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Motor Innervation of the Short Muscles of the Thumb: Anatomic and Clinical Implications

Mohammed O. Salman[1] *MBChB*, **Akram A. Jaffar**[1] *PhD*, **Farqad B. Hamdan**[2] *PhD*

Abstract

Background: The motor innervation of the thumb muscles though important for the hand surgeon, yet it is still a subject of debate since median and ulnar nerves play variable roles.

Objectives: To describe the innervation of the short muscles of the thumb and the possible presence of a 1st palmar interosseous muscle. To correlate the variations of innervation to prognostic values in nerve diseases and injuries.

Methods: A dissection of 15 adult embalmed hands was performed. An EMG study on 42 hands of healthy volunteers was done in which the compound muscle action potential and the interference pattern were studied by sampling separate muscles.

Results: In (86.6%) of the dissections the muscular branch of the median nerve was the first branch in the palm. A median-ulnar anastomosis was found in (53.3%) of the dissected hands, demonstrated at different levels. In spite of special attention to reveal a first palmar interosseous muscle, it was not detected as a separate entity. In the EMG study, innervation showed considerable variations. Adductor pollicis did not receive pure median innervation, it received the ulnar nerve in (90.5%) of the cases. Abductor pollicis brevis did not show a pure ulnar supply, it was mainly supplied by median nerve (66.7%). The highest percentage (66.7%) of a mixed innervation was shown in opponens pollicis.

Conclusions: The palmar median-ulnar anastomosis at different levels makes it vulnerable in surgical interventions. The absence of significant laterality in the mode of innervation of specific muscles may help the prognosis of the affected hand from an EMG study done on the contralateral side. The conventional EMG method does not specify the exact innervation of each muscle. From the prognostic point of view if the method used in this study is applied conventionally, the severity of the injury can be expressed in terms of muscles involved. Many muscles received mixed innervation and will retain their function on the long run.

Key words: thenar muscles, motor innervation, EMG

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Introduction

The gripping mechanism of the palm is an important adjunct to manual dexterity. Of the hand digits, the thumb is as important as the rest of the digits. Functionally speaking, the most important movement of the thumb is opposition. Though many thumb muscles participate in this movement, the thenar muscles are chiefly responsible.

The intrinsic muscles of the thumb are quite ill-defined. The name of thenar eminence is as vague as its content: while French and German speaking anatomists describe a superficial (external) thenar, comprising the abductor pollicis brevis, flexor pollicis brevis, opponens pollicis muscles and a deep (internal) one formed by the adductor pollicis, most English-speaking ones accept under the name of thenar only the first three muscles[1].

The 1st palmar interosseous muscle in the thumb is still debatable. some authorities have preferred to regard the 1st palmar interosseous muscle as a deep striation of flexor pollicis brevis[2]. This issue is to be further clarified herein.

In this study, the German speaking anatomists were followed so the short muscles of the thumb are counted as: opponens pollicis, flexor pollicis brevis,

[1]Dept. Human Anatomy, [2]Dept. Physiology
Address correspondence to: Dr. Akram A. Jaafar,
Email: akramjfr@yahoo.com
Website:
http://www.geocities.com/akramjfr/index.html

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abductor pollicis brevis, and adductor pollicis. Generally speaking, the former three are supplied by the recurrent branch of the median nerve while adductor pollicis is supplied by the ulnar nerve. However, it is neccessory to qualify this statement. Any of the thenar muscles may receive median, ulnar or dual innervation[1,3-6]. The nerve supply of flexor pollicis brevis muscle is subject to more variation than that of any other muscle in the body[4].

Variations in the course and branches of the recurrent branch of the median nerve were reported[5-7]. Because of its recurrent course, the branch from the median nerve to the thenar muscles is vulnerable to lesions that affect these muscles[6]. Median to ulnar communication constitute sources of error in evaluation of nerve coduction velocity & electromyographical (EMG) study[7].

This study aims to report the source of innervation of the thenar muscles through dissection and EMG study of each muscle separately and correlate that to prognostic values in nerve diseases and injuries.

Subjects and Methods

The electromyographical study was performed on 38 apparently healthy volunteers aged between (30y-60y) from both sexes. In two volunteers the muscles on both sides were tested rendering the total number of the hands examined in this study 42. Direct observation of the innervations of the short muscles of the thumb was performed on 15 adult embalmed cadaveric hands of both sexes. In order to restore the softness and flexibility of cadaveric specimen preserved in formalin, the method of Tschernezky[8] was used.

Dissection steps were performed according to Grant' dissector[9] in order to reveal the muscles and follow the branches of the median and ulnar nerves in the palm.

To reaffirm the source of motor innervation of the short muscles of the thumb and explore variations of innervations an EMG was contemplated. This was performed using DANTEC counterpoint 4-channel electromyography.

The test procedure was explained in brief for each subject in order to alleviate any fear, anxiety or apprehension that may be present in the subject. During the procedure the subjects were lying in a supine position on an examination couch. The room temperature was maintained between (25-28°C) during the test procedures. The compound muscle action potential (CMAP) and EMG interference pattern were studied by sampling the four thenar muscles. In addition to stimulation of the median and ulnar nerves while the recording done from each muscle separately.

The stimulation was done using a bipolar stimulating electrode (DANTEC 13L36), which was placed such that the cathode is closer to the recording electrode[10]. The cathode was placed 2-3 cm proximal to the distal crease on the palmar surface, between the flexor carpi radialis and the palmaris longus tendons for stimulation of the median nerve[11]and just over the flexor carpi ulnaris tendon for stimulation of the ulnar nerve[12].

Recording was done by using concentric needle electrodes (DANTEC 13L50). The subject was grounded by special electrode (DANTEC 13S93) with a 15 mm dimension width, usually located between the stimulation & recording electrodes. Muscles were located according to their contraction in action or resisted action[14].

Results

The muscular (recurrent) branch of the median nerve was a thick short nerve that arose from the median nerve at different levels. In (86.6%) of the dissections, it was the first branch of the median nerve in the palm. It may arise as a terminal branch at the same level as the digital branches. Usually it gives branches to the flexor pollicis brevis either crossing it superficially or passing through the muscle itself. In (6.6%) of the cases, the muscular branch appeared in the carpal tunnel and pierced the flexor retinaculum. Yet in another

(6.6%) it arose as a terminal branch at the same level of digital branches.

Median-ulnar anastomosis in the palm

Dissection revealed a connection between median and ulnar nerves in (53.3%) of the hands. The connection was demonstrated at different levels: In (20%) there was a loop connection in the palm just beneath the distal edge of the flexor retinaculum. In (33.3%) there was a connection between the superficial branches of the ulnar nerve with the digital branches of median nerve. In (6.7%) a twig from the deep branch of the ulnar nerve reached the deep stratum of the flexor pollicis brevis after giving its supply to the adductor pollicis muscle while the twig from the median nerve supplies the superficial stratum of flexor pollicis brevis.

The gross anatomy and attachment of the short muscles of the thumb was identical to that mentioned in all established anatomical literature[13,14]. In spite of special attention to reveal any clue of first palmar interosseous muscle. It was not detected as a separate muscle in all the dissected hands.

Variations in the nerve supply of the thenar muscles

In the EMG study, the nerve supply to the short muscles of the thumb showed considerable variations (Table-1).

It is clear from the table that adductor pollicis muscle did not receive pure median nerve innervation. On the other hand, the abductor pollicis brevis muscle did not show a pure ulnar nerve supply. The highest percentage of a mixed innervation was shown in the opponens pollicis muscle (66.7%). The abductor pollicis brevis muscle was mainly supplied by median nerve (66.7%). The adductor pollicis muscle received mainly the ulnar nerve (90.5%).

Table 1: Variations in the nerve supply of the thenar muscles according to the EMG study

Muscles (N=42)	Pure median nerve supply (%)	Pure ulnar nerve supply (%)	Mixed innervations (%)
Abductor pollicis brevis	28(66.7%)	Zero %	14(33.3%)
Flexor pollicis brevis	12(28.7%)	6(14.3%)	24(57%)
Opponens pollicis	4(9.5%)	10(24%)	28(66.5%)
Adductor pollicis	Zero %	38(90.5%)	4(9.5%)

Laterality in the mode of innervation of the thenar muscles

Tables 2 and 3 show pooled observations of right and left hands respectively. In only two cases, the observations were paired. It should be mentioned that in only two cases, right and left hands were examined in the same subject. It is obvious from the EMG procedure that the maneuver is painful for the subject and can hardly be performed for both right and left hands in the same session.

It is clear that the percentage of the mode of innervation of abductor pollicis brevis is exactly identical. However, numerical variations may be suggested when merely observing the percentage of the mode of innervation of the other three muscles, namely flexor pollicis bervis, opponens pollicis, and adductor pollicis.

Chi-square test[15] did not show statistical significance ($p<0.01$) regarding laterality in the mode of innervation of specific muscles.

Table 2: Variation in the nerve supply of the thenar muscles according to the EMG study in the ***right*** hands (total No. of the hands = 30)

Muscles (right)	Pure median	Pure ulnar	Mixed
Abductor pollicis brevis	20 (66.7%)	0%	10 (33.3%)
Flexor pollicis brevis	12 (40%)	0%	18 (60%)
Opponens pollicis	4 (13.3%)	2(6.7%)	24 (80%)
Adductor pollicis	0%	100%	

Table 3: Variation in the nerve supply of the thenar muscles according to the EMG study in the ***left*** hands (total No. of the hands = 12)

Muscles (left)	Pure median	Pure ulnar	mixed
Abductor pollicis brevis	8(66.7%)	0%	4(33.3%)
Flexor pollicis brevis	2(16.7%)	0%	10(88.3%)
Opponens pollicis	0%	4(33.3%)	8(66.7%)
Adductor pollicis	0%	10(83.3%)	2(16.7%)

Discussion

The unsettled issue of the first palmar interosseous muscle

In spite of careful attention through hand dissection to reveal any clue of the first palmar interosseous muscle, it was not detected as a separate muscle in all the dissected hands. In general, a muscle is considered as a separate entity during dissection when it can be easily separated from its surrounding structures. The epimysium that envelopes a muscle can be regarded as a natural boundary that identifies its independent status. For each muscle identified in this study, a clear cleaving fibrous sheath made separation of muscles unambiguous.

To consider the deep stratum of flexor pollicis brevis as a separate muscle[2] and tag it as the first palmar interosseous is an undesirable assumption. Even though these deep fibers have separate innervations, dual innervations, from the ulnar nerve; it is not a justification to consider them as separate entities.

The human body poses many similar examples as in the case of pectineus muscle (femoral and obturator nerve supply); adductor magnus is yet another example being supplied by the sciatic and obturator nerves; However, neither of these muscles were considered to be two separate entities, but were considered as a composite muscle on the account that no fibrous cleavage was found between its components.

To consider flexor pollicis brevis muscle as a composite muscle on the account of the above mentioned similarities may be more justified than considering it as two separate muscles. Even this, we suppose, is adopted to cheer supporters of four palmar interossei muscles as opposed to the well established four dorsal interossei muscles.

Furthermore, functionally speaking, the palmar interossei adduct the fingers. Thus the thumb requires no palmar interosseous muscle because of the thumb already possessing its own powerful adductor pollicis muscle.

Palmar neural anastomosis: another alert to surgeons

The palmar anastomosis between median and ulnar nerves at different levels especially the anastomosis in the palm beneath the distal edge of the flexor retinaculum makes this anastomosis vulnerable in the surgical interventions, like the carpal tunnel release. Surgery literature[16-18] did not bring this issue to the alert of surgeons operating in the palm field.

The high percentage (53.3%) of the presence of anastomosis and the variation in

its location as revealed in this study should be considered by surgeons to be avoided in procedures carried on to decompress carpal tunnel in the carpal tunnel syndrome, drain palmar abscess and to free Dupyturen's contracture.

Variation in the mode of origin of the recurrent branch of the median nerve:

The mode of origin of the recurrent branch of median nerve closely confirm with another study[6] that reported the recurrent branch of median nerve as the first branch in the palm in 83.3% of the cases. This usual position of the motor branch makes it vulnerable in blind median nerve decompression. The surgeon is thus advised to release the retinaculum starting at its proximal edge.

Bilateral identical innervation:

The results of the present study concluded that there was no significant laterality in the mode of innervation of each of the thenar muscles studied. To our knowledge, no literature dealt with this issue. According to current results, it may be said that when one hand is affected by disease or injury of its nerves, an EMG study performed on its lateral counterpart may help the physician to have a better idea about the prognosis of the affected hand since both hands are likely identical regarding the mode of innervation of muscles. If thenar muscles were shown to have a double ulnar and median innervation then an injury of the median nerve will prove less crippling to the thumb of the patient bearing in mind the ulnar nerve compensation especially for flexor pollicis brevis and opponens pollicis (Table-1).

The EMG method used in this study versus conventional EMG study

The conventional EMG method records the compound muscle action potential (CMAP) from the thenar muscles as a bulk. It considers thenar muscles as being supplied by the median nerve. Thus, the conventional method does not specify the exact innervation of each muscle as has been demonstrated in this study.

With the EMG conventional method, to say that a specific nerve was severely or mildly injured, this depends on many observations such as including increased insertion activity, spontaneous activity of positive sharp waves, fibrillation and/or fasciculation potentials and a decrease in the sensory action potential and/or CMAP amplitude. In addition to prolonged latencies and a decrease in the conduction velocity or even absent responses. Yet, in clinical practice, many patients though they suffer a complete injury as revealed in conventional EMG method they can still perform almost normal movements of the thumb and even the bulk of the thenar muscles is affected to a small extent after a considerable time following the injury. This could attract the attention of the electromyographer and may mislead him.

From prognostic point of view if the method used in this study is applied conventionally, the severity of the injury can be expressed in terms of muscles involved. Many muscles as revealed in this study received mixed innervation and will retain their function on the long run.

The possible drawback of the EMG method used in this study may be the multiple painful needle insertion sites. Anatomically speaking, there might be a possible radial artery injury. The radial artery passes into the hand between the two heads of 1st dorsal interosseous muscle then enters the two heads of adductor pollicis to form the deep palmer arch. The penetration of the recording needle to adductor pollicis muscle may jeopardize the radial artery or its branches leading to radial artery injury and bleeding. This problem should be kept in mind though it was not confronted in this study.

Studies to date have been criticized in that volume conduction from adjacent muscles and stimulus spread to adjacent nerves can complicate electrophysiologic interpretation[17]. The use of collision studies may lessen the likelihood of these pitfalls, but it dose not eliminate them[18].

Only anatomic confirmation can be considered definitive.

Refrences

1. Fahrer M.: The thenar eminence: an introduction. In Tubiana R.: The hand vol.1. Saunders. Phladelphia. 1981; p.p. 255-8.
2. Williams PL and Warwick R: Gray's Anatomy. 36th ed. Churchill Livingstone. Edinbourgh. 1980; p.p. 590, 378.
3. LoMonaco M, Padua L, Gregori B, Valente EM, and Tonali P: Ulnar innervation of the thenar eminence with preservation of median innervation of first lumbrical muscle. Muscle Nerve, 1997; 20: 629-30.
4. Ganes T: Complete ulnar innervation of the thenar muscles combined with normal sensory fibers in a subject with no periphral nerve lesion. Electromyogr Clin Neurophysiol, 1992; 32: 559-63.
5. Backhouse KM: Nerve supply in the arm and the hand. In Tubiana R.: The hand. vol. 1. Saunders. Philadelphia. 1981; p.p. 275-90.
6. Chiou TF: EMG secrets. Hanley and Belfus. Philadelphia. 2004; p. 61.
7. McMinn RMH: Last's anatomy: Regional and applied. 9th ed. Churchill Livingstone. Edinburgh. 1998; p.p. 109.
8. Rotman MB and Donovan JP: Practical anatomy of the carpal tunnel. Hand Clin 2003; 18: 219-30.
9. Okuno Y and Kasai T: Anatomical studies on the thumb muscles of the human hand. Kaibogaku Zasshi, 1995; 69: 765-75.
10. Falconer D and Spinner M: Anatomic variations in the motor and sensory supply of the thumb. Clin Orthop, 1985; 195: 83-96.
11. Olave E, Prates JC, Gabrielli C, and Pardi P: Morphometric studies of the muscular branch of the median nerve. J Anat, 1997; 189: 445-9.
12. Uncini A, Lange DJ, and Lovelace RE: Anomalous intrinsic muscle innervation in median and ulnar nerve lesions: an electrophysiological study. Ital. J Neurol Sci, 1989; 9: 467-503.
13. Tschernezky W: Restoration of the Softness and Flexibility of Cadavers Preserved in Formalin. Acta Anat, 1984; 118: 159-63.
14. Sauderland EK: Grant's dissector. 9th ed. Williams & Wilkins. Baltimore. 1984; p.p. 115.
15. Buchthal F: Electrophysiological findings in entrapment of the median nerve at wrist and elbow. J Neurol Neurosurg Psychiat, 1974; 37: 340-60.
16. Melvin JL, Harris DH and Johnson EW: Sensory and motor conduction velocities in the ulnar and median nerves. In Delisa JA and Mackenzie K (ed.) Manual of nerve conduction velocity techniques. Raven Press. New York. 1966. p.p. 33-6.
17. Chu-Andrews J and Johnson RJ: Electrodiagnosis An Anatomical and Clinical Approach. Lippincott. Philadelphia. 1986; p.p. 304(b).
18. Snell RS: Clinical Anatomy for Medical Students. 6th ed. Lippincott. Philadelphia. 2000; p.p. 461.
19. Moore KL and Dalley AF: Clinically oriented anatomy. Williams and Wilkins Pheladelphia. 1999; p.p. 777.
20. Duncan RC, Knapp RG and Clinton Miller M: Introductory biostatistics for the health sciences. 2nd ed. Wiley. New York. 1983; p.p. 161-78.
21. Dudley HAF: Hamilton Bailey's emergency surgery. 11th ed. Wright. Bristol. 1986; p.p. 655-67.
22. Townsend CM and Harri JW: Sabiston textbook of surgery. 16th ed. Vol.3. Saunders. Philadelphia. 2001; p.p. 1570-88.
23. Mann CV, Russell RCG and Williams NS: Bailey and Love's: short practice of Surger. 22nd ed. Chapman & Hall. Madrid. 1999; p.p. 334-7.
24. Dumitru D: Electrodiagnostic Medicine. Hanley and Belfus. Philadelphia. 1995; p.p. 144.
25. Kimura J, Murphy MJ and Varda DJ: Electrophysiological study of anomalous innervation of intrinsic hand muscles. Arch Neurol, 1976; 33: 842-4.

HISTOLOGICAL CHANGES IN PLACENTA OF PATIENTS WITH PRE ECLAMPSIA

Kawkab Al-Kaisy[1], May F. Al-Habib[2] *MSc, PhD*, Shatha T. Al-Zuheiri[3]

Abstract

Background: Preeclampsia is a major disease of human reproduction, with 10% of human births being affected, mainly a systemic endothelial disease causing activation of platelets and diffuse ischemic disorders.

Objectives: The study aims to demonstrate the histological changes in placenta of women suffering from hypertensive disease.

Methods: Placental samples were obtained from 15 healthy uncomplicated pregnancies and 35 pregnancies complicated by intrauterine growth restriction due to sever preeclampsia. Samples were prepared and examined by light microscope.

Results: Variable changes have been observed in different patients, including degeneration of most of endothelial cells, degeneration of major trophoblast cells, hyalinization and fibrotic trophoblast cells in some patient sections and fatty infiltration within trophoblast cells in other patient sections.

Conclusion: The primary cause of preeclampsia is a disturbed growth of trophoblast cells with the degeneration of major endothelial cells.

Key words: Preeclampsia, Placenta, histological changes.

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Introduction

The hypertensive disorders of pregnancy complicate about 7-10% of all pregnancies[1]. Pregnancy induced hypertension, which includes preeclampsia, and eclampsia is responsible for 70%, where as chronic hypertension represents 30% of hypertensive disorders in pregnancy. Gestational hypertension (Preeclampsia) is a major disease of human reproduction, with 10% of human births being affected[2].

Preeclampsia is mainly a systemic endothelial disease causing activation of platelets and diffuse ischemic disorders[3,4]. It is characterized by generalized activation of maternal endothelial cells. Oxidative stress of the placenta is considered a key intermediary step, precipitating deportation of apoptotic fragments into the maternal circulation[5,6].

Since studies over the past decade have provided a better understanding of the potential mechanisms responsible for the pathogenesis of preeclampsia, the initiating events in preeclampsia has been postulated to be reduced uteroplacental perfusion as a result of abnormal cytotrophoblast invasion of spiral arterioles. Placental ischemia is thought to lead to widespread activation dysfunction of the maternal vascular endothelium that results in enhanced formation of endothelium and thromboxane[7,8].

The present paper was designed to study the histological changes in placenta of women suffering from hypertensive disease.

Materials and Methods

This work was carried out in the department of Obstetrics and Gynecology, at Al-Kadhiymia Teaching Hospital in Baghdad, for a period of ten months, from 1st August, 2000 to the 1st June 2001. 50 women were enrolled in the study, all were in early labor and sharing the following criteria:

1. All were singleton pregnancies.
2. Their parities were between 0- 4,

[1]Electron Microscopy Unit, [2]Dept. Human Anatomy-Section of Histology, [3]Dept Obstetric & Gynecology, College of Medicine, Al-Nahrain University.

Address correspondence to Dr. Kawkab Al-Kaisy.

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3. All had full term pregnancies, confirmed by known last menstrual period and early ultrasound report.

They were divided into two groups. The first group (normal or control group) consisted of 15 women who had the following additional criteria:

1. No history of medical problems, smoking or drug intake.
2. No history of obstetrical problems.
3. Their newborns had birth weight within the 10th percentile of the individualized birth weight ratio.

The second group (patients group) consisted of 35 women with pregnancies complicated by intrauterine growth restriction (IUGR) and had the following additional criteria:

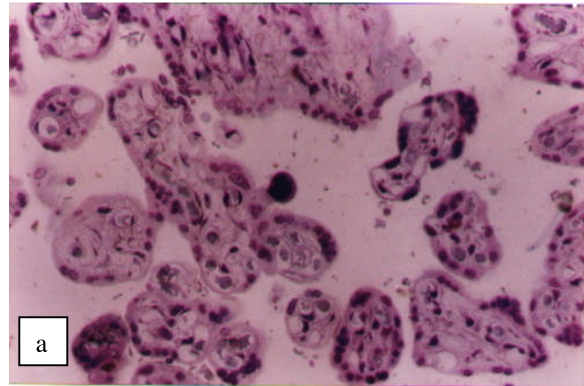
1. All had history of risk factors of complicated current pregnancy with pre-eclampsia or history chronic hypertension.
2. Examination of these women revealed inadequate fundal growth.
3. They had an ultrasonographic evidence of (IUGR), deviation from an appropriate growth percentile depending on biparietal diameter, head and abdominal circumferences measurements, with amniotic fluid index of less than 10cm.
4. Their newborns had birth weight less than the 10th percentile of the individualized birth rate ratio.

Tissues Preparation

Placental tissue samples were obtained from the outer area of the maternal surface of the placenta and cut into small pieces (2x2x2 mm) and prefixed in 2.5% Gluteraldehyde in phosphate buffer pH(7.2), the specimens were prepared for semi-thin sections of 0.5- 1um, stained with 1% Methylene blue and special stain consists of Azar II and Basic fuchsin[9].

Results

In comparsion with normal women group (control group) (Figures 1a and b) shows variable changes in cells of placenta were observed including these following changes:



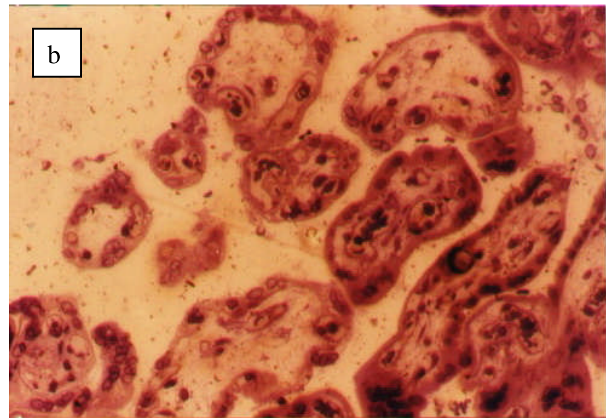


Figure 1a & b: Placental sections from normal women show normal appearance of placental cells.
a: Methylene blue (100X).
b: Basic fuchsin & Azur II (200X).

1. All placental sections of the diseased women have shown an acute type of inflammatory reaction of moderate degree expressed as inflammatory cells infiltration (Figure 2).

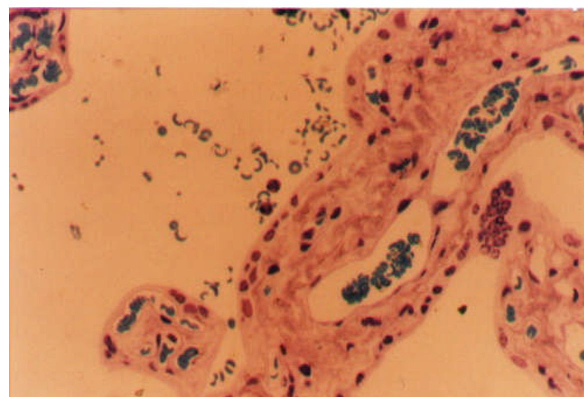


Figure 2: Placental section of hypertensive women shows inflammatory cells.
Basic fucshin – Azur II (100X).

2. All placental sections of preecalmptic women have shown a degeneration of some endothelial cells that reduced the thickness of vessel wall lead to occurrence of hemorrhage (Figure 3).

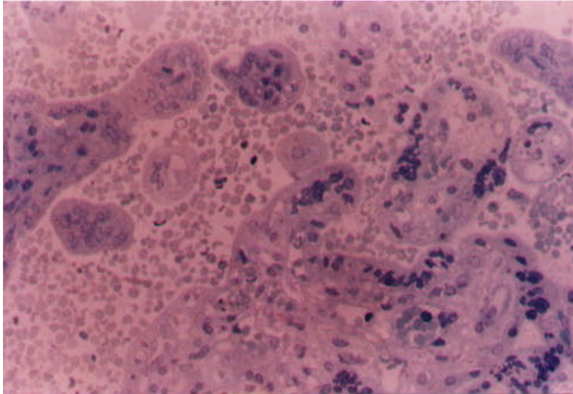


Figure 3: Placental section of hypertensive women shows haemorrage.
Methelene blue (100X)

3. In 70% of patients, fatty infiltration within trophoblast cells was observed (Figure 4).

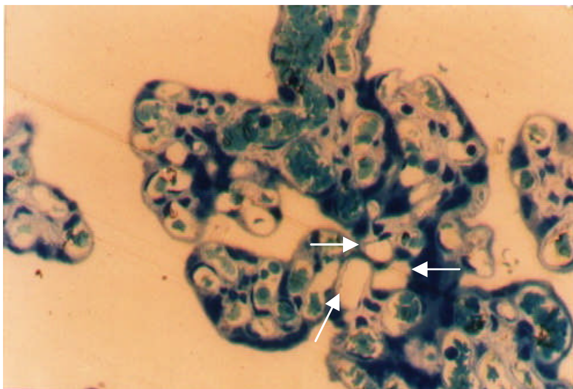


Figure 4: Placental sections of diseased women showing fatty infiltration within trophoblasic cells.
Methylen blue (200X).

4. Placental sections from other women with pre eclampsia, showed a hyalinization and fibrotic trophoblast cells with the degeneration of major trophoblast cells (Figure 5).

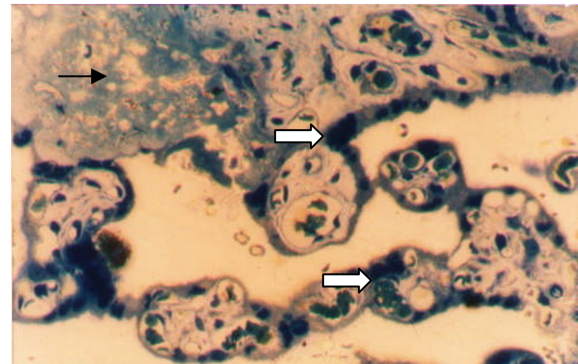


Figure 5: Placental section of hypertensive women showing hyalinization (⟶), and degeneration of trophoblastic cells (⇨)Methelyn blue (200X)

Discussion

Normal pregnancy is associated with reductions in total vascular resistance and arterial pressure possibly due to enhanced endothelium dependent vascular relaxation and decreased vascular reactivity to vasoconstrictor agonists. These beneficial haemo-dynamic and vascular changes do not occur in women who develop preeclampsia; instead, severe increases in vascular resistance and arterial pressure are observed[10].

Preeclampsia is triggered by various factors which can be immunological, vascular, or abnormalities of haemostasis. These defective placental results in a systemic endothelial disease with vasoconstriction[8].

In our study degeneration in major endothelial cells were observed, which reveled the explanation of abnormalities of haemostasis. These changes was accompanied with the degeneration of trophoblast which considered as the primary cause of preeclampsia[11]. This state is accompanied with the hemorrhage which reflects an absolute or relative placental ischemia due to vascular disease or hypertrophic placenta[12].

Moreover, in addition to these changes a fatty infiltration in the placental sections of some patients while the histological examination of other patients reflects a state of hyalinization, these

changes could be referred to the changes in the decidual cells, since the change in cell composition results in certain disturbance of physiological equilibrium of biological active substances produced by the decidual cells[13].

From this study, we can conclude that the primary cause of preeclampsia is a disturbed growth of trophoblast cells with the degeneration of or endothelial cells.

Reference

1. Sibai BM: Preeclampsia-eclampsia current problems in obstetrics gynecology and fertility. 1990; 13: 1-45.
2. Sibai BM: chronic hypertension during pregnancy. In: Sciarra J (ed): Gynecology and Obstetrics. Phelafelphia: JB Lippincott, 1989; p.p.1-18.
3. Robillard PY: Interest in preeclampsia for researchers in reproduction. J Reprod Immunol, 2002; 53(1-2): 279-87.
4. Perloff D: Hypertension and pregnancy-related hypertension. Cardiol Clin, 1998; 16(1): 79-101.
5. Fournier A, Fievet P, el-Esper I et al.: Hypertension ands pregnancy. Diagnosis, physiology and treatment. Schweiz Med Wochenschr, 1995; 125 (47): 2273-98.
6. Hung T, Skepper JN, Charnock JD et al.: Hypoxia a potent inducer of apototic changes in the human placenta and possible aetiological factor in preeclampsia. Circ Res, 2002; 90 (12): 1274-81.
7. Ishihara N, Matsuo H, Murakoshi H, et al.: Increased apoptosis in the syncytiotrophoblast in human term placentas complicated by either preeclampsia or intrauterine growth retardation. Am J Obstet Gynecol, 2002; 186 (1): 158-66.
8. Granger JP, Alexander BT, Llinas MT et al.: Pathophysiology of hypertension during preeclampsia linking placental ischemia with endothelial dysfunction. Hypertension, 2001; 8 (3): 718-22.
9. Beaufils M: Arterial hypertension of the pregnant woman. Ann, 1999; 150(5): 425-31.
10. Hayat MA: Basic technique for transmission electron microscopy. Acad Press Inc, 1986; p.p. 226-31.
11. KhaliI RA and Granger JP: Vascular mechanisms of increased arterial pressure in preeclampsia: lesions from animal models. Am J Regul lntegr Comp Physiol, 2002; 283 (1): 29-45.
12. Wolf G, Wenzel U, Stahl RA, and Huneke B: Hypertensive Erkrankungen in der Schwangerschaft. Med Kim, 2001; 96 (2): 78-86.
13. Vinatier D, and Monnier JC: Pre-eclampsia: physiology and immunological aspects. Eur J Obstet Gynecol Reprod Biol, 1995; 61(2): 85-97.
14. Kottsova NA, Bystrovs OA, Susloparov LA, and Mikhailov VM: Changes in the cellular composition of the deciduous membrane in physiologic pregnancy and late toxicosis. Arkh Anat Gistol Embriol, 1989; 97 (11): 76-82.

ISOLATION AND PURIFICATION OF MYELOPEROXIDASE FROM HUMAN POLYMORPHONEUCLEAR-CELLS (PMN)

Essam F.A. Al-Jumaily[1] *BSc MSc PhD*, **Muntaha A.K. Al-Safar**[2] *BSc MSc PhD*, **Rhagad A.A. AlSuhail**[3] *BSc MSc PhD*

Abstract

Background: Myeloperoxidase (MPO) oxidoreductase, EC.1.11.1.7 stored in granules of neutrophils ingest microorganisms by generating of reactive oxidants.

Objective: Isolation and Purification of (MPO) from polymorphonuclear cells.

Methods: The enzyme was purified from polymorphonuclear blood cells by Ion exchange chromatography by CMC and gel filtration Sephacryl S.200 column and SDS electrophoresis.

Results: Polymorphonuclear cell (PMN) were isolated from human blood; cell extract was prepared by homogenizetion of cell pellets in 0.34 M sucrose. Human (PMN) Myeloperoxidase (MPO) has been purified to homogeneity by two- steps procedure, which included CM-cellulose ion exchange chromatography and Sephacyl S- 200 column at purification fold and recovery of 1.281 and 43.94% respectively. The final product was homogeneous when examined by SDS -polyacrylamide gel electrophoresis. The molecular weight of the enzyme is 80,000 daltons as determined by SDS- PAGE and 88,000 daltons by Sephacyl S-200.

Conclusion: The purification of MPO with accepted yield may open new approaches for its using in the medical application as preparing of monclonal antibodies and diagnostic kits for detection of antimyeloperxidase that are required for some inflammatory diseases

Keywords: Polymorphonulear cells; Myeloperoxidase

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Introduction

Myeloperoxidase (MPO) (donor, hydrogen peroxide oxidoreductase, E.C. 1.11.1.7 is a heme-containing enzyme stored in immense amounts in azurophilic granules of neutrophils, these granulocytic cells ingest microorganisms into phagosomes where it is killed by generating an array of reactive oxidants[1,2]. It is assumed that MPO acts by producing hypochlorous acid, HOCl, which is also likely to contribute to the tissue damage caused by neutrophils at sites of inflammation[3,4]. MPO belongs to the mammalian peroxidase superfamily the enzyme is a disulfide-linked dimer ($□□_2$) of 145KDa with each heavy subunit containing a heme group and this enzymes includes eosinophil peroxidase, lactoperoxidase, thyroid peroxidase[5,6].

The enzyme represents 5% of neutrophil 1% of monocyte protein but has been believed to be absent from macrophags[7]. MPO is a major neutrophil protein and may be involved in the nitration of tyrosine residues observed in a wide range of inflammatory diseases that involve neutrophils and macrophage activation, MPO is released into the extracellular medium where its measurement can be used as an index of neutrophil activation[8]. Most of hydrogen peroxide generated by neutrophils is consumed by MPO[9].

Methods

Isolation of polymorphonuclear cells (PMN). The method of vasiliauskas was followed by[10] and the blood was aspirated from 40 healthy donor's.

Purification of human leukocyte Myeloperoxidase:-

[1]Genetic Engineering and Biotechnology Institute, Baghdad University, [2]Health Dept. Medical Tech. Institute, Baghdad, [3]Dept. Biology, Science College, University of Baghdad.
Address correspondence to be addressed e-mail samgen992003@ yahoo.com
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First step: *Preparation of crude extract*
1- (PMN) extracts were freezer and thawed several times[11].
2-Pellet suspended in 0.34 M sucrose
3-This is considered the crude.

Second step: *CM-Cellulose column chromatography*

The concentrated crude cell extract which contained all the Myeloperoxidase and then applied to a column of CM-Cellulose (3.5x15cm) was equilibrated with 0.02M sodium acetate and 0.1M NaCl (pH=5.0) overnight at 4°C . The column of CMC washed with 60ml of the same buffer and the enzyme was eluted with a linear gradient in the same buffer from 0-0.5M, NaCl, total gradient was 150ml, fractions of 3ml were collected.

Three step: *Sephacryl S-200 column chromatography*

The active concentrated Myeloperoxidase was placed on column (2 x 70 cm) which had been equilibrated with 0.1 potassium phosphate (pH=7.3) and washed with the same solvent. Fractions of 3ml were collected.

Estimation of Myeloperoxidase activity:

Myeloperoxidase activity was determined by the method of Chance and Maehly[12]. The reaction mixture (3ml) contained 1ml of 50mM Sodium phosphate (pH=7.3). 2ml of 20mM guaiacol, 20ml of 40mM H_2O_2 and then enzyme. The reaction was started by adding H_2O_2 and increase in absorbance at 470nm was followed in Spectrophotometer. One unit Myeloperoxidase was defined as the amount of enzyme causing increase of 1 unit in the absorbance at 470nm in 1min. at 20°C under these Conditions.

Protein Determination:

The protein content of the cell free extract was determined by the method of Barford *et al.*,[13]. With Bovine Serum Albumin (BSA) as the standard.

Sodium Dodecyl Sulphate-Polyacrylamide Gel Electrophoresis (SDS-PAGE).

This was parformd as described in Garfine[14] for the Lamemmli system using Bio-Red vertical slab gel cell. Electrophoresis utilized a 0.1M Tris-glycine buffer, pH=8.3 and 1% SDS and 2-mercaptoethanol. A 7.5% gel was employed.

Results

The purification of human (MPO) from (PMN) cells (Table I) included two steps, firstly, CM-cellulose ion exchange chromatography, in this step one peak was obtained (Figure1) which had fold and recovery of 2.96 and 92.12% respectively.

Table 1: Purification of Myeloperoxidase form human neutrophil

Sample	Volume	Enzyme activity u\ml	Total unit	Protein mg\ml	Specific activity u\mg	Fold	Yield
Crude	20	117.3	2346	1.35	86.9	1	100
CM-Cellulose	12	180.09	2161.08	0.7	257.27	2.96	92.12
Sephacryl S-200	9	105.5	949.5	0.32	329.69	1. 281	43.94

The active product of the CMC step which was loaded on Sephacyl S-200 column gave one peak (Figure 2) and with fold and yield 1.281; 43.94% respectively. The specific activity of the purified human MPO was 234.44 unit/ mg.

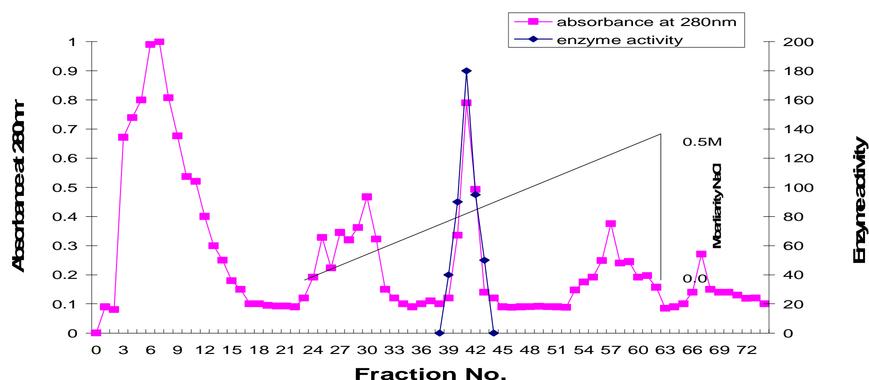

Figure 1: CM-Cellulose chromatography of human polymorphonuclear myeloperoxidase fractions obtained from crude sample. The column (3.5x15 cm) was equilibrated with 0.02M sodium acetate and 0.1M NaCl, pH 5.0 and eluted with a linear gradient in the same buffer from 0-0.5M NaCl. Total gradient was 250 ml.

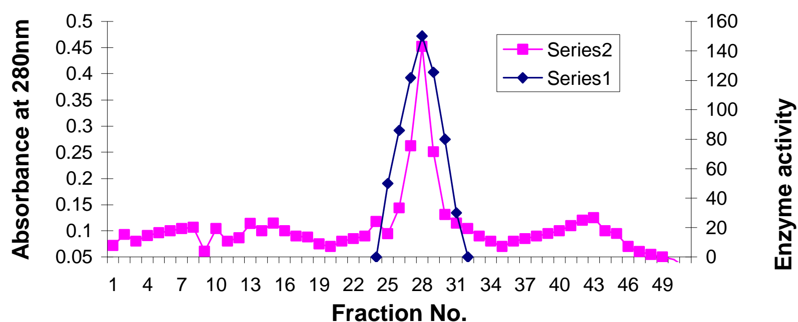

Figure 2: Sephacryl S-200 gel filtration of myeloperoxidase. Concentrated solution from CM-Cellulose (3 ml) was loadead on a Sephacryl S-200 column (2x70 cm) which was equilibrated Myeloperoxidase was eluted with the same solution and fractions of 3 ml were collected

The molecular mass estimated by two methods; the gel filtration by using Sephacryl S-200 column and with the standard molecular weight protein (Figure 3). The molecular weight was estimated to be 88,000 Daltons .The second method was SDS-polyacrylamide gel electrophoresis, the purified (MPO) appeared as one band at position corresponding to molecular weight of 80,000 daltons (Figures 4 and 5).

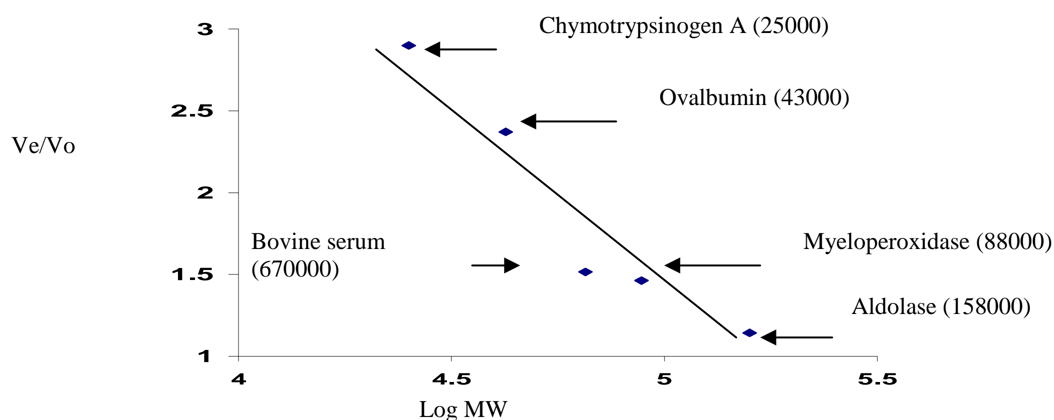

Figure 3: Determination of molecular weight for human MPO by gel filtration by using Sephacryl S-200 Column (70x2 cm)

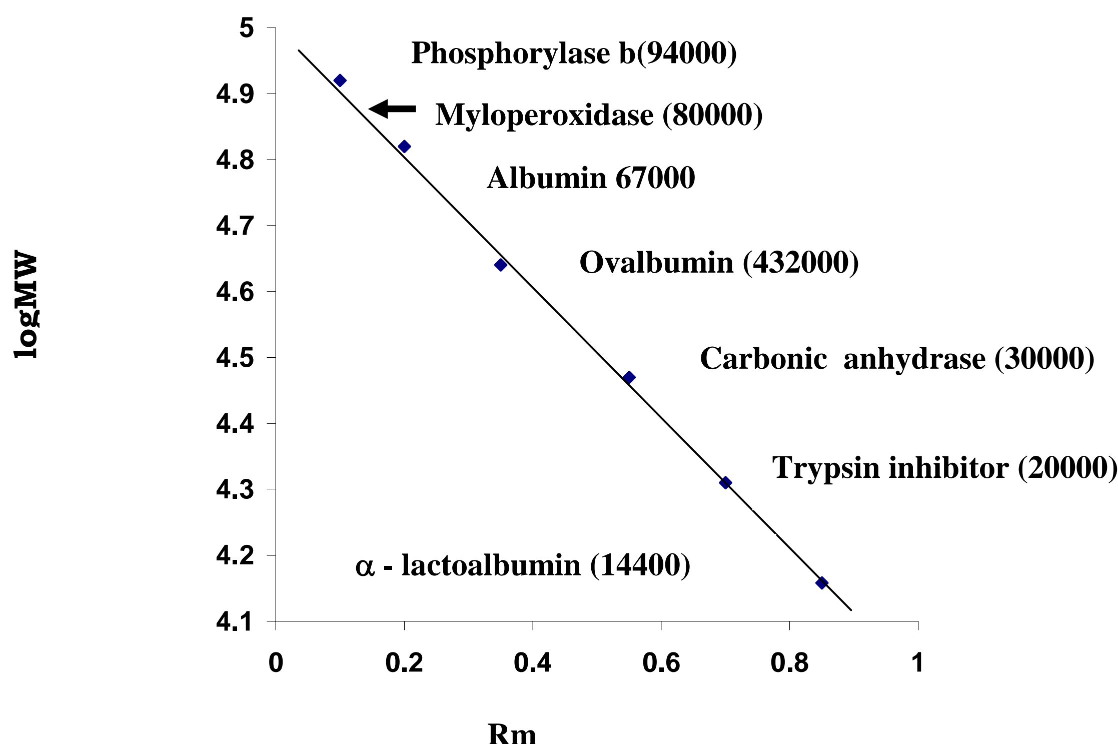

Figure 4: Determination of molecular weight for human MPO by SDS polyacrylamide gel electrophoresis.

In previous studies human (MPO) was purified from Leukocytes of pool peripheral blood from several donors, heterogeneity has been observed in the purified (MPO) obtained in this way possibly due to the heterogenecity of its source and several form of human (MPO) have been separated by polyacrylamide gel electrophoresis[15].

Discussion

Matheson[16] purified human leukocyte (MPO) to homogeneity by three steps namely, dialysis of agranule extract against low salt buffer, Sephadex G-75 chromatography and Carboxy methyl cellulose chromatography. The final yield of activity was excellent and represented 79% of the orginal activity in leukocyte homogenate, the final homogeneous when examined by acid polyacrylaimde gel electrophoresis and Sedimentation equilibrium ultracenterfugation, while[17] purified human (MPO) from Leukemia HL-60 by Carboxymethyl Sepharose CL-6B column chromatography, and Sephacryl S-200 gel Filtration with 31.2% yield and 737.1 unit/mg specific activity. They found that MPO consisted of a small size; Mr 79,000 daltons in addition to large size Mr, 153,000 daltons the small MPO differed in immunological properties from large MPO.

Brown[18] purified human myloperoxidase to sodium dodecyl sulfate-polyacrylaimde gel electrophoresis and a minor band with apparent molecular masses of 60000 Daltons and 15000 Daltons respectively, were recognized by both antibodies- under reducing and denaturing conditions on polyacrylaimde gel electrophoresis , human myelperoxidase gave rise to bands of Mr 57,000;39,000;500[5].

Kettle[19] purified (MPO) from neutrophils, azurophilic granules released by sonication of cells are extracted using cetyltrimethyl- aminonium bromide (CTAB), purification of the enzyme was

done by column chromatographys using concanavalin A bound Sepharose followed by CM-Sepharose, final purification achieved by Chromatography on a phenyl - Sepharose column.

The purification of MPO with accepted yield may open new approaches for its using in the medical application as preparing of monclonal antibodies and diagnostic kits for detection of antimyeloperxidase that are required for some inflammatory diseases[19-21].

References

1. Spry CJF: Eosinophils: Comprehensive Review &Guide to the Medical Literature. Oxford university Press, Oxford, U.K., 1988.
2. Arnhold J, Paul GF, Gunther R, and Christian O: Redox properties of the couple compound I/native enzyme of myeloperoxidase and eosinophil peroxidase. Eur J Biochem, 2001; 268: 5142-8.
3. Klebanoff SJ: Myeloperoxidase: occurrence & biological function in peroxidases in chemistry and Biology (Everse J, Everese KE, and Grisham MB eds), VRC Press, Boston, MA., 1991; p.p.1-3.
4. Heinecke JW: Mechanisms of oxidative damage of low-density lipoprotion in human atherocderosis. Curr Pin Lipid. Biochemistry, 1997; 8: 268-74.
5. Olson RL, and little C: Purification and some properties of myeloperoxidase and eosinophil peroxidase from human blood. Bichem J, 1983; 209: 781-7.
6. Kimara S, and Ikeda-satio M: Human Myeloperoxidase and thyroid peroxidase two enzymes with separate and distinct physiological functions are evolution aridly related members of the same genefamily proteins. Biochem J, 1988; 3: 113-120.
7. Tobler A, and koeffler HP: Myeloperxidase: localization, structure and function. Blood cell Biochem, 1991; 3: 255-88.
8. Burner U, Paul GF, Kettle, AJ, Koppennol, WH, and Obinger C: Mechanism of Reaction of Myeloperoxidase with nitrite. J Biolo Chem, 2000; 275(27): 20597-601.
9. Hampton MB, kettle AJ, and Christine C: Winterbourn. Inside the neutrophil phagosome: oxidants, Myeloperoxidase and Bacterial Killing. Blood, 1998; 92(9): 3007-17.
10. Al-Suhail RGA: Study of anti-neutrophil cytoplasmic antibody response in inflammatory Bowel disease. PhD Thesis. Science College, Baghdad University, 2003.
11. Yamada M, Midori M, and Takashi S: Purification and characterization of small Molecular weight Myeloperoxidase form human promyelocytic leukemia HL-60-cells Biochmistry, 1981; 20: 766-71.
12. Chance B, and Maehly AC: Methods in Enzymology, 1955; 2: 764-75 (Cited from Yamada 1981).
13. Bradford MM: A rapid and sensitive method for the quantitation of microgram, quantition of protein utilizing the principle of protein dye binding. Analy Biochm, 1976; 72: 248-54.
14. Garfin DE: Purification procedure: electrophoretic methods. In: Methods in Enzymology (Deutscher MP edition). Vol. 182: 425-441, Academic Press, New York, 1990.
15. Felbery NT and Schultz J: Arch Biochem Biophys, 1972; 148: 407-13 (cited from Yamada *et al*., 1981).
16. Metheson NR, Wong PS, and Travis J: Isolation and properties of human neutrophil Myeloperoxidase. Biochemistry, 1981; 20: 325-30.
17. Brown KE, Brunt EM, and Heinecke JW: Immunohistochemical Detection of Myeloperoxidase and its oxidation products in kupffer cells of human livers. Am J Pathol, 2001; 159: 2081-8.
18. Kettel AJ: Detection of 3-chlorotyrosine in proteins exposed to neutrophil oxdants. Methods Enzymol, 1999; 300: 111-20.
19. Nakamura RM, and Barry M: Serological markers in inflammatory bowel diseases. Inflamm Bowel Dis, 2001; 8: 9-15.
20. Cabral VL, Miszputn J, and Catapani WR: Antineutrophile cytoplasmic antibodyies in Brazilian patients with inflammatory Bowel disease. Hepto Gastroenterol, 2003; 50: 412-5.
21. Vasiliauskas E, Kam LY, Karp C, Gaiennie J, Yang H, and Taragan SR: Marker antibody expression stratifies Crohn's disease into immunologically homogeneous subgroups with distinct clinical characteristics. Gut, 2000; 47: 487-96.

ROLE OF TOTAL AND LIPID -BOUND SIALIC ACID IN DISCRIMINATIG ACTIVITES OF RHEUMTOID ARTHRIRIS PATIENTS

Alaa K.Mohammed[1] *Ph.D* , **Yahya Y.Z.Farid[2]** *Ph.D* **Israa S.Mohammed[1]** *B.Sc*

Abstract:
Background: It is known that total serum sialic acid (TSA) and lipid bound sialic acid (LSA) levels may be altered by different types of disease included Rheumatoid Arthritis (RA) and inflammatory disease, therefore the evaluation of these compounds in serum patients with RA may elucidate the relationship between its levels and disease activity.

Aim: This study was designed to evaluate the clinical application of serum total sialic acid (TSA) and lipid bound sialic acid (LSA) levels in patients with RA, considering the disease activity and compare the levels with a normal group.

Method: The study was carried out on ninety-seven healthy and sick adults. Fifty-four (37 female, 17 male) patients with RA disease. Of them 29 patients (21female, 8 male) had low disease activity [age range 32-65 years] and twenty-five patients (16 female, 9 male) had high disease activity [age range 22-52 years]. They were compared with 43 healthy persons [age range 19-64 years]. Colorimetric methods were used for determination TSA and LSA in serum samples.

Results: Total sialic acid and lipid bound sialic acid levels in serum patients with RA show significantly increased when compared to normal group, and more increased in patients with high activity disease.

Conclusions: Based on our results, serum TSA and LSA level would be used as a good marker for discriminating between activities of RA disease.

Key words: Rheumatoid Arthritis, sialic acid, and lipid bound sialic acid.

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Introduction

Sialic acid (SA), a class of important ketoses that contain nine-carbon atom, is an acetylated derivative of neuraminic acid (2-keto-5-amino-3, 5-dideoxy-d-nonulosonic acid)[1]. Sialic acid is widely distributed in nature as non-reducing termini of glycoprotein and glycolipids. About 70% of the total sialic acid (TSA) of eukaryotic cell is found on the cell surface and the remainder is distributed primarily in the endoplasmic reticulum, mitochondria and lysosomes[2].

Because of their acidic nature, SA impart a negative charge to the cell surface and are important in cell-to-cell or cell-to-matrix interactions.SA residues on the cell surface may also be involved in masking cell surface antigens and may serve as receptors for lectins, virus particles, some hormones and antibodies[3].However, they can also act as critical components of ligands recognized by a varity of animal, plant and microbial proteins termed sialic acid binding lectins[4].

In human, SA is present in Alpha-1-acid glycoprotein(AAG), haptoglobin, ceruloplasmin and transferrin , which are acute phase rectants[5,6]. Sialic acid levels vary physiologically with age, but their levels may also be influenced by such conditions as inflammation, neoplastic tumor or in born genetic disorders, which cause abnormal sialic acid metabolism[7].

Marked elevation of serum (TSA and LSA) that correlate with the clinical activity of a disease have been documented in many pathological states, included cardiovascular disease, different types of cancer and inflammatory reaction, where the

[1]Dept. Chemistry, College of Education (Ibn-Al-Haitham), Baghdad University, [2]Dept.Chemistry and Biochemistry, College of Medicine, Al-Nahrain Uiversity.
Address Correspondence to: Dr. Alaa K. Mohammed. E-mail:Alaakareem@maktoob.com.
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underlying pathology is either of tissue destruction, tissue proliferation, depolymerization or inflammation[8,9]. The role of total sialic acid (TSA) includes a small amount of free sialic acid as well as glycoprotein and glycolipid-bound sialic acid (LSA) in the various pathological processes promoted us to investigate and find some typical differences between the content of TSA and LSA values in sera patient with Rheumatoid Arthritis and compared with normal group.

Materials and Methods

Chemicals: Standard solution for sialic acid of 500µg/ml concentration was prepared by dissolving 50 mg of standard N-acetylneuraminic acid in 100 ml of distilled water, and on the day of determination, the stock solution was diluted with phosphate buffer saline at pH 7.4 to give the following standard solutions (5.0, 10.0, 15.0, 20.0, 25.0, 30.0 µg/ml) for calibration curve measurement.

Subjects: Sera samples of the measurement of TSA and LSA were obtained from five groups of subjects who were attending Rheumatology Outpatient Clinic Division in Baghdad Teaching Hospital- Medical City from April to August 2004.

The patients with RA were divided in to two groups after evaluation of their disease activity by the Tentative EULAR Criteria[10]. [Number of tender joints: maximum 28; number of swollen joints; maximum 28; pain score: zero (no pain) to 10 (very sever pain), erythrocyte sedimentation and morning stiffness of at least 30 min duration].

Group (1) consisted of 43 adult healthy blood donors as control subjects. Mean age was 44.3±4.2 (range: 19-64) with the male to female ratio being 0.72.

Group (2) and (3) consisted of patients (8 male, 21 female) were enrolled with less than 14 swollen joints and one hour morning stiffness were considered to have low activity of the disease.

Group (4) and (5) consisted of 25 patients (9 male, 16 female) had been diagnosed with a higher number of swollen joints and more prolonged morning stiffness, were considered to have a high degree of inflammation. Characteristics of the above groups were presented in table 1 and 2.

Table 1: Characteristics of the Rheumatoid Arthritis Groups (2) and (3) low activity

Character	Group (2)	Group (3)
Sex	Male	Female
No. of patients	8.0	21.0
Age range (yrs)	32-65	19-16
Mean age (yrs)	55.30±1.90	48.8±2.30
Duration of disease (yrs)	6.90±1.10	5.90±1.20
Morning stiffness (h)	0.70±0.20	1.10±0.30
No. of tender joints	15.20±3.0	16.20±2.30
No. of swollen joints	6.10±1.20	8.90±2.0
ESR (mm/h)	36.0±8.0	34.0±2.0
Positive rheumatoid factor	5/8	17/21

Table 2: Characteristics of the Rheumatoid Arthritis Groups (4) and (5) high activity

Character	Group (4)	Group (5)
Sex	Male	Female
No. of patients	9.0	16.0
Age range (yrs)	22-52	19-58
Mean age (yrs)	45.6±7.9	43.8±4.8
Duration of disease (yrs)	5.2±1.2	6.6±2.3
Morning stiffness (h)	3.4±1.1	3.3±0.3
No. of tender joints	18.9±2.3	24.0±0.8
No. of swollen joints	15.1±3.5	14.2±2.5
ESR (mm/h)	65.0±5.0	78.0±5.0
Positive rheumatoid factor	9/9	

Serum preparation:

Venous blood samples were collected by utilizing disposable needle and plastic syringes from each patient and control, and then allowed at room temperature for 10 minutes for clotting; sera were separated by centrifugation at 3000 rpm (10 min) and stored at (-20C^o) until tested for TSA and LSA levels.

Measurement of serum TSA by resorcinol reagent:

Serum TSA values determination was performed as previously described [11]. Briefly, 20 µl of serum was diluted to 500 µl in to screw-capped tubes with distilled water; the tubes were vortexes and placed in ice.

To each tube for TSA test, 1ml of resorcinol reagent (including 10ml 2%(w/v) stock resorcinol in water, 9.75 ml water, 0.25 ml 0.1M $CuSO_4$, brought to a final volume of 100 ml with concentrated HCL). Then each tube was capped, vortexed, and placed in100C^o boiling water (15minutes), then cooled for 10 minutes in an ice bath. One ml of butylacetate/n-butanol (85:15 v/v) was added to the reaction mixture, the tubes were vortexes and centrifuged at 2500 rpm for 10 minutes at room temperature. The absorbance of the blue color supernatant was recorded at 580 nm.

Measurement of serum LSA:

LSA was measured according to the method described by Katopodis and co-workers[12,13]. Fifty-µl serum was placed in screw-capped tubes, 3ml of cold (4°C) chloroform/methanol (2:1;v/v) mixture was added to each tube for total lipid extraction, the tube were capped and vortexed for 30 seconds, 0.5 ml cold water was added to each tube and the tubes were centrifuged for 5 minutes at 2500 rpm at room temperature.

The upper phase (aqueous layer containing LSA) was transferred to another screw-capped tube. Fifty µl of phosphotungstic acid (1g/ml) was added to each tube .The tubes were vortexed and allowed to sit at room temperature for 5 minutes. The tubes were then centrifuged at room temperature for 5 minutes at 2500 rpm. After that the supernatants were decanted and the remaining pellets were redissolved in 1ml at 37°C water by vigorously vortexing for 1 minute and sialic acid content was determined as mentioned for TSA.

Statistical analysis:

TSA and LSA sensitivity was calculated as the percentage of patients having values above cut-off level 2SD (standard deviation). Student's t-test was used to compare the levels of serum mean values in patients with normal.

Results

The aim of the present investigation is to find some typical role of TSA and LSA as a diagnostic pointer in inflammatory active disease to enable us for clinically meaningful difference with respect to serum TSA and LSA levels by comparing its levels

between normal subjects and different categories of patients with RA and related to degree of inflammation.

Normal subjects:

Comparison of the distributions of the serum TSA and LSA in both sexes, Table (3); shows that the mean serum values for TSA in female and male was (63.61±10.69) and (71.52±5.62) mg/dl respectively, while the mean serum value for LSA was (20.50±3.68) and (23.90±4.26) mg/dl for female and male respectively. The results show little differences between females and males for both TSA and LSA, so that the total means for both sexes was (67.57±7.56) and (22.20±3.29) mg/dl respectively.

Table 3: Serum levels of TSA and LSA in normal

Sex	TSA mg/dl (Mean±SD)	LSA mg/dl (Mean±SD)
Female (n=25) **Male (n=18)**	63.31±10.69 71.52±8.82	20.50±5.68 23.90±4.26
Total (n=43)	67.57±7.56	22.20±3.29

Rheumatoid Arthritis (RA) patients:

TSA and LSA were analyzed with respect to degree of inflammation of the disease, so separate calculation was made for each test's levels of 43 normal and 54 patients with RA are shown in table 4. In the normal sera samples the overall TSA level was found to be (67.57±7.56) mg/dl, while the corresponding level in 54 patients sera samples was (88.25±12.96) mg/dl, this increase of 31% in TSA level was statistically significant (P<0.001).

Both in the normal and the various groups of patients with different degree of disease, there were significant difference between values obtained for females and males for each characteristics of the disease state, therefore they were grouped independent. The mean serum TSA levels was found to be (79.22±11.01) and (76.90±10.23) mg/dl for female and male with low activity; and (104.23±27.22) and (92.68±17.27) mg/dl for serum female and male with high active disease.

The magnitude of the increase in the value varied between 17% for female and 14% for male patients with low activity. In contrast, the magnitude of the increase in serum TSA level varied between 54% and 37% for female and male with active disease respectively.

Table 4: Serum TSA values of patients with RA patients.

Cases	mg/dl (Mean±SD)	Change in TSA level (%)
RA with Low activity **Female (n=21)** **Male (n=8)**	 79.22±11.01 76.90±10.23	 +17 +14
RA with High activity **Female (n=16)** **Male (n=9.0)**	104.23±27.22 92.68±17.26	+54 +37
Total (n=54)	88.25±12.96	+31
Normal (n=43)	67.57±7..56	-

The sensitivity of TSA observed in our study fell from 38% (8 of 21) to 25% (2 of 8) for female and male with low activity disease, while the values reached 75% (12 of 16) to 56% (5 of 9) for female and male with high activity disease figure 1, this increase of sensitivity might contribute to the development of RA and to the progression of the disease itself.

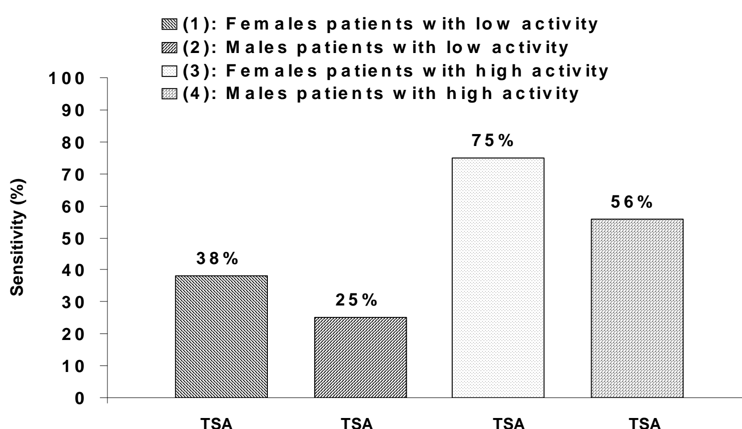

Figure 1: Sensitivity of TSA in patients with (RA)

Striking differences were found in the levels of serum LSA (Table 5). The mean LSA level in 43 normal was found to be (22.20±3.29) mg/dl, while the mean LSA in overall sera patients with RA was (30.92±4.11) mg/dl, this 39% increase in the LSA level was statistically significant (P<0.001).

The results show significant change between values obtained for both sexes, so the increase in the LSA serum level was more pronounced change when the patients were grouped according to the high activity of the disease and the extent of the increase varied between 62% for female and 45% for male with active disease.

Table 5: Serum LSA values of patients with RA patients

Cases	mg/dl (Mean±SD)	Change in LSA level (%)
RA with low activity		
Female (n=21)	28.69±5.38	+29
Male (n=8)	26.71±3.41	+20
RA with high activity		
Female (n=16)	36.05±8.09	+62
Male (n=9)	32.26±6.95	+45
Total (n=54)	30.92±4.11	+39
Normal (n=43)	22.20±3.69	-

Figure 2 shows the distribution and the sensitivity of LSA in the serum normal and in the RA patients according to the activation and severity of the disease. The mean serum LSA level in the total groups of RA was significantly elevated than normal value and the sensitivity fell from 43% (9 of 21) to 37% (3 of 8) for female and male with low activity and 56% (9 of 16) to 44% (4 of 9) for female and male patients with high activity disease.

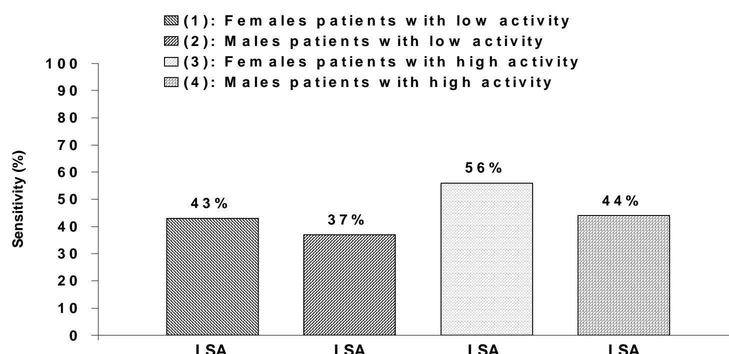

Figure 2: Sensitivity of LSA in patients with (RA)

Discussion

The present study demonstrates that the mean serum TSA and LSA levels in patients with RA was significantly increased compared to normal, this results confirm those of studies that observed high plasma or serum TSA and LSA in patients with cardiovascular disease, inflammatory reaction and different types of cancer[14-17].

TSA includes a small amount of free sialic acid as well as glycoprotein and glycolipid-bound sialic acid, these oligosaccharide chains, with sialic acid on the N-terminal position, are present on the cell surface.Sialic acid induces an electronegative charge[12] because, as a relatively strong acid (pKa=2.6), it is completely ionized at physioiogical pH[18].

This ionization plays a major role because the distribution of cell surface dense anionic sites is correlated, in vitro, with abnormal cell aggregation[19]. The mechanism of increased serum SA level in inflammatory conditions is unclear, but several explanations have been proposed these include: (a) spontaneous release of aberrant SA-containing cell surface glycoconjugates, (b) increased levels and /or glycosylation of normal serum glycoproteins[16].

One of hypothesis of increase of plasma or serum TSA levels in patients with inflammatory state could be explained by an increased output of serum proteins from the liver due to some type of acute phase protein. This also requires an increase in the activity of sialidase, which will catalyze and remove the sialic residues from an acute proteins[20].

References

1. Bhavanandan VP, and Sheykhazari M: Adaptation of the periodate-resorcinol method for determination of sialic acids to a microassay using microtiter plate reader. Anal Biochem, 1993; 213: 438-40.
2. Warren L: The distribution of sialic acids within the eukaryotic cell. In: Biological Roles of Sialic Acid (Rosenberg A, Schengrund C, Eds), plenum Press, New York 1976; pp103-121.
3. Jeanloz RW, and Codington JF. The biological role of sialic acid at the surface of the cell. In: Biological Roles of Sialic Acid (Rosenberg A, Schengrund C, Eds), Plenum Press, New York, 1976; pp201-238.
4. Varki A: Sialic acids as ligands in recognition phenomena. FASEB J, 1997; 11: 248-55.
5. Lopez-Saez JJB, and Senra-Varela A: Evaluation of lipid-bound sialic acid as a tumor marker. J Biol Markers, 1995; 10: 174-9.
6. Taysis, Akcay F, Uslu C, Dogru Y, and Gulcin I: Trace elements and some extracellular antioxidant proteins levels in serum of patients with laryngeal cancer. Biol Trace Elem Res, 2003; 91: 11-8.
7. Frang-Kirscher SG: Comparision of sialic acids axcretion in spot urines and 24-hour-urines of children and adults. Eur J Clin Chem, 1997; 35: 47-52.
8. Shauer R: Sialic acids and their role as biological markers. Trends Biochem Sci, 1985; 10: 357.
9. Haq M, Hag S and Tutt P: Serum total sialic acid and lipid associated sialic acid in normal subjects and those having a myocardial infraction and their relation to acute phase proteins. Ann Clin Biochem, 1993; 30: 383-6.
10. Tentative EULAR criteria for disease activity measurement of rheumatoid arthritis (EULUR Core Set). Rheumatol Eur, 1994; 23: 37-9.
11. Svennerholm L: Quantitative estimation of sialic acids. A colorimetric resorcinol-hydrochloric acid method. Biochem Biophys Acta, 1957; 24: 604-11.
12. Katopodis N, Hirshaut Y, and Geller NL: Lipid-associated sialic acid test for the detection of human cancer. Cancer Res, 1982; 42: 5270-5.
13. Katopodis N, and Stock CC: Improved method to determine lipid-bound sialic acid in plasma or serum. Res Commun Chem Path Pharmacol, 1980; 30: 171-80.
14. Crook M, Lump P, Andrews V, and Swaminathan R: Serum total sialic acid, a reputed cardiovascular risk factor, and its relationship to lipids, plasma fasting insulin, blood pressure and body mass index in normal individuals. Clin Sci, 1999; 95: 535-42.
15. Rogers M, Williams D, and Niththananthan R: Decrease in erythrocyte glycophorin sialic acid content is associatet with increased erythrocyte aggregation in human diabetes. Clin Sci, 1992; 82: 309-13.
16. Stefenelli N, Klotz H, Engel A, and Bauer P: Serum sialic acid in malignant tumors, bacterial infections and chronic liver disease. J Cancer Res Clin Oncol, 1985; 109: 55-9.
17. Pervin V, Mukaddes C, and Dilek SL: Total and lipid-bound sialic acid levels in actinin keeatpsis and basal cell carcinoma. J Med Sci, 1999; 29: 419-23.
18. Jaques LW, Brown EB, Barrett JM, Brey WS, and Weltner WJ: Sialic acid: A calcium binding carbohydrate. J Biol Chem, 1977; 252: 4533-8.

19. Raz A, Bucana C, Mclellan W, and Fidler IJ: Distribution of membrane anionic sites on B16 melanoma variants with differing lugs colonsing potential. Nature, 1980; 284: 363-4.
20. Wakabayashi I, Sankamoto K, and Yoshimato S: Relation of serum sialic acid to lipid concentration. BMJ, 1992; 305: 562-3.

THE ROLE OF CERVICAL SCREENING IN EARLY DETECTION OF CERVICAL LESIONS

Maysaloun M. Al-Ani[1] *MBChB, FICMC(Com. Med.)*, **Abdul-Hussain Al-Hadi**[1], *MBChB, PhD*, **Faiza A. Al-Rawi**[2] *MBChB, FICMC (Pathology)*

Abstract

Background: Papanicolaou (pap) smear is the most effective cancer prevention screening tool ever devised.

Objective: To study the prevalence of cervical inflammatory, premalignant and malignant lesions and to identify factors related to the prevalence of those lesions.

Methods: A cross sectional study was conducted over 3 months period on 302 women attending a Gynecology and Obstetrics outpatient clinic. A detailed history was recruited and pap smear was performed after taking patient consent.

Results: Among the total of 300 satisfactory pap smears, pathological changes were found in 209 (67.7%) while the rest 91 (30.3%) did not show any abnormality. More than one type of pathology were found in some cervical smears. The prevalence of different pathological changes revealed by cytological diagnosis of 302 pap smear were: Non-specific cervicitis 188(62.7%), Squamous metaplasia 23 (7.7%), Moniliasis 11 (3.7%), Squamous dysplasia (mild and moderate) 7 (2.3%), trichomouns vaginalis 4(1.3%), HPV infection 4(1.3%), IUCD changes 4(1.3%) and 2 smears (0.7%) were unsatisfactory and excluded from the study.

Conclusions: The prevalence of pre-carcerous and carcerous cervical lesions are low and comparable to other studies. Low educational level and improper genital and sexual hygienic practices are associated with inflammatory smears, and they are common findings among women with pre-carcerous lesions. History of prior genital infection, using contraception, abortion and curettage are associated with cervicitis.

Keywords: pap smear, cervical cancer, cervicitis

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Introduction

Cervical cancer is the second or third most common neoplastic disease affecting women[1]. In Iraq cervical cancer ranks the tenth within the leading cancers for females[2]. The Pap test is a screening tool that identifies women likely to have pre-malignant disease and high risk for cervical cancer[3].

Cervical Intraepithelial neoplasia (CIN) and cervical cancer remain important health problems for women worldwide, with high morbidity and mortality[4,5]. Risk factors for cervical cancer are: multiple sexual partners, male partners with multiple sexual partners, early age of first intercourse, male sex partner who has had a partner with cervical cancer, current or prior HPV infection or condyloma, history of herpes simplex infection, HIV infection, history of STD, immuno-suppression (*e.g.* renal transplant), smokers of abusers of other substances including alcohol, history of lower genital tract dysplasia or cancer, and low socioeconomic status[6-8]. This study aims at studying the prevalence and factors associated with various cervical lesions (inflammatory, pre-malignant, and malignant).

Patients and methods

A cross sectional study was carried out at the gynecology and obstetric outpatient

[1]Dept. Community Medicine, [2]Dept. Pathology, College of Medicine, Al-Nahrain University.
Address correspondence to: Dr: Maysaloun M. Al-Ani, e-mail: mayshams92@hotmail.com
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clinic of Al-Kadhimiya teaching hospital during the period between the first of July to the end of September 2000. A convenient method of sampling was used sample was obtained from women attending the outpatient clinic of Gynecology and Obstetric at Al-Khadhimiya teaching hospital.

A pre-coded, pre-tested questionnaire form was administered to each woman and a gynecological examination was performed after taking patient consent. Cervical smear was done to each woman. Inclusion criteria was married woman (divorced and widows are included also) while the exclusion criteria were pregnancy, abortion, and vaginal bleeding of any type.

Data obtained from the study sample included information on demographic and socioeconomic status like age in years, age at marriage and edcational level, hygienic practices like using sanitary pads,method of underwear drying and washing after intercourse, and other data related to reproductive, obstetrical and gynecological history such as aage at menarche, age at first livebirth, gravidity, parity, prior lower genitaltract infection for the last year and curettage, and contraception.

Each woman was examined gynaecologicaly, then cervical smear was taken. The prevalence of Different pathological changes detected by pap smear were studied in relation to different variables.. Statistical analysis were done using SPSS version 7.5 computer software (statistical package for social sciences) in association with EPI.6.2 computer software.

Results

Of the total of 302 women to whom pap smear was performed, 99.3% (300 women) had satisfactory smears and only 2 (0.7%) were inadequate. Pathological changes were found in 209(69.7%) women while 91(30.3%) had normal cervical smears. It was possible to find more than on cytological changes in one smear, that,s why addition of the number of different pathological changes in some tables of this study are higher than 209. Results showed that 62.7% of women had non-specific cervicitis.

The prevalence of pathological changes evident by pap smear and its types were; Non-specific cervicitis 188(62.7%), Squamous metaplasia 23 (7.7%), Moniliasis 11 (3.7%), Squamous dysplasia (mild and moderate) 7 (2.3%), trichomouns vaginalis 4(1.3%), HPV infection 4(1.3%), and IUCD changes 4(1.3%) (Table 1).

Table 1: The prevalence 95% CI (%) of pathological changes evident by pap smear

Type of pathological changes		No.	%
Non specific cervicitis		188	62.7
Squamous metaplasia		23	7.7
Moniliasis		11	3.7
Squamous dysplasia	Mild	6	2
	Moderate	1	0.3
	Severe	0	0
Trichomonus vaginalis		4	1.3
HPV		4	1.3
IUCD changes		4	1.3
All pathology*		209	69.7
No pathology		91	30.3
Total		300	100

*More than one type of pathology is possible

Table 2 shows that the prevalence of non-specific cervicitis was more among women below 30 years of age (69.5), while all cases of HPV and squamous dysplasia were among those below 40 years of age. The mean age ±SD of women with non-specific cervicitis was 34.6±9.4 , mean age at marriage and age at first live birth was 18.4± 4.4 and 19.9± 4.3 respectively , the latter two were found to be significantly lower than those without cervicitis (P<0.05) by using t-test.

Table 2: The prevalence of different pathological changes shown by pap smear by age of women

Type of pathological* changes	Age of women (years)						
	<30		30-39		40+		Total
	No. (n=95)	%	No. (n=103)	%	No. (n=102)	%	No.
Non specific cervicitis	66	69.5	62	60.2	60	58.8	188
Squamous metaplasia	7	7.4	8	7.8	8	7.8	23
Moniliasis	3	3.2	5	4.9	3	2.9	11
Squamous dysplasia	2	2.1	5	4.9	0	0	7
Trichomonus vaginalis	0	0	2	1.9	2	2	4
HPV	1	1.1	3	2.9	0	0	4
IUCD changes	2	2.1	0	0	2	2	4

* More than one type of pathology is possible

Table 3 summarizes the studied factors that were significantly associated with cervicitis. The prevalence of non-specific cervicitis were found more among women with lower educational level (P<0.001). results also showed that more prevalence of cervicitis was among women not using sanitary pads 65.5%, women using indoor method of underwear drying 86.9%, and those who did not use to wash after intercourse 88.7% and the difference was found to be significant (P<0.001). It was also found that the prevalence of cervicitis was significantly higher among women with history of lower GTI 73.1% and those with history of curettage 80.4% (P<0.001).

The mean age±SD of women with HPV and dysplasia was 34± 1.6 and 32± 1.3, mean age at menarche 12± 1.2 and 13± 1.1, age at marriage 20± 1.4 and 21± 1.2, age at first live birth 22± 1.5 and 23± 1.4, gravidity 4± 0.8 and 3± 0.7,and parity 4± 0.7 and 3± 0.4 respectively.

Table 3: The prevalence of non-specific cervicitis by different Variables

Variable	Non specific cervicitis						X^2	df	$P(X^2)$
	Present		Absent		Total				
	No.	%	No.	%	No.	%			
Educational level									
Illiterate	51	67.1	25	32.9	76	100	23.8	5	<0.001*
Read & write	39	72.2	15	27.8	54	100			
Primary ed.	52	71.2	21	28.8	73	100			
Intermediate ed.	29	63.0	17	37.0	46	100			
Secondary ed.	10	34.5	19	65.5	29	100			
Higher ed.	7	31.8	15	68.2	22	100			

Table 3: ***Continued***

Using sanitary pads									
Yes	1	9.1	10	90.9	11	100	14.5	1	<0.001*
No	173	65.5	91	34.5	264	100			
Method of underwear drying									
Indoor	133	86.9	20	13.1	153	100	78.6	2	<0.001*
Outdoor	54	37.5	90	62.5	144	100			
Both	1	33.3	2	66.7	3	100			
Washing after intercourse									
Yes	77	45.0	94	55.0	171	100	56	1	<0.001*
No	102	88.7	13	11.3	115	100			
History of prior lower G.T.I.									
Yes	171	73.1	63	26.9	234	100	49.3	1	<0.001*
No	17	25.8	49	74.2	66	100			
History of curettage									
Yes	78	80.4	19	19.6	97	100	19.3	1	<0.001*
No	110	54.2	93	45.8	203	100			
Using contraception									
Yes	113	68.9	51	31.1	164	100	7	1	<0.001*
No	55	52.9	49	47.1	104	100			

* significant association

Table 4 shows that all women with HPV were housewives with history of prior lower GTI (during the last year) and all of them were not using sanitary pads. Half of those womwn had genital warts and used indoor method of underwear drying. One quarter of them were illiterate but none of them were using barrier method of contraception.

Table 4: The prevalence of some characteristics of women with HPV *

Characteristics	Prevalence % (no.)
Housewife	100 (4)
Illiterate	25 (1)
Not using sanitary pads	100(4)
Indoor underwear drying	50 (2)
Not washing after intercourse	25 (1)
Genital warts	50 (2)
Prior lower G.T.I.(for the last year)	100 (4)
Using Contraceptive pills	25 (1)
Using IUCD	25 (1)

***number of cases=4**

Table 5 shows that majority of women with cervical intraepithelial neoplasia (squamous dysplasia) were housewives 85.7% and had history of lower GTI 85.7%. All of them were not using sanitary pads but 71.4% were using indoor underwear drying and 28.6% did not use to wash after intercourse. However none of them were using barrier method of contraception although 40% used contraceptive pills.

Table 5: The prevalence of some characteristics of women with dysplasia (shown by pap smear)*

Characteristics	Prevalence %(no.)
Housewife	85.7 (6)
Illiterate	14.3 (1)
Not using sanitary pads	100 (7)
Indoor underwear drying	71.4 (5)
Not washing after intercourse	28.6 (2)
Genital warts	28.6 (2)
Prior lower G.T.I. (for the last year)	85.7 (6)
Using Contraceptive pills	40 (2)
Using IUCD	20 (1)

* number of cases=7

Discussion

The percentage of total different pathological changes revealed by pap smear was higher than in other study[9], this may be due to differences; in population's characteristics, smear taking and other technical factors. but regarding the type of pathological changes the prevalence of Trichomonus vaginalis infection was lower than in other studies[9,10] and The percentage of non-specific cervicitis was lower than in other studies too[11,12] and was more common among younger age groups[11] which may be related to socioeconomic, reproductive and behavioral differences.

On the other hand the percentage of HPV infection was in agreement with that of Hassan[9]. Squamous dysplasia was mainly prevalent in cervical smears of women aged from 30-39 years which was in agreement with other studies[13,14]. It has been found that inflammatory changes (cervicitis) are important in the pathogenesis of pre-malignant lesions[15,16] and they are related to sexually transmitted infections[17] and in 40-47% of them, microbiological tests proved to have specific infections[5,18,19].

The mean age at marriage and at first live birth among women with non-specific cervicitis were significantly lower than those without such pathology which was in agreement with other studies[10,20], these changes were more common among women with lower educational level because they were adopting unhygienic genital and sexual practices[11] and the percentage of those lesions was significantly lower among women using proper genital and sexual hygienic practices. The percentage of non-specific cervicitis was significantly higher among women who had history of previous lower genital tract infection, history of abortion and curettage which was in agreement with other study[10].

The mean age of women with HPV in this study were approximately similar to that found by other study in Iraq[8], but higher than in other European studies[21], this is probably due to early sexual practicing habits and multiple partners leading to a higher chance of harboring the virus in European women.

The prevalence of HPV findings on pap smear was within the range found by other studies which lies between <1% and 3%. The percentage of Squamous dysplastic changes (CIN) that were found in cervical smears of women with HPV infection was similar to that reported by other studies[8]. Women who had dysplastic epithelial changes in cervical smear were below 39 years old which was in agreement with other studies[13,14] while other studies in United States reported the peak age of first coitus with multiple sexual partners which may be surrogate markers of sexually transmitted infectious pathogens involved in cervical neoplasia.

The mean gravidity and parity of women with CIN were similar to that in other studies[10,13]. Generally those women with

HPV and/or CIN had poor sexual and genital hygienic practices, and none of them were using barrier method of contraception, these factors might be responsible for increasing the possibility of infection, cervical neoplasia and cancer.

Conclusions

The prevalence of pre-carcerous and carcerous cervical lesions are low and comparable to other studies. Low educational level and improper genital and sexual hygienic practices are associated with inflammatory smears, and they are common findings among women with pre-carcerous lesions. History of prior genital infection, abortion and curettage are associated with cervicitis.

In view of the results obtained from this study, the recommendations suggested were; establishment of cervical clinics at all maternity hospitals and at general hospitals with maternity departments, these clinics would enhance the establishment of screening programs for cervical lesions so that women are encouraged to participate and to have cervical smears annually or biannually, and health education programs emphasizing on the importance of papanicolaou screening test in prevention and early diagnosis of cervical cancer and the need for genital and sexual hygiene and proper treatment of genital infection.

These programs should be directed also at men since genital tract infections are sexually transmitted; adoption of the Bethesda System to unify the terminologies in the reporting of papanicolaou smears by different pathologists and laboratories; and inclusion in intermediate and secondary school curricula of specific messages on reproductive and sexual health with special emphasis on the risk factors of cervical pre-cancerous and cancerous lesions.

References

1. Franco ELF: Epidemiology of anogenital warts and cancer. Obst Gyne Clin North Am, 1996; 23(3): 597-623.
2. IRAQI cancer registry, Iraqi MOH, 1999.
3. Borisove I, Shopova E, and Mainkhard K: The etiology of infectious cervicitis in women. Akush Ginekol Sofiia, 1999; 38(2): 23-5.
4. Clement KD, and Connor PD. Tumors of the female reproductive organs. Family medicine principles and practice. 5th ed. Volume 2. New York Springer-Verlag, 1997.
5. Parsons WL, Godwin M, Robbins C, Butler R: Prevalence of cervical pathogens in women with and without inflammatory changes on smear testing. BMJ, 1993; 306(6886): 1173-4.
6. Women's Cancer Information Center WCC Research. Cervical cancer prevention, 2000.
7. Hart DM, and Norman J: Gyncology illustrated. 5th ed. Edinburgh Churchill living stone, 2000.
8. Al-Saadi R: Sub-clinical human papilloma virus infection of the cervix uteri: A clinico-pathological study. FICMS thesis, Iraqi commission for medical specialization in dermatology and venereology, 1998.
9. Hassan IS. The role of cervical and vaginal cytology in the diagnosis of gynecological disease. MSc thesis, College of Medicine-University of Mosul, 1989.
10. Al-Alwan N. Pre-cancerous lesions of the uterine cervix. MSc thesis, College of Medicine, University of Baghdad, November, 1987.
11. Varghese C, Amma NS, Chitrathara K, *et al:* Risk factors for cervical dysplasia in Kerala, India. Bulletin of the World Health Organization, 1999; 77(3): 281-3.
12. Keshavars H, Duffy SW, Sotodeh Maram E, *et al:* Factors related to cervicitis in Qashghaee nomadic Women of southern Iran. Rev. Epidemiol Sante Publique, 1997; 45(4): 279-85.
13. Al-Badri T: Accuracy of cytology and colposcopy in the diagnosis of cervical squamous intraepithelial neoplasia. ICOG thesis, Iraqi commission for medical specialization in obstetrics and gynecology, 2000.
14. Parazzini F, Sideri M, Restelli S, *et al.*: Determinants of high grade dysplasia among women with mild dyskariosis on cervical smear. Obst Gynecol, 1995; 85(5): 754-6.
15. Parashari A, Singh V, Gupta MM, *et al.*: Significance of inflammatory cervical smears. APMIS, 1995; 103(4): 273-8.
16. Neuer A, and Menton M. Bacteriological findings in patients with cervical intraepithelial neoplasia. Zentralbl-Gynakol, 1995; 117(8): 435-8.

17. Molodysky E: Urethritis and cervicitis. Aust Fam Physician, 1999; 28(4): 333-8.
18. Kelly BA, and Black AS. The inflammatory cervical smear: A study in general practice. Br J Gen Pract, 1990; 40(335): 238-40.
19. Wilson JD, Robinson AJ, Kinghorn S, and Hicks DA: Implications of inflammatory changes on cervical pathology. BMJ, 1990; 300: 638-40.
20. Morris M, Totolero-Luna G, Malpica A, *et al*.: Cervical intraepithelial neoplasia and cervical cancer. Obst Gyne Clin North Am, 1996; 23(2): 347-410.
21. Jamison JH, Kaplan DW, Hamman R, *et al*.: Spectrum of human Papillomavirus infection in a female adolescent population. Sex Trans Dis, 1995; 22: 236-42.

TRACE ELEMENTS AND HEMATOLOGICAL CHANGES IN THALASSAEMIA MAJOR AND MINOR

Subh S. Al-Mudallal[1] *MSc FICMS*, **Saad S.M. Abadah**[1] *MBChB FRCPath*, **Y.Y. Z. Farid**[2] *BSc Ph.D.*

Abstract

Background: The Thalassaemias are hereditary hemolytic anaemia due to genetic defect in the DNA or in the messenger RNA. Many studies on B-thalassaemia (major and minor) have reported variation in the level of the trace elements zinc, Copper and Magnesium. These variations may play a role in cellular abnormalities, which characterize this disorder. Thus, these trace elements have been used as indices for following up these patients.

Objective: To assess the level of serum Cu, Zn and Mg in thalassaemic patients (major and minor) and to investigate different hematological parameters as well as serum ferritin in those patients.

Methods: 72 patients with B-thalassaemia major (Th. M.), 17 patients with B- thalassaemia minor (Th.Mn.) and 30 healthy age matched subjects who served as control. Trace elements, Hb, PCV, MCH and MCHC and serum ferritin were estimated.

Results: A significant decrease in Hb concentration and MCHC was found in Th.M. compared to control group whereas a significant increase in MCV, MCH and MCHC was found in Th.M. compared to Th.Mn. In addition, MCV, MCH and MCHC were significantly reduced in Th.Mn. compared to control, while PCV level was significantly higher when compared to Th.M. patients and control group.
Serum Cu and Zn in Th.M. were significantly increased when compared to control group while serum Mg in Th.M. was significantly reduced when compared to healthy control but not in Th.Mn. In Th.Mn., serum Cu was significantly increase when compared to control and to Th.M. patients.
Serum ferritin was significantly high in B-thalassaemia major and minor when compared to control. In addition, it was significantly high in Th. M. compared to Th.Mn. Furthermore, only serum Zn and Mg showed a significant positive correlation with PCV in B-Th.M. patients.

Conclusion: Serum Cu and ferritin were high in B-Th.M. and Th.Mn., serum Mg was low and serum Zn was high in B-Th.M. Since serum Zn and Mg were closely related to PCV, therefore the therapeutic value of Mg and Zn supplementation should be tested in those patients.

Keywords: Trace Elements, Thalassaemia Major and Minor, Hematological Changes

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Introduction

Copper (Cu), Zinc (Zn) and magnesium (Mg) are biological elements that are called as trace elements because small amount of them are found in human body[1], however they are essential metals that are required for growth and proliferation of healthy cells and for normal lymphocytes maturation and regulation of the immune function[2]. Moreover, changes in the level of these elements may impair cellular and physiological functions through changes in the activities of metalloenzymes which require a small and constant number of metal per mole to attain full activity of these enzymes[3].

[1]Dept. pathology, [2]Dept. Chemistry and Biochemistry, College of Medicine, Al-Nahrian University.
Address correspondence to Dr. Subh Al-Mudallal
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The thalassaemia syndromes are characterized by an inherited defect in the synthesis of one or more of the peptide chains of haemoglobin[4]. The biochemical changes especially of the trace elements such as Cu, Zn and Mg had been investigated. Many clinicians had noticed growth retardation[5-7], various retinal abnormalities[6,8] and different abnormalities in the RBC of thalassaeic patients[9], which were attributed to alteration in the level of these trace elements. Therefore, trace elements had been critically examined in the follow up and treatment of patients with B thalassaemia.

Aims of the study

1. Assess the level of Cu, Zn and Mg in the blood of thalassaemia major and minor and compare it with control level.
2. Evaluate the change in different haematological parameters including Hb, PCV, MCV, MCH, MCHC and serum ferritin in B thalassaemia major and minor and compare it with control values.

Materials and Methods

This study was conducted on 72 patients with B thalassaemia major and 17 patients with B thalassaemia minor who were attended Ibn Al-Balady hospital from 2002-2003 along with 30 healthy age matched control subjects table 1.

All those patients were previously diagnosed as having thalassaemia by clinical, family history, hematological and electrophoresis study.

Blood samples collected from the patients before blood transfusion, in an EDTA tube and in plain tube. Serum Cu, Zn and Mg were estimated by Atomic Absorption Spectrophotometer, Perkin Elmer, model 103, serum ferritin was estimated by Immunotech Kit, using Automatic Gamma Counter (Wallac-Wizard). Other haematological parameters, Hb, PCV, MCV, MCH and MCHC were estimated by multichannel analyser, Coulter S Plus.

All data were analyzed by unpaired student t-test and by linear regression test, taking $p<0.05$ as the lowest limit of significant.

Results

This study was performed on 72 patients with thalassaemia major, 17 patients with thalassaemia minor, and 30 healthy age matched control subjects, table 1.

Table 1: The demographic characters of the patients and control subjects enrolled in this study

Parameters	Study Groups		
	Thalassaemia major	**Thalassaemia minor**	**Control**
Number	72	17	30
Age (years)	4-29	20-33	4-30
Sex (female/male)	20/52	9/5	11/19

This study had revealed that in B-thalassaemia major, serum Cu was significantly increased compared to control individuals and to thalassaemia minor patients ($p<0.01$ and $p<0.05$) respectively (Table 2 and figure 1). In addition, serum Zn was significantly increased in relation to healthy control and to thalassaemia minor patients, ($p<0.05$) {table 2 and figure 1}, while serum Mg in thalassaemia major patients was significantly decreased in relation to healthy control ($p<0.05$) but its relation with thalassaemia minor was insignificant (Table 2 and figure 1).

In thalassaemia minor although serum Cu was significantly high when compared to healthy control but both serum Zn and Mg were not significantly changed when compared to control group (Table 2 and figure 1).

Table 2: Levels of serum Cu, Zn, Mg and ferritin in thalassaemia major, minor and control group

Parameters	Thalassaemia major n=72	Thalassaemia minor n=14	Control n=16
Cu (nmol/L)	30.094±6.319**†	24.58±10.95*	21.38±6.125
Zn (nmol/L)	1.14±0.198*†	0.899±0.334	0.858±0.240
Mg (mg/dl)	2.111±0.427*	2.145±0.623	2.428±0.684
S. ferritin µg/L (mean±SE)	1359.5±147.03****†††	83.0±30.55**	44.0±18.34

* All data are expressed as mean±SD except S. ferritin is expressed as mean±SE.
* $p<0.05$ correlation with control. ** $p<0.001$ correlation with control. **** $p<0.0001$ correlation with control.
† $p<0.05$ correlation with thalassaemia minor. ††† $p<0.001$ correlation with thalassaemia minor.

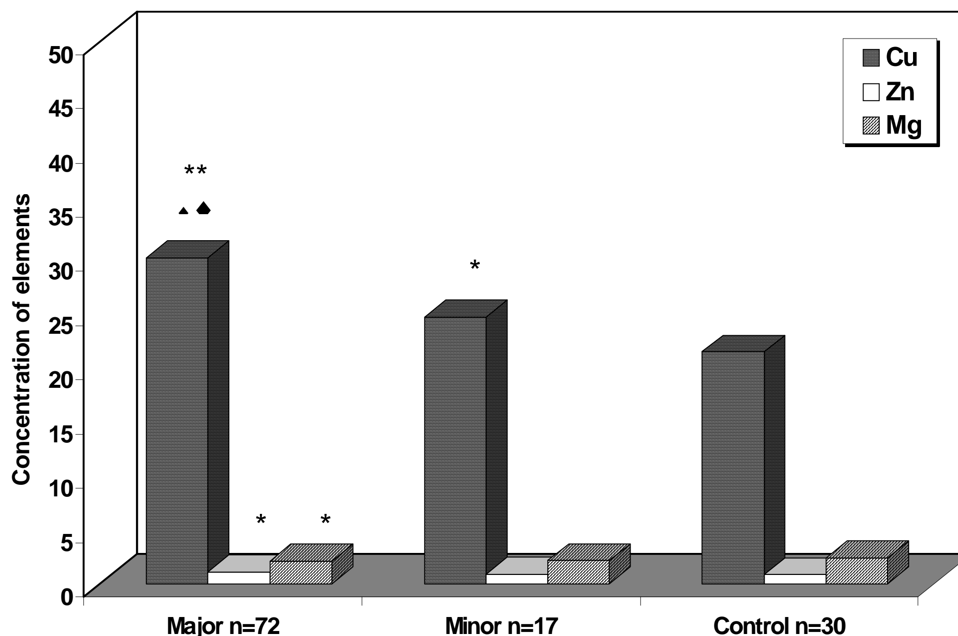

Figure 1: The concentration of serum Cu, Zn and Mg in thalassaemia major, minor and control.

* $p<0.05$ correspondent to control. ** $p<0.001$ correspondent to control.
♦♦ $p<0.01$ correspondent to minor.

The level of serum ferritin was significantly increased in thalassaemia major in relation to its level in thalassaemia minor and control ($p<0.001$, $p<0.00001$). Similarly it was significantly increased in thalassaemia minor in relation to its level in control ($p<0.001$) {Table 2 and figure 2}.

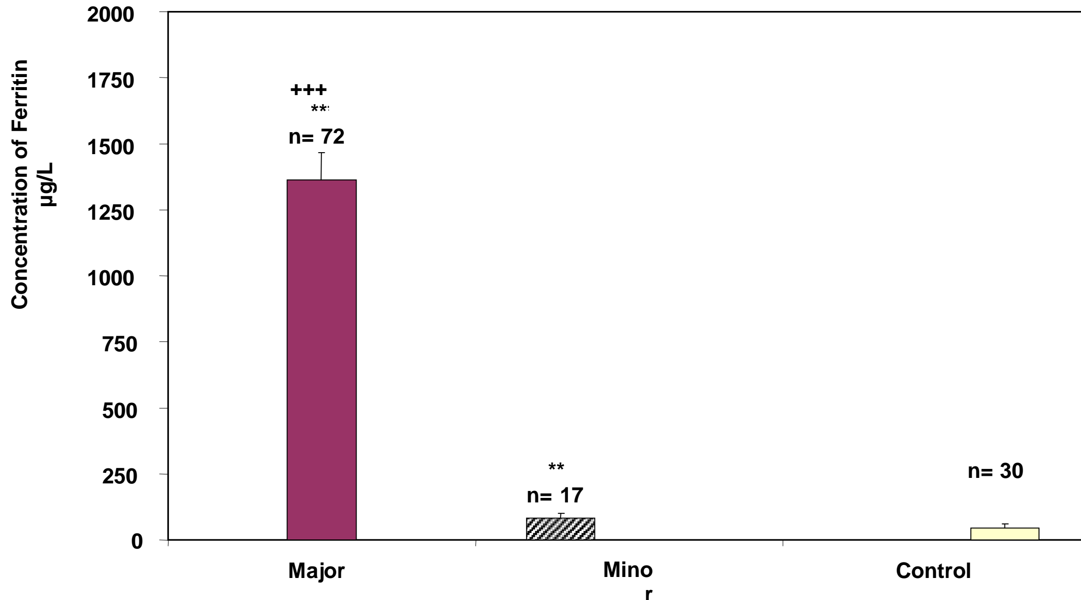

Figure 2: The serum ferritin concentration in thalassaemia major, minor and control.

*** p<0.001 correspondent to control. ** p<0.01 correspondent to control.
+++ p<0.001 correspondent to minor.

This study had revealed a positive correlation between serum Mg and the haemoglobin level in B- Thalassaemia major patients (r=0.457) (Figure 3). Moreover, there was a positive correlation between serum Zn, haemoglobin and PCV (r= 0.278, r=0.306) respectively (figures 4 and 5).

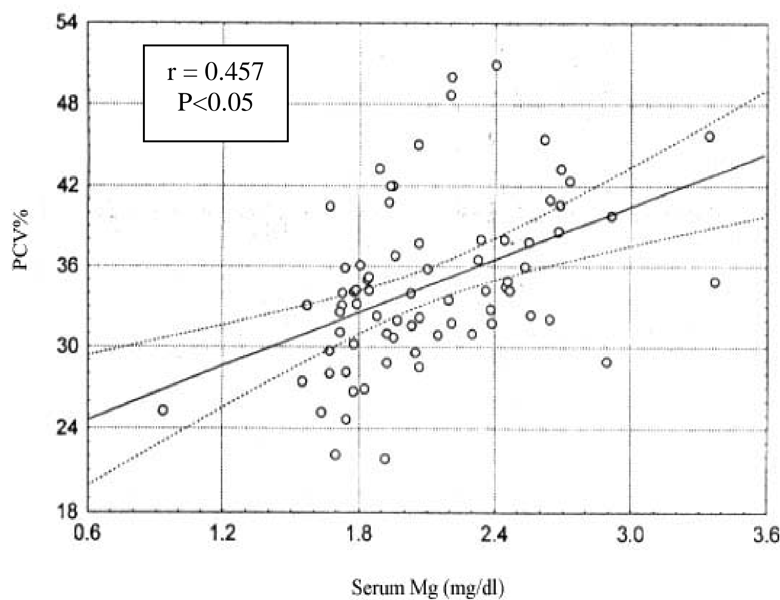

Figure 3: Positive correlation between Serum Mg and PCV in B thalassaemia major patients

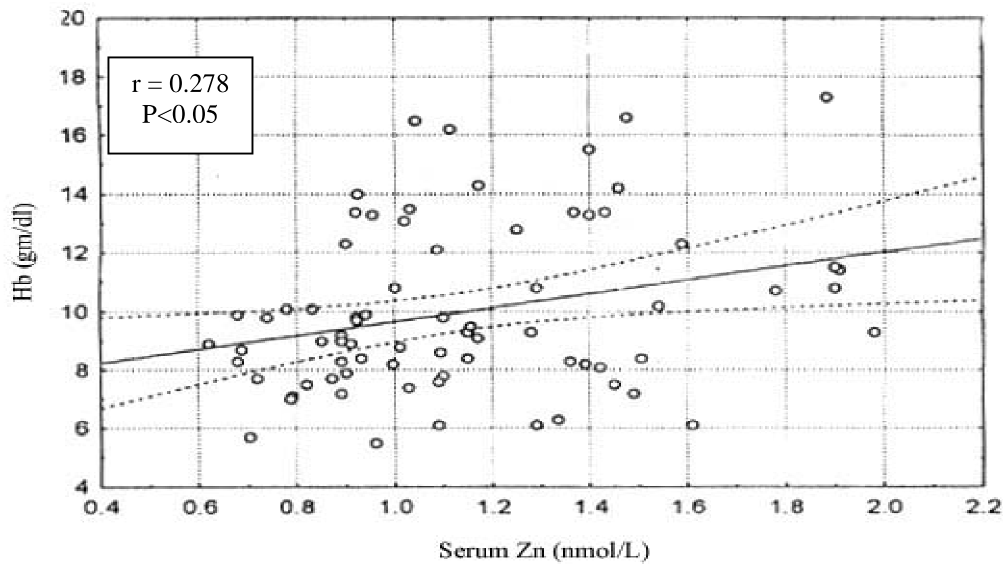

Figure 4: Positive correlation between Serum Zn and Hb level in B thalassaemia major patients

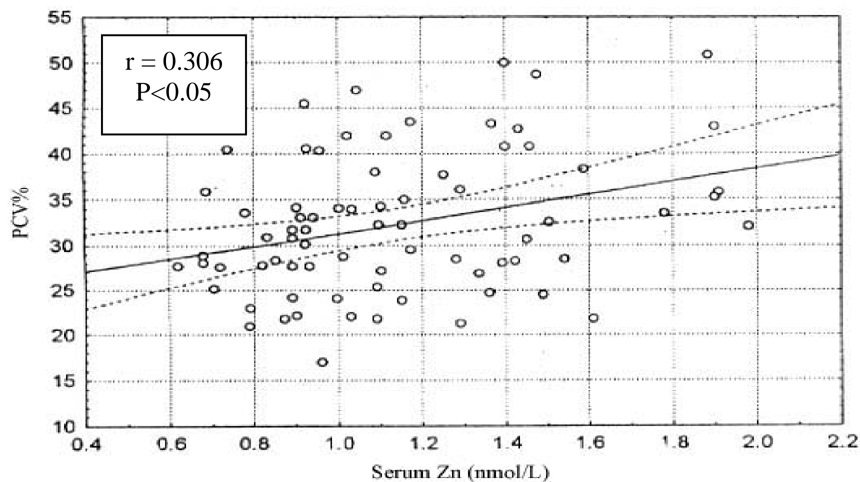

Figure 5: Positive correlation between Serum Zn and PCV level in B thalassaemia major patients

Furthermore the mean haemoglobin level and the MCHC were significantly lower and the MCV was significantly higher in thalassaemia major than in control ($p<0.05$ and $p<0.01$) respectively, while the MCV, MCH and MCHC were significantly higher in thalassaemia major compared to thalassaemia minor (p<p<0.001, $p<0.01$, $p<0.05$) respectively (Table 3).

In thalassaemia minor the mean MCV, MCH and MCHC were significantly lower than a control mean level ($p<0.001$, $p<0.0001$ and $p<0.001$) respectively while the PCV was significantly higher compared to both thalassaemia major and control group ($p<0.05$) {Table 3}.

Table 3: The level of Hb, PCV, MCV, MCH and MCHC in thalassaemia major, minor and control group

Parameters	Study group (mean±SD)		
	Thalassaemia major n=72	**Thalassaemia minor n=17**	**Control n=30**
Hb concentration (gm/dl)	10.8±3.0*	11.1±1.18	11.5±1.79
PCV(%)	34.6±8.6⁺	36.4±7.10♦	34.3±6.02
MCV(fl)	83.0±6.35**+++	64.9±1.76♦♦	76.3±16.17
MCH(pg)	26.9±3.67++*	18.8±1.57♦♦♦	28.5±1.57
MCHC(gm/dl)	31.2±3.78*+	29.0±2.53♦♦	35.5±2.10

* p<0.05 correspondent to control. ** p<0.01 correspondent to control. ♦ p<0.05 correspondent to control. ♦♦ p<0.01 correspondent to control. ♦♦♦ p<0.001 correspondent to control. + p<0.05 correspondent to minor. ++ p<0.01 correspondent to minor. +++ p<0.001 correspondent to minor.

Discussion

Trace elements are increasingly playing a diagnostic, curative and preventive role in many diseases[10].

Iron overload is one of the major complication in B- thalassaemia which due partly to the ineffective erythropoiesis and the constant hemolysis from early infancy and partly to the frequent blood transfusion required for the treatment of the severe anaemia[11].

Our study in agreement with other studies found that serum Cu and iron were significantly increased in thalassaemia patients[12-14]. The close relation between serum Cu and iron can be explained by the importance of copper containing enzymes and co-factors for iron absorption and the effect of copper on the release of iron from the body stores as well as the utilization of iron in haemoglobin synthesis[15,16]. This will explain the hypercupraemia usually encountered in haemochromatosis which is a principle complication in thalassaemia[17,18].

In thalassaemia minor serum ferritin and serum Cu level was significantly lower than in thalassaemia major, table 2, figure 1 and 2, which is expected since those patients are non transfusion dependant and have less extend of ineffective erythropoiesis[11]. Therefore, we expect the increase in serum Cu will be much less than thalassaemia major.

Bashir NA et al[13] study was similar to our study revealed a significant increase in serum Zn and Cu in thalassaemia major patients, and he had contributed it to impairment in kidney function and to disturbances in the metabolism of Zn and Cu which usually encountered in those patients[13].

Moreover since Zn concentration in the RBC is approximately 12 time that found in the plasma[12] and during transfusion of blood a considerable proportion of transfused cells will be destroyed[11], therefore we may propose that these patients who are transfusion dependant will have higher serum zinc comparing to control subjects and to thalassaemia minor patients who are not transfusion dependant.

It is well known that liver function as major storage organ for zinc[19] and since iron overload in the liver may generate oxygen free radical and may induce peroxidative

tissue[20], therefore we may expect an increase in serum zinc from damaged hepatocytes[19].

Pakarek et al hypothesized that variation of serum Zn level may be due to leukocyte endogenous mediator (LEM) which has the property of mobilizing zinc from its stores in liver and other tissue to the serum[21].

The present study found significant low serum Mg in thalassaemia major patients which is similar to other studies[15,16,22]. Magnesium is the second most abundant intracellular metal after potassium and it plays an essential role in the activity of many enzymes involved in cellular metabolism[23]. De-Franceschi L. et al[22] had speculated that the hypomagnesemia in B-thalassaemia may be due to chelation by citrate in chronically transfused patients or could just be a consequence of cellular iron overload[22,23].

Thalassaemia major patients were found to have significant anaemia compared to control, table 2, which was expected since those patients suffer from chronic hemolytic anaemia which is due to ineffective erythropoiesis and the hemolysis of RBC[11,18].

Generally, thalassaemia minor is a milder disease; therefore, the reduction in haemoglobin did not reach the level of significant. On the other hand, PCV was significantly increased in thalassaemia minor because of the increased RBC count usually encountered in thalassaemia minor patients[11,18].

In agreement with previous studies, the MCV, MCH and MCHC were significantly reduced in thalassaemia minor[11,18] who were transfusion independent. While in thalassaemia major, they were significantly increased because all the thalassaemia major patients were transfusion dependant and the transfused RBCs will affect these haematological parameters. Also the increase in MCV may be contributed to folic acid deficiency usually encountered in B- thalassaemia patients as a result of increased cell turnover in the bone marrow[11,17,18].

Additionally, this study had revealed a positive correlation between serum Mg and PCV (r=0.457) in B- thalassaemia major patients (Figure 3), which was in agreement with a study of De-Franceschi L et al[22], who had found that supplement of Mg may improve anaemia and reduce dehydration in B- thalassaemia major rats which was attributed to the effect of Mg on membrane stability by decreasing the activity of the K-Cl contrasport system, thus decrease the abnormal erythrocyte K loss and increase the life span of the RBCs[22].

Many studies had emphasized on the importance of Zn supplementation in the treatment of iron deficiency anaemia particularly in pregnant female[25] and in children[26]. This study revealed a positive correlation between serum Zn, haemoglobin and PCV (r=0.278, r= 0.306 respectively) in B- thalassaemia major patients (figure 4 and figure 5). These results were in agreement with many other studies who found that zinc supplementation in B- thalassaemia major had a positive effect on linear growth and anaemia[25,27].

References

1. Leimore IA, and Earl EM: Basic and dental Sciences. 2nd Ed. Mosby Company. St. Louis Washington, D.C. Toronto,1988; p.p. 255-64.
2. Carpentieri U, Myers J, Thorpe I, Daeshnner CW, and Haggard ME: Zinc and iron in normal and leukemic lymphocytes from children. Cancer Res, 1986; 46: 981-4.

3. Vyas RK, Gupta AP, and Aeron AK: Serum copper, zinc, magnesium and calcium levels in various human diseases. Indian J Med Res, 1982; 76: 301-4.
4. Weatherall DJ, and Clegg JB: The thalassaemia syndromes. 2nd ed. Chap 2, London. Blackwell Scientific Publications, 1972; p.p. 19-48.
5. Arcasay A, and Cavdar AO: Zinc levels of plasma erythrocytes, hair and urine in homozygous Beta-thalassaemia. Acta Haemat, 1979; 62: 41-4.
6. De virgilis S, Congia M, Turco MP, Fran F, Argiolu F, Sorcinelli R, et al.: Depletion by desferrioxamine in patients with thalassaemia. Arch Dis Child, 1988; 83: 250-5.
7. Schiliro G, Russo A, and Azzia N: Leukocyte alkaline phosphatase (LAP), a useful marker of zinc in B- thalassaemia patients. Am J Pedi Haematol / Oncolo, 1987; 9 (2): 149-52.
8. Oliveri NF, Buneie JR, and Chew E: Visual and Auditory neurotoxicity in patients receiving subcutaneous desferoxamine infusions. N Engl J Med, 1986; 314: 869-73.
9. Kajanachimpol S, Tatu T, Sasanakul W, Chuansumrit A, and Hathirat P: Zinc and copper status in thalassaemic children. Southeast Asian J Trop Med Public Health, 1997; 28 (4): 877-80.
10. Abora RB, Hameed A, Subramanian TAV, et al: Differential serum zinc concentration in some cardiovasccular diseases and bronchial asthma. In: Abdulla M, Dashti H, Sarkar B, Al-Sayer H, Al-Naqeeb N. Metabolism of minerals and Trace Elements in Human Diseases. 1st edition. New Delhi, India. Smith-Gordon and Company Limited, 1989; p.p. 57-60.
11. Wainscoat JS: The haemoglobinopathies. In: Hoffbrand AV and Lewis M. Postgraduate Haematology. 4th edition. Great Britain, Oxford. Heinemann Medical Books, 1999.
12. Arcasoy A, and Cardar AO: Changes of trace minerals (serum Iron, Zinc, Copper and Magnesium) in thalassaemia. Acta Haemat, 1975; 53: 341-6.
13. Bashir NA: Serum zinc and copper levels in sickle cell anaemia and beta thalassaemia in North Jordan. Ann Trop Paediatr, 1995; 15 (4): 291-3.
14. Hamid MO, and Pradhan PS: Study of trace elements in splenectomized beta thalassaemia major children. Indian J Haematol blood trans, 1999; 17 (4): 92-4.
15. Erlandson ME, Golubow J, and Smith CH: Bivalent cations in homozygous thalassaemia. J Pediat, 1965; 66: 637.
16. Erlandson ME, Golubow J, Wehran T, and Smith CH: Metabolism of iron, calcium and magnesium in homozygous thalassaemia. Ann NY Acad Sci, 1964; 119: 769.
17. William WJ, Beutler E, Erslev AJ, and Rundles RW: ed. New York. McGraw Hill 1972: 360.
18. Dispenzieri A: The thalassaemia syndromes and the haemoglobinopathies. In: Tefferi A. Primary Haematology. Totowa, New Jersey. Humana Press, 2001; p.p. 41-64.
19. Al-Sayer H, Dashti H, and Christenson J, et al: The role of superoxide dismutase and allupurinal in the prevention of CCl4 induced liver cirrhosis. In: Abdulla M., Dashti H. Sarkar B., Al-Sayer H. and Al-Naqeeb N. Metabolism of minerals and trace elements in human disease. 1st ed. New Delhi, India Smith-Gordon and Company Limited, 1989; p.p. 65-85.
20. Tavazzi D, Duca L, Graziadei G, Lomino A, Fiorelli G, and Coppelini MD: Membrane found iron contributes to oxidative damage of beta-thalassaemia intermedia erythrocyte. Br J Haematol, 2001; 112 (1): 48-50.
21. Arora RB, Hameed A, and Subramanian et al: Differential serum zinc concentration in some cardiovascular diseases and bronchial asthma. In: Abdulla M, Dashti H, Sarkar B, Al-Sayer H, and Al-Naqeeb N. Metabolism of minerals and trace elements in human disease. 1st ed. New Delhi, India. Smith-Gordon and Company Limited, 1989; p.p. 57-60.
22. De Franceshi L, Braugnara and Beuzard Y: Dietary magnesium supplementation ameliorates anaemia in a mouse model of beta-thalassaemia. Blood, 1997; 90 (3): 1283-90.
23. Rayssignier Y, Guenx E, and Motta C: Magnesium deficiency effects on fluidity and function of plasma and subcellular membranes. In: Lasserre B Durlach J. Magnesium, a relevention. New York, NY, Libbey, 1991; p.p. 311.
24. De Franceschi, Benzardy, Tonault H, and Brugnara C: Modulation of erythrocyte potassium chloride contransport, potassium content and density by dietary magnessium intake in transgenic SAD mouse. Blood, 1996; 88: 2738.

25. Nishiyama S, Kiwaki K, Miyazaki Y, and Hasuda T: Zinc and IGF-1 concentrations in pregnant woman with anaemia before and after supplementation with iron and/or zinc. J Am Coll Nutr, 1999; 18(3): 261-7.
26. Ece A, Uyamik BS, Iscan A, et al: Increased serum copper and decreased serum zinc levels in children with iron deficiency anaemia. Biol Trace Elem Res, 1997; 59 (1-3): 31-9.
27. Arcasor A: Zinc status and zinc therapy in Beta thalassaemia. Am J Pediat Hematol/Oncol, 1989; 11 (2): 245-6.

ASPIRATION CYTOLOGY OF OVARIAN CYSTS AND CYSTIC NEOPLASM: A STUDY OF 39 ASPIRATES

Yarub I. Khattab[1] *MSc, FICMS(Path)*, **Liqaa R. Mussa**[2] *FICMS(Gyne & Obst)*, **Alaa G. Hussein**[1] *MSc, FICMS(path)*

Abstract:
Background: Fine needle aspiration cytology has been widely applied in the diagnosis of ovarian cysts and cystic neoplasms, it offers the advantages of rapid diagnosis, minimal trauma to the patient and low cost, yielding relatively acceptable diagnostic sensitivity and specificity.

Objective: To evaluate the sensitivity and specificity of aspiration cytology in the diagnosis of ovarian cystic lesions.

Methods: 39 female patients with clinico-radiological diagnosis of ovarian cysts and cystic neoplasm underwent ultrasonic guided aspiration cytology in the period from January 2000 to June 2003 at Al-Kadhimiya Teaching Hospital.

Results: Thirty-nine female patients with an age range of 21-48 years underwent ultrasonic guided aspiration cytology of an ovarian cystic lesion. There were thirteen female patients with non neoplastic cysts while twenty six female patients had neoplastic cystic lesion with various histogenesis including (surface epithelial and germ cell type) predominantly benign tumors, while three cases revealed malignant serous cystadenocarcinoma. The over all sensitivity around 37.5% considering the inadequate cytological smears as a false negative result and specificity of 100%.

Conclusion: Fine needle aspiration cytology in the evaluation of ovarian cysts and cystic neoplastic lesions in safe, reliable and rapid diagnostic procedure yielding relatively acceptable sensitivity and specificity.

Key word: FNAC, ovarian cystic lesions

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Introduction

Fine needle cytology has been widely applied in the diagnosis of tumors and tumor like lesions of the abdomen including ovarian lesions[1-7]. It offers the advantages of rapid diagnosis, minimal trauma to the patient, low cost when compared with the standard surgical biopsy. The objective of this study is to evaluate the sensitivity and specificity of fine needle aspiration cytology in the diagnosis of ovarian cystic lesions.

Material & methods

From January 2000 to June 2003, 39 female patients with clinico-radiological diagnosis of ovarian cysts and cystic neoplasm underwent fine needle aspiration cytology under ultrasonic guidance using different approaches and different needle lengths and gauges, all patients were seen at Al-Kadhimiya Teaching Hospital at radiology unit.

The aspirates were done using 20-23 gauge spinal disposable needles with different lengths depending on the depth of the lesion and ideal approach for the aspiration with the patient in supine position. Four smears were made from the precipitate after cytospin, the obtained fluidy sample for each patient was fixed immediately in 25% ethyl alcohol for at least 30 minutes. Smears were stained by hematoxylin and eosin H&E. If fragments of tissues were obtained from the solid part of the cystic neoplasm lesions, they were processed as cellblock preparation. An immediate post aspiration sonography was done to assess and monitor complications. It was possible to compare the cytological result with the histopathological result in 29 cases.

[1]Dept. Pathology, [2]Dept. Gynecology & Obstetric, College of Medicine, Al-Nahrain University
Address correspondence to Dr. Yarub I. Khattab.
Received 2nd January 2005: Accepted 23rd May 2005.

Results

Thirty nine female patients with clinico-radiological diagnosis of ovarian cysts and cystic neoplasm underwent fine needle aspiration cytology under ultrasonic guidance using different approaches depending on the site of the lesion (Tables 1 and 2) with an age range of (21-48) years.

Table 1: Site of ovarian cystic lesions in 39 patients

Site	No. of patients	%
Rt. Ovary	21	53.8
Lt. Ovary	14	35.8
Both	4	10.4
Total	39	100

Table 2: Approaches of FNAC for different ovarian cystic lesions

Approach	No. of patients	%
Transvaginal	3	7.7
Transrectal	0	0
Percutaneous	31	79.5
Intraoperative and /or immediately after surgical removal	5	12.8
Total	39	100

There were thirteen female patients with a non-neoplastic functional cystic lesions underwent cytological diagnosis, while twenty-six female patients had neoplastic ovarian cystic lesions, twenty-three were benign and three were malignant in cytological diagnosis. Inadequate material was obtained in five patients (Tables 3 and 4).

Table 3: Cytological diagnosis in 39 cases with ovarian cystic lesions

Cytological Diagnosis	No. of patients	%
Non-neoplastic cyst	13	33.3
follicular cyst	9	23.0
corpus luteum cyst	4	10.3
endometroid cyst	0	0
Neoplastic cyst	26	66.7
Benign		
serous cystadenoma	17	43.5
mucinous cystadenoma	4	10.3
mature cystic teratoma	2	5.2
Malignant		
Serous cystadenocarcinoma	3	7.7
Total	39	100

Table 4: Correlation between cytological and histological and/or clinico-radiological diagnosis

Cytological diagnosis	No. of patients	Histological and/or radiological diagnosis	
		Benign	Malignant
Benign	31	27	0
Malignant	3	0	3
Inadequate	5	5	0
Total	39	32	3

Considering non-neoplastic cystic lesions as benign lesion and inadequate cases as false negative results, Sensitivity: 37.5%, Specificity: 100%

The elaboration of the numbers reported in table 4 on the correlation between fine needle aspiration results and histopathological and/or clinico-pathological follow up. Final diagnosis especially in non-neoplastic cystic lesion yielded the following:
A sensitivity of (37.5%) considering the inadequate cytological smears as false negative results and a specificity of (100%), complication was encountered in one case in the form of vasovagal attack (7.6%).
Cytological diagnoses were classified into:
1. Non-neoplastic cysts; thirteen patients were presented with non-neoplastic functional cystic lesions, nine follicular cysts; four corpus luteal cysts (Figure 1).

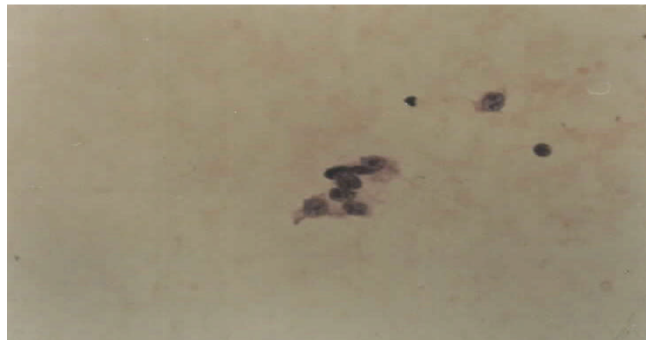

Figure 1: Corpus luteal cyst: smear reveals scattered luteinized granulosa and theca cells on a clean background (RBCs were lysed using glacial acitic acid)(X400).

Three confirmed by biopsy others by close clinico-radiological follow up.
2. Neoplastic cysts: Twenty-six female patients were presented with neoplastic cystic lesions. Twenty three were benign in the form of benign serous cystadenoma in seventeen cases, four mucinous cystadenoma and two cases of mature cystic teratoma, while three cases revealed malignant cytological diagnosis of malignant serous cystadenocarcinoma, all cases confirmed by histopathological final report (Tables 3 and 4; figure 2).

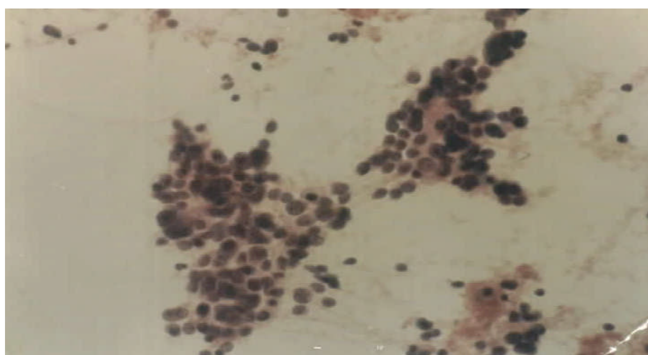

Figure 2: Serous cystadenocarcinoma : smear reveals cellular cohesive clusters of malignant epithelial cells with pleomorphic nuclei. (X400).

Discussion

The use of fine needle aspiration cytology in gynecology was relatively unknown until experiences with ovarian cystic lesions were published in 1970[8]. Ultrasound experiences was led by the work of Ramzy et al detailing the cytopathologic characteristics of ovarian epithelial neoplasms in 1978[2,10], then the use of fine needle aspiration cytological diagnosis in other aspect of gynecology was reported by Sevin et al[11], Moriarity and others[12,13]. Since then a proliferation of highly accurate radiographic imaging and guidance technique has made possible accurate cytological sampling of deep pelvic ovarian cystic lesions with low morbidity. The target organ can be reached by percutaneous, transvaginal, or during laparoscopy route.

Transvaginal aspiration is used in three cases only with deeply seated pelvic ovarian cystic lesions especially when solid portions of the cystic neoplasm gravitated deep in the pelvic cavity in which cannot be reached through percutaneous rout.

Regarding the non-neoplastic functional ovarian cystic lesions, there is controversy whether to aspirate or not. In the present study, the policy to do the aspirate was the following[4,14]:

1. Persistent ovarian cystic lesion though the size of the cyst was less than 5 cm in volume using ultrasound.
2. When the size of the cyst was more than 5 cm in volume.
3. When ultrasonic features of thick irregular wall, multiloculation, or presence of solid portion within the cystic lesions were encountered.

Recently ultrasonic guided ovarian cyst puncture with our without instillation of sclerotherapeutic agents would appear to be a valid alternative to surgery for carefully selected benign ovarian-whether neoplastic or non neoplastic- cysts, especially when the (size is more than 10 cm, the presence of multiloculation, or the suspicion of malignancy) was encountered as a therapeutics management[14,15].

The sensitivity of the test in the present study is relatively lower in comparison to others because of (the limited number of cases, we had no previous experience in that field before, and the fact that nearly all samples were fluidy which usually yielded scattered epithelial cells for cytological study)[2,4,5,7,8,14-].

No significant complications were encountered in this study apart from a vasovagal attack encountered in one case (7.6%).

Conclusion

Fine needle aspiration cytology in the evaluation of ovarian cysts and cystic neoplastic lesions is safe, reliable, and rapid diagnostic procedure yielding relatively acceptable sensitivity and specificity.

Recommendation

Further study are advisable regarding the increasing of number of patients, and the use of this technique not only for diagnosis but also for therapeutic management.

References

1. Khanna AK, and Misra MK: Fine needle aspiration cytology of abdominal masses. J Surg Oncol, 1990; 44 (1): 15-9.
2. Dey P, and Saha SC: Fine needle aspiration biopsy of ovarian neoplasm. Indian J Pathol Microbiol, 2001; 44 (2): 103-6.
3. Rubenchick I, and Auger M: Fine needle aspiration cytology of ovarian cysts in in-vitro fertilization patients: a study of 125 cases. Diagn Cytopahol, 1996; 15(4): 341-4.
4. Mulvany NJ: Aspiration cytology of ovarian cysts and cystic neoplasm: a study of 235 aspirates. Acta Cytol, 1996; 405): 911-20.
5. Ganjei P: Fine needle aspiration cytology of the ovary. Clin Lab Med, 1995; 15 (3): 705-26.
6. Anerson WA, and Nichols GE: Cytologic diagnosis of ovarian tumors: factors influencing accuracy in previously undiagnosed cases. Am J Obstet Gynecol, 1995; 173 (2): 457-63, discussion: 463-4.
7. Granados R: Aspiration cytology of ovarian tumor. Curr Opin Obstet Gynecol, 1995; 7 (1): 43-8.
8. Tord A, and Kjellyren MD: The cytologic diagnosis of ovarian tumors by mean of aspiration biopsy. Acta Cytol, 1972; 26 (4): 336-42.
9. Ramzy I, and De lancy M: Fine needle aspiration of ovarian masses: correlative cytologic and

histologic study of celomic epithelial neoplasms. Acta Cyto, 1979; 23: 27-104.
10. Ramzy I, and De lancy M: Fine needle aspiration of ovarian masses II, correlative cytologic and histologic study of non-neoplastic cysts and non-celomic epithelial neoplasm. Acta cytol, 1979; 23: 185-93.
11. Sevin BU, and Greening SE: Fine needle aspiration cytology in gynecology in clinical aspect. Acta Cytol, 1979; 23: 277-81.
12. Moriarity AT: The role of fine needle aspiration cytology in the management of gynecologic malignancies. Acta Cytol, 1986; 30: 59-64.
13. Imachi M, and Tsu Kamoto N: Fine needle aspiration cytology in patients with gynecologic malignancies. Gynecology, 1992; 4 (2): 309-12.
14. Dordoni D, and Zaglios S: The role of sonographically guided aspiration in the clinical management of ovarian cysts. J Ultrasound Med, 1993; 12(1): 27-31.
15. Lachlan CH, and De Crespigny: The simple ovarian cyst : aspirate or operate. Br J Obstet Gynecol, 1989; 69: 1035-93.
16. Geier GR, and Strecker JR: Aspiration Cytology and E2 content in ovarian tumors. Acta Cytol, 1981; 25: 400-6.
17. Ouinn SF, and Erickson S: Cystic ovarian teratomas: the sonographic appearance of dermoid plug. Radiology, 1985; 155: 477-8.

THE SIGNIFICANCE OF LIPOPROTEIN DEPOSITION IN THE RETINA OF PREGNANT WOMEN AS A MARKER OF PREECLAMPSIA, IUGR AND OTHER RELATED MATERNAL AND FETAL COMPLICATIONS

Wisam A. Ismaeel[1] *FICOG*, **Inas T. Ahmad**[2] *FICOG,* **Akram Sadik**[3]

Abstract

Background: Preeclampsia is a disorder characterized by three main clinical features, these are hypertension, edema and proteinuria. Fibrin deposition in various tissues can be easily demonstrated by microscopical examination, however this requires a biopsy. Fortunately, the eye is the mirror of the body.

Objective: To detect the deposition of fibrin in the eyes of pregnant women within the first two weeks of the third trimester before the women become preeclamptic, through simple ophthalmoscopy

Methods: 42 primigravida pregnant women were chosen as a study group, which include those women in whom hard exudate (Lipoprotein deposition) was detected at 28-30 weeks of gestation in their retina through simple ophthalmoscopy examination. While another control group of 46 women were selected in whom examination of the eye for hard exudate was negative. Both groups were followed routinely in the third trimester and screened for any complication, which may develop.

Results: The incidence of preeclampsia, oligohydramnios, placenta abruption was significantly higher in the study group than in the control group, 45.23% vs 13.04, 28.57% vs 2.17 % and 16.66 % vs 2.17 % respectively. While the incidence of IUGR and perinatal death was still higher in the study group than in the control group 28.57 % vs 2.17 % and 9.52 % vs 0.00 %.

Conclusion: Simple ophthalmoscopic examination of the pregnant women eyes for the presence of hard exudate (Lipoprotein deposition) may be useful in sorting high-risk women for preeclampsia and its related complications.

Key words ; Preeclampsia, hard exudate, retina, lipoprotein

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Introduction

Preeclampsia is a human pregnancy specific disorder characterized by three main clinical features these are hypertension, edema and proteinuria[1-3]. In the last twenty years, much work has been done to elucidate its precise etiological factors. Nowadays it is well established that preeclampsia is a disease of the placenta itself.

The placenta of those women liable to develop preeclampsia differ from those with normal pregnancy by two aspects from biochemical point of view, they produce higher ratio of PG F2@ to that of PG E2. Also, they produce higher amount of placental renin, which is physiologically indistinguishable from the renal renin.the increased production of placental PG F2@ is responsible for the wide spread but low grade activation of the coagulation cascade, ultimately leading to wide spread deposition of fibrin in almost every organ in the body like the liver, spleen and kidneys.

While the increased production of placental renin is responsible for the development of hypertension, through the mechanism of angiotensin[3,4]. The entire above finding is mediated by defective placentation and occurs as early as 12-14 weeks of gestation, and probably mediated by defective immune response[6,7]. Fibrin deposition in various tissues can be easily demonstrated by microscopical examination

[1]Dept. Obstetric & Gynecology, Al-Kindy College of Medicine, Baghdad University, [2]Dept. Obstetric & Gynecology, Al-Elwiya Maternity Hospital, Baghdad, [3]Military Services

Address correspondence to Dr. Wisam A. Ismaeel, e-mail: wisamakram@hotmail.com

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of tissues stained with hematoxylin-eiosin stains[8-10].

However, this requires a biopsy has to be taken from specific organs. Fortunately, the eye is the mirror of the body. That is why we have adopted the principle of examining the pregnant women's eyes for the sake of early detection of lipoprotein deposition in the form of hard exudate, before the signs and symptoms of preeclampsia have yet developed. That is the pivotal idea that we have adopted in committing this paper. To assess whether lipoprotein deposition in the eye of pregnant women can be useful as a screening test for the development of pregnancy induced hypertension.

Preeclampsia and the eye

Actually, most of the studies that have been found in the literature, which describe the retinal changes with preeclampsia have been conducted among women with already established disease. Few if any have been found which describe the early changes in the retina of pregnant women shortly before the onset of preeclampsia. The most prominent findings in the eyes of preeclamptic women include retinal detachment, thrombosis of retinal vessels and retinal hemorrhage[11-14]. In this study, we have tried to detect the deposition of fibrin in the eyes of pregnant women within the first two weeks of the third trimester before the women become preeclamptic, through simple ophthalmoscopy. In addition, to give more accurate assessment about the presence of fibrin deposition in the eye, further confirmation was done by a colleague ophthalmologist. As will be detailed further detailed in the section of methods and patients. Lipoprotein deposition in the retina was described as a lesion indistinguishable from typical hard exudate.

Patients and Methods

1. Population and patients' collection

The study was conducted in Baghdad City at two major maternity hospitals, al-Elwyia maternity hospital and AL-Habibyia maternity hospital. All the patients enrolled in this study were taken from the antenatal clinics from the above-mentioned hospitals. It has included a wide spectrum from Baghdad's inhabitants with different social status and educational degrees.

All the women who have been enrolled in this study have accepted to participate through their verbal consent and supplied with pre-printed data collection sheet to record exactly the events in the third trimester and the outcome of their pregnancy. All the women who have been enrolled in this study were primigravida to make their statistical analysis more conclusive. The study lasted about three years from June 2001 up to March 2004.

2. Follow up protocol

Starting as early as booking all the women were assessed initially by taking full history and meticulous physical examination especially for the blood pressure and uterine size. In addition, all the women were further assessed by the routine antenatal investigation, which include hemoglobin level, random blood sugar, general urine examination, blood urea, serum creatinine, serum uric acid, ABO- Rh blood group, VDRL, as well as Coomb's test for Rh –ve women.

In addition routine ultrasound scan was taken at 16 weeks of gestation. Those women who have been found hypertensive, proteinuric, multiple gestation and low-lying placenta were excluded from the study. The remaining women were followed in the routine way throughout the second trimester up to 28 weeks of gestation. Should as woman remain normotensive she is scheduled for simple outpatient ophthalmoscopic examination to assess the presence of hard exudate in the retina.

In addition, a colleague Ophthalmologist at AL-Jarah private hospital was sent those with positive hard exudate in their retina for further examination and confirmation. Fibrin deposition was described as a grayish

circular lesion, which are arranged in circular or radial arrangement. Accordingly all the women who have accepted to participate in the study were allocated either to the study group (N=42) which include those women in whom hard exudate was detected and confirmed at 28-30 weeks of gestation while they are normotensive.

While the control group (N=46) included normotensive women in whom hard exudate in the retina was absent as assessed by the dual examination. In addition to the above-mentioned screening test, rollover test and serum creatinine samples were assessed for each woman after being assigned to the group mentioned above. The aim is to find any correlation between the presence of hard exudate in the eye and positive roll over test and elevated serum uric acid. After assigning the women to their groups, they were followed up to delivery with the same way.

Those women in whom hypertension did not develop, where secluded to have spontaneous vaginal delivery unless guided by other obstetrical indication like cephalopelvic disproportion or malpresentation, in such cases elective cesarean section was done. Those women in whom hypertension and/ or preeclampsia has developed, were further assessed for the severity of hypertension and the fetal well being test. Those women with preeclampsia has developed in the absence of IUGR, were scheduled for planned delivery at 36 weeks of gestation. While those women with preeclampsia and IUGR, were scheduled to have planned delivery at 34 weeks of gestation or even earlier as guided by the severity of hypertension or the fetal well being tests.

The following parameters were recorded and expressed for each woman in the study, hypertension, preeclampsia, IUGR, oligohydramnios, placental abruption, development of late and variable intrapartum deceleration, number of cesarean sections for fetal distress, Apgar score at 1-5 minutes and the number of infants with neonatal jaundice. Unfortunately Doppler indices and fetal pH estimation could not be collected from all the patients, so skipped from the results. Tanner Thompson's standards were used to assess fetal birth weight percentile.

3. Statistical analysis

The results were expressed as mean and standard deviation for the continuous data and number and percent for the discrete data. Student t test was used to compare the continuous data while Chi square test was used to compare the numerical data. P values less that 0.05 were considered as significant.

Results

After the analysis of the data, the following tables were constructed to show the results and their statistical comparisons. In table number one the overall epidemiological characteristics were presented for both study and control groups. It is interesting to note that the only significant difference between the study group and control groups at 28 weeks of gestation was the significantly higher mean serum uric acid and number of women with positive rollover test.

Table 1: Shows the overall epidemiological characteristics among both study groups

Characteristics	Study group (N=42)	Control group (N=46)	P-value
No. of women who's age is less than 20 years	6 (14.28%)	3 (6.52%)	NS
No. of women who's age is between 20-30 years	33 (78.575)	41 (89.1350	NS
No. of women who's age is more than 30 years	3 (7.14%)	2 (4.34%)	NS
Mean age	23.19+4.41	24.19+3.78	NS
Mean systolic blood pressure at 28 weeks of gestation	111.66+14.51	109.35+14.49	NS
Mean diastolic blood pressure at 28 weeks of gestation	63.78+7.22	62.06+7.19	NS
Mean serum uric acid at 28 weeks of gestation	3.77+0.47	1.72+0.68	<0.05
No. of women with positive rollover test	24 (57.14%)	3 (6.525%)	<0.05

NS = Not significant statistically

Table 2: Shows the overall maternal outcome in women among both groups with their statistical analysis

Characteristics	Study group (N=42)	Control group (N=46)	P value
No. of women who have developed preeclampsia	19 (45.23%)	6 (13.04%)	P<0.05
No. of women who have developed oligohydramnios	12 (28.57%)	1 (2.17%)	P<0.05
No. of women with meconium stained liquor	7 (16.66%)	1 (2.17%)	P<0.05
No. of women with late dceleration	7 16.66%)	2 (4.34%)	NS
No. of women with late and variable deceleration	9 (21.42%)	2 (4.34%)	NS
No. of cesarean section foe fetal distress	7 (16.66%)	1 (2.17%)	P<0.05P
Mean gestational age at delivery in weeks	34.1+1.3	37.12+1.8	<0.05

NS; Not significant statistically

The overall maternal outcome for both groups can be summarized as bellow

1. Number of women who developed preeclampsia was significantly higher in the study group than in the control group {nine (45.23%) vs. six (13.04%): P<0.05}.
2. The number of women who developed oligohydramnios was significantly higher in the study group than in the control group {12 (28.57%) vs. 1(2.17%): P<0.05}
3. The number of women who developed placental abruption was significantly higher in the study group {seven (16.66%) vs. one (2.17%): P<0.05}.
4. The number of women with meconium stained liquor was higher in the study group, yet the difference was not significant {7 (16.66% vs. 2 (4.34%): P<0.05}
5. The number of women who have developed late and variable deceleration was higher in the study group than in the control group {9 (21.425 vs. 2(4.34%); P<0.05}
6. The number of cesarean sections for fetal distress was higher among women in the study group than in the control group {7 (16.66%) vs. 1 (2.17%): P<0.05}
7. The mean gestational age at delivery was lower among women in the study group than in the control group {34.1+1.3 vs. 37.12+1.8: P<0.05}

It is worth mentioning in this regard that among women in the study group who have developed placental abruption two were complete with intrauterine death, while the remaining 5 were partial as confirmed at time of cesarean section by the presence of retroplacental clot. While the only woman in the control group who has developed placental abruption was diagnosed as a case of fetal distress upon developing variable deceleration in the first stage of labor, upon doing cesarean section retroplacental clot was found.

Table 3: Shows the overall neonatal outcome among both study groups

Characteristics	Study group (N=42)	Control group (N=46)	P value
Mean birth weight at delivery	2.1+0.41	3.3+0.54	P<0.05
Number of infants with IUGR	12(28.57%)	1(2.17%)	P<0.05
Apgar score less than 5 at 1 minute	14(33.33%)	7(15.215%)	P<0.05
Apgar score less than 7 at 5 minutes	10(23.80%)	3(6.52%)	P<0.05
Number of infants who developed neonatal jaundice requiring phototherapy	8(19.04%)	1(2.17%)	P<0.05
Number of perinatal death	4(9.52%)	0(0%0)	P<0.05

Table three, summarizes the overall neonatal outcome among infants in both study groups as bellow:

1. The mean birth weight is significantly lower in the study group than in the control group {2.1+0.41 vs. 3.3 + 0.54: P<0.05}
2. Number of infants with IUGR is significantly higher in the study group than in the control group {12(28.57%) vs. 1(2.17%); P <0.05}
3. The number of infants who's Apgar score was lower tan 5 at 1 minute was significantly higher in the study group{ 14(33.33%) vs. 3(6.52%):p<0.05}
4. Number of infants who's Apgar score was less than 7 at 5 minutes was significantly higher in the study group(10923.80%) vs. 3(6.52%);P<0.05}
5. The number of infants with neonatal jaundice requiring phototherapy was higher in the study group {8(19.04%) vs. 1(2.17%); P<0.05}

Among infants in the study group, there were four perinatal deaths. Two cases were associated with complete placental abruption while another two cases were diagnosed as septicemia.

Discussion

There is a direct relationship between positive rollover test at 28 weeks of gestation and the presence of hard exudate in the retina of pregnant women liable to develop hypertension according to the results obtained in this study. Figure 1 shows that up to 57.14% of women in the study group showed positive rollover test compared o 6.52% in the control group.

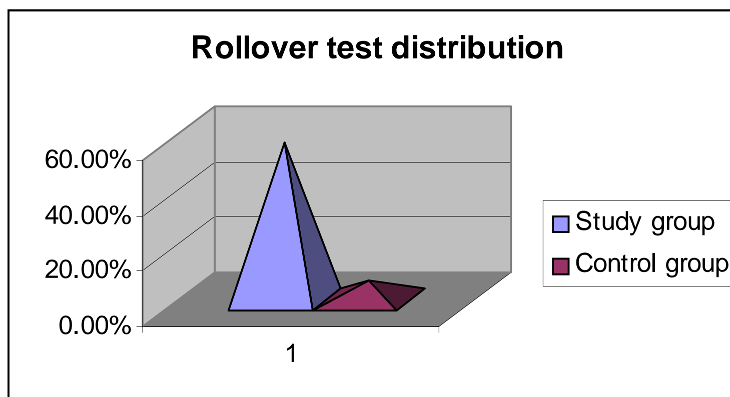

Figure 1: Shows the distribution of positive rollover test among the study groups

There is a direct relationship between elevated maternal serum uric acid and the presence of hard exudate in the retina of pregnant women at 28 weeks of gestation according to the results obtained in this study. Figure 2 shows a histogram of the mean serum uric acid at 28 weeks of gestation between both study groups.

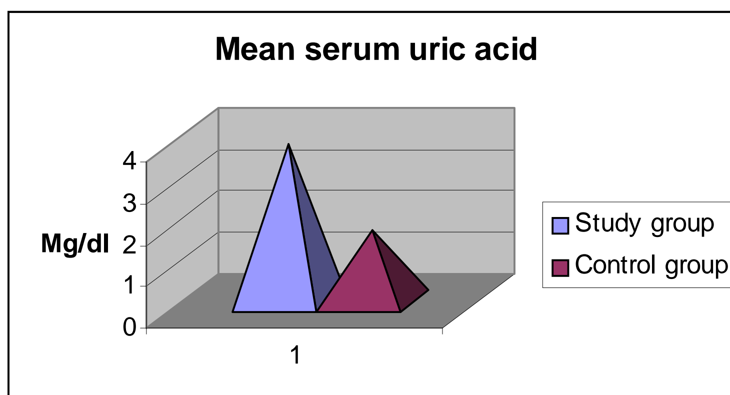

Figure 2: Shows mean serum uric acid at 28 weeks of gestation between both study groups

The idea behind showing increased maternal serum uric acid and the presence of positive rollover test between both study groups is that both are associated with increased incidence of preeclampsia, IUGR and other maternal and fetal complications. This is consistent with the results obtained by Christiansen et al, Yemini et al, Onuonga et al[15-17] as all have stressed the importance of positive rollover test early in the third trimester and later liability to develop preeclampsia in pregnancy. While other researchers like Weeasekera et al; Yoneyama et al and Garrone et al[18-20] have stressed on the direct relationship between elevated maternal serum uric acid early in the third trimester and later liability to develop preeclampsia.

However despite the results we have reached in this study so far, other researchers like Capoor et al[21], described another clinical course through the retinal changes which precedes the development of preeclampsia. In his paper, macular hemorrhage appeared rapidly and shortly before the patient develops preeclampsia. However in the original design of this paper we didn't include examination of the retina after the patient bypass 30 weeks of gestation of pregnancy or the patient already develop preeclampsia. Further researches are required to define the exact pathology, which commence in the eye as the earliest sign of preeclampsia.

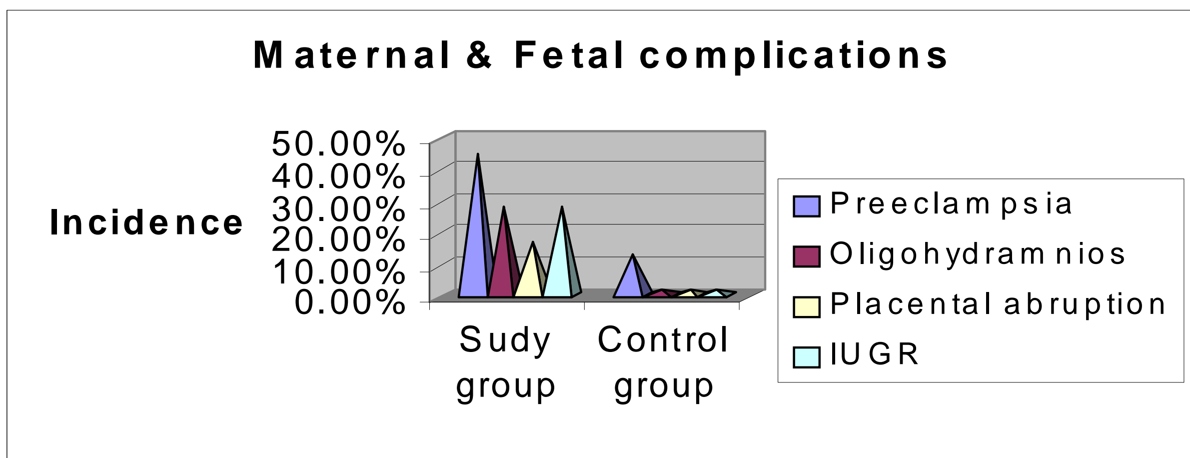

Figure 3: Show the distribution of various maternal and fetal complications between both study groups

The incidence of wide varieties of maternal and fetal complications was higher in the study group than in the control group as shown in figure number three. As it has been explained above that, there was a direct relationship between positive rollover test and elevated maternal serum uric acid with positive hard exudate in the retina of such women. In the same context the incidence of various maternal and fetal complications associated with those tests are expected to be higher among women with hard exudate in their eyes.

Actually, the incidence of preeclampsia placental abruption, IUGR and oligohydramnios were significantly higher in the study group than in the control group. These findings are consistent with results obtained from other researchers like Sanchez et al, Schiff et al; Tewari et al; Yemini et al and Verma et al[22-26]. Among all the above-mentioned papers, the relationship between positive rollover test early in the third trimester and later liability for preeclampsia development has been well clarified.

Conclusion

Simple ophthalmoscopic examination of the retina for the presence of hard exudate in pregnant women early in the third trimester may be helpful in sorting out high-risk women for preeclampsia, IUGR and other related complications. However, it would be much better to follow conservative policy in interpreting the results obtained in this study. We would like to induce other colleagues to commit similar or related researchers to define the exact pathology in the eyes, which precede the development of preeclampsia, and to define the significance of such changes as a screening signs for this disorder.

References

1. Dietl J: The pathogenesis of preeclampsia. J Perinat Med, 2000; 28(6): 464-71.
2. Maquoi E, Schaaps JP, Jacobs JL, et al: Preeclampsia results from placental defects; biology to clinical consideration. Rev Med liege, 1997; 52(7): 478.
3. Toppozada MK: Role of prostaglandins in preeclampsia. Acta Obstet Gynecol Scand, 1990; 69(5): 375-7.
4. Symonds EM: The placenta and the renin-angiotensin system. J Reprod Med, 1979; 23(3): 129-33.
5. Downing GJ, Poisner AM, Barnea ER: First trimester villous placenta has high prorenin and active renin concentration. Am J Obstet Gynecol, 1995; 172(3): 864-7.
6. Loke YW, and Kin A: Immunology of human placental implantation: clinical implication of our current understanding. Mol Med Today, 1997; 3(4): 153-9.
7. Redman CW: Immunological aspects of preeclampsia. Bailliers Clin Obstet Gynecol, 1992; 6(3): 601-15.
8. Moe N: The deposits of fibrin and fibrin like material in the basal plate of the normal human placenta. Acta Patol Microbiol Scand, 1969; 75(1): 1-17.
9. Moe N, and Jorgensen L.: Fibrin deposits in the syncytium of the normal human placenta; evidence of their thrombogenic origin. Acta Path microbiol Scand, 1968; 7294: 519-41.
10. Scheuner G, and gabler W: Histochemical and optic polarization studies of fibrin in ripe human placenta. Acta Histochem, 1966; 23(5): 199-221.
11. Bui BV, Rees SM, Loelinger M, et al: Altered retinal function and structure after chorionic placental insufficiency. Invest Ophthalmol Vis Sci, 2002; 43(3): 805-12.
12. Capoor S, Goble RR, Wheatley T, et al: White centered early hemorrhage as an early sign of preeclampsia. Am J Ophthalmol, 1995; 119(5): 804-6.
13. Shukla BK, and Prasad GN: Retinal hemoorhage in eclampsia and preeclampsia. J Indian Med Assoc, 1976; 66(1): 8-10.
14. Dornan KJ, Mallek DR, and Wittmann BK: The sequelae of serous retinal detachment in pegnant women. Obstet Gynecol, 1982; 60(5): 657-63.
15. Christiansen OB, and Knudsen A: Blood pressure measurement and rollover test at the start of the 3d trimester s predictors of preeclampsia. Ugeskr Laeger (Denmark), 1988; 150(8): 487-9.
16. Yemini M, lancet M, Mass N, et al: Predictor value of rollover test in women with mild preeclampsia. Am J Obstet Gynecol, 1985; 153(1): 77-8.
17. Onuonga O, and Rogo KO: The value of rollover test in the prediction of preeclampsia. East Afr Med J (Kenya), 1988; 65(1): 22-7.
18. Weerasekera DS, and Peiris H: The significance of serum uric acid, creatinine and urinary microprotein levels in predicting preeclampsia. J Obstet Gynecol, 2003; 23(1): 17-9.
19. Yoneyama Y, Suzuki S, Sawa R, et al: Plasma 5-nucleotidase activities and uric acid levels in women with preeclampsia. Gynecol Obstet Invest, 2002; 54(3): 168-71.
20. Garrone C, and Broso P: Uric acid and preeclampsia. Minerva Ginecol (Italy), 1997; 49(5): 213-6.
21. Capoor S, Goble RR, Wheately T, et al: White centered retinal hemorrhages as an early sign of preeclampsia. Am J Ophthalmol, 1995; 119(6): 804-6.
22. Sanchez RL, Briones DK, Kaunitz AM, et al: Prevention of pregnancy induced hypertension by calcium supplementation in angiotensin II sensitive patients. Obstet Gynecol, 1994; 84(3): 349-53.
23. Schiff E, Peleg E, Goldenberg M, et al: The uses of aspirin to prevent pregnancy induced hypertension and lower the ratio of thromboxane A2 to prostacyclin in relatively high-risk pregnancies. N Engl J Med, 1989; 321(6): 351-6.
24. Tewari S, Kaushish R, Sharma S, et al: Role of low dose aspirin in prevention of pregnancy induced hypertension. J Indian Med Assoc, 1997; 95(2): 43-4, 47.
25. Yemini M, Lancet M, Mass N, et al: Predictive value of rollover test in women with mild preeclampsia. Am J Obstet Gynecol, 1985; 153(1): 77-8.
26. Verma UL, Tejani NA, Chatterjee S, et al: Screening for IUGR by rollover test. Obstet Gynecol, 1980; 56(5): 591-4.

TITANIUM MINIPLATE OSTEOSYNTHESIS OF MANDIBULAR FRACTURES

Haithem A. Ziarah[1] *M Med. Sc., F.F.D.R.C.S.I*, **Ammar Yass Khidr**[2] *F.I.C.M.S*

Abstract

Background: The treatment of mandibular fractures (#s) had traditionally involved re-establishment of occlusion by Intermaxillary Fixation (IMF). In order to eliminate the morbidity associated with IMF. IMF has associated morbidity such as weight loss, interference with oral hygiene measures and patient discomfort. Increasingly over the three past decades, oral and maxillofacial surgeons have developed techniques of treating mandibular fractures using internal fixation. The routine use of miniplats in oral and maxillofacial surgery has gained popularity since **Champy** reporded modification of the original technique of **Michelet**.

Objective: Evaluation of titanium miniplate osteosynthesis in treatment of mandibular fractures and comparison of the out come, advantages, disadvantages and complications between intra oral and extra oral approach for the application of titanium miniplates.

Methods: This study was performed prospectively on 24 patients with 28 isolated mandibular fractures and treated by open reduction and titanium miniplate osteosynthesis. Ten patients had intra oral approach and fourteen patients had extra oral approach.

Result: The overall complication rate was 25%. In the group, in which extra oral approach was used, the complication rate was 21.4% (3patients): 2 patients with facial scarring and 1 patient with malocclusion. In the group, in which intra oral approach was used, the complication rate was 30% (3 patients): 1 patient with paraesthesia of the lip, 1 patient with postoperative infection and 1 patient with root injury.

Conclusion: Miniplate osteosynthesis gives acceptable result, and it can be recommended as a routine method for treatment of all mandibular #s. Intra oral approach is advantageous to, and gives comparable results with extra oral approach. Experience in the technique is an important factor in the outcome.

Key words: Trauma-Mandibular fracture-Miniplate osteosynthesis

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Introduction

In an attempt to overcome the disadvantages of intermaxillary fixation (IMF) several authors described the use of bone plates without IMF but the complication rate was unacceptably high, notably, infection, external scar, occlusal derangement and the need for the plate removal. The initial plate either made of stainless steel or of vitalliurm, suffered from a major disadvantage by lacking malleability, which easily resulted in fracture of the plate when attempts were made to bend them[1-3].

The first compression osteosynthesis was performed by Luhr in 1967; he achieved axial compression of the fracture (#) segments by eccentric compression holes and bicortical screws performed exclusively via extra oral approach. Luhr in 1985 demonstrated a significantly reduced rate of infection with an intra oral approach[4].

Michelet in 1973 described osteosynthesis of mandibular #s utilizing miniaturized non-compression plates and self-tapping monocortical screws applied intra orally and the sulcus just medial to external oblique line was used for angular #s, and a juxta-alveolar position in body #s[5].

Champy in 1976 modified Michelet technique using miniaturized malleable non-

[1]**Dept. Surgery-Division of Maxillofacial Surgery College of Medicine, Al-Nahrain University, [2]Al-Kadhihmyia Teaching Hospital Maxillofacial Unit Address correspondence to: Dr. Haithem A. Ziarah, phone No. 5429792**

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compression plates without IMF based on theoretical, biomechanical and experimental studies; he was able to develop **ideal lines of osteosynthesis.** Anterior to mental foramen: 2 miniplates are used, one subapical and another 5mm below to resist the strong torsional forces on this area. Posterior to mental foramen: 1 miniplate is placed subapically.

In the angle region, the vestibular osseous flat area located medial to external oblique line is chosen. He suggested that fixation along alveolar border is stronger than that along the lower border and that only the tractional strains at the alveolar border are needed to be neutralized[6].

We have moved from an era when our primary concern is bony healing and stabilization while preserving maximal periosteal blood supply, to an era when precise reduction and stabilization can be achieved with absolute rigid internal fixation (RIF)[7].

Patients and Methods

This study prospectively reviewed and discussed the result of 24 patients with mandibular # who were admitted to the Maxillofacial Department in Al-Kadhihymia Teaching Hospital during the period from November 2001 to November 2002.

The age of patients ranged from 16-45years, 15 patients were males and 9 were females. Only patients with #s of symphyseal, parasymphyseal, body and angular parts of the mandible were selected (excluding ramus, coronoid and condylar #s) patients in whom mandibular #s were associated with mid-facial #s were also excluded. Diagnosis was based on history, clinical examination and radiographical examination.

Every patient was requested about past medical history to reveal any pre-existing systemic disease that may adversely affect healing or that could be a contraindication to surgery. Level of consciousness and pupil size and reaction were assessed and intracranial nerves examinations were performed to exclude the presence of head injury.

The patients were examined for the presence of blood stained saliva, foeter oris, ecchmosis, sublingual haematoma, and for the presence of pain tenderness on palpation. The oral hygiene was evaluated and classified into good, fair and bad oral hygiene.

Dental status of the patients was evaluated for the presence of missing teeth, badly broken teeth, mobile teeth, painful or tender teeth; periodentally involved teeth and impacted teeth. Electric pulp testing was done to check the vitality of teeth. The occlusion was examined for the presence of step deformity, overriding, posterior gagging, posterior openbite and displacement of midline.

Radiographs were taken to confirm the diagnosis of #s, and to assess the direction of # line, displacement, comminution and the state of teeth in the # line. The radiographs that were commonly used are the orthopantomographs (OPG), oblique lateral mandible, and postero-anterior (PA) mandible. periapical, occlusal and CT-scans were sometimes used. The # site was approached either intra orally or extra orally (including approach through the scar). Combined approach was not used in this study.

Intra oral approach: Wide mucogingival incision through only mucosa, then blunt dissection was done before incising periostum to facilitate subsequent suturing. Periosteum was separated from bone using periosteal elevator exposing, and taking care not to damage the mental nerve.

Extra-oral approach: for symphyseal and parasymphyseal #s, submental incision 1 cm behind the mandible was used. For mandibular body #s, submandibular incision 2cm bellow the inferior border of mandible, the platysma muscle and the deep cervical fascia were incised and reflected to preserve the marginal mandibular branch of facial nerve, the facial artery and vein may have to be ligated. For angular #s, a retro-

mandibular (Risdon) incision was used 2cm behind and bellow the angle of mandible.

When extra-oral was used, the plate was placed at the inferior border of the mandible. However, when an intra oral approach was used, the plate was placed along the Champy's ideal line of osteosynthesis, that is Anterior to the mental foramen, 2 miniplates were used, one sub-apical and another 5mm bellow. If the # was minimally displaced or when segmental arch bar used, 1 miniplate was used. Posterior to the mental foramen 1 miniplate was placed subapically above the inferior dental canal.

Result

From the 24 patients in this study, 15(62.5%) were males and 9(37.5%) were females. The age of patients ranged between 16-45 years. The mean age of the patients was 26.6 years.

The distribution of the etiological factors of mandibular #s, reveals that road traffic accidents (RTA) (11 patients), followed by altercation (9 patients) were the most common etiological factors. Sports, bullet injury, occupational and fallen from height (FFH) were the laest common etiological factors in the study with one patient for each.

From the 24 patients in the study, there was a total of 28 #s, which was treated by RIF. Twenty-one patients (87.5%) had only 1# line, 2 patients (8.33%) had 2# lines and only one patient (4.16%) had comminuted #.

The distribution of the 28 #s was as follow:

1. Symphyseal and prasymphyseal region, 11 #s (39.28%).
2. Mandibular body region, 7 #s (25%).
3. Mandibular angular region, 10 #s (35.71%).

The patients were grouped into tow groups according to the approach to the # site.

Group I included 10 patients in which intra oral approach was used. The vestibular incision was used to approach the body, parasymphyseal and symphyseal #s. The mental nerve was identified in all the patients except in the case of symphyseal # and another patient in whom the area of the nerve was surrounded by fibrosis, postoperative IMF was not used in any patient in this group, and the patients resume normal function soon after operation. This group included 1 patient with symphyseal #, 6 patients with parasymphyseal #s and 3 patients with mandibular body #s. all the patients in the group had a single # line.

Group II included 14 patients in which extra oral approach was used. The submental incision was used for symphyseal and parasymphyseal #s. the submandibular incision was used for mandibular body #s. the angular #s was approach through retromandibular (Risdon) incision. There was one patient in which the parasymphyseal # was approached through the trauma scar. IMF was used for variable periods ranging between 2-6 weeks. In this group, there were seven patients with isolated angular #, two patients with isolated body # and 2 patients with isolated parasymphyseal #. There was one patient with body and contra lateral angular #s, and another patient with angular and contra lateral parasymphyseal #s. There was only patient with comminuted # involving angle and body in one side, and parasymphyseal region in the other side. This group included 4 parasymphyseal #s, 4 mandibular body #s and 10 mandibular angular #s.

In the group in which extra oral approach was used, IMF was used for 2-6 weeks because the plate was applied along the inferior border of mandible. Only in one patient, in whom the approach was through the trauma scar, IMF was not used. When a single # is present, IMF was used for 2 weeks postoperatively, while when 2 # lines are present, IMF was used postoperatively for 3 weeks. IMF for 6 weeks postoperatively was used in 2 patients, the first one had comminuted # and the other one had a delay in treatment of 70 days. The

mean period of IMF in this group was 2.57 weeks (18 days).

In the group in which intra oral approach was used, IMF was not used postoperatively in any patient. Intra operative IMF was done in five patients, while the other five patients, manual reduction were used to establish occlusion.

In the group, in which extra oral approach was used, the complication rate was 21.4% (3 patients): two patients with facial scarring (14.2%) and one patient with malocclusion (7.14%).

In the group, in which intra oral approach was used, the complication rate was 30% (3 patients): one patient with paraesthesis of the lip (10%) and one patient with postoperative infection (10%) and 1 patient with root injury (10%). The overall complication rate in both groups was 25% (6 patients).

Discussion

Many authors have documented the advantages of miniplates, compared with conventional IMF, IMF is either unnecessary or shorter period is needed. Therefore, it allows immediate or early return of function.

In one patient the # was treated by closed reduction and IMF. Postoperatively, radiographs show mal-reduction, the patient was returned to theatre and RIF was used. So we agree with Jaque et al (1997)[8] that closed reduction is more likely to require secondary open reduction than primary open reduction.

We agree with Cawood (1985)[9] that the miniplate system was easy to use, it require less surgeon experience than other systems (compression plates, reconstruction plates, microplates and resorbable plates).

We agree with Baker (1997)[10] and Prein (1998)[11] in that titanium miniplates dose not inferfere with CT scanning. However, we did not see any starburst artifact associated with miniplates, instead the view was very clear. Three-dimensional (3D) reconstructions of CT scans were very clear and were not degraded by artifacts. However, the only disadvantages were that the miniplate could not be differentiated from bone (it appears as bone). We conclude that titanium miniplates should be used when subsequent imaging will be needed in the future.

IMF was applied in all patients in which extra oral approach was used because the plates can only be placed along the inferior border of mandible and not along the ideal lines of osteosynthesis. If IMF is not applied, a gap will be created along the superior border of the mandible during function; this movement may lead to infection and delayed healing. The mean period of IMF in this group is 18 days, which is shorter than the time required for healing if IMF is used alone (6 weeks).

IMF was not applied in the patients in whom intra oral approach was used, as the plates were placed along the ideal lines of osteosynthisis. However, the patients were instructed to use soft diet for 2 weeks.

The intraoral vestibular incision is easily and readily performed without the fear of marginal mandibular nerve injury or scar formation. It readily exposes the #. However, the access is limited rendering the plating technique more difficult especially in the mandibular body and angular regions. In addition, there is increased risk of postoperative infection and injury to the inferior alveolar nerve and the roots of teeth. All of these complications occurred in our sample. These complications should not occur with increased in operator experience in the technique.

The extra oral approach is more difficult and there is fear of marginal mandibular nerve injury and scar formation, and is uncomfortable to the patient. However, it provides wide access rendering the plating technique easier. In addition, it eliminates the risk of postoperative infection and injury to the inferior alveolar nerve and the roots of teeth.

The complication rates observed in this study (25%) were above the previous reports. Nakamura (1994)[12] reported 15.5%complication rate, while Jaque et al

(1997)[8] reported 7% incidence, our limited experience may cause this relatively high complication rates.

However, all the complications in this study (except the case of malocclusion 4.16%) were minor. There was no case of nonunion, no case of severe infection, and no case of motor nerve injury. In addition, there were two cases of unacceptable facial scaring. In addition, the small sample of the study makes the interpretation of the results unreliable. Another cause is that most of the studies are retrospective and many of the complications are not mentioned especially injury to the sensory nerve and roots of teeth.

There was no case of wound dehiscence in the study, this agree with the results of Smith in 1991[13] but in contrast to Cawood in (1985)[9] who reported 12% wound dehiscence. This mostly occur when intra oral approach is used for the posterior parts of the mandibular body angular regions, as the plates, in this situation, is positioned high on the alveolar process near the vestibule. This explains the absence of wound dehiscence in this study.

No patients complained of unusual sensory abnormality, spontaneous pain, hypoesthesia or hyperesthesia in the region of the retained miniplate.

Conclusion

We conclude that

1. The short-term retention of titanium seems to be harmless and without complications.
2. With exception of condylar #s, miniplate osteosynthesis gave acceptable results, and it can be recommended as a routine method for the treatment of mandibular #s.
3. Miniplates produce no scattering during CT. scanning and this is advantageous when subsequent imaging will be needed.
4. Miniplates are rigid enough for stabilization of comminuted fractures especially when supplemented with IMF.

5. Its preferable to place the miniplate along the Champyl' ideal lines of osteosynthesis than along the lower border to avoid postoperalive IMF.
6. Intra oral approach is advantageous to, and gives comparable results with extra oral approach.
7. The application of miniplates is associated with many technically related failures, which can be reduced largely by increasing the experience in its application.

References

1. Roberts WR: The case of mandibular plating. Br J Oral Surg, 1964; 1: 200.
2. Battersby TG: Plating of mandibular #s. Br J Oral Surg, 1967; 4: 194.
3. Becker R: Stable compression plate fixation of mandibular #s. Br J Oral Surg, 1974; 12: 13.
4. Luhr HG, Drommer R, Hotcher U et al: Comparative study between the extraoral and intraoral approach to compression osteosynthesis of mandibular #s. Hjorting-Hansen textbook of Oral & maxillo-facial surgery. 1st edition, Quintessence, Chicago, 1985; (133): p.p. 137.
5. Michelet FX, Deymes J, and Dessns B: Osteosynthesis with miniaturized plates in maxillo-facial surgery. J Oral Maxillofac Surg, 1973; l: 79-84.
6. Champy M, Lodde J.P, Schumitt R, et al: Mandibular osteosynthesis by miniature screwed plates via a buccal approach. J Maxillofac Surg, 1978; 14: 18.
7. Shafer D: Treatment of mandibular angle #s using two miniplates J Oral Maxillofacial Surg, 1994; 52: 1036-7.
8. Jaque B, Richter M, and Arza A: Treatment of mandibular #s with RIF using AO system. J Oral Maxillofac Surg, 1997; 55: 1402-6.
9. Cawood JI: Small plates osteosynthesis of mandibular #s. Br J Oral Maxillofac Surg, 1985; 23: 77-82.
10. Baker S: Concepts and techniques of rigid fixation. Fonseca & Wlker textbook of Oral & maxillofacial Trauma. Second edition, volume II, W.B. Saunders company, 1997; p.p. 1274-1328.
11. Prein J: The use of RIF in mandibular #s complicated by osteomyelitis. J Oral Macillofac Surg, 1994; 52: 1119-21.
12. Nakamura S, Takenoshita Y, and Masnichiro L: Complications of miniplates osteosynthesis for mandibular #s. J Oral Maxillofac Surg, 1994; 52: 233-8.
13. Smith WP: Delayed miniplate osteosynthesis for mandibular #s. Br J Oral Maxillofac Surg, 1991; 29: 73-6.

THE INCIDENCE OF SCROTAL VARICOCELE AS FOUND IN INFERTILE PATIENTS BY CLINICAL EXAMINATION, B-MODE AND COLOR DOPPLER ULTRASOUND.

Usama Al-Nasiri[1] *FRCS*, **FAWAZ A. RASSAM**[2] *FICMS*

Abstract

Background: Considerable attention has been made for improving the diagnosis of varicocele none invasively by color Doppler ultrasound due to the association between it and male sub fertility and the potential for enhanced fertility after varicocelectomy. The color Doppler ultrasound diagnostic criteria for varicocele were; a. Dilatation of pampiniform plexus more than 2mm. b. Retrograde flow in the upright position regardless the size of pampiniform plexus.

Objectives: To assess the value of color Doppler ultrasound compared to clinical examination and B.mode ultrasound in diagnosis of scrotal varicocele.

Methods: A cross sectional study was done on one hundred infertile or hypo fertile patients with clinical suspicion of varicocele who were examined by B. mode and color Doppler US for confirmation or exclusion of the diagnosis or to exclude recurrence after varicocelectomy.

Results: Color Doppler US including B.mode facility increased the incidence of false negative clinical cases by 22% which is very significant and was important to correct the diagnosis of false positive clinical cases in 6% of patients , they were highly valuable in confirming the clinical suspicion of varicocele in 62%.Color Doppler US was highly essential to detect subclinical cases in 5% and detecting recurrence after varicocelectomy in 37% of postoperative cases.

Conclusion: Color Doppler Ultrasound became the standard reference non-invasive imaging modality for diagnosis of scrotal varicocele and following patients after varicocelectomy.

Keywords: Varicocele, B.mode ultrasound, Color Doppler imaging (CDI).

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Introduction

The most common method for identifying varicocele is physical examination, which is convenient, inexpensive and non-invasive. Clinical signs of varicocele are: scrotal swelling, infertility, and abnormally warm scrotum which are due to increased blood flow[1]. Palpable varicocele has been classified clinically in three grades[2]:

Grade I: Varicocele is palpable only during Valsalva maneuver.

Grade II: Varicocele is palpable without Valsalva maneuver.

Grade III: Varicocele is visible and palpable without Valsalva maneuver.

However, physical examination is subjective and is dependent on the experience of examining physician and its limitation was demonstrated in a multicenter study by WHO[3].

It has been suggested that small varicocele not detectable by physical examination alone (subclinical varicocele) may have a role in sub fertility and merit correction[4,5]. Patients with fertility problems may be referred for scrotal ultrasound examination to evaluate testicular size and parenchyma texture, to assess epididymal integrity and to evaluate the presence of subclinical varicocele[6].

[1]Dept. Surgery, College of Medicine, Al-Nahrain University, [2]Rassam X-ray and Colored Doppler Clinic, Al-Saadoon Street, Baghdad.

Address Correspondence to: Dr. Fawaz Rassam, e-mail: fawaz_rassam@yahoo.com

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Ultrasound and /Doppler criteria for diagnosis of varicocele are dilatation of veins of pampiniform plexus > 2mm[7,12] and retrograde flow during Valsalva and/or in the up right position regardless the size of pampiniform plexus[13].

Varicocele occurs in 10-15% of adult men[9,10], and Meacham et al stated that the incidence of varicocele in the general population is 13.4% and in patients with hypo fertility 37%[14]. They are more common on the left side[7,9,12,15,16].

There are two types of varicocele: primary and secondary. Primary varicocele is idiopathic and occurs between the ages of 15 and 25 years. They are the most common correctable cause of infertility[12]. The primary varicocele is believed to be due to incompetent valves in the internal spermatic veins[12].

Veins of pampiniform plexus normally have a diameter between 1-2mm; they change little in size with the patient in erect position, with abdominal compression or with Valsalva maneuver.

The primary type of varicocele may disappear with the patient in supine position therefore CDI should be performed in supine and erect position with Valsalva maneuver to detect varices[18]. Secondary varicocele may result from elevated pressure in the internal spermatic vein produced by tumor, hydronephrosis or muscle strain[12,16], they do not disappear with patient in supine position[12], and in this situation the abdomen and pelvis should be scanned carefully to exclude a mass compressing the spermatic veins on the involved side[9,15] .

Methods

A cross sectional study was done on one hundred infertile or hypofertile male patients proved by two seminal fluid analysis and referred by the urologist with clinical suspicion for varicocele and were examined by B-mode and color Doppler ultrasound for confirmation or exclusion of the diagnosis during the period from June 2001 to Nov. 2002 using Siemens versapro color Doppler ultrasound machine.

The physical examination was done by a urologist and was performed in supine and standing position before and during Valsalva maneuver, to give the clinical impression before knowing the results of ultrasound examination.

All patients were selected depending on the history and at least two abnormal seminal fluid analyses in accordance to WHO seminal fluid normal values[19] which are volume = 1.5-5cc, concentration ≥ 20 million/cc, motility ≥ 50%, and morphology ≥ 50%.

Values less than those mentioned above were regarded as abnormal. Laboratory personnel performing the seminal fluid analysis were unaware of the results of physical and ultrasound examination.

Once the physical and seminal fluid examinations were completed, the patient was referred for color Doppler ultrasound examination using linear high-resolution (7.5 MHz) transducer, initially in supine position and the patient was asked to hold his penis suprapubicaly.

For each patient the following steps were carried out by color Doppler ultrasound examination:-

1. Size of each testis: Testicular atrophy was diagnosed when testicular size was less than 3x2 cm depending on Feld and Hricak Criteria[7,9].
2. Parenchyma texture, for exclusion of focal lesion.
3. The epididymes for any sign of inflammation, epididymal cyst or spermatocele.
4. Identification of pampiniform plexus by B-mode and color Doppler ultrasound to assess their size accurately in supine and erect positions.
5. Detection of reversed flow in pampiniform plexus using color Doppler ultrasound in supine and erect positions with Valsalva maneuver.

Results

One hundred male patients having at least one-year history of infertility, were examined, their age range was 21- 41 years. (Mean = 33 years).

Table 1: The incidence of varicocele by physical examination, B-mode ultrasound and CDI

Type of Examination	RT (%)	LT (%)	Total no. of patients
Physical	8	70	100
B-mode UIS	39	69	
Color Doppler UIS	21	50	

The incidence of patients with negative physical examination and positive B-mode and/or CDI for varicocele was 22%. The total incidence of right varicocele was 8% by clinical examination, 39% by B-mode ultrasound and reversed flow was detected by CDI in 21% of patients. The total incidence of left varicocele was 70% by clinical examination, 69% by B-mode ultrasound and reversed flow was detected by CDI in 50% of patients.

Only 6% of patients had high clinical suspicion for varicocele with no B-mode and / or CDI evidence. The high clinical suspicion for varicocele was supported by B-mode and/or CD ultrasound in 62% of patients, 54% were supported by B-mode only while 36% were supported by CDI ± B.mode ultrasound.

Recurrence of varicocele was diagnosed 6 months-6 years after varicocelectomy by reversed flow in 3/8(37%) of patients by CDI. Varicocele was detected on the other side instead of or in addition to the clinically suspected side in 19% of patients by B-mode ultrasound and in 10% of patients by CDI.

CDI was the only clue for subclinical varicocele in the absence of clinical or B-mode ultrasound findings in 5% of patients, all of them were on the left side.

Size of the testes:

Testicular atrophy was found in 11% of patients with infertility, 5% on the right side and 6% on the left side. Bilateral testicular atrophy was present in 4% of patients.

Discussion

CDI is a valuable non-invasive new imaging modality which altered the diagnosis and management of causes of infertility, but it is an operator dependent procedure and needs sufficient experience, i.e., any case with suspicion of varicocele should be examined in supine and standing position as reversed flow which is very important to detect subclinical varicocele may be seen only on standing position with Valsalva maneuver. If such patients are examined in supine position only, the reversed flow may not be detected and these cases may be missed as normal, meanwhile they are also normal on physical examination, therefore, CDI has now become the most reliable test to detect non palpable reflux or confirm questionable reflux[2].

CDI has superseded ultrasound in measurement of venous diameter which shows too much overlap between competent and incompetent veins, a spermatic veins > 3mm in diameter can be competent while veins less than 2mm can be incompetent, between 2 and 3 mm the overlap is such that B-mode ultrasound is not reliable, so CDI is more sensitive and can detect up to 93% of reflux.

Brief reflux that lasts less than a second is physiological and can be seen in 42-50% of normofertile men without palpable varicocele[14].

Permanent reflux is non palpable in 20% of cases and lasts more than 2 seconds, it does not correlate with the diameter of spermatic veins[2], in such cases CDI is very helpful to detect subclinical varicocele. Intermediate reflux is never palpable and lasts 1-2 seconds; it keeps decreasing during

Valsalva and stops before the end of the maneuver.

In this study permanent reflux only was regarded as subclinical varicocele depending on the study of Cornud et al[20] who found that the Doppler aspect and change after treatment in patients with permanent reflux on CDI are identical to those with palpable varicocele. This standard helped a lot to clarify the controversy over whether or not non palpable reflux should be treated in hypofertile men, as Marsman and Schats stated that only patients with permanent reflux should be treated[20].

B-mode and CDI were positive in 22% of patients with negative physical examination, which indicates that B-mode plus CDI can increase the incidence of varicocele by 22%, which is very significant and subsequently alters the management of infertile cases.

B-mode and CDI were very useful as a standard reference method to correct the diagnosis of false positive clinical cases, which were not uncommon (6%) in the current study, and this was useful to avoid unnecessary surgery.

B-mode and CDI were highly valuable in confirming the high clinical suspicion of varicocele in 62% of patients, and this is very important for surgery from the medico legal point of view as some of the patients who do not benefit from varicocelectomy claim that the clinical diagnosis was wrong and there was no solid evidence by a documented test, so CDI became solid documented investigation to convince the patient.

B-mode and CDI are very important in follow up of patients after varicocelectomy, especially in patients with persistent poor seminal fluid results. The usual expected findings after successful varicocelectomy are reduction in size of pampiniform plexus to normal with no reversed flow, this should be checked ideally at least 6 months after the operation to give enough time for the pampiniform plexus to decrease in size, this if they were mildly dilated, but if the pampiniform plexus was markedly dilated it will be found dilated but thrombosed with no upward or reversed flow postoperatively. This group of patients are important for follow up by CDI as it is the only method which ensure successful varicocelectomy with no detectable flow regardless the size while clinically they may be regarded falsely as recurrent varicocele. Therefore B-mode ultrasound alone is not sufficient to diagnose recurrence of varicocele and in this study significant number of patients 3/8(37%) showed recurrence of varicocele depending not only on persistence of large pampiniform plexus on B.mode ultrasound but also the presence of reversed flow on CDI.

The high sensitivity of ultrasound in diagnosing varicocele was essential in detecting it on the other side or in addition to the clinically suspected side in 19% of patients by B-mode ultrasound and in 10% of patients by CDI which is important to prepare the patient for bilateral instead of unilateral varicocelectomy and missing varicocele on one side may be the cause of persistent poor seminal fluid results after unilateral varicocelectomy.

Detecting subclinical varicocele by CDI in 5% of patients with infertility is significant in this study making referral of infertile patients for CDI is mandatory. B-mode ultrasound was useful in detecting testicular atrophy as a medical cause of infertility, which was found in 11% of patients in this study, and it was bilateral in 4% of patients. CDI was useful in excluding or confirming varicocele as a surgical cause of infertility in patients with testicular atrophy.

The total incidence of right varicocele was significantly high by B-mode (39%) and CDI (21%) than by clinical exam (8%) and this is due to the fact that clinicians always concentrate in their examination on the left side because left varicocele is more common, as a result they easily miss subclinical (or even RT varicocele) while the total incidence of left varicocele in this study was almost the

same by clinical examination (70%) and B-mode ultrasound (69%) due to the dedicated clinical examination for the left side, but CDI was also useful in detecting reversed flow in 50% of patients with left varicocele.

The high incidence of left varicocele in this study compared to Meacham et al[14] was (37%) was due to high selection criteria for CDI, referring only patients with high clinical suspicion rather than the patients with negative clinical examination.

Conclusion

Color Doppler ultrasound examination became the standard non-invasive investigation for varicocele as it is much more accurate than clinical examination and B-mode ultrasound. It is highly valuable for following patients after varicocelectomy, its main limitation that it is operator dependent.

Recommendation

Color Doppler ultrasound examination should be the standard reference investigation for diagnosis of scrotal varicocele before any varicocelectomy and can be a good base line for follow up after surgery. Color Doppler ultrasound should be the gold standard non-invasive imaging modality for subclinical varicocele.

References

1. Mark Sigman, MD, Jonathan P.Jarow, MD: Male infertility. In: Walsh PC, Retik AB, Vaughan ED, Wein AJ, eds. Campbell's Urology. Philadelphia: W, B Saunders. 2003, 43: 1507.
2. Cornud E, Amar E, Hamidak, et al.: Ultrasound Findings in male hypo fertility and importance. Europ Radiol, 2001; 11.
3. WHO. Comparison among different methods for the diagnosis of varicocele. Fertil Steril, 1985; 43: 575-81.
4. Mc Clure RD, and Hricak H: Scrotal ultrasound in the infertile man: Detection of subclinical unilateral and bilateral varicocele. J Urol, 1986; 135: 711.
5. Mc Clure RD, Khoo D, Jarri K, and Hricak H: Subclinical varicocele: the effectiveness of varicocelectomy. J Urol, 1991; 145: 789.
6. Pierik FH, Dohle GR, Van Muiswinke JM, Vreeburg JTM, and Weber RFA. Is routine scrotal ultrasound advantageous in infertile men. J Urol, 1999; 162: 1618-20.
7. Feld R, and Middleton WD: Recent advances in sonography of the testis and scrotum. Radiol Clin North Am, 1992; 30: 1033-51.
8. Gerscovich EO: High resolution ultrasonography in the diagnosis of scrotal pathology: I Normal scrotum and being disease. J Clin Ultrasound, 1993; 21: 355-73.
9. Hricak HH, Hamm B, Kim B (Eds): Imaging of the scrotum. New York, Raven Press, 1995.
10. Rifkin MD, Foy PM, Kurtz AB, et al: The role of diagnostic ultrasonography in varicocele evaluation. J Ultrasound Med, 1983; 2: 271-5.
11. Sigmund G, Gall H, and Bahren W: Stop type and shunt type varicocele: venographic findings. Radiology, 1987; 163: 103-10.
12. Stewart R, and Carroll BA: The scrotum. In Rumack CM, Wilson SR, Charboneau JW (Eds): Diagnostic ultrasound, vol 1. St.Louis, Mosby year Book; 1991.
13. Oyen RH: Scrotal ultrasound. Europ Radiol, 2001; 11.
14. Meacham RB, Towsend RR, Rademacher D, and Drose JA: The incidence of varicocele in the general population evaluated by physical examination, gray scale and color Doppler sonography. J Urol, 1994; 151: 1535-8.
15. Hill MC, and Sanders RC. Sonography of benign disease of the scrotum. In sanders RC, Hill MC (Eds): Ultrasound Annual. New York, Raven Press; 1986.
16. Hricak HH, and Frilly RA: Sonography of the scrotum. Invest Radio1, 1983; 18: 112-21.
17. Philips GN, Abrams HJ, and Subaijas K: Scrotal ultrasonography. In sanders RC, Hill MC (Eds): ultrasound Annual; 1983, New York, Raven Press.
18. Leonard WC: Scrotum. In: Kawamura DM. Diagnostic medical sonography, a guide to clinical practice, 2nd Ed, New York, Lippincott; 1997; p.p. 750.
19. World Health organization. Laboratory Manual for the Examination of Human Semen and semen-cervical Mucus Interaction. Cambridge: Cambridge university press; 1987, appendix 1.A, p.p. 27.
20. Marsman J, and Schats R: The subclinical varicocele debate. Human Reprod, 1994; 9: 1-8.

PREVALENCE OF PARKINSON'S DISEASE IN AL-KADHIYMIA DISTRICT (BAGHDAD CITY): COMMUNITY-BASED STUDY

A. Mutalib A. Kareem[1] *FRCP*, **Amjad D. Niazi**[2], *DTPH, PhD*, **A.F. Abdullah**[1] *MBChB*

Abstract

Background: Parkinson's disease is a chronic neurodegerative disorder affects mostly people above 40ys. Studying its prevalence is crucial for health public planning especially as worldwide communities are getting older. There are some worldwide variations in the estimated prevalence rates and the figures are unknown in our country.

Objective: To estimate the prevalence of Parkinson's disease in Al-Kadhiymia district.

Methods: Community-based study was conducted as cross-sectional survey on random sample of the population of the district. Suspected cases of Parkinson's disease identified during home visits were referred to the neurological department at the University Hospital of Iraqi Medical College in order to confirm the diagnosis of the senior neurologist. Diagnosis is made by identifying at least two cardinal features of the disease (resting tremor, bradykinesia, rigidity and postural instability) in the absence of signs of secondary parkinsonism.

Results: 25 cases of Parkinson's disease collected from a random sample of 22,988 individuals (13 were males, 23 were females. 6 lived in rural areas and 19 in urban). Three cases (12%) were newly diagnosed. Tremor was the predominant symptom of onset (80%). 19 cases had bilateral involvement of the disease, in spite of the unilateral onset of all cases. The crude prevalence rate was 108.75 per 10^5 population. Age adjusted prevalence rates showed constant increase with age. Gender-adjusted prevalence rates were calculated for male $114/10^5$ populations and for $103/10^5$ population. Residency-adjusted prevalence rates were 114,3 and 94,3 per 10^5 population for urban and rural living respectively.

Conclusion: Prevalence rate of Parkinson's disease is just lower than the figures in Europe and North America, but higher than those of Africa and China. It increases constantly with increasing age. There was no significant gender or rural difference in the prevalence rates. The prevalence figure can be applied to the population of Baghdad City because of the similar population structure and characteristics to those of Al-Kadhimiya district.

Key words: Parkinson disease, prevalence, Baghdad, Cross sectional study

IRAQI J MED SCI, 2005; VOL. 4 (2): 179-186

Introduction

Parkinson's disease (PD) is a progressive neuro-degenerative disorder, which affects the movement or the control of movement including speech and "body language". Four cardinal signs dominate it: bradykinesia, tremor and rest, rigidity and postural instability. It mostly affects elderly people with overall prevalence of about 1.6 percent in persons over 65 years of age[1].

The disease is chronic and progressive. Its life expectancy increased since the introduction of L-dopa treatment. Other medical therapies and some resent surgical techniques providing continuous improvement of the disease disability[2].

In addition, the population of the world, in general, is growing older because of the improving health services. This gives an indication that PD will affect more people i.e incidence and prevalence of the disease will continue to rise[1]. These facts indicate that the importance of PD as a population health

[1]Dept. Medicine-Section of Neurology, College of Medicine, Al-Nahrain University [2]Iraqi Committee of Medical Specialization.

Address correspondence to: Dr. A. Muttaleb A. Kareem BO box 70041. Email a_mutaleb@yahoo.com

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issue is expected to increase in the near future.

The prevalence of PD is studied worldwide with some variations in the figures[3-33]. In 1990 worldwide estimated 4 million people were suffering from PD, approximately one million of them in North America[1,2].

In Iraq, there is no local record about the prevalence of PD or the epidemiological factors that affect it. Therefore, we found that it was necessary to study this subject in order to identify the disease extent and to plan useful measures for health care institutions and personnel.

Patients and Methods

Area of the study

The area chosen for the study was Al-Kadhiymia district, one of the original regions in Baghdad City (Capital of Iraq). It is located north of Baghdad on the western bank of the Tigris River. The total population of Al-Kadhiymia district is 480686 people, according to the Iraqi population survey of 1997 distributed into 38 sectors in urban area and 16 villages in rural one. It was chosen because of:

1. Large number of its population,
2. Its inhabitants are of different social classes,
3. Containing both rural and urban areas,
4. Its proximity to the University Hospital.

All individuals involved in the study were asked to take part in it. Their consent was taken while visiting their families at home.

Sample of the study

A random sample was determined from the general population of the district as follows:

* For urban area: in each sector, 20% of the families were selected randomly and included in the study.

* For rural area: 20% of the families living in villages were randomly selected to be enrolled in the study.

The sample was about 23 000 individuals described as families in order to involve all age groups. The random sample was determined and designed in cooperation with the Central Statistical Organization authorities that provided us with a detailed account on the names, locations and numbers of the selected streets and sectors. These numbers were listed by local authorities, in marks at beginning and end of streets.

Survey

This is a community-based study, designed as a cross-sectional survey on the general population. A suitable questionnaire was designed *(see appendix)* in two parts so that the study was run in two phases:

* Phase 1 (screening phase): Families were interviewed at home. One of the researchers did the interviewing to explain the purpose of the study, ask questions about age and gender of each family member and the presence of any of the following:

1- Tremor of the hands "Is there any trembling movement of the hands?"

2- Bradykinesia "Is there any slowness of movement during walking?"

3- Previous diagnoses of PD "Is there any member who has the diagnoses of PD?"

4- Use of antiparkinsonian drugs "Is there any one who is taking L-lopa, Trihexyphenidyl or Bromocriptine tablets?"

Any member who had positive answer would be examined to demonstrate the parkinsonian signs. Patients excluded from further assessment if they had used anti parkinsonian drugs for other indications (e.g. Bromocriptine for pituitary adenoma or prophylactic treatment with anticholinergic drugs in association with dopamine antagonists in patients with psychotic disorder).

* Phase 2 (confirmatory phase): All suspected cases of PD were referred to the neurological department of the Al-Kadhimiya Teaching Hospital where history and neurological examination is performed by senior neurologist.

Patients with confirmed diagnosis of PD would be questioned about: the duration of their symptoms, type of onset, drug ingestion (neuroleptics, antidopaminergic), family history of PD or tremor, presence of medical illness, smoking, number of rooms, family members and total years of education (*see appendix*).

Diagnosis criteria

According to the WHO definition[1] PD was diagnosed when the patient had at least 2 of the cardinal signs (resting tremor, bradykinesia, rigidity and postural instability) with no signs of nervous system involvement, such as corticospinal deficit, cerebral dysfunction, conjugate down or lateral gaze impairment, or prominent early automatic nervous system involvement. Patients with drug induced Parkinsonism were excluded.

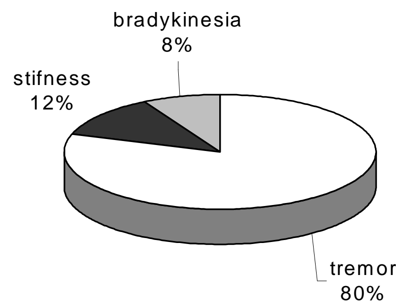

Figure 1: The distribution of onset symptoms in patients with Parkinson's disease

Statistical analysis

Our data was computed in Pentium III system using the statistical analysis system (SPSS version 10.0). We calculated the prevalence rates as crude and category specific (age, gender, and residency). The quantitative data were expressed as mean (X) standard deviation (SD). We determined whether differences in prevalence rates were statistically significant or not by student (t) test and chi-square (X^2) test. A probability limit (p value) of <0.05 was considered statistically significant.

Results

The cross-sectional survey started from December 1st 2000 till September 15th, 2001. The total population involved in this study was 22988 individuals, 11645 (50.7%) of them were females and 11334 (49.3%) were males. Individuals living in urban areas were 16646 (72.3%) and in rural ones were 6364 (27.7%). 76 individuals with suspected PD were referred to the Al-Kadhimiya Teaching Hospital, 55 (72.3%) of them responded and 21 did not.

Those who did not respond two of them had written certificate of diagnosis of PD by a senior neurologist, two were bedridden with full certificate of PD confirmed by the neurologist, and for the other a second visit was done for reassessment that revealed exclusion of diagnosis. A total number of 25 cases of PD were collected, 13 males (52%) and 12 females (48%). Three cases (12%) were not diagnosed yet by the time of the survey. Those who were referred but not diagnosed as

PD, they had benign essential tremor, dementia or cerebrovascular accident.

The crude prevalence rate of PD was **108.75 per 100 000** population. The results of category specific prevalence rates are as follows:

Age: Table 1 shows the age-specific prevalence rates for the total population of the study. The age is categorized a below 20 years and groups of 10 years age for those 20 years and above. The youngest case was 35 years of age and 94 years was the age of the oldest. The most prominent collection of cases was in the age group of 60-69 years, it contained nine cases representing 36% of the total.

Table-1: Age-specific prevalence rates of Parkinson's disease in Al-Kadhiymia district

Age group	Population	Prevalence per 10^5
<20	12585	0
20-29	3671	0
30-39	2852	35.06
40-49	1693	118.13
50-59	1026	487.33
60-69	699	1287.55
70-79	317	1892.74
>80	113	2068.97
Total	22988	108.75

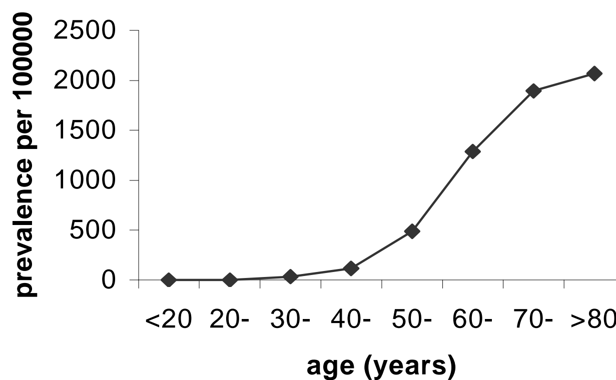

Figure 2: Correlation between prevalence rates of Parkinson's disease with age in Al-Kadhiymia

Gender: The prevalence rate of PD in men ($114.7/10^5$) was slightly higher than in women ($103.05/10^5$), as shown in table-2. This difference was statistically not significant (X^2= 0.07, p=0.789).

Table-2: gender-specific prevalence rates of Parkinson's disease in Al-Kadhiymia district

Age group	Male			Female		
	Total	**PD**	**Prevalence per 10^5**	**Total**	**PD**	**Prevalence per 10^5**
<20	6161	0	0	6424	0	0
20-29	1821	0	0	1850	0	0
30-39	1407	1	0	1445	1	69.2
40-49	865	0	115.61	828	1	120.77
50-59	551	4	725.95	475	1	210.53
60-69	315	3	952.38	384	6	1562.5
70-79	150	3	2000	167	3	1769.4
>80	64	2	3125	81	0	0
Total	11334	13	114.7	11645	12	103.05

Residency: Table-3 shows the distribution of cases of PD among rural and urban areas. Ninteen cases were living in urban areas (10 males, 9 females) and six cases in rural one (three males, 3 females). There was slight increase in prevalence rate of PD in urban ($114.29/10^5$) more than in rural ($94.28/10^5$). This difference was of no statistical significance (p=0.68).

Table 3: residency-specific prevalence rates of Parkinson's disease in Al-Kadhiymia district

Area	**Cases**	**Total**	**Prevalence per 10^5**	**Significance**
Urban	19 (76%)	16624 (72.3%)	114.29	X^2 = 0.17 P = 0.68
Rural	6 (24%)	6364 (27.7%)	94.28	
Total	25	22988	108.75	

Onset: The predominant symptom of onset was tremor, which was present in 20 cases (80%), stiffness of the back was the initial symptom in three cases (12%) while bradykinesia was in two cases (8%), as shown in figure-2. All cases had a unilateral onset.

Laterality: The disease was unilateral in six cases (24%), and bilateral in 19 cases (76%). The average duration of the illness in unilateral disease was about half that of bilateral one. This is shown in table-4.

Table 4: The relation between average duration and laterality of illness in patients with Parkinson's disease

Laterality	**cases**	**Duration (years)**	**significance**
Unilateral	6 (24%)	2.5±2.41	T = -1.57 P = 0.13
Bilateral	19 (76%)	4.42±2.68	
total	25	3.84±2.68	

Discussion

Studies on the prevalence of PD had been conducted in many parts of the world, for example: Australia, New Zealand, Japan, United States, United Kingdom and Iceland. These studies generally relied on record of providers of health services (mainly hospitals and medical practitioners) in the identification of cases. Such maneuvers exclude individuals who failed to seek medical attention for their PD symptoms, as well as those who were improperly diagnosed.

This study was a community-based one where the approach of case finding was to go into the community and screen for patients with PD at home. This approach is more accurate especially in our circumstances where medical records are improperly handled and seeking medical advice is incomplete because of ignorance of symptoms, socio-economic difficulties or disease disability.

This is the first community-based study for PD carried out in our country to estimate prevalence of PD.

We found the crude prevalence of PD in the study population was 108 per 100 000 population. This figure is slightly lower than those reported in Europe and North America: London (193)[1], Finland (166)[4], San Morino (152)[5] and Canada (244)[6]. However, it is much higher than African figures: Nigeria (10)[7], Libya (31)[8], as well as China (57)[9].

These differences may be attributed to a different genetic susceptibility in different race[10]. However, other factors should be considered. Different age structures where older age population is more in developed countries may contribute to the higher prevalence rates[11].

Morens et al [12] found that incidence rates were similar in Asian-American men and in white men. This may be explained by more complete ascertainment than the previous studies. Alternatively, it may reflect a real increase in disease frequency among nonwhite U.S. residents because of increased exposure to an environment factor.

Recent prevalence study on PD was conducted in Taiwan on people of similar ethnic group to Chinese. It revealed a prevalence rate similar to European figures, suggesting that environmental factors might be more important than racial factors in the pathogenesis of PD[13].

Age: There was a consistent and rapid increase in prevalence PD with increasing age, even in the highest age categories. It increases about 20 times from age group of forty to that of eighty. This finding was similar to other studies[4,11,13,33]. Three cases (12%) were identified below the age of 50, corresponding to WHO figure[1].

Although PD is intimately related to aging, it has been demonstrated that its underlying process is distinct from natural aging. There is a marked microglial reaction to neuronal damage in PD that is not seen in normal aging[36]. It may be explained by aging-related factors like chronic exposure to neurotoxicants.

Gender: Prevalence of PD regarding gender distribution is controversial. Some studies show higher prevalence rates in men than women[3,4,6,8,19,20,21,22,37]. Other studies[5,10,14,15,24] found equal gender prevalence. In our study, there was no significant gender difference in the prevalence rate.

Residency: Several studies[6,24-26] indicated that rural living is a significant risk factor for PD. As Gorell study[38] we found no association between rural living and PD. It's the individual characteristics of rural living like farming, use of well water and exposure to herbicides and insecticides are the factors associated with PS[24,25]. These factors need to be evaluated in a specific case-control study with larger sample size.

Characteristics of the disease: The onset symptom was predominately tremor 80%, while stiffness only 12%. This may be

explained, as both stiffness and slowness of movement are manifestations of getting older, so the patients were unaware of them.

Three cases were first diagnosed at the time of study representing 12% of total cases. This is agreed with the assumption of Shrag[3] that 10-20% of all community patients remain undiagnosed.

Conclusion

1- Prevalence of Parkinson's disease in Al-Kadhiymia district is 108.75 per 100 000 population. This figure is slightly lower than European and American figures but more than in Africa and China.

2- There were no significant gender or residency differences in the prevalence of Parkinson's disease.

3- The prevalence figure can be applied to the population of Baghdad City because of similar population structure and characteristics to those of Al-Kadhiymia district.

References

1. Press Release WHO / 71. Parkinson's disease: A unique survey launched. 14 October 1998. Internet : www. Who. Org
2. Adams RD et al: Principles of neurology. 5th edition, NewYork, McGraw-Hill, Inc. 1993, chapter 43: p.p. 937-42.
3. Schrag A, Quinn NP, Ben-Shlomo Y: Cross sectional prevalence survey of idiopathic Parkinson's disease and parkinsonism in London. BMJ, 2000; 321: 21-2.
4. Kuopio AM, Moarttila RJ, Helenius H, Rinne UK: Changing epidemiology of Parkinson's disease in southwestern Finland. Neurology 1999; 52: 302-7.
5. Alessandro RD, Gamberini G, Granieri E, Naccarato S, Manzaroli D: Prevalence of Parkinson's disease in the republic of San Marino. Neurology, 1987; 37: 1679-82.
6. Svenson LW, Platt GH, Woodhead SE: Geographic variation in the prevalence rates of Parkinson's disease in Alberta. Can J Neurol Sci, 1993; 20: 307-11.
7. Osuntokun BO, Adeuja AO, Schoenberg BS, Bademosi O, Nottidge VA, Olumide AO: Neurological disorders in Nigerian Africans: a community- based study. Acta Neurol Scand, 1987; 75: 13-21.
8. Ashok PP, Radhakrishnan K, Sridharan R, Moussa ME: Epidemiology of Parkinson's disease in Benghazi, North-East Libya. Clic Neurol Neurosurg, 1986; 88: 109-13.
9. Li SC, Schoenberg BS, Wang CC, etal. A prevalence survey of Parkinson's disease and other movement disorders in the People's Republic of China. Arch Neurol, 1985; 42: 655-7.
10. Kessler I. Epidemiological studies of Parkinson's disease: A community- based study. AM J Epidemiology, 1972; 96: 242-54.
11. Kusumi M, Nakashima K, Harada H, Nakayama H, Takahashi K. Epidemiology of Parkinson's disease in Yonago City, Japan: comparison with a study carried out 12 years ago. Neuroepidemiol, 1996; 15: 201-7.
12. Morens DM, Davis JW, Gradinetti A, Ross GW, Popper JS, WhiteLR. A prospective study of middle-aged men. Neurology, 1996; 46: 1044-50.
13. Chen RC, Chang SF, Su CL, etal. Prevalence, incidence and mortality of Parkinson's disease: A door to door survey in Ilan Country, Taiwan. Neurology, 2001; 57: 1679-86.
14. DE Rijk MC, Breteler MM, Graveland GA, Grobbee De, Meché FG, Hofman A. Prevalence of Parkinson's disease in elderly: The Rotterdam study. Neurology, 1995; 45: 2143-46.
15. Morgante L, Rocca WA, Rosa AE, etal. Prevalence of Parkinson's disease and other types of parkinsonism: A door to door survey in three Sicilian municipalities. Neurology, 1992; 42: 1901-7.
16. Mutch WJ, Fordyce ID, Downie AW, Paterson JG, Roy SK: Prevalence of Parkinson's disease in a Scottish city. BMJ, 1986; 292: 534-6.
17. Tanner CM, Chen B, Wang W, et al: Environmental factors and Parkinson's disease: A case-control study in China. Neurology, 1989; 39: 660-4.
18. Schoenberg BS, Anderson DW, Haerer AF: Prevalence of Parkinson's disease in the biracial population of Copiah Country, Mississippi. Neurology, 1985; 35: 841-5.
19. Tandberg E, Larsen JP, Nessler EG, Riise T, Aarli JA: The epidemiology of v in the country of Rogland, Norway. Mov Disord, 1995; 10: 541-5.
20. Mayeux R, Marder K, Cote LJ, et al: The frequency of idiopathic Parkinson's disease by age, ethnic group and sex in northern Manhaten., 1988-1993. Am J Epidemiol, 1995; 142: 820-7.
21. Baldereschi M, Carlo A Di, Rocca WA, et al: Parkinson's disease and parkinsonism in a longitudinal study. Neurology, 2000; 55: 1358-63.
22. Al-Shekhli K, Abdul-Zehra IK: Clinical features and primitive reflexes in Parkinson's disease, thesis 1998.

23. Schoenberg BS, Osuntokun BO, Adeuja AO, et al: Comparison of the prevalence of Parkinson's disease in Black population in the rural United States and in rural Nigeria. Neurology, 1988; 38: 645-6.
24. Ho SC, Woo J, Lee CM: Epidemiological study of Parkinson's disease in Hong Kong. Neurology, 1989; 39: 1314-8.
25. Marder K, Tang MX, Mejia H, et al: Risk of Parkinson's disease among first-degree relatives. Neurology, 1996; 47: 55-60.
26. McCann SJ, Le Couteur DG, Green AC, et al.: The epidemiology of Parkinson's disease in an Australian population. Neuroepidemiol, 1999; 22: 405-8.
27. Wang SJ, Fuh JL, Teng EL, et al.: A door-to-door survey of Parkinson's disease in a Chinese population in Kinmen. Arch Neurol, 1996; 53: 66-71.
28. Wermuth L, Joensen P, Bunger N, Jeune B: High prevalence of Parkinson's disease in the Faroe Island. Neurology, 1997; 49: 426-32.
29. Sveinbjornsdottir S, Hicks A, Jonsson T, et al.: Familial aggregation of Parkinson's disease in Iceland. N Engl J Med, 2000; 343: 1765-70.
30. Bennet DA, Beckett LA, Murray AM, et al.: Prevalence of parkinsonian signs and associated mortality in a community population of older people. N Engl J Med, 1996; 334: 71-6.
31. Bharucha NE, Bharucha EP, Schoenberg BS: Pilot survey of the prevalence of neurological disorders in the parsi community of Bombay. Am J Prev Med, 1987; 3: 293-9.
32. Smagiassi A, Mutti A, DE Rosa A,DE palma G, Negrotti A, Calzetti S: A case-control study of occupational and environmental risk factors for Parkinson's disease in the Emilia-Romagna region of Italy. J Neurol Sci ,1997; 143: 157-62.
33. MacDonald BK, Cockerell OC, Shorvon JW: The incidence and lifetime prevalence of neurological disorders in a prospective community- based study in UK. Neurology, 2000; 123: 665-76.
34. Woolson RF: Statistical methods for the analysis for biochemical data. New York, Robert F. Woolson Ed., John Willy & sons, Inc. 1987; p.p. 14-185.
35. Daniel WW: Biostatistics: A foundation for analysis in the health sciences. John Willy & sons, Inc. 1987; ch 10: p.p. 343-56.
36. McGeer L, Itagaki S, Akiyama H, McGeer EG: Rate of cell death in parkinsonism indicate active neuropahtological process. Ann Neurol, 1988; 24: 574-6.
37. Kurtzke JF, Murphy FM: The changing pattern of death rates in parkinsonism. Neurology, 1990; 40: 42-9.
38. Gorell JM, Johnson CC, Rybicki BA, Peterson EL, Richardson RJ: The risk of Parkinson's disease with exposure to pesticide, farming, well water, and rural living. Neurology, 1998; 50: 1346-50.

AN EVALUATION OF HAEMOGLOBIN DETERMINATION USING SODIUM LAURYL SULFATE

Saad Sh. Mansoor[1] *FRCPath*, **Raad J. Musa**[1] *MSc, FICMS*, **Mohammad J. Kudher**[2] *FICMS*

Abstract

Introduction: Various methods for the quantitative determination of Hb have been developed during the last decade, based on chemical and physical principles of Hb, gasometry or spectrophotometry.

Methods: Spectrophotometric measurement of Hb as HiCN was carried out by the ICSH recommended reference method and by cyanide free (sodium lauryl sulfate) reagent. Measurements were carried out in 347 venous blood collected into K_2EDTA anticoagulant. Normal samples were obtained from volunteers and other samples were obtained from patient specimens in routine diagnostic service. The latter included Leukaemia with high WBC count, Lipaemia, Cord blood, other abnormalities (uraemia, jaundice thalassemia minor and major) and methaemoglobinemia. The Hb value then determined by using HiCN method and SLS method.

Results: Maximum absorbance is at 535 nm, the conversion time of Hb to SLS-Hb was rapid of less than 15 seconds. The freshly prepared SLS-Hb was stabile in the first 120 minutes after dilution. Haemoglobin converts almost instantaneously and there is a direct relationship of absorbance to Hb concentration over awid range of measurements (r = 0.999).

Conclusion: its reliability is equal to that of HiCN method with routine blood specimens, but slightly more reliable than HiCN method when there is interference by lipaemia. There is no significant difference in measurements on samples containing HbF (r = 0.995). It measures methamoglobin with correlation coefficient (r) of 0.999. It has a major advantage in that the reagent is non-hazardous compound.

Keywords: Haemoglobin, Sodium lauryl sulphate

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Introduction

Haemoglobin is a chromoproteid[1]. It is calculated to have a molecular weight of 64 458[2]. Each red cell contains approximately 640-million haemoglobin molecule[3]. Haemoglobin possess certain chemical and physical properties, which may be used in estimating its concentration in blood[4].

The depth of red color of blood is directly proportional to the concentration of iron and the concentration of Hb present. The red color of blood, therefore may be compared with that of various preparation used as standards and the concentration of Hb determined[4].

[1]Dept. of Pathology, College of Medicine, AL Nahrain University, [2]Al Kindy General Hospital, Ministry of Health.

Address correspondence to Dr. Raad Jaber Musa

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Various methods for the quantitative determination of Hb have been developed during the last decade, based on chemical and physical principles of Hb, gasometry or spectrophotometer.

The cyanmcthaemoglobin (HiCN) has been accepted worldwide during 1953-1963[5]. In addition, it's recommended by the International Committee of Standardization in Haematology (ICSH) in 1965 and 1967, due to the accuracy and stability of result[6-9]. However, the presence of potassium cyanide (KCN) and potassium ferricyanide (K_3-Fe $(CN)_6$) in the reagents has raised problems of laboratory and environmental pollution, and may constitute a potential toxic hazard[8].

Oshiro et al. (1982) developed cyanide free method of Hb determination that it based on a low toxicity compound sodium lauryl sulfate (SLS), a surfactant[10]. Haemoglobin is rapidly converted into SLS-Hb (10 seconds). The majority of Hb

derivatives encountered in clinical practice are converted to SLS-hemichrome complex using this method. Therefore, unlike others, this method does not need oxidative reagents and does not generate toxic wastes such as KCN and NaN3 which cause environmental pollution[11]. Therefore, the aim of this study was to evaluate sodium lauryl sulfate method for determination of haemoglobin in routine blood specimens.

Materials and Methods

This study was conducted at AL-Kadhimyia General Hospital, during the period from 4th of June 2002 till December 2002.

Spectrophotometric measurement of Hb as HiCN was carried out by the ICSH recommended reference method and by cyanide-free (sodium lauryl sulfate) reagent.

Measurements were carried out on 347 venous blood collected into K2EDTA anticoagulant (1.5mg/ml blood). Normal samples were obtained from volunteers and other samples were obtained from patients specimens in the routine diagnostic service. The latter included:

1- Leukaemias with high leukocyte counts (12 samples).
2- Lipaemias (15 samples).
3- Cord blood (35 samples).
4- Methaemoglobin (35 samples), was produced by exposing normal blood to sodium nitrate (2ml of blood to 0.1ml of sodium nitrate)[12].

The Hb value then determined by using two sets of reagents:

1. Drabkins (cyanide-ferricyanid solution) by using haemoglobincyanid (HiCN) method
2. New cyanide-free (sodium lauryl sulfate SLS) reagent (2g of SLS, 2.5g of disodium EDTA and one liter deionized distilled water).

Two types of Drabkins solution were used one supplied by Randox and other by Iraqi manufacturer; Hb value was determined by these reagents and means value then calculated.

<u>*1. Reagents and instruments:*</u>

1- Drabkins (cyanide-ferricyanid) solution one supplied by Randox and the other by Iraqi manufacturer.
2- HiCN standard so recommended by ICSH.
3- HiCN standard (Iraqi manufacturer).
4- SLS reagent (2 g of SLS, 2.5g of EDTA in one liter deionized distilled water).
5- Spectrophotometer.
6- Automated pipette.
7- Glass tube.

<u>*2. Methods:*</u>

<u>A) HICN method</u>[5]

Twenty microliter of EDTA blood were added to 5 ml of diluent (Drabkins solution), stopper the tube containing the solution & invert it several times, allow to stand at room temperature for about 5–15 minutes to ensure the completion of the reaction , the solution of HiCN is compared with reagent blank in spectrophotometer at 540 nm wave length in which the absorbance is read and the results obtained from stander curve , in which the stander curve prepared by measurement of HiCN reference solution (standard) by the same spectrophotometer as is to be used for the subsequent hemoglobinometry. The absorbance of the solution (standard) against blank of cyanide-ferricyanide reagent. We make reading with the same standard solution diluted with the reagent (Drabkins) 1 in 2 ,1 in 3 ,1 in 4,…etc. translate the Hb value of the solution into terms of g/l. Plot the reading on linear graph paper using arithmetical scales, with absorbance as ordinates (vertical scale). The point should fit a straight line that passes through the origin; this provides a check that the calibration of spectrophotometer is linear (assuming that the standard has been correctly diluted). We make two standard curve one for randox reagent & other for Iraqi manufactured reagent in which each one has it standard[14].

We taken the mean of duplicated measurement of each sample and tabulated

in order to be compared with the new method (SLS).

B) SLS METHOD[10]

Twenty microliter of fresh blood with EDTA was added into 3 ml of SLS reagent & read the absorbance within 20 second on spectrophotometer at 535nm wavelength.

Thus for preparing a calibration graph or standard graph it is necessary to adopt a two–stage procedure using fresh blood as an intermediate reference preparation, the Hb value of which must first be established by the HiCN method. For this purpose blood collected into APD or ACD anticoagulant is suitable for 2-3 weeks if stored at 4 c providing it remain sterile. A standard sample will maintain its assigned value for several months, especially if stored frozen.

After established of Hb value by HiCN method we make dilution by SLS reagent 1 in 2, 1 in 3, 1 in 4…. etc. Plot the reading into linear graph paper using arithmetical scale, with absorbance as ordinates (vertical scale). The points should fit astriaght line that pass through the origin. From the standard curve, we calculate the Hb value and plot in table.

Results

1. Spectrophotometry:

a. Conversion Time:

Total conversion to Hb-SLS was extremely rapid-less than 15 seconds required to obtain an initial measurement on the spectrophotometer.

b. Stability:

In freshly prepared solution of Hb-SLS kept at room temperature no significant difference were seen in absorbance at 535nm on repeated measurements during the first 120 minutes after dilution (Figure 1).

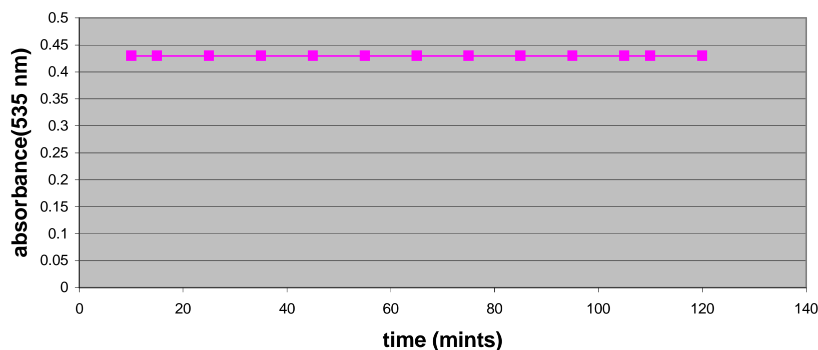

Figure 1: Haemoglobin SLS colorization

2. Technical Assessment:

a. Linearity:

Using a sample of fresh normal blood a series of 10 dilutions were made of packed red cells in platelet poor plasma. 2 replicate analyses were performed by spectrophotometer on each dilution and the mean results were plotted. (Table 1 and figure 2), with significant correlation (r = 0.999).

Table 1: The effect of dilution on measurement of SLS-Hb

Hb(g/l)	Slope	Intercept	Correlation coefficient(r)
SLS-Hb method	2.174	2.323	0.999

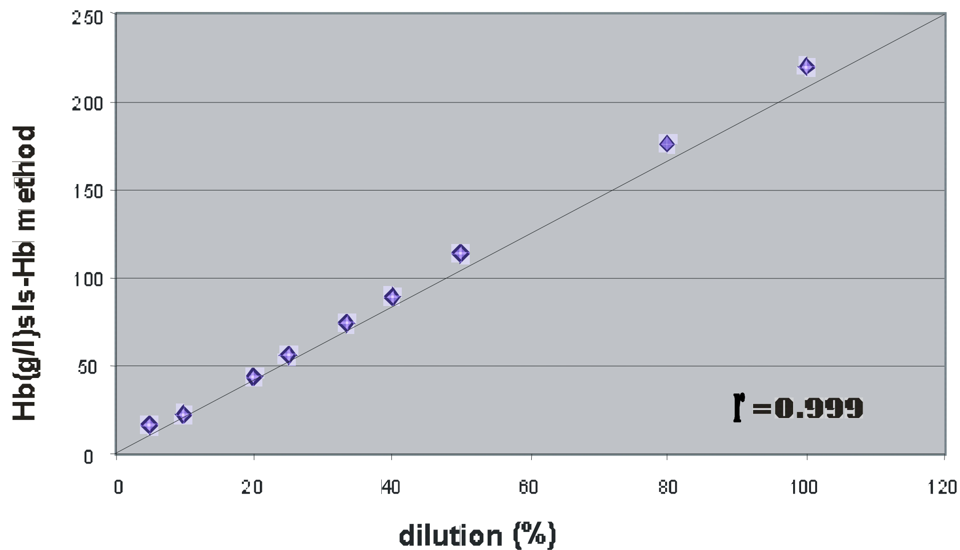

Figure 2: Showing the effect of dilution on measurement of SLS-Hb (linearity)

b. Comparability:

Results on 250 samples are shown in (Table 2 and figure. 3). Mean Hb (g/l) of 250 samples by SLS-Hb method was 116.9 ±31.05 (31-162), while the mean (g/l) by HiCN method was 116.84 ± 31.13 (31-162), with significant correlation (r = 0.998).

Table 2: Comparison of measurement of haemoglobin by SLS-Hb method and routine HiCN method

Method	No. of samples	*Mean / SD (g/l)*	Min (g/l)	Max (g/l)	Correlation (r)
Hb-SLS Method	250	116.9 ± 31.05	31	162	0.9983
HiCN Method	250	116.84 ± 31.13	31	162	

Figure 3: Comparison of measurement of haemoglobin by SLS-Hb method and routine HiCN

3. Interfering Substances:

a. Leukocytosis:

The effect of high leukocyte is illustrated in (Table 3 and figure 4). The differences from the reference method carried out in washed samples.

Table 3: The effect of WBC on measurement of Hb by SLS-Hb method and HiCN method

Method	No. of samples	Mean WBC count/cm	Hb(g/l) mean+SD	min (g/l)	max (g/l)	% Difference from Reference method (washed RBC)
SLS-Hb	12	63416.66	80.25±28.04	38	120	0.11%
HiCN	12	63416.66	80.66±27.81	39	120	0.62%
Hb after washed RBC	12	20583.33	80.16±30.51	37	125	

Note ;t = 0.890 p = 0.392 (non-significant)

b. Lipaemia:

The Hb on 15 selected samples were measured as SLS and as HiCN on whole blood and on spun plasma. Plasma reading were subtracted to obtain turbidity corrected measurements (Table 4).

C. Haemoglobin F:

Thirty-five specimens of neonatal (cord) blood were measured by SLS method and HiCN method, comparison of SLS and HiCN is show in (Table 5 and figure 5). Mean (g/l) of Hb by SLS method was 158.62 + 21.47 (110-187), while mean (g/l) by HICN was 158.85 + 59 (110-189), with significant correlation (r = 0.995).

4. Haemoglobin Derivatives:

Haemoglobin concentration was measured on blood samples by spectrophotometery as HiCN and Hb-SLS after converting proportion of Hb to methaemoglobin, (Table 6 and figure 6). Mean of Hb (g/l) by SLS method was 115.254+33.01 (36-163), while mean by HiCN was 115.08 + 32.74 (35-161), with significant correlation (r=0.999).

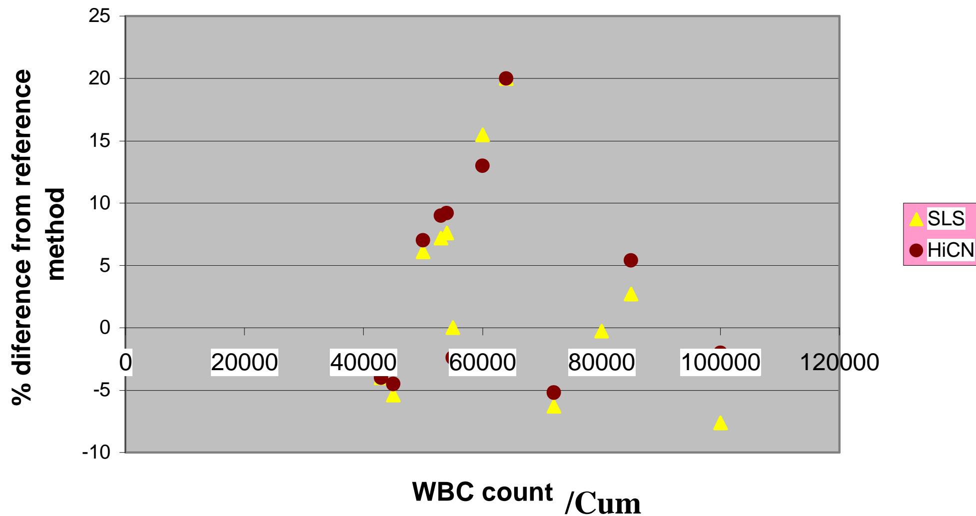

Figure 6: Effect of WBC on measurment of Hb by SLS-Hb method and HiCN method

Table 4: Mean ± SD of Hb (g/l) by SLS-Hb method and HiCN method on lipaemic samples pre and post correction

Samples	SLS – method (g/l)		HiCN method (g/l)	
	Direct	Corrected	Direct	Corrected
1	139	131	133	133
2	74	71	75	71
3	88	80	89	82
4	72	70	75	74
5	99	94	99	94
6	110	90	113	97
7	75	73	78	79
8	140	136	148	139
9	96	90	98	92
10	103	101	105	102
11	144	140	147	143
12	65	63	68	65
13	102	100	105	102
14	99	91	100	93
15	120	115	124	117
Mean±SD	101.73±25.35	96.26±24.62	103.8±25.44	98.86±24.49

Note; t-test for direct (SLS method) Vs direct (HiCN) = -2.77, p= 0.015 (significant)
T-test for direct Vs corrected = for (SLS) method t-test= -4.591, p=0.0001; for (HiCN) method t-test= 4.606, p= 0.0001

Table 5: The relationship between SLS-Hb method and HiCN method in measurement of cord blood (Hb F)

Method	No. of samples	Mean∣SD (g/l)	Min (g/l)	Max (g/l)	Correlation coefficient(r)
SLS-Hb method	33	158.62±21.47	110	187	r=0.9956,
HiCN method	33	158.85±21.59	110	189	p=0.0001

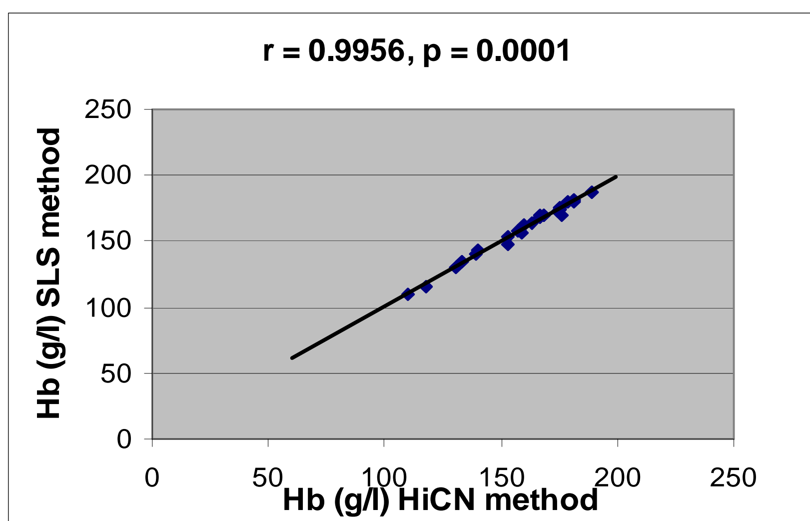

Figure 5: Comparison of measurement of HbF by HiCN method and SLS-Hb method

Table 6: The relationship between SLS-Hb method and HiCN method in measurement of methaemoglobin

Method	NO. of samples	Mean∣SD (g/l)	Min (g/l)	Max (g/l)	Correlation coefficient(r)
SLS-Hb	35	115.25±33.01	36	163	0.999
HiCN	35	115.08±32.74	35	161	

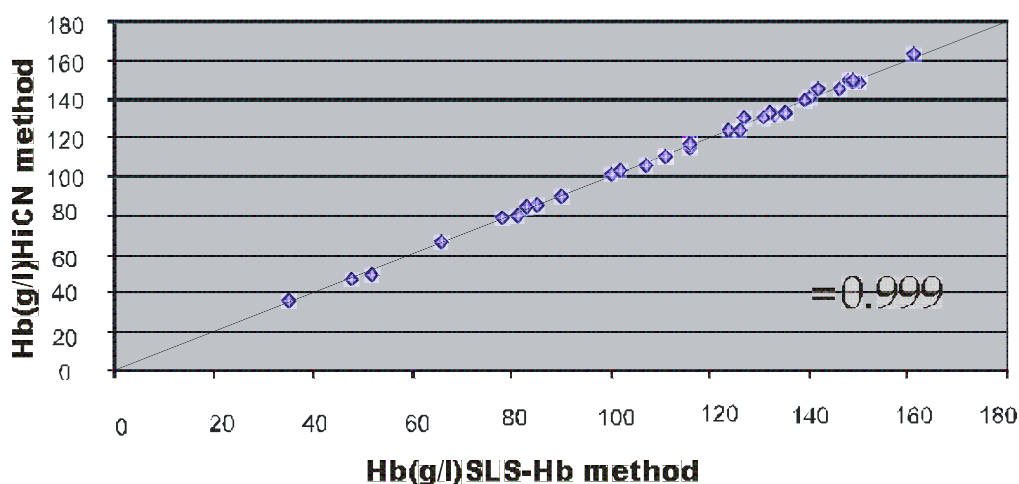

Figure 6: Comparison of measurement of methaemoglobin By SLS-Hb method and HiCN method

Discussion

The ideal method for haemoglobinometry should meet the following requirements:

1. Directly referable to a stable reference standard.
2. Immediate reaction between reagent and blood for total conversion of haemoglobin.
3. Perfect correlation between absorbance at an appropriate wavelength and haemoglobin concentration.
4. Stability of converted haemoglobin for at least several hours.
5. All haemoglobin derivatives are converted.

Reagent is non-toxic and does not affect the apparatus in which it is used in the ICSH recommended haemiglobincyanide (HiCN) method haemoglobin is converted by means of a ferricyanide reagent based on Drabkin's reagent[13]. This method, which has been universally accepted, meets the most important criteria of being linked with a stable standard, the international haemoglobincyanide reference standard[14]. This standard has a stability of at least six years.

The disadvantage of the HiCN method is that the reagent contains cyanide, and although this is used in a harmless amount for each assay, there is potential toxic risk where large volumes are discharged as waste: in some countries, this is regarded as a health hazard, which must be controlled in accordance with complex health and safety regulations. It is thus desirable to use a non-toxic alternative in routine practice[13].

Several alternatives are available[12], especially oxyhaemoglobin (HbO_2) using ammoniated water as reagent[15], but the product is unstable, and the method is unreliable in the presence of carboxyhemoglobin, methenoglobin, or sulfhemoglobin[16]. Nor do any other established methods meet the requirements set out above. It has been suggested that a better method might be to convert haemoglobin to a sulfate derivative by means of sodium Laury sulfate[11], this is a non-toxic substance[13]. It has now been developed as a commercial reagent by to a Medical Electronics for use in their automated haematology analysers and a preliminary report on its usefullness has been published[17]. This present study was undertaken to assess its use for haemoglobinometry in general.

The conversion time was extremely rapid less than 15 seconds. Lewis (1991) found the conversion time was less than 13

seconds[13], this was documented by this study. Similar results were also described by Karsan et al (1993)[11].

Repeated measurement of freshly prepared Hb-SLS during the first 120 mints after dilution shows no significant differences, (Figure 1). These findings were consistent with the findings of Lewis (1991) whose found that repeated measurements of freshly prepared solution of Hb-SLS during the first 2 hr, after dilution shows no significant differences.

Correlation coefficient (r) was 0.999 (Table 1and figure 2), in which there were some instrument differences, but no significant discrepancy for linearity. Lewis (1991) found that there was a direct relationship of absorbance to Hb concentration over awid range of measurements with (r) of 1.000[13].Similar results were encountered in this study.

Mean Hb (g/L) by SLS method was 116.9 $\pm$ 31.03 while mean Hb (g/L) by the HiCN method was 116.84 $\pm$ 31.13, with correlation coefficient of 0.998, (Table 2 and figure 3). Lewis (1991) found that there is a strong correlation between HiCN method and SLS method in measurement of Hb on routine blood specimens with a correlation coefficient of 0.998[13]. This was documented by this study. The same findings were also reported by Tsuda, Ntatsumi (1998) with correlation coefficient of 0.998[10]. The effect of high leucocyte count is illustrated in table 3 and figure4).

The differences from reference method carried out on washed red cells samples[18], showed no significant difference in measurement of Hb by SLS method and HiCN method. The findings of Lewis et al (1991) were not confirmed in this study, this may be due to the use of washed RBCs indicated the measurement of Hb by SLS method is less affected than HiCN method by the leucocytes[13].

The findings of Lewis (1991) did not confirmed by this study, this may be due to use washed RBC rather than centrifuged / filtered samples, because of unavailability of filters in Iraq.

Mean Hb (g/L) by SLS method was (101.73 $\pm$ 25.35 uncorrected and 96.26 $\pm$ 24.62 corrected), and by HiCN method was (103.8 $\pm$ 25.44 uncorrected and 98.86 $\pm$ 24.49 corrected) (Table 4). This findings was consistent with those of Lewis (1991) who founds that the SLS method was slightly more reliable on measurement of Hb when there's interference substance like lipaemia or WBC[13]. This was confirmed by this study.

Mean Hb (g/L) by SLS method was 158.62 $\pm$ 21.47 and by HiCN method was 158.85 $\pm$ 21.59, with correlation coefficient (r) of 0.995. There was no significant difference between SLS method and HiCN method on measurement on samples containing HbF (Table 5 and figure 5).

Lewis found that there was no difference on measurement of sample containing HbF by SLS and HiCN, with correlation coefficient of 0.993[13]. Similar results were encountered in this study.

Mean of Hb (g/L) by SLS method was 115.25$\pm$33.01 and by HiCN method was 115.08 $\pm$ 32.74 with correlation coefficient of 0.999 (Table 6 and figure 6). The findings of Lewis et al (1991) were confirmed in this study[13] in which there was no significant difference in measurement of methaemoglobin by SLS and HiCN methods[13].

Conclusions

The measurement of Hb by sodium lauryl sulfate showed:

1. It is non-toxic substance.
2. Hb converts almost instantaneously.
3. There is a direct relationship of absorbance to Hb concentration over a wide range of measurements.
4. It is stable for 2 hours without significant effect.
5. It is reliability is equal to that of haemoglobinocyanid method.
6. Slightly more reliable method when there is interference by lipaemia.
7. Measures Hb containing high concentration of HbF and methaemoglobin.

Recommendations

1. Haemoglobin sulphate (SLS) method provides a reliable routine method with the advantage of non-hazardous reagent.
2. We recommend another study using sodium lauryl sulfate in automated analysers.

References

1. ICSH: Recommendations for reference method for haemoglobinometry in human blood (ICSH standard Ep 6/2: 1977) and specification for international haemoglobinocyanide reference preparation (ICSH standard Ep 6/3: 1977). J Clin Pathol, 1978; 13: 139-43.
2. Recommendation for reference method for haemoglobinometry in human blood (ICSH standard 1986) and specifications for international haemoglobincyanide reference preparation (3rd edition). Clin Lab Haematol, 1987; 9: 73-9.
3. Hoffbrand AV, Pettit JE, and Moss PAH: Essential Haematology, 4th edition, Blackwell Science, London, 2001; p.p. 14-7.
4. Sunderman FW, Fate RPM, Fadyen DAM, Steven GF, and Copeland BE: Symposium on Clinical Haemoglobinometry. Am J Clinical Pathol, 1953; 23: 519-98.
5. Sunderman FW: Status of clinical haemoglobinometry in the United States. Am J Clin Pathol, 1965; 93: 9-15.
6. Wolf HU, Lang W, and Zarder R: Alkaline haematin D-575, anew tool for the determination of haemoglobin as alternative to the cyanhaemoglobin method. II. Standardization of the method using pure chlorohaemin. Clin Chim Acta, 1984; 136: 95-104.
7. Severini G: The extinction coefficient of human hemiglobincyanide as determined by nitrogen analysis. Clin Chem Acta, 1966; 14: 376-7.
8. Hamaguchi Y: Overview of the principles of Sysmex's Haemoglobinometry. Sysmex J Internat, 1999; 7(1): 45-51.
9. Matsubara T: Evaluation of the internationally standardized method for haemoglobinometry. Clin Chem Acta, 1969; 23: 427-30.
10. Oshiro I, et al.: New manual and automatical method of haemoglobin determination by using sodium lauryl sulfate (SLS). Clin Pathol, 1981; 29: Abstract.
11. Oshiro I, Tuke Naka T, and Maeda J: New method for haemoglobin determination by using sodium laury sulfate (SLS). Clin Biochem, 1982; 15(2): Abstract.
12. Dacie JV, and Lewis SM: Practical Haematology, 8th edition, Churchill Living Stone, Edinburgh, 1995; p.p. 49-54.
13. Lewis SM, Garvey B, Manning R, Sharp SA, and Wardle J: Lauryl sulfate haemoglobin: a non hazar dous substitute for HiCN in haemoglobinometry. Clin Lab Haemat, 1991; 13: 279-90.
14. ICSH: Recommendation for reference method for haemoglobinometry in human blood. Clin Lab Haematol, 1986; 9: 73-9.
15. Dacie JV, and Lewis SM: Practical haematology, 7th edition. Churchill Living hstane, Edinburgh, 1991; p.p. 37-41.
16. Karsan A, Maclaren I, Conn D, and Wadsworth L: An evaluation of haemoglobin determination using sodium lauryl sulfate. Am J Clin Pathol, 1993; 100(2): 123-6.
17. Fujiware C, et al.: The reagent sulfolyser for Hb measurement by haematology analyzers Sysmex J Internat, 1990; 13: Abstract.
18. Harmening D: Modern Blood banking and transfusion practice, 2nd edition, F. A. Davis Company Philadelphia, 1989; p.p. 65.

PITUITARY ADENOMA: RETROSPECTIVE STUDY AND ANALYSIS OF FACTORS THAT AFFECT THE OUTCOME OF SURGICAL TREATMENT

Samir H. Abood *FRCS*, **Sarmad A. Ibraheem** *FICMS*, **Abdul-Amir Jasim** *FICMS*

Abstract

Background: Pituitary adenomas are benign epithelial tumors, representing about 10% of intracranial tumors. They present as visual or hormonal disturbances or both. Attention to details in the treatment of theses benign tumors can't be overemphasized in order to achieve best results.

Objective: The aim of this work is to review the clinical and radiological features of these relatively common tumors, and identify factors that affect the outcome of surgical treatment and recurrence.

Methods: This study was carried out retrospectively by reviewing the medical records of all patients with pituitary adenomas treated in the period of four years, and collecting clinical, radiological, laboratory, and surgical data. Follow up of 1-4 years was obtained on all patients.

Results: There were 55 patients (31 male and 24 female) the median age was 37 year. The main presenting symptoms were visual deterioration in 49%, symptoms of endocrine disturbance in 35.3%, headache in14.5%, behavioural changes in 1.8% and seizure in 1.8%. The most common hormonal disturbance was hyperprolactinemia (32.7%). Lateral skull x-ray showed grade III sellar enlargement with erosion of the dorsum sellae in 87.2%. Brain CT scan showed isodense mass in 76% of cases, with suprasellar extension in 82.6% and contrast enhancement in 85.7%. The operative approaches were right subfrontal craniotomy in 83.6% and tarns-sphenoidal (sub labial) approach in 16.3%. Intracapsular (subtotal) removal was achieved in 70.9%, while capsular (total) removal was achieved in 29.1%. Tumour recurred in 3 patients; two of them had postoperative radiotherapy and all had originally aggressive tumors.. There were 2 cases of post radiation glioma.

Conclusion: The results of the study were similar to those published. Pituitary adenoma is usually benign relatively avascular soft in consistency with characteristic clinical and radiological features. Unless associated with advanced optic atrophy with visual loss there is dramatic improvement of optic nerve function following surgery. Transient diabetes insipidus is the commonest postoperative complication mainly following trans-sphenoidal approach. The only factor that enhances recurrence is tumour aggressiveness. Postoperative radiotherapy is only indicated for aggressive invasive pituitary adenoma.

Key words: Pituitary, Adenoma, Outcome.

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Introduction

Pituitary adenomas are common benign epithelial neoplasms that are composed of and derived from adenohypophysial cells. They represent about 10% of intracranial tumors[1]. In most cases they are histologically benign, slow growing small neoplasms confined to the sella tursica. Some however grow faster, invading surrounding tissues, and cause local symptoms such as visual disturbances, headache, and compression of non tumorous pituitary tissue, resulting in varying degrees of hypopituitarism[2].

Pituitary adenomas can be classified according to their size, radiolographic appearance, endocrine function, morphology, and cytogenesis. Neurosurgeons frequently classify pituitary adenoma on the basis of size

Section of Neurosurgery, Dept. Surgery, College of Medicine, Al-Nahrain University
Address correspondence to Dr. Samir H. Abood. E-mail: dr_samirabood@hotmail.com, samirabood@yahoo.com
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and invasiveness, as determined by imaging studies (MRI, CT scan and X-ray films of the skull), with respect to the size, microadenomas are smaller than 1cm in greatest diameter, and macroadenomas are larger than this[1].

Microscopically pituitary adenomas are generally monomorphous proliferation, consists of a single relatively uniform, homogenous staining cell population with no acinar arrangement, surrounded by a condensed rim of non tumorous pituitary tissue and its condensed reticuline network, which collectively constitute the adenomas pseudocapsule. Pituitary adenomas can be classified into acidophilic, basophilic, and cromophobe adenomas according to their staining property with hematoxylin-eosin preparation[1].

Pituitary adenomas present either as hormonal dysfunction, or as visual disturbance due to chiasmal compression. The management of pituitary adenoma is challenging, as complete surgical removal is sometimes difficult to achieve and is usually associated with substantial morbidity and mortality, on the other hand, other therapeutic modalities such as radiosurgery and radiotherapy are of limited efficacy on their own, and are used as adjuvant to surgical treatment[3-6].

Methods

The material of this study included 55 consecutive patients with pituitary adenoma surgically treated during the period of January1997 to April 1999 in four neurosurgical centers in Baghdad, Al-Kadmya hospital (T), the neurosurgical hospital, the neurosurgical dep. Medical City (T) and the Nursing home, Medical City. The patient's files were reviewed and all biographic, clinical, radiological, and treatment data uniformly collected according to previously designed data sheet. The collected data included: patients age (according to decades), sex, duration of illness and the presenting symptom.

The physical signs documented include visual acuity and visual fields, presence of optic atrophy on fundus examination, evidence of endocrine dysfunction as acromegaly, hyperprolactinemia and the development of secondary sexual characteristics. Serum hormonal level (prolactin, growth hormone, thyroxine, cortisol, follicular stimulating hormone (FSH) and leutenizing hormone (LH) was recorded. On lateral skull X-ray the findings were classified into grades as follow[7].

G_{I}: Normal size sella with focal erosion

G_{II}: Enlarged not eroded sella

G_{III}: Enlarged eroded, ballooned or double floor sella

G_{IV}: Completely destructed sella (Ghost sella)

On CT scan, MRI and MRA data such as extension of the tumor to the frontal, and temporal lobes and to the cavernous sinus as well as hydrocephalus was recorded.

Operative data were documented including the surgical approach (right sub frontal craniotomy or sub labial trans-sphenoidal approach). The choice of surgical approach depended on the presence or absence of extrasellar extension of the tumor. The extent of tumor excision whether total (capsular) or subtotal (intracapsular) resection, Outcome including complications, mortality, and Postoperative radiotherapy was recorded.

Follow-up notes were reviewed and the patient's clinical, endocrinological and radiological findings were documented. Tumor recurrence was defined as re-growth of tumor as appeared in the follow up CT scan and MRI or both, or recurrence of symptoms. The outcome of treatment was considered good if the patient is alive, having improved or stable useful vision, good school performance or is employed and poor

outcome if the patient condition did not fulfill one or more of the above criteria or is dead.

Results

The study included 55 patients (31 males and 24 females) the median age was 36 years. Figure I summarize the age and gender distribution. Most of the patients were in the forth decade. There is female preponderance in the 2nd and 3rd decades, male incidence increased in the 4th and 5th decades.

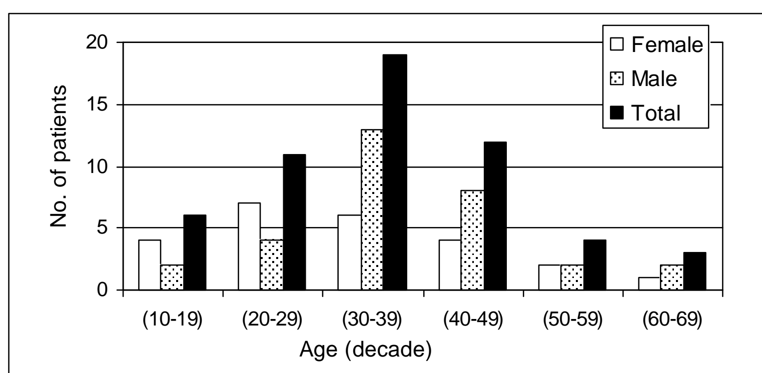

Figure 1: Age and Gender

Visual disturbance in the form of blurring of vision was the commonest presenting symptom (45.4%) followed by amenorrhea 18% and acromegalic feature 16.3% (Table 1). The one patient presented with diplopia as the first complain was diagnosed later to have had pituitary apoplexy.

Table 1: The presenting symptoms of pituitary adenomas

Symptom	No.	Percent
Visual disturbance (decreased visual acuity and field defect)	25	45.4
Headache (non specific)	8	14.5
Diplopia	1	1.8
Loss of vision	1	1.8
Impotence and loss of libido	1	1.8
Amenorrhea	10	18
Acromegalic features	9	16.3

Hyperprolactinemia, proved by hormonal assay, presenting as loss of libido and impotence in males and amerorrhea-galactarrhea in females was present in the majority of patients 72%, followed by excessive growth hormone secretion presenting as acromegaly or gigantism in 64% of cases (Table 2).

Table 2: Hormonal disturbances associated with Pituitary adenoma (25 patients).

Type of hormonal disturbance	No.	Percent	Female	Male	Percent
Acromegaly and gigantism	16	64	6	10	29
Hyperprolactinemia	18	72	6	12	32.7
Cushing's disease	1	4	1	-	1.8
Hypogonadism	1	4	-	1	1.8

Headache was the commonest symptom (90.9%). Progressive visual deterioration coming next in frequency (78%0, all of them had decreased visual acuity and 63.3% of patients had visual field defect usually in the form bitemporal hemianopia. Unilateral visual loss was detected in 27%, and primary optic atrophy was observed in 29% of patients. Papilledema was seen in four patients (7.2%) two of them had hydrocephalus. Diplopia was complained of in 4 patients (7.2%) three patients had pure sixth cranial nerve palsy and one patient had complete left sided ophthalmoplegia (Tables 3 and 4).

Table 3: Symptoms due to pressure effect of pituitary adenoma (55 patients)

Symptoms	No.	Percent
Headache	50	90.9
Visual field defect	43	78
Diplopia	4	7.2
Unilateral loss of vision	15	27.2
Behavioural changes	1	1.8
Seizure	1	1.8
Pituitary apoplexy	1	1.8

Table 4: Signs of space-occupying effect in relation to the total number of patients with pituitary adenomas

Signs	No.	Percent
Decreased visual acuity	43	78.1
Visual field defect (temporal hemianopia)	35	63.3
Primary optic atrophy	16	29
Papilledema	4	7.2
Ocular nerve palsy	4	7.2
Trigeminal nerve affection	1	1.8

On skull X-ray the majority of patients had sellar enlargement (94.5%), while normal size sella found in only (5.4%). Grade III sellar enlargement was the commonest finding (87.2%), (Table 5).

Table 5: Skull X-ray findings in patients with pituitary adenomas (55 patients)

Appearance	Grade	No.	Percent
Normal sella with focal erosion	I	3	5.4
Intrasellar calcification	-	1	1.8
Enlarged sella turcica	II → IV	52	94.5
Enlarged not eroded sella	II	1	1.8
Enlarged eroded sella	III	30	54.5
Enlarged double floor sella	III	3	5.4
Ballooned sella	III	15	27.2
Completely destructed sella (Ghost sella)	IV	3	5.4

Pituitary adenomas were isodense in the majority of patients (76%). Supra sellar extension was the commonest finding (82.6%), while no tumor extension was detected in 17.3%. - Hydrocephalus was seen in only two patients (4.3%). In contrast, in a study, which was performed in 35 patients, 85.7% had enhancing adenoma (Table 6).

Table 6: C.T. Finding in patients with pituitary adenomas (46 patients)

Finding	No.	Percent
CT without contrast		
1- Density	35	76%
- isodense	3	6.5%
- hypo dense	8	17.3%
- hyper dense		
2- Extension		
- suprasellar	38	82.6%
- lateral	13	28.2%
- anterior	1	2.1%
- no extension	8	17.3%
3- Ventricular dilatation (Hydrocephalus)	2	4.3%
CT with contrast (35 patients)		
1- Enhanced	30/35	85.7%
2- Not enhanced	5/35	14.3%

MRI and MRA were the most useful imaging modalities in evaluation they were performed in nine patients (Figures 2 and 3).

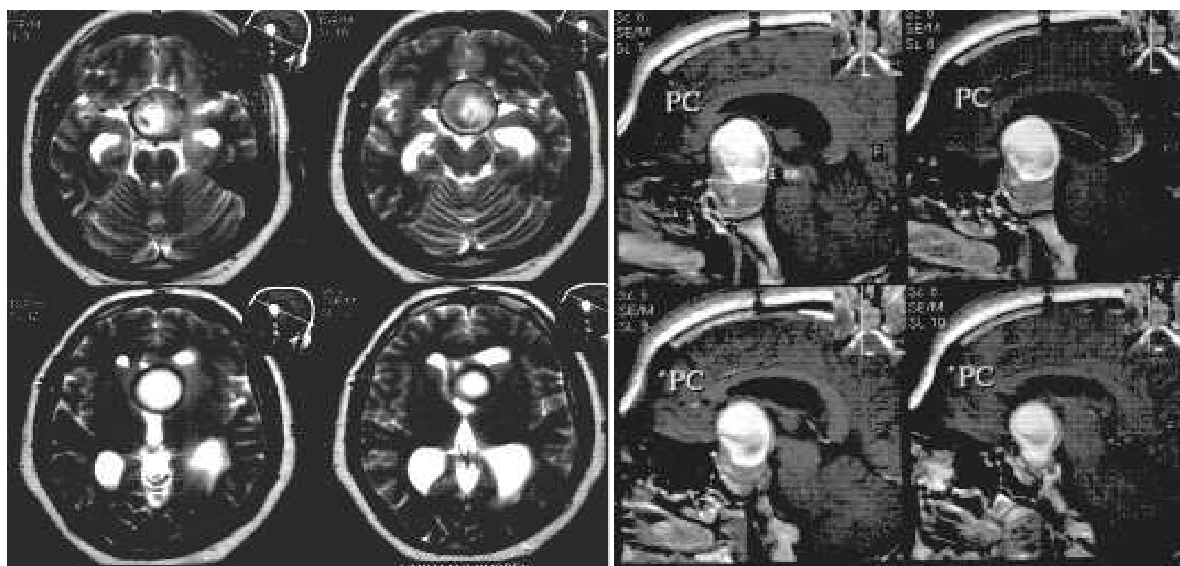

Figure 2: Pituitary adenoma, with suprasellar extention (MRI, Axial and sagital sections)

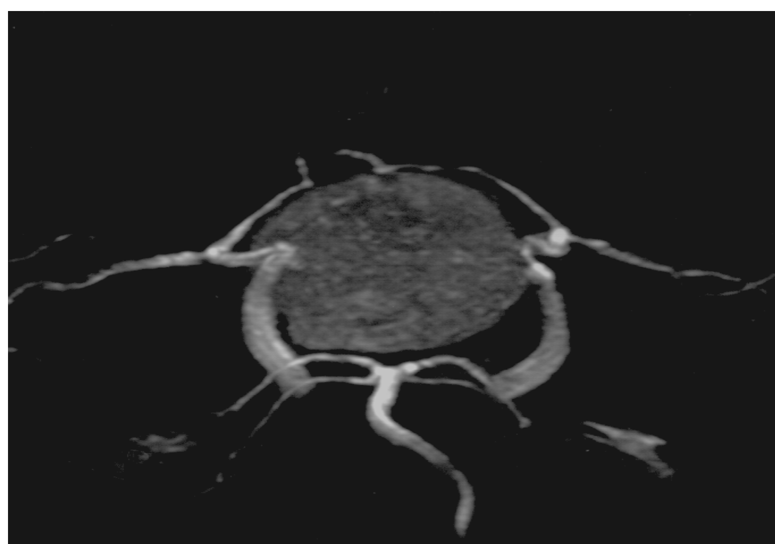

Figure 3: Pituitary adenoma (MRA)

Most of the adenomas were soft, non-cystic (Table 7). Chromophob adenoma was the most common (56.3%) followed by acidophilic adenomas (41.8%). Histologically (90.9%) of pituitary adenomas were benign, while aggressive (invasive) pituitary adenoma was seen in (9.1%) (Table 8).

Table 7: Texture and consistency of the adenoma

Texture and consistency	No.	Percent
Cystic	8	14.5
Non cystic (solid)	47	85.4
Soft	49	89
Firm	6	10.9

Table 8: Histopathological results of pituitary adenomas

Histopathology	No.	Percent
Acidophilic	23	41.8
Chromophob	31	56.3
Basophilic	1	1.8
Aggressive	5	9

Five patients had aggressive adenomas; all were acidophilic adenomas, firm, and highly vascular. All had suprasellar and lateral extension (Table 9).

Table 9: Staining, vascularity, texture and extension of aggressive pituitary adenomas

Histopathology	Vascularity	Texture	Extension
Acidophilic	High	firm, non-cystic	superior & lateral
Acidophilic	High	firm, cystic	superior & lateral
Acidophilic	High	firm cystic	superior & lateral
Acidophilic	High	firm, non-cystic	sup., ant., & lateral
Acidophilic	High	firm, non-cystic	superior & lateral

Dramatic improvement of the visual fields and acuity was documented in 25 of 30 patients (83.3%). Improvement in the cranial nerve deficit with diplopia was seen in (50%). Patients with sever visual impairment, and advanced optic atrophy did not show similar improvement, but no one got worse (Table 10).

Table 10: Post-operative visual improvement

Improving sings and symptoms	No.	Percent
Visual field and acuity	25/30	83.3
Diplopia and squint	2/4	50
Ptosis	1/1	100

Right sub frontal craniotomy was the approach used in 46 patients (83.6%) and trans-sphenoidal approach was performed in 9 patients (16.4%). Gross total resection of the tumor (capsular) was achieved in 29.1% (Figure 4) and subtotal resection (intracapsular) in 70.9%. Benign chromophobe adenoma was the commonest pathological finding, which accounted for 56.3%of cases, followed by acidophilic adenoma in 41.8%. While all aggressive pituitary adenomas were acidophilic and account about 9% (5 patients).

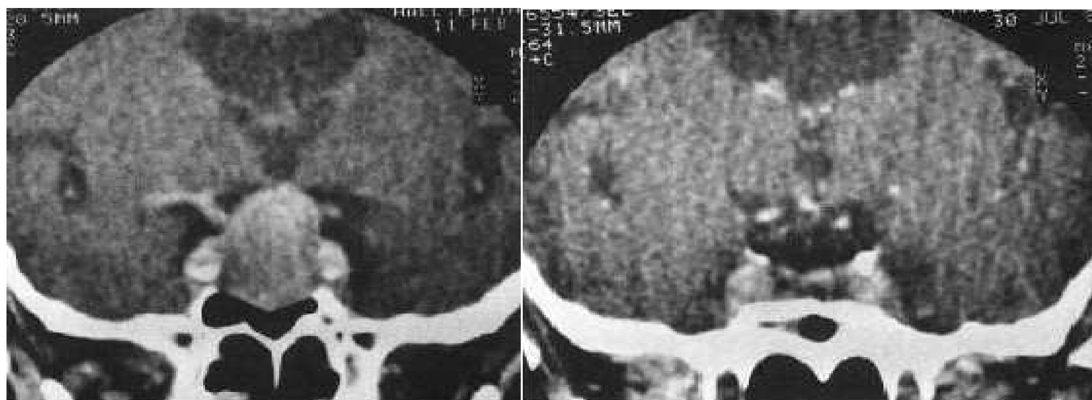

Figure 4: Brain CT scan (coronal sections) of pituitary adenoma showing suprasellar extension (Pre- operative Lt. and post operative Rt.)

All aggressive adenomas had postoperative radiotherapy (all of them did not complete their course) however three of them had recurrences after one year, there was no relation between recurrence and the type of adenoma or the extent of surgical resection. Early postoperative complications (Table 11) included diabetes insipid (DI) in 6 patients, 2 of them following trans-sphenoidal approach, and fortunately only one patient had permanent DI who need permanent antidiuretic hormone (DDAVP) treatment.

Table 11: Post-operative complications

Complication	Approach	No.	Percentage	Percentage from total
CS leak + meningitis	Transcranial	1/46	2.1	1.8
Infected wound	Transcranial	1/46	2.1	1.8
Diabetes Iinsipidus	Transcranial	4/46	8.6	10.9
	Trans sphenoidal	2/9	22.2	
Hypothyroidism	Transcranial	4/46	8.6	7.2
Unilateral anosmia	Transcranial	46/46	100	83.6
Hematoma	Transcranial	1/46	2.1	1.8
Recurrence	Transcranial	3/46	6.5	5.4
Post radiotherapy glioma	Transcranial (Previous op.)	2/46	4.3	3.6
Death	Transcranial	3/46	6.5	5.4

Recurrent CSF leak and meningitis was detected in one patient who transcranial surgery for aggressive pituitary adenoma. Wound infection was recorded in one patient. Hypothyroidism, proved by hormonal assay was recorded in 4 patients (7.2%). Intracerebral hematoma detected in one patient who died in the third postoperative day. Two other patients died one of them developed pulmonary embolism and the other had hypothalamic ischemia. Post radiotherapy glioma proved by histopathology was recorded in 2 patients from a total of thirty-seven who had radiotherapy (5.4%).

Discussion

In Large series of intracranial neoplasm (Burrow et al., 1981; Kovacs et al., 1986)[8,9], pituitary adenomas comprise about 10-15 percent of brain tumors. The true incidence in Iraq is unknown.

Regarding the sex distribution, pituitary adenomas are equally distributed between the sexes. From series (Minderman et al., 1994)[10] one notes a slight female predominates in prolactin hormone (PRL), thyroid stimulating hormone (TSH), and adrenocorticotropic hormone secreting adenomas, whereas male dominance was recorded in growth hormone (GH) secreting adenomas, and non-functioning pituitary adenomas. Equal sex distribution is found in older patients. In our study, slight male preponderance (56.3%) was recorded.

Pituitary adenomas are more apt to be found in specific age groups. They are tumors of the middle decades of life, although isolated examples have been reported in children with pituitary adenomas. One also notices that the average age in lower in acidophil adenomas than chromophobe adenomas[11-13]**.** The results in this study are very similar to the published figures.

Symptoms and Signs

The signs and symptoms of pituitary adenomas are produced by the local mass effect of the adenoma and by the systemic manifestations, which are related to the hormonal disturbances.

In this study non-functioning pituitary adenomas comprise about (54.5%), whereas functional pituitary adenomas were found in (45.5%). Sassolas et al., (1993), and Nedvidkova et al., (2000)[14,15] found non-functioning adenomas in only 40% of cases. This may be due new and more thorough hormonal analysis.

The signs and symptoms produced by the space occupying effect are due to the direct pressure or invasion of the adjacent parasellar structures and the optic nerves and chiasm.

We found the most common symptom was headache (90.9%), the cause of the headache is stretching of the diaphragma sellae, However many of the cases with headache, especially females with hormonal disturbances have psychological cause for their headache. Generally the reported incidence of headache is about 40%[15-17]. The headache is often non-specific, found primarily in the vertex as a dull ache, and does not change with position. In pituitary apoplexy however headache is sudden, severe and is accompanied by neck stiffness.

Visual deterioration was found in 78% of cases, in which decreased visual acuity was detected in all of them, while visual field defect (uni or bitemporal hemianopia) was found in (62.3%)of cases. Comparing the results with other series of Sassolas et al., (1993); Fajardo (1982), and Burrow et al., 1990[14.17,18], visual defect was reported in about 50-70% of patients, at the time of diagnosis. The higher incidence of visual deterioration probably indicates late presentation.

Primary optic atrophy was found in 29% of cases, whereas papilledema was detected in (12.7%) of patients, which is high compared with the reported incidence of 6% by Chang et al., (2000)[19]. However the

typical visual field defects seen in some of these patients with reported papilledema suggest that the impression made by junior residents was false.

The reported incidence of cranial nerve palsies other than the optic nerve is 5-17%. Usually the 3rd, 4th 6th and less commonly the 5th nerves are involved[11-13]. Generally cranial nerve palsies suggest lateral extension into the cavernous sinus, and is mostly associated with aggressive adenomas. The 4 patients who had cranial nerve palsies (7.2%) all had lateral extension and all were aggressive tumors.

Regarding epilepsy, seizures were reported in 1-10% of patients with pituitary adenoma. Most series[11,12,20] in this study it was noticed in one patient (1.8%), We could find no reason or association between epilepsy and the pathology or extension of the tumor.

Hypopituitarism, according to many series, was seen in a good proportion of patients. One important feature for example is Insufficient gonadotropic hormone secretion, which was found in 50-90% of patients (Bakay, 1950 and Arafah et al., 1986)[11,21] In this study only one patient was diagnosed to have hypogonadism due to deficient gonadotropic hormone (GnH) secretion, confirmed by hormonal assessment (1.8%). This lowly incidence of hypogonadism in males, is probably only apparent, due to denial of patient for social reasons, and failure on the part of physicians to asses this important clinical feature of pituitary adenomas.

Regarding functional pituitary adenoma, we found that hyperprolactinemia comprises 72% (32% from total number), by comparing our results with those of other studies (Visot et al., 2001, Davis et al., 1985 and Kleinberg, 1983)[22-24]. We found prolactin secreting adenomas comprising 25%. From this we expect that the higher percentage was not due to pure PRL producing adenoma rather than secondary hyperprolactinoma due to local mass effects causing amenorrhea - galactorrhea Syndrome primarily in females and loss of libido with impotence in males.

Growth hormone (GH) secreting adenoma detected in 64% of patients with functioning pituitary adenomas (29% from total) in which acromegalic features detected in all of them , whereas gigantism detected in only one patient (4%), which is exactly the same percentage found in other series. Insulin dependent diabetes mellitus (IDDM) detected in 62% of patients with Acromegaly, Compared with about 50% reported in literatures. Hypertension detected (37.5%) similar with other studies 36% (Jackson et al., 1999)[25].

Cushing's disease was found in 4% of functioning pituitary adenomas (1.8% from total), which is almost the same of other series (Tindall et al., 1985 and Sheithauer et al., 1986)[2,26]. This disease is rare, and because most of them are due to a microadenoma (80%) it rarely gives rise to other hormonal or compression symptoms.

In our study, no patient was found to have hyperthyroidism or excessive secretion of GnH, which indicate their rarity as seen in other series, TSH producing adenoma comprising about 1% (Martin et al., 1977, Black et al., 1984, Linfoot et al., 1979, and Post et al. 1980)[13,27-29].

Radiological investigations

The radiology of the sella tursica is an interesting subject when considering pituitary adenomas, in plane x-ray, non-functional pituitary adenoma enlarge the sella almost without exception. We had three patients in our study that had normal sized sella tursica (5.5%).With high resolution Brain CT scan, microadenomas can be correctly identified in 70% of cases, and the CT scanner was accurate in the detection of macroadenomas in 95% of cases. (Chang et al., 2000)[19], most of which highly enhances with contrast study.

In this study we found most of pituitary macroadenomas were isodense (76%), while they were hyperdense in 17.3% of patients. Good enhancement with contrast study was seen in 85.7% and by comparing these results with those of other studies we found it similar (Chakeres et al., 1990 and Leeds et al. 1977)[30,31].

Histopathology

On the basis of staining properties of the cell cytoplasm, pituitary adenomas were previously defined as one of three morphological entities, chromophobic, acidophilic and basophilic adenomas. Since the tincturial characteristics of the adenoma cells cannot be correlated reliably with the cell type, secretary activity or cytogenesis, this basis for classification is no longer considered useful (Kamal et al., 1995)[1].

In spite of what is said above, above, our histopathological labs continue to use the same type of morphological classification according to the staining property with hematoxylin and eosin, which revealed that chromophobe adenoma comprise about 56.3%, whereas acidophilic adenomas account for 41.8% and basophilic adenomas 1.3%. By comparing with other series (Wilson et al., 1978 and Tindall et al., 1995, found that chromophobe adenomas account for 75%, whereas acidophilic adenomas about 17%[32,33], this indicate that in our study chromophobe adenoma is much less, this could be due to the poor preparation of the histopathological section. Regarding basophilic adenomas, the result coincides with those of other series of 1-2%.

Macroscopically the consistency of pituitary adenomas is classified into either cystic (partial or complete) and solid. In this study, pituitary adenomas were cystic in 14.5% which is somewhat similar to those of other studies (18-20% by Asa et al, 1993)[34].

Regarding the texture, soft suckable pituitary adenoma was detected in 89%, which is almost the same compared with other series, (92% by Kamal et al., 1995)[1].

Aggressive invasive pituitary adenoma was detected in 9.1% of all types of pituitary adenomas, 50% of them were acidophilic.

Operative findings and surgical management

Regarding the optic chiasm, normal position of the chiasm was found in 89.1%, prefixed chiasm in 2.1% and postfixed chiasm in 8.6% of cases. By comparing with other studies, the percentage were 80%, 9% and 11% respectively (Tindall, 1995)[33], and 70%, 15% and 15% respectively (Albert et al., 1995)[35] which indicate approximately similar results. Regarding extension of the pituitary adenomas we detected that during operation the incidence of tumor extension was 86.9%. Whereas preoperative C.T scan reported 82.6% tumor extension. This indicates that C.T scan is not 100% accurate in the diagnosis of extension.

In the series of Wilson & Demsey (1978)[32], 48.4% pituitary adenomas were confined to the sella tursica, significant suprasellar extension was observed in 34.8%, infrasellar extension in 17.6% and both in 6.8% of cases. In this study, suprasellar extension was seen in 86.6%, whereas infrasellar extension in 22.2% of cases. The increase in the incidence of suprasellar extension may reflect a delay in the diagnosis, and the higher number of non-functioning pituitary adenomas.

As regard to the surgical outcome in both approaches, many series were reviewed regarding the mortality rate and these were ranging from 1.4 - 35% in transcranial subfrontal approach, and from zero to 1.5% following trans-sphenoidal approach (Tindall et al., 1986)[2]. In our study, 3 patients died (5.4%) all of them following transcranial approach, while there was no mortality following trans-sphenoidal approach.

Regarding patient's condition following the operation we found that 83.3%

of patients have improved vision, which is a good result compared with the reported improvement rate of 41-90%[11,19,36].

Tumor recurrence was recorded in 3 patients (6.5%) following transcranial approach, with no recurrence following trans-sphenoidal approach. The previous histopathological results of those with recurrent adenoma were of invasive character, and all had radiotherapy. The result is quite good compared with those registered from other series, which ranged between 6 and 11%, usually following transcranial approach of highly invasive adenoma (Linfoot, 1979; Ciric, et al, 1983; Domingue, et al., 1980; Faria, et al., 1982)[28,36-38]. Invasiveness was the only factor that was associated with recurrence, which raises question regarding the necessity and effectiveness of conventional radiotherapy. Postradiotherapy (glioma) was detected in 2 patients(3.6%), which is similar to other series (George, et al., 1995 and Fajardo, et al., 1982)[33,39].

Conclusions

1. Pituitary adenoma is usually benign relatively avascular soft in consistency with characteristic clinical and radiological features, commonly present in 4th decade.
2. Hyperprolactinenia was the most common hormonal disturbance, and visual deterioration was the most common presenting symptom.
3. Regarding the postoperative prognosis, unless associated advanced primary optic atrophy, there was dramatic improvement in the optic nerve function (83.3%), and other cranial nerves function (50%), which indicate that the primary lesion is due to the pressure effect rather than invasion.
4. Diabetes insipidus is the commonest postoperative complication especially following trans-sphenoidal approach (10.9%) and it is transient in most cases.
5. Tumor invasiveness was the only factor that was found to be associated with recurrence of pituitary adenoma.
6. Postoperative radiotherapy should be considered for invasive adenomas; however the risk of tumor genesis makes one think of an alternative treatment such as Gamma knife surgery.

References

1. Kamal Thapar K, Kovacs E, Horvath SL, Asa: Classifications and pathology of pituitary tumors. Neurosurgery by R.H. Wilkins, S.S. Rengachari: Chap 124: 1995; p.p. 1247-7.
2. Tindall GT, and Barrow DL: Disorders of the pituitary. St. Louis: Mosby. 1986; p. 498.
3. Rush SC, and Newall J: Pituitary adenom: The efficacy of radiotherapy as the sole treatment. Int Radiat Oncol Biol Phys, 1989; 17: 165-9.
4. Vladyka V, Liscak R, Simonova G, Chytka T, Novotny JJr, Vymazal J, et al.: Radiosurgical treatment of hypophyseal adenomas with the gamma knife: results in a group of 163 patients during a 5-year period. Cas Lek Cesk, 2000; 139(24): 757-66.
5. Tsagarakis S, Grossman A, Plowman PN, et al.: Megavoltage pituitary irradiation in the management of prolactinomas long term follow-up. Clin Endocrinol (Oxf.), 1991; 34: 399-406.
6. Grigsby PW, Simpson JR, Stokes S, et al.: Results of surgery and irradiation or irradiation alone for pituitary adenomas. J Neurooncol, 1988; 6: 129-34.
7. Howlett TA, Plowman PN, Wass JA, et al.: Megavoltage pituitary irradiation in the management of Cushing’s disease and Nelson’s Syndrome: long term follow-up. Clin Endocrinol (Oxf.), 1989; 31: 309-23.
8. Burrow GN, Wortzman G, Rewcastle NB, et al.: Microadenoma of the pituitary. N Engl J Med, 1981; 304: 156-8.
9. Kovacs K, and Horvath E: Tumors of pituitary gland. (Atlas of Tumor Pathology. Fascicle 21; 2nd series). Washington, DC: Armed Forces Institute of Pathology, 1986.
10. Mindermann T, and Wilson CB: Age and gender related occurrence of pituitary adenomas. Clin Endocrinol (Oxf.), 1994; 41 (3): 359-64.
11. Bakay L: The results of 300 pituitary adenoma operations (Prof. Herbert Olivecrona's series). J Neurosurg, 1950; 7(3): 240-55.
12. Nurnberger JF, and Kosey SR: New York, Springer Publishing Co. Inc., 1953.

13. Martin JB, Reichlin S., Brown GM (eds). Clinical Neuroendocrinology. Philadelphia: Davis, 1977.
14. Sassolas G, Tronillas J, Treluyes C, et al: Management of non-functioning pituitary adenomas. Acta Endocrinol, 1993; 129 (suppl 1): 21-6.
15. Nedvidkova J, Schreiber V, Starka L, and Pacak K: Pharmacologic control of hypophyseal tumors: interactions of estrogens, thyroid hormones, growth and anti-growth factors. Cas Lek Cesk, 2000; 139 (Suppl 1): 19-21.
16. Katznelson L, Alexander JM, and Klibanski A: Clinical review. 45: Clinicaly nonfunctioning pituitary adenoma. J Clin Endocrinol Metab, 1993; 76: 1089-94.
17. Fajardo LF: Pathogenesis of radiation injury, pathology of radiation injury, New York, Masson Publishing U.S.A. Inc. 1982.
18. Barrow DL, and Tindall GT. Loss of vision after trans-sphenoidal surgery. Neurosurgery, 1990; 27: 60-8.
19. Chang CY, Luo CB, Teng MM, Guo WY, Chen SS, Lirng JF, et al.: Computed tomography and magnetic resonance imaging characteristics of giant pituitary adenomas. J Formos Med Assoc, 2000; 99(11): 833-8.
20. Henderson WR. Br J Surg, 1939; 26: 811.
21. Arafah BM: Reversible hypopituitary in patients with large non-functioning pituitary adenomas. J Clin Endocrinol Metab, 1986; 62: 1173-9.
22. Visot A: Neurosurgery and pituitary tumors: surgical indications and outcome. Presse Med, 2001; 30(8): 401-4.
23. Davis PC, Hoffman JC Jr, Tindall GT, and Braun IF: Prolactin secreting pituitary microadenoma: In accuracy of high resolution CT imaging. Am J Neuroradiol, 1985; 144: 151-6.
24. Kleinberg DL, Boyd AE III, Wardlaw S, et al: Pergolide for the treatment of pituitary tumors secreting prolactin or growth hormone. N Engl J Med, 1983; 309: 704-9.
25. Jackson IM, and Noren G: Role of gamma knife radiosurgery in acromegaly. Pituitary, 1999; 2(1): 71-7.
26. Sheithauer BW, Kovacs KT, Laws ER, and Randall RV: Pathology of invasive pituitary tumors with special reference of functional classification. J Neurosurg, 1986; 65: 733-44.
27. Black Mccl, Zervas NT, Ridgway EC, Martin JB (eds). Secretory tumors of the pituitary gland. New York: Raven, 1984.
28. Linfoot JA (ed.) Recent Advances in the Diagnosis and Treatment of Pituitary Tumors. New York: Raven Press, 1979.
29. Post KD, Jackson IMD, Reichlin S. (Eds). The pituitary adenoma. New York: Plenum, 1980.
30. Chakeres DW, Curtin A, and Ford G: Magnetic resonance imaging of pituitary and parasellar abnormalities. Radiol Clin North Am, 1990; 17: 255-65.
31. Leeds NE, and Naldich TP, Computerized Tomography in the diagnosis of sellar and parasellar lesions. Semin Roentgenol, 1977; 12: 121-35.
32. Wilson CB and Dempsey LC: Transsphenoidal microsurgical removal of 250 pituitary adenomas. J Neurosurg, 1978; 48(1): 13-22.
33. Tindall GT, and Reisner A: Prolactinomas: Neurosurgery by R.H. Wilkins, S.S. Rengachari. Chap. 126: 1995; p.p.1305.
34. Asa SL: Tissue culture in the diagnosis and study of pituitary adenomas. Lloyd RV (ed.): Surgical pathology of the pituitary gland. Philadelphia: Saunders, 1993; pp 94-115.
35. Albert L, and Rhoton Jr: Microsurgical anatomy of the sellar region. Neurosurg. R. H. Wilkins, S.S. Rengachari, Chap. 122, 1995; p.p. 1243-4.
36. Ciric I, Mikael M Stafford T, et al: Trans-sphenoidal microsurgery of pituitary microadenomas with long term follow-up results. J Neurosurg, 1983; 59: 395-401.
37. Dominque JN, Richmond IL, and Wilson CB: Results of surgery in 114 patients with prolactin secreting pituitary adenomas. Am J Obstet Gynecol, 1980; 137: 102-8.
38. Faria MA, and Tindall GT: Transsphenoidal microsurgery for prolactin secreting pituitary adenomas. J Neurosurg, 1982; 56: 33-43.
39. Fajardo LF: Pathogenesis of radiation injury, pathology of radiation injury, New York, Masson Publishing U.S.A. Inc. 1982.

THE COPING STYLES OF ADOLESCENTS WITH TYPE 1 DIABETES

Jawad KA Al-Diwan *MBChB, MSc*

Abstract

Background: The importance of developmental prospective in understanding the variability in diabetic adolescent's coping efforts is underscored in Iraq.

Objective: This work was carried out to assess the association between the coping styles and higher level of perceived diabetes related worries.

Methods: 160 adolescents with type 1 diabetes were enrolled in this study from different diabetes centers in Baghdad city for the period 1st June to 21st Dec. 2000. Each participant interviewed individually. Full information including age, sex, and duration of diabetes, sport activity and visits to diabetic clinics and a diabetic related worries subscales were used. Coping styles (active and aggressive coping styles) were assessed by selected subscales. The influences of coping styles and other variables on diabetic related worries were examined by stepwise regression analysis.

Results: Higher perception of diabetic related worry was significantly associated with active coping, aggressive coping, emotional support and sex. Age and visits to diabetic clinics were not associated with higher perceived diabetic related worry.

Conclusion: The finding demonstrated the importance of coping behavior to perceived diabetic worries, which in turn affects the metabolic control.

Key words: diabetic adolescents, coping styles, Iraq

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Introduction

Adolescents with type 1diabetes faced with a complex set of developmental changes as well as changing demands of the disease[1]. Coping skills are vital for emotional and social development among young people[2]. Coping styles refers to typical and habitual preferences for way of approaching problems and might be regarded as strategies that people generally use to cope across stressors. Several studies showed that avoidance coping (refers to reduce emotional distress caused by stressful event and to manage and regulate emotions that might accompany stressors) in adolescents with type 1diabetes predicts a poor illness specific self care behavior[3-5]. The importance of developmental prospective in understanding the variability in diabetic adolescent's coping efforts is underscored in Iraq.

This work was carried out to assess the association between the coping styles and higher level of perceived diabetes related worries.

Dept. Community Medicine, College of Medicine, Al-Anbar University

Address correspondence to Dr. Jawad K. Al-Diwan, P.O. Box 70017, Baghdad, e-mail: jawadkadhim@yahoo.com

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Materials and methods

160 adolescents with type 1diabetes were enrolled in this study from different diabetic centers (National Diabetic Center at Al-Yarmouk teaching hospital, Diabetic Consultancy Clinic at Al-Kadh.miya teaching hospital, Diabetic Consultancy Clinic at Al-Mansour teaching hospital and Diabetic Consultancy Clinic at Ibn Al-Beldy teaching hospital) in Baghdad city for the period 1st June to 21st Dec. 2000. Adolescence period is considered between 10 – 21 years[6]. Their mean age was 15.1 ± 2.3 years; 53.8% of them were females; 51.3% of them had the disease for more than five years and 76.9% of them reported a history of a frequent hospitalization.

Each participant was interviewed individually. Full information including age,

sex, and duration of illness, sport activity and visits to diabetic clinics were collected.

A diabetic related worries subscales was assessed[7,8]. Diabetic related worry subscale was linearly transformed so that the worst and the best possible scores were zero and 100, respectively. Higher scores indicate lower perceived diabetes related worry.

To cover different coping styles relevant to the management of diabetes in adolescents, Active coping, seeking social support for instrumental reasons, seeking social support for emotional reasons, behavioral disengagement and mental disengagement were assessed by subscales selected from COPE scale developed by Carver et al[9]. Aggressive coping was assessed by modified ways of coping questionnaires[10]. Scores for assessing coping styles were computed as the mean score across items, yielding a scoring range of scale that correspondent with single item: zero (lowest possible use of each coping style) through three (highest possible use of each coping style).

Internal consistency of diabetes related worry and coping styles was assessed by Cronbach's α score. The influence of coping style and other variables on diabetic related worries were examined by stepwise regression analyses[11]. P value less than 0.05 was considered as statistically significant.

Results

Higher perception of diabetic related worry was significantly correlated with active coping, aggressive coping, emotional support and sex ($p < 0.05$). There was no significant association of higher perceptions of diabetic related worry with social support. Lower perception of diabetic related worry was significantly associated with aggressive coping and emotional support ($p < 0.05$). Age and visits to diabetic clinics were not associated with higher perceived diabetic related worry. These findings are shown in Table 1.

Table 1: Regression partial coefficient of diabetic related worry with the studied variables

Variable	Diabetic related worry	
	Partial coefficient	P value
Active coping	- 0.16	0.03
Aggressive coping	0.088	0.01
Emotional support	- 0.07	0.001
Social support	- 0.45	NS
Age	0.05	NS
Sex	- 0.2	0.001
Visit to diabetic clinic	- 0.1	NS

Discussion

An obvious lack of accurate informations on the exact magnitude of diabetes in Eastern Mediterranean Region was reported[12]. Previous studies tend to focus on relationship between treatment related factors (poor adherence to insulin treatment and specifically mission shots) and poor metabolic control[13,14]. However, others demonstrated that stress is a significant risk factor for medical maladjustment[15,16]. Since publication of Diabetes Control and Complication Trials (DCCT) results[17], it has been widely accepted that improving metabolic control must be a fundamental priority in type 1 diabetes care. Improving patient's metabolic control was primarily to a combination of insulin management and psychosocial support provided by DCCT care team[18].

This study revealed that higher perceived diabetic related worry showed a negative significant association with active coping (refers to efforts directed toward rational management of a problem and aimed to change the situation causing the stress) and a positive significant association with aggressive coping (avoidance coping and emotional focus coping such as behavioral and mental disengagement). This finding may be due to the fact that diabetic adolescents were struggling in the social-emotional and peer relationships areas of functioning which lead to emotional problems such as anxiety and depression. It is the maladaptive coping styles, which predict stress. Problem focus coping or active coping is generally associated with better adjustment[19]. Avoidance coping (refers to reduce emotional stress caused by stressful situation and to manage and to regulate emotions that might accompany stressors) is associated with poor specific self-care behavior[20]. Greater use of active coping (problem focus coping) was related to improved metabolic control and diabetic life satisfaction while aggressive coping was related to poor metabolic control[16]. This finding indicated that anxious adolescents may be harder working in monitoring diabetes and may take actions that are more effective in response to signs of poor blood glucose.

Diabetic related worry was significantly associated with emotional support. It may refer to that diabetic adolescents cope with illness by ventilating feelings through shouting and arguing in family. The finding that social support had no role in reducing diabetic related worry may reflect a higher degree of self-blame among diabetic adolescents. Although self-blame might stimulate adolescent's responsibility taking[16], too much self-blame might be linked to internalization of emotional problems[21]. This finding may be due to parental perception of the adolescent's adjustment at school and level of social and emotional functioning with peers. In previous communication, the role of peers in management of diabetes was demonstrated in Iraq[22].

This study revealed, also, that diabetic related worry was significantly associated with sex. It is consistent with other reports[22,23]. It may be due to general family factors such as warmth, cohesion and adaptability, which are the primary drivers for emotional instability. Source of stress for adolescent girls with diabetes include frequent changes in daily routines, academic challenges, interpersonal conflicts with family and peers, and societal messages regarding what is consider attractive contribute to adolescent's concern about their body image (when girls are becoming more concern about their body shape and size and particularly vulnerable to opinion of peers[24], especially boys) may lead to intentional compromising in disease management[22,25] (sever dietary indiscretion and repeated insulin omission) and eating disorders (anorexia nervosa, bulimia nervosa, excessive exercising and food deprivation). It seems that the situation in Iraq is different than in Western countries.

Recently, Wills et al[26] reported that there are failing to achieve high standards of care for young adults with type 1 diabetes, a problem which likely to affect the entire United Kingdom. They demonstrated the need to learn from European centers that achieved better results. The dose adjustment for normal eating program (DAFNE) was a success in United Kingdom[27].

In conclusion, the findings demonstrate the importance of coping behavior on perceived diabetic worries, which are in turn affect the metabolic control. Health promotion intervention in schools and others are important in helping the adolescents to better integrate challenges of the disease, and to adapt a more constructive behavior.

References

1. Hamilton J, and Danewan D: Deterioration in diabetes control during adolescence: physiological or psychological? J Pediatr Endocrinol Metab, 2002; 15: 115-26.

2. Dumont M, and Provost MA: Resilience in adolescents: protective role of social support, coping strategies, self-esteem and social activities on experience of stress and depression. J Youth Adolesc, 1999; 3: 343-63.
3. Hanson CL, Cigrang JA, Harris MA, Carle DL, Reylea G, and Burghen GA: Coping style in youth with insulin-dependent diabetes. J Consult Clin Psychol, 1989; 57: 644-51.
4. Ebata AT, and Maas RH. Personal, situation and contextural correlates of coping in adolescents. J Res Adolesc, 1994; 4: 99-125.
5. Seiffage-Kreke I, and Stemmer M: Coping with every day stress and link to medical, psychological adaptation in diabetic adolescents. J Adolesc Health, 2003; 33: 180-8.
6. Paxaman JM, and Zuckerman RP: Laws and policies affecting adolescent's health. Geneva, 1987; p.p. 4-10.
7. Bott V, Muhlhauser I, Overmann H, and Berger M: Validation of specific quality of life scale for patients with type 1 diabetes. Diabetes care, 1998; 21: 757-68.
8. Ma'ala EG: Assessment of health related quality of life of diabetic adolescents (type 1 diabetes). PhD thesis, College of Nursing, Baghdad University, 2001.
9. Carver CS, Scheier MF, and Weintraub JK. Assessing coping strategies: a theoretically based approach. J Pers Soc Psychol, 1989; 2: 267-83.
10. Folkmans S, and Lazarus RS: Ways of coping questionnaire sampler set manual, Test booklet scoring key palo Alto CA Mind Garden Consulting psychology press. 1988.
11. Daniel WW: Biostatistics: A foundation for analysis in health sciences. 7th edition. New York, John Wiley and Sons Inc., 1999.
12. Al-Alwan AA: Diabetes in WHO Eastern Mediterranean Region. Bulletin Arab Group Study Diabet, 1992; 1: 17-21.
13. Morris AD, Boyle DR, McMahan AD, Greene SA, MaeDonald TM, and Newton RW: Adherence to insulin treatment, glycaemic control and ketoacidisis in IDDM. Lancet, 1997; 350: 1505-10.
14. Weissberg-Benchett J, Glasgow AM, Tynan WD, Wirtz P, Turej J, and Ward J: Adolescent diabetes management and mismanagement. Diabetes care, 1995; 8: 77-2.
15. Grossman HY, Brink S, and Hanser ST: Self-efficacy in adolescent girls and boys with IDDM. Diabetes care, 1987; 10: 324-9.
16. Graue M, Wentzel-Larsen T, Brue E, Hanstead BR, and Soviko O: The coping styles of adolescents with type 1 diabetes are associated with degree of metabolic control. Diabetes care, 2004; 27: 1313-7.
17. Diabetes Control and Complications Trial Research Group. Effect of intensive diabetes treatment on the development and progression of long term complications in adolescents with insulin dependent diabetes mellitus. Diabetes control and Complications Trial. J Pediatr, 1994; 125: 127-88.
18. Skinner TC, and Hampson SE: Personal model of diabetes in relation to self-care and wellbeing and glycaemic control. Diabetes care, 2001; 24: 828-33.
19. Hansen CL, Cograng JA, Harris MA, Carle DL, Relyea G, and Barghen GA: Coping styles in youths with IDDM. J Consult Clin Psychol, 1989; 57: 644-51.
20. Ebata AT, and Moas RH: Personal situation and contextual correlates with coping in adolescents. J Res Adolesc, 1994; 4: 99-125.
21. Grey M, Boland EA, and Davidson M: Short time effects of coping skill training as adjunct to intensive therapy in adolescents. Diabetes care, 1998; 21: 902-8.
22. Jack L: Biopsychosocial factors affecting metabolic control among female adolescents with type 1 diabetes. Diabetes Spectrum, 2003; 16: 154-9.
23. Snock FJ, and Skinner TC: Psychosocial counseling in problematic diabetes: does it help. Diabetes Med, 2002; 19: 265-73.
24. Harrson K, and Cantor J: The relationship between media consumption and eating disorders. J Comm Health, 1986; 47: 40-76.
25. Bryden KS, Dunger DB, Mayon RA, Pevler RC, and Neil AW: Poor prognosis of young adults with type 1 diabetes. Diabetes Care, 2003; 20: 1052-7.
26. Wills CJ, Scott A, Swift PG, Davies MJ, and Mackie AD: Retrospective review of care and outcomes in young adults with type 1 diabetes. Br Med J, 2003; 327: 260-1.
27. DAFNE study group. Training in flexible, intensive insulin management to enable dietary freedom in people with type 1 diabetes: dose adjustment for normal eating randomized control trial. Br Med J, 2002; 325: 746-9.

SUBJECTIVE AND QUANTITATIVE EVALUATION OF BONE MARROW TREPHINE BIOPSY IN IDIOPATHIC MYELOFIBROSIS

Ahlam Al-Mashhadani[1] *FIBMS*, **Saad Sh. Mansoor**[2] *FRCPath*, **Falih S. Sarhan**[2] *FIBMS*

Abstract

Background: Idiopathic Myelofibrosis (IMF) is one of the myeloproliferative disorders in which various degrees of bone marrow fibrosis constitute the cardinal pathogenetic criteria for the disease therefore the study of bone marrow trephine biopsy is a major step in diagnosis .

Objectives: Are reticulin and iron stain are essential for the diagnosis and staging of idiopathic myelofibrosis

Methods: Trephine biopsies of 30 patients with IMF were reevaluated and the paraffin blocks were further sectioned, stained for H & E and reticulin and Perl's reaction. The patients were classified into four groups according to Cologne criteria for which both subjective and quantitative evaluations of trephine biopsies were performed.

Results: Both qualitative and quantitative evaluation was performed on trephine biopsies.

The most consistent finding with progression of diseases was megakaryocytic and granulocytic proliferation with preponderance of megakaryocytes. The study also proves that most patients showed an increase in the number of hemopoietic cells, reticulin fibers, trabecular bone width, osteoblastic index, blood vessels, and a reduction in the iron stores.

Conclusion: A thorough assessment of bone marrow biopsy including adequate tissue sampling stained for reticulin and iron stain are essential for the diagnosis and staging of idiopathic myelofibrosis.

Keywords: Idiopathic myelofibrosis, trephine biopsy, subjective, quantitative

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Introduction

Idiopathic myelofibrosis is an interesting myeloproliferative disease, which presents in middle aged and elderly people with signs and symptoms of anemia, splenomegaly, bone pain and hypermetabolic state with a leucoerythroblastic blood picture, teardrop cells and varying degree of bone marrow fibrosis[1,2].

The principal pathogenetic criteria are an abnormal megakaryocyte resulting from an abnormal neoplastic ste+m cell. The death of these abnormal megakaryocytes results in the production of mitogens that result in bone marrow fibrosis[3-7].

The aspiration of bone marrow in IMF patient usually yields dry tap or unsatisfactory marrow is collected[3,4,8]. Therefore, trephine biopsy is essential for diagnosis, which shows an increase in reticulin fiber density and thickness[3,4,10].

The diagnosis and staging of IMF is based on the Cologne Criteria[11,12] and because of the prognostic significance of the histopathological features of IMF, this study

[1]Specialist in Hematology, Ministry of Health-Al-Noor Hospital, [2]Dept. Pathology, college of Medicine, Al-Nahrain University.
Address correspondence to: Dr. Falih Salim Sarhan.
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is performed to assess the marrow trephine findings in different stages of the disease.

Material & Methods

Using paraffin blocks of 30 patients with IMF, processed from Sep.1998 to Sep. 2002, 18 patients were males and 12 were females. Three sections were obtained from the original paraffin blocks and stained for H &E, reticulum and Perl's reaction. Trephine biopsies were re-evaluated both subjectively and quantitatively.

Subjective evaluation involves the systemic examination of H & E slides for

1. Cellularity: graded as follows[13,14] decreased <35%, normal 35-49%, slight Increase 50-59%, moderate Increase 60-89%, marked Increase 90-100%
2. Megakarocylic concentration[13,14] decreased <3/mm2, normal 3-5/mm2, slight increase 6-8/mm2, moderate increase 9-14/mm2, marked increase ≥15/mm2
3. Iron content: after staining the slides for Perl's reaction the results were graded as follows[15], 0: No iron, 1: Minimal iron, , 2: Slight & Patchy, 3: Moderate & Diffused, 4: Strong and extensively diffused, Grades 0-1 indicate iron deficiency.
4-Reticulin fibrosis was evaluated as in Ellis et al[14].

The quantitative evaluation of bone marrow biopsies involved counting the number of megakaryocytes, and blood vessels in one cubic mm using a planimetric method[16]. Using the Chalkley's point counting method, the amount of hemopoietic tissue was measured as percentage[9].

Both of the osteoblastic index and the trabecular bone width was measured using an ocular graticule[17]. Bone marrow biopsies of 30 subjects with normal histology was chosen as a base line data.

Statistical analysis was done using one way ANOVA and t-test, with P value less than 0.05 was considered significant.

Results

The subjective evaluation of trephine biopsies showed that most patients examined have increased cellularity (Table 1).

Table 1: Subjective evaluation of cellularity

Cellularity	No.	Percentage (%)
Reduced	12	40
Normal	4	13.34
Slightly increased	6	20
Moderately increased	5	16.66
Markedly increased	3	10

Megakaryocytes are increased in 76.66% of cases, being markedly increased in 16.66%. In 20% of cases, clustering of megakaryocytes was recognized (Table 2).

Table 2: Subjective evaluation of megakaryocytes

Megakaryocytes	Evaluation	No. of cases	Percentages (%)
Number	Reduced	4	13.34
	Normal	3	10
	Slight increase	1	36.66
	Moderate increase	7	23.34
	Marked increase	5	16.66
Distribution	Diffuse	21	70
	Cluster	6	20
	Sheets	3	10

Reticulin fibrosis was evident in all cases; however, it was marked in 22 patients (Table 3).

Table 3: Subjective evaluation of reticulin fibrosis

Reticulin fibrosis	Number	Percentage (%)
Slight increase	3	10
Moderate increase	5	16.66
Marked increase	22	73.34

One third of patients showed iron deficient erythropoiesis with six patients showed no demonstratable iron in stores (Table 4).

Table 4: Semi quantitative evaluation of iron pigment

Marrow iron grade	**Number of cases**	**Percentage**
0	6	20
+	4	13.34
++	14	46.66
+++	6	20

The results of quantitative evaluation of trephine biopsies in patients with IMF are shown in table 5, which also compares the results with that of control.

Table 5: Comparison between quantitative evaluation of IMF patients and controls

	Patient mean	**Control mean**	**P value**
Hemopoietic tissue	44.32	50.93	<0.01
Fatty tissue	5.99	49.01	<0.001
Megakaryocytes (MKC)	43.60	19.09	<0.001
Osteoblastic index (OBI)	0.66	0.35	<0.001
Blood vessels (BV)	4021	2613	<0.001
Trabecular bone width (TBW)	116	78	<0.001

A correlation between quantitative evaluation of bone marrow biopsy & the degree of fibrosis was obtained and is shown in table 6.

Table 6: Correlation between degree of reticulin fibrosis & quantitative evaluation of bone marrow biopsy

Reticulin fibrosis	**No. (%)**	**Hemopoietic. Tissue**	**Fatty*** tissue**	**Fibrous tissue**	**TBW**	**MKC**	**OBI**	**BV**
Slight increase	3(10)	92.68	3.81	7.32	80.03	35.62	0.612	3025.5
Moderate increase	5(16.34)	80.06	7.79	33.67	102.12	37.15	0.635	3712.3
Marked increase	22(73.66)	31.53	10.60	45.05	116.88	46.37	0.676	4101.08
p value		<0.01	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

The Cologne criteria was used in this study for staging of IMF which showed that 10% of patients were stage I, 15% stage II, 40% stage III and 35% stage IV. Bone marrow biopsy finding at different stage of IMF is shown in table 7.

Table 7: BMB finding at different stages of IMF (one way ANOVA)

	Stage I	Stage II	Stage III	Stage IV	P value
Hemopoietic tissue	92.68	80.06	49.31	41.10	<0.05
Fatty tissue	3.81	7.79	9.24	16.92	<0.05
Fibrous tissue	7.32	33.67	44.58	49.06	<0.05
TBW	80.03	102.12	116.88	119.35	<0.05
MKC	35.62	37.15	45.91	49.36	<0.001
OBI	0.612	0.635	0.676	0.789	<0.05
BV	3025.5	3712.3	4059.6	4300.8	<0.05

Discussion

A better understanding of the myeloproliferative process and prognosis of IMF patients can be obtained through study of how this disease is expressed in the bone marrow. In this study both subjective and quantitative evaluation of bone marrow trephine biopsies was performed correlating the findings with the amount of iron pigment, reticulin fibrosis and staging of the disease.

The majority of cases showed hemopoietic hypercellularity rather than hypocellularity, a result similar to that of Varki et al, 1983[18]. Many of the megakaryocytes showed morphologic abnormalities as those described by other studies[19,20]. These abnormalities include bizarre nuclear configuration, most of the cells show hypo segmented nuclei while few megakaryocytes have hyper segmented nuclei[19, 20].

A significant degree of marrow fibrosis was recognized in most patients (73.34%) which is higher than that reported earlier by Varki et al, 1983[18], but lower than the figure reported by Jalal (1988) who found that all cases showed marked degree of fibrosis[21].

The semi quantitative evaluation of marrow iron stores revealed the presence of depleted iron stores in 33.34% of patients, presumably because of secondary iron deficiency due to blood loss either resulting from the presence of extramedullary hemopoietic foci leading to peptic ulcer[22] or due to platelet dysfunction, acquired factor V deficiency, thrombocytopenia and DIC[23].

The quantitative evaluation of bone marrow biopsies showed that IMF patients have lower mean values of hemopoietic tissue and fatty tissue and by higher mean values of fibrous tissue; trabecular bone width, OBI, MKC concentration, and blood vessels (p value < 0.001). Similar findings were described by Frisch et al, (1985), who also described the correlation between the degree of reticulin fibrosis and quantitative evaluation of BMB[17]. He proved that the mean value of hemopoietic tissue volume is reduced with increasing amount of reticulin fibrosis, while the mean values of fatty tissue volume, fibrous tissue volume, MKC concentration, OBI, TBW & blood vessels tend to correlate with the degree and reticulin fibrosis[17]. Our study also proves these findings, which were very highly significant statistically.

The number and character of reticulin fibers vary considerably[10]. In sections with abundant hemopoietic cells, there is only a slight-moderate increase in reticulin while in areas with markedly reduced hemopoietic tissue, greatly thickened, more abundant and highly intertwining bundles of reticulin fibers are recognized[10].

We also showed that clusters of megakaryocytes are usually present and may be the only recognizable hemopoietic cells in areas of dense fibrosis. This finding

supports what is written about the close relationship between the marrow fibrosis and megakaryocytes, and the role of the megakaryocytes-derived growth factors in the pathophysiology of IMF[13,17,20]. The marrow sinusoids are usually distended and contain hemopoietic cells. Bone trabeculae may be widened and residual fat cells may be seen in both cellular and fibrotic phase[8].

The quantitative evaluation of bone marrow biopsies also revealed that there is a progressive reduction of hemopoietic tissue, increase in fatty tissue, fibrous tissue, OBI, TBW and blood vessels with increasing stage of IMF. This was in accordance with two earlier studies by Burchardt et al in 1982, and 1984[13,14].

Increase number of MKCs with progression of the disease was a constant and a very highly significant finding in our study, which is consistent with previous findings reported by Jalal in 1988 who showed that collagen fibrosis was found in 86% of patients reviewed and megakaryocytes increased in numbers with tendency for clustering and abnormal morphology[21].

References

1. Manoharn A: Myelofibrosis: Prognostic factors and treatment. Br J Hematol, 1988; 69: 295-8.
2. Garcia S, Miguel A, Linares M, Novarro M, and Colomina P: Idiopathic myelofibrosis terminating in erythroleukemia. Am J Hematol, 1989; 32: 70-1.
3. Lichtman MA: Idiopathic myelofibrosis (Agnogenic Myeloid Metaplasia). In: Lichtman MA, Beutler E, Kipps TJ, Williams WJ editors. Williams Manual of Hematology. 6th edition. New York: McGraw-Hill, 2003; p.p. 259-63.
4. Pearson TC, and Lewis SM: Non-Leukemic myeloproliferative disorders. In: Hoffbrand AV, Lewis SM, Tuddenham EG editors. Postgraduate Hematology. 4th edition. Oxford: Butter Worth-Heinemann, 1999; p.p. 520-7.
5. McCarthy DM: Fibrosis of the bone marrow: content and causes. Br J Hematol, 1985; 59: 1-7.
6. Kimura A, Katoh O, Hyoda H, and Kuramoto A: Transforming growth factor β regulates growth as well as collagen and fibronectin synthesis of human marrow fibroblasts. Br J Hematol, 1989; 72: 468-91.
7. Hasselbalch H: Pathogenesis of angiogenesis in idiopathic myelofibrosis. Am J Hematol, 1990; 33: 151.
8. Kaufman S, Inclea S, and Reif R: Idiopathic myelofibrosis complicated by chronic lymphatic leukemia. Clin Lab Hematol, 1987; 9: 81-4.
9. Hartsock RJ, Smith EB, and Petty CS: Normal variations with aging of the amount of hemopoietic tissue in bone marrow from the anterior iliac crest. Am J Hematol, 1964; 43(4): 326-8.
10. Acherman RD: Bone marrow .In: Roza, J. editor. Ackerman's surgical pathology. 8th edition. New York.Mosby, 1995; p.p. 1797-915.
11. Thiele J, Kvasnicka HM, Diehl V, Fischer R, and Michiels JJ: Clinicopathological diagnosis and differential criteria of thrombocythemias in various myeloproliferative disorders by histopatholgy, Histochemistry and immunostaining from bone marrow biopsies. Leuk Lymph, 1999; 33: 207-18.
12. Visani G, Finelli C, Castelli U, Pett MC, Ricci P, and Vianell N: Myelofibrosis with myeloid metaplasia: Clinical and hematological parameters predicting survival in a series of 133 patients. Br J Hematol, 1990; 75: 4-9.
13. Burkhardt R, Frisch B, and Bartle R: Bone marrow biopsy in hematological disorders. J Clin Pathol, 1982; 35: 257-84.
14. Ellis JT, Silver RT, Coleman M, and Geller SA: The bone marrow in polycythemia vera. Seminars in Hematology, 1975; 433-43.
15. Al-Ani RJ: Evaluation of the anemia of rheumatoid arthritis. A thesis submitted to the scientific council of pathology. 1996.
16. Lazzarino M, Morra E, Castello A, Inverardi D, Coci A, and Pagnuceo G: Myelofibrosis in chronic granulocytic leukemia: Clinicopathologic correlations and prognostic significance. Br J Hematol, 1986; 64: 227-40.
17. Firsch B, Lewis SM, and Burkhardt R: Biopsy pathology of bone marrow London: Champan & Hall; 1985.
18. Varki A, Lottenberg R, Griffith R, and Reinhard E: The syndrome of idiopathic myelofibrosis. Medicine, 1983; 62(6): 353-70.
19. Wolf BC, and Neiman RS: Myelofibrosis with myeloid metapalsia: Pathophysiologic implications of the correlation between bone marrow changes and progression of splenomegaly. Blood, 1985; 65: 803-9.
20. Burchardt R, Bartle R, Jager K, Frisch B, Kettner G, and Mahl G: Working classification of chronic myeloproliferative disorders based on histological, hematological and clinical findings. J Clin Pathol, 1986; 39: 237-52.
21. Jalal ES: Fibrosis of the bone marrow pattern and significance. MSc Hematology thesis, College of Medicine Baghdad University, 1988.

22. Lee GR, Fcerster J, Lukeus J, Paraskevas F, Greer JP, and Rodgers GM: Wintrobe's Clinical Hematology New York:Williams and Wilkins, 1999.
23. Tafferi A: Proceedings of the outreach of Hematology, bone marrow transplantation. Educational Symposium, Jeddah, 1998.
24. Burkhardt R, Bartle R, and Jager K: Chronic myeloproliferative disorders. Path Res Pract, 1984; 179: 131-86.

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التعصيب الحركي للعضلات القصيرة في ابهام اليد: المضامين التشريحية والسريرية

محمد عودة سلمان[١]، اكرم عبود جعفر[١]، فرقد بدر حمدان[٢]

الخلاصة

خلفية الدراسة : يشكل الفهم الواضح لتعصيب العضلات القصيرة لابهام اليد اهمية كبرى لجراح الكف. و لايزال هذا التعصيب يشكل موضوعاً للنقاش حيث ان العصبين المتوسط و الزندي يلعبان دوراً متبايناً في ذلك.

هدف الدراسة : وصف مصدر تعصيب العضلات القصيرة لابهام اليد، و احتمالية وجود العضلة بين العظام الراحية الاولى. كما تهدف الى ربط الاختلافات التعصيبية بالمدلولات المآلية لامراض الاعصاب و اصاباتها.

طريقة العمل : تم تشريح (١٥) كف محنطة لبالغين. كما اجري مخطط كهربية العضل على (٤٢) كف لمتطوعين اصحاء حيث تمت دراسة جهد الفعل المركب و طراز التداخل باعتيان كل عضلة على حدة.

النتائج : ظهر في (٨٦،٦٪) من النماذج المشرحة ان الفرع العضلي للعصب المتوسط هو فرعه الاول في الراحة. وجد التفاغر بين العصبين المتوسط و الزندي في (٥٣،٣٪) من الكفوف المشرحة و قد تم اظهاره في مستويات مختلفة. رغم الاهتمام الخاص لاظهار العضلة بين العظام الراحية الاولى الا انه لم يكن بالامكان استبيانها ككيان منفصل. اظهرت دراسة مخطط كهربية العضل اختلافات جديرة بالاعتبار في التعصيب حيث ان العضلة المقربة لابهام اليد لم تستلم تعصيبا خالصا من العصب المتوسط الا انها استلمت تعصيبا من العصب الزندي في (٩٠،٥٪) من الحالات. اما العضلة القصيرة المبعدة لابهام اليد فلم تستلم تعصيبا خالصا من العصب الزندي حين انها كانت تعتمد في تعصيبها على العصب المتوسط (٦٦،٧٪). ظهرت اكبر نسبة لتعصيب مختلط (٦٦،٧٪) للعصبين المتوسط والزندي في العضلة المقابلة لابهام اليد.

الاستنتاج : ان وجود التفاغر بين العصبين المتوسط والزندي في راحة اليد وفي مستويات مختلفة يجعله عرضة للانجراح اثناء التداخلات الجراحية. ان غياب الاختلافات على الجانبين في نمط التعصيب لكل عضلة على حدة يمكن ان يكون عونا في تقدير المآل لليد المتأثرة من خلال دراسة مخطط كهربية العضل للجانب المعاكس. ان الطريقة التقليدية لدراسة مخطط كهربية العضل لا يمكن ان تخصص بدقة التعصيب لكل عضلة. و من الناحية المآلية فان الطريقة المستخدمة في هذه الدراسة لو تم تطبيقها بشكل تقليدي فان شدة الاصابة يمكن التعبير عنها بصيغة العضلات المتأثرة. ان العديد من هذه العضلات تستلم تعصيبا مختلطا مما يجعلها قابلة للاحتفاظ بوظيفتها على المدى الطويل.

مفتاح الكلمات: عضلات الرانفة، التعصيب الحركي، مخطط كهربية العضل

[١] فرع التشريح البشري (كلية الطب-جامعة النهرين)
[٢] فرع الفسلجة (كلية الطب-جامعة النهرين)

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الملخصات العربية

التغيرات النسجيه الحاصله في مشيمة المصابات بمقدمة الارتعاج

كوكب سليم القيسي[1]، مي فاضل ماجد[2]، شذى الزهيري[3]

الخلاصة:

خلفية الدراسة: يعد مرض متقدمة الارتعاج من الأمراض الرئيسية التي تصيب التكاثر البشري، و تتأثر فيها نسبه ١٠٪ من الولادات البشرية و بالدرجة الرئيسية بعدد من الأمراض الجهاز يه لخلايا المتن التي تسبب تنشيط و انتشار الصفيحات الدموية التي تسبب فقر الدم الموضعي .

هدف الدراسة: تهدف الدراسة الى توضيح التغيرات النسيجيه في مشيمة النساء المريضات المصابات بمرض ارتفاع ضغط الدم.

طريقة العمل: شملت الدراسة خمسين مخاضا في حاله ولآده مبكرة، منهن خمسه عشر حامل بحمل طبيعي و خمسه وثلاثون حامل مصابات بارتفاع ضغط الدم مع بطا نمو الجنين في الرحم. تم اخذ العينات و حضرت للفحص بالمجهر الضوئي.

النتائج: لوحظ وجود العديد من التغيرات عند مختلف المريضات متضمنة ما يلي:

١. تنكس بعض خلايا البطانة.

٢. تنكس في غالبية خلايا ألارومه الغاذيه.

٣. تنكس زجاجي و تليف ألارومه الغاذيه في بعض المقاطع النسيجية للمريضات.

٤. تشرب خلايا ألارومه الغاذيه بالقطيرات الدهنية في بعض المقاطع النسيجية للمريضات.

الاستنتاج: إن الأسباب ألاوليه لحدوث حالات مقدمة الارتعاج يعود لخلل في نمو خلايا ألارومه الغاذيه و تنكس غالبيه خلايا البطانة.

مفتاح الكلمات: مقدمه الارتعاج، المشيمه، تغيرات نسجيه

[1] وحدة المجهر الألكتروني (كلية الطب-جامعة النهرين)
[2] قسم الأنسجة (كلية الطب-جامعة النهرين)
[3] فرع النسائية و التوليد (كلية الطب-جامعة النهرين)

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الملخصات العربية

عزل و تنمية المايلوبيروكسيديز من الخلايا متعددة الانوية في الانسان

عصام الجميلي[1]، مثنى الصفار[2]، رغد السهيل[3]

الخلاصة:
خلفية الدراسة: يعد انزيم المايلوبيروكسيديز (EC 1.11.1.7) من مجموعة انزيمات الاكسدة و الاختزال الموجودة في حبيبات الخلايا العدلة الهاضمة للاحياء المجهرية بفعل انتاجها لبعض المؤكسدات القاتلة لها.

هدف الدراسة: عزل و تنقية الانزيم (MPO) من الخلايا متعددة الانوية.

طريقة العمل: تمت تنقية الانزيم من حبيبات الخلايا متعددة الانوية من الدم باستخدام كروماتوغرافيا التبادل الايوني (CMC) و الترشيح الهلامي Sephacryl -200 و الترحيل الكهربائي بوجود SDS.

النتائج: عزلت حبيبات الخلايا متعددة الانوية من الدم و حضر مستخلص الحبيبات بتجانس راسب الحبيبات بمحلول السكروز بتركيزM ٠,٣٤ نقي انزيم المايلوبيروكسيديز (MPO) المعزول من الخلايا متعددة الانوية في الانسان الى حد التجانس بخطوتين شملت كروماتوغرافي التبادل الايوني عمود CMC و عمود السيفاكريل S-200 بعدد مرات تنقية بحصيلة بلغت ١,٢٨١ و ٤٣,٩٤٪ على التوالي. تجانس النتائج النهائي بواسطة الترحيل الكهربائي بوجود SDS و بلغ الوزن الجزيئي للانزيم ٨٠,٠٠٠ دالتن بطريقة الترحيل الكهربائي بوجود SDS و٨٨,٠٠٠ دالتن باستخدام عمود السيفاكريل S-200

الأستنتاج: ان تنقية الانزيم سوف تفيد مستقبلا في البحوث الطبية و من جملتها الألتهابات.

مفتاح الكلمات:- الخلايا متعددة الانوية، انزيم المايلوبيروكسيديز من الخلايا متعددة الانوية، تنقية انزيم المايلوبيروكسيديز.

[1]معهد الهندسة الوراثية و التقانة الأحيائية (بغداد)
[2] المعهد الطبي التقني (بغداد)
[3] قسم علوم الحياة (كلية العلوم -جامعة بغداد)

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الملخصات العربية

دور حامض السايليك الكلي و الدهن المرتبط بحامض السايليك للتمييز بين فاعليات مرضى التهاب المفاصل الرثوى

علاء كريم محمد[1]، اسراء سالم محمد[١]، يحيى يحيى زكى فريد[٢]

الخلاصة:

خلفية الدراسة: من المعلوم ان مستويات حامض السايليك الكلي و الدهن المرتبط بحامض السايليك تتغير بمختلف الامراض بضمنها التهاب المفاصل الرثوى و حالات التهيج المختلفة، لذلك تثمين تلك المركبات فى مصل مرضى التهاب المفاصل الرثوى ربما توضح العلاقة بين مستواها و بين فاعلية المرض.

هدف الدراسة: الدراسة مصممة لتثمين التطبيق السريرى لمستويات حامض السايليك الكلى و الدهن المرتبط بحامض السايليك فى مصل مرضى التهاب المفاصل الرثوى مع الاخذ بنظر الاعتبار فاعلية المرض و مقارنة المستويات مع مجموعة الاصحاء.

طريقة العمل: اجريت الدراسة على ٩٧ شخصا مقسمين الى اربعة و خمسون مريضا(٣٧ اناث، ١٧ ذكور) يعانون من مرض التهاب المفاصل الرثوى و مقسمين الى ٢٩ مريضا (٢١ اناث، ٨ ذكور) بمدى عمر يتراوح بين (٣٢ —٦٥سنة) و لديهم فعالية قليلة للمرض و ٢٥ مريضا (١٦ اناث، ٩ ذكور) بمدى عمر يتراوح بين (٢٢–٥٢ سنة) و لديهم فعالية مرتفعة للمرض و مقارنة النتائج مع ٤٣ شخص سليم بمدى عمر يتراوح بين (١٩–٦٤ سنة). تم اعتماد الطريقة اللونية لتقدير كلا الحامضين فى مصل النماذج.

النتائج: تزداد مستويات حامض السايليك و الدهن المرتبط بحامض السايليك فى مصل مرضى التهاب المفاصل الرثوى عند مقارنتها مع مصل مجموعة الاصحاء مع زيادة ملحوظة عند المرضى ذو الفاعلية العالية للمرض.

الاستنتاج: اعتمادا على الدراسة، يمكن اعتبار مستوى حامض السايليك و الدهن المرتبط بحامض السايليك كمؤشر جيد للتميز بين فاعلية مرضى التهاب المفاصل الرثوى.

مفتاح الكلمة: التهاب المفاصل الرثوى، حامض السايليك، الدهن المرتبط بحامض السايليك

[١] قسم الكيمياء (كلية التربية-ابن الهيثم-جامعة بغداد)
[٢] قسم الكيمياء والكيمياء الحياتية (كلية الطب- جامعة النهرين)

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الملخصات العربية

دور تقصي لطاخات عنق الرحم في التشخيص المبكر للافات

ميسلون محمد العاني[1]، عبدالحسين الهادي[1] ،فائزة عفتان الراوي[2]

الخلاصة

خلفية الدراسة: تعتبر لطاخة بابانيكولاو لعنق الرحم من اكثر وسائل التقصي فاعلية لمنع سرطان عنق الرحم

هدف الدراسة: تهدف هذه الدراسة الى ايجاد معدل الانتشار للافات الالتهابية و ما قبل الخبيثة والخبيثة بين النساء و تحديد العوامل المتعلقة بمعدل انتشار هذه الافات

طريقة العمل: اجريت دراسة مقطعية على مدى ثلاثة اشهر شملت ٣٠٢ امراة مراجعة للعيادة الخارجية للنسائية و التوليد . تم استحصال المعلومات من كل مريضة و اجريت بعدها لطاخة عنق الرحم بعد اخذ موافقة المريضة

النتائج: كانت معدلات الانتشار للافات المختلفة كالاتي؛ التهاب عنق الرحم غير النوعي ١٨٨(٦٢ر٧٪)، الحؤول الصدفي ٢٣(٧ر٧٪)، داء المبيضات١١(٣ر٧٪)، الثدن الصدفي ٧ (٢ر٣٪)، المشعرات المهبلية ٤ (١ر٣٪)، حمة الحليموم البشري ٤(١ر٣٪)، اما الافات الناتجة من اللولب الرحمي لمنع الحمل فكانت ٤ (١ر٣٪). اثنان من اللطاخات كانت غير كافية و قد استثنتا من الدراسة

الاستنتاج: كانت معدلات الانتشار للافات المختلفة مقاربة للدراسات الاخرى. و لقد تبين وجود علاقة ذات معنى احصائي بين لطاخات عنق الرحم الالتهابية و بين المستوى الثقافي المنخفض، اتباع ممارسات تصحح خاطئة، حدوث التهابات تناسلية سابقة، و وجود اجهاض و تجريف رحمي سابق .

مفتاح الكلمات: لطاخات عنق الرحم، سرطان عنق الرحم، التهاب عنق الرحم

[1] فرع طب المجتمع (كلية الطب- جامعة النهرين)
[2] فرع الباثولوجي (كلية الطب- جامعة النهرين)

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الملخصات العربية

تقييم نسبة المعادن الضئيلة و متغيرات الدم المختلفة عند مرضى الثالاسيميا بنوعيه الرئيسي و النوعي

صبح سالم المدلل[1]، سعد شوقي منصور[1]، يحيى يحيى زكي فريد[2]

الخلاصـة

خلفية الدراسة: ان ملاض الثالاسيميا هو من الامراض الوراثية التي تتسبب في تحلل الدم. لقد اجريت عدة دراسات على مرض الثالاسيميا بنوعيه الرئبسي و الثانوي، و وجد ان هناك فروق في نسب المعادن الثقيلة و خصوصا لمعدن الزنك و النحاس و المغنيسيوم. و وجد ان هذه التغيرات مهمة في تفسير عدد من الظواهر التي تميز هذا المرض.

هدف الدراسة: تقييم مستوى معدن الزنك و المغنيسيوم في مصل المصابين بالثالاسيميا بنوعيه الرئيسي و الثانوي و كذلك تقييم متغيرات الدم المختلفة و نسبة الفيرتين في مصل الدم عند هؤلاء المرضى .

طريقة العمل: تم فحص ٧٢ مريضا مصابا بمرض الثالاسيميا الرئيسي و ١٧ مريضا مصابا بمرض الثالاسيميا الثانوي و ٣٠ شخص سوي عوملوا كمجموعة طبيعية. تم قياس نسبة المعادن الضئيلة، خضاب الدم، مجموع حجم الكريات الحمر، معدل تركيز خضاب الدم في الكرية الحمراء، معدل حجم الكرية الحمراء و معدل نسبة خضاب الدم في الكرية الحمراء.

النتائج: أظهرت هذه الدراسة انخفاضا معنويا مهما في مستوى خضاب الدم و معدل تركيز خضاب الدم في المرية الحمراء عند مرضى الثالاسيميا الرئيسي مقارنة بالمجموعة الضابطة. بينما كان هناك زيادة في معدل حجم الكرية الحمراء و معدل خضاب الدم في الكرية الحمراء و كذلك معدل تركيز خضاب الدم عند مرضى الثالاسيميا الرئيسي مقارنة بمرض الثالاسيميا الثانوي. أما عند عند مرضى الثالاسيميا الثانوي كانت نسبة MCV ، MCH ، MCHC منخفضة و ذات أهمية احصائية مقارنة بالمجموعة الضابطة، بينما كان مجموع حجم الكريات الحمر (PCV) مرتفع و ذو أهمية إحصائية عند مقارنته بمرض الثالاسيميا الرئيسي و المجموعة الضابطة.

أظهرت هذه الدراسة ان مستوى معدني النحاس و الزنك كان مرتفع و ذا أهمية إحصائية عند مقارنته بالمجموعة الضابطة، و قد لوحظ ان زيادة نسبة النحاس عند هؤلاء المرضى هو اكثر من نسبة الزنك. أما النسبة لمعدن المغنيسيوم فقد كان منخفض و ذو أهمية احصائية عند مرضى الثالاسيميا الرئيسي فقط عند مقارنته بالمجموعة الضابطة.

وعند دراسة مرض الثالاسيميا الثانوي، وجد ان نسبة النحاس في المصل كان مرتفع عندهم و مهم معنويا عند مقارنته بالمجموعة الضابطة و لكنه كان منخفضا في مرضى الثالاسيميا الرئيسي. أما مستوى الفيرتين فقد كان مرتفع عند مرضى الثالاسيميا عند مقارنته بالمجموعة الضابطة و مرض الثالاسيميا الثانوي. و كذلك كان مرتفعا في مرضى الثالاسيميا الثانوي مقارنة بالمجموعة الضابطة. أظهرت هذه الدراسة ان نسبة الزنك و المغنيسيوم قد أظهرت علاقة معنوية موجبة مع مجموع حجوم الكريات الحمراء (PCV) عند مرضى الثالاسيميا الرئيسي فقط.

الاستنتاج: اوضحت هذه الدراسة علاقة قوية بين مستوى الزنك و المغنيسيوم في مصل المرضى وبين مجموع حجوم الكريات الحمر (PCV) عند مرضى الثالاسيميا الرئيسي، و هذا يؤكد على أهمية دراسة تأثير اعطاء هذان المعدنان و امكانية التقليل من فقر الدم وبالتالي اعطاء الدم لهؤلاء المرضى.

مفتاح الكلمات: المعادن الضئيلة، الثالاسيميا (الرئيسي و الثانوي)، متغيرات الدم.

[1] فرع الباثولوجي (كلية الطب- جامعة النهرين)
[2] فرع الكيمياء و الكيمياء الحياتية (كلية الطب- جامعة النهرين)

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الملخصات العربية

الفحص الخلوي بالرشف بالابرة الدقيقة للاكياس و الاورام الكيسية للمبيض ، دراسة (٣٩) حالة.

يعرب ادريس خطاب[١]، لقاء رياض موسى[٢]، علاء غني حسين[١]

الخلاصة:
خلفية الدراسة: استخدام الفحص الخلوي بالرشف بالابرة الدقيقة بشكل واسع في تشخيص الاكياس و الاورام الكيسية للمبيض، و هو يتميز بسرعة التشخيص، و ادنى تأثير على المريض و قلة الكلفة ويؤدي الى تشخيص مقبول نسباً و ذو حساسية و نوعية عاليتين .

هدف الدراسة: لتقييم الحساسية للفحص الخلوي باستخدام الابرة الدقيقة في تشخيص الاكياس و الاورام الكيسية للمبيض.
طريقة العمل: تم اجراء الفحص الخلوي بالرشف بالابرة الدقيقة تحت توجية الامواج فوق الصوتية لثلاث و تسعون حالة لنساء مصابة بأفات كيسية و اورام كيسية للمبيض تراوحت اعمارهن ما بين ٢١-٤٨ سنة.
النتائج: كان هناك ١٣ حالة غير ورمية مقابل ٢٦ حالة ورمية من مختلف التولدات النسجية و تشمل (النسيج الطلائي السطحي و الخلايا الاورامية) و معظمها اورام حميدة، فيما اظهرت ثلاث حالات اورام سرطانية خبيثة.
الاستنتاج: يعد الرشف باستخدام الابرة الدقيقة لتشخيص الافات الكيسية و الورمية الكيسية للمبيض فحصاً امينا، و مقبولا من الناحية التشخيصية و يعطي نتائج مقبولة فيما يخص الحساسية والنوعية.

مفتاح الكلمات: الفحص الخلوي بالرشف بالابرة الدقيقة، الافات الكيسية للمبيض.

[١] فرع الباثولوجي (كلية الطب- جامعة النهرين)
[٢] فرع النسئية و التوليد (كلية الطب- جامعة النهرين)

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الملخصات العربية

القيمة التنبئية للترسبات الصلدة في قعر العين للحوامل في الأشهر الثلاث الأخيرة من الحمل كمؤشر لارتفاع ضغط الدم و تباطؤ نموا الجنين و المضاعفات الأخرى

وسام أكرم اسماعيل[١]، أيناس طه احمد الحمداني[٢]، اكرم صادق[٣]

الخلاصة:

خلفية الدراسة: ان مقدمة الأرتعاج عادة ما يصاحبها ارتفاع ضغط الدم، زيادة في بروتين الأدرار و تورم في انسجة الجسم.

هدف الدراسة: البحث عن وجود الترسبات الصلدة في العين عند الأسبوع ٢٨ من الحمل قيل الأصابة بارتفاع ضغط الحمل بواسطة منظار العين البسيط.

طريقة العمل: تم اختيار مجموعة الدراسة المكونة من ٤٢ أمراه حامل بناء على وجود الترسبات الصلدة في العين عند الأسبوع ٢٨ من الحمل. و تم أيضا اختيار مجموعة ضابطة مكونة من ٤٦ أمراه حامل و اللواتي كانت أعينهن خالية من أي ترسبات عند الأسبوع ٢٨ من الحمل. تم متابعة المجموعتين و بشكل روتيني قي الأشهر الثلاث الأخيرة من الحمل و دونت أي مضاعفات فد تحدث.

النتائج: كان معدل ارتفاع ضغط الدم، قلة السائل الامنيوني و كذاك انفصال المشيمة قبل الولادة أكثر و بفارق إحصائي مميز في مجموعة الدراسة عن المعدلات في المجموعة الضابطة. و في نفس السياق كان معدل الولادات للخدج المصابين بتباطؤ النمو و معدل الوفيات خلال الأسبوع الأول من الحمل اكثر في مجموعة الدراسة.

الاستنتاج: أن فحص عين الحوامل في بداية الشهر السابع من الحمل لوجود الترسبات الصلدة قد يكون مفيدا لرصد الحوامل المعرضات لارتفاع ضغط الدم و المضاعفات المصاحبة.

مفتاح الكلمات: مقدمة الأرتعاج، ملتحمة العين، البروتين، الترسبات الصلدة

[١] قسم النسائية و التوليد (كلية طب الكندي-جامعة بغداد)
[٢] مستشفى العلوية للولادة (بغداد)
[٣] طب و جراحة العيون

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الملخصات العربية

تثبيت كسور الفك الاسفل بواسطة الصفائح التيتانيومية العظمية المصغرة

هيثم عبد العال زيارة[١]، عمار ياس خضر[٢]

الخلاصة

خلفية الدراسة: كان العلاج التقليدي و الشائع لكسور الفك الاسفل يعتمد على اعادة ترتيب الاطباق بواسطة التثبيت عن طريق ربط الفك الاسفل بالفك الاعلى، طريقة (IMF) يصحبها معوقات مثل فقدان الوزن و التداخل مع تنظيف الفم و ازعاج المريض. في السنوات الاخيرة و بشكل متزايد استخدم جراحو الوجه و الفكين طريقة التثبيت الداخلي في علاج كسور الفك السفلي. و استخدام صفائح التيتانيومية المصغرة اصبح شائعا في جراحة الوجه والفكين منذو ان طور (Champy) طريقة (Michelet).

هدف الدراسة: تقييم الصفائح التيتانيومية العظمية المصغرة في علاج كسور الفك السفلي و مقارنة النتائج، المحاسن، المساؤى، المضاعفات بين تثبيت من داخل و خارج الفم لصفائح التيتانيومية المصغرة.

طريقة العمل: انجزت هذه الدراسة على ٢٤ مريضا كانوا يعانون من ٢٨ كسرا في الفك الاسفل عولجت بواسطة التثبيت الداخلي و الصفائح التيتانيومية العظمية المصغرة. اجريت عملية التثبيت عن طريق داخل الفم لعشر مرضى و عن طريق خارج الفم لاربعة عشر مريضا.

النتائج: النسبة الكلية للمضاعفات في الدراسة كانت ٢٥٪. و كانت نسبة المضاعفات في المجموعة التي اجريت لهم عملية تثبيت من خارج الفم ٢١،٤ ٪ (٣ مرضى): تندب الوجه مريضان واطباق غير ملائم مريض واحد. و كانت نسبة المضاعفات في المجموعة التي اجريت لهم عملية تثبيت من داخل الفم ٣٠٪ (ثلاث مرضى): تنمل الشفة السفلى مريض واحد و عدوى ما بعد العملية مريض واحد و تضرر لجذر السن مريض واحد.

الاستنتاج: ربط العظام بواسطة الصفائح العظمية تعطي نتائج مقبولة و يمكن التوصية باستخدامها كطريقة روتينية في علاج كسور الفك السفلي. التثبيت عن طريق داخل الفم اكثر فائدة و يعطي نتائج قابلة للمقارنة مع التثبيت عن طريق خارج الفم. الخبرة هي عامل اساسي في النتائج.

مفتاح الكلمات: حوادث، كسور الفك السفلي، تثبيت بواسطة الصفائح التيتانيومية المصغرة.

[١] فرع الجراحة –الوجه و الفكين (كلية الطب- جامعة النهرين)
[٢] عيادة طب الأسنان (مستشفى الكاظمية التعليمي-بغداد)

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الملخصات العربية

حدوث دوالي الخصية في الرجال العقيمين كما وجد بالفحص السريري ، و فحص الموجات فوق الصوتية الاعتيادي و الدوبلر الملون

اسامة الناصري[١]، فواز انيس رسام[٢]

الخلاصة:

خلفية الدراسة: تتيجة للعلاقة الوثيقة بين دوالي الخصية و عقم الرجال واحتمال ازدياد الخصوبة بعد رفع الدوالى بالتداخل الجراحي اصبح هناك اهتماما واضحا في تشخيص الدوالي بطريقة غير مضرة بواسطة الدوبلر الملون اعتمادا على المواصفات التالية :

(ا) توسع الاوردة المنوية فوق ٢ ملم (ب).رجوع الدم الى الاسفل في الاوردة المنوية إلى الأسفل في وضع الوقوف بغض النظر عن حجمها.

هدف الدراسة: لتقييم أهمية الدوبلر الملون في تشخيص دوالي الخصية مقارنة بالسونار الاعتيادي و الفحص السريري.

طريقة العمل: اجريت دراسة لمائة رجل عقيم يشتبه سريريا باصابتهم بدوالي الخصية اذ احيلوا للفحص بالموجات فوق الصوتيه الاعتيادي و الدوبلر الملون لتاكيد او استبعاد التشخيص او استبعاد عودة الدوالي بعد إجراء التداخل الجراحي.

النتائج: أثبتت الدراسة ان الدوبلر الملون بضمنه فحص الموجات فوق الصوتيه الاعتيادي ادى الى زيادة في نسبة تشخيص دوالي الخصية غير المشخصة سريريا بنسبة ٢٢٪ و هي نسبة كبيرة و كان مهما في تصحيح التشخيص السريري الخاطى للدوالي في ٦٪ من المرضى. كان للدوبلر الملون بضمنه الفحص بالموجات فوق الصوتيه الاعتيادي اهمية كبيرة في تاكيد التشخيص السريري للدوالي في ٦٢٪ من المرضى كما كان مهما في تشخيص الدوالي غير المشخصة سريريا في ٥ ٪ من المرضى و اكتشاف عودة الدوالي بعد التداخل الجراحي في ٣٧

الاستنتاج: اصبح الدوبلر الملون المرجع المثالي غير المضر في تشخيص دوالي الخصية و متابعة المرضى بعد اجراء التداخل الجراحي.

مفتاح الكلمات: دوالي الخصية ، الدوبلر الملون.

[١] فرع الجراحة (كلية الطب- جامعة النهرين)
[٢] أختصاصي الأشعة (متقاعد)

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الملخصات العربية

شيوع مرض باركنسن في منطقة الكاظمية من مدينة بغداد

عبد المطلب عبد الكريم[1]، أمجد نيازي[2]، عمار فاضل[3]

الخلاصة

خلفية الدراسة: مرض باركنسن هو اكثر الأمراض المزمنة النمطية التي تصيب الجهاز العصبي، يصيب الأفراد من الأعمار اكثر من ٤٠ سنة. دراسة شيوع المرض يشكل ركيزة اساسية للتخطيط الطبي خصوصا" أن أعمار الأفراد في معظم المجتمعات في تزايد. هناك بعض الأختلافات في نسبة شيوع المرض في المجتمعات المختلفة في العالم و النسبة غير معروفة في بلادنا.

هدف الدراسة: معرفة نسبة الأصابة بالمرض في منطقة الكاظمية.

طريقة العمل: الدراسة معتمدة على الكتلة الأجتماعية أقيمت بشكل مسحي بمشاهدة المرض مرة واحدة من العينة التي أختيرت عشوائيا" من المنطقة. المرضى الذين يشك في أصابتهم بالمرض يحالون الى المستشفى التعليمي في الكاظمية لغرض تثبيت التشخيص بهذا المرض من قبل اختصاصي طب الجملة العصبية. المرض يشخص سريريا" و تدرس الحالة لإستبعاد الأحتمالات الأخرى.

النتائج: ٢٥ مريض مصاب بمرض باركنسن جمعت من عينة عشوائية ل (٢٢٩٨٨) شخص (١٣ ذكور و ٢٣ أناث). (٦) من منطقة ريفية و (١٩) من منطقة حضرية. ثلاث حالات شخصت لأول مرة. الأرتعاش هو اكثر العلامات شيوعا" (٨٠٪) . (١٩) مريض الأصابة كانت في الجهتين على الرغم من أن بداية المرض كانت في جهة واحدة . نسبة شيوع المرض تزداد بتقدم الأعمار. نسبة شيوع المرض كانت ١٠٨,٧٥ لكل ١٠٠٠٠٠ شخص. عند الأناث كانت ١٠٣ لكل ١٠٠٠٠٠ شخص و عند الذكور ١١٤ لكل ١٠٠٠٠٠ شخص. النسبة كانت ١١٤,٣ لكل ١٠٠٠٠٠ شخص للساكنين في مناطق حضرية و ٩٤,٣ لكل١٠٠٠٠٠ شخص للساكنين في الريف.

الأستنتاج: نسبة شيوع المرض في بلادنا اقل بقليل من الولايات المتحدة و اوربا لكنها أكثر بقليل من افريقيا و الصين. النسبة تزداد بتقدم العمر. و لا يوجد فرق مهم في نسبة الشيوع بين الأناث و الذكور او بالنسبة للسكن في الريف او الحضر. هذه النسبة يمكن ان تعطى لعموم بغداد و ذلك لتشابة بغداد و الكاظمية من حيث التركيبة السكانية.

مفتاح الكلمات : مرض باركنسن، شيوع

[1] فرع الطب الباطني (كلية الطب- جامعة النهرين)
[2] الهيئة العراقية للأختصاصات الطبية
[3] مستشفى الحلة العام

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تقييم قياس الهيموغلوبين باستخدام طريقة الصوديوم لوريل سولفيت

سعد شوقي منصور[1]، رعد جابر العاني[1]، محمد جابر خضر[2]

الخلاصــة

خلفية الدراسة: هنالك عدة طرق تطورت خلال القرن الماضي لقياس كمية الهيموغلوبين، اعتمدت على الخصائص الكيميائية و الفيزيائية للهيموغلوبين، الطريقة الغازية أو الطبقية. طريقة السيان – ميتهيموغلوبين.

هدف الدراسة: لأعتماد هذه الطريقة

طريقة العمل: لقد تم قياس الهيموغلوبين الطبقي الضوئي بطريقة السيانو هيموغلوبين و الطريقة الخالية من السيانيد (صوديوم لوريل سولفيت). قد تمت القياسات على ٣٤٧ عينة دم وريدي وضعت في مانع التخثر K2EDTA. العينات الطبيعية أُخذت من متبرعين و العينات الأخرى أخذت من نماذج المرضى في الخدمة الروتينية التشخيصية و التي تشمل: إبيضاض الدم مع ارتفاع كريات الدم البيض، تدهن الدم، دم الحبل السري، نماذج أخرى غير طبيعية (تبولن الدم، اليرقان، فقر الدم البحر الأبيض المتوسط البسيط و العظيم)، ميتهيموغلوبين. و بعد ذلك تم تحديد كمية الهيموغلوبين باستخدام طريقة الهيموغلوبينوسيانيد و طريقة الصوديوم لوريل سولفيت.

النتائج: أعلى امتصاص كان عند ٥٣٥ نانوميتر. و قد تحول الهيموغلوبين إلى صوديوم لوريل سولفيت– هيموغلوبين بشكل سريع جداً أقل من (١٥ ثانية). صوديوم لوريل سولفيت – هيموغلوبين المحضر آنياً يبقى مستقر خلال أول ١٢٠ دقيقة بعد التخفيف، و الهيموغلوبين تحول فورياً إلى صوديوم لوريل سولفيت – هيموغلوبين. هنالك علاقة طردية بين الامتصاص و تركيز الهيموغلوبين لسلسلة قياسات واسعة. الحول على طريقة الصوديوم لوريل سولفيت في نماذج الدم الروتينية هو مساوٍ بالنسبة لطريقة الهيموغلوبين سيانيد، أكثر قليلاً عند وجود عائق بتدهن الدم. لم يكن هنالك فرق واضح في قياس النماذج التي تحتوي على الهيموغلوبين F. (معامل الارتباط = ٩٩٥ر٠).

الأستنتاج: الصوديوم لوريل سولفيت قاس الميتهيموغلوبين (معامل الارتباط = ٩٩٩ر٠).الصوديوم لوريل سولفيت يملك فائدة عظيمة وهي ان الكاشف مركب ليس فيه خطورة.

مفتاح الكلمات: هيموغلوبين، صوديوم لوريل

[1]قسم الباثولوجي (كلية الطب جامعة النهرين)

[2] مستشفى الكندي التعليمي (وزارة الصحة)

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الملخصات العربية

دراسة سريرية، و تحليل للعوامل المؤثرة في نتائج العلاج الجراحي لأورام الغدة النخامية

سمير حسن عبود، سرمد عزيز ابراهيم، عبدالامير جاسم

الخلاصة

خلفية الدراسة: أورام الغدة النخامية اورام طلائية حميدة تكون حوالي ١٠٪ من الاورام داخل الجمجمة. اعراضها تدهور النظر أو اختلال افراز الهورمونات، او كليهما. للحصول على افضل النتائج في علاج هذه الاورام الحميدة يجب الاهتمام بالتفاصيل المميزة لها.

هدف الدراسة: مراجعة الخواص السريرية والشعاعية لهذه الاورام، والتعرف على العوامل المؤثرة في نتائج العلاج الجراحي.

طريقة البحث: دراسه رجعية للمرضى المصابين بورم النخامية خلال فترة اربعة اعوام، من خلال مراجعة طبلاتهم و متابعة حالتهم لفترة ١–٤ اعوام.

النتائج: عدد المرضى ٥٥ (٣١ ذكر ٢٤ انثى) متوسط الاعمار كان ٣٧ سنة، الاعرا ض الابتدائية كانت تدهور النظر ٤٩٪، اعراض اختلال الهورمونات ٣٥,٥٪،الصداع ١٤,٥٪، اضطراب التصرف ١,٨٪ و الصرع في ١,٨٪ من الحالات. ألاضطراب الهورموني الرئيسي كان زيادة افراز هورمون الحليب ٣٢,٧٪. اشعة الجمجمة الجانبية اظهرت توسع من الدرجة (٣) في السرج التركي مع تنخر في ٨٧,٢٪ من الحالات. مفراس الدماغ اظهر وجود كتلة مساوية الكثافة في ٧٦٪ من الحالات مع وجود امتداد الى ما فوق السرج التركي في ٨٢,٦٪ و تظهير بالصبغة في ٨٥,٧٪ من الحالات. عولج ٤٦ مريضا جراحيا بفتح الجمجمة (٨٣,٦٪)، و ٩ مرضى عن طريق الانف (١٦,٣٪). استؤصل الورم من داخل الكبسولة (شبه تام) في ٧٠,٩٪ من الحالات و استؤصل الورم بصورة تامة في ٢٩,١٪ من الحالات. توفي (٣) مرضى و رجع الورم لدى ثلاثة اخرين كانت اورامهم عنيفة، و قد عولج اثنان منهم بالاشعة العميقة. أثنان من اللذين عولجو بالاشعة العميقة اصيبو باورام في الدماغ.

الاستنتاج: نتائج البحث كانت متوافقة مع البحوث المنشورة في الموضوع. أورام الغدة النخامية حميدة في معظم الحالات منخفضة الدموية و لينة، ذات صفات سريرية و شعاعية مميزة. في ما عدا الحالات المتقدمة من ضمور العصب البصري و فقدان البصر، كان هناك تحسن ممتاز في النظر بعد العملية. السكر الكاذب المؤقت كان ابرز المضاعفات بعد العمليات من الانف. العامل الوحيد المؤثر في رجوع المرض هو طبيعة الورم العنيفة. العلاج بالاشعة العميقة يتوجب فقط في الاورام العنبفة.

مفتاح الكلمات: الغده، النخاميه، نتائج العلاج

فرع الجراحة (كلية الطب- جامعة النهرين)

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الملخصات العربية

اساليب التغلب على المشاكل و المصاعب لدى المراهقين المصابين بداء السكر نوع ١

جواد كاظم الديوان

الملخص:
خلفية الدراسة: لم يتم دراسة التطور في جهد المراهقين المصابين بداء السكر نوع ١ في التغلب على المشاكل و المصاعب في العراق.
هدف الدراسة: تقييم العلاقة بين اساليب التغلب على المشاكل و المصاعب لدى المراهقين المصابين بداء السكر و ادراك خطورة مرض السكر.
طريقة العمل: شملت الدراسة ١٦٠ مراهقا مصابا بداء السكر نوع ١ من مختلف مراكز معالجة داء السكر في بغداد للفترة ١ / ٦ الى ٢١ / ١٢ / ٢٠٠٠. تمت مقابلة كل مراهق بشكل منفصل. المعلومات التي تم جمعها شملت العمر و الجنس و فترة الاصابة بداء السكر و ممارسة الرياضة و عدد الزيارات لداء السكر و مقياس القلق و الاضطراب لداء السكر (ادراك خطورة مرض السكر). و تم تقييم اساليب التغلب على المشاكل و المصاعب (الفاعلة و العدائية) بمقاييس مختارة. تأثير اساليب التغلب على المشاكل و المصاعب و غيرها من المتغيرات على القلق و الاضطراب من داء السكر (ادراك خطورة داء السكر) تم تقيمها بالانحدار المتعدد.
النتائج: الادراك لخطورة مرض داء السكر ارتبط معنويا مع اساليب التغلب على المشاكل (الفاعل و العدائي) و الاسناد العاطفي و الجنس. لم يلاحظ ارتباط ذا قيمة معنوية للعمر و الزيارات لعيادة السكر مع اساليب التغلب على المشاكل والمصاعب.
الاستنتاجات: اظهرت الدراسة اهمية اساليب التغلب على المشاكل و المصاعب لادراك خطورة داء السكر و الذي بدوره يؤثر على السيطرة و المعالجة بداء السكر.

مفتاح الكلمات: مراهقون مصابون بداء السكر، اساليب التغلب على المشاكل، العراق

قسم طب المجتمع (كلية الطب-جامعة الأنبار)

المجلة العراقية للعلوم الطبية ٢٠٠٥ م المجلد ٤ العدد ٢ ص ٢١٠-٢١٣

الملخصات العربية

ألتقييم الشخصياتي و الكمي لخزع نقي العظم في مرض تليف نقي العظم الابتدائي

احلام المشهداني[1]، سعد شوقي منصور[2]، فالح سالم سرحان[2]

الخلاصة :

خلفية الدراسة : ان مرض تليف نقي العظم الابتدائي و الذي يعتبر احد الاضطرابات التكاثرية النخاعية غير الابيضاضية، يتميز بحدوث درجات مختلفة من تليف نقي العظام و التي تشكل العلاقة المميزة و التشخيصية للمرضى. لهذا السبب فأن دراسة خزع نقي العظم تعتبر من أهم الخطوات في تشخيص المرض.

هدف الدراسة : اعادة تقييم خزع نقي العظم في المرضى المصابين بتليف نقي العظم الابتدائي.

طريقة العمل : اعيد في هذه الدراسة تقييم خزع نقي العظم لـ (٣٠) مريضاً بتليف نخاع العظم الابتدائي. اعيد تقطيع كتل البارافين و تجهيز شرائح جديدة و صبغها بصبغة الهيماتوكسلين و الايوزين، صبغة الالياف الشبكية و صبغة الحديد. اجري نوعان من التحليل على الشرائح احدهما شخصياتيا و الاخر كمياً. كذلك لقد تم الاعتماد على طريقة كولون في تصنيف المرضى الى اربعة مجاميع.

النتائج : بينت الدراسة ان اكثر العلامات ثبوتيا هي العلامات الخاصة بخلايا النواء كزيادة عددها و وجودها على شكل تجمعات. بالاضافة الى ذلك اثبتت الدراسة ان اغلب المرضى اظهروا زيادة في عدد خلايا النخاع و الالياف الشبكية و نقصان كمية الحديد في النقي العظمي. كذلك تم اثبات وجود زيادة في عرض العظم الحويجري، زيادة مؤشر ليانية العظم و زيادة الاوعية الدموية.

الاستنتاج : ان الدراسة المستفيضة لخزع نقي العظم مع ضمان كمية كافية من العينة النسيجية و استعمال صبغات الالياف الشبكية و الحديد بالاضافة الى الصبغة الاعتيادية (الهيماتوكسيلين و الايوزين) تعتبر هامة جداً في التشخيص و التقسيم المرحلي لمرضى تليف نقي العظم الابتدائي.

مفتاح الكلمات: تليف نقي العظم الابتدائي،خزع نقي العظم، تشخصياتي، كمي

[1] مستشفى النور (وزارة الصحة)
[2] فرع الباثولوجي (كلية الطب-جامعة النهرين)

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حكمت عبد الرســـــول حاتم

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اسامة الناصـــــــــــــري	علاء غني حســـــــــــــين
أنعم رشــــيد الصالحـــي	غسان الشـــــــــــماع
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عقيل جبار البهادلــــــــي	ر
فرق د ب در حم دان	محـــــــــــــر
	ر
	محـــــــــــــرر

سكرتارية المجلة

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MEDICAL PRACTICE: IS THE TEXTBOOK ALWAYS RIGHT?

Kahlid Abdulla *FRCP*

Most practicing doctors are aware of the gap between their practice and the book. They usually regard what the book states as the correct thing and anything short of or different from it as incorrect or inferior. Doctors working in highly developed institutions in developed countries, which frequently provide tertiary care, usually write chapters of textbooks. The situation in most hospitals in developing countries and in small clinics is quite different.

The aim of the doctor is to do what is in the best interest of his patients in the environment he or she is working in. This should be the yardstick by which a certain behavior is judged as right or wrong. What is an appropriate decision in one place may be inappropriate in another. Sticking to the book is not always the proper behavior. Following are examples of this at various stages of the relationship between doctor and patient.

Physical Examination

Women are a special problem in our community when the examiner is a man. They frequently resist exposing parts of their body. Some may request to be examined with their cloths on. The doctor has then two choices. One is to refer the patient to a female doctor. The patient usually does not like that either because she trusts the doctor she has come to and wants to be treated by him or because of the inconvenience of having to go to a different place and have another booking. The second choice is to do what the patient wants and examine her with clothes on.

The doctor has to judge whether such an examination is acceptable in the circumstances. Examining the abdomen with a thin internal cloth on does not interfere significantly with palpation but it interferes with inspection. In some cases, it may be reasonable to assume that this is unlikely to affect your judgment and doing it should not be considered wrong. Taking blood pressure with a thin sleeve on usually does not interfere with measurement[1] and doing it to a patient who wears a sleeve that cannot be wrapped up is another example.

Investigations

Many tests done in our institutions are not as accurate as they are described in the book. The values given to them in the book in the form of sensitivity, specificity, predictive values and likelihood ratios may be quite different from the values they have in our actual practice. Their weight in the diagnostic and management process may consequently be considerably less than the weight given to them in the book.

In other words, their value compared to the information obtained by history taking and physical examination may be considerably less than what is stated in the book. This should always be kept in mind in making a final judgment on diagnosis and management. It should also reflects on the decision to do the test in certain situations when the probability of a diagnosis or a management action built on clinical criteria (pretest odds) is high and the likelihood of it being affected by the test result is low (because the likelihood ratio of the test is low). For example, if you have a very strong suspicion of typhoid fever on clinical grounds and your laboratory is not reliable, it may be prudent to treat the patient without wasting time and money by asking for a Widal test.

Treatment

A certain treatment, especially complicated procedures, is advised when it is judged that its possible benefits outweigh its possible harms for the patient concerned. Consequently, it should be judged according to the situation in the place of practice. It should not follow the instructions of the book blindly. Those who write book chapters usually work, as stated earlier, in advanced institutions with higher expertise and better facilities.

The results of various therapeutic procedures and their complications are not the same as they are in less developed places. Consequently, the balance between benefits and risks is different. So, a treatment, which according to the book is indicated in a particular situation, may not be indicated for the same situation in the place one is working in. Dialysis is an example. In a place where maintenance dialysis is good with few complications and a reasonable quality of life of patients, one may advice patients with chronic renal failure to go on maintenance dialysis when their creatinine clearance comes down to 10m./min. One would expect their life on dialysis to be better than it is without it and their long-term prognosis better.

In another place where the quality of maintenance dialysis is poor and complications are many, one tend to wait longer until the patients' condition become severe enough so that their life on dialysis in spite of its poor quality and frequent complications represents an improvement on their life without it. The policy may then be to wait until creatinine clearance comes down to 5 ml/min. before putting the patient on maintenance dialysis as it is indeed the case in less developed parts of the world[2].

References

1. Hovsrpian R, Al-Haddad M, and Abdulla K: Comparison of blood pressure measurements in bare arm, clothed arm and forearm. J Fac Med Baghdad, 1996; 38: 221-4.
2. Swyter J: International ESRD experience: An anthropological perspective. Dialysis and Transplantation, 1985; 14: 328-38.

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ARABIC ABSTRACTS

في ذمة الله

بمزيد من الحزن والأسى تنعى المجلة العراقية للعلوم الطبية الزميلين الفقيدين الأستاذ المساعد الدكتور محمد فلاح الجزائري والأستاذ المساعد الدكتور عقيل جبار البهادلي أعضاء الهيئة التدريسية في كلية الطب-جامعة النهرين الذين استشهدا بنار الغدر والعدوان. أن فقدانهما لخسارة كبيرة لطلابهما و زملائهما ووطنهما. نسأل الله تعالى أن يتغمدهما برحمته ويسكنهما فسيح جناته ويلهم ذويهما الصبر والسلوان و انا لله وانا إليه راجعون.

نبذة عن حياة الفقيدين الغاليين