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General The Dohuk Medical Journal is a signatory journal to the uniform requirement for manuscripts submitted to biomedical journals, February 2006 (<http://www.icmje.org>).

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Dear Colleagues

I am proud to introduce to you the first issue of Dohuk Medical Journal, the official journal of Dohuk College of Medicine. The college is one of the three medical schools in Kurdistan region / Iraq. It was established in 1992 to accomplish a mission of providing well trained doctors to the community. The mission also aimed to activating scientific researches in the field of medicine and health.

I hope that the journal will be useful in distributing and sharing knowledge, views and experiences among personnel involved in providing health services to the community.

**Dr. Farhad K. Sulayvani
MBChB, CABS, FRCS
Dean, Dohuk College of Medicine,
University of Dohuk**



Dohuk Medical Journal

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EDITORIAL

SPECIALIZATION IN FAMILY MEDICINE; A NECESSITY OR A FASHION

SAMIM A. AL-DABBAGH, MBChB, DTM&H, D. Phil (Oxon.)*

DMJ 2007;1(1):1-3.

For more than one hundred years ago, to be a family doctor was often a lonesome, competitive occupation; single handed practitioner, not especially well trained worked alone for long hours, doing almost every thing themselves with few instruments, and with little or no continuing education. They had meager encouragement apart from love of their work and gratitude of their patients.^{1,2}

Over the years, patterns of diseases have changed. Medicine became more complicated and expensive, with more efficient diagnosis and treatment. The specialists have increased in number. In the last decades also primary care has developed around these services historically provided by the general practitioner. Since Alma-Ata declaration, primary health care has been considered to be the first level of contact of individuals with the national health system bringing health care as close as possible to where people live and work and constitutes the first element of continuing health care process.³

Family medicine has frequently been referred to as the key element of primary

care system. A number of medical academic bodies from Europe collaborated to produce a definition of the work of a general practitioner which was subsequently adopted in its entirety as a policy of the United Kingdom Royal College of General Practitioner, and often referred to as the "Leeuwenhorst definition".⁴ The European model of primary medical care has tended to emphasize the importance of first contact (primary care) and of generalism (general practice). American and Canadian practice, on the other hand, emphasizes the family context of care. The term "generalist" is not accepted as it apparently has a pejorative connotation. Hence the terms "family practice" or family health care" are commonly used to distinguish primary generalist services from those of specialists.⁵ Both schools, however, have emphasized the importance of specialization in general practice or family medicine.

Family medicine continues to struggle for a legitimate place in academic medicine. It has been in conflict about whether it is a specialty or a generalist discipline. Throughout the history, the ideological distinction between generalist and specialist has created a great divide. This polarity has been played out in the

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distinction between family doctor, the new breed of specialist, and general practitioner, the old guard. The specialty has struggled from its inception to possess something of high status that is uniquely its own, not borrowed, not grafted.^{6,7} Whatever the name of this specialization is, it is still unfortunately misunderstood by many individuals within and outside the medical practice.⁷ Family medicine is not a summation of the whole range of specialties. It has, however, special factors and attributes of its own and complements specialist practice. Accordingly the role of a specialist in family medicine is not limited to treating simple cases and referring others to hospitals. Scientific studies show that family physicians could competently and efficiently deal with more than 85% of the health problems of families.⁸ It should not be looked at as a specialization suitable only for developing countries. The practice of family medicine is indeed more applied in developed than developing countries. The specialist in family medicine is the one responsible to provide continuing and comprehensive health care for the individuals and families. It is the specialty with breadth, which integrates the biological, clinical and behavioral sciences. The scope of the practice should encompass all ages, sexes and types of illness.

Looking at this concept in a deeper view, a specialist in family medicine is considered as the one who is specialized in providing preventive, curative and rehabilitation care for all family members, males and females, young and old. He will make an initial decision about every case

and undertake continuing management for his patients with acute, chronic, recurrent or terminal illnesses. The family physician looks at the patient from a wider scale. He does not segregate between a somatic or a psychiatric illness and he should pay attention to the social aspects of the illness. He also has continuous and deep relations with family members during health and disease. Moreover, prolonged contact means that he can use this to gather information and build up a relationship of trust which he can use professionally. He also will practice in cooperation with other colleagues, medical and non-medical. This means that he will treat the family members when they are suffering from illness and he will refer those needing further management. He will also be responsible for arranging the referral with a feedback. A family physician might also be carrying or arranging for home care for his patients. The procedure of referral, by it, should be well organized and there ought to be a feedback from the point of destination. This will not only help the family physician to get the needed information about his patient, but it also helps his patients to get the benefit of that consultation.⁸

Looking forward to this millennium as a time of both challenge and opportunity; confidently expects that new medical roles requiring new competencies will continue to emerge. This depends heavily on interpersonal communication and leadership skills.⁹ All countries, all over the world, are paying more attention towards activating all aspects towards

improving health and health standards of their community members. Several obstacles are facing their goal. Among them is that the health service system is emphasizing curative services, meeting demands rather than actual needs, lack health team approach, with inadequate community participation and insufficient integration between health and socio-economic development. Family physicians might be able to breach most of these obstacles. The general practice characteristics are accessibility, availability, comprehensiveness, and continuing responsibility and long term doctor-patient relationship.⁵ Moreover the traditional view of the general practice has changed and the adequacy of symptomatic management approach is being questioned. The specialization in family medicine, therefore, has a vital role as a link between self-care and professional medical care profoundly influencing the overall level of care and the use of resources for providing a complete primary health care which is the milestone of any health care system. Recently in Iraq, there has been an increasing emphasis on this specialization. It is hoped that doctors specialized in family medicine will be present in all primary health care centers in the near future.

REFERENCES

1. Hunt JH. The future of general practice. *Practitioner* 1968;201: 94-105.
2. Elliott BC. An analysis of lay medicine. *J Royal Coll Gen Pract* 1986;36:542.
3. World Health Organization. Study group on community involvement on health development: challenging health services. *Tech Rep Ser* 1991; (No. 809).
4. Royal College of General Practitioner. The Work of the general practitioner: statement by a working party of the second European conference on the teaching of general practice. London, 1977:117.
5. Henley DE. A trial of increased access to primary care. *N Engl J Med* 1996;335(12):895-8.
6. Stein HF. Family medicine's identity: being generalist in specialist culture? *Ann Fam Med* 2006;4:455-9.
7. Welsh Council of the Royal College of General Practitioner and the Welsh General Medical Services Committee. Patient care and the general practitioner. *BMJ* 1994;301:1144-7.
8. Kurashi NY. Patient referral versus coordinating patient's health care. *Saudi J Fam Com Med* 1999;5(2): 13-4.
9. Tweed WA. Medical education for the next millennium, the medium and the message. *Bahrain Med Bull* 1998;20:25-7.

PERINATAL MORTALITY IN AZADI TEACHING HOSPITAL, DOHUK CITY, IRAQ

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Submitted 1 November 2006; accepted 26 February 2007

ABSTRACT

Background Still birth and perinatal mortality has always been a major problematic issue in clinical practice. They a devastating experience for the parents specially the mother. Few local studies have addressed these problems.

Objectives Estimation of the perinatal mortality rate and studying the major local etiological and predisposing factors in order to highlight the preventive strategies and form a base for future plans.

Design and setting A descriptive study was carried out for all perinatal deaths from 1st January to 31st December 2004 at Azadi teaching hospital in Dohuk city, Kurdistan region, Iraq.

Results During the study period, the number of total births recorded was 10879. There were 427 perinatal deaths giving a perinatal mortality rate of 39.2 per 1000 births. The ratio of fresh stillbirths to macerated cases was 2/1(126 and 60) respectively. The direct leading causes behind fresh stillbirths were placental abruption (25.4%), pre-eclampsia (16.7%), and congenital anomalies (14.3%). The main causes of maceration were pre-eclampsia (33.3%), diabetes mellitus (21.6%) and congenital anomalies (20%). Prematurity (46.9%), birth asphyxia (19.9%) and congenital anomalies (12.4%) were strongly associated with early neonatal deaths (241 cases). A significantly higher risk of perinatal death was observed among women under 20 or over 40 years of age, grand-multiparty, low socioeconomic status, and poor antenatal care attendance.

Conclusions The profile of perinatal deaths among the studied group suggests the need for improvement in obstetric care, early referral, and availability of advanced neonatal care services including neonatal screening protocols.

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Key words: Perinatal mortality, Incidence, Types, Predisposing factors

Perinatal mortality is an important indicator of obstetric care, health

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status and socioeconomic development.^{1,2}

It is also used as an indicator of reproductive health world wide.³ Perinatal mortality has been more difficult to prevent than infant mortality and has only recently received global attention. Being closely linked to maternal outcomes, perinatal mortality can be used as a proxy indicator for maternal health care.⁴ The perinatal mortality rate varies with the quality and degree of utilization of antenatal care and perinatal services.⁵

The World Health Organization had estimated that perinatal conditions took more than 2.4 million lives in the year 2000, representing 4.4% of all deaths in the world.⁶ In Africa, perinatal mortality rate is estimated at 75 per 1000 births and ranged from 36 - 74 per 1000 births in Asia.¹ In England and Wales, stillbirths were registered for the first time in 1928. In 1949, they were combined with early neonatal deaths to form the perinatal mortality rate, which was then found to be 38.1 per 1000 total births.⁷ This combination of stillbirths and early neonatal deaths was later adopted internationally in an effort to permit valid international, regional and temporal comparisons.⁸

Earlier studies carried out in both developed and developing countries had revealed a large number of risk factors with respect to perinatal mortality. Some of these risk factors are related to the mother, others to the child. Perinatal deaths are largely the result of poor maternal health, adverse social conditions, and inadequate care during pregnancy, delivery, and the immediate postpartum period.⁹ Studies of individual cases of death in various European countries during 1993-1998 have shown that perinatal deaths are related to suboptimal care before, during and after childbirth. Richardus et al found that four out of ten deaths are attributed to inadequate care.¹⁰

Stillbirths and early neonatal deaths differ substantially with respect to their principle causes, although conditions such as birth asphyxia and abruptio placenta play a major role for both stillbirths and

early neonatal deaths.⁸ The etiological determinants differ widely according to whether stillbirth occurs before or during labor. Ante-partum stillbirths are often combined with severe maternal, placental or fetal abnormalities, including pre-eclampsia, diabetes mellitus and congenital anomalies, while intrapartum fetal death is usually the result of fetal distress and /or obstructed labor.¹¹

In our region, the estimation of data on the frequency, distribution and predisposing factors of adverse birth outcome is an essential step for future planning to promote maternal and child health care services.

PATIENTS AND METHODS

This retrospective study was carried out in Azadi teaching hospital in Dohuk city, one of the main cities in the Kurdistan Regional Government of Iraq. The hospital contains the main maternity and obstetric unit which receives most of the referral cases from the rural areas.

The study period was from 1st January to 31st December 2004. Information regarding all perinatal deaths was retrieved from the maternity and neonatal registry records. Collected data had included cases with late fetal death (at or after 28th week of gestation) and early neonatal deaths (up to the seventh day of life). Cases with death in utero diagnosed before the onset of labour was regarded as macerated stillbirth, where as death of fetus which took place during labor or at time of delivery been included as fresh stillbirth.

The rate of stillbirth is presented as a proportion of all births and early neonatal death rate presented as a proportion of live births.

Demographic and clinical data pertaining to the perinatal deaths were analyzed including age, parity, and socioeconomic status (family income). Details about antenatal attendance were recorded. Information regarding complications of pregnancy and labor such as ante-partum hemorrhage, pre-eclampsia, diabetes mellitus, prolonged labor and other illnesses were recorded. Pregnancy outcome including birth weight, obvious congenital anomalies and the cause of death were also recorded.

Associations between variable were assessed using the Chi-square test with a critical P value of <0.05.

RESULTS

During the period of the study, the total births recorded were 10879. The perinatal deaths were (427) giving a perinatal mortality of (39.2 /1000 births). There were 241 early neonatal deaths giving a rate of (22.5/1000 live birth) which was higher than still birth rate 186 (17.1/1000 birth). Out of 186 stillbirths, fresh stillbirth were the highest 126 (67.7%) nearly twice the proportion of macerated stillbirth 60 (32.3%).

Placental Abruptio was the major cause behind fresh stillbirths (25.4%) followed by pre-eclampsia (16.7%), congenital anomalies (14.3%), prolonged

labor (11.9%) and breech presentation (10.3%). As for macerated stillbirths, the three main predisposing factors were pre-eclampsia, (33.3%), diabetes mellitus (21.7%) and congenital anomalies (20%) (Table 1). The causes of early neonatal deaths were identified and recorded in table 2. Prematurity was the highest (46.9%), followed by birth asphyxia (19.9%) and congenital anomalies (12.4%) were, infections (7.5%), diabetes mellitus (7%) and hemolytic diseases (6.3%).

Women whose age was lower than 20 or higher than 40 years had a higher prevalence of perinatal death compared to the other age groups ($P < 0.0001$). The lowest perinatal mortality rate were found in the "20-29" years age group (26.2 /1000 birth), followed by the "30-39" (43.09/1000 birth) (Table 3).

The safest outcome was found among mothers with Para 1-4 (31.7%) whereas the highest perinatal mortality was encountered in primiparas (43.8 per 1000 births) and grand multiparas (51.1 per 1000 births) ($P < 0.001$) (Table 4).

A highly significant associations was found between low birth weight (less than 2500 gram) and perinatal death (245/1000 birth) ($P < 0.001$) (Table 5). Women with poor antenatal care had a higher prevalence of perinatal deaths forming more than half of the cases (55.3 %) (Table 6). The majority of mothers with perinatal deaths were of lower socioeconomic status (77.8%), and lower proportions were of middle (16.6%) or high (5.6%) social standards.

Table 1. Etiological factors behind stillbirths

Causes	Fresh Stillbirths	Macerated Stillbirths
	No. (%)	No. (%)
Placental Abruption	32 (25.4)	0
Pre-eclampsia	21 (16.7)	20 (33.3)
Congenital anomaly	18 (14.3)	12 (20)
Prolonged labor	15 (11.9)	0
Breech presentation	13 (10.3)	0
Cord accident	8 (6.3)	0
Unknown cause	8 (6.3)	9 (15)
Diabetes Mellitus	7 (5.6)	13 (21.7)
Second twin	4 (3.2)	0
Rhesus isoimmunization	0	6 (10)
Total	126 (100.0)	60 (100.0)

Table 2. Etiological factors of early neonatal death

Causes	No. (%)
Prematurity	113 (46.9)
Birth asphyxia	48 (19.9)
Congenital Anomaly	30 (12.4)
Infection	18 (7.5)
Diabetes Mellitus	17 (7.0)
Hemolytic diseases	15 (6.3)
Total	241 (100.0)

Table 3. Distribution of perinatal death by different maternal-age groups

Age (years)	Perinatal deaths		
	No.	Total birth	Rate/ 1000 births
<20	35	497	70.42
20-29	148	5632	26.27
30-39	191	4424	43.19
≥ 40	53	326	162.57
Total	427	10879	

(P < 0.0001)

Table 4. Distribution of perinatal deaths according to the parity

Parity	Perinatal deaths		
	No.	Total birth	Rate/ 1000 births
Primi	81	1849	43.8
P1-4	190	5983	31.7
Grand multipara	156	3047	51.1
Total	427	10879	

($P < 0.0001$)

Table 5. Distribution of perinatal death according to the birth weight

Birth weight (grams)	Perinatal deaths		
	No.	Total birth	Rate/ 1000 births
1000-1499	108	604	178 .8
1500-2499	124	1869	66 .3
2500-2999	70	3650	19.1
3000-3999	107	3906	27.3
≥ 4000	18	850	21.1
Total	427	10879	

($P < 0.0001$)

Table 6. Distribution of perinatal death according to frequency of antenatal visits

No. of antenatal visits	Perinatal deaths
	No. (%)
0-2 (poor)	236 (55.3)
3-5 (acceptable)	138 (32.3)
5 (good)	53 (12.4)
Total	427 (100.0)

DISCUSSION

Perinatal mortality remains a challenge in the care of pregnant women worldwide, particularly in developing countries.^{12,13} The perinatal mortality rate in the present study was 39.2/1000 births, which

compares to rates of most developing countries.⁹ The highest mortality rates were registered in developing countries.¹⁴ A similar study was conducted by Chalumeau in West Africa and found the perinatal mortality rate to be 41.8/1000.¹⁵ Lower rates were reported by Manandhar

and Hinderaker (Nepal and Tanzania) 30.7/1000 and 27/ 1000 respectively.^{16,17} On the other hand, higher incidences were noted by S.M. Banajeh and Kuti et al. Their PMR were 87.4/1000 and 77/1000 respectively^{18,19} in both Yemen and Nigeria. In developed countries, the rate of (10/1000 births)²⁰ reflects the wide gap between the two worlds. In the United Kingdom the overall late fetal death rate was 4.7 per 1000 births. This low rate is mostly related to the substantial improvement in the antenatal care and modes of deliveries. The largest reduction occurred in intra-partum related deaths, and deaths due to congenital anomalies, antepartum hemorrhage and preeclampsia.²¹

Although many studies suggested that stillbirths form the major proportion of perinatal deaths,^{16,22,23} our results suggest that early neonatal deaths are higher than stillbirths. The main reasons behind this finding are attributed to the high incidence of prematurity and birth asphyxia which provide lower chances of survival after delivery.

Fresh stillbirths were found to be two fold higher than macerated stillbirths. A similar result was found by Manandhar who reported a very high rate of fresh stillbirths.¹⁶ Macerated stillbirths are often associated with insults that occur in utero during the antenatal period, while fresh stillbirths and early neonatal deaths may suggest problems with the care available during labour.^{24, 25}

As for the causes of stillbirths, different etiological factors were identified. Antepartum hemorrhage, pre-

eclampsia, congenital anomalies, prolonged labour and diabetes mellitus were the major predisposing factors. Many studies conducted in developing countries show the same results.^{17,26-28} A study conducted in Kenya by Weiner found that labour complications played a major role behind the increase in perinatal mortality. Complications like hemorrhage, eclampsia, prematurity and prolonged labour increased perinatal deaths by 8-62 folds.²⁹

Prematurity, birth asphyxia and congenital anomalies were the major contributing factors behind perinatal mortality. Prematurity had been identified as a major cause for early neonatal deaths in many countries.¹⁶⁻¹⁸ Lucy in India had reported that prematurity accounted for 42.5% and asphyxia for 26.2% as a cause of their neonatal deaths.²⁶ A study performed in Bangladesh by Elahi revealed a neonatal mortality rate of 53.5/1000 live births. The causes of deaths were prematurity / low birth weight (30%) and birth asphyxia (16%).³⁰

A community based study conducted by Nguyen showed that prematurity, congenital anomalies and birth asphyxia were the leading causes behind early neonatal deaths.³¹

A high perinatal mortality rate was recognized among mothers aged under 20 or over 40 years. This finding goes with the classical U- shaped association with maternal age.^{23, 32}

First pregnancies and high parity had been associated with poor perinatal outcome. A safer pregnancy and a better perinatal survival rate have been

recognized in mothers with parity of 1 to 4. These results were comparable to studies conducted by Hinderaker and Lucy.^{17, 26}

In the current study, poor antenatal care and low socioeconomic status were contributing factors for the high perinatal mortality. A significant reduction can be achieved by improving antenatal services and enhancing social development.²¹

CONCLUSIONS

The high perinatal mortality rate seen in the present study was mainly related to pregnancy and birth complications with contributing factors such as high parity and poor antenatal attendance. The findings indicate that early prenatal care can assist in rapid identification and management of risk factors for perinatal deaths.

REFERENCES

1. World Health Organization. Perinatal mortality: a listing of available information. Geneva: World Health Organization; 1996.
2. Bakketeig LS, Hoffman HJ, Oakley ART. Perinatal mortality. In: Bracken M, editor. Perinatal epidemiology. Oxford: Oxford University Press; 1984. p.111-20.
3. Richardus JH, Graafmans WC, Verloove-Vanhorick SP, Mackenbach JP. The perinatal mortality rate as an indicator of quality of care in international comparisons. *Med Care* 1998;36:54-66.
4. Joseph KS, Marcoux S, Ohlsson A, Liu S, Allen AC, Kramer MS. Changes in stillbirth and infant mortality associated with increases in preterm birth among twins. *Pediatrics* 2001;108(5):1055-61.
5. Akinla O. The influence of maternal and child health services on maternal and perinatal mortality. The Finnish example. *Nig Med J* 1976;6:437-45.
6. World Health Organization. World health report 2001. Geneva: World Health Organization, 2001.
7. O'Dowd MJ, Philipp EE. The history of obstetrics and gynecology. New York (NY): Parthenon Publishing Group, 1994.
8. Golding J. The epidemiology of perinatal death. In: Kiely M, editor. Reproductive and perinatal epidemiology. Boca Raton: CRC Press; 1991. p. 408-36.
9. World Health Organization. Mother-baby package: implementing safe motherhood in countries. Geneva: World Health Organization; 1994.
10. Richardus JH, Graafmans WC, Verloove-Vanhorick SP, Mackenbach JP. Differences in perinatal mortality and suboptimal care between 10 European regions. *Br J Obstet and Gynaecol* 2003;110:97-105.
11. Hefler LA, Hersh DR, Moore PJ, Gregg AR. Clinical value of perinatal autopsy and genetics consultation in fetal death. *Am J Med Genet* 2001;104(2):165-8.
12. Kramer MS. The Epidemiology of adverse pregnancy outcomes; an

- overview. *J Nut* 2003;133(5 Suppl 2):1592-6.
13. Kambarami RA: Levels and risk factors for mortality in infants with birth weights between 500 and 1800 grams in a developing country: a hospital based study. *Central Africa J Med* 2002;48(11/127):133-6.
14. Dutta DC. Textbook of obstetrics. Calcutta: New Central Book Agency (P) Ltd; 2004.
15. Chalumeau M, Colle B, Breart G. Can clinical risk factors for late stillbirth in West Africa be detected during antenatal care or only during labor? *Int J Epidemiol* 2002;31:661-8.
16. Manandhar SR, Manandhar DS, Baral MR. One year audit of perinatal mortality. *KUMJ* 2003;2(7):198-202.
17. Hinderaker SG, Olsen BE, Bergsjø PB, Gasheka P, Lie RT, Kvale G. Perinatal mortality in Northern rural Tanzania. *J Health Popul Nutr* 2003;21(1):8-17.
18. Banajeh SM, Al-Rabee AM and Al-Arashi IH. Burden of perinatal conditions in Yemen: a 12 –year hospital based study. *East Mediterr Health J* 2005; 11(4):680-9.
19. Kuti O, Orji EO, Ogunlola IO. Analysis of perinatal mortality in Nigerian teaching hospital. *J Obs Gyn* 2003;23(5):512-4.
20. Foursaas E, Gissler M, Hemminki E. Declining perinatal mortality in Finland between 1987 and 1994: contribution of different subgroups. *Eur J Obstet Gynecol Reprod Biol* 1998; 80(2):177-81.
21. Bell R, Parker L, Macphail S, Wright C. Trends in the cause of late fetal death, 1982 – 2000. *BJOG* 2004;111(12):1400-7.
22. Feresu SA, Welch K, Gillespie BW, Harlow SD. Incidence of and sociodemographic risk factors for stillbirth, pre-term birth and low birth weight in Zimbabwean women. *Pediatr Perinat Epidemiol* 2004;18(2):154-63.
23. Bell R, Glinianaia SV, Rankin J. Changing patterns of perinatal death, 1882-2000: a retrospective cohort study in Northern Region of England. *BMJ* 2004;89:531-6.
24. Conde-Agudelo A, Belizan JM, Diaz - Rossello JL. Epidemiology of fetal death in Latin America. *Acta Obstet Gynecol Scand* 2000;79:371-8.
25. Osman NB, Challis K, Cotiro M, Nordahl G, Bergstrom S. Perinatal outcome in an obstetric cohort of Mozambican women. *J Trop Pediatr* 2001;47:30-8.
26. Das Lucy, Satapathy Umakant, Panda Niharika. Perinatal mortality in referral hospital of Orissa- a 10 year review. *J Obstet Gynecol India* 2005;55(6):517-20.
27. Raksha A, Uma D, Majumdar K. Perinatal morbidity and mortality in antepartum hemorrhage. *J Obstet Gynecol India* 2001; 51(3):102-4.
28. Kumar MR, Bhat BV, Oumachigui A. Perinatal mortality trends in referral hospital. *Indian J Pediatr* 1996;63(3): 357-61.
29. Weiner R. Labor complications remain the most important risk factors for perinatal mortality in rural Kenya.

- Bull World Health Organ 2003; 81(8):561-6.
30. Chowdhury ME, Akhter HH, Chongsuvivatwong V, Geater AF.. Neonatal mortality in rural Bangladesh: an exploratory study. J Health Popul Nutr 2005; 23(1):16-24.
31. Nguyen TH, Chongsuvivatwong V. Impact of prenatal care on perinatal morality. Southeast Asian J Trop Med Public Heath 1997;28(1):55-61.
32. Garssen J, Van der Meulen A. Perinatal mortality in the Netherlands. Backgrounds of a worsening international ranking. Demographic Res [Serial online] 2004 [cited 26 Jul 2005]; 11 article13:357-94. Available from: URL: <http://www.demographic-research.org/Volumes/Vol11/13/11-13.pdf>

پوخته

مرنا نوی بويا ل نه خوشخانا نازادی يا فيرکرنی / باژیری دهوکی

پاشخایه ن: مرنا زاروکی دناڤ مال بچیکي دا یان لدهمی زاروک بوونی ههردهم وهك سهر هاتیه کا زوور نه خوش بو دهیکي و ئالوزی یه که بو کارمه ندین ساخه می یین په یوه ندی دار. کیم لیکولین یین نافخوی بو هه لسه نگاندنا فی ئاریشی بهرده ست هه نه.

ئارمانج: نه ڤ لیکولینه هاتیه کرن بو دیارکرن ریژا مرنی دهورو بهری زاروک بوئی PMR، نه گهر وفاکته رین ریخوشکهر، و پیشیارکرن هندهك ستراتیجیین دشیاندا بو کیمکرن فی ریژی.

رینک وجهی لیکولینی: زفراندن بو هه می حاله تین مرنی کو لدهورو بهری زاروک بوئی دماوی ساله کی دا دناڤهرا کانوینا دووی بو کانوینا ئیکی ل سالا 2004 ل نه خوشخانا نازادی يا فيرکرنی ل باژیری دهوکی، حکومه تا ههریما کوردستانی – عیراق چی بوین. ییزانیی چقاکی – دیموگرافی یین په یوه ندی بیقه و زانیاریت کلینیکی لسه حاله تین مرنی لدهور و بهری هاتنه کوم کرن و شلوفه کرن.

ئه نجام: سهرجه می 10879 حالین زاروک بوونی دماوی لیکولینی دا هاتینه تومارکرن. حاله تین مرنی دهورو بهری زاروک بوئی 427 حاله ت بون. ریژا مرنا دهورو بهری زاروک بوونی 39.2 بو ههر 1000 حاله تین زاروک بوونی. ژمارا زاروکین نوی مری دوو جارا هندی ژمارا زاروکین که فن مرین مهللخی 126 و 60 لیدی ئیک. نه گهری راسته خو بو مرنا تازه نوی زای بههرا پتر دزفرینه ڤه بو ڤه ڤه تیان هه ڤال بچیکي 25.4% بهری ژهرکرن دوو گیانیی (فشارا خوینا بلند) 16.7%، نه سروشتیا زک ماکی 14.3%، دریژبوونا زاروک بوئی 11.9% و زاروکی روینشتی 10.3%.

نه گهری سهره کی بو زاینا زاروکی مهللخی دزفرینه ڤه بو ژ ههر بونا دوو گیانیی 33.3%، نه خوشیا شه کری 21.6%، و نه سروشتیا زک ماکی 20%. زاروک بونا بهری وهختی و ڤه تسینا زاروکی په یوه ندیه کا بهیز دگهل مرنا دهفتیا ئیکی دژانیی دا هه به. زنده بونه کا بهرچاڤ یا مرنا دهو رو بهری زاروک بوئی دناڤ ژنکین ژبی وان دناڤهرا زی کیم تر ژ 20 و مهزتر ژ 40 سالا دا هاتیه دیارکرن، و ل بهر نه گهری هه بونا گه لهك زاروکا باری، نابوری یی خراب و نه بونا چاڤدیرییا بهری زاروک بوئی.

بیقه رین سهره کی: مرنا دهور و بهری بوئی، مرنا زاروکی نوی، بونا زاروکی مری، فاکته ر و نه گهرین په یوه ندیدار.

دوماهیك ئه نجام: ریژه کا بهرچاڤ ژ مرنا دهور و بهری زاروک بوونی لدهڤهرا مه دزفرینه ڤه بو پروبلیمیت دوو گیانیی وده می زاروک بوونی ئه ڤه وی چهندی دگه هینیت کو مه پیوستیه کا مه زن جاقدیرییا بهری زاروک بوونی هه ی، فریکرنا زوی، وهه بونا خزمه تین جاقدیرییا ساخه می بو نوی زایا لدهمی زاینی. ئه نجامین مه وی چهندی رادگه هینن کو چاڤدیرییا زوی بو حاله تین زاینی دشین هاریکاریا مه بکه ن بو نه هیلانا فاکته رین ریخوشکهر بو مرنا دهور و بهری زاروک بوونی.

داکوکی کرن ل سهر پروگرامین رهوشه نبیری و بشته ڤانیا ژنکین دوو گیان لسه جاقدیرییا بهری زاروک بوونی. باشکرن خزمه تین ساخه می ل بنگه هین ساخه می یین دهستیکی ئه ڤه هه می دشین هاریکاریا کیمکرن ریژا مرنا دهور و بهری.

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CASUALTIES AND DEATHS FROM ROAD TRAFFIC ACCIDENTS IN DOHUK, IRAQ

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ABSTRACT

Background Motor vehicle injuries have been an important cause of morbidity and mortality in developed countries. In developing countries, however, the impact of such injuries is rapidly increasing due to the increased passenger car ownership and low compliance to safety measures. In Iraq, as well as in other neighbouring countries, high rates of serious Road Traffic Accidents (RTAs) have been reported.

Aims The aim of the present study is to study the pattern of RTAs crashes, injuries and fatalities in Dohuk city, Kurdistan region in Iraq and to investigate any possible increase after 2003.

Methods The study was conducted in Dohuk for a 2 year period 2003-2004. The data was collected from two main sources. The first was the emergency hospital in Dohuk. Information regarding age, gender, type and site of injury and clinical outcome were obtained from the statistical office of the hospital. The other source of data collection was the Directorate of Dohuk Traffic Police.

Results The study revealed increases in RTAs casualties and fatalities (14.3% and 66.3% respectively) during the year 2004 in comparison with 2003. Fractures in different parts of the body constituted the majority of injuries. The most common types were skull fractures (31.9%) followed by lower limb fractures (23.3%). The study documented a total increase in the number of registered cars during 2003 – 2004 (132.2%) with a difference of (32.9%) for the year 2004 over that of 2003.

Conclusion There have been increases in both casualties and fatalities due to RTAs in parallel with an increase in the number of imported second hand cars. Fractures of different parts of the body constituted the majority of injuries with evident male preponderance all through.

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Key words: Road traffic, Accidents, Iraq

Motor vehicle injuries have been an important cause of morbidity and mortality in developed countries.^{1,2} Despite that, there has been a significant reduction in traffic related fatalities in these countries in the last 30 years. Most

analyses have attributed these to changes in vehicle designs, better road designs and strict compliance with safety measures regulations particularly seat belt use.²

In developing countries, however, the impact of such injuries is rapidly increasing due to the increased passenger car ownership and low compliance to safety measures. Globally in 2002 there were 1.2 million deaths resulted from road traffic accidents (RTAs) and about 10

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times that where injuries had occurred in less developed countries.³⁻⁵

In Iraq, as well as in other neighbouring countries, high rates of serious RTAs have been reported. In the United Arab Emirates as well as in other Gulf countries a steady increase of RTAs has been reported.⁶⁻⁹ In Iraq also there has been a tremendous increase of imported cars after the war and the toppling of the regime in April 2003. This was mainly due to the lifting of regulations on importing used cars which were imported with almost no taxation or any other safety regulations. Accordingly it was a concern for doctors and traffic police for an expected increase in RTAs.

The aim of the present study is to study the pattern of RTAs, injuries and fatalities in Dohuk city, Kurdistan region in Iraq and to investigate any possible increase after 2003.

MATERIAL AND METHODS

The study was a survey conducted in Dohuk for a 2 year period 2003-2004. Dohuk is the third largest city in Kurdistan region in Iraq and the centre of Dohuk governorate, with about one million inhabitants. The data were collected from two main sources. The first was the emergency hospital in Dohuk. This hospital is the only one specialized in dealing with emergencies, including RTAs, in Dohuk governorate with 120 beds. Usually all victims of motor vehicle injuries should be admitted to this hospital; where a police report is indicated for each patient. A data sheet was specially

prepared for this study that included all the variables needed to achieve the objectives of the study. The variables are: age and gender of the patient, type and site of the injury and the clinical outcome. This information was obtained from the statistical office in the emergency hospital. The other source of data was the Directorate of Dohuk Traffic Police (DDTP), where information regarding the number of cars and types of RTAs was obtained.

RESULTS

Figures 1 and 2 show the distribution of RTAs casualties and deaths by gender for the two years period 2003-2004. There has been an increase in total casualties in Dohuk with an increase in mortality in 2004 in comparison with that of 2003.

Table 1 reveals the types of RTAs casualties. The majority of the victims have been brought with fractures at different sites. Fractures of the skull followed by those of the lower limbs were the commonest types and constituted about three quarter of all fractures. The trend in the types of fractures has remained almost the same during both 2003 and 2004.

Table 2 shows a (132.2%) increase in the number of different types of cars registered in Dohuk in 2004 in comparison with that of 2003, with Lorries making the highest increase followed by private cars. Also there has been (32.9%) increase in the total number of vehicles in Dohuk between 2003 and 2004.

The data from DDTP also revealed that high speed, bad quality roads and

weather constituted 80.8%, 11.8% and 7.4% respectively of all causes of RTAs in 2003 in comparison with approximate

rates of 76.1%, 16.3% and 6.8% for the year 2004.

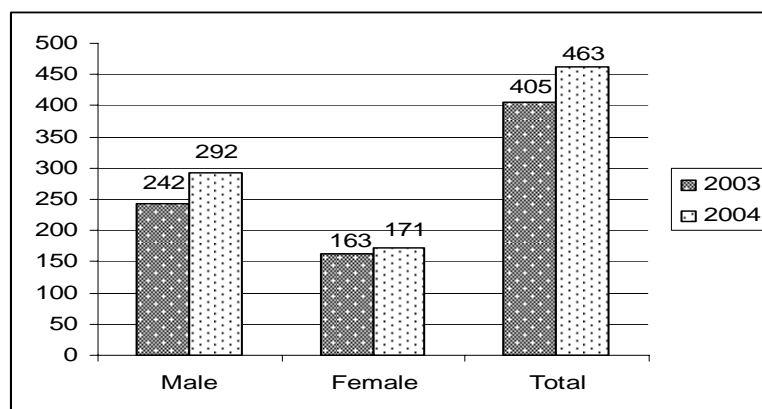


Figure 1. Distribution of RTAs casualties by gender in Dohuk 2003-2004

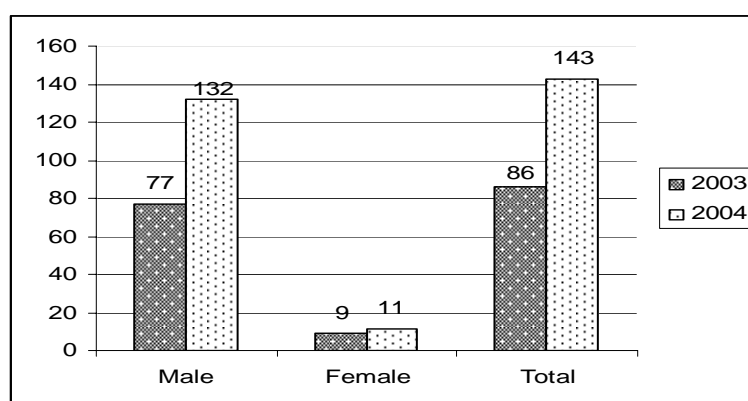


Figure 1. Distribution of RTAs deaths by gender in Dohuk 2003-2004

Table 1. Types of casualties among patients admitted to emergency hospital in Dohuk 2003-2004

Type of casualty	2003 No. (%)	2004 No. (%)	Total No. (%)
Skull fracture *	130 (32.1)	147 (31.7)	277 (31.9)
Lower limb fractures	92 (22.7)	110 (23.8)	202 (23.3)
Upper limb fractures	48 (11.9)	55 (11.9)	103 (11.9)
Spine fracture	29 (7.2)	20 (4.3)	49 (5.6)
Combined soft tissue injury	29 (7.2)	32 (6.9)	61 (7.0)
Chest injury	21 (5.2)	24 (5.2)	45 (5.2)
Multiple fracture	14 (3.5)	19 (4.1)	33 (3.8)
Abdominal injury	13 (3.2)	16 (3.5)	29 (3.3)
Pelvic injury	12 (3.0)	18 (3.9)	30 (3.5)
Others	17 (4.2)	22 (4.8)	39 (4.5)
Total	405 (100.0)	463 (100.0)	868 (100.0)

* With or without head injury

Table 2. Number and types of new cars registered in Dohuk between 2003 and 2004

Type of vehicle	2003	2004	% increase
	No. (%)	No. (%)	
Private cars	5236 (82.1)	11597 (78.3)	121.5
Taxi cars	437 (6.9)	752 (5.1)	72.1
Lorries	686 (10.8)	2426 (16.4)	254
Others	17 (0.3)	31 (0.2)	82.4
Total	6376 (100.0)	14806 (100.0)	132.2

DISCUSSION

The study revealed an evident increase in cars and in RTAs in Dohuk in 2004 in comparison with 2003. Increases occurred both in the total casualties and deaths from RTAs. It is anticipated that the unrestricted importation of second hand used cars after the War may have been the main reason of this increase. The data could have been more decisive if the comparison was made with data before 2003. That is because importation of used cars has started at June 2003. Accordingly one would anticipate that the increase in RTAs have actually started in 2003, thus making such differences even more significant. The DDTP has ranked high speed as the main cause of these accidents. This is in agreement with the expectation that these cars could achieve higher speed than already existing cars. Also there has been no subsequent improvement in the quality of roads and/or traffic legislations. Several studies all over the world have linked RTAs with high speeds and ignoring traffic legislations.^{10,11}

The study revealed that the casualties

and deaths were clearly higher in males than females. This is due to the low prevalence of female drivers. Also accidents are more frequent among youth males driving together and specially while being under the effect of alcohol. Similar findings have been observed in Turkey and in other countries.¹² Also in Sulaimani governorate in Kurdistan region a study conducted on road traffic accidents and the male to female ratio among victims was 4:1.⁹

The study shows that most of the injuries were of severe types; where skull and lower limbs fractures constituted about two thirds of all casualties. Similar findings were also found in Sulaimani governorate.⁹ Lower limb injuries have also been the main cause of casualties in other studies.¹² The high severity of cases admitted might indicate that only the most severe casualties are admitted to the hospital while mild cases might have escaped registration. This will results in under registration of RTAs. Similar results have also been observed in similar studies conducted in other countries.^{4,13}

CONCLUSIONS

1. RTAs casualties and fatalities show increases during 2004 compared to that of 2003. The number of registered imported cars increased in parallel by (132.2%) during the same period.
2. The most common types of casualties were skull fractures followed by lower limb fractures. Male preponderance was evident all through.
3. The commonest registered cause of RTAs was high speed followed by bad quality of roads and weather.

RECOMMENDATIONS

1. Provision of training programs for doctors and paramedics on sorting out and dealing with RTAs casualties.
2. Strict checking of imported cars before permission for use.
3. Improving road quality in parallel with the increasing number of imported cars.
4. Strict implication of traffic legislations and compliance with safety measures regulations.
5. Implementing proper educational programs making use of the different available mass media.
6. The conduct of more extensive studies for delineation of proper preventive measures capable of decreasing RTAs casualties and deaths.
7. Increasing the number of peripheral emergency treatment centers.

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REFERENCES

1. Soderlund N, Zwi AB. Traffic-related mortality in industrialized and less developed countries. Bull World Health Organ 1995;73(2):175-82.
2. Noland RB. Medical treatment and traffic fatality reductions in industrialized countries. Accid Anal Prev 2003;35(6):877-83.
3. Hazen A, Ehiri JE. Road traffic injuries: hidden epidemic in less developed countries. J Natl Med Asso 2006;98(1):73-82.
4. Mock CN, Forjuoh SN, Rivara FP. Epidemiology of transport-related injuries in Ghana. Accid Anal Prev 1999;31(4):359-70.
5. Chao TC, Khoo JH, Poon WN. Road traffic accident casualties in Singapore (with special reference to drivers and front seat passengers). Ann Acad Med Singapore 1984;13(1):96-101.
6. El-Sadig M, Norman JN, Lloyd OL, Romilly P, Bener A. Road traffic accidents in the United Arab Emirates: trends of morbidity and mortality during 1977-1998. Accid Anal Prev 2002;34(4):465-76.
7. Bener A. The neglected epidemic: road traffic accidents in a developing

- country, State of Qatar. *Int J Inj Cont Safety Prom* 2005;12(1):45-7.
8. Jamel H, Mukhlis GM, Al-Habboubi G. Epidemiological study of road traffic accidents in Baghdad area. *J Fac Med* 1981;23(3):267-83.
 9. Quaradaghi SHS, Chawash HAH. Road traffic accidents in Sulaimani governorate, review of 300 consecutive cases. *J Dohuk Univ* 2004;7(1):23-7.
 10. Babio GO, Daponte-Codina A. Factors associated with seatbelt, helmet, and child safety seat use in a spanish high-risk injury area. *J Trauma* 2006;60(3):620-6.
 11. Vorko-Jovic A, Kern J, Biloglav Z. Risk factors in urban road traffic accidents. *J Safety Res* 2006;37(1):93-8.
 12. Esiyok B, Korkusuz I, Canturk G, Alkan HA, Karaman AG, Hanci IH. Road traffic accidents and disability: a cross-section study from Turkey. *Disabil Rehabil* 2005;27(21):1333-8.
 13. Aptel I, Salmi LR, Masson F, Bourde A, Henrion G, Erny P. Road accident statistics: discrepancies between police and hospital data in a French island. *Accid Anal Prev* 1998;31(1):101-8.

پوخته

توشبوون و مرن ب رویدانی هاتن و چوونی ل دهوك، عیراق

شه‌نگستی فه‌کولینی: توشبوون ب رویدانی ترومیلا دینه نه‌گه‌ری نه‌خوشیی و مرنی ل دهوله‌تین پیشکەفتی. به‌لی ریژا فان رویدانا ل دهوله‌تین باشکەفتی به‌ره‌ف بلندبوونی فه‌یه ب شیوه‌کی ب له‌ز ژ به‌ر زیدده‌بوونا خودان ترومیلا و نه‌پیگیریا دروست ب رینمایین ته‌ناهی. ل عیراقی و دهوله‌تین جیران ریژده‌ما مه‌زن ژ فان رویدانا تومارکریه.

ئارمانجا فه‌کولینی: ئارمانجا فی فه‌کولینی دیارکرنا جووری نه‌خوشیی و مرنی یه ژ رویدانی ریکین هاتن و چوونی ل پارێزگه‌ها دهوکی و زانینا زیدده‌بوونا دیتن نه‌نجام بدهت پشی سالا 2003 ی.

ریکین فه‌کولینی: فه‌کولین هاته‌ کرن بو سالی 2003-2004 و پیزانین هاتنه‌ کومکرن ژ دوو ژیدده‌را، یی ئیکی نه‌خوشخانا ته‌نگافیا ل پارێزگه‌ها دهوکی و یی دووی ریژه‌به‌ریا هاتن و چوونی ل پارێزگه‌ها دهوکی.

نه‌نجامین فه‌کولینی: فی فه‌کولینی زیدده‌بونه‌ک دیارکر د ریژا توشبوون و مرنی دا ل سالا 2004 ی به‌رامبه‌ر سالا 2003 ی (14.3% و 66.3% لدویف ئیک). شکه‌ستین هه‌ستیا یین جووره‌ وجور پرانیا توشبوویا بوون و شکه‌ستین کلوخی ژ هه‌میان پتر بوون (31.9%) و دویفدا شکه‌ستین پیا (23.3%). هه‌روه‌سا فه‌کولینی زیدده‌بوونه‌ک دیارکر د ژمارا ترومیلا تومارکری ل سالی 2003 و 2004 ی ب ریژا (132.2%) و ب جوداهیا (32.9%) ل سالا 2004 ی ژ سالا 2003 ی.

ده‌رئه‌نجام: زیدده‌بوونه‌ک یا هه‌ی د توشبوون و مرنی ژ رویدانی ترومیلا ل سالی 2003 و 2004 ی دگه‌ل زیدده‌بوونا چیبوی د ئینانا ترومیلا لاواز. توشبووین نیرا پتر بوون ژ یین مییا وشکه‌ستین هه‌ستیا پرانیا توشبوونا بوون.



THE VALUE OF 2-MERCAPTOETHANOL TEST IN DIAGNOSING RECURRENT ACTIVE BRUCELLOSIS

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ABSTRACT

Background Brucellosis is endemic in Iraq; particularly in Northern parts. Several problems are encountered in diagnosing the disease depending on serological tests; especially among cases suspected to have reactivation of the disease.

Aim to evaluate the value of 2-Mercaptoethanol (2ME) test in diagnosing recurrent active brucellosis.

Patients and Methods The study population consisted of 466 patients who had been treated and cured from a previous attack of brucellosis (diagnosed clinically or serologically), who returned suffering from symptoms suggesting a new attack of brucellosis with positive slide agglutination (SAT) test and on whom a 2ME test was conducted. Patients were collected from Al-Salam teaching hospital in Mosul during 2 years period (1999-2000).

Results The 2ME test was positive in 184/408 (45%). The probability of having a positive 2ME test increased if the SAT was positive at high titers. The study also revealed high IgG antibody titers, mainly ranged between 1:160 and 1:640, with doubled positive rate among females in comparison to males.

Conclusions The study revealed that 2ME test is useful in diagnosing about 45% of suspected cases with activation of previously treated and cured brucellosis. A direct SAT should first be conducted for suspected cases and the probability of having a positive 2ME test has increased if the primary SAT was positive at higher titers.

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Key words: Brucellosis, 2ME test, SAT, Diagnosis

Brucellosis is endemic in Iraq, particularly in Northern parts where the main mode of transmission is via crude unpasteurized cheese. An annual epidemic is usually occurring in spring which is the season of crude cheese made from sheep or goat milk.^{1,2}

Clinical diagnosis of brucellosis should always be supported by laboratory tests. The definite diagnosis is by isolation

of brucella species from the patient.³ This, however, has several drawbacks. The slow growth of brucella in primary culture may delay diagnosis. In addition brucella culture usually needs an incubation time between 3-6 weeks.⁴ Moreover, the sensitivity is often low ranging from 40 to 90% depending on the stage of the disease, species, and culture technique.⁵ This makes culture impractical for clinicians who need quick results to start treatment. Accordingly serological tests, like Rose Bengal screening test and standard tube agglutination test, are commonly requested.⁶ However, these tests are unable

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to differentiate between previous exposures or recent active infections, as they are designed to test the presence of total IgM and IgG antibodies.⁷ Moreover, brucella species are intracellular microorganism which can go into a dormant stage with remission and exacerbation leading to chronic pattern of infection. Serological tests are also unable to differentiate between recurrent activation (i.e. acute on chronic cases) and the smegma of previous exposures.^{7,8} This problem will be more obvious by the sustained high levels of antibody titers, particularly of IgM type.^{8,9} The latter is more marked in Iraqi community due to the high prevalence of antibody titers from previous exposures; which was found to be above 13% among villagers in Northern Iraq in comparison to 1.8% in India and 4.5% in Saudi Arabia.^{8,10,11}

The 2-mercaptoethanol (2ME) will abolish IgM antibodies allowing the detection of IgG antibodies in serum samples. Accordingly it will be valuable in differentiating between a recurrent activation and an increase in IgM antibodies from previous exposure.^{8,12,13}

The aim of this study is to evaluate the value of 2ME test in the diagnosis of recurrent active cases of Brucellosis in Iraq.

PATIENTS AND METHODS

The study group consisted of 466 patients who had been treated and cured from a previous attack of brucellosis diagnosed clinically or serologically and who have returned after a period suffering from

symptoms suggesting a new attack of brucellosis. The patients were seen in the outpatient department of Al-Salam teaching hospital in Mosul from 1/1 – 31/12/2000. All patients were sent to the laboratory with a request for 2ME test. A slide agglutination test (SAT) was done for all cases and only those with a positive SAT (408) test were included in the study.

Sampling

Blood samples were collected in plain sterile tubes, and transferred immediately to the laboratory. The samples were incubated in a water bath at 37°C for clot formation and serum separation. Hemolysed and lypaemic samples were rejected. No inactivation was done.

Principle

The test used was the direct standard slide agglutination test for *Brucella abortus*, to detect and quantify specific antibody using stained bacterial antigen suspension (Cambridge Biomedical, Febrile Antigen Kit). The 2ME treatment was carried out in the day before the test by treating the samples with 0.2 mol/l of 2ME solution. After that all tubes were placed in a 37°C water-bath for one hour.

The SAT was conducted by using a suitable pipette delivering 0.08 ml of serum on the test plate and 0.03 of a well shaken *Brucella abortus* antigen, mixed by stirring for few seconds and the mixture spread over the entire area of the circle. Slow rotation was then applied for one minute and continuous observation of the

agglutination at one minute was reported. A negative and a positive control were run together with each test. Titration was done simultaneously for untreated and treated serum. Six serial dilutions were prepared corresponding to titers ranging from 1:40 - 1:1280 together with a control. Thereafter, the SAT was conducted as above for each dilution. The positive titer is taken as the last dilution to show macroscopic agglutination in one minute.

RESULTS

Table 1 shows that 408/466 patients (87.5%) referred to the laboratory suspected of activation of previous brucellosis had a positive SAT. All the 408 patients were included in the study; while the 58 patients were excluded. Table 2 reveals that 184/408 (45%) had a positive 2 ME test, with relatively higher rates of

positivity among females in comparison to males.

Table 3 shows that the probability of a positive 2ME test increases if direct SAT was positive at high titers., where 55/184 (30%) of patients with positive 2ME test had also a positive result at 1:1280 while only 4/184 (1%) of the negative 2ME test cases had a positive SAT titer at such dilution. Similarly 85/224 (37.9%) of negative 2ME test cases had a positive SAT at 1:160 titers in comparison with none among positive 2ME cases.

Table 4 illustrates the IgG anti-brucella antibody titers among positive 2ME test cases. A total of 66/184 (35.9%) had a titer at 1:160 followed by 59/184 (32.1%), 43/184 (23.4%) and 13/184 (7.1%) at titers of 1:320, 1:640 and 1:1280 respectively. Table 4 also shows that the positivity was more than double among females in comparison to males.

Table 1. Distribution of the study population according to result of SAT

Gender	SAT		Total No. (%)
	Positive No. (%)	Negative No. (%)	
Male	177 (43.3)	14 (24.1)	191 (41.0)
Female	231 (56.6)	44 (75.9)	275 (59.0)
Total	408 (100.0)	58 (100.0)	466 (100.0)

Table 2. Distribution of the study population according to result of 2ME test

Gender	2 ME		Total No. (%)
	Positive No. (%)	Negative No. (%)	
Male	56 (31.6)	121 (68.4)	177 (100.0)
Female	128 (55.4)	103 (44.6)	231 (100.0)
Total	184 (45.1)	224 (54.1)	408 (100.0)

Table 3. Distribution of the antibrucella antibody (Total IgG +IgM) titers among study population according to 2ME test results

Gender	2ME test positive (IgG positive titer)						2ME test negative (IgG negative titer)					
	1:80	1:160	1:320	1:640	1:1280	All	1:80	1:160	1:320	1:640	1:1280	All
Male	-	-	2.2	14.1	14.1	30.4	0.5	37.9	9.4	5.8	0.5	54.0
Female	-	-	20.1	33.7	15.8	69.6	-	-	34.8	10.7	0.4	46.0
Total	No. (%)	-	41 (22.3)	88 (47.8)	55 (29.9)	184 (100.0)	1 (0.5)	85 (37.9)	99 (44.2)	37 (16.5)	2 (0.9)	224 (100.0)

Table 4. IgG titer among positive 2ME test cases

Gender	IgG antibrucella antibody titers						
	1:40 No. (%)	1:80 No. (%)	1:160 No. (%)	1:320 No. (%)	1:640 No. (%)	1:1280 No. (%)	All titers No. (%)
Male	-	-	11 (6.0)	20 (10.9)	16 (8.7)	9 (4.9)	56 (30.4)
Female	1 (0.5)	2 (1.1)	55 (29.9)	39 (21.2)	27 (14.7)	4 (2.2)	128 (69.6)
Total	1 (0.5)	2 (1.1)	66 (35.9)	59 (32.1)	43 (23.3)	13 (7.1)	184 (100.0)

DISCUSSION

The study found that 2ME test was requested by clinicians directly, without screening by SAT for patients who have had a previous attack of brucellosis and returning with symptoms suggesting a new activation. A total of 58/466 (12.5%) referred cases were found to have a negative SAT and accordingly 2ME test was not conducted. This indicates that some clinicians are still unclear about the use and limitation of this test. The 2ME test is not superior to SAT in diagnosing acute brucellosis. In fact, SAT might be more sensitive in diagnosing such cases than 2ME test.^{6,8,12} The latter should only be requested after a positive SAT in cases suspected to have an activation of previous attack of brucellosis. The 2ME breaks the disulphide links of IgM pentamer, thus

interfering with its highly efficient agglutinating capacity while not affecting IgG molecules.^{8,12} Dithiothreitol can act as 2ME by inactivating IgM with the privilege of not having the offensive odor or irritant properties of 2ME. Controversial results have been reported, however, about the effectiveness of this substance in comparison to 2ME.^{14,15} Accordingly the 2ME test is still the one currently used routinely in Iraq to test for IgG antibodies, rather than ELISA specific IgG Test, which is more specific but also more elaborate and expensive to be used in practice.¹⁶

A positive 2ME test was found in 45% (184/408) of cases. This shows that less than one half of the suspected cases have IgG antibodies which might reflect acute exacerbation of the previous attack of brucellosis. The increase in IgG titer

was moderate (1:160) in 36% (66/184) of cases compared to about 7% (13/184) of high titer of 1:1280. This might indicate that IgG titers in chronic cases in Iraq are lower than IgG titers detected in acute cases of brucellosis. The 2 ME test might be valuable in diagnosing about half of suspected cases. In a study conducted by Buchanan and Faber,¹² about 50% of positive 2ME cases had signs and symptoms and required further treatment. Also, negative 2ME was found to be strong evidence against the diagnosis of chronic active brucellosis.^{8,12} The value of this test in diagnosing reactivation and/or incomplete recovery from brucellosis has also been reported in other studies. However, one can not totally eliminate the possibility of recent infection, as brucellosis gives no permanent immunity and re-infection is always a possibility; especially in endemic areas.^{13,17,18}

The study also found that more than 50% of cases with negative 2ME test were positive for SAT. In one study, SAT remained positive at titers equal or more than 160 for one and a half years in about 48% of cases despite adequate treatment, while 2ME test remained positive in only 4% after the same period of follow up.¹² Similarly in another study the SAT gave measurable titers ranging 1:160 to 1:640 for a long period despite effective therapy.⁸ Similar results have also been reported in other studies.^{3,9} The SAT titers, however, were much lower than those with a positive 2ME test. This indicates that those cases are in the acute exacerbation reflected by high peak titers.

Higher rates of 2ME positive tests were noticed in females in comparison to males. This might be a reflection of low immunity among females; which is usually determined by the burden of pregnancy.¹⁹ This is more obvious in Iraq due to high fertility rates. In addition, females have also been affected by the adverse effect of malnutrition resulting from wars and sanctions on Iraq.^{20,21}

Finally, this study documented the value of 2ME test in diagnosing cases suspected to have reactivation of a previous attack of brucellosis. The test should only be requested for cases proved to have a positive SAT. The probability of having a positive 2ME test is increased if the primary SAT was positive at high titers. A more extended study is indicated to look on the variations of IgG titers of brucellosis cases during the course of the illness till recovery and compared with specific IgG estimated by ELISA technique.

REFERENCES

1. Karim MA, Penjouiian EK, Dessouky FI. The prevalence of brucellosis among sheep and goats in northern Iraq. *Trop Animal Health Pro* 1979;11(3):186-8.
2. Abu Shaqra QM. Epidemiological aspects of brucellosis in Jordan. *Europ J Epidemiol* 2000;16(6):581-4.
3. Araj GF. Human brucellosis: a classical infectious disease with persistent diagnostic challenge. *Clin Lab Sci* 1999;12(4):207-12.

4. Yagupsky P. Detection of brucellae in blood culture. *J Clin Microbiol* 1999;37:3437-42.
5. Mantur BG, Mangalgi SS. Evaluation of conventional Castaneda and lysis centrifugation blood culture techniques for diagnosis of human brucellosis. *J Clin Microbiol* 2004; 42:4323-8.
6. Coppola N. New diagnostic frontiers in brucellosis [English abstract]. *Infect Med* 2001;9(3):130-6.
7. Memish ZA, Almuneef M, Mah MW, Qassem LA, Osaba AO. Comparison of brucella standard agglutination test with the ELISA IgG and IgM in patients with brucella bacteremia. *Diagn Microbiol Infect Dis* 2002;44(2):129-32.
8. Mantur BG, Biradar MS, Bidri RC, Mulimani MS, Veerappa, Kariholu P, et.al. Protean clinical manifestations and diagnostic challenges of human brucellosis in adults: 16 years' experience in an endemic area. *J Med Microbiol* 2006;55(7):897-903.
9. Almuneef M, Memish ZA. Prevalence of brucella antibodies after acute brucellosis. *J Chemother* 2003;15(2): 148-51.
10. Al-Dabbagh S, Al-Derzi N, Al-Hamdani R. The prevalence of brucella antibodies among rural communities near Mosul, Iraq. *JIMA* 1997;29:67-70.
11. Al-Sekait MA. Seroepidemiological survey of brucellosis antibodies in Saudi Arabia. *Ann Saudi Med* 1999;19:219-22.
12. Buchanan TM, Faber LC. 2-Mercaptoethanol brucella agglutination test: usefulness for predicting recovery from brucellosis. *J Clin Microbiol* 1980;11:691-3.
13. Pellicer T, Ariza T, Foz A, Pallares R, Gudiol F. Specific antibodies detection during relapse of human brucellosis. *J Infect Dis* 1988;157(5): 918-24.
14. Klein GC, Behan KA. Determination of brucella immunoglobulin G agglutinating antibody titer with dithiothreitol. *J Clin Microbiol* 1981; 14(1):24-5.
15. McMahon KJ. Comparison of the 2 mercaptoethanol and dithiothreitol tests for detecting brucella immunoglobulin agglutinating antibody in bovine serum. *Can J Comp Med* 1983;47:370-2.
16. Al Dahouk S, Tomaso H, Nockler K, Neubaur H, Franqoulidis D. Laboratory-based diagnosis of brucellosis- a review of the literature. Part 1: techniques for direct detection and identification of brucella spp. *Clin Lab* 2003;49(9,10):487-505.
17. Gazapo E, Gonzales LJ, Subiza JE, Baquero M, Gill J, de la Concha EG. Changes in IgM and IgG antibody concentrations in brucellosis over time: importance for diagnosis and follow up. *J Infect Dis* 1989;159 (2):219-25.
18. Baldi PC, Miguel SE, Fossati CA, Wallach JC. Serological follow-up of human brucellosis by measuring IgG antibodies to lipopolysaccharide and

- cytoplasmic proteins of *Brucella* species. Clin Infect Dis 1996;22(3): 446-55.
19. Harkness RA. Oestrogens and host resistance. J R Soc Med 1980;73(3): 161-4.
20. Al-Kafajei A, Al-Neema B. Evaluation of population health needs. In: Al-Kafajei A, Ablahad Y, Al-Mallah N, Khamargo T, Al-Jawadi A. The second field exercise of Mosul University; Qazfakhra and Shamsiat exercise. Mosul: Mosul University press; 1989. p. 34-67.
21. Buck L, Gallant N, Nossal KR. Sanction as a gendered instrument of statecraft: the case of Iraq. Rev Intern Studies 1998;24:69-84.

پوخته

مفایه حسا ۲- ژبو دهسنيشانكرنا تايما مالتايي يا دووباره كرى

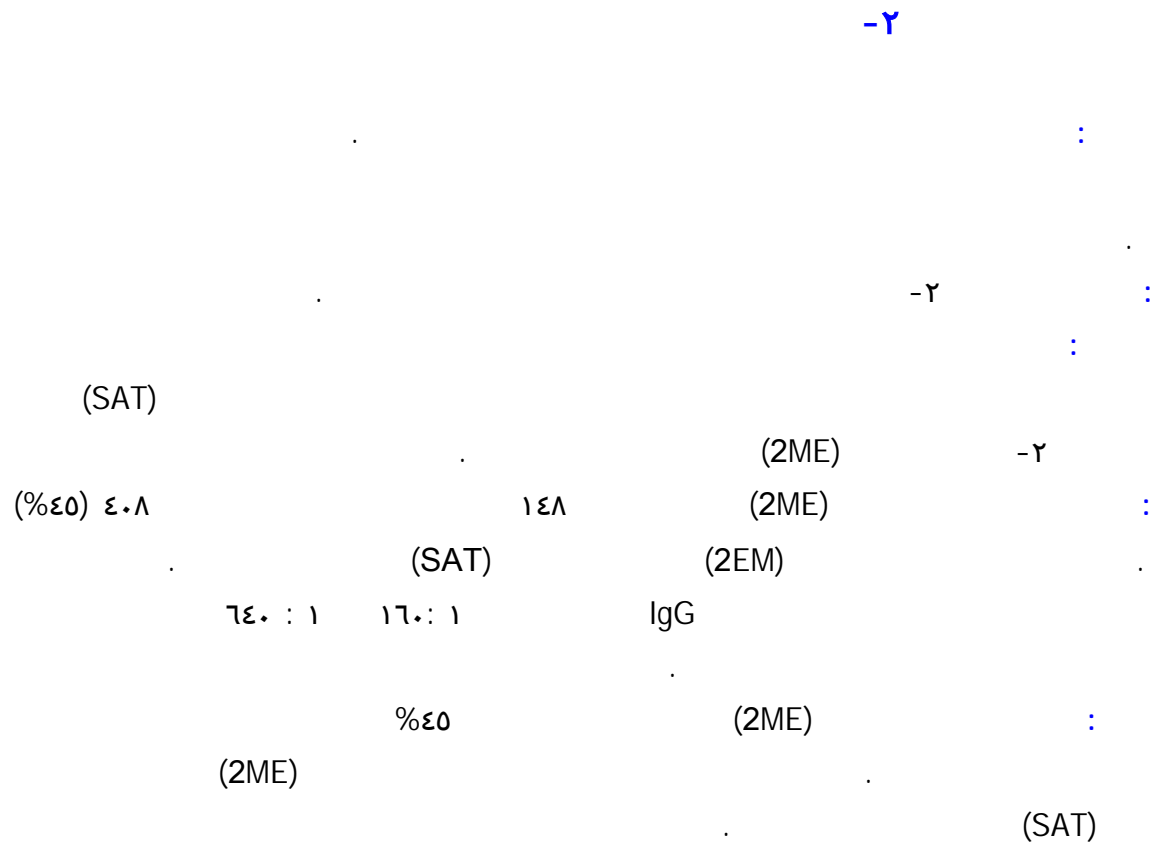
دهسنيپك: نهخوشيا تايما مالتايي يا بهربه لافه ل عيراقى نهمازه ل دهفهرين باكوورى. چهند گرفتارى هه نه ژبو دهسنيشانكرنا نهساخيي ته گهر پشت بهستني ده نه فحسين نه زموونگه هه، نهمازه ل كهسين گومانليكرى كو نهخوشى يا لي زفري پشتى كو هاتينه چاره سهركرن و ساخووين.

نارمانج: هه لسه نگاندا رولي فحسا ۲- بو دهسنيشانكرنا تايما مالتايي يا دووباره كرى.

ريكا فه كوئيني: نمووني فه كوئيني ژ وان نهخوشا فه گرت نهوين تووشى تايما مالتايي بووين و هاتبوونه دهسنيشانكرن ب ريبن كلينيكى و تافيكه هه و هاتنه چاره سهركرن و ساخووين و پشتى ماوه كي چهند نيشانين نهساخيي لي زفري دگه ل نه رينيا فحسا پيكفه نووسيانى ل سه ر سلايدى (SAT)، و فحسا ۲- (2ME) بو هاته كرن ژبو هه لسه نگاندا رولي وي بو دهسنيشانكرنى.

نه نجام: هاته ديار كرن ل في فه كوئيني دا كو نه نجامي فحسا (2ME) بالهر بوو ل ۱۴۸ ژ سه رجه مي نمووني هاتيه وه رگرتن ل فه كوئيني دا كو ۴۰۸ (۴۵٪) نه ساخ بوون. له وان هيا نه ريني يا فحسا (2ME) زيده بوو ده مي كو فحسا (SAT) بالهر بوو ل تايته رين بلندتر. هه روه سا هاته ديار كرن كو تايته رين دزين تايما مالتايي ژ جورى IgG دنا فبه را ۱:۱۶۰ هه تا ۱:۶۴۰ بوون و ريزه يا نه ريني يا فحسى ل ده فافره تا دوو جارى پياوا بوو.

دهر نه نجام: ل فه كوئيني هاته ديار كرن كو فحسا 2ME يا مفايه ژبو دهسنيشانكرنا نزيكى ۴۵٪ ژ كهسين گومانليكرى ب زفراندا چه له نكيا نهساخيي پشتى چاره سه ربي و ساخوويني. هه روه سا فه كوئيني ئاشكرا كر زيده بوونا له وان هيا نه ريني يا فحسا (2ME) ل نهخوشين كو فحسا (SAT) بالهر بوو ل تايته رين بلند تر.



SOME HISTOCHEMICAL CHANGES IN THE PLACENTAE OF PREECLAMPSIA

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ABSTRACT

Background The placenta has been implicated in the pathophysiology of preeclampsia. Preeclampsia is more common in multifetal gestations which have an increased placental mass compared to singleton pregnancies

Objective Detecting the effects of preeclampsia on the availability of enzymes in the full term placenta.

Methods Two groups of placentae were taken from full term pregnant women immediately after labour, each consisting of ten placentae. The first group are placentae obtained from women having an uneventful pregnancy with no history of disease or complication (as a control group) while the second group consists of placentae obtained from women with a history of preeclampsia. The materials were obtained from Al-Batool and Al-Khansaa Teaching Hospitals in Mosul, between February and July (2006).

Results Significant histochemical changes were detected in the placentae of the second group when compared with those from the first group, such changes result from syncytial damage and destruction affecting the preeclamptic placentae, leading to the loss of alkaline phosphatase enzyme with an increase in the amount of the degenerating acid phosphatase enzyme.

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Key words: Placentae, Preeclampsia, Histochemical changes

The placenta has been implicated in the pathophysiology of preeclampsia. Preeclampsia is more common in multifetal gestations which have an increased placental mass compared to

singleton pregnancies.¹ An initiating event in preeclampsia has been postulated to be a reduction in the placental perfusion and destruction of the placental tissue both leading to widespread dysfunction of the maternal vascular endothelium by mechanisms that remain unknown.² Removal of the placenta in preeclampsia is regarded as the main step in the treatment.³

Normally there is invasion of the uterine spiral vessels by cytotrophoblasts and with the end of the second trimester of the pregnancy, the uterine spiral arteries are lined exclusively by the cytotrophoblasts and the endothelial cells are no longer present in the endometrial

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and superficial myometrial region of the uterus.⁴ This remodeling of the uterine spiral arteries was referred to as physiological changes and it changes the spiral arteries from thick walled muscular vessels to sac-like flaccid vessels.⁵ Failure of the spiral arteries to remodel has been postulated to be the morphological basis for decreased placental perfusion in preeclampsia.⁶ Particular attention has been paid to the alkaline and acid phosphatase enzymes largely because they are thought to play an important role in the function of the placenta.

There is an inverse relationship between the amount of acid and alkaline phosphatase enzymes. Normally the alkaline phosphatase enzyme is produced from the basement membrane of the syncytiotrophoblast and the microvilli on their surfaces. This enzyme is formed in small amount during the first and second trimester, increases in amount towards the third trimester and reaches maximum quantity in the full term placentae.⁷ The acid phosphatase enzyme is dominating during the first half of pregnancy due to a remodeling process occurring normally in the vasculature of the placentae during the first trimester while it is absent in normal full term placentae.⁸

In the present study histochemical techniques were used to demonstrate low concentration of alkaline phosphatase and high concentration of acid phosphatase in the placentae of preeclamptic women.

METHODS

A histochemical study was carried out on

placentae obtained from full term pregnant women. The specimens were obtained from Al-Batool and Al-Khansaa Teaching Hospitals in Mosul, between February and July (2006) and studied in the Department of Anatomy, College of Medicine, University of Mosul.

Twenty placentae were used in this study, 10 placentae were collected from women who had normal antenatal blood pressure and urine examination and had no other complication throughout their pregnancy (as a control group) and other 10 placentae were collected from women who were diagnosed to have a history of preeclampsia by measuring their blood pressure and performing urine examination for proteinuria in addition to their history of generalized edema during pregnancy particularly edema of the hands and face.

A complete record for every pregnant woman was reported including: name, age, parity, gestational age (estimated by taking into account the menstrual history, early ultrasound and clinical examination), serial measurements of the blood pressure, any medications taken, history of generalized edema and edema of the hands and/or face, review of the past medical history, obstetric history (abortion, dead babies), investigations including ultrasound, urine examination for proteinuria, in addition to any antepartum complications, diabetes mellitus, placenta praevia, fetal anomalies and abruptio placenta.

Following delivery of the fetus and the placenta two pieces were chosen from each placenta, one from the fetal surface and the other from the maternal surface, and then the specimens were put in a

fixative solution (10% neutral formalin) for 24 hour. Each specimen was cut into 1 cm thick slices and dehydrated in graded alcohol solutions (70% alcohol for overnight, two changes in 90% alcohol one hour for each and two changes in 100% alcohol for two hours). The specimens were then immersed in xylene using three changes with one-hour interval for each.

Complete removal of the clearing solution was made by immersing the tissue specimens into three successive paraffin bathes in oven, one hour for each. Finally paraffin blocks were prepared by embedding the tissue specimens using paraffin wax (melting point is 55-60°C) and these paraffin blocks were now ready for sectioning using Reichert Rotary Microtome, serial paraffin sections of 4 micrometers in thickness were cut from each block, the sections were collected and mounted (using DPX) on glass slides then the slides were put for one hour at room temperature then stained to detect the alkaline and acid phosphatase enzymes activities in full term placentae using Gomori's alkaline phosphatase at PH (9) and Gomori's acid phosphatase at PH (3.5) respectively.⁹

Sections of positive and negative control were used for the assurance of accurate reactions of these enzymes. A positive control for alkaline phosphatase enzyme was a rat kidney processed in the same method and treated by Gomori's method while positive control for acid phosphatase enzyme was a small part of the human prostate obtained from Al-Jumhuri Teaching Hospital and processed in the same method and treated by

Gomori's method. Negative control for both alkaline and acid phosphatase enzymes reactions were a placental sections processed in the same method and treated by Gomori's reaction but incubated without using substrate solution.

RESULTS

The full term placentae obtained from the women having no history of preeclampsia or any other maternal complications (i.e. the control group) showed very strong reaction to the alkaline phosphatase enzyme (Figure 1). The villous stroma showed moderate reaction to the alkaline phosphatase enzyme while the cytotrophoblasts showed negative reaction to alkaline phosphatase enzyme. Maternal decidua showed moderate reaction to the same enzyme (Figure 2). The full term placenta obtained from the control group showed negative reaction to the acid phosphatase enzyme in syncytiotrophoblast, villous stroma and in the maternal decidua.

The full term placentae obtained from the preeclamptic women showed diminished alkaline phosphatase activity in the syncytiotrophoblast, villous stroma and in the maternal decidua (Figure 3). Full term placentae of the control group showed negative reaction to acid phosphatase in the maternal decidua and chorionic villi (Figure 4) while there is a considerable increase in the activity of acid phosphatase enzyme in the villi of the placentae obtained from the preeclamptic women (Figure 5).

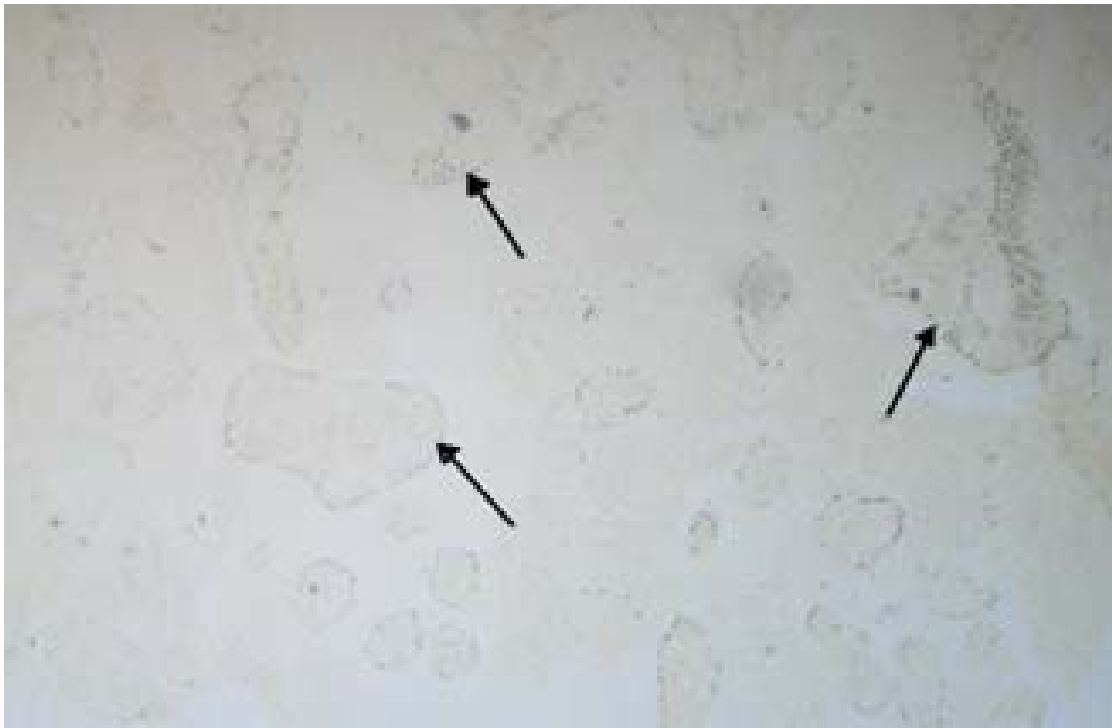


Figure 1. Light microscopical appearance of the normal full term placenta obtained from the control group showing very strong reaction to the alkaline phosphatase enzyme (arrows) (Alk. Ph. X100)

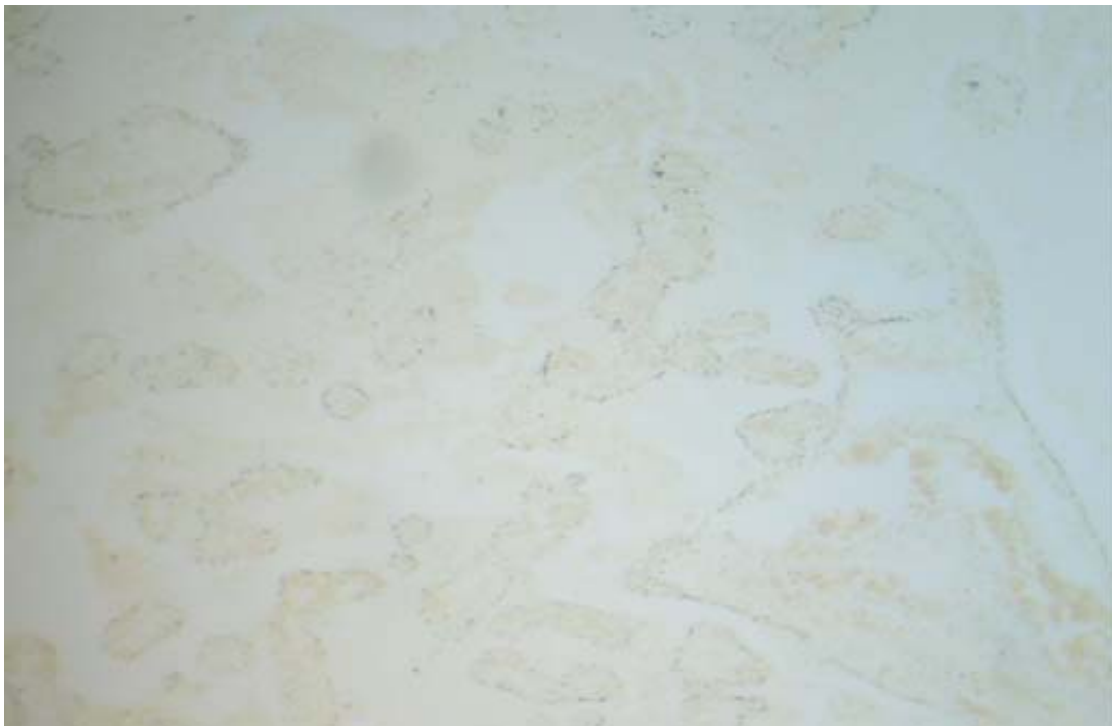


Figure 2. Light microscopical appearance of maternal decidua of the normal full term placenta obtained from the control group showing moderate reaction to the alkaline phosphatase enzyme (Alk. Ph. X100)



Figure 3. Light microscopical appearance of the normal full term placenta obtained from the control group showing negative reaction to the alkaline phosphatase enzyme in the syncytiotrophoblasts (Alk. Ph. X100)

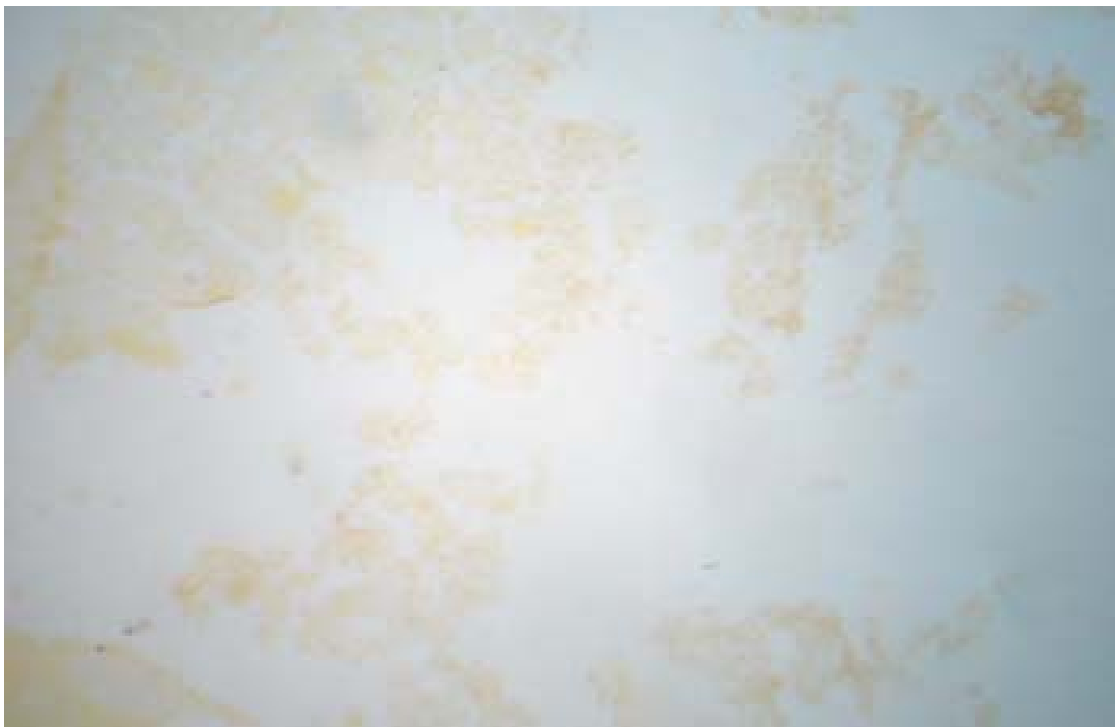


Figure 4. Light microscopical appearance of the normal full term placenta obtained from the control group showing negative reaction to the acid phosphatase enzyme (Acid. Ph. X100)

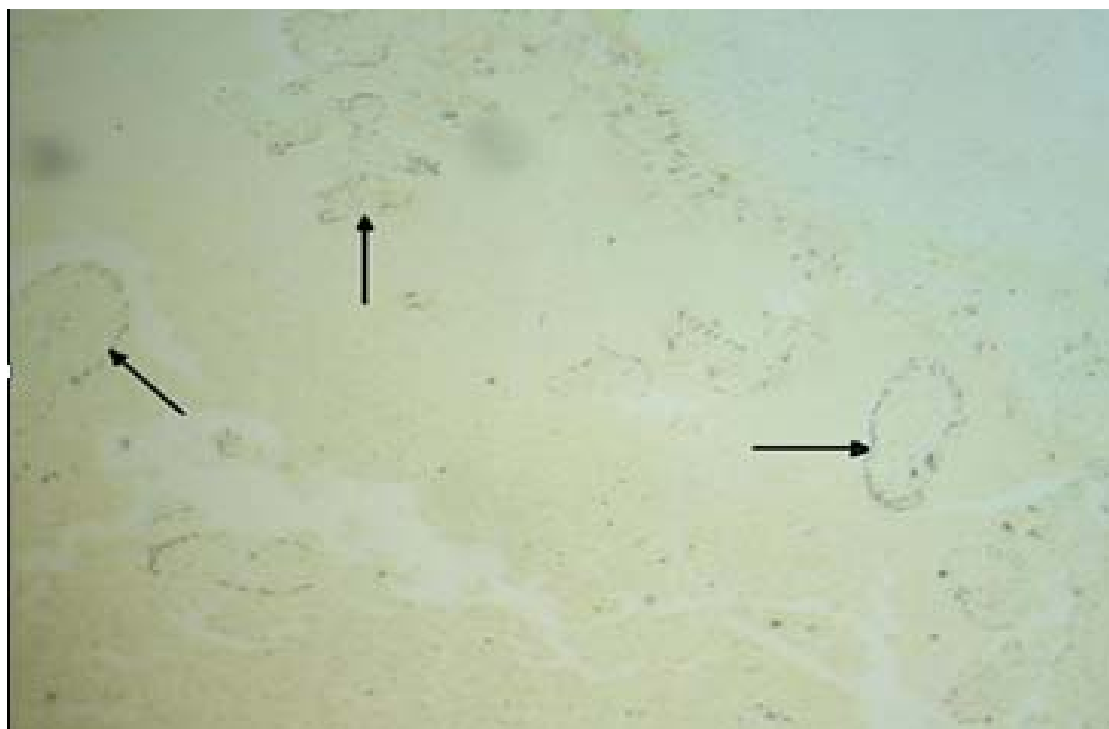


Figure 5. Light microscopical appearance of the full term placenta obtained from the preeclamptic group showing positive reaction to the acid phosphatase enzyme (arrows) (Acid. Ph. X100)

DISCUSSION

Our finding in regard to the distribution of alkaline phosphatase and acid phosphatase enzymes in the placentae obtained from the full term women with no history of preeclampsia or any other maternal complications (i.e. the control group) were similar to those noted by other workers.¹⁰ The alkaline phosphatase is an important enzyme for the trophoblastic transfer thus the full term placenta is adequately equipped with this enzyme.¹¹ It has a vital role in the endocytosis process occurring within the placentae and this function is indicated by the abundant alkaline phosphatase content of the syncytiotrophoblastic basement membrane and their microvilli.

The trophoblasts have two important phosphatase-linked transfer systems, one depends principally upon acid phosphatase enzyme being utilized mainly during the first half of pregnancy and the other depends on alkaline phosphatase enzyme and it dominates during the second half of pregnancy.¹²

The alkaline phosphatase of the placentae obtained from full term preeclamptic women appeared to be affected by the placental ischemia and reduced uteroplacental perfusion leading to a progressive decline in the availability of this enzyme. This is presumably considered as a response to tissue hypoxia which alters the tissue PH of the trophoblast.¹¹

In the present study it is clear that

destruction of the syncytiotrophoblasts due to placental ischemia is the most important factor in decreasing the availability of this enzyme largely because it is formed from the basement membrane of the syncytiotrophoblasts and their microvilli.⁷

It is also appears that syncytial damage and destruction in the placenta obtained from the preeclamptic women is responsible for the increased activity of acid phosphatase which is normally a degenerating enzyme and it is absent in the normal full term placenta.

In normal full term placenta, alkaline phosphatase enzyme gradually increases and it becomes abundant in full term, while acid phosphatase enzyme decreases progressively as gestation proceeds and it become absent at full term. In the placenta obtained from preeclamptic women, this trend is reversed thus alkaline phosphatase enzyme progressively decreases until it disappears. This is usually accompanied by a marked gradual increase in the acid phosphatase activity, such observation is attributed to the continued destructive process within the placenta resulted from reduced uteroplacental perfusion, endothelial cell damage and placental ischemia. This finding differs from the observation of previous workers^{13,14} who found that alkaline phosphatase activity is not lost in the syncytium of the preeclamptic placenta but only there is increase in the activity of acid phosphatase enzyme.

REFERENCES

1. Complications of pregnancy. In: Stead

SM, Stead LG, Kaufman MS, Feig RL, Johnson NC, editors. First aid for the obstetrics and gynecology. NewYork: McGraw-Hill Book Company; 2002. p. 109-12.

2. Granger JP, Alexander BT, Linas MT, Bennet WA, Khalil RA. Pathophysiology of preeclampsia linking placental ischemia/hypoxia with microvascular dysfunction. *Microcirculation* 2002;9(3):147-60.
3. Livingston JC and Maxwell BD. Preeclampsia: theories and speculations. *Wien Klin Wochenschr* 2003;115(5-6):145-8.
4. Gifford RW, August PA, Cunningham G, Green LA, Lindheimer MD, McNellis D, et al. National high blood pressure education program, working group on high blood pressure in pregnancy. *Am J Obstet Gynecol* 2000;183(1):1-26.
5. Roberts JM, Pearson G, Cutler J, Lindheimer. Summary of the NHBPI, working group on researches on hypertension during pregnancy. *Hypertension* 2003;41: 437-41.
6. Kliman HJ. Uteroplacental blood flow: the story of decidualization, menstruation, and trophoblast invasion. *Am J Pathol* 2000;157:1759-68.
7. Al-Sammak MA. Alkaline phosphatase activity during different stages of placental development. *Tekrit Med J* 2002;63:5-12.
8. Johansson S, Wide M. Changes in the pattern of expression of alkaline phosphatase in the mouse uterus and placenta during gestation. *Anat*

- Embryol 1994;190(3):287-96.
9. MacManus JFA, Mowry RW. Staining: histological and histochemical. New York: Harper and Row; 1964.
 10. Messer RH. Heat stable alkaline phosphatase as an index of placental function. Am J Obstet Gynecol 1967;15:459-65.
 11. Jones JP, Fox AC. An ultrahistochemical study of distribution of acid and alkaline phosphatase in placentae from normal and complicated pregnancies. Am J Obstet Gynecol 1975;180(6):10-5.
 12. Pears AGE. Histochemistry. 3rd ed. Edinburgh: Livingstone; 1977.
 13. Luis A. The normal and abnormal placentae. Am J Obstet Gynecol 1974;118:273-5.
 14. Demsey EW. Regional specialization in the syncytial trophoblasts of human placentae. J Anat 1971;108:545-61.

پوخته

گوهورینین هستوکیماوی ل سهر هه فالجویکی بهری (الشنج النفاسي)

ئارمانج: دیار کرنا کارتیکرنا (الشنج النفاسي) ل سهر هه بوونا نه نزیما تین گرتن لئا هه فالجویکی دا.

ریتکین فه کولینی: دوو گروپین هه فالجویکا هاتنه وەرگرتن پستی زاروک بوونی ژ ئافره تین کو ده می دوو گیانی ته واکری و ههر گروپه ک پیکهاتی بوو ژ ده ها. گروپی ئیکی پیکهاتی بوو ژ هه فالجویکین هاتینه وەرگرتن ژ ئافره تین دوو گیانی یین ساخه م و تووشی چی نه خوشیا نه بووین ل بهری یان پستی زاروک بوونی (وهک کونترول) و گروپی دووی پیکهاتی بوو ژ هه فالجویکین هاتینه وەرگرتن ژ ئافره تین دوو گیانی یین تووشی (الشنج النفاسي) بووین. نموونی هه فالجویکی هاته وەرگرتن ژ نه خوشخانا به تول و خه نسا یین فیرکردنی ل میسل ژ شوباتی تا ته مموزا 2006.

ئه نجام: گوهورینین گرتن هاتن دیتن ل گروپا دووی و ئه فه ژی دزفريت بو بو هه لوه شاندا مالکین قاتی جه به لی یی هه فالجویکا ل گروپی دووی و ئه فه ژی ژ بهر کیمبوونا نه نزیمی (فوسفاتین) یی قه لوی و ئه و ژی نه نزیمه که کو ئاماژه ی هه بوونا حه لاندنی دناف هه فالجویکی دا دکه ت.

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THE PREVALENCE OF NEURAL TUBE DEFECTS AMONG NEWBORNS DELIVERED IN AZADI HOSPITAL IN DOHUK CITY, KURDISTAN REGION, IRAQ

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ABSTRACT

Aim The study was performed to highlight the prevalence of neural tube defects (NTDs) among newborns delivered in Azadi teaching hospital in Dohuk.

Methods Study lasted over one year period started from 1st of December 2004 to 30th of November 2005. Fifty six newborns (alive and stillbirth), 40 females and 16 males, were found to have different types of NTDs.

Results The prevalence of NTDs was found to be 4.7 per 1000 live births, which was higher than similar rates estimated in other areas in the world. The mothers of the affected newborns were not consuming folic acid before conception, which may, to some extent, explain the high rate in this study.

Conclusions Further similar studies, however, are required in North of Iraq to document the findings of this study. All women should consume 0.4 mg folic acid daily during their reproductive years to protect against this defect.

DMJ 2007;1(1):42-8.

Key words: Neural Tube Defects (NTDs), Newborns, Congenital anomalies

Neural tube defects (NTDs) are among the most serious and common birth defects affecting more than 300,000 fetuses or infants in the world each year. Approximately 4000 pregnancies in the United States are affected each year, one third of which are spontaneously lost or electively terminated. Such defects include anencephaly, encephalocele, spina bifida occulta, spina bifida cystica (meningocele and myelomeningocele) as well as several less common forms. They account for

most congenital anomalies of the central nervous system (CNS). These defects are the result of failure of the neural tube to close spontaneously between the 3rd and 4th week of in utero development.¹⁻³

The NTDs are thought to arise from a combination of genetic and environmental factors.²⁻⁴ Over the last century, teratogens implicated in the etiology of NTDs in human include potato blight, maternal hyperthermia, low economic status, antihistamines, sulfonamides, nutritional deficiencies, vitamin deficiencies and anticonvulsants. Of all the suspected teratogens, folate deficiency and anticonvulsants (carbamazepine and valproic acid) have been most strongly tied to the development of NTDs.¹⁻⁴

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Females are affected more frequently than males.^{2,3,5} Considerable geographical variation in NTDs rates are also exist with noted hotspots in Guatemala, Northern China, Mexico and parts of the United Kingdom.³⁻⁵ Anencephaly is incompatible with life, other NTDs may give rise to progressive neurological deterioration which may present early after birth or later in life. The neurological deficit may be due to accompanying hydrocephalus, a Chiari II malformation, tethering of the cord, cystic mass or fibrous band compressing the neural elements. Another possible complication is meningitis especially in open NTDs.^{2,3} Although the mechanism of action remains unknown , epidemiologic studies have shown that periconceptional use of folic acid can prevent at least 50% of NTDs and may reduce the occurrence of other birth defects.¹⁻⁶ Public health agencies recommend that all women who are capable of becoming pregnant consume 0.4 mg of folic acid daily in the form of fortified foods or dietary supplements.^{7,8} Because NTDs occur just 3 to 4 weeks after conception, often before pregnancy is confirmed, it is important that women who are capable of becoming pregnant, regardless of pregnancy intention, take folic acid daily.^{9,10}

The aim of this study is to evaluate the size of NTDs in Dohuk, Iraq.

PATIENTS AND METHODS

The study was undertaken in Dohuk city over a period of one year started from the 1st of December 2004 to the 30th of

November 2005. Dohuk is the 3rd largest city in Kurdistan with a population of about 280,000 (according to the last census in 2006). A prospective study was conducted to record all live births in Azadi teaching Hospital, which is the main general Hospital in Dohuk, and to estimate the number of those with NTDs. All newborns were examined by senior house officer and those with overt NTDs were re-evaluated by the author to determine the type of the defect. A thorough history was taken from the mothers of the affected newborns including exposure to teratogens, maternal illnesses during pregnancy, a previous offspring with NTD, the degree of consanguinity, and we asked specifically about taking folic acid before and during early weeks of pregnancy.

RESULTS

The total number of live births during the period of the study was 11945 births, about 20% of deliveries occurred outside the hospital according to the preventive health department, directorate of Dohuk, 2006. Fifty six of the newborns were found to have overt NTDs. Spina bifida occulta was not included because most individuals are asymptomatic and the condition is usually of no consequence. All newborns with anencephaly and encephalocele died during first 24 hours of life and surgical operation done for those with meningocele and myelomeningocele. The prevalence of NTDs in Dohuk was 4.7 per 1000 live births.

All interviewed mothers were not taking folic acid before conception. There

was no history of exposure to teratogens, and no history of maternal diseases during pregnancy. Consanguinity was present in 23 (41%) cases. Other anomalies (especially of the extremities) were seen in 16 (28.5 %) cases. In 4 (7%) cases there was a history of affected sibling with NTD. The type of the defect and the sex of the newborns are shown in the table1.

DISCUSSION

Neural tube defects are among the most common birth defects contributing to infant mortality and serious disability¹¹ In the United Kingdom and Ireland , yearly prevalence of NTDs has declined from 4.5 per 1000 births in 1980 to 1-1.5 per 1000 in the 1990s, in contrast ,in the rest of Europe the prevalence during the 1980s and thereafter was close to 1 per 1000 births.¹² The incidence of NTDs has declined by 50% between the years 1970 and 1989 (1.3 per 1000 to 0.6 per 1000 live births) in the United States.^{7,8,11} In this study, which is the first to be carried out in Dohuk, the prevalence of NTDs is found to be 4.7 per 1000 live births; it is regarded as a high rate when compared with that of other areas in the world. A similar high prevalence of 4.48 per 1000 live births was

seen by Alrabaty in Erbil city (Northern Iraq).¹³ One possible explanation for this high NTDs rate in our area is that most of the pregnancies were unplanned and the women were not taking folic acid before conception. However, the high prevalence in this study may be because only the Azadi hospital deliveries have been included. Probably other genetic and environmental factors play a role.

Evidence to date suggests that supplementation with a multivitamin containing 0.4 mg of folic acid prevent the occurrence of more than 50% of NTDs when it is taken before conception and continued throughout the first trimester of pregnancy.⁷ A significant decrease in the incidence of NTDs was seen in Northern region of China, an area known to have a high prevalence of NTDs, after taking folic acid by pregnant women before pregnancy.¹⁴ Study of NTDs in the United States by Center for Disease Control and Prevention shows a significant reduction of anencephaly and other NTDs following the introduction of fortification of wheat flour with folic acid. However, it is likely that there is genetic variation in response to folic acid, which helps to explain why most mothers with folic acid deficiency do not bear children with NTD and why some

Table 1. Types of NTDs and the sex of the affected newborns

Type of NTD	Cases No. (%)	Male No. (%)	Female No. (%)
Anencephaly	21 (37.5%)	11 (52%)	10 (48%)
Meningocele	19 (34%)	3 (16%)	16 (84%)
Myelomeningocele	12 (21.5%)	2 (17%)	10 (83%)
Encephalocele	4 (7%)	-	4 (100%)
Total	56 (100.0)	16 (28%)	40 (72%)

who ingest adequate amounts nonetheless do bear children with NTDs.⁵ Forty (72%) cases in this study were females.

The sex difference seems to be consistent in most studies, in which about 55-70% of NTDs occur in females. This female predominance is seen in both still and live births.¹⁻⁴ Consanguineous mating was present in 23 (41%) cases. The rate of consanguineous marriage in Dohuk was found to be 25%.¹⁵ This may, partly, explain the high prevalence of NTDs as consanguinity increases the chance that a mating couple will both carry the same disease gene.³ In 4 (7%) cases there was a history of a previous sibling with NTD. Being a multifactorial disorder, the risk of recurrence of NTDs depends on many factors including the number of affected siblings and the severity of the defect. In contrast to most single-gene diseases, recurrence risk for multifactorial diseases can change substantially from one population to another. This is because gene frequencies as well as environmental factors can differ among populations. For example, the recurrence risk for British parents who have had one child with a neural tube defect is 5% while in North America it is about 2% to 3%. Consistent with a multifactorial model, the recurrence risk increase with additional affected siblings. A recent Hungarian study showed that the overall prevalence of NTDs in that country was 1/300 births and that the sibling recurrence risk were 3%, 12%, and 25% after one, two and three affected offsprings respectively.³

The efficacy of folic acid in preventing a subsequent occurrence of

neural tube defect among the fetuses or infants of women with a previous affected fetus or infants was first conclusively demonstrated by the Medical Research Council randomized study in the United Kingdom. Women who took 4 mg of folic acid daily during the periconceptional period in a subsequent pregnancy had a 72% reduction in the risk having an affected fetus or infant.⁵

CONCLUSIONS AND RECOMMENDATIONS

This study and that conducted in Erbil showed a high rate of NTDs in Northern Iraq. This might partly be due to the lack of preventive measures (i.e. the periconceptional use of folic acid). Further community based studies are required in North of Iraq to affirm the findings seen in this study and to evaluate other possible risk factors. In the meantime it is highly recommended that all women of child bearing age who are capable of becoming pregnant should consume 0.4 mg of folic acid daily. The importance of fertile women's taking daily multivitamins that contain folic acid should be stressed in public health education and physician counseling, both for women who are contemplating and those who are not contemplating pregnancy. Schools should encourage girls to take daily multivitamins that contain folic acid.

REFERENCES

1. American Academy of Pediatrics Committee on Genetics. Folic acid for

- the prevention of neural tube defects. *Pediatrics* 1999;104:325-7.
2. Behrman RE, Kliegman RM, Jenson HB. *Nelson textbook of pediatrics*. 17th ed. USA: Saunders; 2004.
3. Jorde LB, Carey JC, Bamshad MJ, White RL. *Medical genetics*. 2nd ed. USA: Mosby; 2000.
4. Watkins ML. The efficacy of folic acid prophylaxis for the prevention of neural tube defects. *Mental Retard Dev Disabil Res Rev* 1998;4:282-90.
5. MRC Vitamin Study Research Group. Prevention of neural tube defects: results of the Medical Research Council Vitamin Study. *Lancet* 1991;338:131-7.
6. Czeizel AE, Dudas I. Prevention of the first occurrence of neural tube defects by periconceptional vitamin supplementation. *N Engl J Med* 1992;327:1832-5.
7. Centers for Disease Control and Prevention. Recommendations for the use of folic acid to reduce the number of cases of spina bifida and other neural tube defects. *MMWR* 1992;41(RR-14):1-7.
8. Institute of Medicine. *Dietary reference intake: folate, other B vitamins and choline*. Washington DC: National Academy Press; 1998.
9. Moore KL, Persaud TVN. *The Developing human: clinically oriented embryology*. 6th ed. Philadelphia: W.B. Saunders; 1998.
10. McNulty H, Cuskelly GJ, Ward M. Response of red blood cell folate to intervention: implications for folate recommendations for the prevention of neural tube defects. *Am J Clin Nutr* 2000;71 Suppl 1:1308-11.
11. Centers for Disease Control and Prevention. Surveillance for anencephaly and spina bifida and the impact of prenatal diagnosis, United States, 1985-1994. *MMWR* 1996; 44(SS-4):1-13.
12. Eurocast Folic Acid Working Group. Eurocast special report: prevention of neural tube defect by periconceptional folic acid supplementation in Europe. University of Ulster. Eurocast central registry. [online]. 2003 [cited 4 Aug 2006]. Available from: URL: <http://www.eurocast.ulst.ac.uk/pubdata/folic%20acid.htm>
13. Alrabaty A. Birth defects among neonates admitted to ICU in Erbil maternity and children hospital. *Zanco Journal of Medical Sciences* 2001;1:66-77.
14. Robert JB, Zhu Li, David E, Song Li, Synthia AM, Hong wang, et al. Prevention of neural tube defects with folic acid in China. *N Eng J Med* 1999;341(24):1864.
15. Al-Alawi NA, Jubrael JM, Hughson M. Molecular characterization of β -thalassemia defects in Dohuk –Iraq. *Hemoglobin* 2006;30(4):479-86.

پوخته

رێژی دیاربوون و بلاڤ بوونا پیشیل بوونین لوولا ده‌ماری لجه‌م بچیکیت نی بووی نه‌خوشخانا نازادی لبارێری ده‌وکی هه‌ریما کوردستانا عیراقی

ئارمانج: ئەڤ لیکولینه هاته‌ کرن ژبوو دیارکنا رێژی پیشیلبوونیت لوولا ده‌ماری لجه‌م بچیکیت بووی ل نه‌خوشخانا نازادی یاقیرکرنی ل بارێری ده‌وکی (هه‌ریما کوردستانا عیراقی).

رێکێن قه‌کولینی: قه‌کولین قه‌کیشا بو ماوی ساله‌کی ئانکو ژ ده‌سپیکا هه‌یفا کانوونا ئیکی سال 2004 ی وتا دوماهیکا هه‌یفا چریا دووی سال 2005 ی، و جه‌ژی نه‌خوشخانا نازادی لبارێری ده‌وکی بوو.

ئه‌نجام: ژمارا بچیکیت ساخ هاتینه‌ سه‌ر دنیایی دفی ماوه‌ی دا (11945) بوون . (56) پینچی وشه‌ش ژوان بچیکا (40) کچک و 16 کورک تووشی پیشیلبوونیت لوولا ده‌ماری ببوون و ئەڤ پیشیلبوونیت نه‌ سروشتی دجیاجیا بوون . رێژا بلاڤ بوونا فان پیشیل بوونا دفی لیکولینی دا (4.7) بوو بو هه‌ر هزار بچیکیت ساخیت نی بووی پینتییه‌ دیارکهن کو ئەڤه‌ رێژه‌ یه‌کا بلنده‌ هه‌که‌ هاتوبه‌راورد که‌بن دگه‌ل جیهانی . ده‌یکیت وان بچیکیت تووشی فان پیشیلبوونا بووین ده‌رمانی (فولیک ئەسید) وه‌رنه‌دگرت به‌ری کو دووزک بین وه‌روسا ده‌می دووزکی دا وه‌رنه‌دگرت ، ونه‌دیره‌ ئەڤه‌ییت ئەگه‌را فی چهن‌دی ، کو دیاربوونا فی رێژی بلندیی پیشیلبوونا.

ده‌رئه‌نجام: ئەم دپیتی لیکولینیت دیتترین لسه‌ر فی بابته‌ی لکوردستانی بو په‌سهند کرنا فی لیکولینا قه‌کری . وژبو کیم کرنا رێژا دیاربوونا فان پیشیلبوونا ئەم بباشتر دینن و ئاموژگاریا مه‌ ئەوه‌ کو ژنکیت دووگیان فولیک ئەسیدی وه‌رگرن بدرێژا هیا ماوی دووزکی.

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OPTICAL INTERNAL URETHROTOMY IN THE TREATMENT OF URETHRAL STRICTURE DISEASE

SHAKER S. BALINDI, MBChB, FICU*

Submitted 8 March 2007; accepted 16 August 2007

ABSTRACT

Objectives To evaluate the efficacy of internal urethrotomy in the treatment of urethral stricture disease as a first line of treatment.

Patients and methods The study has been prospectively undertaken for forty five patients with urethral stricture disease who were treated with cold-knife internal urethrotomy followed by regular self calibration or hydrostatic urethral dilatation via starting micturations while applying pressure over distal urethra so leading to urethral distention.

Results The age of the patients ranged between 22-80 years.

The follow up period was from 6 months to 3 years. The success rate was 88%. The complication occurred in 6.6% of cases.

Conclusion Internal urethrotomy could be regarded as the first treatment of choice in patients with a single, short urethral stricture or post urethroplasty stricture.

DMJ 2007;1(1):49-57.

Key words: Urethral stricture, Internal urethrotomy, Hydrostatic urethral dilatation

Urethral strictures are fibrotic narrowing composed of dense collagen and fibroblasts. The narrowing restricts urine flow and cause dilation of the proximal urethra and prostatic ducts.¹

The male urethra can be divided into 2 parts; the posterior urethra, including the membranous and prostatic urethra and the anterior urethra. The anterior urethra includes the navicularis and penile and bulbar tracts and is surrounded by the corpus spongiosum soft tissue.²

Currently urethral stricture disease is relatively common, most strictures being acquired from injury or infection. Blunt perineal trauma causes injury to the bulbar urethra; pelvic fractures result in urethral distraction defects in the posterior urethra, but iatrogenic causes, including urological instrumentation and placing indwelling catheters, which result in strictures anywhere in the urethra, are the most common causes.³

Patients who have urethral strictures most often present with obstructive voiding symptoms or urinary tract infections, such as prostatitis or epididymitis.⁴

To devise an appropriate treatment plan, it is important to determine the location, length, depth, and density of the stricture. The length and location of the stricture can be determined with

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radiography, urethroscopy and ultrasonography and the depth and density of fibrosis as evidenced by ultrasound evaluation of the urethra.⁴

The traditional treatment for urethral stricture disease is urethral dilatation or urethrotomy. Both have an ancient history but currently urethrotomy tends to be associated with visual urethrotomy, which is of course a recent development; traditional urethrotomy was blind.⁵

The purpose of this study was to evaluate the clinical experience of a tertiary regional center by applying the internal urethrotomy in the treatment of urethral stricture disease in Duhok as a first line of treatment.

PATIENTS AND METHODS

Forty five male patients were identified prospectively with signs & symptoms of urethral stricture disease. All were evaluated and operated upon in Azadi

general teaching hospital by direct internal urethrotomy using a guide wire or ureteric catheter Fr 4 to introduce it through a stenosed area.

The incision has been done at 12 o'clock position via a cold knife (Figure 1). In cases of severe stricture fibrous tissue was resected.

The ages ranged from 22 to 80 years (mean age 46.9 years).

All operations were done under general anesthesia by one urologist. Following the operations, the urethral catheter Fr 16 was left inside for 72 hours. All patients were instructed to carry out daily urethral calibration with a Foley catheter Fr 16 for 4 weeks and then biweekly for 2 months. Two patients have refused to do self catheterization.

Retrograde urethrogram and voiding cystourethrogram were considered for patients with suprapubic catheters and retrograde urethrogram was done for all patients for definite diagnosis.



Figure 1: Photograph of urethral stricture using colling knife

RESULTS

Between January 2002 and December 2005, 45 patients with urethral stricture disease (Figure 2 and Figure 3) were treated by internal urethrotomy and regular urethral calibration, and the results were as follow:

The presenting symptoms are shown in table 1. The most common presentation was weak urinary stream (24), followed by suprapubic catheter (7) and dribbling of urine (7), retention of urine (5) and renal failure (2).

The etiology of the stricture in patients included in the study is shown in the table 2.

The site and number of stricture in these patients are shown in tables 3 and 4.

Figure 4 demonstrates urethral stricture after correction.

Following internal urethrotomy the median hospital stay was 42.9 hours.

The patients were followed up for a mean of 19.2 months (3-35) months.

The results of analysis after first internal urethrotomy were as follow: Good results 30, improved 10, poor results 5.

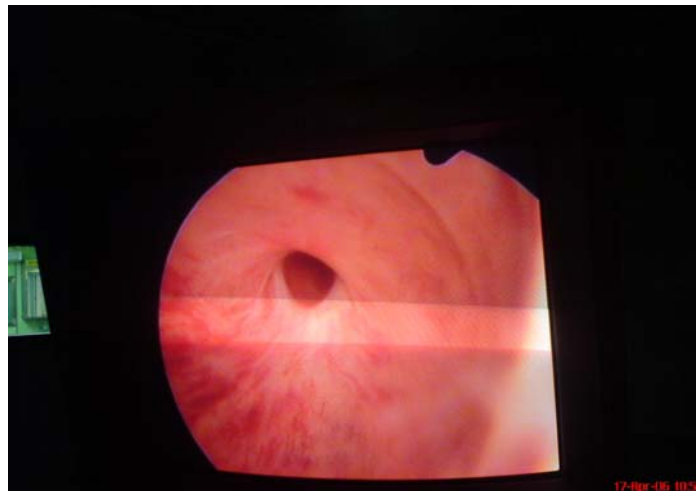


Figure 2. Photograph showing urethral stricture

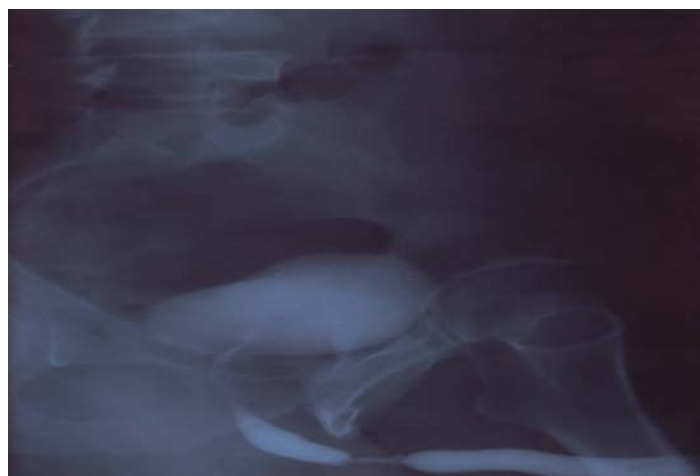


Figure 3. Retrograde urethrogram demonstrating bulbar urethral stricture

Good results

All patients have achieved normal voiding after single attempt of urethrotomy and over a period of follow up between (3- 35) months.

Improvement

The patients have improved, but they have needed another session of internal urethrotomy within 6 months period.

Poor results

The patients have required repeated admissions and repeated procedure within a short period of time (1-2 months).

All patients included in the study were potent and continent preoperatively and remained like that postoperatively except one patient who had preoperative erectile

dysfunction (a case of pelvic fracture distraction injury) and has remained impotent postoperatively.

All patients were received per and post-operative parental antibiotics.

Two patients have developed severe hematuria postoperatively which necessitate blood transfusion (2 units of blood for each).

Two patients had balanitis xerotica obliterans and have developed recurrences of stricture after a short period of internal urethrotomy, so Y-V meatoplasty were done for both of them with considerable improvement.

Four patients have developed fever and rigor and responded very well to parenteral antibiotics.

Table 1. Clinical presentation

Signs &symptoms	No. (%)
Weak stream	24 (53.3)
Dribbling	7 (15.5)
Retention	5 (11.1)
Suprapubic cystostomy	7 (15.5)
Renal failure	2 (4.4)
Total	45 (100.0)

Table 2. Etiology of urethral stricture

Etiology	No. (%)
Traumatic	12 (26.6)
Infection	11 (24.4)
Catheterization	8 (17.7)
Post-TURP	5 (11.1)
Post-urethroplasty	4 (8.8)
Others	5 (11.1)
Total	45 (100.0)

Table 3. Site of Urethral Stricture

Site	No. (%)
Bulbomembranous	24 (53.3)
Membranous	11 (24.4)
Penile	5 (11.1)
Prostatic	5 (11.1)
Total	45 (100.0)

Table 4. No. of Strictures

Type	No. (%)
Single	39 (96.6)
Multiple	6 (13.3)
Total	45 (100.0)

**Figure 4. Retrograde urethrogram of urethral stricture after correction**

DISCUSSION

Urethral stricture is one of the oldest known urological diseases and remains a common problem with a high morbidity despite earlier prediction to the contrary.⁶

Urethral dilatation has long been the standard treatment for patients with urethral stricture. However, in many patients such dilatation may be difficult, painful, or have to be done at frequent

intervals.⁷

Direct vision internal urethrotomy that was described by Sacher in 1974 is one of the most popular primary modalities of treatment.⁸

The technique of urethral dilatation was recorded in the sixth century BC in Hindu writings that described various surgical instruments, such as urethral dilators and catheters, to manage urethral obstruction.⁹

In this study we found a mean age of 46.9 years (range 22-80 years) which is similar to the finding of Meneghini et al.¹⁰

The most common presentations in our study were obstructive voiding symptoms & urinary retention. Andrew et al.³ also found that obstructive voiding symptoms remain the typical reason for evaluating urethral stricture disease.

The etiology of urethral stricture in the present study is traumatic in 26.6%, infection in 24.4%, catheterization in 17.7%, post-TURP in 11.1%, post urethroplasty in 8.8% and others in 11.1. This is similar to report of Dobrowolski et al.¹¹ who documented that the most common cause of posterior urethral stricture is RTA (Trauma). While for anterior urethral stricture is iatrogenic (during catheterization or cystoscopy).

Thirty patients (66.6%) achieved good results after single attempt of internal urethrotomy, which is in concordance with other studies.^{12,13}

Among five patients who had poor results two of them had BXO who were treated first by internal urethrotomy and later on by Y-V meatoplasty which is the treatment of choice. Other three patients had multiple and long urethral strictures and refused to do self urethral calibration post operatively.

Frequent and regular post-internal urethrotomy self dilation of the urethra could reduce stricture recurrence rate at one year by as much as 46 %.¹⁴

Patients who are poor candidates for initial or repeated internal urethrotomy include those with multiple, long (2-5 cm) penile or posterior strictures.¹⁴ All patients

were sexually potent pre operatively and remained like that except one of them who had pelvic fracture distraction injury and became impotent after injury. This patient, initially, underwent urethroplasty and internal urethrotomy later on but still is suffering from erectile dysfunction.

Many urologists suggested that incontinence and erectile dysfunction were purely traumatic rather than surgical.¹⁵

CONCLUSION

In keeping with reported literatures, internal urethrotomy could be regarded as the treatment of choice in patients with a single, short (<2cm) anterior urethral stricture or stricture following posterior urethroplasty if followed by regular self calibration for 3 months.

REFERENCES

1. Mc Aninch JW. Disorders of the penis and male urethra. In: Tanagho EA, Mc Aninch JW, editors. *Smith's General Urology*. 16th ed. USA: Appleton and Lange; 2003. p. 436-45.
2. Barbagli G, Palminteri E, Lazzeri M, Guazzoni G. Anterior urethral stricture. *BJU Int* 2003;92(5):497-505.
3. Peterson AC, Webster GD. Management of urethral stricture disease: developing options for surgical intervention. *BJU Int* 2004; 94(7):971-7.
4. Jordan GH, Schlossberg SM. Surgery of the penis and urethra. In: Walsh PC, Retik AB, Vaughan ED Jr, Wein AJ, editors. *Campbell's Urology*. 8th

- ed. Vol. 3. Philadelphia: WB Saunder; 2002. p. 3886-952.
5. Andrich DE, Mundy AR. Urethral strictures and their surgical treatment. *BJU Int* 2000;86(5):571-80.
 6. Steenkamp JW, Heyns CF, De Kock MLS. Internal urethrotomy versus dilation as treatment for male urethral strictures: a prospective randomized comparison. *J Urol* 1997;157(1):98-101.
 7. Smith PJB, Dunn M, Roberts JBM. Surgical management of urethral stricture in the male. *Urology* 1981;18(6):582-7.
 8. Gnanaraji J, Devasia A, Gnanaraji L, Pandey AP. Intermittent self catheterization versus regular out patient dilatation in urethral stricture: A comparison. *ANZ J Surg* 1999; 69(1):41.
 9. Khoury JM. Internal Urethrotomy. In: Graham SD, Glenn JF, editors. *Glenn's urologic surgery*. 5th ed. Philadelphia: Lippincott Raven; 1998. p.124-31.
 10. Meneghini A, Cacciola A, Cavarretta L, Abatangelo G, Ferrarrese P, Tasca A. Bulbar urethral stricture repair with mucosa graft urethroplasty. *Eur Urol* 2001;39(3): 64-7.
 11. Dobrowolski ZF, Weglarz W, Jakubik P, Lipczynski W, Dobrowolski B. Treatment of posterior and anterior urethral trauma. *BJU Int* 2002;89(7):752-4.
 12. Shittu OB. Internal optical urethrotomy in the management of urethral strictures in Nigerians: technique and outcome. *Afr J Urol* 2001;7(2):62-5.
 13. Wright JL, Wessells H, Nathens AB, Hollingworth W. What is the most cost-effective treatment for 1 to 2-cm bulbar urethral strictures: societal approach using decision analysis. *Urology* 2006;67(5):889-3.
 14. Naudé AM, Heyns CF. What is the place of internal urethrotomy in the treatment of urethral stricture disease? *Nat Clin Prac Urol* 2005;2:538-45.
 15. Jalbani MH, Shaikh NA. Experience with cold knife optical internal urethrotomy and temporary dilatation. *Pakistan J Med Res*;41(4):145-7.

پوخته

چاره‌سهریا ته‌نگیا میزروا کورت ب برینا دهرونیا میزروا کورت ب ریکا دیتنی

نارمانج: بو هه‌لسه‌نگاندنا کار ئەمه‌دیا برینا میزروا کورت یا دهرونی دده‌رمانی نه‌خوشیا ته‌نگیا میزروا کورت وه‌ک چاره‌سهریا سهره‌کی.

نه‌خوش وشپوه: فه‌کولینه‌ک ب شیوی ناینده‌نگه‌ری بو 45 نه‌خوشین توشی ته‌نگیا میزروا کورت هاته نه‌نجامدان کو هاته چاره‌سهرکرن ب برینا دهرونی میزروا کورت ب ریکا دیتنی وپاشی فره‌کنا میزروی ب ریکا مایعی وده‌ست پیکرنا میزکرنی وده‌ره‌قی وه‌ختی دا فشار دئێخسته سهر دوماهی یا میزروا کورت دا میزرو فره‌بیت.

ئه‌نجام: ژبی نه‌خوشان 22-80 سال بو، وه‌ختی دوویف چوونی 6 هه‌یف هه‌تا 3 سالان بو. پلا سهرکه‌فتنی 88% بو ئاریشین پشتی نشته‌رگه‌ریی بو 6.6% ژ نه‌خوشان پشهان.

به‌ره‌م: دشین یئژین کو برینا دهرونیا میزروی چاره‌سهریا سهره‌کی یه بو وان نه‌خوشین ته‌نگیا و به‌راهییا میزروی یان ته‌نگیا پشتی نشته‌رگه‌ریی هه‌ی.

45) :
3 6 80-22 :
6.6% 88% :
:

CARPAL TUNNEL SYNDROME AND ASSOCIATED ENTRAPMENT MONONEUROPATHIES

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ABSTRACT

Aim This study was carried out to assess the electroneurophysiological findings and associated entrapment mononeuropathies in cases of carpal tunnel syndrome (C.T.S.).

Patients and Method One hundred and fifty patients (122 females and 28 males) with clinical suspicion of C.T.S. were included in the study. A questionnaire containing all important information was completed for each case. Nerve conduction study for median nerves and other peripheral nerves was performed according to criteria of Hull Royal Infirmary, using computerized EMG machine.

Results About 80% of referred cases were in the age range of 21-50 years and 70% of them had symptoms for less than 2 years, 120 (80%) of referred cases showed positive C.T.S., 108 (72%) of positive cases had right C.T.S. whereas 44% had left C.T.S. The most common associated entrapment mononeuropathies were right and/or left ulnar tunnel syndrome (U.T.S.). All those with right U.T.S. were right handed, in addition to that 7% of right handed patients had left U.T.S. Entrapment of other peripheral nerves were also reported but at different rates. Parasthesia / numbness of hand(s) was the major presenting symptom and 58% of positive cases had symptoms other than those of C.T.S.. Out of 99 positive females, 79 were housewives and 33% of positive males were building constructors.

Conclusions Major peripheral nerves are liable to compression in some points along their course. Many occupations may affect more than one peripheral nerve, rendering them vulnerable to entrapment. When performing Nerve Conduction Studies (N.C.S.) for suspected cases with C.T.S., it is important to do N.C.S. for other nerves to confirm or exclude their entrapment.

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Key words: Carpal tunnel syndrome, Entrapment mononeuropathies, Nerve conduction study

Compression of the median nerve at the wrist is usually referred to as carpal tunnel syndrome (C.T.S.). The term (C.T.S.) was first described in 1883 by Sir James Paget; in 1951 the syndrome received added attention when Phalen published the first report of a large series

of decompression. Phalen also described the well-known diagnostic wrist flexion maneuver that was named after him (Phalen's test).¹

C.T.S. is by far the most common entrapment neuropathy of the upper extremity.²⁻⁴ The carpal tunnel, a fibro-oseous tunnel in the wrist, is bounded below by bones and a thick fibrous sheet that form the roof of this canal.⁵ It has been shown that the size of myelinated nerve fibers is reduced under the ligament

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(fibrous sheet) and because the tunnel is rigidly bounded in all sides, therefore, any condition that leads to swelling of the synovial tissue or increased volume of structures inside the carpal tunnel will cause compression of the median nerve.²

The syndrome affects an estimated 3% of adult American's,⁶ and approximately three times more common in women than in men,^{2,6,7} usually affects the dominant hand and the symptoms often appear in people with jobs that require heavy and repeated use of hands such as food processing, manufacturing, logging and others.^{8,9} C.T.S. in United States is quite costly in terms of time lost from work and expensive medical treatment. The United States Department of Labor reported that in 2003 the average number of missed days of work due to C.T.S. was 23 days, costing over 2 billions \$ / year.⁷

Although many cases are idiopathic, C.T.S. is also associated with a number of systemic conditions including rheumatoid arthritis, hypothyroidism, diabetes mellitus, gout and colle's fracture.^{4,10,11} The classical symptoms of C.T.S. are pain, numbness and tingling in the distribution of the median nerve, although numbness in all fingers may be a more common presentation.^{12,13} Symptoms usually worsen at night and can awaken the patient from sleep. The pain and parasthesia may radiate to forearm, elbow and shoulder. Decreased grip strength may result in loss of dexterity and in severe cases thenar muscle atrophy may develop. Although one hand typically has more severe symptoms, both hands often are affected.¹⁴ The combination of proper history,

physical examination and electrodiagnostic studies such as nerve conduction studies (NCS) permits the most accurate diagnosis.¹⁵ NCS are useful for confirming the diagnosis in suspected cases and ruling out neuropathy and other nerve entrapments.

Prolonged distal latency at wrist and/or decreased compound muscle action potential (CMAP) of thenar muscle and/or abnormalities in sensory conduction and/or sensory nerve action potential (SNAP) are potentially diagnostic findings of C.T.S.¹⁶⁻¹⁸

The purpose of this study is to find out the major presenting symptoms, predisposing factors and occupation of patients with clinical suspicion of C.T.S. in Dohuk City / Iraqi Kurdistan Region; to asses the electrophysiological changes in suspected cases; and to detect associated entrapments of other peripheral nerves (namely ulnar, posterior tibial and common peroneal nerves).

PATIENTS AND METHODS

The study was conducted at the EMG Unit, Azadi Teaching Hospital in Dohuk City from 1/2/2005 to 1/12/2006. All participants (patients and controls) were adult of both sexes.

Twenty three apparently healthy subjects (16 females and 9 males), with an age range of 19-42 years (mean 34±11) approved to participate in the study for measurement of normal values of peripheral nerves.

One hundred and fifty referred patients to EMG Unit with clinical

suspicion of C.T.S. were selected by simple random selection, their age ranging between 18-70 years (Mean \pm SD of mean 39 ± 19) and (122 were female, 28 were male). All persons were informed about the experimental design and accepted to be enrolled in the study. The questionnaire form was completed for each followed by NCS of the median nerves and other nerves.

Each nerve has its specific technique for electrophysiological examination. The same techniques reported in the Clinical Neurophysiological Manual of Department of Neurophysiology in Hull Royal Infirmary Teaching Hospital,¹⁸ were used in the present study.

Computerized EMG machine (D-81245, 2000, Munich, Schwarzer), Silver made surface electrodes (Schwarzer 630506, 11-2002), ring electrodes (Schwarzer, 518801, 2-2000) and electrodes jell (Spectra Gel, Schwarzer, 605125) were all used for accomplishing the work. Confidence limits for distal latency, nerve conduction velocity (NCV), CMAP, SNAP obtained from normal subjects were used as control data. Values greater than the confidence limits of distal latency was regarded as prolonged distal latency (which indicates entrapment of the nerve). However, values of NCV and/or

CMAP and/or SNEP lower than the confidence values were regarded as abnormal and indicated entrapment of the examined nerve. Cases with abnormal NCS due to peripheral polyneuropathy were excluded from the study.

RESULTS

The age range of the study group was between 18-70 years (mean 39 ± 19). There were 122 females and 28 males. More than 80% of the patients involved in the study were between 21-50 years of age. Almost 70% of patients had symptoms for less than 2 years. Of the 150 cases, 120 (99 female and 21 male) had unilateral or bilateral positive C.T.S., while 30 were negative (Table 1).

Table 2 shows that 108 (72%) cases had evidence of electroneurophysiologically positive right C.T.S. (105 were right handed and 3 were left handed). Moreover, 66 (44%) of cases had left C.T.S. (58 were right handed and 8 were left handed). Compression of right and left ulnar nerves at the wrist(s) (ulnar tunnel tunnel syndrome) was the most common associated entrapment neuropathies (11% and 9% respectively). All patients having right U.T.S. were right handed, 25% of left handed and 7% of

Table 1. Distribution of cases by sex

Sex	Positive No. (%)	Negative No. (%)	Total No. (%)
Female	99 (81)	23 (19)	122 (100)
Male	21 (75)	7 (25)	28 (100)
Total	120 (80)	30 (20)	150 (100)

right handed had left U.T.S.

Other associated nerve entrapments in right handed patients were right peroneal and cubital tunnel syndrome, each in 4 (3%) patients. Right tibial and left tibial compressions were found in 7 (5%) and 3 (2%) of patients. entrapment at the ankle(s) and compression of the left peroneal at the knee(s) were observed each

in 2 (1.5%) cases. Meanwhile, 8% of left handed patients showed neurophysiologic findings of the right peroneal, left tibial nerves entrapments and through elbow entrapment of ulnar nerve (cubital tunnel syndrome). No features of compartment (entrapment) syndrome were found in right ulnar, left peroneal, right tibial and entrapment of peroneal nerve at the knees.

Table 2. The Findings of Nerve Conduction Studies of referred cases*

Nerve	Suspicion of C.T.S. in both hands		Suspicion of C.T.S. in one hands		Suspicion of C.T.S. in Lt hand only in Right handed patients N=14	Total No. of Lt handed patients N=12	Total No. of Rt handed patients N=138	Grand Total N=150
	Lt Handed Patients N=6	Rt Handed Patients N=72	Lt Handed Patients (Lt hand only) N=6	Rt Handed Patients (Rt hand only) N= 52				
	+ve No. (%)	+ve No. (%)	+ve No. (%)	+ve No. (%)	+ve No. (%)	+ve No. (%)	+ve No. (%)	+ve No. (%)
Rt Median	3 (50)	63 (88)	0	40 (77)	2 (14)	3 (25)	105 (76)	108 (72)
Lt Median	4 (67)	42 (58)	4 (67)	7 (13)	9 (64)	8 (67)	58 (42)	66 (44)
Rt Ulnar	0	12 (17)	0	5 (10)	0	0	17 (12)	17 (11)
Lt Ulnar	2 (33)	7 (10)	1 (17)	1 (2)	2 (14)	3 (25)	10 (7)	13 (9)
Rt Peroneal	1 (17)	3 (4)	0	0	1 (7)	1 (8)	4 (3)	5 (3)
Lt Peroneal	0	0	0	2 (4)	0	0	2 (1.5)	2 (1.3)
Rt Tibial	0	5 (7)	0	2 (4)	0	0	7 (5)	7 (4.7)
Lt Tibial	0	2 (3)	1 (17)	1 (2)	0	1 (8)	3 (2)	4 (2.6)
Delayed NCV of Ulnar N. Through the Elbow (Rt or Lt)	0	3 (4)	1 (17)	1 (2)	0	1 (8)	4 (3)	5 (3)
Delayed NCV of peroneal N. (Rt or Lt)	0	2 (3)	0	0	0	0	2 (1.5)	2 (1.3)

* N=150, +ve = Abnormal = indicates delayed motor or sensory distal latency and / or decreased CMAP and / or SNAP. Lt = left, Rt = right

The major presenting symptoms in positive cases are shown in table 3. Parasthesia and / or numbness in one or both hands were the commonest complaints and were present in 89 (74%) of patients. Pain in the wrist(s) and hand(s) was responsible for the presentation of slightly less than half (54 of total 120, 45%) number of positive cases. Meanwhile, unilateral or bilateral shoulder pain was the chief complaint in only 6 (5%) patients, 70 patients (58%) of the positive cases were suffering from symptoms other than symptoms of C.T.S. The commonest associated symptoms are knee(s) and ankle(s) pain.

Of the 99 positive females 79 were housewives (Table 4), 7% were teachers,

computer or clerical workers. The main occupations of males were building constructors (7 of total 21 cases, 33%), teachers and officers (24%), and drivers (14%). The other 14% were elderly and not working.

The periodicity of the symptoms is shown in table 5. The highest number of cases complained of night symptoms (35%). Daily ordinary work was the second leading cause of symptoms (25%). However, fine delicate works were responsible in 15% of positive cases. About 3% and 2% of patients respectively, assumed that manual milking and seasonal variations are the leading factor for their complaint.

Table 3. Major presenting symptoms of positive cases*

Chief complaint	No. (%)
Parasthesia and / or numbness of hands	89 (74)
pain in the wrist and/or fingers, palm	54 (45)
Weakness and /or stiffness, swollen, heaviness of hands	14 (12)
Elbow and forearm pain	11 (9)
Shoulder pain	6 (5)

70 patients (58%) had other associated symptoms

Associated symptoms	No. (%)
Knee(s) pain	27 (39)
Solar pain and numbness of feet	7 (10)
Ankle(s) pain	21 (30)
Low backache	13 (19)
Polyarthralgia	8 (11)
Hip(s) pain	2 (3)

** Some patients gave more than one complaint*

Table 4. Distribution of the positive cases by sex and occupation

Sex	Occupation	No. (%)
Female	Housewife (ordinary house works)	79 (80)
	Computing and writing	7 (7)
	Teacher	7 (7)
	Sewing	4 (4)
	Cleaning (offices)	2 (2)
	Total	99 (82.5)
Male	Construction workers	7 (33)
	Teachers and officers	5 (24)
	Drivers	3 (14)
	Farmers	2 (10)
	Cookers	1 (5)
	Aged (not working)	3 (14)
	Total	21 (17.5)
Grand Total		120 (100.0)

Table 5. Periodicity and triggering factor of symptoms among the positive cases

Periodicity and cause of symptoms	No. (%)
Nocturnal symptoms	42 (35)
Daily ordinary works	30 (25)
Fine works (typing, writing, sewing...etc.)	17 (14)
Continuous symptoms (throughout the day and night)	9 (7.5)
Pregnancy	7 (6)
Manual milking	4 (3.3)
During resting time	4 (3.3)
Driving	3 (2.5)
Seasonal (only during spring and summer)	2 (1.7)
Postoperative (following caesarian section and internal fixation in the forearm)	2 (1.7)
Grand Total	120 (100.0)

DISCUSSION

The total number of referred cases was 150, 120 of whom were found to be positive cases of C.T.S. either in one or both hands. Unlike other parts of the world,^{4,6,7} women in our locality are five times more prone to develop C.T.S. than

men. The early attendance of our patients to clinics (70% had symptoms for less than 2 years) might indicate that symptoms evoked by C.T.S. are very annoying and severe which make the patient obliged to seek for management.

Physiologically speaking, about 90-95% of the populations are dominantly

right handed.¹⁹ For that reason, C.T.S. more commonly affects the right hand. Moreover, right handed patients are more vulnerable to develop C.T.S. in both hands than left handed patients. Adding to this, right handed patients are more prone to develop C.T.S. in the opposite hand (42% have left C.T.S.) than left handed patients (25% have C.T.S. of the right hand). Therefore, complaining of symptoms in one hand will greatly raise the suspicion in the other hand. Also, evidence of entrapment of other peripheral nerves is more observed in right handed patients, mostly affecting both the ulnar and the right tibial nerves. Regarding left handed patients entrapment neuropathies were not reported in the right ulnar nerve, right tibial nerve and some other nerves.

Three quarters and one half of the patients, respectively, were complaining of parasthesia (and/or numbness) and pain in the wrist (and/or fingers and palm), which indicates that the use of hands accentuates the symptoms particularly at and below the site of entrapment. Other associated symptoms were found in 58% of patients indicating that predisposing factors for C.T.S. are stressful and rendering our patients to report multiple complaints.

Although detailed job information was not obtained in the present study, a number of occupational factors have been suggested to be potentially etiologic for symptoms of C.T.S. including repetitive / prolonged intensive hand activities, forceful exertion, awkward and static posture, and localized mechanical stress.²⁰ Most of these factors are probably responsible for presentation of our patients

particularly housewives and construction workers.

Because of increasing number of people using computer and communication technology, we can observe that a good number of referred cases particularly among females (7%), got symptoms related to spending many hours in this field. This has been also observed in other communities,²¹ 30% of frequent computer users complain of hand parasthesia and 10% meet clinical criteria for C.T.S.²⁰

It has been reported that symptoms usually worsen at night and can awaken the patient,^{12,13,22,23} which was clearly observed among positive cases in the present study. Interestingly, four female patients (3.3%) were getting symptoms only during manual milking. Two cases showed seasonal variation (spring and summer symptoms). These are seasons when people in our locality particularly workers and farmers spend most of their time of working in the fields.

These findings conclude that major peripheral nerves are subjected to compression at certain points along their course. Mononeuropathies due to nerve entrapment may give rise to symptoms which initially cause only minor distress, and for which the diagnosis may be vague. Adding to that, many occupational factors may affect more than one peripheral nerve, and may render them particularly vulnerable to compression or trauma. Therefore, it is important, whenever nerve conduction studies are carried out on suspected cases of compression neuropathy, to study nerves other than those which appear to be clinically

affected. Moreover, electrodiagnostic studies are the most objective tests to demonstrate different nerves entrapment in symptomatic patients.

REFERENCES

1. Dehaan M, Wilson R. Diagnosis and management of carpal tunnel syndrome. *J Musculoskel Med* 1989;6:47-60.
2. Zimmerman GR. Carpal tunnel syndrome. *J Athl Train* 1994; 29(1): 22-30.
3. Anthony JV. Management of carpal tunnel syndrome. *Am Fam Physician* 2003;68(2):255-72.
4. Goodyear-Smith F, Aroll B. What can family physicians offer patients with carpal tunnel syndrome other than surgery? A systematic Review or Nonsurgical Management. *Ann Fam Med* 2004;2(3):267-72.
5. Tortora JG, Derrickson B. Principles of anatomy and physiology. 11th ed. New York: John Wiley & Sons; 2006.
6. Atroshi I, Gummesson C, Johnson R, Ornstein E, Ranstam J, Rosen I. Prevalence of carpal tunnel syndrome in general population. *JAMA* 1999;282:153-8.
7. National Women's Health Information Center, U.S. Department of Health and Human Services, Office on Women's Health. Frequently asked questions. [online]. 2005 [cited 14 Apr 2006]. p. 1-5. Available from: URL: [http:// www.womenshealth.gov](http://www.womenshealth.gov)
8. Bernard BP. Musculoskeletal disorders and workplace factors: a critical review of epidemiologic evidence for work-related musculoskeletal disorders of the neck, upper extremity and low back. Cincinnati: National Institute for Occupational Safety and Health; 1997.
9. Katz JN, Simmons Bp. Carpal tunnel syndrome. *N Engl J Med* 2002;346(23):1807-12.
10. Solomon DH, Katz JN, Bahn R, Mogun H, Avorn J. Non occupational risk factors for carpal tunnel syndrome. *J Gen Intern Med* 1999;14:310-4.
11. Stevens JC, Beard CM, O'Fallon WM, Kurland LT. Conditions associated with carpal tunnel syndrome. *Mayo Clin Proc* 1992;67:541-8.
12. Stevens JC, Witt JC, Smith BE, Weaver AL, Bosch EP, Deen HG, et al. Symptoms of 100 patients with electromyographically verified carpal tunnel syndrome. *Muscle Nerve* 1999;22:1448-56.
13. Hephher L, Bentley R. Carpal Tunnel Syndrome. [online]. 2006 [cited 29 Apr 2006];1-3. Available form: URL: [http:// www.addenbrookes.org.uk](http://www.addenbrookes.org.uk)
14. Von Schroeder HP, Botte MJ. Carpal Tunnel Syndrome. *Hand Clin* 1996;12:643-55.
15. Rempel D, Evanoff B, Amadio PC. Consensus Criteria for the classification of carpal tunnel syndrome in epidemiologic studies. *Am J Public Health* 1998;88:1447-51.
16. Lenman JAR, Ritchie AE. Clinical electromyography. 1st ed. Great Britain: Pitman Medical and Scientific

- Publishing Co LTD; 1970.
17. Brown WF, Bolton FC. Clinical electromyography. 2nd ed. United Kingdom: Butterworth-Heinemann; 1993.
 18. Bajalan AAA. Clinical neurophysiological manual of the department of neurophysiology. London: Hull Royal Infirmary Teaching Hospital; 2000.
 19. Guyton AC, Hall JE. Textbook of Medical Physiology. 9th ed. Philadelphia: W.B. Saunders company; 1996.
 20. Keyserling WM. Occupational ergonomics: promoting safety and health through work-design. 3rd ed. Boston: Little Brown; 1994.
 21. Kim JY, Kim JI, Son JE, and Yun SK. Prevalence of carpal tunnel syndrome in meat and fish processing plants. J Occup Health 2004;46:230-4.
 22. Stevens JC, Witt JC, Smith BE, Weaver AL. The frequency of carpal tunnel syndrome in computer users at a medical facility. Neurology 2001;56:1568-70.
 23. Vinkin P, Bruyn G. Handbook of Neurology. Vol 8. New York (NY): Elsevier; 1970.

پوخته

زیده‌بوونا فشاری لسه‌ر ده‌مارا نیفا ده‌ستی و دیتنا فشاری لسه‌ر ده‌مارین دی یین ده‌ست وپییا

مه‌ره‌م : ئەو ئەو کۆلینە هاتە کەرن ژبو هەلەسەنگاندنا گۆهرینین فیسوئەلکتریکێ ل وان نەخوشی زیده‌بوونا فشاری لسه‌ر ده‌مارا نیفا ده‌ستی وه‌ه‌ر وه‌سا دیتنا فشاری لسه‌ر ده‌مارین دی یین ده‌ست وپییا .

نەخوشی وشیووی کاری: (150) نەخوش (122 مێ و 28 نیتر) ئەوین ب تاقیکرنا کلینکی دیاربوون کو زیده‌بوونا فشاری لسه‌ر ده‌مارا نیفا ده‌ستی هەین هاتنه‌ تومارکەرن بو ئی ئە کۆلینێ ، هەر نەخوشه‌کی فورمه‌کا پیزانینان بو هاتنه‌ پرکەرن وپاش نەخشه‌ کیشانا ده‌مارا نیفا ده‌ستی وه‌ه‌روه‌سا ده‌مارین دن یین ده‌ست وپییا بو هاتنه‌ جیکەرن لیدیف مه‌رجین انسیتوتا شاهی یابریتانی ل هەل ، و بکارئینانا ئامیری پروگرامکری یی نەخشه‌ کیشانا ده‌مارا و ماسولکا .

ئەنجام: نزیک (80 %) ژ نەخوشین هاتینه‌ ره‌وانه‌ کەرن ژبی وان دنایه‌را (21 – 50) سالان بوو و (70 %) ژ وان گازنده‌ هەبوون بو ماوی کبتر ژ دوو سالان ، 120 (80 %) ژ پرانی نەخوشان فشارا لسه‌ر ده‌مارا نیفا ده‌ستی یا پوزیتیف بو ، 108 (72 %) ژ وان ل ده‌ستی راستی و 44 % ل ده‌ستی چه‌پی پترین زیده‌بوونا فشاری لسه‌ر ده‌مارین دی ئەو بوو یا لسه‌ر ده‌مارا (Ulnar) ، وه‌می نەخوشین ئەو چه‌نده‌ لده‌فه‌ی ل ره‌خی راست ئەو ده‌ست راست بوون و (7 %) ژ نەخوشین ده‌ست چه‌پ ئەو چه‌نده‌ هەبوو ل ره‌خی راستی . زیده‌بوونا فشاری لسه‌ر ده‌مارین دن یین ده‌ست وپییا هاتنه‌ تومارکەرن و بریژین جودا – جودا بو هەر ده‌ماره‌کی . گەزاندن و ته‌زته‌زینک ئەو گازندا پتر به‌ر به‌لا‌ف بوو لده‌فه‌ نەخوشان و (58 %) ژ نەخوشین پوزیتیف گازندین دن هەبوون زیده‌باری گازندین ده‌ستان ، (79) ژ کوما (99) ژنان ئەقین پوزیتیف کابانین مالان بوون و (33 %) ژ زه‌لامین پوزیتیف پاله‌بوون .

به‌ره‌مه‌م: ده‌مارین ده‌ست وپییا یین سه‌ره‌کی ل به‌رده‌ستن بو فشاری لسه‌ر وان ل هنده‌ک جهین دیار ده‌می بوورینا وان هنده‌ک کار وشولین جوهره‌ وجور ئاکامی لسه‌ر ده‌ماران دکه‌ن و فشاری لسه‌ر زیده‌دکه‌ن . ده‌می نەخشه‌ کیشانا ده‌مارا بو نەخوشین زیده‌بوونا فشاری لسه‌ر ده‌مارا نیفا ده‌ستی تپته‌ کەرن ، یا پیتفی یه‌ بو نەخشه‌ کیشان بو ده‌مارین دن یین ده‌ست وپییا بیتنه‌ نه‌جامدان .

CEREBRAL PALSY AMONG IRAQI CHILDREN: CASE-SERIES STUDY

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ABSTRACT

Background Cerebral palsy is defined as any non-progressive central motor deficit dating to events in the prenatal or perinatal periods. It is one of the most common crippling conditions of childhood, and is not a specific disease but a group of disorders of varied causes.

There is little epidemiological data from developing countries; the prevalence is around 2 / 1000 live births in most developed countries.

Objectives To describe the epidemiologic distribution of the cerebral palsy cases, its different types and associated abnormalities with determination of common conditions reported with those cases.

Patients and Methods This is a case-series descriptive study conducted in the Specialized Center of Medical Rehabilitation in Baghdad (Sadir Al Kanat) on 60 children with cerebral palsy who were attending that center from June 2004 - May 2005 inclusive.

Results The present study revealed that the male: female ratio was 1.5:1. Prolonged & vaginal labor were the most frequently reported perinatal conditions (53.4% & 66.6% respectively); while neonatal jaundice and/or kernicterus with cyanosis and/or asphyxia were the main postnatal reported conditions (46.6% and 31.7% respectively). Spastic cerebral palsy was found to be the most common type (76.7%). Hearing, visual and dental problems were the most frequent associated complications (65%, 56.7%, and 53.3% respectively). There was higher proportion of cerebral palsy in children from consanguine parents (58.3%).

Conclusions and Recommendations All the associated perinatal and postnatal conditions suggest that good prenatal, natal and early neonatal care can prevent the occurrence of a high proportion of cerebral palsy cases. There is a need for enhancing health education directed both at mothers and at the general public with promotion of maternal child health services in Iraq.

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Key words: Cerebral palsy, Iraqi children

Cerebral palsy (CP) first described by Dr. Little in 1826 and it was known for many years by his name. It is defined as any non-progressive central motor deficit dating to events in the prenatal or

perinatal periods. It is one of the most common crippling conditions of childhood, and is not a specific disease but a group of disorders of varied causes.¹

The child with CP and damage to the motor mechanisms would be expected to have damage to the other parts of the brain as well. This may be manifested as epilepsy, specific learning problems, organic behavioral disturbances, language deficit, and intellectual, visual and hearing

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impairment.²⁻⁴ CP consists of a wide range of life time physical disabilities resulting in a varying degree of functional deficit.^{5,6} It is difficult to find two CP children who are alike for the impairment in their brain development because it can take so many different forms.⁷ CP is neither contagious nor is usually inherited from one generation to the next. At this time it cannot be cured although a scientific research continues to yield improved treatment and methods of prevention.⁸

The clinical manifestation of CP often differ according to gestational age at birth, the chronological age, the distribution of lesions and the underlying diseases.⁹ The subtypes of CP are spastic, extra pyramidal and mixed.¹⁰

There is little epidemiological data from developing countries and the prevalence is around 2 / 1000 live births in most developed countries. In recent years the neonatal intensive care management has increased the survival rate of low birth weight babies resulting in an overall increase in incidence of CP in both term and preterm infants.^{1,11}

Cerebral palsy is the most common cause of physical disability in childhood, occurring in 2.0 to 2.5 per 1000 live births and it is the most common cause of motor impairment in children of Australian and European societies.¹⁰⁻¹⁵

The present study aims at describing the epidemiologic distribution of the CP cases attending the Specialized Center of Medical Rehabilitation in Baghdad (SCMR), to specify its different types, and to determine common conditions reported in the patients' history.

PATIENTS AND METHODS

A case-series, descriptive study was conducted on 60 patients with CP, attending the Specialized Center of Medical Rehabilitation in Baghdad, Iraq (SCMR) for the period from 1st June 2004 to 31st May 2005. The center was established during the 1980's to receive referred cases with physical disabilities from different public and private health facilities in Baghdad and surrounding governorates. A questionnaire form was designed to interview parents or the patients' companions for all CP cases attending the center, by direct interview. The questionnaire included information on age, sex, address, type of delivery, parent's consanguinity and details about postnatal history (for any history of neonatal jaundice, cyanosis, fever or diarrhea).

Weighting with neurological examination of all cases was done to diagnose and specify types of CP, with examination of eye and visual acuity, hearing status and dental conditions under the supervision of the specialists in the center.

Analysis of the data was done by using ratios, frequencies and percentages to describe the distribution of the cases.

RESULTS

The distribution of CP cases by their gender reveals that the male to female ratio was (1.5:1) i.e. 60% male and 40% female. Table 1 shows the distribution of the cases by their types of delivery. It indicates that prolonged and vaginal labor was the

commonest type (53.4% and 66.6% respectively).

Figure 1 shows the distribution of cases by their types; it is clear that spastic type is the most common type (76.7%) and figure 2 shows that the common reported conditions, jaundice and/or kernicterus with neonatal asphyxi and/or cyanosis were frequent postnatal conditions reported in the history of CP cases (46.6% and 31.7% respectively). Figure 3 reveals

the distribution of the cases by their associated complications or problems. It shows that hearing problems are among the commonest problems followed by dental and visual problems (65%, 56.7%, and 53.3% respectively). Figure 4 illustrates the distribution of cases by their parent's consanguinity showing that there are a high proportion of consanguine parents (58.3%) among the group studied.

Table 1. Distribution of the CP cases by their types of delivery

Prolonged Labor	Types of Delivery		Total No. (%)
	Vaginal No. (%)	Cesarean Section No. (%)	
Yes	20 (33.3)	12 (20.1)	32 (53.4)
No	20 (33.3)	8 (13.3)	28 (46.6)
Total	40 (66.6)	20 (33.4)	60 (100)

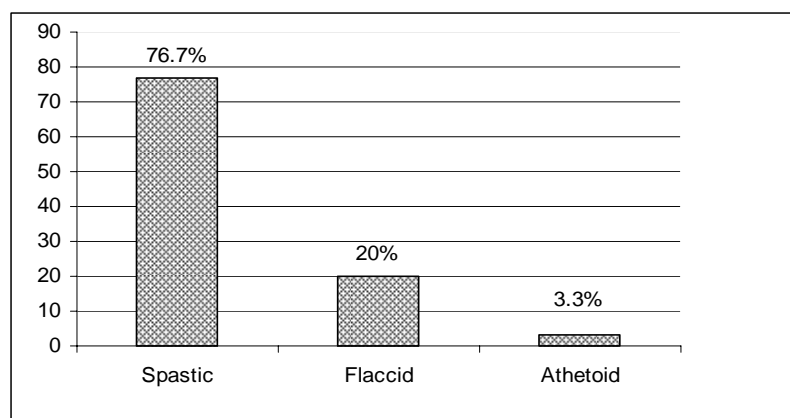


Figure 1. Percentage distribution of CP cases by their types

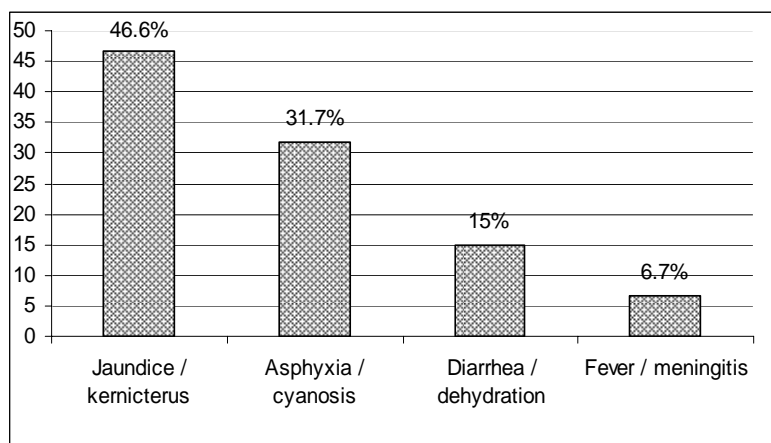


Figure 2. Percentage distribution of CP cases by common conditions

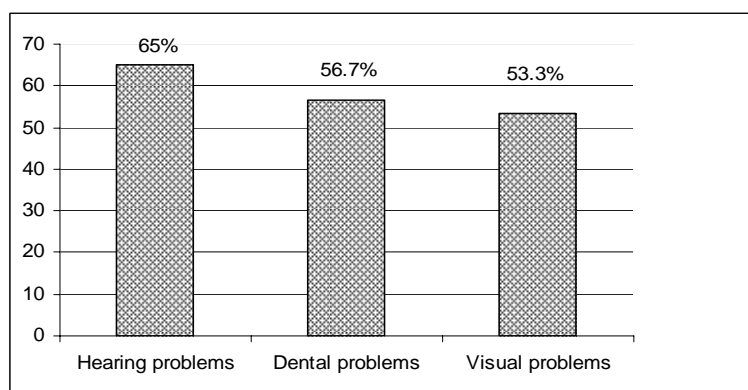


Figure 3. Percentage distribution of CP cases by their associated disorders

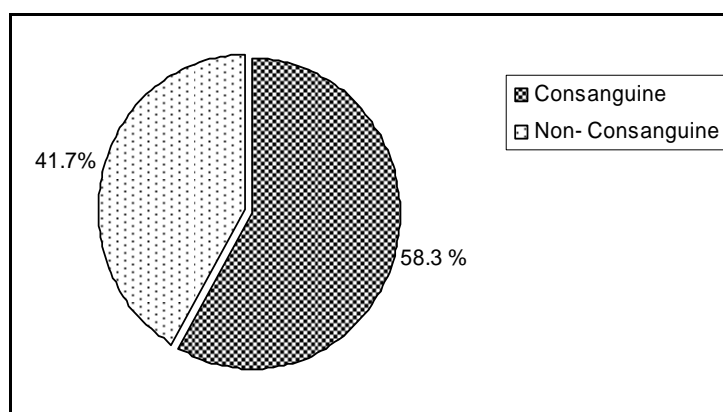


Figure 4. Distribution of CP cases by their parental consanguinity

DISCUSSION

The rate of cerebral palsy was found to be more in the male than in the female,

against what was found in many literatures which stated the relatively same rate or same ratio in both sex.^{2,13,14} This result may reflect the special and intensive care

provided by the Iraqi families towards the male child. In this study the commonest type of delivery was the prolonged and vaginal labor, which may had an effect on the future neonatal growth and development, which is similar to what was studied by other authorities.¹⁶⁻¹⁹ Spastic type of CP was the most common type. This is similar to what was reported in other papers.^{20,21} Neonatal jaundice and/or kernicterus with asphyxia and/or cyanosis were found to be the commonest among other postnatal conditions reported with CP. These conditions may affect neurological growth of the newborns, and this is same to what was mentioned in several other studies.²²⁻²⁵

Hearing, dental and visual problems were the most common complications that were associated with CP, which may be due to neurological damage or complications of CP. This is similar to what was published by Al-Naddawi et al in Baghdad and also by other authors.²⁶⁻²⁹ There was increased proportion of CP in children of consanguineous parents, which may explained the genetic or familiar predisposition, as what was studied by Terzidon and Bennett in 2001.³⁰

CONCLUSIONS AND RECOMMENDATIONS

Male patients were found to be more affected than female. Prolonged and vaginal labor was the commonest type of delivery. Spastic type of cerebral palsy was noticed to be the most common type. Neonatal jaundice and/or kernicterus with neonatal asphyxia and/or cyanosis were

commonest among other reported conditions. Hearing problems were noticed to be the most common associated abnormalities followed by dental and visual abnormalities. There was an increased proportion of cerebral palsy in children born to consanguineous parents. So there is a need for enhancing maternal and public health education with promotion of maternal child health services as important measures for prevention and control of cerebral palsy problem.

REFERENCES

1. Haslam RHA. Cerebral palsy. In: Behrman RE, Kliegman RM, Jenson HB, editors. Nelson Textbook of Pediatrics. 16th ed. Philadelphia: W.B. Saunders Company; 2000. p. 1843-5.
2. Campbell A, Macintosh N. Cerebral palsy. Textbook of Pediatrics .5th ed. New York: Churchill Livingstone; 1998.
3. Al-Suliman AA. Epilepsy in Saudi children with cerebral palsy. Saudi Med J 2001;22(1):19-21.
4. Senbil N, Sonel B, Ayden OF, GurerYKY. Epileptic and non epileptic cerebral palsy: EEG and cranial imaging findings. Brain Dev 2002;3:166.
5. Perry JE, Davis BL, Lucuano. Quantifying muscle activity in non-ambulatory children with spastic cerebral palsy before and after selective dorsal rhizotomy. J Electromyogr Kinesiol 2001;11(1):31-

- 7.
6. Blickstein I. Cerebral palsy in multifetal pregnancies. *Dev Med Child Neurol* 2002;44:531-3.
7. Al-Janabi NM. Impacts of factors upon the level of functioning of children with cerebral palsy [PhD dissertation]. Nursing College: Baghdad Univ.; 2002.
8. Al-Fahad S. Cerebral palsy introduction. The abstract book of the scientific committee of Iraqi Medical Association 2002;1-2.
9. Al-Zaidi MA. Forms of cerebral palsy. The abstract book of the scientific committee of Iraqi Medical Association 2002;3-6.
10. Redihough DS, Collins KJ. The epidemiology and causes of cerebral palsy. *Aust J Physiother* 2003;49(1):7-12.
11. Bhatia M, Joseph B. Rehabilitation of cerebral palsy in developing countries: The need for comprehensive assessment [Abstract]. *Pediatr Rehabil* 2000;4(2):83.
12. SCPE [Surveillance of Cerebral Palsy in Europe]. *Dev Med Child Neurol* 2000;42(12):816.
13. Haslam RHA. Cerebral palsy. In: Behrman RE, Kliegman RM, Jenson HB, editors. *Nelson Textbook of Pediatrics*. 17th ed. Philadelphia: Saunders; 2004. p. 2024-25.
14. Liptak GS, Donnel MO, Conaway M. Health status of children with moderate to severe cerebral palsy. *Dev Med Child Neurol* 2001;43:364-70.
15. William HB. Prevention of disability. In: Maxcy, Rosenau, last, editors. *Public Health & Preventive Medicine*. 14th ed. Connecticut: Appleton & Lange Stamford;1998. p. 1059-64.
16. Rose J, Wolf DR, Jones VK, Bloch DA, Oehlert JW, Gamble JG. Postural balance in children with cerebral palsy. *Dev Med Child Neurol* 2002;44:58-63.
17. Keith LG, Oleszczuk JJ, Keith DM. Multiple gestation: reflection on epidemiology: causes and consequences. *Int J Fertil Women Med* 2000;45(3):206-14.
18. Al-Naddawi MN. Causes of cerebral palsy. The abstract book of scientific committee of Iraqi Medical Association 2002;7-10.
19. Al-Omeri W. Cerebral palsy an almost cleared old obstetricians stigma. The abstract book of the scientific committee of Iraqi Medical Association 2002;11-3.
20. Engsberg J, Ross S, Olree KS, Park TS. Ankle spasticity and strength in children with spastic diplegia. *Dev Med Child Neurol* 2000;42:42-7.
21. Einspieler C, Cioni GG, Paolicelli PB. The early markers for later dyskinetic cerebral palsy are different from those for spastic cerebral palsy. *Neuropediatrics* 2002;33(2):73-8.
22. Asakura H, Kawabata K, Tani A, Satoh M. Perinatal risk factors to neurological outcomes of term newborns with asphyxia at birth : a prospective study . *J Obst Gynecol Res* 2000;26(5):324.

23. Lyon. Chronic lung disease of prematurity. The role of intrauterine infection. *Eur J Pediatr* 2000;159(11):798-802.
24. Psyrrer ER, Yemans ER. Does asphyxia cause cerebral palsy? [Abstract]. *Semin perinatol J* 2000;24(3):215-20.
25. Johnston MV, Tresher WH, Ishida A, Nakajima W. Neurobiology of hypoxic-ischemic injury in the developing brain. *Ped Res J* 2001;49(6):735-41.
26. Al-Naddawi MN, Al-Kanani S, Ayis G. Visual disorders associated with cerebral palsy. *J Fac Med Baghdad* 1989;31(3):277-84.
27. Ottenbacher K. Functional assessment and care of children with neurodevelopment disabilities. *Am J Phys Med Rehabil* 2000;79(2):114-23.
28. Jan J, Lyons CJ, Heaven RK, Matsuba C. Visual impairment due to a dyskinetic eye movement disorder in children with dyskinetic cerebral palsy. *Dev Med Child Neurol* 2001;43:108–12.
29. Krageloh, M, Ingeborg, Helber, Alexandra, Mader, Linda, Bilateral lesions of thalamus and basal ganglia: origin and outcome. *Dev Med Child Neurol* 2002;44(7):477-84.
30. Terzidou V, Bennett P. Maternal risk factors for fetal and neonatal brain damage. *Biol Neonate* 2001;79(3-4):157-62.

پوخته

وهسفا فالنجا مېشكى ل زاروكين عيراقى (فه كولينه كا زنجيره كا حاله تا)

پېشه كى: فالنجا مېشكى دهېته پېناسه كرن وهك قوسيريه كا ناوهندى ل مهزى يا نه وه رار كرى كو نه گهرېن وى زفريته بو ده مې پېش زاروك بوونى يان ده ورو بهرېن وى ، و دهېته هژمارتن كو ئېك ژ گرنكترين نه گهرېن په ككه فتنى ل زاروكا، و ناهېته هژمارتن وهك نه ساخيه كا تا بهت بهلكو ژ كومه كا تيكچونا ژ نه گهرېن جوړاوجوړ .

كىم پېزانين يېن پهزى ههين ژ دهوله تېن گه شنده. رېژه هه ر دمىيت 2 ژ هه ر 1000 ل زاروكين تازه بووين ل پرانيا دهوله تېن پېشكه فتى.

نارمانج: بو ديار كونا به لافه كونا پهزى يېن حاله تېن فالنجا مېشكى و بو ديار كونا جوړېن وى و ديار كونا گرفتېن دى د وان حاله تا.

رېكېن فه كولېنى: نه ف فه كولينا وهسفى يا زنجيره كا حاله تا هاته كرن ل سهنه رى سهدر ولكه نات يى شاياندا ن پزېشكى ل به غدا. نمونه پېكهاتى بوو ژ 60 زاروك يېن توشى فالنجا مېشكى يېن كو سه ره دانا بنگه هى كرين ژ خزي رانا 2004 تا گولانا 2005.

نه نجام: هاته ديار كرن ل فه كولېنى دا كو رېژه يا كور: كچ (1.5 : 1) بوو. زاروك بوونا ب زه حمهت و زاروك بوونا سروشتى گرنكترين فاكته رى مه ترسيى بو ل ده مې زاروك بوونى (53.4٪، 66.6٪ ل دويف ئېك) و لدويف دا زهركا بچويك/يان پويچ بوونا مهزى ژ نه گهرى زهركا بلند (46.6٪)، و شېنبوون/يان خه نده قين ل پشتى زاروك بوونى (31.7٪). جوړى گرژى ژ هه مې جوړين فالنجا مېشكى پتربوو (76.7٪).

گرفتېن چافى، ددانا، و گولې بوونى هاته ديار كرن كو گرفتېن گرنكن دگه ل فالنجا مېشكى (65٪، 56.7٪، 53.3٪ ل دويف ئېك). گه لك ژ حاله تېن فالنجا مېشكى هه بوون ل زاروكين كو دهيك و بايېن وان خزمين ئېك بوون (58.3٪).

دهر نه نجام و راسپارد: هه مې فاكته رېن بهرى زاروك بوونى وى هندى رادگه هيېن كو چاقديريا باش بو دهيك و زاروكا ناهيليت فالنجا مېشكى پيدايت. هه روه سا پيويسى يه كا زور دقيت ژ بو به زكرنا ناستى رهوشنير كونا ساخله مې بو دهيك و كومه لكه هى و هه روه سا پېشته فانى و پېشېخستنا خزمه تېن دهيكينى و زاروكا وهك ريك بو خو پاراستن و كونترول كونا فالنجا مېشكى.

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ACUTE HEAD TRAUMA - CT SCANNING STUDY

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Submitted 10 February 2007; accepted 16 August 2007

ABSTRACT

Background Head trauma now represents a major cause of death and disability among young otherwise healthy people, and it is claimed that it is more common than ever.

Radiographs are now replaced by CT scanning as the primary method of assessing head trauma, but MRI might be needed too.

Aim It is to evaluate the role of CT scanning in cases of acute head trauma of different severity.

Patients A retrospective study has been carried out of 50 cases of acute head trauma with positive CT scanning referred to Azadi Teaching Hospital in Duhok city from March 2003 through December 2005.

Results All the intracranial sequelae of acute head trauma are documented and analyzed, where the males are found to be involved in 78% of the cases, and in up to 60% of the cases the victims are in the first three decades of life, RTA-the main cause shows a dramatic increase compared with other studies.

Fractures are present in the majority of the cases indicating a significant trauma, moreover, more than one sequelae can be detected in many cases.

A localized brain oedema, contusions, and intracerebral hematomas are the most commonly found sequelae, while other sequelae such as subarachnoid hematomas, subdural hematomas, and epidural hematomas are encountered less. Diffuse brain swelling as has been declared in other studies is more common among children.

Conclusion CT scanning remains the first diagnostic imaging tool to detect the different intracranial post-traumatic lesions of acute head trauma, many of which are life-threatening, on the other hand, MRI is spared for full assessment of head trauma, and for follow up too.

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Key words: Acute head trauma, CT scanning study, Hematoma

Trauma is the most common cause of death and permanent disability in the early decades of life with neurological trauma causing the majority.¹ Cranial trauma is responsible for 150 000 hospital

admissions per year in the UK, while in United States 500 000 new cases of head injury occur every year,¹ and it has been claimed that head trauma is more common than ever.

With the ubiquitous availability of CT scanning since the early 1970s the diagnosis and management of head trauma had been changed significantly,²⁻⁴ where CT scanning is considered even less cost effective than admission.^{5,6} This study aims to evaluate the role of CT scanning in acute head trauma, showing various

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intracranial sequelae, their frequencies correlated with age, sex, and the presence of a skull fracture.

MATERIAL AND METHODS

During a 34-month period, from March 2003 through December 2005, 50 cases of acute head trauma with positive CT scan findings are studied retrospectively.

The ages are ranging from 1-69 years (mean is 27.1 years, median is 24.1 years, standard deviation is 19.70). In each case, the age, and sex of the patient, type of trauma, any associated fracture, and CT sequelae with their percentages are all documented.

RESULTS

Out of 50 cases included in the study, 39 cases (78%) are males, while female cases are only 11 cases constituting 22%.

The age distribution is clarified in figure 1.

An associated fracture is documented in 41 cases (82%) of the cases; the non-fractured cases are only 9, constituting 18% .

Regarding the etiologies, road traffic accidents (RTA) is the cause of injury in 86% of the cases, while fall from heights (FFH), and bullet injuries constitute 8%, and 6% of the etiology, respectively .

The incidence of different intracranial sequelae is shown in figure 2, where a localized brain edema is shown in 29 cases, the other sequelae: i.e.: an intracerebral hematoma in 23 cases, a brain contusion in 22 cases, a subdural

hematoma in 19 cases, a diffuse brain swelling in 15 cases, a subarachnoid, epidural, and an intraventricular hematoma in 9, 8 and 5 cases respectively .

Other less common sequelae, such as acute subdural effusion, and pneumatocephalus are encountered less frequently, although not shown in the histogram.

DISCUSSION

It has been noticed in patients with multiple injuries that head is the most commonly injured part, and in fatal RTAs, injury to the brain is found in 75% of victims at autopsy, where most of the serious head traumas occur in people under age of 30 years of age.⁷

A male predominance is obvious, where males constitute 78%, (39 case), and the female victims constitute 22 % (only 11 cases).

The male to female ratio is almost 4:1, compared with other series showing 3:1 and 3:1.5 respectively,⁸ where males are seen to be involved more in all age groups.

This discrepancy reflects more involvement of males in the social, and economic life, in spite of the female predominance observed in our community.

This study elicits that the highest incidence of cases is in the first decade, constituting 30%, while the fifth, sixth, and seventh decades collectively form only 28%, whereas, more than half of the total cases, a 60%, occur in the first three decades of life (Figure 1), this finding is similar to a study done in 1989, where the children and young adults were the main

victims of acute head trauma in 61% of cases.⁷

Generally, in this study, there is a decrease in the incidence of head trauma with increasing age, except for the second decade. In other two separate studies, pediatric age group constituted 53% and 39% respectively.^{7,8}

This series shows that more than two thirds of the cases are due to road traffic accidents (RTA) constituting 86% of all the cases, whereas, fall from height (FFH), and bullet injuries constitute 8% and 6% respectively. The high incidence of RTAs reflects a high contribution to the etiology which is in deed an almost double of that figure found in a previous study a 46 %.⁷

Many authors have noted that in children RTA, and FFH are the main causes in acute head trauma, while in adults assaults are added too.⁴ However, there are authors who considered falls as the main cause of acute head trauma.^{7,8}

The steep raise in the incidence of acute head traumas is, probably, reflecting

an increasing number of vehicles on badly paved roads with ignorance of commitment to the traffic regulations, and laws.

There is a lot of debate concerning the significance of finding a fracture in the post-traumatic skull radiographs,⁹ however, no much stress is laid on this point since the majority of the cases were referred immediately for an urgent CT.

In this study 82% of the cases are associated with fractures, while, the non-fractured cases constitute 18% only, this high incidence might be explained on the basis of severity, and the selectivity of the cases .

A fracture on radiography indicates a significant force has applied to the bony vault. However, the lack of visualization of a fracture does not exclude a significant injury to the underlying brain; therefore, a skull fracture may or may not indicate a brain injury.¹⁰ The literature shows an incidence of 2%-42% of fractures in acute head trauma.⁷

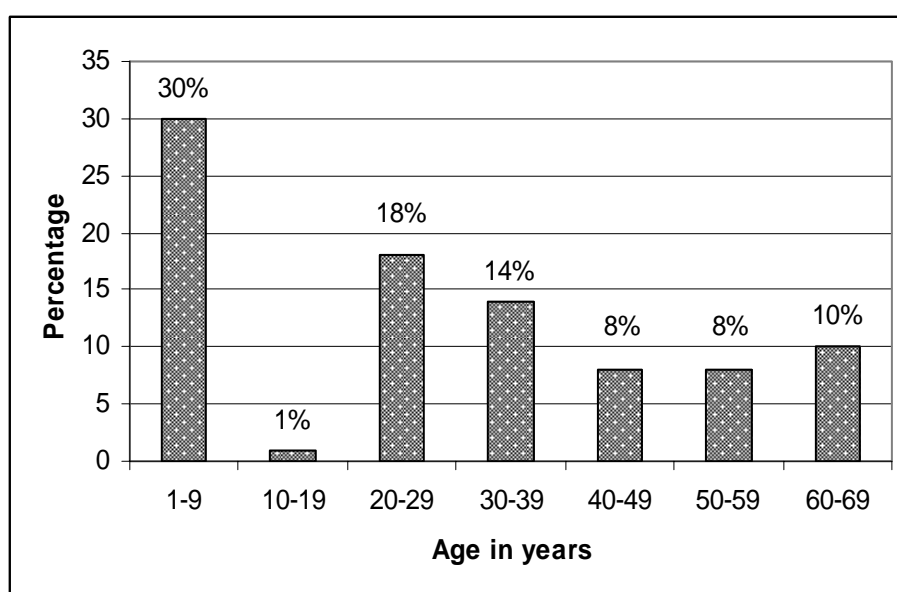


Figure 1. The incidence of acute head trauma in different age groups

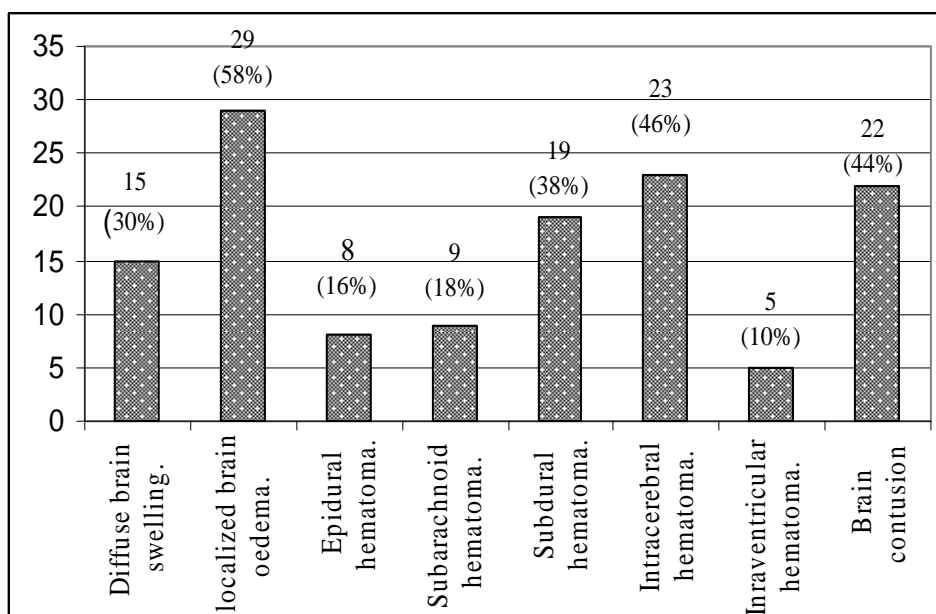


Figure 2. The incidence and number of different post-traumatic sequelae of acute head trauma

PATHOLOGICAL TYPES OF INTRACRANIAL SEQUELAE SEEN ON CT SCANNING

The neuroradiology of trauma has undergone a dramatic change since the advent of CT scanning, which is regarded now as the primary method of assessing head injury, supplemented by a lateral radiograph of cervical spine.⁴

In the early reports, it was established that generally there was a direct relationship between the severity of clinical presentation, and the demonstration of abnormalities,⁸ where all the cases in this series show one or more post-traumatic sequel, indicating a considerable severity, and it was estimated that up to 60% of this study showed two or even three intracranial sequelae, such as

an epidural, and an intracerebral hematomas, or a subarachnoid, a subdural, and an intracerebral hematomas all together.

The incidence of abnormal CT findings are variable in different series ranging from 37%, 73%, 86% to 98%.⁷

Diffuse Brain Swelling (DBS)

DBS (Figure 3) is due to loss of auto regulation resulting in an increase in blood flow and blood volume forcing CSF out of the ventricles and subarachnoid spaces, leading to compression of ventricles and cisterns with no significant change in white matter, though other authors claim some increase in density.⁴

DBS is observed more among pediatric cases following head trauma, and it is usually due to a rapid hyperaemia,

since children differ in mode of injury, flexibility due to presence of sutures and poor myelination, and in a rapid response to trauma in the form of vasodilatation and increase in cerebral blood flow.

DBS is documented more among pediatric age group, a 33% compared with 28% among the adults (Figure 2). Others had recorded incidence of 21% with a ratio of 3.5 times more than in adults, while a separate study showed 27% with a pediatric to adult incidence ratio of 2:1.⁷

Localized Brain Oedema (LBO)

LBO (Figure 4 and 5) appears as a poorly defined hypodense area with shift of the midline structures to the contralateral side which may be the only presenting feature.¹²

LBO is usually noted to accompany other sequelae, which might explain its high incidence, in this study 58% of cases show LBO (Figure 2). In another study the incidence was 41%.⁷

Brain Contusion and Intracerebral Hematoma (ICH)

Brain contusion (Figure 4) lesions are pathologically areas of haemorrhage, necrosis and oedema, appearing as multiple small scattered, usually ill-defined heterogeneously high densities interspersed with low density areas of oedema or normal density.⁴

ICH (Figure 5 and 10) appears as a well-defined circumscribed high density area surrounded by a low density area of oedema. Some authors believe that both contusions and ICH often coexist in the same case, and the incidence of ICH is

higher than estimated,⁷ were ICH is frequently associating other complications.

This study documented a high incidence of contusions, and ICH of 44% and 46%, respectively (Figure 2), where the previous reports declared low figures 1, this might reflect the severity, and the selectivity of the cases of head traumas included in this study. In a separate study the incidence of contusion and ICH were 13% and 25% respectively.⁷

A countercoup injury is regarded rare, while others claim the reverse,¹⁰ and it can be seen in the opposite side of the injury (Figure 6), and nowadays Holburn's force shear theory is accepted, to some, to explain a countercoup lesion on the basis of rotational acceleration leading to shear force strains.^{11,12}

In this study, a countercoup injury is found in 3% of the cases, while a previous study showed 0.7%.⁷

Acute Subdural Hematoma

ASD (Figure 3, 5 and 6) appears as a hyperdense extracerebral crescent-shaped blood collection with a convex lateral border and concave medial border, and a rarely a SDH can be isodense or even hypodenses, moreover, an acute SDH can occur in the posterosuperior aspect of interhemispheric fissure.^{8,13}

Acute SDH is found in 38% of this study (Figure 2), other studies have shown 19%, 9%, 12.6%, 16% and 59%.^{7,1,14-16}

A subdural hygroma is an extracerebral crescentic collection equal in density to CSF, and it is not possible to distinguish it from a chronic SDH on CT scanning, on the other hand it can be life-

threatening and may have the same clinical significance as a subdural hematoma.

Subdural hygromas are found in two cases of this series (4%) (Figure 2), while others had documented 5%, and 9.5%, respectively.^{7,15}

Epidural Hematoma

An EDH (Figure 7 and 8) appears as an extra-axial hyperdense biconvex lenticular collection, with a possible shift of ventricles, due a laceration to middle meningeal artery or disruption of dural venous sinuses. Bilateral EDH have been reported, and the majority are associated with fractures and laceration of adjacent dural vessels (Figure 7 and 8).

This study shows an incidence of 16% (Figure 2), while others had recorded 2%, 6-7%, 9% and 18.3%, respectively.^{7,14-16}

Subarachnoid Hematoma (SAH)

SAH (Figure 9) appears as a hyperdensity in the basal cisterns, Sylvian fissure, sulci, or in the interhemispheric fissure. A calcified falx in an elderly patient should not be misinterpreted as an SAH .

A SAH may coexist with any of other sequelae, but usually found isolated in basal cistern with a fractured base, whilst a SAH over the hemisphere may be related to a fracture, local hematoma or contusion.¹⁶

In this study the incidence of SAH is 18% (Figure 2); while other studies showed 37%, 2% and 7%, respectively.^{7,8,15}

Incidence of SAH increases with increasing severity of the head trauma; this

might explain the wide range of incidences in different series.⁹

Diffuse Axonal Injury

DAI was originally referred to as "shearing injury", and is common with severe head trauma. DAI appears as small lesions of 5-15 mm, usually multiple typically found near the gray-white matter junction, corpus callosum, dorsal lateral upper brain stem, or in the basal ganglia, however, others add brain swelling and intraventricular hematoma too. DAI is associated with a poor prognosis,^{1,11} and the mortality may reach to 50% of the cases .

DAI is thought to be due to a rotational acceleration of brain causing a sort of shear-strain deformity and hence indirect mechanical injury in a top-down pattern.¹⁸

MRI is far more sensitive than CT scanning in detecting DAI, and the latter modality can only detect the lesions which are greater than 15 mm, or when they are present in internal capsule or corona radiata.⁸

No DAI can be diagnosed confidently in this study, in spite of the severity of many cases, explained on the basis that CT often fails to detect DAI since most of the lesions are small and more than 70% are nonhemorrhagic.⁸ Two separate studies have shown incidences of 0.8% & 2.7% respectively.^{1,7}

Intraventricular Hematoma

IVH (Figure 9) is frequently associated with a parenchymal damage and it is an

ominous sign indicating a severe head trauma.³

Some previous studies recorded low incidences of 3% and 0.8%,⁷ however, more recent studies recorded high incidences of up to 35% of the cases,⁸ while this study shows an incidence of 10% , and when diffuse axonal injury of corpus callosum is encountered , then an associated IVH can be found in up to 60% of the cases.

Pneumocephalus and Hydrocephalus

A pnematocephalus can result from a basal fracture, mastoid bone fracture, or a fracture through a paranasal sinus (Figure 10).

This study shows an incidence of 16%, while other two studies showed 2.5%, and 0%, respectively.^{7,14}

Hydrocephalus implies obstruction within CSF pathway where it can be found proximal to the level of obstruction, due to an intraventricular clot, or a mass effect from oedema, hematoma or a brain contusion,² the clinical outcome is much worse , but the number of cases severe enough to require shunting is 5% (Figure 9).

This study shows an incidence of 16%, while the reported incidences are highly variable because of inconsistent definition of the entity.¹



Figure 3. A 26 years old female with a diffuse brain swelling, white cerebellar sign, and a left subdural hematoma



Figure 4. A 40 years old male with a left temporal contusion, a skull fracture, and a subdural hematoma

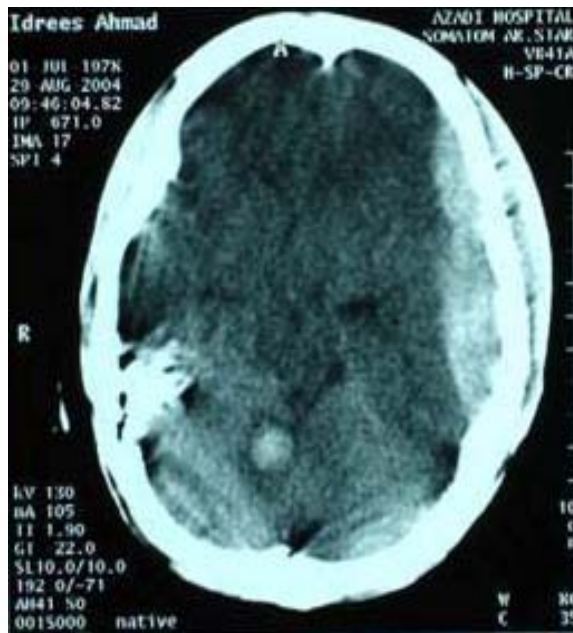


Figure 5. A 34 years old male with a left scalp hematoma, a skull fracture, a left subdural, and a right occipital intracerebral hematomas



Figure 6. An adult male involved in a RTA with a right subdural hematoma, skull fracture, and a left counter-coup contusion

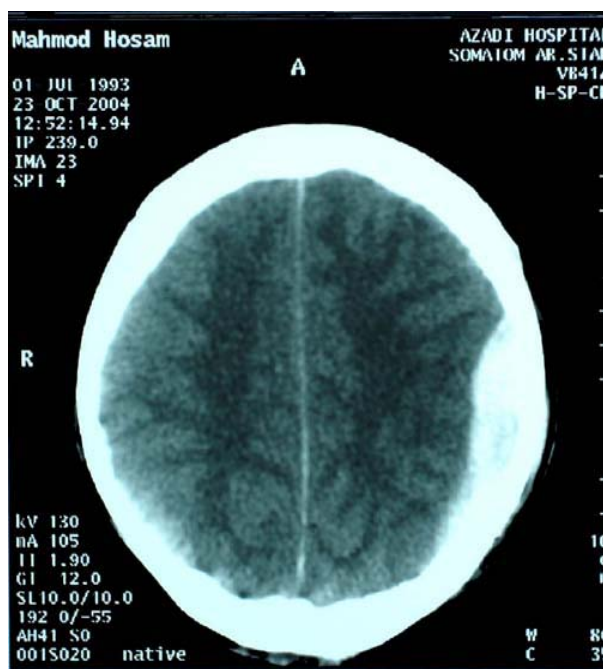


Figure 7. An 11 years old boy involved in a RTA, with a left parietal epidural hematoma



Figure 8. A 7 years old boy with a left temporal epidural hematoma, and a mass effect on the left lateral ventricle

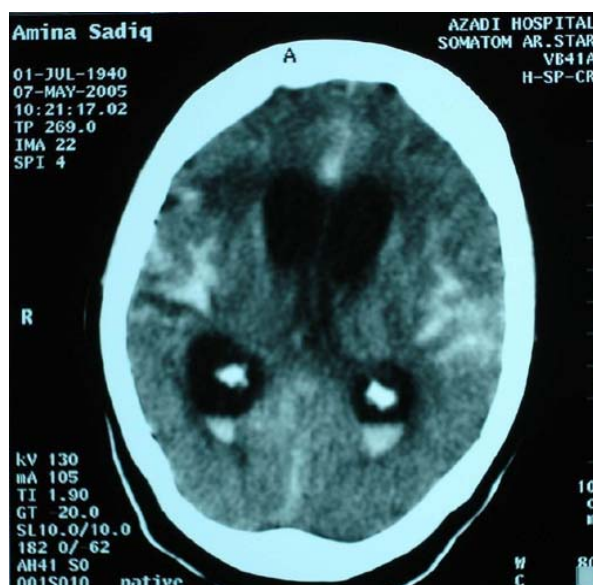


Figure 9. A 65 years old female presented with dilated lateral, and third ventricles, subarachnoid, and intraventricular hematomas

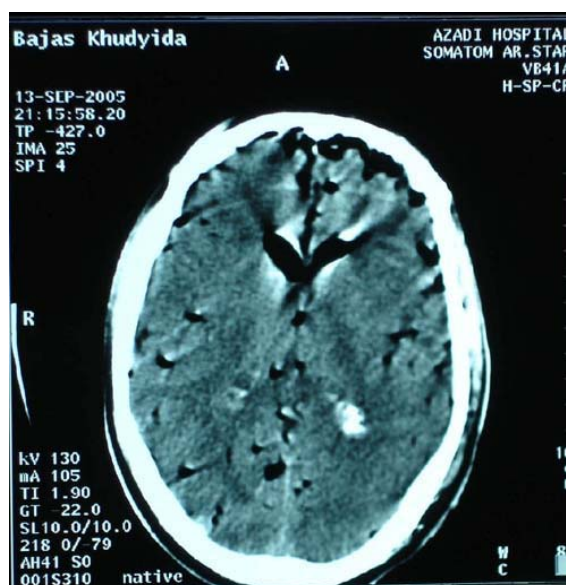


Figure 10. A 52 years old male with a skull fracture, intracerebral hematoma, extensive intracerebral, and intraventricular air

REFERENCES

1. Zimmerman RA, Bilaniuk LT, Genneralli T, Bruce D, Dolinskas C, Uzzell B. Cranial CT in diagnosis and management of acute head trauma. Am J Roentgenol 1978;131:27-34.
2. Sutton D. Textbook of radiology and imaging. 7th ed. Vol 2. China: Curchill Livingstone; 2003.
3. Hagga JR, Lanzieri CF, Gilkeson RC. CT and MRI imaging of the whole body. USA: Mosby; 2001.
4. Hydell MJ, Preston CA, Mills TJ, Lubner S, Blaudean E, Deblieux MC. Indications for computerized tomography in patients with minor head injury. N Engl J Med 2000;343: 100-5.
5. Norlund A, Marke LA, Geijerstam JL, Ordsson S, Britton M, OCTOPUS study investigators. Immediate CT scanning or admission after mild head injury: cost comparison in randomized controlled trial. BMJ 2006;333:469 .
6. Geijerstam JL, Ordsson S, Britton M, OCTOPUS study investigators. Medical outcome after immediate computerized tomography or admission for observation in patients with mild head injury: randomized controlled trial. BMJ 2006;333: 465.
7. Hidayat SKh. Acute head trauma, an evaluation by CT scanning and conventional radiology [DMRD dissertation]. College of Medicine: Mosul Univ.; 1989 .

8. Youmans JR. Neurological Surgery. Vol 4. Philadelphia: Saunders; 1982.
9. Khalili AH. The value of a skull XR in the early management of head injury. Postgrad Doct Middle East 1988;11(8):3891-3.
10. Quayle KS, Quayle KS, Jaffe DM, Kuppermann N, Kaufman BA, Lee BC, et al. Diagnostic testing for acute head injury in children: when are head CT and head radiographs indicated? Pediatrics 1997;99:11.
11. Swischuk LE. Imaging of the newborn, infant, and young child. 4th ed. USA: Lippincott Williams & Wilkins; 1997.
12. Zimmerman RA, Gibby WA, Carmody RF. Neuroimaging, clinical and physical principles. New York: Springer-Verley; 2000.
13. Tuny GA, Kumar A, Richardson RC, Jenny C, Brown DB. Comparison of incidental and non incidental traumatic head injury in children on non-contrast computerized tomography. Pediatrics 2006;118:626-33.
14. Koo AH, La Rouge RL. Evaluation of head trauma by CT. Radiology 1977;123:334-350.
15. French BN, Dublin AB. The value of CT scanning in the management of 1000consecutive cases of head injuries. Radiology 1977;125:464.
16. Al-rawi W, Ameen A, Altaee M. Computerized tomographic scan findings with persistent acute post-traumatic headache. Basrah Journal of Surgery 1995;1:74-8.
17. Armstrong P, Wastie ML. A concise textbook of radiology. GB: Arnold; 2001.
18. Stark DD , Bradely WG. Magnetic resonance imaging. USA: Mosby; 1999.

پوخته

هلنگافتنا سهری - فه کولینه که بئامیری سی تی سکائی

پیشه کی: بریندار بونا سهری نه گهری سهره کی یه بوژ دهست دانا گیانی و په ککهفتنی ل جهم گه نجان , و دیسان نها به لافه تر بویه ژ ههر دهمه کی پیشین.

و نها دهست نیشانکرنا سهربارکین بریندار بونا سهری بئامیری تیشکی یا هاتیه گوهورین بئامیری سی تی سکائی ژ بو هه لسه نگاندا دهسپکی د حاله تین هلنگافتنا سهری دا , لی ئامیری MRI دهیته بکارئینان دهنده حاله تین تایبه تی دا.

ئارمانج: هه لسه نگاندا رولی سی تی سکائی د هلنگافتنا سهری دا.

رئیکین فه کولینی: دفی فه کولینی دا سهربارکین هلنگافتنا سهری بئامیری سی تی د 50 حاله تان دا, ئه وین لنه خوشخانا نازادی هاتینه نشاندن ژ هه یفا ئاداری 2003 هه تا دوماهیا کانونا یه کی 2005 یین هاتینه تومارکرن و گه نکه شه کرن.

ئه نجام: وه سا ناشکه رابویه کو رویدانین هاتوچوی نه گهری سهره کی یه , و دیسان شکه ستنا کلوخی سهری یا هاتیه دیتن د باراپتر ژ فان حاله تان دا.

د فی فه کولینی localized brain oedema, brain contusion, intracerebral hematoma, لي subdural , subarachnoid, epidural hematomas کیمتر هاتینه تومارکرن.

سهرئه نجام ئه وه کو ئامیری سی تی گرنگ و کاریگه ره د ناشکراکرنا سهربارکین بریندار بونا سهری دا , لهوا پیدفی دهست نیشانکرنا کا زو و چاره سهری و لدوف چونه کا ریک و پیکه.

ده ئه نجام: سهره ت نه گهری سهره کی ژی, ئانکو رودانین هاتوچوی , پیدفی هه مو که نالین میدیای رولی خو ببینن ژبو کیمکرنا رودانین ریکان و بهر زکرنا ئاستی ره وشه نبیری ی دفی بواری دا.

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ANTI- β 2- GLYCOPROTEIN I AUTOANTIBODIES MORE CORRELATES WITH STROKES IN ANTIPHOSPHOLIPID SYNDROME

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ABSTRACT

Objectives To determine the significance of testing for anti-beta-2 glycoprotein I dependent ($\alpha\beta$ 2-GPI) among stroke patients of antiphospholipid syndrome (APS).

Subjects and Methods Fifty selected patients with strokes and 30 healthy individuals were tested for IgG isotype of anticardiolipin (aCL), $\alpha\beta$ 2-GPI, and antiphosphatidyl serine (aPS) autoantibodies. The study was conducted in the Postgraduate Laboratory Center, College of Medicine, and University of Dohuk, Iraq during April 2005. The indirect solid phase enzyme – linked immuno-sorbent assay (ELISA) technique was used for the detection of IgG aCL, $\alpha\beta$ 2-GPI, and aPS. The studied cases and controls were under 50 years of age and had no recognizable risk factors.

Results One or more of the tested APLAs were detected in 14/50 (28%) of cases with stroke. The IgG aCL was detected in 11/50 (22%), IgG $\alpha\beta$ 2-GPI in 14/50 (28%), and IgG aPS in 9/50 (18%) of the studied patients. However, IgG aCL was detected in 2/30 (6.7%) of controls with absence of other antiphospholipids antibodies (APLAs). All IgG aPS positive cases (9/14, 18%) were found to be positive for IgG $\alpha\beta$ 2-GPI and IgG aCL markers. Moreover, all IgG aCL positive cases (11/50, 22%) were positive for IgG $\alpha\beta$ 2-GPI marker. On the other hand, IgG $\alpha\beta$ 2-GPI as a sole marker was detected in another 3 positive APLAs cases with (3/14, 21.4%).

Conclusions In this study, IgG $\alpha\beta$ 2-GPI autoantibodies were found to correlate more with stroke cases of APS than other APLAs. These findings necessitate testing for this marker and ascertain the evidence accumulating which would assign a central role played by $\alpha\beta$ 2-GPI in strokes of APS.

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Key words: Anti- β 2 glycoprotein I autoantibodies, Stroke

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There is a substantial number of gaps in the knowledge about the association between stroke and APS.¹ Cerebral ischaemia associated with APS occurs at a younger age than the typical atherothrombotic cerebrovascular disease and is often recurrent and associated with high positive GPL APLAs(1 GPL equal to 1 μ g/ml of IgG) values and usually linked to the presence of aCL antibodies.^{2,3} Other data suggested that subsequent thrombo-

occlusive events and death after focal cerebral ischaemia associated with IgG aCL and might occur sooner.⁴

The APLAs were found in 25% of patients under the age of 45 with cerebrovascular accident with unclear etiology.⁵ Another study reported the prevalence of APLAs in 20% of stroke victims under the age of 50 years.⁶ The relative risk of stroke associated with the presence of APLAs had been assessed and an increased risk of stroke associated with the presence of antibodies of IgG type that were dependent upon the presence of β 2-GPI was observed.⁷

The APS should be suspected in young patients with stroke in setting of detection of APLAs, particularly in the absence of other risk factors.⁷ Therefore, the aim of this study was to determine the significance of testing for anti-beta-2 glycoprotein I dependent ($\alpha\beta$ 2-GPI) among stroke patients of APS.

MATERIALS AND METHODS

Fifty selected patients suffering from stroke attacks and 30 healthy individuals were tested for APLAs in the Postgraduate Laboratory of Medical College, University of Dohuk, Iraq during April 2005. The cases were enrolled from those seen in the main hospitals in Mosul City, Iraq between March 2004 and March 2005. The patients were not being previously diagnosed as SLE cases. Both groups were under 50 years of age and having no recognizable risk factors including hypertension (systolic blood pressure <140 mm Hg and / or diastolic of 90 mm Hg), diabetes

mellitus (fasting blood glucose of ≤ 126 mg/dl and/or 2-hours Post-prandial glucose of ≤ 140 mg/dl), dyslipidemia (total cholesterol level of <240 mg/dl and HDL of >35 mg/dl), and smoking (current or previous). The stroke cases were defined by clinical criteria of focal neurological deficits of sudden onset lasting ≥ 24 hours. The cases were clinically diagnosed by neurologists and the majority (44/50, 88%) of cases confirmed by image techniques such as Computerized Tomography (CT) scanning or Magnetic Resonance Imaging (MRI).

The Enzyme-linked immuno-sorbent assay kits (Orgentic Diagnostika Mainz/Germany) were used for the detection of APLAs, which were IgG of aCL, $\alpha\beta$ 2-GPI, and aPS autoantibodies, by indirect solid phase enzyme immunoassay (ELISA) technique. The IgG isotype results were assessed in IgG phospholipid (GPL) units, with one unit equal to 1 μ g/ml of IgG. According to manufacturer's instructions, cases were considered to be significantly positive if the titer was >10 GPL units for aCL, >8 GPL for $\alpha\beta$ 2-GPL, and >10 GPL for aPS autoantibodies.

RESULTS

Among the studied stroke patients, IgG aCL was detected in 11/50 (22%), IgG $\alpha\beta$ 2-GPI in 14/50 (28%), and IgG aPS in 9/50 (18%). Among the control group, IgG aCL was detected in 2/30 (6.7%) with absence of other autoantibodies. Among the APLAs positive stroke patients, combined profile of IgG aCL with IgG $\alpha\beta$ 2-GPI was observed in all IgG aCL

positive cases and profile of IgG aPS with IgG α 2-GPI in all IgG aPS positive cases too. Moreover, IgG α 2-GPI marker as a sole marker was detected in another 3/14 (21.4%) of patients in the absence of other APLAs.

The neuro-imaging of the brain (CTS or MRI) among the 14 APLAs positive stroke cases revealed normal findings in 4/14 (28.6%), localized ischaemic infarcted area in 6/14 (42.8%), and multiple infarcted areas in 4/14 (28.6%) cases. On the other hand, none of the studied cases showed imaging evidence of hemorrhage. All cases with positive neuroimaging findings of localized area were with history of single stroke attack, while cases with multiple affected areas were with history of recurrent episodes of stroke.

DISCUSSION

The APS is an autoimmune disease characterized by existence of one or more of APLAs and at least one clinical manifestation, the most common being venous or arterial thrombosis and recurrent fetal loss. Since the discovery in 1990 that β 2-GPI is involved in APS as a co-factor the interest in this protein has increases tremendously, but crucial information is still lacking. This protein has low affinity for anionic phospholipids compared to other plasma proteins such as clotting factors; meanwhile this affinity increases up to 100 times after interaction with α 2-GPI.^{8,9} Therefore, as a result of this increase in affinity, these proteins interfere to a significant degree with the interaction

of other plasma proteins with anionic phospholipid surfaces and can inhibit their coagulation function.¹⁰

In this study, one or more of positive APLAs were detected in 28% of stroke cases and this finding is notable in the region. The IgG aCL was detected in 22%, IgG aPS in 18%, and IgG α 2-GPI in 28% of stroke cases. Moreover, all IgG aCL and IgG aPS positive cases were found to be positive for IgG α 2-GP too. Therefore, all detected APLAs positive stroke patients were found to harbor IgG α 2-GPI autoantibodies. In addition, 3 other stroke patients were detected to be positive for IgG α 2-GPI as a sole marker (i.e., absence of IgG aCL and IgG aPS). From these findings, IgG α 2-GPI autoantibodies were found to correlates more with stroke patients than other APLAs markers in APS.

The term "antiphospholipid cofactor syndrome" was used to describe cases in which clinical manifestations are related to APS in the presence of α 2-GPI and absence of other APLAs markers.¹¹ This highlights the importance of testing for α 2-GPI antibodies assay in diagnosing clinical events related to APS to avoid underestimation of cases. In addition, this syndrome was also reported by Picillo et al¹² in cases with recurrent arterial and/or venous thrombosis without SLE. Moreover, Katsarau et al¹³ reported this phenomenon among a case with ischemic stroke.

The mechanisms by which α 2-GPI initiate prothrombotic states are multiple and many receptors on different cell types have been proposed to bind the β 2-GPI-

antibody complex and affecting the coagulation cascade. The induction of tissue factor, intracellular adhesion molecules (ICAM) and vascular adhesion molecule (VCAM) are examples of this pathogenic activity of $\alpha\beta 2$ -GPI on endothelial cells.^{14,15} The neuro-radiological imaging procedures performed here revealed positive findings in 71.4% (10/14) in APLAs positive cases and multiple involved areas were diagnosed in all cases with recurrent attacks. Therefore, this imaging technique may be helpful aid in the diagnosis of these events related to APS and localizing the lesion.

Beyers et al¹⁶ and Arnout et al¹⁷ reported that both Lupus anticoagulant (LA) and conventional aCL to be non-specific tests since LA can detect antibodies to both human prothrombin and human $\beta 2$ -GPI or other antibodies. Moreover, the conventional aCL assay can detect a range of antibodies, including antihuman $\beta 2$ -GPI, antibodies directly bind cardiolipin, and antibodies directed to other cardiolipin-binding plasma proteins.^{18,19} Although, in 1999 the determination of $\alpha\beta 2$ -GPI was not included in the Sapporo laboratory criteria for diagnosis of APS,²⁰ but it was suggested as important testing marker in meeting in Boston in 2002.²¹ The main problem in different previous studies was the lack of standardization of $\alpha\beta 2$ -GPI antibody ELISA kits. New ELISA assay techniques with improved sensitivity and specificity for testing $\alpha\beta 2$ -GPI dependent methods are in use.

In conclusion, the detected rate of APLAs among stroke cases in this region is notable and worthwhile. Moreover, IgG $\alpha\beta 2$ -GPI autoantibodies rate was found to correlates more with stroke cases of APS than IgG aCL and IgG aPS autoantibodies. These findings necessitate testing for this marker in stroke patients and ascertain the evidence accumulating which would assign a central role played by $\alpha\beta 2$ -GPI in stroke cases of APS.

REFERENCES

1. Gatenby PA. Controversies in the antiphospholipid syndrome and stroke. *Thromb Res* 2004; 114:483-8.
2. Brey RL, Hart RG, Sherman DG, Tegeler CH. Antiphospholipid antibodies in cerebral ischemia in young people. *Neurology* 1990; 40:1190-6.
3. Nencini P, Baruffi MC, Abbate R, Massai G, Amaducci L, Inzitari D. Lupus anticoagulant and anticardiolipin antibodies in young adults with cerebral ischemia. *Stroke* 1992;23:189-93.
4. Brey RL, Stallworth CL, McGlasson DL, Wozniak MA, Wityk RJ, Stern BJ, et al. Antiphospholipid antibodies and stroke in young women. *Stroke* 2002;33:2396-400.
5. Munts AG, van Genderen PJ, Dippel DW, van Kooten F, Koudstaal PJ. Coagulation disorders in young adults with acute cerebral ischemia. *J Neurol* 1998;245:21-5.

6. Gromnica-Ihle E, Schossler W. Antiphospholipid syndrome. *Int Arch Allergy Immunol* 2000;123:67-76.
7. Brey RL, Abbott RD, Sharp DS, Ross GW, Stallworth CL, Kittner SJ. Beta(2)-Glycoprotein I-dependent anticardiolipin antibodies and risk of ischemia stroke and myocardial infarction: the Honolulu heart program. *Stroke* 2001;32:1701-6.
8. Willems GM, Janssen MP, Pelsers MM, Comfurius P, Galli M, Zwaal RF, et al. Role of divalency in the high-affinity binding of anticardiolipin antibody- β 2-glycoprotein I complexes to lipid membranes [Abstract]. *Biochemistry* 1996;35:13833-42.
9. Lutters BC, Meijers JC, Derksen RH, Arnout J, de Groot PG. Dimers of β 2-glycoprotein I mimic the in vitro effects of β 2-glycoprotein I- anti- β 2-glycoprotein I antibody complexes. *J Biol Chem* 2001;276:3060-7.
10. de Laat HB, Derksen RHW, de Groot PH. β 2-Glycoprotein I, the playmaker of the antiphospholipid syndrome. *Clin Immunol* 2004;112:161-8.
11. Cabral AR, Amigo MC, Cabiedes J, Alarcon-Segovia D. The antiphospholipid/cofactor syndromes: a primary variant with antibodies to β 2-glycoprotein I but no antibodies detectable in standard antiphospholipid assays. *Am J Med* 1996;101:472-81.
12. Picillo U, Marcialis MR, Italiano G. Antibodies to β 2-glycoprotein I in anticardiolipin negative patients. *J Rheumatol* 1998;25:1440-2.
13. Katsarou E, Attilakos A, Fessatou S, Tsapra H, Tzavara V, Dracou C. Anti-B2-Glycoprotein I antibodies and ischemic stroke in a 20 month-old boy. *Pediatrics* 2003;112(1):188-90.
14. Gharavi AE, Pierangeli SS, Colden-Stanfield M, Liu XW, Espinola RG, Dracou C. GDKV induced antiphospholipid antibodies enhance thrombosis and activate endothelial cells in vivo and in vitro. *J Immunol* 1999;163:2922-7.
15. Salemink I, Blezer R, Willems GM, Galli M, Bevers E, Lindhout T. Antibodies to β 2-glycoprotein I associated with antiphospholipid syndrome suppresses the inhibitory activity to tissue factor pathway inhibitor. *Thromb Haemost* 2000; 84:653-6.
16. Bevers EM, Galli M, Barbui T, Comfurius P, Zwaal RF. Lupus anticoagulant IgG (LA) are not directed to phospholipids only, but to a complex of lipid-bound human prothrombin. *Thromb Haemost* 1991; 66:629-32.
17. Arnout J, Meijer P, Vermeylen J. Lupus anticoagulant testing in Europe: an analysis of results from the First European Concerted Action on Thrombophilia (ECAT) survey using plasmas spiked with monoclonal antibodies against human β 2-glycoprotein I. *Thromb Haemost* 1999;81:929-34.
18. Roudey RA. Immunology of the

- antiphospholipid antibody syndrome. *Arthritis Rheum* 1996;39:1444-54.
19. Rampazzo P, Biasiolo A, Garin J, Rosato A, Betterle C, Ruffatti A, et al. Some patients with antiphospholipid syndrome express hitherto undescribed antibodies to cardiolipin-binding proteins. *Thromb Haemost* 2001; 85:57-62.
20. Hughes GR. Hugh's syndrome: the antiphospholipid syndrome. A historical view. *Lupus* 1998;7 Suppl 2:1-4.
21. Pengo V. Communication. 48th Annual SSC Meeting, Boston, MA, USA. [online]. 18 Jul 2002 [cited 24 Apr 2007]. Available from: URL: [http:// www.med.unc.edu/isth/](http://www.med.unc.edu/isth/)

پوخته

دزی بیتا گلوبولین په یوه نډیه کا پتر یا هه ی ب نه خوشیښ نه ویښ میښکی ل ئیښا جوړښ فوسفاتی

ریښیښ څه کولینی: نه ډ څه کولینه هاته نه نجامدان ل سهر 50 نه خوشیښ نه ویښ میښکی هه یښ ژ نه خوشخانیښ دهوک و میسل و هه ولیر دگه ل 30 که سیښ ساخلم ل سالا 2006 . ل څی څه کولینی دا فه حسیښ antiphosphatidyl serine (aPS) autoantibodies , a β 2-GPI anticardiolipin (aCL) , بو هه می که سیښ به شدار د څه کولینی دا.

نه نجام: دیار بوو کو IgG aCL پوزه تیښ بوو ل 50/11 (%22) و IgG a β 2-GPI ل 50/14 (%28) و IgG aPS ل 50/9 (%18) ژ نه خوشان.

دهر نه نجام: ل څی څه کولینی نه شکر ابوو کو ئیښا جوړښ فوسفاتی ل 28% ژ فان نه خوشان هه بوو و هه می نه ډ نه خوشه پوزه تیښ بوون بو که ره سته دزی بیتا گلوبولین. ژ بهر فان راستیښ دیار بوو یښ نه م دیږ یښ دزی بیتا گلوبولین په یوه نډیه کا پتر یا هه ی ب نه خوشیښ نه ویښ میښکی ل ئیښا جوړښ فوسفاتی.

	(a β 2-GPI)	:
	30	50
		:
	(aCL)	
	(aPS)	(a β 2-GPI)
	(%28) 50/14	:
(%28) 50/14	(%22) 50/11	
	(%18) 50/9	
	(%28)	:
	2	

RELIABILITY AND ACCURACY OF EXERCISE TOLERANCE TEST IN THE DIAGNOSIS OF CORONARY HEART DISEASE

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ABSTRACT

Objectives Estimation of the reliability and accuracy rate of exercise tolerance test in the diagnosis of coronary heart diseases.

Setting Ibn Al Betar Hospital / Baghdad / Iraq.

Methodology The study enrolled 53 patients (36 males and 17 females) from those referred to Ibn Al Betar Hospital with a provisional diagnosis of coronary heart disease during the period May / June 2005. All patients were subjected to the exercise tolerance test. The results were analyzed with reference to the results of subsequent coronary angiography in order to estimate the reliability and accuracy rate of the test.

Results The results revealed moderate test reliability (Kappa value = 0.47) with an accuracy rate of (62.2%). It was less accurate in females but this gender difference did not achieve statistical significance (p value > 0.05).

Conclusions The moderate test reliability needs more extensive investigation to delineate the various sources of variability, whereas improving its awkward accuracy rate mandates the expanded use of other supportive tests such as nuclear imaging and 2D echocardiography.

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Key words: Coronary heart disease, Exercise tolerance test, Reliability, Accuracy rate

Ischemia that is not present at rest can be detected by precipitation of typical chest pain or ST segment depression. Treadmill, stress, or exercise tolerance electrocardiography (ETT) is the most useful non – invasive procedure for evaluating patients with coronary heart disease. While being sensitive and useful in the detection and quantitation of coronary heart disease yet, it is not infallible and may produce false positive results. The results are also influenced by the severity of coronary heart disease and

hence false negative results are low in severe cases and vice versa. The predictive accuracy of (ETT) is lower in women than in men.¹⁻⁴

The test should be classified as non-contributory (and not negative) if the patient can not achieve an adequate level of exercise because of locomotor or other non cardiac problems.

Coronary angiography provides detailed information about the extent and nature of coronary artery disease, and may be indicated when non-invasive tests have failed to elucidate the cause of atypical chest pain.²

Accuracy is the test ability to correctly identify positive and negative cases while reliability refers to the consistency of the

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results when repeat examinations are performed under constant conditions. Sources of variability in any test may come from biological variations; instrumental variations, intra and inter observer errors. Reliability is measured by the Kappa value (κ) which relates actual measure of agreement between two examiners and the agreement by chance.⁵⁻¹¹

Different and widely diverse figures were reported by different studies and authors regarding the evaluation of the results of the exercise tolerance test (ETT).^{12,13}

The study has been designed to explore this problem and to extract local figures from routine hospital practice.

Aims of the Study: Estimation of the reliability (Kappa value) and accuracy rate of ETT.

PATIENTS AND METHODS

The sample included 53 patients aged 40 – 70 years (36 males and 17 females) referred to Ibn Al-Betar hospital with a provisional diagnosis of angina pectoris during the period May/ June 2005. Ibn Al Betar Hospital, Baghdad/Iraq is a specialized third level center in cardiovascular surgery.

Stress ECG testing was performed for all patients on motorized treadmill using Bruce Protocol up to a predicted maximal heart rate for age.¹⁴

The results were compared with the results of subsequent coronary angiogram

which is considered as a reference test. The data were summarized and tabulated in a fourfold table in order to estimate the required rates.

The calculated rates are the following:

1. Sensitivity (%) = $\frac{a}{a + c} \times 100$
2. Specificity (%) = $\frac{d}{b + d} \times 100$
3. Accuracy (%) = $\frac{a + d}{a + b + c + d} \times 100$
4. False Positive (%) = 1 – specificity
5. False Negative (%) = 1 – sensitivity
6. Reliability: expressed as Kappa value (κ).

$$\text{Kappa } (\kappa) = \frac{\text{Observed agreement} - \text{total chance agreement}}{100 - \text{Total chance agreement}}$$

Kappa (κ) is expressed as parts of one, thus:

$$\text{Moderate agreement} = 0.4 - 0.6$$

$$\text{Substantial agreement} = 0.6 - 0.8$$

$$\text{Good agreement} = > 0.8$$

$$\text{Total agreement} = 1$$

ETT	Coronary Angiography		Total
	Positive	negative	
Positive	a	b	a + b
Negative	c	d	c + d
Total	a + c	b + d	a + b + c + d

a= True Positive, b= False Positive, c= False Negative, d= True Negative

RESULTS AND DISCUSSION

38 cases proved positive by (ETT) (26 males and 12 females) compared to an angiogram yield of 30 cases (22 males and 8 females). 15 cases proved negative by (ETT) (10 males and 5 females) compared to a negative angiogram in 23 cases (14 males and 9 females) (Table 1).

Table 2 shows a summary of the estimated rates. The overall sensitivity, specificity and accuracy rates were 80%, 39.1% and 62.2% respectively. Male figures (81.8%, 42.8% and 66.6% respectively) were higher, compared to female figures (75%, 33.3% and 52.9% respectively). The lower sensitivity and specificity in females led to higher false positive and false negative rates (66.7% and 25% respectively).

Statistical analysis of table 2 using Z- test to compare the different rates

showed that all comparisons proved statistically insignificant ($P_{\text{value}} > 0.05$).

It is evident that there is a crucial need for improving accuracy rate. Increased yield might be expected with the use of other supportive maneuvers like the combined use of nuclear imaging and 2D echocardiography. In addition to that, matters related to careful interpretation should be followed. These include the meticulous search for signs other than ST – T changes that may correlate with the presence or severity of coronary heart disease e.g. sub maximal pulse response or ventricular ectopy.^{3,14}

Kappa value (κ) equals 0.47 which indicates moderate agreement. Such a limited value might be due to errors in interpretation, inherited biological variations, instrumental defects or poor pre- test preparation e.g. ignoring the prerequisite of fasting status and the exclusion of prior anti – ischemic drugs.

Table 1. ETT / Coronary Angiogram Results for Males & females

ETT *		Coronary Angiography		Total	
		Positive	negative		
Positive	Male	18	8	26	38
	Female	6	6	12	
Negative	Male	4	6	10	15
	Female	2	3	5	
Total		30	23	53	

* (ETT) Reliability (κ) = 0.47

Table 2. Summary statistics for the estimated rates by gender

Rates	Males & Females	Males	Females
Sensitivity	80% *	81.8%	75%
Specificity	39.1%	42.8%	33.3%
Accuracy	62.2%	66.6%	52.9%
False Positive	60.9%	57.2%	66.7%
False Negative	20%	18.2%	25%

* Based on Z- Test

 $P_{value} > 0.05$ for all comparisons

CONCLUSIONS

1. The test reliability shows moderate agreement as evidenced by a Kappa value (κ) of 0.47.
2. The accuracy rate of (ETT) is 62.2%. It is less accurate in females (52.9%) than in males (66.6%). The gender difference did not achieve statistical significance ($P_{value} > 0.05$).
3. The high false positive rate (60.9%) is a reflection of the low specificity of the test (39.1%). Similarly, the low false negative rate (20%) is a reflection of its sensitivity rate (80%).

RECOMMENDATIONS

1. To improve reliability, a larger prospective study including larger sample size is needed to highlight the different sources of variability and correct them accordingly.
2. To increase the accuracy rate, other supportive tests might be used in conjunction with (ETT) such as nuclear imaging and 2D echocardiography.

REFERENCES

1. Massie BM, Amidon TM. Heart. In: Tierney LM, McPhee ST, Papadakis M A, editors. Current medical diagnosis and treatment. 42nd ed. New York: Lange/ McGraw-Hill; 2003. p. 378.
2. Boon NA, Fox KA, Bloomfield P, Bradbury A. Cardiovascular disease. In: Hunter JA, editor. Davidson's principles and practice of medicine. 19th ed. Edinburgh: Churchill Livingstone; 2002. p. 426.
3. Hegar JW, Niemann JT. Cardiology. 3rd ed. Baltimore: Williams & Wilkins; 1997.
4. Sokolow M, McIlroy MB. Clinical cardiology. New York: Lange Medical Publications; 1999.
5. Cassens BJ. Preventive medicine and public health. 2nd ed. Baltimore: Williams & Wilkins; 1992.
6. Niazi AD. Statistical analysis in medical Research. Baghdad: Alnahrain University; 2001.
7. Hennekens CH, Buring J. Epidemiology in medicine. Boston:

- Little Brown Company; 1987.
8. Moser C, Kalton G. Survey methods in social investigation. 2nd ed. London: Heinemann Educational Books; 1999.
 9. Macmahon B, Tricopoulos D. Epidemiology principles and methods. 2nd ed. Boston: Little Brown Company; 1996.
 10. Mausner JS, Kramer S. Epidemiology: an introductory text. 2nd ed. New York: W.B. Saunders Company; 1986.
 11. Schulz KF, Grimes DA. The lancet handbook of essential concepts in clinical research. Edinburgh: Elsevier; 2006.
 12. Thwaites BC, Quyum AA, Taphael M J. Comparison of ST/heart rate slope with the modified Bruce protocol exercise test in the detection of coronary artery disease. Am J Cardiol 1986;57(8):554-6.
 13. Kotler TS, Diamond GA. Exercise thallium-201 scintigraphy in the diagnosis and prognosis of coronary artery disease. Ann Intern Med 1990;113:684-6.
 14. McInnis KJ, Balady GJ, Weiner DA, Ryan TJ. Comparison of ischemic and physiological responses during exercise tests in men using the standard and modified Bruce protocols. Am J Cardiol 1992;69(1):84-9.

پوخته

متمانندن و هوپرکا فهحسا رهنج یا دلی بو دهست نیشانکرنا نه خوشیپن رههپن دلی

ئارمانج: ژماردنا متمانندن و هوپرکا فهحسا رهنج یا دلی بو دهست نیشانکرنا نه خوشیپن رههپن دلی.

جهیّ قه کولینی: نه خوشخانا () / به غدا / عیراق کو یا تایبه ته ب نشته گه ریا دل و رههپن خوینی.

ریکین قه کولینی: نموونه پیکهاتی بوو ژ 53 نه خوشا (36 نیر و 17 یپن می) ئه وین هاتینه رهوانه کرن بو نه خوشخانا () یپن گومان لی کری کو تووشی نه خوشیپن رههپن دلی بووین ل هه یفا گولانی و خزیرانی ل سالا 2005. فهحسا رهنج یا دلی بو هه می نه خوشا هاته کرن. ئه نجام هاته هه قبه رکرک دگهل ئه نجامین قه سته را رههپن تاجی ژ بو ژماردنا متمانندن و هوپرکا فهحسا رهنج یا دلی.

ئه نجام: هاته دیار کرن دقه کولینی دا متمانندنه کا نافنجی (Kappa value= 0.47) و هوپرکا ب ریژه یا 62.2%. هوپرکا فهحسی کیمتر بوو ل می یا، به لی جیاوازی چی مفایین ئاماری نه بوون ($p>0.05$).

THE FREQUENCY OF 2 HOURS POST GLUCOSE LOAD HYPERGLYCEMIA IN SUBJECTS WITH NORMAL FASTING GLUCOSE

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ABSTRACT

Background The prevalence of impaired glucose tolerance (IGT) and previously undiagnosed diabetes mellitus (PU-DM) in variety of ethnic group has been well documented. Limited information, however, is available about the presence in Iraqi population.

Objective The aim of this study is to investigate the prevalence of the glucose intolerance and undiagnosed diabetes among healthy adult population.

Materials and methods A total of 918 members of the general population of Baghdad city were studied. They presented different age groups (ranging from 20 to 60 years). All subjects were in good health, and had normal fasting serum glucose levels (<6.1 mmol/L). For assessment of hyperglycemia, all subjects underwent an oral glucose tolerance test.

Results Among 918 subjects (401 males and 517 females), 19.0% males and 16.8% females had IGT. The prevalence of PU-DM was 1.2% for males and 5.6% for females. The prevalence increased with age in both genders. IGT and PU-DM were more commonly seen in obese subjects than in normal weight subjects (IGT: 24.5% vs 11.9%) and (PU-DM: 6.3% vs 1.9%).

Conclusions This report on the IGT and PU-DM from Baghdad population showed a high prevalence of these disorders.

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Key words: Asymptomatic Hyperglycemia, Impaired glucose tolerance, Diabetes mellitus, Prevalence

The rising burden of obesity and diabetes worldwide represents a tremendous challenge for health-care systems.¹

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Published studies regarding this problem in Iraq are limited and most of them concentrated on the prevalence of overt type-2 diabetes.² The aim of this study was to determine the prevalence of glucose intolerance and undiagnosed diabetes among apparently healthy adults, especially those with normal fasting serum glucose levels.

SUBJECTS & METHODS

From January through December 2005, 918 members of the general population

(401 males and 517 females), who were recruited through several medical institutes in Baghdad city were studied. They represented different age groups (ranging from 20 to 60 years) of adult urban living in various parts of Baghdad city. All subjects were in good health, had normal fasting serum glucose levels (< 6.1 mmol/L), and were not taking any medication. They had no history or clinical evidence of severe chronic diseases and no history of excessive alcohol intake. None of the females was pregnant. All subjects underwent an Oral Glucose Tolerance Test (OGTT). The OGTT was performed according to World Health Organization (WHO) specifications.³ The subjects were given a 300-ml beverage containing 75 g glucose and were asked to consume it in < 5 min. Blood specimens were collected into plain tubes immediately before and 2 h after the glucose load. The specimens were centrifuged, serum was separated immediately, and serum glucose levels were determined using ACE Bench-Top automated analyzer, which uses a glucose oxidase method. Fasting serum glucose after 12-14 hours overnight fasting and serum glucose 2 hours after ingestion of 75 g glucose were measured. The participants were divided into three main categories on the bases of glucose tolerance: Normal Glucose Metabolism (NGM, $n=721$), Impaired Glucose Tolerance (IGT, $n=163$), or Previously Undiagnosed Diabetes Mellitus (PU-DM, $n=34$).

The diagnosis was made according to the criteria adopted by the American Diabetes Association (ADA) in 1997⁴;

(NGM: FPG < 6.1 mmol/L, and 2-h post load-plasma glucose < 7.8 mmol/L; IGT: FPG < 7.0 mmol/L and 2-h post load-plasma glucose 7.8-11.1 mmol/L; type 2 diabetes: FPG ≥ 7.0 mmol/L or 2-h post load-plasma glucose ≥ 11.1 mmol/L). For assessment of over weight and obesity, the height and weight measurements for each subject were used to calculate the body mass index. [BMI=weight (kg)/height (m^2)]. Subjects with a BMI < 25.0 were considered to have normal weight, overweight ≥ 25.0 to 29.9 and obese ≥ 30.0 .⁵

RESULTS

Table 1 shows the prevalence of IGT and PU-DM among the studied population. The prevalence of IGT was relatively higher in males than in females (19% vs 16.8%), whereas the prevalence of PU-DM was 1.2% for males and 5.6% for females. However, the over all prevalence of IGT was 17.8% and PU-DM was 3.7%. The higher prevalence of IGT and PU-DM was in the older age subjects > 50 years, (29.0% and 7.5%) compared to the middle age subjects < 50 years (16.1% and 2.1%) respectively. Obese subjects had higher prevalence of IGT and PU-DM than normal weight subjects (IGT: 24.5% vs 11.9 %, and PU-DM: 6.3% vs 1.9%) the mean value of 2-h post carbohydrate serum glucose concentrations in normal weight subjects was 6.15 ± 2.1 mmol/L, overweight 6.52 ± 2.3 mmol/L and in obese 7.22 ± 2.6 mmol/L, the differences were statistically significant (p value < 0.01). Subjects with positive family history for

Table 1. Prevalence of IGT& PU-DM among study population (N=918)

		NGM N=721	IGM N= 163	PU-DM N=34
		No. (%)	No. (%)	No. (%)
Sex				
males	401	320 (79.8)	76 (19.0)	5 (1.2)
Females	517	401 (77.6)	87 (16.8)	29 (5.6)
Age in years				
21-30	366	316 (86.3)	41 (11.2)	9 (2.5)
31-40	285	220 (77.2)	52 (18.2)	13 (4.6)
41-50	160	117 (73.2)	39 (24.3)	4 (2.5)
51-60	107	68 (63.5)	31 (29.0)	8 (7.5)
BMI in kg/m²				
Normal weight <25.0	268	231 (86.2)	32 (11.9)	5 (1.9)
Overweight 25.0-29.9	381	304 (79.8)	65 (17.1)	12 (3.1)
Obese >30.0	269	186 (69.2)	66 (24.5)	17 (6.3)
Family history for DM				
Positive	147	93 (63.3)	45 (30.6)	9 (6.1)
Negative	771	628 (81.5)	118 (15.3)	25 (3.2)

diabetes also had higher prevalence for IGT and PU-DM compared with those with negative family history of DM (IGT: 30.6% vs 15.5% and PU-DM: 6.1 % vs 3.2).

DISCUSSION

The prevalence of IGT varies among different population.⁶⁻⁸ The present study showed 17.8 % had IGT; and 3.7% had PU-DM; which increases progressively with increasing age, ranging from 11.2 to 29.0 % and 2.5 to 7.5 %.

Were these percentages applied to the 27 million Iraqi populations, beside the fact that a half population in Iraq is adults, about 2.4 million adults aged 20-60 would have IGT and almost half million would have type -2 diabetes.

Several studies documented similar findings; an IGT prevalence of 11.0 % in US adults with no previous diagnosis of diabetes was evident in the International Health and Nutrition Examination study.⁹ Results from the European Horn Study,¹⁰ which investigated Dutch population, showed an IGT prevalence of 10.3% with large difference between different age groups. It is worrying to find such a high prevalence of IGT among our population. Since IGT is an independent risk for type-2 diabetes.¹¹ Accordingly, we can see that type-2 diabetes is on the high scale. It is therefore conceivable that a high prevalence of IGT among our population, especially older age subjects may suggest a high potential for further rise in type-2 diabetes. Many risk factors are observed for the high prevalence of IGT or DM,¹²

such as high racial susceptibility to diabetes, high familial aggregation, central obesity, insulin resistance and life style change due to urbanization. In Iraq, as well as in other countries, these factors are at increased rate, and expected to be more prevalent. Studies suggest that weight loss and increased physical activity among people with pre-diabetes prevent or delay diabetes and may return blood glucose levels to normal.¹³ In our population, the risk confirmed by the high level of BMI found in a large proportion of the studied sample. Indeed, 68.3% of the subjects were overweight or obese. This observation was noted before.¹⁴ Despite the fact that subjects with impaired fasting glucose (IFG) were not included in our study, the mean serum glucose levels during OGTT were high in obese subjects, and in correlation analysis a significant relationship was found between glucose levels and BMI values ($r=0.189$, p value <0.01).

Thus, we found that obese subjects had higher prevalence of IGT and PU-DM in comparison with normal weight subjects. In view of the confounding effect of the high familial aggregation of diabetes,¹⁵ the prevalence of IGT among offspring with one or both diabetic parent was 30.6% and PU-DM 6.1 %. In this respect, It is of interest to note that of the 147 subjects who had positive family history of DM, 70(48%) were obese; such a finding may partly explain the high prevalence of IGT and PU-DM in our population. The association between obesity and hyperglycemia is stronger in the presence of parental history of

diabetes.¹⁶

CONCLUSIONS

These data indicate an increase in the frequency of hyperglycemic state in Iraqi adults. In addition, increased hyperglycemic state is related to unknown genetic defect combined with environmental factors, predominantly obesity.

REFERENCES

1. Betteridge DJ. Diabetic dyslipidemia. Diabetes, obesity and metabolism. Diabetes 2000;2 suppl 1:531-6.
2. Mula-Abed WS, Al-Naemi AH. Prevalence of diabetes mellitus in Mosull city. Ann Coll Med Mosul 2002;28(2):109-16.
3. World Health Organization: Definition, diagnosis and classification of diabetes mellitus and its complications: report of a WHO consultation. Part I: diagnosis and classification of diabetes mellitus. Geneva: World Health Organization; 1999.
4. American Diabetes Association. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care 1997;20:1183-97.
5. Garrow Js. Obesity and related disease. Edinburgh: Churchill Livingstone; 1988.
6. American Diabetes Association: Screening for type 2 diabetes (Position Statement). Diabetes Care

- 2003;26 Suppl 1:21-4.
7. El-Hazami MAF, Warsy AS, Al-Swailem AR, Al-Swailem AM, Suliaman R, Al-Meshari AA. Diabetes mellitus and impaired glucose tolerance in Saudi Arabia. *Ann Saudi Med* 1996;16(4):381-5.
 8. Al-Mahroos F, McKeigue P. Obesity, physical activity and prevalence of diabetes in Bahrain Arab native population. *Bahrain Med Bull* 1998;20(3):114-8.
 9. Harris MI. IGT prevalence and conversion to NIDDM. *Diabet Med* 1996;13:9-11.
 10. Heine RJ, Nipels G, Mooy JM. New data on the rate progression of IGT to NIDDM and predicting factors. *Diabet Med* 1996;13:12-4.
 11. Ramochandran A, Snehalatha C, Vijay V. Temporal changes in prevalence of type 2 diabetes and IGT in Urban Southern India. *Diabetes Res Clin Pract* 2002;58:55-60.
 12. Tsamura K, Hayashi T, Suematsu C. Life style and the risk or NIDDM. *Diabetes* 1998;47:148-51.
 13. American Diabetes Association. Clinical practice recommendations 1999: diabetes mellitus and exercise. *Diabetes Care* 1999;22 Suppl 1:49-53.
 14. AL-Timimi DJ, Esmail W. Serum Lipids and lipoproteins in the normal weight, over weight and obese women. *Iraqi Postgrad Med J* 2002;4:286-9.
 15. Davey G, Ramachandran A, Snehalatha C. Familial aggregation of central adiposity among Southern Indians. *Int J Obes* 2000;24:1523-7.
 16. Knowler WC, Pettit DJ, Saad MF. Obesity in the Pima Indians: its magnitude and relationship with diabetes. *Am J Clin Nutr* 1991;53:1543-51.

بوخته

زیده بوونا ریژهیا شه کری پستی دوو ده مژمیرا ژ وەرگرتنا گلوکوزی ل که سین کو ریژهیا شه کری دناف خوینی باش روژی بوونی نورمال بوون

پیشه کی: بهر به لافبوونا نه خوشیا شه کری یا نه دهست نیشانگری (PU-DM) و نه ریخستنا بارهه لگریا شه کری (IGT) ل نه تهوین جودا نوکه ب شیوهیه کی باش هاتیه دوکیومنتکرن. سه بارهت عیراقی وهسا یا خویا یه کو پیزانین لسه رفی دیاردی گهله ک ییت سنوردایه.

ئارمانج: دیارکرن بهر به لافبوونا نه خوشیا شه کری یا نه دهست نیشانگری (PU-DM) و نه شیاننا بارهه لگریا شه کری (IGT) دناف عیراقیین دژیین 20-60 سالی دا.

رێک و ئامیرین فه کولینی: ژمارا 918 که سان هاتنه فه کولین کرن ژ گشت باژی ری یه غدا. ژیی وان د نافهرا 20-60 سالان دا بوو. هه میا ته ندروستی یا وان باش بوو ئاستی شه کری دناف خوینی دا نورمال بوو پستی روژی بوونی دا.

ئه نجام: دناف 918 که سان (401 نیر و 517 می)، 19% ژ که سین نیر و 16.8% ژ که سین می نه ریخستنا بارهه لگریا شه کری (IGT) هه بوون و بهر به لافبوونا نه خوشیا شه کری یا نه دهست نیشانگری (PU-DM) دناف دا دریژا 1.2% ژ که سین نیر و 5.6% ژ که سین بوون. دگهل زیده بوونا ژی بهر به لافبوونا نه خوشیی دناف بهرا ههردوو ره گهزا دا بلند دبوو. ریژهیا (IGT و PU-DM) پتر هاته دیتن دناف که سین قهلهو دا بهراوهر دگهل وان که سین لسه تگ نورمال (IGT: 24.5% vs 11.9%) و (PU-DM 6.3% vs 1.9%).

دهرئه نجام: ئه فی راپورتی دیارکر کو بهر به لافبوونا نه خوشیین PU-DM و IGT دناف خه لکی به غدا دا گهله که.

(PU-DM)	:	(IGT)
918	:	
60 – 20		
%16.8	%19	:
	%5.6	%1.2
	:	

CASE REPORT

LEECH INFESTATION PRESENTING AS METROMENORRHAGIA

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ABSTRACT

Objective This case is presented to show that leech infestation is still not uncommon in Iraq especially of the genital tract. Leech infestation, could cause menorrhagia in women.

Case profile A 45years old multiparous woman from Sinjar's rural areas in Mosul province, attending the out patient department with severe vaginal bleeding, and a history of moderate, continuous vaginal bleeding for four months. She underwent diagnostic curettage three months before, which revealed normal secretory endometrium. The infected woman was pale and hypotensive with features of chronic iron deficiency anemia. Speculum and gynecological examinations demonstrated no abnormalities. Total abdominal hysterectomy as an emergency was done with preservation of both ovaries; bleeding persisted postoperatively; re-assessment by speculum pelvic examination revealed leech infestation of the vagina.

The Leech belongs to Phylum Annelida, class Hirudinea, and order Rhynchobdellida. An infestation with leeches should be considered in patients who present with menorrhagia and history of immersion in fresh water lakes or streams in tropical infested areas. Leech infestation is not common in gynecological practice, and is rare nowadays as water supplied to all cities and villages is passing through filtration and disinfection process.

Conclusion Leech infestation should be remembered, as a cause of menorrhagea, in areas where using river water and sitting at riverside is habitual for women in infested rural areas.

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Key words: Leech infestation, Menorrhagia

Leech of the Phylum Annelida, class Hirudinea are blood sucking hermaphroditic parasite that attach themselves to vertebrate host, bite through the skin, and suck out a quantity of blood, they are egg laying Annelida which have elongated annular bodies. To feed itself the leech applies its anterior sucker containing a mouth armed with three medially arranged jaws, which make a Y-shaped

incision. When leeches feed, they secrete an anticoagulant (hirudin), which helps them to obtain a full meal of blood.^{1,2} To prevent blood clotting, the saliva contains a histamine-like vasodilator and anticoagulant such as hirudin or hementerin. Cells involved in leech inflammatory response have been characterized by morphological, histochemical, and immunohistochemical methods, macrophage like cells NK like cells, and granulocytic migration. Other enzymes include (alin esterase) antitrypsin, antiplasmin and antielastase.^{3,4}

Leeches vary in shape from elongated and cylindrical to broad or ovoid. They

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may be black, brightly colored, or mottled; they have muscular suckers at both their anterior and posterior ends.⁵ Their length varies from 5-45cm.^{1,2}

Many different types of leech occur worldwide. Those that attack man may be divided into two groups: land terrestrial leech and aquatic leech.^{1,3,4,6} Aquatic leech live exclusively in fresh water.^{1,4} The leech attach to the external surfaces of the host named external leech causing external hirudiniasis, while the leech attach to the internal surfaces or mucous membrane of the host called internal leech, which causing internal hirudiniasis.⁵⁻⁷ Leeches may cause human morbidity or even mortality:

The first group; Land leech, species related to the genera *haomadipsa* and *phyrobdella* are 1-8cm long. They usually attach themselves to the lower legs or ankles and are adapted for penetrating clothes. They ingest about 1ml of blood in one hour then drop off.⁶⁻⁸

The second group; Aquatic leeches, a species related to the genera *Hirudinaria*, *limnatis* and *Derobdela* swims in water and attach themselves to the bathers. They

may enter the mouth, nostrils, eyes, vulva, vagina, urethra and anus.^{1,9,10}

The main pathological effect of the leech is the blood loss, but other manifestations include pain caused by the biting, secondary infection, itching and phobia. Leech should not be pulled off roughly when removed.

The most important step in the diagnosis of leech infestation depends on presence of leech in situ. It may present at any time and in any place. Diagnosis depends on the history of the habitation and living in the infested areas, and by examination of leech morphology which appear dark brown in color, engorged with blood and presence of fresh pink unclotted blood around the attachment site (Figure 1). Examination of the leech showed it is dark brown in color, filled with blood, and as it secretes heparin like material, there is pink fresh unclotted blood around the site of attachment.^{4,11,12}

Prophylaxis of leech infestation is by using central water supply, of filtered disinfected water, and avoidance of drinking and bathing in stream water.



Figure 1. Leech morphology

CASE PROFILE

A 45years old, multiparous woman presented to the out patient gynecology clinic, with heavy and continuous vaginal bleeding for four months, the blood was fresh without clots. She had no pain and there were no other factors for that bleeding. On examination she was very pale, tachycardia (pulse rate was 120beats/minute) with hypotension (blood pressure was 90/50 mmHg with signs of chronic iron deficiency anemia as koilonychia. Abdominal examination revealed no abnormalities. Pelvic examination using Cusco's speculum which is passing through direct anteroposterior position in the vagina, yielded fresh unclotted blood which was assessed as moderate to severe in amount, no other lesion was found in the lateral walls of the vagina or cervix. The bimanual digital examination revealed an anteverted bulky uniformly enlarged multiparous uterus with no adnexial mass.

RESULTS

Other laboratory Investigation were done, and revealed hemoglobin concentration 7gm/l, blood urea 30mg/dl, and fasting blood sugar 90mg/dl and coagulation screen was normal; blood group was A rhesus positive. Abdominal ultrasound was done which showed no abnormalities apart from just bulky multiparous uterus with normal ovaries.

The patient underwent a diagnostic curettage for this problem three months

ago; the histopathological result showed normal secretory phase endometrium. She was given a progestagen drug that made no difference.

Her anemia was corrected by giving two units of cross-matched blood. After two days under general anesthesia and through lower abdominal transverse incision, laparotomy was done; a classical total abdominal hysterectomy was done, with preservation of both ovaries. She passed motion in the second postoperative day. The first 4 days postoperative period passed smoothly with no complications. On the 5th postoperative day vaginal bleeding started which was mild to moderate in amount.

She was examined in the theater in lithotomic position, without anesthesia, under aseptic condition, when Sims speculum was passed to her vagina, there was a motile annular body of 6 cm length, dark brown in color, filled with blood, with pink fresh unclotted blood around attachment site, typical of a leech parasite, the leech was held by a sponge forceps and was pulled out gently, removal was not easy; the mass was instilled with about 5 ml (1%) Lidocaine after which, it detached itself and started movement. The mass was an aquatic leech, and was sent in a special container to the Parasitology department of the medical college, and found to be aquatic leech, a species of the genera *Hirudinaria* *Limnatis* which are endemic in north of Iraq.

The patient was discharged one day after removal of the stitches, and then, unfortunately did not return for follow up.

DISCUSSION

This pathological condition is extremely rare in urban areas, the aquatic leech have weak jaws and require soft tissue, such as the mucous membrane.⁸ Emphasis should also be made to create awareness among professionals working in areas where leech infestation is prevalent.¹³

A case with postmenopausal vaginal bleeding and hemorrhagic shock had been recorded in Ethiopia.¹⁴

CONCLUSIONS AND RECOMMENDATIONS

Leech infestation should be considered as a cause of abnormal vaginal bleeding, in endemic areas, other wise the diagnosis will be missed.

1. Stream Water should not be used for drinking or washing before space filtration and chlorination, or boiling especially in rural places.
2. In endemic areas using and sitting at riverside by women should be avoided.

REFERENCES

1. Guerrant RL, Walker DH, Weller PF. Essentials of tropical infectious diseases. Philadelphia: Churchill Livingstone; 2001.
2. Guerrant RL, Walker DH, Weller PF. Tropical infectious diseases, principles, pathogens, & practice. Philadelphia: Churchill Livingstone; 1999.
3. Warrell DA, Cox TA, Firth JD, Benz EJ Jr. Oxford textbook of medicine. 2nd ed. Oxford: Oxford University Press; 1978.
4. Husban HK, Al-Jundy AM. IMJ 1998;47(1,2,3,4):138-41.
5. Hunters SG. Tropical medicine and emerging infectious diseases. 8th ed. Philadelphia: W.B. Saunders Company; 2000.
6. Bilgen C, Karci B, Uluoz U. A nasopharyngeal mass: leech in the nasopharynx. Int J Pediatric Otorhinolaryngol 2002;64:73-6.
7. White GB. Leech infestation. In: Cook GC, editor. Manson's tropical diseases. 20th ed. London: Saunders Company; 1998. p. 1523-5.
8. Fitzpatrick TB. Fitzpatrick's dermatology in general medicine. 4th ed. McGraw-Hill Book Co; 1993.
9. Goldman Bennett G, Gill D, Kokko G, Powell M. Cecil textbook of medicine. 21 ed. Philadelphia: Saunders Company; 2004.
10. Hegner RW, Engemanr JG. Invertebrae Zoology. 2nd ed. USA: Mosby; 1968.
11. Rook A, Wilkinson D. Textbook of dermatology. 4th ed. 1986.
12. Camerson A. Haematemesi from leeches. BMJ 1950;3:679-80.
13. Maun KH. Leeches (Hirudinia): their structure, physiology, ecology, and embryology. Oxford: Pergamon Press; 1962.
14. Hailemariam B. Post menopausal vaginal bleeding due to vaginal wall leech infestation. Ethiop Med J 1955;33(4):271.

پوخته

توشبوون ب کرمی (leech) بهیته نیشاندان ب شیوی خوینرشتنی (metromenorrhagia) –
راپورتا حاله ته کی

ئارمانج: ئەو حاله ته هاته پیشکیشکر بو دیار کرنا ئەوی هندی کو توشبوون ب کرمی (leech) هیشتا نهیا به لافه ل عیراقی تایبته یا ئەندامین (تناسلی). توشبوون ب کرمی (leech) دیته ئەگه ری خوینرشتنی (menorrhagia) ل دهف نافرەتان.

پیناسه کرنا حاله تی: نه خوشه کا 45 سالی و خودان زارو و ژ دهفەرا شنگالی ل پارێزگه ها موسل سه رده دانا کلینیکا ده رفه کر بوو کو تووشی خوینرشتنه کا بۆش ژ زهاری ببوو و به ری هندی تووشی خوینرشتنه کا به رده وام و ژ ئاست نافنجی بوو بو ماوی 4 هه یغان. کورتاج بو هاته کرن پیشی هندی ب سی هه یغان و تیدا دیار بوو کو ئەو له به را دنا هه قالبچویکی یا نورمال بوو. نه خوش یا پویتی بوو و فیشارا خوینی یا کیم بوو و هه روه سا نیشاین کیم خوینی هه بوون. جی دیار نه بوو ده می هاتیه پشکنین ب ریکا سپیکولام. هه لکیشانا هه قالبچویکی بو هاته کرن دگه ل هیلانا هه ر دوو خه رزه گه هکولیا، خوینرشتن به رده وام بوو پاش نشته گه ری، هه لسانگاندن جارا کا دی هاته کرن ب ریکا سپیکولام و تیدا کرمی (leech) ل زهاری هاته دیتن.

کرمی (leech) ژ بنه مالا (Annelida) جو ری (Hirudinea) ئوردی (Rhychobdellida). توشبوون ب کرمی (leech) دقیت ژ بیرنه چیت ل نه خوشیت کو خوینرشتنا زهاری هه ی و میژوو هه ییت کو چوو به دنا ف نفا ده ریا و ربارا ل ده فەرین تووشبووی. توشبوون ب کرمی (leech) نهیا به لافه ل دهف نافرەتان و نوکه یا کیمه ژ بهر کو ئەو نفا دچیت بو باژیر و گوندا ده ربازیت دنا ف فلترو پروسپسین پاقر کرنی.

ده رنه نجام: توشبوون ب کرمی (leech) دقیت هه ر ل بیریت وه كه ئەگه ره کی خوینرشتنا زهاری ل ده فەرین کو نفا ربارا ده یته بکارئیمان و روینشتن ل لاین ربارا بویه ره وشته كه بو نافرەتا ل ده فەرین تووشبووی.

(Rhychobdellida) (Hirudinea) (Annelida)