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SPECIFIC IDENTIFICATION OF *CAMPYLOBACTER JEJUNI* USING A PCR ASSAY BASED ON THE *HIP-O* GENE

JALADET MS. JUBRAEL, PhD*
NAJIM A. YASSIN, PHD**

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ABSTRACT

Background and objectives In recent years, the use of molecular methods based on polymerase chain reaction amplification may provide an alternative to classical methods and are increasingly applied to the detection and confirmation identification of *Campylobacter jejuni* cultures directly. The aim of this study was to confirm the identification of phenotypically-identified *C. jejuni* isolates by polymerase chain reaction assays using a species-specific primer, *hipO*.

Methods From 48 previously phenotypically-identified *C. jejuni* isolates, 12 isolates were selected according to differences in their sources, biotyping and resistotyping patterns. These isolates were subjected to species-specific polymerase chain reaction assay using *hipO* primer.

Results *hipO* primer produced appropriate and successful results which yielded amplified products with all selected isolates.

Conclusions Using *hipO* primer makes further confirmation of phenotypically-identified *C jejuni* by subjection them to species-specific polymerase chain reaction assay with specificity 100%.

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Key words: *Campylobacter jejuni*, Polymerase chain reaction, *HipO* primer

Campylobacter jejuni is gram-negative, microaerophilic bacteria colonizing intestinal tract of different animals and poultry birds.[1] The most common human infections are acute bacterial gastro-enteritis. Although they are usually self-limiting in healthy adults, but complications resulting from *Campylobacteriosis* also occurred such as reactive arthritis, pancreatitis, and Guillain-Barre′ and Miller-Fisher syndrome in approximately 0.1% of all cases.[2] However, the major source of human infection is the handling and consumption of contaminated poultry meat. Other sources of infection like raw milk and untreated surface water implicated as sources or vehicles.[3]

Several phenotypic methods for typing of *C jejuni* isolates have been described. These methods include biotyping, serotyping, phage typing, and resistotyping. These methods could be challenging because of the several limitations such as lack of specific reagents, restricted differentiation power, high proportion of strains are non typeable, relative long time 5-7 days of identification , and subjective interpretation of biochemical test results. These limitations often hinder the identification of *C jejuni* from infected

** Professor, Director of Scientific Research Center, College of Sciences, Duhok University, Iraq*
*** Medical-Microbiolog dept, School of Medicine, Faculty of Medical Sciences, Duhok University, Iraq*
Correspondence author: Najm A. Yassin. Email: najim56@yahoo.com

patients, thereby obscure diagnosis and treatment.[4]

In recent years, the use of molecular methods based on PCR amplification may provide an alternative to classical methods and are increasingly applied to the detection of *Campylobacter* directly from specimens, thereby avoiding the need for culture.[5] The aim of this study was to confirm the identification of phenotypically-identified *C jejuni* isolates by PCR assays using a species-specific primer *hipO*.

METHODS

Bacterial Isolates: From June to November 2006, 572 samples were investigated which distributed between 267 samples from infant's diarrheic clinical specimens in Heevy Children Hospital in Duhok city, 177 samples from poultry cloacal swabs, and 128 samples from environmental surface water (raw water) from Duhok region. The samples were collected using clean containers with screw-cap containing Cary-Blair broth. The collected samples were inoculated in selective enriched broth (Hunt broth) containing 5% defibrinated sheep blood, *Campylobacter* selective antibiotic (Butzler's), and *Campylobacter* growth factors (FBP), and dispensed in 3ml aliquots in bottles with loosen caps. Incubate lasted for 18 hrs at 42˚C in microaerobic condition (5% O_2, 10 % Co_2, and 85% N_2) using Gas-pack generating kit (Campy-Gen) (Oxoid) (see appendix IV) in anaerobic jar. One-two loopful of enrichment broth was streaked onto two selective agar medium: The first medium was brucella agar supplemented with 5% sheep blood, *Campylobacter*-growth factors (FBP) and *Campylobacter* selective antibiotic (Butzler's). The second medium was charcoal cefoperazon agar. Both media were incubated in microaerobic condition in anaerobic jar at 42˚C for 2-3 days. The growths identified after 48-72 hrs of incubation periods, and suspect colonies were identified as a *Campylobacter jejuni* according to.[6]

Genomic DNA Extraction from Pure Cultures: For genome-based identification of *C jejuni*, out of 48 phenotypically-identified *C. jejuni* isolates 12 isolates were selected according to differences in their sources as shown in table 1. The selection of 12 samples was to represent overall pictures of *C. jejuni* collected and subjected to PCR**.** Genomic DNA preparation of *C. jejuni* isolates from pure cultures was done as described by Lind et al[7] with minor modifications, briefly; a single colony of *C jejuni* was inoculated into 30 ml of the brain heart infusion broth and incubated overnight at 42˚C under microaerobic conditions using Gas-pack kit generator in anaerobic jar. Cells were harvested by centrifugation at 3000rpm for 30mintes. The pellet was re-suspended in 540 µl 0.01M Tris-EDTA (TE) buffer (pH 8.0) mixed well and 100 µl of Lysozyme enzyme (5 mg/ml) was added. A 60 µl volume of 10 % (SDS) sodium dodecyl sulfate was added, and the solution was incubated at 37˚C for 15mints. A 150µl of a solution containing (25mg/ml) of proteinase K per ml was added, and the combination was mixed and re-incubated for one hour at 37˚C. Centrifuge at 10,000

rpm for 15mints, 700 µl of the supernatant was transferred to another Eppendorf tube. Repeat the step of proteinase K but incubation at 37˚C for 15 mints. For DNA extraction, 700 µl of phenol was added and the mixture was mixed by inverting them several times for 30mints and then centrifuge at 10,000 rpm for 5 mints. Approximately 75% of the viscous supernatant was transferred to a another new Eppendorf tube, an equal volume of chloroform-isoamyl alcohol was added and the mixture was mixed by inverting them several times for 30mints and then centrifuge at 10,000 rpm for 5 mints. Repeat the later step. The supernatant was again transferred to another fresh Eppendorf tube. The supernatant was made 1 M by adding 5 M NaCL, and the DNA was precipitated with 2 volumes of 96% ethanol for 30 mints or overnight at –20˚C.Centrifuge at 10,000 rpm for 10mints, the pellet was suspended in 100 µl TE buffer (pH 7.6) and stores at freeze temperature and used as template DNA.

Table 1. Diversity of C jejuni isolates from various sources selected for species-specific PCR assay

No. of Isolates	Source
4	Surface raw water
4	Poultry cloacal swab
4	Infant feces specimens
12	Total

Species-Specific PCR Amplification: The elements in table 2 represent the components required for species-specific PCR reaction. Master reaction was prepared for 13 samples by using Eppendorf tube (1.5 ml) by mixing 30 µl of 10X PCR buffer with 30 µl dNTPs, 24 µl from each primer (table 3) and 2.4 µl of *Taq* polymerase enzyme, the volume was made up to 276 µl by deionized distilled water(all these steps were done on ice). Twenty three µl (23 µl) of master mix reaction was added into each Eppendorf tube (0.5 ml) then, 2 µl of DNA of each sample was added to these tubes individually and mixed gently. Thus, the final volume of each tube was 25 µl as shown in table 2.

Table 2. Components required for Species-Specific PCR reaction

Additional Order	Component	Volume	The final concentration
1	10X PCR buffer	2.5 µl	1X
2	dNTPs	2.5 µl	0.2 mM
3	Forward primer	2 µl	10 Peco-mol
	Reverse primer	2 µl	10 Peco-mol
4	*Taq* polymerase	0.2 µl	1 U
5	deionized distilled water	13.8µl	-
6	DNA	2 µl	25-50 ng
Total		25µl	

Table 3. Sequences of hipO primer

Primer	Sequences
hipO **Forward 735**	**5-GAA GAG GGT TTG GGT GGT-3**
hipO **Reverse 735**	**5-AGC TAG CTT CGC ATA ATA ACT TG-3**

The reaction mixture in each tube was overlaid with 25 µl of mineral oil to prevent evaporation. All these tubes were transferred to thermal cycler to start the amplification. The amplification program was run as follows:

Number of Cycles	Temperature° C	Duration (minutes)
1 (denaturation)	94°C	3
30 (denaturation)	94°C	1
30 (annealing)	57°C	1
30 (extension)	72°C	1
1 (extension)	72°C	5

Then the amplified DNA was subjected to 1.4% agarose gel electrophoresis, stained with ethidium bromide, and viewed under UV (366 wavelength) and photographed with digital camera.[8]

RESULTS

Genomic DNA Isolation from Pure Bacterial Cultures: Genomic DNA was isolated from pure cultures of *C jejuni* according to the method described by.[7] Suitable yields of genomic DNA were obtained from repeated experiments with an average yield of 3.34-6.10 µg/ml with a purity about (1.7-1.8) determined by spectrophotometer ratio A 260/A 280. The molecular weight of DNA samples was estimated using 1% agarose gel electrophoresis containing ג DNA sample as control, and it was found to be 50 Kb (Figure 1).

Species-Specific PCR Assay: Species-specific PCR assay using *hipO* (735bp) specific primer was used in the present study. The agarose gel electrophoresis of amplification products with *hipO* gene are shown in the figure 2. Examining this figure, an amplicon sized 735 bp may clearly be seen across all 12 samples of phenotypically-identified *C jejuni* isolates.

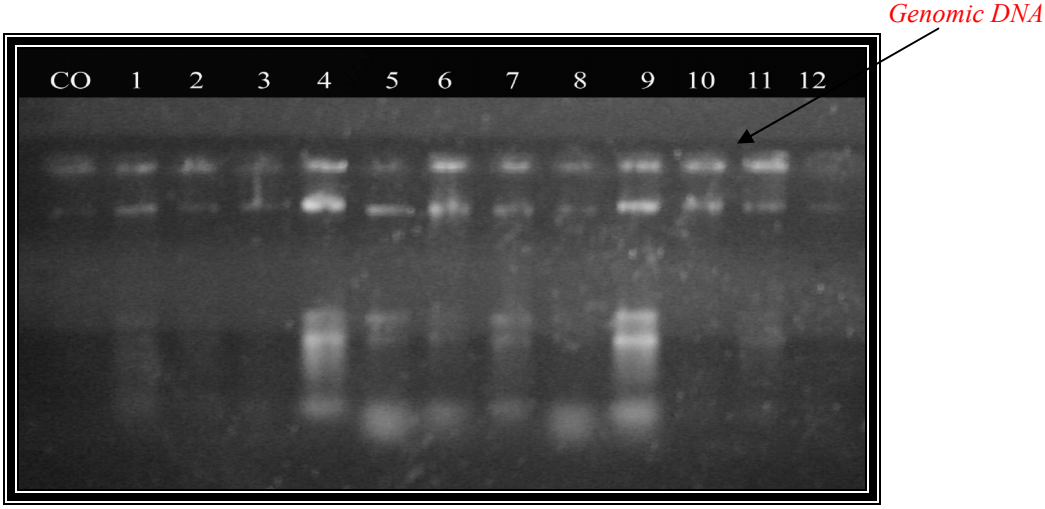

Figure 1. Genomic DNA isolated from twelve isolates of C jejuni collected from different sources. Electrophoresis was performed on 1% agarose gel and run with 3 volt/cm for 3hrs. The lanes 1, 2, 3, and 4 represents C jejuni strains isolated from surface raw water samples whereas lanes 5, 6, 7, and 8 represents C jejuni strains isolated from poultry cloacal swabs, lanes 9, 10, 11, and 12 represents C jejuni strains isolated from infant feces specimen. Lane CO represents undigested lambda ג DNA marker as standard molecular weight marker

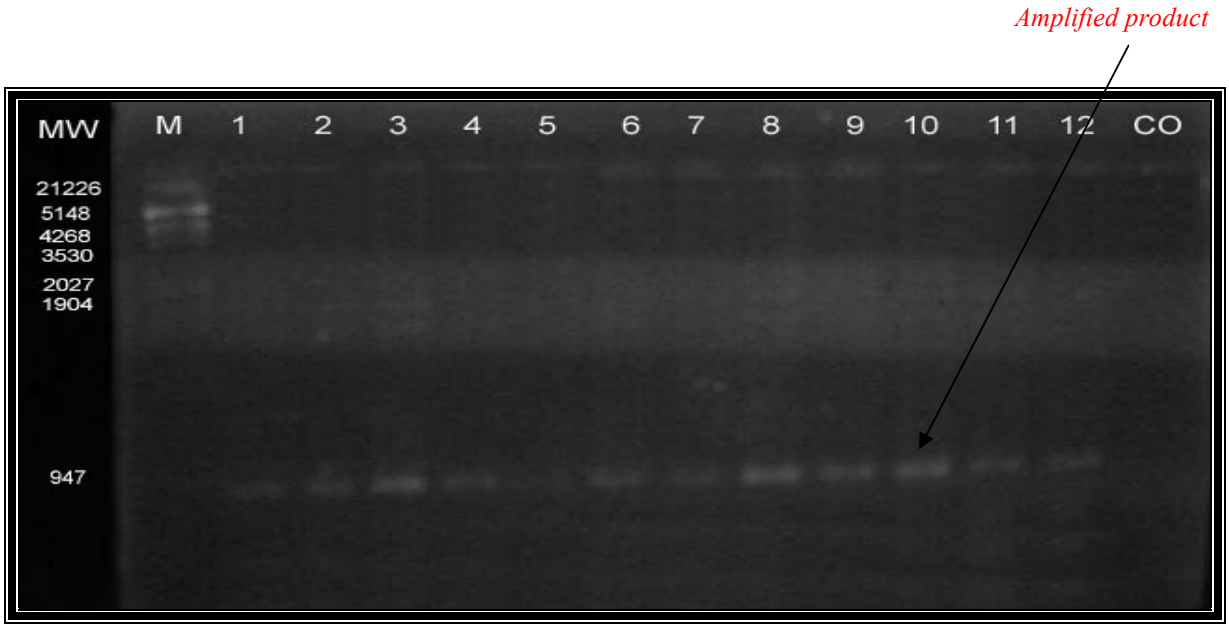

Figure 2. Species-specific PCR amplification of genomic C jejuni isolates obtained with specific primer hipO. Electrophoresis was performed on (1.4%) agarose gel and run with 3 volt/cm for 3 hrs. The lanes 1, 2, 3, and 4 represents C jejuni strains isolated from surface raw water samples whereas lanes 5, 6, 7, and 8 represents C jejuni strains isolated from poultry cloacal swabs, lanes 9, 10, 11, and 12 represents C jejuni strains isolated from infant feces specimen. Lane Co represents the control sample. Lane M indicates the Eco RI and Hind III digested lambda ג DNA marker

DISCUSSION

Extraction buffer contained EDTA, as a chelating agent can bind the divalent ions, like magnesium Mg^{+2}, that are required for activity of most DNAase, and thus reducing the nuclease activity.[11] Sodium dodecyl sulfate (SDS) was also used as strong proteins degradation agents. Saturated phenol acted as an efficient organic solvent capable of protein denaturation and other cell materials.[12] Chlorophorm-isoamyl also acted as further organic solvent but in addition to that leaves nucleic acid in aqueous phase as well as remove phenol residue from solution which affect DNA polymerase activity.[9] The latter nucleic acid was precipitated addition of NaCL (monocationic) and absolute ethanol in precipitation of DNA.[7] The total nucleic acids extracted from *C jejuni* strains tested included also RNA. To remove RNA and obtain only DNA, the samples were usually subjected to RNase enzyme treatment and then further phenol-chlorophorm purification will be required. However, as PCR technique does not require the elimination of RNA since, RNA is a single strand molecule which will completely be denaturated by heat in denaturation step of the first PCR cycle. RNA does not have annealing site to primers used, therefore, it will not be amplified.[9]

The agarose gel electrophoresis of amplification products with *hipO* gene yielded an amplicon sized 735 bp may clearly be seen across all 12 samples of phenotypically-identified *C jejuni* isolates. Detection of this band may provide an effective identification marker for this

pathogen and confirm the phenotypic results, especially certain strains that may failed to do phenotypically hippurate hydrolysis test, which misclassified as *C coli*.[13] It has been reported that the detection by PCR assay of *hipO* gene was shown to be highly conserved in *C jejuni* genome and provided an effective species-level identification marker for this pathogen. [14] Another study stated that the *hipO* could differentiate *C. jejuni* from culture-positive non-*C jejuni*. [15] Other study announced that the PCR assay targeting the *hipO* gene could differentiate between *C jejuni* and *C coli* strains from human and poultry origin, but *flaA* primer failed to do so. [6] Nevertheless, mutation in *hipO* gene was previously been identified as a source of failure for the PCR assay targeting this gene.[16]

This study concluded that the PCR assay based on *hipO* primer confirms identification of phenotypically-identified *C jejuni* by conventional methods. The results highlighted the species-specific PCR assay which was found to be sensitive, fast and reliable that may act as an appropriate supplementary method for identification of *C jejuni*. Therefore, the simplicity and speed of this approach make it indeed highly applicable for early routine detection of *C jejuni* directly in clinical specimens, foods, and sewage water, notably in screening large numbers of samples in endemics outbreaks.

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پوختە

ناسینا تایبەتا کەمپیلوبەکتر جوجونی *Camplyobacter jejuni* ب تێستا PCR لسەر بنەمایێ جینا *hip0*

پێشەکی و ئارمانج: ل سالێن بوری دا، بکارئینانا شێوەیێن مولکیولی لسەر بنەمایێ بهێز کرنا PCR رەنگە شێوەیێن بەرگوهێر حەتا کلاسیکی دروست بکەت و ب بەرفرەهی بو ناسین و تەکەز کرنا چاندێن کەمپیلوبەکتر جوجونی ب شێوەیەکێ ئێکسەر دهێنە ب کارئینان. ئارمانجا ڤێ ڤەکولینێ تەکەز کرن لسەر ناسینا کەمپیلوبەکتەر جوجونیا فنوتوپیکە کو ب تێستێن PCR و پرایمرا تایبەتا hip0 هاتیە جوداکرن.

ریکێن ڤەکولینێ: ژ 48 جوداکریێن کەمپیلوبەکتەر جوجونی یێن فنوتوپیکین بەرێ، 12 جوداکری ل دەڤ جوداهییێن ژێدەران، نمونەیێن زندی و خوراگر هاتنە نیاسین، ئەڤ جوداکریە پرایمرا hip0، ل تێستا PCR یا تایبەت هاتنە کونترولکرن.

ئەنجام: پرایمرا hip0، ئەنجامێن سەرکەفتی و باش بدەستئینان کو بەهەمێن بهێز دگەل هەمو جوداکرین هەلبژارتی بدەست هاتن.

دەرئەنجام: ب پرایمرا hip0، تەکەزیەکا پتر بو کەمپیلوبەکتەر جوجونی یا فنوتوپیك بدەست هاتن، کو ئەڤ تەکەزیە ب کونترولکرنا وان ل تێستا PCR یا سەداسەد دهێتە دەستەبەرکرن.

الخلاصة

التشخيص الدقيق لبكتريا *Campylobacter jejuni* باستخدام تقنية تفاعل التضاعف المتسلسل للحامض النووي أعتمادا على جين *hipO*

خلفية واهداف البحث: في السنوات الحديثة بدأ استخدام التقنيات الجزيئية بلأعتماد على تفاعل التضاعف المتسلسل للحامض النووي عوضا عن الطرق التقليدية في مجالات تشخيص البكتريا ولاسيما في تحديد وتشخيص التأكيدي لمزارع لبكتريا *Campylobacter jejuni*. الهدف من هذا البحث هو لتطبيق التقنية التضاعف المتسلسل للدنا DNA (Polymerase chain reaction) باستخدام البادئ المتخصص(*hipO*) من أجل التشخيص الدقيق والتأكيدي ولتحديد الهوية النوعيه للجرثومة *C jejuni* المشخصة سابقا بالطريقة التقليدية والمأخوذة من المصادر المختلفة وذات أنماط حيوية ومقاومة مختلفة.

طرق البحث: شملت هذه الدراسة انتخاب اثنا عشر عزلة من جرثومة *C jejuni* المشخصة سابقا بالطريقة التقليدية والمأخوذة من المصادر المختلفة وذات أنماط حيوية ومقاومة مختلفة و تم عزل الدنا الجيني DNA اولا من جميع العزلات تم اخضاعها لتقنية التضاعف المتسلسل للدناDNA (polymerase chain reaction) باستخدام البادئ المتخصص(*hipO*) من أجل التشخيص الدقيق والتأكيدي ولتحديد الهوية النوعيه للجرثومة C jejuni.

النتائج: أعطت جميع العزلات (12) المنتخبة من جرثومة *C jejuni* باستخدام البادئ المتخصص(*hipO*) نتائج تضاعف دقيقة وتفاعلا موجبا تجاه تقنية التضاعف المتسلسل للدنDNA (Polymerase chain reaction).

الاستنتاجات: ونستخلص من هذه الدراسه أن تقنية تضاعف سلسلة الدنا وباستخدام البادئات المتخصصة تكون حساسة و سريعة و يعتمد عليها ومن الممكن أن تكون طريقة مناسبة و على الأقل طريقة ثانوية ومساعدة خصوصا عندما تكون نتيجة الزرع سالبا .ويستفاد منها ايضا لتقصي المسببات الجرثومية اثناء حدوث التفشيات الوبائية.

NOISE INDUCED OXIDATIVE STRESS AND HEARING LOSS IN ELECTRICAL GENERATOR WORKERS

RAED S. AL-NAEMI, MSc, PhD*
TARIK A. ABDAL, MSc**

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ABSTRACT

Background and objectives This work was designed to study the effect of prolonged exposure to high noise thresholds on oxidative stress status and hearing ability in large private electrical generators workers.
Methods This study was carried out on 89 adult males. Sixty of them were electrical generator workers and 29 healthy control subjects. The intensity of noise inside and outside the electrical generators rooms was estimated. In both groups, hearing ability, blood pressure, body mass index, serum total antioxidants, malondialdehyde and lipid profile were measured.
Results A significant decrease in serum total antioxidants and a significant increase in serum malondialdehyde when compared to healthy control group. Significantly higher values of mean serum cholesterol, triglycerides, and very low density lipoprotein-cholesterol, body mass index and systolic blood pressure were observed in workers compared to the control group. However, mean values of high density lipoprotein-cholesterol were found to be significantly lower among workers than in the control group. Audiometric study revealed that, hearing abilities were significantly decrease among the workers at frequencies 250, 500, 1000, 2000, 4000, 6000 and 8000 Hz. However, no significant difference was reported between the two groups at frequency 125 Hz.
Conclusions The level of noise exposure in electrical generator workers of Duhok city was high and exceeding the 85dB allowed by municipality. Noise inducing imbalance oxidative stress status and impaired hearing in these workers was reported and got more evident when the employment duration increased.

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Key words: Noise, Oxidative stress, Hearing loss, Audiometry

Noise is derived from the Latin word "nausea" implying unwanted or undesirable sound, or sound that is loud, unpleasant or unexpected, which disturbs the human beings physically and physiologically causing environmental pollution and producing reactive oxygen species (ROS) that cause damage to all vital organs.[1-4] Noise induced hearing loss (NIHL) has been known since the beginning of written history, and thus deafness is found early in the first century A.D., among people who lived near the Nile falls.[5] The most common reason of sensory-neural hearing impairment is the noxious stimuli.[6] The noxious stimuli was thought to cause metabolic exhaustion and results in an overproduction of waste products, ROS in the cochlea, which in turn could cause oxidative stress damage to vital hearing structures and tissues.[7] The noise level and exposure time are the key

*Assistant Professor, Medical Physiology, Department of Physiology, School of Medicine, Faculty of Medicine, University of Duhok
**Assistant Lecturer, Medical Physiology, Department of Physiology, Faculty of Veterinary Medicine, University of Duhok
Correspondence: Raed S. Al-Naemi. E-mail: aosraid@yahoo.com. Mobile: 00964 750 411 5262

determents for how noise affects hearing, and is also the basics for the two different ways that noise can destroy the hearing organs. The first is the high level, short noise exposure time that can stretch inner ear tissues and structure beyond their elastic limits, thus tearing them apart. The second is the low level, long duration noise exposure time can fatigue the ear's delicate tissue.

Overstimulation by intense of sound gives rise to several structural and functional changes in the organ of Corti, these changes include shrinking of the tectorial membrane,[8] disruption of the tip-links of the stereocilia,[9] fracture of the actin core and bending of the stereocilia,[10] shortening and swelling of outer hair cells (OHCs) bodies,[11] swelling of the afferent nerve endings,[12] and degeneration of afferent neurons.[13] The objective of this work was to study the effect of prolonged exposure to high noise thresholds on oxidative stress status as assessed by serum total antioxidants (TAS), malondialdehyde (MDA) and lipid profile in workers of large private electrical generators in Duhok city.

METHODS

This study was conducted at the Department of Physiology, College of Medicine, University of Duhok and Azady teaching hospital in Duhok city, one of the main cities in Kurdistan Regional Government of Iraq. The study period was from first of October, 2009 to fifteenth of June 2010. The study protocol was approved by Post-graduate Committee of Faculty of Medicine/ Duhok University.

This study was carried out on (60) male electrical generator workers (GWs), who worked inside large private electrical generator (LPEG) rooms. A total of 77 LPEG rooms or stations present inside Duhok City. The inclusion criteria for GWs were apparently healthy males with history of no medical treatment, less than 40 years aged, who has been working at LPEG for > one year and exposed to 4 hours or more/day.

The control group was chosen as apparently non smoker, who are not alcohol drinker males, and who were usually far away from any high level of noise stress in their working and living places. The intensity of noise was recorded inside and outside the electrical generator room by using a CEL-254 Digital Sound Survey Meter. A Beltone-model 119 Pure Tune Audiometer was used in this study to estimate the changes of both ears in pure-tone thresholds at 125 to 8000 kHz for both GWs and control groups**.** Blood pressure, body mass index (BMI), serum total antioxidants (TAS), oxidative stress biomarker; malondialdehyde (MDA) and lipid profile were estimated. Smoking and alcohol drinking were also considered. The cutoff values of oxidative stress and total antioxidants were calculated according to the previous study done in Duhok city[14] and for blood pressure using other reference values.[15] The standard audiogram evaluation data of Pallant was used as a reference scale measure of the level of hearing losses.[16]

The statistical analysis was done using Graph Pad software Prism.[17] The level of

significance was determined when P≤ 0.05.

RESULTS

The GWs ages ranged between 17- 39 years (mean ± SD = 27.42 ± 6.46), BMI ranged between 21.27 to 31.55 kg/m^2 (26.41 ± 5.14), systolic blood pressure (SBP) ranged from 120.29 to 136.61 (128.45 ± 8.16) mm Hg, and diastolic blood pressure (DBP) ranged 72.72 to 89.54 (mean ± SD = 81.13±8.4) mm Hg (Table 1). Smoking habit was present in 58.33% of GWs (total smokers number = 35) and all were non alcohol drinkers.

In the control subjects, the age ranged between 21.03 to 29.17 years (25.10 ± 4.07), BMI ranged between 21.14 to 27.06 (24.10±2.96) SBP ranged from 107.4 to 125.8 (116.6 ± 9.21) mm Hg, and DBP ranged from 72.3 to 86.5 (mean ± SD =79.4 ± 7.1) mm Hg. Significant differences were found between the two groups regarding BMI and SBP parameters (Table 1). The intensity of noise inside LPEG rooms were (95.18± 0.87 dB) compared to (90.80 ± 0.94 dB) outside the generator rooms i.e. at the worker resting rooms (Figure 1). Only the data about the right ear were used in this study since the results of paired t-test analysis reveal that there was no statistically significant difference at all frequencies between the two ears in terms of hearing losses of the GWs and the controls. Significant differences were reported between the worker and control groups at the frequencies 250, 500, 1000, 2000, 4000, 6000 and 8000 Hz. Whereas no significant difference was reported between the two groups at frequency 125 Hz, as shown in table 2. According to the employment duration and hearing ability test, the GWs were classified into five groups (Table 3). Group1 (GWs with normal hearing), group 2(GWs with mild hearing loss), group 3(GWs with moderate hearing loss), group 4 (GWs with sever hearing loss) and group 5 (GWs with profound hearing loss). The percentages of the GWs in the five groups were 21.7%, 48.3%, 20.0%, 5.0% and 5.0 % respectively, which showed that, impaired hearing functions started in GWs within the first four years of their starting work with LPEG and got more evident when the employment duration increased.

Table 1. The mean ± SD of the Age, BMI, systolic and diastolic blood pressure and presence of smoking habit in GWs and control groups

Parameters	Generator Workers (n= 60) mean±SD	Controls (n=29) mean±SD	Unpaired Student t-test t-value
Age (years)	27.42 ± 6.46	25.12 ± 4.07	1.76*
BMI (kg/m^2)	26.41 ± 5.13	24.10 ± 2.96	2.08**
SBP (mmHg)	128.45 ± 8.16	116.56 ± 9.20	5.89***
DBP (mmHg)	81.13 ± 8.4	79.4 ± 7.12	0.90*
Smokers number (%)	35(58%)	0	-

non significant (P>0.05) **significant(P≤0.05) * significant(P≤0.001)*
SBP = systolic blood pressure DBP= diastolic blood pressure

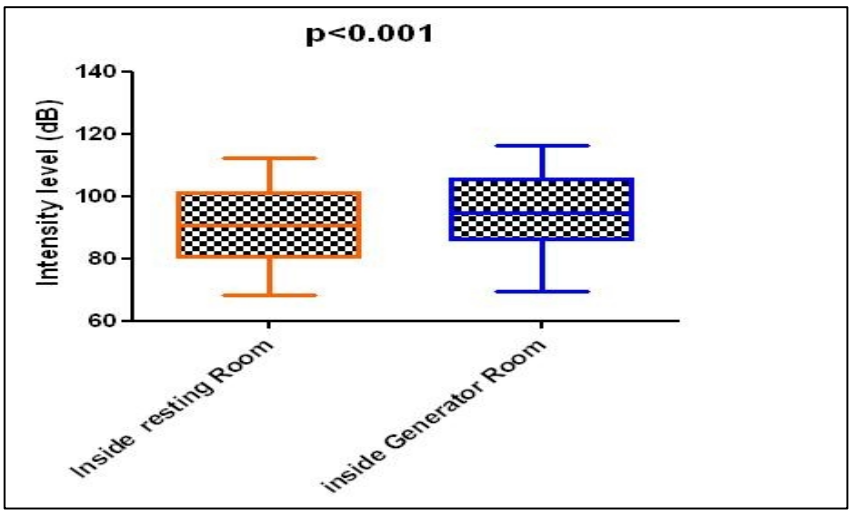

Figure 1. Intensity of noise exposure inside the electrical generator room and outside in the workers resting room

Table 2. Audiometric thresholds of hearing frequencies of generator worker and control groups

Frequency (Hz)	Generator Workers (n= 60) mean±SD	Controls (n=29) mean±SD	Unpaired Student t-test t-value
125	21.83 ± 2.16	16.03 ± 0.79	1.83*
250	24.50 ± 1.97	15.86 ± 0.86	2.97**
500	29.48 ± 2.12	17.24 ± 0.64	3.96**
1000	31.7 ± 2.08	9.66 ± 0.89	7.19***
2000	32.92 ± 2.18	5.69 ± 1.10	8.40***
4000	34.42 ± 2.87	9.14 ± 1.13	5.99***
6000	40.00 ± 2.82	11.21 ± 1.23	6.93***
8000	33.92 ± 3.27	10.34 ± 1.16	4.93***

non significant (P>0.05)* ***significant(P≤0.05)* ** significant(P≤0.001)*

Table 3. Evaluation of hearing ability in generator workers divided according to the standard reference evaluation values

WGs divided according to the hearing ability* (n=60)	Number (%)	Employment Duration (years) mean ± SE
Group I (with normal hearing)	13(21.7)	2.07 ±0.32
Group II (with mild hearing loss)	29(48.3)	4.17 ± 0.73
Group III (with moderate hearing loss)	12(20.0)	6.39 ±1.16
Group IV (with sever hearing loss)	3(5.0)	9.5 ± 0.5
Group V (with profound hearing loss)	3(5.0)	14.5 ± 3.5

**Evaluation of hearing ability done according to the reference value[16]*

According to the employment duration of noise exposure with the LPEG, the GWs were classified into three groups (Table 4): group I included 28 workers (46.67%) with the employment duration > 1 and < 3years (1.33 ±0.08). Group II consisted of 14 workers (23.33%) with the employment duration ≥3 and < 5years (3.57±0.13). Group III composed of 18 workers (30%) with the employment duration ≥ 5 year (9.38±0.65). Significant differences were observed between the 3 groups at the hearing thresholds frequencies 125,1000 and 2000 Hz. Significant lower TAS values were observed in GWs than in the control group ($p<0.01$). The mean values ± SE of TAS in serum of GWs and control were 983.6 ± 24.09 µm/L and 1110 ± 13.41 µm/L respectively. The level of serum MDA was found to be significantly higher ($p< 0.05$) in GWs (mean ± SE =1.12 ± 0.12 nm/L) when compared to the control group (mean ± SE = 0.77 ± 0.03 nm/L), (Figure 2). Significant higher values of total serum cholesterol, triglycerides, and very low density lipoprotein-cholesterol (VLDL-C) were observed in GWs compared to control group (Table 5). However, the high density lipoprotein-cholesterol (HDL-C) was found to be significantly lower in GWs than in the control group. The abnormal values of serum TAS and MDA with other abnormal measurable variables were calculated from the cutoff values for all GWs (including the smoker group). Accordingly, smoker GWs were found to have more risk of imbalanced oxidative stress status, abnormal lipid profile and blood pressure measurements (Table 6).

Table 4. Hearing thresholds at different frequencies in the three groups of GWs divided according to employment duration

Frequency (Hz)	Hearing thresholds (dB)			ANOVA Test
	Group I (n=28)	Group II (n=14)	Group III (n=18)	
	mean ± SE	mean ± SE	mean ± SE	F-value
	A	AB	B	
125	19.66±2.30	18.42± 3.39	32.31± 6.83	3.33**
250	31.72±2.06	39.47±4.74	34.61±5.14	1.31*
500	22.59±2.29	23.42±4.22	31.92±4.36	1.82*
	A	AB	B	
1000	28.79±1.82	30±3.33	41.15±6.89	3.05**
	A	AB	B	
2000	28.79±2.03	31.84±3.45	43.85±6.89	4.05***
4000	32.93±3.71	30±4.52	47.69±8.14	2.74*
6000	35.52±3.25	5±7.59	46.05±5.79	1.56*
8000	32.61±4.59	6.15±10.33	40± 8.86	4.57***

**non significant (P>0.05) **significant(P≤0.05) ***significant(P≤0.01)*

• (A, B, AB): letters of Tukey's statistical test done for significant F value only

• Tukey's same letters (e.g. A, A) mean no difference present while that for different letters (e.g. A, B) mean differences are present

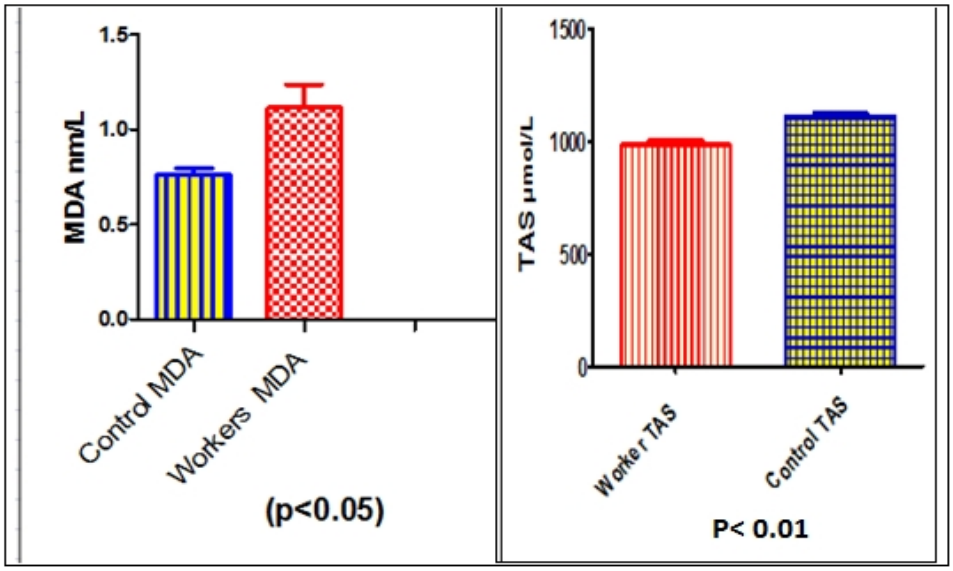

Figure 2. Serum total antioxidants and MDA levels among generator worker and control groups

Table 5. The mean ± SE of serum cholesterols, triglyceride, HDL and VLDL in generator worker and control groups

Serum lipid	Normal reference range•	Generator Workers (n= 60) mean±SD	Controls (n=29) mean±SD	Unpaired Student t-test t-value
Cholesterols (mg/dl)	140-240	167.3 ± 3.41	152.3 ± 6.31	2.23*
Triglyceride (mg/dl)	40- 150	109.7 ± 7.11	74.96 ± 4.96	2.96**
HDL-C (mg/dl)	35-55	32.42 ± 1.86	41.46 ± 2.76	2.77**
VLDL-C (mg/dl)	<40	22.94 ± 1.95	15.00 ± 1.01	2.51*

**significant(P≤0.05) ** significant(P≤0.01)*

• Reference values of the standards samples supplied with the specific kits provided for biochemical auto-analyzer machine

Table 6. The cutoff values of serum TAS, MDA, numbers and percentage of generator workers with abnormal measurable data for lipid profile and high blood pressure

Variables	GWs Total Number (%)	Smokers Number (%)
TAS ≤ 830 µm/L*	7 (11.7)	5 (8.3)
MDA ≥ 1.4 nm/L*	30 (50)	13 (20)
Hypercholesterolemia > 240 mg/dl	5 (8.3)	1(1.7)
Hypertriglyceridemia > 150 mg/dl	13 (20)	10 (15.4)
High VLDL >40 mg/dl	6 (10)	6 (10)
Low HDL cholesterol < 35 mg/dl	24 (40)	13(20)
High SBP >140 mmHg**	7 (11.7)	5 (8.3)
High DBP> 90 mmHg**	4 (6.7)	0 (0)

** The reference cutoff values of the previous study in Duhok city[14]*

*** Other references values from auto-analyzer standard kits and for abnormal blood pressure measurements[15]*

DISCUSSION

Acute and chronic loud noise exposure generate excessive free radicals and cause disorders that involve extra-auditory organs such as neural cells, endocrine functions and cardiovascular homeostasis.[18] Although normal speech occurs at approximately 45 dB, annoying will occur at level exceeds 75 dB.[19] Our work demonstrated that, the daily noise exposure (> 95dB for > 1year) significantly decreased hearing ability in GWs ; by comparing their hearing measurable data with those of the controls. Remarkable hearing losses at high frequencies were found in subjects working in Turkish textile factory.[20] They found that hearing loss occurred with intensity of noise about 95 dB or more and for noise exposure duration periods from 5-8 years. The same finding was recorded in the forest workers who were exposed to high levels of noise (>95 dB) during forestry activities when using a chainsaw machine operator.[21] It was found that the hearing loss occurred at 500, 2000 and 4000 Hz frequencies but the most specific difference observed among forestry chainsaw machine operators at 4000 Hz frequency. Therefore, they named the 4000 Hz as 4000 Hz dip, which is the primary indicator of sensory-neural hearing loss which develops as a result of noise exposure. Our results revealed that hearing loss levels in the two ears were similar to each other (no significant differences present between the two ears). The same previous researchers concluded that noise induce hearing loss was mostly bilateral and showed a similar pattern in both ears.[21] The results of the evaluation of hearing ability in GWs compared with the normal control group significantly decreased at all frequencies (250, 500, 1000, 2000, 4000 and 8000 Hz) while no significant difference at 125 Hz frequency was found between the two groups. These results provide clear evidence that high noise level can produce hearing loss by acute and chronic changes to the ear structures and functions in GWs. In the present study, also a comparison between the three subgroups of GWs, who were classified according to the employment duration of the noise exposure, was done. The most affected subgroup of GWs with hearing loss was a GWs with employment duration ≥ 5 year, followed by those with employment duration ≥3 and <5 years and then with >1 and <3 years respectively. A similar finding was also recorded among textile workers.[20] The audiogram results were analyzed and revealed that the mild hearing losses among the GWs started within the first four years of working period inside LPEG rooms, and progressed into moderate, severe and profound hearing loss when the working period increased. The impact of hearing loss gradually occurred due to acute or chronic exposure to high intensity of noise leading to decrease hearing ability. Recently, it was assumed that the contributing factor in noise induced hearing loss is the cochlear and hair cell injuries ,occur by glutamate excitotoxicity or synaptic exhaustion.[4] The recovery of both hearing cells and their toxic environment (high glutamate and ROS production) may need prolong time,

i.e. about 7-10 days after cessation of noise exposure.[22] However, the GWs in this study had only 3 resting days during each month, so there is no complete recovery and hearing ability may be more progressed toward hearing loss. The level of noise exposure of electrical generator workers in Duhok city was found to be high; it may reach to the cellular damaging level inside and outside the LPEG rooms with absence of using any type of hearing protection measures. However, noise laws and ordinances of Kurdistan cities municipalities contain the level of noise allowable (< 85dB) during daylight and (< 70dB) at night. This indicates the importance in passing a obligatory legislation for using protective measures. The induction of oxidative stress by noise exposure with expected unhealthy nutritional status, inhalation of harmful toxic gases from generator machines and smoking effect in the GWs may contribute in elevating systolic blood pressure, which is a cardiovascular risk factor, especially in the presence of other dyslipidaemic risk factors including hypercholesterolemia, hypertriglyceridaemia, and decreased HDL.

Oxidative stress species induced by prolonged noise exposure, have a negative effect on hearing functionality and cause damage to vital tissues and structures of the cochlea. As a consequence of this degeneration, hearing is impaired, which results in elevated hearing threshold.

The Ministry of Health should inform all owners of the industrial factories and electrical generators about the harmful effects of the noxious sounds on human health and the number of resting days, as a recovery period, must be 7-10 days per month for all workers who are working in a nuisance environment with the imposing of using hearing protective measures.

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پوختە

ژاوژاو دبیتە ئەگەرێ نەهەڤسەنگیا بارێ ماندیبوونا ئوکسانی و ژدەستدانا بهیستنێ لدەڤ گرنگیا موەلیدێن ئلکتریکێ

پێشەکی و ئارمانج: گەلەك كەس توشی ژاوژاوێ دبن ئو ئەڤ توشبونە دبیتە ئەگەرێ ئاریشێن ساخلەمی پێن جورەوجور وەك ژدەستدانا بهیستنێ ژ ئەگەرێ ژاوژاوێ. ئەف كارە هاتیە كرن بو دیاركرنا كاریگەریا توشبونا ژاوژاوێ ب ئاستێن بلند و بو دەمێن درێژ لسەر بارێ ماندیبونا ئوكسانی و شیانا بهیستنێ لدەف كرێكارین موەلیدێن ئلەكتریكێ.

ریكێن ڤەكولینێ: ئەف خویندنە لسەر89 كسێن نێر پێن پێ گەهشتی هاتەكرن. شێست (60) ژوان كرێكارین موەلیدێن ئلەكتریكێ ئو 29 كەسێن ساخلەم ئەوێن نە لبن ماندیبونا ژاوژاوێ لجهێن كاری و لمالێن خو. دژواریا ژاوژاوێ دناڤ و ژدەرڤەی ژورێن كرێكارا هاتە پیڤان. لهەردوو گروپان شیانا بهیستنێ، فشارا خوینێ، بارستەی لەشێ مروفی، malondialdehyde، serum total antioxidants، ئاستێن چەوری د خوینێ هاتنە پیڤان.

ئەنجام: شلوڤەكرنا سەرژمێریاریێ دڤی كاریدا جێوازیەكا بەرچاڤ دیاركر دەربارەی بارستەی لەشێ مروفی و فشارا خوینێ یا سیستولی. نیشاندەرێن ماندیبوونا ئوكسینی ئان كو كێمبونا (serum total antioxidants) و زیدەبوونا (serum malondialdehyde) د كریكاران دا ب شێوەكێ بەر چافتربو ژ گروپێ دابینكری یێ ساخلەم دا. زیدە بونا بەرچاڤ د كولیسترول، چەوریێن سیانی VDRL هانە دیتن دكرێكاراندا دەمێ هاتینە بەراوردكرن دگەل گروپێ دابینكریدا. بەڵێ یا (high density lipoprotein-cholesterol) بەرزتربوون دگروپێ دابینكریدا ژ گروپێ كرێكارا. شلوڤەكرنا ئودیومیتری دیاركر ژ دەستدانا بهیستنێ یا جێوازبو دناڤبەرا كریكاران و كەسێن ساخلەم د لەرەیێن (250، 500، 1000، 2000، 4000، 6000، 8000) هرتز دا بەڵێ چ جوداهیا مەزن دناڤبەرا هەردوو گروپادا نەبۆ د لەرەیا (125) هرتز.

دەرئەنجام: ئاستێ توشبوونا ژاوژاوێ یا كرێكارین موەلیدێن ئلەكتریكێ ل دهوكێ زوور بەرزە (دگەهتە ئاستێ كوژەك بو شانێن گوهی 95 ≤) دناڤ و دەرڤەی ژوورێن كرێكاران دا دگل نەبوونا بكارئینانا چ تشتا بۆ پاراستنا گوها . ماندیبوونا ئوكسینی ژ ئەگەرێ ژاوژاوێ كو بویە ئەگەرێ ژ دەستدانا بهیستنێ ل دەف كریكاران. ژ دەستدانا بهیستنێ دەست پێكر ژ چار سالین دەست پێكی و زێدە دبوو دەمی ماوێ دەمێ خزمەتكاری زێدە دبوو.

الخلاصة

الضوضاء متسببة في حالة الاجهاد التأكسدي الغيرمتوازن وفقدان السمع لدى عمال مولدات الكهرباء

خلفية واهداف البحث: كثير من الناس يتعرّضونَ للضوضاء والتي تسبب مشاكلِ صحيةِ مُخْتَلِفةِ وتؤدي الى خسارةِ سمعيةِ سببها الضوضاءَ. هذا العملِ صُمِّمَ لدراسَة تأثيرِ التعرّضِ المطوّلِ إلى عتباتِ الضوضاءِ العاليةِ على حالة توازن الاجهاد التأكسدي مع قدرةَ السمع لدى عُمّالِ المولّداتِ الكهربائيةِ الخاصة الكبيرة. الهدف من البحث هو التحقق في تأثير الضوضاء على حالة التوازن بين عوامل الإجهاد ألتأكسدي ومضادات الأكسدة على فقدان السمع عند الإنسان

طرق البحث: أجريت هذه الدراسة على (89) من الذكور البالغين ، ستون منهم من عمال المولدات الكهربائية الكبيرة و (29) من الاصحاء كمجموعة سيطرة . تم قياس شدة الصوت والضوضاء داخل غرف المولدات وخارجها . في كلا المجموعتين تم قياس قدرة السمع وضغط الدم ومؤشر كتلة الجسم ومستوى مجموع مضادات الأكسدة والمالونديالديهايد في مصل الدم وصورة دهون الدم لكلا المجموعتين.

النتائج: وجود فروق ذات دلالة إحصائية بين المجموعتين في ارتفاع قيم كل من مؤشر كتلة الجسم وضغط الدم الانقباضي .تم كشف عن وجود حالة عدم اتزان الاجهاد التأكسدي لدى عمال المولدات والذي اتضح بنقصان معنوي لمجموع مضادات الاكسدة وزيادة معنوية للمالونديالديهايد في مصل الدم لدى مجموعة العمال عند مقارنتها مع مجموعة السيطرة (الاصحاء). لوحظ وجود فروق ذات دلالة معنوية لارتفاع مستويات الكوليسترول وثلاثي الكليسرايد والبروتينات الدهنية ذات الكثافة واطئة جدا في مصل الدم لدى عمال المولدات عند مقارنتها بمجموعة السيطرة. استنتج من دراسة تخطيط السمع ان قابلية السمع تختلف بشكل معنوي بين مجموعتي العمال والسيطرة عند الترددات: 250، 500، 1000، 2000، 4000، 6000، 8000 هرتز. على اية حال لم يلاحظ وجود ذات فروق معنوية في تردد 125 هرتز بين المجموعتين.

الاستنتاجات: مستوى التعرّضِ للضوضاءِ في عُمّالِ المولّداتِ الكهربائية في مدينةِ دهوك كَانَ عالي جداً وقد يصل الى المستوى المؤذي للخلايا (≥dB 95) داخل وخارج غرف المولدات الكهربائية مع عدم وجود أي إجراءات الحمايةِ السمعيةِ. تم تسجيل وجود حالة عدم الاتزان الاجهاد التأكسدي في عُمّالِ المولّداتِ الكهربائية وهي قَدْ تَتَقدّمُ لخسارة السَمْع. السمع الضعيف لدى عمال المولدات الكهربائية يبَدأ ضمن السَنَواتِ الأولى الأربعة من بداية توظيفهم ويصبح أكثر وضوحاً عندما تزداد مدّةَ التوظيفَ.

SPERM MOTILITY, CONCENTRATION, MORPHOLOGY AND PREGNANCY OUTCOME POST-VARICOCELECTOMY IN DUHOK

KHALIS S. AMMO, MBChB, FIBMS*

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ABSTRACT

Background and objectives Varicocele is a dilatation and tortuousity of pampiniform plexus. It is a very common condition present in 15% of the general male population and 40% of men evaluated for primary infertility and 80% of men with secondary infertility. A varicocele develops because of defective valves that normally allow blood to flow away from the testicle toward the abdomen. The improvement of seminal parameters and rate of pregnancy after varicocele correction had been reported by several investigators in clinical series. The aim of this study is to evaluate the sperm motility, concentration, morphology and pregnancy rate post-varicocelectomy.

Methods A retrospective study conducted on 160 male patients who underwent surgery for correction of varicocele for the period between January 2005 to January 2011. All the included patients had primary infertility. Pre and post operative evaluation was held by frequent seminal fluid analysis. The place of the study was Azadi teaching hospital.

Results The improvement in the sperm concentration observed in 80 patients (50%) at 3 months, 112 patients (70%) at 6 months, and 128 patients (80%) at 12 months. The sperm motility were improved in 64 patients (40%) at 3 months , 80 patients (50%) at 6 months and 112 patient (70%) at the 12 months .While the improvement in the sperm morphology was seen in 56 patients (35%) at 3 months , 80 patients (50%) at 6 months and 104 patients (65%) at the 12 months. Accordingly the best results obtained after 12 months and the greatest changes were in the sperm concentrations (80%) followed by motility (70%) and morphology (65%). On the other hand the pregnancy rate was 8.1% at 3 months, 21.8% at 6 months and 30% after 12 months.

Conclusions We conclude that the palpable varicocele had a bad effect on the sperm parameters , the repair of varicocele improved these parameters, and in the same time it improves the rate of spontaneous pregnancy but not to the same extend as the improvements in the sperm parameters.

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Key words: Infertility, Sperm motility, Sperm count ,Varicocelectomy

Varicocele is a dilatation of internal spermatic veins that drain the testicle.[1] It is very common condition present in 15% of the general male population and 40% of men evaluated for infertility.[2] A varicocele develops because of defective valves that normally allow for blood to flow away from the testicle toward the abdomen.[3] Testicular injury occurs due to abnormal back flow of blood from the abdomen into the scrotum and this create a hostel environment for sperm development.[4] A unilateral varicocele may affect both testicles.[5] The most probable explanation for the more frequent development of A varicocele of the left side alone is because the left spermatic vein is longer than the right.[6] The left vein

* *Lecturer at the department of surgery, School of medicine, Faculty of Medical Sciences, University of Duhok, Head of Department of surgery, Urologist at Azadi teaching Hospital in Duhok. Email: khalis_sabri@yahoo.com*

enter the left renal vein at a right angle near a site of compression by the mesenteric artery while the right spermatic vein drains at a softer angle into the vena cava. The anatomical factors promote back flow of blood in the left spermatic vein, resulting in pooling of blood and increased temperature and congestion in the testicle.[5,7] The diagnosis of varicocele can usually be made on physical examination of the scrotum while the patient is standing. The varicocele feels like a bag of worms and disappears or became significantly reduced when the patient lies down.[8] Repair of the varicocele is indicated when the couple has documented infertility with normal female partner but a male with one or more abnormal semen parameters and the presence of varicocele on physical examination and also when a varicocele cause testicular pain or discomfort or there is a significant discrepancy between the size of two testicles.[9] The important sperm functions are impaired in patients with varicocele.[10]

The improvement of seminal parameters after varicocele correction has been reported by several investigators in clinical series.[11-14] Furthermore some researches suggest that varicocelectomy can improve human sperm DNA integrity in infertile men with varicocele.[15]

The aim of the study is to evaluate the sperm motility, concentration, morphology and pregnancy rate post-varicocelectomy.

METHODS

A retrospective study was conducted in the period of six years from January, 2005-January, 2011 in the Azadi teaching hospital in duhok.

The study included 160 patients with palpable varicocele with history of primary infertility.

The age of the patients ranged from 22-40 years. Preoperative evaluations of the patients included a complete history, physical examination, ultrasound, hormonal study, and two seminal fluid analysis with two months interval.

Sub-inguinal varicocelectomy for all the patients done under general anesthesia as a day case surgery. The follow up of the cases all the patients were followed with three seminal fluid analysis with calculation of the pregnancy rate at three, six and twelve months.

RESULTS

The age of the patients ranged from (22-40) years, 96 patients (60%) were bellow 30 years and 66 patients (40%) were above 30 years (Table 1).

Regarding the distribution of the varicocele, the left sided pathology was in 138 patients (68.25%), 15 patients (9.75%) were with bilateral pathology and only 7 patients (4.3%) were with right sided Varicocele (Table 2).

Table 1. Age distribution of patients

Age	No. of patients (%)
Below 30	96 (60)
Above 30	64 (40)
Total	160

Table 2. Distribution of the site of varicocele

Site	No. of patients (%)
Left	138 (86)
bilateral	15 (9.6)
right	7 (4.3)
Total	160

All the included cases were with palpable varicocele and an abnormal seminal fluid analysis, at least two were abnormal, the affections were in the sperm concentration, motility and the morphology, any patient with palpable varicocele and normal seminal fluid analysis was excluded from this study. The severity of the effect was different from case to another.

We found that the improvement in the sperm concentration was in 80 patients (50%) at 3 months, in 112 patients (70%) at 6 months, and in 128 patients (80%) at 12 months (Table 3).

The improvement in the sperm motility were in 64 patients (40%) at 3 months, in 80 patients (50%) at 6 months and in the 112 patient (70%) at the 12 months as shown in table 3.While the improvement in the sperm morphology was in 56 patients (35%) at 3 months, in 80 patients (50%) at 6 months and in the 104 patients (65%) at the 12 months table as shown in table 3. So we found the best results were after 12 months and the higher results were in the sperm concentrations (80%), motility (70%) and morphology (65%). On the other hand the rate of pregnancy was positive in 13 cases (8.1%) at 3 months, 35 cases (21.8%) at 6 months and in 48 cases (30%) after 12 months table 4. so the pregnancy rate was higher after 12 months.

Table 3. The improvement of sperm concentration, motility and morphology (n = 160)

Time	3 months No. (%)	6 months No. (%)	12 months No. (%)
Sperm conc.	80 (50)	112 (70)	128 (80)
Sperm motility	64 (40)	80 (50)	112 (70)
Sperm morphology	56 (35)	80 (50)	104 (65)

Table 4. The pregnancy rate

Time	No.	Paternity %
3 months	13	8.1
6 months	35	21.8
12 month	48	30

DISCUSSION

Varicocele is a treatable cause of male infertility, with prevalence of 15% of normal males and 40% of the infertile population.[16] It has been mentioned since ancient times.[17] with surgical management performed by Abu al-Qasim Khalaf ibn Abbas al-Zahravi.[18]

Konodo et al said that the varicocele affects fertility and is the most common known cause of infertility.[1] various mechanisms have been suggested to account for the testicular dysfunction associated with varicocele , including retrograde flow of toxic metabolites from the adrenal gland or kidneys , venous stasis with germinal epithelial hypoxia, alteration in the hypothalamic-pituitary-gonadal axis and increase in testicular temperature .[19] In addition, deregulations of nitric oxide.[20] Reactive oxygen sepsis.[21] And regulators of apoptosis have been

implicated in the pathophysiology of varicocele. [22]

The results of our study demonstrate that the repair of varicocele by surgery results in highly significant improvement of sperm concentrations 50%,70%, and 80% at 3,6 and 12 months respectively. The sperm motility improvement was 40%, 50% and 70% at 3, 6 and 12 months respectively. While sperm morphology improved in 35%, 50%, and 65% respectively. Shamsa in 2010 found that the improvement in sperm concentration, motility and morphology after one year were 55,51 and 46% respectively.[23] In 2009, Jasemi et al found that the sperm condensation and motility at 12 months after repair of varicocele was significantly high.[24] Agarwal et al and Marmar et al said that varicocelectomy significantly improves semen parameters in infertile men with palpable varicocele and abnormal semen parameters.[25,26]

Baazeem et al in 2011 found that the varicocelectomy is associated with significant increase in sperm concentration and improvement in progressive motility and morphology.[27] Another report by Johnson and colleagues showed that 70% of healthy, asymptomatic military recruits with palpable varicoceles had an abnormality on semen analysis.[28]

Furthermore, studies in humans suggest that varicoceles cause progressive testicular damage over time.[29] It appears that surgical repair of varicoceles not only halts this declines in testicular function but often reverses it.[30] And the improvements in semen parameters seen in 70-80% of men after varicocele ligation.[30,31] Others found Improved motility in 70% of patients and improved sperm densities in 51% and improved morphology in 44% of patients .[31]

The ultimate goal of varicocele repair is the pregnancy. The rate of spontaneous pregnancy after surgery was 30% within one year in our study, the pregnancy rate is improved probably due to improvement in the semen parameters .Paternity was 56% in a study done in Iran in 2010 by Shamsa and his colleagues.[23] Baazeem et al in 2011 found that the rate of pregnancy was 41.9% after on year of varicocele repair.[27]

We conclude that the palpable varicocele had a bad effect on the sperm parameters, the repair of varicocele improved these parameters, and in the same times it improves the rate of spontaneous pregnancy but not to the same extend as the improvements in the sperm parameters. We advice surgical repair of palpable varicocele in infertile male patients with abnormal seminal fluid analysis, provided that the female side is normal.

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پوختە

بزاڤا سپێرمی، جۆرێن وی ورێژەییێت پەیدابوونا دووگیانیێ و جووتبوونێ پشتی نشتەرگەریا چاك كرنا دەوالیا گونان

پێشەكی و ئارمانج: دەوالیا گونان بەرفرەهبوون ولێكبادانەكە د توڕا دەمارێن گونانە ئەڤە ژی بوارەكێ بەربەلاڤە و ڕێژا وێ دگەهتە 15 ٪ ژ هەمی نێرا و 40 ٪ ژ ڕێژا نێرا ئەوێن تووشی نەزۆكیا دەسپێكی بووین و 80 ٪ ژ ئەوێن تووشی نەزۆكیا نەسەرەكی بووین . پەیدابوونا دەوالیا گونان ژ ئەنجامێ تێكچوونەكێ یە د كارێ ئەزمانكێن هەی د وان دەماراندا ئەوێن خوینێ ژ گونان ڤە دگوهێزنە زكی. باشبوون درێژە وبزاڤ وجۆرێن سپێرمی پشتی نشتەرگەریا چاك كرنا دەوالی بو جهێ گرنگیدانا ڤەكولەرا. ئارمانج ژ ڤەكولینێ خواندن و هەلسەنگاندنا ڕێژە وبزاڤ وجۆرێن سپێرمایە دگەل خواندنا ڕێژێن پەیدابوونا دوو گیانیێ پشتی نشتەرگەریا چاك كرنا دەوالیا گونان..

ریكێن ڤەكولینێ: خواندنەكا پاشەڕۆژێ یە ول سەر 160 نەخۆشان هاتەكرن. كو نشتەرگەریا دەوالیا گونان ژ كانینا دووێ یا سالا 2005 هەتا كانینا دوو 2011 بو هاتیە كرن. هەمی نەخۆشێن فبەر خواندنێ كەفتین ب نەزۆكیا دەسپێكی تووشببوون. زنجیرەكا لێنێرینێن تاقیگەهێ یا سیمەنی بو هەمی نەخۆشا بەری و پشتی نشتەرگەریێ هاتە كرن. جهێ خواندن وكارێ نەخۆشخانا ئازادی یا فێركرنێ ل دهۆكێ بوو.

ئەنجام: باشبوون درێژا سپێرمێدا 50 ٪ بوو پشتی سێ هەیڤا ژ نشتەرگەریێ و 70 ٪ پشتی شەش هەیڤا و 80 ٪ پشتی دوازدە هەیڤان بەلێ باشبوون د بزاڤا سپێرمێ 40 ٪ و 50 ٪ و 70 ٪ بوون پشتی 3 و 6 و 12 هەیڤا لدویف ئێك لایەكێ دیڤە ئەنجامێن ڕێژا پەیدابوونا دووگیانیێ 8.1 ٪ و 21.8 ٪ و 30 ٪ پشتی 3 و 6 و 12 هەیڤان ژ نشتەرگەریێ.

دەرئەنجام: دەوالیا گونان كارتێكرنەكا خراب هەیە لسەر بزاڤ وڕێژە و جۆرێن سپێرمی ونشتەرگەریا چاك كرنا دەوالیا مفایەك هەیە بو ڕێژە وبزاڤ وجۆرێن سپێرمێ هەر وەسا لسەر ڕێژا پەیدابوونا دووگیانیێ ، بەلێ ب پلەكا كێمتر.

الخلاصة

حركة النطف، أشكالها ونسب حدوث الحمل والإخصاب بعد عملية إصلاح دوالي الخصية في دهوك

خلفية واهداف البحث: دوالي الخصية هو توسع والتواء يحصل في شبكة أوردة الخصية وهي حالة شائعة تصل نسبتها إلى 15% من عامة الذكور و40% من نسبة الذكور الذين يعانون من العقم الأولي و80% من أولائك الذين يعانون من العقم الثانوي. حدوث دوالي الخصية هو نتيجة خلل في عمل الصمامات الموجودة في الأوردة الناقلة للدم من الخصية باتجاه البطن. التحسن في نسب وحركة وأشكال النطف بعد عملية إصلاح الدوالي أخذت اهتمام الكثير من الباحثين. الهدف من الدراسة هو دراسة وتقييم نسب وحركة وأشكال النطف مع دراسة نسب حدوث الحمل بعد عملية إصلاح دوالي الخصية.

طرق البحث: هي دراسة مستقبلية أجريت على 160 مريض. أجريت لهم عملية دوالي الخصية من الفترة كانون الثاني من العام 2005 ولغاية كانون الثاني 2011. جميع المرضى الذين شملوا بالدراسة كانوا يعانون من العقم الأولي. سلسلة من الفحص ألمختبري للسائل المنوي اجري لجميع المرضى قبل وبعد العملية. مكان الدراسة والعمل كان مستشفى أزادي التعليمي في دهوك.

النتائج: كان التحسن في نسب النطف 50% بعد ثلاثة أشهر من العملية و70% بعد (6)أشهر و 80% بعد 12 شهرا. أما التحسن في حركة النطف فكان 40% , 50% , و 70% بعد 3 ,6 ,12 شهرا على التوالي. من جهة أخرى كانت نتائج نسبة حدوث الحمل 8.1%، 21.8% و 30% بعد 3،6،12 شهرا من العملية.

الاستنتاجات: دوالي الخصية له تأثيرات سيئة على حركة ونسب وإشكال النطف، وعملية إصلاح الدوالي لها فائدة لكل من نسب وحركة وأشكال النطف وكذلك على نسبة حصول الحمل ولكن بدرجة أقل.

SERUM LIPID PROFILE IN SUBJECTS WITH TYPE 2 DIABETES MELLITUS AND HYPERTENSION IN RELATION TO METABOLIC SYNDROME: A CASE CONTROL STUDY

SAMIR B. AL-MUKHTAR, MBChB, DCH, MSc, PhD*
NABEEL N. FADHIL, MBChB, CABM, FRCP (London)**
BASSAM E. HANNA, MBChB, FICMS (Chemical Pathology)***

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ABSTRACT

Background and objectives Dyslipidemia is a major modifiable risk factor for cardiovascular disease. It commonly coexists with hypertension, type 2 diabetes and metabolic syndrome. The objective is to study the trends of serum lipid profile among subjects with type 2 diabetes and/or arterial hypertension in relation to metabolic syndrome.
Methods A case control study involved 256 subjects with lone type 2 diabetes, 205 subjects with concurrent type 2 diabetes/hypertension, 173 subjects with lone hypertension and 250 controls attending Al-Zahrawi Private Hospital, Mosul, Iraq from 1st June to 31st December 2011. Fasting plasma glucose, glycated hemoglobin and lipid profile parameters were estimated and the data were analyzed statistically.
Results According to WHO criteria, metabolic syndrome constituted 34.4% of the enrolled hypertensives in concurrent type 2 diabetes/hypertension and 30.6% of the enrolled diabetics in lone type 2diabetes and concurrent type 2 diabetes/hypertension. The mean ages of the different study groups were similar. The means of body mass indices, glycemic control, blood pressures, low density lipoprotein cholesterol, total cholesterol and the total cholesterol/ high density lipoprotein cholesterol ratio of all the groups were significantly higher than controls. The mean triglycerides level of subjects with lone type 2 diabetes and concurrent type 2 diabetes/hypertension were higher than the controls and the mean of high density lipoprotein cholesterol was only, significantly, low in concurrent type 2 diabetes/hypertension subjects in comparison with the controls.
Conclusions Concurrent type 2 diabetes and hypertension adds the risk of exaggerating dyslipidemia in addition to their own potential cardiovascular risk. This is specially prominent when both are part of metabolic syndrome.

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Key words: Type 2 Diabetes mellitus, Hypertension, Metabolic syndrome, Lipid profile, Plasma glucose, Glycated hemoglobin, Dyslipidemia

Coronary heart disease (CHD) is the leading cause of death in adults worldwide and its most important treatable or modifiable risk factors are diabetes mellitus (DM), dyslipidemia and systemic hypertension.[1,2] For unknown cause, dyslipidemia commonly coexists with hypertension.[3] Serum cholesterol has been thought to influence the regulation of blood pressure by adrenergic stimulation.[4]

Moreover, hypercholesterolemia, resulting from imbalanced mobilization of

* *Lecturer of Biochemistry, Department of Biochemistry, Nineveh College of Medicine, University of Mosul, Mosul-Iraq*
** *Assistant Professor of Medicine, Department of Medicine, Nineveh College of Medicine, University of Mosul, Mosul-Iraq*
*** *Assistant Professor of Biochemistry, Department of Biochemistry, Nineveh College of Medicine, University of Mosul, Mosul-Iraq*
Correspondence author: Samir B. Al-Mukhtar. Email: samiralmukhtar@yahoo.com. Mobile: 07701657321

cholesterol, accounts for the earliest stages of atherogenesis, is a risk factor for CHD, and is said to interfere with blood pressure regulation.[4] Vice versa, hypertension when treated with thiazide diuretics accentuates the hyperlipidemia, perhaps by causing potassium or sodium depletion.[5] The risk of CHD in patients with co-morbid hypertension and dyslipidemia is greater than the sum of CHD risks of hypertension and dyslipidemia when they occur alone.[3,6] Wilson et al asserted that the rate of CHD is significantly associated with the specified categories of blood pressure, total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), and low density lipoprotein cholesterol (LDL-C).[7]

Essential hypertension is being increasingly recognized as a part of a complex multi-faceted disorder, the metabolic syndrome (Met S), which may include other abnormalities including dyslipidemia, central obesity, glucose intolerance and hyperinsulinemia, all of which may increase the risk of CHD.[8-10] Both, central adiposity and insulin resistance can exert effect on blood pressure, serum lipids and several cardiovascular factors.[11]

Type 2 DM is characterized by hypertriglyceridemia, low concentrations of HDL-C, increased small-dense low density lipoprotein, greater postprandial lipidemia and a considerable increase in vascular risk.[12] Indeed, non-fasting triglycerides (TGs) predict the vascular risk better than fasting measurements.[13] Lipoprotein abnormalities are manifested during the largely asymptomatic diabetic prodrome and contribute substantially to the increased risk of macrovascular disease, whereas hyperglycemia is a very late stage in the sequence of events from insulin resistance to frank diabetes. The insulin-resistant diabetes course affects virtually all lipids and lipoproteins. Chylomicron and very low density lipoprotein remnants accumulate, and as TGs increase, still within the so-called normal range, abnormalities in high density and low density lipoproteins become more apparent.[14] In Japanese community-dwelling adults, the increase of lipid ratios of TG/HDL-C, TC/HDL-C, LDL-C/HDL-C as well as TGs and reduction of HDL-C were consistently associated with Met S, insulin resistance and serum high molecular weight adiponectin.[15]

In this study, we aimed to explore the trends of serum lipids among subjects with isolated or concurrent type 2 DM and arterial hypertension in relation to Met S and in comparison with non-hypertensive and non-diabetic controls.

METHODS

This case-control study enrolled 884 age and sex-matched subjects in four groups: 256 (28.9%) lone type 2 diabetics (lone T2D), 205 (23.2%) type 2 diabetic and hypertensive (T2D/Ht), 173 (19.6%) lone hypertensive (lone-Ht), and 250 (28.3%) apparently healthy normotensive subjects served as controls. In another way the study population were 461 (217 females, 244 males) of T2D subjects and 423 (196 females, 227 males) of non diabetic subjects age ranged between 33-78 years

with a mean age (SD) of 52.26 (7.64) years. All subjects were attending Out-Patient Clinic of Al-Zahrawi Private Hospital in Mosul city during the period from 1st June to 31st December 2011.

The enrolled diabetic subjects were taking oral hypoglycemic drugs in addition to alleged dieting, while the hypertensive subjects were using antihypertensive drugs. The subjects, after obtaining their written consent were examined clinically and information pertaining to age, sex, habits and history of hypertension, diabetes and other concurrent morbidities were recorded in specially preformed case record. Exclusion criteria included frank proteinuria detected by albustix, cigarette smoking and lipid altering drugs, such as oral contraceptive, diuretics, beta-blockers and lipid lowering drugs. Subjects with lipid-altering diseases: hepato-biliary disease, hypothyroidism, chronic kidney disease and nephrotic syndrome were also excluded. A series of laboratory investigations using standard protocols for estimation of fasting plasma glucose (FPG), glycated hemoglobin (HbA1c), and lipid profile parameters were done. Body mass index (BMI) was calculated as weight in kilograms divided by squared height in meter. [16, 17]

Classification of dyslipidemia was based on ATP III recommendation using lipoprotein cut-off limits of ≥ 1.7 mmol/L for TGs , ≥ 5.2 mmol/L for TC, <1.0 mmol/L for HDL-C, ≥ 2.6 mmol/L for LDL-C in lone T2D and concurrent T2D/Ht groups, and ≥ 3.4 mmol/L for LDL-C in some of lone Ht group who are having 2 or more risk factors and ≥4.1 mmol/L for LDL-C in some of lone Ht and control groups who are having 0-1 risk factor.[18] For TC/HDL-C ratio, a figure of ≥ 5 was considered as a cut-off limit according to the recommendation of the British Hyperlipidaemia Association.[19]

The presence of first-degree relatives with manifest CHD whether premature (development of coronary artery disease at age under 50 years in males and under 60 years in females)[20] or not premature and/or the presence of cerebrovascular diseases and/or peripheral artery diseases were considered as indicator of a positive familial history as well.

Diagnostic criteria for diabetes were used according to the American Diabetes Association 2007 guidelines: Fasting plasma glucose (FPG) up to 7 mmol/L and above was considered to be diabetic and levels between 5.55 mmol/L and 6.9 mmol/L was considered as impaired fasting glucose.[21] Glycemic control among diabetics was based on the WHO recommendation. The subjects were classified according to fasting plasma glucose (mmol/L) into good FPG<6.7, fair FPG 6.7-8.9 and bad control FPG ≥8.9.[22] Depending on HbA1c, the glycemic control was categorized as optimal when HbA1c is <6.8%, acceptable: HbA1c is 6.8-7.6% and poor control: HbA1c is >7.6 %.[23]

Regarding blood pressure, hypertension was defined by self-reporting of the diagnosis and/or using an anti-hypertensive medication, or if the systolic blood pressure was >140 mmHg or if the diastolic blood pressure was >90 mmHg, for both men and women.[24]

The subjects attended the Out-Patient Clinic in the morning (0800-0900 a.m) after an overnight fast. Venous blood samples (10) mL was collected from each subject during single outpatient visit. The blood samples were divided into three parts, each of which was measured in different ways as follows:

For FPG measurement, 1 mL of blood was transferred into fluoride tube. For HbA1c measurement, 2.5 mL of blood was transferred into EDTA tube. The remaining blood was transferred into a disposable plain tube for the measurement of other biochemical parameters. Plasma glucose was measured by oxidase peroxide method.[25] Glycated hemoglobin measured in whole blood by ion exchange resin quantitative colorimetric determination.[26] Determination of (TC), high HDL-C and TGs were performed using enzymatic methods.[27] The LDL-C was calculated by using Friedewald formula for those with TGs <4.5 mmol/L [28]{LDL-C(mmol/L)=TC –[HDL-C +(TG ×0.455)]}. Very low density lipoprotein cholesterol (VLDL-C) (mmol/L) was calculated from the formula: VLDL-C (mmol/L)=TG × 0.455.[28]

The subjects with lone T2D and with concurrent T2D/Ht where segregated according to WHO criteria into two groups for each, a group fulfilling the criteria of Met S and a group that does not. The adopted diagnosis criteria of Met S were those suggested by the WHO requiring the presence of: diabetes mellitus, impaired glucose tolerance, impaired FPG or insulin resistance plus any 2 of the following: blood pressure: ≥140/90 mmHg, dyslipidemia [TGs: ≥1.695 mmol/L and HDL-C ≤0.9 mmol/L (male), ≤ 1.0 mmol/L (female)], central obesity [waist:hip ratio >0.90 (male); >0.85 (female), or BMI >30 kg/m^2], microalbuminuria [urinary albumin excretion ≥20 µg/min or albumin: creatinine ratio ≥30 mg/g].[29, 30]

Data were presented as mean ±SD. Analysis of variance (ANOVA), Duncan and Z test tests were utilized to compare parametric values. Chi-square was used for non parametric data. *p* value <0.05 was considered statistically significant.

RESULTS

Out of the (634) randomized hypertensive and/or diabetic subjects, lone T2D was the most prevalent (40,37%), followed by concurrent T2D/Ht (32.3%) and lone Ht subjects (27.3%). The mean BMIs of subjects in all subgroups were significantly higher than that of the healthy controls (Table 1). Systolic and diastolic blood pressures were significantly higher in concurrent T2D/Ht group than lone Ht (160.17±17.5 / 96.68±7.44 mmHg vs. 154.16 ±10.93 / 93.79 ±4.14 mmHg, p<0.001) (Table 1).

The FPG was significantly higher among the concurrent T2D/Ht subjects (11.84 ± 3.63 mmol/L) than lone T2D (9.53 ± 2.97 mmol/L) (p< 0.001) (Table 2). The mean duration of diabetes and the number of subjects with duration of diabetes beyond 5 years were significantly higher among concurrent T2D/Ht subjects than lone T2D (8.74 ± 3.68 years vs. 4.59 ±3.14 years, p< 0.001) (Table 2 and 3).

Table 1. Anthropometric measures and blood pressure in the studied groups

Parameters	Controls N = 250	Lone Ht N = 173	Lone T2D N = 256	T2D/Ht N = 205
Age (Years)	52.77 ± 7.93 a	51.11 ± 7.06 a	52.75 ± 7.82 a	52.01 ± 7.45 a
Weight (Kg)	64.70 ± 8.36 a	66.95 ± 10.51 b	77.75 ± 9.10 c	85.65 ± 8.72 d
Height (cm)	166.11 ± 6.10 a	166.26 ± 6.26 a	167.53 ± 5.94 b	168.87 ± 5.36 c
BMI (Kg/m^2)	23.38 ± 2.08 a	24.16 ± 3.04 b	27.70 ± 2.91 c	30.04 ± 2.79 d
Systolic BP (mmHg)	122.16 ± 4.98 a	154.16 ± 10.93 c	127.19 ± 5.95 b	160.17 ± 17.50 d
Diastolic BP (mmHg)	79.80 ± 2.93 a	93.79 ± 4.14 c	81.05 ± 2.95 b	96.68 ± 7.44 d

Different letters horizontally indicate significant at P<0.05.

Table 2. Comparison of glycemic status in the studied group

Parameters	Controls N = 250	Lone Ht N = 173	Lone T2D N = 256	T2D/Ht N = 205
FPG (mmol/L)	4.85 ± 0.50 a	4.89 ± 0.36 a	9.53 ± 2.97 b	11.84 ± 3.63 c
HbA1c (%)			9.92 ± 0.93 a	10.22 ± 0.90 b
Duration of DM (Years)			4.59 ± 3.14 a	8.74 ± 3.68 b

Different letters horizontally indicate significant at P<0.05.

Table 3. Frequency distribution of the study samples by demographic variables and some biochemical parameters. Values represented as number (%)

Parameters	Controls N = 250	Lone Ht N = 173	Lone T2D N = 256	T2D/Ht N = 205
		Lipid Profile		
T-C ≥ 5.2 mmol/L	22 (8.8) a	131 (75.7) c	97 (37.9) b	154 (75.1) c
TGs ≥ 1.7 mmol/L	86 (34.4) a	68 (39.3) a	123 (48.0) ab*	129 (62.9) c
HDL-C < 1 mmol/L	47 (18.8) a	24 (13.9) a	53 (20.7) a	84 (41.0) b
LDL-C•	1 (0.4) a	82 (47.4) b	181 (70.7) c	183 (89.3) d
T-C/HDL-C Ratio ≥ 5	19 (7.6) a	77 (44.5) c	68 (26.6) b	165 (80.5) d
		Duration of Diabetes Mellitus (Years)		
< 5			168 (65.6) b	36 (17.6) a
5-10			69 (27.0) a	82 (40) b ***
≥ 10			19 (7.4) a	87 (42.4) b

Letters from a-d horizontally means progressive significant elevation according to Chi-square test. p<0.001.
** Significant higher than control, P<0.005. No significant difference from lone-Ht.*
*** Significant higher than control and lone T2D, p<0.001 and <0.01 respectively. No significant difference from lone-Ht.*
**** p<0.005*
•Lone T2D and concurrent T2D/Ht groups, ≥ 3.4 mmol/L for some of lone Ht group who are having 2 or more risk factors and ≥4.1 mmol/L for some of lone Ht and control groups who are having 0-1 risk factor.

The lipidemic profiles of the studied subgroups were differ than in the control group. Significantly higher means of (TC, LDL-C, VLDL-C and TG) and higher TC/HDL-C ratio in the studied subgroups than in the controls were observed (Table 4).

Comparing the subgroups' lipid, outcomes showed that lone Ht subjects had a

higher (TC and LDL-C) and higher TC/HDL-C ratio (p<0.001) than lone T2D subjects (Table 4).

In regard to the Met S, out of the 461 enrolled diabetic subjects, the prevalence of Met S according to WHO definition was 30.6% (11 lone T2D and 130 concurrent T2D/Ht) vs. 69.4% without Met S (245 lone T2D and 75 concurrent T2D/Ht). Meanwhile, out of the 378 enrolled hypertensive subjects, Met S constituted 34.4% (Table 5).

The highest levels of TC and TC/HDL-C ratio were found in the Met S with concurrent T2D/Ht (5.96±1.18 and 6.33±1.12 respectively) and the highest levels of TGs and VLDL-C (3.25 ± 0.96 and 1.48 ± 0.43 respectively) and lowest level of HDL-C (0.88 ± 0.09) were detected in Met S with loneT2D (Table 5).

DISCUSSION

Diabetes mellitus is a major public-health problem worldwide. Its prevalence is rising in many parts of the developing world and Iraq is not an exempt. In this study, out of the 634 randomly collected T2D and Ht subjects, lone T2D was the most prevalent (40.37%), followed by concurrent T2D/Ht (32.3%) and lone Ht subjects (27.3%). This makes the percent of T2D and Ht patients in the study sample: 72.4% and 59.6%, respectively, reflecting a higher prevalence

Table 4. Selected biochemical parameters in the studied groups

Parameters	Controls N = 250	Lone Ht N = 173	Lone T2D N = 256	T2D/Ht N = 205
T-C (mmol/L)	4.35 ± 0.66 a	5.53 ± 0.53 c	4.98 ± 0.86 b	5.92 ± 1.11 d
TG (mmol/L)	1.54 ± 0.78 a	1.63 ± 0.76 a	1.81 ± 0.94 b	2.26 ± 1.31 c
HDL-C (mmol/L)	1.14 ± 0.23 b	1.18 ± 0.26 b	1.14 ± 0.24 b	1.02 ± 0.23 a
LDL-C (mmol/L)	2.50 ± 0.65 a	3.60 ± 0.58 c	3.01 ± 0.90 b	3.87 ± 1.12 d
VLDL-C (mmol/L)	0.70 ± 0.35 a	0.74 ± 0.35 a	0.83 ± 0.45 b	1.03 ± 0.60 c
TC/HDL-C Ratio	3.91 ± 0.80 a	4.84 ± 0.96 c	4.46 ± 0.83 b	5.97 ± 1.21d

Different letters horizontally indicate significant at P<0.05.

Table 5. Lipid profile parameters in all groups. Values represented as mean ±SD

Parameters	Controls N = 250	Non-Metabolic Syndrome			Metabolic Syndrome	
		Lone-Ht N = 173	Lone T2D N = 245	T2D/Ht N = 75	Lone T2D N = 11	T2D/Ht N = 130
TC (mmol/L)	4.35 ± 0.66 b	5.53 ± 0.53 d	5.03 ± 0.83 c	5.85 ± 0.97 e	3.85 ± 0.77 a*	5.96 ± 1.18 e
TGs (mmol/L)	1.54 ± 0.78 a	1.63 ± 0.76 a	1.75 ± 0.89 a	1.84 ± 0.80 a	3.25 ± 0.96 c	2.50 ± 1.48 b*
HDL-C (mmol/L)	1.14 ± 0.23 b	1.18 ± 0.26 b	1.15 ± 0.24 b	1.14 ± 0.24 b	0.88 ± 0.09 a	0.96 ± 0.20 a
LDL-C (mmol/L)	2.50 ± 0.65 b	3.60 ± 0.58 d	3.08 ± 0.83 c	3.88 ± 0.86 d	1.49 ± 1.02 a	3.87 ± 1.24 d
VLDL-C (mmol/L)	0.70 ± 0.35 a	0.74 ± 0.35 a	0.80 ± 0.40 a	0.84 ± 0.36 a	1.48 ± 0.43 c	1.14 ± 0.67 b*
TC/HDL-C Ratio	3.91 ± 0.80 a	4.84 ± 0.96 c	4.46 ± 0.81 b	5.34 ± 1.12 d	4.47 ± 1.27 b	6.33 ± 1.12 e

*Letters from a-e horizontally means progressive significant elevation according to Duncan test ($p < 0.001$, * $p < 0.05$)*

of T2D than Ht among our randomly enrolled sample, and possibly among Mosul population in general. In regard to the prevalence of Met S, out of the 461enrolled T2D patients, 30.6% fulfilled the WHO criteria of Met S (11 loneT2D and 130 concurrent T2D/Ht) vs. 69.4% who did not fit the Met S (245 lone T2D and 75 T2D/Ht). These proportions reflect that when T2D is associated with Ht the state is more likely to be a Met S than when T2D or Ht is present alone.

In spite of being not a primary aim of this study, we revised the comparative results elsewhere in the world. Using the same criteria (WHO definition), the prevalence of Met S among T2D in native African and in Karachi (Pakistan) was 59.1% and 46% respectively.[31, 32] Involving families of Finland and Sweden descent, the prevalence of Met S in male and female subjects with T2D was 84% and 78%, respectively,[33] while the prevalence of Met S in Ireland was 21 % (29). Our figures were lower than those of native African, Karachi (Pakistan), Finland and Sweden, but higher than the result of Ireland study. Disparities in the prevalence of Met S are largely due to differences in lifestyles, age of the studied populations and on application of uniform diagnostic criteria.[34]

Concerning the hypertension/Met S relationship, the prevalence of Met S among the enrolled Ht subjects (n 378) constituted 34.4%. This prevalence is, strangely, nearly the same as what is reported in other countries. In Germany, for example, the frequency of occurrence of Met S among hypertensive was 34.5% according to WHO criteria, 35.0% according to NCEP ATP III, and 42.5% according to IDF criteria.[35] In a Kuwaiti study, the prevalence of hypertensive patients who met the criteria for Met S, [using criteria similar to ATP III] was (34%).[36]

The BMIs of all subgroups (lone T2D, lone Ht and concurrent T2D/ Ht) were significantly higher than that of the healthy controls. The role of BMI as a predictor for hypertension has been precedingly described.[37] A recent population survey carried out in Italy (the Gubbio study) involving 5376 individuals showed that, up to the age of 64 years, hypertensive men were more markedly obese than normotensive men, whereas in women the prevalence of obesity was higher in hypertensive women than in normotensive women at all ages.[38]

In addition to the association with hypertension, increased BMI is associated with insulin resistance as well.[39] A study in Saudi population has shown an increased association of diabetes and hypertension with BMI, starting at a BMI as low as 21 kg/m^2.[40] This finding is consistent with another study.[25]

Hypertension frequently exists at time of diagnosis of T2D and is associated with Met S.[41] In the present study it was found that hypertension increases the severity of hyperglycemia in T2D. The FPG was significantly higher among the concurrent T2D/Ht subjects (11.84 ± 3.63 mmol/L) than among lone T2D subject (9.53 ± 2.97 mmol/L) ($p< 0.001$).

Vice versa, we found that the presence of T2D together with Ht makes systolic

and diastolic pressures significantly higher than them in lone Ht [160.17±17.5 / 96.68±7.44 mmHg] vs. [154.16 ±10.93 / 93.79 ± 4.14 mmHg] (p<0.001), a result that is consistent with the other study.[42] This could be attributed mainly to the common genetic and environmental factors promoting both T2D and Ht in addition to that Insulin resistance, increased tissue inflammation and reactive oxygen species production resulting in endothelial dysfunction, increased tissue renin angioten- aldosterone system, and increased sympathetic nervous system.[43]

In fact, in T2D subjects, the benefits of tight blood pressure control may be even greater than the benefits of a more intensive glycemic control, as shown in the UK Prospective Diabetes Study (UKPDS) study.[44] Blood pressure and glycemic control of the patients, in general, were poor in spite of having been commenced on treatment. Poor blood pressure control among these patients may be attributed to poor compliance. The possible explanation for poor glycemic control may be attributed to the long duration of T2D (for many years as evident from history).

In regard to the lipidemic status, the target objective of our study, the lipidemic profiles of all the studied subjects were worse than that of the controls as all of the subgroups showed a significantly higher means of (TC, LDL-C, VLDL-C and TGs) and a higher TC/HDL-C ratio than the apparently healthy controls. These results are consistent with studies elsewhere in the world.

Hypertension and hyperlipidemia occur together more often than it is expected by chance. There is some evidence that hyperlipidemia itself may predispose to hypertension and that lipid-lowering interventions may have a beneficial effect on blood pressure, or at least on vascular reactivity.[45] Hypertension and T2D are frequently coexist and they are independent risk factors for dyslipidemia.[46] Meanwhile, Zanchetti has reported that the prevalence of hypercholesterolemia was uniformly higher in hypertensive subjects compared with normotensive men except in the oldest age group.[37]

The TC level in T2D appears to be no more than in nondiabetic subjects. In the Framingham Heart Study,[47] 13% of men and 24% of women with diabetes mellitus had increased TC levels, compared with 14% of men and 21% of women without diabetes mellitus. In the same way, UKPDS found that the mean TC and LDL-C concentrations in those with T2D may not differ significantly from non-diabetic subjects.[48]

However, type 2 DM is usually associated with low plasma levels of HDL-C.[49] Chahil and Ginsberg in 2006 found that the prevalence of low HDL-C in those with diabetes mellitus was almost twice as high as the prevalence in non-diabetic individuals and the prevalence of high LDL-C in diabetes mellitus did not differ significantly from the rates in non-diabetic subjects. Also, the prevalence of high plasma TGs levels in individuals with DM was significantly higher than in those without DM.[47]

The precise pathogenesis of dyslipidemia in diabetes is not known;

however, evidence suggests that insulin resistance has a central role in its development.[12, 50] The main cause of the major features of diabetic dyslipidemia is the increased free fatty-acid release from insulin-resistant fat cells.[12, 50] The increased flux of free fatty acids into the liver in the presence of adequate glycogen stores promotes triglyceride production, which in turn stimulates the secretion of apolipoprotein B (ApoB) and VLDL-C, thus the impaired ability of insulin to inhibit free fatty-acid release from fat cells leads to enhanced hepatic VLDL-C production,[51] which correlates with the degree of hepatic fat accumulation.[52] Elevated serum TGs in diabetic patients with hypertension may be the result of increased hepatic production of TGs and/or a reduction in their catabolism.[53]

The abnormally increased TGs enrich high-density lipoprotein and low-density lipoprotein, leading to high levels of potentially atherogenic particles and low levels of HDL-C.[14] In addition, high TGs levels cause increased transfer of cholesteryl esters from HDL-C and LDL-C to VLDL-C via cholesteryl ester transfer protein, thus forming cholesteryl ester-depleted small dense LDL-C particles.[54] These small dense lipoprotein particles are taken up by arterial wall macrophages, resulting in atherogenesis.[55] Furthermore, HDL-C is a ready substrate for the increased activity of hepatic lipase which converts it into smaller particles that are readily cleared from the plasma.[27,56]

In the present study, the most risky figures of serum lipid (i.e. the highest levels of TC and TC/HDL-C ratio) were seen in the Met S with concurrent T2D/Ht, followed by concurrent T2D/Ht without Met S, then by lone T2D and lone Ht. This sequence shows that the existence of Met S is associated with propensity for more severe dyslipidemia.

On the other hand, the highest levels of TGs and VLDL-C and the lowest level of HDL-C were detected in the Met S where there is diabetes without hypertension. These findings suggest that dyslipidemia in Met S is a spectrum ranging between hypercholesterolemia and high TC/HDL-C ratio in one side where hypertension predominates and hypertriglyceridemia (and excess VLDL-C) in another side where diabetes predominates. In other words, in the Met S, diabetes is more likely to be present where hypertrigliceridemia predominates and hypertension is more likely to be present where hypercholesterolemia and high TC/HDL-C ratio predominate. Thus, the presence of hypertension and T2D together makes the lipidemic figures more badly than when they occur alone.

Total cholesterol, LDL-C and TGs in concurrent T2D/Ht subjects were found to be significantly higher than in lone T2D subjects. This is consistent with another study[59] while contradictory to another study.[42]

Serum HDL-C level was significantly more in lone T2D and lone Ht in comparison to T2D/Ht group. This is in agreement with other study.[58] The TC/HDL-C ratio is a sensitive and specific index of cardiovascular risk.[59] Apart from HDL-C, the ratio of TC/HDL-C is regarded as a predictor of CHD risk,

especially with values >6.0.[60] High density lipoprotein acts by enhancing the removal of cholesterol from the peripheral tissues and so reduces the body's cholesterol pool.

In conclusion, concurrent type 2 diabetes and hypertension adds the risk of exaggerating dyslipidemia in addition to their own potential cardiovascular risk. This is specially prominent when both are part of metabolic syndrome.

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پوختە

پروفایلێ چەوریا دناف خوینێ دا لجەم نەخوشیێن نەخوشیا شەکرێ ژ جورێ 2 و فشارا خوینێ یا زێدە هەین و پەیوەندیا وێ ب (المتلازمة الايضية)

پێشەکی و ئارمانج: تێکچوونا چەوریا دناف خوینێ دا ژ فاکتەرێن مەترسیێ نە کو دهێتە چارەکرن و ئەڤە پتر دگەل زێدەبوونا فشارا خوینێ و نەخوشیا شەکرێ ژ جورێ 2 یا هەی. ئارمانج ژ ڤەکولینێ دیراسەتکرنا پروفایلێ چەوریێ دناڤ خوینێ دا لجەم نەخوشیێن شەکرێ ژ جوری 2 و/یان فشارا خوینێ زێدە هەی و پەیوەندیا وێ ب (المتلازمة الايضية).

ریکێن ڤەکولینێ: 265 نەخوشیێن کو ب تنێ نەخوشیا شەکرێ ژ جورێ 2، 205 حالەت کو نەخوشیا شەکرێ ژ جورێ 2 دگەل زێدەبوونا فشارا خوینێ، 173 حالەت کو ب تنێ فشارا خوینێ یا زێدە هەی، هەروەسا 250 مروڤ کو چێ ژ ڤان نەخوشیا نەبوون هاتنە وەرگرتن ل نەخوشخانا زەهراوی ل میسل / عیراق دناڤبەرا حزیرانا تا کانونا ئێکێ 2011. بو ڤان پشکدارا پشکنینێن خوینێ هاتنە وەرگرتن.

ئەنجام: (المتلازمة الايضية) لدویف پێناسا ریکخراوا ساخلەمیا جیهانی هاتیە دیتن لجەم 34.4٪ ژ نەخوشیێن کو زێدەبوونا فشارا خوینێ هەی و 30.6٪ ژ نەخوشییێن شەکرێ ژ جورێ 2. سەنگا پشکدارا، ئاستێ کونترولکرنا ریژا شەکرێ، فشارا خوینێ کولیسترولێ کێم سەنگ، کولسترولێ گشتی، ریژا کولسترولێ گشتی بو کولسترولێ ژ سەنگا بلند دناف هەمی گروبا دا بلندتر بو ژ وان کەسان کو هاتبوونە وەرگرتن وەك کونترول. هەروەسا دیاربوو لجەم نەخوشیێن نەخوشیا شەکرێ ب تنێ هەی و بێن نەخوشیا شەکرێ دگەل زێدەبوونا فشارا خوینێ کو ریژا گلیسیرایدێ سیانی بلندتربوو بەرواوردکرن دگەل گروبێ کونترول، کولسترولێ سەنگ گران هاتە دیتن ب ریژەکا کێمتر لجەم نەخوشییێن کو نەخوشیا شەکرێ و فشارا خوینێ یا زێدە پێکڤە هەی بەراوردکرن دگەل گروبێ کونترول.

دەرئەنجام: هەبوونا نەخوشیا شەکرێ ژ جورێ 2 دگەل فشارا خوینێ یا زێدە مەترسیا زێدەبوونا تێکچوونا پروفایلێ چەوریێ یا هەی دگەل مەترسیا وێ لسەر بورییێن خوینێ بێن سەرەکی. و ئەڤە پترە ئەگەر (المتلازمة الايضية) هەبیت.

الخلاصة

واجهة شحوم مصل الدم في داء السكري نمط 2 و فرط ضغط الدم الشرياني وعلاقته بالمتلازمة الأيضية: دراسة الحالة– مجموعة القياس

خلفية واهداف البحث: يعد الخلل في شحوم الدم عامل خطورة يمكن معالجته. وهو كثيرا ما يتواجد مع فرط ضغط الدم الشرياني وداء السكري نمط 2 والمتلازمة الأيضية. الهدف من الدراسة هو دراسة خصائص واجهة شحوم مصل الدم لدى المصابين بداء السكري نمط 2 و/أو فرط ضغط الدم الشرياني بالنسبة إلى المتلازمة الأيضية.

طرق البحث: اشتملت دراسة الحالة – مجموعة القياس على 265 حالة داء سكري منفرد نمط 2 و205 حالة داء سكري نمط 2 وفرط ضغط الدم الشرياني المشترك، و 173 حالة فرط ضغط الدم الشرياني المنفرد و250 حالة غير مصابة كمجموعة قياس، في مستشفى الزهراوي الأهلي في الموصل– العراق، في الفترة ما بين حزيران إلى كانون الأول 2011. وقد أجريت فحوص الدم اللازمة وحللت إحصائيا.

النتائج: شكّلت المتلازمة الأيضية وفقا لمواصفات منظمة الصحة العالمية نسبة قدرها 34,4% من حالات فرط ضغط الدم الشرياني و30.6% من حالات السكري نمط 2. وكان معدل أعمار المشاركين في المجموعات كلها متقاربا، كما كانت معدلات مؤشر كتلة الجسم ونسبة السيطرة على سكر الدم وضغط الدم والكولستيرول واطئ الكثافة والكولستيرول الكلي ونسبة الكولستيرول الكلي/الكولستيرول عالي الكثافة في المجاميع كلها أعلى بشكل معتد إحصائيا من مجموعة القياس. كما ظهر أن مجموعة داء السكري نمط 2 المنفرد ومجموعة داء السكري نمط 2 المشترك مع فرط ضغط الدم تتصفان بزيادة الغلسريدات الثلاثية بالمقارنة مع مجموعة القياس. أما معدل الكولستيرول عالي الكثافة فقد وجد بنسبة قليلة معتدة إحصائياً في مجموعة داء السكري نمط 2 المشترك مع فرط ضغط الدم الشرياني بالمقارنة مع مجموعة القياس.

الاستنتاجات: إن اجتماع داء السكري نمط 2 مع فرط ضغط الدم الشرياني يضيف خطورة زيادة الخلل في واجهة شحوم مصل الدم فضلاً عن مخاطرهما الوعائية القلبية الأولية. ويبدو ذلك جلياً عند اجتماعهما في المتلازمة الأيضية.

RELAPSE OF CLUBFOOT DEFORMITY AFTER CORRECTION: EFFECT OF TYPE OF SPLINT IN UNILATERAL CASES

JAGER O. AHMED, MBChB, FICMS*

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ABSTRACT

Background and objectives Talipes equinovarus is the most common type of clubfoot deformities. Diagnosis is mainly clinical. The aim of treatment is to produce and maintain a plantigrade supple foot that functions well. The treatment is usually started by conservative method by serial manipulations and casting but may need surgical correction. After complete correction of the deformity, there is a significant risk of relapse, which can be prevented by using different types of splints for 3-4 years. The classical splint is Denis Browne Brace in which both feet are hold together in external rotation position. But the non compliance to this splint make adherence to it difficult. The aim of the study was to study incidence of relapse after using different types of splints after correction of clubfoot deformity.

Methods This retrospective study included 48 patients with unilateral clubfoot who wear a splint in their foot after their deformity had been corrected. Twenty six cases wear unilateral ankle foot orthosis while 22 cases wear foot abduction brace. They had been followed for about 1.3-3 years.

Results In this study the incidence of relapse in patients with unilateral clubfoot was 35.4%. thirteen (50%) out of 26 cases who used unilateral Ankle Foot Orthosis developed relapse, while 4 (18.2%) out of 22 cases who wear Foot Abduction Brace developed relapse. The results were statistically significant.

Conclusions The use of Foot Abduction Brace is associated with low risk of relapse because it maintains the foot in external rotation position and cause stretching of posteromedial soft tissue structures while unilateral Ankle Foot Orthosis can't hold this position.

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Key words: Splint, Clubfoot, Relapse

Club foot is the congenital abnormality of the foot in which there is a change in the shape and position of the foot.[1] The most common type is talipes equinovarus, in which the ankle become in plantar flexion, hindfoot in varus and midfoot with forefoot in adduction and supination.[2]

The incidence is 1-2/1000 live birth. The cause may be idiopathic, a germ defect, arrest of growth, neuromuscular disorders, postural deformity or may be in association with arthrogryposis, tibial deficiency and constriction rings. The soft tissue in the posteromedial aspect of calf, ankle and foot are short and underdeveloped. The bones are displaced and rotated medially.[2-4]

Diagnosis is mainly clinical, from its apparent change of different parts of ankle and foot with internal rotation of leg.[3] However, plain X-rays of the foot (both anteroposterior & lateral views) are also valuable for diagnosis and follow up of patient to assess progress after treatment.[2]

There is no international unique classification for talipes equinovarus

* *Lecturer of Orthopedics, Department of Surgery, Faculty of Medical Sciences, University of Duhok, Kurdistan Region, Iraq. E-mail: jager.doski@yahoo.co.uk*

deformity, but the simple one is that the deformity is either postural (flexible) or structural (resistant). The resistant type is characterized by thin calf, small high heel, deep creases in the posterior aspect of the ankle and the medial aspect of foot and usually associated with neuromuscular disorders as spina bifida or arthrogryposis. Both Pirani and Dimeglio have developed scoring systems based on the appearance of the foot in the position of maximal correction and they can be used to predict response to treatment and to monitor progress.[3,4]

The aim of treatment is to produce and maintain a plantigrade supple foot that functions well.[2] The treatment is usually started by conservative method, and as early as possible; starting by serial manipulations and casting weekly during first 6 weeks of life with or without percutaneous tenotomy of tendo Achilles to overcome the equinus. A number of centers now believe that most clubfeet can be treated by Ponseti casting method rather than surgery.[2,3,5]

If the patient doesn't improve with conservative method or if present late after few months of life, he may be in need for surgical treatment (by lengthening of short tendons and release of joint tethers as capsule and ligaments). The corrected position is immobilized in a plaster cast for 6-8 weeks.[2-4]

After complete correction of the deformity whether by conservative or operative method, there is a significant risk of relapse, which can be prevented by using different types of splints (such as ankle foot orthosis, Dennis Browne splint or foot abduction brace, …) for 3-4 years or till active dosiflexion and eversion are established.[2,5,6] In case of bilateral club feet, usually the two feet splints are connected together by a metallic bar to maintain them in external rotation. While in unilateral clubfoot, there is a controversy about the type of splint, whether by a simple ankle foot orthosis (AFO) or using other splints that hold both feet together by a bar because of orthosis noncompliance by the family (anxiety about the damage done to the normal foot during treatment of unilateral clubfoot with Denis-Browne splint) which make adherence to the splint difficult and patient resist to use it.[6-11]

The aim of this study is to compare the incidence of relapse in patients with unilateral clubfoot after complete correction of their foot deformity, between those who use unilateral ankle foot Orthosis(UAFO) and those who use the standard foot abduction brace (FAB) (i.e. two AFO connected together by a metallic bar and are in external rotation position).

METHODS

This is a retrospective comparative cohort study which had been conducted in Duhok city which is located in Kurdistan region at the north of Iraq. In this city there is a special medical center for children disabilities named Early Detection of Childhood Disability Center. In this center there is a special sector for paediatric orthopaedic patients which receive referred cases from other hospitals and primary health centers since 1998. Each

patient is examined by a specialist of orthopedic surgery, then investigations done if needed and lastly decision of treatment is made. These data are documented in a special file for each patient for follow up and for any statistical survey.

This study includes those patients with unilateral clubfoot who had been treated in this center at the period between 2005-2011. They were 48 patients, 31 male and 17 female; 27 of them had the deformity in their right foot while 21 in left. Thirt-eight of them had been treated conservatively by serial manipulation and casting, while 10 patients had been operated by postero-medial release. All patients had been followed after complete correction of their foot deformity by a splint to prevent relapse. Twenty-six of them wear unilateral ankle foot orthosis (UAFO) while 22 patient use foot abduction brace (FAB = 2 AFO connected together by a metallic bar in external rotation position; 70^{o} for the affected side and 30^{o} for the normal side). Those patients wear their splint continuously day and night for the first three months, then only during the sleep time for the remaining period. They had been followed for about 1.3-3 years. Relapse was considered if there is any inversion or medial deviation of any part of the foot.

Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) software. Results were presented as frequencies and percentages. P value was identified by using chi-square test. P value of less than 0.05 was regarded as statistically significant.

RESULTS

From the 48 patients who had been included in this study, 31 were male and 17 were female. Twelve out of 31 (38.7%) of male cases developed relapse, while 5 out of 17 (29.4%) of female patients developed relapse. The difference was statistically not significant (P=0.519) (Table 1). Twenty-seven patients had club foot in their right foot versus 21 cases in left foot. Of those who had deformity in their right foot 40.7% developed relapse, while those who had deformity in their left foot 28.6% showed relapse. The P-value of chi-square was 0.382 which was statistically not significant (Table 2).

Table 1. Sex – relapse relationship

Sex	Relapse No. (%)	No relapse No. (%)	Total
Male	12 (38.7)	19 (61.3)	31
Female	5 (29.4)	12 (70,6)	17
Total	17 (35.4)	31 (64.6)	48

Table 2. Side – relapse relationship

Side	Relapse No. (%)	No relapse No. (%)	Total
Right	11 (40.7)	16 (59.3)	27
Left	6 (28.6)	15 (71.4)	21
Total	17 (35.4)	31 (64.6)	48

Those patient who use Unilateral Ankle Foot Orthosis (UAFO) were 26 patients, 13 of them develop relapse; 9 of them had only forefoot adduction, 2 of them had mild degree that didn't need any further interference while the rest were moderate or severe degree that necessitate lateral transfer of Tibialis Anterior tendon to base of 4th metatarsal. The rest 4 cases had significant ankle planter flexion and heel inversion that need revision of

postero-medial release surgery. While those patient who wear Foot Abduction Brace (FAB) which hold the feet in external rotation position were 22 patients, only 4 of them developed relapse and they were of mild degree forefoot adduction that didn't need any further surgery apart from one case who had moderate degree that require Tibialis Anterior lateral transfer (Table 3 and Table 4).

Table 3. Type of splint and relapse relationship

Type of splint used	Relapse No. (%)	No relapse No. (%)	Total
UAFO	13 (50)	13 (50)	26
FAB	4 (18.2)	18 (81.8)	22
Total	17 (35.4)	31 (64.6)	48

The p-value of chi-square equation of relapse between those who wear UAFO and those who used FAB was 0.022 which was considered statistically significant.

DISCUSSION

Recurrence of clubfoot deformity (i.e. relapse) after successful initial correction continues to be a common problem and is often caused by non compliance with wear of the traditional foot abduction brace.[11] Added to that, the anxiety of parents about the damage done to the normal foot during the treatment of unilateral clubfoot with the Denis Browne splint.[7] For this purpose, different modifications had been done to the original Denis Browne splint to make it more dynamic to protect the normal foot or to increase the compliance of patients specially for the kicking behavior of the child.[7,10,12] While others try to use unilateral AFO (with or without modifications) to improve the compliance with the splint in order to encourage the patient to wear it for 3-4 years.[6,8,11]

Findings of this study showed significant difference in the relapse rates between those who used unilateral ankle foot orthosis (50%) and those who wear the ordinary foot abduction brace (18.2%).the P value was < 0.05. Even those who used FAB and developed relapse had only forefoot adduction of mild degree which didn't require any further treatment apart from one case who required tibialis anterior lateral transfer; while those who used the UAFO and developed relapse had more severe degrees of foot deformity that 7 of them needed tibialis anterior lateral transfer and 4 cases required posteromedial release.

Table 4. Types of treatment for relapsed cases

Type of splint used	Number of cases	Relapse	Type of relapse		Type of treatment		
			Forefoot adduction	Ankle flexion and foot inversion	No treatment needed	TA lateral transfer	Revision of posteromedial release
UAFO	26	13	9	4	2	7	4
FAB	22	4	4	0	3	1	0
Total	48	17	13	4	5	8	4

From these results we conclude that use of UAFO increase the risk of relapse of clubfoot and if occur it is more sever. this can be explained by failure of UAFO to maintain the foot in external rotation position which is important for stretching of posteromedial soft tissue structures of ankle and foot.

We agree with Ponseti's believe that relapses are caused by the same pathology that initiated the deformity. It starts at the 3th month of intrauterine life, by unknown dysfunction in the posterior and medial aspects of the lower leg, ankle and foot. There is a decrease in size of the muscles, and an excess of collagen synthesis with retracting fibrosis in the medial and posterior tarsal ligaments, in the deep fascia, the tendo Achilles and the posterior tibial tendon. The period of dysfunction causing the deformity starting in the middle third of pregnancy lasts to the 3th or 4th year of life. Therefore; splinting for several months or years is indispensable to help prevent relapses. Unless the feet are splinted in firm external rotation, the pull of the retracting fibrosis in the ligaments of the medial aspect of the ankle and of the tibialis posterior and the toe flexors is strong enough to cause a recurrence of the deformity in most feet.[13]

Other studies showed similar results. George et al found that recurrence (the need for further casting or operation) was observed in 31.4% of feet who used UAFO, six of them respond favorably to a further period of serial casting and three responded to tibialis anterior transfer and only two required a traditional posteromedial release.[8] While Janicki et al found that recurrence requiring additional treatment occurred in 83 % of AFO group and 31% of the group with boots and bar ($P< 0.001$). Those who developed relapse in AFO group, 11 of them required tenotomy or limited posterior release, but 14 of them needed posteromedial release or midfoot osteotomy; while those of boots and bar group, 5 required tenotomy and only 2 need posteromedial release.[14]

In this study, 17 cases developed relapse; 38.7 % of male cases developed relapse and 29.4% of female cases developed relapse. The greater number of relapsed cases was male but this can be returned the greater number of male patients in our study. However; the difference between both groups was statistically not significant. So we conclude that sex factor had no relation with relapse of clubfoot deformity.

According to the side affected,; from those who had clubfoot deformity in their right foot 40.7% developed relapse while 28.6% were in left. The difference between both groups although was high statistically was not significant. So we conclude that side of deformity did nothing with relapse.

In conclusion the use of splint after correction of club foot deformity is important to prevent relapse. The type of splint should maintain the foot in external rotation position for stretching of posteromedial soft tissue structures. This position can be achieved by holding both feet in boots or 2 AFO connected together by a bar in external rotation position. Unilateral AFO can't hold this position; therefore, it associated with high incidence

of relapse of clubfoot deformity and if this occurs it is more sever and need more difficult treatment. Sex of patient and side of foot affected has no relation with relapse.

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پوخته

ڤەزڤرینا خوارنا پیی یا زکماکی پشتی راستڤەکرنی: رولی جوری گولتەی لدەف نەخوشین نەریکی هەی د پییەکیدا

پێشەکی و ئارمانج: خوارنا پیی بەلاڤترین جوری نەریکیا زکماکیا پییە، کو گوزەکا پیی یا ڤەکیشایە بو خواری و پی یی وەربادایە بو ژ ناڤدا. هەردەم دەستنیشانکرن ل بالگەی دهیتە کرن. ئارمانج ژ چارەسەریی بدەستڤەهینانا پییەکی راست و نەرمە. هەردەم چارەسەری بریکا پارازتنی دەستپیدکەت د زنجیرەکا رونیشتنین چارەسەرییدا ژ لڤاندن و راستکرنا گەهین پیی دەستپیدکەت و پاشی جبسکرنا حەفتیانە بو ماوی شەش حەفتیان، و هەکە ڤی ریکی مفانەبو یان گیروبو، وی دەمی نشتەگەری پی دڤیت. پشتی راستکرنا نەریکیا پیی ب تمامی، ریژەکا ڤەزڤرینا نەریکیی پەیدادبیتەڤە. گەلەك جورین گولتان هەنە دا ئەڤ چەندە روی نەدەت، یی ژ هەمیا باشتر گولتی (دنیس براون) ە کو پیی ب شیوی بادای بو ژ دەرڤە دراوەستینیت. بەلی دیاردا بیزکرنی ژ ڤی گولتەی هەیە ژ بەر هندی بکارهیناناوی یا ب زەحمەتە.

ریکێن ڤەکولینێ: د ڤی لیکولینا پاشڤەیی، 48 زاروك ئەڤیت خوارنا پیی یا زکماکی د پییەکیدا هەی و هاتینە چارەسەرکرن و گولتە ب کارئینان. بیست و شەشان گولتی کت پی ب کار ئینا و بیست و دووا گولتی جوت پی ب کار ئینا.

ئەنجام: د ڤی لیکولینیدا ریژەیا ڤە زڤرینا نە ریکیا پیی پشتی چارەسەرکنی ل دەف وان نەساخین نەریکیا زکماکیا پیی د پییەکیدا هەی 35.4٪ بو. ئەڤیت گولتی کتپی بکارهینای 50٪ ژوان ڤەزڤرینا نەریکیا پیی لی پەیدابو. بەلی ئەڤیت گولتی جوتپیی بکارهینای 18.2٪ ژوان ڤەزڤرینا نەریکیا پیی لی پەیدابو. ئەڤ ئەنجامە ژلایی هژماریڤە یی بەرچاڤبو.

دەرئەنجام: ژ ئەنجامی ڤی لیکولینی بو مە دیاردبیت کو گولتی جوتپی ریژەکا کیما ڤەزڤرینا نەریکیا زکماکیا پیی هەیە، چونکو پیی دهیلتە بادای بو ژدەرڤە و دەمارین پشت و بنی گوزەکا پیی ژیکدکیشیت، کو گولتی کتپی نەشیت ڤیکاری بکەت.

الخلاصة

ارتداد اعوجاج القدم الولادي بعد تعديله: تأثير نوع المسند في حالات احادية الجانب

خلفية واهداف البحث: اعوجاج القدم هو من اكثر انواع تشوه القدم الولادي شيوعا، حيث يكون فيه الكاحل منقبضا للاسفل و القدم منحرفا للداخل. التشخيص دائما يكون سريريا. الهدف من العلاج هوالحصول على قدم مستوية و مرنة. يبدأ العلاج عادة بطريقة تحفظية عن طريق سلسلة جلسات من تحريك مفاصل القدم و تعديلها ثم وضعها في قالب من الجبس اسبوعيا لمدة ستة اسابيع. واذا لم يستجب المريض لهذه الطريقة من العلاج او حضر متأخرا، فسيحتاج الى تداخل جراحي. بعد تعديل تشوه القدم بشكل كامل، هناك نسبة مميزة لعودة التشوه و للحيلولة دون ذلك هناك مساند متعددة اهمها مسند دنيس براون و الذي يمسك القدم في وضعية الدوران للخارج. و لكن هناك ظاهرة عدم اذعان لهذا المسند مما يجعل الالتزام باستخدامه مهمة صعبة. الهدف من البحث هو ملاحظة نسبة ارتداد الاعواج مع استخدام انواع مختلفة من المساند.

طرق البحث: هذه الدراسة الرجعية شملت 48 طفلا من المصابين باعوجاج القدم الولادي احادي الجانب الذين استخدموا انواع مختلفة من المساند بعد تعديل اقدامهم المشوهة. ستا وعشرون طفلا استخدموا مسن القدم الاحادي و اثنان و عشرون استخدموا المسند الثنائي. فترة استخدام المسند تراوحت بين 1.3–3 سنوات.

النتائج: في هذا البحث نسبة ارتداد التشوه للقدم بعد العلاج في المرضى الذين كانوا يعانون من اعوجاج القدم الولادي في قدم واحدة كانت 35.4%. المرضى الذين استخدموا مسند القدم الاحادي فان 50% منهم اظهروا ارتدادا لاعوجاج اقدامهم؛ بينما الذين استخدموا مسند القدم الثنائي فقط 18.2% منهم اظهروا ارتدادا لحالتهم. و كانت هذه النتائج من الناحية الاحصائية مميزة.

الاستنتاجات: نستنتج من نتائج هذا البحث ان مسند القدم الثنائي يرتبط بنسبة ضئيلة من ارتداد اعوجاج القدم الولادي وذلك لانه يحافظ على وضعية دوران القدم للخارج و يؤدي الى مط الانسجة الرخوة في الجهة الخلفية و الداخلية للكاحل والقدم، وهو الشيء الذي لا يستطيع مسند القدم الاحادي القيام به.

TESTICULAR BIOPSY IN AZOOSPERMIC MEN: A STUDY OF THE MORPHOLOGICAL PATTERNS IN DUHOK CITY AND AN ATTEMPT TOWARD THE DEVELOPMENT OF A NEW EVALUATION SYSTEM

ALAA H. RAZAK, MBChB, MSc (Path.), MIBMS*

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ABSTRACT

Background and objectives Infertility is a common medical problem and about (10-15%) of infertile men had azoospermia. Testicular biopsy in these men has not only a diagnostic role but also a therapeutic one. The frequencies of histopathological patterns are greatly varied in different studies. This is probably attributed to the selection criteria of patients or due to the absence of a uniform system for evaluation of these biopsies. The present work is a cross sectional study which included bilateral testicular biopsy obtained from (80) azoospermic patients over a period of three years from 2009-2011. Biopsies were obtained from Histopathology Laboratory of Azady Teaching Hospital and some private laboratories in Duhok City. The objectives of this study are to determine the histopathological patterns seen in the testicular biopsies in azoospermic male and to develop a uniform system for evaluation of these biopsies.

Methods This study included (80) infertile men with azoospermia. Bilateral testicular biopsies were examined and the histopathological findings were classified into three major categories; in the first one, sperms are seen regardless their number, in the second category, there is maturation arrest and in the last category, there is absence of germinal cells regardless the presence of Sertoli cells.

Results This study revealed that (37.5%) of the included patients had sperms in their testicular biopsies, (33.7%) had maturation arrest and (28.7%) had no germinal cells.

Conclusions There are great dissimilarities in the patterns of testicular biopsy among different workers. In the present work, the frequencies of normal spermatogenesis (regardless the number of sperms) (37.5%), maturation arrest (33.7%) and absence of germ cells (28.7%) are nearly equal. While none of our patients had generalized fibrosis. The criteria for selection of azoospermic patients who will be subjected to testicular biopsy need to be standardized. The proposed evaluation scheme is informative and reproducible among pathologists and easily understandable by the surgeon.

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Key words: Azoospermia, Testicular biopsy, Maturation arrest

Male infertility contributes to more than half of all cases of childlessness; yet, it is a reproductive health problem that is poorly studied and understood.[1] About 10%-15% of infertile men present with azoospermia.[2] Azoospermia may be obstructive (blockage of the genital ducts) or non-obstructive (a lack of testicular production). The distinction is based on an ensemble of clinical, spermiological, hormonal, ultrasound, genetic and histological data. Azoospermia is the main indication for testicular biopsy for therapeutic and diagnostic purposes.[3]

Biopsy specimens from infertile male with total lack of sperms (azoospermia) usually show one of the following

* *Assistant Professor in Pathology, Pathology Department, School of Medicine, Faculty of Medical Sciences-Duhok University, Duhok-Iraq. E-mail: ala_hani@yahoo.com. Telephone: 009647504801310*

conditions:

1- Germ cell aplasia (29%).
2- Spermatocytic arrest (26%)
3- Generalized fibrosis (18%)
4- Normal spermatogenesis (27%).[4]

Different studies in this field had demonstrated great dissimilarity in the frequencies of these patterns. [5,6]

The assessment of spermatogenesis in a testicular biopsy is a key for proper evaluation, and until now, the Johnson score is widely used by the pathologist. The method of Johnson applies a score of 1 to 10 for each tubule cross section examined. The criteria are as follows: 10, complete spermatogenesis and perfect tubules; 9, many spermatozoa present but disorganized spermatogenesis; 8, only a few spermatozoa present; 7, no spermatozoa but many spermatids present; 6, only a few spermatids present; 5, no spermatozoa or spermatids present but many spermatocytes present; 4, only a few spermatocytes present; 3, only spermatogonia present; 2, no germ cells present; 1, neither germ cells nor Sertoli cells present.[7]

The aims of this study are to find the histopathologic patterns of testicular biopsy in azoospermic men in Duhok City and an attempt to develop an applicable, reproducible and easily understandable evaluation system.

METHODS

This study included bilateral testicular biopsies obtained from (80) infertile azoospermic men.

Biopsies were fixed in Bouin's solution and submitted for histopathologic study using the paraffin block method; sections were made from these blocks and the slides were stained by H&E stain.[8]

The following parameters were assessed:

1- Density of the seminiferous tubules in the section
2- Spermatogenesis
3- Basement membrane of the seminiferous tubules
4- Leydig cells
5- Sertoli cells
6- Any other findings

Then patients were grouped into 3 categories:

1- Obstructive cases when spermatozoa are identified in the section no matter how much are their number. These include cases of Johnson's score 10, 9 and 8.
2- Maturation arrest:
    a. Early arrest when no spermatids are seen, i.e only germ cells and spermatocytes. These included cases of Johnson's score 3, 4, 5.
    b. Late arrest when spermatids are seen regardless their number. These include cases of Johnson's score 6 and 7.
3- Absence of germ cells. These include cases of Johnson's score 1 and 2.

All the other parameters were looked for and mentioned. When there were no germ cells, further information were described to help reaching the final diagnosis like tubular hyalinization, presence or absence of Sertoli cells and Leydig cell status.

RESULTS

This study included (80) patients. Their ages ranged from (17-39) years over a period of three years from 2009-2011.

Histopathologic study of these biopsies revealed the following:

1- Presence of spermatozooa – regardless their number- in the seminiferous tubules was detected in (30) patients (37.5%) (Figure 1).
2- Maturation arrest was found in (27) patients (33.75%) (Figure 2).
3- Absence of germinal cells was noted in (23) patients (28.75%) (Figure 3 and 4).

Table 1 summarizes the histopathological findings of testicular biopsies in the included eighty patients. Only in one patient, there was a different scoring of the testes.

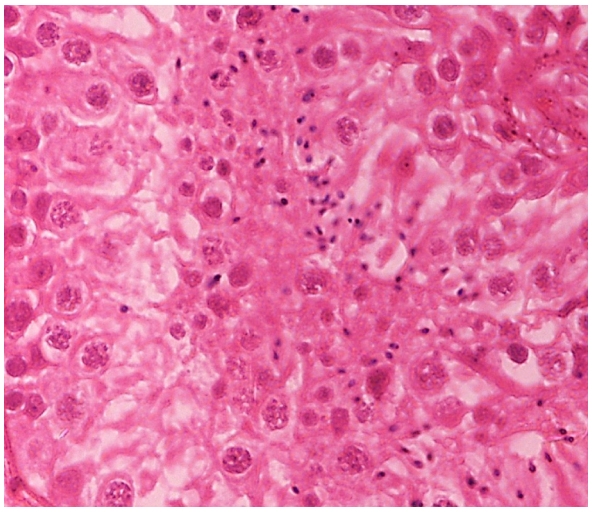

Figure 1. Complete spermatogenesis

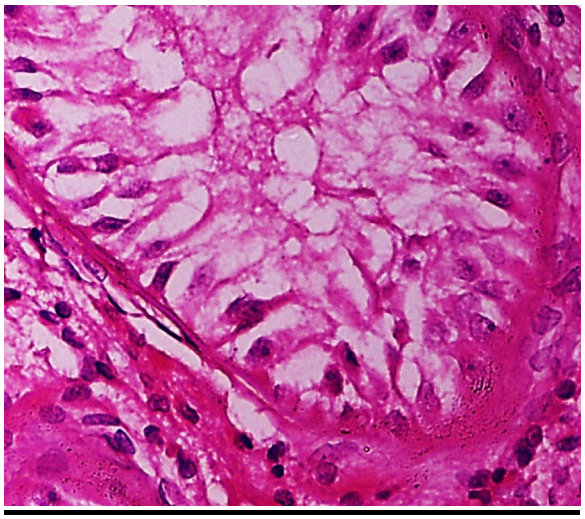

Figure 3. Sertoli cell only syndrome

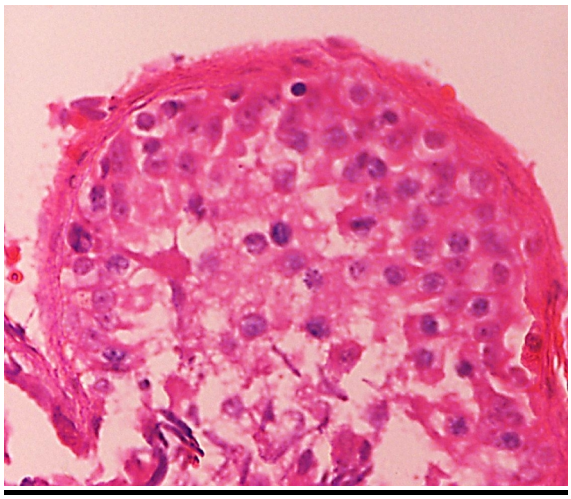

Figure 2. Maturation arrest, many germ cells and spermatocytes are seen but no spermatids are seen

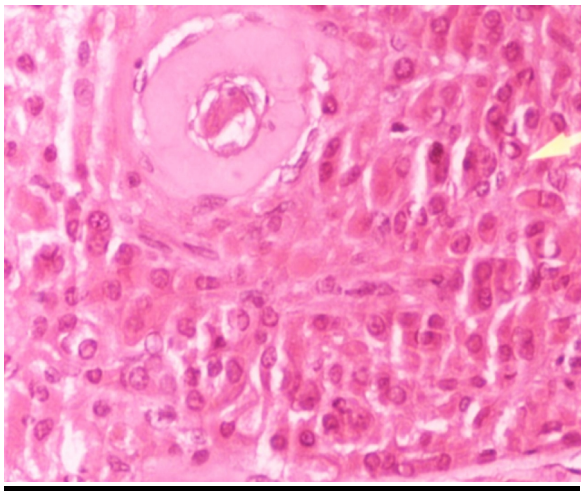

Figure 4. Tubular hyalinization and Leydig cell hyperplasia.

Table 1. summarizes the results in details

Histopathological findings		Johnson's score	No. of patients (%)	Tubular hyalinizatiom	Leydig cell hyperplasia
Presence of spermatozooa		9	26		
		8	3		
		8 & 5*	1		
	Total		30 (37.5)		
Maturation arrest	Late	7,6	12		
	Early	5,4,3	15		
	Total		27 (33.7)		
Germ Cells	Sertoli cell only	2	19		
	germ cell & Sertoli cell	1	4	3	3
	Total		23 (28.7)		

**This was the only patient who had different scores in the left testis and the right testis.*

DISCUSSION

The incidence of male infertility and the subsequent histological findings in testicular biopsies differ significantly from one part of the world to another due to several underlying etiological factors including social habits, genetic causes and environmental conditions such as underlying infections, chemicals, radiation and exposure to heat.[9,10] In our opinion, two additional and seemingly very important factors –which govern the results of histopathological examination of testicular biopsy and the subsequent frequencies of each pathology – should be mentioned here; the first one is the criteria of selection of azoospermic patients whom are candidates for testicular biopsy and the second one is the way of evaluation of the histological sections. If surgeons exclude those with small fibrotic testes and those with elevated serum FSH levels, the frequency of generalized testicular fibrosis will be diminished significantly. Furthermore, there is no standard quantitative method to assign that a given patient has hypospermatogenesis, a term which we exclude it in the proposed evaluation system, since the advances in the reproductive technology allow even those with very low sperm count to be fathers.

Layla Abdullah and Nabeel Bondagji studied hundred testicular biopsies and found that (14%) of their patient had normal spermatogenesis and (29.29%) had hypospermatogenesis and only (12%) had maturation arrest,[11] while in the present work we found that the majority of patients with sperms in their tubular sections had normal spermatogenesis (Johnson's score 9) and they represent (26) of (30) patients. Maturation arrest was detected in (37.5%) of our patients and this represents the largest category. Clearly these results are influenced by the selection criteria of the surgeon.

Ahmed et al had also reported different figures, they found that

hypospermatogenesis accounted for (68.2%) of their patients and (11.4%) had normal morphology.[12]

Al-Rayess and Al-Rikabi used a different evaluation scheme and also gave different results. They found that (90) of (230) patients to have germ cell aplasia or germ cell aplasia with focal spermatogenesis.[13]

Al-Samawi et al had a comparable result to our study and they reported germinal cell aplasia in (33.8%) of their patients and maturation arrest in (23.9%).[14] These different results necessitate the utilization of standard criteria for selection of patients and for a uniform evaluation scheme. And we think that the proposed scheme in the present work may fulfill these needs.

The idea behind using the proposed evaluation scheme belongs to its simplicity, its understandability by the surgeon, reproducibility among the pathologists and the fact that it provides an idea about the present hope or the future hope for these unlucky patients to be child fathers in the future.

When spermatozooa are present in the biopsy, it is possible for these patients to be candidates for in vitro fertilization and intracytoplasmic sperm injection (IVF and ICSI). While if there no germ cells, they can be told that they have no hope. However, the big dilemma is with those who have maturation arrest. For those patients with spermatids, they may have a chance of child fathering in the future, since these cells (The spermatids) are haploid and they had passed the second meiotic division,[15] so probably, in the near future with the advances of reproductive technology, these cells may be used for ICSI after artificial induction of maturation. And this is the reason why we grouped them together under the name of late maturation arrest. However, this is still an idea or a dream which needs a lot of work to be achieved. While if only germ cells and spermatocytes are seen in the biopsy – these cells are diploid or haploid with double chromatin material - so there is no benefit from harvesting them, since their genetic material is not suitable for fertilization.

There is a great dissimilarities in the patterns of testicular biopsy among different workers and in the present work the frequencies of normal spermatogenesis (regardless the number of spermatozooa), maturation arrest and absence of germ cells are nearly equal. Also this study clarifies the need for standardized criteria for selection of azoospermic patients whom will be subjected to testicular biopsy. And finally the use of the proposed evaluation scheme which is informative and reproducible system by the pathologists and easily understandable by the surgeon is to be adopted.

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پوختە

پارچەکا گونی ژ وان زەلامان یێت کو سپێرما ناچێکەن

پێشەکی و ئارمانج: نەزوکی ئاریشەکا ساخلەمیا بەربەلاڤە و(10–15٪) ژ وان زەلامان کو سپێرم نینن دناڤ ئافا زەلامیدا ,پشکنینا پشکەکێ ژ گونی هەنگافەکە بو دیتنا چارەسەرکرنی. جیاوازی دریژەیا بەریخودانادنا هەیە د وەرگرتنا وی پارجا ژ گونی ڤە وەرگرتنی دڤەکولینین جوراوجوردا رەنگە زڤرینا ڤان جیاوازیان ڤەگەرین ژبەر ریکا هەلبژارتنا نەساخین پالیراو بۆ ڤەکولیاری یان ژبەر نەبونا سیستەمین ئیك لا کەر بو هەلسەنگاندنا پارچەیا گونی. ئەڤ ڤەکولینە ل سەر 80 زەلامان هاتیە ئەنجامدان و کومکرن دماوی 3 سالا دا ژ (2009–2011) ل تاقیگەها پشکنینا پوشەکی(histopathology) ل نەخوشخانا ئازادی یا فیرکرنی ل هندەك تاقیگەهین تایبەت ل باژیری دهوکی. مەرەم ژ ئەنجامدانا ڤان ڤەکولینا ژبو دیتنا سیستەمەکی نوی یی گونجای بو هەلسەنگاندنا پارچەیا گونی.

ریکێن ڤەکولینێ: ئەڤ ڤەکولینە 80 نەخوشین توشی نەبوونا توڤی زەلاما بخوڤە دگریت و پارچە خاتینە وەرگرتن ژ هەردوو گونین وان و شیوەیین پوشەکی هاتینە دابەشکرن بو 3 جوران: یەکەم توڤ دگونادان هەنە ژبلی بەرەخودانا ژمارا وان, دووەم راوەستیانا پیگەهشتنا خانین بەرهەم هینەر بو ڤان توڤان, سییەم خانین بەرهەم هینەر بو ڤان توڤان نین.

ئەنجام: دیاردبیت ژ ڤەکولینی کو) 37.5٪ ژڤان نەساخان کو توڤ یین دگونادا هەین و (33.7٪) راوەستیانا پیگەهشتنی هەیە و (28.7٪) خانەیین بەرهەم هینەر بو سپیرمان نینن.

دەرئەنجام: جیاوازیین مەزن دئەنجامین ڤەکولینین جیاواز دا دەربارەی ڤی بابەتی هەنە و دیاردکەت کو ئەڤ ڤەکولینە دریژەیا هەر سی جوران ئەوە کو توڤ دگونیدان هەنە بریژەیا (37.5٪) و پیگەهشتن نینە بریژەیا (33.7٪) و نەبونا خانین بەرهەم هینەر بو توڤا بریژەیا (28.7٪) کو ئەڤ ریژەیە نزیکی ئیکن و نەهاتیە دیارکرن لدەڤ هیچ نەساخەکی کو پیچ بوونا گونی ب تەمامی ژبەر هندی دڤیت بناتەك بهیتە دانان ژبو هەلبژاردنا نەخوشی بو ڤی کرداری و بکارئینانا سیستەمی بەراوردکرنی بسەناهیە وتایبەتمەندین هیستوپاتولوجی ریك ئیخستینە لسەر و بساناهیە بو تیگەهشتنا نوژدارین نشتەرگەر.

الخلاصة

خزعة الخصيه في الرجال المصابين بانعدام الحيامن: دراسه للانماط الشكليه في مدينة دهوك و محاوله لوضع نظام جديد للتقييم

خلفية واهداف البحث: العقم مشكله طبيه شائعه و (10–15%) من الرجال لاتوجد لديهم حيوانات منويه في السائل المنوي. فحص خزعة الخصيه له دور تشخيصي و علاجي. تختلف نسب المشاهدات النسيجيه لعينة الخصيه كثيرا في دراسات مختلفه. و قد يعود سبب الاختلاف الى طريقة اختيار المرضى المرشحين لاجراء هذه العمليه او الى عدم وجود نظم متفق عليه لتقييم خزعة الخصيه. ان هذا البحث هو دراسه لخزع الخصيتين ماخوذه من ثمانين مريضا مصابين بانعدام الحيامن في السائل المنوي جمعت خلال ثلاث سنوات من 2009–2011 في مختبر الفحص النسيجي لمستشفى ازادي التعليمي و بعض المختبرات الاهليه في مدينة دهوك. وتهدف هذه الدراسه لمعرفة الانماط الشكليه لنسيج الخصيه في هؤلاء المرضى و لوضع نظام جديد لتقييم خزع الخصيه.

طرق البحث: شملت هذه الدراسه (80) مريضا لديهم انعدام الحيوانات المنويه في السائل المنوي و اخذت منهم خزع من كلا الخصيتين و تم تصنيف الانماط النسيجيه الى ثلاثة انواع. النوع الاول لديهم حيامن في الخصيه بغض النظر عن عددها و النوع الثاني لديهم توقف نضج الخلايا المنتجه للحيامن و النوع الثالث لاتوجد لديهم الخلايا الام المولده للحيامن.

النتائج: اظهرت الدراسه ان (37.5%) من المرضى المشمولين بالدراسه لديهم حيامن في الخصيه و (33.7%) لديهم توقف النضج و (28.7%) ليس لديهم خلايا ام مولده للحيامن.

الاستنتاجات: هناك فروقات كبيره في نتائج الدراسات المختلفه حول هذا الموضوع. اظهرت هذه الدراسه ان نسب الانماط الثلاثه وهي وجود الحيامن في الخصيه (37.5%) و توقف النضج (33.7%) و غياب الخلايا الام المولده للحيامن (28.7%) هي نسب متقاربه. و لم يشخص اي مريض بالتليف العام للخصيه. و لذلك يجب وضع اسس لاختيار المرضى لهذه العمليه. و ان استعمال نظام التقييم هذا سهل و يتفق عليه اختصاصيي التشخيص النسيجي و سهل الفهم بالنسبه للجراحين.

RECORDING ERRORS IN CONVENTIONAL ELECTROCARDIOGRAPHY IN CLINICAL PRACTICE IN DUHOK CITY, IRAQ

SUAD N. JAAFAR, MBChB, Higher Diploma in Family Medicine*
LARS A. PESCHKE, M.D., ABFM**
QAYSER S. HABEEB, MBChB, MSc, DIM***

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ABSTRACT

Background and objectives The 12-lead electrocardiogram is a useful tool to record and analyze the electrical activity of the heart. If correct recording procedure and high quality standards are not assured, registration mistakes may lead to potentially serious diagnostic errors. As far as we know, this subject has not been studied in Kurdistan Region of Iraq. This study was conducted to investigate the electrocardiographic recording er-rors in clinical practice in Duhok City..

Methods Data were collected from January 20 to May 31, 2011. A cross-sectional design was adopted to assess electrocardiogram records at Azadi Hospital, Zanest Family Medicine Center, and private clinics in Duhok City. All records were assessed for types of errors in accordance with predefined assessment criteria.

Results The total number of recording errors was 6858, which constituted around one fifth (20.8%) of the maximum possible number of errors (33,000). Most major errors were seen at Azadi Hospital (3.3%), followed by private clinics and Zanest Center (2.4% and 1%, respectively). Minor non-technical errors in private clinics (17.2%) were almost double those of Azadi Hospital and Zanest Center. About one third (34.2%) of the total number of records contained at least one major error, 75% contained at least one minor technical error and almost all records (98%) contained at least one minor non-technical error.

Conclusions Electrocardiographic recording errors are a problem in the three sectors of clinical practice in Duhok City, more so in the governmental institutions. Immediate and decisive measures should be put into action to prevent potentially serious sequelae.

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Key words: Electrocardiography, Recording errors, Iraq

The 12-lead electrocardiogram (ECG) is a diagnostic test used in many different clinical settings.[1] It is a noninvasive technique that is inexpensive, simple, reproducible, and can be recorded rapidly with usually easily obtainable portable equipment. Electrocardiography records the heart's electrical activity, also known as action potential, as wave forms. By interpreting these wave forms or ECGs accurately one can identify physiological and pathological phenomena,[2,3] such as rhythm disturbances, conduction abnormalities, electrolyte imbalances, the size of the heart chambers, and the heart position in the chest. It also aids in diagnosing and monitoring conditions such as myocardial infarction, pericarditis,

* *Primary health care doctor at Shahidan Primary Health Care Center, Duhok, Iraq*
** *Lecturer, Instructor of medical students and family medicine resident doctors at the Department of Family and Community Medicine, Duhok Medical School, University of Duhok, Duhok, Iraq*
*** *Professor, Department of Family and Community Medicine, Duhok Medical School, University of Duhok, Duhok, Iraq*

Correspondence author: Suad N. Jaafar. Email: SuadNoori@ymail.com. Phone: 00964-750-474-8852

the effects of medications, and the function of an artificial pacemaker.[4] The 12-lead ECG is a very useful investigation, however, it can be misleading, particularly when interpreted out of context. The value of standard 12-lead ECG depends upon the accuracy of its recording. Correct recording procedure is essential for proper analysis.[5] Common errors in clinical electrocardiography include inaccurate lead placement, inappropriate serial comparisons using different lead sets, lead wire reversals, inappropriate filter settings, and excessively noisy signals.[6]

Artifacts are defined as ECG abnormalities that may be due to sources other than the electrical activity of the heart. Failure to correctly distinguish between arrhythmia and artifact can result in misdiagnosis and unnecessary interventions.[7,8] Errors committed during the various steps of recording procedure may have different effects on the proper interpretation of the ECG depending on their types, extent, and frequency. Major errors may make the record totally useless or a source of wrong diagnoses even for experts like incorrect lead connection.[5] Minor non-technical errors have only minor effects on the quality of the ECG tracing like errors in documenting identity information; such errors usually do not hinder interpretation significantly.[9] In between these two extremes are technical errors that can affect the shape, clarity, and stability of different wave forms and segments resulting in variable effects on the quality of the ECG tracing depending on the type, number, and extent of the errors. They can be a source of wrong diagnosis, especially for the less experienced. Damping errors and baseline instability are examples of such errors.[10]

Study Rationale

Due to the lack of studies that address this problem in Kurdistan Region, this study was designed to investigate electrocardiographic recording pitfalls in clinical practice in Duhok.

Objectives of the Study

The aim of the study was to investigate types and frequencies of electrocardiographic recording errors as they occur in the three sectors of clinical practice in Duhok City, namely hospitals (represented by Azadi hospital), primary health centers (represented by Zanest Family Medicine Center), and the private sector (represented by private clinics of senior physicians). To achieve that aim, frequencies of the different types of errors between the different sectors were compared.

METHODS

A cross-sectional study was adopted to achieve the study objectives. A total of 2750 ECG tracings were collected from January 20 to May 31, 2011, from designated places of the three sectors of clinical practice in Duhok City. At Azadi Teaching Hospital 1000 tracings from the outpatient cardiology clinic, 750 tracings from the intensive care unit, and 400 tracings from the medical emergency department which made a total of 2150 tracings; at Zanest Family Medicine Center 300 tracings; and at private clinics 300 tracings.

Recording errors were categorized into three main groups; each group was further subdivided into four types according to their potential effects on ECG interpretation as follows:

1. Major errors (errors in the core steps): Connection of leads, calibration setting, speed adjustment, number of recorded leads.
2. Technical errors (errors in the technical adjustments): Stylus artifact, damping, interference control (muscular and electrical), baseline stability.
3. Minor Errors (errors in non-technical complementary steps): Number of beats per lead, long strip recording, lead label, and identity information. If an ECG record contained any one of them, it was considered faulty.

All data were entered into Microsoft Office Excel 2007 and transferred to PASW Statistical Editor for statistical analysis. The chi square (χ^2) test was used to test differences between categorical data and a p-value of ≤ 0.05 was considered significant.

RESULTS

The total number of recording errors committed at the three sites was 6858, which constituted around one fifth of the maximum possible number of errors (33,000). The overall errors were more common in private clinic recordings than Azadi Hospital and Zanest Center. Minor errors greatly exceeded the major ones where minor non-technical errors occurred slightly more often than technical ones as is demonstrated in table 1.

Most major errors were seen at Azadi Hospital, and of those more than three quarters were calibration errors. Errors in lead number were few and lead connection errors were rare while no ECG record with speed adjustment error was found, as can be seen in table 2.

The total number of minor technical recording errors committed at the three sites was 2615, which constituted 7.9 % of the maximum possible number of errors. Slightly more errors were seen in Azadi Hospital than at private clinics and Zanest Center. The most commonly encountered errors were interference control issues and stylus artifacts while baseline stability problems did not occur very often. No damping errors were detected at any site, as is shown in table 3.

The total number of minor non-technical recording errors committed at the three sites was 3259, which constituted around one tenth of the maximum possible number of errors. The errors were detected about twice as often at private clinics as compared to Azadi Hospital and Zanest Center. By far the most common type of error was missing or incomplete identity information, followed by long strip errors. Errors in the number of beats per lead and lead label were rare, as can be seen in table 4.

Table 5 demonstrates that while about one third of the total number of records contained major errors, about three quarters contained minor technical errors and almost all records contained minor non-technical errors. The highest percentage of faulty records containing major and minor technical errors were

those recorded at Azadi Hospital. Faulty records with minor non-technical errors were very common at all clinical sites where identity information error accounted for almost all of that type of error in the governmental sector. In private clinics almost all records contained long strip as well as identity information errors.

Table 1. Total number of recording errors by source and type

Source	No. of Potential Errors*	Major Error No. (%)	Minor T. Error No. (%)	Minor N-T. Error No. (%)	Total No. (%)
Azadi (N=2150)	25800	859 (3.3)	2078 (8.1)	2341 (9.1)	5278 (20.4)
Zanest (N= 300)	3600	38 (1.1)	261 (7.3)	299 (8.3)	598 (16.6)
Private (N= 300)	3600	87 (2.4)	276 (7.7)	619 (17.2)	982 (27.3)
Total (N=2750)	33000	984 (3.0)	2615 (7.9)	3259 (9.9)	6858 (20.8)

χ^2 = 155; p < 0.001; N = Number of ECG records; T. = Technical; N-T. Non-Technical
** An ECG record might have up to 12 recording errors, 4 of each type.*

Table 2. Major errors by source and type

Source	No. of Potential Errors*	Lead Connection No. (%)	Calibration Adjustment No. (%)	Speed Adjustment No.(%)	Leads Number No. (%)	Total No. (%)
Azadi (N=2150)	25800	6 (0.0)	764 (3.0)	0 (0)	89 (0.3)	859 (3.3)
Zanest (N= 300)	3600	22 (0.6)	1 (0.0)	0 (0)	15 (0.4)	38 (1.1)
Private (N= 300)	3600	7 (0.2)	60 (1.7)	0 (0)	20 (0.6)	87 (2.4)
Total (N=2750)	33000	35 (0.1)	825 (2.5)	0 (0)	124 (0.4)	984 (3.0)

N = Number of ECG records
** An ECG record might have up to 12 recording errors; 4 of them of the major type.*

Table 3. Minor technical errors by source and type

Source	No. of Potential Errors*	Stylus Artifact No. (%)	Damping Adjustment No.(%)	Interference Control No. (%)	Baseline Stability No. (%)	Total No. (%)
Azadi (N=2150)	25800	467 (1.8)	0 (0)	1492 (5.8)	119 (0.5)	2078 (8.1)
Zanest (N= 300)	3600	64 (1.8)	0 (0)	173 (4.8)	24 (0.7)	261 (7.3)
Private (N= 300)	3600	75 (2.1)	0 (0)	179 (5.0)	22 (0.6)	276 (7.7)
Total (N=2750)	33000	606 (1.8)	0 (0)	1844 (5.6)	165 (0.5)	2615 (7.9)

N = Number of ECG records
** An ECG record might have up to 12 recording errors; 4 of them of the minor technical type.*

Table 4. Minor non-technical errors by source and type

Source	No. of Potential Errors*	Number of Beats/Lead .No. (%)	Long Strip No. (%)	Lead Label .No. (%)	Identity Information .No. (%)	Total No. (%)
Azadi (N=2150)	25800	5(0.0)	237 (0.9)	0 (0)	2099 (0.3)	2341(9.1)
Zanest (N= 300)	3600	0(0)	0 (0)	4 (0.1)	295 (0.4)	299(8.3)
Private (N= 300)	3600	13(0.4)	298 (8.3)	8 (0.2)	300 (0.6)	619(17.2)
Total (N=2750)	33000	18(0.1)	535 (1.6)	12 (0.0)	2694 (0.4)	3259(9.9)

N = Number of ECG records
** An ECG record might have up to 12 recording errors; 4 of them of the minor non-technical type.*

Table 5. Faulty records* by source and type of error

Source	No. of Records	Records w/ Major Errors No. (%)	Records w/ Minor T. Errors No. (%)	Records w/ Minor N-T. Errors No. (%)	Total No. (%)
Azadi	2150	821 (38.2)	1650 (76.7)	2102 (97.8)	2102 (97.8)
Zanest	300	37 (12.3)	203 (67.7)	295 (98.3)	295 (98.3)
Private	300	83 (27.7)	212 (70.7)	300 (100)	300 (100)
Total	2750	941 (34.2)	2065 (75.1)	2697 (98.1)	2697 (98.1)

$\chi 2 = 48$; $p < 0.001$; w/ = with; T. = Technical; N-T. Non-Technical
** A record is considered faulty when it shows any of the 12 recording errors.*

DISCUSSION

ECG recordings are often corrupted by artifacts. These artifacts severely limit the utility of recorded ECGs and thus need to be removed for better clinical evaluation.[3]

The study sample included 2750 records, 300 from private clinics, 300 from Zanest Center, and 2150 from Azadi Teaching Hospital. Each ECG record may show up to 12 possible errors, 4 major, 4 minor technical, and 4 minor non-technical errors, resulting in a total number of 33,000 possible errors (2750 x 12).

The overall number of errors at the three sites was 6858, which constitutes about one fifth of the maximum possible total, thus reflecting a high error rate, which is unacceptable in such a critical field of clinical practice. Most major errors were seen at Azadi Hospital (3.3%) followed by private clinics (2.4%) and Zanest Center (1%). This may be due to a larger number of patients at Azadi

Hospital, which may cause the medical personnel to be overworked, thus allowing more major errors to happen.

Al Habeeb[11] studied the problem of recording errors in three teaching hospitals in Baghdad comparing them with those of private clinics of senior physicians in Baghdad. The data revealed that the highest percentage of major errors occurred at hospitals (Al Yarmouk Hospital 4.2% and Ibn Al Nafeece Hospital 2.9%) more than at private clinics (1.3%).

Calibration errors very much exceeded other major errors especially at Azadi hospital while the highest percentage of minor errors was registered at private clinics. Al Habeeb[11] reported a higher overall percentage of minor errors at both hospitals and private clinics (Baghdad Teaching Hospital 19.5%, Ibn Al Nafeece Hospital 29.6%, Al Yarmouk Hospital 24.2%, and private clinics 34.8%) but with a similar trend of a higher number of minor errors in the private clinics.

Minor technical errors were seen almost equally at the three sites. Their presence in any ECG record may cause confusion for those with limited experience like junior house officers. Minor non-technical errors were the most commonly encountered errors at all three sites. Their frequency in private clinics is double that seen at Azadi Hospital and Zanest Center, yet, they have only minimal effect on the quality of the ECG and do not materially hinder interpretation.

The higher error percentage at private clinics gives a false impression of inefficiency or incompetence, but this is not the case, since the proper application of these auxiliary steps is closely related to the available resources and time. As such considerations are of high importance in private work, the increased error percentage in these elementary non-technical steps does not reflect poor performance in the private sector.

Of the minor technical errors those with interference control were found to be the most common, which may be muscular in origin or due to electricity interference. Electromyogram artifacts often contaminate the ECG.[12] Baranchuk et al.[13] reported in their study that electromuscular interference (EMI) was incorrectly diagnosed in 18% of the cases. Most frequently EMI was confused with atrial fibrillation or flutter (52%), ventricular arrhythmias (22%), and pacemaker dysfunction (26%). Medical students and non-cardiology residents demonstrated significantly worse performance on EMI interpretation. Knight et al [14] found that interference artifacts that look like wide ventricular complexes were not recognized as artifact by 94% of internists, 58% of cardiologists and 38% of electrophysiologists. Furthermore, 88% of the electrophysiologists, 53% of the cardiologists, and 31% of the internists who misdiagnosed the rhythm as ventricular tachycardia recommended an invasive procedure for further evaluation or therapy.

Faulty records with major and minor technical errors were mostly seen at Azadi Hospital, which discloses a clear performance defect. The relatively high number of stylus artifacts raises concerns

about poor maintenance and/or poor capabilities of the staff for proper adjustment of the electrocardiographs.

Baseline instability was mostly encountered at Zanest Center and private clinics. Its occurrence renders interpretation of S-T segment changes impossible and seriously interferes with T wave interpretation.[15] Movement of the patient, loose contacts in any place in the circuit or cutaneous currents and swinging of loose wires conducting electricity, all can cause baseline drift producing a false impression of S-T segment depression or elevation.[16]

Records with major and minor technical errors are more common in the governmental sector than the private sector. This is an expected finding and correlates well with the proficiency of internists and cardiologists practicing at the private sector.

Overall, ECG recording errors at the three health care sites constituted one fifth of all possible errors, comprising 3% major errors, 8% minor technical errors, and 10% minor non-technical errors where major errors were mostly seen at Azadi Teaching Hospital and least at private clinics. About one third of the total ECG records contained major errors while three quarters contained minor technical errors and nearly all records contained minor non-technical errors.

Immediate and decisive measures should be put into action to prevent potentially serious sequelae. Mandatory continued medical education should focus on errors of great impact on the clinical outcome, namely major errors. Furthermore, the final year medical school curriculum should allow for sufficient theory and practice on ECG interpretation, including its potential recording errors.

COMPETING INTETESTS

None of the authors have any competing interests in the study. The authors themselves did not receive any funds for conducting the study.

AUTHORS' CONTRIBUTION

Dr. Suad N. Jaafar: developed the design of the study, collected and interpreted the data, and translated abstract into Kurdish and Arabic. Dr. Lars A. Peschke: assisted revising initial thesis and writing of this journal article manuscript. Dr. Qayser S. Habeeb provided the original idea of the study, guidance and input along the study.

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پوخته

دەستنیشاکرنا خەلەتییێن تومارکرنا هێلکاریا دلی ل دەمێ پێرابونێن نەخوشیێ ل باژێرێ دهوکێ / عیراق

پێشەکی و ئارمانج: هێلکاریا دلی یێ 12 کەنال یێ مفادارە هەتا رادەکێ مەزن وڤەکولینەکا کارتێکەرە، بابەتێ هێلکاریا دلی رامانا وێ تومارکرن و شروڤەکرنا کارەبایی یە بو دلی و تومارکرنا کارێ دروستە یێ ساخلەتێن جیهانی وپێدڤی بو مفا وەرگرتن ژ هێلکاریا دلی وەك ئامیرەکێ دەست نیشانکرنێ. هەکە خەلەتییێن دیارکری ب بەرچاڤ نەهاتنە وەرگرتن. دێ ئاریشێن زیاندار ژ خەلەتییێن دەست نیشانکرنێ پەیدابن. ئەڤ بابەتە هەتا نوکە ل هەرێمێ نەهاتیە خواندن .ئارمانجا ڤەکولینێ چێدبیت ڤەکولینەك بهێتە کرن ل دور هێلکاریا دلی وتومارکرنا خەلەتییێن پەیدا دبن ل دەمێ پێرابوونێن نەخوشیێ ل باژێرێ دهوکێ.

ریکێن ڤەکولینێ: کۆمکرنا رووتکرنا دێ ژ 2011/1/20 هەتا 2011/5/31 ڤەکێشیت. کەرەستە و ئامیرە: دیزاینا پانیێ هاتە وەرگرتن بو هەلسەنگاندنا سەرجەمێ تومارکرنێن هێلکاریا دلی 20750 کو ل سێ کەرتا هاتە تومارکرن بو پێرابوونێن نەخوشیێ ل نەخوشخانا ئازادی ومەلبەندێ زانست یێ نوژداریا خێزانێ وکلینیکێن تایبەت ل باژێرێ دهوکێ. هەمی تومارکرن هاتنە هەلسەنگاندن بو گەلەك جوڕێن خەلەتیا ئەوێن بخوڤە گرتین لدویف پیڤەرێن خەملاندنێ ئەوێن د بەرێدا هاتینە دەست نیشانکرن.

ئەنجام: سەرجەمێ هژمارێ بو تومارکرنا خەلەتیا 6858 کو نێزیکی 5/1 ئانکو (20.78 ٪) بو بلندترین ژمارە ژ خەلەتیا (33000) هاتە پێك ئینان. هەمی خەلەتییێن سەرەکی ل نەخوشخانا ئازادی (3.32 ٪) هاتنە دیتن. پشتی هینگێ دەورێ کلینیکێن تایبەت ومەلبەندێ زانست (2.41 ٪) و (1.05 ٪) لدویف ئێك هات وخەلەتییێن تەکنیکی یێن نەسەرەکی د نێزیکی ئێك بوون د سێ جهاندا. بەلێ خەلەتییێن نەسەرەکی یێن نەتەکنیکی ل کلینیکێن تایبەت بریژا (17.19) د دووجارکی بوون بو ژمارا هەی ل نەخوشخانا ئازادی ومەلبەندی زانست ونێزیکی 3.1 (34.21 ٪) ژ هژمارا دروست بو تومارکرنا خەلەتییێن سەرەکی تێدا بوون. هەر وەسا هەمی تومارکرن ب ریژا (98.0 ٪) خەلەتییێن نەسەرەکی ونەتەکنیکی بخوڤە گرت.

دەرئەنجام: خەلەتییێن تومارکرنێ یێن هێلکاریا دلی ئاریشەك ل سێ کەرتێن پێرابوونا نەخوشیێ ل دهوکێ پەیداکر پترپا وان ل دەزگەهێن حکومەتێ بوون و دڤێت پیڤانێن بلەز وبهێز بو بجهئینانێ بهێنە دانان بو پاراستنێ ژ کارتێکرنێن نەخوشیێ.

الخلاصة

تقصي الاخطاء التسجيلية في مخطط كهربائية القلب التي تحصل اثناء الممارسة السريرية في مدينة دهوك

خلفية واهداف البحث: ان مخطط كهربائية القلب ذي الوصلات الاثنى عشر جدير بالاهتمام لفائدته وتعدد استعمالاته. ان موضوع تخطيط القلب الكهربائي يختص بتسجيل وتحليل النشاط الكهربائي للقلب. ان طريقة التسجيل الصحيحة واعتماد معايير النوعية القياسية تمثل الاساس للاستغلال الامثل لعملية تخطيط القلب كوسيلة تشخيص. اذا لم تؤخذ بعض الاخطاء التسجيلية بنظر الاعتبار فان اخطاء تشخيصية يمكن ان تحدث مع ما يليها من تبعات غير محمودة العواقب. هدف الدراسة هو تقصي الاخطاء التسجيلية في مخطط كهربائية القلب التي تحصل اثناء الممارسة السريرية في مدينة دهوك.

طرق البحث: استغرقت عملية جمع العينات الفترة من 20 / كانون الثاني الى 31 / مايس / 2011. استخدم تصميم الدراسة المقطعية لتقصي ما مجموعه 2750 مخطط تم تسجيلهم في ثلاثة قواطع للممارسة السريرية هي: مستشفى ازادي ومركز زانست لطب الاسرة والعيادات الخاصة في دهوك. اخضعت كل المخططات للفحص بهدف تحري وجود اي نوع من الاخطاء التسجيلية وذلك وفقا لمعايير ضابطة اعدت مسبقا لهذا الغرض.

النتائج: بلغت كل انواع الاخطاء 6858 خطأ اي ما يعادل الخمس من اكبر عدد ممكن من الاخطاء. شكلت الاخطاء الكبرى في مستشفى ازادي (3.32%) تليها ا لعيادات الخاصة ثم على التوالي (2.41%) و (1.05%) مركز زانست. كانت الاخطاء الصغرى مقاربة لبعضها في القواطع الثلاثة. الاخطاء الصغرى غير التقنية لدى العيادات الخاصة كانت مقاربة لضعف نظيراتها في مستشفى ازادي ومركز زانست. كما ان ثلث العدد الكلي للمخططات كان حاويا على اخطاء كبرى بينما احتوى ثلاثة ارباعها على اخطاء صغرى تقنية وتقريبا كلها احتوت على اخطاء صغرى غير تقنية.

الاستنتاجات: شكلت اخطاء التسجيل مشكلة في مجالات الممارسة السريرية الثلاث في دهوك خصوصا في المؤسسات الحكومية. الوضع الحالي يستدعي الشروع الفوري والنشيط في اتخاذ ما يلزم لتجنب العواقب السريرية المتوقعة.

PAPILLARY CARCINOMA IN THYROGLOSSAL CYST: A CASE REPORT

SARDAR H. ARIF, MBChB, FIBMS*
BASHAR A. HASSAWI, MBChB, FIBMS**

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SUMMARY

Thirty two years female patient presented with small cyst in the neck for 15 years. The last 3 years it enlarge in the size, fine needle aspiration cytology showed atypical cells then the CT scan performed of the neck showed no evidence of foci in thyroid gland, No lymph nodes, Sistrunk's operation was done that mean excision of thyroglossal cyst. The biopsy revealed papillary carcinoma.

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Key words: Thyroid, Thyroglossal cyst, Papillary carcinoma

Malignant thyroid tumors are still the commonest malignant tumors of the endocrine glands.[1,2] It accounts about 90% of the whole types of malignant endocrine tumors,[3] however it constitute about 1% of the malignant tumors in the body.[4]

Thyroid carcinoma can be classified into many histological subtypes[5]: papillary, follicular, medullary, anaplastic

Papillary carcinoma is the commonest type in this group; it accounts 70-75% of thyroid carcinoma and has great relation with exposure to irradiation in pathogenesis of this tumor, especially those who exposed to radiotherapy of the neck region. This is because of high sensitivity of thyroid gland to the carcinogenic effects of radiation during childhood. [6,7]

Papillary carcinoma known to extend to lymph nodes in approximately 40% of cases, the most common nodes involved are internal jugular and recurrent laryngeal chain on the side of the lesion[8] and rarely may extend to submandibular group.[9]

A thyroglossal duct cyst is the most common congenital anomaly of the thyroid gland and midline masses in childhood (70% abnormality in childhood, 7% in adult). Carcinomas arising from a thyroglossal duct cyst are rare (only 1% of thyroglossal duct cyst cases) and characterized by relatively non-aggressive behaviour with rare lymphatic spread. They are also diagnosed mostly during the third and fourth decades of life. About 85% to 92% of all thyroglossal duct cyst carcinomas are papillary carcinomas.[10]

CASE PRESENTATION

Thirty two years female patient presented with small cyst in the neck for 15 years. The last 3 years it enlarge in the size. On examination it was hard in consistency mobile in anterior triangle of the neck at subhyoid region in midline. Ultrasound of the neck revealed thyroid cyst. Thyroid

* *Lecturer, Department of surgery, Faculty of medical sciences, Duhok university.*
** *Assistant professor, Department of pathology, Faculty of medical sciences, Duhok university*

Correspondence author: Bashar A. Hassawi. Email: b_hassawi@yahoo.com. Tel: 00964 7706545899/ 00964 7507644861

function test were normal, fine needle aspiration cytology showed atypical cells then the computered tomography showed no evidence of neoplastic foci in thyroid gland, no lymph nodes , Sistrunk's operation was done that mean excision of thyroglossal cyst and it's tract including middle third of hyoid bone (Figure 1 and 2).

The histopathological examination revealed wall of a cyst that lined by flattened to cuboidal cell surrounding the typical appearances of thyroid papillary carcinoma (Figure 3). It comprises complex, branching and randomly oriented papillae with a central fibrovascular core and a single or stratified lining of cuboidal and columnar cells. In addition, nuclear features showed optically clear (ground glass) nuclei and nuclear grooves indicating a malignant papillary carcinoma (Figure 4).

Thyroidectomy and bilateral neck dissection performed. The histopathology revealed normal thyroid gland and only one lymph node showed metastasis.

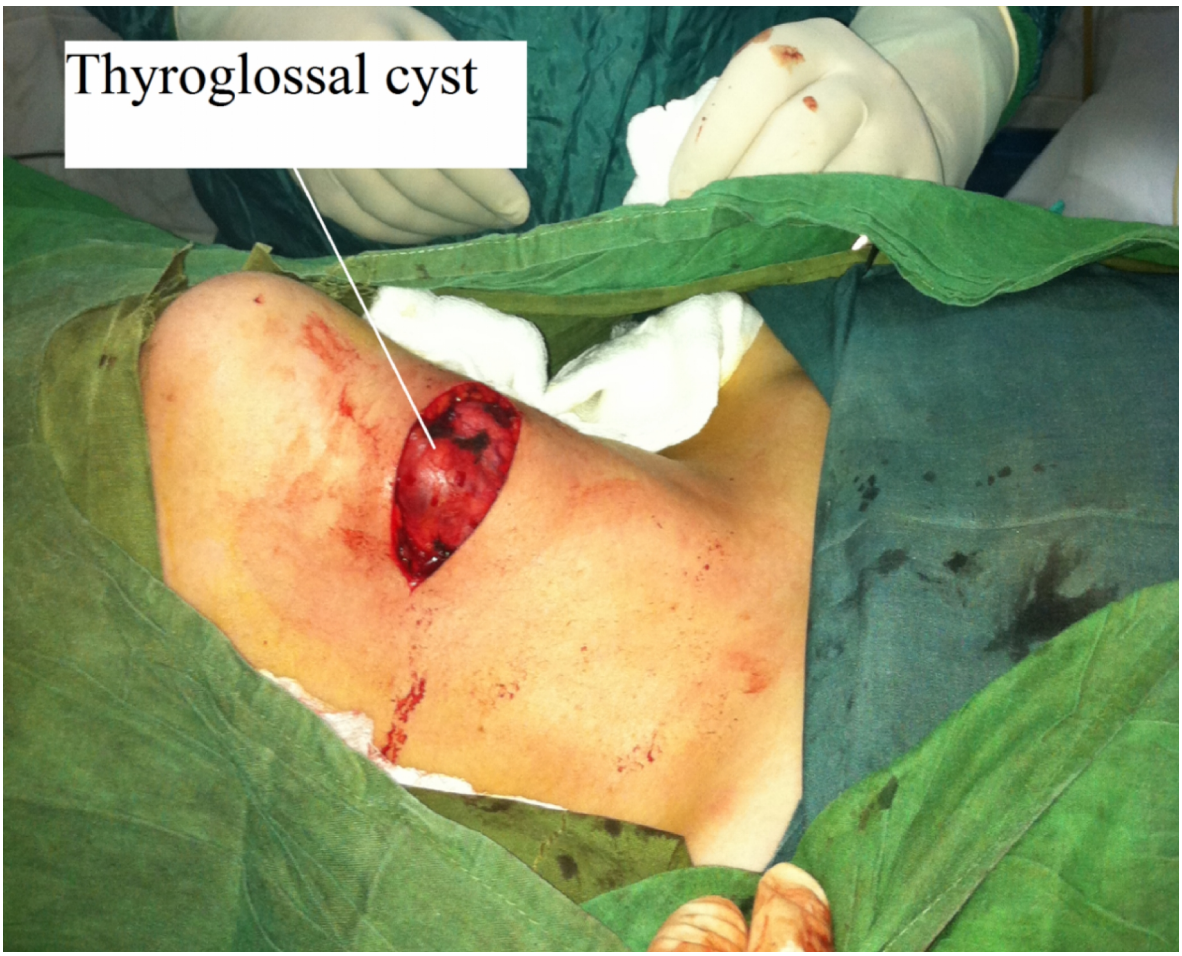

Figure 1. thyroglossal cyst located in subhyoid region

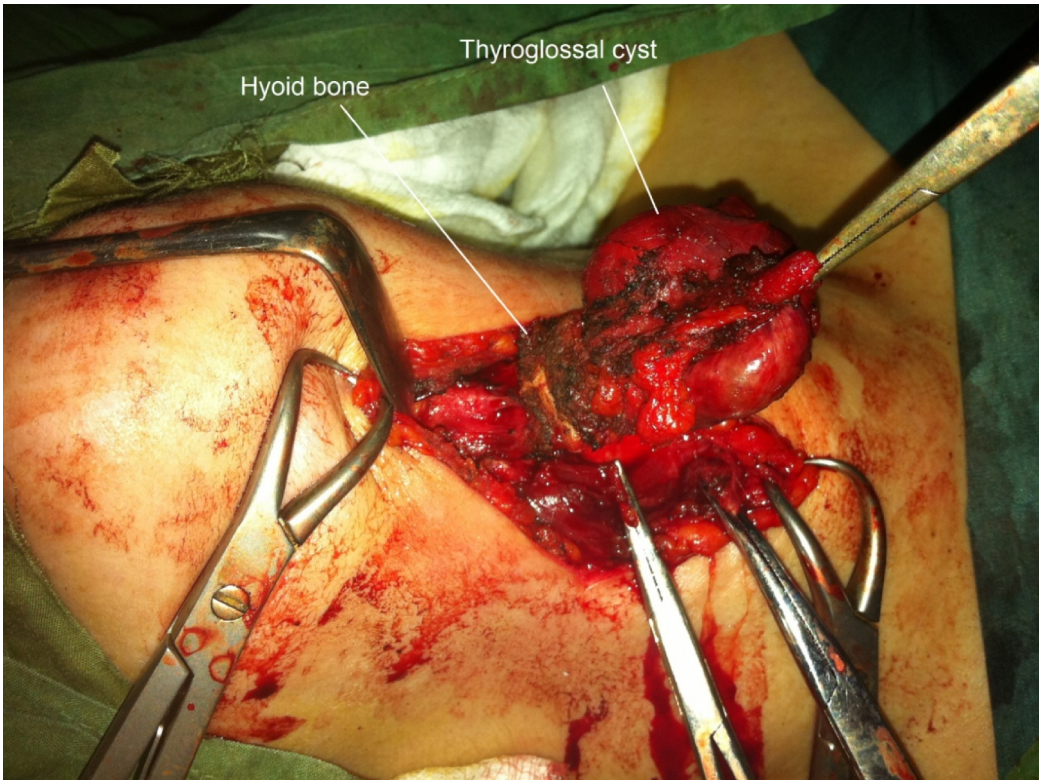

Figure 2 .Excision of thyroglossal cyst and middle third of hyoid bone

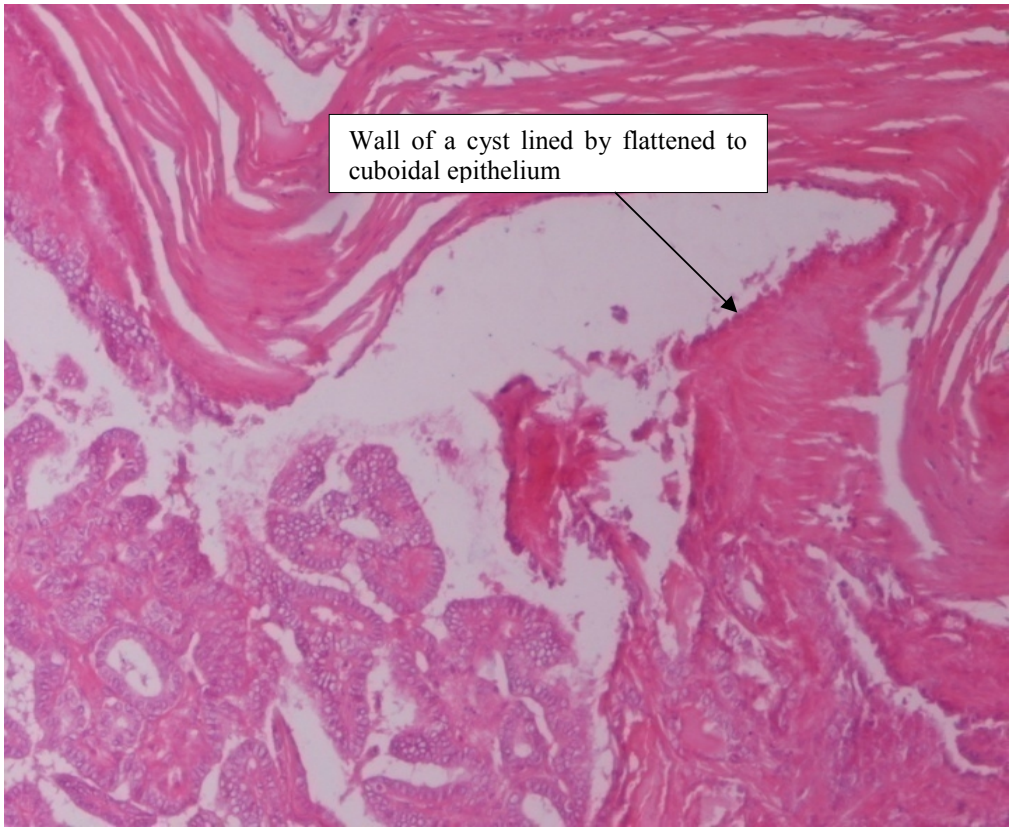

Figure 3. Wall of a cyst shows lining epithelium (×100)

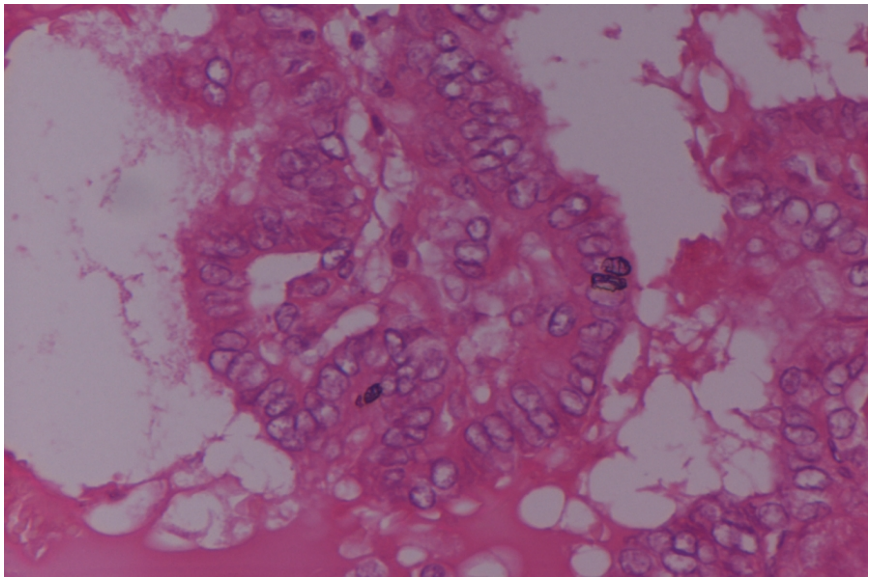

Figure 4. Papillary projections with lining malignant epithelial cells (×400)

DISCUSSION

More than 200 cases of thyroglossal duct carcinomas have been reported in which papillary carcinoma accounts for 80% of cases, with the rest being Squamous cell carcinoma arising from metaplastic columnar cells that line the duct.[11-13]

A cervical nodule is a common clinical finding that includes wide range of differential diagnosis. Although most masses are due to benign processes; however, malignant diseases must be included. Therefore, it is important to develop a systematic approach for the diagnosis and management of neck masses.

Benign thyroglossal duct cysts usually present as asymptomatic, soft, firm or hard midline mass at the anterior aspect of the neck, generally movable and not tender. Malignant thyroglossal duct cysts present in the same manner. Carcinoma should be suspected in any thyroglossal duct cyst that is hard, fixed and irregular or which has undergone recent change. A history of irradiation of the head and neck or mediastinum during childhood or adolescence should also arouse suspicion of carcinoma.[6]

Malignant tumors developing from the thyroglossal duct have two origins: thyrogenic carcinoma arising from thyroembryonic remnants in the duct or a cyst, and Squamous cell carcinoma arising from metaplastic columnar cells that line the duct.[6]

Excluding medullary carcinoma, which arises from parafollicular cell embryologically unrelated to the thyroid, all forms of primary thyroid carcinoma can arise in the thyroglossal duct.[6]

There is still controversy regarding the need to remove the thyroid gland in the case of a papillary carcinoma of the thyroglossal cyst. However thyroidectomy is recommended in cases where (a) the thyroid gland is found to be nodular, with a cold nodule in a thyroid iodine uptake scan; (b) enlarged lymph nodes are present, or (c) a history of neck irradiation exists.[14]

This case concludes that papillary thyroid carcinoma and other malignancy

within a thyroglossal duct cyst is rare but should be included in the differential diagnosis of a neck mass especially of long standing cyst, especially if there is increasing in size. This condition is may suspected by FNAC. Therapy includes surgery and further measures like radioactive iodine and follow up according to differentiation and extend of tumor.

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پوخته

بەنجەشێرا کیسەکێ دەرهافیژی

حالەتەکێ بالکەهی بو کچەکا 32 سالی کو کیسەکئ دەرهافیزی بەری 15 سالان بو بەیدابو و د 3سالین دوماهیئ دا قەوارئ وی مەزن ببو و ب ریکا نشتەرکەریئ هاتە راکرن و دیاربو کو وەرەما بەنجەشێرێ یە.

الخلاصة

السرطان الحليمي للكيس الدرقي اللساني

حالة سريرية لانثى عمرها 32 عاما لديها كيس درقي منذ 15 عاما وزاد في الحجم منذ ثلاثة سنوات وتم رفعه جراحيا وتبين انه يحتوي على ورم سرطاني درقي حليمي.