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# IRAQI JOURNAL OF MEDICAL SCIENCES

# Iraqi Journal of Medical Sciences



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# Iraqi Journal of Medical Sciences

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**Iraqi Journal of Medical Sciences** publishes original articles, case reports, and letters to the editor, editorials, investigative medicine, and review articles. They include forensic medicine, history of medicine, medical ethics, and religious aspects of medicine, and other selected topics.

# IRAQI JMS FORMAT
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**TYPES OF CONTRIBUTIONS:** Original articles, review articles, case studies, editorials, medical education, history of medicine, ethics, practical points, medical quiz, conferences, meetings and letters to the Editor.

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3. Chapter in book: Phillips SJ, and Whisnant JP. Hypertension and strock. In: Laragh JH, and Brenner BM. editors. Hypertension: Pathophysiology, diagnosis, and management. 2nd ed. NewYork: Raven Press; 1995. p. 465-78.

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# Editorial:

## Blood Type Diet: Scientific evaluation

**Enas Talib Abdul-Karim** *PhD.*

Developed by naturopathic physician Dr. Peter D'Adamo, the Blood Type Diet is based on the theory that protein-like compounds in food called lectins react with different blood types to cause a wide variety of health complaints.

The Blood Type Diet is the culmination of nearly four decades of work conducted by Peter D'Adamo, and his father, James D'Adamo, as a naturopathic physician practicing in the 1960s, James wondered why some of his patients did well on the therapeutic diets he prescribed, including strict vegetarian and low-fat diets, while others did not improve or even became worse. James suspected that the difference in the way his patients reacted to the same foods might be rooted in some aspect of the blood. With this hypothesis in mind, he began to record the blood type of his patients and observe their individualized reactions to different diets. Over the years, distinct patterns began to emerge. He noticed that patients with Type A blood did well on a vegetarian diet, while patients with Type O did well on a high-protein, meat-based diet. In 1980, James D'Adamo published his clinical observations in a book titled One Man' Food. That same years, Peter D'Adamo began to research the scientific basis for his father' theories. In 1996, nearly twenty years later, Dr. Peter D'Adamo's findings were published in his book, Eat Right for Your Type [(1)].

Dr. D'Adamo's ideas on the relationship between diet and health are rooted in a simple concept: the blood type-O, A, B or AB-determines the body's ability to absorb nutrients, fight off diseases, and handle stress. Dr. Peter D'Adamo's diet recommendations were born out of evolutionary history, and specifically, the observation that the different blood types emerged as the environmental conditions and eating styles of our ancestors changed. Between 50,000 BC and 25,000 BC, all humans shared the same blood type-Type O. these early humans were skilled hunters, and thrived on a meat-based diet. The type A blood type emerged between 25,000 BC and 15,000 BC, a necessary adaptation to a more agrarian lifestyle. Climactic changes in the Western Himalaya Mountains led to the appearance of Type B, and the blending of Type A and Type B in modern civilization resulted in the appearance of Type AB blood type. Because the emergence of new blood types made it possible for our ancestors to survive the changing environmental conditions, Boyd divided the world population into 13 geographically distinct races with slightly different frequency distributions of blood group genes. Dr. D'Adamo believes that blood type, diet, and health are intricately related; he grouped those thirteen races together by ABO blood group, each type within this group having unique dietary recommendations.

The physiological reason why people should eat according to their blood type relates to lectins, which are protein-like substances found in many

commonly eaten foods. Lectins, also known as phytohemagglutins, were first identified in 1888, at which time it was discovered that lectins interact with sugar-containing molecules on the surface of cells. This discovery allowed certain lectins to be used in blood typing, since blood type is determined by the presence (or absence) of specific sugar-protein residues on the surface of red blood cells.

Although most of the lectins found in food are destroyed by cooking, digestive enzymes, or are inactivated within the gut, at least 5% of the lectins we take in through our diet are absorbed into the bloodstream, and some of these are incompatible with our blood type. Many food lectins look very similar to the antigen that determines one of the four blood types or else bind directly to blood type antigens. In either case, this resemblance can lead to agglutination. According to Dr. D'Adamo "simply put, when you eat a food containing lectins that are incompatible with your blood type antigen, the lectins target an organ or bodily system and begin to agglutinate blood cells in the area". It is believed that if a person want to prevent health problems, it is important to eat foods that are compatible with his blood type based upon their lectin content.

The research done by Nachbar and Oppenheim done in 1980 [(2)] support Dr. D'Adamo support his work, they studied the edible portions of 88 commonly eaten foods including fresh fruits, roasted nuts, and processed cereals, and found that 29 of the 88 foods tested possessed significant lectin-like activity. The researchers also determined that dry heat does not completely destroy the lectin activity in wheat germ, peanuts, and the dry cereals. in addition, wheat germ agglutinin, tomato lectin, and navy bean lect6in have been found to resist breakdown by digestive juices. As a result, it can be concluded that at least some of the lectins found in food make it into the blood stream.

In the Blood Type Diet, foods are divided into sixteen categories: meats and poultry; seafood; dairy and eggs; oils and fats; nuts and seeds; beans and legumes; cereals; breads and muffins; grains and pasta; vegetables; fruit; juices and fluids; spices; condiments; herbal teas; and miscellaneous beverages. For each blood type, lists of foods described as "highly beneficial", "neutral", or "avoid". in support of his dietary recommendation, Dr. D'Adamo points to research that provides evidence for the presence of lectins in food, the agglutination effects of lectins, and the connection between blood type and the development of disease.

Type O was the blood type of the earliest humans, who were skilled hunters and subsisted on a diet of wild game and edible plants growing wild in the forests. As a result, the diet for individuals with Type O blood emphasizes the importance of animal flesh and vegetables.

Individuals with Type A blood thrive on a plant-based diet and should consume large amounts of raw or steamed vegetables, lentils, soy beans, pinto beans, black beans and whole grains. Berries and plums are also beneficial. Occasional consumption of poultry and fermented dairy products is also well tolerated.

The diet for individuals with Type B blood is more varied than the other blood type diets. They can eat seafood, beef, lamb, and dairy products. Oats and millet, green vegetables, and all fruits are beneficial.

***Scientific evaluation***

**1.** Questions of lectin actions

Dr. D'Adamo claims there are many ABO specific lectins in foods [2]. This claim is unsubstantiated by established biochemical research, which has not found differences in how the lectins react with a given human ABO type. In fact6, research shows that lectins which are specific for a particular ABO type are not found in foods (except for one or two rare exceptions, e.g. lima bean), and that lectins with ABO specificity are more found in non-food plants or animals [3, 4], while, the Nachbar study has been cited in support of Dr. D'Adamo's theories.

**2.** Lack of clinical trials

Another criticism is that there are no clinical trials of the Blood Type Diet, but the self-reported internet survey with 6627 respondents conducted by D'Adamo's website reported that individuals following the Blood Type Diet for a period of one month or more, in 71-78% of cases, had significant improvement in a variety of health conditions. The most common improvement was with weight [5]. These results, however, are "self-reported".

**3.** Blood type evolution issues

In a Brazilian medical research journal, Luiz C. de Mattos and Haroldo W. Moreira point out that D'Adamo's assertion that the O blood type was the first human blood type requires that the O gene evolved before the A and B genes in the ABO locus[6]. However, phylogenetic networks of human and non-human ABO alleles show that the A gene was the first to evolve [7].The authors argue that, in the evolutionary sense, it would be extraordinary for normal gene (those of type A and B) to have evolved from abnormal genes (for type O)

Another study from 2004 concluded that: "Assuming constancy of evolutionary rate, diversification of the representative alleles of the three human ABO lineages (A101, B101, and O02) was estimated at 4.5 to 6 million years ago"[8]. This finding directly contradicts D'Adamo's assertion of blood type evolution. However, D'Adamo has been quite clear in the past that these conclusions were drawn from studies of the epidemiologic effects of migration patterns and infectious disease susceptibility in relation to blood groups distribution and the migration patterns, not natural selection via mutation in any Mendelian sense[9].

I wish for everyone optimal health, happiness, and longevity. We owe it to ourselves, to our children, and to all who come after them, to see how optimal function and life span can be achieved on diets that are truly sustainable. It is, after all, the food of all our futures.

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# Genotyping of HLA-class-I by PCR-SSP of Iraqi Breast Cancer Patients

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## *Abstract*

**Background:** The aetiology of breast cancer is multifactorial, in which genetic predisposition; environmental factors, hormones and even the infectious agents are thought to interact in the manifestation of disease. In this regard, alleles of HLA are important immunogenetic risk factors, but their associations show different frequencies in different populations.

**Objectives:** This study was established to shed light on the possible association of HLA class I alleles with BC in Iraqi female patients.

**Subjects and Methods:** The study included 60 subjects: 30 breast cancer patients, 12 patients with benign breast lesions as first control and 18 apparently healthy subjects as second control. Polymerase chain reaction-specific sequence primers (PCR- SSP) assay was conducted to assess HLA- typing.

**Results:** Out of 95 HLA class I alleles (A= 24; B= 48; C= 23), one allele (HLA- A*03010101-07, 09-11N, 13-16 allele) showed a significant variation between breast cancer patients and control groups (healthy and disease controls) (50% vs. 16.6%, OR=5, EF= 0.40, P= 0.041), (50% vs. 8.3%, OR=11, EF=O.45, P=0.024) respectively.

**Conclusions:** The results demonstrated that HLA- A*03010101-07, 09-11N, 13-16 allele may played a role in the etiology of the disease.

**Keywords:** Breast cancer, HLA, PCR.

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## *Introduction*

With more than 1 million new cases in the world each year, breast cancer (BC) is the commonest malignancy and the leading cause of cancer death in women [(1)]. In Iraq, the BC is the commonest cancer in females; furthermore, currently there is a general trend towards an increase the incidence of disease in females of younger ages [(2)].

Human based studies have suggested that the host genetics predisposition is important in disease pathogenesis and protection [(3)], and considering the importance of immune surveillance during tumorigenesis [(4, 5)], some individuals who inherit specific alleles or haplotypes of the highly polymorphic human leukocyte antigen (HLA) system may be exposed or may resist to specific types of cancers [(6-9)].

Breast cancer occurrence differs among women of different racial and/or ethnic groups, and accordingly, several HLA associations have linked HLA system with susceptibility or protection in the disease. However, the studies have been consistent with respect to the influence of these alleles in immune clearance of tumor cells in a way that could affect BC development. Therefore, HLA genotypes have been suggested to be a biologically based risk factor for BC [(10, 11)]. In the present study, the polymorphism of HLA-A, - B and -Cw alleles was analyzed in a sample of Iraqi females with BC using the PCR – SSP method.

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## ***Subjects and Methods***

### *Subjects:*

Thirty breast cancer female patients (invasive ductal carcinoma, invasive lobular carcinoma and in situ ductal carcinoma), with an age range of 28 - 73 years, were eligible for this study. The patients were admitted for surgery at Al-Kadhimia Teaching Hospital and Nursing Home Hospital (Medical City) in Baghdad, for the period March 2006 - March 2007. Pathological data (histologic tumor type grade, tumor stage and lymph node status) were obtained from the medical records of patients and validated by an experienced histopathologist.

Two control groups were included: 12females with benign breast lesions (BBL) (6 cases with fibrocystic disease and 6 with fibroadenoma) and 18 apparently healthy females. The latter subjects had no history or clinical evidence of any breast lesions and matched by age and ethnic backgrounds to BC patients.

### *Methods:*

#### *Blood collection*

Two milliliters of venous blood with EDTA as anticoagulant were collected from each subject.

#### *DNA extraction*

Extraction of DNA from peripheral blood was done according to the modified method of Miller [12], using the EXTRA-GENE-I kit (BAG-Germany), which is the most suitable method for isolation since pure DNA can be obtained from whole blood in a short time without the use of toxic chemicals or solvents.

#### *PCR amplification*

HLA-genotyping was performed by PCR-SSP according to a method presented by Olerup and Zetterquist [13,14], using low resolution typing kits (HISTO TYPE / DNA-SSP Kits-BAG- Germany). Appropriate amounts of DNA and Taq polymerase (Recombinant Taq polymerase from QIAGEN-company) were added to pre-aliquoted primers and PCR conditions were set according to the manufacturer instructions.

#### *Detection of PCR products*

PCR products were loaded in 2 % agarose gel containing 0.5 mg/ml ethidium bromide, electrophoresed for 25 min at 12 V/cm, and examined under ultraviolet light. The individual alleles were assigned for the specific pattern of appropriately sized bands.

### ***Statistical analysis***

The results were presented in terms of percentage frequencies, and alleles showing variations between patients and controls were further presented in terms of odds ratio (OR), etiological fraction (EF) and preventive fraction (PF). The significance of these differences were assessed by fisher's exact probability (P), which was corrected for the number of alleles at each locus [15,16].

## ***Results***

In this study the mean age of BC patients was 48.1 years with a range of 28 - 73 years, while the mean age of BBL was 35.33 years with a range of 21 - 50 years.

In the PCR-SSP method, 24 HLA-A, 48 HLA-B and 23 HLA-Cw specific primer mixes were employed as well as, a negative control and ladder mixes. A successful amplification resulted in the generation of a defined length band as a positive internal control in all lanes except the negative control lane, and when there was no amplification, there was no band. In addition, a positive specific amplification resulted in the generation of a specific band in addition to an internal control band (Figures 1-2).

The observed percentage frequencies of HLA-A,-B and –Cw alleles in the investigated groups are given in tables 1, 2 and 3, respectively, while allele showing a significant

variation is presented in table 4. Out of 95 HLA-class I alleles, one allele (A*03010101-07, 09-11N, 13-16) showed a significant variation between patients and controls. In breast cancer patients, the A*03010101-07,09-11N,13-16 allele accounted to 50% of patients, while its frequency in benign breast lesion patients and healthy controls was 8.3%, 16.6% respectively, such difference was significant (P= 0.024, P=0.041), and associated with OR and EF values of (11,5) and (0.45, O.40) respectively. However, the corrected probability of these differences failed to attend a significant level.

***Discussion***

The role of genetic factors in the etiology of BC was documented decades ago. As a result, the investigative efforts have focused on the genetic markers of susceptibility to this disease. In particular, HLA system plays a pivotal role in cellular immunity and may be an important genetically determined host trait [17, 18]. This study is the first attempt on the association between HLA class I alleles and BC in Iraqi patients, in which the alleles were characterized by PCR-SSP.

In the present work, there was a significant positive association between HLA- A*03010101-07, 09-11N, 13-16 allele and BC as compared with healthy control. The OR for this allele was 5 and this means that the individuals with A*03010101-07, 09-11N, 13-16 allele have 5 times a greater chance of acquiring BC than those of the same population who lack it. Also, in the present study the comparison of BC patients with a second control (patient control) revealed a significant increased frequency of A*03010101-07, 09-11N, 13-16 allele in BC patients. Therefore, this association of BC with A*03010101-07, 09-11N, 13-16 allele may be considered important in the etiology of BC because it was based in the comparison with two different control groups and unlikely to be attributed to chance factor.

In the context of anti-tumor immunity, HLA- A*03010101-07,09-11N,13-16 allele may differ in its ability to present p53 or other relevant tumor associated antigen derived peptides, thereby contributing to the modulation of risk to develop BC. A similar mechanism has been suggested to operate in case of progression of cervical cancer in HLA-B7 positive patients harboring an HPV variant with uniquely altered peptide sequence of the E6 oncoprotein. The association of HLA- A*03010101-07,09-11N,13-16 allele with breast cancer may also be due to other risk modulating loci that are in linkage disequilibrium with this allele [19].

This result is at variance with some other studies, in serological studies, Bouillenne and Deneufbourg, in (1979), identified HLA - A28 as high risk allele amongst nulliparous pre-menopausal breast cancer patients [20]. A study from India reported a higher frequency of HLA-A2, in breast cancer patients whereas HLA-A11, HLA-Aw19 were suggested to be protective alleles [21]. In addition, Glaser (2005) noticed that the risk increased for whites with A-23 and African-Americans with A-32 after adjustment for age and reproductive risk factors [22].

In the absence of similar findings elsewhere, we must suppose that this association is local, and since, HLA class I proteins are entrusted with the presentation of viral antigens, the A*03010101-07,09-11N,13-16 allele could in fact be related with a viral agent, which in turn is associated with breast cancer in this part of the world. In agreement with this scope, different research groups have recently reported

the involvement of a viral agent in human mammary carcinogenesis (murine mammary tumor virus) [23, 24]. In conclusion, these findings demonstrated that HLA-A*03010101-07, 09-11N, 13-16 allele might play a role in BC susceptibility, suggesting HLA-based different etiopathogenesis.

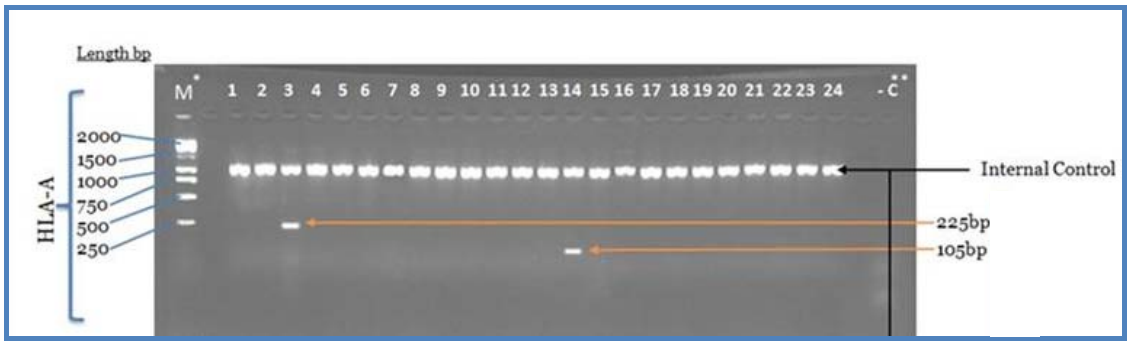


**Figure 1: Electrophoresis of HLA-A alleles amplification by PCR-SSP**. *Lane M represent 1 Kb DNA Ladder, **Lane-C represent negative control. HLA-A genotyping was obtained by using primers that detect the following alleles as they were present in the numbered wells, respectively:1= A1/ - / Null; 2= A2 /Low A2 / A203 / - / A210 / Null; 3= A2 / - ; 4= A3 / Null /- ; 5= A11 / - / Null ; 6= A23(9) / - / Null ; 7= A24(9) / Low A24/ Null / A2403 / A9 / - ; 8= A24(9) / - ; 9= A25(10) / - ; 10= A26(10),A10,Null ; 11= A26(10) ; 12= A29(19) / Null /- ; 13= A30(19) / - ; 14= A31(19) / - ; 15= - / A31(19) ; 16= A32(19) / - ; 17= A34(10) / - ; 18= A36 / - ; 19= A43 ; 20= A66(10) / - ; 21= A68(28) / A28 / Null / - ; 22= A69(28) ; 23= A74(19) / - ; 24= A80.


**Figure 2: Electrophoresis of HLA-A alleles amplification by PCR-SSP.** *Lane M represent 1 Kb DNA Ladder, **Lane-C represent negative control. HLA-A genotyping was obtained by using primers that detect the following alleles as they were present in the numbered wells, respectively:1= A1/ - / Null; 2= A2 /Low A2 / A203 / - / A210 / Null; 3= A2 / - ; 4= A3 / Null /- ; 5= A11 / - / Null ; 6= A23(9) / - / Null ; 7= A24(9) / Low A24/ Null / A2403 / A9 / - ; 8= A24(9) / - ; 9= A25(10) / - ; 10= A26(10),A10,Null ; 11= A26(10) ; 12= A29(19) / Null /- ; 13= A30(19) / - ; 14= A31(19) / - ; 15= - / A31(19) ; 16= A32(19) / - ; 17= A34(10) / - ; 18= A36 / - ; 19= A43 ; 20= A66(10) / - ; 21= A68(28) / A28 / Null / - ; 22= A69(28) ; 23= A74(19) / - ; 24= A80.

**Table 1: Observed percentage frequencies of HLA-A alleles in healthy controls, breast cancer patients and benign breast lesion.**

| Specificities | Healthy controls (n=18) | | Cases (breast Ca) (n=30) | | Benign breast lesion (n=12) | |
|---|---|---|---|---|---|---|
| HLA-A-Allele | N | % | N | % | N | % |
| A*010101-01010102N,0102-04N,06-11N,12,14,15N | 4 | 22.2 | 6 | 20 | 2 | 16.7 |
| A*02010101-22,24-33,36-45,47,49-54,57-61,63,64,66-69,71-77,79-86 | 4 | 22.2 | 6 | 20 | 3 | 25 |
| A*0246,48,70 | 3 | 16.7 | 3 | 10 | 3 | 25 |
| A*03010101-07,09-11N,13-16 | 3 | 16.7 | 15 | 50 | 1 | 8.3 |
| A*110101-*1116,20,21N-23 | 1 | 5.6 | 0 | 0 | 1 | 8.3 |
| A*2301,02,04-07N,08N,10,11N,12 | 1 | 5.6 | 3 | 10 | 1 | 8.3 |
| A*2408,21,29,42 | 2 | 11.1 | 0 | 0 | 1 | 8.3 |
| A*260101-0104,03,05,0701-08,10-12,14-18,21-25N,26 | 2 | 11.1 | 3 | 10 | 1 | 8.3 |
| A*300101-*3004,06-14L,15 | 3 | 16.7 | 6 | 20 | 2 | 16.7 |
| A*310102,02,05,06,08,09,11,12 | 1 | 5.6 | 3 | 10 | 1 | 8.3 |
| A*3201-08/B*1595 | 1 | 5.6 | 3 | 10 | 1 | 8.3 |
| A*3301,0301-07 | 1 | 5.6 | 3 | 10 | 1 | 8.3 |
| A*4301 | 0 | 0 | 0 | 0 | 1 | 8.3 |
| A*6601*6604 | 2 | 11.1 | 3 | 10 | 1 | 8.3 |
| A*680101-28 | 2 | 11.1 | 3 | 10 | 1 | 8.3 |
| Blank | 6 | 33.3 | 3 | 10 | 3 | 25 |

**Table 2: Observed percentage frequencies of HLA-B alleles in healthy controls, breast cancer patients and benign breast lesion.**

| Specificities | Healthy controls (n=18) | | Cases (breast Ca) (n=30) | | Benign breast lesion (n=12) | |
|---|---|---|---|---|---|---|
| HLA-B-Allele | N | % | N | % | N | % |
| B*0714 | 1 | 5.6 | 0 | 0 | 1 | 8.3 |
| B*0809 | 1 | 5.6 | 0 | 0 | 1 | 8.3 |
| B*15170101-1702 | 2 | 11.1 | 3 | 10 | 1 | 8.3 |
| B*1551,52 | 2 | 11.1 | 3 | 10 | 1 | 8.3 |
| B*1820 | 1 | 5.6 | 3 | 10 | 1 | 8.3 |
| B*35010104,03,05,07,08,1401-15,17,23, 24,29,30,32,33,36,38,40N-42,48,50,52, 53N-55-57 | 1 | 5.6 | 3 | 10 | 1 | 8.3 |
| B*350201,0202,0401,06,0901,0902,18 | 2 | 11.1 | 3 | 10 | 1 | 8.3 |
| B*3527,56 | 1 | 5.6 | 3 | 10 | 1 | 8.3 |
| B*3705 | 1 | 5.6 | 3 | 10 | 1 | 8.3 |

| | | | | | | |
|---|---|---|---|---|---|---|
| **B*4039,51** | **1** | **5.6** | **3** | **10** | **1** | **8.3** |
| **B*4101-07** | **1** | **5.6** | **3** | **10** | **1** | **8.3** |
| **B*440301,0302,04,07,13,26,28, 30,32,35,36,38,39** | **2** | **11.1** | **3** | **10** | **1** | **8.3** |
| **B*4416** | **0** | **0** | **0** | **0** | **1** | **8.3** |
| **B*4418** | **1** | **5.6** | **3** | **10** | **1** | **8.3** |
| **B*4442** | **1** | **5.6** | **3** | **10** | **1** | **8.3** |
| **B*4803** | **0** | **0** | **0** | **0** | **1** | **8.3** |
| **B*4901** | **1** | **5.6** | **0** | **0** | **0** | **0** |
| **B*5001,04** | **3** | **16.7** | **6** | **20** | **2** | **16.7** |
| **B*510102,0202,05** | **3** | **16.7** | **3** | **10** | **2** | **16.7** |
| **B*520101,0103,04,05,07** | **2** | **11.1** | **6** | **20** | **2** | **16.7** |
| **B*5208** | **1** | **5.6** | **3** | **10** | **1** | **8.3** |
| **B*180101-06,08,10-13,17N19** | **2** | **11.1** | **3** | **10** | **1** | **8.3** |
| **Blank** | **6** | **33.3** | **3** | **10** | **0** | **0** |

**Table 3: Observed percentage frequencies of HLA-Cw alleles in healthy controls, breast cancer patients and benign breast lesion.**

| **Specificities** | **Healthy controls (n=18)** | | **Cases(breast Ca) (n=30)** | | **Benign breast lesion( n=12)** | |
|---|---|---|---|---|---|---|
| **HLA-Cw-Allele** | **N** | **%** | **N** | **%** | **N** | **%** |
| **Cw*010201-03,06-09** | **1** | **5.6** | **0** | **0** | **1** | **8.3** |
| **Cw*020201-0205,04-08** | **1** | **5.6** | **0** | **0** | **1** | **8.3** |
| **Cw*030401-0403,05-10** | **1** | **5.6** | **0** | **0** | **1** | **8.3** |
| **Cw*0315** | **0** | **0** | **0** | **0** | **1** | **8.3** |
| **Cw*04010101-0102,03-09N,10-13** | **4** | **22.2** | **9** | **30** | **3** | **25** |
| **Cw*0501-07N** | **1** | **5.6** | **0** | **0** | **1** | **8.3** |
| **Cw*0602,03,07,09,10** | **3** | **16.7** | **6** | **20** | **2** | **16.7** |
| **Cw*070101,0102,05,06,08,14,16,18,20-22** | **3** | **16.7** | **6** | **20** | **2** | **16.7** |
| **Cw*120201-0203,08** | **1** | **5.6** | **3** | **10** | **1** | **8.3** |
| **Cw*120301,06,07,11** | **2** | **11.1** | **6** | **20** | **1** | **8.3** |
| **Cw*120401** | **2** | **11.1** | **6** | **20** | **2** | **16.7** |
| **Cw*120402,05** | **1** | **5.6** | **3** | **10** | **0** | **0** |
| **Cw*1210** | **1** | **5.6** | **3** | **10** | **1** | **8.3** |
| **Cw*1701-03** | **2** | **11.1** | **6** | **20** | **1** | **8.3** |
| **Cw07020101,020102,10,13,15,19** | **1** | **5.6** | **3** | **10** | **1** | **8.3** |
| **Blank** | **12** | **66.7** | **9** | **30** | **٥** | **50** |

**Table 4: HLA- A*03010101-07, 09-11N, 13-16 allele showing significant variations between breast cancer patients and controls.**

| HLA-A*03010101-07,09-11N,13-16 | Patients | | Controls | | Statistical evaluations | | |
|---|---|---|---|---|---|---|---|
| | N | % | N | % | Odds ratio | EF | P |
| Breast cancer patients vs. healthy controls | 15 | 50 | 3 | 16.6 | 5 | 0.40 | 0.041 |
| Breast cancer patients vs. benign breast lesion patients | 15 | 50 | 1 | 8.3 | 11 | 0.45 | 0.024 |

EF: Etiological fraction; P: Fisher's exact two-sided Probability.

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# The Expression of CD74 Molecule in *H.pylori* Infected Gastric Mucosal Tissue

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## *Abstract*

***Background:*** *Helicobacter pylori* cause gastric inflammation. Recent interest has been focused on the role of CD74 (the class II MHC-associated invariant chain expressed on the surface of gastric epithelial cells) as an adhesion molecules used by *H.pylori* that may contribute to the proinflammatory immune response seen during infection.

***Objective:*** The aim of this study was to detect the CD74 mucosal expression in *H.pylori* infected patients and compare it with uninfected patients.

***Patients and Methods:*** Sixty-four patients' age mean (34± 1.7) years (14-66 years) who underwent upper gastrointestinal endoscopy because of gastrointestinal complaints, were studied.

A number of both invasive and non-invasive diagnostic tests were used for the diagnosis of *H. pylori* infection, as well as immunohistochemical study of biopsy specimens to detect the CD74 mucosal expression.

***Results:*** After the diagnosis of *H.pylori* infection, patients were grouped as *H. pylori* positive,(*n*=47)and *H. pylori* negative (*n*=17). According to immunohistochemical study of biopsy specimens, the expression of CD74 was observed in infected subjects, and there was a significant difference in the CD74 expression ($p$= 0.005) between infected and uninfected patients.

***Conclusion:*** According to immunohistochemical study of biopsy specimens an overexpression of CD74 was observed in infected subjects

***Keywords:*** *Helicobacter pylori*; CD74; gastric epithelial cells; Immunohistochemistry (IHC)

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## *Introduction*

*Helicobacter pylori* infection provokes a vigorous humoral and cellular immune response in humans, but the organism is rarely eliminated from the gastric mucosa and infection persists lifelong in the absence of treatment [(1)]. *H.* pylori colonize the human stomach and is usually found either as an extracellular pathogen in the gastric mucosa or tightly attached to the cells of the gastric epithelium. Colonization by *Helicobacter pylori* always causes chronic gastritis and leads to the development of severe gastroduodenal diseases such as peptic ulcers, gastric adenocarcinoma, or Lymphoma of the mucosa associated lymphoid tissue (MALT) [(2, 3)].Although the mucosal colonization of *H. pylori* induces a mixed Th1/2-mediated mucosal cytokine milieu [(4,5)] and the generation of *H. pylori* -specific T- and B-cell clones, the inflammatory response is not sufficient to eradicate the organism from its host [(5,6)]. The chronic immune response induced could afford a colonization advantage for the bacteria by providing improved availability of adhesion places. An example of this is the resulting increase in class II major histocompatibility complex (MHC) and CD74, induced by IFN-γ and IL-8 that are used as receptors by *H. pylori* [(7,8,9)]. The CD74 chain was thought to function mainly as an MHC class II chaperone, which promotes an endoplasmic reticulum (ER) exit of MHC class II molecules, directs them

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to endocytic compartments, prevents peptide binding in the ER, and contributes to peptide editing in the MHC class II compartment [10]. Class II MHC and invariant chain expression was believed to be restricted to classical antigen-presenting cell (APC); but during inflammation, other cell types including human mucosal epithelial cells, have also been reported to express class II MHC molecules.These cells have a high-level expression of surface CD74, which is polarized to the apical surface [11]. However, in addition to its function as a chaperone molecule, CD74 was shown to have a role as an accessory signaling molecule.

Beswick *et al*. [12] studied in details binding of the *H. pylori* urease A and B subunits to class II MHC and the class II MHC-associated invariant chain CD74. Consequently, the suggestion of the role for CD74 in gastric epithelial cell interaction with *H. pylori* leading to NF-$_K$B signaling results in IL-8 secretion, and that CD74 plays an important role in these events.

## ***Patients and Methods***

### *Patients:*

A total of 64 patients (41females and 23 males), aged between 14 and 66 years (34±1.7years), were screened for this study. Patients attended the Gastroenterology Unit at AL-Kadhimiya Teaching Hospital in Baghdad from 1st April to 1st October 2007, because of recurrent abdominal pain and other gastrointestinal complaints. All patients filled a questionnaire sheet with regard to their general health and were excluded if they had been previously treated for *H.pylori* infection and usage of non steroidal anti-inflammatory drugs (NSAIDs); also Patients with actively bleeding peptic ulcer disease were excluded, as this is a well recognized cause of a false-negative urease test [13]. The study was approved by the ethics committee of the Hospital.

### Determination *of H. pylori*

Endoscopic examination was performed under local pharyngeal anesthesia, during which three biopsies were obtained from grossly inflamed areas of the antrum. One biopsy was used for Ultra Rapid Urease test (URUT) and slide impression smear, while the other biopsy specimens were fixed with 10% buffered formalinized saline, for preparation the paraffin embedded tissue blocks to histological evaluation and Immunohistochemical staining tests (IHC). In addition, blood samples were aspirated from each patient after the endoscopy.

A number of invasive URUT Test, slide impression smear) and non-invasive (anti-*H. pylori* IgG ELISA Test) diagnostic tests were used for the diagnosis of *H. pylori* infection according to [14].

### Immunohistochemical Analysis of CD74

Immunohistochemistry was performed using the labeled streptavidin-biotin (LSAB) immunostaining method and the four-micrometer-thick, formalin-fixed, paraffin embedded serial sections of all biopsies were de-paraffinized and Re-hydrated. For antigen retrieval, pretreatment was performed by microwave heating in *Glyca* solution (BioGenex's U.S. Cat. No HK167-5K) for 5 min. on high power (~700 watts). Peroxidase block then incubation of each one with Mouse anti-Human CD74 (mouse monoclonal antibody, C2430-01E, dilution 1: 4, USBiological) was conducted at 37 $^0$C for 1hour and followed by phosphate-buffered saline washing. Positive immunohistochemical reactions were revealed using DakoCytomation LSAB 2 System-HRP Code K0673 (DakoCytomation, USA), using

immunohistochemistry detection kit as chromogen substrate. Tissue section were counterstained with hematoxylin and mounted with DPX. In negative controls, the primary antibody was omitted**.**

Slides were examined by light Microscope; the expression of CD74 was measured as the same scoring system used by Beswick *et al*. [9]. High expression of the examined tissue was graded as 2 when > 30% of epithelial cells stained positive for CD74, and low expression was graded as 1 when < 30% of epithelial cells stained positive for CD74.

***Results***

According to the non-invasive and invasive diagnostic methods used for the diagnosis of *H. pylori* infection; a significant difference was noticed ($P < 0.05$) between positive and negative *H.pylori* infected patients. Accordingly, Patients were grouped as *H. pylori* positive (n= 47; 73.4%) and *H. pylori* negative (n=17; 26.5%).

**Increase CD74 expression on Gastric Epithelial Cell in Biopsy Samples during *H. pylori* Infection:**

In order to study the expression of CD74 molecules, staining was done by using anti-CD74 clone LN-2. As seen in Figure (1), there was a marked increase in CD74 staining of epithelial cells during *H. pylori* infection. In this study, 64 patient biopsies were examined for CD74 expression. Of the 64 patients, 47 were positive for *H. pylori* infection, and as determined by staining of the 47 biopsies, 44(93.6%) were found to have high CD74 expression and 3 out of 47 (6.4%) were found to have low CD74 expression (Table-1), suggesting there is also a link between inflammation, which is a hallmark response to *H. pylori* infection, and CD74 expression. Of the17 samples from negative *H.pylori* patients, 13 (76.5%) had low CD74 expression and 4 (23.5%) had high CD74 expression. The results revealed that there was a highly significantly difference ($p$=0.0001) between *H.pylori* positive and negative groups.

**Table 1: The CD74 expression on gastric epithelial cells in patient biopsy samples in the presence or the absence of *H. pylori*:**

| CD74 expression on gastric epithelial cells ($P$=0.0001*) | *H. pylori* positive | | *H. pylori* negative | |
|---|---|---|---|---|
| | No. | % | No. | % |
| Low expression | 3 | 6.4 | 13 | 76.5 |
| High expression | 44 | 93.6 | 4 | 23.5 |

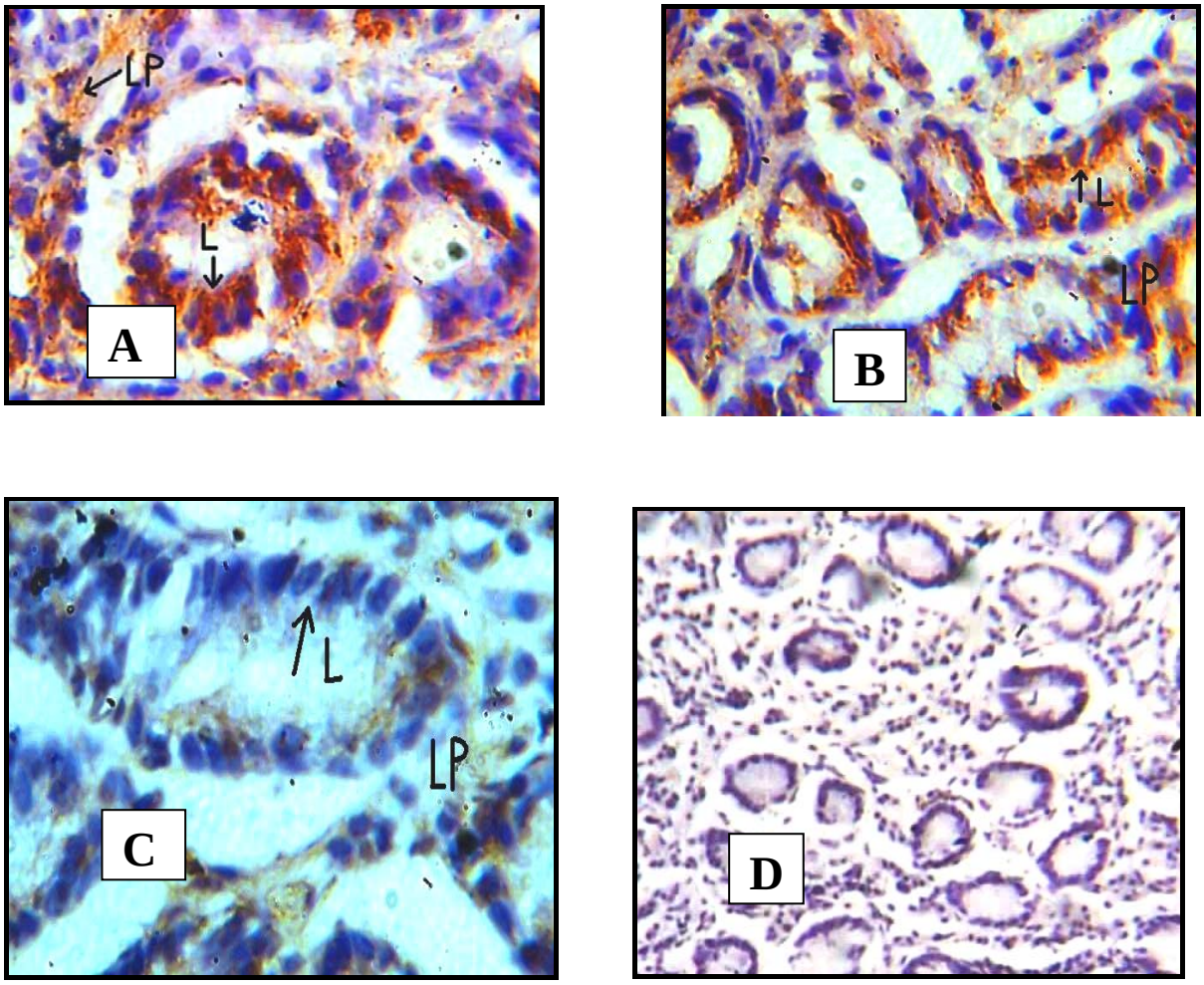


**Figure 1: Immunohistochemical staining (IHC) in formalin fixed paraffin embedded antral biopsies from *Helicobacter pylori* infected (A and B) and uninfected (C)for the expression of the invariant chain CD74 . The sections were stained by DAB chromogen (brown) and counterstained with Hematoxylin (blue). (A and B) CD74 shows increased expression by epithelial cells in samples positive for *H. pylori*; compared with similar biopsies from uninfected biopsies (C); (D) Negative control section. Arrow points to intense apical staining on epithelial cells. L = lumen; LP = lamina propria.**

## ***Discussion***

The variety of functions and signaling leading to immune responses that have recently been implicated in CD74 suggest that there is much more to be revealed about the functions of this molecule.

In this study, immunohistochemistry examination was performed on gastric biopsies from patients infected with *H.pylori* to examine the expression of CD74. Interestingly, the expression of CD74 was evident in uninfected tissue, but the infected tissue showed a marked increase in CD74 expression with a highly significantly difference ($p$ = 0.0001) between *H.pylori* positive and negative cases. The inflamed tissue also had other cells in the lamina

propria that expressed CD74. Specific staining using anti-CD74 clone LN-2 antibodies showed that the highest density of CD74 expression occurred along the apical side of the cells and faint staining was detected at the basolateral side. These results indicate a correlation between *H. pylori* infection and CD74 expression. There was a highly significant association between the high expression of CD74in infected and uninfected patients. These results could be explained in the light of other studies, Beswick and colleagues [8, 9] investigated the interaction of *H. pylori* with the class II major histocompatibility complex (MHC)-associated invariant chain (CD74), which found to be highly expressed by gastric epithelial cells, and they suggested a role for CD74 in gastric epithelial cell interaction with *H. pylori* leading to NF-$_K$B signaling that results in IL-8 secretion. Moreover, Bacterial binding was increased when CD74 surface expression was increased by IFN-γ treatment or by fibroblast cells transfected with CD74, while binding was decreased by CD74 blocking antibodies, enzyme cleavage of CD74, and CD74-coated bacteria. *H. pylori* was also shown to bind directly to affinity-purified CD74 in the absence of class II MHC .Increased CD74 expression by cells that showed increased IL-8 production in response to *H. pylori*, and agents that block CD74 decreased these responses. Therefore, adherence of the bacteria to the gastric mucosa is one of the initial steps of *H. pylori* infection and is an important virulence factor. Many different *H. pylori* adhesins have been identified [15] implying that adherence is a multifactorial process.

In conclusion, the present results show that in *H.pylori* positive biopsy specimens an overexpression of CD74 were observed in infected patients.

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# Detection of Antisperm Antibodies in Sera of Iraqi Males and Females and Their Role in Fertilizing Capacity

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## *Abstract*

***Background:*** Antisperm antibodies (ASAs) have a main role in the immunological infertility. Antisperm antibodies negatively affect sperm movement and interfere with fertilization and may cause abortion .

***Objective:*** to Investigate the occurrence of antisperm antibodies in sera of men and women and their role in fertilizing capacity.

***Method:*** Sixty men and thirteen women were involved in this study . Indirect immunofluorescent test kit was used . As a counterstain , Evans blue pigment was used . The fluorescent microscope was used . For sixty males, seminal fluid analyses were performed. For thirteen females, direct microscopic vaginal tests were done.

***Results:*** Forty five men (75%) and ten women (76.9%) showed positive reactions and antibody titres were either 1/10 or 1/32 .

***Conclusions:*** Serum antisperm antibodies play a significant role in autoimmune infertility and should be treated.

***Keywords:*** serum, antisperm antibodies, infertility, immunity.

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## *Introduction*

Antisperm antibodies can be defined as immmunoglobulins of the IgG, IgA and / or IgM isotype that is directed to various parts of the spermatozoa (head, tail, midpiece or combination thereof )[1]. Antisperm antibodies can be detected in seminal fluid, cervical mucus, oviductal fluid or follicular fluid of women and blood serum of men and women [2]. The occurrence of antisperm antibodies give rise to immunological infertility [3]. In males, testicular trauma, infection, cancer, cryptorchidism and varicocele are involved in generation of antisperm antibodies [1].

In females, the contributing factors include: mechanical such as uterine cervix surgery or chemical disruption of the mucosal layer of the genital tract, foreign antigens gaining access to the female genital tract, lymphocytes in semen , sperm with surface bound antibodies abnormal, senescent or damage sperm, gastrointestinal exposure to sperm and sperm within the peritoneal cavity after transtubal passage[1].

The possible effects of immunologic reaction to fertility are disordered spermatogenesis, inhibiting the effective transport of spermatozoa in male reproductive tract, autoagglutination of ejaculated spermatozoa, sperm cytotoxicity, immobilizing of sperm in the female tract, enhancement of phagocytic clearance of spermatozoa by macrophages, inadequate spermatozoal traverse of cervical mucus, disordered acrosome reaction, blockage of sperm-ovum interaction, induction of sperm immunity in the female, and postfertilization reproductive failure and occult abortion[1,2,4]. Therefore, this study is designed to detect sASAs in both infertile men and women , their causes and role in immunological infertility .

## *Materials and Methods*

Seventy–three infertile patients [ sixty males (82.2%) and thirteen

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females (17.8%) ] attending Institute of Embryo Research and Infertility Treatment at AL–Nahrain University were included in this study during the period from March 2007 to May 2008.

Ages of males ranged from twenty-four to fifty-seven years. Ages of females ranged from twenty-one to thirty-five years. Information concerning smoking, drinking alcohol, varicocele and varicocelectomy were collected from them. To detect antisperm antibodies in the sera of these patients indirect immunofluorescent test kit (Euroimmune,Germany) was used.

This kit contained BIOCHIP slides and each slide contained ten BIOCHIPs coated with smears of human spermatozoa. As a counterstainer, Evans Blue pigment was used. Once the blood sample was collected from each patient, it was centrifuged till the serum was obtained. This serum was collected into an eppendorf tube and was kept at 0 C until performing the test. To perform the test, four serial dilutions for each sample were prepared (1/10, 1/100, 1/1000, 1/10000).

To prepare 1/10 dilution, 11.1 microleters of the serum were added to 100 microleters of phosphate buffer saline-Tween (PBS–Tween). For the other dilutions, 11.1 microleters of each previous dilution were added to 100 microleters of PBS–Tween. Then 25 microleters of each dilution of each sample were applied to each reaction field of the reagent tray. The reaction started by fitting the BIOCHIP slide into the corresponding recesses of the reagent tray. Each sample contacted with its BIOCHIP. Then the slide was incubated for 30 minutes at room temperature.

After incubation, the BIOCHIP slide was rinsed with a flush of PBS–Tween and was immediately immersed in a dish containing PBS-Tween. After 5 minutes, 20 microleters of fluorescein–labeled anti–human globulin were added to each reaction field of a clean reagent tray and within 5 seconds, the BIOCHIP slide was removed from the dish and the slide was immediately put into the recesses of the reagent tray. The slide was protected from the direct sunlight and was incubated for 30 minutes at room temperature.

After 30 minutes, the BIOCHIP slide was rinsed with a flush of PBS-Tween and was put into a dish containing 150 millileters of phosphate buffer added to it 10 drops Evans Blue pigment as a counterstainer and was left for 5 minutes. Then, 10 microleters of glycerol per each reaction field was added onto a coverglass and within5 seconds the BIOCHIP slide was removed. The BIOCHIP slide with the BIOCHIPs facing downwards was put onto the prepared coverglass and it was now ready for checking by using fluorescent microscope at power 40X.

Under this power, any portion of spermatozoa with green colour indicated positive reaction and any portion of spermatozoa with red colour indicated negative reaction .

Of 60 males, seminal fluids were collected by masturbation after three days of abstinence and seminal fluid analyses were performed within two hours. It was estimated according to WHO guideline in year 1999. The following parameters were concerned in this study: sperm agglutination, sperm motility and presence of pus cells. Of 13 females included in this study, vaginal swabs were done and subjected to direct microscopic examination.

***Results***

In this research only the first dilution (1/10) showed positive reaction and the other dilutions (1/100, 1/1000, 1/10000) exhibited no positive reactions

**Table 1: the reactivity exhibited by both males and females.**

| Reactivity and % | Sex | Male | Female |
|---|---|---|---|
| Negative reaction and % | | 15 (25%) | 3 (23%) |
| Weak reaction and % | | 22 (36.7%) | 4 (30.8%) |
| Moderate reaction and % | | 23 (38.3%) | 6 (46.2%) |
| Total | | 60 (100%) | 13 (100%) |

In this test, weak reactions were given the antibody titer 1/10 and moderate reactions were given the antibody titer 1/32.

**Table 2: the attachment of serum antisperm antibodies to various portions of spermatozoa .**

| Sex and nomber | Portion of spermatozoa | Only head | Only mid - piece | Only tail | Head and mid – piece | Head and tail | Mid – piece and tail | Head mid-piece and tail |
|---|---|---|---|---|---|---|---|---|
| Nomber of males | | 13 | 0 | 4 | 1 | 24 | 0 | 3 |
| Nomber of females | | 1 | 0 | 1 | 0 | 8 | 0 | 0 |

**Table 3: Descriptive data of some sperm parameters in infertile males.**

| Sperm parameters | Percentage of infertile patients | Normal value |
|---|---|---|
| Percentage of sperm activity grade A | 1-Lower than 25% [No.= 54 (90%)]<br>2-Positive serum ASAs and sperm grade A activity = zero [ No. = 34 (56.67%)]<br>3-Positive serum ASAs and normal sperm grade A activity [No.= 1 (1.67%)] | ≥ 25% |
| Positive Serum ASAs | 1- No.= 45 (75%) | Nil |
| Percentage of sperm agglutination | 1-More than 10% sperm agglutination [No.= 26 (43.33%)]<br>2-Negative serum ASAs reaction in the normal range of sperm agglutination. | < 10% |
| Pus cells count | 1-Patients with notable no. of pus cells [No.= 11 (18.33%)]<br>2-Patients with notable no. of pus cells and positive serum ASAs [No.=10 (16.67%)]<br>3-Patients with notable no. of pus cells and negative serum ASAs No.=1 (1.67%) | ≤1 cell/HPF |

Of thirteen females subjected to direct microscopic vaginal examination, notable pus cells were recognized in the smears of four females (30.77%).

Of sixty males, thirty–nine (65%) were suffering from primary infertility. Fifteen (25%) of them exerted weak reaction, sixteen (26.67%) exerted moderate reaction and eight (13.33%) exerted no reaction. Also of sixty males, twenty–one (35%) were suffering from secondary infertility. Of these twenty–one males, eight (13.33%) exhibited weak reaction, six (10%) exhibited moderate reaction and seven (11.67%) exhibited no reaction.

Of thirteen females, six (46.15%) were suffering from primary infertility. Of these six females, two (15.38%) showed weak reaction, three (23.08%) showed moderate reaction, and one (7.69%) showed negative reaction. Out of thirteen females, seven (53.85%) were suffering from secondary infertility. Of these seven females, two (15.38%) showed weak reaction, three (23.08%) showed moderate reaction, and two (15.38%) showed negative reaction.

In this study, none of males or females was alcoholic. In addition, none of females was smokers. Out of sixty males, thirty–eight (63.33%) were mild to heavy smokers. Of these thirty–eight, seventeen (28.33%) exerted moderate reaction, fourteen (23.33%) showed weak reaction, and seven (11.67%) exhibited no reaction.

In this study, of three primary infertile males (5%) subjected to varicocelectomy, one (1.67%) showed weak reaction and the other two (3.33%) gave no reaction. Varicocele was detected in six males (10%). Of these six males, four (6.67%) were primary infertile and of these four males, three (5%) exerted moderate reaction and one (1.67%) showed no reaction. The other two males (3.33%) were secondary infertile and one (1.67%) showed moderate reaction and the other (1.67%) exerted weak reaction.

## ***Discussion***

Infertility is defined as the inability of a couple to conceive after a period of twelve months of intercourse without the use of contraception[1]. Table 2 shows the attachment of serum antisperm antibodies to various portions of spermatozoa contained in BIOCHIP slides. Antisperm antibodies is an important cause of immunological infertility in humans and may result from the presence of antisperm antibodies in sera of individuals [5, 6]. Therefore, this study was designed to study antisperm antibodies in circulating blood of infertile patients.

The results revealed the prevalence of serum antisperm antibodies in both primary and secondary infertile males and females. These results agreed with Dorr *et al*. [7] who mentioned that antisperm antibodies were present in a high percentage of infertile patients.

The results indicated that there was a significant correlation between smoking and immune infertility. Moreira and Lipshultz[8] demonstrated that exogenous agents as nicotine which considered as a gonadotoxic agent, might interfere with male infertility. Autoimmune infertility may result from gonadotoxins[4].

Bes [3] stated that there was an association of varicocele with an autoimmune response against spermatozoa.

Antisperm antibodies cause clumping or agglutination of sperms [4]. Hijort[9] revealed that when notable antisperm antibodies titres were detected in serum the amount of antisperm antibodies on the spermatozoa would also be notable . Antisperm antibodies

had a significant negative effect on sperm motility and increase the proportion of motile sperms involved in agglutination [2, 10]. (Table 3) shows the negative effect of antisperm antibodies on sperm motility.

Jose [1] demonstrated that infections in the genital tract were associated with the generation of antisperm antibodies.

For the treatment, the immunosuppressive corticosteroid prednisolone was administered orally to the patients in dosages 5mg /three times a day for two weeks. This drug showed considerable good results. Khudher [11] demonstrated that prednisolone was administered to patients with antisperm antibodies and the patients had reduced serum antisperm antibodies after treatment and, in some cases, an apparent increased chance of pregnancy.

## ***Conclusion***

Serum antisperm antibodies play a considerable role in immune infertility and should be treated.

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# Comparison between Bacterial Vaginosis and Candidiasis in Relation to Estradiol Level and Vaginal pH in Some Infertile Iraqi Women

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## *Abstract*

***Background:*** Female fertility are affected by several factors including microbial and non microbial agents. Microbial infection is one of the most important causes for female infertility. The level of pathogenicity of microbial infections are affected by wide range of factor including age, physiological status, phase of menstrual cycle and race.

***Objective:*** Comparison between bacterial vaginosis (B.V) and candidiasis in relation to Estradiol (E2) level and vaginal pH in some infertile Iraqi women

***Methods:*** The study population was a subset of 109 infertile women attending Institute of Embryo Research and Infertility Treatment at Baghdad University, throughout the period from June till November 2004. Those infertile women were subjected to clinical examination by measuring vaginal pH, vaginal swabs collection to diagnosis of B.V using Amsel clinical criteria beside various micro-biological methods and diagnosis of candidiasis using mycological methods and serum collection from aspirated venous blood at late follicular phase for detection of E2 level.

***Result*:** Forty eight infected infertile women were diagnosed with B.V from 109 infertile women. In those women the Estradiol mean was 41.17 Pg/mL near to lower limit of normal range of E2 level ( 18-147 pg/mL) and lower than E2 mean of healthy control group 132.5 Pg/mL in this study and most of them 93.75% had vaginal pH greater than 4.5. 24 cases with candidiasis were diagnosed from 109 infertile women. In those women the E2 mean was 183.2 Pg/mL higher than upper limit of normal range of E2 level and higher than E2 mean of healthy control group and candidial infection occur in normal pH range of 3.5 to 4.5

***Conclusions:*** The results of the present study appeared that the hormonal disturbance which was associated with different infertility conditions may be predisposing factor in development of B.V and develop candidiasis among infertile women.

Elevated vaginal pH in infertile women who had B.V could be due to estrogen deficiency while normal pH in candidial infection because estrogen hormone increases cellular glycogen content which favors growth of Lactobacilli that metabolize glycogen to lactic acid and then producing an acidic environment.

***Kay words:*** Bacterial vaginosis, candidiasis, Estradiol level, infertile women

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## *Introduction*

The normal vaginal environment is characterized by a dynamic interrelationship between *Lactobacilli acidophilus* and other endogenous flora, estrogen, vaginal pH and metabolic byproducts of flora and pathogens. Vaginitis develops when the vaginal flora has been altered by introduction of pathogen or by changes in the vaginal environment [1, 2].

Bacterial vaginosis is the most common infectious cause of vaginitis characterized by imbalance of vaginal ecosystem while candidiasis is the second most common cause of vaginitis [1,3,4] .These types of vaginal infections

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differ from others in their relation to estradiol level, cellular glycogen content and the acidity of vaginal fluid [5-7]. Hypoestrogenic problems that may be caused by hypothalamic, pituitary and ovarian disorders which are the most common causes of infertility [8-11] that lead to interruption of estrogen production, produces mucosal atrophy, reduction in cellular glycogen which decreases number of *Lactobacilli* leading to increased vaginal pH which enhances the proliferation of *B.V* related bacteria resulting in vaginal infection with *B.V* [1,7,12].Polycystic ovary syndrome(PCO) is one of the most common endocrine disorders and many PCO women have elevated estrogen level [13,14] which is one of the most important risk factors of candidal infection [15] ,because elevated estrogen induce increase in glycogen content in vaginal epithelial cells which favors growth of yeast cells, as well as favors growth of *Lactobacilli* that metabolize glycogen to lactic acid producing an acid pH of 3.5-4.5 [12,16-21] Furthermore, yeast cells possess receptors for estrogen which enhance mycelial formation as well as estrogen was found to reduce the ability of vaginal epithelial cells to inhibit the growth of *C. albican* [22-24].

## Patients, Materials and Methods

### Study population

One hundred nine infertile women within reproductive age. 72 representing patients group which complaining from vaginal discharge with or without other symptoms and 37 were regarded as control group without any signs and symptoms of vaginal infection were attending Institute of Embryo Research and Infertility Treatment at Baghdad University through the period June till November 2004 were studied. Infertile women were given a self-administered questionnaire with following information was collected Age, Last menstrual period, Length of cycle, Length of period of last antibiotic treatment, Vaginal discharge and associated symptoms. (Itching, Lower abdominal pain and Dyspareunia) and Infertility duration.

### Microbial Examination:

Bacterial vaginosis is diagnosed conventionally when at least three of four Amsel clinical criteria are present including (Thin homogenous discharge, vaginal pH greater than 4.5, fishy odor of vaginal discharge and presence of clue cell) [25-27]. Accurate diagnosis of vaginal candidiasis depends on culture techniques that will yield correct identification of fungal pathogens [4] by using germ tube test which perform by inoculating several colonies into the test substrate (such as fetal bovine serum) and incubating the suspension at 37°C for 3 hours. [28-29].

### Clinical examination

The external genitalia are first inspection for erythema, edema or excoriation [11, 30]. Non-lubricated bivalve speculum was inserted into the vagina for inspection of vaginal and cervical erosion and color of mucus. Vaginal discharge was also inspected for its characters [31-32].

### Vaginal pH

pH measurement was easily carried out by clipping a short piece of pH paper (range pH 1-14) to a forceps and dipping it into the vaginal discharge in the lateral fornices, [33]. The color was then compared to the colors and corresponding pH values on a standard chart.

### Sampling collection

### Serum collection

Before vaginal examination, about five mL of venous blood sample was

collected from each woman at cycle day 12.

The blood was delivered into a sterile screw plastic tube, and then centrifuged at 3000 RPM for 5 minutes the serum was then collected into another sterile tube and was kept in deep freeze at -20°C for estimation of estrogen level.

***Swabs Collection***

Three sterile cotton tipped swabs were used to collect vaginal discharge from the posterior fornix [32,34,35]. Two swabs were used for cultivation. One blood agar and chocolate agar were incubated anaerobically at 37°C. The second blood agar MacConkey's and Sabouraud's agar were incubated aerobically at 37°C for 72 hour. The third swab was used for the preparation of Gram's stain, Whiff test and KOH preparation by rolling this swab on two clean glass slides first smear was mixed with a drop of 10% KOH for Whiff test then examine for detection of Candida organisms and the second smear was fixed by heat and then stained according to the Gram's stain procedure [21].

**Estrogen *($E_2$) assay***

Estrogen level of the frozen serum of infertile women was estimated by VIDAS Estradiol 11 ($E_2$ 11) kit-from biomerieux sa-France.

**Table 1: show the ranges of expected normal values of $E_2$ (from biomeriex Sa).**

| Normal subjected female | $E_2$ range, pg/mL |
|---|---|
| **- Follicular phase** | **18 – 147** |
| **- Pre-ovulatory peak** | **93 – 575** |
| **- Luteal phase** | **43 – 214** |
| **- Menopause** | **< 58** |

**Note:**

$E_2$ 11 is an automated quantitative test for use on the VIDAS instruments for the quantitative measurement of 17β estradiol in human serum or plasma (lithium heparinate), using the (ELFA) technique.

***Statistical methods***

The Statistical methods that were used in study to analysis the results of this study include Chi-square test and ANOVA test

***Result***

**Vaginal pH as predictor for candidiasis and bacterial vaginosis in infertile women**

The results of vaginal pH testing in both patients and control groups are shown in table (2), which showed highly significant ($P<0.001$) relationship between *B.V* and vaginal pH when compared to the control group 45/48 (93.75%) of infertile women who had *B.V* were found to have vaginal pH equal or above 4.5 versus 31/37 (83.8%) of healthy control group had vaginal pH less than 4.5. While no significant relationship between high vaginal pH and candidial infection compared to the control group, however 18/24 (75%) of infertile women with candidal infection had vaginal pH less than 4.5.

**Table 2: Vaginal pH as predictor for for candidiasis and bacterial vaginosis in infertile women**

| Type of vaginal infection | Vaginal PH | | | | Chi-square Test p-value |
|---|---|---|---|---|---|
| | < 4.5 | | ≥4.5 | | |
| | N | % | N | % | |
| Bacterial vaginosis N=48 | 3 | 6.25 | 45 | 93.75 | P<0.001 HS |
| Candidiasis N=24 | 18 | 75 | 6 | 25.0 | P>0.05 NS |
| Normal N=37 | 31 | 83.8 | 6 | 16.2 | * |

***P-value in relation to normal group **NS: Non significant**
*****HS: High significant **** N: number of patient**

**The relation between $E_2$ mean and incidence of different types of vaginal infection among infertile women**

The different effects of $E_2$ level on occurrence of B.V and candidiasis among infertile women are descried in table (3). Highly significant difference (P<0.001) was demonstrate between both B.V and Candidiasis and healthy control group in $E_2$ mean and between each other . The $E_2$ mean of 48 infertile women who had *B.V* was 41.17 Pg/mL ±4.15 lower than $E_2$ mean of 37 healthy infertile women 132.5 Pg/mL±11 and $E_2$ mean of candidiasis cases, while $E_2$ mean of 24 infertile women with candidaisis was 183.2 Pg/mL ±32.6 higher than $E_2$ mean of control group and $E_2$ mean of B.V. From the above results,*B.V* occurred at lower $E_2$ level (mean 41.17 Pg/mL) while candidial infection occurred at higher $E_2$ level (mean 183.2 Pg/mL)

**Table (3) The relation between $E_2$ mean and incidence of different types of vaginal infections among infertile women**

| Type of vaginal infection | N | E2 Mean ± S.E.M |
|---|---|---|
| Bacterial vaginosis | 48 | 41.17 ± 4.15 |
| Candidiasis | 24 | 183.2 ± 32.6 |
| Normal | 37 | 132.5 ± 11.0 |

* ANOVA test P<0.001 High significant
Date are Mean ± SEM
N: number of patient
H.S: High significant

***Discussion***

The recurrent hormonal treatment and personal hygiene of infertile women were considered.

**Vaginal pH as predictor for candidiasis and bacterial vaginosis in infertile women.**

Measurement of vaginal pH is useful, effective and inexpensive for screening purposes. In the present study, table (2) shows highly significant relationship (P<0.001) between vaginal pH and *B.V*. The pH value in 93.75% of infertile women who had *B.V* was greater than 4.5. This result is in agreement with other studies who depend on Amsel clinical criteria which have considered vaginal pH ≥ 4.5 as one of four criteria to confirm the diagnosis of *B.V* [27, 36-38]. The failure of any of the following three endocrine glands hypothalamus, pituitary and ovary lead to inhibition of estrogens production by ovary in women within reproductive age [8, 9, 11]. On the other hand, estrogen deficiency in menopausal women lead to elevated vaginal pH, this is due to lack of glycogen content and disappearance of *Lactobacilli* [39]. Therefore, the two states are comparable. As well as, anaerobic bacteria that are associated with *B.V* produce organic amine, which raise vaginal pH [17, 33, 40]. Our data suggest that the elevated vaginal pH in infertile women who had *B.V* could be due to estrogen deficiency and presence of amines, which are produced by anaerobic bacteria that are responsible for further increase in vaginal pH in those women.

Only 16.2% of healthy control group had vaginal pH ≥ 4.5 this may be due to either recent sexual intercourse or douching or touching cervical mucus [41, 42].

This study found no significant correlation between vaginal pH and candidial infection when compared to the control group, because 75% of infertile women with candidiasis and 83.8% of healthy control group had vaginal pH less than 4.5. Thus, the results of this study are in good accordance with many studies demonstrating candidial infection occur in normal pH range from 3.5 to 4.5 [11, 43-45].

Many recent studies emphasized that some infertile women with polycystic ovary, and other causes of hyperandrogenemia have elevated or normal level of estrogens [9,13,14,46]. It was reported that estrogen increases cellular glycogen content which favors growth of *Lactobacilli* that metabolize glycogen to lactic acid producing an acid pH of 3.5-4.5 [12,16-19].

The result of our study suggests that the normal vaginal pH in some infertile women infected with candidiasis may be due to elevated estrogen level and heavy colonization of vagina by *Lactobacilli.*

**The relation between $E_2$ level and occurrence candidiasis and bacterial vaginosis among infertile women**

The relation between serum estrogen ($E_2$) level and occurrence of candidiasis and bacterial vaginosis among infertile women are shown in table (3). The results of this study demonstrated that the $E_2$ mean of 48 infertile women who had *B.V* was 41.17 Pg/mL near to lower limit of normal range of $E_2$ level at follicular phase. From the above result, one can conclude two things, these women were more likely to have hypoestrogenic problems, these problems may be caused by hypothalamic or pituitary or ovarian disorders which are the most common causes of infertility [8-11]. The other thing is *B.V* occurred at low estrogen level, the reason of this state is explained by many recent studies who reported estrogen deplation caused by castration, aging or other causes that lead to interruption of estrogen production, produces mucosal atrophy, reduction in cellular glycogen which decreases number of *Lactobacilli*

leading to increased vaginal pH which enhances the proliferation of *B.V* related bacteria resulting in vaginal infection with *B.V* [1,7,12]. The results of this study suggest that the hypoestrogenic state which is associated with some infertility conditions could play a role in development of *B.V* among those infertile women. In the present study, serum $E_2$ level was found higher than the upper limit of normal range of $E_2$ level at follicular phase in 24 infertile women who had candidiasis 183.2 Pg/mL this may be due to endocrine disorders which is one of the most important risk factors of candidial infection [15]. Elevated estrogen induce increase in glycogen content in vaginal epithelial cells which favors growth of yeast cells [20, 21].

Furthermore, yeast cells possess receptors for estrogen which enhance mycelial formation as well as estrogen was found to reduce the ability of vaginal epithelial cells to inhibit the growth of *C. albican* [22-24].

Polycystic ovary syndrome is one of the most common endocrine disorders and many infertile women with this syndrome have elevated estrogen level [13, 14].

From the above results of this study it was concluded that elevated estrogen level in some infertile women may encourage colonization of *Candida spp*. in vagina leading to candidiasis

From table (3) it can be also observed that $E_2$ mean of infertile women with *B.V* was lower than $E_2$ mean of healthy women which were regarded as control group which was within normal range of $E_2$ level also $E_2$ mean of infertile women with candidiasis was higher than $E_2$ mean of those healthy women.

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# Some Diagnostic Aspects of Celiac Disease in Iraqi children.

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## *Abstract*

***Background***: There has been an increasing appreciation of the high prevalence of celiac diseas around the world and efforts are continuing to clarify the variable diagnostic problems of the disease.

**Objective:** We tried to throw light over some of these problems in a group of Iraqi children.

**Methods:** Ninety-three patients with features of malabsorption were evaluated for celiac diseas, by assessing serum IgA tTG, both IgG & IgA AGA, serum IgA level and a small intestinal biopsy.

***Results:*** Fifty-eight out of ninety-three patients proved to have celiac diseas according to the histopathological picture. Sensitivity of serological tests in general ranged between 50- 77%, but tTG was 100% specific. Patients with more severe histopathological changes showed more serological positivity and higher antibody titers.

Eleven cases of Giardiasis were diagnosed (on biopsy specimen) out of the whole sample, giving variable histopathological changes & serological responses.

***Conclusion:*** celiac diseas is a prevalent problem in Iraqi children. We share with other countries the diagnostic problems of the disease, but there seems to be some additional aspects, that are peculiar to developing countries, implying the need for diagnostic strategies specific to these areas.

***Key words:*** celiac disease, serological tests, histopathology

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## *Introduction*

Celiac disease (CD) is a problem prevalent around the world. It's prevalence has been increasingly appreciated recently, studies primarily in Europe, but also in the United States, now suggest that it's prevalence is roughly 1%, among the general population[(1)]. This better appreciation occurred with the advent of newer diagnostic techniques (including the easy-to-administer serology tests), that clarified many of the non-specificity of the diagnostic techniques [(2)].

Has been frequently rejected by families as being unduly invasive, especially when evaluating problems as anemia, short stature or even chronic diarrhea in their children.

Reports of CD in Iraq first appeared in 1975 by Al-Hassany [(3)], relying on having a proper small intestinal biopsy showing characteristic histology. However, in Iraq as in other developing countries, the facility for obtaining a biopsy is not always available; in addition, this invasive procedure

On the other hand, children in developing countries are prone to multiple disease states that may lead to clinical manifestations mimicking CD, and even to proximal small intestinal mucosal lesions like: tropical sprue, persistent infection or infestation, post infectious complications or protein energy malnutrition [(5, 6)]. A situation which is pressing to provide better diagnostic tools, to avoid misinterpretation of test results and if at all possible to

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spare the child from costly, invasive techniques.

## ***Patients & Methods***

A total of 93 consecutive cases, with malabsorption, presenting with different complaints: chronic diarrhea, chronic abdominal pain, abdominal bloating, anemia, growth retardation….etc, were evaluated for having celiac disease. Their ages ranged from 1- 18years. All those patients were referred to the Iraqi center for gastrointestinal diseases in Baghdad, during the period from Sept. 2001-Apr 2002. After fulfilling the routine initial clinical and Lab evaluation, they have undergone an endoscopic small intestinal biopsy and serological evaluation for celiac disease.

Biopsies were interpreted histopathologically according to the gluten-sensitivity spectrum described as follows [7]: Marsh 0: normal, Marsh I: there is intraepithelial lymphocytes (IELs), Marsh II: IELs+ hyperplasia of crypts, Marsh III: influx of inflammatory cells, hyperplasia of crypts and villous atrophy, III A: partial villous atrophy (PVA)-(villous / crypt ratio< 1:1)

III B: subtotal villous atrophy (SVA), villi are clearly atrophic, but separated villi are still recognizable.

III C: total villous atrophy (TVA), villi are rudimentary or absent.

Diagnosis of CD was only been made by demonstration of Marsh III lesion in small intestinal biopsy [7].

Serological testing included:

- Enzyme immunoassay kit for detection of human anti-tissue transglutaminase IgA in serum (Biohit-Finland)

Values <10 AU (arbitrary unit) were regarded –ve, 10-15 weak +ve, >15 +ve.

- Enzyme immunoassay kits for detection of both Anti-gliadin IgG and IgA antibodies in serum (Biohit-Finland)

In<2yr of age: a titer <50 regarded as –ve, 50-100 weak +ve, >100 +ve

- Single radial immunodiffusion test (SAIDT) plates (Sanofi Diagnostic Pasteur, Inc-USA): for measurement of total IgA level in serum.

Analysis between different variables was done using Chi-square test and student T-test. The level of significance was considered when P-value <0.05.

## ***Results***

A total of 93 patients with different complaints suggestive of malabsorption were evaluated for the diagnosis of CD (51 males), their ages ranged from 1 yr- 18 yrs. Fifty eight were diagnosed as having celiac disease according to histopathological picture (28 males, male/female ratio:0.93), their ages ranged from 18 mo- 18yrs, with a mean of 9.5yrs.

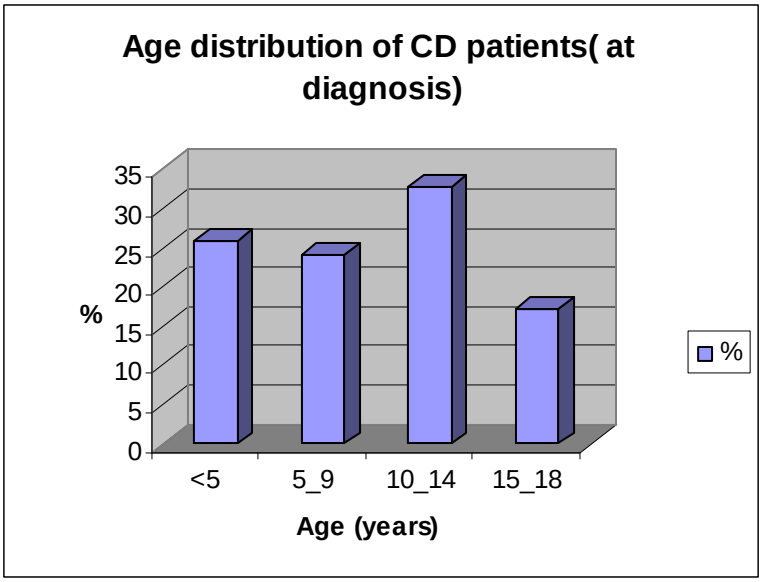


**Figure 1: shows that the disease was diagnosed mostly in the age group 10-14 yrs, next to it those < 5yr of age.**

The diagnosis relied on histopathological findings (the presence of Marsh III changes), from table (1), it can be calculated that 50% of the patients were having severe HP changes, Marsh III C (total villous atrophy TVA), and especially for the older aged patients (>10yr), in whom TVA was seen in 80% of them.

**Table 1: Correlation between age & histopathological severity of CD patients**

| Age(years) | HP stage | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | Marsh A | | Marsh B | | Marsh C | | | |
| | No. | % | No. | % | No. | % | No. | % |
| <5 | 5 | 33.3% | 4 | 26.6% | 6 | **40%** | 15 | 100 |
| 5-9 | 3 | 21.4% | 5 | 35.7% | 6 | **42.8%** | 14 | 100 |
| 10-14 | 6 | 31.5% | 4 | 21% | 9 | **47.4%** | 19 | 100 |
| 15-18 | 2 | 20% | 0 | 0 | 8 | **80%** | 10 | 100 |

Table (2) , shows that tTG assay gave moderate sensitivity and perfect (100%) specificity, while the antigliadine antibodies (igG and IgA) being both less specific, the IgG type was somewhat more sensitive.

**Table 2: Serological markers in celiac patients & controls**

| Patients | Total | tTG | | IgA AGA | | IgG AGA | |
|---|---|---|---|---|---|---|---|
| | | +ve | -ve | +ve | -ve | +ve | -ve |
| Celiac | 58 | 35 | 23 | 29 | 29 | 45 | 13 |
| Non Celiac | 35 | 0 | 35 | 5 | 30 | 5 | 30 |
| Sensitivity | | 60.3% | | 50% | | 77.6% | |
| Specificity | | 100% | | 85.7% | | 85.7% | |

In patients with severe histopathological changes (total villous atrophy or Marsh IIIC) about 90% of them were seropositive (~90%) for tTG, and such patients showed much higher titers than others with milder histological changes, the difference was statistically significant. The same picture was shown with the antigliadin antibodies, table (3)

**Table 3: Serological titers against severity of histopathological changes**

| Marsh III | No. | tTG+ve | tTG titer | | | |
|---|---|---|---|---|---|---|
| | | No. | % | Mean±SD | Median | |
| A | 16 | 6 | 37.5 | 9.69±10.54 | 4 | P value: 0.000289 (significant) |
| B | 13 | 3 | 23.1 | 7.31±11.39 | 3 | |
| C | 29 | 26 | **89.7** | 24.03±14.44 | **25** | |
| Marsh III | No. | IgA AGA+ve | IgA AGA titer | | | |
| | | No. | % | Mean±SD | Median | |
| A | 16 | 5 | 31.3 | 40.5±56.18 | 14.5 | P value: 0.00045 (significant) |
| B | 13 | 2 | 15.4 | 17.23±34.68 | 0 | |
| C | 29 | 22 | **75.9** | 75.45±75.16 | **68** | |
| Marsh III | No. | IgG AGA +ve | IgG AGA titer | | | |
| | | No. | % | Mean±SD | Median | |
| A | 16 | 12 | 75 | 60.56±61.55 | 41.5 | P value: 0.00246 (significant) |
| B | 13 | 7 | 53.8 | 32.69±63.08 | 30 | |
| C | 29 | 26 | **89.7** | 100.35±61.93 | **98** | |

Giardiasis was diagnosed in 11 (out of the 93 studied cases), by demonstrating the parasite attached to the mucosal surface of the biopsy, three of these patients showed histological changes (Marsh III A or

B), two of them with +ve IgG AGA, the third one was seronegative for all the 3 serological markers but was at the same time deficient in the total IgA, one patient reacted positively to the IgG antigliadin only with normal histology and was dismissed as Giardiasis. The remaining 7 cases of giardiasis were seronegative to all tests with normal s.IgA level. whether these results were due to Giardiasis or a concomitant celiac disease?, anyhow the 3 patients were put on gluten free diet (GFD) in addition to antiGiardia therapy, but unfortunately long-term follow up was not possible for these patients.

**Table 4: Cases of Giardiasis**

| Marsh score | | AGA | | tTG | IgA |
|---|---|---|---|---|---|
| | | IgG | IgA | | |
| 1 | 0 | -ve | -ve | -ve | Normal |
| 2 | III B | -ve | -ve | -ve | ***Deficient*** |
| 3 | 0 | -ve | -ve | -ve | Normal |
| 4 | 0 | +ve | -ve | -ve | Normal |
| 5 | III A | +ve | -ve | -ve | Normal |
| 6 | III B | +ve | -ve | -ve | Normal |
| 7 | 0 | -ve | -ve | -ve | Normal |
| 8 | 0 | -ve | -ve | -ve | Normal |
| 9 | 0 | -ve | -ve | -ve | Normal |
| 10 | 0 | -ve | -ve | -ve | Normal |
| 11 | 0 | -ve | -ve | -ve | Normal |

***Discussion***

People until recently thought that the geographical distribution of celiac disease was mostly restricted to Europe and other developed countries, nowadays in part due to the availability of the simple serological diagnostic tests, globalization of the problem of Celiac disease is ensured, following the recognition of increasing reports of the various forms of the disease from developing countries [8,9].

Celiac disease formed a significant part of our patients evaluated for malabsorption (about 62%). In an Indian study by Behera etal [10], CD formed 72% of causes of malabsorption in children and 52% of those in adults, pointing to the higher propensity of CD as a cause of malabsorption in developing countries.

About half of our CD patients were older than 10 years,with a similar age incidence also documented by a previous Iraqi study. (Mohammed et al, 2001)[11], whether this was due to delayed diagnosis of the problem in our pediatric population, or probably mimicking the changing picture of CD reported by several researchers [12,13] in which the median age at presentation in children has shifted from early to late in the first decade of life. Still, most of our patients showed severe H.P changes (TVA), especially demonstrated in the older aged group, strengthening the probability of delayed diagnosis.

Serological markers showed variable sensitivity rates in our patients, in general ranging between 50% -77%, while tTG seemed to be specific enough to ensure the presence of celiac disease. The combined determination of AGA (IgG & IgA)

and tTG (IgA) gave a better diagnostic ability, especially if there was concordance in the results of the 3 antibodies. The positive predictive value of tTG was 100%, while the negative predictive value of the 3 antibodies was 60.3%, this may offer a good strategy for disease identification with 100% specificity of the 60.3% of untreated CD patients, and for the exclusion of nearly 100% of non-celiac patients from unnecessary biopsy.

The validity of testing for serological markers in our studied group seemed to be comparable with the average international figures(14-18), accept for a lower sensitivity of tTG; our data gave sensitivity 60%, while most researchers agree to a sensitivity around 90-100% even those from developing countries[12,19,20,21]. In general these antibody tests are thought to fare less well in the clinical practice setting than in the research setting [22,23]. Standardization and quality control of these tests is an important issue [24,25], and attempting a national standardization initiative to achieve this goal specifically in developing countries [26,27] due to the some what peculiar state of CD diagnosis there.

Tissue transglutaminase, being simpler to perform than antiendomysial antibodies, with the high concordance between the two, has been repeatedly recommended as the serology of choice in developing countries [19, 28]. According to our results, we may recommend the cumulative outcome of tTG and both Antigliadines giving better prediction of the disease in these areas.

Patients with severe H.P changes, in the studied patients, showed higher positivity of serological markers, 89.7% for tTG, 75% for IgA AGA, and 89.7% for IgG AGA antibodies, in addition those patients gave the highest titers, much above the cutoff value. This same picture has led Barker etal [29] from Canada, and Danaldson et al [30] ,at the University of Utah and the University of California Irvine, in their retrospective analysis to suggest raising the cutoff value of tTG to >100 IU (in patients with normal IgA), finding that about 96-98% of such patients had positive biopsy and proved to have CD, hoping in future to avoid totally the need for biopsy [29], but still this would need further approval by other studies in order to be applied practically, and the time to abandon an intestinal biopsy in the diagnosis of CD seems not to have come yet [2,13,21,31].

Roughly, about 10% of cases of CD are difficult to diagnose because of lack of concordance among serologic, clinical, and histologic findings [19]. Even mild mucosal changes (Marsh I, II), could be a presentation of celiac disease, and in symptomatic patients, provided other diagnostic criteria were included (HLA haplotype …etc), GFD would be indicated [32-35]. While in children in developing countries, mild–moderate mucosal changes are less specific as they could be induced by persistent enteric infection or parasitic infestation…etc, the so-called environmental enteropathy [28,36] rather than CD, still some recent reports are appearing from these countries, that even there, CD might present with variable histological pictures and the diagnosis maybe missed or delayed if based only on severe enteropathy [27].

Behera [India], have shown that patients with malabsorption harbor more pathogenic parasites as compared to healthy controls [8]. Giardiasis was diagnosed by duodenal biopsy in our studied patients in 11 out of 93 cases. All the 11 patients were seronegative for tTG & IgA AGA, 3 of them showed severe mucosal changes (Marsh III), with variable reactions towards only the IgG AGA, whether

this whole picture was inflicted by Giardial infection alone or a coexistent gluten hypersensitivity, unfortunately follow up was not possible to evaluate their response to gluten free diet.

Revision of the diagnostic criterias of Celiac disease (in the form of standardization of serologic tests and pathologic criteria) is persistently called for by workers in this field [1,35], with the need for a special consensus for diagnosis in developing countries.

Celiac disease is a common cause of malabsorption in Iraqi children, presenting a persistent diagnostic challenge to pediatricians and gastroenterologists, with the variable efficacy and availability of serological tests in the country, and the endemicity of parasitic infestations in the pediatric population with the mucosal changes they may induce, adding a challenge for additional efforts to clarify diagnostic criteria of CD, especially in developing countries.

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# Estimation of Platelet Count on the Basis of Red cell: Platelet Ratio

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## Abstract

***Background:*** Modern haematology analyzers are able to produce platelet counts with great precision and accuracy. However, in certain cases these analyzers produce erroneous platelet results.Therefore, the estimation of platelet count from blood smears should be systematic each time the automated count is erroneous. However, in comparison with the procedure for an automated count, the examination of a blood smear using a counting Neubauer chamber is a labor-intensive and therefore relatively expensive investigation.

***Objective:*** Verification of the reliability of the estimation technique of platelet count on the basis of red cell: platelet ratio.

***Material and Methods:*** In the period between January 2006 and March 2006 one hundred platelet counts were executed in the National Center for Haematological Diseases by two laboratory methods: an automated count using an impedance cell counter and then a manual method by reviewing microscopic blood smears. The number of platelets per 1000 erythrocytes was multiplied by the automated RBC ($x10^6$ cells/µl) to give an approximate manual count ($x10^3$ cells/µl). Two-paired t-test was used for comparison of the two methods.

***Results:*** Platelet count using the manual method was as follow: the range was 100-499$x10^3$/µl, the mean count was 263.11±104.07 $x10^3$/µl, and the median was 247.5 $x10^3$/µl. using the automated method, platelet count ranged between 95-484 $x10^3$/µl, the mean was 258.43 $x10^3$/µl, and the median was 242.5 $x10^3$/µl. There was no significant difference in results of platelet count using both methods ($P<0.05$). Regression analyses gave the following equation by comparing the automated (y) to the manual method (x): $y=0.9893x - 1.8621$ ($r= 0.966$). The paired t-test showed no significant difference between the two methods ($p<0.05$). The ICC was equal to 0.988. The plot of the differences between the automated and manual values against their means showed that the difference mean was 2.116 with a standard deviation SD= 40.215. It was noticed that 93% of the differences were within the agreement limits (mean±2SD).

***Conclusion:*** Red blood cell:platelet ratio method requires only an accurate RBC count performed on a calibrated hematology analyzer to calculate platelet count. This method is precise, simple, and consumes less time than using a counting chamber, and therefore, potentially should supersede ordinary manual counting.


***Key words:*** red cell: platelet ratio, platelet count

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## Introduction

The estimation of platelet count from blood smears must be systematic each time the automated count is erroneous because even the most expensive and most effective machine is not able to replace human judgement. With the development of sophisticated automated blood-cell analyzers, the proportion of blood-count samples that require a blood smear has steadily diminished and in many clinical settings is now 10 to 15 percent or less.Nevertheless, the blood smear remains a crucial diagnostic aid [(1)].Modern haematology analyzers are able to produce platelet counts with great precision and accuracy. However, in certain cases these analyzers produce erroneous platelet results, for example pseudothrombocytopenia [(2)], or pseudothrombocytosis or at least obvious overestimation of the real number of platelets as in patients with acute leukaemia. Because of their shape and size, haematology analyzers add several undefined particles to the platelet cluster. In some cases, this

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may even lead to the masking of a (possible life threatening) thrombocytopenia, and consequently the withholding of proper medication or other crucial supportive measures [3].

The International Council for Standardization in Haematology (ICSH) and the International Society of Laboratory Hematology (ISLH) recommend the counting of specifically labeled platelets relative to the RBCs with a fluorescence flow cytometer, together with an accurate RBC count determined with a semiautomated, single-channel aperture-impedance counter as a reference method for the enumeration of platelets [4].

***Aim of the study***

Verification of the reliability of the estimation technique of platelet count manually on the basis of red cell : platelet ratio.

***Material and Methods***

***Blood sample collection and processing:*** A total of 100 blood specimens were obtained from patients between January 2006 and March 2006 in the National Center for Haematological Diseases. Specific diseases or conditions of a patient were not considered for inclusion or exclusion in the study.

All venous blood specimens were collected into tubes containing ethylenediaminetetraacetic acid (EDTA) and then were stored at room temperature until analyzed within four hours.

Notation was made if clots were seen in the blood sample or if the amount of blood in the tube was grossly inadequate such that a high concentration of EDTA would be present; these samples were excluded from the study.

***Automated Method:*** After thorough mixing of each blood sample on an automated mixer for 10 min, a complete automated blood count was performed using an impedance cell counter (Coulter Counter), which was maintained and calibrated as recommended by the manufacturer.

***Manual Method:*** Thin air-dried blood smears made after thorough mixing of each sample were stained manually, using Leishman stain, and examined under light microscopy with a X100 oil-immersion lens. The slides were entirely scanned for platelet aggregates and/or macrothrombocytes and, if any, the samples were excluded from the study.

The red cell: platelet ratio was calculated in the monolayer zone of the smear as follows: The number of erythrocytes observed in a quarter of the oil-immersion field was multiplied by four instead of counting all the erythrocytes in the field. Then all the platelets in the same field were counted.

Other fields were examined in the same way until a minimum number of 1000 erythrocytes was reached. The number of platelets per 1000 erythrocytes was multiplied by the automated Red Blood Count (RBC) ($x10^6$cells/μl) to give an approximate manual count ($x10^3$cells/μl) [5].

***Statistical Method***

The mean, median, and range of platelet count using the two laboratory methods were calculated. Simple linear regression plot was used to compare the manual with the automated platelet counts.

Intra-class Correlation Coefficient (ICC) was calculated in order to identify the degree of correspondence and the agreement between the two methods. The ICC value is measured on a scale of 0 to 1, good reliability was assumed as an ICC>0.75. A paired t-test was performed, a statistically significant difference in platelet level was set at a level of $p<0.05$.

***Results***

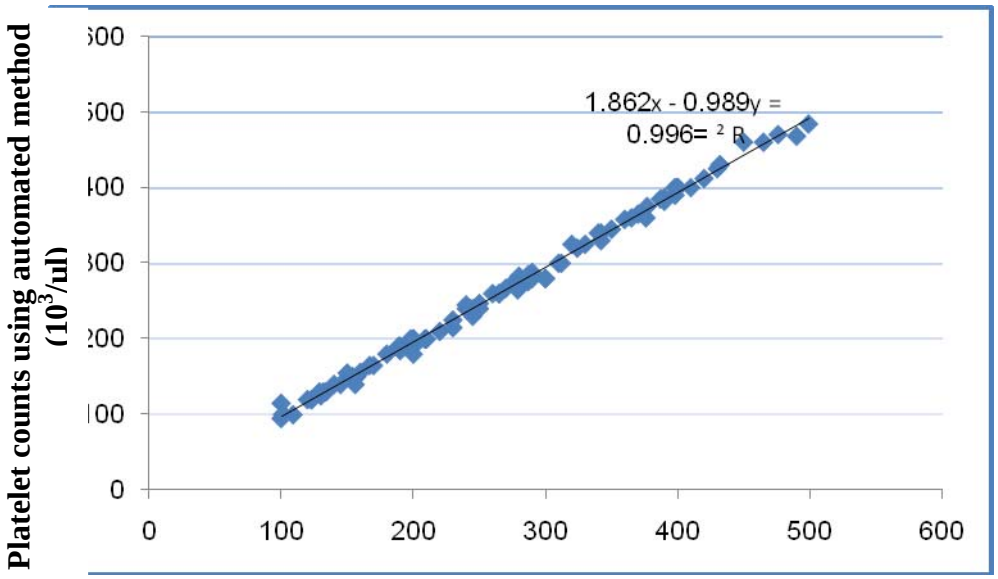
Results of platelet count using the manual method were as follows: the range was between 100-499x$10^3$/µl, the mean platelet count was 263.11±104.07 x$10^3$/µl, and the median was 247.5 x$10^3$/µl. By using the automated method, platelet count ranged between 95-484 x$10^3$/µl, the mean was 258.43 ±103.13 x$10^3$/µl, and the median was 242.5 x$10^3$/µl (table 1). The report of evaluation with the two laboratory methods gave the following equation by comparing the automated (y) to the manual method (x): y=0.9893x - 1.8621 (r= 0.966) (Figure 1).

The paired t-test showed no significant difference between the two methods ($p<0.05$). The ICC was equal to 0.988.

The plot of the differences between the automated and manual values against their means showed that the difference mean was 2.116 with a standard deviation SD= 40.215 (Figure 2). It was noticed that 93% of the differences were within the agreement limits (mean±2SD).

**Table 1: Results of platelet count using manual and automated methods**

| | Manual platelet count method (x$10^3$/µl) | Automated platelet count method (x$10^3$/µl) | P-value |
|---|---|---|---|
| Range | 100-499 | 95-484 | <0.05 |
| Mean ±SD | 263.11±104.07 | 258.43±103.13 | |
| Median | 247.5 | 242.5 | |




**Platelet counts using manual method ($10^3$/µl)**

**Figure 1: The regression analyses for the entire data set using manual and automated methods**

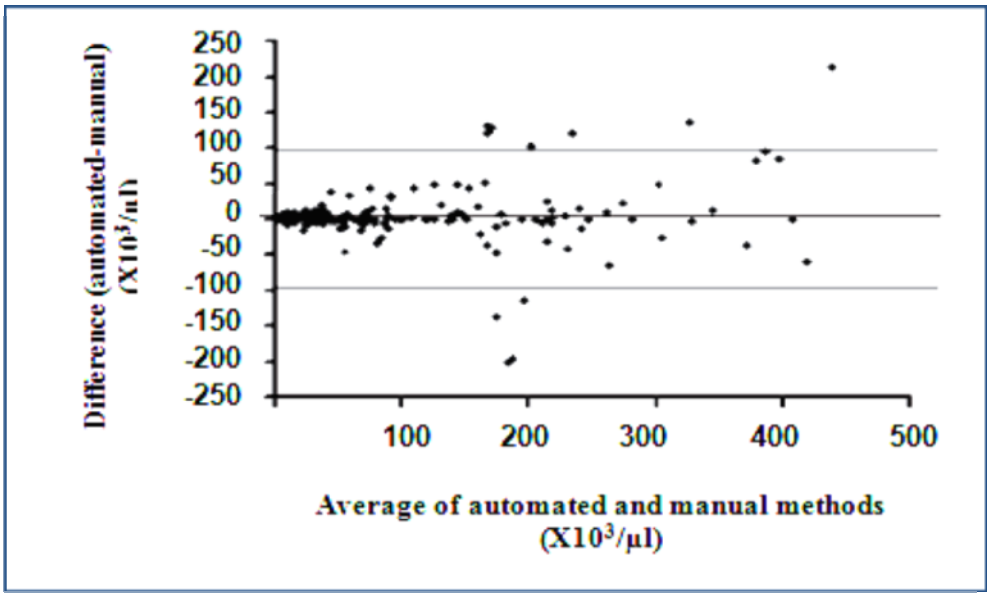



**Figure 2: Difference versus mean plots for automated and manual platelet counts. The middle solid line is the mean of the difference; the outer solid lines are the upper and lower limits of agreement (mean±2SD)**

***Discussion***

Even in the age of molecular analysis, the blood smear remains an important diagnostic tool. Physicians should request a blood smear when there are clinical indications for it. If error is to be avoided, sophisticated modern investigations of hematologic disorders should be interpreted in the light of peripheral-blood features as well as the clinical context. In comparison with the procedure for an automated count, the examination of a blood smear is a labor-intensive and therefore relatively expensive investigation. A request for a blood smear is usually the result of an abnormality in the complete blood count or a response to "flags" produced by an automated instrument [1].

Obtaining an accurate platelet count by using an automated hematology analyzer may be complicated by the presence of particles of similar size and/or light scatter properties (red cell fragments, microcytic red cells, apoptotic white blood cell fragments) and by giant platelets and platelet clumps [6,7]. Falsely low platelet counts may be the result of small clots, platelet clumping, platelet satellitism, or abnormally large platelets. Underlying causes that may be revealed by the blood smear include the May–Hegglin anomaly,

microangiopathic thrombopathies, and leukemias and lymphomas. High platelet counts should be confirmed microscopically with a blood smear; falsely high counts may be the result of other particles (red-cell fragments, fragments of leukemic cells, or fungi) being counted as platelets [8,9,10].

Examination of the blood smear is also important in patients with thrombocytosis to look for evidence of a myeloproliferative disorder, such as giant platelets, or an increase in the basophil count; the latter is not reliably detected by automated counters. A sudden, unexpected improvement in the platelet count also should be confirmed by blood-smear examination, since such an improvement may be factitious [9].

Until recently, the only reference method for platelet counting was the manual phase contrast microscope chamber counts (11) in which platelets are counted manually with a haemocytometer, such as Neubauer chamber. This is laborious, time-consuming and above all, an imprecise technique. The interoperater coefficient variant of this method can be up to 25%. However, it is still most widely used reference method [12].

Even if the manual platelet numeration, using a counting chamber, remains the technique of reference, it consumes more time and, to be more precise, requires a phase-contrast microscope, which is not always available in routine laboratories [13]. That is why the proposed method is better, since it is faster, taking only five minutes on average per patient, while demonstrating good precision.

Some authors recommend calculating the average number of platelets counted in 10 immersion fields; the adequate values are included between 8 to 20 platelets per field [14,15]. The average number of platelets is then multiplied by a factor of 20,000 for wedge preparations or 15,000 for monolayer preparations in order to obtain and estimate the platelet count, but this method is approximative and does not give the real number of platelets.

Comparing automated and manual, using red cell:platelet ratio method, platelets counting techniques showed that there was no significant difference ($P< 0.05$) between the mean, median, and range of platelet counts using these two methods.

The ICC was calculated in order to identify the reliability of the manual technique in comparison to the automated method [16]. The ICC value is measured on a scale of 0 to 1, and good reliability was generally assumed as an ICC > 0.75 [17]. In this study, the ICC was equal to 0.988, which is widely greater than this limit. In addition, 93% of the differences between automated and manual counting methods were within the agreement limits (mean±2SD).

Red blood cell: platelet ratio method requires only an accurate RBC count performed on a calibrated hematology analyzer to calculate platelet count. This method is precise, simple, and consumes less time than using a counting chamber, and therefore, potentially should supersede ordinary manual counting.

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# Measles outbreak in AL-kadhimiyia, Iraq, 2008-2009 and its common complications

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## *Abstract*

***Background:*** Worldwide efforts for measles elimination are made possible due to the availability of highly effective measles vaccine. In spite of that, there is high percentage of unvaccinated children in our country-making outbreak of measles easy and highly occurred.

***Objective:*** To identify the outbreak of measles in AL-Kadhimiya-Baghdad and its common complications with fatality causes and rate.

***Patients and methods:*** Cross-sectional study was conducted during the period between 20 th December 2008 to 30 th April 2009 on 494 patients with measles attending AL-kadhimiya Teaching Hospital ,AL-kadhimiya Hospital for pediatrics and two Primary Health Center ( Al shaheed Basher Al jasaery Primary health center in AL-shaula city and Al Noor Primary health center in AL-Jawaden city )and they were divided into four group according to their age which were (below 1year ),(1-4yr ),(5-9yr)and above 10 years ,regarding immunization status was assessed by examining the immunization card or parental enquiry on this regard.

***Result:*** Male are nearly equal to female (49.80 %)and (50.20 %) respectively. Of 494 reported cases 97 (19.64 %) were under one year of age and 287(58.97 % )were 1-4 years old and this mean that more than two-third 384 (78.61 % ) of patient were under 4 years ,small group (10.53 %) were vaccinated against measles .Complications were Pneumonia, Diarrhea and Vomiting, Croup and Encephalitis ,in (83.85 % ) ,(11.46 %) , (2.60 % ) , (2.09 % ) respectively. Mortality rate was (2.43%) which occured most commonly in age group below 5 years (91.7 %) and slightly more in male (58.4 %) than female (41.6 %) causes of deaths were pneumonia (83.4 %) and encephalitis (16.6 %).

***Conclusion:*** This outbreak of measles demonstrates the increased susceptibility of unvaccinated children who are below 5 years old .Pneumonia, Diarrhea and Vomiting, Croup and encephalitis are complications of measles and higher mortality rate occur in male sex and younger age group, (below5 years old).

***Key words:*** measles, vaccine, outbreak, complications, children.

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## *Introduction*

Measles is a communicable viral illness [(1)]. Caused by a virus, **paramyxovirus** of the genus ***Morbillivirus***. Symptoms include **fever**, **cough**, **runny nose**, **red eyes** and a generalized, **maculopapular**, **erythematous** rash. Measles is spread through respiration (contact with **fluids** from an **infected** person's nose and mouth, either directly or through **aerosol** transmission), and is highly contagious—90% of people without **immunity** sharing a house with an infected person [(2)].endemic form [(3)].Globally about 40 million cases of measles occur every year out of which 777000 death occur due to measles [(4)].

Its incidence in childhood varies from 58 %in epidemic to 10-15 %in

Complications with measles are relatively common, ranging from relatively mild and less serious diarrhea, to pneumonia and encephalitis (sub acute sclerosing panencephalitis), corneal ulceration leading to corneal scarring [(5)]. The fatality rate from measles for otherwise healthy people in developed countries is 3 deaths per thousand cases. In underdeveloped nations with high rates of malnutrition and poor healthcare,

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fatality rates have been as high as 28% [6]. In immunocompromised patients, the fatality rate is approximately 30 percent [7].According to the World Health Organization (WHO), measles is a leading cause of vaccine-preventable childhood mortality. Worldwide, the fatality rate has been significantly reduced by partners in the Measles Initiative: the American Red Cross, the United States Centers for Disease Control and Prevention (CDC), the United Nations Foundation, UNICEF and the World Health Organization (WHO). Globally, measles deaths are down 60 percent, from an estimated 873,000 deaths in 1999 to 345,000 in 2005. Africa has seen the most success, with annual measles deaths falling by 75 percent in just 5 years, from an estimated 506,000 to 126,000 [8]. By using vaccine we can reduce the morbidity and mortality .although it had been shown that outbreak of the disease occur from time to time, In 2007, a large measles outbreak in Japan caused a number of Universities and other institutions to close in an attempt to contain the disease [9,10]. In developing countries where measles is highly endemic. The WHO recommend that two doses of vaccine be given at six months and at nine months of age [11]. Some countries like Iran, Syria and U.A.E have started second dose of measles at 15 months of age with high coverage of 90 %or more [12]. Low vaccines coverage rate with low vaccine efficacy leads to higher rate of complication which causes financial burden [13]. Therefore, children hospitalized with complications of measles can provide the magnitude of problem and its future preventive strategies.

***Aim of the study***

To identify the outbreak of measles in AL-Kadhimiya-Baghdad and its common complications with mortality causes and rate.

***Patients and methods***

Cross sectional study was conducted at children department of AL-kadhimiya Teaching Hospital ,AL-kadhimiya Hospital for pediatrics and two Primary Health Center in Baghdad-AL-kadhimiya(Al shaheed Basher Al jasaery Primary health center in AL-shaula city and Al noor Primary health center in AL-Jawaden city) from 20th of December 2008 to 30th of April 2009 and involve four hundred ninety four patients , the youngest one was two months old and the oldest one was sixteen years old .All children diagnosed as a case of measles on clinical ground, according to appearance of maculopapular rash, fever of 38 c° or more with cough ,coryza and conjunctivitis and appearance of kopliks spots in some of them .pneumonia was defined according to WHO criteria of respiratory rate [14] , and presence of pulmonary infiltrate on chest radiography .Central nervous system was considered to be involved if there was lethargy ,irritability, headache, fits, disorientation or other neurological deficit .The detailed history, physical examination and measles complications including diarrhea, pneumonia, Croup, and encephalitis were filled in case report form. Immunization status was assessed by examining the immunization card or parental enquiry on this regard. Clinical outcome was compared between male and female as well as different age groups. The patients were divided into four groups according to age. Statistical analysis was done by using chi square and p value of less than 0.05 was designated as statistically significant.

***Results***

During the period of the study (from 20th of December 2008 to 30th of April 2009), the total number of

children diagnosed as measles were 494 and the findings were.

Total number of male was 246 (49.80 %) while female number was 248 (50.20 %).

Regarding age 97 (19.64 % ) patients were less than one year and 287 (58.97 %) patients were between 1-4years Which mean that 384 (78.61 % )patients were under 4 years as shown in( Table 1) , distribution of patients regarding age group and sex Was shown in( Figure 1) , the ratio of unvaccinated to vaccinated patients was 9.5:1.

**Table 1: Age distribution of patients.**

| Age categories | number | % |
|---|---|---|
| < 1 yr | 97 | 19.64 |
| 1-4 yr | 287 | 58.97 |
| 5-9 yr | 94 | 19.03 |
| > 10yr | 16 | 2.36 |
| Total | 494 | 100 |

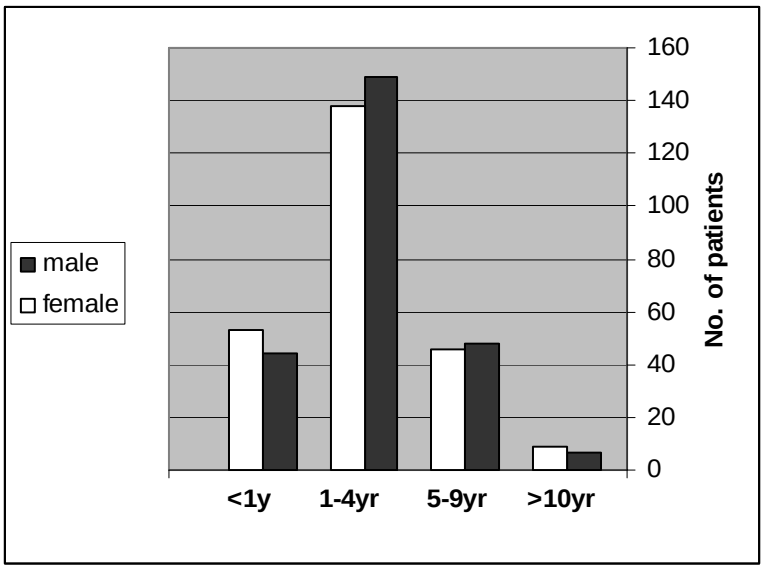


**Figure 1:distribution of patients according to age groups and sex.**

Vaccination against measles was present in 52 (10.53 %) patients, 65.38 % of them were male and 34.62 % of them were female.

Male to female ratio was 1.9:1 as it is shown in (Figure 2).

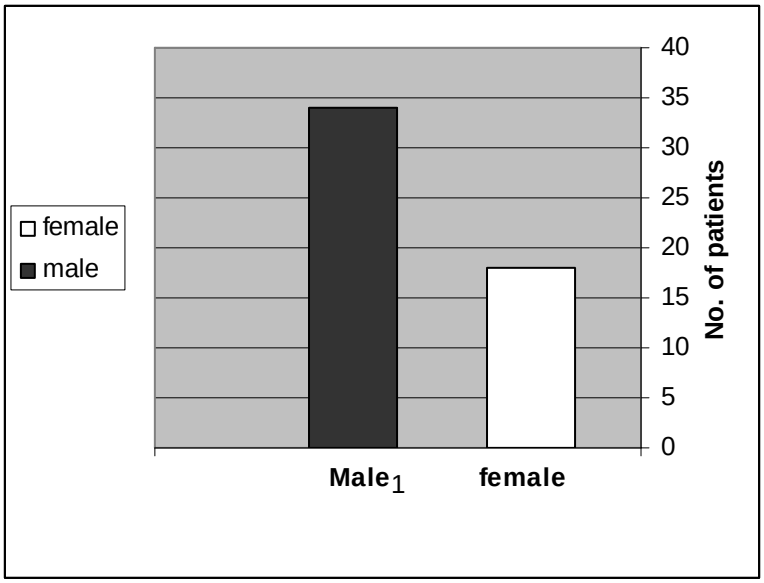


**Figure 2:vaccination status of patients in the sample**.

The most common complications of measles were pneumonia 161 (83.85 %), diarrhea and vomiting 22 (11.46 %), croup 5 (2.60 %), and encephalitis 4 (2.09 %) as it is shown in (Table 2).

**Table 2: Complications and outcome in measles patients.**

| Complications | NO. | % | Improvement | | Death | |
|---|---|---|---|---|---|---|
| | | | NO | % | NO. | % |
| Pneumonia | 161 | 83.85 | 151 | 83.9 | 10 | 83.4 |
| Diarrhea and vomiting | 22 | 11.46 | 22 | 12.2 | 0 | 0 |
| Croup | 5 | 2.60 | 5 | 2.8 | 0 | 0 |
| Encephalitis | 4 | 2.09 | 2 | 1.1 | 2 | 16.6 |
| Total | 192 | 100 | 180 | 100 | 12 | 100 |

Total number of death was 12 and mortality rate was 2.43 %, pneumonia was the leading cause of death (83.4 % of death), followed by encephalitis (16.6 % of death) as it is shown in (Table 2). The mortality was more in male 7 (58.4 % )as well as below one year 7 (58.4 % ) while in age group 1 -4 years was 4 (33.4 % ) which mean that 91.8 % of death occur bellow 5 years as shown in( Table 3 )

**Table 3:Demographic profile and morality (n=12).**

| Parameter | | No. of Death | % |
|---|---|---|---|
| SEX | Male | 7 | 58.4 |
| | Female | 5 | 41.6 |
| | Total | 12 | 100 |
| Age group (years ) | <1yr | 7 | 58.4 |
| | 1-4yrs | 4 | 33.4 |
| | 5-9yrs | 1 | 8.2 |
| | Above10yrs | 0 | 0 |
| | Total | 12 | 100 |

***Discussion***

The study demonstrate an outbreak of measles in Baghdad from the end of 2008 and beginning of 2009 which is similar to the outbreak occurring in different countries like Saudi Arabia in 2007 [15], Pakistan [16] , and Vietnam with Ho Noi [17]Also the study show high percentage of the disease in age group below 5 years which is similar to reported in developing countries [18] whereas.

In contrast with the data from developed countries that the incidence is higher in second decade [19] , because the disease is still endemic in developing countries .In this study there are no difference in the incidence of the disease between males and females which is differ from that obtain from recent study

In Saudi Arabia which show high percentage of the disease in males than females [15] ,The result in this study explained by equal affection of Male and female in most of the viral infections

In this study 10.53 % of patients were vaccinated against measles which is similar to other studies from Islamabad [16] , Rawalpindi [20] ,And Lahore [21] , this could be due to unavailability of vaccine at times where mothers visit the primary health center in addition to poor storage in previous months .

Pneumonia in our study is the commonest complication of measles which is similar to the reported from South east Asia and Europe [22 ,23] While diarrhea and vomiting is a second common complication in this study in contrast to Indian studies where diarrhea and vomiting was the commonest complications [24], this may be due to occurrence of the disease in time when there is high incidence of respiratory tract infection.

Croup was uncommon complication of measles in this study which is differ from study reported from Islamabad [16] .Encephalitis also was uncommon complication in our study which is differ from study reported from Saudi Arabia [15] .The case fatality rate in this study was lower than Islamabad [16] , Saudi Arabia [15] ,and Pakistan [16] ,this due to early detection of the Disease and early diagnoses and perfect managements of its complications.

Now there is need to improve measles vaccination coverage at national level and indicates the urgency to improve vaccinations coverage to protect unvaccinated children and introduce two doses of

measles vaccine schedule to boost the Immunity of vaccinated children. .

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# In vitro treatment of Ham's F-10 medium supplemented with vitamin C and E on human semen characteristic in asthenozoospermic men

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## *Abstract*

***Background:*** The levels of reactive oxygen species are normally limited by antioxidant defense mechanisms such as vitamin C and E that are present within seminal plasma and sperm plasma membrane. The Supplementing infertile males with antioxidant vitamin C and E is suggested as a potential treatment for idiopathic male infertility.

***Objective:*** This study was designed to determine the effect of Ham's F-10 preparation medium supplemented with antioxidant vitamin C or E on semen samples prepared by conventional layering technique**.**

***Methods:*** Liquefied semen (1ml) was layered beneath Ham's F-10 (1ml) enriched with 0.75 mg/ml vitamin C or E after in vitro sperm processing. However, semen samples were collected from a total of 60 asthenozoospermic men by masturbation after 3-5 days abstinence and allowed to liquefy at 37ºC in 5% $CO_2$ for 30 minutes and evaluated according to standard world health organization (WHO) criteria before and after in vitro sperm activation. The semen samples were divided into three groups, one group considered as a control group which had no antioxidant added, and the other two groups were prepared in the presence of antioxidant treatment (either vitamin C or vitamin E).

***Results:*** The supplementation of sperm preparation medium with vitamin C or vitamin E significantly (P<0.001) improved and augmented the seminal parameters including sperm concentration, sperm motility, progressive sperm motility, and normal sperm morphology when compared to that of the control group.

***Conclusion:*** It was concluded that supplementation of medium with antioxidant vitamin C or E actually improve sperm quality, but the better improvement appeared to be with vitamin C.


***Key words:*** Antioxidant, vitamin C, sperm preparation technique, asthenozoospermia

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## *Introduction*

Infertility affects 15% of couples and is cause of infertility in 30% of those couples is associated with aberrations found in male partner termed male infertility. Many cases of male infertility were previously considered idiopathic but are now being attributed to oxidative sperm damage resulting from the pathologically increased levels of reactive oxygen species [(1)].

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In contrast, high levels of ROS are harmful and lead to lipid peroxidation However, ROS can be produced by immature spermatozoa and leukocytes [(2)]. In normal sperm physiology, low levels of ROS are beneficial to stimulate sperm capacitation, enhance zona pellucida binding and promote acrosome reaction [(3).]

of sperm plasma membrane and DNA fragmentation [(4)]. Increased lipid peroxidation is associated with impaired sperm motility and diminished capacity for sperm-oocyte fusion [(5)]. One study found that men with high levels of ROS were 7 times less likely to achieve a pregnancy than men with low levels [(6)].

The levels of ROS are normally limited by antioxidant defense mechanisms such as vitamin C and E that are present within the seminal plasma and sperm plasma membrane [7]. However, the supplementing infertile males with antioxidant vitamin C and E suggested as a potential treatment for idiopathic male infertility [8] .Vitamin E is a chain-breaking antioxidant because of its ability to terminate a free radical chain reaction and play an important role in pathogenesis of male infertility and protecting against oxidative attack both in vivo and in vitro [9]. Specifically, vitamin E inhibits peroxidation of polyunsaturated fatty acids (PUFA) which is especially important in spermatozoa due to their high PUFA content [10]. While, Vitamin C actually secreted from seminal vesicles during ejaculation and protect human sperm from endogenous oxidative DNA damage [11]. It is acts as a scavenger of a wide range of ROS which explains its ability to successfully counteract the effects of DNA damage and ROS production [12]. It has previously been shown to be the major antioxidant in seminal plasma of fertile men contributing up to 65% of the total chain-breaking antioxidant capacity [13]. In addition, concentration of Vitamin C in seminal plasma is 10 times greater than the concentration found in blood plasma [14]. The semen quality is an important factor in determining suitability of the couples in achieving pregnancy. The fertilization occurs despite an abnormal semen analysis, or it fails to occur when analysis values are normal. The semen analysis cannot ascertain the functional capacity of sperm and frequently fails to predict the outcome of male infertility [15].

## ***Subjects, Materials and Methods***

### *Collection of semen samples:*

Semen samples were obtained from a total of 60 asthenozoospermic men and attendance of IVF Institute of Embryo Research and Infertility Treatment/Al-Nahrain University between March and May 2007. The mean of age ± S.E.M for infertile subjects was 30.05 ± 4.87 years. The ejaculates were collected by masturbation after 3-5 days abstinence and allow liquefying at 37ºC in 5% $CO_2$ for 30 minutes. The liquefied semen is carefully mixed for few seconds, and then seminal fluid analysis parameters including sperm concentration, sperm motility, progressive sperm motility, and normal sperm morphology was examined before and after in vitro sperm treatment. However, WHO (1999) criteria for normal semen values were applied.

### *Sperm preparation technique and in vitro antioxidant treatment:*

Sperm processing prepared using conventional layering technique by mixing 1ml of the liquefied semen was layered beneath 1ml culture medium (Ham's F-10) after finished the routine semen analysis and confirmed the results before in vitro sperm preparation which is regarded as a control. The supernatant was removed and divided into two tubes, 0.5 ml for each tube. One tube was mixed with 0.75 mg/ml antioxidant vitamin C (Sigma Aldrich Co. Ltd, Poole, UK) and another tube was mixed with 0.75 mg/ml antioxidant vitamin E (Trolox, Sigma Aldrich, UK).

### ***Statistical analysis***

Statistical analysis was performed with the SPSS version 12.00. The data analysis was done using paired sample t-test to assess the statistical differences in the results . Mean and standard error of mean (S.E.M) obtained from crude data to

compare between Pre-and Post-activation for semen parameters. P-value < 0.05 was used as a level of statistically significance.

***Results***

The results of the present study showed that semen samples supplemented with antioxidant vitamin C and E improved the results for semen parameter as compared with control group used Ham's F-10 only (Tables 1-3; respectively), but vitamin C gives the best seminal parameter including sperm concentration, sperm motility, progressive sperm motility, and normal sperm morphology as compared with vitamin E. However, it was noticed a significant (P<0.001) differences in sperm function and seminal fluid parameters were assessed post in vitro sperm activation as compared with results of pre-activation of human spermatozoa in all infertile patients.

The markedly reduction in sperm concentration was observed following in vitro sperm preparation using Ham's F-10 medium with and without vitamin C and vitamin E supplementation. The handling yielded significantly lower sperm concentration as compared with pre-activation. But, these parameters of spermatozoa significantly increased not only by addition of antioxidant within culture medium for sperm preparation as compared with unprepared semen, but also in absence of antioxidant within Ham's F-10 medium. It was recognized that Ham's F-10 medium contains protein, inorganic ions as well as carbohydrates, and most necessary requirement for improvement sperm functions that cause an increase in migration of normal mature active sperm to upper layer of culture medium.

**Table 1: Effect of Ham's F-10 medium on semen parameters pre and post in vitro sperm activation for infertile patients* with asthenozoospermia**

| Parameters | Conventional layering technique | |
|---|---|---|
| | Pre-activation | Post-activation |
| Sperm Concentration ($\times 10^6$ sperm/ml) | 37.33±5.83 | 22.01±3.54 † |
| Sperm Motility (%) | 52.56±2.63 | 81.18±1.19 † |
| Progressive sperm Motility (%) | 31.45±2.41 | 54.43±2.25 † |
| Normal Sperm morphology (%) | 53.66±2.57 | 87.33±1.07 † |

Values are Mean ± S.E.M

†: Means a highly significant (P<0.001) difference from pre-activation

*No. of infertile patients=20

**Table 2: Effect of Ham's F-10 medium supplemented with vitamin E on semen parameters pre and post in vitro sperm activation for infertile patients* with asthenozoospermia**

| Parameters | Conventional layering technique | |
|---|---|---|
| | Pre-activation | Post-activation |
| Sperm Concentration ($\times 10^6$ sperm/ml) | 40.35±6.31 | 22.75±3.65 † |
| Sperm Motility (%) | 54.00±2.55 | 86.60±2.07 † |
| Progressive sperm Motility (%) | 31.90±1.66 | 54.85±1.43 † |
| Normal Sperm morphology (%) | 44.50±3.20 | 80.25±2.09 † |

Values are Mean ± S.E.M
†: Means a highly significance (P<0.001) different from pre-activation
*No. of infertile patients=20

**Table 3: Effect of Ham's F-10 medium supplemented with vitamin C on semen parameters pre and post in vitro sperm activation for infertile patients* with asthenozoospermia**

| Parameters | Conventional layering technique | |
|---|---|---|
| | Pre-activation | Post-activation |
| Sperm Concentration ($\times 10^6$ sperm/ml) | 48.12±4.52 | 25.63±1.41 † |
| Sperm Motility (%) | 54.12±3.63 | 88.34±1.29 † |
| Progressive sperm Motility (%) | 38.72±1.45 | 67.90±1.38 † |
| Normal Sperm morphology (%) | 42.41±2.50 | 83.40±1.81 † |

Values are Mean ± S.E.M
†: Means a highly significance (P<0.001) different from pre-activation
*No. of infertile patients=20

***Discussion***

The results of the present study are in a good agreement with results obtained by Zavos et al. [16] who reported that layering technique significantly has higher percentage of recovery of motile spermatozoa, progressive motile spermatozoa, higher DNA integrity, and numbers of pregnancies than other sperm preparation method. However, it was assessed that sperm concentration, motility, morphology, viability, membrane integrity, acrosomal status, ROS formation, and chromatin

maturity results could be evaluated with usefulness of sperm preparation techniques [17]. In addition, Sills *et al.* [18] mentioned that the selection of sperm preparation methods depend on the quality of the ejaculates. The ejaculates with ROS production by spermatozoa and leukocytes should not be separated by centrifugation method due to severely spermatozoa damage.

It was noticed that the problem caused by ROS can resolve by performed directly from liquefied semen underneath an overlay of culture medium and aspirate directly from the interface region with total number of spermatozoa recovered [19]. However, Aitken and Clarkson [20] suggested that poor IUI outcome may be related to improper preparation techniques with release of harmful ROS as well as the separation of motile and active sperm from the rest of the semen can significantly improve pregnancy rates [21]. Furthermore, it was reported that common laboratory factors like centrifugation, washing, temperature fluctuation, and processing delay harmfully affect semen quality both positively and negatively due to direct influence of laboratory interventions on the cytoskeletal assemblies of sperm [22].

Many studies focus on isolating the population of infertile men who are most likely to benefit from vitamin E supplementation. Potential populations could include men with increased ROS levels, increased DNA fragmentation or asthenozoospermia. The supplementation of sperm preparation medium with vitamins C and E may reduce free radical production and decrease ROS induced DNA damage in patients with poor sperm quality. This in turn may provide a greater chance of successful fertilization, as there is an inverse correlation between percentage of sperm with DNA fragmentation and fertilization rates in vitro with both IVF [23] and ICSI [24]. There is also possibility that oral administration of ascorbate may facilitate a reduction in induced DNA damage, although this is an area that requires further investigation before any firm conclusions can be drawn. As a result, vitamin E enhanced has the potential to help numerous couples that suffer from male infertility.

The dosage and duration of vitamin E supplementation also needs to be explored and optimized. While, vitamin C act as a scavenger of a wide range of ROS [25], which explains its ability to successfully counteract the effects of free radicals both in terms of induced DNA damage and ROS production. It has previously been shown to be the major antioxidant in seminal plasma of fertile men, contributing up to 65% of the total chain breaking antioxidant capacity [26]. The concentration of this antioxidant in seminal plasma is 10 times greater than the concentration found in blood plasma. The study by Moilanen and Hovatta suggested that vitamin E is less possible to have a protective role given that its seminal plasma concentrations were below the beneficial levels. The study found that this vitamin concentration in the spermatozoal membrane rather than in the seminal plasma is positively correlated with improved sperm parameters [27].

The combination of vitamins could substantially reduce ROS levels and impair its normal physiologic function. The current study outlines the beneficial effects of antioxidant supplementation on induced DNA damage. Previous studies have shown that vitamin E affords sperm cells some protection from oxidative attack both in vivo [28] and in vitro [29] studies. The oral administration of vitamin E has also been shown to lead

to a significant improvement in the in vitro function of human sperm as assessed using the zona-binding test and has been suggested as a treatment for ROS-associated male infertility [30].

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# Shigellae – associated diarrhoea in children in Baghdad – Iraq

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## *Abstract*

***Background:*** *shigella* spp. reported to be the second commonest bacterial agent responsible for childhood diarrhea after *E.coli*.Currently, isolation of the bacterium and confirmation of the diagnosis by bacteriological and biochemical methods remains the "gold standard".

***Objective:*** To determine the prevalence of *shigella* spp. among children below 3 years with acute diarrhoea and susceptibility of the isolates to commonly used antimicrobials.

***Methods:*** This study was carried out in the outpatient's clinic of Children Central Teaching Hospital in Baghdad from May 2007 – April 2008. One hundred and fifty children below 3 years with acute dirrhea were the source of stool specimens to detect *shigella* spp.All isolates were diagnosed according to bacteriological and biochemical standard methods. Available antimicrobial were used to determine the susceptibility of isolates to antibiotics

***Results:*** *Shigella flexneri* type 2 was the predominate serotype out of 9 isolates.The prevalence of *shigellae* isolates was significantly higher in children older than one year .All patients were on artificial feeding, 78% were using untreated water for drinking. All isolates were sensitive to ceftriaxone , ciprofloxacin , nalidixic acid, norfloxacin and gentamicin. Drug resistant to 3 or more drugs was found in 56% of the isolates.

***Conclusion:*** *Shigella flexneri* type 2 was the predominate serotype and most isolates were resistant to trimethoprim -sulphamethoxazole (89%).

***Keywords:*** *Shigellae* , Diarrhoea, Antibiotic, Children, Iraq .

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## *Introduction*

Diarrhoeal diseases are one of the main causes of death especially among young children which leads to at least five million deaths each year [1]. Endemic bacillary dysentery accounts for about 10% of the disease in children aged less than five years [2, 3].Among the different pathogens responsible for diarrhoea , *shigella* spp. Play an important role in causing inflammatory diarrhoea and dysentery [4, 5] , with a significant morbidity and mortality in developing countries [6] .

Shigellosis is an acute diarrhoeal disease caused by *shigella* spp.

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It has caused and continued to be responsible for morbidity and / or mortality in high risk populations such as children under 5 years of age [7]. Spread of shigellosis is through contaminated water, poor sanitation and overcrowded areas [8]. Over the past decades, *shigella* spp. has become progressively resistant to the most widely - used and inexpensive antimicrobials [4, 9, 10, 11]. Moreover changes in the virulence of *shigella* spp. make it difficult to formulate a drug of choice for the treatment of shigellosis [12].

## *Patients and methods*

### *Patients:*

This study was carried out in the outpatient Clinic of Children Central Teaching Hospital in Baghdad during the period from May 2007 - April 2008. One hundred and fifty children with acute diarrhoea age rang from few

days to 3 years.( Most children (84%) were less than one ).

***Specimens collection:***

Stool specimens were collected in sterile wide mouth containers.

***Methodology:***

All isolates were diagnosed according to well known microbiological methods [13].For optimal isolation, three different media and an enrichment medium were used. The samples were inoculated directly on MacConkey agar, xylose – lysine deoxycholate agar and *Salmonella-Shigella* agar.Enrichment was done in Na – Tetra thionat broth and incubated at 37C overnight. Biochemical identification of *shigella* spp. has been performed according to standard methods[14]. Confirmation of diagnosis was through slide agglutination test using commercially available antisera (Wellcome Diagnosis, UK)[14].

***Antimicrobial susceptibility tests:***

*Shigella* isolates were examined for their susceptibilities to ampicillin , chloramphenicol,ceftriaxone,ciprofloxacin,gentamicin,kanamycin,nalidixic acid, norfloxacin and co - trimoxazole by the standard disc - diffusion method[15].

***Results***

Table ( 1 ) shows that *shigella flexneri* type 2 was the predominate serotype out of 9 isolates. Of children with diarrhoea , *shigellae* were isolated equally from both sexes (5 from 80 males and 4 from 70 females) . A total of 6 *shigella flexneri* and 3 *shigella sonnei* were isolated. Of the 6 strains of *shigella flexneri* 4 were *shigella flexneri* type 2 , 1 of *shigella flexneri* type 1 and 1 of *shigella flexneri* type 3.Out of 9 *shigella* strains , 5 ( 56% ) were isolated in summer , 1 (11%) in autumn , 2 (22%) in winter , 1 ( 11% ) in spring. Three *shigella* strains were isolated from 50 children with diarrhoea aged 6 months to one year , 6 strains out of 24 patients aged more than one year and none were isolated from 76 children less than 6 months old . All patients with *shigella* isolates (100%) were on artificial feeding, 7 (78%) were using untreated water for drinking,1 (11%) took Bactrim 2 days prior to stool collection and none travelled abroad in the last 30 days before stool collection . Blood and mucus were seen in the stool of 5 (56%) patients with diarrhoea . Clinical examination showed 6 (67%) with fever and 3 ( 33% ) with vomiting ,1 (11%) was diagnosed to have septicemia and 1(11% ) had generalized convulsion. Clinical finding are shown in Table - 1 . The drug susceptibility patterns of 9 isolates of *shigella* strains were determined . Resistant strains to ampicillin was found to be 78% , 67% to chloramphenicol , 67% to kanamycin ,56% to streptomycin and 89% to trimethoprim-sulphamethoxazole . All isolates were sensitive to ceftriaxone, ciprofloxacin, nalidixic acid, norfloxacin and gentamicin as compared to Table-2. Drug resistance to 3 or more drugs was shown by 56% of strains.

**Table 1 :Information about the children with diarrhoea and their *shigella* species isolates.**

| Patients number | Gender | Age | Month of Occurrence | Use of untreated drinking water | Stool with mucus | Stool with blood | Fever | Vomiting | Duratin diarhea in (days) | Freque-ncy | Species and Serotype of *Shigella* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | F | 11 mo. | May | No | + | + | - | - | 1 | 5 | *Sh. sonnei* |
| 2 | F | 30 mo. | June | Yes | - | - | - | - | 2 | 3 | *Sh .flexneri* type 2 |
| 3 | M | 27 mo. | July | Yes | - | - | + | - | 1 | 6 | *Sh. sonnei* |
| 4 | F | 18 mo | July | Yes | - | - | - | - | 1 | 8 | *Sh .flexneri* type 2 |
| 5 | F | 3 yrs. | August | No | + | + | + | - | 2 | 5 - 7 | *Sh. flexneri* type 2 |
| 6 | M | 6 mo. | September | Yes | - | - | + | - | 7 | 7 - 8 | *Sh.flexneri* type 1 |
| 7 | M | 6 mo. | November | Yes | + | + | + | + | 1 | 10 | *Sh. flexneri* type 2 |
| 8 | M | 13 mo. | December | Yes | + | + | + | + | 10 | 6 - 7 | *Sh. flexneri* type 3 |
| 9 | M | 32 mo. | March | Yes | + | + | + | + | 1 | 4 | *Sh .sonnei* |

**Table 2: Antimicrobial susceptibility patterns of *shigella* spp. isolates from outpatients presenting at Hospital in Baghdad - Iraq.**

| Antimicrobial agent | Resistant | | | Intermediate | | | Susceptible | | |
|---|---|---|---|---|---|---|---|---|---|
| | Zone size(mm) | No. | % | Zone size(mm) | No. | % | Zone size(mm) | No. | % |
| Ampicillin | ≤ 13 | 7 | 78 | 14 - 16 | 0 | 0.0 | ≥ 17 | 2 | 22 |
| Chloramphenicol | ≤ 12 | 6 | 67 | 13 - 17 | 1 | 11 | ≥ 18 | 2 | 22 |
| Ceftriaxone | ≤ 14 | 1 | 11 | 15 - 16 | 0 | 0.0 | ≥ 17 | 8 | 89 |
| Ciprofloxacin | ≤ 12 | 1 | 11 | 13 - 14 | 1 | 11 | ≥ 15 | 7 | 78 |
| Gentamicin | ≤ 15 | 2 | 22 | 16 - 19 | 2 | 22 | ≥ 20 | 5 | 56 |
| Kanamycin | ≤ 13 | 6 | 67 | 14-17 | 1 | 11 | ≥ 18 | 2 | 22 |
| Nalidixic acid | ≤ 14 | 2 | 22 | 15 - 18 | 1 | 11 | ≥ 19 | 6 | 67 |
| Norfloxacin | ≤ 13 | 3 | 33 | 14 - 17 | 0 | 0.0 | ≥ 18 | 6 | 67 |
| Streptomycin | ≤ 11 | 5 | 56 | 12 - 14 | 2 | 22 | ≥ 15 | 2 | 22 |
| Trimethoprim-Sulphamethoxazole | ≤ 10 | 8 | 89 | 11 - 15 | 0 | 0.0 | ≥ 16 | 1 | 11 |

In total 9 *shigella* strains were tested.

***Discussions***

In the present study *shigella flexneri* was found to be the most frequent isolate in children with *shigellae* associated diarrhea in our study, rate in Baghdad with type 2 being the predominating serotype. These findings conform with several studies [16,17,18], and other developing countries [19,20,21], but they are in contrast with studies in developed countries where *shigella sonnei* is dominant and *shigella flexneri* is the second most prevalent isolate [4,22,23]. In some countries, *shigellae* were isolated more often from children older than 2 years of age than from younger children [24,25,26]. In our experience *shigella* spp. were isolated only from children who were not breast - fed and significantly more frequently from children older than one year of age[27]. Human milk has been shown to protect against severe shigellosis in children up to 35 months of age [28]. Studies showed that all milk samples obtained from mothers contained antibodies to antigens encoded by the large virulence plasmid in strains of *shigella* [29] . In addition to this, children more than one year old are capable of moving around and come into more direct contact with other children and adults which expose them under certain circumstances, to infection with these organisms. Food and waterborne outbreak of shigellosis have been reported from different parts of the world [8, 30, 31]. Most (78%) of our patients with stools positive for *shigella* have used untreated drinking water. We found blood and mucus in the stools of 56% and fever in 67% of our patients. These clinical findings are in line with those reported by other investigators in under - developed countries[19,20]. Several investigators reported cases of generalized convulsions associated with shigellosis[16,32,33]. Of our patients, only one (11%) had generalized convulsions and we believe this to be the first time such a case has been reported from Iraq which conform by Daoud *et al.* [16],who studied 93 children with shigellosis and found 15% of the patients developed generalized convulsions. They reported than neither specific diagnostic procedures nor drug therapy were usually necessary due to benign and self – limiting nature of convulsions associated with shigellosis.

Antibiotics can be useful in the treatment of *shigella* – associated diarrhoea, however trimethoprim - sulphamethoxazole no longer to be considered the drug of choice in our hospital as the majority (89%) of the local isolates were resistant to this antibiotic . Several studies reported the same findings [16, 17].AL - Eissa *et al.* [17], from Saudi Arabia reported 74% of their *shigella* isolates were resistant to trimethoprim - sulphamethoxazole. All our isolates were sensitive to ceftriaxone, ciprofloxacin,nalidixic acid, norfloxacin and most of them to gentamicin , therefore they should be considered the drugs of choice for treatment of diarrhea due to *shigellae.* , however, susceptibility testing to antibiotics before administering the drug is recommended.

The present study shows that *shigellae* associated diarrhea in children is still a public health problem.

*Shigella flexneri* type 2 was the predominate serotype and most isolates were resistant to trimethoprim - sulphamethoxazole (89%), thus it no longer to be considered the drug of choice in the treatment of *shigellae* associated diarrhea in children below 3 years in our study.

Since all isolates were sensitive to ceftriaxone, ciprofloxacin, nalidixic

acid, so they can be considered as the drugs of choice.

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# Removal of Tattoo By 1064 and 532nm Q-switched Nd: YAG laser

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## *Abstract*

***Background:*** A tattoo is made of particles of pigment injected into the skin. Although the body attempts to remove them, the particles of tattoo are too large to be removed and the body responds by encapsulating the whole tattoo in a wall of collagen that traps it within the skin permanently.

***Objective:*** to evaluate Nd-YAG laser effects for removing tattoo.

***Materials & Methods****:* The study was done on 99 tattoo lesions (in both genders) on different parts of the body (hand, foot, face and chest), ages were between 21- 60 years, and most common age group was between 20-30 years. In this work, tattoo were divided according to its colors, into black, blue, green, and red (The red color always shares with other colors), and each subdivided to amateur and professional tattoos. Quality-switched (Q-switched) 1064 nm, Nd: YAG laser, pulse width of 10 nanosecond and repetition rate (R.R) 5 Hz with different fluencies (energy/area) was used. These parameters were used for black, green, and blue tattoos. To red color tattoo 532nm Nd: YAG Q-switched laser, 7 ns pulse duration, R.R 10Hz, fluencies from 7.3 - 10.3 Joule/cm$^2$ was used. The exposure time needed for treatment was from 2-5 min. according to the size of tattoo. Time interval between two sessions was from 3-4 weeks.

***Results****:* Black and blue color tattoo removed rather completely with faint shadow. Red color tattoo removed completely. For green color tattoo there was no responses. In this study the treatment with Q-switched Nd: YAG laser offers bloodless, low-risk, no permanent complication, no scarring, and no disfigurements in skin.

***Conclusion:*** Laser use is considered standard treatment for patient seeking tattoo removal.

***Keyword****:* Tattoo/ Q-switched Nd: YAG laser/ Fluencies.

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## *Introduction*

The origin of tattooing among humans is as old as humanity itself [(1)] .A tattoo is a permanent make or design made on the body when pigment is inserted into the dermal layer of the skin through ruptures in the skin's top layer [(9)]. The National Institute of Health recognizes five types of tattoo; amateur, professional, cosmetic, medical, and traumatic (natural).

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Amateur tattoo are usually done with commonly available inks or pigments introduced into the skin manually. Professional tattoo uses commercial grade inks applied with a "gun" or specially designed apparatus. The main reasons for removing tattoos are enhancement of self-esteem and social, work or family reasons [(30)]. There are medical complications may be seen in patients with tattoo like bacterial endocarditis, dermatofibrosarcoma protuberans,hepatitis, lichenoid, metal toxicity and unusual cutaneous lesions like Boeck's sarcoid, secondary syphilis, discoid lupus erythematosus and eruption due to mercury sensitivity. Many different kinds of removal methods have been used throughout the

centuries, such as chemical (trichloacetic acid) [21, 29], mechanical (dermabrasion) [5], surgical (excision) [20] and thermal (electrocautery) [5]. All these techniques left some kind of hypo or hyper-pigmentation as well as scarring [19] With the advent of lasers in the late 1960's the outcome of tattoo removal completely changed, due to its specific absorption by the pigment itself. One study resumes the final cosmetic outcome in removing tattoos with the (continuous wave) CW $CO_2$ laser [4].Even though the $CO_2$ laser gave more acceptable results than chemical or mechanical methods, there was still unacceptable scarring. All patients treated by argon and carbon dioxide laser have some form of secondary scar formation making both of them are not the ideal treatment modalities [3,6]. Leon Goldman published his first report of the use of a Q-switched ruby laser on a tattoo removal [12]. The technique was abandoned for almost 20 years. The reasons were mainly technical and commercial. The Q-switched ruby laser at this time was more a laboratory system than a laser which could be used in daily dermatological clinics. The first no scarring tattoo removal was demonstrated in Scotland by Reid et al [23].later on other teams refined the technique and used other Q-switched laser [17] .Within the last 13 years, Q-switched laser systems have been available, which leave minimal damage. They are able to remove some tattoos, depending on pigments that used [19], without scarring, hypo or hyper-pigmentation. Caution must be taken with the use of these lasers to avoid complications such as darkening. Some tattoo colors including flesh tones, light red, white, peach, and light brown containing pigments as well as some green and blue tattoo pigments, changed to black when irradiated with Q-switched laser pulses [2].Therefore in case of a multicolor tattoo treatment it is best to perform first a spot test on suspicious areas [22].

***Materials and methods***

After clinical assessments of tattoo, (99) lesions were divided according to their tattoo's colors, then subdivided into amateur and professional (see table 1). Complete medical history was taken about blood diseases, viral hepatitis, history of medications that were used by patient (aspirin, steroid, and anticoagulant), type of ink and instrument which were employed to make tattoo and the age of tattoo. Then prepare the person, shaving the hair, clean the area, apply a topical anesthetic (Emla cream) to skin, covered with an occlusive dressing and left for 30 minutes. This study was done at the dermatology department in al-zahura hospital, Damascus / Syria from 1st of august to 20th of December 2007.

**Table 1**: **Number of lesions, color of tattoo, and type of tattoo.**

| No. of lesions | Color of tattoo | Type of tattoo |
|---|---|---|
| 20 | blue | professional |
| 38 | blue | amateur |
| 10 | black | professional |
| 6 | black | amateur |
| 8 | green | professional |
| 3 | green | amateur |
| 14 | red(usually with other color) | professional |

The laser employed in this present work was a Q-Switched Nd: YAG laser which is characterized by: Maximum output energy is 1Joule. Peak power is [1J/10ns] =100 Megawatts. Pulse duration is 7-10ns. Wavelength is 1064 and 532nm. Repetition frequency is (1, 3, 5, and 10Hz). Diameter of light spot is 2-8mm (for 1064nm). Diameter of light spot is 2-6mm (for 532nm). Energy of pulse is 1000mJ (for 1064nm). Energy of pulse is 500mJ (for 532nm). Different fluencies were used according to treatment session (see table 2). Aiming beam is 3.0 mW of 635nm diode laser.

***Results***

The study revealed the following results (table 2 and figures 1-7):Blue color tattoo, amateur type responded faster than professional, needed 3-4 sessions, while the professional needed 5-6 sessions with faint shadow (figure 1-2). Black color amateur tattoo was responded faster than professional, needed 2-4 sessions (figure 3 and 5). While the professional black color tattoo needed 3-5 treatment sessions (see figure 4). Complete removal of red tattoo after 2-3 treatment sessions was obtained (figure 2, 4 and 6). For green color tattoo there was no response after 10 sessions (figure 7).

**Table 2: Parameters and Results**

| Wavelength(nm) | Parameters | Color of tattoo | Type of tattoo | Results |
|---|---|---|---|---|
| 1064 | Pulse width 10ns<br>Spot size (3mm)<br>R.R 5Hz<br>Fluence 6.3-10.1j/cm$^2$ | Blue | Professional | (20) Persons required (5-6) treatment sessions but all patients still have shadow. |
| | | | amateur | (38) Persons required (3-4) treatment sessions but all patients still have slight shadow. |
| 1064 | Pulse width 10ns<br>Spot size (3mm)<br>R.R 5Hz<br>Fluence 6.3-10.1j/cm$^2$ | Black | Professional | (10) persons required (3-5) treatment sessions for removing completely except (6) Patients still have cosmetically acceptable slight shadow. |
| | | | amateur | (6) Persons required (2-4) treatment sessions for removing completely tattoos except (2) patients still have cosmetically acceptable slight shadow. |
| 1064 | Pulse width 10ns<br>Spot size (3mm)<br>R.R 5Hz<br>Fluence 6.3-10.1j/cm$^2$ | Green | Professional | (8) Persons not response to treatment after (9-10).secessions. |
| | | | amateur | (3) Persons not response to treatment after (9-10).secessions except (1) patient slight faint in color after (9) sessions. |
| 532 | Pulse width 7ns<br>Spot size (2mm)<br>R.R 10Hz<br>Fluence 7.3-10.3j/cm$^2$ | Red | Professional<br>And amateur | Always the red color tattoo in companion with other color (Blue, black, green). (14) Red color tattoos all of them professional required (2-3) treatment sessions to be removed completely. |

**Blue color tattoo**

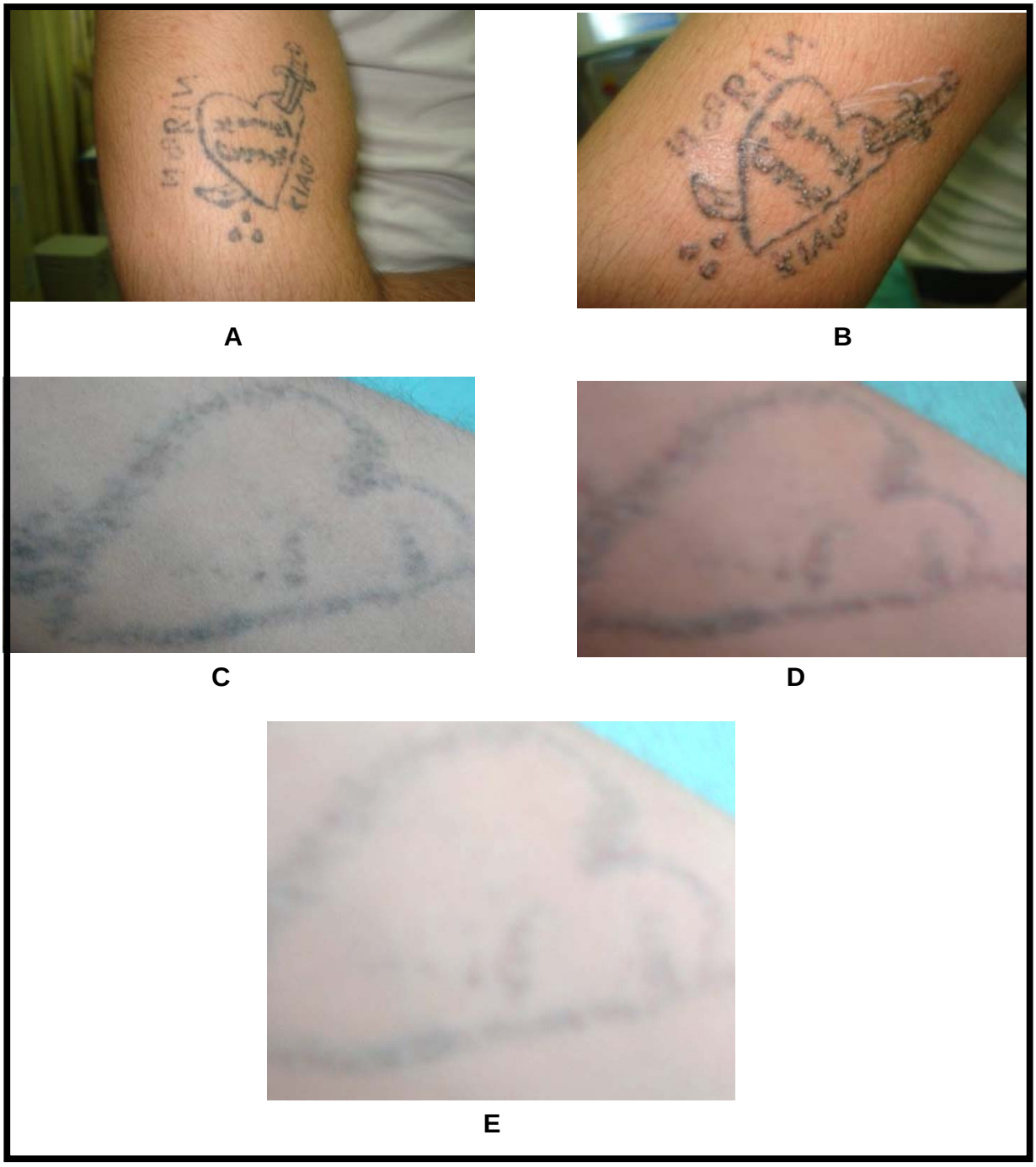


**Figure 1: Professional blue tattoo A=2nd session.B= 3rd session. C= 4th session. D= 5th session and E= 6th session.**

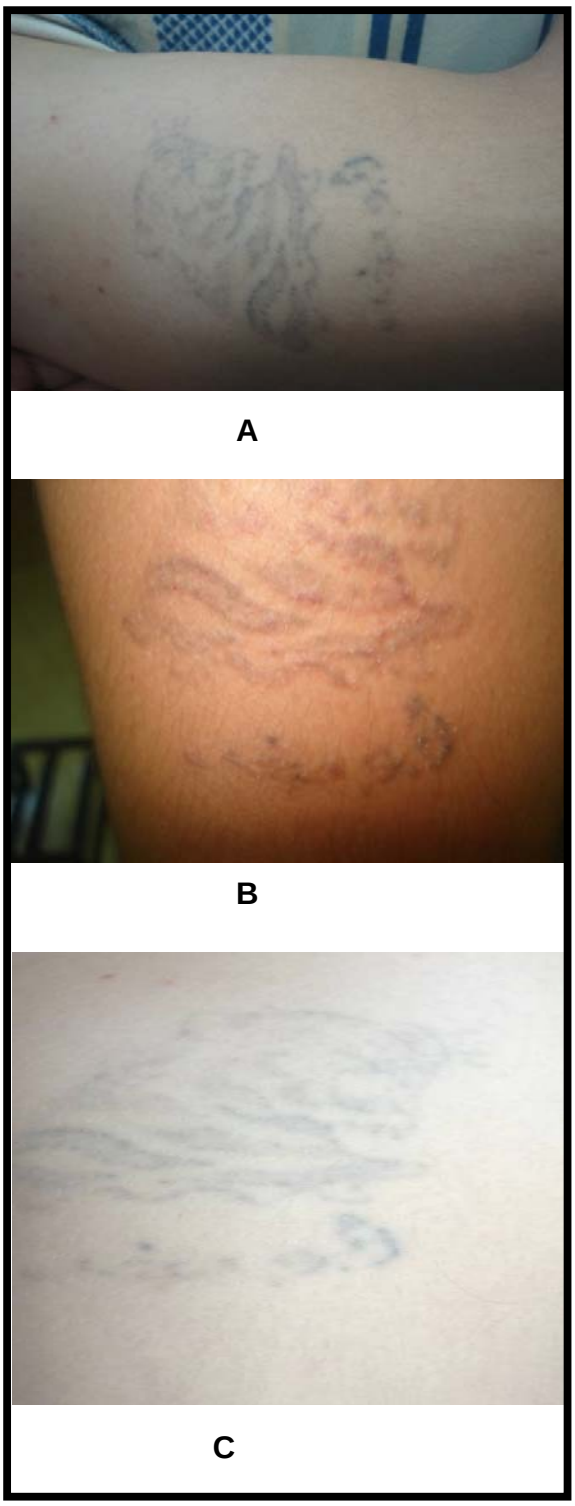


**Figure 2: Professional blue and red tattoo. A=4th session for blue and 1st session for red. B=5th session for blue and 2nd session for red. C= 6th session for blue and 3rd for red.**

**Black color tattoo**

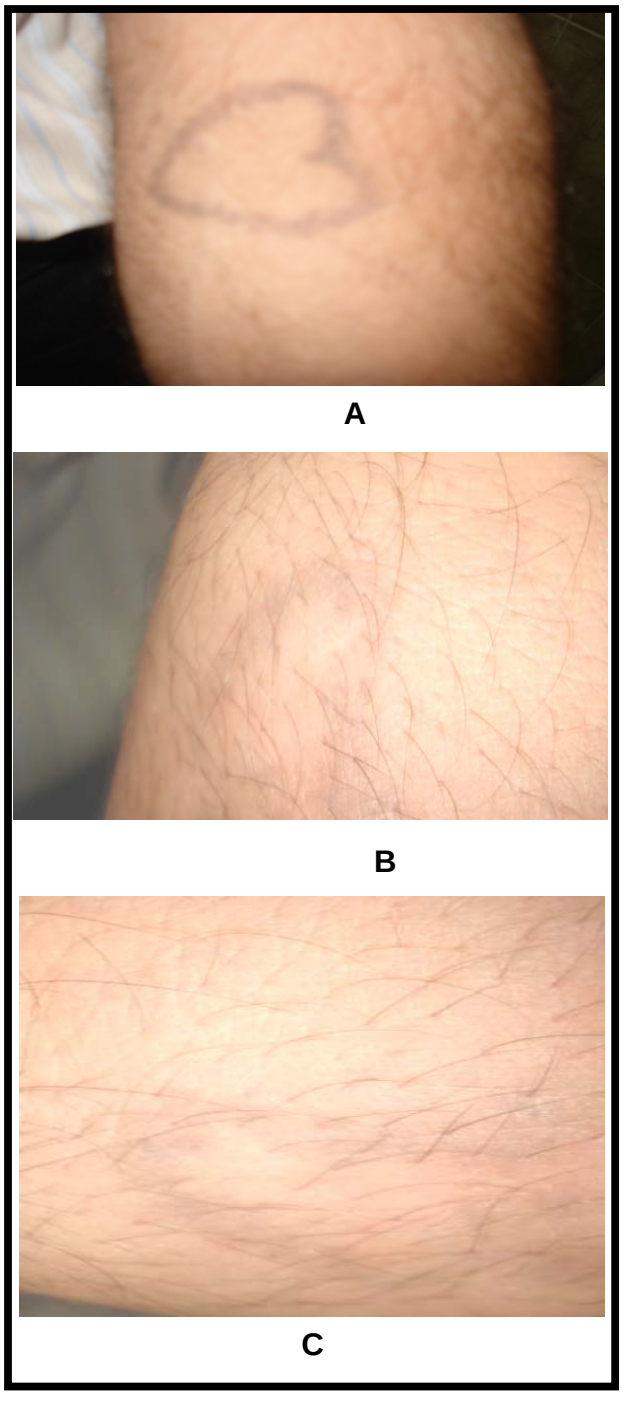


**Figure 3: Amateur black tattoo. A= 2nd session. B= 3rd session .C= 4th session**

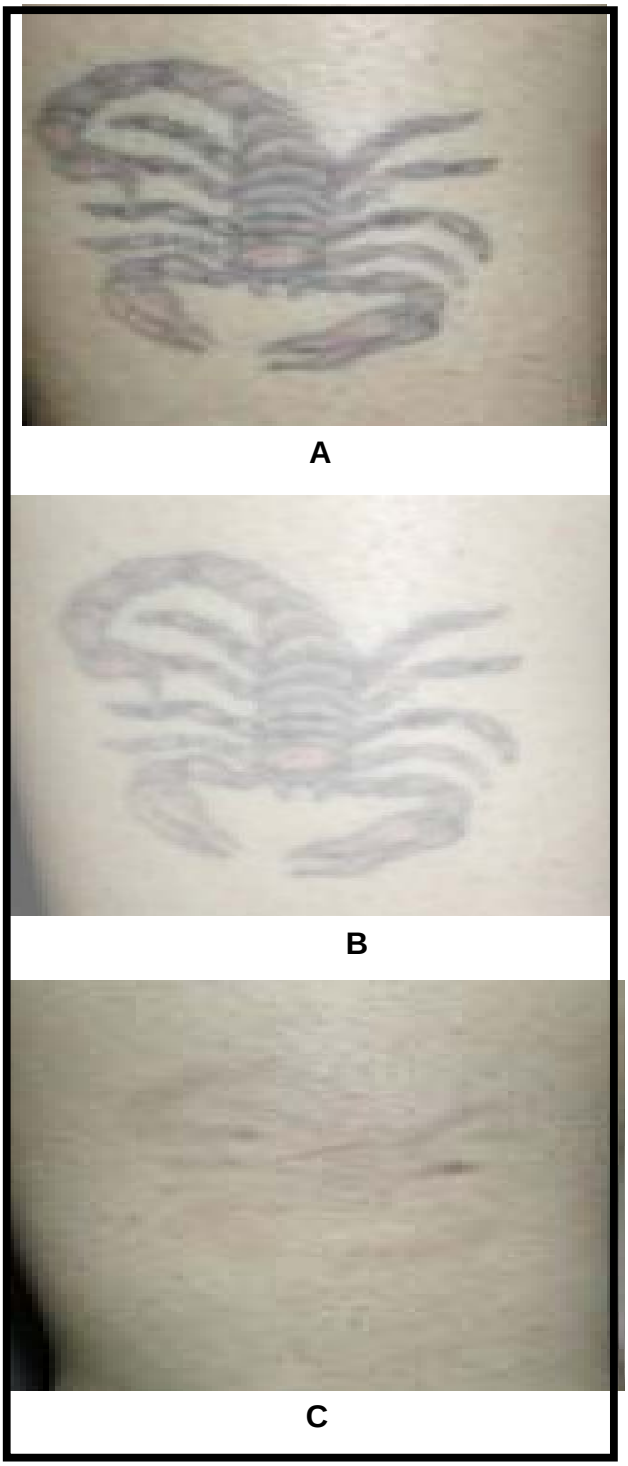


**Figure 4: Professional black and red tattoo. A= 2nd session for black and 1st session for red. B= 3rd for black and 2nd for red. C= 4th for black and 3rd for red.**

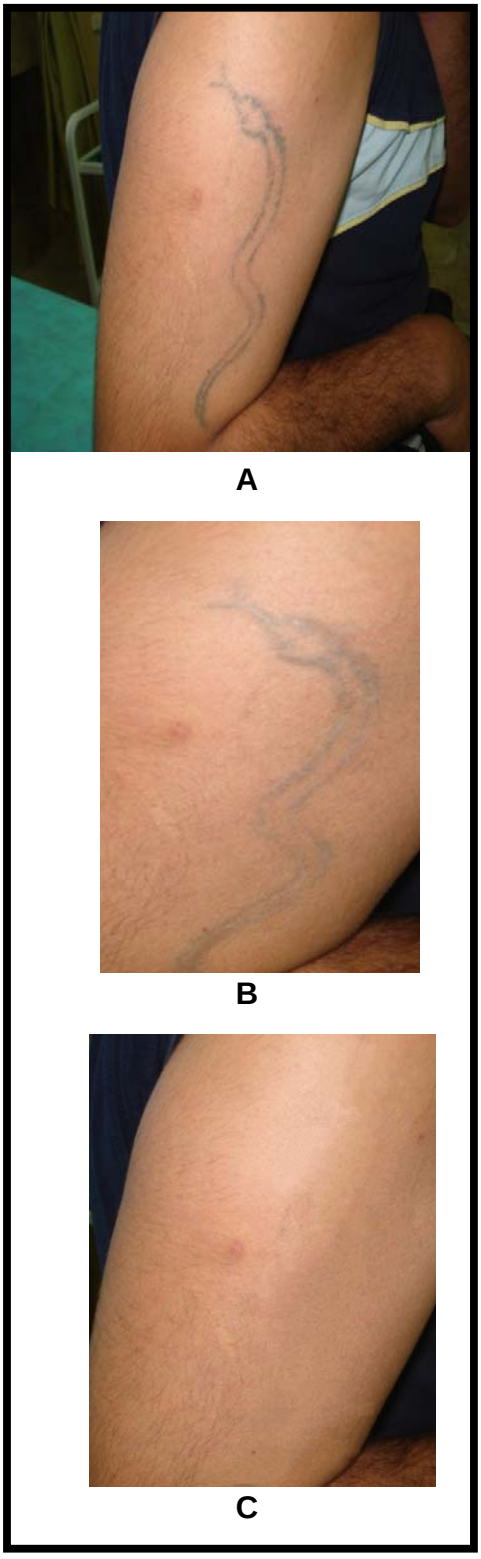


**Figure 5: Amateur black tattoo. A= 2nd session .B= 3rd and C=4th session.**

**Red color tattoo**

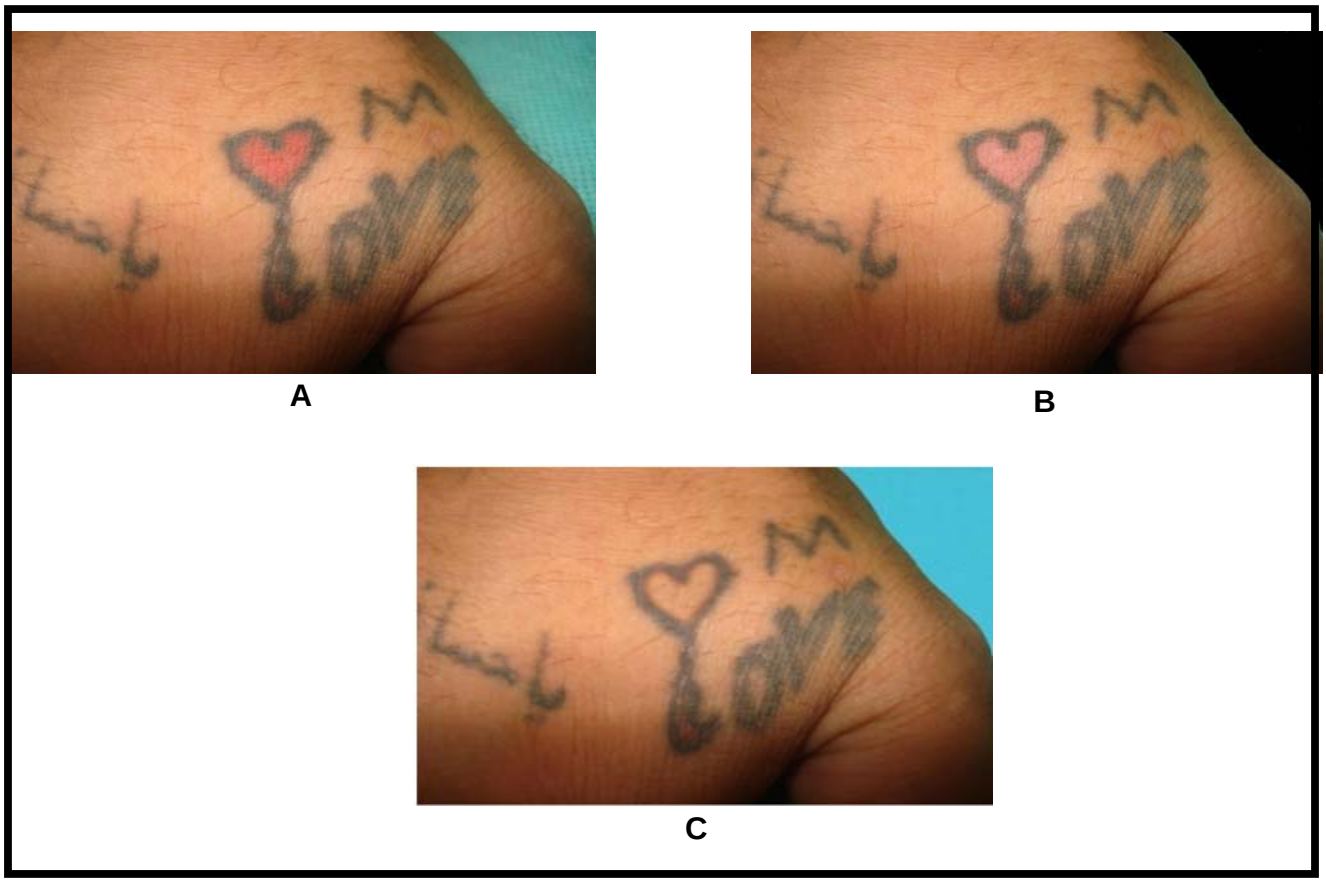


**Figure 6: Red tattoo. A=1st session. B= 2nd session. C= 3rd session**

**Green color tattoo**

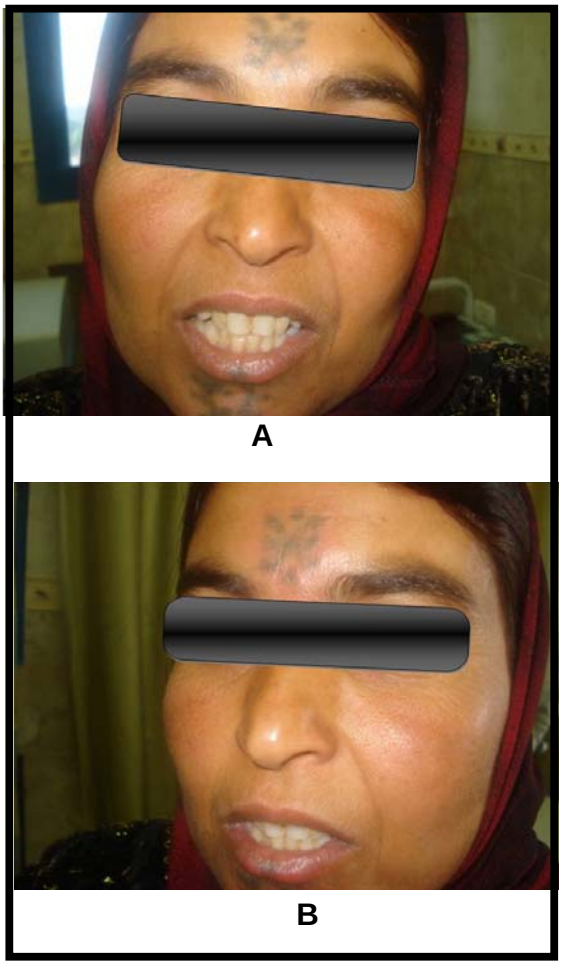


**Figure 7: Green tattoo. A= 9th session. B= 10th session.**

***Complications***

No complications have been seen like: scar, disfigurement or changing in the skin pigment, except the following transient complications have been noticed during this study (table 3 and figure 8).

**1.** Transient textural change was seen in (15) cases; occurred after multiple treatment sessions (figure 8, E and F). The textural change was resolved completely after 4 weeks without scarring; bepanthen ointment was used for treatment.

**2.** Pin-point bleeding: - occurred in (20) cases (figure 8, E and F) this due to in direct vascular injury from photo acoustic waves generated by the laser's interaction with tattoo pigment [40]. The pin-point bleeding was resolved within 5-7 days; it was treated by cleaning the area and topically applying antibiotic ointment and protective dressing to prevent secondary infection.

**3.** Systemic allergy occurred in one case only; the patient gave previous history of allergic rhinitis, the patient suffered, from joint pain, chills rhinitis, myalgia and itching after each laser treatment. Oral antihistamines and oral steroids before laser treatment are useful in this

case. The Q-switched laser is mobilizing the ink which may generate systemic allergic response.

**4.** Local allergic response (figure 8, D) happened in two cases, probably due to photo allergic reaction .Erythema, pruritus was happened in two cases after Q- switched laser treatment for red ink tattoo, this probably because the red ink contain cinnabar (mercuric sulphide) . This complication was treated by locally applied (steroid + antibiotic) ointment, oral antihistamines and sunscreen after laser treatment.

**Table 3: Complications after laser treatment.**

| Type of Complication | Number of lesions | % | notes |
|---|---|---|---|
| Hypopigmentation | Nil | - | - |
| Depigmentation | Nil | - | - |
| Hyperpigmentation | Nil | - | - |
| Transient textural change | 15 | 17.64 | Often resolved within one month |
| Permanent textural change | Nil | - | - |
| Scar | Nil | - | - |
| blister | Nil | - | - |
| Local allergic response | 2 | 2.35 | - |
| Erythema, pruritus | 2 | 2.35 | In red color tattoo (red ink contain cinnabar) |
| Inflamed nodules, varicose papules. | Nil | - | - |
| granulomas | Nil | - | - |
| Systemic allergic | 1 | 1.17 | Responded to antihistamines and steroids due to Q-switched mobilizing the ink which may generate systemic allergic response |
| Rupture blood vessels and aerosolize tissue | Nil | - | - |
| Pin-point bleeding | 20 | 23.52 | - |

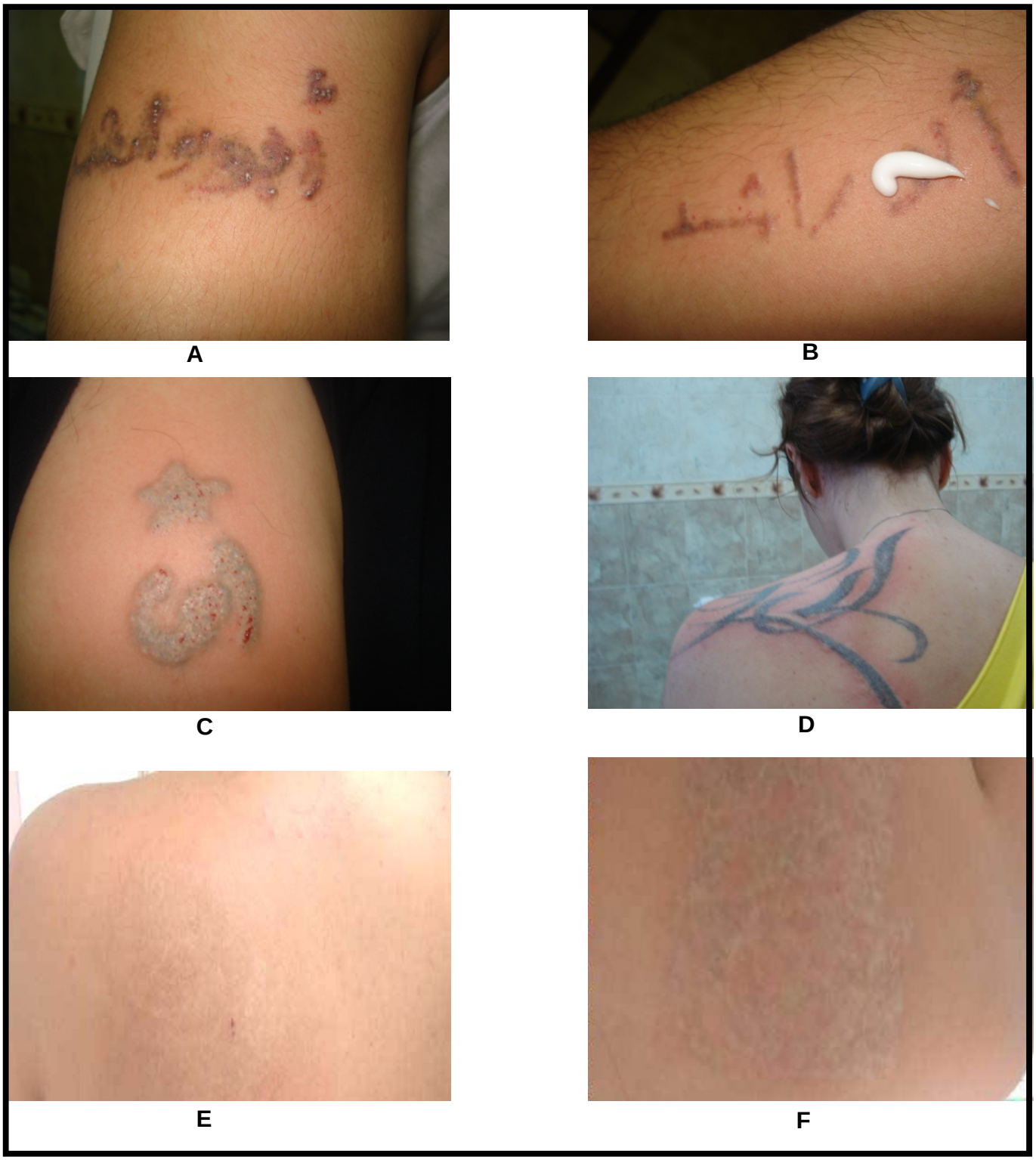


**Figure 8: A, B and C = Pin-point bleeding. D= local allergic response. E and F= transient textural changes.**

***Discussion***

The absorption of light pulses by the tattoo pigments is the first and most important step. If there is no absorption, there is no reaction [19]. When the laser light is absorbed by the pigment molecule, the light energy is converted to heat or break chemical bonds in the pigment molecules [30]. As the pigment heats up, the pigment molecule becomes extremely hot and causes rapid expansion of the water surrounding the molecule within the cell [30]. This in turn creates negative pressure and a shock wave is created near the surface of the pigment molecule which helps to destroy it [30]. The newly created smaller fragments are released into the lymphatic system and removed from the body [30].

The only part of the pigment which is really eliminated from the body is a very superficial one as it is eliminated by desquamation of epidermis during its repair; this phenomenon is called transepidermal elimination[35]. Removal of tattoo pigments probably depend on variables factor such as color of ink and on the chemical composition of ink, amount of ink injected in the skin, the immune ability to remove the disrupted ink particles, natural skin colors and how the depth of injected ink [16].

The reason that amateur tattoo ink is eliminated faster than professional ink is most likely due to a less uniform, most shallow distribution in the dermis as well as larger size of the individual ink particles found in amateur tattoo. For that reason amateur tattoo in blue and black colors needs less treatment session than professional. The study which done by Reid R [22] [24] stated that, professional tattoos required 1 - 3 additional treatment sessions for complete pigment removal. Another study done by Stratigos AJ [26] stated that Amateur tattoos require less number of treatments than professional tattoos. In this study red color tattoo required from 2-3 treatment sessions to be removed completely by 532nm Nd: YAG Q-switched laser. This explained in one study stated that, the red color well absorbed by 532nm [11] [16]. Other study stated that the red and black response well in most instances [25].Study done by Kilmer et al, stated that all tattoos containing red ink were removed completely in 1-3 treatment sessions [15].Green color tattoo not response to Nd:YAG laser 1064nm, only one case slight faint in color after 9 sessions this may be due to the green color ink contain titanium dioxide (TiO2) as explain in study stated that {white ink, composed of about 95% (TiO2), is commonly used to brighten green, yellow, and purple tattoos. the resulting mixtures are made such that green inks, for example, are typically composed of 30% to 40% (TiO2) [25][28]. Therefore, it is possible that tattoos with a large titanium fraction turn deeply black with Q-switched laser treatment, whereas other tattoos with smaller titanium "burdens" darken so little with laser irradiation that they appear grossly not to lighten. With repeated treatments of green tattoos, one possible scenario is that the green "organic" portion, at least superficially, is being eliminated, where as the titanium portion is darkening [25]. It is unknown, however, whether TiO2 is playing a role in resistance or, alternately, whether there is simply a higher damage threshold for the organic azo-dyes commonly used as green tattoo inks [25]. Other study stated that green ink is best treated by a red wavelength (694nm or 755nm) because the absorption of green ink is greatest at this

wavelength. [11][17][18]. The Q-switched Nd: YAG presented better initial as well as long-term results [13] [14]. Other study done by Jone A, and Grevelink JM, showed that the benefit of Q-switched Nd: YAG laser, it is safe used in darker-skinned patient in whom melanin absorption is concerned [10] [12].

In the present study the treatment with Q-switched Nd: YAG laser offers bloodless, low risk, no permanent complication no scarring and no disfigurement in skin was seen, The complication in this study is mainly transient textural change (no scarring) often resolved within one month, and pinpoint bleeding resolved within 5-7 days. There is no change in pigmentation of skin (no hypopigmentation or hyperpigmentation) this probably due to good preparation to patients; sun block is used by patients before and after laser treatment. So it is considered standard treatment for patient seeking tattoo removal.

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# Prevalence of hyaline membrane disease in cesarean section in al-kadhamia teaching hospital

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## *Abstract*

***Background:*** Hyaline membrane disease ,one of the commonest cause of sever respiratory distress early in life ,which is caused by surfactant deficiency is described not only in preterm infant but also in near term babies after cesarean section .

***Objective:*** The study aimed to identify the occurrence of hyaline membrane disease following cesarean section (cs) in AL-Kadhimiyia Teaching Hospital.

***Patient and methods:*** The study was conducted during the period between 1st January-30 April 2009, on 372 neonate born in AL-Kadhimiya Teaching Hospital .All patients were singletons, their gestational age between 37-40 weeks, and their body weight >2.5 kg. They were grouped into 3 groups according to the mode of delivery, normal vaginal delivery (NVD), emergency or elective CS.

***Result:*** Males were affected more than females (14.1%&9.2% respectively), occurrence of hyaline membrane disease was much higher after delivery by CS(18.5%)., than after NVD (4.76 %).There was little difference in the occurrence of hyaline membrane disease between emergency and elective CS and the lower the body weight of the neonate the more the occurrence of hyaline membrane disease was noted.

***Conclusion:*** Normal vaginal delivery has a possible protective effect against hyaline membrane disease as the frequencies of it's occurrence was noticed to be less in normal vaginal deliveries than in cesarean section deliveries.

***Key words:*** Hyaline membrane disease, normal vaginal delivery, cesarean section ,elective ,emergency .

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## *Introduction*

Hyaline membrane disease (HMD) which is characterized by stiff , non – compliant lungs and is due to surfactant deficiency ,is one of the commonest cause of Respiratory distress syndrome(RDS ) that occur early in life [(1)],This syndrome defined as the presence of at least two of the following clinical sings: tachypnea >60 , dyspnea with inspiratory sub costal and intercostals retractions, nasal flaring, expiratory grunting and cyanosis in room air . the most frequent underlying cause of RDS during the first 48 hours are transient tachypnea of the new born , infections, meconium aspiration syndrome , hyaline membrane disease (HMD) and perinatal asphyxia [(2)].The diagnosis of HMD will depend on clinical feature in addition to chest radiography which shows diffuse reticulogranular opacities , air

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branchograms and small lung volume the main factors predisposing to HMD include prematurely , male sex , and maternal diabetes[3] ·also cesarean section in particular is associated with increase incidence of hyaline membrane disease[4],and delivery by cesarean section continue to increase in both developed and developing countries [5].

***Patient and method***

A cross -sectional study was done in Al-Kadhimiyia Teaching hospital at the period from 1st of January-30 April 2009. A three hundred seventy two neonate were included in the study 189(50.8%), delivered by normal vaginal delivery and 183(49.2%) delivered by cesarean section (160 of them delivered by emergency cesarean section and 23 by elective cesarean section), all of them were singletons, their gestational age from 37-40 weeks and without apparent congenital malformations, all pregnancies associated with pre-eclampsia , chronic hypertension, diabetes, multiple gestations and meconium stained amniotic fluid were excluded.

We recognized cesarean sections done after the start of labor or rupture of membrane as emergency cesarean section, while those done before the onset of labor or by scheduled surgery were considered as elective cesarean section. Vaginal delivery was considered normal if the onset of labor was spontaneous and there was no complication of pregnancy or instrumentations.

Then all neonates were grouped into three groups according to their body weight, the first group weight was 2.5-3, kg, the second group weight was 3.1-3.5 kg, the third group weight> 3.5 kg, then we compare the incidence of HMD in each group. All result were expressed in numbers and percentages while statistical analysis was done by using chi sequire and p value of equal or less than 0.05 was designated as statistically significant .

***Results***

In our study total number of neonate was 372, 189( 50.81%) of them delivered by NVD and183( 49.19 % ) delivered by CS and the percentage of emergency CS was 160 ( 87.43 %) ,and the elective CS was 23 (12.43 %) of total CS, also it had been found that 9 (4.76 %) 0f neonate delivered by NVD had HMD in contrast to 17.3 % (34 ) of those delivered by CS, which is statistical significant since p value was 0.00017 .this is shown in table 1.

**Table 1:Distibution of HMD and mode of delivery.**

| Type of delivery / HMD | Em.CS | % | El.CS | % | NVD | % | total | % | |
|---|---|---|---|---|---|---|---|---|---|
| **HMD +ve** | 30 | 18.7% | 4 | 17.3% | 9 | 4.76% | 43 | 11.5% | $\mathbf{X^2}$=17.4 **df**=2 **P**=0.00017 |
| **HMD -ve** | 130 | 81.3% | 19 | 82.6% | 180 | 95.2% | 329 | 88.4% | |
| **Total** | 160 | 100% | 23 | 100% | 189 | 100% | 372 | 100% | |

**Em.CS :Emergency cesarean section El.CS :Elective cesarean section**

Table 2 . Shows that 34.85 % of those neonate whose body weight (B. wt ) was bellow 3 Kg and the percentage decrease with increasing in B. wt until it reach 4.73 % of neonate with B.wt more than 3.5 Kg which statistically significant result , p value equal to 0.00000012.

**Table 2: Distribution HMD according to body weight.**

| B.wt / HMD | 2.5-3 kg | | 3.1-3.5kg | | >3.5kg | | total | |
|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | | |
| **HMD+ve** | 23 | 34.85% | 13 | 8.23 % | 7 | 4.73 % | 43 | $\mathbf{X^2}$=35.03 **df**=3 **P**=o.oooooo12 |
| **HMD-ve** | 43 | 65.15% | 145 | 91.77% | 141 | 95.27% | 329 | |
| **total** | 66 | 100 % | 158 | 100 % | 148 | 100 % | 372 | |

**B.wt :body weight**

In this study it had been found that male were slightly affected more than female (14.1% and 9.2% respectively) as shown in table 3.

**Table 3: Distribution of HMD according to sex**.

| | Male | | females | | Total |
|---|---|---|---|---|---|
| | **No.** | **%** | **No** | **%** | |
| **HMD+ve** | 25 | 14.2 | 18 | 9.3 | 43 |
| **HMD-ve** | 152 | 85.8 | 177 | 90.7 | 329 |
| **Total** | 177 | 100 | 195 | 100 | 372 |

***Discussion***

In this study the occurrence of HMD in term babies was high (11.5%) in comparism with other studies like a study in England[6] where the incidence is 0.1%.

Delivery by CS showed increased risk of HMD in both emergency and elective CS, but the risk is low in neonate delivered by NVD. These results are similar to many other studies which had demonstrated the protective nature of NVD in lowering the incidence of HMD.In Norwegian population –based cohort study they found cesarean delivery to be a major risk factor associated with the development of HMD[7] .A hospital based case control study in Beirut showed that after controlling for other factors HMD was twice as likely in infants delivered by cesarean delivery [8] . Another study done in Pakistan showed that HMD is three folds higher in CS group than those delivered vaginally[9].Some authors have suggested that the mechanism for decreased HMD in vaginally delivered neonates is associated with endogenous prostaglandin production stimulated by uterine activity [10,11] . Some have suggested labor results in the release of lung surfactant into the airways, other theories to explain HMD in cesarean delivered infants include persistent fetal circulation as well as increased retention of pulmonary fluid in neonate delivered by CS.[12] During vaginal delivery about one third of fetal lung fluid is removed by squeezing the babies chest, this removal is missing during delivery by CS[13]. Others have suggested that a beta-adrenergic surge during labor may be responsible for the ultimate fetal lung expulsion of surfactant in preparation for birth[14-16] .

In this study HMD was nearly the same in those delivered by emergency CS and in those delivered by elective CS, while in other studies like Curet et al , and Kim A found cesarean delivery before labor associated with higher incidence of HMD compared with cesarean done after the onset of labor pain[17-19] .This can be explained by the fact that the time of delivery that matters because that is what determines lung functional development – a view borne out by Gabert et al [20] . who showed that CS was not associated with HMD when the lecithin –sphingomylin ratio offered a good prognosis. In this study HMD was inversely proportional to body weight of neonate , similar results were obtained by many other studies like in Australian study showing that higher risk of HMD was associated with body weights lower than 2.5kg and the risk is lower in those Weighing > 3.5 kg [9] .

This was explained by the fact that HMD is thought to be caused by high lung alveolar surface tension, causing atelactesis and lack of pulmonary surfactant, which is a combination of lecithin ,phosphatidylglycerol , cholesterol and surfactant apoproteins. The production of surfactant by fetal lung begins by week 20 but does not reach the surface of the fetal lung until much later[15] .In this study males are

affected more than females and this is similar to all the above studies**.**

All doctors should always remember that NVD has a protective effect against hyaline membrane disease, so CS should be limited only to those patients with real indications, and elective CS should always be planed at 39-40 weeks of gestation.

Also patients should be educated about the risk of CS on the baby.

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# Redo hypospadias surgery; experience with 27 patients with prior distal or proximal hypospadias repair failure

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## *Abstract*

***Background :***Urethral reconstruction in failed hypospadias poses a significant challenge. We report our experience using tabularized incised plate method in distal type of hypospadias and excision of fistulous tract with dartos flab in more proximal types of hypospadias.

***Objective:*** To retrospectively review our experience in a series of re-operative hypospadias repairs for distal and proximal types of hypospadias repair failure.

***Materials and Methods*:** Between December 2006 and June 2009, 27 children (mean age 4.8 years, range 2 years to 11 years) were referred for re-operation of failure of hypospadias repair. The patients were divided into 2 groups; group1 (20 patients) with distal and midshaft hypospadias. In these cases, the Tubularized incised plate (TIP) urethroplasty was covered with an additional layer of subcutaneous tissue or dartos flap. Group 2(7 patients) with proximal shaft or penoscrotal hypospadias types that were complicated with fistula formation, excision of the fistula tract was done with closure with interrupted sutures and a second layer covering (dartos) was performed

The original location, associated complications and results were recorded.

***Results:*** for group 1 There were 5 (25%), incidences of complications of TIP re-operation, 3 meatal stenosis, one stenosis with small fistula and one dehiscence. Re-operation was necessary in only one patient of our series (7.6%) and the others were cured by dilatation.

Group 2: 2 patients out of 7(28.5%) had failure of repair with persistence of the fistula that required reoperation

***Conclusion:*** for distal type Hypospadias using TIP urethroplasty as described by Snodgrass, is a suitable method for treating primary and re-operative cases. While for more proximal hypospadias closure of the defect and a second layer covering to prevent fistula is a viable option treatment.

***Key words:*** urethroplasty; hypospadias; urethral plate; tubularized incised plate (TIP)

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## *Introduction*

Numerous methods for repair of hypospadias have been introduced. However, urethrocutaneous fistula or neourethral dehiscence was the most troublesome complication. These problems are the main difficulty in re-operations, because in these cases urethral reconstruction is required, but only a small amount of penile foreskin is available.On the other hand, the vasculature of previously operated tissues may be suboptimal, resulting in further complications. In 1994, Warren Snodgrass described a procedure using tubularized incised plate (TIP) urethroplasty with excellent results [(1)]. The TIP urethroplasty has also been used successfully in re-operative and complex hypospadias repairs [(2-4)].

Although, tubularized incised plate urethroplasty is well described, there are few reported experiences pertaining to complicated hypospadias or circumcised patients that are re-operated by this technique. We report our results in using

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the TIP urethroplasty with a local flap in previous hypospadias repair complicated by fistula or dehiscence.

On the other hand fistula that develops after repair of more proximal types of hypospadias are more challenging, they require more delicate procedures and a higher failure rate.

***Materials and methods***

Between December 2006 and June 2009, 27 children (mean age 4.8 years, range 2 years to 11 years) were referred for re-operation of failure of hypospadias repair.

The patients were divided into two groups

***Group 1***: twenty [20] patients had a failed hypospadias repair of distal [18 patients] and mid shaft [2 patients] penile hypospadias (Figure 1) who had previously undergone a failed hypospadias repair. The previous techniques utilized were Mathieu repair in 6, MAGPI in 4 and unknown in 10. Glanular hypospadias were excluded from this study. The interval from the last surgery to TIP re-operation was between 6 months to 9 years.

After the primary evaluation, tubularized incised plate urethroplasty (TIP) was performed for correction of complications related to the previous hypospadias surgery. All of the re-operations were performed by the same surgeon. After general anesthesia, a stay suture was placed through the glans for traction. Then the penis was degloved and any meatal stenosis or fistula opened widely, to prevent subsequent stricture formation. An artificial erection was carried out for ventral curvature, as a necessary step. Parallel incisions separated the glans wings from the urethral plate and the plate was incised in the mid-line as described by Snodgrass [(1)]. An 8F or 10F Foleys catheter was passed into the bladder for post operative urinary diversion, then, an urethroplasty was performed using subcuticular 4/0 vicryl continuous sutures. The epithelium of the urethral plate was inverted toward the lumen to avoid fistula formation. Care was taken to avoid suturing the distal urethral plate too tightly, which may result in meatal stenosis. Usually only 1 or 2 sutures beyond the mid glans penis level of the plate were needed, leaving the neomeatus oval in configuration [(3)]. The neourethra was covered by a second layer of dartos tissue pedicle then closing the skin with interrupted sutures and a compression dressing was applied. All patients were discharged from the hospital one to two days after surgery. Catheter and dressing were removed after five days. Patients were examined twice in the first month (Figure 2), with follow-up within a 6 month period. Patients who had an acceptable cosmetic appearance and voided from the end of the penis with no difficulty were considered as successful surgery.

***Group 2***: 7 patients had failed hypospadias repair of proximal penile [4 patients] and penoscrotal [3 patients] hypospadias (Figure 3).

All the patients had fistula formation near the proximal end of the neourethra of the previous repair

After the initial assessment of the patients, repair of the fistula was decided and under general anesthesia, the procedure started with the assessment of the neourethra with calibration to exclude any stenosis or stricture, a 8 or 10 Fr Foleys' catheter was inserted to the bladder, excision of the fistula and closure of the defect with interrupted 4/0 vicryl sutures with inversion of the urethral edges towards the lumen then covering the area with a second layer of

dartos tissue pedicle harvested from the nearby tissue (Figure 4), the skin was closed by interrupted sutures (Figure 5).

At the end of the procedure suprapubic cystostmy tube was inserted as safety measure to divert the urine and a compression dressing was applied. All patients were discharged from the hospital one to two days after surgery. Catheter and dressing were removed after four days and suprapubic cystostomy on day 6. Patients were examined twice in the first month, with follow-up within a 6 month period. Patients who had an acceptable cosmetic appearance and no recurrence of the fistula and voided from the end of the penis with no difficulty were considered as successful surgery.

***Results***

The mean follow-up after surgery was 6 months (range 4-9 months)

Group1

There were five complications; a six-year old boy that was referred after a failed repair. The day after surgery he developed severe bladder spasms. Subsequently, the patient's glanuloplasty dehisced which required re-operation. Three children developed meatal stenosis that responded to 2-3 times calibration. The fifth child developed a pin hole fistula and stenosis. After 6 weeks calibration (twice per week), both of them were cured.

Group2:

Two children developed complication including failure of the fistula closure after repair that required another operation after 6-12 months

***Discussion***

In the correction of complicated hypospadias, it is preferable to use vascularized preputial or penile skin. When genital skin is unavailable or insufficient, it may be necessary to choose extragenital tissues such as skin, bladder mucosa and buccal mucosa, in order to complete a successful repair. Duckett et al.[5] comment that buccal mucosa grafts are the best urethral replacement for redo surgery and for stricture disease, and the meatus will be durable.

In contrast, hypospadias repair with Snodgrass incised plate urethroplasty in primary cases, has gained widespread acceptance because it is versatile, and has the advantages of reliably creating a vertically oriented meatus, while having a lower complication rate than other techniques. These excellent results have been reported in literature as primary repair [1, 3]. Although the use of Snodgrass urethroplasty has been extended from primary to re-operative hypospadias [2, 4, 6, 7], these reports do not appear to be very conclusive.

In group 1 patients (n=20), our complications rate (25%) was related to five patients: i.e. three meatal stenoses, one stenosis with a small fistula and one dehiscence. This study, and also the report of Yang et al. [8], demonstrated that the meatal stenosis is the most frequent form of complications in re-operative TIP urethroplasty especially in distal types. Although a wide neomeatus has been made, the meatal stenosis had the most complications.

The results would be similar to Snodgrass and Lorenzo [3] who reported the usage of TIP urethroplasty to repair proximal hypospadias (33%). Although their cases were proximal, complications in re-operation (2) were 3 in 15 (20%), and is similar to those reported by Shanberg et al. [6] and Borer et al. [9] 24%, 15%, respectively. It is very important to note that in only one patient of our series, re-operation was necessary

while others were cured by dilatation; this indicates that the ultimate success rate without another operation was 92.4%. We had a patient with dehiscence glanuloplasty that underwent a successful second redo tubularized incised plate urethroplasty re-operation and responded satisfactorily

For prevention of fistula, when possible, the neo-urethra was covered with a blanket of tunica vaginalis (Figure 4) or some other buffering vascularized layer as an alternative flap for multilayer coverage of the urethroplasty. Therefore, the incidence of fistula was only one case that could be due to meatal stricture. Meatal stenosis is the most reported form of complication and usually responds to dilatation. Although uroflowmetry was not performed, meatal stenosis was evaluated clinically. Based on the opinion of Duckett et al.[5], flowmetry is a good objective measure of caliber, but observation of a good full stream is subsequently more revealing in follow-up. Ideally one should have both [5].

In conclusion, using the TIP urethroplasty as described by Snodgrass et al. is a suitable method for treating the re-operative cases. It can also be used successfully in patients who do not have a healthy skin flap and for circumcised patients when there is a complete lack of foreskin.

Group2:

In the management of fistula of more proximal hypospadias failed repair, the same principles of excision of the fisula tract, tension free closure and a second layer covering help decreasing the incidence of complications specially dehiscence and fistula after the redo-repair

Bracka [10] reported his experience with 600 patients with primary and secondary hypospadias repairs, he concluded that the second layer largely decrease the incidence of fistula from 63% without a second layer to only 5.4% with the use of dartos flap as a second covering layer

While Massimo Catti et al[11] described the incidence of fistula in redo hypospadias repair was as high as 20% and this was more common with free graft use like buccal mucosal graft than grafts with a pedicle.

In the second group series seven [7] patients had fistula as a complication of prior proximal hypospadias repair two of them developed fistula postoperatively making the fistula rate as high as 28.5%.Although the patients' sample is small (seven patients only) this high failure rate can be explained by several reasons like the early experience of the surgeon and the type of suture material used which is vicryl 4/0 while ideally it is 6/0 or 7/0 vicryl as described by Massimo Catti[11]

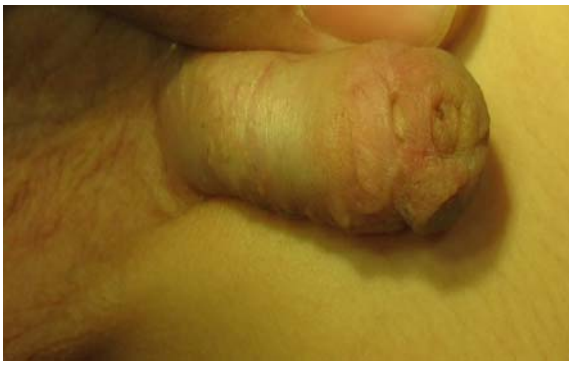

**Figure 1**

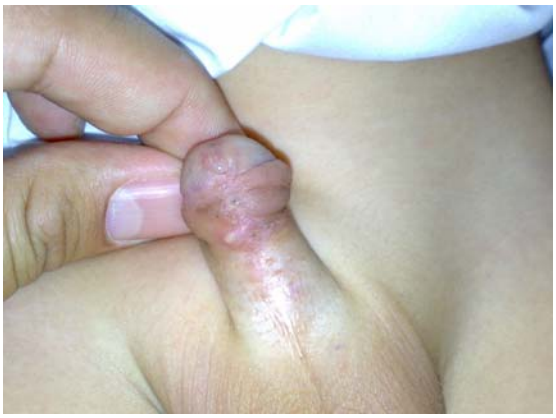

**Figure 2**

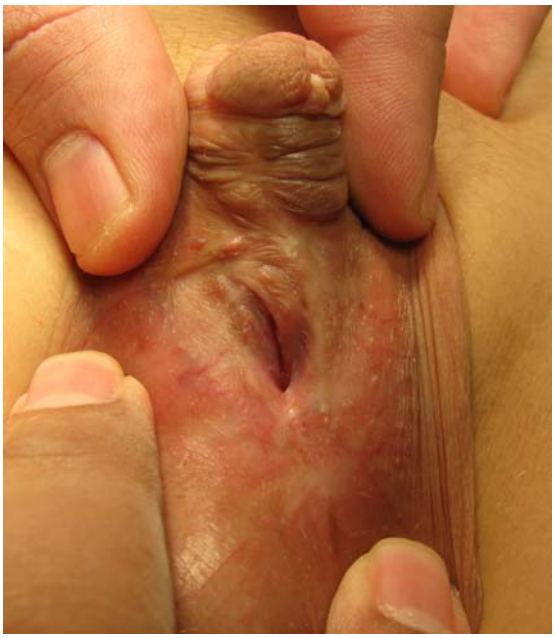

**Figure 3**

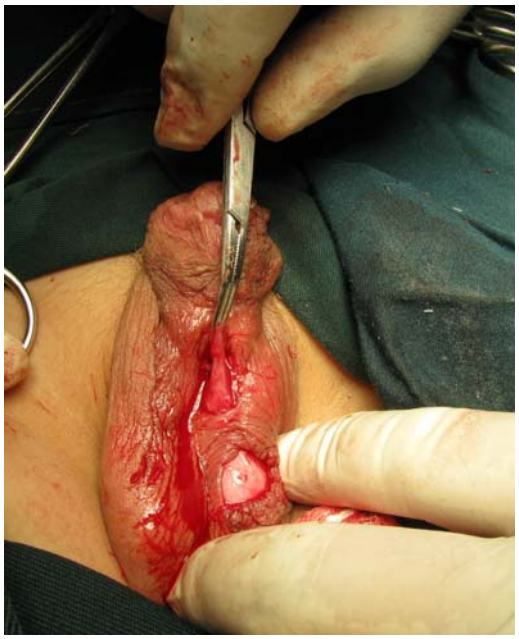

**Figure 4**

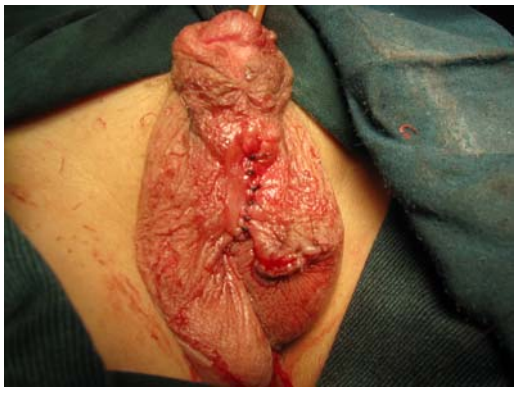

**Figure 5**

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# Study of Socio-demographic characteristics of patients with congenital coagulation disorders

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## Abstract

***Background:*** patients with genetic deficiencies of plasma coagulation factors exhibit life-long recurrent bleeding episodes into joints, muscles and closed space, either spontaneously or following an injury.

***Objective:*** The present study aimed to determine the socio-demographic characteristics of patients with congenital coagulation disorders.

***Methods:*** A cross-sectional study was conducted at the Centre of Congenital Coagulation Disorders in the Al-Mansour Pediatric Teaching Hospital in Baghdad for the period between 1st of March to 31st of August 2008.

The study sample was comprised of 243 patients with different congenital coagulation disorders who attended the Centre during the study period.

Data were collected through well structured questionnaire form introduced only by the researcher by interviewing with the patient or his/her relative or care giver.

***Results:*** showed that hemophilia and Von Willebrand Disease (VWD) constituted the majority (90.1%), of the studied sample

The mean age of all patients was 14.31 ±10.42 years. About 77% of patients were from Baghdad governorate. Some families (15.6%) of the studied patients had three or more members with congenital coagulation disorders. For those patients aged ≥7 years, 41% of them had not attended or left school due to their disease. For those patients aged ≥18 years, 68.4% of them were unmarried, and 45.07% of them had no work due to their disease. Treatments were available to the majority (97.1%) of patients

***Conclusions:*** The features in this study were similar to other studies in Mediterranean region and Western countries, except that the mean age of patients in this study was lower than that in other studies, blood groups showed no significant effect on types of congenital coagulation disorders, the disease showed adverse effects on all levels of education of patients

***Key words:*** Study of Congenital coagulation disorders in Baghdad city

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## Introduction

Coagulation disorders deal with disruption of the body's ability to control clotting.(1) Coagulation factor disorders can arise either from deficiency, usually congenital, of a single factor- e.g. factor VIII in hemophilia A or from multiple factor deficiencies which are often acquired, e.g. secondary to liver disease (2) Patients with genetic deficiency of plasma coagulation factors exhibit lifelong recurrent bleeding episodes into joints, muscles, and closed spaces,either spontaneously or following an injury. The most common inherited factor deficiencies are the hemophilias; X-linked diseases caused by deficiency of factor VIII (hemophilia A) or factor IX (hemophilia B). Rare congenital bleeding disorders due to the deficiencies of other factors including factor II (prothrombin), factor V, factor VII, factor X, factor XIII, and fibrinogen are usually inherited in an autosomal recessive manner (3).

Hemophilia A (factor VIII deficiency) one out of every 5000 males worldwide; Christmas disease, or hemophilia B, is less common than hemophilia A. both are the most common severe inherited bleeding

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disorders [4], Von Willebrand's disease affects both males and females and is often diagnosed in children, the remaining defects, generally transmitted as autosomal recessive traits in both sexes, are rare, with prevalence of the presumably homozygous forms in the general population ranging from approximately 1 in 2 million to 1 in 500,000 [5]. Frequently these conditions produce life-threatening and limb- threatening complications. Moderate and mild coagulopathies may remain clinically silent until they are detected serendipitously on routine laboratory screening assay for global coagulation (e.g. PT or aPTT ) or when these assays are ordered to evaluate the cause of abnormal bleeding or easy bruisability [6].

This study was done to assess the social and demographic characteristics of patients with congenital coagulation disorders.

***Patients and methods***

A cross-sectional study was conducted at the Centre of Congenital Coagulation Disorder (CCCD) in Baghdad city, this centre is the only centre in Iraq, was opened in 1997 and located within the Al-Mansour Pediatric Teaching Hospital which is connected with the General Administration of Medical City in Baghdad, it has a special Laboratory for diagnosis and wards inside the hospital for diagnosis and treatment for patients (from all age groups) who visited the centre from all over the country, The centre gives medical services to all patients with congenital coagulation disorder (CCD) who attend the centre except hemophilic patients with AIDS (referred to Al-Towaitha hospital for treatment), the centre also provides medical consultation about surgery for patients with CCD. The number of patients who attended the Centre each day was around 15 patients (new& old ones), and by the time the study was finished there were 1048 files for all patients who attended the Centre since it was opened until the end of the study period.

All patients (old and new cases) attending the CCCD for diagnosis, treatment, consultation, and follow up were included in the study during the period from the 1st of March to the 31st of August 2008, the work was for 5 days per week with a working hours of 4-5 hours/day. Two hundred forty three patients with different congenital coagulation disorders were included through well structured questionnaire form, data collected by the researcher only by direct interviewing with the patients or his\her parents or close relative. Information collected included different socioeconomic and demographic characteristics, family history, Some information were taken from the file of the patients like type of CCD, blood levels of factor VIII and factor IX for hemophilic patients, blood groups, , and availability of treatment.

In this study, the severity of hemophilia was classified on the basis of the patients baseline level of factor VIII or factor IX, patients having less than 1.0 unit/dl (<1%) of the specific clotting factor were considered as severe hemophilia, patients having 1-5 unit/dl as moderate hemophilia and patients having greater than 5 unit/dl were considered as mild hemophilia [4].

Data analysis was done using descriptive measures including mean, median, standard deviation, percentages, and test of significant done using chi-square. P value≤ 0.05 was considered significant.

***Results***

Hemophilia (A & B) and Von Willebrand disease constituted the majority (90.1%) of patients and corresponded to 64.6% of patients with hemophilia A, 16% hemophilia B, and

9.5% with Von Willebrand disease. Rare bleeding disorders (RBD) including factor 1, V, VII, X, and factor XIII deficiencies constituted 9.9% of the study sample. The proportion of cases with hemophilia A to B was 5:1 (table 1).

Generally, the studied sample included 90.1% males, and 9.9% females. All cases of hemophilia were males while the other congenital coagulation disorders include males and females totally in about equal number and even in each disease, the sex distribution is close in numbers, Of 243 patients; 239 (98.4%) were Arabic, while only 1.6% were Kurdish and they were hemophilic. All cases were Muslims, The mean age with standard deviation for all patients was 14.31± 10.42. The largest range of age was found among patients with factor VII deficiency 3.5 - 50 years. The least range of age 5 – 15 years was found among patients with factor XIII deficiency (Table 1).

Table-2: shows that 73.7% of patients with congenital coagulation disorders were below 20 years of age on attending the centre during the study period. It was found that most cases of hemophilia were either severe (66.3%) or moderate (22.5%), while the mild cases constituted only 11.2% of hemophilia patient. Statistically, the difference between the degree of severity of patients with hemophilia A and patients with hemophilia B was not significant P=0.494 (Table 3)

It was found that 187 patients (77%) were from Baghdad and 23% were from other governorates mainly those around Baghdad such as Dyala [26 patients,(10.6%)], Babil [6 patients,(2.5%)], Salahaddin [6 patients, (2.5%)], Anbar [5 patients, (2.1%)], and Wasit 2.1% (Table 4)

Out of 178 patients of ≥7 years of age; 7.3% were not attending primary school due to their diseases, and 41% had not attended or left school due to their diseases in comparison to 10.7% of patients who had not attended or left school not due to their diseases . Also it was noticed that slightly less than one third of them were either illiterates (9%) or just read and write (23%). On the other hand; 6 patients (3.4%) finished only the secondary school, and only 3 patients (1.7%) finished the university. Also 43.2% were still studying in different stages and more than half of them were still studying in primary school.(Table 5)

It was found that out of 79 patients above 18 years old; about two third (54 patients, 68.4%) of them were single, and about one third 31.6% of them were married.(Figure 1)

It was found that 45.07 had no work due to their diseases (Figure 2).

Generally speaking, slightly more than two third of patients (68.7%) had positive family pedigree and less than one third (31.3%) had negative family pedigree.(Table 6)

Figure 3 shows that:

- 134 patient's families (55.1%) had only one patient with CCD.
- 71 patient's families (29.2%) had two patients with CCD.
- About 10% of patients families had three patients with CCD.
- 6 patient's families (2.5%) had four patients with CCD.
- 8 patient's families (3.3%) had five patients with CCD.

It was found that 110 patients (45.3%) had blood group O, 70 patients (28.8%) had blood group B, 47 patients (19.3%) had blood group A, and 16 patients (6.6%) had blood group AB. Blood group showed no significant effect on type of CCDs, P value was 0.384.(Table 7)

During the study period, recombinant factor VII and factor IX were available to all patients with deficiencies of these factors.

Recombinant factor VIII was available to the majority of hemophilia A patients except 7 patients who received cryoprecipitate instead of recombinant F VIII concentrate When the latter was unavailable.(Table 8).

The median of times of attendance of all studied sample was 4 times per a year with a range of 0-39 times. Hemophilic patients had the largest range (up to 39 times) of attending the Centre per a year, while patients with factor X deficiency had the largest median of times (10 times) of attending the Centre per a year. The least median of times of attending the Centre was noticed in patients with Von Willebrand disease and patients with factor XIII deficiency which was only 2 times per a year.

Days of admission during the last 12 months: For all patients, the median was 5 days of admission per a year with a range of 0-55 days. The largest median of days of admission to the centre per a year was noticed among patients with factor X deficiency (13 days) , while patients with factor XIII deficiency or Von Willebrand disease had the lowest days of admissions to the centre (2 days, 3 days respectively) per a year, patients with hemophilia A were found to have the largest number of admission days (up to 55 days) per a year, and then followed by patients with hemophilia B (up to 38 days per a year). Other congenital coagulation disorders had admission of 20 days or less per a year. Treatment were available for 97.1% of patients with CCDs (Table 8)

**Table 1: Distribution of patients with congenital coagulation disorders in the sample**

| Types of CCDs* | Sex | | | | Race | | Mean age ± SD**(year) | Range of age | total | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Male | | Female | | Arabic | Kurdish | | | | |
| | No | % | No | % | | | | | No | % |
| Hemophilia A | 157 | 100 | --- | ---- | 154 | 3 | 14.95±10.3 | 5 m***-43y**** | 157 | 64.6 |
| Hemophilia B | 39 | !00 | ---- | ----- | 38 | 1 | 10.313.29± | 7 m-41 y | 39 | 16.0 |
| Von Willebrand disease | 10 | 43.5 | 13 | 56.5 | 23 | --------- | 11.29± 8.3 | 1 y-35 y | 23 | 9.5 |
| Factor I deficiency | 4 | 66.7 | 2 | 33.3 | 6 | ------- | 13.27 ±7.9 | 1.5 y-21 y | 6 | 2.5 |
| Factor V deficiency | 0 | ----- | 1 | 100 | 1 | --------- | 18.25 | 18 y | 1 | 0.4 |
| FactorVII dificiency | 4 | 44.4 | 5 | 55.6 | 9 | ------ | 15.8±16.3 | 3.5 y50 y | 9 | 3.7 |
| Factor X deficiency | 2 | 66.7 | 1 | 33.3 | 3 | ------- | 23.8 ±17.1 | 6 y-40 y | 3 | 1.2 |
| Factor XIII deficiency | 3 | 60 | 2 | 40 | 5 | ------- | 8.6 ±3.7 | 5 y-15 y | 5 | 2.1 |
| Total | 219 | 90.1 | 24 | 9.9 | 239 | 4 | 14.3 ± 10.4 | 5 m-50 y | 243 | 100 |

*CCDs= congenital coagulation disorders
**SD=Standard Deviation
*** m=months
**** y=years

**Table 2: Distribution of congenital coagulation disorders by age**

| Age (years) | Hemophilia A | | Hemophilia B | | Von Willebrand disease | | Rare inherited bleeding disorders | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Freq | % | Freq | % | Freq | % | Freq | % | Freq | % |
| **< 1** | 2 | 0.82 | 2 | 0.82 | - | | - | | 4 | 1.64 |
| **1-4** | 29 | 11..93 | 10 | 4.12 | 5 | 2.06 | 5 | 2.06 | 49 | 20.17 |
| **5-9** | 28 | 11.53 | 7 | 2.88 | 7 | 2.88 | 7 | 2.88 | 49 | 20.17 |
| **10-14** | 32 | 13.17 | 6 | 2.47 | 5 | 2.06 | 2 | 0.82 | 45 | 18..52 |
| **15-19** | 22 | 9.05 | 3 | 1.23 | 3 | 1.23 | 4 | 1.65 | 32 | 13.16 |
| **20-29** | 28 | 11.52 | 8 | 3.3 | 2 | 0.82 | 3 | 1.23 | 41 | 16.87 |
| **30-49** | 16 | 6.58 | 3 | 1.24 | 1 | 0.41 | 3 | 1.24 | 23 | 9.47 |
| **Total** | 157 | | 39 | | 23 | | 24 | | 243 | 100 |

**Table 3: Degree of severity of hemophilic patients in the studied sample**

| Degree of severity | Hemophilia A | | Hemophilia B | | Total | | $\chi^2$ | P value |
|---|---|---|---|---|---|---|---|---|
| | Freq | % | Freq | % | Freq | % | | |
| **Mild** | 17 | 10.8 | 5 | 12.8 | 22 | 11.2 | 1.41 df=2 | 0.494 |
| **Moderate** | 38 | 24.2 | 6 | 15.4 | 44 | 22.5 | | |
| **Severe** | 102 | 65.0 | 28 | 71.8 | 130 | 66.3 | | |
| **Total** | 157 | 100 | 39 | 100 | 196 | 100 | | |

**Table 4: Governoratal distribution of patients with congenital coagulation disorders**

| Governorates | No. of patients | % |
|---|---|---|
| **Baghdad** | 187 | 77 |
| **Dyala** | 26 | 10.7 |
| **Babil** | 6 | 2.5 |
| **Salahaddin** | 6 | 2.5 |
| **Anbar** | 5 | 2.1 |
| **Wasit** | 5 | 2.1 |
| **Najaf** | 2 | 0.8 |
| **Al-Quadessia** | 1 | 0.4 |
| **karbala** | 1 | 0.4 |
| **Mesan** | 1 | 0.4 |
| **Basrah** | 1 | 0.4 |
| **Al-Mothanna** | 1 | 0.4 |
| **Nainawa** | 1 | 0.4 |
| **Total** | 243 | 100.0 |

**Table 5: Schooling situation and educational status of patients ≥7 years old with congenital coagulation disorders**

| Educational level | Still studying | Reason for not obtaining optimal educational level | | Finishing secondary or university study |
|---|---|---|---|---|
| | | Leave school due to disease | Leave school for other causes | |
| **Not attending school (n=16)** | - | 13 (81.3%) | 3 (18.7%) | - |
| **Primary school (n=83)** | 42 (50.6%) | 36 (43.4%) | 5 (6%) | - |
| **Intermediate school (n=49)** | 23 (46.9%) | 17 (34.7%) | 9 (18.4%) | - |
| **Secondary school (n=20)** | 6 (30%) | 6 (30%) | 2 (10%) | 6 (30%) |
| **University (n=10)** | 6 (60%) | 1 (10%) | - | 3 (30%) |
| **Total (n=178)** | 77 (43.2%) | 73 (41%) | 19 (10.7%) | 9 (5%) |

**Table 6: Family pedigree of patients with congenital coagulation disorders**

| Congenital coagulation disorders | Family pedigree | | | |
|---|---|---|---|---|
| | yes | | no | |
| | No. | % | No | % |
| **Hemophilia A** | 116 | 73.9 | 41 | 26.1 |
| **Hemophilia B** | 27 | 69.2 | 12 | 30.8 |
| **Von Willebrand disease** | 12 | 52.2 | 11 | 47.8 |
| **Rare inherited bleeding disorders** | 12 | 50.0 | 12 | 50.0 |
| **Total** | 167 | 68.7 | 76 | 31.3 |

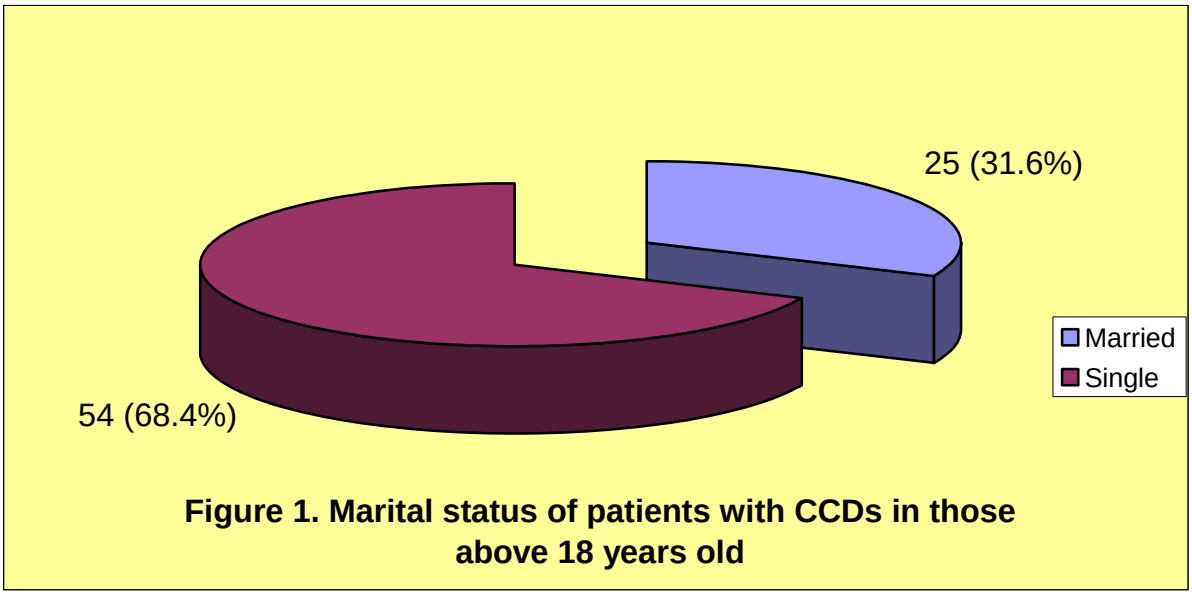


**Figure 1. Marital status of patients with CCDs in those above 18 years old**

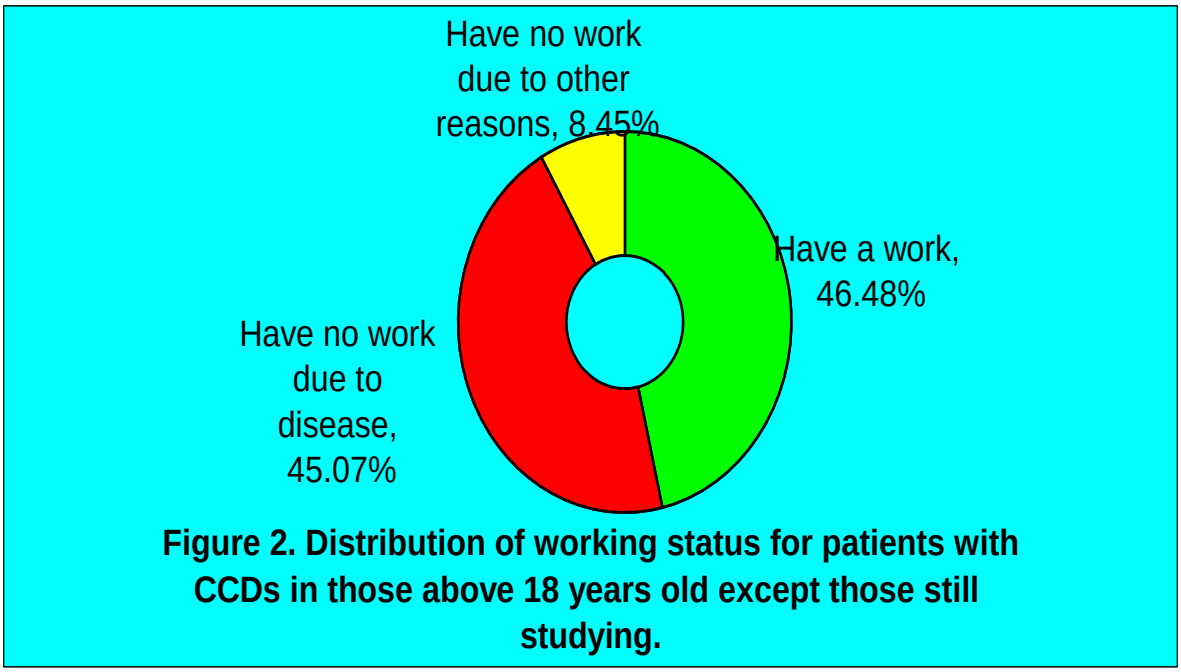


**Figure 2. Distribution of working status for patients with CCDs in those above 18 years old except those still studying.**

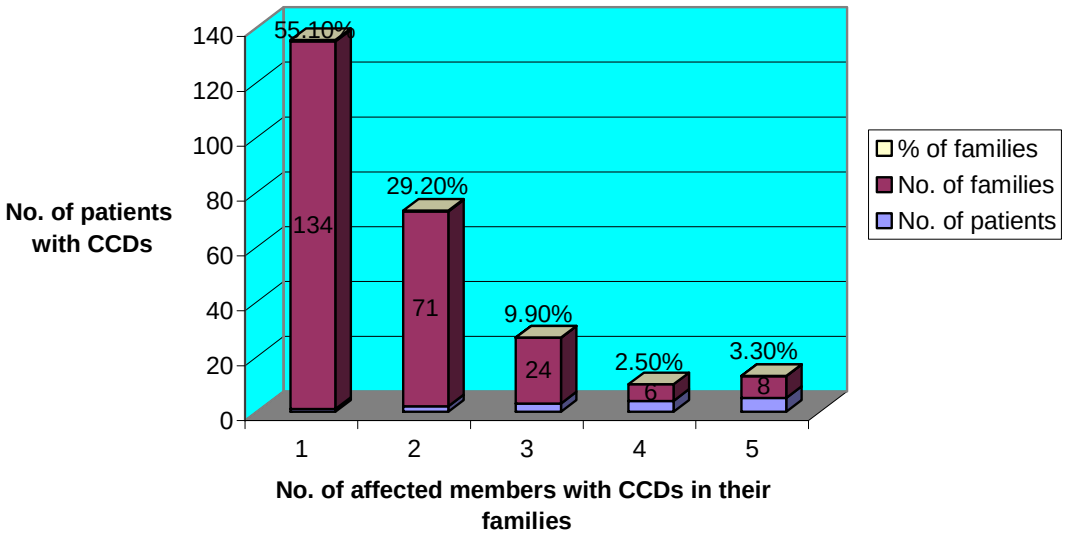


**Figure 3. Ditribution of the number of affected members with CCDs within their families**

**Table 7: Blood group distribution among patients with congenital coagulation disorders**

| Blood group | Hemophilia A | Hemophilia B | Von Willebrand disease | Rare bleeding disorders | Total | $\chi^2$ | P value |
|---|---|---|---|---|---|---|---|
| **A** | 32 | 8 | 6 | 1 | 47 (19.3%) | 9.6 df=9 | 0.384 |
| **B** | 45 | 10 | 6 | 9 | 70 (28.8%) | | |
| **AB** | 10 | 1 | 1 | 4 | 16 (6.6%) | | |
| **O** | 70 | 20 | 10 | 10 | 110 (45.3%) | | |
| **Total** | 157 | 39 | 23 | 24 | 243 | | |

**Table 8. Distribution of times of attendance and days of admission to the Centre during the last 12 months for patients with CCDs**

| Types of CCDs* | Times of attendance to centre during last 12 months | | Days of admission to centre during last 12 months | | Treatment availability* | | | |
|---|---|---|---|---|---|---|---|---|
| | Median | Range | Median | Range | Yes | % | No | % |
| **Hemophilia A** | 5 | 0-39 | 6 | 0-55 | 150 | 95.5 % | 7 | 4.5 |
| **Hemophilia B** | 4 | 0-30 | 6 | 0-38 | 39 | 100 | -- | ---- |
| **Von Willebrand bisease** | 2 | 0-9 | 3 | 0-17 | 23 | 100 | ---- | ---- |
| **Factor I deficiency** | 6 | 2-12 | 6 | 2-14 | 24 | 100 | ---- | ---- |
| **Factor V deficiency** | 1 | ------ | 1 | ----- | | | | |
| **Factor VII deficiency** | 5 | 1-20 | 6 | 2-14 | | | | |
| **Factor X deficiency** | 10 | 2-20 | 13 | 2-20 | | | | |
| **Factor XIII deficiency** | 2 | 1-6 | 2 | 1-7 | | | | |
| **Average** | 4 | 0-39 | 5 | 0-55 | | | | |
| **Total** | | | | | 236 | 97.1 | 7 | 2.9 |

* Treatment availability for haemophilic patients includes only recombinant factors

***Discussion***

This is the first study on the socio-demographic aspects of congenital coagulation disorders in the Centre of Congenital Coagulation Disorders in Iraq, which was established in Baghdad city on 1997, that make it difficult to compare the result of this study with other Iraqi studies except with the only two studies found; one related to the prevalence of viral hepatitis markers in patients with hereditary bleeding disorders carried on 1997 by Dilshad Saber [7], and the other was a prospective on-going study by AL-Mondhiry [8] and was published at Thrombosis and Hemostat Journal 1977. In this study, the majority of patients (90.1%) had either hemophilia or Von Willebrand disease and this result is similar to studies in Iraq [8], KSA[9], Iran [10], Poland [11], Pakistan [12], Brazil [13], Europe [14], and Thailand [15]. In this study, hemophilia-A was the most prevalent (corresponded to 64.6%) among patients with congenital coagulation disorders. This result was in agreement with studies in Iraq [8], Saudi Arabia [9], Iran [10], USA [16], Canada [17], Italia [18], Poland [11], Brazil [13], Thailand [15], and India [19]. The ratio of hemophilia-A to B was 4:1, this result was similar to other studies in which the ratio range 3.7:1 to 6.5:1 as such that in:

Iran by Mehdizadeh et al., [20] the ratio was 5: 1.

India by Kulkarni et al., [19] the ratio was 5.3: 1.

USA by Michael Soucie et al., [16] the ratio was 3.7: 1.

Brazil by Rezende et al., [13] the ratio was 5: 1.

Canada by Ronald O. Barr et al., [17] the ratio was 4: 1.

Spain by Aznar et al., [21] the ratio was 6.5: 1.

Poland by Windyqa et al., [11] the ratio was 6: 1.

Most cases of hemophilia were severe (66.3%), followed by moderate (22.5%), and mild (11.2%). This distribution of severity, as expected, reflect that the severe cases attended the Centre more frequently than moderate or mild cases and caused increase in their prevalence. This result was in concordance with report of National hemophilia foundation [22]. The result that severe form was more prevalent than the other form was in agreement with a study in USA [16], Italia [18], Poland [11], Canada [18], Brazil [13], and Netherlands [23]. In this study, the proportion of moderate and mild forms had disagreement with other studies [9, 20], this discrepancy in the prevalence of degree of severity of hemophilia in different studies may be related to differences in the types of studies and on the criteria on which hemophilia was defined.

In von Willebrand disease, male to female ratio was 1: 1.3 and this was in agreement to a study in Brazil [13] with a ratio of 1: 1.5. In this study, 47.9% of patients with VWD were between 10-49 years old in contrast to 76.7% in a study in Brazil conducted by Rezende et al. [13] This discrepancy may be due to that about half of patients (52.2%) in this study were between 1-10 years of age. `In rare inherited disorders, the most common one was factor VII deficiency and constituted 37.5% of rare bleeding disorders. This result was in agreement with a study in Iran conducted by Karimi et al. [10], a study in Pakistan [12] and a study in Poland [11].

While rare inherited bleeding disorders (RIBD) was found in this study to constitute the minority (9.9%) of the studied sample and is similar to studies in Iraq [8] (11.3%), Iran[10] (9.1%), KSA[9] (14.3%), India [19] ( 12.5%), and Pakistan [12] (5.9%) , but is

more than studies in Europe [14] (3-5%), Brazil [13] (2.4%) , and Thailand [15] (3.86%). This discrepancy is most likely due to high rate of consanguinity marriages and large number of births per family in Iraq. It was slightly higher than that in Brazil (2.4%) [13], Europe (3-5%) [14] and Thailand (3.9%) [15]. In all these countries in addition to a study in North America and Canada conducted by Acharya [24] factor VII was the more prevalent among RIBD and in agreement to the result of this study.

The mean age of all patients on examination in this study was 14.31±10.42 years which was less than a study in Iran carried by Mehdizadeh, et al.[20] (29.92±15.19 years), and another study also in Iran carried by Mohssen Nassirtoosi, et al.[25] with a mean of 26.6±12.1 years and most of the patients were in the third decade of life. It has been estimated that > 80% of patients older than 20 years of age are HCV antibody positive as of 2006 [3], in the present sample majority of patients are young ones who are born after 1986 which might indicate that some of them had already received non- virucidal factor concentrate, the study shows that 73.7% of patients were below 20 years old, while a study carried in Iran [20] showed that half of the population studied was younger than 24 years of age.

It was found that 77% of patients were from Baghdad, and 23% were from governorates other than Baghdad especially those governorates close to Baghdad geographically. This finding may be due to inaccessibility of patients from these governorates. The low levels of educational status of patients with CCDs may be explained by fearing of patients parents from getting trauma to their affected children in school and so prevent them from attending school, or due to frequent absences from school because of their frequent attendances and admissions to hospital.

Unfortunately, there were no other studies accessible for comparison.

It was found that 68.4% of all patients above 18 years old were unmarried. And apart from those who still studying, 53.5% had no work whether due to their disease or not. Also blood groups showed no significant effect on types of CCD. Unfortunately, there were no other studies accessible for comparison.

It was found that 29.2% of families had two patients, and 15.7% of families had more than two patients with this disorder. This finding is due to inherent nature of these diseases, large Iraqi family size, and sharply pointed to the need of proper education and genetic counseling for family planning for those families. Unfortunately, there were no other studies were accessed for comparison. In this study, family pedigree was negative in 26.1% and 30.8%of patients with hemophilia A and B respectively. This result was in agreement with what was published in USA [27] , and was in consistent with the hypothesis of Heldane [28] that lethal X-linked recessive disorders in approximately one third of patients and are due to spontaneous mutation, but this result was in disagreement with a study in Poland [11] which showed that about 50% of the hemophiliacs have no history of bleeding diathesis in the family.

When the study considered rare inherited disorders, the proportion of male (54.2%) and female (45.8%) in this study was in agreement to that found in a study in Pakistan [12] Family pedigree was positive in 50% of patients with RIBD. This finding was in agreement with a study in North America and Canada[ 24] and a study conducted by Herrmann et al on

subjects from Europe and Latin America with mutation in the factor VII gene[29] . Although about half of patients were with blood group O, there were no significant differences between the different types of CCDS regarding their blood group, this possibly could be due to sample size and also those with rare bleeding disorders were only 24 patients in the sample.

The median times of annual attendance to the Centre by patients with congenital coagulation disorders was 4 times, with a range of 0-39 times. Also these patients had a median of 5 days admission per a year with a range of 0-55 days of admission per a year. This reflect the high rate of attendance and admission of patients with congenital coagulation disorders to this Centre and it was mainly due to the nature of their diseases, treatment were available to 97.1% of patients with CCDs, which in some way reflect the support of the government to this centre in spite of the difficulties facing the country in the current situation.

There is a clear need for extensive study in the whole country to determine the exact prevalence and other epidemiological distribution of CCDS, a need to establish new centers in governorates other than Baghdad and planning for administering home-treatment (prophylaxis treatment) for patients with severe hemophilia.

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# Urethral Diverticulum after Endoscopic Urethrotomy, A Case Report

**Ula M.R.Al-Kawaz** *MBChB; FIBMS (Urology).*

***Abstract***

Diverticula of the male penile urethra are rare clinical entities. Urethral diverticula in males may be associated with trauma, infection, impacted calculi or stricture disease. Here in, we present an unusual case of a 55-year-old man with and a symptomatic urethral diverticulum after endoscopic urethrotomy for a bulbar urethral stricture. Surgical repair involving urethral stricture excision, end-to-end primary urethroplasty, and closure of the diverticular neck, the patient is voiding well but has persistent erectile dysfunction unresponsive to phosphodiesterase-5 inhibitors.

***Keywords:*** urethral diverticulum, urethroplasty, stricture

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## *Introduction*

Diverticulum of the male[1] bulbar urethra is a rare clinical entities with unknown exact incidence[(1)] , unlike the in females in which diverculum is relatively a common disease. Urethral diverticula in males may be caused by or associated with several pathological conditions including trauma, infection, impacted calculi or stricture disease [(1, 2)].

The pathophysiology of most cases of urethral diverticula appears to revolve around obstruction of and infection within the paraurethral glands. The glands are thought to become enlarged and inflamed, eventually forming a retention cyst and then an abscess, which ruptures back into the urethra [(3, 4)].

The clinical presentation of urethral diverticula varies considerably from patient to patient and also may vary depending on when during the natural history of the disorder the diagnosis is made. Early in the natural history, when the periurethral gland initially becomes infected, the predominant symptoms may be related to urination.At this stage, dysuria, frequency, and postmicturition dribbling may bring the patient to clinical attention. Later, as chronic and recurrent inflammation develops around the diverticulum, low pelvic pain may be reported as well. Clinical signs such as pyuria, a palpable suburethral mass, suburethral indurations, and tenderness may be present. Diagnosis can be confirmed by Voiding cystourethrography, Ultrasonography, Urethral pressure profilometry and Urethroscopy [(5)].

## *The case*

A 55 years old man that presented with dysuria and post micturation dribbling, he gave a history of trauma to the pelvis 10 years ago since then he had a urethral stricture that was treated four times with endoscopic urethrotomy, the patient neglected his condition for 5 years then presented to our unit with his urinary symptoms .

Examination revealed a compressible lump at the perineum that evacuates pus and urine through the urethra. Urinalysis revealed pus cells and bacteria. Cystourethrography revealed a huge diverticulum at the bulbar part of the urethra Figure (1); Urethroscopy revealed a stricture distal to the Diverticulum that was treated by cold knife urethrotomy and

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a wide neck urethral Diverticulum was found. An open urethroplasty Figures (2, 3) was decided with excision of the whole diverticulum and the stricture site was done with end-to-end anastamosis and closing the urethra and overlying layers.

***Discussion***

Urethral diverticula in males is a rare disease, very few reports in the literature about this condition,
Exact incidence is unknown [4].

Parker WR, Wheat J [5] reported their unusual case of a 57-year-old man with erectile dysfunction and a symptomatic urethral diverticulum after endoscopic urethrotomy for a pendulous urethral stricture. One year after surgical repair involving urethral stricture excision, end-to-end primary urethroplasty, and closure of the diverticular neck, the patient is voiding well but has persistent erectile dysfunction unresponsive to phospho-diesterase-5 inhibitors.

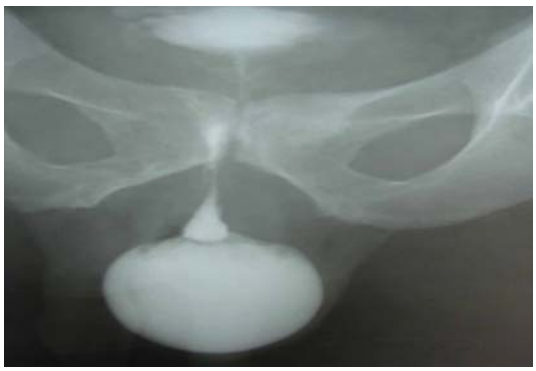

**Figure 1**

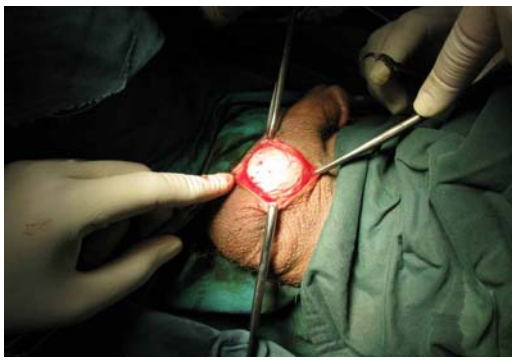

**Figure 2**

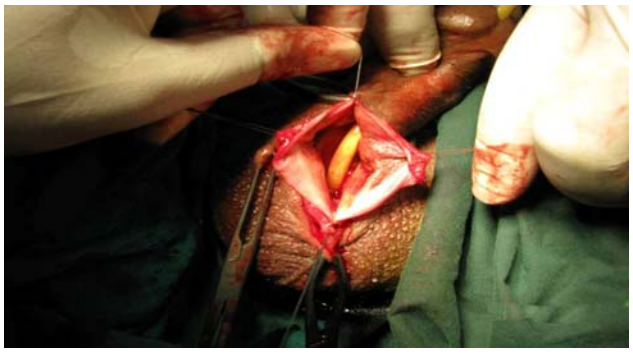

**Figure 3**

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# Torsion of the Gallbladder in an Adult: A Rare Case of Acute Cholecystitis

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***Abstract***

Torsion of the gallbladder is a rare condition that is generally due to an abnormal anatomical variation, *i.e.*, the presence of a long mesocyst with loss of fixation of gall-bladder to the inferior margin of the liver. The clinical features closely mimic those of acute cholecystitis. In any case, the definitive diagnosis is made during surgery [1, 2].

***Keywords:*** Torsion gallbladder- Laparoscopy - Acute cholecystitis

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***Case report***

An old woman aged 60 years was admitted with a 12-hour history of right hypochondriac pain, nausea, and vomiting. Her medical history included hypertension. Physical examination revealed tachycardia (heart rate, 110 beats/min). Her blood pressure was 165/90 mmHg and her temperature was 37.5°C. She was not jaundiced. Plain abdominal X-ray revealed dilated small bowel consistent with localized ileus. Abdominal ultra-sonography demonstrated a distended, fluid-filled, thick-walled gallbladder with surrounding edema. There were no stones. The preoperative diagnosis was acute cholecystitis. At exploration, the gallbladder was suspended by a long cystic duct and a short mesentery. It was gangrenous and twisted more than 360° clockwise on the cystic duct. Cholecystectomy was carried out without incident.

***Discussion***

Acute torsion of the gallbladder is a rare abdominal disease [1, 2].

It affects women more frequently than men; particularly among the elderly. [3] Elderly females are most frequently affected. Because of the rarity of the condition, misdiagnosis occurs frequently and patients are generally diagnosed as having acute cholecystitis [4, 5]. Two types of torsion, incomplete (rotation less than 180°) and complete (rotation more than 180°), are described. According to several authors, the initiator of the torsion would be peristalsis in the transverse colon, duodenum, or gallbladder itself [6].

Diagnosis of the condition is extremely difficult because of the absence of specific clinical or imaging signs; in particular, echography cannot differentiate acute volvulus from other causes of acute cholecystitis. [7] Gallstones may or may be not present. Surgical treatment is technically easy because traction on the mobile gallbladder enables the anatomy of Calot's triangle to be readily identified. This should lead to more laparoscopic management of this condition.

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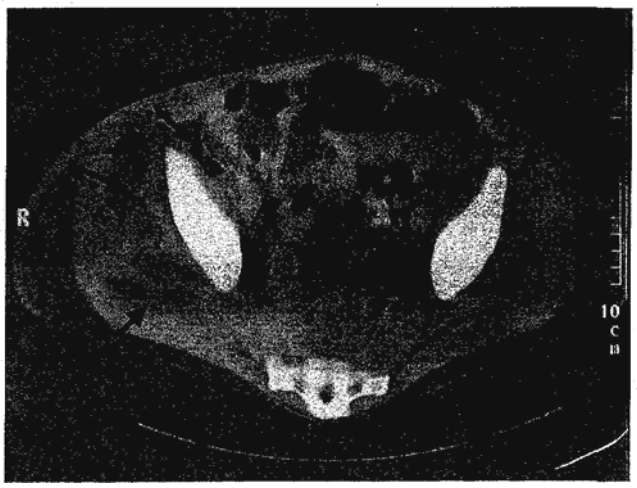

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# المجلة العراقية للعلوم الطبية
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**المقالات**

المجلد السابع، العدد الثالث، ١٤٣٠ هـ، ٢٠٠٩م

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**CONTENTS**

## Case Report

# التنميط الوراثي لمستضدات الخلايا البيض البشرية-الصنف الأول بواسطة تفاعل البلمرة المتسلسل – بتقنية الباديء المعين لسلسلة جينية معينة لمريضات سرطان الثدي العراقيات

احمد عبد الحسن عباس الحسن[1] , نضال عبد المهيمن[1] , علاء غني حسين[2] , أميرة النعمة[3]


## الخلاصة

**خلفية الدراسة :** إن مسببات سرطان الثدي متعددة, فالمهيئات الوراثية ,العوامل البيئية والهرمونية وحتى الخمجية يعتقد بأنها تتداخل لإظهار المرض. في هذا الخصوص, أليلات مستضدات خلايا الدم البيض البشرية تعتبر مهمة كعوامل وراثية مناعية خطرة لكن مصاحبتها للمرض تظهر تكرارية مختلفة باختلاف الشعوب .

**هدف الدراسة:** نظمت هذه الدراسة لتسلط الضوء على احتمالية وجود مصاحبة بين أليلات مستضدات خلايا الدم البيض البشرية (الصنف الأول) مع سرطان الثدي في المريضات العراقيات.

**الأشخاص وطرائق العمل:** تضمنت الدراسة 60 شخص: 30 مريضة مصابة بسرطان الثدي, 12 مريضة مصابة باورام الثدي الحميدة كمجموعة ضابطه أولى و 18 امرأة سليمة ظاهريا كمجموعة ضابطه ثانية. استخدم فحص تفاعل البلمرة المتسلسل – بتقنية الباديء المعين لسلسلة جينية معينة لتقييم تنميط أليلات مستضدات الخلايا البيض البشرية.

**النتائج:** من 95 أليل لمستضدات خلايا الدم البيض البشرية الصنف الأول, أليل واحد (A*03010101-07,09-11N,13-16) اظهر تـ باي ـن معنوي بين المريضات والمجموعتين الضابطتين (مجموعة النساء السويات ومجموعة مريضات أورام الثدي الحميدة) (50% vs. 16.6%, OR=5, EF= 0.40, P= 0.041), (50% vs. 8.3%, OR=11, EF=O.45, P=0.024) نسبة إلى كل منهما.

**الاستنتاجات:** النتائج أظهرت بان الاليل: (A*03010101-07,09-11N,13-16) ربما لعب دور في أحداث المرض.


**مفتاح الكلمات:** سرطان الثدي،مستضدات الخلايا البيض البشرية ، تفاعل البلمرة المتسلسل.

[1] فرع الأحياء المجهرية [ كلية الطب _ جامعة النهرين]

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[3] معهد الطب العدلي

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## تعبير جزيئة CD74 في عدوى Helicobacter pylori للنسيج ألطلائي المعوي

نضال رؤوف مهدي[1] ، نضال عبد المهيمن[2]

## الخلاصة

**خلفية الدراسة :** تُسبّبُ H.pylori التهابا معوياً.الاهتمامات ألحديثه ركزت على دور CD74 والذي هو عبارة عن السلسلة الثابتة المرتبطة بالصنف الثاني للـMHC تظهر على سطح الخلايا الطلائيه المعوية تستعملها H.pylori للالتصاق، وقد تسهم في بدءا لتفاعلات المناعية الالتهابية.

**هدف الدراسة :** هدف هذه الدراسةِ هو الكشف عن تعبير CD74 في النسيج المعوي عند حصول عدوىH.pylori ومُقارنُتها مَع نماذج من المرضى الغير مُصابينِ بتلك البكتريا.

**المرضى وطرق العمل :** شملت الدراسة 64 مريض بعمر14 -66 سنه (متوسط العمر 34 سنه) تم فحصهم بناظور الجزء العلوي للمعدة والأمعاء بسب وجود اعتلال في منطقه المعدة والأمعاء.لتشخيص ألاصابه بال H.pylori استخدم عدد من ألاختبارات منها المتداخلة ( اختبار أنزيم الureaseالسريع جدا و فحص المسحة ألمضغوطة على السلايد) وغير المتداخلة ( ELISA )

بالإضافة إلى دراسةِ التحليل المناعي النسيجي الكيميائي (IHC ) لفحص العينات،والكشف عن تعبير جزيئه CD74 ألنسيج ألطلائي المعوي.

**النتائج :** بَعْدَ تشخيص الاصابه بال H.pylori؛ قسم المرضى إلى مجموعتين (مجموعه مصابه بال H.pylori وعددها 47) و(مجموعه سلبيه للبكتريا وعددها 17 ).، وطبقاً للدراسةِ التحليل المناعي النسيجي الكيميائي للمقاطع ألنسيجيه فأن تعبير جزيئه CD74 لوحظَ في المقاطع المُصَابةِ؛ وكان هناك فرق هامّ في التعبيرِ ($p = 0.005$) بين أنسجة المرضى المُصَابينِ والغير المُصابينِ بالبكتريا.

**الاستنتاجات :** طبقا لدراسة التحليل المناعي النسيجي الكيميائي لفحص العينات، هنالك تعبير فائق لظهورCD74 في العينات المصابة بالبكتريا.

**مفتاح الكلمات :** Helicobacter pylori؛ألتحليل المناعي ألنسيجي ألكيميائي (IHC) ؛ CD74 ؛ الخلايا الطلائيه المعوية.

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[2] فرع الأحياء ألمجهريه [كليه الطب –جامعه النهرين]

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# التحري عن الأجسام المضادة للنطف في مصل دم الذكور و الإناث العراقيين وعلاقتها بالقدرة الإخصابية

محمد باقر محمد رشاد فخر الدين , سندس فاضل حنتوش


## الخلاصة

**خلفية الدراسة:** إنَ المسبب الرئيس للعقم المناعي هو وجود الأجسام المضادة للنطف حيث تجعل حركة النطف بطيئة جدا" و تتداخل بعملية الإخصاب و قد تتسبب بالإجهاض .

**هدف الدراسة:** تهدف هذه الدراسة إلى التحري عن الأجسام المضادة للنطف في مصل المرضى من كلا الجنسين و بيان دورها في التسبب في العقم .

**طريقة العمل:** تم التحري عن هذه الأجسام بإجراء الاختبار المناعي غير المباشر . استخدمت أجسام معلمة للأجسام المضادة للنطف . استخدمت صبغة الإيفانس الزرقاء كصبغة مضادة . كشف عن هذه الأجسام باستخدام مجهر الفلورسنت . اشتملت الدراسة على ثلاثة و سبعين مراجعا" لمعهد أبحاث الأجنة و علاج العقم (ستين رجلا" و ثلاثة عشر امرأة ) . تم إجراء اختبار تحليل السائل المنوي لستين رجلا. تم إجراء الفحص المجهري المباشر للمسحات المهبلية لثلاثة عشر امرأة .

**النتائج:** أظهر أربعون رجلا"(75%) و عشر نساء(76.9%) وجود هذه الأجسام في مصلهم و كانت المعيارية لنسبة وجودهم هي إما 10/1 او 32/1 مما استوجب خضوعهم إلى العلاج . استخدم عقار البردنوسولون كعقار فعال ضد هذه الأجسام .

**الاستنتاج :** تلعب الأجسام المضادة للنطف في مصل مرضى العقم دورا" مهما" في التسبب بالعقم مما يستوجب العلاج .


**مفتاح الكلمات :** مصل الدم، الأجسام المضادة للنطف، عدم الخصوبة، المناعة.

معهد أبحاث الأجنة و علاج العقم / جامعة النهرين

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص19-23

# المقارنة بين الكاردينلا المهبلية والتهاب المهبل الفطري (المونيليا) وعلاقتهما بمستوى الاستروجين في الدم وحامضية المهبل في بعض النساء العراقيات العقيمات

رحاب شفيق عبد السيد[1]، إكرام أمين خاكي [2]، صباح النجار[2]


## الخلاصة

**خلفية الدراسة:** تُعرّض خصوبة النساء لعده عوامل تتضمن العوامل الجرثومية وغير الجرثومية . الإصابات الجرثومية هي واحده من أهم مسببات العقم في النساء، مستوى أمراضيه الاصابه الجرثومية تتأثر بمدى واسع من العوامل والتي تتضمن العمر والحالة الفسلجية (طور الدورة الشهرية) والعرق أو الجنس.

**هدف الدراسة:** هدفت هذه الدراسة إلى المقارنة بين الإصابة بالكاردينلا المهبلية والتهاب المهبل الفطري (المونيليا) وعلاقتهما بمستوى الاستروجين في الدم وحامضيه المهبل في بعض النساء العراقيات العقيمات

**طرق العمل :** نفذت هذه الدراسة على 109 امرأة عقيمة راجعن معهد أبحاث الأجنة وعلاج العقم في جامعة بغداد للفترة من حزيران حتى تشرين الثاني لعام 2004 . تلك النساء خضعن للفحص السريري وقياس حامضيه المهبل كما وجُمعت منهن المسحات المهبلية لغرض تشخيص الإصابة بالكاردينلا باستخدام معيار Amsel السريري مع عدد من طرق التشخيص الجرثومية. كما تم تشخيص الإصابة بالمونيليا باستخدام طرق تشخيص الفطريات. بالإضافة إلى ذلك تم جمع عينات المصل بواسطة سحب الدم من النساء وهّن في الطور الجريبي المتأخر لغرض معرفه مستوى الاستروجين.

**النتائج:** ثمانية وأربعون امرأة عقيمة ُشخّصت بأنها مصابه بالكاردينلا المهبلية من 109 امرأة عقيمة وكان معدل الاستروجين في هذه النساء حوالي 41.17 بيكوغرام /مل قريب من الحد الأدنى للمعدل الطبيعي لمستوى الاستروجين (18-147 بيكوغرام /مل) واقل من معدل الاستروجين لنساء مجموعه السيطرة 13.25 بيكوغرام /مل وفي هذه الدراسة كانت حامضيه المهبل لحوالي 93.75% من تلك النساء أكثر من 4.5.

شُخّصت أربعه وعشرون امرأة عقيمة بأنها مصابه بالمونيليا المهبلية من 109 امرأة عقيمة وكان معدل الاستروجين لديهن 183.2 بيكوغرام /مل أعلى من الحد الأعلى للمعدل الطبيعي لمستوى الاستروجين وأعلى من معدل الاستروجين لمجموعه السيطرة , وان الإصابة بالمونيليا المهبلية قد حدثت في مدى حامضيه المهبل الطبيعية التي تتراوح بين 3.5-4.5 .

**الاستنتاج :** أظهرت نتائج هذه الدراسة بان الاضطراب الهرموني الذي يكون مرافق لمختلف حالات العقم عند النساء قد يكون المهيئ للإصابة بالكاردينلا المهبلية والمونيليا المهبلية بين النساء العقيمات، وان ارتفاع حامضيه المهبل بين النساء العقيمات المصابات بالكاردينلا المهبلية قد يكون نتيجة نقص الاستروجين، بينما الحامضيه المهبلية تكون طبيعيه في النساء المصابات بالمونيليا المهبلية لان الاستروجين يزيد محتوى الكلايكوجين الخلوي الذي يزيد من نمو عصيات حامض اللبنيك التي تؤيض الكلايكوجين الى حامض اللبنيك منتجا المحيط الحامضي.


**مفتاح الكلمات:** الكاردينلا المهبليه ، المونيليا ، مستوى الاستروجين ، النساء العقيمات.

[1] معهد أبحاث الأجنة و علاج العقم / جامعة النهرين

[2] فرع الأحياء المجهرية [ كلية الطب _ جامعة بغداد ]

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص24-31

# بعض النواحي التشخيصية لمرض داء الزلاق البطني (حساسية الحنطة) لدى الأطفال في العراق

هالة سامح عارف[1] , راجي الحديثي[2], سرمد عبد الاله[2]

**الخلاصة:**

**خلفية الدراسة:** هناك ازدياد ملموس لوجود مرض الزلاق البطني في مختلف أنحاء العالم, والبحوث مستمرة لتلافي المشاكل التشخيصية لهذا المرض.

**هدف الدراسة:** دراسة مجوعة من الأطفال ذوي داء الزلاق البطني والمشاكل التشخيصية لهذا المرض.

**طرق العمل :** تم تقصي 93 حالة من الأطفال الذين كانوا يعانون من مرض سوء الامتصاص في الأمعاء, بواسطة التحاليل السيرولوجية وخزعة االأمعاء الدقيقة, لوجود مرض حساسية الحنطة.

**النتائج:** 58 من 93 من المرضى وجدوا مصابين بمرض حساسية الحنطة حسب الفحص النسيجي لخزعة الأمعاء الدقيقة. تراوحت حساسية الفحوص السيرولوجية لوجود المرض بين 50 – 70 %. المرضى الذين كانوا يعانون من تغيرات نسيجية شديدة أعطوا ايجابية أكثر في الفحوص السيرولوجية مع مستويات أعلى من الأجسام المضادة في هذه الفحوص من سواهم.

وجدت أحدى عشر حالة من عينة المرضى الكلية , مصابون بطفيلي الجيارديا, وأعطى العض منهم تغيرات متفاوتة في الفحص النسيجي والفحوص السيرولوجية.

**الاستنتاجات:** هناك وجود ملحوظ لمرض حساسية الحنطة في العراق. نشترك مع الآخرين في المعاناة من وجود بعض المشاكل التشخيصية, بالإضافة لوجود مشاكل أخرى في التشخيص تخص المرضى في الدول النامية, مما يدعو لإيجاد إستراتيجية خاصة للتشخيص في هذه المناطق.

مفتاح الكلمات: مرض الزلاق البطني, الفحوص السيرولوجية , الأمراض النسيجية.

[1] فرع طب الأطفال [ كلية الطب _ جامعة النهرين]

[2] فرع علم الأمراض [ كلية الطب _ جامعة النهرين]

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص32-39

قياس عدد الصفيحات الدموية بالاعتماد على نسبة كريات الدم الحمر إلى صفيحات الدم

باسم فاضل عبد صالح

## الخلاصة

**خلفية الدراسة:** إن الأجهزة التحليلية الحديثة المستعملة في مجال علم أمراض الدم تمتاز بقدرتها على إعطاء نتائج دقيقة ومحددة لعدد الصفيحات الدموية، لكن في بعض الحالات هذه الأجهزة تعطي نتائج مضللة وغير دقيقة لذلك يجب قياس عدد الصفيحات الدموية بالاعتماد على فحص مسحة الدم والذي يجب أن يكون من الفحوص المختبرية الروتينية خصوصا إذا كانت نتائج الأجهزة المختبرية التلقائية العمل مضللة، ولكن بالمقارنة مع هذه الأجهزة فإن الفحص المختبري لمسحة الدم باستخدام الطريقة التقليدية (حجرة نيوباور التعدادية) يتطلب الكثير من الوقت والجهد لذا فهو أكثر تكلفة اقتصاديا.

**هدف الدراسة:** تقييم مدى صلاحية وموثوقية تقنية قياس عدد صفيحات الدم بالاعتماد على قياس نسبة كريات الدم الحمر إلى صفيحات الدم في مسحات الدم المصبوغة.

**المواد وطرق العمل:** خلال الفترة من كانون الثاني إلى آذار 2006 تم قياس عدد الصفيحات الدموية لمائة عينة من الدم في المركز الوطني لأمراض الدم باستخدام طريقتين مختبريتين: الأولى بالاعتماد على جهاز قياس الخلايا التلقائي والثانية بالاعتماد على الطريقة اليدوية لقياس صفائح الدم باستخدام المجهر الضوئي وذلك بعد عد الصفائح الموجودة في المسحة الدموية المصبوغة لكل ألف كرية دموية ومن ثم ضربها بعدد كريات الدم الحمر المقاسة باستخدام الأجهزة المختبرية التلقائية الحديثة.

**النتائج:** كانت نتائج فحص مسحات الدم باستخدام الطريقة اليدوية كالآتي: تراوح عدد الصفائح الدموية للعينات المقاسة بين 100-499×103/مايكروليتر وكان معدل عددها 263,11± 104,07 ×103/مايكروليتر. في حين كانت النتائج باستخدام الأجهزة المختبرية لنفس العينات كما يلي: تراوحت أعداد الصفائح الدموية بين 95-484 ×103/مايكروليتر ومعدل عدد الصفائح كان 258,43 ±103,13 ×103/مايكروليتر.

أظهرت النتائج عدم وجود فارق إحصائي معتد به بين عدد الصفائح الدموية المقاسة باستخدام الأجهزة المختبرية التلقائية وتلك المقاسة بالطريقة اليدوية بالأعتماد على نسبة كريات الدم الحمر إلى صفيحات الدم ($P<0.05$)

**الاستنتاج:** قياس عدد الصفيحات الدموية بالاعتماد على نسبة كريات الدم الحمر إلى صفيحات الدم هي طريقة معتد بها وتحتاج فقط إلى قياس دقيق لعدد كريات الدم الحمراء باستخدام الأجهزة التحليلية المختبرية, حيث تساعد هذه الطريقة على قياس عدد الصفائح الدموية بدقة اكبر وفي نفس الوقت تستهلك جهدا ووقتا أقل مما تتطلبه الطريقة التقليدية لقياس عدد صفائح الدم باستخدام حجرة نيوباور التعدادية.

**مفتاح الكلمات**: تعداد الصفيحات الدموية، كريات الدم الحمر، نسبة الصفيحات الدموية

فرع علم الأمراض [ كلية الطب _ جامعة النهرين]

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص40-45

## انتشار مرض الحصبة في العراق –الكاظمية لسنة 2008-2009 واهم مضاعفاته

عبد الكريم جاسم محمد البهادلي , أريج عبد العباس

## الخلاصة

**خلفية الدراسة:** الحصبة مرض انتقالي حاد، واسع الانتشار في سن الطفولة، يتسبب عن الإصابة بفيروس الحصبة، ويتميز بارتفاع في درجة الحرارة مصحوب برشح وسعال ورمد، ويتبع ذلك طفح على جميع أجزاء الجسم.

**هدف الدراسة:** لغرض معرفة حجم انتشار مرض الحصبة في مدينة بغداد- الكاظمية مع دراسة أسباب هذا الانتشار واهم المضاعفات التي رافقت المرض.

**طريقة البحث :** الدراسة شملت 494 مريض تم تشخيصهم سريريا في كل من مستشفى الكاظمية التعليمي , مستشفى أطفال الكاظمية ومركزين من مراكز الرعاية الصحية الأولية في الكاظمية وتم تقسيمهم إلى أربع مجاميع حسب الفئات العمرية (اقل من سنه واحده-من سنه إلى أربع سنوات –من خمس سنوات الر تسع سنوات- عشر سنوات فما فوق ) ,وتمت معرفة حالة التحصين ضد مرض الحصبة من خلال فحص سجل التحصين الخاص بالمريض أو من خلال الاستفهام من الوالدين ,إضافة إلى اعتماد المعايير الطبية السريرية من اجل تسجيل المضاعفات.

**النتائج :** أظهرت الدراسة معدل الإصابة عند الذكور (49.80 %) يكاد يكون مقارب للإصابة عند الإناث (50.20 % ) ,وكان عدد المصابين بالمرض بحسب الفئات العمرية كالآتي:
الأطفال الذين تقل أعمارهم عن السنة 97 (19.64 % ) ,من عمر سنة إلى أربع سنوات 287 (58.97 % ) مما يدل على أن عدد الأطفال المصابين بالمرض والذين تقل أعمارهم عن الأربع سنوات 384 (78.61 % ) ,كانت نسبة الأطفال المحصنين ضد المرض (10.53 % ) ,أظهرت الدراسة أن أهم المضاعفات التي رافقت المريض كانت كالآتي , ذات الرئة ,إسهال مع تقيؤ ,التهاب الحنجرة والتهاب الدماغ وحسب النسب الآتية ( 83.85 % ) ,(11.46 % ) ,(2.60 % ) و(2.09 % ) لكل واحد منهما وحسب التسلسل وكانت نسبة الوفيات (2.43 % ) والتي حدثت معظمها في الأطفال الذين تقل أعمارهم عن الأربع سنوات (91.7 % ) وهناك زيادة طفيفة في نسبة الوفيات عند الذكور (58.4 % ) مقارنة بالإناث (41.6 % ) ز

**الاستنتاج :** الفئات العمرية التي تقل أعمارها عن الأربع سنوات هي أكثر إصابة بمرض الحصبة الذي كان انتشاره بسبب قلة التحصين ضد المرض واهم وأكثر المضاعفات هو مرض ذات الرئة كما أن نسبة الوفيات كانت نسبة قليلة بصورة عامة .

**مفتاح الكلمات :** الحصبة , التحصين, انتشار, مضاعفات, أطفال .

فرع طب الأطفال [كلية الطب _ جامعة النهرين ]

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص46-51

## تأثير إضافة وسط تحضير النطف معززاً بفيتامين E أو فيتامين C على مواصفات السائل المنوي لمرضى العقم المصابين بقلة حركة النطف

باسم خميس كوتي

## الخلاصة

**خلفية الدراسة:** إن المستوى الطبيعي للجذور الحرة في السائل المنوي يمكن تحديده عن طريق ميكانيكية عمل مضادات الأكسدة مثلاً فيتامين C أو فيتامين E والتي تكون موجودة بشكل طبيعي في بلازما المني والغشاء البلازمي للنطفة البشرية. أن عملية تعزيز عينة السائل المنوي لمرضى العقم بمضادات الأكسدة كفيتامين C أو فيتامين E يمكن أن يكون له دورٌ كبيرٌ في علاج الكثير من حالات العقم الذكري.

**هدف الدراسة:** تهدف الدراسة إلى تحديد تأثير إضافة فيتامين C أو فيتامين E إلى وسط تحضير النطف باستخدام التقنية الطباقية البسيطة على مواصفات السائل المنوي لمرضى المصابين بقلة حركة النطف.

**طرق العمل:** تم اخذ 60عينة سائل منوي وكل عينة قسمت إلى قسمين وكل قسم يحتوي على ( 0.5 ml) من السائل المنوي وبعد ذلك أضيف (0.75 mg/ml) من فيتامين C إلى القسم الأول (العدد:20عينة) و (0.75 mg/ml) من فيتامين E إلى القسم الثاني (العدد:20عينة) إما مجموعة السيطرة (العدد:20عينة) فتم تحضيرها بإضافة وسط تحضير النطف فقط لأجراء عملية تنشيط النطف خارج الجسم اعتماداً على مقررات منظمة الصحة العالمية (WHO).

**النتائج:** لوحظ إن فحوصات كفاءة النطف والتي تتضمن تركيز النطف, حركة النطف, الحركة التقدمية للنطف, النسبة المئوية للنطف السوية تحسنت بشكل كبير وأعطت ارتفاعاً معنوياً عالياً بعد إضافة فيتامين C أو فيتامين E إلى وسط تحضير النطف مقارنة بمجموعة السيطرة.

**الاستنتاج:** تم الاستنتاج من خلال الدراسة أن إضافة فيتامين C و فيتامين E يودي إلى تغيير واضح في وظائف النطف بعد إجراء عملية التنشيط, ولكن فيتامين C يعد الأحسن في أعطاء أفضل النتائج ويمكن أن يؤثر على معدلات الحمل بعد استعماله في التقنيات المختبرية المساعدة على الإنجاب.


**مفتاح الكلمات:** مضادات الأكسدة, فيتامين C, تقنيات تحضير النطف, متلازمة قلة حركة النطف

فرع علوم الحياة [كلية العلوم _ جامعة ذي قار ]

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص52-58

## المشكلات المصاحبة للإسهال في الأطفال في بغداد – العراق

عبد المنعم ناجي محمد،


## الخلاصة

**خلفية الدراسة:** سجلت أجناس الشيكلاهي البكتريا الشائعة الثانية كعامل مسؤول عن إسهال الأطفال بعد E.coli. حالياً ,عزل هذه البكتريا وتأكيد تشخيصها تم بواسطة الطرق البكتريولوجيه والكيمياء الحياتية وتبقى هذه الطرق المقياس الذهبي

**هدف الدراسة:** لتحديد انتشار أجناس الشيكلا بين الأطفال دون 3 سنوات والمصابون بالإسهال الحاد وفحص حساسية هذه العزلات إلى مضادات الجراثيم الشائعة الاستعمال .

**طرق العمل:** أجريت هذه الدراسة في العيادة الخارجية لمستشفى الأطفال المركزي التعليمي في بغداد من مايس 2007 إلى نيسان 2008 .
مائه وخمسون طفل دون 3 سنوات في حاله إسهال حاد ، كانوا مصدرا لنماذج الخروج لتحديد أجناس الشكيلا فيها،وشخصت كل العزلات طبقا إلى الطرق المعيارية البكتريولوجيه والكيمياء الحياتية،واستعملت مضادات الجراثيم المتوفرة لتحديد حساسية العزلات للأدوية .

**النتائج:**أن (Shigella flexneri type 2 ) هي السائدة في فحص نوع المصل من بين جميع العزلات التسعة . وكان انتشار عزلات الشكيلا عاليه الوضوح في الأطفال الذين أعمارهم أكثر من سنه واحده.
كل المرضى كانوا على الإطعام الاصطناعي،(78%) منهم استعملوا الماء الغير ألمعالج للشرب.
أن جميع العزلات كانت حساسة إلى.(Ceftriaxone,Ciprofloxacin, &Gentamicin Nalidixic Acid, Norfloxacin) ومقاومتها للدواء, لثلاثة أدويه أو أكثر وجد في (56%) من العزلات.

**الاستنتاج والتوصيات:** (Shigella flexneri type ) هي السائدة في فحص نوع المصل ومعظم العزلات كانت مقاومه إلى Trimethoprim – sulphamethoxazole ( 89%)،وهكذا لا يعد اعتبار لهذا الدواء في معالجه الشيكلات المصاحبة للإسهال في عينه لاطفا ل العراق دون 3 سنوات.

**مفتاح الكلمات:**الشيكلات المصاحبة للإسهال في الأطفال ،المضادات الحيوية.

**فرع الأحياء المجهرية [ كلية الطب _ الجامعة المستنصرية]**

**المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص59-65**

## إزالة الوشم باستخدام ليزر 1064 و 532 نانومتر كيو سوج – اندي ياك

**فاطمة عدنان محمد علي , علي شكر محمود**

**الخلاصة**

**خلفية الدراسة:** إن الوشم عادة من أقدم الممارسات التي توارثتها الشعوب, فقد عثر على مومياء فرعونية تحمل اثر لأكثر من وشم عليها.وتوجد طرق قديمة لإزالة الوشم قدم الوشم ذاته ولكن اغلب هذه الطرق بدائية تؤدي إلى حرق الجلد, تشوهات فيه, تغير لون الجلد, تثخن الجلد .وقد ثبت حديثاً إن اسلم طريقة لإزالة الوشم هي استخدام الليزر (كيو سوج) وذلك لقلة تأثيره على الجلد ولعدم حصول عوارض على البشرة.

**هدف الدراسة:** إن هدف هذه الدراسة هو تقييم جهاز الكيو سوج انديك 1064,532 نانو متر في علاج الوشم.

**المواد والطرق:** في هذه الدراسة تم أخذ (99) ,وشم من كلى الجنسين ومن أعمار تتراوح بين 20-60 سنة. وقد تم تشخيص وتقسيم الوشم إلى ألوانه المختلفة حسب المستخدم منها وهي الأخضر والأزرق والأحمر والأسود و كلا منها قسم إلى غير حرفي يتم على يد أشخاص غير متخصصين و حرفي يتم على أيدي أشخاص مهنيين متخصصين . أستخدم في الدراسة جهاز الليزر (كيو سوج- اندي ياك), ذو الطول ألموجي (1064) نانو مترو تردد(5)هيرتز, شدة الإشعاع تراوحت بين (6.3- 10.1) جول/سم2 , حجم البقعة (3) مليمتر, عرض النبضة (10) نانو سكند للون الأخضر والأزرق والأسود. أما اللون الأحمر فقد أستخدم الطول الموجي (532)نانو ميتر, التردد(10) هيرتز, شدة الإشعاع (7.3-10.3) جول/سم2, حجم البقعة (2) مليمتر. كان العلاج يتم بواسطة جلسات, كل جلسة تستغرق من (2-5) دقائق حسب حجم الوشم, كذلك وجود فترة زمنية بين جلسة وأخرى, تتراوح بين (3-4) أسابيع .

**النتائج:** كانت استجابة اللون الأسود جيدة مع وجود ظل خفيف جدا مقبول تجميلياً, أما الأزرق فقد استجاب, لكن يوجد ظل خفيف, أما اللون الأخضر فلم يستجب إلا مريض واحد, بدأ بالاستجابة في الجلسة التاسعة, ذلك بتخفف اللون قليلاً جداً..وكانت الاستجابة جيدة جداً. وقد لوحظ من خلال تقسيم المرضى إلى وشم حرفي وغير حرفي, إن استجابة الوشم غير حرفي أسرع عند المرضى, كونه قريباً من سطح الجلد, واكبر حجماً من الحرفي واقل قيمة.

**الاستنتاج:** يمكن اعتبار الليزر علاج معياري اعتمادا على اللون لمن يريد إزالة الشم.

**مفتاح الكلمات:** وشم, ليزر, ,حرفي وغير حرفي.

معهد الليزر للدراسات العليا / جامعة بغداد.

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص66-81

## متلازمة عسر التنفس وعلاقتها بالعمليات القيصرية التي تجرى في مستشفى الكاظمية التعليمي

أريج عبد العباس

## الخلاصة

**خلفية الدراسة** : تعتبر متلازمة عسر التنفس واحدة من أهم أسباب دخول الأطفال ألحديثي الولادة إلى وحدة الخدج وحديثي الولادة والتي تحدث بسبب نقص مادة السيرفكتنت التي يؤدي نقصها إلى قله التبادل الغازي داخل الحويصلات الرئوية ,وهي تحدث نتيجة أسباب عديدة منها الولادة المبكرة ,مرض السكري عند الأم الحامل والعمليات القيصرية

**هدف الدراسة** :لمعرفة العلاقة بين حدوث متلازمة عسر التنفس و العمليات القيصرية التي تجرى في مستشفى الكاظمية التعليمي

**طريقة الدراسة** :تضمنت هذه الدراسة التي أجريت في مستشفى الكاظمية التعليمي للفترة من اليوم الأول لشهر كانون الثاني لسنة 2009 إلى الثلاثون من شهر نيسان من نفي السنة ,372طفل حديث الولادة مصاب بمتلازمة عسر التنفس والذين تتراوح مدة الحمل بهم من 37 إلى 40 أسبوع وكانت أوزانهم أكثر من 2,5 كغم حيث تم تقسيمهم إلى مجاميع حسب نوع الولادة وهي ولادة طبيعية أو قيصريه

**النتائج** : أظهرت الدراسة إن نسبة الإصابة عند الذكور (14,1 % )أعلى من نسبة الإصابة عند الإناث(9,2 % ), وأظهرت الدراسة كذلك إن الإصابة بمتلازمة عسر التنفس عند الأطفال الذين يولدون بواسطة العمليات القيصرية (18,5 %) أكثر بكثير من الأطفال الذين يولدون بواسطة الولادة الطبيعية وهناك أيضا زيادة نسبة الإصابة عند الأطفال ألحديثي الولادة والذين تقل أوزانهم عن 3 كغم

**الاستنتاج** :الولادة الطبيعية هي واحدة من أهم العوامل التي تقلل نسبة الإصابة بمتلازمة عسر التنفس وكذلك فا ن نقص وزن الطفل يشكل احد العوامل المؤدية للإصابة بها

**مفتاح الكلمات** :الولادة الطبيعية ,العملية القيصرية ,متلازمة عسر التنفس

فرع طب الأطفال [ كلية الطب _ جامعة النهرين]

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص82-87

## عمليات إعادة تجميل الاطليل التحتي: تجربة مع 27 مريض لديهم فشل عملية تجميل الاطليل التحتي من النوع البعيد أو القريب

علا محمد الكواز

## الخلاصة

**خلفية الدراسة:** إن عمليات إعادة تصنيع الاحليل بعد فشل عملية مبال تحتاني سابقة تنطوي على تحد مميز.نعرض تجربتنا بتجميل الاحليل من خلال طريقة سنودجراس بالنسبة للأنواع البعيدة من المبال التحتاني وطريقة قص الناسور مع استعمال طبقة الصفن الليفية في الأنواع القريبة للمبال التحتاني.

**هدف الدراسة :** بأثر رجعي استعراض تجربتنا في سلسلة من عمليات إعادة اصلاح المبال التحتاني للأنواع البعيدة والقريبة منه والتي سبق ان فشل فيها عملية إصلاح سابق.

**المواد وطرق العمل:** بين كانون الأول / ديسمبر 2006 ويونيو 2009 ، 27 طفلا (متوسط العمر 4.8 يتراوح بين سنتين إلى 11 سنة) أُحيلوا لعملية إعادة تصليح المبال التحتاني. المرضى قُسّموا إلى مجموعتين؛ المجموعة الأولى (20 مريض) للمبال التحتاني للنوعين البعيد والمتوسط في هذه الحالات، عملية تجميل الاحليل بطريقة Snodgrass والتي فيها غُطّى الاحليل الجديد بطبقةِ إضافيةِ مِنْ النسيجِ التحت الجلدي. أما المجموعة الثانية (7 مرضى) المبال التحتاني من النوع الأدنى الذي يحوي مضاعفات العمليات السابقة بتشكيلِ الناسور،عملية إعادة التصليح وذلك بقطع منطقةِ الناسورَثم بالإغلاقِ بالخياطاتِ المُقَاطَعةِ وعمل غطاء طَبقةِ ثانيةِ مِنْ النسيجِ التحت الجلدي.

**النَتائِج:** المجموعةِ الأولى كان هناك 5 (25 %)، مضاعفات والتي شملت رجوع تضيّقَ فتحة الاحليل في 3 مرضى، تضيّق واحد مع ناسورِ الصغيرِ وواحد فتح كلي للجرح. إعادة عمليةِ كَانتْ ضروريةَ في فقط واحد مِنْ مريضِ سلسلتِنَا (7.6 %) والآخرون عولجوا بالتوسُّيعِ لفتحة الاحليل.

المجموعة الثانية: مرضى 2 من 7 (28.5 %) كَانَ عِنْدَهُمْ فشلُ التصليحِ ببقاءِ الناسورِ الذي تَطلّبوا إعادة العمليةِ

**الاستنتاج :**بالنسبة للنوعِ الأقصى من المبال التحتاني عملية تجميل الاحليل كما هو مَوْصُوف من قبل Snodgrass هي طريقة مناسبة لعلاج حالات المبال التحتاني من النوع الأقصى بالنسبة للحالات الأساسية أو إعادة العملية. في حين في حالات النوع الأدنى من المبال التحتاني فان إغلاق منطقة الناسور وتغطيتها بطبقة ثانية لمنع الإصابة بالناسور يعد خيارا مقبولا للعلاج .

**مفتاح الكلمات :** تجميل الاحليل ,مبال تحتاني, اللوحة الأحليلية.

فرع الجراحة [ كلية الطب _ جامعة النهرين]

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص88-93

# دراسة صفات اجتماعية وديموغرافية للمرضى المصابين بأمراض نزف الدم الوراثية

ايناس طالب عبد الكريم[1] , سعد فاضل محمد[2]

## الخلاصة

**خلفية الدراسة:** المرضى المصابين بالنقص الوراثي لعوامل التخثر في البلازما يعانون من نزف متكرر مدى الحياة في المفاصل, العضلات، والأماكن المغلقة في الجسم أما بشكل أني أو بعد التعرض لجرح

**هدف الدراسة :** تهدف هذه الدراسة إلى بيان الواقع الاجتماعي والديموغرافي للمرضى المصابين بأمراض نزف الدم الوراثية المراجعين لمركز الأمراض النزفية الوراثية في مستشفى المنصور التعليمي للاطفال في بغداد.

**طريقة الدراسة :**أجريت دراسة مقطعية في الفترة مابين الأول من شهر آذار لسنة ٢٠٠٨م ونهاية شهر آب لسنة ٢٠٠٨م وكانت عينة الدراسة تتألف من 243 مريضا مصابين بمختلف الأمراض النزفية الوراثية. تضمنت البيانات استبيان منظم مليء فقط من قبل الباحث بواسطة الاستجواب للمريض أو والديه أو ذويه المقربين.

**النتائج:** أظهرت الدراسة الحالية مايلي:

المرضى المصابين بالهيموفيليا ومرض فون ويلليبراند يشكلون ٩٠,١٪ من عينة الدراسة ، كان معدل العمر لمرضى العينة حوالي ١٤سنة، كان حوالي ٧٧٪ من عينة الدراسة من سكنة محافظة بغداد. بعض عوائل المرضى ١٥,٦٪ لديهم ثلاثة أفراد أو أكثر يعانون من احد الأمراض النزفية الوراثية. معظم حالات الهيموفيليا التي راجعت مركز الأمراض النزفية الوراثية كانت أما شديدة أو متوسطة الشدة ٦٦,٥٪ و ٢٢,٥٪ على التوالي. بالنسبة للمرضى الذين أعمارهم ٧ سنوات أو أكثر، فان ٤١٪ منهم لم يلتحقوا بالمدرسة أو تركوا المدرسة بسبب مرضهم الذي يعانون منه. بالنسبة للمرضى الذين أعمارهم ١٨ سنة أو أكثر. كان٦٨,٤ % غير متزوجين، ٤٥,٠٧٪ كانوا عاطلين عن العمل بسبب المرض. العلاجات وخاصة عوامل الدم المصنعة كانت متوفرة لمعظم المرضى( ٩٧,١٪ ).

**الاستنتاج:** كانت الظواهر الاجتماعية والديموغرافية لمرضى النزف الوراثي في الدراسة مشابهة للدراسات الأخرى في منطقة حوض بحر الأبيض المتوسط وبلدان الغرب, فيما عدا معدل العمر في هذه الدراسة كان اقل من الدراسات الأخرى. صنف الدم لم يؤشر بعلاقة ايجابية مع مختلف انواع مرض النزف الوراثي، كما ان الأصابة بالمرض كان له تأثير سلبي على مستوى التعليم عند المرضى

**مفتاح الكلمات :** دراسة مرض النزف الوراثي في مدينة بغداد

[1] فرع طب المجتمع [كلية الطب _ جامعة النهرين]

[2] مدينة الطب _بغداد_العراق

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص94-106

## الردبة الإحليلية بعد قص الاحليل بالناظور " تقرير حالة "

علا محمد الكواز

**الخلاصة**

ان رُدَب الإحليلِ القضيبي الذكرِي هي حالاتَ سريريةً نادرةَ. الرُدَب الإحليلية في الذكورِ قَدْ ترتبط باصابة موضعية،او عدوى، او حصاة الاحليل أو تضيق الاحليل. هنا، نُقدّمُ حالةَ غير عاديةَ لرجل بعمر 55 سنةً مَع ردبة إحليلية عرضية بعد قص الاحليل بالناظور لتفنيدِ التضيق الإحليلي. التصليح الجراحي للحالة المرضية يَتضمّنُ قطعَ تفنيدِي للإحليل، تجميل الاحليل، وإغلاق رقبةِ رُدَب الإحليلِ، المريض بعد العملية يتبولُ جيدا ولكن عِنْدَهُ ضعف في الإنتصابُ لم يستجب للادوية جيدا

**مفتاح الكلمات:** رُدب الإحليل ,تجميل الإحليل ,تضيق الإحليل.

فرع الجراحة [ كلية الطب _ جامعة النهرين ]

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص107-109

# التفاف المرارة عند البالغين/ حالة مرضية نادرة لالتهاب المرارة الحاد

## " تقرير حالة "

علي جليل عواد [1]، حسن هادي السكافي [2]

**الخلاصة**

التفاف المرارة هي حالة نادرة وعموماً بسبب الاختلاف التشريحي غير الطبيعي مثل وجود كيس طويل مع فقدان المرارة للحواف السفلى مع الكبد, والعلامات السريرية تقريباً مشابهة لالتهاب المرارة الحاد . وعلى أية حال التشخيص الصحيح يكون خلال الجراحة (أي خلال إجراء العملية).

**مفتاح الكلمات:** التفاف المرارة , المنظار الجراحي , التهاب المرارة الحاد

[1] فرع الجراحة _ مستشفى الكرامة التعليمي

[2] فرع الجراحة _ مدينة الطب

المجلة العراقية للعلوم الطبية 2009 م المجلد 7 العدد3 ص110-111