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# Duhok Med J

## CONTENTS

### THE CLINICAL UTILITY OF LAPAROSCOPY IN THE MANAGEMENT OF IMPALPABLE TESTES IN PEDIATRIC AGE GROUP

**MUSLIM LAYTH ALABDULLAH** ..... 1 - 8

### FACTOR II PROTHROMBIN (G20210A) MUTATION AMONG HEALTHY INDIVIDUALS IN ERBIL / IRAQ

**FARIDA, F. A. NERWEYI, SALAR S. HUSSAIN, JALADET M.S.JUBRAEL**..... 9-15

### BOTOX INJECTION FOR THE MANAGEMENT OF CHRONIC IDIOPATHIC CONSTIPATION IN CHILDREN

**MOHAMMED H. ALDABBAGH, OSAMA ALMASHHADANI**.....16-23

### MORTALITY OF BURNS IN DUHOK, IRAQ

**KURDO A. QRADAGHI**.....24-31

### VASCULAR ACCESS OUTCOMES IN PATIENTS ON HAEMODIALYSIS IN DUHOK KIDNEY DISEASE CENTER

**ASHUR YOHANNA IZAC, ZANA SIDIQ MOHAMMED SALEEM**.....32-40

### AUTISM SPECIFIC PEDAGOGIC INTERVENTION (ASP); CASE REPORT

**ABDULBAGHI A. AHMAD**.....41-46

### CARDIAC HYDATID CYST WITH MULTIPLE ORGANS INVOLVEMENT

**MUSHEER A. GORAN**.....47-51

### CAROTID BODY TUMOR, SURGICAL RESECTION WITH SAPHENOUS VEIN INTERPOSITION GRAFT: A CASE REPORT

**ABDULLAH S. ABDULLAH**.....52-61

### LIPOID PNEUMONIA AS A COMPLICATION OF MINERAL OIL ASPIRATION: A CASE REPORT

**ISRAA FAWZI AL-DABBAGH, QASEM AHMED AL-SALMI**.....62-67

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## THE CLINICAL UTILITY OF LAPAROSCOPY IN THE MANAGEMENT OF IMPALPABLE TESTES IN PEDIATRIC AGE GROUP

MUSLIM L. ALABDULLAH, MBChB, FIBMS (Ped. Surg.)\*

*Submitted 25 May 2013; accepted 30 Dec 2013*

### ABSTRACT

**Background** Laparoscopy is proposed to play a major role in the management of impalpable testes in pediatrics. The Objectives of the study is to assess the role of laparoscopy in the management of impalpable testes.

**Methods** A prospective study for 49 impalpable testes (46 patients) in which three patients had bilateral impalpable testes. All of these patients underwent diagnostic laparoscopy in the Pediatric Surgery Center at Al-Khansaa Teaching Hospital from the period between July 2008 and January 2012. All these patients were proved to have impalpable testes whether unilateral or bilateral by clinical examination.

**Results** Five testes (10.2%) were palpated after anesthesia so traditional orchiopexy were performed. These patients were excluded from the study. Eleven (25%) from the remaining 44 testes were vanished. 33(75%) testes were identified intraabdominally at different sites, Ten (22.7%) were near the internal inguinal ring and directly fixed, Eleven (25%) were fixed after extensive release of the cord, Seven (15.9%) fixed after cutting the testicular vessels, and Five (11.3%) after staged surgery and these were discharged home a day after surgery. There were no intra operative and immediate post laparoscopic complications. Follow up from six months to two years showed that the testes were located in a normal scrotal position, but four (9%) were smaller than the contralateral ones.

**Conclusions** The laparoscopic management of impalpable testes is the most useful method with excellent results and low morbidity. Moreover, it is quick and easy in dealing with all varieties of impalpable testes.

**Duhok Med J 2013; 7 (2): 1-8.**

**Key words:** Laparoscopy, Testes, Intraabdominal, Anesthesia, Internal inguinal ring

Cryptorchidism is usually a common finding in pediatric practice, it is encountered in 21% of preterm male infants,<sup>1</sup> about 1.8-4% of all full term boys,<sup>2</sup> and 0.8% of 1 year old boys.<sup>1</sup> The percentage of cryptorchid boys with impalpable testes has been reported to vary from 8.3% to 20%.<sup>3</sup> Laparoscopy was introduced as a diagnostic technique for the impalpable testis by Cortesi in 1976,<sup>4</sup> and Scott reported the first series in children in 1982.<sup>5</sup> Before laparoscopy, diagnosis was depends on groin exploration, followed by either laparotomy

or extra peritoneal exploration, However diagnostic laparoscopy can be performed before groin exploration, with minimal addition to the operating time, and without complication, provided that an open technique can be used.<sup>6</sup>

**The aim** of this study is to evaluate the clinical utility of laparoscopy in the management of impalpable testes in pediatric age group.

### METHODS

A prospective study carried out for 49 impalpable testes (46 patients) in which

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three patients had bilateral impalpable testes. All of these patients underwent diagnostic laparoscope at the Pediatric Surgery Center in Al-Khansaa Teaching Hospital of Iraq at the period between July 2008 and January 2012. All these patients were proved to have impalpable testes whether unilateral or bilateral by meticulous repeated clinical examinations. Eight of these patients had ultrasound examination which was requested by the referral doctor so we record these results and compared them with their laparoscopic findings (As we are not send such kind of patients an ultrasound examination ). All of these patients were admitted to the hospital a day before surgery in which complete blood picture, renal function test, and chest X ray were done for them. Intravenous antibiotics also was given (ampiclox) as a single dose before surgery. A period of 4-6 hours fasting was required depending on the age of the patients.

The operation was performed under general anesthesia with endotracheal intubation. After complete relaxation of the child meticulous examination was done to the ipsilateral and contralateral scrotum with examination to the inguinal region of the affected side, if the testis was palpated then traditional orchiopexy was performed, otherwise we proceeded with laparoscopy.

Firstly, before introducing the laparoscopic camera we palpate the bladder and the patient was catheterized only if the bladder was palpable. The patient put in supine position and an incision was made at the abdomen through the umbilicus. After that dissection was done till we reach the peritoneum. Then, 10mm blunt trocar was inserted inside the abdominal cavity. The abdomen was insufflated with carbon dioxide to a pressure around 10-15 mm Hg, and then the abdomen was entered by the laparoscopic camera and inspected to rule out any injury.

If an intra-abdominal testis was identified then another one or two ports were inserted in which the first one in the

contralateral iliac region and the other in the opposite side, then the decision was made to perform either a one-stage orchidopexy or two stages Fowler-Stephens procedure depending on the result of the stretching test which was done by stretching the testis with its cord from the affected side to the other side, if it reach it, then one stage orchidopexy was performed otherwise two stages FS procedure was the choice.

If a single stage laparoscopic orchiopexy was appropriate, the peritoneum overlying the iliac vessels was incised with either scissors or diathermy, this is accomplished taking great care to stay away from the vital structures that can be injured by the laparoscopic instruments, laterally, the peritoneum was also incised to the IIR, then using a combination of blunt and sharp dissection, the peritoneum with the underlying testicular cord were swept toward the pelvis from the initial incisions. At this point, a 10mm incision was made in the ipsilateral scrotum and soft tissue dissection carried out to the level of the pubic tubercle, so a tunnel was created through which the testicle can be transposed from its intraabdominal location into the scrotum where it was secured with 3-0 absorbable suture material.

If a two-stage procedure was the plan, then ligation of the testicular vessels proximal to the testis was done either by two endoscopic clips or endoscopic cautery which followed by a period 6-12 months of observation to allow augmentation of the secondary collateral vasculature to the testis. Then after this period another laparoscopy was done and the testicular vessels were divided either with cautery or by endoscopic scissors. After that, release of the cord was done slightly away from it to avoid damaging the collateral vessels, and lastly orchidopexy was performed to the appropriate position in the scrotum.

After completion of the procedure, inspection of the abdominal dissection

confirms proper hemostasis, closure of the umbilical incision was done by approximation of the umbilical fascia and suturing the skin after removal of the laparoscopic ports then a sterile dressing applied.

Postoperatively the bladder catheter if required was removed at the end of surgery, feeding was started after recovery from anesthesia for the patients who treated by direct inguinal orchiopexy while it was started 6-10 hours for those who subjected to laparoscopic examination.

The follow up was ranged from six months to two years after surgery which includes testes size, position and cosmeses. This was performed as an outpatient visits in a schedule of four visits one week, one month, one year and lastly, two years after surgery.

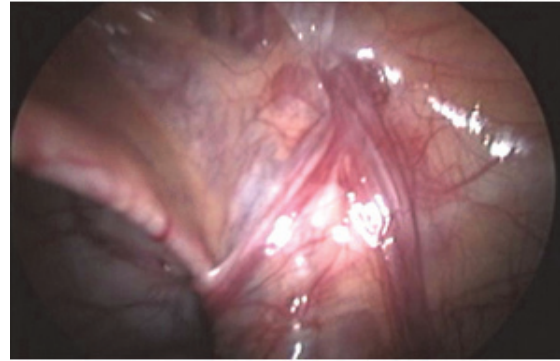
## RESULTS

The surgical findings of the 49 impalpable testes were:

Five (10.2%) from the total 49 testes were palpated in the inguinal region after examining them under general anesthesia, so direct orchiopexy were performed to all of them and the testes were fixed to a normal scrotal position. These were excluded from the study.

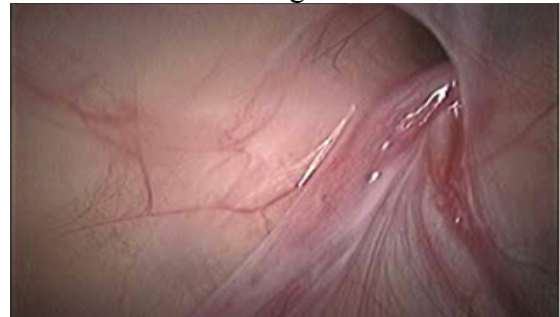
Eleven (25%) from the remaining 44 testes were not identified intraabdominally and rudimentary vas with attenuated testicular vessels which end blindly at a closed Internal Inguinal Ring, inguinal exploration was done for eight (72.7%) testes of the vanished ones and a tissue was taken for biopsy after taking the permission from their families. The result revealed that there was no testicular tissue, so this group is considered as vanishing testes as shown in figure 1.

Thirty three (75%) out of the 44 testes were identified intraabdominally at different locations:



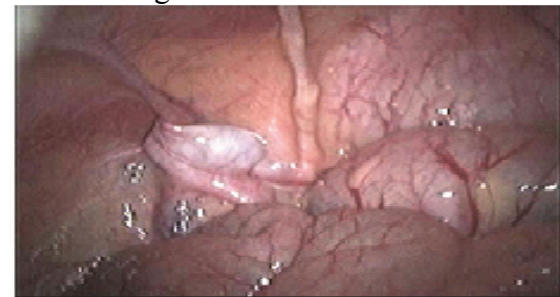
**Figure 1. Vanished Testis.**

Ten (22.7%) testes were identified near the Internal Inguinal Ring (IIR) this group is considered as intracanalicular testes, so direct orchiopexy were performed with no tension as shown in figure 2.



**Figure 2. Intracanalicular Testis.**

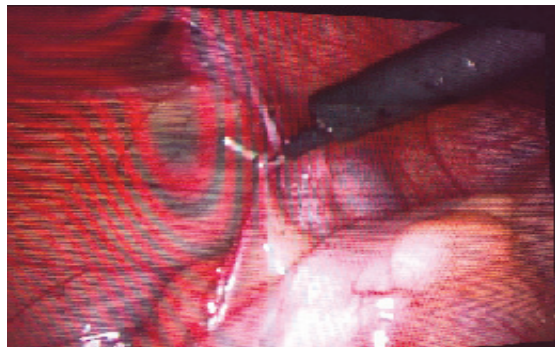
Eleven (25%) testes were identified in the abdomen and fixed to the mid scrotum after extensive intraabdominal release of the cord from the peritoneal reflection as shown in figure 3.



**Figure 3. Extensive intraabdominal release of the cord from the peritoneal reflection.**

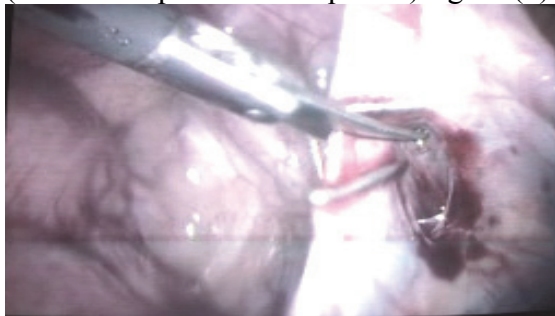


Seven (15.9%) were identified in the abdomen proximal to the IIR with short vessels so extensive dissection from the peritoneal reflection was done with division of the testicular vessels and the testes were fixed to upper scrotum depending on the vas vasculature as the main blood supply of the testes as shown in figure 4.



**Figure 4. Division of the testicular vessels.**

Five (11.3%) also were identified intraabdominally with very short vessels (stretching test was negative), so ligation of these vessels were done between two clips just proximal to the testis and then after 6 months another laparoscope was performed and fixation of these testes were done to mid scrotum with no tension (Fowler-Stephens technique FS) figure (5).



**Figure 5. Ligation of testicular vessels was done by two clips and then after 6 months cut between these clips (Fowler-Stephens technique FS).**

The patients who were treated by direct orchidopexy were discharged from the hospital immediately after recovery from anaesthesia. Those who were treated laparoscopically were discharged on day one following surgery.

There were no intra operative complications and no immediate post laparoscopic complications for all the treated impalpable testes, and after follow up ranging from six months to two years the testes were located in a normal scrotal position, but four (9%) of these testes were smaller than the contralateral ones.

## DISCUSSION

In the present study five testes were palpated at the inguinal region after anesthesia in which all of these patients were obese so direct orchiopepy was performed. This result is similar to another study done by El-Gohary which stated that 19 out of 189 patients were found to have their testes in the superficial inguinal pouch, 11 of those were obese and 6 emerging testes with complete hernia sac.<sup>7</sup>

From our results the most common finding by laparoscope was the presence of intraabdominal testes which was found in 75% of the testes and this result is similar to the study entitled "The Impalpable Testicle—Peeping Through the Key Hole" which was done by Shah A .and Shah AV and to another study done by Satar N., Bayazit Y., and Doran S in which it was 58%<sup>8</sup> and 66.6%<sup>9</sup> respectively.

The percentage of vanished testes in this study (25%) was higher than the study performed by Satar et al, in which it was 14.3%<sup>9</sup>. The exact reason for this difference is not known it may be due to genetic causes, weather changes, traditional hormonal treatment, or delay seeking advice by the patient so this result needs to be further studied in the future.

There is a great controversy regarding inguinal exploration in patients discovered to have a vanished testes, although it is not considered mandatory,<sup>10</sup> but it was carried out in all the seven patients of vanished

testes to eliminate the potential risk of malignancy in the residual testicular stroma.<sup>8,11</sup> Hypo-plastic vessels entering the ring are associated with either testicular absence or a hypo-plastic testicular remnants, the need for inguinal exploration has been questioned.<sup>6,11-14</sup>

In contrast to the study entitled "Laparoscopy in the Management of Impalpable Testicle" which stated that blind ending spermatic vessels obviate other investigational techniques and can be considered absence of testes, but when the spermatic vessels are through the IIR, it is obligatory to assess the inguinal canal.<sup>9</sup>

In the present study the inguinal region was explored in eight (72.7%) of our 11 vanished testes and a nubbin of tissue was taken out and sent for histopathological examination which reveal no seminiferous tubules, so from these results we can assume that if we find an attenuated vas and vessels end before a closed IIR there is no need for further inguinal exploration, while when the vas and vessels end at or in an opened IIR then it is mandatory to explore the inguinal region to exclude intracanalicular vanished or atrophic testes in order to eliminate the potential risk of malignancy by removal of this abnormal testicular tissue.<sup>8,11</sup>

Over the years, many imaging modalities have been used to detect impalpable testes, these include US, C.T. scan, MRI and invasive procedures like arteriography and venography, however none of these modalities have adequate accuracy to detect presence or absence of impalpable testes.<sup>15</sup> In a study carried out in India, the overall diagnostic agreement of US examination was 19 %, <sup>15</sup> and it was 12.5%<sup>8</sup> in another one. A study done by Dr Ghulam Hazart stated that all imaging modalities used for diagnosing impalpable testes are inaccurate as laparoscopy especially in the negative ones.<sup>16</sup> In the present study we only recorded the ultrasound results and compared them with the laparoscopic findings which revealed 50% false results.

In the current study when the testes were palpated under anesthesia and treated by direct orchiopexy, the patients were discharged after recovery from anesthesia, while for those who underwent laparoscopy; they were discharged on the next day. This result is similar to a study result which was done by Amar Shah and Anirudh Shah, the patients who underwent laparoscopic orchiopexy or orchiectomy have been discharged from the hospital during the first 24 hours after surgery and satisfactory results were achieved.<sup>16</sup> While Shah A .and Shah AV stated that 75% of children was treated as a day case procedure and discharged home on the same day, 20% stayed overnight whereas 5% stayed for two days following bilateral orchiopexy.<sup>8</sup>

The follow up was from six months to two years which includes the testes size, position and cosmeses. It was scheduled in the form of four outpatient visits after one week, one month, one year and two years from the time of surgery. The results were excellent regarding testicular size equivalent to their preoperative size and cosmeses of both the scrotum and abdomen. Only four (9%) testes were smaller in size as they compared with their contralateral normally descended ones in which two of them were fixed after cutting the testicular vessels, one after extensive intraabdominal dissection and the other one by FS technique. In comparison to other studies the results are identical. Hazrat Ghulam and Mishra K stated that the cosmeses was excellent with good testicular size.<sup>17</sup> John G. vansavage did a study for 65 impalpable testes the results were one testis had a higher position and three testes had smaller size than that of the opposite side.<sup>18</sup>

The laparoscopic management of impalpable testes is the most useful method with excellent results and low morbidity in addition to that it is quick and easy in dealing with all varieties of impalpable testes.

Laparoscopic abdominal examination should be the first choice for evaluation of every patient with impalpable testes.

## REFERENCES

1. Poenaru D, Homsy YL, Peloquin F, Andze GO. Laparoscopic management of the impalpable abdominal testis. *Urology*. 1993; 42: 574-8.
2. Froeling FM, Sorber MJ, de la Rosette JJ, Joseph DM. The non palpable testis and the changing role of laparoscopy. *Urology*. 1994;43: 222-7.
3. Heiss KF, Shandling B. Laparoscopy for the impalpable testis: experience with 53 testes. *J Pediatr Surg* 1992;27: 175-8.
4. Cortesi N, Ferrari P, Eambarda E, Manenti A, Baldini A, Morano FP. Diagnosis of bilateral abdominal cryptorchidism by laparoscopy. *Endoscopy*. 1976; 8 (1): 33-4.
5. Scott JES. Laparoscopy as an aid in the diagnosis and management of the impalpable testis. *J Pediatr Surg*. 1982;17: 14-6.
6. Baillie C T, Fearn G , Kitteringham L, Turnock RR. Management of the impalpable testis: the role of laparoscopy. *BMJ*. 1998;79: 419-22
7. El-Gohary M. Amin. Role of laparoscopy in the management of impalpable testes. *J Indian Assoc Pediatr Surg*. 2006; Vol 11: 207-10.
8. Shah A .and Shah AV. The Impalpable Testicle-peeping through the key hole. *J Indian Assoc Pediatr Surg*. 2003; Vol 8: 209-12.
9. Satar N., Bayazit Y., and Doran S. Laparoscopy in the Management of Impalpable Testicle. *Acta Chir Belg*. 2005; 105: 662-6.
10. Schleef J, vonBismarck S, Burmucic K, Gutmann A, Mayr J. Groin exploration for nonpalpable testis: laparoscopic approach. *J Pediatr Surg*. 2002; 37:1552-5.
11. Plotzker ED, Rushton HG, Belman AB, Skoog SJ. Laparoscopy for nonpalpable testes in childhood: is inguinal exploration also necessary when vas and vessels exit the inguinal ring? *J Urol* 1992; 148:635-7.
12. Tennenbaum SY, Lerner SE, McAleer IM, Packer MG, Scherz HC, Kaplan GW. Preoperative laparoscopic localization of the nonpalpable testis: a critical analysis of a 10 year experience. *J Urol* 1994;151:732-4.
13. Moore RG, Peters CA, Bauer SB, Mandell J, Retik AB. Laparoscopic evaluation of the nonpalpable testis: a prospective assessment of accuracy. *J Urol*. 1994;151:728-31.
14. Elder JS. Laparoscopy for impalpable testes: significance of the patent processus vaginalis. *J Urol* 1994; 152: 776-8.
15. Amar Shah and Anirudh Shah. Impalpable Testes–Is Imaging Really Helpful?. *Indian Pediatrics*. 2006; 43: 720-3.
16. Hazrat Ghulam and Mishra K. Laparoscopic management of undescended testis. *Indian Pediatrics*. 2007; 45: 733-43.
17. John G. vansavage. Avoidance of inguinal incision in laparoscopically confirmed vanishing testis syndrome. *J Urol*. 2001; 166: 1421-4.
18. Tang PMY, Leung MWY, Chao NSY, Wong BPY, Kwok WK, Liu KKW. Use of Laparoscopy in the Management of Impalpable Testis in Children. *HK J Paediatr*. 2009; 14:172-6.



## پوخته

## به کارئینانا کلینیکی بۆ ئامیرا لاپروسکۆب ل چارهسهرکرن گونا نه دیار ل زاروکا

**پێشهکی و ئارمانج:** دیارکرن رولا ئامیرا لاپروسکۆبی ل چارهسهرکرن گونا نه دیار ل زاروکا **ریکێن فهکولینی:** فهکولینا بۆ ٤٦ زاروکا هاتیه کړن , ٣ ژوان ههردوو گون نه دیار بوون , هه می زاروک ژلایی کلینیکی دیار که گونی وان نه دیاره (ناهیهه گرتن ب دهستی) . ئه ف فهکولینا ب ریکا بکا رئینانا لاپروسکۆبی ل نه خوشخانا الخنساو ل باژییری میسل هاتیه ئه نجامدان ول ماوهی هه یفا گلاقیژی ٢٠٠٨ تا هه یفا به فرباران ٢٠١٢.

**ئه نجام:** پاش پروسه سا سرکرنی پینچ زاروک گونا وان دیار بوون وهاتیه دیتین ل ئه نجامدا نشته گهریا اورکیوپیکی کلاسیکی هاتیه ئه نجامدان ول فهکولینی هاتینه دیرئخستن. دیسان ١١ زاروکا ل ٤٤ زاروکێن مایی گون نه بوو. ل ژمارا مایی ل فهکولینی که ٣٣ زاروک بوون گونا وان لئاڤ زگی بوو. یا پیدفی هاتیه ئه نجامدان ژلایی نشته گهری و روزهک پاش ئه نجامدانی نشته گهری هاتینه ده رئیکهستن ل نه خوشخانی. چ هه ودان یان ئاریشه پاش نشته گهری روونه دا . دیفچوون بۆ ماوهی ٦ هه یفا بۆ دووسالی هاتیه ئه نجامدان و دیاربوو که گون ل جهی خو یا دروست بوو. ل چار زاروکا گونی رهخی نشته گهری بچیک تر بو.

**ده رئه نجام:** بکار ئینانا ئامیرا لاپروسکۆبی بۆ چارهسهرکرن گونا نه دیار , باشتترین ریکێن زانستی یه و دگه ل ئه نجاما نه یاب و کیمترین ئاریشه . زیده باری ریکه کی به له ز و سانایی بۆ چارهسهرکرن هه می جورا گون نه دیار.

## الخلاصة

### الفائدة السريرية لناظور البطن في علاج الخصى الغير محسوسة عند الاطفال

**الخلفية والأهداف:** ناظور البطن يفترض انه يلعب دور مهم في علاج الخصية الغير محسوسة عند الاطفال. الهدف من البحث هو تقييم استخدام ناظور البطن لعلاج الخصية الغير محسوسة.

**طرق البحث:** دراسة مستقبلية ل ٤٩ خصية غير محسوسة (٤٦ مريض) حيث انه كان هنالك ثلاثة مرضى مصابين بخصية غير محسوسة متناظرة. اجريت عملية الناظور التشخيصي لجميع المرضى في مركز جراحة الأطفال في مستشفى الخنساء التعليمي بالموصل للفترة بين تموز ٢٠٠٨ وكانون الثاني ٢٠١٢. لقد تم اثبات ان جميع الاطفال كان لديهم خصى غير محسوسة سواء كانت بجهة واحدة او متناظرة عن طريق الفحص السريري.

**النتائج:** خمسة خصى (١٠,٢%) تم إحساسها بعد التخدير واجري لهم تثبيت مباشر. تم استبعاد هؤلاء المرضى من البحث. احدى عشر (٢٥%) كانت متلاشية ، وثلاثة وثلاثون خصية (٧٥%) كانت موجودة في التجويف البطني في مواقع مختلفة حيث انه عشرة خصى (٢٢,٧%) كانت مستقرة قرب المحبس الداخلي الاريبي فثبتت مباشرة ، احدى عشر (٢٥%) ثبتت بعد تحرير واسع ، سبعة (١٥,٩%) ثبتت بعد قص أوعية الخصية وخمسة (١١,٣%) احتاجوا عملية متدرجة وهؤلاء المرضى الذين اجري لهم ناظور البطن اخرجوا من المستشفى في اليوم التالي.

لا توجد أي مضاعفات أثناء أو بعد عملية الناظور مباشرة. بعد متابعة تراوحت بين ستة اشهر و سنتين كان جميع الخصى في موقع طبيعي في كيس الصفن ماعدا أربعة (٩%) كانوا اصغر من نظائريهم.

**الاستنتاجات:** يعتبر ناظور البطن طريقة مهمة لتشخيص ومعالجة الخصية الغير محسوسة بجميع مواقعها وهو سريع وسهل التعامل مع نتائج ممتازة و قلة المضاعفات.

## FACTOR II PROTHROMBIN (G20210A) MUTATION AMONG HEALTHY INDIVIDUALS IN ERBIL / IRAQ

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### ABSTRACT

**Background and Objectives** The factor II (Prothrombin) is the precursor of thrombin. Factor II G20210A mutation is one of the genetic risk factors for thrombosis and it is associated with elevated prothrombin levels. The goal of this study was to study the frequency of the Factor II mutation in healthy individuals in Erbil-Iraq.

**Methods** A total of 100 healthy individuals attending the premarital screening center in Erbil city were recruited. Factor II gene polymorphism was investigated in all of them by the polymerase chain reaction (PCR) and restriction fragment length polymorphism (RFLP).

**Results** Factor II prothrombin (G20210A) mutation was documented heterozygous state in 2 subjects (2%).

**Conclusions** The prevalence of Prothrombin G20210A mutation differs in different countries and ethnic groups, being highest in Caucasians, especially those in the Southern Europe and in the Eastern Mediterranean; its frequency in Erbil appears slightly lower than these reports and than that from Duhok, probably implying limited role in pathogenesis of venous thrombosis in the former province.

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**Key words:** Factor II prothrombin (G20210A) Mutation, PCR-RFLP

**P**rothrombin (coagulation factor II) is the precursor of thrombin, which participates as a serine protease in the coagulation cascade. Thrombin is essential in the processes of hemostasis and thrombosis<sup>1,2</sup>. The gene that codes for prothrombin is 21 kb in size and contains 14 exons<sup>3</sup>. The gene has been mapped on chromosome 11 at position 11p11-q12<sup>4</sup>.

A G20210A mutation in the<sup>3'</sup> untranslated region of the prothrombin gene results in an elevated serum prothrombin level and an increased risk for venous thrombosis. Individuals heterozygous for the prothrombin G20210A mutation have a two- to three-fold increased risk for venous thrombosis and elevated prothrombin serum level<sup>5,6</sup>. This mutation is inherited in an autosomal dominant fashion<sup>7</sup>.

Several Polymerase Chain Reaction

(PCR) based methods are available to detect the prothrombin 20210 mutation with a high degree of specificity and sensitivity<sup>5</sup>.

Heterozygous prothrombin mutations are found in about 2% of the US white population. The mutation is uncommon in African Americans (approximately 0.5%) and is rare in Asians, Africans, and Native Americans. The homozygous form is considered uncommon, with an expected occurrence of approximately 1 in 10 000 individuals. The prothrombin 20210 mutation is equally as common in men and in women<sup>7</sup>.

The aim of present study was to investigate the presence and determine the frequency of prothrombin G20210A mutations among apparently healthy Kurds in Erbil.

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## METHODS

The study was conducted in Scientific Research Center, Faculty of Science, University of Duhok. The total samples consists of 100 random healthy individuals attending the premarital screening center in Erbil for routine mandatory checkup , included 56 males and 44 females, with age range 15-45 years.

Three mLs of whole blood collected in EDTA was used for the isolation of genomic DNA using a phenol chloroform method<sup>8</sup>.

The Factor II prothrombin G20210A mutation was determined by the polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) method and some individuals were evaluated by using reverse hybridization (RH) to oligonucleotide specific probes (ViennaLab-Austria).

### PCR-RFLP method

By the PCR-RFLP method A 506-bp fragment encompassing nucleotide position 20210 of prothrombin gene was amplified with specific primers, followed with digestion with the restriction nuclease HindIII<sup>8</sup>.

The PCR reactions were done in a sterile 0.2 ml tube. The reaction mixture contained 1×PCR buffer (Promega – USA) with 1.5 mmol/L MgCl<sub>2</sub>, 1.5 unit Taq DNA polymerase, 10 µmol/L dNTP, DNA template 2.5 µl, primers (Forward; 5'-GCA CAG ACG GCT GTT CTC TT -3' and Reverse; 5'- ATA GCA CTG GGA GCA TTG AAG C -3') (0.5µl each), completed to 25 µl with sterile distilled water<sup>9</sup>.

The thermocycling program using an ABI 2720 thermocycler (Applied Biosystems – USA) included Pre-PCR-95°C for 2 min, followed by 40 cycles of denaturation 94°C for 15 seconds, annealing 55°C for 15 seconds and extension 72°C for 30 seconds, and a final extension at 72°C for 1 min<sup>9</sup>.

PCR products and 100 bp DNA ladder (Promega-USA) were run on a 2% agarose

gels followed by staining with ethidium bromide to stain DNA fragments.

The amplicons were subjected to HindIII restriction enzyme digestion at 37°C overnight. Digestion was carried out in a final volume of 10µL, using 8.5µL of PCR product, 5 units of HindIII enzyme, and 1.0µL of buffer. Size analysis of the restriction fragments were visualized by using 6% polyacrylamid gel electrophoreses of digested PCR products and stained with silver staining kit (Promega, USA) as described by the supplier, silver- stained gels were scanned to capture digital images of the gels after air drying. Factor II G20210A creates a recognition sequence for the restriction enzyme HindIII, and this is detected after restriction with HindIII.

Undigested products of PCR-RFLP result in products of 506 bp. HindIII digestion of prothrombin wild-type amplicons yielded fragments 407 bp and 99 bp ; the 99-bp fragment was a result of an invariant HindIII site. Digestion of the prothrombin G20210A heterozygote resulted in fragments of 407 bp, 384 bp, 99 bp, and 23 bp. Prothrombin 20210A homozygotes digested with HindIII yielded fragments of 384 bp, 99 bp, and 23 bp<sup>9</sup>.

### PCR and reverse hybridization

Five individuals (involved 2 heterozygous and 3 wild genotypes) were evaluated by using PCR-RH ViennaLab StripAssay. This assay screens for several gene mutations including prothrombin gene polymorphisms whereby in vitro, the different gene sequences are simultaneously amplified and biotin-labeled in a single amplification reaction (Multiplexing). Briefly, 5 µl of DNA is added to 15 µl of PCR amplification mix in the presence of 5 µl of 0.2 U/µl Diluted Taq DNA polymerase (Promega). The thermocycler (Applied Biosystems 2720) program consists of an initial step of 94°C for 2 min, followed by 30 cycles of 94°C for 10 s, 58°C for 30 s, 72°C for 30 s, and a final extension step of 72°C for 3 min.

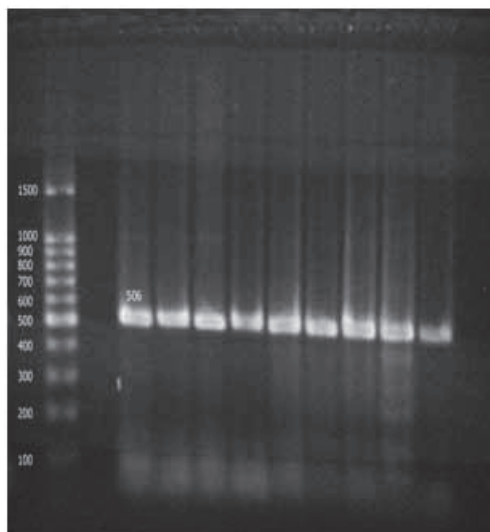
All above procedures were performed according to the instructions of manufacturers (ViennaLab-Austria). The amplified products were selectively hybridized to strip Assay which was containing allele-specific oligonucleotide probes (wild and mutant specific) immobilized as an array of parallel lines, the bound biotinylated sequences were detected using streptavidin-alkaline phosphatase and color substrates (ViennaLab-Austria).

## RESULTS

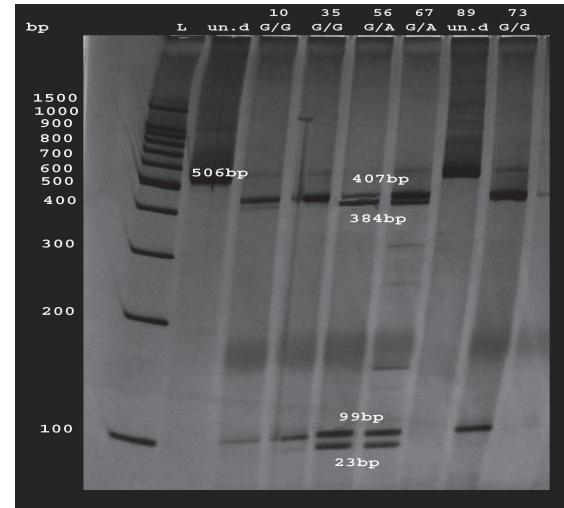
Following amplification using specific primers and HindIII restriction, 98 individuals had the 407 and 99 bp fragments thus were labeled as wild genotype (GG), another two had the 407 bp, 384 bp, 99 bp, and 23 bp fragments and thus labeled as heterozygous for the prothrombin G20210A mutation(GA). No individuals had the 384 bp, 99 bp, and 23 bp bands and thus no homozygous cases (AA) were identified (Figure 1).

Furthermore the five individuals (the 2 heterozygous and 3 random wild genotypes detected by the RFLP method above) were evaluated by using Vienna Lab Strip Assay and the results revealed that the same 2 individuals identified by PCR-RFLP were also heterozygous by RH assay (Figure 2).

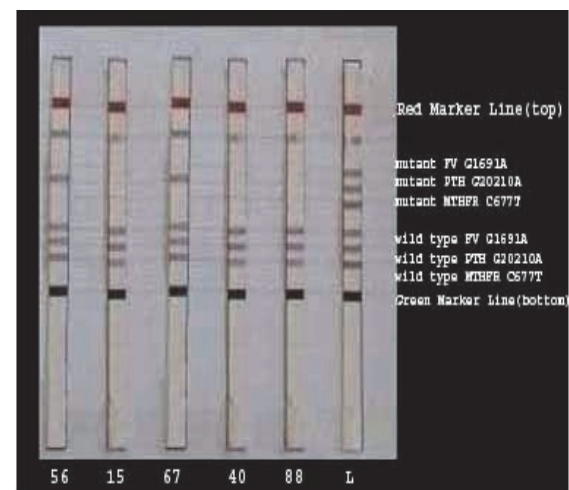
A.



B.



**Figure 1. A)** Represent the PCR product FII G20210A genotype bands in 506bp fragments by agarose gel electrophoresis. **B)** Represent the detection of FII G20210A mutation by (PCR-RFLP) analysis. Amplified undigested and HindIII digested fragments were separated by polyacrylamide. Lane 2 and 8: Undigested 506-bp prothrombin amplicon. Lane 3,4, and 9: A prothrombin G20210G wild type (G/G) with digested products of 407, and 99 bp. Lane 5 and 6: A prothrombin G20210A heterozygote(G/A) with digestion products of 407, 384, 99 bp and 23-bp products are shown. Lane 1: Ladder 100 bp DNA ladder.



**Figure 2. Represent a blot of Reverses Hybridization techniques performed according to supplier's recommended (Vienna Lab kit). Cases (#56 and 67) PTH G20210A heterozygote while cases (#15, 40 and 88) is wild type of PTH G20210A. Strip L illustrate the wild and mutant probes for each of the three genes on test strips.**

## DISCUSSION

In the general population, individuals may develop thrombosis due to multiple risk factors, acquired and genetic. Clearly, acquired factors (smoking, high cholesterol, obesity, etc.) are more frequent and combined with a genetic predisposition further enhance the risk of a thrombotic event<sup>10</sup>. Among the inherited causes of thrombosis is the G20210A polymorphism in the factor II gene, this variant is weak but a consistent risk factor for DVT and for development of CVD<sup>11</sup>.

In the current study, the overall prevalence of Prothrombin G20210A mutation in the 100 healthy individuals was found to be 2% heterozygous, which is less than the previously documented frequency of this mutation among the healthy population of Duhok to the north of Erbil, where a rate of 3% was reported<sup>12</sup>.

Our figures and those obtained from Duhok 12 are more or less consistent with figures those reported from other Eastern Mediterranean countries like Turkey, Iran, Jordan, Lebanon, and Greece 2.7%, 4%, 2%, 1.3-3.6%<sup>13,14,15,16</sup>, and 2.7%<sup>17</sup>, respectively, where it is presumed that the mutation arose some 30000 years ago.

The prevalence of prothrombin G20210A gene mutation varies widely worldwide; it is common in Europe but uncommon in Africa, Asia, and natives of America<sup>18, 19</sup>.

It is interesting to note that Rahimi et al. studied individuals of Kurdish ethnic background from the Kermanshah Province, Western Iran, and reported that the prevalence of prothrombin (G20210A) mutation as 1.6%, which is more or less comparable to our figure of 2% and among the Kurdish population in Erbil<sup>20</sup>.

The reasons for the differences in frequency of this mutation between Caucasians and non-Caucasians may be due to differences in the genetic background as well as environmental factors. In other words, genetic drift by a founder effect or a selective mechanism is

suggested. This is supported by the proposed single origin for prothrombin G20210A mutation that occurred most likely after the divergence of Africans from non-Africans and of Caucasoid from Mongoloid subpopulations<sup>6, 21</sup>.

There are many studies which have evaluated the relationship between venous thrombosis and common inherited factor II mutation as a predisposing factor, but they have reported variable results. A recent study from Duhok however revealed no association between Venous thrombosis and the prothrombin mutation<sup>22</sup>.

Prothrombin G20210A mutation testing is not a priority in our locality, however it may be indicated if the patient of venous thrombosis has some particular conditions like an age less than 50 years, venous thrombosis in unusual sites, recurrent venous thrombosis, strong family history of thrombotic disease or first degree relatives with venous thrombosis under the age of 50 years.

## REFERENCES

1. Jackson CM. Physiology and biochemistry of prothrombin. In: Bloom AL, Forbes CD, Thomas DP, Tuddenham EGD, eds. Hemostasis and thrombosis. Edinburgh: Churchill Livingstone; 1994. p 397.
2. Dang QD, Vindigni A, di Cera E. An allosteric switch controls the procoagulant and anticoagulant activities of thrombin. *Proc Natl Acad Sci U S A*. 1995; 92: 5977.
3. Degen SJF, Davie EW. Nucleotide sequence of the gene for human prothrombin. *Biochemistry* 1987; 26: 6165-77.
4. Royle NJ, Irwin DM, Koschinsky ML, MacGillivray RTA, Hamerton JL. Human genes encoding prothrombin and ceruloplasmin map to 11p11-q12 and 3q21-24, respectively. *Somat Cell Mol Genet* 1987; 13: 285-92.
5. Sharma S, Kumar SI, Poddar U, Yachha SK, Aggarwal R. Factor V Leiden and prothrombin gene G20210A mutations are uncommon in



- portal vein thrombosis in India. *Indian J Gastroenterol.* 2006; 25: 236–9.
6. Wan ZA, Subashini K, Selamah G, Narazah MY. Factor V Leiden and prothrombin G20210A mutations among healthy indians in Malaysia. *Lab. Medicine.* 2010; 41 (5) 284-87.
  7. Foy P, Moll S. Thrombophilia. *Current Treatment Options in Cardiovascular Medicine.* 2009; 11 (2): 114–128.
  8. Ausubel, FM., Brent, R. Kingston, RE, Moore DD, Seidman JG, Smith, JA, Struhl K. *Current Protocols in Molecular Biology*, Greene Publishing Associates/Wiley Interscience, New York 1987.
  9. Suzanne H, Karl VV. Analytical Evaluation of Primer Engineered Multiplex Polymerase Chain Reaction–Restriction Fragment Length Polymorphism for Detection of Factor V Leiden and Prothrombin G20210A. *The Journal of Molecular Diagnostics.* 2000; 2(3); 153-157
  10. Claudia CB, Tânia P, Rita C, Paula RP, Luisa M. Thrombotic genetic risk factors and warfarin pharmacogenetic variants in São Miguel's healthy population (Azores). *Thrombosis Journal.* 2009; 7:9
  11. Bosler D, Mattson J, Crisan D. Phenotypic heterogeneity in patients with homozygous prothrombin 20210AA genotype. *J Mol Diagn.* 2006. 8: 420-425.
  12. Al-Allawi NA, Jubrael JM, Baban NK, and Gedeon GS. Thrombophilic HROMBPHILIC Mutations in Blood Donors in Duhok/ Iraq. *Duhok Medical Journal.* 2009; 3 (1): 25-32.
  13. Akar N, Misirlioglu M, Akar E, Aveu F, Yalcin A, Sozuo A. Prothrombin gene 20210G-A mutation in the Turkish population. *Am J Hematol.* 1997; 58 (3): 249.
  14. Nahid MD, Maryam PMD, Shirin FMD. Factor V G1691A and prothrombin G20210A gene polymorphisms among Iranian patients with cerebral venous thrombosis. *Neurology Asia.* 2012; 17 (3): 199–203.
  15. Abu-Amero KK, Wyngaard CA, Kambouris M, Dzimir N. Prevalence of the 20210 G>A prothrombin variant and its association with coronary artery disease in a middle Eastern population. *Arch Pathol Lab Med.* 2002; 126 (9): 1087-1090.
  16. Tamim H, Finan RR, Almawi WY. Prevalence of two thrombophilia predisposing mutations: factor V G1691A (R506Q; Leiden) and prothrombin G20210A, among healthy Lebanese. *Thromb Haemost.* 2002; 88: 691-692
  17. Zalavras ChG, Giotopoulou S, Dokou E, Mitsis M, Ioannou HV, Tsaousi C, Tzolou A, Kolaitis N, Vartholomatos G. Prevalence of the G20210A prothrombin gene mutation in Northwestern Greece and association with venous thromboembolism. *Int Angiol.* 2003; 22: 55-57
  18. Nagaraja D, Kruthika-Vinod TP, Christopher R. The prothrombin gene G20210A variant and puerperal cerebral venous and sinus thrombosis in south Indian women. *J Clin Neurosci.* 2007; 14: 635-8.
  19. Jadaon MM. Epidemiology of Prothrombin G20210A mutation in the Mediterranean Region. *Mediterr J Hematol Infect Dis.* 2011; 3 (1): 1-11.
  20. Rahimi Z, Vaisi-Raygani A, Mozafari H, Kharrazi H, Rezaei M, Nagel RL. Prevalence of factor V Leiden (G1691A) and prothrombin (G20210A) among Kurdish population from Western Iran. *J Thromb Thrombolysis.* 2008; 25: 280-283.
  21. Hong SH. Genotype distribution of the mutations in the coagulation factor V Gene in the Korean population: Absence of its association with coronary artery disease. *Korean J Biol Sci.* 2003; 7: 255–259.
  22. Balo, H. Prothrombin G20210A polymorphism in patients with deep venous thrombosis – Duhok province. University of Duhok/ faculty of medical sciences. (2013).

## پوخته

### گۆرانکاری له فاکتەری دوویی خوین مەیین (G20210A) له کەسانی تەندروست لە هەولێر/ عێراق

**پێشەکی و ئارمانج:** فاکتەری دوئی یی Prothrombin پێشەکیە بو نەخوشی Thrombin. فاکتەری دوی گۆرانکاری G20210A فاکتەریکی ترسناکی هۆکاری گۆرانکاری جەلەیی دەمارە وە گرێدرایی لە گەل بەرزبونەوهی ئاستی مەیینی خوین (Prothrombin). ئامانج لەم توێژینەوه ئەوەیە کە لیکۆلێنەوهیەک بکۆت لە سەر کرداری ناو بەناوی فاکتەری دوی گۆرانکاری G20210A ئەو کەسە تەندروستانە لە شاری هەولێر/ عێراق.

**ریکۆن فەکۆلینی:** سەرجه مە ١٠٠ کەسین ساخەم یین سەرەدانا سەنتەری پشکنین بەری مەرەبرینی ل هەولێری کری هاتنە بەشدارکرن دقە کۆلینی دا. فاکتەری دوی گۆرانکاری G20210A هاتە پشکنینکرن بو هەمی بەشداربوویا بریک PCR و RFLP.

**ئەنجام:** فاکتەری دوی گۆرانکاری G20210A هاتە دیتن بشیوی هەمەجور ل دوو کەسین ساخەم (٢٪).

**دەرئەنجام:** فاکتەری دوی گۆرانکاری G20210A جیاوازه به جیاوازی ولاتان و جیاوازی رهچهکان . ریزهی ئەم فاکتەرە بلندە لە ناو قەوقازەکان بە تاییبەتی لە باشوری ئەوروپا و رۆژهەلاتی دەریای سپی ناوەرەست. ئەم فاکتەرە (G20210A) دەیتە دیتن ل دەف کەسین ساخەم ل هەولێری، هەرچەندە ریزە کۆمترە ژیا بەری نوکە هاتیە دیتن ل دهوکی ئەمەش پێشنیارە بو سنورداری ئەم نەخوشیە لە مەیینی خوین لە باژیری پیشوو.



## الخلاصة

### عامل الثاني البروثرومبين (G20210A) طفرات الجينية بين الأفراد الأصحاء في أربيل / العراق

**خلفية واهداف البحث:** إن العامل الثاني (البروثرومبين) هو مقدمة للثرومبين. عامل الثاني طفرة G20210A هي واحدة من عوامل الجينية الخطرة لتجلط الدم و أنه يرتبط مع مستويات مرتفعة البروثرومبين . هدف هذه الدراسة كانت دراسة تردد تغير الطفرة العامل الثاني في الأفراد الأصحاء في أربيل/ العراق.

**طرق البحث:** جمع عينة من مائة (١٠٠) الأفراد الأصحاء الذين حضروا مركز الفحص قبل الزواج في مدينة أربيل. عامل الثاني تعدد الأشكال الجيني تُحرى في كُلّ منهم بسلسلة تفاعل البلمرة ( PCR ) و تقييد جزء طول تعدد الأشكال (RFLP).

**النتائج:** عامل الثاني لطفرة البروثرومبين ( G20210A ) وقد تم توثيق حالة heterozygous في ٢ المواضيع (٢%).

**الاستنتاجات:** إنتشار طفرة البروثرومبين G20210A يَختلفُ في البلدانِ المختلفةِ والمجموعات العرقية، ويَكونُ أعلى في Caucasians، خصوصاً أولئك في جنوب أوروبا وفي شرقي البحر الأبيض المتوسط؛ تردده في أربيل يَبْدُو أوطأ قليلاً مِنْ التقاريرِ سجلت في دهوك، هذا يَدُلُّ على الدورِ المحدودِ من المحتمل في النشوء المرضي مِنْ تخثر وريدي في المحافظةِ السابقة.

**BOTOX INJECTION FOR THE MANAGEMENT OF CHRONIC IDIOPATHIC CONSTIPATION IN CHILDREN**

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**ABSTRACT**

**Background and objectives** Chronic constipation stays one of the most common and challenging problems facing the doctors and the family as well. Most of these cases are idiopathic. A lot of medical and surgical treatment modalities are in use; however there is no universal way to manage resistant cases. Each method had its own benefits and side effects. Recently Botox were used in the management of idiopathic constipation in children. The aim of this study is to evaluate the role of Botox injection in the management of these cases.

**Methods** A prospective study of 25 children with idiopathic constipation underwent Botox injection in two pediatric surgery centers. The children condition was assessed before and after the injection using the same scoring system. The material was injected in the internal sphincter.

**Results** Patient's age ranged from 2 to 9 years. Twenty four percent of the patients had significant ( $p$  value  $< 0.05$ ) and sustained improvement at three and six months after injection. While (36%) had significant improvement at three months, but they relapsed at 6 months after injection. Other 40% had no significant improvement ( $p$  value  $> 0.05$ ). No patient had deterioration of his score at the time of therapy. Transient fecal incontinence was noticed in three patients and all recovered within two weeks. No complications were recorded through out the study.

**Conclusions** The use of Botox in managing idiopathic constipation in children is a new and safe method with good response but the problem is the significant relapse rate and should be reserved for selected resistant cases.

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**Key words:** Idiopathic constipation, Botox injection, Children.

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**A** Concern about bowel function has been prevalent throughout history across many cultures. Normal bowel pattern is thought to be a sign of good health. Constipation in children is a very important problem and has reported prevalence rates between 1% and 30% in different communities.<sup>1</sup>

Constipation is defined as "a period of 8 weeks with at least<sup>2</sup> of the following symptoms: defecation frequency less than 3 times per week, fecal incontinence frequency greater than once per week, passage of large stools that clog the toilet,

palpable abdominal or rectal fecal mass, stool withholding behavior, or painful defecation.<sup>2</sup> If there is no underlying cause the condition is termed "Idiopathic constipation" which represents most of the cases. Idiopathic constipation is very common and when it becomes resistant and relapsing it become a very distressing problem for the child and the family.<sup>3</sup> Diagnosis of idiopathic constipation in children can usually be made by the history and physical examination and some investigations if indicated like thyroid function, rectal biopsy, manometry,

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barium enema, endorectal ultrasonography. However, in most infants and children with idiopathic chronic constipation there is no need for extensive investigations.<sup>3</sup> This condition is generally under treated, leading to acquired megarectum. More soiling, aggravating the distress to the family and the child.<sup>3-5</sup> Over activity or achalasia of the internal anal sphincter (IAS) is thought to play the major role in the etiology.<sup>3,6</sup> The treatment is traditionally by laxatives and enemas. Resistant cases treated in different ways all over the world, which include anal dilatation, internal sphincterotomy, myectomy and botox injection in the internal sphincter and in severe cases surgical resection of the dilated segment.<sup>3-5</sup> Anal dilatation and myectomy may carry the risk of damage to the anal sphincters which may not be apparent for many years.<sup>3,7,8</sup> Botox injection claimed to be safe, effective and carry no risk of damaging the sphincters. Botulinum toxin type-A would result in decreased muscle activity by blocking the release of acetylcholine from the neuron, leading to internal anal sphincter relaxation which last up to six months, it is recently used to treat recalcitrant constipation.<sup>3,7,9</sup>

The aim of this study is to evaluate the role of Botox injection in the internal anal sphincter in the management of children with idiopathic constipation.

## METHODS

A prospective study done on 25 children with idiopathic constipation, between Jan.2010 to Jan.2012. The cases were managed by two pediatric surgeons in Duhok and Mosul pediatric surgery centers. Full history was taken and all cases were thoroughly examined. Behavioral and social background of the cases studied were taken into consideration during history assessment. At least one Barium enema was done for every patient and rectal biopsy were done as needed.

Patients with underlying causes like Hirschsprung's disease, anal stenosis and neurological diseases etc. were excluded from the study. All the patients had at least three months treatment with laxatives and enemas with poor or no response. Patients' inclusion criteria were those of functional constipation under the Rome III criteria<sup>2</sup>, symptoms must include at least two of the following: Two or fewer defecations per week. At least one episode per week of fecal incontinence after the child has acquired toileting skills, history of excessive stool retention or retentive posturing, history of painful or hard bowel movements, presence of a large fecal mass in the rectum, history of stools with large diameter that may obstruct the toilet. The children were scored using symptom severity score (SS score)<sup>7</sup> as shown in table 1 before the injection and at 3 and 6 months after the injection. Patients with fecal impaction were dealt with before the time of injection. Finger disimpaction was not done at the time of injection in order not to interfere with the injection results. The injections were done under general anesthesia, lithotomy position. We used Botox which is Botulinum toxins Type A (onabotulinumtoxin A, Allergan Pharmaceuticals, Ireland) vial 100 international unit diluted in 5 cc normal saline. Twenty units were injected in 4 quadrants in the internal sphincter transanally this done by inserting the left index finger in the anus to feel the internal sphincter then injection done 2-3 mm distal to dentate line into the internal sphincter. A total of 80 units were given. The operation done as a day case surgery and all the patient were discharged home at the same day. The patients were followed at regular intervals using the same SS scoring system.<sup>7</sup> The laxative treatment was not stopped during the study.

**Table 1 Symptom severity scoring system for constipation and fecal incontinence in children.<sup>7</sup>**

| item   |  | Score |
|--|--|-------|
| Soiling  | none   | 0     |
|  | Rarely   | 1     |
|  | Occasionally                                   | 2     |
|  | Only if bowel loaded                           | 5     |
|  | Continuous day only                            | 8     |
|  | Continuous day and night                       | 10    |
| Delay in defecation                                  | Daily stool                                    | 0     |
|  | Every 2-3 d                                    | 1     |
|  | Every 3-5 d                                    | 2     |
|  | Every 5-10 d                                   | 5     |
|  | >10 d  | 8     |
|  | Never  | 10    |
| Pain and difficulty with defecation                  | None   | 0     |
|  | Occasionally                                   | 1     |
|  | Often  | 2     |
|  | With most stool                                | 4     |
|  | With every stool                               | 5     |
| Laxatives and enema                                  | None   | 0     |
|  | Softeners only                                 | 1     |
|  | Softeners and daily stimulants                 | 2     |
|  | Extra weekend Movicol or picosulfate           | 4     |
|  | Extra high-dose Movicol or Klean-Prep          | 8     |
|  | Laxatives and regular enemata or suppositories | 10    |
| Child's general health affected by the bowel problem | Well   | 0     |
|  | Occasionally ill                               | 2     |
|  | Often ill                                      | 3     |
|  | Ill most of the days                           | 4     |
| Behavior related to the bowel problem                | Cooperative                                    | 0     |
|  | Needs reminding to use the lavatory or pot     | 2     |
|  | Refuses to use the lavatory or pot             | 3     |
|  | Also refuses medicines                         | 4     |
|  | Also generally difficult behavior              | 5     |
| Overall improvement of the symptoms since last seen  | Nearly completely OK                           | 0     |
|  | Much better                                    | 1     |
|  | Some improvement                               | 4     |
|  | Still as difficult                             | 8     |
|  | Getting worse                                  | 12    |
| Amount of stool detected on abdominal examination    | None palpable                                  | 0     |
|  | Little palpable                                | 1     |
|  | Suprapubic only                                | 2     |
|  | To umbilicus                                   | 3     |
|  | Beyond umbilicus                               | 5     |
|  | Reaching ribs                                  | 8     |

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## RESULTS

The patients age ranged from 2 to 9 years. Sixteen patients (64%) were males and 9 (36%) were females. Analysis of the SS scoring results was obtained. The comparison was between 3 readings: one before injection (laxatives time), then three and six months after injection.

Analysis (table2) showed that 6 patients (24%) had significant and sustained improvement ( $p$  value  $< 0.05$ ) at three and six months after injection. Nine patients (36%) had significant improvement at three months but they relapsed after 6 months of injection. Ten patients (40%) had no significant improvement ( $p$  value  $> 0.05$ ). No patient had deterioration of his SS score at the time of therapy. Transient fecal incontinence was noticed in three patients and all recovered within two weeks. No complications were recorded throughout the study.

**Table 2 Outcomes of Botox Treatment**

| Groups   | No | %   |
|--|----|-----|
| 1 significant and sustained improvement at three and six months    | 6  | 24% |
| 2 significant improvement at three months but relapsed at 6 months | 9  | 36% |
| 3 No significant improvement                                       | 10 | 40% |

## DISCUSSION

When the rectum fails to empty the painful hard stools, willful defecation becomes less and less frequent. More hard stools then further accumulate in the sigmoid-rectum and the vicious cycle perpetuates. Painful defecation leads to sphincteric spasm, which further aggravates the outlet dysfunction as well as the vicious cycle.<sup>10</sup> Anal dilatations and sphincterotomies have long been practiced and benefited many children with refractory constipation. However, these procedures are in many ways traumatic and sphincterotomy risks long-term complications of permanent sphincteric damage with incontinence.<sup>11</sup> More recently, biofeedback relaxation therapies

have been practiced with some success, but its application in young children would be 'cognitively' and technically challenging. Botulinum toxin injection as a muscle relaxant which cause internal sphincter relaxation; however, because of its overall safety profile and clinically reversible effects, has gained popularity in many areas of clinical practice and would be a natural alternative for treating constipation associated with anal sphincter spasm.<sup>12-14</sup>

Although the use of Botox in managing constipation in children is a new subject, researchers like Keshtgar et al from Guy's & St. Thomas hospital obtained higher success rates (76%) than ours with sustained results at 3 and 6 months after injection.<sup>3</sup> Irani K, Rodriguez L et al found that of 24 pediatric patients with intractable constipation, 22 experienced significant improvement in their constipation lasting more than 2 weeks but the duration of effect was variable. Only 12 patients demonstrating benefit lasting 6 months.<sup>15</sup> Other researchers also found that recurrences are common after the pharmacological effect has receded but can be cured with an additional.<sup>16</sup> We did not report any persistent complications related to the use of Botox, however a short general anesthesia is needed at the time of injection. Reviewing the articles on this subject also did not mention any significant complications apart from transient incontinence which had also happened in 3 of our patients.<sup>3,12-15,17-21</sup> However Florian Friedmacher and Prem Puri reported higher rate of transient faecal incontinence and non-response to treatment.<sup>22</sup>

The use of Botox injection in the management of idiopathic constipation in children is safe, but unfortunately good and sustained response was only obtained in about 24% of the resistant cases. The cons of using Botox was a significant relapse and failure rate, and may be the high cost.

That is why Botox injection should be reserved for selected retractable functional constipation not responding to laxative therapy.

## REFERENCES

1. Van den Berg MM, Benninga MA, Di Lorenzo C. Epidemiology of childhood constipation: a systematic review. *Am J Gastroenterol*. 2006; 101 (10): 2401-9.
2. Benninga M, Candy DC, Catto-Smith AG. The Paris Consensus on Childhood Constipation Terminology (PACCT) Group. *J Pediatr Gastroenterol Nutr*. 2005; 40 (3): 273-5.
3. Keshtgar AS, Ward HC, Clayden GS. Diagnosis and management of children with intractable constipation. *Semin Pediatr Surg*. 2004; 13(2): 300-9.
4. Fitzgerald JF. Constipation in children. *Pediatr Rev*. 1987; 8: 299-2.
5. Lord PH. A day case procedure for the cure of third degree hemorrhoids. *Br J Surg*. 1969; 56 (10): 747-9.
6. Talley NJ, Weaver AL, Zinsmeister AR. Functional constipation and outlet delay: A population based study. *Gastroenterology*. 1993 Sep; 105(3): 781-90.
7. Keshtgar AS, Ward HC, Clayden GS, Ahmad S,. Botulinum toxin, a new treatment modality for chronic idiopathic constipation in children: long-term follow-up of a double-blind randomized trial. *J Pediatr Surgery*. 2007; 42: 672-80.
8. Lunniss PJ, Gladman MA, Hetzer FH. Risk factors in acquired fecal incontinence. *J R Soc Med*. 2004; 97: 111-16.
9. Keshtgar AS, Clayden GS, Ward HC. (2004, July) *Double blind controlled trial of botulinum toxin injection and myectomy of internal anal sphincter in the treatment of idiopathic constipation in children*. poster session presented at: The British Association of Pediatric Surgeons international conference; Oxford. pp. 27-30.
10. Ayantunde AA, Debrah SA. Current concepts in anal fissures. *World J. Surg*. 2006; 30(12): 2246-60.
11. Keshtgar AS, Ward HC, Clayden GS. Role of anal dilatation in treatment of idiopathic constipation in children: long-term follow-up of a double-blind randomised controlled study. *Pediatr Surg Int*. 2005; 21(2):100-5.
12. Jankovic J, Brin MF. Therapeutic uses of botulinum toxin. *N Engl J Med*. 1991; 324(17): 1186-94.
13. Gui D, Cassetta E, Anastasio G. Botulinum toxin for chronic anal fissure. *Lancet*. 1994; 344 (8930): 1127-8.
14. Maria G, Cassetta E, Gui D. A comparison of botulinum toxin and saline for the treatment of chronic anal fissure. *N Engl J Med*. 1998; 338(4): 217-20.
15. Irani K, Rodriguez L, Doody DP, Goldstein AM. Botulinum toxin for the treatment of chronic constipation in children with internal anal sphincter dysfunction. *Pediatr Surg Int*. 2008; 24 (7): 779-83.
16. Husberg B, Malmborg P, Strigård K. Treatment with botulinum toxin in children with chronic anal fissure. *Eur J Pediatr Surg*. 2009;19(5):290-2.
17. Pasricha PJ, Ravich WJ, Hendrix TR. Intraspincteric botulinum toxin for the treatment of achalasia. *N Engl J Med*. 1995; 322: 774-8.
18. Hallan RI, Williams NS, Melling J. Treatment of anismus in intractable constipation with botulinum A toxin. *Lancet*. 1988; 2 (8613): 714-7.
19. Ubhi T, Bhakta BB, Ives HL. Randomised double blind placebo controlled trial of the effect of botulinum toxin on walking in cerebral palsy. *Arch Dis Child*. 2000; 83 (6): 481 -87.

20. Minkes RK, Langer JC. A prospective study of botulinum toxin for internal anal sphincter hypertonicity in children with Hirschsprung's disease. *J Pediatr Surg.* 2000; 35: 1733-36.
21. Ciamarra P, Nurko S, Barksdale E, Fishmans, Dilorenzo C. Internal anal sphincter achalasia: clinical characteristics and treatment with *Clostridium botulinum* toxin. *J Pediatr Gastroenterol Nutr.* 2003; 37: 315-9.
22. Florian Friedmacher and Prem Puri: comparison of posterior internal anal sphincter myectomy and intrasphincteric botulinum toxin injection for treatment of internal anal sphincter achalasia: a meta-analysis. *Pediatr Surg Int.* 2012; 28: 765–71.

## پوخته

## چارهسهرکړنا بارین قه بزبونا دوم دريژ لنک زاروکا بکارئینانا فاکسینا بوتوکس

## د زهبله کا گفشوک یا نافخویی یا دهرچي

**پيشهکی و نارمانج:** بارین قه بزبونا دوم دريژ لنک زاروکا ژ ئاريشين گهلهک بهربه لافه کو ب زهحمهت بهيته چارهسهرکړن و چيدبيت هندهک جارن يا ئالوزبيت ئهقهژی کارتيکړنهکا نهريڼی دکته لسهر زاروکی و مالا وی و چيدبيت بهرهقاژی بيت لسهر دهرونی زاروکی و مالا وی. گهلهک جورين ريکا هه نه کو لجيهانی دهينه بکارئینان بو چارهسهرکړنا فی باری بهلی چيدبيت هه می سهر نهگرن. بارين ئالوز چيدبيت پيتفی ب مایتيکړنهکا نشتهرگهري ههبيت و کريارين نشتهرگهري یا قالا نابيت ژ ئاريشا و زيانا. بکارئینانا فاکسینا بوتوکس ريکهکا نوی یه و چيدبيت جهی نشتهرگهري بگريت و زاروکی ژ فی ئاريشی قورتال بکته. نارمانج ژ فی قهکولینی خواندنهکا رژه بو فی ريکی د چارهسهرکړنا بارين ئالوز.

**ريکين قهکولینی:** خواندنهکا پاشهروژي ژ ۲۵ زاروکا گرت ل مهلبهندی دهوک و میسل یا نشتهرگهري زاروکا هه می بوار کهفتنه بهر چارهسهریا ئاسایی ئهوژی دهرمان کو دهمهکی کو کيمتر نهبوو ژ سی ههيفان بهلی چ دیار نهبوون دفی دهمی خواندنهکا دبواری وان هاته کرن پاشی بمادی بوتوکس هاتنه فاکسينکرن و ئهجام هاتنه بهراوردکرن.

**ئهجام:** خواندنئ دیارکر کو ۲۴٪ ژ زاروکا برهنگهکی بهرچاډ باش بوون. بهلی ۳۶٪ ههچهنده د هر سی ههيفين ئيکی د باش بوون بهلی جارهکا پی تووش بوون و پشتی شهش ههيفا نیشان لی پهيدا بوون. ۴۰٪ باری وان یی ساخلهمیی باش نهبوون. چ نيشانين لایهکی ژ چارهسهرکړنی نههاتنه تومارکرن.

**دهرئهجام:** بکارئینانا بوتوکس د چارهسهرکړنا قه بزبونا دوم دريژ لنک زاروکا ريکهکا نوی یه و پشت راسته بهلی ژ خواندنئ دیاربوو کو وهرگرتنا دهرمانا یا باش و بهردهوام بوو بو چاريکا بارين هاتينه خواندن. بهلی بارين دی د وهرگرتنا دهرمانا نه یا ب دل بوو. ئهقجا شيرت دهيته کرن کو بکارئینانا فی ريکی بو هندهک بارين ئالوز دهيته دهست نيشانکرن.



## الخلاصة

### علاج حالات الإمساك المزمن عند الأطفال باستعمال حقن البوتوكس في العضلة العاصرة الداخلية للمخرج

**خلفية واهداف البحث:** تعتبر حالات الإمساك المزمن عند الأطفال من المشاكل الشائعة جدا والتي قد يصعب علاجها وقد تصبح مستعصية أحيانا مما يشكل تأثيرا سلبيا على الطفل والأهل وقد ينعكس على نفسية الطفل وأهله. تستخدم عالميا أنواع كثيرة من الوسائل لعلاج هذه الحالة وقد تفشل جميعها. إن الحالات المستعصية قد تحتاج إلى تدخل جراحي ولا تخلو العمليات الجراحية من مشاكل وأضرار جانبية. يعد استعمال حقن البوتوكس طريقة جديدة قد يحل محل الجراحة ويجنب الطفل هذه المشاكل. الهدف من هذا البحث دراسة جدوى هذه الطريقة في علاج الحالات المستعصية.

**طرق البحث:** دراسة مستقبلية شملت ٢٥ طفلا في مركزي دھوك والموصل لجراحة الأطفال. جميع الحالات خضعت للعلاج التقليدي بالأدوية الملمنة لفترة لا تقل عن ثلاثة اشهر و بدون استجابة ملحوظة. درست حالتهم خلال هذه الفترة ثم تم حقنهم بمادة البوتوكس وقورنت النتائج.

**النتائج:** أظهرت الدراسة ان ٢٤% من الاطفال تحسنوا بشكل ملحوظ ومستديم. بينما ٣٦% بالرغم من تحسنهم في الثلاثة اشهر الاولى لكنهم عانوا من انتكاسة ورجوع الاعراض بعد ستة اشهر. ٤٠% لم تتحسن حالتهم الصحية. لم تسجل أي اعراض جانبية من جراء العلاج.

**الاستنتاجات:** استخدام البوتوكس في علاج الامساك المزمن عند الاطفال تعد طريقة حديثة ومأمونة. لكن لوحظ من الدراسة أن الاستجابة كانت جيدة ومستديمة لربع الحالات المدروسة فقط. أما بقية الحالات فقد كانت الاستجابة غير مرضية. لذا ينصح بتحديد استعمال هذه الطريق لبعض الحالات المستعصية.

MORTALITY OF BURNS IN DUHOK, IRAQ

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ABSTRACT

**Background and Objectives** The mortality rate following burn is an important outcome parameter. This study aimed to identify factors associated with mortality in adult hospitalized burn patients in Duhok, Iraq

**Methods** In a prospective study, 812 burn patients were admitted to the Burns and Plastic surgery Hospital in Duhok-Kurdistan region of Iraq from 3rd January 2011 and 30th December 2012. The age, sex, nationality, cause of burn, extent of injury, cause of death and mortality rates were tabulated and analyzed.

**Results** 310 (38.2%) were males and 502 (61.8%) were females. The highest number of cases occurred in January, with the highest short period incidence occurring in April. Out of 812 patients, 170 patients died. Burn injuries were more frequent and larger with higher mortality in females than in males. Flame was the major cause of burns. Self-inflicted burns were noted mainly in young women.

**Conclusion** A large number of burn injuries, which affect children and females, occur in the domestic setting and could have been prevented. Therefore, it is necessary to implement programs for health education relating to prevention of burn injuries focusing on the domestic setting.

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**Key words:** Mortality, Burns, Duhok

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**B**urns are the major causes of disability and mortality throughout the world. In developed countries, burn mortality rate is 2.1 per 100,000 person-years<sup>1</sup>. The foremost determinants which influence survival in the thermally injured are increasing age and size of burn. Other important factors which have an effect on mortality include an associated inhalation injury, depth of burn, resuscitation protocol, and timing of surgery, infection control and nutritional support<sup>2-6</sup>.

In order to develop preventive programs, it is important that the epidemiology of burns include information about the risk factors that predisposes a person to the occurrence of this type of trauma. Few such data is available for Iraqi burn patients. For this reason, the simple

description of the epidemiologic data of patients treated in our burn centers would be of benefit for the region and these study admissions and statistics of Duhok burn units, and outcome, in terms of mortality, are used to describe the problems of burn injuries in Duhok city and surrounding area.

**METHODS**

Eight hundred and twelve burn patients were admitted to the Burns and Plastic surgery Hospital in Duhok-Kurdistan region of Iraq, between 3rd January 2011 and 30th December 2012. The age, sex, nationality, cause of burn, extent of injury, cause of death and mortality rate were tabulated and analyzed. The total body surface area (TBSA) per cent burn

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involvement was estimated according to the Lund and Browder chart by the burn team doctor. All patients received Advanced Trauma Life Support (ATLS) protocol from the Burn Unit team. Fluid resuscitation was carried out based on the Parkland formula:  $4/\text{kg}\% \text{ TBSA} = \text{total fluid to be administered in the first 24 h}$  and adjusted accordingly to maintain urine output at about 1 ml/kg body weight/hour. Escharotomy are medicated for full thickness

Circumferential burns of the extremity or for full thickness burns of the chest wall when the eschar compromises thoracic cage excursion and, thus, ventilation of the patient. When inhalation injury was suspected for any reason (such as facial burn, stridor, exposure to heavy smoke, burned nasal vibrissae, etc.), the high percent oxygen therapy via (special face mask with reservoir bag) which gives oxygen concentration from 60 to 80 percentage, guided by ABG, if SPO<sub>2</sub> is normal so we stop oxygenation for 20 minutes and do ABG if PaO<sub>2</sub> is normal we do stop oxygen therapy.

Burn wound care included cleansing the area with saline and covering it with 1% cream silver sulphadiazine daily. After successful resuscitation and stabilization of the patient, the early for major burns that are deeply full thickness was carried out. The first session of early tangential excision was done on days 3 or 4 post burn. The extent of excision depended on the availability of the donor sites for skin grafting to the burn wound and the general condition of patient, in the majority of patients at average of 15 per cent of the burn surface area will undergo excision and grafting. As paralytic ileus can be prevented by starting feeds in the immediate post-injury period. Enteral alimentation was started on day 2, as drinking fluid and light food allowed and gradually increased to its optimal need by days 5 or 6 post burn, Stress ulcer prophylaxis (sucralfate, and protein pump inhibitors) are given in those patients who

are not taking oral or enteral feeds or those with a previous history of peptic ulcer disease. No systemic prophylactic antibiotics were given, the antibiotics were given according to sensitivity profiles in the presence of positive cultures<sup>7</sup>.

## RESULTS:

Between 3rd January 2011 to 31 December 2012, 812 burned patients were admitted to Duhok burn and plastic surgery hospital. The mean age of admitted patients was 25.6 years. The 12-25 age groups had the highest rate of burns. Ninety three per cent of patients were under 60 years old. Among 812 cases, 310 (38.2%) were male and 502 (61.8%) were female. Burn rate in females was more than males. The highest number of cases occurred in January, with the highest short period incidence occurring in April. Out of 812 cases, 170 patients died while the remainder all recovering Table (1).

**Table 1. Burn mortality**

| Month | Admission | Death |     |
|-------|-----------|-------|-----|
| Jan   | 82        | 24    | 29% |
| Feb   | 65        | 19    | 29% |
| Mar   | 81        | 22    | 27% |
| Apr   | 58        | 10    | 17% |
| May   | 73        | 15    | 20% |
| Jun   | 60        | 11    | 18% |
| July  | 65        | 12    | 18% |
| Aug   | 70        | 17    | 24% |
| Sep   | 56        | 9     | 16% |
| Oct   | 76        | 14    | 18% |
| Nov   | 69        | 9     | 13% |
| Dec   | 57        | 8     | 14% |

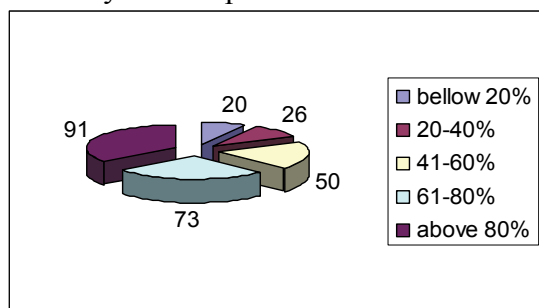
Age the mean age of persons who died was 26.8 years (range 2 months to 79 years). Out of 170 patients 73 (43%) were young (21-40 years), followed by adolescents (15-20 years) and the middle aged (41-50 years). A minimal number of patients were observed in the extreme age groups (Table2).

Sex the male/female ratio in patients that died was 1:5 (27 males and 137 females). The predominance of female deaths was observed throughout the study period except in the extreme age groups, the male/female ratio was 1:1.5 (Table 2).

**Table 2. Age and Sex distribution in burn deaths**

| Age group (years) | N  | Male (%) | N   | Female (%) |
|-------------------|----|----------|-----|------------|
| 0-12              | 12 | 7,.      | 18  | 10,5       |
| 13-20             | 2  | 1.7      | 59  | 34,7       |
| 21-30             | 3  | 1.7      | 44  | 25,8       |
| 31-40             | 6  | 2.3      | 11  | 6,4        |
| 41-50             | 4  | 2,2      | 5   | 2,9        |
| Above 50          | 3  | 1,7      | 3   | 1,7        |
| Total             | 30 | 85,7     | 140 | 14,3       |

Extent of bum out of 170 deaths, 129 (76 per cent) had burns to the extent of 50% of TBSA and more (Figure 1). In the present study the minimum extent of burns (total body surface area) which caused mortality was 15 per cent.



**Figure 1. Extent of bum**

Causes of burn the most common cause of burns was flame (154 cases): (90 per cent) involving more females (139) than males (13). Analysis of mode of flame burn injuries revealed that 129 (76 per cent) burns were due to kerosene, either from a kerosene oil stove bursting (11 per cent) or suicide attempt by burn. Scalds were seen more frequently in children (8 per cent). These were caused due to accidental spillage or fall into hot gravy, boiling water or other hot liquids. Electrical burns (2 per cent) were more

common in males. These occurred mostly at the place of work.

Most of the suicidal burn deaths were observed in female at 83 per cent. Kerosene was the main factor in all the suicidal and homicidal burns.

Causes of death more than half of the deaths (55 per cent) were due to septicemia. Inhalation injury (40 per cent) and hypovolaemic shock (5 per cent)

## DISCUSSION

Mortality rate is an important standard for evaluating the results of burn, and the outcome measures are the first step in evaluating consequences<sup>7</sup>. Furthermore, mortality rates are used in

evaluating new therapeutic interventions and establishing standards of burn survival. The predictive factor for mortality is known for age, TBSA involved and presence of inhalation injury<sup>8-10</sup>. The results of this study have been summarized and compare with those from other countries.

The present study revealed that nearly two-thirds of fatal burn cases were in the young age group (21-40 years) with peak incidence in the 21-30 years group. This is similar to the observation of Jaipur and Jayaram who also reported peak incidence of burn deaths in young adults<sup>11,12</sup>. In countries such as Scotland, Ivory Coast and Angola, the rates were higher among children<sup>13-15</sup>. In Spain, 62 per cent of cases were older than 40 years<sup>16</sup>.

Age has been recorded as one of the strongest prognostic variables for mortality after burn injury. In the elderly with burns, very high mortality rates have been observed. Herd et al<sup>17</sup> reported a 45% mortality rate in 123 burned patients aged  $\geq 65$  years. Despite the low mortality rate in the elderly in our review, other studies revealed opposite findings with the highest mortality rates in the elderly<sup>18-20</sup>. The mean age of persons who died was 26.8 years (range 2 months to 79 years). Out of 170 patients 73(43%) were young (21-40 years), followed by adolescents (15-20

years) and the middle aged (41-50 years).thus this may explain A minimal number of patients were observed in the extreme age Hence, the higher the number of elderly with burns, the higher the overall mortality rate.

A higher incidence of burn deaths among females was observed throughout the study period, except in the extreme age groups, suicide is the major cause. Low economic condition, lack of enough education and opportunity, dependency and teen age love marriage and physical abuse were predisposing factors of suicide. Long-term disappointments, depression and emotional problem resulted suicide by pouring kerosene and lighting thereby. Maximum suicide burnt cases were related to young married females, in the kitchen room using kerosene. Lack of social or family support to love married female was also working as the source of stress. Physical abuse in alcohol drinking situation by husband was supporting more to emotional/impulsive suicide A higher burn mortality rate amongst females was also found in Egypt, Kuwait, Iran, and India, with flame found to be the most common cause of burn morality. Similar sources of fire were responsible for fatalities in India. In Iran and Kuwait, cooking fire was the major source of fatal burn<sup>21-21</sup>, whereas in some other countries (Argentina, Thailand, Uruguay and Saudi Arabia), about 70 per cent of burn cases were male<sup>22</sup>. In Spain, burn cases were observed to be more common among males in all the age groups except in the elderly<sup>23</sup>.

Body surface area burned is also known as an important prognostic factor with an increase of mortality with increased body surface area burned. In the present study, the majority (89 per cent) of burn deaths were in the subjects with > 50 per cent burns, thus demonstrating that such extent of burn is usually incompatible with life in Iraq. Studies from Jaipur, Albania and Saudi Arabia<sup>24</sup>. Also reported 80 per cent

mortality rate in burns to the extent of more than 40-50 per cent TBSA.

The observations of the present study that flame was the major cause of burn are in agreement with the Dalbir study in India<sup>25</sup>. In other countries (Angola, Ivory Coast and Jordon),<sup>16,25,26</sup> calds were reported to be the major cause of burn mortality. In the present study, kerosene was seen to be the most important cause of burn. Kerosene stoves are still widely used for cooking purposes in Iraq and cooking is generally regarded as the responsibility of women. The poor quality of design, and manufacture of pressure stoves also exposes women to a higher risk of accidents.

Suicidal burn, as observed in the present study, was the major cause of burn as compared to accidental burn. Multiple complications that occurred included: particularly sepsis, clinical and radiological diagnosed of bronchopneumonia and septic shock. The commonest cause of death was septicaemic shock, Recognition of septic shock requires identification of features of the systemic inflammatory response syndrome (SIRS), based on American College of Chest Physicians/Society of Critical Care Medicine Consensus Panel guidelines. —mental changes, hyperventilation, distributive hemodynamics, hyperthermia or hypothermia, and a reduced, elevated, or left-shifted white blood cell (WBC) count—along with the existence of a potential source of infection closely followed by extensive of burns. These findings are similar to those reported by other authors<sup>27-28</sup>.

Peak mortality in adolescent and young age groups, predominance of mortality in females in all age groups except extreme age burns were also more severe among females (particularly in the age group of 15-40 year).Suicide was the major cause of burn, The research on patients with all means of suicide attempts is needed further to understand more about the problem.

Septicemia was the major cause of burn death.

A large number of burn injuries affecting females occur in the domestic setting and could have been prevented. Therefore, it is necessary to implement programs for health education relating to prevention of burn injuries focusing on the domestic setting. These strategies might be communicated by means of broadcast flashes on television or the radio, showing risk situations together with epidemiological data about burn accidents and sentences to call attention to strategies to prevent burn accidents. In that way, we believe that implementation of educational programs might reduce the incidence of burn injuries.

## REFERENCES

1. Ryan CM, Schoenfeld DA, Thorpe WP, Sheridan RL, Cassem EH, Tompkins RG. Objective estimates of the probability of death from burn injuries. *N Engl J Med* 1998; 388:362–6
2. Khadim MF, Rashid A, Fogarty B, Khan K. Mortality estimates in the elderly burn patients: the Northern Ireland experience. *Burns* 2009;35:107–13.
3. Germam G, Bartholdl U, Lefering R, Raff T, Hartmann B. The impact of risk factors and pre-existing conditions on the mortality of burn patients and the precision of predictive admission-scoring systems. *Burns* 1997; 23:195–203.
4. Spies M, Herndon DN, Rosenblatt JJ, Sanford AP, Wolf SE. Prediction of mortality from catastrophic burns in children. *Lancet* 2003; 361:989–94.
5. Hogan C, Murray B, Loo L, Hospenhal M, Cancio D, Kim S et al.. Contribution of bacterial and viral infections to attributable mortality in patients with severe burns: an autopsy series. *Burns* 2010; 773–9.
6. Germam G, Bartholdl U, Lefering R, Raff T, Hartmann B. The impact of risk factors and pre-existing conditions on the mortality of burn patients and the precision of predictive admission-scoring systems. *Burns* 1997;23:195–220.
7. Gupta M, Gupta OK, Yaduvanshi KK, Upadhyaya J. Burn epidemiology in Pink city scene. *Burns* 1993; 22:47–51.
8. Jayaraman V, Ramakrishnan KM, Davies MR. Burns in Madras, India: an analysis of 1368 patients in 1 year. *Burns* 1993; 19:339–44.
9. Sarhadi NS, Murray GD, Reid WH. Trends in burn admissions in Scotland during 1970–1992. *Burns* 1995; 21:612–5.
10. Vilasco B, Bondurand A. Burns in Abidjan, Cote D'Ivoire. *Burns* 1995; 21:291–6.
11. Adamo C, Esposito G, Lissia M, Vonella M, Zagaria N, Suderi N. Epidemiological data on burn injuries in Angola: a retrospective study of 7230 patients. *Burns* 1995;21:536–8.
12. Reig A, Tejerina C, Baena P, Mirabet V. Massive burns: a study of epidemiology and mortality. *Burns* 1994; 20:51–4.
13. Herd BM, Herd AN, Tanner NSB. Burns to the elderly: a reappraisal. *Br J Plast Surg* 1987; 40:278–82.
14. Lari AR, Alaghebandan R, Nikui R. Epidemiological study of 3341 burn patients during 3 years in Tehran, Iran. *Burns* 2000; 26:49–53.
15. Souza DAD, Marchesan WG, Greene LJ. Epidemiological data and mortality rate patients hospitalized with burns in Brazil. *Burns* 1998; 24:433–8.
16. Soltani K, Zand R, Mirghasemi A. Epidemiology and mortality of burns in Tehran, Iran. *Burns* 1998; 24:325–8.
17. Saleh S, Gadalla S. Accidental burn deaths to Egyptian women of reproductive age. *Burns* 1986; 12:241–5.



18. Souza DAD, Marchesan WG, Greene LJ. Epidemiological data and mortality rate of patients hospitalized with burns in Brazil. *Burns* 1998;24:433–8.
19. El Danaf A. Burn variables influencing survival: a study of 144 patients. *Burns* 1995; 21:517–20.
20. Singhe D. Singh A. Sharma AK, sodhil. Burn mortality in Chandigarh zone: 25 years autopsy experience from a tertiary care hospital of India. *Burns*. 1998; 24:150–6.
21. Adamo C, Esposito G, Lissia M, Vonella M, Zagaria N, Scuderi N. Epidemiological data on burn injuries in Angola: a retrospective study of 7230 patients. *Burns* 1995; 21:536–8
22. Pereira C. Murphy K. Herndon D. Outcome measures in burn care. Is mortality dead? *Burns* 2004; 30:761–71.
23. Laloë V, Ganesan M, Self-immolation: a common suicidal behaviour in Eastern Sri Lanka. *Burns*. 28 (2002), pp. 475–80
24. K.E. Nega, B. Lindtjorn Epidemiology of burn injuries in Mekele Town. Northern Ethiopia: a community-based study *Ethiopian J Health Dev.* 16 (2002), pp. 1–7
25. Pruitt Jr BA, Mason Jr AD, Goodwin CW. Epidemiology of burn injury and demography of burn care facilities. *Burn*. 1990;32:235-51.
26. Cadier MA, Shakespeare PG. Burns by thermal fire. *Burns*. 1995;21:200-4.
27. Branski LK, Al-Mousawi A, Rivero H, et al. Emerging Infections in Burns. 2009;10(5):389-97.
28. Greenhalgh DG, Saffle JR, Holmes JH, Gamelli RL, Palmieri TL, Horton JW et al. American Burn association consensus conference to define sepsis and infection in Burn Care Res. 2007; 28: 776-90.

## پوخته

## پژوهی مردن به هۆی سوتاوی له دههوك -عیراق

**پێشهکی:** ریزهکانی مه‌رگداری، پاش سوتان، ئه‌نجامی گرنگی پێوه‌ره دانپێدراوه‌کانه. ده‌توانرێت وه‌کو خاڵه هه‌ستپێکراوه دوايه‌یه‌کان به‌کار به‌یnderین بۆ کۆنترۆڵکردنی چه‌شنی. هۆکانی مردن پاش سوتاندنی سه‌خت به هۆی کات تێپه‌رینه‌وه گۆپاون.

له تۆیژینه‌وه‌یه‌کی پێشبیندا هه‌شت سه‌ده‌و دوانزه نه‌خۆشی سوتان، له ساڵی ۲۰۱۱ دا، چوبونه خه‌سته‌خانه‌ی سوتان و نه‌شته‌گه‌ری جوانکاری شارێ ده‌وک له هه‌ریمی کوردستانی عێراق .

**ریکین ئه‌کولینی:** ته‌مه‌ن و ره‌گه‌ز و ره‌گه‌زنامه و هۆی سوتاندن و بری سوتاندنه‌که و هۆی مردن و ریزه‌ی مردنه‌کان کۆکرا‌بونه‌وه‌و شیکرا‌بونه‌وه: ۲۱۰ (۲÷۲۸٪) نێرینه و، ۵۰۲ (۸،۶۱) مێینه بوون. به‌رزترین ژماره‌ی روداوه‌کان له مانگی کانونی دوا و به‌رزترین روداوی ماوه کورته‌کان له مانگی نیساندا بوون. له ۸۱۲ خاڵه‌تانه‌دا ۱۷۰ که‌سیان مردبون.

**ئه‌نجام:** روداوی زیانه‌کانی سوتان زۆر زوو رووداو‌تر بوو، به‌شی مردنی مێینه له نێرینه به‌رز تر بوو. ئاگر هۆی سه‌ره‌کی سوتاندن بوو،

**ده‌رئه‌نجام:** سوتانی خودی خۆی به سه‌ره‌کی له ئافره‌تی گه‌نجدا سه‌رنجده‌درا.

ژماره‌یه‌کی زۆری سوتاندن که کاریگه‌رییان له سه‌ر مندالان و مێینه‌دا هه‌یه، له ده‌ورو به‌ری ماڵدا روویان دا‌بوو، که ده‌توانرا نه‌هێشت‌تریت روو به‌دن . له به‌ر ئه‌وه پێویسته که پرۆگرام بۆ په‌ره بوته‌ندروستی بره‌خسێنرێت، ئه‌وه‌ی په‌یوه‌ندی به رێگرتن له زیانی سوتانه‌وه هه‌یه و ئارسته‌ی ده‌روبه‌ری ناو ماڵ بکری‌ت .



## الخلاصة

### وفيات الحروق في محافظة دهوك

**خلفية وأهداف البحث:** أن معدل وفيات الحروق هي نتيجة مهمة للمعلومات الثابتة ويمكن ان تستخدم بمثابة نقاط موضوعية نهائية للسيطرة النوعية. وبمرور الزمن، فقد تغيرت أسباب الوفيات نتيجة لحروق شديدة، يهدف البحث إلى معرفة الأسباب المرتبطة بوفيات الحروق في محافظة دهوك.

**طرق البحث:** في دراسة تتبعية، تم ادخال ثمانمائة واثنى عشر مريض حروق الى مستشفى الحروق والجراحة التجميلية في مدينة دهوك في اقليم كردستان العراق في سنة للفترة من بين ٣ يناير ٢٠١١ لغاية ٣١ ديسمبر ٢٠١٢، وأجريت جدولة وتحليل بيانات العمر، الجنس، الجنسية، سبب الحرق، مدى الحرق، سبب الوفاة، ونسب الوفيات.

**النتائج:** أظهرت الدراسة بأن ٣١٠ (٣٨.٢%) كانوا من الذكور و٥٠٢ (٦١.٨%) من الاناث. وأن أعلى الحالات قد وقعت في شهر كانون الثاني مع وقوع أعلى معدل لفترة قصيرة في شهر نيسان ومن ضمن العدد ٨١٢ فقد توفي ١٧٠ مريضاً.

كانت إصابات الحروق متكررة الحدوث وأعلى نسبة وفيات الاناث من الذكور. وكان اللهب السبب الرئيسي للحروق وبأن الحروق المسببة ذاتيا كانت ضمن النساء الشابات.

**الاستنتاجات:** أظهرت الدراسة أن عدد كبير من الاضرار والتي تؤثر على الأطفال والانات تقع في بيئة محلية بحيث كان من الممكن منع وقوعها. وعليه، فمن الضروري اجراء برامج التربية الصحية من التي تتعلق بمنع وقوع حوادث الحرق والتركيز على المحيط المحلي.

VASCULAR ACCESS OUTCOMES IN PATIENTS ON  
HAEMODIALYSIS IN DUHOK KIDNEY DISEASE CENTER

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**ABSTRACT**

**Background and objectives** Patients with chronic renal failure have only two options of treatment either renal transplantation or dialysis (peritoneal or haemodialysis), haemodialysis need efficient, comfortable, easy accessible, long duration and least complication vascular access. We try to analyze 102 patients and evaluating their vascular access. The aim of the study is compare between different types of vascular access and assessing, the efficiency, durability, and complications associated with that vascular access.

**Methods** This is retro and prospective study for patients with vascular access on haemodialysis; sample is collected from Jun 2009 to 30th June, 2013 in Duhok Kidney Disease Center.

**Results** Hundred two patients included in this study. Diabetes was the most common cause of end stage renal failure in the studied group. 76.4 % of the studied group was with arteriovenous fistula. Small vein was the most common reason for creation of graft fistula. Edema was the commonest complication among arteriovenous fistula where thrombosis was among the graft fistula.

**Conclusions** Only 10% of the studied group present early for creation of vascular access before needing urgent dialysis. Complications seem to be equal in both sexes in arteriovenous fistula type of access but its more than four folds in women with graft fistula. Aneurysm in patients with arteriovenous fistula is high in this study in compares to other studies.

**Duhok Med J 2013;7(2): 32-40.**

**Key words:** Haemodialysis, Arteriovenous fistula, Vascular access

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The body fluids (volume and composition) are regulated by kidneys, achieved by an ultra filtrate of plasma (120 milliliter/minutes, 170 liters/day). The kidney is primarily responsible for excretion of many metabolic breakdown products (including ammonia, urea and creatinine from protein, and uric acid from nucleic acids), drugs and toxins.<sup>1</sup> Renal failure has three modalities of therapy; hemodialysis, peritoneal dialysis and renal transplantation. End stages renal disease (ESRD) population is projected to grow by about 7% per year.<sup>2</sup> Current targets include

a urea reduction depending on whether urea concentrations are equilibrated. 9 and 12 hours of dialysis are required each week, usually divided into three equal sessions.<sup>3</sup> The ideal vascular access should provide safe and effective therapy, easy to use, reliable and have minimal risk to the individual receiving haemodialysis.<sup>4</sup> The creation of optimal vascular access requires an integrated approach among patient, nephrologist, and surgeon<sup>5</sup>. Planning of vascular access for haemodialysis in pre-dialysis patients often remains an unsolved problem, Prevalence of central venous catheters at first dialysis

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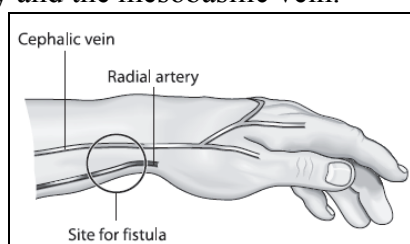
of chronic renal failure patients is also proposed to evaluate the efficiency in access planning.<sup>6</sup>

Types of vascular access

External Arterio-Venous (A-V) shunt.

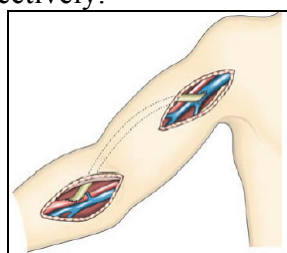
Central venous catheter (subclavian, Jugular, femoral).

Internal Arterio-Venous shunt, is the access of choice in chronic haemodialysis, this shunt is usually placed above the wrist between the radial artery and the cephalic vein as shown in figure 1, and in cases where there is no proper vein it can be done at the elbow between the humeral artery and the mesobasilic vein.

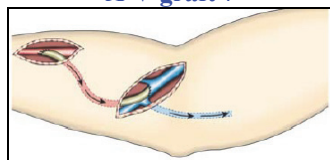


**Figure 1. Radial artery and the cephalic vein fistula<sup>7</sup>**

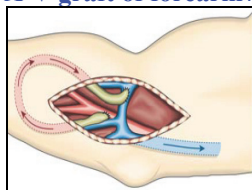
Arteriovenous graft, the A-V grafts are usually placed in the arm between the brachial artery and the axillary vein, either straight, or in loop formation in the forearm between the brachial artery and mesobasilic vein<sup>7</sup> as shown in figures 2,3,4 respectively.



**Figure 2. Brachial axillary A-V graft<sup>7</sup>**



**Figure 3. Radial Mesobasilic A-V graft of forearm<sup>7</sup>**



**Figure 4. brachial-Mesobasilic "en loop" A-V graft<sup>7</sup>**

Complications:

Venous Stenosis and venous hypertension.

Aneurysm is recognized as a localized ballooning of the vein.

Ischemia, Patients with severe peripheral vascular disease who are more predispose to the development of ischemia.

Infection of A-V fistula, Failure of vascular access because of thrombosis with or without associated infection.<sup>8</sup>

Cardiac failure.

Seroma perigraft Seroma is relatively rare complication of synthetic vascular prosthesis.<sup>9</sup>

## METHODS

### Patients and Study Design

This is retro and prospective study was conducted at the Duhok Kidney Disease Center (DKDC) and Azadi Teaching Hospital, Duhok Directorate of Health from 1st June, 2009 to 30th June, 2013. Hundred two patients with End Stage Renal Disease on regular Hemodialysis (aged 11 – 75 years) living in different areas of Duhok Governorate were enrolled in the study. Among these, 46 patients were males and 56 were females.

Oral consent was obtained from the subjects after the nature of the study had been explained to them. The study protocol was approved by Medical and Ethical committee of School of Medicine, Faculty of Medical Sciences – University of Duhok.

A pre-tested questionnaire was designed to obtain information on age, gender, residence, cause of ESRD, duration of being with renal failure, sort of access that patient start haemodialysis, duration of hemodialysis, duration of having vascular access, treatment plan, type of vascular access, place of creating vascular access, history of previous attempt, type of vascular access complications if present, and reason of creating graft fistula.

Methods

Data collected from patient's history and case sheets of vascular access operations collected from Azadi Teaching Hospital and Private Hospital statistics units. Patients were followed at Duhok Kidney Disease Center from 1st June, 2009 to 30th June, 2013.

#### Statistical Analyses

**Table 1. Characteristic of Participant**

|  |           |
|--|-----------|
| Age (Years)                                  | 47.7 ± 16 |
| Residence                                    |           |
| Urban  | 57        |
| Rural  | 45        |
| Sex  |           |
| Male   | 46        |
| Female                                       | 56        |
| Mean Duration of ESRD (in months)            | 48        |
| Mean Duration of Dialysis (in months)        | 28        |
| Plan of Treatment                            |           |
| Kidney Transplantation                       | 51        |
| Dialysis                                     | 51        |
| Type of Vascular Access                      |           |
| AVF  | 78        |
| GF   | 24        |
| Type of Vascular Access at Start of Dialysis |           |
| Double Lumen catheter                        | 92        |
| AVF  | 8         |
| GF   | 2         |
| Failed Previous attempt                      | 43        |

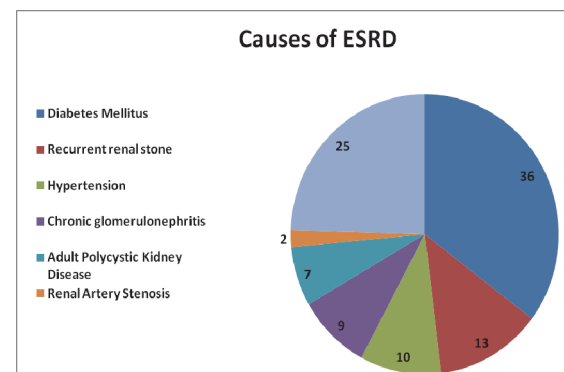
All data were analyzed using the Statistical Package for Social Science (SPSS); paired student t- test was calculated to assess differences among groups. Significance of association between various risk factors was assessed using Chi-square test. Level of statistical significance was set at < 0.05.

## RESULTS

Of 102 studied patients 45% were male. The characteristics of participant are summarized in Table 1. Plan of 50% of

participants were kidney transplantations. 90% of patients start haemodialysis through central venous line

The diagnosed causes of end stage renal diseases are, Diabetes Mellitus, unknown, renal stones, and uncontrolled hypertension in order of decreasing frequency as shown in Figure 1. While 25 % of patients with end stage renal failure still have undiagnosed etiology.



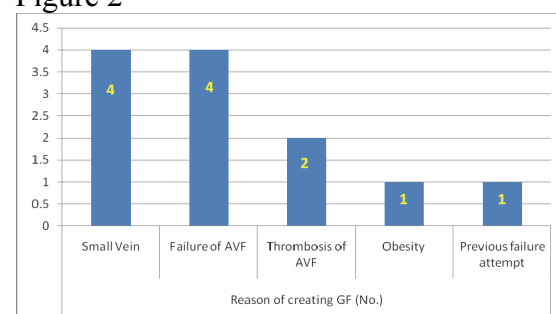
**Figure 1 Causes of ESRD in percentage**

The site and type of vascular access was forearm as the most common for AVF while arm AVG was the commonest as shown in table 2. Forearm vascular accesses were associated more with complications than arm accesses.

**Table 2. Location and Complication of Vascular Access**

| Type and Location | No. | Complication No. (%) |
|-------------------|-----|----------------------|
| Forearm AVF       | 52  | 31 (60)              |
| Arm AVF           | 26  | 13 (50)              |
| Forearm AVG       | 7   | 4 (57)               |
| Arm AVG           | 17  | 7 (41)               |

Twenty four patients were with graft fistula and the most common reason for creating AVG was small veins as shown in Figure 2



**Figure 2. Reason of creating Graft Fistula**

Complication observed in the studied patients with vascular accesses were edema, aneurysm, thrombosis and mixed complications as shown in table 3

Table 4.1 and 4.2 shows the complication of vascular accesses by gender. Edema was the most common complication among those with AVF

especially among females, while thrombosis was the commonest complication among those with GF, again the females more affected.

Table 5 shows the differences in complications among those with AVF and GF in regard of duration of hemodialysis

**Table 3. Complication of vascular access**

| Type of complications | AVF No. (%)     | GF No. (%)     | Total No. (%)   | P Value  |
|-----------------------|-----------------|----------------|-----------------|----------|
| Edema                 | 18 (23)         | 4 (16.7)       | 22 (23)         | 0.2522   |
| Aneurysm              | 11(14)          | 0              | 11 (12)         | 0.02572  |
| Thrombosis            | 6 (7.6)         | 6 (25)         | 12 (9)          | 0.005122 |
| Insufficient flow     | 2 (3)           | 0              | 2 (2)           | 0.2141   |
| HF                    | 4 (4)           | 0              | 4 (3)           | 0.1293   |
| Bleeding              | 1 (1)           | 0              | 1 (1)           | 0.2886   |
| Infections            | 0(0)            | 3 (12.5)       | 3(3)            | 0.001527 |
| Mixed complications   | 4 (5)           | 1( 4.2)        | 5 (5)           | 0.4243   |
| <b>Total</b>          | <b>46 (59%)</b> | <b>14(58%)</b> | <b>60 (59%)</b> |          |

**Table 4.1 Complication of vascular access (AVF) by gender**

| Type of complications | AVF       |            | P Value |
|-----------------------|-----------|------------|---------|
|                       | Male No.  | Female No. |         |
| Edema                 | 5         | 13         | 0.02410 |
| Aneurysm              | 8         | 3          | 0.06238 |
| Thrombosis            | 4         | 2          | 0.2163  |
| Insufficient flow     | 1         | 1          | 0.4853  |
| HF                    | 4         | 0          | 0.02268 |
| Bleeding              | 0         | 1          | 0.1518  |
| Mixed complications   | 2         | 2          | 0.4790  |
| <b>Total</b>          | <b>24</b> | <b>22</b>  |         |

**Table 4.2 Complication of vascular access (GF) by gender**

| Type of complications | GF       |            | P Value |
|-----------------------|----------|------------|---------|
|                       | Male No. | Female No. |         |
| Edema                 | 0        | 4          | 0.1031  |
| Aneurysm              | 0        | 0          |         |
| Thrombosis            | 2        | 4          | 0.2931  |
| Insufficient flow     | 0        | 0          |         |
| HF                    | 0        | 0          |         |
| Bleeding              | 0        | 0          |         |
| Infections            | 0        | 3          | 0.2864  |
| Mixed complications   | 0        | 1          | 0.2777  |
| <b>Total</b>          | <b>2</b> | <b>12</b>  |         |

Table 5. Complication of vascular access by duration of Hemodialysis

| Comp              | Less than 2 yr | More than 2 yr dialysis | P value  |
|-------------------|----------------|-------------------------|----------|
| Edema             | 11             | 11                      | 0.05159  |
| Aneurysm          | 3              | 8                       | 0.002975 |
| Thrombosis        | 10             | 1                       | 0.02710  |
| Insufficient flow | 1              | 1                       | 0.3301   |
| HF                | 0              | 3                       | 0.008645 |
| Bleeding          | 1              | 0                       | 0.2290   |
| Infections        | 3              | 0                       | 0.008645 |
| Multiple          | 1              | 4                       | 0.01597  |
| <b>Total</b>      | <b>30</b>      | <b>28</b>               |          |

## DISCUSSION

The diagnosed etiology of end stage renal disease in our study revealed that diabetes mellitus is the major cause of renal failure followed by renal stones followed by hypertension 35 %, 13 % and 10% respectively which are not comparable to other study (chronic glomerulonephritis and chronic Pyelonephritis) while unknown causes is still same around 25 %.<sup>10</sup> Another study in Brazil revealed renal hypertension the main cause (40.5 %) of end stage renal disease.<sup>11</sup>

Incidence of complications in AV fistula is relatively higher than GF which is not compatible to other studies which mentioned the GF as higher complications rate than fistula.<sup>4,5,12-14</sup> This is may be due to poor planning for permanent access (poor vessels for cannulation) in comparison to direct cannulation of graft adding unskilled nurse in cannulation and poor care of vascular access.

About 56 percent of patients with vascular access developed complications which are high rate of complications in our study which increase the morbidity, hospitalization and cost in treating patients with haemodialysis, but still near to other published data.<sup>15</sup>

Ninety percent of new ESRD patients in our study initiate HD with a catheter (temporarily or semi permanent) and only 10% with an AVF, this is very low in comparison to other studies and

guidelines.<sup>4,12-17</sup> Our explanation is that majority of our patients with chronic kidney disease are being seen by nephrologists with insufficient time to arrange for the creation and maturation of an AVF (time of diagnosis and time of initiation of dialysis was short and patient in need in urgent or semi urgent), another issue may be due to The substantially lower number of vascular access surgeons and hours devoted to vascular access surgery, long time table for creation vascular access creation, which is comparable to study in united state and Europe.<sup>17</sup>

In our study majority of vascular access is A-V fistula (76.5) which is comparable to European and American guidelines which strongly recommend creation of autogenous fistulae as the dialysis access of choice in patients requiring hemodialysis.<sup>4,11-18</sup>

Outcome and/ or complications according to gender it seems to be equal in both sexes in AV fistula type of access but its more than four folds in women with graft fistula comparing to men with graft fistula, majority of our patients with graft fistula were females (75%) and the most common reason for creating AVG was small veins this is comparable to other study.<sup>15,18,19</sup>

DOPPS III data (2005-2007) indicate 4-13% of HD patients using synthetic or bovine grafts in all countries except the united state which is less than in our study



where patients with GF 23.5 % of patients with vascular access.<sup>16,17</sup>

Aneurysm is one of important complications in our study regarding patients with AVF (14 %) and it forms 30 % of complications in patients with native AVF, while no aneurysm recorded in patients with GF. Which is not compatible to other studies,<sup>5,8,12,13</sup> and study done in united state showed that aneurysm in patient with graft fistula is rare complication.<sup>20</sup>

Other complications likes thrombosis and insufficient flow which are the major cause for the vascular access failure occur in about 13.7% of all patients with vascular access it end with vascular access failure where no periodic fallow up and flow assessment done in our center or other diagnostic procedures like angiography which help in detection of early vascular or graft stenosis and low flow rate as recommended.<sup>4,5,12,15</sup>

Infection complicating the vascular access in our study only 3 cases (12.5%) reported in patients with graft fistula and no infection in AVF reported which support that graft fistula has higher rate of infection and need careful aseptic handling of the vascular access during cannulation.<sup>8,11,12,16,19</sup>

Finally we recommend early referral of patients for permanent vascular access, arm preservation (vein and artery) for fistula creation, and follow up of vascular access with periodic blood flow measuring by Doppler ultrasound.

## REFERENCES

1. J. Goddard, A.N. Turner, A.D. Cumming and L.H. Stewart. Kidney and urinary tract disease (Chapter 17). In: Nicholas A. Boon, Nicki R. Colledge, Brian R. Walker and John A. A. Hunter, editors. Davidson's principles and practice of medicine, part 2 Practice of medicine. Churchill Livingstone; 20 edition; 2006. P 458-60.
2. Nina Tolkoff-Rubin. Treatment of irreversible renal failure (Chapter 133). In Section XI – Renal and Genitourinary Diseases. In: Lee Goldman, Dennis Ausiello, editors. Cecil Medicine. Philadelphia: Saunders Elsevier; 23rd edition ;2007.
3. Fauci. Braunwald. Kasper. Hauser Longo. Jameson. Loscalzo. Harrison's principles of internal medicine. Disorders of the kidney and urinary tract. (Part 12) Disorders of the kidneys and urinary tract, (Chapter 275) Dialysis in the Treatment of Renal Failure. United States of America: McGraw-Hill Companies; 2008, 17th edition.
4. Richard Fluck, Mick Kumwenda. Clinical practice guidelines vascular access for haemodialysis. UK Renal Association, Final Version (5.01.11), 5th Edition, 2008-2011.
5. Toros Kapoian, Jeffrey L. Kaufman, John Nosher, Richard A. Sherman. Dialysis Access and Recirculation (Chapter 5). In: William L Henrich and William M. Bennet (Volume 5, Section I: Dialysis as Treatment of End-Stage Renal Disease ).In: Robert W. Schrier. Series Editor. Atlas of Diseases of the Kidney. On-line edition, by ISN Informatics Commission and NKF cyberNephrology. Figure 5.1. available from: <http://www.kidneyatlas.org/toc.htm#vol5>
6. D Bonucchi, A. D'Amelio, M. Grosoli, A. Baraldi and G. Cappelli. Vascular access for haemodialysis. Surgical procedure to an integrated therapeutic approach. Nephrology Dialysis Transplantation. 1998, Volume 13, Issue 7; P 78-81.
- A. Kostakis, D. Mantas. Vascular Access to patients in Hemodialysis (Chapter 13.1). In: C.D.Liapis, K.Balzer, F. Benedetti-Valentini, J. Fernandes, Editors. Vascular Surgery , European Manual



- of Medicine. Springer –Verlag Berlin Heidelberg; 2007, P 587-94.
7. Gerald Beathard. A Practitioner's Resource Guide to Physical Examination of Dialysis Vascular Access. Fistula First National Vascular Access Improvement Initiative. Distributed by: End Stage Renal Disease Network of Texas November 2003, P 9-11.
8. Harry Schanzer and Milan Skladany. Vascular Access for Dialysis (Chapter 77) In: Henry Haimovici, Enrico Ascer, Larry H. Hollier, D. Eugene Strandness, Jonathan B. Towne, Haimovici's Vascular Surgery Principles and Techniques, United States of America; Blackwell Science, 4th Edition, 1996. P-1038.
9. Mark A. Little, Aisling O'Riordan, Brian Lucey, Michael Farrell, Michael Lee, Peter J. Conlon and J. Joseph Walshe. A prospective study of complications associated with cuffed, tunneled haemodialysis catheters. *Nephrology Dialysis Transplantation*, (2001) 16: 2194-2200.
10. Guilherme Centofanti, Eliane Y. Fujii, Rafael N. Cavalcante, Edgar Bortolini, Luiz Carlos de Abreu, Vitor E. Valenti, et al. An experience of vascular access for hemodialysis in Brazil. *International Archives of Medicine* 2011, No. 4, P-16.
11. Hung Michael Choi, Brajesh K. Lal, Joaquim J. Cerveira, Frank T. Padberg, Michael B. Silva, Robert W. Hobson and Peter J. Pappas. Durability and cumulative functional patency of transposed and non transposed arteriovenous fistulas. *Journal of vascular surgery*, December 2003, Volume 38, Number 6; P-1206.
12. O. EL Minshawy, T. Abdel Aziz, H. Abdel Ghani. Evaluation of vascular access complications in acute and chronic hemodialysis, *The Journal of Vascular Access* 2004; no. 5: P 76-82.
13. V.D. Nenov and G.P. Stefanov. Our Experience with Tunneled Cuffed Hemodialysis Catheters. *Bantao Journal* 2007: Vol. 5 no. 1: P 16-18.
14. Chih-Ching Lin, Wu-Chang Yang. Prognostic Factors Influencing the Patency of Hemodialysis Vascular Access. *J Chin Med Assoc* March 2009; Vol. 72. No.3: P 109–116.
15. Maria Goretti Penido. Vascular Access for Hemodialysis, Technical Problems in Patients on Hemodialysis, In Tech, Available from: <http://www.intechopen.com/books/technical-problems-in-patients-on-hemodialysis/vascular-access-for-hemodialysis>. Technical Problems in Patients on Hemodialysis / Edited by Prof. Maria Goretti Penido / ISBN 978-953-307-403-0 / Hard cover, 312 pages / Publisher InTech / Published online 07, December, 2011 / Published in print edition December, 2011.
16. Jean Ethier, David C. Mendelssohn, Stacey J. Elder, Takeshi Hasegawa, Tadao Akizawa, Takashi Akiba, Bernard J. Canaud and Ronald L. Pisoni. Vascular access use and outcomes: an international perspective from the dialysis outcomes and practice patterns study, *Nephrology Dialysis Transplantation*, 2008. Vol. 23: P 3219–26.
17. Christopher D. Miller, Michelle L. Robbin, and Michael Allon. Gender differences in outcomes of arteriovenous fistulas in hemodialysis patients. *Kidney International*, 2003 Vol. 63, P 346–52.
18. Dirk M. Hentschel. Vascular Access for Hemodialysis, *Nephrology Rounds*. January 2008, Vol. 6, Issue 1, P 304-59.
19. Lauren R. Pandolfe, B.A. Angelo P. Malamis, Kenneth Pierce and Marc A. Borge. Treatment of Hemodialysis Graft Pseudoaneurysms with Stent Grafts: Institutional Experience and Review of the Literature. *Seminars in International Radiology*, 2009. vol. 26, no. 2, P 89-95.

## پوخته

## نهجامین پیگههین خوینی بۆ نهخووشین دهیلهزا خوینی

## ل سهنته ری دهوک بی نهخووشین گولجیسکا

**پیشهکی و ئارمانجین فهکولینی:** دوو ریک هه نه بۆ جاره سه ریا نهخووشیا گولجیسکا ژکارکهفتی، چاندنا گولجیسکی یان دهیلهز (یا خوینی یان یا پیریتوونی). دهیلهزا خوینی پیتقی ب پیگههکا خوینی یا باش، بی ئازار، ب ئاسان بۆ بکارئینانی، ب ژیهکی دوومدریژ، وبگیمترین موزاعهفات. ههول ددهین خاندن وشرووفه کرنا سه د و دوو (۱۰۲) نهخووشان بکهین، دگهل ههلسهنگاندنا پیگههین وان یین خوینی. مه رهم ژ فهکولینی: بهراوه ریکرنا جورین جوداجودا یین پیگههین خوینی، ههلسهنگاندنا کارکرنا وان، دوومدریژیا ژیهی پیگههان، موزاعهفاتین پیرا دیاردین.

**ریکین فهکولینی:** ئەف فهکولینه فهکولینهکا پاشنیرین و پیشنیرینه، بۆ وان نهخووشان ئەفین پیگههین خوینی بۆ هاتینه چیکر ل پارێزگهها دهوک هه ر ژ خزیرانا ۲۰۰۹ ی تا خزیرانا ۲۰۱۳ ی ل سهنته ری دهوک بی نهخووشین گولجیسکا.

**ئهجام:** سه د و دوو نهخووش کهفتنه دفی فهکولینی دا، نهخووشیا شهکری ئەگه ری ژ خر ئەگه ران پتر دیاربوو بۆ نهخووشیا گولجیسکا ژکارکهفتی دفی فهکولینی دا. ۷۴٪ ژ نهخووشان ناسوورا بوریهخوونقهگێر وبوریهخوونبرا هه بوو، بجویکیا بوریهخوونقهگێر ئەگه ری هه ره سه رهکی بوو بۆ چیکرنا ناسوورا بریکا گرافتی پیشچیکری. دیارترین موزاعهفات دگهل ناسوورا بوریهخوونقهگێر وبوریهخوونبرا وهرماندنوو، ههروهها دیارترین موزاعهفات دگهل ناسوورا بریکا گرافتی پیشچیکری ههشکبوونا خوینی بوو د گرافتی دا.

**دهرئهجام:** بتنی ۱۰٪ ژ نهخووشان ئەفین هاتینه فهکولین، هاتبوو بۆ چیکرنا پیگههی خوینی بهری کو ئەوان پیویستی ب دهیلهزهکا بلهز ههبت. خویابوو کو موزاعهفات وکی ئیک دیاردبوون ل دهف ژن وزهلامان، ئەفین ناسوورا بوریهخوونقهگێر وبوریهخوونبرا هه ی، بهلی دیاربووونا موزاعهفاتا جوارجاران پر دیاردبوو ل دهف ژنان پر ژ زهلامان بۆ وان نهخووشان ئەفین ناسوورا بریکا گرافتی پیشچیکری هه ی. ریژا ژیکهاته ده ر و فرهههینا ده مارین خوینی ل دهف وان نهخووشین ناسوورا بوریهخوونقهگێر وبوریهخوونبرا هه ی، پتر بوو دفی فهکولینیدا ب بهراوردی دگهل فهکولینین دی.

## الخلاصة

### نتائج الموصلات الدموية للمرضى الخاضعين للديليزة الدموية في مركز دهوك لأمراض الكلى

**خلفية وأهداف البحث:** يوجد خيارين اثنين لاستمرارية الحياة لمرضى عجز الكلية وهما زرع الكلية أو الديليزة (الدُموية أو البريتونية)، الديليزة الدموية تحتاج الى موصل دموي كفؤ، مريح، سهل الاستعمال يعمل لأطول فترة ممكنة مع أقل المضاعفات، سنحاول دراسة وتحليل ١٠٢ من المرضى وتقييم الموصلات الدموية لديهم. الهدف من الدراسة هو مقارنة الأنواع المختلفة من الموصلات وتقييم كفاءتها، العمر الزمني والمضاعفات المصاحبة لها.

**طرق البحث:** هذه دراسة استيعادية ومسبقية للمرضى الذين اجريت لهم تصنيع الموصلات الدموية في محافظة دهوك من حزيران ٢٠٠٩ ولغاية حزيران ٢٠١٣ في مركز دهوك لأمراض الكلى.

**النتائج:** تم شكل مائة اثنين من المرضى ا في هذه الدراسة. وكان داء السكر السبب الأكثر شيوعا في المرضى الذي يعانون من الفشل الكلوي في المجموعة التي شملتها الدراسة ٧٦,٤% من المرضى لديهم ناسور شرياني وريدي. كان الاوردة الصغيرة السبب الأكثر شيوعا لإنشاء ناسور شرياني وريدي بمجازة اصطناعية. وكان أشيع المضاعفات هي الوذمة في ناسور شرياني وريدي بينما كان التخثر الاكثر شيوعا في ناسور شرياني وريدي بمجازة اصطناعية.

**الاستنتاجات:** ١٠% فقط من عينة الدراسة تقدموا لخلق موصل الاوعية الدموية قبل أن يحتاجوا لغسيل الكلى العاجل. يبدو أن المضاعفات على قدم المساواة في كلا الجنسين في نوع ناسور شرياني وريدي من الوصول ولكنها أكثر من أربعة أضعاف في النساء مع ناسور شرياني وريدي بمجازة اصطناعية. تمدد الأوعية الدموية في المرضى الذين يعانون من ناسور شرياني وريدي عالية في هذه الدراسة في مقارنة مع غيرها من الدراسات.

## AUTISM SPECIFIC PEDAGOGIC INTERVENTION (ASP); CASE REPORT

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*Submitted 16 May, 2013; accepted 30 Dec. 2014*

## SUMMARY

Autism Spectrum Disorder is an increasingly diagnosed developmental disorder with a clinical picture consisting of deficit social interaction, inadequate speech, and stereotype behavior. It has unknown etiology, chronic course, and dire psychosocial consequences both on the affected child and his family. Several treatment methods have been applied with controversial outcomes and no curative effect. Based on cognitive behavior therapy, Autism Specific Pedagogic intervention (ASP) was developed at the Metin Health House (a private clinic for child mental health supported by the Investment Law). In a 2-month individual plan with measurable aims the ASP was applied to improve social interaction for two autistic boys aged 3 and 4 years. In the first plan, the first boy was taught to respond to his name. Then, both patients were trained in individually adjusted sessions to learn words and use them in meaningful sentences. Significant improvement was achieved according to the daily structured assessment of the therapists confirmed by the reports of the parents. The results are promising regarding a pedagogic use of cognitive behavioral therapy in early diagnosed Autism.

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**Key words:** Autism treatment, CBT, Investment Law, Social Interaction, Speech therapy

Autism is a developmental disorder that was classified first by Leo Kenner in 1943<sup>(1)</sup>. It has been continuously updated through intensive research due to its severe consequences on the affected child throughout the life span. The parents usually become exhausted, and the family system usually suffers in seeking adjustment to the non-functional system of the autistic child. The diagnostic criteria have been recently revised in the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) collecting all previous subtypes to only one and the same diagnose Autism Spectrum Disorder (ASD)<sup>(2)</sup>. The worldwide prevalence of ASD is increasing, estimated to be about 6 per 1000 children in 2012<sup>(3)</sup>. While DSM does not outline recommended treatment and services for mental disorders, determining

an accurate diagnosis is a first step for a clinician in defining a treatment plan for a patient. In the Kurdistan region of Iraq, the first diagnosis of Autism was confirmed in 2008 at the reception of the Metin Health House (MHH), a private clinic supported by the Investment Law to provide preventive and treatment services concerning child mental health from gestation to 18 years of age.

Despite comprehensive research, no certain etiology or curative treatment has been identified for ASD, yet. Although specific medication has been successfully used for short term symptomatic treatment, it is usually accompanied with high rates of side effects<sup>(4)</sup>. While some controlled studies suggest Hyperbaric Oxygen Therapy (HBO) to be effective for treatment of ASD (5), a recent review shows insufficient evidence to draw

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conclusions on the efficacy of any complementary or alternative medicine, including the HBO<sup>(6)</sup>. More recent research suggests systemic multimodal approach of speech therapy<sup>(7)</sup>, melodic based communication therapy<sup>(8)</sup>, and Cognitive Behavioral Therapy (CBT) to be valid forms of intervention for treatment of ASD<sup>(9)</sup>. According to our knowledge, no pedagogic application of CBT has been used for treatment of autism in children, yet. Our case report demonstrates the use of such a pedagogic technique based on CBT.

### **CASE PRESENTATION**

During February 2013, 2 boys aged 3 and 4 years fulfilled ASD criteria after investigation at the MHH, both having deficit social response and lack of speech as chief complaints. The first boy was first child to his parents, while the second boy was nr 6/6 siblings. Each of them was living in a complete family with stable conditions and good economic status. The parents of both children were university educated and state employed. The mothers were forced to stay at home from job to take care of the child, while the fathers could retain employment. Neither the first nor the second boy showed any psychiatric or physical co-morbidity. No complication was found during pregnancy, delivery or neonatal period. All the investigations revealed normal findings. Both boys were free from medication during at least 1 month before starting the intervention.

### **INTERVENTION**

Problem analysis with the parents according to the CBT principles identified deficit social interaction, and lack of speech among the most urgent problems to be addressed in the treatment plan. The parents of the first boy put “responding to his name” at the top of the priority list to be focused in the treatment. Another priority for both boys addressed by the parents was “learning meaningful words”. An individual plan was agreed on by the

child psychiatrist and the parents of the first child to start targeting the first goal of treatment directly after the diagnosis. An Autism Specific Pedagogic technique (ASP) was developed based on CBT principles. It was conducted by an experienced pedagogue at the MHH during February–April, 2013. The intervention composed 10-20 sessions per week, each consisting of 1–2 hours distributed over the week in collaboration with the caregiver of the child. The same pedagogue used, then, the same technique and procedure to treat the second goal for the first child, while a trained nurse assistant applied ASP simultaneously on the second boy during April–June, 2013.

The parents took responsibility for bringing the children to treatment at the MHH. They participated in the intervention whenever the therapist asked them to do. A special instrument called Daily Assessment Schedule (DAS) was developed to be applied by the therapist to measure the progress in social response and speech development during the therapy. The parents were spontaneously reporting observation at home during the treatment period.

### **RESULTS**

The first boy showed increasing response rates when the therapist called him by his name. The progress was increasing even after the initial reinforcement was withdrawn (Figure 1).

The reinforcement was gradually withdrawn when the response to the name was increasing until no reinforcement was needed any more at the middle of the intervention period.

Both boys showed significant increase in vocabulary (first boy from 13 to 41 words, and second boy from 32 to 95 words) (Figure 2 and 3). The second boy also showed an increasing tendency to use meaningful sentences consisting of maximal 4 words, while the first boy only learned to use up to 2 words in short meaningful sentences. Both boys gained

observable improvement in communication with the surrounding according to the parents who were

positively surprised to the changes of their boys' social interaction skills.

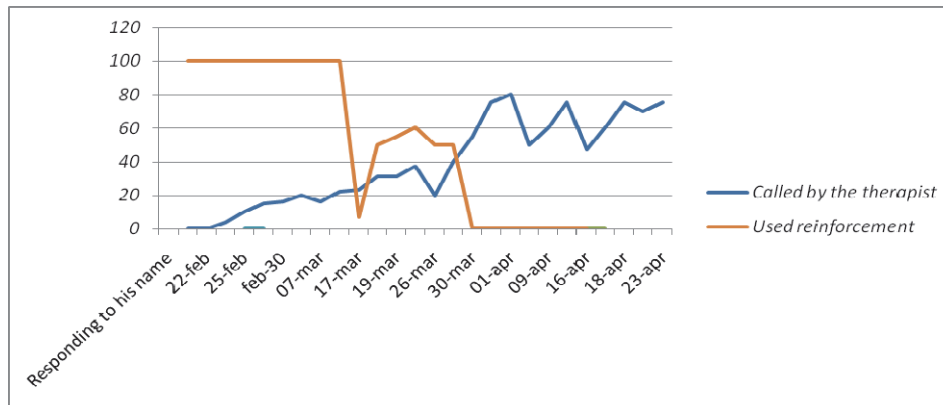


Figure 1. Frequency of the first boy's response to his name, with and without positive reinforcement.

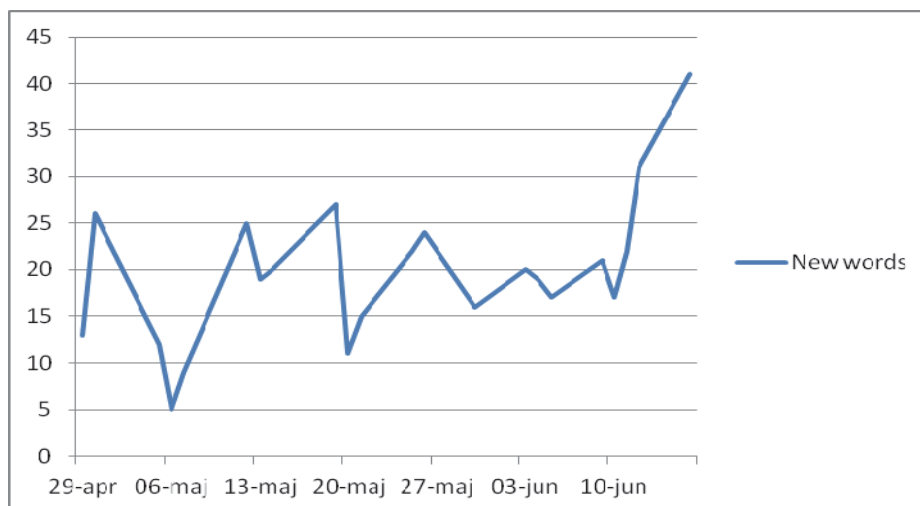


Figure 2. Number of new words in the first boy during the treatment.

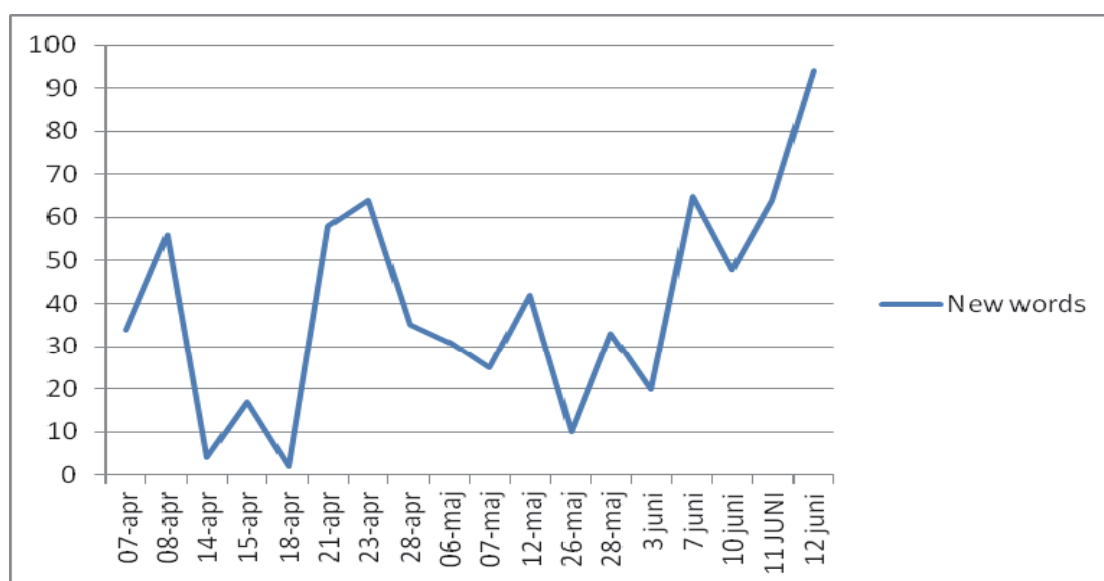


Figure 3. Number of new words in the second boy during the treatment.

## DISCUSSION

A pedagogic technique based on CBT principles was successfully applied to teach a 3 year-old boy to respond to his name, and to elicit speech both in this boy and another one aged 4 years, both of them suffering from ASD as only diagnosis. This application is the first kind of structured intervention using CBT in a pedagogic intervention as far as we know. The results confirm recent research suggesting multimodal speech therapy, rhythmic communication, and CBT for treatment of ASD<sup>(7-9)</sup>. Furthermore, this case report suggests pedagogic approach in applying CBT principles to constitute an important therapeutic element for the core pathology of ASD. Early age at the time of diagnosis and treatment, and the severity of symptoms pushing the parents to seek help so early, might be positive factors contributing to the effectiveness of this type of intervention. Being one year older than the first boy, the second boy gained more speech skills than the first one. This indicates the normalizing characteristics of the ASP intervention. One might even think of the possibility for both boys to have developed normal speech and social interaction at this age if they were free from ASD. Again this might suggest the ability of the ASP technique to normalize the condition of the autistic child by attacking its core pathology. Responding to the name confirms the specific effect of this type of intervention for Autism, as it controls one of the most important core symptoms of ASD. These findings generate a hypothesis telling that autism specific pedagogic intervention (ASP) constitutes a new model of treatment for ASD which also contributes to the knowledge about the etiology and pathogenesis of this mysterious condition. However, this is to be confirmed in more rigorous research in the future.

## REFERENCES

1. Sadock BJ, Sadock VA. Kaplan and Sadock's Synopsis of Psychiatry, 10th ed. Philadelphia: Lippincott Williams and Wilkins; 2007. p. 1191-9.
2. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (DSM-V), 5th ed. Washington DC: American Psychiatric Association; 2013. p. 1269.
3. Williams JG, Higgins JPT, Brayne CVG. Systematic review of prevalence studies of autism spectrum disorders. Arch Dis Child. 2006; 91 (1): 8-15.
4. Caccia S. Safety and pharmacokinetics of atypical antipsychotics in children and adolescents. Paediatr Drugs. 2013; 15 (3): 217- 33.
5. Sampanthavivat M, Singkhwa W, Chaikyul T, Karoonyawanich S, Ajpru H. Hyperbaric oxygen in the treatment of childhood autism: a randomised controlled trial. Diving Hyperb Med. 2012; 42 (3): 128- 33.
6. Whitehouse AG. Complementary and alternative medicine for autism spectrum disorders; Rationale, safety and efficacy. J Paediatr Child Health. 2013; 49 (9): 438- 42.
7. Tamas D, Marković S, Milankov V. Systemic multimodal approach to speech therapy treatment in autistic children. Med Pregl. 2013; 66 (5-6): 233- 9.
8. Sandiford GA, Mainess KJ, Daher NS. A pilot study on the efficacy of melodic communication therapy for eliciting speech in nonverbal children with autism. J Autism Dev Disord. 2013; 43 (6): 1298- 307.
9. Danial JT, Wood JJ. Cognitive Behavioral Therapy for Children with Autism: Review and Considerations for Future Research. J Dev Behav Pediatr. 2013; 34 (9): 702-15.



## پوخته

## چارهسهریه‌کا په‌روه‌دا تاییه‌ت بو خوه‌ییس‌م، رایۆرتا دوو‌حاله‌تا

خوه‌ییس‌م (ئوتیس‌م) نه‌ساخیه‌کا وه‌رارا زاروکا‌یه. نیشانن وی ئه‌فه‌نه: لاوازییا تیکه‌لییا جفاکی، کیماسیا په‌یوه‌ندیین ب گوتنی، و ره‌فتاره‌کا بی مه‌ئنا. ئه‌گه‌ر نه‌دیاره. نه‌ساخی درێژخایه‌نه و کاریگه‌ریین خراب ل سهر زاروکی و خیزانا وی هه‌نه. چهند جورین چاره‌سهری یین هاتین جه‌رباندن لی چ ئه‌نجامین قوت‌بر نه‌په‌یدا‌بوینه. ل سهر بنه‌مایین چاره‌سهریا ره‌فتار و هزری میتوده‌کا نوی یا په‌روه‌دا تاییه‌ت ل ساخله‌مخانا مه‌تین (بنگه‌هه‌ک تاییه‌ت بو پاراستن و چاره‌سهرکرنا ساخله‌میا ده‌رونی یا زاروک و گه‌نجا ب پپی یاسا وه‌به‌ره‌ینانی) هات چیکرن و ب کارئینان. پلانه‌کا ۲ هه‌یقی دگه‌ل ئامانجین پیقانه‌بر ل سهر ۲ کورکین ۳ سالی و ۴ سالی یین نه‌ساخیا خوه‌ییس‌م (ئوتیس‌م) هه‌ین ئه‌نجامین ئه‌رینی نیشادان.

## الخلاصة

### تداخل علاجي تربوي خاص بالتوحد، تقرير حالتين

مرض التوحد يصيب تطور الطفل فيما يتعلق بالتواصل الاجتماعي، والتكلم والسلوك. الأسباب غير معلومة، والمرض مزمن ذو تأثير سلبي على الطفل وعائلته. لحد الآن أعطت عدة وسائل علاجية نتائج متناقضة ولا يوجد علاج شاف. بناءً على نظرية العلاج الإدراكي سلوكي إختترعت وسيلة تربوية خاصة في بيت متين الصحي (مركز خصوصي لوقاية ومعالجة الصحة النفسية للأطفال والشباب وفق قانون الإستثمار) وأعطت نتائج إيجابية عند تطبيقه على ولدين ٣ و ٤ سنوات من العمر يعانون من التوحد.

## CARDIAC HYDATID CYST WITH MULTIPLE ORGANS INVOLVEMENT

MUSHEER A. GORAN, MBChB, DMRD\*

*Submitted 28 Ap. 2013; accepted 30 Dec 2013*

## SUMMARY

A 17 years old female referred to radiology department with hepato–splenomegaly and a chest X–ray with multiple high dense masses with rounded outline. Computerized Tomography scan done for the patient and showed multiple similar rounded masses in the liver, spleen and right lung of different size and single one in wall of left ventricle of the heart measuring 42 mm by 41 mm in diameter. This report is the first case report from Iraq of cardiac hydatid cyst with multiple hydatid cysts of lung, liver and spleen.

Duhok Med J 2013;7(2): 47-51.

**Key words:** Hydatid cyst, x-ray, cardiac hydatid

**H** ydatid cyst is endemic in various regions of the world. Unlike liver and pulmonary involvement, cardiac echinococcosis is rare with an incidence of 0.2 to 2%.<sup>1</sup> Pulmonary and cardiac cysts require various methods for differential diagnosis because conventional radiography is inadequate. On the other hand, computed tomography CT scan is superior for differential diagnosis by demonstrating fluid in the cystic lesion. Echo-cardiography, magnetic resonance imaging (MRI) and conventional angiography are other tools for the diagnosis of cardiac echinococcosis.<sup>2,3</sup>

## CASE REPORT

A 17 years old female patient was referred to radiology department for CT scan following an abdominal ultrasound suggesting a hydatid cyst in the liver and spleen. A chest X–ray showed multiple opacities in the right lung. The CT of the abdomen and chest revealed multiple cysts of right lung, and liver, and a large cyst in the spleen and another one in the wall of left ventricle of heart (Figure 1).

## DISCUSSION

Human echinococcosis is zoonotic infection caused by tapeworm of the genus echinococcosis. Hydatid cyst is endemic in most sheep raising countries in Asia, Europe, South America, New Zealand and Australia.<sup>4,5</sup> Exposure to food and water contaminated by the feces of an infected host or poor hygiene can lead to echinococcosis.<sup>4</sup> Of the 4-four known species echinococcosis, echinococcosis granulosis is the most important and causes cystic echinococcosis (CE). The incidence of CE in endemic areas ranges from 1–220 cases per 100,000 inhabitants.<sup>4</sup> Morbidity is usually secondary to free rupture of echinococcal cyst (with or without anaphylaxis), infection of the cyst or dysfunction of affected organs.<sup>5,6</sup> Examples of dysfunction of affected organs include: biliary dysfunction, cirrhosis, bronchial obstruction, renal outflow obstruction, increased intracranial pressure secondary to mass effect and hydrocephalous secondary to cerebrospinal fluid outflow obstruction. Symptoms can be produced by mass effect or cyst complications. Symptoms due to pressure usually take long time to manifest, except when they occur in the brain or the eyes.

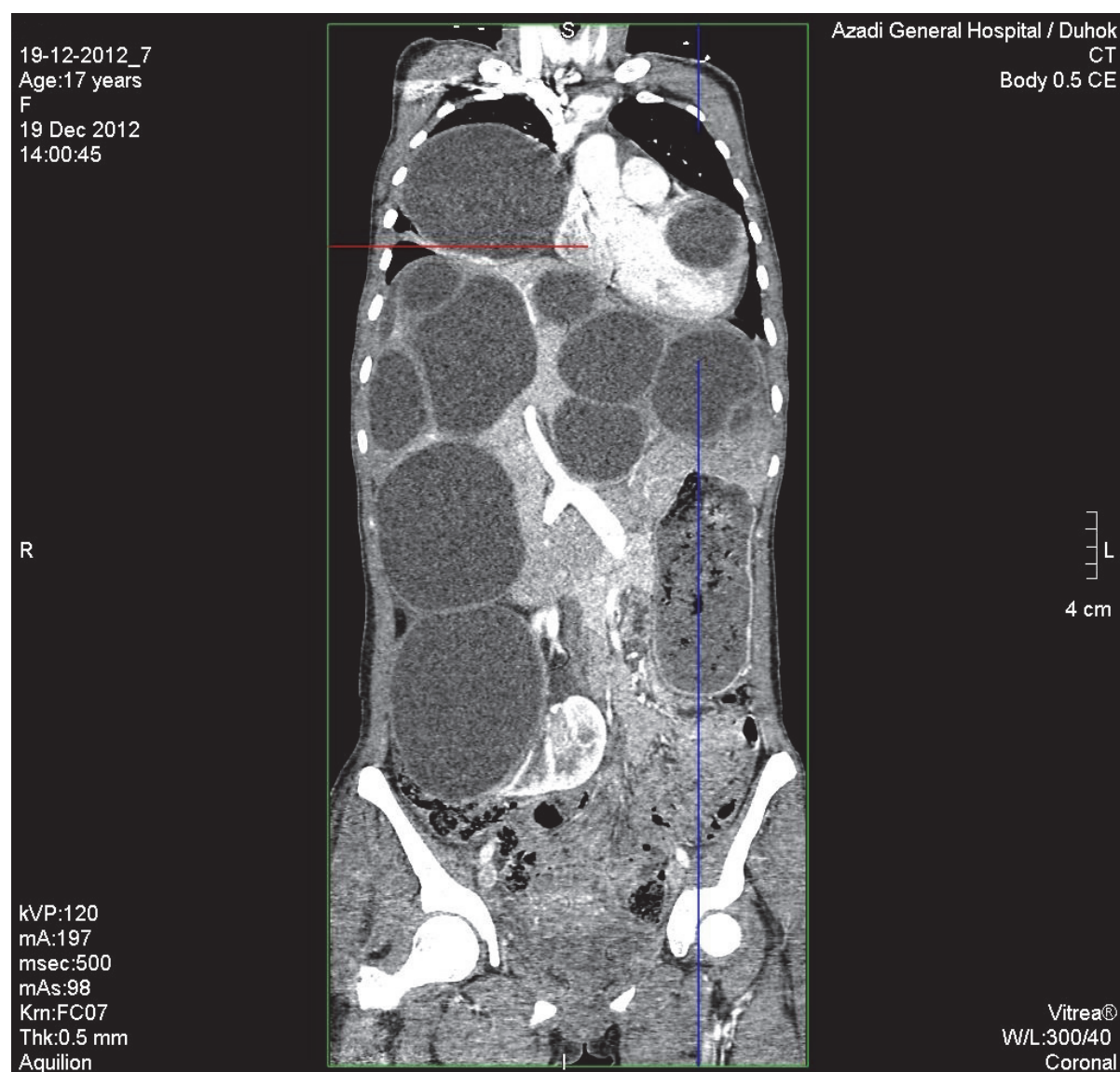
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Most of the cyst symptomatic cysts are larger than 5cm in diameter. Organs affected by *E. granulosus* are the liver (63%), lungs (25%), muscles (5%), bones (3%), kidneys (2%), brain (1%) and spleen (1%).<sup>7,8</sup>

Mortality is secondary to anaphylaxis, systemic complication of the cysts (sepsis, cirrhosis, respiratory failure) or operative complication.

Cardiac cyst has been reported in (0.2 to 2%). It could be seen in any age and sex, although it's more common in those with ages of 20 to 40 years and in females (M: F of 1.6/ 1).<sup>9,10,11,12,13,14,15</sup> The clinical presentation varies depending on the location, sizes and integrity of cardiac cyst.

Several studies reported that the most common locations of cardiac CE were the left ventricles (60%) and the ventricular septum (9-20%), but the right ventricle and right atrium can also be involved in (4 to 17%).<sup>3,9,10</sup> However, Sadeghpour and coworkers reported that the most common sites of cardiac involvement were the interventricular septum (46%), followed by the right atrium (15.3%), left ventricular free wall (15.3%), pericardium (7.7%), right ventricular free wall (7.7%) and left atrium (7.7%).<sup>11</sup> Cardiac hydatid cyst may be fatal due to complications such as cardiac failure, cyst rupture embolization.<sup>7,11</sup> Early diagnosis and surgical treatment are utmost importance.



**Figure 1. Cardiac hydatid cyst with multiple hydatid cysts of lung, liver and spleen.**

## REFERENCES

1. Akhtar MJ. Hydatid disease of the right ventricle and role of tomographic scanning in its diagnosis. *Int J cardiol*. 1991; 33: 432-4.
2. Cantonis, Frola C, Gatto R, Loria F, Tezzi MI, Vallebona A. Hydatid of the inter ventricular septum of the heart: MR findings. *AJR*. 1993; 161: 753-4.
3. Miralles, A, Bracamonte L, Pavie A, Bors U, Rabago G, Gandjbakhch I. Cardiac echinococcosis: surgical treatment and results. *J Thorac Cardiovasc Surg*. 1994; 107: 184-90.
4. Flisser A. Larval cestodes. In: Collier L, Balows A, Sussman M, editors. *Topley and Wilson's microbiology and microbial infections*. Parasitology. Vol. 5. 9th ed. New York: Oxford University Press 1998; P. 539-60.
5. Salih OK, Celik SK, Topuuoğlu MS, Kısacıkoğlu B, Tokcan A. Surgical treatment of hydatid cysts of the heart: a report of 3 cases and a review of the literature. *Can J Surg*. 1998; 41: 321-7.
6. Odev K, Aeikgozoglu S, Gormus N, Aribas OK, Kiresi DA, Solak H. Pulmonary embolism due to cardiac hydatid disease, Imaging Findings of unusual complication of hydatid cyst. *Eur Radiol*. 2002; 12: 627-33.
7. Elbeyli L, Kervancioglu R, Bayram M, Filiz A. Hydatid cysts with pulmonary and cardiac involvement. *Asian Cardiovasc Thorac Ann*. 1999; 7: 236-7.
8. Iglesias LF, Morales MZ, Mazcos G. Pericarditis secondary to the rupture of the hydatid cyst. *Rev. Esp Cardiol*. 1999; 52: 135-8.
9. Ben-Hamda K, Maatouk F, Ben-Farhat M, Betbout F, Gamra H, Addad F, Fatima A, et al. Eighteen-year experience with echinococcosus of the heart: clinical and echocardiographic features in 14 patients. *Int J Cardiol*. 2003; 91(2-3): 145-51.
10. Thamear H, Abdelmoula S, Chenik S, Beyn, Ziadi M Mestiri T et al., Cardiopericardial hydatid cysts. *World J Surg*. 2001; 25: 58-67.
11. Sadeghpour A, Nemati B, Arefi S, Raissi K, Vahedian J, Omrani G, Givtaj N, Hosseini S, Mollasadeghi G. 11 years experience with cardiac hydatid cyst operation at Rajaei Heart Center. *Iranian Heart Journal*. 2004; 5(4): 40-4.
12. Von Sinner WN, Linjawi T, Al Watban J. Mediastinal hydatid disease: report of three cases. *Can Assoc Radiol J*. 1990; 41: 79-82.
13. Miralles A, Bracamonte L, Pavie A. Cardiac echinococcosis: surgical treatment and results. *J Thorac Cardiovasc Surg* 1994; 107: 184-90.
14. Birincioglu CL, Bardakci H, Küçük SA. A clinical dilemma: cardiac and pericardiac echinococcosis. *Ann Thorac Surg*. 1999; 68: 1290-4.
15. Kaplan M, Demirtaş M, Cimen S, Ozler A. Cardiac hydatid cysts with intracavitary expansion. *Ann Thorac Surg*. 2001; 71: 1587-90.

## پوخته

## کورتہ فہکولین لسہر توشبوونا ہندہک کوئہندامین لہشی دگہل دلی ب نہخوشیا کیسکین ٹافی

ئہف نہخوشیہ ہاتیہ دیتن ل دہف نہخوشہکی کو توش ببو ب کیسکین ٹافی ل کوئہندامین دلی، سیہ، میلاک و تحیل ب ریکا ٹامیری پشکینا مفراسی ل دہمی پشکینہکا ئاسایی کو ہاتہ ئہنجام دان بو سنگ و زکی نہخوشی، کیسکین ٹافی ئیکہ ژ وان نہخوشیین بہر بہلاف ل عیراقی بشیوہیہکی گشتی و ہندہک دہقہرین ہریمما کوردستانی بشیوہیہکی تایبہت۔

کیسکین ٹافی ل کوئہندامی دلی ئیکہ ژ ہرہ کہیسین گہلہک کیم کو ریدہن و ئہفہ ئیکہمین کہیسہ کو دہیتہ تومارکرن ل نہخوشخانا ئازادی ل باژیری دہوکی ہر ژ وختی ئینانا ٹامیری مفراسی ل سالاً ۱۹۹۸۔

### الخلاصة

#### ملخص البحث لإصابة اعضاء متعددة في الجسم مع القلب بالأكياس المائية

تم اكتشاف حالة المريضة المصابة بكيس مائي في القلب عن طرق الفحص الروتيني بواسطة اشعة المفراس الحزوني للصدر والبطن في جلسة واحدة و تبين انها مصابة بكيس مائي في القلب بالاضافة الى الرئة والكبد والطحال، الاكياس المائية هي من الامراض الشائعة في العراق ومناطق معينة من اقليم كردستان. تعتبر الاصابة بالاكياس المائية في القلب من الحالات النادرة وهذه الحالة هي الاولى من نوعها التي يتم تشخيصها في مستشفى آزادي في مدينة دهوك منذ وصول جهاز المفراس الحزوني الى المحافظة سنة ١٩٩٨.



## CAROTID BODY TUMOR,SURGICAL RESECTION WITH SAPHENOUS VEIN INTERPOSITION GRAFT: A CASE REPORT

ABDULLAH S. ABDULLAH, MD, FICS, FACS\*

*Submitted 25 Nov 2013; accepted 30 Dec 2013*

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### SUMMARY

Paraganglioma or carotid body tumor is a rare asymptomatic,painless tumor, originate from paraganglionic body of autonomous nerve system of the neural crest cells in the periadventitia tissue of the carotid artery bifurcation .despite its gradual developing nature ,it is critical to reach early diagnosis and surgery for Carotid body tumor due to its potential of being malignant and local invasion or pressure on the adjacent neurovascular tissues.

I report here a case of carotid body tumor , presented with a large painless neck swelling, that treated surgically in the light of literature review.

In conclusions surgical resection is the treatment of choice for carotid body tumors(CBTs) .

Observation of these tumors is not recommended because of their potential for malignancy and progressive local growth that associated with increase risk of vascular and neurological deficits.Early surgical management is recommended to avoid these deficits due to tumor enlargement .

**Duhok Med J 2013;7(2): 52-61.**

**Key words:** Paraganglioma, Carotid body tumor, Surgery

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**T**he carotid body first described by Van Haller in 1743, is located in the periadventitial tissue, on the posteromedial aspect of the carotid artery bifurcation and embryologically is derived from the neuroectodermal tissue<sup>1</sup>. the normal gland measures 3-5 mm and weight's less than 15 gr. on average<sup>2</sup>. Locating of carotid body tumor within periadvantitial tissue of the carotid bifurcation is very important during surgical resection of these tumors, as dissection in the dipper planes of the carotid artery are associated with higher risk for complications from vessel injury<sup>3</sup>. The gland is highly vascular and receive its blood supply from feeder vessels running through the Mayer ligaments, primarily from the external carotid artery (ECA), typically the ascending pharyngeal artery .it is innervated by the Hearing nerve, originating from the Glossopharyngeal nerve<sup>4</sup>.

The carotid body works as a chemoreceptor organ stimulated by hypoxia, hypercapnia and acidosis. Although the etiology of carotid body tumor is not known<sup>5</sup>, but there is an increase incidence in populations living at high altitudes and in patients with chronic obstructive pulmonary disease (COPD), which has led to the hypothesis that chronic stimulation of the carotid body by hypoxia may predispose certain people to the development of these tumors<sup>6</sup>. Paraganglioma (PGL) is a rare tumor of the head and neck and has an incidence of about 0.012%,which arises from the neural crest cells. Histologically it is similar to the adrenal gland neoplasm, but usually they are non functioning tumors.

Carotid body tumors (CBTs) are the most common form of PGL of the head and neck<sup>7</sup>.

Although my patient's had a very big tumor(tumor sized 5x3 cm and Shamblin

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type III) They are generally benign, usually unilateral (95%); and there is no report of spontaneous regression<sup>5</sup>. CBTs occur at any age but are typically diagnosed between the third and sixth decades of life and the average age of onset is 45 years<sup>8</sup>. These tumors are usually sporadic, but a familial form can be seen in 10% of cases. the sporadic tumors are unilateral in 95%, but in familial form, the incidence of bilateral tumors increase to 30%<sup>9</sup>.

CBTs are found in both sides of the neck and both genders at the same rate of frequency<sup>10</sup>, but interestingly, the male/female ratio is higher (M/F=1/8.3) in patients dwelling at high altitude above 2000 meters than those patients dwelling at sea level (1/1.0-1.4)<sup>11</sup>.

The malignant rate of CBTs are between 5 and 10%<sup>(12)</sup>, but in young patients, this rate is higher, up to 30%<sup>5,12</sup>.

Malignancy is determined by the detection of metastases in local lymph nodes or distant organs, such as the lungs, bones, liver, pancreas, breast, rather than by the histological criteria or development of malignancy in neoplasms. the incidence of local or distant metastases is less than 10%. Carotid body tumors are most commonly presented as an asymptomatic palpable neck mass in the anterior triangle of the neck. they are slowly growing tumors. On examination, the mass is vertically fixed because of its attachment to the bifurcation of carotid artery (Fontaine sign). Approximately 10% of cases of CBTs present with cranial nerves palsy of the hypoglossal, Glossopharyngeal, recurrent laryngeal, or spinal accessory nerves, or involvement of the cervical sympathetic chain. Enlarged CBTs compress the carotid artery and the surrounding nerves, may therefore be associated with pain, hoarseness, dysphagia, Horner syndrome, or shoulder drop<sup>13</sup>.

Based on the tumor size, a surgical classification for CBTs was proposed by Shamblin in 1971; Group I: tumors are relatively small with minimal attachment to the carotid vessels, and surgical excision can be performed safely.

Group II: tumors are large with moderate arterial attachments and can be resected with careful surgical dissection.

Group III: tumors are large neoplasms encasing the carotid arteries and can be resected only with arterial sacrifice<sup>14</sup>.

### CASE REPORT:

The patient (H.N) is a 55 year old women, was presented to me, complaining of a painless left sided neck swelling for more than 5 years. Her past medical and familial history was non specific, except smoking and hypertension.

On examination there was a painless, solid 5x3 cm mass in the left anterior triangle of the neck (Figure1).

The mass was pulsatile and had a bruit over it. It was vertically fixed, because of its attachment to the bifurcation of the carotid artery, but movable horizontally (Fontaine sign).

There were no cervical lymphadenopathy or any sign or symptom of cranial nerves deficit. Also there was no any mass in the right side of the neck. Other systems were



**Figure 1. mass in the left anterior triangle of the neck**

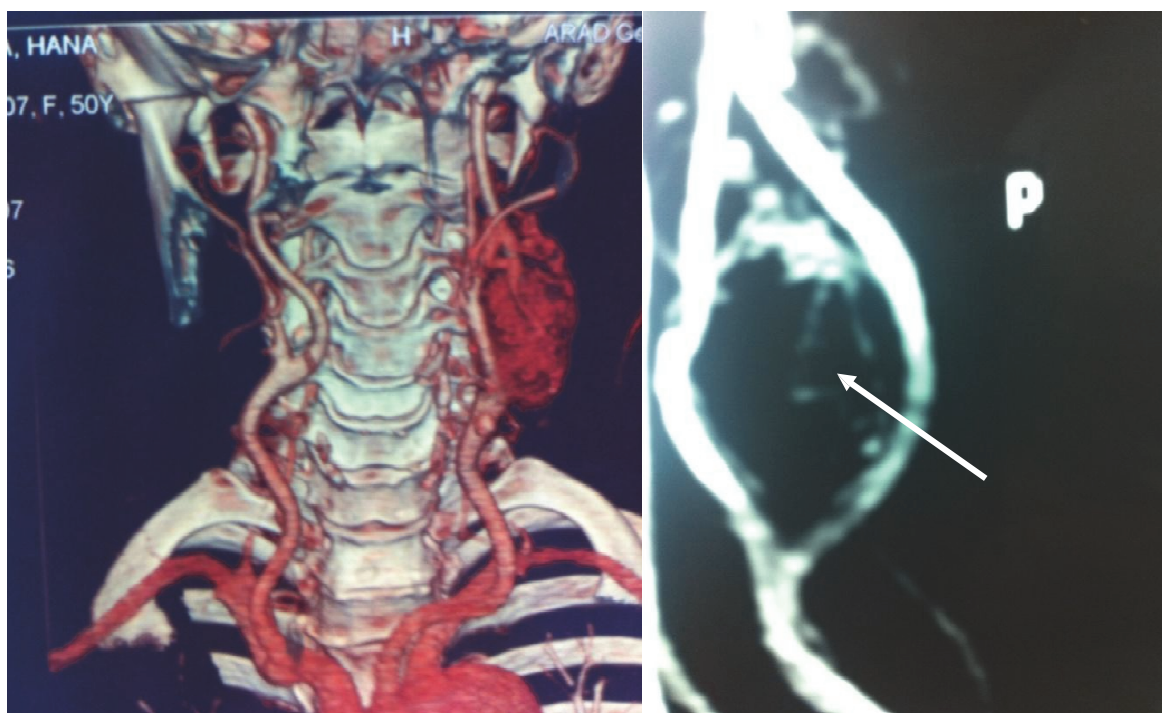
normal, except moderately elevated blood pressure (BP=140/95mmHg).

Laboratory investigations including; CBP, FBS, BUN, Serum Creatinine and urinary level of

Vanillylmandelic Acid (VMA), were done and all were in normal range.

CXR was normal and ECG shows no any significant changes. Doppler U/S of the neck vessels was done ; it shows an oval shape, hyper-vascular mass between the left internal and external carotid arteries. Multidimension CT-angiography of the neck as an excellent non invasive imaging study was done; a lobulated iso to mildly hyper-dense mass of about 5x3 cm,

located in the left carotid bifurcation was seen. the mass was markedly enhanced following contrast administration, represent of carotid body tumor. The tumor is highly vascular and splaying the internal carotid artery (ICA) and external carotid artery (ECA), from each other's (Lyric sign) .The carotid vessels in the right side of the neck were normal. (Figure 2-a , b)



**Figure 2. CT-angiography of carotid vessels revealed a highly vascular mass in the left carotid artery bifurcation (a) Fig.2 Lyric sign: splaying of the left ICA & ECA (b)**

The clinical diagnosis of left side carotid body tumor (CBT) was established and the patient was prepared for surgery, at 15 Th. Sep. 2013 in Vajeen privet Hospital in Duhok city.

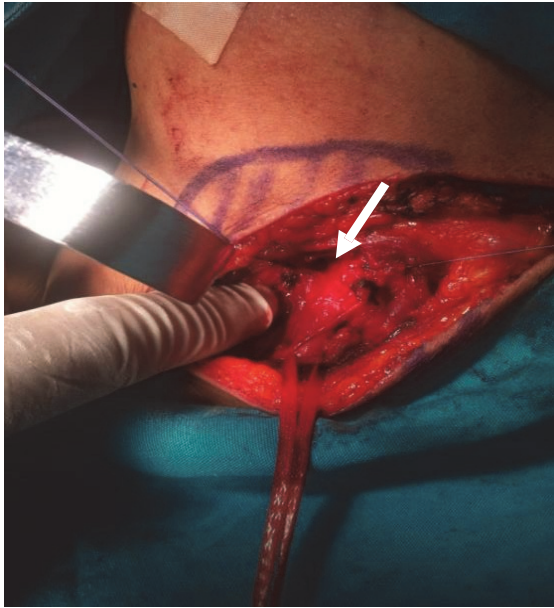
The preoperative preparation of the patient's includes ; explain of her condition for the patient's and her family's and discuss the surgical outcome and possibility of serious risks of operation and postoperative complications, cardiac and anesthesia consultations, control of high BP and reservation of 4 points of blood.

Under general anesthesia, the left side of the neck was explored by an oblique incision, parallel to the anterior border of Sternocleidomastoid (SCM) muscle. After elevation of skin flaps, the SCM muscle

was retracted, and the carotid sheath's was opened. There was a large sized (5x3cm), highly vascularized, lobulated mass, that located in the bifurcation of the carotid artery and separate (splay) both the ICA and ECA. The tumor was completely engulfed the carotid bifurcation and both it's branches, mainly the ICA (Shamblin \type III-CBT). These surgical findings was matched with preoperative clinical diagnosis of CBT. (Figure 3)

First of all, proximal and distal control of the carotid arteries; the common carotid artery (CCA), ICA, ECA, were done with a tape.





**Figure 3. Operative picture revealing CBT engulfing the carotid vessels.**

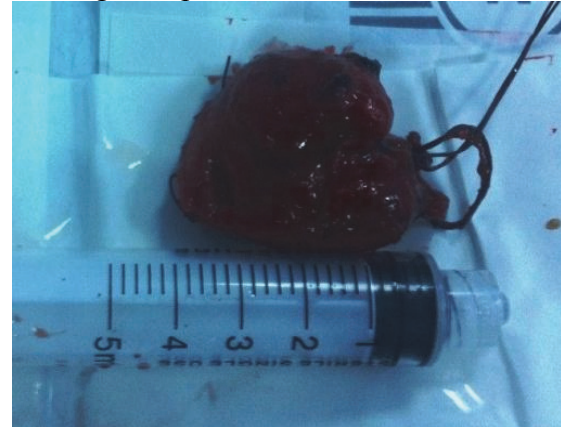
The internal jugular vein and Ansa cervicalis nerve were separated and displaced laterally and posteriorly. Then dissection with care was continued to separate the cranial nerves close to the tumor; First the Hypoglossal nerve's that coursing over the distal end of the tumor and then the Vagus nerve's, that lying posterior to it.

Because of the complete encasing of the carotid vessels ,by the tumor, periadvantitial dissection was impossible (Shamblin type III-CBT). (Figure 4) So decision for resection of the tumor with the involved part of the carotid vessels was done.

Before further dissection of the tumor, I decide to remove a segment of the Saphenous vein for maintain of continuity of the left internal carotid artery.

Through a longitudinal incision in the Left upper thigh a segment of 8 cm of the long Saphenous vein was removed and prepared for interposition graft. Then for continuous cerebral perfusion during period of the tumor resection; a synthetic catheter was used as an intraluminal shunt, first inserted in to the ICA distal to the tumor (to prevent air embolism to the cerebral blood circulation) and then to the CCA proximal to the tumor.

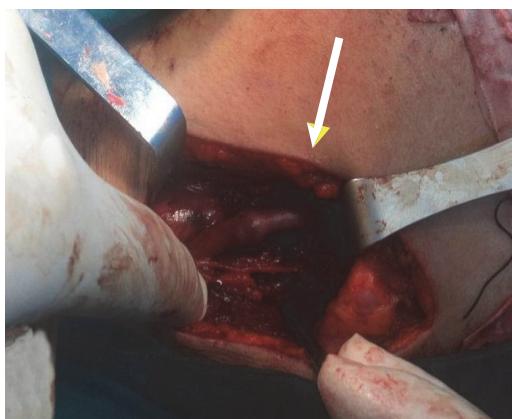
After fixation of the catheter inside the carotid arteries, the cerebral blood flow was shifted through the shunt and then the CCA and the ICA were closed with bulldog clamps.



**Figure 4. surgical specimen of shamblin III CBT**

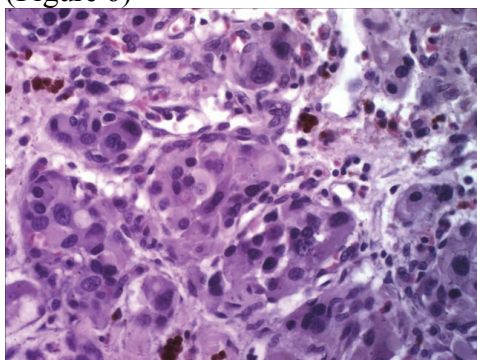
In order to prevent excessive bleeding, first the ECA and it's branches were clamped and suture ligated. Then the tumor with the involved part of the common carotid artery (CCA), its bifurcation and the ICA were completely resected During resection of the tumor, careful dissection was done to avoid injury to the adjacent cranial nerves. After complete resection of the tumor and control of bleedings, the shunt was removed and a prepared segment of Saphenous vein was reversed and end to end anastomosed (by using the 6/0 proline suture) first to the internal carotid artery (ICA) and then to the common carotid artery (the carotid artery occlusion time took 8 minutes). Then the carotid arteries were opened and the cerebral blood flow was restored, through the Saphenous vein interposition graft (Figure 5)

At the end of operation the patient has recovered from anesthesia, with no any sign or symptoms of brain damage or cranial nerves injuries. The post operative period was past without any complication and the patient was discharged from hospital after three days, on plavex 75mg. tablet once daily.



**Figure 5. Saphenous vein interposition graft**

The histopathology report of the resected mass was confirm the clinical diagnosis of CBT or carotid paraganglioma; "section shows proliferation of Nesting (Zellballen) or trabecular pattern of cells within a prominent vascular network. there is melanin-like pigment. Mitosis is rare" (Figure 6)



**Figure 6. Cytoplasmic Chromogranin Positive**

After two months of operation, the patient has recovered completely with no any complains. (Figure 7)



**Figure 7. Two months after surgery**

## DISCUSSION:

Carotid body tumors (CBTs) are rare , approximately 1out of 30000 tumors of head and neck region corresponds to a CBTs (paragangliomas), of which the most frequent site is the carotid body(45%)<sup>15,16</sup>.

About 1000 cases of CBTs had been reported in the literature<sup>17</sup>.

CBTs are generally present as a painless, rubber like and slowly enlarging neck mass (5mm annually)<sup>18</sup>.

They are located at the anterior border of sternocleidomastoid muscle at level of the hyoid bone. As the tumor grows ; dysphagia, odynophagia, dysphonia, and other symptoms due to compression of cranial nerves 9 to 12 may be observed<sup>18</sup>.

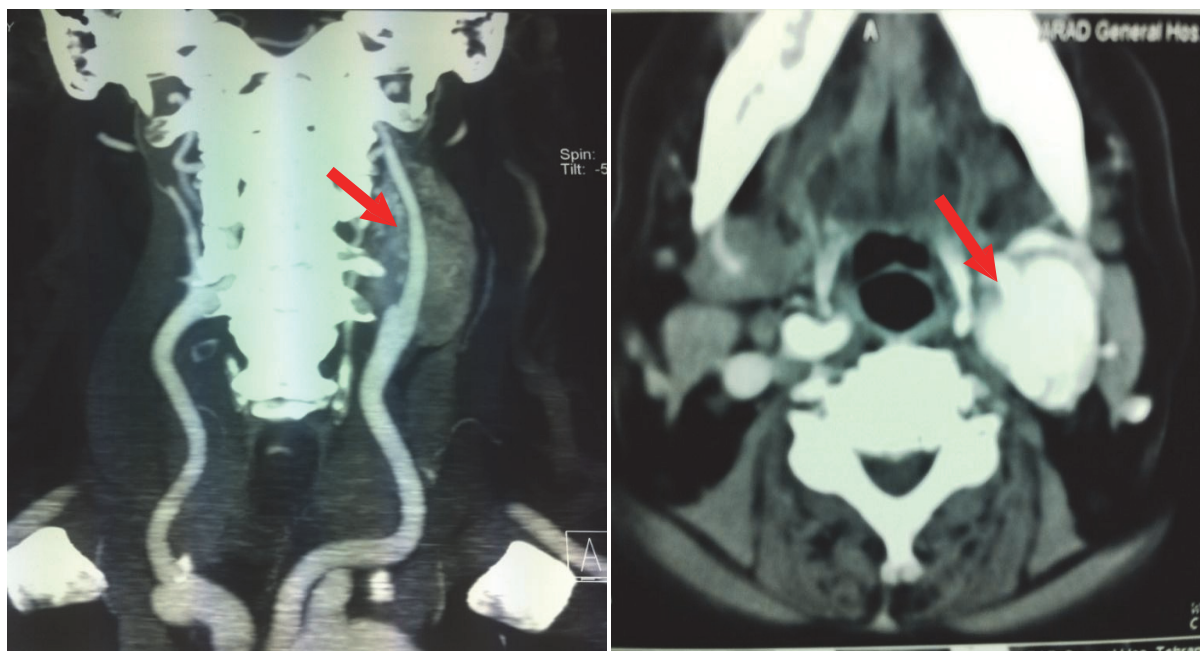
Bilateral rate of sporadic CBTs has been reported in 5-10% of cases<sup>15</sup>.

My patient presented with asymptomatic left sided neck mass, she didn't had any sign or symptoms of cranial nerves deficit.

For the diagnosis of CBTs, color Doppler U/S is the first non-invasive procedure, that allows for differentiation between solid and cystic nature of the tumor and carotid artery aneurysm<sup>19</sup>. Carotid artery angiography is the gold standard technique for diagnosis, which provides both diagnostic and therapeutic purposes<sup>20</sup>. it can detect multiple lesions, tumor size and vascularity and the major vessel tributaries supplying the tumor. During angiography embolization of the feeding arteries of CBT can also be performed.

Magnetic resonance Angiography (MRA) and CT angiography also can demonstrate the extent of the tumor, it's relationship to the adjacent structures and rule out multi-centric lesion<sup>10</sup>

Finally, Figure 8 (a ,b) shows the preparative computed tomography angiography of the left corotid body tunor.



**Figure (8-a,b) Preoperative computed tomography angiography of a left carotid body tumor**

In differential diagnosis of CBTs; bronchial cysts, salivary gland tumors, carotid artery aneurysm, lymphoma, neurofibromas, tuberculosis and metastatic carcinomas should be considered<sup>5,12</sup>. Biopsy and even fine middle aspiration are contraindicated in cases of suspected paraganglioma<sup>5</sup>.

I used the Doppler U/S, MRA and multidimensional CT-angiography for evaluation and diagnosis of my patient's.

Surgical resection is the treatment of choice, because it will remove the tumor and the malignant potential of CBTs.

Radiotherapy may be considered as an alternative treatment for unresectable tumor or an elderly patient in poor general condition<sup>5,21,22,23</sup>.

Shamblin type 1 and 2 tumors can often be dissected in a periadvantitial plane without traumatizing the carotid vessels. If the proper plane between the tumor and carotid vessels cannot be found or if the tumor completely surrounded the carotid vessels (Shamblin type 3), resection

of the involved part of vessels may be required.

The ICA should then be anastomosed to the CCA or replaced with a Saphenous vein or synthetic interposition graft. Ligation of the ICA is associated with a

high risk of stroke and a high mortality rate (50%), and should be avoided if possible<sup>24</sup>. On the other hand reconstruction of the ECA following tumor resection is not necessary, and it may be ligated<sup>25</sup>.

In my case, because the carotid vessels were completely surrounded by the tumor (Shamblin type III tumor), periadvantitial dissection of the tumor was impossible, so complete resection of the tumor with the involved part of the carotid vessels was done. I used a reversed segment of the

Saphenous vein as an interposition graft for maintaining of continuity of the carotid vessels. Operation for CBTs are not risk free<sup>26</sup>. As the tumor grows, the surgery becomes more

difficult and the postoperative complication rate will increase.

The high rate of cranial nerve deficit on the first postoperative day may arrive 49%, but the majority of these deficits are reversible, and the incidence of permanent cranial nerve palsy is reported to be low in the literature<sup>23,26</sup>.

Although my patient's had a very big tumor (tumor sized 5x3 cm and Shamblin type-III) she had not any sign or symptom of cranial nerve deficits in pre and postoperative period. During surgical resection, Surgeons should be prepared



themselves to do vascular reconstruction , if necessary.

Integrity of the ICA should be maintained by either a Saphenous vein or synthetic graft. during repair of the carotid artery, if the clamping period is less than 10 minutes, the risk of developing neurological damage is quite low<sup>27</sup>. many authors have emphasized that radical resection prevent local recurrences<sup>28</sup>.

During operation of my patient ,because of encasing of the carotid vessels by tumor, I decided to resect the tumor with involved part of the carotid vessel. Before resection, I maintained the cerebral perfusion by bypass shunt and the total period of carotid occlusion was 8 minutes. In postoperative period not any sign or symptom of neurological damage was noted.

Although CBTs are slowly growing tumors,early diagnosis as well as surgical treatment are important ,because of their malignant potential,progressive local growth and pressure on adjacent neurovascular elements.

Early surgery will decrease the neurological and vascular complications associated with CBTs.

#### REFERENCES:

1. Van Haller(1743). Cited in Kohn A. Die paraganglien .Arch Mikr. Anat. 1903;62:263-8.
2. Lack EE. Anatomy and physiology of peripheral arterial chemoreceptors in : pathology of adrenal and extra adrenal paraganglia .Philadelphia : W.B Saunders;1994:1-14
3. Maxwell JG ,Jones SW,Wilson E, Kotwall CA,Hall T,Hamann S,et al.Carotid body tumor excisions: adverse outcomes of adding carotid endarterectomy .J Am Coll Surg. 2004;198(1):36-41.
4. TA, Day JK. Joe. Primary neoplasms of the neck. In: Cummings: Otolaryngology: Head& Neck Surgery. 2005. P133. St Louis: Elsevier- Mosby; 2005: 113.
5. Wang SJ, Wang MB, Barauskas TM, Calcaterra TC. Surgical management of carotid body tumors. Otolaryngol Head Neck Surg. 2000;123:202–6.
6. Chedid A,Jao W. Hereditary tumors of the carotid bodies and chronic obstructive pulmonary disease.Cancer 1974;33(6):1635-41.
7. Bishop BG ,Urist MM, Gammal T, Petres GE ,Maddo WA.Paragangliomas of the neck. Arch Surg.1992;127(12):1441-5.
8. Ward PH, Jenkins HA,Hanaffe WN.Diagnosis and treatment of carotid body tumors. Ann Otol Rhinol Laryngol.1978;87(5 Pt 1):614-21.
9. Muhm M., Polterauer P., Gstotner W. et al. Diagnosis and therapeutic approaches to carotid body tumors. Arch Surg, 1997, 132: 279-84
10. Muhm M, Polterauer P, Gstöttner W, Temmel A, Richling B, Undt G.,et al. Diagnosis and therapeutic approaches to carotid body tumors. Arch Surg. 1997;132(3):279-84.
11. Dickinson PH, Griffin SM, Guy AJ, McNeill IF. Carotid body tumor: 30 years experience .Br J Surg. 1986;73(1):14-6
12. Barnes L,Tse LLY,Hunt JL. Carotid Body paragangliomas.In: pathology and genetics of the head and neck tumors.IARC;2005:364-65.
13. McPherson GA,Halliday AW,Mansfield AO. Carotid Body tumors and other cervical paragangliomas: diagnosis and management in 25 patients.Br J Surg .1989;76 (1):33-6.
14. Isik AC,Imamoglu M,Erem C,Sari A. Paragangliomas of the head and neck. Med Print Practice .2007;16 (3):209-14.
15. Shamblin WR, ReMine WH, Sheps SG, Harrison EG Jr. Carotid Body tumors(chemodectoma): clinicopathologic analysis of 90 cases. Am J Surg.1971;122(1) :732-9.
16. Parry DM, Li FP, Strong LC, Carney JA, Schottenfeld D, Reimer RR,



- Grufferman S. Carotid body tumors in humans: genetics and epidemiology. *J Nat Cancer Inst.* 1982; 68: 573-8.
17. Luna-Ortiz K, Rascon-Ortiz M, Villavicencio-Valencia V, Granados-Garcia M, Herrera-Gomez A. Carotid body tumors: review of 20 year experience. *Oral Oncol.* 2005; 41: 56-61.
  18. Pisanu A, Cois A, Uccheddu A, Cagetti M. Carotid body tumor: Case report and literature review. *Minerva Chir.* 2001; 56 (1): 101-9.
  19. Leonetti JP, Donzelli JJ, Littooy FN, Farrell BP. Perioperative strategies in the management of carotid body tumors. *Otolaryngol Head Neck Surg.* 1997; 117 (1): 111-5.
  20. Seabrook GR, Haimovici H, Ascer E, Hollier LH, Strandness DE, Towne JB. Nonatherosclerotic cerebrovascular disease. *Haimovici's vascular surgery, USA: Blackwell science, Inc;* 1996.
  21. Van der Mey AG, Frijns JH, Cornelisse CJ, Brons EN, Van Dulken H, Terpstra HL, et al. Dose intervention improve the natural course of glom us tumors? A series of 108 patients seen in a 32- year period. *Ann Otol Rhinol Laryngol.* 1992; 101 (8): 635-42.
  22. Evenson LJ, Mendenhall WM, Parsons JT, Cassisi NJ. Radiotherapy in the management of chemodectomas of the carotid body and glom us vagale. *Head Neck.* 1998; 20 (1): 609-13.
  23. Valdagni R, Amichetti M. Radiation therapy of carotid body tumors. *Am J Clin Oncol.* 1990; 13 (1): 45-8.
  24. Plukker JT, Brongers EP, Vermay A, Krikke A, Van den Dungen JJ. Outcome of surgical treatment for carotid body paraganglioma. *Br J Surg.* 2001; 88 (10): 1382-6.
  25. Dardik A, Eisele DW, Williams GM, Perler BA. A contemporary assessment of carotid body tumor surgery. *Vasc Endovascular Surg.* 2002; 36 (4): 277-83.
  26. Krupaki WC. Carotid body tumors. In: Rutherford RB, editor, *Vascular surgery*, 6th Ed. Philadelphia: W.B Saunders; 2006. p. 2066-73.
  27. Sajid MS, Hamilton G, Baker DM; joint vascular research Group. A multi center review of carotid body tumor management. *Eur J Vasc Endovasc Surg.* 2007; 34: 127-30.
  28. Patetsios P, Gable DR, Garrett WV, Lamont JP, Kuhen JA, Shutze WP, et al. management of carotid body paragangliomas and review of 30-year experience. *Ann Vasc Surg.* 2002; 16 (3): 331-8.

### پوخته

#### چاره سهرکړنا گړۍ يا جهستۍ شا دهمارا ستوی، ب ریکا نشته رگه ریی ویکار ئینانا خوین به را سافن

گړۍ يا جهستۍ شا دهمارا ستوی، وهرمه کا زور يا کیمه و چ نیشان یان ژان نینه. نه ډگرۍ په ل جهی دو شاخ بونا شا ده مارا ستوی، سهرهل ددهت. سهره رای هندۍ کو نه ډگرۍ په زور ل سهرخو مهن دبیت، بهی ژیه رکو چی دبیت ببیته وهرمه کا په نجه شیرۍ یان ژۍ کار تی کرنۍ ل دهمار یان ره هین خوینی یین ده ورویه رین خو بکته، پیټفی په ب زویترین دم بهیته ده ست نیشانکرن و نشته رگه ری بق بهیته نه نجام دان.

من ل فیره حاله ته کی، گړۍ په کا مهن يا جهستۍ شا دهمارا ستوی، راپورت کریه، کو نشته رگه ری بو هاتی نه نجام دان، دگهل پیداجونا بابته تی ل گوفارین پزشکی.

## الخلاصة

ورم الجسم السباتي، استئصال الجراحي، مع تطعيم توسط الوريد الصافن (إشهار حالة)

ورم الجسم السباتي، ورم غير مؤلم ونادرة، تنشأ من الخلايا العصبية في الجسم السباتي في التشعب الشريان السباتي في الرقبة. على الرغم من طبيعته النامي والتدريجي، الوصول الى تشخيص المبكر وتدخل الجراحي السريع مهم جداً، بسبب إمكانية تحولها الى ورم الخبيثة وتهاجم او الضغط على الانسجة المجاورة (أو عيته الدموية او الاعصاب). أنا هنا اقدم حالة الورم الجسم السباتي الكبير في الرقبة، إن عالجتها بتدخل الجراحي في ضوء مراجعة المجالات الطبية.

LIPOID PNEUMONIA AS A COMPLICATION OF MINERAL  
OIL ASPIRATION: A CASE REPORT

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SUMMARY

Lipoid pneumonia is an uncommon disease that may result from lipid aspiration. It was first described in patients with chronic laxative ingestion and use of oil base nasal drops. Presentation of lipoid pneumonia can vary from asymptomatic to a severe symptomatic disease. In this case report, we present a 10 years old boy with a background of cerebral palsy diagnosed to have lipoid pneumonia due to aspiration of liquid paraffin prescribed for chronic constipation. The diagnosis was made based on the basis of radiological and histopathological investigations. Following diagnosis, antibiotics therapy was stopped and a course of steroid therapy was given for six weeks. The mother was advised to stop giving her son liquid paraffin, and on follow up of the patient after six weeks, chest x ray showed minimal improvement.

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**Key words:** Pneumonia, Lipoid, Mineral oil, Aspiration

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**P**neumonia is a common disease that represents an inflammation of the lungs usually caused by an infection. It can affect all ages with variable mortality that range from mild to life-threatening condition. It is caused by many different organisms or agents including bacteria, viruses and fungi with different categories. It can be severe particularly in infants and young children, elderly people, and those with underlying debilitating diseases. Pneumonia be classified into different forms whether being acquired or not, the type of causative agent or the affected area of lung. Other risk factors include age, type of microorganisms, presence of associated lung disease or systemic disease, and history of hospitalization.<sup>1</sup> In infants and children aged less than 5 years pneumonia is the leading cause of mortality with an estimated incidence of 0.29 episodes per child-year in developing and 0.05 episodes per child-year in developed countries. This represent about

156 million new cases annually worldwide, of which 151 million cases are in the developing world.<sup>2</sup>

CASE REPORT

A 10-year-old boy who had cerebral damage as a result of Reye's syndrome since the age of 6 months presented with a one week history of cough and fever. Physical examination showed signs of his chronic neurological problem . Chest examination revealed reduced breath sounds and bronchial breathing on the right infra-clavicular area. Chest x-ray showed bilateral infiltrates (Figure 1). He was admitted and received Amoxiclavulanic-acid and later Cefotaxim for one week. He was discharged home but re-admitted again after few days with the same complaints and findings on chest x-ray. He received Ceftriaxone, Amikacin and Clarithromycin for 14 days. Influenza virus screen was negative, however while awaiting the screen result, he received

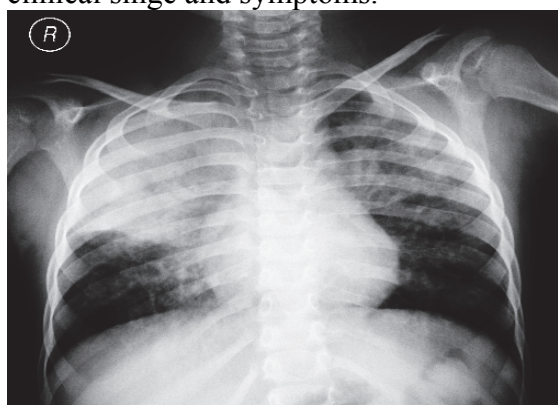
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Oseltamivir for five days. He improved clinically and was discharged home. At home, he remained afebrile for five days then started to have fever again and was brought back to the hospital for further evaluation. His repeated chest x-ray showed the same findings. He was started on Meropenem and then Gentamycin was added, however no appreciable response was noted. The antibiotics were changed to Linezolid and Ciprofloxacin. Despite negative culture he was given multiple courses of AB empirically because of the clinical singe and symptoms.



**Figure 1. Chest x- ray at presentation showing bilateral infiltrates.**

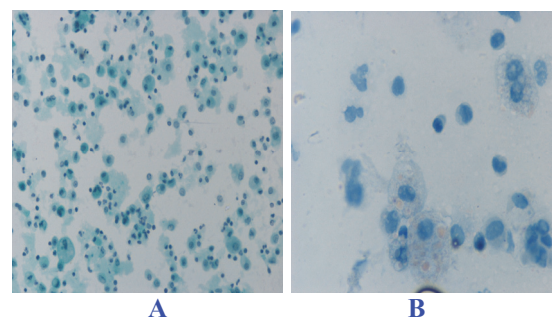
At the time of his last admission, computed tomography (CT) scan was done and showed large areas of consolidation in both upper lobes of the lungs. Sputum cultures on three occasions, sputum examination for acid-fast bacilli and mantoux test were all negative (Unfortunately sputum cytology sample was sent but for some technique error the sample was rejected. Full blood count showed hypochromic microcytic anaemia, thrombocytosis, with no leucocytosis or neutrophilia. He had high serum C-reactive protein concentration. The child had gastro-esophageal reflux and difficulty in swallowing as part of his neurological condition and he was on oral feed as his parents refused naso-gastric tube feeding.

The medical team noticed during the last admission the mother giving the child liquid paraffin which was prescribed to him earlier for constipation. Further history revealed that the child was found to spit out fumes while taking the liquid

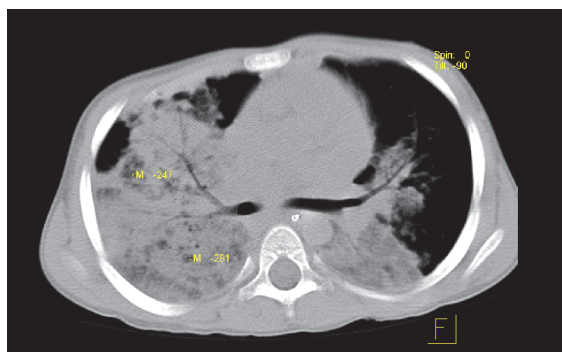
paraffin. In view of the persistent lung opacity, diagnostic bronchoscopy showed normal airway anatomy with no obstruction and on lavage, an oily turbid fluid was drawn back (Figure 2). Microscopical examination of the lavaged fluid showed abundant foamy macrophages and macrophages with lipid droplets within the cytoplasm (Figure 3). Repeat CT scan showed a large area of consolidation in both upper lobes of the lungs with innumerable small areas of low attenuation seen bilaterally in the area of consolidation with minus Hounsfield unit close to fat density (Figure 4). Hence, antibiotics therapy was stopped and a course of steroid therapy was given for six weeks. The mother was advised to stop the liquid paraffin, and on follow up of the patient after six weeks, he was found still having on-off low grade fever. His chest x ray showed minimal improvement.



**Figure 2. Fluid obtained by bronchoscopy showing its turbid appearance.**



**Figure 3. Microscopical examination of the lavaged fluid, showing abundant foamy macrophages using papaniouloua stain (A) and macrophages with lipid droplets within the cytoplasm using Oil re O (B)**



**Figure 4.** Chest CT scan showing large area of consolidation in both upper lobes of the lungs with innumerable small areas of low attenuation seen bilaterally in the area of consolidation with minus Hounsfield unit close to fat density.

## DISUSSION

Lipoid pneumonia is an uncommon under-diagnosed disease that may result from lipid aspiration.<sup>3</sup> It was first described by Laughlen in 1925 in four patients with chronic laxative ingestion and use of oil base nasal drops.<sup>4</sup> Clinically, it can vary from asymptomatic to a severe symptomatic disease. On the other hand, mineral oil is a common household product which is used usually for chronic constipation, a condition common among pediatric age group. Mineral oil is a mixture of liquid saturated hydrocarbons obtained from petroleum.<sup>5</sup> It is colorless and tasteless material that is used to aid bowel movement in patients with chronic constipation. It passes through the gastrointestinal tract without being taken into the body, but it limits the amount of water removed from the stool. Mineral oil can be easily obtained without prescriptions.<sup>3</sup> In our reported patient, there is a background of a neurological disorder in which swallowing dysfunction is part of the problem. Hence, he is at a higher risk for aspiration. Although the precise mechanism underlying oil induced damage is unclear, it is suggested that the aspirated oil is emulsified and phagocytosed by the alveolar macrophages which are then subsequently damaged. The lipid also causes the release of local inflammatory mediators causing chronic

inflammation which may eventually lead to fibrosis.<sup>6</sup>

The history of ingestion of substances containing oil is vital to establish the diagnosis of lipoid pneumonia. However, children with their parents may ignore or fail to mention this piece of information in the history.<sup>7</sup> Both bronchoscopy and bronchoalveolar lavage are attributing in achieving the diagnosis of lipoid pneumonia by demonstration the lipid laden macrophages.<sup>8</sup> Simple chest x-ray usually shows basal opacities, diffuse opacities or occasionally a solitary low-density mass known as paraffinoma. High resolution CT scan reveals consolidation with low attenuation, ground-glass opacity or a mosaic pattern of attenuation.<sup>3</sup> Although pneumothorax and pneumomediastinum are rare, however they have been reported in these patients.<sup>4</sup> CT scan can also reveal area of fat attenuation as low as -30 HU within the consolidation opacities and the nodules finding is diagnostic of lipoid pneumonia. At initial presentation, the attenuation of the opacities and the nodules can be low.<sup>4</sup>

Lipoid pneumonia is an indolent condition however it might also present as a progressive illness. The risk factors include concurrent debilitating illness and continued exposure to mineral oil. Therefore, early diagnosis and avoidance of exposure to oily substances is the most important step in avoiding the long term sequels.<sup>9</sup> The treatment of this disorder is not well-defined, and may be beyond avoiding ongoing exposure and providing supportive care. Systemic steroid therapy has been used to weaken the inflammatory response but most of the cases will resolve spontaneously with cessation of exposure. Although in some cases it is reported that steroid therapy seems to control the illness but not to solve the inflammatory response at least for severe intoxication.<sup>10</sup> Lung lavage with an emulsifying liquid has been used with good outcome in severe cases of exogenous lipoid pneumonia.<sup>11</sup> Finally, Erasmus et al<sup>12</sup> in their recent review on



swallowing problems in children with cerebral palsy have pointed to the increasing morbidity and mortality of these patients with the importance of care towards dysphagia and aspiration problems and their substantial contribution to the burden on the child and his/her family.

**Conclusion:** Lipoid pneumonia is an under-estimated diagnosis that may still be faced in the daily practice. It may result from chronic mineral oil use mainly when taken for chronic constipation. The diagnosis of exogenous lipoid pneumonia needs a relevant history of ingesting oil-based products in high risk individuals. Early diagnosis by bronchoalveolar lavage with CT scan is required to prevent long term sequels. The aim from presenting this report is to highlight the potential side effect of mineral oil and how it should be used with caution in the pediatric age group and in patients with neurological disorders causing swallowing dysfunction.

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## REFERENCES

1. Dharmage SC, Rajapaksa LC, Fernando DN. Risk factors of acute lower respiratory tract infections in children under five years of age. *Southeast Asian J Trop Med Public Health*. 1996; 27 (1): 107-10.
2. Rudan I, Boschi-Pinto C, Biloglav Z, Mulholland K, Campbell H. Epidemiology and etiology of childhood pneumonia. *Bull World Health Organ*. 2008; 86 (5): 408-16.
3. de Albuquerque Filho APL. Exogenous lipoid pneumonia: importance of clinical history to the diagnosis. *J Bras Pneumol*. 2006; 32 (6): 596-8.
4. Laughlen GF. Studies on pneumonia following nasopharyngeal injection of oil. *Am J Pathol*. 1925; 1: 407-14.
5. Betancourt SL, Martinez-Jimenez S, Rossi SE, Truong MT, Carrillo J, Erasmus JJ. Lipoid Pneumonia: Spectrum of Clinical and Radiologic Manifestations. *Am J. Roentgenol*. 2010; 194 (1): 103-9.
6. Midulla F, Strappini PM, Ascoli V, Villa MP, Indinnimeo L, Falasca C, et al. Bronchoalveolar lavage cell analysis in a child with chronic lipid pneumonia. *Eur Respir J*. 1998; 11 (1): 239-42.
7. Zanetti G, Marchiori E, Gasparetto TD, Escuissato DL, Soares Souza AJr. Lipoid pneumonia in children following aspiration of mineral oil used in the treatment of constipation: high-resolution CT findings in 17 patients. *Pediatr Radiol*. 2007; 37 (11): 1135-9.
8. Hadda V, Khilnani GC, Bhalla AS, Mathur S. Lipoid pneumonia presenting as non resolving community acquired pneumonia: a case report. *Cases J*. 2009; 2: 9332-5.
9. Bandla HP, Davis SH, Hopkins HE. Lipoid Pneumonia: A Silent Complication of Mineral Oil Aspiration. *Pediatrics*. 1999; 103 (2): 1-4.
10. Russo R, Chiumello D, Cassani G, Maiocchi G, Gattinoni L. Case of Exogenous lipoid pneumonia: Steroid therapy and lung lavage with an emulsifier. *Anesthesiology*. 2006; 104 (1): 197-8.
11. Simmons A, Rouf E, Whittle J. Not Your Typical Pneumonia: A Case of Exogenous Lipoid Pneumonia. *J Gen Intern Med*. 2007; 22 (11): 1613-6.
12. Erasmus CE, van Hulst K, Rotteveel JJ, Willemsen MA, Jongerius PH. Clinical practice: swallowing problems in cerebral palsy. *Eur J Pediatr*. 2012; 171 (3): 409-14.



### پوخته

#### سينك كولى چهورى نيك ژ ئيشين نه بهربه لاف و دروست بيت ژ نهگهري هلكيشياني مادي چهورى

بو جاري ئىكى ئەف ئىشە ھاتىە ديتىن ل نه خوشين ئووين مادا ملين بشيوكى بهردهوام بكارده ئينين ديسان ئووين چىكا دفىنى يى ل مادا چهورى ھاتىە دروست كرن. نه خوشى توشى سينك كولى چهورى يان بى نيشانه يان نيشانين گران ھەبيت. كىسەك ھاتىە توماركن زاروكەك كور ژىي وى ۱۰ سال و توشى ئيفليجى ميشكى بو وژ ئەگهرا بكار ئينانا مادا پارافين بو ماوهكى دريژ توشى سينگ كولى چهورى ب و، ودهستنيشان كرنا ئيشى ھاتە كرن ب ريكا تيشكى و ھيستوپاتولوجى. پىشتى دەستنيشان كرنا ئيشى دەرمانا دژى زينده ھاتىە راوہستاندن و نه خوش ھاتە چارەسەر كرن ب مادا ستىرويد بو شەش ھەفتىي وپىشتى بدىماھى ئينانا كورسا ستىرود ى، گھورانكى كىم ھاتىە ديتىن ل تيشكى سنگى، ودايكا زاروكى ھاتىە ئاگە ھار كرن بو قەدەغە كرنا بكار ئينانا مادا پارافين.

### الخلاصة

#### الالتهاب الرئوي الدهني نتيجة مضاعفات تطلع الزيوت المعدني: تقرير حالة

الالتهاب الرئوي الدهني هو مرض شائع قد ينجم عن تطلع الدهون. تم وصف الحالات الأولى للالتهاب الرئوي الدهني في المرضى الذين يعانون من تناول المليينات المزمنة واستخدام قطرات الأنف الزيتية. حالات واعراض الالتهاب الرئوي الدهني يمكن أن تختلف من حالة بدون أعراض الى أعراض مرضية شديدة. في هذا التقرير نقدم حالة للالتهاب الرئوي الدهني تم تشخيصها لدى صبي عمره ١٠ سنة مع خلفية إصابته بالشلل الدماغي حيث تم تشخيص إصابته بالالتهاب الرئوي الدهني بسبب تطلع البارافين السائل الذي كان يتناوله لعلاج الإمساك المزمن. تم إجراء التشخيص إستثناء على التحقيقات الإشعاعية والتشريحية المرضية. بعد التشخيص، تم إيقاف العلاج بالمضادات الحيوية، وأعطيت للمريض دورة من العلاج بالستيرويد لمدة ستة أسابيع. تم إبلاغ الأم بالتوقف عن إعطاء ابنها البارافين السائل، وعند متابعة المريض بعد ستة أسابيع، أظهرت الأشعة السينية للصدر حد أدنى من التحسن.