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EDITORIAL
SWINE FLU: HOW TO PROTECT OURSELVES?

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Key words: Influenza A(H1N1), Reassortment, One dose vaccine, Oseltamivir

The swine flu outbreak was clinically identified in April 2009.[1] The rapid global spread of a novel influenza A (H1N1) 2009 virus prompted the World Health Organization (WHO), on June 11, 2009, to declare the first influenza pandemic in 41 years and raised its alert level to phase 6 out of 6 possible.[2,3]

Pigs play an important role in interspecies transmission of influenza virus.[4] Susceptible pig cells possess receptors for both avian and human influenza strains which allow for the reassortment of influenza virus genes from different species if a pig cell is infected with more than one strain.This new strain appears to be a result of the reassortment of two swine influenza viruses, one from North America and one from Europe, the North American pig strain was itself the product of previous reassortments and has carried an avian PB2 gene for at least ten years and a human PB1 gene since 1993.[5-8]

In temperate regions influenza epidemics recur with marked seasonality: in the Northern hemisphere the influenza season spans from November to March, while in the Southern hemisphere epidemics last from May until September. Indoor crowding during cold weather, seasonal fluctuations in host immune responses, and environmental factors, including relative humidity, temperature, and UV radiation have all been suggested to account for this phenomenon, but none of these hypotheses have been tested directly.[9] Using the guinea pig model, a study found that low relative humidity of 20%–35% were most favorable, while transmission was completely blocked at a high relative humidity of 80%. Furthermore, at 5 °C, transmission occurred with greater frequency than at 20 °C, while at 30 °C, no transmission was detected. These data implicate low relative humidity produced by indoor heating and cold temperature as features of winter that favor influenza virus spread.[10]

This indicates that area with high humidity rates like Basrah, might have low transmission rates. In Kurdistan cold weather with low relative humidity during winter might favor such transmission. Nevertheless several other factors are implicated and one cannot usually draw such simple extrapolation, especially the relatively high rate of UV radiation is available in our region around the year.

Spread, presentation and complications of novel H1N1 virus are thought to be similar to that of seasonal flu spread although transmissibility appears substantially

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higher compared with seasonal flu.

Unlike seasonal influenza, however adults older than 64 years did not appear to be at increased risk of novel H1N1-related complications as a result of preexisting immunity against antigenically similar influenza virus that circulated prior to 1957. Information analyzed by Centers for Disease Control and Prevention (CDC) supports the conclusion that novel H1N1 flu has caused greater disease burden in people younger than 25 years of age than older people.[11]

A seasonal flu virus in the same H1N1 family as the pandemic virus has been circulating since 1977, but until now it was thought that this seasonal virus did not induce immunity to the pandemic strain. This was because the pandemic virus spread faster than would be expected if there were widespread immunity to it, and because antibodies to the seasonal vaccine do not cross-react with it. Much of the current global pandemic planning is predicated on previous experience that two doses of vaccine are required to elicit a protective immune response in populations that are immunologically naive to a new influenza strain making it very hard to immunize many people in time to protect them. Experimental pandemic vaccines "unexpectedly" developed protective antibodies after single dose.[12] "Somehow people's immunity has been primed". Cross protection that was afforded by exposure to antigenically drifted strains of the same influenza subtype has been described. The finding suggests that seasonal flu vaccine boosts those antibodies slightly, perhaps a reason to get a shot of seasonal flu vaccine this year.

These antibodies can't be the whole story, because people without them also responded swiftly to the vaccine. Seasonal H1N1 infection may have primed cell-mediated immunity, which may not prevent infection but limits the severity of the disease. This could be why the pandemic has spread fast but remained mild in many people, though not all.

Accordingly a one dose vaccination to novel H1N1 will ease conduct and hasten providing the general public with immunity. Relatively relieving news is the availability of vaccine. The food and drug administration gave its permission in mid-September-to novel H1N1 vaccine. The vaccine was prepared in embryonated chicken eggs with the same standard techniques that are used for the production of seasonal trivalent inactivated vaccine. The CDC indicates that the vaccine's effectiveness varies depending on the age and health of the person receiving it. Millions of doses are now available and priority is now given to health care personnel, school children and high risk groups.[12]

People infected with seasonal and novel H1N1 virus shed virus and may be able to infect others from 1 day before getting sick to 5 to 7 days thereafter. This can be longer in children and immunocompromised people.[13]

General actions that are implicated to help prevent the spread of infectious agents that cause respiratory infections are still indicated and thought to be effective in preventing novel H1N1 spread.[14]

Swine flu virus is fragile to

environmental conditions and can only retain infectivity for 2 to 8 hours after being deposit on surfaces. It is destroyed by heat [75-100°C]. This is much lower than other viruses such as SARS virus which can survive for 48 hours on plastic surfaces. In contrast SARS virus is inactivated by heating at 56◦C for 30 minutes.[15]

In addition, several antiseptics including chlorine, hydrogen peroxide, detergents (soap), iodophors (iodine-based antiseptics), and 70% alcohols are effective if used in proper concentration for a sufficient length of time. A study showed that 4 %chlorhexidine gluconate (CHG) product, reduced the virus by 99.94 percent after 30 second, exposures. A 0.5% CHG and 70% isopropyl alcohol formulation, reduced the virus by 99.99 percent after 15 second, both were more effective than soap and water which was used as a comparative test product.

To prevent the spread of influenza virus it is important to keep surfaces clean by wiping them down with a household disinfectant. Linens, eating utensils, and dishes belonging to those who are sick do not need to be cleaned separately, but importantly these items should not be shared without washing thoroughly by household laundry soap and tumbled dry on a hot setting.[16]

No research has been completed on the susceptibility of novel H1N1 flu virus to conventional drinking water treatment processes. Current drinking water treatment regulations provide a high degree of protection from viruses. Nevertheless tap water treated by conventional disinfection processes does not likely pose a risk for transmission of influenza viruses. However, recent studies have demonstrated that free chlorine levels typically used in drinking water treatment and recommended for swimming pools are adequate to inactivate highly pathogenic H5N1 avian influenza. It is likely that other influenza viruses such as novel H1N1 would also be similarly inactivated by chlorination. To date, there have been no documented human cases of influenza caused by exposure to influenza-contaminated drinking water.

Facemasks help stop droplets from being spread by the person wearing them. They also keep splashes or sprays from reaching the mouth and nose of person wearing them. They are not designed to protect against breathing in very small particle aerosols that may contain viruses. This mask can reduce but not completely abolish transmission of novel H1N1 virus. Respirators refers to an N95 has 95% efficiency in filtering out particles greater than 0.3 micron so it is designed to protect the person wearing it against breathing in very small particle aerosol that may contain viruses. It fits tightly on face and contains air filter.

The CDC recommends the use of oseltamivir or zanamivir for the treatment and/or prevention of infection with novel H1N1 flu virus. They may also prevent serious flu complications. They work best if started within two days of symptoms appearance. The priority use for influenza antiviral drugs is to treat severe influenza illness and high risk patients.[17,18]

School closure and the distribution of

antiviral medicines for prevention are not recommended because the virus is widespread within the community. People are likely to be repeatedly exposed to the virus in their every day lives. Closing school will no longer be effective in slowing the spread of the virus as people could still be exposed outside school.

In conclusion general personal protection measures against upper respiratory infection are effective and should be conducted. The mortality rate of swine flu virus is not markedly different from that of seasonal flu virus. Vaccination is now available and expected to be effective by single dose. Nevertheless health authorities should keep strict preventive measures to overcome any false sense of security of this pandemic which might change it's pathogenesis at any time.

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EFFECT OF PURIFIED 1-HYDROXYPHENAZINE PIGMENT ON ACTIVE AND TOTAL T ROSETTE FORMATION AGAINEST SECONDARY HYDATIDOSIS

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ABSTRACT

Background The effect of 1-hydroxyphenazine pigment which was isolated and purified from *Pseudomonas aeruginosa* on specific immune response T cells inside the body of white male BALB/C mice against experimental secondary hydatidosis and the infectivity of protoscoleces was studied.

Objectives The aim of this study was to found out the effect of this phenazine pigment (1-hydroxyphenazine) on one of the specific cell-mediated reaction against experimental hyadatidosis *In vivo* which may affect the infectivity of hydatid cyst protoscoleces.

Methods Six mice groups were used in this research, four of them were injected with(Primary and booster doses) of four concentrations of purified 1-hydroxyphenazine pigment (25, 50 , 75 and 100 µmole/ml) isolated from *Pseudomonas areuginos*a and then injected with 2000 protoscoleces /ml as a challenge dose after 7 days, active and total T rosette were calculated,and after 25 weeks the infectivity of prtoscoleces were studied in relative with the numbers and diameters of hydatid cysts which formed *in vitro*.

Results In comparison with negative control mice groups (P.B.S.) the results showed that the higher purified concentrations (50, 75 and 100 µmole/ml) of the pigment had suppressive effect on the specific immune response T cells and this effect was highly significant ($P<0.01$) after 6 weeks from challenge dose with protoscoleces intraperitoneally (I.P) against this pigment. This effect reflects that the protoscoleces infectivity was increased due to suppression of T rosette formation activity of T lymphocytes while the mitogen Phytohaemagglutinin (PHA) showed a significant stimulation of the specific cellular response which decrement protoscoleces infectivity in comparison with higher pigment concentrations.

Conclusion 1-hydroxyphenazine is a toxic pigment (dose dependent) causing decrementation of T cells activity especially at higher concentrations which allow protoscoleces development and growth.

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Key words: *Pseudomonas,* 1-hydroxyphenazine, T rosette, Protoscolex

Cystic echinococcosis is the most serious zoonotic disease caused by the metacestode of the dog tapeworm Echinococcus granulosus which affects human causing serious public health problem[1] and also infected a wide range of livestock species.[2]

Ultimate growth of the cyst depends on location inside the body of the host, so in some organs of the body they are unable to expand freely, whereas in others, most growth results in serious functional impairments of vital organs or even in death.[3]

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This parasite secretes some antigens that are thought to be responsible for immunomodulatory activities promoting its survival within a mammalian host[4] and they have extraordinary abilities to control host immune rejection mechanisms.[5]

Recent experimental studies were directed to improve this relationship between the parasite and the immune system arms (specific and non specific) by using chemicals or natural substances or parasite derived antigens that may modulates the immune response to control and prevent the infection.[6-8]

Pseudomonas aeruginosa

It is a prevalent opportunistic pathogen colonizing the respiratory tract in human causing many adverse effects, like chronic obstructive pulmonary disease[9] or direct tissue damage with a greater cytotoxic potential than others[10] or urinary tract infection.[11]

Many virulence factors produced by this pathogen affect the immune system during infection causing both acute and chronic diseases, these factors are either enzymes like proteinases[12] or may be endotoxins like Lipopolysaccharides (LPS) that suppresses host immune response causing persistent infection,[13] and sometimes poison pigments like a redox cycling phenazine pyocyanin[14] and its derivative 1-hydroxyphenazine.[15]

Many of these products have biological effects on host cells that may contribute to some inflammatory states like epithelial cell apoptosis,[16] or immunological effects on some of the specific immune response cells like T lymphocytes[17] and interleukins[18] while others may affects some of the innate immune response like macrophage[19] and complement.[20]

T Rosettings

T lymphocytes play a very important role against parasitic infections especially *Echinococcus granulosus* whether this role is pathogenic or protective in the immune response[21] and they accordingly elaborates a majority of soluble molecules that mediate interaction between cells called interleukins.[22] These cells have ability to bind with Sheep Red Blood Cells (SRBC) directly by its surface receptor (CD $+^2$) forming rosettings phenomenon which consist of central lymphocyte cell surrounded by bound red blood cells,[23] and this phenomenon is an index which correlated well with cell-mediated immune function.[24] This test is widely used in clinical immunity to determined the percentage of functional T lymphocytes in lymphocytes population of patients who is suffering from a variety of diseases and infections,[25-28] and diminishing of this phenomenon is also well known even in autoimmune disease.[29]

MATERIALS AND METHODS

Six groups of white male BABL/C mice (each group of 10-12 mice; aged 8-10 weeks; weight 21-24gm) were used for experimental infection which performed in animal house of biology department in college of science, Al-Musatansiriya

University, and hydatid cysts were collected from resident's patients in some of Baghdad hospitals from 1/2/1999 to 1/9/1999.

Protoscoleces were isolated from cysts in sterile conditions according to[30] method, and their numbers were adjusted to 2000 protoscoleces / 1ml of sterile Phosphate Buffered Saline (P.B.S) with pH = 7.2 and their viabilities were tested according to[31] method using eosin Stain (Viability must be more than 98%).

Design of experiments

The inbred males (Females excluded) BALB/C mice groups were injected as follow:

1. Four groups were inoculated intraperitoneally (I.P) with four purified concentrations of 1-hydroxyphenazine (25,50,75 and 100) µmole/1ml [32] and after seven days they were given the same concentrations of the pigment as a booster dose. Then after seven days from the booster dose, they were injected (I.P) with 2000 protoscoleces /1 ml of (P.B.S) as a challenge dose.
2. The fifth group was inoculated I.P with 1ml of sterile P.B.S and used as a negative control group.
3. The sixth group was inoculated I.P with 100 µgm /ml of non-specific mitogen Phytohaemagglutinin (PHA) and a challenge dose of the same number of protoscoleces and used as a positive control.
4. After 2, 4, and 6 weeks, T lymphocytes were separated according to [28] method and incubated with Sheep red blood cells (SRBC) according to [25] method.
5. Thin films of this mixture on very clean slides were prepared after 1 hour incubation for active T rosette and after 4 hours incubation for Total T rosette.
6. All the films were fixed with 70% alcohol and stained with Wright-Giemsa stain.
7. All slides were examined microscopically and 100 lymphocytes were counted.
8. T rosette forming cells (At least 3-5 SRBC bound to T Lymphocytes) were considered positive and counted.
9. After 25 weeks all mice were killed and dissected under dissecting microscope and the infectivity of protoscoleces was investigated and cysts number and diameters were recorded.

Statistical Analysis

The usual statistical analysis methods were done to analysis and asses all results obtained using SPSS vers.10 under windows XP as follow [33];

1- Mean. 2-Standerd Deviation (S.D). 3-Standerd Error (S.E). 4-Analysis of Variance (ANOVA). 5-Least Significant Difference (LSD) and results were expressed as follow: highly significant if $P<0.01$, significant if $P<0.05$, and non significant if $P>0.05$.

RESULTS

After two weeks of exposure to protoscoleces challenge dose, 1-

hydroxyphenazine caused decrementation in the active and total T rosetting formation and this decrementation was highly significant (P<0.01) specially among mice groups exposed to a concentrations of 75 µmole/ml of the pigment which were 41.25± 4.27 and 47.38 ± 2.56 for both active and total T rosetting respectively. In mice group exposed to 100 µmole/ml the results were 24.25± 3.99 and 25.00± 3.59 for both active and total T rosetting respectively. On other hand mice group exposed to low concentration 25 µmole showed no significant difference (P>0.05) for both active and total T rosetting formation in comparison with negative and positive control groups as shown in (Table 1).

A similar trend of activity was observed in mice exposed after 4 weeks of a challenge dose of protoscoleces to a concentration of 75 µmole/ml, decrementation was continued at a highly significant level (P<0.01) which were 18.38±2.26 and 25.75±3.77 for both active and total T rosettings respectively, and the results of the group exposed to 100 µmole/ml were 12.13±2.59 and 18.00±3.46 for both active and total T resetting. While for negative control group PBS were 82.25±2.05 and 84.00±3.02, and for positive control group PHA were 84.00±2.51 and 87.00±3.21 as shown in (Table 2).

Pigment concentration of 75 µmole/ml showed high significant decrementation (P<0.01) in active and total T rosetting in mice exposed after 6 weeks of challenge dose which were 19.25±4.80 and 19.88±1.81 respectively as compared with negative control group 70.25 ±1.39 , 74.75±8.83 and the positive control groups 84.25±1.49 ; 86.25±2.12 , while the mice groups exposed to (100) µmole/ml showed highly Significant decrement(P<0.01) in active and total T rosetting in comparison with both negative and positive control groups as shown in table 3.

The suppression of T lymphocytes rosette formation may indicate the ability of the protoscoleces to avoid the specific immune response as evidence by the significant (P <0.05) increment of cysts number and sizes recovered from mice injected with 75 and 100 µmoles which were 15.63±5.50 ,16.13±3.00 and 1.875±0.420, 2.213±1.792 respectively in comparison with the cysts recovered from the mice injected with PHA which decreased significantly the numbers and sizes of cysts but this decrement was less or not significant (P>0.05) between each two pigment concentrations as shown in table 4.

DISCUSSION

As we know, no previous studies have been conducted to study the effect of this pigment, which has been isolated and purified from *P. aeruginosa,* on T lymphocytes as immunomodulators against parasites and in particular against experimental hydatidosis.

The metacestode of *E. granulosus* requires the ability to alter or suppress the functional lymphocytes and avoid the host

Table1. Effect of purified 1- hydroxyphenazine on active and total T resetting *In vivo* after 2 weeks from Protoscoleces infection

Pigment concentrations (µmole/ml)	T Rosetting	
	Active Mean ± S.D	Total Mean ± S.D
P.B.S (- control) #	* 80.25 ± 5.12	*82.50 ± 6.07
P.H.A (+ control)	# 84.50 ± 5.53	$85.63 ± 1.85
25	*76.25 ± 3.88	*78.13 ± 6.31
50	$ 65.13 ± 2.23	Γ 65.88 ± 5.59
75	€ 41.25 ± 4.27	¥47.38 ± 2.56
100	γ 24.25 ± 3.99	# 25.00 ± 3.59

Different symbols are indicated significant differences between each two groups in the same column.

Identical symbols are indicated no significant differences between each two groups in the same column

Where significant difference (P<0.05), highly significant difference (P<0.01), non significant difference (P>0.05).

Table 2. Effect of purified 1- hydroxyphenazine on active and total T resetting *In vivo* after 4 weeks from Protoscoleces infection

Pigment concentrations (µmole/ml)	T Rosetting	
	Active Mean ± S.D	Total Mean ± S.D
P.B.S (- control)	*82.25 ± 2.06	* 84.00 ± 3.02
P.H.A (+ control)	*84.00 ± 2.51	* 87.00 ± 3.21
25	$71.13 ± 3.18	$ 79.13 ± 5.36
50	#50.50 ± 3.16	# 51.25 ± 4.o6
75	¥18.38 ± 2.26	Γ25.75 ± 3.77
100	£12.13 ± 2.59	€18.00 ± 3.46

Different symbols are indicated significant differences between each two groups in the same column.

Identical symbols are indicated no significant differences between each two groups in the same column.

Where significant difference (P<0.05), highly significant difference (P<0.01), non significant difference (P>0.05).

Table 3. Effect of purified 1- hydroxyphenazine on active and total T resetting *In vivo* after 6 weeks from Protoscoleces infection

Pigment concentrations (µmole/ml)	T Rosetting	
	Active Mean ± S.D	Total Mean ± S.D
P.B.S (- control)	*70.25 ± 1.39	*74.7 ± 8.53
P.H.A (+ control)	$ 84.25 ± 1.49	$86.25 ± 2.12
25	* 70.13 ± 4.45	*73.5 ± 3.16
50	#40.63 ± 5.53	#50.75 ± 6.23
75	£19.25 ± 4.80	Γ19.88 ± 1.81
100	¥ 9.25 ± 1.91	€9.63 ± 2.88

Different symbols are indicated significant differences between each two groups in the same column.

Identical symbols are indicated no significant differences between each two groups in the same column

Where significant difference (P<0.05), highly significant difference (P<0.01), non significant difference (P>0.05).

Table 4. Effect of purified 1- hydroxyphenazine pigment on cysts numbers and diameters after 25 weeks from Protoscoleces infection

Pigment concentrations (µmole/ml)	Cyst number	Cyst diameters (mm)
	Mean ± S.D	Mean ± S.D
P.B.S (- control)	0 ± 0	# 0 ± 0
P.H.A (+ control)	*1.75 ± 0.71	*0.738 ± 0.250
25	*3.88 ± 1.46	*1.088 ± 0.380
50	$9.25 ± 3.69	$1813 ± 0.577
75	#15.63 ± 5.50	$1.875 ± 0.420
100	# 16.13 ± 3.00	$2.213 ± 1.792

One way ANOVA of cysts numbers showed highly significant (P<0.01)

One way ANOVA of cysts diameters showed significant (P<0.05).

Different symbols are indicated significant differences between each two groups in the same column.

Identical symbols are indicated no significant differences between each two groups in the same column

Where significant difference (P<0.05), highly significant difference (P<0.01), non significant difference (P>0.05).

immunocompetent cells, thus facilitating the long term survival of the parasite.[34]

The principal finding of this study is that 1-HP pigment, isolated and purified from *P. aeruginosa,* is toxic product able to suppress the T lymphocytes against the experimental hydatidosis in mice infected with freshly isolated protoscoleces of *Echinococcus granulosus*, especially at higher concentrations (75 and 100 µmole/ml).

Pseudomonas aeruginosa phenazine pigment causes inhibition of human lymphocytes proliferation i*n vitro* in presence of killed *P. aeruginosa.* Purified phenazines are stronger inhibitors of T cells proliferation than crude pigment,[35] while[36] demonstrated that phenazine pigment caused local suppression of T lymphocytes proliferation and may interfere with cellular immune responses and this pigment inhibits the production of one of the essential lymphokines, Interleukin-2 (IL-2) and it's receptor on the T cell membrane.

Although the mechanism of phenazine pigment was not well known [37] and till now numerous questions regarding this mechanism remain unanswered,[38] but some authors reported that phenazine pigments induce intracellular reactive oxygen intermediate (ROI) which is toxic and in turn persist over a period of 5 hours, exposing the cells to sustained oxidative stress.[39]

1-Hydroxyphenazine like all phenazines pigments secreted by *Pseudomonas aeruginosa* may affects the activity of SRBC receptors on T lymphocytes ($CD+^2$, Ti) and causes suppression of the binding of SRBC to the T lymphocytes or T cell lines which led to diminishing of this phenomenon. On other hand, phenazine pigment has dual dose-dependent stimulatory as well as inhibitory effects in which high concentration resulted in an inhibition of Interleukin-2 (IL-2) production from T helper cells which are responsible for T lymphocytes activation,[40] and inoculation of mice with alive protoscoleces manifested high markers (IL-4, 5, 10) which they are responsible for disease progressions and establishment.[8] In addition to the toxicity of the pigment, the juvenile hydatid cyst fluids are able to suppress or destroy lymphocytes in direct contact with cyst[5] causing suppression *In vitro* blast transformation of T cells.[41] Moreover infection with higher dose of this parasite may result in non-specific suppression of T cell activity.[42]

In addition, our results agree with[32,43] who said that this pigment had a toxic effect on T lymphocytes activity and functions as well as against different species of bacterial growth *in vitro.*

From these results, it can be concluded that high concentrations of 1-hydroxyphenazine had suppressive effects on the active and total T rosettings phenomenon, while PHA is a good mitogenic, which is able to stimulate and proliferate T lymphocytes,[23] in addition this mitogen stimulates the expression of the receptors for sheep red blood cells. The ability of human T lymphocyte proliferation in response to

phytohaemagglutinin (PHA) is a traditional property.[44]

CONCLUSION

1-hydroxyphenazine excreted from *Pseudomonas aeruginosa* is a toxic pigment (dose dependent) induces suppression of T rosetting formation against experimental hydatidosis and this bacterium pave the way for hydatid cyst infection which may become more aggressive in patients colonized with this pathogen.

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پوختە

کارتێکرنا کەرەستێ 1-Hydroxyphenazine ل سەر تێکرایا پێکئینانا و گەشەپێدانا کیسێن ئاقێ

پێشەکی: کارتیکرنا کەرەستێ 1-Hydroxyphenazine ئەوێ هاتیە دەرئینان ژ بەکتریا Pseudomonas aeroginosa ل سەر شانێن بەرگریێ د لەشێ دا ژ جورێ T د مشکێن جورێ BALB/C ئەوێن هاتینە توشکرن ب نەخوشیا کیسێن ئاقێ هاتە پشکنین.

ئارمانج: ئارمانجا سەرەکی یا قێ پشکنینێ ئەوە کو کارتیکرنا کەرەستێ 1-Hydroxyphenazine ل سەر ئەرکێ شانێن جورێ T د بەرگریا نەخوشیا کیسێن ئاقێ د مشکاندا بهێتە رونکرن.

ڕێکێن ڤەکولینێ: شەش گروپێن مشکێن پشکینانا هاتینە بکارئینان د قێ ڤەکولینێ دا هاتنە بکارئینان. چار ژ ڤان گروپان هاتنە دەرزیکدان دوو جارکی ب کەرەستێ 1-Hydroxyphenazine ئەوێ هاتیە دەرئینان ژ بەکتریا Pseudomonas aeroginosa و ب چار ڕێژا ئەوژی (25, 50, 75, and 100 umol/ml) و پشتی حەفت روژا ب (2000/ml) ژ سەرکێن کیسێن ئاقێ هاتنە دەرزیکدان و پاشی گەشەپێدانا کیسێن ئاقێ هاتە پشکنین.

ئەنجام: بەراورکرن دگەل گروپێ کنترولی، هاتە دیتن کو ڕێژێن 1-Hydroxyphenazine (50, 75, 100) کارتێکرنەکا داشکاندنێ هەبو ل سەر رولێ شانێن بەرگریێ ژ جورێ T و ئەڤ کارتێکرنە یا مەعنەوی بو و ژ بەر وێ چەندێ سەرکێن کیسێن ئاقێ گەشەدا و دروستکرنا نەخوشیێ زێدە کر. بەروڤاژی کەرەستێ Phytohaemagglutinin ئەوێ شیای گەشەپێدانا سەرکێن کیسێن ئاقێ کێم کەت.

دەرئەنجام: کەرەستێ 1-Hydroxyphenazine کەرەستەکێ ژاراوایە و چالاکیێن شانێن بەرگریێ ژ جورێ T کێم دکەت.

الخلاصة

تأثيرصبغة 1-هيدروكسيفينازين ألنقيه في ألتشكل ألزهري التائي الفعال وألكلي ضد ألخمج التجريبي بالأكياس ألعدريه الثانويه

الخلفيه: تم دراسة تأثير صبغة 1-هيدروكسيفينازين ألنقيه ألمستخلصه من بكتريا ألزوائف الزنجاريه *Pseudomonas aeruginosa* في ألخلايا التائيه النوعيه المتخصصه ضد ألخمج التجريبي بالأكياس ألعدريه ألثانويه داخل الجسم ألحي ومعرفة مدى أمراضيتها.

ألهدف: يهدف البحث لمعرفة ألتأثير ألمناعي لصبغة1-هيدروكسيفينازين ألنقيه في ألتشكل ألزهري التائي الفعال وألكلي ضد ألخمج التجريبي بالأكياس ألعدريه ألثانويه وتأثير ذلك على خمجية ألروؤيسات ألأوليه داخل ألجسم ألحي.

طريقة ألبحث: أستعملت أربع مجموعات من ألفئران البيضاء الذكور للخمج التجريبي بـ ٢٠٠٠ روؤيس أولي/مل للأكياس العدريه كجرعة تحدي ضد اربعة تراكيز (٢٥ ، ٥٠ ، ٧٥ و١٠٠ مايكرومول/مل)على التوالي مع جرعه مقويه بعد سبعة أيام من ألجرعه الأولى لصبغة1هيدروكسيفينازين مقارنة مع مجموعةألسيطره سالبه ألمحلول ألملحي ألفسلجي ألدارئ ومجموعة ألسيطره ألموجبه(مشطر لانوعي) ثم تم حساب ألتشكل ألزهري ألتائي ألفعال وألكلي بعد مرور (٢، ٤ و٦ أسبوع) من جرعة ألتحدي وبعد مرور ٢٥ اسبوع تم حساب خمجية ألروؤيسات ألأوليه بدلالة معدل عدد وقطر ألأكياس ألعدريه المتكونه داخل جسم ألفئران ألبيضاء ألمخمجه.

ألنتائج: مقارنة مع مجموعة السيطره ألسالبه والموجبه اظهرت النتائج بان التراكيز ألمستعمله قيد ألبحث وخصوصا العاليه للصبغه (٥٠،٧٥ و١٠٠) مايكرومول/مل لها تأثيرآمثبطآ وبمعنويه عاليه(P<0٠01) في فعالية الخلايا أللمفاويه التائيه وتبين بأن هذا التأثير كان معنوياً بدرجة عاليه(P<0٠01)وذلك بعد ستة اسابيع من حقن ألروؤيسات الأوليه في ألفئران المعامله.أظهر هذا ألتأثير زياده في أمراضية وخمجية ألروؤيسات ألأوليه بسبب تثبيط فعالية الخلايا التائيه ألمناعيه، فيما أظهر ألمشطر أللانوعي(Phytohaemagglutinin) تحفيزآ في ألأستجابه ألمناعيه ألخلويه ألمتخصصه وأالتي أدت الى نقصان في أمراضية ألروؤيسات ألبدائيه مقارنة مع تراكيز ألصباغ كافه.

ألأستنتاج: صبغة 1-هيدروكسيفينازين هي صبغة سامه ويزداد تأثيرها بأزدياد تركيز ألجرعه وتسبب تقليل فاعلية ألخلايا ألمناعيه ألتائيه وخاصة عند ألتراكيز ألعاليه وألتي تسمح بنمو وتطور ألكيس ألعدري.

CARDIAC COMPLICATIONS OF HOMOZYGOUS-β THALASSAEMIA

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ABSTRACT

Background Homozygous – β thalassaemia has frequently been reported to be associated with cardiac complications mainly due to the deposition of iron in the heart.
Objective To find effects of homozygous β-thalassemia on cardiac functions and its relation with Haemosiderosis.
Patients and Methods Eighty patients with homozygous β thalasseamia were included in the study. Thalassaemic center attach to Ibn-Alatheer paediatric hospital, Haematology unit, Coronary care unit and Echo unit in Ibn-Sina teaching hospital in Mosul (February to September 2005). All patients were subjected to full clinical, laboratory, ECG and Echocardiographic evaluat
Results There were 50 males and 30 females. Age ranged between 11-35 years, their mean was 15.8 years. 18 (22.5%) patients had cardiac dysfunction, of them 12 (15%) had systolic dysfunction and 6 (7.5%) had diastolic dysfunction. ECG changes were present in 27(33.75%) patients and 31(38.75%) had valvular abnormalities. Congestive heart failure present in 3(3.75%) patients and 3(3.75%) patients had pulmonary hypertension.
Conclusion Cardiac Complications are common in Homozygous-β Thalassaemia older than the age of 10 years. Further studies utilizing more advanced techniques to detect earlier cardiac dysfunction more accurately may be useful.

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Key words: Thalassaemia, Myocardial complications, Haemosiderosis

β-thalassaemia is inherited haemoglobin disorder (autosomal recessive) caused by impaired synthesis of β-globin chain and resulting in chronic haemolytic anaemia.[1-3]

According to clinical severity, three forms of thalassaemia are distinguished, Thalassaemia major (TM), intermedia and minor.

The diagnosis of β-thalassaemia major is readily made during childhood on basis of severe anaemia, accompanied by the characteristic sign of massive ineffective erythropoiesis, hepatosplenomegaly, profound microcytosis and characteristic blood smear and elevated levels of HbF, with or without HbA2 elevation.[4-6]

In thalassaemic patients, long term

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transfusion therapy, extravascular haemolysis and increased intestinal absorption of iron result in systemic iron overload.[7-10] In patients who are not receiving transfusion, abnormally regulated iron absorption result in increase body iron burden ranging from 2-5 gram/year, depending on severity of erythroid expansion.[11,12] After approximately one year of transfusion iron began to be deposited in parathyroid tissue.[13]

Tissue iron overload is fatal with or without transfusion if not prevented by iron chelation therapy which considered a major focus of management.[14]

Within the heart, changes associated with chronic anaemia which is aggravated by iron deposition. Excessive iron deposition associated with cardiac hypertrophy and dilatation, degeneration of myocardial fibers and in rare cases fibrosis. In patients who are receiving transfusion but not chelating therapy symptomatic cardiac disease has been reported within 10 years after the start of transfusion.[15-19]

Haemochromatosis of the heart is considered as a variety of dilated cardiomyopathy with increased left ventricular diastolic cavity dimension and depressed systolic function.[20-21]

PATIENTS AND METHODS

The study comprised 80 patients (older than 10 years) with the diagnosis of homozygous β-thalassaemia, attending the thalassaemia center attached to Ibn-Alatheer paediatric hospital and the haematological unit at Ibn-Sina teaching hospital in Mosul for follow up or blood transfusion during the period between 1st February and 30th September 2005.

All patients were fully assessed clinically, laboratory investigation included Hemoglobin and iron studies, in addition to echocardiography (2 dimensional and M modes) and electrocardiography were performed to all patients.

Patients were classified into three age groups <15, 15-20, >20 years.

RESULTS

There were 50 male and 30 female with ratio of (1.7:1; their ages ranged between 11-35 years (mean 15.8). The duration of illness ranged between 11-31 years with a mean of 15.5 years). Frequency of blood transfusion (unit) ranged between 116-340 units with a mean of 232 units (Table 1).

Eighteen patients had cardiac dysfunction, 12 of them had systolic dysfunction and 6 patients had diastolic dysfunction (p value < 0.05). The mean Left ventricular end diastolic dimension (LVESD) was 3.45 cm, left ventricular end diastolic dimension (LVEDD) was 4.65 cm and both were more in patients in the cardiac dysfunction group by echo i.

The mean ejection fraction (EF%) was 48.8%, fractional shorteneing (FS) was 22.4%. For patients with abnormal cardiac function, it was much lower in comparison with non cardiac dysfunction group.

Figure 1 indicates that cardiac dysfunction, systolic and diastolic, was higher among patient aged more than 20 years.

Valvular abnormalities in the form of

mitral, aortic and pulmonary regurgitation were found in 31 patients with higher incidence in older age group, P value < 0.01. Mitral regurgitation was found in 26 patients, 4 patients had pulmonary regurgitation and only 1 patient had aortic regurgitation. Figure 2 shows that patients aged more than 20 years had higher percentage of valvular abnormalities.

Twelve of those with cardiac dysfunction had haemoglobins ≤ 60 g/l while the remaining 6 patients were haemoglobin was 60-90 g/l, with P-value < 0.01). Regarding splenectomy, 12 patients with cardiac dysfunction are splenectomised and 6 patients were non splenectomised. Thirteen of those with cardiac dysfunction were on regular desferroxamine therapy while five were on irregular therapy. On the other hand, of the 12 males with cardiac dysfunction, 10 had transferrin saturation of 60-100% while the other 2 had transferrin saturation < 60%. Of the six females with cardiac dysfunction, 5 had transferrin saturation of 50-100% while the remaining one had a transferrin saturation < 50% (Table 2).

ECG changes: 6 patients had Left Ventricular Hypertrophy (LVH), 3 had Right Ventricular Hypertrophy (RVH), 4 had Left bundle branch block (LBBB), 3 had right bundle Branch block (RBBB), 14 patients had ST and T wave changes, P value < 0.05 (Table 3).

DISCUSSION

Homozygous transfusion dependant ß thalassaemia patients manifest cardiac, hepatic, endocrine and metabolic disorders attributable to chronic hypoxia and iron overload.

Quality and duration of life of thalassaemic patients can be expected now to extend beyond the 3rd decade with regular blood transfusion and iron chelating therapy. Although premature from cardiac complications is still a major problem in the management of these patient as a consequence of chronic anaemia and iron overload.

Table 1. Mean of age; duration; frequency of blood transfusion and LV

Parameters	Maximum	Minimum	Mean
Age (years)	**35**	**11**	**15.8**
Duration (years)	**31**	**11**	**15.5**
Frequency of blood transfusion (units)	**340**	**116**	**232**
LV parameters by echo			
LVESD	**5.2**	**2.4**	**3.485**
LVESD	**6.8**	**3.2**	**4.656**
EF%	**89**	**35**	**60.562**
FS	**41.3**	**13.7**	**32.893**

LVESD: left ventncular end systolic dimension
LVEDD: left ventncular end diastolic dimension
EF%: Ejection fraction
FS: Fractional shortening

Iron overload can result from increased intestinal absorption of iron which result also in increased marrow expansion, extramedullary haematopoiesis, splenomegaly, susceptibility to infection and hypercoagulability.[22-24]

In developing countries most of thalassaemic patients are under transfused and have poor quality of life and developed hypersplenism and most die from combination of anaemia, infection and complications of hypersplenism before age of 12 years.[25]

The present study show that clinically evident cardiac dysfunction was present in 48 patients in the form of dyspnoea, chest pain, palpitation, exercise intolerance, elevated jagular venous pressure, gallop rhythm, hepatomegaly, peripheral oedema), these symptoms and signs were higher in older age group.[26]

Anaemia is another contributing factor for cardiac dysfunction in thalassaemic patients, abnormal cardiac function was higher in patients with anaemia of moderate severity, although statistically not significant it indicates a positive correlation between severity of anaemia and cardiac dysfunction.[27]

The present study shows a significant incidence of abnormal cardiac function in patients who receive > 100 units of blood, P value < 0.05.

The present study shows that patients with elevated transferrin saturation percent and poor compliance to chelating therapy are at high risk of severe heart haemochromatosis, abnormal cardiac function increased by 10% when transferring saturation > 60% in male patients and by 70% when transferring saturation > 50% in female patients.[28]

Abnormal cardiac function is more common in splenectomised than non splenectomised patients because the mean value of transferring saturation in splenectomised cases was significantly higher compared to that of non splenectomised group.[29-32] ECG is standard method for investigating thalassemic patients,[33,34] in our study ECG changes were present in 27 patients (33%) which is statistically significant (P value < 0.05), of them 6 (7.5%) patients had LVH, 3 (3.75%) patients had RVH, Conduction defect due to deposition of iron conduction tissue and myocardium,[35] in form of LBBB, RBBB and arrhythmias are present in 9 (8.75%) patients. Infiltrative cardiomyopathy as ST, T wave changes was present in 14 (17.5%) and present mainly in chest lead. Some patients had chambers hypertrophy and at the same time had conductive abnormality and ST, T wave changes.

Echocardiography appear to be more reliable tool than clinical, ECG and/or chest x-ray in the assessment of cardiac function of thalassaemic patients. The first echo abnormality in patient with systolic function seems to be an increased LVEDD while left ventricle mass not altered.

In the present study the parameter LVEDD shows dilatitive cardiomyopathy andarrhythemias which are late complications of Cooley's anaemia which contribute to severe heart failure which is resistant to conventional treatment.[36,37]

Systolic cardiac dysfunction present in 12(15%) of patients where diastolic

dysfunction present in 6(7.5%) of patients with high incidence in older age group and male gender, and congestive heart failure present in 3(3.75%) of patients.

Valvular abnormalities are also hard to characterized and to grade with precision by mean of echo, leaflet thickening and endocardial calcification, followed by valvular regurgitation probably caused by volume overload and the resultant cardiac dilatation.[38-40]

In the present study valve lesion were present in 31(38.75%), of them 26(32.5%) had mitral valve regurgitation, the other 4(5%) patients had pulmonary valve regurgitation and only 1(1.25%) patient had aortic regurgitation which is statistically significant P value < 0.01.

Pulmonary hypertension occur in older age thalassaemic patients and result in right heart failure, it is evaluated by the peak systolic right ventricular to right atrial (tricuspid) pressure gradient derived by continuous Doppler tracing of tricuspid valve flow, it is defined by tricuspid gradient of > 30 mmHg,[41-43] this is not done in our study, we depend on clinical diagnosis of pulmonary hypertension. Three (3.75%) of our patients had pulmonary hypertension.

CONCLUSION

Thalassaemia is one important cause of cardiomyopathy with serious effect on the hole structure of the heart including the valves and conductive tissue.

Table 2. Cardiac dysfunction according to clinical and echo

Parameter	Cardiac dysfunction (No. of patients)	
	Positive	Negative
Hb		
<6	**12**	**34**
6-9	**6**	**28**
Splenectomy		
Splenectomized	**12**	**42**
Non splenectomized	**6**	**20**
Transferin saturation		
Male		
<60	**2**	**10**
60-100	**10**	**28**
Female		
< 50	**1**	**6**
50-100	**5**	**18**
Cardiac function by echo (systolic and diastolic dysfunction)	**18**	**62**
Desferrioxamin therapy		
Regular	**13**	**47**
Irregular	**5**	**15**

Table 3. Abnormal ECG parameters in the studied group

ECG changes	Number of patients
LVH	**6**
RVH	**3**
LBBB	**4**
RBBB	**3**
ST and T wave changes	**14**
Arrhythmias	**3**

LVH: left ventricular hypertrophy
RVH: Right ventricular hypertrophy
LBBB:left bundle branch block
RBBB: Right bundle branch block

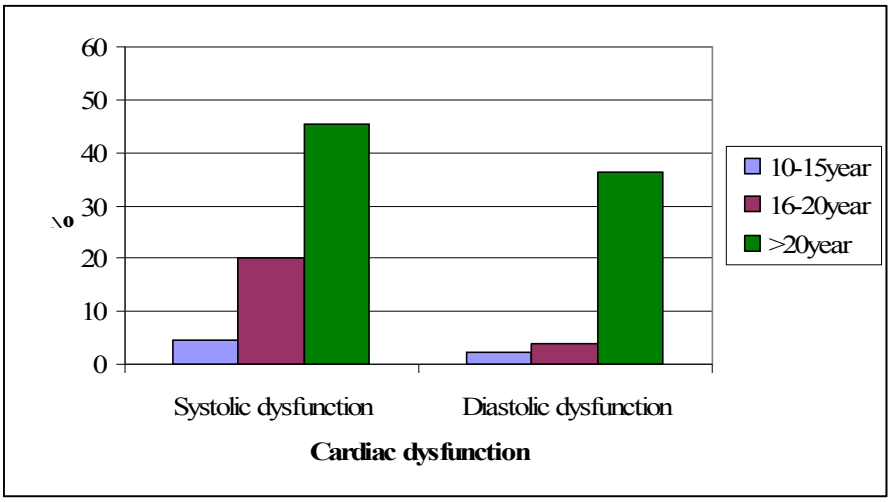

Figure 1. Systolic and diastolic dysfunction according to the age group

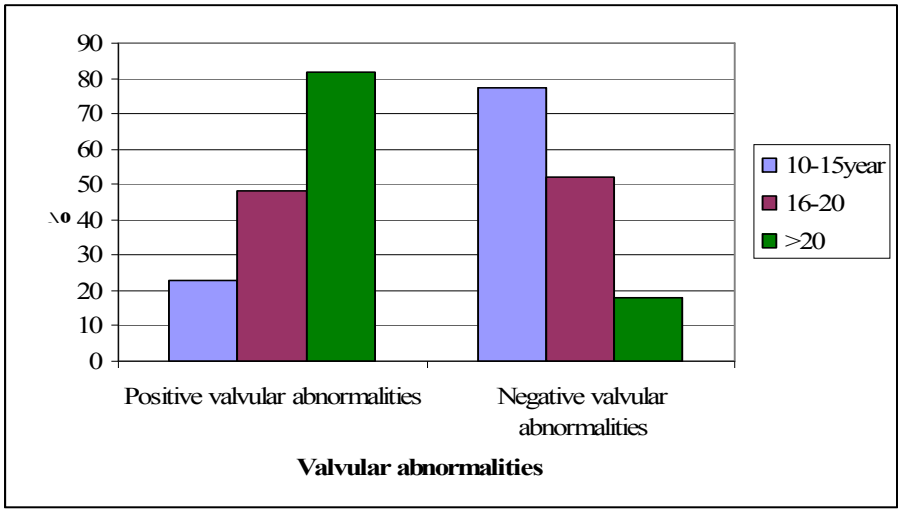

Figure 2. Valvular abnormalities according to age group

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پوختە

نەخوشخانا دلی ژ ئەنجامێ نەخوشیا تەلاسیما ژ جورێ ب

پێشەکی: گەلەك جارا هاتیە دیارکرن کو نەخوشیا تەلاسیما ژ جوری ب پەیوەندیەك یا هەي دگەل تێکدانا کارێ دلی ژ بەر زوربونا ریژا ئاسنی ل دلی دا .

ئارمانج: واستیانا دلێ ژ ئەنجامێ نەخوشیا تەلاسیمێ ژ جوری ب و پەیوندیا وی دگەل زوربونا ریژا ئاسنی خوینیدا .

نەخوش و ریکا ڤەکولینێ: پشتی ل بنکەهی نەخوشخانا(ئەبن ئەسیر)یا تەلاسیمیا زاروکان,یەکێت نەخوشێن خوینی وجارسەرین دلی ویەکەي کارێت دلی ل نەخوشخانا(ئەبن سینا)یا فیرکرنی ل میسل هە رژ ئیکی هەیڤا شباتێ تاسیهێ هەیڤا ئە یلوڵێ2005 .

ئەنجام: هاتە شلوڤەکرن هەشتێ نەخوش ئەوێن توشی نەخوشیا تەلاسیمیایی بووین ئەڤا لیکدایی ژجوری ب,ژی وان ل ناڤبرا(11_35) سالی بوون, ل تاقیگەهی شلوڤەکرنی بو گشت نەخوشیا هیموکلوبین و ریژا ئاسنی دناڤ خوینیدا,هەروەسا پشتی شلوڤەکرنا پیلێن دلی سەر دەنگیدا وکیشانا دلی یا کارەبای ژزێدەڤە بو شلوڤەکرنێن دەماغ و ماسولکێن ودەماربوهەمی ئەوان نەخوشیا . هەژدە نەخوشین (22.5%) ێن توشبوین ونەخوشین دلی ژوان (15%)ئەوبوون نەخوشین وان گران هەین(7.5%) ئەوین نەخوشین سڤك هەین(3.75%) ئەوین دەمارین دلێ وان کرتي(33.75%)ئەوین بێ گوهرین کیشانا دلێ ژجوری کارەبێ و(38.75%) یاتوشبونی کاری خوناکەت بورین دلێ (3.75%)ئەوین توشبوین ژ بلندبونا گیڤاژێن خوینی لدەمارین میلاکا سوور .

دەرئەنجام: تالاسیمیا جورێ ب کارتێکرنەکا زور یا لسەر پێکهاتێن دلی هەین و ئەگەرەکێ گرنکە تێکدانا کارێ دلی .

الخلاصة

مخالطات مرض القلب بسبب ثلاسيميا المتجانسة نوع ب

خلفيةالبحث: الثلاسيميا المتجانســـــــــة من نوع ب لديها علاقة بالمضاعفات القلبية نتيجة زيادة نسبة الحديد في انسجة القلب .

الهدف: تقييم مضاعفات القلب الناتجة عن الثلاسيميا المتجانســـــــــة نوع ب و علاقتها مع زيادة نسبة الحديد في الأنسجة.

المرضى و طرق البحث: أجريت الدراسة في مركز الثلاسيميا بمستشفى ابن الأثير للأطفال, وحدات أمراض الدم و العناية القلبية و وحدة وظائف القلب في مستشفى ابن سينا التعليمي في الموصل خلال الفترة من الأول من شباط و لغاية الثلاثين من شهر أيلول 2005.

النتائج: تضمنت الدراسة ثمانين مريضا مصابين بمرض الثلاسيميا المتجانسة نوع - ب , تتراوح أعمارهم مابين 11-35 سنة. أجريت فحوصات مختبرية لجميع المرضى شملت فحص الهيموغلوبين وحالة الحديد في الدم, كما تم فحص القلب بالأمواج فوق الصوتية وتخطيط القلب الكهربائي بالأضافة الى الفحص السريري لكافة المرضى. ثمانية عشر مريضا (22،5%) مصابون باعتلال في عمل القلب منهم 15% اعتلال انقباضي و 7،5% اعتلال انبساطي, 3،75% لديهم عجز القلب الأحتقاني و33،75% لديهم تغيرات في تخطيط القلب الكهربائي و 38،75% يعانون من عدم كفاءة الصمامات القلبية و 3،75% مصابون بارتفاع ضغط الدم للشريان الرئوى

الاستنتاجات: ثالاسيميا نوع ب ذات تاثير جسيم على مكونات القلب جميعا وسبب هام فى تعطيل الوظائف التى تقوم بها .

DERMATOGLYPHIC PATTERNS IN IRAQI PATIENTS WITH INSULIN DEPENDENT DIABETES MELLITUS

SAMEH S. AKKILA, MBChB, MSc*

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ABSTRACT

Background The scientific study of papillary ridges on the hands and feet (dermatoglyphics) is a field of increasing interest in medicine. Because fingerprints and line formations form during vital stages of fetal development and are age-stable, dermatoglyphic studies are in a unique position to evaluate the effect of maternal, genetic and environmental factors on early fetal development.

Objectives Insulin dependent diabetes mellitus (IDDM) is in part genetically determined and the study aimed to reveal any association of certain dermatoglyphic patterns with IDDM in Iraqi patients.

Patients and Methods Patients were scanned for 3 dermatoglyphic characteristics (Fingerprint pattern, digital ridge counts and A-B ridge counts) and compared to unrelated controls. Two commonly associated conditions with IDDM were also included in the study: hypertension and hyperlipidemia.

Results The study revealed statistical difference in the form of increased whorls and arches but decreased loops in diabetics. The digital ridge counts were significantly reduced in certain digits and the A-B ridge counts were also reduced.

Conclusions Further studies are required to establish a data base for Iraqi patients with diabetes (and other conditions) using larger samples and other dermatoglyphic parameters so that dermatoglyphic features may be used as a screening tool for individuals at risk of certain genetically predisposed conditions.

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Key words: Dermatoglyphics, Dibetes mellitus, Hypertension, Hyperlipidemia, Digital ridge count, Palmar A-B ridge, Dibetogenous screening

Dermatoglyphics, the study of the patterns of ridges on the skin of the fingers, palms, toes, and soles is of interest in anthropology, criminology, and medicine.[1] The importance of dermatoglyphic studies in clinical medicine is that, during development, ridge formation is affected by maternal environment, gene deviants, and chromosomal aberrations. Once formed, they are age and environment stable, becoming a reliable indicator of genetic damage.[2] The intervention of the factors responsible for the self-immune process of β insulinic cells' destruction is manifesting as early as the intrauterine life, when the papillary ridges are formed, which supports the idea of their possible utilization as "markers" in predicting persons with diabetogeneous risk.[3]

The process of dermal ridge formation begins in the sixth to seventh week of development with the formation of the elevated fetal volar pads of mesenchymal

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tissue over the end of the most distal metacarpal bone on each finger, in the interdigital areas just below the fingers, and on the hypothenar and thenar areas of the palms and soles. The epidermal ridges first appear in the form of localized cell proliferations around the 10th to 11th week of gestation. These proliferations form shallow corrugations that project into the superficial layer of the dermis. The number of ridges continue to increase, being formed either between or adjacent to existing ridges. It is during this period of primary ridge formation that the characteristic patterns are formed. At about 14 weeks the primary ridge formation ceases and secondary ridges begin to form as sweat gland anlagen begin to develop along the apices of the primary ridges at uniform intervals.[4] It's suggested that the underlying bony skeleton, the time of ossification and blood vessels and nerve formation also affect the pattern of ridge formation.[5] It should also be noted that the onset for spontaneous movement of the hand has not been reported until about the middle of the 11th week of pregnancy and fetuses are reported to begin to tightly grasp at 16 to 20 weeks. It would therefore appear that the palmar creases are genetically rather than mechanically induced.[6]

It's well-known that skin cells and the entire vertebrate nervous system develop from the ectoderm, and what controls this regional identification of the neural plate and skin is the adjacent mesoderm; the precursor of bone, connective tissue, muscle, blood, vascular and lymphatic tissue.[7]

Therefore, we believe that a set of tissues formed in the early stages of embryonic development can react identically to different dysmorphogenetic causes. This may be why some observations of dermatoglyphic patterns can be related to several physical conditions.

A number of congenital problems have left their marks on both the brain and the hand. Examples of such associations are the significant increases in palmar single flexion creases ("simian line") and Sydney creases (distal or proximal transverse crease that completely crosses the palm) and mental retardation in Down's syndrome, missing interphalangeal flexion creases in mentally retarded individuals, and "sandal" plantar creases on the soles of those with Down's syndrome and Rubinstein-Taybi syndrome.[6] Although genetic oriented diseases (e.g. Trisomy, Tay Sachs) have received the most scrutiny, correlations have been found to Alzheimer's disease, diabetes, cancer and several heart diseases.[8]

PATIENTS AND METHODS

The aim of the current study is to compare and evaluate the dermatoglyphic patterns in individuals with insulin-dependent diabetes mellitus (IDDM) with a normal population. The study included 100 IDDM patients (58 males and 42 females) randomly selected from the National Center for Diabetes Treatment and Research (Baghdad) and 80 healthy controls (50 males and 30 females)

belonging to the same demographic profile randomly selected as unrelated subjects from the staff of the center and the College of Medicine of Al-Mustansiriyah university. All the patients had a disease onset after the age of 18. The patients and controls belonged to the 20 – 50 years age range. A screening questionnaire excluded participants with diseases or congenital abnormalities thought to be associated with dermatoglyphic abnormalities. A group of patients were selected with an associated chronic condition in the form of hypertension (HPT) or hyperlipidemia (HPL) as shown in Figure 1.

Digital scans of the right hand were obtained using a high resolution (600 dpi) Genx ® computer scanner and a laptop. The scans were analyzed and tabulated for 1 qualitative variable and 2 quantitative parameters:

1. The fingerprint pattern style (qualitative): the three basic types of fingerprints were looked for regardless of the variations in the subtypes. These types are the arch, loop and whorl (Figure 2).
2. The digital ridge count: ridge counts for each finger were calculated from the number of dermal ridges that intersected or touched a straight line drawn from the central core of the fingerprint pattern to one or two adjacent triradius points. A triradius is a point where the pattern deviates into three directions (Figure 2). Consistent with standard methods, fingertips with an arch pattern were assigned a ridge count of zero and those with a loop pattern received a ridge count equal to the number of ridges crossing the single straight line to the nearest triradius point. For fingertips of the whorl patter (with two triradii) the ridge count was calculated using the following formula:

   Ridge count = (ridge number crossing the longer line) + (1/2 the ridge number crossing the shorter line).[9]
3. The palmar A-B ridge count: is the number of ridges between the palmar triradii in the second interdigital area between the index and middle fingers.[1]

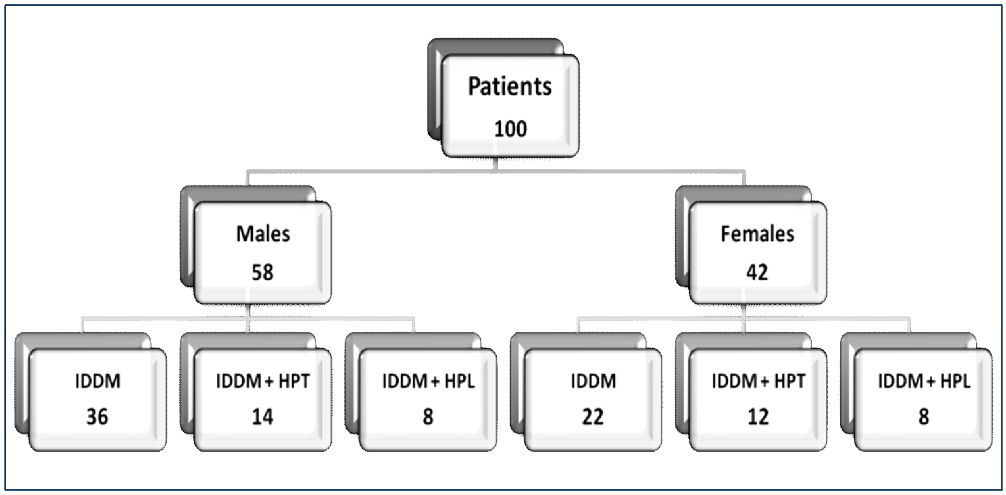


Figure 1. The distribution of the patients group

Figure 2. The Basic fingerprint patterns

RESULTS

Fingerprint Pattern Style (Table 1)

All patients with IDDM showed a significantly lower frequency of loops and higher frequency of whorls compared to controls.

The male diabetics accounted for the greater frequency of whorls compared to the female diabetics on one hand and to either sex of the control group.

The female diabetics showed a statistically significant increase in arch frequency compared to male diabetics and to either sex of the controls.

Both male and female diabetics showed whorl pattern abundance in the radial digits with decreasing frequency towards the ulnar side.

At the same time the loops appeared to be increasing in frequency of occurrence in the ulnar rather than the radial digits.

The arches were most frequent in the middle digits of diabetics, especially the ring finger of females and the middle finger of both males and females.

There was no significant difference in pattern distribution in relation to the associated hypertension or hyperlipideamia. The percentages were consistent whether the patient had diabetes alone or diabetes with hypertension or hyperlipidemia.

Digital Ridge Counts (Table 2)

There was an overall significant decrease in digital ridge counts in all digits of diabetic patients. The statistical significance ($P<0.05$), however, was limited to the ring and little fingers in males and the index, middle and ring fingers in females. Association with HPT or HPL demonstrated no statistical difference from IDDM alone.

The A-B Ridge Counts (Figure 3)

A significant decrease in A-B ridge counts was noted in all patients with IDDM. Patients with IDDM alone and IDDM with hyperlipidemia had the same significance in both male and female patients. Patients with IDDM and hypertension had a highly significant ($P<0.001$) difference in the ridge count from controls in both sexes.

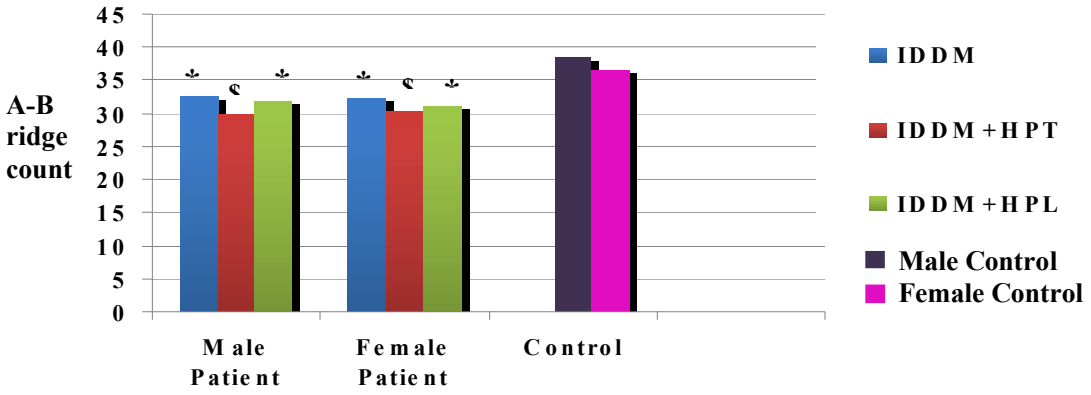
Table 1. The frequency of distribution of fingerprint patterns in the fingers of the right hands of diabetic patients and controls (data represent percentage of total patients or controls) in percentages

Digit	Pattern Style											
	Patient						Control					
	Male			Female			Male			Female		
	Whorl	Loop	Arch	Whorl	Loop	Arch	Whorl	Loop	Arch	Whorl	Loop	Arch
Thumb	11	0.6	0	5.6	1.6	1.2	3.75	8.75	0	2	4.25	1.25
Index	10	1	0.6	4	2.2	2.2	3	9.5	0	0.25	6.5	0.75
Middle	7.2	3	1.4	2	4	2.4	2.75	9.25	0.5	2.75	4.5	0.25
Ring	4.4	7.2	0	1.6	4.2	2.6	1	11.25	0.25	1.75	4.75	1
Little	4.4	7.2	0	0.8	6	1.6	2	10	0.5	1.5	5.5	0.5
Total	37	19	2	14	18	10	12.5	48.75	1.25	8.25	25.5	3.75
Summary	Whorl			51			Whorl			20.75		
	Loop			37			Loop			74.25		
	Arch			12			Arch			5		

Table 2. The digital ridge counts of the right hand fingers of diabetic patients and controls.

Digit	Ridge Count			
	Patient		Control	
	Male	Female	Male	Female
Thumb	22.5 ± 8.6	19.2 ± 7.8	24.1 ± 8.3	21.2 ± 8.2
Index	18.1 ± 7.1	12.1 ± 5.1 (*)	19.6 ± 7.9	16.3 ± 6.7
Middle	14.2 ± 5.8	11.8 ± 5.1 (*)	18.5 ± 7.7	19.2 ± 7.2
Ring	16.7 ± 7.4 (*)	11.2 ± 4.7 (*)	22.7 ± 7.6	17.2 ± 6.5
Little	11.5 ± 4.8 (*)	12.4 ± 5.5	17.5 ± 6.9	16.8 ± 6.1

(Data represent mean ± standard deviation,=P<0.05 {significant})*



*(Data presented as mean ± standard deviation, *=P<0.05 {significant}, $=P<0.001 {highly significant})*

Figure 3. The A-B ridge counts in n the right hands of diabetic males and females compared to the controls

DISCUSSION

It is a general truth that disordered early fetal development accounts in part for the aetiology of IDDM.[10] It's also documented that on each fingertip, the number of primary dermal ridges (digital ridge count); which is dependent on the fingerprint pattern; provides a measure of fingertip growth activity during the early fetal period.[11]. Kahn *et al* [9] neurologically related each fingertip to a spinal cord segment in a range that includes the sixth to the eighth cervical levels (C6-C8). The thumb is linked to the cephalic part of C6 and the little finger is linked to the caudal part of C8. Therefore it was hypothesized by some authors that changes in the radio-ulnar direction might reflect circumstances associated with cepalo-caudal growth of the fetus. We too suggest that a prenatal exposure that contributes to metabolic programming by impairing or redistributing fetal growth occurs at a critical time window that affects the digital and palmar dermal ridge growth in a specific pattern.

Our result of the increasing whorls and decreasing loops in the radio-ulnar direction supports the cephalo-cuadal growth change theory.

Although the whorl patterns were greater in diabetics, the digital ridge counts were decreased. This may be associated with the increase in the arch patterns (arches have a zero ridge count) but there also appears to be a change in the pattern but a decrease in dermal ridge growth (i.e. whorls having less ridges than loops).

The arch pattern frequencies being highest in the middle and ring fingers may be associated with the timing of an event during fetal growth (related to the growth of these digits) rather than a progressive cephalo-caudal (radio-ulnar) process of changes.

Our results were consistent with the study of Sant *et al* [12] regarding the increase in the frequency of whorls in diabetic males and females and the specific increase of arch patterns in diabetic females. Vera *et al* [13] and Verbov [14] found similar results regarding the arch pattern frequency. However, Verbov described a decrease rather than an increase in whorl patterns.

Kahn *et al* [9] also reported the peculiar decrease of digital ridge counts associated with an increase in whorls and hypothesized a growth inhibition event affecting the whorl growth in fetal life.

Our finding of a decreased A-B ridge count concurs with the findings of Ziegler *et al*[15] and Verbov. The greatest association of decreased A-B ridge counts of patients with IDDM and HPT suggests a greater fetal event or possibly more than one event affecting more proximal fetal tissue growth. Shield *et al* [10] was unable to reproduce the A-B ridge changes in their study.

The absence of statistical significance in our three parameters in patients with IDDM and HPL as compared to other diabetics suggests that the hyperlipidemia in these patients is a metabolic complication rather than an associated or predisposing condition with prenatal grounds.

CONCLUSION AND RECOMMENDATIONS

One of the problems with dermatoglyphic studies is the capacity to look at large numbers of variables within very complex formations in small sample sizes. Statistical errors, control group inadequacy and racial differences also result in disagreement between the findings of different authors.

The results of this study suggest that there can be an association between dermatoglyphics and IDDM and possibly hypertension. The degree of predictability and the possible use to support medical diagnoses requires further studies to be undertaken on larger samples using more dermatoglyphic variables and parameters. The ability to reproduce certain results will greatly support this study and any other future studies. Then, dermatoglyphics can serve as a ready screener to select and follow-up individuals from a larger population for further investigations to confirm or rule out conditions like IDDM.

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پوختە

نیشانێت ڤەبر ییێت پیستی لجەم نەخوشێن عیراقی ییێن تووشی نەخوشیا شەکرێ یا بهیڤیا ئەنسولینی بووین.

پیشەکی: نیشانێت ڤەبر ییێت پیستێ سەرڤە (نەخشا ناڤا دەستی) ل دەلیڤێت تەنگاڤ پێك دهێت ژ پێشکەفتا گیانەوەری دەمێ پاشماییێت خەلەوی تووشی وان هوکارا دبن کە دبنە ئەگەرێ پەیدابوونا کێماسییێ و چەند دۆخێت نەخوشیا ل تەمەنێت داهاتی دا پەیدا دبن. نیشانێت پیستی ییێت شوین تبلا و ناڤا دەستی دهێنە هژمارتن وەن ئامیرەك گونجای بو زانینا سالوخەتێت ویراسی و گیانەوەری ییێت کاریگەر ل هندەك نەخوشیا چونکە ئەڤ نیشانە د گونجاینە بدرێژاهیا دەمی.

ئارمانج: هوکارێت ویراسی دهێنە هژمارتن ئێك ژ ئەگەرێت سەرەکی بو نەخوشیا (شەکرێ) یا بهیڤیا ئەنسولینێ ڤە یە. ژ بەر ڤێ چەندێ و ژ ئارمانجێت ئەڤێ خاندنێ دیارکرنا پەیوەندییێ دناڤبەرا ئەڤێ هوکارێ ویراسی و جورێت نیشانێت پیستی ب سەرڤە ل هەر دوو دەستا

ڕێکێن ڤەکولینێ: سێ بیڤەر ژ نیشانێت پیستی ییێت دەستی هاتینە خاندن ئەوژی جورێ شوین تبلا و هژمارەك ژ ڤەبرێت پیستی ییێت هەر شوین تبلەکێ و هژمارا ڤەبرا ل دەڤەرا (A-B) ل ناڤا دەستییە و ڤەکولینێت سەرژمێری لسەر ڤان پیڤەرا هاتیە کرن و ئەنجامێت وێ دیاردکەن ب زێدەبوونەکا بەرچاڤ شوین تبلێت بشێوێ بازنە و کڤان.

ئەنجام: نزم بوونەکا بەرچاڤ بو هژمارا ڤەبرێت پیستی ل هندەك تبلا و ل دەڤەرا (A-B) ژ ناڤا دەستی ئەڤ ئەنجامە د وەك هەڤن د گەل هژمارەکا زور ژ وان خاندنێت هاتینە کرن ل دەڤەرێت دی ژ جیهانێ بەلێ د جیاواز بوون دگەل هندەك خاندنێت دی.

دەرئەنجام: هژمارەکا زۆر ژ ڤەکولینا دڤێت بهێنە کرن و بکارئینانا پیڤەرێن دی بو خاندنێ لسەر نموونێت مەزنتر ژ نەخوشا ژ بو داناانا بناغەیەکێ ئاشکرا کە بیتە بنیات ل پاشەرۆژێ ژ بو ڤەوژاردنێ و لدیڤ چوونا ئەوان کەسێت توشی نەخوشیا شەکرێ دبن لدیڤ نەخشا ناڤا دەستی.

الخلاصة

أنماط مرتسم الكف لدى المرضى العراقيين المصابين بالسكري المعتمد على الإنسولين

خلفية البحث: تتكون علامات الحيود الجلدية السطحية في فترات حرجة من التطور الجنيني حين تكون المورثات الخلوية عرضة للعوامل التي قد تؤدي الى خلل أيضي ينتج عنه حالات مرضية في سنوات العمر اللاحقة. و تعتبر العلامات الجلدية لبصمات الأصابع والكف (مرتسم الكف) أداة مناسبة للتعرف على الخواص الوراثية و الجنينية المؤثرة في بعض الأمراض كون هذه العلامات ثابتة بمرور الوقت.

الأهداف: يعتبر العامل الوراثي أحد المسببات الرئيسية لمرض السكري المعتمد على الأنسولين، و عليه هدفت هذه الدراسة الى كشف العلاقة بين هذا العامل الوراثي و أنماط العلامات الجلدية السطحية في اليدين .

طرق البحث: تم دراسة ثلاثة معايير من العلامات الجلدية اليدوية لمئة من مرضى السكر المعتمد على الإنسولين ، و هي نمط بصمات الأصابع وعدد الحيود الجلدية لكل بصمة و تعداد الحيود في المنطقة A-B من كف اليد و أجريت البحوث الإحصائية على هذه المعايير.

النتائج: أظهرت النتائج زيادة ملحوظة في تواتر البصمات الدوامية و القوسية الشكل و انخفاض ملحوظ لعدد الحيود الجلدية في بعض الأصابع و في المنطقة A-B من الكف لدى المرضى.

الاستنتاجات: توافقت هذه النتائج مع العديد من الدراسات التي أجريت في مناطق أخرى من العالم و لكنها اختلفت مع بعض الدراسات الأخرى مما يدعو لإجراء المزيد من البحث وشمول مقاييس اخرى للدراسة على عينات أكبر من المرضى لوضع اساس بياني قد يمكن الاعتماد عليه مستقبلا لفرز و متابعة الأشخاص الذين قد يكونون عرضة للإصابة بالسكري اعتمادا على مرتسم الكف لديهم.

COMPARATIVE STUDY OF CURRENT ANTIMICROBIAL RESISTANCE PATTERNS OF UROPATHOGENS: EVALUATION OF EMPIRICAL TREATMENT

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ABSTRACT

Background Urinary tract infection is a common disease in the community. There is a huge problem in management of these cases in general practice, which mainly attributed to misdiagnosis and misuse of empirical therapy.
Objective The aim of this study is to determine the distribution of uropathogens in Northern Iraq together with their in vitro susceptibility profiles to antimicrobial agents
Methods The study included urine samples submitted for culture and antibiotic susceptibility examination for inpatient and outpatient cases. Mosul samples were from inpatient admitted to Al-Salam teaching hospital for a one year period of 2005. Duhok samples were from outpatients sent to the Laboratory of one of the two main private hospital in the city for a one year period of 2006. All samples were tested microbiologically by standard procedures and cultured quantitatively. Colony count was estimated after overnight incubation at 35°C.
Results A total of 1692 and 842 samples were sent for urine culture for inpatient and out patient cases of which 31% and 60.6% of them showed a significant bacteruria respectively. *Escherichia coli* was the commonest bacteria isolated represented above 40% of total isolates in both groups. The other main bacterial isolates from inpatients were *Klebsiella spp.* 18.9%, *Staphylococcus. aureus* 8.6%, *Psendomonas aeruginosa* 7.0% and *Proteus spp* 6.5%, while in out patients were; according to frequency, *Proteus spp.* 30.7%, *Enterococcus. fecalis* 7.8% and *Staphylococcus saprophyticus* 7.4%. The study showed a high emerging resistance for most commonly used antibiotics in general practice; with overall increase in antibiotic resistance profile mainly among inpatients. Empirical treatment was chosen as a general guideline for treating urinary tract infections in the North of Iraq.
Conclusions The data provide much needed information on the prevalence of antimicrobial resistance amongst pathogens currently causing UTI in hospitals and community in the North of Iraq.

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Key words: Urine, Antimicrobial susceptibility, Resistance

Urinary tract infection (UTI) is the most common nasocomial infection among hospitalized patients representing about 40-60% of all hospital infections.[1-3] Also the community acquired UTI is the most frequent problem in primary care, mainly among women without underlying diseases or anomalies.[4]

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Antimicrobial susceptibility of bacteria causing UTI has evolved over several decades as antimicrobial exposure has repeatedly been followed by emergence of resistance.[1,4] Older populations in the community and long-term care facilities, have increased prevalence of resistant bacteria isolated from UTI.[5] Also microorganisms responsible and their antimicrobial susceptibility have been varied between a community acquired infections and patients admitted to hospital. The latter has shown a wider resistant to commonly used antibiotics.[5,6] Moreover it has been postulated that infections with resistant organisms may increase the risk of treatment failure and morbidity. Accordingly it is highly essential to measure the incidence of resistance in uropathogens and to find the suitable effective antibiotic profiles.[7]

The aim of this study was to determine the distribution of uropathogens in Northern Iraq together with their in vitro susceptibility profiles to antimicrobial agents between community acquired and nasocomial infections. This is expected to act as a local guideline for empirical treatment of UTI and to minimize the emerging resistance from misuse of antibiotics.

PATIENTS AND METHODS

The study was conducted in Mosul and Duhok, by prospective follow up of cultures by authors. Mosul is the center of Nineveh governorate which is inhabited by more than 2 million population and situated 400Km North of Baghdad. Duhok is the center of Duhok governorate with a population of about 1 million situated at about 65Km North of Mosul in the Kurdistan region of Iraq.[8,9]

The study included all urine samples submitted for culture and antibiotic susceptibility examination at microbiology laboratory. Mosul samples were from inpatients admitted to Al-Salam teaching hospital for a one year period from 1st January to 31st December 2005. This hospital is one of the two major teaching hospitals in Mosul serving mainly the west bank of Mosul city. A total of 3359 samples have been sent to the microbiology laboratory for culture; of which 1692 samples were for urine culture.

Duhok samples were from outpatients sent to the Laboratory of one of the two main private hospitals in the city for a one year period from 1st January to 31st December 2006. A total of 2216 samples were sent to this laboratory of which 842 samples were for urine culture.

All Urine samples were expected to be a mid stream samples collected in a sterile container. All urine samples were tested microbiologically by standard procedures and cultured quantitatively on blood and MacConkey agar by calibrated loop technique, using a standard 1microliter of urine inoculums.[10]

Colony count was estimated after overnight incubation at 35^0C and results were interpreted and pathogen identified with utmost care according to clinical condition of the patients.

Urine culture results were divided

according to WHO classification into three major groups[10]:

A. Significant bacteruria where total counts were considered and reported as clinically relevant cases for diagnosis of UTI.

B. No significant bacteruria This included cases where the total counts were considered and reported as probable absence of U.T.I when a mixed growth equal or more than three organism's types. No pure predominating pathogen.

C. No growth this included all cases showing sterile urine with no growth after 48 hours incubation.

Significant bacteruria was subdivided by colony count into three minor subgroups:

Category I

Total count was more than 10^5 colony forming unit (CFU)/ml. These bacterial counts are strongly suggestive of UTI in all patients; including asymptomatic females if one or two different colony types of bacteria are present.

Category II

Total count of at least 10^4 -10^5 colony forming unit (CFU)/ml of a single potential pathogen or for each of two potential pathogens in clean catch midstream (CCMS) urine strongly suggest UTI in symptomatic patients, in cases of pyelonephritis, acute cystitis, and asymptomatic bacteruria.

Category III

Total count of $\geq 10^4$CFU/ml of a single potential pathogen in CCMS urine/in cases of symptomatic males or catheterized urines or acute urethral syndrome.

$\geq 10^2$ CFU/ml of any number of organism types in cases of symptomatic young females, suprapubic aspirates, or any other surgically obtained urines (including cystoscopy specimens).

All cases of significant bacteruria were submitted for antibiotic susceptibility tests for commonly used antibiotics by conventional agar diffusion antibiotic sensitivity assay, using modified Kerby-Bauwer technique according to CLSI (Clinical and Laboratory Standards Institute) guidelines , using Mueller - Hinton agar standard media.[10] The inhibition zone standards for antimicrobial susceptibility were considered from tables for interpretive zone diameters of Clinical and Laboratory Standards Institute (CLSI).[11]

RESULTS

Out of 1692 urine cultures in Mosul, 1280 samples (75.7%) were from females. Table 1 shows culture results of hospital cases in Mosul, where a total of 525(31%) patients had a significant bacteruria with 387(73.7%), 135(25.9%) and 3 (0.6%), for colony count category I, II, and III, respectively. Moreover 414 (24.5%) cases showed no significant bacteruria with no significant growth; while the rest of 753 cases (44.5%) had no bacteruria with sterile urine on culture.

Table 1. Inpatients' urine culture results/ Al-Salam hospital in Mosul

Type of growth	Colony forming unit (CFU) / ml	No. (%)
Significant growth	$<10^4$ _ $\geq 10^2$	3 (0.6)
	$>10^4$ - 10^5	135 (25.9)
	$> 10^5$	387 (73.7)
	Total	525 (31.0)*
No significant growth		414 (24.5)*
No growth		753 (44.5)*
All categories		1692 (100.0)

* *Out of total urine cultures*

In Duhok out of 842 urine cultures 594(70.5%) were from females. Table 2 reveals culture results for community cases in Duhok where a total of 496 (59.0%) patients had a significant bacteruria with 275(30.5%), 147(17.5%) and 92(11.0%), for colony count category I, II, and III respectively. Moreover 116 cases (13.8%) showed no significant bacteruria with no significant growth; while the rest of 230 (27.3%) cases had no bacteruria with sterile urine on culture. Tables 3 and 4 show the frequency of bacterial isolates from urine culture for both categories. *Escherichia coli* were the commonest bacteria isolated represented above 40% of total isolates in both groups. The other main bacterial isolates from inpatients were *Klebsiella spp.* 18.9%, *Staphylococcus. aureus* 8.6%, *Psendomonas aeruginosa* 7.0% and *Proteus spp* 6.5%, while in out patients were; according to frequency, *Proteus spp.* 30.7%, *Enterococcus fecalis* 7.8%, *Staphylococcus saprophyticus* 7.4%. The least common isolates detected were *Enterococcus fecalis* 5%, *Staphylococcus epidermidis* 4.6% and *Candida albicans* 1.1%.

Table 5 reveals the resistance frequency among bacteria isolated from inpatient samples. *E.coli* was found to be highly resistant to Ampicillin and Trimethoprim 94.6%, Co-Trimoxazole 91.9% and Rifampicin 98.7%, with lower resistance to Ciprofloxacin, Cephalexin, Tetracycline and Cefotaxime 85.7%, 78.5%, 75.8, 69.1% respectively, However the lowest resistance was with Nitrofurantoin 13.5%, Nalidixic acid 48.9% and Gentamicin 49.3%.

As for other coliforms including *Klebsiella spp., Enterobacter aerogenes,* and *Proteus spp.,* they were found to be resistant to Ampicillin, in a range of 93.9%-96.2%, except for Proteus 76.5%. High resistance was noted for both sulpha drugs in a range of 96.2%- 100% with the exception of *Klebsiella spp* 86.9%; however, lower resistance was noted for other gram negative uropathogens compared to *E.coli* except for *Proteus spp* .reflected by a resistance ranges of 78.7-88.5%, 67.6-73.1%, 68.0%-69.2%, 57.7%-70.6% for Ciprofloxacin Cephalexin, Tetracycline, and Cefotaxime respectively with the lowest range with Nitrofurantoin19.2%-26.9%, and

Gentamicin 26.9-36.4%. *Proteus spp* showed a striking increase in resistance with Ciprofloxacin 97.1%, Tetracycline 91.2% and Nitrofurantoin 82.4% compared to lower resistance only with Gentamicin 38.2% and Nalidixic acid 50.5%.

On the other hand the gram positive bacteria including, *Staphylococcus epidermidis, Staphlococcus aureus Enterococcus fecalis* showed relatively lower pattern of resistance for Ampicillin in 91.7%, 86.7%, and *51.6*% respectively, but not for Trimethoprim and Co-Trimoxazole drugs where high resistance was noted for the majority and the lowest was in *Staphylococcus aureus* 79.8%.

High resistance was also recorded for Rifampicin and Nalidixic acid in all gram positive uropathogens however, the first generation Cephalosporin Cephalexin showed a relatively moderately resistance rates ranging between 80.0-87.5 %, as well as Ciprofloxacin 70.8-88.9% Tetracycline 71.1-83.9%,compared to a lower rates with and 3rd generation cephalosporines Cefotaxime 42.2-75.0%.

The Lowest resistance frequencies were noted in gram positive uropathogens with Gentamicin 38.7-50.5% and Nitrofurantoin 9.7-20.8 %.

Finally table 6 shows the resistance frequency among bacteria isolated from out patient sample. *E. coli* was found to resist Trimethoprim, Amikacin, Tetracycline and Gentamicin in 76.4%, 71.4%, 66.4% and 58.8% respectively. Relatively moderately resistance rates were detected for Cephalexin 11.8% Ciprofloxacin 23.3%, Amoxiclave 24.4%, Nalidixic adic acid 30.2% and Nitrofurantoin 37.8%. On the other hand the resistant profile was relatively similar for *Proteus spp.*and Stapylococcus. *saprophyticus* isolates when the highest resistance rates were reported for Tetracycline 80.3% and 88.1% respectively. Above 50% of both bacteria were resistance to Gentamicin and around approximately one third to Nalidixic acid, Amikacin, and Amoxiclav.

The resistance rate however, was higher among, *Proteus spp.*, *Enterococcus fecalis and Stapylococcus saprophyticus* for Ciprofloxacin , Nitrofurantoin and Trimethoprim, 76.9%,48.6 % and 49.7 % in comparisons to 27.3% 47.7%, 29.5% and 35.7%, 31.0%, 21.4% respectively.

DISCUSSION

The study revealed that urine culture represented about 50% of total culture load submitted for microbiology laboratory. Also about three quarters of samples were for female in a rate of (75.7%), (70.5%) in Mosul and Duhok respectively. This is expected as UTIs are very common complaint encountered both in hospitals and community particularly among females.[1] This also indicates the necessity of improving the quality of this procedure and the importance of continuous monitoring of bacterial resistance profile.[1,12]

The study revealed that urine culture represented about 50% of total culture load submitted for microbiology laboratory. Also about three quarters of samples were

Table 2. Outpatients' urine culture results in Duhok

Type of growth	Colony forming unit (CFU) / ml	No. (%)
Significant growth	$<10^4$ _ $\geq 10^2$	92 (11.0)
	10^4 - 10^5	147 (17.5)
	$> 10^5$	257 (30.5)
	Total	496 (59.0)*
No significant growth		116 (13.7)*
No growth		230 (27.3)*
All categories		842 (100.0)

* *Out of total urine cultures*

Table 3. Microorganisms isolates from urine culture- inpatients

Isolates	No. (%)
Escherichia.coli	223 (42.5)
Klebsiella spp.	99 (18.9)
Staphlococcus aureus	45 (8.6)
Psendomonas aeruginosa	37 (7.0)
Proteus spp.	34 (6.5)
Enterobacter aerogenes	31 (5.9)
Enterococcus fecalis	26 (5.0)
coagulase-negative Staphlococcus epidermidis	24 (4.6)
Candida albicans	6 (1.1)
Total	525 (100.0)

Table 4. Microorganisms isolates from urine culture- outpatients

Isolates	No. (%)
Escherichia coli	262 (46.5)
Proteus spp.	173 (30.7)
Enterococcus fecalis	44 (7.8)
Staphylococcus saprophyticus	42 (7.4)
Klebsiella spp.	25 (4.4)
Psendomonas aeruginosa	18 (3.2)
	564 (100.0)

NB. Total isolates were more than the number of cases as some had mixed growth

Table 5. Resistance frequency among isolated bacterial pathogens towards antimicrobial agents – inpatients

Antimicrobial	Resistance frequency (%)						
	E. coli	*Klebsiella spp.*	*Staphylococcus aureus*	*Proteus spp.*	*Enterococcus. fecalis*	*Enterob. aerogenese*	*Staphylococcus epidermidis*
Ampicillin	94.6	93.9	86.7	76.5	51.6	96.2	91.7
Co-Trimoxazole	91.9	86.9	88.9	97.1	93.5	100.0	91.7
Trimethoprim	94.6	100.0	79.8	100.0	93.5	96.2	100.0
Cephalexin	78.5	72.7	80.0	67.6	83.9	73.1	87.5
Cefotaxime	69.1	67.7	42.2	70.6	64.5	57.7	75.0
Gentamicin	49.3	36.4	40.0	38.2	38.7	26.9	50.5
Nitrofurantoin	13.5	19.2	15.6	82.4	9.7	26.9	20.8
Tetracycline	75.8	68.0	71.1	91.2	83.9	69.2	75.0
Nalidixic acid	48.9	50.5	97.8	50.5	83.9	8.5	100.0
Ciprofloxacin	85.7	78.8	88.9	97.1	87.1	88.5	70.8
Rifampicin	98.7	100.0	97.8	100. 0	93.5	96.2	100.0

Table 6. Resistance frequency among isolated bacterial pathogens – outpatients

	Escherichia coli	*Proteus spp.*	*Enterococcusfecalis*	*Staphylococcus saprophyticus*	*Klebsiella spp.*
Amoxiclav	24.4	24.3	40.9	31.0	16.0
Trimethoprim	76.7	49.7	29.5	21.4	32.0
Cephalexin	11.8	16.8	90.9	4.8	16.0
Nalidixic Acid	30.2	37.0	70.5	26.2	20.0
Nitrofurantoin	37.8	48.6	47.7	3.0	16.0
Tetracycline	66.4	80.3	43.2	88.1	64
Gentamicin	58.8	58.4	72.7	54.8	48.0
Amikacin	71.4	36.4	36.4	38.1	56.0
Ciprofloxacin	23.3	76.9	27.3	35.7	4.0
Total number tested	262	173	44	42	25

for female in a rate of (75.7%), (70.5%) in Mosul and Duhok respectively. This is expected as UTIs are very common complaint encountered both in hospitals and community particularly among females.[1] This also indicates the necessity of improving the quality of this procedure and the importance of continuous monitoring of bacterial resistance profile.[1,12]

About 31% of hospital samples showed a significant bacterial growth out of which 73.7% of cases with total count more than 10^5 CFU/ml in comparison to about 59% from community derived samples with a rate of 30% with a total count more than 10^5 CFU/ml.

The study showed that cases referred to hospital had high rates of no significant / no growth results (69%) compared to community cases where the rates were much lower for both categories (41 %). This might be explained by different factors. First the selection of highly indicated patients to be send for urine culture in the community due to the high expenses of the procedure in comparison to the randomized referral by clinicians for free culture test without presumptive evidence of infection by lab test like general urine examination in governmental hospitals. The second reason might be the factor that the majority of community urine cultured is requested by specialist in comparison to specialist and senior house officers in hospital. The precision of clinically suspected UTI is certainly higher among specialist in comparison to general doctors. Moreover the data revealed that the quality of sampling was much better with the community cases where strict instruction and care was expected for patients in sampling, this was reflected by the low rates of no significant growth results which was 13.8 % in community cases, compared to higher rates 24.5% in hospital cases. Another important finding was Candida albicans infections in female patients detected in a rate of 1.1% in hospital acquired infections which can be explained by the misdiagnosis of UTI with vaginosis due to shared symptoms,[13] or the suggestion of over treatment with antibiotics which can ultimately end with Candidiasis.

E.coli was the commonest isolates from both in and out patients representing above 40% of all isolates.This is similar to rates reported in Gaza strip, Palestine and Germany[14,15] but slightly lower than those reported in similar studies conducted in Nepal India, Italy where the rates were 50.7%, 54.7% and 59.4% respectively.[2,12,16] The rates of *E.coli* reported in this study were, however, significantly lower than those found in Turkey (87%), United Kingdom (56.3%-77.3%) and USA (75%-90%).[17-19] These variation illustrates the importance of conducting similar local studies, especially as a significant number of UTIs are treated empirically; even in developed countries.

The commonest four pathogens isolated from inpatients were *Klebsiella spp., Staphylococcus aureus* followed by *Pseudomonas and Proteus spp.*, while in community samples they were *Proteus spp., Enterococcus fecalis, Staphylococcus saprophyticus* and *Klebsiella spp*. Despite variation in their

frequencies this is similar to findings of other studies .[2,13,16,17]

Pseudomonus aeruginosa was isolated from about 3% of outpatient samples compared to inpatients 7% and this is similar to the finding reported in Croatia and Canada from a community acquired infections, where the rates were at 3.5% and 2.9% respectively.[20,21]

Evaluation of antimicrobial resistance of isolates from urine samples is highly essential both for empirical and specific treatment of UTIs. There has been a difference in the quantity of types antibiotic discs used in cultures conducts in the hospital versus those conducted in the private lab. The latter has used relatively smaller number of antibiotic disc, mainly to reduce the expenses of the test.

Nevertheless, the resistance of *E.coli* to Sulpha drugs was very low in both settings; 94.6% in hospital and 76.4% in outpatient samples. This is important as these drugs are frequently used for empirical treatment in suspected cases of UTI. These findings were similar to studies conducted in Italy and Turkey which also indicated that the empirical antibiotic therapy of patients with UTI should always be guided according to current antimicrobial resistance.[16, 17] *E.coli* was found to be resistant to amoxiclav in only 24.4% of community samples. This is similar to the rate reported in United Kingdom (22.2%) and was thought to be due to inhibition of beta-lactamase action due to the addition of clavulanic acid, in comparison to high resistant rate reported with amoxicillin.[18]

The study showed that the profile of resistance frequencies was much higher in hospital than community samples and for almost all isolates. This is expected as nasocomial infections have originally highest primary resistance.[22]

CONCLUSIONS AND RECOMMENDATIONS

The data provide the prevalence of antimicrobial resistance amongst pathogens currently causing UTI in hospitals and community in the North of Iraq.

The study reflected the high resistance frequencies to both most commonly empirically used drugs in UTI (Ampicillin and Co-Trimoxazole). Accordingly it is recommended to consider the use of the first line drugs Amoxiclav and first generation Cephalosporines with Nalidixic acid and Nitrofurantoins as the drugs of choice in empirical UTI treatment of simple community acquired cases in, keeping the Ciprofloxacin as a second line measure due to its highly acquired resistance as well as its beneficial indications in other infections.

The study also reflects the alarming emerging uropathogens resistance in cases of hospital acquired U.T.I with the recommendation of Ciprofloxacin, Gentamicin, and second generation Cephalosporins for empirical treatment while awaiting the culture and sensitivity results. It is also recommended to continuously monitor the pattern of urinary pathogens and updating their resistance profiles to clinicians. However simple

measures applied in governmental hospitals including training of staff for proper sampling and strict rules should be restrained on guidelines and formulary for resident doctors and general practitioners is indicated to minimize the misdiagnosis and misuse of antibiotics in addition to decrease the expenses on patients in private laboratory and using culture only in chronic cases by treating simple community cases with empirical profile above.

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پوختە

لێکولینەکا بەرامبەردار لسەر شێوازێ بەرگرتنا ئەنتی بایوتیکا لدەف بەکتریایێن ڕێزکا میزێ

پێشەکی: هەودانا ڕێزکا میزێ نەساخیەکا بەربەلاڤە دناڤ کومە لگەهێدا . ئارێشەکا مەزن یاهەی د چارەسەرکرنا ئەڤان نەخوشیاندا لدەف پێشەکارێن گشتی , ئەو ژی د گرێدای شاشیێ د نیشان ناسینێ و بکارئینانا دەرمانان بشێوێ ئەزمونکاری .

ئارمانج: ئارمانج ژ ڤێ ڤەکولینێ ئەو بو دیارکرنا ڕێژا بەڵف بوونا بەکتریایێن ڕێزکا میزێ ل باکورێ عیراقێ , دگەل دیارکرنا ڕێژا بەرگرتنا وان بو ئەنتی بایوتیکا ئەوێت هێنە بکارئینان.

ڕێکا کاری: د ڤێ ڤەکولینێ دا نمونێت میزێ ئەوێت هاتینە هنارتن ژبو چاندنێ و دیارکرنا کارتێکرنا دەرمانان, ییت وان نەخوشان بوون ئەوێت تقستی د نەخوشخانا السلام ل باژێرێ میسل ل سالا 2005 هەر وەسا نەخوشێت کلینیکا دەرڤە ل ئێك ژ دوو نەخوشخانێت تایبەت ل باژێرێ دهوکێ ل سالا 2006 .

هەمی نمونە هاتنە تاقیکرن بکارئینانا ڕێکێن ستاندار بو چاندنێ و ئێش ناسینێ و کارتێکرنا دەرمانان و هەر وەسا هەژمارتنا ژمارا کولونییێت بەکتریا .

ئەنجام: 1692 و 842 دا نمونێت میزێ هاتینە هنارتن ژبو چاندنێ ژ وان نەخوشان بوون ئەوێت تقستی د نەخوشخانێ دا هەر وەسا نەخوشێت کلینیکا دەرڤە . ئەنجاما دیارکر کو 31٪ و 60.6٪ ژوان ڕێژەکا بەرچاڤ هەبو ژ شینبوونا ئەو بەکتریت بنە ئەگەرێ هەودانا ڕێزکا میزێ ل دیڤ یەك. بەکتریت E.coli ڕێژا پتر ژ 40٪ ژ وان بەکتریت هاتینە جوداکرن بوون . گرنگترین جوریت بەکتریت هاتینە جوداکرن لدف نەخوشێت تقستی د نەخوشخانێ دا ئەڤە بوون: Klebsiella spp. 18.6٪، Staph. Aureus 8.6٪، Pseudomonas spp. 7.0٪، Proteus spp. 6.5٪. بەلێ پا لدف نەخوشێت کلینیکا دەرڤە ئەڤە بوون: Proteus spp. 30.7٪، Enterococcus spp. 7.8٪، Staph. Saprophyticus 7.4٪. ئەڤێ ڤەکولینێ دیارکر پەیدابوونا ڕێژەکا بلند ژ بەرگرتنێ ژ بو ئەنتی بایوتیکێت تێنە بکارئینان بشێوەکێ بەربەلاڤ دکریارێن نوژداریێن گشتی دگەل بلندیەکا بەرز بتایبەتی لدف نەخوشێت تقستی د نەخوشخانێ دا . پیشنیارا بکارئینانا چارەسەریا دەست پێکی و یا باش بو چارەسەرکرنا هەودانا ڕێزکا میزێ هاتە دان بو کریارێن نوژداریێن گشتی ل با کورێ عیراقێ.

دەر ئەنجام: ئەڤێ ڤەکولینێ پێزانینێن گرنگ دیارکرن ل دور بەربەلاڤبوونا بەرچاڤ یا بەرگرتنێ ژ بو دەرمانان لدەف ئەو بەکتریت بو ینە ئەکرێ هەودانا ڕێزکا میزێ ل با کورێ عیراقێ.

الخلاصة

دراسة مقارنة لنمط المقاومه للمضادات الحيوية في جراثيم المجاري البولية: تقييم استخدام العلاج الاولي

الخلفية: يعتبر خمج الجهاز البولي من الامراض الشائعة في المجتمع. هنالك مشكلة كبيرة في علاج هذه الحالات في الممارسة الطبية العامة والتي تعزى اساسا الى عدم دقة التشخيص وسوء استخدام العلاج الاولي للحالات المسببه للالتهاب

الهدف : هدفت الدراسة الى قياس نسبة انتشار الجراثيم المجاري البولية في شمال العراق مع قياس نسبة مقاومتها للمضادات الحيوية المستخدمة.

طريقة البحث: شملت الدراسة نماذج الادرار المرسلة لغرض الزرع وقياس الحساسية الدوائية للمرضى الراقدين في مستشفى السلام في مدينة الموصل خلال سنة 2005 والمرضى المراجعين للعيادة الخارجية في واحدة من المستشفيين الخاصين في مدينة دهوك لعام 2006. تم فحص جميع النماذج باستخدام الطرق القياسيه للزرع و التشخيص و لحساسية الدوائية. تم قياس عدد المستعمرات بعد الحضن بدرجة 35 درجة مئوية.

النتائج: تم ارسال 1692 و 842 نموذج ادرار لغرض الزرع من المرضى الداخلين في المستشفى ومرضى العيادة الخارجية وقد اظهرت النتائج ان 31% و 60.6% منهم نمو قياسي لخمج المجاري البويلة علي التوالي. شكلت نسبة جرثومة ال E.coli اكثر من 40% من العزلات في كلا المجموعتين. كانت اهم العزلات الراقدين في المستشفى هي: Klebsiella spp 18.6%، Staph. Aureus 8.6%، Pseudomonas spp. 7.0%، Proteus 6.5%. بينما كانت عزلات مرضى العيادة الخارجية:Proteus spp. 30.7%، Enterococcus spp. 7.8%، Staph. Saprophticus 7.4%. اظهرت الدراسة ظهور نسبة مقاومة عالية للمضادات المستخدمة بصورة واسعة في الممارسة الطبية العامة مع زيادة كبيرة خاصة للمرضى الراقدين في المستشفى. تم اقترح العلاج الذي يحبذ استخدامه كخط اول لعلاج خمج المجاري البولية للمارسة العامة في شمال العراق.

الاستنتاجات: اظهرت الدراسة معلومات مهمة حول الانتشار الفقطي للمقاومة الدوائية لجراثيم المسببة للخمج البولي في المجتمع شمال العراق.

CASE REPORT
SINUS PERICRANII: AN UNCOMMON TORTUOUS VASCULAR ANOMALY WITH TRAUMATIC ORIGIN

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ABSTRACT

Sinus pericranii is an unusual venous anomaly characterized by communication of pericranial varicosities with an underlying dural sinus. The varicosities are intimately associated with the periostium, are distensible, and vary in size with changes in intracranial pressure. Symptoms are infrequent but include headache, vertigo, feelings of fullness, local pain, or dermatological lesion. It presents at the paediatric age group more than other age groups and it affects males more than females. Although its aetiology has not been settled yet, however, computerized tomography scan discloses those of traumatic nature. Although, in many instances, it raises a cosmetic concern to the patient and parents, the condition is not without possible "significant" complications, e.g., haemorrhage, infection, dermatological, and air embolism during operative intervention. We describe the clinical features, imaging and operative findings of a case of Sinus pericranii presented to our department, the reason for the surgical treatment of which was the cosmetic concern of the patient's parents.

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Key words: Sinus pericranii, Trauma-induced vascular anomaly, Spontaneous vascular thrombosis

Sinus pericranii (SP) is a rare abnormal venous communication between extracranial and intracranial dural sinuses, which usually involves the superior sagittal sinus more than the transverse sinus.[1,2] The intracranial pressure (ICP) decides the size of the distension of this periosteum-associated vascular anomaly.[3] It can be either traumatic or congenital in origin; however, the computerized tomography (CT) scan is capable of disclosing the former. It is commoner in males than females; the usual presentation is at the paediatric age group.[2] Although it

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may give rise to symptoms,[4] however, from the cosmetic point of view, it may raise the concern of the patient's parents, or the patient, who may ask for an excision. In other situations, it may undergo spontaneous thrombosis.[1] In order to avoid the possible complications, and for the cosmetic reasons, it is advisable to have it dealt with by a suitable surgical technique.[2] Moreover, follow-up is needed to disclose the recurrence,[2] or the presence of other lesions which are mentioned in the literature although this is unlikely.

CASE REPORT

A 3-year-old boy, from the northwest part of Iraq, has presented to our services with right fronto-temporal varicose-like swelling; the lesion has not caused pain or bleeding. The scalp covering is intact, pink, normothermic, non-pulsatile, soft and compressible; the vascular channel has communicating vessels at the frontal and parietal sides. It is of 1.5 cm in diameter, not tender and there is neither palpable thrill nor audible bruit. The lesion becomes more prominent on crying, on assuming head down position and when pressing on the jugular veins (Figure1). There has been no history of trauma. There is no similar case in the family or the relatives.

The plain skull shows bony thinning at the site of the lesion that coincides with course of the vascular structure, which is along the coronal suture, and at its cephalic extremity where it dips down below the bone and communicates, presumably, with the venous diploes (Figure 2).

The brain spiral 3D - CT scan although it shows normal brain parenchyma, however, there is a locally detached piece of fronto-temporal bone with two bony defects (interruptions) at the cephalic and caudal aspects suggestive, and in favor, of the condition being traumatic in origin, though the event of trauma has been denied, probably missed or passed un-noticed (Figure 3).

The MR angiography shows, at the site of the lesion, more dilated vascular channels joining the superior sagittal sinus than its counterpart on the other side (Figure 4). As the child was unco-operative, no direct contrast injection into the SP was performed to study the direction of blood flow.

At operation a horse shoe flap is raised with its base at the pterion, (Figure 5). The tortuous vein is dissected of the scalp tissue down to where it emerges from underneath the bone and the two draining veins have been interrupted with diathermy; they eventually collapsed. At the site of communication with the bony diploe, the 3-cm-widened major vessel is ligated with 3/0 silk suture and then diathermised flush with the surface of the bone (Figure 5). Neither craniectomy nor craniotomy has been done. A decision is taken not to attempt to pursue the venous channel, or its communication(s), neither under the bone nor to the sagittal sinus. Such a step will be taken in case there is any recurrence in the future. The post-operative recovery has been uneventful; there were no wound complications; the patient was sent home one week following surgery (Figure 6).

As the resected lesion was sent for histopathological examination, the report was read as follow: " macroscopic description: an irregular piece of tissue with a central cavity inside; microscopical examination: sections show vascular lesion comprises large and dilated central vessel containing thrombus with features of recanalization, surrounded by small congested vessels. The appearances are suggestive of arteriovenous connection, could be due to malformation or old trauma".

DISCUSSION

SP is a small circumscribed fluctuating vascular swelling of the scalp that directly communicates with the intracranial venous system.[5] SP was first described by Hecker in 1845 as a soft midline subcutaneous tumour connected to the venous system.[5] However; it was also described in 1850 by Stromeyer.[6]

Newton and Troost described it as a communication between the extracranial and intracranial venous circulation via large, tortuous, thin walled vascular channels.[7] Volkmann described two types of SP: "true sinus" which fills with increased intracranial pressure and disappears completely with compression and "pseudo sinus", which fails to disappear completely with compression. They considered the latter to be angioma or cavernoma of mainly venous component. Three possible theories have been postulated regarding the pathogenesis of this anomaly, i.e., congenital, spontaneous and traumatic. The congenital theory is widely accepted.[8] Heinke stated that vomiting, forceful coughing, respiratory disturbances and other forces might cause tearing of emissary veins at the bone surface eventually resulting into a communicating blood cyst on the skull surface.[9] Cohn stated that the endothelial lining of the wall of the SP differentiates the congenital type from the acquired type.[10] However, in the case we are presenting here there is no sign of raised intracranial pressure and the case is of traumatic origin.

Most cases of SP are located near the midline, the frontal region is most frequently involved followed by the parietal region.[11] A lateral location is unusual.[12] Though Desai et al have reported a frontal SP,[13] however, they stated that frontal SP with an orbital extension had not been reported in the literature. In this case it is found to be at the fronto-temporal region.

Usually the patients are not symptomatic and the problems are cosmetic.[13] Rarely there may be headache, giddiness and vertigo.[13] The primary concern is often cosmetic, but common symptoms include headaches, sensations of pressure or fullness, or local pain.[14] In this case, it was the parents' concern about the cosmetic appearance of their child rather than other symptoms, or other possible complications which were absent. Authors mention that in rare cases, severe symptoms such as bradycardia, bradypnoea,[15] hearing loss,[16] or dermatologic manifestation[3] have been described; these did not occur in this case; the parents denied that their child had any complaint. The varicosities tend to

increase in size slowly, although there has been a report of spontaneous regression. [17]

The diagnosis of SP can be made by the appearance of soft, fluctuant mass located near the intracranial sinus, which usually vary in size with change in intracranial pressure.[13] Digital subtraction angiography (DSA) is diagnostic, as it demonstrates the SP in the venous phase. Carpenter et al stated that DSA had confirmed the transosseous connection with flow of blood from the superior sagittal sinus to the overlying pericranial varicosity and from the varicosity to an adjacent intracranial vein.[1] Magnetic resonance imaging shows a signal void.[11]

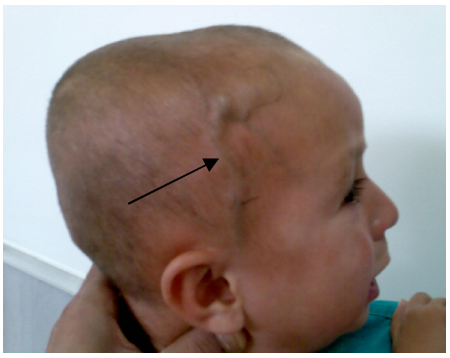
The differential diagnosis includes arteriovenous malformation, epicranial varix, cavernoma, growing skull fracture, eosinophilic granuloma, epidermoid tumour, dermoid, meningocele and encephalocele. Most cases appear to be congenital in nature and are frequently associated with vascular malformation, haemangioma of the cerebellum or retina as a part of the von Hippel-Lindau syndrome, blue naevus syndrome, venous cavernoma of the scalp and haemangioma of the tongue.[18] However, this case, we think it was traumatic in origin as the fracture site was clearly seen in the radiological studies; the diagnosis has been established on clinical, radiological and histopathological grounds. Further follow-up is necessary to exclude the possible development of other lesions mentioned in the literature.

Although spontaneous regression of the SP has been reported,[17] however, most patients require removal of the sinus and blocking the communicating veins[13]; this has been done in this case. In keeping with other author's findings, the occlusion of this abnormal pathway has not been reported to result in intracranial venous hypertension.[8] The possible complications during surgery are haemorrhage and air embolism,[11] did not occur in this case. Moreover, the histopathological findings, in the specimen of this study, of partial (mild) thrombosis, which was not enough to cause the complete obliteration and thrombosis, is, logically, not in contradiction with others' reports of spontaneous thrombosis.

CONCLUSION

SP a rare vascular disorder communicating pericranial with intracranial venous structures. It can be of congenital or traumatic origin, and may have different modes of clinical presentation. Clinical, radiological and operative materials subjected for histopathological examination, all should be able to establish the diagnosis. Since it may carry with it life threatening complication(s) such as haemorrhage, infection and air embolism, therefore a high index of suspicion is always warranted. Surgical excision, with craniotomy, or craniectomy, should provide a definitive treatment choice. Few authors had, also, performed acrylic cranioplasty after obliteration of the vascular channels. However, follow-up is mandatory to exclude recurrence or the association of other lesion(s).

A.



B.

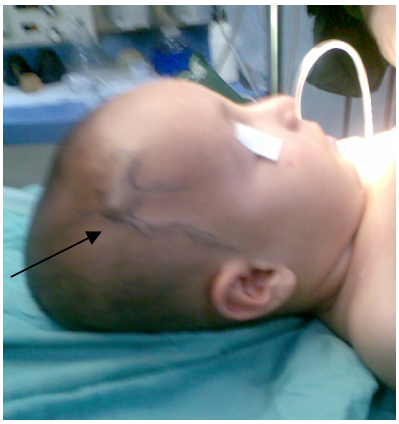
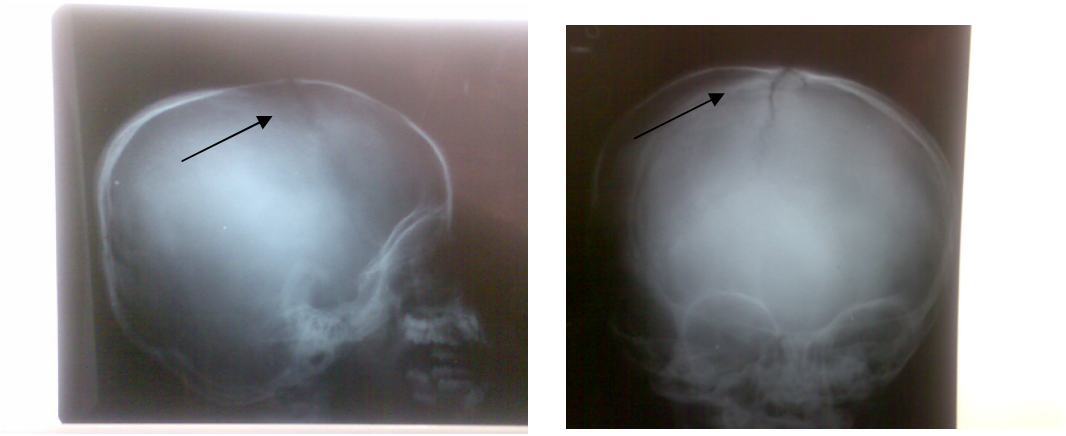
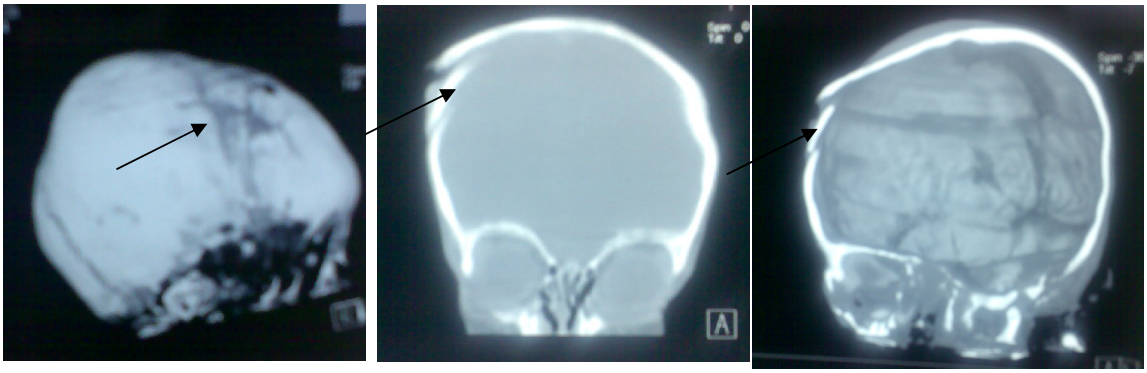


Figure 1. The prominent sinus pericranii, A. on crying and B. while patient is under anaesthesia, marked with indelible ink

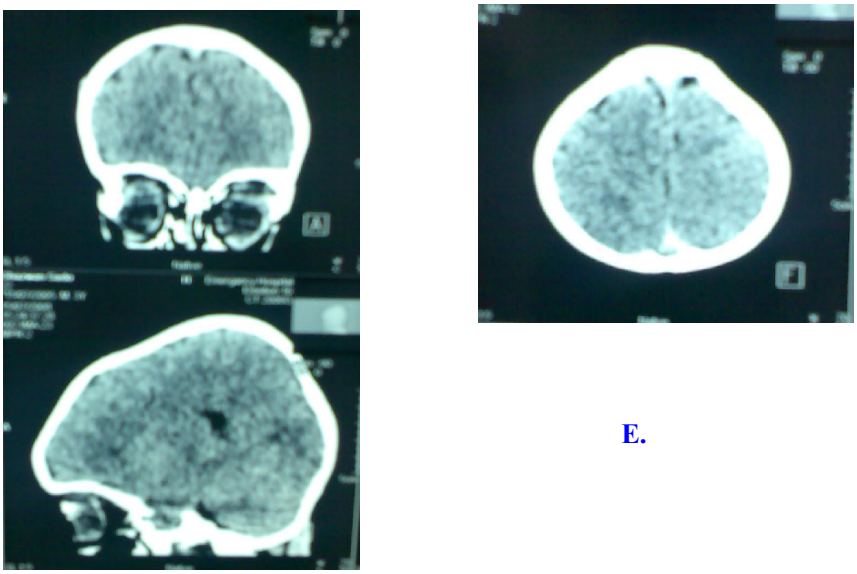


A. **B.**

Figure 2. A. and B. The plain skull X-ray films show diminished bone density at the site of the lesion (please see arrows)

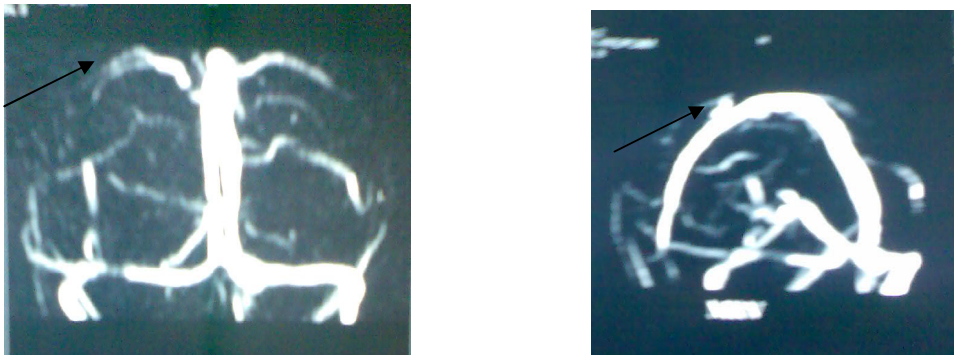


A. B. C.



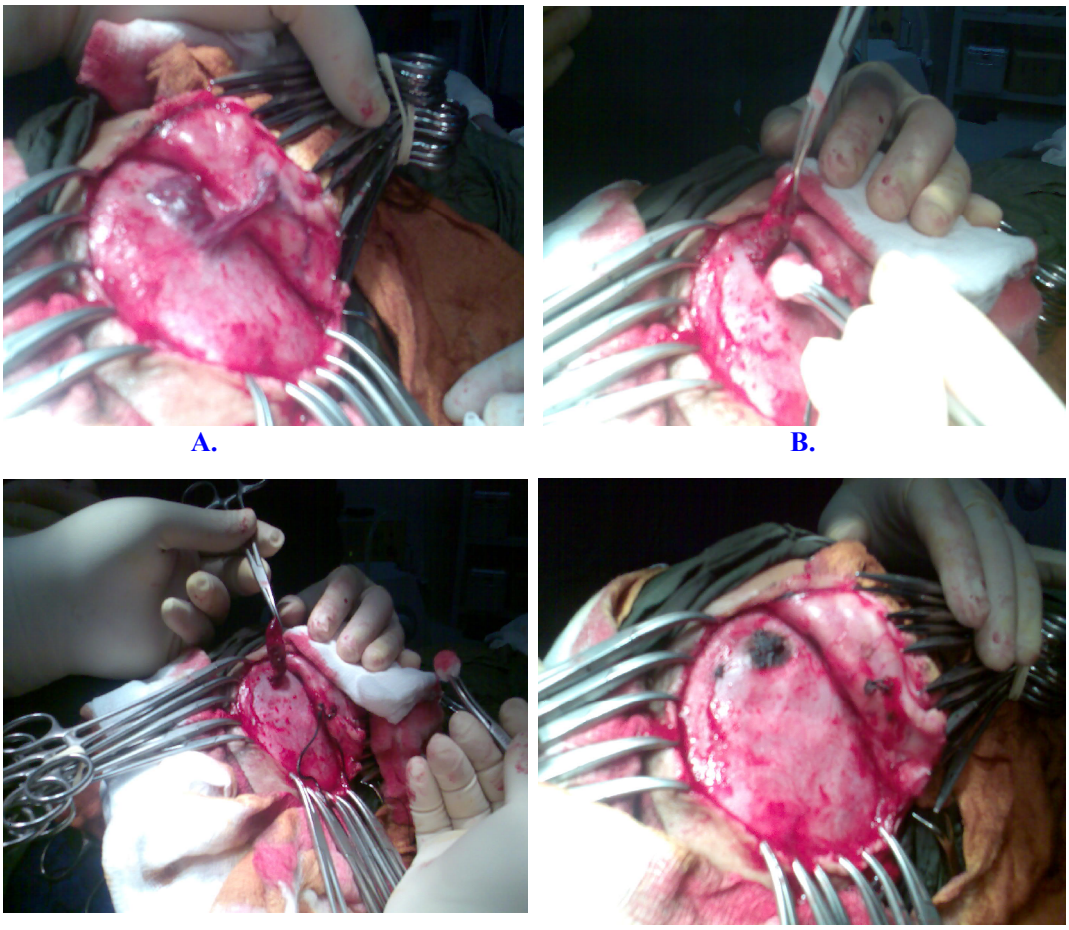
D. E.

Figure 3. The computerized tomography. A, B and C show the bony defect under the vascular confluence of the anomaly, at the site of bone penetration as well as the site of bone fracture (please see arrows). D and E show normal brain parenchyma



A. B.

Figure 4. A and B The magnetic resonance venography showing the dilated vascular channels draining into the superior sagittal sinus (see the arrows)



A. B.

C. D.

Figure 5. A – D. The vascular anomaly as shown at operation and during the steps of dissection and resection. The communicating draining veins have been disconnected

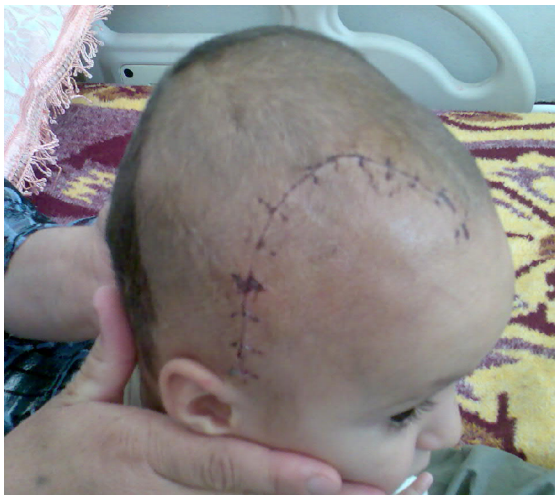


Figure 6. A post-operative picture showing the disappearance of the sinus pericranii

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پوختە

گیرفانێ دەوروبەریێ قافە : شیێواندنا لوولەیا خوینێ لو چوارەنەیێ ئاسایی یە درویدانێدا وجهێ چێبوونێ کێمە

گێرفانێ دەوروبەریێ قافە ، ئەو شیێواندنەکا خوین هێنەریە و نە یێ ئاسایی یە درویدانێدا ساخلەتا وی ئەوە کو دەوالیێن دەوروبەریێ قافێ دگەهینتە گیرفانێ دەیکا دژوار، ئەف دەوالیە پەیوەندیەکا ب وان شانا هەی ئەوێن هەستیێن قافێ چێدکەن و شیانێن پیربونێ هەنە و قەباریێ وی یێ جیاوازە دگەل گوهورینا ئەوژی ب بلند بونا پەستانێ دناف کلوخیدا ، نیشانێن نەخوشیێ ، سەرئێشی و گێژبونێ و هەست ب پیربونێ یان ژانەکا دجهدا یان ئێشا پیستی بخوڤە دگریت ، پێشکەفتنا ئامادەیا وێ دکومەلا ژیێ زاروکینێدایە پتر ژکومەلێن ژی بیێن دی و بیێت نێر پتر پێ توشدبن ژبیێن مێ ، هەرچەندە نەهاتیە زانین کاچەوا چێدبیت ، بەلێ (مفراس) دەزگەها هیێلکاریا میێشکی جهێ کێم بێ چێبوونی دیاردکەت هەرچەندە شەپرزەیي و گرنگیا جوانیێ پەیدادکەت . بەلێ نەیا ڤالایە ژتێکەلبونێ بەری یان ل دەمێ تێکداچونێن نشتەرگەریێ وەك خوین رشتنێ و کولبونێ و گرتنا بای ، ل ڤیرێ ساخلەتا نەخوشیێ و تیشکی و لدەمێ تێکداچونێن نشتەر گەریێ هەیە، ئەگەرێ تێکداچونا نشتەر گەریێ دڤێ رەوشتێدا ئەوژی شەپرزەیا دەیابێن زاروکیە لسەر سەر و سیمایێ جوانیا زاروکي وان.

الخلاصة

الجيب المحيط قحفي : شوه وعائي دموي متعرج غير اعتيادي الحدوث رضحي المنشأ

ان الجيب المحيط قحفي هو شوه وريدي غير اعتيادي الحدوث يتميز باتّصال الدوالي المحيطية بالقحف بجيوب الأم القاسية. و هذه الدوالي ذات علاقة متّصلة بالنسيج المكون للعظم القحفي و هي لها قابلية الامتلاء و يختلف حجمها مع التغيرات بارتفاع الضغط داخل الجمجمة. ان الأعراض السريرية تشمل الصداع و الدوخة و شعور الامتلاء أو الألم الموضعي أو الأذى الجلدي. تقدم حضورها في مجموعة عمر الطفولة أكثر من المجاميع العمرية الأخرى و تصيب الذكور أكثر من الاناث. بالرغم من عدم معرفة كيفية منشاها، الاّ ان مفراس الدماغ يكشف ذات المنشأ الرضحي. بالرغم من انّه يثير القلق و الاهتمام الجمالي، الاّ أنّه لا يخلو من احتمال الاختلاطات قبل و أثناء التداخلات الجراحية كالنزف الدموي و الالتهاب و السد الهوائي. هنا وصف سريري و شعاعي و أثناء التداخل الجراحي. ان سبب التداخل الجراحي في هذه الحالة هو قلق والدي الطفل على المظهر الجمالي لطفلهم.