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CASE REPORT

Editorial:

Mass Casualty Management and Emergency Care System (E M S)

Hikmat A.R.Hatam, *FRCS.*

A characteristic of the EMS is the mechanism by which sudden increasing demands on one local system are met by shifting resources from less involved areas to the scene of the demand. A common term for the process by which resources are temporarily loaned to the system being taxed by emergency demands is "mutual aid ". In this manner, peak emergency care demands are often met by use of shared facilities or temporarily borrowed resources from systems in neighboring areas.

EMS systems are triggered locally several times a year and therefore planning and operations are kept current through repeated use.

The plan for managing victims of disasters should be built around an existing EMS system. Since detection, notification and primary dispatch of rescue teams and on-site early care are all part of the EMS, it is the pre-hospital component of EMS that will commonly provide hospitals with their initial notification and assessment of the scope and nature of the disaster.

Experience in handling large numbers of injured patients is relatively limited only to those who involved in Iraq, Iran conflict and much of the accumulated experience has been military rather than civilian. It is unlikely that we will ever have much carefully controlled data on which to base our management of this type of problem.

Primary care consists mainly of basic life support (BLS) measures together with such advanced life Supports (ALS) measures as may be necessary. (These are usually devoted to airway and ventilation factors, control of hemorrhage, anti-shock treatment and preparation for transportation).

Principles of Disaster Management

Advance Planning

The most important and generally agreed – upon principles that have emerged from the experience of the medical profession in handling disasters is the need for realistic advance planning. In spite of the importance and wide acceptance of this principles, there has been less thoughtful planning for handling mass casualties than there should Shaft an summarizes this well in stating that most description of civilian disasters are concerned with implementation of hospital disaster plans and casualty care after the patient reaches the hospital triage area .

In many cases central medical authority cannot be designated effectively in time for any important decisions to be made. Obviously, criteria for such decision making should have been discussed in planning sessions with representative of all involved personnel (fire and police departments, medical planners and the support and mutual aid agencies commonly utilized, including nearby military recourses).

Disasters may range from episodes of violence in an urban setting, in which scope of the occurrence is relatively easy to define, to the large acts of nature with disruption of communication and

transportation over wide geographical areas.

Disasters may be natural (floods, earth quakes, windstorms, large fires, volcano eruptions) or man – made (transportation explosion, fire, riot and civil unrest, war). There are many proponents of planning and exercises designed to meet the needs of the hospital involved in a disaster. So many organizations would be involved in such an exercise that the undertaking would be difficult and expensive.

It is obviously difficult to develop plans that will be suitable for the limitless type and magnitude of disasters that may occur. Some disasters cause a general disruption in a community and others are localized to a building or two. There are certain features that are sufficiently common to enough different types and sizes of disasters to justify the effort involved in planning. By definition in mass casualty situations the demands always exceed the capacities of the personnel and facilities.

The purpose of advanced planning is therefore to establish a system that will assure the optimal utilization of personnel and facilities for the particular situation.

Casualty predictability

As previously stated, the key to effective handling of disaster situations is realistic advance planning.

Use of effective maneuvers

A third principle is that certain maneuvers that are economical of personnel, facilities and time may produce a decrease in mortality, early morbidity and long-term functional loss. More Sophisticated techniques that require the prolonged services of highly trained individuals using complex equipment and many supplies though extremely valuable in ordinary practice, may not be a wise investment of resources in handling large numbers of injured people in a brief period of time.

Treatment modifications

This principle is that the way in which we handle specific types of injuries in ordinary practice must often be modified when we are dealing with casualties from a disaster this shift in thinking and action is extremely difficult for many physicians to make unaware of the modifications that must be made in a mass casualty situation are likely to continue to utilize conventional techniques in such a situation unless there is forceful direction from those in charge in a mass casualty situation are likely to continue to utilize conventional techniques in such a situation unless there is forceful direction from those in charge.

Teamwork

This brings us to a fourth principle of mass casualty management: teamwork. In ordinary practice each physician is accustomed to working in a more or less independent capacity. The effective management of large numbers of casualties in a short time demands a totally different or organizational structure. There must be someone in charge, in the person of the disaster plan director, who by experience and training is capable of giving orders, and other must be able and willing to have control as close to absolute authority as is seen in medical practice.

Philosophical Approach

Special attention should be given to the readjustment of thinking- literally of philosophy- that is necessary if the best possible results are to be obtained from the medical care of disaster victims. The physician is ordinarily committed to the highest quality of care for his individual patient. When a hospital is flooded with tremendous numbers of seriously injured individuals, an abrupt

modification of this philosophy is essential. For example, certain individuals will arrive at the hospital in such condition that, under the disaster circumstances, there is no hope of salvaging them, though had they arrived in isolated circumstances, aggressive treatment might have permitted their survival. In the disaster situation we have no reasonable choice but to regard these individuals as hopelessly injured and to turn the bulk of our efforts to those less seriously wounded.

Disaster Planning for the Hospital

A key feature of the hospital management of disasters is the provision of separate space for triage, stabilization, major surgery, minor surgery and recovery. Special provision should be made for supplying space for waiting families of disaster victims, for the handling of the dead and for accommodation of representative of communication of representatives of communications media. The integration of these facilities, the provision of adequate resources and staff and mobilization of a disaster plan require finely tuned coordination. Such coordination can be achieved only if the plan is exercised at regular intervals through disaster drills.

Surgery in Most hospitals, the major surgery area will be the main set of operating rooms in disasters. Ample numbers of surgical staff, anesthesia staff and nursing staff must be provided and a plan must be at hand for orderly addition of staff as needed. A minor surgery area (and possibly a special fracture area) should be provided so that patients need not remain for definitive care in the stabilization area and so that patients at the same time will not overload the major surgical area. The minor surgery are must be supervised by an experienced individual who can maintain a steady flow of patients. It is imperative to note that here, as elsewhere in the handling of disasters victims, it may be necessary to compromise the highest quality of care in the name of efficiency.

Recovery Area Plans must provide for the easy evacuation of regular hospital patients from areas normally used for recovery or for intensive care to provide large open areas for recovering disaster victims.

Intensive care unit personnel must constantly be aware of patients who could be moved out if a need should arise suddenly.

It is particularly important that an appropriate individual have the authority to make decisions about patient moving and that a crisis of authority not be allowed to arise that would be superimposed on the crisis imposed by the disaster itself.

Logistics

The key feature in coordination of hospital disaster efforts is successful communication among those responsible for resources. In order to coordinate the various resources and facilities, an information system manned by trained personnel must provide the communication s connection. A single individual should be in charge of coordinating disaster resources and facilities.

The disaster control center should include representative of the medical staff, nursing staff, administration, materials management, security, public affairs and support services. Specific communication support should be provided. The individual in the control center must have the authority to call in staff from outside.

Drills

As indicated earlier the effective coordinating of facilities, resources and manpower requires both planning and practice. It is commonplace that the requisite

disaster drills are given little attention beyond that necessary to comply with external standards. Complex problems that may arise to challenge key coordinating staff in an actual disaster are not covered in many drills.

Triage

The classification of patients into categories is critical in determining the success in handling a disaster. These categories may include patients who need immediate stabilization, those who can proceed to definitive care and those with relatively minor injuries. Physicians performing such triage must be experienced in the care of trauma patients and sensitive to unusual clinical problems. It is imperative that this task not be relegated to junior staff or house officers. The triage area must be capable of expansion to accommodate all patients that may be brought to a given hospital. Since triage is best performed at the entry point to the hospital the emergency department should have been planned to serve this purpose. Ideally, the registration and waiting areas should be capable of conversion to triage.

The details of patient sorting will, of course, depend upon the particular circumstances. Patients arriving at the hospital may be classified into one of four major categories by the triage officer. These are:

I. Patients with minimal injuries who will do well on self-care or "buddy" care. Medicolegal responsibility makes it necessary not only to allow any patient to register if he desires but also to provide "medically trained" personnel to render care. This holds true for the disaster situation and may make the self-care or "buddy" system not feasible and force these patients to be grouped with category II patients

II. Patients whose injuries are less trivial and will require medical attention but are not of a serious nature; these patients will not require intensive care.

III. Patients whose injuries will require major medical attentions. This group may be subdivided into the following.

A. Require early operation

1- Immediate

2- After an interval

B. Do not require operation or operation will be performed only later in their course.

IV. Patients who are either dead on arrival or so hopelessly wounded that under the circumstances of disaster there is no reasonable chance of saving them.

In some disaster situations , the patient flow may be so great that initially triage should be made according to the most basic classifications, i.e., (A) those who will live no matter what, (B) those who will die no matter what and (C) those whose survival depends upon early critical care. It may be necessary to have "tiered triage in which category C patients are subdivided by another team according to whether or not there is need for surgery, and early operation or delayed operation.

In Addition to sorting patients into categories, the triage officer may or may not be assigned two additional responsibilities .The first is the establishment of priorities among Category III patients. In other words, the triage officer may determine which patients most urgently need surgical attention, blood transfusions and other care. The other responsibility sometimes as signed to triage officers is the institution of certain measures of immediate care such as the relief of airway obstruction and the control of hemorrhage. If it is elected to assign to the triage officer the responsibility of priority of determination for Category III patients or the responsibility for execution of some immediate care measures.

Patient Identification and Record-Keeping

System that serves to identify patients in a disaster situation should be different from the hospital routine in several respects. A system such as D1, D2, D3, would identify the disaster victim as being such. Later permanent hospital numbers could be assigned so that disaster numbers could be used again.

Patient Care Categories

Patients in each category should be cared for in a separate location. The segregation of patients on this basis, which in ordinary hospital practice is called progressive patient care, is properly the most efficient means of handling large numbers of causalities in a brief period of time with limited resources.

Category I – Minimal Care. Almost no medical personnel are necessary to handle patients in this category.

Category II- Light Medical Attention. Again , very little medical expertise needs to be expended . The principal duties to be carried out are perhaps the administration of tetanus shots, the application of light dressing and other chores that can safely be performed by medical students.

Category III-Major Medical Attention. It is this category that will utilize most of the personnel, equipment and supplies. The specific organizational structure of Category III care is best determined by the individual hospital on the basis of its particular resources. The designation of a senior person to supervise this large portion of the mass casualty management is probably advisable in most hospital.

Patients who require early operation treatment must, if priority has not already been determined by the triage officer, be sorted with respect to the urgency of operative intervention. The decision regarding the timing of operation will, of course, depend in large measure upon the nature and size of the disaster- several patients with moderately severe head injuries may require decompression quite early. On the other hand, in the event of a major catastrophe with hundreds of soft – tissue injuries to be cared for by a few surgeons, the talents may be much better utilized in the performance of 30 or 40 wound debridements than in the performance of three or four cranial decompressions. It is probably desirable for a relatively high- ranking member of the surgical staff to serve as a deputy disaster plan director in change of Category III patients. His major responsibility is to keep the workload reasonably well distributed among the personnel caring for these patients. Those with the greatest expertise and leadership ability should be utilized to fill the position of disaster plan director.

Category IV- Hopelessly injured a D. O. A. The emotional difficulty – involved in classifying these patients and the importances of assigning some patients who arrive at the hospital alive to this category have already been discussed. Patients in Category IV should be made as comfortable as possible with the facilities at hand. A few nurses equipped with drugs can ordinarily do this.

Conclusion

Optimal medical care in disasters of all sizes and types is dependent upon realistic advance planning by the community and its hospitals. The type of catastrophe that will occur in a particular community cannot be anticipated, but planning can assure that when a disaster occurs, appropriate individuals will be in a position to deal effectively with the specific problems that arise. The fact that planning cannot be complete is no

justification for the absence of preparation. The integration of hospital disaster planning to the regional EMS plan is essential for realistic preparedness in the event of a real disaster.

ONE-YEAR (PATIENT AND RENAL ALLOGRAFT) SURVIVAL FOLLOWING RENAL TRANSPLANTATION

Ausama S. A. Muhsin[1] *FIBMS,* **Usama S. Alnasiri**[1] *FRCS,*
Usama N. Rifat[2] *FRCS, FACS*

Abstract

Background: Renal transplantation offers a realistic therapeutic option to patients with end-stage renal disease (ESRD).

Objective: To evaluate one- year (patient and renal allograft) survival and comparing age and HLA-matching results as possible risk factors.

Methods: Fifty (50) patients underwent renal transplantation in the renal transplantation unit of Surgical Specialties Hospital-Baghdad from September 2000 to October 2002. None had diabetes mellitus or clinical evidence of symptomatic cardiac disease. All the transplanted kidneys were from living donors. Direct matching between the serum of recipient and lymphocytes of the donor was negative. HLA class I matching was performed. Recipients were followed for one year following renal transplantation clinically and by regular laboratory tests. Ultrasound and color Doppler examinations were performed when there was evidence of decreased urinary output, allograft dysfunction, or clinical suspicion of rejection. Graft nephrectomy, when needed, was done in the same center.

Results: Thirty-nine patients (78%) continued their lives one year following renal transplantation while eleven patients (22%) died during the first year following renal transplantation, due to cardiovascular complications and sepsis. Death following renal transplantation was compared with age and HLA-matching as possible risk factors. The comparison was not statistically significant. In thirty-eight patients (76%) the transplanted kidney was functioning normally after one year from renal transplantation. Twelve (12) patients (24%) needed graft nephrectomy on the basis of clinical picture of acute rejection aided by conventional sonographic and color Doppler examinations. Acute rejection was not confirmed by histopathological examination prior to graft nephrectomy.

Conclusions: Cardiovascular disease is common in renal transplant recipients and is a major cause of mortality in this population followed by sepsis. Age of recipient and HLA- matching results were not correlated to the one-year recipient mortality.

Key words: Acute rejection, cardiovascular diseases, one-year survival, renal transplantation.

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Introduction

Renal transplantation can restore patients with end-stage renal disease (ESRD) to nearly normal health. Regardless of whether the treatment modality is dialysis or transplantation, the major causes of death are, in order, heart disease, sepsis, and stroke[1].

It has been known for some time that cardiovascular mortality and morbidity are higher in renal transplantation than in the general population[2]. There is an approximate 10-fold higher incidence of cardiovascular mortality in renal transplant recipients than equivalent patients without renal disease. In contrast, when one considers all patients with ESRD, cardiovascular mortality is lower in transplant recipients than patients on maintenance hemodialysis. Kasiske[3] examined a large cohort of renal transplant

[1]Dept. Surgeury-Section of Urology, College of Medicine, Al-Nahrain University, [2] College of Medicine, Baghdad University Chairman of Iraqi Council for Urology.
Address correspondence to Dr: Ausama S.A. Muhsin, Email: ausamasaadi@yahoo.com
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recipients and found that, in a broad sense, traditional factors such as lipids, HgbA1C, and diabetes mellitus were associated with cardiac morbidity and mortality in a similar quantitative manner as in the general population.

An often-overlooked phenomenon in renal transplant recipients is cardiomyopathy, which in this population is thought to be multifactorial. Once again, the incidence of cardiomyopathy is significantly less in renal transplant recipients (10%) compared with patients on maintenance dialysis. Unfortunately, several commonly used immunosuppressive drugs interfere with the cardiovascular system.

One year graft survival rates are reported to be 80% for mismatched cadaveric renal grafts, 90% for non-identical living related grafts and 95% for human lymphocyte antigen-identical grafts[4]. A variety of medical and surgical catastrophes can occur following renal transplantation which compromise graft outcome. Technical failures, infections, and recurrence of the disease for which the transplant was performed are among the problems occasionally encountered in these patients. However, except for transplants performed between identical twins, transplant rejection continues to be the most important contributor to graft loss.

The aim of the study is to evaluate one- year (patient and renal allograft) survival and comparing age and HLA-matching results as possible risk factors and under the difficult circumstances of sanctions.

Patients & Methods

From September 2000 to October 2002, 50 patients underwent renal transplantation in the renal transplant unit of Surgical Specialties Hospital, Baghdad. The recipients and their potential donors were evaluated prior to transplantation. None was shown to have diabetes mellitus or clinical evidence of symptomatic cardiac disease. All transplanted kidneys were from living donors (LDs).

Recipients and their potential donors were ABO compatible. Direct matching was negative. HLA-matching class I only was performed as class II was not available. Panel reactive antibodies (PRA) test was performed. Recipients with less than 30 per cent reaction were chosen.

The hot ischemia time was ranging between 4-14 minutes. The cold ischemia time was ranging between 60-180 minutes. In (45) patients (90%) arterial anastomosis was to the external iliac artery (according to the surgeon's preference), while in (5) patients (10%) the anastomosis was to the internal iliac artery. The renal vein was anastomosed to the external iliac vein. The arterial anastomosis was done in an interrupted fashion, while the venous one was continuous. Extravesical technique for ureteroneocystostomy was used. Triple immunosuppressive therapy that consisted of cyclosporine, corticosteroids and azathioprine was used. Newer agents were not available.

Data collection

The recipients were followed for one year clinically and biochemically. Renal allograft dysfunction was defined as a persistent/or progressive elevation of serum creatinine. Conventional sonographic and color Doppler examinations were performed when there was clinical evidence of decreased urinary output, and/or laboratory findings of graft dysfunction.

Statistical analysis

Data were tabulated in a mean (±SD), number and percentage. Association between different variables was measured by using Fisher's exact test. P value < 0.05 was considered as statistically significant.

Results

Fifty patients aged (15-62) years; with a mean age (34.46 ± 12.4) years underwent renal transplantation. They were (35) males

(70%) and (15) females (30%). Thirty-nine patients (78%) continued their lives one year following renal transplantation while eleven patients (22%) died during the first year following renal transplantation, due to cardiovascular complications and sepsis. Cardiothoracic complications were responsible for death of (7) patients (63.63%).

Two of them died (they were 46 years and 25years) due to cardiac arrest in the immediate 24-hour period. No autopsy could be performed so the real cause of death could not be verified. Two patients developed acute rejection and after failure of anti-rejection medical therapy, graft nephrectomy was done and they were returned to hemodialysis but later died due to acute pulmonary edema. The remaining three patients died due to respiratory failure secondary to chest infection. Sepsis was responsible for death in (4) patients (36.36% of cases). One developed disseminated pulmonary tuberculosis. The other three had septic shock leading to death.

Table (1) shows the causes of death among recipients of transplanted kidney. Death following renal transplantation was compared with recipients' age and HLA matching as possible risk factors. The comparison was not statistically significant. Table (2) and table (3) show the correlation between death and both HLA matching and recipients' age respectively.

Table 1: Causes of death among recipients of transplanted kidney

Cause of death	**(n=11)**
Cardiopulmonary complication (s)	7 (63.63%)
Sepsis	4 (36.36%)

Table 2: Donor- recipient HLA class I – matching and recipients' death

One-year recipients' fate *	**Less than one haplotype (n=20)**	**One haplotype (n=30)**	**Total (n=50)**
Death	4	7	11 (22%)
Survival	16	23	39 (78%)

*** P value not significant**

Table 3: Age and recipients' death *

Age (years)	Dead (n=11)	Survived (n=39)	Total (n=50)
10-19	1	4	5 (10%)
20-29	2	13	15 (30%)
30-39	0	12	12 (24%)
40-49	6	5	11 (22%)
50-59	1	5	6 (12%)
60-69	1	0	1 (2%)

Thirty-three patients (68.75%) developed renal allograft dysfunction, which ranged from mild reversible dysfunction to severe deterioration that necessitated graft nephrectomy. After one year from renal transplantation the transplanted kidney was functioning normally in thirty-eight patients (76%) while twelve (12) patients (24%) needed graft nephrectomy on the basis of clinical picture of acute rejection aided by laboratory, conventional sonographic and color Doppler examinations. The diagnosis of acute rejection was confirmed by biopsy in two patients. Figure (1) illustrates the monthly percentage of deaths and renal allograft nephrectomy during the first year following renal transplantation.

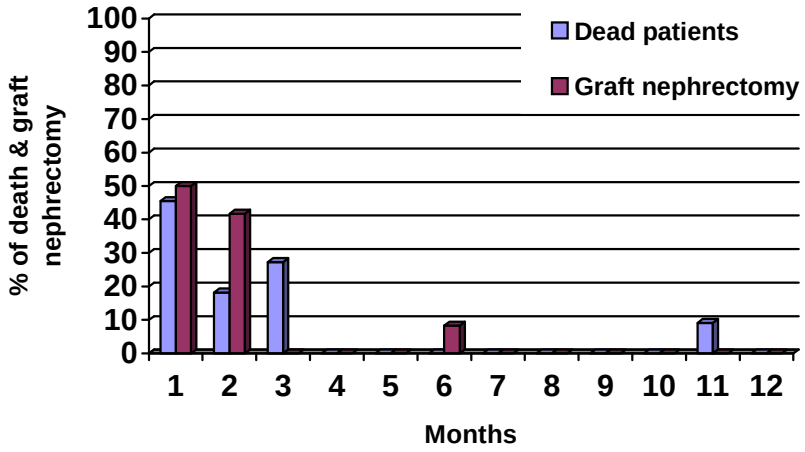

Figure 1: Monthly percentage of recipients' death and renal allograft nephrectomy

Discussion

Transplantation has revolutionized treatment of end- stage renal disease (ESRD) by proving more cost effective than hemodialysis, with a lower morbidity and improved quality of life. Both patient mortality and graft loss were excessive prior to 1970, reflecting the limitations of immunosuppressive therapy available at the time. As immunosuppressive therapy was

refined, patient survival improved. This was due to a decrease in the frequency of life-threatening infections. Currently, a 6-month patient survival of 95% is achievable at most centers, despite the fact that criteria for recipient selection have been liberalized to include older individuals and patients with systemic illnesses such as diabetes mellitus[5]. Other Registries now report 2-year patient survival exceeding 90% for HLA identical matches, and 85-90% for cadaveric and living-related non-HLA identical transplants[6-8].

Since cardiovascular disease (CVD) is the main cause of death in renal transplant recipients, optimal control of cardiovascular risk factors is essential in the long-term management of these patients[9]. Evidence is very suggestive that pre-transplant screening for CVD, treatment of hypertension, the use of low-dose aspirin, and smoking cessation will also help to reduce the incidence of post-transplant CVD. Less compelling are data suggesting that intensive glucose control in diabetics will safely decrease the incidence of CVD. Although there is little evidence that treatment of erythrocytosis will reduce CVD, hematocrits above 55% to 60% should probably be treated to prevent venous thrombosis. Vitamins for reducing homocysteine, antioxidant vitamins, and prophylaxis for potentially atherogenic infections are therapies that warrant additional study[3].

An attempt was made to evaluate the effectiveness of the clinical history and current screening techniques available in predicting post-transplant coronary artery disease and also to assess the role of coronary angiography as a pre-transplant screening technique. The conclusion was that clinical history and electrocardiogram (ECG) results are good, practical and low-cost screening methods, and that exercise stress testing and echocardiography were found to be of limited value. Coronary angiography is appropriate in certain high-risk groups but not necessary as part of screening in all potential renal transplant recipients[10].

The first renal transplantation in Iraq was performed in 1973. Renal transplantation surgery started in the Medical City in 1985. Several social and ethical issues of such surgical procedure were encountered.

In this study thirty-nine patients (78%) continued their lives one year following renal transplantation while eleven patients (22%) died during the first year following renal transplantation, due to cardiovascular complications and sepsis. Although recipients did not have symptoms or otherwise clear clinical evidence of diabetes mellitus or active cardiac disease, two of them died due to cardiac arrest in the immediate 24-hour period raising a question of anesthetic protocol during surgery or whether any further preoperative work up was needed. In our study Age of recipient and HLA-matching results were not proved to be correlated to the one-year recipient mortality.

Cardiovascular disease is common among renal transplant recipients and is a major cause of mortality in this population. Calcineurin inhibitors such as cyclosporin, although minimizing early acute rejection, are responsible for considerable nephrotoxicity, leading to progressive renal dysfunction and graft loss. The recent introduction of non-nephrotoxic immunosuppressants offers the possibility of improved renal function post-transplantation.

After one year from renal transplantation the transplanted kidney was functioning normally in thirty-eight patients (76%) while twelve (12) patients (24%) needed graft nephrectomy on the basis of clinical picture of acute rejection aided by laboratory, conventional sonographic and color Doppler examinations. The diagnosis of acute rejection was confirmed by biopsy in two patients.

The indications for allograft nephrectomy are to remove a symptomatic irreversibly rejected kidney and, in the case of a chronically rejected asymptomatic graft, to withdraw immunosuppression and to prevent the development of anti-HLA antibodies that could delay or prevent a subsequent transplantation[1].

In a previous study (42%) of non-functioning renal transplants required removal at some time. Graft failure due to acute or early acute rejection invariably necessitated removal. The recommendation was that transplant nephrectomy is reserved for the symptomatic cases[11].

Our figures of one-year patient and graft survival are less than the standard international figures. This study was undertaken while the country was under sanctions[12, 13]. We were short of many appliances, anesthetic drugs, antibiotics, anti sera, immunosuppressive drugs and kidney perfusion solutions. HLA class II was and still is not available. It was a hard decision to continue working with limited success or to give up. We hope that with a better supply of required drugs and equipment the results will pick up.

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BACTERIAL INFECTIONS IN NEONATAL UNIT IN TRIPOLI MEDICAL CENTER, LIBYA

Jawad K. Al-Diwan[1] *MSc*, **Tariq S. Al-Hadithi**[2] *PhD*
Abdul Latif Shaban[1] *CABP*, **Mohammed Dekna**[1] *PhD.*

Abstract

Background: Infection is a frequent and important cause of morbidity and mortality in the neonatal period.

Objective: This work was carried out to investigate the prevalence of bacterial infection and the frequency of different pathogens among newborns admitted to the Neonatal Intensive Care Unit (NICU) at Tripoli Medical Center (TMC), Libya.

Methods: The case records of all neonates admitted to the NICU of TMC, Libya for the period Sept. 1996 through August 1997, inclusive, were reviewed. Blood and/or CSF cultures were used to establish the diagnosis of bacterial infection. The admissions were categorized as sterile and unsterile.

Results: A total of 1123 newborns were admitted to NICU over the period of the study, 129 (11.5%) of them were proved to be bacterially infected, 10.6% and 24% of the sterile and unsterile admissions, respectively, had bacterial infection. Blood culture was positive in 115 (10.2%) of the admitted newborns, while CSF culture was positive in 24 (2.1%) of them. Gram-negative bacteria were the predominantly isolated bacteria. Serratia spp. was isolated from 38.3% and 50% of blood and CSF cultures, respectively. Klebsilla pneumoniae was isolated from about 25% of both blood and CSF cultures. Coagulase negative staphylococcus (CONS) was isolated from 11.3% of blood cultures.

Conclusion: It can be concluded from this study that neonatal infection is still a problem facing the country and there is a need for study of bacterial colonization of anogenital tract of Libyan pregnant women and its relation to neonatal infections .

Key words: neonatal infection, gram-negative bacteria, Libya

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Introduction

Infection is a frequent and important cause of morbidity and mortality in the neonatal period. Infections affect neonates either through transplacental haematogenous vertical transmission or exposure to infectious diseases in the community[1, 2]. The frequency of different pathogens varies between geographical areas and should be defined in each setting[3-5].

This work was carried out to investigate the prevalence of bacterial infection and the frequency of different Pathogens among newborns admitted to the Neonatal Intensive Care Unit (NICU) at Tripoli Medical Center (TMC), Libya.

Materials and Methods

The case records of all neonates admitted to the NICU of TMC, Libya for the period Sept. 1996 through August 1997, inclusive, were reviewed. Data regarding date of admission, gestational age, birth weight and laboratory results were collected. Blood and/or CSF cultures were used to establish the diagnosis of bacterial infection.

The admissions were categorized as sterile and unsterile. The sterile category refers to neonates who delivered at TMC and admitted to the NICU, while unsterile category includes neonates who were

[1]**Dept Paediatrics, College of Medicine, Al-Fateh University, Libya** [2]**Dept. Community Medicine, College of Medicine, Baghdad University, Iraq.**
Address correspondence to Professor Tariq Al-Hadithi, e-mail: alhadithit47@yahoo.com
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delivered at home or others hospitals and then admitted to the NICU.

Data analysis was carried out using scientific package for social sciences program (SPSS) for windows version 11. Chi-square was used for comparison of prevalence rates. P value less than 0.05 was considered as statistically significant.

Results

A total of 1123 newborns were admitted to NICU over the period of the study, 129 (11.5%) of them were proved to be bacterially infected, 10.6% and 24% of the sterile and unsterile admissions, respectively, had culture proven bacterial infection. The difference between the two rates is statistically significant ($p < 0.05$) (Table 1).

Table 1 : Prevalence rates of neonatal bacterial infection

Type of admission	Total Number	Infected newborns*	
		No.	%
Sterile	1048	111	10.6.2006
Un Sterile	75	18	24.0
Total	1123	129	11.5

* Blood and / or CSF culture positive ($X^2 = 18.3$, d.f. = 1, $p < 0.05$)

Blood culture was positive in 115 (10.2%) of the admitted newborns, while CSF culture was positive in 24 (2.1%) of them. Prematurity (gestational age less than 37 weeks) was reported in 49.3% of newborns, while low birth weight (LBW) was reported in 43% of newborns. Microorganisms isolated from bacterially infected newborns are shown in Table 2.

Table 2: Microorganisms isolated from blood and CSF cultures

Blood culture			**CSF culture**		
Microorganism	**No.**	**%**	**Microorganism**	**No.**	%
Serratia species	44	38.3	Serratia species	12	50.0
Klebsilla pneumoniae	28	24.3	Klebsilla pneumoniae	6	25.0
Enterobacter species	15	13.0	E. Coli	2	8.3
Coagulase negative staphylococcus (CONS) Staph. Epidermidis	13	11.3	Acinetobacter species	2	8.3
			Others	2	8.3
Others	15	13.0			
Total	115	89.2	**Total**	24	18.6

Gram- negative bacteria were the predominantly isolated bacteria. Serratia spp. was isolated from 38.3% and 50% of blood and CSF cultures, respectively. Klebsilla pneumoniae was isolated from about 25% of both blood and CSF cultures. Coagulase negative staphylococcus (CONS) was isolated from 11.3% of blood cultures. Figure 1 showed the monthly variations of prevalence of bacterially infection among sterile and unsterile admissions to the NICU. Neonatal infection shows an increase in the prevalence with time in both admissions.

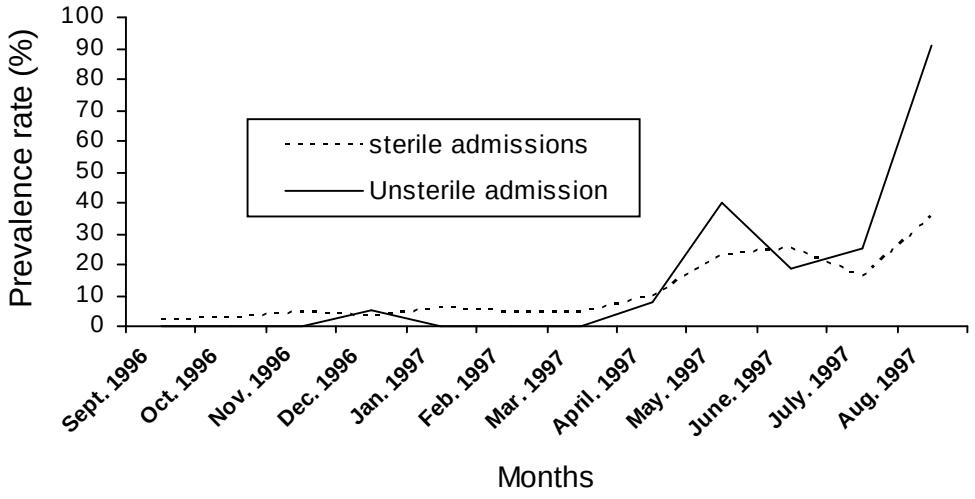

Figure 1: Monthly variations of prevalence of bacterial infections among sterile and un sterile admissions to NICU in TMC

Discussion

The prevalence of neonatal sepsis varies with considerable fluctuation overtime and geographical location, and even from hospital to hospital. These variations may be related to rates of prematurely, low birth weight (LBW) [6, 7], prenatal care [8], conduct of labor [9], and environmental conditions [10].

This study revealed that 11.5% of neonates admitted to the NICU had culture proven bacterial sepsis of blood and / or CSF culture; 10.2% had bacteraemia only. Neonatal bacteraemia is estimated to occur in 1-8 infant per 1000 live births in developed countries [11]. In developing world neonatal sepsis is a greater problem, a rate of 5-10% was reported in Malaysia [12] and a rate of 6% of neonatal septicaemia was reported in Saudi Arabia[13]. The relatively high prevalence rate of neonatal sepsis revealed by this study may be attributed to the finding of high prevalence of prematurity and LBW which in turn could be due to the admission policy in TMC, as it was a common practice to admit premature and LBW neonates to the NICU. The risk of infection is inversely related to gestational age and birth weight [6,7].

The finding that a significantly higher percent of unsterile admission than the sterile admission could be due to the fact that unsterile deliveries, whether home deliveries which are largely in hands of untrained birth attendants or hospital deliveries which are mostly in hands of nurses or midwives in the district and subdistrict hospitals; these deliveries presumably conducted in poor hygienic practices with increased risk of neonatal infection during delivery or thereafter. Nosocomial infection may account for large proportion of both forms of neonatal infections [7]. Abdul Latif[14], In Iraq reported a significant association between neonatal infection and type of delivery place and birth attendant. A similar finding was reported from India [15] and Bangladesh [16].

Several investigators reported variations in the frequency of different pathogens between geographical areas; the bacterial pathogens affecting infant tend to be those, which colonize the anogenital tract of the mother. In Western and developed countries, group B streptococci (GBS) has emerged as the leading cause of neonatal sepsis [3,17,18]. The picture of neonatal

infection in the developing world is quite different, gram- negative organisms still predominate, as revealed by this study with insignificance of GBS as a pathogen. This is the picture in India [19,20], Pakistan [21]. Sri Lanka [22], Bangladesh [16] and Jordan [23]. In Saudi Arabia, also, many workers reported a rate of GBS neonatal infection, although some of them found a high rate of colonization of anogenital tracts of pregnant women with GBS [13,24–27]. Coagulase negative staphylococcus (CONS) which was isolated from 11.3% of blood culture in this study, and staphylococcus aureus seem to emerge as important pathogens as these developing countries implement modern neonatal practices [13,20,25].

The increase in the prevalence rate of infection among neonates with time may reflect just a simple increase in admission rate due to increased referral to this specialized center, a relative deterioration in the health services with time in the NICU after its recent establishment in 1996, with subsequent increase in nosocomial infections.

It can be concluded from this study that neonatal infection is still a problem facing the country and there is a need for study of bacterial colonization of anogenital tract of Libyan pregnant women and its relation to neonatal infections.

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ELECTROCARDIOGRAPHIC STUDY ON THE SIGNIFICANCE OF CHEST PAIN IN PATIENTS WITH ACUTE ASTHMATIC ATTACK

ZAIDAN K. AL-HERGANI *FRCPI, FRCPG.*

Abstract

Background: Patients with acute asthma are usually presented with dyspnoea, wheezing and cough, but some are presented with chest pain, which is usually overlooked. The pain may be part of the clinical features or due to associated ischaemic heart disease.

Objective: To assess the origin of chest pain in acute asthmatic patients.

Methods: Tow hundred patients with acute asthmatic attacks were studied for their symptoms and those with chest pain were especially selected and studied by ECG with other investigations. ECG was done on admission and repeated 48 hours later.

Results: Thirty cases out of the total 200 with acute asthma were found to have chest pain [15%] as alone or part of the clinical features. The cases with chest pain were commoner in patients older Than 50 years [80%]. ST depression and T wave inversion were the most common abnormalities to be found in cases with chest pain [67%]. After 48 hours some of the ECG changes return back to normal and the remaining cases with ECG changes were [40%] which was considered as a substantial ischaemia.

Conclusion: It appears that chest pain occurring in some of the acute asthmatic cases may be due to ischaemia rather than only as a apart of the clinical presentation and it is recommended to be investigated by repeated ECG in all cases.

Key words: Asthma, Chest pain ,Ischaemic heart disease

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Introduction

Bronchial asthma is presented usually with various clinical features including dyspnoea, wheezing, chest tightness, and cough and diagnosis can often made quickly and accurately from the patient description and complaints, but sometimes difficulty arises to differentiate between pain and tightness. Peak expiratory flow rate measurement may be needed with other investigations including eosinophils count, chest x-ray, electrocardiography [ECG], and specific mediators to confirm the diagnosis and complications[1].

Doctors cared for the management of acute asthmatic patients have often faced Patients who complain of chest or epigastric pain alone or with the association of other

Symptoms, which occur during or after cessation of acute attack[2].

The chest pain in acute asthmatic attack is usually overlooked, because the severity of other symptoms masks it[3], however chest pain as the presenting symptom is seldom noted. Non-cardiac pain is a common clinical problem in patients with various respiratory diseases, but some of these pains could be cardiac rather than respiratory in origin[4].

In this study of patients with acute asthmatic attacks, a repeated ECG with other investigations have been performed in order to know the significance of chest pain associated with, and whether it is part of the clinical feature or an ischaemic anginal pain.

Dept. Medicine, Al-kindy Medical College, Baghdad University
Email: zalhergani@yahoo.com
Received 25th December 2005: Accepted 10th May 2006.

Patients and Methods

Tow hundred cases with acute asthma were studied at casualty and as inpatients regarding various symptoms analysis during the period between 1998-2005 at Al-Karama Teaching Hospital, and Al-Kindy Teaching Hospital in Baghdad city. They were 110 female and 90 male patients. Their age ranges between 15-70 year.

All patients had an ECG examination on admission and another ECG after 48 hours. Chest pain, dyspnoea, and Tightness was recorded in studied patients with other symptoms of asthma. The chest pain varies in duration between few minutes to many hours and was acute and not recorded by the patients previously.

Results

Of the total 200 cases with acute asthma, only 30 cases found to have chest pain [15%] while most of them [85%] had other symptoms but without chest pain. Wheezing and tight chest were the most predominant symptoms as shown in table 1.

Table 1: Clinical presentation of acute asthma causes (200cases)

Symptom	No. /cases	Percentage
Wheezing	190	95
Tightness	190	95
Cough	120	60
Dyspneoa	60	30
Chest pain	30	15

There was no difference in the clinical presentation between male and female patients, but the cases with chest pain were seen in patients older than 504 year [80%] compared to younger age group [20%] as seen in table 2.

Table 2: Age related chest pain in asthmatic patients

Age in years	No./chest pain cases	Percentage
> 50	24	80
<50	6	20
Total	30 cases	100

The site of pain was retrosternal in 50% of cases while less frequent in other chest areas as seen in table 3.

Interpretation of ECG changes revealed that T wave inversion and ST segment depression were the commonest findings and was recorded in 12 cases (40%). Only one patient with chest pain found to have acute inferolateral myocardial infarction. Four cases with ventricular ectopic beats were found (13%) which were unrelated to the T wave and ST segment Changes. The T wave inversion and ST segment depression were in 20 cases of the total number of 30 cases with chest pain (67%). The ECG findings are shown in table 4.

ECG was repeated after 48 hours of the acute attack and it was found that 7 out of 12 T wave inversion and 5 out of 8 ST segment depression returned back to normal which means about 60% of the abnormalities were disappeared. There was no significant difference in ECG changes

between male and female patients with acute asthmatic attacks and chest pain.

Table 3: The site of chest pain in acute asthmatic patients

Site	No./ cases	Percentage
Retrosternal	15	50
Left sided	8	27
Epigastric	4	13
Right sided	3	10
Total	**30 cases**	**100**

Table 4: ECC changes in asthmatics with chest pain (30 cases)

ECG changes	No. of cases	Percentage
T inversion	12	40
ST depression	8	27
Ventricular ectopic beats	4	13
Myocardial infarction	1	3
Normal ECG	5	17
Total	**30 cases**	**100**

Discussion

Asthma is an extremely common disorder and though most common before the age of 25 years, it may develop at any time throughout life. The worldwide prevalence of asthma has increased more than 40% since the late 1970. It is among the most common reason to seek medical treatment[5].

The symptoms of asthma consist of a triad of dyspnoea, cough and wheezing, the last often being regarded as the sine qua non. In its most typical form all these symptoms coexist[6].

Asthma is not a uniform disease but rather abroad spectrum dynamic clinical syndrome and the variable nature of symptoms is a characteristic feature[7]. In this study acute asthmatic patients with chest pain were selected and studied for the significance of their pain. The chest pain was in 50% retrosternal. The other 50% recorded sites of pain, were left sided, epigastic and right sided. ECG on admission revealed that 67% of abnormalities included T and ST segment but the repeat ECG after 48 hours showed only 15% of them persist and considered. Unfortunately it was not possible to compare our ECG changes to previous patients ECG because they were unavailable.

As a substantial ischaemia which were more evident in elder population. A study was done by Karwat k. in 2002 on asthmatic patients with and without chest pain showed 18.9% of patients had ST-T changes[8]. In comparison to this study which showed only 10% ST-T changes in all patients with and without pain and which rises up to 67% in those specifically with chest pain.

Out of the 30 cases with chest pain in this study, one was found to have myocardial infarction, which was confirmed, by ECG and cardiac enzymes. A similar report by Rubinsztajn et al was published on a 39 year old woman without any previous history of heart disease[9].

Various studies regarding the cause of death in asthma agreed that the top causes of death were acute myocardial infarction, ischaemic heart disease and heart failure and this is explained by hypoxia and the adverse effect of beta-agonist drugs with tachycardia and hypokalaemia[10, 11].

It is also reported that the use of inhaled beta-agonists were associated with a tow fold increased risk of primary cardiac arrest among patients with asthma especially when inhaled steroids were not used[12]. In various studies of ECG in acute asthma, many of the ECG changes have been observed to disappear within hours after initiation of effective asthma therapy, but return of ECG to normal may be delayed for up to 9 days[13, 14].

The effect of asthma on cardiovascular system has been appreciated for decades. During normal inspiration there is an increased venous return to the right heart as intrathoracic pressure becomes more negative[15-17]. In contrast maneuvers that increase intrathoracic pressure such as Valsalva may decrease venous return that transiently decrease cardiac output and systemic blood pressure and during acute asthmatic exacerbation the interrelation between ventilation and cardiovascular function becomes much more complex with flattening of the interventricular septum interfering with ventricular systolic function[18-20].

There are several factors that predispose to myocardial damage including hypoxia,vasospasm related to mediators release and electrolytes disturbances and dysarrhythmia associated with medications used to treat asthma[21,22].

Hypovolaemia may be a complication of asthma reflecting increased insensible fluid losses from excessive sweating or hyperventilation with decreased fluid intake in severely dyspnoec patient and the patient may become hypotensive during acute exacerbation[23]. It is postulated that dyspnoea associated with severe asthma may mask the pain of myocardial ischaemia. Other complications associated with acute asthma including metabolic acidosis, hypoxaemia, vasospasm which may lead to myocardial contraction band-necrosis,circadian fluctuation in epinephrine and cortisol level ,pulmonary hypertention, increased intrathoracic pressure and left ventricular afterload which may lead to pulmonary oedema[24].

It is concluded from all above that chest pain associated with acute asthma attacks may signify underlying ischaemic episode which is usually missed and it is recommended to consider chest pain in acute asthmatic attack as an important association and to be investigated carefully to avoid cardiac complications in addition to the complications of underlying acute asthmatic episode.

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PLEURAL EFFUSION, ADENOSINE DEAMINASE (ADA) AND LACTATE DEHYDROGENASE (LDH) ENZYMES LEVEL, CORRELATED WITH CYTOLOGICAL EVALUATION.

F.A. Al-Rawi[1] *FICPath,* **N.J. Metib**[1] *MBChB,* **Z. Talib**[2] *PhD.*

Abstract:

Background: Measurement of pleural fluid adenosine deaminase (ADA) and Lactate dehydrogenase (LDH) enzymes activity has gained increasing popularity as a diagnostic test for tuberculous and non-tuberculous pleuritis, especially in countries where the prevalence of TB is high. It carries a high sensitivity, inexpensive and easy.

Objective: To demonstrate the diagnostic value of increased level of ADA and LDH in pleural effusion correlated with the cytological, biochemical and bacteriological assessment.

Methods: seventy-five patients presented with pleural effusions were studied (53 males and 22 females) their mean age was 43.8 years. In all cases after the clinical assessment, evaluation of the pleural fluid was done and this included cytological exam with biochemical tests (adenosine deaminase "ADA" enzyme, lactate dehydrogenase "LDH", protein and glucose level) and bacteriological tests (Gram stain, and Ziehl-Neelsen stain).

Results: From the clinical data and lab tests, patients were divided into six groups according to the etiology of pleural effusion. Most (32 patients) were tuberculous, malignant effusion13 patients, infection 10 cases, heart failure 8 cases, idiopathic effusion 6 cases and miscellaneous 6 cases. Significant difference was found in ADA level in different effusions ($P<0.005$). Highest value of ADA was in TB effusions (the mean was 76.6 u/l), compared to malignant effusions (the mean was 32.4 u/l) and less values in other effusions. LDH highest value was in malignant and TB effusions (mean 321.1 and 314u/l respectively).

Conclusion: Increased ADA levels in TB effusions can be used to differentiate tuberculous from non-tuberculous effusions. And high LDH levels were useful in confirmation malignant effusions.

Keywords: ADA, Pleural effusion, TB.

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Introduction

Measurement of pleural fluid ADA activity has gained increasing popularity as a diagnostic test for tuberculous pleuritis since 1978, especially in countries where the prevalence of TB is high. It carries a high sensitivity (90-100%), inexpensive and easy to measure[1, 2]. ADA is an enzyme of purine catabolism, which catalyzes deoxyadenosine and adenosine to deoxyinosine and inosine and ammonia. High level of ADA is available in activated CD4+ T-lymphocytes, therefore ADA considered as a marker of cell mediated Immunity and play a role in maturation of monocytes to macrophages [3-5].

It has been reported that TB pleural effusion has significantly higher ADA level than other non-tuberculous effusion, and in the latter is seldom exceeded the diagnostic cut off for TB effusion [6-8]. Moreover no significant correlation between activities of ADA in pleural fluid and serum was observed.[9]. This indicates that ADA is being locally synthesized by cells within the pleural cavity in these diseases (local cell mediated immune response) [9, 10] ADA expresses the sum of two isoenzymes ADA1 and ADA2. ADA1 is ubiquitous in all cells including lymphocytes and monocytes,

[1]Dept. Pathology [2]Research Institute, College of Medicine, Al-Nahrain University.
Address correspondence to Professor Dr. FA Al-Rawi, E-mail: faizarawi@yahoo.com.
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where as ADA2 is found mainly in monocytes[11-13].

ADA in TB pleuritis increases at the expense of ADA2 because it produces by monocytes and that the ADA1/ADA total activity ratio improves performance in terms of sensitivity, specificity, and accuracy. But this procedure is highly elaborate [3-5, 11-13]. Studies on this enzyme show wide range of cut-off values (from 25u/l to 70u/l) [3-5,8,9].

Two possible causes in the variation of cut-off values were suggested. The first is related to the method of ADA activity estimation, which is either colorimetric or spectrophotometric method [8]. The second source of discrepancy is related to the characteristics of the population studied in each case, considering areas with a high incidence of both HIV and TB infection.. Further studies show that ADA is independent of HIV serology [1,2,6,7].

ADA level of more than 33u/l considered diagnostic for TB effusion, the sensitivity raised to 100%, the specificity to 95%, and the accuracy to 96% [9]. Others reported that ADA above 70u/l is highly suggestive of tuberculous effusion, whereas level below 40u/l rules out this diagnosis [3-7]. Cytology is an important test for diagnosing malignant cells in pleural effusion with overall accuracy 50-90%, increases by submission of a second specimen and or combined cytology and pleural biopsy [14,15]. Acid fast smears are positive in less than 20% of tuberculous effusions and cultures are positive in 67%, but culture combined with histological examination establish the diagnosis in about 95% of tuberculous pleuritis [6,16]. The aim of this study is to demonstrate the diagnostic value of increased level of ADA in the tuberculous effusion with the application of cytological, biochemical and bacteriological tests. . Ideally the workup of a pleural effusion begins with classification of fluid into either transudate or exudates according to Light et al criteria (1972) [17].

Patients & Methods

This prospective study was carried out during the period from December 2003 to June 2004 in Dept of Pathology and Medical Research Center in College of Medicine Al-Nahrin University, and Al-Kadhemia Teaching Hospital in Baghdad-Iraq. Seventy-five patients with pleural effusion (53 males and 22 females) their age ranged from 6-79 years (mean=43.8 years) were enrolled in this study. Detailed clinical history, physical examination was done.

Pleural fluid specimens were aspirated and submitted for cytological, bacteriological (direct smears and culture) and biochemical exam. Five smears for each case were prepared from the sediment, 3 smears were fixed in 95% alcohol for 20 minutes and stained with H&E for cytological exam and two air dried smears one for gram stain and the second for Ziehl-Neelsen stain. The supernatant of pleural fluid were submitted for biochemical tests (ADA enzyme level measured by colorimetric method (Galanti and Guisti method) [17] and the cutoff value used in this study was 33u/l, LDH activity was measured according to Wroblewski and Ladue method [18] total protein was determined by Biuret method [18] and Glucose was measured by enzyme colorimetric method [18].

Total and differential cell count of pleural fluid was done by dilution of 0.4ml of fluid with 0.4ml of glacial acetic acid using counting chamber for calculation and differentiation. The results were analyzed by appropriate computer soft ware program (SPSS 10.0).

Results

From the clinical data, and lab tests, patients were divided into six groups according to the etiology of pleural effusion. Tuberculous (TB) effusion 32 cases, malignant effusion13 cases, infection 10 cases, heart failure 8 cases, idiopathic effusion (no specific etiology demonstrated) 6 cases and miscellaneous (include uremia, connective tissue disorders, and other rare causes of pleural effusion) 6 cases. All TB effusions, malignant effusions and infection cases were exudates. (Table 1).

Table 1: Levels of Different Parameters in Transudates and Exudates.

Effusion type	ADA U/L mean±SD	LDH U/L mean±SD	Protein gm/L mean±SD	Glucose mol/L mean±SD
Transudate (n =20)	11.9±9.2	174.6±23.9	21.2±7.5	5.3±1.7
Exudate (n = 55)	55.4±45.9	301.5±70.6	43.8±9.6	2.1±1.1

TB effusions (n=32); Form 43% of the cases of idiopathic pleural effusion. Twenty three cases were left sided effusions, and 9 were right sided. The mean ADA value was 76.7u/l, in 30 cases (93.7%) exceeded the cutoff value (33u/l) and only 2 cases (6.3%) were below the cutoff value. Ziehl Neelsen stain was positive in two smears (6.3%). Cytological smears and cell count revealed moderate-severe chronic inflammatory reaction with paucity of mesothelial cells. LDH, mean value was 314.2u/l. (Table-2).

Table 2: Mean Age of Patients and Levels of Different Parameters in the Pleural Fluid of the studied groups

Diagnosis (Cause of pleural effusion)	Age Years	ADA U/L mean±SD	LDH U/L mean±SD	Protein gm/L mean±SD	Glucose mmol/L mean±SD	Cell count/ccm
Idiopathic* (N=6)	57.2	14.9±10.6	177.2±16.2	24.0±7.7	4.5±2.0	816.7±1075.5
Infection** (N=10)	36.7	21.1±14.3 2 cases > 33U/L 8 cases < 33U/L	279.8±42.4	38.5±4.6	2.6±1.2	3650.0±2848.5
TB (N=32)	32.4	76.7±41.1 30 cases > 33U/L 2 cases < 33U/L	314.2±69.7	43.7±8.6	2.1±.9	3218.8±2232.2 Lymphocytes form 98% of the cells.
Heart failure (N=8)	63.6	24.8±17.8	171.9±16.0	27.1±5.8	4.9±1.7	1137.5±1627.2
Malignancy (N=13	60.8	32.4±51.3 3 cases > 33U/L 10 cases < 33U/L	321.1±60.2	50.5±11.3	1.6±.9	2007.7±1651.0
Miscellaneous*** (N=6)	39.5	6.7±5.9	165.3±11.8	16.5±7.8	6.0±1.3	366.7±310.9

***Undiagnosed conditions inspite of all possible clinical and lab tests. **Non-specific infection. *** Include nephrotic syndrome, celiac disease, liver cirrhosis, hypothyroidism & connective tissue disorders.**

Malignant effusions (n=13); Form 17% of the cases. ADA was below the cutoff value in (77%) 10 cases and only in 3 cases (23%) were above the cutoff value (Table 2). Five were right sided, 6 were left and 2 were bilateral effusions. Cytological smears were positive in 7 cases (53.8%) and negative in 6 (46.2%). Nine cases were metastatic adenocarcinoma, 3 were squamous cell carcinoma, and one small cell lung carcinoma. LDH, mean values were 321.1u/l. (Table-2). Details of other tests and other effusions are listed in table 2.

Discussion

Evaluation of pleural effusion usually includes complete clinical assessment, radiographic studies lab tests of pleural fluid and pleural biopsy. However following these procedures approximately 20% of patients still has undiagnosed conditions [19]. Current study shows marginal significant correlation between final diagnosis and age of the patients, but not with the side of effusion. Highest level of ADA activity in this study was measured in tuberculous effusions.

Cutoff value of ADA was 33u/l gave 93.7% sensitivity, 86.1% specificity and 89.3% accuracy. These results were comparable with other studies [20,21]. The relationship between ADA and final diagnosis was significant (P<0.005). Only two TB effusions (out of 32) showed ADA below the cutoff value and 3 malignant effusions (out of 13) showed ADA level above the cutoff value. The high ADA level correspond to an increase in CD4+ T-lymphocytes as in TB effusion, while its low level correlated with a higher percentage of CD8+ T lymphocytes and a fall in the CD4+ T lymphocytes as neoplastic effusions [20] *Talib Z. et.al. 2001,* showed sensitivity and specificity of 83% and 70% respectively [21]**.** Determination of individual ADA isoenzymes ADA1 and ADA2 could help in distinguishing various causes of increased ADA activity [4, 5].

High LDH associated with increased lactic acid production from polymorph leukocytes and activated lymphocytes[22] Pleural fluid LDH activity has been used to discriminate malignant from non-malignant effusions[21, 22]. In this study the exudative effusions have relatively higher level of LDH than transudate, which is in agreement with other studies [23, 24]. And it was characteristically high in malignant effusions and nearly all-benign effusions have low LDH values.

The cytological examination and evaluation of cells in effusions can be difficult, as in interpretation of long standing transudate effusions characterized by accumulation of few enlarged mesothelial cells, an erroneous false positive diagnosis of cancer can be made [14,15]. While in tuberculous effusions, the differential diagnosis from lymphoma and leukemia depending on the high proportion of mature lymphocytes with paucity of mesothelial cells, the latter is attributed to deposition of fibrin on the pleural surface, either sealing off or destroying it[6, 7]. A further difficulty was in evaluating the accuracy of neoplastic effusions cytology. It is obvious that no single cellular structural changes are diagnostic by itself, a combination of several abnormalities is necessary for accurate diagnosis. In the current study no false positive results was recorded. The sensitivity, specificity and accuracy of cytological diagnosis was 53%, 100% and 72% respectively. Other workers 14, 19, also obtained similar accuracy rate.

Acid-fast bacilli detection by Ziehl Nelseen stain was positive in only two smears of TB cases, similar percentages reported by other studies. TB effusion is usually the result of delayed hypersensitivity reaction to the protein of mycobacterium and the actual bacterial load in the pleural space is low [6, 7, 16].

In conclusion increased ADA levels in TB effusions may reflect highly local cell mediated immune activity in these patients and can be used to differentiate tuberculous

and non-tuberculous effusions. The LDH, protein and glucose level were useful in separation of exudative and Transudate pleural effusions.

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BLEEDING AND THROMBOSIS IN PATIENTS WITH CHRONIC MYELOGENOUS LEUKEMIA

Saad Sh Mansour[1] *FRCPath*, **Raad J Musa**[1] *FICMS*,
Wakas F Al-Sammerai[2] *MSc.*

Abstract

Backgroud: There is considerable variation in the incidence of bleeding and thrombotic complications noted among patients with myeloproliferative disorders (cMPDs).

Objective: To explore the rate of thrombotic and hemorrhagic complications in cMPD and to identify parameters that might be associated with these complications.

Methods: Fourty five patients with various entities of cMPDs were enrolled in this study, which was conducted from January, 2003 to July, 2004 and involves three medical centers in Baghdad. Additionally, 25 apparently healthy individuals were included as control group. The patients and healthy subjects were submitted for the following investigations; (plasma fibrinogen concentration, factor VIII:C, factor VII:Ag, plasma factor X:Ag and plasma D-Dimers.

Results: The total rate of haemostatic complications among cMPD patients was 20 %. These complications was significantly associated with increasing patients' ages (P=0.005) and inversely correlated with the disease duration (r =-315, P<0.05). Factor VII:Ag level was found to be significantly lower in CML patients in comparison to control (P=0.001). Concerning the plasma factor VIII: C, FX:Ag levels and plasma D-Dimer, no association was found between any of these three parameters and the occurrence of thrombohaemorrhagic complications.

Conclusion: Bleeding and thrombosis are frequent complications in patients with cMPD.

Keywords: bleeding, thrombosis, chronic myelogenous leukemia.

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Introduction

The chronic myelogenous leukemia is clonal neoplastic diseases of the bone marrow [1].Bleeding and thrombosis have been recognized as major causes of morbidity and mortality [2]. Moreover, there is considerable variation in the incidence of bleeding and thrombotic complications noted among different series[3, 4]. However, the aim of this study is to explore the rate of thrombotic and haemorrhagic complications in patients with various entities of chronic myelogenous leukemia to identify parameters that may be associated and/ or predictive for the occurrence of these haemostatic complications in those patients.

[1]Dept. Pathology, College of Medicine, Al-Nahrain University [2]Dept. Pathology, College of Medicine, Al-Mustansiriya University.
Address correspondence to Dr. Raad J Musa
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Materials& Methods

Forty five patients with various entities of chronic myeloproliferative disorders (cMPD) were studied and collected from three medical centers in Baghdad: AL-Kadhimiya Teaching Hospital, the National Center of Hematology/AL-Mustansiriya University, and Baghdad teaching hospital. Patients who were on drugs that may affect haemostatic parameters; and those with pregnancy, chronic liver disease, chronic renal failure, and active infection were excluded from the study.

Six patients (4 with CML, 1 with PRV, and 1 with ET) were not receiving any treatment (newly diagnosed). Thirty-nine patients (32 with CML, 5 with PRV, and 2 with IMF) were on treatment.

Additionally, 25 sex-matched apparently healthy subjects of comparable age (13 men and 12 women) with a mean

age (± SD) 41.4 years (range between 24 and 66 years) were enrolled in this series as a control group. The patients and healthy subjects were submitted for the following investigations; (plasma fibrinogen concentration by clotting method of Clauss) [5], plasma factor VIII: C (FVIII:C) level by activated partial thromboplastin time (aPTT) based assay[6], plasma factor VII:Ag (FVII:Ag)[7] and plasma factor X:Ag (FX:Ag)[8] levels by enzyme linked immunosorbent assay (ELISA), and plasma D-Dimers determonation by latex agglutination test [9].

Statistical analysis:

Statistical analyses were done using SPSS version 7.5computer software (Statistical Package for Social Sciences). The statistical significance of the difference in mean of age, fibrinogen concentration, FVIII: C activity, FVII:Ag activity, and FX:Ag activity, between study groups was a tested by ANOVA and Student's *t*-test.

Results

Fourty five patients with various entities of cMPD were enrolled in the present study; thirty-six patients had CML; six patients had PV; two patients had IMF; and one patient had ET. The mean age (±SD) of cMPD patients was 41.35±10.9 years (range between 19 and 65 years). Twenty-four males and 21 females with a male: female ratio (M: F = 1.1: 1). A group of 25 apparently healthy subjects were enrolled in the current study; there were 13 men and 12 women, with male: female ratio (M: F = 1.1: 1). The mean age (±SD) of the control group was 42.2±12.0 years. Descriptions of clinical and laboratory characteristics in the different study groups are listed in Table 1.

Table 1: Description of clinical and laboratory characteristics in the different study groups

Characteristics		Study groups				P value*
		Control n=25	CML n=36	PRV n=6	(IMF and ET) n=3	
Age (Years)	Range	24-66	24-60	40-65	19-61	0.024*
	Mean±SD	42.2±12.0	39.1±7.6	53.0±8.8	45.0±22.7	
Disease duration (Years)	Range	.	0.1-3.0	0.1-5.0	0.1-1.5	0.477*
	Mean±SD	.	1.5±0.8	2.0±2.1	0.9±0.7	
Plasma fibrinogen Conc. (g/L)	Range	1.8-4.2	1.8-5.5	2.3-3.9	2.6-3.9	0.003*
	Mean±SD	2.7±0.7	3.6±1.0	3.1±0.6	3.2±0.7	
Plasma factor FVIII:C (%)	Range	63-100	60-110	70-95	80-105	0.332*
	Mean±SD	78.2±9.1	81.2±12.6	82.5±10.4	90.0±13.2	
Plasma factor FVII:Ag (%)	Range	75-105	60-97	75-92	80-90	0.011*
	Median	95	80	85	85	
	Mean±SD	90.6±8.8	82.3±10.0	85.3±5.9	85.0±5.0	
Plasma factor FX:Ag (%)	Range	75-110	85-105	70-105	95-105	0.735*
	Median	95	95	95	95	
	Mean±SD	94.8±11.0	93.9±6.5	91.7±12.5	98.3±5.8	

*** Test of significance for difference in mean by ANOVA, ** Test of significance for difference in median by Kruskal wallis test.**

Hemorrhagic complications were observed in 5 out of 45 patients (11.1%), while thrombotic complications occurred in 4 out of 45 patients (8.9%). The total rate of occurrence of thrombohaemorrhagic complications was (20%). In addition, there were fewer complications in CML group (5.6 %) than in other cMPD groups (77.8 %), with statistical significance (χ^2-test, P<0.001)(Table 2).

Table 2: Occurrence rate of thrombohaemorrhagic complications in the different cMPD groups

Classification of cMPD patients by clinical evidence of coagulation derrangement	cMPD groups						P value χ^2-test
	CML n=36		(PRV, IMF, ET) n=9		Total n=45		
	No.	%	No.	%	No.	%	
Bleeding	2	5.6	3	33.3	5	11.1	0.04
Thrombosis	0	0	4	44.4	4	8.9	0.001
Asymptomatic	34	94.4	2	22.2	36	80	0.001
Symptomatic patients*	2	5.6	7	77.8	9	20	0.001

***Symptomatic patients; total number of patients with haemostatic complications.**

The rate of these complications was 83.3 % in PV group and 66.7% in (IMF and ET) group without significant difference between these two groups (P=1), However, it was significantly higher in these two groups as compared to CML group (P<0.001, and P=0.02), respectively.

The occurrence rates of thrombo-haemorrhagic complications were significantly associated with increasing age trend (P=0.005)(Table-3) and these complications were directly correlated with age (r =0.469, P<0.01) and were inversely correlated with the disease duration (r=-315, p<0.05).

Table 3: The rate of having disturbed haemostasis (bleeding/ thrombosis) in cMPD patients by certain clinical parameters

Parameters	Disturbed hemostasis (bleeding/thrombosis)						P value χ^2-test
	Negative Asymptomatic		Positive Symptomatic		Total		
	No.	%	No.	%	No.	%	
Age group (years)							0.005
<30	4	80	1	20	5	100	
30-39	14	100	0	0	14	100	
40-49	15	88.2	2	11.8	17	100	
50+	3	33.3	6	66.7	9	100	
Gender							
Female	17	81	4	19	21	100	ns
Male	19	79.2	5	20.8	24	100	

ns= non significant

The mean plasma fibrinogen concentration in CML group was significantly higher than control group (P<0.001) (Table 1). But the differences between other cMPD groups and control were insignificant (P>0.05). However the mean plasma fibrinogen concentration (±SD) in patients with disturbed haemostasis was insignificantly different in comparison with the asymptomatic group of patients (P=0.732). The difference in mean FVIII: C level among these four groups was insignificant (P=0.332).

The mean plasma FVII:Ag levels (±SD) in patients with disturbed haemostasis (81.9±9.9%) was insignificantly different from asymptomatic patients (83.1±9.2%), (P=0.728). The mean plasma FX:Ag (±SD) levels in patients with disturbed haemostasis (95.6±11.6%) was insignificantly different from asymptomatic patients (93.5±6.1%), (P=0.463). The difference in rate of positive plasma D-Dimers between patients with disturbed haemostasis and those who are asymptomatic was statistically insignificant (P=0.5).

Discussion

Bleeding and thrombosis in cMPD occur in varied patterns and incidence. Schafer *et al*[2] found that thrombohaemorrhagic complications occur in about 60% of patients with cMPD. The bleeding syndrome is more frequent than thrombosis, accordingly, the former is most frequent in IMF, while thrombotic complications are most common in PV patients [2,10]. In CML, disordered haemostasis is rare [3]. Besides, disorders of the microcirculation are the most common complaint in patients with ET [10].

Data presented in this series revealed that the total rate of occurrence of haemostatic complications in cMPD patients was 20% (11.1% bleeding episodes, and 8.9% was thrombotic episodes). The previous studies reported a wide range of occurrence rate for the thrombohaemorrhagic events in cMPD patients. For examples, the total incidence of haemostatic complications was 21% as reported by Barbui *et al*[11] while Schafer [2] mentioned a total incidence of 60%. Bleeding events were observed in 33.3% of patients with (IMF and ET) group, 33.3% with PV, and only 5.6% in CML patients, while, thrombotic events were most common in PV patients (50%), followed by (IMF and ET) group (33.3%), whereas, CML patients did not experience thrombotic complications. Accordingly, the occurrence of haemostatic complications was most frequent in PV (83.3%), and least frequent in CML (5.6%), (P<0.001).

Data from various reports indicated that in PV, thromboembolic and haemorrhagic complications occur at rates of 26-63 % and 16-35 %, respectively [12]. Therefore, these figures are comparable with the rates of 50 % and 33.3 % observed in the present study.

Increasing patients' ages were regarded as an important risk factor for cardiovascular events in cMPD patients [2,13,14] For example, in ECLAP study, the incidence of cardiovascular complications was much higher in patients aged more than 60 years or with a history of thrombosis than in younger subjects with no history of thrombosis [15] In agreement with these reports, the occurrence of thrombohaemorrhagic complication was significantly associated with increasing patient age, so 66.7% of patients aged more than 50 years had haemostatic complications, while only 20% of those less than 30 years age had haemostatic complications (p=0.005). Moreover there was significant correlation between age and occurrence of these complications (r =0.469, P<0.01).

Although, the clinical observations revealed insignificant association between the occurrence of haemostatic complication and disease duration (P =0.454), there were out of nine patients with thrombohaemorrhagic complications, six

(66,6%) were newly diagnosed (less than 3 months). As well, correlation study revealed significant inverse correlation between disease duration and the occurrence of these complications (r=-315, P<0.05). These observations be consistent with that of Wehmeier *et al* [16] who reported that the rate of bleeding and thrombosis was highest just before and during the first months after diagnosis and decline there after.

In this study the mean plasma fibrinogen concentration in CML patients were significantly higher as compared to control subjects (2.7±0.7 g/L), (P<0.001). While, the mean plasma fibrinogen concentration in patients with thrombohaemorrhagic complications was insignificantly different from asymptomatic patients (P=0.732). These findings were in agreement with Günay and Öztürk[17] who reported a significantly elevated plasma fibrinogen level in PV (3.83 g/L), and CML (3.73 g/L) patients, though, these elevations were not related with the increased risk of thrombotic episodes in cMPD patients.

Our data revealed that FVIII: C level in cMPD patients were only slightly but not significantly higher than the control subjects (p>0.05). Also, there was insignificant difference between patients with disturbed haemostasis and those with out complications (p=0.786). So the alteration in FVIII: C level does not relate with the occurrence of these complications in cMPD patients. These results are similar to previous reports by Günay and Öztürk[17]. FVII:Ag level were significantly lower in cMPD patients than the respective values in the plasma of healthy subjects (p<0.001), but there was no association with the occurrence of thrombo-haemorrhagic complications in cMPD patients (P=728).

Falanga *et al* found that FVIIt and FVIIz parameters were higher in (30 %) of ET patients than the respective values in the plasma of healthy control subjects, although the elevation in mean concentrations of these two FVII parameters in ET was not significant, but it indicates an increased in vivo proteolysis of FVII in ET that is consistent with hypercoagulation state in ET, and this may be a contributory factor for the increased rates of thrombosis associated with ET [18]. Therefore, further studies are required to elucidate the role of this parameter in haemostatic complications in cMPD patients.

Results in the current study revealed that the FX:Ag level were within the normal range. The mean FX:Ag level in cMPD patients did not significantly differ from that in healthy subjects (p>0.05). As well, there was no association with the occurrence of bleeding or thrombotic complications in cMPD patients. Although, there were no previous reports available about the alteration of FX in the cMPD disorders, results which obtained in the present study might suggest that FX had neither play an important role in the pathogenesis of thrombohaemorrhagic complications nor has a predictive value for these complications in cMPD.

So, it is obvious that both FVII and FX:Ag assay were of little help in the exploration of part of the problem of haemostasis in MPD. It may be suggested that antigenic assay does not reflect a qualitative alteration of these factors and another factor parameters may be more helpful (e.g., procoagulant activity).

Plasma D-Dimers are by products of the coagulation reactions, liberated during clotting activation, that provide a biochemical tool for the definition of the hypercoagulable state and are modulated by therapy. In the current study only 3 (8.3 %) patients (all within CML group) had elevated plasma D-Dimer level (>0.05µg/ml). Moreover, there were insignificant difference in the rate of positive plasma for D-Dimer between patients with disturbed haemostasis and those who are asymptomatic (p=0.5). Falanga *et al*, found a significantly elevated D-Dimer level in PV compared with controls [19], a finding that support a previous observation of a hypercoagulable state in a group of patients with ET [20]**.** This difference in results between the current study and

these previous reports might result from a different way of analysis used in these studies (ELISA method) for measuring plasma D-Dimer level.

Conclusion

1. Bleeding and thrombosis are frequent complications in patients with cMPD. These complications occur
in varied patterns, most commonly in PV, but rarely in CML.
2. Plasma FVIII and plasma FX:Ag activity may have no role in the pathogenesis of haemostatic complications in cMPD. While Plasma fibrinogen and plasma FVII:Ag may play a limited role in the pathogenesis of these complications in cMPD.

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COMPARISON OF BLOOD LEVELS OF ANTICHLAMYDIA TRACHOMATIS ANTIBODIES AMONG MOTHERS AND THEIR NEWBORN BABIES FOLLOWING NORMAL DELIVERIES VERSUS MOTHERS AND NEWBORN BABIES FOLLOWING CESAREAN SECTION

Enas Talib Abdul- Karim[1] *PhD*, **Nidhal Abdul-Muhymen [2]***PhD*, **Tara S. Al-Chrmawindi [3]** *MSc*.

Abstract:

Background: A number of studies have demonstrated that chlamydia trachomatis plays a prominent role in disorders of the human reproductive system.

Objective: This study was carried out to determine antibody levels of Chlamydia trachomatis among mothers with either normal deliveries or had cesarean section and their newborn babies, and the effect of various epidemiological, obstetric, and medical factors on antibody levels among the studied groups.

Method: Serum specimens from 166 women with normal deliveries and their babies (group one) and 32 women with cesarean section and their babies (group two), were screened for C. Trachomatis antibodies by Micro ELISA method.

Result: C. Trachomatis infection rate was 24% and 20.5% among women and babies in-group one, while it was 40.6%and 38.1% in-group two. History of bleeding (significant negative correlation), discharge and urinary tract

Infection (significant negative correlation) during pregnancy, weight of newborn, had higher rate among group two, while fever and anemia during pregnancy, number of previous abortions were higher among women in group one.

Conclusion: Chlamydia trachomatis infection rate was higher among women and their babies following cesarean section than among those with normal delivery.

Keyword: chlamydia trachomatis antibodies in women after delivery

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Introduction

Chlamydia trachomatis is an obligate intracellular parasite that was once thought to be a virus. It has discrete cell walls that resemble Gram-negative bacteria and responds to antibiotic therapy [1]. The Center for Disease Control (CDC) in Atlanta estimates that 3 million people are infected annually, with 75% of infected women having few or no recognized symptom [2]. The increasing incidence of Chlamydia infection in the community has been well documented, along with an increase in cases of neonatal Chlamydia [3]. Prenatal implications of Chlamydia infection for the mother and newborn include associations with ectopic pregnancy, spontaneous abortion, preterm labor, ammionitis, premature rupture of membranes, low birth weight, prematurity, still birth, and neonatal death [4]. Women with Chlamydia during pregnancy are also more likely to develop intrapartum fever and or late onset postpartum endometritis after vaginal delivery [5]. Vertical transmission of C. trachomatis to the neonate occurs in approximately 50% of cases[4], maternal-

[1]Dept.Community medicine,[2]Dept. Microbiology, [3]Dept.Medical Research center, College of Medicine,Al-Nahrain University

Address Correspondence to Dr Enas Talib Abdul- Karim, AL-Kadimiyia P.O. Box 14222 Baghdad Iraq

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infant transfer of this disease occurs in approximately 23 to 70% of infants born to infected mothers, there are also rare cases of C. trachomatis infections in infants born by cesarean section [4]. For the newborn of untreated mothers, inclusion conjunctivitis occurs in 11%-44% of cases, and pneumonia occurs in 11-20% of cases. Furthermore, C.trachomatis in infancy has also been associated with otitis media, broncholitis, pharyngitis, rhinitis and gastroenteritis [6].

Screening for C.trachomatis in pregnancy is considered best practice internationally for detecting and subsequently treating Chlamydia infection in pregnant women, and reducing the associated morbidity [7, 8]. This study was carried out to determine antibody levels of C. trachomatis among mothers with either normal deliveries or had cesarean section and their babies, and the effect of various epidemiological factors on antibody levels among the studied groups

Subjects and Method

A cross sectional study was designed in which the study sample was divided into two groups, group one included 166 mothers with normal vaginal deliveries and their newborn babies, group two included 32 mothers delivered by cesarean section and their babies. Data on the demographic and socioeconomic status of the family, medical and obstetric history of the mother during pregnancy was obtained through well-structured questionnaire form. Both groups were collected from Al-Kadhimyia teaching hospital during the period December 2004- July 2005.

Blood samples obtained from both mothers and babies to measure antichlamydia trachomatis antibody levels via micro ELISA technique.

Standardization procedures were carried out for the antigen (Chlamydia antigen from Virion), conjugate (Antihuman IgG Fab specific, peroxidase conjugate, Sigma) and antisera, and the optical dilutions were found to be 1/10, 1/500 and 1/2 respectively. ELISA test was used following the WHO standard method [9] using the above antigen in proper concentration for coating microwells as a solid phase.

The antibody levels to C. trachomatis (absolute optical density values) were divided into the following groups [10].

- Negative: < 0.91
- Equivocal: 0.91- 1.09
- Positive: > 1.09

Analysis of data was done using SPSS statistical program version10.0 to obtain frequencies, percentages. t test of significant was used. P value of ≤ 0.05 was considered significant

Result

Percentage of low level of antichlamydia antibodies (< 0.91) was higher in the group of mothers with normal delivery and their newborn babies than with cesarean section (76.0%, and 79.5% versus 59.4 and 71.9%), while it was the reverse for antibody levels of more than 1.09 (12.0% and12.0% versus 21.8% and 15.6%). There were no significant differences between mothers and their babies in the two groups table 1.The study showed that history of bleeding during pregnancy was positive in 4.2% of women in first group and 6.3% in the second group, vaginal discharge and fever during pregnancy were 5.4%, 6.6% and 9.4%, 3.1% respectively. Urinary tract infection during pregnancy shows higher percentage (21.9%) among women with cesarean section (group two) than those with normal delivery (10.8%), anemia during pregnancy was higher among women in group one 24.7% than among those in group two (12.5%). The study also shows that 7.8% and 3.1% of babies have weight < 2.5 kg among group of normal delivery and cesarean section respectively table 2.

History of urinary tract infection and vaginal bleeding during pregnancy shows negative and significant correlation with Chlamydia antibody levels only in the second group table 3.

Table (1): Distribution of serum level of antichlamydia trachomatis antibodies among group of mothers and their newborn babies following normal vaginal deliveries and cesarean section

Anti-chlamydia antibody	Normal vaginal delivery		Cesarean section		Significant
	Frequency	Percent	Frequency	Percent	
Mothers					
< 0.91	126	76.0	19	59.4	X^2 =3.77
0.91-1.09	20	12.0	6	18.8	DF= 4
> 1.09	20	12.0	7	21.8	P=
Total	166	100.0	32	100.0	
Babies					
< 0.91	132	79.5	23	71.9	X^2 =0.98
0.91-1.09	14	8.5	4	12.5	DF= 4
> 1.09	20	12.0	5	15.6	P=
Total	166	100.0	32	100.0	

Table (2): Distribution of sample of mothers following normal vaginal deliveries and cesarean section according to some medical, obstetric problems and outcomes of pregnancy

Variables	Normal deliveries		Cesarean section		Significant
	Freq	Percent	Freq	Percent	
Age					X^2 =9.79
<25	80	48.2	12	37.5	DF= 1
≥25	86	51.8	20	62.5	P < 0.01
Bleeding\pregnancy					X^2 =0.97
Yes	7	4.2	2	6.3	DF= 1
No	159	95.8	30	93.8	P > 0.05
Total	166	100.0	32	100.0	
Discharge\pregnancy					X^2 = 1.14
Yes	9	5.4	3	9.4	DF= 1
No	157	94.6	29	90.6	P > 0.05
Total	166	100.0	32	100.0	
Fever\pregnancy					X^2 = 1.06
Yes	11	6.6	1	3.1	DF= 1
No	155	93.4	31	96.9	P > 0.05
Total	166	100.0	32	100.0	
Number of previous deliveries	62	37.3	12	37.5	X^2 = 0.18
0 pregnancy	59	35.5	11	34.4	DF= 1
1 -2	45	17.2	9	28.1	P > 0.05
> 3.0	166	100.0	32	100.0	
Total					

Number of previous abortion 0 abortion 1 -2 3+ Total	138 18 10 166	83.2 10.8 6.0 100.0	23 8 1 32	71.9 25.0 3.1 100.0	$X^2 = 4.92$ DF= 2 P < 0.05
URI \pregnancy Yes No Total	18 148 166	10.8 89.2 100.0	7 25 32	21.9 78.1 100.0	$X^2 = 1.75$ DF= 1 P > 0.05
HB g/dl level\pregnancy > 33 ≤ 33 Total	41 125 166	24.7 75.3 100.0	4 28 32	12.5 87.5 100.0	$X^2 = 7.14$ DF= 1 P < 0.01
Weight of newborn ≥ 2.5 < 2.5 Total	153 13 166	92.2 7.8 100.0	31 1 32	96.9 3.1 100.0	$X^2 = 0.34$ DF= 1 P > 0.05
Gestational age ≥ 37 weeks < 37 weeks Total	151 15 166	91.0 9.0 100.0	32 ---- 32	100.0 ----- 100.0	---------

Table (3): Comparison result of correlation test using antichlamydia antibodies level and different demographic, socioeconomic, medical and obstetrics problems among mothers and their babies with normal deliveries and with cesarean section

Variables	Normal deliveries		Cesarean section	
	Mothers	Babies	Mothers	Babies
Age\years P. Correlation Significant Number	 .039 .621 166	 .099 .203 166	 .314 .080 32	 .251 .166 32
Residency P. Correlation Significant Number	 - .014 .861 166	 .015 .846 166	 .179 .327 32	 .108 .558 32
Mother\education P. Correlation Significant Number	 - .067 .389 166	 -.117 .133 166	 -.065 .725 32	 -.036 .845 32
Crowding index P. Correlation Significant Number	 .098 .209 166	 .161* .039 166	 .328 .066 32	 .279 .121 32
Bleeding\ pregnancy P. Correlation Significant	 -.022 .774	 -.062 .427	 -.353* .048	 -.423* .016

Number	166	166	32	32
Discharge \ pregnancy				
P. Correlation	.066	.065	.053	-.035
Significant	.396	.404	.775	.850
Number	166	166	32	32
Fever\Pregnancy				
P. Correlation	.058	-.081	.081	.097
Significant	.461	.300	.658	.598
Number	166	166	32	32
Number of previous deliveries P. Correlation	.020	.137	-.315	-.191
Significant	.801	.078	.079	.295
Number	166	166	32	32
Number\abortion				
P. Correlation	.092	.048	.004	-.083
Significant	.237	.542	.981	.652
Number	166	166	32	32
UTI\pregnancy				
P. Correlation	- .032	-.035	-.643**	-.470**
Significant	.681	.656	.000	.007
Number	166	166	32	32
Weight of newborn				
P. Correlation	-.127	-.136	-.179	-.145
Significant	.103	.081	.326	.427
Number	166	166	166	32

Discussion

Chlamydia trachomatis infection showed higher rate of infection and higher levels of antibody titers among women and their newborn babies in group two than those in group one. Popovich DM et al [1] 2004 suggested that perinatal transmission usually occurs via vaginal delivery, but infection can also occur secondary to ruptured fetal membrane, directly contaminated the infant's nasopharynx and lungs, there also cases of C. Trachomatis infection in infants born by cesarean section. The finding in the present study could possibly be due to obstetric problems and early rupture of membrane that lead to performance of cesarean section in the studied group, further studies is needed to confer this finding and a larger group of pregnant women in labor is needed.

The finding of increase percentage of women with history of vaginal bleeding, discharge and urinary tract infection during pregnancy (significant correlation for vaginal bleeding and UTI) among women exposed to cesarean section than those with normal vaginal delivery agreed with the study done in USA and published by the American Social Health Association [11]. Babies born with low birth weight (<2.5 kg) represented 7.8% of babies in group one and 9.0% of them were born before 37 weeks of gestation, C. trachomatis infection has been associated with intrauterine growth restriction and prematurity [12, 13], this percentage was less among babies of mothers with cesarean section, this possibly explained by the fact that the number of women in group two was small. A study done in Hungary found that Chlamydia infection was a significant predictor of low birth weight [14]. The finding of no significant association between weights of newborn with Chlamydia trachomatis infection is in agreement with some studies [15, 16], and disagreed with other studies [12, 17, 18].

Conclusion and recommendation: Chlamydia trachomatis infection rate was higher among women and their babies following cesarean section than among those with normal delivery, the antibody levels show significant and negative correlation with history of bleeding and UTI during pregnancy. These finding highlight the need for a routine antibody testing of C. Trachomatis, treatment of women during pregnancy and also advocating for newborn assessment and treatment to reduce the significant, yet preventable morbidity associated with C. Trachomatis infection in both mothers and neonates.

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Serum Magnesium Level in Chronic Asthma In pediatrics

Najim Al-Ruznamaj [1] *FAAP*, **Hussam M. Al-Alwany** [2] *CABP*, **Ibtisam T. Alobusi** [3] *PhD*, **Ammar Al-Shebli**[3] *CABP*.

Abstract :

Background: Asthma in Latin means difficult breathing; it's the most common chronic illness in children. Asthma defined as reversible airway obstruction due to hyper- reactivity of the airways, and its still raising a lot of concern regarding mortality and morbidity, which are still increasing regardless of the advance of management.

The Serum level of Magnesium (Mg) in asthmatics & if any variation from normal children is the subject of this study.

Aim: Aim of the study is to measure the serum level of Mg in asthmatics with different degrees of severity & compare it to normal children.

Patients and method: A total number of 100 patients subjected to study, 50 asthmatics patients & 50 controls patients.

Assessment of asthmatics attacks & measurement of serum Mg for the cases & measurement of serum level of magnesium for the non-asthmatics controls.

Results: The results show that there is lower serum Mg in asthmatics than the control group but no significant correlation to severity of asthma.

Conclusion: Serum magnesium is important element to look for in asthma hence the low serum level compare to other children, which may help in management.

Key words: asthma, magnesium, and severity

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Introduction

Asthma in Latin means difficult breathing & it is also known as reactive airway disease. Asthma is the most frequent admitting diagnosis in children hospitals result in 5-8 lost school days /yr/child [1.]

Asthma defined as reversible airway obstruction due to hyper- reactivity of the airway [1]. It's the most common chronic disease in childhood. Both small (<2mm) and large (>2mm)

Airways involved to varying degree in this hyper reactivity. [2.]

The prevalence, morbidity and mortality of asthma have increased during the last two decades, without specific causes [3].

Airway obstruction in asthma is due to bronchoconstruction, hypersecretion of mucous and mucosal edema due to inflammatory cells. Various allergic and non-specific stimuli and wide variation of factors can cause bronchoconstriction leading to asthmatic attack [4].

Magnesium is the second most abundant intracellular cation. It is

[1]Chief of Iraqi committee of Medical specialization of Pediatrics
[2]Dep.Pediatrics of medical college/Al-Nahrain University.
[3]Dept.of chemistry & biochemistry ,college of Medicine,Al-Nahrain University.
Address Correspondence to Dr. Hussam M. Al-Alwany
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Essential for the activity of many enzymes including the phosphotransferases. Bone contains about 50% of the body magnesium; small proportion of the body·s content is in the ECF(extracellular fluid) [5].

Magnesium found in significant amount in gastric & biliary secretions. Factors concerned with the control of Mg absorption have not been defined, but may involve active transport across intestinal mucosa by a process involving vitamin D. Renal conservation of Mg is at least partly controlled by PTH & aldosteron [6].

Assessed by clinical criteria (mild, moderate and severe) and supported by

The Serum level of Mg in asthmatics is the focus of this study which is an interesting issue.

Aim

To measure the serum level of magnesium in asthmatics & compare it to controls & to see if any change of serum level with severity of their disease.

Patients and Methods

A total number of 50 asthmatic children were subjected to a prospective study regarding the severity of their asthma, which was spirometry measurement of FEV^1 taken by portable spirometer (table 1).

Severity of Asthma attacks(1)

Parameters	Mild	Moderate	Severe	Respiratory arrest imminent
Talking alertness	Sentences, may be agitated	Phrases, usually agitated	Words, usually agitated	Drowsy or confuse
Respiratory rate	Increased	Increased	Mark increase	Parodoxical
Accessory muscle	Usually not	Usually	Usually	Paradoxical thoraco-abdo movement
Wheeze	Moderate, often only end expiratory	Loud	Usually Loud	Absence of wheeze
Pulse / min	<100	100-200	>120	Bradycardia
PEF*	over 80%	60-80%	<60%	

***PEF: peek expiratory flow rate**

A sample of blood was collected from each patient & control in non- heprenized test tube & serum taken after centrifugation.

The principle of test is that Mg ions react with calmagite in alkaline medium to produce a red complex that is measured photometrically at 532 nm . The intensity of color produced is directly proportional to magnesium concentration. Calcium interference is virtually eliminated by EGTA.

The procedure is summarized in table (2).

Table (2)

	Blank	Calibrator	Sample
Doubledist.water	10 µl	--------	----------
Calibrator	---------	10 µl	---------
Sample	---------	---------	10 µl
Color reagentR1	500 µl	500 µl	500 µl
AlkalinereagentR2	500 µl	500 µl	500 µl

Mix & incubate for one minute at 37C or 5 minutes at 20-25 c
Read absorbance (A) against reagent blank. The final color is stable for at least one hour

Calculation of Mg concentration = A sample /A calibrator * concent.calib

Results

The mean serum magnesium concentration of asthmatic patient is 2.6 mg/dl while that of control group is 3.7 mg/dl (p value < 0.01).

No significant correlation between the severity of asthmatic attacks & serum level of magnesium.

Discussion

Many studies suggest the change in serum level of magnesium in hyper reactive airway diseases.

Zervas E., shows that there is 20% decrease in serum Mg & response to nebulized Mg [7].

Alamoudi,O.S., declares that hypomagnesaemia is common in chronic asthmatics. Chronic asthmatics with low Mg tend to have more hospitalizations than chronic asthmatics with normal Mg.Hypomagnesaemia was also associated with more severe asthma [8] .

Hashimoto& colleagues show that 40% of asthmatic patients demonstrated magnesium deficiency, and that the low magnesium

Concentration in erythrocytes reflects decreased magnesium stores in patients with bronchial asthma [9.]

Other studies show that there is normal serum level of Mg in asthma. e.g.

Kakish,K.S., shows that serum magnesium levels in asthmatic children during acute attacks and between exacerbations are not significantly different from those of controls.[10]

Vural & collegues show that No changes were found in serum magnesium and iron levels in patients with asthma as compared to controls [11].

Zervas & colleagues present that the acute asthma is associated with lower erythrocyte Mg content while plasma levels remain unchanged. This decrease in intracellular Mg content occurs regardless of the severity of the exacerbation and returns to normal values after control has been achieved[12].

Conclusion

The serum level of magnesium in asthmatics is lower than other children .No significant correlation of magnesium level to severity of attacks.

Recommendations

- Measurement of serum magnesium level can be added to usual investigations of asthmatic children.
-Magnesium can be used in management of asthmatic attacks because of low level of serum magnesium.
-Measurement of intracellular magnesium can help in asthmatic patients.

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Causes of Neonatal Deaths In Al- Kadhymia Teaching Hospital

Lamia Abdul Karim Al- Saady , *CABP*.

Abstract

Background: neonatal death is the death take place in the first 28 days of life.

Although the neonatal mortality has been declining more rapidly than the post neonatal mortality in the recent decades, neonatal mortality continue to account for close to two third of all infants death.

Aim*: to* review the main causes of neonatal death among the neonates admitted to the nursery care unit (NCU) in Al- Kadhymia Teaching hospital for ten years period. in order to prevent or treat the treatable ones.

Patients and Methods*:* Through a retrospective study, analysis of the medical records of all the admitted neonates to the NCU in Al- Kadhymia Teaching Hospital during the period between 1995 -2005, the medical information were analyzed to find the important causes of neonatal deaths.

Results*:* the number of admitted cases during this period was 2683 cases and the total numbers of deaths were 982 cases (36.6%). We found that the main causes of death were Respiratory distress syndrome (RDS), neonatal sepsis, birth asphyxia, congenital anomalies, meconium aspiration and infant of diabetic mother.

Conclusion*:* the most important causes of deaths were sepsis, birth asphyxia and congenital anomalies. Prevention of prematurity as a major cause for RDS will lead to a decrease in neonatal mortality and morbidity, and a significant reduction will depend on genetic counseling and prevention of congenital anomalies.

Key Words: Neonatal Death, NCU, RDS .

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Introduction

Neonatal mortality now account for approximately 2\3 of the eight

Million deaths in children less than one year of age, its highest level occurs in the first 24 hour of life. World wide 98%of deaths occur in the developing countries and largely attributed to infections, birth asphyxia, a consequences of prematurity and low birth weight and congenital anomalies. There are important variations in the leading causes of deaths noted for neonatal and post neonatal periods. The leading cause of death for 2000were congenital malformations, deformities and chromosomal abnormalities, disorders related to short gestational age and low birth weight, respiratory distress, bacterial sepsis, intrauterine hypoxia and birth asphyxia [1,2].

Aim

To find the main causes of death in Al – Kadhymia Teaching Hospital and to see where further

Dept. of pediatric, Medical Collegeof| Al – Nahrian University.

Address Corespondence to Dr. Lamia Al- Saady. Lecturer in College of Medicine in Al- Nahrain University.

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improvement may be possible and preventive measures can be applied to decrease the mortality and morbidity.

Patients and Methods

This is a retrospective study through which analysis of the medical records of all the admitted neonates to the NCU in Al-Kadhymia Teaching Hospital during the period between 1995- 2005 to find the major causes of deaths among the admitted cases also we study the gestational age of the admitted cases Although the detailed data were not available due to inadequate collection of health information during this period.

Results

The study showed that the total no. Of the admitted cases were 2683 live born and the no. Of the died cases were 982(36.6%) of all the admitted neonates.

The main causes of deaths were RDS, sepsis, birth asphyxia and congenital anomalies (lethal), these results shown in table (1).

The cases of RDS occur mainly in the premature neonates 524(97.76%) while there were only 10cases(1.90%) in the term neonates. The distribution of prematurity and

RDS were shown in table (2).

The cases of birth asphyxia occurred mainly in preterm neonates (65) cases while the Term neonates were constituted only 15 cases of birth asphyxia.

The cases of meconium aspiration occur mostly in term infants (27) cases, 3 cases post term and only 5 cases were preterm.

Discussion

The major cause of death was RDS which constitute 536(54.52%) of all the deaths there are similar results which agree with our study that problems of RDS, preterm birth, sepsis, lethal malformations, asphyxia were still the main causes of neonatal deaths and account for 95% of deaths [3,4,5.]

For the RDS it is estimated that 30% of all neonatal deaths result from RDS or its complications, it occur primarily in preterm infants, the incidence inversely related to gestational age and birth weight, it occur in 60-80%of infants <28 weeks gestation, in15-30%of those between 32-34weeks, in about 5% beyond 37 weeks and rarely at term [6]. In our study 48.09% of the cases occur in neonates < 28 weeks, 29-32weeks were 155(29.58%), 33-36 weeks were 107 (20.41%) and only 10 cases beyond 37 weeks (1.90%). Most of cases of RDS were preterm 524(97.76 %), similar results of prematurity and its complications is the leading cause of neonatal mortality and substantial portion of all birth related short and long-term morbidity [7].

The incidence of sepsis is 1-8\1000 live births, the mortality rate is high (13-25%), higher in premature and those with early fulminante disease [8].

Infections in the neonates still constitute a significant cause of death in our study I was 22.5% of all deaths, in a study done in Gambia, West Africa, infections accounted for 37% of all deaths [9].

As many as 2% of fetuses are infected in utero, and up to 10% of infants are infected during delivery or the first month of life. The infant acquired the organisms from the delivery room (contaminated equipments), in the nursery care unit (hospital personnel, or visiting families) and it can be transmitted by direct contact or indirect contact with contaminated vehicles (intravenous fluid, respiratory equipments), antibiotics interfere with colonization by normal flora, crow dining and inadequate infections control techniques (hand washing between patients examination) may also contribute to the problem, also low birth weight , long stay in the nursery , invasive procedures and catheters , endotreachal tubes and alterations in the skin and mucous membranes barrier all these may contribute to high incidence of infections [10].

Neonatal mortality due to congenital malformations or genetic disorders has no decrement despite a decrease in overall

neonatal mortality with recent advances in medical technology, as a consequence an increase in percentage of neonatal deaths attributable to congenital malformations and genetic disorders.

In our study the congenital anomalies were not specified in most of the cases and didn't give the precise diagnosis, it was found that it constitute to9.57%of all deaths. While in another retrospective study reviewed the neonatal deaths in NCU at Kosair Children Hospital, Kentucky. The congenital malformations were responsible for approximately 45% (range 32-61%), other major causes of deaths were extreme prematurity, respiratory disorders, sepsis, asphyxia and primary pulmonary hypertension [11].

Another cause of death was birth asphyxia which constitute for 3.76%of all neonatal deaths, the distribution of cases were 65 cases preterm and 15 cases term infants in a study done in south Africa found complications of prematurity and hypoxia were the most conmen final cause of death in neonates. This occur in spite of major advances in monitoring technology and knowledge of fetal and neonatal pathologies, perinatal asphyxia or more appropriately hypoxic –ischemic encephalopathy(HIE) remain a serous cause of perinatal mortality and long term morbidity in developing countries [12].

The death rate in term infant with severe hypoxia is about 11% and about 0.3in1000 live term births are severely affected. The incidence of hypoxic- ischemic encephalopathy, death and handicap rates all are significantly high in preterm infants [13].

Although the incidence of HIE and its consequences in term infants has fallen significantly, meconium aspiration (represent fetal asphyxia and distress) which usually occur in term and post term infants 5% of these infants develop aspiration pneumonia of which 30%require mechanical ventilation and 5-10% may expire [6]. In our study there were only 3.76% of all deaths were due to this condition the term infants were 27, postterm5 and 5 preterm. , The ultimate prognosis depend on the extent of CNS injury from asphyxia .The passage of meconium in an asphyxiated infants < 34 weeks gestation is unusual and may represent bilious secretion secondary to intestinal obstruction (ileus) [14].

Of the less common causes of neonatal deaths were infant of diabetic mothers which contribute to only 1.42%of all deaths which may be explained by the fact that good control of maternal diabetes is the key factor in determining the fetal outcome. Data indicate that perinatal mortality and morbidity in the neonates have improved with dietary management and insulin therapy [15].

The mortality rate is over 5 times higher for non diabetic mothers and is higher at all gestational age and in every birth weight for gestational age category [16, 17].

These infants are three times the risk for malformations compared with offspring of non-diabetic mothers, it present in about 1in 2000delivary [18]. Poor control in the first trimester is associated with higher percentage of congenital malformations, it account now to 50% of perinatal deaths and include cardiac,GIT and CNS defects[19].

Conclusion Of the important causes of deaths were RDS, sepsis, birth asphyxia, and congenital anomalies.

Prevention of preterm delivery and low birth weight continue to be a priority for reducing neonatal mortality.

Genetic counseling could lead to further decline in neonatal mortality.

The diagnosis, treatment and prevention of congenital anomalies are critical for reducing over all neonatal mortality.

Important factors for prevention of infections are scrub suits for the nurses and residents, hand washing hands between the patients adequate nursing staff and avoidance of overcrowding.

Recommendations: Early provision of intensive observation and caring to high-

risk newborn infants can significantly reduce morbidity and mortality. Provision of experienced and skilled personnel especially designed and organized regional hospital units, proper equipments. Prevention of premature birth should be more emphasized to decrease neonatal mortality and morbidity.

Prevention and early diagnosis and intervention of the causes of death and regionalization of perinatal care with more comprehensive transport system are mandatory.

Better antiseptic measures would significantly reduce sepsis as a major cause of death,

Table (1): The major causes of death in the NCU.

Cause of death	No.	%
RDS	536	54.58%
Sepsis	221	22.5%
Congenital Anomalies	94	9.57%
Birth asphyxia	80	8.14%
Meconium Aspiration	37	3.76%
Infant of diabetic mother	14	1.42%

Table (2): The distribution of RDS cases according to the gestational age.

%	No.	Gestational Age
48.09%	252	<28week
29.58%	155	29-32 week
20.41%	107	33-36 week
1.90%	10	>37 week

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Lactate Dehydrogenase Isoenzymes Pattern in Differential Diagnosis of Pleural Effusions.

Hussam H. Ali[1] *MBChB,* **A.W.R. Hammed** [2]*PhD*, **Zainab T. Al-Okab**[3] *PhD.*

Abstract

Objectives: Total lactate dehydrogenase (LD) in the pleural fluid (PF) is of little value in the discrimination of various types of exudative effusions such as malignant from non-malignant effusions.
The aim of this study is to assess the diagnostic value of LD isoenzymes activity in serum & pleural fluid in the differentiation between various exudative pleural effusions.

Methods: Sixty-Six patients with pleural effusions were included in the study. Activity of total LD & isoenzyme were measured in pleural fluid & serum. Isoenzymes were separated by agarose gel electrophoresis & the quantity of each isoenzyme was measured by spectrophotometer.

Results: Exudative (inflammatory, neoplastic) effusions had a relatively high LD levels compared to transudates.
LD isoenzymes pattern was significantly different between transudates & exudates. PF LD isoenzymes pattern differs from that in serum. Our results showed that mainly the pattern of LD3 in pleural fluid & serum was helpful in discriminating inflammatory exudates from neoplastic exudates.

Conclusion: The LD isoenzyme pattern differed between pleural effusions of transudative and exudative origin. Moreover including the LD isoenzyne activities in the biochemical work up of pleural effusions reveal an additional discriminatory value in the separation between various exudative effusions, especially between inflammatory exudate & neoplastic exudates.

Keywords: Pleural effusion,lactate ehydrogenase isoenzymes

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Introduction

Lactate dehydrogenase (LDH) is a cytoplasmic enzyme present in essentially all major organ systems. The extracellular appearance of LDH is used to detect cell damage or cell death [1]. Due to its extraordinarily widespread distribution in the body, serum LD is abnormal in a host of disorders [2, 3].

[1]. Dept. of Pathology
[2]Dept of Chemistry and Biochemistry
[3]Dept Lecturer Medical Research Center, College of Medicine, Al-Nahrain University.
Address Correspondences to Dr. Zainab T. AL-Okab,E-mail : Zainabakab@hotmail.com
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Therefore, LDH measurement is a sensitive, but rather non-specific test.
LDH activity has been extensively used in the analysis of pleural effusions, especially in distinguishing between transudate & exudate [4, 5]. However, total LDH activity in the pleural fluid (PF) is of little value in the discrimination of various types of exudative effusions such as malignant from non malignant effusions [4, 6].
Eventhough the total PF LDH activity is not useful in distinguishing among various exudative pleural effusions; one might suppose that LDH isoenzymes could be of additional value in the differentiation. Few studies reporting the analysis of LDH isoenzymes in pleural

effusions were found and the results were conflicting [7-9].

Patients and Methods:

Patients

From 1st of February 2000 to the end of October 2000, 66 pleural effusion fluids, as well as blood samples were obtained from 66 in patients admitted to the kahdemyia hospital. Patients were categorized into three groups (table 1).

Table (2) shows the age & sex distribution among patients presented with pleural effusions on all PF samples, the following analyses were performed: protein, LD, LD isoenzymes, bacterial culture, acid-fast bacilli smear and cytology. Simultaneously a sample of serum was obtained to measure biochemical parameters.

The aspirated PF, blood (10 ml each) were separated by centrifugation by 2000 xg for 10 minutes, then supernatant and serum were aspirated and dispensed into 0.2 ml tubes and stored at room temperature (20-25° c) for not more than 2-3 days.

Determination of LD activity

LD activity was determined according to the method of Wroblewski and La Due [10].

Separation & measurement of LD isoenzymes by electrophoresis

1- LD isoenzymes separated on agarose gels according to the method of Eleritch 1966 [11] with some modification: colorimetric determination of the relative amounts of each isoenzyme present is accomplished by the addition of substrate containing lactate (500 mM), nicotinamide adenine dinucleotide (NAD)(10 mg), nitroblue tetrazolium salt (NBT) (1 mg/ml), phenazine methosulphate PMS (1 mg/ml), Tris-Hcl buffer (0.057 M,PH 8.0).
2- After the isoenzyme have been separated by agarose gel. Cellulose acetate membrane was soacked in the above reaction mixture and then layered over the separation gel; the plate is incubated for 15-20 min. in 37° c oven. After incubation, the membrane is removed, fixed with 5% acetic acid and stored for elution.
3- To estimate the relative amount of each isoenzyme, the strips were cut into sections, each section was transferred to a test tube with tight cup and both the dye and the membrane were completely dissolved by solvent mixture (ethanol: chloroform).
4- The substance was read at 546 nm against a blank made by using part of cellulose strip with a similar area.
5- The absorbance of given fraction divided by the sum of all the absorbance, yield the fractional amount, in percent of the given isoenzyme. This fraction when multiplied by the total LDH activity gives the total amount of the fraction in U/L.

Statistical analysis

Student's t-test was used for comparison of pleural fluid and serum LD activity and ANOVA was used for comparison among different groups. The linear regression and the Pearson coefficient of correlation (r) were determined.

Results

Table (3) shows the mean PF LD isoenzymes activity. Among groups, LD1 activity in male patients did not show significant difference, while the mean LD isoenzymes activity from LD2 to LD5 were significantly high in group II and III as compared to group I ($P< 0.01$), but there was no significant difference in LD isoenzyme activities between group II & III.

In female patients, the pattern differes from that in male patients with a significant high LD1 activity in group II as compared to both group I & II ($P< 0.01$; $P< 0.05$, respectively).

LD3 activity was higher in group II as compared to both group I and group

III (P< 0.01), as well as group III as compared to group I (P< 0.01).

While the results of LD isoenzymes in serum of male patients revealed that the mean serum LD1, LD2 and LD3 activities were higher in group I as compared to group II (P< 0.01), and non significantly different as compared with group III (table 4). The mean LD4 and LD5 activities did not show significant difference between the three groups.

In female patients the isoenzyme pattern differs completely from that in male patients, LD1 was higher in group I compared to group II (P< 0.05), but non significantly different as compared to group III (P> 0.05). In addition LD2, LD3, LD4 and LD5 did not show significant difference among the three groups

Serum Vs pleural fluid LD isoenzymes activity:

Figure (1) illustrates the distribution of individual results for both PF and serum LD isoenzyme activities for both sexes.

LD3 isoenzyme activity had distinct pattern in the three groups. Since in group I, serum LD3 activity was significantly higher than that in PF (P< 0.01), and vice versa in group II, while there was no significant difference between PF and serum LD3 activity in group III patients (table 3,4). Figure (2) demonstrates a suggested scheme for separation of the three groups of pleural effusion patients according to their LD isoenzyme activities.

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Table 1: Classification of 66 pleural effusions.

Group I	No.	Group II	No.	Group III	No.
Transudate	12	Inflammatory exudates	54	Neoplastic effusion	23
CHF	6	Pulmonary TB	23	Lung CA	5
Renal disease	6	Pneumonia	4	Breast CA	3
		Empyema	4	Larynx CA	2
				Bronchial CA	1
				Bladder CA	1
				Thyroid CA	1
				Pancrease CA	1
				Lymphoma	4
				Unknown primary	5

*** CHF : Congestive Heart failure**
*** TB : Tuberculosis**
*** CA : Cancer**

Table 2: Age and sex distribution among patients with pleural effusions.

Group (No.)	Mean ± SEM (yr)	Range (yr)
Group I Transudate		
Male (7)	*53 ± 4.8 Years	40-80
Female (5)	47.4 ± 7.6 Years	29-72
Group II Inflammatory exudate		
Male (24)	34.6 ± 3.6 Years	12-70
Female (7)	34.3 ± 6.9 Years	15-65
Group III Neoplastic effusion		
Male (11)	**60.8 ± 3.13 Years	45-80
Female (12)	49.1 ± 6.3 Years	14-88

* P<0.05 versus group II

** P< 0.01 versus group II

Table (3): Lactate dehydrogenase (LD) and LD isoenzymes activity in pleural effusion fluids:

	Male (U/L)							Female (U/L)						
Groups	No	LD	LD_1	LD_2	LD_3	LD_4	LD_5	No	LD	LD_1	LD_2	LD_3	LD_4	LD_5
Group I	7	120±22**	31±5.4	26±5.0**	28±5.0**	20±3.2**	19.5±4.7**	5	105±9**	21±4.0	23±3.0**	23±3.0	20±1.6**	17±2.0**
Group II	24	342±23	50±3.6	63±5	72±6.5	78±6.5	79±7.7	7	318±47	59±12*#	68±10	85±15##	60±8	62±10
Group III	11	283±43	36±8.8	58±10	60±10	68±11	72±11	12	235±45	39±8	52±11	52#±11	50±10	44±9

- Date were expressed as (Mean ± SEM)

* P<0.05 versus group III

\# P<0.01 versus group I

\## P<0.01 versus group I and III

** P<0.01 versus group II and III

Table (4): Lactate dehydrogenase (LD) and LD isoenzymes activity in serum:

	Male (U/L)							Female (U/L)						
Groups	No	LD	LD_1	LD_2	LD_3	LD_4	LD_5	No	LD	LD_1	LD_2	LD_3	LD_4	LD_5
Group I	7	299±35	75±8.7**	74±10.7**	71±12.7**	43±4.6	36±3.4	5	263±38	75±11*	79±11.6	57±11.2	26±3.6	24±5.5
Group II	24	225±16	48±4.3	51±3.8	47±3.8	38±3	40±4.6	7	237±39	53±9	67±17	55±12	36±2.6	28±3.4
Group III	11	234±22	59±11.7	59±7.6	51±3	35±2.5	31±2.4	12	250±27	59±6.7	69±10.2	54±6.5	35±4.8	34±4.5

- Date were expressed as Mean ± SEM

* P<0.05 versus group II

** P<0.01 versus group II

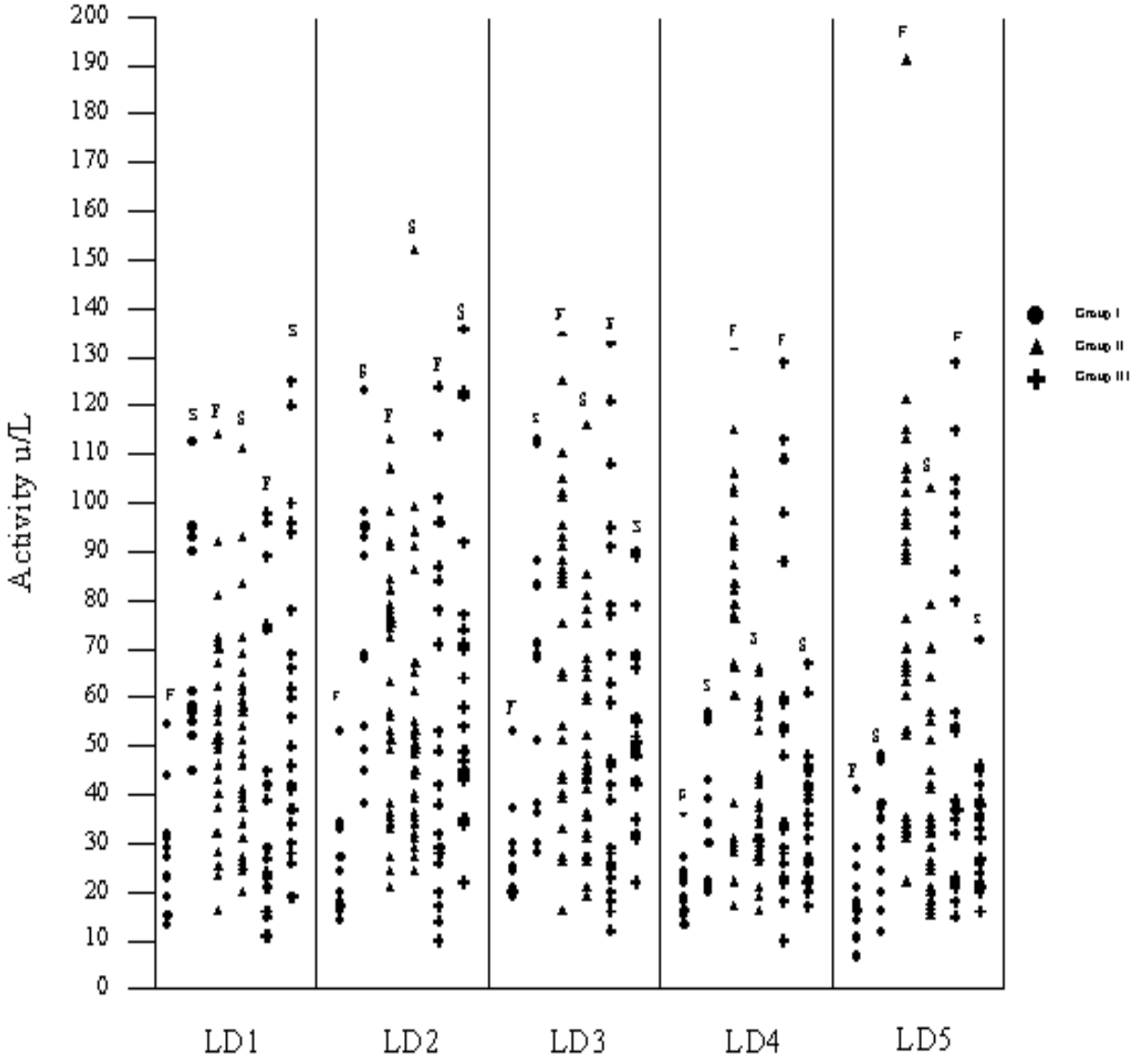

Figure (1): Distributions of pleural effusion fluids (F) and serum (S) Lactate dehydrogenase (LD) isoenzymes.

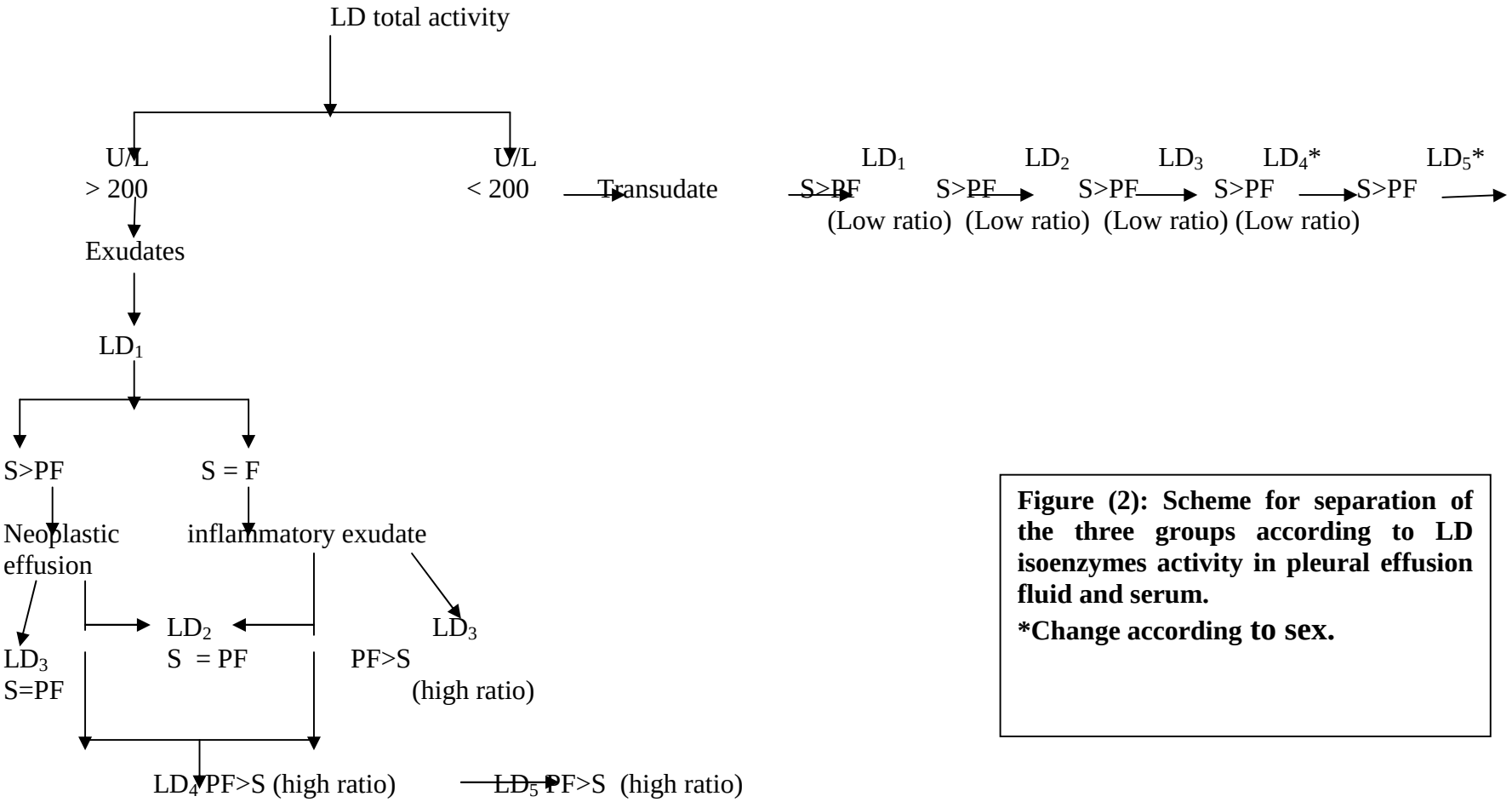

Figure (2): Scheme for separation of the three groups according to LD isoenzymes activity in pleural effusion fluid and serum.

***Change according to sex.**

Discussion

Pleural fluid LD activity has among others, been used in the analysis of pleural effusion especially, to discriminate transudates from exudates [3, 4].

The current study indicates that exudative (inflammatory, neoplastic) effusions have a relatively high LD levels compared to transudates, which is in agreement with other workers [5, 6, 7], and in contrast with others [10, 12, 13], who reported that an elevated PF LD was characteristic of malignant effusions and that nearly all benign effusions had low LD levels.

Cytoplasmic, cellular enzymes, such as LD in the extracellular space are suggestive indicators for disturbance of the cellular integrity induced by pathological conditions.

As LD is present in essentially all major organ system [14], LD measurement is a sensitive, but rather non specific test. The concentration of the PF LD is a reliable indicator of pleural inflammation even though, the total PF LD activity is not useful in distinguishing among various exudative PF, one might suppose that LD isoenzymes could be of additional value in the differentiation.

Reviewing the literature conflicting data were found and only the relative values of LDH isoenzymes, as percentage of total LDH were studied. The current study evaluates the absolute LDH isoenzymes activity in various PF as well as among different sex groups. In this study LDH isoenzymes pattern was significantly different between transudates and exudates (inflammatory & neoplastic) which is in agreement with other worker [15].

Moreover, in exudates the absolute activity of LD3 was higher in inflammatory than that in neoplastic effusions, although it was significant only in female patients. In the current study PF LD isoenzymes pattern differs from that in serum, a result was comparable to the result found by Paavonen and associates [16]. Most of the isoenzymes activity in transudates was lower than that in serum, which is in agreement with other workers [17].

All inflammatory exudates were characterized by higher activity of LD3, LD4, and LD5 than the corresponding serum isoenzymes. Similar results were obtained by other workers [7, 18].

The finding that inflammatory exudates effusions had a high LD4 and LD5 as compared to the corresponding serum may be explained by the following observations. Because a marked PF leukocytosis usually occurs in disease in which injury to the lung occurs, the LD4, and LD5 from PMN leukocytes probably contributes in the elevation of these isoemzymes in the PF [19.] Processes characterized by mesothelial proliferation would show mostly elevation of LD4 & LD5, since these isoenzymes predominate in mesothelial cells [20]. Moreover, the PF lymphocytes in disease states such as tuberculosis are probably immunologically stimulated, and although lymphocytes usually contain LD1 & LD2, immunologically stimulated lymphocytes contain mostly LD4 & LD5 (M), where as in neoplastic effusions only LD4, LD5 activities in effusions were higher than their corresponding serum activities. However, it was significant only in male patients. This result was in agreement with other worker [17] and in contrast to Frohlich & associates [18], who reported that neoplastic effusions were characterized by maximal enzyme activity in LD2, LD3 and LD4. The high activity of PF LDH4 and LDH5 in neoplastic effusions indicates that the origin of these isoenzyme in effusions is unlikely to be from the serum. Since no correlation has been found in the current study and local LD4, LD5

concentration exceeding those found in serum with a high fluid to serum ratio.

Our results showed that mainly the pattern of LD3 in PF & serum was helpful in discriminating inflammatory exudates from neoplastic exudates. The high LD3 activity in pleural inflammatory exudates indicate that the source of LD3 in effusion is unlikely to be from the serum since no correlation was observed, and probably LD3 contribution from the lung and from the inflammatory cells in the pleura cavity. In contrast to Cobben and associates [21], who reported that mainly the percentage of LD4 & LD5 are helpful in discriminating malignant effusions from benign exudative effusions (i.e parapneumonia effusions).

In the current study, neoplastic pleural effusion had variable LD isoenzymes pattern. This could be due to the various neoplastic tissues that secrete different LD isoenzymes. It has been shown that malignant lymphoma and small cell lung carcinoma differ from other malignancy by a low LD5 isoenzyme secretion. Alternatively, the extent of the pleural inflammatory response to malignancy and the variable degree of pleural PMN leukocytosis may determine the relative levels of LD4 & LD5 isoenzymes [8]. The marked heterogenity of malignant etiologies and the relative small number of patients with neoplastic effusions in the current study precluded separation between various LD isoenzymes pattern according to the cytopathologic diagnosis.

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THE VALUE OF PANORAMIC RADIOGRAPHY IN THE DIAGNOSIS OF MAXILLARY SINUS DISEASES

Tahrir N. N. Aldelaimi *MSc(BAGH)*

Abstract

Background: Diseases of the maxillary sinus may create symptoms that the patient might interpret as of dental origin, and conversely, dental diseases may adversely influence the health of the sinus

Objective: To interpret the panoramic radiograph of maxillary sinus in a sample from Anbar population,

Methods:120 subject aged from 30 to 70 years, mean age 58 years , who underwent orthopantomographic examination for different medical & dental treatment purposes including males (56%) and females (44%) . Panoramic radiographs were taken in College of Dentistry, Anbar University, Ramady City, Anbar. With Cranex – Soredex panoramic x-ray machine (Helsinki, Finland).

Results: Normal maxillary sinus were found in (58%) while radiographical changes (maxillary sinus findings) were found in (42%) including mucosal thickening were (32%) and (4%) of the findings were classified as mucous retention cysts.

Conclusion: The maxillary sinus findings were more common in fifth decade of life and slightly higher percentage in male group and the majority of findings were found in dentate subjects.

Key words :axillary sinus,OPG,Mucosal thickening.

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INTRODUCTION

Diseases of the maxillary sinus may create symptoms that the patient might interpret as of dental origin, and conversely, dental diseases may adversely influence the health of the sinus [1]. The response of the sinus mucosa to the odontogenic inflammation has been called periapical mucositis[2]. This is usually defined as localized thickening of the sinus mucosa, which reach sometimes 10 – 15 mm as a result of irritating stimuli [3]. This is considered the most common antral lesion and requires differentiation from a mucous retention cyst [4, 5, 6]. Mucous inflammatory lesion is believed to be caused by products of pulpal or periodontal diseases that penetrate the antral floor and reach the mucosa causing it to thicken locally [7]. Clinical and radiographic studies have shown that mucous thickening in the maxillary sinus is common in individuals with apical infections at the upper molars and premolars than in individual with healthy periodontal tissues [8, 9]. The close contact between the roots of the upper molars and premolars and the maxillary sinus, and the numerous anastomoses in the apical region of these teeth and corresponding vessels in sinus mucosa

Dept surgery college of dentistry, Anbar University
Address correspondence to Dr Tahrir N. N. Aldelaimi: P.O.Box 55317 Baghdad E-Mail; tahrir_aldelaimi@yahoo.com
Mobile: 07901896381
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have been found to permit the spread of odonogenic pathological processes from the periodontium and pulpal spaces both directly and via vessels to the maxillary sinus [10, 11]. In radiographical studies of both dentate and edentulous subjects. Prevalence figures ranging from 2% to 13% have been reported [3, 12, 13]. The diffuse mucosal thickening is more common with frequencies up to 50% of the radiographic incidental findings [14]. Mucous cysts which are included in the paranasal sinuses are more common in the maxillary sinus [15]. Bjorn et al. [16] and Lindhall et al. [17] found radiographic signs of long standing mucosal changes in the maxillary sinus in 10.6% of statistical sample of a Swedish population. Prevalence figures for sinusitis due to dental causes vary between 4.6 and 47%. However it has been suggested that, mucous retention cysts are insignificant clinically and only of radiograph interest [18]. Further more mucous cysts and mucosal thickening usually cause no symptoms, but occasionally they have been related to a variety of symptoms, mainly, facial pain, headache and toothache [3. 19]. Mucosal thickening resolve when their caused is removed. In symptomatic cases, however surgical removal of the cyst may be indicated [(20,21,22)]. Myall et al in 1974 [6] stated that benign mucosal cyst is the most maxillarymolars · Its incidence varies by Halstead in 1973 [20] To 9.6% in one retention cysts are round, ovoid or domeshape shadow of uniform density within the maxillary sinus whose base is continuous with the floor or the wall of the maxillary sinus and the free surface of the lesion should be smooth and sharply defined and adjacent to an air shadow. Also, there should be no osseous cortex [6]. Layon [24] has discussed the reliability of panoramic radiography in the diagnosis of maxillary antral pathosis. The main disadvantage of panoramic radiography arises from their dynamic projection technique, distortion levels may reach 30% in the third molar region [25, 26]. The maxillary sinus is clearly imaged in panoramic radiography, but small changes out side the 2 –3 mm thick sharply depicted layer are not visualized in the normal panoramic projection, the roof of the maxillary sinus is not imaged because of superimposition of bones [27]. However mucous cysts and other mucosal thickening are usually well demonstrated as they almost always arises from the antral floor not from roof [30, 29, 28, 23].

Statistical analysis: includes percentages, mean, standard deviation and student "t" test. The finding was considered as statistically significant if the p value <0.005, Karl –person coffiecient of correlation (r) was used to find inter observer reliability (-1<r<+1).

MATERIALS AND METHODS:

120 subject aged from 30 to 70 years, mean age 58±8 years, who underwent orthopantomographic examination for different medical & dental treatment purposes including 66 males (56%) and 54 females (44%). Panoramic radiographs were taken in college of Dentistry, Anbar University, Ramady City, Anbar. With Cranex – Soredex panoramic x-ray machine (Helsinki, Finland), All patient were referred to college of dentistry requesting OPG examinations, panoramic films were processed by Kodak RP X-omat automatic processor. The radiographs then were studied under standardized condition by two independent examiners (double blind technique) with the use of magnifying lens of radiographic viewer. Panoramic radiographs were interpreted for these findings using a standardized radiographic criterion of mucosal thickening and mucous retention cyst of the maxillary sinus (24,91,6). The mucous retention cyst is a well defined dome-shaped opacity with convex outline arising from the floor of the maxillary sinus, while the mucosal thickening is represented by the more diffuse opacities along the margins of the sinus without well-defined rounded outline, as mentioned both are usually well demonstrated as they almost always arises from the antral floor not from roof. [(30,29,28,23)].

Result

The study sample was including 66 (56%) males and 54 (44%) females with age ranged from 30-70 years of mean age 58±8 year. The distribution of the number of patients and age groups are summarized in (Table 1). Normal radiographical (maxillary sinus findings) were 70 subjects (58%) while maxillary sinus findings were found in 50 subjects (42%). Including mucosal thickening in 38 patients (32%) and 4 patients (4%) have mucous retention cysts (Table 2). The highest percentage of mucosal thickening was found that in the age group (40-49) years represent (14%) within the age group. Regarding the mucous retention cyst the highest percentage was found also among the age group years representing (2%) (Table 3). Regarding the sex (Table 4), the maxillary findings were slightly higher in the males rather than the females, where the mucosal thickening was found in (18%) within gender. While the mucous retention cysts were found in (3%) within the gender. Table 5 showed that the prevalence of mucosal thickening in dentate and edentulous patients representing (20%) and (12%). Other maxillary sinus findings were also recorded in this study. There were (4%) of patients showed impaction & displacement of a tooth inside the maxillary sinus. The impacted maxillary teeth or tooth were either canine or second molar, also severe pneumaitization of the maxillary sinus floor down to the alveolar crest was seen in (2%).

TABLE 1: T he distribution of age group in relation to sex

AGE GROUP	MALE	FEMALE
30-39	٥%	٣%
40-49	24%	28%
50-59	18%	9%
60-69	9%	4%
TOTAL 120(100%)	66(56%)	54(44%)

TABLE 2: The distribution of radiographical maxillary sinus findings*

MAXILLARY SINUS FINDINGS		PERCENT
Normal		70(58%)
Mucosal thickenings		38(32%)
Mucous retention cyst		4(4%)
Others	Root inside antrum	4(4%)
	Pneumatization (sinus floor to alveolar ridge)	3(2%)
TOTAL		120 (100)%

** r=0.9*

TABLE 3: The distribution of maxillary sinus finding in relation to patients age group

AGE GROUP	NORMAL	MUCOSAL THICKENING	MUCOUS RETENTION CYST	OTHERS
30-39	4%	2%	0%	1%
40-49	26%	14%	2%	2%
50-59	17%	10%	1%	2%
60-69	11%	6%	1%	1%
TOTAL 120(100%)	70(58%)	38(32%)	4(4%)	7(6%)

TABLE 4: The distribution of radiographical maxillary sinus findings in relation to sex

SEX	NORMAL	MUCOSAL THICKENING	MUCOUS RETENTION CYST	OTHERS
MALE	32%	18%	3%	4%
FEMALE	26%	14%	1%	2%
TOTAL 120(100%)	70(58%)	38(32%)	4(4%)	7(6%)

TABLE 5: The distribution of radiographical maxillary sinus finding in relation to maxillary arch

MAXILLARY ARCH	NORMAL	MUCOSAL THICKENING	MUCOUS RETENTION CYST	OTHERS
DENTATE	34%	20%	2%	3%
EDENTULOUS	24%	12%	2%	3%
TOTAL 120(100%)	70(58%)	38(32%)	4(4%)	7(6%)

DISCUSSION

The prevalence of mucous and diffuse mucosal thickening in all the paranasal sinuses has occasionally been as high as 50% in facial radiographs taken for indications other than suspected sinus disease [32]. In magnetic resonance imaging study of incidental findings in the paranasal sinuses of 438 subjects, the prevalence of incidental findings in all sinuses was 37.5% and they were most common in the maxillary sinus [32]. The prevalence of the maxillary sinus findings among elderly edentulous in previous studies of variable ranges, however figures ranging from 2.6% to 20% have been reported [10,12]. In a study of Soikkonen and Ainomo in 1994 [14], The prevalence of mucous cysts and diffuse mucosal thickening in the maxillary sinuses of elderly edentulous subject was 7% studies of rounded shadows (mucous cysts) in maxillary sinus found in both dentate and edentulous subject with figures ranging from 2% to 13% [12, 3, 13].

Our figures of 4% for the prevalence of mucous retention within that range. According to mattila, [29] the prevalence of mucous cysts is not age-dependant. This was in accordance with our results, where no statically significant difference was found between age groups ($p<0.005$). In studies including younger age groups, maxillary sinus findings have been most prevalent in the third decade and they have also been found to be more prevalent in men [12, 3, 27]. This result is on the contrary with ours, where the findings were more common in the fifth decade of life and comes in accordance with ours regarding the slightly higher percentage in the male group. In the rather wide age-range of the present study old subjects, the number of maxillary sinus findings showed no age-dependent tendencies. The diffuse mucosal thickening, however, were more prevalent in the younger age group, the majority of the diffuse mucosal thickening were found in dentate subject of younger age group. More important (than dental origin) is that allergic sinusitis especially due to dust inhalation especially in this region of Iraq due to sentimental characteristic of the region and it can be suspected that odontogenic causes may not be a major contributing factor in their formation. This result comes in accordance with previous who stated that, the prevalence maxillary sinus findings in sites of periapical or periodontal pathosis and in sites without pathologic findings have also been similar [33]. Neither that findings nor ours support the findings of Halstead in 1973 [20], Who reported that a possible odontognic cause could be indicated in 90% of subjects with maxillary sinus findings. Regarding The diffuse mucosal thickenings, it was reported that those findings always indicate the presence of irritating stimuli, after an infection of dental origin [8,14]. Although our results showed no statistical significant difference ($p<0.005$) between dentate and edentulous patients in relation to the mucosal thickening found in the floor of the sinus. It has been stated that, the chronic apical periodontitis, deep infra-bony pockets are usually unaccompanied by any major subjective symptoms. Their accurate diagnosis may sometimes be vital to the patient, for if the host resistance for same reason, it will give this infection the opportunity to become exacerbated and cause acute sinusitis, whereas the possibility also exists of further spread systemic manifestation [34, 28].

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ANEMIA IN WOMEN DURING REPRODUCTIVE YEARS IN RURAL AREA

Maida Y. Shamdeen[1] *MRCOG, FRCOG*, **Baybeen K.Alselevany** [2] *PhD*.

Abstract

Background: iron deficiency anemia **(IDA)** is a medical and public health problem of prime importance, causing few deaths, but contributing seriously to the weakness and substandard performance of millions of people**.**

Objectives: To determine the prevalence of anemia, 10 years after sanction among women, at reproductive years in rural areas.

Patients & Methods: The study was carried out in September 2002 within field application for university of Mosul on women in reproductive years in Badoosh areas, 20 Km to the North of Mosul city. The study was conducted in rural areas, where 98 women were evaluated clinically, after a questionnaire with 17 items including age, marital status, and social status, number of children, lactation, and menstrual blood loss. A blood sample was taken to evaluate hemoglobin level (Hb), Hematocrit (hct), serum iron level (SI), total iron binding capacity (TIBC), and transferrin saturation (TS).

Results: The mean age of the women with all tests available was 28.75±10.6 years (range15-50 years); the mean number of previous pregnancies in parous women was 5 pregnancies. 58 women were found to be anemic (57.14%). The mean values of their Hb, hct, SI, TIBC and TS in anemic and non anemic group were; (106.8g/l,126.79g/L),(0.32L/L,37.9L/L),(13.53µmol/dl,15.42µmol/L),(69.85µmol/L,62.55 µmol/L) and(19.37%,24.7%) respectively, while the over all results for the same values for all women were 115.4g/L, 0.34L/L, 14.34 µmol/L, 61.01µmol/L and 23.50% respectively. In the anemic group 37 women were married (66.07 %),10 women (17.3%) were lactating, 28women (48.3 %) had more than 4 children, 98 % of the sponsors of the family were workers of low socioeconomic status, 12 (12.3%) married women had heavy menstrual cycle and 84(85.7%) of the families had more than 6 persons in the house.

In the present study the level of Hb was lower and TIBC was higher in anemic as compared to non-anemic patients ($p< 0.05$), while there was no significant difference in the levels of hct, SI, and TS% in anemic patients from that of non-anemic patients ($P>0.05$).

Conclusions: Almost all the anemic women were suffering from iron deficiency (ID) ،which is mainly due to nutritional factors and low socioeconomic status, multiparity, lactation and heavy menstrual loss. This may reflect the effects of the blockade on the nutritional and social status in the rural areas.

Recommendations: For girls ages 12-18 and non-pregnant women of childbearing ages, it is recommended to screen for anemia every 5 years, and annual screen for women with risk factors for iron deficiency anemia, and more frequent in pregnant women. Give iron supplements to all women in reproductive years in rural areas.

Key words: IDA, reproductive years of women life.

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Introduction

The WHO criterion for anemia in women is Hb less than 120 gm/L and less than 110gm/L in pregnant women due to physiological anemia [1- 7]. Anemia may be difficult to define in countries in which malnutrition, infection, high altitude, air pollution and smoke or congenital hematological disorders are common [1-3, 6-12]. The prevalence of ID is 10-15% in pregnant compared to3-4% in non-pregnant women. Flemings et al [13], found that approximately 50% of the anemic women were ID. The signs and symptoms

[1]**Dept.of Obstet & Gynecol, College of Medicine Duhok University,** [2]**Dept. Of Medical Physiology College of Medicine Mosul University Mosul – Iraq**

ddress correspondence to:Dr.BaybeenK.AlselevanyEmail: bselevany@yahoo.comMobile: 0770 161 0242

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of anemia are dependent upon the degree of anemia, as well as the rate at which the anemia has evolved. The history, physical examination, and simple laboratory testing are all useful in evaluating the anemic patient. One or more of the three independent mechanisms can cause anemia: decreased RBC production, increased RBC destruction, and RBC loss [1, 3, 14-15].

The classical presentation of IDA is, multigravid woman in her forties, presents with chronic blood loss from menometrorrhgia, weakness, headache, irritability and varying degrees of fatigue and exercise intolerance, however many patients are asymptomatic and present only with anemia. The Plummer– Vincent or Patterson– Kelly syndrome (dysphasia, esophageal web, and atrophic glossitis), koilonychias, Chlorosis and blue sclera. Pica and pagophagia are specific for ID state; an occasional manifestation of ID is beeturia [15-22]. Reduced absorption of iron and a diet deficient in iron can cause ID [17, 23-28]. Physical examination will show pallor of the palms, nail beds, face or conjunctivae. In developed countries the prevalence of anemia is stated as below 20 %, while in developing countries the prevalence is 40-70 % [3-4, 13].

The manifestations of ID occur in several stages. They are defined by the extent of depletion, first of iron stores and then of iron available for hemoglobin synthesis [14, 20, 25-26].

Laboratory evaluation: the initial testing should include Hb, hct, RBC count and RBC indices. Important discriminating features are low SF and ST, an increased TIBC and low SI, which is excellent indicator of iron store, there appears to be a direct quantitative relationship between the SF and iron stores [23-24,26,27,29-37,39-41].Pregnant women have an elevated serum transferrin in the absence of ID [24,35-37,40-43)]. In severe IDA, SI is reduced and the TIBC is elevated; the latter finding reflects the reciprocal relationship between SI and transferrin gene expression in most nonerythroid cells [35]. The low SI and high TIBC result in a low TS (often less than 10% compared to the normal value of 25-45%)(40,42-43). One problem in pregnancy and oral contraceptives users is increase in the plasma transferrin concentration; as a result, the percent saturation may be low in such patients in the absence of ID [44-45]. Once the diagnosis of anemia due to ID is established, attempts to find out the cause should follow [27, 31, 35, 41, 46-49].

Patients & Methods

A cross-sectional study was conducted in September 2002 on women in reproductive age in Badoosh area 20Km north to Mosul city. Ninety-eight women were selected randomly: almost all in the childbearing age (14-52years), with a mean age of 28.75±10.6 years. Demographic, socioeconomic, menstrual, obstetric, and medical data were collected. Clinical evaluations for symptoms and signs of anemia were done. About 5ml of venous blood was drawn from antecubital vein .The blood sample was divided into two parts: first one ml of blood was added to a tube containing EDTA for the estimation of Hb, and hct. Second, 4ml were put in a clean dry disposable plain tube and centrifuged at 3000 rpm for 15 minutes. The serum obtained was used for estimation of the SI and TIBC, SF was not available to be done .Hb (gm/L) was measured by using cyanomethemoglobinometry, and hct (L/L) was estimated by microhematocrit methods according to Dacie and Lewis (50), SI (µmol/L) and TIBC (µmol/L) were estimated by an enzymatic colorimetric assay (Giesse Diagnostics Kit -Italy), and TS (%) was calculated by the formula; TS% = SI/TIBC X 100.

Statistical analysis was performed using student-unpaired t –test. All values were expressed as mean ± SD. The accepted level of significance was at $P<0.05$.

Results. Evaluation of the results showed that 58 women had low Hb and hct, the prevalence of anemia was 57.14% .The mean age of the women in this study was 28.75± 10.6 years, peak incidence was found in the age group 25-35years as shown in the Figure 1. The non-anemic group was 40 women. The results respectively in the anemic and non- anemic group: concerning marital status, lactation, having more than four offspring or not, and presence of heavy menstrual 37(66.07%), 22(55%) were married, 10 (17.3%), 1(2.5%) were lactating, 28 (48.3%), 10(25%) had more than four children, and 12 (12.3%),6(15%)had heavy loss as shown in (Table1), The sponsor of the families in 99%,98% of cases were workers of low socioeconomic status and (92%),(90%) 0f the families had more than six person in the house (ranging between 6 -20).The distribution of anemia according to the ages is shown in (Figure 1).

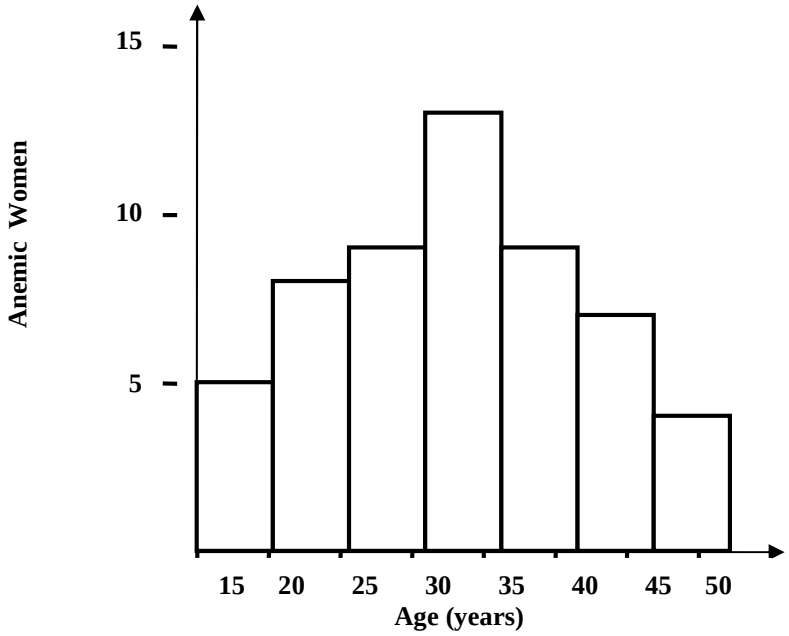

Figure 1: Distribution of anemia according to ages.

The mean number of previous pregnancies, marital status, and lactation, presence of pregnancy, and those with menorrhagia, and others are shown in (Table1).

Table 1: percentage (%) values for anemic, non anemic and mean of both group concerning marital, menstrual and pregnancy statuses.

State	Non-anemic patients n=40	Anemic patients n=58	% Of total
Married	**55%**	**66%**	**60.5%**
Single	**45%**	**34%**	**39,5%**
Lactating	**2.5%**	**17.3%**	**9.9%**

Pregnant	**2.5%**	**7.1%**	**4.8%**
Married with more than 4children	**25%**	**82.4%**	**53.7%**
More than 6person in the family	**97.5%**	**92%**	**94.8%**
Menorrhagea	**15%**	**13%**	**14%**
Low socioeconomic class	**95%**	**98%**	**96.5%**

In the present study the level of Hb is significantly lower and TIBC is significantly higher ($p<0.05$) in anemic as compared to non-anemic patients. The values of hct, SI, and TS % were lower in anemic than in non-anemic patients but these were statistically non-significant as shown in (Table 2).

Table 2: mean± SD of all variables in anemic compared to non- anemic patients.

Variables	Overall group n=98	Non-anemic group n = 40	Anemic group n = 58	p-value
Hb (gm/L)	**115.4± 14.9**	**126.79 ±5.31**	**106.8±14.2***	**0.000**
hct (L/L)	**0.345± 4.45**	**0.379±1.66**	**0.320±4.29** ^{NS}	**0. 934**
SI (µmol/L)	**14.34±6.67**	**15.42± 6.52**	**13.53±6.73** ^{NS}	**0.170**
TIBC(µmol/L)	**61.01±13.94**	**62.55±12.76**	**69.85±14.76***	**0.013**
TS (%)	**23.50±15**	**24.70±11.88**	**19.37±17.1** ^{NS}	**0.091**

NS: non-significant.

***: significant difference** * **p <0.05**

Discussion

The study showed that 57.14% of the women in this locality were anemic; this is compatible with data from other study in developing countries (1-4). The three types of factor responsible for the high prevalence of women anemia in such setting were, iron deficiencies, due to under feeding, consumption of cereal with low iron content, short intervals between pregnancies, and helminthes infestation. Poverty impairs all these factors and limited access to health care and lack of medicine. Although iron and folic acid supplementation are generally recommended, there are numerous economic, cultural and social obstacles to this simple preventive measure [2]. Logistic regression was found that anemia significantly related to the age, socioeconomic status, parity and lactation.

Conclusions

Almost all the anemic women were suffering from iron deficiency, mainly due to nutritional factors and low socioeconomic status, multi parity, lactation and heavy menstrual loss. This may reflect the effects of the sanction on the nutritional and social status in the rural areas.

Recommendations

For girls ages 12-18 and non-pregnant women of child bearing ages, it is recommended to screen for anemia every 5years, and annual screen for women with

risk factors for ID anemia. Supply Iron supplementations for all women in reproductive years of live.

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Effects of AflatoxinB_1 on Some Skeletal Muscle Resident Cells Using a Nuclear Differentiating Stain Technique

May F. Al-Habib *PhD*.

Abstract

Background: Aflatoxins are one of the toxigenic fungi that draw attention for researcher, they are a group of closely related mycotoxins that can contaminate food. The problem of using contaminated food with toxigenic fungi is still one of the most important stigmas in the field of nourishment of human and animals

Objectives: This study was designed to determine how Aflatoxin B_1 contaminated food and feeding regimen might affect and induce specific changes in the muscle resident cells.

Methods: Two groups of animals were studied one fed with Aflatoxin B_1 contaminated food and the other fed with Aflatoxins free diet. Rats were fed daily with diet contaminated with the spore. The Extensor digitorum longus muscle wasremoved and cut into small pieces and prepared by the method of Torikata (1988). Semi thin sections were obtained and stained by a nuclear differentiation stain.

Results: Animals treated with AFB_1 have shown a marked increase in body weight. Aflatoxin B_1 showed pronounced effects on muscle nuclei and on the vascularity of skeletal muscle fibers.

Conclusions: It has been concluded that AFB_1 have marked effects on the number of cells found in skeletal muscles.

Keywords: Aflatoxins B1- Skeletal muscle- nuclear differentiating stain

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INTRODUCTION

Normal mature skeletal muscle is among a growing list of tissue and organs now known to contain rich resident population of different types of cells and especially mononuclear phagocytic cells. Two groups of cells were observed:

Endogenous myonuclei: These are seen within muscle fibers, they include satellite cells and myonucli exogenous cells: These include cells seen outside the muscle,

1. Blood mononuclear cells:
Including monocytes and lymphocytes.
2. Macrophages.
3. Others: include dendritic cells, fibroblasts, and vessel related cells [1].

Satellite cells are a small population of morphologically undifferentiated cells located between the external lamina and sarcolemma of uninjured muscle fiber [2]. They are probably derived from embryonic myoblasts. During postnatal muscle growth they fuse with their adjacent growing myofibers resulting in an increase in the number of nuclei [2&3].

A considerable increase in the number of nuclei during muscle growth in rats was noticed by many researchers, further radiography with 3H-thymidin has shown that some nuclei located within the basement membrane of the muscle fiber have mitotic figures [4].

Dept. histology and embryology,Al Nahrain College of medicine
Address Correspondence to Dr. May F. AL-Habib
P.O.Box 14222
Baghdad/ Iraq
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There are certain powerful biological toxins called myonecrotic agents that can affect muscles. Clostridial toxins, found to destroy connective tissue with muscle fibers necrosis [5]. Fatty degeneration in cardiac muscle was also recorded after the administration of diphtheria toxins to guinea pigs [6]. Snake toxins cause skeletal muscle degeneration and subsequent regeneration when injected into the rats [7].

Aflatoxins are group of closely related mycotoxins that are widely distributed in the nature in different agricultural comities produced by Aspergillus flavors group of fungi [8]. It causes great economic losses and health hazards both to human and farm animals. The most important group of Aflatoxins produced by this type of fungi is B_1 (AFB), which have a very wide range of biological activities [9].

The problem of using contaminated food with toxigenic fungi is still one of the most important stigmas in the field of nourishment of human and animals. These toxigenic fungi are able to produce secondary metabolites that may produce a toxic biological effect [10].

Acute exposure may not reflect the exposure pattern of individual whose diet may contain Aflatoxin contaminated foodstuff. Low-level exposure to AFB_1 may present health risk where it was found to impair specific and non-specific immune responses [11&12].

Aflatoxin B_1 is a known hepatocarcinogen. Several investigations have shown the serious effects of Aflatoxins on liver, lymphocytes, macrophages, and lung [8, 13, and 14].

However, Studies of its effects on muscle have not been taken in consideration. In an attempt to analyze the relationship between some muscle resident cells and the effects ofAFB1 on them this study was designed.

MATERIAL AND METHODS:

1. Isolation of fungi

The Aflatoxins producing fungi were isolated from seed samples (rice, peanut and wheat) according to method of Shotwell et al. in the Department of Technical Biology, College of Science, Al - Nahrain University [15]. The fungi isolates were identified by direct examination with light microscope using lacto phenol stain

2. Spore suspension preparation

Slants containing Czapek's dox agar medium were inoculated with the isolation of A. parasiticus then the slants were incubated at 30 °C for 7 days and kept under 5 °C in the refrigerator. Spore suspensions were prepared according to Faraj method [16]

3. Laboratory animals.

Mature albino rats were used in this study. Animals were isolated in a relatively controlled environment at a temperature of about 37 °C. They were given free access of tap water and food. The albino rats were divided into 2 groups (4 rats for each age group) as follows:

a. Group I

Rats which were fed daily 25 gm of the diet for 30 days considered as a control group.

b. Group II

A pilot study was done before starting this experiment using different doses of diet contaminated with the spore of isolated A. parasiticus. Rats were fed daily with diet contaminated with the spore of isolated A. parasiticus 200 mg/Kg of body weight for 30 days. At the end of the treatment, all animals were killed by spinal dislocation and dissected. The Extensor digitorum longus muscle was removed and cut into small pieces (1 mm x 1 mm x 1 mm).

4. Tissue Preparation for semi thin sections:

The method of Torikata (1988) [17] was employed. Tissue blocks were fixed for 3 hours in 2.5% gluteraldehyde in phosphate buffer (pH 7.2) with tannic acid. Tissue blocks were then washed with the phosphate buffer 3-4 times and left in the buffer for 12 hours.

Specimens were fixed with 1%osmium tetraoxide for one hour and dehydrated then transferred to propylene

oxide for 20 minutes. Blocks were then passed to a mixture of propylene oxide and araldite for one hour, left in araldite for 12 hours at room temperature. All pieces were

Cleaned by filter paper and placed in a plastic capsule. The capsule filled with araldite was then transferred to an oven at 60°C for 48 hours. The capsule was left for 1-2 days at room temperature to be ready for sectioning.

Glass knives were made by (LKB) knife maker then, tissue blocks were cut using this knife in an electrical ultramicrotome. Semi-thin sections 0.5-1 \µ were obtained

5. Nuclear Differentiation Special Stain:

Semi thin sections were placed on glass slides heated to 60°C and stained with 2 solutions [18].

Solution A: This was prepared by adding 0.4% basic fuchsin to 25% methanol.

Solution B: This was Prepared by mixing equal volumes of 1% azure II in distilled water, 1% Methylene blue in distilled water, 5% Na2Co3 in distilled water, Absolute methyl alcohol. Resulting solution was diluted to half with distilled water

6. Staining technique:

The specimens were stained with solution (A) for 3 minutes on a hot plate to 54°C, and then it was washed with distilled water. Staining with solution (B) was done for15 seconds on the hot plate and rinsed well with distilled water. If the stain is too weak, repeat staining for an additional 15 seconds

The slides were then air dried and mounted with synthetic resin. Cell counting was done by using measuring graticule eyepiece. Twenty semi thin sections from the experimental and the control groups were examined.

7.Counting of Resident cells, blood vessels

Sections stained with nuclear differentiation stain were examined by selection of a field in each section, in which counting of resident cells was done by defining each type of cell depending on it's characteristic features as described by

(Ontell, 1974). An eyepiece graticule of a single lattice pattern of (X10) magnification was used for this counting method with a field area of (0.75) mm^2 divided into 100 equal squares. Counting was done by systematic scanning of the whole 100 squares, and counting them at X100 magnification (oil immersion) in Reichert-Jung Diastar photomicroscope. ANOVA (single factor) test was applied for the resident cell, and the mean values and P-value were calculated for each of them [19].

Results

Animals treated with AFB_1 have shown a marked increase in body weight from (303.5+ SE 133 - 363.5+ SE 126) increase in the body weight of animals treated with Aflatoxin B_1.

Nuclear differentiation stain used in this study was capable of differentiating all types described to be seen in normal muscle were clearly identified (figure 1&2). The over all number of nuclei was markedly decreased from (35.0 +SE 4.6 to 25.5+ SE 4.4).

Myonucli seems to be not affected (figure3) but, the numbers of satellite cells and fibroblasts seems to be markedly affected (figure3). Fibroblasts are resident cells located out side the muscle fibers within the connective tissue compartments they show a significant difference in their distribution between treated and non treated animals with (P-value < 0.001) (table1). The amount of connective tissue seen in between muscles can be noticed to be more and loose (figure 3)

In addition to the resident cells, enumeration of the blood vessels per fixed field area was done on the semithin sections that stained with NDS, there was a significant difference in their distribution between treated and non treated animals as seen in (figure 2&3) with (P-value <0.001) (table1).

Table (1): Shows the mean distribution of the some skeletal muscle resident cells, blood vessels in treated and non treated groups, with their P-values.

	Control	Treated	P value
Myonuclei	19	27	<0.001
Satellite cells	9	3	<0.001
Fibroblasts	3	12	<0.001
capilleries	6	1	<0.001

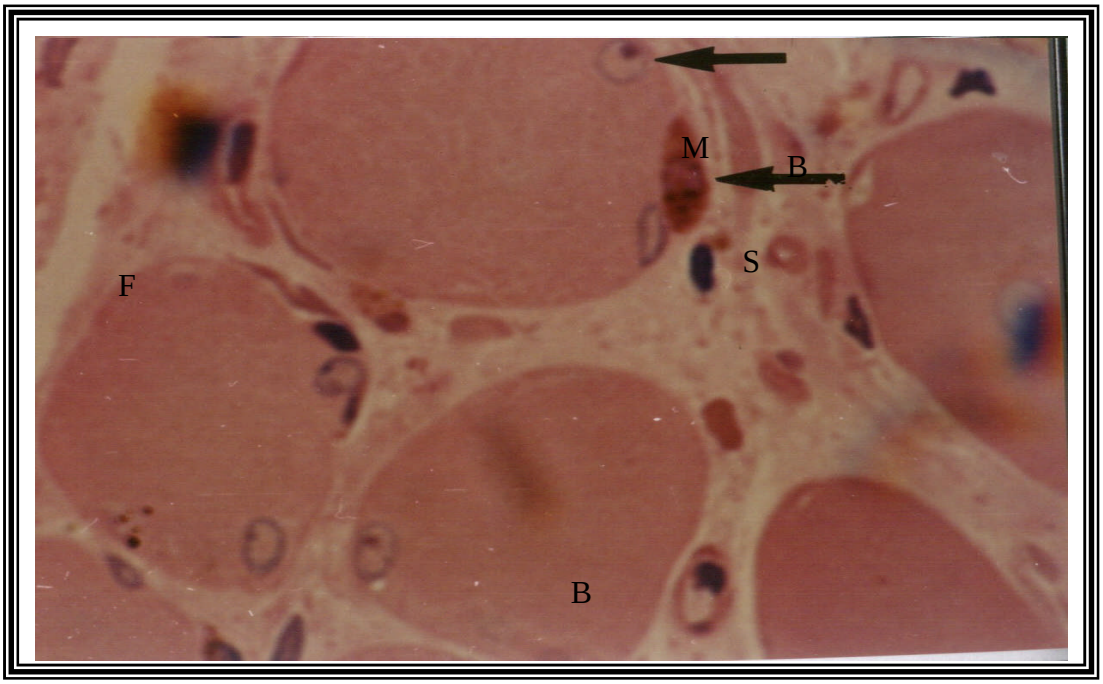

Figure (1): Multiple forms of nuclei in non treated animals. S: satellite cells; M: myonuclei; B: blood vessels; F: fibroblasts
(Nuclear differentiating stain, X225).

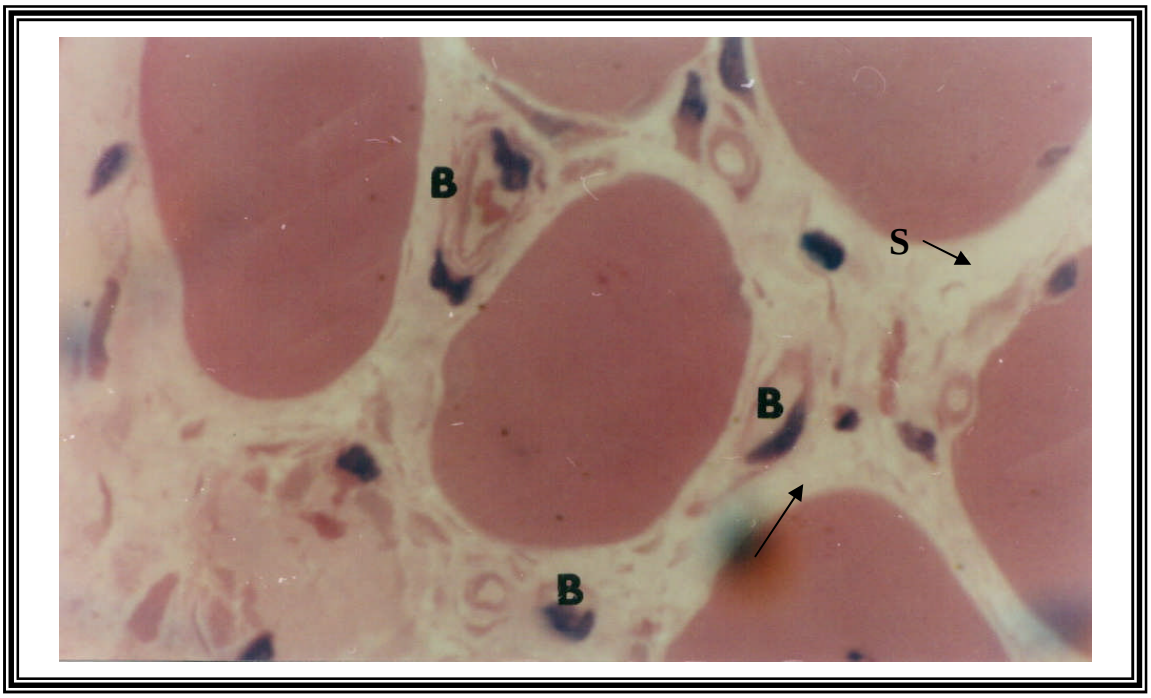

Figure (2): Rich vascularity in non treated animals S: satellite cells. (Nuclear differentiating stain, X320).

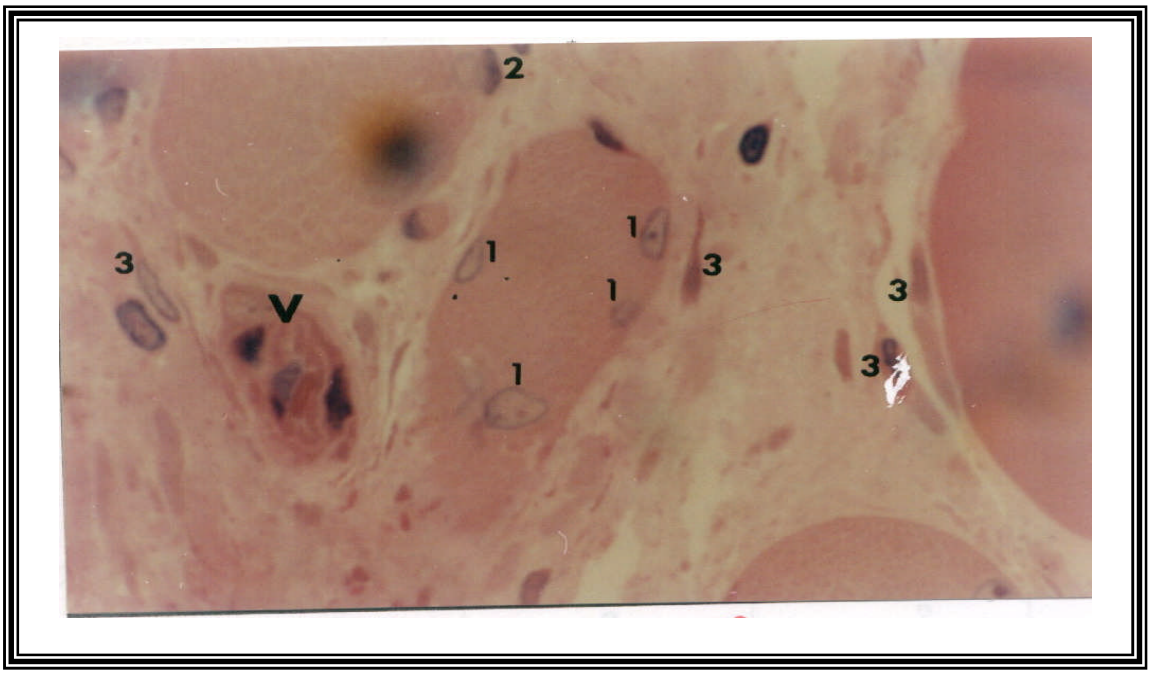

Figure (3): Treated animal with marked decrease in cellularity and vascularity. 1: Myonuclei, 2: Satellite cells, 3: fibroblasts, V: blood vessels. (Nuclear differentiating stain, X320).

Discussion:

Aflatoxin B_1 is a potent carcinogen produced by certain Aspergillus species. Several investigations have shown serious effects of AFB_1 on many organs like liver, lung, spleen, lymphocytes and immune system [11].

The amount AFB_1 contaminated diet that might produce toxicity differs according to the type of tissue we are studying and also differs according to the animal species [20]. We have found that the dose200 mg/Kg of body weight for 30 days have important effects on skeletal muscle.

In this study animals treated with AFB_1 have shown an increase in total body weight which might be due to increase of water intake that have been noticed during this experiment and swelling of some organs after treatment as a reaction for the effect of Aflatoxin B_1 which perhaps have stimulated the thirst center in the rats resulting in an increase in water consumption as an attempt to assist in the excretion in the body metabolism [21].Gain in body weight was recorded in aflatoxins treated animals, it was observed in turkey, poults and rabbits[22]

Nuclear differentiation stain used in this study was capable of differentiating cell types that are difficult to be identified using H &E stain.

It seems that the number of myonuclei was markedly affected. There was an increase in the number of myonuclei in treated animals with a P value <0.001 . The number of satellite cells was markedly decreased with P<0.001 value of. Satellite cell nuclei can be easily identified based on the criteria of proximity to its principle muscle fiber, chromatin density exceeding that of adjacent myonuclei and, the presence of a space or halo between the nuclei and its muscle fiber [23].

In this study the satellite cells show a significant reduction, while the myonuclei showed a marked increase. This suggests that there is an apparent reciprocal relationship between the number of satellite cells and myonuclei. Satellite cells produce myonuclei by mitosis, which are then added continuously to the post mitotic pool of myonuclei, they divided repeatedly in young rats and function as the source of true muscle nuclei [24].It seems that Aflatoxin B_1 might cause some sort of injury to the muscle, this will stimulate satellite cell to proliferate in response to injury to give rise to regenerated muscle

The overall decrease in the number of vessels include areas of focal myofibril disorganization, seems to be a feature of necrosis, the vascularity was significantly decreased in treated animals (P< 0.001). It seems that Aflatoxins induces a form of ischemia and ischemic muscle fibers become necrotic and die [25].This might be due to the deficit and impairment of O_2 exchange within skeletal muscle of senescent individuals due to decrease in the number of capillaries.

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IMATINIB MESYLATE IN IRAQI PATIENTS WITH CHRONIC MYELOID LEUKEMIA

Nabeel S. Murad *FRCP*, **Ali M. Al Ameri** *CABM*.

Abstract:

Background: Chronic myeloid leukemia (CML) is a clonal proliferation of stem cells that is characterized by granulocytosis with granulocytic immaturity. The molecular abnormality involving the ABL gene on chromosome 9 and the BCR gene on chromosome 22 have been established as being the proximate cause of chronic phase CML.

Objective: To study the clinical, and hematological responses to imatinib mesylate and the main side effects in Iraqi patients with CML in the three phases of disease.

Methods: Three hundred and sixty two patients with CML were enrolled .they were diagnosed by peripheral blood and bone marrow aspirate examination and were treated with imatinib mesylate 400 mg/day as one single dose orally and followed up every 4 weeks for clinical , hematological responses and evaluation of side effects.

Results: The frequency of CML cases by residence was 17. 40%, 21.8% and 61.6% from south, north and middle regions of Iraq respectively. The age of patients ranged 14-70 years, 192 males (53%) and 170 females (47%). Complete clinical and hematological responses were observed in 325 (90%) of patients within 3 months from the initiation of imatinib in the chronic phase of the disease, only 4/10 responded in the accelerated phase at higher dosage of 600-800mg/day , no one in the blastic phase responded. Side effects were generally mild and tolerable.

Conclusion: Imatinib mesylate is effective and safe in achieving high clinical and hematological responses in chronic phase CML patients, but has poor response in accelerated and acute blastic phases. Side effects are generally mild.

Key words: Chronic Myeloid Leukemia, Imatinib Myseglate

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Introduction

Chronic myeloid leukemia (CML) is a clonal proliferation of the stem cell, that is characterized by anemia,extreme peripheral blood granulocytosis and granulocytic immaturity, basophilia, often thrombocytosis and splenomegaly. The hematopoietic cells contain a reciprocal translocation between chromosome 9and 22 in over 90% of patients which leads to an overtly short, long arm of chromosome 22, referred to as Philadelphia (Ph1) chromosome(22q-) [1]. A rearrangement of the break point cluster region on chr.22 is probably present in all patients with CML and the molecular abnormality involving theABL gene on chr. 9 and the BCR gene on chr. 22 have been established as being the proximate cause of chronic phase CML [1,2].

The disease has a very high propensity to evolve into an accelerated or acute fatal phase resembling acute leukemia. The incidence in USA is 1-2 patients in 100000 of population [2]. Until recently the only treatment choices were, stem cell transplantation which, though curative, is limited to a small proportion of patients with CML, and hydroxyurea-based , or interferon -alfa (IFN) based regimen [4, 5].

Treatment with IFN has a deleterious effect on patients quality of life and is associated with physical toxicities as fever

[1]**Dept. Medicine, College of Medicine, Al-Nahrain University** [2]**Dept. Medicine, College of Medicine, Al-Mustansiriya University.**

Address correspondence to Dr. Nabeel S. Murad, Email : nabeelmurad@yahoo.com

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and chills, hypotension and fatigue, impaired memory and inability to concentrate [4-7]. Hydroxyurea is well tolerated but is of limited efficacy with no effect on disease progression or survival [8].

Imatinib mesylate is an oral targeted therapy, a selective Bcr- Abl tyrosine kinase inhibitor with significant activity in the treatment of Ph positive CML and in Ph +ALL patients [9]. In clinical trials [10-13], imatinib has demonstrated a high level of efficacy, clinically and hematologically in the three phases of the disease and is associated with significantly less toxicity, which is likely to translate into quality of life benefit and survival advantage [14].

In patients with CML chronic phase –(post IFN alfa failure), imatinib induced complete cytogenetic response in 48% and major cytogenetic response (Ph chromosome less than 35%) in 65% of patients. The two year transformation rate was 13% and the two year survival rate was 92%[10, 14].

In the International Randomized Study (IRIS) [13], comparing IFN and low dose Ara-c, versus imatinib in patients with newly diagnosed CML in chronic phase, Imatinib was associated with significantly better 18 months rate of complete cytogenetic response (7 versus 14%), respectively. In the most recent study, imatinib versus other therapies, imatinib was a significant independent favorable prognostic factor for survival [14].

Aim of this work

Is to study the clinical and hematological responses to, and the side effects of Imatinib in Iraqi patients with chronic myeloid leukemia in the three phases of the Disease and to highlight some aspects of the epidemiology of this disease in this country.

Patients and methods

Late in 2002, a committee was assigned by the Ministry of Health to help delivering imatinib (glivec) to Iraqi CML patients and the National Center for the Treatment of Blood Disorders (at Al-Mustansiriya University) was chosen for prescribing and dispensing this drug agent. At the time of starting writing the results of this work we had already seen 362 patients with CML who were diagnosed on clinical and hematological grounds by experienced physicians and hematologists.

Cytogenetics was unfortunately not performed because of technical difficulties. They were 53% males and 47% females with an age range of 14-70 years. Full investigations were performed for each including, CBC and ESR, Bone marrow aspirate, FBS and BU, uric acid and hepatic transaminases. There were no clear-cut exclusion criteria in this pilot study except patients with advanced organ failure.

The dose of imatinib was 400 mg to be taken orally in one single dose preferably after breakfast. Patients were instructed to attend every 4 weeks and report on a special sheet, their subjective body responses, daily activity and side effects, and to undergo careful physical examination for splenic size, jaundice, edema or any skin reaction, also to have their peripheral blood examined for Hemoglobin, WBC and differential count and platelet count to assess the hematological response and the disease phase.

Statistics

Parameters were represented as means& percentages on the figures.

Results

The prevalence of the CML in Iraq is about 2/100000, assuming the population is 25 millions and the No. of CML patients in mid 2005 was approaching 560 in the (NCH) center. Figure 1 shows that 51% of patients have an age range between 30-49 years and around 30% of the total were younger than 30 years.

Figure 2 shows no obvious difference in sex distribution, 53.07% males and 46.9% females. Figure 3 shows the distribution

according to the geographical area. North of Iraq: 21.9%, Middle 61.6% and the South 17.4%. Figure 4 shows the relationship to occupation, ordinary laborers were the commonest class and the farmers least affected! Figure 5 shows the Hb level before therapy. Figure 6 shows the WBC count 3 months after therapy. Figure 7 shows the response in accelerated phase. Figure 8 demonstrates the response in blastic phase.

Ten patients were in accelerated phase, four of them reverted to chronic phase on higher dose imatinib therapy. Six patients were in acute blastic phase and showed no response.

Table 1 shows the distribution of registered side effects of Imatinib in all patients treated. Side effects were generally mild and tolerable. Of the non hematological: muscle and joint pain seen in 325 patients (90%), nausea and indigestion in 304 patients (84%), peri orbital swelling and weight gain in 144 patients (40%). Of the hematological side effects. granulocytopenia grade 1,2: in 100 patients (30%), grade 3,4: in 28 patients (8%) and thrombocytopenia Grade 1,2: in 72 patients (20%), Grade 3,4: in 18 patients (5%).

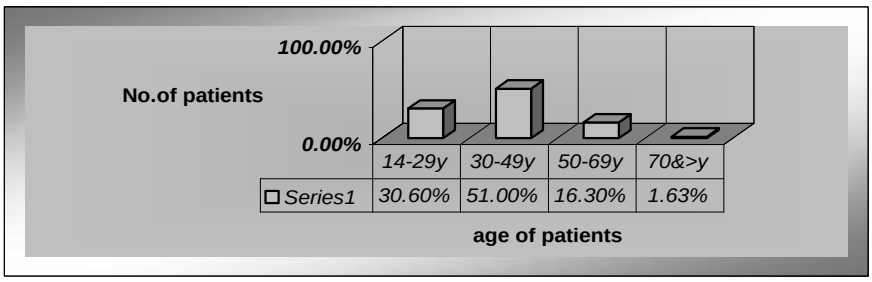

Figure 1: Age Distribution

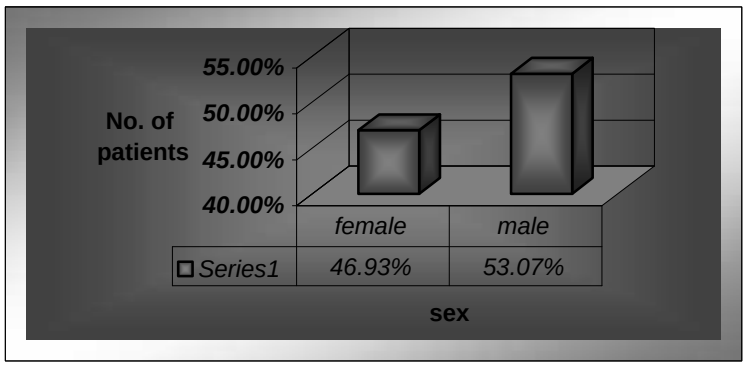

Figure 2: Sex distribution

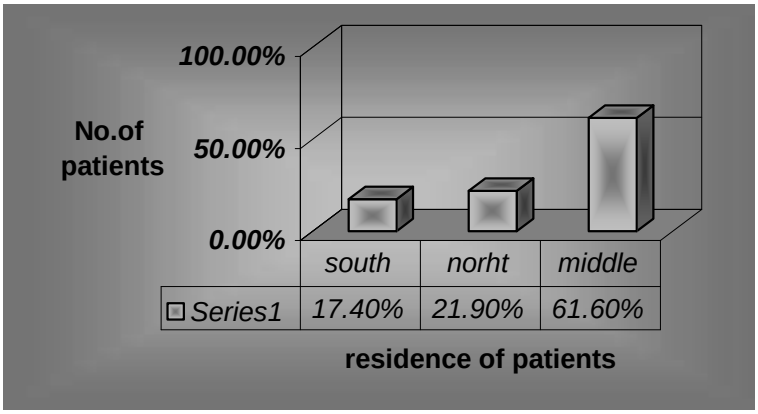

Figure 3: Residence

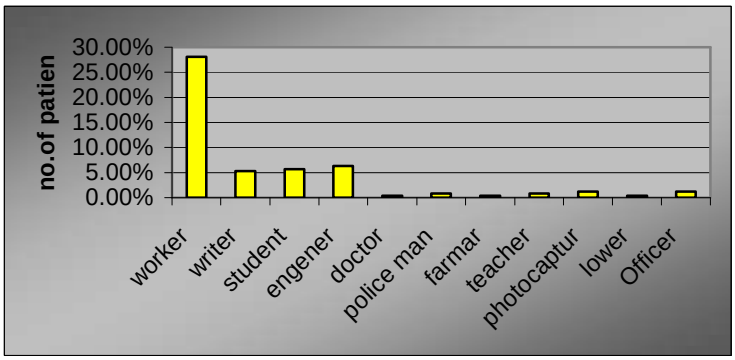

Figure 4: Occupation Of Patients

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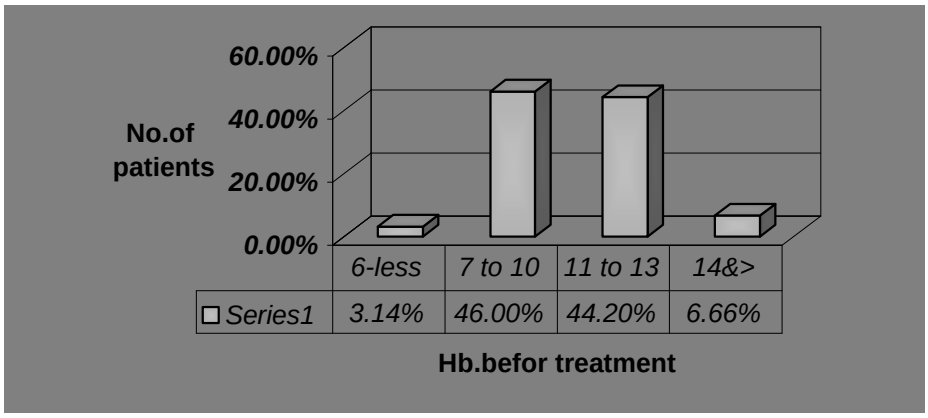

Figure 5: Hb concentration at presentation

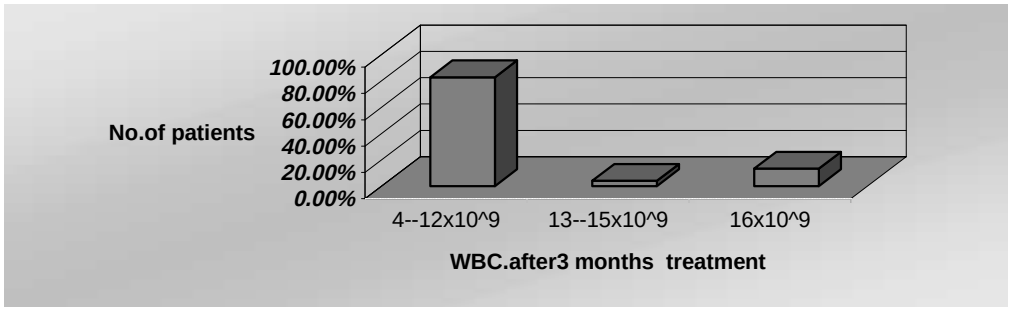

Figure 6 :WBC count after 3 months of treatment

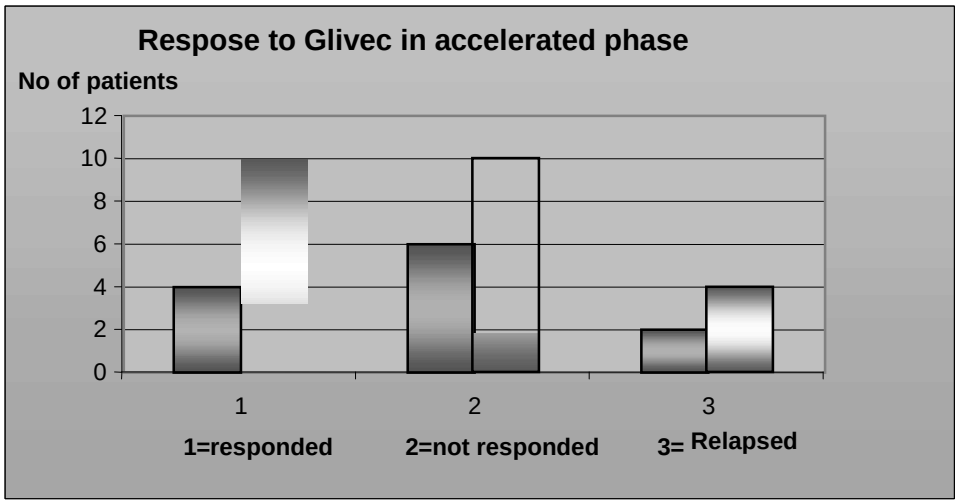

Figure 7: Response in accelerated phase

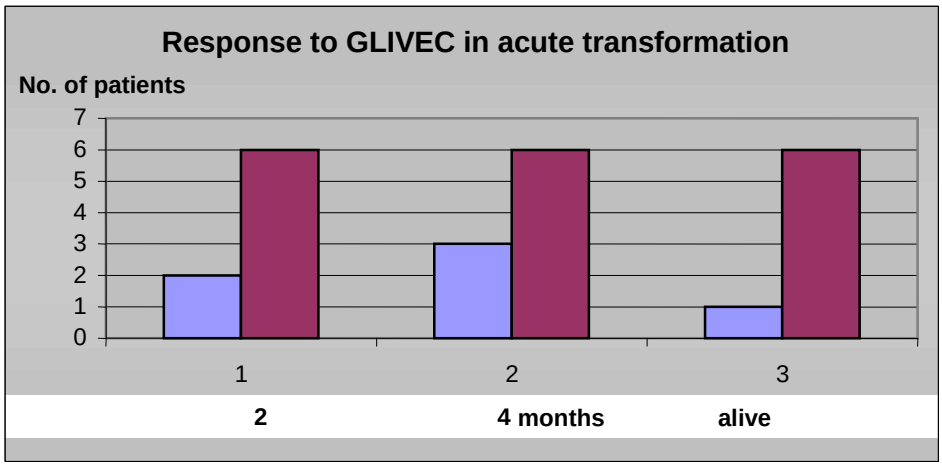

Figure 8: Response in blastic phase

Table 1: Side Effects of Imatinib

Side Effects	Very Common (› 1/10)	Common (› 1/100 ‹ 1 / 10)	Uncommon (‹ 1 /100 › 1/ 1000)
Infections			
Sepsis, pneumonia			+
H zoster & simplex			+
CNS			
Headache			+
Dizziness, Parasthesia, Epileptic fits			+
Eye			
Conjunctivitis			+
blurred vision		+	
Dry eye			+
periorbital swelling		+	
Ear			
Vertigo & Deafness			+
tinnitus			+

Cardiovascular			
hypertension, hypotension			+
palpitation, chest pain, dyspnoea			+
Gastro intestinal			
Nausea, dyspepsia	+	+	
vomiting			
Diarrhea & flatulence & constipation			+
Hepatic			
jaundice			+
increased SGPT & ALP			+
Skin			+
Dermatitis		+	
Facial oedema &eyelid oedema	+		
dryness & photosensitivity			
Musculo skeletal			
muscle pain & cramps	+		
joint swelling			+
bone pain, sciatica			+
Genitourinary			
renal insufficiency			+
gynaecomostia			+
menstrual disturbance			+
polyuria & polydepsia			+
Blood			
mild Neutropenia	+		
mild thrombocytopenia	+		
pyrexia & Rigor			+
Weight gain			
Early		+	
Later	+		

Discussion

The prevalence of CML in Iraq is approaching 2/100000 which is nearly the maximum figure found in the literature, 1-2/100000[1,2] By April 2005 the number of patients in the NCH rose to 545. Has the disease increasing in frequency or incidence in our country?. The answer may be (yes) in view of the environmental pollution which the country had been exposed to, over the past two decades and the hot issue of the depleted uranium and its adverse radiation effect. Another point is that chemicals and insecticides do not seem to be a causative agent for CML due to the emerging observation that farmers had the lowest rate of prevalence despite heavy exposure to these agents, this is also compatible with the literature that, chemicals or insecticides are not incriminated in CML development[1, 2].

Chronic myeloid leukemia is a killing disease in 3-6 years by progressing into accelerated or acute phase. Of the 362 patients enrolled in this study 289(80%) are alive and in hematological remission at the time of reporting i.e., two and a half years from the start of imatinib.

This is consistence with the results of several previous cohorts in the world[8-11,14]. With regard to the age, about 30%of patients were under the age of 30 years and 81% under the age of 49 years, this may give an impression that CML is not a disease of middle aged population, but probably if a larger number of patients were enrolled it would have shown different results.

Twenty patients developed accelerated or blastic transformation during the past two years after they were

previously in remission on imatinib, most of them have had the disease for more than 4 years. This may be attributed to the development of resistance perhaps due to a mutation at the BCR-ABL that reduce the binding affinity of imatinib as it has been reported lately [15].

Side effects were mild in the majority of our patients, both the hematological and the non hematological which is consistent with the results of several previous studies[16]

Conclusion

Imatinib mesylate induces good and durable clinical and hematological response in patients with chronic phase CML with acceptable mild side effects. Patients with accelerated phase and those in blastic crises showed poor response.

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HETEROTOPIC PREGNANCY: A CASE REPORT

Liqaa R. AL-Khuzai *FICMS.*

Abstract:

The incidence 0f heterotopic pregnancy increased in the recent years with wide spread of ovulation induction drugs and assisted reproduction techniques. There is delay in the diagnosis of heterotopic pregnancy and about 50% of patients are admitted for emergency surgery following rupture. Early diagnosis and treatment of heterotopic pregnancy lead to decrease maternal mortality, morbidity, and salvage of intrauterine pregnancy.

Key words: Heterotopic pregnancy

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Clinical History

A 29 years old woman gravida 2 Para 0 abortion 1 conceived after ovulation induction using clomiphine citrate; her last menstrual period (LMP) was on the first of October. She was admitted as a case of acute abdominal pain of 2 days duration on 23.12.2004; she was 12 weeks pregnant. The pain was all over the abdomen with radiation to both shoulders. She consulted two hospitals before she came to our hospital, where she received an intravenous fluid and analgesics and discharged home. The pain increased in severity, and her general condition was deteriorating in the last day. She was attending a private doctor who did for her3-ultrasound examinations were normal except for the diagnosis of cervical incompetence for which cervical cerclage have been done.

On admission, the patient was severely pale, her rate was100 pulse per minute BP 110/50. There was a generalized abdominal distention with tenderness all over the abdomen; vaginal examination performed which revealed bulky uterus 12 weeks size and fullness in the pouch of Douglas. Immediate expletory laparotomy was performed revealed haemoperitoneum (abdomen filled with blood), ruptured chronic left ampullary ectopic pregnancy that was also involving the left ovary. The right tube was edematous; right ovary was normal.

Uterus was 12 weeks size. Left salpingectomy was performed. Hemostasis secured, cleaning of the abdominal cavity from blood; estimated blood loss was three liters. The fetus was found floating in the abdominal cavity. Tube drain inserted in the left iliac fossa. Patient received five units of blood and one unit plasma. In her second postoperative day, there was incomplete abortion of the intrauterine fetus followed by curettage under general anesthesia and removal of placenta. The postoperative period was smooth .She was discharged home on her third postoperative day.

In this case report I present a case of heterotopic pregnancy complicated by rupture with review of literature.

Discussion

Heterotopic pregnancy (HP) is the coexistence of an intrauterine pregnancy and ectopic pregnancy. In 1948, the spontaneous HP rate was calculated as one in 30,000[1]

Dept. Obstetric & Gynecology, College of Medicine, Al-Nahrain University
Address correspondence to Dr. Liqaa R. Al-Kuzai, e.mail: dr_liqaa@yahoo.com
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pregnancies. Today HP actually occurs one in 3889 to 1 in 6778 pregnancies [2]. The increased incidence of HP is a consequence of assisted reproduction and the wider use of ovulation induction agents. The diagnosis of HP is frequently done not as earlier as it should be and it has serious repercussions. Delay in the diagnosis is because of visualization of intrauterine gestational sac.

The HP in our case was associated with the use of clomiphine citrate an ovulation induction drug, there are many case reports about this association [3-5].The fetomaternal prognosis can be improved by early diagnosis. There is a need to maintain a high index of suspicion and to intervene early to salvage the intrauterine pregnancy and prevent maternal mortality and morbidity associated with ectopic pregnancy. Treatment of HP pregnancy is surgical by salpingectomy done through laparotomy or laparoscopically and there are case reports of salvage the intrauterine pregnancy that continued to term without complications [6-9].

There are two case reports of ultrasound-guided transvaginal injection of potassium chloride or hyperosmolar glucose in to the abdominal pregnancy resulting in a systole and spontaneous resorption of the ectopic fetus while the intrauterine pregnancy continued and resulted in alive delivery at full term [10, 11].

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Brugada Syndrome, A case Report

Mohamaad Hashim[1] *CABM,***Tahseen AL-Kinani**[1] *CABM,* **Kamil Namiq**[1] *MBChB,***Amar AL-Hamdi**[2] *FRCP(Ed),* **Kais Al-Mudares**[3] *MBChB*

Introduction:

Brugada syndrome is a clinical and electrocardiographic diagnosis based on syncopal sudden death episodes in patients with a structurally normal heart and characteristic ECG pattern composed of right bundle branch block (RBBB) and a specific shape ST-segment elevation in V1 to V3 [2].

The first report on this syndrome was published in 1992, although some reports on a similar condition has been reported since 1989, since 1992 there has been an exponential increase in the number of patients recognized all over the world [(9)]. Its incidence and prevalence are difficult to estimate, however asymptomatic subjects with Brugada type ST-segment shift were present at a rate of 0.14% in the general Japanese population [1, 3].

Key words: Brugada Syndrome, Ventricular arrhythmia, sudden cardiac death.

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Patient characteristics:

A 15 years old Iraqi boy presented with history of recurrent syncopal attacks of seven years duration. He gave a strong family history of sudden death; two of his brothers died suddenly without previous complaints, one at the age of 16 years and the other at the age of 12 years. No one of his remaining family (two sisters, three brothers and his father) is symptomatic. His mother died due to obstetric problem.

Thorough examination of the patient was essentially normal. His ECG showed sinus rhythm, PR interval of 230 msec., RBBB pattern with a QRS width of 84 msec. and a specific Brugada type ST-segment elevation in V1-V3 (Figure 1). Holter monitor showed a varying ST segment shift over the 24 hours of the recording (Figure 2).

Echocardiogram was normal. EEG, complete blood count, blood biochemistry, serum electrolyte and chest X-ray are all within normal.

A diagnosis of Brugada syndrome was made and an Implantable Cardioverter Defibrillator (ICD) Ventack Prizm II VR,Guidant, St Paul ,MN. With a defibrillator lead: Endotak reliance from Guidant was implanted.

An ECG screening for the rest of the family showed no similar abnormalities in any other member.

Discussion:

Brugada syndrome is usually identified as sporadic cases. However 50% of individuals who have this syndrome have a family history of the disease [1].

Genetic mutations composed of abnormalities in SCN 5 A gene results in abnormalities in the cardiac sodium channels and those patients are predisposed to rapid polymorphic ventricular tachycardia (VT) or ventricular fibrillation (VF).

In our patient the 12 lead ECG (Figure 1) showed a prolonged PR interval, which has been reported in 18% of patients with Brugada syndrome

[5]. It also showed a RBBB pattern with QRS width of 84msc. and a specific shaped ST-segment elevation in V1-V3.

The Holter study showed a long short cycle length, which could precede the occurrence of polymorphic VT in few cases of Brugada syndrome. The diurnal variation in the degree of ST-segment elevation in the same lead has been noticed in our patient [1].

We proceeded to ICD implantation directly without EPS because a negative EPS will not change our decision for ICD implantation in this high risk patient for sudden cardiac death (SCD) as symptomatic Brugada patients require protective treatment from SCD even when the VT are not inducible during EPS [6].

In this type of symptomatic patient with syncope and classical ECG pattern the risk of new arrhythmic event is estimated to be 19% within 54 months [7].

The rest of the family of this patient were asymptomatic and the 12 lead ECG for all of them showed no abnormal ECG pattern, procainamide challenge [8] for the family members were done and no abnormality appeared in the ECG after challenge.

In this syndrome there are few predictors of events occurrence:

1. A spontaneously abnormal ECG is a marker of possible sudden arrhythmic death in comparison to those who had abnormal ECG after drug challenge [8].
2. Male sex is considered as a risk factor for SCD as compared to female [1].
3. The inducibility of sustained VT during EPS, which may be the strongest marker of prognosis [1].
4. Symptomatic patients have unacceptably high rate of arrhythmic events which are more frequent in patients who present with aborted SCD compared to patients who present with repetitive syncopal episodes [9].

No effective antiarrhythmic drug is available [1, 9]. ICD is indicated in symptomatic patients, however the group of asymptomatic individuals in whom the abnormal ECG was recognized only after drug challenge and they have very low event rate during follow up warrants no treatment [9].

As far as we know this is the first case of Brugada syndrome diagnosed in Iraq, we hope that this case report will make physicians and cardiologist oriented about this condition.

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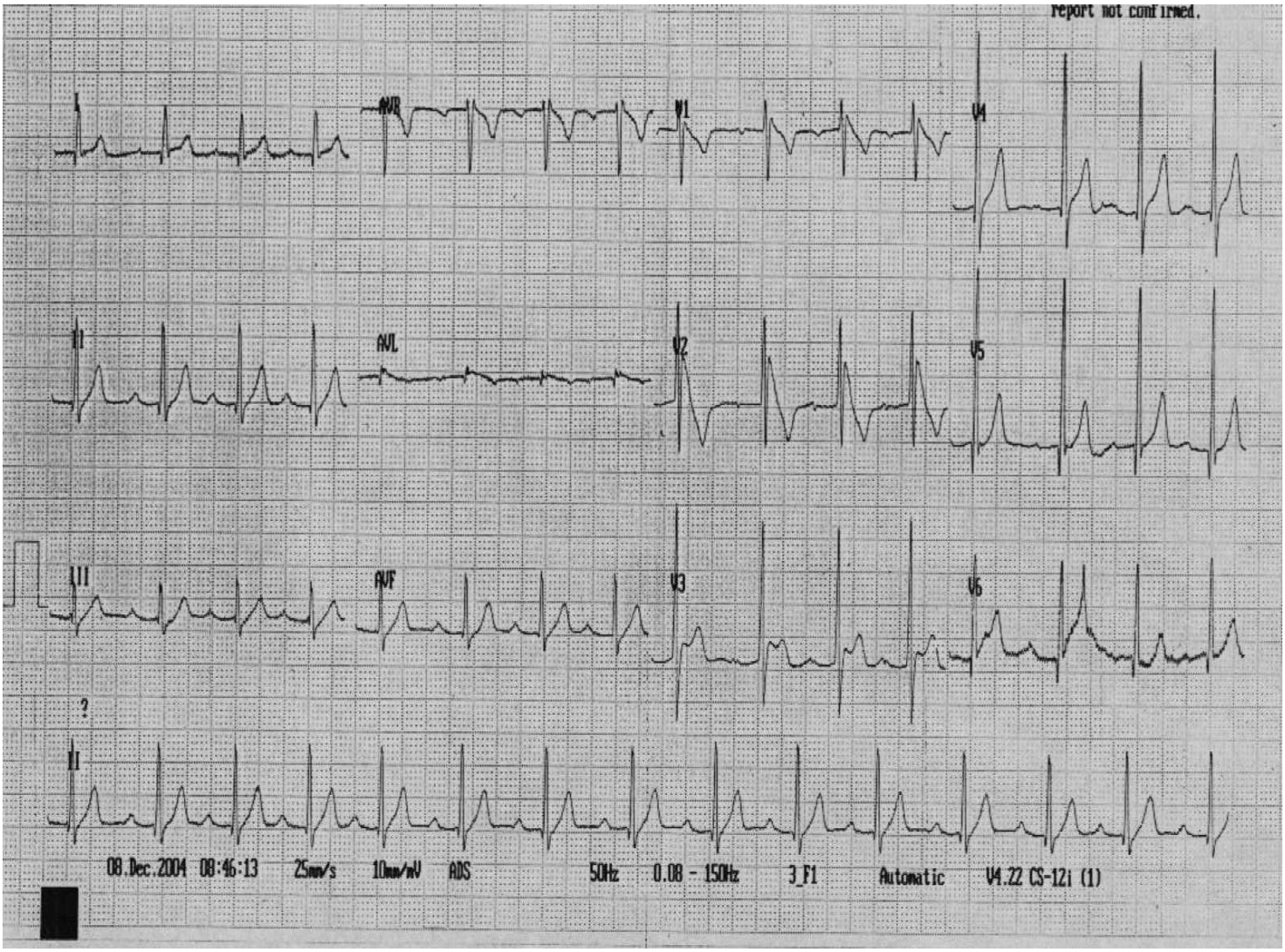

FIGURE 1. 12 LEAD ECG SHOWING TYPICAL COVED ST ELEVATION IN V1,V2,V3.

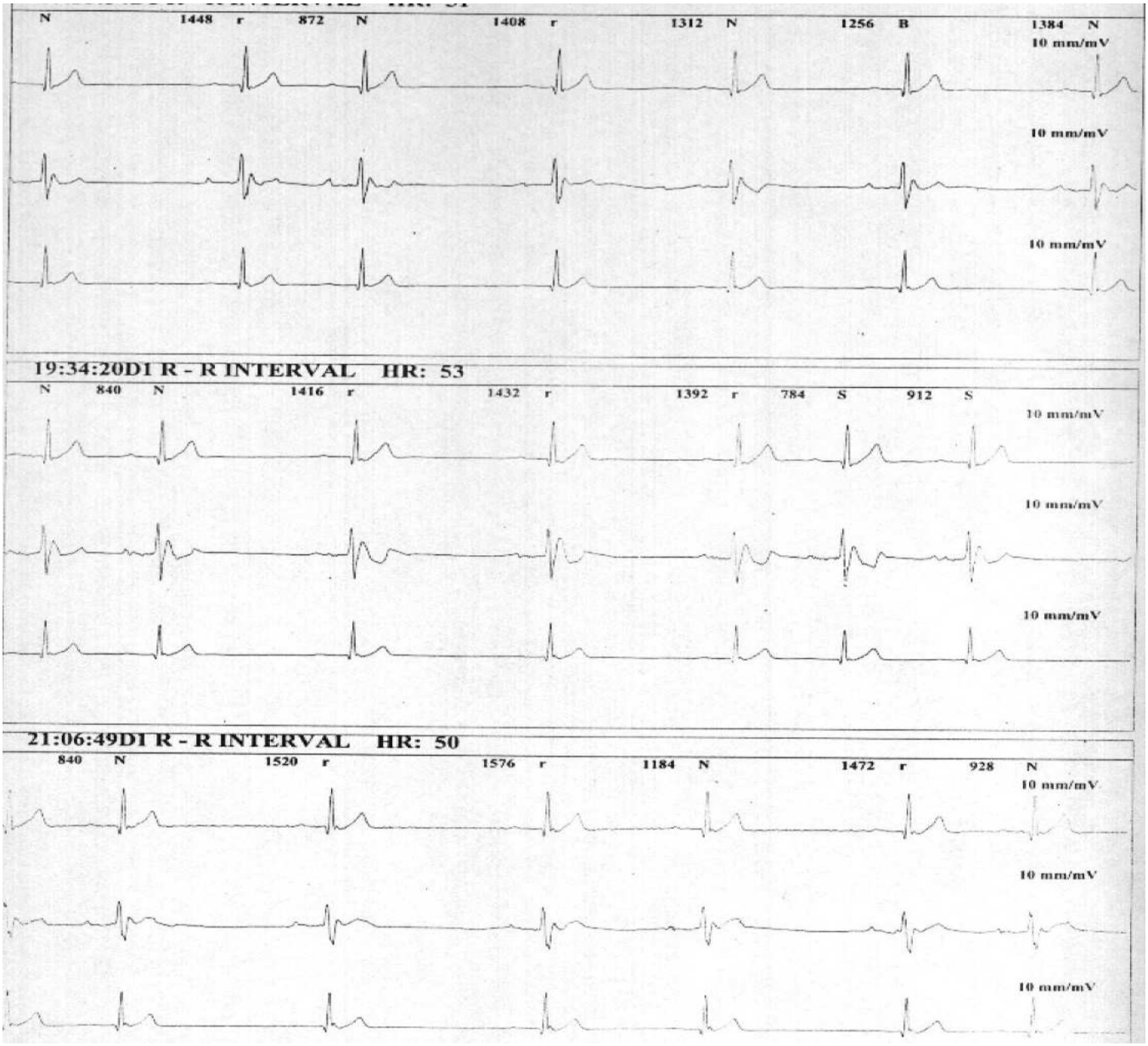

FIGURE 2. HOLTER SHOWING SHORT-LONG CYCLE LENGTHS.

النجاة السنوية لمستلمي الكلية المزروعة وحيوية الكلية المزروعة بعد سنة من اجراء عملية زراعة الكلية

أسامة سعدي عبد المحسن[١] ، أسامة الناصري[١] ، أسامة نهاد رفعت[٢]

الخلاصة

خلفية الدراسة: تمثل الكلية المزروعة علاجا ناجعا لمرضى عجز الكلية المزمن النهائي ، وقد أدى التطوير المستمر لأدوية كبح الجهاز المناعي مع بقية الأدوية والرعاية الطبية المقدمة للمستلم بعد إجراء العملية إلى تحسن ملحوظ في أداء المريض و العضو المزروع معا

هدف الدراسة: تقويم حيوية المستلم والكلية المزروعة بعد سنة واحدة من اجراء عملية الكلية المزروعة ، ومحاولة ربط الوفاة بعد العملية مع عاملي العمر و نوع التطابق النسيجي بين المتبرع و المستلم.

طريقة العمل: اجريت دراسة تشمل خمسين مريضا تتراوح اعمارهم بين (١٥-٦٢) سنة في وحدة زراعة الكلية في مستشفى الجراحات التخصصية- بغداد للفترة من شهر ايلول ٢٠٠٠ الى شهر تشرين الاول ٢٠٠٢ ، تم خلالها متابعة المرضى (مستلمي الكلية المزروعة) مع حيوية ووظيفة العضو المزروع لمدة سنة بعد اجراء العملية. تم أخذ الكلية المزروعة في جميع الحالات من متبرع حي وتراوحت نتائج الفحص النسيجي (HLA-matching) بين المتبرع و المستلم بين (غير مطابق تماما) و (تطابق نسيجي جزئي احادي). شملت المتابعة الحالة السريرية ، الفحوصات المختبرية متضمنة فحوصات وظائف الكلية المزروعة ، فضلا عن الفحوصات الدورية بجهاز السونار و الدوبلر. حدثت الوفيات في ردهة العناية المركزة للمركز كما تم اجراء جميع عمليات رفع الكلية المزروعة ، عند الحاجة في المركز أيضا.

النتائج: بعد مرور سنة على عملية زراعة الكلية استمر تسعة و ثلاثون مريضا (٧٨%) على قيد الحياة بينما توفي أحد عشر مريضا (٢٢%) خلال السنة الاولى من اجراء العملية بسبب مضاعفات جهاز القلب و الاوعية الدموية والتسمم الجرثومي. فيما يتعلق بوظيفة الكلية المزروعة، فقد كانت طبيعية بعد سنة من اجراء العملية عند ثمانية و ثلاثين مريضا (٧٦%) بينما احتاج اثنا عشر مريضا (٢٤%) الى اجراء جراحة لرفع الكلية المزروعة بسبب الصورة السريرية لوجود رفض حاد للكلية المزروعة ام يستجب للعلاج التحفظي المقدم في المركز. لم يثبت احصائيا وجود علاقة بين العمر و نوع التطابق النسيجي مع الوفاة بعد اجراء زراعة الكلية.

الاستنتاج: العمل المبكر للكلية المزروعة مع استمرارها في نشاطها الطبيعي هو عامل اساسي في بقاء العضو المزروع فعالا لفترة طويلة. ألعوامل الاساسية في وفاة مستلمي الكلية المزروعة هي مضاعفات جهاز القلب و الاوعية الدموية ثم التسمم الجرثومي. لم يثبت وجود ربط بين عمر المريض المستلم ونوع التطابق النسيجي مع وفاة المرضى خلال السنة الأولى بعد إجراء العملية.

مفتاح الكلمات: زرع الكلية ، النجاة السنوية للمريض ، أمراض القلب و الاوعية الدموية ، الرفض الحاد

الملخصات العربية

[١] فرع الجراحة البولية[كلية الطب-جامعة النهرين]
[٢]فرع الجراحة البولية[كلية الطب-جامعة بغداد]

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الخمج في وحدة الرعاية المركزية للخدج في طرابلس - ليبيا .

جواد كاظم الديوان[١] ، طارق الحديثي[٢] ، عبد اللطيف شعبان[١] ، محمد ديكنة[١]

الخلاصة

خلفية الدراسة: الاخماج سبب شائع ومهم من اسباب مراضة ووفيات الخدج. والهدف من البحث هي دراسة انتشار الاخماج البكترية وانواعها بين الخدج الراقدين في ردهة العناية المركزة في مركز طرابلس الطبي – ليبيا .
طريقة العمل: تم مراجعة ملفات الخدج الراقدين في مركزطرابلس الطبي للفترة أيلول – ١٩٩٦ ولغاية اب ١٩٩٨. زرع عينات الدم هي الوسيلة لتحديد الخمج البكتري . دخول المرضى تم تصنيفه الى ملوث وغير ملوث .
النتائج: خلال فترة الدراسة رقد ١١٢٣ خديجا في ردهة العناية المركزة . ١٢٩ (١١.٥%) اصيبوا بالخمج البكتيري . ١٠.٦% و ٢٤% من الخدج الملوثين وغير الملوثين على التوالي . كانت نتائج زرع عينات الدم موجبة لدى ١١٥ (١٠.٢%) من الخدج بينما كانت زرع عينات السائل الشوكي موجبا لدى ٢٤ (٢.١%) . البكتريا ذات الصبغة (كرام) السالبة هي السائدة لدى الاطفال المصابين بالخمج . انواع السريشا لدى ٣٨.٣% و ٥٠% من عينات الدم وسائل النخاع الشوكي . البكتريا سالبة التخثر CONS تم عزلها من ١١.٣% من نتائج عينات الدم .
الأستنتاج: خكج الخدج لايزال يشكل مشكلة تواجه البلد و هنالك حاجة لدراسة التلوث البكتيري في الجهاز التناسلي للنساء الليبيات الحوامل و علاقتها بالخمج للخدج.
مفتاح الكلمات: خمج الخدج، البكتيريا ذات الصبغة السالبة لكَرام، ليبيا

[١]فرع طب الأطفال [كلية الطب-جامعة الفاتح-ليبيا]
[٢]فرع طب مجتمع [كلية الطب -جامعة بغداد]

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دراسة في تخطيط القلب للمرضى المصابين بالربو القصبي الحاد المصحوب بالم الصدر

زيدان خلف الحركاني

الخلاصة

خلفية الدراسة: أن أعراض مرض الربو القصبي الحاد متعددة وتشمل ضيق التنفس الشديد،السعال والاختناق ولكن البعض يصاب بألم الصدر أيضا والذي لا يتم التركيز عليه عادة تداخل الاعراض و حاجة المريض للعلاج الآني

هدف الدراسة: لمعرفة مصدر الم الصدر في مرضي الربو القصبي الحاد وهل هو جزء من الاعراض اوناتج من قصور الشرايين التاجية؟

طريقة العمل: تضمنت الدراسة ٢٠٠ حالة ربو قصبي حاد وسجلت الاعراض السريرية المختلفة كما تم اختيار الحالات المصحوبة بالم الصدر وتم عمل تخطيط القلب تجميع الحالات مع الفحوصات الاخري واعيد تخطيط القلب بعد مرور ٤٨ ساعة من بداية الحالة وتم تسجيل ودراسة التغيرات الحاصلةالنتائج :تبين من الدراسة ان٣٠ حالة من اصل ٢٠٠ حالة للربو القصبي الحاد كانت مصحوبة بالم الصدر (١٥%) وان اغلب الحالات كانت فى الاعمار اكثرمن ٥٠ سنة(٨٠%). ان التغيرات الحاصلة في تخطيط القلب كانت تدلل على احتمال وجودالدبحة الصدرية في (٦٧%) في المرضي المصابين بالم الصدر وتراجعت النسبة الي (٤٠%) بعد اعادة التخطيط للقلب بعد مرور ٤٨ ساعة

الاستنتاج: أن نسبة لايستهان بها من مرضى الربو القصبي الحاد تشكو من الم الصدر والذي تبين من الدراسة انه قد يكون ناتجا من قصور الشرايين التاجية للقلب وليس فقط احد الاعراض المصاحبة لحالة الربو والدي يتطلب الانتباه له والتحري عن مصدره.

مفتاح الكلمات: الربو القصبي، الم الصدر، قصور الشرايين التاجية

رئيس فرع الطب [كلية طب الكندي -جامعة بغداد]

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الأنضباب الجنبي، دراسه خلويه، كيميائيه حياتيه (تشمل أنزيم ألأدينوسين دي أمينيز و لاكتك دي هايدروجينيز) ودراسه جرثوميه.

فائزة عفتان الراوي[١] ، نزار جبار متعب[١] ، زينب طالب[٢]

الخلاصة

خلفيه الدراسه: الأنضباب الجنبي الغير معروف سببه من المشاكل السريريه المهمه. لذلك فان قياس كمية أنزيم ألأدينوسين دي أمينيز و لاكتك دي هايدروجينيز في سائل الأنضباب الجنبي اكتسب أهميه متزايده للتميز بين ألأنضباب الدرني، والسرطاني والخمجي ألألتهابي.

هـدف الدراسـة: تقييم الفائدة التشخيصية لأنزيمي ألأدينوسين دي أمينيز و لاكتك دي هايدروجينيز في سائل الأنضباب الجنبي لتميز الأنضباب ألدرني عن الأنضباب لأسباب أخرى.

المرضى، المواد وطرق العمل: دراسه تقدميه ل ٧٥ مريض مصابين بالأنضباب الجنبي (٥٣ ذكور و ٢٢ اناث)، معدل أعمارهم ٤٣.٨ سنه. درست الحالات سريريا مع اجراء الفحوصات المختبريه للسائل الجنبي. أجري الفحص الخلوي بأستعمال صبغة H&E والفحوصات الجرتوميه (صبغتي Gram و AFB) و فحوصات كيميائيه (كمية أنزيم ألأدينوسين دي أمينيز و تاكتك دي هايدروجينيز، البروتين و السكر) و حساب العدد الكلي و التفريقي للخلايا المحتمل ايجادها في السائل الجنبي.

النتائج: من المعلومات و الفحوصات السريريه و المختبريه قسمت الحالات المرضيه الى ستة مجاميع: مجموعة الأنضباي الجنبي الدرني (٣٢ حاله)، السرطاني (١٣ حاله)، الجنبي الخمجي (١٠ حاله)، انضباب ناتج عن عجز القلب (٨ حالات)، غير معروفه أسبابه (٦ حالات) و متفرق الأسباب (٦ حالات). أعلى معدل لأنزيم ألأدينوسين دي أمينيز كانت ٧٦.٧ وحده فياسيه/لتر في الأنضباب التدرني مقارنة ب٣٢.٤ وحده فياسيه/لتر في الحالات السرطانيه. أما انزيم لاكتك دي هايدروجينيز فأعلى قياس كان في ألأنضباب السرطاني ٣٢١.١ وحده فياسيه/لتر و كان كلا ألأنزيمين ذو فائده تشخيصيه للتميز بين أنواع ألأنضباب الجنبي ($P<0.005$).

الاستنتاج: لأنزيمي ألأدينوسين دي أمينيز و لاكتك دي هايدروجينيز في سائل الأنضباب الجنبي أهميه تشخيصيه يمكن ألأستفاده منها في ألتميز بين الأنضباب الجنبي ألدرني والسرطاني عن الأنضباب لأسباب أخرى.

مفتاح ألكلمات: ألأنضباب الجنبي، أنزيم ألأدينوسين دي أمينيز، أنزيم لاكتك دي هايدروجينيز.

[١] فرع الباثولوجي [كلية الطب - جامعة النهرين]
[٢] مركز البحوث الطبية

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المضاعفات النزفية والخثارية لدى المرضى المصابين باضطرابات تكاثرية نقوية مزمنة

سعد شوقي منصور [١] ، رعد جابر موسى [١] ، وقاص فاضل السامرائي [٢]

الخلاصة:

خلفية الدراسة: ان نسبة حدوث المضاعفات الخثارية والنزفية تكون مرتفعة بين المرضى المصابين بابيضاض الدم التقياني المزمن . وأن معدل تكرار المضاعفات النزفية كانت أعلى من معدل تكرار المضاعفات الخثارية.

هدف الدراسة: استهدفت هذه الدراسة استكشاف نسبة المضاعفات الخثارية والمضاعفات النزفية لدى المرضى المصابين باضطرابات تكاثرية نخاعية مزمنة وكذلك لتحديد المعايير الأكثر ترافقا و / أو الأكثر تنبا لحدوث تلك الاختلاطات المرقئة .

طريقة العمل: خمسة وأربعون مريضا مصابين بمختلف أنماط الاضطرابات التكاثرية النخاعية المزمنة (٢٤ رجل، ١٧ إمراة) تم شمولهم بهذه الدراسة الاستقدامية بمعدل عمر المرضى (± الأنحراف المعياري) ٤١,٣٥± ١٠,٩ سنة

أجريت هذه الدراسة للفترة من كانون الثاني ٢٠٠٣ إلى حزيران ٢٠٠٤ وتم اختيار المرضى من ثلاثة مراكز طبية في مدينة بغداد .

فضلا عن ذلك ، ٢٥ من الأشخاص الأصحاء ظاهريا (١٣ رجل ، ١٢ إمراة) كمجموعة سيطرة بمعدل عمر (+ الانحراف المعياري) ٤٢,٢ ±١٢،. سنة

مجموعة المرضى والسيطرة أخضعوا للاختبارات التالية ؛ تركيز الفابيرينوجين ؛ حساب فاعلية العامل الثامن في بلازما الدم ؛ حساب الفاعلية ألأنتيجينية للعامل السابع والعامل العاشر في بلازما الدم ؛ وقياس تركيز مثنويات- D (D-Dimers) في بلازما الدم.

النتائج: أظهرت النتائج أن النسبة الكلية لحدوث المضاعفات المرقئة بين المرضى المصابين بأضطرابات نقي العظم التكاثرية المزمنة كان ٢٠%

أن حدوث المضاعفات الخثارية النزقية كانت تترافق وبشكل معتمد مع انحدار العمر (P= 0.005) .

لقد أظهرت النتائج بانة لم يكن هنالك ترافق معتد بين حدوث الاضطرابات الخثارية النزفية مع ارتفاع تركيز الفابيرنيوجين في بلازما الدم (P = 0.4).

الفاعلية الانتيجينية للعامل السابع في بلازما الدم كانت منخفضة بشكل معتد لدى المرضى المصابين بابيضاض الدم النقياني المزمن مقارنة بمجموعة السيطرة (P = 0.001). فيما يتعلق بفاعلية العامل الثامن في بلازما الدم والفاعلية الانتجينية للعامل العاشر في بلازما الدم وزمن النزف تظهر الإحصائيات فأنه لم يظهر التحليل المتعدد بين مختتلف مجاميع المرضى ومجموعة السيطرة

أي اختلافات معتدة (P > 0.05) .بالإضافة إلى ذلك، لم يوجد ترافق معتد بين تلك المعايير وحدوث الاضطرابات الخثارية النترفية (P > 0.05).

أن الاختلافات في نسبة الإيجابية لمثنويات – D في بلازما الدم بين المرضى الذين يعانون من اضطرابات خثارية نزفية وبين المرضى الذين لم تظهر عليهم تلك الأعراض لم تكن معتدة (P > 0.05).

الملخصات العربية

الاستنتاج:عليه يمكن الاستنتاج بأن المرضى المصابين باضطرابات تكاثرية نخاعية مزمنة ربما يكونون أكثر عرضة لحدوث تلك الاضطرابات وأن كثرة الصفيحات الدموية ربما يكون لها دور مهم في أمراضية تلك المضاعفات الخثارية النزفية .

مفتاح الكلمات: نزفيه، تخثريه،تكاثرية نقويه مزمنة

[1]فرع الباثولوجي- أمراض الدم [كلية الطب -جامعة النهرين]
[2]فرع الباثولوجي [كلية الطب - الجامعة المستنصرية]

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مقارنة بين مستوى المضادات في الدم للكلاميديا تراكوماتس عند أمهات وأطفالهن الحديثي الولادة بعد الولادة الطبيعيةوعند أمهات وأطفالهن بعد الولادة القيصرية

إيناس طالب عبد الكريم [١] ، نضال عبد المهيمن[٢] ، . تارة الجرموندي [٣]

الخلاصة:

خلفية الدراسة : أثبتت الدراسات العديدة التي أجريت إن الكلاميديا تراكوماتس تلعب دور بارز في ألاضطراب الذي يصيب الجهاز التكاثري عند البشر

هدف الدراسة : أجرى هذا البحث لتحديد مستوى الأجسام المضادة للكلاميديا تراكوماتس عند الأمهات بعد الولادة الطبيعية او بعد اجراء عملية قيصرية وعلى أطفالهن الحديثى الولادة وتأثير مختلف العوامل الوبائية، الطبية والعوامل التي تؤثر أثناء الحمل على مجموعة الدراسة.

طرائق الدراسة: تم أخذ نموذج دم من ١٦٦ امرأة بعد الولادة الطبيعية واطفالهن (المجموعة الاولى) وكذلك من ٣٢ امرأة بعد أجراء عملية قيصرية وأطفالهن (المجموعة الثانية) ثم أجراء فحص الأليزا على نماذج الدم لمعرفة مستوى المضادات للكلاميديا تراكوماتس فيها.

النتائج: كان معدل الإصابة بلكلاميديا تراكوماتس ٢٤% و ٢٠.٥% عند الأمهات وأطفالهن في المجموعة الأولى بينما كانت ٤٠.٥% و ٣٨.١% في المجموعة الثانية. كانت عوامل وجود نزف أثناء الحمل (علاقة ذات مغزى إحصائي سالب) وجود إفرازات أثناء الحمل، التهاب المجاري البولية (علاقة ذات مغزى إحصائي) وكذالك وزن الوليد تحمل معدلات أعلى عند الأمهات في المجموعة الثانية بينما كان وجود حمى وفقر الدم أثناء الحمل وعدد الإسقاطات السابقة تحمل معدلات أعلى عند الأمهات في المجموعة الأولى.

الاستنتاجات: أظهرت الدراسة أن معدل الإصابة بلكلاميديا تراكوماتس كانت أعلى عند الأمهات اللواتي أجري لهن عملية قيصرية وأطفالهن من الأمهات اللواتي وضعن بواسطة ولادة طبيعية

كلمات المفتاح: المضادات للكلاميديا تراكوماتس عند النساء بعد الولادة.

[١] فرع طب المجتمع [كلية الطب - جامعة النهرين]
[٢] فرع الأحياء المجهرية [كلية الطب- جامعة النهرين]
[٣] مركز البحوث الطبية [كلية الطب - جامعة النهرين]

المجلة العراقية للعلوم الطبية ٢٠٠٧ م المجلد ٥ العدد٢ ص٣٤-٣٩

مستوى المغنيسيوم في مصل دم الاطفال المصابين بالربو القصبى المزمن

نجم الدين الروزنامجى[١] ، حسام محي العلواني[١] ، ابتسام العبوسي[٢]،عمار الشبلى[١]

الخلاصة

خلفية الدراسة : الربو القصبي(Asthma) في اللغة اللاتينية تعني عسر التنفس وهو أكثر أمراض الأطفال المزمنة شيوعا وهو عبارة عن انسداد في المجاري التنفسية ينتج عن تحسس المجارى الهوائية لمحفزات مختلفة . لقد شهد المرض زيادة في نسبة الإصابة وشدتها ونسبه الوفيات في الآونة الأخيرة رغم التقدم الحاصل فى وسائل العلاج .

مستوى المغنيسيوم الذي هو من العناصر المهمة في دم المرضى المصابين بالربو القصبي المزمن وتغيراته هو محور هذه الدر اسه.

هدف الدراسة : هو دراسة مستوى المغنيسيوم في دم الأطفال من مرض الربو وتغيراته مع نوبات المرض .

طريقة العمل : تضمنت الدراسة ٥٠ طفل مصاب بالربو القصبي ، ودرست شدة الربو لديهم عن طريق العلامات السريرية ، وجهاز سبايرومتري ، وأخذت عينة دم وريدي من كل مريض لقياس مستوى المغنيسيوم لديهم وكذلك أخذت ٥٠ عينه أخرى من أطفال غير مصابين بالربو وقيس مستوى المغنيسيوم للمقارنة .

النتائج : بينت النتائج ان مستوى المغنيسيوم في مصل دم مرضى الربو القصبي هو اقل منه لدى العينه غير المصابه بالمرض بمعدل ٢,٦ ملغ/١٠٠ الى ٣,٧ ملغ/١٠٠ بالتسلسل ولا توجد علاقة قويه بين شده المرض ومستوى المغنيسيوم في الدم .

الاستنتاج : المغنيسيوم من العناصرالمهمه فى دم المريض بالربو، وتكون مستوياته أدنى من الأطفال الآخرين الأصحاء وقد تفيد هذه المعلومة في العلاج.

مفتاح الكلمات: الربوالقصبى،المغنيسيوم ، شدة المرض

[١]فرع طب الأطفال [كلية الطب-جامعة النهرين]

[٢]فرع الكيمياء الحياتية [كلية الطب-جامعة النهرين]

المجلة العراقية للعلوم الطبية ٢٠٠٧ م المجلد ٥ العدد٢ ص٤٠-٤٣

أسباب الوفيات ت لحديثي الولادة في مستشفى الكاظمية التعليمي

لمياء عبد الكريم السعدي

الخلاصة

خلفية الدراسة : على الرغم من الانخفاض الحاصل في الوفيات لحديثي الولادة في العقود المتأخرة عن الوفيات للأطفال الذين تجاوزوا سن حديثي الولادة ولكن الوفيات لحدثي الولادة لا زالت تشكل تقريبا ثلثي الوفيات لدى صغار الأطفال.

الهدف : لإيجاد أهم الأسباب المؤدية إلى الوفاة لدى جميع حديثي الولادة اللذين ادخلوا إلى ردهة العناية المركزة لحديثي الولادة في مستشفى الكاظمية التعليمي وذلك لمنع والوقاية ومعالجة ما يمكن من هذه الأسباب.

طريقة العمل: لقد تمت مراجعة السجلات الطبية لكل المرضى اللذين ادخلوا إلى ردهة حديثي ا لولادة في مستشفى الكاظمية التعليمي للفترة الزمنية بين ١٩٩٥-٢٠٠٥ لايجا أهم الأسباب المؤدية إلى الوفاة خلال هذه الفترة.

النتائج: كانت النتائج إن عدد المرضى الداخلين الى ردهة حدثي الولادة هو ٢٦٨٣مريض ولمختلف التشخيصات وكان عدد الوفيات لجميع الأسباب هو ٩٨٢حالة مرضية . إن أهم أسباب الوفاة لدى المرضى الراقدين هو وحسب ما هو مسجل في السجلات الطبية للمرضى كالأتي :طفل خديج مع متلازمة عسر التنفس ،تسمم الدم الجرثومي،الاختناق الولادي ، التشوهات الخلقية ، استنشاق العق ، والطفل لأم مصابة بداء السكر .

الاستنتاج: إن من أهم العوامل التي تؤدي الى انخفاض الوفيات لدى حديثي الولادة هو محاولة منع حدوث الولادات المسبقة ومعالجة الأطفال الخدج وقليلي الوزن سوف يؤدي الى انخفاض في نسبة الوفيات بشكل كبير كذلك الأمراض الوراثية والتشوهات الخلقية ومعالجة ما يمكن معالجته منها ومحاولة منع حدوثها.

مفتاح الكلمات: الوفيات لحديثي الولادة ،ردهة العناية المركزة لحديثي الولادة، متلازمة عسر التنفس.

فرع طب الأطفال [كلية الطب-جامعة النهرين]

المجلة العراقية للعلوم الطبية ٢٠٠٧ م المجلد ٥ العدد٢ ص٤٤-٤٨

نمط نظائر أنزيم لاكتيت دي هيدروجينيز في التشخيص التفريقي لسوائل انصباب الجنب

حسام حسون علي[١]، عبد الوهاب رزوقي حمد[٢] ، زينب طالب آل عكاب[٣]

الخلاصة

خلفية الدراسة: إن فعالية إنزيم اللاكتيت دي هايدروجينيز في سوائل انصباب الجنب ليس لها قيمة في التميز بين الأنواع المختلفة من سوائل النتحية مثل سوائل انصباب سرطاني وغير سرطاني.

هدف الدراسة: تقييم القيمة التشخيصية لفعالية نظائر أنزيم اللاكتيت دي هايدروجينيز في مصل وسوائل انصباب الجنب في التميز بين سوائل النتحية المختلفة(نتحي التهابي وسرطاني).

طريقة العمل: تمت دراسة ٦٦ مريضا راقدا في مستشفى الكاظمية التعليمي في الفترة الواقعة بين (شباط ٢٠٠٠- تشرين الاول ٢٠٠٠) يشكون من سوائل انصباب الجنب وتم تقسيمهم الى ثلاثة مجاميع: المجموعة الاول: تتضمن ١٢ حالة كانت ناتجة عن انصباب نتوح مصلي والمجموعة الثانية وتتضمن ٣١ حالة كانت ناتجة عن انصباب نتحي التهابي والثالثة تتضمن ٢٣ حالة كانت ناتجة عن انصباب سرطاني.

النتائج: أظهرت النتائج إن فعالية انزيم اللاكيت دي هايدروجينز كان معنويا اعلى في سوائل النتحيه (نتحي التهابي وسرطاني) مقارنة بسوائل النتوح المصلي (حبيبي). إن فعالية أنزيم(LD) في سائل الجنب كانت ذو قيمه منخفضة في تميز مابين النتيجة الالتهابية والسرطانية. بينما أظهرت دراسة فعالية نظائر الأنزيم (LD) إن فعالية (LD3) كانت عالية في سوائل النتحيه الالتهابية مقارنة مع سوائل الانصباب السرطاني مع أنها فقط معنويا أعلى في المرضى الإناث. إضافة إلى ذلك كان للنظير (LD3) نمط مميز في مصل وسوائل انصباب الجنب وفي المجاميع الثلاث.

الاستنتاج: يختلف نمط نظائر أنزيم اللاكتيت دي هايدروجينيز بين سوائل النتوح المصلي (حبيبي) وسوائل النتحية (التهابي وسرطاني).
إن تضمين فعالية نظائر انزيم LDفي التحليل البيوكيميائي لسوائل انصباب الجذب اظهر قيمة تميزية إضافية في الفصل بين سوائل النتحية المختلفة وخصوصا بين سوائل نتحي التهابي وسوائل نتحي سرطاني.
الأسباب والاحتمالات التي تؤدي إلى هذه التغييرات تم الإشارة لها في هذه الدراسة.

مفتاح الكلمات: سوائل انصباب الجنب، نظائر انزيم لاكتيت دي هايدروجينيز .

الملخصات العربية

[1]فرع الباثولوجي [كلية الطب - جامعة النهرين]
[2]فرع الكيمياء الحياتية [كلية الطب-جامعة النهرين]
[3]مركز البحوث الطبية

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الفحص ألشعاعي لتجويف الفك الأعلى

تحرير نزال الدليمي

الخلاصة

خلفية الدراسة : أمراض التجويف الفكي تشكل أعراض وعلامات التي لها أسباب سنية وكذلك من الممكن أن تكون أمراض الفم والأسنان سبب مؤثر في حالة التجويف الفكي.

هدف الدراسة : لفحص التجويف الفكي لعينة ما في محافظة الانبار باستخدام جهاز الأشعة الفكية.

طرق الدراسة : تم فحص ١٢٠ مريضا" بين ٣٠ – ٧٠ سنة بالفحص الشعاعي وهم ٥٦ % ذكور و ٤٤ % إناث في كلية طب الأسنان / جامعة الانبار .

النتائج : وجد ان ٤٢ % من المرضى لديهم علامات مرضية للتجويف الفكي .

الاستنتاجات : أكثر العلامات المرضية وجدت في العقد الخامس من العمر ومن الذكور بنسبة أكثر من الإناث .

الكلمات المفاتيح : تجويف الفك الأعلى ، الأشعة الفكية ، تليف الأنسجة الرخوة .

كلية طب الأسنان -جامعة الأنبار

المجلة العراقية للعلوم الطبية ٢٠٠٧ م المجلد ٥ العدد٢ ص٥٩-٦٤

فقر الدم عند النساء خلال فترة سن الإنجاب في القرى

مائدة يوسف شمدين [١] , بيبين خورشيد السليفانى [٢]

الخلاصة

خلفية الدراسة: ان فقر الدم عوز الحديد هو مشكلة طبية واجتماعية ذات اهمية قصوى، مسببة وفيات قليلة ولكنها تساهم مساهمة خطيرة في ضعف الصحة العامة وانجاز العمل لملايين الناس.

أهداف البحث: معرفة مدى انتشار فقر ألدم عند النساء في فترة سن الإنجاب عشر سنوات بعد المقاطعة .

طريقة العمل : تمت الدراسة في ناحية بادوش ٢٠ كيلو متر شمال مدينة الموصل، على النساء خلال فترة سن الإنجاب في أيلول ٢٠٠٢ خلال الممارسة الميدانية لجامعة الموصل . شملت الدراسة ثمانية وتسعين امرأة، قيمن سريريا بعد استجوابهن عن العمر،ألحاله الزوجية،ألحاله الاجتماعية، عدد الأطفال، الرضاعة، كمية الدم في الدورة الشهرية و مشاكل طبية أخرى .تم اخذ عينة من الدم و اجريت الفحوصات التالية : تركيز الهيموكلوبين ، تراص كريات الدم الحمراء ، تركيز الحديد ، سعة الربط الكلية للحديد و نسبة إشباع الترانسفرين.

النتائج : معدل العمر للنساء **كان28.75 ± 10.6** سنة (يتراوح بين 15-50سنة) . معدل الحمل السابق هو 5 . و تبين ان 58 امرأة(57.14%) يعانون من فقر الدم اعتمادا على تركيز الهيموكلوبين ، تراص كريات الدم الحمراء ، تركيز الحديد ، سعة الربط الكلية للحديد و نسبة إشباع الترانسفرين . كان معدلات القيم عند النساء المصابات بفقر الدم والغير مصابات كالآتي: ,(19.37%, 24.7%) (106.8gm/l, 126.79gm/L) ,(0.32L/L, 37.9L/L) (13.53µmol/dl, 15.42 µmol/l), (69.85 µmol /L, 62.55µmol) على التوالي .وكانت النتائج الكلية لكل المجاميع كالاتى,61.01µmol/L 14.34µmol/L, 0.34L/L,115.4gm/L و23.5%.

أظهرت نتائج هذا البحث انخفاض معنوي فى مستوى الهيموكلوبين وارتفاع معنوى فى سعة الربط الكلية للحديد (احتمالية P<0.05) فى مرضى فقر الدم مقارنة مع الغير المصابين بقر الدم بينما لم توكد النتائج وجود فرق معنوي في قيم تراص كريات الدم ، تركيز الحديد ونسبة إشباع الترانسفرين في مرضى فقر الدم مقارنة مع الغير المصابات بفقر الدم احتمالية (p>0.05).

الاستنتاجات: ثبت إن معظم النساء المصابات بفقر الدم سببه نقص الحديد، الناتج من سوء التغذية، تعدد الولادات، الرضاعة ونزف الدورة الشهرية.هذه النتائج ربما تعكس تأثيرات الحصار على التغذية والحالة المعيشية في المناطق الريفية.

الملخصات العربية

التوصيات: إجراء مسح الهيموكلوبين للنساء كل خمسة سنوات في سن ١٢-١٨سنة،وكل سنة للنساء الحوامل والمعرضين للإصابة بفقر الدم .إعطاء الأقراص الحاوية على الحديد لكل النساء في مرحلة الإنجاب في المناطق الريفية .

مفتاح الكلمات: فقر الدم نقص الحديد، سنوات الإنجاب من عمر المرأة.

[١] قسم النسائية والتوليد (كلية الطب - جامعة دهوك)
[٢] قسم الفسلجة الطبية(كلية الطب- جامعة الموصل)

المجلة العراقية للعلوم الطبية ٢٠٠٧ م المجلد ٥ العدد٢ ص٦٥-٧٠

تأثيرات الافلاتوكسين ب١ في بعض الخلايا القاطنة في العضلات الهيكلية باستخدام تقنيه صبغة تمايز الانويه

مي فاضل الحبيب

الخلاصة

خلفيه الدراسة : سموم الافلاتوكسين ب ١ هي واحده من سموم الفطريات التي جذبت انتباه العلماء للأبحاث وهذه السموم عبارة عن مجموعه مترابطة من سموم الفطريات. إن مشكله استخدام الاغذيه الملوثة بسموم الفطريات لا تزال واحده من أهم السمات في حقل التغذية للإنسان والحيوان.

هدف الدراسة : صممت هذه الدراسة لقياس ومتابعه مدى تأثير استعمال أغذيه ملوثهبالافلاتوكسين ب١ في العضلات الهيكلية والتغيرات التي تحدثها في الخلايا القاطنة للعضلات الهيكلية.

طرائق الدراسة : استخدمت في هذه الدراسة مجموعتين من الجرذان البيض، أعطيت المجموعة الأولى سموم الافلاتوكسين ب١ مع الغذاء والمجموعة الثانية وهي مجموعه السيطرة أعطيت غذاء بدون الافلاتوكسين.
استأصلت العضلة الباسطة للأصابع وقطعت إلى قطع صغيره ثم تم تحضير الانسجه للحصول على مقاطع شبه خفيفة وصبغت بصبغه تمايز الانويه.

النتائج:

- هنالك ازدياد ملحوظ في وزن الجسم للحيوانات التي استعملت الغذاء الملوث بسموم الافلاتوكسين ب١.
- انويه الخلايا العضلية الهيكلية أظهرت قله بالعدد مع قله بعدد ألاوعيه الدموية في الحيوانات المعالجة بغذاء الافلاتوكسين ب١.

الاستنتاجات : لقد تم استنتاج وجود تأثيرات ملحوظة لسموم الافلاتوكسين على عدد الخلايا القاطنة في العضلات الهيكلية.

مفتاح الكلمات : سموم الافلاتوكسين ب١ _العضلات الهيكلية – صبغه تمايز الانويه.

فرع الأنسجة و الأجنة [كلية الطب - جامعة النهرين]

المجلة العراقية للعلوم الطبية ٢٠٠٧ م المجلد ٥ العدد٢ ص٧١-٧٧

اماتنب ميسيليت (كليفيك) في المرضى العراقيين المصابين بابيضاض الدم النقياني المزمن

نبيل سلمان مراد[١] ، علي مسلم العامري[٢]

الخلاصة

خلفية الدراسة: مرض ابيضاض الدم النقياني المزمن ينشأ من خلية واحدة جذعية من خلايا النخاع العظمي . يتميز المرض بازدياد كبير في عدد الخلايا البيضاء الحبيبية البالغة و الابتدائية بأنواعها العدلة و الحمضة و القعدة . ثبت إن الخلل الجزئي الذي ينشأ من تبادل لمواد جينية بين صبغي (كروموسوم) 22.9 وهي ABL و BCRوما ينتجه من بروتين تا يروسيني هو السبب المباشر في حدوث المرض بحالته المستقرة أو المزمنة .

هدف الدراسة: هو دراسة الاستجابات السريرية و المختبرية للعقار اماتنيب ميسيليت مع تحديد أهم التأثيرات الجانبية في المرضى العراقيين المصابين بمرض ابيضاض الدم النقياني المزمن بحالاته الثلاثة المزمنة و المتسارعة و الحادة .

طريقة العمل: تم دراسة ثلاثمائة و اثنان و ستون مصاباً بالمرض مستقبلياً بعد تشخيصهم بواسطة فحص الدم المحيطي و نخاع العظم . ثم أعطي لكل منهم العقار اماتنيب بجرع ثابتة 400 ملغم تعطى كجرعة واحدة عن طريق الفم يومياً ،وتمت متابعتهم كل 4 أسابيع بإجراء الفحص الطبي السريري و المختبري لتحديد استجابتهم للعلاج .

النتائج: كان تكرار حدوث حالات الـ CML حسب الرقعة الجغرافية كالتالي : 17.4 %، 21.8 %، 61.6%، في الجنوب، الشمال، الوسط على التوالي.

تراوح عمر المريض بين 14 - 70 سنة بمعدل يقدر بـ 39.4 .شكل الجنس 192(53%) من الرجال و 170(43%) من النساء. لوحظت الاستجابة الكاملة السريرية و المختبرية في 325 مريضاً(90%)خلال الثلاث أشهر الأولى من بدء العلاج في المرضى بالحالة المزمنة. حوالي 4 من10 مريض استجاب في المرحلة المتسارعة من المرض بجرع تتراوح بين 600 - 800 ملغم يومياً. لم يستجب أي من المرضى الستة بالحالة الحادة من المرض . كانت معظم التأثيرات الجانبية للعقار طفيفة وسهلة التحمل.

الأستنتاج: استنتجنا إن العقار اماتنيب ميسيليت هو علاج فعال وقليل المخاطر في تحقيق الاستجابة السريرية أو المختبرية العالية لمرض ابيضاض الدم النقياني المزمن في العراق.

الكلمات المفتاحية: ابيضاض الدم المزمن النقياني . اماتنيب ميسيليت

[١]كلية الطب - جامعة النهرين
[٢]مركز أمراض الدم [كلية الطب - الجامعة المستنصرية]

المجلة العراقية للعلوم الطبية ٢٠٠٧ م المجلد ٥ العدد٢ ص٧٨-٨٤

الحمل متباين الموضع " تقرير حالة "

لقاء رياض الخزاعي

الخلاصة

هناك تزايد في حالات الحمل متباين الموضع في السنوات الأخيرة متزامنة مع زيادة استخدام الأدوية المنشطة للمبايض يوجد عادة هناك تأخير في تشخيص الحمل متباين الموضع حيث أكثر من ٥٠ % من الحالات يتم التشخيص بعد حدوث انفجار الحمل خارج الرحم. هناك حاجة إلى التشخيص المبكر لتقليل المضاعفات للأم مع الحفاظ على الحمل الموجود داخل الرحم.
في هذا التقرير نسجل حالة حمل متباين الموضع مع مراجعة المصادر.

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حالة لمتلازمة بروكادا " تقرير حالة "

محمد هاشم [١] ، تحسين الكناني[١] ، كامل نامق [١]، عمار طالب الحمدي[٢] ، قيس محمد سعيد المدرس[٣]

كلمات المفتاح : متلازمة بروكادا، لا نظميات بطينية، الموت القلبي المفاجئ.

[١]فرع أمراض القلب [مستشفى ابن البيطار]
[٢]فرع الباطنية [كلية الطب - جامعة النهرين]
[٣]فرع الفسلجة [كلية الطب - جامعة النهرين]

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الهيئة الأستشارية

اسامة نهاد رفعت (الهيئة العراقية للأختصاصات الطبية)

أكرم جرجيس (جامعة الموصل)

ألهام الطائي (الجامعة المستنصرية)

امجد داود نيازي (الهيئة العراقية للأختصاصات الطبية)

أميرة شبر (الجامعة المستنصرية)

أنعم رشيد الصالحي (معهد أبحاث الأجنة و العقم-جامعة النهرين)

ثامر أحمد حمدان (جامعة البصرة)

حسن أحمد حسن (جامعة النهرين)

حكمت الشعرباف (جامعة بغداد)

خالد عبدالله (جامعة النهرين)

داود الثامري (جامعة النهرين)

راجي الحديثي (الهيئة العراقية للأختصاصات الطبية)

رافع الراوي (جامعة النهرين)

رجاء مصطفى (الجامعة المستنصرية)

رياض العزاوي (الجامعة المستنصرية)

زكريا الحبال (جامعة الموصل)

سركيس كريكور ستراك (جامعة البصرة)

سرمد الفهد (جامعة بغداد)

سرمد خوندة (جامعة بغداد)

سميرة عبد الحسين (جامعة تكريت)

طاهر الدباغ (جامعة الموصل)

ظافر زهدي الياســـين (جامعة بغداد)

عبد الا له الجوادي (جامعة الموصل)

عدنان عنوز (جامعة النهرين)

فوزان النائب (الجامعة المستنصرية)

محمود حياوي حماش (جامعة النهرين)

نجم الدين الروزنامجي (الهيئة العراقية للأختصاصات الطبية)

نزار طه مكي (جامعة النهرين)

نزار الحسني (الهيئة العراقية للأختصاصات الطبية)

المجلة العراقية للعلوم الطبية
قائمة المحتويات

المقالات

تقرير حالة