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MICROBIAL INFECTION AND BILHARZIASIS AS RISK FACTORS IN RENAL OXALATE - CALCULI DISEASE

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ABSTRACT

Sixty nine of bilharzial and non - bilharzial stone formers, with uniary oxalate - calculi (71 stones), were categorized according to the chemical composition of stones into 4 groups. Biochemical and microbiological parameters for these groups were determined. It was found that urinary calculus was mostly found in patients suffered from bilharziasis (70.4%). Stone recurrence rate was frequently in 50.7% of patients with urinary tract infection. *Pseudomonas* , *E. coli* , *Staphylococci* and *Enterococci* were the common types isolated from urine and stone (surface) samples . The biochemical results demonstrated the significant increase in serum total protein and its fractions, (except albumin), in all groups, compared to the mean normal value. , α_2- glycoprotein revealed significant increased value in groups 2 and 4 (with stones containing calcium phosphate. Significantly increased value of β -glycoprotein was obtained in groups 1 and 3 (calcium oxalate - calcium phosphate (Ca Ox- CaP) or calcium oxalate - uric acid (CaOx - U stones). α_2 and β - glycoprotein were associated with high bacterial infection and incidence of acute inflammation. In the present work it could be confirmed that, bilharziasis increased the incidence of urinary tract infection and formation of stone. The stone recurrence rate was frequently high in the patients with chronic urinary tract infection, referring that the bacterial infection play an important role in the formation of stones.

INTRODUCTION

Urolithiasis is prevalent in certain areas of the world such as Middle East , North Africa, North India , Egypt, Holland and South China, although the exact mechanism of stone formation is unknown.

Some investigators; reported that, bilharziasis may increase the incidence of urinary tract obstruction, infection and stone formation[1]. Also **Abdel-Aziz et al.** [2] concluded that, urinary Schistosoma and the resulting immunopathological changes were predisposing factors in one way or another to the formation of renal stones. Further, **Grasso et al.** [3] found that 60% of the stone fragments were composed of pure calcium oxalate monohydrate in the patients with *Schistosoma haematobium*.

The infection may play an important role in stone initiation [4] ; when the bacteriurea is truly persistent , a renal stone is usually present as an anatomic or pathophysiological disorder. It is highly probable that infection may set the stage for the initiation of stone formation by providing a nucleus upon which insoluble salts are deposited. Recently, it was reported [5] that, recent bacteriological studies and investigation using the scanning electron microscope have also shown microorganisms to be present in calcium oxalate and other sterile stones, these microorganisms were assumed not to be able to split urea.

Diet rich in calcium phosphate and oxalate containing food may lead to increased renal excretion of those substances and an increased incidence of stone formation in susceptible individuals [6].

Approximately, 40% of the excreted oxalate comes from the metabolism of ascorbic acid and about 40-50% comes from metabolic reactions involving glyoxalic acid [7] . Changes in H^+ concentration of urine could affect the solubility of certain crystalloids and hence influence the formation of calculi , which are formed in acidic urine. These calculi are usually calcium oxalate or a mixture of calcium oxalate - calcium phosphate or calcium oxalate - uric acid.

Most of calcium oxalate stones are not composed of pure calcium oxalate but calcium oxalate predominated with small quantities of calcium phosphate and uric acid . Calcium phosphate can be present as either the more common apatite ($Ca_{10}(PO_4)_6(OH)_2$ or relatively unusual brushite ($CaHPO_4 2H_2O$).

Uric acid , is the major end product of purine metabolism in human. It accounts for the formation of uric acid calculi. Its crystals have a suitable size and shape to act as a nucleus for deposition of calcium oxalate [8].

Polyclonal increases in serum immunoglobulins are the normal response to infections. Glomerular damage allows the escape of serum intermediate molecular size proteins in the urine, e.g. albumin, alpha macroglobulins, transferin and immunoglobulin G (IgG) [9].

The present work has been carried out to evaluate quantitatively calcium, magnesium, phsphorus, and uric acid in serum of non-struvite stone formers compared to the controlled normal subjects. Total serum protein, electrophoretic fractions and serum glycoprotein fractions were estimated . The bacteriological investigation of both urine and stone surface were evaluated to identify the different types of microorganisms in all the studied groups with stone formation . Basides the releationship between the composition of stone , its location and type of microorganisms as well as the effect of bilharzial infection on the incidence of stone formation.

MATERIAL AND METHODS

Material of the study consisted of 69 cases (male and female) of stone formers (age range 13 -70 years), 50 out of the cases had bilharziasis , 17 healthy normal subjects (age range 26-45 years) were selected as control . The patients with stone formation were classified according to chemical composition of the surgically removed - stones, into 4 groups:

Group 1 : includes 12 cases with pure calcium oxalate (CaOx) stones.

Group 2 : includes 26 cases with 28 calcium oxalate + calcium phosphate (Ca Ox - CaP) stones.

Group 3 : includes 15 cases with calcium oxalate + uric acid (Ca Ox - U) stones.

Group 4 : includes 16 cases with calcium oxalate + calcium phosphate + uric acid (Ca Ox - CaP-U) stones.

All patients were seeking medical care in the Urology Department, Faculty of Medicine , Cairo University. Serum and urine samples were collected before the initiation of any treatment . After the surgical operation the stone samples were collected from the patients in sterile saline.

The following clinical parameters were evaluated :

1-Under complete aseptic conditions , clean mid - stream urine specimens and crushed pieces of stone surface were directly cultured on Nutrient agar, Baird Parker plates, Mannitol salt agar , CLED agar (for urine) , MacConkey agar , Shigella - Salmonella medium , endo agar, EMB agar and X LD agar. Isolation and identification of the pathogenic microorganisms were established by previously reported methods : (10-12) .

2-Serum total protein was determined by the biuret method as described before(13) . Evaluation of protein and glycoprotein fractions in serum, by Agarose Simple Electrophoresis , was carried out as previously discribed (14,15) .

3- Serum uric acid, calcium and magnesium were determined by using colorimetric test, the reagents were obtained from Boehringer Mannheim/ Germany, Bio Mericux / France and Química Clinica Applicada S. A./ Spain, respectively, (16,17) . Serum inorganic phosphorus was determined by photometric U.V. test, the reagents were obtained from Human / Germany (18).

RESULTS AND DISCUSSION

The patients with urolithiasis were divided according to the chemical composition of stones, (which were free from magnesium ammonium phosphate "MAP"), into 4 groups:

Group 1 and 2 : The composition of stones was Ca Ox as pure or combined with hydroxyl apatite.

Group 3 and 4 : The uric acid crystals may act as a nucleus for the deposition of Ca - ureate or Ca Ox - $CaPO_4$.

The most commonly calcium oxalate stones predominate with small quantities of calcium phosphate and uric acid, this was observed in group 4. Most of the patients had bilharziasis (72.5%). Many investigators suggested that bilharziasis increases the incidence of urinary tract infection and in turn the stone formation , especially multiple urolithiasis (1) . Furether , **Abdel -Aziz et al.,** (2) found that, urinary schistosoma and the resulting immunopathological changes were factors predisposing to formation of renal stones.

The results demonstrated that (Table 1) the bilharzial patients with calcium - stones were 83.3% , 78.6% , 46.6% and 68.8% of the patients of groups 1-4, respectively. The incidence of bacterial infection in bilharzial patients was 40.8%, 33.8% of urine and surface of stone samples, respectively, as compared to 16.9%, 15.5% in their corresponding samples of non - bilharzial patients. Thus the bilharziasis increases the incidence of infection rate in the urinary tract, which is leading to or contributing in calculus formation. Most of the isolated stones 45 out of 71 stones were removed from kidney, 25 stones from bladder, thus the majority of stones were formed in kidney.

In group 1 , most of the stones were removed from kidney (8 out of 12 Ca Ox stones) . However, **Holmgren et al.** (19) reported that most of the Ca Ox stones were located in ureter . Stone recurrence was found in 58.3% of all patients , 83.8% of patients had bilharziasis . Bacteria was found in 66.7%, 58.3% of all urine and stone (surface) samples , respectively . **Gault et al.** (20) stated that , positive cultures were common in the oxalate stone formers . **Shortiffe et al.** (21) suggested that calcium oxalate and other sterile stones in the urinary tract may become infected with urea - splitting or non - urea spliting bacteria during a urinary tract infection. **Sabinksi and Leusmann** (5) found that, calcium oxalate stone may be infected with microorganisms , which were not able to split urea . In

Table (1) : Type of stone , its location and percentage of recurrence inc ases with or without bilharziasis.

Stone type	Kidney stone (n=45)		Urter stone (n=25)		Bladder stone (n=1)		Recurrence
	Bilharzial casses (%)	Non bilharzial	Bilharzial	Non bilharzial	Bilharzial	Non bilharzial	
Ca. Ox (12 stones)	50.0	16.7	33.3	-	-	-	58.3
Ca.Ox-Ca. P (28 stones)	39.3	10.7	39.3	10.7	-	-	35.7
Ca.Ox-U (15 stones)	33.3	46.7	13.3	6.7	-	-	60.0
Ca. Ox-CaP-U (16 stones)	37.5	31.3	25.0	-	6.3	-	62.5

the light of these results, the matrix theory of calcium oxalate stone development with need some modifications.

On the contrary , **Holmgren et al.** [19] reported that the least frequency of infection was found in urine of patients with Ca Ox alate stones.

Figure (a) , shows the *Escherichia coli* was the most present organism in stone (surface) samples of group 1, *E. coli* is non-urease producer. Mixed infection (more than one organism) was found in 16.7% of urine samples.

In group 2 (with Ca Ox - Ca P stones) , 50% of stones were located in kidney, 78.6% had bilharzia. Stone recurrence rate represented 35.7% of all patients, this reflects the lower bacterial infection in this group than the first group. Thus the incidence of bacterial infection was 53.6% of urine samples while in surface of stones was 39.3%. The organism that infected most stone (surface) and urine samples of this group was *Pseudomonas* (non - urease producer species) (Fig. (b).

In group 3 , patients with Ca Ox - U stones, 80% of stones were removed from kidney. Stone recurrence rate represented 60% of all patients , 46.6% had bilharziasis . Bacteria infected 46.7% of both urine and stone samples. Figure (c) shows that *Staphylococcus epidermidis* was the most common strain (20% in stone and 13.3% in urine samples).

In patients with Ca Ox - CaP -U stones (group 4), 68.8% of the stones were located in kidney in cases that had bilharzia.

The incidence rate of stone recurrence was 62.5% , this reflects the highest bacterial infection in stone samples, bacterial infection was 62.5% of both urine and stone samples. Similar finding was reported by **Shortliffe and Spigelman** [22] . *Enterococcus* spp. was the prevalent bacteria in 18.8% of stone (surface) of group 4 (Fig. (d) . *E. coli* was the major strain (18.8%) isolated from urine samples . Also it was isolated from all groups (1-4), inspite of it is non - urease producer *Proteus mirabilis* (urease producer) was isolated only from group 3. *enterobacter* was isolated in low frequency in both urine and stone samples. It has been found that. *Alcaligenes* was isolated from urine samples with Ca Ox - U stone , similar observation was found by **Durlach et al.** [23] who isolated *Alcaligenes oderance* from 2 cases .

In the present study , serum total protein was estimated Fig. (1a) . It has been shown that groups 2 and 3 had moderately significant increased levels in the serum total protein, while slightly significant increased level was obtained in groups 1 and 4 , as compared to the normal subjects .

Figure (1b) showed that serum albumin was decreased several folds in groups of patients (1-4) . The observed hypo- albuminemia was associated with hyper - albuminuria due to renal tissue involvement under the effect of calculus formation [24] .

From Fig. (1c, 1d, 1e , 1f) , the data obtained for serum α_1 - α_2, β- and γ - globulin, in group from 1-4 , showed highly significant increased level due to the effect of acute phase response to inflammation. **Latner** [25] found that slight to moderate increases in one or all of the three globulin fractions may occur, inconsistently, in acute glomerulonephritis . Bacterial infection and active tissue damage lead to increase of globulin fractions, consequently serum albumin was decreased simultaneously . Glomerular damage allows the escape of serum albumin , IgG and transferrin in the urine [9].

In comparison to the normal healthy persons, α_1-glycoprotein (seromucoid) showed insignificant value Fig. (2a) in patients of group 1, very high signifciant decreased in group 3 . The decrease in seromucoid may be attributed to the type of the infected microbes in urine or stone samples.

Fig.(a):Percentage of occurrence of different microorganisms in urine and stone(surface)sample with Ca-Ox.

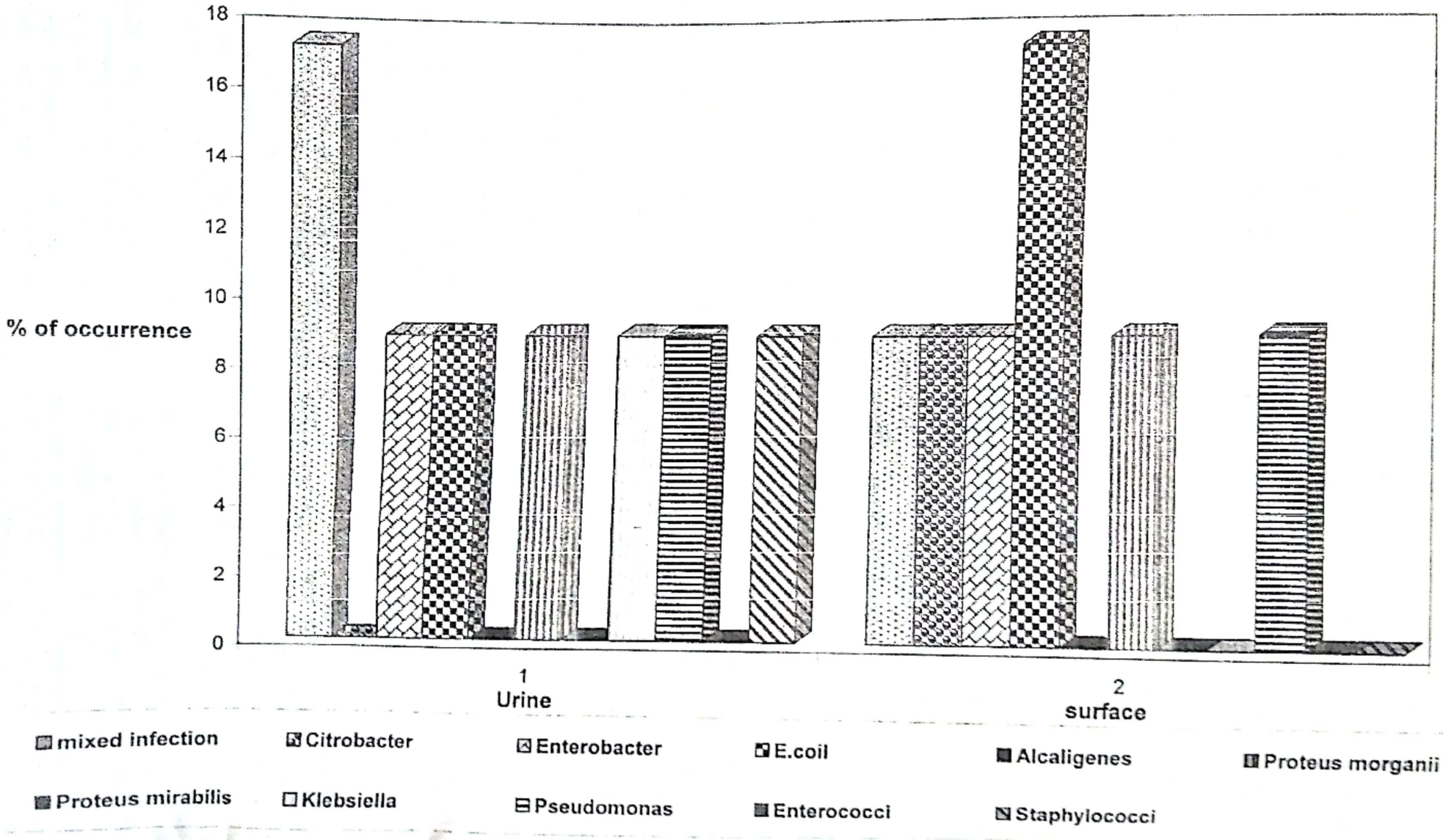


Fig.(b): Percentage of occurrence of different microorganisms in urine and stone(surface)samples with Ca-Ox + Ca-P.

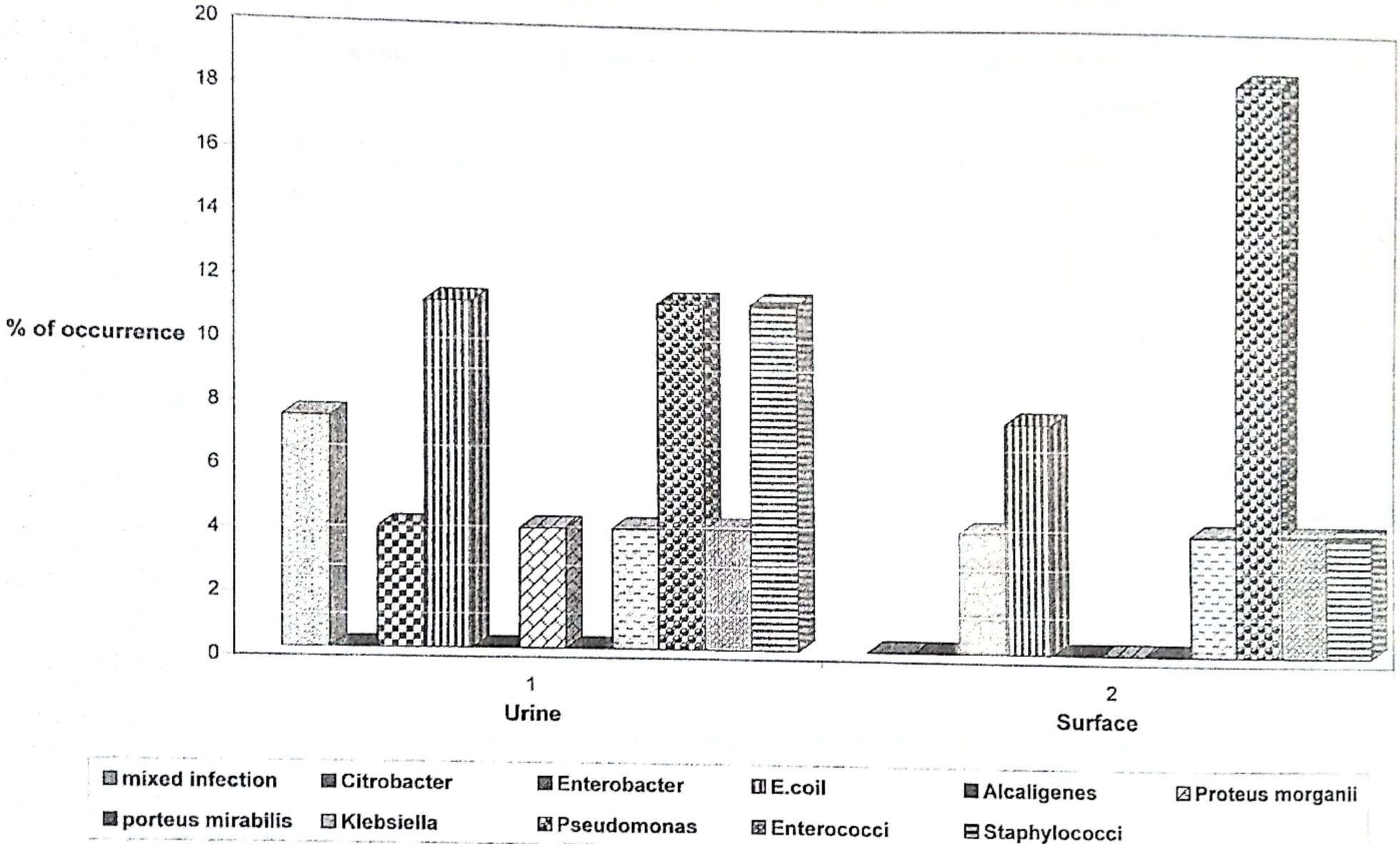


Fig.(c): Percentage of occurrence of different microorganisms in urine and stone(surface)samples with Ca-Ox + Uric acid.

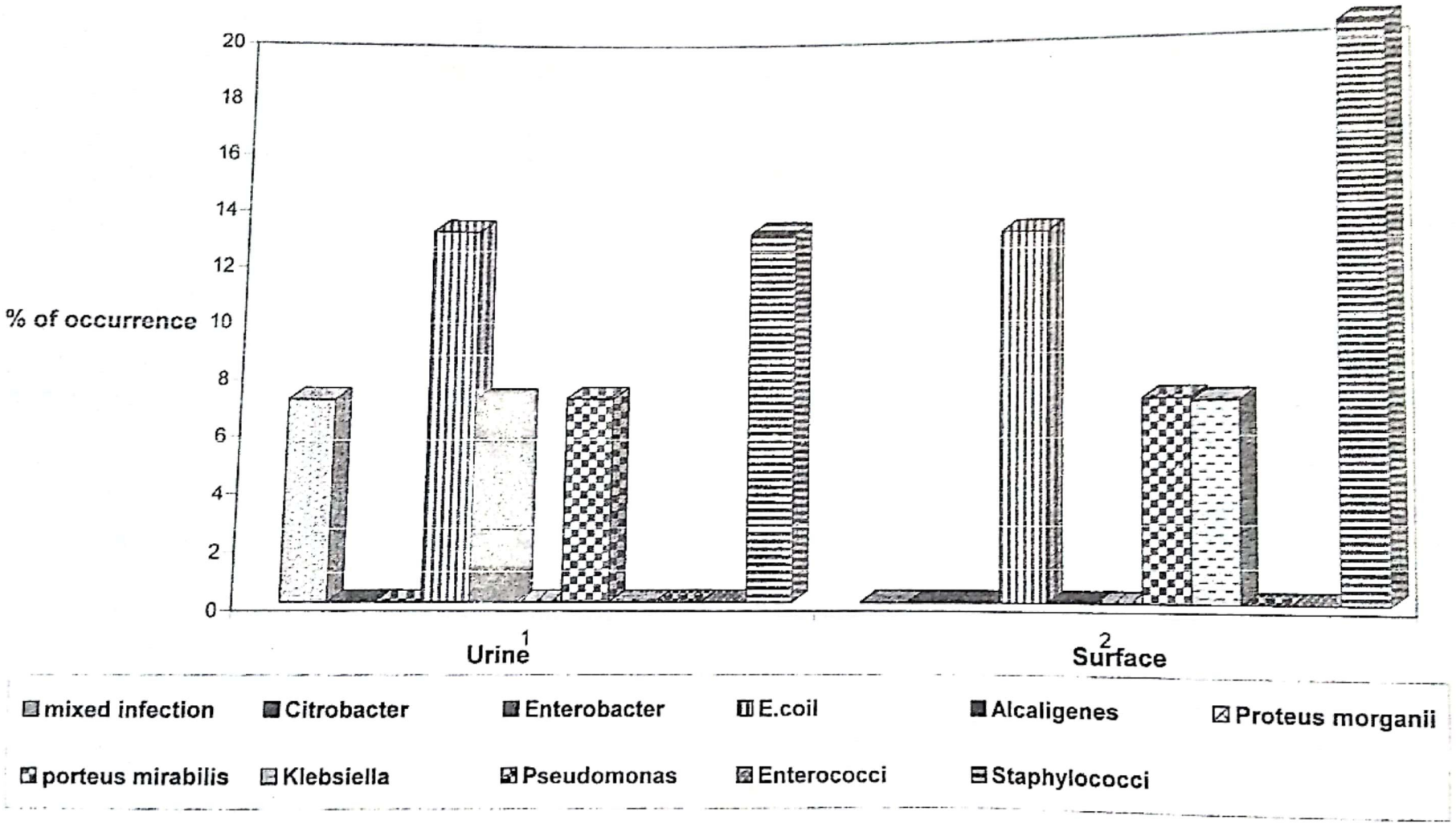
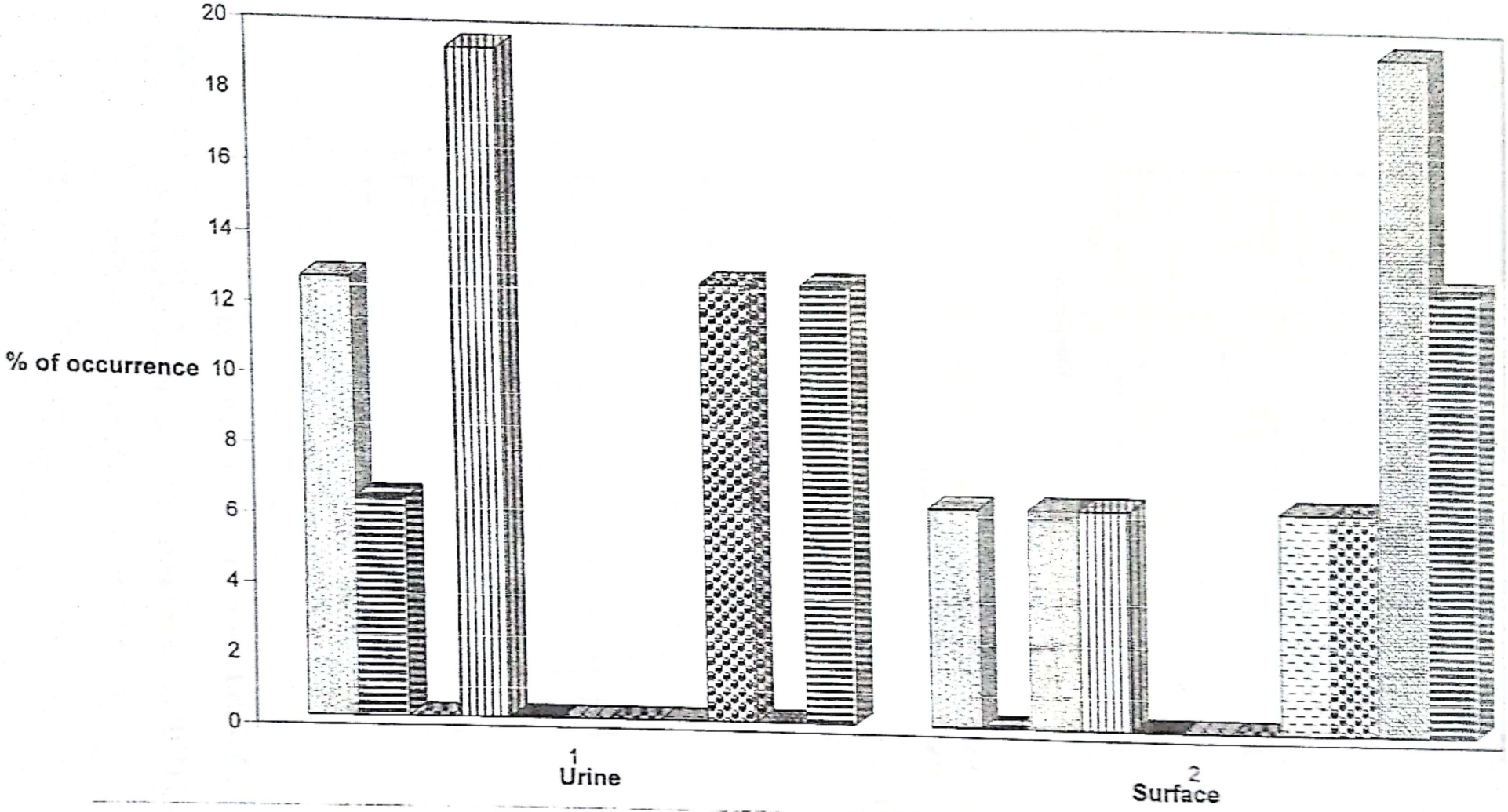
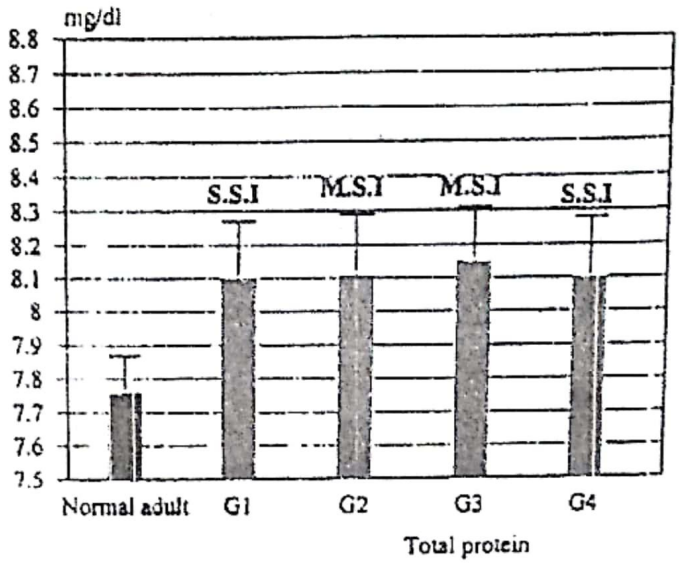


Fig.(d): Percentage of occurrence of different microorganisms in urine and stone(surface)samples with Ca-Ox + Ca-P + Uric acid.

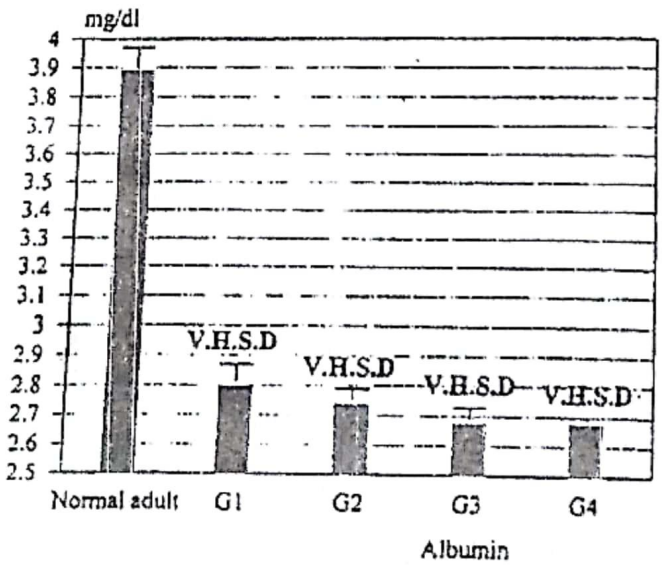




S.S.I = Slightly Significant Increase

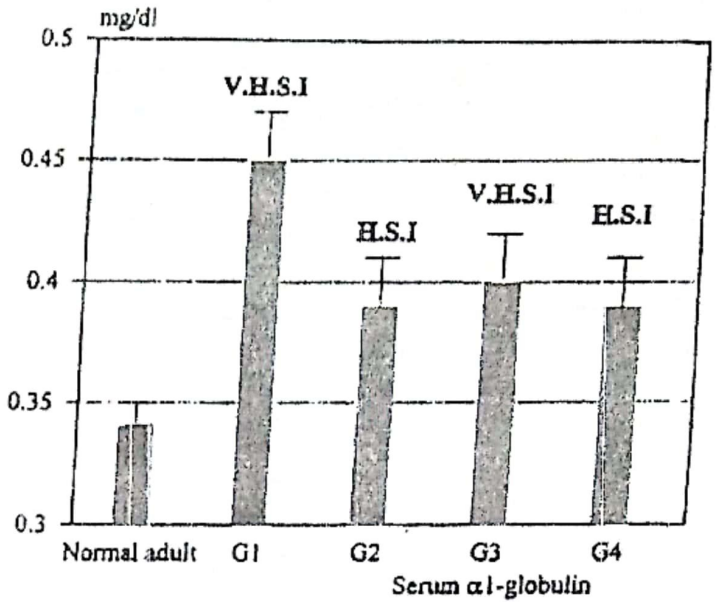
M.S.I = Moderate Significant Increase

Fig. (1a): The mean values ± S.E of serum total protein concentration (mg/dl) in the different studied groups.



V.H.S.I = very high significant increase

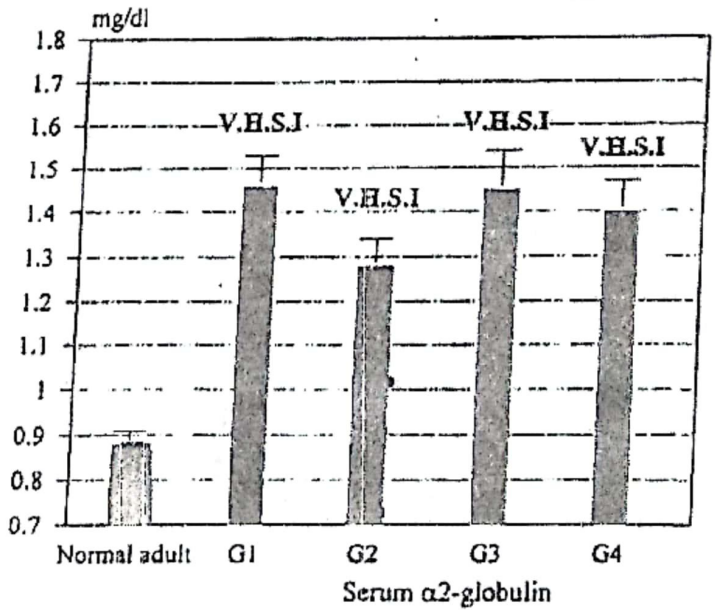
Fig. (1b): The mean values ± S.E of serum albumin concentration (mg/dl) in the different studied groups.



V.H.S.I = Very High Significant Increase

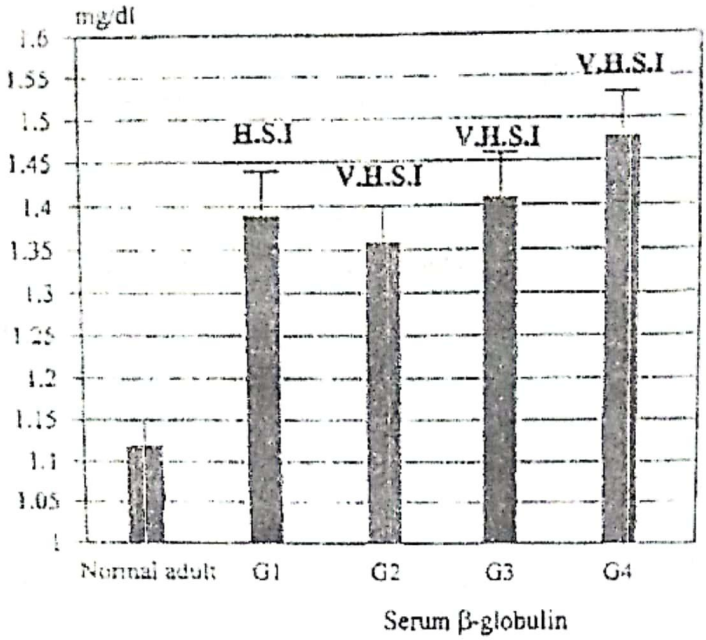
H.S.I = High Significant Increase

Fig.(1c): The mean values ± S.E of serum α_1-G concentration (mg/dl) in the different studied groups.



V.H.S.I = Very High Significant Increase

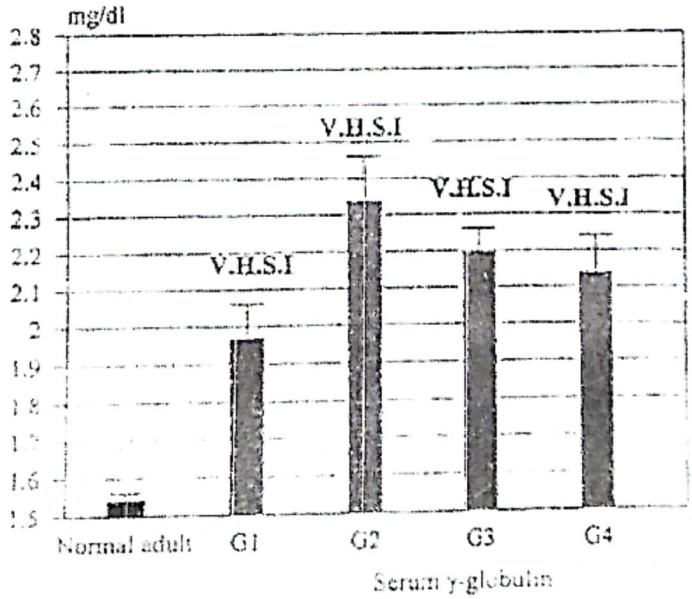
Fig. (1d): The mean values ± S.E of serum α_2-G concentration (mg/dl) in the different studied groups.



H.S.I = High Significant Increase

V.H.S.I = Very High Significant Increase

Fig. (1e): the mean values ± S.E of serum β- G concentration (mg/ dl) in the different studied groups.



V.H.S.I = very high significant increase

Fig. (1f): the mean values ± S.E of serum γ-G concentration (mg/dl) in the different studied groups.

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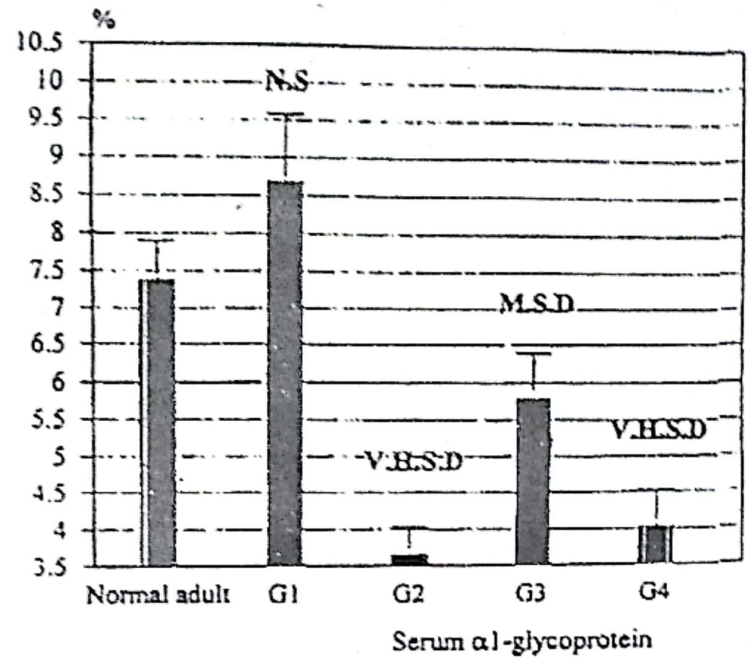
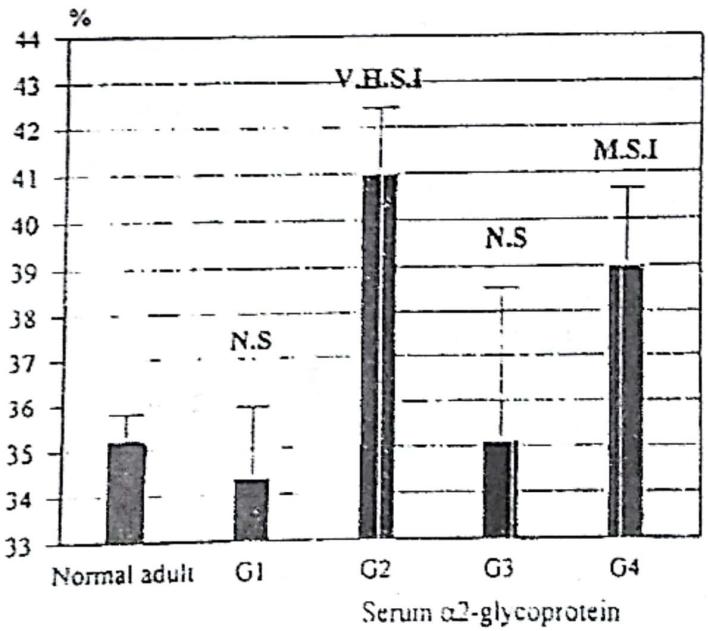


M.S.D = Moderate Significant Decrease

V.H.S.D = Very High Significant Decrease

N.S = Insignificant

Fig. (2a): The mean values ± S.E of serum α_1-glycoprotein concentration in the different studied groups.

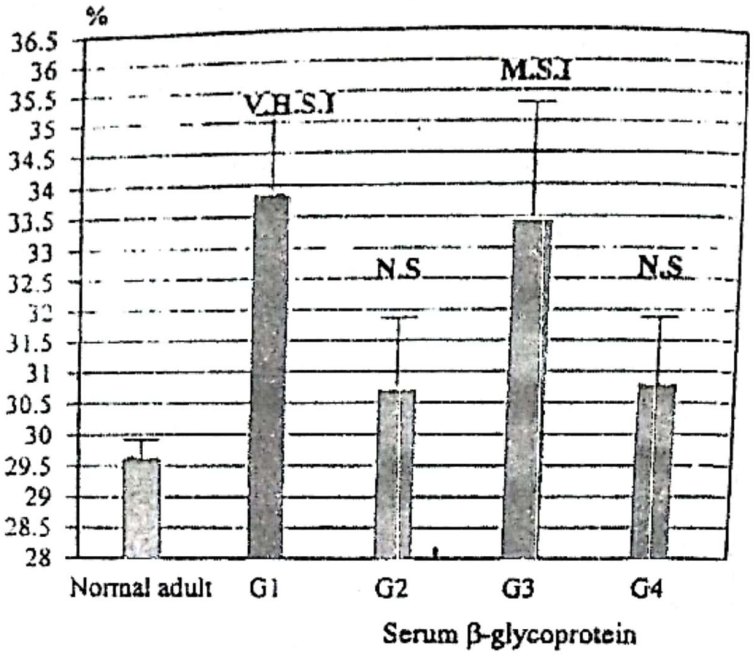


V.H.S.I = Very High Significant Increase

N.S = Insignificant

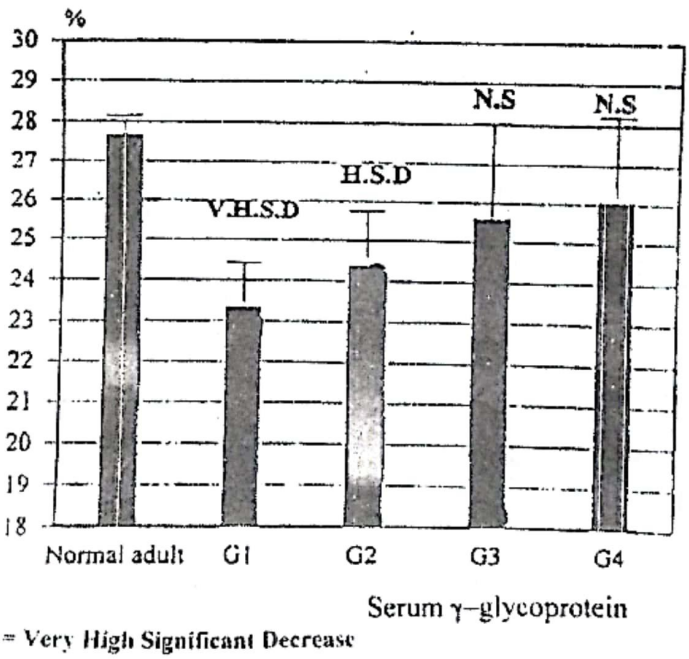
M.S.I = Moderate Significant Increase

Fig. (2b): the mean values ± S.E of serum α_2- glycoprotein concentration (mg/dI) in the different studied groups.



V.H.S.I = Very High Significant Increase

Fig. (2c): the mean values ± S.E of serum γ - glycoprotein concentration (mg/dl) in the different studied groups.

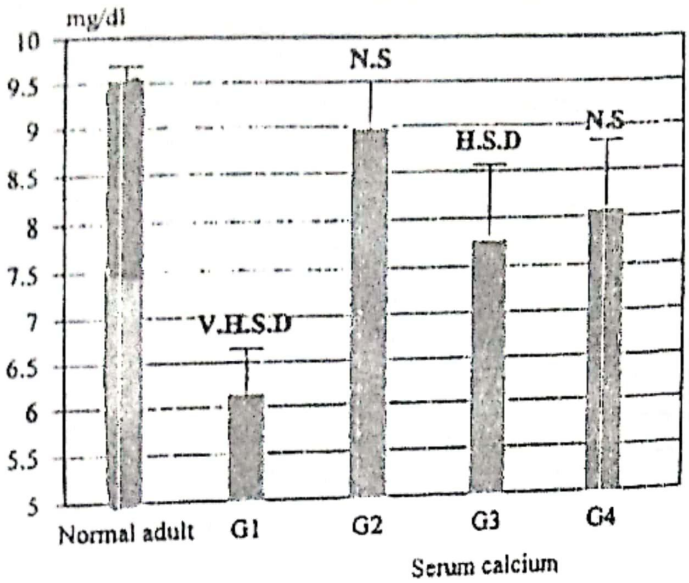


V.H.S.D = Very High Significant Decrease

H.S.D = High Significant Decrease

N.S = Insignificant

Fig.(2d): the mean values ± S.E of serum γ-glycoprotein concentration in the different studied groups.

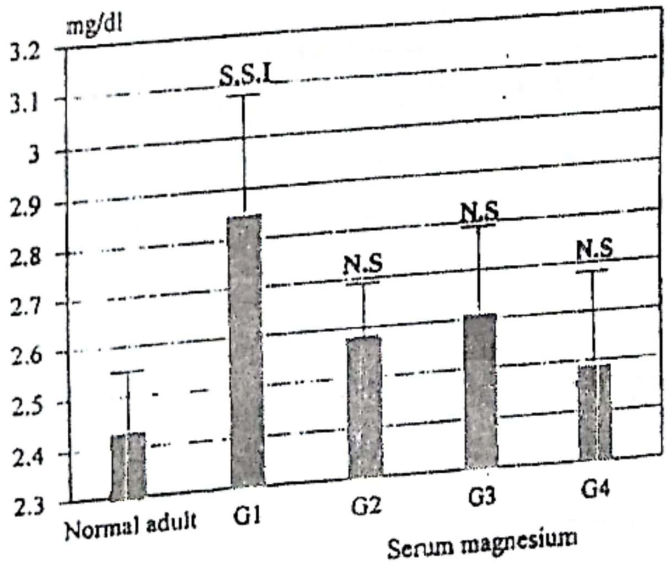


V.H.S.D = Very High Significant Decrease

N.S = Insignificant

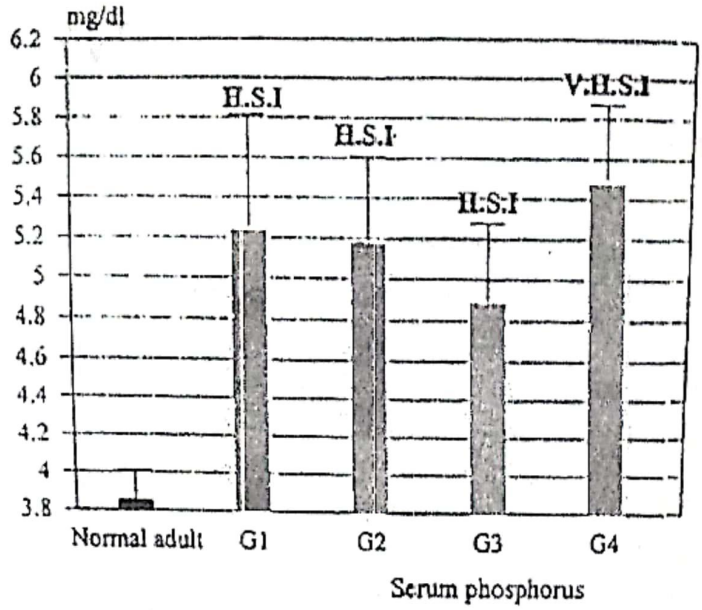
H.S.D = High Significant Decrease

Fig. (3a): The mean values ± S.E of serum calcium concentration (mg/dl) in the different studied groups.



S.S.I = slightly significant increase
N.S = insignificant

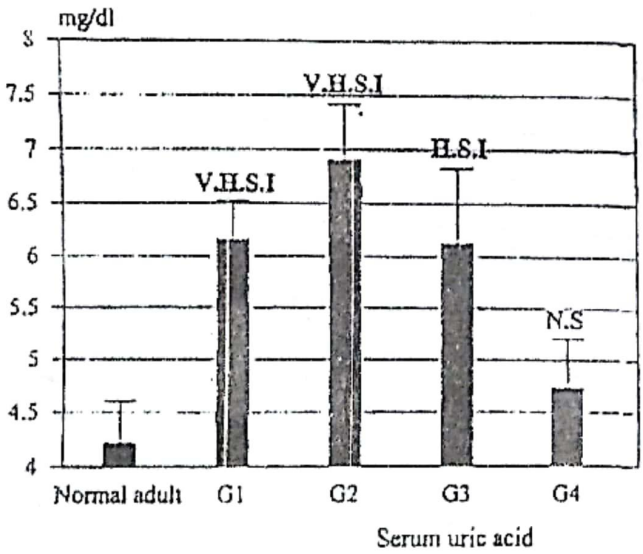
Fig. (3b): The mean values ± S.E of serum magnesium concentration (mg/dl) in the different studied groups.



H.S.I = High Significant Increase

V.H.S.I = Very High Significant Increase

Fig. (3c): The mean values ± S.E of serum phosphorus concentration (mg/dl) in the different studied groups.



V.H.S.I = Very High Significant Increase

H.S.I = High Significant Increase

N.S = Insignificant

Fig. (3d): The mean values ± S.E of serum uric acid concentration (mg/dl) in the different studied groups.

The alpha 2-glycoprotein revealed insignificant change in groups 1 and 3 whereas in groups 2 , 4 Fig. (2b).(Ca P was found in the 2 groups), significant increased level was observed. This due to tissue destruction and inflammation. In the meantime, **McCrory and Fleisher**[26] found that, differential changes in glycoproteins associated with various electrophoretic components occur in viral or bacterial infections, the rise in these substances reflects the degree of the severity of damage.

In β-glycoprotein , insignificant difference in both groups 2 and 4 was obtained Fig. (2c), moderately significant increased level in group 3 and very high significant increased level in group 1, as compared to the normal value . Serum γ - glycoprotein showed insignificant change in groups 3 and 4 , while highly significant decreased levels were obtained in groups 1 and 2 as compared to normal level Fig. (2d). Albumin fraction was absent in both healthy subjects and the stone formers.

The results obtained for calcium , magnesium , phosphorus and uric acid in the sera Fig. (3a), (3b) (3c) are in agreement with the corresponding data reported by **Chernecky et al.** [27] . In patients of groups 1 and 3 , the level of serum calcium was highly and signifiacntly decreased and this decrement of calcium may be attributed to the withdrawing of calcium from serum and deposited to from crystals, or the decreasing of calcium may parathyroidism. However, serum calcium showed insignificant changes in groups 2 and 4.

Serum magnesium revealed slightly significant increased level in group 1 and non - significant change in groups 2,3 and 4 . Magnesium salts do not participate in stone composition of these groups.

The increment of phosphorus level in serum was showed in all studied groups. This could be attributed to either hypo- parathyroidism associated with a low serum calcium [27] , or to the destructive kidney lesions as pyelonephritis due to bacterial infection.

In spite of uric acid which was not participating in stone formation of groups 1 and 2 a highly significantly increased level of this acid was observed in serum Fig. (3d) . Group 4 showed insignificant value , while in group3, uric acid showed highly significant increase . **Freeman** [28] showed that the formation of uric acid in stones could be due to the increased value of uric acid in serum.

In conclusion, the incidence of bacterial infection was high in urine and stone samples of bilharzial patients as compared to non- bilharzial ones. Bilharziasis frequently plays an important role in the formation of stones in cases associated with bacterial infection.

Non - urea spliting bacteria such as *E. coli* , *enterobacter* , *Citrobacter* and some strains of *Pseudomonas* were isolated from stone samples (surfaces) , especially *E. coli* was the most type isolated from urine and stone (surface) samples . Some investigators suggesed that this organism was found as secondary infection and non- stone producer.

Serum total protein and their fractions were significantly high, except albumin fraction, as compared to the mean normal value . This may be the result of infection, chronic inflammatory states and active tissue damage (acute phase reaction) . The increased level of both α_2- and β- glycoprotein was associated with high bacterial infection and incidence of acute inflammation. The stone recurrence rate was frequently high in the patients associated with chronic urinary tract infection, This shows that the bacterial infection plays an important role in the formation of stone.

REFERENCES

1-Bazeed, M. A. ; Nabeen, A. and Atwan, N.: Xanthogranulomatous pyelonephritis in Bilharzial patients : A report of 25 cases. J. Urol. 141 (2) : 261 (1989).

2- Abdel - Aziz, M. K. ; Ekiaby, A. P. ; Mangoud, A. M. ; Eissa M. F. ; Salam, M. M. and Morsy, T. A.: Schistosomiasis affection of the kidney as a possible cause of renal stone formation. **J. Egypt Soc. Parasitology** , 1 (2): 187-195 (1991).

3-Grasso, M. ; Shalaby, M. ; El-Akkad, M. and Bagley , D. H.: Techniques in Endoscopic lithotripsy using pulsed dye laser. **Urology**, 37 (2) : 138 (1991).

4-Giannakopoulox , X .; Evangelou, A. ; Tsoumanis, P.; Papadopoulou C. ; Charamboupoulos, C. and Antoniadis , G.: Urinary infection in urolithiasis patients in the epirus district (northeastern greece) . **Am- Urol - Paris**, 30 (3) 118-123 (1996).

5- Sabinski, F. and Leusmann, D. B. : Potential contribution of optional urease positive bacteria to idiopathic urinary calcium stone formation, I. expression of urease Activity in bacteria from the urinary tract that are commonly classified as urease negative. **Urol. Res.** 24 (1) : 51 (1996).

6- Black Lock, N. F.: The epidemiology of renal lithiasis in : Urinary calculus disease (edited by Wickham, J.E. A) chap.2, pp. 21-39. Churchill Livingstone. (1979).

7-Menon, M. and Mahle, C. J.: Oxalate metabolism and renal calculi J. Urol. 127 : 148 (1982).

8-Lonsdale, K.: Human stones. **Science**, 150 : 1199-1206 (1968).

9-Keogh, J. B. ; Saltarelli, C. G. ; Blatt, A. and Murphy, G. P.: A new quantitative procedure for proteinuria. **Invest. Urol.** 14 : 446 (1977).

10- Edwards , P. R. and Ewing, W. H. : Identification of enterobacteriaceae . Burgess Publishign Co. Minneapolis. (1972).

11-Hawetz , E. ; Melnick, J. L. ; Adelberg, E. A. Brooks, G. F.; Butel, J. S. and Omston, L. N.: Medical Microbiology, 19 th ed., Lange Medical Book (1991).

12- Sligh, D. J. and Timbury, C. M. : Medical bacteriology; 4th ed. London. (1994).

13-Wooton, . Microanalysis in Medical Biochemistry 5th ed. Churchill, London. (1974).

14- Carlstrom, A. and Johansson, B. G. : Agarose Gel Electrophoresis- Immunofixation. **Scand. J. Immunol.**, 17, Suppl. 10 : 23 - 32. (1983).

15-Nehi, V. S. and Penn, G. M. : Electrophoretic and immunological charactenzation of immunoglobulins. In Manual of clinicial laboratory immunology (eds. Rose. N. R. ; Friendman, H. and Fahey , J. L.) 3rd ed , p. 126 . The C. V. Mosby Company, Philadelhia. (1986).

16-Town, et al., **J. Clin. Chem. Clin. Biochem**. 23 : 591. (1985)

17-Gindler, E. ; Melvin, K. and John, D. : Rapid colorimetric determination of calcium in biological fluids. **Am. J. Clin. Path.**, 58 : 376 (1972).

18-Gamst, O. and Try, K. : Determination of serum phosphate without Deproteinization by Ultraviolet Spectrophotometry of the Phosphomolybdic acid Complex. **Scand. J. Clin. Lab. Invest.**, 40: 483-486 (1980).

19-Holmgren, K.; Danielson, B. G. ; Fellstrom, B. ; Lunghall, S. ; Niklasson, F. and Wikstrom, B. : The Relation between urinary tract infections and stone composition in renal stone - formers . **Scand. J. Urol. Nephrol.**, 23 : 131-136 (1989).

20-Gault, M. H. ; Paul, M. D. and Longerich, L. : Comparison of urinary tract infection in calcium oxalate and calcium phosphate stone formers . **Nehron**, 55 (4): 408 (1990).

21-Shortiffe, L. D.; McNeal , J.; Wehner, N. and Stamey , T. A.: Persistent urinary tract infections in a female with bilateral stone. **J. Urol.** 131 : 1147-1151 (1984).

22-Shortiffe, D. M. L. and Spigelman, S. S. : Infection stones: Evaluation and Management . **Urol. Clin. of North Am.** 13 (4) (1986).

23-Durlach, R. A. ; Toblli, J. E. ; Gigler, C. ; Domeq, P. ; Vazequez, R. Cucci, V. ; Romas, H. and Ghirlanda , J. M.: Staghorn renal lithiasis treated with shock waves. **Bacteriologic Aspects. Medicina B. Aires** , 54 (5 PT1) 411 (1994).

24-Ibrahim, M. A. ; Shaker, M. Y.; El-Hawary, S. F. ; Fayek, K. I. Zahran, M. M. and El-Shawarby, K. N.: Immunochemical studies of serum , Urine, and Calculus protein in urolithiasis . **Clin. Physiol. Biochem.** , 3 : 16-22 (1985).

25-Latner, A. L. : Serum protein abnormalities in disease. in: Clinical biochemistry (eds. Cantarow & Tumper) 7th edition, p. 200 W. G. Saunders Company. London.

26-McCrory, W. W. and Heisher, D. S.: The nephrotic syndrome . Recent advances in pediatrics , 2nd edition (edited by gairdner D) Churchill limited , London. p. 244 (1958).

27- Chemecky, C. C. ; Krech, R. L. and Berger, B. J. : Protein electrophoresis in serum . In : Laboratory tests and diagnostic procedures : 778-780. W. B. Saunders Company, USA (1993).

28- Freeman, M. R. : Genitourinary tract . In : Current Medical Diagnosis and Treatment (Eds., Tiemey , M. L. Mc Phee, J. S.; Papadakis, A. M. and Schroeder, A.S.) International Edition , p. 707 , Hall International Inc. (1993).

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التلوث الميكروبى والبلهارسيا كعوامل خطورة فى مرض حصوات الأوكسالات البولية

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تسعة وستون من المرضى المصابين بحصوات المسالك البولية (٧١ حصوة أو كسالات) بعضهم كانوا مصابين بالبلهارسيا والبعض غير مصاب . تم تقسيم الحالات تبعاً للتحليل الكيميائى للحصوة إلى ٤ مجاميع . أجريت عليهم فحوص بيوكيميائية وميكروبية.

وجد أن ٤ر٧٠٪ من مكونوا الحصوات كانوا من مرض البلهارسيا . وأن معدل تكرار تكون الحصوات كانت فى ٧ر٥٠٪ من المرضى الذين يعانون من التلوث الميكروبى فى الجهاز البولى . سيدوموناس وشريشياكولاى وستافيلوكوكاى وإنيتركوكاى من أكثر الميكروبات التى تم عزلها من عينات بول المرضى ومن سطح الحصوات. أوضحت النتائج البيوكيميائية أن هناك إرتفاعاً فى مستوى البروتينات الكلية بالسيرم بالمقارنه مع أشخاص أصحاء وما عدا الألبومين. وكان مستوى الألفا ٢- جليكوبروتين أعلى من الطبيعى فى المجموعتين ٢ ، ٤ (التى تحتوى حصواتهم على فوسفات الكالسيوم) أما بيتا - جليكوبروتين فكان مرتفعاً فى المجموعتين ١ ، ٣ التى تكون حصوات أوكسالات الكالسيوم مع أو بدون حامض اليوريك . وقد لوحظ أن هذه الزيادة كانت مصحوبة بتلوث بكتيرى وإلتهابات حادة. وفى هذا البحث تأكد أن البلهارسيا التى تزيد من العدوى الميكروبية تجرى البول وبالتالى من حدوث تكون للحصوة وتلوثها وتكرارها. وأن معدل تكرار تكوين الحصوة كان غالبا عاليا فى المرضى بالتوث الميكروبى المزمن لمجرى البول . وهذا يشير إلى أن التلوث البكتيرى يلعب دوراً هاماً فى تكوين الحصوات.