

UROLITHIASIS AT THE
KENYATTA NATIONAL HOSPITAL
NAIROBI, JANUARY 1978 - DECEMBER 1987

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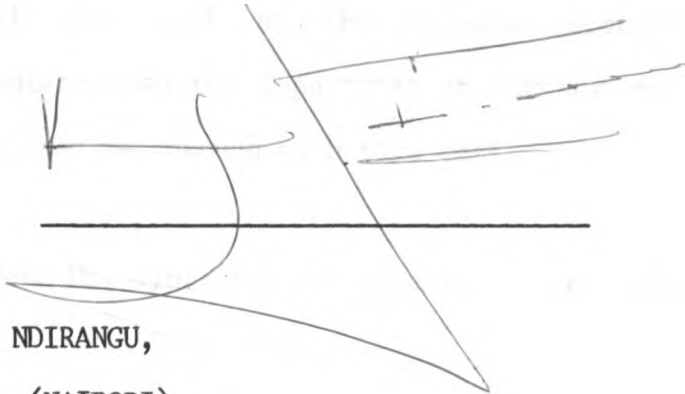
DECLARATION

I hereby certify that this dissertation is my own original work and has not been presented for a degree in any other university.

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DR. NICK Y. NGWANYAM (M.D.)

DEDICATION

To my parents: JEREMIAH & MAGDALENE NGWANYAM,
my wife : FLORENCE TITU and
my children : REMMY ROYEH and
CYNTHIA BERI

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SUMMARY

We carried out a retrospective study to establish the epidemiological and clinical picture of urolithiasis and its management at the Kenyatta National Hospital General Surgery Units.

82 patients with 89 episodes were seen and managed for stone disease in 10 years with an annual incidence of 8.9. The sex ratio is 4.5 males: 1 female. The majority of the patients have only one episode of severe stone disease mostly in the bladder. The left and right are equally predisposed.

Pain, dysuria, frequency, retention, infection and haematuria are quite common. Urinalysis, plain abdominal x-rays, intravenous urograms and cystoscopy are quite adequate for establishing the diagnosis.

Almost all patients are hospitalised for about a month or more and cystolithotomy is the surgical procedure that is most indicated. Only a tenth of our patients are not operated. No associated deaths have been noticed.

Two-thirds of the stones are single and only a tiny proportion of those are staghorn. Calcium and oxalate predominate in the formation of the stones. Pure uric acid stones are hardly seen and cystine is inexistent.

C H A P T E R 1

INTRODUCTION:

Urinary stone disease is an ancient affliction. However, the character, and clinical manifestations have varied with culture and societal changes (Donald, 1981). Hans-Joachim (1982) observes that great differences prevail in various countries concerning opinions on its frequency, distribution of types, localisation and ratio of sexes, and age of patients. Bladder stones used to predominate and children were affected most often.

Uroliths (stones) are solid structures that arise from disturbances of the physico-chemical balance and/or of the hydrodynamic system of the urine, and the urinary tract from the collection system down to the urethra. These structures have minimal size of 1,000um and consist mainly of crystalline and to a lesser degree, of amorphous organic and/or inorganic components, which may be mixed with a non crystalline high molecular substance (Matrix) (Hans-Joachim, 1982). Urinary calculus is thought by many to be very rare or absent in blacks. Some report that they did not see stones in any black African during years of urologic practice in East Africa (Tshipeta et al 1983).

There is a lot of literature in the journals about urolithiasis in many parts of the world especially Europe (McClean, 1980; Christian et al, 1982), America (Royce et al, 1987; John et al 1985) and Japan (Takuo et al, 1982). Most of the literature from 1980 deals more with the pathophysiology of the problem (Try, 1981; Fellstrom, 1983) as well as advanced techniques in its management (McClean, 1980; Bernard, 1982). There is very little on the epidemiological and clinical aspects (Power et al, 1987; Verbaeys et al, 1985; Scott, 1987). Some treat specific complications (Goldwasser et al, 1985; Bishop et al, 1987).

Very little of the works have been carried out in the African continent (Reddy et al, 1985; Kitonyi, 1986). Few isolated cases can be picked up in Index Medicus (Kampal, 1981, Tshipeta et al, 1983) and in the literature review of such articles. Most of these works certainly end up unpublished in University and hospital libraries as thesis and observations. The facts are that such information is not available to the scientific world.

As from 1980, some of the published works on this topic have included that of Tshipeta et al in Zaire (1983) who did a study on urolithiasis in black Africans in that zone. Kampal in Khartoum (1981) looked at the relation of urinary bilharziasis to vesical stones in children. A recent study from Nigeria by Udeh (1987) deals with urinary calculi in

general; stating their experience with this pathological entity in the Eastern states of Nigeria.

The only available study on this subject in East Africa is that of Kitonyi (1986). It had been observed in a pilot study that along the Kenyan coast the male incidence of renal calculosis as seen on X-ray tended to be more than in females. He therefore, reviewed 1453 excretion urographies performed in the Department of Diagnostic Radiology of the Kenyatta National Hospital between January 1982 and December, 1984. He came to the conclusion that the male incidence of renal calculosis is approximately twice that in females.

Kitonyi's population ranged from 3 months to 85 years and 90% of patients were above 14 years. 4.8% of the males and 2.5% of the females examined had one or more renal calculi. Calculi were most frequently found in the bladder. 5 of the 6 patients with prostatic enlargement had calculi in the bladder. 4 of these males had bilateral upper urinary tract calculi and were all above 50 years. None of the females had bilateral calculi. In his discussion, Kitonyi says that over 90% of renal calculi are radiopaque, that renal stones are uncommon in children and that caucasians are more prone than Negroes. He under scores the fact that very little work on renal tract calculi has been done in the African population.

It is this paucity in the availability of such works that motivated me to study the epidemiological and clinical picture of urolithiasis at the Kenyatta National Hospital. This is intended to highlight the salient aspects of this disease in this community. I hope that the findings, conclusions and recommendations that would be deduced from the study will be applicable to the problem as it is here and the neighbouring countries with similar patterns.

1.1 AIM:

To establish the epidemiological and clinical picture of urolithiasis and its management at the Kenyatta National Hospital Surgical Units.

1.2 SPECIFIC OBJECTIVES:

- 1.2.1 To establish the incidence of stone disease in the Surgical Units.
- 1.2.2 To highlight the epidemiological pattern as regards age, sex, profession, residence.
- 1.2.3 To state the clinical picture and evaluate the management procedures.

- 1.2.4 To establish the magnitude of complications due to urolithiasis (morbidity/mortality).
- 1.2.5 To determine the chemical nature of the stones seen.
- 1.2.6 To recommend a public health programme for the prevention of stone disease in this and similar communities.

CHAPTER 2

REVIEW OF LITERATURE

2.1 EPIDEMIOLOGY:

In this review of literature starting from 1980, four were available on the problem in Africa: from Zaire (Tshipeta et al, 1983), Khartoum (Kampal, 1981), Nigeria (Udeh, 1987); Kenya (Kitonyi, 1986). The authors cited some African literature which was not available for the present study.

In their epidemiological approach; Tshipeta et al in Zaire found out that the causation of urolithiasis in their population remains mostly undetermined. Populations along the Zaire river appeared to be more affected and the ages ranged from 2 to 69 years, with a mean of 43 years. The sex ratio was 4:1; M:F. Obvious causes of urinary stones were demonstrated in 40% of the cases, whereas 60% did not have any somatic demonstrable causes. The lower urinary tract was involved in only 14% of the patients. In the upper urinary tract, 43% of the stones were located in the pelvis, 32% in the ureter and 11% in the kidney. The right and left side were affected equally. Calcium containing stones were most common;

calcium oxalate in 54% and calcium phosphate (along with magnesium ammonium phosphate) in 24%. Uric acid stones were found in 17% of the cases and cystine stones had a low frequency.

Urinary tract infection was present in 60% of cases; Escherichia coli in 25%, Klebsiella in 16% and Proteus mirabilis in 2.9%. Only 30% had sterile urine. Hydronephrosis and pyonephrosis accounted for 30% of the complications with most deaths associated with acute fulgurant bacteremia due to Klebsiella. Medical treatment for the stones was the most used procedure (26%). Most of the patients were lost to follow up.

Kampal (1981) found out that urinary bilharziasis plays no role in the aetiology of vesical stones in Khartoum. However, it is believed that bilharziasis may predispose to vesical stones in children. This is not usually associated with any abnormality of the urinary tract and they rarely recur after removal.

Udeh from Nigeria (1987) writes on the retrospective study of 140 cases of urinary calculi occurring in indigenes from the 4 Eastern states of Nigeria. A low incidence rate of 7 in 100,000 was established with a male: female ratio of 5:1. Renal/ureteric calculi

accounted for 79 cases while vesical/urethral calculi accounted for 16 cases.

Urinary tract obstruction was incriminated in the pathogenesis of calculi in 40% of cases, other factors being urinary tract infections (17%), hypercalciuria and hyperuricaemia (4%). Approximately 40% of cases were considered to be idiopathic calcium calculi. A generally low level of daily urinary calcium excretion was noted (mean 105.9mg/24hrs) and there was no endemic vesical calculi in children despite the general poor dietary and nutritional status of children in Nigeria.

In a similar study Emil et al (1982) in Israel noted a high family incidence of urinary calculi. The aetiology of idiopathic urinary calculi in cases in which no metabolic defect was found was never clearly established. In their review of literature, they stated the following facts:-

- Urinary calculi are known to appear with varying frequency in different parts of the world. The regions where this disease is relatively common used to be called the 'Stone belt.' Many hypothesis were forwarded to explain this phenomenon.

- Climatic conditions were often believed to play a role in the formation of urinary calculi by dehydration in areas with high temperatures.
- Drinking and nutritional habits were also considered as aetiologic factors.
- Several pathologic conditions are known to predispose to the formation of calculi such as; infection of the urinary tract, malformations, and immobilisation, however, this could hardly explain the increased incidence of this disease in certain regions.
- Some of the urinary calculi are due to metabolic disturbances such as cystinosis, oxalosis, renal tubular acidosis and hyperthyroidism. All these diseases are genetically transmitted.
- In the so-called 'idiopathic' stones, the metabolic studies did not reveal any disturbances, and the basic cause remained unknown.
- Some authors have advocated socio-economic factors as an explanation and have stressed the necessity for further studies of this problem.

Scot et al (1987) in their study on the prevalence of calcified stone disease drew attention to some sources of error. They say figures of true prevalence are difficult to obtain and with respect to stone disease it is usual to refer to incidence as being the number of patients discharged or dealt with in hospital. When the incidence of stone disease is taken from hospital statistics it is clearly dependent upon a number of variables such as the availability and quality of radiological backup, the aggression in pursuit of the diagnosis and the awareness and reference of patients from general practitioners. An example of the difficulties of interpretation of stone incidence by these methods is that where there are special centres interested in stone disease, patients will gravitate towards these hospitals.

A paediatric survey in the 1970s by Churchill et al (1980) yielded some interesting results. They state that there are many causes of urolithiasis in children. Endemic lower urinary tract calculi, common throughout the world in the nineteenth century are now found primarily in developing countries and the prevalence correlates well with protein malnutrition. The sex ratio was 1:1 and the mean age was 9.4 years. A positive family history was found in 36% of the cases. Urinary tract abnormalities predisposing to infective urolithiasis was found in 18%, hypercalcemia in 8% while

74% had idiopathic urolithiasis. Cases of idiopathic hypercalcaemia were found amongst the last group. In this series they noticed that the common mode of presentation was abdominal or flank pain with or without haematuria in 79% of cases. Fever and/flank masses were present in the others.

The other extreme is that obtained in the health survey of 50 year old men followed up for 10 years. Within this period 19% had a history of renal stone formation (Ljunghall et al, 1987). In 47% there had been recurrences. These authors say though renal stones appear to be common, there is comparatively little information on the epidemiology and natural history of stones in the unselected population. Most information concerning stones stems from studies of patients investigated at specialised out-patient stone clinics or subjected to detailed inpatient metabolic evaluation.

Power et al (1987) checked on the incidence of stones in 18 British towns. Their findings were that stones in the upper urinary tract are more common in industrialised than non-industrialised countries. Some of their findings show that a greater proportion of calcium oxalate stones occur in younger men, while infective stones are common in older men and women. The effect of

water hardness, climatic factors and latitude did not exert a great influence.

Verbaeys et al (1985) paid more attention to urometabolic evaluation in patients with renal calculous disease. They had a male/female ratio of 1.59%. Their mean age was 45 years. Not one struvite stone was spontaneously eliminated in their study. All stones so passed were calcium oxalate or calcium-oxalate-phosphate. They advocate that stone formers must be evaluated urologically and metabolically. In 90% of highly selected patients a stone promoting factor was identified. In those with calyceal stones, regular urinalysis is mandatory since infection plus metabolic disorders mean that the chances for spontaneous stone elimination are significantly reduced, that operation will be necessary and that staghorn formation is possible. Hypercalciuria and hyperuricosuria were the main metabolic disorders present in most patient groups. Therefore, stone patients who are young, the operated and those who are recurrent stone formers should be investigated.

Renal stones is a condition affecting about 80% of the Irish population at some time in their lives. There are many types of stones and great variations in the incidence of each type. Calcium oxalate stones account

for 50 to 80% of all renal stones, calcium phosphate for 20% and mixed stones for approximately 10%. Uric acid, cystine, xanthine and oxalate stones account for approximately 5% (Helen et al, 1981).

2.2 PATHOPHYSIOLOGY:

Urine is a complex solution of organic and inorganic compounds, often oversaturated with salts of oxalate, urate and phosphate, even in healthy persons. For stone formation to start, there must be a seeding site in the urinary tract. For growth of the stone, continuous presence of oversaturated urine is mandatory (Try, 1981).

Proposed causes of the increasing incidence of kidney stones seen over the world have included an increased intake of proteins in Western civilizations, an increased intake of refined sugar and a decreased fluid intake. Gillenwater (1980) in this study noted that males have a 12% chance and females a 5% chance of having a stone by age 70. 44% of patients required hospitalisation for stone management and 0.4% required surgery. No seasonal variation in incidence was detected.

The majority of primary urinary stones arise in an unknown way but a few factors are worthy of consideration: increased content of crystalloids or

abnormal crystalloids in urine e.g. xanthinuria; hypercalciuria; hyperoxaluria; hyperuricosuria; hypercalciuria in hyperparathyroidism, sarcoidosis, immobilisation, and renal tubular acidosis. The mechanism of formation of calcium containing calculi in the many instances in which there is no obvious abnormality in calcium metabolism is not known. Thus the traditionally held deleterious effect of dehydration on stone formation needs reconsideration (Fredric, 1980). Other contributing factors are foci of calcification in the kidney (Randall's plaques), stasis, urinary pH; infection with urea-splitting organisms notably Proteus Mirabilis; and Vit A deficiency in rats (Fredric, 1980).

2.2.1 THE PRINCIPAL DIVISIONS OF STONE DISEASE.

The four main types are calcium, uric acid, cystine, and struvite. Most patients have calcium stones (calcium oxalate 88.6%, calcium phosphate 2.1%, calcium and uric acid 4.2%). Uric acid stones make 1.5%, cystine 0.6% and struvite 3%. The overwhelming numerical dominance of calcium oxalate stones especially has been well documented by many studies (Fredric, 1980).

2.2.2 PHYSICAL CHEMISTRY OF STONE FORMATION.

At the moment of its genesis, a stone appears in its embryonic form as a solid-phase nucleus in a region of a solution that had, until that moment, no solid phase; nucleation had thus occurred. At microscopic dimensions, a vast proportional change had occurred (Gillenwater, 1985).

Nucleation and crystal growth are complimentary and separate facets of kidney stone development. Solid phase, once formed, may undergo a coarsening process, essentially redistributive and not drawing additional material from the liquid to the solid phase, as nucleation and crystal growth do. Small particles may aggregate to form stable or unstable masses, a process called agglomeration. In a different process called ripening, small particles dissolve and their molecules are taken up by larger particles (Fredric, 1980).

2.2.3 CALCIUM STONES.

Most idiopathic stone formers have idiopathic hypercalciuria or hyperuricosuria that appear to cause stones and are amenable to medical treatment. Primary hyperparathyroidism; distal hereditary tubular acidosis, secondary hyperoxaluria, inflammatory bowel disease and sarcoidosis were some observed causes in its pathogenesis (Fredric, 1980).

2.2.3.1 CALCIUM OXALATE.

Spontaneous crystalluria is not rare in recurrent stone formers, a fact that illustrates vividly the oversaturation of their urine. Uric acid and sodium hydrogen urate, its salt, are both present in urine, and each is reasonably an efficient heterogeneous nucleator of calcium oxalate. Pyrophosphate and other substances in urine normally inhibit calcium oxalate nucleation. slow crystal growth and aggregation. The balance of the forces determines who forms stones (Fredric, 1980).

The mineralisation of calcium oxalate, which is found in more than two thirds of all human urinary stones, is complicated by the existence of 3 hydrates. Whewellite (Calcium oxalate monohydrate) and weddelite (Calcium oxalate dihydrate) are the 2 major crystalline forms in urinary calculi (Sung-Tsuen, et al, 1982). Natural inhibitors in urine have been recognised for many years and components such as polyphosphate, tricarboxylic acids, magnesium, trace metal ions, unidentified polypeptides and naturally occurring polymers have all been involved as possible agents (Sung-Tsuen, et al, 1982).

James et al (1980) analysed the sizes of calcium crystals in urine. The mean size in normal subjects was $12.0 \pm 7.8 \mu\text{m}$. Calcium oxalate monohydrate crystals are significantly smaller than calcium oxalate dihydrate ($p < 0.01$).

In the calcium oxalate stone-former the urine is sterile, the sex ratio is 3 males to 1 female subject and classification within the prostate is more frequent. The biochemical profile of the urine tends to reflect hypercalciuria with a wide range of liability, hypophosphaturia, decreased urinary citrate and hyperuricosuria in some 8 to 20% of cases. Urinary magnesium and oxalate here are not quantitatively abnormal. Of all urinary crystalloids, oxalate is theoretically the most important ion in determining calcium oxalate crystallizations and yet it is quantitatively the least variable in our experience, and has the least correlation with severity of stone formation of any of the urinary crystalloids (William, 1982).

Richard et al (1980) looked at the calcium and oxalate concentrations in human renal tissue and wondered if it had anything to do with the pathogenesis of stone formation.

They found out that there was a higher concentration of oxalate in the papillae compared to the medulla of normal kidneys. The medulla also had more compared to the cortex. Calcium showed a similar gradient. Both substances were appreciably higher in the papillae than in urine; Oxalate 25 times and calcium 6 times more.

The medulla and papillae correspond to the sites of formation of Randall's plaques. The latter are macroscopic subepithelial deposits of calcium crystals. The epithelium over the plaques is eroded and crystal growth continues into the pelvis. Intraluminal calcium deposits can evolve to interstitial positions. They came to the conclusion that the higher concentrations of calcium deposits, are the basis for at least some stone formation, and also have emphasised the importance of the renal papillae as the site of early stone formation.

Richard et al (1980) looked at the renal oxalate excretion in calcium urolithiasis. Urinary oxalate excretion was not significantly different when the diet was changed from a random to a calcium restricted diet. They state that the renal oxalate excretion in an ambulatory setting is not critically dependent on the state of calcium absorption and intake. Therefore, impositions of a low calcium absorption in stone formers does not necessarily augment oxalate excretion.

The seasonal variations in urinary excretion of calcium, oxalate, magnesium and phosphate on free and standard mineral diet in men with urolithiasis was studied by Junti et al (1981). Some review of their literature suggest that the incidence of urolithiasis depends partly on seasons and climate. They noticed that 24-hour urine volume, urinary calcium, oxalate, magnesium or phosphate did not vary significantly

with seasons on a standard mineral diet. There were significant variations on a free diet that did not differ by age. Magnesium excretion was not affected by season or age. They came to the conclusion that dietary intake probably best explains seasonal changes in the urinary excretion of calcium, oxalate and phosphate.

Wunderlich (1981) in Berlin stated that magnesium ions may induce an increase in the solubility of calcium oxalate but in contrast it may also broaden the Ostwald-Miers' range thus favouring the formation of larger crystals.

In a control study of dietary factors in renal stone formation by Griffith et al (1981) the percentage of energy derived from fat was higher in patients with stones, previous observations had suggested that dietary fiber and dietary fat may affect absorption from the intestine

of calcium and phosphate. They suggested that studies would be needed to test whether high fiber, low fat diets would be helpful in the management of patients with recurrent nephrolithiasis. Helen et al (1981) in Dublin came to the same conclusion in a similar study. They state that the cause varies with the type of stone but that the precise causative factor can be identified in only about 15% of Irish patients. Many metabolic, dietary and non-dietary factors have been identified. Some of these include: fluids, carbohydrates, animal proteins, fats, lysine, arginine, refined sugar, fiber, calcium, phosphate, zinc, aluminium, iron, copper, sulphur, magnesium, Vitamin A, Vit. B, folic acid, Vit. C, Vit. D, phytic acid, oxalate, purine, climate, sunshine and humidity.

2.2.3.2 CALCIUM PHOSPHATE

These stones are common in primary hyperparathyroidism and distal renal tubular acidosis. In the latter the urine is excessively alkaline favouring calcium phosphate stones formation. In primary hyperparathyroidism, the urine is not abnormally alkaline and the reason for calcium phosphate stones, as opposed to calcium oxalate stones are unknown. Alkali abusers and patients infected with urea-splitting bacteria may readily form calcium phosphate stones because of undue alkalinity (Fredric, 1980).

Brushite, (CaHPO_4) , is not commonly a significant component of renal calculi but it may have significance as the nidus of calcium phosphate crystals, which later mature to hydroxyapatite, $(\text{Ca}^{2+})_5(\text{OH}^-)(\text{PO}_4^{3-})_3$, the crystal usually observed in calcium phosphate stones. Brushite may also be heterogenous

nuclei for calcium oxalate (Fredric, 1980).

Try in Norway (1981) analysed stones by infrared spectrophotometry. He says urine is a complex solution of organic and inorganic compounds, often oversaturated with salts of oxalate, urate and phosphate even in healthy persons. For stone formation to start, there must be a seeding site in the urinary tract and for growth, the continued presence of oversaturated urine is mandatory. The male:female ratio was 2:1 and phosphate was found in 69.9% males and 94.2% females. Oxalate was present in 59.6% males and 39.1% females.

Calcium phosphate in saturated solutions precipitate mainly as hydroxyapatite at pH levels above 5.47. The solubility of calcium phosphate greatly increased with lowering of the pH. Increasing the ionic activity of the saturated

solution with sodium chloride increases the solubility of calcium phosphate only to a limited extent (Try, 1981).

2.2.3.3. STRUVITE (STAGHORN CALCULUS)

The formation and solubility products of this crystal; magnesium ammonium phosphate (MAP); are very close together. Normal people and stone formers without urinary infection do not exceed these products. Most of the stones occur when the urinary tract is infected with urea-splitting organisms such as Proteus mirabilis. The concentration of ammonium is greatly increased; the urine pH is high and increases ionized phosphate by removing protons so that solubility and formation products are exceeded (Fredric, 1978). Other organisms include Providencia, Klebsiella, Pseudomonas, Serratia, Enterobacter, Staphylococcus and Escherichia Coli. These organisms produce urease. In an extensive

study on staghorn calculi, Alexander et al (1982) noted that the most common clinical manifestation of these calculi were pain, fever, recurrent urinary tract infection and hematuria. These were more prominent if there was a complication. A palpable flank mass indicated pyonephrosis or xanthogranulomatous pyelonephritis. Other complications included azotemia, sepsis, and death. Only 1% of their patients could be considered as having a silent stone while other authors say 24%.

The natural history is that of progressive deterioration, obstruction and sepsis. At least one complication was present in 53% of their cases. The recurrence rate after surgical removal varies from 5 to 54%.

2.2.3.4. URIC ACID STONES.

The causes are well understood unlike with the calcium stones. Pre-

existing stones can be dissolved or reduced medically so the disease is preventable. Uric acid and its salt, sodium hydrogen urate, also produce intrarenal disease (Fredric, 1980).

They make up 5-10% of renal stones in the United States, 5% in most of Europe and the highest percentages are from Israel where Atsmon et al reported that 75% of stones were composed of uric acid (Fredric, 1980).

Ts'si Fan (1981) wrote a review article on urolithiasis in hyperuricemia and gout. He says most stones in ancient times were in the bladder, few being found in the upper urinary tract. Since uric acid, the end product of purine metabolism in man, is of limited solubility; man is vulnerable to suffer from uric acid stones. The end product of purine metabolism in most mammals is allantion, which is converted from uric acid by uricase. Allantion is

freely water soluble and thus most mammals do not get stones except some dogs (Dalmatians). Birds and reptiles excrete uric acid through the capacious cloaca.

In man about two-thirds to three quarters of uric acid formed from purine metabolism is excreted by the kidney under normal circumstances. In patients with gout and hyperuricemia there is an over production of uric acid that must be excreted to maintain homeostasis.

The chance of having renal calculi is 40 to 50% if urine uric acid is more than 1gm per day and if uric acid nitrogen makes up more than 2.2% of the total urine nitrogen or if serum urate is more than 11mg/dl.

In patients with renal calculi and gout uric acid is the chief component in 80% of the cases; calcium oxalate 10%; mixed with calcium oxalate in 7% and with calcium phosphate in 2 to

3%. Some states of increased uric acid production are: partial or complete hypoxanthine-guanine-phosphoribosyl transferase deficiency, myeloproliferative and neoplastic diseases (blood dyscrasias like polycythemia, leukemia, lymphomas, myelomas).

Fellstorm et al (1983) also stress that hyperuricemia and gout are associated with a high incidence of urolithiasis. These are pure uric acid, mixed uric acid and calcium containing stones.

Bengt et al (1982) came to the conclusion that hyperuricosuria is not a feature of calcium stone disease but when present, particularly in combination with renal acidification defects, the stone disease is more severe in terms of stone operation. It was earlier suggested that urate salts could possibly facilitate calcium stone formation in different ways. One

possible mechanism is epitaxial growth of calcium oxalate on a nidus of uric acid or monosodium urate because of structural similarities. Urate may also act as an anti-inhibitor of crystal precipitation and growth through an adsorption of naturally occurring macro-molecular inhibitors of crystallisation in the urine.

2.2.3.5. CYSTINE STONES

The overall frequency of cystine stone formers seems to be 1-3% of the stone-forming patients. Most patients have recurrent stone disease and morbidity may be high especially for men. The stones precipitate from urine oversaturated with excessive cystine excretion, a direct consequence of defective renal tubule cystine reabsorption (Fredric, 1980).

Cystine stones are not seen commonly. Cystinuria occurs in 1 of every 15,000 persons with only 2 to 3% of

those with cystinuria having stone formation. Under ordinary circumstances very little cystine is excreted in the urine. Thus it is unusual to have mixed stones of uric acid and cystine in patients with gout. On the other hand, certain cystinuric patients are hyperuricemic (Ts'si-Fan, 1981).

Urolithiasis in children is quite uncommon and usually a metabolic or other cause can be found in 40% of the patients. In adults with cystinuria, cystine stones usually are found in the upper urinary tract, although vesical calculi are sometimes observed. There is an associated inborn error of tubular reabsorption of cystine, lysine, ornithine and arginine (O'regon et al, 1980).

2.2.3.6. SILICA STONES

Pure silicon dioxide (silica) were removed from the bladder of an 81-

year old man in conjunction with transurethral resection of the prostate; Levisin et al (1982). Silica compounds contrary to widely held impressions, do enter into human metabolism. Silica is absorbed in diet and about 10mg per day are excreted in urine. Its bladder stones may not be as rare as believed. Many stones that cannot be analysed chemically are not submitted routinely for infra-red and x-ray analysis.

2.2.4 SOME OTHER PATHOPHYSIOLOGICAL
CONSIDERATIONS.

Pak et al (1981) state that the cause of urolithiasis in patients with primary hyperparathyroidism is not known. They did not find any distinctive features to distinguish the physiology of patients with stone forming and non-forming hyperparathyroidism.

It has been suggested that certain exchange resins bind, and lower intestinal

absorption of calcium thus modifying its physiology. Scholz et al (1981) found that this was not an effective method of control because with time there was mild transitory hyperparathyroidism and increased intestinal calcium transport thus bringing urinary calcium to pre-treatment levels.

Toluene intoxication induces renal tubular acidosis (RTA) (Michael et al, 1980). The finding of hyperchloremic acidosis in a stone former immediately suggests RTA. There is an altered metabolism of calcium and citrate in systemic acidosis. Chronic acid retention titrates alkaline bone salts, leading to mobilisation of calcium and hypercalciuria. Direct effects of acidosis on the renal tubule also may have a role in increasing urinary calcium.

Acidosis reduces urinary citrate, an inhibitor of crystallisation (Michael et al, 1983; Michael et al, 1980). Alkaline urine decreases the solubility of calcium salts particularly calcium phosphate. Impaired sodium-hydrogen exchange in the

distal nephron leads to sodium depletion, secondary hyperaldosteronism, potassium wasting then severe acidosis and hypokalaemic paralysis (Michael et al, 1980).

Laor et al (1985) state that indwelling calculi in the urinary tract have no influence on the twenty four hour urinary metabolite output.

William as an editorial guest (1982) rightly concludes that urinary calculus is a troublesome disease which is still largely poorly understood. It may be that we have not yet mastered the problem, in which case a redefinition may be in order. Without such knowledge we are unlikely to arrive at a true understanding of the mechanisms of calcium formation. We may solve the clinical problem by accident but the clear message is that it has not yet been solved.

2.3. DIAGNOSIS:

Clinical assessment, history and physical examination, are the basis for assessing activity, morbidity, complications as well as for detecting a few of the causes of stones. Laboratory studies are the key to the diagnosis of most causes of stones (Reddy et al, 1985).

The formulation of improved diagnostic criteria based on physiological derangements has led to the elucidation of specific metabolic causes in nearly 90% of the patients with nephrolithiasis. These include absorptive hypercalciuria, renal hypercalciuria, primary hyperparathyroidism, hyperuricosuria, hyperoxaluria, renal tubular acidosis, uric acid lithiasis and infection lithiasis. Diagnostic separation is critical since it would ensure delivery of a selective treatment program based on correction of specific underlying physiological derangements (Charles, 1982).

Gillenwater (1982) did a study on urolithiasis in children. The most common clinical features were abdominal pain and hematuria 40%; 14.5% had painless hematuria; 58% had infection; 25% congenital abnormalities; 31% metabolic abnormalities and 9% had neurogenic bladders. Others had dysuria and enuresis. Urolithiasis in children is variable in its presentation.

Renal pain is thought to result from distension and increased tension of the renal pelvis and capsule. The pain is mediated through autonomic nerves via the iliac ganglia. Acute ureteral occlusion involves an increased blood flow caused in part by prostaglandin release. The potent prostaglandin synthetase inhibitor diclofenac sodium, relieves renal colic through reduction in the rise in intrapelvic pressure mediated by prostaglandin released in the kidney during ureteral obstruction (Gillenwater, 1982).

In addition to the routine diagnostic studies one should pay particular attention to several points for precise diagnosis (Ts'si-Fan, 1981). Examine urine for microscopic hematuria and pyuria. Urine may remain clear at times even if there is complete obstruction of the ureter by a calculus. Urine collected might be from the unobstructed ureter. Both ureters can be obstructed by impacted crystals or stones leading to anuria and acute elevations of blood urea nitrogen and creatinine (Ts'si-Fan, 1981). The traditional 24-hours urine metabolic screening is not useful in evaluating stone formers (Gillenwater, 1984).

The routine technique of stone analysis by crushing or powdering the fragments is not quite satisfactory. It is better to split the calculus and to scrape off bits of

calculus material for chemical analysis or for direct examination under the light microscope. Other useful *in vivo*/*in vitro* techniques are chromogenicity, ultraviolet spectrophotometry, polarised microscopy, scanning electron microscopy, powdered diffraction analysis using x-rays, radionuclide imaging with radioactive technetium (^{99m}Te). Computerised axial tomography,

calculus material for chemical analysis or for direct examination under the light microscope. Other useful invivo/invitro techniques are chromogenicity, ultraviolet spectrophotometry, polarised microscopy, scanning electron microscopy, powdered diffraction analysis using x-rays, radionucleotide imaging with radioactive technetium (^{99m}Te). Computerised axial tomography, sonography, (Holm-Nielson et al, 1981; Ts'si-Fan, 1981), autoradiographic analysis (Bridget, 1980), and exfoliative urinary cytology (Morten, 1981).

Approximately 80% of all renal and ureteral calculi are non-opaque or of low density. The diagnosis may be difficult. These calculi usually consist of uric acid, urate, xanthine and/or cystine components that must be differentiated from tumours and blood clots. Painless hematuria is caused by a tumour unless proved otherwise is an old medical axiom. History, blood chemistry, urinalysis, and urine cytology are not diagnostic. Tumour signs as 'inverted goblet' sign on IVP are inconclusive. Other methods include retrograde pyelography, ureteral brush biopsy, angiography, sonography, and computerised axial tomography with or without contrast have all been used to aid in diagnosis (Morten, 1981; Stiris, 1981).

Pure uric acid stones are relatively small, smooth, rounded and brownish red or reddish black. A cystine stone usually is yellowish brown and is much firmer than a uric acid stone. When a uric acid stone is mixed with calcium or with cystine, it may be radiopaque though the opacity is not as dense as for a pure calcium stone. A staghorn stone may be encountered, containing free uric acid layered with deposits of oxalate and/or phosphate (Ts'si-Fan, 1981).

Uric acid crystals look like needles and cystine crystals are hexagonal. Under polarised microscopy, uric acid has a strongly negative birefringence. Calcium pyrophosphate stones are much thicker and of various shapes, usually rhomboid. Calcium crystals are weakly positive on birefringence. Under scanning electron microscopy uric acid crystals in a calculus show orderly stratified crystal structures and non-orderly areas of cementation of intercrystalline attachments (Ts'si-Fan, 1981).

Gillenwater (1984) looked at the value of the 24-hour urine analysis in the assessment of stone formers. They observed that the 24-hour urine metabolic screen is not useful in evaluating stone formers. In their study, 5% of non-stone formers and 8% of stone formers excreted calcium at daily rates greater than the normal 95th percentile. Oxalate excretion was increased above the

95th percentile in 5% of non-stone formers and 22% of stone formers.

Diagnostic irradiation is the second most important burden on the population after natural background irradiation (Gillenwater, 1981). Most techniques to locate stones *invivo* be they plain X-rays, IVP, CT evaluation, and radionuclide imaging or probe renography deliver some form of irradiation energy to tissues (Holm-Nielsen et al, 1981; Noble et al, 1981). Sonographic evaluation is devoid of this and has the added advantage of picking up radiolucent stones which show as echo-dense areas.

Autoradiographic analysis (Bridget, 1980) and powder Diffraction analysis of urinary calculi using cobalt x-ray tube (David et al, 1982) are some radiological techniques used to analyse stones *invitro*.

One area of difficulty in diagnosis is the origin of gross hematuria in children which is often unknown even after extensive investigations. The effectiveness of thiazides in preventing further episodes makes one wonder if bleeding is not caused by tiny calculi, not large enough to be seen on excretory urography (Kalia et al, 1981). Rubben et al (1982) have recommended the differentiation between uric acid stone and epithelial

tumour in the upper urinary tract. The overall accuracy rate of the cytologic diagnosis was 70%, with about 10% of false positive results in patients with stones. Other sources of false positives are bladder carcinoma, urinary tract infections, urolithiasis, trauma, tuberculosis, irradiation of the small pelvis and systemic cytotoxic therapy.

2.4 TREATMENT:

A lot of work has been devoted to this aspect of established urolithiasis. The methods are conservative (Backman, 1980; Fellstrom et al, 1983) and surgical (Frank et al, 1981; Donald, 1981; Jeffrey et al, 1981). Some of the newer techniques include cystoscopy (Jeffrey et al, 1982), ureteroscopy (Alvin, 1983), percutaneous nephrolithotomy (Gillenwater, 1982), pulsed dye laser fragmentation (Stephen et al, 1987) extracorporeal shock wave lithotripsy (Scott, 1987), electrohydraulic and ultrasonic lithotripsy (Gillenwater, 1982). More than 80% of ureteric stones are passed spontaneously, but the behaviour of any particular stone cannot be predicted. Impaction of a ureteric stone causes inflammatory changes; these result in the formation of 'bars' i.e. oedematous areas of mucosa engulfing the stone. The ureter dilates with thinning and fibrotic changes. Most stones are passed within 6 weeks and the smaller and

lower down the ureter the stone is, the more likely it is to pass. There is no urgent reason for removing a ureteric stone unless:

- (a). the pain is severe.
- (b). there is persisting increasing dilatation.
- (c). the patient becomes pyrexial.
- (d). there is evidence of pyelonephritis or pyonephrosis.

The size and position may also indicate early removal (O'Flynn, 1980).

Total obstruction by a stone is a rare occurrence. O'Flynn (1980) concludes that no form of drug therapy presently used is likely to affect the passage of a stone. Even a high fluid intake is useless. Ureterolithotomy is a highly successful procedure and failure to recover the stone by this method is rare.

Glenn et al (1985) suggest that appropriately applied medical treatment could affect the course of stone disease favourably and influence significantly the need for an operation and also remain an appropriate adjuvant to stone removal by whatever means.

2.4.1. MEDICAL TREATMENT:

Pak in Dallas (1982) observed the heterogeneity of the pathogenetic background for nephrolithiasis. He set out selected treatment programs directed at ameliorating or correcting the specific underlying derangements. Some of these include sodium cellulose phosphate; thiazides for absorptive hypercalciuria, thiazides for renal calciuria; orthophosphate for hypophosphatemic absorptive hypercalciuria; allopurinol for hyperuricosuric calcium oxalate nephrolithiasis (Pak, 1982; Ts'si-Fan, 1981).

An increased urine volume reduces urinary saturation of calcium oxalate, brushite, monosodium urate and inhibits spontaneous nucleation of calcium oxalate. Potassium citrate is useful for hypocitraturic calcium nephrolithiasis (Pak. 1982). Backman et al (1980) do not advocate the use of sodium cellulose phosphate as the drug of choice in calcium oxalate stones because of the many side effects encountered.

Ts'si-Fan (1981) thinks that the efficacy of medical management depends to a large extent on

patient compliance. When a relatively small sized stone is lodged in the ureter, a high urine flow may promote its travel down the ureter.

Adjustment of urine pH depends upon the nature of the stone (Backman, 1980; Fellstrom, 1983). Uric acid, calcium oxalate and cystine precipitate in acid urine. Calcium phosphate precipitate from solutions with pH more than 6. Magnesium ammonium phosphate deposits in alkaline urine. Dietary prudence is important e.g. decreasing purine and protein intake in uric acid stones. Control of urinary infection when present is important. The most effective way to prevent recurrent calculi is the reduction of solute production and/or its excretion. Calcium stones are not easy to prevent because of the relative insolubility of calcium salts throughout the pH range of urine.

Hydrochlorothiazide inhibits the excretion of calcium as well as uric acid. (Ts'si-Fan, 1981; Try 1981). Cystine stones are relatively rare and when present tend to recur often requiring surgery. Penicillamine, a cystine analogue, acts as a chelating agent to form a

mixed cysteine-penicillamine disulfide and penicillamine disulfide, which are many times as soluble as cystine. Alfa-mercaptopropionylglycine (MPG) dissolve stones invitro more than penicillamine (Takuo et al, 1982).

In the management of staghorn calculi, operative procedures must be followed by adjunctive measures. It is imperative to maintain sterile urine with appropriate bactericidal antibiotics followed by long-term suppressive antibactericidal therapy and any metabolic abnormality should be corrected promptly (Alexander et al, 1982).

Buck et al (1981) selected hypercalciuric patients who were treated with indomethacin, which resulted in a significant decrease in urinary calcium excretion. This suggested that PGE_2 is the hormone that determines the renal handling of calcium by controlling renal tubular function.

Joseph et al (1982) managed urolithiasis in pregnant women. 42% of them had idiopathic hypercalcuria, 13% hyperuricosuria, 10% primary

hyperparathyroidism, 13% infected stones, 3% cystinuria, 19% idiopathic lithiasis. This distribution is not different from that of the general population. Most of the symptoms were flank pain 100%, premature labour 67%, abdominal pain 50%, urgency 78%, dysuria 67%, nausea and vomiting 33%, gross hematuria 22%, chills 11% and fever 22%. 50% of these patients passed their stones spontaneously on conservative management (hydration and analgesics). Surgical intervention was required in less than 50% of them.

2.4.2. SURGICAL MANAGEMENT:

The first successful renal lithotomy procedure was performed in 1879 by Heineke. Successful removal of renal calculi has occurred only in modern times. All lithotomies are faced with the problem of retained or residual calculi (Donald, 1981).

Renal calculi are commonly removed through a parenchymal renal incision (Nephrolithotomy) or through an incision into the renal pelvis (pyelolithotomy). Removal of a large branched calculus may be facilitated by using an

extended pyelolithotomy. Partial and total nephrectomy are sometimes indicated. Numerous adjunctive techniques include: regional renal hypothermia, operative radiology, operative nephroscopy, coagulum pyelolithotomy, and post operative renal irrigations with stone solvents. Complete surgical removal of all stones is not always possible even in the most expert hands. Operative mortality is less than 1% and surgical morbidity is small (Fredric, 1980). Complete surgical removal is the initial step in the treatment of staghorn calculi and should be done as the diagnosis is made if clinical conditions allow. Extended pyelolithotomy and the anatomic nephrolithotomy are used the most. A partial nephrectomy may be indicated when severe atrophic changes have occurred in the lower pole of the kidney. In the authors series, extended pyelolithotomy was done in 35% of cases, anatomic nephrolithotomy 25%, pyelonephrotomy in 17% and partial nephrectomy in 6%. A nephrectomy is indicated only in advanced renal deterioration (Alexander et al, 1982).

Gillenwater (1980) reviewed cases of partial nephrectomies and found that today the greatest

indication is stone disease. It has been used in other cases of localised parenchymal disease in which there is need to preserve renal parenchyma. A flank approach was mostly used in elective cases and transperitoneal approach in cases of trauma.

Frank et al (1981) state that staghorn renal calculi seem infinitely variable in configuration. These features have a bearing on surgical management. Branched calculi typically arise in the intrarenal pelvis, expand to touch the pelvic wall and then grow peripherally, keeping contact with the walls of the pelvis.

James et al (1983) have demonstrated that bilateral renal surgery for stone removal in one operative session can be done safely with results comparable to those of unilateral staged procedures. They advocate aggressive surgical removal of all stones and appropriate metabolic and antibiotic therapy. The preferred approach is the anatomic nephrolithotomy.

Other surgical techniques of management include surgical removal of parathyroid adenomas or hyperplasias. Serum calcium and serum parathyroid hormone levels serve to confirm the presence of parathyroid pathology. Jeffrey et al (1981) came to the conclusion that parathyroidectomy is an effective means to prevent patients with metabolically active stones from getting new calculi.

2.4.3. CHEMOLITHOLYSIS:

Berkhoff et al (1987) have confirmed that the dissolution of cystine calculi by irrigation with tromethamine-E, mercapto-propionylglycine, D-penicillamine and acetylcysteine is possible. In patients intelligent enough to handle the irrigation system properly it is possible to proceed to percutaneous dissolution of cystine calculi on an ambulatory basis after an initial stay in hospital.

Gillenwater (1985) acknowledges the fact that struvite calculi can be dissolved with hemiacidrin, a modification of an isotonic citrate solution pH 4.0. It has been used for the dissolution of small fragments retained

after open surgical removal of a large stone or after percutaneous ultrasonic nephrolithotripsy.

Percutaneous techniques have improved irrigation procedures. Marcus et al (1982) have described an antegrade catheter technique to dissolve uric acid ureteral calculi. Sodium bicarbonate irrigation gets in via a nephrostomy tube and an antegrade tube provides drainage in cases of obstructed ureters. THAM (trihydroxy methyl aminomethane) can also be used.

Calcium oxalate stones are the least soluble type of urinary tract stones. Their dissolution in situ has not been reported. Charles et al (1982) presented a case report which suggests that an increased urinary output and thiazides can do it. The latter lower calcium excretion and decrease the state of saturation. Thiazides also increase pyrophosphate excretion, a recognised inhibitor of stone formation. Zinc and magnesium are inhibitors of stone formation and excretion of these two ions are increased by thiazides.

2.4.4. ADJUNCTIVE SURGICAL TECHNIQUES

Three basic approaches are used to get to the stone in the tract. The percutaneous approach (John et al, 1985; Reddy et al, 1985) the transurethral approach (Bishop et al, 1987) and the extracorporeal approach (Christian et al, 1982; Royce et al, 1987) have all been extensively described.

Since it is extremely important to remove all calculi to reduce the likelihood of recurrence, a number of measures have been introduced to improve stone localisation and removal. This reduces the number and extent of incisions made into the renal parenchyma. Amongst the techniques used are operative radiography, nephroscopy, plasma coagulum and ultrasonography (Bernard et al 1982; Peter et al, 1981).

2.4.4.1. TRANSURETHRAL ENDOSCOPIC/NON- ENDOSCOPIC TECHNIQUES

Besides open ureterolithotomy, ureteric stones can be removed by 'blind' extraction using a basket

extractor of standard type (DORMIA) or the Pfister-schwartz pattern. Endoscopy avoids the blind manipulation of stones and increases the success rates in bigger and impacted stones. Other procedures like ureteral meatotomy, electrohydraulic lithotripsy (EHL), ultrasonic lithotripsy, pulsed dye laser fragmentation and ballon dilatation can easily be performed with lesser complications (Alvin, 1983; Mclean et al, 1980).

2.4.4.2 PERCUTANEOUS TECHNIQUES.

Methods for establishing percutaneous access to the urinary tract have developed considerably since 1955. The traditional methods of managing ureteral calculi that could not pass spontaneously were: retrograde stone manipulation for calculi situated in the lower ureter below the pelvic brim and open ureterolithotomy for stones situated in the middle, upper and lower ureter, which could not be

manipulated successfully in retrograde fashion.

Initially, the percutaneous approach for ureteral stones was difficult because on many occasions it was impossible to visualise the calculus directly endoscopically and success depended on being able to basket the stone under flourosopic control (John et al, 1985).

In percutaneous nephrostolithotomy, large rigid Teflon tubes are introduced with the use of flexible polyurethane dilators into the pelvis (Gillenwater, 1982). Percutaneous access to the collecting system can be rapidly and safely achieved and the tract dilated (Gillenwater, 1984). A nephrostogram, flexible nephroscopy (Reddy et al, 1985) or rigid nephroscopy (Gillenwater, 1984) using a Storz or Wolf percutaneous nephroscope can be carried out. Other procedures possible through the nephroscope are:- forceps and basket

extraction, flushing or fragmentation with an ultrasonic lithotrite or electrohydraulic probes (Gillenwater, 1982). Nephroscopy is used as an auxiliary technique in complete stone surgery; permitting visualisation of about 60% of calices and localisation of small stone fragments.

Reddy et al (1985) had an overall success rate of 99% for renal and 94.5% for ureteral stones. They did percutaneous removal of renal and ureteral stones in 400 patients. The enhanced success rate was due to greater experience and modification of techniques allowing extremely accurate placement of nephrostomy tubes.

2.4.4.3 EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY (ESWL)

The search for methods alternative to surgical treatment of kidney stones has gone on for years.

Extracorporeally generated shock waves focused at the stone or target, are generated by an underwater, high-voltage condenser spark discharge lasting 1 second. Gillenwater (1981) treated 20 out of 21 patients this way and the resulting fragments were passed in urine. Extracorporeally induced shock waves are completely different from ultrasound-litholapaxy or electrohydraulic waves and is non-invasive. Royce et al (1987) used ESWL specifically on uric acid calculi. They enunciated that ESWL and urinary alkalinisation are complementary non-invasive methods of treating uric acid calculi and in the present series they rendered 76% of the patients stone-free at 3 months and 90% at 5 months.

In Gillenwater's article (1981), 19 patients had oxalate stones, one had uric acid and another had calcium phosphate. The struvite was broken

up into large pieces too large to pass spontaneously.

2.5 COMPLICATIONS AND RELATED PROBLEMS IN RENAL STONE DISEASE

Most complications are related to the natural history of the stone and its composition. Others are associated with the type of management. Related problems are mostly of economic and technical nature. For instance surgical, endoscopic or ESWL treatment per course is about US\$6,000 to \$10,000. In contrast one can treat and follow stone patients medically for less than \$1000 per year (Glenn et al, 1985).

Calculi have a great tendency to recur in 75% of cases. Medical treatment is effective in preventing or reducing the occurrence of new stone formation in 95% of cases (Glenn et al 1985)

Staghorn calculi are usually associated with pain, fever, recurrent urinary tract infection and hematuria. These become more prominent if there are complications like hydronephrosis, pyonephrosis, xanthogranulomatous pyelonephritis and end stage kidney disease. The recurrence rate after staghorn removal can be from 5 to 54%. The natural history is that of progressive morbidity and mortality (Alexander et al, 1982).

Goldwasser et al (1985) state that ureterocutaneous fistulae can occur following ureteral calculi, ureteral infections, surgery, ureteral tumours, colonic and duodenal disease.

The most frequent complications in percutaneous techniques are bleeding and difficulties in passing the stone fragments. Others include lacerations, arteriovenous fistulae, perirenal abscess and extrarenal extravasation of fluids (Glenn et al, 1985).

During transurethral endoscopic manoeuvres the common complications are perforations near or at the vesico-ureteral junction and the pelvic brim; stenosis; temporary dilatation of the ureter; vesico ureteral reflux; urinary infection and thrombo-embolic disorders (Bishop et al, 1987).

2.6. PREVENTION

Some preventive measures have been advocated for some particular stone diseases. Maschio et al (1981) have demonstrated that low dose thiazide, amiloride and allopurinol are effective in preventing recurrent calcium nephrolithiasis.

Glenn et al (1985) concluded that potassium citrate might correct the underlying metabolic derangements and prevent the complications of nephrolithiasis in patients with distal renal tubular acidosis. Gillenwater (1984) holds the same views.

CHAPTER 3

MATERIALS AND METHOD

This is a retrospective study carried out at the Kenyatta National Hospital, Nairobi, Kenya. It is the largest and the best structured Government Hospital offering primary, secondary and tertiary health care to the people.

The files of patients hospitalised in the four General Surgical Wards and the Paediatric Surgical Ward from January 1978 - December 1987 were considered. The discharge registers, theatre registers and the computed filing system were used to sort out the patients managed for urolithiasis.

3.1 SAMPLE SIZE

The files of patients with urolithiasis were studied critically to make sure that most or all of the information on the questionnaire could be retrieved. Where gross doubts existed as to the accuracy of the diagnosis, such files were excluded from the study.

Those cases with urolithiasis that were seen and managed in the medical, gynaecological, orthopaedic or oncology units were also excluded.

3.2. PROCEDURE OF DATA COLLECTION.

Information was collected for each member of the sample using a questionnaire which also served as a check list for clinical observations. I critically studied the files myself to determine who was and who was not to be considered an element of the sample. Each case was entered into a separate questionnaire.

Data was collected from the Department of Biochemical Pathology to determine the nature of stones. All the available information was thereafter analysed.

CHAPTER 4

RESULTS

4.1 82 patients with 89 episodes were seen and managed for stone disease in 10 years. This puts the incidence of stone disease at 8.9 cases per year in the General Surgery Units of the Kenyatta National Hospital.

4.2 **TABLE 1: AGE DISTRIBUTION**

5-YEAR INTERVAL	NO	10-YEAR INTERVAL	NO	%
1 - 5	2			
6 - 10	5	1 - 10	7	8.5
11 - 15	5	11 - 20	10	12.2
16 - 20	5	21 - 30	21	25.6
21 - 25	10	31 - 40	15	18.3
26 - 30	11	41 - 50	12	14.6
31 - 35	9	51 - 60	7	8.5
36 - 40	6	61 - 70	5	6.1
41 - 45	6	71 - 80	3	3.7
46 - 50	6			
51 - 55	5			
56 - 60	2			
61 - 65	1			
66 - 70	4			
71 - 75	2			
76 - 80	1			

The age was not stated for 2 patients. The youngest patient was 2 years old and the oldest 78 years. The greatest number of patients ranged between 11 and 50 years. The peak incidence was in the 3rd decade.

4.3 SEX DISTRIBUTION

67 Males (81.7%) and 15 females (18.3%) were seen. The sex ratio is 4.5 males: 1 female.

4.4 MARITAL STATUS

43 patients (52.4) were married, 34 (41.5%) were single. One was divorced and another a widower. The status was not stated for 3 others.

Most of the single ones were below 20 years (17 patients = 20.7%). 14 of these (17.1% were students or pupils).

4.5 TABLE 2: OCCUPATION

OCCUPATION	NO	%
Salaried employment	26	31.7
Students & Pupils	14	17.1
Farmers/Grazers	12	14.6
Jobless	10	
Owens business	7	8.5
Housewife	6	7.3
Retired	4	4.9
Not stated	3	3.7
TOTAL	82	100

4.6 TABLE 3: PLACE OF RESIDENCE

DISTRICT	NO	%
NAIROBI	19	23.2
KIAMBU	7	8.5
NYERI	5	6.1
EMBU	4	4.9
GARISSA	4	4.9
MACHAKOS	4	4.9
MOMBASA	4	4.9
MURANGA	4	4.9
KAKAMEGA	3	3.7
KITUI	3	3.7
MERU	3	3.7
NAKURU	3	3.7
SOUTH NYANZA	2	2.4
WEST POKOT	2	2.4
FOREIGNERS (REFERRED)	2	2.4
BARINGO	1	1.2
BUSIA	1	1.2
KAJIADO	1	1.2
KISUMU	1	1.2
LAMU	1	1.2
MARSABI	1	1.2
NYANDARUA	1	1.2
SIAYA	1	1.2
WAJIR	1	1.2

The place of residence for 2 patients was not noted.

The results are a reflection of population densities and proximity to secondary and tertiary health care services. Some other factors like human behaviour, attitudes to modern health care and acceptance of surgery could be other contributing factors that might affect this distribution.

4.7 RACE

76 patients were negroes (92.7%). 4(4.9%) were of Asian origin. The race was not stated in 2 cases. No caucasian was treated in this group. They hardly ever attend our hospital.

4.8 HISTORY OF DISEASE AND RESULTS OF EXAMINATION

4.8.1 NO OF EPISODES

65 Patients (79.3%) had only one episode. 10 patients (12.3%) had more than one distributed thus: 8 patients had 2, 1 had 3 and another had 4 documented episodes.

The number of episodes was not stated in 7 patients (8.5%).

4.8.2 SIDE INVOLVED

20 Patients (24.4%) had pain on the right side and 20 others on the left. It was described as bilateral in 5 cases (6.1%).

In some (43 cases = 52.4%) the symptoms were non-lateralising especially in disease of the lower urinary tract.

Because some patients had more than one episode of disease with varying locations of the stone and symptomatology, it is understandable that the total is more than the sample size.

4.8.3 **TABLE 4: PRIMARY SITE OF PAIN AND TENDERNESS**

SITE	NO	%
Flanks and loins	45	56.3
Hypogastrium	37	46.3
Iliac fossae	11	13.8
Periumbilical	3	3.8
Genitalia	2	2.5
Subcostal	2	2.5
Epigastrium	0	0.0
TOTAL	80	100

In one patient there was a communication barrier and in another it was not mentioned.

Pain in the flanks, and hypogastrium were the most common sites.

4.8.4 TABLE 5: RADIATION OF PAIN

This was documented in 32 patients.

SITE	NO	%
Hypogastrium	15	46.8
Testicle/Scrotum	5	15.6
Penis	5	15.6
Groin	4	12.5
Labia	2	6.3
Back	1	3.2
TOTAL	32	100.0

4.8.5 TABLE 6: SIGNS AND SYMPTOMS

SIGN AND SYMPTOM	NO	% of 82
Colic/pain/discomfort/tenderness	71	86.6
Dysuria	42	51.2
Frequency	27	32.9
Urinary retention	23	28.1
Chills	15	18.3
Dribbling	14	17.1
Urgency	14	17.1
Vomiting/nausea	11	13.4
Incontinence	11	13.4
Nocturia	7	8.5
Frank hematuria	6	7.3
Straining	6	7.3
Urethral discharge	3	3.7
Swelling	2	2.4
Constipation	1	1.2
Headaches	1	1.2

The major presenting complaints were pain and dysuria. Frequency, urinary retention, chills, dribbling and urgency were other concomittant symptoms.

Frank hematuria (7.3%) was not very significant. 50 patients (64.9%) of the 77 urine specimens examined microscopically were positive for hematuria.

4.8.6 **TABLE 7: 77 URINALYSIS**

CRITERIA	NO	%
Cloudy (frank pus, pus cells, bacteria, proteinuria)	52	67.5
Hematuria	50	64.9
Foul smelling	5	6.5
Clear	5	6.5
Gravel	1	1.3

Infection (67.5%) and hematuria (64.9%) were very prominent findings in the urine specimens either on the first or subsequent investigations.

4.8.7 **TABLE 8: PERSONAL HISTORY AND OTHER PATHOLOGY DIRECTLY OR INDIRECTLY RELATED TO STONE DISEASE.**

Event	No	% of 82
Urinary tract infection	45	54.9 *
Hydronephrosis (Uni/bilateral)	20	24.4 *
Severe trauma (fractures, gun shots)	11	13.4 *
Benign prostatic hypertrophy	9	11.0
Catheterisation	7	8.5
Fistulae (vesico-vaginal, recto-vaginal; uretero-rectal)	7	8.5
Cardiovascular disease (hypertension, heart failure)	6	7.3
Urinary instrumentation	5	6.1
Hydroureter	5	6.1
Pregnancy	4	4.9
Non-functioning kidney	5	6.1

Pyonephrosis	4	4.9
Schistosomiasis	3	3.7
Gout	2	2.4
Duplex ureters	2	2.4
Aberrant arteries and poorly developed renal vessels	2	2.4
Pill	2	2.4
Pulmonary tuberculosis	1	1.2
Deaf and dumb	1	1.2
Cystocele	1	1.2
Hypertensive encephalopathy	1	1.2
Kidney ptosis	1	1.2
Bladder diverticulum	1	1.2
Horse shoe kidney	1	1.2
Rectal prolapse	1	1.2
Varicose veins	1	1.2
Pelvi-ureteral junction obstruction	1	1.2
Hemiparesis	1	1.2
Underweight	1	1.2
Contracted bladder	1	1.2
Uretero-vesical junction stenosis	1	1.2

There was a patient with paraplegia, posterior dislocation of a hip, congenital talipes equinovarus and an anorectal malformation.

A lady had secondary infertility, ovarian cysts, urinary tract infection and had undergone appendicectomy.

4.8.8. FAMILY HISTORY OF STONE DISEASE

This was positive in 2 cases (2.4%).

4.8.9 TABLE 9: SPECIFIC INVESTIGATIONS

INVESTIGATIONS	No	% of 82
Urinalysis	77	93.9
Plain abdominal x-ray	75	91.5
Intravenous urogram	71	86.6
Diagnostic cystoscopy	14	17.1
Retrograde ureteropyelography	4	4.9
Abdominal ultrasound	2	2.4
Micturition cystourethrogram	2	2.4
Barium enema	1	1.4

Most of our diagnosis were confirmed by urinalysis, plain abdominal x-rays and intravenous urograms. The cystoscopies were done to establish the causes of bladder outlet obstruction and to determine the cause of hematuria when a stone was not evident on plain x-rays.

4.8.10 **TABLE 10: LOCATION OF STONES**

SITE	No	% of 82
Vesical	47	57.3
Kidney (pelvis)	25	30.5
Ureter: lower 1/3	6	7.3
Ureter: middle 1/3	6	7.3
Ureter: upper 1/3	4	4.9
Uretero-vesical junction	3	3.7
Urethra	3	3.7
Pelvi-ureteral junction	2	2.4

After rearranging into few groups we get:

SITE	No	% of 82
Lower urinary tract (bladder+urethra)	53	64.6
Kidney - Pelvi-ureteral junction	27	32.9
Ureters proper	16	19.5

These tables emphasise that our stones are formed mostly in the lower urinary tract. The bladder is the most incriminated site (57.3%), followed by the renal pelvis (30.5%).

4.9 TABLE 11: MANAGEMENT PROCEDURES (DEFINITIVE AND ASSOCIATED)

PROCEDURE	No	% of 82
Hospitalisation	81	98.8
Cystolithotomy	42	51.2
Pyelo-nephrolithotomy	17	20.7
Uneventful passage	9	11.0
Ureterolithotomy	8	9.8
Therapeutic cystoscopy (successful/failed)	7	8.5
Fistula repair	4	4.9
Total nephrectomy	3	3.7
Prostatectomy	2	2.4
V - Y plasty	1	1.2
Ureterolysis	1	1.2
Vaginal examination and milking	1	1.2
Circumcision	1	1.2
Repair of cystocele	1	1.2
Appendicectomy	1	1.2
Bladder neck reconstruction	1	1.2
Caesarean Section	1	1.2
Passage of sound	1	1.2
Sigmoidoscopy	1	1.2
Reimplantation of ureter	1	1.2
Colostomy	1	1.2

One patient (1.2%) was not hospitalised. 11.0% passed their stones uneventfully. Cystolithotomy was the most indicated operation.

4.10 TABLE 12: ASSOCIATED MEDICAL TREATMENT

DRUG USED	NO	% of 82
Analgesics	81	98.8
Antibiotics	80	97.7
Fluids (given or recommended)	64	78.1
Antispasmodics	51	62.2
Diuretics	18	22.0
Allopurinol	4	4.9
Antihypertensives	2	2.4
Urinary alkalinisation	0	0.0
Urinary acidification	0	0.0

Analgesics, antibiotics, fluids and antispasmodics constituted the main stay in this supportive therapy.

4.11 TABLE 13: OUTCOME OF TREATMENT

EVENT	NO	% of 82
Eventually got well/better	64	78.1
Post-operative infection	14	17.1
Recurrence	10	12.2
Lost to follow-up	7	8.5
Renal failure	1	1.2
No improvement	1	1.2
Other urological complications	1	1.2
Related deaths	0	0.0

4.12 TABLE 14: DURATION OF HOSPITALISATION IN DAYS

DAYS	No of cases	% of 89
30 +	51	57.3
11 - 15	7	7.9
26 - 30	6	6.7
21 - 25	5	5.6
6 - 10	3	3.4
16 - 20	3	3.4

The 82 patients had a total of 89 admissions as some of them had recurrences or had to come back for elective surgery after investigations.

The majority of patients stayed on for more than one month. Only 14 cases (15.7%) were admitted for less than a fortnight.

This long stay in hospital certainly had a lot of economic, social, moral and professional impact on the state, the patient, those on the waiting lists, medical staff and the families.

4.13 PHYSICAL NATURE OF THE STONES

Of the 82 documented descriptions, 52 (64.4%) were single and 30 (36.6%) were multiple.

5 of the single stones (6.1%) were stated as being of the staghorn variety.

4.14 TABLE 15: COMPOSITION OF 56 STONES

ANIONS & AMORPHOUS MOLECULES	NO	% of 56	CATIONS	NO	% of 56
Oxalate	43	76.8	Calcium	46	82.1
Urate	33	58.9	Ammonium	23	41.1
Phosphate	16	28.6	Magnesium	18	32.1
Carbonate	9	16.1			
Xanthine	4	7.1			
Sulfonamide	4	7.1			
Chloride	1	1.9			
Cystine	0	0.0			

4.15 TABLE 16: TYPES OF STONES

TYPE OF STONE	NO	% of 56
Calcium stones		
x Calcium Oxalate	4	7.1
x Calcium Phosphae	2	3.6
x Calcium Urate	1	1.8
x Calcium-oxalate-urate	9	16.1
	(16)	(28.6)
	-----	-----
Uric acid	0	0
Cystine	0	0
Struvite	5	8.9
(Magnesium Ammonium Phosphate only)		
Mixed stones	35	62.5

Most of the particles mentioned in table 15 were present in varied combinations in the mixed stones (62.5%). 7.1% had Xanthine, 7.1% Sulfonamide and 1.9% Chloride.

Cystine was not present at all. Most of the uric acid was present in the mixed stones.

The dominance of calcium 82.1% and oxalate (76.8%) as the most common constituents have been clearly demonstrated.

CHAPTER 5

DISCUSSION

5.1 INCIDENCE

Our incidence of 8.9 cases per year is a figure that is difficult to interpret as Scot et al (1987) rightly state. They are difficult to compare because authors like Uden (1987) use figures like 7 in 100,000. The denominator is not common. One is temporal and the other is numerical and the numbers could be referring to hospital admissions or the general population. Helen et al (1981) found out that renal stones affect about 80% of the Irish population at sometime in their lives.

5.2 AGE DISTRIBUTION

90% of Kitonyi's population (1986) was more than 14 years old. Tshipeta et al (1983) had the same trend.

Our study confirms their findings. The highest incidence was in the third decade. 20.7% of our patients were less than 20 years old.

5.3 SEX DISTRIBUTION

Our sex ratio is 4.5 male: 1 female. Kitonyi's was 4.8 male: 2.5 female. Udeh came up with 5.1; Tshipeta had 4:1. These figures are probably a good reflection of the African situation with an unquestionable male dominance.

Churchill et al (1980) had a 1:1 ratio and Verbaeys et al (1985) got 1:59:1

The disparity in these results are quite significant and underscore Hans-Joachim's (1982) observations about the varying epidemiological patterns of stone disease.

5.4 NUMBER OF EPISODES

12.3% of our patients had more than one episode. Ljunghall et al (1987) recorded a 47% recurrence rate in a selected group of old patients over a 10 year period.

5.5 SIDE AND SITE INVOLVED

We found an equal chance for the involvement of either side in the disease. Tshipeta et al had the same finding. The probability of having bilateral stones was the least while the greatest was that of having stones in

the midline structures (lower urinary tract especially the bladder).

Power et al (1987) found stones in the upper urinary tract to be more common in industrialised than non-industrialised countries. Churchill et al (1980) hold the same opinion.

Udeh's findings in Nigeria grossly differ from these observations as 79 cases were from the lower urinary tract. Tshipeta et al from Zaire had findings similar to Udeh's.

Kitonyi had a predominance of bladder stones. We have shown in this paper that bladder stones predominate (57.3%).

5.6 MODE OF PRESENTATION

Churchil et al (1980) state that the common mode of presentation is abdominal or flank pain with or without hematuria in 79% of cases. We confirm this because 86.6% of our patients had pain and 64.9% had frank or microscopic hematuria. Other important findings were the presence of dysuria and frequency.

5.7 RATE OF INFECTION

Tshipeta et al (1983) stated an infection rate of 60% our values range between 54.9% and 67.5% depending on the criteria used.

5.8 COMPLICATIONS

These are difficult to assess in their own right. The problem lies with determining which occurred first as the stone and the complication are usually present when the patient is first seen. The so called complication e.g. hydronephrosis could actually be the basis for the stone formation.

Tshipeta had an infection rate of 60%. Hydronephrosis and pyonephrosis were present in 30% of the cases and deaths were associated with acute severe klebsiella septicemia.

Alexander et al (1982) said pain, fever, recurrent urinary tract infection and hematuria were the most common symptoms in stone disease which became more accentuated with complications like pyonephritis, azotemia, sepsis and eventually death. Only 1% of their patients had a silent stone.

Glenn et al (1985) found most of these complications to be related to the natural history of the stone, composition, and the management technique. Medical treatment is cheaper than any form of surgical management. Percutaneous techniques can cause bleeding, lacerations, arterio-venous fistulae, abscesses and extravasation.

Goldwasser et al (1985) included uretero-cutaneous fistulae, ureteral calculi. Bishops et al (1987) increased the list when transurethral techniques are used. They mention perforations, stenosis, temporary dilatation of the ureter, vesico-ureteral reflex and thrombo-embolic disorders.

Our extensive list on table 8 demonstrates in part some of these problems. These include infection, pyonephrosis, hydronephrosis, heart failure, hydroureters, non-functioning kidneys, fistulae, stenosis, contracted bladder etc.

5.9 FAMILY HISTORY

Emil et al (1982) stressed the high family incidence of calculi in Israel. In our sample 2 patients (2.4%) had a positive family history of stone disease.

5.10 INVESTIGATIONS

Many authors have emphasised the different aspects of diagnostic techniques (Reddy et al, 1985; Charles, 1982; Gillenwater, 1982; Ts'si-Fan, 1981; Bridget, 1980; Morten, 1981; etc)

We used simple available techniques with a lot of success. These included urinalysis (93.9%), plain abdominal X-ray, (19.5%), intravenous urogram (86.6%) and others (see table 9).

The percentages do not reflect the success rate but rather how often the procedure was used. In most of the cases, the combined analysis of the findings was satisfactory for our purposes.

5.11 MANAGEMENT PROCEDURES

Many authors have contributed to the different facets of this issue be they medical, surgical or adjunctive surgical procedures. The last aspect has been of particular interest in the developed nations and need complex and expensive machines.

O'Flynn (1980) said 80% of stones can be passed on their own and thus puts forward a set of criteria to be met if

a ureteric stone needs removal. He says no form of drug therapy is likely to affect the passage of a stone.

Gillenwater (1980) concluded that 44% of patients required hospitalisation and 0.4% needed surgery. In Tshipeta's group medical treatment was the most used procedure.

Our figures look quite alarming at first sight. It should be appreciated that most of them were referred to us and had failed on other forms of medical treatment. We do not have facilities for extensive and refined endoscopic manipulations nor any form of lithotrites.

We had 89 admissions, 57.3% of them needed to be in hospital for more than one month to be treated. Only 15.7% were in hospital for less than 14 days. One patient needed no hospitalisation.

98.8% needed analgesics, 97.7% antibiotics and 4.9% allopurinol. Only 11% passed their stones peacefully. The others needed some form of surgery (see table 11). Cystolithotomy was the most widely done operation (51.2%).

5.12 TYPES OF STONES

Gillenwater (1984) and other authors have condemned the routine technique of stone analysis after crushing or powdering the fragments. They say it is not quite satisfactory. It is the only technique available in this set up and was used in this study.

Scot et al (1987) have noticed that many variables affect the establishment of the prevalences or incidences of disease and their types. Some of these include the degree of aggression in pursuit of a diagnosis.

Levisin et al (1982) also noticed that many stones that cannot be analysed chemically are not routinely submitted for infra-red and x-ray analysis.

We had no pure uric acid stones nor cystine stones. We had mostly mixed stones (62.5%). Calcium was present in 82.1% of the specimens and oxalate in 76.8%.

The dominance of calcium and oxalate in urolithiasis has been observed by many authors including Fredric (1978).

C H A P T E R 6

CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

The paucity of information on stone disease in this environment and Africa as a whole was the sole motive for undertaking this study. It has been quite exciting making a dream come true and now we are in a position to spell out our disease pattern.

We now know the incidence is 8.9 cases every year with a peak in the third decade. There are 4.5 males for every female and the majority are married. Most of our cases are drawn from the indigenes and the proximity to the health units is quite contributory.

Most of our patients have only one episode though a tiny percentage can have as many as four. The stones are formed mostly in the lower urinary tract (bladder). In the upper urinary tract either side is equally predisposed and the chance of having bilateral disease is low.

The common presenting complaints are pain in the flanks, loins, hypogastrium and iliac fossae; dysuria; frequency; urinary retention; chills; dribbling and urgency. Infection and hematuria are quite common.

Patients on admission can have quite a variety of associated problems directly or indirectly related to their stones.

Family histories of urolithiasis are not common. Urinalysis, plain abdominal X-rays, intravenous urograms and cystoscopy are quite adequate for establishing the diagnosis.

Almost all the patients seen in the surgical units require hospitalisation for about a month or more. Cystolithotomy is the most practised surgical procedure. Analgesics, antibiotics, antispasmodics and fluids are widely used. Only a tenth of our patients are treated conservatively.

The majority of our patients get well and no related deaths have been noticed.

Two-thirds of the stones are single and only a tiny proportion of these are staghorn. Calcium and oxalate predominate in the formation of the majority of the

stones which are mixed. We hardly see pure uric acid stones. Cystine is inexistent.

6.2 RECOMMENDATIONS

- I The management of stone disease is not a major problem in our environment. The need for sophisticated and costly equipment as it is the case in the developed nations is uncalled for.
- II Proper training of personnel to handle this problem in the peripheral hospitals offering secondary medical care would be the best approach. This will decrease the necessity for lengthy hospitalisations and curb the inconveniences that are faced at the moment.
- III There is a need to increase public awareness on the presence of this disease and the need for early diagnosis to decrease the associated complications observed.
- IV There is a need for similar studies to be carried out in the other African countries so that a common statement can be made about the problem in our continent.
- V This same study can be carried out in this environment with emphasis shifted to some specific aspects of urolithiasis as the present one is rather global.

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A P P E N D I X IQUESTIONNAIREUROLITHIASIS AT THE KENYATTA NATIONAL HOSPITAL 1978 - 1987**1. PATIENT'S IDENTITY**1.1. Name 1.2 I.P. No. 1.3 Age 1.4 Sex
Male Female 1.5 MARITAL STATUS1. Single 3. Divorced 2. Married 4. Widower 1.6 Number of Children 1.7 OCCUPATION1. Jobless 6. Salaried Employment 2. Farmer 7. Housewife 3. Casual labourer 8. Retired 4. Owns business 9. Student/Pupil 5. Family Business 10. Not stated 1.8 Place of birth or tribe 1.9 Place of residence (USE MAP AND CODE NUMBER ATTACHED)RACE1.10 1. Negro 2. Asian/Arab 3. Caucasian

2. HISTORY OF DISEASE AND EXAMINATION

Yes = 1 No = 2 Not specified = 3

1. Date of first examination

2, No. of episodes

3. Side involved

Left	<input type="text"/>
Right	<input type="text"/>
Both	<input type="text"/>
Not applicable	<input type="text"/>

3. LOCATION OF PAIN AND TENDERNESS

1. Epigastrium 6. Iliac Fossa

2. Genitalia 7. Subcostal

3. Flank 8. Loin

4. Hypogastrium 9. Others

5. Periumbilical

4. RADIATION

1. Hypochondrium 5. Back

2. Labia 6. Penis

3. Testicle/Scrotum 7. Others

4. Groin

5. MANIFESTATION

1. Colic/Pain 7. Vomitting/Nausea

2. Chills 8. Urgency

3. Dysuria 9. Urinary Retention

4. Frequency 10. Hematuria

5. Headaches 11. Dribbling

6. Nocturia 12. Others

6. URINE ANALYSIS

- | | | | |
|--|-------|-------------------------|-------|
| 1. Clear | _____ | 4. Hematuria | _____ |
| 2. Cloudy | _____ | 5. Pus Cells & Bacteria | _____ |
| 3. Foul Smelling/
frank pus discharge | _____ | | |

7. ANTECEDENTS

- | | | | |
|-------------------------|-------|--------------------------------|-------|
| 1. Bone Disease | _____ | 10. Prior Immobilization | _____ |
| 2. Bowel Disease | _____ | 11. Protracted febrile illness | _____ |
| 3. Catheterisation | _____ | 12. Sarcoidosis | _____ |
| 4. Chronic Medications | _____ | 13. Severe Trauma | _____ |
| 5. Gout | _____ | 14. Urinary Instrumentation | _____ |
| 6. Hyperparathyroidism | _____ | 15. UTI | _____ |
| 7. Malignancy | _____ | 16. Vitamins | _____ |
| 8. Mineral Supplement | _____ | 17. Antacids | _____ |
| 9. Peptic Ulcer Disease | _____ | 18. Other Associated Diseases | _____ |

8. FAMILY HISTORY

- | | |
|--------------------|-------|
| 1. Stone Disease | _____ |
| 2. Gout | _____ |
| 3. Not Significant | _____ |

9. SPECIFIC INVESTIGATIONS

- | | | | |
|--------------------------|-------|---------------------------|-------|
| 1. Plain ABD xray | _____ | 4. Urinalysis | _____ |
| 2. Abdominal Ultra Sound | _____ | 5. Retrograde Pyelography | _____ |
| 3. I.V.U. | _____ | 6. Cystoscopy | _____ |

10. LOCATIONS OF STONE/STONES

- | | |
|----------------------------------|-----------------------------------|
| 1. Kidney (Pelvis) _____ | 5. Ureter Lower 1/3 _____ |
| 2. Pelvi-Ureteral Junction _____ | 6. Uretero-Vesical Junction _____ |
| 3. Ureter Upper 1/3 _____ | 7. Vesical _____ |
| 4. Ureter Middle 1/3 _____ | 8. Urethral _____ |

11. MANAGEMENT (SURGICAL)

- | | |
|---|-----------------------------|
| 1. Hospitalization _____ | 6. Total Nephrectomy _____ |
| 2. Partial Nephrectomy _____ | 7. Uneventful passage _____ |
| 3. Pyelolithotomy/
Pyelonephroli-
thotomy _____ | 8. Ureterolithotomy _____ |
| 4. Removal by
Cystoscopy _____ | 9. Others (Specify) _____ |

12. ASSOCIATED MEDICAL TREATMENT

- | | |
|--------------------------------------|--------------------------------|
| 1. Analgesics _____ | 5. Urinary Acidification _____ |
| 2. Antispasmodics _____ | 6. Diuretic _____ |
| 3. Fluids given
(recommend) _____ | 7. Antibiotics _____ |
| 4. Urinary
Alkalinization _____ | 8. Others _____ |

13. OUTCOME OF TREATMENT

- | | |
|--------------------------|----------------------------|
| 1. Got well/Better _____ | 5. Recurrence _____ |
| 2. Died _____ | 6. Lost to follow-up _____ |
| 3. Infection _____ | 7. Others _____ |
| 4. Renal Failure _____ | |

14. DURATION OF HOSPITALIZATION IN DAYS

- | | |
|------------------|------------------|
| 1. 1 - 5 _____ | 5. 21 - 25 _____ |
| 2. 6 - 10 _____ | 6. 26 - 30 _____ |
| 3. 11 - 15 _____ | 7. 30 X _____ |
| 4. 16 - 20 _____ | |

15. PHYSICAL NATURE OF STONES

1. Single _____

2. Multiple _____

16. CHEMICAL COMPOSITION

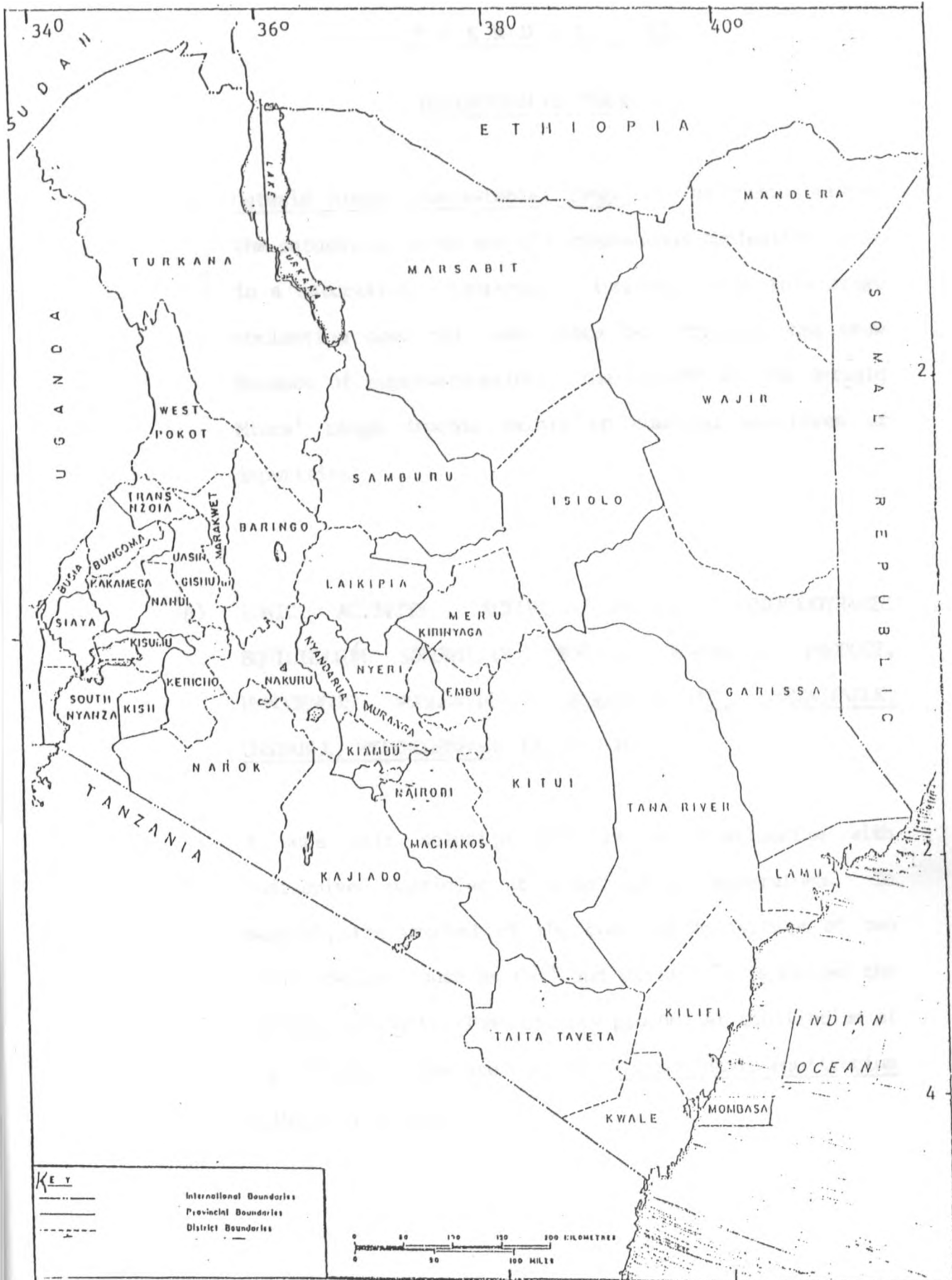
MOLECULE	+/-	MOLECULE	+/-
Calcium		Urate	
Oxalate		Carbonate	
Phosphate		Xanthine	
Cystine		Others	
Magnesium Ammonium Phosphate			

CODES FOR THE DISTRICTS IN KENYA. (USE IN CONJUNCTION WITH
NO. 1.8 AND 1.9 ABOVE)

- | | |
|--------------------|------------------|
| 1. Baringo | 21. Marsabit |
| 2. Bungoma | 22. Meru |
| 3. Busia | 23. Mombasa |
| 4. Elgeyo Marakwet | 24. Muranga |
| 5. Embu | 25. Nairobi |
| 6. Garissa | 26. Nakuru |
| 7. Isiolo | 27. Nandi |
| 8. Kakamega | 28. Narok |
| 9. Kilifi | 29. Nyandarua |
| 10. Kajiado | 30. Nyeri |
| 11. Kiambu | 31. Samburu |
| 12. Kericho | 32. Siaya |
| 13. Kirinyaga | 33. South Nyanza |
| 14. Kisumu | 34. Taita |
| 15. Kitui | 35. Tana River |
| 16. Kwale | 36. Trans Nzoia |
| 17. Laikipia | 37. Turkana |
| 18. Lamu | 38. Uasin Gishu |
| 19. Machakos | 39. Wajir |
| 20. Mandera | 40. West Pokot |

APPENDIX II

MAP OF KENYA



DISTRICTS IN KENYA (1987)

A P P E N D I X IIIDEFINITION OF TERMS

- A) Ostwald Miers' (metastable) range is the space between the saturation curve and the spontaneous nucleation curve in a saturation - temperature diagram. With this range nucleation does not take place but crystals can grow because of supersaturation. The breadth of the Ostwald Miers' range depends mainly on chemical additives or impurities.
- B) IONIC ACTIVITY, ACTIVITY PRODUCT, THERMODYNAMIC EQUILIBRIUM SOLUBILITY PRODUCT, FORMATION PRODUCT, HOMOGENOUS NUCLEATION, UNDERSATURATED, METASTABLE, UNSTABLE, HETEROGENEOUS NUCLEATION.

A pure salt solution will be at equilibrium with undissolved particles at a particular temperature. In general, the product of the chemical activities of two ionic species, such as Ca^{2+} and Oxalate^{2-} , is called the activity product. The activity product at equilibrium at a particular temperature is the Thermodynamic equilibrium solubility product.

If the solid phase is removed, the activity product will not change, but no new solid phase will form. If the activity product is raised gradually by adding calcium, oxalate, or both, and there are no surface impurities or irregularities on the wall of the container or debris in the solution to act as non specific starting points for precipitation, the solution will remain clear until a critical product is reached, the so called formation product. At that point, new solid phase nuclei will begin to form rapidly and then grow. This spontaneous precipitation is called homogeneous nucleation. A solution is called undersaturated with respect to a given ion pair when their activity product is less than the solubility product, metastable when between the solubility and formation products, unstable and prone to homogeneous nucleation when greater than the formation product.

Homogeneous nucleation is not common in nature or the laboratory. Heterogeneous nucleation can be initiated by any solid phase (impurities) that have reasonable structural similarities to the crystal formed by the ion pair in question when the solution is metastable (Fredric, 1980).

C) HEMATURIA

Is the presence of 5 or more red blood cells per high - poweredfield on microscopic examination of the urinary sediment. Apparently healthy, school children or adults have it in 3 - 4% of those tested (Gary, 1983).