DISSERTATION ON

Urolithiasis Associated with Renal Insufficiency

Factors Predicting Outcome-Prospective study

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CERTIFICATE

This is to certify that the dissertation titled "**Urolithiasis Associated with Renal Insufficiency Factors Predicting Outcome-Prospective study"** submitted by **Dr. T. Senthil Kumar** to the faculty of Urology, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in M.Ch. Degree in Urology Branch IV is a bonafide research work carried out by him under direct supervision and guidance for the years 2008 to 2011.

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I **Dr. T. SENTHIL KUMAR** solemnly declare that this dissertation titled "**Urolithiasis Associated with Renal Insufficiency Factors Predicting Outcome-Prospective study"** is a bonafide work done by me in the Department of Urology, Govt. Kilpauk Medical College and Hospital and Govt. Royapettah Hospital under the guidance and supervision of my Professor **PROF. V. SELVARAJ M.Ch.** Professor, Department of Urology, Govt. Kilpauk Medical College and Hospital and **Prof. C.ILAMPARUTHI M.Ch., DNB.,** Prof. Department of Urology Govt. Royapettah Hospital.

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CONTENTS

INTRODUCTION

Kidney stones are a common entity, affecting approximately 5% of women and 12% of men in general population.

Prevalence of kidney stone disease is increasing and likewise the complications associated with stone disease .The most life threatening being sepsis and renal failure. Both could be treated with return of good renal function if managed early and meticulously.

The pathophysiology of renal insufficiency in obstructive uropathy is well studied.

Similar mechanism is seen in stone disease also.but specific feautures of calculous disease like duration of obstruction, type of stone,Infected system,location of stone plays an additional role along with obstruction.

Renal calculous disease may be associated with various degrees of renal insufficiency secondary to a combination of obstruction, urinary infection, frequent surgical intervention, and coexisting medical disease.

Many patients with calculous disease on conservative management by themself or by a nonspecialists land up in renal insufficiency due to

lack of timely referral to urologist and intervention.This type of presentation was seen in many patients .there are many queries about the recoverability of renal function in them if renal parenchyma was thinned out.

In 25 patients with stone disease and elevated renal parameters we have studied the various factors influencing the outcome -preoperative, intraoperative, postoperative.

We have analysed all the entities and the results after intervention in these patients.

AIM OF STUDY

1. TO ANALYZE THE PREOPERATIVE FACTORS CAUSING RENAL INSUFFICIENCY IN STONE DISEASE.
2. TO ASSESS THE INTERVENTIONS DONE TO IMPROVE RENAL FUNCTION.
3. TO DISCUSS THE FACTORS THAT DECIDE THE OUTCOME
4. TO ANALYSE THE IMPROVEMENT IN RENAL FUNCTION AFTER INTERVENTION
5. FOLLOW UP IN TREATED PATIENTS.

REVIEW OF LITERATURE

Urinary lithiases have been a major urological problem. A very high incidence was reported in Pakistan, Northern India, Thailand, Afghanistan, Turkey, Egypt, Japan, Indonesia, Middle East, Europe, Netherlands and Scandinavian countries. Urolithiasis constitutes about 10% to 25% of the total work load in the urological practice. In about 5% of the patients presenting with acute renal failure, the cause is obstructive uropathy and urolithiasis is the most common cause of obstruction.

Many patients with bilateral stones have oliguria .Calculus anuria is a urological emergency due to bilateral ureteric calculus impaction or unilateral ureteric calculus impaction of solitary kidney or the only functioning kidney. After the onset of obstruction, there is increased intrapelvic pressure, resulting in pyelolymphatic and pyelovenous urine back flow as well as fornix rupture and urine extravasation.

Therefore, the obstruction of the urinary tract causes significant kidney damage. Prompt and early intervention can save the patient from developing irreversible renal damage.

The patho physiological changes occurring in calculous obstruction is similar to the obstruction due to congenital obstructions which have been extensively studied.

RENAL FUNCTIONAL CHANGES IN CALCULOUS OBSTRUCTION

1) GLOMERULAR FILTRATION

There are many functional changes in the kidney influenced by the extent and severity of obstruction, whether the obstruction is unilateral or bilateral, and whether the obstruction currently persists or has been relieved.

Obstruction can transiently or permanently alter GFR .After complete bilateral obstruction GFR is decreased but maintained due to

- Continuous salt and water absorption along nephron.
- Ability of renal tract to dilate.
- Alteration in renal hemodynamics.

2) RENAL BLOOD FLOW

There are differences between unilateral ureteral obstruction (UUO) and bilateral obstruction (BUO).

RBF was measured directly with various types of flow probes, and indirectly by measuring RPF using secretory markers and indexing this to hematocrit.

A number of vasoactive substances are thought to play a role in the changes in RBF and GFR. The varying hemodynamic patterns during the time course of obstruction may be due to a combination of vasoactive hormones synthesized and released at different rates, physical damage to glomerular and tubular units, and extrarenal compensatory mechanisms.

Triphasic pattern of RBF and ureteral pressure changes.

Unilateral Ureteral Obstruction-

1st phase- **1 to 2 hours** – Vasoactive substances -prostaglandin E_2 (PGE_2) & Nitric oxide (NO) produced causing Afferent arteriolar vasodilation- RBF increases inspite of high tubular and collecting system pressure.

2nd phase- **3 to 4 hours**- pressure remain elevated but RBF begins to decline.

3rd phase- **5 hours**- further decline in RBF,decrease in tubular and collecting system pressure shifts of blood flow from outer to inner cortex is seen.

Bilateral Ureteral Occlusion

Modest increase in RBF due to Nitric oxide and platelet-activating factor- lasting 90 minutes. Followed by profound decrease in RBF –due to increased renal nerve stimulation - vasoconstriction related to increased renorenal reflex activity Endothelin,Angiotensin II and TXA_2 contribute to these responses. Blood flow showed a 92% decrease in inner medullary plasma flow.

3) Tubular pressure

Ureteral pressure is higher with BUO than with UUO.

Both cases ureteral and tubular pressure is increased for the first 4 to 5 hours, the ureteral pressure remains elevated for at least 24 hours with BUO, whereas it begins to decline and approaches preocclusion pressures by 24 hours with UUO.

The prolonged elevation in intratubular pressure contributes to the profound decrease in SNGFR and whole kidney GFR. Ureteral pressure remains high in BUO -Due to preglomerular vasodilation due to **Atrial natriuretic peptide (ANP)** and then a prolonged postglomerular vasoconstriction.

Post obstructive diuresis - Urine flow and sodium excretion are increased after release of BUO. ANP appears to play a prominent role in this response based on its natriuretic properties.

4) Tubular Function

Urinary Concentrating Abillity

Aquaporin 2 (AQP2) is present in the apical membranes of the collecting duct cells& Aquaporin 1 (AQP1), in renal proximal tubules, the thin descending limb of Henle - promotes urinary concentration through the countercurrent multiplier by facilitating water transport from the descending limb of Henle into the interstitium .

The onset of concentration defects - 6 minutes of ureteral obstruction due to development of vasopressin resistance, Vascular changes -decrease in inner medullary plasma flow Necrosis of both the inner and outer medullae Release of obstruction resulted in polyuria that gradually decreased over a 30-day period.

Sodium Transport

Unilateral Ureteral Obstruction- increased fractional excretion of sodium (FE_{Na}) in the previously obstructed kidney due to contralateral kidney compensating for the sodium losses of its mate.

Bilateral Ureteral Obstruction- The FE_{Na} may be increased to as much as 20 times normal in this setting due to ANP.

When the BUO is relieved, both intrarenal and extrarenal factors greatly enhance salt and water excretion. FE_{Na} following relief of BUO is typically greater than that after UUO because BUO causes retention of Na, water, urea nitrogen, and other osmolar substances and increased production of ANP, all of which stimulate a profound natriuresis.

Potassium Transport

Decrease in K^+ excretion due to reduced delivery of Na to the distal nephron, low volume flow rate that would minimize the transmembrane gradient for K^+ secretion and intrinsic defect in K^+ secretion

Hydrogen Ion Transport and Urinary Acidification

Obstruction causes a deficit in urinary acidification. Major acidification defect is in the distal nephron due to defects in H^+-ATPase

or H^+,K^+-ATPase, Cl^-/HCO_3^- exchange, a back leak of protons into the renal interstitium

Effects of Obstruction on ionTransport

Phosphate –Rapidly excreted when BUO is released -

Decrease in UUO.

Magnesium -increased after the release of either UUO or BUO.

Calcium -disruption of transport in areas of the nephron where Ca^{2+} and Mg^{2+} are differentially transported may account for varying effects of obstruction on their net transport and excretion.

Effect of Obstruction on the Excretion of Proteins

Proteins increased in urine- Monocyte chemoattractant protein 1 is a mediator of the inflammatory process –is an index of tubular damage. Urinary enzymes from the proximal tubule - alkaline phosphatase, γ-glutamyltransferase, *N*-acetyl-β-D-glucosaminidase, and leucine aminopeptidas are increased.

Proteins decreased in urine -epidermal growth factor (EGF),

Tamm-Horsfall protein

Metabolic changes

Shift from oxidative metabolism to anaerobic respiration. Reduction of renal ATP levels, an increase in amounts of adenosine diphosphate (ADP) and adenosine monophosphate (AMP), and an increase in the renal lactate-to-pyruvate ratio.

Chronic interstitial fibrosis

Tubulointerstitial fibrosis, tubular atrophy and apoptosis, and interstitial inflammation.Due to imbalance between Extracellular Matrix synthesis matrix deposition and matrix degradation. TGF-β, angiotensin II and tumor necrosis factor α (TNF-α) from the renal tubular and interstitial cells and infiltrating macrophages -increase extracellular matrix synthesis and deposition.

Matrix metalloproteinases like collagenase decrease and Tissue inhibitors of metalloproteinases Increase- resulting in the accumulation of extracellular matrix. Infiltrating macrophages stimulate TGF-β synthesis, and interleukin 2, interleukin 6, fibroblast growth factor, and

platelet-derived growth factor (PDGF) that appear to contribute to this inflammatory and fibrotic process.

Angiotensin II -upregulates the expression of TGF-β1 & TNF-α-regulates vascular smooth muscles-neointimal thickening. Angiotensin II activates the transcription factor NFκB, which in turn increases the expression of several chemokines and cytokines involved with the fibrotic process.

Whereas UUO increased iNOS in the obstructed renal medulla and increased neuronal NOS (nNOS) and endothelial NOS (eNOS) in the cortex, losartan treatment downregulated iNOS and nNOS with unchanged levels of eNOS. Obstruction increases inducible, neuronal, and endothelial NOS and high concentrations of NO can result in peroxynitrite production- Administration of L-arginine by infusion or even by oral administration prevents the upregulation of iNOS, blunts the increase in renal interstitial volume, and attenuates the infiltration of the renal parenchyma by macrophages in rats with obstructed kidneys.

Bone morphogenic protein 7, a structural relative of TGF-β, was effective in preventing the tubulointerstitial changes and accelerating the return of renal function-this agent inhibited apoptosis.

Hepatocyte growth factor works by suppressing expression of TGF-β and PDGF. Pirfenidone- inhibits collagen synthesis, downregulates production of multiple cytokines and blocks fibroblast proliferation.

Renal Recovery after Obstruction

1) The duration of obstruction .

- When acute- obstruction (3DAYS)is promptly relieved, full recovery of global GFR can occur.

- After 3 days of UUO, GFR and RBF were reduced to less than 10% of their baseline values and Both returned to their baseline within 14 days of relief of obstruction

- In BUO in adults-two phases of functional improvement: initial phase -first 2 weeks -tubular function improved

  later phase -next 10 weeks –Glomerular function improved.

2) Degree of obstruction-mild to moderare hydronephrosis cause parenchymal damage less than severe hydronephrosis.

3) Compliance of the collecting system-extrarenal pelvis is more compliant than intrarenal pelvis.

4) Presence of pyelolymphatic backflow –reduces the renal pelvic pressure and also tubular pressure and subsequent renal damage.

PATHOLOGIC CHANGES OF OBSTRUCTION

Gross Pathologic Findings

- Dilation of the pelvis and ureter and blunting of the papillary tips were present in the obstructed kidney at 42 hours after obstruction and the weight of this renal unit was heavier.

- The cortex and medullary tissue in the obstructed kidney were diffusely thinned.

Microscopic Pathologic Findings

- lymphatic dilation, interstitial edema.

Collecting duct and tubular dilatation was prominent by 7 days.

Interstitial edema, widening of Bowman's space, tubular basement membrane thickening, Interstitial fibrosis and thickening of the tubular basement membrane

Glomerular collapse and tubular atrophy, interstitial fibrosis, and proliferation of connective tissue in the collecting system were reported at 5 to 6 weeks after obstruction.

Stone disease-a brief review of the types of stones and their pathophysiology.

Urinary tract stone disease is likely caused by two basic phenomena. The first phenomenon is supersaturation of the urine by stone-forming constituents, including calcium, oxalate, and uric acid. Crystals or foreign bodies can act as nidi, upon which ions from the supersaturated urine form microscopic crystalline structures. The resulting calculi give rise to symptoms when they become impacted within the ureter as they pass toward the urinary bladder.

The overwhelming majority of renal calculi contain calcium. Uric acid calculi and crystals of uric acid, with or without other contaminating ions, comprise the bulk of the remaining minority. Other, less frequent stone types include cystine, ammonium acid urate, xanthine, dihydroxyadenine, and various rare stones related to precipitation of medications in the urinary tract. Supersaturation of the urine is likely the underlying cause of uric and cystine stones, but calcium-based stones (especially calcium oxalate stones) may have a more complex etiology.

The second phenomenon, which is most likely responsible for calcium oxalate stones, is deposition of stone material on a renal papillary calcium phosphate nidus, typically a Randall plaque.

Calcium phosphate precipitates in the basement membrane of the thin loops of Henle, erodes into the interstitium, and then accumulates in the subepithelial space of the renal papilla. The subepithelial deposits, which have long been known as Randall plaques, eventually erode through the papillary urothelium. Stone matrix, calcium phosphate, and calcium oxalate gradually deposit on the substrate to create a urinary calculus.

Stone Varieties

A. Calcium Calculi

Eighty to eighty-five percent of all urinary stones are calcareous. Calcium nephrolithiasis is most commonly due to elevated urinary calcium, elevated urinary uric acid, elevated urinary oxalate, or a decreased level of urinary citrate.

Hypercalciuira is found as a solitary defect in 12% of patients and in combinationwith other defects in an additional 18%.

Hyperuricosuria is identified as a solitary defect in 8% of patients and associated with additional defects in 16%.

Elevated urinary oxalate is found as a solitary finding in 5% of patients and as a combined defect in 16%.

Finally, decreased urinary citrate is found as an isolated defect in 17% of patients and as a combined defect in an additional 10%.

Approximately one-third- no identiable metabolic defect.

1. Absorptive hypercalciuric nephrolithiasis.

Normal calcium intake averages approximately 900-1000 mg/d. one-third is absorbed by the small bowel, and 150- 200 mg excreted in urine. Absorptive hypercalciuria is secondary to increased calcium absorption from the small bowel, predominantly from the jejunum.

This results in an increased load of calcium filtered from the glomerulus. The result is suppression of parathyroid hormone, leading to decreased tubular reabsorption of calcium, an increased absorption of calcium from the small bowel.

Absorptive hypercalciuria can be subdivided into 3 types.

Type I absorptive hypercalciuria is independent of diet and 15% of all calcareous calculi.There is an elevated urinary calcium level (> 150-200 mg/24 h) even during a calcium-restricted diet.

Treatment -Cellulose phosphate is an effective nonabsorbable exchange resin. This effectively binds the calcium in the gut, preventing bowel absorption.

Cellulose phosphate has no impact on the calcium transport defect. Urinary calcium excretion returns to normal values with therapy.

Cellulose phosphate must be taken with meals to be available when calcium is ingested. A typical dose is 10-15 g orally in 3 divided doses and is well tolerated.

Hydrochlorothiazides are an alternative treatment for type I absorptive hypercalciuria.

Type II absorptive hypercalciuria is dietary dependent and is a common cause of urinary stone disease.

Treatment -There is no specific medical therapy. Calcium excretion returns to normal on a calcium-restricted diet.

Patients should limit their calcium intake to 400-600 mg/d.

Type III absorptive hypercalciuria is secondary to a phosphate renal leak and accounts for 5% of all urinary calculi.

Decreased serum phosphate leads to an increase in 1,25-dihydroxyvitamin D synthesis.

Treatment - Orthophosphate - 250 mg 3-4 times daily. It is best taken after meals and before bedtime. Orthophosphates do not alter intestinal calcium absorption.

2. Resorptive hypercalciuric nephrolithiasis-About half the patients with clinically obvious primary hyperparathyroidism present with nephrolithiasis.

This group represents less than 5-10% of all patients with urinary stones.

Hypercalcemia is the most consistent sign of hyperparathyroidism.

Surgical removal of the offending parathyroid adenoma is the only effective way of treating this disease. Attempts at medical management are futile.

3. Renal hypercalciuric nephrolithiasis—Hypercalciuria of renal origin is due to an intrinsic renal tubular defect in calcium excretion. This creates a physiologically vicious cycle. Excessive urinary calcium excretion - decrease serum calcium-secondarily increased parathyroid hormone - increases calcium absorption from the gut.

These patients have an elevated fasting urinary calcium level, normal serum calcium level, and an elevated parathyroid hormone level.

Renal hypercalciuria is effectively treated with hydrochlorothiazides. Stimulate proximal tubular absorption of calcium as well as other constituents. They also increase reabsorption at the distal tubule.

4. Hyperuricosuric calcium nephrolithiasis—Hyperuricosuric calcium

Nephrolithiasis is due to either an excessive dietary intake of purines or an increase in endogenous uric acid production. In both situations there is an increase in urinary monosodium urates.

Monosodium urates absorb and adsorb urinary stone inhibitors and facilitate heterogeneous nucleation.

Patients have elevated urinary uric acid levels (> 600 mg/24 h in women and > 750mg/24 h in men) and consistently have a urinary pH > 5.5. The urinary pH helps to differentiate hyperuricosuric calcium from hyperuricosuric uric acid stone formation.

Patients with excessive purine intake can be effectively treated by changing their diet to one with low purines. Those with excessive endogenous uric acid production can be successfully treated with allopurinol. Allopurinol is a xanthine oxidase inhibitor.

Allopurinol reduces uric acid synthesis and renal excretion of uric acid. It also inhibits uric acid-calcium oxalate crystallization.

Allopurinol has many potential side effects- skin rashes and liver toxicity, and should be administered with careful monitoring (300 mg daily). Potassium citrate is an alternative treatment, especially when associated with hypocitraturia.

5. Hyperoxaluric calcium nephrolithiasis

Secondary to increased urinary oxalate levels (> 40 mg/24 h). found in patients with inflammatory bowel disease or other chronic diarrheal states . It is rarely associated with excessive oxalate intake

Chronic diarrheal states alter oxalate metabolism. Malabsorption leads to increased luminal fat and bile. Intraluminal calcium readily binds to fat, resulting in a saponification process. Urinary calcium levels are usually low (< 100 mg/24 h). The intraluminal gut calcium that normally would have bound to oxalate is decreased.

The unbound oxalate is readily absorbed and is unaffected by the usual metabolic inhibitors of energy-dependent pumps. Bile salts may increase the passive bowel absorption of oxalate.

A small increase in oxalate absorption and subsequent urinary excretion dramatically increases the formation product of calcium-oxalate. This increases the potential for heterogeneous nucleation and crystal growth in this metastable environment.

Other factors-dehydration, hypocitraturia , decreased magnesium, and protein malabsorption. Enteric hyperoxaluric calcium nephrolithiasis is successfully treated with oral calcium supplementation. The calcium binds to the intraluminal oxalate and limits its absorption. It must be given with meals when the oxalate is present. Other oral cations are effective, including magnesium supplements.

Primary hyperoxaluria is a rare hereditary disease. It is associated with calcium oxalate renal calculi, nephrocalcinosis, and other distant deposits of oxalate, culminating in progressive renal failure and eventual death.

Type I enzyme deficiency of alanine glyoxylate transaminase resulting in elevated urinary levels of glycolic acid and oxalic acid.

Type II has increased excretory levels of L-glyceric acid rather than elevated levels of glycolic acid. It is associated with a D-glycerate dehydrogenase enzyme deficiency.

Combined liver and renal transplantation has cured this previously fatal rare disease.

6. Hypocitraturic calcium nephrolithiasis

Citrate is an important inhibitor of urinary stone disease. Increased metabolic demands on the mitochondria of renal cells decrease the excretion of citrate. Such conditions include intracellular metabolic acidosis, hypokalemia (as with thiazide therapy), fasting, hypomagnesemia, androgens, gluconeogenesis, and an acid-ash diet. Citrate may be consumed in the urine by bacteria during a urinary tract infection. The cause of hypocitraturia may be unknown in some cases. In contrast, alkalosis, alkaline-ash diet, estrogen, and vitamin D increase urinary citrate levels.

Citrate has its action in solution. It complexes with calcium, thereby decreasing the ionic calcium concentration and thus the activity product and thereby decreasing the energy for crystallization. Citrate decreases agglomeration, spontaneous nucleation, and crystal growth of calcium oxalate. Citrate also decreases calcium oxalate calculi by decreasing monosodium urates that can absorb inhibitors and facilitate heterogeneous nucleation.

Hypocitraturic (< 320 mg/24 h) calcium nephrolithiasis is associated commonly with renal tubular acidosis type I (distal tubule)

thiazide therapy (accompanied by potassium wastage), and chronic diarrhea.

Treatment - potassium citrate supplementation. Routine dosage is 20-30 mEq 2-3 times daily and is usually well tolerated.

B. Noncalcium Calculi

1. Struvite—Struvite stones are composed of magnesium, ammonium, and phosphate (MAP). They are found most commonly in women and may recur rapidly.

They frequently present as renal staghorn calculi and rarely present as ureteral stones except after surgical intervention .

Struvite stones are infection stones associated with urea-splitting organisms-Proteus, Pseudomonas, Providencia, Klebsiella, Staphylococci etc.

The high ammonium results in an alkaline urinary pH.below 7.2 and MAP crystals precipitate.

MAP crystals are soluble in the normal urinary pH range of 5-7.

Foreign bodies and neurogenic bladders may predispose patients to urinary infections and subsequent struvite stone formation.

Women with recurrent infections despite apparently appropriate antibiotic therapy should be evaluated for struvite calculi with a conventional kidney-ureter-bladder (KUB) film or renal ultrasound, or both.

Culture-specific antibiotics can reduce urease levels by 99% and help reduce stone recurrence. Stone removal is therapeutic.

Long-term management is optimized with the removal of all foreign bodies, including catheters of all varieties. A short ileal loop helps decrease the risk of stones in those with supravesical urinary diversion. All stone fragments should be removed with or without the aid of follow-up irrigations. Hemiacidrin (Renacidin) irrigations should be used with caution if at all.

Acetohydroxamic acid inhibits the action of bacterial urease, thereby reducing the urinary pH and decreasing the likelihood of precipitation.

2. Uric acid—Uric acid stones compose less than 5% of all urinary calculi and are usually found in men.

Patients with gout, myeloproliferative diseases, or rapid weight loss,malignant conditions with cytotoxic drugs . Most patients with uric acid calculi, however, do not have hyperuricemia.

Elevated uric acid levels are frequently due to dehydration and excessive purine intake. Patients present with a urinary pH consistently < 5.5, in contrast to patients with hyperuricosuric calcium nephrolithiasis, who have a urinary pH > 5.5. As the urinary pH increases above the dissociation constant pKa of 5.75, it dissociates into a relatively soluble urate ion.

Treatment-urine volume > 2 L/d and a urinary pH > 6.0. Reducing dietary purines or the administration of allopurinol also helps reduce uric acid excretion.

3. Cystine—Cystine lithiasis is secondary to an inborn error of metabolism resulting in abnormal intestinal (small bowel) mucosal absorption and renal tubular absorption of dibasic amino acids, including cystine, ornithine, lysine, and arginine.

They may present as single, multiple, or staghorn stones. Family history of urinary stones and the radiographic appearance of a faintly opaque, ground-glass, smooth-edged stone. Urinalysis- hexagonal

crystals. Stone analysis confirms the diagnosis. Quantitative urinary cystine - confirm the diagnosis Treatment -

Medical therapy includes high fluid intake (> 3 L/d) and urinary alkalinization.low-methionine (precursor to cystine) diet Penicillamine can reduce urinary cystine levels. Mercaptopropionylglycine (Thiola), 300-1200 mg in divided doses

4. Xanthine—Xanthine stones are secondary to a congenital deficiency of xanthine oxidase. This enzyme normally catalyzes the oxidation of hypoxanthine to xanthine and of xanthine to uric acid. It is of interest that allopurinol, used to treat hyperuricosuric calcium nephrolithiasis and uric acid lithiasis, produces iatrogenic xanthinuria.

Blood and urine levels of uric acid are lowered, and hypoxanthine and xanthine levels are increased. Urinary stones develop in 25% of patients with a xanthine oxidase deficiency. The stones are radiolucent and are tannish-yellow in color.

Treatment- High fluid intake and urinary alkalinization are required for prophylaxis. If stones recur, a trial of allopurinol and a purine-restricted diet is appropriate.

5. Indinavir—Protease inhibitors are a popular and effective treatment in patients with acquired immunodeficiency syndrome. Indinavir is the most common protease inhibitor that results in radiolucent stones in up to 6% of patients who are prescribed this medication. Indinavir calculi are the only urinary stones to be radiolucent on noncontrast CT scans.

6. Rare—Silicate stones are very rare and are usually associated with long-term useof antacids containing silica. Surgical treatment is similar to that of other calculi.

Triamterene stones are radiolucent and have been identified with an increased frequency. They are associated with antihypertensive medications containing triamterene, such as Dyazide. Discontinuing the medication eliminates stone recurrences. Other medications - glafenine and antrafenine.

Urolithiasis and renal failure

Urolithiasis is not a common cause of renal failure. Occur in bilateral stone with obstruction, solitary functional kidney,associated risk factors that cause renal injury.

Risk factors

1) Diabetes Mellitus

2) Hypertension

3) struvite staghorn calculi-increased risk of proteus infection,recurrence of stone if not cleared,chances of complete obstruction.

4) Urinary tract infections-urosepsis.

5) Percutaneous and extracorporeal urological methods -chronic deterioration of renal function if treated with multiple therapeutic sessions.

PREDICTORS OF RENAL FUNCTION IN UROLITHIASIS

Preoperative

Duration of symptoms,Solitary functioning kidney,bilateral stone disease,Coexisting hypertension and diabetes,Stone burden,Atrophic renal cortex .,NSAID intake,functional or anatomical urological anomalies .

Intraoperative -Number of PCNL tracts, ESWL—sessions,

Postoperative- Residual fragments, Recurrent infection Proteinuria, Recurrence of calculus

Management of urolithiasis patients with obstruction and infection-Emergency settings

- Correct dehydration.
- Ranal function tests to assess renal function.
- Treat urinary infections –start the patient on empirical third generation cephalopsporins.
- Avoid using medications that may be detrimental to renal function.
- Identifying patients with a solitary functional kidney,
- Reduce risks of acute renal failure from contrast nephrotoxicity, particularly in patients with preexisting azotemia (creatinine >2 mg/dL), diabetes, dehydration, or multiple myeloma
- Choosing imaging studies that do not require intravenous contrast (eg, ultrasound, abdominal flat plate radiographs, noncontrast CT scans) .

- Releive obstruction-PCN OR Double J stenting.

- Definitive treatment-once sepsis settles.

Function Assessment

SCr level is widely used as an index of renal fuction. Total creatinine clearance exceeds the GFR because of tubular secretion, whereas the urea clearance is usually lower than the GFR because of tubular reabsorption. The mean of the creatinine and the urea clearance is a more accurate estimate of the GFR than either is separately. Factors associated with creatinine excretion (age, gender, ethnicity, tubular creatinine secretion and inhibition) may affect the accuracy of standard SCr measurements. The MDRD study equation to predict the GFR incorporates SCr concentration, demographic characteristics (age, gender, ethnicity), and other serum factors (urea, serum albumin). It is more accurate than other more widely used predictive equations or parameters

The MDRD equation for demographic and serum variables is:

$GFR = 170 \times [P_{Cr}]^{-0.999} \times [age]^{-0.176} \times [0.762$ if patient is female$] \times [1.180$ if patient is black$] \times [SUN]^{-0.170} \times [Alb]^{-0.318}$

Although the MDRD equation is better than other standard measures for GFR, it is inaccurate for patients not in a steady state for creatinine balance or those with a medical condition interfering with creatinine excretion or creatinine assay or both

Considering the current K/DOQI staging of renal function linked to level of GFR, a number of medical centers and commercial laboratories are using estimated GFR (eGFR) from SCr as a primary method of reporting kidney function because of inadequacies in SCr alone. The MDRD study equation is used most commonly to estimate GFR. The estimation of GFR from the MDRD study equation is not appropriate for patients who have rapidly changing kidney function, are at the extremes of age and body size, exhibit malnutrition or obesity, are paraplegic or quadriplegic, consume a vegetarian diet, or have diseases that have an impact on the skeletal muscle status. Moreover, in sick, hospitalized patients with moderately advanced renal failure, the Cockcroft-Gault, MDRD equations, and eGFR perform poorly when estimating GFR in this patient population and are not reliable measurements of RRF . In instances when the eGFR may not be accurate, a 24-hour urine collection for creatinine plus urea clearance divided by 2 is recommended.

Proteinuria

Increased excretion of albumin is a sensitive marker for renal damage attributable to diabetes mellitus, glomerular disease, interstitial disease, and hypertension. In adults albuminuria can be identified by the use of albumin dipstick, urinary albumin concentration (UAC), albumin/creatinine ratio (ACR) measured in a spot morning urine sample, or urinary albumin excretion greater than or equal to 30 mg over 24 hours . Traditionally the cutoff value indicating a urinary albumin excretion greater than 30 mg/24 hr has been 3 mg/L . The standard urine protein dipstick is insensitive for low concentrations of albumin (<10 mg/dL) and for some immunoglobulin light chains. Dehydration, hematuria, exercise, infection, and extremely alkaline urine (pH > 8) can cause false-positive dipstick readings.

Radiographic Assessment

The radiographic assessment should take into account the impact of contrast on renal function. Imaging techniques in CKD are usually utilized for investigation of new ARF or for investigation of potential problems in nonrenal sites (e.g., cardiac catheterization, peripheral vascular concerns, abdominal investigations). Contrast material can induce worsening of underlying renal disease to the point of requiring

RRT. All patients with abnormal SCr greater than 2 mg/dL should be considered for alternative diagnostic testing and prophylactic preventive strategies to avoid worsening renal function. Specific measures may help lower the risk for ARF in patients undergoing radiographic assessment. These include volume expansion, hydration with intravenous administration of normal saline solution (sodium chloride 0.9%), one-half strength saline solution (sodium chloride 0.45%), sodium bicarbonate solution, infusion of mannitol, administration of atrial natriuretic peptide, loop diuretics, calcium antagonists, theophylline, dopamine, *N*-acetylcysteine, low-osmolar contrast, iso-osmolar contrast media, use of gadolinium-based contrast medium, CO_2 angiography, hemodialysis shortly after contrast agent administration, and avoidance of short intervals between contrast studies

A number of reports have emphasized the value of normal saline solution hydration in patients to generate a urine flow rate of 100 to 150 mL/hr, thus decreasing the risk for contrast agent–induced nephropathy . Merely hydrating patients without ensuring effective urine flow is less valuable. There is no additional benefit to mannitol in the diabetic patient undergoing radiocontrast procedures; however, sodium bicarbonate solution infusion along with *N*-acetylcysteine may be of help. The antioxidant *N*-acetylcysteine (600 mg orally twice a day on the day

before and on the day of administration of the contrast agent) along with hydration using a 0.45% saline solution intravenously may decrease the risk of ARF from radiographic contrast agents in this patient group

Measures to decrease renal deterioration

Regardless of the nature of the initial insult, once a critical number of nephrons is destroyed, a steady decline in GFR occurs as the progressive loss in viable nephrons occurs. And yet, it is becoming increasingly clear that well-designed renal assessment and management programs are feasible and can be systematically applied to large numbers of patients to decrease the rate of progression before and after stone management.

International Society of Nephrology 2004 Consensus Workshop on Prevention of Progression of Renal Disease Lifestyle modifications, blood pressure control, glycemic control, reduction of proteinuria, protein restriction, lipid control, avoidance of nephrotoxic agents, early referral to a nephrologist, correction of anemia, optimization of calcium-phosphorus product, correction of acidosis, and maintenance of fluid balance.

Various pharmacologic trials comparing different medications have shown that patients with better blood pressure control have significantly slower rates of deteriorating kidney function.

Angiotensin II is critical in causing progressive renal disease by both hemodynamic and nonhemodynamic mechanisms. Blockade of the renin-angiotensin system contributes to preservation of renal function by decreasing glomerular pressure and proteinuria. Because proteinuria plays a sentinel role in renal scarring, a reduction in proteinuria correlates with slowing of disease progression .

Clinical research findings support the view that preservation of renal function through angiotensin blockade can be achieved in patients with either diabetes mellitus or nondiabetic nephropathy.

Patients with diabetes mellitus and microalbuminuria were treated with ramipril ACE inhibitors also prevent the slow decline in P_{O_2} within the kidney. Considering the “hypoxia injury hypothesis,” a potentially important renoprotective mechanism of ACE inhibition could stem from this improvement in interstitial capillary P_{O_2} levels, therefore decreasing the risk of renal sclerosis.

Dietary modifications can reduce renal hyperfiltration injury. Formulations include

Low-protein (0.6 g protein/kg ideal body weight) diets,

Very low-protein diet (0.3 g protein/kg/day predominantly vegetable protein) supplemented with essential amino acids,

Very low-protein diet supplemented with both essential amino acids and nitrogen-free analogs of amino acids

MATERIALS AND METHODS

Study group: Patients who were admitted in the Kilpauk Medical College Hospital and Govt. Royapettah hospital, Chennai , with clinical diagnosis of urolithiasis from January 2009 – March 2011.

1) Study design: cross sectional study

Inclusion criteria

1) Patients with stone disease.

2) Increased renal parameters

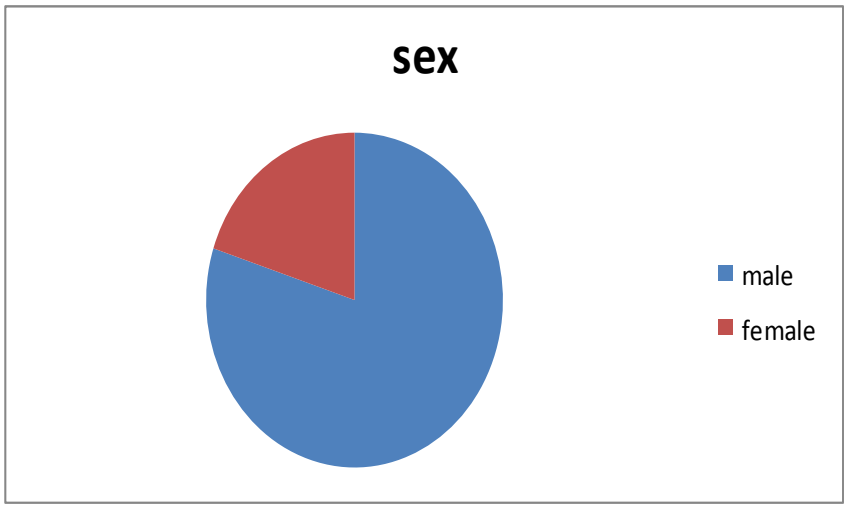
Exclusion criteria:

1) Known case of medical renal disease.

2) Patients with associated congenital anomalies of urinary tract causing obstruction.

Total number of patients included in the study are 25.

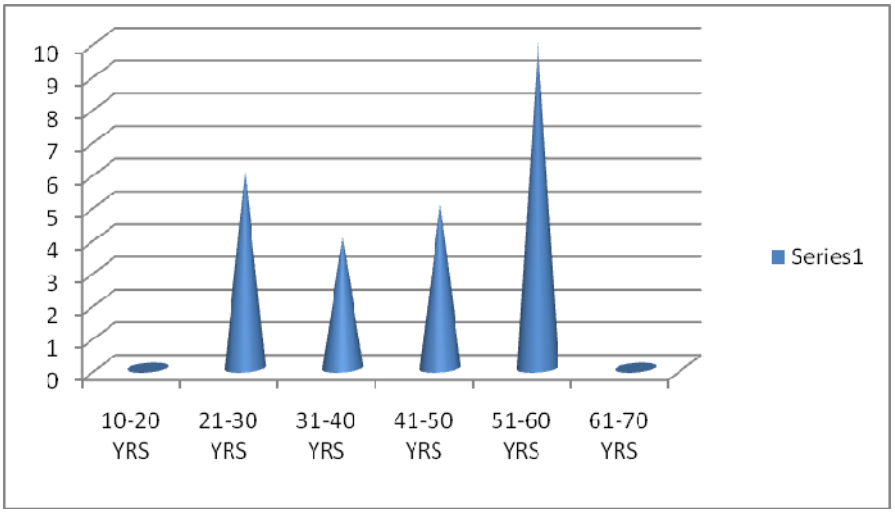
Among them 20 were male and 5 were female patients



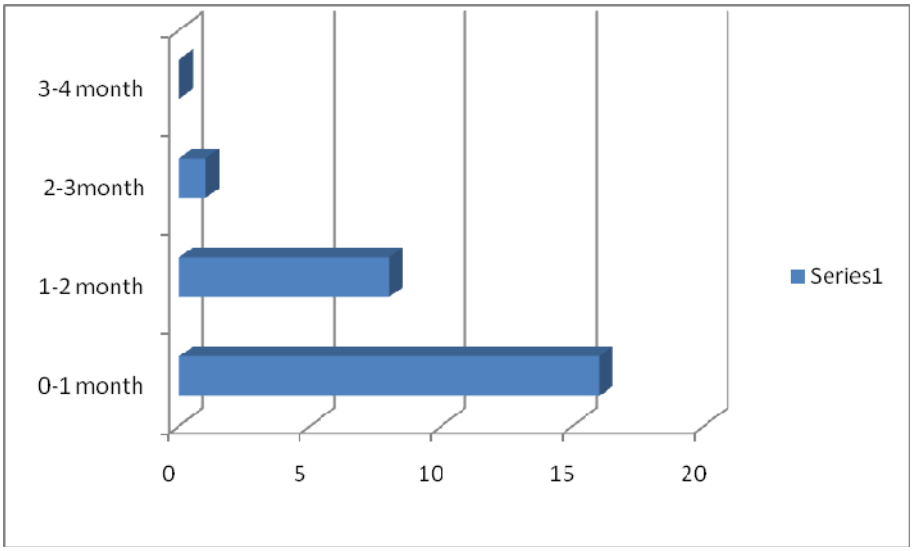
1) **AGE(yrs)** **No**

20-30	6
31-40	4
41-50	5
51-60	10

Age distribution of the patients is mainly middle age.



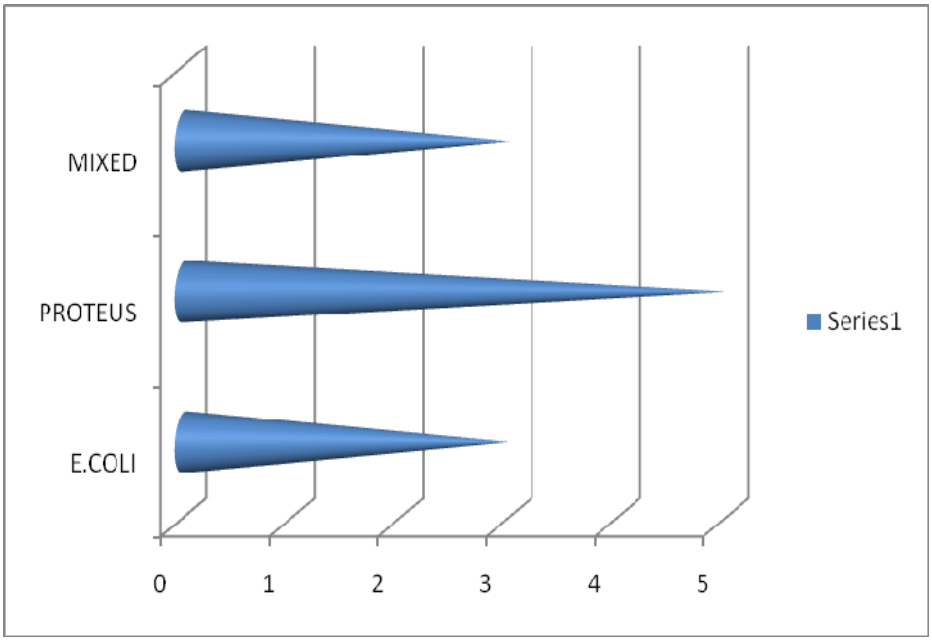
2) DURATION OF SYMPTOMS



Most of the patients had vague loin pain . 4 patients presented with fever, chills, they are initially managed with systemic antibiotics before proceeding with further evaluation

3) URINE CULTURE

After urine routine culture was done which showed E.Coli growth in 3 patients and Proteus growth in 4 patients Mixed growth was seen in 2 patients Appropriate antibiotics were given according to the sensitivity.



4) COMORBID ILLNESS

Patients history of medical,surgical illness is obtained.

Blood investigations – sugar ,urea, creatinine, electrolytes, Chest X-Ray PA view taken to find any pulmonary tuberculosis

Blood pressure recorded

Electrocardiogram taken in all patients

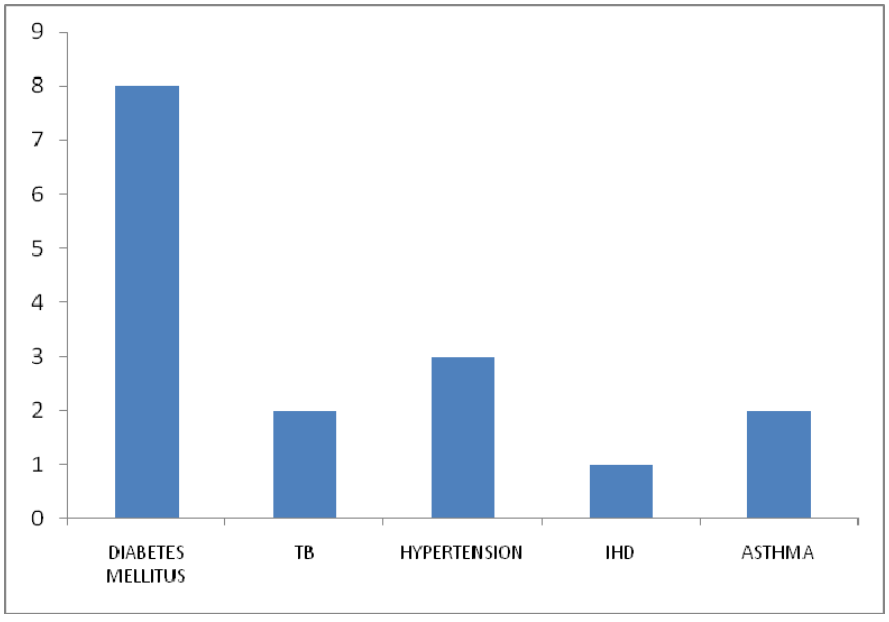
8 had Diabetes mellitus,out of which 3 patients had associated hypertension.

All the patients were evaluated for glycosylated hemoglobin and GTT done .Anti diabetic treatment and anti hypertensives were started

2 had TB

1 had Ischemic heart disease

2 had Asthma



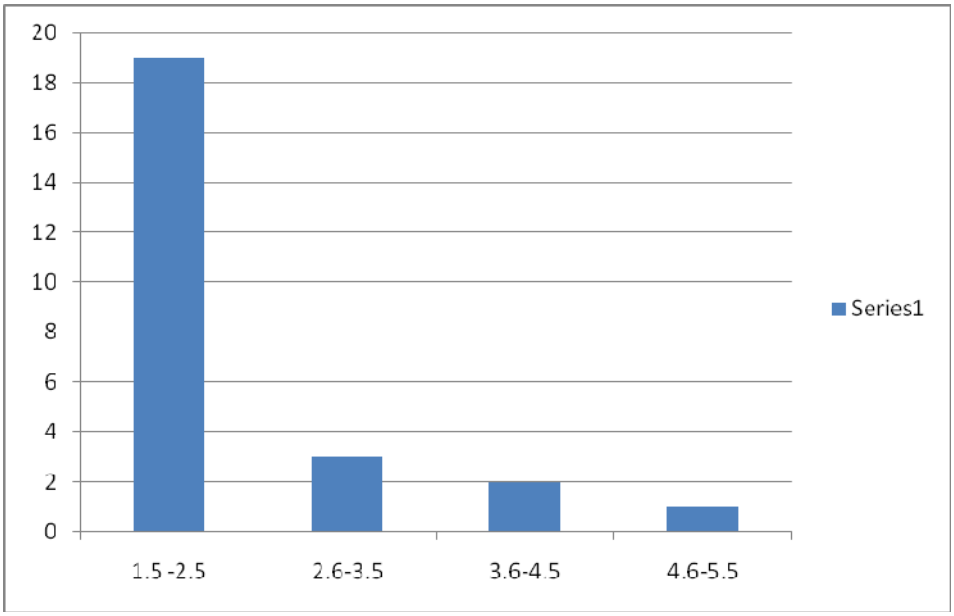
5) SERUM CREATININE

Creatinine is measured using jaffes method .

Patients who are known cases of medical renal disease are excluded from my study

Base line creatinine of 1.5 mg% is taken as the criteria of renal insufficiency

19 had creatinine 1.5-2.5,3 had 2.6-3.5,2 had 3.6-4.5,1 had 4.6-5.5 Dialysis was done in the patient with creatinine 5mg% and operated

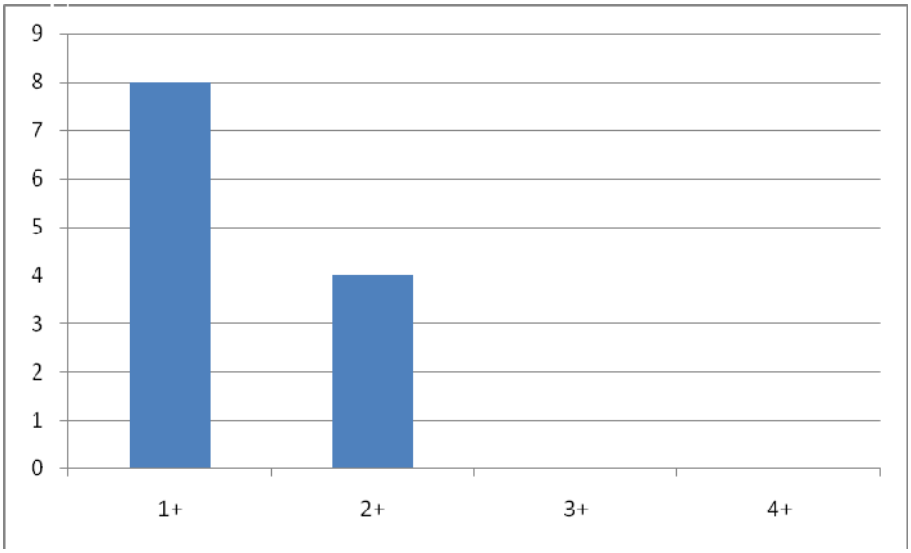


6) PROTEINURIA

Urine protein estimated since it represents the degree of renal damage and it correlates directly with the recoverability of renal function after surgery

8 patients had 1+ proteinuria,

4 Patients had 2+ proteinuria.

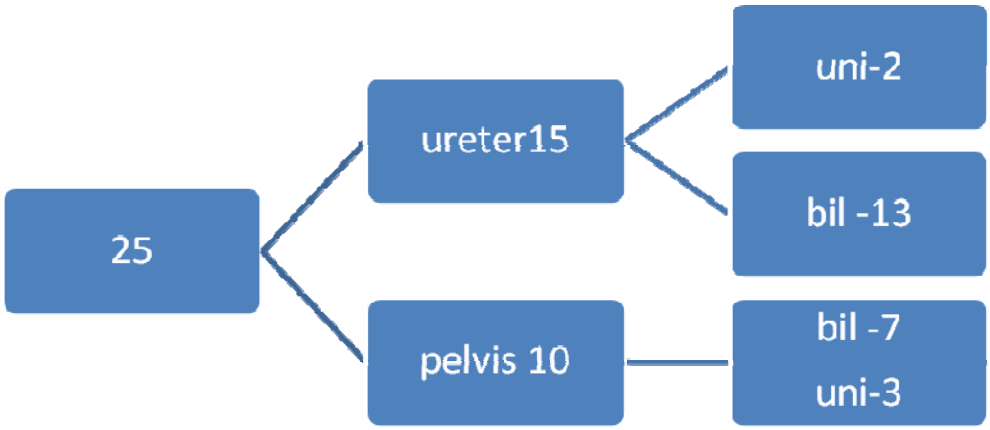


7) STONE LOCATION

All the patients were evaluated with USG and X-Ray KUBU.

Plain CT KUBU is taken in patients with irregularity in renal contour.

Kidney size, contour, degree of hydronephrosis, parenchymal thickness, internal echoes, perinephric collections, malignancy were assessed Size, location ,density of stone is assessed.



STONE SIZE

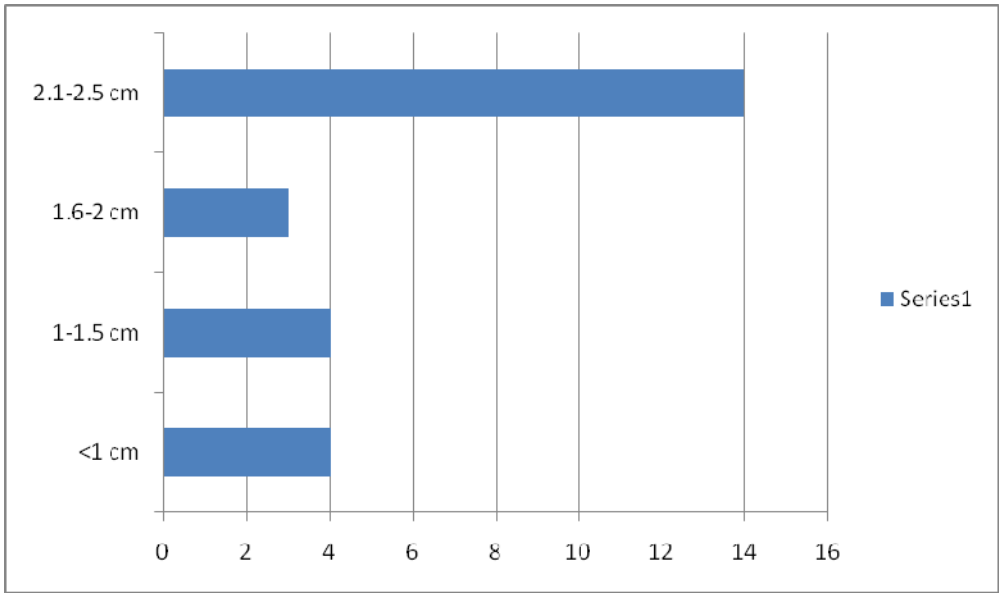
Ranged from 1cm to 5.8cm

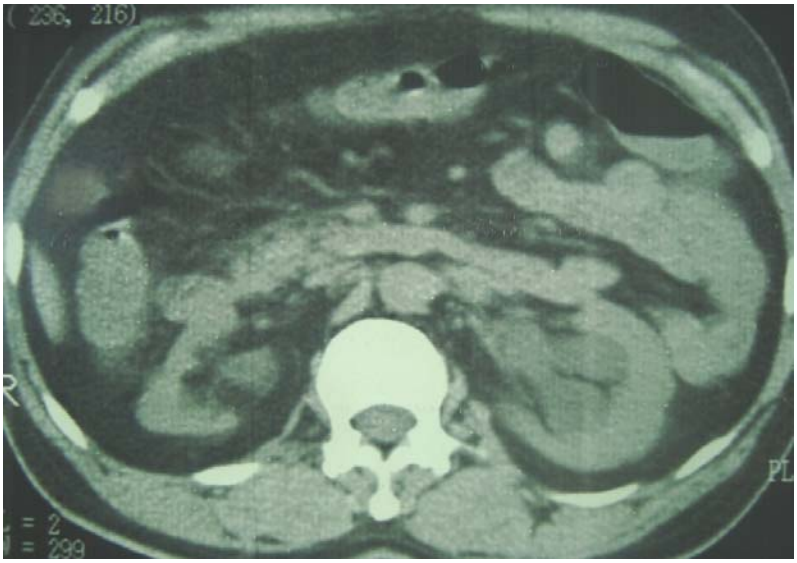
PARENCHYMAL THICKNESS

Assessed by USG,CT-KUBU(Plain)

17 patients had good parenchymal thickness 2 -2.5cm

8 patients had reduced parenchymal thickness





Bilateral ureteric calculus with decreased cortical thickness

METABOLIC EVALUATION-

Serum calcium,uric acid,PTH,electrolytes,urine ph,urine calcium was evaluated in all patients.

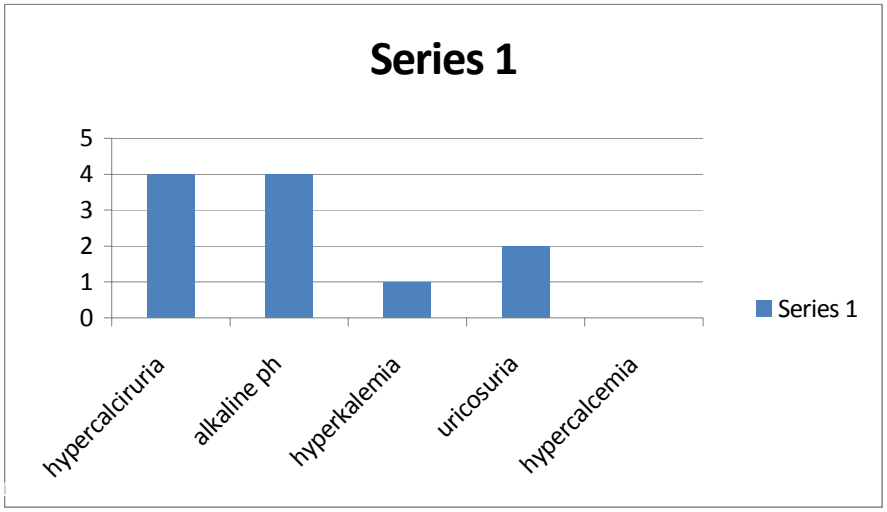
Hypercalciruria is seen in 4 patients.

PTH was normal in all patients.

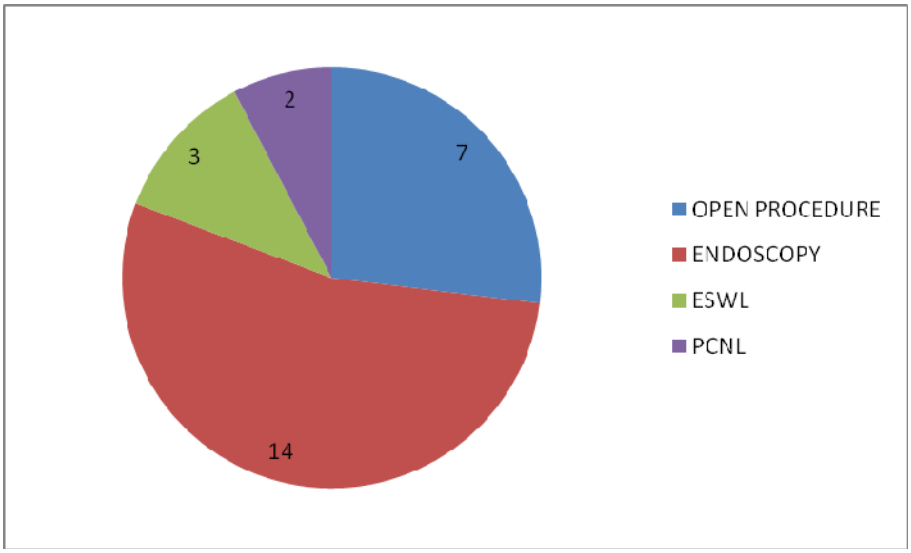
All the patients had normal electrolytes.

Urine ph is alkaline in 4 patients suggesting of infection by urease splitting organisms.

Stone analysis- out of 15 cases for which stone analysis was done five were struvite stones and rest of them calcium oxalate stones.



Type of procedure



In four patients who presented with fever and pain initial PCN was done under USG Guidance

Turbid urine was drained and sent for urinalysis and culture and sensitivity.

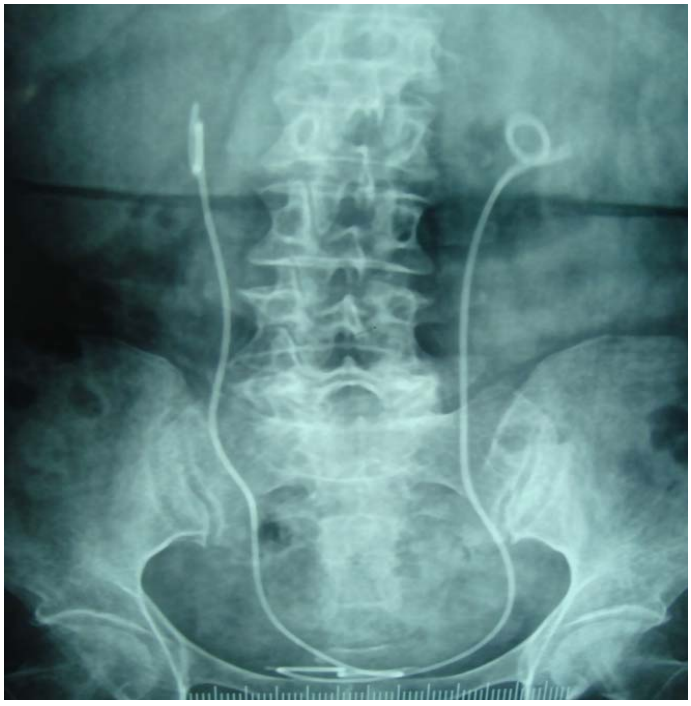
In these patients open procedure was undertaken after the infection settled. Open procedure was done in 7 patients with renal stones.bilateral renal stones procedure was done on the symptomatic or better functioning side first followed by the other side after 3 months and there is no per operative difficulty in any of these patients.post operative X-Ray showed residual stones in the lower calyx in 2 cases for which ESWL was done.

ESWL is done in 3 patients with stone size 1cm after stenting.two patients had bilateral upper ureteric calculous obstruction.one patient had bilateral renal calculus for which stenting was done before ESWL.complete stone clearance was achieved .stent removal was done after 3 weeks.

PCNL was done in 2 patients.one was two staged procedure in which PCNL was undertaken after 2 weeks of PCN.

Nephroscope with pneumatic lithotripsy was used and stones were fragmented,antegrade stenting was done, Ureteroscopy with pneumatic lithotripsy was done in 14 patients with Good fragmentation was achieved and stenting was done. Ureterolithotomy was done in one patient with an impacted upper ureteric calculus,

POST RIGHT PYELOLITHOTOMY WITH LT URS WITH DJ STENT



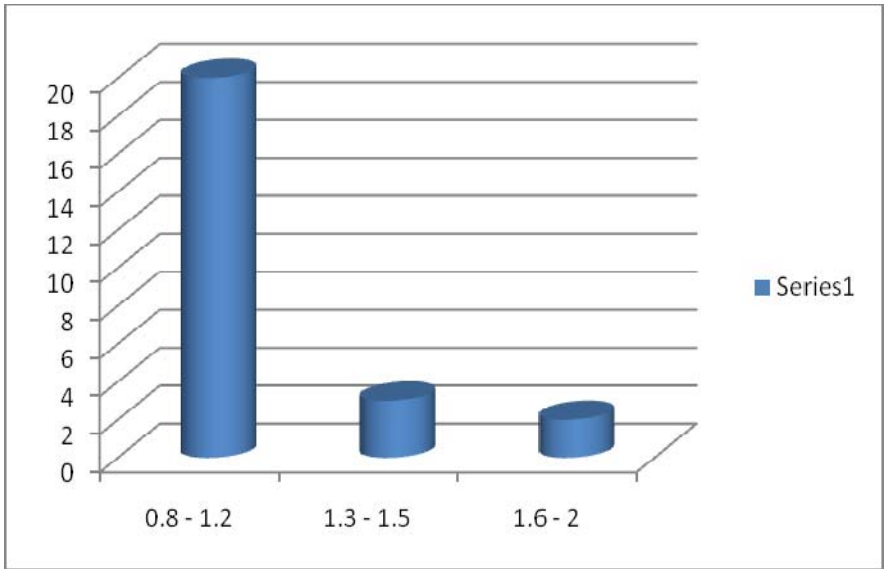
POST OPERATIVE CREATININE

After a duration of two weeks the follow up creatinine gradually decreased and reached various nadir levels.

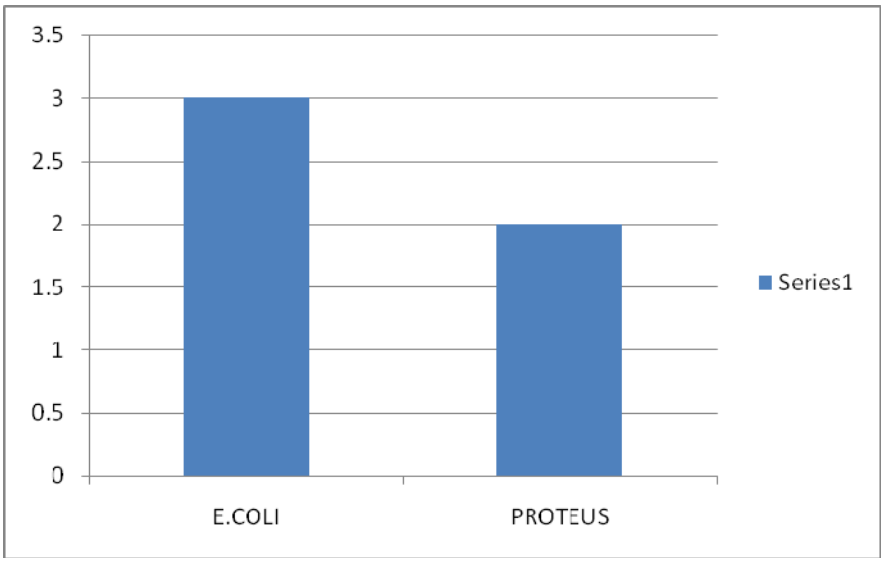
Normal creatinine values seen In 20 patients .

1.2-1.5mg% in 3 patients,

1.6 -2mg% achieved in 2 patients



POST OPERATIVE UTI



5 patients had post operative fever and urine culture in these patients showed E.Coli in 3 pts and proteus growth in 2 pts. All the 5 patients settled on antibiotic treatment

CORTICAL ATROPY

Post Obstructive atrophy changes were seen in 2 patients but renal function maintained in borderline. None of these needed dialysis support on follow up.

PROTEINURIA

Proteinuria resolved in 10 patients .The other two had minimal proteinuria

STONE RECURRENCE

All the patients on follow up of 2 yrs

No stone recurrence was seen

RESIDUAL FRAGMENTS –

Seen in 3 patients and ESWL was done in two of these patients. Third patient is on follow up.

STONE ANALYSIS –

5 stones were found to be struvite stones and rest are calcium oxalate stones.

RESULTS

Complete clearance defined as no visible fragment on KUB films and renal ultrasonography at 1 month follow-up was achieved in 22 patients (88%), while 3 patients (12%) had residual fragments.

Over a mean follow-up of 1 to 2 years , 1 patients showed evidence of recurrent calculi, and 5 patients had recurrent urinary tract infection.

Overall, 20 patients (80%) showed improvement, 5 patients (20 %) showed stabilization.

The baseline serum creatinine concentration correlated well with the postoperative renal function which stabilized or improved in nearly all patients.

DEMOGRAPHIC DATA

No. of patients 25

Mean age (years) -40 yrs

Sex (% male) 72%

Side (% right) 53%

Complete staghorn -3

Partial staghorn -2

Pelvic -5

Mean calculus size -3cm

Prior stone treatment -2

Associated hypertension -3

Associated diabetes -8

Associated urinary infection -8

Nephrostomy drainage required -4

Dialysis required preoperatively -1

Mean postoperative serum creatinine (mg/dL)-1mg%

FACTORS EVALUATED

Preoperative -Age **,**Duration of symptoms**,**Coexisting hypertension and diabetes**,**Stone burden**,**Atrophic renal cortex

Intraoperative-Procedure done URS,ESWL,OPEN,PCNL.

Postoperative- Creatinine, Residual fragments, Recurrent infection Proteinuria, Recurrence of calculus. Out of the 5 patients who had stable renal function but elevated serum creatinine 3 patients were diabetic with superimposed infection.

Serum creatinine improved in them after antibiotics .these patients are referred to nephrologist for further assessment and treatment to improve renal function.

They were adviced to follow the protein and salt restricted diet ACE inhibitors were prescribed and the patients responded well to this treatment.

Strict follow up is adviced in both urology and nephrology departments. 2 patients in this group had dilated and thinned out parenchyma and post operative creatinine was higher than normal.

None of them required dialysis support in the follow up period of 2 years. In patients with high base line serum creatinine post operative creatinine decreased only moderately depicting the glomerular injury that has occurred. In our study out of the various parameters studied Stone location rather than stone size was found to be an important factor since ureteral calculus in both kidneys or stone in solitary kidney were having high creatinine but good post operative improvement.

Choice of procedure does not seem to alter the outcome in these group of people.as per literature each PCNL procedure has a 1% loss of nephrons and each ESWL session has 2%-4% loss of nephrons.but with limited number of cases studied we are not able to comment on this aspect authentically.but for a large calculus where complete stone clearance is a must open procedure will minimize the loss of nephrons and also gives a better stone clearance.

Large stag horn calculus if nonobstructing had good preserved renal function. Of the various factors Preop serum creatinine ,cortical thickness, diabetes mellitus, proteinuria, and recurrent urinary infection were found to correlate with post operative renal deterioration .

DISCUSSION

Few studies have addressed the problem of renal insufficiency in patients with urolithiasis (1–4).

Marengella and coworkers (2) reported an 18% incidence of renal insufficiency in frequently relapsing idiopathic calcium stone formers.

Patients with renal calculous disease, particularly those with struvite staghorn calculi, are prone to renal damage because of various combinations of renal obstruction, infection, and surgical intervention. (1,3-5)

Calcium oxalate crystals are known to stimulate the proliferation of renal interstitial cells, thus triggering interstitial scarring (5).

Once chronic renal insufficiency is established, further progression has been attributed to hyperfiltration of the remaining functional nephrons (6).

Witherow and Wickham(7) in their studies suggested that long-term renal function preservation in patients with complex stone disease

depends on adequate blood pressure control, stone size, and stone clearance status.

They also reported that renal deterioration occurs more frequently in patients with stone disease in a solitary kidney (7).

Amanullah and associates (17) in a study at Chandka Medical College, Larkana, Pakistan found that Out come of patients with calculus anuria is good, if early diagnosis is made and prompt treatment should be given on emergency basis

Renal-specific mortality was 30% in the conservatively managed patients. **Chandhoke and associates(11)** found no significant deterioration in renal function in patients with mild or moderate renal insufficiency who underwent treatment for renal calculi with either SWL or PCNL. However, four of the five patients with creatinine values 3 mg/dL showed deterioration. In a study of long-term renal function after staghorn calculus management -the renal cause-specific mortality was 0 for patients with complete stone clearance (by PCNL, SWL, or combination therapy) and 67% in patients managed expectantly.

Renal functional outcome

In our study, the preoperative baseline serum creatinine concentration was an accurate predictor of postoperative renal function deterioration:

100% of patients with serum creatinine 2.5 mg/dL, 96% of patients with creatinine between 2.5 and 3.5 mg/dL, and 40% of patients with serum creatinine between 3.5 and 5.5 mg/dL revealed stabilized or improved renal function over a mean follow-up of1 years.

Our study revealed atrophic renal parenchyma, large stone burden, diabetes mellitus,proteinuria at follow-up, and recurrent urinary infection were predictors of postoperative renal function deterioration

Significant proteinuria and cortical atrophy indicate extensive glomerular and tubular functional loss(12,13).

Patients with struvite calculi develop renal function impairment because of obstructive or pyelonephritic episodes(5).

Aggressive treatment of obstruction and infection has been known to improve acute renal dysfunction.

Large stone burdens may be associated with a greater degree of tubular obstruction, intrarenal interstitial fibrosis, and glomerulosclerosis, which may lead to nephron loss and a reduced glomerular filtration rate.

Teichman and coworkers (10) reported solitary kidneys to be more frequently associated with renal deterioration in patients with staghorn calculi.

The safety of PCNL with respect to functional nephron loss is well established. (14,15)

SWL being reserved for residual calculi. serum creatinine alone is not the ideal estimate of renal function.

Creatinine clearance and renal scans may be more reliable in this regard.

Few studies have addressed the problem of renal insufficiency in patients with nephrolithiasis.

In a survey of 2000 patients by Gupta and associates,3 only 33 patients (1.7%) experienced low to moderate grade renal insufficiency.

In our series 23 patients improved after adequate relief of obstruction.

CONCLUSION

Most patients presenting with renal insufficiency due to calculous disease experience improvement or stabilization of renal function with early aggressive intervention aimed at complete stone clearance.

Serum creatinine- was the most important predictor of renal recovery high baseline creatinine indicated severity of renal damage, post operative creatinine showed only moderate decrease in these patients.

Cortical atrophy –if associated with bilateral obstruction recoverability is moderate.even in unilateral cortical thinning opposite kidney goes for hyperfiltration injury and focal nephrosclerosis.

Renal preservative measures followed during this period helps in preventing hyperfiltration renal damage.

Diabetes mellitus- patients with DM had more chances of progressing to renal insufficiency following calculous obstruction due to diabetic nephropathy with superimposed resistant urinary tract infections.even in this group early intervention by diabetic control,infection control and stone removal showed good results

Large stone burden- implied more fibrotic reaction .stag horn calculus were having preserved parenchyma due to lack of obstruction.Recurrent or persistent urinary infection in these patients were associated with high incidence of post operative renal deterioration.

Residual stones- Meticulous attention should be given towards stone clearance since residual stones can regrow, cause obstruction, and urinary tract infection

Morbidity and mortality is reduced in these group of patients by judiciously handling the condition with timely interventions and further prevention of renal deterioration and follow up of these patients.

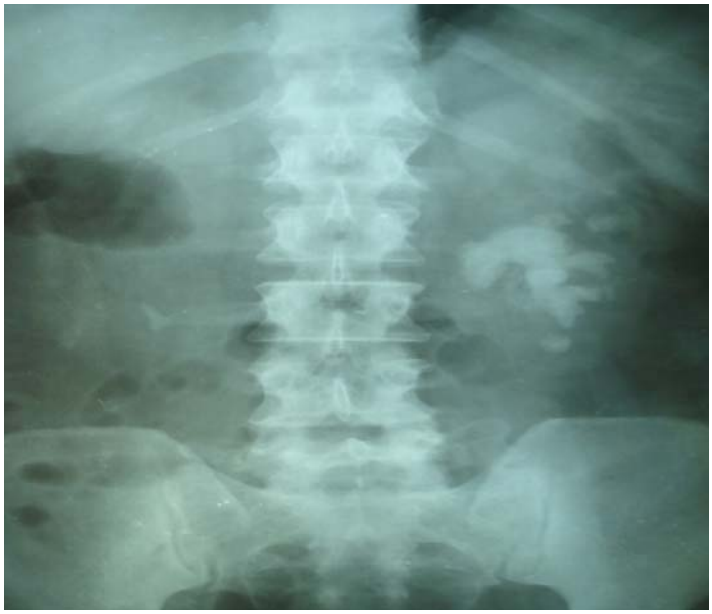
MASTER CHART

S. NO	NAME	AGE/SEX	IP. NO	STONESIZE&LOCATION	CREATININE	U -C/S	U-PROTEIN
1	ARUN KUMAR	40/M	10034	B/L URETER-1CM	1.6	NG	NIL
2	SELVAM	49/M	12429	LT.PELVIS-5CM	1.8	E.COLI	2+
3	SELVANATHAN	50Y/M	11307	LT.PELVIS-3.5CM	3.4	NG	1+
4	KRISHNAMOORTHY	30Y/M	961199	RT.PELVIC-1.5CM	1.9	E.COLI	1+
5	GOVINDARAJ	60/M	961299	LT URETER-1CM	1.9	PROTEUS	2+
6	GURUVAPPAN	50/M	12131	RT.RENAL -3.5CM	3.2	E.COLI	1+
7	DEVARAJ	50/M	2398	RT.RENAL -2CM	2	PROTEUS	NIL
8	RAJENDRAN	48/M	28872	B/L URETER-1CM	1.5	NG	NIL
9	SARAVANAN	41/M	30262	B/L URETER-1CM	2	NG	NIL
10	KRISHNAN	24/M	9912	B/L STAGHORN	1.7	NG	1+
11	MANIKANDAN	31/M	9909	B/L URETER-0.8CM	2.8	E.COLI	NIL
12	THIRUMALAI KUMAR	52/M	943284	B/L VUJ CALCULUS	1.8	NG	NIL
13	MURUGAN	31/M	947722	B/L PELVIC CALCULUS	2	PROTEUS	1+
14	VENKATESH	70/M	942801	LT URETER-1CM	2.1	E.COLI	1+
15	MOHANAMBAL	41/F	942803	B/L VUJ CALCULUS	2	NG	NIL
16	DURAI	56/M	941600	LT URETER-1CM	1.8	NG	NIL
17	JAIRAM SINGH	36/M	970829	B/L URETER-1CM	2	NG	NIL
18	DANIEL	44/M	969121	B/L URETER-1CM	3	NG	2+
19	HARIDASS	34/M	959068	B/LPELVIC-1.5CM	2	MIXED	1+
20	RANGANATHAN	48/M	957008	B/L URETER -1.5CM	2	NG	NIL
21	ADITHYAN	18/M	967856	B/L URETER-1.5 CM	1.5	PROTEUS	NIL
22	JULIET	41/F	930747	LT PELVIC -3 CM	5.5	PROTEUS	2+
23	MALLIKA	42/F	11232	B/L URETER-1.5 CM	1.6	NG	NIL
24	MUNIAMMAL	60/F	921931	RT URETER-2 CM	4.5	MIXED	1+
25	INDIRA	38/F	928713	B/L URETER -1.5CM	1.8	NG	NIL

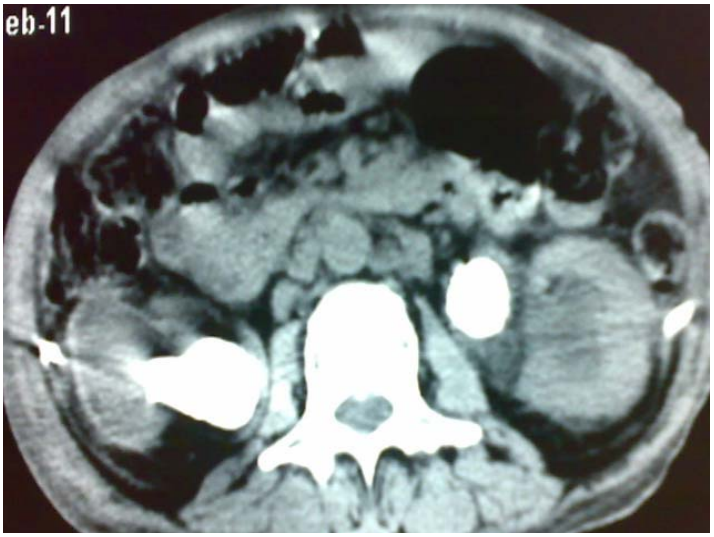
S.NO.	NAME	COMORBID	CORTEX	PROCEDURE	POST OP CREATININE	UTI
1	ARUN KUMAR	NIL	3CM	B/L URS	0.8	NIL
2	SELVAM	DM	2CM	LTPYELOLITHOTOMY	1.5	NIL
3	SELVANATHAN	DM/HT	2CM	LTPYELOLITHOTOMY	1	NIL
4	KRISHNAMOORTHY	NIL	2CM	ESWL	0.8	NIL
5	GOVINDARAJ	DM	1.3CM	LT URS	1.4	E.COLI
6	GURUVAPPAN	NIL	1.5CM	RTPYELOLITHOTOMY	2	NIL
7	DEVARAJ	DM/TB	2CM	PCNL	1	NIL
8	RAJENDRAN	NIL	2.5 CM	B/L URS	1	NIL
9	SARAVANAN	Asthma	2CM	B/L URS	0.9	NIL
10	KRISHNAN	NIL	2CM	B/L PYELOLITHOTOMY	1	NIL
11	MANIKANDAN	NIL	2.5CM	B/L URS	1.2	NIL
12	THIRUMALAI KUMAR	Asthma	3CM	B/L URS	0.8	NIL
13	MURUGAN	NIL	1.4CM	RT OPEN /LT ESWL	1.4	PROTEUS
14	VENKATESH	DM	1.5 CM	ESWL	2	ECOLI
15	MOHANAMBAL	NIL	2CM	B/L URS	1	NIL
16	DURAI	TB	2CM	ESWL	1	NIL
17	JAIRAM SINGH	NIL	2CM	B/L URS	1	NIL
18	DANIEL	DM/HT	2CM	B/L URS	1	NIL
19	HARIDASS	NIL	2CM	PCNL	1	NIL
20	RANGANATHAN	NIL	2CM	B/L URS	1	NIL
21	ADITHYAN	NIL	2.5CM	B/L URS	0.7	NIL
22	JULIET	DM/HT	1.3CM	LT PYELOLITHOTOMY	1.8	ECOLI
23	MALLIKA	NIL	3CM	B/L URS	0.9	NIL
24	MUNIAMMAL	DM	1.5CM	RT URETEROLITHOTOMY	1.3	PROTEUS
25	INDIRA	NIL	2.5CM	B/L URS	1	NIL

LEFT STAG HORN CALCULUS

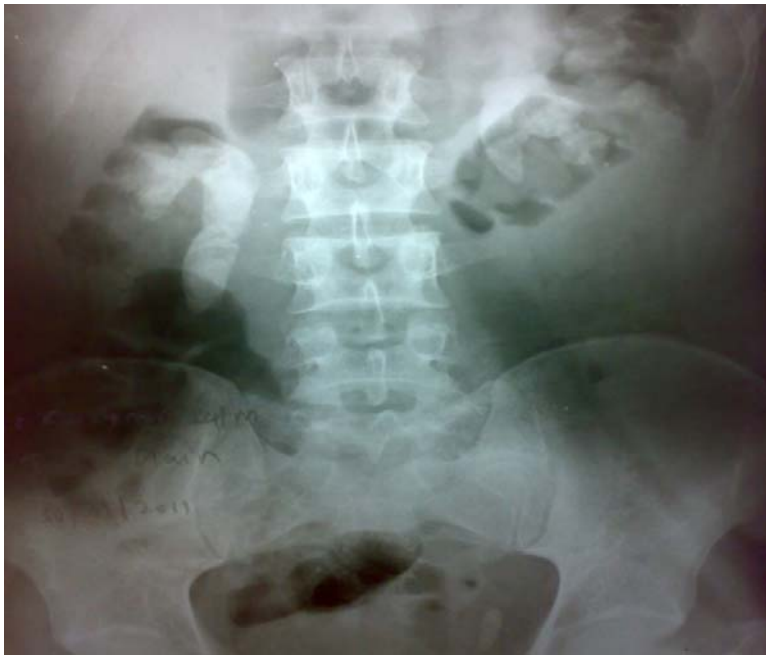
.



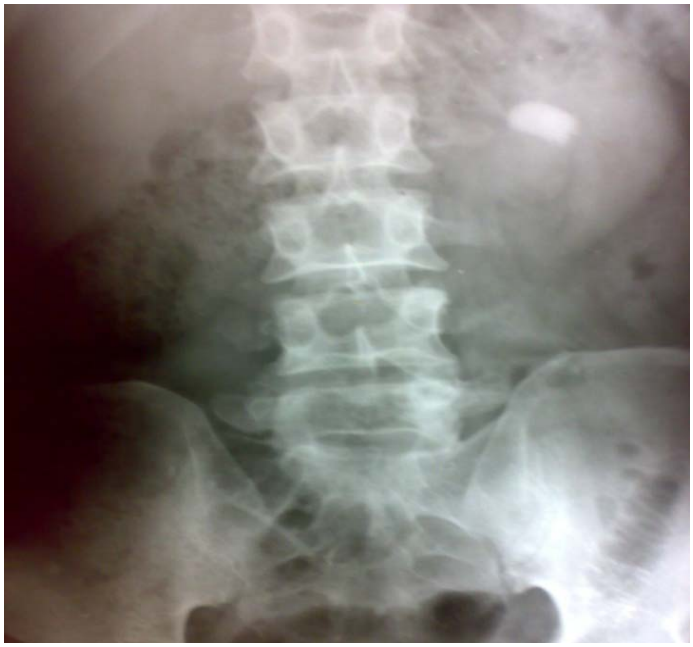
BILATERAL PELVIC CALCULUS



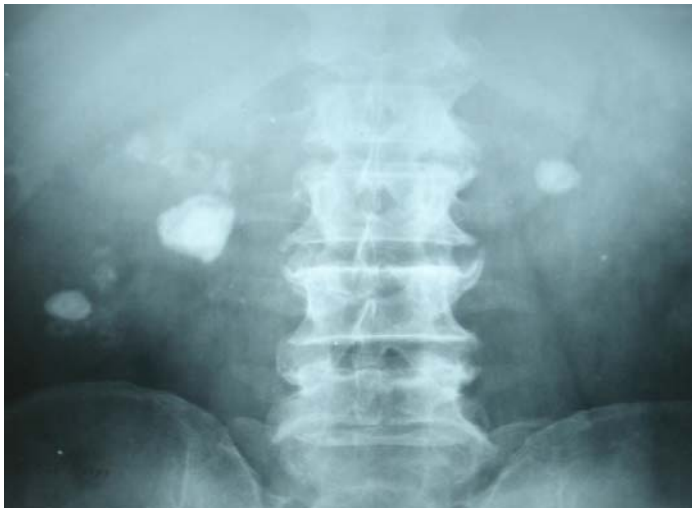
BILATERAL STAGHORN CALCULUS WITH LEFT URETERIC CALCULUS



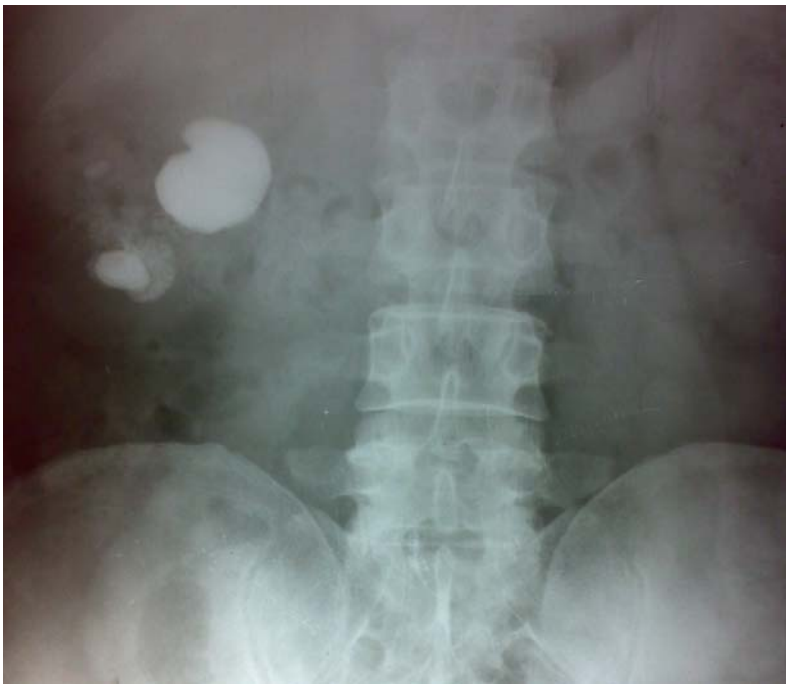
LEFT PELVIC CALCULUS



BILATERAL RENAL PELVIC CALCULUS



RIGHT RENAL CALCULUS



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