STUDY ON FACTORS INFLUENCING THE COURSE AND OUTCOME OF INFLAMMATORY LESIONS OF KIDNEY IN ADULTS .

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CERTIFICATE

This is to certify that this dissertation entitled **"STUDY ON FACTORS INFLUENCING THE CLINICAL COURSE AND OUTCOME OF INFLAMMATORY LESIONS OF KIDNEY IN ADULTS"** submitted by **Dr. A. LARIF** appearing for **M.Ch (Urology)** degree examination in August 2013 is a original bonafide record of work done by him under direct supervision and guidance in partial fulfillment of requirement of the Tamil Nadu Dr.M.G.R. Medical University, Chennai.

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ABBREVIATIONS USED

APN - Acute Pyelonephritis

CT - Computed Tomography

DM - Diabetes Mellitus

EPN - Emphysematous Pyelonephritis

PCN - Percutaneous Nephrostomy

PUJ - Pelvi Ureteric Junction

USG - Ultrasonogram

UTI - Urinary Tract Infection

VUR - Vesico Ureteric Reflux

WBC - White Blood Cell

XGP - Xanthogranulomatous Pyelonephritis

VUJ - Vesico Ureteric Junction

INTRODUCTION

Inflammation of renal parenchyma and collecting system are usually secondary to microbial infections . Interstitial renal inflammation caused predominantly by bacterial infection, is nowadays recognized as a non specific histopathological change that can occur due to various immunologic, congenital, or toxin induced lesions that develop in the absence of bacterial infection.

Urinary Tract Infection (UTI) is considered as an inflammatory response of the urothelium to microbial invasion . UTIs exist as one of the most prevalent microbial diseases in the society with a substantial financial burden .

Spectrum of infectious disease process in the kidney presenting as acute inflammatory lesion commonly encountered in urological practice include

Acute Pyelonephritis

Focal and multifocal bacterial nephritis

Renal cortical abscess , Perinephric abscess

Emphysematous Pyelonephritis

Pyonephrosis

Course of the renal inflammation depends on the pathogenic virulence factors , predisposing comorbid illness and conditions in the host , severity of the infectious process , the time of presentation, and the effectiveness of therapy instituted**.**

Urosepsis occurring as a result of severe urinary tract infection is a serious condition that can ultimately lead to septic shock and death. High degree of suspicion on possible occurrence of urosepsis in all cases of presumed acute pyelonephritis is the single most essential factor in making an early diagnosis of complicated renal infection and preventing sepsis .

Definitive guidelines for optimal management of acute renal inflammatory lesions have yet to be established. The best treatment strategy must be the one that improves the patient survival , relieves the distressing symptoms and at the same time maximizes renal salvage. Minimal invasive interventions have been advocated nowadays so as to conserve the renal unit . Unfortunately, these minimal invasive management strategies have variable treatment outcome and can be predicted to little extent by the presence of certain risk factors in the individual

This study is intended to analyse the various factors that can influence the course and alter the outcome of the inflammatory pathologies of the kidney.

Continuous active monitoring is required in patients with these risk factors which might help in taking timely decision either to preserve the kidney or perform nephrectomy if they fail to respond to the initial conservative management. Nephrectomy contributes often to a complete resolution of life threatening renal infection in these situations .

AIMS OF THE STUDY

Aims & Objectives :

1. To analyse the risk factors predisposing the acute inflammatory lesions in kidney .

2. To evaluate the factors determining their clinical course – Recovery from disease / Nephrectomy

3. To discuss the management options and factors influencing their success.

REVIEW OF LITERATURE

Urinary tract infections (UTIs) are among the most common conditions requiring medical attention . It results from invasion of the urothelium by bacteria that terminate in an inflammatory response.

Interactions between the uropathogen and host result in UTIs and the pathogenesis often involves several processes. The uropathogen initially attaches to the epithelial surface where it colonises and disseminates throughout the mucosa causing tissue damage. Pathogens can then ascend in to the urinary bladder resulting either in symptomatic infection or asymptomatic bacteriuria. Disease progression further may lead to pyelonephritis and renal impairment.

Virulence factors of the uropathogens are responsible for bacterial resistance to the normally effective defence mechanisms of the host.

Depending on the anatomic or functional status of the urinary tract and the health of the affected host, UTIs can be divided in to

Uncomplicated UTI and Complicated UTI

Uncomplicated UTI :

Describes an infection in a healthy patient having a structurally and functionally normal urinary tract [1]. The virulence properties of the causative bacteria serve as the predominant factor leading to the infection. Majority are women presenting with isolated or recurrent bacterial cystitis or acute pyelonephritis. The infecting pathogens are usually susceptible and eliminated by a short course of commonly used oral antimicrobial therapy.

Complicated UTI :

Describes an infection occurring in a patient with an anatomically abnormal urinary tract or significant medical or surgical comorbidities. The host immune deficiency in its various forms serve as the major cause . Structurally or functionally abnormal urinary tract, compromised host , increased virulence or antimicrobial resistance of bacteria contribute. Majority are men. Removal of the complicating factor in addition to the high doses of antibiotics is essential for successful treatment .

A complicated infection is usually associated with factors that increase the chance of acquiring microbes and decrease the efficacy of therapy (Table 1).

TABLE 1 : Risk Factors For Complicated UTI

Risk factors for complicated urinary tract infection	
• Male sex	• Urinary tract instrumentation
• Old age	• Pregnancy
• Febrile urinary tract infection	• Diabetes
• Symptoms for > 7 days	• Immunosuppression
• Haematuria	• Infection with drug resistant organism
• History of stone disease	• Functional or structural abnormality
• Recent hospitalisation	

Both pathogenic (organism related) and host factors determine the severity of the infectious process and thus influence the course and outcome of the inflammatory lesions of the kidney.

Host factors: Patients at risk for complicated UTI

A. Intubated urinary tract:

Patients with an intubated urinary tract, with an indwelling catheter (urethral, suprapubic, Nephrostomy, or others) or an internal one, such as a ureteral or urethral stents are at risk for complicated UTI . Reasons being

i) These patients are always at increased risk for infection and due to subsequent exposure to multiple courses of antibiotics ; they are prone to infections with resistant organisms.

ii) Urinary findings of infection are indistinguishable from those with sterile urine, from the perspective of the urinary chemistry and microscopy. In the presence of an indwelling catheter ,the urine will become colonized, if not infected by the 2-week time period. The usual organisms tend to be

uropathogens, but occasionally skin flora or vaginal organisms predominate. Often it is difficult to distinguish colonization from frank infection in the setting of an indwelling catheter.

B. Urinary obstruction

The patient with a known urinary obstruction, whether it involves the upper or lower urinary tract, have high propensity to develop complicated UTI. [2] Obstruction cause prolongation of bacteruria by mechanical means and also interferes with the local and systemic immune response, preventing its functioning at optimal levels. Obstruction prevents the normal flow of urine, and the resulting urinary stasis compromise renal and bladder defence mechanisms. Bacterial growth in urine and their ability to adhere to the urothelial cells is increased following stasis .

The infectious process itself increases the degree of obstruction. This is accomplished by bacterial alterations of the environment in the course of the infection, resulting in swelling and prevention of peristalsis .[12] The presence of obstruction causes the kidney to have a reduced ability to excrete and concentrate antibiotics as well, and also reduce the ability of the drug to function because of the extended presence of bacterial products in the environment.

In the setting of obstruction, an infection is usually viewed as an undrained abscess which depend upon the degree of obstruction and also the level of obstruction . In most cases, drainage is indicated, usually on an

urgent or even emergent basis. Even mild episodes of pyelonephritis or cystitis in situations where obstruction to urine flow is present can become life threatening . It becomes essential to relieve the obstruction in these situations for a successful outcome.

C. Male Gender

Urinary tract infections are more prevalent in women because of their short urethral length which allows easy bacterial colonisation of the bladder. At least 20% of women experience a UTI in their life time.

Most clinicians suggest that male gender alone can be a criteria for being a complicated UTI . [1,3] This is especially true in the older individual who are likely to have benign prostatic hyperplasia, and thus presumably a lower urinary tract obstruction. [4] Males have several advantages over females when it comes to urinary tract infection susceptibility; an infection in male should raise suspicion of an underlying condition. The presence of an elongated urethra and the antibacterial nature of prostatic secretions combine to reduce the incidence of UTIs in male as a whole. [5] Thus, infection in a male suggests underlying abnormalities predisposing to complicated infection.

D. Age

Age is always a consideration when considering the existence of a complicated UTI. Old bed ridden patients with catheter poses greater risk. Infections in childhood gives clue to certain abnormalities of the genitourinary tract. [6,7] Vesicoureteral reflux, urethral valves, ureteropelvic obstruction, or ureterovesical obstruction are some of the relatively common conditions where there is increased incidence of urinary tract infections, as well as increased severity. Gender here is important as well, as most of the congenital genitourinary abnormalities are commoner in boys than girls.

E. Diabetes mellitus

A number of medical conditions have found to be risk factors for UTIs, and they lead to a more protracted course of therapy with potentially adverse outcomes because of specific complications. Most important among them is Diabetes mellitus (DM). Not only are these patients more susceptible to UTIs in general, complications are observed far more often than the normal individuals.[8] Perinephric abscesses are rarely seen in patients without obstruction, except in the setting of diabetes, particularly when there is poor glucose control. This is also true for lobar nephronia, intrarenal abscesses, or carbuncles. One condition that is almost exclusive to diabetics is **emphysematous pyelonephritis.**[9] This specific

case of renal infection is manifested by the presence of gas in the renal parenchyma. The condition has a mortality rate that exceeds 40% and often results in loss of the affected kidney.

Secondary obstruction may also be seen in DM because of the sloughing of papillae. This condition called as **renal papillary necrosis** results in obstruction of the upper urinary tract by mechanical means. The pathophysiology is a result of pyelonephritis in a patient with compromised intrarenal bloodflow like diabetics. The condition presents as an emergency requiring immediate drainage by either a retrograde or antegrade technique.

While women with diabetes mellitus have high occurrence of clinical asymptomatic and symptomatic UTIs , there is no increase incidence among diabetic men. Hospitalizations for acute pyelonephritis in diabetics are more common among women (10.86/10,000) than for men (3.32/10,000).

Upper urinary tract is involved in nearly 80% of diabetic patients as evident by localization studies (Forland et al, 1977). Infections in diabetics are commonly caused by atypical organisms such as yeast and can result in upper tract infections with significant complications such as papillary necrosis, emphysematous pyelonephritis, renal abscess or perinephric abscess.

F. Renal insufficiency

Patients with renal insufficiency are another high-risk group known to develop complicated UTIs. The most common reason being reduction in renal blood flow that most often accompanies this disease. The secondary effects include a reduced immune response, both on the local and systemic level . Reduced urinary volume and impaired host defences increase the bacterial capability to colonize the urinary tract. The delivery of antibiotics is compromised, making eradication of infection difficult . Lastly, patients on both hemodialysis and peritoneal dialysis have an increased incidence of UTIs, and some are secondary to infections elsewhere .[26]

G. Immunosuppression

Patients who are immunosuppressed, either by medications or by other comorbid conditions, must always be treated aggressively. [10] The secondary effect of the immunosuppressant drugs is the increased susceptibity for infections throughout the body, including the urinary tract. The prototype of these is corticosteroids. These drugs irrespective of their route of administration cause immunosuppression in a dose dependent fashion by reducing the cell-mediated immune responses and local immune reaction. There is also an effect on the humoral immune system, although this is not as pronounced.

Drugs such as the calcineurine inhibitors (cyclosporine and tacrolimus) are cell-mediated immunity specific. Monoclonal antibodies such as muromonab-CD3 and a host of others which are currently being used to treat transplant rejection have profound effects on the treatment of UTI. The cell-cycle nonspecific drugs, such as azathioprine and mycophenolate mofetil, are also a concern, as they are not only used in treatment of organ transplantation, but also in other diseases.

H. Urolithiasis

Urolithiasisis certainly one of the medical conditions that confers the designation of ‘‘complicated’’.[11] The most ominous complication of stones is obstruction. The association of an obstructing calculus along with febrile UTI is usually considered an emergency, because of the risk of sepsis. Intervention is mandatory in most cases, specifically by employing either a nephrostomy tube or a ureteral stent.

Stones often prolong the treatment of UTIs if they are infected. The bacteria become concealed in the interstices of the stone and form their own biofilm environment which make infection difficult to eradicate without removal of the calculus. Stones also cause injury to the urinary tract, and this damage can give bacteria a place to establish colonization.

Certain stones form as a direct result of infections, specifically magnesium ammonium phosphate or calcium carbonate stones. Urea splitting microorganisms, by altering their microenvironment, facilitate precipitation of these salts, promoting de novo stone formation and thus persistence of infection. If the bacteria come in contact with other types of stones , they may lay down new matrix upon the existing stone, creating an eclectic mix of calculus material.

I. Surgery of the Urinary tract

Whenever surgery of the urinary tract performed, either for obstruction, calculus, or other causes, the urinary tract becomes more susceptible to UTIs and persistence or recurrence of infection occurs . Disruptions or irregularities of the urothelium may serve as an initiation point for UTIs or the creation of obstruction. The underlying abnormality may not have been entirely corrected which may serve as a risk factor for infection . Sutures utilised in the surgery may persist in the urinary tract, even when the material is designed to dissolve. If sutures remain long enough, colonization may occur, and stones may occur.

J. Functional and Anatomical abnormalities of the Urinary tract

There are a number of urinary tract abnormalities which can be either anatomical or functional that may lead to complicated UTIs.

Most of these are congenital and present early in life with voiding dysfunction . Vesico-ureteral reflux, ureteropelvic junction obstruction, urethral valves (anterior or posterior), and congenital megaureter are some of the most common abnormalities which are associated with an increased incidence of UTIs, as well as a need for prolonged antibiotic therapy or intervention. Correction of these abnormalities may reduce the risk of UTIs, but their occurrences in the setting of an infection implies a complicated label .[12] Structural abnormalities like polycystic kidney disease, calyceal diverticula, medullary sponge kidney, renal artery stenosis, renal vein varices or thrombosis, and many others lead on to complicated infection. Although some of these may be corrected or near-corrected with surgery and medications, the risk for a complicated UTI remains greater than among the general population, and should be managed accordingly.

K. Pregnancy

Pregnancy always confers complicated status on any UTI .[13,14] Complicated infection lead to increased risk to both the mother and fetus. The gravid uterus, depending on the trimester, will cause an anatomic alteration that involves a relative obstructive uropathy. Drainage is not needed usually , but must be considered in high grade obstruction to relieve symptoms . After clearance of the UTI, decision regarding prophylactic antibiotics is warranted.

The hormone status also has a significant bearing on the infection management .A reduction in smooth muscle contractility, not only of the uterus, but also of the ureter and bladder, may confer a degree of obstructive uropathy.It has been shown that progesterone levels correlate closely with reduced ureteral motility and may both predispose to UTIs and prolong their treatment. The voiding dysfunction manifesting as frequency and urgency, along with stress incontinence, that almost invariably accompanies a pregnancy also negatively impacts the resistance to and treatment of UTIs.

Increase in bladder pressure and associated venous congestion may further contribute to the increased susceptibility. These conditions disrupt the tight junctions in the bladder epithelium and the uromucoid layer (a protective layer of mucus, Tamm- Horsfall protein and other substances) and they fail to prevent infection.

Bacteriuria prevalence in the pregnant women varies from 4% to 7%, and the incidence of acute clinical pyelonephritis is about 25% to 35% if left untreated .

L. Voiding Dysfunction

Patients with voiding dysfunction, including those with neurogenic bladders, comprise a significant proportion of complicated UTIs. [15] Prime reasons are :

i) **Frequent presence of residual urine**

Conditions such as low spinal cord injury, myogenic atony,spina bifida, sacral agenesis and others may cause incomplete emptying and thus an enhanced milieu for bacterial growth. This can also be a result of bladder outlet obstruction, either anatomic or physiologic.

ii) **Presence of high pressure voiding and its effects**

The first is a reduction in blood supply, impairing immune response and host response. Second, the high pressures disrupt the uromucoid layer and may separate the urothelial cells, all causing a facilitation of bacterial binding and ultimately colonization. Lastly ,as the bladder damage progresses over time, cellules and diverticula may emerge,as does secondary vesicoureteral reflux. These lead to incomplete emptying, which greatly contributes to the sequestration of bacteria, establishment of individual microbial ecosystems, and reduced clearance by drugs, as well as renal insufficiency.

M. Environment – Nosocomial infections

Hospital acquired or nosocomial infections must be considered complicated because of a host of factors. [16] Indwelling catheters and other breaks in the body's integument provide for initiation points for the infection. The enormous amount of antibiotics used in this environment leads to emergence of resistant bacterial species . Another problem is cross contamination from patient to patient transmitted by the health care providers

themselves. Hospitalized patients contracting UTIs have mortality rate reaching 30% .[17]

Pathogenic factors: Organism related factors contributing to Complicated UTI.

The organisms implicated in causing complicated UTIs are varied (Table 2)

TABLE 2: Common Bacterial Pathogens

Common urinary bacterial pathogens	
• *Escherichia coli*	• *Klebsiella* spp
• *Staphylococcus saprophyticus*	• *Providencia* spp
• *Streptococcus faecalis*	• *Citrobacter* spp
• *Proteus* spp	• *Serratia* spp
• *Pseudomonas* spp	• *Enterococcus faecalis*

I. Gram negative Organisms :

The initial infection typically is caused by Escherichia coli. It is the most common facultative aerobic organism in the gastrointestinal tract and is in close proximity to the genitourinary tract. E. coli has a number of survival advantages to establish a colony, and ultimately an infection, over other Gram-negative species. Many subtypes of E.coli are possessing surface structures that bind to specific locations on the urothelial cell surface. Most important of these are fimbria which includes

a) **Type I fimbriae**, which are inhibited by mannose and avidly bind to latex catheters and urothelial cells [18].

b) **P-fimbriae,**which bind to a urothelial cell surface receptor referred to as the P-blood group antigen present in the majority of the world population and located on the urothelial cells as well [18]. Those with the P-blood group antigen are able to ascend the urinary tract easily, even in the anatomically normal system. P fimbriae is implicated in E. coli pyelonephritis. Epidemiologic studies have consistently demonstrated that these adhesions are present in almost 100% of strains causing pyelonephritis .[19]

Many other Gram-negative species may also be found in the complicated UTI group. Most of these are nosocomially acquired . In the setting of an indwelling catheter multiple species may colonize the catheter, and thus the urine. Not all species are capable of causing a true infection, but they may complicate the circumstances in other ways which include

a) Sharing of genetic material, both chromosomal or naked DNA, often referred to as **‘‘plasmids.’’** which may readily spread throughout a bacterial population by conjugation and other means, result in multiple antibiotic resistant infections [20].

b) Certain bacterial species appear to have a great survival advantage due to presence of matrix or glycocalyx synthesis (also referred to as **biofilm**) [21,,22].

Certain bacteria, the prototype being Pseudomonas aeruginosa, are able to synthesize a substance into their micro environment, which forms a protective shell around themselves which protect them from the body's immune response as well as cause exclusion of antibiotics .

II. Gram Positive Organisms

Other than Staphylococcus saprophyticus, and rarely group D streptococci (enterococci), infections caused by Gram-positive species are more commonly seen in complicated UTI groups. The reasons being

i) Most species lack the external appendages to bind avidly to the urothelial cell surface, and thus have a lower probability of establishing a colony to initiate the infectious process.

ii) These species are not as ubiquitous in the area in and around the urethral opening nor seen in the colon or mucosal surfaces.

iii) They often lack specific virulence factors to allow them establish a colony and invade.

Gram-positive organisms may spread via the hematogenous route and tend to be a result of a significant insult to the body's defense mechanisms. An example of this is Staphylococcus aureus infection, which often can come from an infected site elsewhere in the body, causing bacteremia and therefore seeding of the kidney [23]. This usually occur in the

circumstance of an intravenous drug abuser or someone with infected indwelling line. These infections cause complicated UTIs, specifically lobar nephronia (renal carbuncle) or perinephric abscess, requiring long-term antibiotics or intervention ,either endoscopic or surgical.

III . Fungal infections

Another group of pathogens that are included in the complicated group are the fungi. The most among them is the genus Candida, with the species albicans, tropicalis ,and kruzei predominating. These yeast forms are frequently found in the urinary tract as a colonizing agent, rather than an infecting organism[24] and acquire greater significance in the setting of an intubated urinary tract.

Candida accounts for less than 5% of complicated UTIs. Other fungi comprise a very tiny percentage, with isolated case reports of prostatitis, pyelonephritis, and other infections caused by fungi other than candida.

SPECTRUM OF ACUTE INFLAMMATORY RENAL LESIONS

I. Acute Pyelonephritis (APN)

Traditionally defined as inflammation of the kidney and renal pelvis, but the diagnosis is mostly clinical. Many patients with APN present with mild symptoms and can be managed in as outpatient.[25] About 10% to 30% of patients with APN require hospitalisation [26] and

may even present with life-threatening complications including shock, septicaemia and multi-organ dysfunction syndromes. The mortality rates ranges from 1% to 12%.[27, 28]

Clinical Presentation :

Patients have acute onset of fever (100° F or greater), chills , unilateral or bilateral flank or costo vertebral angle pain and / or tenderness. Lower tract symptoms like dysuria, increased urinary frequency, and urgency may be present . Hill [29] defined APN pathologically as a suppurative inflammation of the renal parenchyma and pyelocaliceal system typically distributed along one or more medullary rays supporting an ascending route of infection. There is often poor correlation between the clinical symptoms and the site of infection . Previous history of lower UTIs is present in approximately 75% cases. There is costovertebral angle tenderness during deep palpation on physical examination .

Acute pyelonephritis may have asymptomatic progression to chronic pyelonephritis, especially in compromised hosts, without any symptoms. Acute renal failure may be the presentation in rare cases .

Laboratory Diagnosis:

Numerous WBCs, often in clumps, and bacterial rods or chains of cocci are seen in urine analysis . Leukocytes showing Brownian motion in the cytoplasm (glitter cells) are seen in hypotonic

urine , but are not diagnostic of pyelonephritis. Granular or leukocyte casts are suggestive of acute pyelonephritis and these are readily demonstrated by staining urinary sediment with basic dyes like dilute toluidine blue or KOVA stain.

Blood tests show leukocytosis with a predominance of neutrophils, increased erythrocyte sedimentation rate, elevated C-reactive protein levels, and elevated creatinine levels if renal failure is present. Creatinine clearance may be decreased. Blood cultures may be positive.

Bacteriology:

Urine cultures are usually positive, but are negative in 20% of patients due to lesser colony count . E. coli, accounts for 80% of the cases. Multiple antimicrobial resistant clonal groups are seen in community –acquired pyelonephritis . More resistant species like Proteus, Pseudomonas, Klebsiella , Enterobacter, Serratia or Citrobacter should be suspected in patients presenting with recurrent UTIs. Except for E. faecalis, S. epidermidis, and S. aureus, gram-positive bacteria rarely cause pyelonephritis.

Blood cultures are positive in about 25% of cases and most resemble the urine culture . They do not influence decisions regarding therapy. Blood cultures should therefore routinely not obtained for the evaluation of uncomplicated pyelonephritis but performed only in those with systemic toxicity or those requiring

hospitalization or with demonstrable risk factors such as pregnancy (Velasco et al, 2003).

Imaging Studies:

Ultrasonography and Computed Tomography are utilized to evaluate patients initially for complicated UTIs and are useful in re-evaluation of patients who are not responding after 72 hours of therapy. Ultrasonography depict hypoechoic or attenuated parenchyma, renal enlargement and a compressed collecting system. Contrast computed tomography (CT) scans show hypoenhancing regions with or without renal swelling and can be focal or diffuse. To standardize terminologies, Talner et al [30] in 1994 suggested that all radiological parenchymal abnormalities without abscess attributable to acute infection be called APN. CT scan reveal disorganized parenchyma and abscess formation in the lalater stages with renal parenchymal damage.

Radionuclide imaging are useful to demonstrate functional changes associated with acute pyelonephritis (decrease in renal blood flow, delay in peak function, and delay in excretion of the radionuclide) and renal cortical defects associated with vesicoureteral reflux.

Management :

Patients with presumed uncomplicated pyelonephritis are managed as outpatients and radiologic evaluation are usually deferred. Single drug oral therapy with a fluoroquinolone is effective . Single

parenteral dose of an antimicrobial agent such as ceftriaxone, gentamicin, or a fluoroquinolone are administered by some before starting oral therapy. Amoxicillin or amoxicillin / clavulanic acid is recommended for gram-positive organisms .

If a patient show uncomplicated infection but is sufficiently ill (with evidence of sepsis) to require hospitalization or has complicated pyelonephritis, or fails to improve with outpatient therapy , a parenteral fluoroquinolone, an aminoglycoside with or without ampicillin, or broad spectrum cephalosporin with or without an aminoglycoside is administered .

In patients with known or suspected complicated pyelonephritis renal ultrasound evaluation rules out stones or obstruction. Excellent assessment of the urinary tract status , extent and severity of the infection is done by CT scan. These patients require hospitalization, with complete bed rest, intravenous fluids, parenteral antibiotics and antipyretics. An obstructed kidney have difficulty in concentrating and excreting antimicrobial agents and hence any significant obstruction has to be relieved expediently by the safest and simplest method .

Unfavorable Response to Therapy:

Immediate reevaluation is needed in these patients .Repeating the urine and blood cultures and making alterations in antimicrobial therapy

is necessary. CT scan is employed to identify unsuspected obstructive uropathy, urolithiasis , or underlying anatomic abnormalities which aid in prolonging the infection, preventing a rapid therapeutic response and producing complications such as renal or perinephric abscess.

Follow-Up:

Repeat urine cultures are done on the seventh day of therapy and 10 to 14 days after discontinuing antibiotics to ensure that the urinary tract remains free of infections. 10% to 30% cases relapse after a 14-day course of therapy.

A second 14-day course of antibiotics is necessary for relapse treatment but occasionally a 6-week course may be needed. Some patients may require additional investigations such as voiding cystourethrogram, cystoscopy or bacterial localization studies and correction of an underlying abnormality of the urinary tract.

II. Acute Focal or Multifocal Bacterial Nephritis

Acute focal or multifocal bacterial nephritis is an uncommon, more severe form of acute renal infection in which a heavy leukocyte infiltrate is confined to a single renal lobe (focal) or multiple lobes(multifocal).

Clinical Presentation:

Symptoms and signs are similar to that of acute pyelonephritis but are usually more severe. More than half of the patients are diabetic, and

sepsis is common . Leukocytosis and gram-negative urine culture are found . Blood culture is positive in more than 50% of cases.

Radiologic Findings:

The diagnosis is evident on radiologic examination. On ultrasonography lesion is relatively sonolucent and poorly marginated with occasional low-amplitude echoes that disrupt the cortical medullary junction . Enhanced CT scan studies is necessary because the lesion is difficult to visualize on the unenhanced study. Wedge shaped areas of decreased enhancement are characteristic. Definite wall is not evident and frank liquefaction is absent. It has to be differentiated from renal abscess. Abscesses are usually round with liquid centers and are present both before and after contrast medium enhancement. Chronic abscesses have a ring shaped area of increased enhancement surrounding the lesion (Corriere and Sandler, 1982).

Management:

Acute bacterial nephritis usually represents a relatively early phase of abscess formation and most cases progressed to frank abscess formation. Treatment includes hydration and intravenous antibiotics for at least 7 days, followed by 7 days of oral antimicrobial therapy. Patients rightly respond to medical therapy, and resolution of the wedge-shaped zones of diminished attenuation can be seen in followup imaging studies .

Failure to respond to therapy is an indication for imaging studies to rule out obstructive uropathy, renal or perirenal abscess, renal carcinoma, or acute renal vein thrombosis.

III. Emphysematous Pyelonephritis (EPN)

Urologic emergency characterized by an acute necrotizing parenchymal and perirenal infection caused by gas-forming uropathogens. Majority of the cases occur in diabetic patients. High tissue glucose levels serve as a substrate for microorganisms such as E. coli, which produce carbon dioxide by the fermentation of sugar. In addition to diabetes, most patients have urinary tract obstruction associated with urinary calculi or renal papillary necrosis and exhibit significant renal functional impairment. The overall mortality rate is reported to be between 19% to 43% in various studies.

Clinical Presentation:

All documented cases are adults and there is no increased risk in juvenile diabetics . Women are affected more often . The usual clinical presentation is severe acute pyelonephritis, but in some situations a chronic infection precedes the acute attack. Many patients present with the classic triad of fever, vomiting, and flank pain. Pneumaturia is absent unless the infection involves the collecting system.

Labarotory Investigations :

Urine cultures are usually positive with E. coli being the most commonly identified organism. Klebsiella and Proteus are less common.

Imaging Studies:

Plain X-ray shows tissue gas distributed in the renal parenchyma as mottled gas shadows over the involved kidney. A crescentic collection of gas over the upper pole of the kidney is more characteristic .Gas extends to the perinephric space and retroperitoneum as the infection progresses. USG usually demonstrates strong focal echoes suggesting the presence of intraparenchymal gas. CT Scan is the imaging procedure of choice in defining the extent of the emphysematous process and guiding management .

25% of cases demonstrate presence of obstruction evident by imaging . Radioisotope scan should be performed to assess the degree of renal function impairment in the involved kidney and the status of the contralateral kidney.

Management :

Emphysematous pyelonephritis is considered a surgical emergency. Most patients present with sepsis ; fluid resuscitation with broad spectrum antibiotics are essential. Medical therapy can be considered in a functioning kidney . Nephrectomy is recommended in

patients who fail to improve after a few days of therapy. If the affected kidney is non-functioning and not obstructed, nephrectomy should be performed since medical treatment alone in such situation is usually lethal. If a kidney is obstructed, prompt drainage must be instituted. If condition of the patient improves, nephrectomy may be deferred pending a complete urologic evaluation.

IV. Renal Abscess

Renal abscess or carbuncle is a collection of purulent material confined to the renal parenchyma .Previously 80% of cases were attributed to hematogenous seeding by staphylococci but since 1970, gram-negative infections are the major source of the cases.

Ascending infection along with tubular obstruction from previous calculi or infections appears to be the primary pathway .Two-thirds of gram-negative abscesses are associated with renal calculi or damaged kidneys. Hematogenous renal seeding by gram-negative organisms may occur but rare. Recent studies indicate that reflux is frequently associated with renal abscesses due to persisting infections.

Clinical Presentation:

Fever, chills, abdominal or flank pain and occasionally weight loss and malaise . Symptoms of cystitis may occur. Occasionally symptoms are vague and may delay the diagnosis .

Thorough history may reveal a gram positive source of infection before the onset of urinary tract symptoms. Multiple skin carbuncles and intravenous drug abuse introduce gram positive organisms in to the blood stream. Complicated UTIs associated with pregnancy , calculi , neurogenic bladder and diabetes mellitus predispose the patient to abscess formation.

Laboratory Diagnosis:

Marked leukocytosis present . Blood cultures are usually positive. Pyuria and bacteriuria not present unless the abscess has communication with the collecting system. Urine cultures shows no growth or a different microorganism in gram-positive infection while the same organism is isolated from the abscess in gram negative infection .

Imaging studies:

Ultrasonography show an echo-free or low-echo density space occupying lesion with increased transmission .The margins of an abscess are indistinguishable in the acute phase but become well defined in the later stages. Internal appearance of the abscesss may vary from a virtually solid lucent mass to one with increased numbers of low-level internal echoes.

CT Scan is the diagnostic procedure of choice and provides excellent delineation of the tissue . Initially, CT shows renal enlargement and focal, rounded areas of decreased attenuation , later thick fibrotic wall begins to form around the abscess. An echo free or slightly echogenic

mass due to the presence of necrotic debris is seen. Chronic abscess are characterized by the obliteration of adjacent tissue planes and thickening of Gerota fascia. Round or oval parenchymal mass of low attenuation with a surrounding inflammatory wall of slightly higher attenuation forms a ring

The **ring sign** is caused by the increased vascularity of the abscess wall. Radionuclide imaging with gallium or indium is sometimes useful .

Management :

Small abscess less than 3 cm in diameter are managed conservatively with the course of intravenous antibiotics and careful observation . In hematogenous dissemination where there is higher chance of penicillin-resistant Staphylococcus, a penicillinase resistant penicillin is prescribed . In case of Penicillin hypersensitivity, Vancomycin is utilised. Gram -negative pathogens are treated with intravenous third-generation cephalosporins, antipseudomonal penicillins, or aminoglycosides .

In larger abscess , Percutaneous or Open incision and drainage is the classical treatment . CT scan or USG guided needle aspiration is necessary to differentiate an abscess from a hypervascular tumor. Culture of the aspirated material done to direct appropriate antimicrobial therapy.

Patients are followed up with serial examinations with USG or CT scan until the abscess resolves. The common causes of deteriorating clinical course include misdiagnosis , uncontrolled infection with formation of perinephric abscess and infection with resistant organisms.

Abscesses 3 to 5 cm in diameter , smaller abscesses in immunocompromised hosts and abscesses that do not respond to antimicrobial therapy should be drained percutaneously. Surgical drainage, however, currently remains the procedure of choice for most renal abscesses greater than 5 cm in diameter.

V. Perinephric Abscess

Etiology:

Perinephric abscess usually results from

1. Rupture of an acute cortical abscess in to the perinephric space
2. Hematogenous seeding from sites of infection (1/3 rd of the cases)
3. Secondarily infected perirenal hematoma by the hematogenous route or by direct extension of the primary renal infection.

High risk patients include patients with pyonephrosis ; particularly associated with calculus, Diabetes mellitus (approximately one third of patients) and polycystic renal disease patients who are on hemodialysis (mechanism is not clear; may be due to limited bioavailability of drugs in cysts).

Perinephric infection ruptures through the Gerota fascia in to the pararenal space to form paranephric abscess. It can also result from infections of the bowel, pancreas, or pleural cavity.

Perinephric or psoas abscess can occur as a result of of bowel perforation, Crohn ‘s disease or spread of osteomyelitis from thoracolumbar spine. E. coli, Proteus, and S. aureus account for most infections.

Clinical Presentation :

The onset of symptoms is usually insidious. Symptoms are present for more than five days (only 10% have it in pyelonephritis) . Clinical feature is similar to that of pyelonephritis but more than one third of patients may be afebrile. Abdominal or flank mass can be felt in about half of the cases. Psoas abscess should be suspected if the patient has a limp and flexion and external rotation of the ipsilateral hip.

Laboratory Investigations :

There is marked leucocytosis. Elevated levels of serum creatinine, and pyuria in more than 75% of cases. Urine cultures are positive in only 37% of cases. Blood culture, particularly with multiple organisms, was often indicative of perinephric abscess (Positive in only 42% of cases). Therapy based on the results of urine and blood cultures often may be inadequate.

IMAGING:

CT Scan is valuable for demonstrating the primary abscess. The abscess may be confined to the perinephric space or extend in to the flank or psoas muscle. CT scan delineate the route of spread of infection in to the surrounding tissues and thus helpful in surgical drainage.

Ultrasonography shows anechoic mass displacing the kidney to an echogenic collection that tends to blend with normally echogenic fat within the Gerota fascia .

MANAGEMENT :

Primary treatment for perinephric abscess is drainage; successful treatment by antimicrobial agents alone are unusual . Image guided percutaneous aspiration and drainage of small perirenal collections is possible. They are usually contraindicated in large abscess cavities filled with thick, purulent fluid. Gram stain of the purulent material guide in appropriate antimicrobial therapy.

If the patient's condition is good, nephrectomy can sometimes be performed along with drainage of the perinephric abscess. In other situations it is best to drain the perinephric abscess first and correct the underlying problem or perform a nephrectomy when the patient's condition improves.

VI. Pyonephrosis

Infected hydronephrosis is bacterial infection in a hydronephrotic kidney. The term pyonephrosis refers to infected hydronephrosis being associated with suppurative destruction of the parenchyma of the kidney resulting in total or nearly total loss of renal function.Where infected hydronephrosis ends and pyonephrosis begins is difficult to determine clinically. Rapid diagnosis and treatment of pyonephrosis are important to avoid permanent loss of renal function and to prevent urosepsis.

Clinical Presentation:

Patient is usually severely ill, with presence of high fever, chills, flank pain, and tenderness. Sometimes a patient may have only an elevated temperature and a complaint of vague gastro intestinal discomfort. Past history of urinary tract calculi, infection, or surgery is common. Bacteriuria may be absent if the ureter is completely obstructed.

Radiologic Findings:

Ultrasonography in infected hydronephrosis demonstrates internal echoes within the dependent portion of a dilated pyelocalyceal system. Focal areas of decreased echogenicity seen within the hydronephrotic parenchyma suggests pyonephrosis. CT is

usually nonspecific but may reveal thickening of the renal pelvis, perirenal fat straining , and a striated nephrogram.

Management:

Once diagnosis of pyonephrosis is made, treatment is initiated with appropriate antimicrobial drugs and drainage of the infected pelvis. A ureteral stent can be passed to drain the kidney but thickened pus may clog the stent resulting in poor drainage .In such cases percutaneous nephrostomy tube should be placed (Camunez et al, 1989) When the patient becomes hemodynamically stable, other procedures are needed to identify and treat the source of the obstruction.

MATERIALS AND METHODS

1. Study Group : Patients who were admitted with features of acute renal inflammation at 1) Kilpauk Medical College Hospital and 2) Government Royapettah Hospitals , Chennai.

2. Study Design : Prospective clinical study

3. Period of Study: January 2012 - January 2013

4. Total number of cases : 30 patients

4. Data Analysed : Risk factors, Clinical manifestations,
Laboratory Investigations, Imaging Studies,
Therapeutic interventions , Final outcome , Follow up

Inclusion criteria :

- Patients presenting with acute onset Flank or Loin pain , Fever with chills, with or without lower urinary tract symptoms.
- Radiological evidence of Renal inflammatory pathology.

Exclusion criteria :

- Patients unwilling to undergo intervention as a part of therapy

Study Protocol

- **Diagnosis of renal inflammatory lesions was made by**

Complete History (which included enquiry on predisposing risk factors) , Clinical Examination and confirmed by imaging studies.

Image 1 : CT Scan of Patient Number 2 with Left EPN

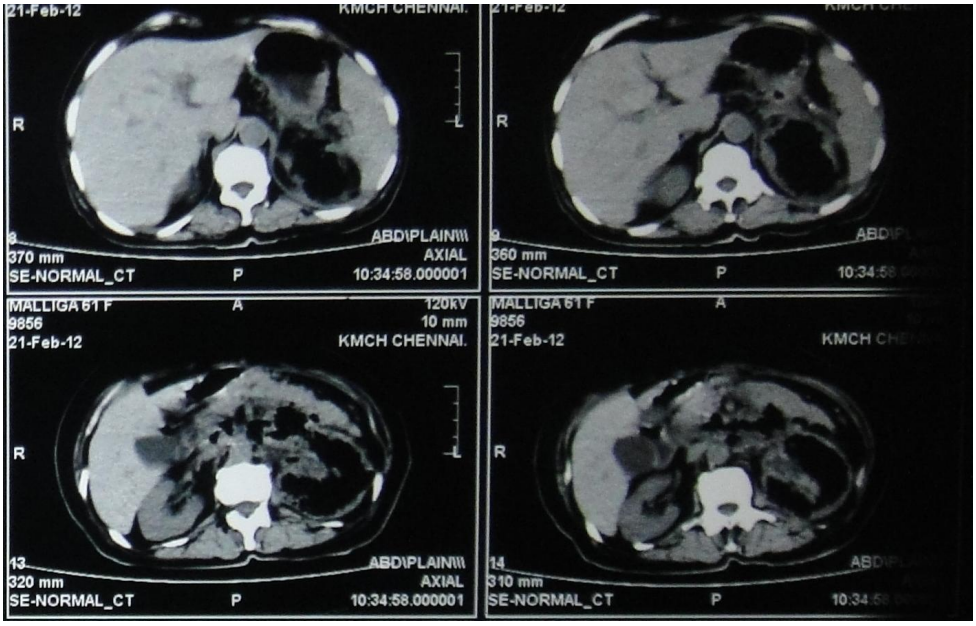


Image 2 : Isotope Study of Patient Number 2 with Left EPN

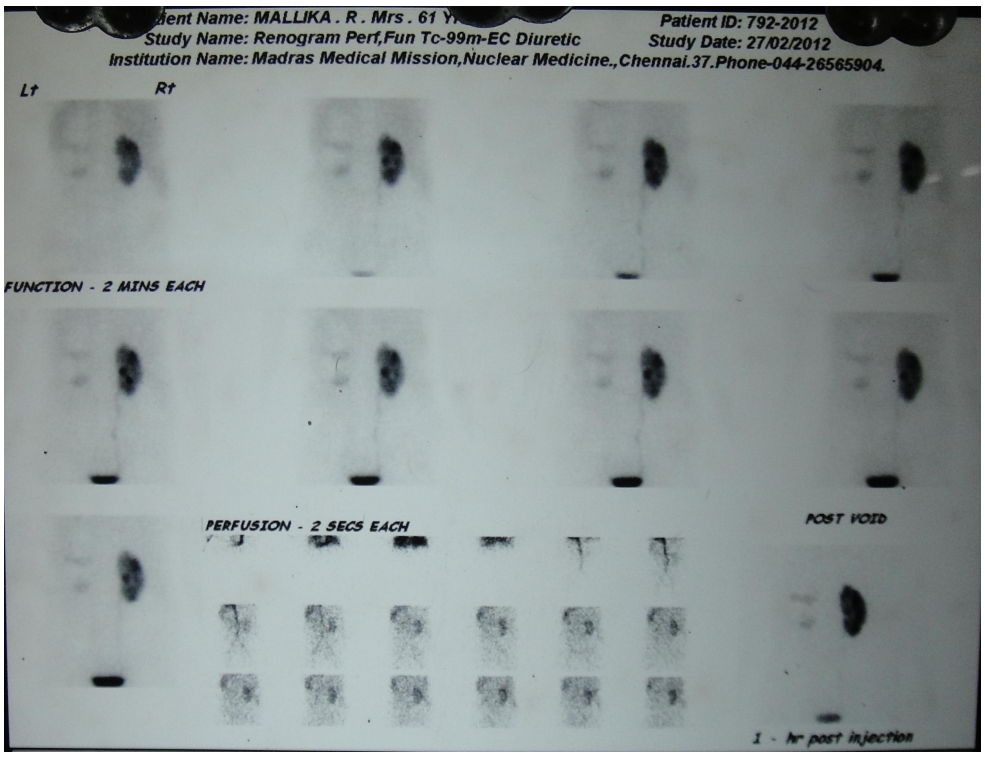


Image 3 : CT Scan of Patient Number 3 with Right Cortical Abscess

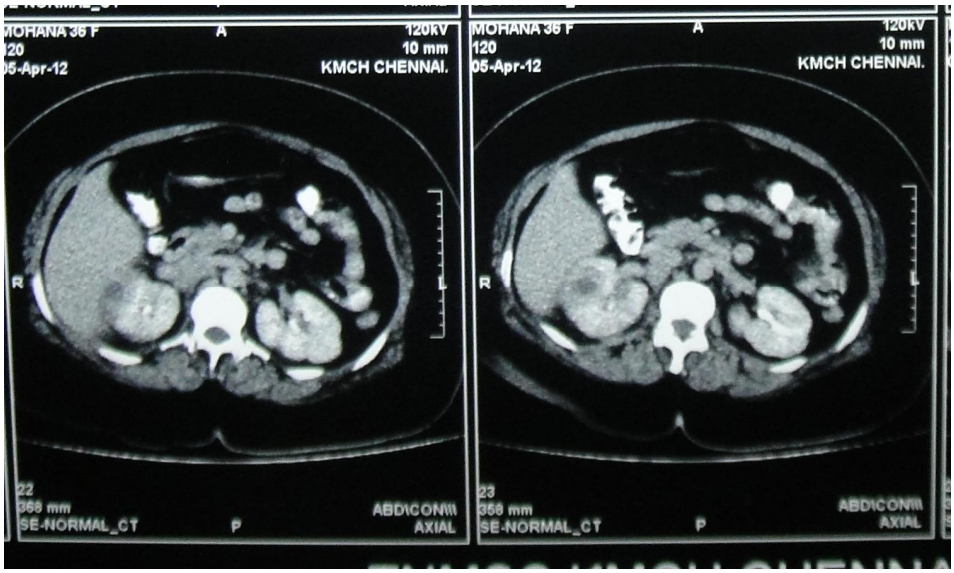
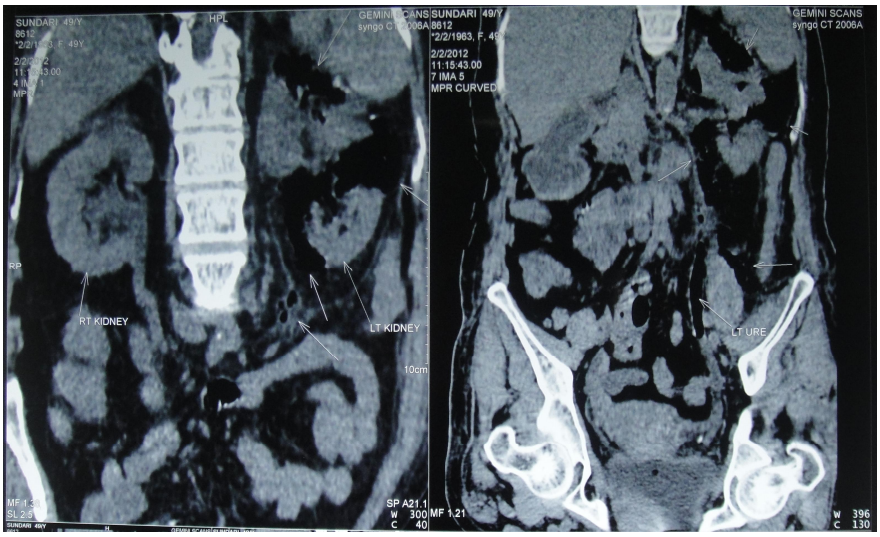


Image 4 : CT Scan of Patient Number 4 with Bilateral EPN



- **Patients were admitted and evaluated with** Urine analysis , Urine and Blood culture study, Complete Hemogram,Blood sugar and HbA1c (In Diabetics) , Renal Function Tests, Liver Function Tests , Blood coagulation profile , relevant additional imaging studies .

Image 5 : CT Scan of Patient Number 5 showing Left Pyonephrosis

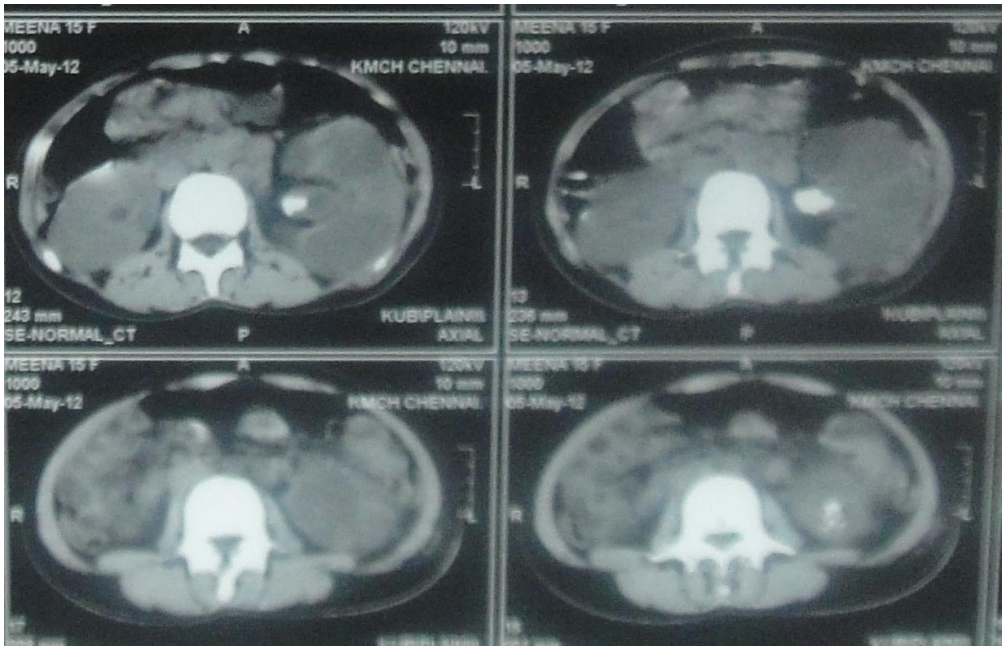
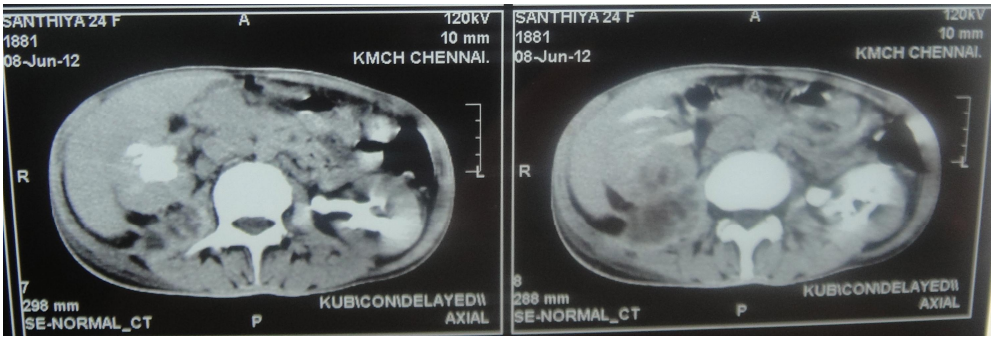


Image 6 : Patient Number 5 with Pyonephrosis Undergoing PCN

- **Managed with**

Hemodynamic Support , Fluid and electrolyte management , aggressive sugar control in diabetics , and antibiotics ; initially with broad spectrum parenteral (Injn. Cefotaxime 1gm twice daily) then changed according to culture & sensitivity report.

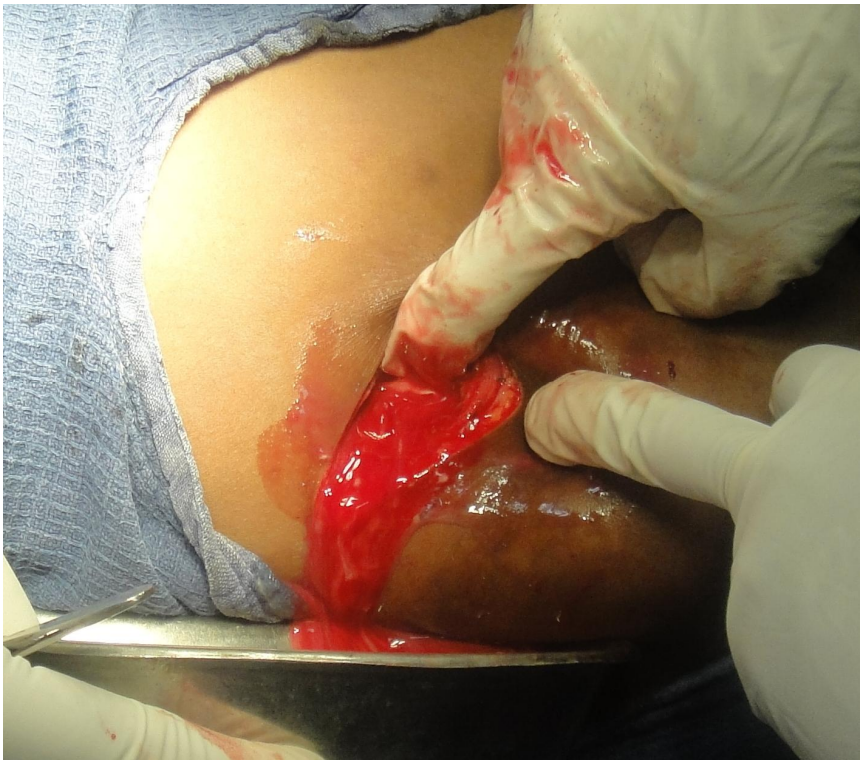
Image 7 : CT Scan of Patient Number 14 with Perinephric abscess



All patients were initially subjected to minimal invasive interventions like Percutaneous Nephrostomy (PCN) drainage under ultrasonogram guidance , Placement of Double J (DJ) Stenting, Both percutaneous nephrostomy and DJ stenting , Image guided percutaneous abscess drainage or Open Abscess drainage depending on the type of lesion diagnosed with the intention to conserve the renal unit.

The procedures were carried out after obtaining informed consent , explaining the pros and cons of the interventions and the need for strict followup to the patient and relatives .

Image 8 : Drainage of Perinephric abscess in Patient Number 14



Patients who did not recover with the medical and minimal invasive interventions were offered additional investigations like percutaneous nephrostomy fluid analysis , follow up CT scan , radioisotope scan and those who showed clinical deterioration , increasing renal parameters and non functional status in isotope scan (less than 15% split renal function) were subjected to Nephrectomy after obtaining cardiac , anaesthetic fitness and correcting any coagulation disorders if present.

Definitive Treatment of Complicating Factors were also carried out in the patients where the kidney was salvaged.

- **Follow up**

Patient were followed with 1 month and 3 months after discharge. Symptom analysis , Clinical Examination, Urine Culture & Sensitivity Renal Function test were done during their visit and those patients who showed recovery were included in the study.

- **Data collection and Statistical analysis**

Patients' demographics, medical histories, clinical parameters, biochemical and laboratory variables, types of renal lesions, the relevant therapeutic interventions and condition at discharge were analysed and compared among two groups :

Group I -- Recovered with Minimal Invasive interventions (16 pts)

Group II -- Required Nephrectomy (14 pts)

Statistical analysis was performed with **Statistical Package for the Social Sciences (SPSS) Version 15** software using Pearson's Chi-Square Test and Fisher's Exact Test with $P < 0.05$ considered to indicate statistical significance.

OBSERVATIONS AND RESULTS

Total number of Patients : 30

TABLE 1 : AGE STATISTICS

	Minimum	Maximum	Mean	Std. Deviation
Age in years	15	67	46.67	14.35

TABLE 2 : AGE WISE DISTRIBUTION

Age Group	Frequency
≤ 30 Years	6
31 – 50 Years	14
≥ 50 Years	10

CHART 1: AGE WISE DISTRIBUTION

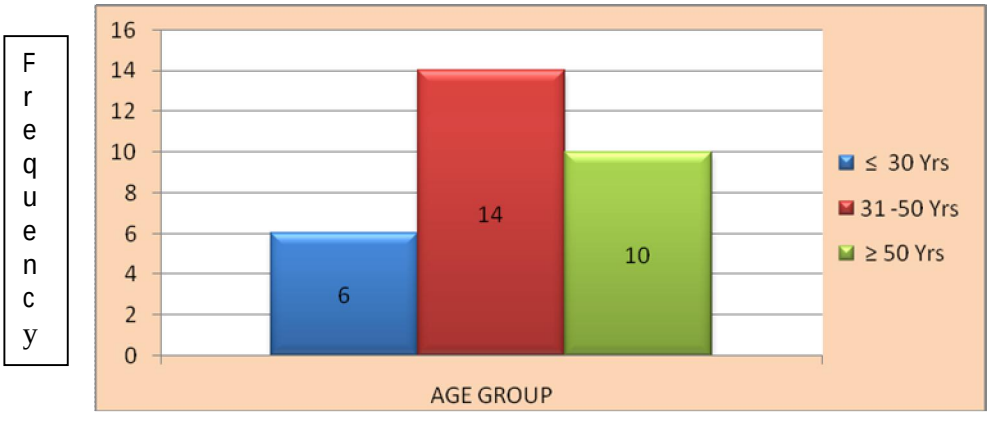


TABLE 2 : GENDER DISTRIBUTION

	Number	Percentage
FEMALE	20	66.7%
MALE	9	30.0%
TRANS GENDER	1	3.3%
Total	30	100.0%

CHART 2 : GENDER DISTRIBUTION

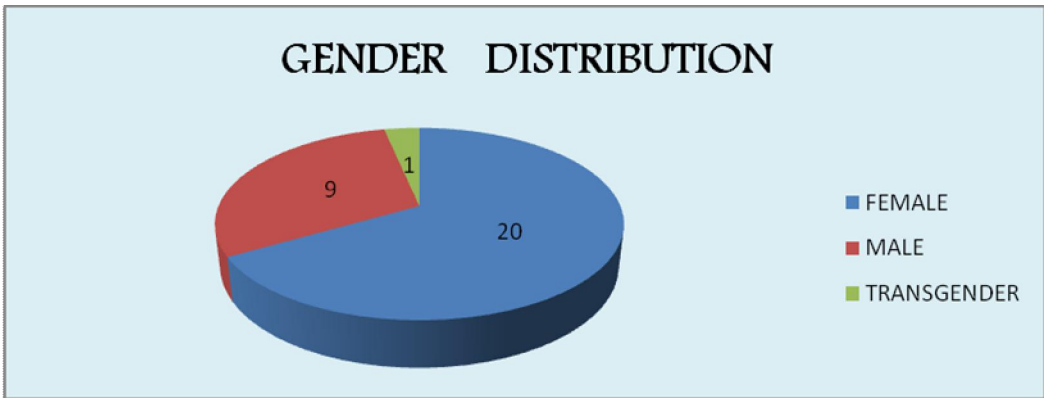


TABLE 3 : LATERALITY OF LESION

	Number	Percentage
BILATERAL	1	3.3 %
LEFT	16	53.3 %
RIGHT	13	43.3 %
Total	30	100.0 %

CHART 3 : LATERALITY DISTRIBUTION

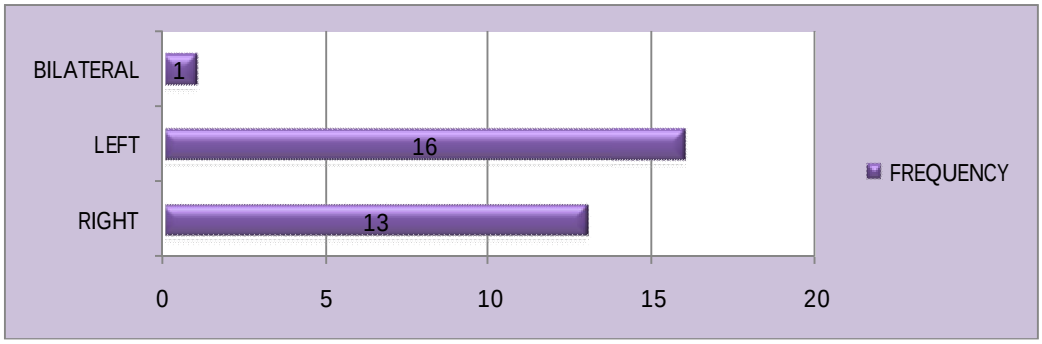
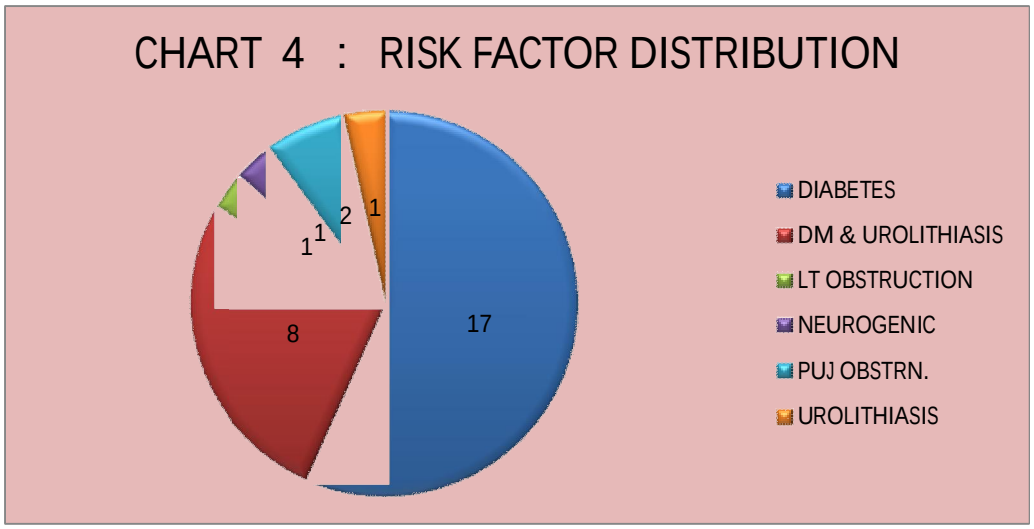


TABLE 4 : PREDISPOSING RISK FACTORS

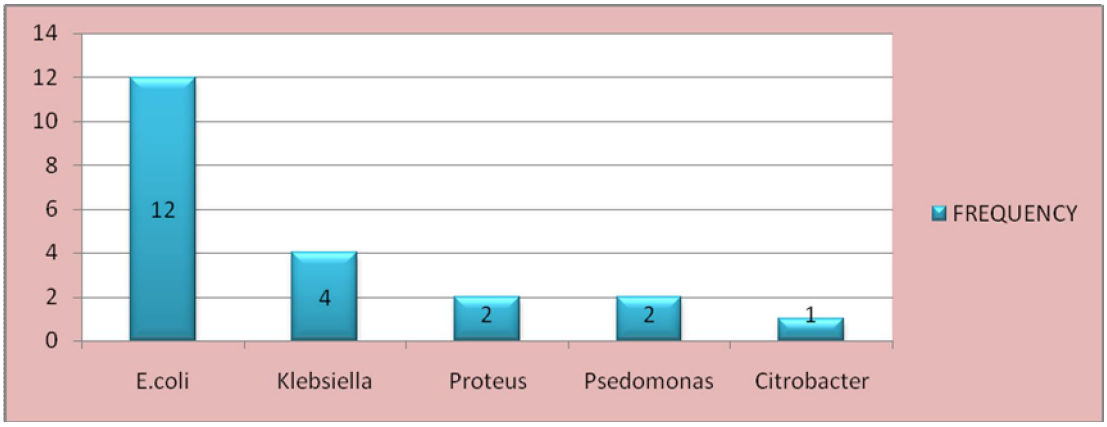
	FREQUENCY	PERCENTAGE
DIABETES MELLITUS	17	56.7 %
DM & UROLITHIASIS	8	26.7 %
LOWER TRACT OBSTRUCTION	1	3.3 %
NEUROGENIC DYSFUNCTION	1	3.3 %
PUJ OBSTRUCTION	2	6.7 %
UROLITHIASIS	1	3.3 %
Total	30	100.0 %



POSITIVE URINE CULTURE

Total Number : 21 (70%)

CHART 5 : DISTRIBUTION OF ORGANISMS IN URINE CULTURE



POSITIVE BLOOD CULTURE

Total Number : 9 (30 %)

CHART 6 : DISTRIBUTION OF ORGANISMS IN BLOOD CULTURE

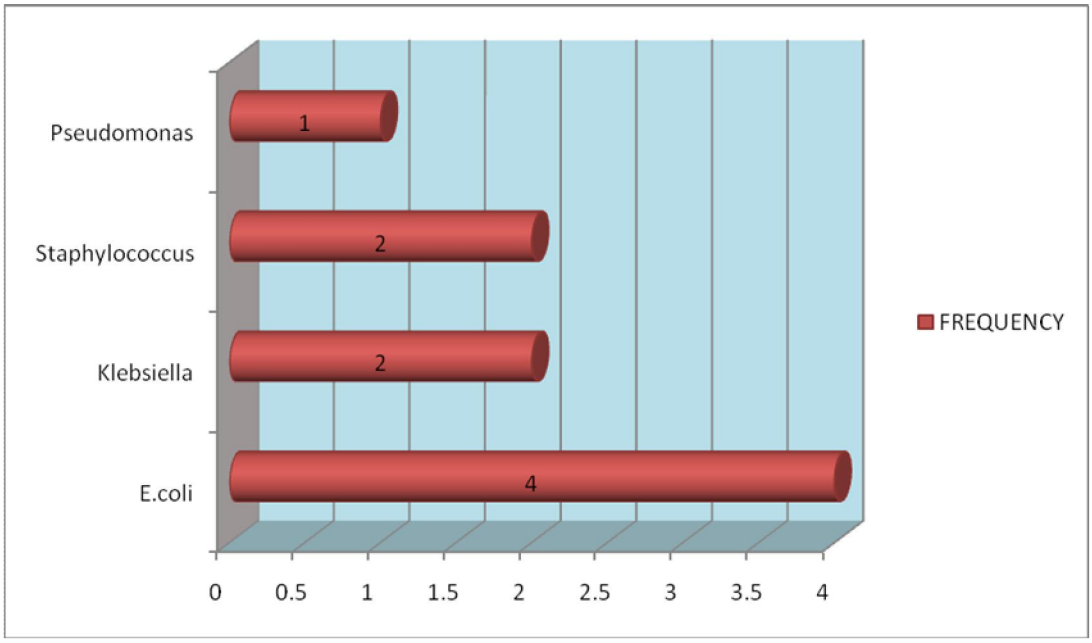


TABLE 5 : TYPE OF LESION AT PRESENTATION
(Based on Imaging Studies)

	Frequency	Percentage
BILATERAL . EPN	1	3.3 %
EPN	14	46.7 %
PERINEPHRIC ABSCESS	2	6.7 %
PYONEPHROSIS	10	33.3 %
RENAL ABSCESS	3	10.0 %
Total	30	100.0 %

CHART 7: DISTRIBUTION OF TYPE OF LESION

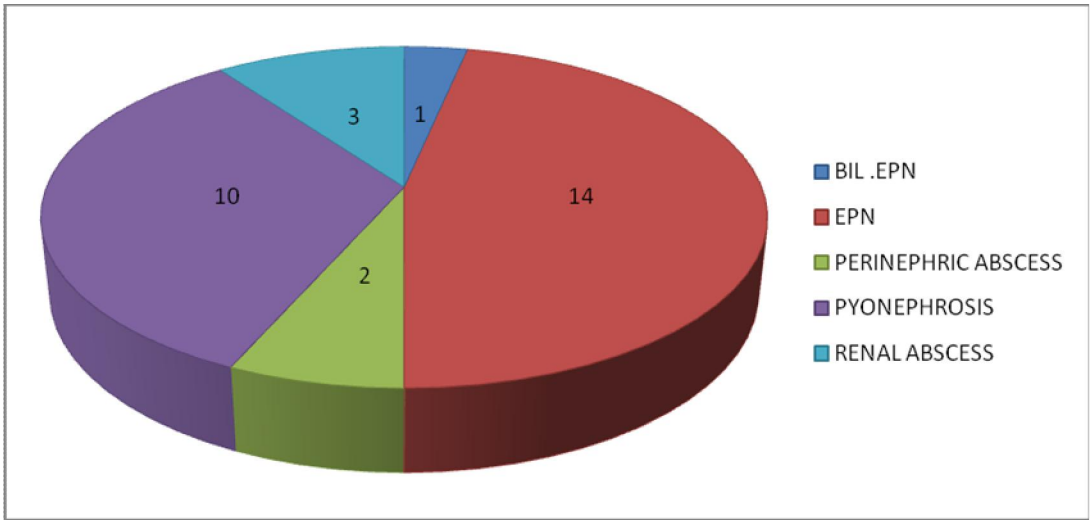


TABLE 6: THERAPEUTIC INTERVENTIONS

MANAGMEMENT	*NUMBERS (%)*	*REQUIRED NEPHRECTOMY*
MEDICAL MANAGEMENT	2 /30 (6.6%)	NIL
PERCUTANEOUS NEPHROSTOMY (PCN)	12/30 (46%)	11
DJ STENTING	7 /30 (23.3%)	NIL
PCD & DJ STENTING	4 /30 (13.33%)	1
OPEN DRAINAGE	3 / 30 (10%)	2
IMAGE GUIDED ASPIRATION	1 / 30 (3.3 %)	NIL

CHART 8 : THERAPEUTIC INTERVENTIONS

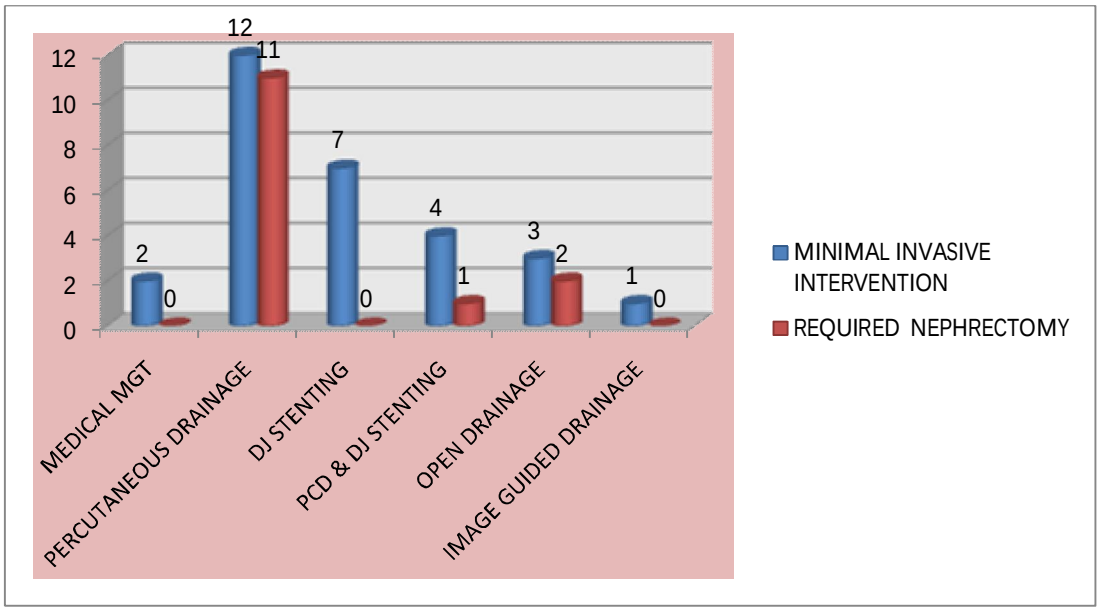
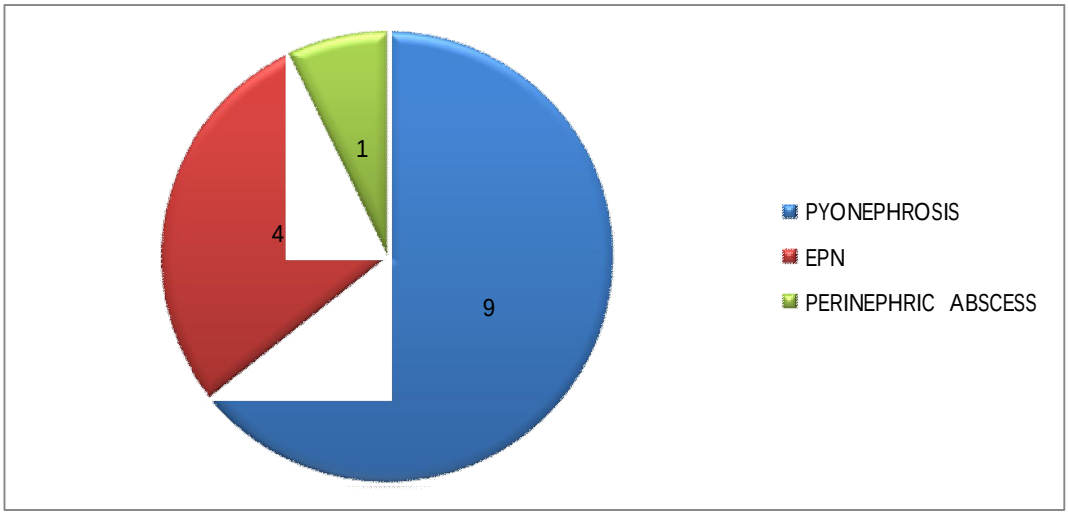


CHART 9 : NEPHRECTOMY AND TYPE OF LESION



The collected data on the various demographic , clinical , biochemical , imaging variables were compared between both the groups to derive out those factors that had significant association with occurrence of nephrectomy implying poorer outcome of the disease process.

STATISTICAL ANALYSIS

TABLE 7 : CORRELATION OF AGE-GROUP WITH OCCURRENCE OF NEPHRECTOMY

Cross tab

			NEPHRECTOMY		
			NO	YES	Total
AGE GROUP	1 (≤ 30 Yrs)	Count	2	4	6
		% of Total	6.7%	13.3%	20.0%
	2 (31 -50 Yrs)	Count	7	7	14
		% of Total	23.3%	23.3%	46.7%
	3 (> 50 Yrs)	Count	7	3	10
		% of Total	23.3%	10.0%	33.3%
	Total	Count	16	14	30
		% of Total	53.3%	46.7%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.143[a]	2	.343
Likelihood Ratio	2.192	2	.334
No. of Valid Cases	30		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is 2.80.

CHI SQUARE : 2.143 P = 0.343 NOT SIGNIFICANT

TABLE 8 : CORRELATION OF GENDER WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		SEX			Total
		FEMALE	MALE	TG	
NEPHRECTOMY	NO	11	4	1	16
	YES	9	5	0	14
Total		20	9	1	30

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.183(a)	2	.553
Likelihood Ratio	1.565	2	.457
No. of Valid Cases	30		

(a) 4 cells (66.7%) have expected count less than 5. The minimum expected count is .47.

CHI SQUARE : 1.183 P = 0.553 NOT SIGNIFICANT

TABLE 9 : CORRELATION OF LATERALITY OF LESION WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		LATERALITY OF LESION			Total
		BILATERAL	LEFT	RIGHT	
NEPHRECTOMY	NO	1	8	7	16
	YES	0	8	6	14
Total		1	16	13	30

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.948(a)	2	.623
Likelihood Ratio	1.330	2	.514
No. of Valid Cases	30		

(a) 2 cells (33.3%) have expected count less than 5. The minimum expected count is .47.

CHI SQUARE : .948 P = 0.623 NOT SIGNIFICANT

TABLE 10 : CORRELATION OF PREDISPOSING RISK FACTOR WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		PREDISPOSING RISK FACTORS						Total
		DIABETES	DIABETES &UROLITHIASIS	LOWER TRACT OBSTRUCTION	NEUROGENIC	PUJO	UROLITHIASIS	
NEPHRECTOMY	NO	13	2	1	0	0	0	16
	YES	4	6	0	1	2	1	14
Total		17	8	1	1	2	1	30

Chi-Square Tests			
	Value	**df**	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	**11.683(a)**	**5**	**.039**
Likelihood Ratio	**13.908**	**5**	**.016**
No. of Valid Cases	**30**		
(a) 10 cells (83.3%) have expected count less than 5. The minimum expected count is .47.			

CHI SQUARE : 11.683 P = 0.039 SIGNIFICANT

TABLE 11:CORRELATION OF ALTERED SENSORIUM AT PRESENTATION WITH OCCURRENCE OF NEPHRECTOMY

Crosstab Count				
		ALTERED SENSORIUM		**Total**
		NO	**YES**	
NEPHRECTOMY	**NO**	16	0	16
	YES	6	8	14
Total		22	8	30

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	12.468(b)	1	.000		
Continuity Correction(a)	9.717	1	.002		
Likelihood Ratio	15.673	1	.000		
Fisher's Exact Test				.001	.001
No. of Valid Cases	30				
(a) Computed only for a 2x2 table					
(b) 2 cells (50.0%) have expected count less than 5. The minimum expected count is 3.73.					

CHI SQUARE : 12.468 P< 0.001 SIGNIFICANT

TABLE 12 : CORRELATION OF HYPOTENSION (SYSTOLIC BP < 90mm Hg) AT PRESENTATION WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		HYPOTENSION		Total
		NO	YES	
NEPHRECTOMY	NO	13	3	16
	YES	8	6	14
Total		21	9	30

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.066(b)	1	.151		
Continuity Correction(a)	1.078	1	.299		
Likelihood Ratio	2.088	1	.148		
Fisher's Exact Test				.236	.150
No. of Valid Cases	30				

(a) Computed only for a 2x2 table

(b) 2 cells (50.0%) have expected count less than 5. The minimum expected count is 4.20.

FISHER'S EXACT TEST P = .236 NOT SIGNIFICANT

TABLE 13 : CORRELATION OF POOR GLYCEMIC CONTROL (HbA1c > 7) WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		INC.HbA1C		Total
		NO	YES	
NEPHRECTOMY	NO	12	4	16
	YES	5	9	14
Total		17	13	30

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	4.693(b)	1	.030		
Continuity Correction(a)	3.229	1	.072		
Likelihood Ratio	4.810	1	.028		
Fisher's Exact Test				.063	.035
No. of Valid Cases	30				
(a) Computed only for a 2x2 table					
(b) 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.07.					

CHI SQUARE : 4.693 P = 0.030 SIGNIFICANT

TABLE 14 : CORRELATION OF LEUCOCYTOSIS (WBC Count > 15,000 /cu.mm) WITH OCCURRENCE OF NEPHRECTOMY

Crosstab Count				
		LEUCOCYTOSIS		**Total**
		NO	**YES**	
NEPHRECTOMY	**NO**	6	10	16
	YES	8	6	14
Total		14	16	30

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	1.158(b)	1	.282		
Continuity Correction(a)	.503	1	.478		
Likelihood Ratio	1.164	1	.281		
Fisher's Exact Test				.464	.240
No. of Valid Cases	30				
(a) Computed only for a 2x2 table					
(b) 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.53.					

FISHER'S EXACT TEST P = .464 NOT SIGNIFICANT

TABLE 15 : CORRELATION OF THROMBOCYTOPENIA (Platelets < 1,00,000/cu.mm)WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		THROMBOCYTOPENIA		Total
		NO	YES	
NEPHRECTOMY	NO	16	0	16
	YES	3	11	14
Total		19	11	30

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	19.850(b)	1	.000		
Continuity Correction(a)	16.610	1	.000		
Likelihood Ratio	24.881	1	.000		
Fisher's Exact Test				.000	.000
No. of Valid Cases	30				

(a) Computed only for a 2x2 table

(b) 0 cells (.0%) have expected count less than 5. The minimum expected count is 5.13.

FISHER'S EXACT TEST P < 0.001 SIGNIFICANT

TABLE 16 : CORRELATION OF INCREASED SERUM CREATININE (Sr. Creatinine > 2 mg /dl) WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		INCREASED SERUM CREATININE		Total
		NO	YES	NO
NEPHRECTOMY	NO	9	7	16
	YES	0	14	14
Total		9	21	30

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	11.250(b)	1	.001		
Continuity Correction(a)	8.731	1	.003		
Likelihood Ratio	14.722	1	.000		
Fisher's Exact Test				.001	.001
No. of Valid Cases	30				
(a) Computed only for a 2x2 table					
(b) 2 cells (50.0%) have expected count less than 5. The minimum expected count is 4.20.					

FISHER'S EXACT TEST P = .001 SIGNIFICANT

TABLE 17: CORRELATION OF POSITIVE URINE CULTURE WITH OCCURRENCE OF NEPHRECTOMY

Crosstab Count				
		URINE CULTURE POSITIVE		**Total**
		NO	**YES**	
NEPHRECTOMY	**NO**	3	13	16
	YES	6	8	14
Total		9	21	30

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	2.066(b)	1	.151		
Continuity Correction(a)	1.078	1	.299		
Likelihood Ratio	2.088	1	.148		
Fisher's Exact Test				.236	.150
No. of Valid Cases	30				
(a) Computed only for a 2x2 table					
(b) 2 cells (50.0%) have expected count less than 5. The minimum expected count is 4.20.					

FISHER'S EXACT TEST P = .236 NOT SIGNIFICANT

TABLE 18 : CORRELATION OF POSITIVE BLOOD CULTURE WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		BLOOD CULTURE POSITIVE		Total
		NO	YES	
NEPHRECTOMY	NO	13	3	16
	YES	8	6	14
Total		21	9	30

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.066(b)	1	.151		
Continuity Correction(a)	1.078	1	.299		
Likelihood Ratio	2.088	1	.148		
Fisher's Exact Test				.236	.150
No. of Valid Cases	30				

(a) Computed only for a 2x2 table

(b) 2 cells (50.0%) have expected count less than 5. The minimum expected count is 4.20.

FISHER'S EXACT TEST P = .236 NOT SIGNIFICANT

TABLE 19 : CORRELATION OF PRESENCE OF HYDRONEPHROSIS WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		HYDRONEPHROSIS +		Total
		NO	YES	
NEPHRECTOMY	NO	12	4	16
	YES	2	12	14
Total		14	16	30

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	11.059(b)	1	.001		
Continuity Correction(a)	8.754	1	.003		
Likelihood Ratio	11.977	1	.001		
Fisher's Exact Test				.001	.001
No. of Valid Cases	30				
(a) Computed only for a 2x2 table					
(b) 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.53.					

FISHER'S EXACT TEST P = .001 SIGNIFICANT

TABLE 20 : CORRELATION OF TYPE OF LESION WITH OCCURRENCE OF NEPHRECTOMY

Crosstab
Count

		TYPE OF LESION					**Total**
		BIL.EPN	**EPN**	**PERINEPHRIC ABSCESS**	**PYONEPHROSIS**	**RENAL ABSCESS**	
NEPHRECTOMY	**NO**	1	10	1	1	3	16
	YES	0	4	1	9	0	14
Total		1	14	2	10	3	30

Chi-Square Tests			
	Value	**df**	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	12.895(a)	4	.012
Likelihood Ratio	15.430	4	.004
No. of Valid Cases	30		
(a) 7 cells (70.0%) have expected count less than 5. The minimum expected count is .47.			

CHI SQUARE : 12.895 P = 0.012 SIGNIFICANT

TABLE 21 : ASSOCIATION OF FACTORS WITH NEPHRECTOMY (DEMOGRAPHIC AND CLINICAL VARIABLES)

	TOTAL NUMBER OF CASES	GROUP I RENAL SALVAGE	GROUP II NEPHRECTOMY	'P' VALUE	SIGNIFICANCE
AGE GROUP I GROUP II GROUP III	 6 14 10	 2 7 7	 4 7 3	0.343	NOT SIGNIFICANT
GENDER MALE FEMALE TRANSGENDER	 9 20 1	 4 11 1	 5 9 0	0.553	NOT SIGNIFICANT
LATERALITY RIGHT LEFT BILATERAL	 13 16 1	 7 8 1	 6 8 0	0.623	NOT SIGNIFICANT
RISK FACTORS 1 DIABETES 2.DM& UROLITHIASIS 3 LOWER TRACT OBSTRUCTION 4.NEUROGENIC DYSFUNCTION 5. PUJ OBSTRUCTION 6. UROLITHIASIS	 17 8 1 1 2 1	 13 2 1 0 0 0	 4 6 0 1 2 1	0.039	SIGNIFICANT
ALTERED SENSORIUM	8	0	8	< 0.001	SIGNIFICANT
HYPOTENSION	9	3	6	0.236	NOT SIGNIFICANT

TABLE 8 : ASSOCIATION OF FACTORS WITH NEPHRECTOMY (LABARATORY AND RADIOLOGICAL VARIABLES)

	TOTAL NUMBER OF CASES	GROUP I RENAL SALVAGE	GROUP II NEPHRECTOMY	'P' VALUE	SIGNIFICANCE
INCREASED HBA1c	13	4	9	0.030	SIGNIFICANT
LEUCOCYTOSIS	16	10	6	0.464	NOT SIGNIFICANT
THROMBO-CYTOPENIA	11	0	11	< 0.001	SIGNIFICANT
INCREASED SERUM CREATININE	21	7	14	0.001	SIGNIFICANT
POSITIVE URINE CULTURE	21	13	8	0.236	NOT SIGNIFICANT
POSITIVE BLOOD CULTURE	9	3	6	0.236	NOT SIGNIFICANT
PRESENCE OF HYDRONEPHROSIS	16	4	12	0.001	SIGNIFICANT
TYPE OF LESION PYONEPHROSIS EPN BILATERAL EPN PERINEPHRIC-ABSCESS CORTICAL – ABSCESS	 10 14 1 2 3	 1 10 1 1 3	 9 4 0 1 0	 0.012 	 SIGNIFICANT

ANALYSIS AND DISCUSSION

Inflammatory lesions of kidney commonly encountered in urological practice are secondary to infectious processs although a variety of immunologic, congenital, metabolic or toxin induced diseases can present as renal inflammation. The inflammatory process may be acute usually following a bacterial infection (Bacterial nephritis) and includes spectrum of clinical entities, progressing from mild acute pyelonephritis to renal abscesses or emphysematous pyelonephritis. Inflammation as a chronic process occur as a consequence of either infection superadded on structural and functional abnormalities such as vesicoureteral reflux disease or reactivation of latent infection with organisms like Mycobacterium tuberculosis. There are no specific symptoms for chronic pyelonephritis until it produces renal insufficiency, and then the symptoms are similar to those of any other form of chronic renal failure.

The severity of the renal infection depends on the virulence factors of the offending agent and the strength of host defence mechanisms. We conducted this study to analyse the host factors that influences the clinical course and the final outcome of acute inflammatory pathology.

We included in the study patients who were admitted with symptoms suggestive of acute renal inflammation which included presence of loin or flank pain of short duration , fever with chills with or with out lower urinary tract symptoms and whose imaging studies revealed the presence of inflammation either in the renal parenchyma or collecting system.

37 patients fulfilled the inclusion criteria but among them 4 patients who had recurrence of the disease in the followup period and 3 patients who did not attend the followup clinics were excluded from the study and hence the data from 30 patients were taken up for the statistical analysis.

In our study the mean age of the patients was 46.67 years and minimum and maximum ages were 15 years and 67 years respectively. Female out - numbered male (20 : 9) . There was one transgender patient (patient number 14 in chart) who was admitted with right perinephric abscess consequent to urethral stenosis following the sex realignment surgery.

The renal lesions were more common in the left side in our study than right (16 : 13) . One of the patient (patient number 4 in chart) had bilateral presentation of emphysematous pyelonephritis.

The predominant risk factor leading to the complicated renal infections in our study was the presence of diabetes (17 patients ;

56.7%) . Concomitant diabetes and urinary stones were present in 8 patients (26.7%) most of them landing up with pyonephrosis. Upper tract abnormality in the form of pelvi-ureteric junction obstruction was seen in 2 patients (6.7%) . Presence of lower urinary tract obstruction in the form of urethral stenosis, neurogenic bladder dysfunction with vesicoureteric reflux and non obstructive renal stone were also noted as predisposing risk factor in our patients .

The patients were grouped into two for the purpose of analysing the statistical analysis of the collected data . Group I (16 in number) were the patients whose affected renal unit were conserved by medical and minimally invasive surgical interventions. Group II comprising patients (14 in number) where there was deterioration of clinical condition inspite of these interventions warranting nephrectomy to avoid mortality.

Mortality in our study occurred in 3 patients (10%). One patient with Class III (Huang and Tseng et al [42]) emphysematous pyelonephritis (patient number 6 in chart) and another patient with pyonephrosis (patient number 27 in chart) expired following nephrectomy in the immediate post operative period due to acute respiratory distress syndrome (ARDS) .Another patient (patient number 26 in chart) who had nephrectomy for emphysematous pyelonephritis expired due to myocardial infarction in the followup period .

The various demographic , clinical , biochemical , imaging variables were compared between both the groups to derive out those factors that had significant association with occurrence of nephrectomy implying poorer outcome of the disease process.

The patients were categorised in to 3 groups according to their age ; Group I $\leq$ 30 years (6 patients) , Group II 31 – 50 years (14 patients) and Group III $\geq$ 50 years (10 patients) for statistical analysis. Age Group did not have any significant association with the occurrence of nephrectomy (P value of 0.343) (Table. 7)

Although females formed the predominant gender group in our study (16 patients) , the gender of the patient showed no statistical significance with the nephrectomy (P value of 0.553) (Table. 8)

Left sided lesions were more common in our study (16 patients) but the side of the lesion did not have any influence in the progression of the disease to terminate in nephrectomy . Test of significance showed a P value of 0.623 (Table. 9) which was not statistically significant.

Diabetes was the predominant predisposing risk factor in our patients in the study (totally 25 patients with 8 of the patients had concomitant urolithiasis) . This was in concordance to the various studies available which highlight the role of diabetes in upper tract infections. Studies by Muller et al [31] and Joshi et al [32] showed that diabetes may predispose patients to more severe infections and

complications of the upper urinary tract with its inherent capacity to reduce the host immune response. Hoepelman et al [33] and Patterson et al [34] in their studies emphasised the fact that infections in diabetics are more difficult to treat and tend to recur often. Some studies showed that diabetes increases the risk of hospitalization and mortality from infections. [35,36] Only 4 of the 17 patients in the diabetes alone group progressed to nephrectomy (23.5%) but 6 out of the 8 patients (75 %) in the concomitant diabetes and urolithiasis group had severe disease process progressing to nephrectomy. This constitutes the reason for significant statistical association of the type of inflammatory lesion in our study to nephrectomy occurrence (P value of 0.039). (Table. 10)

Two important clinical presentation at the time of admission; altered mental status and the presence of hemodynamic instability were analysed in our study to derive out their influence in the occurrence of nephrectomy.

Altered sensorium evidenced by the presence of mental confusion, disorientation and stupor was observed in 8 of our patients (26.66%) as they presented with overwhelming sepsis at the time of admission. Although all these patients recovered their sensorium following resuscitation , antibiotics and minimal invasive procedures their renal units were grossly tampered by the disease process and all these 8 patients (100%) landed up with nephrectomy .In statistical analysis there

was significant statistical association with altered sensorium at clinical presentation and nephrectomy (P value of <0.001) (Table. 11) confirming it as one of the important prognostic factor influencing outcome of complicated renal infections .

Hemodynamic instability as evidenced by hypotension with systolic pressure of less than 90 mm Hg was present in 9 of the patients (30 %) in our study. These patients required resuscitation with the use of intravenous fluids and administration of ionotropic agents . 6 of the patients (66.6%) had nephrectomy as their final treatment outcome and the statistical analysis showed no statistical association between the presence of hypotension at clinical presentation and nephrectomy . P value in the test of significance was 0.236 which was not significant. (Table. 12)

Efstathiou et al.[37] in their study on predictors of poor treatment outcome of complicated pyelonephritis showed that age > 65 years , septic shock and bedridden status had greater risk of death . Our study revealed altered sensorium but not presence of hemodynamic stability as one of the factors predicting poor outcome .

The laboratary biochemical parameters of HbA1c (glycosylated haemoglobin), total white blood cell count, serum creatinine level ,blood platelet levels , urine and blood culture of the patients were analysed between the two groups to determine their predicting ability for the occurrence of nephrectomy.

HbA1c level in the blood reflects the adequacy of control of diabetes with normal value of 7 indicating good glycemic control. HbA1c levels were checked in all of our diabetic patients (25 in number) . Increased HbA1c (value of > 7) was present in 13 of the 25 patients (52%). 9 among them had the occurrence of nephrectomy as their terminal event (69%) . Statistical analysis revealed significant statistical association with the P value of 0.030. (Table. 13) Although previously suggested as possible risk factors, duration of diabetes or elevated HbA1c levels have not been shown to increase the risk of UTI in recent studies. by Geerlings et al. [38] and Boyko et al. [39]

Leucocytosis is a frequent accompaniment in acute infectious process . A white blood cell count more than 15,000 / cubic millimeter was considered as leucocytosis in our study and it was present in 16 patients (53.33%) . Statistical analysis showed no significant association between the presence of leucocytosis and nephrectomy occurrence (P value of 0.464) (Table. 14)

Infection can induce secondary immune mediated destruction of platelets and because of the short life span the platelet reserves get exhausted in a quicker time to result in significant thrombocytopenia. We had count of platelets less than 1,00,000 / cubic millimeter as defining criteria of thrombocytopenia in our study . This was present in 11 patients (36.6%) and all had nephrectomy (100%). Statistical analysis

showed significant statistical association with occurrence of nephrectomy with the P value of < 0.001. (Table. 15)Thrombocytopenia has been considered as a significant poor prognostic factor for emphysematous pyelonephritis in various studies by Falagas *et al.* [40] and Wan et al. [41]

Raised serum creatinine level (> 2 mg / deciliter) at initial presentation was present in 21 of the patients (70%) reflecting the severity of the infection . Among these patients 14 (66.6%) landed up with nephrectomy . Stastistical analysis showed significant association between the raised creatinine level and nephrectomy (P value =0.001) (Table. 16) implicating the fact that patient with acute renal insufficiency had more protracted disease outcome . Falagas et al [40] showed in his study that raised serum creatinine (2.5mg /dl) at the time of presentation is associated with poor prognosis increasing mortality in emphysematous pyelonephritis.

Urine culture was positive in 21 patients (70%) in our study but there was no statistical association of this variable among the nephrectomy group (P value = 0.236). (Table. 17) Blood culture was positive in a lesser number of patients; only 9 patients (30%) had significant bacteremia but statistical analysis failed to show any significant association between bacteremia and nephrectomy. (Table. 18) Prior antibiotic usage accounted for significant number of patients having negative culture.

Presence of hydronephrosis and the type of presenting inflammatory lesion in the imaging studies were analysed to probe their association with the need of nephrectomy in our study .

Hydronephrosis of varying degrees was apparent in 16 patients (53.33%) ; 10 among them was associated with pyonephrosis and 6 with emphysematous pyelonephritis . 12 of these patients had to be performed with nephrectomy following failure of initial percutaneous nephrostomy drainage as the PCN fluid analysis and radio isotope scan revealed non functioning renal moiety. Statistical analysis showed significant association between the presence of hydronephrosis and nephrectomy procedure with test of significance P value being 0.001. (Table. 19)

Presenting lesion type greatly influence the therapeutic option excised on the patient . Nephrectomy was inevitable in 9 patients with pyonephrosis (30%) and 4 patients of severe emphysematous pyelonephritis which belong to class III of Huang and Tseng et al. CT classification [42] and these lesions formed the major bulk of nephrectomy in our study. Group statistics rightly showed significant association between the type of presenting lesion and the need for nephrectomy with the P value being 0.012. (Table. 20)

In the study conducted by Kapoor et al[43] on predictive factors for the need of nephrectomy in emphysematous pyelonephritis cases, extensive renal parenchymal destruction of 50% (based on

computed tomography) significantly predicted the need for nephrectomy . Presence of hydronephrosis nor Huang and Tseng et al. CT scan classification [42] of EPN cases did not have significant association in their study . Our study had higher number of pyonephrotic lesions landing up with nephrectomy and hence the reason for significant association of presence of hydronephrosis and type of lesion with nephrectomy .

Limitations of our Study

There are few limitations considering the outcome of this study

- Our study may not be representative of the general population in view of the small cohort and short duration of the study
- Our hospital being a tertiary institute , the cohort consisted more of complicated cases referred from other places which resulted in skewing of increased nephrectomy rates (40%)
- Followup period in our study is short (up to 3 months) and a lengthier followup is necessary inorder to give a more predictable outcome.

CONCLUSION

- Diabetes is the most common predisposing risk factor for the occurrence of acute inflammatory lesion of kidneys in our study .

- Concomitant presence of diabetes and urolithiasis confers higher incidence of severe renal parenchymal damage leading to nephrectomy

- The presence of an altered mental status at presentation ,poor glycemic control (increased HbA1c levels) , increased serum creatinine , thrombocytopenia and presence of hydronephrosis at presentation are associated with higher need for nephrectomy in our study

- Type of renal lesion at presentation significantly influence the therapeutic option excised and the need for performing nephrectomy.

- Our study shows no significant association with age, gender ,laterality of lesion , haemodynamic instability at presentation , leukocytosis, positive urine culture and bacteraemia with the occurrence of nephrectomy.

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ANNEXURE I - Ethical Committee Approval

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.N.1463/MEI(Ethics)/2012 Dt:03.04.2012

CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College , Chennai reviewed and discussed the application for approval entitled "Factors influencing the course and outcome of Inflammatory lesions of Kidney in adults" submitted by Dr.A.Larif, Mch(Urology) PG Student, Govt. Kilpauk Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE
15 JUN 2012
Kilpauk, Chennai-10.

13/6/12
CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

ANNEXURE II -
ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சித் தலைப்பு : சீறுநீரக அழற்சி நோய்கக‌ளுக்கான காரணங்கள் மற்றும் நோயின் தன்மை மற்றும் நோய் பாதிப்பு குறித்த ஆய்வு (STUDY ON FACTORS INFLUENCING THE COURSE AND OUTCOME OF INFLAMMATORY LESIONS OF KIDNEY IN ADULTS.)

பெயர் : தேதி :
வயது : உள்நோயாளி எண் :
பால் : ஆராய்ச்சிச் சேர்க்கை எண் :

சீறுநீரக அழற்சி நோய்கக‌ளுக்கான காரணங்கள் மற்றும் நோயின் தன்மை மற்றும் நோய் பாதிப்பு குறித்த ஆய்வில் பங்கு பெற விரும்புகிறேன். இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் எனக்கு முழுமையாகவும் தெளிவாகவும் விளக்கப்பட்டன. எனக்கு விளக்கப் பட்ட விஷயங்களைப் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தருகிறேன்.

எனது சிறுநீரகங்களின் செயல்பாடு குறித்த பல பரிசோதனைகளை செய்து கொள்ள வேண்டும் என்றும் , இது தொடர்பான மருத்துவ சிகிச்சைகள், மருந்து உட்கொள்ளுதல், மருந்து செலுத்துதல் மற்றும் அறுவை சிகிச்சைகளை செய்ய வேண்டிய அவசியம் வரலாம் என்பது எனக்கு விளக்கி கூறப்பட்டது

சிறுநீரகத்தில் அடைப்பு இருந்தால் அதை நீக்கவேண்டுமா? இல்லையா? என்பது குறித்து அறிய எனது சிறுநீரகத்தில் ஒரு குழாய் (PCN) வைத்து பரிசோதனை செய்யப்படும் என்பதும், சிறுநீரகம் பாதிப்படைதிருந்தால், அதை நீக்க வேண்டிவரும் (NEPHRECTOMY) என்பது எனக்கு விளக்கி கூறப்பட்டது இது தொடர்பான பக்க விளைவுகளும் பிற விபரங்களும் எனக்கு விளக்கி கூறப்பட்டுள்ளன.

சீறுநீரக அழற்சி நோய்கக‌ளுக்கான காரணங்கள் மற்றும் நோயின் தன்மை மற்றும் நோய் பாதிப்பு குறித்த ஆராய்ச்சியில் பங்கேற்க நான் சம்மதம் தெரிவிக்கின்றேன்.

இந்த ஆராய்ச்சியில் பிறர் நிர்ப்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகிறேன் . மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் விலகலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னைச் சேர்த்துக் கொள்ளச் சம்மதிக்கிறேன்

கையொப்பம்

ANNEXURE III - Copy of Informed Consent

I _____ willing to participate in the study titled

‘ STUDY ON FACTORS INFLUENCING THE COURSE AND OUTCOME OF INFLAMMATORY LESIONS OF KIDNEY IN ADULTS .’

I am aware that I will be undergoing various tests to study my kidney function. I am willing to undergo any medical and surgical intervention to treat my ailment. I am aware that a tube may be placed in my affected kidney to assess whether it is advisable to repair the block.

The final decision to remove affected kidney or repair it ,will be based on the results. The complications and other details have been extensively discussed with me. The purpose and the advantage of the study has been explained in the mother tongue by the investigator. I am also aware that I can discontinue from the study whenever I wished to do so.

Date:

Place:

Signature of the Patient/Guardian

ANNEXURE IV - COPY OF PROFORMA

INFLAMMATORY LESIONS OF KIDNEY

Name - **Age/Sex:** **IP.No-** **D.O.A**

Address- **D.O.D**

COMPLAINTS :

PRESENT H/0 :

PAST H/O – Complicating factors

Intubated Urinary tract : Indwelling catheters --

Internal Stents -- Ureteral or Urethral

Obstruction : Upper Tract / Lower Tract Treatment (if any)

Childhood Infections :

Diabetes mellitus: Period : Treatment : OHA / Insulin

Presence of Ketosis:

Immunosuppression: HIV: TB:

Use of steroids:

Urolithiasis : Site: Intervention :

Clearance:

Surgery of the urinary tract:

Functional and anatomical abnormalities of the urinary tract

Voiding dysfunction / Neurogenic :

EXAMINATION : G/E: Temp: PR:

BP: Mental Status :

P/A:

External Genitals:

PR:

CLINICAL DIAGNOSIS:

INVESTIGATIONS :

Urine Routine Alb: Sugar: Deposits:

Ketones:

Urine Culture & Sensitivity :

Blood Hb : TC: DC: ESR:

Platelet:

Coagulation Profile :

Blood Sugar: HbA1c : Blood Urea:

Sr . Creatinine: Sr . Electrolytes :

Blood Culture & Sensitivity :

X ray KUB:

USG KUB:

IVU:

CT KUB :

Chest X Ray :

ECG : :

Blood Grouping & Typing:

Renal isotope study : DTPA / DMSA

COMPLICATIONS:

PLAN –

Medical Treatment:

Blood Transfusion (if any):

Surgical Procedure done : STENTING / PCN / Open Drainage / NEPHRECTOMY / OTHERS

Post Operative Complications :

Post Operative Biochemistry & Imaging (if any):

Follow up:

ANNEXURE V – MASTER CHART

SL.NO	NAME	AGE	SEX	IP.No	Date of Admisssion	Institution	Predisposing Risk Factor	Altered Sensorium At Presentation	Hypotension At Presentation	Urine Culture	Blood Hemoglobin (gm%)	Total WBC Count (per cub.mm)	Differential count	Platelet Count (per cub.mm)	Random Blood Sugar (mgm%)	HbA1c (%)	Blood Urea (mgm%)	Serum Creatinine (mg/dl)	Blood Culture	Type of Lesion	Presence of Hydronephrosis	Primsry Therapeutic Intervention	Nephrectomy	Death
1.	Mrs. Amsavalli	61	Female	2834	4.1.12	KMCH	Diabetes	NO	NO	E.coli	10.2	16,700	P78,L20,E2	2,50,000	212	8	58	2.8	No Growth	Lt. Emphysematous Pyelonephritis	YES	Lt.Percutaneous Nephrostomy→ Lt.DJ Stenting	NO	NO
2.	Mrs. Mallika	62	Female	4122	20.2.12	KMCH	Diabetes	YES	YES	E.coli	9.3	12,200	P65,L32,E3	80,000	256	9.2	48	2.9	E.coli	Lt. Emphysematous Pyelonephritis	YES	Lt.Percutaneous Nephrostomy	YES	NO
3.	Mrs. Mohana	36	Female	8472	2.4.12	KMCH	Diabetes	NO	NO	No Growth	11.2	11,500	P66,L33,E1	2,75,000	187	6.3	23	1.1	No Growth	Rt. Renal Cortical Abscess	NO	Medical Management	NO	NO
4.	Mrs.Sundari	49	Female	996609	11.4.12	GRH	Diabetes	NO	NO	Pseudomonas	9.4	17,200	P82,L17,E1	1,85,000	235	8.2	57	2.7	E.coli	Bilateral Emphysematous Pyelonephritis	NO	Bilateral DJ Stenting	NO	NO
5.	Ms. Meena	15	Female	10497	20.4.12	KMCH	PUJ obstruction	NO	NO	No Growth	11	16,900	P78,L21,E1	2,85,000	98	-	45	2.6	No Growth	Lt. Pyonephrosis	YES	Lt. Percutaneous Nephrostomy	YES	NO
6.	Mr.Murali	42	Male	996909	2.5.12	GRH	Diabetes	YES	NO	Klebsiella	10.3	18,200	P75,L23,E2	79,000	220	7.9	50	2.5	No Growth	Lt. Emphysematous Pyelonephritis	NO	Lt.Open Drainage	YES	YES
7.	Mr. Pavadai	56	Male	11961	3.5.12	KMCH	Diabetes	NO	NO	Proteus	11.8	17,500	P72,L26,E2	1,80,000	149	5.3	27	1.2	Klebsiella	Lt.Emphysematous Pyelonephritis	NO	Lt. DJ Stenting	NO	NO
8.	Mr.Sajid Khan	64	Male	9261	5.5.12	KMCH	Diabetes	NO	YES	E.coli	12	8,700	P66,L33,E1	2,75,000	153	6.1	30	1.1	No Growth	Rt.Emphysematous Pyelonephritis	YES	Rt. DJ Stenting	NO	NO
9.	Mrs .Unnamalai	48	Female	12334	7.5.12	KMCH	Diabetes	NO	NO	Pseudomonas	9.2	11,000	P70,L27,E3	' 2,50,000	176	6.4	48	2.9	No Growth	Rt.Emphysematous Pyelonephritis	YES	Rt. DJ Stenting	NO	NO
10.	Mrs. Sumathi	49	Female	13213	12.5.12	KMCH	Diabetes	NO	NO	E.coli	10.4	17,600	P74,L23,E3	3,12,000	156	5.1	31	1.4	No Growth	Lt. Emphysematous Pyelonephritis	NO	Lt. Percutaneous Nephrostomy	NO	NO
11.	Mrs.Kannagi	67	Female	997382	15.5.12	GRH	Diabetes	NO	YES	E.coli	8.6	9,800	P68,L30,E2	2,50,000	178	5.6	26	1.3	No Growth	Lt. Emphysematous Pyelonephritis	NO	Lt. Percutaneous Nephrostomy → Lt.DJ Stenting	NO	NO
12.	Mrs.Parvathy	48	Female	13622	18.5.12	KMCH	Diabetes & Urolithiasis	YES	NO	Klebsiella	9.4	10,200	P63,L35,E2	76,000	257	9.3	60	2.9	E.coli	Lt. Pyonephrosis	YES	Lt. Percutaneous Nephrostomy	YES	NO
13.	Mrs.Jothi	50	Female	998998	25.5.12	GRH	Diabetes & Urolithiasis	NO	NO	Proteus	9.5	9,500	P65,L34,E1	80,000	211	6.7	45	2.7	No Growth	Rt. Perinephric Abscess	NO	Open Drainage	YES	NO
14.	Ms.Santhya	24	Trans Gender	15786	4.6.12	KMCH	Lower Urinary Tract Obstruction	NO	YES	E.coli	8.7	19,800	P80,L19,E1	1,78,000	123	-	55	2.6	No Growth	Rt.Perinephric Abscess	NO	Open Drainage	NO	NO
15.	Mr.Mohan	50	Male	16824	13.6.12	KMCH	Diabetes	NO	NO	No Growth	12	8,900	P65,L34,E1	2,30,000	110	5.2	26	1.2	No Growth	Lt. Renal Cortical Abscess	NO	Medical Management	NO	NO
16.	Mr.Murugan	28	Male	100927	20.6.12	GRH	PUJ Obstruction	NO	NO	No Growth	11.5	16,500	P72,L36,E2	73,000	114	-	49	2.7	No Growth	Rt.Pyonephrosis	YES	Rt. Percutaneous Nephrostomy	YES	NO

SL.NO	NAME	AGE	SEX	IP. No	Date of Admission	Institution	Predisposing Risk Factor	Altered Sensorium At Presentation	Hypotension At Presentation	Urine Culture	Blood Hemoglobin (gm%)	Total WBC Count (per cub.mm)	Differential count	Platelet Count (per cub.mm)	Random Blood Sugar (mgm%)	HbA1c (%)	Blood Urea (mgm%)	Serum Creatinine (mg/dl)	Blood Culture	Type of Lesion	Presence of Hydronephrosis	Primsry Therapeutic Intervention	Nephrectomy	Death
17.	Mr.Sankar	40	Male	101834	4.7.12	GRH	Diabetes & Urolithiasis	NO	NO	E.coli	10.2	17,000	P73,L25,E2	1,78,000	230	7.8	32	1.6	No Growth	Lt. Emphysematous Pyelonephritis	NO	Lt.DJ Stenting	NO	NO
18.	Mrs.Prabhavathi	45	Female	101654	13.7.12	GRH	Diabetes & Urolithiasis	YES	NO	No Growth	9.3	15,500	P78,L21,E1	2,50,000	209	8.4	59	2.8	No Growth	Lt. Pyonephrosis	YES	Lt. Percutaneous Nephrostomy	YES	NO
19.	Mrs.Parameswari	67	Female	25293	7.8.12	KMCH	Diabetes	YES	YES	Klebsiella	9.6	8,900	P70,L28,E2	80,000	289	11.2	60	3.1	Pseudomonas	Lt. Emphysematous Pyelonephritis	YES	Lt. Percutaneous Nephrostomy	YES	NO
20.	Mrs.Pencillama	56	Female	105192	13.8.12	GRH	Diabetes	NO	NO	No Growth	11.3	7,900	P67,L32,E1	2,30,000	156	5.1	28	1.3	No Growth	Rt.Renal Cortical Abscess	NO	USG guided Aspiration	NO	NO
21.	Ms. Lakshmi	22	Female	105212	22.8.12	GRH	Diabetes & Urolithiasis	NO	NO	E.coli	11.2	16,500	P72,L26,E2	3,10,000	145	5.7	52	2.7	Staphylococcus	Lt. Pyonephrosis	YES	Lt. Percutaneous Nephrostomy	NO	NO
22.	Mr.Thangadurai	39	Male	105894	24.8.12	GRH	Diabetes & Urolithiasis	YES	YES	No Growth	10.2	7,900	P68,L30,E2	87,000	267	8.5	50	3.1	No Growth	Rt. Pyonephrosis	YES	Rt. Percutaneous Nephrostomy	YES	NO
23.	Mrs. Muthamil	63	Female	106254	13.9.12	GRH	Diabetes	NO	NO	Klebsiella	11	18,700	P75,L24,E1	2,10,000	178	6.4	39	2.6	No Growth	Lt. Emphysematous Pyelonephritis	NO	Lt. Percutaneous Nephrostomy → Lt.DJ Stenting	NO	NO
24.	Mrs. Mydeen Meeral	53	Female	107694	17.9.12	GRH	Diabetes	NO	NO	Citrobacter	9.8	15,600	P78,L21,E1	3,19,000	165	6.1	30	1.5	No Growth	Rt. Emphysematous Pyelonephritis	NO	Rt. DJ Stenting	NO	NO
25.	Mr.Subramani	50	Male	28436	29.9.12	KMCH	Neurogenic Dysfunction	NO	YES	No Growth	10.2	10,800	P70,L29,E1	73,000	112	-	57	2.7	No Growth	Rt. Pyonephrosis	YES	Rt. Percutaneous Nephrostomy	YES	NO
26.	Mrs. Vasanthi	67	Female	31334	7.10.12	KMCH	Diabetes	NO	NO	E.coli	10.3	9,600	P69,L30,E1	67,000	332	9.5	60	2.9	E.coli	Lt. Emphysematous Pyonephrosis	YES	Lt. Percutaneous Nephrostomy → Lt.DJ Stenting	YES	YES
27.	Mrs.Anthoniammal	47	Female	32374	5.11.12	KMCH	Diabetes & Urolithiasis	YES	YES	E.coli	8.3	11,700	P66,L32,E2	84,000	275	8.3	47	2.6	Staphylococcus	Rt. Pyonephrosis	YES	Rt. Percutaneous Nephrostomy	YES	YES
28.	Mr. Abdul Kani	23	Male	113045	9.12.12	GRH	Urolithiasis	NO	NO	E.coli	12.2	16,800	P72,L27,E1	2,75,000	98	-	65	3.1	No Growth	Rt.Pyonephrosis	YES	Rt. Percutaneous Nephrostomy	YES	NO
29.	Mrs. Radha	30	Female	113302	17.12.12	GRH	Diabetes & Urolithiasis	YES	YES	No Growth	10.3	19,200	P81,L17,E1	91,000	234	7.8	72	2.8	Klebsiella	Lt. Pyonephrosis	YES	Lt. Percutaneous Nephrostomy	YES	NO
30.	Mrs. Vatchala	47	Female	113937	26.12.12	GRH	Diabetes	NO	NO	E.coli	11	15,500	P72,L26,E2	1,87,000	254	7.3	64	2.9	No Growth	Rt. Emphysematous Pyelonephritis	NO	Rt. DJ Stenting	NO	NO

ANNEXURE VI – TURNITIN REPORT FOR PLAGIARISM