“STUDY OF CLINICAL AND BIOCHEMICAL FACTORS DETERMINING PROGNOSIS OF PATIENTS WITH ACUTE PYELONEPHRITIS”

A DISSERTATION SUBMITTED TO THE TAMILNADU
Dr. MGR MEDICAL UNIVERSITY

CHENNAI

In partial fulfilment of the Regulations

for the award of the Degree of

M.D. (GENERAL MEDICINE) BRANCH - I

DEPARTMENT OF GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

MAY 2018

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CONTENTS

ABBREVIATIONS

DM-DIABETES MELLITUS

UTI-URINARY TRACT INFECTIONS

EPN-EMPHYSEMATOUS PYELONEPHRITIS

NEPN-NON EMPHYSEMATOUS PYELONEPHRITIS

CECT-CONTRAST ENHANCED CT SCAN

DMSA SCAN-DIMERCAPTO SUCCINIC ACID SCAN

TMP-SMX-TRIMETHOPRIM –SULFAMETHOXAZOLE

ASB-ASYMPTOMATIC BACTERIURIA

CAUTI-CATHETER ASSOCIATED URINARY TRACT INFECTIONS

PCN-PERCUTANEOUS NEPHROSTOMY

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ANNEXURE

PROFORMA

STUDY OF CLINICAL AND BIOCHEMICAL FACTORS DETERMINING PROGNOSIS IN PATIENTS WITH ACUTE PYELONEPHRITIS

PATIENT DETAILS Sl.No:

NAME: AGE : SEX : IP NO:

ADDRESS: OCCUPATION: DOA:

DOD:

CO-MORBIDITIES:

T1 DM	T2 DM	S H T	CA HD	PT B	BRONC HIAL ASTHM A	IMMUNOCOMP ROMISED STATE	CK D	MISCELLA NEOUS

IF DIABETIC:

DURATION OF DIABETES

DRUG COMPLIANCE

PERSONAL HISTORY

FAMILY HISTORY

SOCIO-ECONOMIC STATUS

TREATMENT HISTORY

PRESENTING COMPLAINTS

GENERAL EXAMINATION

VITALS:

BP:

PULSE:

TEMPERATURE:

R.R:

SYSTEMIC EXAMINATION:

CVS:

RS:

P/A:

CNS:

LABORATORY DATA:

1)CBC:

TC			
DC			
PLATELETS			
Hb			

2)RFT & S.ELECTROLYTES:

BLOOD UREA			
SERUM CREATININE			
SODIUM			
POTASSIUM			

3)LFT:

S.BILIRUBIN		
DIRECT		
INDIRECT		
TOTAL PROTEIN		
ALBUMIN		
GLOBULIN		
SGPT		
SGOT		
ALP		

FBS				
PPBS				
HBA1C				
RBS				

ALBUMIN	SUGAR	DEPOSIT	C/S	ACETONE

USG ABDOMEN & KUB:

CECT ABDOMEN & KUB:

OUTCOMES:

NEED FOR PERCUTANEOUS DRAINAGE :

NEED FOR NEPHRECTOMY:

IMPROVED & DISCHARGED:

DEATH:

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CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO:855/GM/2016

PROTOCOL TITLE: STUDY OF CLINICAL AND BIOCHEMICAL FACTORS DETERMINING PROGNOSIS OF PATIENTS WITH ACUTE PYELONEPHRITIS.

PRINCIPAL INVESTIGATOR: Dr. G.BHARATHI MBBS

DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE IN GENERAL MEDICINE

DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear , Dr. G. Bharathi, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 05.08.2016.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

Dr.K.Shantaraman MD
Registrar, TIREC
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Dr.J.Suresh Durai, MD
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CERTIFICATE - II

This is certify that this dissertation work title **"STUDY OF CLINICAL AND BIOCHEMICAL FACTORS DETERMINING PROGNOSIS OF PATIENTS WITH ACUTE PYELONEPHRITIS"** of the candidate **Dr.G.BHARATHI** with registration Number **201511351** for the award of M.D. in the branch of **GENERAL MEDICINE.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **1 percentage** of plagiarism in the dissertation.

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INTRODUCTION

In a recent community based estimate, UTI were found to be second only to LRTI among older diabetics.The extent of involvement ranges from inconsequential lower urinary tract colonization to cystitis, pyelonephritis, renal or perirenal abscess.

Emphysematous pyelonephritis(EPN) is the necrotizing infection of renal parenchyma with the presence of gas in the renal parenchyma, collecting system or perinephric tissue. EPN is an uncommon life threatening condition precipitated mainly by poorly controlled blood sugars & urinary tract obstruction.

Prevalence of diabetes in patient with emphysematous pyelonephritis ranges from 53-90 %. It is treated with conventional parenteral antibiotics with percutaneous /open surgical drainage with or without nephrectomy.

However there have been few large studies, which have selectively looked into the clinical, microbial profile & treatment outcome of diabetic patients with pyelonephritis both Non emphysematous pyelonephritis (NEPN) & EPN.

Hence to address this issue this prospective observational study is undertaken to evaluate factors determining prognosis of patients with pyelonephritis.

AIM AND OBJECTIVES

To study the clinical & biochemical factors determining prognosis of patients with acute pyelonephritis

REVIEW OF LITERATURE

DEFINTIONS:

1. URINARY TRACT INFECTION:

UTI is currently defined as the inflammatory response of the urothelium to bacterial invasion, which comprises a constellation of clinical entities.

2. BACTERIURIA:

Bacteriuria is the presence of bacteria in urine which indicates colonization rather than active infection.

3. PYURIA:

Pyuria indicates the presence of white blood cells in urine which in turn is either due to infection or due to other inflammatory causes such as carcinoma in situ, bladder stones etc (sterile pyuria).

4.UNCOMPLICATED UTI:

Uncomplicated UTI refers to acute cystitis or pyelonephritis in non pregnant outpatient women in a structurally and functionally normal urinary

tract. Usually common in women and has got good prognosis with a course of antibiotics.

5. COMPLICATED UTI:

Complicated UTI is that which occurs in a structurally and functionally abnormal urinary tract such as in bladder outlet obstruction due to BPH, bladder stones, or due to immunosuppression, diabetes mellitus, indwelling catheters, and recent urinary tract interventions etc.

Complicated form of UTI is the commonest form occurring in men and has a delayed prognosis with antibiotic treatment. Recurrence is also common.

TYPES OF UTI:

1. ISOLATED UTI:

In this the time interval between two episodes of uti is more than 6 months.

2. RECURRENT UTI:

Two or more episodes in 6 months or three episodes in one year are referred to as recurrent UTI. This is either due to inhabitance of bacteria (in bladder stones etc) or due to reinfection with mixed organisms.

3.UNRESOLVED UTI:

In this type the UTI is not cured due to inappropriate usage of antibiotics, mixed infection, or due to rapid reinfections.

The entire urinary tract is divided into upper and lower urinary tract.

UPPER URINARY TRACT:

The upper urinary tract comprises of the kidneys and the ureters.

LOWER URINARY TRACT:

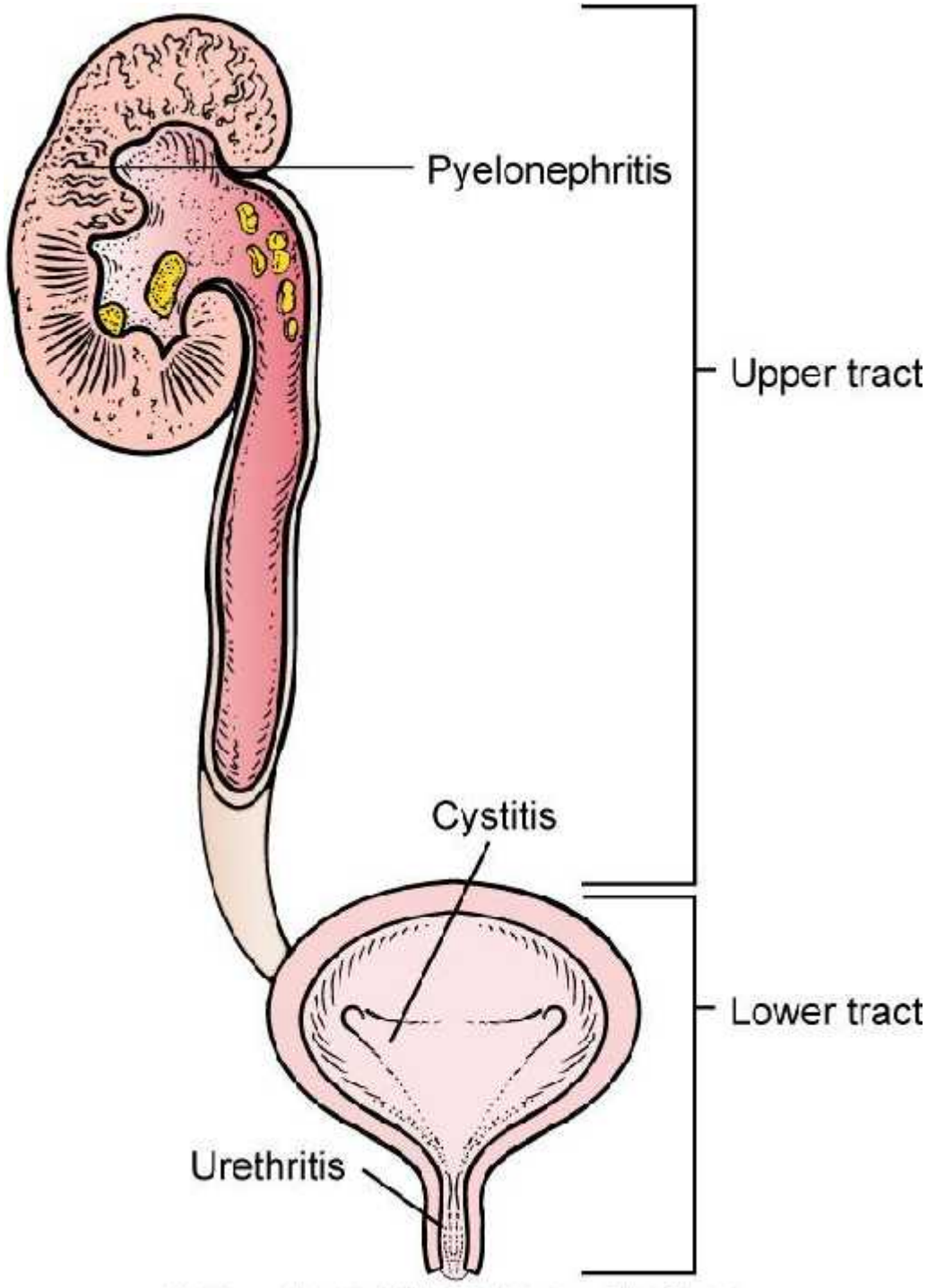
The lower urinary tract comprises of the bladder and the urethra.

CYSTITIS:

It is the infection of the bladder.

PYELONEPHRITIS:

It is the infection of the renal parenchyma



SPECTRUM OF PRESENTATION OF URINARY TRACT INFECTION

- **ASYMPTOMATIC BACTERIURIA**
- **ACUTE URETHRITIS AND CYSTITIS**
- **ACUTE PYELONEPHRITIS**
- **PROSTATITIS**
- **SEPTICEMIA**

Of the above manifestations, taking pyelonephritis into consideration

75% -sporadic pyelonephritis

25%-recurrent pyelonephritis

2%-complicated pyelonephritis

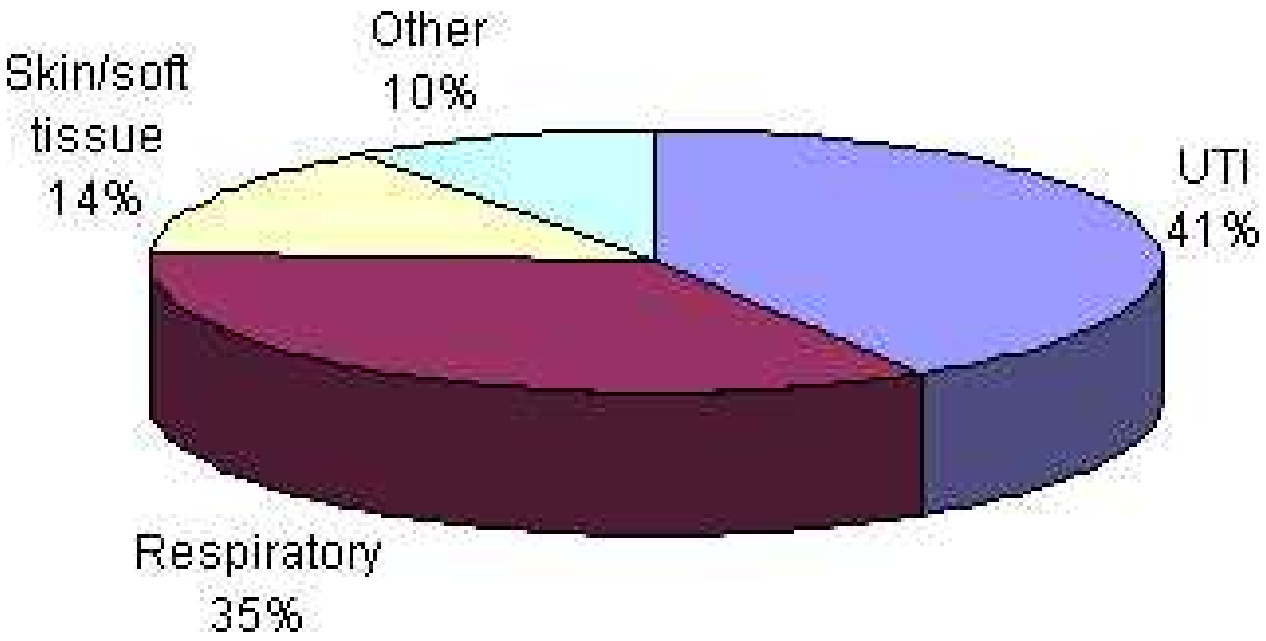
In total it is estimated that 50-80%of women would report an UTI in their lifetime.

The incidence is about 50000 per million persons per year and it accounts for 1-2% of primary care.

Pyelonephritis causes considerable morbidity and can also lead to end stage renal disease.

EPIDEMIOLOGY:

UTI is one of the most common infections occurring in human. All forms of UTI are more common in women than in men. During the neonatal period UTI is common in male because of the increased incidence of urinary tract abnormalities among male. While in the older age group, incidence of UTI is equal in both men and women. This is because of benign prostatic hypertrophy which leads to increased risk of UTI.



PREVALANCE OF BACTERIURIA:

AGE	FEMALE	MALE
INFANTS	1%	3%
SCHOOL<15 YR	1-3%	<1%
REPRODUCTIVE	4%	<1%
ELDERLY	20-30%	10%

It is stated that the prevalence of UTI in women, is about 3% at the age of 20. This prevalence tends to increase constantly by 1% every 10 years. In men the incidence of UTI is usually more in the infant period and also after the age of 60 years.

The prevalence of asymptomatic bacteriuria is estimated to be about 5% between the ages 20-40 years while this trend tends to rise to about 50% at above the age of 50 years.

It is reported that worldwide there are 150million cases of UTI per year, of which pyelonephritis account for about 10% of cases.

RISK FACTORS OF PYELONEPHRITIS:

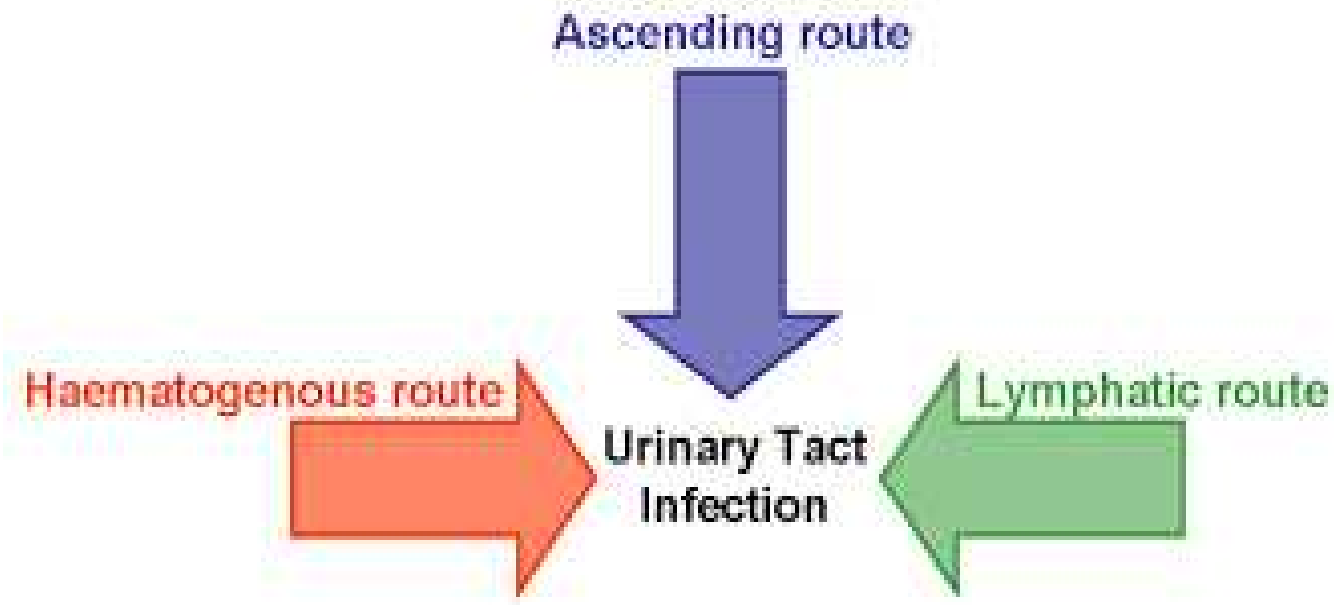
- FEMALE SEX
- EXTREMES OF AGE
- MENOPAUSAL AGE
- PREGNANCY
- DIABETES MELLITUS
- PREVIOUS OR RECURRENT PYELONEPHRITIS
- CATHETERISATION
- URINARY TRACT ANOMALIES
- FREQUENT SEXUAL ACTIVITY
- NEW SEXUAL PARTNER
- INCONTINENCE
- RENAL STONES
- NEUROLOGICAL DYSFUNTION OF BLADDER
- BLADDER OUTFLOW OBSTUCTION
    - URETHRAL STRICTURE
    - BENIGN PROSTATIC HYPERTROPY
    - VESICO URETRIC REFLUX
    - PROSTATE CANCER

All these factors are attributed to be the risk factors for pyelonephritis.

ACUTE PYELONEPHRITIS:

It is defined as the inflammation of the parenchyma and lining of the renal pelvis. Small cortical abscesses and streaks of pus are noted in the renal medulla. Histologically, there are polymorphonuclear cells which occlude the tubular lumen.

ROUTES OF INFECTION:



PYELONEPHRITIS CAN OCCUR BY 3 ROUTES:

1.**ASCENDING ROUTE:**

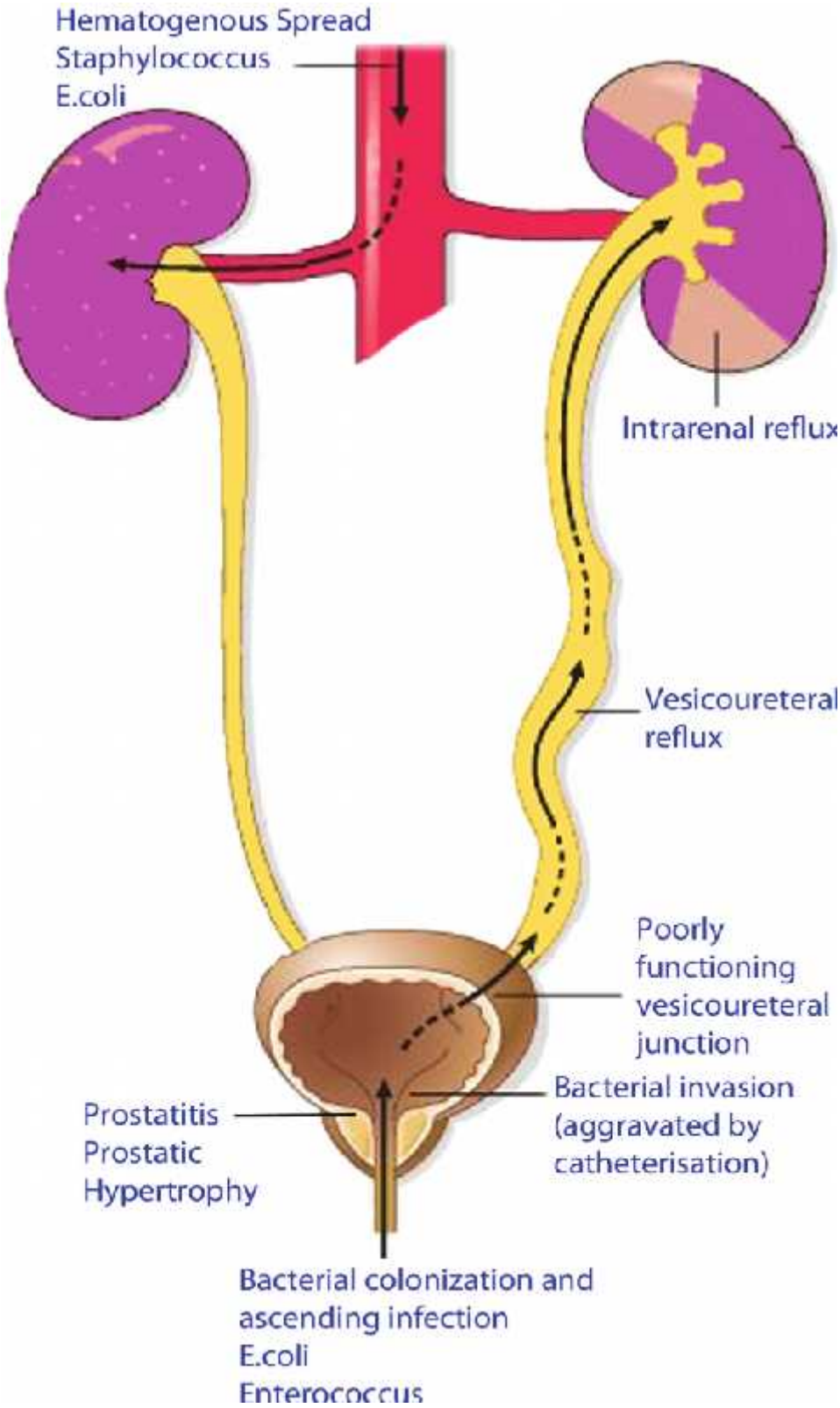
Pyelonephritis usually occurs due to retrograde infection ascending up to kidneys right from the urethra. The organisms causing pyelonephritis are those that colonize the perineum, vagina and distal urethra cause cystitis and then ascend up to cause pyelonephritis. Reflux is not essential but when present, it aggravates the infection. In conditions such as ureteric obstruction, pregnancy, endotoxins, septicemia, the ureteric peristalisis is impaired. Similarly the infection rate is also high in bacteria with P pili (fimbriae) which in turn adhere to the endothelium of the urinary tract.

2. HAEMATOGENOUS ROUTE:

This is not common but is seen to occur in infections involving Staphlyococcus aureus, Mycobacterium tuberculosis etc. Presence of candiduria (candida) is indicative of haemotogenous spread.

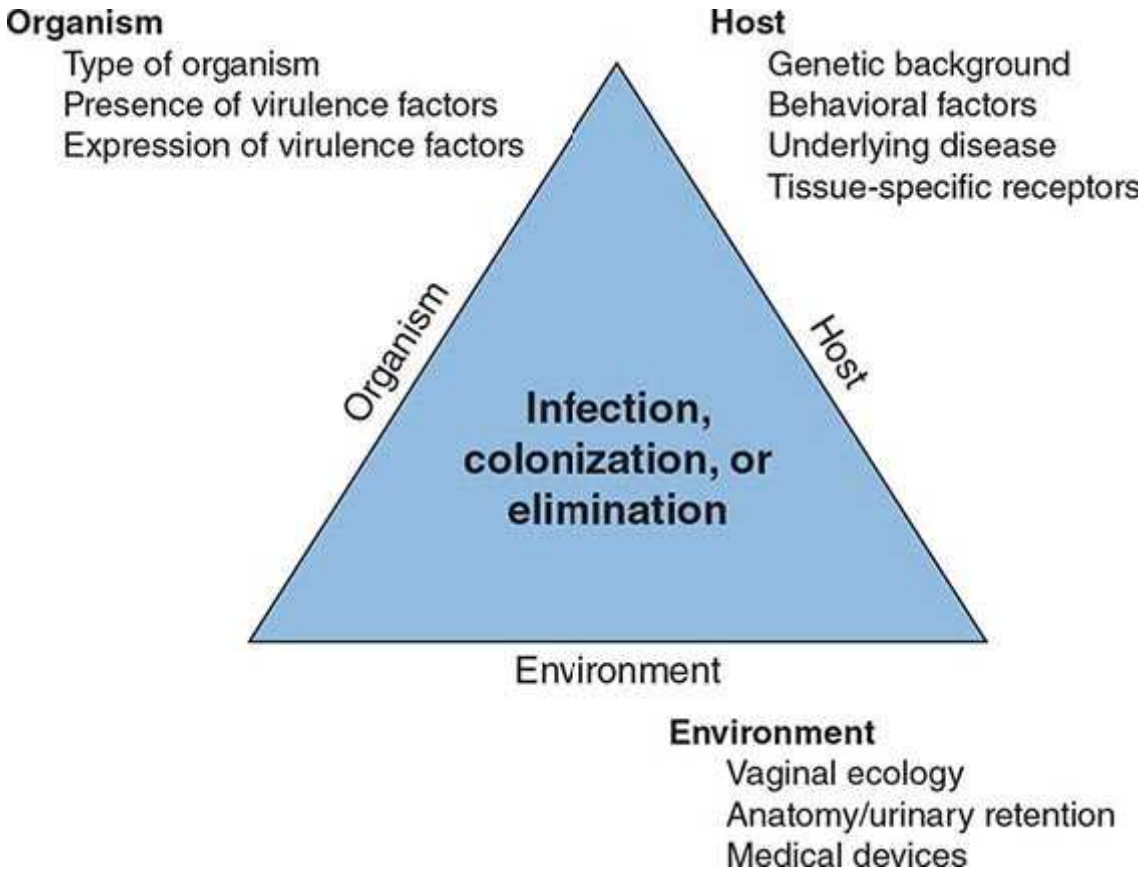
3. LYMPHATIC ROUTE:

Rare route and is seldom seen in cases such as inflammatory bowel disease, retroperitoneal abscess etc.


Hematogenous Spread
Staphylococcus
E.coli
Intrarenal reflux
Vesicoureteral reflux
Poorly functioning vesicoureteral junction
Bacterial invasion (aggravated by catheterisation)
Prostatitis
Prostatic Hypertrophy
Bacterial colonization and ascending infection
E.coli
Enterococcus

PATHOGENESIS OF PYELONEPHRITIS:

The interplay between the host, organism and environmental factors helps in the establishment of pyelonephritis.



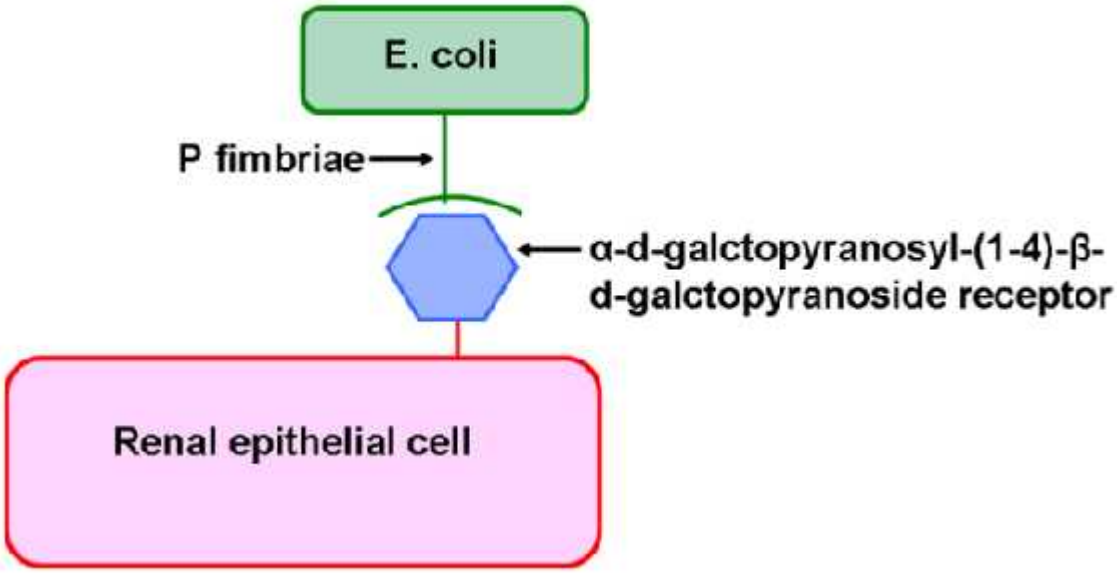
FACTORS INCREASING THE VIRULENCE OF ORGANISMS:

1. ADHESION MECHANISM:

Presence of **P fimbriae** favours the virulence of many bacteria. These pili also known as fimbriae, are found on the surface of bacteria and get attached to the urothelial cells of the host. There is about 100-400 pili in a pilated cell with a diameter of 5-10mm and a length of 2 micrometers.

The P fimbriae is known so because it can bind with the P antigen in the blood which contains D-galactose-D-galactose residue. Adhesion to the mucosal surface is mediated by the presence of various adhesions. One of the most documented adhesion is PapG adhesion which is found at the tip of P fimbriae. This serves as an important pathogenesis in pyelonephritis.

The pathogenic strains of E.coli that cause pyelonephritis produce mannose resistant pili. Type 1 pilus is mainly found in E.coli strains. Yet many strains do not express it. This is mainly indicated in cystitis. These pili initiate apoptotic signals and lead to shedding of bladder urothelial cells along with the bacteria.



2. **TOXINS:**

The host erythrocytes are directly affected by the toxins released by certain strains of E.coli.

3. **CAPSULAR PROTECTION:**

Effective phagocytosis is prevented by the presence of an extracellular capsule as in E.coli or by preventing the formation of phagolysosome as in Mycobacterium tuberculosis.

4. **ENZYMES:**

Urease is an enzyme which breaks down urea in urine to ammonia, thus resulting in the formation of struvite stone formation. This type of enzyme is produced by certain species of Proteus.

5. **ANTIMICROBIAL RESISTANCE:**

Antimicrobial resistance is produced by one of the following mechanisms.

- Production of beta lactamase enzyme: This in turn breaks open the beta lactam ring of certain antibiotics such as penicillins, carbapenems etc thus rendering these antibiotics useless.

- Genetic variation in binding site: Due to variations which take place the antigen binding sites which are the targets of antibiotic mediated therapy are altered thus leaving the organism sensitive.

HOST DEFENSE MECHANISMS:

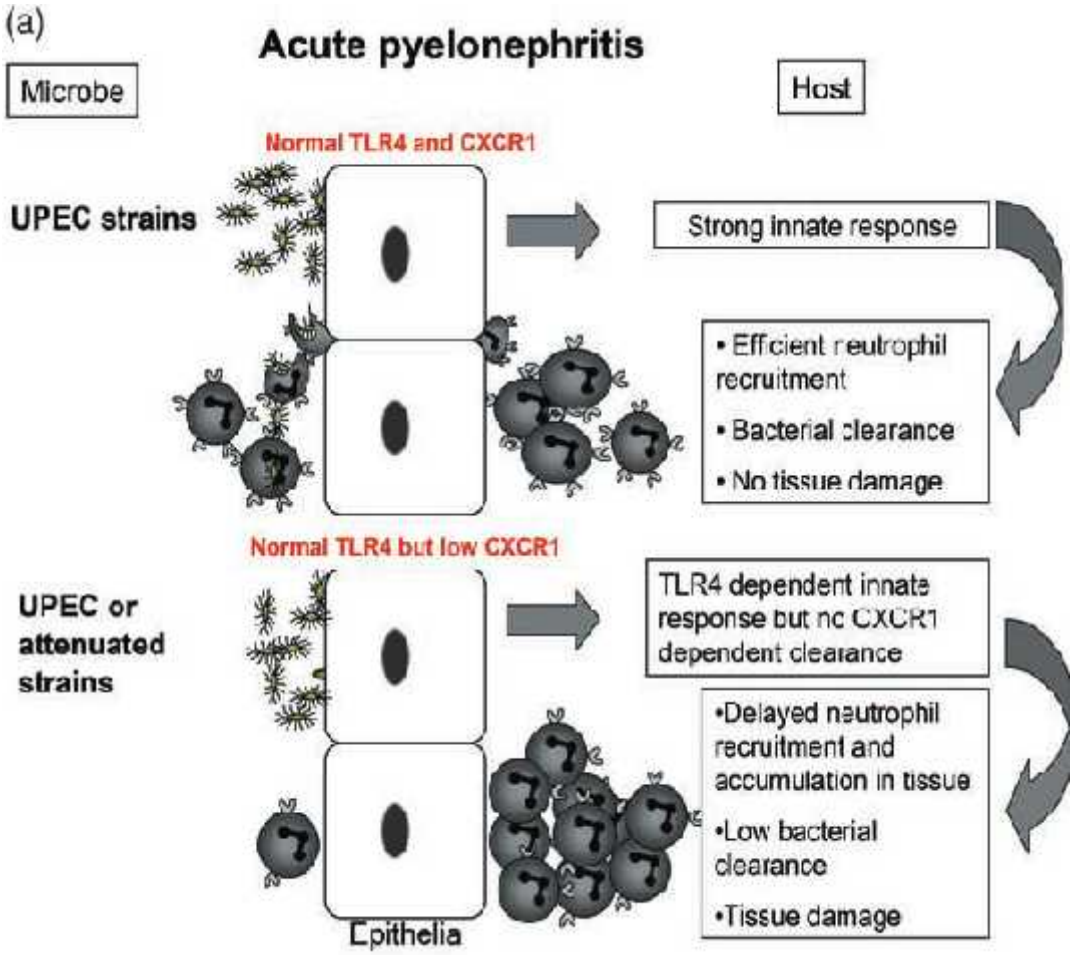
1. GENETIC SUSCEPTIBILITY:

It is found that genetic predisposition is also a contributing factor in developing UTI especially among women. Polymorphisms in CXCR1 are related to increased risk of pyelonephritis.

2. FAMILIAL HISTORY:

Familial influence is noted in developing pyelonephritis. It was found that women with a maternal history of UTI developed recurrent UTI or had developed UTI even before the age of 15 years. These susceptible women were found to have high affinity to bacteria ie) they could bind upto three fold bacteria than a woman without recurrent UTI.

It was also found that women who were non secretors of certain blood antigens such as ABH were having higher affinity towards binding of bacteria thus leading to pyelonephritis and recurrent UTI.


(a)
Acute pyelonephritis
Microbe
Host
Normal TLR4 and CXCR1
UPEC strains
Strong innate response
• Efficient neutrophil recruitment
• Bacterial clearance
• No tissue damage
Normal TLR4 but low CXCR1
UPEC or attenuated strains
TLR4 dependent innate response but no CXCR1 dependent clearance
•Delayed neutrophil recruitment and accumulation in tissue
•Low bacterial clearance
•Tissue damage
Epithelia

3.**IMMUNE RESPONSES:**

Once bacteria adhere to the urothelial cells the neutrophils get activated. The adhesion molecules activate Toll receptor 4, on the mucosal surface. This leads to release of interleukins IL-8 and the expression of its corresponding receptor CXCR1 on the surface of the neutrophils. Thus, the neutrophils get activated and aid in bacterial killing. When this entire process is disturbed by genetic alterations the resultant neutrophil activity is affected. Hence the individual becomes more susceptible to pyelonephritis (acute invasion of renal parenchyma) because of the decreased immune response mediated by the neutrophils.

4. **COMMENSAL ORGANISMS**:

Commensal organisms provide protection by the following mechanisms:

- Reduction of the pH levels
- Production of bacteriocin
- Competing for nutrients
- Stimulation of immune system

The commensals include lactobacilli, streptococci, cornyebacteria and bacteroides. Using spermicidal jelly or extensive use of antibiotics is known to destroy this natural flora thus resulting in increased incidence of pyelonephritis.

5. **URINE PH AND OSMOLALITY:**

Bacterial invasion was found to be less in conditions where the urine osmolality is >800mosm/L and in low pH states. Lactobacillus acidophilus is known to convert glycogen to lactic acid and thus produce a low pH thereby inhibiting the growth of pathogenic bacteria.

6.**URINARY FLOW:**

The anterograde flow of urine flushes out all the bacteria in the tract and thus prevents the colonization of urinary tract by bacteria.

7. **URINARY TRACT OBSTRUCTION:**

Due to indwelling catheters or stones urinary stasis occurs which leads to a biofilm formation and paves an easy towards developing pyelonephritis. On the other hand in conditions such as vesicoureteral reflex, neurogenic bladder urinary diversion surgeries and in urethral obstruction due to benign prostatic hypertrophy there is urinary stasis producing a suitable site for the growth of bacteria.

8.**VAGINAL ECOLOGY:**

The vagina gets colonized from the periurethral area and the vaginal introitus by the intestinal microflora. Sexual intercourse is associated with an increased risk of UTI. Nonoxynol is a spermicide which is known to destroy normal vaginal flora. Similarly in postmenopausal women the lactobacillus gets reduced and chances of UTI are thus increased.

9.**MUCOSA OF URINARY TRACT:**

Mucosal surface secretes substances such as lysozyme which splits out the muramic acid in the cell walls of gram positive organisms and also lactoferrin which interferes with the normal metabolism of the bacteria. The colonization is thus prevented. It is because of breech of the mucosa in urinary catheterization the way to infection begins.

10. **UROEPITHELIUM:**

The uroepithelium is known to contain mannosylated proteins such as Tomm Horsfall protein which gets attached to mucosa and a glycocalyx layer is also found covering the urothelium. These defensive substances are known to prevent bacterial adhesion.

11. **IMMUNOGLOBULINS:**

Ig A immunoglobulins are secreted by the urothelium and these are known to activate the complement system. This helps in preventing the

adherence of bacteria and thus also play a major role in the pathogenesis of pyelonephritis.

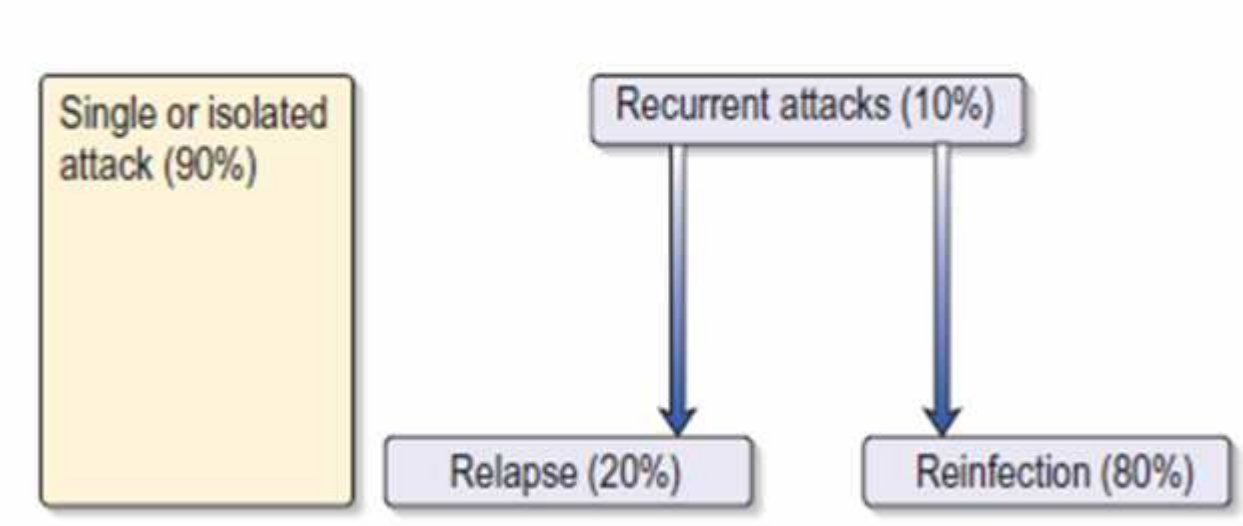
NATURAL HISTORY OF UTI:

ORGANISMS INVOLVED IN CAUSING PYELONEPHRITIS:

Organisms that cause pyelonephritis and other forms of UTI can be sorted based on community acquired or hospital acquired.

In the community acquired pyelonephritis the major causative organisms include E.coli (from gastrointestinal tract) which accounts for nearly 75% of infections. Other organisms include Pseudomonas, Proteus, Streptococci, Staphylococcus epidermidis etc.

In the hospital acquired pyelonephritis the major causative organism is again E.coli yet the other organisms which predominate include Klebsiella, and Streptococci.



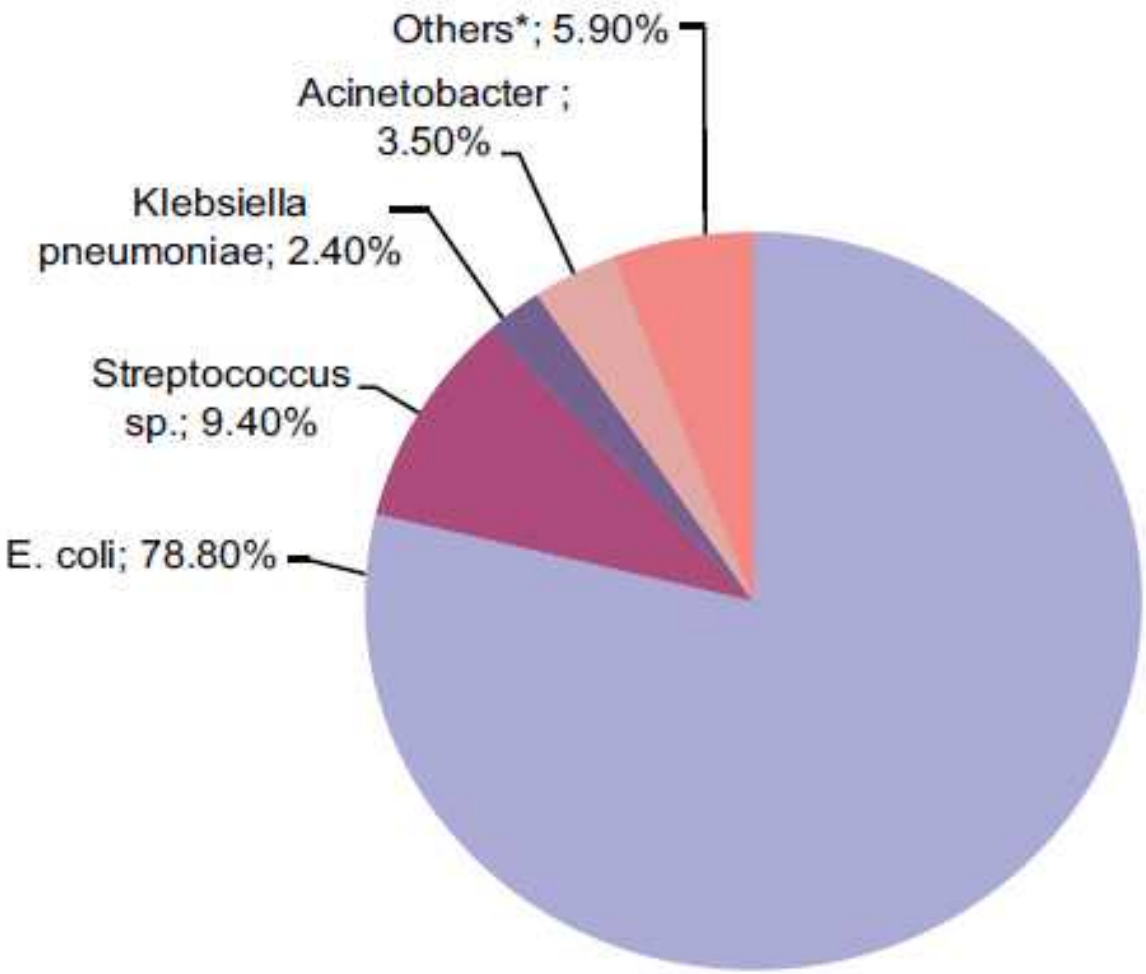
ORGANISMS WHICH CAUSE PYELONEPHRITIS AND OTHER FORMS OF UTI

Organism	Approximate frequency (%)
Escherichia coli and other 'coliforms'	68+
Proteus mirabilis	12
*Klebsiella aerogenes**	4
*Enterococcus faecalis**	6
Staphylococcus saprophyticus or *epidermidis*†	10

*More common in hospital practice
†More common in young women (20–30%)

ORGANISMS THAT CAUSE PYELONEPHRITIS				
Org	Gram	Aerobes/ anaerobes	Genus	Species
COCCI	+ve	Aerobes	Streptococci	Non hemolytic enterococcus α-hemolytic: S.viridians, β- hemolytic streptococci
			Staphylococci	S.saprophytics S.aureus S.epidermidis
	-ve	Aerobes	Neisseria	Neisseria gonorrhoea
Bacilli	+ve	Aerobes	Cornyebacteria	C. urealyticum
	Acid Fast		Mycobacterium	M. tuberculosis
	+ve	Anaerobes	Lactobacillus	L.crispatus, L.jensii Clostridium perfringens
	-ve	Aerobes	Enterobacteria	Escherichia.coli Klebsiella Proteus vulgaris
			Non fermenters	Pseudomonas aeruginosa
	-ve	Anaerobes	Bacteroides	Bacteroides fragilis
Other	organisms		Chlamydia	Chlamydiatrachomatis
			Mycoplasma	Mycoplasma hominis
			Ureaplasma	U .urealyticum
			Candida	Candida albicans

Frequency of pyelonephritis caused by various organisms in the community



CLINICAL FEATURES OF ACUTE PYELONEPHRITIS:

In case of acute pyelonephritis the patient presents with the following features:

- Fever (low grade in case of mild pyelonephritis and high grade in case of severe pyelonephritis)
- Rigors
- Flank pain
- Loin pain
- Nausea
- Vomiting
- Tenderness over the kidneys
- Bacteriuria

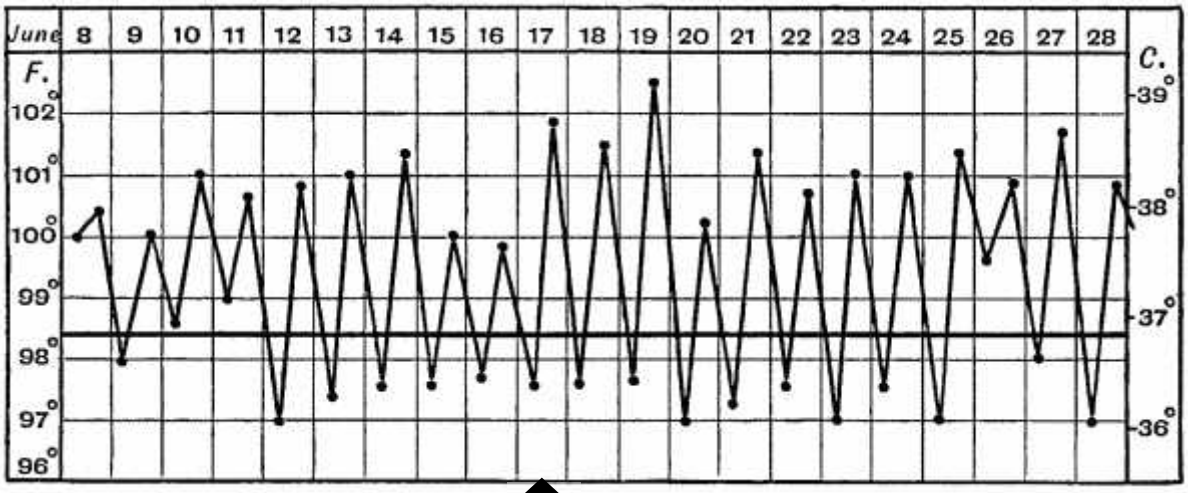
CLASSIC TRIAD OF PYELONEPHRITIS:

FEVER

LOIN PAIN

TENDERNESS OVER THE LUMBAR REGION

FEVER IN ACUTE PYELONEPHRITIS:



PICKET FENCE PATTERN

Fever in case of acute pyelonephritis has a high resolving "**PICKET FENCE"** pattern and resolves over 72 hours of therapy. Fever is the main distinguishing feature between cystitis and pyelonephritis.

ASSOCIATED SYMPTOMS IN PYELONEPHRITIS:

In case of ascending infection of the kidneys there are accompanying symptoms like

- Urgency
- Frequency
- Suprapubic pain
- Burning sensation during micturition

This is also accompanied by loin pain and fever in case of involvement of renal pelvis (pyelonephritis)

DIFFERENTIAL DIAGNOSIS:

Other conditions which mimic acute pyelonephritis include appendicitis, Cholecystitis, Pancreatitis, Diverticulitis, salpingitis, ruptured ovarian cyst, pyelonephrosis. In pyelonephrosis due to upper

urinary tract obstruction, patients may become extremely ill, with fever, leucocytosis and positive blood cultures. With a perinephric abscess, there is marked pain and tenderness, and often bulging of the loin on the affected side.

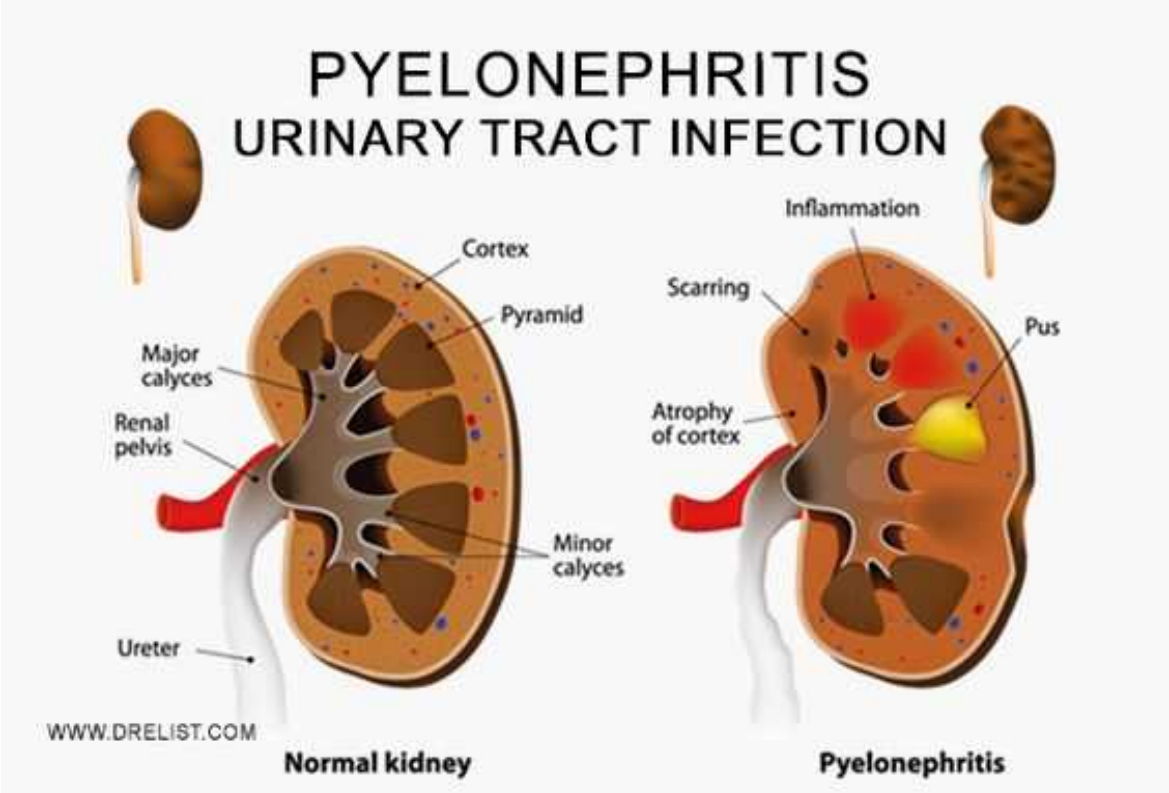
PATHOLOGY:

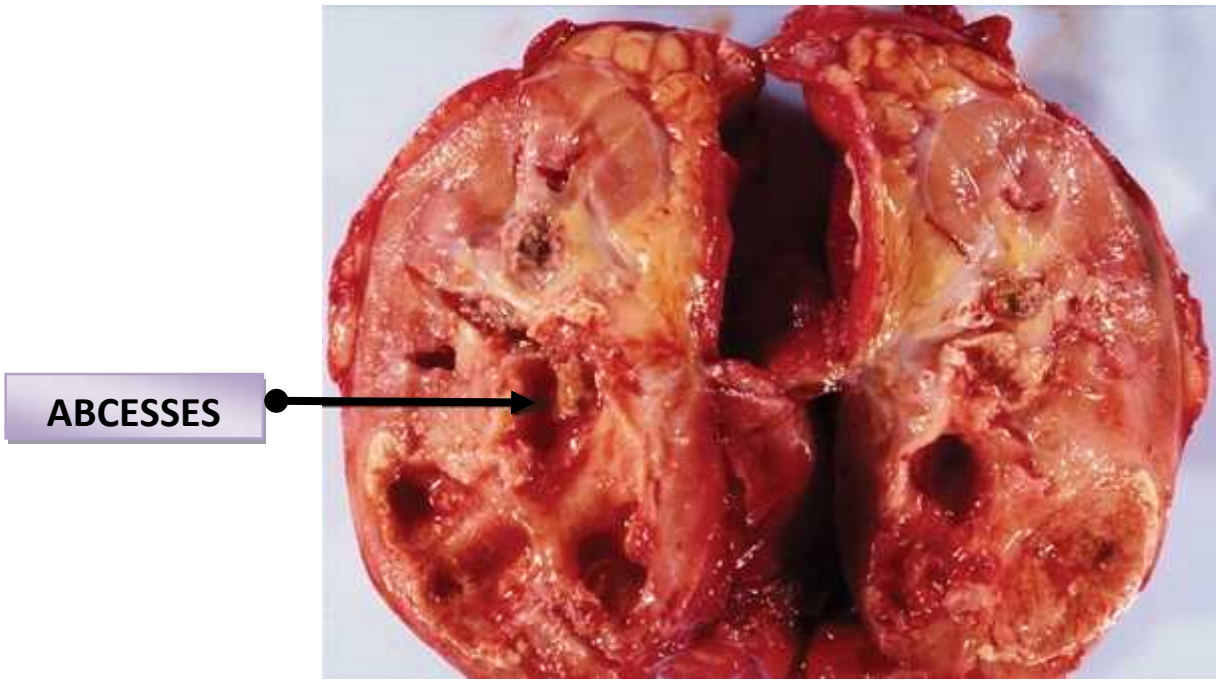
MACROSCPIC FEATURES:

The size of the affected kidneys is normal or slightly enlarged.

CUT SURFACE:

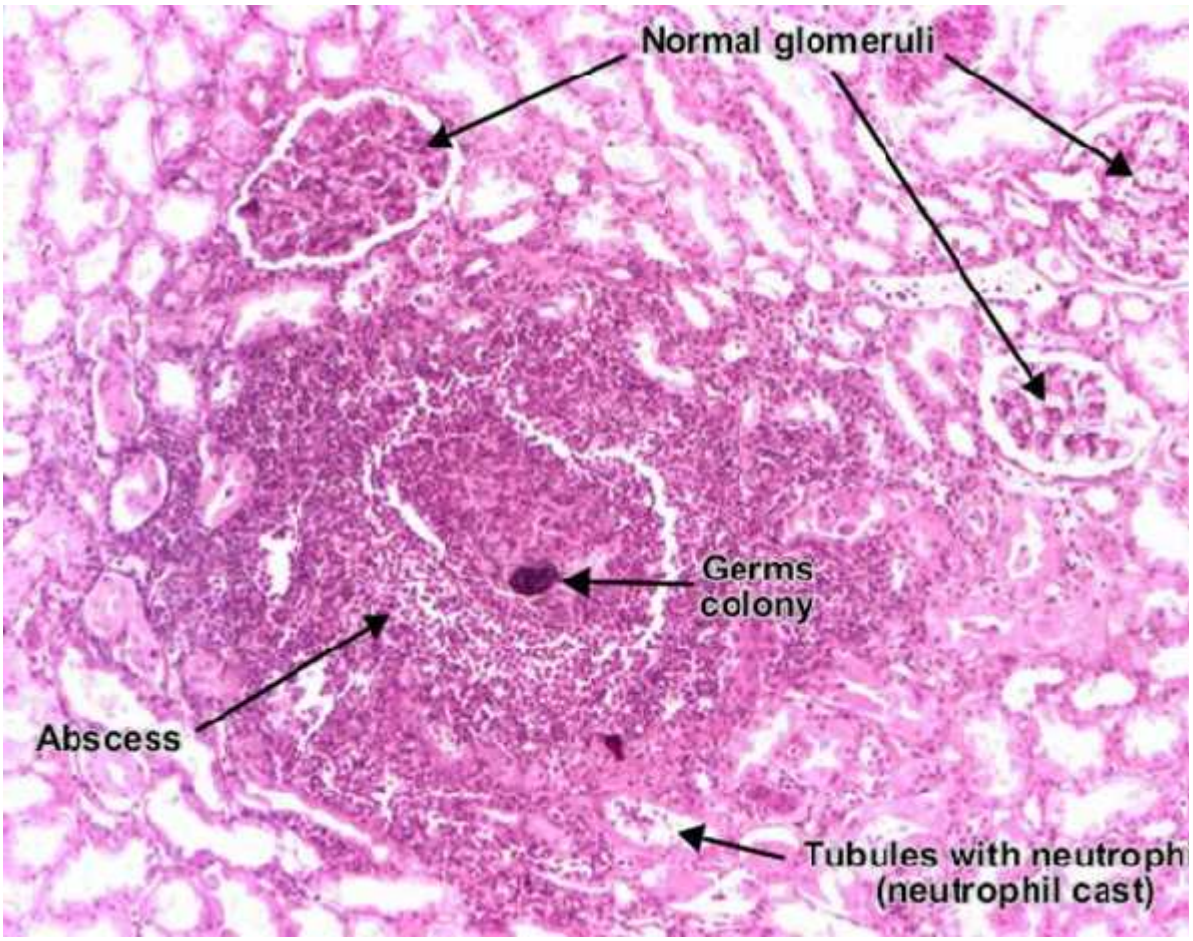
The cut surface of the kidneys show multiple isolated pale yellowish abcesses(1-5 mm in diameter) with a hyperaemic halo in the cortical area. Mucosa of the renal pelvis and calyx is congestive and covered by a purulent exudate.

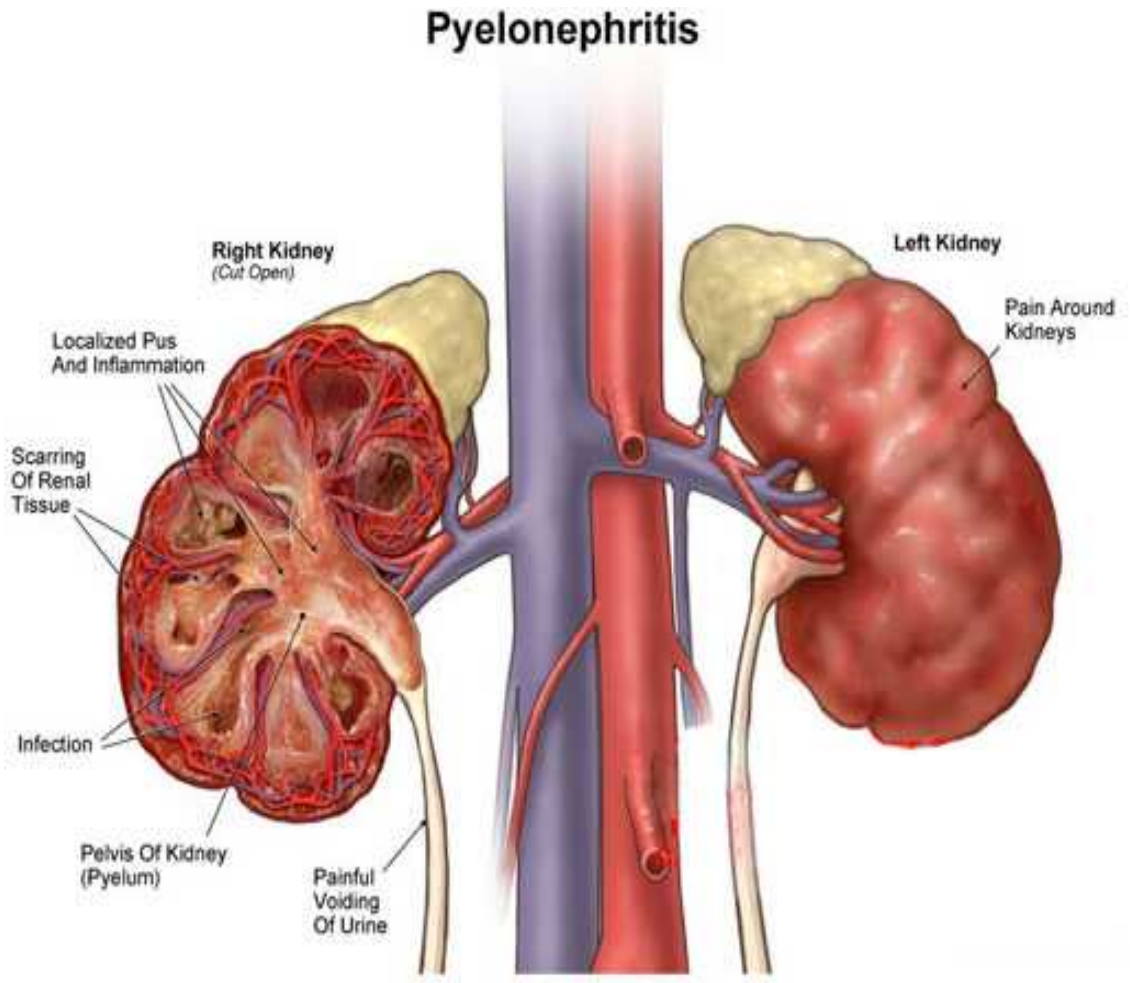




MICROSCOPY:

The renal interstitium presents abscesses (suppurative necrosis) consisting of purulent exudates which consists of neutrophils, fibrin, cell debris and central germ colonies(heamatoxylinophils). The tubules get damaged by the exudates and may contain neutrophil cast, which can be detected in the urine samples. In the early stages glomeruli and vessels are spared.


Normal glomeruli
Germs colony
Abscess
Tubules with neutroph
(neutrophil cast)


Pyelonephritis
Right Kidney
(Cut Open)
Left Kidney
Localized Pus
And Inflammation
Pain Around
Kidneys
Scarring
Of Renal
Tissue
Infection
Pelvis Of Kidney
(Pyelum)
Painful
Voiding
Of Urine

PAPILLARY NECROSIS:

Papillary necrosis is one of the manifestations of pyelonephritis. It is the necrosis of the renal papillae as a result of the decreased vascular supply to the renal tissues leading to ischaemic changes. Conditions which cause papillary necrosis include the following:

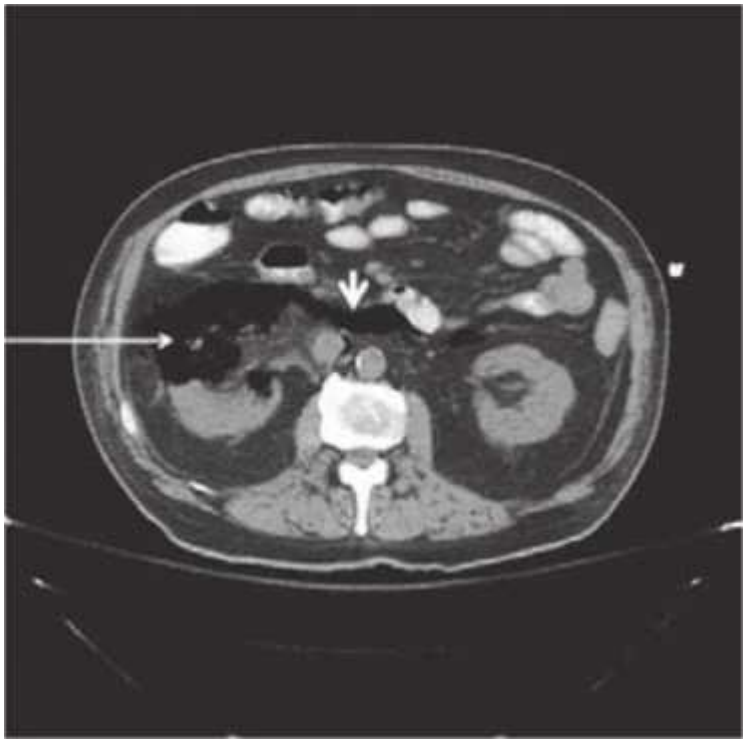
- **Pyelonephritis**
- Obstructive uropathy
- Sickle cell disease
- Tuberculosis
- Cirrhosis of the liver
- Analgesic or alcohol abuse
- Renal vein thrombosis
- Diabetes mellitus
- Systemic vasculitis

In cases of bilateral papillary necrosis the patients usually present with increase in creatinine levels. Papillary necrosis is usually more common in diabetics presenting along with obstructive uropathy. In complicated pyelonephritis there occurs sloughing of papillae that obstruct the ureters and thus lead to increased risk of pyelonephritis.

EMPHYSEMATOUS PYELONEPHRITIS:

This is one of the most severe forms of pyelonephritis in which there occurs severe necrosis of the renal parenchyma and perirenal tissues associated with the production of gas. It manifests similar to acute pyelonephritis. It is a life threatening condition and is found almost only in diabetic patients. It serves as a bad prognostic factor. The poor control of sugar levels serves as an excellent fermenting media for organisms such as E.coli. Emphysematous pyelonephritis presents in the form of severe fever and systemic features not responding with iv antibiotics.

CT ABDOMEN SHOWING EMPHYSEMATOUS PYELONEPHRITIS IN WHICH THERE IS DESTRUCTION OF RENAL PARENCHYMA (RED ARROW) AND ALSO PRESENCE OF GAS (ARROWHEAD) IN THE RETROPERITONEAL SPACE



XANTHOGRANULOMATOUS PYELONEPHRITIS:

This is a severe renal infection which occurs in conditions of chronic urinary obstruction such as renal calculi (staghorn calculus) and renal obstruction. Chronic infection sets in which leads to suppurative destruction of renal tissue. It is of three types focal, segmental, and diffuse.

Macroscopically kidneys appear enlarged and consist of yellowish nodules along with areas of hemorrhagic necrosis. Microscopically there is presence of lipid laden foamy macrophages that are found around abcesses within the parenchyma of the kidney. The most important differential diagnosis of xanthogranulomatous pyelonephritis is renal cancer.

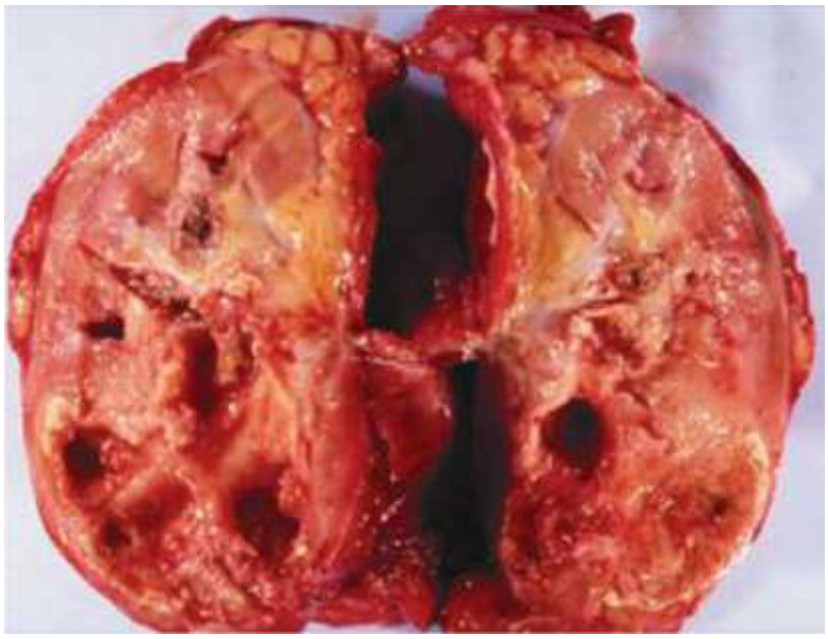
CLINICAL PRESENTATION:

Xanthogranulomatous pyelonephritis presents with flank pain, fever, hematuria, and in some with a tender flank mass.

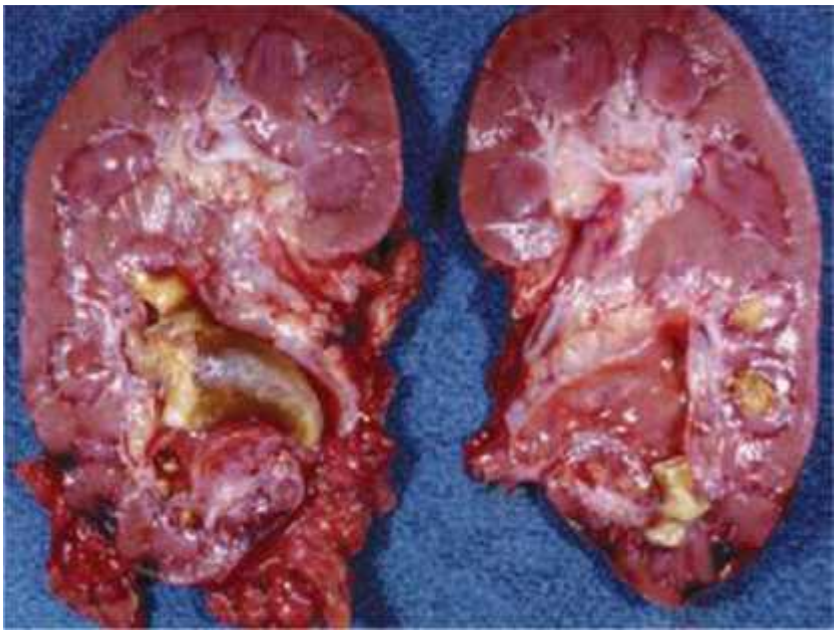
It is more common in females than in males.

COMPLICATIONS:

Paranephric abscess, psoas abscess and nephrocutaneous fistula.



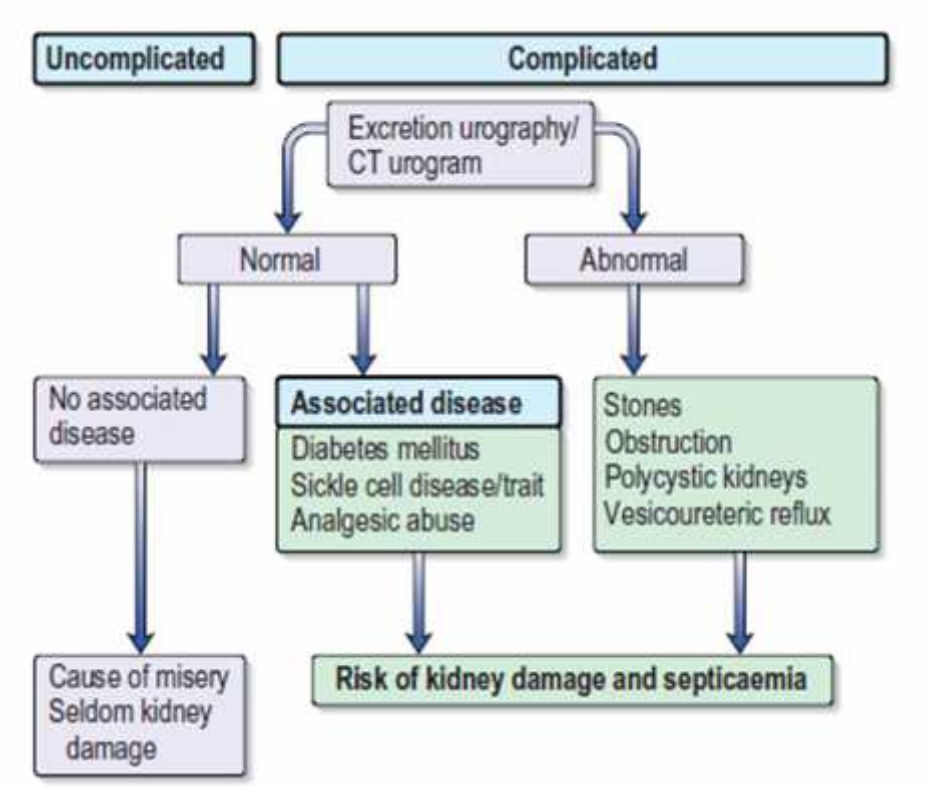
XANTHOGRANULOMATOUS PYELONEPHRITIS



PERINEPHRIC ABSCESS:

Perinephric abscess develops when the infection of acute pyelonephritis extends beyond the renal parenchyma to involve the perinephric tissues as a result of rupture of cortical abscess. This abscess develops within Gerota's fascia. Perinephric abscess can be precipitated by diabetes, immunocompromised state, obstructive ureteric calculus. This is usually caused by Staphylococcus aureus, E.coli, Proteus.

APPROACH TO COMPLICATED AND UNCOMPLICATED UTI:



INVESTIGATIONS:

Prior to investigations the tool used in the diagnosis is history. The history which is uttered by the patient has more diagnostic value than a positive urine analysis or other forms of diagnostic tests.

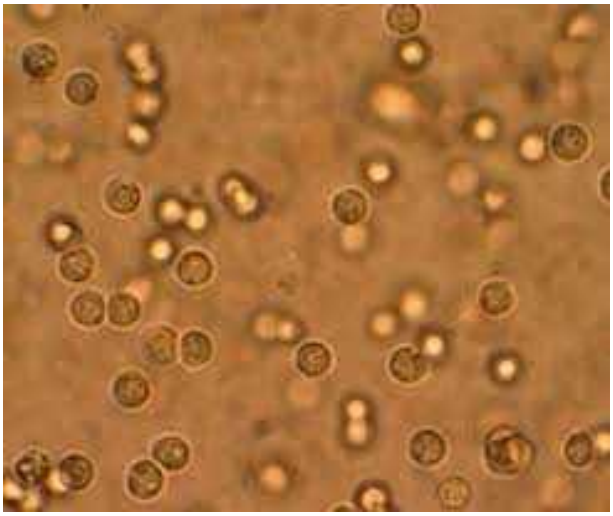
The results of a meta-analysis stated out that,

SYMPTOMS	COMPLICATING FACTORS	RISK FACTORS	PROBABILITY OF PYELONEPHRITIS
ONE SYMPTOM OF UTI	+/-	-	50%
NO SYMPTOMS	-	+	90%
DYSURIA OR URINARY FREQUENCY	-	-	96%

One of the differential diagnosis of dysuria is sexually transmitted infections when diagnosis is approached as history.

The primary investigation of choice is to do urine analysis by the use of a clean catch midstream urine sample. The next aim is the urine culture and evaluation of presence or absence of pyuria. There are numerous dipstick tests for the detection of pyuria. In these tests, pyuria is detected by the release of esterases from white blood cells. In case where detection of nitrite is the primary mechanism, reduction of nitrates to nitrites by gram negative organisms results in production of red colour in the reagent kit. It has been analysed that the reliability of diptstick tests increases when the results are positive for both nitrate and leucocyte esterase with a sensitivity of about 75% and specificity of about 82%.

PYURIA



URINE-PUS CELLS

INVESTIGATIONS FOR PATIENTS WITH PYELONEPHRITIS: (AS PER DIFFERENT CATEGORY)

ALL PATIENTS:

Dipstick estimation of nirite leucocyte esterase and glucoseMicroscopy /cytometry of urine for white blood cellsUrine culture

Infants, children and anyone with fever or complicated infection:

Complete blood count

Urea, creatinine

Electrolytes

Blood cultures

Recurrent pyelonephritis:

Renal tract ultrasound or CT

Pelvic examination in women and rectal examination in male

CRITERIA FOR DIAGNOSIS OF BACTERIURIA

Symptomatic young women

$\geq 10^2$ coliform organisms/mL urine plus pyuria (>10 WCC/mm^3)

OR

$\geq 10^5$ any pathogenic organism/mL urine

OR

any growth of pathogenic organisms in urine by suprapubic aspiration

Symptomatic men

$\geq 10^3$ pathogenic organisms/mL urine

Asymptomatic patients

$\geq 10^5$ pathogenic organisms/mL urine on two occasions

IMAGING IN PYELONEPHRITIS:

In cases of uncomplicated pyelonephritis, there is more often no need for imaging techniques. Radiological evaluation is necessary in uncomplicated pyelonephritis in conditions such as

- Recurrent infections
- Male gender
- Children
- Presence of predisposing factors such as diabetes mellitus
- Immunocompromised states

Yet in cases of complicated pyelonephritis, the need for radiological investigation gains importance. The imaging modalities used are

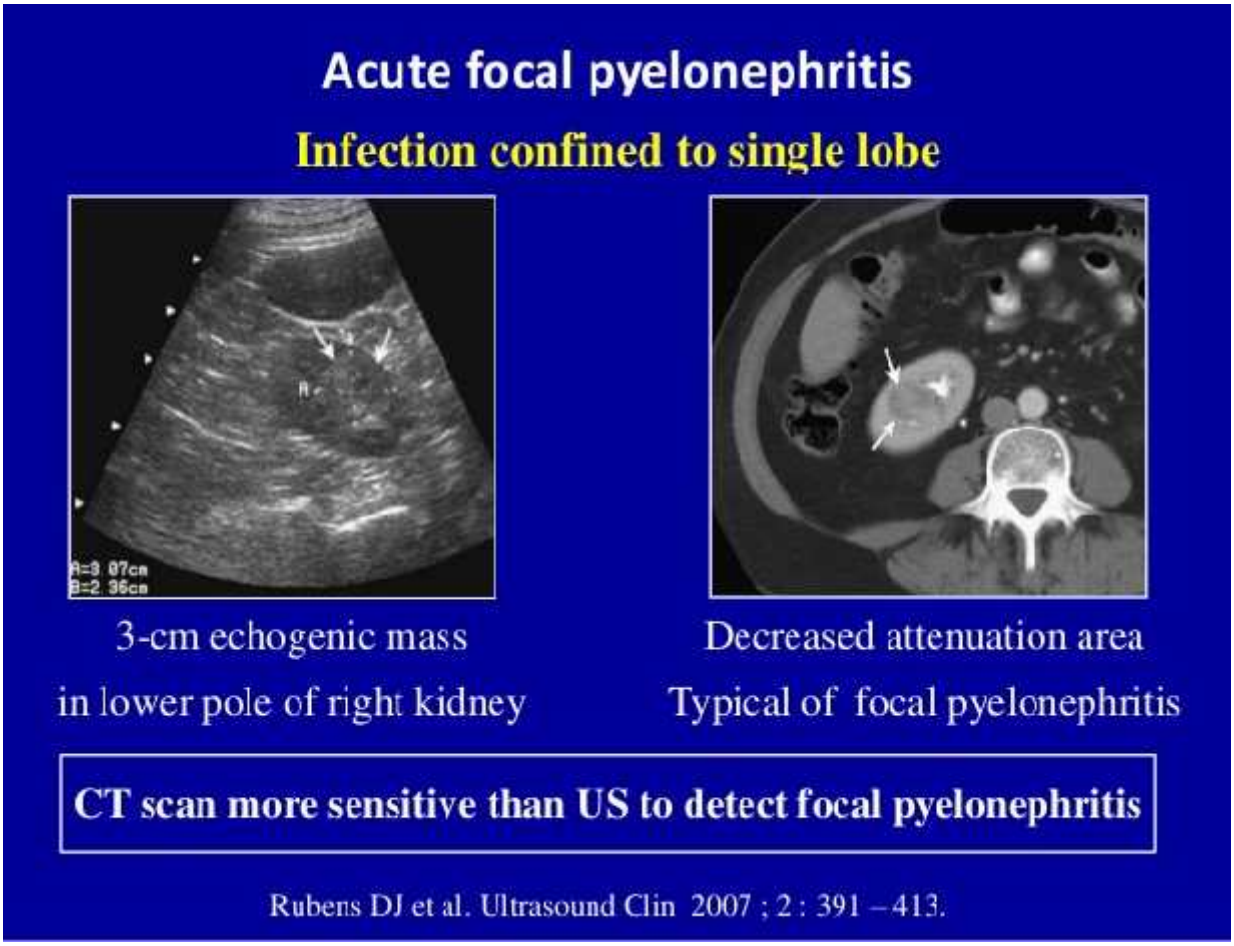
- Ultrasound abdomen
- CT abdomen (more specific is contrast enhanced CT)
- MRI
- Nuclear scans(DMSA scan)

ULTRASOUND ABDOMEN:

Ultrasound is done in patients with suspected pyelonephritis requiring drainage. Through ultrasound presence of calculi, obstruction, and also incomplete emptying could be easily diagnosed.

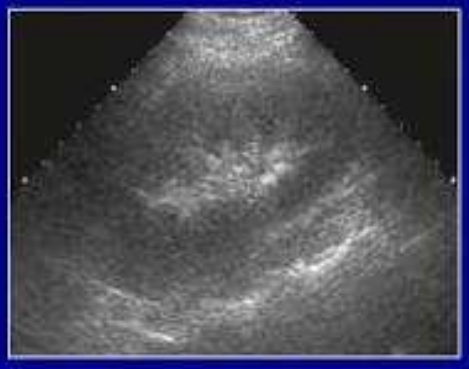
In xanthogranulomatous pyelonephritis, granuloma can be visualized along with other features of pyelonephritis.

In conditions of emphysematous pyelonephritis there is seepage of gas around the renal shadows.



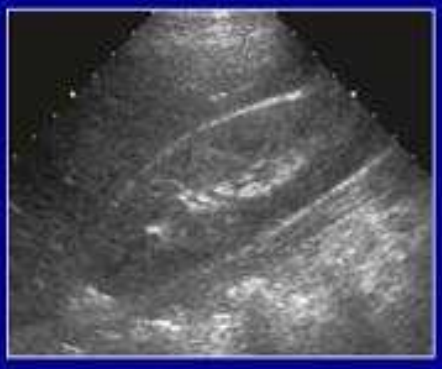
Acute pyelonephritis

Longitudinal US of left kidney



Diffuse renal enlargement
Decreased echogenicity
Loss of corticomedullary differentiation

Longitudinal US of right kidney

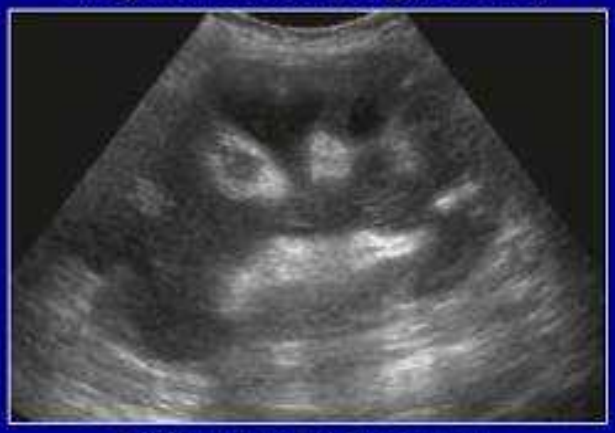


Normal right kidney

Kao HW et al. J Med Ultrasound 2008 ; 16 : 113 – 122.

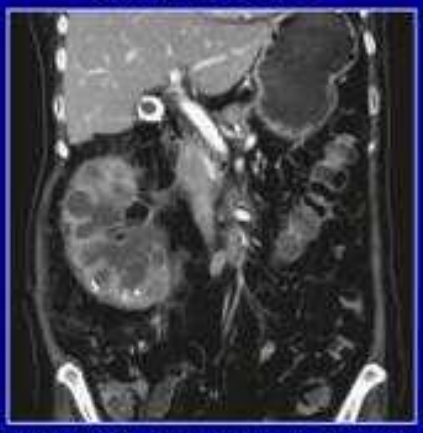
US of Xanthogranulomatous pyelonephritis

Longitudinal US of right kidney



Diffuse renal enlargement
with echogenic foci

Coronal CECT

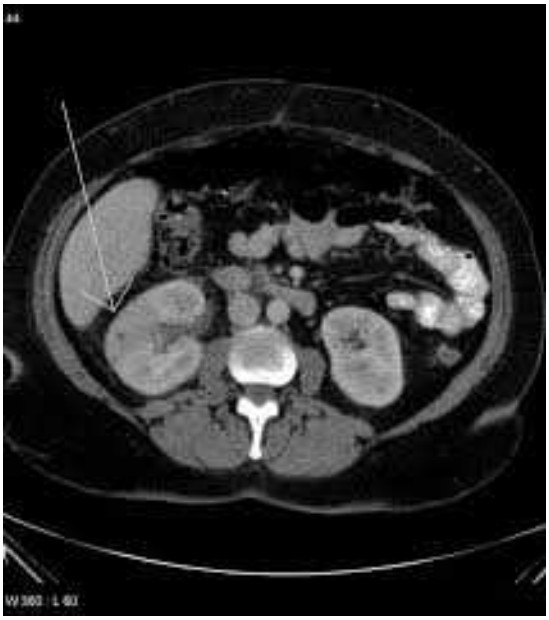


Right kidney hydronephrosis
with stones in lower pole

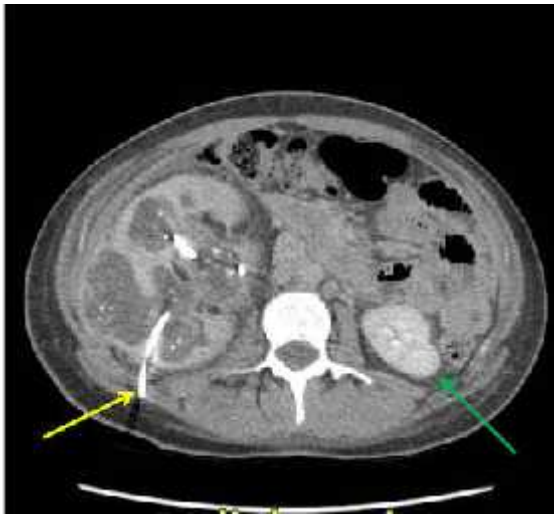
Kao HW et al. J Med Ultrasound 2008 ; 16 : 113 – 122.

CT ABDOMEN:

This is a more sensitive modality than USG abdomen. This is highly effective is cases of complicated pyelonephritis. This diagnostic modality is used to define the extent of the disease and other associated complications such as obstruction or calculi.



CT ABDOMEN SHOWING EMPHYSEMATOUS PYELONEPHRITIS



MRI ABDOMEN:

MRI abdomen is particularly useful in those with iodinatedcontrast allergies, offering an ionizing radiation-freealternative in the diagnosis of both medical and surgicaldiseases of the kidney.

NUCLEAR MEDICINE:

Nuclear medicine has a limited role in the evaluation ofUTI in adults. Its main role is in the assessment of renalfunction and detection of scars by DMSA scan, oftenprior to surgery.

TREATMENT:

Antimicrobial therapy is must for any UTI with symptoms.

Thus, treatment choices should be according to local resistance, drug availability, and individual patient factors such as recent travel and antimicrobial use.

UNCOMPLICATED CYSTITIS IN WOMEN:

Nitrofurantoin is highly effective against *E. coli* and most non–*E. coli* isolates. *Proteus*, *Pseudomonas, Serratia*, *Enterobacter*, and yeasts are all intrinsically resistant to this drug.

. Nitrofurantoin do not attain significant tissue levels and cannot be used for treating pyelonephritis. The fluoroquinolones commonly used for UTI include ofloxacin, ciprofloxacin, and levofloxacin.Quinolone use in certain populations,including adults >60 years of age, has been associated with anincreased risk of Achilles tendon rupture.

Apart from pivmecillinam, β-lactam agents generally is not that effective as TMP-SMX or fluoroquinolones in acute cystitis. The generally accepted explanation is that β-lactams fail to eradicate uropathogens from the vaginal reservoir.

PYELONEPHRITIS:

Due to high rates of TMP-SMX-resistant *E. coli* in patients with pyelonephritis fluoroquinolones still remains the first-line treatment for acute uncomplicated pyelonephritis. . A randomized clinical trial showed that

a 7-day treatment with oral ciprofloxacin(500 mg twice daily, with or without an initial IV 400-mg dose)was highly effective for the initial treatment of pyelonephritis in the outpatient set up.

Oral TMP-SMX (one double-strength tablet twice daily for 14 days) also is effective for treatment of acute uncomplicated pyelonephritis if the micro-organism is known to be susceptible. If the pathogen's susceptibility is not known and TMP-SMX is started, an initial IV 1-g dose of ceftriaxone is needed.

Choices for parenteral therapy for uncomplicated pyelonephritis include fluoroquinolones, an extended-spectrum cephalosporin along with or without an aminoglycoside, or a carbapenem.

Combinations of a β-lactam and a β-lactamase inhibitor (e.g., ampicillin-sulbactam, ticarcillin -clavulanate, piperacillin-tazobactam) or imipenem-cilastatin can beused in patients with more complicated histories, previous episodes of pyelonephritis, or recent urinary tract instrumentation; in general, the treatment of such patients should be according to urine culture results. Once the patient has improved clinically, oral therapy

should be substituted for parenteral therapy.

UTI IN PREGNANT WOMEN:

Nitrofurantoin, ampicillin, and the cephalosporins are found relatively safe in early pregnancy.

Fluoroquinolones are not used because of possible side effects on fetal cartilage development. Ampicillin and cephalosporins have been used extensively in pregnancy and are the therapy of choice for the treatment of asymptomatic or symptomatic UTI in pregnant patients. For pregnant women with overt pyelonephritis, parenteral β-lactam therapy along with or without aminoglycosides is the standard line of treatment.

UTI IN MEN:

Since the prostate is involved in the most of the cases of febrile UTI in men, the aim in these patients is to eradicate the prostatic infection as well as the bladder infection. A 7- to 14-day course of a fluoroquinolone or TMP-SMX is recommended if the micro-organism is susceptible.

For documented chronic bacterialprostatitis, a 4- to 6-week course of antibiotics is often necessary.

Recurrences, which is usual in chronic prostatitis, often need a 12-week course of therapy.

COMPLICATED UTI:

Complicated UTI occurs in a heterogeneous class of patients with various structural and functional anomalies of the urinary tract and kidneys. As a result, treatment for complicated UTI must be individualized and guided by urine culture results. Xanthogranulomatous pyelonephritis is treated with nephrectomy. Percutaneous drainage can be used as the starting therapy in emphysematous pyelonephritis and can be proceeded to elective nephrectomy if needed. Papillary necrosis with obstruction needs intervention to remove the obstruction and to coserve renal function.

ASYMPTOMATIC BACTERIURIA (ASB):

Treatment of ASB in pregnant women and patients with urologic procedures must treated according to urine culture results. In rest of other populations, screening for and treatment of ASB should not be encouraged.

CATHETER-ASSOCIATED UTI (CAUTI):

Various institutions have given guidelines for the treatment of CAUTI, which is defined by bacteriuria and symptoms in a catheterized patient. The accepted threshold for bacteriuria to meet the definition of CAUTI is $\geq 10^3$ CFU/mL, while the threshold for bacteriuria to meet the definition of ASB is $\geq 10^5$ CFU/mL.

In general, a 7- to 14-day courseof antibiotics is often needed.. The best modality for prevention of CAUTI is to avoid insertion of unnecessary catheters and to take away catheters once they are not needed.

CANDIDURIA:

Removing unnecessary in-dwelling catheters is definitely helpful.

Fluconazole (200–400 mg/d for 14 days) reaches high levels in urine and is the first-line regimen for *Candida* infections of the urinary tract. For *Candida* isolates with high levels of resistance to fluconazole, oral flucytosine and/or parenteral amphotericin B are options.

PREVENTION OF RECURRENT UTI IN WOMEN:

Three prophylactic strategies are used: continuous, postictal, and patient-initiated therapy. Continuous prophylaxis and postcoital prophylaxis usually involve using low doses of TMP-SMX, a fluoroquinolone, or nitrofurantoin., a prophylactic regimen is given for 6 months and stopped.

PROGNOSIS:

In the presence of no anatomic abnormalities, recurrent infection in children and adults does not lead to chronic pyelonephritis or to renal failure. In spinal cord–injury patients, use of a longterm indwelling bladder catheter is an important risk factor for bladder cancer.

MATERIALS AND METHODS

In our study done from june 2016 –june 2017 in Tirunelveli medical college,50 patients with pyelonephritis with classical clinical features of fever ,chills & flank pain & tenderness, with radiological evidence of acute pyelonephritis were studied. Associated co-morbidities like DM, urolithiasis were noted. Blood investigations CBC, Serial RBS,FBS & PPBS ,RFTS.S.Electrolytes,LFT,HBA1C,Urine albumin, sugar, deposits acetone (if needed) & c/s done.

Radiological imaging USG abdomen & KUB, CT scan abdomen taken patients prospectively then followed for a period of 2 months & factors determining the prognosis of patients studied. Patients who recovered with antibiotics ,percutaneous nephrostomy & other supportive measures were considered to have good prognosis .Those who required nephrectomy or died were considered to have poor prognosis.

INCLUSION CRITERIA:

All patients admitted to Tirunelveli medical college & hospital with acute pyelonephritis during the study period june2016- June 2017.

EXCLUSION CRITERIA:

Terminally ill patients

LIMITATIONS OF THE TEST:

Small sample size

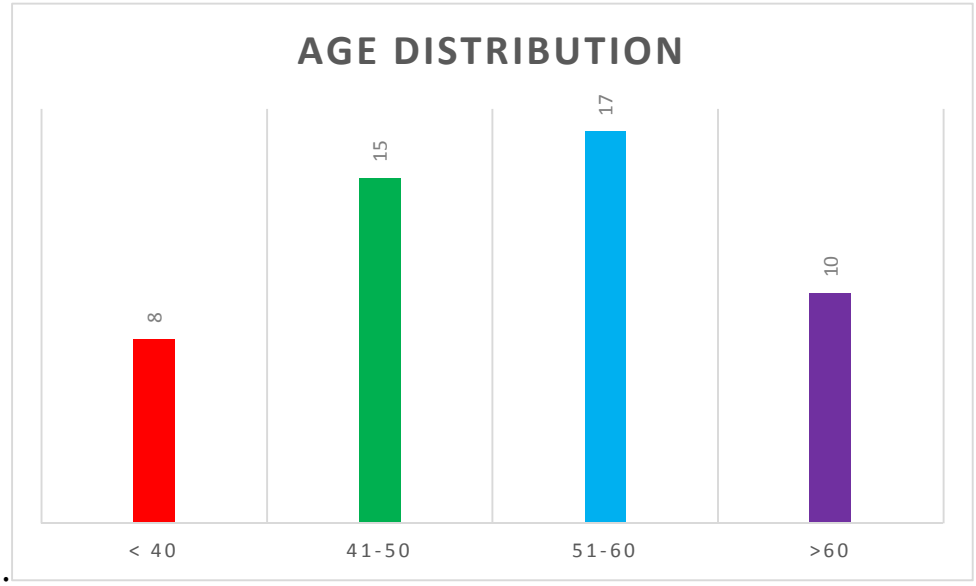
OBSERVATION & RESULTS

AGE DISTRIBUTION

Table 1

AGE(IN YEARS)	NO OF PATIENTS	PERCENTAGE
< 40	8	16%
41-50	15	30%
51-60	17	34%
>60	10	205

Chart 1



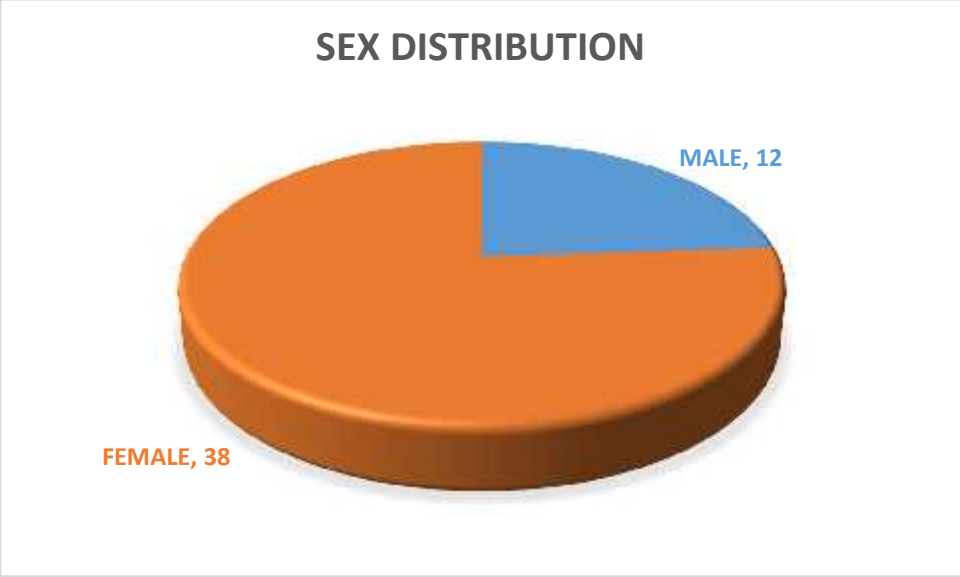
34% of patients belonged to the age group 51-60 years

SEX DISTRIBUTION

Table 2

SEX	NO OF PATIENTS	PERCENTAGE
MALE	12	24%
FEMALE	38	76%

Chart 2

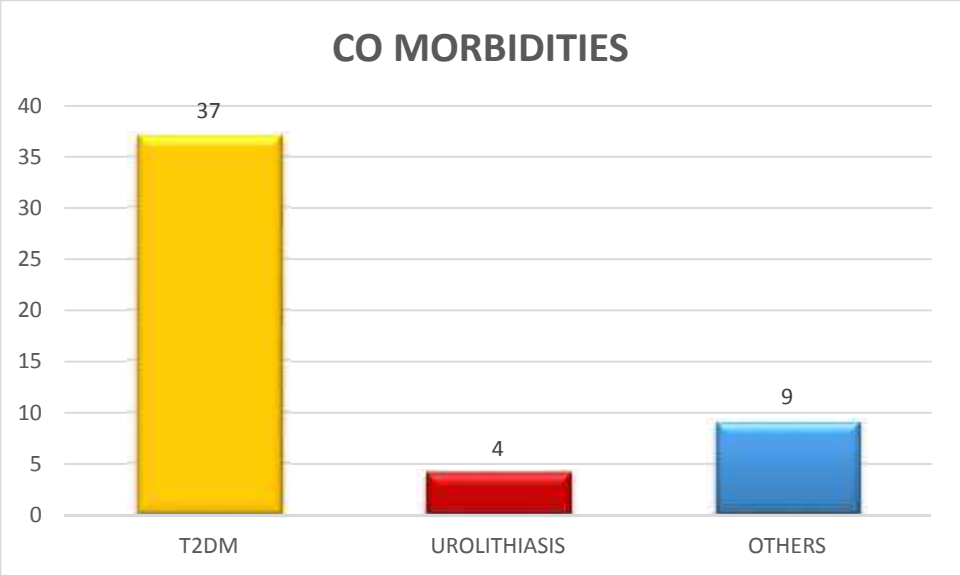


38 patients were females,12 were males

Table 3

CO MORBIDITIES	NO OF PATIENTS	PERCENTAGE
T2DM	37	74%
UROLITHIASIS	4	8%
OTHERS	9	18%

Chart 3



37 (74%)patients were diabetics, & 4(8%) had urolithiasis.

Table 4

SYMPTOMS	NO OF PATIENTS	PERCENTAGE
FEVER	41	82%
ABD PAIN	32	64%
DYSURIA	28	56%
PEDAL EDEMA	3	6%

Chart 4

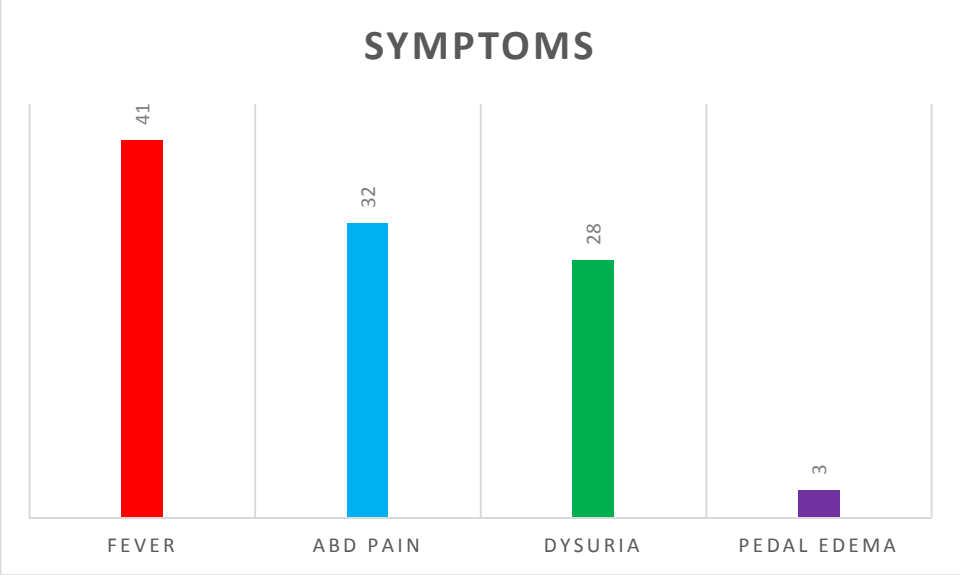
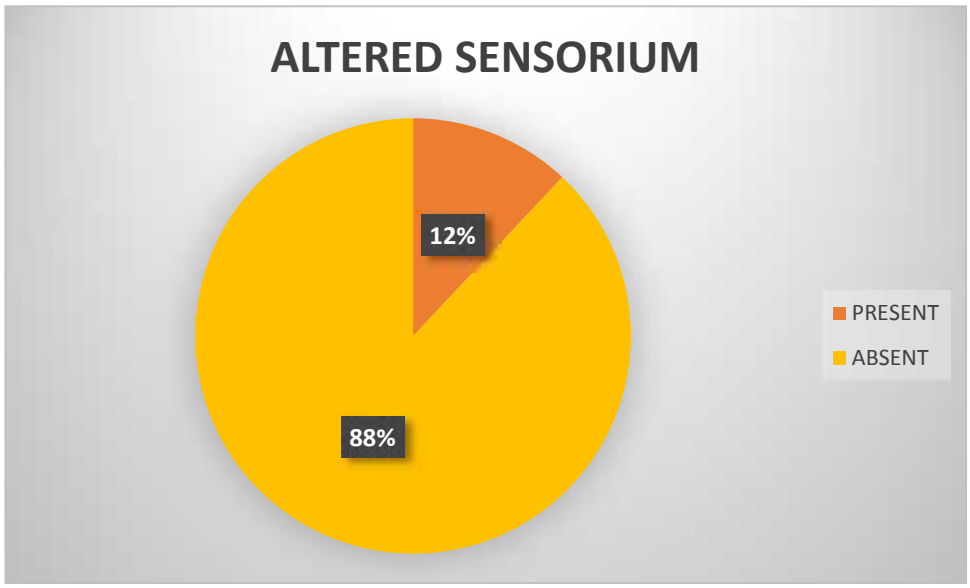


Table 5

ALTERED SENSORIUM	NO OF PATIENTS	PERCENTAGE
PRESENT	6	12%
ABSENT	44	88%

Chart 5

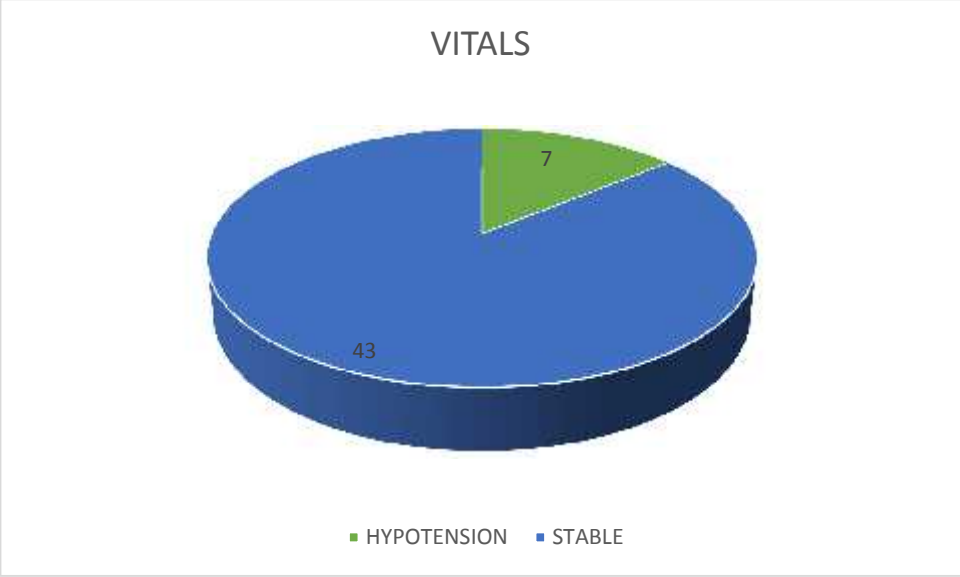


Altered sensorium present in 12 % of patients.

Table 6

VITALS	NO OF PATIENTS	PERCENTAGE
HYPOTENSION	7	14%
STABLE	43	86%

Chart 6

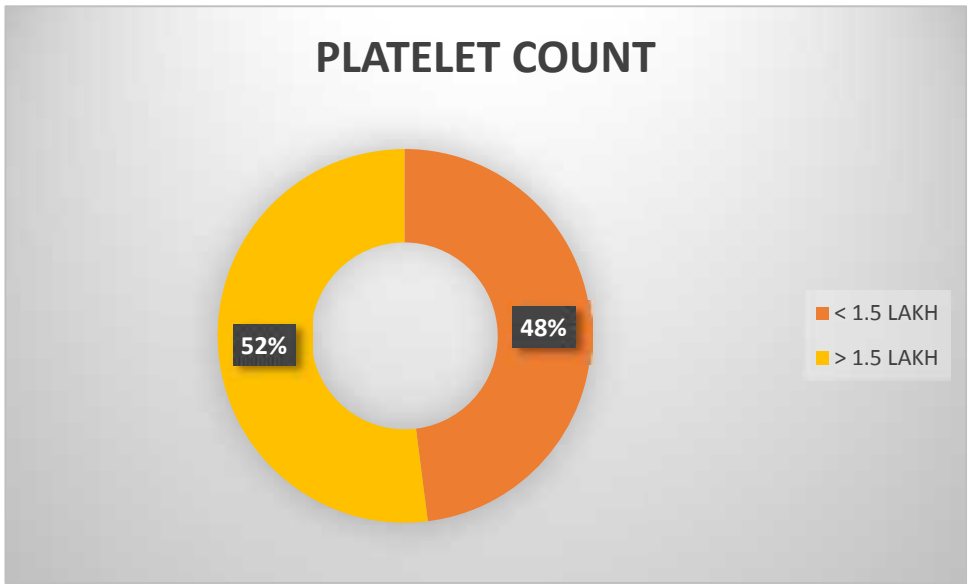


Hypotension present in 7(14%) patients.

Table 7

PLATELET COUNT	NO OF PATIENTS	PERCENTAGE
< 1.5 LAKH	24	48%
> 1.5 LAKH	26	52%

Chart 7



48% of patients had thrombocytopenia.

Table 8

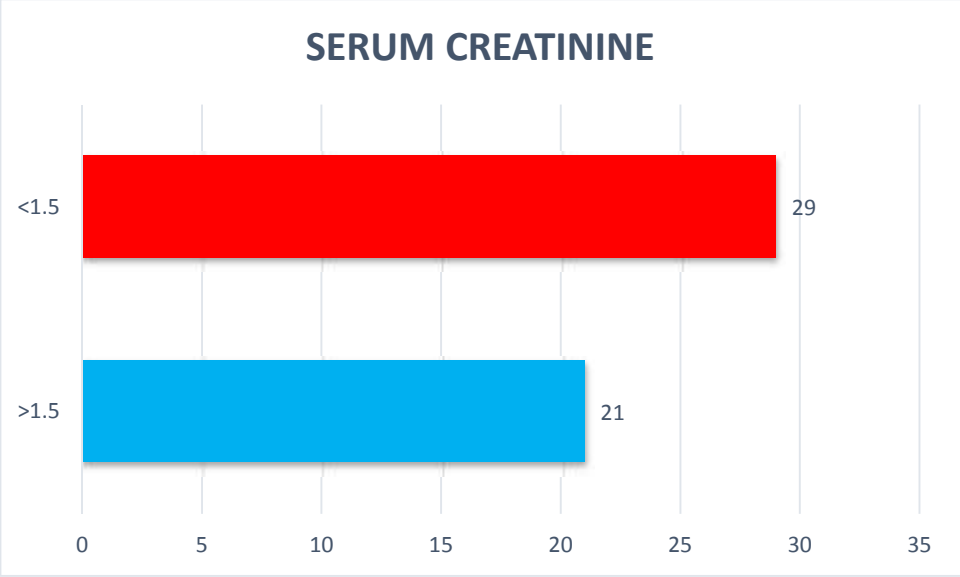
HBA1C	NO OF PATIENTS	PERCENTAGE
> 7.5 %	21	42%
<7.5 %	29	58%

29(58%) patients had poor glycaemic control.

Table 9

CREATININE	NO OF PATIENTS	PERCENTAGE
>1.5	21	42%
<1.5	29	58%

Chart 8



29(58%) patients had renal dysfunction.

Table 10

LFT	NO OF PATIENTS	PERCENTAGE
ALTERED	6	12%
NORMAL	44	88%

6 (12%) had altered LFT.

Chart 9

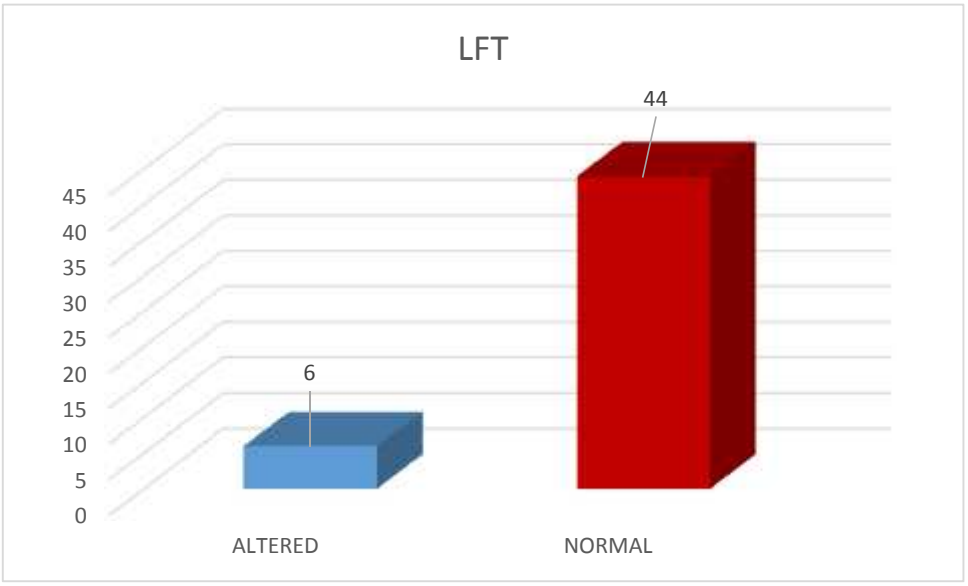
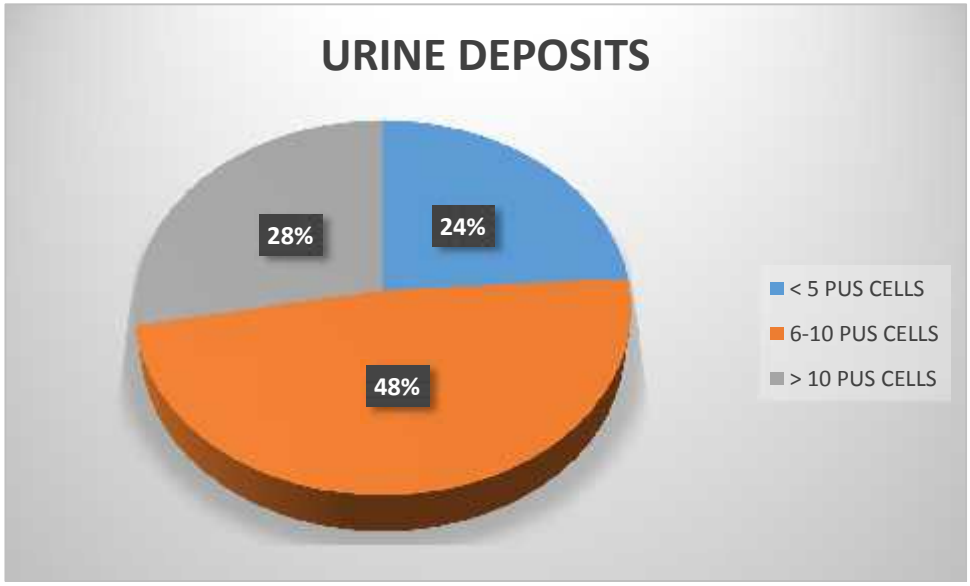


Table 11

URINE DEPOSITS	NO OF PATIENTS	PERCENTAGE
< 5 PUS CELLS	12	24%
6-10 PUS CELLS	24	48%
> 10 PUS CELLS	14	28%

Chart 10



48 % had pus cells 6-10.

Table 12

URINE CULTURE	NO OF PATIENTS	PERCENTAGE
GROWTH +VE	22	44%
GROWTH - VE	28	56%

Urine culture showed positive growth in 44 % patients

Chart 11

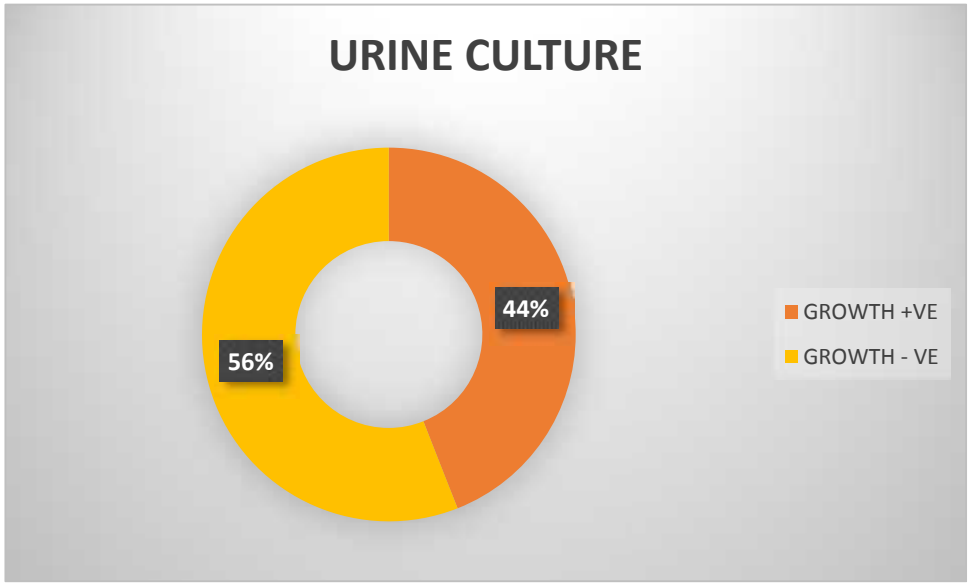


Table 13

ORGANISM GROWN(N-22)	NO OF PATIENTS	PERCENTAGE
E.COLI	16	72.70%
KLEBSIELLA	4	18.30%
PSEUDOMONAS	1	4.50%
FUNGAL SPECIES	1	4.50%

E.COLI CULTURED IN 16(72.7%), KLEBSIELLA IN 4(18.3%),PSEUDOMONAS & FUNGAL SPECIES IN 1(4%) PATIENT

Chart 12

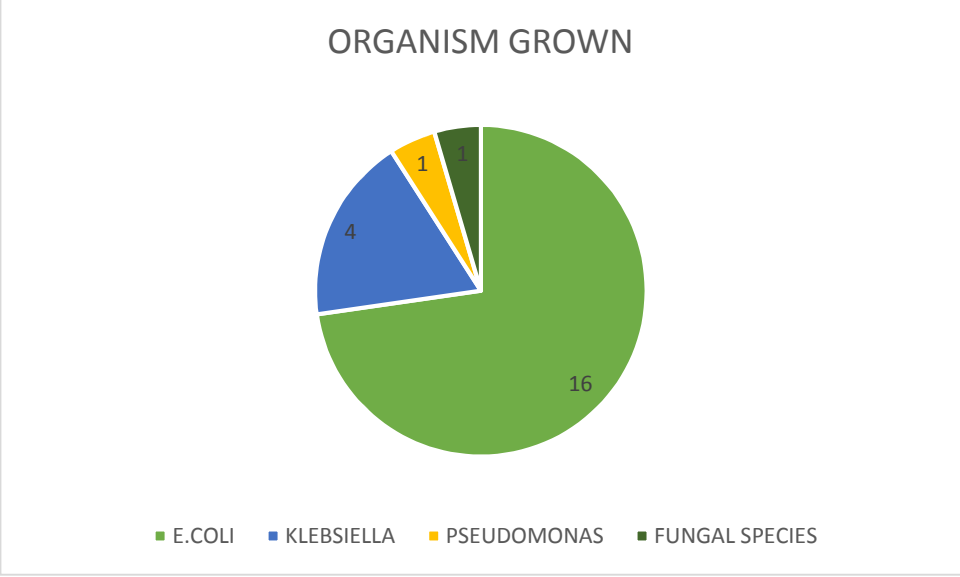
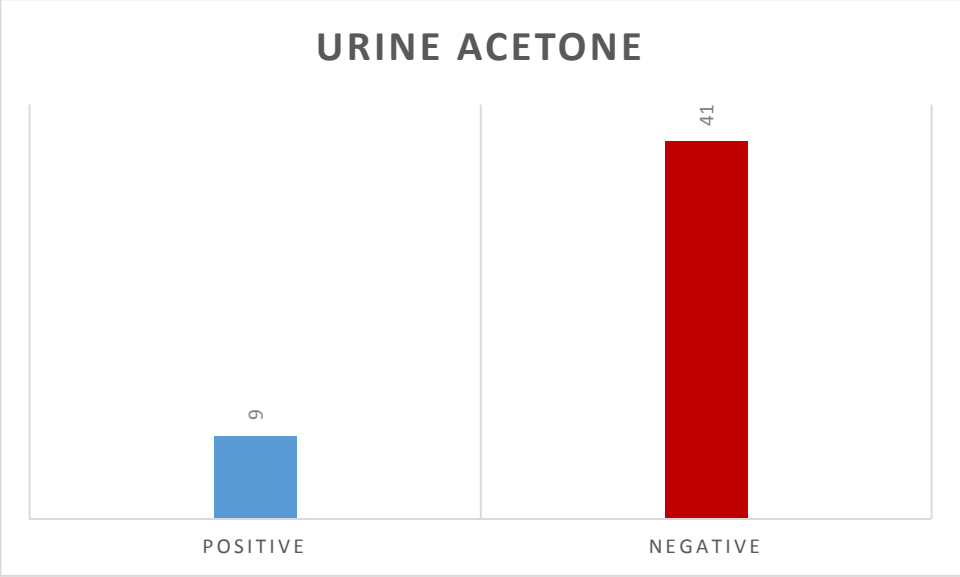


Table 14

URINE ACETONE	NO OF PATIENTS	PERCENTAGE
POSITIVE	9	18%
NEGATIVE	41	82%

Chart 13



Urine acetone positive in 9 patients.

Table 15

USG & CT	NO OF PATIENTS	PERCENTAGE
HIGH RISK	12	24%
LOW RISK	38	76%

High risk – B/L Pyelonephritis, Emphysematous pyelonephritis, Papillary necrosis, Renal abscess –present in 12 (24%) patients.

Chart 14

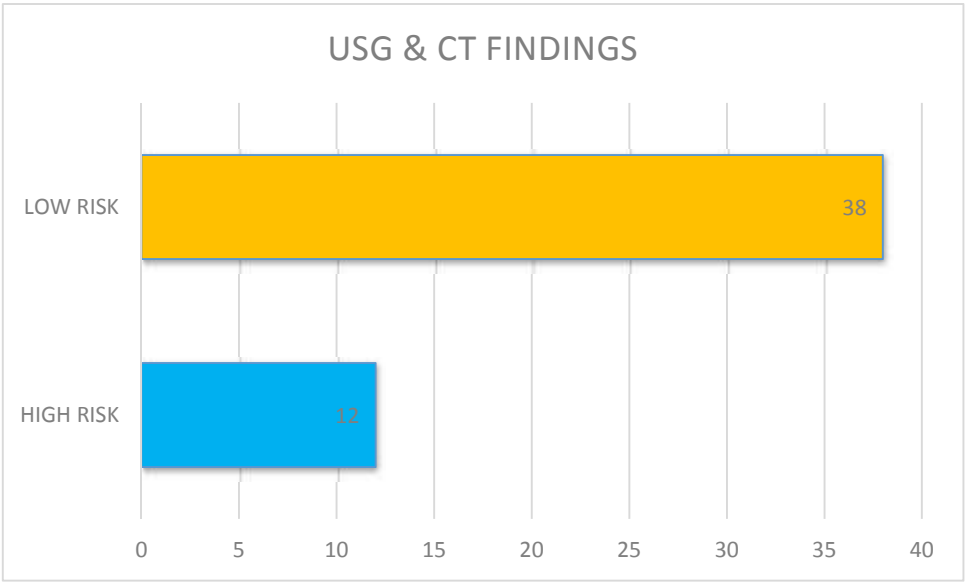
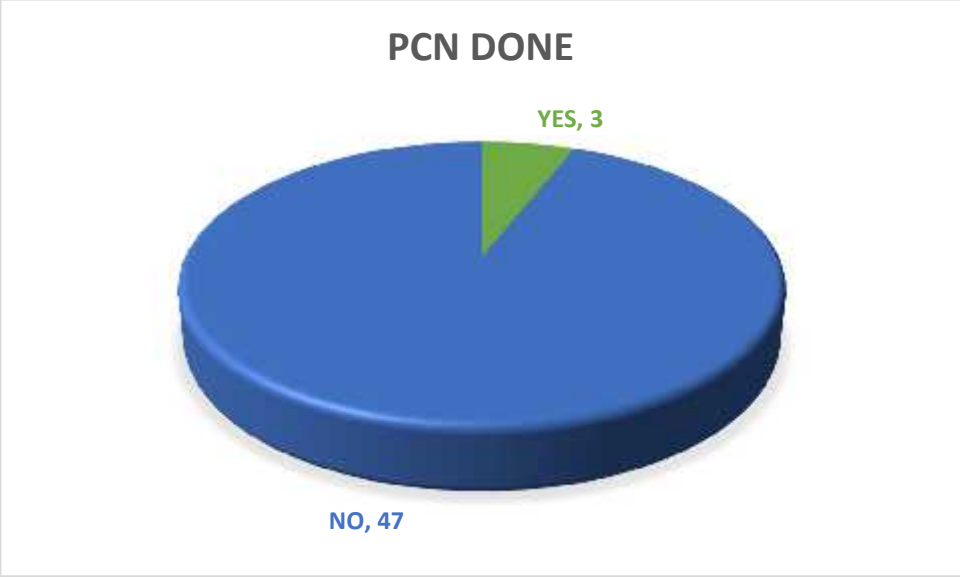


Table 16

PCN DONE	NO OF PATIENTS	PERCENTAGE
YES	3	6%
NO	47	94%

Chart 15



PERCUTANEOUS NEPHROSTOMY DONE IN 3 (6%)PATIENTS

Table 17

PROGNOSIS	NO OF PATIENTS	PERCENTAGE
DEATH	6	12%
ALIVE	44	88%

Chart 16

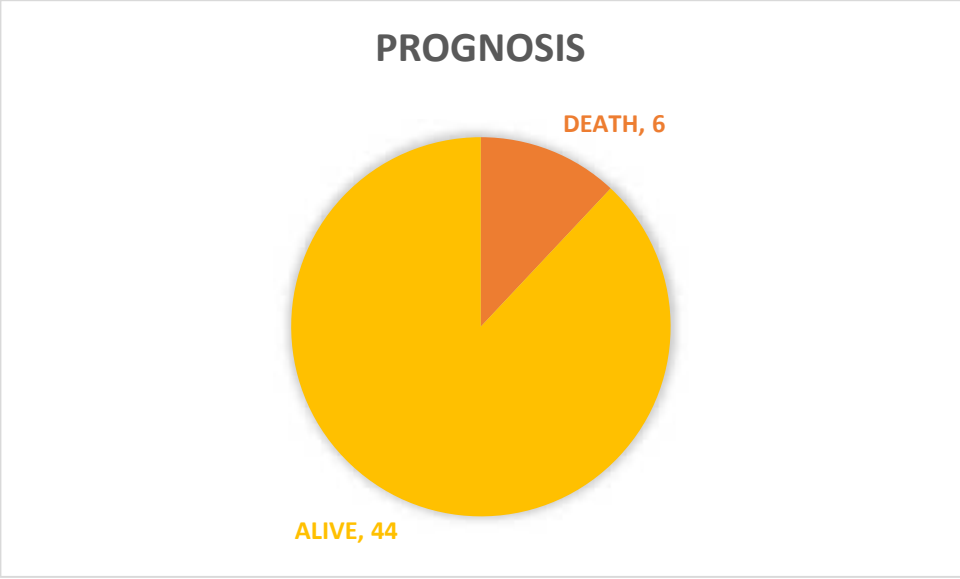


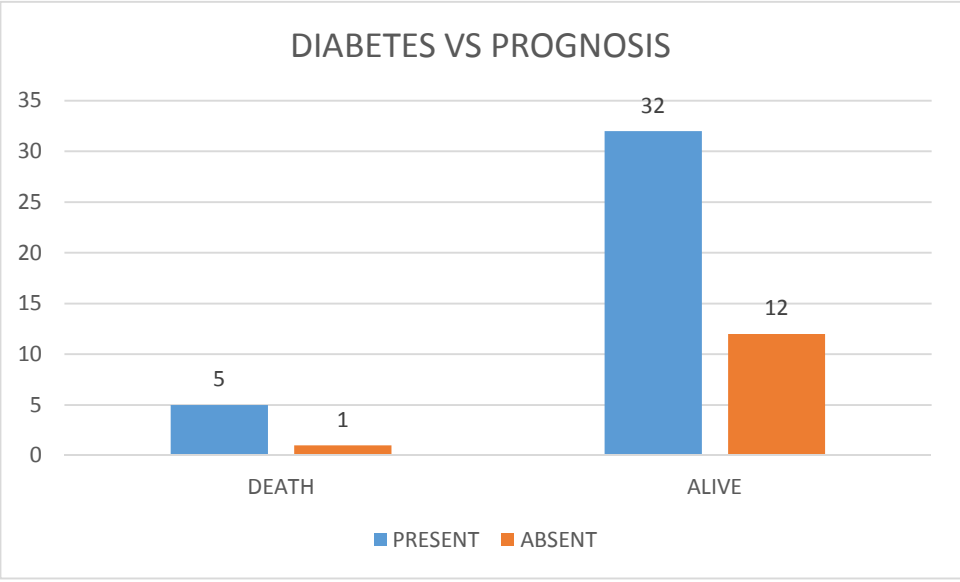
Table 18

DESCRIPTIVE STATISTICS				
	Minimum	**Maximum**	**Mean**	**SD**
AGE	24	70	51.12	11.888
TC	5200	35800	14590.00	5073.470
Hb	6.5	16.8	10.806	1.5495
PLATELETS	18000	460000	175580.00	97190.763
HbA1C	5.0	12.0	7.408	1.6829
UREA	15	241	65.98	44.015
CREATININE	.6	6.9	1.922	1.2572
SODIUM	120	146	135.72	5.268
POTASSIUM	.8	5.3	3.912	.7858

Table 19

	PROGNOSIS	
DIABETES	DEATH	ALIVE
PRESENT	5	32
ABSENT	1	12
P VALUE - 0.578		
NON SIGNIFICANT		
CHI SQUARE TEST		

Chart 17

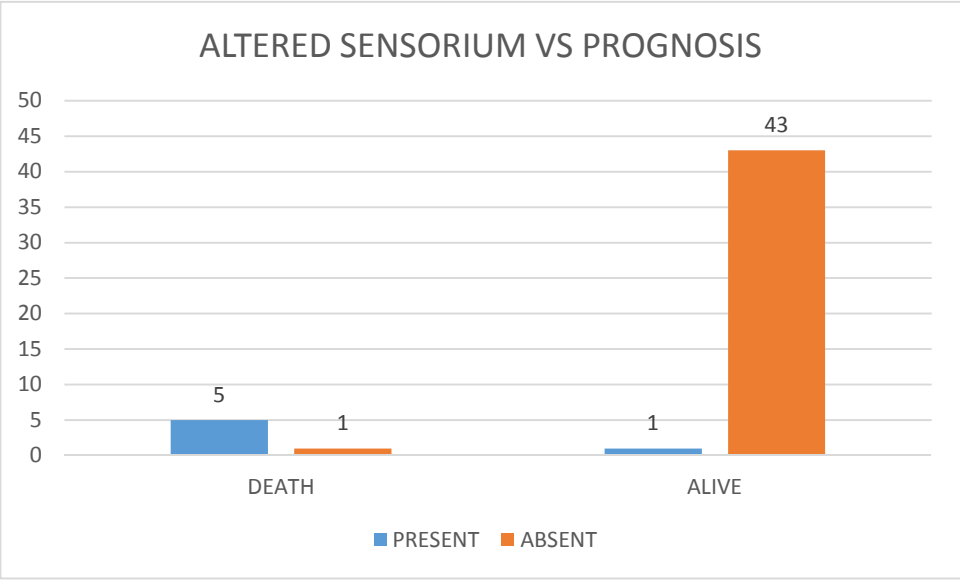


5 out of 6 patients who died were diabetics.

Table 20

	PROGNOSIS	
ALTERED SENSORIUM	DEATH	ALIVE
PRESENT	5	1
ABSENT	1	43
P VALUE - 0.001		
SIGNIFICANT		
CHI SQUARE TEST		

Chart 18

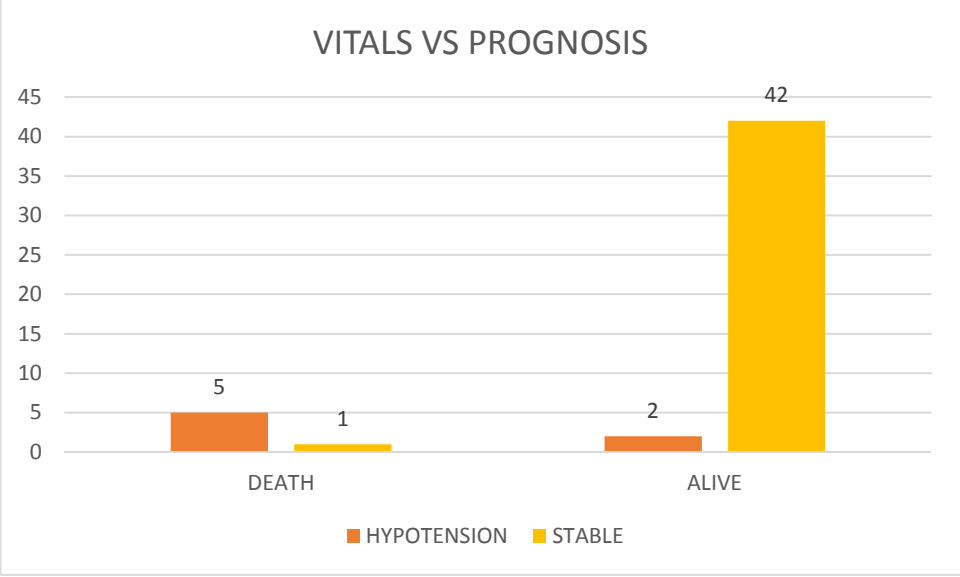


5 out of 6 deaths presented with altered sensorium

Table 21

	PROGNOSIS	
VITALS	DEATH	ALIVE
HYPOTENSION	5	2
STABLE	1	42
P VALUE - 0.001		
SIGNIFICANT		
CHI SQUARE TEST		

Chart 19

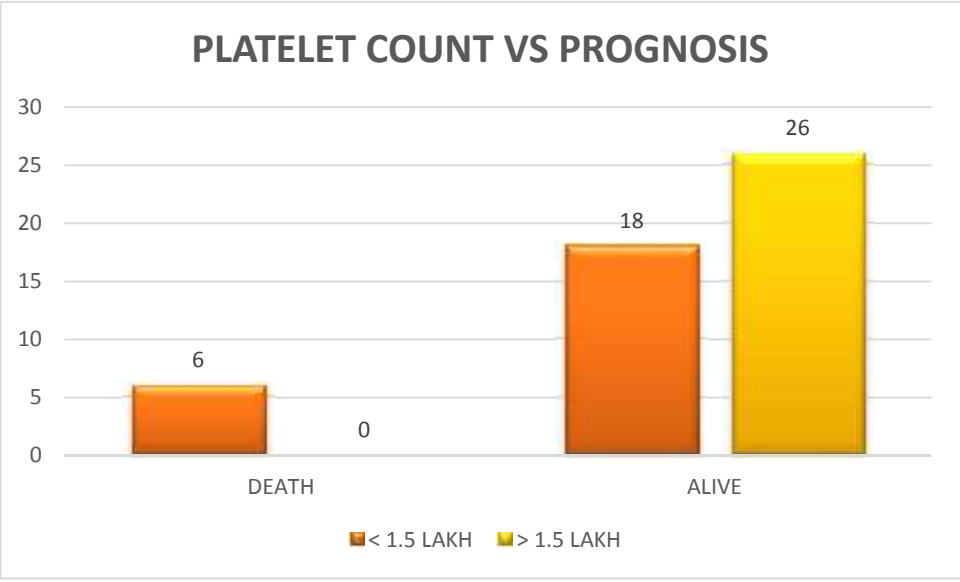


5 out of 6 patients who died presented with hypotension

Table 22

	PROGNOSIS	
PLATELET COUNT	DEATH	ALIVE
< 1.5 LAKH	6	18
> 1.5 LAKH	0	26
P VALUE - 0.007		
SIGNIFICANT		
CHI SQUARE TEST		

Chart 20

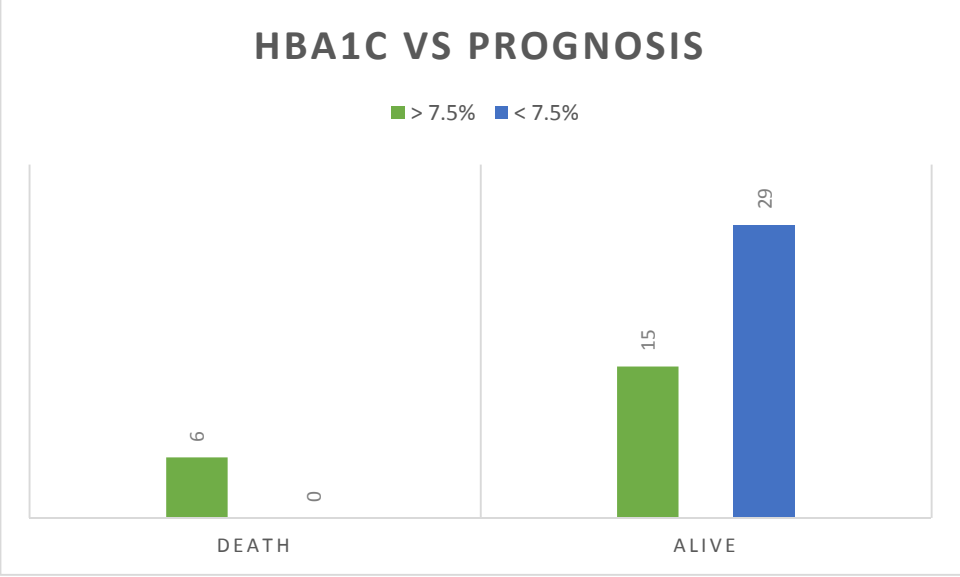


6 out of 6 patients who died had thrombocytopenia

Table 23

	PROGNOSIS	
HBA1C	DEATH	ALIVE
> 7.5%	6	15
< 7.5%	0	29
P VALUE - 0.002		
SIGNIFICANT		
CHI SQUARE TEST		

Chart 21

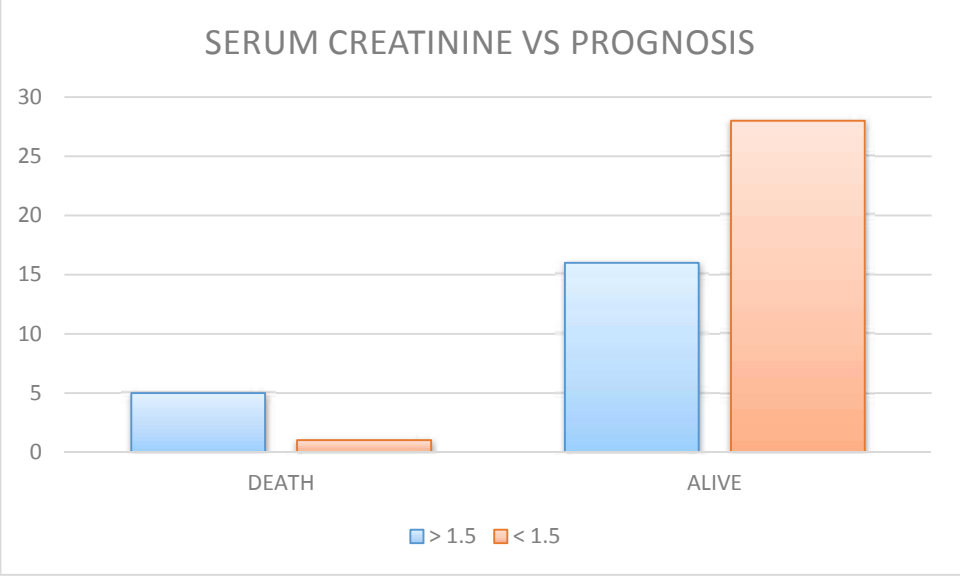


ALL 6 PATIENTS WHO DIED HAD POORLY CONTROLLED GLYCAEMIC STATUS

Table 24

	PROGNOSIS	
SERUM CREATININE	DEATH	ALIVE
> 1.5	5	16
< 1.5	1	28
P VALUE - 0.029		
SIGNIFICANT		
CHI SQUARE TEST		

Chart 22

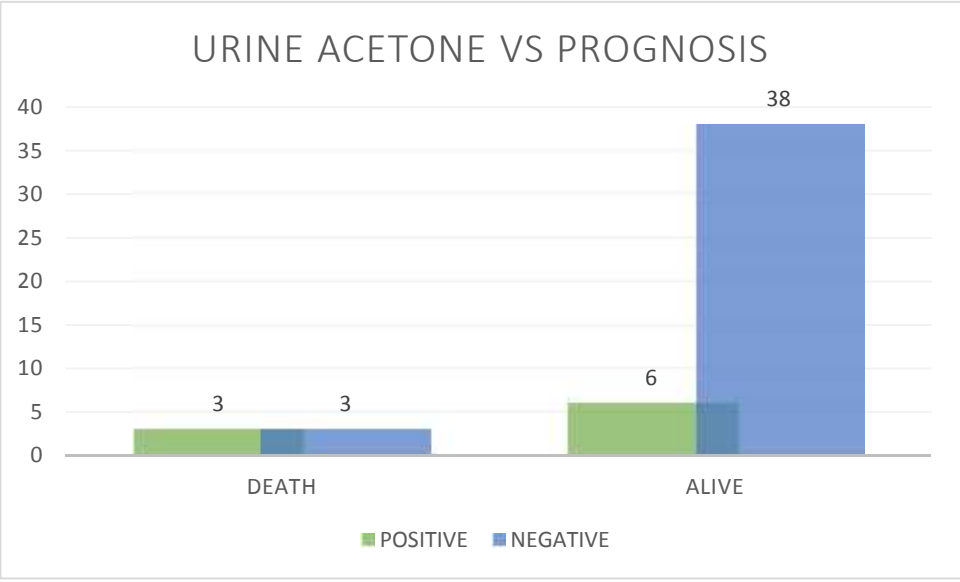


5 OUT OF 6 PATIENTS WHO DIED HAD RENAL DYSFUNCTION

Table 25

	PROGNOSIS	
URINE ACETONE	DEATH	ALIVE
POSITIVE	3	6
NEGATIVE	3	38
P VALUE - 0.030		
SIGNIFICANT		
UNPAIRED T TEST		

Chart 23

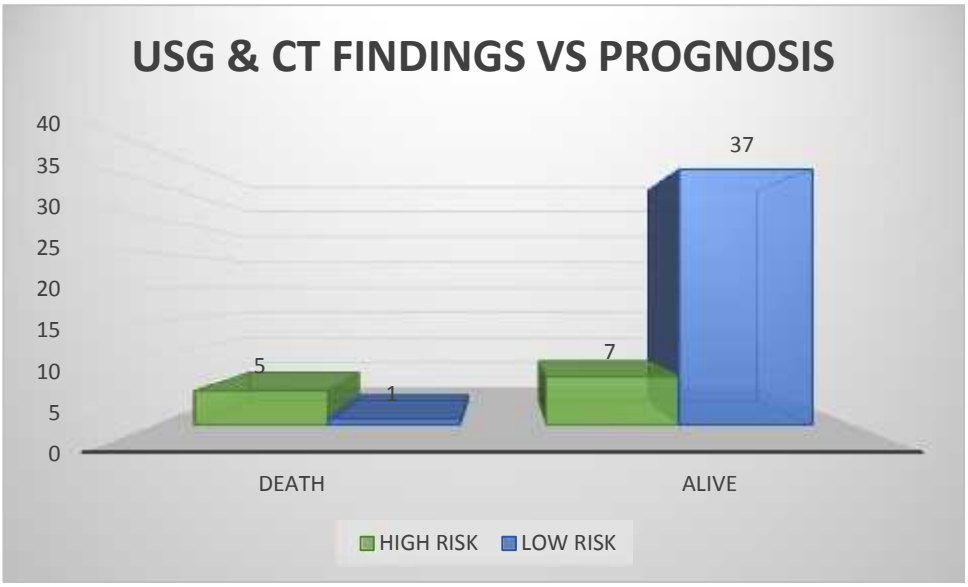


ACETONE POSITIVE IN 3 OUT OF 6 PATIENTS WHO DIED

Table 26

	PROGNOSIS	
USG & CT FINDINGS	DEATH	ALIVE
HIGH RISK	5	7
LOW RISK	1	37
P VALUE - 0.001		
SIGNIFICANT		
CHI SQUARE TEST		

Chart 24

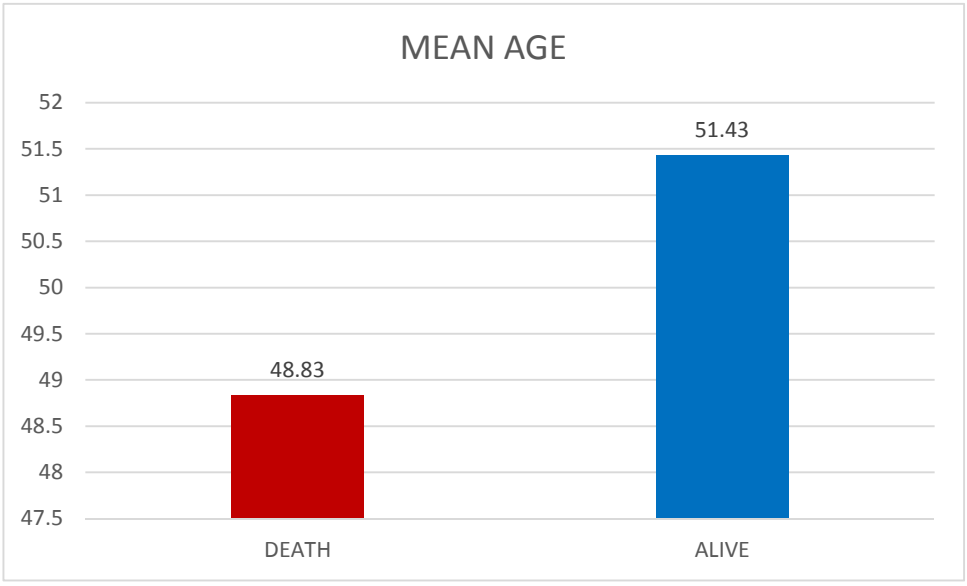


5 OUT OF 6 PATIENTS WHO DIED HAD HIGH RISK RADIOLOGICAL FEATURES

Table 27

	AGE	
PROGNOSIS	MEAN	SD
DEATH	48.83	12.68
ALIVE	51.43	11.89
P VALUE - 0.620		
SIGNIFICANT		
UNPAIRED T TEST		

Chart 25

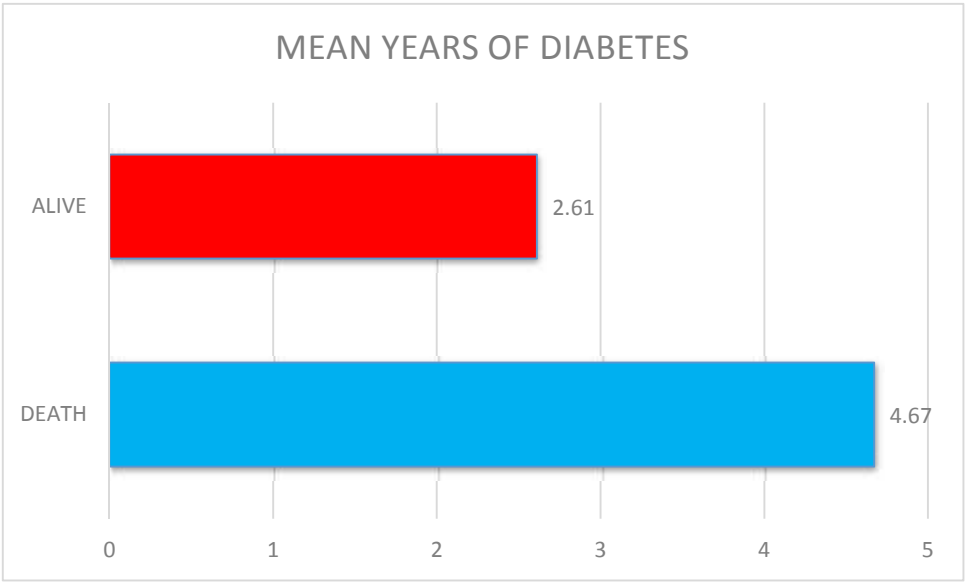


MEAN AGE AT DEATH WAS 48.83%

Table 28

	DM -NO OF YEARS	
PROGNOSIS	MEAN	SD
DEATH	4.67	2.87
ALIVE	2.61	2.36
P VALUE -0.05		
SIGNIFICANT		
UNPAIRED T TEST		

Chart 26

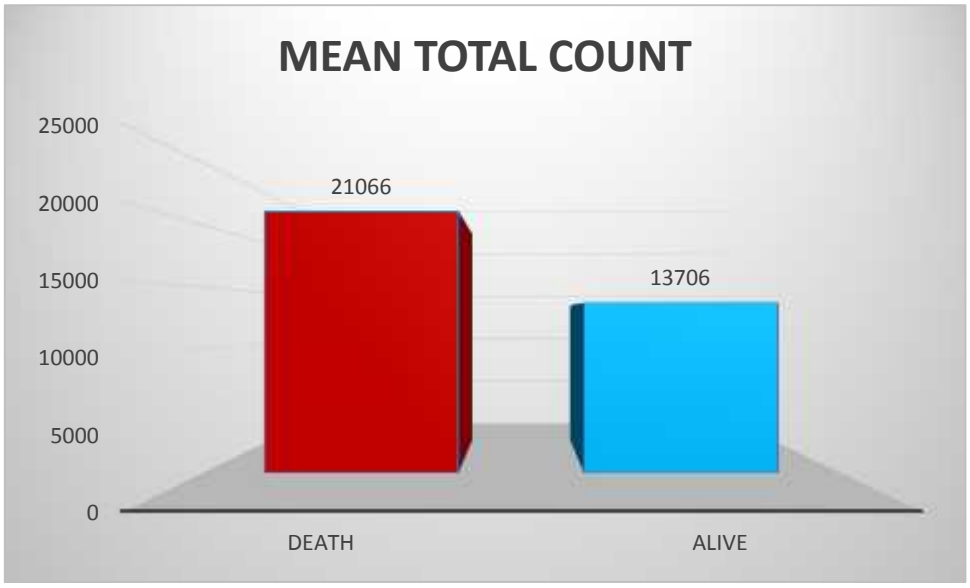


PATIENTS WHO DIED HAD 4.67 MEAN YEARS OF DIABETES

Table 29

	TOTAL COUNT	
PROGNOSIS	MEAN	SD
DEATH	21066	9199
ALIVE	13706	3583
P VALUE - 0.001		
SIGNIFICANT		
UNPAIRED T TEST		

Chart 27



MEAN TOTAL COUNT AMONG PATIENTS WHO DIED WAS 21066

Table 30

	HEMOGLOBIN	
PROGNOSIS	MEAN	SD
DEATH	10.95	1.48
ALIVE	10.78	1.57
P VALUE - 0.811		
NON SIGNIFICANT		
UNPAIRED T TEST		

Chart 28

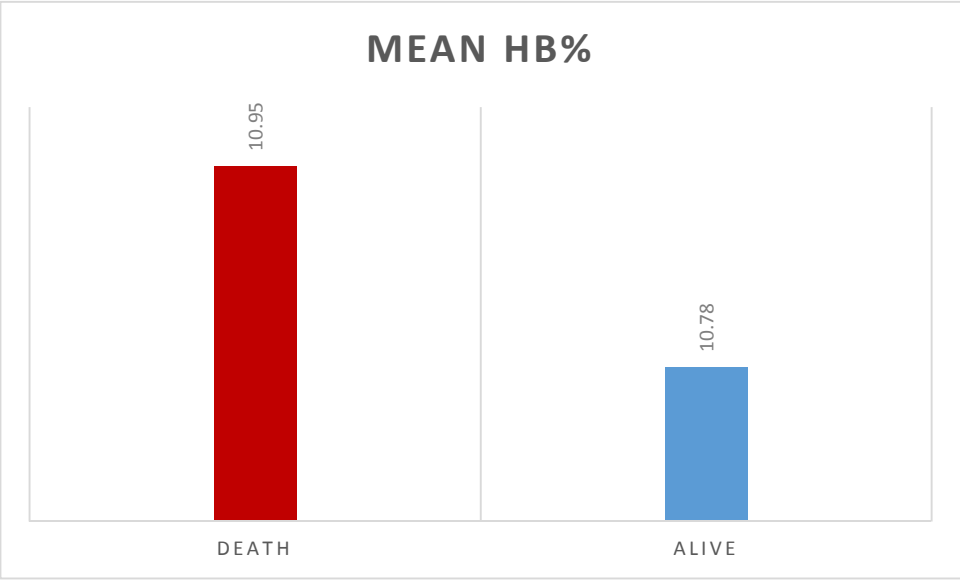
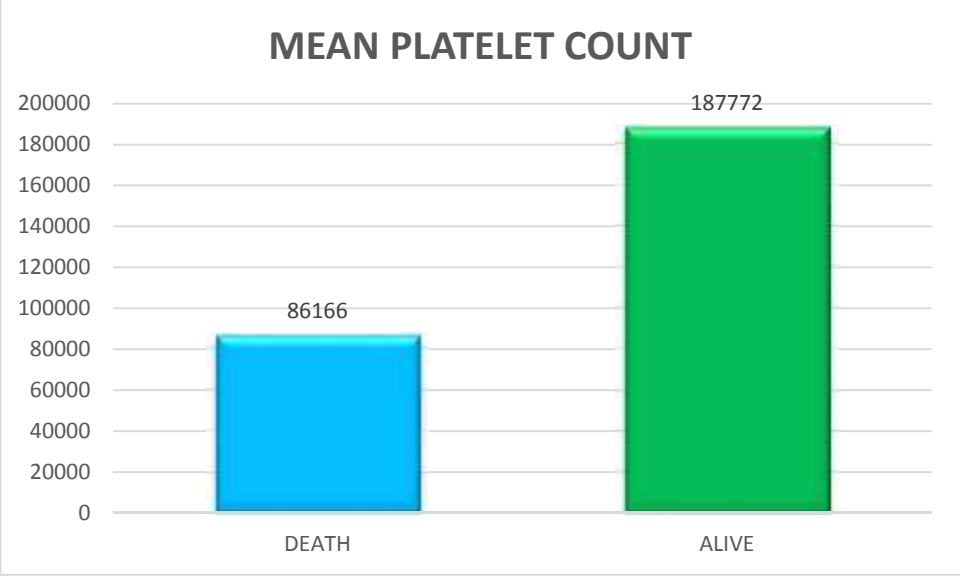


Table 31

	PLATELET COUNT	
PROGNOSIS	MEAN	SD
DEATH	86166	22489
ALIVE	187772	97147
P VALUE - 0.015		
SIGNIFICANT		
UNPAIRED T TEST		

Chart 29

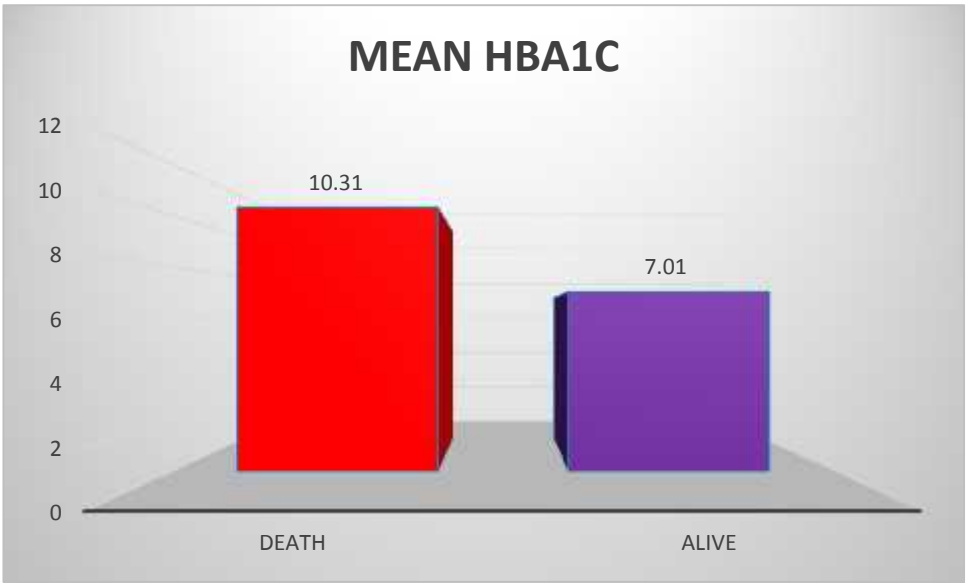


MEAN PLATELET COUNT AMONG PATIENTS WHO DIED WAS 86166

Table 32

	HBA1C	
PROGNOSIS	MEAN	SD
DEATH	10.31	1.52
ALIVE	7.01	1.27
P VALUE - 0.001		
SIGNIFICANT		
UNPAIRED T TEST		

Chart 30



MEAN HBA1C WAS 10.13

Table 33

	BLOOD UREA	
PROGNOSIS	MEAN	SD
DEATH	120.17	69.18
ALIVE	58.59	34.43
P VALUE - 0.001		
SIGNIFICANT		
UNPAIRED T TEST		

Chart 31

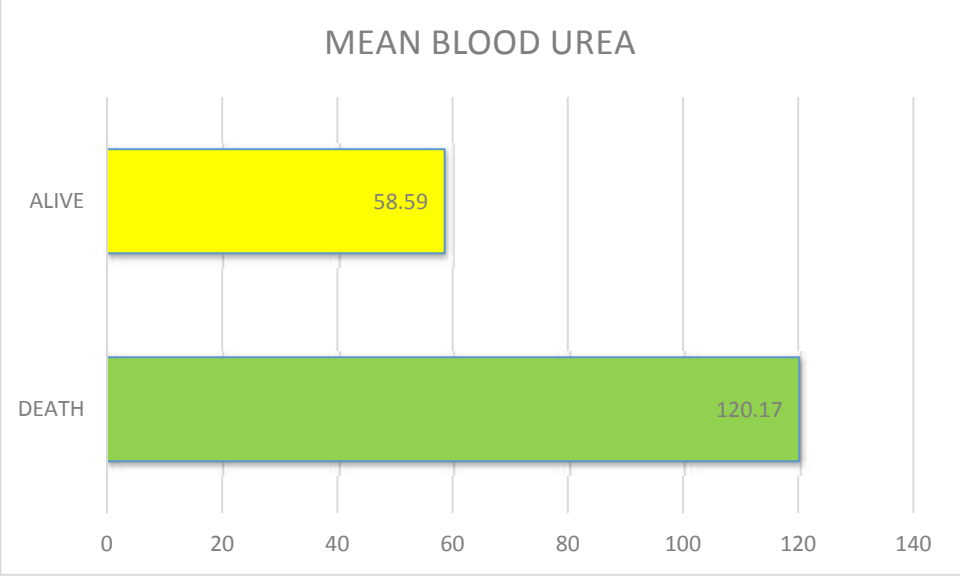
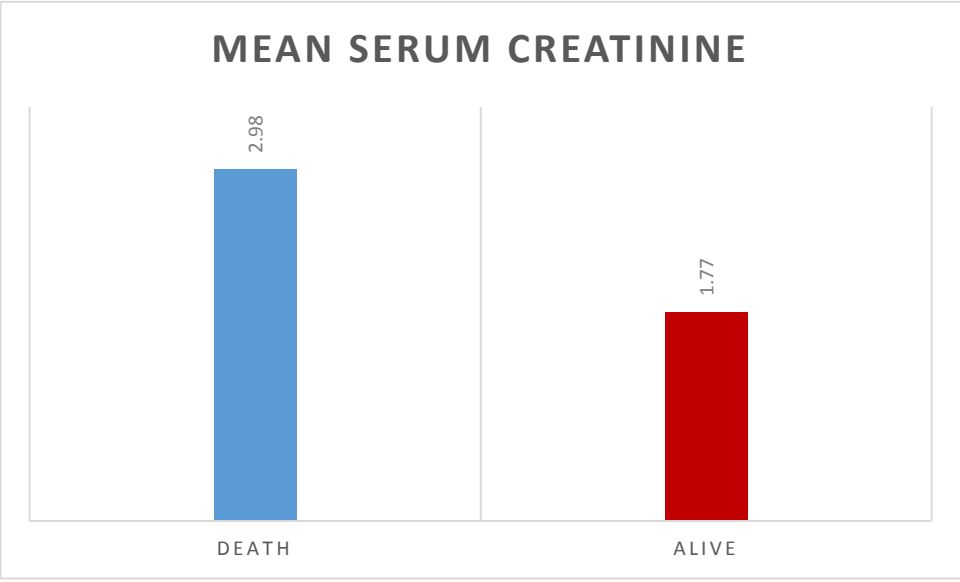


Table 34

	SERUM CREATININE	
PROGNOSIS	MEAN	SD
DEATH	2.98	1.52
ALIVE	1.77	1.16
P VALUE - 0.026		
SIGNIFICANT		
UNPAIRED T TEST		

Chart 32

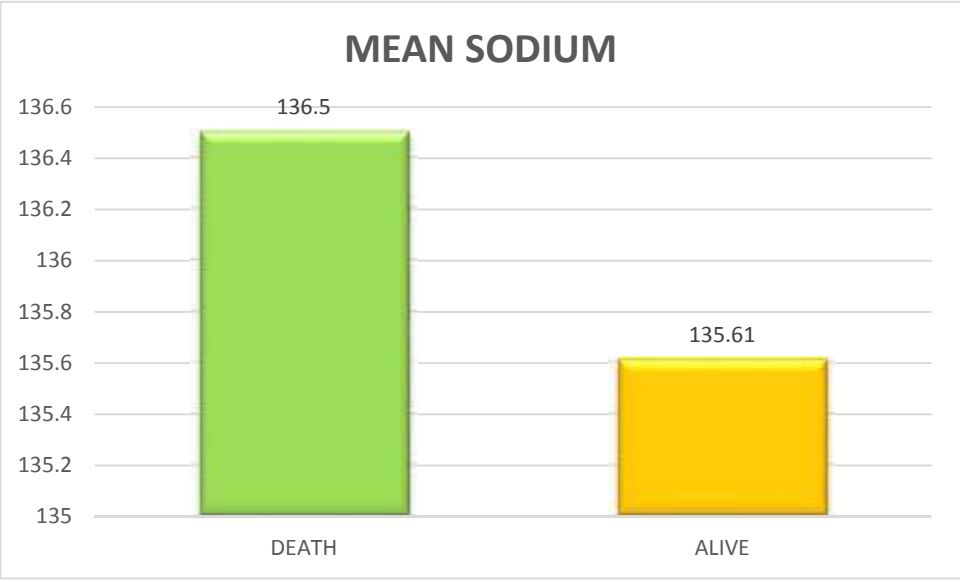


MEAN S.CREATININE WAS 2.98 IN PATIENTS WHO DIED

Table 35

	SODIUM	
PROGNOSIS	MEAN	SD
DEATH	136.5	5.46
ALIVE	135.61	5.29
P VALUE - 0.703		
NON SIGNIFICANT		
UNPAIRED T TEST		

Chart 33



MEAN SODIUM WAS 136.5 AMONG PATIENTS WHO DIED

Table 36

	POTASSIUM	
PROGNOSIS	MEAN	SD
DEATH	4	0.62
ALIVE	3.9	0.81
P VALUE - 0.773		
NON SIGNIFICANT		
UNPAIRED T TEST		

DISCUSSION

In a recent community based estimate, UTI were found to be second only to LRTI among older diabetics. The extent of involvement ranges from inconsequential lower urinary tract colonization to cystitis, pyelonephritis, renal or perirenal abscess.

Prevalence of diabetes in patient with emphysematous pyelonephritis ranges from 53-90 %. It is treated with conventional parenteral antibiotics with percutaneous /open surgical drainage with or without nephrectomy.

However there have been few large studies, which have selectively looked into the clinical, microbial profile & treatment outcome of diabetic patients with pyelonephritis both NEPN & EPN.

Hence to address this issue this prospective observational study is undertaken to evaluate factors determining prognosis of patients with pyelonephritis.

In a previous study done by Akhaira et al, Int urol nephrol 41(4) 2009 april, clinical profile &prognostic factors & outcomes of 19 patients with emphysematous pyelonephritis. Patients were followed for a period of 6 months. From 2001-2007, 19 cases were studied, out of which 16 were females,3 males .14 were diabetic patients. E.Coli was the predominant

causative organism. Shock (P=0.03), S.CREATININE > 5 mg/dl (p=0.035) & DIC (p=0.017) were independent poor prognostic factors.5 cases underwent percutaneous drainage,3 underwent nephrectomy,10.5% expired

In another study of Acute pyelonephritis in diabetes mellitus single centre experience, done in 2010 -2012 where 105 diabetic patients were studied ,in which 79 had non emphysematous pyelonephritis (75.2%) ,26 had emphysematous pyelonephritis. E.Coli was the most common cultured organism. Renal abscess were seen in 13 % & papillary necrosis in 4 % of cases. Worsening renal function observed in 92 % of EPN &93 % of NEPN .Nephrectomy done in 5 patients (19.2 %).13 patients expired . 4 had EPN & 9 had NEPN. EPN patients presented with shock & had poorly controlled blood sugar levels. Shock, altered sensorium associated with poor outcome in patients with EPN .DM with pyelonephritis associated with severe disease .Emphysematous pyelonephritis had poor treatment outcome than NEPN ,but no difficulty in mortality between them. There was greater need of nephrectomy in EPN compared to NEPN.

In our study done from June 2016 –June 2017 in Tirunelveli medical college ,50 patients with pyelonephritis with classical clinical features of fever ,chills & flank pain & tenderness ,with radiological evidence of acute pyelonephritis were studied. Associated co-morbidities like DM, urolithiasis

were noted. Blood investigations CBC, Serial RBS, FBS & PPBS ,RFTS.S.Electrolytes,LFT,HBA1C,Urine albumin, sugar, deposits acetone (if needed) & c/s done, radiological imaging USG abdomen & KUB ,CT scan abdomen taken patients then followed for a period of 2 months & factors determining the prognosis of patients studied. Patients who recovered with antibiotics, percutaneous nephrostomy & other supportive measures were considered to have good prognosis .Those who required nephrectomy or died were considered to have poor prognosis.

Out of 50 patients studied 34% belonged to the age group 51-60 .12 were males(24%) ,38 were females(76%).T2DM present in 37 patients (74%), Urolithiasis in 4 patients (8%).6 patients presented with altered sensorium (12 %).7 patients presented with hypotension .(BP<90/60 mm hg).Thrombocytopenia (platelets <1.5 lakh)seen in 24 patients.(48%). Glycaemic status were stratified as good (<7%),moderate (7-7.5%) & poor (>7.5%).21 patients had poorly controlled diabetic status (42 %).Renal dysfunction was considered with a creatinine level >1.5 .21 patients had renal dysfunction (42 %)

Alterd LFT seen in 6 patients (12 %).Urine culture showed positive in 22 patients (44%).E.Coli in 16 patients (72 %)., Klebsiella in 4 patients,

Pseudomonas & fungal species in 1 patient each .Urine acetone positive in 9 patients .

Radiological imaging showing B/L Pyelonephritis, Emphysematous pyelonephritis, Papillary necrosis & renal abscess were considered high risk & were present in 12 patients. Emphysematous pyelonephritis seen in 11 patients (22%)After follow up period of 2 months ,41 patients recovered with antibiotics ,3 needed percutaneous nephrostomy & 6 patients died.

Diabetes with pyelonephritis associated in 5 deaths.(p value 0.578).-non significant. 5 out of 6 patients who died presented with altered sensorium (p value 0.001 –significant). 5 out of 6 Patients who died presented with hypotension (p value 0.001 –significant (chi square test).All 6 patients who died presented with thrombocytopenia (p value 0.007 –significant).All 6 patients who died had HBA1C OF > 7.5 % (P VALUE 0.002 –significant)

5 patients out of 6 who died presented with renal dysfunction. High risk radiological findings seen in 5 out of 6 patients who died.p value 0.001 –significant.

CONCLUSION

Acute Pyelonephritis is more common in females. Most common age group is 51-60 years. Diabetes mellitus is the most common co-morbidity associated followed by urolithiasis .E.Coli was the predominant organism cultured in urine. Presence of altered sensorium & hypotension at admission associated with poor outcome. Thrombocytopenia and Renal dysfunction are associated with poor prognosis. Long standing duration of DM with poor glycaemic control associated with poor prognosis. Presence of radiological features of Emphysematous pyelonephritis, B/L pyelonephritis, renal abscess & papillary necrosis is associated with poor prognosis.

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S.NO	NAME	AGE	SEX	CO-MORBIDITIES	SYMPTOMS	SIGNS	VITALS	TC	Hb	PLATELETS	HbA1C	UREA	CREATININE	SODIUM	POTASSIUM
1	LEELAVATHY	70	F	T2DM ,3 YEARS	FEVER,ABDOMINAL PAIN	LEFT LOIN TENDERNESS	STABLE	15700	10.9	96000	11.2	98	1.5	142	3.8
2	SYED ALI FATHIMA	30	F	T2DM,5 YEARS	ABDOMINAL PAIN,DYSURIA	SUPRAPUBIC TENDERNESS	STABLE	16600	9.5	3.8 LAKHS	8.6	49	0.6	130	3.5
3	MEENA	47	F	T2DM,3 YEARS	FEVERABDOMINAL PAIN,DYSURIA	LEFT LOIN & SUPRAPUBIC TENDERNESS	STABLE	16400	10	4.4 LAKHS	10.8	21	1.1	139	4.3
4	MARIAPPAN	25	M	UROLITHIASIS	FEVER ,ABDOMINAL PAIN	DROWSY,P/A;DISTENDED:LEFT LOIN TENDERNESS ,BS:SLUGGISH	HYPOTENSION	35800	9.7	92000	5.2	135	3.7	135	3.2
5	RAMALAKSHMI	56	F	T2DM,5 YEARS	VOMITING,ABDOMINAL PAIN	P/A :SOFT	STABLE	13200	9.5	1.5 LAKHS	8.6	62	2	132	3
6	PETCHIAMMAL	24	F	NIL	DYSURIA,FEVER	P/A :SOFT	STABLE	5900	10.6	1.7 LAKHS	5.3	65	1.7	132	3.9
7	SHENBAGAM	59	F	T2DM,3 YEARS	FEVER,ABDOMINAL PAIN	P/A :SOFT	STABLE	17800	9	3.2 LAKHS	7.5	18	1.5	136	4.1
8	BALAPUSHPAM	60	F	T2DM,3 YEARS	FEVER,ALTERED SENSORIUM	DROWSY,P/A :SOFT	STABLE	13800	10.8	2.2 LAKHS	7.2	46	0.9	146	3.5
9	SAROJA	60	F	T2DM,15 YEARS	FEVER,BREATHLESSNESS	DYSPNOEIC,P/A :SOFT	STABLE	12700	10.8	1.O6 LAKHS	8.3	64	2.4	146	3.7
10	CHELLAMMAL	68	F	T2DM,3 YEARS	FEVER,ABDOMINAL PAIN	OMPHALOCELE +	STABLE	14400	11.6	1.05 LAKHS	8.3	41	1.3	137	4
11	SARAVANA KUMARI	43	F	T2DM ,1 YEAR	FEVER,ABDOMINAL PAIN	P/A:DIFFUSE TENDERNESS	STABLE	27300	9.1	1.9 LAKH	6.2	79	2.4	130	3.2
12	SATHIYAPPAN	59	M	T2DM ,7 YEARS	DYSURIA,BOTH LEG SWELLING	TENDERNESS RIGHT LOIN	STABLE	14500	10.3	1.2 LAKH	11.2	41	1.1	136	4
13	ULAGESHWARI	50	F	T2DM,15 YEARS	FEVER,CHILLS,ABDOMINAL PAIN	LEFT LOIN TENDERNESS	STABLE	12500	8.3	7.6 LAKHS	5.7	16	1	132	3.8
14	KANAGAMMAL	50	F	T2DM,5 YEARS	FEVER,DYSURIA	P/A :SOFT	STABLE	14700	12	1.75 LAKHS	12.00	52	1	136	4.8
15	ESAKKIRAJ	24	M	SEIZURE DISORDER	FEVER,ABDOMINAL PAIN,SEIZURES 1 EPISODE	TENDERNESS LEFT LOIN	STABLE	8500	13.1	2.03 LAKHS	5.2	111	6.9	138	4.9

S.NO	NAME	AGE	SEX	CO-MORBIDITIES	SYMPTOMS	SIGNS	VITALS	TC	Hb	PLATELETS	HbA1C	UREA	CREATININE	SODIUM	POTASSIUM
16	ESAKKIRAJ	54	M	T2DM,5 YEARS	FEVER ,ABDOMINAL PAIN	LEFT LOIN TENDERNESS	STABLE	11500	13.6	2.5 LAKHS	8.2	100	2.9	127	4
17	MYDEEN	65	M	T2DM,5 YEARS	FEVER ,ABDOMINAL PAIN	TENDERNESS LEFT LOIN & LUMBAR REGION	STABLE	23700	10.7	18000	10.7	131	3.4	121	4.6
18	SEENISAMY	68	M	RENAL & VESICAL CALCULI	FEVER,DYSURIA	P/A:SOFT	STABLE	6000	12	1.9 LAKH	5	47	2.8	131	4.6
19	PUSHPAM	60	F	,PTB	ABDOMINAL PAIN,DYSURIA	RS:BRONCHIAL BREATH SOUNDS,P/A:DIFFUSE TENDERNESS,HSM	STABLE	5200	6.5	2.3 LAKHS	5.6	15	0.8	138	4.1
20	VALLIAMMAL	50	F	T2DM,7 YEARS	FEVER,DYSURIA	P/A SOFT	STABLE	16300	16.8	1.2 LAKHS	8.9	121	2.5	139	5
21	PARVATHI	65	F	T2DM,5 YEARS	FEVER,ABDOMINAL PAIN	TENDERNESS LEFT HYPOCHONDRIUM	STABLE	12900	8.9	1.09 LAKHS	10.8	47	1.9	120	5.3
22	ALAGARSAMY	60	M	CKD	FEVER,BREATHLESSNESS,DYSURIA	TENDERNESS IN SUPRAPUBIC & RIGHT LOIN TENDERNESS	STABLE	22200	13.1	50000	5.1	241	5.5	129	3.3
23	AMALIN ROSY	45	F	T2DM	ABDOMINAL PAIN,VOMITING,FEVER	TENDERNESS LEFT LUMBAR REGION & LOIN	STABLE	17500	9.3	3.5 LAKHS	9.6	28	1.3	144	4.3
24	AMUTHA	41	F	T2DM	FEVER,ABDOMINAL PAIN	TENDERNESS LEFT LUMBAR & RENAL ANGLE	STABLE	10700	11.5	3.6 LAKHS	9.6	19	1	139	4.3
25	HELEN	38	F	T2DM,10 YEARS,PTB	FEVER ,ABDOMINAL PAIN	TENDERNESS LEFT LUMBAR & RENAL ANGLE	STABLE	12500	10.8	2.5 LAKHS	10.3	47	2.2	137	5.2
26	SOWMYA	35	F	T2DM,3 YEARS	FEVER,DYSURIA	TENDERNESS SUPRAPUBIC REGION	STABLE	11900	11.6	2.1 LAKHS	9.6	34	1.2	135	3.6

S.NO	NAME	AGE	SEX	CO-MORBIDITIES	SYMPTOMS	SIGNS	VITALS	TC	Hb	PLATELETS	HbA1C	UREA	CREATININE	SODIUM	POTASSIUM
27	PETCHIAMMAL	65	F	T2DM,2 YEARS	FEVER,ABDOMINAL PAIN,DYSURIA	TENDERNESS LEFT LUMBAR & RENAL ANGLE	STABLE	16700	9.8	1.6 LAKHS	11.2	123	3.2	136	4.3
28	PERACHI	48	F	LEFT RENAL CALCULI	FEVER,ABDOMINAL PAIN	TENDERNESS LEFT LUMBAR & RENAL ANGLE	STABLE	18600	10.6	1.8 LAKHS	5.6	56	1.3	135	3.8
29	VEERAMANI	56	M	T2DM ,5 YEARS,RIGHT RENAL CALCULI	FEVER,ABDOMINAL PAIN	TENDERNESS RIGHT LUMBAR REGION	STABLE	21800	12.8	98000	10.8	142	3.8	132	3.8
30	KARUPPASAMY	63	M	T2DM,3 YEARS	FEVER,ABDOMINAL PAIN	RIGHT RENAL ANGLE TENDERNESS	STABLE	15600	11.8	1.5 LAKHS	12.2	42	1.1	134	3.4
31	VANAJA	46	F	NIL	FEVER,DYSURIA	SUPRAPUBIC TENDERNESS	STABLE	16800	10.8	1.7 LAKHS	5.6	35	1.2	136	4.3
32	PAPPATHI	56	F	T2DM,5 YEARS	FEVER,ABDOMINAL PAIN,DYSURIA	TENDERNES LEFT LUMBAR,LEFT RENAL ANGLE	STABLE	13500	10.7	1.2 LAKHS	11.2	56	2.2	135	3.8
33	JANAKI	38	F	NIL	FEVER,DYSURIA	TENDERNESS LEFT LUMBAR & RENAL ANGLE	STABLE	12600	11.6	1.8 LAKHS	5.4	38	1.2	136	3.8
34	VALLIAMMAL	45	F	T2DM,2 YEARS	FEVER,ABDOMINAL PAIN,DYSURIA	TENDERNESS LEFT LUMBAR REGION	STABLE	18900	10.4	2.2 LAKHS	10.6	46	1	136	3.8
35	RAJATHI	56	F	T2DM,5 YEARS	FEVER,ABDOMINAL PAIN,DYSURIA	DROWSY,AROUSABLE,TENDERNESS LEFT LUMBAR & SUPRAPUBIC REGION	HYPOTENSION	26700	9.6	83000	11.2	76	2.8	146	4.3
36	RANI	62	F	T2DM,5 YEARS	FEVER,DYSURIA	TENDERNESS LEFT LUMBAR REGION	STABLE	18600	11.8	1.6 LAKHS	8.2	45	1.2	135	3.8
37	SRIRENGANAYAKI	53	F	CAHD	FEVER,DYSURIA	SUPRAPUBIC TENDERNESS	STABLE	21600	10.6	1.6 LAKHS	5.2	38	1.2	136	4.2
38	CHRISTY	51	F	NIL	FEVER,DYSURIA	RIGHT RENAL ANGLE TENDERNESS	STABLE	21000	11.6	1.8 LAKHS	5.6	45	1.1	135	3.8

S.NO	NAME	AGE	SEX	CO-MORBIDITIES	SYMPTOMS	SIGNS	VITALS	TC	Hb	PLATELETS	HbA1C	UREA	CREATININE	SODIUM	POTASSIUM
39	ATHIMOOLAM	56	M	T2DM,3 YEARS	ABDOMINAL PAIN,DYSURIA	TENDERNESS LEFT LUMBAR REGION	STABLE	16500	11.6	2.2 LAKHS	11.8	75	2.8	136	4.2
40	BALAMMAL	52	F	T2DM,5 YEARS	FEVER,ABDOMINAL PAIN,DYSURIA	TENDERNESS SUPRAPUBIC REGION	STABLE	12800	10.6	1.5 LAKHS	7.9	46	1.2	136	3.8
41	REKHA	43	F	T2DM,3 YEARS	FEVER,DYSURIA	TENDERNESS LEFT RENAL ANGLE & LUMBAR REGION	STABLE	14600	10.2	1.8 LAKHS	12.2	40	1.1	135	3.9
42	CHELLAMMAL	65	F	T2DM,4YEARS	ABDOMINAL PAIN,DYSURIA	SUPRAPUBIC TENDERNESS	STABLE	13600	9.6	1..5 LAKHS	8.2	36	1.4	136	4.6
43	KEERTHI	32	F	NIL	FEVER,ABDOMINAL PAIN,DYSURIA	TENDERNESS LEFT LUMBAR & RENAL ANGLE	STABLE	11500	10.8	2.8 LAKHS	5.2	46	1	146	4.8
44	SUMATHI	69	F	T2DM,8 YEARS	FEVER,ABDOMINAL PAIN,DYSURIA	TENDERNESS LEFT LUMBAR & RENAL ANGLE	STABLE	18900	11.2	86000	11.2	113	4.3	138	3.8
45	SASI	45	F	T2DM,5 YEARS	FEVER,DYSURIA	TENDERNESS RIGHT LUMBAR REGION	STABLE	13600	11.6	1.6 LAKHS	7.8	46	1	135	3.6
46	MADASAMY	48	M	RENAL & VESICAL CALCULI	FEVER,ABDOMINAL PAIN	TENDERNESS LEFT LUMBAR & RENAL ANGLE	STABLE	12600	10.2	1.6 LAKHS	5.2	42	1.3	142	3.8
47	RAMU	54	F	PTB	ABDOMINAL PAIN	TENDERNESS LEFT RENAL ANGLE & LUMBAR REGION	STABLE	11500	10.1	1.8 LAKHS	5	40	0.9	135	3.8
48	LAKSHMI	46	F	T2DM,7 YEARS	FEVER,DYSURIA,ABDOMINAL PAIN	LEFT LUMBAR & RENAL ANGLE TENDERNESS	STABLE	10500	9.7	2.6 LAKHS	9.2	142	2.1	137	4.2
49	CHELLARANI	56	F	T2DM,5 YEARS	FEVER,DYSURIA	SUPRAPUBIC TENDERNESS	STABLE	12300	11.8	2.8 LAKHS	8.2	135	1.2	135	1.2

S.NO	NAME	AGE	SEX	CO-MORBIDITIES	SYMPTOMS	SIGNS	VITALS	TC	Hb	PLATELETS	HbA1C	UREA	CREATININE	SODIUM	POTASSIUM
50	SURESH	45	M	T2DM,2 YEARS	FEVER,DYSURIA,ABDOMINAL PAIN	LEFT LUMBAR & RENAL ANGLE TENDERNESS	STABLE	10800	10.8	2.6 LAKHS	10.2	68	1.9	136	0.8

S.NO	NAME	LFT	URINE DEPOSITS	URINE CULTURE	URINE ACETONE	USG ABDOMEN	CT ABDOMEN	NEED FOR PCN/NEPHRECTOMY	OUTCOME
1	LEELAVATHY	WNL	4-6 PUS CELLS	NO GROWTH	POSITIVE	LEFT PYELONEPHRITIS	LEFT EMPHYSEMATOUS PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
2	SYED ALI FATHIMA	WNL	PLENTY OF PUS CELLS	KLEBSIELLAGROWN	POSITIVE	LEFT PYELONEPHRITIS	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
3	MEENA	WNL	1-2 PUS CELL	E.COLI GROWN	NEGATIVE	B/L PYELONEPHRITIS	PAPILLARY NECROSIS BOTH KIDNEYS	NO	IMPROVED & DISCHARGED
4	MARIAPPAN	ALTERE	PLENTY OF PUS CELLS	NO GROWTH	NEGATIVE	,LEFT RENAL & URETERIC CALCULUS	,LEFT RENAL & UPPER URETERIC CALCULUS,LEFT RENAL PERINEPHRIC COLLECTION	NO	DEATH
5	RAMALAKSHMI	WNL	4-6 PUS CELLS	NO GROWTH	POSITIVE	MILD LEFT HUN,?PAPILLARY NECROSIS	,LEFT HUN,PAPILLARY NECROSIS	NO	IMPROVED & DISCHARGED
6	PETCHIAMMAL	WNL	4-5 PUS CELLS	E.COLI GROWN	NOT DONE	B/L PYELONEPHRITIS	B/L PYELONEPHRITIS,BLADDER ECHOES,MESENTERIC LYMPHADENOPATHY	NO	IMPROVED & DISCHARGED
7	SHENBAGAM	WNL	5-10 PUS CELLS	NO GROWTH	NEGATIVE	LEFT RENAL CALCULUS,LEFT HUN	LEFT PYELONEPHRITIS,LEFT HUN,	NO	IMPROVED & DISCHARGED
8	BALAPUSHPAM	WNL	3-5 PUS CELLS	NO GROWTH	NEGATIVE	GRADE 1 MRD,B/L PYELONEPHRITIS	B/L PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
9	SAROJA	WNL	PLENTY OF PUS CELLS	NO GROWTH	NEGATIVE	B/L PYELONEPHRITIS	B/L PYELONEPHRIT	NO	IMPROVED & DISCHARGED
10	CHELLAMMAL	WNL	10-15 PUS CELLS	E.COLI GROWN	NEGATIVE	RIGHT PYELONEPHRITIS	RIGHT PYELONEPHRITIS,FATTY LIVER	NO	IMPROVED & DISCHARGED
11	SARAVANA KUMARI	wnl	10-15 pus cells,3-5 RBC S	NO GROWTH	NEGATIVE	LEFT PYELONEPHRITIS	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
12	SATHIYAPPAN	WNL	2-4 PUS CELLS	KLEBSIELLAGROWN	NEGATIVE	RIGHT EMPHYSEMATOUS PYELONEPHRITIS	RIGHT EMPHYSEMATOUS PYELONEPHRITIS ,PERI RENAL COLLECTION	NO	IMPROVED & DISCHARGED
13	ULAGESHWARI	WNL	4-6 PUS CELLS	NO GROWTH	NOT DONE	LEFT PYELONEPHRITIS	B/L EMPHYSEMATOUS PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
14	KANAGAMMAL	WNL	5-8 PUS CELLS	E.COLI GROWN	NEGATIVE	B/L PYELONEPHRITIS	B/L PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
15	ESAKKIRAJ	WNL	1-2 PUS CELL	NO GROWTH	NOT DONE	RIGHT PLEURAL EFFUSION	B/L PYELONEPHRITIS,MRD,MILD ASCITES	NO	IMPROVED & DISCHARGED

S.NO	NAME	LFT	URINE DEPOSITS	URINE CULTURE	URINE ACETONE	USG ABDOMEN	CT ABDOMEN	NEED FOR PCN/NEPHRECTOMY	OUTCOME
16	ESAKKIRAJ	WNL	5-6 PUS CELLS	E.COLI GROWN	NEGATIVE	LEFT PYELONEPHRITIS	B/L PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
17	MYDEEN	ALTERE	2=4 PUS CELLS,1-2 RBC "S	FUNGAL SPECIES GROWN	POSITIVE	LEFT PYELONEPHRITIS	LEFT EMPHYSEMATOUS PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
18	SEENISAMY	WNL	6-8 PUS CELLS	E.COLI GROWN	NOT DONE	B/L PYELONEPHRITIS	B/L EMPHYSEMATOUS PYELONEPHRITIS,B/L RENAL CALCULI,VESICAL CALCULUS	NO	IMPROVED & DISCHARGED
19	PUSHPAM	ALTERE	,1-2 RBC"S	NO GROWTH	NEGATIVE	,HEPATO SPLENOMEGALY	B/L PYELONEPHRITIS,HEPATOSPLENOMEGALY	NO	IMPROVED & DISCHARGED
20	VALLIAMMAL	WNL	1-2 PUS CELL	E.COLI GROWN	POSITIVE	LEFT PYELONEPHRITIS	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
21	PARVATHI	WNL	5-7 PUS CELLS	NO GROWTH	NEGATIVE	LEFT EMPHYSEMATOUS PYELONEPHRITIS	LEFT EMPHYSEMATOUS PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
22	ALAGARSAMY	WNL	5-6 PUS CELLS	NO GROWTH	NOT DONE	RIGHT HUN,HSM,FREE FLUID	RIGHT PYELONEPHRITIS,MULTIPLE CORTICAL CYST,MILD SPLENOMEGALY	NO	IMPROVED & DISCHARGED
23	AMALIN ROSY	WNL	PLENTY OF PUS CELLS	PSEUDOMONAS GROWN	POSITIVE	PYELONEPHRITIS LEFT KIDNEY	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
24	AMUTHA	WNL	5-6 PUS CELLS	NO GROWTH	NEGATIVE	ENLARGED LEFT KIDNEY	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
25	HELEN	WNL	1-2 PUS CELL	NO GROWTH	POSITIVE	ENLARGED LEFT KIDNEYS	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
26	SOWMYA	WNL	5-6 PUS CELLS	NO GROWTH	NEGATIVE	RIGHT PYELONEPHRITIS	RIGHT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED

S.NO	NAME	LFT	URINE DEPOSITS	URINE CULTURE	URINE ACETONE	USG ABDOMEN	CT ABDOMEN	NEED FOR PCN/NEPHRECTOMY	OUTCOME
27	PETCHIAMMAL	WNL	1-2 PUS CELL	E.COLI GROWN	NEGATIVE	B/L PYELONEPHRITIS	B/L PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
28	PERACHI	WNL	PLENTY OF PUS CELLS	NO GROWTH	NOT DONE	LEFT RENAL CALCULUS,LEFT HUN	LEFT RENAL CALCULI,LEFT PYELONEPHRITIS,LEFT HUN	NO	IMPROVED & DISCHARGED
29	VEERAMANI	WNL	5-6 PUS CELLS	KLEBSIELLA GROWN	NEGATIVE	B/L RENAL CALCULI,B/L PYELONEPHRITIS	B/L RENAL CALCULI,B/L PYELONEPHRITIS	PCN DONE	IMPROVED & DISCHARGED
30	KARUPPASAMY	WNL	PLENTY OF PUS CELLS	NO GROWTH	POSITIVE	RIGHT PYELONEPHRITIS	RIGHT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
31	VANAJA	WNL	2-3 PUS CELLS	NO GROWTH	NOT DONE	RIGHT PYELONEPHRITIS	RIGHT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
32	PAPPATHI	WNL	PLENTY OF PUS CELLS	KLEBSIELLA GROWN		LEFT PYELONEPHRITIS	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
33	JANAKI	WNL	5-6 PUS CELLS	NO GROWTH	NOT DONE	LEFT KIDNEY ENLARGED	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
34	VALLIAMMAL	WNL	PLENTY OF PUS CELLS	NO GROWTH	NEGATIVE	LEFT PYELONEPHRITIS	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
35	RAJATHI	ALTERE	PLENTY OF PUS CELLS	E.COLI GROWN	POSITIVE	B/L PYELONEPHRITIS	B/L EMPHYSEMATOUS PYELONEPHRITIS,PAPILLARY NECROSIS	PCN DONE	DEATH
36	RANI	WNL	5-6 PUS CELLS	NO GROWTH	NEGATIVE	LEFT PYELONEPHRITIS	LEFT PYISELONEPGRIT	NO	IMPROVED & DISCHARGED
37	SRIRENGANAYAKI	WNL	1-2 PUS CELL	E.COLI GROWN	NOT DONE	B/L PYELONEPHRITIS	B/L PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
38	CHRISTY	WNL	5-6 PUS CELLS	NO GROWTH	NOT DONE	RIGHT PYELONEPHRITIS	RIGHT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED

S.NO	NAME	LFT	URINE DEPOSITS	URINE CULTURE	URINE ACETONE	USG ABDOMEN	CT ABDOMEN	NEED FOR PCN/NEPHRECTOMY	OUTCOME
39	ATHIMOOLAM	WNL	1-2 PUS CELL	E.COLI GROWN	POSITIVE	LEFT PYELONEPHRITIS	LEFT EMPHYSEMATOUS PYELONEPHRITIS	PCN DONE	IMPROVED & DISCHARGED
40	BALAMMAL	WNL	5-6 PUS CELLS	NO GROWTH	NEGATIVE	B/L PYELONEPHRITIS	B/L PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
41	REKHA	WNL	PLENTY OF PUS CELLS	E.COLI GROWN	POSITIVE	LEFT PYELONEPHRITIS	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
42	CHELLAMMAL	WNL	5-6 PUS CELLS	NO GROWTH	NEGATIVE	B/L PYELONEPHRITIS	B/L PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
43	KEERTHI	WNL	5-6 PUS CELLS	NO GROWTH	NOT DONE	LEFT PYELONEPHRITIS	LEFT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
44	SUMATHI	ALTERE	PLENTY OF PUS CELLS	NO GROWTH	POSITIVE	B/L EMPHYSEMATOUS PYELONEPHRITIS	B/L EMPHYSEMATOUS PYELONEPHRITIS,RIGHT RENAL ABSCESS	PCN DONE	IMPROVED & DISCHARGED
45	SASI	WNL	5-6 PUS CELLS	NO GROWTH	NEGATIVE	RIGHT KIDNEY ENLARGED	RIGHT PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
46	MADASAMY	WNL	PLENTY OF PUS CELLS	E.COLI GROWN	NOT DONE	LEFT RENAL CALCULUS,LEFT HUN	LEFT RENAL CALCULI,LEFT PYELONEPHRITIS,LEFT HUN	NO	IMPROVED & DISCHARGED
47	RAMU	WNL	5-6 PUS CELLS	NO GROWTH	NOT DONE	LEFT PYELONEPHRITIS	B/L PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
48	LAKSHMI	WNL	PLENTY OF PUS CELLS	E.COLI GROWN	LEFT PYELONEPH	LEFT PYELONEPHRITIS	LEFT EMPHYSEMATOUS PYELONEPHRITIS	NO	IMPROVED & DISCHARGED
49	CHELLARANI	WNL	1-2 PUS CELL	E.COLI GROWN	B/L PPYELONEPH	B/L PYELONEPHRITIS		NO	IMPROVED & DISCHARGED

S.NO	NAME	LFT	URINE DEPOSITS	URINE CULTURE	URINE ACETONE	USG ABDOMEN	CT ABDOMEN	NEED FOR PCN/NEPHRECTOMY	OUTCOME
50	SURESH	WNL	5-6 PUS CELLS	E.COLI GROWN	POSITIVE	LEFT EMPHYSEMATOUS PYELONEPHRITIS	LEFT EMPHYSEMATOUS PYELONEPHRITIS	NO	IMPROVED & DISCHARGED

S.NO	NAME
1	LEELAVATHY
2	SYED ALI FATHIMA
3	MEENA
4	MARIAPPAN
5	RAMALAKSHMI
6	PETCHIAMMAL
7	SHENBAGAM
8	BALAPUSHPAM
9	SAROJA
10	CHELLAMMAL
11	SARAVANA KUMARI
12	SATHIYAPPAN
13	ULAGESHWARI
14	KANAGAMMAL
15	ESAKKIRAJ

S.NO	NAME
16	ESAKKIRAJ
17	MYDEEN
18	SEENISAMY
19	PUSHPAM
20	VALLIAMMAL
21	PARVATHI
22	ALAGARSAMY
23	AMALIN ROSY
24	AMUTHA
25	HELEN
26	SOWMYA

S.NO	NAME
27	PETCHIAMMAL
28	PERACHI
29	VEERAMANI
30	KARUPPASAMY
31	VANAJA
32	PAPPATHI
33	JANAKI
34	VALLIAMMAL
35	RAJATHI
36	RANI
37	SRIRENGANAYAKI
38	CHRISTY

S.NO	NAME
39	ATHIMOOLAM
40	BALAMMAL
41	REKHA
42	CHELLAMMAL
43	KEERTHI
44	SUMATHI
45	SASI
46	MADASAMY
47	RAMU
48	LAKSHMI
49	CHELLARANI

S.NO	NAME
50	SURESH