"A STUDY ON CLINICAL ,MICROBIOLOGICAL,RADIOLOGICAL ABNORMALITIES IN ACUTE PYELONEPHRITIS "

Dissertation Submitted to

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY
Chennai

In partial fulfillment of the regulations for the award of the degree of

M.D. BRANCH – I
(GENERAL MEDICINE)
REGISTRATION NUMBER: 200120101025

DEPARTMENT OF GENERAL MEDICINE
STANLEY MEDICAL COLLEGE, CHENNAI
THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY
TAMILNADU, INDIA
2022

CERTIFICATE

This is to certify that the dissertation titled **"A STUDY ON CLINICAL., MICROBIOLOGICAL, RADIOLOGICAL ABNORMALITIES IN ACUTE PYELONEPHRITIS "** presented here is the original work done by Dr. SANDESH JADHAV in the Department of General Medicine, Government Stanley Hospital, Stanley Medical College, Chennai – 600 001, in partial fulfillment of the University rules and regulation for the award of M.D. DEGREE BRANCH I GENERAL MEDICINE – under my guidance and supervision during the academic period from 2020 – 2021.

Guide

Prof. Dr.G.RANJANI M.D.,
Department of Internal Medicine,
Govt Stanley Medical College
Chennai – 600 001.

Dr. G. RANJANI, M.D.,
Professor of Medicine
Govt. Stanley Medical College and Hospital
Chennai-600 001.
Reg. No. 50382

HOD

Prof. Dr.S.PARIMALASUNDARI M.D.
Department of Internal Medicine,
Govt Stanley Medical college
Chennai -600 001

Professor and Head of the Department
Department of Medicine
Govt. Stanley Medical College & Hospital
Chennai - 600 001

Prof. Dr. P. Balaji, MS., FRCS., PhD., FCLS,
Dean,
Government Stanley Medical
College and Hospital, Chennai- 600 001

DEAN
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

DECLARATION

I, Dr. Sandesh Jadhav solemnly declare that Dissertation titled **"A STUDY ON CLINICAL,MICROBIOLOGICAL,RADIOLOGICAL,ABNORMALITIES IN ACUTE PYELONEPHRITIS"** is a bonafide work done by me at Government Stanley Hospital Chennai, during April 2021 to September 2021 under the guidance and supervision of Prof. Dr. Ranjani G. M.D., Professor of Medicine, Government Stanley Hospital, Chennai. I also declare that this bonafide work or a part of this work was not submitted by me or any other for award degree or diploma to any other university, board either in India or abroad.

This dissertation is submitted to the Tamil Nadu DR. M.G.R Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree (Branch – I) in General Medicine.

Place: Chennai

Signature of the candidate

Date: 21.11.2022

(Dr SANDESH JADHAV)

Reg. No:200120101025

CERTIFICATE – II

This is to certify that this dissertation work titled ""**A STUDY ON CLINICAL , MICROBIOLOGICAL ,RADIOLOGICAL ABNORMALITIES IN ACUTE PYELONEPHRITIS** of the candidate **Dr. SANDESH JADHAV** with registration Number **200120101025** for the award of **M.D. DEGREE** 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 pages and result shows **Fifteen Percentage** of plagiarism in the dissertation.

Dr. G. RANJANI, M.D.,
Professor of Medicine
Govt. Stanley Medical College and Hospital
Chennai-600 001.
Reg. No: 50382

Guide Sign with Seal

ACKNOWLEDGEMENT

I owe my thanks to the Dean, Government Stanley Medical College and Hospital, **Prof. Dr. P. Balaji, MS.,** for allowing me to avail the facilities needed at his disposal for my dissertation work.

I am very grateful to **Prof. Dr S. CHANDRASEKAR, M.D.,** Professor and Head of the Department of General Medicine, Government Stanley Medical College and Hospital for permitting me to do this study and for his encouragement.

I am very grateful to my unit chief **Prof. Dr G. RANJANI, M.D**., Government Stanley Medical College & Hospital for her valuable assistance and guidance.

I am extremely thankful to Assistant Professors **Dr. N. Karunakaran, M.D.,** and **Dr. Balamurugan. A, M.D,** for their guidance and encouragement.

I am also thankful to my colleagues for their valuable help rendered to complete this study. My great thanks to the patients who cooperated for this study, without whom, this study could not have been undertaken.

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01
INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK	: "A STUDY ON CLINICAL, MICROBIOLOGICAL PROFILE, RADIOLOGICAL ABNORMALITIES IN ACUTE PYELONEPHRITIS"
PRINCIPAL INVESTIGATOR	: DR. SANDESH JADHAV,
DESIGNATION	: PG IN GENERAL MEDICINE,
DEPARTMENT	: DEPARTMENT OF GENERAL MEDICINE

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 24.03.2021 at the Council Hall, Stanley Medical College, Chennai-1 at 11 am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI.

Ouriginal
by Turnitin

Document Information

Analyzed document	THESIS (2).docx (D153404050)
Submitted	12/14/2022 6:54:00 PM
Submitted by	Dr Sandesh Jadhav
Submitter email	sjsandesh2@gmail.com
Similarity	15%
Analysis address	sjsandesh2.tnmg@analysis.urkund.com

Sources included in the report

TABLE OF CONTENTS

ABBREVATIONS

- DM- Diabetes Mellitus
- NIDDM- Non insulin dependent diabetes mellitus
- BMI- Body mass index
- HbA1C- Glycosylated hemoglobin
- HOMA B- Homeostatic model assessment beta
- FBS- Fasting Blood Sugar
- PPBS- Post Prandial Blood Sugar
- LDL- Low Density Lipoprotein
- HDL- High Density Lipoprotein
- VLDL- Very Low Density Lipoprotein
- GLP- Glucagon Like Peptide
- SGLT 2- Sodium Glucose Cotransporter 2
- DPP4- Dipeptidyl peptidase 4
- GDM- Gestational Diabetes Mellitus
- ESRD- End Stage renal Disease
- DSPN- Distal Symmetric Peripheral Neuropathy
- IGT- Impaired Glucose Tolerance
- CKD- Chronic Kidney Disease
- PAD- Peripheral Arterial Disease
- MM-Medical Management

INTRODUCTION

Acute pyelonephritis (APN) is an disease of the renal parenchyma tissues that culminate in the occurrence of infection in the renal parenchyma or perinephric tissue. 1 A patient infected with Acute pyelonephritis exhibit promising prognosis with medical management (MM), while APN deserves specific attention (owing to its life-threatening situations) under MM and or surgical management. Mortality from APN is primarily attributable to septic complications. APN was marked and noted for heightened mortality rate (as high as 20%), but, in the recent decades, advancements in management techniques has curtailed the mortality. APN 2 If not treated early, it may lead to fulminant sepsis and, therefore, carries a high mortality. The subjects predominantly presented with fevers and flank pain.2

ASSOCIATED :FACTORS:

Diabetes Mellitus (DM) is the prime associated factor screened together with APN. More than 95% of patients diagnosed

with APN have lurking unrestrained DM. Other established co-factors correlated with the advancement of APN; drug abuse; neurogenic bladder; alcoholism and few anatomic anomalies. There is a predominance of APN in females; as the female to male ratio established in many independent investigations is 6:1. Heightened susceptibleness to UTI portrayed to be the reason for this larger and contrary occurrence in female subjects and for the general population the risk of acquiring APN auxiliary to a urinary tract obstruction is under 40%. Even though the occurrence of APN have been proven and published as a global phenomenon, it appears to be more common in Asia countries with many investigations originated from there.3 3 Figure1: Pathogenesis due

toEColi

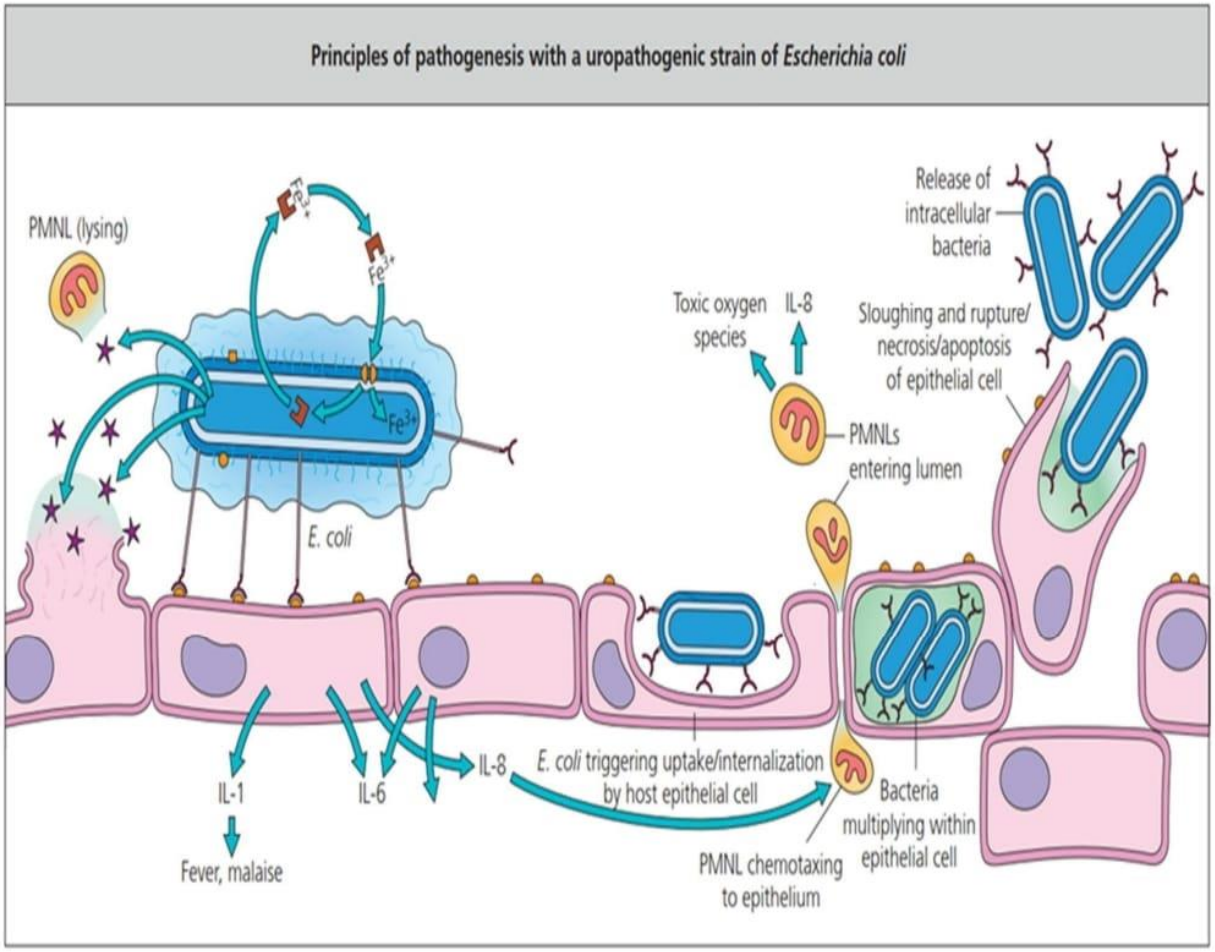


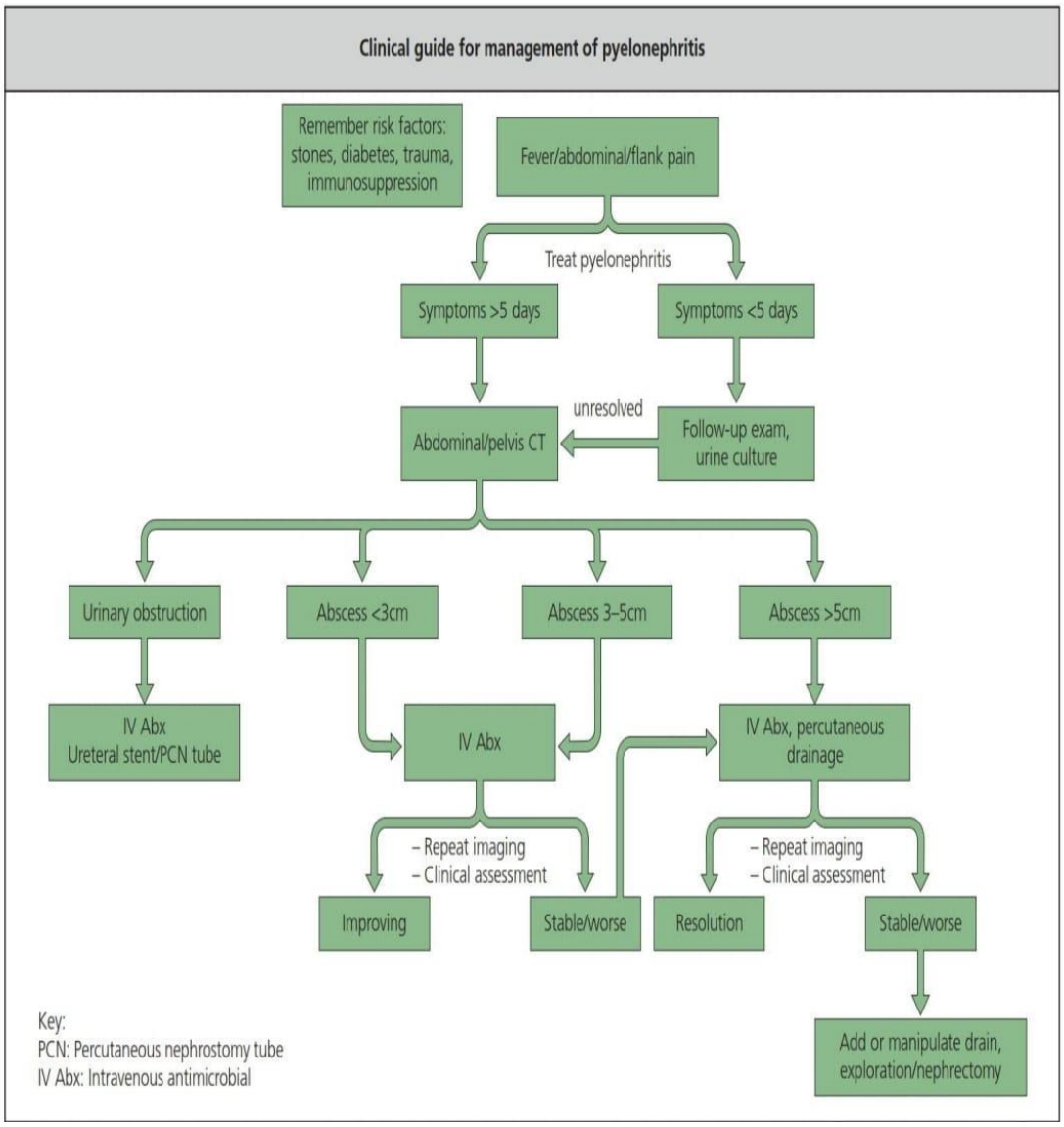
Figure1: Pathogenesis due to *E Coli*

AETIOLOGY AND PATHOGENESIS:

APN is a , necrotizing form of acute bacterial pyelonephritis and Escherichia coli remains the most common causative pathogen; the organism has been isolated from urine

samples across more than 70% of the established cases, but there is proof of Proteus mirabilis, Klebsiella pneumoniae, Streptococcus and Staphylococcus being the responsible microbes for instigating APN. 4 Anaerobic microbes Clostridium septicum, Candida albicans, Cryptococcus neoformans and Pneumocystis jiroveci have also been proven to instigate APN . Bacteraemia is expounded in more than half of the reported cases of APN. Radiological features are mandatory to rule out the possibility of APN presence (figure 1).4 Assorted and an array of attributes are correlated in the pathogenesis of APN; heightened concentration of glucose in the tissues ; debilitated vascular blood distribution; deteriorated host immunity and obstruction(s) noted in the urinary tract. An elevated tissue glucose compounded with disrupted blood supply to the kidneys (usual in DM subjects), further aid anaerobic metabolism. Gram-negative facultative anaerobic micro-organisms (E. coli) generate gas owing to the fermentation of glucose and lactate, culminating in the production and accumulation of CO2 and H2. Radiologically guided needle aspiration of the gases released by the tissues was done by Huang and Tseng, N2 and O2 have also

been reported with rare instances of NH4, CO and CH4. Infection might spread beyond the inflamed site to the sub-capsular, perinephric and pararenal spaces, rare case is reported with gas in the scrotal sac and spermatic cord. Pathological examination of the kidney exhibit countenance of abscess development, micro-macro-infarctions, vascular thrombosis.



MANAGEMENT OF ACUTE PYELONEPHRITIS

The outcome of APN can be extreme and life-threatening if not diagnosed and treated at an earlier stage. The element for mortality in APN is primarily attributed to septic complications. Up to 45% of the cases with APN have underlying uncontrolled DM. The risk of developing APN secondary to a urinary tract obstruction is about 25-40%. E coli deemed the prime microbe to be associated and isolated on urine or

pus cultures (70%).8 9 Many investigations published have proven that patients successfully treated with percutaneous drainage (PCD) in addition to medical management (MM), has a great impact in lowering the mortality rates. PCD should be performed on patients with localized areas of gas and in functioning renal tissue. The treatment strategies include medical management MM alone, PCD+MM, MM+emergency nephrectomy, and PCD+MM+ emergency nephrectomy .9 In studies of cases with APN wherein they were managed with MM and or PCD, ultimately nephrectomy was deemed mandatory and, but with the combined therapy management in these cases, the reported mortality is just 6.6%. Nephrectomy in subjects with APN achieved via intervention (radical or laparoscopic).

6).

Venue	Clinical Situation	Drug	Route	Dose/ Frequency	Duration	Adjunctive Therapy
Outpatient	Uncomplicated infection <10% regional fluoroquinolone resistance	Ciprofloxacin Ciprofloxacin Levofloxacin	Oral	500mg BD 1000mg OD 750mg OD	7days 7days 5days	Single IV ciprofloxacin 400mg or ceftriaxone 1g
	Uncomplicated infection >10% regional fluoroquinolone resistance	Ciprofloxacin Ciprofloxacin Levofloxacin Strongly consider adjunctive agent	Oral	500mg BD 1000mg OD 750mg OD	7days 7days 5days	Single IV ceftriaxone 1g or 24h dosage of aminoglycoside
	Uncomplicated infection, culture data available	Trimethoprim-Sulfamethoxazole	Oral	160/800mg BD	14days	None
Inpatient	Unable to tolerate oral intake, complicated infection, clinical instability	Ciprofloxacin Ceftriaxone Carbapenems Aminoglycoside± ampicillin or extended spectrum penicillin	IV	400mg BD 1g BD 500mg BD 250mg±500 mg BD 250mg BD	Until clinical improvement or cultural data available	

Figure 5: Antimicrobial Therapy for Pyelonephritis

When to image for pyelonephritis

Clinical Risk Factors	Concerning Symptoms	Abnormal Laboratory Results
Diabetes	Symptoms >72 h, despite antibiotics	Urine pH >7.0
Immunocompromised	Acute renal colic	GFR <40
Elderly	Unstable patient (AMS, low BP)	50% decline in renal function
History of stone disease	Recurrent symptoms after completion of antibiotics	—

Abbreviations: AMS, altered mental status; BP, blood pressure; GFR, glomerular filtration rate.

Figure 6: Conditions under which to advice for scanning

AIM OF THE STUDY

To analyse different clinical presentations, laboratory profile, and outcome in Acute pyelonephritis.

OBJECTIVE

1. To analyse age and sex distribution, risk factors and clinical features of Acute pyelonephritis.
2. To study the causative organisms and sensitivity pattern.

METHODOLOGY AND MATERIALS

Study design **:**

Acute pyelonephritis (APN) is a necrotizing infection of renal parenchyma and surrounding tissues by microbes like E coli, Klebsiella, Proteus with increased case fatality rate. Major risk includes Diabetes Mellitus (DM), immunocompromised state, renal stone(s), prior surgery or instrumentation of urinary tract. Patients may have varying presentations like fever, dysuria, loin pain, altered sensorium, AKI, shock. APN was a rare condition but not so uncommon in recent years, with early diagnostics, and with the aid of computed assisted tomography, medical management (MM) with appropriate antibiotics, relieving urethral obstructions, surgical drainage and or nephrectomy, mortality rate can be greatly minimized.

Setting:

A prospective observational study to analyse clinical, laboratory profile, and outcome of patients admitted in medical, nephrology and urology wards, Government Stanley Medical College Hospital, Chennai, over a period between April 2021 to September 2021. 42

Approval :

The current study was approved by the ethical committee of Government Stanley Medical College Hospital, Chennai (April 2021)

Study population A sample size of 80 , of APN patients admitted to medical, nephrology and urology wards. The patients were interviewed for demographic and other details like age, sex, past medical history of comorbidity, along with presenting complaints were noted. Further these patients were subjected to physical examination for clinical signs and these were recorded separately in a separate proforma. Lab parameters were analysed for arriving at the statistical significance and correlation.

Inclusion criteria Age ≥18, sex, APN patients based on USG/ CT KUB.

Exclusion criteria: 1.Denial

2.patients with existing renal pathology

3.pregnant women

Consent

Informed consent was procured from patients for their inclusion in this study and no ethical issues was involved.

Statistical analysis

Statistical analysis was done using Minitab 17 and Microsoft Excel. Quantitative factors were computed with means+/SD and the qualitative factors in percentage.Informed consent was taken from all the patients or his/her legally valid immediate relative.

Other information

This study had no financial support from any source and the current work did not impose any additional financial burden to the patient. A complete physical check-up was performed on patients, inclusive of noting their pulse, blood pressure, respiratory rate etc. 44

Laboratory parameters

Serum creatinine, Blood Sugar, HbA1c, Platelet count, WBC, LFT, Blood culture, Urine culture, Urine acetone

. Imaging

Ultrasound KUB, CT KUB

REVIEW OF LITERATURE

Acute pyelonephritis is a bacterial infection causing inflammation of the kidneys and is one of the most common diseases of the kidney. Pyelonephritis occurs as a complication of an ascending urinary tract infection (UTI) which spreads from the bladder to the kidneys and their collecting systems. Symptoms usually include fever, flank pain, nausea, vomiting, burning on urination, increased frequency, and urgency. The 2 most common symptoms are usually fever and flank pain. Acute pyelonephritis can be divided into uncomplicated and complicated. Complicated pyelonephritis includes pregnant patients, patients with uncontrolled diabetes, kidney transplants, urinary anatomical abnormalities, acute or chronic kidney failure, as well as immunocompromised patients, and those with hospital-acquired bacterial infections. It is important to make a distinction between complicated and uncomplicated pyelonephritis, as patient management and disposition depend on it.[1]

ETIOLOGY :

The main cause of acute pyelonephritis is gram-negative bacteria, the most common being Escherichia coli. Other gram-negative bacteria which cause acute pyelonephritis include Proteus, Klebsiella, and

Enterobacter. In most patients, the infecting organism will come from their fecal flora. Bacteria can reach the kidneys in 2 ways: hematogenous spread and through ascending infection from the lower urinary tract. Hematogenous spread is less common and usually occurs in patients with ureteral obstructions or immunocompromised and debilitated patients. Most patients will get acute pyelonephritis through ascending infection. Ascending infection happens through several steps. Bacteria will first attach to urethral mucosal epithelial cells and will then travel to the bladder via the urethra either through instrumentation or urinary tract infections which occur more frequently in females. UTIs are more common in females than in males due to shorter urethras, hormonal changes, and close distance to the anus. Urinary tract obstruction caused by something such as a kidney stone can also lead to acute pyelonephritis. An outflow obstruction of urine can lead to incomplete emptying and urinary stasis, which causes bacteria to multiply without being flushed out. A less common cause of acute pyelonephritis is vesicoureteral reflux, which is a congenital condition where urine flows backward from the bladder into the kidneys.

PATHOPHYSIOLOGY:

Pyelonephritis largely occur due to bacterial ascension from the lower urinary tract. Although the hematogenous, lymphatic transmission is

found, but lymphatic transmission reported as rare in healthy subjects. The hematogenous spread arise due to the establishment of staphylococcal bacteremia or candidemia, while lymphatic transmission happens due to the candid development of the local infection. Similar to the traits with lower urinary tract contagion, Escherichia coli is the prime causative agent mostly associated with emphysematous pyelonephritis accounting for 70–87% of all such infections, followed by Klebsiella, Enterococcus and Proteus species. E. coli over the years has developed several significant factors that not only proved virulent, but also abide it in its adherence, ascension, and escape the immune system. Both the pathogenic and non-pathogenic species types were reported to have established itself in the genitourinary tract due to production of a marker: adhesin, especially the mannose susceptible, type 1 pili; adhesins largely expressed among all known E. coli, pivotal to colonization as well spreading infection. In array of studies with suitable animal models (bladder inoculated) these microbial stains tracted higher colonization than their counterparts. Augmentation with anti-type 1 pili antibody and mannose-sourced antagonistic inhibitors bestowed a protective action against the occurrence of urinary 17 tract infection in mice. Type 1 pili also noted for its capacity to empower the intracellular displacement of the bacteria, triggering the formulation of intracellular bacterial communities (IBC). IBC protect these microbes from assorted

antimicrobials and inherent host immune response, and also provisioning a reservoir for time-triggered ‘fluxing’ or shedding of these pathogens into the bladder, invigorating surface colonization after initial escape from the immune system. Mannose-tolerant types collogue tropism in the kidney and are asseverated by most pyelonephritogenic strains of uropathogenic E. coli (MTEC). MTEC- adhesin are largely adhering/ anchoring type and are hence predominant in retrograde ureteral ascent. In an independent investigation, this was noted in 91% of MTEC strains contrary to just 19% of cystitis-correlated strains and 14% of pertaining to asymptomatic bacteriuria.13,14 Bacteria anchored to the urothelium evokes an inflammatory torrent that triggers the discharge of cytokines (IL)-1, IL-6, IL-8 and TNF-tumor necrosis factor, together with the commencement of neutrophils and lymphocytic action via complementation. This culminates in the accumulation of ROS-reactive oxygen species, leukotrienes, prostaglandins, other cytokines responsible for inflammation; coupled with bacterial signal molecules enhance tissue 18 damage, edema, and vasoconstriction in that infected area, culminating in the rise of enlarged kidney (owing to cortical swelling). Micro-abscesses with surrounding hyperemia can occur on the subcapsular surface; medulla presents yellow streaks denoting the occurrence of pus; discharge within the collecting ducts, subsequently the collecting system mucosa and submucosa, tubules and interstitium and edematous are

undulated with neutrophils; culminating in tubular necrosis and subsequent micro-abscess development within the mucosa and interstitium. Finally, these abscesses consolidate and integrate in to macroscopic abscesses. Macroscopic abscesses when developed can aggregate in the corticomedullary junction but also reported in the subcapsular and in the perirenal gaps. In DM cases with unmanaged glycemic regulation, acid fermentation of glucose generate gas within the renal parenchyma or collecting system, resulting in EPN; pyelitis. These patients are also at higher liableness for papillary necrosis in acute EPNs, arise of exuviate in papillary tissue and end with upper urinary tract block. Occasionally, the interstitial and tubular inflammation of acute EPN can cause deteriorated urinary function owing to the damage occurring in the distal and collecting tubules, though this dysfunction rarely manifests it is also reversible.15

CLINICAL FEATURES :

Pyelonephritis is classically characterized by fever and flank pain in the setting of a urinalysis suggestive of infection. However, the clinical presentation of this entity can vary widely depending on a number of host and pathogen factors; patients can range from afebrile and wellappearing to floridly septic, with hemodynamic instability and multiorgan dysfunction. Accurate and timely diagnosis can

be challenging. Thorough and pertinent history and physical examination is the first best tool. The typical history is a prodrome of malaise and low-grade fevers, with or without antecedent cystitis (dysuria, urinary frequency/ urgency, suprapubic pain), that evolves into aching flank pain, rigors, fevers, nausea and vomiting.16 On physical exam, the patient is ill- appearing, febrile and tachycardic with the pathognomonic finding of costovertebral angle (CVA) tenderness on percussion or deep palpation, either unilaterally or bilaterally. Presentations that deviate from this classic picture are common . Children, the elderly or debilitated, and even healthy individuals can fail to show some of the usual signs and symptoms or have nonspecific symptoms like nonlocalized abdominal, back or pelvic pain, predominant gastrointestinal symptoms, or isolated malaise. Patients with neurologic dysfunction or immunologic suppression can have deceptively benign presentations, without fever, 20 constitutional symptoms or pain, even in the face of severe infections. Spinal cord injured patients or those with spina bifida often exhibit generalized back or abdominal discomfort, increased incontinence or leakage between catheterizations, worsening spasticity or autonomic dysreflexia (spinal cord injuries at or above T6) as their predominant symptoms. Having a high index of suspicion for pyelonephritis is important in those who present with abdominal, flank pain and fevers.

Risk Factors for Complicated Acute Pyelonephritis[2]

Age
- Infants
- Elderly (> 60 years of age)

Anatomic/functional abnormality
- Polycystic kidney disease
- Horseshoe kidney
- Double ureter
- Ureterocele
- Vesicoureteric reflux

Foreign body
- Urinary, ureteric, or nephrostomy catheters
- Calculus

Immunosuppressed state
- Diabetes mellitus
- Sickle cell disease
- Transplantation
- Malignancy
- Chemoradiation
- HIV infections
- Corticosteroid use

Male sex
- Anatomic abnormalities
- Prostatic obstruction

Obstruction

Foreign body

Calculi

Bladder neck obstruction

Posterior urethral valve

Benign prostatic hypertrophy

Neurogenic bladder

Pregnancy

Miscellaneous

Inappropriate antibiotics

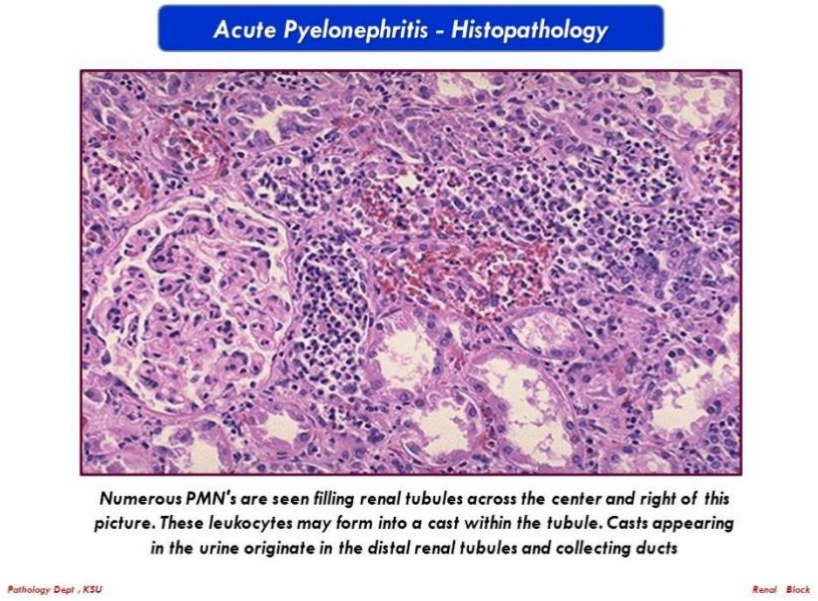
Resistant organisms

Instrumentation

HIV = human immunodeficiency virus.[2]

HISTOPATHOLOGY;

Histopathology will usually reveal necrosis or putrid abscess formation within the renal parenchyma. The renal tissues are infiltrated with neutrophils, macrophages, and plasma cells. However, the architecture is not completely disorganized.



HISTORY AND PHYSICAL EXAMINATION:

Acute pyelonephritis will classically present as a triad of fever, flank pain, and nausea or vomiting, but not all symptoms have to be present. Symptoms will usually develop within several hours or over the course of a day. Symptoms of cystitis, such as dysuria and hematuria, will be present in women usually. In children, common symptoms of acute pyelonephritis can be absent. Symptoms such as failure to thrive, fever, and feeding difficulty are most common in neonates and children under 2 years old. Elderly patients may present with altered mental status, fever, deterioration, and damage to other organ systems. On physical examination, the patient's general appearance will be variable. Some patients will appear ill and uncomfortable, while others may appear healthy. Patients will usually not appear toxic. When a patient is febrile, fever may be high, often over 103 F. Costovertebral angle tenderness is commonly unilateral over the affected kidney, but in some cases, bilateral costovertebral angle tenderness may be present. Suprapubic tenderness during the abdominal examination will vary from mild to moderate with or without rebound tenderness.

DIFFERENTIAL DIAGNOSIS:

When diagnosing acute pyelonephritis, keeping the differential broad is a wise idea. Physicians should consider other disorders as well when patients present with fever, flank pain, and costovertebral angle tenderness. Because symptoms can be variable (unilateral, bilateral, radiating, sharp, dull) and because pyelonephritis can progress to sepsis and shock, the differential diagnoses associated with pyelonephritis can be extensive. Common mimics of acute pyelonephritis can include but are not limited to:

- Appendicitis
- Abdominal abscess
- Nephrolithiasis
- Cholecystitis
- Urinary tract obstruction
- Pelvic inflammatory disease
- Pancreatitis
- Ectopic pregnancy

EVALUATION:

Though it is a clinical diagnosis, the heterogeneity of clinical presentations makes diagnostic tests useful adjuncts. The initial diagnostic evaluation should aim to efficiently identify patients with factors that necessitate urgent intervention and those who require hospitalization for close monitoring. The minimum laboratory testing to make the diagnosis of pyelonephritis include 1) a carefully collected midstream or catheterized urinalysis which demonstrates pyuria and bacteriuria; 2) urine culture collected prior to administration of antimicrobials to allow for pathogen identification and antimicrobial 22 susceptibility data. Additional laboratory tests should include

complete blood count, basic serum chemistry, and pregnancy testing in women of child- bearing age. The degree of leukocytosis can provide an assessment of the severity of infection. de Jager et al. (2010) found that peak leukocytosis correlated with the extent of renal parenchymal suppuration on computed tomography (CT). Patients with 25% renal parenchymal involvement had a mean white blood cell count (WBC) of 10.2cellsmL-1 compared with 16 cellsmL-1 in those with greater than 50% parenchymal involvement.17 Serum chemistry will aid to expose the electrolyte abnormalities and creatinine elevations that would heighten suspicion for significant dehydration or urinary obstruction, or hyperglycemia that could require correction. Blood cultures should be obtained in patients at increased risk of bacteremia. Imaging is required for diagnosis, can be useful in patients in whom another acute intra-abdominal process is possible, those with known or suspected complicating anatomic or host factors, recurrent infections, or those who fail to respond to appropriate therapy. Approximately 16% of patients with pyelonephritis will have an associated genitourinary (GU) tract abnormality. The choice of imaging should be informed by the clinical status of the patient at presentation and the leading clinical suspicion to be evaluated. Ultrasound is a good first choice in pregnant patients and the clinically unstable patients in whom 23 one needs to exclude complicating factors. It is widely available, costeffective and spares the risks of ionizing radiation. It can delineate upper and lower urinary tract anatomy, identify hydronephrosis, solid and cystic lesions, abscesses, some renal and bladder stones and evaluate for elevated post-void residual urine volume. It has poor sensitivity to detect the parenchymal changes in acute pyelonephritis and ureteral stones. The sensitivity and specificity for detecting urinary calculi in all locations with ultrasound is 78% and 31%, respectively.18-21 CT is the gold standard for urinary tract imaging and is an excellent choice for

fine anatomic detail, rapid identification of conditions that may necessitate intervention (e.g. urinary obstruction, renal abscess) and evaluation of alternative causes of fever and/or flank pain. Non-contrast CT scan is the gold standard for the identification of urinary calculi, while contrast-enhanced CT provides delineation of parenchymal changes in acute pyelonephritis and evolving abscess. Acute pyelonephritis can be characterized by enlargement and wedgedshaped regions of poor enhancement (i.e. striated nephrogram), with or without perinephric fat stranding. Abscess appears as a defined mass with a thickened, hyper-enhancing wall and low attenuation, centrally. CT is the best choice in patients who are acutely ill or clinically decompensating despite appropriate therapeutic measures (figure 9- 12).22,23 24 The drawbacks of CT include exposure to ionizing radiation and intravenous contrast agents, but in well-selected patients, the benefit may well outweigh the risks. It is important to note here that while ultrasound (US) remains the mainstay imaging modality in pregnancy, CT can be used safely in pregnant patients who are clinically ill or fail to respond to appropriate therapy and previously nondiagnostic methods

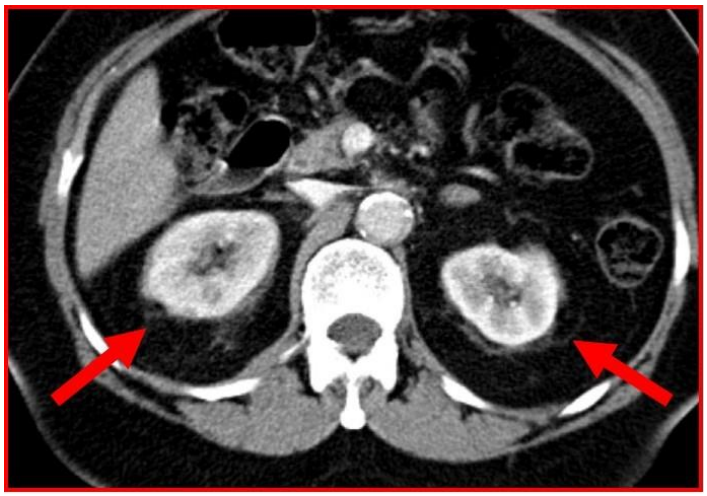


Figure shows fat stranding on both the kidneys

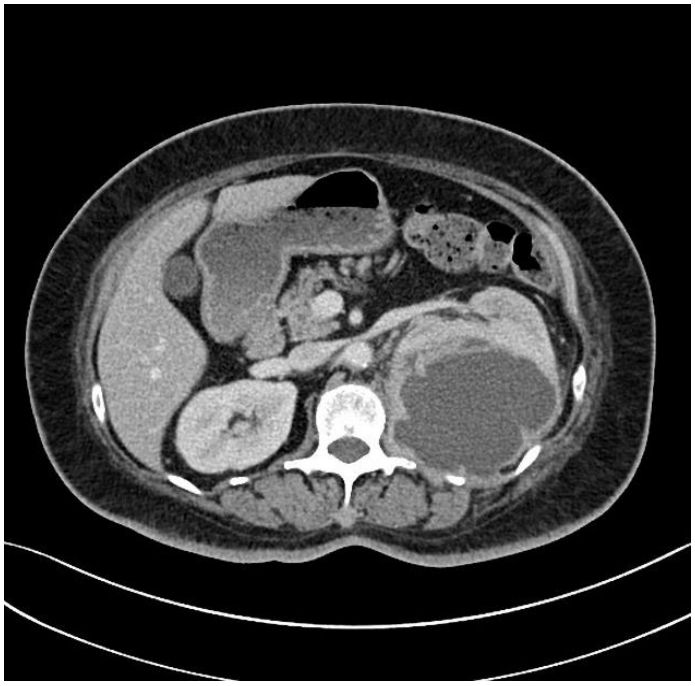


Figure showing Renal abscess in left kidney

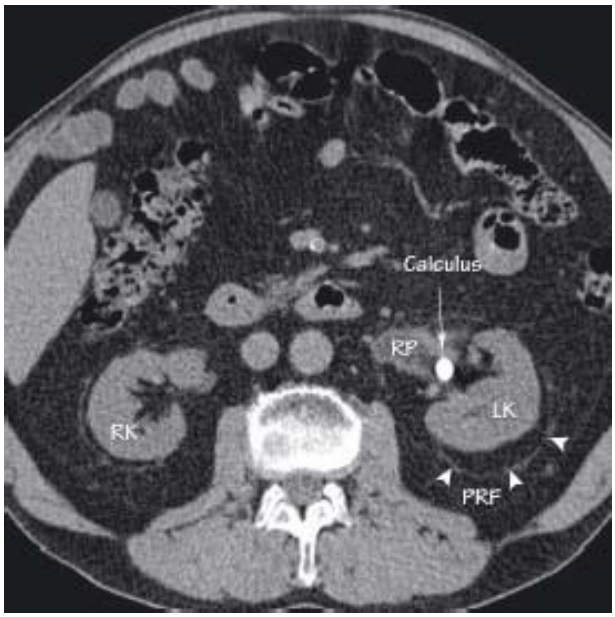
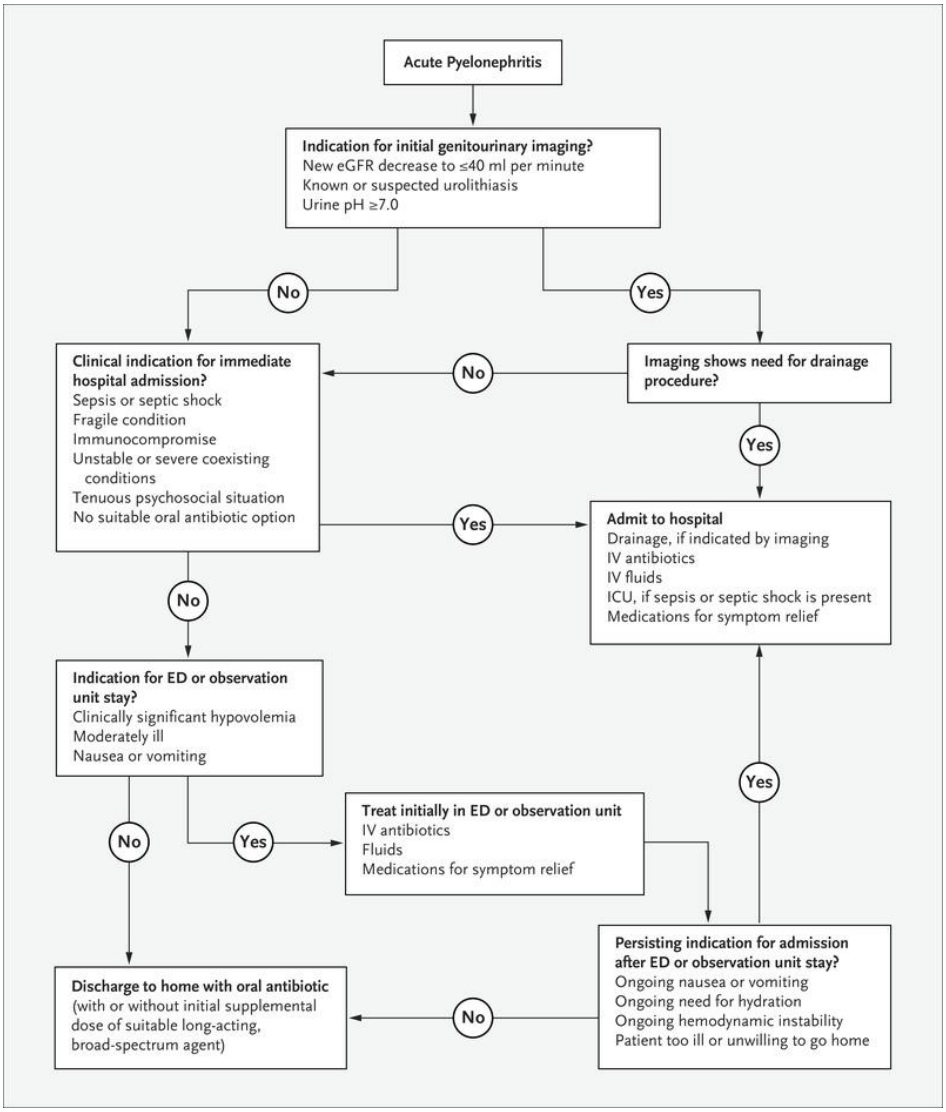


Figure shows calculi in the left pelvis causing mild hydronephrosis

MANAGEMENT :

Historically, most patients were admitted for parenteral antimicrobials and observation. Increasing evidence indicates that oral therapies and outpatient management can be safe and effective in properly selected patients. The decision of whether inpatient admission or outpatient management is to be pursued should be based on the patient's hemodynamic stability, ability to tolerate oral intake, adequacy of pain control, co-morbidities, capacity for compliance and access to care. Patients with hemodynamic instability, urinary obstruction, poorly controlled diabetes, poor response to appropriate outpatient therapy, or high likelihood for infection with multidrug-resistant organisms should be admitted.



ANTIMICROBIAL SELECTION :

The overwhelming majority of antimicrobials selected for initial treatment of pyelonephritis will be selected empirically. In general, the agent of choice should have good activity against the typical gramnegative bacteria that cause the majority of UTIs, and exhibit good tissue penetration and low rates of resistance in the geographical region in question. Many centres compile ‘hospital antibiograms’, summaries of hospital-specific patterns in antimicrobial resistance that

can aid in the selection of empiric therapy that is most effective in the specific region.31 For patients with complicated infections, consideration for coverage of gram-positive and/or multidrug-resistant gram-negative organisms should be considered. In patients who have a history of recurrent urinary tract infections, prior recent culture data can be informative as to patterns of infecting organisms and resistance. In general, fluoroquinolones are the agent of choice for the empiric therapy 32 of uncomplicated pyelonephritis to be managed on an outpatient basis. In areas where the rate of community-acquired uropathogen resistance to fluoroquinolones is ≤10%, 5–7 days of oral fluoroquinolone therapy is suggested. An initial intravenous dose was not shown to impact cure or clinical outcome. For regions with ≥10% fluoroquinolone resistance, an initial dose of long-acting antimicrobial, like ceftriaxone or a consolidated 24-hour dose of aminoglycoside followed by a 5–7day course of fluoroquinolone is recommended.32 Trimethoprim–sulfamethoxazole (TMP–SMX) exhibits good tissue penetration and activity against susceptible uropathogens but with widespread, high rates of resistance, it is no longer generally recommended as empiric therapy. When used as part of culture-directed therapy, a 14-day course is recommended. However, if one elects to use it empirically based on prior culture data or hospital antibiograms, an intravenous dose of ceftriaxone or consolidated aminoglycoside should be given. It is important to note

that when ciprofloxacin and TMP–SMX were compared in a randomized control trial in women presenting with mild-to-moderate pyelonephritis in an outpatient setting, ciprofloxacin had a significantly higher clinical cure rate (99% vs 89%) than TMP– SMX. This study's findings were also significant for greater success when using TMP–SMX if a single dose of intravenous ceftriaxone is given in conjunction with the outpatient oral therapy. For empiric 33 inpatient therapy, intravenous fluoroquinolones and extended spectrum cephalosporins (i.e. ceftriaxone) are appropriate for individuals in whom community-acquired infection is suspected. For those with suspected hospital-acquired infection, multidrug- resistant pathogens or other complicating factors, the selection of agents with a broader spectrum, such as aminoglycosides with ampicillin or carbapenems, is advised.33

EXPECTED CLINICALCOURSE :

Expected clinical course The majority of patients will achieve cure after a single course of appropriate antibiotics, but 9.6% of women and 5.7% of men will experience recurrence within 12 months. Of the patients who achieve clinical cure, most will begin to show signs of clinical improvement (i.e. normalization of blood pressure, resolution of malaise, improved oral intake, improvement in cystitis symptoms)

within 2 days of the initiation of therapy. Fever can persist for several days after the initiation of therapy. If after 48–72 hours there is no improvement in any clinical parameters, aggressive re-evaluation is required to consider alternative diagnoses, confirm appropriateness of therapy, or identify evolving complications such as abscess or unrecognized obstruction.34 A thorough history and physical examination, repeat laboratory testing (including blood cultures, serum chemistry, urinalysis and urine culture plus Gram stain) and urinary tract imaging should be performed. 34 In this clinical scenario, proceeding directly to CT is recommended. If imaging reveals a focal finding, like abscess or urinary obstruction, which warrants intervention, urologic consultation should be pursued. If culture data fails to be informative or reveals significant antimicrobial resistance, infectious disease consultation is prudent.[35]

OTHER STUDIES:

As per a study done by Colgan et al., 2011, intense pyelonephritis was attributed to bacterial infection of the renal pelvis and kidney (observed higher among young women in larger number in their study group; 83nos.), flank pain was found as routine; urinalysis could be the basic

tool to confirm the diagnosis in patients to initiate antibiotic therapy. Escherichia coli was the prime and ubiquitous microbe encountered among their study subjects. Imaging, with contrastenhanced computed tomography, was deemed avoidable or irrelevant with good response to antibiotic treatment, inpatient management were deemed necessary with cases who had severe illness with complications when suspected. Oral fluoroquinolones were the first recommended as the initial outpatient therapy (under community resistance <10%, when >10%, intravenous dose of ceftriaxone or gentamicin advised).37

Kiranmala et al., 2019 reported a study on the significance microbial profiles among UTI subjects (100 numbers) contrasting T2DM and normal population. T2DM patients with UTI had notably higher bacteriuria (32%) but with previous history of UTI additional 25% chances and catheterization impacted with added 16%. E. coli was the prime microbe isolated and portrayed sensitivity in the order of meropenem >netilmicin >amikacin >nitrofurantoin. Ceftriaxone was preferred choice of antibiotics assiduously selected despite of the prevailing low sensitivity of E. coli. 34

RESULTS AND DISCUSSION

STASTICAL OUTPUT OF THE SAMPLE POPULATION

A prospective observatory study to analyse clinical and laboratory profile of patients admitted in medical, nephrology and urology wards, Government Stanley Medical College Hospital, chennai, over a period between April 2021 to September 2021. A sample size of 80, of APN patients admitted to medical, nephrology, and urology wards. The patients were interviewed for demographic and other details like age, sex, past medical history of comorbidity, along with presenting complaints were noted. Further these patients were subjected to physical examination for clinical signs and these were recorded separately in a separate proforma. Lab parameters were analysed for arriving at the statistical significance and correlation. [46]

Total number of subjects used in the study = 80

Total number of males = 13

Total number of females = 67

Acute pyelonephritis (APN) is a necrotizing infection of renal parenchyma and surrounding tissues by microbes like E coli, Klebsiella, and Proteus with increased case fatality rate. Major risk includes Diabetes Mellitus (DM), immunocompromised state, renal

stone(s), prior surgery or instrumentation of urinary tract. Patients may have varying presentations like fever, dysuria, loin pain, altered sensorium, AKI, shock. With early diagnostics, and with the aid of computed assisted tomography, medical management (MM) with appropriate antibiotics, relieving urethritic obstructions, surgical drainage and or nephrectomy, mortality rate can be greatly minimized.

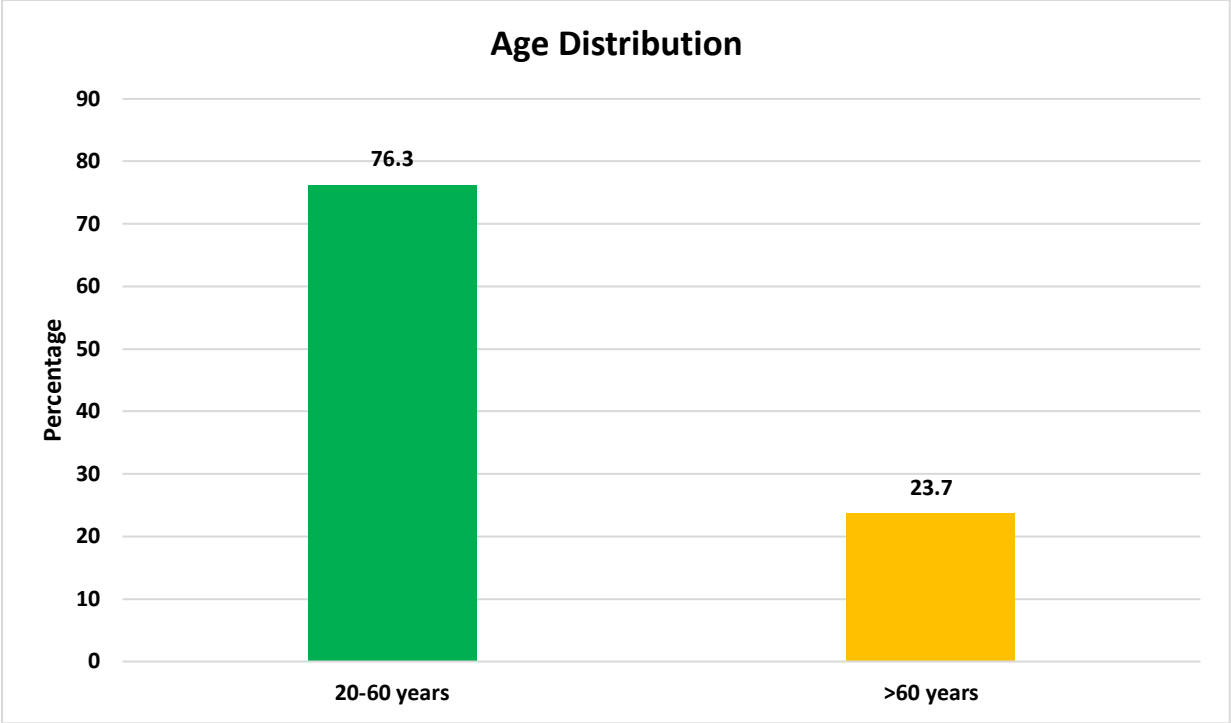
AGE DISTRIBUTION:

In our study, 76.3% were in the age group of 20-60 years and in the age category >60 years,23.7% were found

Table: Age Distribution of the study population

Age Distribution	Frequency (N)	Percentage (%)
20-60 years	61	76.3
>60 years	19	23.7

Figure: Age Distribution of the study population



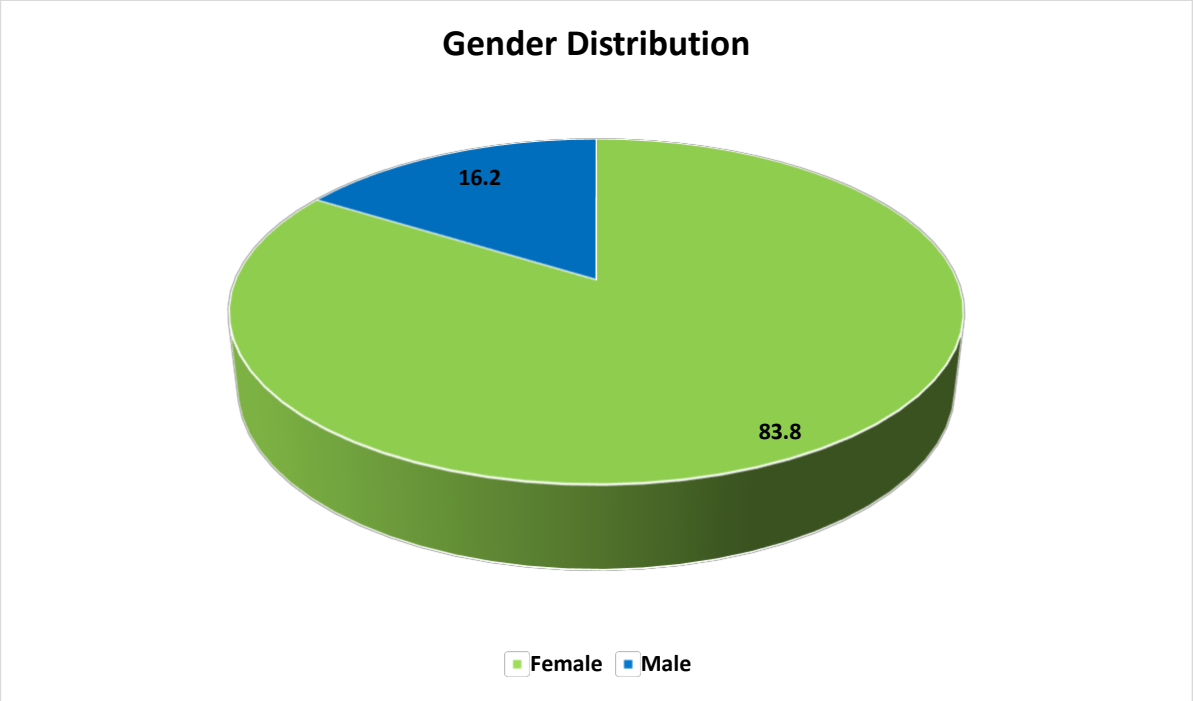
GENDER DISTRIBUTION:

In our study, 83.8% of the patients who were having pyelonephritis were females and the rest were males

Table: Gender distribution of the study population

Gender	Frequency (N)	Percentage (%)
Female	67	83.8
Male	13	16.2

Figure: Gender distribution of the study population



CLINICAL PROFILE

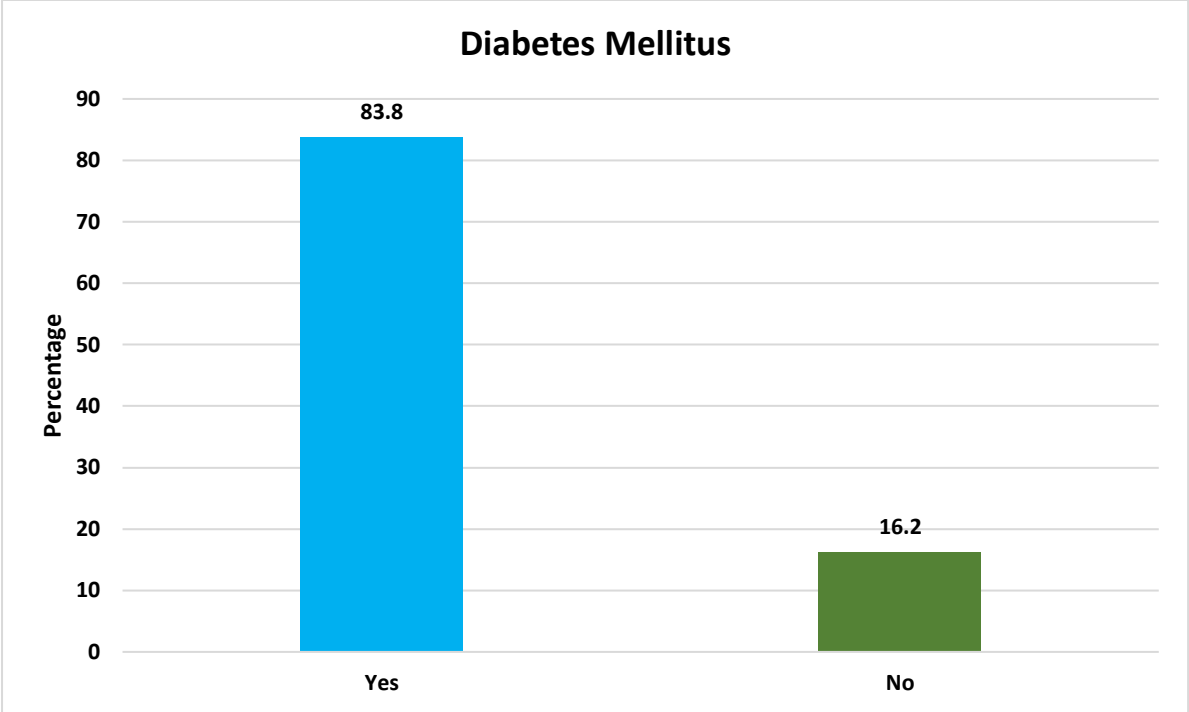
Diabetes Mellitus

In our study, 83.8% of them were having diabetes and 16.2% of them were non diabetics

Table: Diabetes Mellitus status of the study population

Diabetes Mellitus	Frequency (N)	Percentage (%)
Yes	67	83.8
No	13	16.2

Figure: Diabetes Mellitus status of the study population



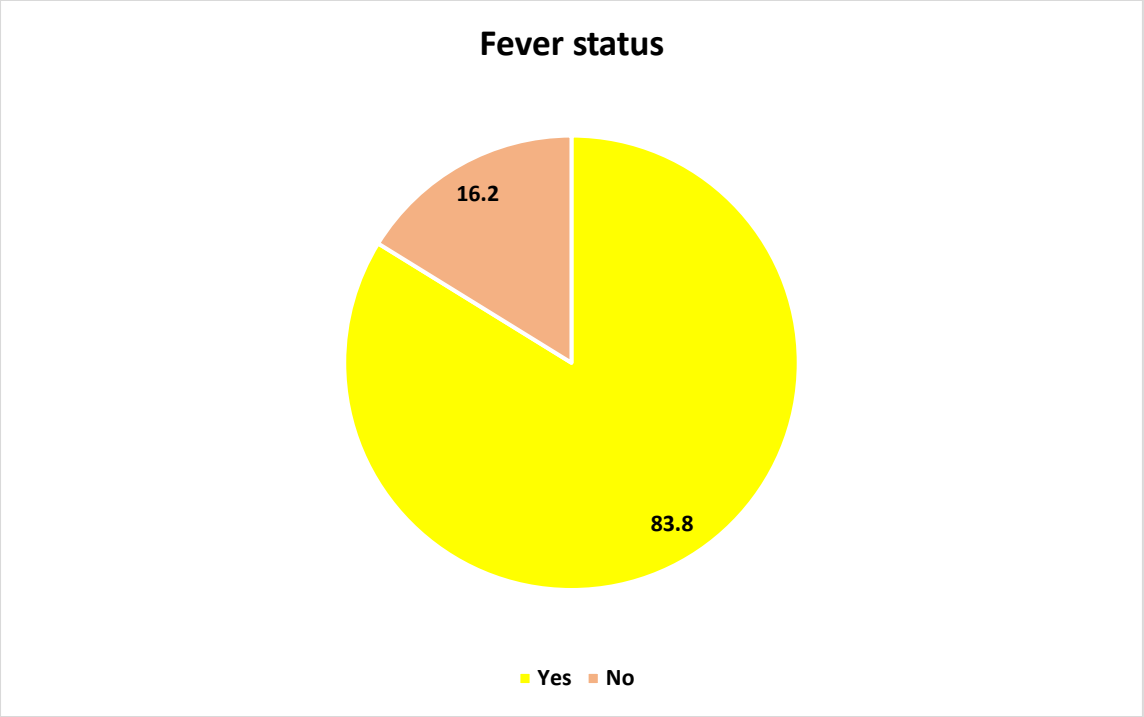
Fever

In our study, 83.8% had fever and 16.2% did not have fever symptom

Table: Fever status of the study population

Fever	Frequency (N)	Percentage (%)
Yes	67	83.8
No	13	16.2

Figure: Fever status of the study population



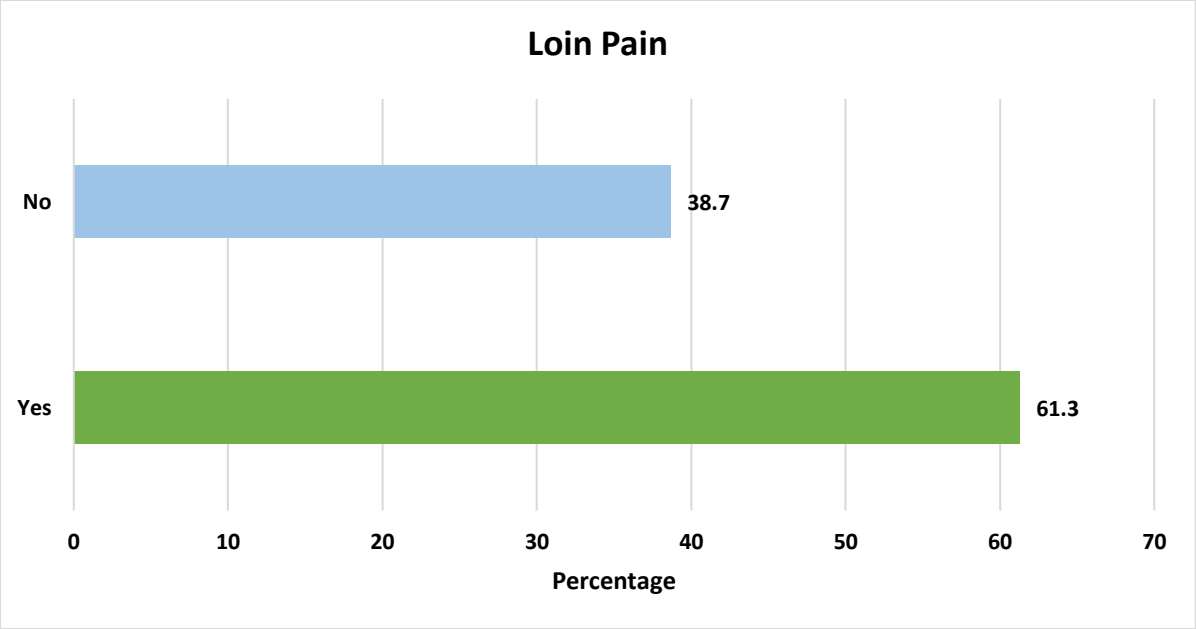
Loin Pain

The study population had loin pain which contributes to 61.3% and 38.7% did not have loin pain

Table: Loin pain complaint

Loin Pain	Frequency (N)	Percentage (%)
Yes	49	61.3
No	31	38.7

Figure: Loin pain complaint



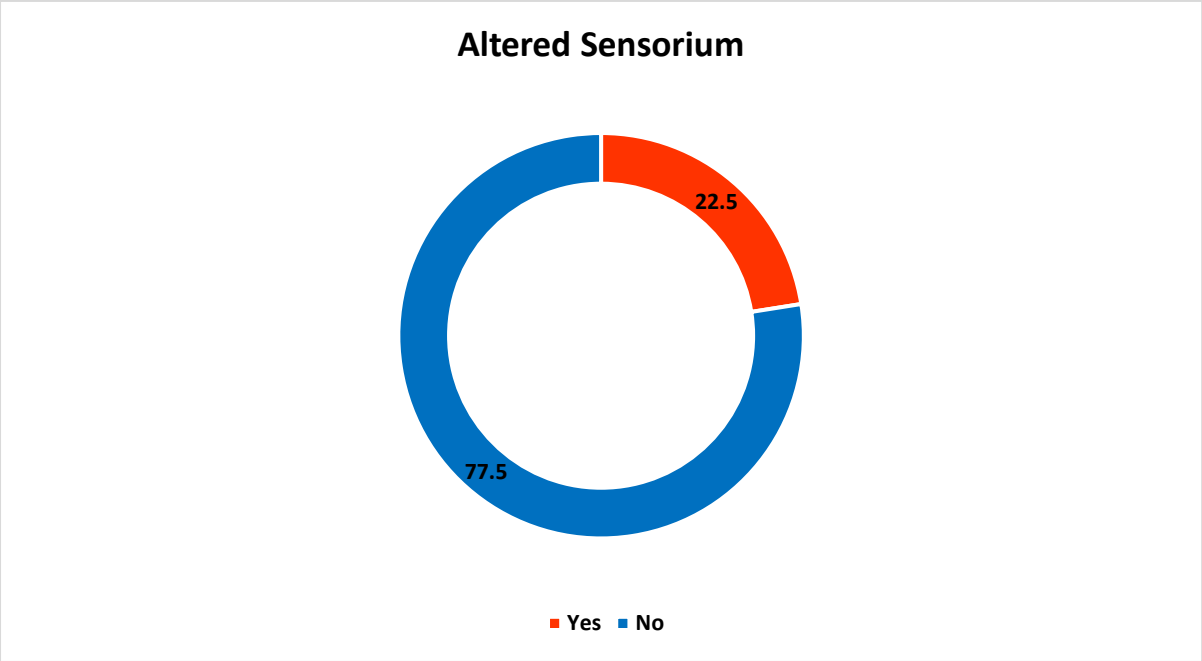
Altered Sensorium

22.5% of the study population had altered sensorium and 77.5% of them were having no such symptoms

Table: Altered Sensorium Status of the study population

Altered Sensorium	Frequency (N)	Percentage (%)
Yes	18	22.5
No	62	77.5

Figure: Altered Sensorium Status of the study population



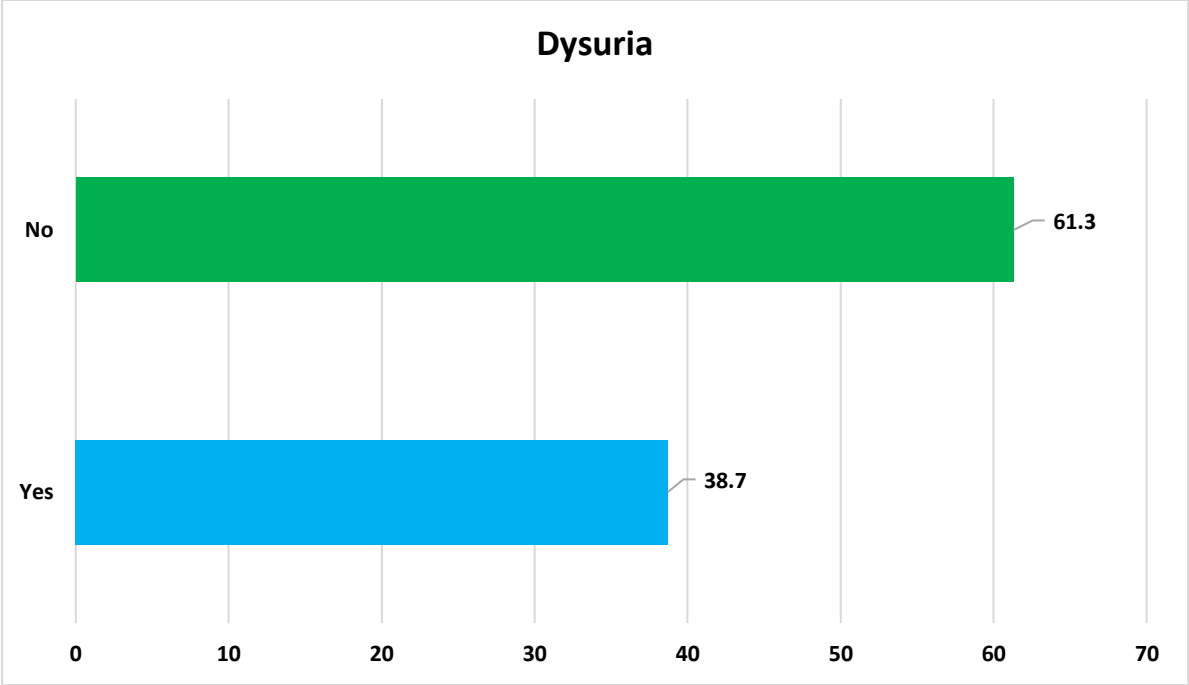
Dysuria

38.7% had dysuria and 61.3% had no difficulty during passing urine in our study

Table: Dysuria complaint of the study population

Dysuria	Frequency (N)	Percentage (%)
Yes	31	38.7
No	49	61.3

Figure: Dysuria complaint of the study population



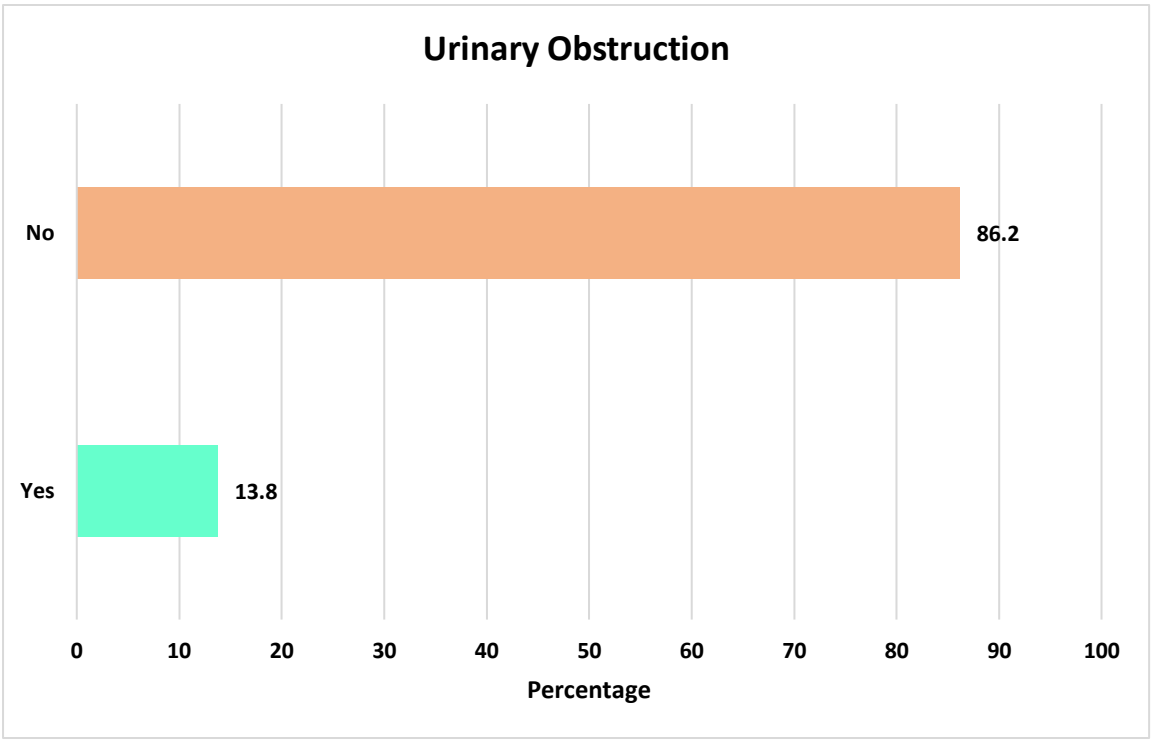
Urinary Obstruction

13.8% had urinary obstruction and 86.2% of them had signs and symptoms of urinary obstruction in our study

Table: Urinary Obstruction of the study population

Urinary Obstruction	Frequency (N)	Percentage (%)
Yes	11	13.8
No	69	86.2

Figure: Urinary Obstruction of the study population



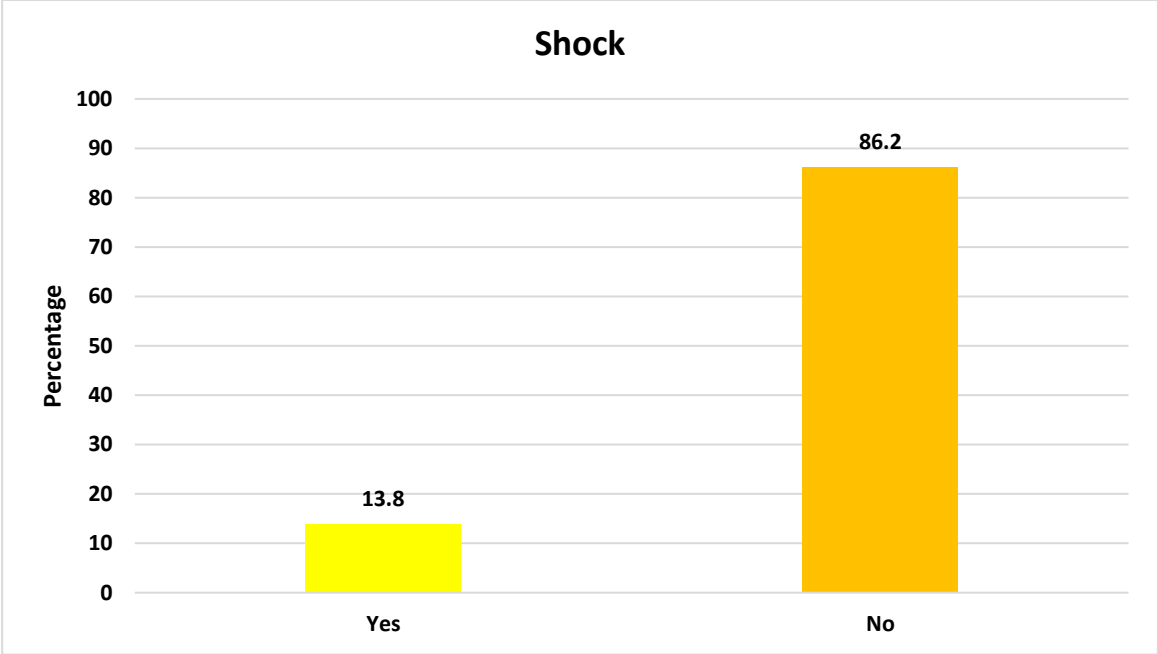
Shock

In our study, 13.8% of the study population had shock

Table: Shock status of the study population

Shock	Frequency (N)	Percentage (%)
Yes	11	13.8
No	69	86.2

Figure: Shock status of the study population



LABORATORY INVESTIGATIONS

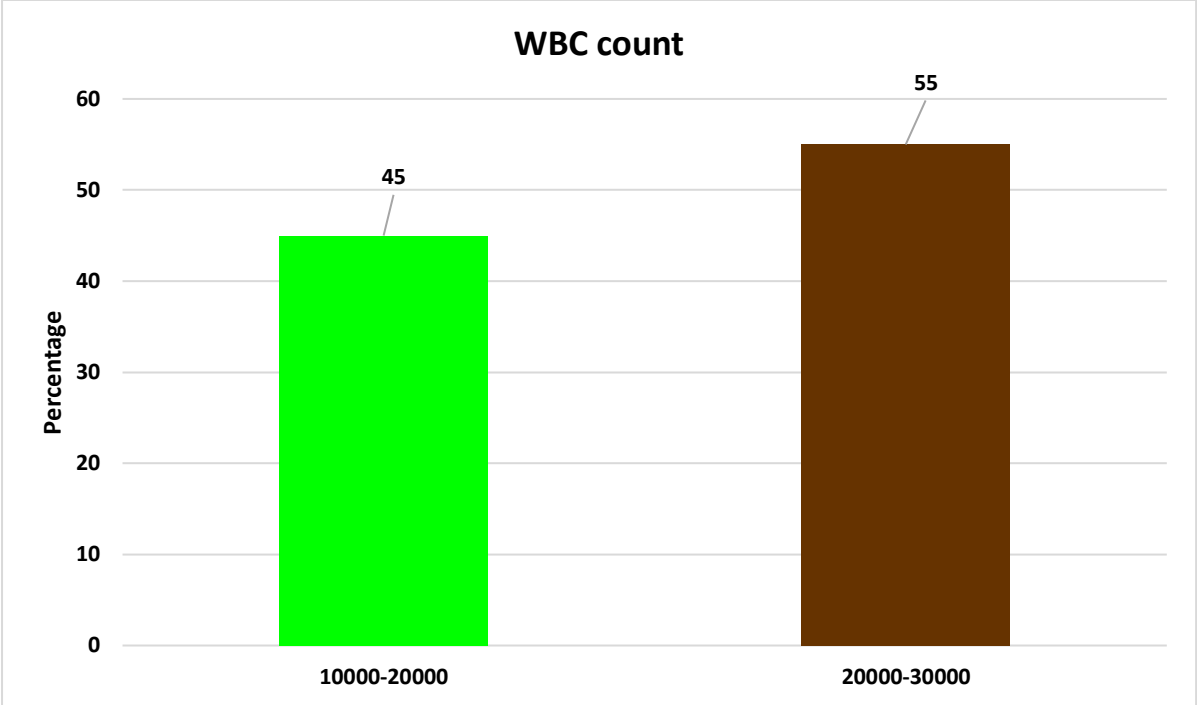
WBC count

45% of the study population had WBC values in the range 10000-20000 per microliter while 55% were having values in the range of 20000-30000 indicating very large infection

Table: WBC count of the study population

WBC count	Frequency (N)	Percentage (%)
10000-20000	36	45
20000-30000	44	55

Figure: WBC count of the study population



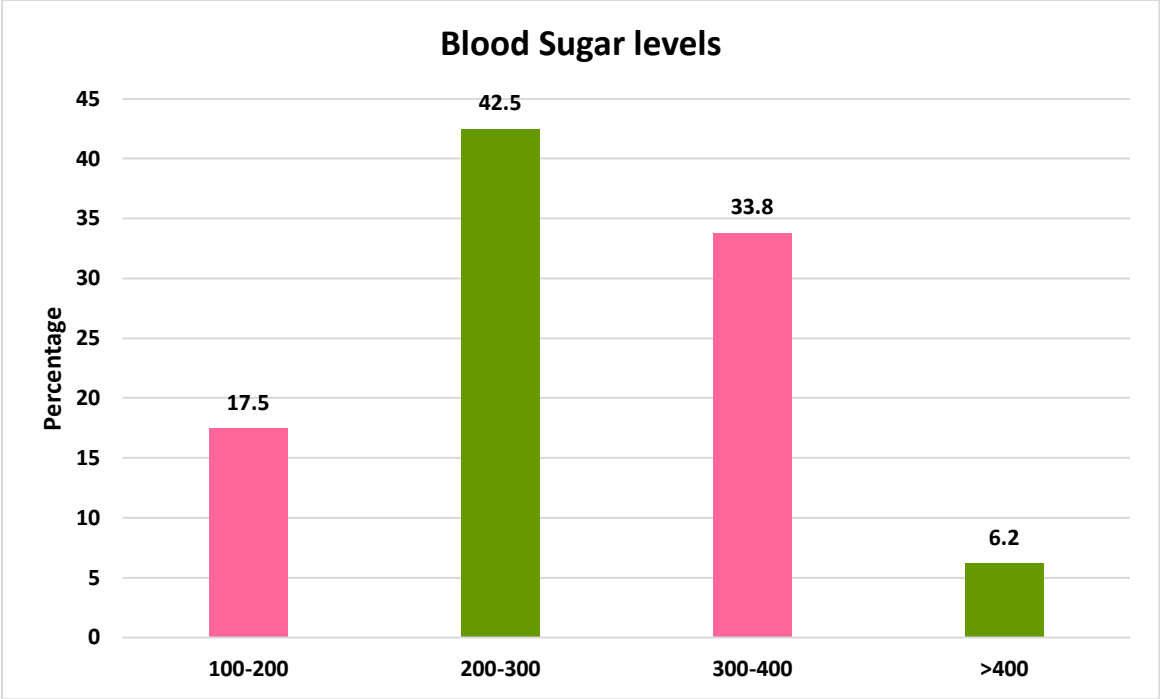
Blood Sugar levels

17.5% had blood sugar levels between **100-200 md/dl**, 42.5% had blood sugar levels between 200-300 mg/dl, 33.8% had blood sugars between 300-400 mg/dl and 6.2% had blood sugar level more than 400 mg/dl

Table: Blood Sugar levels of the study population

Blood Sugar levels (mg/dl)	Frequency (N)	Percentage (%)
100-200	14	17.5
200-300	34	42.5
300-400	27	33.8
>400	5	6.2

Figure: Blood Sugar levels of the study population



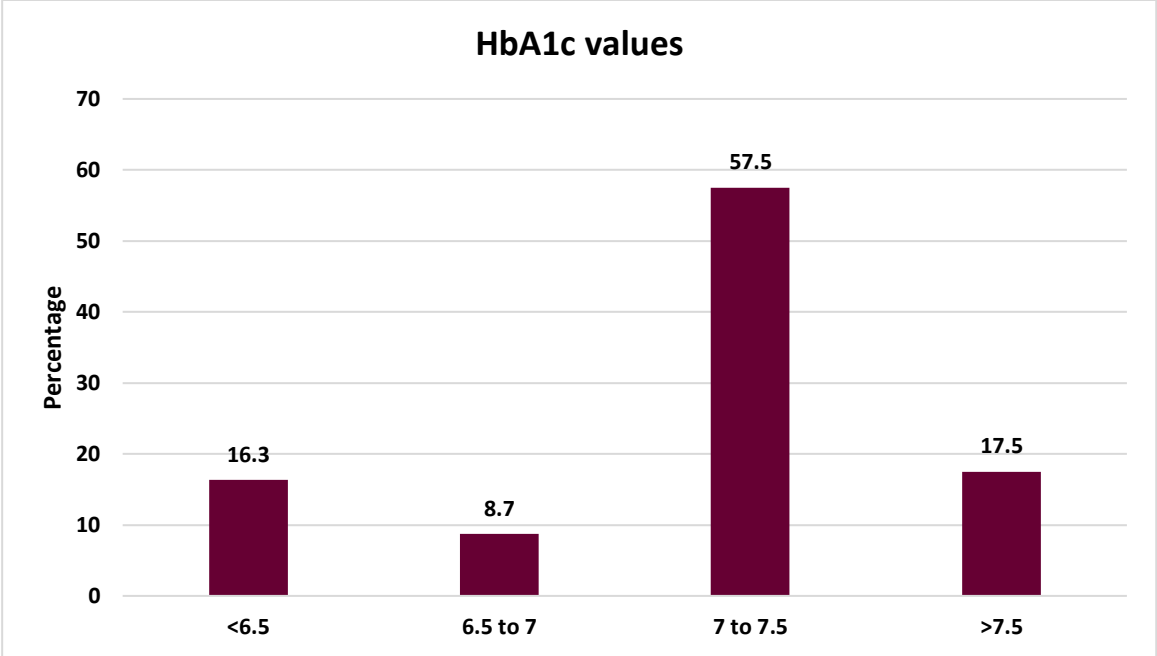
HbA1c values

Almost 57.5% had HbA1c in the 6.5 to 7 % range,17.5% had HbA1c in the greater than 7.5% range indicating that the uncontrolled diabetes could have lead to the development of pyelonephritis

Table: HbA1c values of the study population

HbA1c (%)	Frequency (N)	Percentage (%)
<6.5	13	16.3
6.5 to 7	7	8.7
7 to 7.5	46	57.5
>7.5	14	17.5

Figure: HbA1c values of the study population



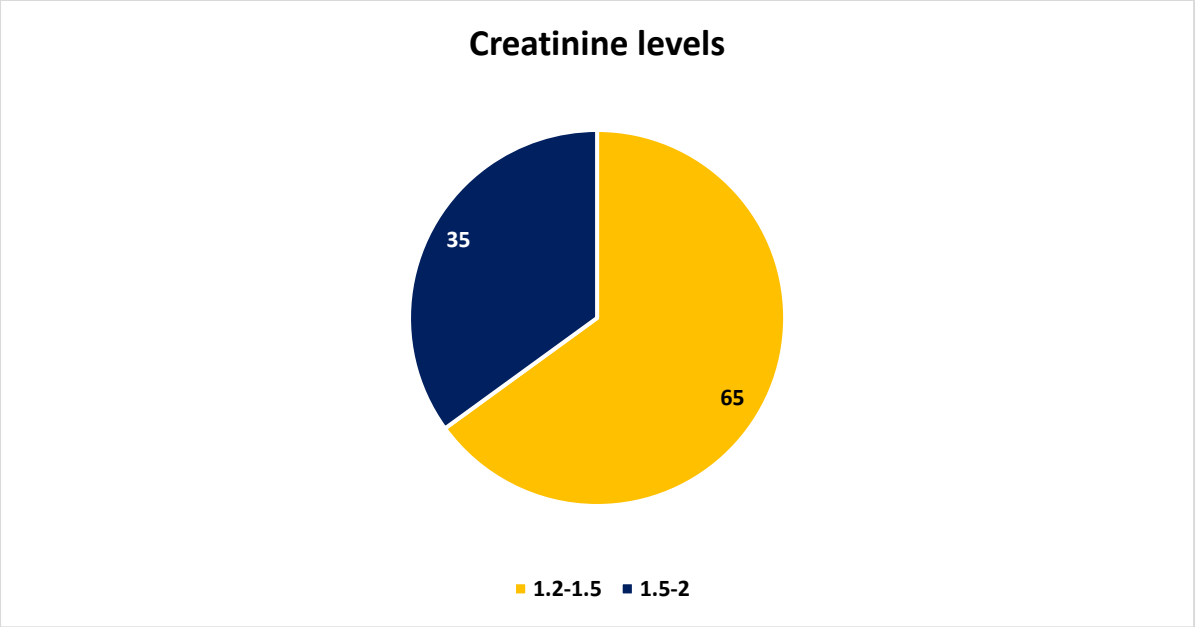
Creatinine levels

65% had creatinine levels in the range of 1.2-1.5 and 35% had creatinine values in the range 1.5-2

Table: Creatinine levels of the study population

Creatinine	Frequency (N)	Percentage (%)
1.2-1.5	52	65
1.5-2	28	35

Figure: Creatinine levels of the study population



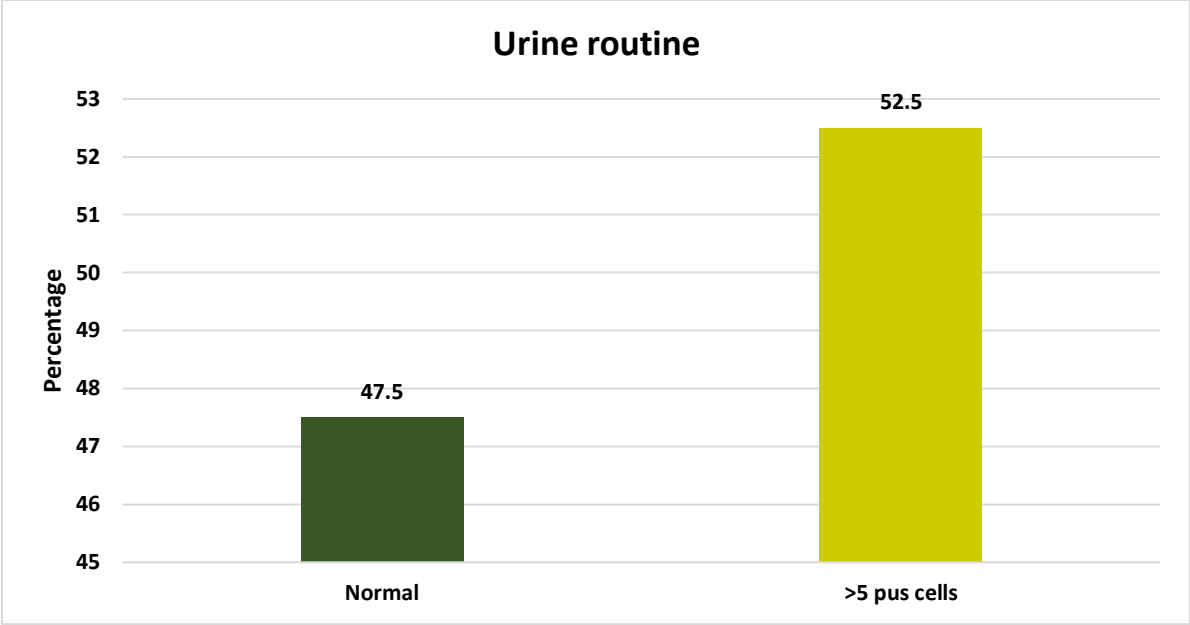
Urine Routine

52.5% of the study population had >5 pus cells in the routine urine examination while 47.5% had normal urine routine examination

Table: Urine routine interpretation of the study population

Urine Routine	Frequency (N)	Percentage (%)
Normal	38	47.5
>5 pus cells	42	52.5

Figure: Urine routine interpretation of the study population



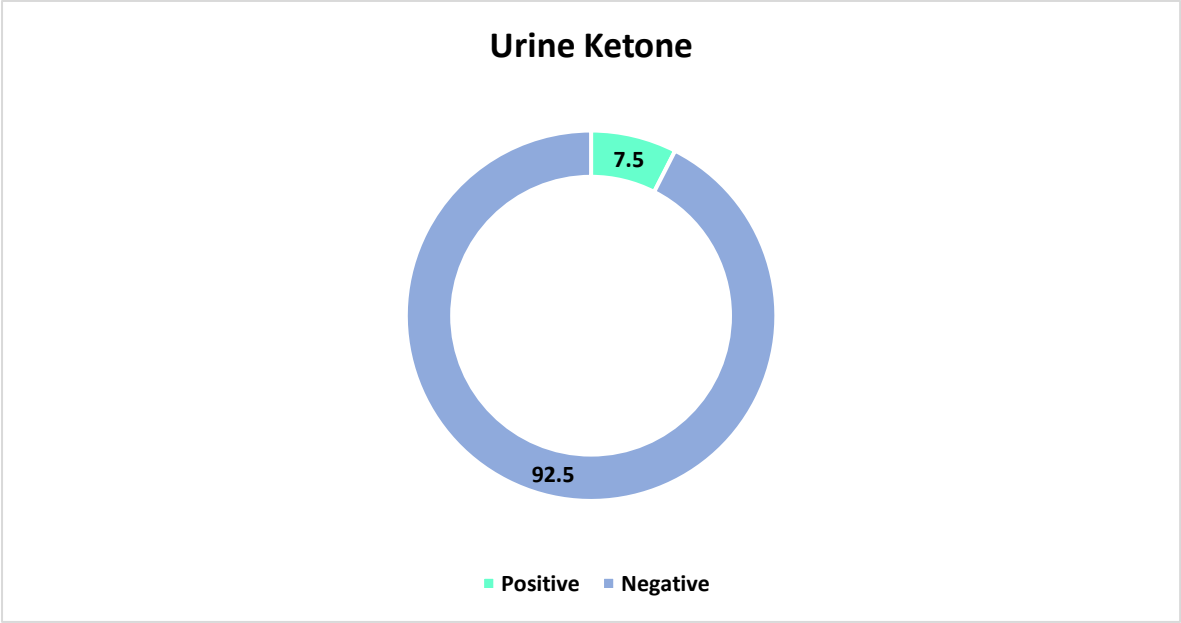
Urine ketones

7.5% of the study population had ketones in their urine sample

Table: Urine ketones levels of the study population

Urine Ketones	Frequency (N)	Percentage (%)
Positive	6	7.5
Negative	74	92.5

Figure: Urine ketones levels of the study population



MICROBIOLOGICAL PROFILE

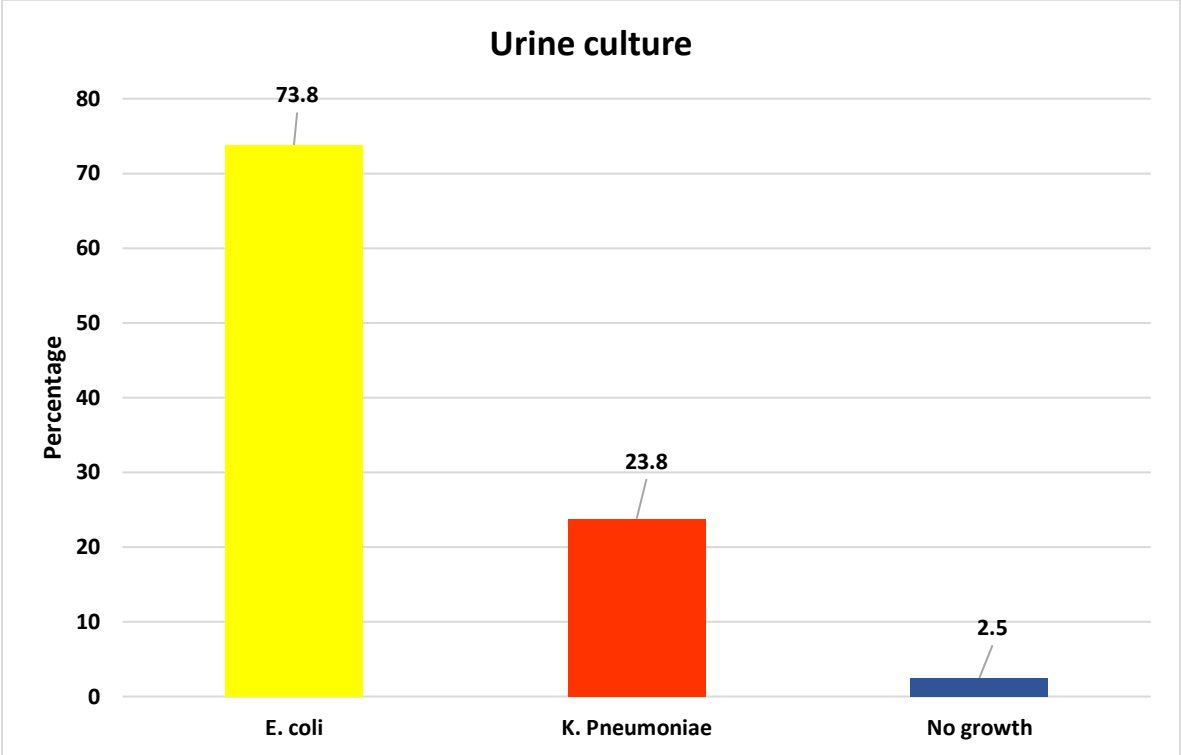
Urine Culture

73.8% of the study population had E.coli on urine culture, 23.8% had K. pneumoniae in culture and 2.5% had no growth

Table: Urine Culture of the study population

Urine Culture	Frequency (N)	Percentage (%)
E. coli	59	73.8
K. Pneumoniae	19	23.8
No growth	2	2.5

Figure: Urine Culture of the study population



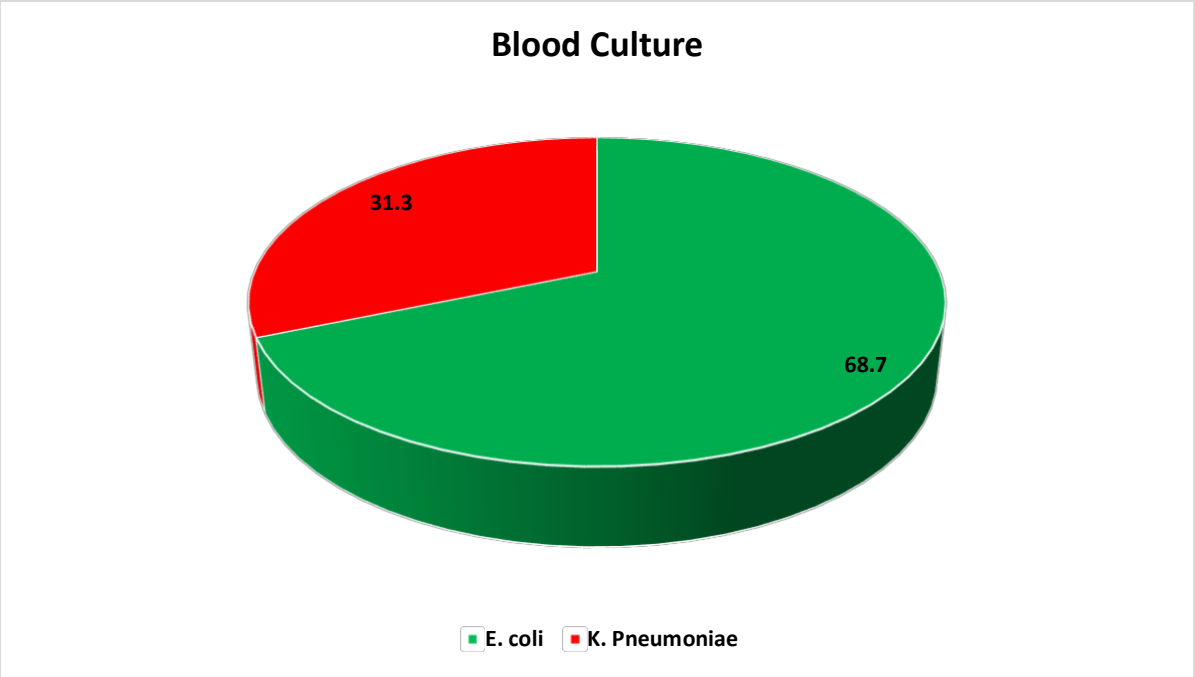
Blood Culture

The blood culture report of the study population also showed that 68.7% had E.coli and 31.3% had K. pneumoniae

Table: Blood culture of the study population

Blood Culture	Frequency (N)	Percentage (%)
E. coli	55	68.7
K. Pneumoniae	25	31.3

Figure: Blood culture of the study population



RADIOLOGICAL ABNORMALITIES

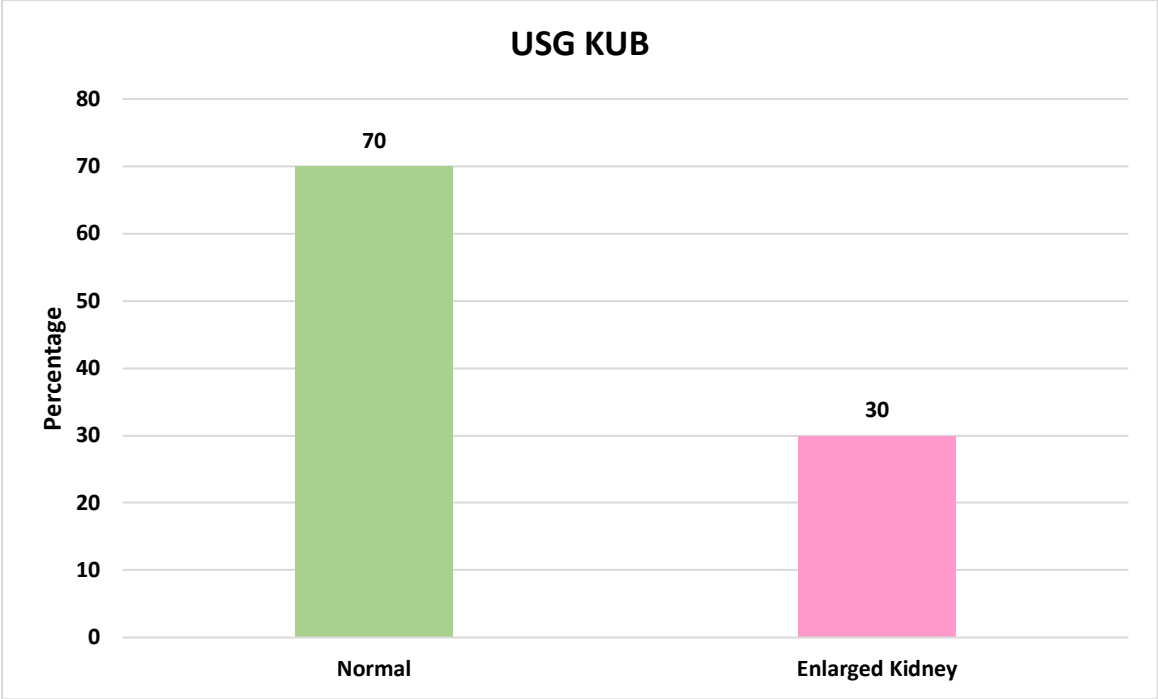
USG KUB

30% had enlarged kidney in USG KUB among the study population

Table: USG KUB findings of the study population

USG KUB	Frequency (N)	Percentage (%)
Normal	56	70
Enlarged Kidney	24	30

Figure: USG KUB findings of the study population



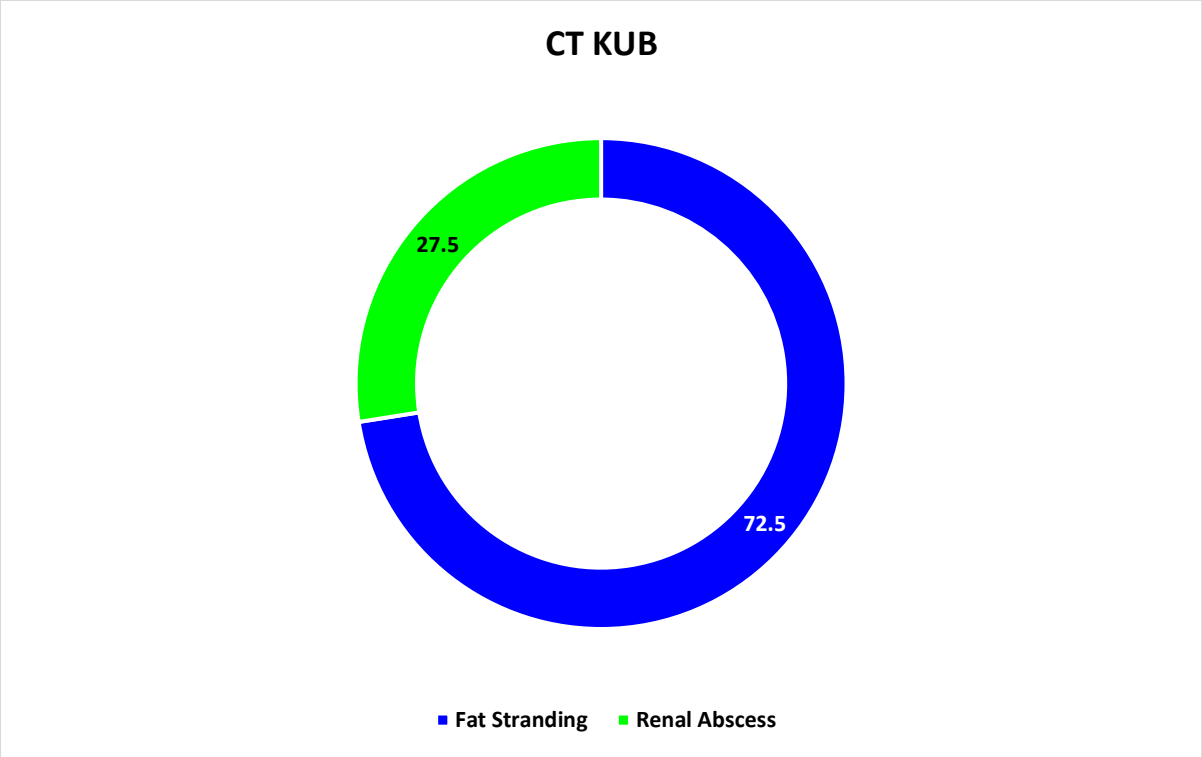
CT KUB

72.5% had fat stranding and 27.5% had renal abscess in CT KUB in our study

Table: CT KUB findings of the study population

CT KUB	Frequency (N)	Percentage (%)
Fat Stranding	58	72.5
Renal Abscess	22	27.5

Figure: CT KUB findings of the study population



SUMMARY

In this presented investigation of APN the sample populations mean age was 56.74±9.36 (SD), the lowest recorded age being 38years and highest being 74. There were 13 male patients admitted for APN complications and 67 women were included during the entire study period. The sample populations duration of diabetes was 12.65±7.42 with lowest duration of 4 years in a patient with highest recorded duration being 27 years Blood sugar level in the overall sample

population average was arrived as 339.19±75.54mg/dL, with least value noted as 220mg/dL and highest noted value at 480mg/dL. There was a strong correlation between the sample population's diabetic profile analysis with respect to age; duration of diabetics, blood sugar level and HbA1C. When the blood samples were profiled for serum creatinine (Sr C) the population's mean was found to be 5.22±2.44mg/dL. Altered sensorium was noted among 18 subjects, shock was reported in 11 subjects with 4 men and 5 women had altered sensorium; 4 men and 6 women presented shock during admission. 81 All the patients diagnosed with different grades of APN Among all the cases 67 were diabetic and their treatment consisted of insulin and different combination of antibiotics,

CONCLUSION □

Fever, loin pain and dysuria were the predominant symptoms prevailed in significant number of patients. □

E coli was the predominant causative organism isolated from the culture, but multi-organ dysfunction was more common in K pneumonia. □

Elevated serum creatinine levels, high WBC count, thrombocytopenia, poor glycaemic control; associated with poor outcome, and required timely aggressive management. □

BIBLIOGRAPHY

1. Ubee SS, McGlynn L, Fordham M. Emphysematous pyelonephritis. BJU international. 2011;107(9):1474-8.

2. Czaja CA, Scholes D, Hooton TM, Stamm WE. Population-based epidemiologic analysis of acute pyelonephritis. Clin Infect Dis. 2007;45(3):273-280

3. Ramakrishanan K, Scheid DC. Diagnosis and management of acute pyelonephritis in adults. Am Fam Physician 2005;71:933-42.

4. Nicolle LE. Epidemiology of urinary tract infection. Infect Med. 2001;18:153-162. Stamm WE, Hooton TM. Management of urinary tract infections in adults. N Engl J Med 1993;329:1328-34

5. Eswarappa M, Suryadevara S, John MM, Kumar M, Reddy SB, Suhail M. Emphysematous pyelonephritis case series from South India. Kidney international reports. 2018;3(4):950-5.

6. Scholes D, Hooton TM, Roberts PL, Gupta K, Stapleton AE, Stamm WE. Risk factors associated with acute pyelonephritis in healthy women. Ann Intern Med. 2005;142(1):20-27

. 7. Sokhal AK, Kumar M, Purkait B, Jhanwar A, Singh K, Bansal A, Sankhwar S. Emphysematous pyelonephritis: changing trend of clinical spectrum, pathogenesis, management and outcome. Turkish journal of urology. 2017 Jun;43(2):202.

8. Nicolle LE. Complicated pyelonephritis: unresolved issues. Curr Infect Dis Rep. 2007;9(6):501-507.

9. Somani BK, Nabi G, Thorpe P, Hussey J, Cook J, N'Dow J, ABACUS Research Group. Is percutaneous drainage the new gold standard in the management of emphysematous pyelonephritis? Evidence from a systematic review. The Journal of urology. 2008;179(5):1844-9.

10. Falagas ME, Alexiou VG, Giannopoulou KP, Siempos II. Risk factors for mortality in patients with emphysematous pyelonephritis: a metaanalysis. The Journal of urology. 2007;178(3):880-5.

11. Pontin AR, Barnes RD, Joffe J, Kahn D. Emphysematous pyelonephritis in diabetic patients. Br J Urol. 1995;75(1):71-4. https://doi.org/10.1111/j.1464-410x.1995.tb07237.x.

12. Efstathiou SP, Pefanis AV, Tsioulos DI, et al. Acute pyelonephritis in adults: prediction of mortality and failure of treatment. Arch Int Med 2003;163:1206-12.

13. Tilden W, Valliani S, Chana HS. Severe emphysematous pyelonephritis: conservative management plus image-guided percutaneous drainage prior to successful elective nephrectomy. A case report and review of the literature. BJR| case reports. 2015:20140004.

14. Alonso JV, Turpie J, Farhad I, Ruffino G. Protocols for point-of-careultrasound (POCUS) in a patient with sepsis; an algorithmic approach. Bulletin of Emergency & Trauma. 2019;7(1):67.

15.Buttar S, Cooper Jr D, Olivieri P, Barca M, Drake AB, Ku M, Rose G, Siadecki SD, Saul T. Air and its sonographic appearance: understanding the artifacts. The Journal of Emergency Medicine. 2017;53(2):241-7.

15. Rollino C, Beltrame G, Ferro M, Quattrocchio G, Sandrone M, Quarello F. Acute pyelonephritis in adults: a case series of 223 patients. Nephrol Dial Transplant 2012;27:3488-93

16. Chen KC, Hung SW, Seow VK, Chong CF, Wang TL, Li YC, Chang H. The role of emergency ultrasound for evaluating acute pyelonephritis in the ED. The American journal of emergency medicine. 2011;29(7):721- 4.

17. de Jager CP, van Wijk PT, Mathoera RB, de Jongh-Leuvenink J, van der Poll T, Wever PC. Lymphocytopenia and neutrophil-lymphocyte count ratio predict bacteremia better than conventional infection markers in an emergency care unit. Crit Care 2010;14(5):R192. DOI: https://doi.org/10.1186/cc9309.

18. Hindi A, Peterson C, Barr RG. Artifacts in diagnostic ultrasound. Reports in Medical Imaging. 2013;6:29-48.

19. Hudson MA, Weyman PJ, van derVliet AH, Catalona WJ. Emphysematous pyelonephritis: Successful management by percutaneous drainage. Journal of Urology 1986;136(4):884-886. https://doi.org/10.1016/s0022-5347(17)45115-9.

20. Huang JJ, Tseng CC. Emphysematous pyelonephritis: Clinicoradiological classification, management, prognosis, and

pathogenesis. Archives of Internal Medicine 2000;160(6), 797- 805. https://doi.org/10.1001/archinte.160.6.797.

21. Brown N, Petersen P, Kinas D, Newberry M, Emphysematous Pyelonephritis Presenting as Pneumaturia and the Use of Point-of-Care Ultrasound in the Emergency Department, Case Reports in Emergency Medicine, 2019(2019), Article ID 6903193. https://doi.org/10.1155/2019/6903193

22. Wan YL, Lee TY, Bullard MJ, Tsai CC. Acute gas-producing bacterial renal infection: correlation between imaging findings and clinical outcome. Radiology. 1996;198(2):433-8.

23. McCollough CH, Schueler BA, Atwell TD, et al. Radiation exposure and pregnancy: When should we be concerned? Radiographics. 2007;27:909- 917; discussion 917-908.

24. Alsharif M, Mohammedkhalil A, Alsaywid B, Alhazmy A, Lamy S. Emphysematous pyelonephritis: Is nephrectomy warranted? Urol Ann 2015;7:494-8. doi:10.4103/0974-7796.158503

25. Kolla PK, Madhav D, Reddy S, Pentyala S, Kumar P, Pathapati RM. Clinical profile and outcome of conservatively managed emphysematous pyelonephritis. ISRN Urol 2012;2012:931982. DOI: https://doi.org/10.5402/2012/931982.

26. Patel SJ, Reede DL, Katz DS, et al. Imaging the pregnant patient for nonobstetric conditions: algorithms and radiation dose considerations. Radiographics. 2007;27:1705-1722.

27. Foxman B, Klemstine KL, Brown PD. Acute pyelonephritis in US hospitals in 1997: hospitalization and in hospital mortality. Ann Epidemiol. 2003;13(2):144-150.

28. Levey A. S., Bosch J. P., Lewis J. B. , Greene T., Rogers N., Roth D., A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. modification of diet in renal disease study group, Annals of Internal Medicine, 1999;130(6):461–470

29. Yamamoto Y, Fujita K, Nakazawa S, et al. Clinical characteristics and risk factors for septic shock in patients receiving emergency drainage for acute pyelonephritis with upper urinary tract calculi. BMC Urology 2012;12:4.

30. Zagoria RJ, Dyer RB, Harrison LH, Adams PL. Percutaneous management of localized emphysematous pyelonephritis. J Vasc Interv Radiol 1991;2(1):156-158. https://doi.org/10.1016/s1051-0443(91)72491-3

31. Lu Y-C, C B-J, Pong Y-H, Chen C-H, Pu Y-S, Hsuch P-R, Huang C-Y. Emphysematous pyelonephritis: Clinical characteristics and prognostic

factors. International Journal of Urology 2014;21:277–282. doi: 10.1111/iju.12244

32. Kapoor R, Muruganandham R, Gulia AK, Singla M, Agrawal S, Mandhani A, Ansari M, Srivastava A. Predictive factors for mortality and need for nephrectomy in patients with emphysematous pyelonephritis. BJU International, 2009;105: 986-989. doi:10.1111/j.1464-410X.2009.08930.x

33. Kiranmala K, Johnson R, Savio J, Idiculla J. Microbiologic profile and clinical practices in urinary tract infections in a tertiary care center in Southern India. J Family Med Prim Care 2019;8:2888-92. doi:10.4103/jfmpc.jfmpc_346_19.

34. Punatar C, Jadhav K, Kumar V, Joshi V, Sagade S. Neutrophil:Lymphocyte Ratio as a Predictive Factor for Success of Nephron-Sparing Procedures in Patients with Emphysematous Pyelonephritis. Permanente Journal 2019;23:18-044, https://doi.org/10.7812/TPP/18-044

35. Chung VY, Tai CK, Fan CW, Tang CN. Severe acute pyelonephritis: a review of clinical outcome and risk factors for mortality. Hong Kong Medical Journal 2014;20:285–289. doi: 10.12809/hkmj134061

36. Colgan R, Williams M. Diagnosis and Treatment of Acute Pyelonephritis in Women. American Family Physician 2011;84(5): 519-526.

37. Khade AL, Lad SK, Shah VB. Pathology of emphysematous pyelonephritis: A study of 11 cases. Med J DY Patil Univ 2016;9:722-6.doi:10.4103/0975-2870.194191

38. Das D, Pal DK. Double J stenting: rewarding option in the management of emphysematous pyelonephritis. Urol Ann 2016;8:261-4. doi:10.4103/0974-7796.184881

CONSENT FORM

STUDY TITLE:

"A STUDY ON CLINICAL PROFILE, MICROBIOLOGICAL

PROFILE ,RADIOLOGICAL ABNORMALITIES IN ACUTE PYELONEPHRITIS "

DEPARTMENT OF GENERAL MEDICINE GSMC-CHENNAI

PARTICIPANT NAME:

AGE

: SEX

: I.P. NO:

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during and after medical procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study.

Time: Patient name;

Date: Signature / Thumb Impression of Patient:

Place

Name and signature of the Investigator

ஆராvசி ஒ~©தபவ• ெபயt: ேததி : வய¢: உ ேநாயாள• எz:

பாலின• : ஆ€ º ேசtைக எz: இ|த ஆ€ வŽ } ேநா‚க• ம, ²•

வŽ வரuக,, «,ைமயாக எனt ெதள• வாக வŽ ள‚க~பyட¢ .

இ† வா€ வŽ ƒ இ¯|¢ நா} எ|த ேநர«• பŽ }வாuகலா• எ}பைத•

அதனாƒ எனt எ|த பாதி~©• இ ைல எ}பைத• ெதள• வாக ©T|¢

ெகாzேட}. «œºகைள அƒ ல¢ க¯{¢கைள ெவள• யŽ ž • ேபாேதா

அƒ ல¢ ஆ€ வŽ } ேபாேதா எ}§ைடய ெபயைரேயா அƒ ல¢

அைடயாளuகைளேயா ெவள• யŽ ட மyடா‚க,, எ}பைத• அறி|¢

ெகாzேட}. இ|த ஆ€ வŽ ƒ எ† வŽ த நி‚ப|த«• இ}றி என¢ ெசா|த

வŽ ¯~ப{தி} ேபTƒ நா} பu ெப²கி}ேற} . நா} யநிைனºட§• «,

த|திர{¢ட§• இ|த ம¯{¢வ ஆரா€ vசியŽ ƒ ேசt{¢tெகா,, ள

ச• மதtகி}ேற}. ஆரா€ சியாள‚ ஒ~ப• பuேக, பாள‚ ஒ~ப•)அ(இட¢

ெப¯வŽ ர ேரைக

ANNEXURE II – PROFORMA

Case No: Name:

Age / Sex: In Patient Number:

Address: Occupation:

Complaints at presentation

Fever

Vomiting

Abdominal Pain

, Flank Pain

Urinary Symptoms

Other Symptoms

PAST HISTORY

History of Diabetes Mellitus

- Duration -

Mode of Therapy -

Control Status

History of Comorbid Illness -

Hypertension -

Coronary Artery Disease -

Previous Hospitalisation

History of Urinary tract obstruction

History of Renal Stones

History of Instrumentation, trauma

History of Immunosuppressive illness, drugs

FAMILY HISTORY

1. Diabetes Mellitus

2. Other Illness

PERSONAL HISTORY

Whether Smoker, Alcoholic

ClINICAL EXAMINATION

Height Weight

VITALS

Pulse Rate Respiratory Rate

Blood Pressure Temperature

SYSTEMIC EXAMINATION

Abdomen

INVESTIGATION

1. CBC

2. Blood sugar

3. RFT- Renal Function Test

4. LFT- Liver function Test

5. Acetone

6. Hb1Ac

7. Urine Culture and sensitivity

8. Blood Culture and Sensitivity

9. USG KUB

10. CT KUB

MASTER CHART

NO	AGE	GE	DM	FEVER	LOIN	ALTE	DYURI	SHOC	BLO	URIN	URI	URI	CR	BLOOD	USG KUB	CT KU	URINAR

		GENDER			PAIN	REDSENSORIUM	A	K	ODSUGARS	EROUTINE	NEKETONES	NECULTURE	EATINNE	CULTURE		B	YOBSTRUCTION
1	65	F	1	1	1	2	2	2	2	1	2	1	1	1	1	1	2
2	48	F	1	1	1	2	1	2	2	2	2	1	1	1	1	1	2
3	68	F	2	2	2	1	2	1		2	2	1	2	2	1	1	2
4	59	M	1	1	1	2	1	2	2	2	2	1	1	1	1	1	2
5	86	F	1	2	2	1	2	1	2	1	2	1	2	2	1	1	2
6	73	F	1	1	1	2	2	2	3	1	2	3	2	2	1	1	2
7	89	F	1	2	2	2	2	2	2	1	2	1	2	1	1	1	2
8	62	M	2	2	2	1	2	2	1	2	2	1	2	2	1	2	2
9	62	F	1	1	1	2	1	2	3	2	2	1	1	1	2	1	2
10	48	F	1	1	2	2	2	2	4	2	1	1	1	1	1	1	2
11	54	M	1	1	1	2	1	2	3	2	2	1	1	1	1	2	2
12	52	F	1	1	1	1	2	2	3	1	2	1	1	1	1	1	2
13	62	F	2	2	2	1	2	2	1	2	2	3	1	2	1	1	2
14	49	F	1	1	1	2	1	2	2	2	2	1	1	1	1	1	1
15	53	F	2	1	1	2	1	2	1	2	2	1	1	1	1	1	2
16	50	F	1	1	1	2	1	2	3	2	2	2	1	1	1	1	2
17	62	M	2	1	2	1	2	1	1	1	2	1	1	1	1	1	2
18	49	F	1	1	1	2	1	2	3	1	2	1	1	2	2	1	2
19	38	F	1	1	2	1	2	2	3	2	1	1	1	2	1	1	2
20	54	F	2	2	2	1	2	1	1	2	2	1	2	2	1	1	2
21	49	F	1	1	1	2	1	2	3	2	2	1	2	1	1	1	1
22	58	F	1	1	1	2	2	2	3	1	2	1	2	1	2	1	2
23	72	F	1	1	2	2	2	2	3	2	2	1	1	1	2	1	2
24	52	M	2	1	1	2	1	2	1	2	2	1	1	1	1	1	2
25	54	F	1	1	2	2	2	2		1	2	1	1	2	2	2	1
26	72	F	1	2	2	2	2	2	2	1	2	1	2	1	1	1	2
27	47	F	2	1	1	2	1	2	1	1	2	2	1	1	2	2	1
28	62	F	1	1	2	2	1	2	2	1	2	1	1	1	1	1	2
29	51	F	1	1	1	2	1	2	2	2	2	1	2	1	1	1	2
30	61	F	1	1	1	2	2	2	2	1	2	1	1	1	1	1	2
31	71	F	1	1	1	2	2	2	2	1	2	1	2	2	1	2	2
32	45	F	2	2	2	0	2	2	1	1	2	1	1	1	1	1	2
33	59	M	2	1	2	1	2	1	1	1	2	1	1	1	1	1	2
34	54	F	1	1	2	2	2	2	2	1	2	1	1	2	2	2	1
35	48	F	1	1	1	2	2	2	3	1	2	1	2	2	1	1	2
36	68	M	1	1	1	2	1	2	2	2	2	1	1	2	1	2	2
37	57	F	1	1	1	2	2	2	3	1	2	1	1	1	1	1	2
38	61	F	1	2	2	2	2	2	2	1	2	2	1	1	1	1	1

39	51	F	1	1	1	2	2	2	2	1	2	1	2	1	2	1	2
40	61	F	1	1	2	1	2	2	3	2	2	2	2	1	1	2	1
41	44	F	1	1	1	2	1	2	3	2	2	1	1	1	1	2	2
42	52	F	2	1	1	2	1	2	1	2	2	1	1	1	1	2	2
43	53	F	1	2	1	1	2	1	2	2	2	1	2	2	2	2	1
44	39	F	1	1	1	2	1	2	4	2	1	2	1	1	1	1	2
45	81	F	1	1	2	1	2	1	2	1	2	2	1	2	2	2	1
46	71	F	1	1	1	1	2	1	3	2	2	2	1	1	1	1	2
47	69	M	1	1	1	2	1	2	2	1	2	1	1	2	2	1	2
48	61	F	2	1	1	2	1	2	1	1	2	1	1	1	1	1	2
49	58	F	1	1	2	2	2	2	2	1	2	1	1	2	2	2	2
50	48	F	1	2	2	2	2	2	2	1	2	1	2	1	1	1	2
51	44	F	1	1	2	2	2	2	3	1	1	1	1	2	2	2	2
52	51	M	1	1	1	2	2	2	2	2	2	2	2	1	2	2	2
53	39	F	1	1	1	2	1	2	4	2	2	2	1	1	1	1	2
54	77	F	1	1	2	2	2	2	2	2	2	2	1	1	1	1	2
55	60	M	1	1	2	2	2	2	2	2	2	1	1	1	1	1	2
56	49	F	1	1	1	2	1	2	4	1	2	1	1	2	2	1	2
57	50	F	1	1	1	2	1	2	3	2	2	1	2	2	1	1	2
58	53	F	2	1	1	2	1	2	1	2	2	1	1	1	1	1	2
59	61	F	1	1	1	2	2	2	2	1	2	1	2	2	1	1	2
60	58	F	1	1	1	2	1	2	3	2	2	1	2	1	2	1	2
61	46	F	1	1	1	2	2	2	2	1	2	1	2	1	2	1	2
62	47	M	1	1	2	2	1	2	3	2	2	1	1	1	1	1	2
63	49	F	1	1	1	2	2	2	3	1	2	2	1	2	1	1	2
64	50	F	1	1	1	2	2	2	4	2	2	2	2	1	2	2	2
65	54	F	1	1	2	2	2	2	3	1	2	1	1	2	2	2	2
66	73	F	1	1	1	1	2	1	3	2	2	2	1	1	1	1	2
67	46	F	1	1	2	2	2	2	3	1	2	1	1	1	1	1	2
68	52	F	1	1	1	2	2	2	3	1	2	1	1	1	1	1	2
69	48	F	1	1	1	2	1	2	2	2	2	1	2	1	2	1	2
70	55	F	1	1	1	2	1	2	2	1	2	1	2		1	2	2
71	61	F	1	1	1	1	2	1	3	2	2	2	1	1	1	1	2
72	61	M	1	1	1	2	1	2	2	1	2	1	2	1	2	2	2
73	47	F	1	1	1	2	1	2	2	2	2	2	1	1	1	1	2
74	55	F	1	1	2	1	2	2	3	2	1	2	2	1	1	2	1
75	45	F	1	1	1	2	1	2	2	2	2	1	2	1	2	1	2
76	33	F	1	1	2	1	2	1	3	1	1	2	1	1	1	1	2
77	46	F	1	1	2	2	2	2	2	1	2	1	1	1	1	1	2
78	61	F	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2
79	47	F	1	2	1	2	2	2	2	2	2	2	1	2	1	1	2
80	50	M	1	1	2	2	1	2	2	2	2	2	1	1	2	2	1