# STUDY OF CLINICAL PROFILE OF ACUTE KIDNEY INJURY IN INTENSIVE CARE UNIT

**BY:**

**DR.SMIT S. SHAH (M.B.B.S)**

**DISSERTATION SUBMITTED TO**

**S.B.K.SMEDICAL INSTITUTE & RESEARCH CENTRE**

SUMANDEEP VIDYAPEETH, PIPARIA, VADODARA

**IN PART FULFILLMENT**

**OF THE REQUIREMENTS FOR THE DEGREE OF**

**M.D. IN GENERAL MEDICINE**

**Under the Guidance of**

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**YEAR 2015-2018**

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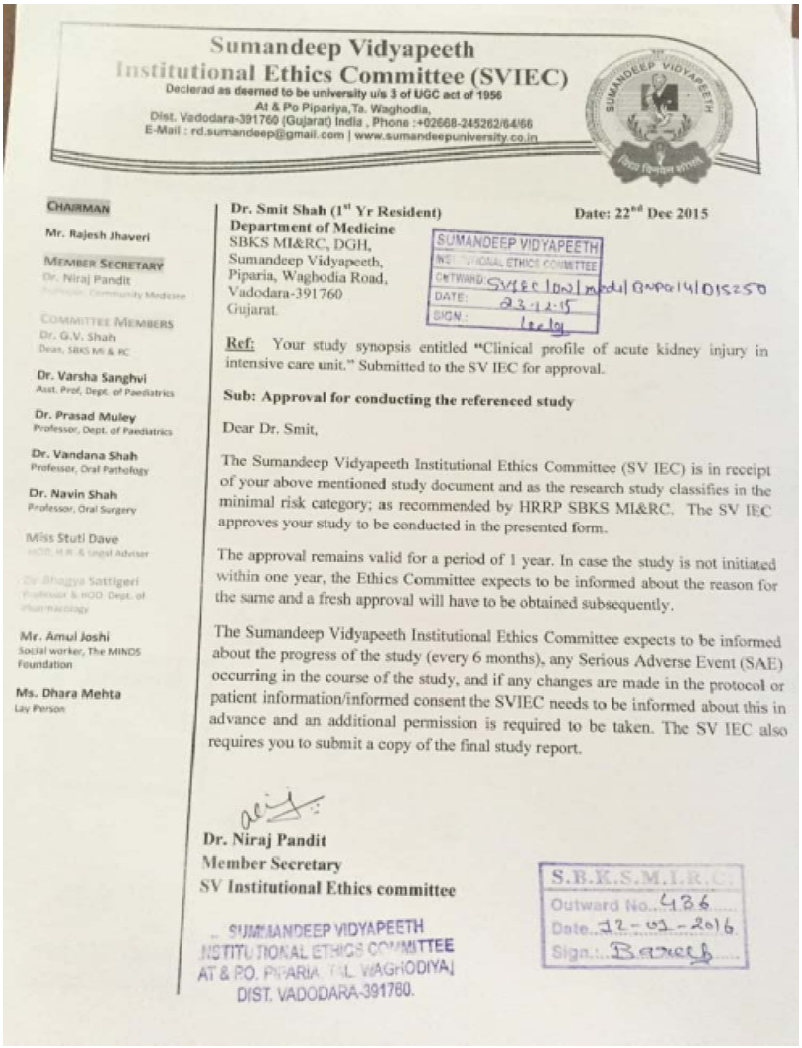

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**Ref:** Your study synopsis entitled "Clinical profile of acute kidney injury in intensive care unit." Submitted to the SV IEC for approval.

**Sub: Approval for conducting the referenced study**

Dear Dr. Smit,

The Sumandeep Vidyapeeth Institutional Ethics Committee (SV IEC) is in receipt of your above mentioned study document and as the research study classifies in the minimal risk category; as recommended by HRRP SBKS MI&RC. The SV IEC approves your study to be conducted in the presented form.

The approval remains valid for a period of 1 year. In case the study is not initiated within one year, the Ethics Committee expects to be informed about the reason for the same and a fresh approval will have to be obtained subsequently.

The Sumandeep Vidyapeeth Institutional Ethics Committee expects to be informed about the progress of the study (every 6 months), any Serious Adverse Event (SAE) occurring in the course of the study, and if any changes are made in the protocol or patient information/informed consent the SVIEC needs to be informed about this in advance and an additional permission is required to be taken. The SV IEC also requires you to submit a copy of the final study report.

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**Member Secretary**
**SV Institutional Ethics committee**

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## STUDY COMPLETION CERTIFICATE

This is to certify that your study synopsis entitled: "Clinical Profile of Acute Kidney Injury in Intensive Care Unit." Research Project was done by **"Dr. Smit Shah"** (PG Student, Dept of Medicine, S.B.K.S MI & RC, Dhiraj Hospital, Piparia, Waghodia road, Vadodara-391760, Gujarat) and it was conducted to the satisfaction of the Sumandeep Vidyapeeth Institutional Ethics committee.

**Dr. Niraj Pandit**
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927
21/09/2017

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## DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled "**STUDY OF CLINICAL PROFILE OF ACUTE KIDNEY INJURY IN INTENSIVE CARE UNIT"**is a bonafide and genuine research work carried out by me under the guidance of **DR. PRAMOD R. JHA , Professor, Department of MEDICINE, SBKS Medical Institute & Research Centre, Piparia, Vadodara.**

**Date:**
**Place: PIPARIA**

**Signature of the Candidate**
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## SUMANDEEP VIDYAPEETH

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# ACKNOWLEDGEMENT

*Words may fail to express my deep sense of gratitude that I felt towards my respected professor, excellent teacher and guide* ***DR.PRAMOD JHA,*** *PROFESSOR, DEPARTMENT OF GENERAL MEDICINE, SMT. BK SHAH MEDICAL INSTITUTE & RESEARCH CENTRE, VADODARA for this dissertation, without whom this dissertation would not be possible.*

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**Dr SMIT S. SHAH**
**M.B.B.S**

# INDEX

# INTRODUCTION

The first detailed report of fatal acute kidney injury has been accredited to Hackradt, a pathologist from Germany in 1917 and was based on soldiers who sustained severe traumatic injuries. Since then, a number of publications have discussed the pathogenesis of Acute Kidney Injury. These reports have focused on isolating the etiopathogenesis, risk factors, mortality rate and treatment. Compared to western literature, reports from our country are scarce and thus, a clinical profile study of Acute Kidney Injury is necessary. In view of this, a pilot study of patients developing Acute Kidney Injury was conducted.

# AIM& OBJECTIVES

1. Study incidence of Acute Kidney Injury at our centre

2. Study various causes of Acute Kidney Injury

3. Assess clinical spectrum of Acute Kidney Injury

4. Analyse course of Acute Kidney Injury in medicalcases

5. Study complications of Acute Kidney Injury

# REVIEW OF LITERATURE

Acute Kidney Injury (AKI) is a syndrome of rapid decline in glomerular filtration rate (hours to days), nitrogenous waste products retention,and perturbation of extracellular fluid volume and electrolytes and acid base balance[1].

On the basis of cause, Acute Kidney Injury may be divided into prerenal, intrinsic or post renal. Majority of the patients develop prerenal,acute kidney injury or acute tubular necrosis (a variety of intrinsic acute kidney injury which is usually caused by ischemia or toxins). With a systematic approach, physicians can elicit the underlying cause of acute kidney injury in most of the patients. This approach consists of a thorough history and physical examination, blood investigations, urine examination and a renal ultrasonographic evaluation.

In certain situations, such as when a patient has glomerular disease, micro vascular disease or obstructive disease, early diagnosis and management are necessary to prevent renal damage. By maintaining euvolemia, identifying patients who are,at increased risk of developing AKIand minimizing exposure to nephrotoxic agents, physicians can decrease the incidence of acute kidney injury.Once acute kidney injury develops, supportive therapy is critical in maintaining the balance of fluid and electrolytes, minimization,of nitrogenous waste production and sustaining nutrition. Death is most often caused by infection or cardio-respiratory complications (Am Fam Physician 2000; 61:2077-88).

Infection accounts for approximately 75 % deaths in patients with Acute Kidney Injury. The second most common cause is cardiorespiratory complications[14-16]. On the

basis of the severity of renal failure, the mortality rate can range from as low as 7 percent to as high as 80 percent [15-17].

In Acute Kidney Injury, there is decline in the glomerular filtration rate over days to weeks. Because of this, the excretion of nitrogenous waste is reduced, and the balance of fluid and electrolytes cannot be maintained. Patients with Acute Kidney Injury,are often found to be asymptomatic, and the underlying disease can only be diagnosed by observed elevations of Blood Urea Nitrogen (BUN) and Serum Creatinine Levels.

Most authorities define Acute Kidney Injury as an acute increase in serum creatinine levels from baseline (i.e. an increase of at least 0.5 mg per dl[15]. Complete renal shutdown is considered when the serum creatinine levels rise by at least 0.5 mg per dl per day and there should also be urine output less than 400 ml per day (oliguria).

Elevations in blood urea nitrogen and serum creatinine levels donot always arise from Acute Kidney Injury. Drugs such as cephalosporins& trimethoprim-sulfamethaxazole may cause AKI as a secondary to that of interstitial disease, but these agents often result in elevated serum creatinine levels simply by inhibition of tubular secretion of creatinine without causing any real damage to kidneys. In patients who are on corticosteroid therapy, those with increased catabolism and those with gastrointestinal bleeding, the blood urea nitrogen may be elevated.

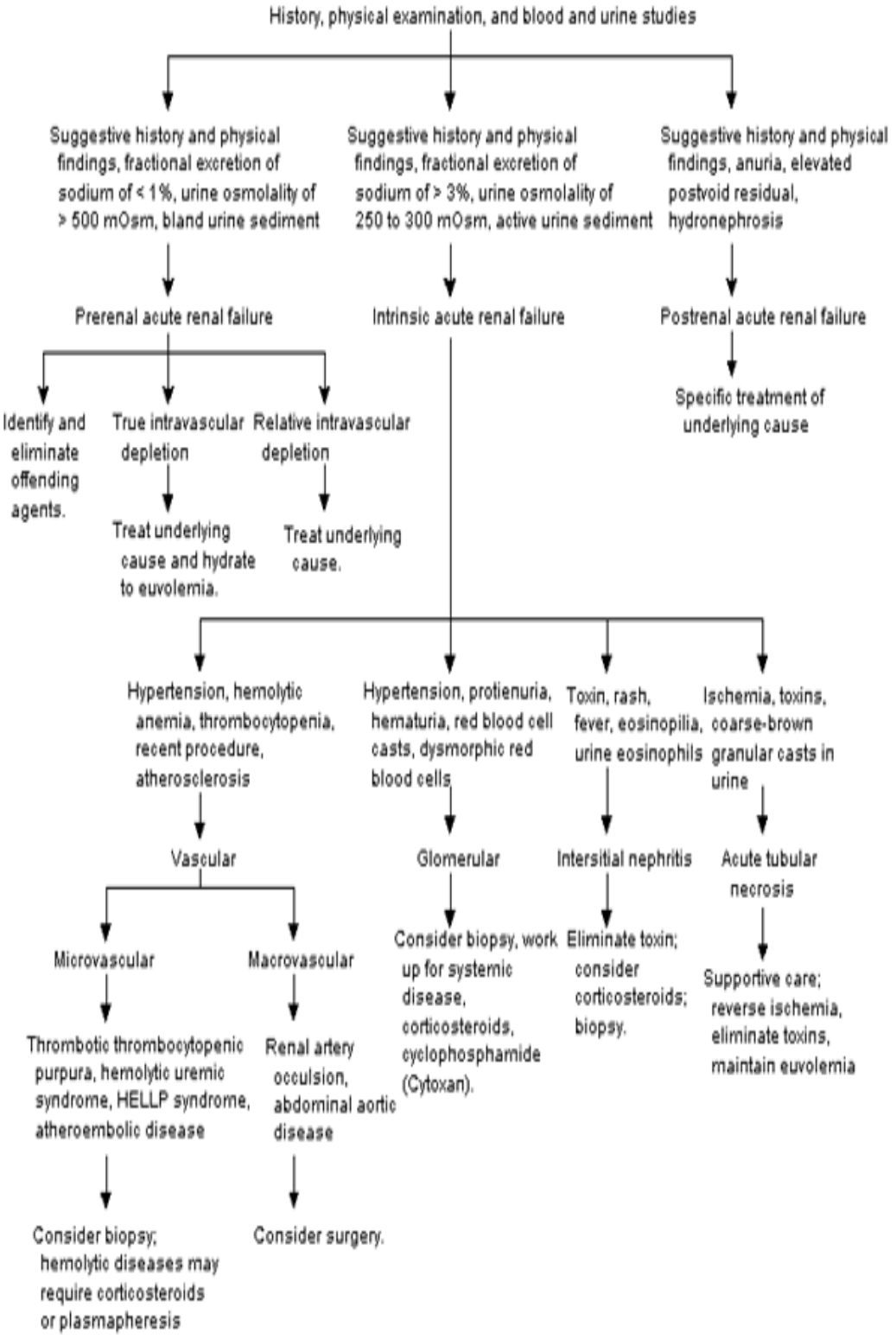



**Figure 1: Algorithm for the Diagnosis & Treatment of Acute Kidney Injury**

## POINTS OF DIFFERENTIATION & DIAGNOSIS

Acute kidney injury can be classified into three categories. (pre renal, intrinsic and post renal). Pre renal acute kidney injury is characterised by diminished renal blood flow (most common, occurs in 60 to 70 percent of cases). In intrinsic AKI, there is damage to the renal parenchyma (occurs in 25 to 40 percent of cases). Post renal,AKI occurs due to some obstructive cause such as urinary tract obstruction (least common, occurs in about 5 to 10 percent of cases)[14]. Various etiologies of AKI can be classified as:

Pre renal: low blood volume, low blood pressure, heart failure, liver cirrhosis, local changes to the blood vessels supplying the kidney, renal artery stenosis, inadequate cardiac output and hypovolemia.

Intrinsic:Glomerulonephritis, acute tubular necrosis (ATN), and acute interstitial nephritis (AIN)

Post renal: Benign prostatic hyperplasia, renal stones, obstructed urinary catheter, bladder stone; bladder, ureteral or renal malignancy.

Acute kidney injury can be classified as following:

Introduced by KDIGO,in 2012, specific criteria exist for diagnosis of AKI. Acute kidney injury can be diagnosed if any one of these is present:

- Increase in serum creatinine by ≥0.3 mg/dl (≥26.5 μmol/l) within 48 hours; or
- Increase in serum creatinine to ≥1.5 times baseline, which have occurred within the prior 7 days; or
- Urine volume < 0.5 ml/kg/h for 6 hours.

**Staging:**

The *RIFLE,criteria*, proposed by the Acute Dialysis Quality Initiative (ADQI) group, aid in the staging of patients with AKI:[12][13]

- Risk: 1.5-fold increase in the serum creatinine, or glomerular filtration rate (GFR) decrease by 25 percent, or urine output <0.5 mL/kg per hour for six hours.
- Injury: Two-fold increase in the serum creatinine, or GFR decrease by 50 percent, or urine output <0.5 mL/kg per hour for 12 hours
- Failure: Three-fold increase in the serum creatinine, or GFR decrease by 75 percent, or urine output of <0.3 mL/kg per hour for 24 hours, or anuria for 12 hours
- Loss: Complete loss of kidney function (e.g., need for renal replacement therapy) for more than four weeks
- End-stage renal disease: Complete loss of kidney function (e.g., need for renal replacement therapy) for more than three months

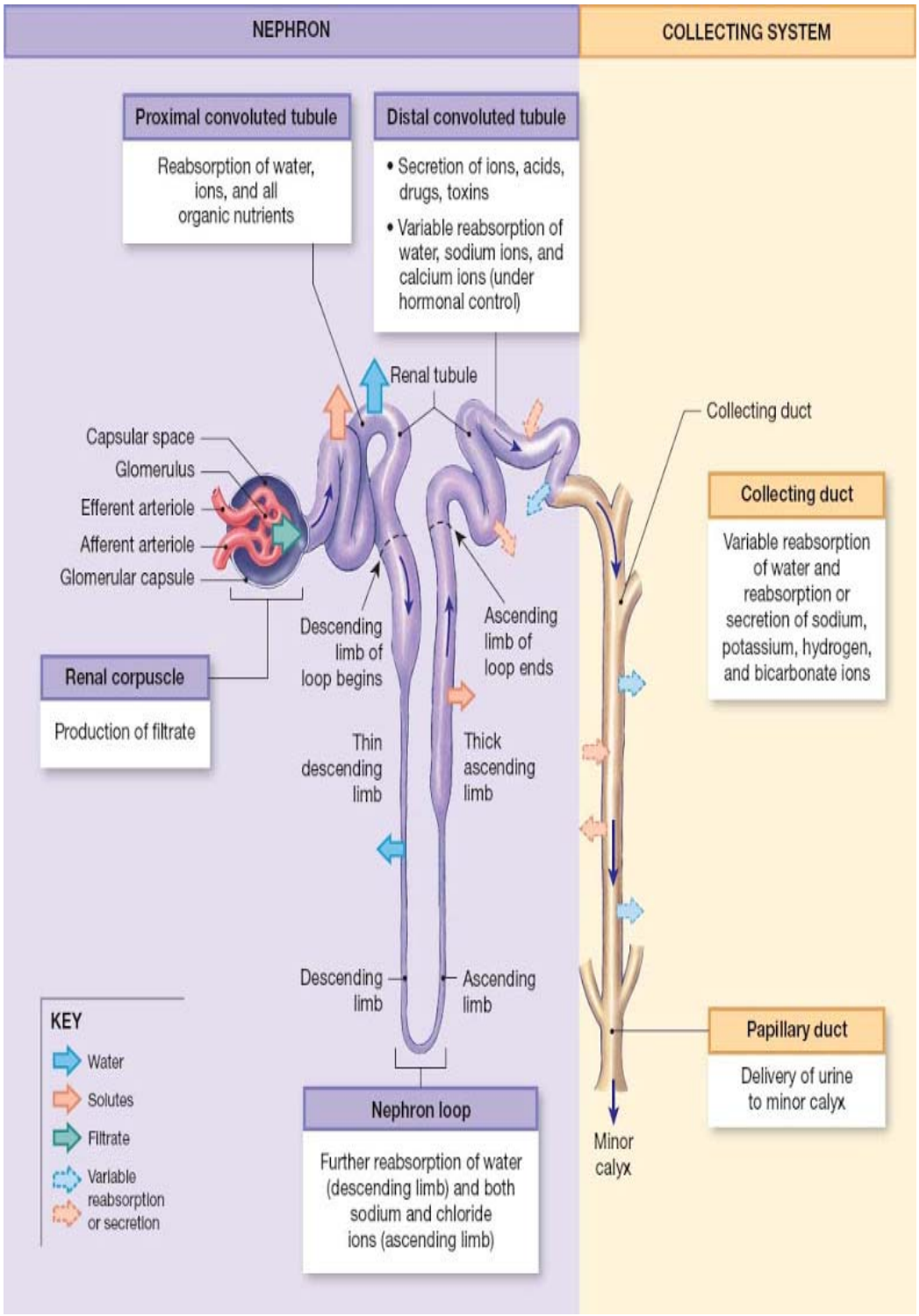



**Figure 2: Relative,hypoxia in the outer medulla predisposes to ischemic injury in the S3 segment of the proximal tubule.**

With the help of a step by step approach, physicians are able to determine the cause of AKI in most patients. According to one team evaluating AKI, " The time honoured approach to evaluating a patient with AKI is to exclude prerenal and post renal causes and then, if necessary, initiate an examination to determine the potential renal(intrinsic) etiologies"[13].

The first and primary step is to evaluate a thorough history and perform a complete and detailed physical examination. Key symptoms,and physical findings for AKI and uremia have been listed in detail in Table 1. On the basis of history and complete physical examination, the possible causes of AKI have been listed in Table 2 and 3, respectively.

*Supportive data* can be provided by blood and urinary examination. BUN, Electrolytes, Creatinine, Calcium, Phosphorous, Albumin levels along with complete blood count should be done in all patients. In certain specific,conditions, few other tests are indicated (Table 4). All patients must have to undergo following urine studies such as Dipstick Test, Urine Routine and microscopical examination, Serum creatinine and sodium levels as well as urine osmolality examination.

Often, it may be necessary to do a renal biopsy to establish a diagnosis, evaluate the prognosis or guide the therapy. Only small number of patients have indications for biopsy[18]. Biopsy is usually performed when a patients has intrinsic Acute Kidney Injury that is not Acute Tubular Necrosis. The most common complications,of biopsy are bleeding, arteriovenous fistula and death, but life threatening complications develop only in about 1 percent of the cases[15]. The differential diagnosis of Acute Kidney Injury is presented in Table 5.

**TABLE 1 : Key Symptoms and Physical Findings in Patients with Acute kidney injury and Uremia***

| **Symptoms** |
|---|
| Anorexia |
| Fatigue |
| Mental status changes |
| Nausea and vomiting |
| Pruritus |
| Seizures (if blood urea,nitrogen level is very high) |
| Shortness of breath (if volume overload is present) |
| **Physical findings** |
| Asterixis and myoclonus |
| Pericardial or pleural rub |
| Peripheral edema (if volume overload is present) |
| Pulmonary rales (if volume overload is present) |
| Elevated right atrial pressure (if volume overload is present) |

**TABLE 2 : Probable causes of acute kidney injury based on the findings of the history**

| HISTORY | PROBABLE CAUSES OF AKI |
|---|---|
| Review of systems | |
| Pulmonary system | |
| Sinus, upper respiratory or pulmonary symptoms | Pulmonary-renal syndrome or vasculitis |
| Cardiac system | |
| Symptoms of heart failure | Decreased renal perfusion |
| Intravenous drug abuse, prosthetic valve or valvular disease | Endocarditis |
| Gastrointestinal system | |
| Diarrhea, vomiting or poor intake | Hypovolemia |
| Colicky abdominal pain radiating from flank to groin | Urolithiasis |
| Genitourinary system | |
| Symptoms of benign prostatic hypertrophy | Obstruction |
| Musculoskeletal,system | |
| Bone pain in the elderly | Multiple myeloma or prostate cancer |
| Trauma or prolonged immobilization | Rhabdomyolysis (pigment nephropathy) |
| Skin | |
| Rash | Allergic interstitial nephritis, vasculitis, systemic lupus,erythematosus, atheroemboli or thrombotic thrombocytopenic purpura |
| Constitutional symptoms | |
| Fever, weight loss, fatigue or anorexia | Malignancy or vasculitis |
| Past medical history | |
| Multiple sclerosis, diabetes mellitus or stroke | Neurogenic bladder |
| Past surgical history | |
| Recent surgery or procedure | Ischemia, atheroemboli, endocarditis or exposure to contrast agent |
| Medication history | |
| Angiotensin-converting enzyme inhibitors, nonsteroidal anti-inflammatory drugs, antibiotics or acyclovir (Zovirax) | Decreased renal perfusion, acute tubular necrosis or allergic interstitial nephritis |

**TABLE 3 : Probable Causes of Acute kidney injury,based on the physical findings**

| **PHYSICAL EXAMINATION** | | **PROBABLE CAUSES OF AKI** |
|---|---|---|
| Vital signs | | |
| | Temperature | Possible infection |
| | Blood pressure | Hypertension: nephrotic syndrome or malignant hypertension |
| | | Hypotension: volume depletion or sepsis |
| | Weight loss,or gain | Hypovolemia or hypervolemia |
| Mouth | | Dehydration |
| Jugular veins and axillae (perspiration) | | Hypovolemia or hypervolemia |
| Pulmonary system | | Signs of congestive heart failure |
| Heart | | New murmur of endocarditis or signs of congestive heart failure |
| Abdomen | | Bladder distension suggesting urethral obstruction |
| Pelvis | | Pelvic mass |
| Rectum | | Prostate enlargement |
| Skin | | Rash of, interstitial nephritis, purpura of micro vascular disease, livedoreticularis suggestive of atheroembolic disease, or splinter hemorrhages or Osler's nodes of endocarditis |

**TABLE 4 : Findings of Blood Tests for Specific Types of Acute,kidney injury**

| ***FINDINGS ON BLOOD TESTS*** | ***DIAGNOSES TO CONSIDER*** |
|---|---|
| *Elevated uric acid level* | *Suggestive of malignancy or tumor lysis syndrome leading to uric acid crystals; also seen in prerenal acute kidney injury* |
| *Elevated creatine kinase or myoglobin levels* | *Rhabdomyolysis,* |
| *Elevated prostate-specific antigen* | *Prostate cancer* |
| *Abnormal serum protein electrophoresis* | *Multiple myeloma* |
| *Low complement levels* | *Systemic lupus erythematosus, post infectious glomerulonephritis, sub acute bacterial endocarditis* |
| *Positive antineutrophilic cytoplasmic antibody* | *Small-vessel vasculitis (Wegener's granulomatosis or polyarteritisnodosa)* |
| *Positive antinuclear,antibody or antibody to double-stranded DNA* | *Systemic lupus erythematosus* |
| *Positive antibody to glomerular basement membrane* | *Goodpasture's syndrome* |
| *Positive antibodies to streptolysin O, streptokinase or hyaluronidase* | *Post streptococcal glomerulonephritis* |
| *Schistocytes on peripheral smear, decreased haptoglobin level, elevated lactate dehydrogenase level or elevated serum bilirubin level* | *Hemolytic uremic syndrome or thrombotic thrombocytopenic,purpura* |
| *Low albumin level* | *Liver disease or nephrotic syndrome* |

**TABLE 5 : Differential Diagnosis of Acute kidney injury**

| TYPES OF ACUTE KIDNEY INJURY,AND UNDERLYING PROBLEM | POSSIBLE DISORDERS |
|---|---|
| Prerenal acute kidney injury | |
| True intravascular depletion | Sepsis, haemorrhage, over diuresis, poor fluid intake, vomiting, diarrhoea |
| Decreased effective circulating volume to the kidneys | Congestive heart failure, cirrhosis or hepatorenal syndrome, nephrotic syndrome |
| Impaired renal blood flow because of exogenous agents | Angiotensin-converting enzyme,inhibitors, nonsteroidal anti-inflammatory drugs |
| Intrinsic acute kidney injury | |
| Acute tubular necrosis | Ischemia |
| | Toxins: drugs (e.g., aminoglycosides), contrast agents, pigments (myoglobin or hemoglobin) |
| Glomerular disease | Rapidly progressive glomerulonephritis: systemic lupus erythematosus, small-vessel vasculitis (Wegener's granulomatosis or polyarteritisnodosa), Henoch-Schönleinpurpura,(immunoglobulin A nephropathy), Goodpasture's syndrome |
| | Acute proliferative glomerulonephritis: endocarditis, post streptococcal infection, post pneumococcal infection |
| Vascular disease | Microvascular disease: atheroembolic disease (cholesterol-plaque microembolism), thrombotic thrombocytopenic purpura, hemolytic uremic syndrome,,HELLP syndrome or postpartum acute kidney injury |
| | Macrovascular disease: renal artery occlusion, severe abdominal aortic disease (aneurysm) |
| Interstitial disease | Allergic reaction to drugs, autoimmune disease: (systemic lupus erythematosus or mixed connective tissue disease), pyelonephritis, infiltrative disease (lymphoma or leukemia) |
| Post renalacute kidney injury | Benign prostatic hypertrophy or prostate cancer, cervical cancer, retroperitoneal disorders, intratubular,obstruction (crystals or myeloma light chains), pelvic mass or invasive pelvic malignancy, intraluminal bladder mass (clot, tumor or fungus ball), neurogenic bladder, urethral strictures |

Pre Renal Acute Kidney Injury

In pre renal Acute Kidney Injury, the main problem is impairment of renal blood flow due to true intravascular depletion, decrease in effective circulating volume to the kidneys or agents which lead to impairment of renal blood flow. Urine and blood investigations are useful in diagnosing pre renal Acute Kidney Injury. Differentiating features include bland urine sediment (Table 6)[3] a urine osmolality of greater than 500 mOsm and a BUN to serum creatinine ratio of greater than 20:1. (Table 7)

It is necessary to determine the fractional exertion of sodium. The level of filtered sodium which is ultimately excreted is equal to,100 X 3 (urine sodium/serum sodium) X (urine creatinine/ serum creatinine). The value is found to be less than 1 percent in majority of the patients with pre renal Acute Kidney Injury. In patients with pre renal Acute Kidney Injury, the renal parenchyma is not damaged and the kidneys respond as if volume depletion has developed. Thus, sodium is reabsorbed by the kidneys in order to reabsorb water.

There are very few causes for fractional excretion of sodium of less than,1 percent which are not the result of pre renal Acute Kidney Injury such as Contrast Induced Nephropathy and Pigment Nephropathy.

Patients receiving diuretics may truly have developed pre renal Acute Kidney Injury, but there may be fractional excretion of sodium which may be increased by diuretic induced sodium excretion.

Now, patients on diuretic therapy might have pre renal Acute Kidney Injury, but the sodium fractional excretion may increase due to sodium,excretion (secondary to diuretic use). Also, there would be impairment in sodium reabsorption in patients with

chronic renal insufficiency. Thus, if they develop pre renal Acute Kidney Injury, they may not,be able to reabsorb enough sodium to have a less than 1 percent sodium fractional excretion.

Acute Kidney Injury in patients with congestive cardiac failure develops due to fall in renal blood flow. This develops due to either hypovolemia – due to overdiuresis or hypervolemia – due to elevated filling pressures of left ventricle leading to decrease in cardiac output.

Patients in hypovolemic group may respond to hydration with discontinuation of diuretics. Patients in the hypervolemic group require diuretics, inotropes and vasodilators. Fluid management with invasive hemodynamic monitoring is also needed.

The agents which cause pre renal Acute Kidney Injury are ACEI(Angiotensin converting enzyme inhibitors) and NSAIDs(Non steroidalanti inflammatory drugs). ACEI(Angiotensin converting enzyme inhibitors) prevents the conversion of,Angiotensin I to Angiotensin II, leading to fall in levels of Angiotensin II. Angiotensin II leads to rise in GFR(glomerular filtration rate) by efferent arteriole constriction; its absence decreases GFR(glomerular filtration rate) by efferent arteriole dilatation.

In patients with hypovlemia or bilateral renal artery stenosis, GFR(glomerular filtration rate)is dependent on the effects of Angiotensin II. If such patients take an ACEI (Angiotensin converting enzyme inhibitors), their GFR(glomerular filtration rate) falls leading to pre renal Acute Kidney Injury.

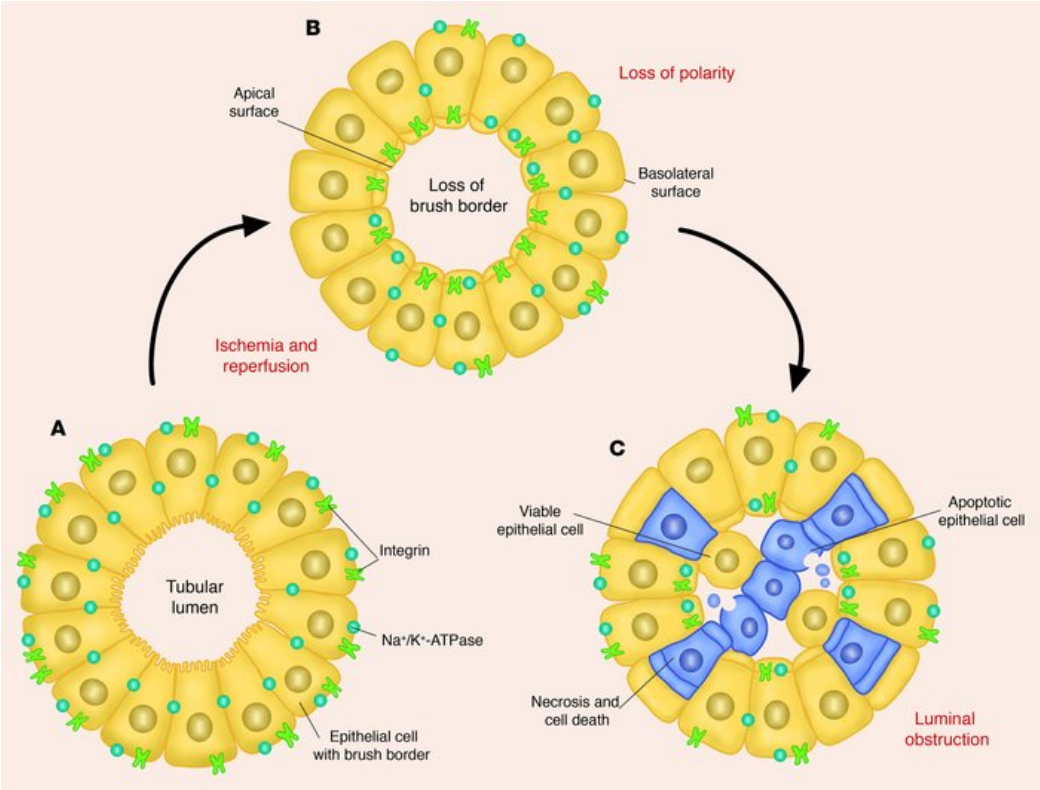
As the patient starts an ACEI (Angiotensin converting enzyme inhibitors), levels of serum potassium, blood-urea-nitrogen and serum creatinine must be monitored. NSAIDs (Non steroidal anti-inflammatory drugs) alter the local,glomerular arteriolar perfusion leading to pre renal Acute Kidney Injury by blocking prostaglandin production.

Decreased renal blood flow leads to ischemia in renal parenchyma. ATN (Acute Tubular Necrosis) develops if ischemia is prolonged. Early restoration of renal blood flow shortens ischemia time and prevents parenchymal injury. A response to restoration of renal blood flow must take place within 24 to 48 hours.

The keys to therapy are – treat underlying disorder, maintain euvolemia and eliminate toxic agents.

Intrinsic Acute Kidney Injury

Intrinsic Acute Kidney Injury is mainly of 4 types – Tubular Disease, Glomerular Disease, Vascular Disease and Interstitial Disease. Intrinsic Acute Kidney Injury, the renal parenchyma is injured. The damage to tubule cells leads to certain urinary microscopic findings. Impairment in sodium reabsorption occurs due to,parenchymal injury resulting in sodium fractional excretion greater than 3 percent and an isotonic urine osmolality of 250-300 mOsm.

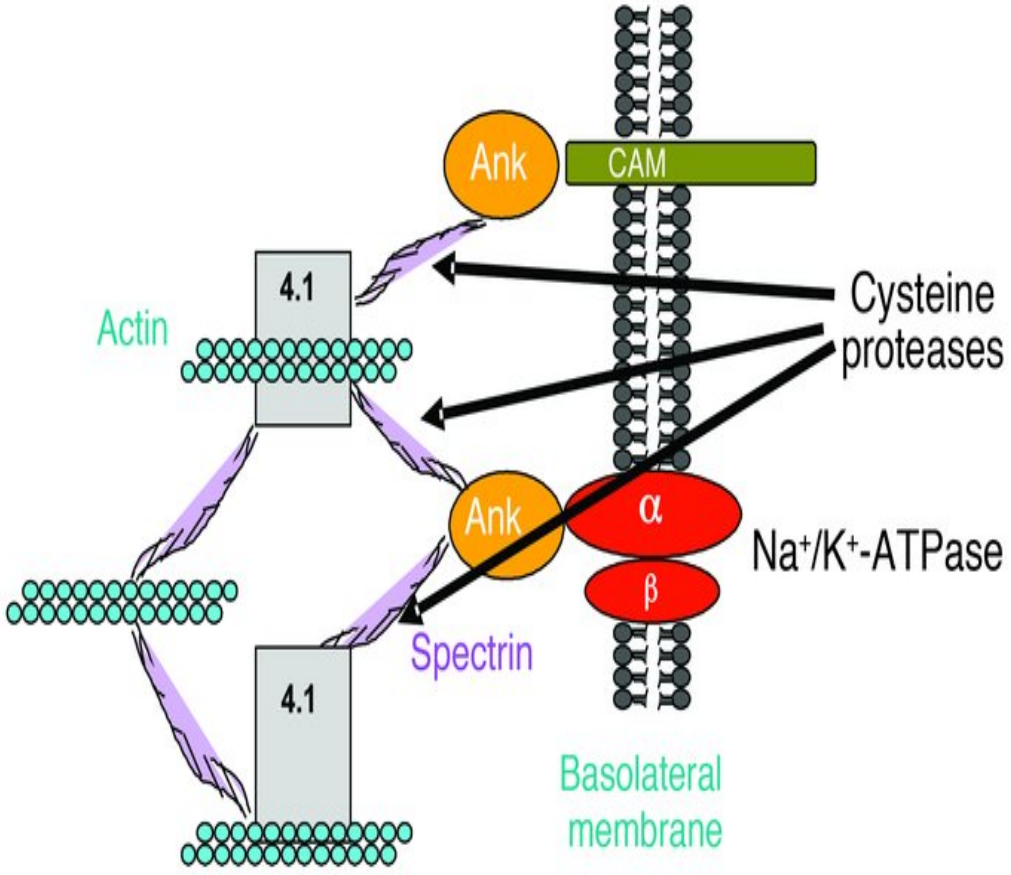



Above figure indicates that following ischemia and reperfusion, morphological changes occur in the proximal tubules, including loss of polarity, loss of the brush border, and redistribution of integrins and $Na^+/K^+$-ATPase to the apical surface. Calcium and reactive oxygen species may also have a role in these morphological changes, in addition,to subsequent cell death resulting from necrosis and apoptosis. Both viable and nonviable cells are shed into the tubular lumen, resulting in the formation,of casts and luminal obstruction and contributing to the reduction in the GFR.

Tubular Disease

ATN (Acute Tubular Necrosis) is the commonest cause of Intrinsic Acute Kidney Injury in hospitalized patients. In Ischemic ATN (Acute Tubular Necrosis), unlike pre renal Acute Kidney Injury, the GFR (Glomerular filtration rate) does not improve

with renal blood flow restoration. Ischemic ATN( Acute Tubular Necrosis) is usually, reversible unless necrosis has already occurred[14-15].




Above figure shows potential cytoskeletal targets of cysteine proteases during hypoxia/ischemia in proximal tubules.

Agents which cause Acute Tubular Necrosis are aminoglycosides and contrast agents. It may result by pigment from hemoglobinuria(hemolysis) or myoglobinuria (rhabdomyolysis).

Acute Tubular Necrosis has mainly 3 phases[14] – Initiation Phase,,in which renal injury evolves lasting hours to days; Maintenance Phase, in which glomerular filtration rate and urine output becomes low lasting from days to weeks; Recovery Phase which

begins with post acute tubular necrosis diuresis lasting for days. Main concern in this phase is hypovolemia due to excessive urine output. Inspite of urinary production recovery, patients may have problems like uremia, disturbance in electrolytes and acid levels due to incomplete recovery of tubular function. All phases of acute tubular necrosis require management and monitoring.

Patients with Diabetes Mellitus, congestive cardiac failure or chronic renal insufficiency are at higher risk for acute tubular necrosis. By treating patients with reversible causes of ischemia or pre renal acute kidney injury and through,appropriate hydration in patients who are getting nephrotoxic agents, acute tubular necrosis can be prevented. Animal studies show improvement in acute tubular necrosis by using agents such as mannitol, loop diuretics, dopamine and calcium channel blockers[19].

Glomerular Disease

Glomerulonephritis is mainly characterised by 3 parts – Hypertension, Proteinuria and Hematuria[6]. Of the various types of glomerulonephritis, most varieties,are associated with chronic kidney disease.

Furthermore, glomerulonephritis can be further divided into two types – Rapidly Progressive Glomerulonephritis(RPGN) and Acute Proliferative Glomerulonephritis. Patients with bacteria endocarditis or other such infectious conditions develop the second type.

Rapidly Progressive Glomerulonephritis(RPGN) can develop primarily or secondary to a systemic disorder. Once such a condition is suspected, the systemic,disease must be south via serological markers or renal biopsy and must be treated accordingly.

In patients of Rapidly Progressive Glomerulonephritis(RPGN), renal function[20] may quickly deteriorate and end-stage renal disease may develop over days to weeks.

Main line of therapy for patients with Rapidly Progressive Glomerulonephritis (RPGN) is cyclophosphamide and glucocorticoids. Patients with Goodpasture's syndrome have shown improvement with plasma exchange but similar findings have not been seen in other types of glomerulonephritis.

Post renal Acute kidney injury if both the outflow tract of obstructed. Mainly occurs in obstruction of lower urinary tract. Patients with severe oliguria or anuria (output less than 100 ml of urine per day) are more susceptible to develop post renal,acute kidney injury. Although, all patients of post renal acute kidney injury are not oliguric.

Primary causes of post renal acute kidney injury are benign hypertrophy of prostate, Carcinoma of prostate, Carcinoma of cervix, Retroperitoneal disorders. Intratubular causes are crystals and myeloma light chains[15].

Initially in patients with Acute Kidney Injury, it must be found whether a patient has post renal acute kidney injury, as the management is easy and there is an inverse, relationship between duration of obstruction and renal function recovery. Patients with obstruction in bladder or urethra may require bladder catheterization.

In ultrasonographic examination, obstruction may be indicated by development of hydronephrosis. Sensitivity of above diagnostic technique is 90 percent and specificity is 100 percent. If the obstruction Is very early or if there is retroperitoneal fibrosis, ultrasound may give a false positive result. In patients of post renal acute kidney injury, management must be directed towards the underlying disease.

Various techniques available for management are – Percutaneous Nephrostomy, Ureteral Stenting, Bladder Catheterization, Lithotripsy, Urethral Stenting.

**Causes of Anuria**

| |
|---|
| Obstruction (vast majority of patients with anuria) |
| Bilateral renal cortical necrosis |
| Fulminant glomerulonephritis (usually some type of rapidly progressive glomerulonephritis) |

***Findings on,Urinalysis in the Broad Categories of Acute kidney injury***

| **TYPE OF RENAL FAILURE** | **FINDINGS ON URINALYSIS** |
|---|---|
| Prerenal acute kidney injury | Scant; few hyaline casts |
| Post renalacute kidney injury | Scant; few hyaline casts, possible red cells |
| Acute tubular necrosis | Epithelial cells, muddy-brown,coarsely granular casts, white blood cells, low-grade proteinuria |
| Allergic interstitial nephritis | White blood cells, red blood cells, epithelial cells, eosinophils, possible white blood cell cast, low to moderate proteinuria |
| Glomerulonephritis | Red blood cell casts, dysmorphic red cells, severe proteinuria |

***Blood and Urine Studies to Distinguish,Prerenal from IntrinsicAcute kidney injury***

| TYPE OF RENAL FAILURE | BUN-TO-CREATININE RATIO | URINE OSMOLALITY | FRACTIONAL EXCRETIONOF SODIUM* |
|---|---|---|---|
| Prerenal acute kidney injury | > 20:1 | > 500 mOsm | < 1% |
| Intrinsic acute kidney injury | < 20:1 | 250 to 300 mOsm | > 3% |

BUN = blood urea nitrogen (mg per dL).*The fractional excretion of sodium is calculated using the following formula: 100 × (urine sodium/serum sodium) ÷ (urine creatinine/serum creatinine)

Vascular Disease

Acute Kidney Injury may be due to micro vascular or macrovasculardisease( major renal artery occlusion or severe abdominal aortic disease)[22].

The classic micro vascular diseases have a presentation of microangiopathic hemolysis and acute kidney injury due to thrombosis or occlusion of glomerular capillary plus thrombocytopenia. Such a condition is seen in HUS,(Hemolytic Uremic Syndrome), TTP(Thrombotic Thrombocytopenic Purpura), HELLP Syndrome(Hemolysis, Elevated Liver Enzymes and Low Platelets).

The classic pentad of TTP(Thrombotic Thrombocytopenic Purpura) consists of pyrexia, neurological changes, thrombocytopenia and microangiopathichaemolytic anemia. HUS( Hemolytic Uremic Syndrome) presents with all changes of TTP,except neurological changes. HELLP Syndrome is a type of HUS occurring in pregnancy with rise in levels of transaminases.

Plasmapheresis and corticosteroids are required to treat patients of micro vasculardisease leading to acute kidney injury. In patients with parturition-related acute kidney injury (HELLP syndrome), expedition of delivery is the initial treatment of choice.

Atheroembolic disease is another important cause of irreversible acute kidney injury. Patients with atherosclerotic disease who undergo an invasive procedure (e.g., vascular surgery or interventional vascular studies) or have an acute arrhythmia are at increased risk for acute kidney injury induced by atheroemboli. Acute kidney injury from embolic disease may present one day to seven weeks after the inciting event.

Atheroembolism is relatively common in tertiary care and intensive care units, presenting classically with "purple toes and renal failure." Evidence of microembolism may be present in other organs (livedoreticularis, gastrointestinal,tract bleeding, pancreatitis, persisting encephalopathy and retinal embolism seen as "Hollenhorst" plaques). The diagnosis of atheroembolic disease can be confirmed on skin or renal biopsy. Treatment is nonspecific, but avoiding further vascular intervention and anticoagulation is strongly recommended.

Intrinsic Disease

Acute interstitial nephritis usually presents with fever, rash and,eosinophilia. Urine staining that is positive for eosinophils is suggestive of this condition. Acute interstitial nephritis is usually the result of an allergic reaction to a drug, but it may also be caused by autoimmune disease, infection or infiltrative disease.

Many drugs can cause acute interstitial nephritis, but the more common ones are NSAIDs, penicillins, cephalosporins, sulfonamides, diuretics and allopurinol

(Zyloprim). Renal function should improve after the offending agent,is withdrawn. Corticosteroids are sometimes helpful in speeding recovery[26].

# MATERIALS & METHODS

- Study Site:- This study wascarried out at DHIRAJ GENERAL HOSPITAL, PIPARIA. It is a tertiary care centre with 1200 beds, a 24-hour emergency department and Intensive Care Units.
- Type of Study:- Pilot Study
- Sample Size:- Minimum 45 patients and maximum as many as possible.
- Study Period:-The study wascarried out over a period of one yearfrom January 2016 to December 2016.

DATA COLLECTION & ANALYSIS-

- The study was carried out in the medicine department at SBKS & MIRC after approval from the institutional ethics committee.
- The study was carried out over a period of one year.
- All patients with AKI at the time of admission in ICU in the department of medicine and fulfilling inclusion criteria were enrolled for the study.
- Detailed history and clinical examination was carried out in all these patients.
- Laboratory investigations were carried out rationally based on clinical differential diagnosis.
- Results were analyzed applying appropriate statistical methods and formulas.

INCLUSION CRITERIA

- Both Sexes, Age 18 to 70 years
- Patients presenting with AKI during ICU stay
- Newly detected HTN/DM

EXCLUSION CRITERIA

- Patients who refuse to give the informed written consent.
- K/C/O Type II Diabetes Mellitus and Hypertension
- Chronic Kidney Disease patients on maintenance Haemodialysis

INVESTIGATIONS PERFORMED:

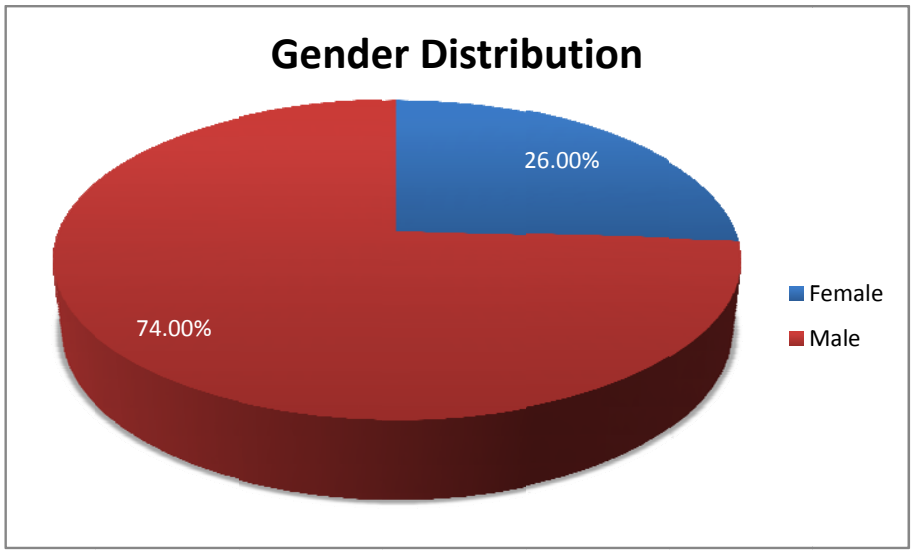
- Hemogram with Peripheral Smear
- Urea
- S. Creatinine
- S. $Na^{+}$
- S. $K^{+}$
- S. $Cl^{-}$
- S. $HCO_3^{-}$
- Urine – RM
- Urine input/ output
- S. Uric Acid
- USG – Abdomen and pelvis
- LFT
- S. $Ca^{++}$
- S. $PO_4^{3-}$
- Sickling
- PS for MP
- Urine – CS[as and when indicated]

# RESULTS

**Table 1: Gender Distribution in AKI patient**

| Gender | N | % |
|---|---|---|
| **Female** | 13 | 26.0 |
| **Male** | 37 | 74.0 |
| **Total** | 50 | 100.0 |

Graph 1: Gender Distribution in AKI patients

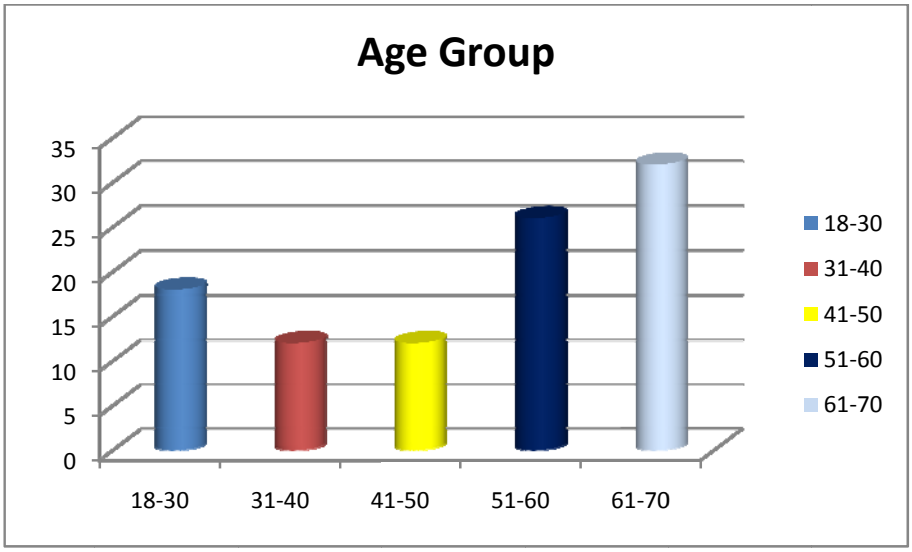



Among 50 patients studied, majority of the patients were male 37(74%) and females were 13(26%) with a male to female ratio of 2.84:1(Table 1)

**Table 2: Age Group in AKI patients**

| Age group | N | % |
|---|---|---|
| **18-30** | 9 | 18.0 |
| **31-40** | 6 | 12.0 |
| **41-50** | 6 | 12.0 |
| **51-60** | 13 | 26.0 |
| **61-70** | 16 | 32.0 |

**Graph 2: Age Group in AKI patients**

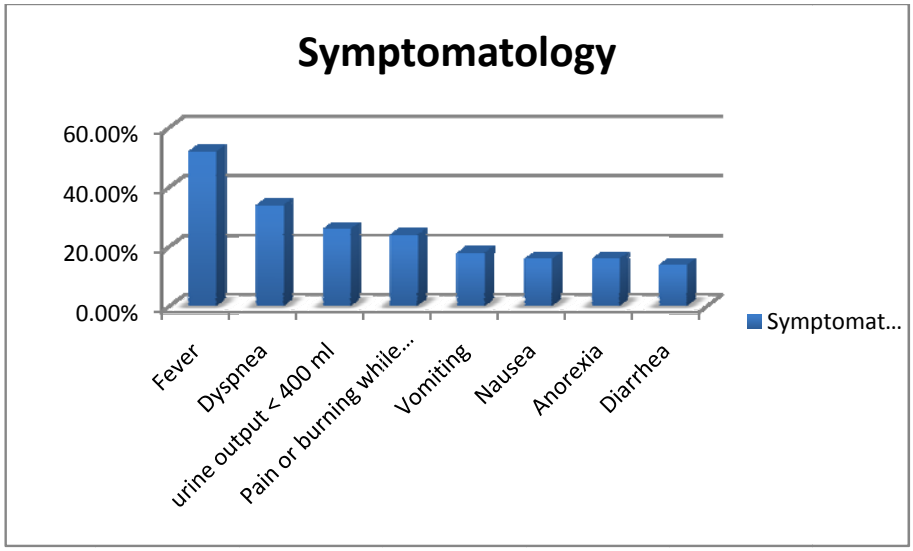



Amongst the studied patients, mean age was found to be 50.40 ± 16.21, minimum age being 18 years and maximum age being 70 years. Majority of the patients were in the age group of 61 – 70 years (32%) followed by 51-60 years (26%), 18-30 years (18%), 31-40 years (12%) and 41-50 years (12%)(Table 2).

**Table 3: Symptomatology in AKI patients**

| Symptomatology | N | % |
|---|---|---|
| **Fever** | 26 | 52.00% |
| **Dyspnea** | 17 | 34.00% |
| **Oliguria** | 13 | 26.00% |
| **Dysuria** | 12 | 24.00% |
| **Vomiting** | 9 | 18.00% |
| **Nausea** | 8 | 16.00% |
| **Anorexia** | 8 | 16.00% |
| **Diarrhea** | 7 | 14.00% |

**Graph 3: Symptomatology in AKI patients**



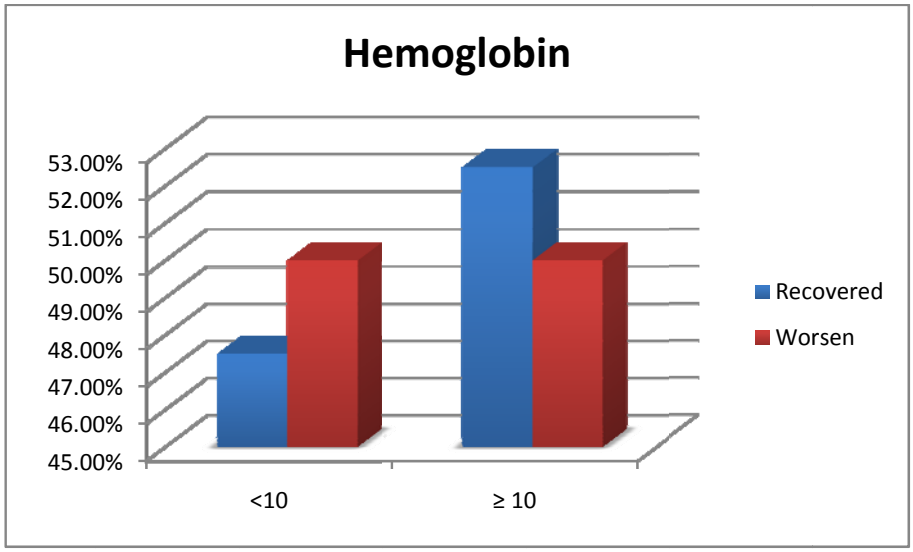

Among the studied patients, fever, dyspnea and oliguria were most common presentations. Symptoms recorded were fever in 26(52%) followed by dyspnea in 17 (34.00%), oliguria in 13 (26.00%), burning micturition in 12 (24.00%), vomiting in 9 (18.00%), nausea and anorexia in 8 (16.00% each), diarrhea (Table 3).

**Table 4: Laboratory Parameters in AKI patients**

**Table 4.1 Hemoglobin in AKI patients**

| Hb (gm%) | Total | Recovered | | Worsen | |
|---|---|---|---|---|---|
| | | N | % | N | % |
| **<10** | 24 | 19 | 79.16% | 5 | 20.84% |
| **≥ 10** | 26 | 21 | 80.76% | 5 | 19.24% |
| **Total** | 50 | 40 | 80.00% | 10 | 20.00% |

**Graph 4.1 Hemoglobin in AKI patients**



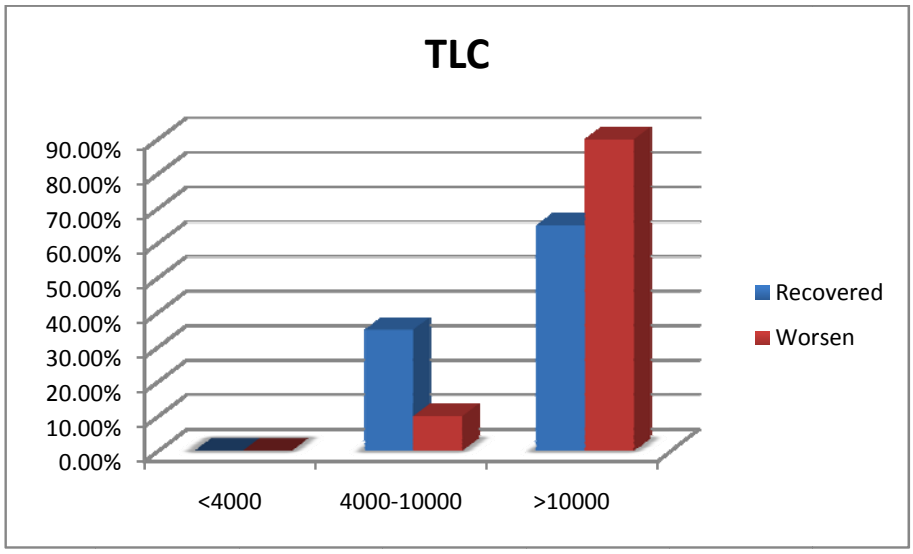

Among studied patients, 24 had haemoglobin less than 10 gm/dl and 26 had haemoglobin more than 10 gm/dl. Of 40 recovered patients, 19 had hemoglobin <10 and 21 had >10 and among 10 worsened patients, 5 had haemoglobin>10 and 5 had >10gm/dl(Table 4.1).

**Table 4.2 Total Leukocytic Count in AKI patients**

| TLC( cells/ cu.mm) | Total | Recovered | | Worsen | |
|---|---|---|---|---|---|
| | | N | % | N | % |
| **<4000** | 0 | 0 | 0.00% | 0 | 0.00% |
| **4000-10000** | 15 | 14 | 93.33% | 1 | 6.67% |
| **>10000** | 35 | 26 | 74.28% | 9 | 25.72% |
| **Total** | 50 | 40 | 80.00% | 10 | 20.00% |

| Parameter | No. | <10000 | SD | No. | >10000 | SD | p value |
|---|---|---|---|---|---|---|---|
| TLC | 30 | 7826.67 | 1574.11 | 20 | 15248.57 | 4571.61 | 0.000 |

**Graph 4.2 Total Leukocytic Count in AKI patients**



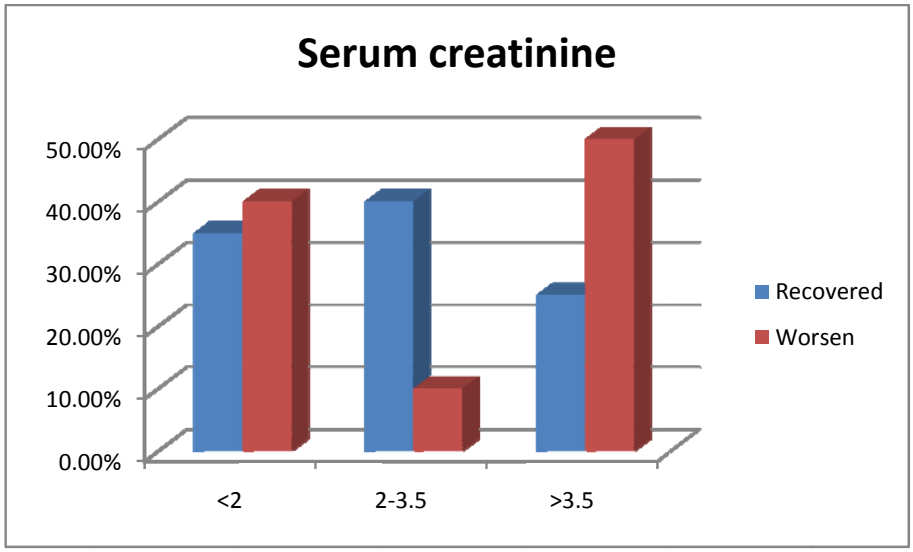

Among the 50 patients studied, 15 had TLC 4000-10000 and 35 had more than 10000. Of 40 recovered patients, 14 had TLC 4000-10000 and 26 had TLC >10000 and among 10 worsened patients, 1 had TLC 4000-10000 and 9 had TLC >10000(Table 4.2). Also, comparison of patients with <10000 cells/ cu.mm with >10000 cells/ cu.mm showed a highly significant p value of 0.000(Table 4.2).

**Table 4.3 Serum Creatinine in AKI patients**

| Creatinine (mg/dl) | Total | Recovered | | Worsen | |
|---|---|---|---|---|---|
| | | N | % | N | % |
| **<2** | 18 | 14 | 77.78% | 4 | 22.22% |
| **2-3.5** | 17 | 16 | 94.12% | 1 | 5.88% |
| **>3.5** | 15 | 10 | 66.67% | 5 | 33.33% |
| **Total** | 50 | 40 | 80.00% | 10 | 20.00% |

| Parameter | No. | < 5 | SD | >5 | Abnormal | SD | p value |
|---|---|---|---|---|---|---|---|
| Creatinine | 38 | 2.23 | 0.75 | 12 | 8.88 | 5.02 | 0.000 |

**Graph 4.3 Serum Creatinine in AKI patients**



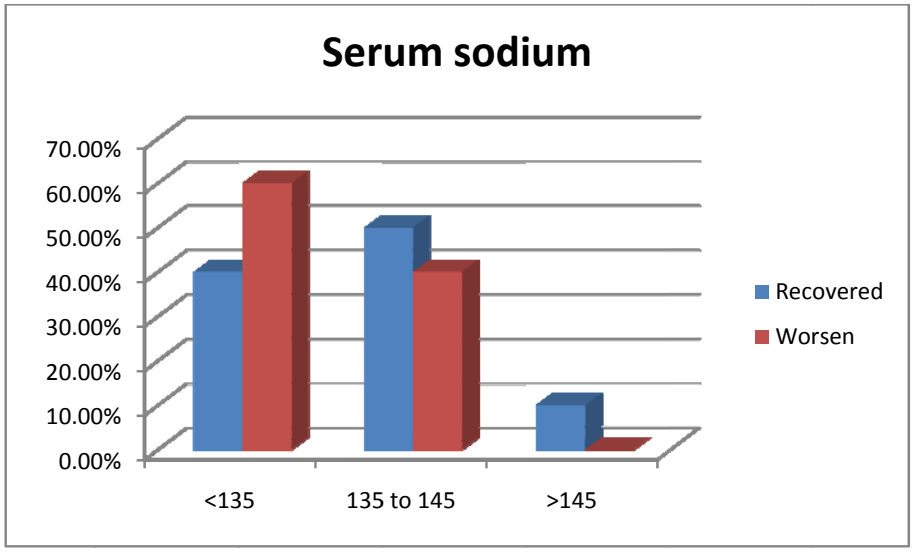

Among 50 patients studied, 18 had creatinine less than 2, 17 had creatinine between 2-3.5and 15 had more than 3.5. Of 40 recovered patients, 14 had creatinine <2, 16 had 2-3.5 and 10 had >3.5 and among 10 worsened patients, 4 had creatinine <2, 1 had 2-3.5 and 5 had >3.5(Table 4.3). Also, comparison of patients with <5 mg/dl with >5 mg/dl showed a highly significant p value of 0.000(Table 4.3).

### 4.4 Serum Sodium in AKI patients

| Na( mmol/L) | Total | Recovered | | Worsen | |
|---|---|---|---|---|---|
| | | N | % | N | % |
| **<135** | 22 | 16 | 72.72% | 6 | 27.28% |
| **135 to 145** | 24 | 20 | 83.33% | 4 | 16.67% |
| **>145** | 4 | 4 | 100.00% | 0 | 0.00% |
| **Total** | 50 | 40 | 80.00% | 10 | 20.00% |

| Parameter | No. | <135 | SD | No. | >135 | SD | p value |
|---|---|---|---|---|---|---|---|
| Sodium | 24 | 128.17 | 5.51 | 26 | 142.00 | 5.22 | 0.000 |

**Graph 4.4 Serum Sodium in AKI patients**

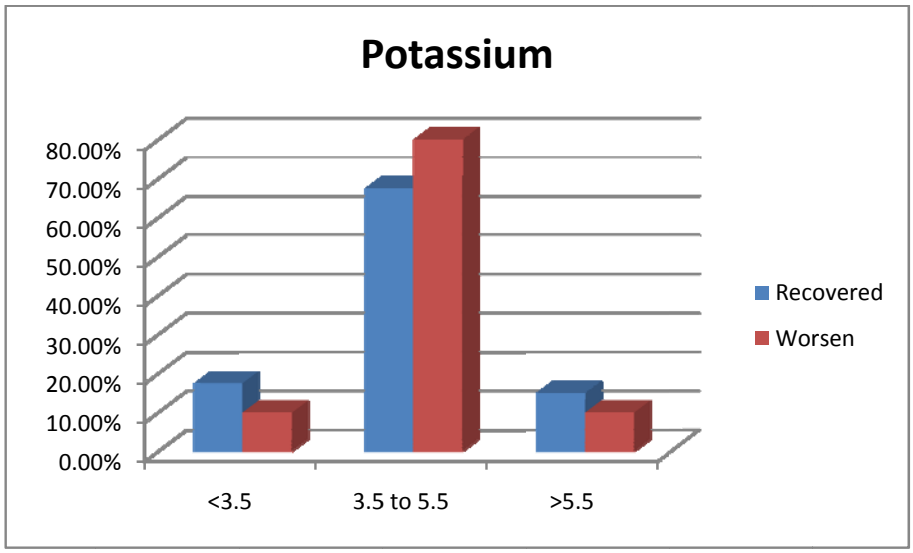



Among 50 patients studied, serum sodium was less than 135 in 22 patients, 135 to 145 in 24 patients and more than 145 in 4 patients. Of 40 recovered patients, 16 had sodium <135, 20 had 135-145 and 4 had >145 and among 10 worsened patients, 6 had sodium <135, 4 had 135-145 (Table 4.4). Also, comparison of patients with <135 mg/dl with >135 mg/dl showed a highly significant p value of 0.000(Table 4.4).

**Table 4.5 Serum Potassium in AKI patients**

| K ( mmol/L) | Total | Recovered | | Worsen | |
|---|---|---|---|---|---|
| | | N | % | N | % |
| **<3.5** | 8 | 7 | 87.50% | 1 | 12.50% |
| **3.5 to 5.5** | 35 | 27 | 77.14% | 8 | 22.86% |
| **>5.5** | 7 | 6 | 85.72% | 1 | 14.28% |
| **Total** | 50 | 40 | 80.00% | 10 | 20.00% |

**Graph 4.5 Serum Potassium in AKI patients**




Among 50 patients studied, serum potassium was less than 3.5 in 8 patients, 3.5 to 5.5 in 35 patients and more than 5.5 in 7 patients. Of 40 recovered patients, 7 had potassium <3.5, 27 had 3.5-5.5 and 6 had >5.5 and among 10 worsened patients, 1 had potassium<3.5, 8 had 3.5-5.5 and 1 had >5.5(Table 4.5)

**Table 5: Urine culture in AKI patients**

| Microbiology | | | | |
|---|---|---|---|---|
| Parameter | **Positive** | **%** | **Negative** | **%** |
| **Urine Culture** | 14 | 28.00% | 36 | 72.00% |

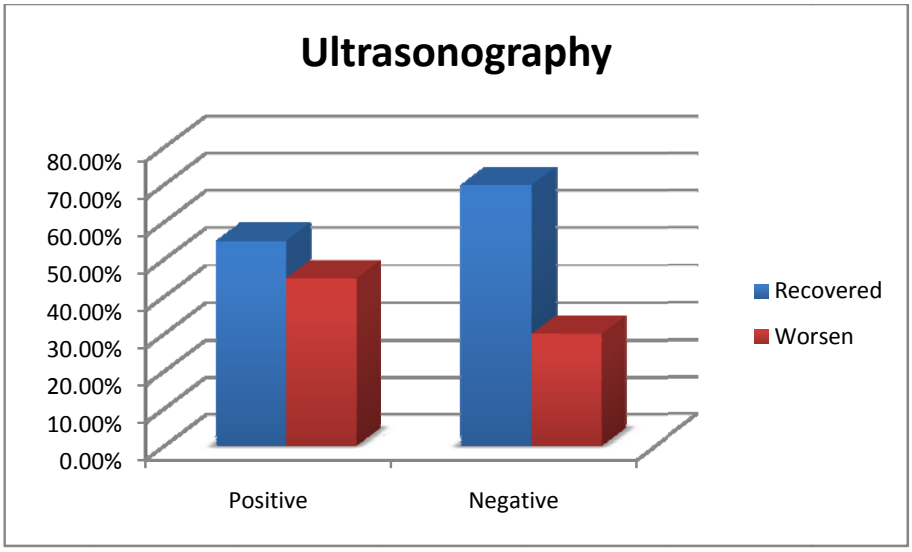
| **Recovered** | | **Worsened** | |
|---|---|---|---|
| **Positive** | **%** | **Positive** | **%** |
| **10** | 71.42% | 4 | 28.58% |

In this study 28.00% had positive urine culture, of which 10(71.42%) recovered and 4(28.58%) worsened. Microorganisms found positive in urine culture were candida albicans, Escherichia coli, proteus mirabilis, klebsiella species and pseudomonas species (Table 5).

**Table 6 Ultrasonography in AKI patients**

| **Ultrasono-graphy** | **Total** | **Recovered** | | **Worsen** | |
|---|---|---|---|---|---|
| | | **N** | **%** | **N** | **%** |
| **Positive finding** | 29 | 22 | 75.86% | 7 | 24.14% |
| **Negative finding** | 21 | 18 | 85.72% | 3 | 14.28% |
| **Total** | 50 | 40 | 80.00% | 10 | 20.00% |

**Graph 6 Ultrasonography in AKI patients**

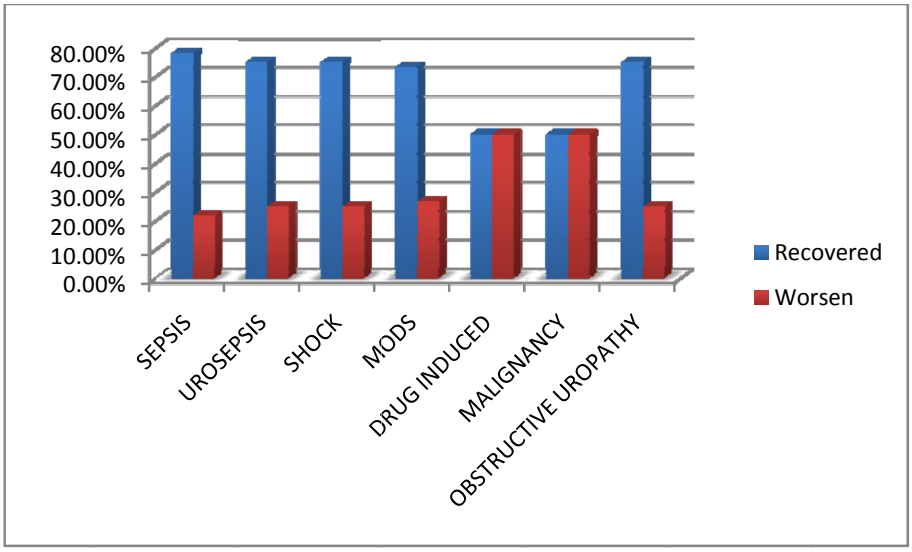



Among 50 patients studied, 29 had positive ultrasonographic finding as renal or bladder calculi, hydronephrosis, hydroureter, stricture urethra, pyelonephritis, raised renal echotexture while in 21 patients, ultrasonography was normal(Table 6).

**Table 7.1: Underlying causes in AKI patients**

| Underlying cause | Total | Recovered | % | Worsen | % |
|---|---|---|---|---|---|
| SEPSIS | 32 | 25 | 78.13% | 7 | 21.88% |
| UROSEPSIS | 20 | 15 | 75.00% | 5 | 25.00% |
| SHOCK | 16 | 12 | 75.00% | 4 | 25.00% |
| MODS | 15 | 11 | 73.33% | 4 | 26.67% |
| DRUG INDUCED | 8 | 4 | 50.00% | 4 | 50.00% |
| MALIGNANCY | 2 | 1 | 50.00% | 1 | 50.00% |
| OBSTRUCTIVE UROPATHY | 4 | 3 | 75.00% | 1 | 25.00% |

**Graph 7.1: Underlying causes in AKI patients**



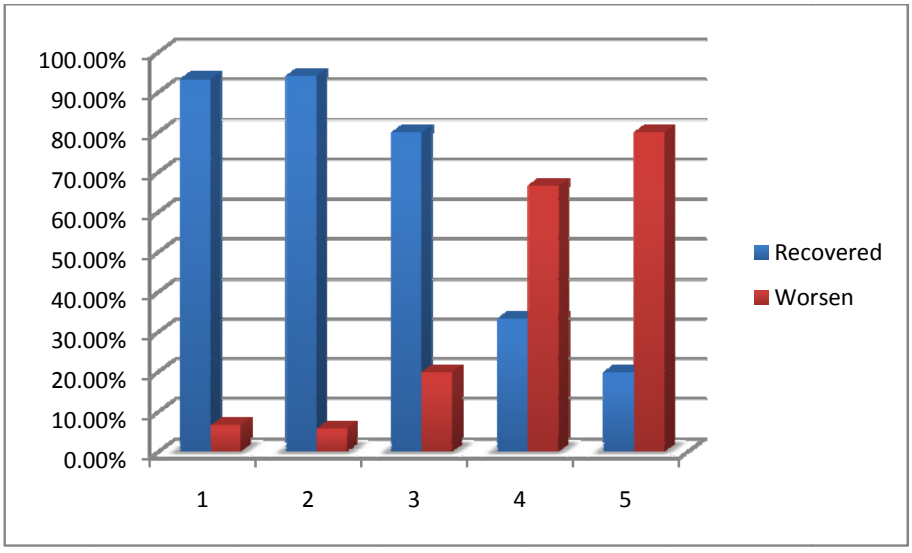

From above table and graph it has been concluded that Sepsis was the most common condition in patients(32 patients) and out of them 25(78.13%) patients were recovered and 7(21.88%) patients were not recovered. Urosepsis was found in 20 patients, out of them 15(75%) were recovered and 5(25%) were not recovered. Shock was found in 16 patients out of them 12(75%) were recovered and 4(25%) were not recovered. MODS were found in 15 patients, out of them 11(73.33%) patients were recovered and 4(26.67%) patients were not recovered. Drug induced was found in 8 patients out of them 4(50%) were recovered and 4(50%) were not recovered. Malignancy was found in 2 patients out them 1(50%) patient was recovered and 1(50%) patient was not recovered. Obstructive Uropathy was found in 4 patients out of them 3(75%) patient were recovered and 1(25%) patient was not recovered.

**Table 7.2: "MODUS" Score in AKI patients**

| MODUS Score | Total | % | Recovered | % | Worsen | % |
|---|---|---|---|---|---|---|
| 1 | 15 | 30% | 14 | 93.33% | 1 | 6.67% |
| 2 | 17 | 34% | 16 | 94.12% | 1 | 5.88% |
| 3 | 10 | 20% | 8 | 80.00% | 2 | 20.00% |
| 4 | 3 | 6% | 1 | 33.33% | 2 | 66.67% |
| 5 | 5 | 10% | 1 | 20.00% | 4 | 80.00% |
| Total | 50 | 100% | 40 | 80.00% | 10 | 20.00% |

As per data in Table 7.1, each cause has been assigned a score. Sepsis = 1, urosepsis = 1,shock = 1, MODS = 1, drug induced = 1, malignancy = 1, obstructive uropathy = , ventilator support = 1 and hemodialysis = 1. **MODUS Score** stands for M = MODS, Malignancy; O = Obstructive Uropathy; D = Drug Induced; U = Urosepsis; S = Sepsis, Shock.

**Graph 7.2: "MODUS" Score in AKI patients**



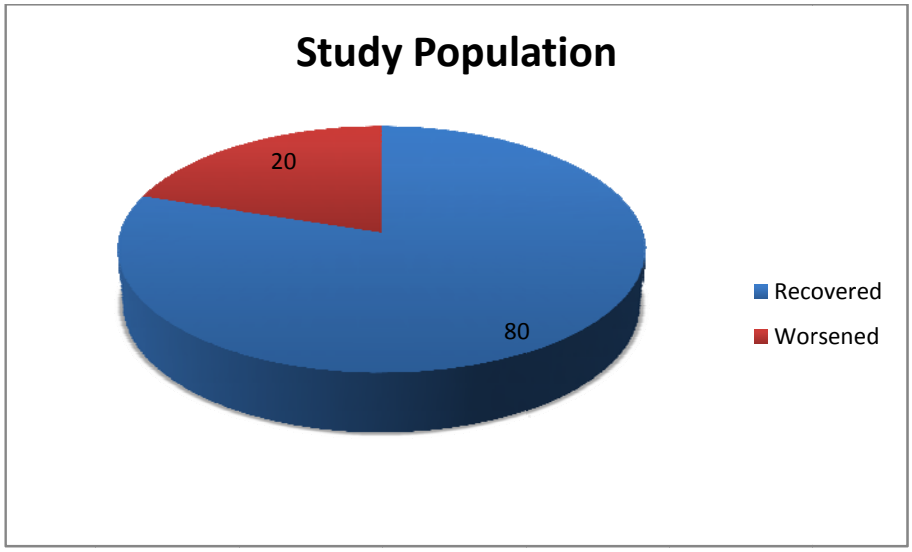
Among the studied patients, score 2 had 17 patients(34%), followed by score 1 with 15 patients(30%), score 3 with 10 patients(20%), score 5 with 5 patients(10%) and score 4 with 3 patients(6%). Out of 17 patients with score 2, 16(94.12%) recovered while 1(5.88%) worsened. Out of 15 patients with score 1, 14(93.33%) recovered while 1(6.67%) worsened. Out of 10 patients with score 3, 8(80%) recovered while 2(207%) worsened. Out of 5 patients with score 5, 1(20%) recovered while 4(80%) worsened. Out of 3 patients with score 4, 1(33.33%) recovered while 2(66.67%) worsened.

As per above data, patients having 3 or less than 3 conditions have higher chance of recovery while patients having 4 or more conditions have higher chance of worsening. Out of the worsened patients, 1 had sepsis, 1 had sepsis + urosepsis, 2 patients had 3 conditions( no. 1 had sepsis + drug induced + on hemodialysis and no. 2 had urosepsis + drug induced + on hemodialysis), 2 patients had 4 conditions (no. 1 had sepsis + urosepsis + drug induced + on hemodialysis and no. 2 had urosepsis + MODS + malignancy + on hemodialysis) and 4 patients had 5 conditions(no.1 had sepsis + urosepsis + shock + MODS + on hemodialysis, no. 2 had sepsis + urosepsis + shock + obstructive uropathy + on hemodialysis, no. 3 and no. 4 had sepsis + shock + MODS + on hemodialysis + on ventilator support.

**Table 8 Overall Outcome in AKI patients**

| Outcome | N | % |
|---|---|---|
| **Recovered** | 40 | 80.0 |
| **worsened** | 10 | 20.0 |
| **Total** | 50 | 100.0 |

Graph 8: Overall Outcomein AKI patients




Among 50 patients studied, 40 patients AKI recovered, and 10 patients AKI worsened(Table 8).

# DISCUSSION

Present study is aimed at finding out etiological pattern, clinical profile, investigation profile and clinical outcome in 50 patients of an intensive,care unit of a tertiary care hospital in which AKI was detected according to the KDIGO and RIFLE criteria.

We have compared various parameters relating to acute kidney injury with data available from other studies. Very few recently conducted studies are available regarding AKI from an Indian tertiary care centre.

In the study by Turney et al carried out across 54 centres in 23 countries, 29269 patients were screened and 1738 were found to have AKI, incidence rate of 5.7% and male to female ratio of 1.74:1.In the study by J Prakash et al carried out at Varanasi, the sample size and study time are similar to our study. They observed a low incidence rate of AKI at 3.79%. They had male predominance at 1.3:1[29]. In the study by R Bhadade et al carried out at Mumbai, they had a bigger sample size over a similar period as ours. They found a much higher incidence of 37.71%. Male to female ratio was found to be higher, 2:1 with majority patients below age group of less than 60 years[30].

In our study, patients with AKI were found to be older and having male predominance with male to female ratio of 2.84:1. Incidence rate of acute kidney injury in ICU was observed to be 6.3% patients. Maximum patients were found in age group of 61-70 years.

Symptomatology done by J Prakash et al shows dyspnea (47%), urinary symptoms such as oliguria (38%), obstetric symptoms (10%), neurological symptoms (2%).

Present study showed that fever, dyspnea and oliguria were most common presentations - fever in 26(52%) followed by dyspnea in 17 (34.00%), oliguria in 13 (26.00%), burning micturition in 12 (24.00%), vomiting in 9 (18.00%), nausea and anorexia in 8 (16.00% each), diarrhea, drug h/o in 6 (12.00%) with trauma in 3(6.00%) and other complaints in 2(4.00%).

While in the study done by J,Prakash et al, it was observed that mean haemoglobin of 10.25 ± 1.71; TC 12008.7 ± 2113.43 and Platelets 75500 ± 36400. Serum creatinine was 5.28 ± 1.42. urea was 142 ± 63.92. Serum sodium was 132.95 ± 7.82. Serum potassium was 4.66 ± 0.92. urine culture was found to be positive in 26 cases.

Our study had mean haemoglobin of 10.1± 2.51; TC 13022 ± 5197.46 and platelets 1.84 ± 1.07. Serum creatinine is 3.83 ± 3.78. urea is 99.36 ± 58. Serum sodium is 135.36 ± 8.77. Serum potassium is 4.36 ± 0.92. Urine culture was found to be positive in 14 cases. Serum chloride as 100.16 ± 8.98. Also, serum calcium is 7.98 ± 0.75; serum phosphorous as 4.81 ± 1.14 and serum uric acid as 5.52 ± 1.31.

In comparison of patients who had total leucocyte count <10000 cells/cu.mm with >10000 cells/ cu.mm, a highly significant p value of 0.000 was observed which means that patient having total leucocyte count > 10000 cells/ cu.mm has higher probability of worsening in comparison to patient having total leucocyte count >10000 cells/ cu.mm.

Among studied patients, 35 had sepsis. Out of that, 26 patients(74.28%) recovered which means that if sepsis is corrected, there is higher probability of recovery of AKI. Thus, in comparison of outcome of patients with sepsis, p value was detected to be highly significant i.e. 0.000. It means that outcome of patients in whom sepsis has

been corrected have a better chance of prognosis than those in whom it hasn't been corrected.

Study carried out by J Prakash et al, R Bhadade et al and Turney et al[29, 30] strongly asserts the role of sepsis as the primary entity for development of AKI.

In patients of sepsis, inflammatory mediators,derived from bacteria or immune cells are filtered in the glomerulus, enter the tubular space and can subsequently inflict injury by tubular cells by binding to their respective receptors. In addition, these inflammatory mediators are released from extravasated leukocytes and can also activate tubular cells from the interstitial side. The activation of cytokines,or DAMP/PAMP (damage associated molecular pattern/ pathogen associated molecular pattern) receptors may induce apoptosis or cell cycle arrest.

So, the mechanisms included are endothelial dysfunction, inflammation, coagulation disturbance, and adaptive cell responses to injury. Therefore, a key event in the early dysfunction of the kidney during sepsis is a bio-energetic stress of the tubular epithelial cells, in response to the amplified inflammatory signal that peritubular microvascular dysfunction generates. Sepsis causes a profound alteration of the macro- and microcirculation and is characterized by a decreased peripheral vascular resistance, mal-distribution of tissue blood flow, and derangement of microcirculatory perfusion.[31]

Among studied patients, 16 had shock. Out of that, 12 patients(75 %) recovered which means that if shock is corrected, there is higher probability of recovery of AKI. Thus, in comparison of outcome of patients with shock, p value was detected to be highly significant i.e. 0.000. It means that outcome of patients in whom shock has been

corrected have a better chance of prognosis than those in whom it hasn't been corrected.

Patients in whom renal,perfusion declines due to any cause, kidneys try to maintain the glomerular capillary pressure via afferent arteriolar vasodilation and efferent arteriolar vasoconstriction- the auto-regulation. So apart from causes of renal hypo-perfusion, anything that impairs renal auto-regulation will alsoresult in fall in GFR.[32]

Similarly, in comparison of patients who had serum sodium <135 mg/dL with >135 mg/dL, a highly significant p value of 0.000 was observed which means that patients having serum sodium <135 mg/dL has higher probability of worsening in comparison to patients having serum sodium >135 mg/dL. Also, among studied patients, 22 had hyponatremia. Out of that, 16 patients(72.72%) recovered which means that if underlying hyponatremia is corrected, there is higher probability of recovery of AKI. Thus, in comparison of outcome of patients with hyponatremia, p value was detected to be highly significant i.e. 0.000. It means that outcome of patients in whom hyponatremia has been corrected have a better chance of prognosis than those in whom it hasn't been corrected.

Low serum sodium level reflects hypo-osmolality state. So, hypo-osmolality,always indicates excess total body water relative to body solutes or excess water relative to solute in the extracellular fluid, as water moves freely between the intracellular and the extracellular compartments. This imbalance can be due to,solute depletion, solute dilution, or a combination of both.Under normal conditions, renal handling of water is sufficient to excrete as much as 15-20 litres of free water per day. When osmolality is reduced, there is impairment of renal excretion of free water resulting in hyponatremia and AKI.[33]

Among comparison of patients which had serum creatinine <5 mg/dl with >5 mg/dl, a highly significant p value of 0.000 was observed which indicates that patients having serum creatinine >5 mg/dl have higher probability of worsening in comparison to patients having serum creatinine <5 mg/dl.

In the study by Turney et al, in 47.5% of patients, AKI was associated with septic shock. 34% of AKI was associated with major surgery, 27% was related to cardiogenic shock, 26% was related to hypovolemia, and 19% of ARF was potentially drug-related.In study by J Prakash et al, hypotension (71.7%), sepsis (69.6%) and nephrotoxic medications (67.4%) were the major causes.In study by R Bhadade et al, causes were cardiogenic (72.2%); sepsis (69.3%); nephrotoxic medications (66.63%); hypovolemia (58.8%); tropical fever (39.9%); post operative (33.3%).

In our study, sepsis was the most common condition in patients (64%)followed by urosepsis(40%), shock (32%), MODS (30%), drug induced (16%),obstructive uropathy (8%) andmalignancy (4%). Sepsis remains a major cause of AKI. Our patients with sepsis had higher mortality than in those without the evidence of sepsis.Of the 10 patients that worsened, 8 had sepsis as primary cause of development of AKI. This observation was similar to other studies.

Another important cause of AKI is drug induced or contrast induced AKI. Normal kidney functioning depends upon adequate blood flow to the kidney. Blood flow to the kidney is a complex, tightly regulated process that relies ona number of hormones and other small molecules, such as prostaglandins. Under normal circumstances, prostaglandin E2 ($PGE_2$) produced by the kidney is necessary to support adequate blood flow to the kidney. Like all prostaglandins, $PGE_2$,synthesis depends upon the cyclooxygenases. The pathophysiology of contrast-induced

nephropathy (CIN) is based on three distinct but interacting mechanisms: medullary ischaemia, formation of reactive oxygen species and direct tubular cell toxicity.

Aspirin and other NSAIDs are inhibitors of the,cyclooxygenases. In the kidney, this inhibition results in decreased $PGE_2$ concentration causing a reduction in blood flow. Because blood flow to the kidney first reaches the renal cortex and then the renal medulla, the deeper structures of the kidney are most sensitive to decreased blood flow. Thus the innermost structures of the kidney, known as,the renal papillae, are especially dependent on prostaglandin synthesis to maintain adequate blood flow. Inhibition of cyclooxygenases therefore damages the renal papillae, increasing the risk,of renal papillary necrosis and leading to development and progression of AKI.

Patients having 3 or less MODUS score (vide page no 48 for score of AKI) have higher chance of recovery while patients having 4 or more MODUS scorehave higher chance of worsening of AKI.

Considering other parameters in the 10 patients that worsened, 9 were male and 1 was female. 3 patients had age less than 50 years, 2 patients were in 51-60 years age group and 5 patients were in 61-70 years age group. 6 patients had hyponatremia and 4 had normal sodium levels. 1 patient had hypokalemia, 1 patient had hyperkalemia and 8 had normal potassium levels. 5 patients had creatinine level more than 5 gm/dl and they also had more conditions alongside leading to higher chance of worsening of AKI.

The benefits of present study are : we are able to evaluate factors affecting AKI in detail; using MODUS criteria for AKI, we are able to classify patients on basis of underlying condition present according to which outcome of AKI could be elicited;

study parameters are cost effective. Very few studies are available regarding AKI in ICU at tertiary care hospital in an Indian setup.

The limitations of present study are: study is carried out over a small group of patients so results cannot be applied to a larger group. Another disadvantage is that only the patients admitted in medical ICU have been included.

In patients having high MODUS score for AKI alongside older age group, the higher chance they had of worsening and in patients having lower MODUS score for AKI alongside younger age group, the higher chance they had of recovery.

# SUMMARY & CONCLUSION

- Present study was undertaken to study clinical profile of AKI in ICU setup of a tertiary care centre. This was an observational pilot study. It was conducted from January 2016 to December 2016 in the Department of Medicine of SBKS MI & RC, SumandeepVidyapeeth, Piparia. The study was started after procuring approval from the institutional ethics committee.

- All patients admitted in ICU in which AKI was detected were included in the study. All included patients were evaluated for risk factors. Data was collected using a pretested proforma meeting the objectives of the study. Detailed history was taken regarding duration of illness, symptoms, comorbid conditions. All included participants were subjected to CBC, renal function test, urine routine microscopy, urine culture, ultrasonography of abdomen and pelvis, liver function test, serum calcium, serum phosphorous, serum uric acid, serum electrolytes, peripheral smear for malarial parasite and sickling solubility test. Laboratory evaluations were performed in the institutional pathology and biochemistry labs. The data so collected was then analyzed.

- Total 50 patients were included in present study which included 37 males(74%), 13 females(26%) with M:F ratio is 2.8:1 showing male predominance.

- The mean age of patients was 50.38$\pm$19.92 years. This observation indicated that acute kidney injury affected mainly middle and older age group as majority of patients were in 51-70 age group.

- Most common presenting symptoms were fever, dyspnea, oliguria, dysuria, loose stools, vomiting. Other symptoms included nausea, pain in abdomen, anorexia.

- Duration of stay in hospital was around 3 to 10 days.

- Sepsis was the most common cause followed by urosepsis, shock, MODS, drug induced , malignancy, obstructive uropathy.

- In patients having high MODUS score for AKI alongside older age group, the higher chance they had of worsening and in patients having lower MODUS score for AKI alongside younger age group, the higher chance they hadof recovery.

- Bedside monitoring of vital parameters such as temperature, pulse, blood pressure, respiratory rate, pulse oximetry and urine output monitoring correlated well with severity of illness suggesting that even simple bedside parameter monitoring can be of great help.

- Thus, from this study, we conclude that a patient has higher probability of developing AKI if:

    - Patient has sepsis

    - Patient is more than 50 years of age

    - Patients with MODUS (**MODUS** stands for M = MODS and Malignancy; O = Obstructive Uropathy; D = Drug Induced; U = Urosepsis; S = Sepsis and Shock) score $\geq$ 4 had higher chance of worsening and patients with MODUS score $\leq$ 3 had higher chance of recovery. (vide page no 48 for score of AKI)

    - Patient who had serum creatinine > 5 mg/dL and serum sodium < 135 mg/dL( p value 0.000) in comparison to patients who had serum creatinine < 5 mg/dL and serum sodium > 135 mg/dL had higher chance of worsening in comparison to the latter group.

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# ANNEXURE

**ABBREVIATIONS IN DATA**

| | |
|---|---|
| ACE | Angiotensin Converting Enzyme |
| AGE | Acute Gastro Enteritis |
| AGN | Acute Glomerulonephritis |
| AIN | Acute Interstitial Nephritis |
| AKI | Acute Kidney Injury |
| ATN | Acute Tubular Necrosis |
| BUN | Blood Urea Nitrogen |
| CCF | Congestive Cardiac Failure |
| CKD | Chronic Kidney Disease |
| CVP | Central Venous Pressure |
| CVS | Cardiovascular System |
| DIC | Disseminated Intravascular Coagulation |
| GFR | Glomerular Filtration Rate |
| HD | Hemodialysis |
| HRS | Hepato Renal Syndrome |
| HUS | Hemolytic Uremic Syndrome |
| IUD | Intra Uterine Death |
| LVF | Left Ventricular Failure |
| NSAID | Non-Steroidal Anti Inflammatory Drug |
| PD | Peritoneal Dialysis |
| PPH | Post-Partum Hemorrhage |
| RPGN | Rapidly Progressive Glomerulo Nephritis |

**ABBREVIATIONS IN MASTERCHART**

| | |
|---|---|
| DOC | Date of Collection |
| M | Male |
| F | Female |
| YRS | Years |
| Sx | Symptoms |
| H/O | History Of |
| Hb | Hemoglobin |
| TC | Total Count |
| PLT | Platelets |
| PSMP | Peripheral Smear for Malarial Parasite |
| RFT | Renal Function Test |
| LFT | Liver Function Test |
| SGPT | Serum Glutamic Pyruvic Transaminase |
| SGOT | Serum Glutamic OxaloaceticTranaminase |
| RM | Routine Microscopy |
| IO | Input Output |
| CS | Culture and Sensitivity |
| USG AP | Ultrasonography Abdomen and Pelvis |
| R | Recovered |
| W | Worsened |
| A | Albumin in urine |
| P | Pus cells in urine |

PROFORMA

| Profile | On Admission | On Discharge |
|---|---|---|
| Name | | |
| Age | | |
| Sex | | |
| IPD | | |
| Residence | | |
| DOA | | |
| DOD | | |
| Chief Complaint | | |
| • Fever | | |
| • Diarrhea/Vomiting | | |
| • Blood Loss | | |
| • Cough | | |
| • Expectoration | | |
| • Urinary Symptoms | | |
| • Gynaec Symptoms | | |
| • Jaundice | | |
| • Drug Ingestion | | |
| • h/o Recent Injury | | |
| Diagnosis | | |
| Hemogram(Hb,TC,Plt) | | |
| Urea | | |
| S. Creatinine | | |
| S. $Na^+$ | | |
| S. $K^+$ | | |

| | | |
|---|---|---|
| S. $Cl^-$ | | |
| S. $HCO_3^-$ | | |
| Urine-RM(Ab,Sug,P,E) | | |
| Urine Input/Output | | |
| S. Calcium | | |
| S. Phosphorous | | |
| S. Uric Acid | | |
| LFT | | |
| Urine-CS | | |
| USG | | |
| Sickling | | |
| MP | | |
| Cause | | |
| Outcome | | |

## Informed Consent Form (ICF) for Participants in Research Programmesinvolving studies on human beings

**TITLE**: - Clinical Profile of Acute Kidney Injury in Intensive Care Unit.

Study Number: SVU/SBKS/ /2015-____

Participants Initials: _____

Participant's Name _____

Date of Birth / Age _____ ( Years)

- I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions.

- I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.

- I understand that the investigator of this study, others working on the investigator's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records, both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I agree to this access. However, I understand that my identity will not be revealed in any information related to third party or published. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

- I agree to take part in the above study.

Signature (or thumb impression) of the participant

Legally acceptable representative:

_____

Signatory's Name:_____Date:_____

_____

Signature of the investigator: _____

_____

Date:_____

Study Investigator's Name: Dr.Smit S. Shah.

Signature of the impartial witness: _____

Date:_____

Name of the witness: _____

# PARTICIPANT INFORMATION SHEET

**TITLE**: Clinical Profile of Acute Kidney Injury in Intensive Care Unit.

**DATE:**

You are being cordially invited to participate in the above titled study. The proposed study is a cross-sectional observational study to know clinical profile of Acute Kidney Injury.

**Purpose & Nature of the study: -**It is a cross-sectional study. The purpose of the study is to see clinical profile of Acute Kidney Injury.

**Voluntary nature of participation: -** Your participation in this study is voluntary and at your free will. You can refuse to participate in the study. Moreover, you are also free to withdraw at any time without having to give a reason. Despite this, you will continue to receive your standard medical care and treatment.

- **Study methods: -** It is a cross-sectional study. In this study, patient came at Dhiraj general hospital will be selected and their numerous investigations will be done.

- **Participant's responsibility: -**
    - You will share information regarding the health problem with the investigator as required.

- You will agree to allow necessary investigations like blood, urine tests. Cost of the same will be borne by the investigator.

- **Expected adverse events, risks and solution: -**This is a cross-sectional study only. There are no adverse effects of this study.

- **Benefits of participation: -**This is a cross-sectional observational study only. Causes of AKI can be detected.

- **Confidentiality**: -
    - Your information will remain strictly confidential and will not be revealed to any third party and will not be published anywhere without your prior permission.
    - If your photographs are taken for documentation, it will be dealt with strict confidentiality. However, it will be used for scientific purpose, without your identity being revealed.

- **Investigator's Contact Information: -** Since this is a study, no additional problem expected to arise. However if you need to share any information or seek advice with regard to the study, you can contact –

  Dr.Smit S. Shah (7405310006)

  Room No. 44, NRI BLOCK,

SumandeepVidyapeeth, Piparia,

Taluka: Waghodia, District: Vadodara

- **Financial consideration**: - You will not have to bear any extra cost purely for the purpose of the study. However, if the investigator desires to carry out any additional investigation, other than the ones suggested by your treating doctor or the ones which are a part of treating protocols for your disease condition, the cost of the same will be borne by the investigator. You will not get any financial incentives for participating.

- **Protection and security**: -It is a cross-sectional study and no new drugs/procedure/technique is being tested, so this does not apply.
- **Obtaining additional information**: -If you need any additional information with regard to the study, or if you require any clarification, or in case of any doubt, you are free to ask questions to the Investigator. You will be given a copy of this participant information sheet for your information and record. If you need more information at a later date, you may call the investigator or meet him.

## પરિશિષ્ટ-૪

## સુમનદીપવિદ્યાપીઠ

પીપરીયા. તા. વાઘોડિયા, જીલ્લો વડોદરા ૩૯૧૭૬૦

## માનવીઓમાં થતા સંશોધન માટેનું ઇન્ફોર્મડ કન્સેન્ટ ફોર્મ

અભ્યાસનું શીર્ષક: -

અભ્યાસ નંબર: એસ.વી.યુ./એસ.બી.કે.એસ./ /૨૦૧૩ - _____

સહભાગીના પુરાનામનાપહેલાઅક્ષરો:_____

સહ્ભાગીનું નામ_____જન્મતારીખ/ ઉમર_____( વર્ષ)

- હું તારીખ _____ અને ઉપરના અભ્યાસનું માહિતીપત્રક વાંચી અને સમજીને અભ્યાસમાં સામેલ થવાની ખાતરી આપું છું અને મને કોઈપણ પ્રશ્નો પૂછવાની તક આપવામાં આવશે. જેની મને ખબર છે.
- મારી અભ્યાસમાં ભાગીદારી સ્વૈચ્છિક છે અને હું મારી ભાગીદારી કોઈપણ કારણ આપ્યા વગર, મારી તબીબી સારવાર ને અસરના પહોચે અથવા મારા કાનૂની અધિકારોને અસર ના થાય તેમ ગમે ત્યારે પાછી ખેચી લેવા સ્વતંત્ર છું.

- હું આ અભ્યાસના અભ્યાસકર્તા, અભ્યાસકર્તાના સહયોગી, એથીક્સકમિટી અને રેગ્યુલેટરી ઓથોરીટીસ ને મારી પરવાનગી વગર મારા સ્વાસ્થ્યનો રેકોર્ડ કે જે આ અભ્યાસ કે આભવિષ્યમાં થનારા પરીક્ષણના સંદર્ભમાં થશે તે ને જોવાની સંમતિ આપું છું. જો હું મારી સંમતિ પછી ખેચી લઉં તો પણ મારી માહિતી અભ્યાસકર્તા મેળવી શકશે.છતાં, હું સમજુ છું કે, મારી ઓળખાણ કોઈપણ સ્વરૂપમાં ત્રીજી વ્યક્તિને અપાશે નહી કે જાહેરમાં પ્રકાશિત કરવામાં નહિ આવે.
- આ અભ્યાસ પરથી જે પરિણામ કે માહિતી મળે તેની પર હું પ્રતિબંધ નહિ મુકું. તેનો ઉપયોગ ફક્ત વૈજ્ઞાનિક હેતુ માટે કરવામાં આવશે.

- હું ઉપરના અભ્યાસમાં ભાગ લેવાની સંમતિ આપું છું.

Patient સહી અથવા અંગુઠાનું નિશાન_____ અથવા

કાયદાકીય રીતે સ્વીકાર્ય પ્રતિનિધિ_____

સહીકર્તાનું નામ_____તારીખ_____

અભ્યાસકર્તાની સહી_____તારીખ_____

અભ્યાસના અભ્યાસકર્તાનું નામ_____

નિષ્પક્ષસાક્ષીની સહી_____તારીખ_____

નિષ્પક્ષસાક્ષીનું નામ_____

# ભાગીદારીમાહિતીશીટ

અભ્યાસનું શીર્ષક: - Clinical Profile of Acute Kidney Injury in Intensive Care Unit.

તારીખ:

તમને ઉપરોકત શીર્ષક અભ્યાસમાં સામેલ થવા માટે આમંત્રિત કરવામાં આવે છે. આ અભ્યાસનો મુખ્ય હેતુ ભારતીયો માં દવાનો ઉપયોગ અને દવાથી થતી વિપરિત અસરો કે જેની પૂરતી માહિતી અત્યાર સુધી ઉપલબ્ધ નથી. ,તે છે.

- અભ્યાસનો મુખ્ય હેતુ
- **ભાગીદારી ની સ્વેચ્છિક પ્રકૃતિ: -**

  આ અભ્યાસમાં તમારી ભાગીદારી સ્વેચ્છિક છે. તમે આ અભ્યાસમાં ભાગ લેવાનો ઇન્કાર કરી શકો છો. વધુમાં, તમે કોઈ કારણ આપ્યા વગર ગમે ત્યારે સંમતિ પાછી ખેચી શકો છો. આમ છતાં તમારી પ્રમાણભૂત તબીબી સારવાર ચાલુ રહેશે.

- **અભ્યાસની પદ્ધતિઓ: -**
- **સહભાગીની જવાબદારી: -**
    - તમે જરૂર પડે ત્યારે, તમે તમારા રોગ વિષેની માહિતી અભ્યાસકર્તાને આપશો.
    - તમે અભ્યાસકર્તા ને દરેક મુલાકાત માં સહકાર આપશો.
    - તમને દવાની આડઅસર થઇ હશે તો તમારે લોહી, પેશાબનું પરીક્ષણ કરવું જરૂરી થઇ શકે છે. જોકે, આ પરીક્ષણ ભાગ્યે જ જરૂર પડે છે અને તેનો સંપૂર્ણ ખર્ચો અભ્યાસકર્તા ઉપાડશે.
- **અપેક્ષિત આડ અસરો,તેના થી રહેલા જોખમો અને તેના ઉપાયો: -**

આ એક પર આધારિત અભ્યાસ છે. તમારા રોગની સારવાર કે દવાઓથી થતી આડઅસરો નો ઈલાજ તમારા ડોક્ટર જ કરશે,અભ્યાસકર્તા તમારો ઈલાજ નહિ કરે. આ અભ્યાસના કારણે તમને કોઈ આડઅસરો નું જોખમ રેહવાનું નથી.

- સહભાગી થવાના ફાયદાઓ: -તમારા રોગની ઉંડાણપૂર્વક તપાસ કરવામાં આવશે. જેનાથી તમારા રોગનું સચોટ નિદાન અને સારવાર મળશે. જેનાથી તમને યોગ્ય અને ગુણકારક સારવાર મળશે.
- જો તમને સારવાર દરમ્યાન આડઅસર થશે તો તેની પહેલી જાણકારી તમને આપવામાં આવશે કે જેથી તેના યોગ્ય સુધારાત્માક પગલા લેવામાં આવે. જેનાથી તમારે ઓછી પીડા સહન કરવી પડશે.
- તમને દવાઓથી થતી આડઅસરોની માહિતી આપવામાં આવશે કે જે થી તમે ભવિષ્યમાં એ દવાઓથી થતી આડઅસરોને રોકી શકો.

- **ગુપ્તતા: -**
    - તમારી માહિતી ચોક્કસ રીતે ગુપ્ત રાખવામાં આવશે. તમારી માહિતીની જાણ,કોઈ ત્રીજી વ્યક્તિને કે જાહેરમાં પ્રસિદ્ધ કરવામાં નહિ આવે.
    - જો માહિતીના સંગ્રહ માટે તમારા ફોટોગ્રાફ્સ લેવામાં આવશે તો તેને ગુપ્ત રીતે સાચવવામાં આવશે. પણ જો તેનો વૈજ્ઞાનિક હેતુ માટે ઉપયોગ કરવામાં આવશે તો તમારી ઓળખ જાહેર ના થાય તેવી રીતે પ્રસિદ્ધ કરવામાં આવશે.
- **અભ્યાસકર્તા નો સંપર્ક: -**

    આ એક નિરીક્ષણ પર આધારિત અભ્યાસ છે . તમને સારવાર આપતા ડોક્ટરે આપેલી દવાઓથી થતી આડઅસરો સિવાય બીજી

કોઈ સમસ્યા થવાની શક્યતા નથી. જો તમને બીજી કોઈપણ માહિતી જોઈતી હોય કે તમને કોઈ સલાહની જરૂર હોય તો તમેની ચેની વ્યક્તિનો સંપર્ક કરી શકો છો.

Dr.Smit S. Shah (7405310006)

Room No. 44, NRI BLOCK,

SumandeepVidyapeeth, Piparia,

Taluka: Waghodia, District: Vadodara

- **નાણાકીયખુલાસો: -**

  તમારે આ અભ્યાસ માટે કોઈ વધારાનો ખર્ચો ઉપાડવાનો નથી. જો તમારી સારવાર આપતા ડોકટરે કરાવેલા પરીક્ષણો સિવાય અભ્યાસકર્તા કોઈ અન્ય પરીક્ષણો કરાવવા માંગે તો તેનો સંપૂર્ણ ખર્ચો અભ્યાસકર્તા ઉપાડશે. અભ્યાસમાં સામેલ થવાના લીધે તમને કોઈ વળતર અપાશે નહી કે નાણાકીય ફાયદો મળશે નહી.

- **રક્ષણ અને સલામતી: -**

  આ એક નિરીક્ષણ પર આધારિત અભ્યાસ છે. તેમાં કોઈ નવી દવાઓ કે પ્રક્રિયાનું પરીક્ષણ કરવામાં આવવાનું નથી. જેથી આ પ્રશ્ન ઉપસ્થિત થતો નથી.

- **વધારાની જાણકારી મેળવવી: -**

  જો તમને અભ્યાસ સંદર્ભે કોઈપણ પણ વધારાની જાણકારી જોઈતી હોય અથવા તમને કોઇ સ્પષ્ટતાની જરૂર હોય તો,અથવા કોઇ શંકા હોય તો,તમે અભ્યાસકર્તાને પ્રશ્નો પૂછી શકો છો.તમને તમારી માહિતી માટે આ પત્રક ની નકલ આપવામાં આવશે. જો

ભવિષ્યમાં તમને વધારે માહિતી જોઇતી હોય,તો તમે અભ્યાસકર્તાને ફોન કરી શકો છો અથવા તેમને રૂબરૂ મળવા આવી શકો છો.

| SR. NO. | DOC | AGE(YRS) | SEX | ADDRESS | DURATION(DAY) | FEVER | DIARRHEA | VOMITING | ANOREXIA | DYSPNEA | OLIGURIA | DYSURIA | NAUSEA | PAST | PERSONAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 03-05-16 | 18 | F | MP | 3 | 1 | 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 |
| 2 | 03-05-16 | 67 | M | GUJ | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 3 | 29-04-16 | 27 | M | GUJ | 7 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 |
| 4 | 04-05-16 | 58 | M | GUJ | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 |
| 5 | 04-05-16 | 24 | F | MP | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 6 | 06-05-16 | 20 | F | GUJ | 15 | 1 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 2 |
| 7 | 09-05-16 | 56 | F | MP | 15 | 2 | 2 | 2 | 1 | 1 | 1 | 2 | 2 | 1 | 2 |
| 8 | 05-05-16 | 70 | M | GUJ | 5 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 9 | 30-04-16 | 65 | F | MP | 15 | 1 | 2 | 1 | 2 | 1 | 1 | 2 | 2 | 2 | 2 |
| 10 | 16-05-16 | 19 | M | MP | 10 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 |
| 11 | 10-05-16 | 50 | M | GUJ | 10 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 |
| 12 | 11-05-16 | 57 | M | GUJ | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 |
| 13 | 09-05-16 | 25 | M | GUJ | 2 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 14 | 11-05-16 | 65 | M | MP | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 |
| 15 | 15-05-16 | 40 | M | MP | 10 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 16 | 17-05-16 | 38 | M | GUJ | 30 | 1 | 2 | 2 | 1 | 2 | 1 | 1 | 1 | 1 | 1 |
| 17 | 16-05-16 | 65 | M | MP | 2 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 |
| 18 | 09-05-16 | 35 | M | MP | 10 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 |
| 19 | 21-05-16 | 52 | M | GUJ | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 20 | 26-05-16 | 68 | M | MP | 3 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 21 | 20-05-16 | 70 | F | GUJ | 3 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 22 | 21-05-16 | 34 | M | GUJ | 10 | 1 | 1 | 1 | 1 | 2 | 2 | 1 | 1 | 1 | 1 |
| 23 | 20-05-16 | 45 | M | GUJ | 3 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 |
| 24 | 21-05-16 | 66 | M | GUJ | 7 | 1 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 |
| 25 | 24-05-16 | 35 | M | GUJ | 3 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 26 | 25-05-16 | 35 | M | MP | 15 | 2 | 1 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 2 |
| 27 | 21-05-16 | 68 | M | GUJ | 10 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 |
| 28 | 25-05-16 | 68 | F | GUJ | 7 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 |
| 29 | 24-05-16 | 65 | M | GUJ | 15 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 1 | 2 |
| 30 | 24-05-16 | 24 | M | GUJ | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 |
| 31 | 27-05-16 | 62 | M | GUJ | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 2 |
| 32 | 27-05-16 | 70 | F | GUJ | 15 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 |
| 33 | 29-05-16 | 66 | M | GUJ | 3 | 1 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 2 |
| 34 | 26-10-16 | 30 | F | GUJ | 5 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 |
| 35 | 29-05-16 | 23 | M | MP | 10 | 1 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 2 |
| 36 | 25-10-16 | 50 | M | GUJ | 3 | 2 | 1 | 1 | 1 | 1 | 2 | 2 | 1 | 2 | 2 |
| 37 | 26-10-16 | 60 | M | GUJ | 5 | 1 | 1 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 |
| 38 | 16-12-16 | 64 | M | GUJ | 5 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 2 | 1 |
| 39 | 16-12-16 | 58 | M | GUJ | 4 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 |
| 40 | 16-12-16 | 58 | F | GUJ | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |

| SR. NO. | DOC | AGE(YRS) | SEX | ADDRESS | DURATION(DAY) | FEVER | DIARRHEA | VOMITING | ANOREXIA | DYSPNEA | OLIGURIA | DYSURIA | NAUSEA | PAST | PERSONAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 41 | 15-12-16 | 55 | M | GUJ | 5 | 1 | 2 | 1 | 1 | 1 | 2 | 1 | 1 | 1 | 2 |
| 42 | 21-12-16 | 61 | F | GUJ | 10 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 2 |
| 43 | 21-12-16 | 58 | F | GUJ | 15 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 |
| 44 | 22-12-16 | 60 | M | MP | 60 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 | 2 | 2 |
| 45 | 24-12-16 | 53 | M | GUJ | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 1 | 1 |
| 46 | 27-12-16 | 58 | F | MP | 4 | 2 | 1 | 2 | 2 | 1 | 2 | 2 | 1 | 1 | 1 |
| 47 | 29-12-16 | 50 | M | MP | 5 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 |
| 48 | 29-12-16 | 50 | M | MP | 15 | 2 | 2 | 2 | 1 | 1 | 2 | 2 | 2 | 1 | 1 |
| 49 | 31-12-16 | 60 | M | GUJ | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 |
| 50 | 31-12-16 | 46 | M | GUJ | 8 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 |

| SR. NO. | DRUG | FAMILY | HB | SCORE | TC | SCORE | PLT | SICKLING | PSMP | UREA | CREATININE | SCORE | SGPT | SGOT | BILIRUBIN | DIRECT | INDIRECT | SODIUM |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 2 | 5.8 | 1 | 7200 | 2 | 1.3 | 1 | 2 | 103 | 5.3 | 3 | 37 | 19 | 0.6 | 0.4 | 0.2 | 150 |
| 2 | 2 | 2 | 10.1 | 2 | 13900 | 3 | 2.59 | 2 | 2 | 66 | 2.9 | 2 | 25 | 14 | 0.5 | 0.2 | 0.3 | 143 |
| 3 | 2 | 2 | 5.9 | 1 | 6400 | 2 | 1.95 | 2 | 2 | 167 | 23.9 | 3 | 19 | 18 | 0.4 | 0.2 | 0.2 | 133 |
| 4 | 2 | 2 | 8.9 | 1 | 24500 | 3 | 2.28 | 2 | 2 | 92 | 2.1 | 2 | 1335 | 415 | 4.1 | 1.6 | 2.5 | 138 |
| 5 | 2 | 2 | 6.8 | 1 | 25100 | 3 | 0.4 | 2 | 2 | 46 | 2.6 | 2 | 243 | 108 | 5.5 | 0.7 | 4.8 | 140 |
| 6 | 1 | 2 | 6.8 | 1 | 5400 | 2 | 0.5 | 2 | 2 | 99 | 1.6 | 1 | 32 | 16 | 2.2 | 1 | 1.2 | 140 |
| 7 | 1 | 1 | 11.7 | 2 | 7600 | 2 | 1.54 | 2 | 2 | 55 | 1.6 | 1 | 95 | 139 | 1.7 | 1.2 | 0.5 | 140 |
| 8 | 2 | 2 | 12.1 | 2 | 5000 | 2 | 1.5 | 2 | 2 | 41 | 1.6 | 1 | 18 | 19 | 0.5 | 0.2 | 0.3 | 140 |
| 9 | 2 | 2 | 9.8 | 1 | 10600 | 3 | 2.5 | 2 | 2 | 87 | 2.7 | 2 | 24 | 24 | 0.7 | 0.3 | 4 | 140 |
| 10 | 2 | 2 | 9.6 | 1 | 13500 | 3 | 0.65 | 1 | 1 | 163 | 2.3 | 2 | 91 | 95 | 27.5 | 18.2 | 9.3 | 132 |
| 11 | 2 | 2 | 10.5 | 2 | 9400 | 2 | 1.79 | 2 | 2 | 36 | 1.9 | 1 | 41 | 27 | 0.4 | 0.2 | 0.2 | 140 |
| 12 | 2 | 2 | 14.9 | 2 | 11400 | 3 | 1.8 | 2 | 2 | 76 | 1.7 | 1 | 25 | 29 | 0.9 | 0.3 | 0.6 | 143 |
| 13 | 2 | 2 | 13.5 | 2 | 18000 | 3 | 1.32 | 2 | 2 | 46 | 1.4 | 1 | 20 | 15 | 1 | 0.4 | 0.6 | 124 |
| 14 | 2 | 2 | 5.8 | 1 | 10800 | 3 | 4.52 | 2 | 2 | 46 | 1.5 | 1 | 24 | 28 | 0.8 | 0.4 | 0.4 | 125 |
| 15 | 2 | 2 | 12.9 | 2 | 18000 | 3 | 1.71 | 2 | 2 | 69 | 1.5 | 1 | 240 | 201 | 1.3 | 0.9 | 0.6 | 131 |
| 16 | 1 | 2 | 6.7 | 1 | 16800 | 3 | 0.7 | 2 | 2 | 107 | 7 | 3 | 322 | 443 | 34.6 | 15.9 | 18.7 | 136 |
| 17 | 2 | 2 | 13.6 | 2 | 7900 | 2 | 2.75 | 2 | 2 | 43 | 1.6 | 1 | 19 | 15 | 0.4 | 0.2 | 0.2 | 137 |
| 18 | 1 | 2 | 9.9 | 1 | 8500 | 2 | 1.45 | 2 | 2 | 144 | 8.3 | 3 | 97 | 41 | 22.2 | 12.2 | 10 | 142 |
| 19 | 2 | 2 | 12 | 2 | 13000 | 3 | 0.6 | 2 | 1 | 62 | 2.2 | 2 | 74 | 54 | 1.1 | 0.5 | 0.6 | 131 |
| 20 | 2 | 2 | 14 | 2 | 11400 | 3 | 2.01 | 2 | 2 | 64 | 1.5 | 1 | 373 | 152 | 1.1 | 0.5 | 0.6 | 139 |
| 21 | 2 | 2 | 10.7 | 2 | 11100 | 3 | 1.37 | 2 | 2 | 87 | 3.8 | 3 | 40 | 15 | 0.9 | 0.3 | 0.6 | 133 |
| 22 | 1 | 2 | 4.1 | 1 | 12300 | 3 | 1 | 2 | 2 | 26 | 1.6 | 1 | 33 | 29 | 3.7 | 1.5 | 2.2 | 140 |
| 23 | 2 | 2 | 13.6 | 2 | 12100 | 3 | 2.26 | 2 | 2 | 74 | 3.3 | 2 | 90 | 82 | 1.3 | 0.6 | 0.7 | 119 |
| 24 | 1 | 2 | 9.9 | 1 | 14000 | 3 | 2.14 | 2 | 2 | 138 | 1.9 | 1 | 32 | 13 | 0.5 | 0.2 | 0.3 | 126 |
| 25 | 2 | 2 | 9.4 | 1 | 12200 | 3 | 1.5 | 2 | 2 | 98 | 11.6 | 3 | 19 | 16 | 0.5 | 0.2 | 0.3 | 132 |
| 26 | 2 | 2 | 12.4 | 2 | 19200 | 3 | 4.77 | 2 | 2 | 124 | 8.3 | 3 | 22 | 26 | 0.4 | 0.2 | 0.2 | 152 |

| SR. NO. | DRUG | FAMILY | HB | SCORE | TC | SCORE | PLT | SICKLING | PSMP | UREA | CREATININE | SCORE | SGPT | SGOT | BILIRUBIN | DIRECT | INDIRECT | SODIUM |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 27 | 2 | 1 | 9.2 | 1 | 12100 | 3 | 3.73 | 2 | 2 | 132 | 5.4 | 3 | 17 | 18 | 0.3 | 0.2 | 0.1 | 144 |
| 28 | 1 | 2 | 10.3 | 2 | 16300 | 3 | 2.43 | 2 | 2 | 95 | 2.8 | 2 | 29 | 28 | 0.4 | 0.2 | 0.2 | 130 |
| 29 | 1 | 2 | 11.8 | 2 | 11000 | 3 | 2.12 | 2 | 2 | 95 | 2.6 | 2 | 1450 | 918 | 1.2 | 0.6 | 0.6 | 131 |
| 30 | 2 | 2 | 9.5 | 1 | 10700 | 3 | 1 | 2 | 2 | 112 | 2 | 2 | 110 | 20 | 1 | 0.5 | 0.5 | 140 |
| 31 | 1 | 2 | 10.8 | 2 | 6600 | 2 | 1.1 | 2 | 2 | 34 | 1.5 | 1 | 220 | 119 | 1.2 | 0.6 | 0.6 | 134 |
| 32 | 1 | 2 | 9.6 | 1 | 18000 | 3 | 1.5 | 2 | 2 | 34 | 2.7 | 2 | 100 | 80 | 0.8 | 0.4 | 0.4 | 160 |
| 33 | 2 | 2 | 10.6 | 2 | 10600 | 3 | 0.3 | 2 | 2 | 134 | 2 | 2 | 188 | 86 | 13.2 | 8.4 | 4.8 | 128 |
| 34 | 2 | 2 | 9.8 | 1 | 17100 | 3 | 0.7 | 2 | 1 | 237 | 2.8 | 2 | 49 | 24 | 4.1 | 2 | 2.1 | 115 |
| 35 | 2 | 2 | 7.8 | 1 | 9400 | 2 | 0.8 | 2 | 2 | 206 | 7.2 | 3 | 307 | 126 | 0.4 | 0.2 | 0.2 | 135 |
| 36 | 2 | 2 | 14.4 | 2 | 9200 | 2 | 1.5 | 2 | 2 | 45 | 1.5 | 1 | 1068 | 1952 | 2.9 | 1.1 | 1.8 | 133 |
| 37 | 2 | 2 | 13.2 | 2 | 14300 | 3 | 3.4 | 2 | 2 | 35 | 1.8 | 1 | 14 | 19 | 0.6 | 0.2 | 0.4 | 130 |
| 38 | 2 | 2 | 8.5 | 1 | 30000 | 3 | 2.01 | 2 | 2 | 210 | 8.5 | 3 | 340 | 206 | 10.1 | 5.5 | 5.6 | 125 |
| 39 | 1 | 1 | 8 | 1 | 8500 | 2 | 1.68 | 2 | 2 | 95 | 6.1 | 3 | 40 | 36 | 0.4 | 0.2 | 0.2 | 135 |
| 40 | 2 | 2 | 10.3 | 2 | 16800 | 3 | 1.77 | 2 | 2 | 60 | 2.2 | 2 | 28 | 26 | 0.6 | 0.3 | 0.3 | 143 |
| 41 | 1 | 2 | 10.5 | 2 | 20200 | 3 | 3.6 | 2 | 2 | 50 | 1.6 | 1 | 40 | 30 | 0.4 | 0.2 | 0.2 | 141 |
| 42 | 1 | 1 | 9 | 1 | 9700 | 2 | 2.69 | 2 | 2 | 48 | 2.1 | 2 | 40 | 30 | 0.8 | 0.4 | 0.4 | 136 |
| 43 | 1 | 2 | 8.1 | 1 | 13900 | 3 | 1.81 | 2 | 2 | 133 | 3.6 | 3 | 30 | 28 | 0.6 | 0.3 | 0.3 | 140 |
| 44 | 2 | 2 | 10.8 | 2 | 6700 | 2 | 2.85 | 2 | 2 | 152 | 6.8 | 3 | 30 | 22 | 0.8 | 0.4 | 0.4 | 146 |
| 45 | 1 | 2 | 6.2 | 1 | 13000 | 3 | 1 | 2 | 2 | 227 | 8.2 | 3 | 560 | 480 | 18 | 9 | 9 | 126 |
| 46 | 1 | 2 | 10.4 | 2 | 18400 | 3 | 0.5 | 2 | 2 | 108 | 3.3 | 2 | 400 | 326 | 10 | 5.5 | 4.5 | 119 |
| 47 | 2 | 2 | 11.2 | 2 | 11700 | 3 | 0.8 | 2 | 2 | 111 | 1.9 | 1 | 20 | 20 | 0.6 | 0.3 | 0.3 | 121 |
| 48 | 1 | 2 | 8.3 | 1 | 18000 | 3 | 4.4 | 2 | 2 | 152 | 4.4 | 3 | 48 | 36 | 0.6 | 0.3 | 0.3 | 128 |
| 49 | 1 | 1 | 10.7 | 2 | 9900 | 2 | 1 | 2 | 2 | 44 | 1.7 | 1 | 504 | 480 | 15 | 8 | 7 | 139 |
| 50 | 2 | 2 | 12.6 | 2 | 13700 | 3 | 2.56 | 2 | 2 | 260 | 3.1 | 2 | 30 | 28 | 0.4 | 0.2 | 0.2 | 143 |

| SR. NO. | SCORE | POTASSIUM | SCORE | CHLORIDE | BICARBONATE | URINE RM | URINE IO | Urine Out Put | URINE CS | CALCIUM | PHOSPHOROUS | URIC ACID | USG AP | OUTCOME |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 3 | 2.7 | 1 | 124 | 10 | 1(A 3, P 10) | 6250 | 500 | 2 | 5.8 | 4.4 | 4.3 | 1 | 1 |
| 2 | 2 | 4.4 | 2 | 104 | 21 | 1(A 3, P 8) | 1300 | 600 | 2 | 8.5 | 2.5 | 4.9 | 2 | 1 |
| 3 | 1 | 4.3 | 2 | 101 | 20 | 1(A 3, P 8) | 2500 | 1800 | 2 | 7.5 | 2.3 | 2.1 | 1 | 1 |
| 4 | 2 | 5 | 2 | 100 | 22 | 1(A 1, P 4) | 1600 | 1000 | 1 | 8 | 4.3 | 4.4 | 2 | 1 |
| 5 | 2 | 4 | 2 | 102 | 18 | 1(A 1, P 12) | 2700 | 1600 | 1 | 7 | 4.4 | 4.5 | 1 | 1 |
| 6 | 2 | 4 | 2 | 105 | 22 | 1(A 1, P 4) | 1100 | 400 | 2 | 8.4 | 4 | 4.5 | 2 | 1 |
| 7 | 2 | 5.01 | 2 | 101 | 18 | 1(A 1, P 4) | 1000 | 700 | 2 | 9 | 6 | 4.4 | 2 | 1 |
| 8 | 2 | 3.8 | 2 | 110 | 14 | 1(A 0, P 2) | 900 | 1700 | 2 | 7.6 | 4.2 | 5.1 | 2 | 1 |
| 9 | 2 | 4.7 | 2 | 104 | 20 | 1(A 1, P 4) | 3200 | 5000 | 2 | 7 | 4.6 | 4.4 | 2 | 1 |
| 10 | 1 | 4.6 | 2 | 99 | 15 | 2(A 0, P 0) | 2200 | 1600 | 2 | 7.5 | 6.6 | 2.5 | 1 | 1 |
| 11 | 2 | 4.2 | 2 | 101 | 18 | 1(A 4, P10) | 1800 | 1000 | 2 | 8 | 6.1 | 4.8 | 1 | 1 |
| 12 | 2 | 4 | 2 | 99 | 10 | 1(A 1, P 8) | 1500 | 1600 | 2 | 7.5 | 4 | 4.3 | 1 | 2 |

| SR. NO. | SCORE | POTASSIUM | SCORE | CHLORIDE | BICARBONATE | URINE RM | URINE IO | Urine Out Put | URINE CS | CALCIUM | PHOSPHOROUS | URIC ACID | USG AP | OUTCOME |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 13 | 1 | 3.9 | 2 | 90 | 18 | 1(A 0, P 4) | 1200 | 900 | 1 | 8 | 4.1 | 4.3 | 2 | 2 |
| 14 | 1 | 4.5 | 2 | 92 | 17 | 2(A 0, P 0) | 1000 | 700 | 2 | 7 | 4.2 | 6.1 | 2 | 2 |
| 15 | 1 | 4.3 | 2 | 95 | 11 | 2(A 0, P 0) | 900 | 600 | 2 | 7 | 3.7 | 6 | 1 | 1 |
| 16 | 2 | 5.1 | 2 | 99 | 16 | 1(A 2, P 14) | 1000 | 600 | 1 | 8.7 | 7.5 | 8.4 | 1 | 2 |
| 17 | 2 | 4 | 2 | 100 | 20 | 1(A 0, P 3) | 3300 | 400 | 2 | 8.3 | 4.8 | 7.1 | 2 | 1 |
| 18 | 2 | 4.1 | 2 | 106 | 11 | 1(A 1, P 8) | 2050 | 1000 | 2 | 7.9 | 4.8 | 6.1 | 1 | 2 |
| 19 | 1 | 3.3 | 1 | 93 | 18 | 1(A 1, P 3) | 2100 | 1500 | 2 | 9.7 | 5 | 3.6 | 2 | 1 |
| 20 | 2 | 3.6 | 2 | 101 | 16 | 2(A 0, P 0) | 2000 | 700 | 2 | 7.5 | 4.3 | 5.7 | 2 | 2 |
| 21 | 1 | 3.2 | 1 | 98 | 13 | 1(A 1, P 4) | 1600 | 800 | 1 | 7.5 | 5 | 6.3 | 1 | 2 |
| 22 | 2 | 4 | 2 | 105 | 12 | 1(A 1, P 8) | 1000 | 400 | 2 | 7 | 4.8 | 6.2 | 1 | 1 |
| 23 | 1 | 3.4 | 1 | 79 | 12 | 2(A 0, P 0) | 1000 | 100 | 2 | 8.3 | 6.9 | 6.2 | 1 | 1 |
| 24 | 1 | 4 | 2 | 96 | 14 | 1(A 2, P 12) | 1800 | 600 | 1 | 8 | 5.4 | 8.9 | 1 | 1 |
| 25 | 1 | 3.3 | 1 | 98 | 12 | 1(A 3, P 6) | 500 | 30 | 2 | 8.5 | 4.8 | 6.7 | 1 | 1 |
| 26 | 3 | 2.5 | 1 | 122 | 6 | 1(A 2, P 6) | 1700 | 800 | 2 | 8.5 | 4 | 5.5 | 1 | 1 |
| 27 | 2 | 5.7 | 3 | 117 | 6 | 1(A 3, P 12) | 1200 | 1000 | 2 | 8.4 | 6.2 | 8.5 | 1 | 1 |
| 28 | 1 | 4.8 | 2 | 100 | 16 | 2(A 0, P 0) | 1050 | 1300 | 2 | 9.6 | 3 | 4.8 | 2 | 1 |
| 29 | 1 | 4.5 | 2 | 94 | 19 | 1(A 2, P 4) | 1800 | 400 | 2 | 6.6 | 4.9 | 5.2 | 1 | 2 |
| 30 | 2 | 3.6 | 2 | 107 | 20 | 1(A 0, P 6)0 | 2750 | 1600 | 2 | 8.3 | 6.2 | 6.5 | 2 | 1 |
| 31 | 1 | 3.4 | 1 | 102 | 16 | 2(A 0, P 0) | 1800 | 1500 | 2 | 8.4 | 7 | 5.7 | 2 | 1 |
| 32 | 3 | 4.4 | 2 | 127 | 17 | 1(A 1, P 12) | 1000 | 600 | 1 | 8.4 | 7.2 | 6 | 1 | 1 |
| 33 | 1 | 3.7 | 2 | 99 | 15 | 2(A 0, P 0) | 1500 | 600 | 2 | 8 | 4.5 | 6.2 | 1 | 1 |
| 34 | 1 | 4.3 | 2 | 83 | 15 | 1(A 1, P 4) | 1800 | 1000 | 1 | 8 | 4.5 | 6.3 | 1 | 1 |
| 35 | 2 | 3.8 | 2 | 108 | 12 | 2(A 0, P 0) | 1000 | 200 | 2 | 8.5 | 6.1 | 4.1 | 1 | 1 |
| 36 | 1 | 5.8 | 3 | 102 | 12 | 2(A 0, P 0) | 1200 | 800 | 2 | 8.5 | 4.3 | 6.2 | 2 | 1 |
| 37 | 1 | 3 | 1 | 100 | 13 | 2(A 0, P 0) | 1200 | 801 | 2 | 8 | 4.2 | 5.8 | 2 | 1 |
| 38 | 1 | 6.5 | 3 | 90 | 8 | 1(A 0, P 12) | 2000 | 400 | 2 | 7.5 | 6 | 6.8 | 1 | 2 |
| 39 | 2 | 6.1 | 3 | 99 | 20 | 2(A 0, P 0) | 1600 | 1200 | 2 | 8.1 | 5.6 | 6.3 | 2 | 1 |
| 40 | 2 | 4.2 | 2 | 94 | 28 | 1(A 0, P 4) | 1200 | 800 | 1 | 8.6 | 6 | 5.2 | 2 | 1 |
| 41 | 2 | 4.7 | 2 | 95 | 19 | 2(A 0, P 0) | 1500 | 1300 | 2 | 9.1 | 3.7 | 5.2 | 2 | 1 |
| 42 | 2 | 4.1 | 2 | 90 | 20 | 2(A 0, P 0) | 1000 | 600 | 2 | 7.2 | 4.5 | 6.1 | 2 | 1 |
| 43 | 2 | 7 | 3 | 104 | 14 | 1(A 0, P 12) | 1200 | 800 | 1 | 8.5 | 3.8 | 5.8 | 2 | 1 |
| 44 | 3 | 5 | 2 | 90 | 11 | 1(A 0, P 8) | 2400 | 2000 | 1 | 7.6 | 4.5 | 5.3 | 1 | 1 |
| 45 | 1 | 4.4 | 2 | 90 | 10 | 1(A 0, P 16) | 1500 | 300 | 1 | 7 | 4 | 7.5 | 1 | 2 |
| 46 | 1 | 5.4 | 2 | 96 | 16 | 1(A 0, P 4) | 1000 | 400 | 2 | 8.5 | 3.9 | 4.9 | 1 | 1 |
| 47 | 1 | 5.5 | 3 | 100 | 17 | 1(A 0, P 16) | 2000 | 1500 | 1 | 8.9 | 5 | 5.2 | 1 | 1 |
| 48 | 1 | 5.5 | 3 | 99 | 22 | 2(A 0, P 0) | 1000 | 1200 | 2 | 7.5 | 4.5 | 5.2 | 1 | 1 |
| 49 | 2 | 4.5 | 2 | 99 | 14 | 1(A 0, P 4) | 800 | 600 | 2 | 8.1 | 3.8 | 5.1 | 1 | 1 |
| 50 | 2 | 4.1 | 2 | 99 | 24 | 1(A 0, P 12) | 1000 | 600 | 1 | 8.2 | 4.5 | 5 | 1 | 1 |

**Score 1= Yes, 2= No.**

| SR. NO. | DOC | AGE(YRS) | SEX | ADDRESS | DURATION | FEVER | DIARRHEA | VOMITING |
|---|---|---|---|---|---|---|---|---|
| 1 | 5/3/2016 | 18 | F | MP | 3 | 1 | 1 | 1 |
| 2 | 5/3/2016 | 67 | M | GUJ | 1 | 1 | 2 | 2 |
| 3 | 4/29/2016 | 27 | M | GUJ | 7 | 1 | 2 | 2 |
| 4 | 5/4/2016 | 58 | M | GUJ | 2 | 2 | 2 | 2 |
| 5 | 5/4/2016 | 24 | F | MP | 2 | 2 | 2 | 2 |
| 6 | 5/6/2016 | 20 | F | GUJ | 15 | 1 | 2 | 2 |
| 7 | 5/9/2016 | 56 | F | MP | 15 | 2 | 2 | 2 |
| 8 | 5/5/2016 | 70 | M | GUJ | 5 | 2 | 2 | 2 |
| 9 | 4/30/2016 | 65 | F | MP | 15 | 1 | 2 | 1 |
| 10 | 5/16/2016 | 19 | M | MP | 10 | 1 | 2 | 2 |
| 11 | 5/10/2016 | 50 | M | GUJ | 10 | 1 | 2 | 2 |
| 12 | 5/11/2016 | 57 | M | GUJ | 2 | 2 | 2 | 2 |
| 13 | 5/9/2016 | 25 | M | GUJ | 2 | 1 | 2 | 1 |
| 14 | 5/11/2016 | 65 | M | MP | 2 | 2 | 2 | 2 |
| 15 | 5/15/2016 | 40 | M | MP | 10 | 2 | 2 | 2 |
| 16 | 5/17/2016 | 38 | M | GUJ | 30 | 1 | 2 | 2 |
| 17 | 5/16/2016 | 65 | M | MP | 2 | 1 | 2 | 2 |
| 18 | 5/9/2016 | 35 | M | MP | 10 | 2 | 2 | 2 |
| 19 | 5/21/2016 | 52 | M | GUJ | 1 | 1 | 2 | 2 |
| 20 | 5/26/2016 | 68 | M | MP | 3 | 1 | 2 | 2 |
| 21 | 5/20/2016 | 70 | F | GUJ | 3 | 1 | 1 | 1 |
| 22 | 5/21/2016 | 34 | M | GUJ | 10 | 1 | 1 | 1 |
| 23 | 5/20/2016 | 45 | M | GUJ | 3 | 1 | 2 | 2 |
| 24 | 5/21/2016 | 66 | M | GUJ | 7 | 1 | 2 | 2 |
| 25 | 5/24/2016 | 35 | M | GUJ | 3 | 1 | 2 | 2 |
| 26 | 5/25/2016 | 35 | M | MP | 15 | 2 | 1 | 1 |
| 27 | 5/21/2016 | 68 | M | GUJ | 10 | 2 | 2 | 2 |
| 28 | 5/25/2016 | 68 | F | GUJ | 7 | 2 | 2 | 2 |
| 29 | 5/24/2016 | 65 | M | GUJ | 15 | 2 | 2 | 2 |
| 30 | 5/24/2016 | 24 | M | GUJ | 2 | 1 | 2 | 2 |
| 31 | 5/27/2016 | 62 | M | GUJ | 2 | 2 | 2 | 2 |
| 32 | 5/27/2016 | 70 | F | GUJ | 15 | 1 | 2 | 2 |
| 33 | 5/29/2016 | 66 | M | GUJ | 3 | 1 | 2 | 2 |
| 34 | 10/26/2016 | 30 | F | GUJ | 5 | 1 | 2 | 2 |
| 35 | 5/29/2016 | 23 | M | MP | 10 | 1 | 2 | 2 |
| 36 | 10/25/2016 | 50 | M | GUJ | 3 | 2 | 1 | 1 |
| 37 | 10/26/2016 | 60 | M | GUJ | 5 | 1 | 1 | 1 |
| 38 | 12/16/2016 | 64 | M | GUJ | 5 | 2 | 2 | 2 |
| 39 | 12/16/2016 | 58 | M | GUJ | 4 | 2 | 2 | 2 |
| 40 | 12/16/2016 | 58 | F | GUJ | 1 | 2 | 2 | 2 |
| 41 | 12/15/2016 | 55 | M | GUJ | 5 | 1 | 2 | 1 |
| 42 | 12/21/2016 | 61 | F | GUJ | 10 | 2 | 2 | 2 |
| 43 | 12/21/2016 | 58 | F | GUJ | 15 | 2 | 2 | 2 |
| 44 | 12/22/2016 | 60 | M | MP | 60 | 2 | 2 | 2 |
| 45 | 12/24/2016 | 53 | M | GUJ | 1 | 2 | 2 | 2 |
| 46 | 12/27/2016 | 58 | F | MP | 4 | 2 | 1 | 2 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 47 | 12/29/2016 | 50 | M | MP | 5 | 1 | 2 | 2 |
| 48 | 12/29/2016 | 50 | M | MP | 15 | 2 | 2 | 2 |
| 49 | 12/31/2016 | 60 | M | GUJ | 1 | 1 | 2 | 2 |
| 50 | 12/31/2016 | 46 | M | GUJ | 8 | 2 | 2 | 2 |

| ANOREXIA | DYSPNEA | OLIGURIA | DYSURIA | NAUSEA | PAST | PERSONAL | DRUG | FAMILY |
|---|---|---|---|---|---|---|---|---|
| 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 1 | 1 | 2 | 2 | 2 | 1 | 2 | 1 | 2 |
| 1 | 1 | 1 | 2 | 2 | 1 | 2 | 1 | 1 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 |
| 1 | 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 |
| 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 1 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 2 |
| 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 1 | 2 | 2 | 1 | 2 | 1 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 1 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 2 |
| 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 1 | 2 | 2 | 1 | 2 | 1 | 2 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 1 | 2 | 2 | 2 | 1 | 2 | 1 |
| 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 |
| 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 |
| 2 | 2 | 2 | 2 | 2 | 1 | 2 | 2 | 2 |
| 2 | 1 | 2 | 1 | 2 | 2 | 2 | 1 | 2 |
| 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 |
| 2 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 |
| 2 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 |
| 1 | 1 | 2 | 2 | 1 | 2 | 2 | 2 | 2 |
| 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 2 | 1 | 1 | 2 | 1 | 2 | 2 |
| 2 | 1 | 2 | 2 | 2 | 1 | 2 | 1 | 1 |
| 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
| 1 | 1 | 2 | 1 | 1 | 1 | 2 | 1 | 2 |
| 2 | 2 | 1 | 2 | 2 | 1 | 2 | 1 | 1 |
| 2 | 2 | 2 | 2 | 2 | 1 | 2 | 1 | 2 |
| 2 | 1 | 2 | 1 | 2 | 2 | 2 | 2 | 2 |
| 2 | 2 | 1 | 2 | 2 | 1 | 1 | 1 | 2 |
| 2 | 1 | 2 | 2 | 1 | 1 | 1 | 1 | 2 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 2 | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 |
| 1 | 1 | 2 | 2 | 2 | 1 | 1 | 1 | 2 |
| 2 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 1 |
| 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |

| HB | SCORE | TC | SCORE | PLT | SICKLING | PSMP | UREA | CREATININ |
|---|---|---|---|---|---|---|---|---|
| 5.8 | 1 | 7200 | 2 | 1.3 | 1 | 2 | 103 | 5.3 |
| 10.1 | 2 | 13900 | 3 | 2.59 | 2 | 2 | 66 | 2.9 |
| 5.9 | 1 | 6400 | 2 | 1.95 | 2 | 2 | 167 | 23.9 |
| 8.9 | 1 | 24500 | 3 | 2.28 | 2 | 2 | 92 | 2.1 |
| 6.8 | 1 | 25100 | 3 | 0.4 | 2 | 2 | 46 | 2.6 |
| 6.8 | 1 | 5400 | 2 | 0.5 | 2 | 2 | 99 | 1.6 |
| 11.7 | 2 | 7600 | 2 | 1.54 | 2 | 2 | 55 | 1.6 |
| 12.1 | 2 | 5000 | 2 | 1.5 | 2 | 2 | 41 | 1.6 |
| 9.8 | 1 | 10600 | 3 | 2.5 | 2 | 2 | 87 | 2.7 |
| 9.6 | 1 | 13500 | 3 | 0.65 | 1 | 1 | 163 | 2.3 |
| 10.5 | 2 | 9400 | 2 | 1.79 | 2 | 2 | 36 | 1.9 |
| 14.9 | 2 | 11400 | 3 | 1.8 | 2 | 2 | 76 | 1.7 |
| 13.5 | 2 | 18000 | 3 | 1.32 | 2 | 2 | 46 | 1.4 |
| 5.8 | 1 | 10800 | 3 | 4.52 | 2 | 2 | 46 | 1.5 |
| 12.9 | 2 | 18000 | 3 | 1.71 | 2 | 2 | 69 | 1.5 |
| 6.7 | 1 | 16800 | 3 | 0.7 | 2 | 2 | 107 | 7 |
| 13.6 | 2 | 7900 | 2 | 2.75 | 2 | 2 | 43 | 1.6 |
| 9.9 | 1 | 8500 | 2 | 1.45 | 2 | 2 | 144 | 8.3 |
| 12 | 2 | 13000 | 3 | 0.6 | 2 | 1 | 62 | 2.2 |
| 14 | 2 | 11400 | 3 | 2.01 | 2 | 2 | 64 | 1.5 |
| 10.7 | 2 | 11100 | 3 | 1.37 | 2 | 2 | 87 | 3.8 |
| 4.1 | 1 | 12300 | 3 | 1 | 2 | 2 | 26 | 1.6 |
| 13.6 | 2 | 12100 | 3 | 2.26 | 2 | 2 | 74 | 3.3 |
| 9.9 | 1 | 14000 | 3 | 2.14 | 2 | 2 | 138 | 1.9 |
| 9.4 | 1 | 12200 | 3 | 1.5 | 2 | 2 | 98 | 11.6 |
| 12.4 | 2 | 19200 | 3 | 4.77 | 2 | 2 | 124 | 8.3 |
| 9.2 | 1 | 12100 | 3 | 3.73 | 2 | 2 | 132 | 5.4 |
| 10.3 | 2 | 16300 | 3 | 2.43 | 2 | 2 | 95 | 2.8 |
| 11.8 | 2 | 11000 | 3 | 2.12 | 2 | 2 | 95 | 2.6 |
| 9.5 | 1 | 10700 | 3 | 1 | 2 | 2 | 112 | 2 |
| 10.8 | 2 | 6600 | 2 | 1.1 | 2 | 2 | 34 | 1.5 |
| 9.6 | 1 | 18000 | 3 | 1.5 | 2 | 2 | 34 | 2.7 |
| 10.6 | 2 | 10600 | 3 | 0.3 | 2 | 2 | 134 | 2 |
| 9.8 | 1 | 17100 | 3 | 0.7 | 2 | 1 | 237 | 2.8 |
| 7.8 | 1 | 9400 | 2 | 0.8 | 2 | 2 | 206 | 7.2 |
| 14.4 | 2 | 9200 | 2 | 1.5 | 2 | 2 | 45 | 1.5 |
| 13.2 | 2 | 14300 | 3 | 3.4 | 2 | 2 | 35 | 1.8 |
| 8.5 | 1 | 30000 | 3 | 2.01 | 2 | 2 | 210 | 8.5 |
| 8 | 1 | 8500 | 2 | 1.68 | 2 | 2 | 95 | 6.1 |
| 10.3 | 2 | 16800 | 3 | 1.77 | 2 | 2 | 60 | 2.2 |
| 10.5 | 2 | 20200 | 3 | 3.6 | 2 | 2 | 50 | 1.6 |
| 9 | 1 | 9700 | 2 | 2.69 | 2 | 2 | 48 | 2.1 |
| 8.1 | 1 | 13900 | 3 | 1.81 | 2 | 2 | 133 | 3.6 |
| 10.8 | 2 | 6700 | 2 | 2.85 | 2 | 2 | 152 | 6.8 |
| 6.2 | 1 | 13000 | 3 | 1 | 2 | 2 | 227 | 8.2 |
| 10.4 | 2 | 18400 | 3 | 0.5 | 2 | 2 | 108 | 3.3 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 11.2 | 2 | 11700 | 3 | 0.8 | 2 | 2 | 111 | 1.9 |
| 8.3 | 1 | 18000 | 3 | 4.4 | 2 | 2 | 152 | 4.4 |
| 10.7 | 2 | 9900 | 2 | 1 | 2 | 2 | 44 | 1.7 |
| 12.6 | 2 | 13700 | 3 | 2.56 | 2 | 2 | 260 | 3.1 |

| SCORE | SGPT | SGOT | BILIRUBIN | DIRECT | INDIRECT | SODIUM | SCORE | POTASSIUM |
|---|---|---|---|---|---|---|---|---|
| 3 | 37 | 19 | 0.6 | 0.4 | 0.2 | 150 | 3 | 2.7 |
| 2 | 25 | 14 | 0.5 | 0.2 | 0.3 | 143 | 2 | 4.4 |
| 3 | 19 | 18 | 0.4 | 0.2 | 0.2 | 133 | 1 | 4.3 |
| 2 | 1335 | 415 | 4.1 | 1.6 | 2.5 | 138 | 2 | 5 |
| 2 | 243 | 108 | 5.5 | 0.7 | 4.8 | 140 | 2 | 4 |
| 1 | 32 | 16 | 2.2 | 1 | 1.2 | 140 | 2 | 4 |
| 1 | 95 | 139 | 1.7 | 1.2 | 0.5 | 140 | 2 | 5.01 |
| 1 | 18 | 19 | 0.5 | 0.2 | 0.3 | 140 | 2 | 3.8 |
| 2 | 24 | 24 | 0.7 | 0.3 | 4 | 140 | 2 | 4.7 |
| 2 | 91 | 95 | 27.5 | 18.2 | 9.3 | 132 | 1 | 4.6 |
| 1 | 41 | 27 | 0.4 | 0.2 | 0.2 | 140 | 2 | 4.2 |
| 1 | 25 | 29 | 0.9 | 0.3 | 0.6 | 143 | 2 | 4 |
| 1 | 20 | 15 | 1 | 0.4 | 0.6 | 124 | 1 | 3.9 |
| 1 | 24 | 28 | 0.8 | 0.4 | 0.4 | 125 | 1 | 4.5 |
| 1 | 240 | 201 | 1.3 | 0.9 | 0.6 | 131 | 1 | 4.3 |
| 3 | 322 | 443 | 34.6 | 15.9 | 18.7 | 136 | 2 | 5.1 |
| 1 | 19 | 15 | 0.4 | 0.2 | 0.2 | 137 | 2 | 4 |
| 3 | 97 | 41 | 22.2 | 12.2 | 10 | 142 | 2 | 4.1 |
| 2 | 74 | 54 | 1.1 | 0.5 | 0.6 | 131 | 1 | 3.3 |
| 1 | 373 | 152 | 1.1 | 0.5 | 0.6 | 139 | 2 | 3.6 |
| 3 | 40 | 15 | 0.9 | 0.3 | 0.6 | 133 | 1 | 3.2 |
| 1 | 33 | 29 | 3.7 | 1.5 | 2.2 | 140 | 2 | 4 |
| 2 | 90 | 82 | 1.3 | 0.6 | 0.7 | 119 | 1 | 3.4 |
| 1 | 32 | 13 | 0.5 | 0.2 | 0.3 | 126 | 1 | 4 |
| 3 | 19 | 16 | 0.5 | 0.2 | 0.3 | 132 | 1 | 3.3 |
| 3 | 22 | 26 | 0.4 | 0.2 | 0.2 | 152 | 3 | 2.5 |
| 3 | 17 | 18 | 0.3 | 0.2 | 0.1 | 144 | 2 | 5.7 |
| 2 | 29 | 28 | 0.4 | 0.2 | 0.2 | 130 | 1 | 4.8 |
| 2 | 1450 | 918 | 1.2 | 0.6 | 0.6 | 131 | 1 | 4.5 |
| 2 | 110 | 20 | 1 | 0.5 | 0.5 | 140 | 2 | 3.6 |
| 1 | 220 | 119 | 1.2 | 0.6 | 0.6 | 134 | 1 | 3.4 |
| 2 | 100 | 80 | 0.8 | 0.4 | 0.4 | 160 | 3 | 4.4 |
| 2 | 188 | 86 | 13.2 | 8.4 | 4.8 | 128 | 1 | 3.7 |
| 2 | 49 | 24 | 4.1 | 2 | 2.1 | 115 | 1 | 4.3 |
| 3 | 307 | 126 | 0.4 | 0.2 | 0.2 | 135 | 2 | 3.8 |
| 1 | 1068 | 1952 | 2.9 | 1.1 | 1.8 | 133 | 1 | 5.8 |
| 1 | 14 | 19 | 0.6 | 0.2 | 0.4 | 130 | 1 | 3 |
| 3 | 340 | 206 | 10.1 | 5.5 | 5.6 | 125 | 1 | 6.5 |
| 3 | 40 | 36 | 0.4 | 0.2 | 0.2 | 135 | 2 | 6.1 |
| 2 | 28 | 26 | 0.6 | 0.3 | 0.3 | 143 | 2 | 4.2 |
| 1 | 40 | 30 | 0.4 | 0.2 | 0.2 | 141 | 2 | 4.7 |
| 2 | 40 | 30 | 0.8 | 0.4 | 0.4 | 136 | 2 | 4.1 |
| 3 | 30 | 28 | 0.6 | 0.3 | 0.3 | 140 | 2 | 7 |
| 3 | 30 | 22 | 0.8 | 0.4 | 0.4 | 146 | 3 | 5 |
| 3 | 560 | 480 | 18 | 9 | 9 | 126 | 1 | 4.4 |
| 2 | 400 | 326 | 10 | 5.5 | 4.5 | 119 | 1 | 5.4 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 1 | 20 | 20 | 0.6 | 0.3 | 0.3 | 121 | 1 | 5.5 |
| 3 | 48 | 36 | 0.6 | 0.3 | 0.3 | 128 | 1 | 5.5 |
| 1 | 504 | 480 | 15 | 8 | 7 | 139 | 2 | 4.5 |
| 2 | 30 | 28 | 0.4 | 0.2 | 0.2 | 143 | 2 | 4.1 |

| SCORE | CHLORIDE | BICARBON/ | URINE RM | URINE IO | Utrine Out | URINE CS | CALCIUM | PHOSPHOR |
|---|---|---|---|---|---|---|---|---|
| 1 | 124 | 10 | 1(A 3, P 10) | 6250 | 500 | 2 | 5.8 | 4.4 |
| 2 | 104 | 21 | 1(A 3, P 8) | 1300 | 600 | 2 | 8.5 | 2.5 |
| 2 | 101 | 20 | 1(A 3, P 8) | 2500 | 1800 | 2 | 7.5 | 2.3 |
| 2 | 100 | 22 | 1(A 1, P 4) | 1600 | 1000 | 1 | 8 | 4.3 |
| 2 | 102 | 18 | 1(A 1, P 12) | 2700 | 1600 | 1 | 7 | 4.4 |
| 2 | 105 | 22 | 1(A 1, P 4) | 1100 | 400 | 2 | 8.4 | 4 |
| 2 | 101 | 18 | 1(A 1, P 4) | 1000 | 700 | 2 | 9 | 6 |
| 2 | 110 | 14 | 1(A 0, P 2) | 900 | 1700 | 2 | 7.6 | 4.2 |
| 2 | 104 | 20 | 1(A 1, P 4) | 3200 | 5000 | 2 | 7 | 4.6 |
| 2 | 99 | 15 | 2(A 0, P 0) | 2200 | 1600 | 2 | 7.5 | 6.6 |
| 2 | 101 | 18 | 1(A 4, P10) | 1800 | 1000 | 2 | 8 | 6.1 |
| 2 | 99 | 10 | 1(A 1, P 8) | 1500 | 1600 | 2 | 7.5 | 4 |
| 2 | 90 | 18 | 1(A 0, P 4) | 1200 | 900 | 1 | 8 | 4.1 |
| 2 | 92 | 17 | 2(A 0, P 0) | 1000 | 700 | 2 | 7 | 4.2 |
| 2 | 95 | 11 | 2(A 0, P 0) | 900 | 600 | 2 | 7 | 3.7 |
| 2 | 99 | 16 | 1(A 2, P 14) | 1000 | 600 | 1 | 8.7 | 7.5 |
| 2 | 100 | 20 | 1(A 0, P 3) | 3300 | 400 | 2 | 8.3 | 4.8 |
| 2 | 106 | 11 | 1(A 1, P 8) | 2050 | 1000 | 2 | 7.9 | 4.8 |
| 1 | 93 | 18 | 1(A 1, P 3) | 2100 | 1500 | 2 | 9.7 | 5 |
| 2 | 101 | 16 | 2(A 0, P 0) | 2000 | 700 | 2 | 7.5 | 4.3 |
| 1 | 98 | 13 | 1(A 1, P 4) | 1600 | 800 | 1 | 7.5 | 5 |
| 2 | 105 | 12 | 1(A 1, P 8) | 1000 | 400 | 2 | 7 | 4.8 |
| 1 | 79 | 12 | 2(A 0, P 0) | 1000 | 100 | 2 | 8.3 | 6.9 |
| 2 | 96 | 14 | 1(A 2, P 12) | 1800 | 600 | 1 | 8 | 5.4 |
| 1 | 98 | 12 | 1(A 3, P 6) | 500 | 30 | 2 | 8.5 | 4.8 |
| 1 | 122 | 6 | 1(A 2, P 6) | 1700 | 800 | 2 | 8.5 | 4 |
| 3 | 117 | 6 | 1(A 3, P 12) | 1200 | 1000 | 2 | 8.4 | 6.2 |
| 2 | 100 | 16 | 2(A 0, P 0) | 1050 | 1300 | 2 | 9.6 | 3 |
| 2 | 94 | 19 | 1(A 2, P 4) | 1800 | 400 | 2 | 6.6 | 4.9 |
| 2 | 107 | 20 | 1(A 0, P 6)( | 2750 | 1600 | 2 | 8.3 | 6.2 |
| 1 | 102 | 16 | 2(A 0, P 0) | 1800 | 1500 | 2 | 8.4 | 7 |
| 2 | 127 | 17 | 1(A 1, P 12) | 1000 | 600 | 1 | 8.4 | 7.2 |
| 2 | 99 | 15 | 2(A 0, P 0) | 1500 | 600 | 2 | 8 | 4.5 |
| 2 | 83 | 15 | 1(A 1, P 4) | 1800 | 1000 | 1 | 8 | 4.5 |
| 2 | 108 | 12 | 2(A 0, P 0) | 1000 | 200 | 2 | 8.5 | 6.1 |
| 3 | 102 | 12 | 2(A 0, P 0) | 1200 | 800 | 2 | 8.5 | 4.3 |
| 1 | 100 | 13 | 2(A 0, P 0) | 1200 | 801 | 2 | 8 | 4.2 |
| 3 | 90 | 8 | 1(A 0, P 12) | 2000 | 400 | 2 | 7.5 | 6 |
| 3 | 99 | 20 | 2(A 0, P 0) | 1600 | 1200 | 2 | 8.1 | 5.6 |
| 2 | 94 | 28 | 1(A 0, P 4) | 1200 | 800 | 1 | 8.6 | 6 |
| 2 | 95 | 19 | 2(A 0, P 0) | 1500 | 1300 | 2 | 9.1 | 3.7 |
| 2 | 90 | 20 | 2(A 0, P 0) | 1000 | 600 | 2 | 7.2 | 4.5 |
| 3 | 104 | 14 | 1(A 0, P 12) | 1200 | 800 | 1 | 8.5 | 3.8 |
| 2 | 90 | 11 | 1(A 0, P 8) | 2400 | 2000 | 1 | 7.6 | 4.5 |
| 2 | 90 | 10 | 1(A 0, P 16) | 1500 | 300 | 1 | 7 | 4 |
| 2 | 96 | 16 | 1(A 0, P 4) | 1000 | 400 | 2 | 8.5 | 3.9 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 3 | 100 | 17 | 1(A 0, P 16) | 2000 | 1500 | 1 | 8.9 | 5 |
| 3 | 99 | 22 | 2(A 0, P 0) | 1000 | 1200 | 2 | 7.5 | 4.5 |
| 2 | 99 | 14 | 1(A 0, P 4) | 800 | 600 | 2 | 8.1 | 3.8 |
| 2 | 99 | 24 | 1(A 0, P 12) | 1000 | 600 | 1 | 8.2 | 4.5 |

| URIC ACID | USG AP | OUTCOME |
|---|---|---|
| 4.3 | 1 | 1 |
| 4.9 | 2 | 1 |
| 2.1 | 1 | 1 |
| 4.4 | 2 | 1 |
| 4.5 | 1 | 1 |
| 4.5 | 2 | 1 |
| 4.4 | 2 | 1 |
| 5.1 | 2 | 1 |
| 4.4 | 2 | 1 |
| 2.5 | 1 | 1 |
| 4.8 | 1 | 1 |
| 4.3 | 1 | 2 |
| 4.3 | 2 | 2 |
| 6.1 | 2 | 2 |
| 6 | 1 | 1 |
| 8.4 | 1 | 2 |
| 7.1 | 2 | 1 |
| 6.1 | 1 | 2 |
| 3.6 | 2 | 1 |
| 5.7 | 2 | 2 |
| 6.3 | 1 | 2 |
| 6.2 | 1 | 1 |
| 6.2 | 1 | 1 |
| 8.9 | 1 | 1 |
| 6.7 | 1 | 1 |
| 5.5 | 1 | 1 |
| 8.5 | 1 | 1 |
| 4.8 | 2 | 1 |
| 5.2 | 1 | 2 |
| 6.5 | 2 | 1 |
| 5.7 | 2 | 1 |
| 6 | 1 | 1 |
| 6.2 | 1 | 1 |
| 6.3 | 1 | 1 |
| 4.1 | 1 | 1 |
| 6.2 | 2 | 1 |
| 5.8 | 2 | 1 |
| 6.8 | 1 | 2 |
| 6.3 | 2 | 1 |
| 5.2 | 2 | 1 |
| 5.2 | 2 | 1 |
| 6.1 | 2 | 1 |
| 5.8 | 2 | 1 |
| 5.3 | 1 | 1 |
| 7.5 | 1 | 2 |
| 4.9 | 1 | 1 |

| | | |
|---|---|---|
| 5.2 | 1 | 1 |
| 5.2 | 1 | 1 |
| 5.1 | 1 | 1 |
| 5 | 1 | 1 |