CLINICAL PROFILE OF ACUTE KIDNEY INJURY IN INTENSIVE CARE UNIT PATIENTS

By

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DISSERTATION SUBMITTED TO BLDE DEEMED UNIVERSITY, VIJAYAPURA

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

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IN

GENERAL MEDICINE

Under the guidance of

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Dr. VAITLA CHANDRAKANTH

LIST OF ABBREVATIONS

AKI- Acute Kidney Injury

ICU- Intensive Care Unit

KDIGO- Kidney Disease Improving Global Outcomes

RIFLE - Risk, Injury, Failure, Loss of kidney function, and End stage kidney disease

AKIN- Acute Kidney Injury Network

AGE- Acute Gastro Enteritis

CVA- Cerebro Vascular Accident

CKD- Chronic Kidney Disease

ATN- Acute Tubular Necrosis

NSAIDs- Nonsteroidal Anti-Inflammatory Drugs

ESRD- End Stage Renal Disease

DCLD- Decompensated Chronic Liver Disease

HRS- Hepato Renal Syndrome

HUS- Hemolytic Uremic Syndrome

IHD- Ischemic Heart Disease

AV- Arterio Venous

ADQI-Acute Disease Quality Initiative

ISN- International Society of Nephrology

NKF- National Kidney Foundation

ASN- American Society of Nephrology

SCr- Serum Creatinine

HIV-Human Immunodeficiency Virus

ARF- Acute Renal Failure

MODS- Multi Organ Dysfunction Syndrome

GFR-Glomerular Filtration Rate

BUN-Blood Urea Nitrogen

APACHE- Acute Physiology And Chronic Health Evaluation

R- Risk

I- Injury

F- Failure

USG- Ultrasound Sonography

ACEi- Angiotensin Converting Enzyme Inhibitors

ARBs- Angiotensin Receptor blockers

RRT- Renal Replacement Therapy

CRRT- Continuous Renal Replacement Therapy

FENa- Fractional Excretion of Sodium

RPGN- Rapidly Progressive Glomerulonephritis

WHO- World Health Organisation

TTP- Thrombotic Thrombocytopenic purpura

DIC- Disseminated Intravascular Coagulation

CCF- Congestive Cardiac Failure

MDRD- Modification of Diet in Renal Disease

GIT- Gastro Intestinal Tract

ANA- Anti Nuclear Antibody

ANCA- Anti Neutrophil Cytoplasmic Antibody

GBM- Glomerular Basement Membrane

ASO- Anti Streptolysin O

SIRS- Systemic Inflammatory Response Syndrome

MAP- Mean Arterial Pressure

PARP- Poly ADP Ribose Polymerase

iNOS- Inducible Nitric Oxide Synthase

ANP- Atrial Natriuretic Peptide

PD- Peritoneal Dialysis

HD- Hemodialysis

InHD- Intermittent Hemodialysis

EDD- Extended Daily Dialysis

SLED- Sustained Low Efficiency dialysis

CM- Conservative Management

ARDS- Acute Respiratory Distress Syndrome

NGAL-Neutrophil Gelatinase Associated Lipocalin

KIM- Kidney Injury Molecule

NHE 3- Sodium Hydrogen Exchanger Isoform 3

NAG- N Acetyl Glucosaminidase

SSAT- Spermidine/Spermine N1 Acetyl Transferase

CYR61- Cysteine Rich Protein 61

GRO Alpha- Growth Regulatory Protein Alpha

KC- Keratinocyte Derived Chemokine

DGF- Delayed Graft Function

IL- Inter Leukin

ELISA- Enzyme Linked Immunosorbent Assay

RFT- Renal Function Test

SOB- Shortness of Breath

RTA- Road Traffic Accident

KUB- Kidney Ureter Bladder

ABG- Arterial Blood Gas

OP- Organo Phosphorous Compound

R- Recovered

RI- Recovering

DM- Diabetes Mellitus

LD- Liver Disease

A- Anuria

DKA- Diabetic Keto Acidosis

LOC- Loss of Consciousness

CO- Cardiac Output

LL- Lower Limb

UL- Upper Limb

ABSTRACT

INTRODUCTION

- Patients in the intensive care unit (ICU) frequently experience acute kidney injury (AKI), which is a clinical problem that predicts a poor result on its own.
- AKI is one among the leading causes of ICU admissions and common contributor to in hospital morbidity and mortality rates.
- AKI is linked to prolonged hospital stays, which raises the cost of treatment.
- AKI may be treatable with early detection and treatment, but a higher rate of morbidity and mortality may result from a delayed diagnosis.

AIM

- Analysis of the clinical spectrum of AKI patients in ICU.
- Study the cause and risk factors of AKI.
- Analysis of final outcome of patients with AKI.

MATERIALS AND METHODS

The study was conducted at B.L.D. E (DEEMED TO BE UNIVERSITY), Shri B.M. Patil Medical College, Hospital & Research Centre, Vijayapura. All patients above 18 years of age, admitted in ICU diagnosed with Acute Kidney Injury using KDIGO criteria or developed Acute Kidney Injury after admission to ICU were taken for study. Chronic Kidney Disease patients were not included in the study. All patients were classified according to KDIGO criteria and the outcome was analyzed.

RESULTS

- In our study most of the cases were over the 50 years of age.
- In our study sepsis was the most common etiology, second most common being Acute Gastroenteritis. Other Etiologies include hepatorenal syndrome, cardiorenal syndrome, CVA, snake bite, post operative cases and poisoning and others.
- 64.10% patients were managed conservatively, 35.90 % required hemodialysis.
- Out of 78 patients, 50 patients survived. Mortality was seen in 28 patients.

CONCLUSION

The most frequent cause of AKI in ICU patients is sepsis. It also has a high death rate. In each case of sepsis, preventing the development of multi-organ failure is crucial. Dialysis was used to treat our patients cautiously and when necessary. Common causes of increased mortality include multi-organ dysfunction, lung and other infections, delayed diagnosis and treatment, and the frequent occurrence of comorbidities. Although there was a correlation between AKI and hospital outcomes, the prognosis of AKI severity is more affected by organ failure.

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INTRODUCTION

Acute kidney injury denotes a sudden impairment in renal function that often happens over a time period of hours to days and results in fluid, electrolyte, and acid-base homeostasis dysregulation as well as the retention of waste products of metabolism.[1,2]

In patients in intensive care unit (ICU), acute kidney injury (AKI) is a prevalent clinical problem that predicts a poor outcome on its own. According to two recent major multi-centre cohort studies, around 36% of all patients admitted to intensive care unit had AKI. Furthermore, more observational data showed that AKI is becoming more common.

AKI is a most important complication in critically ill patients admitted to ICU. AKI is now considered to occur at a substantially greater rate than anticipated previously, with more than 50% of the patients admitted to ICU getting AKI at some time during their critical illness. Mortality rate among intensive care unit patients with Acute kidney injury and multiorgan dysfunction has been identified to be greater than 50%. Patients who need renal replacement therapy (RRT) have a mortality rate of up to 80%.[3] AKI's pathophysiology is complicated by a variety of etiologies and risk factors. Increased age, the existence of heart failure, liver disease, nephrotoxic substances such as antibiotics, non-steroidal anti-inflammatory medications (NSAIDs) and

radiocontrast dyes are all risk factors. Need for artificial ventilation, infection, shock, sepsis and surgery are known risk factors for developing AKI.[4] Despite major advancements in treatments, AKI related mortality and morbidity remain high. In poor countries, the risk factors and incidence for AKI may differ from those in developed countries.[5]

Many people remain dialysis dependent or suffer from severe renal impairment, even though the majority of patients who survive an episode of AKI restore their renal function. Even individuals who have recovered their renal function completely or almost so are at a higher risk of developing chronic kidney disease (CKD), and superimposition of Acute on chronic kidney disease is linked to an accelerated rate of progression to end-stage renal disease (ESRD).

Data on overall incidence, risk factors, and outcome of the AKI could help in the development of AKI prevention and treatment techniques. Assessing the risk factors & clinical outcomes of AKI in patients hospitalized to ICU at a teaching hospital is the aim of this study.

AIMS AND OBJECTIVES

- Analysis of the clinical spectrum of AKI patients in ICU.
- Study the cause and risk factors for AKI.
- Analysis of final outcome of the patients with AKI.

REVIEW OF LITERATURE

• Bowman capsule and the link between the proximal tubules and the glomerular capillary tuft were described by William Bowman (1816–1892).

• DALAFIELD outlines the microscopic pathology of AKI in 1888.

• AKI brought on by trauma during World War I was diagnosed in 1977 as warnephritis.

• In 1941 LANDMARK ARTICLE reported by Waters and Brall at the time of World War II showed extensive tubular injury and a pigmented cast inside the tubular lumen, followed by reduced renal function in crush victims.

• In his 1951 textbook, "The Kidney Structure and Function in Health Science," HORMER W. SMITH coined the phrase "acute renal failure." [6]

• Silverstein Lee Henderson first proposed hemodialysis in 1967.

• Kraner discusses continuous AV hemodialysis in 1979.

In 2004, acute kidney injury, as defined by the RIFLE criteria, replaced the term acute renal failure. This phrase also encompasses the entire spectrum of disorders, from slight changes in kidney function to the need for renal replacement therapy.

DEFINITION AND STAGING OF AKI:

In 2004, Acute Disease Quality Initiative (ADQI) group, American Society of Nephrology (ASN), National Kidney Foundation (NKF) and International Society of Nephrology (ISN) met and proposed the term 'Acute Kidney Injury.

AKI is defined by any one of the following (KDIGO):

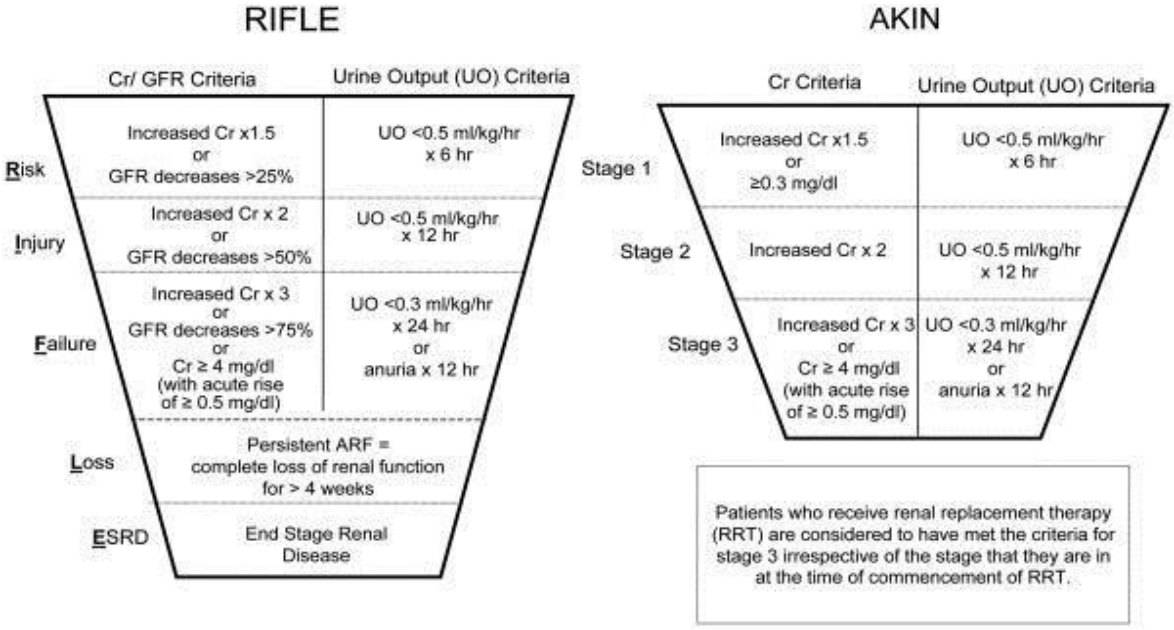
- Increase in serum creatinine by ≥ 0.3mg/dl (≥26.5µmol/l) within 48 hours.
- Increase in serum creatinine to ≥ 1.5 times of baseline value, within a period of 7 days.
- Urine output < 0.5ml/kg/hr for greater than 6 hours.

As per the KDIGO criteria, the definition and staging of acute kidney injury is done for clinical and practical purposes.

Table 1: KDIGO Criteria

AKI Stage	Serum Creatinine	Urine Output
1	1.5-1.9 times baseline Or >/=0.3mg/dl increase	<0.5ml/kg/hr for 6-12 hours
2	2.0-2.9 times baseline	<0.5 ml/kg/hr for >/=12 hours
3	3.0 times baseline Or Increase in SCr to >/=4 mg/dl Or Initiation of RRT	<0.3ml/kg/hr for >/=24 hours Or Anuria for >/= 12hours

FIGURE 1: RIFLE AND AKIN CRITERIA



EPIDEMIOLOGY

The first reported case of lethal AKI was based on military personnel who had suffered severe traumatic injuries and was licensed to Hackradh, a German pathologist, in 1917. During and after World War II, the idea of AKI in a previously healthy kidney was increasingly appreciated. Bywaters and Beall illustrated crush injury syndrome in a London victim in 1941. Acute and potentially reversible kidney damage with the histopathological characteristics of ATN was revealed in later research. Other reasons included sepsis, unmatched blood transfusions, foetal loss, circulatory collapse and different types of nephrotoxic drugs.[7]

Hantaan virus, raw fish consumption, quinine hypersensitivity, ecstasy, contracting HIV, myotoxin inhalation, gelatin infusion, and herbal medicine were among the newly discovered reasons that have been documented in recent years.[8]

Recurrence of AKI in hospitalized patients has increased to 7.2%, according to Nash et al. (2002), who used the same AKI characterization criteria at a varied institute. The rate of in-hospital death was 19.4%. Renal hypoperfusion (39%), nephrotoxic drug administration (16%), major surgery (9%), and contrast administration (16%) and were identified as the main reasons. In 1996, Liano et al. conducted research in 13 centres with 2.4 million participants. There were 200 incidences of AKI per million overall. Acute tubular necrosis 45%, Obstructive 10%, Acute on chronic kidney disease 12.7%, Prerenal 21%. Patients with AKI had a greater mortality rate (45%) than other hospitalized patients (5.4%).

AKI incidence was 5.7% in multi-centre trials involving 29,269 patients with serious illness in 54 hospitals across 23 countries, and 72.5% of these patients required RRT. 52% of patients die in intensive care units, while 8% of patients die after being released from ICU. 60.3% is the overall mortality rate. 13.8% of the survivors still needed RRT after they were discharged from the hospital.

A study carried out in intensive care unit of a referral hospital in India revealed that ARF was noted among 3.79% of cases and was associated with bad prognosis. A higher death rate among ARF patients in intensive care units was linked to the prevalence of blood infections, mods, higher APACHE-II scores,

and the need for ventilation.[10] AKI was demonstrated to be an independent risk factor for death by Ostermann, M. Chang RW: CCM 2007.

They detected the mortality rate of different classes of RIFLE Criteria as:

Class F - 57% mortality

Class I - 45% mortality

Class R - 2% mortality

When compared to 8% mortality of patients with no AKI.

The etiologies of AKI differ by location. Sepsis is the most frequent cause in our nation. Northern India has a greater incidence of AKI due to from insects.[11] Malarial AKI is prevalent in eastern India, but AKI due to Leptospira is frequently found in Chennai and Kerala.[12] In African hospitals, between 25 and 35 percent of all medical ARFs occur after using plant-based medications (Joubert and Sebata, 1982).[13] The true incidence of ARF caused by chemicals and plant-based medications is unknown in India. Information regarding the precise occurrence of ARF in various geographic locations is still sparse, even when it comes to snake bites. In other nations, the incidence ranges from 1 to 27%.

INCIDENCE

Almost 30% of ICU admissions and 5-7% of hospital admissions are complicated by AKI.[14] Clinical aspects that are frequently observed include retaining nitrogen waste products, oliguria (urine output less than 400 ml/d), which contributes to electrolyte imbalance, acid-base abnormalities, and extracellular fluid overload. Aki is typically asymptomatic and is identified on biochemical testing of the patient reveals a recent rise in serum creatinine and blood urea levels.

Three categories have been established for the diagnosis and treatment of AKI:

1. A condition when there is renal hypoperfusion, with preserving renal parenchyma(prerenal).

2. Conditions affecting renal parenchymal tissue (intrarenal).

3. Illness due to acute blockage of the urine tract (postrenal).

Although it may not always be feasible to recover to the baseline serum creatinine level following injury, AKI is frequently thought to be reversible. AKI has a considerable in-hospital morbidity and mortality rate, where later can range from 30 to 60% based on the clinical features and functional derangement of other internal organs is present or not. This is necessary to identify clinically important irreversible injury that may eventually lead to the development of CKD.[15]

PATHOPHYSIOLOGY

Although the above categorisation is very helpful for distinguishing diagnosis, many pathophysiological characteristics are shared between different types. Patients developing AKI may be with reduced or normal urine output, have a delayed or fast increase in creatinine levels and may have a difference in quality of concentration of urinary solute. The deficiency of unified clinical picture defines the varying nature of injury. Classification of Acute kidney injury as oliguric or non-oliguric depending on the daily urine output has prognostic importance. With the exception of prerenal failure, oliguria, which is defined as a daily urine output of less than 400 millilitres, has a poor prognosis. Less than 100 millilitres of urine output per day is known as anuria, and if it appears suddenly, it indicates bilateral blockage or a catastrophic injury to the two kidneys.

The stratification of kidney failure, in this sense contributes to deciding (for example, the time of beginning dialysis) and is an important criteria for the patient's response to treatment.

ETIOLOGIES

❖ PRERENAL AKI:

- **VOLUME DEPLETION:**

- **EXTRA-CELLULAR FLUID LOSS:**

  Haemorrhage

  Vomiting, diarrhoea

  Use of diuretics

  Osmotic diuresis

  Hypoadrenalism

  Nephrogenic diabetes insipidus.

- **EXTRAVASCULAR FLUID LOSS:**

  Burns

  Hypoproteinaemia.

- **REDUCED CONSUMPTION OF WATER:**

  Dehydration.

- **REDUCED CARDIAC OUTPUT:**

  Diseases of the heart (myocardial, pericardial or diseases of valves)

  Pulmonary embolism or pulmonary hypertension

  Reduced venous return.

- **SYSTEMIC VASODILATION:**

  Septic shock

  Antihypertensive drugs

  Anaphylactic reaction.

- **RENAL VASCULAR CONSTRICTION:**

  Low levels of calcium

  Raised Catecholamines

  Calcineurin inhibitor

  Amphotericin B.

- **LOSS OF AUTOREGULATION:**

  Cyclo-oxygenase Inhibitors

  ACE Inhibitors or ARBs

  HRS.

PATHOGENESIS

Early kidney function recovery occurs in prerenal AKI when normal kidney perfusion is restored. On the other hand, persistent renal hypoperfusion causes irreversible kidney damage. The Renin Angiotensin system and sympathetic system are activated with renal hypoperfusion or effective circulatory volume of arteries is decreased.

Raised angiotensin II levels causes constriction of both preglomerular & post glomerular arterioles, with predominant post glomerular vasoconstriction, which maintains normal intraglomerular capillary pressure and near normal GFR. This action is opposed by vasodilator prostaglandins.[16]

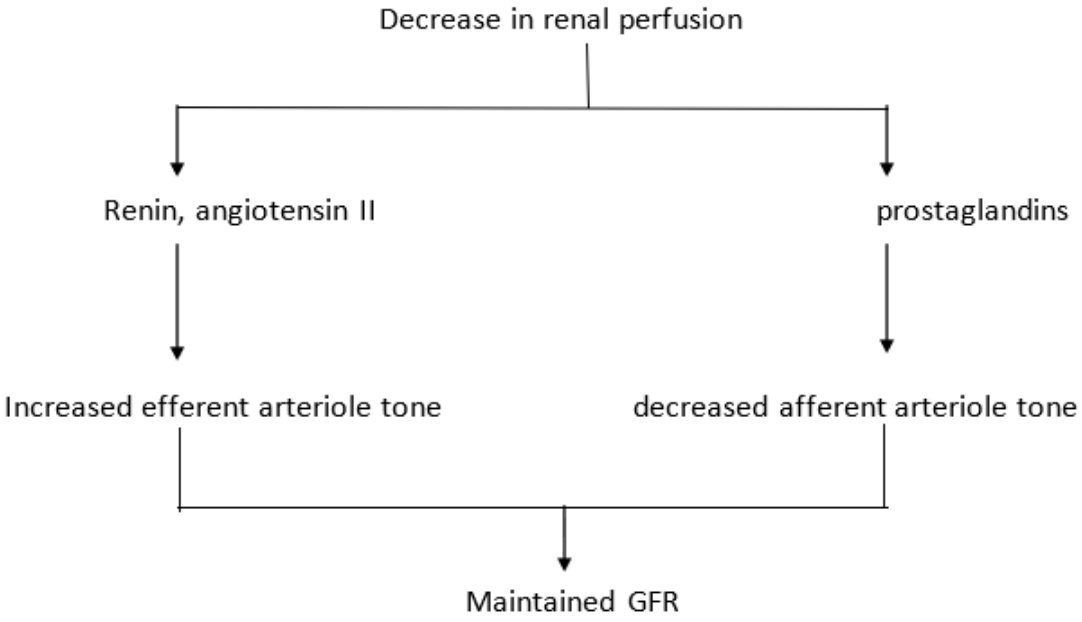
Hemodynamic factors, increasing Angiotensin II levels, activation of sympathetic system increases proximal tubular reabsorption of sodium and water. The secretion of Aldosterone & Vasopressine [17] will lead to an increase in the reabsorption of water, sodium and urea in distal tubules. Therefore, concentration of urine with a decrease in sodium levels is seen because of physiological response.

In prerenal AKI, the mechanism of regulation is unable to combat for even serious level hypoperfusion. This leads to reduction of GFR.

The kidneys first line of defence against blood pressure variations is autoregulation [18]. The afferent arteriole recognizes the extent of stretch and relaxes when renal perfusion decreases. We refer to this as the myogenic reflex.

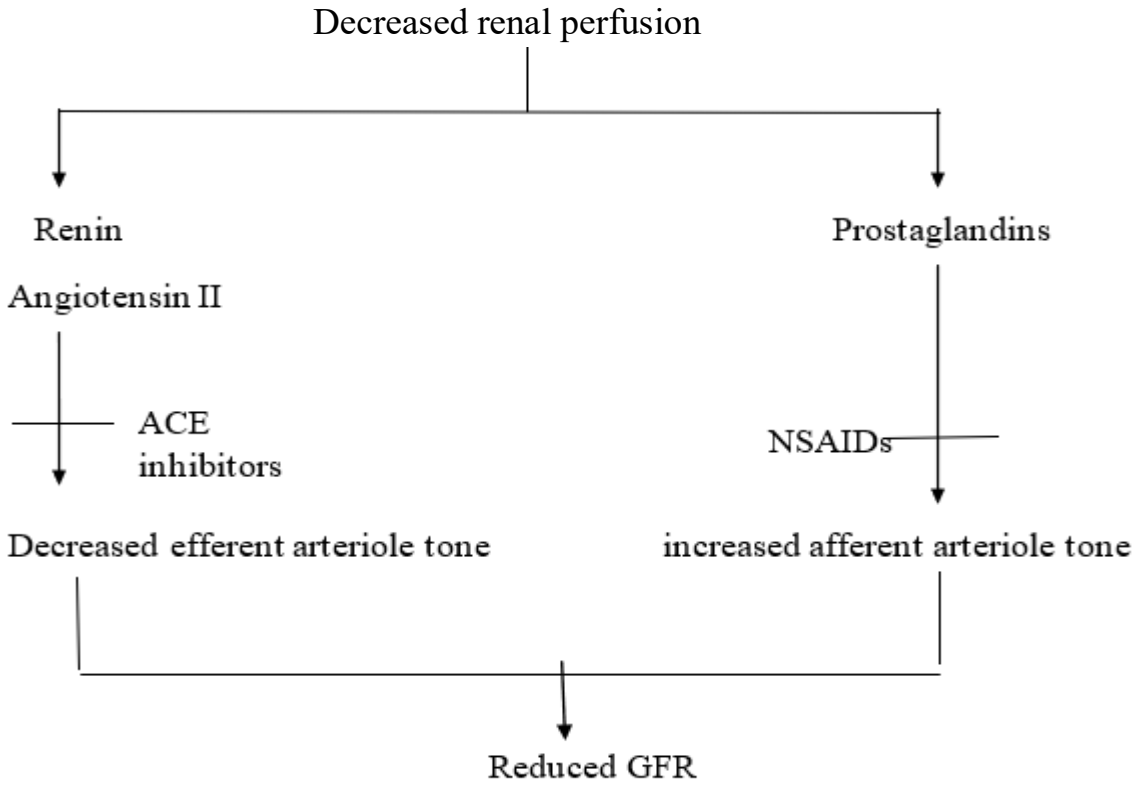
In autoregulation, tubulo-glomerular feedback plays a crucial role. In the cortical collecting duct, when macula densa detects a reduction in solute supply in the distal convoluted tubules, it releases nitric oxide, which causes the afferent arteriole to dilate.

AUTOREGULATION



After the systolic blood pressure drops below 80 mmHg, autoregulation and other natural compensating systems stop working.

DYSFUNCTION OF THE AUTOREGULATION



Clinical features include

Reduced urinary sodium concentration <20 mmol/lit

Reduced fractional excretion of sodium < 1%

Reduced fractional excretion of urea <30%

Raised urinary osmolality.

- ❖ **INTRINSIC AKI**
- ➢ **OBSTRUCTION OF RENAL VASCULATURE:**

  Renal artery Atherosclerosis

  Aneurysmal dissection

Small & large vessel vasculitis

Thromboembolism

Renal vessel obstruction due to compression.

- **DISEASES OF VASCULATURE IN GLOMERULI:**

Glomerulonephritis

vasculitis

DIC

Severe Preeclampsia

Microangiopathy

Malignant hypertension

Systemic lupus erythematosus

Scleroderma.

- **ATN:**

- Ischemia [19]

Same etiologies as for prerenal AKI, but with more severe and prolonged effect can cause ATN.

- Toxin related

EXOGENOUS - Calcineurin inhibitor

Aminoglycosides antibiotics [20,21]

Radio-contrast [22]

Cisplatin chemotherapy [23]

Amphotericin B [21]

Ethylene glycol.

ENDOGENOUS- Haemolysis

Rhabdomyolysis.

- **ACUTE INTERSTITIAL NEPHRITIS:**

Drugs: Antibiotics: Sulphonamides, Quinolones, β-lactams, Rifampicin.

Diuretics, NSAIDs.

Bilateral Pyelonephritis.

Cancers: Lymphoma, Leukemia.

Sarcoidosis.

Sjögren's syndrome, Tubulointerstitial nephritis.

Intraluminal obstruction: Intrinsic- Myeloma proteins [24]

Tumour lysis syndrome (uric acid) [25]

Systemic oxalosis.

Extrinsic-Anti viral (Acyclovir, ganciclovir, indinavir)

Methotrexate.

PATHOPHYSIOLOGY

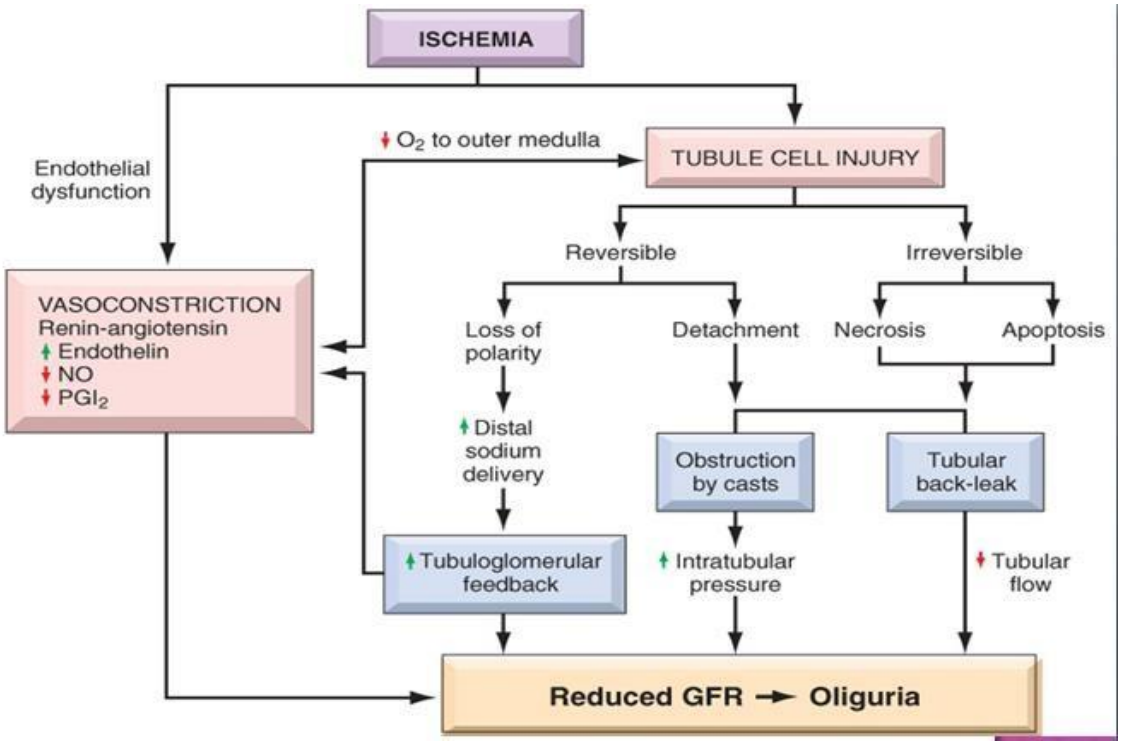
The structural lesions in the kidneys are the pathognomonic of the intrinsic AKI, and commonly seen form is acute tubular necrosis, ischemia or cytotoxic.

Mostly patchy necrosis is seen, frank necrosis is not prominent in humans. Less common injuries are injury to the brush borders, damage of the epithelium, desquamation of the cells, dilatation of lumen and intratubular casts. Even though damage is prominently seen in the proximal tubes, distal tubular damage can also be seen. In addition, desquamated cells and cell debris may block the distal tubules.

Intrinsic renal vascular constriction is predominant mechanism for the decreased GFR in patients with Acute Tubular Necrosis. The intermediate substances for this vascular constriction are not known, but tubular damage seems to be a significant concomitant observation. Intratubular obstruction due to desquamated cells and debris is the cause of decline in net ultrafiltration. The importance of this mechanism is emphasized by improving kidney function after reducing such intracellular obstruction. Furthermore, the tubulo-glomerular feedback mechanism, which is assumed to be mediated by adenosine & activated when proximal tubular injury occurs and Macula densa is exposed to an elevated chloride load, contributes to the pronounced vasoconstriction that occurs when obstruction lasts a long time.

In addition to increasing basal renal vascular tone, the stressed renal microvasculature is more sensitive to vasoconstrictive drugs and normally tolerated changes in systemic blood pressure. In the injured kidney, the vasculature has a dysregulated response to vasodilation and autoregulatory behaviour is lost. This later phenomenon is significant clinically because the

reduction of systemic pressure regularly during the intermittent hemodialysis can cause additional damage and can delay the recovery of ATN. Typically, injury leads to atubular glomeruli, in which glomerular function is preserved, but the lack of tubular flow obviates its function.



A physiological feature of ATN is not able to dilute or concentrate urine maximally (isosthenuria). This defect does not respond to pharmacological dosage of vasopressin. The injured kidney does not produce and maintain a high solute gradient in the medulla, as the accumulation of solute in the medulla depends on the normal distal tubule function. Not able to excrete concentrated urine even in the presence of Oliguria is a useful diagnostic indicator to differentiate between prerenal and intrinsic kidney disease. In prerenal azotemia,

the urinary osmolality is usually greater than 500 m Osm / kg, in intrinsic AKI, urine osmolality is less than 300 m Osm / kg.

Table 2: Nephrotoxic ATN: [26]

Mechanism	Agent
Direct tubular cell injury	Aminoglycosides, vancomycin, cisplatin, radiocontrast agents, amphotericin B, heavy metals, foscamet, cyclosporin.
Endothelial cell injury	Cyclosporin, cocaine, tacrolimus, mitomycin C, quinine.
Vasoconstriction	NSAIDs, radiocontrast agents, cyclosporin, amphotericin, heme pigments.
Efferent arteriolar vasodilation	Angiotensin-converting enzyme inhibitors.
Crystalluria	Ethylene glycol, sulphonamides, uric acid (tumour lysis syndrome), triamterene, acyclovir, methotrexate, protease inhibitors.
Interstitial nephritis	Any drug.
Glomerulopathy	Gold, penicillamine, NSAIDs.
Hemolytic uremic syndrome	Conjugated estrogens, cyclosporin, tacrolimus, mitomycin, cocaine, quinine.

- **ACUTE TUBULAR NECROSIS DUE TO SEPSIS:** [27,28]

  Systemic hypoperfusion

  Renal hypoperfusion

  Endotoxins

  Inflammatory mediator activation

  Microvascular endothelial injury.

- **ACUTE INTERSTITIAL NEPHRITIS:**

Interstitial lymphocytic infiltration. In just 10 to 30% of cases, it manifests clinically as rash, fever, eosinophilia & eosinophiluria. Antibiotics, NSAIDs, infections, cancer, and systemic diseases are the most frequent causes.

Urinary features include hematuria, pyuria, Non-nephrotic range proteinuria, WBC casts. Diagnosis is by renal histopathological examination.

- **GLOMERULONEPHRITIS:**

  AKI can be caused by glomerulonephritis and often comes under a category called Rapidly progressive glomerulonephritis (RPGN). On biopsy, glomerular crescents (an indication of glomerular injury) are

seen in RPGN, if crescents are found in greater than 50% of glomeruli, renal function typically declines significantly. Acute glomerulonephritis should be taken into account when diagnosing AKI, despite its relative rarity.

- **RADIOCONTRAST NEPHROPATHY:** [22]

Renal function rapidly deteriorates following the injection of radiocontrast. After contrast is administered, the serum creatinine starts to increase 24 to 76 hours later, peaks 3–5 days later, and then returns to baseline in 3–5 days.

- **POSTRENAL AKI:** [25]

The obstruction of the urine collecting system causes post-renal AKI.

ETIOLOGY:

- UPPER TRACT OBSTRUCTION:

- Internal: Renal calculi

  Papillary necrosis

  Blood clot

  Transitional cell carcinoma.

- External: Pelvic or retroperitoneal carcinomas

Retroperitoneal adenopathy

Retroperitoneal fibrosis

Endometritis

Aneurysm of abdominal aorta

Injury during surgery.

- LOWER TRACT OBSTRUCTION:

- Bladder: Neurogenic bladder

  Transitional cell carcinoma

  Blood clots

  Bladder calculi

  Prostate carcinoma

  Benign prostatic hyperplasia.

- Urethra: Urethral stricture or obstruction of urethral valve, Phimosis.

PATHOPHYSIOLOGY

The mechanical obstruction of the urinary collecting system, including renal pelvis, ureter, bladder or urethra, leads to obstructive uropathy or postrenal AKI. When there is unilateral obstruction, the increase in the level of creatinine in the serum may not be obvious due to contralateral kidney function. Although

the serum creatinine level may remain low with unilateral obstruction, a significant loss of GFR can be noted and in patients with partial block, progressive loss of GFR may occur if the block is not relieved. Causes of blockage include renal calculi, intramural, intraluminal or extraluminal tumor stricture. Bilateral obstruction is generally the result of enlargement of the prostate or cancers in men & gynaecological, urologic cancers in women. Patients with anuria usually have blocked at the level of bladder or downstream to bladder.

- **AKI DUE TO SNAKE BITE:**

Five to thirty percent of snake bites are complicated with acute renal failure. Viper bites account for the majority of acute kidney injury caused by snake bites. According to WHO estimates, 1,25,000 people die globally each year. It causes 10,000 deaths annually in India.

Snake bite related AKI is diagnosed with to following features.

Reduction of GFR to < 60 ml/m^2 in less than 72 hours after the snake bite. GFR was estimated with Cockcroft gaults equation. Snake bite induced AKI risk was increased with an abnormal whole blood clotting time and 1.5 times rise in aPTT, PT.

BLOOD PICTURE:

Created RBC in peripheral smears

Rise in sr. creatinine to >30% of baseline

Proteinuria

Increased D dimer

Decreased platelet count < 1 Lakh/m^3.

ALTERED HEMODYNAMICS AND HYPOTENSION:

Hemorrhage

Vasodilation

Increased vascular permeability

Direct or indirect cardiotoxicity.

NEPHROPATHY DUE TO PIGMENTS:

Myoglobinuria

Hemoglobinuria.

PATHOGENESIS OF ACUTE KIDNEY INJURY:

Nearly 100 types of proteins or peptides, lipids extracted from venom. Activating or inhibiting different blood clotting proteins, endothelial damage

with serine protease, phospholipase A2, lectins and metalloproteinases leading to coagulation disorders.

Venom contains over 100 distinct kinds of proteins, peptides, and lipids. causing coagulation issues by activating or inhibiting different blood clotting proteins, endothelium damage with serine protease, lectins, matrix metalloproteinase, and phospholipase A2.

Because haemorrhage directly damages the endothelium, it causes spontaneous bleeding. Local tissue damage and necrosis is brought on by the digestive hydrolase, hyaluronidase and polypeptide cytotoxin. Hypotension is caused by permeability factors that promote the leakage of the plasma from intravascular smooth muscle.

GUPTA et al. discovered that 15 out of 121 patients of snake bite have AKI, seven of the above 15 patients were managed conservatively, and eight required dialysis.

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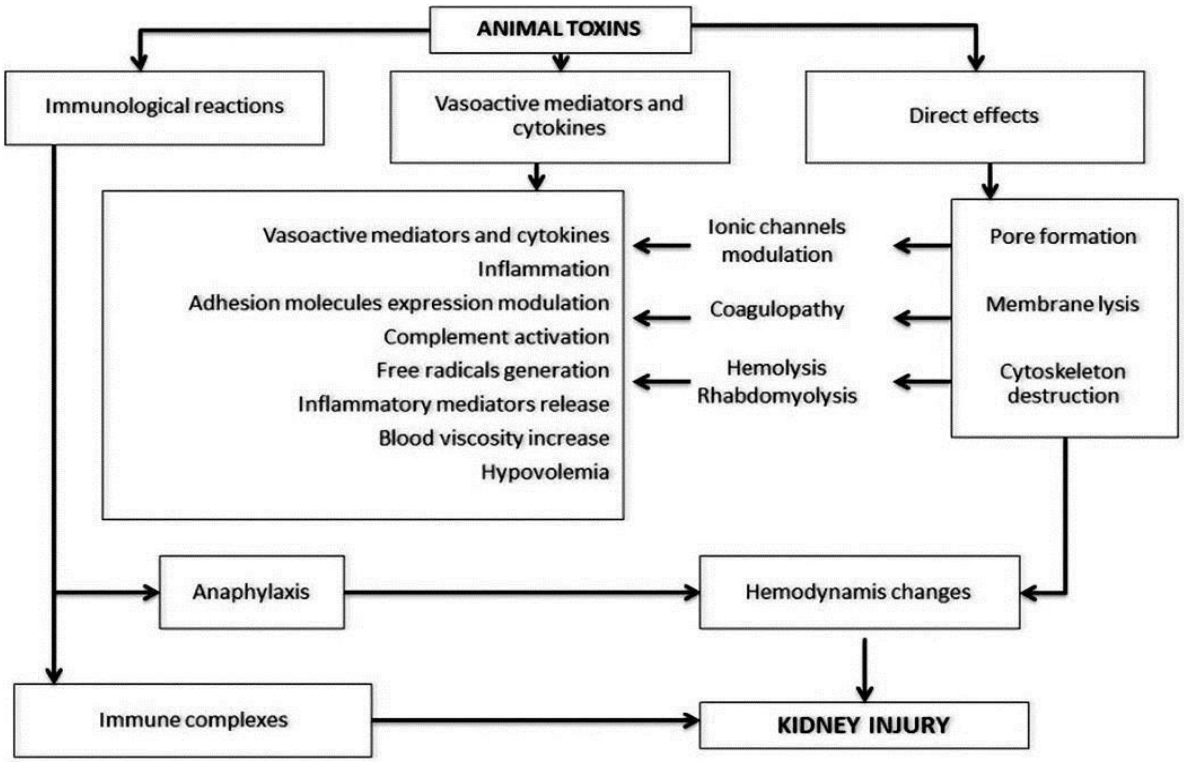

- **AKI DUE TO ACUTE GASTROENTERITIS:**

Acute gastroenteritis induced AKI is common in adults and the elderly. Five percent of cardiac output is sent to the kidneys. Reduced renal perfusion and severe vasoconstriction are caused by acute diarrhoea and electrolyte and fluid loss. Moreover, desquamation of epithelial cells cause tubular dysfunction. The most typical symptoms include hypokalemia, metabolic acidosis brought on by bicarbonate depletion, and oliguria. The histologic manifestation is of acute tubular necrosis in most the cases. There is also acute corticalnecrosis.

Late referral, severe metabolic acidosis, anuria, multiorgan failure, and metabolic encephalopathy are the main causes of AKI mortality rates.

According to Chugh et al., AGE is the primary cause of 63% of medical renal illnesses. Over the past 20 years, the incidence of post diarrheal AKI has decreased from 23% to lesser than 10% because of improved sanitation and early treatment.

- **AKI DUE TO SEPSIS:**

  Sepsis is the most common cause of AKI, it is seen in about 40% of ICU patients. AKI diagnosis is a stronger predictor of death in sepsis. Patients older than 65, those with hypertension, a higher APACHE score, severe anemia, elevated phosphate, and hyperkalemia are at higher risk for AKI and death.

  The most frequent causes of AKI during pregnancy are postpartum puerperal infections and septic abortion. Ischemia, direct inflammatory damage, reperfusion injury, coagulation, endothelial cell dysfunction, and apoptosis are the most common causes of pathogenesis.

- **DRUG INDUCED AKI**

  • Heme pigments, diuretics, aminoglycosides, and amphotericin B21 in a volume-depleted state.

  • Older individuals, congestive heart failure, ACE inhibitors, angiotensin receptor blockers, diabetes, jaundice, hypertension, NSAIDs, and radiocontrast agents if there is preexisting renal illness or bilateral renal arterial disease.

- **FEVER INDUCED AKI:** [29]

  Malaria frequently causes AKI. AKI incidence is greater than 4% and falciparum malaria mortality is greater than 45% in endemic areas.[30]

  The pathogenesis includes jaundice, disseminated intravascular coagulation, intravascular hemorrhage, hypovolemia, and compromised microcirculation caused by parasitized red blood cells.

  Panda Sk et al. discovered in a study that AKI had been linked to 20% of malaria. Within five to seven days of the fever starting, renal failure was observed. Typhoid, dengue, leptospirosis, and viral hemorrhagic fever are further causes.

DIAGNOSTIC EVALUATION OF ACUTE KIDNEY INJURY

- **History taking:**

  To diagnose the type of AKI and decide on the best course of treatment, a thorough and precise medical history is essential. Making the right diagnosis requires a thorough history, a physical examination, and standard laboratory testing.

- Although it can be challenging, it is crucial to distinguish AKI from chronic renal failure. Chronic renal failure is suggested by a history of persistent symptoms, such as pruritus, nocturia, anorexia, easy fatigability, and weight loss.

- Physical examination findings:

- Low blood pressure
- Volume depletion
- Congestive cardiac failure
- Use of Nephrotoxic drugs
- Trauma history or unaccustomed exertion
- Blood transfusions
- History of any autoimmune diseases or connective tissue disorders
- Exposure to toxins, such as ethylene glycol or ethyl alcohol
- Exposure to heavy metals like mercury, cadmium, lead, which are commonly seen with miners and welders

- The following comorbidities increase a person's risk of developing AKI:

- Hypertension
- Congestive heart failure
- Diabetes mellitus
- Infections
- Myeloproliferative disorders
- Multiple myeloma.

- The history of reduced urine output often promotes Acute kidney injury: A sudden anuria denotes obstruction, renal artery blockage due to embolism or acute & severe glomerulonephritis. A gradual decrease in

urine output may indicate urethra stricture or obstruction of the bladder outlet due to the enlargement of the prostate.

- AKI may be superimposed on chronic renal failure due to a reduction in functioning nephrons, which can happen with a minor nephrotoxic insult.

❖ **Physical examination**

Performing a comprehensive physical examination is crucial for gathering information regarding the cause of AKI.

- **Skin**

- Conditions such as purpura, petechiae, livedo reticularis, ecchymosis can reveal information about the vascular and inflammatory causes of AKI.
- Typical cutaneous changes can be caused by infectious disorders, disseminated intravascular coagulation (DIC), embolic phenomena, and thrombotic thrombocytopenic purpura (TTP).

- **Eyes**

- Interstitial nephritis and necrotizing vasculitis may be indicated by uveitis, while ethylene glycol intoxication or necrotizing vasculitis may be indicated by ocular palsy.
- A thorough examination of the eyes may reveal findings suggestive of endocarditis, atheroembolic illness, and severe hypertension.

- **Cardiovascular system**

- The assessment of cardiovascular system is the most crucial component of the physical examination.

- The physical examination must include blood pressure and pulse rate recording taken both standing & supine, a close look at the jugular vein pulse, a thorough examination of the heart, lungs, mucous membranes and skin turgor as well as a check for peripheral edema.
- Daily weight measurement and record of fluid consumption and urine output are important for hospitalized patients.
- Recordings of blood pressure can be useful diagnostic measure.
- Hypotension is seen with hypovolemia, but hypotension may not always be a sign of hypovolemia.
- Hypotension is also a possible side effect of severe congestive cardiac failure (CCF). Despite having low blood pressure, patients with CCF may have volume expansion and inadequate renal perfusion, both of which can lead to AKI.
- Severe hypertension combined with renal failure may include atheroembolic disease, glomerulonephritis, vasculitis, or renovascular disease.

➢ **Abdomen**

• When renal failure is caused by obstruction at the level of bladder outlet, which may be caused by an enlarged prostate or cancer, the results of an abdominal examination can be useful.

• Renal vascular hypertension is indicated by an epigastric bruit and may be a risk factor for AKI.

- Tight ascites may indicate high intra-abdominal pressure, which might delay renal venous return and cause AKI.

❖ **Investigations:**

AKI's etiology can be evaluated using a number of laboratory tests, and the results can help with appropriate treatment. Complete blood cell (CBC) count, serum biochemistry, urine microscopic analysis, & urinary electrolytes are some of these tests.

➢ Blood urea nitrogen (BUN) and serum creatinine

- Although elevated BUN and creatinine levels are a hallmark of renal failure, the rate of increase is dependent upon the extent of kidney damage and protein intake in relation to BUN.
- The creatinine to BUN ratio is a significant finding, because it can surpass 1:20 under circumstances that favour increased urea reabsorption (such as volume contraction), which may indicate prerenal AKI.
- Patients receiving steroid therapy, diet rich in protein, or GI or mucosal hemorrhage may have high BUN.
- If kidney functioning is absent, following formula can be used to roughly forecast a 24-hour rise in BUN:

24 hours protein intake in milligrams x 0.16 divided by the total body water in mg/dl added to the BUN value

- **CREATININE AS A MARKER FOR KIDNEY FUNCTION:**

Skeletal muscles and dietary meat are the main sources of creatinine, a metabolic product of creatine. Males produce 20–25 mg/kg of creatinine per day, while females produce 15–20 mg/kg per day.

Both proximal tubular secretion and glomerular filtration eliminate creatinine. In healthy people, glomerular filtration removes more than 90% of creatinine, with tubular secretion handling the remaining portion. However, the percentage of creatinine removed by tubular secretion rises to 50% when renal function begins to decline.[31]

Glomerular filtration rate (GFR):

The filtration rate of all functioning nephrons together is known as GFR. For men, the typical figure is approximately 125 ml/min/1.73 m2, whereas for women, it is approximately 100 ml/min/1.73 m2. GFR is calculated using the formula below:

$$GFR = U * V / P.$$

Where, U - concentration of substance in the urine

P - concentration of substance in the plasma

V - urine flow rate.

Substrate used, need to be biologically inert, that is not produced or absorbed by tubules, is free and completely filtered by glomeruli, and is not be degraded by the kidneys.

The gold standard of exogenously administered indicator for measuring GFR was inulin. However, the functionality of inulin as the marker for GFR is limited by a number of variables, including high cost, sparsity and issues with collection of urine to evaluate inulin clearance. GFR is frequently calculated using creatinine.

Estimation of creatinine clearance & GFR by creatinine based equations:

The two most used creatinine-based formulas for estimating GFR in adults are the Cockcroft-Gault formula and the modification of diet in renal disease study (MDRD) equation.[32]

COCKCROFT – GAULT EQUATION

$$\text{Est. creatinine clearance} = \frac{\text{body weight x (140-age)} \times 0.85 \text{ (if female)}}{72 \times \text{plasma creatinine}}$$

MDRD EQUATION

$$\text{Est. GFR} = 170 \times (\text{PCr})^{-0.999} \times (\text{age})^{-0.175} \times (0.762 \text{ if female}) \times (1.180 \text{ if African American}) \times (\text{BUN})^{-0.170} \times (\text{albumin})^{+0.318}$$

The Cockcroft-Gault & MDRD equations are significant because they have the potential to overstate GFR in cases of severe renal impairment and understate GFR in cases of normal renal function.

LIMITATIONS OF CREATININE AS A MARKER FOR GFR:

GFR calculations based on creatinine can overestimate renal function.

Therefore, the following restrictions should be taken into account when using creatinine to evaluate renal function.

1) A patient's body mass, race, age, and other characteristics affect creatinine production.
2) There is no linear relationship between the drop in renal function and the change in plasma creatinine.

3) The baseline creatinine value should be used to interpret an individual's creatinine variations. This requires a baseline normal value, which is typically unavailable.

4) Substances that disrupt creatinine tubular secretion can also change the amount of creatinine. For instance, medications such as trimethoprim and cimetidine can raise creatinine levels because they inhibit tubular secretion.

5) In the steady state only, creatinine serves as a helpful indicator of GFR. Abrupt changes in GFR are not associated with acute renal damage.

As a result, variations in serum creatinine levels occur several days after variations in GFR. Additionally, increase in serum creatinine levels for slight variations in GFR are not very specific or sensitive. As a result, numerous different low molecular weight blood proteins have been studied as potential endogenous GFR indicators.

- Assuming that there is no renal function, the increase in serum creatinine value can be determined using the following formulas:

For males: weight in kg X [28 – 0.2(age)] divided by total body water in mg/dL, which is added to the creatinine value.

For females: weight in kg X [23.8 – 0.17(age)] divided by total body water, which is added to the creatinine value

- As a thumb rule, if serum creatinine raises to >1.5 mg/dl/day, there is a need to rule out rhabdomyolysis.

- **CBC count, peripheral smear, and serology**

  - Schistocytes may be seen in the peripheral smear in disorders like HUS or TTP.
  - Increased rouleaux production indicates multiple myeloma, and focus of the investigations should be on urine and serum immuno-electrophoresis.
  - Etiology of AKI may be further defined by presence of hemoglobin or myoglobin, elevated serum uric acid levels, and other relevant features.
  - Glomerular disease may be included or excluded with the use of serologic tests for anti-GBM antibodies, antinuclear antibody (ANA), antistreptolysin (ASO), hepatitis, and complement level. Serological tests can be instructive, but if they are not ordered carefully, the expenses can be excessive.

- **Urine analysis**

- Granular, muddy-brown castings were discovered, which may indicate tubular necrosis.
- The diagnosis of ATN is further supported by the presence of tubular cells or tubular cell casts. Oxalate crystals are frequently seen in ATN patients.
- Reddish brown or cola coloured urine indicates the presence of hemoglobin or myoglobin, particularly when a dipstick test for heme is positive and there are no red blood cells visible under a microscope.
- Significant proteinuria detected by the dipstick assay may indicate glomerular or interstitial disease.
- Urine containing red blood cells is invariably pathogenic. RBCs that are Eumorphic indicate bleeding along the collecting system. RBC casts or dysmorphic RBCs are signs of glomerular inflammation, which implies the presence of glomerulonephritis.
- Pyelonephritis or acute interstitial nephritis may be indicated by the presence of WBCs or WBC casts. Urine eosinophils are useful in diagnosing allergic interstitial nephritis, although they are not required for the condition to exist.

Table 3: Intrinsic AKI Features

Intrinsic Renal Azotemia	Proteinuria	Sediments
Ischemic	Mild to moderate proteinuria	Pigmented granular cast
Toxins	Mild to moderate proteinuria	Pigmented granular cast
Acute Interstitial Nephritis	Mild to moderate proteinuria Haemoglobin Leukocytes	White cell and white cell cast, eosinophils and eosinophil cast Red cells

•When eosinophils are visible using Hansel stain or Wright stain, it indicates interstitial nephritis, but it can also be observed in atheroembolic illness, urinary tract infections and glomerulonephritis.

• Calcium oxalate crystals are typically seen in ethylene glycol poisoning.

- The presence of uric acid crystals may indicate ATN linked to uric acid nephropathy.

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Urine electrolytes

The most commonly utilized indicator of functioning renal tubules is the fractional excretion of sodium (FENa), which can also be obtained from urine electrolyte results. However, a mistaken diagnosis may result from the interpretation of data from individuals with glomerulonephritis, those in nonoliguric conditions, and those using or swallowing diuretics. In diseases like hepatorenal syndrome, FENa can be a useful test for identifying high renal avidity for salt. The following formula is used to get the FENa:

- FENa = (UNa/PNa) / (UCr/PCr) X 100.
- In AKI, only in the presence of oliguria, calculating the FENa is helpful.
- Prerenal azotemia cases typically have a FENa of less than 1%. The FENa is more than 1% in ATN. ATN brought on by severe burns, acute glomerulonephritis, rhabdomyolysis, and radiocontrast nephropathy are exceptions to this norm.
- FENa may be less than 1% in the presence of ATN when hepatic dysfunction is present. However, these results cannot be considered as the only signs of AKI because usage of the diuretics can lead to FENa higher than 1%.

Table 4: Urinary Diagnostic indices [33]

INDEX	PRE RENAL AKI	RENAL AKI
U_{Na} mEq/l	<10	>20
Urinary Osmolality (mOsmol/kgH_2O)	>500	<250
Urine to plasma urea nitrogen	>8	<3
U_{cr} to P_{cr}	>40	<20
Renal failure index U_{Na} / U_{cr}/P_{cr}	<1	>1
Fractional excretion of Na $\frac{U_{Na} \times P_{cr} \times 100}{P_{Na} \times U_{cr}}$	<1	>1
URINE specific gravity	>1.020	1.010
Plasma BUN / creatinine ratio	>20	<10-15

The most sensitive index is FENa. However, if a patient has prior chronic kidney disease worsened by salt wasting, adrenal insufficiency, bicarbonaturia (accompanied by sodium to maintain electroneutrality), or is taking diuretics, FENa may be greater than 1.0% in prerenal AKI. On the other hand, FENa is frequently <1.0 in AKI because of glomerulonephritis, urinary tract blockage, & vascular dysfunction, and it is <1.0% in around 15% of patients with nonoliguric nephrotoxic or ischemic AKI.[29]

- Since diuretics have no effect on urea transport, a fractional excretion of urea (FEUrea) can be achieved in individuals taking them. The following formula is used to determine the FEUrea:

  FEUrea = (Uurea/Purea) / (UCr/PCr) X 100.
- FEUrea of < 35% is indicative of a prerenal state.

➢ Bladder pressure: Intra-abdominal pressure lesser than 10 mm Hg is taken as normal and indicates that AKI is not caused by abdominal compartment syndrome. Abdominal compartment syndrome is a risk factor for patients with intra-abdominal pressures less than 15–25 mm Hg, and AKI should be suspected in patients with bladder pressures greater than 25 mm Hg.

➢ Emerging biomarkers: Some biological markers are being researched to predict and stratify AKI in individuals who are at risk. The explanation is because a high level of creatinine indicates a significant drop in GFR and is a late indicator of renal damage. Urinary Neutrophil Gelatinase-Associated lipocalin (NGAL) is the most promising biomarker to date, it

has been demonstrated to predict Acute Kidney Injury in children having cardiopulmonary bypass surgery.

❖ **Imaging Studies**

Renal imaging can be helpful in certain situations, particularly when obstruction is the cause of renal failure. The American College of Radiology states that the best imaging technique for AKI is ultrasonography, ideally using Doppler techniques.[34]

➢ **Ultrasonography**

- To evaluate current renal illnesses and obstructions of the urine collecting system, kidney ultrasonography is a highly helpful tool. The degree of blockage need not always correlate with the degree of hydronephrosis. If detected early, minimal hydronephrosis can be seen with total occlusion.
- Obese patients or those with abdominal distension from ascites, gas, or retroperitoneal fluid accumulation may have technical challenges when trying to obtain kidney pictures.
- Imaging examinations such as ultrasonography scans that reveal small kidneys may indicate chronic kidney disease.

➢ **Doppler ultrasonography**

- Doppler scan is helpful in identifying the type & presence of the renal vascular flow.
- Test results are not very helpful in diagnosing AKI because renal vascular flow is decreased in prerenal and intrinsic AKI.

- Doppler scans are a valuable tool for diagnosing renovascular or thromboembolic disorders.
- Patients with hepatorenal syndrome show increased resistance indices.

➢ **Nuclear scans**

- Renal blood flow and tubular functioning can be evaluated by radionucleotide scans using technetium-99m Tc-diethylenetriamine Penta acetic acid (99m Tc-DTPA), 99m mercaptoacetyltriglycine (99m Tc-MAG3) or iodine-131 (131 I)–Hippurate.
- The usefulness of these scans is restricted due to a noticeable lag in radionuclide tubular excretion in prerenal and intrarenal diseases.
- In certain situations of necrotizing vasculitis (e.g., polyarteritis nodosa), atherosclerosis with aortorenal obstruction, renal artery stenosis, and renal atheroembolic diseases, aorto-renal angiography may be useful in establishing the diagnosis of renal vascular disorders.

❖ **Procedures**

➢ **Renal biopsy**

- A renal biopsy can be helpful in confirming the diagnosis of intrinsic causes of AKI and can be justified if it will change management (eg, initiation of immunosuppressive medications). When renal functioning does not improve for an extended length of time and a prognosis is needed to create long-term care, a renal biopsy can also be necessary.

- The results of a kidney biopsy may show an unexpected diagnosis in up to 40% of instances.
- Only by conducting a renal biopsy may acute humoral or cellular rejection can be conclusively identified in a kidney transplanted patient.

❖ **Approach to AKI**

- Prior to starting treatment, it's critical to determine whether the AKI is actually acute or a symptom of CKD, whether the AKI is prerenal, intrinsic, or postrenal, whether the kidneys are small and contracted on ultrasonography, whether the kidneys have broad casts larger than two to three white blood cells in diameter, and whether the AKI is caused by diabetes mellitus, hypertension, glomerulonephritis, or kidney disease.
- Low levels of carbamylated hemoglobin suggest presence of chronic kidney disease
- Optimizing fluid balance, addressing underlying causes, and starting RRT at the ideal time are the top priorities for treating AKI.

➢ **Non Dialytic Therapy**

In the treatment of AKI, Correction of metabolic disturbances and electrolyte imbalance with crystalloids or colloids are examples of non-dialytic treatments. Although pharmacological treatments using dopamine, fenoldopam, loop diuretics, insulin-like growth factor-1, thyroxin, and atrial natriuretic peptide have demonstrated encouraging outcomes in animal trials, they did not significantly affect human research. The interval

between the occurrence of AKI and intervention can be associated with poor outcomes. Frusemide provides no clinical benefit in the prevention or treatment of established AKI, according to the findings of the metaanalysis of the function of loop diuretics. Its use may make ototoxicity more likely.[35]

Dopamine use is linked to compromised splanchnic perfusion, which raises the risk of gram negative bacterial infections and increases the incidence of arrhythmia, in particularly of atrial fibrillation, in patients recovering from open heart surgery. Consequently, dopamine has no part in the management of AKI.[36]
In addition to continuously monitoring the states of volume, renal biomarkers, and electrolytes, medical therapy involves strict blood sugar control. When ARF is caused by SIRS in intensive care unit, protein kinase C is a useful agent. Adequate supportive measures, maintaining renal perfusion pressure (MAP> 80 mm Hg), preventing further nephrotoxin damage, & administering RRT continue to be the cornerstones of ARF management.

Antiapoptotic & antinecrotic drugs (caspase inhibitor, which are both selective and nonselective against Caspases 1, 3, and 7, minocycline, PARP inhibitors), anti-inflammatory drugs (IL-10, iNOS inhibitor, activated protein kinase C), antiseptic drugs (insulin, activated protein kinase C), vasodilators (Endothelin antagonists, ANP) and growth factors (recombinant erythropoietin, hepatocyte growth factor) are emerging pharmacological treatments for AKI.

- **Dialytic therapy**

One of the key therapeutic modalities for AKI is dialysis. In cases of severe electrolyte imbalance (sodium, potassium), acid-base imbalance, volume overload, profound azotemia (BUN greater than 100 mg/dl), and complex uremia features (encephalopathy, pericarditis, hemorrhage, nausea-vomiting), renal replacement therapy (RRT) should be initiated. Continuous renal replacement treatment (CRRT), peritoneal dialysis (PD), extended daily dialysis (EDD), intermittent hemodialysis (InHD), and sustained low efficiency dialysis (SLED) are the choices available. Due to issues like difficulties in managing ultra filtration and inadequate delivery of dosage of dialysis, PD is not given as much priority. However, some research has indicated that continuous peritoneal dialysis has produced better outcomes in terms of lowering ICU morbidity and mortality.

Numerous controlled trials have demonstrated the benefits of InHD, despite the fact that it can exacerbate renal impairment by reducing blood pressure. By lowering complement activation and the synthesis of leukotrienes and other cytokines, the use of membranes with greater biological compatibility, such as polysulfones or polyacrylonitrile, improves the results of AKI.

With AKI better characterized, in a known at-risk category, new biomarkers being developed to detect AKI early, and InHD proving to be a successful dialysis modality, AKI is expected to become a less morbid condition in near future. However, CRRT is the recommended dialysis method for patients with hypovolemia, hypotension, and multiorgan dysfunction syndrome (MODS).

❖ Management of AKI

The symptoms of AKI are not exclusive to the kidneys; it is a morbid illness. It is a multisystem illness and an inflammatory state. The uremic condition mediates the systemic symptoms of AKI, which include metabolic acidosis, endocrine abnormalities (insulin resistance, hyperparathyroidism), and metabolic dysregulation (carbohydrate, lipid, amino acid, and protein metabolism). Through the production of humoral factors, immunocompetent cell activation, and decreased metabolism of cytokines (IL6, IL8, and IL10), the injured kidney induces a proinflammatory state that leads to distal organ damage. Thus, in sepsis-induced AKI, there is a rise in noradrenaline, Angiotensin II, Platelet activating factor, Endothelin, toll-like receptor, tumour necrosis factor, apoptosis, as well as activation of the coagulation cascade.

Long-term effects of AKI are not benign. Chronic renal disease that progresses to end-stage kidney disease is increasingly being linked to AKI. In a clinical study 174 of the 245 kids who had AKI treatment made it out alive. During a 3 to 5years follow-up, 16.6% of the survivors had chronic renal disease.[38] Early detection of AKI, aggressive management of the identified cause, administering Renal replacement therapy when needed, and measures for prevention of AKI in high risk individuals are all important components of management methods, because the morbidity and mortality of AKI are strongly correlated with the timing of its

recognition & intervention. Reversal to normal will be ensured by early detection of AKI and the implementation of remedial measures.

- **Prevention of AKI**

Preventing the AKI is the best way to manage it. Older adults, diabetics (particularly in patients who are not controlled), hypertension, hyperuricemia, preexisting kidney disease, dyslipidaemia heart failure, multiple myeloma, sepsis, volume depletion, those who use nephrotoxic drugs such as aminoglycosides, mannitol, vancomycin, diuretics, tacrolimus, and amphotericin B are at higher risk of developing AKI. Myocardial dysfunction, hepatic failure, endothelial dysfunction, rhabdomyolysis, hemolytic uremic syndrome, blood coagulation disorders, acute respiratory distress syndrome, bacteremia and endotoxemia, and septic shock are among the risk factors for AKI in intensive care units.

- **Early Recognition of AKI**

There are newer biomarkers recognized for Acute kidney injury. These will aid in the early administration of corrective measures in the population at risk for AKI. The AKI panels are anticipated to be helpful in finding the time of initial insult nearly and assessing duration of AKI since they reflect the biomarkers that are expressed sequentially. It is also probable that the AKI panels would differentiate between various AKI etiologies & types based on variations in biomarker expression.

Novel biomarkers of Kidney Injury

Cystatin C, Kidney Injury Molecule-1 (KIM-1), Neutrophil Gelatinase Associated Lipocalin (NGAL), Na+/ H+ Exchanger Isoform 3 (NHE 3),

N Acetyl Glucosaminidase (NAG), Glutamyl transpeptidase, and Glutathione S transferase and Interleukin-18 are some of the biomarkers of AKI. The biomarkers of promise include a plasma panel (NGAL and cystatin C) and a urine panel (NGAL, IL- 18 and KIM-1).

The most reliable and independent indicator of AKI is the level of NGAL in urine (uNGAL) two hours after cardiopulmonary bypass.[39] Urinary NGAL levels were 4–6 times higher than the controls in a prospective study of 140 critically ill children, indicating that it was an excellent predictor of impending AKI. The increase in serum creatinine levels happens 48 hours after the increase in uNGAL. Compared to children without sepsis, children with sepsis had greater uNGAL levels. However the relationship with AKI remained intact.

Table 5: Novel Biomarkers in Acute kidney injury [40,41,42]

Host	Marker	Substrate-test	Comments
Rat	SSAT	Rat kidney- RTPCR and northern blot	SSAT was able to distinguish AKI with tubular injury from AKI without ATN
Rat & mouse	CYR61	Kidney and urine-western blot	CYR61 is upregulated in kidneys with IRI- able to distinguish prerenal from intrarenal AKI
Human	IL-18	Urine- ELISA	IL-8 elevated in human kidney ATN (native and transplanted kidneys)
Human	NHE3	Urine-semiquantitative immunoblotting	NHE3 differentiated prerenal from intrarenal causes of AKI
Human	Urine proteome pattern	Urine- mass spectroscopy	Humans following cardiopulmonary bypass surgery-markers at 2 and 6 hrs postoperatively highly sensitive and predictive of AKI
Human	KIM-1	Urine, kidney-multiple methods	Specific for ischemic AKI/ ATN when compared with other forms of kidney disease
Human, mouse	Gro-α, KC	Urine, blood-ELISA	Gro-α correlates with renal recovery from AKI/DGF in transplant, early increase in urine and blood well before rise in serum creatinine in AKI models
Human	NGAL	Blood, urine-western blot and ELISA	NGAL sensitive, specific and predictive marker of AKI in blood and urine of patients after cardiopulmonary bypass
Human	Actin, IL-6 & IL-8	Urine-dot immunoblot and ELISA	All 3 markers predicted prolonged AKI following renal transplantation in humans

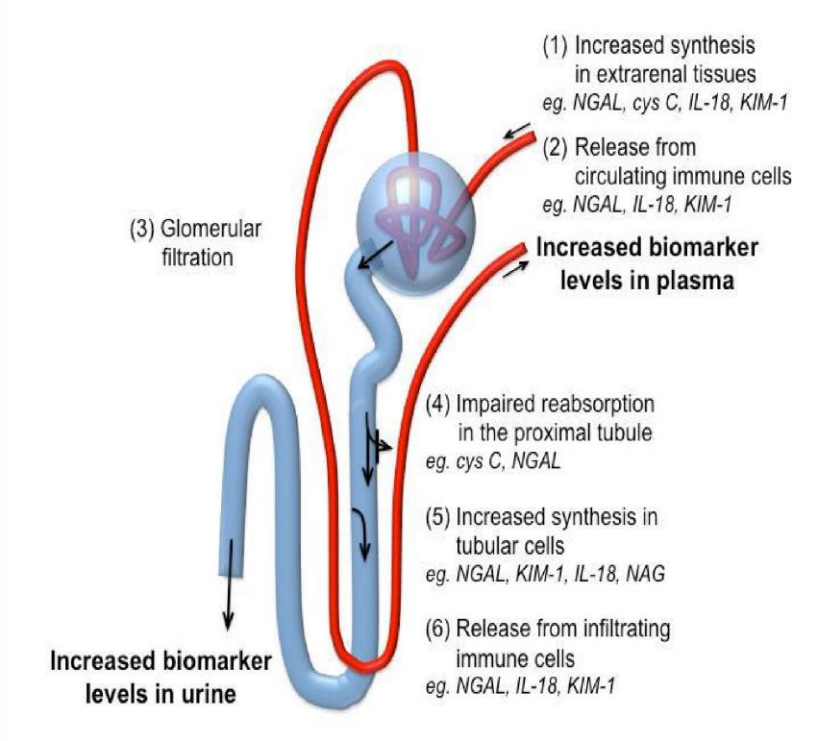


Figure 2: Novel biomarkers in Acute Renal Failure

Clinicians should attempt to prevent ARF because there are no many active treatment options. Correcting volume status, preventing nephrotoxic exposure, and being ready for high-risk procedures like contrast agent use are all things to think about.

- Methods to Prevent ARF in Hospitalized Patients

- Rapid correction of hypotension and its prevention.
- Before any surgery evaluate renal function.
- Avoid nephrotoxic drugs.
- Correct electrolyte imbalances and volume deficit, particularly before any surgery.
- Use non-ionic contrasts in place of traditional contrast agents, and use contrast judiciously.

- Identify and treat infection and oliguria immediately and efficiently.

➢ **Preventing Contrast Nephropathy**

- Decreasing the amount any of the contrast used, substituting non-ionic contrast for conventional contrast, and ensuring patients are properly hydrated prior to the procedure can all help lower the risk of contrast nephropathy.[43]
- The overall risk of contrast nephropathy can be reduced by 50%, from roughly 6% to 3%, by using non-ionic contrast agents. Rudnick and colleagues' study found that baseline CKD (serum creatinine level greater than 1.5 mg/dl), diabetes were risk factors for contrast nephropathy, the use of non-ionic contrasts decreased the occurrence of contrast nephropathy from 24% to 12% in patients who had the above two risk factors.
- Giving IV normal saline at a rate of 1 millilitre per kilogram per hour before to the procedure is the most efficient way to hydrate patients. Adding mannitol or a loop diuretic has no positive effect. Acetylcysteine pretreatment can somewhat slow the rise in creatinine levels, although it may have little clinical impact.

❖ **Outcomes**

- Depending on the patient's comorbidities and the kind of AKI, the mortality rate for severe AKI is about 50%. Those with ATN had a 60% mortality rate in the Madrid research, where as those cases with postrenal or prerenal illness had a mortality rate of 35%.[15] The majority of fatalities are caused by the underlying illness or its consequences rather than the AKI itself.[6] According to the Madrid data, the underlying disease accounted for 60% of deaths, with infection or cardiopulmonary failure accounting for the remaining 40%.
- AKI is more than just a sign of disease. AKI was observed in 183 out of 16,000 individuals who had computed tomography with contrast in a follow-up report [43]. Those with ARF had a mortality rate of 34%, while a matched cohort from the similarly exposed group had a mortality rate of only 7%.
- About 40% of survivors of ATN experience an incomplete recovery, while 50% of survivors fully regain renal function. Only about 5–10% of patients need maintenance hemodialysis.

MATERIALS AND METHODS:

1. SOURCE OF DATA:

The Information for the study will be collected from ACUTE KIDNEY INJURY Patients, (IPD) in Intensive Care Unit of B.L.D.E(DU), SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA-583106, KARNATAKA from May 2023 to December 2024.

2. METHOD OF COLLECTION OF DATA:

- Information collected through prepared proforma from each patient of Acute kidney Injury admitted in Intensive Care Unit.
- The sociodemographic data will be obtained using a face-to-face interview technique.
- A briefing will be given to the participants and/or their relatives about the objective of this study and assured confidentiality in collection of personal data.
- Complete Blood Cell (CBC) Count, Renal Function Test (RFT), Urine microscopy and Urine Routine, Blood culture and sensitivity, Sputum analysis, Peripheral smear for malarial parasite, USG Abdomen and KUB, Arterial blood gas analysis, Chest X-ray, Liver function test.

3. INCLUSION CRITERIA:

1. All patients admitted in ICU diagnosed with Acute kidney injury using KDIGO criteria or developed Acute Kidney Injury after admission to ICU.

2. Age above 18 years.

4.EXCLUSION CRITERIA:

Patients with Chronic Kidney Disease.

5.TYPE OF STUDY:

Observational Cross- Sectional study.

6.SAMPLE SIZE:

With anticipated Proportion of clinical profile of Acute Kidney Injury in ICU patients, the study would require a Sample size of 78 patients with 95% level of confidence with 5% level of significance and margin of error 0.05.

The sample size computed using the following formula:

Sample size (n) = (Z* σ/d) 2 Where, z is the z score= 2.576 d is the margin of error= 0.5 n is the population size σ is the Standard Deviation =1.71 α is the level of significance=0.01

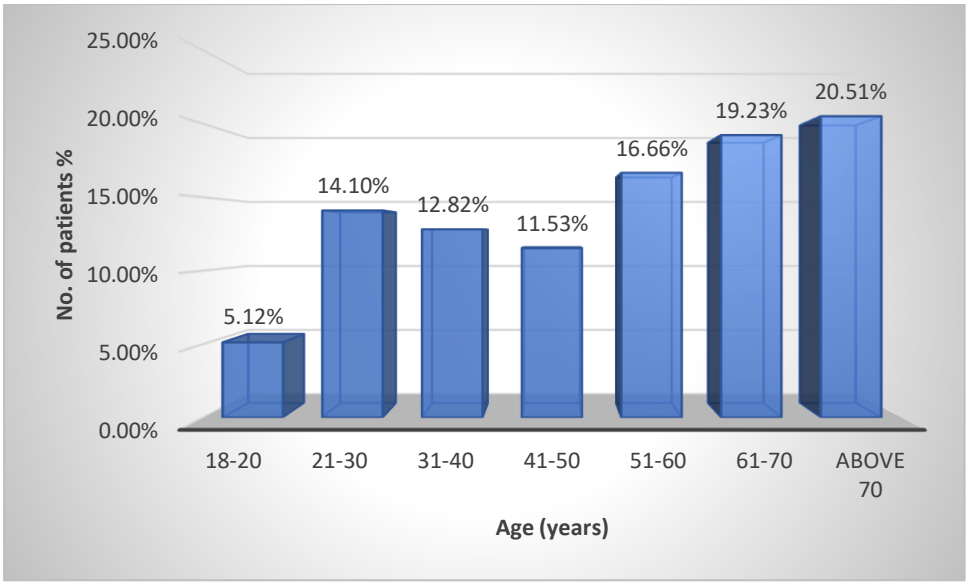
The estimated sample size of this study is 78.

STATISTICAL ANALYSIS: Data obtained is entered into an Excel sheet for statistical analysis. Continuous variables were summarized using mean and standard deviation, while categorical variables were represented using frequency and percentage. Data were analysed using an unpaired t-test for continuous variables and a chi-square test for categorical variables. A p-value of <0.05 was considered statistically significant. The results were illustrated through tables, bar diagrams, and pie charts.

RESULTS

Table 6: Distribution of cases according to age

Age Group	No. of cases	Percentage
18-20	4	5.12%
21-30	11	14.10%
31-40	10	12.82%
41-50	9	11.53%
51-60	13	16.66%
61-70	15	19.23%
Above 70	16	20.51%
Total	78	100

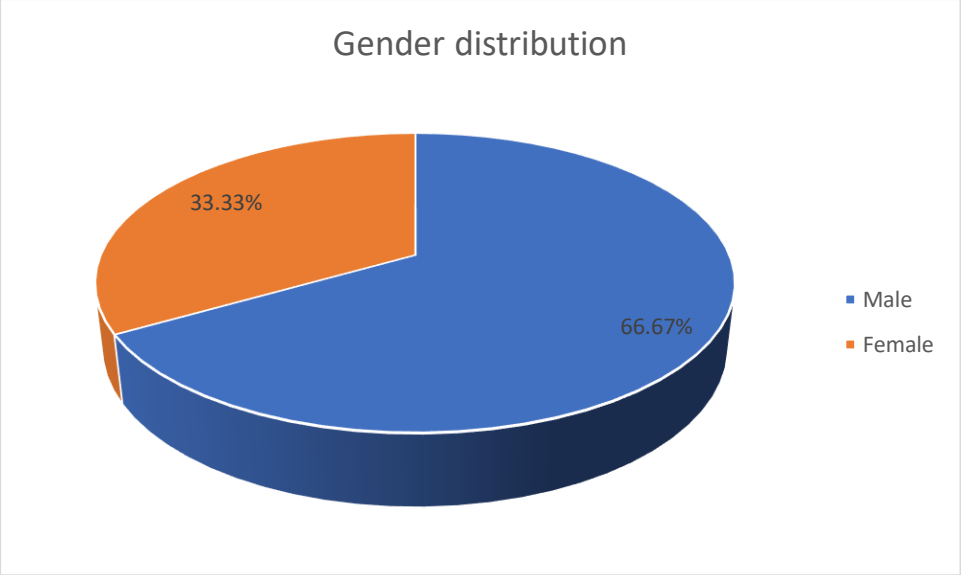


Graph 1: Distribution of cases according to age

In our study peak incidence of AKI is seen above 70 years (20.51%%), with 56.40% of cases seen above 50 years of age.

Table 7: Distribution of cases according to gender

Gender	No. of cases	Percentage
Male	52	66.67%
Female	26	33.33%
Total	78	100



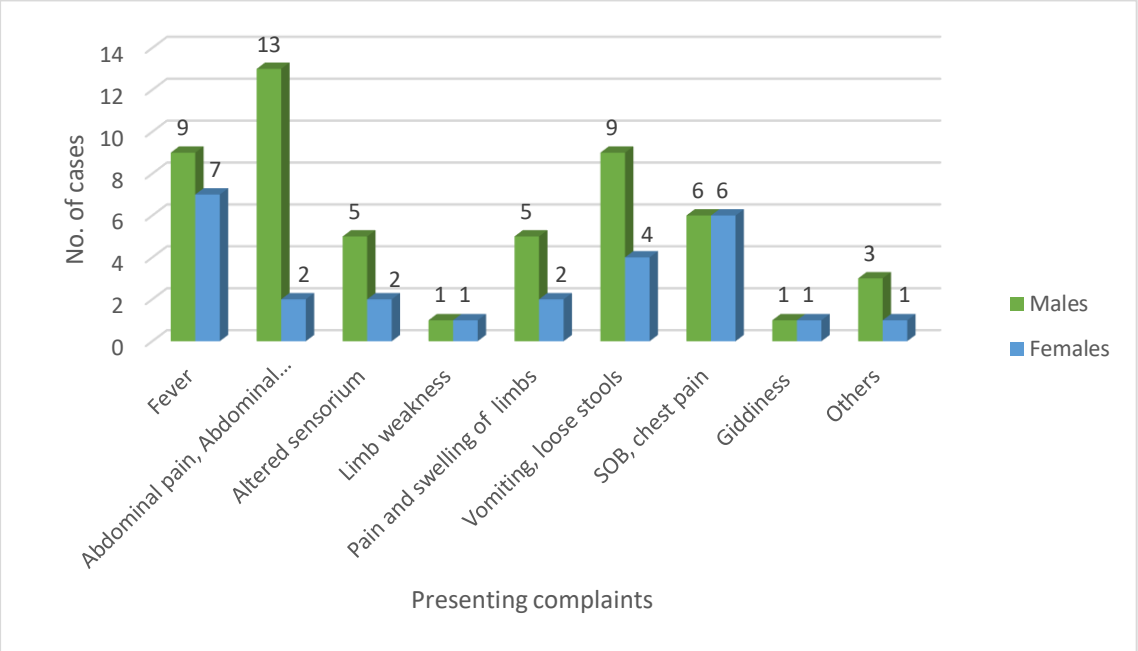
Graph 2: Distribution of cases according to gender

52 patients are male population in our study (66.67%) and 26 patients being females (33.33%).

Table 8: Distribution of cases according to presenting complaints

Presenting complaints	**Males**	**Females**	**Total**
Fever	9	7	16
Abdominal distension, Abdominal pain	13	2	15
Altered sensorium	5	2	7
Limb weakness	1	1	2

Pain and swelling of limbs	5	2	7
Loose stools, vomiting	9	4	13
SOB, chest pain	6	6	12
Giddiness	1	1	2
Others	3	1	4
Total	52	26	78

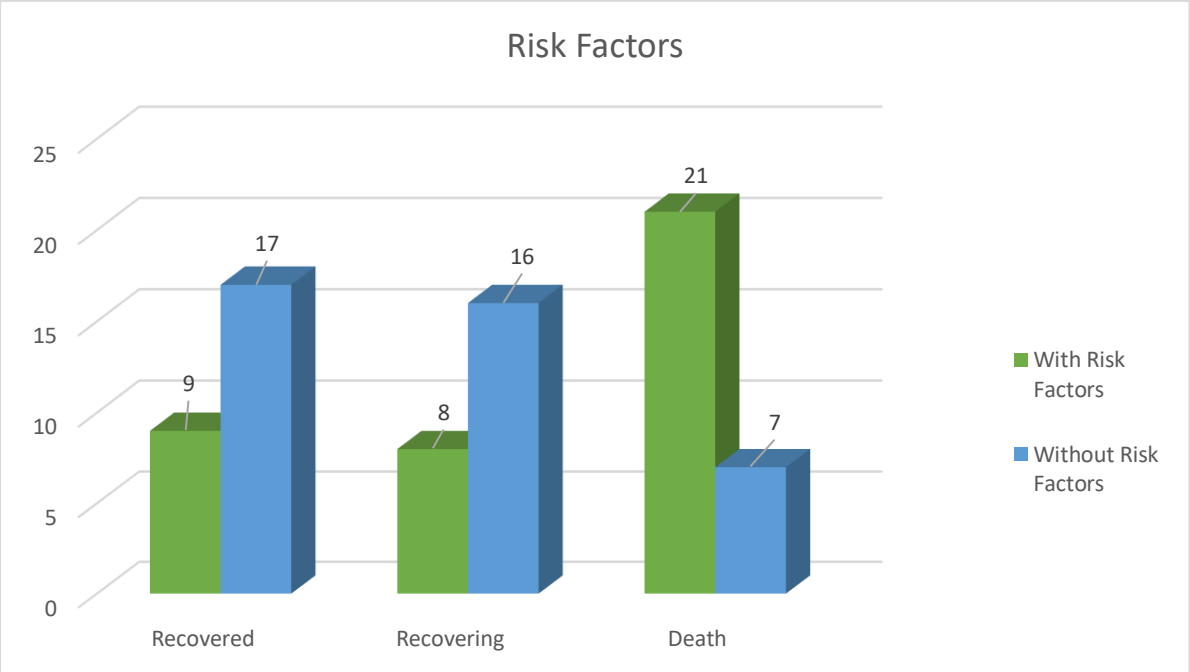


Graph 3: Distribution of cases according to presenting complaints

Predominant presentations in our study include Fever (16 patients), Abdominal pain or distension (15 patients), Altered sensorium (7 patients). 7 patients came with pain and swelling of limbs, 13 patients with loose stools, vomiting and 12 patients with SOB, chest pain. Other less frequent complaints include giddiness, limb weakness etc.

Table 9: Distribution of cases according to Risk factors

Risk Factors	Recovered	Recovering	Death	Total
With Risk Factors	9	8	21	38
Without Risk Factors	17	16	7	40
Total	26	24	28	78

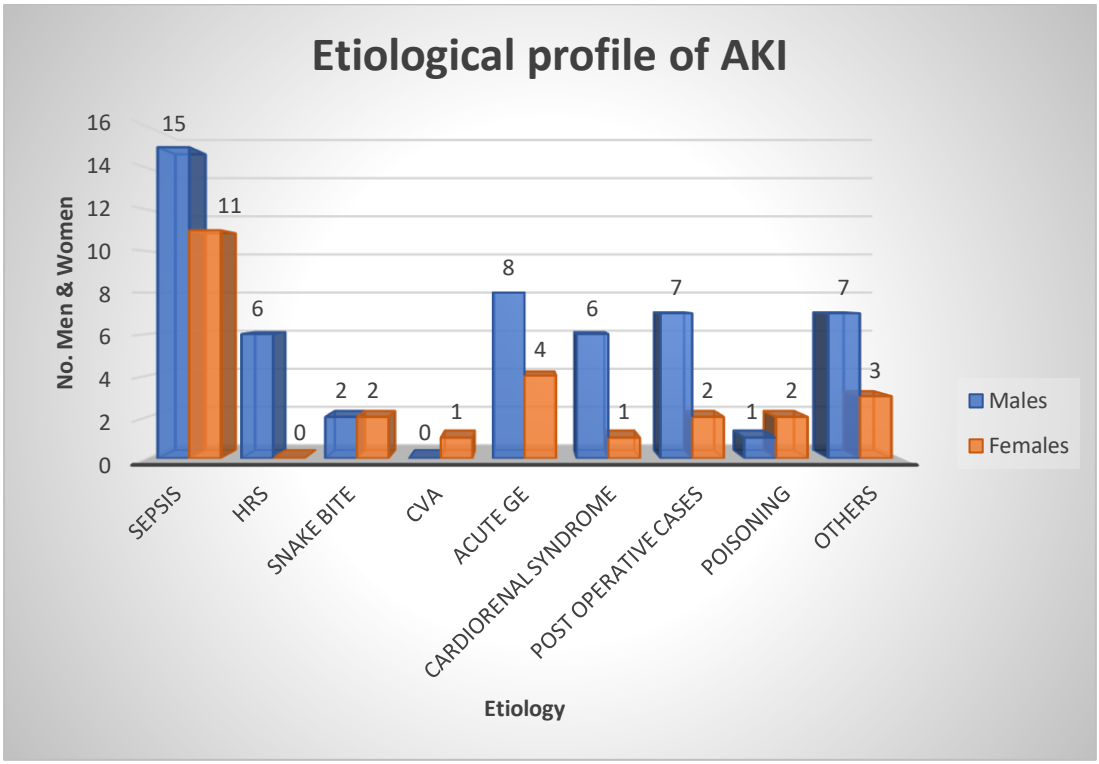


Graph 4: Distribution of cases according to Risk factors

In our study, out of 38 patients with risk factors, 9 patients recovered early, 8 patients showed delayed recovery and mortality was seen in 21 patients. Out of 40 patients without risk factors, 17 patients recovered early, in about 16 patients recovery was delayed, and 7 patients without risk factors died.

Table 10: Distribution of cases according to Etiology

Etiology	Males	Females	Total
Sepsis	15	11	26
HRS	6	0	6
Snake bite	2	2	4
CVA	0	1	1
Acute GE	8	4	12
Cardiorenal syndrome	6	1	7
Post operative cases	7	2	9
Poisoning	1	2	3
Others	7	3	10
Total	52	26	78

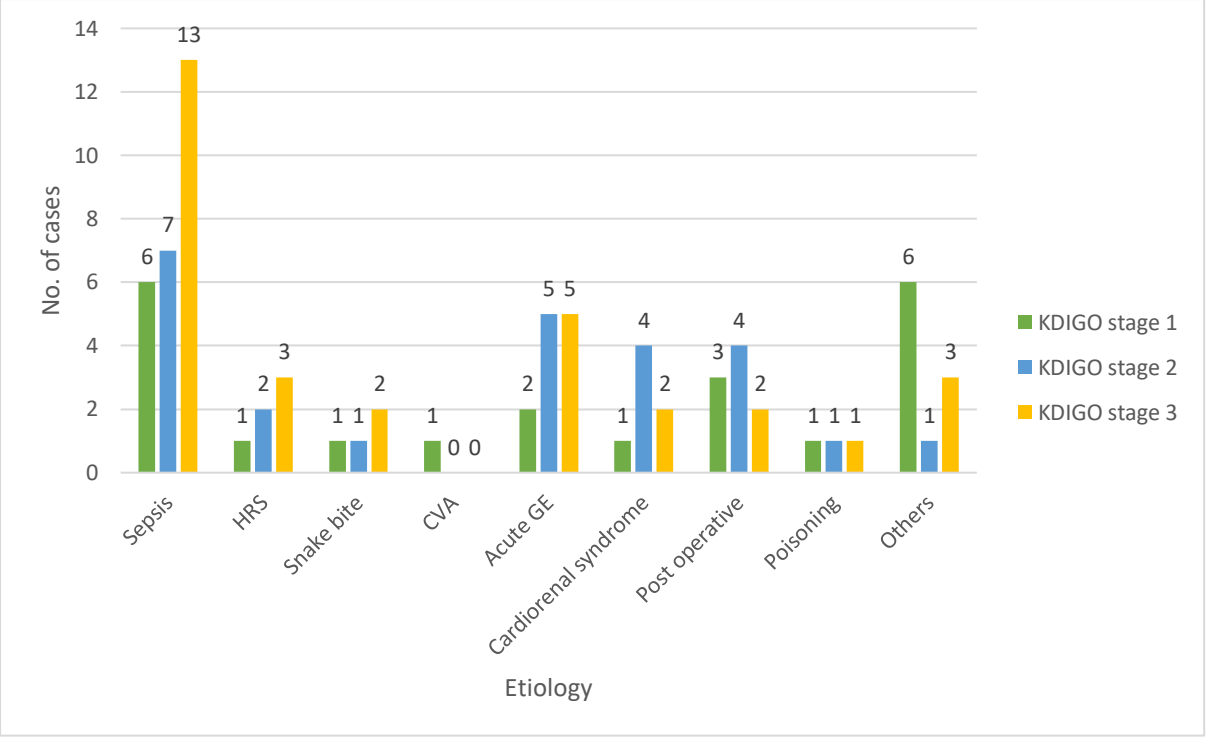


Graph 5: Distribution of cases according to Etiology

Sepsis was the commonest cause of AKI in ICU patients. The second most common cause was Acute GE. Next common etiologies include Cardiorenal syndrome, hepatorenal syndrome, post operative cases. 4 patients were of snake bite and 3 were of poisoning. Other less common causes include infections, CVA, DKA, seizures, HELLP syndrome, acute pancreatitis etc.

Table 11: Distribution of cases according to KDIGO Staging

Etiology	KDIGO stage 1	KDIGO stage 2	KDIGO stage 3	Total
Sepsis	6	7	13	26
HRS	1	2	3	6
Snake bite	1	1	2	4
CVA	1	0	0	1
Acute GE	2	5	5	12
Cardiorenal syndrome	1	4	2	7
Post operative cases	3	4	2	9
Poisoning	1	1	1	3
Others	6	1	3	10
Total	22	25	31	78



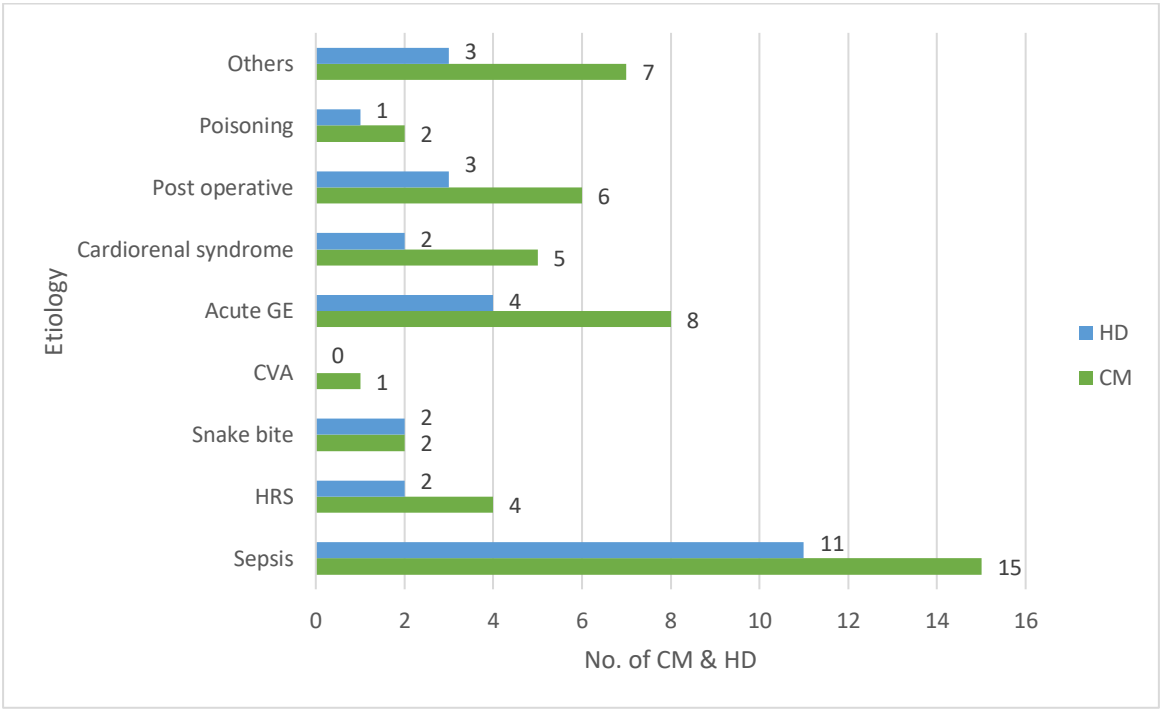
Graph 6: Distribution of cases according to KDIGO Staging

Most of the cases in our study fall under KDIGO stage 3, 39.74%, (31 patients), and 28.20% in KDIGO stage 1(22 patients) and 32.05% in KDIGO stage 2 (25 patients).

Table 12: Distribution of cases According to Management

Etiology	CM	HD	Total
Sepsis	15	11	26
HRS	4	2	6
Snake bite	2	2	4
CVA	1	0	1
Acute GE	8	4	12
Cardiorenal syndrome	5	2	7
Post operative cases	6	3	9

Poisoning	2	1	3
Others	7	3	10
Total	50	28	78

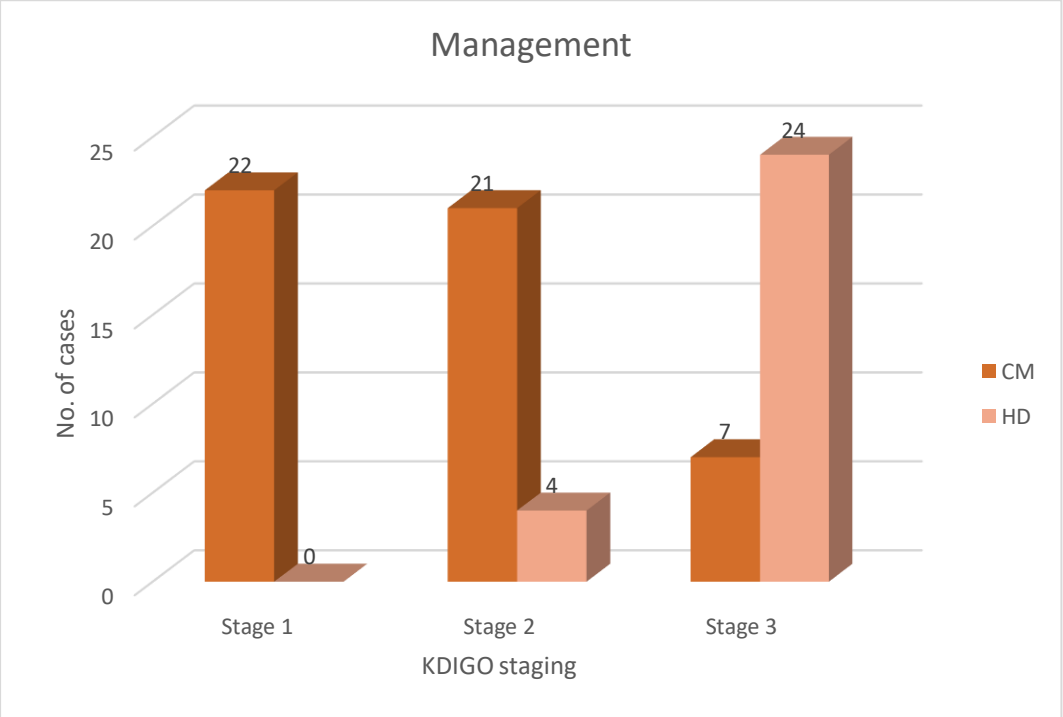


Graph 7: Distribution of cases According to Management

Out of 78 patients in our study, 50 patients were managed conservatively, 28 patients needed hemodialysis.

Table 13: Distribution of cases According to KDIGO Stage wise Management

KDIGO Staging	CM	HD	Total
Stage 1	22	0	22
Stage 2	21	4	25
Stage 3	7	24	31
Total	50	28	78



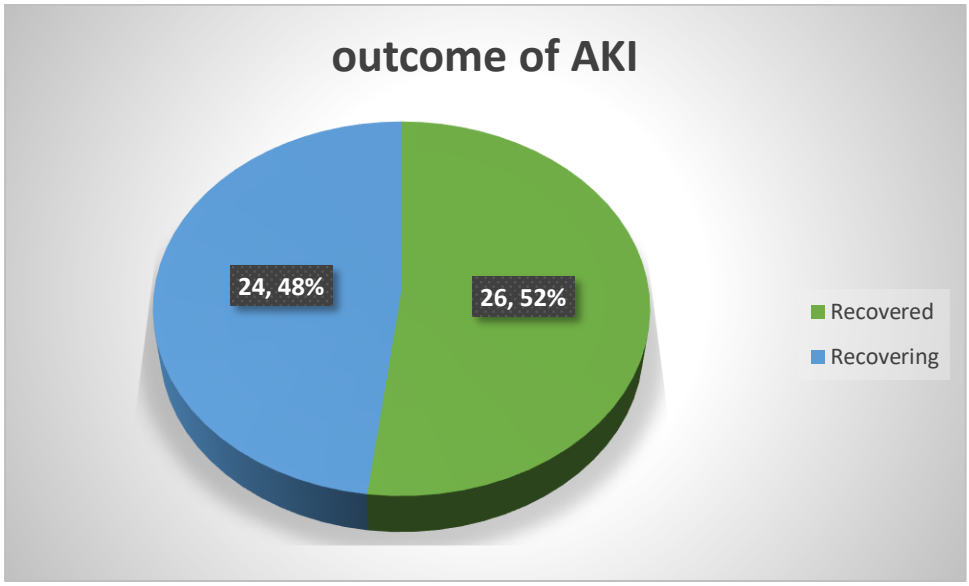
Graph 8: Distribution of cases According to KDIGO Stage wise Management

In our study, conservative management was given to all 22 patients under KDIGO stage 1. Out of 25 patients falling under stage 2, 21 patients were

managed conservatively, 4 patients required hemodialysis. 24 out of 31 patients falling under KDIGO stage 3 required hemodialysis.

Table 14: Distribution of cases according to outcome of AKI

Outcome of AKI	No. of cases
Recovered	26
Recovering	24

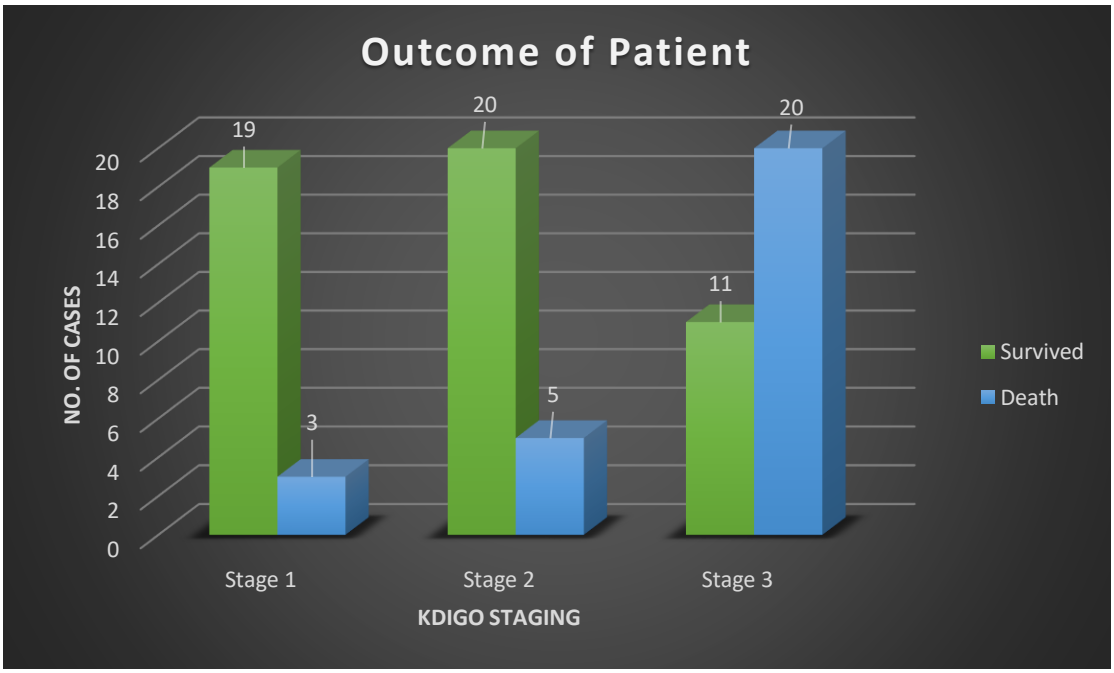


Graph 9: Distribution of cases according to outcome of AKI

In our study, out of 50 patients who survived, 52% patients recovered, & 48% are in the phase of recovery.

Table 15: Distribution of cases according to outcome of patient

KDIGO staging	Survived	Death	Total
Stage 1	19	3	22
Stage 2	20	5	25
Stage 3	11	20	31
Total	50	28	78

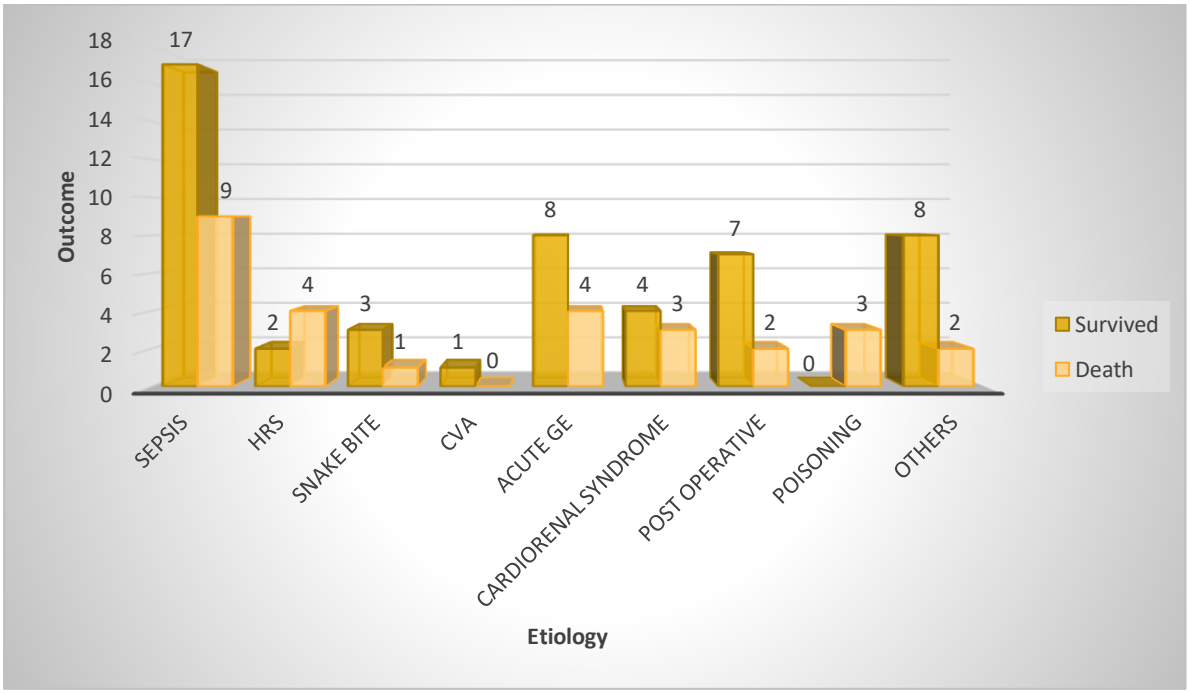


Graph 10: Distribution of cases according to outcome of patient

Out of 78 patients in our study, 28 patients died, 50 patients survived.

Table 16: Outcome depending on Etiology

Etiology	Survived	Death	Total
Sepsis	17	9	26
HRS	2	4	6
Snake bite	3	1	4
CVA	1	0	1
Acute GE	8	4	12
Cardiorenal syndrome	4	3	7
Post operative cases	7	2	9
Poisoning	0	3	3
Others	8	2	10
Total	50	28	78



Graph 11: Outcome depending on Etiology

Sepsis resulted in death of 9 patients out of 26 due to multiorgan dysfunction and failure. 4 patients of hepatorenal syndrome died out of 6. 4 out 12 patients of acute gastroenteritis died. Of the 4 cases of Snake bite one patient died and Cerebrovascular accident has shown no mortality. 3 patients out of 7 of cardiorenal syndrome and all 3 cases of poisoning died. Out of 9 post operative patients 2 died.

DISCUSSION

The main aim of this study was to understand the clinical and etiological characteristics of patients hospitalised to intensive care unit with AKI. Due to geographic variety and varying medical care standards, Etiology, course, and outcome vary throughout the world.

We examined 78 patients in our study. There were 26 females and 52 males present. Maximum number of cases were seen above the age of 50 years. Most common presenting complaints in our study are fever (20.51%), abdominal distension or pain (19.23%), loose stools and vomiting (16.66%). In

Vijaykumar sah, Satyam Prakash et al 2022 study fever was the most common presenting complaint (77%).

The most common cause of acute kidney injury in our study was sepsis (33.33%) of which 57.69% of cases were managed conservatively and remaining 42.31% needed dialysis. Mortality with sepsis was 34.61%. Second most common cause of AKI in our study was Acute Gastroenteritis (15.38%) of which 66.67% cases were managed conservatively and 66.67% patients of Acute GE survived with treatment. S.K. Agarwal et al. study found out that 11% of AKI is due to Acute diarrheal disease. Out of 78 patients, 11.53% of patients developed AKI post operatively, out of which 66.67% patients were managed conservatively and mortality was seen in 22.22%. Snake bite was the cause in 5.12% of cases with 25% mortality and 50% required hemodialysis. Cerebrovascular accident was the cause in one of the cases with no mortality and managed conservatively. Hepatorenal syndrome was the cause in 7.69% of cases with 33.33% requiring dialysis. Mortality in cases of HRS was 66.66%. Cardiorenal syndrome was the cause in 8.97% of cases of which mortality was seen in 42.85% of cases. Poisoning was the cause in 3.84% of cases and mortality was seen in all cases. Malaria was the cause in one case which was managed conservatively and survived.

The AKI definition of the RIFLE and AKIN categories is replaced by the KDIGO classification. As to the research conducted by Heng Chih Pan et al.,

the AKI incidence obtained by the KDIGO classification was greater than that determined through the RIFLE or AKIN classification.[44]

In our study out of 78 patients, 22 patients were diagnosed as KDIGO stage 1 and 25 patients as KDIGO stage 2, and 31 patients as KDIGO stage 3. All stage 1 patients were managed conservatively. 13.63% of KDIGO stage 1 patients died. Mortality may be a result of sicker patients taken admission to the ICU and the increased prevalence of related comorbidities. Out of 38 patients with risk factors, mortality was seen in 21 cases, 9 cases recovered early and 8 cases showed delay in recovery. Out of 40 patients without risk factors, mortality was seen in 7 cases, 17 cases recovered early and 16 cases showed delay in recovery.

Under KDIGO stage 2, 21 patients out of 25 were managed conservatively, of which 20% patients died. 24 patients out of 31 patients of KDIGO stage 3 required hemodialysis, only 7 patients were managed conservatively. Mortality among stage 3 patients was 64.51%. The etiological classification of our patients to be significantly associated with KDIGO criteria, as well we found the mortality also significantly associated with KDIGO criteria.

CONCLUSION

The most frequent cause of AKI in ICU patients is sepsis. It also has a high death rate. In each case of sepsis, preventing the development of multi-organ failure is crucial. Similarly, to reduce the high mortality rate linked to sepsis, multi-organ failure in the context of sepsis should be vigorously managed.

In our study higher mortality was seen in cases of poisoning, hepatorenal syndrome, cardiorenal syndrome and sepsis. Early recovery was seen with no risk factors. Delayed recovery was seen in patients with associated risk factors like DM, HTN, IHD, LD.

Dialysis was used to treat our patients cautiously and when necessary. In our hospital, the preferred mode of dialysis is hemodialysis because of availability of government schemes. The use of Peritoneal dialysis is drastically reduced because of the availability of SLED and CRRT.

Common causes of increased mortality include multi-organ dysfunction, lung and other infections, delayed diagnosis and treatment, and the frequent occurrence of comorbidities. Although there was a correlation between AKI and hospital outcomes, the prognosis of AKI severity is more affected by organ failure.

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ETHICAL CLEARANCE CERTIFICATE

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 914/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "CLINICAL PROFILE OF ACUTE KIDNEY INJURY IN INTENSIVE CARE UNIT (ICU) PATIENTS".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.VAITLA CHANDRAKANTH

NAME OF THE GUIDE: DR.SANJEEVKUMAR N. BENTOOR, PROFESSOR, DEPT. OF MEDICINE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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CONSENT FORM

BLDE(DU) SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA-586103

TITLE OF THE PROJECT: **CLINICAL PROFILE OF ACUTE KIDNEY INJURY IN INTENSIVE CARE UNIT PATIENTS.**

PRINCIPAL INVESTIGATOR: Dr. VAITLA CHANDRAKANTH
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All aspects of this consent form are explained to the patient in the language understood by him/her.

INFORMED PART

PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in this study will help to patient's survival and better outcome.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by code number. The code-key connecting name to numbers will be kept in a separate location.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime.

Dr. VAITLA CHANDRAKANTH is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr. VAITLA CHANDRAKANTH may terminate my participation in the study after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Dr. VAITLA CHANDRAKANTH Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. VAITLA CHANDRAKANTH has explained to me the purpose of research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

_____ _____

Participant / Guardian
Date

_____ _____

Witness to signature Date

CASE PROFORMA

CLINICAL PROFILE OF ACUTE KIDNEY INJURY IN INTESIVE CARE UNIT PATIENTS

NAME:

AGE/SEX: IP NO:

OCCUPATION:

ADDRESS: CASE NO:

DATE OF ADMISSION: DATE OF DISCHARGE:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

PAST HISTORY:

PERSONAL HISTORY:

DIET: APPETITE:

SLEEP: HABITS:

BLADDER/BOWEL:

FAMILY HISTORY:

GENERAL PHYSICAL EXAMINATION:

PALLOR: YES/ NO

ICTERUS: YES/ NO

CYANOSIS: YES/NO

CLUBBING: YES/NO

LYMPHADENOPATHY: YES/NO

EDEMA: YES/NO

VITALS:

PULSE RATE:

BLOOD PRESSURE: SPO2:

RESPIRATORY RATE: TEMPERATURE:

SYSTEMIC EXAMINATION:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

CENTRAL NERVOUS SYSTEM:

PER ABDOMEN:

INVESTIGATIONS:

COMPLETE BLOOD COUNT:

TOTAL COUNT	CELLS/CMM
NEUTROPHILS	%
LYMPHOCYTES	%
EOSINOPHILS	%
BASOPHILS	%
MONOCYTES	%
HEMOGLOBIN	GM/DL
PLATELET COUNT	LAKHS/CMM

URINE COMPLETE:

ALBUMIN	MG/DL
SUGAR	MG/DL
RBC	PER MICRO LT
EPITHELIAL CELLS	PER HPF
PUS CELLS	PER MICRO LT

RENAL FUNCTION TEST:

UREA	MG/DL
CREATININE	MG/DL
SODIUM	MEQ/L
POTASSIUM	MEQ/L

ARTERIAL BLOOD GAS (ABG):

BLOOD PH	
PO2	MMHG
PCO2	MMHG
HCO3	MMOL/L
LACTATE	MMOL/L

USG ABDOMEN AND KUB:

OTHER RELEVANT INVESTIGATIONS ACCORDING TO THE ETIOLOGY:

PROVISIONAL DIAGNOSIS:

OUTCOME:

DATE:

SIGNATURE:

MASTER CHART

S.No	Age	Gender	IP No	Presenting Complaints	Risk Factors	Etiology	Creatinine		Urine Output (ml/kg/hr)	KDIGO Stage	Management	Outcome of AKI	Outcome of patient
							Baseline	Rise					
1.	28	M	231391	Fever, rashes	-	Rickettsial fever	0.9	1.6	0.4	1	CM	R	S
2.	36	M	370757	Abdominal pain, lower limbs Swelling	LD	Hepatorenal syndrome	3	8.2	A	3	HD	RI	S
3.	75	M	239827	Loose stools, vomiting	DM	Acute GE	1.2	5.1	0.2	3	HD	-	D
4.	55	M	151256	SOB, chest pain	IHD	Cardiorenal syndrome	1.8	6.7	A	3	HD	-	D
5.	75	M	245089	Abdominal pain	-	Post operative	1.1	3	0.4	2	CM	RI	S
6.	45	F	349403	Right ULand LL weakness	-	CVA	0.9	1.7	0.4	1	CM	R	S
7.	77	F	214260	Pain and swelling of left foot	-	Snake bite	1.4	3.6	0.4	2	CM	RI	S
8.	60	M	231778	Loose stools, vomiting	DM, IHD	Acute GE	0.9	2.7	0.4	2	CM	RI	S
9.	49	M	16251	Altered sensorium	-	Sepsis	1.1	2.5	0.4	2	CM	R	S
10.	72	F	16017	Fever, SOB	DM	Sepsis	1.5	2.7	0.5	1	CM	R	S
11.	28	F	17712	Bleeding PV	Intubated	Post operative	0.8	1.4	0.5	1	CM	-	D
12.	26	F	243850	SOB, Giddiness	Intubated	HELLP syndrome	1.1	3.5	A	3	HD	-	D
13.	23	F	8176	Fever, vomiting	-	Sepsis	0.8	4.2	0.2	3	CM	RI	S
14.	25	M	234173	Altered sensorium	-	Post operative	0.9	2.9	0.2	3	CM	RI	S
15.	70	M	234488	Pain and swelling of left lower limb	DM	Sepsis	1.1	2.3	0.3	2	CM	R	S
16.	75	M	241256	Loose stools, vomiting	-	Acute GE	1.1	4	0.2	3	HD	-	D

17.	45	M	361248	Fever	-	Sepsis	2.1	6.2	0.2	3	HD	RI	S
18.	20	M	15872	Loose stools, vomiting	-	Acute GE	1.2	3.9	0.2	3	CM	-	D
19.	45	M	234910	Altered sensorium	IHD	Cardiorenal syndrome	1	2.8	0.4	2	CM	RI	S
20.	70	F	234909	Loose stools, vomiting	-	Acute GE	1.1	1.9	0.5	1	CM	R	S
21.	25	F	11346	SOB, vomiting	Intubated	OP poisoning	0.8	1.4	0.4	1	CM	-	D
22.	41	M	373909	SOB	Intubated	Sepsis	1.1	3.4	0.2	3	CM	-	D
23.	33	M	275219	Vomiting blood	LD	Hepatorenal syndrome	0.5	2.7	0.2	3	CM	-	D
24.	28	M	9187	Vomiting	Intubated	Hydrogen cyanide poisoning	0.9	2.5	0.4	2	CM	-	D
25.	80	M	11123	Pain and swelling of Right thumb	-	Scorpion bite	0.9	1.5	0.5	1	CM	R	S
26.	62	F	16148	Fever	-	Sepsis	0.9	2.1	0.4	2	CM	-	D
27.	25	F	15848	Fever	-	Sepsis	0.8	1.5	0.4	1	CM	R	S
28.	38	M	17185	Fever with chills, abdominal pain	-	Plasmodium Vivax Malaria	0.8	1.5	0.5	1	CM	R	S
29.	56	F	15282	Fever, SOB	-	Sepsis	1.1	2.5	0.4	2	CM	R	S
30.	30	F	10993	SOB	-	Paraquat poisoning	0.9	5.9	0.2	3	HD	-	D
31.	80	M	226962	Abdominal pain, vomiting	-	Post operative	1	2.2	0.4	2	CM	R	S
32.	70	M	10362	SOB	DM, HTN, IHD	Cardiorenal syndrome	1.1	2.5	0.4	2	CM	R	S

33.	76	M	13766	Chest pain	DM, HTN, IHD	Cardiorenal syndrome	1.1	2.3	0.4	2	CM	R	S
34.	48	M	237765	SOB, abdominal pain	DM, HTN, Intubated	Post operative	1.1	3	0.3	2	HD	RI	S
35.	19	F	15480	Altered sensorium	Intubated	Sepsis	0.8	3.5	0.2	3	HD	-	D
36.	70	M	184522	Giddiness	HTN, IHD	NSAID related	1	1.6	0.5	1	CM	R	S
37.	30	M	16400	Abdominal distension, abdominal pain	LD	Hepatorenal syndrome	0.9	4.9	0.1	3	HD	RI	S
38.	19	F	11944	Fever, altered sensorium	DM, Intubated	Sepsis	0.9	6.8	0.2	3	HD	-	D
39.	23	M	16665	Fever, vomiting	-	Sepsis	0.9	2.9	0.2	3	CM	-	D
40.	80	F	208522	Loose stools, vomiting	DM, HTN, Intubated	Acute GE	1.2	4.8	0.2	3	HD	-	D
41.	53	M	225697	Fever	-	Sepsis	2.2	6.8	0.2	3	HD	RI	S
42.	38	M	225199	Abdominal distension	LD	Hepatorenal syndrome	1.1	1.9	0.4	1	CM	-	D
43.	56	M	223053	Pain and swelling of Right index finger	-	Snake bite	1.2	2.1	0.4	1	CM	RI	S
44.	70	F	221380	Fever	HTN	Sepsis	1	2.6	0.4	2	CM	R	S
45.	69	M	221069	Abdominal distension	LD	Hepatorenal syndrome	1.1	2.3	0.3	2	CM	-	D
46.	35	M	220471	Involuntary movements, vomiting	-	Seizure disorder	1	2.5	0.4	2	CM	R	S
47.	59	M	14300	Altered sensorium	DM, LD	Hepatorenal syndrome	1.7	3.5	0.4	2	CM	-	D
48.	70	F	218161	SOB	DM, Intubated	DKA	1.1	1.9	0.5	1	CM	R	S
49.	34	M	217048	Pain over left side of face, LOC	-	Post operative	0.8	1.4	0.4	1	CM	R	S

50.	65	M	218101	LL swelling, fever	-	Sepsis	1.8	6.9	0.2	3	HD	RI	S
51.	70	M	242248	Abdominal pain	-	Post operative	1.3	3	0.4	2	HD	RI	S
52.	62	M	215397	Fever, LL swelling	-	Sepsis	1.2	2.3	0.5	1	CM	R	S
53.	72	F	213959	Fever	Intubated	Sepsis	1.5	4.6	0.3	3	CM	-	D
54.	59	M	213919	Fever, abdominal pain	-	Sepsis	2	7	0.2	3	HD	RI	S
55.	56	M	242407	LL weakness	DM, HTN, IHD	Cardiorenal syndrome	1.1	1.9	0.5	1	CM	R	S
56.	66	M	245707	Loose stools, vomiting	DM, HTN	Acute GE	1.2	2	0.5	1	CM	R	S
57.	38	F	2588	Abdominal pain	Intubated	Post operative	2.1	4.8	0.2	3	HD	-	D
58.	60	F	212556	Altered sensorium	DM, IHD	Sepsis	1.5	6	A	3	HD	-	D
59.	45	M	212810	Abdominal pain, vomiting	-	Acute pancreatitis	1	1.8	0.4	1	CM	R	S
60.	55	F	211712	Pain and swelling of left hand	-	Snake bite	2.3	7.1	0.2	3	HD	-	D
61.	56	F	226964	Loose stools, vomiting	-	Acute GE	1.1	1.9	0.4	2	CM	R	S
62.	84	M	16430	Abdominal pain, vomiting	-	Post operative	2.2	4	0.4	1	CM	RI	S
63.	70	F	207167	SOB, Chest pain	IHD	Cardiorenal syndrome	2.1	6.8	0.2	3	HD	-	D
64.	42	F	206489	Abdominal pain, vomiting	DM	DKA	1.2	4.6	0.2	3	HD	-	D
65.	80	F	199792	SOB, LL swelling	DM, HTN	Sepsis	2.4	5.5	0.3	2	HD	RI	S
66.	82	M	243869	Fever	-	Sepsis	1.2	4.6	0.2	3	HD	-	D
67.	80	M	197855	Fever, SOB	-	Sepsis	1.5	2.6	0.5	1	CM	R	S

68.	28	M	194381	Abdominal pain	LD	Acute pancreatitis	0.9	3.4	0.2	3	HD	RI	S
69.	75	M	194791	Loose stools, vomiting	-	Acute GE	2	5	0.3	2	CM	RI	S
70.	70	M	192532	Loose stools, vomiting	HTN, IHD	Acute GE	1.8	5.2	0.3	3	HD	RI	S
71.	40	M	193888	Loose stools, vomiting	-	Acute GE	1.4	3.8	0.3	2	CM	RI	S
72.	32	M	193914	Altered sensorium, fever	-	Sepsis	0.9	1.6	0.4	1	CM	R	S
73.	71	M	191245	SOB, chest pain	IHD, Intubated	Cardiorenal syndrome	0.9	2.1	0.3	2	CM	-	D
74.	20	M	189263	Pain and swelling of left ankle	-	Snake bite	0.8	5.4	0.2	3	HD	RI	S
75.	62	M	186846	Pain and swelling of Right LL	-	Sepsis	1.2	2.1	0.4	1	CM	R	S
76.	45	M	31913	Abdominal pain, vomiting	Intubated	Sepsis	0.9	6.4	0.2	3	HD	-	D
77.	55	F	386372	Loose stools, vomiting	-	Acute GE	1.5	3.2	0.4	2	CM	RI	S
78.	69	M	386410	Pain and swelling of Right LL	-	Sepsis	2.2	5.4	0.4	2	HD	RI	S