"ONE YEAR CROSS SECTIONAL STUDY OF ETIOLOGY AND OUTCOME OF HOSPITAL ACQUIRED ACUTE RENAL FALURE"

By

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KLE University, Belgaum, Karnataka

In Partial Fulfillment
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M. D. MEDICINE

Under the Guidance of

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MAY - 2010

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DECLARATION

I hereby declare that this dissertation entitled **"ONE YEAR CROSS SECTIONAL STUDY OF ETIOLOGY AND OUTCOME OF HOSPITAL ACQUIRED ACUTE RENAL FALURE"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. M. S. KHANPET** **MD, DNB (Nephrol)** Associate Professor, Department of Medicine, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum – 10.

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LIST OF ABBREVIATIONS USED

ADQI	-	Acute dialysis qualitative initiative
ALD	-	Alcoholic liver disease
ANCA	-	Anti neutrophil cytoplasmic antibodies
ANP	-	Atrionatriuretic peptide
ARF	-	Acute renal failure
ATN	-	Acute tubular necrosis
BUN	-	Blood urea nitrogen
Ca++	-	Calcium ion
CAARF	-	Community acquired acute renal failure
Cl^-	-	Cloride
CRRT	-	Continuous renal replacement therapy
CTVS	-	Cardiothoracic and vascular surgery
CVP	-	Central venous pressure
CVVDH	-	Continuous venovenous haemodialysis
CVVHD	-	Continuous venovenous hemodialysis
FENa	-	Fractional excretion of sodium
g/Kg/d	-	Gram per kilogram per day
GBM	-	Glomerular basement membrane
GFR	-	Glomerular filtration rate
HAARF	-	Hospital acquired acute renal failure
HCO_3^-	-	Bicarbonate ion
HTN	-	Hypertension
HUS	-	Hemolytic uremic syndrome
IABP	-	Intraaortic balloon pump counterpulsation

ICCU	-	Intensive cardiac care unit
ICU	-	Intensive care unit
IHD	-	Ischemic heart disease
JVP	-	Jugular venous pressure
K+	-	Potassium ion
KUB	-	Kidney ureter bladder
LDH	-	Lactate dehydrogenase
Meq/L	-	Milli equivalents per litre
mg/dL	-	Milli gram per deciliter
$Mg2^{+}$	-	Magnesium ion
MICU	-	Medical intensive care unit
mm Hg	-	Milligram of mercury
MODS	-	Multiple organ dysfunction syndrome
Na^{+}	-	Sodium ion
$NaHCO_3$	-	Sodium bicarbonate
NS	-	Not significant
NSAID	-	Non steroidal antiinflamatory drugs
NSICU	-	Neurosurgical intensive care unit
PCr	-	Plasma creatinine
PCWP	-	Pulmonary capillary wedge pressure
PEEP	-	Peak end expiratory pressure
Po_4^{3-}	-	Phosphate ion
RIFLE	-	Risk, injury, failure, loss and end stage kidney disease
RVD	-	Retroviral disease
S	-	Significant

S. Cr.	-	Serum creatinine
SCUF	-	Slow continuous ultrafilteration
SG	-	Specific gravity
SICU	-	Surgical intensive care unit
SIRS	-	Systemic inflammatory response syndrome
TIPS	-	Trasjugular porto systemic shunt
TTP	-	Thrombotic thrombocytopenic purpura
UCr	-	Urine creatinine
UNa	-	Urine sodium

ABSTRACT

Background and Objectives

Acute renal failure in the intensive care unit is a common condition in the hospitalized patients. Morbidity and mortality due to hospital acquired acute renal failure is high and varies from 19 to 59%. The present study was conducted to assess the etiology and outcome of the patients developing renal failure in the hospital (hospital acquired acute renal failure) in terms of mortality and morbidity, and to analyse causes, risks and prognostic factors, and final outcome of ARF in ICU setting.

Methods

The present one year cross sectional study was conducted in Department of Medicine on 65 patients admitted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during period of January 2008 to December 2008. Adult patients who developed renal failure (Creatinine ≥ 1.5 mg/dL) any time after 48 hours of admission were included in the study and were referred as HAARF. Patients who were having chronic kidney disease or who had acute renal failure on admission were excluded from study.

Results

The mean age of patients was 52.38±17.02 years, 87.69% were males. Comorbidity was seen in 69.23% (Hypertension, diabetes, CAD). HAARF had developed complicating the medical (58.46%) and surgical conditions (56.92%). Most common cause of HAARF was multifactorial etiology (70.77%) and 53.85% sepsis. MOD was noted in 40% of cases. Altered sensorium (96.43%),

metabolic acidosis (92%), MOD (88.46%), requirement of ionotropic support (84.44%) and mechanical ventilation (82.50%), oliguria (78.72%), hypotension (78.57%) were independent risk factors associated with outcome of HAARF ($p<0.05$). Overall mortality of HAARF was 63.08% and 36.93% of patients had recovery of them, 33.85% patients had complete recovery and 3.08% had partial recovery and they were independent of dialysis.

Conclusion

The HAARF patients had the high mortality despite of aggressive modern treatment. Commonest precipitating factor was multifactorial etiology followed by sepsis.

Key words

Hospital acquired acute renal failure; Sepsis; Multifactorial etiology; Multiple organ dysfunction.

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INTRODUCTION

Acute Renal Failure (ARF) is a common complication in patients admitted to the intensive care unit (ICU). Numerous causes are responsible for development of ARF.[1] Moreover ARF is often due to multifactorial aetiology in critically ill patients.

Acute renal failure (ARF) is the abrupt loss of renal function sufficient to decrease urinary elimination of nitrogenous waste (urea nitrogen and creatinine). although there is consensus about this general definition, few agree on the magnitude of the rise in serum creatinine necessary to ascribe a diagnosis ARF. The differences in the definition plus variances in the methods of patients accrual, population analysed, categorization of causes render development of broad based over view of ARF difficult. However two generalizations about contemporary ARF are compelling. First, ARF is predominantly a hospital acquired disorder. Second, the high mortality of patients with ARF is not explained entirely by comorbid conditions. Recent data indicate that ARF per say increase the risk of development of multiple non renal conditions that lead to death and disability.

The relative importance of factors contributing to ARF will be different depending on the underlying pathology and patient characteristics. A patient population of young trauma patients developing ARF can probably not be compared with older patients with ischemic and congestive heart disease developing ARF after cardiac surgery. The large differences in mortality for patients with ARF, as reported in recent trials (varying between 28 and 83%) can possibly be explained by differences in patient population.[2,3]

While functional outcome after ARF is usually good among the surviving patients, mortality rate is high close to 70% in ICU series. Although it is unfortunate that these mortality rates have remained fairly constant over the past decades, despite advances in critical and renal care, is attributed to the changing pattern of associated pathology and patient population[4,5,6,7] and these patients frequently have multy organ failure and die despite appropriate treatment of renal insufficiency. It should be noted that today's patients are generally much older and display a generally much severe condition than in the past. These factors, together with the more aggressive therapeutical possibilities presently available, could account for this apparent paradox.

The incidence of hospital acquired acute renal failure (HAARF) varies from 4.9 to 7.2%[8,9] and 3.79%[10] in the western[8,9] and Indian studies[10] respectively.

Increasing use of nephrotoxic drugs, invasive procedures,intravascular catheters and major surgical procedures (cardiovascular and abdominal surgeries) predispose patient to sepsis and renal failure.[11] Morbidity and mortality due to hospital acquired renal failure is high.[8] Mortality of hospital acquired renal failure varies from 19% to 59% [9,11] The true magnitude of the problem is not well described in the literature.[12]

The present study is a cross sectional study of HAARF in the ICUs of KLES Dr Prabhakar Kore Hospital with special reference to causative factors, clinical course and the outcome.

OBJECTIVES

The objectives of the present study were;

1. To study the etiology of hospital acquired acute renal failure.
2. To study the outcome of HAARF in terms of mortality and morbidity in the first two weeks.

REVIEW OF LITERATURE

Acute Renal Failure (ARF) is a syndrome characterized by rapid (hours to weeks) decline in glomerular filtration rate (GFR) and retention of nitrogenous waste products such as blood urea nitrogen (BUN) and Creatinine.[13]

ARF complicates approximately 5% of hospital admissions and upto 30% of admissions in ICU.[8]

Hospital acquired acute renal failure (HAARF) is defined as renal failure that developed during hospitalization for non renal related problems in patients whose admission serum creatinine level was normal.[11]

Acute renal failure is a common condition in hospitalized patients. The incidence of hospital acquired renal failure (HAARF) varies from 4.9 to 7.2%[8,9] of patients, Increasing use of nephrotoxic drugs, invasive procedures, intravascular catheters and major surgical procedures (cardiovascular and abdominal surgeries) predisposes patient to sepsis and renal failure. Morbidity and mortality due to hospital acquired renal failure is high.

Mortality of hospital acquired renal failure varies from 19% to 59%.[9,11] The true magnitude of the problem is not well described in the literature[12].

The spectrum of definitions in published studies of ARF is striking, ranging from severe (for example, ARF requiring dialysis) to relatively modest observable increases in serum creatinine concentration (for example, increase in serum creatinine of 0.3 to 0.5 mg/dl above baseline). Solomon et al[14] used the

definition of an increase in serum creatinine of 0.5 mg/dl within 48 h of radiocontrast exposure in a widely cited study that showed a borderline significant difference in ARF among individuals given saline infusion versus furosemide or mannitol before radiocontrast exposure.

Many other definitions of ARF have been applied; some are outlined below. The most liberal of definitions have been used in intervention studies aimed at ARF prevention, usually in the context of radiocontrast exposure, one of the few instances in which ARF can be anticipated.

Whether male gender is a true risk for ARF or simply a risk for being diagnosed with ARF is unclear. Regardless, the association of ARF with male gender highlights one of the limitations of the use of a definition of ARF that is creatinine based and not age, gender, and race adjusted. Changes in serum creatinine are not specific and do not discriminate the nature and type of renal insult (for example, ischemic, nephrotoxic) or the site and extent of glomerular or tubular injury, and levels are relatively insensitive to small changes in GFR.[15] Moreover, changes in serum creatinine may lag behind changes (decline or recovery) in GFR by several days.

Definitions of ARF

- 0.5 mg/dl increase in (SCr) within 48 h.[16,17,18,19,20.]
- 0.5 mg/dl increase in SCr if baseline SCr ≤1.9 mg/dl, or 1.0 mg/dl increase in SCr if baseline SCr 2.0 to 4.9 mg/dl, or 1.5 mg/dl increase in SCr if baseline SCr ≥5.0 mg/dl[8]

- 0.9 mg/dl increase in SCr if baseline SCr <2.0 mg/dl, or 1.5 mg/dl increase in SCr if baseline SCr ≥2.0 mg/dl, and "remained elevated for at least one additional consecutive determination".[21]

- "Sudden" rise of >2 mg/dl in subjects with prior "normal" renal function, or "sudden" increase in SCr of ≥50% with "mild to moderate" basal chronic renal failure with SCr <3.0 mg/dl, or "elevation of SCr at admission with normal or increased renal size (except with myeloma or hydronephrosis with cortical atrophy)".[22]

- 50% increase in SCr to at least SCr of 2.0 mg/dl ("ARF"),100% increase in SCr to at least SCr of 3.0 mg/dl ("severe ARF").[23]

- 25% increase in SCr to at least SCr of 2.0 mg/dl within two days.[24]

- 0.9 mg/dl increase in SCr if baseline SCr <2.0 mg/dl to at least 2.0 mg/dl, or 1.5 mg/dl increase in SCr if baseline SCr ≥2.0 mg/dl (baseline defined as lower of most recent SCr in past 3 months or lowest value during hospitalization).[25]

- 0.5 mg/dl increase in SCr to at least 2.0 mg/dl, or admission SCr ≥2.0 mg/dl with no history of renal disease.[26]

- 0.5 mg/dl increase in SCr or 25% increase from baseline within 48 h.[27]

- SCr ≥3.0 mg/dl with baseline SCr <1.8 mg/dl, or "acute decrease" in creatinine clearance to ≤25 mL/min after surgery, trauma, hypotension, or sepsis.[28]

- 1.0 mg/dl increase in SCr over 2 days.[29]
- >50% increase in SCr to at least 1.4 mg/dl.[30]
- >0.3 mg/dl and >20% increase in SCr.[31]
- ≥1.0 mg/dl increase in SCr, or ≥20 mg/dl or 50% increase in BUN.[32]
- 6 graded criteria - >0.3 mg/dl and >20% increase in SCr on day 1, 2, or 3, and day 5, 6, or 7, or >0.3 mg/dl increase in SCr on day 1, 2, or 3, or >0.3 mg/dl and >20% increase in SCr on day 1 or 2, or ≥2.0 mg/dl increase in SCr on day 1 or 2, or ≥1.0 mg/dl increase in SCr on day 1, or ≥20 mg/dl or ≥50% increase in BUN on day 1[33]
- >50% increase in SCr in absence of "volume responsive prerenal status," or >1 mg/dl increase in SCr with known renal insufficiency.[34]
- ≥0.3 mg/dl increase in SCr.[35]

Rifle criteria

Recently, acute dialysis qualitative initiative (ADQI) group[36] formulated a new classification for ARF. The risk, injury, failure, loss and ESKD (RIFLE) classification, that defines 3 grades of severity Classes R (Risk), Class (F) Failure and Class (I) Injury and two outcome classes. Loss and end stage kidney disease. Many studies[37,38] have applied RIFLE criteria in hospitalized patients particularly in ICU patients.

The intent of ADQI is to provide an objective, dispassionate distillation of the literature and description of the current state of practice of dialysis and related therapies. The purpose is to develop a consensus of opinion, with evidence where possible, on best practice and to articulate a research agenda to focus on important unanswered questions. This approach is a blend of "expert panel" and "evidence appraisal" and was chosen in order to achieve the best of both methods.

The objectives for ADQI are the following:

- Standardize and possibly optimize the process of dialysis for the critically ill patient.
- Develop consensus recommendations for best practice.
- Establish evidence based statements and possibly guidelines where applicable.
- Identify questions for future research and consider study design options

Table 1: RIFLE definition of acute renal failure (Adapted from Bellomo et al, 2004)

GFR criteria	Urine output criteria	
1. Risk increased serum creatinine × 1.5 Or decreased GFR > 25%	< 0.5 ml/kg/h × 6 h	High sensitivity
2. Injury increased serum creatinine × 2 or decreased GFR > 50%	< 0.5 ml/kg/h× 12h	
3. Failure increased serum creatinine × 3 or Decreased GFR > 75% or anuria × 12 h	< 0.3 ml /kg / ×24 h	High specificity
4. Loss of persistent ARF, complete loss of kidney function > 4 weeks		
5. ESRD end stage renal disease (> 3 months)		

In conclusion, ADQI is a moving process that will produce evidence based statements on different issues concerning acute dialysis. The first step was to try to reach consensus on Continuous Renal replacement Therapies, an area where major controversies are still present. The next step will be the development of consensus statements that should provide the basis for recommendations to be used in clinical practice. Their effort aims at obtaining a common ground where acute dialysis should be discussed and optimized. At the present time there is very little agreement on how much, when and how dialysis should be provided.

Differences between HAARF and CAARF [11]

1. The studies have found that Patients with HA-ARF were significantly older compared to patients with CA-ARF.

2. The mean age of all patients was 54 years with a 67% male predominance.

3. There were no significant differences in age, sex, peak serum creatinine levels, or underlying medical history.

4. The incidence of CA-ARF was 3.5 times greater than that of HA-ARF (0.55% vs 0.15%).

5. Prerenal causes of ARF were more common among CA-ARF than HAARF (35% vs 19%; p=0.07), but intrarenal causes were more common among HA-ARF (81% vs 55%; p=0.07). All the cases of obstruction occurred in CAARF

6. Patients with HAARF were sicker than patients with CAARF, patients with CAARF were more likely to require hemodialysis.

7. Prognosis was significantly worse in patients with HAARF compared to CAARF. Mortality rate among HAARF patients was 51% compared to CAARF patients 20%. A one more study mentioned mortality of 59% among HAARF patients compared to 35% among CAARF patients.

8. Full recovery occurred in only 30% among HAARF group by discharge compared to 59% in CAARF group.

9. It is documented that HAARF is associated with increased risk of dying. However, Nash et al have shown mortality of only 37.8%.

10. Mortality was higher in HAARF (59% vs 33%; p=0.03), and the incidence of recognized predictors of mortality was higher in patients with HA-ARF than in those with CA-ARF: oliguria (59% vs 35%; p=0.04); sepsis (73% vs 35%; p=0.004); stay in the intensive care unit (ICU) or mechanical ventilation (55% vs 6%; p=0.001); and multiorgan failure (59% vs 24%; p=0.002).

11. Those with HAARF were twice as likely to require dialysis as those with CAARF. The mortality was high in younger patients with CAARF and in older patients with HAARF, but the dialysis-related mortality rate was three fold higher among patients with HAARF.

12. While mean ± SD length of hospital stay was more prolonged in HA-ARF than CAARF (26 ± 28 days vs 12 ± 11 days; p=0.001), the 120- day survival rate was lower in HA-ARF than CA-ARF (43% vs 66%; p=0.05).

13. High mortality and morbidity noted in HAARF because it is often intra renal type of ARF (ATN) and occurs in older age group of patients with comorbid conditions, and they have severe medical illness which is nonrenal to start with, later on developing into, renal failure who often required RRT which had dialysis related complications and these patients were also associated more frequently with documented independent predictors of mortality such as oliguria, sepsis, hypotension, metabolic

acidosis, MOD, and longer ICU stay or need for mechanical ventilation and ionotropic support occurred.

CLASSIFICATION

ARF may complicate host of diseases that for purpose of diagnosis and management are conveniently divide into three categories, prerenal intra renal and post renal failure.[39]

Table 2: Classification of HAARF

Disease Category	Percentage of Patients with Acute Renal Failure (%)
Prerenal Azotemia	55 – 60
Intrinsic renal azetomia	35 – 40
Postrenal azotemia	< 5

Prerenal Azotemia

Decrease in glomerular filtrate rate resulting from renal hypoperfusion reversed on restoration of renal perfusion and glomerular ultrafiltration pressure without compromising the integrity of renal parenchyma.

Renal azotemia (Intrinsic)

Decrease in glomerular filtration rate resulting from renal hypoperfusion, nephritis or nephrotoxin, not reversed immediately upon discontinuation of insult and associated with involvement of renal parenchyma. Here the sudden decrease

in glomerular filtration rate that characterizes ARF is secondary to intrinsic renal damage mainly affecting tubules, interstitium, glomeruli and/or vessels, we are facing a parenchymatous ARF. Multiple causes have been described,

Acute interstitial nephritis

Disease characterized by decrease in GFR resulting from inflammation of tubulo interstitium. During the last years, acute tubulointerstitial nephritis is increasing in importance as a cause of acute renal failure. For decades infections were the most important cause. At present, antimicrobials and other drugs are the most common causes.

Acute glomerulonephritis or vasculitis

Decrease in GFR resulting from glomerular or vessel inflammation.

Acute renovascular disease

Disease characterized by decrease in GFR resulting from obstruction of renal artery or vein (thrombosis or embolism) in a single functioning kidney or with bilateral kidney disease.

Postrenal azotemia (Obstructive uropathy)

Obstruction at any level of the urinary tract frequently leads to acute renal failure. Decrease in GFR resulting from obstruction in urinary collecting system.

AETIOLOGY OF ARF

In general, the causes of ARF have a dynamic behavior as they change as a function of the economical and medical development of the community. Economic differences justify the different spectrum in the causes of ARF in developed and developing countries. The setting where ARF appears (community versus hospital), or the place where ARF is treated (intensive care units [ICU] versus other hospital areas) also show differences in the causes of ARF.

I. Major Causes Of Prerenal Azotemia

A. Intravascular Volume Depletion

- Hemorrhage: traumatic, surgical, gastrointestinal, postpartum.
- Gastrointestinal losses: vomitting, nasogastric suction, diarrhea.
- Renal losses: drug induced or osmotic diuresis, diabetes insipidus, adrenal insufficiency.
- Skin and mucous membrane losses: burns, hyperthermia, and other causes of increased insensible losses.
- "Third space" losses: pancreatitis, crush syndrome, hypoalbuminemia.

B. Decreased Cardiac Output

- Diseases of myocardium, valves, pericardium, or conducting system.
- Pulmonary hypertension, pulmonary embolism, positive-pressure mechanical ventilation.

C. Systematic Vasodilatation

- Drugs: antihypertensives, afterload reduction, anesthetics, drug overdoses.

- Sepsis, liver failure, anaphylaxis.

D. Renal Vasoconstriction

- Norepinephrine, ergotamine, liver disease, sepsis, hyperkalemia.

E. Pharmacologic Agents That Acutely Impair Autoregulation and GFR in Specific Settings

- Angiotensin- Converting enzyme inhibitors in renal artery stenosis or severe renal hypoperfusion.
- Inhibition of prostaglandin synthesis by nonsteroidal anti-inflammatory drugs during renal hypoperfusion.

II. Major Causes of Acute Intrinsic Renal Azotemia

A. Diseases Involving Large Renal Vessels

- Renal arteries: thrombosis, atheroembolism, thromboembolism, dissection, vasculitis (for example, Takayasu).
- Renal Veins: thrombosis, depression.

B. Diseases of Glomeruli and the Renal Microvasculature

- Inflammatory: acute or rapidly progressive glomerulonephritis, vasculitis, allograft rejection, radiation.
- Vasospastic: malignant hypertension, toxemia of pregnancy, scleroderma, hypercalcemia, drugs, radicontrast agents.
- Hematologic: hemolytic-uremic syndrome or thrombotic thrombocytopenic purpura, disseminated intravascular coagulation, hyperviscosity syndromes.

C. Diseases Characterized By Prominent Injury To Renal Tubules Often With ATN

- Ischemia caused by hypoperfusion.
- Exogenous Toxins (for example, antibiotics, anticancer agents, radio contrast agents, poisons).
- Endogeneous toxins (for example, myoglobin, hemoglobin, myeloma light chains, uric acid, tumorlysis).

D. Acute Disease of the Tubulointerstitium

- Allergic interstitial nephritis (for example, antibiotics, nonsteroidal anti inflammatory drugs).
- Infectious (viral, bacterial, fungal).
- Acute cellular allograft rejection.
- Infiltrations (for example, lymphoma, leukemia, sarcoid).

III. Some Disease Of Glomeruli And The Renal Microvasculature Associated With Acute Intrinsic Renal Azotemia

A. Glomerulonephritis or Vasculitis

- Associated with anti-glomerular basement membrane antibody.
  - anti-GBM Ab.
  - Goodpasture syndrome if associated with lung hemorrhage.
- Associated with antineutrophil cytoplasmic antibodies (ANCA).
  - Wegner granulomatosis.
  - Microscopic or Churg-Strauss variant of polyarteritis nodosa.
  - Renal-limited crescentic glomerulonephritis.

- Associated with glomerular immune complexes and hypocomplementemia.
  - Acute diffuse proliferative glomerulonephritis (postinfectious).
  - Membranoproliferative glomerulonephritis.
  - Subacute bacterial endocarditis.
  - Cryoglobulinemia.
  - Systemic lupus erythematosus (SLE).
- Associated with absence of hypocomplementemia, anti-GBM Ab and ANCA.
  - Immunoglobumin A nephropathy.
  - Schonlein-Henoch purpura.
  - Classic polyarteritis nodosa.
  - Radiation injury.
  - Abdominal abscess.

B. Hyperviscosity syndromes

- Multiple myeloma.
- Waldenstrom macroglobulinemia.
- Polycythemia.

C. Hemolytic-Uremic Syndrome or Thrombotic Thrombocytopenic Purpura

- Infections.
  - Viral (for example, enterovirus, coxsackievirus, influenza, virus, hepatits A virus, human immunodeficiency virus).

- Bacterial (for example, Escherichia coli, Shigella, Salmonella, Yersinia, Campylobacter).

- Chemotheraputic Agents and other drugs
  - Chemotheraputic Agents (mitomycin C, cisplatin + bleomycin).
  - Cyclosporin , oral contraceptives.
- Immunologic Diseases
  - SLE, rheumatoid arthritis, sjogren, ankylosing spondylitis.
- Other
  - Idiopathic, familial, pregnancy and puerperium, after renal or bone marrow transplantation.

D. Miscellaneous

- Accelerated hypertension.
- Sclroderma crisis.
- Toxemia of pregnancy.
- Drugs.
  - Cyclosporine, Amphotericin B.
- Radiocontrast agents.

IV. Some Exogenous Nephrotoxins That Are Common Causes Of Acute Intrinsic Renal Azotemia With Acute Tubule Necrosis

A. Antibiotics

Acyclovir**,** Aminoglycosides**,** Amphotericin B**,** Foscarnet**,** Pentamidine.

B. Organic solvents

- Ethylene glycol, Toluene.

C. Poisons

- Paraquat, Snake bites.

D. Chemotheraputic agents

- Cisplatin, Ifosfamide.

E. Anti inflammatory and immunosuppressive agents

- NSAIDs, Cyclosporine.

F. Radiocontrast agents

G. Bacterial toxins

V. Causes Of Acute Postrenal Azotemia

A. Ureteric Obstruction

- Intraluminal: stones, blood clot, sloughed renal papillae, uric acid or sulfonamide crystals, fungus balls.
- Intramural: postoperative edema after ureteric surgery.
- Extraureteric: iatrogenic (ligation during pelvic surgery).
- Periureteric: hemorrhage, tumor or fibrosis.

B. Bladder Neck Obstruction

- Intraluminal: bladder carcinoma, bladder infection with mural edema, neurogenic, drugs (for example, tricyclic antidepressants, ganglion blockers).
- Extramural: prostatic hypertrophy, prostatic carcinoma.

C. Urethral obstruction

- Phimosis, congenital valves, stricture tumor.

PATHOPHYSIOLOGY OF ACUTE RENAL FAILURE

Prerenal ARF

Prerenal ARF is the most common form of ARF and represents a physiologic response to mild - moderate renal hypoperfusion.[40] It rapidly reverses upon restoration of renal blood flow and glomerular ultrafiltration pressure. Renal parenchymal tissue is not damaged. Severe hypoperfusion may lead to ischemic injury of renal parenchyma and intrinsic renal ARF. Prerenal ARF may complicate any disease that induces hypovolemia, low cardiac output, systemic vasodilatation, or selective intrarenal vasoconstriction.

Autoregulatory dilatation of afferent arterioles is maximal at mean systolic arterial blood pressures of ~ 80 mmHg, and hypotension below this level is associated with a precipitous decline in GFR. Lesser degrees of hypotension may provoke prerenal ARF in the elderly and in patients with diseases affecting the integrity of afferent arterioles. In addition, drugs that interfere with adaptive

responses in renal microcirculation may convert compensated renal hypoperfusion into overt prerenal ARF or trigger progression of prerenal ARF to ischemic intrinsic renal ARF.[40,41]

In the face of azotemia and oliguria, several markers are useful in order to diferentiate prerenal azotemia versus ATN, and are shown in. All those markers are useful in the absence of a prior diuretic dose, except for the fractional excretion of urea which is not affected. It is also important to remember the causes of falsely high/low fractional excretion of sodium presented in an abnormality of the routine urine analysis would suggest underlying acute or chronic renal disease. It is not unusual for the urinary sodium value to suggest a prerenal element, but the urine creatinine to suggest renal dysfunction or vice versa. If even one of many parameters suggests a prerenal element, any potential prerenal factors should be identified and reversed. Prerenal azotemia is confirmed if the urine output improves and the azotemia resolves with the administration of isotonic fluids or improvement in the underlying condition, for example, heart failure.

Ischemic ARF

In ischemic ARF there is hypoperfusion, which induces ischemic injury to renal parenchymal cells, particularly tubular epithelium, and recovery typically takes 1 to 2 weeks after normalization of renal perfusion as it requires repair and regeneration of renal cells. In most extreme forms ischemia leads to bilateral renal cortical necrosis and irreversible renal failure. The course of ischemic ARF characterized by five phases

Prerenal phase

Decrease in glomerular filtrate rate resulting from renal hypoperfusion reversed on restoration of renal perfusion and glomerular ultrafiltration pressure without compromising the integrity of renal parenchyma.

Initiation phase (hours to days)

Numerous ischemic insults, alone or in synergistic combination with nephrotoxins, initiate epithelial and vascular cell injury, resulting in an extremely rapid decrease in GFR appropriately termed the "initiation phase". Initial period of renal hypoperfusion during which ischemic injury is evolving. GFR declines because;

- Glomerular ultrafiltration pressure is reduced as a consequence of fall in renal blood flow.
- The flow of GFR within tubules is obstructed by casts and necrotic debris.
- There is backleak of glomerular filtrate through injured tubular epithelium.

Ischemic injury is most prominent in terminal medullary portion of proximal tubule (S_3 segment, pars recta) and the medullary portion of the thick ascending limb of loop of Henle.

Cellular ischemia results in series of alterations in energetics iron transport and membrane integrity that ultimately leads to cell injury and if severe, cell apoptosis or necrosis. These alterations include depletion of ATP, inhibition of active sodium transport and other solutes, cell swelling, cytoskeletal disruption

and loss of cell polarity, cell to cell and cell to matrix attachment, accumulation of intracellular calcium, altered phospholipid metabolism, oxygen free radical formation, and peroxidation of membrane lipids.

Extension phase

The initiation phase is immediately followed by a phase that has recently been termed the "extension phase".[42] During the extension phase, multiple interrelated events lead to a worsening of epithelial and endothelial cell injury and cell death, primarily in the cortical-medullary region of the kidney. Existence of this phase has been postulated to occur in the clinical arena, but substantiating physiologic data are lacking. Nevertheless this concept is built on sound investigational data from both clinical sources and animal models. It also provides an important instructional framework upon which to base therapy, and it emphasizes the absolute necessity of a rapid diagnosis and institution of therapy in clinical ARF.

Maintenance phase (1 – 2 weeks)

GFR stabilizes (5 to 10 mL/min), urine output is lowest, and uremic complications arise.[43] GFR remains low due to intrarenal vasoconstriction and medullary ischemia triggered by release of vasoactive mediators from injured endothelial cells (for example, decreased nitric oxide, increased endothelin-1, adenosine and platelet activating factor), congestion of medullary blood vessels and reperfusion injury induced by reactive oxygen species derived from leucocytes or renal parenchymal cells.

Epithelial cell injury per se may contribute to persistent intrarenal vasoconstriction by process termed tubuloglomerular feedback.

Recovery phase

It is characterized by renal parenchymal cell, particularly tubule epithelial cell, repair and regeneration and a gradual return of GFR to or towards premorbid levels. It may be complicated by marked diuretic phase due to excretion of retained salt and water and other solutes, continued use of diuretics, and or delayed recovery of epithelial cell function relative to glomerular filtration.

Nephrotoxic ARF

Acute intrinsic renal ARF can complicate exposure to many structurally diverse pharmacological agents. These agents induces an acute fall in renal blood flow and GFR, a relatively benign urine sediment, and low FeNa.[44]

Contrast nephropathy classically presents as an acute (24 to 48 hours) but reversible (peak 3 –5 days, resolution within 1 week). Syndrome appears to be dose related, and its incidence is only slightly reduced in high risk individuals by use of more expensive low osmolality, nonionic contrast agents**.**[45]

Direct toxicity to tubular epithelial cells and/or intratubular obstruction are major pathophysiologic events in ARF induced by many antibiotics and anti-cancer drugs. The formation of intratubular casts containing filtered immunoglobin light chains including Tamm-Horsfall protein produced by thick ascending limb cells, is the major trigger for ARF in multiple myeloma.[46]

Intratubular obstruction is also an important cause of ARF in patients with severe hyperuricosuria and hyperoxaluria.

Although ATN is the most common cause of hospital-acquired ARF, one must consider the various intrinsic renal parenchymal or hemodynamic derangements responsible for ARF. These include diseases that primarily affect the glomerulus (glomerulonephritis), interstitium (interstitial nephritis), blood vessels (vascular occlusion or vasculitis).

Alterations in glomerular hemodynamics

These are increasingly recognized as a cause of ARF. These alterations include afferent arteriolar vasoconstriction (hepatorenal Syndrome) or efferent arteriolar vasodilation (angiotensinconverting enzyme inhibitors). The latter is seen when renal blood flow is already compromised by diuretics, severe cardiac failure, or renal artery stenosis. In addition, less well defined derrangements in intrarenal hemodynamics are likely responsible for the ARF of sepsis, potent vasodilators, and the nonsteroidal antiinflamatory drugs (NSAIDs). In these cases, the urinary sediment is non relevant, and the renal biopsy if performed tend to be normal. Recovery of renal function is expected, provided the offending drug is removed or the underlying condition is corrected.

Acute interstitial nephritis

It is usually due to drug allergy, of which penicillins, cephalosporins, sulfonamides, diuretics and NSAIDs are the most common agents. Patients will present with fever, rash, arthralgias, eosinophilia, and eosinophiluria (excepting

NSAIDs).[8] Other less frequent causes are pyelonephritis, myeloma multiple, uric acid nephropathy and ocasionally infiltrative disorders, such as lymphoma, leukemia, and sarcoidosis. Oxalate nephropathy may complicate acute ethylene glycol ingestion, see. The urine sediment is usually non relevant, but crystalluria, pyuria, and white blood cell casts can be seen, even in the absence of infection.

Vascular disease

It is a frequently overlooked cause of ARF. Malignant hypertension usually accompanied by retinopathy, thrombocitopenia, and microangiopathy can cause ARF. Microangiopathy and thrombocitopenia also accompany hemolytic uremic syndrome or thrombotic thrombocytopenicpurpura (TTP). Renal infarction due to trauma, arterial embolus, or thrombosis can cause ARF with fever, hematuria, acute flank pain, ileus, leucocytosis, and an increased LDH level. This syndrome often mimics an acute abdomen. Renal atheroesclerotic or cholesterol microemboli commonly occur following aortic manipulation (surgery or catheterization), besides ARF, gastrointestinal bleeding (due to microinfarction), livedo reticularis of the lower extremities, patchy areas of ischemic necrosis in the toes, hypocomplementemia, and eosinophilia are common. Finally, renal vasculitis often causes ARF. These syndromes are identified by their multisystem manifestations, very active urine sediment (hematuria, pyuria, red and white blood cell casts, and proteinuria), and in the cases of Wegener's and polyarteritis nodosa, the presence of ANCA in the serum.

Acute tubular necrosis

It is the most common cause of hospital and ICU acquired ARF, which is broadly divided into toxic and ischemic causes, drugs that induce renal damage and mechanisms.[8] Among the more common toxins causing ATN are the aminoglycoside antibiotics. Risk factors for aminoglycoside nephrotoxicity include volume contraction, age, hypokalemia, concomitant use of other nephrotoxins, and a short-dosing interval. After an initial loading dose, the maintenance dose should be adjusted based on the patient's creatinine clearance. The routine use of peak and trough serum levels does not decrease the likelihood of ATN.

Table 3: Drugs inducing renal damage

Drug	Damage
Diuretics, ACE, B blockers	Decrease in renal perfusion
NSAIDS Contrast	Impaired intrarenal hymodynamics
Aminoglycoside, Amphot B	Tubular toxicity
Lactams, NSAIDS, Furosemide, Cimetidine	Allergic interstitial nephritis

Radiographic contrast agents may cause ARF in patients with preexisting renal insufficiency, diabetes mellitus, poor left ventricular function, or when multiple studies are done in a 24-hr period. The volume of contrast used (>1.5 ml/kg) appears directly related to nephrotoxicity. In patients at very high risk for contrast nephropathy, nonionic contrast may be slightly less nephrotoxic.[18]

However, volume expanding these high risk patients with intravenous crystalloids is the BEST prophylaxis.[18]

Intravenous mannitol,furosemide, and calcium channel blockers do not appear to lessen nephrotoxicity, but theophylline may be of adjunctive value. Acetylcysteine appears to offer protection against contrast toxicity[19] though its use in very high risk patients requires further study. Most cases of contrast nephrotoxicity are nonoliguric and resolves within a few days. Rarely, a patient will require acute dialysis.

Permanent loss of renal function likely does not occur. Rhabdomyolisis is producing ARF at an increasing rate. Drugs (for example, heroin, cocaine and lovastatin) and major crush injuries have joined alcohol, seizures and muscle compression as common causes. All have the potential of producing myoglobinuria and ARF, particularly if extracellular volume depletion or shock exists simultaneously. Hyperkalemia, hyperuricemia, hyperphosphatemia, and high levels of creatine-kinase, with low Bun/Cr ratio also result. Hypocalcemia occurs early; hypercalcemia appears during recovery.[47] Dark heme-positive urine without red blood cells is a major diagnostic clue.

Prophylaxis against ATN depends on aggressive intravenous crystalloids administration. The addition of mannitol and bicarbonate (1/2 NS with 12.5 g/L of mannitol and 50 mEq/L of NaHC03/L at 250 to 500 ml/hr) may be a useful adjunct. Ischemic insults to the kidney occur with prolonged hypotension, suprarenal aortic or renal artery occlusion (either with clot or clamp), and sepsis. The renal tubular cells are particularly susceptible to ischemic insults because

their baseline balance between oxygen supply and demand is tenuous;[47] thus, whenever systemic or intrarenal blood flow decreases slightly, ischemic insult to the tubular cell may occur. This imbalance of oxygen supply and demand may help to explain the beneficial effects attributed to loop diuretics in some studies; by inhibiting active chloride and sodium transport in the ascending limb of the loop, these agents decrease metabolic work and, therefore, oxygen requirements. More than 50% of all cases of oliguric ARF in the hospital are due to sepsis. This condition appears related to a simultaneous decrease in systemic vascular resistance, reducing renal plasma flow and GFR. ARF occurs independently of systemic hypotension. Fever, leucocytosis, and overt signs of sepsis may be absent. A mild alteration in mental status or respiratory alkalosis may be the only clinical clue. Oliguria and or azotemia in this setting should be considered occult septicemia unless disproved.

Sepsis and renal failure

The incidence of ARF is approximately 20% to 25% in patients who have moderately severe sepsis and normal blood pressure; once hypotension occurs, the incidence of ARF exceeds 50%.[2,48,49] The fundamental cause of ATN in sepsis, even in the absence of hypotension, is renal hypoperfusion and ischemic injury to proximal tubular cells. An important mechanism of renal hypoperfusion is the combined effect of arterial vasodilation and intrarenal vasoconstriction. Another mechanism is intrarenal microvascular injury caused by PMN and complement-induced endothelial injury and intravascular thrombosis.[50]

The intrarenal vasoconstriction associated with sepsis has been ascribed to the local release of endothelial-derived vasoconstrictors, including endothelin, thromboxane A2, and leukotrienes.[51] Renal hypoperfusion also increases renal susceptibility to superimposed nephrotoxic events, which are common in septic patients.Acute renal failure in sepsis is more often part of the multiorgan dysfunction syndrome (MODS) associated with sepsis. MODS is caused by diffuse microvascular injury, which leads to inadequate perfusion and hypoxia of the lung, heart, liver, and other organs.

Other causes of hospital acquired acute renal failure

Acute renal failure caused by nonsteroidal anti-inflamatory drugs is associated with the presence of certain risk factors as true hypovolemia, cardiac failure, cirrhosis with ascites, and sepsis when the effective arterial blood volume is decreased; and with advanced age, chronic renal insufficiency and diabetes when the effective arterial blood volume is normal.

Some patient populations within intensive care units are at high risk for developing ATN. These patients include postoperative and sepsis.[52,53,54]

Postoperative ARF is associated with substantial increase in morbidity, length of ICU and hospital stay and mortality. The postoperative period is currently one of the most prevalent settings of ARF. For example 27% of 748 cases of ARF reported by liano and pauscal were encountered in post operative settings.[8] older studies by charlson etal indicated 25% of elective, non cardiac surgical procedures were complicated by an acute rise in serum creatinine of 20% or greater[8] in 11% of these patients, 50% decline in endogenous creatiine

clearance occurred[8] more recent studies by chartow et al., using veteran affairs patient databases, indicate that the development of ARF sufficient to require renal replacement therapy occurs in 0.4 to 7.5% of patients undergoing cardiac surgery and 0.6% of patients undergoing general surgery, and is dependent on the number of preoperative risk factors.[18,55]

Risk factors for acute tubular necrosis after surgery

Chronic renal disease: creatinine 2 mg/dl, advanced age, emergency surgery, cardiac dysfunction, diabetes mellitus, atherosclerotic vascular disease, Obstructive jaundice.

- Type of Surgery - Qardiac, AAA repair, hepatobiliary
- Postoperatives Variables

Cardiac dysfunction, Re-do surgery, Number of transfusions Angiographv within 24 hours of surgery.

In many elective procedures, underlying comorbidity (diabetes mellitus, chronic hypertension, vascular disease, congestive heart failure,) leads to diminished baseline GFR and reduced renal reserve.[8,18,55] with this background, the surgical experience appears to potentially induce afferent arteriolar renal vasoconstriction and diminished GFR.[19,56] if additional renal insult is encountered, clinical ARF occurs. These additional renal insults often referred to as second hits and include re-operation, sepsis, nephrotoxin exposure, circulatory/ volume deficits, and heart failure.[8,18,55]

Other causes of Acute Renal failure in the ICU are caused by osmotic nephropathy such as with substances that are added as vehicles to drug formulations as propylene glycol and sucrose. Other causes of osmotic nephropathy include mannitol, methanol or ethylene glycol.

A percentage of contemporary HAARF occurs in patients of ICU. For example, 27% of 748 cases of ARF reported by liano pauscal were encounterd in ICU[8] and frequency of ARF in patients admitted to ICU ranges from 6 to 23%.[57] In the most of ICU acquired ARF, the ARF occurs in the setting of multiorgan failure. For example, more than 90% of cases of ICU acquired ARF have failure of one or more additional organ systems and failure these other systems nearly always proceeds the development of ARF. In the recent ICU experience of Brivet et al[2] failure of another organ system was nearly uniformly present in the patients with ARF.[8] In all series of ICU associated ARF, hemodynamic instability, nephrotoxins, and sepsis are concomitantly present in more that two-thirds of patients.[8,57,58,59]

ARF is commonly encountered in patients with HIV infection.[54] The cause of ARF in HIV population are diverse and include, prerenal condition, post renal failure (due to intratubilar micro cristalisation from sulfa drugs/protease inhibitors or uretric obstructon from lymphoma), and renal disorders, including thrombotic microangiopathy, HIV associated nephropathy, glomerulopathy, interstitial nephropathy, and acute tubular injury occurring in the setting of sepsis and/or nephrotoxins.[47,60]

Patients of neoplastic disease are also high risk for HAARF.[53,57] The frequency development of ARF in the patients of hematological malignancies may be up to 40% in some series.[53] Sepsis and tumour lysis syndrome, hypercalcemia, hyperuricemia, nephrotoxins, and prerenal factors are predominant causes of ARF in the patients of malignancies. Less commonly encountered causes include tumour infiltration, obstructive uropathy and glomerulopathy.

Two of the most common conditions predisposing to HAARF are sepsis and nephrotoxin exposure. Sepsis was the most common factor 48% of the cases predisposing to development of ICU associated ARF in the study Brivet et al[2] not only hospital acquired sepsis often linked to ARF, but community acquired sepsis is also commonly associated with ARF.[2]

Table 4: Clinical approach to the diagnosis of acute renal failure

History, physical examination (including funduscopy and weight), detailed review of Hospital chart, previous records and drug history.
Urinalysis including specific gravity, dipstick, sulfosalicylic acid, microscopy and Staining for eosinophilis.
Flowchart of serial blood pressures, weights, BUN, serum creatinine, major clinical events, interventions and therapies.
Routine blood chemistry assays (BUN, creatinine, Na^+, K^+, Ca^{2+}, HCO_3^-, Cl^-, PO_4^{3-}) and hematologic tests (complete blood count and differential white blood cell count)
Selected special investigations: Urine chemistry, eosinophils, and/or immunoelectrophoresis. Serologic tests: anti-glomerular basement membrane antibodies, antineutrophil cytoplsmic antibodies, complement, antinuclear antibodies, cryoglobulins, serum protein electrophoresis, anti-streptolysin O or anti- Dnase titers. Radiologic evaluation: plain abdominal film, renal ultrasonography, intravenous pyelography, renal angiography. Renal biopsy.

Clinical approach to HAARF and CAARF remains the same once the patient is considered to have community acquired or hospital acquired ARF.

HAARF is basically occurs in hospitalized patients, who are admitted secondary to non renal cause of illness with the basal level of creatinine being normal during the admission of patient in to ICU.

HAARF is associated with severe illness and also it occurs more commonly in contrast induced nephropathy during angiography or angioplasty procedures in the cardiac patients, similarly in the post operative patients, especially CABG, and other common surgical procedures, these patients are belong to older age group with comorbid conditions like HTN, DM, also have high incidence of sepsis, MOD, hypotension, ionotropic support, mechanical ventilation and altered sensorium.

The most common cause of HAARF are drugs and sepsis, hence we have to be vigilant enough to avoid nephrotoxic drugs, better fluid management, and early institution dialysis before the development of severe acute renal failure.

Intra renal type renal failure is the most common cause of HAARF followed by prerenal and post renal ARF. The incidence of HAARF is increasing inspite of advances made in modern era. And it is associated with high mortality and morbidity.

The patients are considered to have CAARF when the patients are admitted to hospital with deranged renal parameters secondary to febrile illness or other cause, CAARF is a less severe illness when compared to HAARF.

The incidence of community acquired acute renal failure (CAARF) is decreasing in the western countries (0.1%) where as tropical country like India

still has the high incidence (1.5%). The factors responsible for this higher incidence include hot climate predisposing to hypovolemic insults and poor nutrional status, and increasing susceptibility to infections.

A high history of G6PD deficiency in certain ethnic groups predispose them to hemolysis when exposed to certain drugs and infections.

CAARF is commonly associated with diarrheal diseases, copper sulfate poisoning, snake bites and insect stings and among the infections most commonly it occurs in complicated malaria, leptospirosis, HUS, leprosy, kalaazar, typhoid, post infectious glomerolonephritis.

CAARF due to Obstetrical emergencies still continues to be high in India. CAARF is associated with low mortality and morbidity, belong to are younger age group, prerenal type of ARF is the most common type in CAARF.

Table 5: Useful clinical features, urinary findings, and confirmatory test in the differential diagnosis of major causes of acute azotemia[39]

Causes of ARF	Some Suggestive Clinical Features	Typical Urinalysis	Some Confirmatory Tests
Prerenal Azotemia	Evidence of true volume depletion (thirst, postural or absolute hypotension and tachycardia, low jugular vein pressure, dry mucous membrane and axillae, weight loss, fluid output > intput) or decreased effective circulatory volume (e.g heart failure, liver failure), treatment with NSAIDs or ACE inhibitor	Hyaline casts FE_{NA} < 1% U_{NA} <10 mEq/L SG >1.018	Occasionally requires invasive hemodynamic monitoring; rapid resolution of ARF on restoration of renal perfusion
Intrinsic Renal Azotemia			
Diseases involving large renal vessels			
Renal Artery Thrombosis	History of atrial fibrillation or recent myocardial infarction, nausea, vomiting, flank or abdominal pain	Mild proteinuria Occasionally red blood cells	Elevated lactate dehydrogenase with normal transaminases, renal arteriogram
Athero-embolism	Usually > 50 years, recent manipulation of aorta, retinal plaques, subcutaneous nodules, palpable purpura, livedo reticularis, vasculopathy, hypertension	Often normal, eosinophiluria, rarely casts	Eosinophilia, hypocomplementemia, skin biopsy, renal biopsy
Renal vein thrombosis	Evidence of nephroic syndrome or pulmonary embolism, flank pain	Proteinuria, hematuria	Inferior venacavogram and selective renal venogram; Doppler flow study; MRI

Diseases of small vessels and glomeruli			
Glomerulo-nephritis or vasculitis	Compatible clinical history (for example, recent infection) sinusitis, lung hemorrhage, rash or skin ulcers, arthralgias, hypertension, edema	RBC or granular casts, RBC's, WBC's, mild proteinuria	Low C3, antineuterophil cytoplasmic antibodies, anti-glomerular basemaent membrane antinuclear antibodies, anti-streptolysin O, anti-DNase, cryoglobulins, renal biopsy.
HUS or TTP	Compatible clinical history (for example, recent gastrointestinal infection, cyclosporine, anovulants), fever, pallor, ecchymoses, neurologic abnormalities	May be normal, RBC's, mild proteinuria, rarely RBC's or granular casts	Anemia, thrombocytopenia, schistocytes on blood smear, increased lactate dehydrogenase, renal biopsy
Malignant hypertension	Severe hypertension with headaches, cardiac failure, retinopathy, neurologic dysfunction, papilledema	RBC's, RBC casts, proteinuria	LVH by echocardiography or electrocardiography, resolution of ARF with control of blood pressure

ARF mediated by ischemia or toxins (ATN)			
Ischemia	Recent hemorrhage, hypotension (for example, cardiac arrest), surgery	Muddy brown granular or tubule epithelial cell casts, FE_{NA}> 1%, U_{NA} >20mEq/L, SG =1.010	Clinical assessment and urinalysis usually sufficient for diagnosis
Exogenous toxins	Recent radiocontrast study, nephrotoxic antibiotics or anticancer agents often coexistent with volume depletion, sepsis, or chronic renal insufficieny	Muddy brown granular or tubule epithelial cell casts, FE_{NA}> 1%, U_{NA} >20mEq/L, SG =1.010	Clinical assessment and urinalysis usually sufficient for diagnosis
Endogenous toxins	History suggestive of rabdomyolysis (seizures, coma, ethanol abuse, trauma)	Urine supernatant tests positive for heme	Hyperkalemia, hyperphosphatemia, hypocalcemia, increased myloglobin, creatinine kinase MM, uric acid
	History suggestive of hemolysis (blood transfusion)	Urine supernatant pink and positive for heme	Hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, pink plasma positive for hemoglobin
	History suggestive of tumor lysis (recent chemotherapy), myeloma (bone pain), or ethylene glycol ingestion	Urate crystals, dipstick- negative proteinuria, oxalate crystals, respectively	Hyperuricemia, hyperkalemia, hyperphosphatemia (for tumorlysis); circulating or urinary monoclonal spike (for myeloma); toxicology screen, acidosis, osmolal gap (ethylene glycol)

Acute disease of the tubulointerstium			
Allergic interstitial nephritis	Recent ingestion of drug and fever, rash, or arthralgias	WBC casts, WBC's (frequently eosinophiluria), rarely RBC casts, proteinuria (occassionaly nephrotic)	Systemic eosinophilia, skin biopsy of rash area (leukocytoclastic vasculitis), renal biopsy
Acute bacterial pyelo-nephritis	Flank pain and tenderness, toxic state, febrile	Leucocytes, proteinuria, RBC's, bacteria	Urine and blood culture
Postrenal azotemia	Abdominal or flank pain, palpable bladder	Frequently normal, hematuria if stones, hemorrhage, malignancy or prostatic hypertrophy	Plain film, renal ultrasonography intravenous pyelography, retrograde or anterograde pyelography, computed tomography

URINALYSIS

Assessment of urine is a mandatory and inexpensive tool in evaluation of ARF. Anuria suggests complete urinary tract obstruction but may complicate severe cases of prerenal or intrinsic renal ARF. Wide fluctuations in urine output suggests, intermittent obstruction while patients with partial urinary tract obstruction may present with polyuria caused by secondary impairment of urine concentrating mechanisms.

Table 6: Urine sediment in the differential diagnosis of acute renal failure

Normal or few red blood cells or white blood cells
Prerenal azotemia
Arterial thrombosis or embolism
Preglomerular vasculitis
HUS or TTP
Scleroderma crisis
Postrenal azotemia
Granular casts
ATN (muddy brown)
Glomerulonephritis or vasculitis
Interstitial nephritis
Red blood cell casts
Glomerulonephritis or vasculitis
Malignant hypertension
Rarely interstitial nephritis
White blood cell casts
Acute interstitial nephritis or exudative glomerulonephritis
Severe pyelonephritis
Marked leukemia or lymphomatous infiltration
Eosinophiluria (> 5%)
Allergic interstitial nephritis (antibiotics > NSAIDs)
Atheroembolic disease
Crystalluria
Acute urate nephropathy
Calcium oxalate (ethylene glycol toxicity)
Acyclovir
Sulfonamides
Radiocontrast agents
Bilirubinuria
Hepatorenal syndrome

RENAL FAILURE INDICES

Analysis of urine and blood biochemistry is particularly useful for distinguishing prerenal from ischemic or nephrotoxic intrinsic renal ARF. The fractional excretion of sodium (FE_{Na}) is most sensitive index for this purpose.[60] The renal failure index provides comparable information, since clinical variations in serum sodium concentration are relatively small.

FE_{Na} may be > 1% in prerenal ARF, if patients are receiving diuretics or have bicarbonaturia, preexisting CRF complicated by salt wasting or adrenal insufficiency. In contrast, FE_{Na} < 1% in ~15% of patients with non-oliguric ischemic or nephrotoxic ARF due to patchy injury to tubular epithelium with preservation of reabsorptive function in some areas. It is also due to urinary tract obstruction, glomerulonephritis and vascular disease.[61]

Table 7: Urine indices used in the differential diagnosis of prerenal and ischemic intrinsic renal azotemia[39]

Diagnostic Index	Prerenal Azotemia	Ischemic Intrinsic Azotemia
Fractional excretion of Na^+ (%), $(U_{Na} \times P_{cr}) / (P_{Na} \times U_{cr}) \times 100$	< 1	> 1
Urinary Na^+ concentration (mEq/L)	< 10	> 20
Urinary creatinine/ plasma creatinine ratio	> 40	< 20
Urinary urea nitrogen / plasma urea nitrogen ratio	> 8	< 3
Urine specific gravity	> 1.018	< 1.012
Urine osmolality (mOsm/kg H_2O)	> 500	< 300
Plasma BUN/ creatinine ratio	> 20	< 10-15
Renal failure index, $U_{Na} / U_{cr} / P_{cr}$	< 1	> 1
Urine sediment	Hyaline casts	Muddy brown granular casts

Table 8: Imaging procedures in the diagnosis of acute renal failure

Procedure	Purpose
Plain film of the abdomen and/or tomography	For kidney size, calcification, calculi, abnormal gas collection
Ultrasonography (screening modality of choice)	For kidney size, function, cortical thickness, corticomedullary differentiation, hydronephrosis, mass.
Intravenous pyelography	Best avoided (contrast nephropathy)
Retrograde and antegrade pyelography	For localization and release of obstruction.
Radionuclide scans (lacks specificity)	For assessing blood flow, glomerular filtration, and infiltration by inflammatory cells.
Doppler USG, MR flow imaging, spiral CT, contrast angiography	For patency of renal artery and vein, suspected vascular obstruction.

Indication for kidney biopsy in acute renal failure

- Patients in whom prerenal and postrenal failure have been excluded and the cause of intrinsic renal azotemia is unclear
- ARF associated with glomerular nephritis, vasculitis, HUS, TTP, allergic interstitial nephritis and acute allograft rejection that may respond to specific therapy.

- Prolonged acute renal failure

COMPLICATIONS OF ACUTE RENAL FAILURE

1. Intravascular volume overload is an almost inevitable consequence of diminished salt and water overload and may present as life threatening pulmonary edema.[39]
2. Hypertension is unusual in ATN and should suggest hypertensive nephrosclerosis, glomerulonephritis, renal artery stenosis.
3. Hyperkalemia is a potentially life threatening complication of ARF. Serum K^+ rises by 0.5 mEq/L/d in oliguric and anuric patients. Coexistent metabolic acidosis may exacerebrate hyperkalemia. Severe hyperkalemia at the time of diagnosis suggests rhabdomyolysis, hemolysis and tumor lysis syndrome. Mild hyperkalemia is usually asymptomatic.
4. ARF is commonly complicated by metabolic acidosis, typically with widening of serum anion gap.
5. Mild hyperphosphatemia (5 to 10 mg/dL) is common consequence of ARF and may be severe (10 to 20 mg/dL) in highly catabolic patients or when associated with rhabdomyolysis, hemolysis and tumor lysis syndrome.
6. Hypocalcemia is due to tissue resistance to the actions of parathyroid hormone and reduced levels of 1, 25-dihydroxyvitamin D and Ca^{2+} sequestration in injured tissues.[62]
7. Anemia develops rapidly in ARF and is usually mild and multifactorial in origin, due to inhibition of erythropoiesis, hemolysis, bleeding, hemodilution, and reduced red blood cell survival time.

8. Prolongation of bleeding time and leukocytosis are also common. The former may result from mild thrombocytopenia, platelet dysfunction and or clotting factors abnormalities. Leucocytosis reflects sepsis stress response, and or other concurrent illness.[63]
9. Infection is the most common and serious complication of ARF, occurring in 50 -90% of cases and accounting for upto 75% of deaths.[21,64]
10. Cardiopulmonary complications of ARF include arrhythmias, myocardial infarction, pericarditis and pericardial effusion, pulmonary embolism.
11. Mild gastrointestinal bleeding is common (10 to 30%) and is usually due to stress ulceration of gastric or small intestinal mucosa.
12. A vigorous diuresis may complicate the recovery phase of ARF and precipate intravascular volume depletion and a delay in recovery of renal function.
13. Malnutrition remains one of the most frustrating and troublesome complications of ARF and majority have net protein breakdown more than 200 g/d in catabolic subjects. It may reflect –
    a) inability to eat or loss of appetite
    b) sepsis, rhabdomyolysis, trauma
    c) nutrient losses in drainage fluids or dialyste
    d) increased breakdown and reduced synthesis of muscle protein and increased hepatic gluconeogenesis
    e) inadequate nutritional support.

MANAGEMENT OF HAARF

Preventive Measures:

As there are no specific therapies for ischemic or nephrotoxic ARF, prevention is of paramount importance. Aggressive restoration of intravascular volume has been shown to reduce dramatically incidence of ischemic ARF after major surgery, trauma, burns or cholera. The incidence of nephrotoxic ARF can be reduced by tailoring dosage of nephrotoxic drugs to body size and GFR.

Allopurinol and forced alkaline diuresis are useful prophylactic measures in patients at high risk for acute urate nephropathy (cancer chemotherapy). Forced alkaline diuresis may prevent or attenuate ARF in patients receiving high dose methotrexate or suffering from rhabdomyolysis. N-acetylcysteine limits acetaminophen induced renal injury if given within 24 hours of ingestion. Ethanol inhibits ethylene glycol intoxication. Because the risk and the mortality of ARF are high in critically ill patients,[65] prevention is the best therapy. The most common risk factor is extracellular volume depletion. Volume expansion can minimize the risk of ARF from radiographic contrast agents, cisplatin, and NSAID's. Mannitol appears to at least partially abrogate the ARF caused by rhabdomyolisis and cisplatin but not that caused by contrast agents. Limiting the dose and simultaneous exposure appears important in avoiding contrast, aminoglycoside, and cisplatin toxicity. Alkali may limit the nephrotoxicity of myoglobinuria and uric acid. Allopurinol should be used before chemotherapy, whenever tumor lysis is anticipated. Adjusting dosing interval for changes in Cr is important to prevent aminoglycoside toxicity. Correcting hypokalemia and

expanding ECV are also helpful. Positive end-expiratory pressure (PEEP), as well as high intrathoracic pressure associated with mechanical ventilator support, may compromise cardiac output and renal perfusion. If possible, PEEP should be minimized and ECV should be expanded in high risk patients. There are animal data to suggest that hyperalimentation may increase the risk of ARF. However, the benefits of nutritional support seem to far outweigh this risk.

It appears to improve renal tubular cell regeneration and survival in patients, particularly those with multiple complications. Whenever possible, enteral hyperalimentation is preferred. Certain drugs have been proposed to decrease renal injury in high risk patients, these include atrial natriuretic peptide (ANP), calcium channel blockers, prostaglandin analogues, endothelial receptor antagonist, alpha adrenoreceptors blockers, and insulin like growth factors; Though many of these are beneficial in experimental models of ATN, they have failed to confer consistent benefits in humans.

Acute renal failure oliguric versus nonoliguric

Converting oliguria to nonoliguria appears helpful. Nonoliguric patients have fewer complications, a decreased dialysis requirement, and in some studies improved survival.[50] Conversion to nonoliguria can often be accomplished by repleting intravascular volume (if deficient) and using high-dose loop diuretics (for example,, 200 mg iv of furosemide or continous infusions at a rate of 10 to 40 mg/hr). The diuretic may have the additional advantage of decreasing tubular cell metabolic activity, thus lessening the oxygen requirement. A renal vasodilatory dose of dopamine (0.5 to 2.0 mg/Kg/min) is sometimes helpful in

stimulating urine volume. However, in a recent placebo controlled trial in critically ill patients, low dose dopamine failed to improve serum creatinine, dialysis requirement, ICU or hospital days.[66]

Neither furosemide nor dopamine have any utility as prophylaxis prior to major surgery.

Unfortunately, there are no specific therapies for most causes of ARF. Steroids and ciclophosphamides are indicated for polyarteritis, Wegeners disease, or diffuse proliferative lupus nephritis. Plasmapheresis is indicated for Goodpasture's syndrome, TTP, and myeloma of the kidney. Antibiotics are useful for sepsis and infectious forms of glomerulonephritis.

Prerenal azotemia

It is rapidly reversible on restoration of renal perfusion. Hypovolemia caused by hemorrhage is ideally corrected with packed red cells and isotonic saline is appropriate for mild to moderate hemorrhage or plasma loss. A recent RCT comparing crystalloid with colloid replacement for resuscitation in critically ill patients concluded that routine use for colloids maybe associated with adverse outcome and is not justified.[67] Urinary and gastrointestinal losses are usually corrected with hypotonic solutions (for example, 0.45% saline) although isotonic saline maybe appropriate in severe cases.

Cardiac failure may require aggressive management with positive inotropes, preload and after load reducing agents, antiarrythmic agents and IABP.

Fluid management maybe particularly challenging in patients with ARF and cirrhosis. It is important to distinguish between hepatorenal syndrome and reversible ARF due to true or "effective" hypovolemia caused by overzealous use of diuretics or sepsis. Hypovolemia can be accessed by fluid challenge and titrated by JVP, CVP, PCWP, abdominal girth, and urine output. Large volume of ascitic fluid can be drained by paracentesis without deterioration of renal function if I.V albumin is administered simultaneously.[68] Shunting of ascitic fluid (peritoneojugular shunt, Leveen or Denver shunts) is alternative approach in refractory cases though RCT have shown no effect on overall survival. Recent evidence has emerged that suggests that insertion of transjugular portosystemic shunt (TIPS) maybe associated with an improvement in renal outcome.

Intrinsic renal ARF

Optimization of cardiovascular function and intravascular volume is single most important maneuver in management of acute intrinsic azotemia. Renal dose dopamine increases renal blood flow and albeit to less extent GFR. It has not been demonstrated to prevent or alter course of ischemic or nephrotoxic ATN in trials. Rather, dopamine even at lower doses is potentially toxic in critically ill patients and can induce tachyarrythmias and myocardial ischemia among other complications. Thus routine administration of dopamine to patients with oliguric ARF is not justified based on clinical evidence.[69]

Atrial natriuretic peptide (ANP) amino acid polypeptide synthesized in cardiac atrial muscle augments GFR by triggering afferent arteriolar vasodilation and inhibition of sodium transport and lowers oxygen requirement in several

nephron segments. Large multicentre trials have failed to show clinically significant dialysis free survival or mortality in ATN.[29]

Administration of high dose intravenous diuretics may minimize fluid overload, there is no indication that it alters mortality or dialysis free survival.[70] Administration of mannitol in severely oliguric or anuric patients, may trigger expansion of intravascular volume and pulmonary edema, and severe hyponatremia.

In humans prostaglandin analogues, antioxidants, insulin like growth factors I, antibodies against leucocyte adhesion molecules have either failed to confer consistent benefit or proved ineffective.

ARF due to acute glomerulonephritis or vasculitis may respond to corticosteroids, alkylating agents, plasmapheresis depending on primary cause. Glucocorticoids also hasten remission in some cases of allergic interstitial nephritis.

Aggressive control of systemic arterial pressure is of paramount importance in limiting renal injury in malignant hypertensive nephrosclerosis, toxemia of pregnancy and other vascular diseases.

Postrenal ARF

Urethral or bladder neck obstruction is usually relieved temporarily by transurethral or suprapubic placement of bladder catheter, which provides temporary relief while obstructing lesion, is identified and treated definitively. Similarly, ureteric obstruction may be treated initially by percutaneous

catheterization of dialated renal pelvis or ureter. Indeed, obstructing lesions can often be removed percutaneously (for example, calculs, sloughed papilla) or bypassed by insertion of a ureteric stent (e.g carcinoma). Most patients experience an appropriate diuresis for several days following relief of obstruction. ~ 5% of patients develop salt wasting nephropathy that may require administration of saline to maintain blood pressure.

Table 9: Supportive management of intrinsic acute renal failure

Complication	Treatment
Intravascular volume overload	Restriction of salt (1-2 g/d) and water (usually < 1 L/d)
	Diuretics (usually loop blockers ± thiazide)
	Ultrafiltration or dialysis
Hyponatremia	Restriction of free water intake (oral and dextrose-containing solutions)
Hyperkalemia	Restriction of dietary potassium intake
	Eliminate K^+ supplements and K^+ - sparing diuretics
	K^+ - binding ion exchange resins
	Glucose (50 mL of 50% dextrose) and insulin (10 U regular)
	Sodium bicarbonate (usually 50-100 mEq)
	Calcium gluconate (10 mL of 10% solution over 5min)
	Dialysis
Metabolic acidosis	Restriction of dietary protein
	Sodium bicarbonate (maintain serum HCO_3^- > 15mEq/L)
	Dialysis
Hyperphosphatemia	Restriction of dietary phosphate intake
	PO_4^{3-} -binding agents (calcium carbonate, aluminium oxide)
Hypocalcemia	Calcium carbonate (if symptomatic or if sodium bicarbonate to be administered)
	Calcium gluconate (10-20 mL of 10% solution)
Hypermagnesemia	Discontinue Mg^{2+} - containing antacids
Hyperuricemia	Treatment usually not necessary (if < 15 mg/dL)
Nutrition	Restriction of dietary protein (~ 0.5 g/kg/d)
	Carbohydrate (~100 g/d)
	Entral or parental nutrition (if recovery prolonged)
Drug dosage	Adjust doses for degree of renal impairment
Indication for dialysis	Clinical evidence (symptoms or signs) of uremia
	Intractable intrvascular volume overload
	Hyperkalemia or severe acidosis resistant to conservative Measures
	? Prophylactic dialysis when BUN > 100-150 mg/dL or creatinine > 8-10 mg/dL

INDICATIONS AND MODALITIES OF DIALYSIS

Studies suggesting that early dialysis therapy, improved prognosis for patients with ARF have not been confirmed.[71] It is unclear whether the choice of dialytic modality or the intensity of dialysis favourably affects outcome.

Indeed, early and unnecessary hemodialysis may potentially exacerbate renal hypoperfusion because transient hypotension is common complication of this treatment and leucocytes activated on exposure to dialysis membranes may potentially aggravate ischemic renal injury.

Potential indications for Continous Renal replacement Therapy in the ICU are Non Obstructive Oliguria or Anuria**,** Severe acidemia (ph < 7.1) due to metabolic acidosis**,** Hyperkalemia (plasma K^+ > 6.5 mmol/L or rapidly raising) **,**Azotemia (BUN>80 mg/dl), Suspected uraemic organ involvement (pericarditis, encephalopathy, neuropathy, myopathy)**,** Progressive severe dysnatremia (Na^+ > 160 or <115 mmol/L)**,** Clinically significant organ edema (especially Lung)**,** Coagulopathy requiring large amounts of blood products in patients with or at risk of pulmonary edema.

Dialysis modalities in the intensive care unit

Intermittent hemodialysis has been the standard therapy for many years because it is efficient, widely available, and generally well tolerated. However hemodialysis is not without potential complications, bleeding (due to heparinization) and hypotension being the most severe. The bleeding can usually be avoided by using citrate as an alternative anticoagulant.

Hypotension often can be modified by fluid removal (ultrafiltration). However, the hypotension of hemodialysis is, at least in part, due to the use of bioincompatible (cellulose) dialysis membranes. Complement activation and alterations in immune function are observed when hemodialysis is performed with these dialyzers. Recent studies have established that hypotension and complement activation can be reduced by the use of more compatible polysulfone, polyacrylonitrile or polymethylmethacrylate dialysis membranes..

Peritoneal Dialysis requires no anticoagulation, and its slow continuous ultrafiltration rates are well suited to patients with baseline hypotension and or poor cardiac output. However, its utility is limited after abdominal surgery and in severe catabolic patients because of relatively slow solute removal. Continuous extracorporeal techniques for solute and fluid removal are valuable in hemodynamically unstable patients, particularly those with multiple organ failure. During the past decade, a number of advances have been made in the field of renal replacement therapy. Clinicians have gained a better appreciation of the need for early and aggressive management of patients with renal failure in the ICU.[71]

Although a number of treatment modalities exist, there seems to be a continued controversy about selection of the most appropriate modality for each individual patient. Appropriate modality selection requires an understanding of the clinical spectrum of renal failure in the ICU. Uncomplicated renal failure refers to an acute and transient decline in glomerular filtration rate without clinically apparent complications. Dialytic support often is not required in

patients with uncomplicated ARF or may be performed for a single indication, such as hyperkalemia.

In complicated ARF, however, multiple metabolic and volume status pertubations are present, the patient is often oliguric, and the renal failure may be present in association with multiorgan failure. The threshold for initiation of dialysis and the choice of dialytic modality differ depending on the associated complications and comorbid conditions. Many nephrologists avoid dialysis initiation for as long as possible and the reasons are because dialysis procedure itself has associated risks (hypotension, arrythmias, vascular access placement) and the concern that dialysis may delay recovery of renal function.[72,73] In the critically ill patient ARF usually does not occur in isolation from other organ system dysfunction and therefore providing dialysis can be viewed as a form of renal support for multi-organ dysfunction rather than renal replacement. In the presence of oliguric renal failure, administration of larges volume of fluid to patients with MOF may lead to impaired oxygenation. In such a setting, early intervention with extracorporeal therapies for management of fluid balance significantly may impact the function of other organs, even in the absence of traditional indices of renal failure such as marked azotemia.

Table 10: Renal replacement therapies

Intermittent	Continuous
Hemodialysis (IHD)	Hemodialysis (CHD)
Ultrafiltration (IUF)	Ultrafiltration (SCUF)
Peritoneal (IPD)	Peritoneal (CPD)
Sustained low efficiency dialysis (SLED)	Hemofiltration (CAVH, CVVH)
	Hemodiafiltration (CA/CVVHDF)

Peritoneal Dialysis

With the development of intermittent hemodialysis, and more latterly the slow continuous blood purification therapies, there has been decline in the use of peritoneal dialysis in acute setting. It is still used in treatment of ARF in regions where access to acute intermittent or slow continuous hemodialysis is not possible.

Table 11: Advantages and disadvantages of peritoneal dialysis[72]

Advantage	Disadvantage
No angioaccess	Risk of visceral injury at catheter insertion
Technically simple	Less predictable ultrafiltration / solute control
No anticoagulation	Risk of peritonitis
Reduced risk of disequilibrium	Difficult in post abdominal surgery
Haemodynamic stability	Hyperglycemia
Clearance, toxins and ultrafiltration of excess intravascular volume, generally adequate	Diaphragmatic splinting

Acute Intermittent Hemodialysis

It has been the mainstay of renal replacement therapy in ARF over past 30 years. Patients undergo dialysis for 3 to 4 hours daily or on alternate days depending on their catabolic state.

The choice of membrane used during dialysis may have an effect on outcome. Several trials indicate that maintenance phase of ATN is significantly shorter with the use of more biocompatible synthetic dialysis membranes (for example, polysulphone, polyacrylonitrile) because of less activation of blood complement leucocytes and other mediator systems.[74]

Anticoagulation with heparin is the standard method for preventing thrombosis of the extracorporeal circuit during acute intermittent dialysis. Heparin free dialysis can be performed in patients at high risk of heamorrhagic complications.

Major complications of acute intermittent hemodialysis

1. Rapid shifts in plasma volume and solute composition.
2. Angioaccess procedure
3. Necessity for anticoagulation
4. Dialysis membrane incompatability
5. Intra-dialytic hypotension[75]
6. Hypotension may be problematic particularly in critically ill patients with ATN and concurrent sepsis, hypoalbuminemia, malnutrition or large third space losses.
7. Dialysis disequilibrium syndrome it typically occurs after first dialysis in very uremic patients. It is a self limited condition characterized by nausea, vomiting, headache, altered consciousness, and rarely seizures or coma. The incidence of this complication has fallen in recent years.

Slow Continuous Hemofiltration And Hemodialysis

Continuous renal replacement therapies (CRRT) are alternatives to conventional hemodialysis techniques for treatment of ARF. They are particularly valuable techniques in patients in whom intermittent hemodialysis fails to control hypervolemia or uremia and for those who do not tolerate intermittent hemodialysis and in whom peritoneal dialysis is not possible.[76]

Advantages:

1. Relative simplicity of operation.
2. Ability to remove large volumes of fluid over a prolonged period with minimal hemodynamic compromise.
3. Capacity to control uremia and electrolyte and acid-base abnormalities with minimal perturbation of plasma osmolality.

Continuous Venovenous Hemodialysis (CVVHD)

It is the technique favored by most centers. Angioaccess is achieved by double lumen venous catheter as a blood pump generates ultrafiltration pressure across dialysis membrane. In more simple techniques of continuous arteriovenous hemofiltration (CAVH) and continuous venovenous hemofiltration (CVVH). The dialysis step is eliminated and ultrafiltrate of plasma is removed across the dialysis membrane and replaced by a crystalloid solution.

Slow Continuous Ultrafiltration (SCUF)

Slow continuous ultrafiltration (SCUF) is a similar technique except that the dialysis flow rate is set at zero and no replacement solution is administered. This technique yields pure ultrafiltration and is typically used in the patient with marked volume overload, as a result of fluid intake and heart failure or capillary leak syndrome.

Disadvantages of continuous hemodialysis techniques

1. Prolonged immobilization in bed
2. Systemic anticoagulation,

3. Arterial canulation (CAVH)
4. Prolonged exposure of blood to synthetic, albeit relatively biocompatible, dialysis membranes.

Several retrospective studies suggest an improvement in outcome in critically ill patients treated with continuous forms of renal replacement present, but to date no prospective data exist showing decreased mortality in patients treated with these newer techniques.[77]

Prognosis and outcome of hospital acquired acute renal failure

ARF is a complex clinical entity with high morbidity and mortality. In 1940's the mortality was as high as 90%, with date occurring from pulmonary edema, hyperkalemia and uremia. With introduction of antibiotics, availability of effective treatment for hyperkalemia and development of dialysis procedures, the mortality declined dramatically to 50 – 60% by the early 1970's.

The crude mortality has improved surprisingly little since that time. The mortality rate among patients with acute intrinsic renal azotemia ~ 50% and has changed little over the past three decades.[78] This finding may be explained in parts by changes in patient population as reflected in an lower frequency of patients with good prognosis, a greater proportion of elderly patients, an increase in severity and number of failing organs, and delayed occurrence of acute renal failure in patients whose survival is prolonged.[79,80] This lack of improvement in outcome, despite significant advances in supportive care, maybe more apparent than real and reflect a reduction in percentage of isolated ARF combined with an increase in ARF, complicating the multiorgan dysfunction syndrome.[78,81]

ARF with no organ damage is rare in ICU patient. Failure of one or more organs is seen in about 90% of patients who have ARF, and most ARF follows other organ failure. The incidence of ARF following cardiac failure is 44% and the incidence following respiratory failure is 27%, while kidney is the first organ to fail in only 22%. The mortality rate is highest (70%) when organ failure preceeds ARF, rather than when it occurs subsequently (55%).[82]

Factors associated with poor prognosis include oliguria (< 400 mL/d) at time of presentation before administration of diuretics and rise in serum creatinine value of greater than 3 mg/dL, probably reflecting more severe renal injury and or comorbid disease.[39] Hypotension, sepsis, ventilatory support for long duration, elderly patients with organ failure are independent factors for poor prognosis.[83,84]

Non-oliguric ARF carries a better prognosis than oliguric ARF, partly because management of fluid and electrolyte abnormalities is easier and possibly because this patients have a less severe renal insult.[85,86]

With appropriate supportive management of ARF, death is usually a consequence of primary disease that induced ARF and rarely of a direct complication per se. ARF is irreversible in approximately 5% of patient, usually as a consequence of complete cortical necrosis, and requires long term renal replacement therapy with dialysis or transplantation.[87]

METHODOLOGY

The present study was conducted in the Department of Medicine on 65 patients admitted to critical care units at Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum between the period from January 2008 to December 2008.

Study design

Cross-sectional study

Study Period

The study was conducted over a period of one year from January 2008 to December 2008.

Source of Data

Patients admitted at critical care units of KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum with HAARF.

Method of collection of data

Sample size

Sixty five (65) patients with HAARF admitted at critical care units of KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum

Sampling procedure

As there was no data available in KLES Dr. Prabhakar Kore Hospital and Medical Research records section regarding HAARF, all the patients presenting with HAARF during the study period were included.

Selection Criteria

Inclusion criteria

- Adult patients who had normal renal function on admission and developed renal failure (Serum creatinine ≥ 1.5 mg%) after 48 hours of admission.

Exclusion criteria

- Patients of ARF on admission.
- Known cases of CRF

Procedure

All the cases were evaluated by detailed medical history and physical examination. The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum.

All patients presenting with HAARF in the Department of Medicine or intensive care units at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre were screened for the eligibility. After evaluating the suitability as per inclusion and exclusion criteria, they were selected for the study and briefed

about the nature of the study, the procedures used and written informed consent was obtained (Annexure–I). The consented patients were enrolled in the present study. Further, descriptive data of the participants like name, age, sex, detailed history, were obtained by interviewing the participants or the patient attenders and recorded on predesigned and pretested proforma (Annexure-II). Detailed history, clinical examination, blood investigations were performed.

Investigations

- Hemogram (HB,TC,DC, ESR)
- Urine analysis
- Fractional excretion of sodium
- Urinary sodium
- Urinary osmolality
- Blood urea
- Serum creatinine
- Urine Creatinine
- Serum electrolytes
- Arterial blood gas analysis as and when required
- Blood sugar
- Electro cardiography
- Ultrasonography – KUB

Other investigations were done as and when required depending on the etiology.

Adult patients who developed renal failure (creatinine ≥ 1.5) anytime after 48 hours admission to ICU were included in the study, and referred to as HAARF.

Sixty five (65) patients of HAARF were evaluated after identification of the patient, a clinical and laboratory data were collected at admission and then on daily basis. Data was recorded included patient characteristics, underlying medical condition responsible for ICU admission, dialysis status, need for assited ventilation, inotropic support, total duration of hospital stay, and the final outcome.

All these patients were followed every daily basis for next 2 weeks in the hospital,a serial recording of urine output and serum creatinine was maintained, the different complication associated with ARF were specifically looked for and recorded.

Hemodialysis or peritoneal dialysis, and conservative treatment instituted according to standard clinical indication. These data were analysed to find out the association of risk factors with the outcome of HAARF patients

Statistical analysis

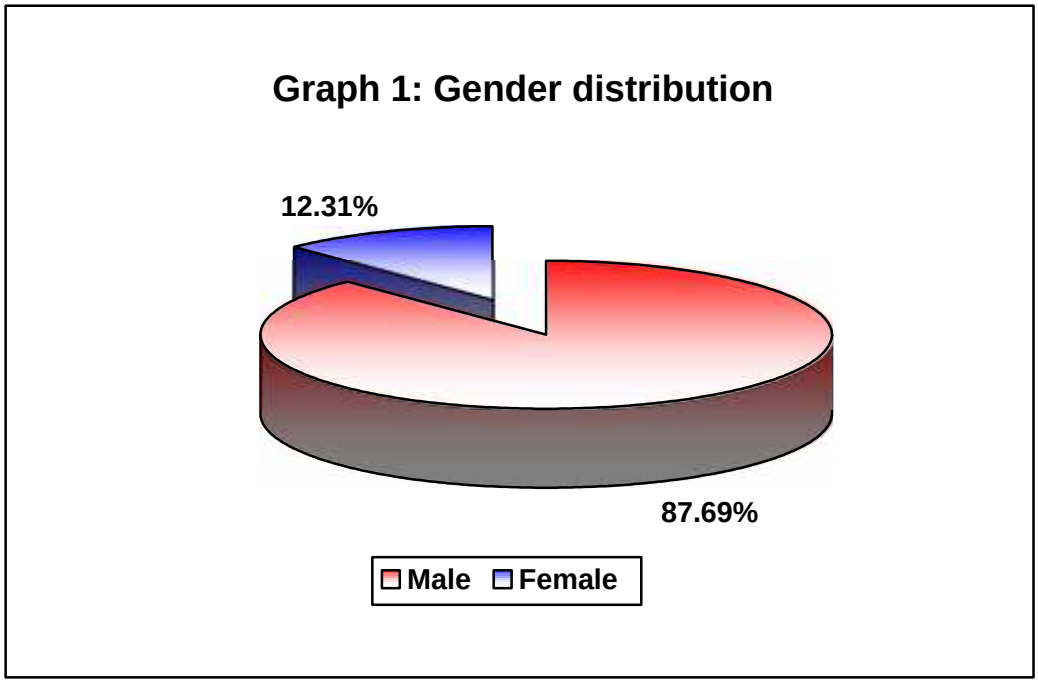
The results were tabulated and the data was analysed using rates, ratios and percentages of different clinical manifestations, signs, etiologies and diagnosis. Continuous variables are expressed in mean (SD), chi square test was applied for qualitative data and a p value < 0.05 was considered significant.

RESULTS

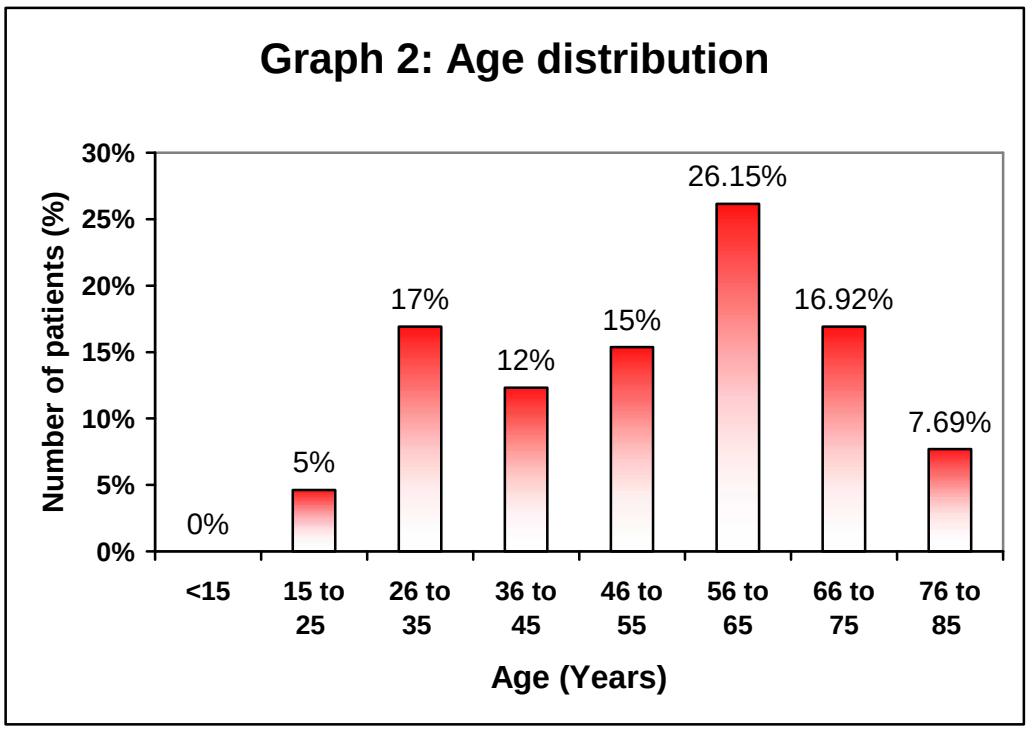
The present study was conducted in the Department of Medicine on 65 patients admitted to critical care units at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum between the period from January 2008 to December 2008. The findings and observation were recorded as below.

Table 12: Demographic characteristics of the study population

Demography		Patients	
		Number	**Percentage**
Gender	**Males**	57	87.69%
	Females	08	12.31%
	Total	65	100%
Age distribution	**< 15**	00	00%
	15 to 25	03	4.62%
	26 to 35	11	16.92%
	36 to 45	08	12.31%
	46 to 55	10	15.38%
	56 to 65	17	26.15%
	66 to 75	11	16.92%
	76 to 85	05	7.69%
	Total	65	100%



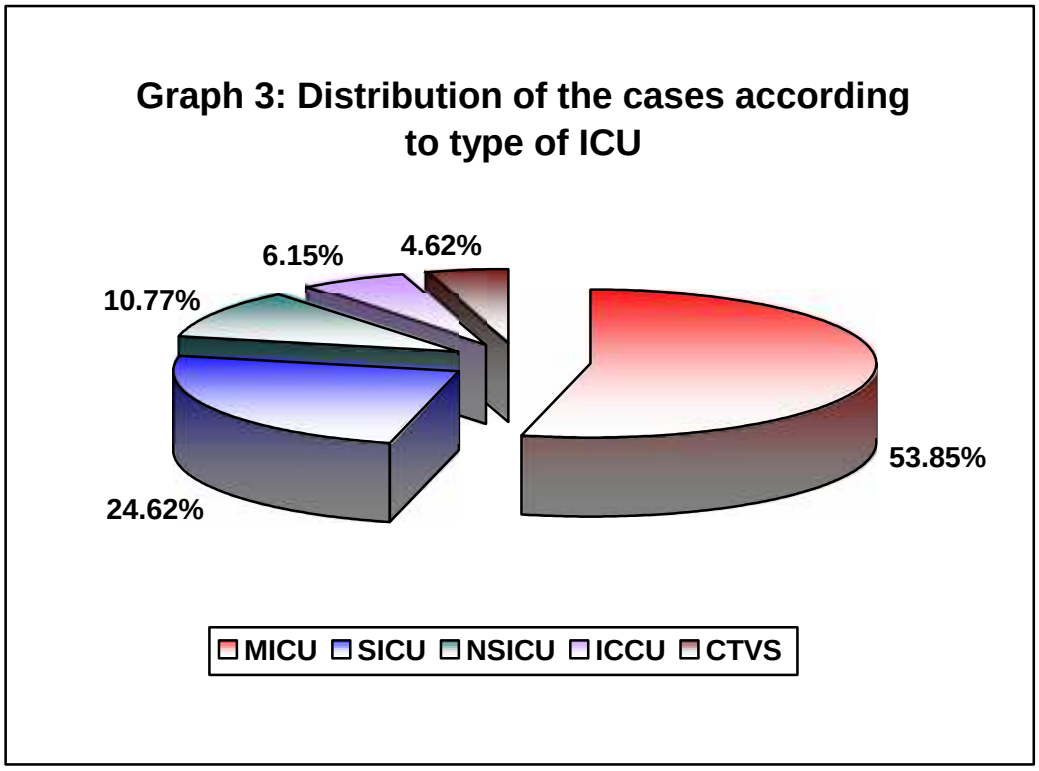
Out of 65 patients, 57 (87.69%) patients were males, and 08 (12.31%) patients are females. Male predominance was present.



The age group varied between 15 to 85 years, maximum number occurred between 56 to 65 years group. The mean age of the cases studied was 52.38 ± 17.02 years.

Table 13: Distribution of the cases according to type of ICU and the speciality

Distribution		Patients	
		Number	**Percentage**
Type of ICU (locality)	**MICU**	35	53.85%
	SICU	16	24.62%
	NSICU	07	10.77%
	ICCU	04	6.15%
	CTVS	03	4.62%
Speciality	**Medicine**	34	52.31%
	General Surgery	14	21.54%
	Neurosurgery	08	12.31%
	Cardiology	03	4.62%
	CTVS	03	4.62%
	Orthopaedics	02	3.08%
	Oncology	01	1.54%

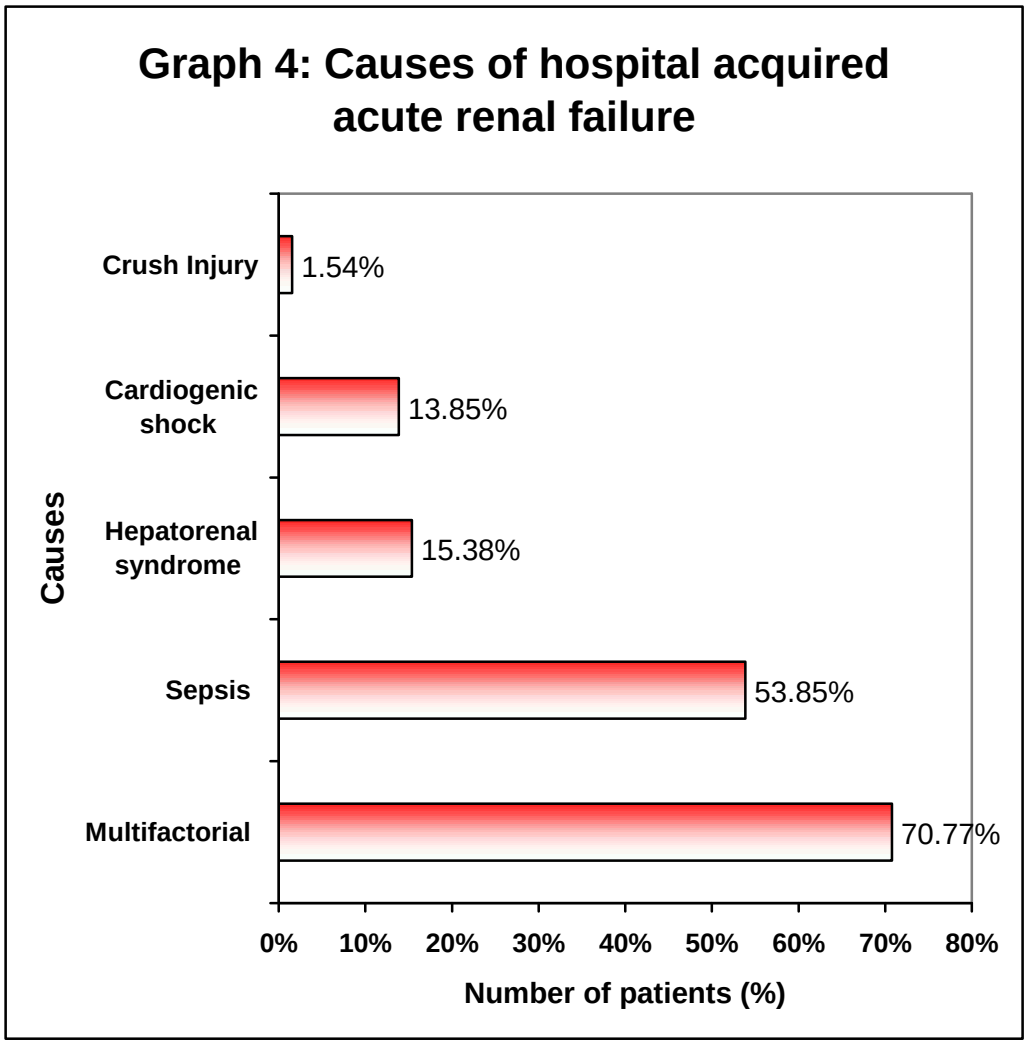


The maximum patients belonged to MICU 35 (53.85%), followed by SICU surgical (24.62%) and NSICU neurosurgical (10.77%), ICCU cardiology (6.15%) and CTVS (4.62%).

Most (52.31%) of the cases were from medical disorders followed by surgical, neurosurgical, cardiology and CTVS, orthopedics, oncology, 14 (21.54%), 08 (12.31), 3 (4.62%) 3 (4.62%), 02 (3%), 1 (1.5%) respectively.

Table 14: Distribution of patients according to causes of hospital acquired acute renal failure

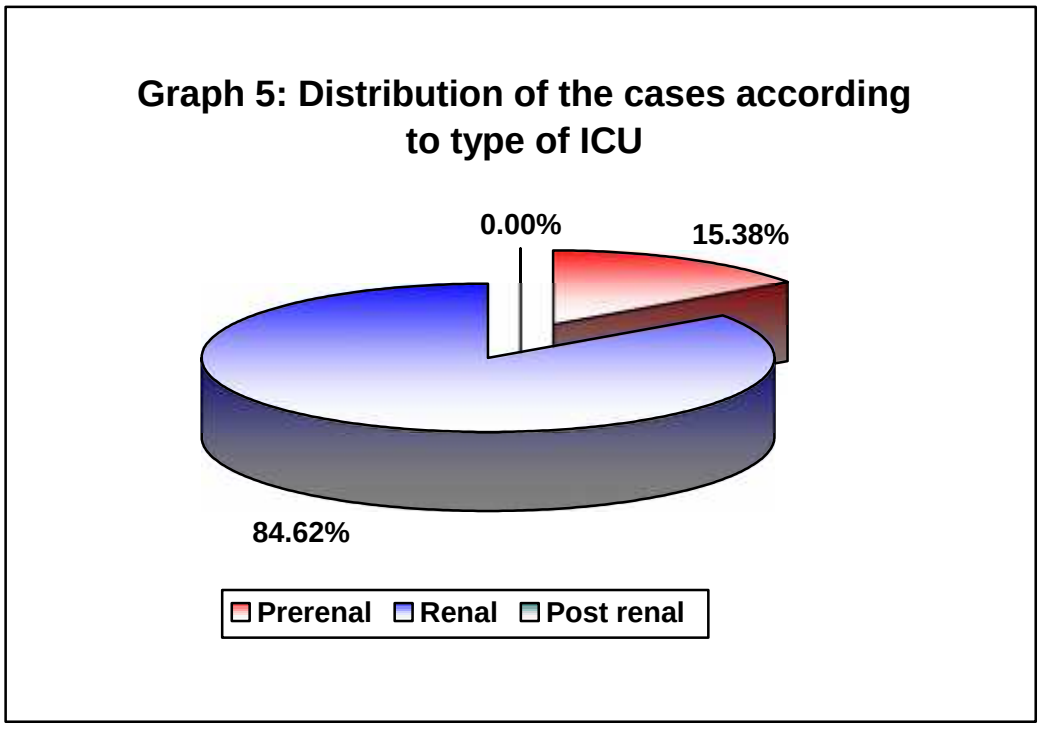
Causes	Patients	
	Number	Percentage
Multifactorial	46	70.77%
Sepsis	35	53.85%
Hepatorenal syndrome	10	15.38%
Cardiogenic shock	09	13.85%
Crush injury	01	1.54%



Most (70.77%) of the cases had multifactorial etiology as the causative risk factor followed by sepsis, hepatoenal syndrome and ischemia. Most common cause of HAARF was multifactorial etiology followed by SEPSIS.

Table 15: Distribution of patients according to Type of hospital acquired acute renal failure

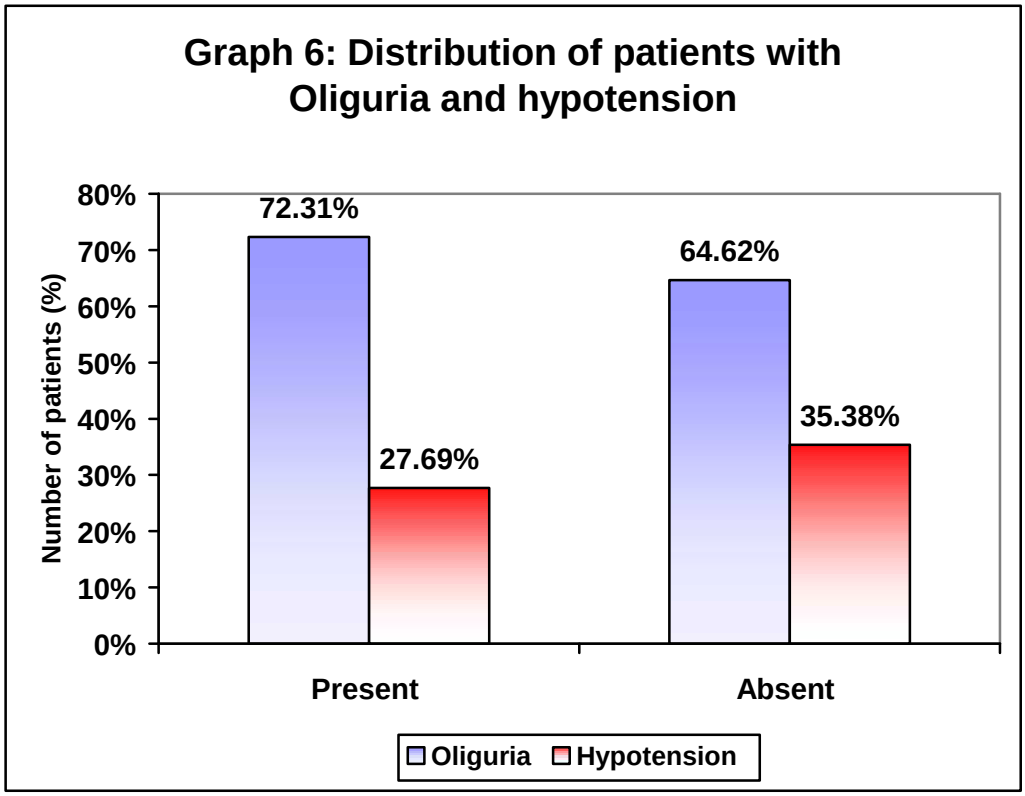
Type of HAARF	Patients	
	Number	Percentage
Prerenal	10	15.38%
Renal	55	84.62%
Post renal	00	0.0%
Total	65	100%



Intrarenal type of ARF (84.62%) was the most common type in HAARF followed by prerenal and none from pos renal ARF.

Table 16: Distribution of patients with oliguria and hypotension

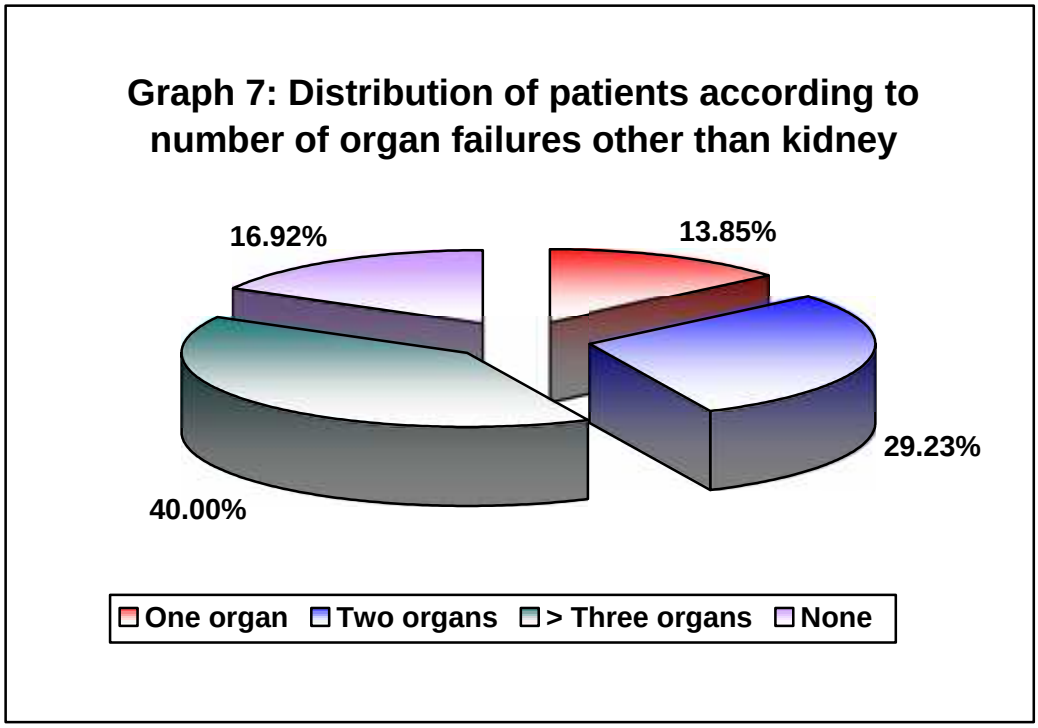
Status	Oliguria		Hypotension	
	Number	Percentage	Number	Percentage
Present	47	72.31%	42	64.62%
Not present	18	27.69%	23	35.38%
Total	65	100%	65	100%



Most (72.31%) of the patients had oliguria and 42 (64.62%) patients were hypotensive. Oliguria and hypotension were important risk factors in HAARF.

Table 17: Distribution of patients according to number of organ failures other than kidney

Causes	Patients	
	No.	**Percentage**
One organ	09	13.85%
Two organs	19	29.23%
≥ three organs (MOD)	26	40.00%
None	11	16.92%



26 (40%) cases had MOD, followed by 2 organs dysfunction (19 cases, 29.23%). 11 cases didn't have organ dysfunction. MOD was the most common occurrence in HAARF.

Table 18: Distribution of patients according to requirement of ionotropic support and assisted ventilation

	Ionotropic support		Assisted ventilation	
	Number	**Percentage**	**Number**	**Percentage**
Required	45	69.23%	40	61.5%
Not required	20	30.77%	25	38.46%
Total	65	100%	65	100%

45 (69.23%) patients required ionotropic support, 40 (61.5%) patients required assisted ventilation.

Table 19: Distribution of patients according to SIRS and sepsis

	SIRS		Sepsis	
	Number	**Percentage**	**Number**	**Percentage**
Present	54	83.08%	44	67.69%
Absent	11	16.92%	21	32.31%
Total	65	100%	65	100%

54 (83.08%) patients had SIRS and 44 (67.69%) had sepsis.

Table 20: Distribution of patients according to level of sensorium

Level of sensorium	Patients	
	Number	Percentage
Conscious	24	36.92%
Altered conscious	13	20.00%
Comatose	28	43.8%
Total	65	100%

Maximum (43.8%) patients were comatosed followed by 24 (36.92%) patients were conscious and 13 (20%) were in altered sensorium.

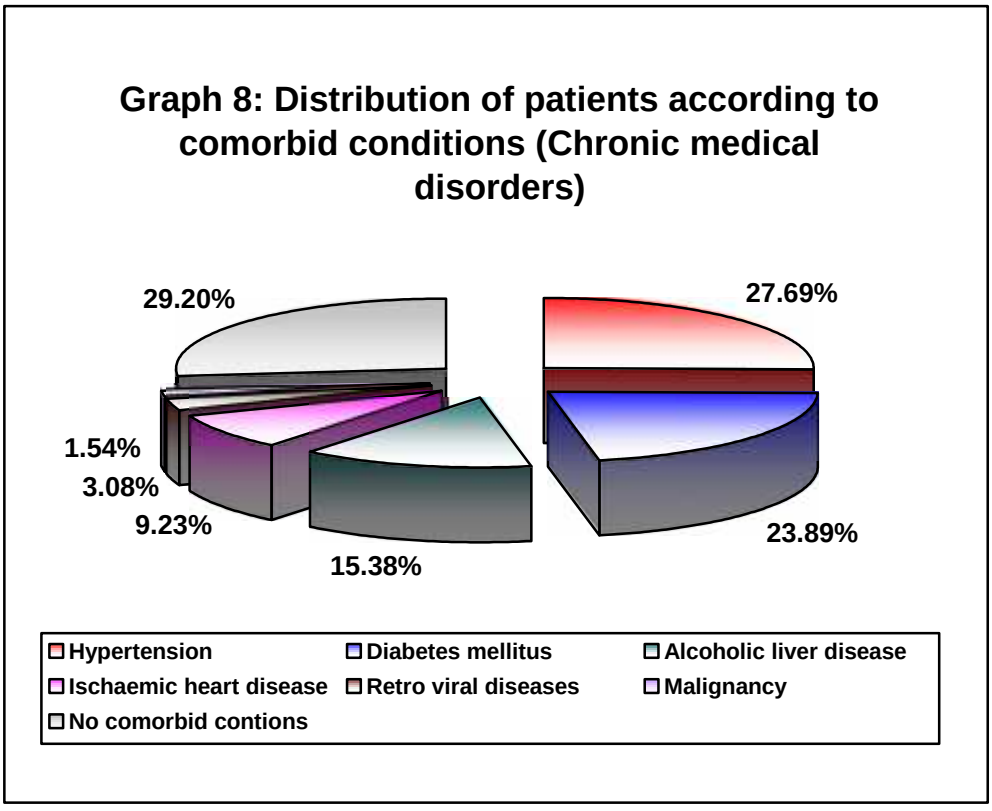
Table 21: Distribution of patients according to hyperkelemia and metabolic acidosis

	Hyperkelemia		Metabolic acidosis	
	Number	Percentage	Number	Percentage
Present	14	21.54%	25	38.46%
Not present	41	63.08%	40	61.54%
Total	65	100%	65	100%

Most (63.08%) of the cases didn't have hyperkelemia and 40 (61.5%) were not in metabolic acidosis. All the cases of HAARF may or may not have hyperkelimia and metabolic acidosis.

Table 22: Distribution of patients according to comorbid conditions (Chronic medical disorders)

Type	Patients	
	Number	Percentage
Hypertension	18	27.69%
Diabetes mellitus	15	23.08%
Alcoholic liver disease	10	15.38%
Ischaemic heart disease	06	9.23%
Retro viral diseases	02	3.08%
Malignancy	01	1.54%
No cormid conditions	19	29.2%
Total	65	100%



Most common comorbid condition was hypertension 18 (27.69%) cases, followed by diabetes mellitus 15 (23.08%) and the remaining cases (29.2%) had no comorbid conditions.

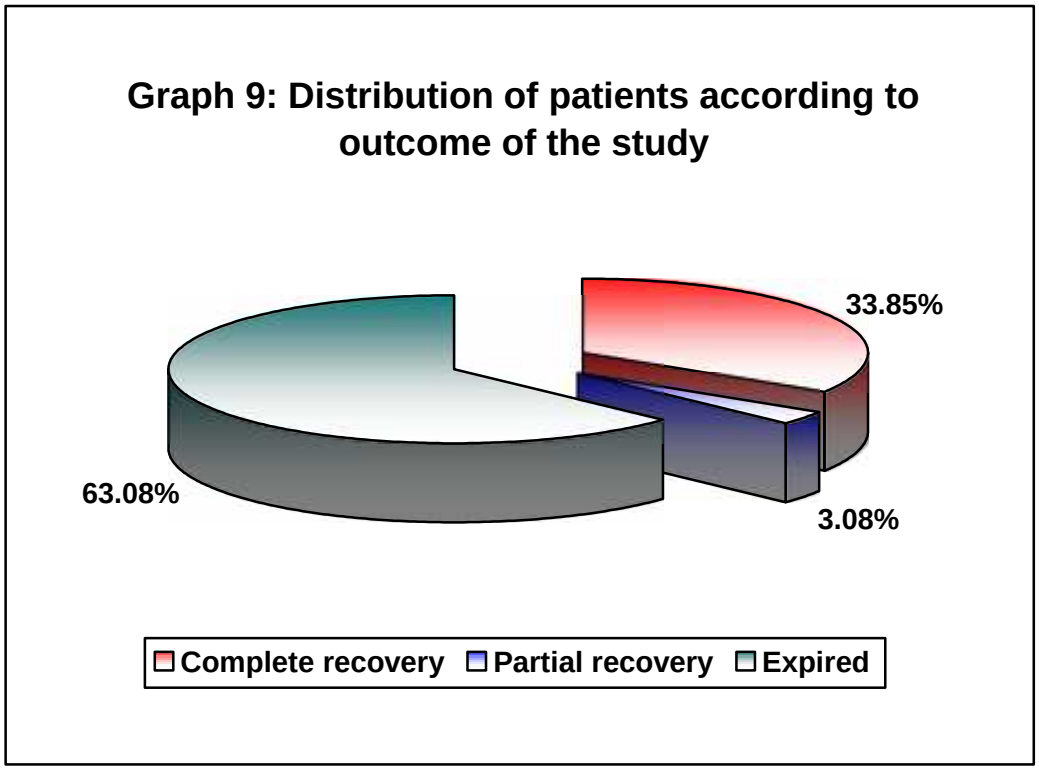
Table 23: Distribution of patients according to type of management

Type	Patients	
	Number	**Percentage**
Hemodialysis	05	7.69%
Peritoneal dialysis	05	7.69%
Conservative	55	84.62%
Total	65	100%

Out of total 65 cases, most (84.62%) of the patients were treated conservatively, 5 cases needed hemodialysis and 5 more cases were treated by peritoneal dialysis. Conservative management is the most common type of management inspite of high requirement of dialysis.

Table 24: Distribution of patients according to outcome of the study

Outcome	Patients	
	Number	Percentage
Complete recovery	22	33.85%
Partial recovery	02	3.08%
Expired	41	63.08%
Total	65	100%



Mortality rate among HAARF patients was 41 (63.08%) patients, and full recovery occurred in 22 (33.85%) cases, partial recovery in 2 (3.08%) cases. HAARF was associated with high mortality (63.08%).

Table 25: Distribution of patients according to total duration of hospital stay and duration of stay prior to development of HAARF

Duration	Prior HARF		Total duration	
	Number	Percentage	Number	Percentage
2 to 5 days	36	55.38%	04	6.15%
6 to 10 days	13	20.00%	20	30.77%
> 10 days	16	24.62%	41	63.08%
Total	65	100%	65	100%

36 (55.38%) cases developed HAARF within 5 days of admission to ICU and total duration of stay was more than 10 days among 41 (63.08%) cases.

Table 26: Association of Age distribution and out come

Age (Years)	Total		Recovered		Expired	
	No.	%	No.	No.	%	%
> 80	01	1.53%	00	0.0%	01	100.0%
> 65 to 79	20	30.76%	10	50.0%	10	50.0%
> 40 to 64	27	41.52%	07	25.93%	20	74.07%
<40	17	26.15%	07	41.18%	10	58.82%

x^2= 3.588; df=3; p=0.3095 NS.

The majority of the cases were between 40 to 64 years age group, which had the highest mortality (74.07%), but in all age groups the mortality being was more than 50%. The above findings showed the mortality in HAARF was not significantly associated with age.

Table 27: Association of type of renal failure with the outcome

Outcome	Total	Intra renal		Prerenal	
		Number	Percentage	Number	Percentage
Complete recovery	22	18	32.73%	4	40.00%
Partial recovery	2	00	0.00%	2	20.00%
Expired	41	37	67.27%	4	40.00%
Total	65	55	84.61%	10	15.38%

x^2= 5.518; df=2; p=0.0634 NS.

Among the intrarenal type of renal failure 37 (67.27%) cases expired, out of 41 cases and among the prerenal type of ARF (10 cases), 4 had complete recovery (40%), 2 had partial recovery (20%) and the remaining 4 cases expired (40%),this shows type renal failure is not significantly associated with outcome of HAARF.

Table 28: Association of creatinine at admission with outcome

Creatinine at admission	Total	Expired		Recovered	
		Number	Percentage	Number	Percentage
<0.8	16	10	62.50%	06	37.50%
>0.8 to 1.4	49	31	63.27%	16	32.65%
= 1.4	7	3	42.86%	4	57.14%
Total	65	41	63.8%	24	36.92%

x^2= 0.738; df=2; p=0.6915 NS.

49 (75%) patients were having serum creatinine level ranging from 0.8 to 1.4 at the time admission to ICU. The level of creatinine at admission did not have statistically significant association with the outcome.

Table 29: Association of peak creatinine during hospital stay

Peak creatinine	Total		Expired		Complete recovery		Partial recovery	
	No	%	No.	%	No.	%	No.	%
Up to 1.5	05	7.69%	02	40.0%	03	60.0%	00	0.0%
> 1.5 to 2.0	13	20.0%	06	46.15%	07	53.85%	00	0.0%
> 2 to 3	14	21.53%	08	57.14%	05	35.71%	01	7.14%
> 3	33	50.76%	25	75.76%	07	21.21%	01	3.03%
Total	65	100%	41	100%	22	100%	02	100%

x^2= 7.356; df=6; p=0.2892 NS.

Higher the creatinine during the hospital stay had poor out come, but not significant statistically.

Table 30: Association of oliguria with outcome

Outcome	Oliguria present		Oliguria absent	
	Number	**Percentage**	**Number**	**Percentage**
Complete recovery	08	17.02%	13	72.22%
Partial recovery	02	4.26%	00	0.0%
Expired	37	78.72%	05	27.78%
Total	47	72.30%	18	27.69%

x^2= 18.270; df=2; p=0.0001 HS.

The patients who were oliguric had high mortality rate (78.72%) when compared to 72.22 % of patients recovered fully who are not oliguric, this is statistically significant, oliguria is highly significant risk factor in all patients of HAARF p= 0.0001

Table 31: Number of patients with SIRS and sepsis

Outcome	SIRS present		Sepsis present	
	Number	**Percentage**	**Number**	**Percentage**
Complete recovery	16	29.63%	11	11.36%
Partial recovery	01	1.85%	01	2.27%
Expired	37	68.52%	32	72.73%
Total	54	83.07%	44	67.69%

x^2= 0.271; df=2; p=0.8734 NS.

54 (83.07%) cases had SIRS, among them 44 (67.69%) progressed SEPSIS, the mortality in SIRS and SEPSIS are 68.5%, 72.73% respectively. The

association of the patients who had SEPSIS or SIRS with out come was not significant statistically.

Table 32: Association of SIRS and with outcome

Outcome	SIRS present		SIRS absent	
	Number	**Percentage**	**Number**	**Percentage**
Complete recovery	16	29.63%	06	54.55%
Partial recovery	01	1.85%	00	0.0%
Expired	37	68.52%	05	45.45%
Total	54	100%	11	100%

x^2= 2.632; df=2; p=0.2682 NS.

37 (68.52%) cases expired out of 54 cases with SIRS.

Table 33: Association of sepsis with outcome

Outcome	Sepsis Present		Sepsis Absent	
	Number	**Percentage**	**Number**	**Percentage**
Complete recovery	11	11.36%	10	47.62%
Partial recovery	01	2.27%	01	4.76%
Expired	32	72.73%	10	47.62%
Total	44	67.67%	21	32.30%

x^2= 3.924; df=2; p=0.1406 NS.

32 (72.73%) patients with SEPSIS expired out of 44 case, Association of sepsis as a risk factor with out come is not statistically significant.

Table 34: Association of hypotension with outcome

Outcome	Hypotension present		Hypotension absent	
	Number	Percentage	Number	Percentage
Complete recovery	07	16.67%	15	65.22%
Partial recovery	02	4.76%	00	00%
Expired	33	78.57%	08	34.78%
Total	42	64.61%	23	35.38%

x^2= 0.15.963; df=2; p=0.0003 HS.

The association of hypotension as a risk factor has the high mortality rate (78.57%) when compared to the patients who are not hypotensive (34.78%). 65.22% of patients had full recovery in absence of hypotension, this is statistically significant. Hypotension is the significant risk factor in the outcome of HAARF (p=0.0003)

Table No. 35: Association patient's level of sensorium with the outcome

Outcome	Conscious		Altered sensorium		Comatosed	
	No.	%	No.	%	No.	%
Complete recovery	18	75.0%	03	23.08%	01	3.57%
Partial recovery	02	8.33%	00	0.0%	00	0.0%
Expired	04	16.67%	10	76.92%	27	96.43%
Total	24	36.92%	13	20%	28	43.07%

x^2= 36.982; df=4; p=0.0001 HS.

Mortality rate is 96.43% in comatosed patients, when compared to 75% patients recovered if they are normal conscious. The level of sensorium had a statistically significant association with out come of HAARF patients (p= 0.0001).

Table 36: Association of requirement of assisted ventilation with outcome

Outcome	Required		Not required	
	Number	**Percentage**	**Number**	**Percentage**
Complete recovery	05	12.50%	17	68.00%
Partial recovery	02	5.00%	00	0.0%
Expired	33	82.50%	08	32.00%
Total	40	61.53%	25	38.46%

x^2= 21.471; df=2; p=0.0001 HS.

33 (82.50%) patients expired who required assisted ventilation expired when compared to 68% patients recovered fully who didn't require assisted ventilation, Requirement of assisted ventilation is a significant risk factor in the outcome of HAARF (p=0.0001).

Table 37: Association of hyperkelemia with outcome

Outcome	Hyperkelemia		No hyperkelemia	
	Number	**Percentage**	**Number**	**Percentage**
Complete recovery	02	14.29%	20	39.22%
Partial recovery	00	0.0%	02	3.92%
Expired	12	85.71%	29	56.86%
Total	14	21.53%	51	78.46%

x^2= 4.016; df=2; p=0.1343 NS.

85.71% patients had hyperkelemia expired when compared to 56.86% of patients expired in the group who ware not hyperkelemic. 40% of the patients without hyperkelemia had recovery. Hyperkelimic patients had high mortality which is not significant statistically. Hyperkelemia may or may not be present in all the cases of HAARF but in presence of hyperkelemia, patients had a poor prognosis.

Table 38: Association of metabolic acidosis with outcome

Outcome	Metabolic acidosis		No metabolic acidosis	
	Number	**Percentage**	**Number**	**Percentage**
Complete recovery	02	8.00%	20	50.00%
Partial recovery	00	0.0%	02	5.00%
Expired	23	92.00%	18	45.00%
Total	25	38.46%	40	61.53%

x^2= 14.656; df=2; p=0.0007 NS.

23 (92%) Of the patients expired who had metabolic acidosis compared to 20(50 %) patients recovered who did not have metabolic acidosis, the above observation is statistically significant. All the patients who had metabolic acidosis were associated with high mortality (p= 0.0007).

Table 39: Association of requirement ionotropic support with outcome

Outcome	Required		Not required	
	Number	**Percentage**	**Number**	**Percentage**
Complete recovery	05	11.11%	17	85.0%
Partial recovery	02	4.44%	00	0.0%
Expired	38	84.44%	03	15.0%
Total	45	69.23%	20	30.76%

x^2= 30.043; df=2; p=0.0001 HS.

The patients who required ionotropic support had high mortality (84.44%) and 85% of the patients who did not require ionotropic support recovered completely. This observation is statistically significant (P=0.0001). This infers requirement of ionotropic support is a significant risk factor in the outcome of HAARF patients.

Table 40: Association of co morbid conditions and their out come

Comorbid conditions	Total		Expired		Recovered	
	No.	%	No.	%	No.	%
Hypertension	18	27.69%	13	72.2%	05	27.28%
Diabetes mellitus	15	23.8%	12	80.0%	03	20.0%
Alcoholic liver disease	10	15.38%	08	80.0%	02	20.0%
Ischaemic heart disease	06	9.23%	02	33.33%	04	66.67%
Retro viral diseases	02	3.08%	01	50.0%	01	50.0%
Malignancy	01	1.54%	01	100%	00	0.0%
No cormid conditions	19	29.23%	09	47.37%	10	52.63%

The maximum patients didn’t have co morbid conditions 19 (29%), out of which 47.37% expired and 52.63% of patients recovered and followed by 18 cases suffering from diabetes which had 80% mortality. These findings were statistically not significant. However comorbid conditions are associated with poor out of HAARF.

Table 41: Association of type of management and its outcome

Outcome	Hemodialysis		Peritoneal dialysis		Conservative	
	No.	**%**	**No.**	**%**	**No.**	**%**
Complete recovery	01	20%	00	0.0%	21	38.18%
Partial recovery	00	00%	00	0.0%	02	3.64%
Expired	04	80%	05	100%	32	58.18%
Total	05	7.69%	05	7.69%	55	84.61%

x^2= 4.161; df=4; p=0.3846 NS.

The highest mortality occurred in dialyzed group (80%), 100% mortality in peritoneal dialysis patients, 55 (84.6%) cases who were treated conservatively of which 58% expired and 41.82% recovered. This shows that the type of dialysis does not play a significant role in the outcome of HAARF.

Table 42: The association of duration of hospital stay prior developing ARF with outcome

Duration of hospital stay	Total		Expired		Recovered	
	No.	%	No.	%	No.	%
2 to 5 days	36	55.38%	20	55.56%	16	44.44%
6 to 10 days	13	20.00%	08	61.54%	05	38.46%
> 10 days	16	24.62%	13	81.25%	03	18.75%

x^2= 3.157; df=2; p=0.2063 NS.

The maximum patients (81.25%) expired in the group of which had > 10 days of hospitalization stay before developing ARF, followed by 6 to 10 day group, this indicates higher is the mortality with later the development of ARF in the hospital. This was not statistically significant.

Table 43: The association of total duration of stay in hospital with the out come

Duration of hospital stay	Total		Expired		Recovered	
	No.	%	No.	%	No.	%
2 to 5 days	04	6.25%	04	100.0%	00	0.0%
6 to 10 days	22	33.85%	13	59.09%	09	40.91%
> 10 days	39	60.00%	24	61.54%	15	38.46%

x^2= 2.531; df=2; p=0.2821 NS.

The patients who had minimal duration of stay had the highest mortality that is 100 %, but over all, total duration of in hospital stay is not statistically significant associated with the outcome of the patients. Mean value of total duration of the stay was 14.42±7.31 days.

Table 44: Association of number of organ dysfunction and the out come

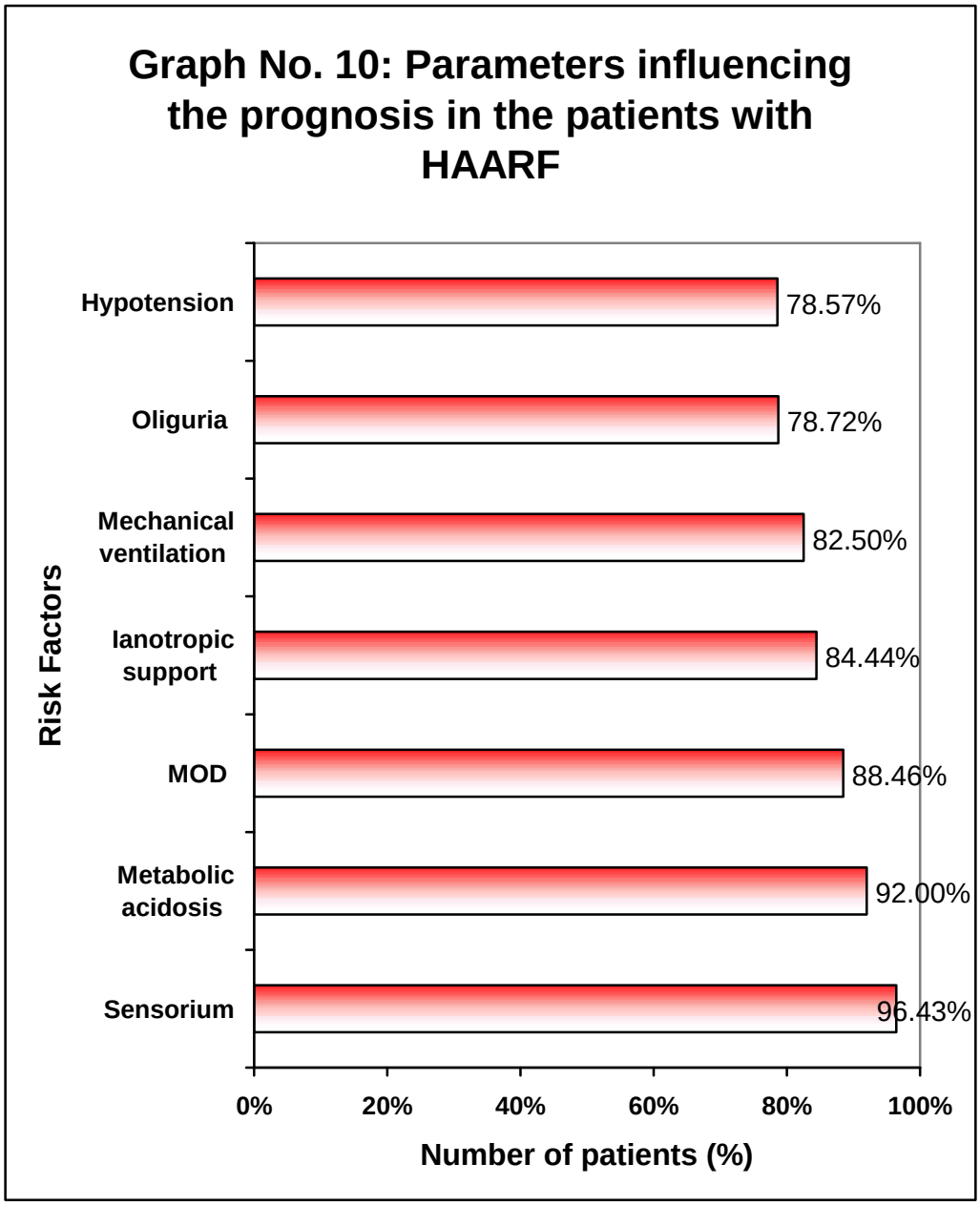
Number of organ dysfunction	Total		Survivors		Non survivors	
	No.	%	No.	%	No.	%
One organ	09	13.85%	06	66.67%	03	33.33%
Two organs	19	29.23%	08	42.10%	11	57.89%
≥ three organs (MOD)	26	40.00%	03	11.54%	23	88.46%
None	11	40.00%	07	63.54%	04	36.36%

x^2= 14.2018; p=0.0026 S.

Out of total 65 cases, 26(40%) cases had MOD, with 88.46% mortality rate and 12% recovery, among the patients with 2 organ dysfunction and 1 organ dysfunction had 57.89% and 33.33% mortality respectively. MOD is a one of the important risk factor which plays a role in the outcome of HAARF.

Table 45: Parameters influencing the prognosis in the patients with HAARF

Risk factors	Mortality
Sensorium	96.43%
Metabolic acidosis	92.0%
MOD	88.46%
Ianotropic support	84.44%
Mechanical ventilation	82.50%
Oliguria	78.72%
Hypotension	78.57%



Parameters influencing the prognosis in the patients with ARF in the intensive care unitthe higher mortality was observed in patients with altered sensorium, metabolic acidosis, MOD, ionotropic support. We also noted higher mortality in patients requiring assisted ventilation, oliguria, and hypotension. Association of these parameters with outcome was statistically significant ($p<0.05$).

DISCUSSION

The present study was conducted in the Department of Medicine between the period of January 2008 to December 2008. A total of 65 patients who developed ARF in ICUs in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were referred to nephrology consultation.

In the present study out 65 cases, 57 (87.69%) cases were males and 8 (12.31%) cases were females. This indicates there was a male predominance in HAARF. A study from Atlanta[11] found male predominance of 67%.

In the our study the age group of patients varied from 15 to 85 years in which maximum patients belonged to age group of 40 to 64 years (41.53%). The mean age of the patients was 52.38 ± 17.02 years. The above findings suggest HAARF can occur mainly in the older age group probably due to associated comorbid conditions. An Indian study from Bangalore[88] also noted the mean age of their patients 48 ± 17 years.

In the this study majority cases were admitted in MICU 35(53.8%), remaining were from SICU 16 (24.62%) cases and from NSICU 7 (10.77%) cases, ICCU 4 (6.15%) cases and, CTVS 3 (4.62%) cases. A study from Pittsburg[91] observed that 58.9% medical, 41.1% in surgical cases of HAARF. An Indian study from Varanasi[10] also noted 71.7% and 23.9% accounted for medical and surgical cases respectively.

In the present study among the medical disorders 10 were suffering from alcoholic liver disease, 8 cases of cerebrovscular accident, 5 cases of acute MI, 2

cases were of complicated malaria and RVD, 1 case of aplastic anemia and out of 14 general surgical cases most were in post operative state, among the others 8 cases were referred from neurosurgery and 2 cases from orthopaedics.

In our study prerenal type of HAARF was found in 10 (15.38%) cases, 55 (84.62%) cases were of intrarenal type of HAARF and we did not have any cases of post renal HAARF. In comparison to a study in Atlanta,[11] also showed 81% of HAARF patients were of intrarenal type and 19% of prerenal type and none from post renal.

In this study out of 65 (70.77%) cases had HAARF secondary to multifactorial etiology which included the combination of post operative state, dehydration, drugs, sepsis and remaining 10 (15.38%) cases were of hepatorenal syndrome, 9 (13.85%) cases of cardiogenic shock during the post MI period and there was 1 case of rhabomiolysis following RTA in comparison to our study, an Indian study done at Varanasi[10] showed etiology of HAARF was multifactorial, which included hypotension, volume depletion, nephrotoxic drugs, and sepsis.

In the present study 47 (72.31%), 42 (64.62%), 14 (21.54%) and 25 (28.46%) cases had oliguria, hypotension, hyperkalemia and metabolic acidosis respectively. An Indian study from Varanasi[10] also noted 71.74% of patients with HAARF had hypotension and 65.2% had oliguria.

In the present study out of 65 cases, 28 (43.8%) were comatosed and 13 (20%) cases were in altered sensorium and 24 (36.92%) cases were of normal consciousness.

In the present study 54 (83.07%) and 44 (67.67%) cases had SIRS and sepsis respectively. A study conducted in France[94] also showed 70.8% of the patients had SIRS and a study from Varanasi[10] found 69.5% of patients with sepsis.

In the present study 40 (61.5%) cases required assisted ventilation and 45 (69.23%) cases required ionotropic support, as most of HAARF patients suffered from sepsis with MOD.

In our study out of 65 cases, 26 (40%) patients had MOD, 19 (29.23%) cases had two organ dysfunction and 9 (13.88%) cases had one organ dysfunction and remaining 11 (16.92%) cases did not have any organ dysfunction leaving out kidney. In comparison to our study, an Indian study from Varanasi[10] showed 63% of the patients with MOSF and 37% of the patients had 2 organ dysfunction.

In the present study 36 (55.38%) patients had comorbid conditions. Among them 18 cases (27.69%) had hypertension followed by DM in 15 (23.08%) cases and 10 (15.38%) cases were of ALD, 6 (9.2%) cases were of IHD, 2 cases of RVD and 1 case of aplastic anemia. In the remaining 19 (29.2%) cases did not have any comorbid conditions. In fact these patients were largest group in this study. Most of these were young patients admitted due to acute illness like complicated malaria, crush injury, hanging, snake bite, acute pancreatitis, post operative cases and head injury cases from neurosurgery. Similarly a Varanasi[10] study from India noted 52.70% of patients had comorbid conditions in which hypertension, diabetes and IHD were the predominant.

In the present study, 49 (75.38%) had creatinine between 0.8 to 1.4 mg/dL and rest of 24.62% of the patients had creatinine < 0.8 mg/dL at the time of admission to the hospital. The mean value of the creatinine at the time of admission was 0.95 ± 0.27 mg/dL. The peak value of creatinine up to 1.5 mg/dL was found in 5 (7.69%) cases, > 1.5 to 2 mg/dL in 13 (20.00%) cases, between > 2 to 3 mg/dL 14 (21.53%) cases and > 3 mg/dL was found in 33 (50.76%) cases. The mean value of the peak creatinine during hospital stay was 3.75 ± 2.06 mg/dL.

In the present study 36 (58.38%) cases developed acute renal failure after 2 to 5 days of admission whereas, 20% in 6 to 10 days, 24.62% after > 10 days of duration respectively. The mean value of duration of hospitalization prior to HAARF was 7.29 ± 5.36 days.

In the present study 41 (63.08%) cases had > 10 days of total duration of stay and 22 (33.85%) of patients had between 6 to 10 days and 4 (6.15%) cases had 2 to 5 days of total duration of stay. This suggests most of the patients of HAARF had prolonged hospital stay. The mean length of stay for HAARF patients in our study was 14.42 ± 7.31 days. A study from Atlanta[11] also had mean length of stay of 26 ± 28 days. These discrepancies were appreciated because in our study, patients have been followed up to either death of the patient or maximum 2 weeks of duration.

In the present study of 65 cases, 37 (56.92%) patients required renal replacement therapy (RRT), among them, 10 cases (15.38 %) underwent renal replacement therapy (RRT). in these, 5 each received intermittent hemodialysis

and peritoneal dialysis. And the remaining 27 (72.97%) patients received conservative treatment instead of RRT, as their condition was too critical and CRRT facilities were not available at our centre.

In our study out of 65 cases, 41 (63.85%) patients expired, 22 (33.85%) patients had complete recovery and 2 (3.08%) patients had partial recovery. The above findings suggest, HAARF has high mortality rate. The mortality rate was 63% in Varanasi[10] study and 60.3% in the study from Pittsburg.[89]

In the present study, mortality was 67.27% in intrarenal and 40% in prerenal type of HAARF. A study conducted in Atlanta[11] showed 81% patients were of intrarenal type, 19% in prerenal type HAARF, since prerenal HAARF was included hepato renal syndrome, the mortality was noted at higher rate. Multicentre study mentioned 4% of patients had post renal type of HAARF but in our study we did not.

In the present study out of 65 cases 47 (72.30%) cases had oliguria in which 37 (78.72%) cases expired. In the remaining 18 cases who were non oliguric 13 (72.22%) patients survived. This shows that, oliguric patients had high mortality than non oliguric patients. The association between oliguria and the outcome of HAARF patients was statistically significant ($p=0.0001$). An Indian study conducted in Varanasi[10] found 65.2% of their patients were oliguric and mortality in these patients was 73.35% compared to 43.75% in those without oliguria ($p<0.05$).

In the present study out of 65 cases, 42 (64.62%) cases had hypotension. In these 33 (78.57%) cases expired p=(0.0003) and 40 (61.53%) of the patients required assisted ventilation, in these 33 (82.50%) expired (p=0.0001), 45 (68.23%) cases required ionotropic support, among these 38 (84.44%) expired (p=0.0001), 14 (26.15%) patients had hyperkalemia with 12 (85.71%) mortality (p=0.013), 25 (38.46%) of patients had metabolic acidosis, in these 23 (92%) expired (p=0.0007). These observations suggest hyperkalemia, requirement of assisted ventilation and ionotropic support have statistically significant association with outcome of HAARF.

In this study out of total 65 cases, 28 (43.8%) cases were comatosed, 24 (36.92%) cases were normally conscious and 13 (20%) cases were in altered sensorium. Among the 28 (96.43%) comatosed patients, 27 (96.43%) cases expired. In patients who were conscious, 20 (83.33%) patients recovered. Whereas among the patients with altered sensorium, 10 (76.92%) patients expired. These findings suggest strong association between patient sensorium with outcome of ARF patients. This association was statistically significant (p=0.0001).

Among the 26 patients with MOD, 23 (88.46%) patients expired. In two organ dysfunction group of 19 cases, 11 (57.89%) cases expired and out 9 patients with single organ dysfunction 6 (66.67%) patients survived and in the remaining 11 patients with no organ dysfunction, 7 (63.54%) patients survived and 4 (36.36%) patients expired. From these results it could be noted that, as the number of organs failure increases mortality rate also aggravates. Similarly as the number of organ dysfunction increases there is a decreased chance of

survivability and this association was statistically significant (p=0.0026). Similar correlation was found in another study.[10]

In our study, out of 65 patients, 54 (83.07%) cases had SIRS, in them 37 (68.52%) expired and 44 (67.69%) had sepsis. Among them 32 (72.73%) patients expired. The above findings show that SIRS and sepsis are very important risk factors which have the association with outcome of HAARF patients. 83.07% patients of SIRS cases progressed to the level of SEPSIS (81.48%), which has high mortality (72.73%) compared to the patients with SIRS (68.52%).

In the present study out of 65 patients, 49 patients had serum creatinine levels between 0.8 to 1.4 mg/dL at admission. Among them 31 (63.27%) patients expired, 16 (32.65%) patients had complete recovery and 2 (4.08%) patient had partial recovery. In the remaining 16 patients had serum creatinine was less than 0.80 mg/dL out of which 10 (62.5%) cases expired. However association of admission serum creatinine with outcome was statistically not significant (p=0.691).

In our study the peak creatinine level during the hospital stay was more than 3 mg/dL was recorded among 33 patients. Among them 25 (75.76%) patients expired. There were 13 cases with serum creatinine between 1.5 to 2.0 mg/dL of which 6 (46.15%) cases expired. However the association of peak serum creatinine during the hospital stay with outcome was statistically not significant (p=0.289).

In the present study duration of hospital stay prior to ARF ranged from 2 to 20 days. Out of 65 patients 36 (55.38%) patients developed ARF within 5 days

of admission. Among them 20 (55.56%) cases expired. In the remaining 13 patients developed ARF between 6 to 10 days after admission in which 8 (61.54%) patients died, and 16 patients developed ARF after > 10 days of hospitalization, of which 13 (81.25%) patients expired. These findings show higher the mortality with later the development of ARF in the hospital, indicating MOD and multifactorial etiology for HAARF. However the association between duration of hospital stay prior to developing ARF with outcome was statistically not significant (p=0.206).

In our study total duration of hospital stay ranged from 3 to 35 days. Out of 65 patients, 39 (60.00%) had hospital stay of more than 10 days. Among these 24 (61.54%) patients expired. In the remaining, 22 patients had hospital stay of 6 to 10 days in which 13 (59.09%) patients died and 4 (6.25%) patients had hospital stay of 2 to 5 days out of which 4 (100%) patients expired. However the association between duration of total hospital stay with outcome was statistically not significant (p=0.282).

In the present study 46 (70.76%) patients had comorbid conditions and 19 (29.23%) patients did not suffer from comorbid illnesses. Among these 10 (52.25%) cases recovered and 9 (47.37%) cases expired. In the remaining majority of the patients had hypertension (27.69%), followed by type 2 DM (23.8%), ALD (15.38%), IHD (9.23%), RVD (3.08%) and malignancy (1.54%). In the patients with hypertension 13 (72.2%) expired. Similarly 12 (80%) patients with DM, 8 (80%) patients with ALD, 2 (33.33%) patients with IHD and 1 (50%) patient each of RVD and malignancy (100%) expired. These findings suggest the patients with comorbid condition in ARF are at higher risk of mortality.

In our study, mode of management to treat ARF was dialysis and conservative type of management depending on the condition of the patients. Out 65 patients, 10 (15.38%) patients were treated by dialysis. Among them 5 (50%) each patients were treated with hemodialysis and peritoneal dialysis. Of the hemodialysed group, 4 (80%) patients expired and 1 (20%) patient recovered fully. In patients who were treated by peritoneal dialysis all patients expired (100%). The remaining 55 (84.62%) patients were treated conservatively. Among them 32 (58.18%) patients expired, 2 (3.64%) cases had partial recovery and 21 (38.18%) patients had complete recovery. These observations suggest type of management does not have statistically significant association with the outcome of the ARF patients (p=0.3846). However the patients who were treated by dialysis had higher mortality, the reason could be related to the fact that these patients frequently have MOD and die despite of appropriate treatment of renal insufficiency. But all the recovered patients were not considered for dialysis as they had mild renal failure.

A study conducted at Varanasi[10] observed that the organ support system was associated with excess mortality in dialyzed group (80% vs 46%) that in non dialyzed group. This apparent paradox in case of this life supporting systems seems to be due to the confounding effect of severity of the underlying disease process, sepsis and also the presence of multi organ dysfunction. Another study conducted in Atlanta[11] noted adverse outcomes in ARF such as dialysis and mortality occurred more frequently in HAARF (59.0%). In our study, higher mortality (90%) was seen among the dialyzed patients. This was probably due to smaller sample size and the severity of the patients.

It may be also noted that the number of days from ICU admission to institution of dialysis seems also to increase the risk dying in ARF patients. Various studies[10,94] found that patients who were dialyzed later in their ICU course had a worst survival rate compared to those dialyzed earlier.

Mode and intensity of RRT might affect outcome of ARF patients but available data are inconsistent.[3,90-93] The need for RRT resulted in a 6 times increased odds for dismal outcome. Recent studies have shown that the patients who needed RRT had a significantly higher mortality compared with patient groups without need for RRT.[11]

It underlines the importance of further research regarding the optimal timing, dose, and mode of RRT. When the patients at risk for development of ARF can already be identified on the first day of sepsis, greater effort can be put in measures to prevent the evolution to ARF and need for RRT; this would also potentially result in a better outcome. One could argue, however, that the decision for starting RRT in patients with ARF is not always taken on objective grounds.

In the present study out of 65 patients 41 (63.08 %) patients expired, 22 (33.85%) patients had complete recovery and 2 (3.08%) patients had partial recovery. The above findings suggest a HAARF has high mortality rate. These observations are consistent with most of the studies in the literature.[10]

Hospital acquired acute renal failure is a significant problem in the ICU patients associated with high mortality and morbidity. Appropriate management requires prospective identification and avoidance of renal insults and correction of pathophysiologic conditions if possible. Extensive research over several

decades have brought to identify pharmacological interventions that prevents renal injury and promotes renal recovery. These efforts have met with very limited success, universal strategies that are supportive have proven effective: avoidance of nephrotoxins, supportive treatments and haemodyanamic support, drug treatments have demonstrated benefits only in specific population.

CONCLUSION

- Hospital acquired acute renal failure most commonly occurred in 40- 64 yrs age group of patients.
- Maximum number of cases were found in MICU (53.85%) followed by SICU (24.62%) and NSICU (10.77%).
- Intrarenal type of HAARF (84.62%) was the most common type of renal failure followed by prerenal type (15.38%) in this study.
- Multifactorial etiology which includes the combination of dehydration, sepsis, nephrotoxic drugs and surgery is the most common contributing factor for HAARF followed by Sepsis.
- Sepsis, level of sensorium, metabolic acidosis, MOD, requirement of ionotropic support and assisted ventilation, oliguria, Hypotension were all independent significant risk factors for mortality in HAARF.
- Antimicrobials (aminoglycosides) and analgesics, osmotic diuretics like mannitol were the commonest contributing drugs in HAARF.
- Later the onset of HAARF poorer the outcome, HAARF was more likely to have prolonged length of the stay in the hospital.
- Mortality due to MOD was high and there was progressive increase in the mortality as the number of organ system failure increased.

- Although patients with HAARF were more likely to require dialysis but was associated with high mortality. In this study the dialyzed patients had high mortality, as this treatment was given in severe renal failure.

SUMMARY

The present cross sectional study was conducted to know the etiology and outcome in the patients with HAARF. This study was conducted on 65 patients, admitted to various ICUs at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2008 to December 2008.

The results of the present study showed that HAARF occurs more commonly in 40 – 64 yr age group of patients with male predominance. The most common cause of HAARF was multifactorial etiology followed by sepsis. Intrarenal type of ARF was most common type of renal failure among these patients. Sepsis, hypotension, requirement of ianotropic support and assisted ventilation, oliguria, MOD, level of sensorium, metabolic acidosis were all independent significant risk factors for the mortality. Most of the patients did not had chronic medical conditions but among the chronic medical conditions like hypertension / diabetes / IHD / liver disease were associated with poor outcome. Among the drugs causing HAARF were antimicrobial (Aminoglycosides), analgesics and osmotic diuretics like mannitol were the most common contributing factors especially in multifactorial cause of HAARF. Later the onset of HAARF poorer the outcome and were more likely to have prolonged length of hospital stay. HAARF was associated with high mortality and morbidity and few cases of recovery. Although most of the patients required dialysis but they were associated with high mortality. Amongst the recovered patients most of them were independent of dialysis. HAARF in the setting of ICU is characterized by increasing comorbidity, high incidence of sepsis, MOD and mortality.

Multifactorial etiology and Sepsis were the major risk factors of mortality in these patients. These findings call for early detection and aggressive management of sepsis and its associated complications, so as to bring down the mortality in the patients admitted to ICU.

It is worth of reemphasizing that therapeutic efforts to prevent the progression of HAARF should be applied early since this problem considerably increases the length and cost of hospitalization in addition to accounting for considerable morbidity and mortality.

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ANNEXURE I - CONSENT FORM

Title of the study

"ONE YEAR CROSS SECTIONAL STUDY OF ETIOLOGY AND OUTCOME OF HOSPITAL ACQUIRED ACUTE RENAL FALURE"

Purpose of study

The patients of HA-ARF in critical care units are at an increased risk of mortality due to severity of the illness, HA-ARF is associated with high hospital mortality and morbidity, By evaluating the etiology and the outcome of HAARF, we will be able to prevent HAARF and manage them in better ways. There will be 65 critically ill patients participating in the study during 1 year period. This study will be under the supervision of Dr. Girish. P. V. and under the guidance of Dr. M. S. Khanpet, Associate Professor, Dept of medicine, J.N. Medical college, Belgaum.

Procedure and treatment

The adult patients who have normal renal function at admission, and developed renal failure (serum creatinine ≥1.5) after 48 hrs of admission in MICU, SICU, medicine or post operative wards in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum are eligible for inclusion in the study group. The baseline concentration of creatinine is estimated in all the patients in critical care units, the creatine levels are again repeated anytime after 48 hours of admission.

To estimate serum creatinine, venepuncture will be done and other investigations as necessary may also be done.

The outcome of the patients will be recorded either in the first two weeks of hosptitalisation.

Risks

There are certain risks and discomforts that you may experience as a result of participating in the study these may include hematoma formation and/or infection at the site of venepuncture, which will be adequately treated.

Benefits

On the basis of new information, the out come of the patients would be better handeled. There will be no extra benefit to the patient otherwise.

Financial incentive for participation

You will not receive any payment for participating in this study .

Alternatives

If you not to participate in the study, you will receive the standard treatment for patients with this condition.

Authorization to publish results

Results of this study may be published for scientific purposes and/or presented to scientific groups; however you will not be identified.

Sponsors policy

There are no sponsors for this study.

Institutional policy

There will neither be any compensation to or for the patient and his/her relatives nor would there be any monetary for damage incurred.

Emergency provision

In the event of emergency you should telephone Dr. Girish P. V. at 9886391465 or Dr. M. S. Khanpet at 9448092537.

Voluntary participation

Your participation in this study is voluntary. Your decision whether or not to participate will not affect your current or future relations with us. You are free to discontinue participation in this study at any time and for any reason. In case you need any further information regarding your rights as a study participant, you may please contact Dr. V. D. Patil, J. N. Medical College, Belgaum and Chairman of the JNMC Institutional Ethics committee on Human Subjects Research, Telephone number 95 831 2471701.

Statement of consent

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me and I may ask questions at any time.

_____ _____

Siganature or Left Hand Thumb Impression Date

(volunteer Subject/Legally authorized representative)

_____ _____

Signature (witness) Date

_____ _____

Signature of investigator Date

ANNEXURE II

PROFOMA

TITLE OF THE STUDY

"ONE YEAR CROSS SECTIONAL STUDY OF ETIOLOGY AND OUTCOME OF HOSPITAL ACQUIRED ACUTE RENAL FALURE"

Patients Name: Address:

Age: Sex:

I.P.No: DOA:

Occupation: DOD:

Ward:

Original disease/ primary diagnosis:

Surgical -

Cardiac -

Infectious –

Miscellaneous -

Chief Presenting Symptoms:

Symptoms	**Duration**
1. Oliguria	
2. Anuria	
3. Hematuria	
4. Nocturia	
5. Pain in Abdomen	

6. Dysuria/ Strangury
7. Loin Pain
8. Fever
9. Vomiting
10. Diarrhoea
11. Altered Sensorium/ Drowsiness/ Confusion
12. Oedema
13. Breathlessness
14. Chest Pain
15. Palpitation
16. Cough with Expectoration
17. Hemoptysis
18. History of snake bite
19. History of acute blood loss or crush injury
20. History of injection of radiocontrast dye
21. History of burns
22. Drug intake

Past History

1. Any major illness like diabetes, hypertension, jaundice, etc.
2. Previous hospitalization
3. Any major operations and / or accidents.
4. History of blood transfusion.
5. Any other.

Family History

History of diabetes / hypertension in the family.

Personal History

Diet

Sleep

Appetite

Bowel bladder

Habits

Drug History

General Physical Examination

Level of Consciousness

Build

Nutrition

Pallor

Cyanosis

Clubbing

Icterus

Pedal Edema

JVP

Signs of Dehydration

Signs of Congestive Cardiac Failure

Vital signs

Pulse:

Blood Pressure: 1 Standing 2 Supine

Temperature:

Respiration:

Systematic Examination:

Cardiovascular System:

Central nervous system:

Per Abdomen:

Respiratory System:

Investigations:

Sr creatinine levels at admission:

1.Complete Blood Count (CBC)					
• Haemoglobin (Hb) A. Male B. Female	 13-17 gm% 11-15 gm%				
• Total Leukocyte count (TLC)	4000-11000/mm3				
• Differential Count A. Neutrophils (N) B. Lymphocytes (L) C. Eosiniphils (E) D. Basophils (B) E. Monocytes (M)	 40-70 % 20-40 % 1-6 % 0-1 % 2-10 %				
• Erythrocyte Sedimentation Rate (ESR) A. Male B. Female	 0-10 mm/Hr. 0-20 mm/Hr.				
2.Urine routine					
• Epithelial Cells	Absent				
• Pus Cells	0-2 /hpf				
• WBC's	0-2 /hpf				
• RBC's	0-2 /hpf				
• Albumin	Absent				

• Sugar	Absent				
• Casts	Absent				
3. Fractional Excretion of Sodium (FeNa)					
4. Urinary Sodium Concentration	75-200 mEq/day				
5. Urinary Osmolality	500-800 mOsm/day				
6. Urine Creatinine	1-1.6 gm/day				
7. Random Blood Sugar	70-160 mg%				
8. Minirenal					
• Urea	10-39 mg%				
• Creatinine	0.5-1.5 mg%				
• Sodium	130-145 mEq/L				
• Potassium	3.5-5 mEq/L				
• Chloride	98-106 mEq/L				
• Bicarbonate	21-30 mEq/L				
9. Arterial Blood Gas					
• PH	7.38-7.44				
• PO_2	80-100 mmHg				
• PCO_2	35-45 mmHg				
• SPO_2	90-100 %				
• HCO_3^-	21-30 mEq/L				

10. Liver Function Test					
• Total Bilirubin	0.2-1.0 mg/dl				
• Direct Bilirubin	0.0-0.3 mg/dl				
• Alkaline Phosphatase	50-140 U/L				
• Total Proteins	6.4-8.2 gm/dl				
• Albumin	3.4-5.0 gm/dl				
• A/G Ratio	1-2				
• SGOT	10-37 U/L				
• SGPT	20-65 U/L				
• Serum Ammonia	10-80 µgm/dl				
11. Electrocardiograph					
12. Ultrasonography for Kidney Ureter Bladder					

Treatment and Follow-up: In hospital

Conservative

Dialysis

Need for mechanical ventilation

Ionotropes / vasopressors

Outcome:

Recovery- (a) Full

(b) Partial

(c) No recovery

Death

Need for the renal replacement therapy at discharge:

ANNEXURE III MASTER CHART

Serial Number	In patient number	Age (Years)	Diagnosis	Oliguria	Duation of stay (Days)	Causes	Ionotropes	Mechanical ventilation	Dialysis	Conservative management	Urine sodium (mEq/L)	Plasma creatine (mg/dL)	Plasma sodium (mEq/L)	Urine creatinine (mg/dL)	Fraction excretion of sodium	U Cr/ PCr	RFI	Creat at admission (mg%)	Urea at admissioin (mg%)
1	250874	65	DH	Y	6	SEPSIS CS POP	Y	N	N	Y	40	1.8	139	57	0.9	31.6	1.26	0.8	25
2	252685	30	FTC SAH	Y	10	SEPSIS BSF	Y	Y	N	Y	138	4.1	168	44	7.7	10.73	12.86	1	15
3	252495	30	DAI SDH	Y	4	DRUGS BSF	N	N	N	Y	21	4.7	116	58	1.5	12.34	1.7	0.6	21
4	252068	77	OR POP	Y	12	POP DRUG	N	N	N	Y	16	4.7	114	39	4.7	8.29	1.93	1	37
5	253767	27	EDH	Y	7	POP DRUG	Y	Y	N	Y	20	4.2	117	29	2.5	6.9	2.89	0.6	17
6	255394	61	ALD HE	Y	9	SEPSIS DRUGS	Y	Y	N	Y	8	2	141	49	0.2	24.5	0.32	1	18
7	255368	60	ALD HE MTB	Y	10	SEPSIS DRUGS, HE	Y	Y	PD	N	76	1.6	60	31	8.2	19.37	3.92	1.2	40
8	255825	30	BT PHV	Y	7	POST OP DRUGS	Y	Y	Y	N	101	3.3	149	60	3.7	18.18	5.55	0.9	33
9	255974	67	AP	Y	4	SEPSIS	Y	Y	N	Y	90	3.6	135	40	6	11.11	8.1	0.5	29
10	256355	51	SDH	Y	8	MOD DRUGS	Y	N	N	Y	37	3	158	40	1.8	13.3	2.78	0.9	12
11	256811	65	CVA HTN DM	Y	17	DRUGS SEPSIS	N	N	N	Y	37	1.5	130	40	1.1	26.66	1.38	1.1	21
12	257879	60	RPT	Y	18	SEPSIS, DRUGS MOD	Y	Y	N	Y	38	2.1	152	38	2.5	18.09	2.1	0.9	41
13	258731	53	BT ABD SAH S	Y	10	SEPSIS,POST OP,DRUGS	N	Y	N	Y	15	1.8	138	10	2	5.55	2.7	1.4	17
14	257748	30	CI POP	Y	13	DRUGS POST OP	N	N	N	Y	8	2.7	146	23	0.6	8.51	0.94	0.7	146
15	260860	81	EX COPD HTN AS OPK	Y	8	DRUGS SEPSIS MOD	Y	Y	N	Y	31	2	118	60	0.9	30	1.03	0.6	29
16	264028	70	RTA #	Y	23	POST OP DRUGS	Y	N	N	Y	228	22	144	46.9	74.3	2.09	109.09	0.8	28
17	263946	12	HI EDH CRI	Y	8	DRUGS, BRAIN STEM FAIL	Y	Y	N	Y	73	1.6	156	30	2.5	18.75	3.89	0.5	27
18	269131	46	AH HE HOCM IE	Y	12	LF DRUGS SEPSIS	Y	N	Y	N	54	4.2	120	342	0.6	81.42	0.66	0.7	23
19	267667	56	AIMI	Y	3	PTL DRUGS ISC	Y	Y	N	Y	72	1.8	133	34	2.9	18.88	3.88	1.4	37
20	269654	61	ME	N	26	SEPSIS DRUGS	N	N	N	Y	80	1.7	152	34.8	2.6	20.47	3.9	1	18
21	276908	59	CS IHD CABG POP	N	18	POP SEPSIS	N	N	N	Y	10	1.5	141	14.9	0.7	9.93	1	0.9	22
22	269687	68	IHD OLD MI HTN DM	N	16	ISC	N	N	N	Y	20	1.8	132	28.96	0.9	1.44	13.81	0.8	16

Serial Number	In patient number	LOS prior to HAARF (Days)	Number of organs failed	Heart failure	Hypotension	SBP (mm Hg)	DBP (mm Hg)	SIRS	SEPSIS	UREA high (mg/dL)	Creatinine high (mg%)	Bilirubin (mg/dL)	Serum Albumin (gm/dL)	Platelet count	Comorbid condition	Department	Level of sensorium	outcome
1	250874	2	PA RS	Y	Y	60	40	Y	Y	70	1.9	0.7	2.6	2.25Lk	NIL	SUR	AS	RC
2	252685	4	CNS RS	N	Y	124	80	Y	Y	168	4.1	0.5	2.6	1.6Lk	NIL	NEU	COM	EX
3	252495	3	CNS	N	N	138	80	Y	Y	153	6.6	0.8	4.8	2.26Lk	HTN	NEU	COM	EX
4	252068	6	-	N	N	130	70	Y	N	114	4.7	5.9	2.2	3.5Lk	NIL	ORTHO	CN	RC
5	253767	5	CNS	N	Y	170	100	Y	N	183	10.2	0.6	3.8	2.29Lk	NIL	NEU	COM	EX
6	255394	6	PA RS CNS CVS	N	Y	120	60	Y	Y	99	4.5	7.2	2	78000	ALD	MED	COM	EX
7	255368	2	CNS PA	N	Y	110	60	Y	Y	116	3.9	0.6	2.1	45000	ALD DM	MED	COM	EX
8	255825	3	PA RS CNS	N	N	120	80	Y	N	106	5.6	1.2	2.3	1.8Lk	NIL	SUR	COM	EX
9	255974	2	CNS PA RS	N	Y	98	50	Y	Y	121	3.6	2.2	2.7	90000	NIL	SUR	COM	EX
10	256355	7	CNS RS	N	Y	120	70	Y	N	157	4.4	3.7	1.8	3Lk	NIL	NEU	COM	EX
11	256811	16	CNS	N	N	140	104	N	N	95	3.1	0.5	3.8	1Lk	HTN DM	MED	AS	EX
12	257879	17	RS PA CNS GIT	N	Y	90	40	Y	Y	133	2.5	0.8	2	1.6Lk	MGN	SUR	COM	EX
13	258731	2	GIT CNS RS	N	Y	144	90	Y	Y	89	2.5	1.3	2	1.1Lk	NIL	SUR	COM	EX
14	257748	8	GIT CNS RS	N	Y	90	50	Y	N	94	2.7	1.6	2.6	3.4Lk	NIL	NEU	CS	RC
15	260860	5	RS CNS	N	Y	150	60	Y	Y	118	2	0.4	2.6	2.5Lk	HTN AS OKC	MED	COM	EX
16	264028	4	-	N	Y	150	60	Y	Y	47	2.2	0.7	2.3	1.8 Lk	NIL	ORTH	CS	RC
17	263946	4	RSCNS	N	Y	80	36	N	N	1.6	8.2	0.1	4	2Lk	NIL	MED	COM	EX
18	269131	4	GIT,CNS CVS MOD	Y	Y	132	80	Y	Y	140	7.6	8.3	2.2	2.72Lk	HOCM	MED	AS	EX
19	267667	2	MOD	Y	Y	80	38	N	N	98	1.8	2.8	4.1	1Lk	DM	CARD	COM	EX
20	269654	7	CNS RS	N	N	100	60	Y	Y	70	1.7	0.8	2.5	2.63Lk	NIL	MED	RL CS	RC
21	276908	3	-	N	N	110	80	Y	Y	44	1.5	1.1	3.2	3 Lk	CABG	SUR	NR	RC
22	269687	4	-	N	N	120	80	N	N	207	4.2	0.1	2.3	2.10Lk	TVD, IHD HTN DM	CARD	NR	RC

Serial Number	In patient number	Age (Years)	Diagnosis	Oliguria	Duation of stay (Days)	Causes	Ionotropes	Mechanical ventilation	Dialysis	Conservative management	Urine sodium (mEq/L)	Plasma creatine (mg/dL)	Plasma sodium (mEq/L)	Urine creatinine (mg/dL)	Fraction excretion of sodium	U Cr/ PCr	RFI	Creat at admission (mg%)	Urea at admissioin (mg%)
23	277657	29	ALD HE S	Y	6	SEPSIS	Y	Y	N	Y	82	1.5	151	108	0.8	72	1.13	1.2	20
24	277444	36	HANGING	Y	11	AP SEPSIS DRUGS	Y	Y	PD	N	135	5.2	141	23.2	21.5	4.46	30.26	1.4	22
25	278410	47	BC SAH SDH	Y	5	DRUGS	N	N	N	Y	27	1.5	153	19.2	1.4	12.8	2.1	1.2	29
26	268766	72	IHD POST CABG	Y	25	ISC	Y	Y	PD	N	54	5.3	140	74	2.8	13.96	3.86	0.8	12
27	278088	35	CRI LAP	Y	23	POP DRUG	Y	Y	N	Y	15	2.7	143	13.74	2.1	5.08	2.95	1.4	21
28	272280	52	SB FMY	N	13	SEPSIS, CRI	N	N	Y	N	30	2.2	140	113.9	0.4	51.7	0.58	0.9	32
29	278755	60	MVR CABG	N	19	ISC POP	N	N	N	Y	47	1.6	142	172	0.3	107.5	0.43	0.7	18
30	279246	39	PA POP	N	18	DRUG POP	N	N	N	Y	164	3.7	130	37	12.6	10	16.4	0.9	21
31	265159	65	DM IHD PTCA	N	10	POP	N	N	N	Y	29	1.9	36	6.31	6.4	3.32	8.73	1.2	21
32	279734	56	CM	Y	15	SEPSIS DRUG	Y	Y	Y	N	55	3.5	135	6.31	11.9	1.8	30.77	1.2	63
33	275006	45	GBS VAP	N	30	SEPSIS DRUG	N	Y	N	Y	59	1.7	133	13.7	5.5	8.05	7.32	0.6	13
34	279368	77	COPD HTN H DM	YS	21	SEPSIS DRUG	Y	Y	N	Y	73	1.8	128	23	1.8	12.7	0.17	1.2	33
35	278626	27	MTB VAP BO	N	15	SEPSIS DRUG	Y	Y	N	Y	43	2.7	140	45.7	1.9	16.92	2.54	1	41
36	283793	49	AP	Y	17	SSH	Y	Y	N	Y	65	2.5	152	27.5	3.9	11	5.9	0.9	21
37	281599	77	BO POP	Y	13	POP	N	N	N	Y	108	1.8	151	47.3	2.7	26.27	4.11	1.4	50
38	313310	48	RVD CCM	N	25	AMP	N	N	N	Y	72	1.7	145	20.01	4.2	11.8	6.1	0.7	24
39	289525	18	CM	Y	9	MOD	Y	Y	PD	N	57	2.2	137	14	6.5	6.36	8.96	0.9	67
40	303427	31	ALD PO HTN HG	N	9	ALD POHTN	N	N	N	Y	18	1.9	132	68.1	0.4	35.84	0.5	1.3	38
41	303245	40	TH POST CR	N	6	SSH	Y	Y	N	Y	74	2.2	143	41.8	2.7	19	2.47	1.1	29
42	305989	42	ALW	Y	17	ALD POHTN	Y	Y	N	Y	40	3.5	161	52.9	1.6	15.11	2.64	0.7	10
43	304537	53	ALD PO HTN HE	Y	22	ALD HE	Y	Y	N	Y	5	2	142	49.5	0.2	14.75	0.33	0.8	60
44	304860	56	CVA HTN IHD	Y	9	CS	Y	Y	N	Y	33	3.1	159	43.4	1.5	13.9	2.37	1	21

Serial Number	In patient number	LOS prior to HAARF (Days)	Number of organs failed	Heart failure	Hypotension	SBP (mm Hg)	DBP (mm Hg)	SIRS	SEPSIS	UREA high (mg/dL)	Creatinine high (mg%)	Bilirubin (mg/dL)	Serum Albumin (gm/dL)	Platelet count	Comorbid condition	Department	Level of sensorium	outcome
23	277657	3	PA CNS	N	Y	50	NR	Y	Y	39	1.5	3.1	1.7	1Lk	ALD	MED	COM	EX
24	277444	11	CNS RS	N	Y	100	60	Y	ES	90	7.7	1.7	3.3	1Lk	NIL	MED	AS	EX
25	278410	4	CNS	N	N	130	80	Y	Y	88	3.4	2	3.3	2.9Lk	DM	MED	COM	EX
26	268766	20	MOD	Y	Y	90	60	Y	Y	191	5.9	2.4	1.4	7000	IHD	ITU	COM	EX
27	278088	5	CNS RS	N	Y	100	60	N	N	98	3.1	1.1	1.8	2.2 Lk	NILL	SUR	COM	RC
28	272280	2	-	N	N	120	80	Y	Y	144	6	1.1	1.6	62000	NONE	SUR	CON	RC
29	278755	13	-	N	N	120	80	Y	Y	116	1.6	1.2	3.1	2.15Lk	NONE	ITU	CS	RC
30	279246	4	-	N	N	90	60	Y	Y	40	3.7	0.8	3.1	3.6Lk	DM	SUR	CS	RC
31	265159	4	CVS	Y	Y	120	70	N	N	144	3.9	0.3	3.3	2Lk	DM IHD HTN	CARD	CS	RC
32	279734	4	RS GIT CNS	N	Y	100	60	Y	Y	144	4.4	14.5	2	30000	NIL	MED	AS	EX
33	275006	8	RS CNS	Y	N	100	60	Y	Y	133	1.7	1	3.5	2.7Lk	NIL	MED	AS	EX
34	279368	3	RS GIT CNS	N	N	100	60	Y	Y	9	4.8	4.3	31	3.19Lk	HTN DM COPD	MED	CON	EX
35	278626	7	RS	N	Y	100	70	Y	Y	91	2.7	0.9	1.6	95000	RS GIT CNS	MED	CS	RC
36	283793	19	CNS RS PA	N	Y	NR	NR	Y	Y	81	2.5	2.3	2.5	1.8Lk	ALD	SUR	COM	EX
37	281599	15	PA	N	N	124	64	Y	N	74	2.8	0.5	3.8	2.45Lk	DM.HTN	SUR	CS	RC
38	313310	19	CNS	N	N	130	80	Y	N	60	0.1	0.8	2.8	1Lk	RVD	MED	CS	RC
39	289525	5	CNS PA CVS	N	N	90	60	Y	N	132	4.2	3.2	1.9	28000	PREGNANCY	MED	COM	EX
40	303427	2	CNS PA	N	N	160	90	Y	Y	86	1.9	1.7	2.1	48000	ALD	MED	AS	RC
41	303245	4	CNS CVS RS	N	Y	70	NR	Y	Y	111	7.7	0.6	4.2	1.5Lk	HTN	NEU	COM	EX
42	305989	9	CNS PA RS CVS	N	Y	160	90	Y	Y	193	4.5	1.6	1.9	1.6 Lk	ALD	MED	COM	EX
43	304537	4	CNS PA RS CVS	N	Y	130	100	Y	Y	210	2.7	2.5	1.1	90000	ALD	MED	HE	EX
44	304860	7	CNS CVS RS	Y	Y	230	100	N	N	109	3.1	0.4	3.5	2.26Lk	IHD HTN	MED	COM	EX

Serial Number	In patient number	Age (Years)	Diagnosis	Oliguria	Duation of stay (Days)	Causes	Ionotropes	Mechanical ventilation	Dialysis	Conservative management	Urine sodium (mEq/L)	Plasma creatine (mg/dL)	Plasma sodium (mEq/L)	Urine creatinine (mg/dL)	Fraction excretion of sodium	U Cr/ PCr	RFI	Creat at admission (mg%)	Urea at admissioin (mg%)
45	304156	78	CVA HTN IHD	Y	15	GE CVA POP DRUGS SSH	Y	Y	PD	N	39	5	144	64.85	2.1	12.96	3	1.1	30
46	286518	54	ALD HE TBL	N	17	HE TB SSH	Y	Y	N	Y	13	1.5	120	620	0	413	0.03	0.7	14
47	312460	75	CVA PM APN	N	13	DRUG PN	Y	Y	N	Y	28	1.6	155	64	0.5	40	2.7	1.3	47
48	293395	36	DVT VF	N	8	DVT DF	N	N	N	Y	12	1.5	124	14	1	26.6	0.45	1.3	18
49	312331	75	AC MI	Y	8	STRPK HYPOT	N	N	N	Y	48	5.1	139	30.69	5.7	5.88	8.16	1.4	29
50	312280	65	IHD AC LVF HTN DM	Y	14	CS	Y	Y	N	Y	12	2.3	146	50.06	0.4	21.7	0.55	1.1	22
51	301644	66	CVA BAO	N	18	HYPOT	Y	Y	N	Y	28	2.8	148	40.06	1.3	14.2	1.97	1.1	42
52	305164	72	SIADH PUL TB	Y	17	SIADH	N	N	N	Y	73	1.8	123	76.23	1.4	42.2	1.72	0.6	22
53	304376	57	CVA HTN BLEED	N	17	DRUGS	N	N	N	Y	87	2.2	156	23.89	5.1	10.8	8.05	0.8	15
54	287126	59	HCGP IHD HTN	Y	14	ISC POP	Y	Y	N	Y	135	2.4	145	60.85	3.7	23.8	5.67	1.2	86
55	292764	40	GBS SEPSIS	Y	10	SSH	Y	Y	Y	N	10	2.7	134	45.43	0.4	16.6	0.6	0.5	41
56	277214	65	CAD CABG POP	Y	6	POP SEPSIS	Y	Y	N	Y	21	2.5	134	32	2.5	1.28	17.35	1.4	56
57	281275	68	RAS POP	OL	13	DRUGS POP SEPSIS A	N	N	N	Y	26	2	149	43.8	0.8	21.9	1.18	0.9	23
58	298106	35	ALD V HE	Y	35	HE SSH	Y	Y	N	Y	14	2.7	129	54.8	2.7	20.29	0.66	0.6	18
59	287044	56	AA DM	Y	30	DRUG MOD IMS	Y	Y	N	Y	59	4.1	152	208.6	4.1	50.73	1.61	1	43
60	303060	32	OPP	N	19	SEPSIS	N	Y	N	Y	23	1.8	144	67.98	0.4	37.4	0.61	0.9	22
61	292135	32	SDH CRA	Y	24	POP SEPSIS MOD	N	Y	N	Y	12	2.8	135	41.8	0.6	14.9	0.8	0.7	16
62	290007	46	CRI SCA	Y	18	BL MOD SSH	Y	Y	N	Y	67	6	133	45.88	6.6	7.63	8.78	0.8	21
63	341745	36	RVD TOX	Y	30	SS APN	Y	N	N	Y	70	1.9	118	23	4.9	12.1	5.78	0.8	20
64	313354	75	PN DM HTN	Y	4	SSH	Y	Y	N	Y	43	2.4	134	1380	0.1	575	0.74	1.2	14
65	313657	70	PN DM HTN MPE PC	N	20	SEPSIS CS CCF	Y	Y	N	Y	16	1.9	130	16	6	8.42	1.9	0.6	20

Serial Number	In patient number	LOS prior to HAARF (Days)	Number of organs failed	Heart failure	Hypotension	SBP (mm Hg)	DBP (mm Hg)	SIRS	SEPSIS	UREA high (mg/dL)	Creatinine high (mg%)	Bilirubin (mg/dL)	Serum Albumin (gm/dL)	Platelet count	Comorbid condition	Department	Level of sensorium	outcome
45	304156	11	PA CNS RS	N	Y	90	60	Y	Y	438	7.3	1.05	2.3	90000	HTN	MED	CS	EX
46	286518	10	PA RS CNS	N	Y	100	60	Y	Y	135	1.5	17.9	2.4	88000	ALD	MED	AS	EX
47	312460	5	CNS RS PA	N	Y	160	90	Y	N	147	1.7	0.7	1.5	1.97Lk		MED	COM	EX
48	293395	3	-	N	N	120	80	Y	N	25	1.9	0.8	4.5	1.37Lk	HCGP	MED	CS	RC
49	312331	3	CVS	Y	N	100	70	N	N	139	5.8	0.8	5	2Lk	DRUGS HYPOT	MED	CS	RC
50	312280	3	CVS RS PA	Y	Y	90	60	N	N	109	3.2	10.3	1.7	1.81Lk	IHD HTN DM	MED	CS	RCP
51	301644	4	CNS CVS	N	Y	110	60	N	N	42	2.8	0.34	3.7	2.8Lk	HTN DM	MED	COM	EX
52	305164	5	CNS RS	N	N	160	100	N	N	40	1.8	0.7	2.8	3.48Lk	HTN TB	MED	CS	RC
53	304376	8	CNS CVS	N	N	150	100	N	N	147	2.2	0.5	4.4	2Lk	HTN IHD	MED	CS	RC
54	287126	2	PA CVS PA	N	Y	110	70	Y	Y	305	8	1.7	1.6	3.21Lk	HTN IHD HCGP	SUR	CS	EX
55	292764	9	CNS MOD	N	Y	130	80	Y	Y	90	2.9	5.1	1.4	1.5Lk	AUTOIMMUNE	MED	CON	EX
56	277214	6	CVS PA RS	Y	Y	90	60	Y	Y	99	2.5	6.8	2.1	1.5Lk	POST CABG POP	ITU	CS	RCP
57	281275	11	CVS CNS	N	N	156	80	Y	N	161	4.9	0.3	4.3	2.6Lk	HTN DM ATH POP	SUR	AS	EX
58	298106	18	PA CNS RS CVS	N	Y	100	70	Y	Y	99	3.6	18	2.3	95000	ALD	MED	AS	EX
59	287044	19	CNS RS HEM	N	Y	100	70	Y	Y	283	3.5	25.7	2.1	45000	AA DM DRUGS HTN MOD F	ONC	COM	EX
60	303060	5	CNS RS	N	N	130	80	Y	Y	81	1.8	0.6	2.3	3Lk	SEPSIS	MED	AS	RC
61	292135	13	MOD	N	Y	90	60	Y	Y	103	5.2	1.1	1.2	4.45Lk	SSH	NEU	COM	EX
62	290007	12	MOD	N	Y	100	60	Y	Y	206	6	2.5	1.6	1.13Lk	SSH	VAS	AS	EX
63	341745	15	MOD	N	Y	142	80	Y	Y	147	3.5	1	1.8	70000	RVD	MED	COM	EX
64	313354	4	SEPSIS.MOD	N	Y	100	70	Y	Y	141	2.4	4.6	1.9	2 Lk	DM HTN	MED	COM	EX
65	313657	20	RS,CVS,	Y	Y	90	40	Y	Y	80	1.9	4.2	2.2	2Lk	DM HTN	MED	CS	EX

ANNEXURE III

KEY TO MASTER CHART

#	-	Fracture
A	-	Angina
AA	-	Aplastic Anaemia
ABD	-	Abdomen
AH	-	Acute hepatitis
AIMI	-	Acute inferior wall myocardial infarction
ALD	-	Alcoholic liver disease
ALW	-	Alcohol withdrawal
AMP	-	Amphoterisin B
AP	-	Acute pancreatitis
AP	-	Aspiration Pneumonia
APN	-	Aspiration pneumonia
AS	-	Altered sensorium
AS	-	Aortic stenosis
ATH	-	Atherosclerosis
BAO	-	Basilar artery occlusion
BC	-	Brainstem contusion
BIA	-	Blunt injury abdomen
BL	-	Blood loss
BO	-	Bowel obstruction, interstinal obstruction
BSF	-	Brainstem failure

BT	-	Blunt trauma
CABG	-	Coronary artery bypass graft
CAD	-	Coronary artery disease
CARD	-	Cardiology
CAS	-	Carcinoma stomach
CCF	-	Congestive cardiac failure
CCM	-	Cryptococcal meningitis
CI	-	Crushing injury
CM	-	Cerebral Malaria / Complicated Malaria
CN	-	Conscious
COM	-	Comatosed
COPD	-	Chronic Obstructive Pulmonary Disease
CR	-	Craniotomy
CRI	-	Cervical injury
CSH	-	Cardiogenic shock
CVA	-	Cerebro vascular accident
DAI	-	Diffuse axonal injury
DBP	-	Diastolic blood pressure
DF	-	Dengue fever
DH	-	Direct hernia
DM	-	Diabetes mellitus
DVT	-	Deep vein thrombosis
EDH	-	Extra dural hematoma
EX COPD	-	Exacerbation of COPD

EX	-	Expired
F	-	Fungal infection
FMY	-	Fasctiotomy
FNa	-	Fractional excretion of Sodium
FTC	-	Fronto temporal contusion
GBS	-	Gullian barre syndrome
GE	-	Gastroenteritis
H	-	Hemorrhage
HAARF	-	Hospital acquired acute renal failure
HCGP	-	Hyper coagulobility state
HD	-	Hemodialysis
HE	-	Hepatic encephaloathy
HEM	-	Hematology
HG	-	Hypoglycemia
HI	-	Head injury
HOCM	-	Hypertrophic cardiomyopathy
HTN	-	Hypertension
HYPOT	-	Hypotension
IE	-	Infective endocarditis
IHD	-	Ischemic heart disease
IMS	-	Immuno suppression
ISC	-	Ischemic
ITU	-	Intensive tharpy unit
LAP	-	Laporotomy

LF	-	Liver failure
Lk	-	Lakhs
LOS	-	Length of stay
LVF	-	Left ventricular failure
ME	-	Meningo encephalitis
MED	-	Medicine
mEq/L	-	Milli equivalent per litre
mg/dL	-	Milli gram per deci litre
mg/dL	-	Milligram per deciliter
MGN	-	Malignancy
MI	-	Myocardial infarction
mm Hg	-	Milli meters of mervy
MOD	-	Multiple organ system dysfunction
MTB	-	Miliary tuberclosis
MTB	-	Miliary tuberculosis
MVR	-	Mitral valve replacement
N	-	No
NEU	-	Neurosurgery
OPP	-	Organo phosphorous poisoning
OR	-	Osteoarthritis
ORTHO	-	Orthopaedics
P KOCH'S	-	Pulmonary Kochs
PA	-	Pelvic abscess
PD	-	Peritonial dialysis

PE	-	Pericardial effusion
PHV	-	Perforation of hollow viscus
Pl Cr	-	Plasma Creatinine
Pl Na	-	Plasma sodium
PM	-	Pontine myelinolysis
PN	-	Pneumonia
POHTN	-	Portal Hypertension
POP	-	Post Operation
POP	-	Post Operative Stage
PRC	-	Partially recovered
PTb	-	Pulmonary Tuberculosis
PTCA	-	Pre catheter transluminal coronary angioplasty
PTL	-	Post thrombolysis
RAS	-	Renal artery stenosis
RC	-	Recovered
RFI	-	Renal failure index
RFI	-	Renal failure index
RPT with OU	-	Retroperitoneal tumour with obstructive uropathy
RTA	-	Road traffic accident
RVD	-	Retrovival disease
SAH	-	Subarachnoid hemorrhage
SB	-	Snakebite
SBP	-	Systolic blood pressure
SCA	-	Subclavican artery

SEPSIS	-	SEPSIS
Si	-	Splenectomy
SSH	-	Septic shock
SSH	-	Septic shock
STADA	-	Syndrome of inappropriate anti diuretri hormone
STRPK	-	Streptokinase
SUR	-	Surgery
TBL	-	TB Lymphadenitis
TbLy	-	Tuberculosis lymphadenitis
TH	-	Trans herniation
TOX	-	Toxoplasmosis
TVD	-	Triple vessel disease
UCr	-	Urine creatinine
UNa	-	Urine sodium
V	-	Varices
VAP	-	Ventilation associated pneumonia
VF	-	Viral fever
Y	-	Yes