"CLINICAL AND LABORATORY EVALUATION OF ACUTE KIDNEY INJURY (AKI) IN ELDERLY POPULATION: A ONE YEAR CROSS-SECTIONAL STUDY AT KLES DR. PRABHAKAR KORE HOSPITAL"

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ENDORSEMENT

This is to certify that the dissertation entitled **"Clinical and Laboratory Evaluation of Acute Kidney Injury (AKI) in Elderly Population: A One Year Cross-Sectional Study at KLES Dr. Prabhakar Kore Hospital" – A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY"** is a bonafide research work done by **REG NO. BG0115006.**

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ABBREVIATION	
ADQI	The Acute Dialysis Quality Initiative
AKI	Acute Kidney Injury
AKIN	Acute Kidney Injury Network
ATN	Acute tubular necrosis
BPH	Benign Prostatic Hypertrophy
BPH	Benign Prostatic Hypertrophy
BUN	Blood urea nitrogen
CIN	Contrast-induced nephropathy
CKD	Chronic kidney disease
HU/HN	Hydroureter/Hydronephrosis
ESRD	End-Stage Renal Disease
ESRF	End Stage Renal Failure
HIVAN	HIV associated Nephropathy
ICD	International Classification of Diseases
IHD	Ischemic Heart Disease
KDIGO	Kidney Disease Improving Global Outcomes
RIFLE	Risk, Injury, Failure, Loss, End Stage
RPGN	Rapidly progressive glomerulonephritis
RRT	Renal Replacement Therapy
SCr	Serum Creatinine
UNFPA	United nations population fund
DCM	Dilated Cardiomyopathy

ALD	Alcoholic Liver Disease
RVD	Retro-Viral Disease
CVA	Cerebro-Vascular Accident
IWMI	Inferior Wall Myocardial Infarction
LVF	Left Ventricular Failure
COPD	Chrpnic Obstructive Pulmonary Disease
HTN	Hypertension
DM	Diabetes Mellitus

ABSTRACT

Need for Study

In the developing countries, the factors responsible for ARF are different from the factors causing ARF in the developed world. In India, the number of studies[9-14]on AKI in elderly are very limited as parallel with the increasing number of AKI cases in the elderly.

Aims & Objectives –

The study was aimed at studying the clinical profile of Acute Kidney Injury (AKI) among elderly subjects presenting to a tertiary care teaching hospital.

Material and Methods –

The current study was a cross sectional study of 200 elderly subjects aged 65 and above with acute kidney injury, conducted in the department of General medicine KLES Dr Prabhakar Kore Hospital, Belagavi for a period of 1 year from january 2016 to december 2016.

Results & Conclusions –

The mean age **was** 70.46 years. The youngest person was 65 years old and the oldest person was 93-years-old.

1. The most common co-morbidities present in the study population were Hypertension (36.50%), Diabetes mellitus (34%) and Ischemic Heart Disease (11%).
2. The most common diagnosis in the study population was Acute Gastroenteritis in 28.50% of the study population, followed by Pyelonephritis in 16% of the population and Urosepsis in 12% of the population. The other common diagnoses were Obstructive Uropathy in 8.5%, Pneumonia in 6.5%, NSAID induced nephropathy in 5.05% and lower limb cellulitis in 4% of the subjects.
3. In histopathology of 29 specimens, ATN (Acute Tubular Necrosis) was the most common abnormality seen in 75.86% of specimens. The other common histopathological findings were AIN (Acute Interstitial Nephritis) in 10.34%, ATIN (Acute Tubular Interstitial Nephritis) was seen in 6.89% and HIVAN (HIV associated nephropathy) and RPGN (Rapidly progressive glomerulonephritis) 3.44% subjects each.

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INTRODUCTION

KIDNEYS are two bean-shaped organs, which have the capacity to excrete wastes[(1)], to maintain fluid balance, to conserve electrolytes, to concentrate urine, to make hormones that regulate blood pressure, keep bones strong, produce Red Blood Cells. But when the kidneys stop working abruptly, it is known as Acute Renal Failure.

The term " **Acute Kidney Injury** "(AKI) which was known previously as "**Acute Renal Failure**"(ARF) is a sudden or rapid decline in Filtration function of the kidneys resulting in the loss of the capacity of kidneys to excrete wastes, to maintain fluid balance, to conserve electrolytes and to concentrate urine[(2)].It is characterized usually by a rise in the concentration of serum creatinine or a rise in the concentration of Blood Urea Nitrogen (azotemia). But, immediately following the injury of the kidney, they may be within normal standard levels and the only indication may be decreased in the production of urine. Cases of AKI have been reported since ages. Acute Renal Failure has affected humans from time centuries old[(3)].During the World War II, in the bombing of London, in crash victims who were severely injured, Bywaters and Beall [(4)]described an acute loss of function in the kidneys. Then, a series of clinical and experimental including pathological studies lead to the development of the theory of ARF in 1951[(3)].

KIDNEYS are not an exception to the process of aging during the course of which changes in the structure and function of the organs [(5)], accumulate together in a living organism with the passage of time and these changes become evident as a decline in the physiological functions of the organs until death.

The aging kidneys are at an increased risk of AKI since they undergo several structural and functional changes[6]. The Incidence of AKI is high, especially among the elderly[7].

AKI in elderly carries an increased risk of mortality. Those who survive also are left to deal with chronic kidney disease (CKD), eventually leading to ESRD (End-Stage Renal Disease).

In the developing countries, the factors responsible for ARF are different from the factors causing ARF in the developed world. The main attention is on the critically ill and elderly with ARF in developed countries while the developing countries, in addition, also have to manage cases of ARF arising as a result of gastroenteritis, infections. The Geriatric population is on the rise in India. A report[8]jointly brought out by united nations population fund (UNFPA) and help age international says“ India has around 100 million elderly at present and the number is expected to increase to 323 million, constituting 20 percent of the total population by 2050 ”.

But in India, the number of studies [9-14]on AKI in elderly are very limited as parallel with the increasing number of AKI cases in the elderly. Most of the literature which is available is from Developed countries. Review articles [7, 15-18] by various authors have stressed that AKI is a public health emergency and stressed the importance of addressing AKI in the elderly.

Kerr, M., et al. (2014) [19]in their study on "The economic impact of acute kidney injury in England”estimated about 0.4 - 0.6% of the total UK National Health Service Budget in 2009–2010 as the total costs spent yearly for treating AKI. In addition to this, there is the additional burden of following CKD, including RRT

(Renal Replacement Therapy) if the stage of ESRF (End Stage Renal Failure) is reached.

So far, AKI has been managed mainly by supportive therapy in elderly because as such no proven treatment measures exist in elderly for treating AKI[16]and hence the outcome is poor in elderly. In order to reduce the incidence and improve the outcome in elderly, an exhaustive and detailed knowledge of epidemiology, aetiology, the natural history of the disease, clinical course, prognosis and mortality rates of AKI in the elderly is essential.

So, our aim here is to study the spectrum of acute kidney injury (AKI) in elderly and to study various aetiological and risk factors leading to AKI in the elderly

OBJECTIVE

The study was aimed at studying the clinical profile of Acute Kidney Injury (AKI) among elderly subjects presenting to a tertiary care teaching hospital.

Objectives:

- To study the spectrum of acute kidney injury (AKI) in elderly.
- To study various risk factors and etiologies leading to acute kidney injury (AKI) in the elderly.

REVIEW OF LITERATURE

DEFINITION OF AKI AND DIAGNOSTIC CRITERIA

According to Schrier's (2008) Manual of Nephrology[20]"Acute Renal Failure (now replaced by the term AKI) is a sudden and usually reversible decrease in the GFR occurring over a period of hours to days. ARF may occur in patients with previously normal renal function or patients with CKD".

According to the recent review article by Makris et al (2016)"AKI is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and impairment (loss of function)" [21]

The concept and definition of ARF have been through various changes and examination through years and this term has been replaced by Acute Kidney Injury (AKI).

According to the Kidney Disease: Improving Global Outcomes (KDIGO) Foundation (2012), the most recent definition is [22, 23]

AKI is defined as any of the following:

1. An increase in Serum Creatinineby $\geqslant 0.3$ mg/dl within 48 hours; or
2. An increase in Serum Creatinineby $\geqslant 1.5$ times baseline value, which is known or presumed to have occurred within the prior 7 days; or
3. Urine volume <0.5 ml/kg/h for 6 hours"

AKI as defined by Harrison's Manual of Medicine (2016)[24] as "Acute kidney injury (AKI), previously known as acute renal failure, is characterized by the sudden impairment of kidney function resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys. AKI is not a single disease but,

rather, a designation for a heterogeneous group of conditions that share common diagnostic features: specifically, an increase in the blood urea nitrogen (BUN) concentration and/or an increase in the plasma or serum creatinine (SCr) concentration, often associated with a reduction in urine volume. It is important to recognize that AKI is a clinical diagnosis and not a structural one. A patient may have AKI without injury to the kidney parenchyma."

Since early days, various operational definitions have been used for defining the Term "Acute Kidney Injury" (previously known as Acute Renal Failure). There has been no operational definition of AKI which is universally accepted.

AKI has been defined by several international and national organizations and groups based on various criteria. Initially, AKI was defined as an abrupt and sustained decrease in the functioning of kidneys which resulted in retention of nitrogenous products such as urea and creatinine and other waste products. But this is vague and cannot be used as an operational definition. Only the most severe forms of AKI were recognized by the definitions that crudely stated that only those cases needing RRT[25] were suffering from AKI.

In the last 2 decades, there is a trend of acknowledging the deterioration of functions in the kidney which are very small[26, 27].But all these minor changes may not be clinically relevant. The techniques and methods used for assessment of kidney function also need attention[28].

The availability of many biomarkers such as NGAL, Cystatin C which is readily available and some being inexpensive, have also added to the dilemma of putting a single worldwide defining criterion of AKI[26].

According to a review [17] by Cooper et al(2015), in various clinical studies across the globe, more than 30 different criteria have been hired in defining AKI. This is due to the heterogenity of the patterns of renal injury, the variability in the level of oliguria, and the time lag which occurs in the rise and fall of the concentration of the serum creatinine with injury and recovery. The two classification systems that attempted to give an operational definition to AKI were the RIFLE (Risk, Injury, Failure, Loss, End Stage) criteria and the AKIN (Acute Kidney Injury Network) criteria.

Prior to 2004, the definition given by ICD (International Classification of Diseases) was in use[29].They were used mainly for administrative purposes only in healthcare systems of developed countries. In 2004,ADQIgroup[30](The Acute Dialysis Quality Initiative) proposed" The Risk, Injury, Failure, Loss, End-stage renal disease (RIFLE) criteria"for the diagnosis and classification of Acute Renal Failure. ADQI group used the term Acute Renal Failure.

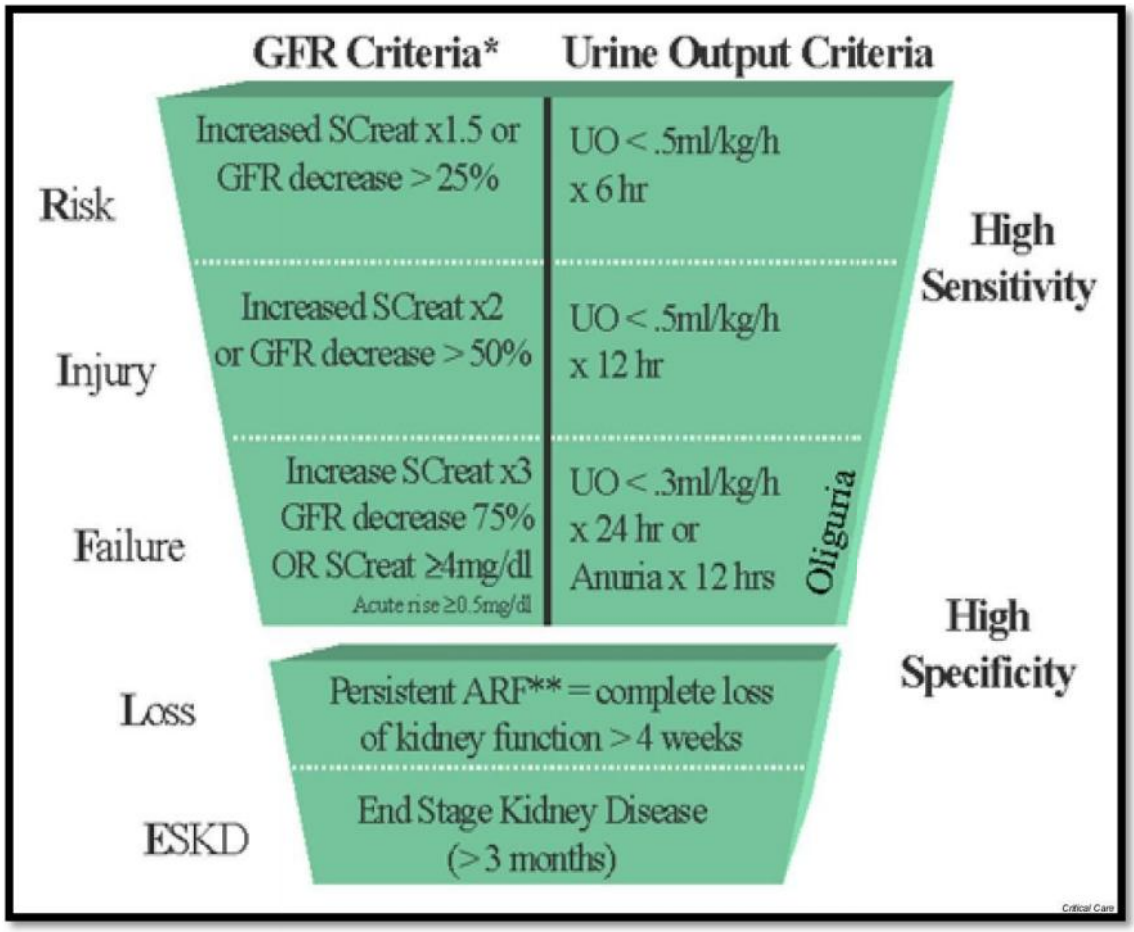
Based on the severity and duration of Renal injury, Acute Renal Failure was stratified into 5 stages

1. Risk
2. Injury
3. Failure
4. Loss
5. End-stage disease

Increasing RIFLE stage leads to greater risk of death.

The degree of Renal dysfunction is defined based on the relative increase in serum creatinine from baseline or the duration of oliguria or severity of oliguria.

Figure 1: The RIFLE Criteria for AKI (ARF)[30]



In this criteria, at the top of the figure, more patients will be incorporated in the mild category and hence there will be high sensitivity but some actually without renal failure may also be included (less specificity). But at the bottom, the criteria are set more strict and hence the specificity will be high, although some with renal failure may be missed.

Multiple studies [31-35]done have also demonstrated a clear association between the clinical outcomes and the RIFLE staging across various settings in different countries. The AKIN criteria are just parallel to the risk, injury, and failure stages of the RIFLE criteria. They are the most applicable criteria while characterizing AKI in the hospital.

The Acute Kidney Injury Network (AKIN) was convened by an international consortium of renal and critical care societies[36].The AKIN had proposed several refinements to the RIFLE criteria. Modifications to the definition of AKI proposed by AKIN included

1. Use of the term "Acute Kidney Injury" instead of the term Acute Renal Failure so that the broad range of Acute Renal Dysfunction is seized and usage of the term "Kidney" instead of "Renal" may also help in patient communication better.
2. The Term "Acute Renal Failure" to be constrained to the severe state of complete organ dysfunction
3. Addition of an absolute increase in serum creatinine of ≥ 0.3 mg/dL and
4. Specification of time that the decline in kidney function must occur within a 48 hour period.

Table 1:AKIN STAGING AND CLASSIFICATION[36]

stage	Criteria for serum creatinine *	Criteria for urine output*
1	Increase in serum Creatinine≥1.5–2 times the baseline value or increase in serum creatinine ≥ 0.3 mg/dL	<0.5mL/kg/hr for more than 6hrs
2	Increase in serum Creatinine 2–3 timesthe baseline value	<0.5mL/kg/hrfor more than 12hrs
3	Increase in serum Creatinine of ≥ 3 times baseline value or a creatinine value ≥4mg/dl with an acute rise of atleast 0.5mg/dL or Individuals receiving RRT	<0.3mL/kg/hr for 24hrs or anuria for 12hrs

*Only either of the one criterion to be fulfilled to qualify for a stage

But both these classifications (RIFLE and AKIN) are based upon changes predominantly in serum Creatinine concentration. But elevations in creatinine are often delayed with relation to the time of onset of AKI. Furthermore,serum creatinine levels are influenced by factors,other than kidney function such as muscle mass, the volume of distribution, catabolic state, and medications[37].

The International Society of Nephrology and Kidney Disease Improving Global Outcomes (KDIGO) in 2012, brought together experts and gave a definition and staging for AKI, to harmonize the RIFLE and AKIN criteria.This is the recently accepted definition for AKI[38].

Table 2: KDIGO STAGING AND CLASSIFICATION[38]

Stage	Criteria for serum creatinine	Criteria for urine output
1	Increase in SCr by ≥ 0.3 mg/dL within 48 hours or increase in SCr 1.5 to 1.9 times baseline which is known or presumed to have occurred within the prior 7 days	<0.5mL/kg/hr for more than 6hrs
2	Increase in SCr to 2.0 to 2.9 times baseline	<0.5mL/kg/hrfor more than 12hrs
3	Increase in SCr to 3.0 times baseline or increase in SCr to ≥ 4.0 mg/dL or initiation of renal replacement therapy	<0.3mL/kg/hr for 24hrs or anuria for 12hrs

Table 3: CLASSIFICATION OF CAUSES OF AKI [39]

Prerenal causes	Renal causes	Postrenal causes
Haemorrhage	Acute tubular necrosis	Bilateral upper tract obstruction
Gastrointestinal losses	Intratubular obstruction	Obstruction of solitary functioning kidney
Renal losses	Acute Glomerulonephritis	Lower tract obstruction
Skin losses	Acute Interstitial Nephritis	Urinary retention
Renal vasoconstriction	Atheroembolic disease	
Reduced effective arterial blood volume	Acute vascular syndrome	
Loss through third spaces		

BURDEN OF AKI

Before the consensus for use of standard definitions to estimate Acute Kidney Injury, there was wide variation[40, 41]in estimates of the prevalence of disease (1–25%) and mortality (15–60%).

A systematic review[42] including 154 large cohort studies that adopted a KDIGO-equivalent AKI definition from 2004–2012 done by Susantitaphong, et al. (2013) with 3,585,911 subjects totally, estimated the pooled incidence rates of AKI as 21.6% in adults with 95% confidence interval of 19.3 to 24.1% while the Pooled AKI-associated mortality rates were about 23.9% in adults with 95% CI, 22.1 to 25.7% .

In the developed world, AKI has been reported in 3.2–9.6% of hospital admissions and the overall in-hospital mortality is around 20%, and it goes up to 50% in ICU patients[43, 44].

In Scotland, a comprehensive population-based study [45]by AliTet al. (2007) with a geographical population base of 523,390 estimated that the overall incidence of AKI was 2147 per million population per year. In this study, the incidence of AKI and ACRF were 1811 and 336 per million population, respectively. The Median age of AKI was 76 yrs in this study and for ACRF,it was 80.5 years.

A community-based study [46]by Hsu C-Y et al. (2007) using the database of Kaiser-Permanente of Northern California from 1996 to 2003 found an overall incidence of AKI of 384.1 per 100,000 person-years which was non-dialysis requiring while overall incidence of dialysis-requiring AKI was 24.4 per 100,000 person-years.
Sural S et al(1999) [47] in their study for 6 year period on 70 consecutive subjects who developed ARF in the ICU with a mean age of 28.6 years, grouped them into Survivors (7),Nonsurvivors (63). 70% of subjects developed ARF after surgery while 21% due to medical causes. 74.3% required dialysis. Even before ARF onset, organ

failure and sepsis was present in 80% of subjects. The overall mortality was 90%. They concluded that in ICU setting, multiple organ failures is a poor prognostic factor in subjects with ARF.[47]

Jayakumar M et al (2006)[48] studied 1112 ARF cases retrospectively from case records of subjects who were admitted to the hospital from 1995-2004.About 60.1% were males and their mean age was 37.08 with a standard deviation of 3.4 yrs. The Proportion of ARF due to Medical causes was 87.6%. About 8.9% and 3.4% of ARF were due to obstetric and surgical causes respectively.The Commonest medical cause of ARF was Acute diarrheal disease. The overall mortality was 19.6 %.Mortality was associated significantly with high serum creatinine (>440 micromoles) at entry, hospital-acquired ARF, presence of jaundice, sepsis, anemia, oliguria, hypoalbuminemia, About 69% of ARF subjects required dialysis. The preferred form of renal replacement therapy was Hemodialysis [48].

AKI IN ELDERLY GLOBAL STUDIES:

Numerous studies[49-51] have demonstrated that the elderly subjects are more susceptible to developing AKI.The incidence of AKI that is reported in the elderly also varies, according to the type of population studied, their community, and whether the patients were hospitalized in general wards or in ICU.

Pascual J et al (1990)[49] have reported that 35% of all ARF cases have occurred in elderly above 70 years of age and the prevalence in this group was reported to be 3.5 times higher than the younger subjects. The most common causes reported were prerenal in 47%, Acute tubular necrosis (ATN) in 40%. Dehydration was the most frequent cause of prerenal ARF in the elderly (51%). The etiological distribution of ATN was similar in both groups, being of multifactorial origin in most

cases. Mortality was also reported to be higher in elderly in all types of ARF. Total recovery from ARF in older persons was less frequent and slower than in younger subjects.The authors concluded that elderly persons are at high risk for developing ARF but they recommended similar therapeutic approach as younger patients.[49]

Liano F, et al (1996) [40]studied the incidence and etiology of ARF, in 4.2 million subjects aged above 14 years in 13 tertiary care centers in Spain over nine months.The overall incidence of ARF was 209 per million population with 95% C.I. of 195 to 223 per million population while the incidence of Acute tubular necrosis (ATN) was reported at 88 per million population with 95% CI of 79 to 97 per million population. The incidence of prerenal ARF was 46 per million population with 95% CI of 40 to 52 per million population while acute-onset chronic ARF was reported at 29 per million population with 95% CI of24 to 34 per million population and obstructive ARF at 23 per million population with 95%CI 19 to 27 per million population .In this study, the criteria for ARF was fixed as a sudden rise in S.Creatinine concentration> 177 mmol/liter in subjects patients with normal renal function, or a sudden rise of 50% or more in subjects who have a mild-to-moderate chronic renal failure(S.Cr< 264 mmol/liter).The subjects had amean age was 63 with a standard deviation of 17 years. ATN was the commonest cause of ARF responsible for about 45% of cases. About 21% of ARF cases were Prerenal. The proportion of ARF cases causedby acute-onset chronic renal failure and obstructive ARF were 12.7% and 10% respectively.The Corrected Proportion of mortality due to ARF was 26.7%.In about 36% of patients, Dialysis was necessary.[40]

Baraldi A et al (1998) [50]in their study on 109 Acute Renal Failure (ARF) subjects of medical aetiology during a 30-month period with a mean age of 67 +/- 7 years. They identified 4 main causes for ARF.

1. Drug-Related ARF was reported in 39 subjects
2. 33 subjects reported reduced perfusion due to Dehydration, Hypotension
3. 20 subjects had multifactorial etiology
4. 14 subjects had Renal Parenchymal disease identified by Biopsy

The criteria for ARF in their study was a rapid increase of serum creatinine more than 2 mg/dl over the baseline level in normal subjects or in CRF, an increase of two times the pre-existing value. The proportion of subjects requiring dialysis was 64.2%.The main risk factors reported were Elderly age, presence of vascular disease and monoclonal gammopathy.[50]

Sesso R et al (2004) [52] in their study on difference in prognosis between hospital-acquired acute renal failure (n = 154) and Community-acquired ARF (n = 171) in a Tertiary Hospital, in 325 elderly subjects aged 60 years or above found a significant difference in mortality rates between the two groups (59% in hospital-acquired ARF Vs 41% in community-acquired ARF). There was a significant difference between the groups with regards to etiology, pre-existing diseases, dialysis performance, sepsis, failure of organs. After adjusting for significant factors affecting mortality, the mortality risk for hospital-acquired ARF group was 2.23 times [95% CI of 1.21 to 4.08] greater than in community-acquired ARF group.

Significant factors affecting mortality in hospital-acquired ARF group were neurological failure with odds ratio of 2.97, Hematologic failure with OR of 4.30 and oliguria with OR of 12.14, While in community-acquired ARF, they were neoplasia, hepatic disease, cardiac disease, oliguria,cardiovascular failure, and sepsis.[52]

Uchino S et al (2005)[41] in their study on 29269 critically ill ICU subjects in 23 different countries,estimate the period prevalence of Acute Renal Failure from September 2000 to December 2001 as 5.7% (1738 subjects) with 95% confidence

interval of 5.5%-6.0%.1260 out of 1738 ARF subjects had been treated with RRT. A case of ARF was defined as a subject who satisfied any one of the predefined criteria for ARF or had been treated with renal replacement therapy. The most frequent cause of ARF was Septic Shock which contributed to 47.5% of the cases. In about 30% of subjects, there was Preadmission Renal Dysfunction. About 13.8% of subjects at the time of discharge from the hospital required dialysis.This study concluded that the ARF period prevalence was high ranging from 5.5% to 6% with a high overall hospital mortality rate of 60.3% .[41]

Xue JL et al(2006) [53]in their study on hospitalized Medicare beneficiaries between 1992 and 2001 on about 5,403,015 discharges estimated the incidence rate of ARF as 23.8 cases per 1000 discharges and these rates were found to be increasing every year by 11%.The factors significantly associated with ARF were male gender, old age and black race ($P < 0.0001$). In discharges without ARF, the in-hospital death rate was about 4.6% while it was 15.2% in discharges with ARF, and it was about 32.6% in discharges where the secondary diagnosis was ARF. Discharges with ARF were more ($P < 0.0001$) likely to have intensive care and other acute organ dysfunction than those without ARF.[53]

Ali T et al(2007) [45] reported the incidence of AKI as 1811 per million population in a population base of 5,23,390.The incidence of ACRF was 336 per million population. They identified all patients with serum creatinine> / = 150 micromol/L in males and> /=130 micromol/L in females as ARF over a six-month period in 2003.The Median age of subjects with AKI was 76 yrs and it was 80.5 yrs for ACRF subjects.

About 47% of patients had sepsis as a precipitating factor. It also concluded that the RIFLE classification was beneficial in predicting full renal function recovery,

duration of hospital stay, renal replacement therapy requirement($P < 0.001$) and in-hospital mortality ($P = 0.035$).[45]

Ishani A et al(2009)[54]studied the Incidence and Hazard ratios for ESRD in 233,803 elderly subjects aged 67 or above from a 5% random sample of Medicare beneficiaries, without any previous ESRD or AKI in 2000.About 3.1% were diagnosed as AKI on discharge, and 5.3 per 1000 developed ESRD. 25.2%of subjects who were treated for ESRD had a previous history of AKI. The Hazard Ratio for development of ESRD was 41.2 with 95% C.I. of 34.6 to 49.1 for subjects with AKI and CKD after adjusting for factors such as Age, Race, Gender, Presence of Diabetes and Hypertension with relation to those without kidney disease. It was 13.0 with 95% CI of 10.6 to 16.0 for subjects without previous CKD but with AKI and 8.4 with 95% CI of 7.4 to 9.6 for subjects without AKI,but with CKD. This study concluded that Elderly subjects with AKI were at an increased risk for ESRD significantly especially those with the previous history of CKD.[54]

Lo LJ et al(2009)[55]in their study on 556,090 adults from Northern California who were hospitalized over an 8 year period studied the outcome in subjects with an episode of ARF which required Dialysis but with normal / Near normal kidney function and had not developed ESRD within 30 days of discharge from hospital. They observed that dialysis-requiring ARF had been associated independently with a 28 fold increase inthe risk of developing CKD stage 4 or 5 and also with a 2 fold increased risk of death.[55]

Fang Y et al(2010) [43] in their study to estimate the incidence and mortality rate ofAKI among 176,155hospitalized adult subjects in a tertiary hospital in China between 2004 to 2008 found the overall incidence rate of AKI as 3.19%.A relative 50% increase /an absolute increase of 0.3 mg/dl in serum creatinine within 48 h

constituted AKI.The In-hospital mortality rate in subjects with AKI was 19.68% while it was 2.84% in all discharges. Elderly, Admission in ICU, AKI Network score, the need for RRT were independent predictors of hospital mortality in subjects with AKI[43].

James MT et al(2010)[56]in their cohort study on 920,985 adults of Alberta, Canada during 2002 to 2007 observed that proteinuria and eGFR jointly altered the risks of AKI and the succeeding adverse outcomes. They enrolled the Subjects attending O.P. with a measurement of Proteinuria identified by urine dipstick or albumin-creatinine ratio and with a measurement of serum creatinine and who did not need chronic dialysis at baseline.Totally 6520 (0.7%) subjects were enrolled.

The adjusted rates of hospital admission with AKI, need for Dialysis were significantly greater in those with heavy dipstick proteinuria (for all eGFR values).The adjusted death rates and renal outcomes were diminished in those presenting with low baseline eGFR but with heavy proteinuria.

This study concluded that data on proteinuria and eGFR when used together while identifying subjects at risk of AKI, also provides long-term prognostic information.[56]

Garzotto F et al(2011)[57]in their study enrolled 601 incident admissions in their study using a web-based data collection tool from 10 ICU's in Italy. 25 subjects with ESRD were excluded. In the remaining 576 subjects, 59.4% were males and the median age was 66 with IQR of 53-76.Within 24 hrs of admission in ICU, 246 (42.7%) subjects had AKI while 133 developed new AKI later. In this study, the RIFLE-initial class was Risk in 54.1% of subjects, Injury in 26.1% while Failure in 19.8% and the progression to a worse RIFLE class was seen in 30.8% of AKI subjects.

Crude ICU mortality was higher in AKI subjects when compared with others (28.8 vs8.1%, p <0.001). The Median length of ICU stay was also significantly longer (7 in AKI vs 3 days in non-AKI; p < 0.001). Both Crude ICU mortality, length of stay increased when the severity of AKI was greater. This study also concluded that use of RIFLE to stage severity of AKI was optimal.[57]

Gong Y et al(2012) [58] in their study on 99 elderly subjects with AKI, aged 65 and above, concluded that presence of concomitant disease and MODS (multiple organ dysfunction syndromes) were significant independent risk factors responsible for Mortality in Elderly with AKI. Ischemia (53.34%) followed by surgery (33.33%), sepsis (10.10%) and intake of nephrotoxic drugs (3.03%) were the main cause of AKI in elderly. The Mortality Rate in Elderly subjects with AKI was about 42%.[58]

Li QL et al(2013)[59]in their study on 232 elderly (215 males, 17 females) AKI patients with a mean ageof 86.7(+/- 5.3)years from June 2008 to December 2009 at Chinese PLA General Hospital observed that 16.4% of subjects died within 28 days after AKI while 24.6% died within 3 months. Infection (43.1%), Hypovolemia (19.0%), use of nephrotoxic drugs (16.8%) and cardiovascular events (15.1%) were major causes of AKI.

Low BMI, hypoalbuminemia, peak serum level of creatinine more than 246.5 micromol/L , oliguria and mechanical ventilation were the significant prognostic factors in those patients dying within 28 days after AKI while low BMI, hypoalbuminemia and high blood level of urea nitrogen were significant prognostic factors in those dying from 29 days to 3 months after AKI.[59]

Wen J et al (2013)[60] reported the overall incidence of AKI in very elderly subjects (older than 80) as 14.8%. In those subjects, infection was the major cause.

Hypovolemia, use of nephrotoxic drugs (Antibiotics were the most common), cardiac dysfunction and respiratory failure were the most common causes of AKI by multifactorial analysis.

For one year survival, MODS, malnutrition, gastrointestinal bleeding, the absolute increase in SCR were the significant risk factors independently. Use of Alpha-ketoacid appeared to be a protective factor with OR of 0.656. For 90 days endpoint, MODS, heart failure, and gastrointestinal bleeding were significant independent risk factors by Cox proportional hazard model.[60]

Turgutalp K et al (2017)[61]in their study of subjects aged 65 and above, admitted to their hospital between May 2012 to 2013, reported an overall incidence of 7.3 %(N=3229). The incidence of AKI-DO (Acute Kidney Injury Developing outside the hospital) that required hospitalization in elderly subjects (65-75 years old) was5.8 % (136/2324) and in very elderly subjects (>75 years old) was 11 % (100/905)($p < 0.001$).The AKI-DO incidence was high, especially in the male gender.

Mortality rates were higher in very elderly subjects (23.5 %) compared to elderly subjects (31 %). The risk factors that were significant for development of AKI-DO in elderly were use of ACEI, ARB, radiocontrast agents and NSAIDS.[61]

Table 4: WORLDWIDE INCIDENCE OF AKI IN THE ELDERLY

AUTHOR WITH PLACE	INCIDENCE
1. Turgutalp K et al, Turkey (2017) [61]	7.3% (in aged ≥ 65)
2. Wen J et al, China (2013) [60]	14.8% (in aged >80) 2.76% (in aged 65-80)
3. Ishani et al., USA (2009)[54]	3.1%
4. Ali et al., Scotland (2007)[45]	AKI- 1,811 cases per million population ACRF- 336 per million population
5. Baraldi et al., Italy (1998) [50]	10 times higher in aged ≥ 65 vs. ≤ 65

AKI IN ELDERLY, INDIAN STUDIES

The literature on AKI in elderly in India is limited compared to other parts of the world.

Arora P et al(1993) [(11)] in their study on 139 ARF subjects, reported that 65% of geriatric ARF were due to surgical causes and in younger patients, 55.1% were due to medical causes. Out of the 139 subjects, 29.4% were elderly and their mean age was 67.1 years while 70.6% were younger and their mean age was 32.3 years. In geriatric subjects, drugs and sepsis were the predominant medical causes (85.7%). Prostate-related problems either due to obstruction or after transurethral resection of prostate contributed to the surgical cause of Geriatric ARF in 74%. Totally Nephrotoxic drugs contributed to Geriatric ARF in 51% of subjects either alone or as a combination of other predisposing factors.

In the elderly, there was a significant delay in recovery from ARF, that is normalization of serum creatinine, when compared to the younger subjects (32 Vs 11.4 days, $P < 0.001$). But this study concluded that there was statistically not significant difference in Mortality rates in the elderly as compared to the younger subjects (9.75% Vs 6.1%).[(11)]

Prakash J et al(1997) [(62)]reported that 80% of geriatric ARF was due to medical causes and 20% was due to surgical causes, in their study on 638 ARF subjects of diverse etiology over 9 years. About 15% of subjects were classified as elderly ARF with a mean age of 72.5 years. They did not observe ARF occurring in association with multiorgan failure. This study reported a Mortality of 25%.[(62)]

Kohli HS et al (2000)[(63)] in their study on 31860 subjects admitted during a 12 month period, of which 4176 (13%) were elderly (>60 years), observed the incidence of ARF related to treatment in the elderly to be 1.4%. The contribution of

Nephrotoxic drugs towards the development of ARF was 66%, 47.5% each due to sepsis and hypoperfusion, postoperative ARF contributed towards 25.4% and contrast medium was responsible in16.9% of subjects. 15.23% of the subjects needed dialysis. 25.4% of the subjects died.Mortality was significantly higher in those with ARF compared to those without ARF (25.4 vs 12.5%, P=0.03). Sepsis with OR of 43, oliguria with OR of 64, Hypotension with OR of 15 were the significant predictors of poor patient outcome independently.[63]

Lou LM et al (2002)[64]reported an incidence of 1,238 cases per million per year in their study. 99 elderly subjects were identified with ARF which contributed to 1.78% of hospital admissions. The most common cause of ARF was Preexisting chronic diseases especially Hypertension (54%), Diabetes (39%). The cause of ARF was Prerenal in 60% of subjects, renal in 31% while it was post-renal in 9%. The Proportion of Oliguria or Anuria in ARF subjects was 44.4%. The mortality rate was 36.4%. Comorbid conditions, Oliguria, Anuria, Renal ARF, Concentration of serum Albumin were the significant factors affecting the mortality rate. They concluded that important aetiological factors in those with ARF outside hospital are Volume depletion, pharmacological treatment and associated cardiovascular pathology.[64]

Kohli HS et al (2006)[65]in their study of 4255 elderly subjects who were either hospitalized or attending outpatient renal clinic reported236 (5.5%) cases with renal failure. Their Mean age was 65.1 with a range of 60-86 years.The Proportion of CRF was 58.1% while ARF was 29.2% and RPRF was observed in 12.7% of Renal failure subjects. 38.7% of CRF subjects presented with ESRD. Sepsis was responsible for ARF in 75.4% of subjects. Acute interstitial nephritis (AIN) was the commonest cause of RPRF seen in 33.3% of subjects.(65)

Mahajan S et al (2006)[14]in their study on datafrom 454 elderly ARF patients aged 60 and above in a tertiary superspecialty center in North India with a mean age of 66.4 years observed that volume depletion was the most common factor precipitating ARF in 33% of cases. Others were Infection/sepsis (21.6%), drugs (11.5%).

The proportion of ARF patients with oliguria (<400 ml/day) was 31.8%. 33.5% required RRT. The most frequent RRT given was Acute peritoneal dialysis (62.5%). Mortality was observed in 41.2% (187) of subjects. Although recovered from ARF, 56 died out of the 187.

The factors significantly associated with poor outcome were presence of chronic illness, oliguria, presence of sepsis, cardiac failure,need for RRT and increasing number of organ failure.[14]

Kohli HS et al (2007)[66]in their study on 33,301 adults hospitalized at a tertiary care center over 1 year in a developing country analyzed the predictors of mortality in elderly with ARF. Totally, 69 elderly (1.6%) had ARF. Out of 4255 elderly subjects, 1.6% (69) had ARF. 60.9% of the elderly ARF patients died.Various predictors analyzed were hospital-acquired ARF, causative factors of ARF, preexisting hypertension and diabetes mellitus, severity of renal failure (initial and peak serum creatinine, need for dialysis), and complications of ARF: infection during the course of illness; serum albumin levels and critical illness defined as presence of two or more organ system failures excluding renal failure.

Age > 60 years, was an independent predictor of mortality in the whole group (odds ratio 5.6, P = 0.001). Infection which occurs during the course of ARF (OR9.72) and critical illness (OR9.97)(Two or more organ system failures exceptrenal failure) were the significant independent predictors of mortality on multivariate analysis although several factors were significant predictors in univariate

analysis like Hospital-acquired ARF, sepsis leading to ARF, Requirement of dialysis (72.5%, P = 0.022), Development of infection during the course of ARF (87.9%, P = 0.000) and in those with a critical illness (90.0%, P = 0.00). [66]

Kohli HS et al (2007) [9] in their study to determine the predictors of mortality in Acute renal failure (ARF) in the developing world on 33,301 hospitalized adults admitted during the study period of one year, found the proportion of subjects who got admitted with ARF or developed ARF after hospitalization was 0.88%. The Mean age of the subjects with ARF was 43.9 +/- 16.9 yrs with a range of 18 to 86 years. The most common cause of ARF was sepsis (63.26%). In comparison with younger subjects, elderly subjects with ARF had a significantly higher proportion of pre-existing diseases like DM, HTN, Malignancy, Diseases of the Respiratory system, Cardiovascular system, Central Nervous system.

The factors significantly associated with mortality in ARF on univariate analysis were Age > 60 yrs, sepsis, hypoperfusion, Diseases of various systems, DM, oliguria, bleeding, critical illness and infection during ARF were predictors of poor outcome. But with multivariate analysis, only critical illness (OR of37.3), Age > 60 years (OR of 5.6), and sepsis-causing ARF (OR of 2.6) were found to be significant predictors of mortality independently.[9]

Mahesh E et al. (2017) [13]in their cross sectional study in a tertiary care teaching hospital on elderly population (>60 yrs) from South India with characteristics of AKI (RIFLE) 1 at admission and those developing it following admission reported the mean age of subjects was 70.5 years. In their study, 59% were males while 41% were females. In the age group of 60–69 years, the peak incidence of AKI was observed. The Proportion of risk factors observed in subjects were 44% with diabetes, 35% had hypertension, ischemic heart disease in 19%, and 12% had

chronic obstructive pulmonary disease. Seven days was the average duration of their hospital stay. Medical factors were the cause in 87% while surgical causes accounted for 11%, and gynecological causes in 2%. Among medical causes, sepsis was most common. Pneumonia and urosepsis were reported as the most common causes of sepsis leading to AKI. In comparison to surgical causes, AKI of medical aetiology had a better outcome. About 28% of subjects required dialysis. 44 received hemodialysis while 12 received peritoneal. The overall mortality was 15% and the mortality rate was higher among postsurgical AKI in comparison to the AKI of medical aetiology(P<0.001) and in patients who required dialysis. The independent predictors of mortality were Oliguria, the need for dialysis and postsurgical AKI.

COMMON CAUSES OF AKI IN ELDERLY:

The kidneys with aging undergo a number of important changes which are age-dependent[6, 67].

The causes of acute kidney injury in elderly can be grouped into three categories:

1. Prerenal,
2. Renal and
3. Postrenal.

In elderly, ischemia (hypovolemia/hypotension) is responsible for about 50% of the AKI cases as reported by Gong Y et al (2012) [58], followed by surgery (33.3%), sepsis (10%), and the nephrotoxins (3%).

The renal mass decreases with aging. At the age of about 70 years, the kidneys would have lost between 30% to 50% of their cortical glomeruli mainly due to ischemic changes. The remaining glomeruli may manifest some degree of sclerosis.

Other changes that occur with aging include a reduction in the number and size of tubules, a decrease in glomerular filtering surface area as a result of an increasing proportion of mesangial cells, increasing tubulointerstitial fibrosis, thickening of glomerular and tubular basement membranes, arteriosclerosis and decreased afferent arteriolar luminal area.

Most of the information for aetiologies for AKI in the elderly is mainly based on clinical judgment since the incidence of renal biopsy in the elderly is low and the data is very limited[58, 68]. In many other larger studies[69][70], Acute tubular necrosis was the most common cause of AKI in the elderly (39%). Prerenal conditions accounted for about 30% of AKI.

Table 5:CLASSIFICATION OF CAUSES OF AKIIN ELDERLY [(71)]

PRERENAL CAUSES	RENAL CAUSES	POSTRENAL CAUSES
1.True volume depletion • Blood loss • Insensible losses • Adrenal insufficiency • Gastrointestinal losses Vomiting Diarrhea • Genitourinary losses Diuretics Osmotic diuresis (hyperglycemia) Third spacing	1. Acute Tubular Necrosis 2. Acute Interstitial Nephritis 3. Acute Renal Vascular Disease 4. Acute Glomerulonephritis	**1.Upper tract obstruction** Nephrolithiasis Blood clots Papillary tissue Pelvic neoplasms Endometriosis Retroperitoneal processes Neoplasms Adenopathy Fibrosis Hematoma Gastrointestinal Neoplasms Radiation treatment
2. Decreased effective arterial blood volume Heart failure Cirrhosis Nephrotic syndrome		**2. Lower tract obstruction** Urethral Strictures Nephrolithiasis Blood clots Phimosis / Paraphimosis Prostatic processes Benign hypertrophy Carcinoma Calculi Bladder processes Carcinoma Calculi Neurogenic bladder
3.Medications Nonsteroidal anti-inflammatory drugs ACEI/ARB Calcineurin inhibitors		
4.Hypercalcemia		

In Prerenal Disease or Prerenal Azotemia, renal hypoperfusion leads to a decrease in the glomerularfiltration rate. It occurs as an adaptive response to various extrarenal insults such as volume depletion, systemic hypotension, significant renal vascular stenosis or thrombosis, severe systolic or diastolic cardiac failure, and activation of the neurohumoral axis increasing renal vascular resistance. It is a leading cause of AKI in the geriatric populations. Prerenal AKI follows only if the fall in

renal perfusion exceeds the ability of these counter-regulatory systems to maintain a near-normal GFR. Prerenal azotemia is only responsible for the bulk of community-acquired AKI. Though classically associated with hypovolemia, prerenal AKI also develops in the background of effective intravascular volume depletion associated with congestive heart failure and other organ failures.

Renal disease is intrinsic acute kidney injury. Acute tubular necrosis is the most frequent cause of intrinsic acute kidney injury[(40)]. In the absence of a kidney biopsy, it is often only a presumptive diagnosis. Other causes include acute glomerulopathies which are often rapidly progressive, acute vasculitis, acute interstitial nephritis. Contrast-induced nephropathy (CIN) is a major cause of AKI in hospitalized elderly patients[(63)].

Postrenal disease or obstructive AKI is a more common entity in the aged than in the young [(69)], accounting for 9% to 30% of cases[(72)]. It usually occurs after obstruction of the urinary tract. In contrast with intrinsic acute kidney injury, when prerenal or postrenal causes are reversed, it usually results in prompt recovery of function, whereas late correction can lead to kidney damage. The most frequent causes of postrenal include benign prostatic hypertrophy (BPH) or prostate cancer, retroperitoneal adenopathy or malignancies, pelvic neoplasms, and neurogenic bladder.

The following reasons attribute to the higher incidence of AKI in elderly persons[(67, 71, 73)]

A) Comorbidities that accumulate with age may facilitate AKI such as Renovascular disease, Congestive heart failure.

B) Comorbidities that may necessitate procedures, drugs or surgery which may function as kidney stressors and nephrotoxins;

C) Age-dependent structural, functional changes over time

EVALUATION OF AKI:

To evaluate a case of AKI, a detailed history,and physical examination is important to differentiate the various aetiologies of AKI such as Prerenal, Renal, and Postrenal. The Prerenal, Renal and Postrenal cases should be evaluated in the subject. The Subject should be examined for volume status, any evidence of atheroembolic disease and extra-renal manifestations of systemic diseases[7, 17].

Initially, along with the baseline blood investigations like Serum urea, Creatinine, Complete Blood Count, a complete analysis of the urine including urine output, urine sediments, urine creatinine, a bedside postvoid bladder sonogram and then ruling out lower urinary tract obstruction by passing a urinary catheter, and renal ultrasound should be done.

If the etiology of AKI remains unclear even after these investigations, then consideration of a kidney biopsy is warranted. A kidney biopsy is a relatively low-risk procedure. It is well tolerated even by elderly subjects. Approximately 30% of diagnoses, in elderly more than 60 years, were altered in one case series of kidney biopsies.

The abrupt change in concentration of serum creatinine is usually used to clinically diagnose the AKI. But the concentration of serum creatinine is dependent on the steady state between creatinine release from muscles and its excretion through the kidneys[68]. The concentration usually reduces with age as the muscle mass declines with age, if GFR is unchanged. After an acute injury, there may be a decline in GFR, the rate and magnitude of the rise in serum creatinine may be diminished in elderly because of the smaller muscle mass.

In the recent years, there has been an increase in the use of novel biomarkers to detect AKI.

Herget-Rosenthal et al. (2004) [74] in their study, demonstrated a 50% rise in serum cystatin C in elderly patients with a mean age 70 ± 8 years for diagnosing AKI one or two days prior to clinical AKI (which is defined by a 50% increase in serum creatinine). Cystatin C is an endogenous protease inhibitor. Cystatin is produced at a constant rate by nucleated cells. It is excreted exclusively by glomerular filtration. Serum levels are stable with changes in age, muscle mass, diet and physical activity. Several urinary biomarkers of AKI that reflect tubular injury rather than changes in GFR have been studied.

For example, in a recent meta-analysis done by Han W et al. (2008) [75]on the performance of NGAL(Neutrophil gelatinase-associated lipocalin) as a biomarker, for the early diagnosis of AKI, it was observed some effect modification by age. The performance was better in children in comparison to adults.

Thus no single biomarker has been excellent, in elderly, for the early diagnosis of AKI. It is also unknown whether the performance of any AKI biomarkers differ in the old or very old compared to younger adults. But in the future, a panel of multiplebiomarkers[76]may be needed for diagnosing AKI early, accurately in older adults.

PREVENTION AND TREATMENT OF AKI IN ELDERLY

The prevention of AKI in elderly involves recognizing their increased vulnerability, because of the structural and functional changes that occur with aging.

1. Avoiding use of nephrotoxic agents[77],
2. Warranting adequate volume expansion, before any known use of stressors such as administration of I.V. contrast or use of nephrotoxic medications.

3. While calculating GFR, MDRD(Modification of Diet in Renal Disease) [78]Study equation or the CKD-EPI equation to be used, in order to determine the high risk status. Although Prevalence of CKD by this method may be highly estimated.
4. Utilizing of off-pump coronary artery bypass surgery[79]in those subjects who have a high risk for AKI.

But Once AKI is established, then there are general supportive measures such as hemodialysis, which should not be withheld based solely on old age. Since there is no evidence supporting inferior outcomes in elderly patients who require dialysis compared to Nondialysis AKI., Dialysis should be done whenever needed.

There is also no sufficient evidence in the literature to support for a specific modality or intensity of RRT during AKI.

METHODOLOGY

Study design: The current study was a cross sectional study.

Study setting: The study was conducted in the department of General medicine KLES Dr Prabhakar Kore Hospital, Belagavi.

Study population: The study population included was all the elderly subjects (65 years and above), who were diagnosed with Acute Kidney Injury

Study duration: The data collection for the study was done between --- 2016 January to --- 2017 December, for a period of 1 year.

Inclusion criteria:

- All the elderly subjects of 65 years and above
- Both males and female
- With Acute Kidney Injury without history of chronic kidney disease

Exclusion criteria:

- All patients below 65 years of age with AKI
- All patients of 65 years and above having chronic kidney disease

Sample size: The sample size was calculated assuming the expected proportion of any particular etiology of AKI I study population as --%, as per the study by – et al. The other parameters considered for sample size calculation were 5% precision and 95% confidence level. The sample size was calculated using the following formula.

$$n = \frac{Z^2 P(1-P)}{d^2}$$

Where n = Sample size

Z = Z statistic for a level of confidence,

P = Expected prevalence of proportion

(If the expected prevalence is 20%, then P= 0.2), and

d = Precision (If the precision is 5%, then d=0.05).

As per the above-mentioned calculation, the required sample size was --- 190 subjects. To account for a non-participation rate of 5%, it was decided to include a total 200 subjects in the study.

Sampling method: All the study subjects were recruited into the study by convenient sampling.

Study procedure: All the subjects admitted in the study setting and have a likelihood of AKI, underwent a clinical evaluation by history, clinical examination as per the institutional protocol. Relevant medical records were reviewed to confirm the presence of AKI.

AKI was defined as per the Harrsion's Manual of Medicine (2006).

Informed consent to participate in the study was obtained after confirmation of the AKI. Subjects willing to provide consent were included in the study and underwent afurther round of evaluation. All the relevant parameters were documented onto a structured proforma.

Ethical considerations

Clearance was obtained from the ethical committee of the institution. Written and informed consent was sought from the patients and their attendants. They were

given the option of quitting from the study if so desired by them. No element of compulsion was exerted. All data were kept confidential.

Statistical methods:

The relevant parameters analyzed were demographic variables like age, gender, the aetiology of AKI, risk factors for AKI like Diabetes mellitus, hypertension etc. laboratory parameters like hemoglobin, Total count, platelet count etc.. Renal function parameters like blood urea nitrogen and serum creatinine were also documented and analyzed. The morphology of the kidney in ultrasonography was also documented. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram, and box plots.

USG Ultrasonography, Risk factors, Diagnosis category, Chief Complaints, Renal biopsy all are Descriptive analysis in the study population.

P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.(80)

RESULT

A total of 200 elderly patients with AKI were included in the study.

Table 6: Descriptive analysis for Age in study population (N= 200)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
Age	70.46 ± 6.02	68.00	65.00	93.00	69.62	71.30

The mean age **was** 70.46 years with a standard deviation of 6.02. The youngest person was 65 years old and the oldest person was 93-years-old.(Table6)

Table 7: Descriptive analysis of Age group in study population (N=200)

Age group	Frequency	Percent
At 65	44	22.00%
66-70	86	43.00%
71-75	34	17.00%
76-80	21	10.50%
81-85	12	6.00%
86 and above	3	1.50%

Majority (43%) subjects belonged to 66-70-year age group, followed by up to 65-year age group (22%).(Table7& figure 2)

Figure2: Bar chart of Age distribution in study population (N=200)

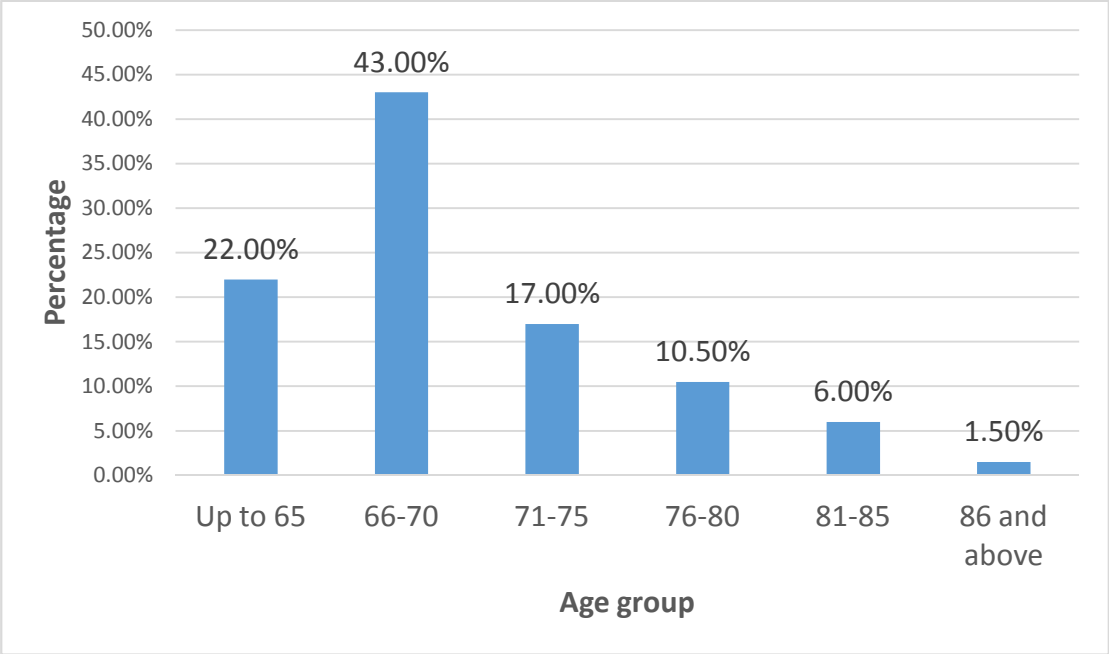


Table 8: Descriptive analysis of Gender in study population (N=200)

Gender	Frequency	Percentage
Male	132	66.00%
Female	68	34.00%

Among the study population Males constituted 66% and Female constituted 34%. (Table 8)

Figure3: Pie chart of Gender distribution in study population (N=200)

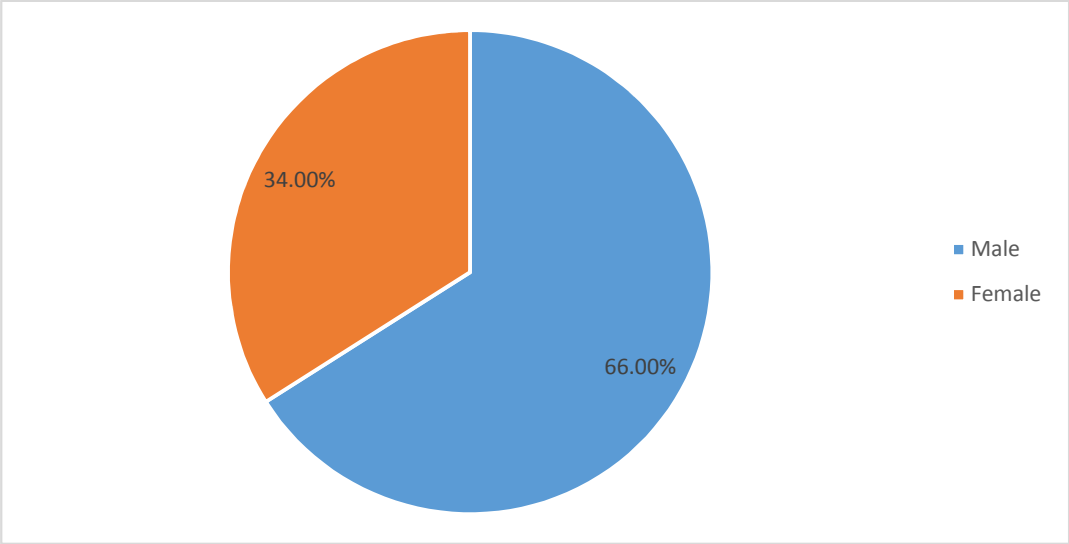


Table 9: Descriptive analysis for Serum Creatinine in study population (N=200)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
Serum Creatinine	4.72 ± 2.91	3.89	1.35	20.45	4.31	5.13

The Mean Serum Creatinine was 4.72 mg/dl with a standard deviation of 2.91. The minimum and maximum values were 1.35 and 20.45 respectivelyin the study population. (Table 9)

Table 10: Descriptive analysis for platelet count in study population (N=200)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
Plateletcount	242334.5 ± 127547.32	218500	18900	720000	224549.51	260119.49

The Mean platelet count was 242334.5 and the standard deviation was 127547.32. The minimum and maximum 18900 and 720000 respectively in the study population. (Table 10)

Table 11: Descriptive analysis for Total leucocyte count in study population (N=200)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
Total leucocyte count	13792.39 ± 6366.12	12500	2900	38300	12897.89	14686.89

The Mean **Total leucocyte count** was 13792.39 and standard deviation 6366.12. The minimum value was2900 and themaximumvalue was 38300 in the study population. (Table 11)

Table 12: Descriptive analysis for BUN Urea in study population (N= 200)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
BUN Urea	120.68 ± 63.01	108.50	20.00	344.00	111.89	129.46

The Mean BUN Urea was 120.68 and standard deviation 63.01. The minimum 20 and maximum 344(95% CI 111.89–129.46) in the study population. (Table12)

Table 13: Descriptive analysis for HB in study population (N=200)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
HB	11.16 ± 1.98	10.90	6.90	16.90	10.89	11.44

The Mean HB was 11.16 and standard deviation 1.98. The minimum 6.90 and maximum 16.90 (95% CI 10.89–11.44) in the study population. (Table13)

Table 14: Descriptive analysis of DM in study population (N=200)

DM	Frequency	Percentage
Yes	68	34.00%
No	132	66.00%

Among the study population, 34% ofsubjects had Diabetes Mellitus. (Table 14& figure4)

Figure 4: Bar chart of DM distribution in study population (N=200)

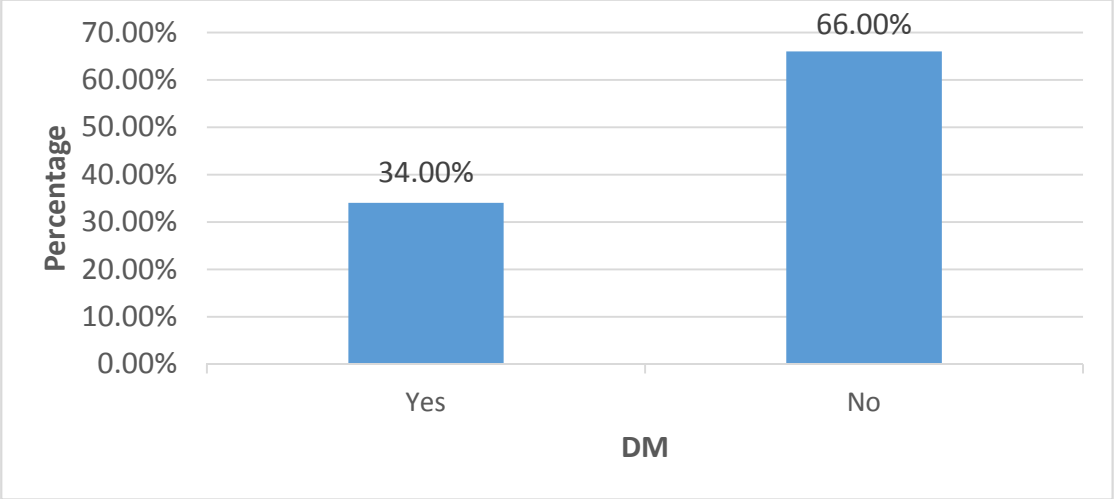


Table 15: Descriptive analysis of HTN in study population (N=200)

HTN	Frequency	Percentage
Yes	73	36.50%
No	127	63.50%

Among the study population, 36.50% subjects had Hypertension (Table 15& figure 5)

Figure 5: Bar chart of HTN distribution in study population (N=200)

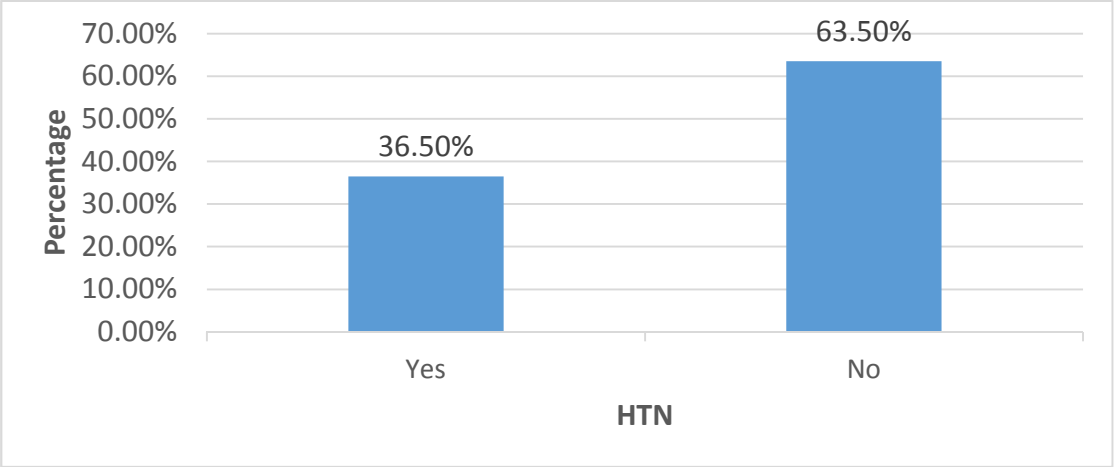


Table 16: Descriptive analysis of IHD in study population (N=200)

IHD	Frequency	Percentage
Yes	22	11.00%
No	178	89.00%

Among the study population, 11% subjects had IHD.(Table 16& figure 6)

Figure 6: Bar chart of IHD distribution in study population (N=200)

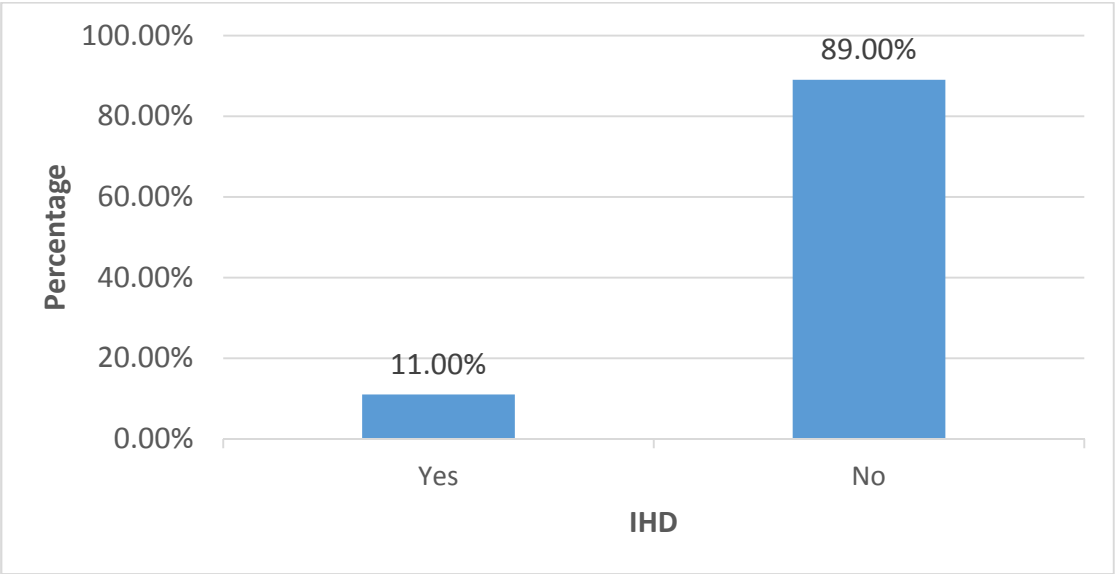


Table 17: Descriptive analysis of COPD in study population (N=200)

COPD	Frequency	Percentage
Yes	4	2.00%
No	196	98.00%

Among the study population,2% subjects had COPD.(Table 17& figure 7)

Figure7: Pie chart of COPD distribution in study population (N=200)

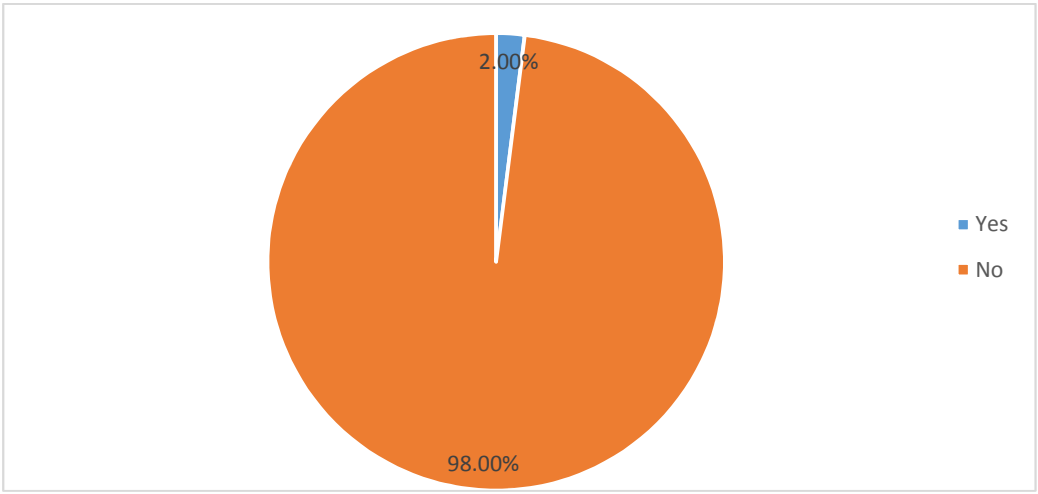


Table 18: Descriptive analysis of RVD in study population (N=200)

RVD	Frequency	Percentage
Yes	2	1.00%
No	198	99.00%

Among the study population,1% subjects had RVD. (Table 18)

Table 19: Descriptive analysis of DCM in study population (N=200)

DCM	Frequency	Percentage
Yes	2	1.00%
No	198	99.00%

Among the study population, 1% subjects had DCM. (Table 19)

Table 20: Descriptive analysis of LVF/LVD in study population (N=200)

LVD	Frequency	Percentage
Yes	1	0.50%
No	199	99.50%

Among the study population, 0.50% subjects had LVD. (Table20)

Table 21: Descriptive analysis of ALD in study population (N=200)

ALD	Frequency	Percentage
Yes	1	0.50%
No	199	99.50%

Among the study population, 0.50% subjects had ALD. (Table21)

Table 22: Descriptive analysis of CVA in study population (N=200)

CVA	Frequency	Percentage
Yes	3	1.50%
No	197	98.50%

Among the study population, 1.50% subjects had CVA.(Table22)

Table 23: Descriptive analysis of IWMI in study population (N=200)

IWMI	Frequency	Percentage
Yes	1	0.50%
No	199	99.50%

Among the study population, 0.50% subjects had IWMI.(Table23)

Table 24: Descriptive analysis of Anemia in study population (N=200)

Anemia	Frequency	Percentage
Yes	2	1.00%
No	198	99.00%

Among the study population,1% subjects had Anemia.(Table24)

Figure 8: Bar chart ofRisk factors distribution in study population (N=200)

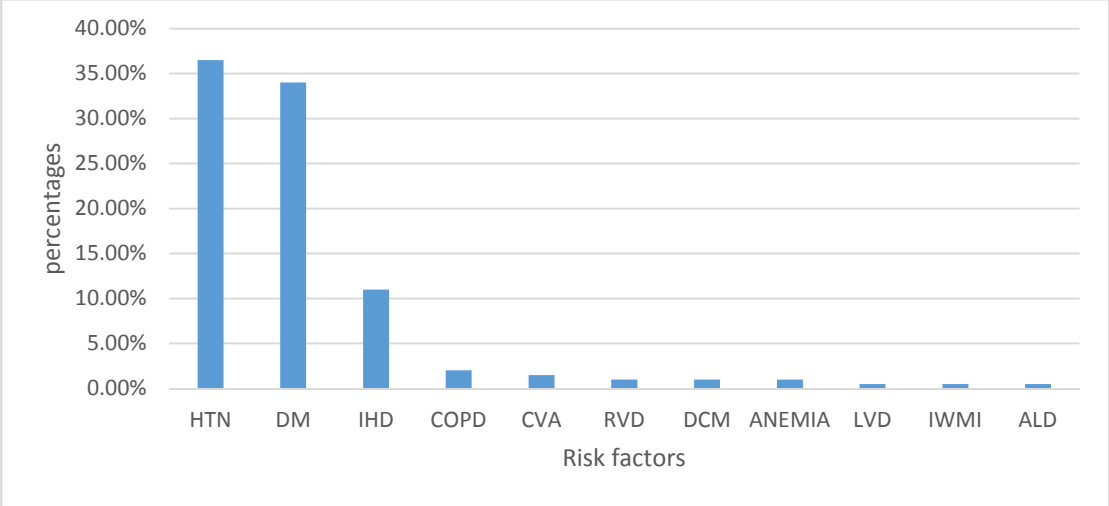


Table 25: Descriptive analysis of Chief Complaints in study population (N=200)

Chief Complaints	Frequency	Percent
Pain Abdomen	114	57.00%
Anuria	113	56.50%
Oliguria	86	43.00%
Fever	83	41.50%
LooseStools/Vomitting	57	28.50%
Burning Micturition	23	11.50%
Breathlessness	22	11.00%
Cough	20	10.00%
Other minor Symptoms	16	8.00%
Weight Loss	4	2.00%
Anemia	2	1.00%
Burns	1	0.50%

Among the study population, the chief complaints of pain abdomen were the most common presenting complaint seen in 57% of subjects. The proportion of subjects presenting with anuria, oliguria, and fever was 56.50%, 43%, 41.50 % respectively. The other common symptoms were Loose stools with vomiting, Burning Micturition, breathlessness, cough.

Figure9: Bar chart Chief Complaints distribution in study population (N=200)

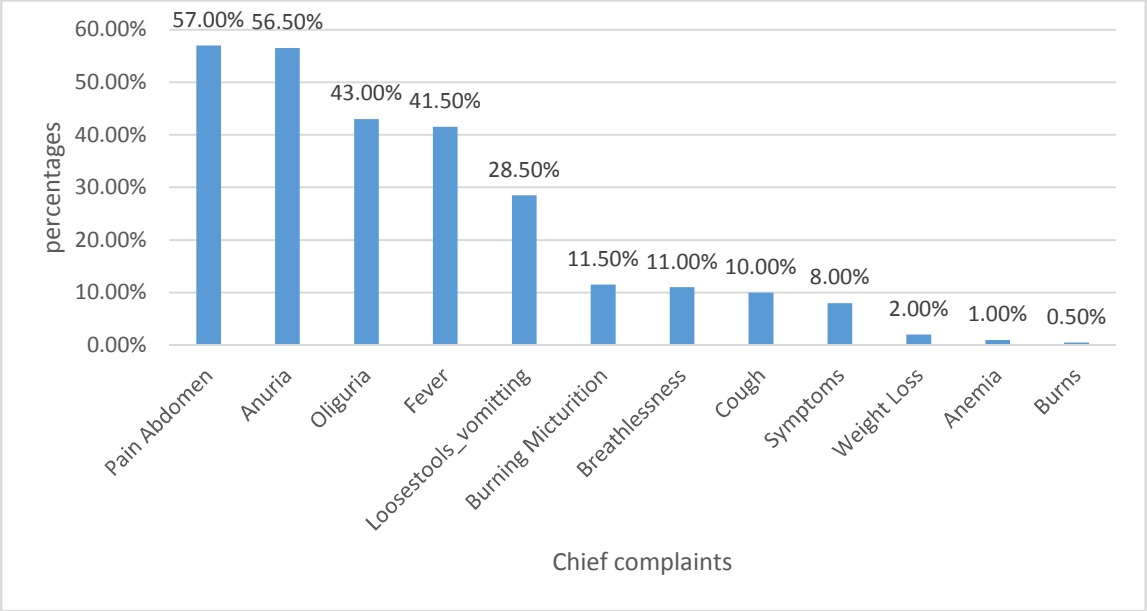


Table 26: Descriptive analysis of USG (Ultrasonography) in study population (N=200)

Ultrasonography	Frequency	Percent
Normal Kidney Size, no urinary obstruction	140	70.00%
Pyelonephritis	28	14.00%
Renal Calculi	17	8.50%
Benign Prostatic Hypertrophy (BPH)	3	1.50%
Chronic Liver Disease	2	1.00%
Small Bowel Perforation	1	0.50%
Chronic Calcific Pancreatitis	1	0.50%
Emphysematous pyelonephritis	1	0.50%
Acute Pancreatitis	1	0.50%
Acute Hepatitis	1	0.50%
Carcinoma cervix with metastasis to ureters	1	0.50%
Periampullary carcinoma with biliary cirrhosis	1	0.50%
Perforated Appendix	1	0.50%
Carcinoma Colon Obstructing on ureters	1	0.50%
B/L moderate HN, with entire HU with B/L Multicystic kidneys.	1	0.50%

Among 140 (70%) subjects, ultrasonography showed normal kidney size and no urinary obstruction.The common abnormalities identified in ultrasound were pyelonephritis in 14%,renal calculi in 8.5% and benign prostatic hypertrophy (BPH) in 1.50% of subjects respectively.(Table 26)

Table 27: Descriptive analysis of Diagnosis in study population (N=200)

Diagnosis category	Frequency	Percent
Acute Gastroenteritis	57	28.50%
Pyelonephritis	32	16.00%
Urosepsis	24	12.00%
Obstructive Uropathy	17	8.50%
Pneumonia	13	6.50%
NSAIDs induced	10	5.05%
Lower Limb Cellulitis	8	4.00%
Chronic Liver Disease	4	2.00%
COPD	4	2.00%
Benign Prostatic Hypertrophy	3	1.50%
Dengue fever	3	1.50%
Pancreatitis	3	1.50%
Acute Bronchitis	3	1.50%
Snake Bite	2	1.00%
Peritonitis due to perforation	2	1.00%
Viral Fever	1	0.50%
Carcinoma colon	1	0.50%
Burns	1	0.50%
Post Coronary Angiography	1	0.50%
Bulbous Pemphigoid	1	0.50%
Parotid abscess	1	0.50%
Diabetic Ketoacidosis	1	0.50%
Multiple myeloma	1	0.50%

The most common diagnosis in the study population was Acute Gastroenteritis in 28.50% of the study population, followed by Pyelonephritis in 16% of the population and urosepsis in 12% of the population. The other common diagnoses were Obstructive Uropathy in 8.5%, Pneumonia in 6.5%, NSAID induces nephropathy in

5.05% and lower limb cellulitis in 4% of the subjects. Various other miscellaneous conditions contributed to the rest of the cases.(Table 27)

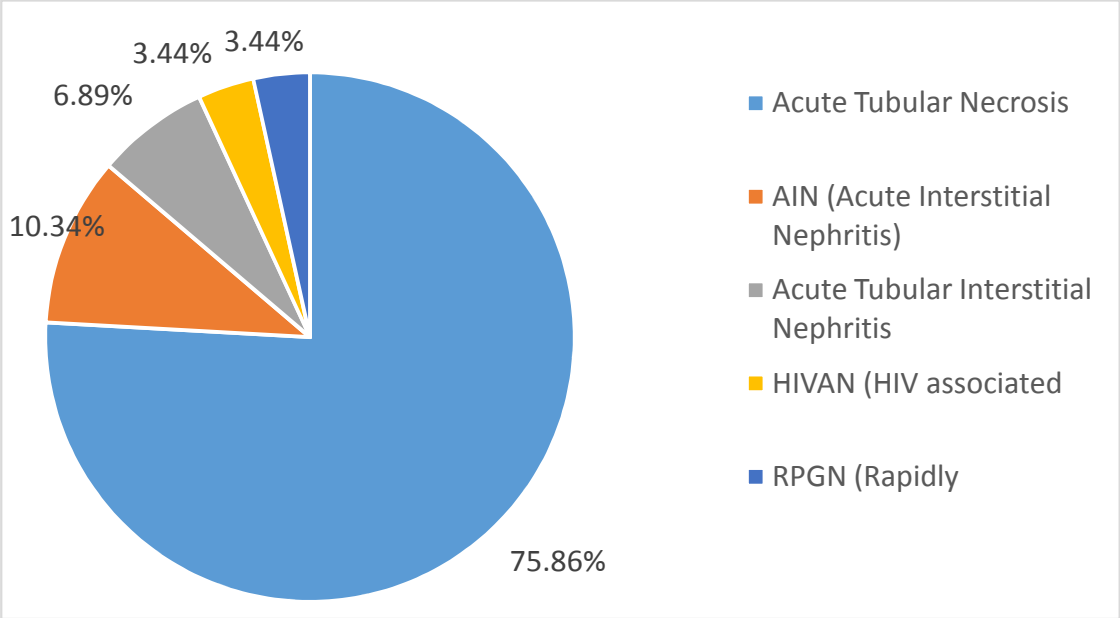
Only 29 patients underwent a renal biopsy in the study.

Table 28: Descriptive analysis of RENAL BIOPSY findings in study population (N=29)

RENALBIOPSY	**Frequency**	**Percentage from 29**
Acute Tubular Necrosis	22	75.86%
AIN (Acute Interstitial Nephritis)	3	10.34%
Acute Tubular Interstitial Nephritis	2	6.89%
HIVAN (HIV associated nephropathy)	1	3.44%
RPGN (Rapidly progressive glomerulonephritis)	1	3.44%

Among the 29 subjects who underwent renal biopsy, Acute Tubular Necrosis was seen in 22 (75.86%),AIN (Acute Interstitial Nephritis) was seen in 3 (10.34%), Acute Tubular Interstitial Nephritis was seen in 2 (6.89%) and HIVAN (HIV associated nephropathy) and RPGN (Rapidly progressive glomerulonephritis) were seen in 1(3.44%) subjects each.

Figure 10: Pie chart of RENALBIOPSY distribution in study population (N=29)



DISCUSSION

In the elderly, the incidence of AKI is very high. The elderly AKI patients with their associated co-morbidities usually pose a different set of challenges to the treating physician both diagnostically and therapeutically. AKI is associated with an amplified risk of mortality and morbidity in the elderly.

Demographic profile:

In our study, a total of about 200 elderly patients with AKI were included in the study. The mean age of subjects in our study was 70.46 years with a standard deviation of 6.02 while the Median age of AKI was 76 yrs in a comprehensive population-based study [45] by **AliTet al. (2007)** with an interquartile range of 66.9 to 83.9 years. Their study reported a higher median age compared to our study because they included all subjects even cared in the ICU, who needed RRT or not. The Median age of ACRF (Acute on Chronic Renal Failure) in their study was 80.5 years. But in our study, we excluded critically ill patients so that terminally ill subjects who were very old could have been excluded resulting in a lower mean age.

Mahesh E et al. (2017) [13]in their cross sectional study in a south Indian tertiary care teaching hospital on elderly population reported a mean age of 70.5 years (Vs 70.46 years in our study) in their subjects which were similar to our study.

Li QL et al (2013) [59] in their study reported a mean age of 86.7 with a standard deviation of 5.3 years on 232 elderly AKI subjects which was higher than our study while **Kohli HS et al (2007)**[9] in their study on 33,301 hospitalized adults reported the Mean age of their subjects as 43.9 +/- 16.9 yrs with a range of 18 to 86 years.

Their study was in the total population i.e. adults, but our study included only subjects 65 and above years.

In our study, the youngest person was 65 years old and the oldest person was 93-years-old and the majority (43%) of the subjects in our study belonged to 66-70 year age group, followed by 65 year old age group (22%).

In our study, Males constituted 66% while Females constituted 34% of the study population which was comparable with other studies. Similar to our study, **Mahesh E et al. (2017)**[13]in their study, observed that 59% of the study population were males while 41% were females. But **Li QL et al(2013)** [59] in their study on 232 elderly subjects found that 93% were males and only 7% were females. It is due to the factors related to admission in Chinese PLA General Hospital that a large difference was seen in the proportion of males relative to the females in their study.

Similar to our study **Turgutalp K et al (2017)** [61], also observed that the incidence of AKI-DO (Acute Kidney Injury Developing outside the hospital) was high especially in the male gender.

Risk factors/ Co-morbidities:

Among our study population, 34% of subjects had Diabetes Mellitus and 36.50% had Hypertension while 11% of the subjects had IHD. But only 2% subjects had COPD, RVD, DCM. Anaemia was seen in only 1% of the subjects 0.50% of subjects had LVD, ALD, IWMI while 1.50% subjects had CVA.

In our study, Diabetes and Hypertension were the major co-morbidities found in the study population with AKI. Similar to our study **Mahesh E et al. (2017)** [13] also observed that diabetes was present in 44% of subjects and35% had hypertension.

But they observed that ischemic heart disease was observed in 19% Vs 11% in our study, and 12% had chronic obstructive pulmonary disease vs only 2% in our study.

Similar to our study**, Kohli HS et al (2007)**[9] also observed that Elderly Subjects with ARF had a significantly higher proportion of pre-existing diseases like DM, HTN, Malignancy, Diseases of the respiratory system, Cardiovascular system, Central Nervous system, but they compared it with the younger generation.

In their study along with other factors (such as Age > 60 yrs, sepsis, hypoperfusion) associated comorbidities like diseases of the Respiratory system, CVS, CNS, DM and oliguria, bleeding, critical illness and infection during ARF were predictors of poor outcome in AKI.

Ali T et al. (2007) [45] in their study found a lower proportion of Diabetes (15.7 % Vs 34%) and Hypertension (27.8% Vs 36.5%) in AKI subjects in comparison to our study. IHD was the most common comorbid condition found in 31.5% of subjects. The difference seen from our study is due to the study population, as they included all adults with AKI above 15 years of age.

Presenting complaints:

In our study population, the chief complaint was pain abdomen. It was the most common presenting complaint seen in 57% of the subjects. The proportion of subjects presenting with anuria, oliguria, and fever was 56.50%, 43%, 41.50 % respectively. The other common symptoms were loose stools with vomiting, burning Micturition, breathlessness, cough. The Findings from other studies on chief complaints of the study population was very limited as it was not very essential as

AKI is a laboratory diagnosis and has no specific signs or symptoms. But, these signs and symptoms may lead to the aetiology.

In a Nationwide survey of clinical characteristics of AKI by **Pan H C et al**. (2016) [81]only 33% of subjects presented with Pain Vs 57% in our study. The Chief complaint in their study was Urinary symptoms. About 36% of AKI subjects in their study presented with Dehydration while only 50% presented with Urinary symptoms.

Those symptoms pointing to poor intake orally, losses of salt and fluid through diarrhoea or vomiting may put forward to a pre-renal aetiology[17] while chief complaints of decreased urine output or other urinary symptoms including anuria may point towards postrenal AKI. But urinary tract obstruction can't be ruled out only by the absence of these symptoms. A diagnosis of Postrenal AKI can also be sorted to by the presence of symptoms such as increased frequency of urination, hesitancy, or incontinence. Pain in the flanks may suggest Nephrolithiasis.

Radiological findings:

A Radiographic workup for the detection of any obstruction leading to AKI starts with USG. But, it may appear normal in subjects with early obstruction or under the presence of retroperitoneal processes which encase the kidneys and ureters, hence stopping ureteral dilation. CT can also be very valuable in defining the level and cause of obstruction in case of failure to detect lesion by Ultrasound.

In our study, among 140 (70%) subjects, ultrasonography showed normal kidney size and no urinary obstruction. The common abnormalities identified in ultrasound were Pyelonephritis in 14%, Renal Calculi in 8.5% and Benign Prostatic Hypertrophy (BPH) in 1.50% of subjects respectively.

The Renal ultrasound is useful for investigating underlying CKD[17] if any besides the AKI. In long-standing diseases of the kidney, they are seen as small, echogenic masses or their architecture can be markedly distorted as in cystic diseases. Urinary tract obstruction can be identified by Hydronephrosis or hydroureter.When there is a suspicion of vascular obstruction, doppler of the renal vasculature will be useful.

Information on radiological work up for AKI is very limited in various studies across the globe as the workup is mainly based on parameters in the blood such as serum creatinine and urea.

Final diagnosis/ aetiology of AKI:

The most common diagnosis responsible for AKI in our study population was acute gastroenteritis in 28.50% of the study population, followed by pyelonephritis in 16% of the population and urosepsis in 12% of the population.

But in the study by Mahesh E et al. (2017) [13], Pneumonia and urosepsis were reported as the most common medical causes of sepsis leading to AKI. They observed that medical factors were the cause in 87% while surgical causes accounted for 11%, and gynecological causes in 2%. Among medical causes, sepsis was most common. In comparison to surgical causes, AKI of Medical aetiology had a better outcome. Similarly, Kohli HS et al (2007) [9] in their study also reported that the most common cause of ARF was sepsis (63.26%).

Li QL et al(2013) [59] in their study found out that Infection (43.1%), Hypovolemia (19.0%), use of nephrotoxic drugs (16.8%) and cardiovascular events (15.1%) were major causes of AKI while Ali T et al. (2007) [45]in their study reported

that Sepsis was the most common precipitating factor (47%) which was followed by hypovolemia (32%).

The other common diagnoses in our study were Obstructive Uropathy in 8.5%, Pneumonia in 6.5%, NSAID induces nephropathy in 5.05% and lower limb cellulitis in 4% of the subjects which were similar to other studies[13, 59].

HPE findings: (Histo Pathological)

The Data with regards to renal biopsy is very limited in the elderly. In our study, among the 29 subjects who underwent renal biopsy, Acute Tubular Necrosis was seen 22 (75.86%), AIN (Acute Interstitial Nephritis) was seen in 3 (10.34%), Acute Tubular Interstitial Nephritis was seen in 2 (6.89%) and HIVAN (HIV associated nephropathy) and RPGN (Rapidly progressive glomerulonephritis) were seen in 1(3.44%) subjects each.

Acute tubular necrosis is the most frequent cause of renal disease or intrinsic acute kidney injury[40]. In the absence of a kidney biopsy, it is often only a presumptive diagnosis.

However, Haas, M., et al. (2000)[82] in their study on Acute Renal Insufficiency, observed that the most frequent primary diagnoses on the biopsy specimens were pauciimmune crescentic glomerulonephritis with/without arteritis, observed in about 31.2% of biopsy specimens while Acute tubular necrosis (ATN) with nephrotic syndrome was seen in only 7.5% of subjects as opposed to 755 of our subjects.

The indication for renal biopsy in a case of AKI[83]is when there is no apparent cause with two standard sized kidneys which is non-obstructed. Renal biopsy is a

relatively safe procedure and in more than 95% of subjects, tissue diagnosis is obtained. The life-threatening complications with Renal Biopsy are very less (< 0.1%).

Despite the presence of many effective approaches to prevent and manage AKI in experimental animals, their role in humans is still unexplored.

Hence as of now, at the present, prevention of AKI in elderly patients is aimed at recognizing the increased vulnerability of subjects to AKI such as the use of nephrotoxic agents, volume depletion due to infectious and noninfectious conditions because the treatment techniques are usually supportive.

CONCLUSION

1. The mean age **was** 70.46 years with a standard deviation of 6.02. The youngest person was 65 years old and the oldest person was 93-years-old. There was a male preponderance with a male to female ratio of about 3: 1
2. The most common co-morbidities present in the study population were Hypertension (36.50%), Diabetes mellitus (34%) and Ischemic Heart Disease (11%)..
3. The other less common co-morbidities include COPD, RVD, LVF etc.
4. Pain Abdomen was the most common presenting complaint seen in 57% of subjects. The proportion of subjects presenting with Anuria, Oliguria, and fever was 56.50%, 43%, 41.50 % respectively.
5. The common abnormalities identified in ultrasound were Pyelonephritis in 14%, Renal Calculi in 8.5% and Benign Prostatic Hypertrophy (BPH) in 1.50% of subjects respectively.
6. The most common diagnosis in the study population was Acute Gastroenteritis in 28.50% of the study population, followed by Pyelonephritis in 16% of the population and Urosepsis in 12% of the population. The other common diagnoses were Obstructive Uropathy in 8.5%, Pneumonia in 6.5%, NSAID induced nephropathy in 5.05% and lower limb cellulitis in 4% of the subjects. Various other miscellaneous conditions contributed to the rest of the cases.
7. In histopathology of 29 specimens, ATN (Acute Tubular Necrosis) was the most common abnormality seen in 75.86% of specimens. The other common histopathological findings were AIN (Acute Interstitial Nephritis) in 10.34%, ATIN (Acute Tubular Interstitial Nephritis) was seen in 6.89% and HIVAN (HIV associated nephropathy) and RPGN (Rapidly progressive glomerulonephritis)

SUMMARY

Acute Kidney Injury is a term used to represent a heterogeneous group of conditions which share common diagnostic features such as an increase of serum creatinine (Sr-Cr), Blood-Urea Nitrogen (BUN), often associated with a reduction in urine volume. Various definitions exist for defining AKI, such as proposed by ICD, RIFLE criteria, AKIN criteria with the recent one being KDIGO criteria. The aging kidneys are at an increased risk of AKI since they undergo several structural and functional changes. In the elderly, the incidence of AKI is very high. The elderly AKI patients with their associated co-morbidities usually pose a different set of challenges to the treating physician both diagnostically and therapeutically.

We conducted our observational cross sectional study in the department of General medicine KLE, Belagavi after obtaining the approval of our Institutional Ethics Committee.

In our observational cross sectional study, we studied the spectrum of Acute Kidney Injury in Elderly along with the risk factors leading to AKI. 200 elderly subjects of 65 years of age and above, who were diagnosed with AKI were studied between 2015 to 2017 and critically ill patients were excluded. Relevant medical records were reviewed to confirm the presence of AKI. All the subjects admitted in our study underwent thorough clinical evaluation by clinical history, physical examination as per the institutional protocol after obtaining written and informed consent from the patients. AKI was defined as per the criteria (Criteria used in our study?). All the relevant parameters were documented onto a structured proforma.

The mean age of subjects in our study was 70.46 years with a standard deviation of 6.02. The Mean Serum Creatinine was 4.72 mg/dl with a standard

deviation of 2.91. Hypertension was the most common co-morbidity found in the study population with AKI (36.50%) followed by Diabetes mellitus (34%) and Ischemic Heart Disease (IHD). Majority of the subjects (57%) presented with Pain Abdomen and Anuria (56.50%). The proportion of subjects presenting with Oliguria and fever were 43%, 41.50 % respectively. The most common diagnosis responsible for AKI in our study population was Acute Gastroenteritis in 28.50% of the study population, followed by Pyelonephritis in 16% of the population and Urosepsis in 12% of the population. By imaging with USG, the most common abnormality identified was Pyelonephritis in 14% of study subjects. Renal Calculi was found in 8.5% while Benign Prostatic Hypertrophy (BPH) was observed in 1.50% of subjects. In histopathology of 29 specimens, Acute Tubular Necrosis was the most common abnormality seen in 75.86% of specimens.

AKI is associated with an amplified risk of mortality and morbidity in the elderly. AKI is often detected more slowly in the elderly or it is even camouflaged so that the risk of future complications and poor outcomes are increased. The management of AKI is tailored according to the possible mechanism of injury. So, when the initial attempts fail to limit the injury, the aetiology should be reconsidered immediately. An early renal biopsy for AKI is indicated if the aetiology remains unclear.

An Early Renal consultation for AKI is recommended for the elderly as the treatment is mainly supportive.

The prevention of AKI in elderly involves recognizing their increased vulnerability, because of the structural and functional changes that occur with aging. A multitude of factors may contribute to AKI and many studies also reveal that the elderly subjects suffer from higher morbidity and mortality from AKI. So, the

healthcare workers must be conscious of AKI and also its devastating effects on the elderly. In the future, more resources should be devoted to reducing the incidence of AKI in this population.

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ANNEXURE-I

PATIENT INFORMATION SHEET AND INFORMED WRITTEN CONSENT

FROM

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mr/Mrs _____ we are requesting you to enroll yourself in study titled **"Clinical and Laboratory Evaluation of Acute Kidney Injury (AKI) in Elderly Population: A One Year Cross-Sectional Study at KLES Dr. Prabhakar Kore Hospital"** conducted by Dr. _____, Post Graduate in M.D. General Medicine under the guidance of Dr._____ , Professor, Department General Medicine, J.N. Medical College, KLE university, Belgaum.

Respected sir/madam we request you to participate in our study as you are eligible for participating in the study.

Your participation in research is voluntary. Your decision whether or not to participate in the study will not affect your relationship with J.N. Medical College. If you decide to participate you are free to withdraw at any time.

The purpose of research is to study Acute Kidney Injury (AKI) in elderly population.

Procedure Involved:

If you agree to enrol yourself in my study, you will be interviewed and asked questions regarding your present, past and family history, then you will be clinically examined in detail and investigated accordingly.

Voluntary Participation/Withdrawal:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study. Your decision will not change present or future health care services offered to you at K.L.E's hospital.

Privacy and Confidentiality:

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Authorization to Publish Results:

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study, that can be identified with you will remain confidential.

Financial Incentives for participation:

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator.

CONSENT STATEMENT

I, the undersigned, have been explained in my own vernacular language about the study and my participation in the study is voluntary. If I want I can withdraw at any time. Also I have been given enough time to clear my doubts about the study and my rights as a study participant.

In case you have any questions related to the study you can contact Dr. _____

In case you have any questions about your rights as a study participant you can contact Dr. _____

Signature or the left thumb impression of the participant or legally authorized representative.

Participant's name: _____Signature: _____

Witness name: _____ Signature: _____

Experimenter's name: _____ Signature: _____

Guardian's name: _____ Signature: _____

Place: _____ Date: _____

ANNEXURE II – PROFORMA

Sr No –

IP NO -

Name –

Age –

Sex-

Occupation –

Religion –

Diagnosis –

Chief Complaints-

History of Present Illness (HOPI)-

Past History-

Personal History-

General Examination-

BP=

P=

Temp=

Systemic Examination-

CVS=

CNS=

RESP=

PA=

Investigations-

CBC(Complete Blood Count) =

Hb –

Platelet-

TC-

Creat -

BUN-

USG-

Renal Biopsy –

ANNEXURE III – KEY TO MASTER CHART

Diagnosis Key

Obstructive Uropathy – 1

Pyelonephritis -2 -

Acute Gastroenteritis – 3

Pneumonia – 4

Urosepsis – 5

Viral Fever – 6

Chronic Liver Disease – 7

Dengur fever – 8

Pancreatitis – 9

Carcinoma colon – 10

COPD – 11

Acute Bronchitis – 12

Benign Prostatic Hypertrophy – 13

Burns -14

Snake Bite – 15

Post Coronary Angiography – 16

Lower Limb Cellulitis – 17

Bulbus Pemphigoid – 18

NSAIDs induced – 19

Parotid abcess – 20

Diabetic Ketoacidosis – 21

Pancreatitis -22

Peritonitis due to perforation – 23

Multiple myeloma – 24

Carcinoma cervix – 25

Ayurvedic medicine induced – 26

Gluteal abcess – 27

Axillary abcess – 28

RCINEX tablet induced - 29

Key to Chief Complaints

Pain Abdomen – 1

Fever – 2

Burning Micturition – 3

Increased Frequency of Micturition – 4

Breathlessness – 5

Cough – 6

Oliguria -7

Anuria – 8

Symptoms related to site of abcess – 9

Loose stools/Vomitting – 10

Weight Loss – 11

Burns – 12

Key to Ultrasonography (USG)

0-Normal Kidney Size,no urinary obstruction

1-Pyelonephritis

2-Renal Calculi present

3-Benign Prostatic Hypertrophy (BPH)

4-Chronic Liver Disease

5-Small bowel Peforation

6-Chronic Calcific Pancreatitis

7-Emphysematous pyelonephritis

8-Acute Pancreatitis

9-Acute Hepatitis

10- Carcinoma cervix with metastasis to ureters

11-Periampullary carcinoma with biliary cirrhosis

12-Perforated Appendix

13-Carcinoma Colon Obstructing on ureters

14-B/L moderate HN,with entire HU with B/L Multicystic kidneys.

Key to Renal Biopsy

Acute Tubular Necrosis – 1

Acute Tubulo Interstitial Nephritis – 2

HIVAN(HIV associated nephropathy) – 3

RPGN(Rapidly progressive glomerulonephritis) – 4

AIN(Acute Interstitial Nephritis) - 5

Reference Ranges

Reference Ranges –

Sr Creatinine – 0.50-0.90 mg/dl

Urea -17-49 mg/dl

Hb – 12.0-15.0 g/dL

TLC-4000-10000 /mm3

Platelet-1,50,000-4,50,000/mm3

Chapter 1

Introduction

Chapter 2

Objectives

Chapter 3

Review of Literature

Chapter 4

Methodology

Chapter 5

Results

Chapter 6

Discussion

Chapter 7

Conclusion

Chapter 8

Summary

Chapter 9

Bibliography

Chapter 10

Annexures

					C								
S NO	IP NO	AGE	SEX	DIAGNOSIS/ETIOLOGIES	Chief Complaint	SR CREATININE mg/dL	HB g %	TLC/mm3	PLATELET/mm3	BUN(Urea)mg/dL	USG (Ultrasonography)	RENAL BIOPSY	RISK FACTORS
1	729287	65	F	1	1,7	6.6	12.2	13500	196000	97	2		
2	733064	67	M	2	1,2,7	7.65	10.2	9200	188000	240	1		DM,HTN
3	748219	66	M	1	1,2,7	12.94	9.4	14300	300000	80	2		HTN
4	712867	65	F	6	2,7	2.43	13.3	6200	147000	140	0		HTN, DM
5	740641	68	F	3	1,8,10	5.7	15.9	7700	288000	78	0		
6	777807	69	M	5	2,3,7	1.85	10.1	9100	284000	66	0		DM
7	730376	79	F	3	1,8,10	2.8	9.9	13400	256000	72	0		IHD
8	778316	77	M	4	2,5,6,8	2.9	9.6	13000	121000	50	0		DM,HTN
9	730756	77	F	5	2,3,7	3.83	9.4	7800	226000	109	0		DM,HTN
10	717033	65	F	2	1,2,7	1.8	9.4	13800	511000	34	1		DM,HTN,IHD
11	726250	66	M	3	1,8,10	11.9	9.8	17000	223000	336	0	2	
12	736451	85	M	2	1,2,7	2.6	8.6	9100	336000	39	1		DM,HTN
13	756681	85	M	12	5,6,7	2.41	13.5	12500	266000	70	0		IHD
14	772819	66	M	3	1,8,10	3.6	6.9	9600	170000	93	0		
15	724923	68	M	5	2,3,7	7.39	9.5	12000	220000	162	0		RVD
16	733064	67	M	2	1,2,7	3.96	10.9	27000	196000	138	1		DM,HTN
17	721957	76	M	7	1,8	9	11.3	21500	131000	207	4		DM,HTN
18	736182	74	M	3	1,8,10	3.42	14.1	32200	99000	74	0		
19	757442	70	M	3	1,8,10	6.21	10.4	10000	85000	152	0	1	DM
20	728235	66	M	3	1,8,10	3.3	8.9	16200	140000	110	0	1	
21	711284	85	M	4	2,5,6,8	5.02	11.9	23500	120000	140	0		DM,HTN
22	722587	67	M	2	1,2,7	3.34	10.7	9200	200000	121	1		DM
23	760377	65	M	5	2,3,7	2.87	13.4	18300	184000	72	0		
24	771017	72	M	2	1,2,7	2.7	10.7	26500	280000	79	1		
25	712778	80	M	1	1,7	9.5	11.4	13800	195000	223	14		
26	737331	69	M	19	8	8.9	6.9	19,500	480000	344	0		
27	750718	85	M	19	8	2.4	16.8	10,900	127000	93	0		
28	729140	65	M	17	2,8,9	2.2	13.3	10600	61000	66	0		
29	762857	65	M	2	1,2,7	2.77	11	8300	173000	95	1		
30	742727	65	M	3	1,8,10	7.74	10.5	12000	223000	270	0	1	
31	752874	65	M	8	2,8	4.28	14.4	3800	42000	217	0		
32	732575	66	M	11	5,6,7	3.1	16	4800	120000	133	0		
33	763794	73	M	3	1,8,10	3.54	12.5	11900	326000	119	0		DM,HTN
34	769530	85	F	1	1,7	5.39	11	7700	410000	260	2		DM,HTN
35	734401	65	F	3	1,8,10	3.4	15.1	12,600	269000	64	0		DM,Hypothyroidism
36	714460	66	F	10	7,11	2.85	12.3	15600	168000	86	13		
37	718069	66	F	2	1,2,7	4.23	10.9	20500	287000	101	1		
38	748381	70	M	13	7	2.07	11.1	10600	171000	74	3		HTN,IHD
39	720781	68	M	11	5,6,7	3.24	10.1	9400	261000	90.3	0		DM,IHD
40	736306	72	F	3	1,8,10	2.8	11.8	6000	210000	60	0	1	
41	708606	65	M	14	8,12	2.09	7.4	8200	89000	48	0		
42	710509	70	M	3	1,8,10	5.91	8	7,000	199000	161	0		
43	725781	68	M	3	1,8,10	1.9	14.1	13800	358000	43	0		
44	729173	66	M	4	2,5,68	3.3	11.9	29200	481000	72	0		
45	726259	67	M	3	1,8,10	6.5	14.8	9100	202000	194	0		IHD
46	712122	80	M	19	8	3.39	15.4	16000	236000	104	0	1	HTN

S NO	IP NO	AGE	SEX	DIAGNOSIS/ETIOLOGIES	Chief Complaint	SR CREATININE mg/dL	HB g %	TLC/mm3	PLATELET/mm3	BUN(Urea)mg/dL	USG (Ultrasonography)	RENAL BIOPSY	RISK FACTORS
47	713701	65	M	4	2,5,6,8	2.2	10.9	9200	320000	93	0		HTN/DM, DCM, LVD
48	765334	70	M	27	8,9	4.17	9	8000	239000	43	0		DM,HTN
49	755236	68	M	19	8	1.35	14.7	10100	345000	20	0		HTN
50	713583	79	M	17	8,9	2.5	13.6	10300	18900	110	0		DM,HTN
51	747461	66	F	3	1,8,10	5.3	12.4	7700	160000	65	0		
52	716840	68	F	1	1,7	4.6	11.2	6300	210000	140	2		HTN
53	760934	80	F	3	1,8,10	1.9	7.9	8100	207000	108	0		HTN
54	758328	65	F	3	1,8,10	6.08	11.4	11400	350000	92	0	1	DM
55	788764	70	F	2	1,2,7	4.1	12.8	18200	200000	106	1		DM,HTN
56	756353	66	F	3	1,8,10	9.53	10.8	9100	302000	132	0		
57	741291	67	F	3	1,8,10	3.34	8.7	7500	278000	108	0		
58	745072	70	F	3	1,8,10	4.06	11.5	20000	280000	86	0		
59	737833	65	F	8	2,7	4.91	10.3	6000	82000	67	0		HTN
60	778596	66	F	2	1,2,7	2.58	10	13400	90000	82	1		DM,HTN
61	738109	65	F	3	1,8,10	6.17	13	11800	240000	152	0		DM
62	754582	70	M	2	1,2,7	2.1	9.3	9700	310000		1		
63	718972	83	M	5	2,3,7	4.58	9.1	18500	162000	61	0		DM,HTN
64	718558	65	M	16	5,8	3.2	11.1	12000	250000	98	0		IHD
65	735079	73	F	3	1,8,10	4.4	8.7	13200	170000	128	0		DM,HTN
66	777308	67	M	19	8	15.4	9.6	11500	320000	340	0		HTN
67	752151	65	M	2	1,2,7	3.4	10.3	6400	550000	110	0	5	
68	735616	66	M	23	1,2,8	1.8	11.9	22100	460000	32	12		
69	751198	67	M	7	1,8	2.86	11.4	23500	92000	111		1	HTN
70	726170	65	M	3	1,8,10	4.29	8.1	24900	720000	86	0		ALD, Pulmonary HTN
71	756197	72	M	1	1,7	2.98	9.8	4800	240000	77	2		
72	744686	65	M	15	8,9	9.54	10.7	15000	92000	280	0	2	
73	748414	66	M	3	1,8,10	2.41	12	13000	250000	80	0	1	DM
74	734403	70	M	5	2,3,7	3.4	11.3	9900	192000	124			DCM
75	749067	80	F	3	1,8,10	2.69	10.3	10300	246000	66	0		COPD/Cor Pulmonale
76	720006	65	M	2	1,2,7	6.74	10.2	12200	283000	124	1		
77	761109	66	M	3	1,8,10	2.1	10.8	14500	298000	65	0		DM,HTN,IHD
78	766451	67	F	25	8,11	14.32	8.8	11200	390000	140	10		
79	778751	75	M	4	2,5,6,8	2.2	13.5	25700	185000	32	0		
80	777203	65	M	5	2,3,7	3.11	9.2	29800	130000	121	0		DM,HTN
81	775996	66	F	3	1,8,10	7.3	8.7	8500	345000	158	0		
82	721146	65	M	2	1,2,7	1.8	10.2	7100	191000	48	1		
83	756582	66	F	2	1,2,7	7.4	10	19200	539000	120	1		HTN
84	779036	65	M	3	1,8,10	4.84	14.3	12600	155000	108	0		
85	733417	67	M	5	2,3,7	20.45	10.6	9700	325000	206	0	3	RVD
86	748438	65	F	21	5,8	2.48	11.7	7300	217000	146	0		DM
87	720810	65	M	4	2,5,6,8	2.31	13.7	34300	227000	76	0		
88	726096	90	M	17	2,8,9	2.73	9.9	25100	95000	102	0	1	DM,HTN,IHD
89	742602	78	M	2	1,2,7	7.52	14	12500	348000	148	0	1	
90	776086	71	M	1	1,7	2.8	8.8	7200	180000	92	2		
91	770043	66	F	2	1,2,7	3.45	10.8	20500	268000	107	1		HTN
92	731973	80	F	4	2,5,6,8	3.21	10.8	19300	559000	57	0		
93	771737	87	F	4	2,5,6,8	2.7	10.2	13400	269000	75	0		HTN
94	733035	65	M	20	8,9	6.59	10.9	12500	563000	180	0		DM,HTN

S NO	IP NO	AGE	SEX	DIAGNOSIS/ETIOLOGIES	Chief Complaint	SR CREATININE mg/dL	HB g %	TLC/mm3	PLATELET/mm3	BUN(Urea)mg/dL	USG (Ultrasonography)	RENAL BIOPSY	RISK FACTORS
95	768038	80	M	5	2,3,7	6.3	12.3	12700	129000	146	0		HTN
96	722900	74	F	12	5,6,7	1.9	11.7	6900	245000	79	0		Old CVA,Gouty Arthritis
97	771800	70	F	11	5,6,7	8.07	13	15400	106000	183	0		HTN
98	760500	74	M	11	5,6,7	6.5	8.3	8900	385000	126	2		DM,HTN
99	747767	70	M	5	2,3,7	5.9	11.7	16600	306000	130	0		DM,IHD
100	742888	66	M	3	1,8,10	2.2	9.3	14200	204000	88	0		DM,HTN,IHD
101	779441	75	M	7	1,8	2.1	12	11000	160000	72	9		
102	740546	65	M	3	1,8,10	8.3	10	7100	199000	136	0		DM/HTN
103	772871	67	F	19	8	5.74	9.1	8500	378000	150	0		
104	717840	67	M	4	2,5,6,8	2.03	17.3	9900	143000	40	0		
105	712386	73	M	2	1,2,7	2.2	10	2900	207000	41	1		
106	728243	93	M	13	7	2.4	11.4	3200	260000	60	3		
107	736019	79	M	3	1,8,10	2.33	10.7	7900	241000	53	0	1	DM,HTN
108	763854	69	M	3	1,8,10	8.42	16.9	13700	347000	168	0	1	
109	778274	75	M	5	2,3,7	3.9	10	12000	213000	105	0		DM
110	751920	66	M	19	8	5.16	9.4	9300	52000	157	0		
111	725396	65	M	9	1,8	4.44	14.4	14700	118000	132	8	1	
112	744757	65	M	2	1,2,7	8.52	9.1	17200	379000	165	1		
113	736740	72	F	3	1,8,10	6.86	11	18500	301000	116	0	1	DM
114	712484	72	M	2	1,2,7	7.55	14.8	23000	674000	307	1		DM,HTN
115	761656	65	M	8	2,8	3.74	11.1	11900	101000	94	0		
116	766143	72	M	5	2,3,7	3.13	9.2	12000	235000	112	0		DM,HTN
117	751519	65	M	3	1,8,10	3.97	10.2	13000	140000	168	0	1	
118	536671	68	M	5	2,3,7	4.2	11	14800	280000	112	0		HTN
119	771940	66	M	3	1,8,10	2.4	11.6	14100	233000	85	0	4	
120	755571	77	F	2	1,2,7	2.1	13.8	7700	120000	70	1		
121	736994	65	F	3	1,8,10	8.13	11.1	18700	395000	118	0	1	
122	734366	66	M	5	2,3,7	9.79	10.6	11500	64000	172	0		
123	751726	81	M	17	2,8,9	4.4	9.3	19000	213000	161	0		HTN
124	739302	66	M	3	1,8,10	3.9	11.6	18200	212000	87	0		DM,HTN.IHD
125	714324	70	M	5	2,3,7	6.4	11.3	9900	260000	155	0		
126	733836	78	F	3	1,8,10	8	11.3	15000	320000	116	0		DM
127	730765	72	M	3	1,8,10	1.9	15	11800	153000	88	0		
128	752215	69	F	4	2,5,6,8	2.4	13.2	14600	175000	70	0		DM,HTN,Left PCA tertiary Infarct
129	770056	70	F	26	8	2.57	10.4	15500	528000	58	0		
130	761931	66	M	2	1,2,7	2.78	10.6	26100	57000	93	1		HTN
131	744591	78	M	3	1,8,10	5.4	11.4	9400	163000	260	0		
132	762458	69	F	3	1,8,10	2	14	13700	260000	31	0		DM,HTN,IHD
133	771340	69	M	2	1,2,7	2.1	12.4	10400	179000	79	1		
134	670758	77	F	5	2,3,7	3.89	10.6	20000	140000	126	0		DM,HTN
135	765932	74	F	19	8	6.06	9.9	12800	250000	109	0		DM
136	773204	66	F	5	2,3,7	5.86	9.7	22100	120000	220	0		
137	775635	65	M	3	1,8,10	3.3	13.8	14800	270000	130	0		DM,HTN,IHD
138	761332	73	M	19	8	13.18	10.8	10000	235000	193	0		DM,HTN
139	731721	65	M	29	8	6.26	12.5	8600	138000	238	0		
140	744976	82	M	2	1,2,7	9.66	11.6	38300	80000	164	1		
141	779083	75	M	17	2,8,9	1.89	11.1	11200	334000	98	0		DM,HTN
142	756426	70	F	15	8,9	7.47	9.6	5200	79000	290	0	1	

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143	733079	70	F	1	1,7	4.46	11.4	13900	89000	188	2		HTN
144	765988	65	F	3	1,8,10	2.5	9.8	8600	173000	84	0	1	
145	744822	75	F	3	1,8,10	5	11.3	17,600	200000	95	0		HTN
146	708512	75	M	3	1,8,10	5.64	13.2		167000	126	0		IHD,IWMI
147	760859	67	M	2	1,2,7	5.61	13.7	25600	215000	210	1		
148	753746	65	F	2	1,2,7	2.2	11.6	6400	191000	62	7		
149	772419	67	F	5	2,3,7	2.14	11.6	16200	244000	56	0		DM,IHD
150	721203	66	M	3	1,8,10	4.98	12.7	10400	130000	113	0		
151	712751	68	F	1	1,7	7.99	9.5	10400	573000	99	2		HTN
152	763815	72	F	19	8	2.25	10.4	9500	272000	66		1	DM,HTN
153	737316	65	M	4	2,5,6,8	2.83	10.3	11800	193000	62	0		DM,HTN,IHD
154	711129	65	F	1	1,7	10.8	9.5	15200	485000	104	2		DM
155	735385	76	F	5	2,3,7	7	10.9	10100	114000	249	0		
156	723750	78	F	1	1,7	2.28	11.8	7800	330000	64	2		
157	764055	68	M	3	1,8,10	3.7	13	8200	240000	116	0		DM
158	769275	68	F	5	2,3,7	2.4	10.3	16100	124000	100	0		DM,HTN
159	771768	65	M	9	1,8	3.5	12	14500	129000	166	6		
160	760925	70	F	5	2,3,7	1.9	8.8	7400	211000	50	0		DM,HTN,IHD
161	763093	68	M	18	2,8,9	4.18	12.6	23200	437000	144	0	1	
162	753411	69	M	1	1,7	6.64	11.1	15800	360000	290	2		
163	772874	70	M	1	1,7	3.82	12.3	21200	120000	168	2		
164	725505	70	M	12	5,6,7	3.4	8.4	10000	220000	136	0		IHD
165	716382	72	M	4	2,5,6,8	3.1	12.2	11,600	150000	104	0		old CVA-SDH with right parietal infarct
166	776383	65	M	1	1,7	2.49	10.9	11800	339000	123	2	1	DM
167	729045	73	M	3	1,8,10	12.3	10	20,500	134000	217	0	5	COPD
168	737157	84	M	23	1,2,8	2.6	14.4	20000	187000	78	5		HTN
169	724195	73	M	3	1,8,10	4.28	10.4	28,500	380000	110	0		DM,HTN
170	773097	77	M	5	2,3,7	4.1	10.1	15600	135000	158	0		HTN
171	713389	83	M	13	7	2	12	14,200	162000	50	3		DM,HTN,LVH
172	719397	75	F	4	2,5,6,8	6.5	13.4	12800	275000	113	0	1	DM
173	713743	70	M	17	2,8,9	2.1	14.3	25,100	460000	97	0		HTN
174	723082	75	M	1	1,7	4.5	11.1	6800	460000	67	2		HTN,IHD
175	747624	65	M	1	1,7	3.94	9.6	25,200	73000	85	2		DM,HTN
176	715572	71	F	2	1,2,7	2.2	8.7	23400	180000	86	1		
177	725379	68	M	3	1,8,10	8.25	12.1	14,200	444000	146	0		DM,HTN
178	75472	80	F	24	8,11	9.76	7	12800	292000	192	0		HTN,COPD
179	748433	70	M	2	1,2,7	3.79	11	15,400	147000	112	1		HTN,BPH
180	756114	66	M	28	8,9	4.01	11.9	5300	61000	139	0		HTN
181	775641	75	F	25	8,11	6.36	9.8	8,200	251000	54	0		Anemia
182	727275	65	F	5	2,3,7	4.73	8.7	11600	165000	128	0		Haemorrhoids/Anemia
183	765334	70	F	27	8,9	4.17	9	16,200	280000	142	0		DM,HTN
184	745177	84	F	2	1,2,7	6.33	9.8	18900	326000	145	1		DM
185	742021	65	F	3	1,8,10	8.67	11.6	24300	167000	113	0		
186	740574	66	M	3	1,8,10	3.05	12.4	16600	160000	71	0		
187	748365	68	M	3	1,8,10	7.3	16.2	20700	400000	103	0		DM,HTN,IHD
188	727540	65	M	17	2,8,9	3.13	9.8		61000	146	0		
189	761430	66	M	1	1,7	4.2	10.1	9200	200000	130	2		
190	723283	67	M	3	1,8,10	4.11	14.8	10100	314000	122	0		Old CVA,HTN

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191	739930	85	M	5	2,3,7	2	11	9800	420000	46	0		IHD,COPD
192	719383	72	F	17	2,8,9	2.7	12.4	18700	210000	72	0		DM
193	737349	66	M	7	1,7	4.33	7	27800	41000	108			
194	735809	65	F	3	1,8,10	10.59	11.8	15700	228000	281	0		
195	727060	74	M	2	1,2,7	2.9	10.6	12700	478000	58	0	5	
196	720470	67	M	3	1,8,10	5.05	15.4	8300	226000	94	0		DM
197	776138	68	F	2	1,2,7	4.74	8.1	13400	356000	220	1		DM
198	709496	72	M	5	2,3,7	3.28	8.3	6600	164000	138	0		
199	717449	70	M	3	1,8,10	2.8	12.6	10800	324000	82	0		
200	755539	72	M	2	1,2,7	7.4	10.1	4100	125000	200	1		