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A study of acute kidney injury in patients admitted in intensive care unit in a tertiary care centre.

Dr D H Choudhary , Dr D O Patil , Dr C A Kante
Junior Resident, Department of Medicine, Dr Ulhas Patil Medical College And Hospital,Jalgaon , Maharashtra
Professor, Department of Medicine, Dr Ulhas Patil Medical College And Hospital, Jalgaon,Maharashtra
Professor, Department of Medicine, Dr Ulhas Patil Medical College And Hospital, Jalgaon ,Maharashtra

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I. INTRODUCTION

Acute kidney injury (AKI) refers to syndrome encompassing kidney damage from mild injury to full loss of functions that seriously disturbs the homeostasis of fluid and electrolyte balances1. An uniform definition for acute kidney injury has existed only since 2004, when the Acute Dialysis Quality Initiative (ADQI) proposed the Risk, Injury, Failure, Loss, End-stage kidney disease (RIFLE) criteria for AKI.2 Since then two modifications of the RIFLE: Acute Kidney Injury Network (AKIN) (2007)3, and Kidney Disease: Improving Global Outcomes (KDIGO) (2012)1 have emerged. All of the three modern definitions are based on changes in serum or plasma creatinine (Cr) and urine output (UO).

As the kidney injury progresses and affects the glomerular filtration rate (GFR) Cr starts to rise. Oliguria or anuria may develop early, but sometimes the UO remains intact for quite long. Later in the course of AKI the severely diminished GFR manifests as electrolyte and acid-base disturbances, most often as elevated potassium and acidosis.

The arising consensus suggests that AKI is a syndrome with several different predisposing factors and mechanisms of pathophysiology. A growing amount of data supports the idea that risk for AKI increases with a growing "burden of illness" whether chronic or acute.1 The traditional division of kidney failure to pre- and post-renal causes has been widely abandoned as the complex nature of the kidney injury syndrome has unfolded.1 Extrarenal causes, without actual kidney damage, such as depletion of fluids or urinary track obstruction naturally still exist but are rare causes for AKI in the intensive care environment. Also these causes, when identified, are quite easy to treat and usually without long-term damage to the kidney or other organs. In the ICU, AKI is usually multifactorial with both chronic conditions and acute events contributing to the development of kidney injury.9 Sepsis is the most common single underlying cause for AKI.

In the diagnosing and staging of AKI, serum creatinine and urine output act as surrogates for glomerular filtration rate, however prominent weaknesses in both as kidney injury markers exist5. A vigorous search for new kidney injury biomarkers has been going on for several years. A hope of easily measurable markers that would be more sensitive and specific to actual injury in the kidneys, would react earlier in the course of AKI, and would be less prone to bias in different physiological situations remains.

AKI has significant consequences. It is associated with morbidity and permanent loss of kidney function. All severity stages of AKI are associated with significantly higher short15 and long-term mortality6. Hence aim of this study was done to evaluate the different etiologies of AKI in critically ill patients AKI

AIMS & OBJECTIVES:

1. To study demographic factors in acute kidney injury in patients.
2. To study aetiologies of acute kidney patients
3. To study clinical features in acute kidney injury
4. To find biochemical changes.

II. MATERIALS AND METHODS

Type of study: It was an observational cross-sectional study.

Sample size: Sample size was calculated to be 100. Approval from Institutional Ethical Committee was taken.

Period of study : Duration from Dec 2022 to Jan 2024.

Sample size : This study was conducted on 100 cases admitted in ICU developing AKI.

The formula used to calculate the sample size-
Sample Size (n) = 4 p q/ l2
Where,

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n = minimum sample size to be calculated
p = prevalence of disease under study which was taken as 20 (Reference study Jose J. et al 7)
q=100-p
l = allowable error
n=4 * 20*80/82
=100

Patient data collection and evaluation:

- Data was collected from all patients, irrespective of their gender/ background /socio economic status. The patients were evaluated and according to protocol.
- 100 cases admitted in ICU developing AKI were studied. A performa was pre designed to gather the minimum but essential information regarding different parameters as depicted in case performa.

INCLUSION CRITERIA

- Patients > 13 years of age getting admitted to ICU with wide variety of medical disorders necessitating ICU care.
- Acute increase in serum creatinine of 0.5mg/dl if baseline serum creatinine is > 1.5 mg/dl or increases at least 1 mg/dl if base line serum creatinine is > 1.5 mg/dl but < 5mg/dl.
- Oliguria urine output < 200 ml in 12 hours.
- Need for dialysis

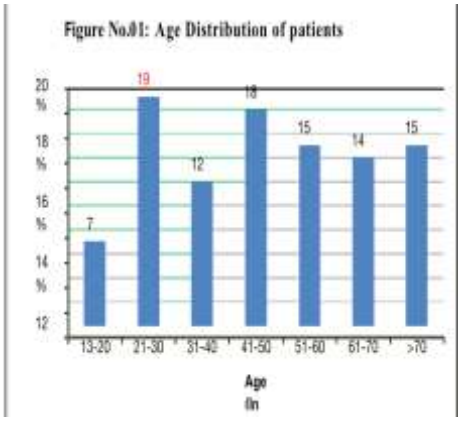
EXCLUSION CRITERIA

- Patients of chronic kidney disease.
- Age < 13 years.
- Patients with retroviral diseases, diabetes mellitus, hypertension and obstructive uropathy.

III. STATISTICAL ANALYSIS:

Data was analysed using statistical software and will be presented in the form of tables, figures, graphs whenever necessary.

IV. OBSERVATIONS AND RESULTS



Majority of AKI patients i.e. 19% were between 21-30 years followed by 18% in 41-50 years and rest 15% each in 51-60 years and >70 years.

Age group (years)	Males	Females	Total
13-20	4	3	7
21-30	9	10	19
31-40	9	3	12
41-50	10	8	18
51-60	12	3	15
61-70	9	5	14
>71	11	4	15
Total	64	36	100

Table No.1: Distribution of patients with respect to Gender

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In present study, 64% were male and 36% participants were females. Most of the males belonged to the age group of 51 to 60 years. Most of the females belonged to the age group of 21 to 30 years.

Table No. 2: Patients with respect to etiology and Age

Etiology	13-20 Years	21-30 Years	31-40 Years	41-50 Years	51-60 Years	61-70 Years	>71 Years	Total
Sepsis	1	8	3	5	10	3	9	39
Vasculotoxic snake bite	1	5	5	7	3	3	1	25
Cardio renal syndrome	0	1	0	2	1	5	2	11
Hepatic causes	2	0	1	1	0	1	0	5
Dehydration	0	0	1	1	0	2	0	4
Obstetric causes	1	3	0	0	0	0	0	4
Malaria	0	1	1	0	1	0	0	3
Poisoning	1	0	1	0	0	0	1	3
Dengue	1	0	0	1	0	0	0	2
AFI	0	0	0	0	0	0	2	2
Others	0	1	0	1	0	0	0	2
Total	7	19	12	18	15	14	15	100

The Table No.02, shows various causes of AKI distributed across the age groups. Most common cause of AKI in the study is sepsis (n=39), followed by vasculotoxic snake bite (n=25) and cardiorenal syndrome (n=11).Hepatic causes were the most common causes in patients < 20 years of age.

Table No.03: Distribution of patients with respect to clinical features (n=100)

	Clinical symptoms	Percentage
	Fever	79
Urinary symptoms	Dysuria	4
	Oliguria	36
Respiratorysymptoms	Cough	4
	Dyspnea and cough	19
	Dyspnea	39
Cardiovascular	Chest pain	3
Gastrointestinal symptoms	Abdominal pain	6
	Vomiting	27
	Loose motions	9
Central Nervous System symptoms	Altered sensorium	25
	Convulsion	5

Fever (79%) was the most common clinical symptom. Next major system involved is respiratory system, with 39% complaining of dyspnoea, 4% complaining of cough and 19% complaining of both. Next major system involved is urinary system.

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Table No. 4: Distribution of patients with respect to biochemical changes

Biochemical parameters		Percentage of cases (%)
Hemoglobin	<10	46.00
	≥10	54.00
Serum creatinine	<1.4	0.00
	≥1.4	100
BUN	<20	0.00
	≥20	100
Serum bilirubin	<1.5	76.00
	≥1.5	24.00
SGOT	<40	82.00
	≥40	18.00
SGPT	<40	82.00
	≥40	18.00
Serum Potassium	Hypokalemia	11.00
	Normal	77.00
	Hyperkalemia	12.00
Serum sodium	Hyponatremia	36.00
	Normal	58.00
	Hypernatremia	06.00

The table No.4, shows distribution of patients with respect to biochemical changes. Hb was low (Hb < 10.00 grams per dL) in 46% of the individuals. Serum creatinine was high in all of the study participants. Blood Urea Nitrogen was also high in all the study participants. 24% of the patients had high serum bilirubin. As for enzymes, SGOT was elevated in 18% of the people and SGPT was elevated in another 18%.

V. DISCUSSION

AKI is a potentially fatal, but reversible renal disease. The etiology, course, outcome differ in various parts of the world and also within India due to its climatic and geographic diversity and the variable standards of medical care. In our study 100 patients were analyzed. There were 64 males and 36 females. Mean age of occurrence was 47.65 years. Maximum number of cases occurred in 3rd and 5th decade. In a study by Kai Singbartl et al 8, age was consistent risk factor. Even in a study by Eswarappa M et al 9 showed that median age of the patients was 55.5 years, lying in the age group of 51- 60 years. This study highlighted the potential risk of AKI in elderly patients especially above the age of 60 years. Sepsis is the most common cause of AKI followed by hypovolemia in the above mentioned studies. However, vasculotoxic snake bite induced AKI is the second most common cause in our study since the incidence of snake bite cases are high in our study area. According to the James Case et al10, patients with sepsis have the highest incidence of AKI. In a study conducted by Kai Singbartl et al,8 sepsis is the major cause of AKI, accounting for nearly 50% of cases. Sepsis in our study was mainly due to infections like pneumonia and urinary tract infections. The excessive systemic inflammatory reaction most likely plays a key role in the development of kidney injury and multiple organ failure. The release of various inflammatory mediators, from pathogens and from immune cells, induces direct toxicity to tubular cells and triggers a complex cascade of inflammation. Hence, sepsis is one of the causes of renal AKI causing deranged renal function test. The most common clinical signs and symptoms of toxicity are nausea, vomiting, abdominal and chest discomfort, profound hypotension, severe metabolic acidosis or mixed metabolic acidosis, respiratory alkalosis, and acute renal failure may occur. Also, some rare complications have been reported such as pulmonary edema, acute pancreatitis, transient leukopenia, and transient hyperglycemia.

VI. CONCLUSION-

The occurrence of acute kidney injury (AKI) is prevalent in patients admitted to the intensive care unit (ICU), leading to a complex hospital course and adverse outcomes. The pathogenesis of AKI in this patient population is multifactorial. The risk factors for the development of AKI in the ICU patients include age and underlying diseases. Consistently, our data demonstrated a significant trend toward an increased number of AKI cases with an increased number of underlying diseases and older age. Laboratory findings have not been previously investigated enough as risk factors of AKI. Although it did not remain significant in the multivariate analysis, the small difference in the levels of BUN and Cr on admission to ICU was highly significant. Hypotension or hypovolemia could have contributed. This fact may have clinical implications since a patient admitted to the ICU with slightly elevated BUN and Cr levels must be considered and treated as a patient at risk to develop AKI. Future studies may benefit by better identifying modifiable risk factors to prevent the development of AKI.

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