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Original Research Article

COMMUNITY ACQUIRED ACUTE KIDNEY INJURY: A PROSPECTIVE OBSERVATIONAL STUDY

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Abstract

Background: Pattern of Acute kidney injury is different from region to region in India. Prospective data on community acquired AKI using KDIGO criteria for AKI are limited. Objective was to study the etiology, clinical, characteristic, short term outcome of CAAKI in adults. **Materials and Methods:** This was prospective observational study in medical ward of tertiary care hospital. patient fulfilling the 2012 KDIGO AKI criteria of community acquired acute kidney injury (CAAKI) were included. Patient who develops AKI 48 hours after admission, those hospitalized >48 hrs elsewhere, patient with chronic kidney disease, Obstetric and surgical cases of AKI were excluded. Serum creatinine and urine output monitored daily. Development of hypotension, oliguria, ARDS, need of renal replacement was noted. **Result:** 150 patients of community acquired AKI were studied between 2021 to 2022. Mean age of the patients was 49.8 years. 26.7% in AKI stage 1, 36.7% in AKI stage 2 &3. Acute gastroenteritis was the commonest cause for AKI, followed by pneumonia and snake bite in our region. 12% required Dialysis & 18% succumbed to the illness. **Conclusion:** CAAKI is a common, serious, often preventable complication of certain condition acquired in community and is therefore a matter of utmost importance for public health.

INTRODUCTION

In 2012 the Kidney Disease Improving Global Outcome (KDIGO) guideline unified the definition of AKI and included stage of severity. AKI has since been defined as increase in serum creatinine of 0.3mg/dl or more within 48 hour of observation or 1.5 times Baseline or greater which is known/ presumed to have occurred within 7 days, or a reduction in urine volume below 0.5/kg/hr for 6 hours.[1-3]

The term acute kidney injury is replaced by acute renal failure. Mean dichotomous relationship between normal kidney function and over organ failure acute kidney attempts to encompass the growing study of data associating small acute and transient decrements in kidney function with serious adverse effects.[3,4]

Kidney is a source of variety of stems cells help in repair after Injury seen in ischemic insult of kidney renal and tubular epithelial cells undergoes regeneration led to recovery.[5] Among the few organs in the body 'Kidney has ability to undergoes virtually complete recovery of structure and function after damage. Despite many advancements in management of Acute Kidney Injury mortality is still a high due to complication. It is also as independent risk factor for death.[6,7] AKI complicates 5.7% of acute care hospital and up to 30% of admission to ICU.[8,9]

In this study we have evaluated the epidemiology, clinical feature, mortality, complication, outcome, prognosis of acute renal failure in community. Most common causes of community acquired acute kidney injury are different in different region so our main objective of this study the aetiology, clinical feature, outcome of community acquire, acute renal failure patient admitted to ICU in RIMS Raichur.

Prerenal AKI: Prerenal azotemia is the most common cause of AKI, accounting for approximately 40% to 50% of all cases.[6,10] Effective hypovolemia causes a decrease in mean arterial pressure that activates baroreceptors and initiates a cascade of neural and humoral responses, leading to activation of the sympathetic nervous system and increased production of catecholamines, especially norepinephrine.[11,12] This response also increases intrarenal angiotensin II (Ang II) activity via

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activation of the renin-angiotensin-aldosterone system (RAAS). Ang II is a potent vasoconstrictor that preferentially increases efferent arteriolar resistance, preserving glomerular filtration rate (GFR) in the setting of decreased renal perfusion by maintaining glomerular hydrostatic pressure.[13-15]

Renal sympathetic nerve activity is significantly increased in prerenal azotemia.[16,17] In hypovolemia, adrenergic activity constricts both the afferent arteriole and changes efferent arteriolar resistance through Ang II. α-Adrenergic activity primarily influences kidney vascular resistance, while renal nerve activity is linked to renin release through β-adrenergic receptors on renin-containing cells.[18]

Contrarily, α2-adrenergic agonists primarily decrease the glomerular ultrafiltration coefficient via Ang II. Even after subacute renal denervation, renal vascular sensitivity to Ang II increases due to major upregulation of Ang II receptors, leading to complex effects on renin-angiotensin activity in prerenal azotemia.[19,20]

Autoregulation, maintained by stretch receptors in afferent arterioles, causes vasodilation in response to reduced perfusion pressure. In physiological conditions, autoregulation works until a mean systemic arterial blood pressure of 75 to 80 mm Hg. Renal production of prostaglandins, kallikrein, kinins, and nitric oxide (NO) increases, contributing to vasodilation.[21]

In patients with advanced liver disease and portal hypertension, hepatorenal syndrome (HRS) represents an extreme form of prerenal disease, characterized by peripheral and splanchnic vasodilation with intense intrarenal vasoconstriction unresponsive to volume resuscitation.[22]

INTRINSIC AKI: Intrinsic AKI can be divided into diseases of larger renal vessels, diseases of renal microcirculation and glomerular, ischemic, nephrotoxic, and tubulointerstitial inflammation. Intrinsic AKI differs from prerenal as hypoperfusion induces ischemic injury to renal parenchymal cells, particularly tubular epithelium, resulting in acute tubular necrosis (ATN). Recovery takes one to two weeks after normalization of renal perfusion, but in extreme cases, it can lead to bilateral renal cortical necrosis and irreversible renal failure.[21,22]

Structural injury in the kidney is the hallmark of intrinsic AKI, with acute tubular injury (ATN), either ischemic or cytotoxic, being the most common form.[23] Frank necrosis is not prominent in most human cases of ATN and tends to be patchy. Other changes include loss of brush borders, flattening of the epithelium, detachment of cells, formation of intratubular casts, and dilatation of the lumen. Injury is predominantly observed in proximal tubules, but injury to the distal nephron can also occur. The distal nephron may also become obstructed by desquamated cells and cellular debris.[24,25]

Vascular factors contributing to ATN include alterations in renal blood flow, increased sensitivity to vasoconstrictors, impaired autoregulation leading to endothelial injury, and various other factors like changes in endothelium, nitric oxide, prostaglandins, and leukocyte adhesion. Apoptotic cell death primarily occurs in the distal nephron during ischemic injury due to alterations in the actin cytoskeleton, leading to flattening of the epithelium.[26]

The investigations and the probable results would be as under,[12,18,27,28]:

1. **BUN/Creatinine Ratio:** Prerenal AKI often elevates BUN disproportionately (BUN: Creatinine ratio > 20:1). In intrinsic AKI, BUN and creatinine rise together (ratio ≈ 10:1).
2. **Creatinine Levels:** Ischemic ATN raises creatinine in 24-48 hours. Contrast-induced AKI peaks in 5-7 days. Nephrotoxic ATN may delay AKI onset (7-10 days).
3. **Indicators of Kidney Conditions:** Hyperkalemia, hyperuricemia, hyperphosphatemia, cell lysis, and hypocalcemia suggest rhabdomyolysis. High uric acid, normal creatine kinase, and urine uric acid/creatinine ratio indicate acute urate nephropathy or tumor lysis syndrome. Severe hypercalcemia can lead to prerenal AKI. Severe anemia hints at hemolysis, multiple myeloma, or thrombotic microangiopathy.
4. **Complete Blood Count (CBC):** Leukocytosis is common in AKI. Leucopenia, thrombocytopenia suggest SLE or TTP. Anemia, rouleaux formation signal multiple myeloma. Microangiopathic anemia suggests thrombotic microangiopathy. Eosinophilia may suggest allergic interstitial nephritis or polyarteritis nodosa.
5. **Blood Chemistry:** CPK elevation occurs in rhabdomyolysis, myocardial infarction. Liver transaminase rise signals liver failure, hepatorenal syndrome. AKI often presents moderate hypocalcemia, hyperkalemia.
6. **Urine Chemical Indices:** Useful to distinguish prerenal from intrinsic AKI. Prerenal: low FeNa, low UNa, high urine creatinine/plasma creatinine ratio, high urine urea nitrogen/plasma urea nitrogen ratio, high urine specific gravity, high urine osmolality, high plasma BUN/creatinine ratio. ATN: high FeNa, high UNa, low urine creatinine/plasma creatinine ratio, low urine urea nitrogen/plasma urea nitrogen ratio, low urine specific gravity, low urine osmolality, plasma BUN/creatinine ratio 10-15.
7. **Imaging Studies:** Chest radiography crucial for volume overload, lung infiltrations (e.g., Wegener's granulomatosis, Goodpasture's syndrome).
8. **Procedures:** Renal biopsy for unclear intrinsic AKI causes (e.g., glomerulonephritis, vasculitis, myeloma cast nephropathy).

Treatment: Prerenal AKI: Restore blood volume with appropriate fluid resuscitation. RBC transfusion for hemorrhagic hypovolemia. Caution with isotonic sodium chloride solutions to avoid renal vasoconstriction. In liver failure and hepatorenal

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syndrome, albumin infusion and vasoconstrictors may improve kidney function.

Pharmacologic Therapies: Limited roles for dopamine and fenoldopam in AKI. Natriuretic peptides, loop diuretics, mannitol have limited or no benefit. Supportive management includes electrolyte correction, addressing metabolic acidosis, nutritional support.

Renal Replacement Therapy (RRT): Options: intermittent hemodialysis (IHD), continuous venovenous hemofiltration (CVVH), peritoneal dialysis (PD). IHD common but risk of hypotension. CVVH suitable for unstable patients. PD involves dialysate instillation/removal in the peritoneal cavity.

Recent Advances: Biomarkers like TIMP-2, IGFBP7, hepcidin, KIM-1, NGAL, cystatin C, IL-18 emerging for early AKI detection. Serum cystatin C may detect small GFR reductions better than creatinine. Urinary cystatin C and IGFBP7/TIMP-2 combo show promise in early AKI detection.

MATERIALS AND METHODS

We conducted a prospective hospital-based observational study at Raichur Institute of Medical Science, Raichur, spanning a period of two years. The study included 150 patients admitted to the emergency medical ICU. The inclusion criteria comprised patients aged over 18 years who met the 2012 KDIGO criteria for AKI. Exclusion criteria included patients below 18 years, those developing AKI more than 48 hours after admission or previously hospitalized elsewhere for over 48 hours, and patients with chronic kidney disease.

The methodology involved admitting patients to the medicine ICU through the emergency, conducting detailed history and clinical examinations and verifying comorbid conditions, assessing baseline parameters at admission, including serum creatinine, serum sodium, serum potassium, liver functions, hemoglobin, total leukocyte count, and platelet count, daily clinical examinations during the hospital stay, monitoring urine output every 6 hours on the first day and every 24 hours thereafter for general ward patients, and every 6 hours for ICU patients. Noting the development of specific complications such as hypotension, oliguria, acute lung injury (ALI), acute respiratory distress syndrome (ARDS), encephalopathy, sepsis, thrombocytopenia, and the need for mechanical ventilation. Defining hypotension as systolic BP <90 mmHg or requiring inotropes to maintain a mean arterial pressure (MAP) of 65 mmHg. Oliguria was defined as urine output <0.5 ml/kg/hour for over 6 hours, and anuria as urine output <100 ml for 12 hours. ARDS was defined by the Berlin definition. Encephalopathy was identified as a decline in mental status with relevant urea and creatinine levels, excluding intracranial pathology or liver cirrhosis. Thrombocytopenia and anemia were defined as platelet count <100,000/dL and hemoglobin (Hb) <9 g/dL, respectively. Sepsis was defined as per the Surviving Sepsis Campaign definition, and hypertension as systolic BP >150 mmHg and/or diastolic BP >90 mmHg. Hyponatremia and hypernatremia were defined as serum sodium levels <135 meq/L and >145 meq/L, respectively. Individualized workup was conducted for each patient to achieve a definitive diagnosis, involving serial serum creatinine tests and noting the maximum AKI stage and serum creatinine. Patient management, including antibiotic use, enteral nutrition, fluids, vasopressors, hypoglycemia management, dialysis decisions, and blood product administration, followed the unit's policy. Dialysis (renal replacement therapy) initiation and type (hemodialysis, slow low efficiency dialysis) were determined in consultation with nephrologists, and the number of sessions was individualized. Patients were followed until discharge or death. Complete renal recovery was defined as serum creatinine returning to <1.5 mg/dL and a non-oliguric state, while partial recovery indicated declining serum creatinine at discharge without returning to normal. Data were recorded in Microsoft Excel and analyzed using SPSS software version 17.0. Age and disease duration were categorized and expressed as percentages. Gender, complications, AKI stage, and outcomes were presented as frequency and percentages for qualitative variables. Hemoglobin, platelet count, serum creatinine at admission and discharge, total leukocyte count, sodium, and potassium were presented as mean values (standard deviation). Data were graphically represented using bar diagrams.

To assess the relationship between age category, gender, disease stage, sodium and potassium levels, anemia, leukopenia, thrombocytopenia, and outcomes, a chi-squared test was employed. The comparison of serum creatinine levels at admission and discharge, SGOT, and SGPT levels between survivors and non-survivors was conducted using an independent t-test. Statistical significance was set at $p < 0.05$.

RESULTS

Between 2021 and 2022, we studied 150 patients with community-acquired AKI, with a mean patient age of 49.8 years. Most patients were aged between 46 and 60 years (39%), followed by those over 60 years (25%), and those under 30 years (17%). The majority were male (58%). Common complications included anemia (54%), hyponatremia (48%), sepsis (29.3%), hyperkalemia (22.7%), hypokalemia (20.7%), thrombocytopenia (15.3%), and leukopenia (4.7%).

Regarding AKI stage and renal replacement therapy (RRT), 40 patients (26.7%) were in stage 1, 55 (36.7%) in stage 2, and 55 (36.7%) in stage 3. The mean creatinine level was 3 in non-survivors and 2.8 in survivors, with 18 patients (12%) requiring RRT.

The etiology of community-acquired AKI was primarily acute gastroenteritis in 66 patients,

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followed by community-acquired pneumonia (17.3%), hepatorenal syndrome secondary to chronic liver disease (8.6%), snake bites (6.6%), sepsis (6.6%), urinary tract infections (3.3%), and rickettsial fever (1.3%). Prerenal AKI was the most common form, predominantly caused by acute gastroenteritis, followed by intrinsic renal AKI. Among non-infective causes, snake bites were the most common. In terms of outcomes, 27 patients died, primarily due to sepsis and pneumonia. Multiorgan failure and hospital-acquired infections were the leading causes of death. Anemia, sepsis, hypotension, thrombocytopenia, leukopenia, and encephalopathy were significantly associated with mortality. There was also a statistically significant association between AKI stage and outcome, with nearly half of non-survivors in stage 3 AKI ($p = 0.04$).

In total, 150 participants were recruited for the study purpose. Out of 150, 58 (39%) were 46-60 years of age, 37 (25%) were >60 years and 26 (17%) were less than or equal to 30 years of age. Of all the participants more than half 87 (58%) of them were men and the remaining 63 (42%) were women. Nearly three-fourth of the patients had a duration of ≤7 days and one-fourth had >7 days. Of all the participants 44% were diagnosed with acute gastroenteritis ,17.3 % community acquired pneumonia, hepatorenal syndrome 8.6%, snake bite and sepsis 6.6%.

Out of 150 patients, 55 (36.7%) were in acute kidney disease of stage 2, another 55 (36.7%) was in stage 3 and the remaining 40 (26.7%) were in AKI stage1. Of all the AKI patients, 18(8%) had undergone dialysis and the rest did not require dialysis. The major complications were anaemia (54%), followed by Hyponatremia (48%), sepsis (29.3%), Hyperkalaemia (22.7%) and hypokalemia in 20.7% of the patients respectively. Others were Thrombocytopenia (15.3%) and Leukopenia (4.7%). Out of 150 patients with AKI, 27 (18%) were expired and 123 (82%) showed improvement. The mean (SD) serum creatinine was 3 (1.3) in non-survivors and the same was 2.8 (1.7%) in survivors. The mean (SD) creatinine at death among non-survivors was 4.6 (2.6) and at discharge among the survivors was 1.6 (1.1) among survivor. There was a significant difference in serum creatinine at discharge was observed between the survivors and non-survivors with the p value of <0.001.

Nearly half of the patients in non-survivors were in stage 3 of AKI and the same was 34% in survivors. Similarly, the proportion of patients with stage 2 AKI was higher in non survivors than survivors (44.4% vs 35%). There was a significant association observed between the severity of AKI and the outcome of the patient ($p=0.04$).

Table 1: Sociodemographic profile and etiology of the AKI

Parameter	Groups	Number	Percentage
Age groups (years)	<30	26	17.3
	31-45	29	19.3
	46-60	58	38.7
	>60	37	24.7
Gender	Male	87	58
	Female	63	42
Hospital stay duration	<7 days	109	72.7
	>7days	41	27.3
Causes	Acute gastroentiritis	66	44
	Community acquired Pneumonia	26	17.3
	Hepatorenal syndrome Secondary to chronic liver disease	13	8.6
	Snake bite	10	6.6
	Sepsis of undetermine origin	10	6.6
	Urinary tract infection	5	3.3
	Miningitis	4	2.6
	Seizure disorder	4	2.6
	Cerebrovascular Accident	4	2.6
	Cardiorenal syndrome secondary to cardiac Failure	4	2.6
	Pancreatitis	2	1.3
	Rickettesial fever	2	1.3

Table 2: Staging, Treatment and outcomes of the participants

Parameter	Groups	Number	Percentage
AKI stage	1	40	26.7
	2	55	36.7
	3	55	36.7
Dialysis done	Yes	18	12
	No	132	88
Complications	Anaemia	81	54.0
	Hyponatremia	72	48.0
	Hypokalaemia	31	20.7
	Sepsis	44	29.3
	Thrombocytopenia	23	15.3
	Leukopenia	7	4.7
	Hypoglycaemia	1	0.7
	ARDS	1	0.7

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	Hypernatremia	9	6.0
	Hyperkalaemia	34	22.7
	Encephalitis	1	0.7
Outcomes	Death	27	18.0
	Improved	123	82.0

Table 3: Investigations done for the participants.

Parameter	Mean	SD	Median	Minimum	Maximum
HB	11.2	3.1	10.9	3.1	18.8
TLC	14777	11475.4	12600	1400	93000
Platelet	2.5	1.4	2.3	0.1	6.3
Sodium	134.3	7.8	134.5	103	158
Potassium	4.3	0.9	4.2	1.5	7.6
SGOT	35.7	56.6	24	11	390
SGPT	32.5	40	24	8	272
Serum Creatinine at admission	2.8	1.6	2.4	0.8	14.8
Nadir Creatinine	3.2	2	2.7	0	16
Serum Creatinine at discharge	2.1	1.8	1.4	0.6	9

DISCUSSION

All patients were evaluated by clinical history, examination and laboratory investigations. A prospective observational study done on community acquired acute kidney injury for period of 2 year in Raichur Karnataka region.

Important observation was most common cause of community acquired AKI is Acute gastroenteritis consist of 66 (44%) cases. In recent study done in Chennai by M Jayakumar most common cause was acute gastroenteritis.[29]

Similarly study done in Lucknow, Uttar Pradesh by Anupam Kaul, acute gastroenteritis consist of 54 (14.8%) cases 2nd most common cause of AKI in that study.[30] Whereas in study done in Himachal Pradesh by S. Kumar in Indira Gandhi college Acute gastroenteritis consist of 21 (20.6%) cases.[31] Study done in Puducherry India 2019 only 9(4.8%) cases.32

In our region Acute gastroenteritis was most common cause of AKI may be due to poor hygiene, overcrowding and poverty contamination of a drinking water. Geographical difference is seen for cause of community Acquired AKI by comparing our study with study done in Himachal Pradesh,[33] and puduchery,[32] where acute gastroenteritis cases were less. Second most common etiological factor for AKI is community acquired pneumonia comprise of 26 (17.3%).

Study done in Himachal Pradesh by Sanjay Vikrant 9.1% were pneumonia cases. Study done in Puducherry had 10(4.8%) cases.[31] Suspected cases sent sputum for tubercular bacilli most of the cases were positive. In our study snake bite cases comprise of 10 (6.6%). In Puducherry snake bite cases were 49 out of 186 and study done in Chennai by Muthusethupathi, only 6 out of 186 cases.[32] In our study all snake bite cases underwent renal replacement therapy.

In India AKI due to acute gastroenteritis constitute 20.6 to 30.5%.[33] In our study it was 44% and our study prerenal AKI is most common. Most of the patient were recovered with a intravenous fluid and antibiotics.

In our study male preponderance is seen with 58% were male which is similar to other study. In our study mortality is 18% with sepsis due to pneumonia was most common cause of death.

Study done in Puducherry in 2019 mortality due to community acquired AKI is 15.1% with common cause was snake bite.[32] Study done in Himachal Pradesh community Acquired AKI had 9.1% mortality.[31] 18% of patients required renal replacement theraphy in our study most of the cases were snake bite. Reason may be the delay in hospitalization. Study done in Puducherry had 22% patients underwent renal replacement theraphy. 96 Study done in Himachal Pradesh it was 53% .[31]

Study done in Lucknow, requirement of renal replacement therapy is 83%.[30] Limitation of our study is our study limited to only community acquired acute Kidney injury excluded hospital acquired and surgical and obstetrical causes.

CONCLUSION

Aetiology was multi factorial with most common cause was Acute Gastroenteritis followed by Pneumonia and Decompensated chronic liver disease. Incidence more among elderly people with a male preponderance. Most common observed laboratory parameter are anaemia, hyponatremia, hypokalaemia, sepsis. Early recognition of the disease and early intervention into disease management prevented many patients progressing for the need of dialysis. Non survivors were mainly in people of Age >46year with male preponderance. Half of the Non survivors were in stage 3 of KIDGO classification and indicated high mortality. CAAKI is a common, serious and often preventable disease and requires high degree of suspicion and early initiation of treatment to minimise morbidity and mortality.

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