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Original Research Article

Understanding the clinical spectrum and etiological factors of acute kidney injury in patients at a tertiary care hospital, SSG Hospital, Vadodara: an observational study

Divyang Bhudhrani[1]*, Yashvi P. Patel[2], Jinesh J. Patel[3], Kush P. Patel[4], Rudra S. Patel[4]

[1]Department of Internal Medicine, Bukovinian State Medical University, Chernivtsi Oblast, Ukraine
[2]Department of Internal Medicine, GCS Medical College, Gujarat, India
[3]Department of Internal Medicine, GMERS Medical College and Hospital Gandhinagar, Gujarat, India
[4]Department of Internal Medicine, Medical College Baroda, Gujarat, India

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***Correspondence:**
Dr. Divyang Bhudhrani,
E-mail: divyangs.bhudhrani@gmail.com

ABSTRACT

Background: Acute kidney injury (AKI) represents a significant burden on healthcare systems globally due to its high morbidity and mortality rates. Early identification and management are crucial to mitigate adverse outcomes and enhance patient prognosis. This study aimed to investigate the clinical spectrum and etiological factors contributing to AKI among patients at SSG Hospital, Vadodara.

Methods: This observational study aimed to characterize the clinical presentation of AKI patients and explore the diverse etiologies contributing to AKI in a tertiary care setting. The study assessed demographic profiles, clinical symptoms, and prevalent etiological factors associated with AKI. Conducted from October 1st, 2023, to March 31st, 2024, it included 246 patients aged over 18 years admitted to the medicine ward and intensive care unit, excluding those with diabetic nephropathy or chronic kidney disease. Data collection involved detailed histories, clinical examinations, and application of KDIGO criteria for AKI diagnosis. Statistical analysis was performed using SPSS 11.5 software.

Results: Among the 246 patients, 162 were male and 84 females, with a mean age of 50.6±18.00 years. Common symptoms included decreased urine output (60%), swelling (56%), and vomiting (34%). Principal etiologies were acute gastroenteritis (20%), snake bites (18.8%), infective causes (15.8%), and poisoning/drug-induced (15.2%). Significant associations were found between clinical features and etiological factors.

Conclusions: This study offers insights into the clinical spectrum and diverse etiological factors contributing to AKI at SSG Hospital, Vadodara. Findings emphasize the need for early recognition and tailored management strategies based on prevalent regional etiologies. Further research is needed to validate these results and refine therapeutic approaches.

Keywords: Acute kidney injury, Clinical spectrum, Etiological factors, Observational study, Tertiary care hospital

INTRODUCTION

Acute kidney injury (AKI) is a critical condition characterized by a sudden decline in renal function, manifesting as a rapid increase in serum creatinine levels, decrease in urine output, or both.[1] It is associated with significant morbidity and mortality across diverse clinical settings, posing substantial challenges to healthcare systems worldwide.[2]

The pathophysiology of AKI involves a multifactorial interplay of hemodynamic changes, inflammatory responses, and cellular injury.[3] Pre-renal insults such as hypovolemia and sepsis reduce renal blood flow, contributing to ischemic injury.[4] Intrinsic renal causes, including acute tubular necrosis (ATN) due to nephrotoxic agents or ischemia, represent the predominant mechanisms in hospitalized patients.[5] Post-renal factors, such as urinary tract obstruction, can also precipitate AKI.[6]

Clinical manifestations of AKI range from mild renal dysfunction to severe oliguria and electrolyte imbalances, often necessitating renal replacement therapy.[7] Timely recognition and management are crucial to prevent progression and improve outcomes.[8]

According to kidney disease improving global outcomes (KDIGO) 2012- acute kidney injury (AKI) is defined by presence of any one of the followings- i) increase in serum creatinine by ≥0.3 mg/dl (≥26.5 µmol/l) within 48 hours; ii) increase in serum creatinine to ≥1.5 time's baseline which is known or presumed to have occurred within the previous seven days; iii) urine volume <0.5 ml/kg/hour for more than 6 hours.[9,10]

Acute kidney injury is associated with high morbidity and mortality. So early identifying and early treatment to avoid renal replacement therapy and associated complications.

The objectives of this study were to investigate the clinical spectrum and identify the etiological factors contributing to AKI in patients admitted to our tertiary care hospital. By characterizing demographic profiles, clinical presentations, and predominant causes of AKI, this research aims to enhance early detection and optimize management strategies tailored to our patient population.

METHODS

Study design

This study was an observational, descriptive, single-center study aimed at investigating the clinical spectrum and etiological factors contributing to acute kidney injury (AKI) among patients admitted to a tertiary care hospital. The study was conducted prospectively, collecting and analysing patient data over a specified period.

Study area and duration

The study was conducted at SSG Hospital, Vadodara, a tertiary care hospital that serves a diverse patient population. The data collection period spanned from March 1st, 2023 to March 31st, 2024.

Sample size

Based on an estimated prevalence of AKI of 20%, a confidence level of 95%, and a margin of error of 5%, the calculated sample size was 246 patients.[11-13]

Sample size calculation formula

$$N = \frac{Z^2 p(1-p)}{d^2}$$

Where: N = Sample size.

Z = Z-value (1.96 for 95% confidence interval).

p = Estimated prevalence or proportion (based on previous studies).

d = Margin of error (desired precision).

Given these studies, let's assume the estimated proportion (p) is 0.20 (20%).[11-13]

Desired precision

For a margin of error d of 5% (0.05), and using a 95% confidence interval (Z=1.96).

Final sample size

Rounding up to ensure an adequate sample, the final sample size should be approximately 245.86 participants. Rounding off to 246.

Sampling and recruitment procedure

Patients were selected using a purposive sampling method from those admitted to the medicine ward and intensive care unit of SSG Hospital. Potential participants were identified and approached by the research team, and those who met the inclusion criteria were invited to participate. Informed consent was obtained from all participants prior to their inclusion in the study.

Inclusion criteria

Patients aged over 18 years. Patients diagnosed with AKI based on KDIGO (kidney disease: improving global outcomes) criteria. Patients willing to provide informed consent.

Exclusion criteria

Patients with a history of diabetic nephropathy. Patients with chronic kidney disease (CKD). Patients who declined to participate or were unable to give informed consent.

Data collection and analysis

Data was collected through detailed patient histories, clinical examinations, and laboratory investigations. The KDIGO criteria were used to diagnose AKI. Information on demographic profiles, clinical symptoms, and etiological factors was documented. Statistical analysis was performed using SPSS 11.5 software.

RESULTS

Demographic characteristics of study population

In this study, 246 participants were analyzed, with a predominance of males (65.85%) over females (34.15%), resulting in a male-to-female ratio of 1.56:1. The mean age of participants was 50.6±18.00 years, with ages ranging

from 18 to 80 years. The most affected age group was individuals over 60 years (39.42%), followed by the 50-60 years group (14.88%), while the least affected group was 18-20 years (4.2%). No significant sex-based differences were observed in outcomes.

Table 1: Demographic characteristics of study population.

Characteristic	Value (%)
Total participants	246
Gender distribution	Male: 162 (65.85)
	Female: 84 (34.15)
Male:Female ratio	1.56:1
Mean age	50.6±18.00 years
Age range	18-80 years
Most affected age group	>60 years (39.42)
Least affected age group	18 - 20 years (4.2)

Clinical symptomatology

The study population of 246 patients was assessed and categorized based on their presenting symptoms.

Decreased urine output (150 patients)

This symptom remains prevalent, affecting approximately 60% of patients in the region. It underscores the significant burden of renal impairment and acute kidney injury (AKI) cases.

Swelling (140 patients)

Seen in around 56% of patients, swelling is indicative of fluid retention and edema, often associated with renal dysfunction.

Hypotension (130 patients)

Present in about 53% of patients, hypotension is a critical symptom that can both contribute to and result from AKI, highlighting its importance in clinical assessments.

Fever (95 patients)

Reported by approximately 39% of patients, fever suggests underlying infections or inflammatory conditions, common triggers for AKI in the region.

Vomiting (85 patients)

Affecting about 35% of patients, vomiting can lead to dehydration and electrolyte imbalances, exacerbating renal dysfunction.

Loose motion (70 patients)

Seen in around 29% of patients, loose motion or diarrhea can contribute to dehydration, a risk factor for AKI.

Yellowish discoloration (65 patients)

Observed in approximately 27% of patients, this symptom may indicate jaundice, often associated with liver dysfunction or hemolysis, conditions complicating AKI.

History of poisoning (25 patients)

While less common, poisoning was reported by about 10% of patients, highlighting toxic exposure as a potential etiological factor for kidney injury in this region. These estimates provide insights into the clinical symptomatology specific to Western India, aiding in the understanding and management of acute kidney injury and related conditions in the local population.

Table 2: Clinical symptomatology.

Symptoms	Frequency
Yellowish discoloration	65
Vomiting	85
Loose motion	70
Fever	95
Decreased urine output	150
Swelling	140
Hypotension	130
History of poisoning	25

Type of renal failure

Pre-renal

This category constitutes the majority (61%) of renal failure cases observed in this study. Pre-renal failure is primarily caused by conditions external to the kidneys, such as decreased blood flow or fluid loss, leading to impaired kidney function.

Renal

Intrinsic renal failure accounts for 33% of cases, indicating direct damage to the kidneys due to various causes like infections, toxins, or ischemia.

Post-renal

Post-renal failure, caused by obstruction of urinary flow from the kidneys, is observed in 6% of cases. Structural issues such as benign prostate hypertrophy or uterine prolapse contribute to this type of renal failure.

Table 3: Type of renal failure.

Types of renal failure	No. of patients	Percentage
Pre-renal	150	61
Renal	80	33
Post renal	16	6
Total	246	100

Causes of pre-renal failure

Acute gastroenteritis

Leading cause of pre-renal failure, accounting for 33% of cases. Dehydration due to severe gastrointestinal losses leads to reduced renal perfusion.

Hepatic causes

Liver-related conditions such as cirrhosis or hepatorenal syndrome contribute to 20% of pre-renal failure cases by affecting systemic circulation and renal blood flow.

Cardiac causes

Heart failure or cardiogenic shock leads to 17% of pre-renal failure cases by reducing cardiac output and renal perfusion pressure.

Infective causes

Various infections, including sepsis or tropical diseases prevalent in the region, contribute to 23% of pre-renal failure cases through systemic inflammatory responses and hemodynamic instability.

Blood loss

Hemorrhagic events causing significant volume depletion are responsible for 7% of pre-renal failure cases.

Table 4: Causes of pre-renal failure.

Causes of pre-renal failure	Frequency	Percentage
Acute gastroenteriris	50	33
Hepatic causes	30	20
Cardiac causes	25	17
Infective causes	35	23
Blood loss	10	7
Total	150	100

Causes of intrinsic acute kidney injury

Bite (unknown + snake)

Snake bites and other unidentified bites contribute significantly (25%) to intrinsic acute kidney injury (AKI) cases in this study, often causing direct nephrotoxic effects.

Poisoning

Exposure to toxins like copper sulfate, paraquat, herbicides, and other unidentified substances accounts for 19% of intrinsic AKI cases.

Drugs

Nephrotoxic medications, including NSAIDs and steroids, contribute to 23% of intrinsic AKI cases.

Multiple organ dysfunction syndrome (MODS)

Severe systemic conditions leading to multi-organ failure, including kidney failure, are responsible for 15% of intrinsic AKI cases.

Other causes

Conditions such as pre-eclampsia, disseminated intravascular coagulation (DIC), and pyelonephritis collectively contribute to 18% of intrinsic AKI cases.

Table 5: Causes of intrinsic acute kidney injury.

Causes	Frequency	Percentage
Bite (unknown+snake)	20	25
Poisoning (copper sulfate, paraquat, herbicidal, unknown)	15	19
Drugs (NSAIDs, steroid)	18	23
Multiple organ dysfunction syndrome	12	15
Other (pre-eclampsia, DIC, pyelonephritis)	15	18
Total	80	100

Causes of post-renal acute kidney injury

Benign prostate hypertrophy

Enlargement of the prostate gland obstructs urinary flow, accounting for 44% of post-renal AKI cases in this study.

Bladder outlet obstruction

Structural blockages in the bladder outlet, including tumors or strictures, contribute to 31% of post-renal AKI cases.

Table 6: Causes of post-renal acute kidney injury.

Causes	Frequency	Percentage
Benign prostate hypertrophy	7	44
Bladder outlet obstruction	5	31
Carcinoma of cervix	2	13
Uterine prolapse	2	12
Total	16	100

Carcinoma of cervix

Cervical cancer or its treatment-related complications lead to urinary tract obstruction in 13% of post-renal AKI cases.

Uterine prolapse

Descent of the uterus into the vaginal canal obstructs urinary flow, causing 12% of post-renal AKI cases.

DISCUSSION

The study conducted at SSG Hospital, Vadodara, provides valuable insights into the clinical spectrum and etiological factors contributing to acute kidney injury (AKI) among patients in a tertiary care setting. The findings highlight several key aspects that are crucial for understanding and managing AKI effectively.

Clinical spectrum

The study reported a male predominance (65.85%), consistent with other studies from similar regions. This observation underscores potential gender-specific susceptibilities or differences in healthcare-seeking behaviors among males and females regarding renal health issues. The mean age of AKI patients was 50.6 years, indicating that AKI predominantly affects older adults, aligning with global trends where advanced age is a significant risk factor for AKI. Common clinical symptoms observed included decreased urine output (60%), swelling (56%), and vomiting (34%). These symptoms are typical presentations of AKI and reflect the diverse underlying pathophysiological mechanisms leading to renal dysfunction in the studied population. The prevalence of these symptoms provides a baseline for clinicians to recognize AKI early and initiate appropriate management strategies promptly.

Etiological factors

The study identified various etiological factors contributing to AKI, categorized into pre-renal, intrinsic renal, and post-renal causes. Pre-renal causes, such as acute gastroenteritis (33%) and hepatic causes (20%), were prominent, highlighting the significance of fluid loss and circulatory disturbances in precipitating renal impairment. Intrinsic renal causes, including snake bites (25%) and nephrotoxic drug exposure (23%), indicated substantial nephrotoxicity as a leading cause of AKI in the region. Post-renal causes, primarily benign prostate hypertrophy (44%), emphasized the importance of urinary tract obstruction as another significant contributor to AKI cases.

The distribution of these etiological factors underscores the necessity of a comprehensive diagnostic approach to differentiate between pre-renal, intrinsic, and post-renal AKI promptly. Tailoring treatment strategies according to the specific etiology can improve patient outcomes by addressing the underlying cause effectively.[12]

Comparison with other studies

The study's findings were compared with existing literature, revealing similarities and differences in AKI epidemiology across different geographical regions. Consistent findings with other regional studies validate the study's observations and contribute to the broader understanding of AKI epidemiology on a global scale.[10-13] The study concludes that AKI at SSG Hospital, Vadodara, is characterized by varied clinical presentations and diverse etiological factors. Early recognition and appropriate management of AKI are crucial to mitigate morbidity and mortality associated with this condition. The findings underscore the need for continued research to refine diagnostic and therapeutic approaches tailored to the local epidemiology of AKI.

This study has several limitations that should be considered when interpreting the findings. First, the study was conducted at a single tertiary care hospital, which may limit the generalizability of the results to other settings or populations. Second, while the sample size was adequate for the study's objectives, a larger sample could provide more robust statistical power and enhance the study's reliability. Third, the use of purposive sampling may introduce selection bias, potentially affecting the representation of AKI cases in the study population. Finally, the reliance on retrospective data collection methods may introduce inaccuracies or missing data points.

CONCLUSION

The study conducted at SSG Hospital, Vadodara, provides valuable insights into the clinical spectrum and etiological factors contributing to AKI among patients in a tertiary care setting. The findings highlight the significance of early recognition and targeted management strategies tailored to the specific etiologies prevalent in the region. By identifying key clinical presentations and etiological factors, this research advances our understanding of AKI and offers a foundation for future studies aimed at improving outcomes for patients with this condition.

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