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A Study on the Etiology and Outcome of Acute Kidney Injury Patients in the Nephrology Center of the Combined Military Hospital in Dhaka

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Abstract **Original Research Article**

Background: Acute kidney injury (AKI) is increasingly prevalent in both developing and developed countries, posing significant morbidity and mortality risks. Many AKI cases can be prevented through interventions at individual, community, and in-hospital levels. However, there has been limited research on acute kidney injury (AKI) within the Armed Forces in Bangladesh. ***Aim of the study:*** This study aimed to assess the etiology and outcome of acute kidney injury patients in the nephrology center of the Combined Military Hospital in Dhaka. ***Methods:*** This cross-sectional study was conducted at the nephrology center of Combined Military Hospital (CMH) in Dhaka, Bangladesh, from September 20, 2012, to March 20, 2013. A total of 50 acute kidney injury (AKI) cases were included using purposive sampling. The study examined the etiology, mode of presentation, laboratory findings, management, and outcomes of AKI cases. Data were analyzed using MS Office tools and SPSS version 23.0. ***Results:*** In this study, more than one-fourth of the participants (26%) experienced AKI due to hypovolemia from diarrhea, vomiting, or hemorrhage. NSAIDs and/or rhabdomyolysis accounted for 14% of cases. Other potential etiologies included septicemia (12%) and falciparum malaria (8%). Most patients (84%) recovered completely, while 4% died due to severe falciparum malaria and septicemia. Complications included peritonitis (8%), RTI (6%), UTI (6%), wound infection (8%), pulmonary edema (4%), and septicemia (4%). ***Conclusion:*** Hypovolemia from diarrhea, vomiting, or hemorrhage, NSAIDs, rhabdomyolysis, septicemia, and falciparum malaria are common etiologies of AKI. Over eighty percent of AKI cases completely recover through one or more treatment procedures, including conservative treatment, hemodialysis, peritoneal dialysis, and continuous renal replacement therapy (CRRT).

Keywords: Acute kidney injury, AKI, Etiology, Nephrology, Oliguria, Hypovolemia, Dialysis.

INTRODUCTION

Acute kidney injury (AKI) has supplanted the term acute renal failure in medical terminology [1]. Clinically, AKI is characterized by a rapid decline in kidney function, leading to an inability to maintain fluid, electrolyte, and acid-base balance [2]. It is increasingly prevalent and poses a significant health challenge in our country [3]. AKI has been documented to complicate 5% of all medical and surgical admissions in a comprehensive study conducted in the United States [4]. Data on the incidence, causes, and outcomes of AKI patients are sparse in our local context [5]. A statistical analysis of patients admitted to the Nephrology department of BSMMU over a decade (1973-1983) indicated that AKI cases constituted 8.8% of all admissions during that period in Bangladesh [5]. The etiological factors contributing to acute kidney injury (AKI) include hypovolemia from various causes, nephrotoxicity induced by non-steroidal anti-inflammatory drugs (NSAIDs), septicemia, rhabdomyolysis following physical trauma or strenuous exercise, glomerulonephritis, acute pyelonephritis, malaria caused by Plasmodium falciparum, obstructive uropathy, contrast-induced nephropathy, vasculitis, HELLP syndrome (Hemolysis, elevated liver enzymes, low platelets), snake bites, and thrombotic thrombocytopenic purpura (TTP) [6]. Oliguria and

vomiting are common initial symptoms, while other manifestations may include edema, fever, anorexia, jaundice, and diarrhea [7]. Treatment of AKI primarily involves supportive measures such as medication adjustments, appropriate fluid management, correction of electrolyte imbalances, and addressing acid-base disturbances [8]. To optimize cardiac output and renal blood flow, it is crucial to promptly identify and manage acute complications associated with acute kidney injury (AKI), such as hyperkalemia, acidosis, pulmonary edema, and bleeding, and initiate dialysis as necessary to prevent uremic complications from developing [9]. AKI often leads to life-threatening pulmonary edema due to inappropriate fluid administration, electrolyte imbalances, uremia, metabolic acidosis, anemia, infections, and cardiopulmonary issues including arrhythmias, pericarditis, and pericardial effusion [10]. Early intervention typically results in rapid improvement in renal function; however, in some cases, treatment may be ineffective, leading to established renal failure [11]. The objective of this study was to assess the etiology and outcome of acute kidney injury patients.

METHODOLOGY

This was a cross-sectional study that was conducted at the nephrology center of the Combined Military Hospital (CMH), Dhaka, Bangladesh from 20 September 2012 to 20 March 2013. As the study subjects, a total of 50 cases of acute kidney injury (AKI) were included in this study. It was a purposive sampling technique that facilitated sample selection. The study examined cases based on etiology, presentation mode, laboratory results, management strategies, and outcomes. Approval for the study was obtained from the hospital's ethical committee, and written consent was duly acquired from all participants before data collection. As per the study's inclusion criteria, 50 patients admitted to the Nephrology Center in CMH-Dhaka during the study period were included, meeting the RIFLE criteria for AKI. Conversely, following the exclusion criteria of this study, patients with AKI who had comorbid conditions such as diabetes mellitus (DM), hypertension (HTN), left ventricular failure (LVF), liver cirrhosis, chronic kidney disease (CKD), and positive for hepatitis B surface antigen (HBsAg) were excluded from the analysis. Data were analyzed and processed using MS Office and SPSS version 23.0 software.

RESULT

The largest proportion of our participants (44%) belonged to the age group of 18-30 years, followed by 38% from the age group of 31-45 years. Most of the participants (44%) were male. In this study, the etiology of AKI was diverse among participants. Hypovolemia due to conditions such as diarrhea, vomiting, and hemorrhage accounted for more than one-fourth of cases (26%). NSAIDs and/or rhabdomyolysis were identified as the cause in 14% of cases. Septicemia (12%), falciparum malaria (8%), glomerulonephritis (4%), obstructive uropathy (4%), acute pyelonephritis (2%), contrast-induced nephropathy (4%), post-operative AKI (4%), vasculitis (4%), HELLP syndrome (2%), and snake bite (2%) were also identified as contributing factors in some cases. The mode of presentation among the cases included oliguria in 50%, edema in 40%, diarrhea in 22%, acidosis in 20%, shortness of breath in 16%, anuria in 12%, loin pain in 12%, fever in 10%, and drowsiness in 10% of the noticeable cases. Among our participants, the mean hemoglobin (Hb) level was 10.93 ± 2.86 g/dL, the mean urea level was 160.40 ± 80.20 mg/dL, the mean creatinine level was 4.40 ± 2.12 mg/dL, and the mean potassium level was 5.00 ± 1.88 mEq/L. In this study, 24 patients (42%) exhibited normal urinary findings, while 17 patients (34%) had the presence of red blood cells (RBCs), and 7 patients (14%) showed the presence of myoglobin in their urine samples. Upon analyzing the blood pH at admission, it was observed that 40 patients (80%) had normal blood pH, while 10 patients (20%) exhibited acidosis. In the current study, the majority of patients, 28 (56%), were managed with conservative treatment. Hemodialysis was required in 17 patients (34%), peritoneal dialysis in 4 patients (8%), and 2 patients (4%) required continuous renal replacement therapy (CRRT). In analyzing the outcomes of AKI, it was found that the majority of patients (84%) recovered completely, 12% recovered incompletely, and 4% of patients died. It was observed that 2% of patients died due to severe malaria, while another 2% succumbed to septicemia as the primary illness. In this study, complications that developed among the cases included peritonitis in 8%, respiratory tract infections (RTI) in 6%, urinary tract infections (UTI) in 6%, wound infections in 8%, pulmonary edema in 4%, septicemia in 4%, convulsions in 4%, hypoglycemia in 4%, and other complications in 8% of the cases.

Table 1: Age distribution of participants (N=50)

Age (Year)	n	%
18-30 Yrs.	22	44%
31-45 Yrs.	19	38%
46-60 Yrs.	6	12%
>60 Yrs.	3	6%

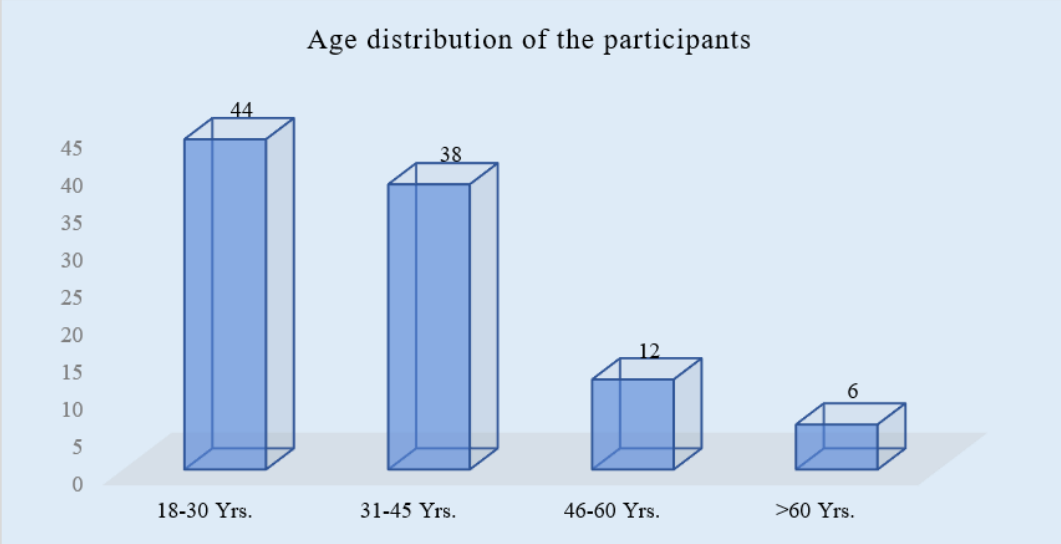

Figure I: Column chart showed age wise participants distribution (N=50)

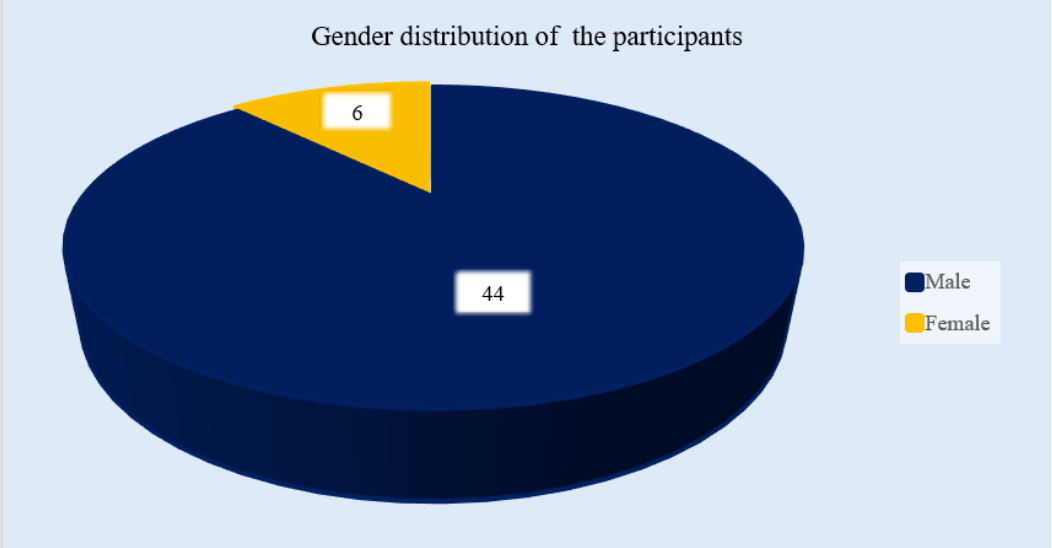

Figure II: Pie chart showed gender wise participants distribution

Table 2: Distribution of etiology of AKI (N=50)

Etiology of AKI	**n**	**%**
Hypovolemia	13	26%
NSAIDs	7	14%
Rhabdomyolysis	7	14%
Septicemia	6	12%
Falciparum malaria	4	8%
Glomerulonephritis	2	4%
Obstructive uropathy	2	4%
Acute pyelonephritis	1	2%
Contrast-induced nephropathy	2	4%
Post-operative	2	4%
Vasculitis	2	4%
HELLP syndrome	1	2%
Snakebite	1	2%

Table 3: Mode of presentation (N=50)

Presentation	n	%
Oliguria	25	50%
Oedema	20	40%
Diarrhea	11	22%
Acidosis	10	20%
Shortness of breath	8	16%
Anuria	6	12%
Loin pain	6	12%
Fever	5	10%
Drowsiness	5	10%
Hematuria	3	6%
Hypotension	3	6%
Hypertension	3	6%
Shock	2	4%
Convulsion	2	4%

Table 4: Haematological profile & blood biochemistry at admission (N=50)

Tests	Mean	SD
Haemoglobin (gm/dl)	10.93	2.86
WBC count (n x 10^9/L)	10.2	5.12
Platelet count (n x 10^9/L)	180.12	80.22
Blood urea (mg/dl)	160.4	80.2
Serum creatinine (mg/dl)	4.4	2.12
Serum sodium (mmol/L)	140	2.5
Serum potassium (mmol/L)	5	1.88
Serum chloride (mm0l/L)	96	4.22

Table 5: Other investigations at admission (N=50)

Investigations	Findings	n	%
CXR P/A view	Pulmonary oedema	20	40%
Plain X-ray of KUB	Renal/Ureteric calculi	5	10%
USG of KUB	Renal/Ureteric calculi	6	12%
ECG	Feature of hyperkalaemia	4	8%
Urine C/S	Growth of organism	6	12%

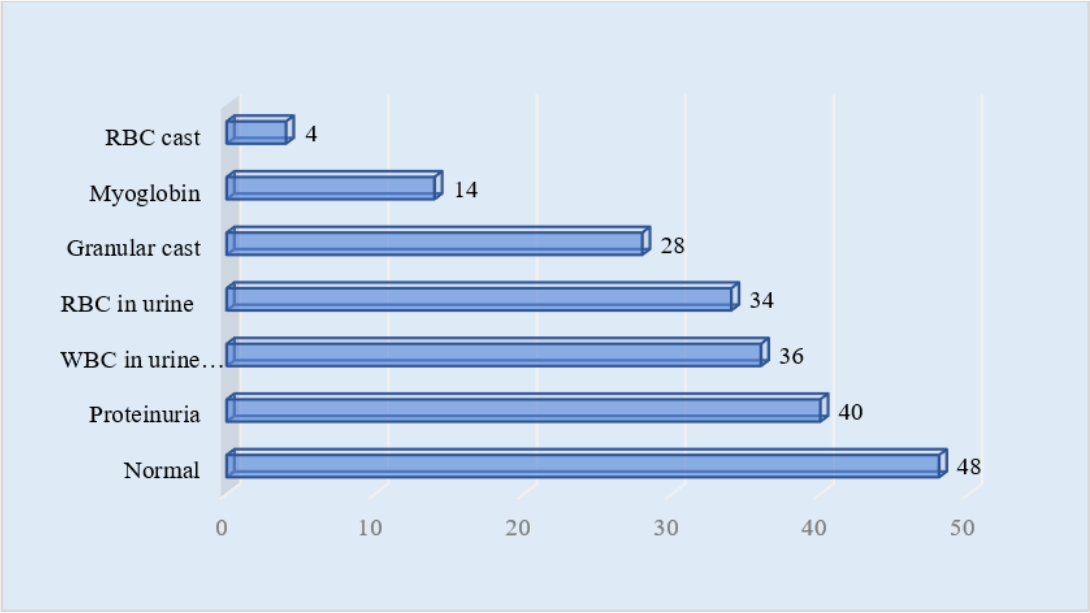

Figure III: Bar chart showed findings of routine urine analysis of the participants (N=50)

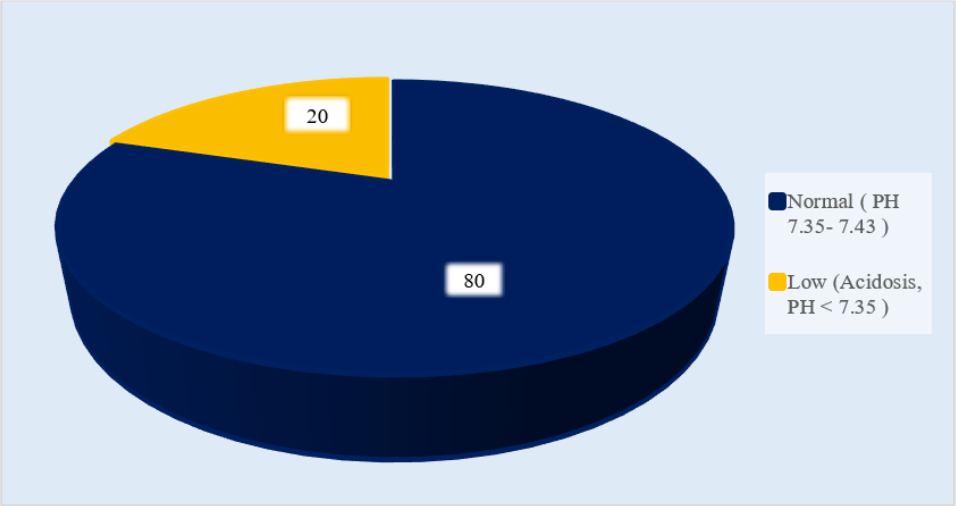

Figure IV: Pie chart showed blood p^{H} at admission wise participants (N=50)

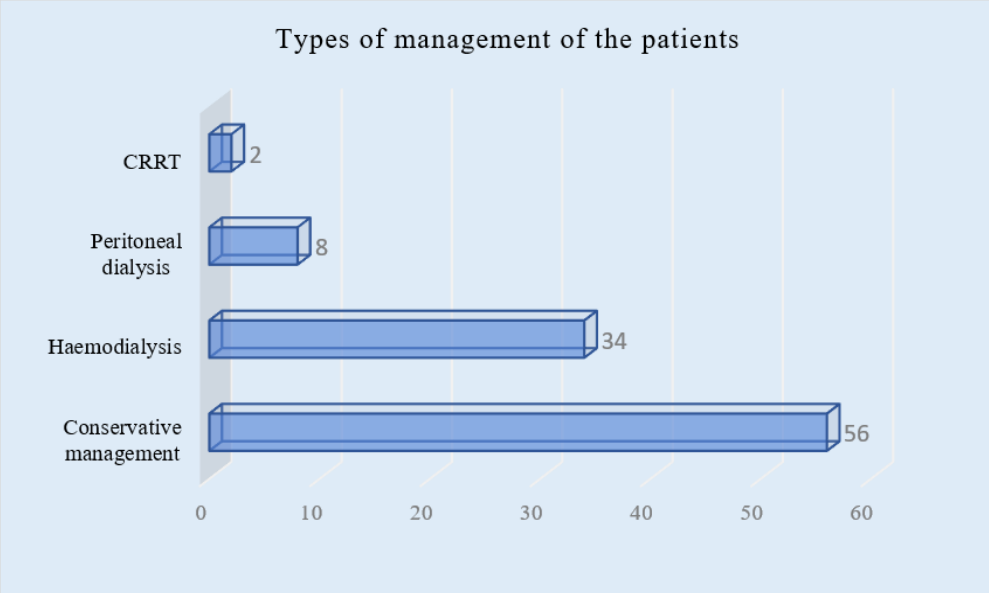

Figure V: Bar chart showed types of management (N=50)

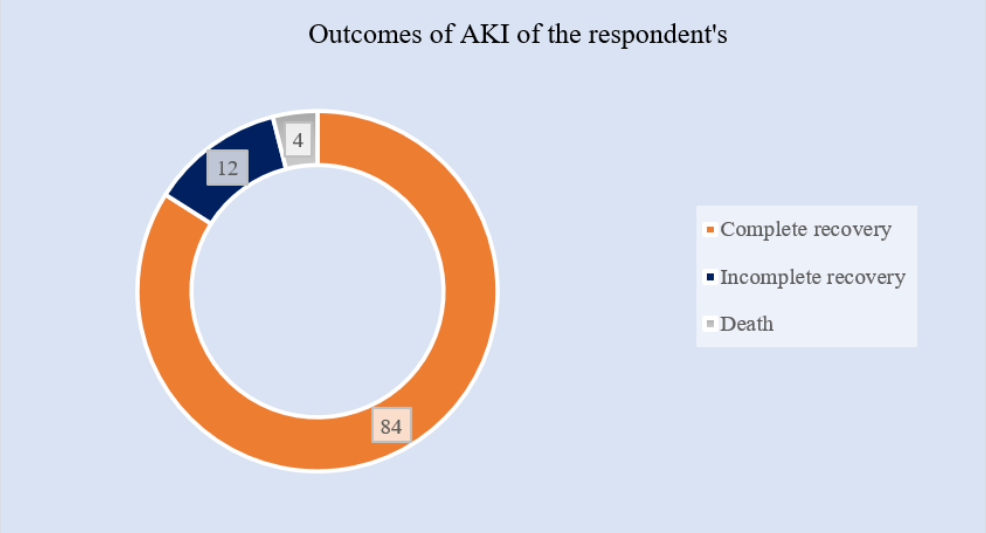

Figure VI: Ring chart showed outcomes of AKI wise participants (N=50)

Table 6: Cause of death as primary illness (N=50)

Cause	n	%
Severe malaria	1	2%
Septicemia	1	2%
Total	2	4%

Table 7: Complications developed (N=50)

Complications	n	%
Peritonitis	4	8%
RTI	3	6%
UTI	3	6%
Wound infection	4	8%
Pulmonary edema	2	4%
Septicemia	2	4%
Convulsion	2	4%
Hypoglycemia	2	4%
Others	4	8%

DISCUSSION

In this study, AKI was predominantly observed in males, comprising 88% of the patients, consistent with findings reported by Shivappa D R [10]. The primary cause of AKI in this study was hypovolemia (26%) due to diarrhea. Other common causes included NSAIDs (14%), and rhabdomyolysis (14%) due to factors like extreme physical exertion, prolonged seizure, crush injury, physical assault, and various drug-induced conditions such as statins and fibrates. Septicemia accounted for 12% of cases. Our findings were consistent with observations in India, where volume depletion from gastrointestinal fluid loss (35.2%) [12] and similar trends were noted locally [13]. In contrast, developed countries report a higher incidence of AKI due to trauma from accidents, cardiovascular surgeries, and cardiogenic shock, accounting for 60% of cases [14]. In this study, NSAIDs were responsible for inducing AKI in seven patients (14%) who had taken them for various types of pain, highlighting the common occurrence of NSAID-induced nephropathy and AKI among military personnel. Typically, military personnel often use various NSAIDs for exercise-induced injuries. However, in this study, these patients fully recovered after discontinuing the offending drugs and receiving adequate hydration and supportive care. AKI caused by various drugs and chemicals is becoming more common in developed countries, with higher risks associated with both short-term and long-term therapy, especially at higher doses [15]. Similarly, drug-induced AKI is prevalent in our country. There is a significant risk due to superstition, self-medication, and exploitation of illiterate individuals by unqualified practitioners, highlighting the need for prevention measures. NSAID users face a threefold higher risk of experiencing clinical AKI compared to non-users in the general population. It's crucial to exercise caution when prescribing NSAIDs to patients with conditions such as diabetes mellitus, hypertension, and heart failure [16]. In this study, 14% of patients developed AKI due to rhabdomyolysis following intense exercise and physical assault, a scenario not uncommon among military personnel and consistent with findings from other studies [17]. AKI is a frequent complication in critically ill patients. Those with septic AKI typically exhibit more severe illness, greater physiological abnormalities, and a higher overall disease burden compared to those with nonseptic AKI. Furthermore, septic AKI is linked independently to increased odds of mortality and prolonged hospital stays [18]. In our study, six patients (12%) developed AKI following septicemia. Acute pyelonephritis represents a severe form of urinary tract infection, ranging from mild discomfort to potentially life-threatening conditions. Complications may lead to renal function impairment, resulting in AKI and chronic renal scarring that can progress to chronic kidney disease (CKD) [19]. Additionally, one patient (2%) in our study developed AKI due to acute pyelonephritis, consistent with findings from other studies [20]. According to the World Health Organization, AKI occurs in less than 1% of cases of severe plasmodium falciparum malaria, but mortality rates can be as high as 45% in these instances [21]. In our study, 4 cases (8%) of AKI were attributed to severe falciparum malaria. Contrast-induced nephropathy has emerged as a notable cause of hospital morbidity and mortality due to the increasing use of iodinated contrast media in diagnostic and interventional procedures like angiography, especially in high-risk patients. It ranks as the third most common cause of hospital-acquired acute renal failure, following surgery and hypotension [22], which contrasts with findings from our study. The onset of AKI during the perioperative period is linked to heightened risks of morbidity and mortality. In our study, 2 cases (4%) developed AKI postoperatively despite having normal renal function before surgery, which aligns with findings from Murray P.'s study [23]. Snakebites are a significant health concern in Asia, where they represent a frequent occurrence. Studies from Nigeria have reported incidences of acute renal failure ranging from 1% to 10% following snakebites [25]. Blood urea, serum creatinine, and serum electrolytes are crucial indicators for diagnosing and managing AKI patients. In our study, the highest mean urea level

observed was 160.40 ± 80.20 mg/dl, and the mean creatinine level was 4.40 ± 2.12 mg/dl, which aligns with findings from another study [26]. Electrolyte imbalances were present in almost all patients. The most common treatment modality used was furosemide in conjunction with conservative management, employed in 56% of AKI cases, with favorable outcomes observed. In this study, hemodialysis was necessary for 17 (34%) patients, representing a cornerstone of patient management. Only 4 (8%) patients received peritoneal dialysis, mainly due to hypotension or other contraindications to hemodialysis. Continuous renal replacement therapy (CRRT) was administered to 1 (2%) hemodynamically unstable patient. Among the 50 AKI patients, 2 (4%) died from multiorgan failure due to severe falciparum malaria and septicemia. Infection was the most common complication of AKI, affecting 32 (64%) patients who required antibiotic treatment. The association between AKI and infections highlights the predisposition to infections in a uremic state, which remains a significant cause of mortality. The treatment outcomes in this study were favorable, with 42 (84%) patients achieving full recovery, 6 (12%) patients experiencing incomplete recovery with residual renal function impairment, and only 2 deaths resulting in a mortality rate of 4%. This contrasts with a previous study reporting a mortality rate of 10.98% during 1996–2008 [27], possibly due to different causes of AKI, younger patient ages, early detection, and effective management. The encouraging outcomes observed in this study may be attributed to the predominance of correctable pre-renal causes treated with conservative measures, prompt patient presentation, and improved management protocols.

LIMITATION OF THE STUDY

This study was conducted at a single center with a limited sample size. Furthermore, the study spanned a relatively short period. Therefore, the findings from this study may not fully represent the overall situation across the entire country.

CONCLUSION

Hypovolemia due to diarrhea, vomiting, or hemorrhage, along with NSAIDs, rhabdomyolysis, septicemia, and falciparum malaria, are common etiologies of acute kidney injury (AKI). Over eighty percent of AKI cases completely recover through one or more treatment procedures, including conservative treatment, hemodialysis, peritoneal dialysis, and continuous renal replacement therapy (CRRT). These therapeutic interventions play a crucial role in managing AKI, aiding in the restoration of kidney function and overall patient recovery. Early recognition and prompt treatment of the underlying causes are essential for improving outcomes and minimizing the risk of long-term kidney damage.

RECOMMENDATIONS

To mitigate morbidity and mortality from AKI, ensure adequate renal perfusion, avoid nephrotoxic drugs, and recognize risk factors like older age and sepsis. Review medications for nephrotoxicity and prioritize prevention through heightened awareness. Adopt team-based strategies for early detection and management. Future research should focus on systematic, kidney-specific protocols over singular treatments.

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