ORIGINAL RESEARCH

To study the clinical features and broad spectrum of etiologies in patients with acute renal injury in a specialized hospital

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ABSTRACT

Aim: To study the clinical features and broad spectrum of etiologies in patients with acute renal injury in a specialized hospital. **Material and methods:** This prospective observational study was conducted in the Department of Medicine, focusing on patients diagnosed with acute renal injury (ARI). The study aimed to examine the clinical characteristics and range of causes of ARI in a cohort of 100 patients.A total of 100 patients diagnosed with acute renal injury were included in the study. Acute renal injury was defined as an abrupt decrease in kidney function, characterized by a significant increase in serum creatinine levels (≥ 0.3 mg/dL within 48 hours or a 50% increase from baseline) and/or a decrease in urine output (oliguria: < 0.5 mL/kg/h for more than six hours). **Results:** A total of 75% of the patients experienced recovery of renal function, with the highest recovery rates observed in the prerenal group (28 out of 35), likely due to the reversible nature of conditions like dehydration or hypotension when promptly treated. However, 15% of the patients progressed to chronic kidney disease (CKD), with the intrarenal group showing the highest progression rate (8 out of 45), underscoring the long-term impact of intrinsic renal damage. The mortality rate in the study was 10%, with intrarenal causes being associated with the highest mortality (5 out of 45), reflecting the severity of conditions like acute tubular necrosis and the challenges in managing these patients. Postrenal causes had a slightly higher mortality rate (3 out of 20) compared to prerenal causes, likely due to complications related to delayed diagnosis or intervention in obstructive uropathy. **Conclusion:** This study highlights the significant impact of acute renal injury (ARI) on middle-aged and older adults, with a higher prevalence among males. The results reveal that intrarenal causes are the most common, followed by prerenal and postrenal causes. The elevated serum creatinine and BUN levels, particularly in the intrarenal group, underscore the severity of renal impairment associated with intrinsic renal damage. Comorbid conditions, such as hypertension and diabetes, were prevalent, further complicating the management of ARI.

Key Words: Acute kidney injury, KDIGO classification, Pre-Renal

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INTRODUCTION

Acute kidney injury (AKI) is a critical condition characterized by a sudden decline in kidney function, which can lead to severe morbidity and mortality if not promptly diagnosed and managed. The clinical profile and etiological spectrum of AKI can vary widely depending on the population studied, geographical location, and healthcare setting. Understanding these variations is crucial for developing effective prevention and treatment strategies. The clinical presentation of AKI can range from asymptomatic elevations in serum creatinine to life-threatening conditions requiring renal replacement therapy. Common symptoms of AKI include oliguria (reduced urine output), edema, fatigue, confusion, and in severe cases, uremic symptoms such as nausea, vomiting, and altered mental status. Laboratory findings typically reveal elevated serum creatinine and blood urea nitrogen (BUN) levels, and in some cases, abnormalities in electrolytes such as hyperkalemia and metabolic acidosis.[1,2] Risk factors for AKI are diverse and include pre-existing chronic kidney disease (CKD), advanced age, diabetes mellitus, hypertension, heart failure, liver disease, and exposure to nephrotoxic medications or contrast agents. Hospitalized patients, especially those in intensive care units (ICUs), are at particularly high risk due to the complexity of their medical conditions and the frequent use of invasive procedures and medications that can impair renal function. The etiology of AKI can be broadly categorized into pre-renal, intrinsic renal, and post-renal causes. Each category encompasses a variety of conditions that can impair renal function through

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different mechanisms. Pre-renal AKI is the most common form and is primarily due to decreased renal perfusion without intrinsic kidney damage. Causes include hypovolemia from dehydration or hemorrhage, decreased cardiac output from heart failure or myocardial infarction, and systemic vasodilation from sepsis or anaphylaxis. Prompt recognition and treatment of pre-renal causes are essential to prevent progression to intrinsic renal damage.[3]

Intrinsic renal AKI, also known as intra-renal AKI, results from direct damage to the kidney parenchyma. Common causes include acute tubular necrosis (ATN), which can occur due to ischemia or nephrotoxins such as aminoglycosides, radiocontrast agents, and nonsteroidal anti-inflammatory drugs (NSAIDs).

Glomerulonephritis, acute interstitial nephritis, and thrombotic microangiopathies are other intrinsic causes. The pathophysiology of ATN involves tubular cell injury and death, inflammation, and microvascular dysfunction. Post-renal AKI, or obstructive AKI, occurs due to obstruction of urine flow at any level from the renal pelvis to the urethra. Causes include benign prostatic hyperplasia, urolithiasis, tumors, and strictures. Prompt relief of the obstruction is critical to prevent permanent renal damage. The incidence of AKI varies widely across different settings. In the general population, AKI is relatively uncommon, but its incidence increases dramatically in hospitalized patients and those undergoing major surgery or critical illness. The prognosis of AKI depends on the underlying cause, severity, and timeliness of treatment. While many patients recover renal function, others may progress to chronic kidney disease (CKD) or end-stage renal disease (ESRD). Long-term outcomes include an increased risk of cardiovascular events and mortality.[4,5]

MATERIAL AND METHODS

This prospective observational study was conducted in the Department of Medicine, focusing on patients diagnosed with acute renal injury (ARI). The study aimed to examine the clinical characteristics and range of causes of ARI in a cohort of 100 patients.A total of 100 patients diagnosed with acute renal injury were included in the study. Acute renal injury was defined as an abrupt decrease in kidney function, characterized by a significant increase in serum creatinine levels (≥ 0.3 mg/dL within 48 hours or a 50% increase from baseline) and/or a decrease in urine output (oliguria: < 0.5 mL/kg/h for more than six hours).

Inclusion Criteria

- Patients aged 18 years or older.
- Patients with newly diagnosed acute renal injury based on the criteria mentioned above.
- Patients providing informed consent to participate in the study.

Exclusion Criteria

- Patients with chronic kidney disease (CKD) or end-stage renal disease (ESRD).
- Patients on dialysis prior to the onset of acute renal injury.
- Patients with pre-existing conditions that could confound the diagnosis of ARI (e.g., known obstructive uropathy, severe dehydration).
- Patients with incomplete medical records.

Methodology

Data collection involved obtaining detailed medical histories from each patient, including demographic data such as age and sex, past medical history, comorbidities like diabetes and hypertension, and any prior episodes of renal dysfunction. The cause of acute renal injury (ARI) was assessed through a combination of clinical evaluations, laboratory findings, imaging studies, and, when necessary, renal biopsy. The causes of ARI were categorized into three primary groups: prerenal, intrarenal, and postrenal. Prerenal causes included conditions leading to reduced renal perfusion, such as dehydration, shock, and heart failure. Intrarenal causes involved direct damage to the kidneys, including acute tubular necrosis, glomerulonephritis, acute interstitial nephritis, and vascular causes like renal artery thrombosis. Postrenal causes were associated with obstructive uropathy due to factors such as stones, tumors, or strictures.

All patients underwent a comprehensive clinical evaluation, which included a physical examination and assessment of symptoms such as oliguria, edema, hypertension, and signs of fluid overload. The severity of ARI was classified based on serum creatinine levels and urine output, following the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Laboratory investigations were conducted for all patients to assess renal function and identify potential causes of ARI. These tests included serum creatinine and blood urea nitrogen (BUN) levels, electrolytes (sodium, potassium, calcium, bicarbonate), urinalysis (including urine sediment examination, proteinuria, and hematuria), complete blood count (CBC) to detect anemia and signs of infection, liver function tests (LFTs) to rule out hepatic causes, and a coagulation profile, including prothrombin time (PT) and activated partial thromboplastin time (aPTT), to assess coagulation status.

Imaging studies were also an integral part of the diagnostic process. Ultrasound imaging of the kidneys was performed to evaluate renal size, parenchymal echogenicity, and the presence of any obstructive pathology. In selected cases, more advanced imaging techniques such as contrast-enhanced computed tomography (CT) or magnetic resonance imaging (MRI) were utilized to further investigate the

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underlying cause of ARI. Treatment was tailored to the specific cause of ARI identified in each patient. Interventions included fluid management, correction of electrolyte imbalances, and, in some cases, renal replacement therapy (RRT) such as hemodialysis or peritoneal dialysis. Patients were closely monitored for clinical outcomes, including recovery of renal function, progression to chronic kidney disease, or mortality. Follow-up assessments were conducted at regular intervals—weekly for the first month, then monthly for three months—to evaluate renal function and overall health status.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Descriptive statistics, including mean, standard deviation, and frequency distributions, were used to summarize the data. Comparisons between different groups (e.g., prerenal, intrarenal, postrenal causes) were made using chi-square tests for categorical variables and t-tests or ANOVA for continuous variables. A p-value of <0.05 was considered statistically significant.

RESULTS

Table 1: Demographic Characteristics of Patients with Acute Renal Injury (ARI)

The demographic characteristics of the 100 patients diagnosed with acute renal injury (ARI) are detailed in Table 1. The average age of the patients was 58.3 ± 12.1 years, indicating that ARI predominantly affects middle-aged and older adults. When comparing the age distribution across the different etiologies, patients with intrarenal causes had a slightly higher mean age (59.5 ± 13.2 years) compared to those with prerenal (56.7 ± 11.4 years) and postrenal causes (58.6 ± 10.9 years). The male-to-female ratio in the study was approximately 3:2, with 61 males and 39 females, showing a higher prevalence of ARI among males. This trend was consistent across all subgroups, with males representing the majority in prerenal (22 out of 35), intrarenal (28 out of 45), and postrenal (11 out of 20) causes.

Table 2: Comorbidities in Patients with Acute Renal Injury (ARI)

Table 2 presents the comorbidities observed in the study population. Hypertension was the most common comorbidity, affecting 75% of the patients, with a slightly higher prevalence among those with intrarenal causes (35 out of 45). Diabetes mellitus was present in 54% of the patients, again most prevalent in the intrarenal group (25 out of 45). Heart failure was noted in 23% of the patients, predominantly among those with prerenal causes (15 out of 35), which is expected as heart failure can lead to reduced renal perfusion. A history of ARI was recorded in 18% of the patients, with a near-equal distribution across the three etiological groups. These comorbidities highlight the complex interplay between ARI and chronic conditions, particularly cardiovascular diseases and diabetes, which are known risk factors for kidney injury.

Table 3: Causes of Acute Renal Injury (ARI)

The causes of ARI were categorized into prerenal, intrarenal, and postrenal origins, as shown in Table 3. Intrarenal causes were the most common, accounting for 45% of the cases. This group includes conditions such as acute tubular necrosis and glomerulonephritis, which directly damage the kidney parenchyma. Prerenal causes, which involve factors leading to decreased renal perfusion like dehydration and shock, were responsible for 35% of the cases. Postrenal causes, related to obstructive uropathy, were the least common, accounting for 20% of the cases. This distribution reflects the typical etiological pattern of ARI in clinical practice, where intrinsic renal damage often predominates.

Table 4: Laboratory Findings in Patients with Acute Renal Injury (ARI)

Table 4 outlines the laboratory findings among the patients. The mean serum creatinine level was elevated across all groups, with the highest levels observed in the intrarenal group (4.2 ± 1.6 mg/dL), reflecting more severe renal impairment. Blood urea nitrogen (BUN) levels were also elevated, particularly in the intrarenal group (46.8 ± 13.5 mg/dL), which is consistent with impaired renal function leading to reduced clearance of nitrogenous waste. Electrolyte imbalances were observed, with sodium levels slightly below the normal range (133.4 ± 4.9 mmol/L), particularly in the intrarenal group. Potassium levels were elevated (4.9 ± 0.8 mmol/L), with the highest values in the intrarenal group (5.1 ± 0.9 mmol/L), indicating a risk of hyperkalemia, a common and dangerous complication in ARI.

Table 5: Clinical Outcomes in Patients with Acute Renal Injury (ARI)

The clinical outcomes of the study population are summarized in Table 5. A total of 75% of the patients experienced recovery of renal function, with the highest recovery rates observed in the prerenal group (28 out of 35), likely due to the reversible nature of conditions like dehydration or hypotension when promptly treated. However, 15% of the patients progressed to chronic kidney disease (CKD), with the intrarenal group showing the highest progression rate (8 out of 45), underscoring the long-term impact of intrinsic renal damage. The mortality rate in the study was 10%, with intrarenal causes being associated with the highest mortality (5 out of 45), reflecting the severity of conditions like acute tubular necrosis and the challenges in managing these patients. Postrenal causes had a slightly higher mortality rate (3 out of 20) compared to prerenal causes, likely due to complications related to delayed diagnosis or intervention in obstructive uropathy.

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Table 1: Demographic Characteristics of Patients with Acute Renal Injury (ARI)

Characteristic	Total (n = 100)	Prerenal Causes (n = 35)	Intrarenal Causes (n = 45)	Postrenal Causes (n = 20)
Age (years)	58.3 ± 12.1	56.7 ± 11.4	59.5 ± 13.2	58.6 ± 10.9
Sex (Male)	61	22	28	11
Sex (Female)	39	13	17	9

Table 2: Comorbidities in Patients with Acute Renal Injury (ARI)

Comorbidity	Total (n = 100)	Prerenal Causes (n = 35)	Intrarenal Causes (n = 45)	Postrenal Causes (n = 20)
Hypertension	75	25	35	15
Diabetes Mellitus	54	20	25	9
Heart Failure	23	15	5	3
History of ARI	18	7	8	3

Table 3: Causes of Acute Renal Injury (ARI)

Cause	Number of Patients (n = 100)	Percentage (%)
Prerenal Causes	35	35%
Intrarenal Causes	45	45%
Postrenal Causes	20	20%

Table 4: Laboratory Findings in Patients with Acute Renal Injury (ARI)

Laboratory Test	Normal Range	Total (n = 100)	Prerenal Causes (n = 35)	Intrarenal Causes (n = 45)	Postrenal Causes (n = 20)
Serum Creatinine (mg/dL)	0.6-1.2	3.6 ± 1.4	3.0 ± 1.2	4.2 ± 1.6	3.5 ± 1.3
BUN (mg/dL)	7-20	42.7 ± 12.9	39.5 ± 10.8	46.8 ± 13.5	41.2 ± 12.3
Sodium (mmol/L)	135-145	133.4 ± 4.9	134.2 ± 5.1	132.8 ± 4.7	133.9 ± 4.6
Potassium (mmol/L)	3.5-5.1	4.9 ± 0.8	4.6 ± 0.7	5.1 ± 0.9	4.8 ± 0.8

Table 5: Clinical Outcomes in Patients with Acute Renal Injury (ARI)

Outcome	Total (n = 100)	Prerenal Causes (n = 35)	Intrarenal Causes (n = 45)	Postrenal Causes (n = 20)
Recovery of Renal Function	75	28	32	15
Progression to Chronic Kidney Disease (CKD)	15	5	8	2
Mortality	10	2	5	3

DISCUSSION

The demographic characteristics of patients with acute renal injury (ARI) in this study reflect findings from earlier research. The average age of 58.3 ± 12.1 years is consistent with other studies, which have shown that ARI is more prevalent in older populations due to the higher incidence of comorbidities and decreased renal reserve with aging. For example, a study by Nash et al. (2002) reported that the mean age of patients with ARI was approximately 60 years, which aligns with our findings.[1] The male predominance (61%) observed in our study is also consistent with previous research. Liaño et al. (1998) found that males are more likely to develop ARI, possibly due to a higher prevalence of cardiovascular diseases and greater exposure to nephrotoxic agents among men.[2]In our study, hypertension was the most common comorbidity (75%), followed by diabetes mellitus (54%), heart failure (23%), and a history of ARI (18%). This distribution mirrors the findings of other studies that have identified hypertension and diabetes as significant risk factors for ARI. For instance, in a study by Coresh et al. (2007), hypertension was present in over 70% of patients with ARI, and diabetes was a comorbidity in more than 40% of cases.[3] The prevalence of heart failure in our study is also comparable to the findings by Coca et al. (2008), who reported heart failure in approximately 25% of ARI patients.[4] The presence of these comorbidities underscores the multifactorial nature of ARI and the need for comprehensive management strategies that address underlying conditions.The distribution of ARI causes in our study, with 45% intrarenal, 35% prerenal, and 20% postrenal, aligns with previous research. Intrarenal causes, particularly acute tubular necrosis, are often the most common, as noted by Hou et al. (1983), who reported intrarenal causes in 40-50% of ARI cases.[5] Prerenal causes, often related to decreased renal perfusion, are also a significant contributor, especially in critically ill patients, as described by Brivet et al. (1996).[6] The lower incidence of postrenal causes in our study (20%) is

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consistent with the findings of Waikar et al. (2008), who found obstructive uropathy to be a less common cause of ARI, particularly in younger populations.[7]

The laboratory findings in our study, including elevated serum creatinine and BUN levels, reflect the severity of renal impairment across the different ARI etiologies. The mean serum creatinine level was highest in the intrarenal group (4.2 ± 1.6 mg/dL), which is consistent with the findings of Shlipak et al. (2004), who reported similar levels in patients with acute tubular necrosis.[8] The elevated potassium levels observed, particularly in the intrarenal group (5.1 ± 0.9 mmol/L), indicate a risk of hyperkalemia, a known complication of ARI, as highlighted by Burnett et al. (1996).[9] The slight hyponatremia noted in all groups is also a common finding in ARI and reflects impaired renal sodium handling, as discussed by Zager et al. (1998).[10]The clinical outcomes in our study show that 75% of patients experienced recovery of renal function, with the highest recovery rates in the prerenal group (80%). This is in line with the reversible nature of prerenal ARI when the underlying cause is promptly addressed, as noted by Perazella (2003).[11] The progression to chronic kidney disease (CKD) was highest in the intrarenal group (17.8%), which is consistent with findings by Kellum et al. (2002), who reported a higher risk of CKD following intrinsic renal injury.[12] The mortality rate of 10% in our study, particularly the higher mortality in the intrarenal group, reflects the findings of Schiffl et al. (2006), who noted increased mortality in patients with acute tubular necrosis due to its severity and complications.[13]

CONCLUSION

This study highlights the significant impact of acute renal injury (ARI) on middle-aged and older adults, with a higher prevalence among males. The results reveal that intrarenal causes are the most common, followed by prerenal and postrenal causes. The elevated serum creatinine and BUN levels, particularly in the intrarenal group, underscore the severity of renal impairment associated with intrinsic renal damage. Comorbid conditions, such as hypertension and diabetes, were prevalent, further complicating the management of ARI. Notably, 75% of patients experienced renal recovery, with the highest recovery rates in the prerenal group, while the intrarenal group showed the highest progression to chronic kidney disease and mortality rates. These findings emphasize the need for early intervention and targeted management strategies to improve outcomes for ARI patients.

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