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**Dr. Divya Reddy Molugu**
General Medicine, Gayatri Hospital, Hasthinapuram, Hyderabad, Telangana, India

**Dr. B Sreenivas Reddy**
General Medicine, Gayatri Hospital, Hasthinapuram, Hyderabad, Telangana, India

**Dr. Greeshma Reddy Molugu**
MBBS, Hyderabad, Telangana, India

**Corresponding Author:**
**Dr. Divya Reddy Molugu**
General Medicine, Gayatri Hospital, Hasthinapuram, Hyderabad, Telangana, India

# Clinical profile of acute kidney injury in ICU patients

**Dr. Divya Reddy Molugu, Dr. B Sreenivas Reddy and Dr. Greeshma Reddy Molugu**

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**Abstract**
**Background:** Deterioration of renal function that is both acute and potentially reversible causes the kidney to be ineffective to eliminate nitrogenous waste products and maintain fluid and electrolyte balance, resulting in Acute Kidney Injury.
**Objective:** To study the Clinical profile of acute kidney injury in ICU patients
**Methods:** A total of 50 patients with acute kidney injury were included after meeting the inclusion exclusion criteria. After obtaining of written informed permission from the patients, a comprehensive medical history, general physical examination, systemic examination, and routine and specific lab investigations were performed to determine the underlying etiology, clinical characteristics, and result of AKI. All patients were clinically and biochemically monitored until they were discharged.
**Results:** Male were predominant with 70% compared to females 30%.The Male: Female ratio was 2.3:1, Fever was the most common symptom observed in 54% of the cases followed by dyspnea. Around 30% of the urine cultures were positive. Escherichia coli, candida albicans, Klebsiella species, proteus mirabilis, and pseudomonas species were among the microorganisms discovered in urine culture. Patients with three or less ailments (82%) have a better probability of recovering, whereas those with four or more ailments (18%) have a larger risk of deteriorating. 78% of the patients had recovered from AKI, whereas AKI deteriorated in 22% of the patients.
**Conclusion:** Acute Kidney Injury is a prevalent clinical issue in critically ill patients, particularly those in the medical ICU. Early identification and appropriate management are crucial for reducing AKI-related and all-cause death in critically ill patients.

**Keywords:** Acute kidney injury, ICU, modus, dialysis**,** creatinine

## Introduction

Acute kidney injury (AKI), formerly known as acute renal failure, is defined by a sudden acute renal failure, which results in the retention of nitrogenous and other waste products typically eliminated by the kidneys. AKI complicates 5–7% of acute care hospital admissions and up to 30% of intensive care unit hospitalizations [1]. It is a significant medical problem in the developing world, especially in the context of diarrheal disorders, infectious diseases such as malaria and leptospirosis, snake bites, and natural catastrophes [2]. AKI can exacerbate a variety of illnesses. It is split into three groups for the purposes of diagnosis and management. There are three types of AKI: prerenal, renal, and postrenal, with different causes for each [3].

AKI can range in severity from asymptomatic and temporary alterations in glomerular filtration rate (GFR) laboratory measurements to overwhelming and swiftly deadly derangements in efficient circulation volume regulation and electrolytes, acid-base composition of the plasma [4]. Nausea, vomiting, tiredness, oliguria/anuria, fluid overload, metabolic acidosis, hyperkalemia, and impaired mental state are all typical clinical symptoms. The primary goal of treating individuals with acute kidney damage is to reverse the underlying cause and restore fluid and electrolyte imbalances. Dialysis is used as an adjunct to supportive care.

AKI is associated with a significantly higher risk of death in hospitalized patients, particularly those admitted to the intensive care unit (ICU), where in-hospital mortality rates can reach 50% [5]. Morbidity is determined by the severity of the damage and the underlying illness.

Other factors influencing patient survival in acute kidney injury include age, the number and severity of coexisting illnesses and associated complications such as intravascular overload, hyperkalemia, and other metabolic complications, as well as systemic life threatening complications such as cardiac arrhythmia, myocardial infarction, pulmonary embolism, gastrointestinal ulcers, seizures, coma, hemolysis, and kidney failure [6].

## Materials and Methods

**Study Design:** This is a random cross sectional observational study included

**Sample size:** 50 patients with acute kidney injury

**Inclusion Criteria**

- Clinically and/or biochemically confirmed acute kidney injury patients.

**Exclusion Criteria**

- Patients with pre-existing chronic renal failure or chronic renal disease.
- Patients aged below 14 years

After obtaining of written informed permission from the patients, a comprehensive medical history, general physical examination, systemic examination, and routine and specific lab investigations were performed to determine the underlying etiology, clinical characteristics, and result of AKI. All patients were clinically and biochemically monitored until they were discharged.

**Ethical Clearance:** Ethical clearance was obtained from the institutional committee prior to the commencement of the study.

**Statistical Methods:** Statistical tables and charts were used to illustrate the statistical data. For statistical analysis SPSS software version 22 was used.

## Observation and Results

**Table 1:** Distribution based on age and gender

| Gender | Number (%) |
|---|---|
| Male | 35(70%) |
| Female | 15(30%) |
| **Age Group** | |
| 18-30 | 10(20%) |
| 31-40 | 6(12%) |
| 41-50 | 7(14%) |
| 51-60 | 12(24%) |
| 61-70 | 15(30%) |
| **Total** | **50(100%)** |

Male were predominant with 70% compared to females 30%.

**The Male:** Female ratio was 2.3:1

Majority of the patients belonged to the older age group with 30% of the cases in 61 to 70 years followed by 51 to 60 years with 24% of the cases and 20% of the cases in the 18 to 30 yrs age group.

**Table 2:** Distribution based on the symptoms

| Symptoms | Number (%) |
|---|---|
| Fever | 27(54%) |
| Dyspnea | 18(36%) |
| Oliguria | 14(28%) |
| Dysuria | 13(26%) |
| Vomiting | 10(20%) |
| Nausea | 9(18%) |
| Anorexia | 9(18%) |
| Diarrhea | 8(16%) |

Fever was the most common symptom observed in 54% of the cases followed by dyspnea in 36% of the cases, oliguria in 28% of the cases, Dysuria in 26% of the cases. Vomiting in 20% of the cases, Nausea and anorexia in 18% of the cases each and diarrhea in 16% of the cases.

**Table 3:** Distribution based on the various parameters

| 3 | Total |
|---|---|
| <10 | 23(46%) |
| ≥ 10 | 27(54%) |
| **TLC (cells/ cu.mm)** | |
| <4000 | 0(0%) |
| 4000 - 10000 | 14(28%) |
| >10000 | 36(72%) |
| **Creatinine (mg/dl)** | |
| <2 | 20(40%) |
| 2-3.5 | 16(32%) |
| >3.5 | 14(28%) |
| **Na (mmol/L)** | |
| <135 | 21(42%) |
| 135 to 145 | 25(50%) |
| >145 | 4(8%) |
| **K (mmol/L)** | |
| <3.5 | 9(18%) |
| 3.5 to 5.5 | 34(68%) |
| >5.5 | 7(14%) |
| **Ultrasonography** | |
| Positive finding | 28(56%) |
| Negative finding | 22(44%) |
| **Microbiology** | |
| Positive | 15(30%) |
| Negative | 35(70%) |
| Total | 50(100%) |

- Among the 50 patients studied, 46% had hemoglobin less than 10 gm/dl and 54% had hemoglobin greater than 10 gm/dl.
- TLC was between 4000-10000 in 28% of the patients, while TLC was higher than 10000 in 72% of the patients.
- 40% of the patients had creatinine levels less than 2, 32% had levels between 2-3.5, and 28% had creatine levels of more than 3.5.
- Serum sodium levels were less than 135 in 42% of patients, 135 to 145 in 50% of patients, and greater than 145 in 8% of patients.
- Serum potassium levels were less than 3.5 in 18% of patients, 3.5 to 5.5 in 68% of patients, and greater than 5.5 in 14% of patients.
- Around 30% of the urine cultures were positive. Escherichia coli, candida albicans, Klebsiella species, proteus mirabilis, and pseudomonas species were among the microorganisms discovered in urine culture.

- Around 56% of patients exhibited positive ultra-Sonographic findings such as hydronephrosis, renal or bladder calculi, hydroureter, pyelonephritis, stricture urethra and increased renal echotexture, whereas 44% had normal ultrasonography.
- MODUS Score stands for M = MODS, Malignancy; O = Obstructive Uropathy; D =Drug Induced; U = Urosepsis S =Sepsis, Shock.

Patients with three or less ailments (82%) have a better probability of recovering, whereas those with four or more ailments (18%) have a larger risk of deteriorating.

**Table 4:** Distribution based on underlying cause

| Underlying cause | Total |
|---|---|
| Sepsis | 32(64%) |
| Urosepsis | 20(40%) |
| Shock | 16(32%) |
| MODS | 15(30%) |
| Drug Induced | 8(16%) |
| Malignancy | 2(4%) |
| Obstructive Uropathy | 4(8%) |

Sepsis was diagnosed in 32%, Urosepsis in 40%, Shock in 32%, MODS in 30%, Drug Induced in 16%, Obstructive uropathy in 8% and Malignancy in 4% of the cases.

**Table 5:** Distribution based on MODUS score

| MODUS Score | Total |
|---|---|
| 1 | 14(28%) |
| 2 | 18(36%) |
| 3 | 9(18%) |
| 4 | 3(6%) |
| 5 | 6(12%) |
| Total | 50 |

**Table 6:** Distribution based on outcomes

| Outcomes | Number (%) |
|---|---|
| Recovered | 39(78%) |
| Worsened | 11(22%) |
| Total | 50(100%) |

Out of the 50 AKI patients, 78% of the patients recovered, whereas AKI deteriorated in 22% of the patients.

## Discussion

Acute Kidney Injury is a prevalent presentation in hospitalized patients, particularly those admitted to the intensive care unit (ICU) with a critical illness. With recent modifications in definition and advancements in diagnostic and therapeutic techniques, it is important to investigate the prevalence and clinical profile of AKI patients in various groups. The availability of appropriate and available epidemiological data aids in the efficient utilization of health-care resources. AKI's etiological range and pattern are extremely diverse. Acute Kidney Injury is a prevalent problem in critically ill patients, and it also puts a financial strain on health-care resources, which is especially true in resource-constrained countries like India. The key to preventing mortality and long-term morbidity is early diagnosis and swift treatment.

In sepsis patients, inflammatory mediators produced from bacteria or immune cells bypass the glomerulus and reach the tubular region, where they can cause damage to tubular cells by attaching to their receptors. Furthermore, extravasated leukocytes produce inflammatory mediators that might activate tubular cells from the interstitial side. Apoptosis or cell cycle arrest can be induced by the activation of cytokines or DAMP/PAMP (damage associated molecular pattern/pathogen associated molecular pattern) receptors [7, 8]. Endothelial malfunction, inflammation, coagulation disruption, and adaptive cell responses to damage are among the processes studied. As a result, a bio-energetic stress of the tubular epithelial cells in response to the increased inflammatory signal generated by peritubular microvascular dysfunction is a critical event in the early kidney failure during sepsis. Sepsis is characterized by a reduction in peripheral vascular resistance, a misdistribution of tissue blood flow, and a disruption of microcirculatory perfusion.

When renal perfusion is compromised for whatever cause, the kidneys strive to maintain glomerular capillary pressure by afferent arteriolar vasodilation and efferent arteriolar vasoconstriction, a process known as auto-regulation [9]. So, in addition to causes of renal hypoperfusion, anything that affects renal auto-regulation will produce a decrease in GFR. Hypo-osmolality is indicated by a low serum sodium level. As water flows easily between the intracellular and extracellular compartments, hypo-osmolality always implies excess total body water compared to body solutes or excess water relative to solute in the extracellular fluid [10]. Solute depletion, dilution, or a combination of the two can cause this imbalance. Under normal circumstances, renal water handling is adequate to excrete up to 15-20 liters of free water each day [11]. When osmolality is decreased, renal excretion of free water is impaired, leading in hyponatremia and AKI [12].

A very significant p value of 0.000 was observed in patients with serum creatinine level of 5 mg/dl when compared to patients with serum creatinine >5 mg/dl, indicating that patients with serum creatinine >5 mg/dl had a greater risk of deteriorating than those patients with serum creatinine level of 5 mg/dl. Turney *et al*. reported that AKI was linked to septic shock in 47.5 % of patients. Major surgery was linked to 34% of AKI, cardiogenic shock was linked to 27% of ARF, hypovolemia was linked to 26% of ARF, and drug-related ARF was linked to 19% of ARF [13]. In a research by J Prakash *et al*., Hypotension (72%), sepsis (70%), and nephrotoxic medicines (67%) were the leading causes [14]. In a study by R. Bhadade *et al*., Cardiogenic (72%), sepsis (69%), nephrotoxic medicines (67%), hypovolemia (59%), tropical fever (40%), and postoperative complications (33%). were among the reasons identified [15].

Patients with a MODUS score of 3 or less have a better chance of recovering, whereas those with a score of 4 or more have a higher risk of AKI deteriorating. Patients with a high MODUS score for AKI and an older age group had a higher risk of deteriorating, whereas patients with a lower MODUS score for AKI and a younger age group had a higher probability of recovery.

## Conclusion

Acute Kidney Injury is a prevalent clinical issue in critically ill patients, particularly those in the medical ICU. Patients with a MODUS score of 3 or less have a better chance of recovering, whereas those with a score of 4 or more have a higher risk of AKI deteriorating Early identification and appropriate management are crucial for reducing AKI-

related and all-cause death in critically ill patients.

**References**


1. Brenner & Rector's The Kidney, 7th Ed. B.M. Brenner (ed) Philadelphia Saundes, 2004.
2. Harrison's Principles of Internal Medicine, 19th Ed.
3. Fernando Liano, Julio Pascual and The Madrid Acute Renal Failure Study group 1, epidemiology of acute renal failure: A prospective, multi centre, community based study. Kidney International 1996, 811-818.
4. Tariq Ali. Incidence and Outcomes in Acute Kidney Injury: A comprehensive population based study. JASN IMPACT FACTOR 7.371.2006
5. Eric Hoste AJ, Norbert H. Lameire, Raymond C. Acute renal failure in patients with sepsis in ICU: Predictive Factors, Incidence, Comorbidity and outcome Jasn Impact Factor 2006, 7.371
6. Jean-Pierre Grunfield, Dominique Ganeval, Francois Bournerias. Acute renal failure I pregnancy. Kidney International 1980;18:179-191.
7. Jose Ramon Perez Valdivieso 1 Prognosis and serum creatinine levels in acute renal failure. BMC Nephrology 2007;8:14.
8. Shigehiko Uchino, John A Kellum. Acute Renal Failure in critically ill patient S: A multinational, multi centre study. JAMA 2005;294:813-818.
9. Neil Shusterman, Brai Storm I, Thomas Murray G *et al*. Rish Factors and outcome of hospital acquired renal failure. AM. J. Med 1987, 83.
10. Mahakur AC *et al*. pattern of acute renal failure in Orissa. JAPI 1978, 26.
11. Ciaran C Doherty *et al.* 10.1 Epidemiology of acute renal failure. Oxford Textbook of Nephrology 3rd Edition.
12. Nolan CR, Anderson RJ. Hospital acquired acute renal failure. J Am Soc Nephrol 1998;9;710-8.
13. Turney, Journal of The Association of Physicians of India 2016, 64.
14. Prakash J, Murthy AS, Vohra R, Rajak M, Mathur SK. Acute Renal Failure in ICU. JAPI 2006, 4.
15. Bhadade R. Journal of the association of physicians of India 2006, 54.