Section: General Medicine

Original Research Article

A PROSPECTIVE OBSERVATIONAL STUDY - AETIOLOGY, CLINICAL PROFILE AND DETERMINANTS OF SHORT-TERM OUTCOME OF ACUTE KIDNEY INJURY IN PATIENTS ADMITTED TO MEDICAL INTENSIVE CARE UNIT AT SOUTHERN RAILWAY HEADQUARTERS HOSPITAL, PERAMBUR

G Arunkumar[1], T S Sreedev[2], K Muruganandam[1]

[1]Consultant Physician, Department of General Medicine, Southern Railway Headquarters Hospital, Ayanavaram, Chennai, Tamil Nadu, India.
[2]DNB Resident, Department of General Medicine, Southern Railway Headquarters Hospital, Ayanavaram, Chennai, Tamil Nadu, India.

Received : 04/08/2025
Received in revised form : 21/09/2025
Accepted : 06/10/2025

Keywords:
Acute Kidney Injury (AKI), Sepsis, mortality, urine output, Coronary Artery disease (CAD), Cerebrovascular Accident (CVA).

Corresponding Author:
Dr. K Muruganandam,
Email: mkmuruganandam@gmail.com

DOI: 10.47009/jamp.2025.7.5.209

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (5); 1107-1113

ABSTRACT

Background: Acute Kidney Injury (AKI) is a common clinical condition associated with significant morbidity and mortality. Identifying predictors of adverse outcomes is essential to improve early recognition, management, and prognosis. The present study aims to assess the aetiology, clinical profile, and determinants of the short-term outcome of acute kidney injury in patients admitted to the intensive care unit. **Materials and Methods:** This was a prospective observational study conducted on 155 patients diagnosed with AKI admitted to our tertiary care center. Detailed demographic data, clinical presentation, underlying comorbidities, laboratory parameters, and treatment interventions were recorded. Patients were followed throughout their hospital stay, and outcomes were categorized as AKI resolved, AKI not resolved, or death. Statistical analysis was performed to identify predictors of mortality. **Result:** Of 155 patients, 135 (87.1%) survived, including 11 (7.1%) with unresolved AKI, while 20 (12.9%) patients died. All the patients who died were aged >65 years, establishing advanced age as a significant predictor of mortality ($p = 0.041$). Sepsis was strongly associated with adverse outcomes ($p = 0.009$), whereas other etiologies, such as acute gastroenteritis and LV dysfunction, were not statistically significant. In terms of clinical presentation, decreased urine output ($p = 0.008$), altered sensorium ($p < 0.001$), and reduced food intake ($p < 0.05$) were significantly related to poor outcomes, highlighting their role as early warning indicators of severity. Regarding comorbidities, coronary artery disease (CAD) and cerebrovascular accident (CVA) showed a statistically significant association with mortality ($p < 0.05$). **Conclusion:** This study demonstrates that advanced age (>65 years), sepsis, CAD, CVA, and critical presenting symptoms (decreased urine output, altered sensorium, and reduced food intake) are significant predictors of mortality in AKI patients. The findings underscore the importance of early recognition of high-risk groups to initiate timely and aggressive management strategies.

INTRODUCTION

Acute kidney injury (AKI) is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and impairment (loss of function).[1] It is a syndrome that has a mixed aetiology where the presence of sepsis, ischaemia, and nephrotoxicity often co-exist and complicate recognition and treatment.[2] AKI is a syndrome that rarely has a sole and distinct pathophysiology, and the kidney plays an active role in the progression of multi-organ dysfunction rather than a single entity. Accurate and prompt diagnosis of AKI and investigating the pathophysiology of the various clinical phenotypes are very important for effective therapeutic interventions.[3] Traditionally, importance was given to the most severe acute reduction in kidney function, as indicated by severe azotaemia and often by oliguria or anuria.[4] But recent studies suggest that even relatively mild injury or impairment of kidney function, manifested by small changes in serum creatinine (sCr) and/or urine

ISSN (O): 2687-5365; ISSN (P): 2753-6556

output (UO), is a predictor of serious clinical consequences.

Acute kidney injury (AKI) is a clinical syndrome with diverse etiologies, many of which are largely preventable. It is potentially reversible if recognized and managed early; however, delayed or inadequate treatment often leads to serious complications, including high morbidity and the risk of permanent loss of kidney function.[5] All stages of AKI are associated with significantly increased short- and long-term adverse outcomes, with varied mortality rates.[2] Nevertheless, timely recognition and appropriate intervention may result in partial or even complete reversal of renal damage.

The underlying pathophysiology of AKI commonly involves an imbalance between oxygen and nutrient delivery to the nephrons due to impaired microcirculation, compounded by increased cellular energy demands triggered by stress responses.[2] Based on etiology, AKI is classified into three categories: pre-renal, when caused by factors affecting renal perfusion; intrinsic, when resulting from direct injury to renal tissues; and post-renal, when attributable to obstructive processes impairing urine outflow.[2]

Significant disparities exist in the incidence and causes of AKI between developing and developed countries. In developing regions, hospital-acquired factors such as renal ischemia, sepsis, and nephrotoxic drugs are predominant in urban centers, whereas community-acquired causes, including diarrheal illness, dehydration, and infectious diseases, are more frequent in rural populations.[6] Under-reporting in these settings further complicates assessment of the true global burden. In developed countries, the prevalence of AKI is rising, particularly among hospitalized and critically ill patients, where rates may reach as high as 60%.[2] Community-acquired AKI is less common but remains clinically relevant, with an estimated incidence of 4.3% among hospital admissions; however, this figure likely underestimates the true burden due to under-referral.[2] Against this background, the present study was conducted in a tertiary care hospital to evaluate the etiology, clinical profile, and short-term prognosis of patients with AKI.

MATERIALS AND METHODS

A prospective, hospital-based, observational study was conducted on all cases of AKI admitted to the department of general medicine in Southern Railway Headquarters hospital, Chennai, located in South India, from February 2023 to May 2024.

Inclusion criteria

All patients, aged above 18 years, with the diagnosis of AKI as per the AKIN criteria[4] suggested by Kidney Disease Improving Global Outcomes Clinical Practice Guideline for AKI were selected as follows: 1. Patients with an increase in serum creatinine by ≥0.3 mg/dl within 48 h; 2. Increase in serum creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or 3. Urine volume ≤0.5 ml/kg/h for 6 h.

Exclusion criteria

Patients hospitalized for less than 48 hours, and patients with known ESRD or who were on dialysis for AKI at the time of enrolment.

A detailed history and thorough clinical examination were performed for all selected patients. Laboratory investigations included complete blood counts, urine analysis, blood urea (urease method), serum creatinine (enzymatic method), blood glucose, liver function tests, chest X-ray, and abdominal ultrasonography performed by a qualified radiologist. All findings were documented in a structured proforma. For blood urea and serum creatinine, samples were collected at admission, at 24 and 48 hours, and subsequently repeated daily for seven days. Urine output was monitored throughout this period for every patient. Sepsis was diagnosed according to the International Sepsis Definitions Conference (2023) criteria. Patients requiring renal replacement therapy underwent intermittent hemodialysis. Ethical clearance was obtained from the Institutional Ethical Committee, and written informed consent was obtained from all study participants.

Statistical analysis: Descriptive analysis was carried out by frequency and proportion for categorical variables. Continuous variables were presented as Mean ± SD or Median (IQR). The chi-square/Fisher's exact test was used to test the statistical significance of cross-tabulation between categorical variables. P values <0.05 were considered statistically significant. RStudio desktop latest version 2023.03.0+ 386 was used for statistical analysis.

RESULTS

A total of 155 patients were included in the study. The majority of patients (73.5%, n = 114) were aged >65 years. Patients aged 55–64 years comprised 21.3% (n = 33), while 3.9% (n = 6) were in the 45–54 years age group, and 1.3% (n = 2) were in the 35–44 years age group, as shown in [Figure 1]. Males accounted for 57.4% (n = 89) and females for 42.6% (n = 66) as illustrated in [Figure 2]. The most common comorbidity observed was diabetes mellitus, present in 79.4% (n = 123) of patients, followed by hypertension in 76.1% (n = 118). Coronary artery disease was seen in 30.3% (n = 47), while chronic obstructive pulmonary disease/bronchial asthma was reported in 7.7% (n = 12), as detailed in [Table 1]. Cerebrovascular accident was noted in 4.5% (n = 7), and 8.4% (n = 13) had other comorbidities such as decompensated chronic liver disease or rheumatoid arthritis. Only 1.3% (n = 2) of patients had no comorbidities.

The most common presenting symptoms were vomiting (27.7%, n = 43), altered sensorium (25.8%,

ISSN (O): 2687-5365; ISSN (P): 2753-6556

n = 40), and fever (25.2%, n = 39) as detailed in Table 2. Breathlessness was reported in 18.7% (n = 29), cough with expectoration in 13.5% (n = 21), and urinary symptoms such as dysuria or increased frequency of micturition in 11.6% (n = 18). Decreased urine output was observed in 9.0% (n = 14), abdominal pain and loose stools each in 5.8% (n = 9). Chest pain was reported by 2.6% (n = 4), while other symptoms accounted for 8.4% (n = 13).

The most common etiology identified was sepsis, accounting for 63.9% (n = 99) of cases. Left ventricular dysfunction was observed in 14.2% (n = 22), while acute gastroenteritis contributed to 11.6% (n = 18). Drug-induced causes and diabetic ketoacidosis (DKA) were each reported in 3.2% (n = 5). Hepatorenal syndrome (HRS) was present in 2.6% (n = 4), obstructive uropathy in 1.9% (n = 3), and both accelerated hypertension and type 2 respiratory failure in 1.3% (n = 2) each, as summarised in [Table 3].

Baseline and 48-Hour Laboratory Parameters are detailed in [Table 4]. At admission, the median hemoglobin was 10 g/dL (IQR 9–12), indicating mild anemia in most patients. The median total count was 15,000/μL (IQR 9,600–18,000), reflecting leukocytosis consistent with underlying infection/sepsis. Baseline renal parameters were relatively preserved, with a median urea of 32 mg/dL (IQR 27–38) and creatinine of 1.0 mg/dL (IQR 0.9–1.1). However, at 48 hours, there was a marked rise in renal indices: urea increased to a median of 75 mg/dL (IQR 52.5–92) and creatinine to 2.0 mg/dL (IQR 1.85–2.3), suggestive of acute kidney injury progression during hospital stay. Urine analysis showed pyuria in 9.77% of patients, while proteinuria was present in 21.9%. No urinary abnormalities were seen in 68.4% of patients, as detailed in [Table 5].

In our study, dialysis was needed in 3.2% of patients, as shown in [Figure 4], and the majority of patients (80%) experienced resolution of acute kidney injury (AKI), indicating good recovery with treatment. However, a small proportion had persistent AKI (7.1%) or succumbed to their illness (12.9%), highlighting that AKI remains a significant contributor to morbidity and mortality in this cohort, as summarised in Table 6. Out of 155 patients, 135 survived and 20 died, as shown in [Figure 3]. All patients who died were older than 65 years. In contrast, among survivors, 69.6% (n = 94) were >65 years, 24.4% (n = 33) were 55–64 years, 4.4% (n = 6) were 45–54 years, and 1.5% (n = 2) were 35–44 years.

In the present study, comorbidities were associated with higher in-hospital mortality, and CAD and CVA were significantly associated with outcome (P<0.05), whereas DM, HTN, COPD/BA, old PTB, nil, and others were not (P>0.05), as summarised in [Table 7]. The association between age group and mortality was statistically significant (p = 0.041). Among aetiological factors, sepsis and age >65 years showed a statistically significant association with outcome (P<0.05). However, LV dysfunction, drug intake, obstructive uropathy, type 2 respiratory failure, DKA, accelerated hypertension, and HRS did not show a significant association (P>0.05). Among symptoms, decreased urine output and altered sensorium/reduced food intake were significantly associated with outcome (P<0.05), while loose stools, vomiting, fever, dysuria, abdominal pain, breathlessness, cough with expectoration, chest pain, and others were not (P>0.05). Regarding comorbidities, CAD and CVA were significantly associated with outcome (P<0.05), whereas DM, HTN, COPD/BA, old PTB, nil, and others were not (P>0.05). No significant association was found between gender or dialysis status and outcome (P>0.05) as shown in [Table 8].

Table 1: Existing comorbidities in patients with acute kidney injury (N=155)

Comorbidities	Frequency	Percentage
DM	123	79.4%
HTN	118	76.1%
CAD	47	30.3%
COPD/BA	12	7.7%
CVA	7	4.5%
Nil comorbidities	2	1.3%
Others (DCLD, RA)	13	8.4%

Table 2: Symptoms in the study population (N=155)

Symptoms	Frequency	Percentage
Loose stools	9	5.8%
Vomiting	43	27.7%
Fever	39	25.2%
Dysuria/Increased frequency of micturition	18	11.6%
Abdominal pain	9	5.8%
Decrease urine output	14	9.0%
Breathlessness	29	18.7%
Altered sensorium	40	25.8%
Cough expectoration	21	13.5%
Chest pain	4	2.6%
Others	13	8.4%

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Table 3: Clinical conditions associated with patients with acute kidney injury (N=155)

Etiology	Frequency	Percentage
Acute gastroenteritis	18	11.6%
Sepsis	99	63.9%
LV Dysfunction	22	14.2%
Drug Induced	5	3.2%
Obstructive uropathy	3	1.9%
Type 2 Respiratory failure	2	1.3%
DKA	5	3.2%
Accelerated HTN	2	1.3%
HRS	4	2.6%

Table 4: Baseline and 48-Hour Laboratory Parameters

Parameters	Median (IQR)
Hb	10 (9, 12)
TC	15000 (9600, 18000)
Urea	32 (27, 38)
Creatinine	1 (0.9, 1.1)
48hr Urea	75 (52.5, 92)
48hr Creatinine	2 (1.85, 2.3)

Table 5: Urine routine parameters in the study population (N=155)

Urine Routine Parameters	Frequency	Percentage
Protein +	34	21.9%
Pus cells plenty	15	9.7%
Normal	106	68.4%

Table 6: Analysis of Outcome in the study population (N=155)

Outcome	Frequency	Percentage
AKI resolved	124	80.0%
AKI not resolved	11	7.1%
Death	20	12.9%

Table 7: Comparison of comorbidities according to outcome in the study population (N=155)

Comorbidities	Outcome		P value
	Alive (n=135)	Death (n=20)	
DM	106 (78.5%)	17 (85%)	0.767
HTN	102 (75.6%)	16 (80%)	0.784
CAD	45 (33.3%)	2 (10%)	0.034
COPD/BA	10 (7.4%)	2 (10%)	0.655
CVA	3 (2.2%)	4 (20%)	0.006
Old PTB	2 (1.5%)	0 (0%)	1
Nil	2 (1.5%)	0 (0%)	1
Others	13 (9.6%)	0 (0%)	0.221

Table 8: Comparison of different parameters according to outcome in AKI (N=155)

Parameters	Outcome		P value
	Alive (n=135)	Death (n=20)	
Age in years			
35 to 44	2 (1.5%)	0 (0%)	
45 to 54	6 (4.4%)	0 (0%)	
55 to 64	33 (24.4%)	0 (0%)	
>65	94 (69.6%)	20 (100%)	0.041
Gender			
Male	75 (55.6%)	14 (70%)	0.223
Female	60 (44.4%)	6 (30%)	
Aetiology			
Acute gastroenteritis	18 (13.3%)	0 (0%)	0.082
Sepsis	81 (60%)	18 (90%)	0.009
LV Dysfunction	20 (14.8%)	2 (10%)	0.565
Drug Induced	5 (3.7%)	0 (0%)	1
Obstructive uropathy	3 (2.2%)	0 (0%)	1
Type 2 Respiratory failure	2 (1.5%)	0 (0%)	1
DKA	5 (3.7%)	0 (0%)	1
Accelerated HTN	2 (1.5%)	0 (0%)	1
HRS	4 (3%)	0 (0%)	1
Dialysis			
Done	5 (3.7%)	0 (0%)	1
Not done	130 (96.3%)	20 (100%)	
Symptoms			

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Loose stools	9 (6.7%)	0 (0%)	0.234
Vomiting	38 (28.1%)	5 (25%)	0.769
Fever	36 (26.7%)	3 (15%)	0.262
Dysuria/Increased frequency of micturition	18 (13.3%)	0 (0%)	0.082
Abdominal pain	9 (6.7%)	0 (0%)	0.234
Decrease urine output	9 (6.7%)	5 (25%)	0.008
Breathlessness	27 (20%)	2 (10%)	0.285
Altered sensorium	29 (21.5%)	11 (55%)	<0.001
Cough expectoration	16 (11.9%)	5 (25%)	0.109
Chest pain	4 (3%)	0 (0%)	1
Others	12 (8.9%)	1 (5%)	0.558

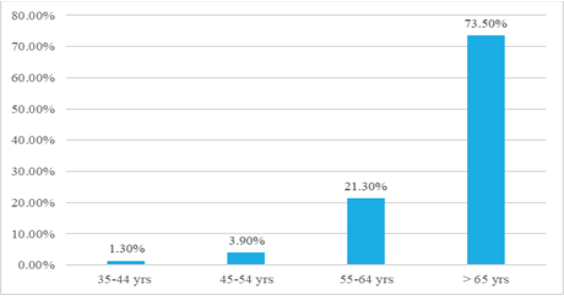

Figure 1: Age distribution of patients with Acute Kidney Injury

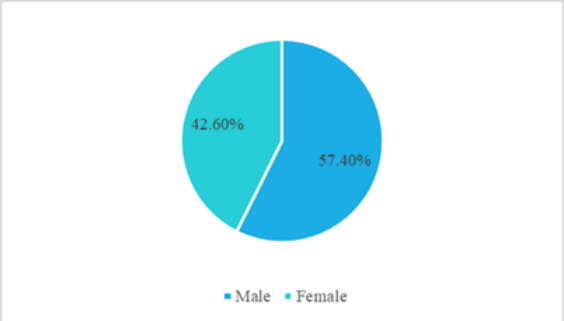

Figure 2: Sex distribution of patients with Acute Kidney Injury

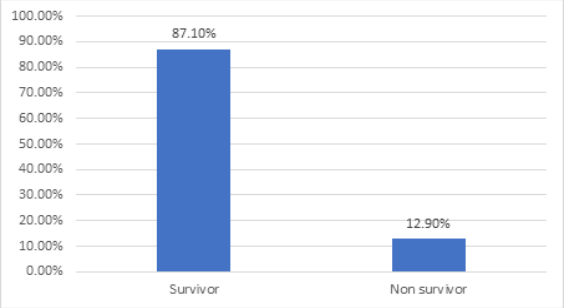

Figure 3: Outcome of patients in Acute Kidney Injury

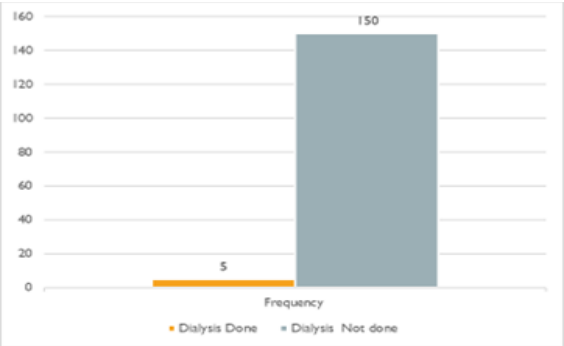

Figure 4: Distribution of Dialysis Requirement Among AKI Patients (n=155)

DISCUSSION

In the present study, the most common age group was >65 years. Bhattacharya PK et al,[8] in their study found that the mean age group was 41.09 ± 16.17 years. However, a review of large pooled data of AKI in the ICU setting from five American teaching universities (the PICARD group) in 618 patients found the mean age of patients to be higher at 59.5 years.[9]

In the present study, comorbidities were associated with higher in-hospital mortality, and CAD and CVA were significantly associated with outcome (P<0.05), whereas DM, HTN, COPD/BA, old PTB, nil, and others were not (P>0.05). The results are comparable to PICARD group,[9] reported extensive comorbidities in patients with AKI, with 37% having coronary artery disease, and 29% having diabetes mellitus. In our study, the most common presenting symptoms were vomiting (27.7%, n = 43), altered sensorium (25.8%, n = 40), and fever (25.2%, n = 39), it is similar to study conducted by Bhattacharya, et al,[8] in their study fever (40%) was the most common presenting symptom followed by oliguria (25.83%). In the present study, sepsis was the most common acute clinical condition associated with AKI, which was present in 63.9% of the patients. In a population-based study done by Ali et al,[10] with 1811 AKI patients, sepsis was the most frequent precipitating factor, being present in 47% of the cases. In another Indian study from the southern state of Tamil Nadu, Basu et al.[11] AKI was seen in 41.1% of patients with tropical acute febrile illness, with the most common causes being scrub typhus, malaria, salmonellosis, dengue, and leptospirosis.

Among the predictors of mortality in our study, older age (>65 years) was found to be a significant factor (p = 0.041), with all deaths occurring in this age group. This observation is consistent with the findings of Bagshaw et al,[12] and Kellum et al,[13] who reported higher mortality rates among elderly AKI patients, confirming advanced age as a major determinant of poor outcomes. Furthermore, a meta-analysis by Schmitt et al,[14] demonstrated that recovery from AKI was 28% lower in patients older than 65 years, further supporting our results. Sepsis was another strong predictor of mortality in our cohort (p = 0.009), while other causes, such as acute gastroenteritis and LV dysfunction, were not significant. This is in agreement with an Egyptian study of 532 patients, Abd ElHafeez et al,[15] which

ISSN (O): 2687-5365; ISSN (P): 2753-6556

found sepsis to be the most common cause of AKI and a predictor of non-recovery and increased mortality. Similarly, Hoste et al,[16] Bellomo et al,[17] and large retrospective data from the UPHS-AKI cohort (n = 6119) all identified sepsis as an independent predictor of mortality in AKI patients.

In addition, clinical features such as decreased urine output ($p = 0.008$) and altered sensorium ($p < 0.001$) were significantly associated with mortality in our study. These findings mirror the work of Schrier et al,[18] and Kaddourah et al,[19] who demonstrated that these symptoms are strong indicators of severe illness and poor prognosis in AKI patients. Dialysis status did not significantly influence mortality ($p = 1$). Also, Wald et al,[20] in their study reported that dialysis alone did not significantly impact mortality, consistent with our results. Gender did not significantly affect mortality ($p = 0.223$). This is comparable to the studies done by Yang et al,[21] and Chertow et al,[22] also found no significant gender differences in AKI outcomes.

CONCLUSION

Our study concluded that mortality was confined entirely to patients >65 years, confirming older age as a strong predictor of death in AKI. In contrast, younger patients (<65 years) demonstrated markedly better survival rates. The most common aetiology for developing Acute Kidney Injury was sepsis, comprising 64% of the total patients studied. Most common clinical presentations were altered sensorium, fever, vomiting, and decreased urine output, of which decreased urine output and altered sensorium were significantly associated with mortality. In our study, the presence of coronary artery disease (CAD) and cerebrovascular accident (CVA) showed a statistically significant association with mortality. Patients with these comorbidities were more likely to experience adverse outcomes compared to those without. This suggests that pre-existing cardiovascular and cerebrovascular conditions substantially increase vulnerability in AKI patients.

Limitations: This was a prospective observational study, which is a key strength as it allowed for systematic data collection, reduced recall bias, and ensured more reliable temporal assessment of exposures and outcomes compared to retrospective studies. The prospective design also enabled closer monitoring of patients, ensuring more accurate recording of symptoms, laboratory changes, and short-term outcomes. In our study, there are certain limitations that should be acknowledged. First, the relatively small sample size may have limited the statistical power to detect differences in some variables. Conditions such as left ventricular dysfunction, drug-induced AKI, or obstructive uropathy may have shown significant associations with larger cohorts. Subgroup analyses within etiologies (e.g., different stages of LV dysfunction or specific categories of drug-induced injury) were not feasible due to the limited number of cases. Additionally, being conducted at a single center, the findings may not be generalizable to broader populations with different demographics or healthcare settings. Although prospective, the study was limited to short-term in-hospital outcomes, and the lack of longitudinal follow-up restricted assessment of long-term renal recovery, recurrent AKI episodes, or survival beyond discharge.

REFERENCES

1. Abdel-Rahman, E. M., Turgut, F., Gautam, J. K., & Gautam, S. C. (2021). Determinants of outcomes of acute kidney injury: clinical predictors and beyond. Journal of Clinical Medicine, 10(6), 1175.
2. Makris K, Spanou L. Acute Kidney Injury: Definition, Pathophysiology and Clinical Phenotypes. Clin Biochem Rev. 2016 May;37(2):85-98. PMID: 28303073; PMCID: PMC5198510.
3. Basile, D. P., Anderson, M. D., & Sutton, T. A. (2012). Pathophysiology of acute kidney injury. Comprehensive physiology, 2(2), 1303-1353.
4. Section 1: Introduction and Methodology. Kidney Int Suppl (2011) [Internet]. 2012Mar;2(1):13–8. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2157171615310303
5. Kolhe, N. V., Staples, D., Reilly, T., Merrison, D., Mcintyre, C. W., Fluck, R. J., ... & Taal, M. W. (2015). Impact of compliance with a care bundle on acute kidney injury outcomes: a prospective observational study. PloS one, 10(7), e0132279.
6. Prakash, J., Singh, T. B., Ghosh, B., Malhotra, V., Rathore, S. S., Vohra, R., ... & Usha. (2013). Changing epidemiology of community-acquired acute kidney injury in developing countries: analysis of 2405 cases in 26 years from eastern India. Clinical kidney journal, 6(2), 150-155.
7. Case, J., Khan, S., Khalid, R., & Khan, A. (2013). Epidemiology of acute kidney injury in the intensive care unit. Critical care research and practice, 2013(1), 479730.
8. Bhattacharya PK, Roy A, Jamil M, Barman B, Murti SV, Marak PR. Clinical profile and determinants of short-term outcome of acute kidney injury: A hospital-based prospective study from Northeastern India. J Lab Physicians. 2019 Jan-Mar;11(1):5-10. doi: 10.4103/JLP.JLP_135_18. PMID: 30983795; PMCID: PMC6437823.
9. Mehta, R. L., Pascual, M. T., Soroko, S., Savage, B. R., Himmelfarb, J., Ikizler, T. A., ... & Program to Improve Care in Acute Renal Disease (PICARD. (2004). Spectrum of acute renal failure in the intensive care unit: the PICARD experience. Kidney international, 66(4), 1613-1621.
10. Ali, T., Khan, I., Simpson, W., Prescott, G., Townend, J., Smith, W., & MacLeod, A. (2007). Incidence and outcomes in acute kidney injury: a comprehensive population-based study. Journal of the American Society of Nephrology, 18(4), 1292-1298.
11. Basu, G., Chrispal, A., Boorugu, H., Gopinath, K. G., Chandy, S., Prakash, J. A. J., ... & John, G. T. (2011). Acute kidney injury in tropical acute febrile illness in a tertiary care centre—RIFLE criteria validation. Nephrology Dialysis Transplantation, 26(2), 524-531.
12. Bagshaw, S. M., Lapinsky, S., Dial, S., Arabi, Y., Dodek, P., Wood, G., ... & Cooperative Antimicrobial Therapy of Septic Shock (CATSS) Database Research Group. (2009). Acute kidney injury in septic shock: clinical outcomes and impact of duration of hypotension prior to initiation of antimicrobial therapy. Intensive care medicine, 35(5), 871-881.
13. Kellum, J. A., Lameire, N., & KDIGO AKI Guideline Work Group. (2013). Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (Part 1). Critical care, 17(1), 204.
14. Schmitt, R., Coca, S., Kanbay, M., Tinetti, M. E., Cantley, L. G., & Parikh, C. R. (2008). Recovery of kidney function after

ISSN (O): 2687-5365; ISSN (P): 2753-6556

acute kidney injury in the elderly: a systematic review and meta-analysis. American Journal of Kidney Diseases, 52(2), 262-271.
15. Abd ElHafeez, S., Tripepi, G., Quinn, R., Naga, Y., Abdelmonem, S., AbdelHady, M., ... & Ravani, P. (2017). Risk, predictors, and outcomes of acute kidney injury in patients admitted to intensive care units in Egypt. Scientific reports, 7(1), 17163.
16. Hoste, E. A., Clermont, G., Kersten, A., Venkataraman, R., Angus, D. C., De Bacquer, D., & Kellum, J. A. (2006). RIFLE criteria for acute kidney injury are associated with hospital mortality in critically ill patients: a cohort analysis. Critical care, 10(3), R73.
17. Bellomo, R., Kellum, J. A., Ronco, C., Wald, R., Martensson, J., Maiden, M., ... & Schneider, A. (2017). Acute kidney injury in sepsis. Intensive care medicine, 43(6), 816-828.
18. Schrier, R. W., Wang, W., Poole, B., & Mitra, A. (2004). Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. The Journal of clinical investigation, 114(1), 5-14.
19. Kaddourah, A., Basu, R. K., Bagshaw, S. M., & Goldstein, S. L. (2017). Epidemiology of acute kidney injury in critically ill children and young adults. New England Journal of Medicine, 376(1), 11-20.
20. Wald, R., Adhikari, N. K., Smith, O. M., Weir, M. A., Pope, K., Cohen, A., ... & Bagshaw, S. M. (2015). Comparison of standard and accelerated initiation of renal replacement therapy in acute kidney injury. Kidney international, 88(4), 897-904.
21. Yang, L., Xing, G., Wang, L., Wu, Y., Li, S., Xu, G., ... & Wang, H. (2015). Acute kidney injury in China: a cross-sectional survey. The Lancet, 386(10002), 1465-1471.
22. Chertow, G. M., Burdick, E., Honour, M., Bonventre, J. V., & Bates, D. W. (2005). Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. Journal of the American Society of Nephrology, 16(11), 3365-3370.

ISSN (O): 2687-5365; ISSN (P): 2753-6556