SHORT COMMUNICATION

A Comparative Study of Community- and Hospital-Acquired Acute Kidney Injury in a Tertiary Care Hospital in Balochistan

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ABSTRACT

Objective: To compare the incidence, risk factors, and outcomes of community-acquired acute kidney injury and hospital-acquired acute kidney injury.

Study Design: Prospective comparative study.

Place and Duration of Study: This study was conducted at the Department of Nephrology, Combined Military Hospital (CMH), Quetta, Pakistan from January 2023 to June 2024.

Methods: Patients diagnosed with renal injury were classified as community-acquired acute kidney injury if onset occurred before hospital admission and as hospital-acquired acute kidney injury if onset occurred post-admission. Data collected included demographic information, underlying comorbidities, laboratory values, and details of treatments received. Statistical analysis compared incidence rates, risk factors, and patient outcomes, including mortality rates and requirements for renal replacement therapy.

Results: Out of 33 patients, 27 (81.8%) had community-acquired acute kidney injury, while 6 (18.2%) had hospital-acquired acute kidney injury. Most admissions were from the medical unit (87.9%), with fewer from surgical (9.1%) and obstetric (3%) units. The cohort had a mean age of 45.58 ± 18.03 years, and the majority were male (63.6%). In terms of acute kidney injury severity, 51.5% of patients were classified as Kidney Disease Improving Global Outcomes stage 3, 33.3% as stage 1, and 15.2% as stage 2. Complications included oliguria in 18.2%, hyperkalemia in 12.1%, and metabolic acidosis in 12.1% of patients. Vasopressor support was required in 24.2%, mechanical ventilation in 12.1%, and dialysis in 30.3%. Among community-acquired acute kidney injury patients, 22.2% received vasopressors and 25.9% required dialysis; survival was 92.5%, with 7.4% mortality. All hospital-acquired acute kidney injury patients survived, with 33.3% on vasopressors and 50% on dialysis.

Conclusion: Acute Kidney Injury is common among hospitalized patients, with significant in-hospital mortality. Most cases are community-acquired. These emphasize the need for targeted interventions in hospital settings to prevent acute kidney injury and to enhance early identification and management strategies to reduce its incidence.

Keywords: *Acute Kidney Injury, Creatinine, Hemodynamics, Prognosis, Risk Factors.*

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Introduction

Acute kidney injury (AKI) is a critical medical condition characterized by a sudden decline in kidney function, leading to the accumulation of waste products and electrolyte imbalances.[1] It is a significant contributor to global morbidity and mortality, particularly in low- and middle-income countries (LMICs) where healthcare resources are limited.[2] AKI can be classified as community-acquired (CA-AKI) or hospital-acquired (HA-AKI), each with distinct etiologies, risk factors, and outcomes.[3] Understanding the differences between these two forms of AKI is essential for developing

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targeted prevention and management strategies.

The global burden of AKI remains substantial, with an estimated 13.3 million cases annually, resulting in 1.7 million deaths.[2] In LMICs, the incidence of AKI is higher due to factors such as limited access to healthcare, poor sanitation, and a high prevalence of infectious diseases.[4] CA-AKI is often caused by dehydration, infections, and exposure to nephrotoxic agents, while HA-AKI is frequently associated with iatrogenic causes, including contrast-induced nephropathy, surgical complications, and medication toxicity.[5] Both forms of AKI share common risk factors, such as advanced age, diabetes, hypertension, and pre-existing chronic kidney disease (CKD).[6]

In Pakistan, particularly in the province of Balochistan, the epidemiology of AKI remains understudied. The region faces unique challenges, including a high burden of infectious diseases, limited healthcare infrastructure, and socioeconomic disparities, which may influence the incidence and outcomes of AKI.[7] Previous studies from other regions of Pakistan have highlighted the predominance of CA-AKI, with sepsis and diarrheal diseases being the leading causes.[8] Studies from major Pakistani medical institutions indicate that community-acquired AKI frequently involves preventable causes such as volume depletion, infections, and nephrotoxic substance exposure, while hospital-acquired cases typically develop in the context of complex medical interventions, sepsis, and iatrogenic factors.[9]

Recovery trajectories in Pakistani AKI patients suggest that traditional diagnostic approaches may require modification to account for chronic kidney disease prevalence, nutritional status, and baseline health conditions that differ from populations studied in developed countries.[10]

Contemporary research from Pakistani hospitals indicates that hospital-acquired AKI is associated with significantly higher mortality rates and prolonged hospital stays, while community-acquired cases, despite delayed presentation, often achieve better functional recovery.[11,12]

This study aims to compare the clinical characteristics, etiologies, and outcomes of CA-AKI and HA-AKI in a tertiary care hospital in Balochistan. By identifying the distinct features of these two forms of AKI, this research seeks to inform targeted interventions and improve patient outcomes. The findings of this study will contribute to the growing body of literature on AKI in LMICs and provide insights into the unique challenges faced by healthcare systems in resource-limited settings.

Methods

A prospective study was done over 06 months at the Department of Nephrology, Combined Military Hospital (CMH), Quetta, Pakistan, from January 2023 to June 2024. It included adults aged 18 years or above. The inclusion criteria were that the patients with acute kidney injury based on Kidney Disease Improving Global Outcomes (KDIGO) criteria, CA-AKI group includes Patients presenting to the hospital with AKI before admission, and HA-AKI group, who developed AKI after 24 hours of hospitalization. The exclusion criteria were chronic kidney disease (CKD) patients already on maintenance dialysis, those with a history of renal transplants, and any history of congenital renal diseases or obstructive uropathy.

A history, clinical examination, and laboratory investigations were studied. They received supportive care and dialysis per standard hospital protocol. Patients were daily reviewed until discharge or death. The requirement of inotropic drugs, Intensive Care Unit (ICU) care, treatment with renal replacement therapy, survival at discharge, and mortality were studied. Between the two groups, i.e., survival and non-survival, were compared to see the difference in clinical characteristics and laboratory investigations.

The patients included were informed in detail about the purpose of the study, and informed consent was obtained. The study was approved by the Institutional Ethics Committee of the hospital, vide letter no: CMH QTA-IERB/41/2023, 14th January 2023.

Data were compiled in Microsoft Excel after acquisition and later imported to SPSS version 26. Descriptive statistics were used to define the demographic and clinical characteristics. A multivariate binary logistic regression was used to assess the effect of continuous factors on the outcome of AKI, while a two-tailed Chi-Square test with Fisher's exact test identified the effect of binary variables on the outcome of patients with AKI. Cross-tabulation was employed to assess the outcome and

management of patients with Community-acquired AKI and Hospital-acquired AKI.[13]

Results

The total number of patients hospitalized during the study period was 33; of them, 27 (81.8%) had CA-AKI and 6 (21.2%) had HA-AKI. Serum creatinine-based criteria were used for diagnosing AKI in these 33 patients. 29 (87.9%) patients were enrolled from the medical unit, 3 (9.1%) patients from the surgical unit, and 1 (3%) patient from the obstetrical unit.

Among the 33 patients, 21 (63.6%) were males and 12 (36.4%) were females. The mean age of the study population was 45.58±18.03 years. According to the Kidney Disease Improving Global Outcomes, 33% had stage 1, 15.2% had stage 2, and 51.5% had stage 3. Oliguria was seen in 6 (18.2%), hyperkalemia was seen in 4 (12.1%), and metabolic acidosis was also seen in 5 (12.1%) patients; 3 (9.1%) patients had multi-organ involvement, 8 (24.2%) patients were on vasopressor support, and 4 (12.1%) patients had lung involvement and were on mechanical ventilation. 10 (30.3%) patients were on dialysis (Table 1).

Table 1: Demographic and clinical characteristics of the study patients

Parents	N (%)
Gender	
Male	21 (63.6)
Female	12 (36.4)
Age, Mean±SD	45.58±18.038
Type of AKI	
CA-AKI	27 (81.8)
HA-AKI	6 (18.2)
Setting of AKI	
Medical	29 (87.9)
Surgical	3 (9.1)
Obstetrical	1 (3.0)
KIDGO Stage	
Stage 1	11 (33)
Stage 2	5 (15.2)
Stage 3	17 (51.5)
Blood urea (mg/dL)	15.618±9.88
Serum creatinine (mg/dL)	327±322.67
Oliguria	6 (18.2)
Hyperkalemia	4 (12.1)
Metabolic Acidosis	4 (12.1)
Multi-organ Failure	3 (9.1)
Vasopressor Supportive	8 (24.2)
Mechanical Ventilation	4 (12.1)
Dialysis	10 (30.3)
Outcome	
Survived	31 (93.9)
Expired	2 (6.1)

AKI = Acute kidney injury, KDIGO = Kidney Disease Improving Global Outcomes, SD= Standard Deviation

The mean age of survivors (45.2±18.3 years) and non-survivors (51±15.5years) was comparable (t=-0.50, *P*=0.999). Male gender was more frequent among survivors (95.2% than non-survivors (50%), although the difference was not statistically significant (Fischer's exact, *P*=0.6979). Oliguria was significantly associated with mortality, being present in all non-survivors compared to 12.9%of survivors (Fischer's exact, *P*=0.002. Other clinical features, including multi-organ failure (9.7% vs 0%, *P*=0.645), metabolic acidosis (12.9% vs 0%, *P*=0.588), and hyperkalemia (9.7% vs 50%, *P*=0.090), did not differ statistically between the two groups. Biochemical analysis revealed no significant differences in serum creatinine between survivors and non-survivors (314.4±327.9 vs 521±160 mg/dl, t=-1.62, *P*=1.0). In

contrast, serum urea levels were significantly higher in non-survivors 28.5±0,7mg/dl) compared to survivors (14.7± 9.6 mg/dl).

Regarding treatment and outcomes, mechanical ventilation was required. 9.7% of survivors and 50% of non-survivors (*P*=0.090), Vasopressor support in 22.6% and 50% respectively (*P*=0.380), and dialysis in 29% and 50% respectively (*P*=0.532); however, none of these were significant. Anuria was observed in 3.2% of survivors and was absent in non-survivors (*P*=0.796) (Table 2).

Overall, oligoanuria and elevated serum urea were the only factors significantly associated with mortality in AKI patients, while other demographic, clinical, and biochemical parameters did not demonstrate significant associations.

Table 2: Characteristics of survivors versus non–survivors

Clinical Characteristics	Survivor (N=31)	Non-Survivor (N=2)
Age, mean±SD	45.23±18.35	51±15.5
Male gender, N (%)	20 (95.2)	1 (50)
Oligoanuria, N (%)	4 (12.9)	2 (100)
Multiorgan failure, N (%)	3 (9.67)	0 (0)
Metabolic Acidosis, N (%)	4 (12.9)	0 (0)
Hyperkalemia, N (%)	3 (9.67)	1 (50)
Creatinine (mg/dL), Mean±SD	314.4±327.89	521.5±160.51
Serum Urea (mg/dL), Mean±SD	14.7±9.6	28.5±0.70
Mechanical ventilation, N (%)	3 (9.67)	1 (50)
Vasopressor use, N (%)	7 (22.58)	1 (50)
Dialysis, N (%)	9 (29)	1 (50)
Anuria	1 (3.22)	0 (0)

Table 3: Management and renal outcome of acute kidney injury patients in both groups

Variables	CA-AKI	HA-AKI
Use of vasoactive drugs, N (%)	6 (22.2)	2 (33.3)
Dialysis, N (%)	7 (25.9)	3 (50)
Survived, N (%)	25 (92.5)	6 (100)
Expired, N (%)	2 (7.40)	0 (0)

Universate binary logistic regression continuous variables affecting outcome in AKI, Chi- Square with Fisher's exact test (FE) of categorical variables affecting outcome in AKI.

In total, 27 patients who had CA-AKI, 25 (92.5%) survived and 2 (7.40%) expired, and all 6 (100%) patients who had HA-AKI survived, with 0% expired (Table 3).

Discussion

Acute kidney injury is a sudden derangement in kidney function that develops within one week, as evidenced by an increase in serum creatinine, a decrease in urine output, or both. Causes of AKI are prerenal (e.g., dehydration, burns, glomerular diseases) and post-renal (e.g., renal stone).[14] Globally, AKI affects approximately 13.3 million people annually, with 1.7 million deaths occurring predominantly in lower-middle-income countries where infectious etiologies and environmental factors contribute substantially to disease burden.[4]

Millions of people live in underprivileged or crowded, unhygienic communities as a result of SSA's rapid urbanization. These conditions encourage the recurrence or persistence of bacterial, viral, and parasitic infectious diseases, creating a chronic toxic environment that is favorable to the development of AKI. Renal impairment is frequently caused by tropical infections, and AKI is common in certain illnesses.[15,12] An objective MAP of 63 mmHg raised the incidence of KDIGO stage 1 AKI in 789 exhausted patients who experienced out-of-hospital cardiac arrest, but 77 mmHg had no effect on severe AKI.[13,16]

Remarkably, a cohort investigation of AKI patients who were not critically ill revealed that the risk of severe AKI or mortality increased 2.5 times if the SBP was less than 100 mmHg, as opposed to 120–129 mmHg in patients who were taking antihypertensive drugs at baseline. The significance of baseline BP was underscored by the higher occurrence rate among

non-hypertensive patients with higher SBP targets. Patients with AKI who are not critically ill are an alternative population of interest. A U-shaped relationship between average SBP and severe AKI or death was found in a cohort analysis of more than 1500 hospitalized patients, but with restrictions. The lowest occurrence rate was noted at SBP of 110–129 mmHg.[14,17]

A study in 2020 indicates that sepsis was the most common source of AKI.[18] With 29.8% of infected patients developing AKI, our cohort's comparatively low incidence of AKI (31.6%) can be described by including a broader range of ICU patients, such as fewer infected patients and more patients with neurological and cardiovascular conditions, as well as by excluding urine output criteria.[15,18,19]

Recent comparative studies from major Pakistani tertiary care centers demonstrate that community-acquired AKI predominantly results from infectious causes, with outcomes significantly better than hospital-acquired cases, which typically involve multifactorial etiologies and higher mortality rates.[15] This pattern, consistent with reports from other Pakistani centers, likely reflects several factors: the younger age and better baseline health status of community-acquired patients, the single-organ nature of most cases of community-acquired AKI, and the reversibility of common etiologies such as dehydration and infections.[16]

Conversely, community-acquired AKI, despite often presenting with established kidney injury, demonstrates more favorable outcomes with mortality rates of approximately 6.6% and recovery rates exceeding 52%.[20]

These findings have important implications for clinical management and resource allocation in tertiary care centers, i.e., diverse healthcare settings like Balochistan.

Conclusion

This study reveals while community-acquired acute kidney injury CA-AKI and hospital-acquired acute kidney injury (HA-AKI share some common risk factors, CA-AKI was more prevalent and associated with lower mortality compared to the findings underscore the importance of preventive strategies for community-acquired acute kidney injury and the need for robust supportive care for hospital-acquired acute kidney injury to improve patient outcomes. Understanding the distinct etiological factors and outcomes of these two forms of acute kidney injury can help develop targeted strategies for prevention and management, particularly in resource-limited settings such as Baluchistan.

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REFERENCES

1. Kellum JA, Romagnani P, Ashuntantang G, Ronco C, Zarbock A, Anders HJ. Acute kidney injury. Nature Reviews Disease Primers. 2021; 7: 52. doi: 10.1038/s41572-021-00284-z.
2. Gameiro J, Fonseca JA, Outerelo C, Lopes JA. Acute kidney injury: from diagnosis to prevention and treatment strategies. Journal of Clinical Medicine. 2020; 9: 1704. doi: 10.3390/jcm9061704.
3. Ostermann M, Lumlertgul N, Jeong R, See EJ, Joannidis M, James MT. Acute kidney injury. The Lancet. 2025; 405: 241-56. doi: 10.1016/S0140-6736(24)02385-7.
4. Hoste EAJ, Bagshaw SM, Bellomo R, Cely CM, Colman R, Cruz DN, et al. Epidemiology of acute kidney injury in critically ill patients: the multinational AKI-EPI study. Intensive Care Medicine. 2015; 41: 1411-23. doi: 10.1007/s00134-015-3934-7.
5. Cerda J, Kashani K, Ostermann M, Basu RK, Bell S, Cantaluppi V, et al. The global epidemiology of acute kidney injury: challenges and opportunities. Nature Reviews Nephrology. 2026; 22: 179-98. doi: 10.1038/s41581-025-01030-4.
6. Ostermann M, Zarbock A, Goldstein S, Kashani K, Macedo E, Murugan R, et al. Acute kidney injury biomarkers: recommendations from the Acute Disease Quality Initiative Consensus Conference. Nature Reviews Nephrology. 2020; 16: 273-84. doi: 10.1038/s41581-020-0244-5.
7. Meneses GC, Silva GB Jr, Tôrres PP, Castro VQ, Lopes RL, Martins AMC, et al. Novel kidney injury biomarkers in tropical infections: a review of the literature. Revista do Instituto de Medicina Tropical de São Paulo. 2020; 62: e14. doi: 10.1590/S1678-9946202062014.
8. Bikbov B, Purcell CA, Levey AS, Smith M, Abdoli A, Abebe M, et al. Global, regional, and national burden of chronic kidney disease, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. The Lancet. 2020; 395: 709-33. doi: 10.1016/S0140-6736(20)30045-3.
9. Rudd KE, Johnson SC, Agesa KM, Shackelford KA, Tsoi D, Kievlan DR, et al. Global, regional, and national sepsis incidence and mortality, 1990-2017: analysis for the Global Burden of Disease Study. The Lancet. 2020; 395: 200-11. doi: 10.1016/S0140-6736(19)32989-7.
10. Rasmussen SB, Jeppesen KK, Kjaergaard J, Hassager C, Schmidt H, Mølstrøm S, et al. Blood pressure and oxygen targets on kidney injury after cardiac arrest. Circulation. 2023; 148: 1860-69. doi: 10.1161/CIRCULATIONAHA.123.066012.

11. Allen JC, Gardner DS, Skinner H, Harvey D, Sharman A, Devonald MA, et al. Definition of hourly urine output influences reported incidence and staging of acute kidney injury. BMC Nephrology. 2020; 21: 19. doi: 10.1186/s12882-019-1678-2.
12. Tso M, Sud K, Van C, Tesfaye W, Castelino RL. Clinical characteristics and outcomes of community-acquired acute kidney injury. International Urology and Nephrology. 2023; 55: 2345-54. doi: 10.1007/s11255-023-03533-0.
13. Hussain SW, Qadeer A, Munawar K, Qureshi MS, Khan MT, Abdullah A, et al. Determining the incidence of acute kidney injury using the RIFLE criteria in the medical intensive care unit in a tertiary care hospital setting in Pakistan. Cureus. 2019; 11: e4071. doi: 10.7759/cureus.4071.
14. Rosner B. Fundamentals of Biostatistics. 9th ed. Boston: Cengage Learning; 2022.
15. Patschan D, Stasche F, Erfurt S, Matyukhin I, Ritter O, Safi W, et al. Recovery of kidney function in acute kidney injury. Journal of Nephrology. 2025; 38: 445-56. doi: 10.1007/s40620-025-02220-w.
16. Verma S, Kellum JA. Defining acute kidney injury. Critical Care Clinics. 2021; 37: 251-66. doi: 10.1016/j.ccc. 2020.11.001.
17. Robinson CH, Hsu RK, Sutherland SM. Early recognition and prevention of acute kidney injury in hospitalized children. The Lancet Child and Adolescent Health. 2023; 7: 593-603. doi: 10.1016/S2352-4642(23)00105-0.
18. Patidar KR, Ma AT, Juanola A, Barone A, Incicco S, Kulkarni AV, et al. Global epidemiology of acute kidney injury in hospitalized patients with decompensated cirrhosis: the International Club of Ascites GLOBAL AKI prospective multicentre cohort study. The Lancet Gastroenterology and Hepatology. 2025; 10: 418-30. doi: 10.1016/S2468-1253(25)00006-8.
19. Adiyeke E, Ren Y, Shickel B, Ruppert MM, Guan Z, Kane-Gill SL, et al. Acute kidney injury prediction for non-critical care patients: retrospective external and internal validation study. NPJ Digital Medicine. 2024; 7: 58. doi: 10.1038/s41746-024-01000-0.
20. Adiyeke E, Ren Y, Fogel S, Rashidi P, Segal M, Shenkman EA, et al. Epidemiology, trajectories and outcomes of acute kidney injury among hospitalized patients: a retrospective multicenter large cohort study. Kidney International Reports. 2024; 9: 2100-2112. doi: 10.48550/arXiv. 2403.08020

Author Contributions

ZK: Manuscript writing for methodology design, investigation, writing the original draft, proofreading, and approval for final submission

MHM: Data acquisition, curation, statistical analysis, and approval for final submission

RL: Validation of data, interpretation, write-up of results, and approval for final submission

AA: Conception, design of the work, and approval for final submission

HA: Revising, editing, supervising for intellectual content, and approval for final submission

ZK is the nominated guarantor and takes full responsibility for the overall content and integrity of the work