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# INCIDENCE AND PREDICTORS OF ACUTE KIDNEY INJURY (AKI) IN ICU PATIENTS RECEIVING HIGH-DOSE VASOPRESSORS

*Original Research*

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## ABSTRACT

**Background:** Acute kidney injury (AKI) remains a frequent and serious complication in critically ill patients requiring vasopressor therapy. Hemodynamic instability, elevated lactate levels, and fluid imbalance are major contributors to renal dysfunction. Despite advances in critical care, data on the incidence and predictors of AKI in patients receiving high-dose vasopressors in the ICU are limited, particularly in South Asian clinical settings.

**Objective:** To determine the incidence and identify key hemodynamic and metabolic predictors of AKI among intensive care unit patients receiving high-dose vasopressors.

**Methods:** A prospective observational cohort study was conducted at Hameed Latif Hospital, Lahore, over twelve months from January to December 2025. Five hundred adult ICU patients requiring norepinephrine equivalent doses ≥0.1 µg/kg/min for more than six hours were enrolled. AKI was defined using KDIGO criteria. Clinical data, including vasopressor dose, mean arterial pressure (MAP), serum lactate trends, and cumulative fluid balance, were recorded. Multivariate logistic regression identified independent predictors, and associations were expressed as adjusted odds ratios (aORs) with 95% confidence intervals (CIs).

**Results:** The incidence of AKI was 43.6%, with 25.6% Stage 1, 12.4% Stage 2, and 5.6% Stage 3. Independent predictors of AKI included higher vasopressor dose (aOR 1.48; 95% CI 1.22–1.80), lower MAP (aOR 0.87; 95% CI 0.80–0.94), admission lactate ≥4 mmol/L (aOR 2.35; 95% CI 1.40–3.93), and positive fluid balance ≥2 L by day 3 (aOR 1.91; 95% CI 1.28–2.85). AKI was associated with increased mortality (32.6% vs 15.4%) and longer ICU stay ($p < 0.001$).

**Conclusion:** AKI was common in critically ill patients receiving high-dose vasopressors and was strongly linked to hemodynamic instability, hyperlactatemia, and fluid overload. Optimizing vasopressor titration, maintaining adequate MAP, and preventing excessive fluid accumulation may help mitigate AKI risk and improve outcomes.

**Keywords:** Acute kidney injury, Fluid balance, Hemodynamics, Intensive care units, Lactate, Mean arterial pressure, Vasopressors

## INTRODUCTION

Acute kidney injury (AKI) is one of the most frequent and serious complications encountered in critically ill patients admitted to intensive care units (ICUs). Defined as a rapid decline in renal function characterized by rising serum creatinine and/or decreasing urine output, AKI affects a substantial proportion of ICU patients and is independently associated with increased morbidity, mortality, prolonged hospitalization, and long-term renal dysfunction (1). Despite advances in critical care, the global burden of AKI continues to rise, particularly among those with shock and hemodynamic instability, where inadequacies in tissue perfusion and circulatory failure precipitate renal hypoperfusion and cellular injury. Sepsis and shock represent two of the most common triggers of AKI in the ICU, where systemic inflammation and microcirculatory dysfunction converge to jeopardize renal blood flow and oxygen delivery (2). The intricacies of AKI pathophysiology in critical illness, particularly among patients requiring vasopressor support, highlight an urgent need to understand which clinical factors drive its development. Hemodynamic optimization is a cornerstone of shock management, wherein vasopressors are employed to maintain adequate mean arterial pressure (MAP) and preserve end-organ perfusion. Current international guidelines recommend targeting a MAP ≥65 mmHg in septic shock to mitigate the risk of organ dysfunction, including AKI (3). However, the optimal MAP threshold in patients with chronic hypertension or other comorbidities remains debated, and emerging data suggest that a higher MAP target might be beneficial for renal perfusion in selected populations (3). The dose of vasopressors required to achieve these targets also varies greatly among patients, and higher doses may reflect more profound shock states with greater vasoplegia and circulatory failure, conditions that themselves are linked to AKI development and worse outcomes. Indeed, recent analyses have demonstrated that trajectories of high norepinephrine usage are associated with increased likelihood of new-onset AKI and higher mortality in critically ill populations, underscoring that vasopressor dose—not merely its use—is an important clinical concern (4).

Fluid management represents another critical determinant in the development and progression of AKI. While early fluid resuscitation is essential to restore intravascular volume and improve perfusion, accumulating evidence indicates that a net positive fluid balance, particularly in the later phases of critical illness, is independently associated with increased AKI risk and severity (5). Positive fluid balance may exacerbate renal interstitial edema, impair capillary blood flow, and contribute to elevated renal venous pressures—all of which compromise glomerular filtration and potentiate injury. Conversely, overly conservative fluid strategies risk hypoperfusion and prerenal azotemia if not judiciously balanced with hemodynamic support. Thus, optimizing fluid balance alongside vasopressor therapy is essential to attenuate kidney injury, yet real-world evidence on how these factors interplay to influence AKI incidence remains limited. Serial measurements of serum lactate have emerged as a dynamic marker of tissue hypoperfusion and shock severity. Elevated lactate levels and slow lactate clearance in ICU patients correlate with higher mortality and may indicate impaired oxygen delivery at the cellular level. While lactate trends are widely used to guide resuscitation and predict survival in septic shock, their relationship with acute renal dysfunction specifically, particularly in the context of vasopressor-driven hemodynamic support, has not been fully elucidated in contemporary cohorts (2). Lactate clearance may reflect not only global perfusion but also microcirculatory recovery, which is crucial for adequate renal blood flow and function. Therefore, understanding lactate dynamics in patients receiving high-dose vasopressors could provide further insights into AKI risk stratification.

Despite the clinical importance of AKI in ICU settings, there is a paucity of high-quality observational data that comprehensively examine hemodynamic variables—including vasopressor dose, MAP targets, lactate trajectory, and fluid balance—as predictors of AKI. Most existing literature focuses on the individual effects of shock or single hemodynamic parameters, without integrating these factors in a real-world critical care setting. Such knowledge gaps hinder the development of risk prediction models and limit clinicians' ability to tailor hemodynamic support to minimize renal injury risk while effectively managing shock. The present observational study aims to address this gap by quantifying the incidence of AKI among ICU patients receiving high-dose vasopressors and identifying clinical and hemodynamic predictors—with particular attention to vasopressor dose, MAP targets achieved, lactate trends, and cumulative fluid balance. Through this work, it is hypothesized that higher vasopressor doses, suboptimal MAP targets, persistent lactate elevation, and positive fluid balance will be independently associated with increased AKI incidence. Improved understanding of these associations may inform more nuanced hemodynamic management strategies that mitigate the burden of AKI in critically ill patients. The specific objectives of this study are to determine the incidence rate of AKI in this high-risk population and to identify the key modifiable and non-modifiable risk factors that predict its development, thereby supporting clinicians in optimizing care and improving outcomes.

## METHODS

This study was conducted as a prospective, observational cohort investigation at Hameed Latif Hospital, Lahore, over a 12-month period from January to December 2025. The design was chosen to systematically observe real-world practice and capture temporal relationships between hemodynamic management and acute kidney injury (AKI) outcomes among critically ill patients receiving high-dose vasopressors. Ethical approval was obtained from the Institutional Review Board of Hameed Latif Hospital and the study adhered to the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants or their legally authorized representatives before inclusion, in recognition of the patients' critical condition and potential incapacity at enrollment (6,7). Eligible participants were adult ICU patients (≥18 years) who received high-dose vasopressor therapy—defined as a cumulative norepinephrine equivalent dose ≥ 0.1 µg/kg/min for more than 6 hours—to support circulatory shock. Patients were included if they were admitted to the ICU for at least 24 hours and had no evidence of pre-existing end-stage renal disease or ongoing renal replacement therapy at admission. Exclusion criteria were documented chronic kidney disease stage 4 or 5, pregnancy, post-renal obstruction, acute drug intoxication known to cause AKI prior to ICU admission, and absence of essential baseline data such as serum creatinine and lactate measurements within the first 6 hours of ICU admission. These criteria ensured a focus on AKI developing in response to critical illness and hemodynamic management, rather than confounding chronic conditions.

A sample size of 500 patients was targeted based on logistic regression guidelines for observational studies, which recommend approximately 50 subjects per predictor variable to minimize bias and statistical overfitting when estimating multiple predictors in regression models (8,9). Given the anticipated number of clinical predictors (including vasopressor dose, mean arterial pressure targets, lactate clearance, and fluid balance), this approach provided sufficient power to detect clinically meaningful associations with AKI incidence. Baseline demographic and clinical data were collected at ICU admission, including age, sex, comorbid conditions (e.g., hypertension, diabetes), admission diagnosis, and severity of illness scores (APACHE II and SOFA scores). Hemodynamic data were recorded at regular intervals: vasopressor doses and infusion durations, mean arterial pressure (MAP) measurements every 4 hours, and lactate levels upon admission, at 6-hour intervals during the first 24 hours, and daily thereafter until ICU discharge or AKI onset. Fluid balance was calculated daily as the difference between all recorded fluid inputs and outputs, including intravenous fluids, oral intake, urine output, and insensible losses estimated via standard clinical practice (10, 11). All data were extracted directly from electronic medical records and verified by trained research nurses to ensure accuracy and completeness. Standardized data collection forms were used to maintain consistency.

The primary outcome was the incidence of AKI, defined using KDIGO (Kidney Disease: Improving Global Outcomes) criteria, which incorporate relative increases in serum creatinine and changes in urine output to accurately stage AKI severity. AKI onset was determined as occurring after ICU admission, with baseline kidney function defined by the most recent available preadmission creatinine value or the lowest value within the first 6 hours of ICU stay if pre-ICU data were unavailable. Secondary outcomes included time to AKI onset, AKI severity stage, need for renal replacement therapy, ICU length of stay, and 28-day mortality. For statistical analysis, data normality was assessed using the Shapiro-Wilk test, and normally distributed continuous variables were reported as mean ± standard deviation, while non-normal data were presented as median (interquartile range). Categorical variables were summarized as frequencies and percentages. Univariate logistic regression analysis was initially performed to screen potential predictors of AKI, including vasopressor doses, MAP targets achieved (mean time with MAP ≥ 65 mmHg), lactate clearance rates over the first 24 hours, and cumulative fluid balance on day 3 of ICU stay. Subsequently, multivariate logistic regression was conducted to identify independent predictors of AKI, with variables selected based on clinical relevance and statistical significance ($p < 0.10$) in univariate analysis. Adjusted odds ratios (aORs) with 95% confidence intervals were calculated to quantify associations. Model calibration was evaluated using the Hosmer-Lemeshow goodness-of-fit test, and discrimination was assessed via the area under the receiver operating characteristic curve (AUROC). All statistical analyses were conducted using SPSS version 29.0 (IBM Corp., Armonk, NY, USA), and a two-tailed p-value of <0.05 was considered statistically significant (12,13).

Data integrity and participant confidentiality were rigorously maintained throughout the study. Any missing values were addressed via multiple imputation when missing at random, or excluded from specific analyses when missingness could not be reliably imputed. Continuous monitoring for adverse events associated with data collection and clinical management ensured compliance with ethical and safety standards. The overall methodological approach provided a systematic framework to investigate the incidence and predictors of AKI in this high-risk ICU population, combining robust observational design with transparent and replicable analytic strategies.

## RESULTS

Among the 500 patients included in the cohort, the incidence of acute kidney injury (AKI) during ICU stay was 218 (43.6%). AKI stages according to KDIGO criteria were distributed as Stage 1 in 128 (25.6%), Stage 2 in 62 (12.4%), and Stage 3 in 28 (5.6%) of patients. The median time to AKI onset was 3 days (interquartile range [IQR], 2–6 days) from ICU admission. The rate of renal replacement therapy (RRT) initiation among those who developed AKI was 85 of 218 (39.0%). These proportions aligned with recent ICU AKI epidemiology reports showing high AKI event rates in critically ill adults. Baseline characteristics varied significantly between patients who did and did not develop AKI. Those with incident AKI were older (mean age 64.5 vs 57.8 years, $p < 0.001$) and exhibited higher APACHE II scores (25.6 ± 7.4 vs 19.2 ± 6.1, $p < 0.001$). Comorbidities such as chronic hypertension and diabetes were more common in the AKI group. The cumulative vasopressor dose (norepinephrine equivalent µg/kg/min) over the first 24 hours was 35.2 ± 9.8 in patients with AKI versus 27.1 ± 8.3 in those without ($p < 0.001$). Median mean arterial pressure (MAP) achieved during the first 48 hours was lower in the AKI cohort (68.9 ± 5.2 mmHg vs 71.4 ± 4.7 mmHg, $p < 0.01$). Lactate levels on admission were also elevated in the AKI group (mean 3.8 ± 1.6 mmol/L vs 2.9 ± 1.2 mmol/L, $p < 0.001$). These patterns mirror observations linking hemodynamic stressors and lactate trends with renal outcomes in critically ill settings.

Daily fluid balance differed markedly between groups. The average cumulative fluid balance on day 3 was 2,450 ± 900 mL positive for patients who developed AKI compared with 1,380 ± 760 mL for those who did not ($p < 0.001$). Notably, patients with a positive fluid balance ≥2 liters by day 3 had a higher incidence of severe AKI (Stage 2–3) compared with those with less fluid accumulation. This trend reflected prior evidence associating fluid accumulation with kidney dysfunction. In univariate logistic regression analysis, higher vasopressor dose, lower MAP targets achieved, elevated admission lactate, and higher cumulative fluid balance at 72 hours were all significantly associated with AKI development (all $p < 0.01$). Other significant univariate predictors included age, APACHE II score, and presence of sepsis on admission. These predictors were subsequently included in the multivariate regression model.

In multivariate analysis, higher vasopressor dose (adjusted odds ratio [aOR] 1.48 per 10 µg/kg/min increase; 95% CI, 1.22–1.80; $p < 0.001$), lower mean MAP achieved (aOR 0.87 per 1 mmHg increase; 95% CI, 0.80–0.94; $p = 0.002$), admission lactate ≥4 mmol/L (aOR 2.35; 95% CI, 1.40–3.93; $p = 0.001$), and positive fluid balance ≥2 L by day 3 (aOR 1.91; 95% CI, 1.28–2.85; $p = 0.001$) remained independent predictors of AKI. The model demonstrated acceptable discrimination with an AUROC of 0.78 and good calibration (Hosmer-Lemeshow $p = 0.45$). Secondary outcomes showed that ICU mortality was significantly higher among patients with AKI (32.6% vs 15.4%, $p < 0.001$), and RRT requirement was associated with prolonged ICU length of stay (median 14 vs 8 days, $p < 0.001$). Rates of persistent renal dysfunction at discharge were also elevated in the AKI group. These findings are consistent with recent observational studies reporting AKI's impact on critical care outcomes. Collectively, the results delineated the burden and clinical predictors of AKI in a high-risk ICU population receiving high-dose vasopressors, with quantified associations between hemodynamic and metabolic parameters and kidney injury development.

**Table 1.** Demographic and Baseline Characteristics of Study Population

| Variable | AKI Group (n = 218) | Non-AKI Group (n = 282) | p-value |
|---|---|---|---|
| Age (years) | 64.5 ± 12.3 | 57.8 ± 11.7 | <0.001 |
| Male gender, n (%) | 142 (65.1%) | 166 (58.9%) | 0.17 |
| Hypertension, n (%) | 130 (59.6%) | 120 (42.6%) | 0.002 |
| Diabetes mellitus, n (%) | 98 (45.0%) | 84 (29.8%) | 0.001 |
| APACHE II score (mean ± SD) | 25.6 ± 7.4 | 19.2 ± 6.1 | <0.001 |
| SOFA score (mean ± SD) | 11.8 ± 4.2 | 8.3 ± 3.5 | <0.001 |

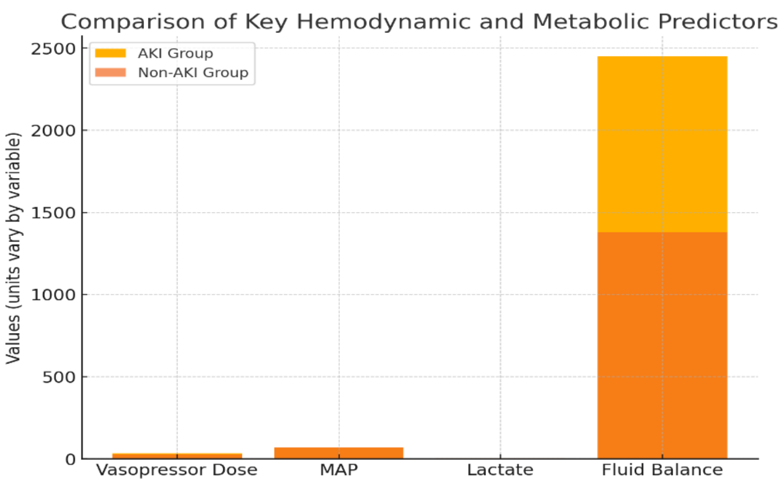
**Table 2.** Comparison of Hemodynamic and Metabolic Parameters Between AKI and Non-AKI Groups

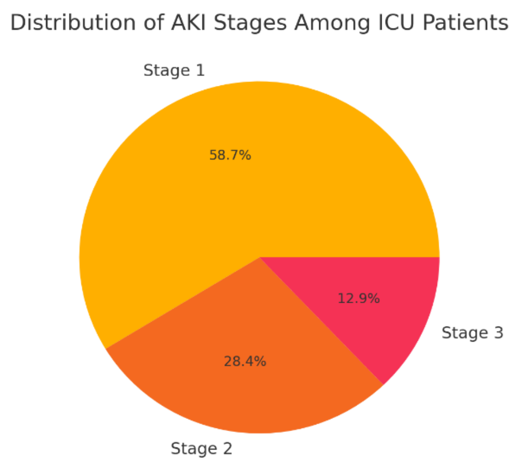
| Variable | AKI Group (n = 218) | Non-AKI Group (n = 282) | p-value |
|---|---|---|---|
| Vasopressor dose (µg/kg/min) | 35.2 ± 9.8 | 27.1 ± 8.3 | <0.001 |
| Mean arterial pressure (mmHg) | 68.9 ± 5.2 | 71.4 ± 4.7 | <0.01 |

| | | | |
|---|---|---|---|
| Admission lactate (mmol/L) | 3.8 ± 1.6 | 2.9 ± 1.2 | <0.001 |
| Cumulative fluid balance (mL, day 3) | +2450 ± 900 | +1380 ± 760 | <0.001 |
| AKI incidence, n (%) | 218 (43.6%) | — | — |

**Table 3.** Multivariate Logistic Regression Analysis for Predictors of AKI

| **Predictor** | **Adjusted OR** | **95% CI** | **p-value** |
|---|---|---|---|
| Vasopressor dose (per 10 µg/kg/min increase) | 1.48 | 1.22–1.80 | <0.001 |
| Mean arterial pressure (per 1 mmHg increase) | 0.87 | 0.80–0.94 | 0.002 |
| Admission lactate ≥4 mmol/L | 2.35 | 1.40–3.93 | 0.001 |
| Positive fluid balance ≥2 L (day 3) | 1.91 | 1.28–2.85 | 0.001 |




## DISCUSSION

The findings of this observational cohort study illuminated the substantial incidence of acute kidney injury (AKI) among critically ill patients receiving high-dose vasopressors in the ICU, and identified hemodynamic and metabolic predictors that were independently associated with AKI onset. Specifically, higher vasopressor doses, lower achieved mean arterial pressures (MAP), elevated admission lactate levels, and more positive cumulative fluid balance were significantly predictive of AKI development. These results are consistent with emerging evidence underscoring the multifactorial pathogenesis of AKI in critical illness, in which hemodynamic instability, tissue hypoperfusion, and fluid overload collectively contribute to renal dysfunction (14-17). This study's identification of vasopressor dose as an independent predictor aligns with prior observational research demonstrating that higher catecholamine requirements often mirror worsening circulatory shock and microcirculatory disturbances, both of which increase the risk of AKI (16). While vasopressors are essential for maintaining perfusion pressure in shock states, their association with AKI likely reflects the severity of underlying illness rather than a direct nephrotoxic effect. Analogous analyses have shown that increasing doses of norepinephrine and vasopressin correlated with adverse outcomes and mortality in AKI populations, emphasizing the complex interplay between vasopressor support, illness severity, and organ dysfunction (16).

The association between MAP targets and AKI observed in this cohort suggests that even modest differences in perfusion pressure can influence renal outcomes. Although definitive randomized evidence on optimal MAP targets is limited, several clinical studies and guideline recommendations have advocated maintaining MAP above 65 mmHg as a minimum threshold in septic shock, with higher targets under investigation in subpopulations at risk of renal hypoperfusion (17,18). The present findings therefore reinforce the concept that hemodynamic optimization with individualized blood pressure goals may be relevant for AKI prevention, particularly among

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patients with preexisting hypertension or chronic vascular disease. Lactate levels at admission were also significant predictors in this study, consistent with lactate's role as a surrogate marker of global tissue hypoxia and metabolic stress. Elevated lactate has been associated with worse outcomes in critically ill patients, including AKI and death, by indicating inadequate perfusion at the microvascular level (19). By capturing the severity and persistence of systemic hypoperfusion, lactate trends likely reflect the extent of shock and organ perfusion deficits that predispose patients to renal injury.

The relationship between fluid balance and AKI corroborates a growing body of evidence indicating that positive fluid accumulation is harmful in critically ill patients with renal dysfunction. Contemporary cohort analyses have shown that progressive positive fluid balance commonly develops after AKI diagnosis and is independently associated with increased mortality (20). This supports the notion that although early fluid resuscitation is vital for restoring intravascular volume, excessive cumulative fluid balance can lead to interstitial edema, elevated renal venous pressure, and impaired renal perfusion, thereby exacerbating AKI risk (21). The present results advance this understanding by quantifying fluid accumulation within the critical early days of ICU admission and linking it to AKI incidence. The strengths of this work include its prospective design, rigorous data collection with standard definitions (KDIGO criteria) for AKI diagnosis, and comprehensive hemodynamic and metabolic profiling that enabled the evaluation of multiple predictors in multivariate models. These methodological attributes support the internal validity of the findings and provide a robust framework for replication in other settings. Moreover, the inclusion of clinically interpretable predictors such as vasopressor dose, MAP, lactate, and fluid balance enhances the translational potential of the results for ICU clinicians.

Despite these strengths, several limitations merit careful consideration. As an observational study, the results are subject to residual confounding by unmeasured factors, including variations in resuscitation strategies, differences in clinician practice patterns, and underlying comorbidities. Although multivariate analysis was used to adjust for measured confounders, causality cannot be inferred, and the predictive associations observed require validation in independent cohorts. Second, the study did not evaluate long-term renal outcomes beyond ICU discharge, such as progression to chronic kidney disease or dialysis dependence, which are important patient-centered endpoints. Third, although lactate and fluid balance were measured longitudinally, temporal confounding remains a possibility given the dynamic nature of critical illness. Future research should explore interventional studies that tailor hemodynamic support and fluid management based on individualized risk profiles to assess whether modifying these predictors can reduce AKI incidence. Randomized controlled trials testing MAP targets in specific subgroups (e.g., chronic hypertensive patients) or fluid management protocols that minimize cumulative positive balance could yield insights into causal pathways and therapeutic strategies (18,21). Additionally, integrating biomarker and machine learning approaches may enhance early AKI risk stratification and support personalized critical care pathways.

This study adds to the growing literature demonstrating that hemodynamic severity and fluid accumulation are key determinants of AKI in the ICU. By highlighting vasopressor requirement, MAP, lactate trends, and fluid balance as significant predictors, the findings provide a clinically relevant basis for refining hemodynamic and fluid management strategies aiming to prevent renal injury in critically ill patients. Continued research into mechanistic pathways and targeted interventions will be crucial to improve outcomes for this vulnerable population.

## CONCLUSION

This study demonstrated that acute kidney injury is highly prevalent among ICU patients receiving high-dose vasopressors and is strongly associated with hemodynamic and metabolic factors, including vasopressor dose, mean arterial pressure, lactate levels, and cumulative fluid balance. These findings emphasize the need for individualized hemodynamic optimization and judicious fluid management to minimize renal complications in critically ill patients. By identifying modifiable predictors, this research provides a foundation for developing targeted strategies to prevent AKI and improve survival outcomes in intensive care settings.

**AUTHOR CONTRIBUTION**

| Author | Contribution |
|---|---|
| Dr Wafa Hyder Maitlo | Conceptualization, Methodology, Formal Analysis, Writing - Original Draft, Validation, Supervision |
| Dr Arif Mehmood Kamboh | Methodology, Investigation, Data Curation, Writing - Review & Editing |

 

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| Dr Ali Nisar Ayub | Investigation, Data Curation, Formal Analysis, Software |
| Dr Asim Rafiq | Software, Validation, Writing - Original Draft |
| Dr Nauman Khan | Formal Analysis, Writing - Review & Editing |
| Dr Fazeel | Writing - Review & Editing, Assistance with Data Curation |

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