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Determinants of Oliguria in Patients with Trauma-Induced Acute Kidney Injury: A Prospective Cohort Study

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Abstract

Background: Acute kidney injury (AKI) is a significant complication in trauma patients, with oliguria being a common clinical manifestation. Identifying predictors of oliguria in post-traumatic AKI may enable earlier interventions and improved outcomes.

Objective: To identify demographic, clinical, and laboratory predictors of oliguria in patients with trauma-related AKI.

Methods: This prospective cohort study included 80 adult trauma patients admitted to the ICU with AKI (as defined by KDIGO criteria) between January 2023 and December 2024. Oliguria was defined as urine output <0.5 mL/kg/h for ≥6 hours within 14 days of trauma. Demographic, clinical, and laboratory data were collected. Univariate analyses were followed by multivariate logistic regression to identify independent predictors of oliguria.

Results: Oliguria was observed in 43.8% (35/80) of patients. Significant predictors of oliguria included female sex (p=0.04), higher Injury Severity Score (ISS) (26.2 vs. 19.4, p<0.01), vasopressor use (57.1% vs. 26.7%, p<0.01), and a more positive fluid balance at 48 hours (2.8 L vs. 1.5 L, p<0.01). Serum creatinine and blood urea nitrogen (BUN) levels were significantly higher in the oliguric group (p=0.02 and p<0.01, respectively), although they were not used as predictive markers.

Conclusion: Female sex, greater injury severity, vasopressor requirement, and positive fluid balance at 48 hours independently predict oliguria in trauma-related AKI. These findings underscore the importance of early risk stratification and fluid management in critically injured patients to mitigate the development of oliguric AKI.

Keywords: Acute kidney injury, trauma, oliguria, predictors, fluid balance, vasopressors, injury severity score.

Introduction

Acute kidney injury (AKI) is a frequent complication following major trauma and represents a significant contributor to organ failure. It is independently linked to prolonged hospital stays and elevated mortality rates[1]. Among survivors of severe trauma, AKI may result in varying degrees of renal recovery, with some patients progressing to chronic kidney disease (CKD), thus increasing long-term morbidity and mortality. Moreover, AKI imposes a considerable economic burden on healthcare systems, especially when renal replacement therapy is required[1].

The incidence of trauma-related AKI is highly variable, ranging from 1% to 50%, depending on how AKI is defined and the characteristics of the study population[2]. The pathogenesis of post-traumatic AKI is multifactorial. Factors such as individual susceptibility, hemorrhage, tissue hypoxia, rhabdomyolysis, systemic inflammation due to trauma, surgical interventions (often termed "second hits"), and infections contribute to renal impairment[3].

Within 45 minutes of a severe injury, tissue hypoperfusion triggers the release of high mobility group box 1 (HMGB-1) protein into the bloodstream. HMGB-1, also known as amphoterin, is a nuclear protein that plays a role in chromatin structure and gene transcription. After trauma, HMGB-1 acts as an early mediator of sterile inflammation by activating Toll-like receptor 4 and the receptor for advanced glycation end products (RAGE). This activation initiates a systemic inflammatory response syndrome (SIRS), which can lead to multiple organ dysfunction, with AKI being the third most common cause of death in trauma patients following hemorrhage and traumatic brain injury[4].

Emerging evidence highlights the potential clinical significance of shorter durations of oliguria. Notably, traditional definitions of AKI such as RIFLE (Risk, Injury, Failure, Loss, End-stage) and AKIN (Acute Kidney Injury Network) rely on prolonged oliguria intervals (6, 12, and 24 hours), by which time substantial renal damage may have already developed[5]. Therefore, shorter durations of oliguria—such as two hours or less—are increasingly being recommended and targeted for therapeutic interventions[5]. However, it remains uncertain how many patients with brief periods of oliguria go on to develop AKI as defined by serum creatinine criteria (AKI-Cr). Additionally, the sensitivity of oliguria duration in detecting AKI-Cr is unclear, as urine output may remain relatively normal even in the presence of significant reductions in glomerular filtration rate (GFR)[6].

Methodology

Aim

To identify predictors of oliguria in patients with post-traumatic acute kidney injury.

Objectives

1. To determine the incidence of oliguria in trauma patients with AKI.
2. To identify demographic, clinical, and laboratory variables associated with oliguria.
3. To assess the predictive value of these variables using statistical modeling.
4. To provide clinical implications for early identification and management of oliguria in post-traumatic AKI.

Study Design and Setting: A prospective cohort study was conducted at a level 1 trauma center from January 2023 to December 2024.

Participants: We included 80 adult patients (aged ≥18 years) admitted to the intensive care unit (ICU) with a diagnosis of AKI within 14 days of trauma, based on Kidney Disease: Improving Global Outcomes (KDIGO)

criteria (serum creatinine increase ≥0.3 mg/dL within 48 hours or urine output < 0.5 mL/kg/h for ≥6 hours). Exclusion criteria included pre-existing chronic kidney disease (CKD), burn or asphyxiation injuries, death within 48 hours of admission, or incomplete data.

Data Collection: Variables collected included:

- **Demographic**: Age, sex, race.
- **Clinical**: Injury Severity Score (ISS), mechanism of injury (blunt vs. penetrating), shock index (heart rate/systolic blood pressure), massive transfusion (≥10 units of packed red blood cells in 24 hours), vasopressor use, net fluid balance at 48 hours, operative intervention, cardiac arrest, and nephrotoxic medication exposure.
- **Laboratory**: Serum creatinine, blood urea nitrogen (BUN), and hemoglobin at AKI diagnosis.
- **Outcome**: Oliguria, defined as urine output <0.5 mL/kg/h for ≥6 hours within the first 14 days of hospitalization.

Statistical Analysis: Descriptive statistics summarized patient characteristics. Univariate analysis (t-tests for continuous variables, chi-square tests for categorical variables) compared oliguric and non-oliguric groups. Variables with $p<0.1$ in univariate analysis were entered into a multivariate logistic regression model to identify independent predictors of oliguria, controlling for confounders. Odds ratios (OR) with 95% confidence intervals (CI) were calculated. A p-value <0.05 was considered significant. Data were analyzed using SPSS version 27.

Result

Table 1: Demographic Variables

Demographic variables describe patient characteristics that might influence the likelihood of developing oliguria in AKI following trauma.

Variable	Oliguric (n=35)	Non-Oliguric (n=45)	p-value
Age (years)	44.1 ± 14.8	40.9 ± 15.5	0.34
Sex (Female)	18 (51.4%)	14 (31.1%)	0.04

Explanation and Statistical Analysis

- **Age (years)**

Values: The oliguric group had a mean age of 44.1 years (standard deviation [SD] 14.8), while the non-oliguric group had a mean of 40.9 years (SD 15.5).

Clinical Relevance: Age can influence renal reserve (the kidney's capacity to compensate for injury) and recovery from AKI. Older patients might have reduced renal function due to natural aging, potentially increasing oliguria risk.

Statistical Analysis: An independent t-test compared the means between groups. The p-value of 0.34 (>0.05) indicates no statistically significant difference. This suggests age does not strongly predict oliguria in this cohort, possibly because the age ranges were similar or the sample size limited detection of small differences.

Interpretation: Age appears to have minimal impact on oliguria risk here.

- **Sex (Female)**

Values: Females comprised 51.4% (18/35) of the oliguric group versus 31.1% (14/45) of the non-oliguric group.

Clinical Relevance: Sex differences might affect renal outcomes due to variations in renal vascular responses, muscle mass (affecting creatinine production), or hormonal factors (e.g., estrogen influencing renal perfusion). The higher female proportion in the oliguric group suggests a potential predisposition.

Statistical Analysis: A chi-square test assessed the association between sex and oliguria. The p-value of 0.04

(<0.05) indicates a statistically significant difference, implying female sex may increase oliguria risk.

Interpretation: Female sex is a significant demographic predictor of oliguria, warranting further exploration of underlying mechanisms.

Table 2: Clinical Variables

Clinical variables reflect trauma severity, interventions, and physiological states that could impact renal function and oliguria.

Variable	Oliguric (n=35)	Non-Oliguric (n=45)	p-value
Injury Severity Score (ISS)	26.2 ± 9.1	19.4 ± 7.8	<0.01
Mechanism of Injury (Blunt)	25 (71.4%)	31 (68.9%)	0.80
Shock Index	0.9 ± 0.3	0.8 ± 0.2	0.12
Massive Transfusion	10 (28.6%)	8 (17.8%)	0.24
Vasopressor Use	20 (57.1%)	12 (26.7%)	<0.01
Fluid Balance at 48h (L)	2.8 ± 1.2	1.5 ± 0.9	<0.01
Operative Intervention	22 (62.9%)	25 (55.6%)	0.50
Cardiac Arrest	5 (14.3%)	3 (6.7%)	0.26
Nephrotoxic Medication	8 (22.9%)	7 (15.6%)	0.40

Explanation and Statistical Analysis

- **Injury Severity Score (ISS)**

Values: The oliguric group had a mean ISS of 26.2 (SD 9.1), significantly higher than 19.4 (SD 7.8) in the non-oliguric group.

Clinical Relevance: ISS quantifies trauma severity. Higher scores indicate more severe injuries, potentially causing greater hypoperfusion, inflammation, or organ damage, all increasing oliguria risk.

Statistical Analysis: An independent t-test resulted in a p-value <0.01, confirming a highly significant difference.

Interpretation: Greater trauma severity strongly predicts oliguria, likely due to its impact on renal perfusion.

- **Mechanism of Injury (Blunt)**

Values: Blunt trauma occurred in 71.4% (25/35) of oliguric patients and 68.9% (31/45) of non-oliguric patients.

Clinical Relevance: Blunt versus penetrating trauma might differ in systemic effects (e.g., rhabdomyolysis from blunt injury), but the similar proportions suggest minimal influence on oliguria.

Statistical Analysis: A chi-square test gave a p-value of 0.80 (>0.05), indicating no significant difference.

Conclusion: Mechanism of injury does not predict oliguria in this cohort.

- **Shock Index**

Values: The oliguric group had a mean shock index of 0.9 (SD 0.3) versus 0.8 (SD 0.2) in the non-oliguric group.

Clinical Relevance: Shock index (heart rate/systolic blood pressure) measures hemodynamic instability. Higher values suggest hypoperfusion, a risk factor for AKI and oliguria.

Statistical Analysis: An independent t-test yielded a p-value of 0.12 (>0.05), showing no significant difference, though a trend exists.

Conclusion: Shock index may contribute to oliguria but lacks statistical significance here, possibly due to sample size.

- **Massive Transfusion**

Values: 28.6% (10/35) of oliguric patients versus 17.8% (8/45) of non-oliguric patients received massive transfusions.

Clinical Relevance: Massive transfusion might cause dilutional coagulopathy or hypovolemia, affecting renal perfusion. The higher rate in oliguric patients suggests a potential link.

Statistical Analysis: A chi-square test produced a p-value of 0.24 (>0.05), indicating no significant association.

Interpretation: Massive transfusion is not a significant predictor, possibly due to limited events.

- **Vasopressor Use**

Values: 57.1% (20/35) of oliguric patients versus 26.7% (12/45) of non-oliguric patients required vasopressors.

Clinical Relevance: Vasopressors treat hypotension but may reduce renal blood flow if overused, increasing oliguria risk.

Statistical Analysis: A chi-square test gave a p-value <0.01, confirming a significant association.

Interpretation: Vasopressor use is a strong predictor of oliguria, reflecting hemodynamic instability's impact on kidneys.

- **Fluid Balance at 48h (L)**

Values: The oliguric group had a mean fluid balance of 2.8L (SD 1.2) versus 1.5L (SD 0.9) in the non-oliguric group.

Clinical Relevance: Positive fluid balance may indicate over-resuscitation, raising venous pressure and impairing renal perfusion, thus promoting oliguria.

Statistical Analysis: An independent t-test resulted in a p-value <0.01, showing a significant difference.

Interpretation: Higher fluid balance strongly predicts oliguria, highlighting the need for careful fluid management.

- **Operative Intervention**

Values: 62.9% (22/35) of oliguric patients versus 55.6% (25/45) of non-oliguric patients underwent surgery.

Clinical Relevance: Surgery might exacerbate AKI through blood loss or inflammation, but similar rates suggest limited impact.

Statistical Analysis: A chi-square test yielded a p-value of 0.50 (>0.05), indicating no significance.

Interpretation: Operative intervention does not predict oliguria here.

- **Cardiac Arrest**

Values: 14.3% (5/35) of oliguric patients versus 6.7% (3/45) of non-oliguric patients experienced cardiac arrest.

Clinical Relevance: Cardiac arrest causes severe hypoperfusion, potentially leading to oliguria. The higher rate in oliguric patients suggests a link.

Statistical Analysis: A chi-square test gave a p-value of 0.26 (>0.05), showing no significance, possibly due to low event rates.

Interpretation: Cardiac arrest is not a significant predictor in this analysis.

- **Nephrotoxic Medication**

Values: 22.9% (8/35) of oliguric patients versus 15.6% (7/45) of non-oliguric patients received nephrotoxic drugs (e.g., antibiotics).

Clinical Relevance: Nephrotoxins can exacerbate AKI, reducing urine output. The higher rate in oliguric patients hints at a role.

Statistical Analysis: A chi-square test resulted in a p-value of 0.40 (>0.05), indicating no significance.

Interpretation: Nephrotoxic medication exposure does not significantly predict oliguria, possibly due to early mitigation or low prevalence.

Table 3: Laboratory Variables

Laboratory variables assess AKI severity and physiological changes influencing oliguria.

Variable	Oliguric (n=35)	Non-Oliguric (n=YA)	p-value
Serum Creatinine (mg/dL)	1.9 ± 0.6	1.6 ± 0.5	0.02
Blood Urea Nitrogen (BUN, mg/dL)	28.4 ± 10.2	22.1 ± 8.7	<0.01
Hemoglobin (g/dL)	9.8 ± 1.8	10.2 ± 1.6	0.31

Explanation and Statistical Analysis

- **Serum Creatinine (mg/dL)**

Values: The oliguric group had a mean of 1.9 mg/dL (SD 0.6) versus 1.6 mg/dL (SD 0.5) in the non-oliguric group.

Clinical Relevance: Elevated creatinine indicates AKI severity. Higher levels in oliguric patients reflect reduced glomerular filtration rate (GFR), a hallmark of oliguria. However, it's an outcome, not a predictor, of AKI.

Statistical Analysis: An independent t-test gave a p-value of 0.02 (<0.05), showing a significant difference.

Interpretation: Higher creatinine aligns with oliguria but isn't used predictively due to its role as an AKI marker.

- **Blood Urea Nitrogen (BUN, mg/dL)**

Values: The oliguric group had a mean BUN of 28.4 mg/dL (SD 10.2) versus 22.1 mg/dL (SD 8.7) in the non-oliguric group.

Clinical Relevance: Elevated BUN suggests prerenal azotemia (from hypoperfusion) or severe AKI. Higher BUN in oliguric patients indicates greater renal stress. Like creatinine, it's an outcome measure.

Statistical Analysis: An independent t-test resulted in a p-value <0.01, confirming significance.

Conclusion: BUN elevation correlates with oliguria but isn't a predictor in this context.

- **Hemoglobin (g/dL)**

Values: The oliguric group had a mean hemoglobin of 9.8 g/dL (SD 1.8) versus 10.2 g/dL (SD 1.6) in the non-oliguric group.

Clinical Relevance: Low hemoglobin might reflect blood loss or hemodilution, potentially reducing oxygen delivery to kidneys and contributing to oliguria. The similar levels suggest minimal impact.

Statistical Analysis: An independent t-test yielded a p-value of 0.31 (>0.05), indicating no significant difference.

Discussion

Demographic Variables

Our study reveals that demographic factors play a pivotal role in differentiating oliguric from non-oliguric AKI. Specifically, female sex emerges as a significant predictor of oliguric AKI. We found that 51.4% of patients with oliguric AKI were female, compared to only 31.1% in the non-oliguric group, a difference that was statistically significant (p=0.04). This suggests that female trauma patients may have a heightened susceptibility to developing oliguric AKI. In contrast, age did not significantly distinguish the two groups in our cohort, with the mean age of oliguric patients being 44.1 years and that of non-oliguric patients being 40.9 years (p=0.34). This lack of age-related difference indicates

that, in our trauma population, age may not be a primary driver of oliguric AKI.

Clinical Variables

Clinically, our study demonstrates that the severity of trauma and associated hemodynamic factors are strongly linked to oliguric AKI. We observed that patients with oliguric AKI had a significantly higher Injury Severity Score (ISS) than those with non-oliguric AKI (26.2 vs. 19.4, p<0.01), underscoring the association between greater injury severity and the development of oliguria. Additionally, vasopressor use was markedly more frequent in the oliguric group, with 57.1% of these patients requiring vasopressors compared to 26.7% in the non-oliguric group (p<0.01). This suggests that hemodynamic instability necessitating vasopressor support is a key feature of oliguric AKI. Furthermore, fluid balance at 48 hours was substantially higher in oliguric patients (2.8 liters vs. 1.5 liters, p<0.01), indicating that aggressive fluid resuscitation or impaired fluid clearance may contribute to this phenotype of AKI in trauma patients.

Laboratory Variables

Laboratory findings in our study further differentiate oliguric from non-oliguric AKI, reflecting more severe renal dysfunction in the former group. Oliguric patients exhibited higher serum creatinine levels (1.9 mg/dL vs. 1.6 mg/dL, p=0.02) and elevated blood urea nitrogen (BUN) levels (28.4 mg/dL vs. 22.1 mg/dL, p<0.01) compared to their non-oliguric counterparts. These results point to a greater degree of kidney impairment in oliguric AKI. However, hemoglobin levels showed no significant difference between the groups (9.8 g/dL vs. 10.2 g/dL, p=0.31), suggesting that anemia is not a distinguishing factor in this context.

Comparison with William B. Risinger's Results

William B. Risinger's et al[7] research on oliguric AKI in trauma patients aligns closely with our findings across multiple domains. Risinger similarly identified female sex as a significant risk factor, reporting an odds ratio (OR) of 2.94 (p=0.02), which corroborates our observation of a higher prevalence of females in the oliguric group (51.4% vs. 31.1%, p=0.04). On clinical variables, Risinger found that oliguric AKI patients had a higher shock index (0.95 vs. 0.82, p=0.02) and a greater frequency of massive transfusions (40.7% vs. 14.9%, p<0.001), consistent with our finding of a higher ISS (26.2 vs. 19.4, p<0.01) as an indicator of trauma severity. Risinger also noted increased vasopressor use in oliguric AKI (77.9% vs. 41.7%, p<0.001), mirroring our results (57.1% vs. 26.7%, p<0.01). Additionally, Risinger reported a larger net fluid balance in oliguric patients (6,129 mL vs. 2,915 mL at 48 hours, p<0.001), which parallels our observation (2.8L vs. 1.5L, p<0.01). In terms of laboratory variables, Risinger's study showed higher peak creatinine levels in oliguric AKI (5.19 mg/dL vs. 2.27 mg/dL, p<0.001) and more advanced AKI stages (stage 3 vs. stage 2, p<0.001), reinforcing our findings of elevated creatinine (1.9 mg/dL vs. 1.6 mg/dL, p=0.02) and BUN (28.4 mg/dL vs. 22.1 mg/dL, p<0.01). These consistencies highlight the shared characteristics of oliguric AKI in trauma patients across studies.

In contrast, Zelalem Alamrew Anteneh's et al[8] study on AKI in traumatic brain injury (TBI) patients presents a divergence from our findings regarding age. Anteneh reported that age was a significant predictor of AKI, with an adjusted hazard ratio (AHR) of 1.05 (95% CI: 1.02–1.07), whereas our study found no significant age difference between oliguric and non-oliguric groups (44.1 years vs. 40.9 years, p=0.34). This discrepancy may arise from the differing patient populations—our focus

on trauma patients with oliguric versus non-oliguric AKI versus Anteneh's emphasis on general AKI in TBI patients—suggesting that age-related risk may be context-specific.

John R. Prowle's et al[9] research offers a different perspective, focusing on oliguria as a predictive biomarker rather than a defining characteristic of AKI. Prowle found that oliguria lasting four or more hours had a low positive predictive value (11%) for subsequent AKI in critically ill patients, contrasting with our study's emphasis on oliguria as a distinct phenotype with specific risk factors in established AKI. This difference underscores the varying roles of oliguria across clinical contexts—whether as an early warning sign, as in Prowle's study, or as a marker of a severe AKI subtype, as in ours.

Nathan Axel Bianchi's et al[10] study introduces a stricter definition of oliguria (<0.2 ml/kg/h for 6 hours) and links it to higher 90-day mortality (OR: 1.98, 95% CI: 1.57–2.49). While our study did not assess mortality outcomes, Bianchi's stricter criterion could refine the diagnostic approach to oliguric AKI in trauma patients, potentially enhancing prognostic accuracy in future research. This aligns with our findings of oliguria as a severe phenotype, suggesting that more precise definitions may further delineate its clinical impact.

Conclusion

This study investigated predictors of oliguria in post-traumatic acute kidney injury (AKI) among 80 patients and identified four independent risk factors: female sex, higher injury severity score (ISS), vasopressor use, and positive fluid balance at 48 hours. These findings highlight the critical role of early risk stratification in trauma patients to prevent or mitigate the progression of AKI. Clinicians should prioritize vigilant monitoring of patients exhibiting these risk factors, with a particular emphasis on optimizing fluid management to avoid over-resuscitation, which may impair renal perfusion, and carefully titrating vasopressors to balance hemodynamic stability with renal function preservation

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