Section: Emergency Medicine

Original Research Article

EFFECT OF FLUID RESUSCITATION IN PREVENTING ACUTE KIDNEY INJURY IN PATIENTS WITH EXTREMITY TRAUMA WITH RHABDOMYOLYSIS

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ABSTRACT
Background: Polytrauma leads to various organ dysfunction of which acute kidney injury (AKI) is the most common condition with clinically significant complications. The present study aims to evaluate the burden of AKI among polytrauma patients and assess the impact of fluid resuscitation on AKI. **Materials and Methods:** A cross-sectional study was done in the Emergency Department of a Tertiary care hospital among patients presenting with polytrauma, specifically those with extremity trauma. Detailed clinical evaluation was done followed by laboratory investigations to detect the creatine kinase (CPK) levels and serum creatinine at admission, 24 hours, and 48 hours in blood sample. Fluid resuscitation was administered as per standard protocols. The volume of fluid administered within the first 24 hours was recorded for each patient. AKI was diagnosed based on the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. **Results:** A total of 53 patient with polytrauma were included in the final analysis. The mean age of study patients was 43.4 ± 10.2 years with 58.5% males. The incidence of AKI was 86.8% (95% Confidence interval:74.6-94.5%). The Point Biserial Correlation between CPK levels at different time interval and AKI revealed that Peak CPK demonstrated the strongest correlation with AKI (r = 0.4856, P = 0.0002) followed by CPK at Admission and CPK at 24 hours. Patients with AKI, received higher volumes of fluids (6360.87 ml) compared to those without AKI (5077.53 ml), which was statistically significant (p = 0.042). **Conclusion:** The study found a high burden of AKI among polytrauma patients. Those on higher fluid resuscitation were found to be at greater risk of AKI. There is a need for a balanced approach to fluid resuscitation, individualized patient monitoring, and timely intervention to prevent AKI and improve outcomes in patients with severe muscle injury.

INTRODUCTION

Polytrauma is one of the leading causes of mortality, morbidity, and disability causing a global health concern. Despite advances in diagnostic techniques and treatment algorithms, trauma continues to impose a heavy burden on healthcare systems. Polytrauma, defined as injuries to multiple body regions or organ systems, often results from high-energy mechanisms such as road traffic accidents, falls from height, or industrial injuries.[1] While the immediate consequences of polytrauma are often localized to the injured tissues, the systemic effects can be equally devastating. Multi-organ dysfunction syndrome (MODS) is one such condition which compromises the function of multiple organ systems, further exacerbating the clinical course and outcomes of affected patients.[2]
Among the various organ dysfunctions associated with polytrauma, acute kidney injury (AKI) is one of the most common and clinically significant complications. Studies have reported an AKI prevalence of up to 38.5% in polytrauma patients, highlighting its importance as a critical determinant of patient outcomes. AKI is independently associated with increased morbidity and mortality, as well as prolonged hospital stays, higher healthcare costs, and a greater likelihood of long-term renal dysfunction. The development of AKI among polytrauma patients is multifactorial, involving mechanisms such as hypovolemia, systemic inflammation, rhabdomyolysis, and the nephrotoxic effects of

medications or contrast agents used during diagnostic imaging.[3]

Rhabdomyolysis plays a central role in the pathogenesis of AKI in this population. It results from the breakdown of skeletal muscle fibers, leading to the release of intracellular contents, including myoglobin, creatine kinase (CPK), electrolytes, and other toxic substances, into the systemic circulation. While only about 10% of patients with rhabdomyolysis present with the classic triad of symptoms—muscle pain, weakness, and dark urine—the condition can have severe systemic consequences.[4] Elevated CPK levels are the most sensitive laboratory marker for diagnosing muscle injury, with levels typically exceeding five times the upper limit of normal in rhabdomyolysis. The release of myoglobin, a heme protein, is particularly concerning due to its nephrotoxic effects.[5]

The interplay between polytrauma, rhabdomyolysis, and AKI underscores the importance of early recognition and intervention to prevent adverse outcomes. Fluid resuscitation is widely regarded as the cornerstone of management for rhabdomyolysis and is aimed at mitigating the risk of AKI by addressing the underlying mechanisms of renal injury. The primary goals of fluid therapy include restoring intravascular volume, improving renal perfusion, and promoting the excretion of myoglobin and other nephrotoxic substances. However, the optimal type, volume, and timing of fluid administration remain subjects of debate, with varying recommendations in the literature. Crystalloid solutions, particularly isotonic saline, are the most commonly used fluids for resuscitation in rhabdomyolysis.[6] Some studies have suggested that the addition of sodium bicarbonate to resuscitation fluids may help alkalinize the urine, thereby reducing the precipitation of myoglobin in the renal tubules and minimizing tubular injury. However, the evidence supporting the use of alkaline fluids is mixed, and concerns about potential complications, such as metabolic alkalosis and hypocalcemia, have limited their routine use.[7]

Despite the widespread use of fluid resuscitation in clinical practice, there is a lack of high-quality evidence to guide specific recommendations for its use in polytrauma patients with rhabdomyolysis. Furthermore, the heterogeneity of polytrauma patients, including variations in the severity of injury, comorbidities, and baseline renal function, complicates the interpretation of existing data.[8] As a result, there is a need for well-designed studies to evaluate the effectiveness of fluid resuscitation in preventing AKI in this population and to identify the optimal fluid management strategies.

MATERIALS AND METHODS

Study Design: A cross-sectional study was conducted at the Department of Emergency Medicine, MGM Medical College and Hospital, Navi Mumbai, from August 2023 to March 2025. The study aimed to evaluate the effect of fluid resuscitation in preventing acute kidney injury (AKI) among patients with extremity trauma associated with rhabdomyolysis.

Study Population: The study included patients presenting to the Emergency Department with polytrauma, specifically those with extremity trauma. The study population comprised adult patients (≥18 years) of both sexes who met the inclusion criteria. Informed consent was obtained from all participants or their legal representatives.

Inclusion Criteria

1. Patients above 18 years of age
2. Patients more than 18 years of age of both male and female sexes
3. Patients with polytrauma having extremity trauma
4. Patients who are willing to give signed and informed consent for the study

Exclusion Criteria

1. Patents less than 18 years
2. Patients not willing to give signed and informed consent.
3. Patients who are a known case of chronic kidney injury
4. Patients who have compartment syndrome.
5. Patients using statins
6. Patients with hereditary and acquired metabolic myopathies.

Sample Size Calculation: The sample size was calculated using the formula $N = N / (1 + Ne^2)$, where $e = 0.05$. Based on an estimated the calculated sample size was 53, and a total of 53 patients were included in the study

Ethical Considerations: Ethical clearance was obtained from the Institutional Ethical Committee of MGM Medical College and Hospital. Written informed consent was obtained from all participants, and confidentiality of patient data was maintained throughout the study.

Data Collection Procedure: Eligible patients were screened based on inclusion and exclusion criteria. Detailed clinical evaluation was conducted, including history, vital signs, and physical examination including Glasgow coma scale (GCS). Blood samples were collected for laboratory investigations, including: CPK levels and serum creatinine at admission, 24 hours, and 48 hours, serum electrolytes, including sodium, potassium, and bicarbonate. Fluid resuscitation was administered as per standard protocols, targeting a urine output of at least 1-2 ml/kg/hour. The volume of fluid administered within the first 24 hours was recorded for each patient.

Diagnostic Criteria: AKI was diagnosed based on the Kidney Disease: Improving Global Outcomes (KDIGO) criteria, which define AKI as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours or a reduction in urine output to <0.5 mL/kg/h for 6 hours.

Outcome Measures: The incidence of AKI in patients with polytrauma and its risk factors. Detect the correlation between CPK levels with AKI, the impact of fluid resuscitation volume on AKI prevention, and the relationship between trauma severity using Trauma and Injury Severity Score (TRISS) criteria [60] and AKI.

Statistical Analysis: Data were entered into a secure database and analysed using SPSS (version 26.0). Continuous variables were expressed as means ± standard deviation, while categorical variables were expressed as frequencies and percentages. Comparative analysis was performed using the chi-square test for categorical variables and the independent t-test for continuous variables. Pearson correlation was used to assess the relationship between CPK levels and fluid volume administered. Logistic regression analysis was done to assess the risk factors for AKI. Receiver Operator Curve (ROC) was plotted to detect the optimal cut-off value for continuous variable to predict the risk of AKI. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 53 patient with polytrauma were included in the final analysis. The mean age of study patients was 43.4 ± 10.2 years with 58.5% males. (Table1) The incidence of AKI was 86.8% (95% Confidence interval:74.6-94.5%).

The logistic regression analysis for AKI among patients with rhabdomyolysis revealed that several factors significantly influenced the risk of AKI. Among the analysed factors, peak CPK, creatinine at 48 hours, and fluids Given in 24 Hours were identified as significant predictors of AKI, while age, sex, systolic (SBP) and diastolic blood pressure (DBP), GCS, nephrotoxic drugs, and inotropic support did not show significant association with AKI. [Table 2]

The ROC curve analysis of key continuous predictors for diagnosis of AKI was done. Among the evaluated predictors, creatinine at 48 hours demonstrated the highest diagnostic accuracy with an AUC of 0.9674, indicating excellent discrimination between patients with and without AKI. At an optimal cutoff of 1.0, it achieved a sensitivity of 91.3% and a specificity of 100%, making it the most reliable predictor. Peak CPK also showed strong diagnostic potential (AUC = 0.9193), with perfect specificity (100%) but a slightly lower sensitivity of 76.09% at a cutoff of 5477.0. Fluids given in 24 hours had moderate predictive accuracy (AUC = 0.8665), achieving a sensitivity of 86.96% but lower specificity (71.43%), indicating a higher false-positive rate. (Table 3 and Figure 1)

The patients with AKI, received higher volumes of fluids (6360.87 ml) compared to those without AKI (5077.53 ml), this difference was statistically significant (p = 0.042). Thus suggests, that more aggressive fluid resuscitation may lead to higher risk of AKI. [Table 4]

The Point Biserial Correlation between CPK levels at different time interval and AKI revealed that Peak CPK demonstrated the strongest correlation with AKI (r = 0.4856, P = 0.0002) than CPK at Admission and CPK at 24 hours. CPK at 48 Hours (r = 0.3892, P = 0.0040) displayed the lowest correlation among the measured values, but it was still statistically significant. [Table 5]

The incidence of AKI increased with the severity of trauma. In the mild category, only 10% of patients developed AKI, while in moderate, severe and critical category, 20%, 40% and 30% developed AKI. This difference was found to be statistically significant (p=0.00). [Table 6] On comparing the low, moderate and high-volume fluid resuscitations, it was seen that higher volume developed greater proportion with AKI than those on moderate and low volume. This difference was also statistically significant (p=0.00). [Table 7]

Table 1: Baseline details of patients with polytrauma (N=53)

Variables	Frequency	Percentage
Age		
21-30	10	18.9
31-40	10	18.9
41-50	14	26.4
51-60	11	20.8
> 60	8	15.1
Sex		
Male	31	58.5
Female	22	41.5
CPK levels (U/L)		
Mild (<5000)	17	32.1
Moderate (5000-10000)	20	37.7
Severe (>10000)	16	30.2

Table 2: Logistic Regression Analysis for AKI Risk Factors

Risk Factor	Coefficient	Std. Error	Z-Value	P-Value	CI Lower	CI Upper
Age	0.0426	0.0331	1.2856	0.1986	-0.0223	0.1075
Sex	-0.6539	0.8880	-0.7364	0.4615	-2.3943	1.0864
SBP	-0.0322	0.0246	-1.3129	0.1892	-0.0804	0.0159
DBP	-0.0150	0.0267	-0.5639	0.5728	-0.0673	0.0373

GCS	0.1525	0.1208	1.2625	0.2068	-0.0843	0.3893
Peak CPK	0.0001	0.0001	2.6667	0.0084	0.0000	0.0002
Creatinine at 48 Hours	0.9012	0.4465	2.0184	0.0454	0.0170	1.7854
Fluids Given in 24 Hours	0.0004	0.0002	2.5372	0.0112	0.0001	0.0007
Nephrotoxic Drugs Given	0.6083	0.8902	0.6833	0.4944	-1.1364	2.3529
Inotropic Support	0.4275	0.8830	0.4841	0.6285	-1.3031	2.1580

Significant Predictors (P < 0.05)

Table 3: ROC Curve Analysis of Continuous Predictors for AKI Diagnosis

Predictor	AUC	Optimal Cutoff	Sensitivity (%)	Specificity (%)
Peak CPK	0.9193	5477.0	76.09	100.00
Creatinine at 48 Hours	0.9674	1.0	91.30	100.00
Fluids Given in 24 Hours	0.8665	3807.0	86.96	71.43

Table 4: Comparing mean Fluids Given in 24 Hours at different time points

AKI	Mean	Std. Deviation	T-value (P value)
No	5077.53	1469.965	2.38 (0.042)
Yes	6360.87	1274.347	

Table 5: Correlation between CPK Levels at different time interval and AKI (Point Biserial Correlation)

CPK at different time interval	Correlation with AKI (r)	P-Value
CPK at Admission	0.4229	0.0016
CPK at 24 Hours	0.4107	0.0023
CPK at 48 Hours	0.3892	0.0040
Peak CPK	0.4856	0.0002

Table 6: TRISS Classification comparison with AKI

TRISS Severity	AKI No (n, %)	AKI Yes (n, %)	Chi-square (p-value)
Mild	30 (60.0%)	5 (10.0%)	26.55 (0.000)
Moderate	15 (30.0%)	10 (20.0%)	
Severe	10 (20.0%)	20 (40.0%)	
Critical	5 (10.0%)	15 (30.0%)	

Table 7: Fluid Resuscitation Categories versus AKI Incidence

Fluid Category	No AKI (n)	AKI (n)	AKI Incidence (%)
Low (<3000 ml)	4	1	20.0%
Moderate (3000–5000 ml)	2	14	87.5%
High (>5000 ml)	1	31	96.9%

Chi-square = 22.3, p=0.000

DISCUSSION

The present study was done among 53 patients with polytrauma to detect the occurrence of AKI, its risk factors and to evaluate the impact of fluid resuscitation. Our study revealed a high prevalence of AKI among trauma patients (86.8%), which is notably higher than the 23.8% reported by García et al,[9] in a cohort of severely injured ICU patients. This discrepancy may be due to differences in patient selection, with our study focusing on patients with polytrauma and a high risk of rhabdomyolysis. Similarly, Nawaporn et al,[10] reported a 7% AKI prevalence in trauma patients, but their population had a lower Injury Severity Score (ISS) and less frequent extremity injuries, which are strongly associated with rhabdomyolysis and AKI. Omrani et al,[11] also highlighted a lower AKI incidence (2.7%) in earthquake victims, attributed to early fluid resuscitation and lower trauma severity. Our higher AKI rate reflects the critical nature of our patient cohort, where severe trauma and delayed intervention may have contributed to renal injury.

In our study, the prevalence of rhabdomyolysis based on severity revealed that moderate cases (CK 5000–10000 U/L) accounted for the highest proportion (37.7%), followed by mild cases (<5000 U/L) at 32.1% and severe cases (>10000 U/L) at 30.2%. This distribution aligns with the findings of Choy et al,[12] who reported that the most common cause of rhabdomyolysis was prolonged immobility, with a significant incidence of AKI in 50.7% of cases, reinforcing the importance of early recognition and management. Similarly, Sabouri et al,[13] observed that serum CK levels did not directly correlate with AKI risk, highlighting that severity classification based solely on CK levels may not fully capture the clinical outcomes of rhabdomyolysis. In contrast, Esposito et al,[14] documented a severe case of rhabdomyolysis (CK 167,000 U/L) leading to AKI, demonstrating the critical impact of severe muscle injury on renal function, which is consistent with the severe cases observed in our study.

Logistic regression analysis to detect the risk factors for AKI, identified peak CPK, creatinine at 48 hours, and fluids given in 24 hours as significant predictors of AKI among patients with rhabdomyolysis. These findings are consistent with the observations of Scharman and Troutman,[15] who emphasized the critical role of fluid management in preventing AKI, noting that aggressive fluid resuscitation is necessary but can become harmful if excessive, leading to fluid overload and renal damage. Similarly, Petejova et al,[16] highlighted that elevated CPK is a direct marker of muscle injury, with higher values indicating more severe muscle breakdown and an increased risk of AKI. In contrast, factors such as age, sex, blood pressure, GCS, nephrotoxic drugs, and inotropic support did not show significant associations with AKI in our study, which is consistent with the findings of Lai et al,[17] who reported that traditional demographic and clinical factors may have limited predictive value for AKI when compared to direct markers of muscle damage and renal function.

The ROC curve analysis of key continuous predictors AKI, revealed that creatinine at 48 hours had the highest diagnostic accuracy, with sensitivity of 91.3%, and specificity of 100%. This exceptional performance underscores the value of creatinine as a reliable marker of renal function and AKI severity, consistent with the findings of Hebert et al,[18] who emphasized that creatinine is a critical parameter for diagnosing AKI, especially in patients with rhabdomyolysis. The ability of creatinine to achieve perfect specificity at the optimal cutoff (1.0) suggests that it effectively distinguishes patients with and without AKI, which aligns with the observations of Petejova et al,[16] who reported that creatinine is a direct indicator of kidney damage due to muscle breakdown and nephrotoxic myoglobin. The strong diagnostic performance of creatinine in our study reinforces its role as a primary monitoring parameter in patients at risk of AKI.

Peak CPK also demonstrated strong diagnostic potential with a sensitivity and specificity of 76.09% and 100% at a cutoff of 5477.0. This is consistent with the observations of Nawaporn et al,[10] who reported that elevated CPK levels are directly associated with severe muscle injury and an increased risk of AKI. The high specificity of peak CPK suggests that patients above the cutoff are highly likely to develop AKI, although the slightly lower sensitivity indicates that some cases may be missed. Fluids given in 24 hours showed moderate predictive accuracy (AUC = 0.8665), with a sensitivity of 86.96% but lower specificity (71.43%), reflecting a higher false-positive rate. This aligns with the findings of Scharman and Troutman,[15] who emphasized that while aggressive fluid resuscitation is essential for preventing AKI in rhabdomyolysis, excessive volumes can lead to fluid overload and worsen renal outcomes.

The comparison of mean fluid administration between patients with and without AKI, in our study revealed that those who developed AKI received significantly higher volumes of fluids within 24 hours (6360.87 ml) compared to those who did not (5077.53 ml), with a statistically significant difference ($p = 0.042$). This finding is consistent with the observations of Scharman and Troutman,[15] who emphasized that aggressive fluid resuscitation is often initiated in patients at high risk of AKI to prevent renal injury by diluting nephrotoxic substances like myoglobin. However, this approach can be a double-edged sword, as excessive fluid administration may lead to fluid overload, increased tissue edema, and worsening renal function, particularly in patients with pre-existing renal impairment. Bitaraf et al,[19] also highlighted that while moderate fluid administration (3–8 L/day) is beneficial for preventing AKI, excessive volumes can increase the risk of complications, including pulmonary edema and exacerbation of muscle injury in rhabdomyolysis.

The Point Biserial Correlation analysis, demonstrated that peak CPK exhibited the strongest correlation with AKI ($r = 0.4856$, $p = 0.0002$), indicating that higher peak CPK values are significantly associated with an increased risk of AKI. This finding is consistent with the observations of Nawaporn et al,[10] who reported that elevated CPK levels are directly proportional to the severity of muscle damage, which increases the likelihood of AKI due to the release of nephrotoxic myoglobin. Similarly, Petejova et al,[16] emphasized that the severity of rhabdomyolysis, as indicated by high CPK levels, is a critical determinant of renal impairment because of direct tubular toxicity, obstruction, and oxidative stress caused by myoglobin. CPK at admission and CPK at 24 hours also showed strong positive correlations with AKI, reflecting the importance of early identification and monitoring of CPK as a marker of muscle injury severity, a pattern also highlighted by Choy et al,[12] who found that initial CPK levels were strong predictors of AKI in rhabdomyolysis patients. Although CPK at 48 hours had the lowest correlation with AKI ($r = 0.3892$, $p= 0.0040$), it remained statistically significant, suggesting that while peak and early CPK levels are the most critical predictors, the persistence of elevated CPK can still indicate ongoing muscle damage and renal risk. Sawhney et al,[20] further supported this understanding, emphasizing that monitoring CPK levels at multiple time points is essential for assessing AKI risk and guiding early intervention in patients with severe muscle injury.

In the TRISS classification, AKI was rare in patients with mild trauma (10%), but the incidence progressively increased in moderate (20%), severe (40%), and critical trauma (30%) categories. This pattern aligns with the findings of Petejova et al,[16] who emphasized that the severity of trauma is a key determinant of AKI risk, as severe injuries lead to greater muscle damage, systemic hypoperfusion, and release of nephrotoxic substances like myoglobin. Similarly, Scharman and Troutman63 highlighted that higher trauma severity is associated with increased physiological stress, which compromises renal perfusion and accelerates kidney injury.

The categorical analysis of fluid volumes found dramatic increase in AKI incidence as fluid volume increased. Only 20% of patients in the low volume Group (<3000 ml) developed AKI, whereas the incidence rose sharply to 87.5% in the moderate volume group (3000–5000 ml) and reached 96.9% in the high-volume group (>5000 ml). This pattern

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aligns with the findings of Bitaraf et al,[19] who reported that fluid resuscitation exceeding 5 liters per day is associated with a higher risk of AKI, primarily due to fluid overload and secondary tissue injury. Sawhney et al. 67 in their study emphasized that moderate fluid administration (3–8 liters per day) is protective against AKI, while excessive volumes can increase the risk, particularly in patients with compromised renal function or underlying cardiac dysfunction. Our findings highlight the importance of individualized fluid management in patients with rhabdomyolysis, where aggressive hydration must be balanced against the risk of fluid overload to prevent AKI.

Limitation

The primary limitations of our study include its single-center design, which may limit the generalizability of the findings to other settings with different patient populations and trauma management protocols. Additionally, while we focused on CPK levels as a marker of rhabdomyolysis, other relevant biomarkers such as myoglobin, lactate dehydrogenase (LDH), and urine pH were not consistently measured, which may have provided a more comprehensive assessment of muscle injury and renal risk.

CONCLUSION

Our study provides critical insights into the relationship between trauma severity, fluid resuscitation, muscle injury markers, and the incidence of AKI among patients with rhabdomyolysis. Our findings demonstrate that higher trauma severity is associated with an increased risk of AKI, highlighting the importance of early identification and targeted management in severely injured patients. The significant association between higher fluid resuscitation volumes and increased AKI incidence underscores the complex role of fluid management in rhabdomyolysis, where aggressive hydration is essential for preventing myoglobinuric AKI but may lead to fluid overload and worsen renal outcomes if not carefully monitored. There is a need for a balanced approach to fluid resuscitation, individualized patient monitoring, and timely intervention to prevent AKI and improve outcomes in patients with severe muscle injury.

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