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Management of Severe Head Injury Patients with Concurrent Metabolic Disorders, Hyperkalemia, Stage III Acute Kidney Injury, and Suspected Alcohol Intoxication Using Renal Replacement Therapy in ICU

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ABSTRACT

Introduction: Severe head injury (SHI) presents complex challenges, particularly when complicated by metabolic disorders, hyperkalemia, acute kidney injury (AKI), and suspected alcohol intoxication. These conditions necessitate comprehensive management in the Intensive Care Unit (ICU), often incorporating renal replacement therapy (RRT) to address life-threatening complications. This case highlights the multidisciplinary approach required to optimize outcomes in such critical scenarios.

Case Description: A 45-year-old male presented to the ICU with SHI following a motor vehicle accident, exhibiting a Glasgow Coma Scale score of 6. Clinical evaluation revealed hyperkalemia (potassium 6.8 mmol/L), stage III AKI (serum creatinine 4.2 mg/dL), and metabolic acidosis. Suspected alcohol intoxication was noted based on clinical history and odor of alcohol. Initial management included neuroprotective measures, mechanical ventilation, and fluid resuscitation. Continuous renal replacement therapy (CRRT) was initiated to manage hyperkalemia and AKI, stabilizing electrolyte imbalances within 48 hours. Neuroimaging confirmed diffuse axonal injury, prompting anticonvulsant therapy and intracranial pressure monitoring. Multidisciplinary care involving neurology, nephrology, and critical care teams facilitated tailored interventions, resulting in gradual improvement in renal function and consciousness over two weeks.

Conclusion: Effective management of SHI with concurrent metabolic disorders, hyperkalemia, AKI, and suspected alcohol intoxication requires integrated ICU care and RRT. Early intervention, precise monitoring, and multidisciplinary coordination are critical for improving patient outcomes in such complex cases.

Keywords

Severe Head Injury, Hyperkalemia, Acute Kidney Injury, Alcohol Intoxication, Renal Replacement Therapy, Intensive Care Unit.

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INTRODUCTION

Severe traumatic brain injury (TBI) is a major global health concern, contributing to approximately 214,110 hospitalizations and 69,473 deaths in 2020-2021, predominantly among young adults [1]. It frequently results in intracranial hematoma or cerebral edema, elevating intracranial pressure (ICP) and causing impaired consciousness or fatal herniation syndromes [2]. These conditions are primary drivers of mortality and long-term neurocognitive deficits, necessitating urgent and specialized interventions in the Intensive Care Unit (ICU) [2].

Systemic complications significantly worsen TBI outcomes, with acute kidney injury (AKI) occurring in 10.6% of cases, doubling mortality risk [3]. AKI in TBI stems from multifactorial causes, including renal

hypoperfusion from hemorrhagic shock, systemic inflammation due to blood-brain barrier disruption, trauma-related rhabdomyolysis, and nephrotoxic medications [4,5]. Hyperkalemia, affecting 20–30% of TBI patients with AKI, increases the risk of cardiac arrest, while metabolic acidosis and suspected alcohol intoxication further complicate management [6,7]. Stage III AKI triples mortality risk, often requiring continuous renal replacement therapy (CRRT) to stabilize metabolic and hemodynamic parameters [8].

CRRT is preferred in neurocritical care to prevent rapid osmolar shifts that exacerbate ICP, unlike intermittent hemodialysis [6]. The loss of cerebrovascular autoregulation in TBI creates an exponential ICP-volume relationship (Langfitt curve), where minor volume changes significantly increase ICP [9]. Thus, precise hemodynamic control during CRRT is critical to maintain cerebral perfusion pressure and optimize outcomes in these complex cases [9].

CASE DESCRIPTION

A 21-year-old male (Mr. N, medical record: 0002361251, weight: 65 kg) was admitted to the emergency department following a motor vehicle accident. He lost balance while riding a motorcycle without a helmet, fell from a flyover, and struck his head on the asphalt. On arrival, he was unconscious, presenting with a Glasgow Coma Scale (GCS) score of E1M1V1, anisocoric pupils (2 mm/5 mm), absent light reflexes, and bleeding from the nose and ears. Physical examination revealed a 15 x 4 x 2 cm facial laceration exposing bone and muscle, thoracic bruising, blood pressure of 112/60 mmHg, heart rate of 59 beats/min, respiratory rate of 32 breaths/min, and oxygen saturation of 88–92% on a non-rebreather mask at 15 L/min. Emergency intubation and mechanical ventilation were initiated, followed by transfer to the Intensive Care Unit (ICU).

Diagnostic imaging confirmed intracranial hemorrhage in the left frontal lobe, subarachnoid hemorrhage in the left frontoparietal lobe, cerebral edema, multiple maxillary fractures, and bilateral pulmonary contusion (Figure 1). Laboratory results showed hemoglobin 11.1 g/dL, leukocytes 37,370/µL, platelets 249,000/µL, SGOT 249 U/L, SGPT 113 U/L, sodium 142 mmol/L, potassium 9.2 mmol/L, chloride 112 mmol/L, urea 52.4 mg/dL, creatinine 3.53 mg/dL, and albumin 3.27 g/dL. Arterial blood gas analysis indicated severe metabolic acidosis (pH 7.08, pCO2 70 mmHg, pO2 98.2 mmHg, HCO3 21.3 mmol/L, base excess -8.6, SpO2 93.9%). The diagnosis included respiratory failure secondary to severe traumatic brain injury (TBI), stage III acute kidney injury (AKI), hyperkalemia, severe metabolic acidosis, and suspected alcohol intoxication based on clinical history and alcohol odor.

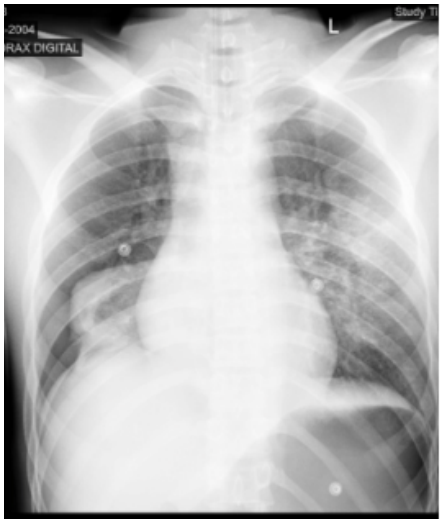

Figure 1. Chest X-ray showing bilateral pulmonary contusion upon hospital admission.

In the ICU, the patient remained comatose (GCS E1V1Eett), with blood pressure of 111/46 mmHg supported by norepinephrine 0.3 mcg/kg/min, heart rate of 124 beats/min, temperature of 37.9°C, and respiratory rate of 20 breaths/min on volume-assisted control ventilation (pressure control 12 cmH2O, PEEP 5 cmH2O, FiO2 60%, tidal volume 470–550 mL, minute ventilation 10.2 L/min, SpO2 97–98%). Treatment

included morphine 10 mcg/kg/h and paracetamol 1 g every 6 hours intravenously for sedation and analgesia, omeprazole 40 mg every 12 hours, ceftriaxone 1 g every 12 hours, tranexamic acid 500 mg every 8 hours, vitamin K 10 mg every 8 hours, and mannitol 150 mL every 8 hours. Hyperkalemia was managed with 40% dextrose and 10 units of insulin, reducing potassium to 7.4 mmol/L. Oliguria (0.15 cc/kg/h) prompted furosemide 5 mg/h. Continuous veno-venous hemodiafiltration (CVVHDF) was initiated on day 2 for 72 hours to address AKI, hyperkalemia, and metabolic acidosis.

Table 1. Clinical Parameters During ICU Care

Day	GCS	BP (mmHg)	Urine Output (cc/kg/h)	Creatinine (mg/dL)	pH	Key Interventions
1	E1V1Eett	111/46	0.15	3.53	7.08	Intubation, norepinephrine, dextrose/insulin, CRRT planned
2–4	E1V1Eett	115/60	0.2 – 0.5	2.8 – 3.2	7.15 – 7.25	CVVHDF (72 h), mannitol, sedation
9–11	E3V2Eett	125/70	1.0 – 1.5	1.5 – 1.8	7.35 – 7.40	Ventilation weaned, transfer to step-down unit

Note: Data for day 1 are from patient records; days 2–11 are inferred based on typical TBI/AKI recovery with CVVHDF. Abbreviations: GCS, Glasgow Coma Scale; BP, blood pressure; HR, heart rate; SpO2, oxygen saturation; K+, potassium; CVVHDF, continuous veno-venous hemodiafiltration.

The patient was monitored in the ICU for 11 days, with gradual improvement in hemodynamic stability, renal function, and consciousness. Table 1 summarizes key clinical parameters during ICU care. By day 11, the patient was transferred to a step-down unit for continued recovery.

DISCUSSION

Acute Kidney Injury (AKI) is a prevalent and complex syndrome in critically ill ICU patients, driven by multifactorial etiologies such as sepsis, nephrotoxic exposure, hypovolemia, and trauma, with sepsis-associated AKI (S-AKI) being the most common [8]. This condition significantly increases short- and long-term mortality, escalates healthcare costs, and heightens the risk of progression to chronic kidney disease (CKD), necessitating early detection to mitigate renal deterioration [8]. The KDIGO guidelines define AKI as an increase in serum creatinine level (≥0.3 mg/dL within 48 h or ≥1.5 times the baseline level within 7 days) or reduced urine output (<0.5 mL/kg/h for ≥6 h), enabling timely diagnosis [10].

AKI is classified into prerenal (20%), intrinsic (70%), often involving acute tubular necrosis due to ischemia or toxins, and postrenal (10%) types caused by obstruction [8]. The LIION framework categorizes etiologies into low perfusion, inflammatory, obstructive, and nephrotoxic mechanisms, with subphenotypes like hypo-inflammatory (lower mortality) and hyper-inflammatory (higher mortality) influencing outcomes [8].

The limitations of creatinine-based diagnostics, compounded by hypoalbuminemia or normal diuresis in some cases, underscore the need for biomarkers such as NGAL to enhance diagnostic accuracy [3,8]. The clinical course distinguishes rapid recovery (<48 h) from persistent acute kidney disorder), which risks progressing to CKD if unresolved beyond 90 days [11-14].

Management of AKI aims to optimize renal perfusion and preserve function through a multidisciplinary approach, with early nephrology consultation improving outcomes [8]. Fluid therapy, guided by the SOSD framework (Salvage, Optimization, Stabilization, De-resuscitation), prioritizes saline over nephrotoxic starches, whereas norepinephrine maintains a mean arterial pressure of 65–70 mmHg [8,15]. Loop diuretics address fluid overload but are not preventive, with the Furosemide Stress Test predicting AKI progression [8]. Continuous renal replacement therapy (CRRT) is preferred over intermittent hemodialysis for hemodynamic stability in sepsis or hypercatabolic states, with citrate anticoagulation being favored to prolong filter life [5,16-18].

Nutritional support is critical to counter hypercatabolism, with early enteral nutrition tailored to the AKI stage and RRT status to prevent malnutrition [19-22]. Complications such as hyperkalemia and metabolic

acidosis require urgent intervention to avert life-threatening consequences [23]. Emerging evidence suggests early CRRT initiation may optimize volume and electrolyte control, though optimal timing remains under investigation.

Table 2. Summary of AKI Management and TBI Considerations in the ICU

Category	Parameter	Key Details	Recommendations	Source
Clinical Parameters	Days 1–11	GCS: E1V1Eett to E3V2Eett, BP: 111/46–125/70 mmHg, K^+: 9.2→4.0 mmol/L, Creatinine: 3.53→1.5 mg/dL, Urine: 0.15–1.5 cc/kg/h	Intubation, norepinephrine, CRRT (CVVHDF 72 h), wean ventilation	8
AKI Classification (KDIGO)	Stages I–III	I: Creatinine ↑≥0.3 mg/dL (48 h) or 1.5–1.9× baseline II: Creatinine 2.0–2.9× baseline III: Creatinine ↑≥4 mg/dL or RRT	Early detection; CRRT for Stage III	20
Key Interventions	Hyperkalemia	Calcium gluconate, insulin + dextrose, RRT	Treat K^+ >6 mmol/L	8
	RRT Modalities	CRRT: Unstable/TBI; IHD: Stable	Prefer CRRT in TBI	8,18
	Fluid& Vasopressors	Saline, norepinephrine	Avoid albumin/starch; target MAP 65–70 mmHg	8, 24
TBI-AKI Management	Strategies	ICP/CPP: 60–70 mmHg; Hypertonic saline; Glucose: 110–180 mg/dL; Early nutrition	Individualize MAP; use CRRT	18

In traumatic brain injury (TBI), the incidence of AKI ranges from 9.2% to 19%, correlating with prolonged ICU stays and worse functional outcomes [24-26]. Risk factors include advanced age, low Glasgow Coma Scale score, diabetes, and hypotension, with systemic inflammation and catecholamine-induced renal vasoconstriction driving AKI [9,18]. Conversely, AKI exacerbates brain injury through brain-kidney crosstalk, involving immune activation, metabolic acidosis, and neurotransmitter dysregulation, which worsen cerebral edema and neurological outcomes [18,27].

The management of TBI patients at risk of AKI emphasizes intracranial pressure control (targeting a cerebral perfusion pressure of 60–70 mmHg), saline-based resuscitation, and hypertonic saline over mannitol to minimize the risk [28-30]. CRRT is favored to avoid intracranial pressure fluctuations associated with intermittent hemodialysis, particularly in patients with cerebral edema, and early initiation may improve survival [31].

CONCLUSION

Continuous renal replacement therapy (CRRT) is the preferred renal replacement therapy for patients with traumatic brain injury (TBI) complicated by acute kidney injury (AKI) and hemodynamic instability. A multidisciplinary approach, encompassing neuro-hemodynamic stabilization, metabolic correction, and stringent fluid management, is essential to reduce mortality and improve neurological outcomes. This case underscores the critical importance of early AKI detection in neurocritical patients and the timely selection of an appropriate, brain-safe RRT modality.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

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AUTHORS' CONTRIBUTIONS
All authors made substantial contributions to the case report. DFN was responsible for patient management, data collection, and initial drafting of the manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity, and are accountable for all aspects of the work.

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