Case Report

Continuous renal replacement therapy for acute kidney injury: a case report

Jianmin Wang[#], Yiyao Bao[#^], Lijun Guan^, Linhua Tan^

Department of Surgical ICU, The Children's Hospital, Zhejiang University School of Medicine, National Clinical Research Center for Child Health, Hangzhou, China

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[#]These authors contributed equally to the work.

Correspondence to: Linhua Tan, MD, PhD. Department of Surgical ICU, The Children's Hospital, Zhejiang University School of Medicine, National Clinical Research Center for Child Health, 3333 Binsheng Road, Hangzhou 310052, China. Email: chtlh@zju.edu.cn.

Background: Acute kidney injury is a severe complication of pediatric trauma with complex and diverse mechanisms and is associated with multiple organ dysfunction. For severely injured children, renal function should be closely monitored, and continuous renal replacement therapy should be promptly initiated if there is a risk of developing severe acute kidney injury. This case report describes the treatment process of a child with acute kidney injury due to trauma, highlighting the need to improve the rapid identification of traumatic acute kidney injury risk and actively implement resuscitation strategies to protect renal function in trauma patients.

Case Description: A 3-year-old boy presented with acute kidney injury following a car accident. Multi-organ dysfunction developed, and laparotomy and liver laceration repair were performed. Continuous renal replacement therapy for 7 days, plasma exchange, antimicrobial therapy, and multi-organ support were provided. A 1-year follow-up showed no organ dysfunction.

Conclusions: This case report describes the treatment process of a child with acute kidney injury due to trauma, highlighting the need to improve the rapid identification of traumatic acute kidney injury risk and actively implement resuscitation strategies to protect renal function in patients with trauma. Prompt continuous renal replacement therapy should be considered when necessary to prevent adverse outcomes.

Keywords: Acute kidney injury (AKI); pediatrics; multi-organ dysfunction; trauma; case report

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Introduction

Acute kidney injury (AKI) in children can have various etiologies and, if not diagnosed promptly, can rapidly progress to acute kidney failure and chronic kidney disease, necessitating lifelong renal replacement therapy or kidney transplantation. Approximately 10% of children admitted to pediatric intensive care units (ICUs) develop AKI, substantially affecting the mortality rate (1). The risk of AKI increases after trauma owing to direct renal damage, shock, hypoxia, microcirculatory disorders, ischemia–reperfusion injury, exposure to pathogens and toxins, nephrotoxic drug use, and rhabdomyolysis (2,3). A meta-analysis of 24 studies (4) reported an AKI incidence of 24% among patients with trauma admitted to ICUs, with mild, moderate, and severe

^ ORCID: Yiyao Bao, 0009-0003-2185-2122; Lijun Guan, 0009-0006-7658-5537; Linhua Tan, 0000-0001-6624-4970.

AKI observed in 13%, 5%, and 4% of patients, respectively. Compared to patients without AKI, those with AKI had a prolonged ICU stay of 6.0 (range, 4.0–7.9) days and high mortality risk [risk ratio =3.4 (95% confidence interval: 2.1–5.7)]. Among patients with AKI, 10% required continuous renal replacement therapy (CRRT), and renal function recovered in 96%. To enhance awareness of traumatic AKI (T-AKI) in children and optimize the timing of renal replacement therapy, we present a case of a child with multiple injuries sustained from a car accident who developed acute kidney failure and was treated with CRRT at The Children's Hospital, Zhejiang University School of Medicine. We present this case in accordance with the CARE reporting checklist (available at https://tp.amegroups.com/article/view/10.21037/tp-2024-500/rc).

Case presentation

This is a case of AKI following trauma. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Publication of this case report and accompanying images was waived from patient consent according to The Children's Hospital, Zhejiang University School of Medicine Ethics Committee (approval No. 2024-IRB-0304-P-01).

Highlight box

Key findings

- This case highlights the successful management of a child with traumatic acute kidney injury (AKI) through the use of continuous renal replacement therapy (CRRT).

What is known and what is new?

- AKI is a severe complication of pediatric trauma with complex and diverse mechanisms and is associated with multiple organ dysfunction.
- For severely injured children, renal function should be closely monitored, and CRRT should be promptly initiated if there is a risk of severe AKI development.

What is the implication, and what should change now?

- This case suggests that for children with severe injuries, renal function should be closely monitored, and if there is a risk of developing severe AKI, CRRT should be initiated promptly.
- Clinical doctors should emphasize the need to rapidly identify the risk of T-AKI and actively implement resuscitation strategies to protect renal function in trauma patients.

A 3-year-old boy was admitted to our surgical ICU because of multiple injuries from a car accident sustained 2 days before and oliguria lasting 1 day. Two days before admission, while riding a scooter uphill, the boy was struck by a four-wheeled car from the right side, thrown approximately 2 m away, and landed on his left side. He immediately lost consciousness and was admitted to a local hospital. Upon arrival, he had cyanotic lips, and cardiopulmonary arrest was noted. Cardiopulmonary resuscitation was initiated and spontaneous circulation was restored after 2 min. Imaging and diagnostic tests revealed a right liver laceration, and the patient underwent emergency surgery for liver laceration repair, cholecystectomy, and intra-abdominal exploration. Postoperatively, the patient was hospitalized at a local facility and received ceftriaxone as antimicrobial therapy; gastroprotection; hepatoprotection; and transfusions of red blood cells, plasma, and platelets. One day before the transfer, he developed dark urine and oliguria (24-h urine <100 mL). He was intubated and transferred to The Children's Hospital, Zhejiang University School of Medicine by ambulance for further treatment. His medical, personal, and family histories were unremarkable.

On admission at The Children's Hospital, Zhejiang University School of Medicine, the patient's physical examination revealed the following: temperature 36.7 ℃, pulse 107 beats/min, respiratory rate 25 breaths/min (on mechanical ventilation), blood pressure 91/40 mmHg, Glasgow Coma Scale score 4 + T, equal and round pupils with a diameter of 1.5 mm, delayed light reflexes, coarse breath sounds bilaterally without rales or rhonchi, regular heart rhythm without significant murmurs, a soft abdomen with a dry surgical wound dressing and patent hepatic drainage tube, negative Babinski signs bilaterally, normal muscle tone in all limbs, capillary refill time of 2 s, and an indwelling urinary catheter. Laboratory tests showed the following: white blood cell count 10.13×10^9/L, neutrophil percentage 83.71%, hemoglobin level 83 g/L, platelet count 71×10^9/L, high-sensitivity C-reactive protein (hs-CRP) level 26.36 mg/L, total bilirubin level 43.2 μmol/L, albumin level 33.9 g/L, amylase level 1,823 U/L, urea level 26.76 mmol/L, creatinine level 152.4 μmol/L, creatine kinase level 9,252 U/L, alanine transaminase level 7,162 U/L, aspartate transaminase level 11,350 U/L, potassium level 6.5 mmol/L, myoglobin level >400 ng/mL, and procalcitonin level 15.40 ng/mL. Chest computed tomography (CT) showed bilateral pulmonary contusions with pleural effusion. Contrast-enhanced abdominal CT revealed post-liver laceration repair with occlusion of the right hepatic artery

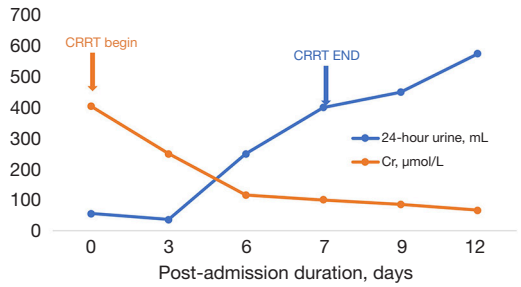

Figure 1 Changes in urine output and serum creatinine level over time. Cr, creatinine; CRRT, continuous renal replacement therapy.

and right portal vein, extensive hypoperfusion in the right lobe, heterogeneous perfusion in the left lobe, a mass-like shadow in the right lobe (possible hematoma), reduced perfusion in both kidneys with poor differentiation between the cortex and medulla, no obvious abnormalities in the main renal arteries and veins, and intra-abdominal fluid collection. Head CT showed unclear gray-white matter differentiation, suggesting possible cerebral edema. Bedside electroencephalography demonstrated diffuse delta wave background activity with frequent beta waves, spikes, and spike-wave discharges in the right parietal region. Brain magnetic resonance imaging indicated a cerebral contusion, and whole-spine magnetic resonance imaging revealed no significant abnormalities.

The initial diagnosis included: (I) traumatic liver laceration; (II) multi-organ (brain, liver, kidney, and lung) dysfunction; (III) hemorrhagic shock; (IV) traumatic coagulopathy; (V) sepsis; (VI) pleural and peritoneal effusions; (VII) post-cardiopulmonary resuscitation; and (VIII) rhabdomyolysis. After the injury, the child underwent emergency surgery. On the day after the operation, urine output was adequate, and fluid balance was stable. By the second day, blood pressure remained stable, and fluid rehydration was administered according to physiological requirements. However, urine output reduced, its color darkened, blood creatinine levels increased, and the glomerular filtration rate declined, though other signs were not significantly aggravated. Enhanced abdominal CT showed decreased perfusion in both kidneys, further elevation of creatinine, dark-colored urine, and low urine volume. The continuous venovenous hemofiltration (CVVH) mode was adopted for CRRT treatment for 1 week. One session of plasma exchange was performed because of markedly elevated myoglobin levels. Considering the post-operative state and trauma, sodium citrate was used for anticoagulation *in vitro* (5), with adjustments based on calcium ion levels. Liver function was closely monitored following liver rupture repair. Daily urine output gradually increased (*Figure 1*), while serum creatinine and urea nitrogen levels progressively decreased (*Figure 1*). The patient also required mechanical ventilation for 6 days and underwent hypothermia therapy for the brain for 72 h, fluid resuscitation, and urine alkalinization. Additional treatments included myocardial nutrition, hepatoprotective therapy, antimicrobial therapy, and multiple thoracenteses to drain pleural effusions. The patient remained in the ICU for 20 days, with an average urine output of 3.0 mL/kg/h and normalized creatinine levels at discharge. The patient was subsequently transferred to a general ward and discharged 2 days later. A follow-up at 1 year post-discharge showed normal renal function.

Discussion

In China, there are no definitive reports on the incidence of kidney injury related to pediatric trauma. However, a meta-analysis (6) of nine relevant studies found that the prevalence of T-AKI in children ranged from 0% to 30.3%, with a pooled prevalence of 9.86% (95% confidence interval: 8.02–11.84%), potentially increasing the risk of death by 5.5 times. Kidney injuries following trauma can be categorized as direct or indirect. The kidneys, as retroperitoneal organs, are protected by a strong fibrous capsule and a thick perirenal fat pad, resulting in a lower incidence of direct kidney injury, which is mainly caused by blunt or penetrating trauma. Low-grade (I–III) injuries are typically confined to the capsule and parenchyma, whereas high-grade injuries involve the major renal vessels and collecting systems (7). Conversely, the kidneys are relatively vulnerable secondary target organs after trauma, and AKI often develops within a few days of injury. Our patient developed oliguria with elevated creatinine levels on the third day after the trauma. The early causes of AKI include hypovolemic shock, hypoxia, and ischemia-reperfusion, whereas secondary causes mainly involve nephrotoxic medications and treatments, and complications such as sepsis, abdominal compartment syndrome, and massive blood transfusions (4,8,9). Myoglobin is directly toxic to the renal tubules, and its rapid accumulation can lead to tubular obstruction and subsequent necrosis, resulting in renal failure. Our patient had multiple injuries, including hemorrhagic shock, liver laceration, a history of massive blood transfusion, high infection markers, and markedly

elevated myoglobin levels, all of which can contribute to kidney injury.

Recent studies have highlighted the important role of systemic and local immune responses in the kidneys following trauma in the development of AKI (10). Within minutes of trauma, the innate immune response is triggered by the autonomic nervous system, which releases various damage-associated molecular patterns (DAMPs) and pathogen-associated molecular patterns (PAMPs). These molecules are recognized by pattern recognition receptors on immune cells, initiating systemic inflammatory and coagulation cascade reactions, exacerbating immune barrier disruption, and leading to further release of DAMPs and PAMPs, creating a vicious cycle of innate immune activation that ultimately results in multi-organ dysfunction, including kidney dysfunction. In the glomeruli, the release of DAMPs, such as high-mobility group box 1 and histones, along with the activation of coagulation and complement systems, induces endothelial changes. Renal tubular epithelial cells contribute to inflammation by producing DAMPs, PAMPs, and various cytokines that further exacerbate kidney injury. This has important implications for identifying new biomarkers and therapeutic targets for kidney injury, as the kidneys are interconnected with almost all other organ systems. For example, in traumatic brain injury (11), sympathetic nerve stimulation, hypothalamic-pituitary-adrenal axis activation, and neuroinflammation are key drivers of the brain-kidney axis. AKI can adversely affect the brain by altering the body's water content and disrupting the blood-brain barrier, which can promote the influx of inflammatory mediators and toxic substances into the injured kidneys. A study of patients with trauma (12) showed that acute lung injury requiring mechanical ventilation was a risk factor for AKI and that mechanical ventilation was independently associated with early AKI progression. The heart, liver, spleen, and gastrointestinal tract interact with the kidneys, complicating the management of severely injured patients. Paradoxically, measures aimed at stabilizing damaged tissues and organs may exacerbate kidney injury.

Severe trauma often leads to rhabdomyolysis, in which the release of myoglobin and creatine kinase from damaged muscles can cause renal injury. Approximately 50% of patients with rhabdomyolysis develop AKI via multiple mechanisms (13). First, fluid loss from muscle injury reduces the intravascular volume, leading to renal vasoconstriction. Second, myoglobin is toxic to renal tubular epithelial cells, causing tubular injury and vasoconstriction in the renal microcirculation, leading to ischemic damage. Third, myoglobin can precipitate and form casts within the renal tubules, causing obstruction and further renal damage. Additionally, systemic inflammation and the release of proinflammatory cytokines following muscle injury can exacerbate renal injury and promote AKI progression. Correcting hypovolemia, improving renal perfusion, and alkalinizing urine are fundamental measures for treating rhabdomyolysis. Renal replacement therapy should be considered for patients with severe AKI due to rhabdomyolysis and life-threatening complications, such as hyperkalemia, electrolyte disturbances, hyperuricemia, and fluid overload. Given that myoglobin has a molecular weight of 17 kDa, conventional dialysis is limited in removing it from the circulation. A previous study (14) has shown that CVVH is effective in clearing myoglobin. Our patient presented with tea-colored urine, hyperkalemia, and anuria after a motor vehicle accident, indicating severe AKI. Timely initiation of CVVH combined with plasma exchange positively affects patient prognosis.

During CRRT, clotting in the extracorporeal circuit can shorten the lifespan of filters and catheters, increase blood loss, reduce solute clearance, and decrease the effectiveness of CRRT, thereby increasing the treatment cost and workload. Therefore, anticoagulation strategies should be carefully considered during CRRT. Unfractionated heparin is commonly used owing to its low cost, ease of administration, simple monitoring, and reversibility with protamine. However, for patients with a high risk of bleeding, such as those with recent active bleeding, trauma, surgery, or uncontrolled hypertension, systemic anticoagulation (e.g., heparin) carries a higher risk of bleeding, and regional citrate anticoagulation (RCA) is recommended (15). Multiple studies (16,17) have shown that RCA is superior to systemic or regional heparin anticoagulation, effectively reducing the risk of circuit loss and filter failure while more safely lowering the risk of bleeding. The potential complications of RCA, such as hypocalcemia, require close monitoring to prevent adverse effects. Our patient was treated with RCA, and monitoring of coagulation function, bleeding status, and electrolytes (including calcium) did not reveal any significant complications.

Conclusions

AKI is a severe complication of pediatric trauma with complex and diverse mechanisms and is associated with multiple organ dysfunction. AKI increases the need for

mechanical ventilation and renal replacement therapy and worsens the 28-day mortality rate (18). Untimely treatment can lead to chronic kidney disease, necessitating increased vigilance. For severely injured children, renal function should be closely monitored, and CRRT should be promptly initiated if there is a risk of severe AKI development. Currently, research on CRRT use and randomized controlled trials in pediatric T-AKI is far less extensive than that in adults, and more high-quality studies are needed to clarify the indications, timing, and anticoagulation strategies for CRRT. This case report describes the treatment process of a child with AKI due to trauma, highlighting the need to improve the rapid identification of T-AKI risk and actively implement resuscitation strategies to protect renal function in patients with trauma. Prompt CRRT should be considered when necessary to prevent adverse outcomes.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://tp.amegroups.com/article/view/10.21037/tp-2024-500/rc

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Publication of this case report and accompanying images was waived from patient consent according to The Children's Hospital, Zhejiang University School of Medicine Ethics Committee (approval No. 2024-IRB-0304-P-01).

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