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CASE REPORT

Septic Shock and Rhabdomyolysis as Rare Complications Following Rectal Perforation: A Case Report

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Abstract: Rectal perforation leads to massive translocation of intestinal bacteria and enteric fluid into the peritoneal cavity, which can cause severe intra-abdominal infection and may progress to sepsis. Beyond prompt surgical intervention and drainage, proactive prevention of potential postoperative complications is critically important. This report describes the case of a patient with septic shock secondary to rectal perforation complicated by rhabdomyolysis, who was managed in our intensive care unit. During treatment, HA380 hemoperfusion was utilized as an adjunctive therapy to assist in controlling the inflammatory response and potentially mitigate the progression of acute kidney injury. Through the analysis of this clinical case, we emphasize that in patients with rectal perforation, halting the progression of sepsis, timely clearance of inflammatory cytokines, and multidimensional prevention of complications are essential components of successful management.

Keywords: septic shock, rectal perforation, rhabdomyolysis, hemoperfusion

Introduction

Rectal perforation represents a surgical emergency that allows massive translocation of enteric bacteria and intestinal fluid into the peritoneal cavity, leading to severe intra-abdominal infection which frequently progresses to septic shock and multiple organ dysfunction syndrome (MODS), conditions associated with persistently high mortality rates.[1] The core pathophysiological processes driving organ injury in sepsis involve a dysregulated inflammatory response and profound microcirculatory dysfunction.[2] Rhabdomyolysis (RM), although an uncommon complication of sepsis, can further exacerbate renal impairment and disrupt internal homeostasis, posing considerable therapeutic challenges.[3] This case report details the successful multidisciplinary management of a patient who developed septic shock, MODS, and RM following rectal perforation surgery, analyzing the underlying mechanisms of the cytokine storm in sepsis and summarizing the integrated use of continuous renal replacement therapy (CRRT) coupled with hemoperfusion (HP) as part of a comprehensive rescue strategy.

Case Presentation

A 60-year-old male patient presented with abdominal pain and was admitted to a local hospital, where a computed tomography scan indicated gastrointestinal perforation. He had no significant past medical history. He subsequently underwent emergent exploratory laparotomy with partial rectal resection, temporary sigmoid colostomy, and peritoneal lavage and drainage in the early hours of the following day. On the second postoperative day, he was transferred to the

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intensive care unit (ICU) of our hospital. Upon admission, the patient was comatose and mechanically ventilated via an orotracheal tube. The abdominal surgical wound dressing was dry, the peritoneal drainage tube was patent, and the colostomy was well-situated (Figure 1). Diffuse skin mottling was observed on all four limbs. A diagnosis of septic shock secondary to rectal perforation and abdominal infection was established.

In strict adherence to the 1-hour sepsis bundle, the patient received rapid crystalloid infusion totaling 31.9 mL/kg within the first 3 hours, resulting in a decrease in lactate levels from 6.8 mmol/L on admission to 2.9 mmol/L. Vasoactive agents and empirical antibiotics were initiated concurrently. Pending results of intra-abdominal pus culture, broad-spectrum antibiotic therapy with imipenem/cilastatin and vancomycin was administered. The patient maintained a mean arterial pressure around 70 mmHg with vasopressor support. Laboratory studies revealed markedly elevated inflammatory markers, including IL-6 >5000 pg/mL and CRP 188.21 mg/L, attributed to a severe inflammatory response secondary to abdominal infection (Figure 2). To mitigate this excessive inflammatory state and remove circulating cytokines, combined HA380 and CRRT was initiated on the second day of admission using a "1+2+1" protocol: one hemoperfusion session was performed on the first day of treatment, followed by two sessions on the second day, and one session on the third day. During the combined therapy, blood flow was maintained at 80–100 mL/min, with close monitoring and maintenance of phosphate (2.5–4.5 mg/dL) and magnesium levels. Following four sessions of this therapy, IL-6 levels decreased significantly to 234.6 pg/mL, and other inflammatory markers also showed notable improvement. Additionally, the patient presented with persistently elevated creatine kinase (CK) levels several times above the normal range, brown-colored urine, elevated serum creatinine, and proteinuria, indicating acute kidney injury. The disproportionate elevation of CK relative to CK-MB, in the absence of new cardiovascular events, together with the confirmed severe abdominal infection, strongly suggested septic shock complicated by RM-induced acute kidney injury. Aggressive fluid resuscitation and CRRT were continued to prevent further renal deterioration.

The patient experienced recurrent bleeding and discharge from the abdominal wound after admission (Figure 3), with no significant improvement following conservative management. This ultimately necessitated surgical wound debridement and hemostasis on the 7th and 10th days post-admission, with continuous vacuum-assisted closure (VSD) applied after the second debridement. On hospital day 13, the wound bleeding worsened markedly, prompting emergency exploratory laparotomy to identify the source and achieve definitive hemostasis. Intraoperative findings revealed necrotic rectus abdominis muscle, confirming the diagnosis of RM. Subsequent rectus abdominis hematoma evacuation, partial muscle resection, and partial peritonectomy were performed to prevent further spread of necrosis. Following these interventions, the patient's symptoms and abdominal wound condition improved significantly, with effective control of the abdominal infection and a marked decline in inflammatory markers. Vital signs stabilized, mental status gradually returned to normal, and muscle strength and tone improved notably after rehabilitation therapy. The patient was eventually transferred out of the ICU. During subsequent follow-up and continued treatment in the general ward for two months, the patient achieved full recovery and was successfully discharged. Six months later, the patient returned to the hospital for ostomy reversal, with no significant complications observed throughout the entire follow-up period.

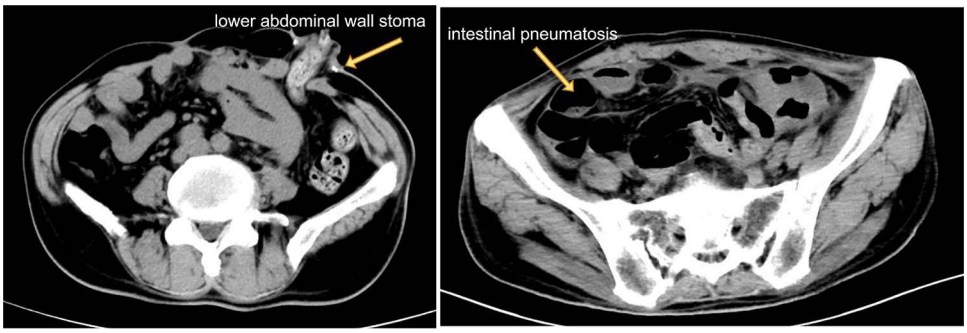

Figure 1 Abdominal CT image of the patient after the operation. The left arrow indicates the left lower abdominal wall stoma. The right arrow indicates intestinal pneumatosis in the right lower abdomen. Additional findings include postoperative exudate in the surgical area, stranding of the fat planes, and a small amount of pelvic fluid.

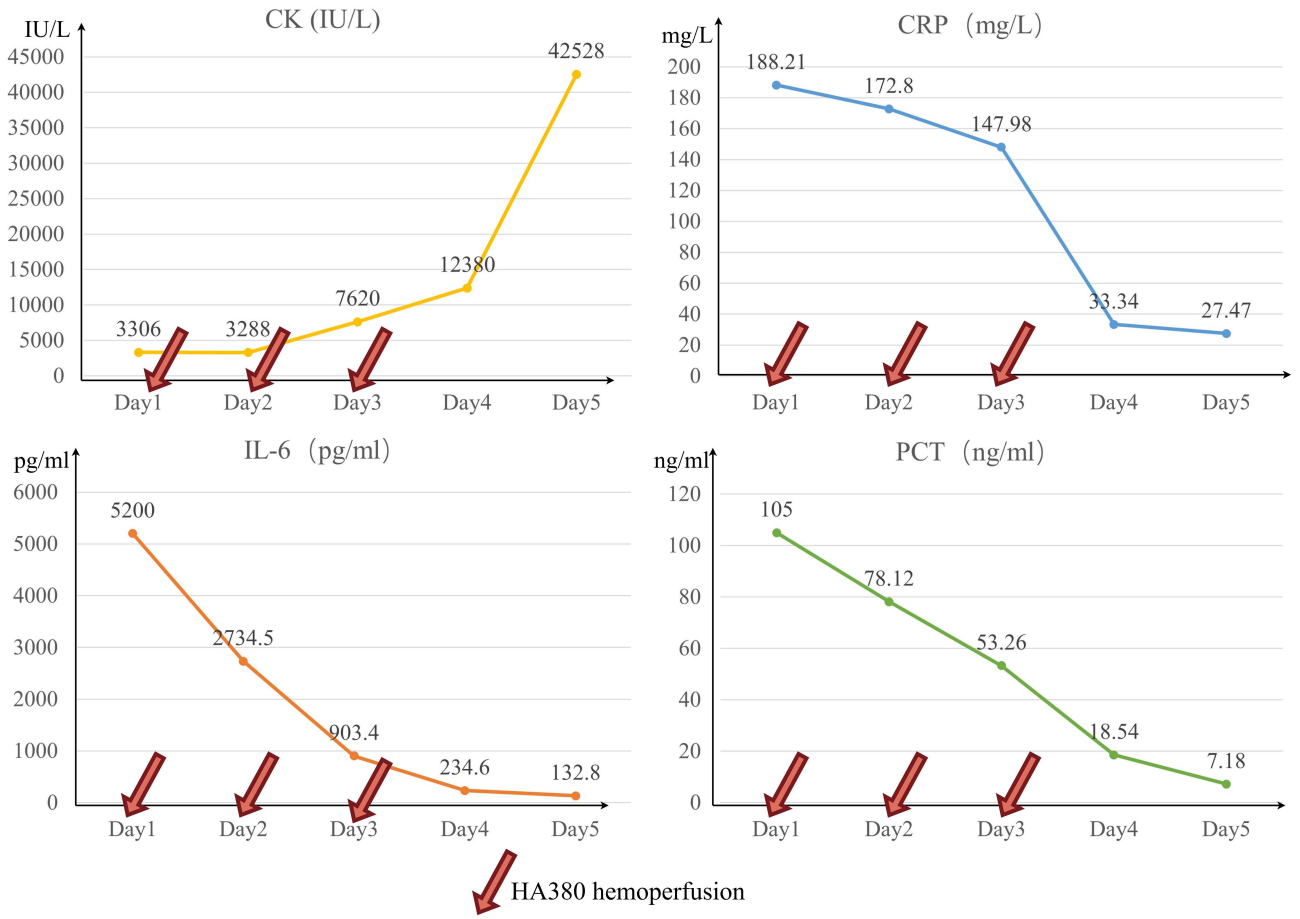

Figure 2 The changing trends of the patient's inflammatory indicators and CK values. The red arrows on the x-axis indicate the days on which HA380 hemoperfusion sessions were performed.
Abbreviations: CK, Creatine Kinase; CRP, C-reaction protein; IL-6, Interleukin6; PCT, Procalcitonin.

Discussion

Rectal perforation represents an acute abdominal condition characterized by the dissemination of enteric bacteria from the fecal stream into the peritoneal cavity, which can subsequently lead to bloodstream infection and trigger an exaggerated inflammatory response. The excessive release of inflammatory mediators contributes to further tissue and organ damage, progressing to sepsis, septic shock, and multiple organ dysfunction syndrome (MODS).[4] Sepsis is defined as a life-threatening systemic inflammatory response syndrome resulting from the invasion of normally sterile tissues or body fluids by pathogenic microorganisms and their metabolites, which activate the humoral immune system to release various cytokines and endogenous mediators.[5] When pathogens or their components breach anatomical barriers, they engage pattern recognition receptors (PRRs), activating downstream signaling pathways that initiate the release of inflammatory factors and create an amplifying cascade.[6] In severe infections, a massive influx of bacteria and endotoxins into the circulation induces an explosive release of pro-inflammatory mediators—a phenomenon known as a cytokine storm—ultimately leading to septic shock and MODS.[7]

In the present case, the initial anti-infective regimen consisted of imipenem/cilastatin combined with vancomycin for broad-spectrum coverage of common pathogens. After culture results identified Escherichia coli and Enterococcus faecalis, therapy was de-escalated to linezolid for targeted treatment. As the patient's clinical status improved, the regimen was further transitioned to cefoperazone/sulbactam plus minocycline for maintenance, which was associated with declining infection markers and gradual recovery of organ function. Effective infection control remains a cornerstone of sepsis management,[8] and accumulating evidence indicates that administering appropriate antibiotics at the correct time and dose can significantly reduce mortality in septic patients, underscoring the vital role of antimicrobial therapy in the overall management of sepsis.[9]

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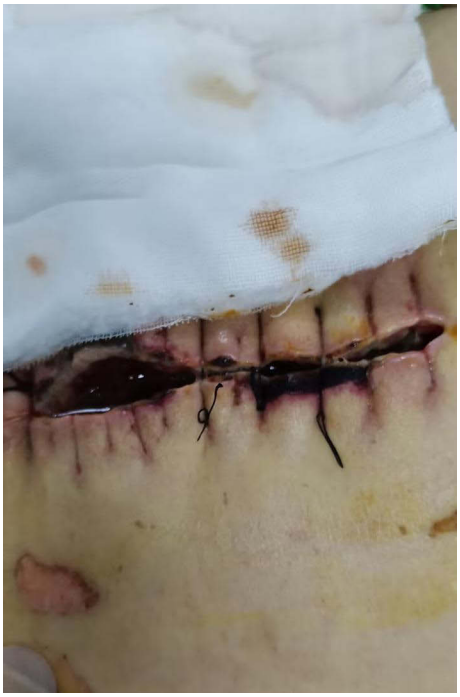

Figure 3 The patient's abdominal wound. The wound healed poorly and obvious bleeding was visible.

Hemoperfusion has demonstrated significant potential in sepsis management by effectively removing inflammatory mediators.[10,11] The HA380 cartridge, a commonly used adsorber in clinical practice, exhibits excellent biocompatibility and broad-spectrum adsorption capacity, enabling efficient clearance of key inflammatory cytokines such as IL-6 and TNF-α.[12,13] Studies have shown that HA380 can markedly ameliorate organ damage and mitigate the "cytokine storm" in patients with heatstroke complicated by multiple organ dysfunction.[14] While previous evidence indicates that continuous renal replacement therapy (CRRT) alone may improve patient outcomes, its efficacy as a standalone treatment is often limited.[15,16] Furthermore, research supports the role of hemoperfusion in reducing circulating myoglobin levels in cases of rhabdomyolysis-induced acute kidney injury (AKI), thereby facilitating renal function recovery.[17]

The decision to initiate combined HA380 hemoperfusion and CRRT early in this patient's clinical course was based on two primary considerations: first, the presence of persistent fever and a confirmed diagnosis of septic shock secondary to abdominal infection warranted timely removal of inflammatory cytokines to attenuate the systemic inflammatory response and prevent exacerbation of infection and shock driven by the cytokine storm; second, the observed persistent rise in serum creatinine indicated sepsis-associated AKI, and early CRRT initiation not only provided solute clearance and fluid management but also contributed to renal tubular protection by clearing circulating inflammatory mediators, restoring metabolic homeostasis, and reducing the burden of damage-associated molecular patterns, thereby potentially preserving renal function.

Rhabdomyolysis (RM) is a clinical syndrome characterized by skeletal muscle cell damage and necrosis resulting from diverse etiologies, leading to the release of intracellular contents into the systemic circulation.[18] When muscle tissue is subjected to direct trauma, ischemia, metabolic disturbances, or severe infection, the integrity of the cell membrane is disrupted, allowing myoglobin and other intracellular components to enter the bloodstream.[19] Under physiological conditions, small amounts of myoglobin can be effectively cleared; however, a rapid and massive release can overwhelm the body's compensatory mechanisms, resulting in systemic complications, the most severe of which is acute kidney injury (AKI).[20] The mechanisms underlying RM-associated renal failure primarily involve renal vasoconstriction, myoglobin cast-induced tubular obstruction, and direct tubular toxicity.[21]

Cases of RM complicating rectal perforation remain rare, and the pathogenesis is multifactorial rather than driven by infection alone. In the present case, although the patient exhibited classic signs of RM such as markedly elevated creatine

kinase and tea-colored urine, its development likely involved several contributing factors. Beyond the systemic inflammatory response and metabolic dysregulation induced by sepsis, technical aspects of surgical intervention—such as excessive suture tension potentially compromising local muscle perfusion—may have precipitated or exacerbated ischemic muscle injury. Additionally, high-dose vasopressor administration (eg, epinephrine) during septic shock resuscitation, while essential for maintaining perfusion pressure, may have induced excessive vasoconstriction, further reduced muscle blood flow and increased RM risk. Reflecting on this case, future management of similar patients should adopt integrated preventive strategies during the perioperative and intensive care phases. Surgical techniques should emphasize meticulous wound closure with appropriate suture tension and strive for thorough debridement to minimize tissue edema resulting from intra-abdominal infection or repeated operations. Vasopressor use requires careful titration to balance end-organ perfusion with the preservation of microcirculatory integrity, thereby avoiding ischemic complications related to excessive dosing. Where feasible, monitoring of intra-abdominal pressure and local tissue perfusion should be implemented to objectively assess abdominal status, with prompt fasciotomy considered if compartment syndrome is suspected to relieve pressure and restore blood flow.

Several limitations in the management of this case merit consideration. Although RM was strongly suspected on the second hospital day due to markedly elevated CK levels and characteristic urine discoloration, the precise anatomical site of muscle injury was not immediately identified or addressed. The combined impact of sepsis and RM likely contributed to the development of AKI. Given the clinical context, more vigilant monitoring of the abdominal wound was warranted; particularly during the sedated period when the patient could not actively report discomfort, a more detailed and repeated abdominal examination might have facilitated earlier detection of rectus abdominis abnormality, enabling prompt surgical intervention to remove the source of ongoing muscle necrosis. Furthermore, during combined HA380 hemoperfusion and CRRT therapy, not all relevant laboratory parameters were dynamically monitored. Although the patient's overall condition improved, finer physiological control during treatment was suboptimal. Future management of similar cases should include systematic blood sampling before and after hemoperfusion sessions to quantitatively demonstrate treatment efficacy. While the successful outcome in this case suggests that hemoperfusion may have contributed to the amelioration of the clinical course of sepsis, it is important to acknowledge that the observed improvement cannot be definitively attributed to hemoperfusion alone. The concurrent use of CRRT, antibiotics, and surgical source control makes it difficult to isolate the specific therapeutic effect of hemoperfusion. Therefore, any benefit of hemoperfusion in this context remains uncertain, and its apparent efficacy may be confounded by other concurrent interventions. While the successful outcome in this case suggests that hemoperfusion may ameliorate the clinical course of sepsis, its generalizability remains limited without larger sample sizes and statistical validation. We propose establishing a dedicated hemoperfusion therapy team, developing a structured patient registry, and initiating multi-center collaborations to further investigate the potential of hemoperfusion combined with CRRT in sepsis. RM remains an uncommon and clinically subtle complication of rectal perforation. In patients presenting with abdominal pain, impaired wound healing, markedly elevated CK, or tea-colored urine, the possibility of RM should be considered, with prompt evaluation and intervention to reduce complications and sequelae.

Conclusion

RM secondary to rectal perforation is an exceedingly rare clinical entity, with only a limited number of cases reported in the literature. This case report highlights the rare occurrence of RM secondary to rectal perforation, primarily attributed to severe infection. RM lacks a single definitive diagnostic gold standard; it is generally diagnosed when plasma CK exceeds five times the upper limit of normal (or >1000 U/L), accompanied by significantly elevated serum myoglobin and urine dipstick positivity for blood without commensurate red blood cells. The therapeutic goals for RM include halting further skeletal muscle injury, preventing acute renal failure, and rapidly identifying life-threatening complications. In this case, early initiation of combined CRRT and hemoperfusion was associated with attenuation of progression of sepsis and provided critical time for subsequent surgical source control. The multifactorial etiology of RM was thoroughly analyzed, underscoring the importance of considering non-infectious contributors in future cases. This report not only enriches the literature on RM but also suggests a potential role for early combined hemoperfusion and CRRT in similar complex cases. Continued research and broader clinical studies are essential to establish standardized therapeutic protocols for such rare and complex presentations.

Ethics Approval and Consent to Participate

Ethical approval for this publication by IRB at Yichang Central People's Hospital.

Patient Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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