Journal of Society Medicine
Research & Review Articles on Diseases
Journal of Society Medicine. 2026; 5 (8)

Gut-Origin Sepsis After Radical Cystectomy and Ileal Conduit: Early Intensive Care Unit Recognition and Targeted Management Leading to Rapid Recovery

Ida Juita Halim [1*], Vera Irawany [2]

[1] *Department of Anesthesiology and Intensive Care, Universitas Indonesia, Cipto Mangunkusumo General Hospital, Jakarta, Indonesia*
[2] *Department of Anesthesiology and Intensive Care, Universitas Indonesia, Fatmawati General Hospital, Jakarta, Indonesia*

***Corresponding Author**: Ida Juita Halim, Email: : ijhalim328@gmail.com

ARTICLE INFO

Article history:
Received
21 May 2026

Revised
4 July 2026

Accepted
31 August 2026

Manuscript ID:
JSOCMED-21052026-58-4

Checked for Plagiarism: Yes

Language Editor:
Rebecca

Editor-Chief:
Prof. Aznan Lelo, PhD

ABSTRACT

Introduction: Gut-origin sepsis is an important but under-recognized source of postoperative systemic inflammation in critically ill patients. Disruption of the intestinal mucosal barrier after major surgery may facilitate bacterial translocation and trigger systemic inflammatory responses, even without overt intra-abdominal infection or positive blood culture. Early recognition remains challenging because clinical manifestations are often nonspecific, underscoring the value of simple, available biomarkers.

Case Description: We report a 74-year-old man who developed early features suggestive of gut-origin sepsis after open radical cystectomy with ileal conduit for muscle-invasive bladder cancer. Despite stable macrocirculatory parameters and no vasopressor requirement, the patient exhibited persistent hyperlactatemia (6.1 mmol/L), lymphopenia (absolute lymphocyte count 711/µL), and an elevated neutrophil-to-lymphocyte ratio (NLR 16.9). Prompt admission to the intensive care unit and early targeted management—including optimized fluid resuscitation with albumin, broad-spectrum antimicrobial therapy, empirical antifungal coverage, and multimodal opioid-sparing analgesia—were followed by rapid clinical and laboratory improvement. Within 72 hours, serum lactate decreased to 2.8 mmol/L, the absolute lymphocyte count rose to 792/µL, and the NLR fell to 13.1. The patient was successfully extubated and discharged from the intensive care unit without progression to multiple organ dysfunction.

Conclusion: This case underscores the clinical value of early intensive care recognition of gut-origin sepsis using simple, routinely available biomarkers, such as serum lactate, absolute lymphocyte count, and NLR. Optimal outcomes depend on a high index of suspicion and timely, physiology-based intervention delivered through a coordinated critical care approach in high-risk postoperative patients.

Keywords

Gut-Origin Sepsis, Intensive Care Unit, Lactate, Neutrophil-to-Lymphocyte Ratio, Postoperative Sepsis

How to cite: Halim IJ, Irawany V. Gut-Origin Sepsis After Radical Cystectomy and Ileal Conduit: Early Intensive Care Unit Recognition and Targeted Management Leading to Rapid Recovery. *Journal of Society Medicine*. 2026; 5 (8): 301-305. **DOI: https://doi.org/10.71197/jsocmed.v5i8.292**

INTRODUCTION

Sepsis remains a leading cause of morbidity and mortality among critically ill patients, particularly in the postoperative setting [1,2]. Although the lungs, urinary tract, and surgical site are commonly identified as sources of infection, the gastrointestinal tract has increasingly been recognized as a potential driver of systemic inflammation and sepsis. The concept of gut-origin sepsis describes a pathological process characterized by disruption of intestinal barrier integrity, alterations in gut microbiota composition, and impaired mucosal immunity, facilitating bacterial translocation from the gut lumen to mesenteric lymphatics and systemic circulation, even in the absence of gastrointestinal pathology or positive blood culture [3-5].

Major surgical stress, prolonged anesthesia, intraoperative hypotension, and splanchnic hypoperfusion have been shown to exacerbate intestinal ischemia-reperfusion injury, which leads to mucosal hypoxia and immune dysfunction. This process promotes bacterial translocation predominantly through the mesenteric lymphatic pathway, with subsequent passage via the thoracic duct into the systemic circulation, triggering a systemic inflammatory response and subsequent organ dysfunction [6,7]. Reports describing gut-origin sepsis in postoperative urological patients, especially following radical cystectomy, remain limited, and early diagnosis often relies on subtle clinical and laboratory findings, particularly dynamic changes in simple biomarkers such as serum lactate, absolute lymphocyte count, and neutrophil-to-lymphocyte ratio (NLR), in the absence of confirmed infection. This case report aims to highlight the clinical presentation, diagnostic challenges, and the potential role of these biomarkers in facilitating early recognition of gut-origin sepsis in postoperative patients.

CASE DESCRIPTION

A 74-year-old man was admitted for elective open radical cystectomy with ileal conduit creation for muscle-invasive bladder cancer (stage T3N0Mx). His medical history included hypertension and ischemic heart disease, with prior percutaneous coronary interventions in 2021 and 2022. Preoperative assessment classified the patient as having American Society of Anesthesiologists (ASA) physical status II.

The procedure was performed under general anesthesia. The total anesthesia duration was approximately 6 hours, with a surgery time of 5 hours and 30 minutes. Throughout the intraoperative period, the patient remained hemodynamically stable without the need for vasopressor support. The estimated blood loss was 1,500 mL. Fluid therapy consisted of 2,000 mL of crystalloid, 500 mL of colloid, and transfusion of 773 mL of packed red blood cells. Intraoperative urine output was 100 mL over 6 hours (0.27 mL/kg/h). Postoperatively, the patient was transferred to the ICU for close monitoring. On admission, he was mechanically ventilated and maintained stable hemodynamics in the absence of vasopressor or inotropic therapy; the corresponding hemodynamic trend during the ICU stay is summarized in Table 1. Laboratory evaluation showed significant metabolic and inflammatory abnormalities, including a high serum lactate level of 6.1 mmol/L, an absolute lymphocyte count of 711/μL, and an NLR of 16.9. Inflammatory markers showed a C-reactive protein (CRP) level of 0.19 mg/dL and a procalcitonin level of 0.97 ng/mL.

Table 1. Hemodynamic Trends in the ICU

Parameter	Day 1	Day 2	Day 3
Blood pressure (mmHg)	92/54	115/65	127/77
Pulse (×/min)	93	69	81
Temperature (°C)	36.8	36.9	36.7
SpO_2 (%)	100	100	100
Fluid balance (mL)	+449	+1258	−2868

The initial ICU management focused on optimizing tissue perfusion and attenuating systemic inflammation. Targeted fluid resuscitation with 250 mL of 5% albumin solution was performed. Analgesic management was adjusted from opioid-based monotherapy to a multimodal analgesic regimen incorporating continuous infusions of tramadol, lidocaine, and ketamine to achieve opioid-sparing analgesia. Empirical broad-spectrum antimicrobial therapy was initiated using intravenous meropenem, following current sepsis management guidelines and standard high-dose administration. The following day, serum lactate levels remained elevated at 5.6 mmol/L, accompanied by persistent lymphopenia (absolute lymphocyte count, 638/μL) and an NLR of 16.4. Additional interventions included a repeat albumin bolus, broadening of antimicrobial coverage with the addition of amikacin, and initiation of empirical antifungal therapy using fluconazole, considering the patient's postoperative risk profile. Subsequently, the patient's clinical condition improved, and extubation was successfully performed. Within 72 h of ICU admission, laboratory parameters demonstrated marked improvement, with a decreased serum lactate level of 2.8 mmol/L, an increase in absolute lymphocyte count to 792/μL, and a reduction in NLR to 13.1. The serial laboratory data are presented in Table 2. The patient maintained stable hemodynamics without vasopressor support and adequate,

spontaneous ventilation. He was discharged to the high-care unit without evidence of progression to multiple organ dysfunction syndrome.

Table 2. Laboratory Parameters in the ICU

Parameter	Day 1	Day 2	Day 3
Haemoglobin (g/dL)	14.8	13.7	11.2
Haematocrit (%)	46.5	41.6	33.3
Leukocyte (10^3/μL)	13.4	11.8	11.8
Thrombocyte (10^3/μL)	160	120	105
Absolute Lymphocyte (/μL)	711	638	792
NLR	16.9	16.4	13.1
Serum Lactate (mmol/L)	6.1	5.6	2.8

Written informed consent was waived as this case report was retrospective in nature, all patient data were fully anonymized, and no identifiable personal information was disclosed. The report was prepared in accordance with the institutional ethical standards.

DISCUSSION

This case is notable for the early suspicion of gut-origin sepsis in a postoperative patient based on dynamic changes in simple biomarkers, despite the absence of overt hemodynamic instability or confirmed infection at the time. The presence of persistent hyperlactatemia, lymphopenia, and an elevated NLR shortly after surgery suggests early microcirculatory dysfunction and systemic inflammatory activation, consistent with the concept of gut-origin sepsis.

The gastrointestinal tract plays a central role in immune homeostasis and serves as a barrier against microbial translocation. Under surgical stress conditions, such as prolonged surgery, tissue manipulation, significant blood loss, and perioperative hypoperfusion, the intestinal mucosa becomes vulnerable to ischemia-reperfusion injury [6]. This process disrupts epithelial tight junctions, increases intestinal permeability, and promotes dysbiosis, facilitating the translocation of bacteria and endotoxins into systemic circulation [4-6]. Evidence from high-risk abdominal surgery populations shows that this translocation may occur predominantly via mesenteric lymphatic pathways, even in the absence of overt anastomotic leakage or positive blood cultures, thereby amplifying systemic inflammation and contributing to postoperative sepsis [2]. Both experimental and clinical studies have described the gut as a potential “motor of sepsis” [3]. In this patient, several established risk factors for gut-origin sepsis were present, including extensive pelvic surgery, substantial intraoperative blood loss, prolonged surgery duration, and reduced urine output, suggesting global hypoperfusion. Notably, these pathological processes occurred despite stable macrocirculatory parameters, highlighting the limitations of conventional hemodynamic monitoring in detecting early microcirculatory and splanchnic dysfunction.

Early recognition of gut-origin sepsis is challenging because of its nonspecific clinical presentation and frequent absence of definitive microbiological confirmation. In this setting, simple and readily available biomarkers may provide important diagnostic clues. Persistent elevation of serum lactate, even in the absence of hypotension, reflects impaired microcirculatory perfusion and altered cellular metabolism [8]. Lymphopenia and an elevated NLR reflect stress-induced immune dysregulation and a shift toward innate immune activation. Several studies have suggested that NLR may outperform conventional inflammatory markers in the early detection of sepsis, particularly in surgical patients [9-12]. The role of NLR as an early predictor of sepsis has also been demonstrated in critically ill patients. Evidence from a prospective cohort of critically ill acute stroke patients has shown that increased NLR is independently associated with the subsequent development of sepsis, suggesting that immune dysregulation may precede overt infection and clinical deterioration [13]. The management of suspected gut-origin sepsis requires a physiology-based approach aimed at restoring splanchnic perfusion, limiting ongoing inflammatory injury, and preventing progression to multiple organ dysfunction. In this case, early ICU admission enabled close monitoring and timely intervention. Targeted fluid resuscitation with albumin was used to support intravascular volume [14]. and microcirculatory flow

while minimizing interstitial edema [10]. Broad-spectrum antimicrobial therapy was initiated to cover enteric pathogens commonly implicated in bacterial translocation, and empirical antifungal therapy was considered in line with existing guidelines for critically ill postoperative patients at high risk of invasive fungal infection [15].

Rapid clinical and laboratory improvement, with resolution of hyperlactatemia and inflammatory abnormalities within 72 hours, suggests that early recognition and ICU-based intervention may alter gut-origin sepsis in high-risk postoperative patients. Although definitive microbiological confirmation was lacking, the clinical course and response to therapy are consistent with the pathophysiological understanding of gut-derived systemic inflammation in high-risk postoperative patients.

CONCLUSION

Gut-origin sepsis is an often-overlooked cause of early postoperative systemic inflammation after major surgery. This case shows that significant gut-derived sepsis may develop despite stable macrocirculatory parameters and no obvious infectious focus. Early recognition using readily available biomarkers, such as serum lactate, absolute lymphocyte count, and NLR, can reveal microcirculatory and immunological dysfunction. Prompt ICU admission and timely physiology-based management were associated with clinical and laboratory improvement, preventing multiple organ dysfunction and supporting early intensive care intervention.

DECLARATIONS

This case report was conducted in accordance with institutional ethical standards and the principles of the Declaration of Helsinki. Written informed consent was waived due to the retrospective nature of the report; all patient data were fully anonymized, and no identifiable personal information was disclosed.

CONSENT FOR PUBLICATION

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

FUNDING

None

COMPETING INTERESTS

The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

I.J.H. was responsible for patient management, data acquisition, and drafting the initial manuscript. V.I. contributed to the clinical supervision and critical revision of the manuscript for important intellectual content. Both authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of this work.

ACKNOWLEDGMENTS

None

REFERENCE

1. Rhodes A, Evans LE, Alhazzani W. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock: 2016. Intensive Care Med. 2017;43(3):77-304.
2. Doudakmanis C, Bouliaris K, Kolla C, Efthimiou M, Koukoulis GD. Bacterial translocation in patients undergoing major gastrointestinal surgery and its role in postoperative sepsis. World J Gastrointest Pathophysiol. 2021;12(6):106-114.
3. Deitch EA. Gut-origin sepsis: evolution of a concept. Surgeon. 2012;10(6):6-350.

4. Fink MP. Intestinal epithelial hyperpermeability: update on the pathogenesis of gut mucosal barrier dysfunction in critical illness. Curr Opin Crit Care. 2003;9(2):51-143.
5. Assimakopoulos SF, Triantos C, Thomopoulos K. Gut-origin sepsis in critically ill patients: pathophysiology and treatment. Infection. 2018;46(6):60-751.
6. Grootjans J, Lenaerts K, Buurman WA, Dejong CHC, Derikx JPM. Life and death at the mucosal-luminal interface: new perspectives on human intestinal ischemia–reperfusion. World J Gastroenterol. 2016;22(9):70-2760.
7. Fay KT, Ford ML, Coopersmith CM. Intestinal microenvironment in sepsis. Biochim Biophys Acta Mol Basis Dis. 2017;1863(10 Pt B):83-2574.
8. Bakker J, Gris P, Coffernils M. Serial blood lactate levels can predict the development of multiple organ failure following septic shock. Am J Surg. 1996;171(2):6-221.
9. Zahorec R. Ratio of neutrophil to lymphocyte counts—rapid and simple parameter of systemic inflammation and stress in critically ill. Bratisl Lek Listy. 2001;102(1):5-14.
10. Gurol G, Ciftci IH, Terizi HA, Atasoy AR, Ozbek A, Koroglu M. Are there standardized cutoff values for neutrophil-lymphocyte ratios in bacteremia or sepsis? J Microbiol Biotechnol. 2019;29(8):7-1243.
11. Drăgoescu AN, Pădureanu V, Stănculescu AD, Chiuțu LC, Tomescu P, Geormăneanu C, et al. Neutrophil to lymphocyte ratio (NLR)—a useful tool for the prognosis of sepsis in the ICU. Biomedicines. 2022;10(1):75.
12. Liu X, Shen Y, Wang H, Ge Q, Fei A, Pan S. Prognostic significance of neutrophil-to-lymphocyte ratio in patients with sepsis: a prospective observational study. Mediators Inflamm. 2016;2016:8191254.
13. Irawany V, Lolobali MC. Neutrophil-to-lymphocyte ratio as a new predictor of sepsis in critically ill stroke patients. Open Access Maced J Med Sci. 2022;10(B):21-1118.
14. Caironi P, Tognoni G, Masson S. Albumin replacement in patients with severe sepsis or septic shock. N Engl J Med. 2014;370(15):21-1412.
15. Pappas PG, Kauffman CA, Andes DR, Clancy CJ, Marr KA, Ostrosky-Zeichner L, et al. Clinical practice guideline for the management of candidiasis: 2016 update by the Infectious Diseases Society of America. Clin Infect Dis. 2016;62(4):e1-50.
16. Hutasuhut AF, Rismawan B. Management of septic shock secondary to submandibular phlegmon and ventilator-associated pneumonia in the intensive care unit. Journal of Society Medicine. 2025;4(9):284-291.
17. Bernadeth B, Erlangga ME. Management of a critically ill post-cesarean section patient with antepartum hemorrhage due to placenta previa totalis in a G2P1A0 at 27–28 weeks' gestation with severe preeclampsia, HELLP syndrome, pulmonary edema, stage 2 acute kidney injury, and hypoalbuminemia. Journal of Society Medicine. 2025;4(7):227-231.
18. Dalimunthe SSM, Daramawan E, Taufiqurrahman R, Lubis B, Lubis AP, Nadeak RF, Purwaamidjaja DB, Chandra F. Bronchoscopy, ultrasound, and video laryngoscopy as guidelines for percutaneous dilatation tracheostomy (PDT) in patients with difficult airway suspected of laryngeal tumor. Journal of Society Medicine. 2026;5(6):241-247.
19. Sahat D, Kestriani ND, Ismandiya AI. Comprehensive management of septic shock secondary to intra-abdominal infection complicated by acute respiratory distress syndrome: a case report. Journal of Society Medicine. 2026;5(2):46-51.