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Suspected Gut-Origin Sepsis Following Pelvic Exenteration for Stage IIIB Cervical Cancer: A Case Report

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Gut-Origin Sepsis; Pelvic Exenteration; Cervical Cancer; Septic Shock; Multiple Organ Dysfunction Syndrome

Abstract

Sepsis is a life-threatening condition that may develop after major surgical procedures, particularly in patients who experience massive hemorrhage, prolonged tissue hypoperfusion, and gastrointestinal barrier dysfunction. This case report describes suspected gut-origin sepsis in a 33-year-old woman with stage IIIB cervical cancer who underwent pelvic exenteration after incomplete chemoradiotherapy. The surgical procedure lasted 11.5 hours and was complicated by massive intraoperative blood loss of approximately 5,000 mL, requiring extensive blood transfusion and intensive postoperative support. After surgery, the patient developed septic shock, acute kidney injury, severe metabolic acidosis, and multiple organ dysfunction syndrome. Intensive care management included vasopressor therapy, mechanical ventilation, continuous renal replacement therapy (CRRT), broad-spectrum antimicrobial therapy, fluid management, and nutritional support. Although definitive evidence of bacterial translocation from the gastrointestinal tract was unavailable, the combination of extensive pelvic surgery, prolonged tissue hypoperfusion, elevated lactate levels, and persistent systemic inflammation suggested gastrointestinal barrier dysfunction and possible gut-origin sepsis. Despite aggressive multidisciplinary management, the patient experienced recurrent cardiac arrest and ultimately died on the tenth day of intensive care. This case highlights the importance of early recognition of gastrointestinal barrier dysfunction, timely hemodynamic resuscitation, appropriate source control, antimicrobial therapy, and comprehensive organ support in critically ill postoperative patients at risk of sepsis and multiple organ dysfunction.

INTRODUCTION

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection (M. Cao et al., 2023; Jacobi, 2022; Joosten et al., 2024; Z. Liu et al., 2024; Zhang et al., 2024). It is a serious complication that may occur after high-risk surgical procedures, particularly in patients with risk factors such as massive hemorrhage, massive transfusion, immune dysfunction, and gastrointestinal ischemia. In patients undergoing high-risk surgery involving the gastrointestinal tract, the risk of sepsis may increase because impaired intestinal mucosal integrity can facilitate bacterial translocation. Although the concept of gut-origin sepsis has been widely discussed in the literature, its diagnosis in clinical practice remains challenging because no gold-standard diagnostic method can directly confirm bacterial translocation from the gastrointestinal tract. In most cases, the diagnosis is considered based on a combination of predisposing factors, the clinical course, and exclusion of other more apparent sources of infection. Therefore, this case report aimed to describe suspected gut-origin sepsis in a patient who underwent pelvic exenteration complicated by massive hemorrhage and prolonged shock and to discuss the mechanisms of intestinal barrier dysfunction that may contribute to the development of sepsis and multiple organ dysfunction.

Gut-origin sepsis refers to a proposed mechanism in which disruption of the intestinal barrier and alterations in the gut microbiota facilitate the translocation of bacteria, microbial products, or inflammatory mediators into the systemic circulation (Bao et al., 2026; Chancharoenthana et al., 2023; Charitos et al., 2025; X. Liu et al., 2026; Tian & Li, 2025; Xu, 2025). The principal mechanisms include increased intestinal permeability resulting from hypoperfusion, ischemia, and dysbiosis, which may be exacerbated by critical illness and antimicrobial exposure. These processes may promote an exaggerated systemic inflammatory response and contribute to septic shock and multiple organ dysfunction (Bitsadze et al., 2025; Z. Liu et al., 2024; Miguel et al., 2026; Vella et al., 2025; Wu et al., 2024).

This case involved a patient with stage IIIB cervical cancer who underwent pelvic exenteration that included hysterectomy, ileal conduit formation, repair of a rectal laceration, and creation of a colostomy. The procedure was complicated by massive intraoperative blood loss of approximately 5,000 mL, requiring extensive blood transfusion (P. Cao et al., 2026; Coccolini et al., 2024; Neethirajan & Akilandeswari, 2026; Zhou et al., 2024). Postoperatively, the patient developed septic shock with suspected involvement of a gut-origin mechanism following major surgery, massive hemorrhage, and prolonged tissue hypoperfusion. Intensive care management included hemodynamic support, mechanical ventilation, continuous renal replacement therapy (CRRT), broad-spectrum antimicrobial therapy, and comprehensive organ support. Lung-protective ventilation strategies were used to reduce ventilator-associated lung injury and support respiratory function in the setting of critical illness. This case report discusses the potential role of gastrointestinal barrier dysfunction in the development of suspected gut-origin sepsis, the intensive care management provided, and the clinical implications for patients undergoing major surgery complicated by massive hemorrhage and multiple organ dysfunction.

The purpose of this case report was to describe the clinical features of suspected sepsis associated with intestinal barrier dysfunction following pelvic exenteration complicated by massive hemorrhage and prolonged shock, as well as to examine the pathophysiological mechanisms underlying intestinal barrier dysfunction and their potential contribution to sepsis and multiple organ dysfunction. This report is expected to increase clinical awareness of possible gut-origin sepsis in postoperative patients with massive hemorrhage and hemodynamic instability, provide insight into comprehensive diagnostic and intensive care approaches—including hemodynamic optimization, antimicrobial therapy, and organ support—and provide a basis for further research on biomarkers of intestinal mucosal injury and the role of the gut microbiota in postoperative sepsis.

METHOD

The research method described the analytical procedures used to address the research problem. The method section included the main analytical or characterization tools, the research setting, the number of participants or samples, data collection procedures, data processing and analysis techniques, and the criteria used to assess research outcomes. Commonly established methods were described concisely with appropriate references rather than explained in unnecessary detail. Experimental or procedural steps were presented in declarative form rather than as commands.

CASE REPORT

Patient Identity

1. Name: Mrs. E
2. Age: 33 years old
3. Gender: Female
4. Main diagnosis: Cervical Ca 3B post chemoradiation.

Disease History

1. **History of Past Illness:**
   a. Hypertension since 5 years ago, is not controlled regularly.
   b. AKI (post Renal) with a history of HD on demand twice before surgery.
   c. There is no history of diabetes mellitus or other chronic diseases.
2. **Case illustration:**

The patient came to the hospital with complaints of vaginal bleeding that had been getting heavier since a few weeks before being admitted to the hospital. The patient also experienced complaints of lower back pain that radiated to the lower abdomen. The patient had a history of partial chemoradiation for stage 3B cervical cancer but it was not complete. The patient underwent elective Pelvic exenteration surgery which included *subtotal hysterectomy, ileal conduit, rectal laceration repair, and colon stoma*. During the surgery lasting 11 hours and 30 minutes, the patient experienced massive bleeding of up to 5000 ml and entered 6250 cc fluid, received a transfusion of PRC 2280 ml, TC 10U (500cc), and FFP of 1052 ml. After surgery, the patient experienced hypovolemic shock which then developed into septic shock with suspected involvement of intestinal barrier disorders (gut barrier dysfunction).

The patient was admitted to the ICU after pelvic excerpt surgery on February 3, 2025. After an extensive surgical procedure, the patient develops an unstable hemodynamic condition with severe hypotension, requiring vasopressor support. In addition, patients must be fitted with mechanical ventilation with SIMV mode, 60% FiO2%, and 5 cmH2O PEEP to support their breathing. In the first 24 hours, the patient showed a fairly high increase in fluid balance (+4000 ml), while the initial examination showed the presence of severe metabolic acidosis with an increase in lactate levels of up to 14 mmol/L.

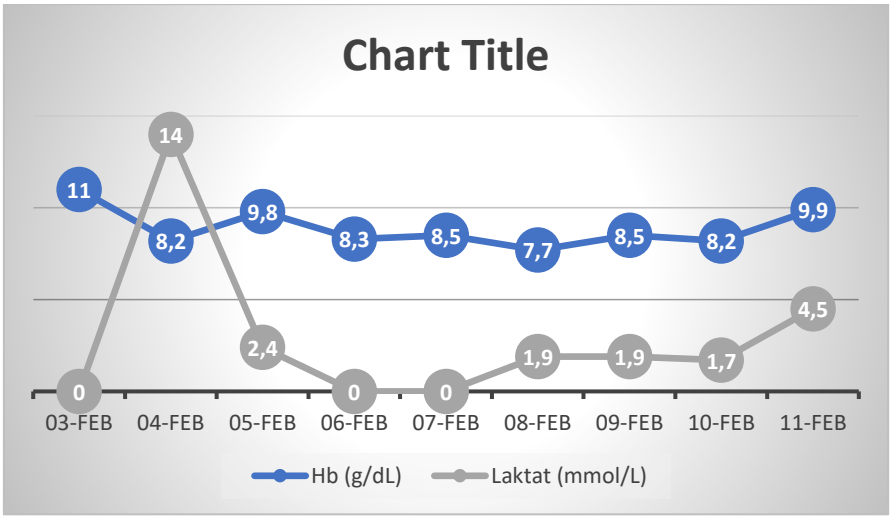


Figure 1. Development of Hemoglobin amounts, and lactate levels, during ICU
Source: Patient medical records, processed by the authors (2025)

In the first three days in the ICU, the patient developed persistent sepsis with hypotension that required a combination of norepinephrine and vasopressin to maintain his blood pressure. Given the existence of acute kidney failure with anuria and fluid overload, CRRT (Continuous Renal Replacement Therapy) therapy begins on the second day in the ICU (February 4, 2025). Laboratory monitoring showed an improvement in lactate levels from 14.0 mmol/L on February 4 to 3.4 mmol/L on February 6. However, at the same time, D-dimer levels increase drastically, indicating the presence of secondary coagulation disorders. The initial antibiotic given was meropenem, and blood and urine cultures were taken for further evaluation.

On February 6, the patient's extubation plan was postponed after the patient experienced a sudden event of bradycardia following the ETT suction procedure. The patient experiences episodes of seizures, severe coughing, and abundant sputum production, which is then followed by severe bradycardia until cardiac arrest occurs. Cardiopulmonary resuscitation was immediately carried out for 4 cycles, with the administration of adrenaline 4 times, as well as an additional bicarbonate of 75 mEq and 1 gram of calcium gluconate. After the procedure, the patient experienced a return of spontaneous circulation (ROSC), but remained in critical condition and had to be retubated with the help of a boogie to ensure that the airway remained patent.

Table 1. Development of Laboratory Results, while in the ICU

Date	Hb (g/dL)	Ht (%)	Leukosi de (/µL)	Platelet s (/µL)	LA (/µL)	RN L	Lactate (mmol/L)	Ureum (mg/dL)	Creatin ine (mg/dL)
03-Feb	11.0	35.2	13.100	191.000	670	17.5	-	-	-
04-Feb	8.2	27.0	25.000	249.000	376	60.1	14.0	79.3	2.92
05-Feb	9.8	30.7	11.700	104.000	398	26.9	2.4	64.3	1.88
06-Feb	8.3	26.1	10.000	110.000	348	25.4	-	76.1	2.18
07-Feb	8.5	26.8	10.900	98.000	491	19.8	-	48.9	1.4
08-Feb	7.7	24.3	7.600	105.000	580	11.2	1.9	52.1	1.39
09-Feb	8.5	26.8	5.300	109.000	484	9.1	1.9	-	-
10-Feb	8.2	25.6	5.300	117.000	358	13	1.7	49.3	1.06
11-Feb	9.9	31.5	10.200	140.000	451	20.8	4.5	51.4	1.24

Source: Patient medical records, processed by the authors (2025)

Post-ROSC, patients experienced tachycardia with a picture of ventricular tachycardia (VT) more than 200 times per minute who did not respond to administration of 5 mg IV metoprolol. Therefore, cardioversion is performed with 50 Joules which successfully lowers the heart rate to 120 times per minute. After initial stabilization, the patient still required intensive hemodynamic support with an infusion of norepinephrine of 0.3 mcg/kg/min.

In the following days, the patient developed pneumonia, which led to a change in antibiotic regimen from meropenem to a combination of meropenem and vancomycin. Despite improved lactate levels and stabilization of fluid balance, CRP and procalcitonin levels remained high, indicating persistent systemic inflammation.

Table 2. Development of fluid balance, while in the ICU

Date	Balance	BK	Uo cc/24jam
04-Feb	512	512	
05-Feb	599	1111	270
06-Feb	1664	2775	480
07-Feb	-1570	1205	2190
08-Feb	-1276	-71	2810
09-Feb	-240	-311	1880
10-Feb	320	9	1700
11-Feb	1365	1374	1670
12-Feb	-944	430	3480

Source: Patient medical records, processed by the authors (2025)

On February 11, after a re-evaluation, the patient was scheduled to weaning the ventilator in preparation for the experimental ectubation and if that failed, a tracheostomy was considered. However, before the procedure was performed, the patient experienced sudden bradycardia which again progressed to cardiac arrest. CPR was again performed for 30 minutes, but did not succeed in restoring the patient's spontaneous circulation. The patient was finally declared dead on February 12, 2025 due to the continued deterioration of his condition.

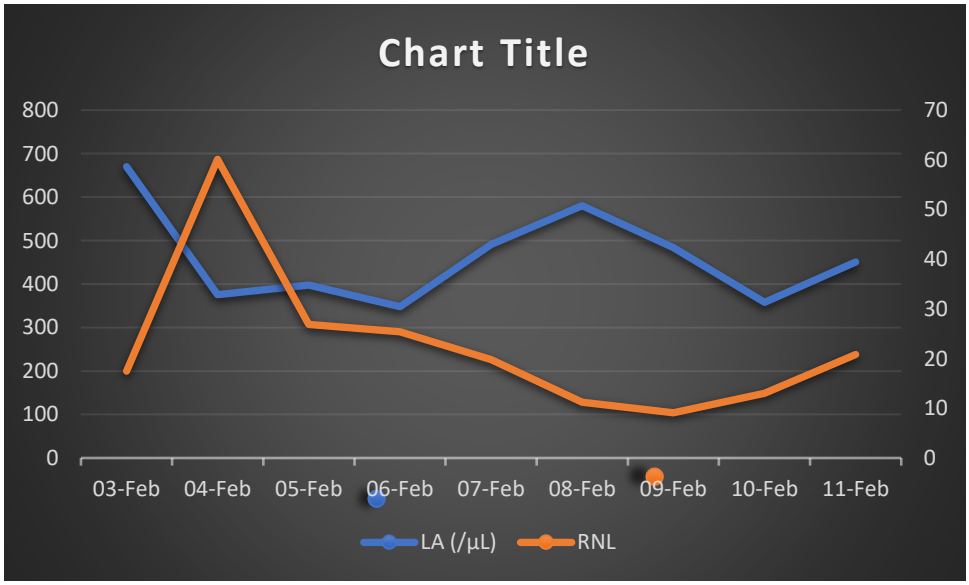
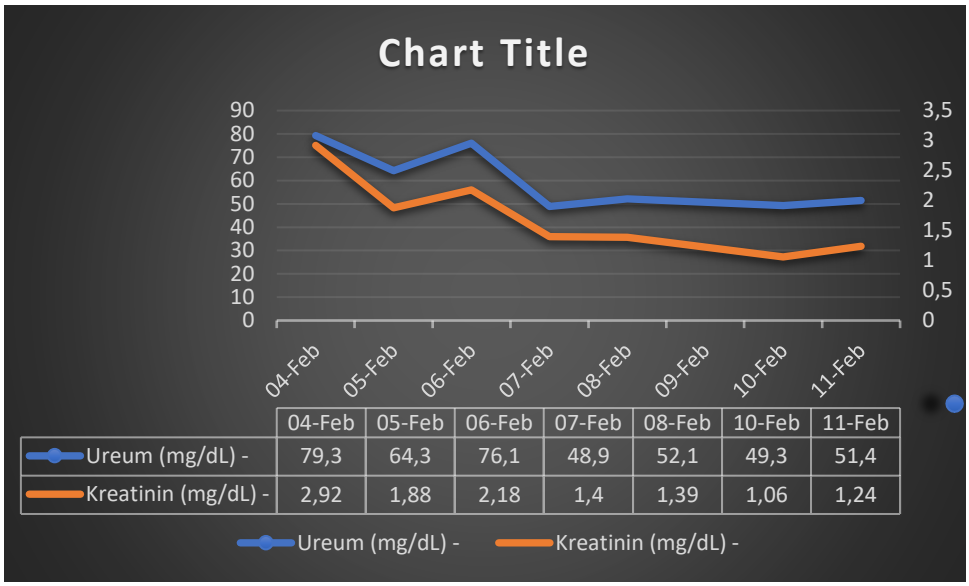


Figure 2. Development of absolute Lymphocytes and RNL, while in the ICU

Source: Patient medical records, processed by the authors (2025).



	04-Feb	05-Feb	06-Feb	07-Feb	08-Feb	10-Feb	11-Feb
Ureum (mg/dL) -	79,3	64,3	76,1	48,9	52,1	49,3	51,4
Kreatinin (mg/dL) -	2,92	1,88	2,18	1,4	1,39	1,06	1,24

Figure 3. Development of urea, creatinine, eGFR levels during treatment

Source: Patient medical records, processed by the authors (2025).

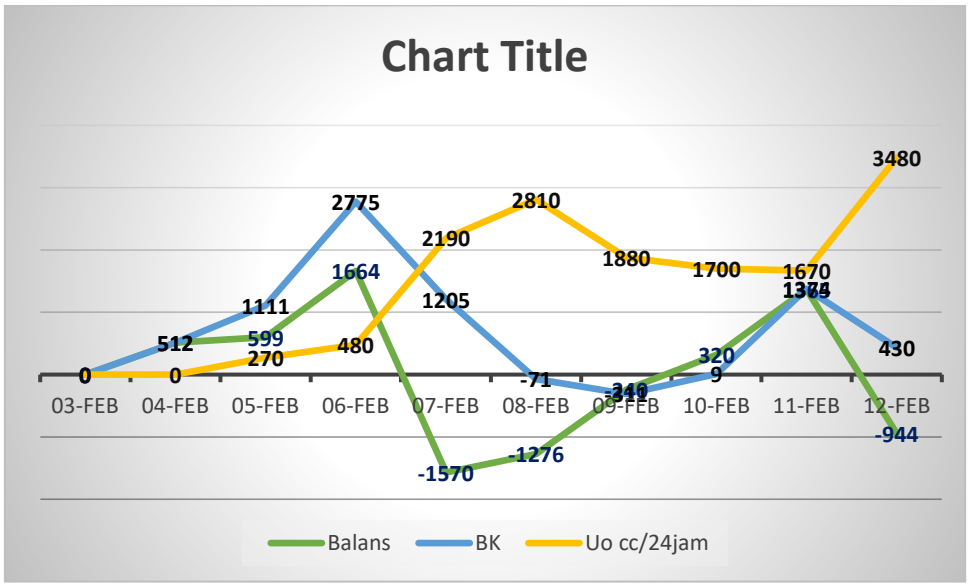


Figure 4. Fluid balance, urine *output*, and cumulative balance in the ICU

Source: Patient medical records, processed by the authors (2025).

Table 3. Chronology of the Patient's Disease Journey in the ICU

Date	Clinical Events	TTV, Liquid Balance & BK	Medical Intervention
3 Feb (Day 1, 22.22 WIB - ICU Admission)	The patient was admitted to the ICU post **Pelvic Exenteration** surgery with intraoperative massive bleeding (**5000 ml**).	**TD:** 124/70 mmHg, **HR:** 118x/min, **RR:** 20x/min, **SpO2:** 100%, **Temperature:** 36°C.	**Vasopressor:** Vascon 0.3mcg/kgbb/mnt, **Analgesia:** Tramadol + Ketamin, **Nutrition:** Smofkabiven 1448/24 hours.
4 Feb (Day 2)	Hemodynamics The patient is still unstable, requiring a vasopressor. **Septic shocked by suspected gut-origin sepsis**. CRRT is carried out due to AKI and sepsis	**TD:** 150/100 mmHg, **HR:** 142x/min, **RR:** 20x/min, **SpO2:** 100%. **Balance:** +512 ml, **BK:** 512 ml.	**CRRT dimulai**, Weaning vasopressor, **AB:** Meropenem, Amikasin, Fluconazole.
5 Feb (Day 3)	The patient is still **in critical condition**, hemodynamics **are starting to stabilize**. **A sign of hypokalemia**, **Hb drops after transfusion**. **Low urine output**.	**TD:** 109/62 mmHg, **HR:** 110x/min, **RR:** 14x/min. **UO:** 270 ml/24 jam. **Balance:** +599 ml, **BK:** 1111 ml.	**PRC transfusions**, **hypokalemia correction**, *ventilator weaning*, advanced antibiotics.
6 Feb (Hari 4 - Cardiac Arrest & ROSC)	The patient experienced **sudden bradycardia → Asistole → CPR**. **ROSC** after **Adrenaline 4x, Bicnat, Ca-gluconate**. **VT >200x/min**, given **Metoprolol IV (failed) → Cardioversion 50 Joules → HR 120 bpm**.	**TD:** 120/68 mmHg, **HR:** 115x/min, **RR:** 15-18x/min. **UO:** 480 ml/24 hours. **Balance:** +1664 ml, **BK:** 2775 ml.	**CT Scan of the Head:** No stroke/bleeding. **A cardiology ECHO** is performed. Vasopressors remain high.
10 Feb (Day 8)	The patient shows improvement in hemodynamics, **extubation plan**. **Bronchoscopy was performed** → a thick brownish-yellow secretion **was found**. Additional diagnoses: **Nosocomial pneumonia, Severe lymphopenia**.	**TD:** 111/82 mmHg, **HR:** 89x/min, **RR:** 18x/min. **UO:** 1700 ml/24 jam. **Balance:** +320 ml, **BK:** 9 ml.	**Extubation discourse plan**, Bronchoscopy + BAL culture. Antibiotic regimen: **Meropenem, Amikasin, Fluconazole**.
11 Feb (Day 9 - NSTEMI Worsening)	Post **bronchoscopy patients found thick** greenish-yellow **secretions**, hyperemic mucosa, **blood in LAKi**. Additional diagnosis: **NSTEMI. Tracheostomy discourse**.	**TD:** 83/45 mmHg, **HR:** 98x/min, **RR:** 20x/min. **UO:** 1670 ml/24 jam. **Balance:** +1365 ml, **BK:** 1374 ml.	**AB:** Meropenem, Amikasin, Fluconazole. **Analgesic:** Fentanyl, Ketamine, Lidocaine.
12 Feb (Day 10 - Terminal Cardiac Worsening & Stop)	**Drastic decrease in consciousness**, **hyperthermia 41.1°C**, **hemodynamic failure**. **Desaturation 88%, TD dropped to 64/45 mmHg**.	**TD** 106/67 →80/40 **HR:** 130x/min, **RR:** 25-30x/min. **UO:** 3480 ml/24 jam. **Balance:** -944 ml, **BK:** 430 ml.	**Vasopressor is elevated (NE 0.3 mcg/kg/min), administration of Albumin 5%, elective tracheostomy is delayed.**
12 Feb	**HR drops drastically to**	**HR:** 50x/min → **0x/min**,	**The patient was**

(13.00 - Terminal Cardiac Stop & DNR)	**50x/minute, pulse is weak, TD is illegible, saturation is 70%. The patient's husband declared DNR**.	**TD:** Illegible, **SpO2:** 70% **→ 0%.**	**declared dead at 13.30 WIB, the ventilator was turned off, the ETT was removed**.

Source: Patient medical records, processed by the authors (2025)

RESULTS AND DISCUSSION

This case shows the complexity of the management of patients after pelvic exenteration with massive bleeding that develops into septic shock and multiorgan dysfunction as well as the suspicion of gut origin sepsis. Factors such as bacterial translocation, disruption of the intestinal mucosal barrier, as well as gut-lung axis involvement play a major role in exacerbating systemic inflammation that eventually leads to multi-organ failure. Intensive management efforts that include hemodynamic stabilization, strict monitoring of fluid balance, and culture-based antibiotic therapy are crucial aspects in the management of patients with severe sepsis in the ICU.

In these cases, gut-origin sepsis is one of the most likely etiologies to consider. The suspicion is based on the existence of pelvic major surgery that lasted for 11.5 hours, massive bleeding of 5000 mL, the need for massive transfusion, tissue hypoperfusion characterized by an increase in lactate up to 14 mmol/L, and the development of multiorgan dysfunction. However, the diagnosis of gut-origin sepsis cannot be definitively confirmed because there are no biomarkers of intestinal mucosal injury or microbiological examinations that directly prove bacterial translocation from the gastrointestinal tract. Suspected bacterial translocation from the gastrointestinal tract likely contributes to systemic inflammation characterized by increased PCT and CRP, although these increased biomarkers are not specific to *gut-origin sepsis*. In these patients, the involvement of the gut-lung axis may have contributed to the deterioration of lung function. Although nosocomial pneumonia is a more likely cause of later lung infections, systemic inflammation derived from a disorder of the intestinal barrier can aggravate the pulmonary inflammatory response and increase susceptibility to secondary infections.

In addition, prolonged hypoperfusion conditions due to hypotension also contribute to intestinal mucosal ischemia that accelerates bacterial translocation. In this condition, the *endothelial glycocalyx shedding* mechanism is thought to play a role, so it can lead to increased vascular permeability and aggravate the sepsis condition. In addition, the worsening can also be caused by an increase in pro-inflammatory cytokines such as TNF-α, IL-6, and IL-10, which will exacerbate systemic inflammatory response syndrome (SIRS) and can develop into multiple organ dysfunction syndrome (MODS).

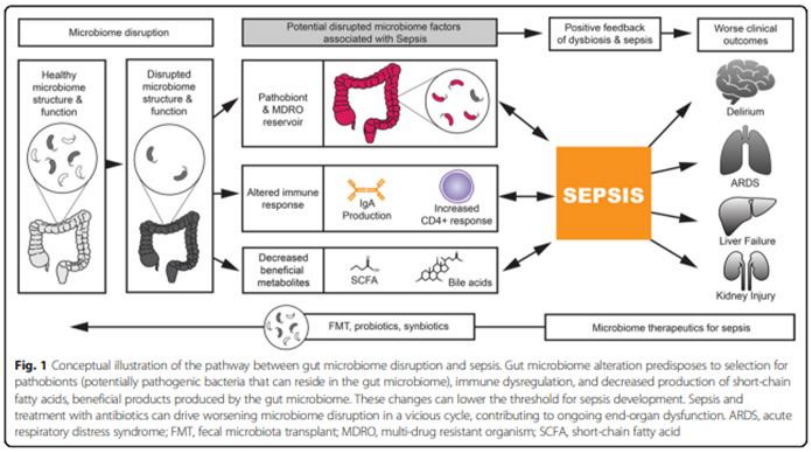


Fig. 1 Conceptual illustration of the pathway between gut microbiome disruption and sepsis. Gut microbiome alteration predisposes to selection for pathobionts (potentially pathogenic bacteria that can reside in the gut microbiome), immune dysregulation, and decreased production of short-chain fatty acids, beneficial products produced by the gut microbiome. These changes can lower the threshold for sepsis development. Sepsis and treatment with antibiotics can drive worsening microbiome disruption in a vicious cycle, contributing to ongoing end-organ dysfunction. ARDS, acute respiratory distress syndrome; FMT, fecal microbiota transplant; MDRO, multi-drug resistant organism; SCFA, short-chain fatty acid

Figure 5. Conceptual illustration of the pathway between gut microbiome disorders and sepsis

Source: Adelman, M.W., Woodworth, M.H., Langelier, C. et al. The gut microbiome's role in the development, maintenance, and outcomes of sepsis. Crit Care 24, 278 (2020). https://doi.org/10.1186/s13054-020-02989-1

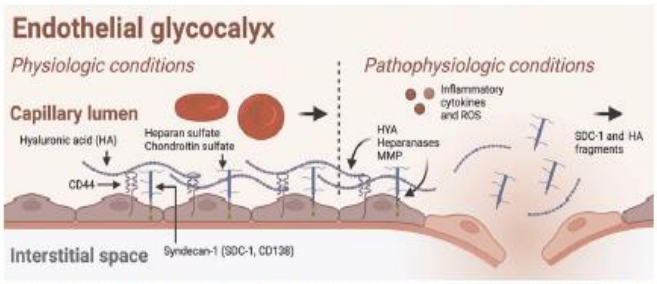


FIG. 1. **Representation of the EGL containing HA anchored to its cell surface receptor, CD44, and syndecan-1 (SDC-1, CD138), with associated heparan sulfate and chondroitin sulfate, during physiologic and pathophysiologic conditions.** HA and SDC-1 are cleaved by HYA, heparanases, and MMP during inflammation or mechanically disrupted during tissue injury, resulting in EGL component shedding into the blood stream (created on Biorender.com). EGL, endothelial glycocalyx layer; HA, hyaluronic acid; HYA, hyaluronidases (HYA); MMP, matrix metalloproteinases.

Figure 6. Comparison of *Endothelial Glycocalyx Layer* (EGL) in physiological conditions with pathophysiological conditions

Source: Carmichael, S. P., Appelbaum, R. D., Renaldo, A., Hauser, N., Rahbar, E., & Nunn, A. M. (2023). Endothelial Glycocalyx Shedding in Intra-Abdominal Sepsis: A Feasibility Study. SHOCK, 59(4), 540–546. https://doi.org/10.1097/SHK.0000000000002079

Several studies have stated that the mesenteric lymphatic system plays an important role in the spread of substances from the intestine to other organs, including the lungs. After intestinal tissue damage, lipophilic and hydrophilic components in the mesenteric lymphatic system can activate endothelial cells, neutrophils, as well as monocytes or macrophages, as shown in Figure 2, These components can cause endothelial barrier dysfunction, slow down neutrophil apoptosis, as well as inhibit erythrocyte deformability and dendritic cell function. This mechanism contributes to systemic inflammation and acute damage to lung tissue.

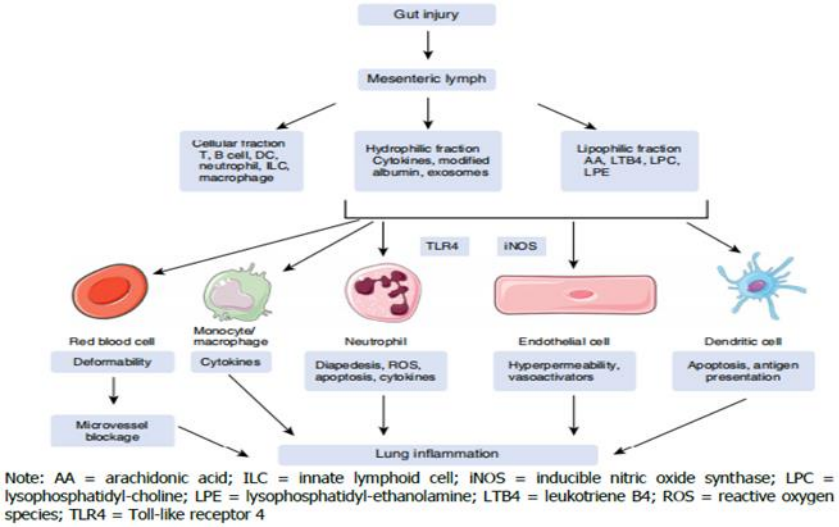


Figure 7. Mechanism of inflammation of the Lungs through mesenteric lymphatic after intestinal injury

Source: Indari, A., Rasmin, M., & Syam, A. F. (2023). Gut-Lung Axis. Respiratory Science, 3(2), 132-143. https://doi.org/10.36497/respirsci.v3i2.68

Optimal early management interventions are needed to prevent further complications, including targeted therapies that take into account microbiota homeostasis and fluid balance. Early antibiotic selection using meropenem and amicasin, then plus vancomycin was tailored broad-spectrum to handle possible anaerobic and aerobic infections of the digestive system. However, antibiotic de-escalation should be carried out based on microbiological cultures to avoid further resistance and disruption of the gut microbiota. the results of BAL culture in this patient have not yet come out, so the de-escalation strategy cannot be applied

Enteral feeding should be started immediately after stabilization of the patient to maintain mucosal integrity. If the patient is intolerant to the usual oral diet, use an immunonutritional formula such as peptamen. These patients received peptamen and Smofkabiven, but their nutritional status remained a challenge.

Monitoring-based resuscitation targets can optimize tissue oxygenation balance. Use hemodynamic targets with a MAP > 65 mmHg, and lactate monitoring. Fluid balance is essential in the management of these patients. Crystalloid fluid is administered carefully to avoid *fluid overload* that can worsen intestinal edema. The choice of Albumin as a resuscitating fluid can provide a protective effect against glycocalyx. The management of negative fluid balance achieved after day 5 is an important factor in the recovery of this patient.

The Role of CRRT in Sepsis and AKI Management

Objectives of CRRT Implementation:

1. Reduces fluid overload which can worsen intestinal edema and increase bacterial translocation.
2. Lowers levels of pro-inflammatory cytokines such as TNF-α, IL-6, and IL-10 which can worsen the systemic inflammatory response.
3. Facilitates the elimination of endotoxins and other inflammatory mediators, which often play a role in cytokine storms in severe sepsis.

4. Balances electrolytes and acid-base status, especially in the face of hyperkalemia and severe metabolic acidosis.

In principle, sepsis management is still focused on timely identification or diagnosis of sepsis, initiation of broad spectrum antimicrobials, control of the source of infection and hemodynamic resuscitation, and therapy can also be added to modulate the immune response when needed.

Case Limitations

The interpretation that sepsis in this patient originated in the gastrointestinal tract has some limitations. Biomarkers of intestinal mucosal damage (e.g., intestinal fatty acid-binding protein/I-FABP, or endotoxin activity assay) are not available. In addition, no microbiota analysis or sequencing examination can confirm the translocation of bacteria from the gastrointestinal tract to systemic circulation. Therefore, *the involvement of gut-origin sepsis* in this case, is a clinical conjecture based on strong risk factors and the course of the patient's disease.

CONCLUSION

This case illustrates the complexity of managing patients after pelvic exenteration complicated by massive hemorrhage, septic shock, and multiple organ dysfunction. Although gut-origin sepsis could not be definitively confirmed, the combination of major surgery, prolonged tissue hypoperfusion, and risk factors for intestinal barrier dysfunction supported the possible involvement of this mechanism in the patient’s clinical course. This case highlights the importance of early identification of risk factors for intestinal barrier dysfunction, optimization of hemodynamic resuscitation, appropriate source control, antimicrobial therapy, and comprehensive organ support in critically ill patients after major surgery.

Based on this case, careful assessment of intestinal barrier dysfunction may be considered when clinically appropriate, including the use of investigational biomarkers such as intestinal fatty acid-binding protein (I-FABP) where available. Hemodynamic resuscitation should be individualized, with careful fluid management to minimize fluid overload and tissue edema. Early enteral nutrition should be considered when clinically feasible to support gastrointestinal function, while antimicrobial therapy should be reassessed and de-escalated according to microbiological findings and the patient’s clinical response. Multidisciplinary collaboration among surgeons, intensivists, clinical nutrition specialists, and other relevant healthcare professionals is essential. Further prospective studies are warranted to evaluate diagnostic biomarkers and targeted interventions for preventing or mitigating suspected gut-origin sepsis in critically ill postoperative patients.

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