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Management of Intra-Abdominal Infection Patients with Septic Shock in the ICU

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ABSTRACT

Introduction: Intra-abdominal infections (IAIs) complicated by septic shock pose a critical challenge in intensive care units (ICUs) due to high morbidity and mortality. Effective management requires rapid diagnosis, source control, and optimized antimicrobial therapy. This case report describes the multidisciplinary approach to managing IAI with septic shock, highlighting evidence-based strategies.

Case Description: A 59-year-old male presented with septic shock secondary to generalized peritonitis caused by an incarcerated hernia. Initial management included fluid resuscitation with 1,500 mL Ringer's lactate, norepinephrine, and broad-spectrum antibiotics (meropenem and metronidazole). Emergency laparotomy within 4.5 hours achieved source control by evacuating 300 mL of purulent peritoneal fluid. ICU care involved mechanical ventilation, analgesia (fentanyl, paracetamol), sedation (midazolam, transitioned to dexmedetomidine), and enteral nutrition starting on day three. Multidisciplinary collaboration facilitated hemodynamic stabilization, extubation on day five, and transfer to the high-care unit on day six, with significant clinical improvement.

Conclusion: Successful management of IAI with septic shock hinges on timely source control, appropriate antimicrobial therapy, and comprehensive ICU care. The absence of microbiological cultures, a key limitation, underscores the need for improved diagnostic access in resource-limited settings. Adherence to evidence-based protocols and multidisciplinary expertise are critical to improving survival. Future efforts should focus on enhancing diagnostics to optimize therapy and reduce antimicrobial resistance.

Keywords

Intra-Abdominal Infection, Septic Shock, Intensive Care Unit, Source Control, Antimicrobial Therapy, Multidisciplinary Care.

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INTRODUCTION

Intra-abdominal infections (IAIs) are a significant cause of sepsis and septic shock, posing a critical challenge to surgeons, intensivists, and other related disciplines worldwide [1]. Despite advancements in preventive measures, such as adherence to the Joint Commission International accreditation standards, sepsis management aligned with the Surviving Sepsis Campaign (SSC) guidelines, and rational antibiotic use, IAIs remain a persistent issue in Indonesia [1–4].

The global mortality rate for abdominal sepsis ranges from 3% to 42%, reflecting the severity and variability of outcomes [5]. In Indonesia, data from six major hospitals indicate that the primary sources of IAIs include appendicitis (26.64%), gastric and duodenal ulcers (22.70%), small intestine (11.84%), large intestine (13.16%), postoperative complications (9.54%), and other causes (16.12%) [5]. Microbiological cultures commonly identify Escherichia coli (35.41%), Klebsiella pneumoniae (13.44%), Enterobacter cloacae (9.34%), Proteus mirabilis (8.69%), and Enterococcus faecalis (7.87%) as the predominant pathogens [5]. Intra-abdominal sepsis originates from infections within the abdominal cavity, often leading to diffuse or

localized peritonitis [6]. The gastrointestinal tract, frequently described as the "motor of sepsis," harbors a high bacterial load, underscoring the need for early diagnosis to prevent the progression to severe sepsis or septic shock [6]. Complications such as acute respiratory distress syndrome (ARDS), characterized by acute-onset respiratory failure within seven days of an insult, bilateral opacities on chest radiography, and hypoxemia not attributed to cardiac failure, further elevate mortality risk [7]. ARDS management often necessitates mechanical ventilation with a lung-protective strategy to mitigate hypoxemia [7]. Delayed diagnosis and inadequate source control significantly increase mortality, emphasizing the importance of prompt surgical intervention and aggressive management [6].

Effective management of IAI with septic shock and ARDS in the intensive care unit (ICU) requires a multifaceted approach, including fluid and nutritional support, analgesia, sedation, thrombosis prophylaxis, glycemic control, stress ulcer prevention, and targeted antibiotic therapy [6]. Additionally, strategies to minimize ventilator-associated complications, such as ventilator-associated pneumonia (VAP), are critical for improving outcomes [6]. This case report examines the diagnosis and management of a patient with an intra-abdominal infection complicated by septic shock and ARDS in the ICU, highlighting evidence-based strategies to optimize clinical outcomes.

CASE DESCRIPTION

A 59-year-old male patient, identified as Mr. T (medical record number: 0002124648), with a body weight of 65 kg and height of 160 cm, presented to the emergency department with a five-day history of diffuse abdominal pain, fever for three days, and inability to defecate or pass flatus for two days. On arrival, the patient appeared lethargic and apathetic, with vital signs indicating septic shock: blood pressure of 76/46 mmHg, heart rate of 146 beats/min, respiratory rate of 28 breaths/min, and oxygen saturation of 97% on a non-rebreather mask at 10 L/min. Physical examination revealed anemic conjunctivae, non-icteric sclerae, vesicular breath sounds bilaterally without adventitious sounds, and regular heart sounds without murmurs. Abdominal examination revealed distension, decreased bowel sounds, and diffuse tenderness. The liver and spleen were not palpable because of distension. An indwelling urinary catheter was placed, yielding 100 mL of turbid yellow urine. Chest radiography revealed cardiomegaly but no other pulmonary abnormalities. The patient was diagnosed with septic shock secondary to generalized peritonitis caused by incarceration of the hernia.

Table 1. Laboratory and Diagnostic Findings on Admission (Hematology & Biochemistry)

Parameter	Result	Interpretation
Hemoglobin, g/dL	9.8	Mild anemia
White Blood Cell Count, /µL	26,000	Leukocytosis
Platelet Count, /µL	357,000	Within normal limits
Sodium (Na^+), mEq/L	132	Hyponatremia
Potassium (K^+), mEq/L	3.6	Normal
Chloride (Cl^-), mEq/L	105	Normal
Total Calcium, mg/dL	4.93	Hypocalcemia
Magnesium, mg/dL	1.9	Normal
Blood Urea Nitrogen (BUN), mg/dL	131.8	Elevated
Creatinine, mg/dL	2.51	Elevated
AST (SGOT), U/L	39	Normal
ALT (SGPT), U/L	39	Normal
Prothrombin Time (PT), seconds	15	Slightly prolonged
Activated Partial Thromboplastin Time (APTT), seconds	28.4	Normal
INR	1.06	Normal
Albumin, g/dL	2.8	Hypoalbuminemia
Lactate, mmol/L	4.7	Elevated (hyperlactatemia)

Initial management included aggressive fluid resuscitation with 1,500 mL of Ringer’s lactate over one hour and norepinephrine titration to achieve a mean arterial pressure (MAP) greater than 65 mmHg. An

emergency exploratory laparotomy was performed, which revealed 300 mL of purulent fluid in the peritoneal cavity. Intraoperative fluid management consisted of 1,000 mL of Ringer's lactate, with an estimated blood loss of 200 mL and a urine output of 150 mL over a four-hour procedure. Postoperatively, the patient was admitted to the intensive care unit (ICU) and placed on mechanical ventilation. Antibiotic therapy was initiated with intravenous meropenem (1 g every 8 h) and metronidazole (500 mg every 8 h). Fluid management included 1,500 mL of intravenous fluid per 24 h. Analgesia was provided with fentanyl 0.5 mcg/kg/hour and paracetamol 1 g every 8 hours, and the patient was kept nil per os (NPO). Sedation was managed with midazolam (3 mg/h), which was transitioned to dexmedetomidine on day four. Omeprazole (40 mg daily) was administered for gastric prophylaxis, but thromboprophylaxis was not administered during the ICU stay.

Table 2. Arterial Blood Gas (ABG) Analysis

Parameter	Result	Interpretation
pH	7.34	Slight acidosis
pO_2	148.7	Hyperoxia (likely supplemental O_2)
pCO_2	24.5	Decreased (respiratory compensation)
Base Excess	-8.5	Metabolic base deficit
HCO_3^-	14.6	Metabolic acidosis
P/F Ratio	341	No acute respiratory distress (ARDS)

The patient's fluid balance was monitored meticulously (Table 3). On the first day, the total fluid intake was 5,636 mL, comprising 5,000 mL from infusions, 500 mL from medications, and 636 mL from transfusions. The fluid output was recorded as 2,849 mL, which included 440 mL of urine, 300 mL from the nasogastric tube, 500 mL from drains, 500 mL of blood loss, and 600 mL of insensible losses, resulting in a positive fluid balance of 2,787 mL. By the sixth day, the cumulative fluid balance had shifted to a negative value of -118 mL, indicating enhanced renal function and diuresis.

Table 3. Fluid Balance During ICU Stay

Day	Total Fluid Input (mL)	Total Fluid Output (mL)	24-Hour Balance (mL)	Cumulative Balance (mL)
1	6,136	2,349	2,787	2,787
2	3,000	4,045	-545	2,212
3	2,800	3,650	-550	1,662
4	3,100	4,550	-850	812
5	3,700	5,000	-700	112
6	3,450	4,350	-300	-118

Note: Total fluid input includes infusion, drugs, transfusion, and diet. The total fluid output includes urine, nasogastric tube (NGT), drainage, bleeding, and insensible water loss (IWL).

Table 4. Laboratory Results During ICU Stay

Day	Hb (g/dL)	WBC (x10^3/µL)	Na (mEq/L)	Cr (mg/dL)	Lac (mmol/L)	Alb (g/dL)	P/F Ratio
1	7.8	13.2	132	0.96	2.7	2.8	341
2	8.2	4.78	136	0.66	4.6	1.6	383
3	9.1	3.82	144	1.06	-	1.9	322
4	8.1	8.66	144	0.56	-	2.2	400
5	8.6	11.59	-	0.56	-	2.3	408
6	9.6	20.3	135	-	-	-	-

Note: Procalcitonin levels, 10.62 ng/mL (day 2), 0.52 ng/mL (day 7)

DISCUSSION

Intra-abdominal infections (IAIs) complicated by septic shock represent a significant clinical challenge owing to their high morbidity and mortality, particularly in intensive care unit (ICU) settings. In the present case, a 59-year-old man with generalized peritonitis secondary to an incarcerated hernia developed septic shock, necessitating urgent surgical intervention and comprehensive ICU management. The patient's condition

aligned with the classification of community-acquired complicated IAI, characterized by peritonitis due to hollow viscus perforation, as described by Sartelli et al. [2].

The intraoperative finding of 300 mL of purulent peritoneal fluid confirmed secondary peritonitis resulting from the migration of gastrointestinal flora into the peritoneal cavity after anatomical barrier disruption [8].The absence of microbiological cultures in this case, despite the recommendation by the Surviving Sepsis Campaign (SSC) to obtain cultures prior to antibiotic initiation, limited the ability to tailor therapy but did not preclude effective empirical treatment with meropenem and metronidazole [3]. This regimen is appropriate given the high prevalence of Escherichia coli and Klebsiella pneumoniae in complicated IAIs in Indonesia, as reported by Moenadjat et al.[5]

Timely diagnosis and management of IAI in this patient were critical for achieving favorable outcomes. The patient presented with classic clinical signs of peritonitis, including diffuse abdominal pain, distension, and muscular guarding, corroborated by leukocytosis (26,000/μL) and elevated lactate levels (2.7 mmol/L), indicative of acute infection and tissue hypoperfusion [9,10]. Although abdominal plain radiography confirmed pneumoperitoneum, suggesting hollow viscus perforation, the decision to proceed with exploratory laparotomy within 4.5 h of presentation was pivotal, aligning with guidelines emphasizing early source control to reduce mortality [4,20]. Delays in surgical intervention for IAI are associated with increased mortality, with each hour of delay contributing to adverse outcomes [20]. Successful source control in this case, as evidenced by the evacuation of purulent fluid and management of the incarcerated hernia, underscores the importance of rapid surgical intervention in mitigating the inflammatory cascade driven by bacterial translocation and cytokine release, including tumor necrosis factor-α (TNF-α) and interleukins (IL-1, IL-6) [11].

Septic shock, diagnosed on day one based on the need for vasopressor support (norepinephrine 0.1 mcg/kg/min) and elevated lactate (4.6 mmol/L), reflects the systemic inflammatory response and endothelial dysfunction characteristics of abdominal sepsis [1,11]. Initial fluid resuscitation with 1,500 mL of Ringer's lactate in the emergency department, equivalent to approximately 23 mL/kg, was slightly below the SSC-recommended 30 mL/kg for septic shock but was sufficient to stabilize hemodynamics in conjunction with norepinephrine [3]. The lack of serial lactate measurements to assess resuscitation adequacy, as recommended by the SSC 2021, represents a deviation from optimal practice, potentially limiting the ability to confirm the resolution of tissue hypoperfusion [3,16]. Fluid management transitioned to a negative balance by day four (-500 to -700 mL/24 h), consistent with deresusitation strategies to minimize complications, such as prolonged ventilation and acute kidney injury [24]. The patient's improving renal function (creatinine decreased from 0.96 mg/dL to 0.56 mg/dL by day five) and decreasing procalcitonin levels (from 10.62 ng/mL on day two to 0.52 ng/mL on day seven) suggested effective infection control and hemodynamic stabilization.

Empirical antimicrobial therapy with meropenem and metronidazole was initiated, targeting the polymicrobial nature of IAIs, particularly gram-negative and anaerobic pathogens prevalent in secondary peritonitis [2,5]. The 10-day duration of therapy aligns with SSC recommendations for short-course therapy (7–10 days) in the presence of adequate source control [2]. However, the absence of microbiological cultures precluded de-escalation to a narrower-spectrum regimen, a practice recommended to reduce antimicrobial resistance and optimize therapy [2,3]. The patient's clinical improvement, shown by extubation on day five and transfer to high-care on day six, supports the regimen's efficacy despite the lack of culture-guided therapy. The high-risk case, due to delayed presentation and diffuse peritonitis, justifies the use of broad-spectrum antibiotics, as outlined in the extra-biliary IAI management algorithm [2].

Supportive care in the ICU followed the FASTHUG framework (Feeding, Analgesia, Sedation, Thromboembolic prophylaxis, Head-of-bed elevation, Ulcer prophylaxis, Glycemic control), with protocol deviations noted. Enteral nutrition was initiated on day three with Peptamen, progressing from 50 mL to 100 mL every four hours, delayed compared to ASPEN's recommendation of starting within 24–48 hours for high-risk critically ill patients [25]. The patient's low malnutrition risk (NRS 2002 <3) may have justified this delay, although earlier initiation could have optimized gut integrity and reduced infectious morbidity [25-37]. Analgesia and sedation with fentanyl, paracetamol, and dexmedetomidine (replacing midazolam on day four) achieved target scores on the Critical Care Pain Observation Tool (CPOT: 0–1) and Richmond Agitation-

Sedation Scale (RASS: 0 to -2), aligning with guidelines to minimize benzodiazepine use and prevent delirium [28,29]. Omitting thromboprophylaxis despite a low VTE risk based on the Padua/IMPROVE scores was reasonable given the postoperative bleeding risk, although daily VTE monitoring should have been emphasized [33–35]. Omeprazole (40 mg/day) for stress ulcer prophylaxis was appropriate given the patient's risk factors (septic shock and mechanical ventilation), although long-term proton pump inhibitor use warrants caution due to the potential risk of Clostridium difficile infection [37].

CONCLUSION

Intra-abdominal infections with septic shock are a critical challenge in the ICU and are associated with high morbidity and mortality. This case highlights the success of rapid diagnosis, timely surgical source control, early broad-spectrum antibiotics, and multidisciplinary ICU care in achieving favorable outcomes. Despite limitations, such as the absence of microbiological cultures and delayed enteral nutrition, adherence to evidence-based protocols improved survival. Enhanced diagnostics and guideline compliance are essential for optimizing care in resource-limited settings.

DECLARATIONS

None

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

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

AUTHORS' CONTRIBUTIONS

All authors made substantial contributions to the case report. GMA was responsible for patient management, data collection, and the 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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