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# Anesthetic Management of Intestinal Ischemic Necrosis Complicated by Septic Shock: A Case Report

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## Abstract

Intestinal ischemic necrosis is a life-threatening condition caused by compromised intestinal blood flow due to arterial/venous occlusion or vasoconstriction, with acute mesenteric ischemia (60–70% of cases, mortality >60%) and colonic ischemia as primary subtypes. This case report describes the anesthetic management of a 56-year-old male with acute intestinal ischemic necrosis complicated by septic shock, requiring emergent laparotomy. Preoperative assessment revealed hemodynamic instability (HR 130 bpm, BP 85/43 mmHg, $SpO_2$ 89%) and peritoneal signs.

Anesthesia involved rapid-sequence induction with etomidate/rocuronium and maintenance with sevoflurane–remifentanil under BIS guidance. Hemodynamic stabilization included:

1. Fluid resuscitation (1500 mL crystalloid, 1000 mL colloid, 500 mL fresh frozen plasma) guided by pulse pressure variation (PPV) / transthoracic echocardiography (TTE)
2. Vasopressors (norepinephrine 0.1–0.3 μg/kg/min + vasopressin 0.01–0.02 U/min)
3. Metabolic correction (sodium bicarbonate for pH 7.246, calcium gluconate for $Ca^{2+}$ 0.87 mmol/L)
4. Epinephrine infusion (0.02–0.1 μg/kg/min) improved left ventricular ejection fraction (35% → 45–50%) and right ventricular function

The patient underwent successful small bowel resection (300 cm) with primary anastomosis (operative time: 108 minutes, EBL: 100 mL), was extubated on postoperative day (POD) 3, and discharged on POD 22.

Key anesthetic principles emphasized:

1. Early hemodynamic optimization (MAP ≥65–70 mmHg, urine output >0.5 mL/kg/h)
2. Multimodal monitoring (PPV, TTE, etc.)
3. Balanced fluid/vasopressor therapy
4. Metabolic and temperature control

This case highlights the critical role of tailored anesthetic strategies in managing this high-mortality condition.

## Keywords

Intestinal Ischemic Necrosis, Septic Shock, Anesthesia, Hemodynamic Stabilization, Emergency Laparotomy

Case Report

## Abbreviations

PPV: Pulse Pressure Variation; BIS: Bispectral Index; TTE: Transthoracic Echocardiography; ABG: Arterial Blood Gas Analysis; POD: Postoperative Day; ABP: Arterial Blood Pressure; CVP: Central Venous Pressure; SVR: Systemic Vascular Resistance.

## Introduction

Intestinal ischemic necrosis, a life-threatening condition affecting either the small or large bowel, occurs when compromised blood flow results from arterial or venous occlusion, or profound vasoconstriction. When involving the small intestine, this condition is clinically designated as acute mesenteric ischemia, accounting for 60–70% of cases, with mortality rates exceeding 60%. Large intestinal involvement is typically classified as colonic ischemia.

Without prompt intervention, severe hypoperfusion can rapidly progress to transmural necrosis, potentially leading to catastrophic complications including sepsis, peritonitis, pneumoperitoneum, or gangrene. Immediate surgical intervention becomes imperative when clinical presentation, imaging studies, or laboratory findings suggest bowel infarction. This case report details the critical anesthetic considerations and management strategies for a patient presenting with intestinal ischemic necrosis complicated by septic shock.

## Case Presentation

*Preoperative Assessment*:

A 56-year-old Asian male (BMI 23 kg/$m^2$) presented with a five-month history of progressive abdominal pain exhibiting acute exacerbation over the preceding 48 hours. The patient was subsequently diagnosed with acute intestinal ischemic necrosis complicated by septic shock, necessitating emergent exploratory laparotomy. Relevant comorbidities included poorly controlled type 2 diabetes mellitus and essential hypertension. Preoperative assessment revealed limited functional capacity (3–5 metabolic equivalents). On admission, the patient demonstrated the following hemodynamic parameters:

- Tachycardia (HR 130 bpm)
- Hypotension (BP 85/43 mmHg)
- Tachypnea (RR 35 breaths/min)
- Marginal oxygenation ($SpO_2$ 89% on 6 L/min $O_2$ via simple face mask)
- Normothermia (36.7°C)

Physical examination was notable for significant abdominal distension with diffuse peritoneal signs, including guarding and rebound tenderness.

*Anesthetic Management*:

Rapid Sequence Induction and Maintenance:

Following 5 minutes of head-up preoxygenation (end-tidal $O_2$ >90%), anesthesia was induced with intravenous etomidate (6 mg) and rocuronium (50 mg) to facilitate rapid sequence intubation. Standard cricoid pressure was maintained during video laryngoscopy-assisted endotracheal intubation. Proper tube placement was verified by waveform capnography and bilateral chest auscultation. Anesthesia maintenance consisted of sevoflurane (1.5–2% end-tidal concentration) and remifentanil infusion (0.08–0.15 μg/kg/min), titrated according to bispectral index (BIS) monitoring (target range 40–60).

Hemodynamic Stabilization Protocol:

Post-intubation, the patient was immediately positioned in a modified shock position (Trendelenburg with elevated lower extremities). Hemodynamic monitoring included large-bore (14-gauge) peripheral IV access, invasive arterial pressure monitoring via left radial artery cannulation, and central venous catheterization (right internal jugular approach).

Based on invasive arterial blood pressure monitoring, pulse pressure variation (PPV), arterial blood gas analysis (ABG), and transthoracic echocardiography findings, the patient received 1500 mL of normal saline (0.9% NaCl), 1000 mL of succinylated gelatin solution (colloid), and 500 mL fresh frozen plasma (FFP). Hemodynamic support was achieved with norepinephrine (0.1–0.3 μg/kg/min) and vasopressin (0.01–0.02 U/min) to optimize systemic vascular resistance and mean arterial pressure.

Physiological Optimization:

For metabolic management, sodium bicarbonate (150 mL) was administered to correct refractory metabolic acidosis (pH = 7.246, BE = −10.5), calcium gluconate (2 g) to address ionized hypocalcemia ($Ca^{2+}$ = 0.87 mmol/L), and an insulin drip was titrated to maintain glycemic control within the target range of 140–180 mg/dL. Initial arterial blood gas analysis demonstrated moderate ARDS ($PaO_2/FiO_2$ ratio <180) with elevated lactate (9.5 mmol/L), while transthoracic echocardiography revealed reduced left ventricular function (LVEF 35%), moderate tricuspid regurgitation, and elevated right ventricular systolic pressure (45 mmHg). Following initiation of epinephrine infusion (0.02–0.1 μg/kg/min), subsequent echocardiographic evaluation showed improved left ventricular ejection fraction (45–50%), reduced right ventricular pressures, and normalized oxygenation ($SpO_2$ 100% on $FiO_2$ 0.6). Active temperature management was maintained through forced-air warming blankets (core temperature 36.5–37°C) and inline fluid warmers for all intravenous infusions, with renal perfusion monitored via hourly urine output (>0.5 mL/kg/h) and serial creatinine measurements.

Surgical Intervention:

The surgical team performed an emergency small bowel resection of necrotic segments (approximately 45 cm) with primary side-to-side functional anastomosis. Total operative duration was 108 minutes with minimal blood loss (approximately 100 mL). The patient was transferred to the intensive care unit (ICU) for postoperative management after surgery. The endotracheal tube was extubated on postoperative day (POD) 3, followed by transfer to the general ward on POD 5. The patient was discharged in stable condition on POD 22 after successful recovery.

## Discussion

Sepsis is the most common cause of distributive shock in surgical patients. Initial therapy includes fluid resuscitation for hypovolemia (which may be severe) and vasopressors (typically norepinephrine) to achieve a MAP ≥65–70 mmHg. Additional vasopressors, inotropes, or blood transfusion may be required. The focus is on ensuring adequate organ perfusion rather than targeting potentially harmful higher MAP. Chronic hypertension patients may require higher MAP. Urine output should ideally be restored to ≥0.5 mL/(kg·h) [1].

*Fluid Resuscitation*:

Similar to hypovolemic shock, rapid IV boluses of balanced crystalloids (e.g., lactated Ringer's solution) in 500 mL increments are administered as needed. Achieving MAP ≥65–70 mmHg may require 2–5 L of fluid [2]. If large crystalloid volumes are needed intraoperatively, albumin may be considered [3].

*Vasopressors*:

If fluids fail to restore MAP, vasopressors (commonly norepinephrine) are initiated [1,4]. If significant tachycardia or arrhythmias occur with norepinephrine, phenylephrine may be substituted. If refractory hypotension persists, vasopressin is added, as acquired vasopressin deficiency may develop in prolonged septic shock [5]. Inotropic support may be needed for refractory shock with low cardiac output. Methylene blue can be considered for vasoplegic shock [6].

*Antibiotics*:

Broad-spectrum IV antibiotics should be administered as soon as possible, ideally within 3 hours of presentation, to reduce mortality. Blood cultures should be obtained before antibiotic administration to guide targeted therapy [7].

*Hyperglycemia*:

Septic shock patients often exhibit hyperglycemia and insulin resistance. IV insulin is typically required to maintain blood glucose between 140–180 mg/dL (7.7–10 mmol/L) [8].

*Relative Adrenal Insufficiency*:

IV corticosteroids are indicated for severe septic shock unresponsive to fluids and vasopressors [9].

## Conclusion

The anesthetic management of intestinal ischemic

necrosis complicated by septic shock requires an individualized, physiology-based strategy focused on three critical objectives: (1) maintenance of hemodynamic stability through precision-guided interventions, (2) optimization of organ perfusion using advanced monitoring techniques, and (3) protection of end-organ function via targeted therapeutic approaches.

## Consent for Publication

Written informed consent was obtained from the patient's son for the publication of this case report and related images.

## Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of West China Hospital of Sichuan University. Written informed consent was obtained from the patient and his wife for the publication of any potentially identifiable images or data included in this article.

## Data Availability Statement

The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding authors.

## Conflict of Interest

The author has read and approved the final version of the manuscript. The author declares no conflicts of interest.

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