Case Report

Intra-abdominal Hernia with Bowel Obstruction Triggering Multi-organ Failure: The Role of Preoperative Enteritis

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Abstract

This report details the clinical course of a 41-year-old male who developed fulminant multiple organ dysfunction syndrome following emergency surgery for a strangulated intra-abdominal hernia. Preoperative manifestations, including diarrhea and marked leukocytosis, pointed towards an underlying infectious enteritis. We posited that surgical release of the obstructed, ischemic bowel segment precipitated a massive systemic influx of endotoxins and inflammatory mediators, triggering septic shock and rapid sequential organ failure. This case underscored the critical need to suspect concomitant gastrointestinal infection in patients presenting with mechanical bowel obstruction accompanied by infectious signs. Aggressive perioperative sepsis management encompassing early empiric antimicrobial therapy, vigilant hemodynamic monitoring, and preparedness for advanced organ support is essential to mitigate this severe complication.

Keywords: Bowel obstruction; enteritis; internal hernia; ischemia-reperfusion injury; multiple organ dysfunction syndrome; septic shock

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Introduction

Internal hernias represent an uncommon but potentially lethal cause of acute bowel obstruction, with a significant risk of intestinal strangulation and ischemia. Their nonspecific clinical presentation often contributes to diagnostic delays. Multiple organ dysfunction syndrome (MODS) (1), a life-threatening condition characterised by the progressive failure of two or more organ systems, carries a high mortality rate. It is most frequently precipitated by severe sepsis, major trauma or extensive surgical procedures. We present an instructive case of catastrophic MODS that ensued after surgical repair of a strangulated intra-abdominal hernia. The distinctive feature of this case was the presence of preoperative signs indicative of active enteritis, a factor that likely played a pivotal role in the pathophysiology of the subsequent systemic collapse. This report highlights a critical clinical scenario where the interplay between localised gastrointestinal infection and mechanical obstruction, followed by surgical intervention, created a perfect storm for fulminant systemic inflammatory response and multi-organ failure.

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Case report

A previously healthy 41-year-old male presented with a 7-hour history of acute-onset, progressively worsening abdominal pain associated with nausea, vomiting and diarrhoea. He was afebrile. Initial examination revealed normal vital signs, abdominal distension, tenderness in the left quadrant and hyperactive bowel sounds with tinkling qualities. Laboratory investigation showed leucocytosis (white blood cell count 14.8 × 10^9/L) with neutrophilia (76.3%). An abdominal computed tomography (CT) scan suggested intestinal obstruction as evidenced by air-fluid levels.

Conservative management with nasogastric decompression, intravenous fluids and antibiotics was initiated. A repeat CT scan on hospital day 2 demonstrated dilated small bowel loops with focalisation, raising strong suspicion for an internal hernia. The patient initially declined surgical intervention but consented on day 3 due to unbearable pain.

Operative findings

Emergency laparotomy revealed a segment of the jejunum (approximately 100 cm in length) herniated and incarcerated through a congenital defect in the small bowel mesentery, the margin of which was formed by the inferior mesenteric vein. The affected bowel was markedly oedematous with areas of focal ischemia but no transmural necrosis. The hernia was manually reduced. Given the vascular nature of the hernial ring, it was not divided; the mesenteric defect was primarily closed. The total operative time was 105 minutes.

Postoperative course

The initial postoperative period was uneventful. However, on the night of surgery, the patient ingested a large volume of water independently. Early on postoperative day 1 (POD1), he developed severe abdominal distension, agitation and profound watery diarrhoea exceeding 20 episodes per day. His condition deteriorated rapidly, characterised by severe metabolic acidosis (pH 7.16), tachycardia, tachypnea and hypoxemia (SpO_2 74%), necessitating urgent transfer to the intensive care unit (ICU).

His clinical decline accelerated, culminating in respiratory failure requiring endotracheal intubation and mechanical ventilation. Laboratory parameters confirmed the onset of MODS: worsening metabolic acidosis, elevated serum lactate (peaking at 8.36 mmol/L), acute kidney injury (creatinine 374 μmol/L), acute hepatic dysfunction (AST 253 U/L) and severe thrombocytopenia (platelets falling to 20 × 10^9/L). Stool microscopy confirmed significant fecal leucocytosis (30-50 white blood cells per high-power field). Management included broad-spectrum antibiotics (imipenem-cilastatin and levornidazole), aggressive fluid resuscitation, sodium bicarbonate infusion and the initiation of continuous renal replacement therapy (CRRT) on POD1 for refractory acidosis and renal failure. Despite maximal supportive care, multi-organ failure persisted, prompting transfer to a tertiary care centre on POD4 at the family's request. Key temporal trends in laboratory parameters were summarised in Tables 1-4.

TABLE 1: Patient hematology results

Time	WBC (10^9/L)	Neutrophils (%)	Platelets (10^9/L)	Hematocrit (%)
Preoperative	14.8	76.3	243	47.5
POD1	5.8	93.2	192	55.9
POD2	9.7	95.0	88	45.4
POD3	17.0	94.0	35	33.5

TABLE 2: Selected biochemical markers of organ dysfunction

Time	ALT (U/L)	AST (U/L)	Creatinine (μmol/L)	Lactate (mmol/L)	NT-proBNP (ng/L)
Preoperative	49	36	84	-	-
POD1	66	150	374	5.39	>35,100.0
POD2	105	253	214	8.36	4397.9

TABLE 3: Stool microscopy findings

Time	Red Blood Cells	White Blood Cells	Pus Cells
POD2	None	2-5 / HPF	Few
POD4	0-1 / HPF	30-50 / HPF	Few

TABLE 4: Serial arterial blood gas analysis (Selected time points)

Time (POD)	pH	pCO_2 (mmHg)	pO_2 (mmHG)	HCO_3^- (mmol/L)	Base Excess (mmol/L)
1 (13:00)	7.16	23.6	89.7	8.4	-18.6
1 (17:00)	7.13	37.3	77.0	11.9	-16.5
2 (07:00)	7.26	51.7	57.0	22.6	-4.4
3 (07:00)	7.28	23.8	72.0	10.8	-14.0

Discussion

This case exemplifies a rapid and severe progression to established MODS (1) following surgical correction of a strangulated internal hernia. The diagnosis was substantiated by the sequential failure of five organ systems within 48 hours: cardiovascular (evidenced by markedly elevated NT-proBNP) (2), respiratory (ARDS requiring mechanical ventilation) (3), hepatic (significant transaminitis), renal (acute kidney injury necessitating CRRT) (4) and hematologic (profound thrombocytopenia).

Pivotal role of concurrent enteritis

The distinctive and instructive element of this presentation is the compelling evidence for a severe, pre-existing infectious enteritis that served as the primary catalyst for the subsequent systemic collapse (5). Preoperative indicators, namely diarrhoea and leucocytosis with neutrophilia, are atypical for simple mechanical obstruction and should raise suspicion of an underlying inflammatory or infectious intestinal process. Postoperatively, the hallmark profuse secretory diarrhoea and confirmed faecal leucocytosis provided definitive microbiological evidence of an invasive gastrointestinal infection. We hypothesise that this infectious milieu contributed to a critical pathophysiological sequence: the inflammatory state and bowel wall oedema may have facilitated herniation through a pre-existing mesenteric defect. More significantly, the strangulated bowel segment became a reservoir for a high bacterial and endotoxin load.

Postulated pathophysiological cascade

The clinical timeline and findings support the following sequence of events: (i) Pre-existing enteritis: An active gastrointestinal infection, likely bacterial, caused mucosal inflammation and altered motility. The reported "diarrhoea" at presentation may have represented "paradoxical diarrhoea" secondary to partial obstruction and irritation of the inflamed bowel; (ii) Obstruction and systemic insult (6): The internal hernia led to complete obstruction, causing fluid sequestration ("third-spacing"), electrolyte disturbances and intravascular volume depletion, contributing to pre-renal azotemia and metabolic acidosis; (iii) Reperfusion injury as the critical trigger: Surgical release of the obstruction, while anatomically corrective, precipitated reperfusion of the ischemic, bacteria-laden bowel segment. This sudden restoration of blood flow facilitated a massive systemic influx of endotoxins (e.g., lipopolysaccharide), microbial products and locally generated inflammatory mediators (e.g., cytokines, oxygen-free radicals); and (iv) Fulminant septic shock and MODS: This abrupt endotoxin and cytokine storm triggered fulminant septic shock, which served as the direct driver for the rapid onset of MODS observed within hours postoperatively. The magnitude of the response overwhelmed endogenous compensatory mechanisms, leading to distributive shock, capillary leak, tissue hypoperfusion and end-organ damage. This mechanism is consistent with the well-documented but perilous consequence of reperfusion following the release of any strangulated bowel, highlighting how a concomitant enteritis drastically amplifies the bacterial and inflammatory burden, thereby exacerbating the systemic response. Other potential sources of sepsis, such as pneumonia or surgical site infection, were reasonably excluded by clinical and radiographic evaluation.

Conclusion

This report illustrates a critical and potentially under-appreciated clinical trajectory leading to MODS: the confluence of severe infectious enteritis and mechanical bowel obstruction, wherein surgical intervention paradoxically initiates the final common pathway of systemic collapse via reperfusion injury.

Key Clinical Implications

(i) Elevated clinical suspicion: In patients presenting with clinical and radiographic evidence of bowel obstruction, the concomitant presence of diarrhoea, leucocytosis or marked neutrophilia should prompt a

high index of suspicion for an underlying gastrointestinal infection, even in the absence of overt peritonitic signs.

(ii) Mandatory aggressive perioperative management: When such a scenario is recognised or suspected, perioperative strategy must prioritise sepsis management. This includes early, empiric antimicrobial therapy and immediate administration of broad-spectrum antibiotics effective against common enteric pathogens.

(iii) Vigilant hemodynamic monitoring and proactive resuscitation: Anticipatory fluid management and vasopressor support to counteract impending distributive shock.

(iv) Preparedness for rapid clinical deterioration: Awareness that surgical relief of the obstruction may precipitate clinical decline. Immediate access to advanced organ support (e.g., mechanical ventilation, CRRT) is crucial.

(v) Pathophysiological awareness: Clinicians must recognise the dangerous synergy wherein localised bowel infection and obstruction create a scenario where the necessary surgical correction can act as the trigger for a systemic inflammatory avalanche via reperfusion-mediated endotoxin release. Heightened awareness of this sequence, coupled with prompt diagnostic suspicion and aggressive, integrated perioperative care, may improve outcomes and mitigate the risk of this devastating complication.

Data availability statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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