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Clinical-radiological and intraoperative discrepancy in massive pneumatosis intestinalis and portomesenteric venous gas in a patient with septic shock

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Abstract

Pneumatosis Intestinalis (PI) and Portomesenteric Venous Gas (PMVG) are radiological findings frequently associated with transmural bowel ischemia and poor prognosis. Their presence usually prompts urgent surgical intervention. A 60-year-old man with a past history of small bowel infarction presented with septic shock, abdominal pain, and elevated serum lactate (3.2 mmol/L). Contrast-enhanced CT demonstrated massive intra- and extrahepatic PMVG with a bubble-like intramural gas pattern in the small bowel. Based on the clinical and imaging findings, emergency laparotomy was performed. Intraoperative exploration revealed bowel distension and mucosal edema with petechial hemorrhage, but no evidence of transmural necrosis. A second-look procedure at 48 hours confirmed the absence of necrosis. This case highlights that even massive PI with PMVG and septic shock may not invariably indicate bowel necrosis. Careful correlation of radiological, laboratory, and clinical findings is essential, and surgical exploration remains mandatory in unstable patients to prevent catastrophic outcomes.

Introduction

Pneumatosis Intestinalis (PI) is defined as the presence of gas within the intestinal wall, most commonly located in the mucosa or submucosa, and may involve any segment of the

gastrointestinal tract.[1,2] PI was first described by Duvernoy in 1730 during a cadaveric dissection,[3] while Portomesenteric Venous Gas (PMVG) was initially reported by Wolf and Evans in 1955.[4] With the increasing availability of Multidetector Computed Tomography (MDCT), both PI and PMVG are now detected more frequently in the emergency setting.

Traditionally, PI was considered an alarming radiological sign, almost always interpreted as a marker of transmural bowel ischemia requiring urgent surgical intervention.[5] The clinical scenario is even more concerning when PI is associated with PMVG, which has been linked to a significantly higher risk of bowel necrosis and poor outcome.[1,2,6]

However, PI should not be regarded as a disease in itself but rather as a radiological manifestation of different underlying conditions.[7] Its pathogenesis is multifactorial, with three main proposed mechanisms: the bacterial theory, in which intramural gas originates from bacterial overgrowth and translocation;[8] the mechanical theory, where increased intraluminal pressure or iatrogenic trauma forces gas into the bowel wall;[8] and the mucosal disruption theory, where epithelial injury facilitates gas penetration, commonly seen in ischemia, inflammation, or after chemotherapy and steroid therapy.[9]

Additionally, PI may occur in patients with chronic obstructive pulmonary disease or in those receiving positive-pressure ventilation. In these cases, the so-called “pulmonary theory” suggests that alveolar rupture with subsequent tracking of air along mediastinal and retroperitoneal planes may lead to gas within the bowel wall.[10]

This complex and heterogeneous pathophysiology explains why PI can be associated with a wide range of clinical scenarios, from incidental findings to life-threatening ischemia. Consequently, predicting the severity of PI and determining the need for surgical intervention remain major clinical challenges.[11] In this report, we present the case of a patient with septic shock and CT evidence of PI and PMVG, in whom exploratory laparotomy revealed no evidence of transmural necrosis.

Case Report

A 60-year-old man with a past medical history of small bowel infarction treated one year earlier with ileal resection and ileostomy was admitted to the emergency department with altered mental status and worsening abdominal pain over the previous three hours.

On arrival, he was hypotensive (blood pressure 70/38 mmHg) and required fluid resuscitation and vasopressor support. Laboratory tests revealed ph value < 7.3, bicarbonate level < 20 ml/L and elevated serum lactate (3.2 mmol/L). Physical examination showed a distended abdomen with diffuse tenderness. Given the suspicion of recurrent intestinal ischemia with sepsis, an urgent contrast-enhanced CT was performed.

CT demonstrated extensive intra- and extrahepatic Portomesenteric Venous Gas (PMVG) associated with intramural gas in small bowel loops adjacent to the ileostomy, showing a bubble-like pattern. The mesenteric arteries and veins were patent, and no free fluid or free air was detected in the peritoneal cavity (Figure 1 and 2a-b).

Based on the patient's status of shock/sepsis, lab results, and CT findings, emergency exploratory laparotomy was performed. Intraoperatively, the small bowel was distended for approximately 40–50 cm proximal to the ileostomy, with mucosal edema and petechial hemorrhage, but no macroscopic evidence of transmural necrosis or pneumatosis was observed (Figure 3 and 2c-d). No resection was required. A planned second-look laparotomy performed 48 hours later confirmed the absence of bowel necrosis.

The patient's postoperative course was uneventful. Lactate levels progressively normalized within 72 hours, with gradual correction of the acid–base imbalance. Arterial blood gas analysis showed that pH and bicarbonate levels, initially decreased (pH 7.25–7.30; bicarbonate <20 mmol/L), progressively returned to normal values within 48–72 hours, in parallel with clinical improvement and normalization of lactate levels.; D-dimer levels were within the normal range. There were no signs of sepsis or intestinal dysfunction. He resumed oral feeding on postoperative day 6 and was discharged home on postoperative day 14 without complications.

Discussion

PI and PMVG are uncommon but alarming radiological findings, traditionally considered ominous signs strongly associated with bowel ischemia and infarction.[1,2,6] Historically, their detection was almost invariably interpreted as transmural necrosis and an immediate indication for surgery.[5] However, PI is not a disease entity in itself but a radiological sign with multifactorial pathogenesis,[8] which may result from bacterial gas production, increased intraluminal pressure, or mucosal barrier

disruption.[9] In some cases, PI and PMVG may even occur as incidental findings or in non-ischemic conditions.[10]

The prognostic interpretation of PI and PMVG must therefore be contextualized within the overall clinical picture. Several studies have reported a strong association between the concomitant presence of PI and PMVG and bowel infarction, with mortality rates reaching 80–90%.[12] PMVG alone is associated with transmural infarction in up to 81% of cases, and when combined with PI, the probability of necrosis exceeds 90%.[12] Some authors have proposed that certain imaging features, such as a bubble-like distribution pattern, are more frequently associated with benign outcomes, whereas linear or circumferential gas patterns correlate more often with irreversible ischemia.[12] However, these distinctions are not universally reliable.[13]

Beyond imaging, biochemical markers are of paramount importance. Elevated serum lactate (> 2 mmol/L) has been consistently associated with irreversible ischemia.[14] In our patient, lactate reached 3.2 mmol/L, a value typically highly predictive of transmural necrosis.[15]

Other indicators/poor predictors of prognosis include acide/base disturbances susch as pH value < 7.3 and bicarbonate level < 20 ml/L.
Among these studies, Bani Hani *et al.*[13] demonstrate that high lactate and low arterial CO2 are correlated with a worst outcome in patients with PI. Also, Horowitz *et al*.[45] tried to understand which laboratory test can predict the outcome of these patients. They found out that low bicarbonate levels (< 20 mmol/L) and low pH (< 7.35) correlate with poor outcome. Although almost each laboratory test has been investigated in different studies, for some studies peritonitis and clinical exam remain the strongest predictors of outcome.[16,17]

According to the most recent guidelines from the World Society of Emergency Surgery (WSES) and the Eastern Association for the Surgery of Trauma (EAST), management of suspected mesenteric ischemia should always rely on the integration of clinical, biochemical, and radiological parameters.[18,19] Hemodynamic instability or signs of peritonitis mandate immediate laparotomy without delaying for imaging, whereas in stable patients, CT angiography remains the diagnostic modality of choice.[19] When bowel viability remains uncertain, a planned second-look laparotomy within 24–48 hours is recommended.[19]

More recent evidence-based decision algorithms have further emphasized a tailored approach that balances the catastrophic risk of missed transmural ischemia with the morbidity of unnecessary

laparotomies, suggesting that surgical intervention should not be based solely on radiological findings but on a comprehensive clinical and biochemical evaluation.[20]

In the present case, several features were highly suggestive of transmural infarction: septic shock, elevated lactate, metabolic acidosis and CT findings of massive PI with PMVG. Despite this, laparotomy revealed only mucosal edema and petechial hemorrhage without necrosis, a finding confirmed during the second-look procedure. The postoperative course was uneventful, with progressive normalization of lactate levels, resolution of abdominal pain, and discharge on postoperative day 14 without complications.

Only a limited number of reports have described patients with PI and PMVG showing a mismatch between alarming radiological findings and relatively benign intraoperative or clinical outcomes. In these rare observations, massive PI and PMVG did not necessarily correspond to transmural necrosis, suggesting that imaging features alone may overestimate disease severity.[21,22]

In a few instances, particularly in septic or hemodynamically unstable patients, conservative or delayed management was associated with favorable evolution.[23,24]

Recent cohort analyses have reinforced the notion that the prognostic weight of PI and PMVG depends more on clinical and biochemical markers, such as shock, metabolic acidosis, and elevated lactate, than on the mere presence or extent of gas on CT.[25]

Within this context, our case further emphasizes the potential clinical–radiological discrepancy in massive PI with PMVG, underlining the need for an integrated assessment to avoid unnecessary or premature surgical intervention.

Considering the high complexity of this topic, analyzing data available in literature, we were able to identify some anamnestic, laboratory and radiological risk factors predictors of life-threatening PI versus benign, synthesized in Table 2.

Clinical decision-making for discordant PI findings involves a careful balance between avoiding unnecessary surgery and preventing a catastrophic outcome from missed ischemia and should prioritize patient stability, using a multi-factorial approach that includes clinical symptoms, lab results, and CT findings, rather than relying solely on imaging. In case of PI at the CT scan, distinction between hemodynamically stable or unstable patients is crucial. In case of instability, surgical exploration is mandatory. In case of stability, clinical presentation plays a central role, considering as symptomatic the presence of abdominal tenderness or peritonism. If the patient is

symptomatic, operative treatment is advocated. Otherwise, we rely on some anamnestic, laboratory and radiological parameters considered as risk factors (Table 2).
A “watch and wait” approach is often best for stable patients without signs of peritonitis or sepsis, while surgical intervention is reserved for those with clinical signs of ischemia like hemodynamic instability, peritonitis, or acidosis. A thorough evaluation of clinical symptoms, physical exam findings, and laboratory tests is paramount, as the radiographic finding of PI does not always correlate with the severity of the underlying pathology. The selection of an appropriate treatment course hinges upon a thorough assessment of the patient’s overall health, clinical manifestations, medical background, and imaging results to ascertain whether the presence of PI poses a benign or life-threatening scenario.

Our working hypothesis is that prolonged hypotension induced transient mesenteric vasospasm and mucosal ischemia, which allowed intramural gas migration and PMVG dissemination. Reperfusion likely occurred before irreversible necrosis developed, leading to the observed discrepancy between radiological severity and intraoperative findings. This mechanism has been hypothesized in previous reports describing similar cases of extensive PI and PMVG without transmural necrosis, suggesting a transient ischemia–reperfusion pathophysiology.[26,27] In these previously reported cases, lactate levels remained stable or decreased over time, emphasizing the importance of integrating biochemical trends and clinical presentation, rather than solely imaging findings.

Furthermore, a recent systematic review concluded that the presence of PI and PMVG alone should not be considered an absolute indication for surgery, and that conservative or delayed approaches may be appropriate in selected patients, particularly when clinical and biochemical parameters are stable.[28]

This case emphasizes that even extensive PI and PMVG in the setting of shock and elevated lactate are not invariably indicative of bowel infarction. Nevertheless, surgical exploration remains mandatory in such scenarios, given the catastrophic risk of missed transmural ischemia. The challenge for clinicians lies in integrating clinical, biochemical, and imaging data to avoid both unnecessary laparotomies and potentially fatal diagnostic delays.

Limitations

This report describes a single case, and thus its generalizability is inherently limited. Nevertheless, it underscores that although PI and PMVG are typically considered strong predictors of bowel necrosis, exceptions exist. Careful integration of radiological, clinical, and laboratory data remains essential for appropriate decision-making.

Conclusions

This case highlights that even massive pneumatosis intestinalis with extensive PMVG, in the context of septic shock and elevated lactate, may not invariably indicate transmural bowel necrosis. Surgical exploration remains mandatory in unstable patients to avoid missing life-threatening ischemia, but radiological and biochemical predictors should always be interpreted in combination with the clinical scenario. A planned second-look laparotomy is crucial whenever bowel viability remains uncertain.

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Table 1. Pathological conditions associated with pneumatosis intestinalis.

Category	**Conditions**
Trauma	Blunt/penetrating abdominal trauma
Mechanical	Bowel obstruction (volvulus, carcinoma, malrotation, intussusception)
Autoimmune	Lupus enteritis; Celiac sprue; Polymyositis; Dermatomyositis; Polyarteritis nodosa; Mixed connective tissue diseases; Graft-versus-host disease; Primary immunodeficiency
Inflammatory	Inflammatory bowel disease; Appendicitis; Diverticulitis; Cholelithiasis; Sarcoidosis
Vascular conditions	Ischemia or infarction; Diabetes
Pulmonary diseases	Chronic obstructive pulmonary disease; Cystic fibrosis; Asthma

Category	Conditions
Drugs	Corticosteroids; Chemotherapy and immunotherapy; Immunosuppression; Lactulose; Trichloroethylene; Sorbitol; Alpha-glucosidase inhibitors; Practolol
Diagnostic/therapeutic procedures	Endoscopy; Enema/colon hydrotherapy; Barium studies
Connective tissue/Neurological	Scleroderma; Multiple sclerosis; Hirschsprung disease; Quadriplegia; Amyloidosis
Other conditions	Hemodialysis; Pseudo-obstruction; Whipple disease; Cytomegalovirus infection

Table 2. Summary of demographic, clinical, laboratory, and radiological factors associated with transmural bowel ischemia in patients presenting with Pneumatosis Intestinalis (PI) and/or Portomesenteric Venous Gas (PMVG).

Category	Predictive Factors	Notes / Implications
Demographic & Clinical	Age > 60 years	Older patients more frequently have ischemic or life-threatening PI.
	Hemodynamic instability, sepsis, or peritonitis	Strongly associated with transmural ischemia and poor outcome.
Laboratory	Lactate > 2 mmol/L	Most reliable biochemical predictor of bowel ischemia.
	Metabolic acidosis	Reflects tissue hypoperfusion and anaerobic metabolism.
	Elevated WBC count	Non-specific but correlates with inflammatory or ischemic damage.
Radiological	Coexistence of PI and PMVG (vs PI or PMVG alone)	Combined findings increase risk of transmural necrosis.

Category	Predictive Factors	Notes / Implications
	Small bowel involvement (vs large bowel)	Strongly predictive of ischemic etiology and need for surgery.

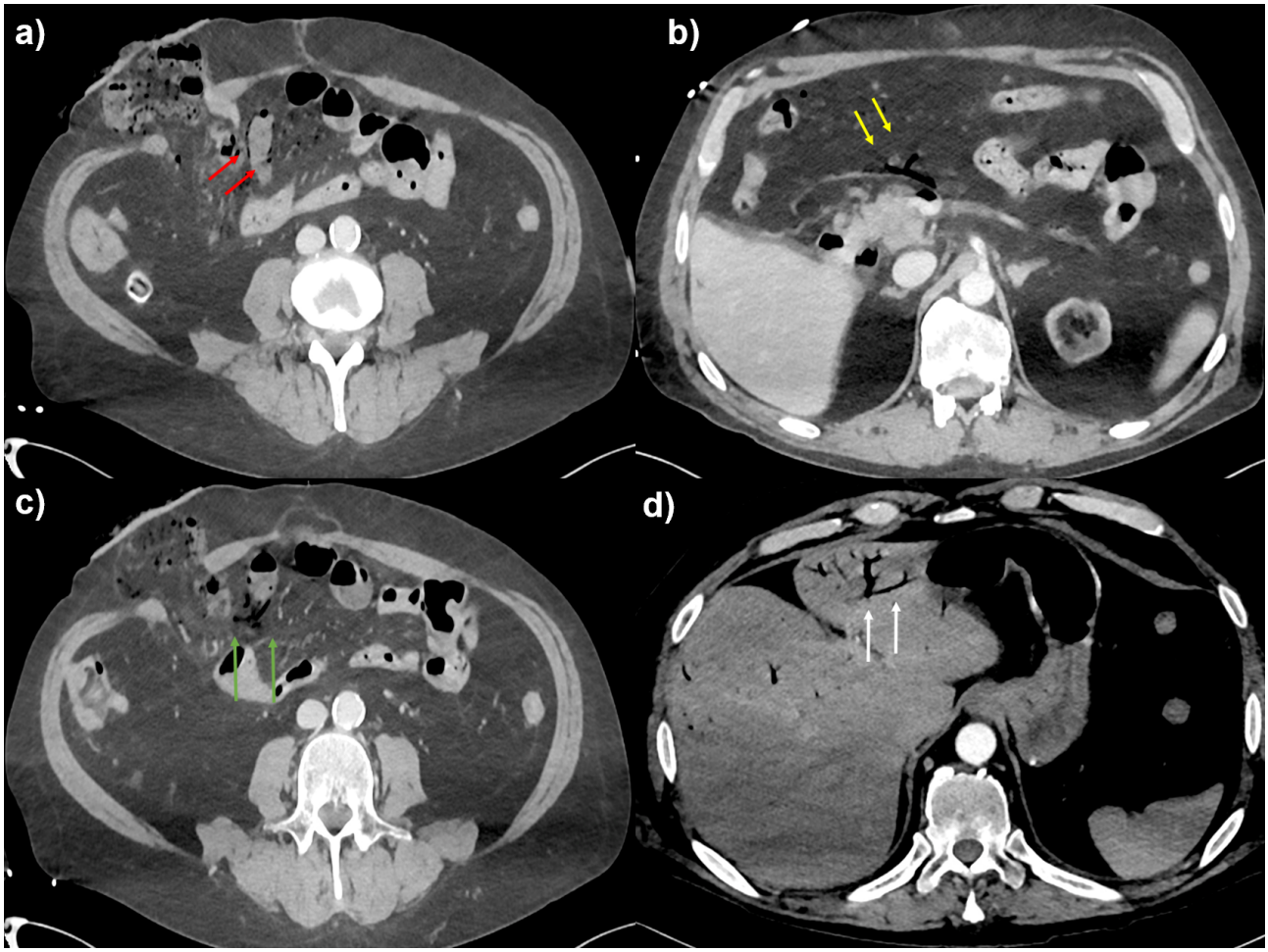

Figure 1. A 60-year-old male patient with septic shock and past history of bowel infarction. Axial computed tomography (CT) scan shows pneumatosis intestinalis (a, red arrows) and large gas collection in the superior mesenteric vein (b, yellow arrows), its tributaries (b, green arrows) and peripherally in the liver (d, white arrows).

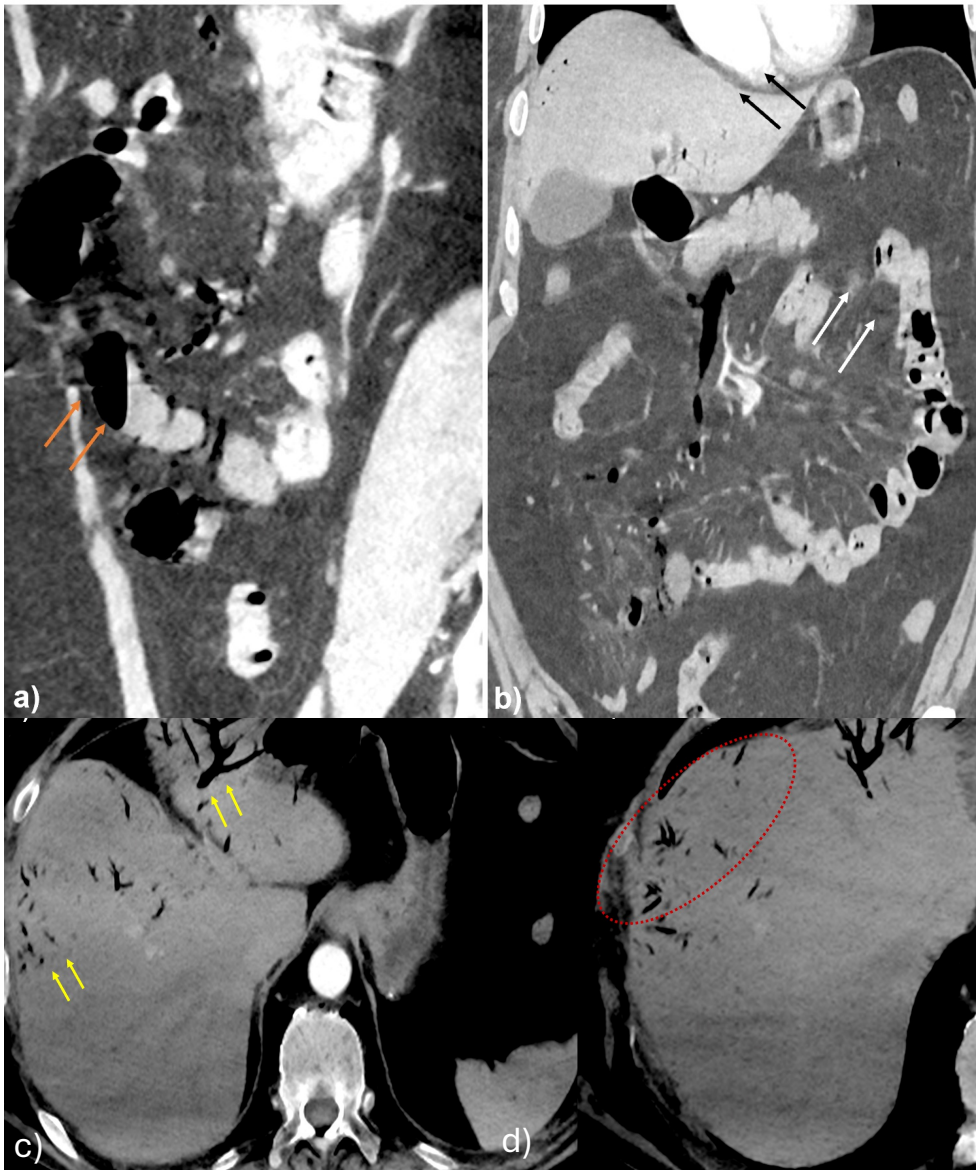

Figure 2. Magnified view sagittal and coronal reformatted CT scan reveal PI (a, orange arrows) with evidence of "bubblelike" pattern involving bowel loops adjacent to the ileostomy and the presence of portomesenteric venous air and portal air in the liver (b, black and white arrows).

Extensive intra-hepatic portal venous gas within both lobes of the liver especially the right and the medial aspects of the left lobe (c, yellow arrows). Typically, the gas in the liver is peripheral (d, dashed oval) which helps differentiate it from more central gas due to pneumobilia.

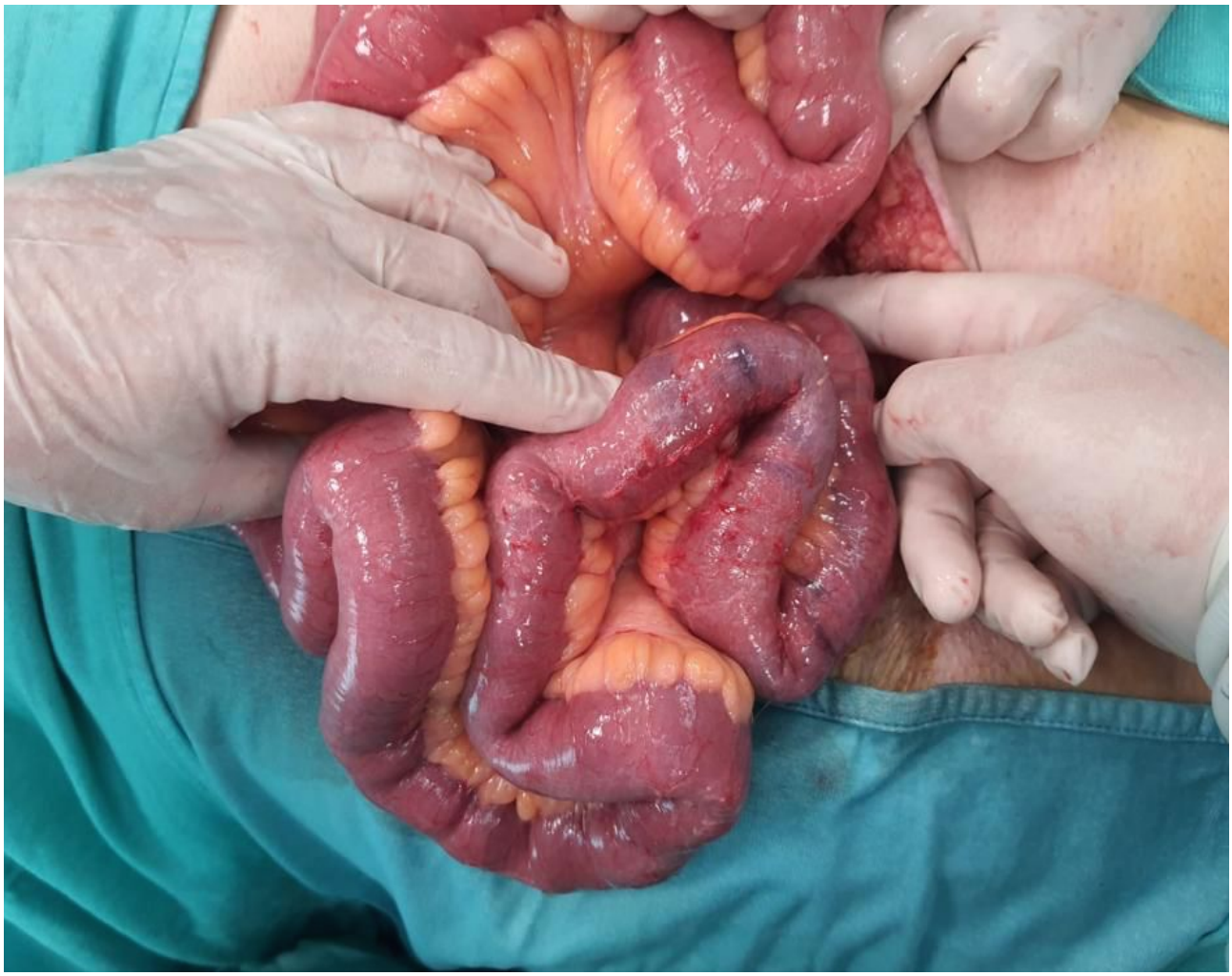

Figure 3. Intraoperative findings: Macroscopic examination of the abdominal cavity shows a swollen small intestine from 40 to 50 cm proximal to the ileostomy with areas of edematous mucosa interspersed with petechial hemorrhage; the small bowel had clear signs of congestion and edema but, despite CT images, no signs of macroscopically evident intestinal pneumatosis and necrosis were visible intraoperatively.