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RESEARCH ARTICLE

UNUSUAL PRESENTATION OF INTESTINAL VASCULITIS LEADING TO PERFORATION PERITONITIS

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ABSTRACT

Bowel perforation secondary to vasculitis of the gastrointestinal tract is rare. We describe the case of an 82-year-old woman who presented with abdominal pain, abdominal distension, breathlessness, anorexia, reduced urine output, and constipation. Laboratory investigations and cross-sectional imaging revealed a ruptured liver abscess with associated pneumoperitoneum, consistent with gastrointestinal perforation. This case underscores the need to investigate the underlying cause of unexplained liver abscesses when the patient is clinically stable. The patient underwent emergency exploratory laparotomy for the ruptured abscess and peritoneal contamination.

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INTRODUCTION

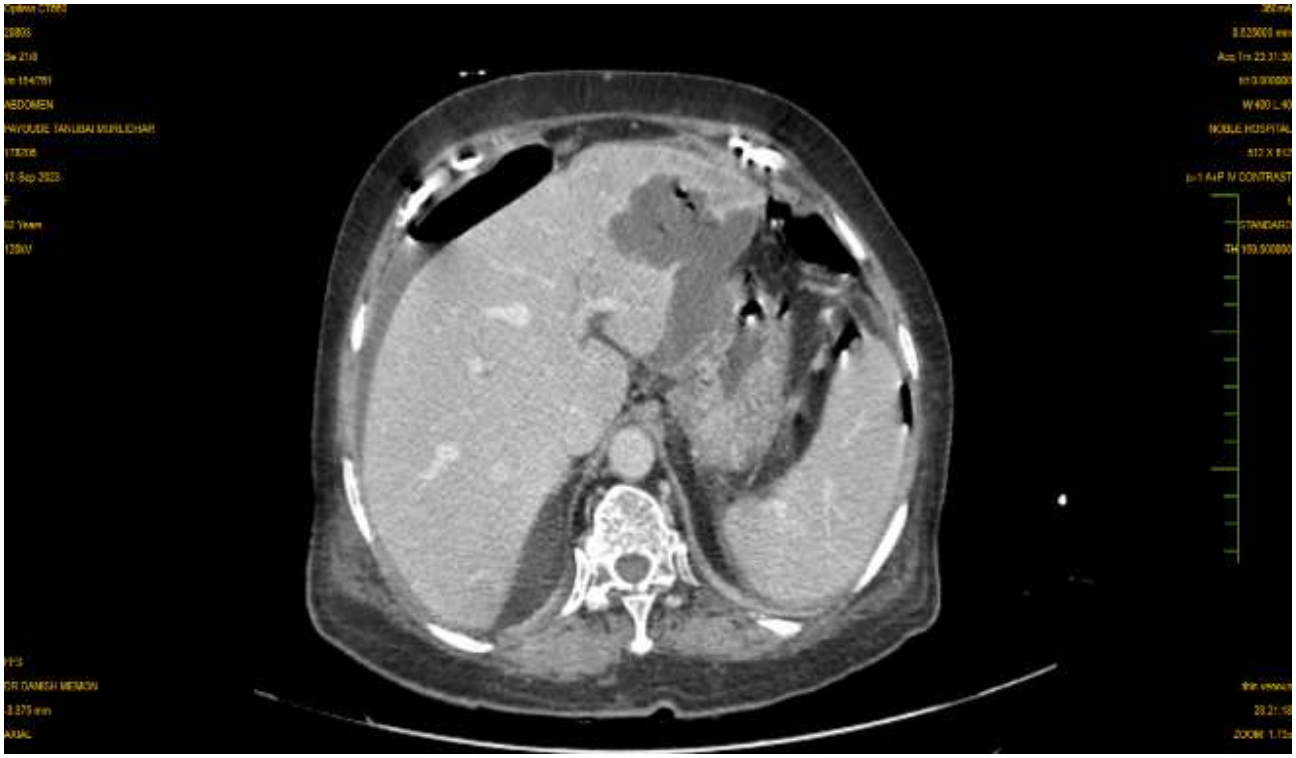
Vasculitis involving the gastrointestinal tract is an uncommon but potentially life-threatening condition(1). When complicated by bowel perforation, diagnosis is often delayed due to nonspecific clinical features. Hepatic abscesses in the setting of vasculitis are rare, and the association is infrequently reported in the literature. We present a case of an elderly woman whose initial presentation of abdominal symptoms was later found to be due to bowel perforation and hepatic abscess secondary to suspected gastrointestinal vasculitis.(2)

CASE PRESENTATION

An 82-year-old female with a history of diabetes, hypertension, and tubal ligation presented with complaints of abdominal pain, distension, constipation, per rectal bleeding, decreased urine output, and breathlessness, which had been gradually worsening over three days. There was no history of similar complaints in the past and no history of vomiting. On general examination, the patient was in septic shock with desaturation, decreased urine output, and hypertension. Abdominal examination revealed distension, a lump-like sensation in the epigastrium, generalized tenderness, guarding, and rigidity. Blood investigations showed a raised total leukocyte count of 13,900/mm³. Two-dimensional echocardiography demonstrated dilated ventricles with mild mitral and tricuspid regurgitation, possibly secondary to atherosclerotic changes.

Ultrasonography revealed a heterogeneously hypoechoic lesion without internal vascularity, likely representing an abscess in the subcapsular region of the left lobe of the liver. Further evaluation with triphasic CT showed a peripherally enhancing, thick-walled collection in segment III of the liver containing several air foci, with adjacent perihepatic and subcapsular free fluid communicating with the collection, suggestive of a ruptured abscess. The collection was closely abutting the lesser curvature of the stomach, with adjacent air foci raising suspicion of a concealed perforation. There was short-segment edematous wall thickening of the proximal jejunal loop with adjacent multiple air foci and a loculated intercommunicating collection, possibly representing a sealed perforation.Multiple air foci were also noted beneath both domes of the diaphragm and in the omental plane, consistent with pneumoperitoneum. The patient underwent an emergency exploratory laparotomy with resection of the partial transverse colon and descending colon (watershed area—splenic flexure), formation of a transverse colostomy, and marsupialization of the liver abscess. The resected specimen was sent for histopathological examination.

Histopathology of the transverse colon revealed a large perforation site with necrotic bowel wall. Omental biopsy demonstrated multiple microabscesses. Intraoperatively, bowel ischemia was noted predominantly around the splenic flexure, the watershed area.



Microscopic examination of the resected bowel showed the perforation site lined by necroinflammatory exudate. The colonic wall surrounding the perforation was completely necrotic. The remaining colon and surgical margins exhibited acute-on-chronic inflammation with features consistent with peritonitis. Postoperatively, the patient was transferred to the intensive care unit. Recovery was delayed due to advanced age, comorbidities, poor nutritional status, and multiple postoperative infections. The patient was eventually discharged with a colostomy and on supplemental oxygen support.

DISCUSSION

The proximal esophagus receives its blood supply from the inferior thyroid artery, while the mid-esophagus is vascularized by bronchial and esophageal branches of the aorta. The distal esophagus and stomach are supplied by branches of the celiac artery, which form a rich anastomotic network that typically prevents ischemic events—except in rare cases. In contrast, the small intestine and colon are more susceptible to ischemic injury. Midgut derivatives, spanning from the ampulla of Vater to the splenic flexure of the colon, are supplied by branches of the superior mesenteric artery (SMA). These vessels form overlapping arcades within the mesentery, offering collateral vascular support.(3) The hindgut, extending from the splenic flexure to the distal sigmoid colon, is supplied by the inferior mesenteric artery (IMA). Notably, regions such as the splenic flexure and rectosigmoid junction are considered “watershed zones” due to relatively poor collateral circulation, predisposing them to ischemic injury, often from non-occlusive causes.

Virchow’s triad outlines three major contributors to thrombosis:

- Hypercoagulability
- Hemodynamic changes (e.g., stasis or turbulence)
- Endothelial injury or dysfunction

Splenic flexure ischemia **may result from:**

- Severe hypotension due to dehydration, heart failure, major surgery, trauma, or shock.
- Arterial occlusion from thrombosis or severe atherosclerosis.
- Volvulus or herniation causing vascular compromise.
- Bowel obstruction from adhesions or tumors leading to distension and impaired perfusion.
- Vasculitis or hematologic disorders such as lupus or sickle cell anemia.
- Vasoconstrictive medications (e.g., used for cardiac or migraine treatment)
- Hormonal medications (e.g., oral contraceptives)
- Substance use (e.g., cocaine or methamphetamine)
- Extreme physical exertion (e.g., long-distance running)

Currently, the diagnosis of vasculitis-induced bowel perforation relies on clinical, histological, and angiographic evidence. However, these diagnostic tools—used individually or in combination—may yield inconclusive results. In fact, histological confirmation of vasculitis is found in only approximately 50% of cases. While case reports often cite histology consistent with vasculitis, larger studies reflect similarly low rates of histological confirmation.

There are two primary reasons for the absence of histological evidence

- **Proximal vascular involvement**: If the vasculitis affects proximal mesenteric vessels, leading to thrombosis and downstream ischemia, the resected specimen may show only patchy ischemia, necrosis, or thrombosis without direct evidence of vasculitis.
- **Necrotic tissue**: In cases of advanced ischemia or perforation, specimens may be predominantly necrotic,

precluding histological assessment of vascular inflammation.

- Given the low sensitivity of histopathology in these settings, a high index of clinical suspicion is essential for diagnosis.
- The case described above represents an example of histopathology-negative, clinically suspected vasculitis leading to bowel perforation in the watershed zone (splenic flexure). The underlying vasculitis may be primary or secondary, possibly triggered by local inflammation from a ruptured liver abscess. This underscores the diagnostic challenge and the need for clinical vigilance in identifying vasculitis as a cause of gastrointestinal ischemia and perforation.

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