Hepatic Portal Venous Gas Secondary to Intestinal Obstruction Complicated by Chronic Myeloid Leukaemia: A Case Report

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ABSTRACT

Hepatic portal venous gas (HPVG) is an exceptionally rare radiological finding characterised by the abnormal accumulation of gas within the portal vein (PV) and its intrahepatic branches, resulting from a variety of underlying causes. The presence of HPVG often signifies severe illness, poor prognosis, and exceedingly high mortality rates, hence regarded as an ominous sign of death. This article reports a case of HPVG secondary to intestinal obstruction in a patient with chronic myeloid leukaemia, accompanied by a thorough review of relevant literature. This report aims to enhance the understanding of clinicians and diagnostic accuracy regarding HPVG and provide valuable insights for its clinical diagnosis and management.

Key Words: *Hepatic portal venous gas, Chronic myeloid leukaemia, Intestinal obstruction, Intestinal necrosis.*

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INTRODUCTION

Hepatic portal venous gas (HPVG) is a rare radiological finding characterised by the abnormal accumulation of gas within the portal vein (PV) and its intrahepatic branches, arising from various aetiologies. It represents an exceptionally rare radiographic phenomenon.[1] In 1978, Liebman *et al*. first documented this radiological finding in adults, reporting a mortality rate of 75%.[2] HPVG generally signifies severe clinical conditions, poor prognosis, and extremely high mortality, earning it the label omen of death.[3]

Recently, our hospital successfully treated a patient with HPVG, which is presented below.

CASE REPORT

A 52-year male with a 10-year history of chronic myeloid leukaemia (CML) and long-term oral imatinib mesylate therapy was admitted in December 2024 with abdominal pain, distension, nausea, vomiting, and a 15-day history of cessation of anal flatus and bowel movements.

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Physical examination revealed abdominal distension without visible gastrointestinal patterns or peristaltic waves, diffuse abdominal tenderness with rebound tenderness and guarding, a tympanic percussion note throughout the abdomen, and hypoactive bowel sounds (approximately 2 per minute). A full abdominal non-contrast CT scan (Figure 1) showed multiple dilated bowel loops with significant pneumatosis and small air-fluid levels in some segments, along with areas of intramural gas. Gas densities were identified within the PV and its intrahepatic branches, the superior mesenteric vein, and local tributaries.

Preoperative diagnosis was made of intestinal obstruction, acute peritonitis, and CML. On surgical exploration, a small amount of yellowish, turbid exudate with a foul odour was identified within the abdominal cavity. Approximately one metre adjacent to the ileocaecal valve, the small intestine displayed adhesions, torsion, and stenosis involving the anterior abdominal wall and mesentery. Proximal to the stenotic adhesion site, approximately two metres of small intestine appeared greyish-black, lacking peristalsis and showing signs of necrosis. Distal to the stenotic adhesion site, 0.85 metres of the small intestine also exhibited greyish-black discolouration and necrosis. After dissecting the adhesions at the stenotic segment, a perforation was identified, indicating that a small intestinal perforation had led to surrounding adhesions and stenosis. No significant thrombosis was observed in the mesenteric vessels. During the operation, a segment of small intestine was resected that appeared dusky grey-black and contained a perforation.

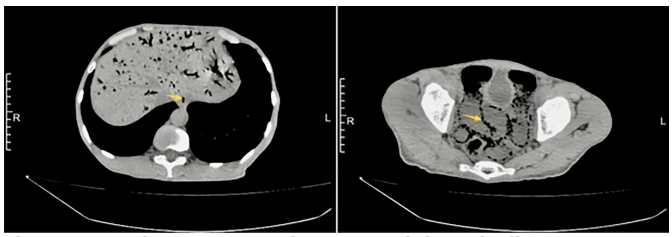

Figure 1: Portal venous gas and pneumatosis intestinalis.

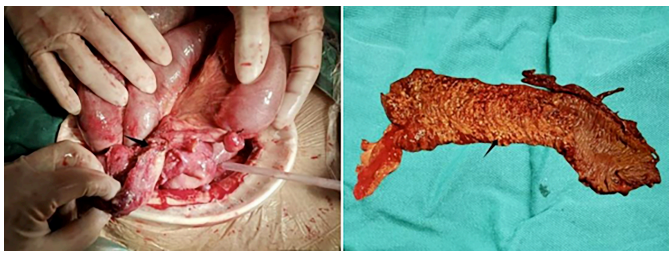

Figure 2: Small bowel perforation and necrosis, colonic mucosal erosion, and ischaemia.

Postoperative pathological examination of the intestinal segments revealed mucosal necrosis and sloughing, submucosal congestion and oedema, and transmural infiltration by numerous neutrophils. No significant reduction in ganglion cells was noted.

Aerobic and anaerobic cultures were performed on xanthochromic peritoneal pus isolates, identifying *Clostridium baratii* as the predominant anaerobe.

No major surgical complications (anastomotic stenosis, dehiscence, or haemoperitoneum) were observed during recovery. Follow-up computed tomography prior to discharge demonstrated total resolution of HPVG, with leucocyte counts returning to physiologic baseline.

DISCUSSION

HPVG can arise through two primary mechanisms:[4] firstly, mucosal damage and necrosis in the digestive tract, combined with increased intraluminal pressure, allow gas to enter the submucosal layer of the intestinal wall and subsequently reach the portal venous system *via* venous return. Secondly, pathogenic, gas-producing bacteria from intra-abdominal mesenteric or intestinal infections infiltrate the intestinal wall and mesentery, forming emphysema that directly introduces gas into the venous system, resulting in HPVG accumulation.[5] In this case, the patient had a 10-year history of CML and long-term oral imatinib mesylate treatment, which compromised immunity. Chronic intestinal obstruction elevated intraluminal pressure, causing ischaemic-hypoxic damage to the intestinal mucosa and necrosis of epithelial cells. These factors synergistically exacerbated the breakdown of the intestinal mucosal barrier, leading to pneumatosis intestinalis in the submucosal layers of the small intestine and colon. Necrotic portions of the small intestine and colon drained into branches of the superior mesenteric vein, allowing intraluminal gas to migrate through the submucosal venous network into the portal system *via* the superior mesenteric vein and its tributaries, ultimately causing HPVG.

HPVG is a pathological change observed in abdominal and intestinal diseases, rather than an independent clinical entity. It typically lacks specific symptoms and is often discovered incidentally during radiological studies,[6] but serves as an indicator of disease progression. Intestinal ischaemic necrosis is the most common cause of HPVG, although non-digestive conditions such as sepsis and abdominal infections may also lead to this clinical manifestation. In 2001, Kinoshita *et al*. reported an overall mortality rate of 39% in adult HPVG cases, with the three most common causes being intestinal necrosis (43%), gastrointestinal distension (12%), and abdominal abscess (11%).[7,8] Rare iatrogenic cases typically result from direct gas entry into the PVs during hepatic or gastrointestinal surgeries. Preoperative abdominal CT and postoperative pathological specimens in this case demonstrated HPVG with extensive intestinal wall pneumatosis (Figure 1). Intraoperative findings confirmed partial small bowel mesenteric torsion, resulting in ischaemic necrosis in the superior mesenteric vein-branch territories, accompanied by small intestinal perforation (Figure 2), thus confirming intestinal necrosis-induced HPVG rather than superior mesenteric vascular thrombosis.

Recent reports of HPVG cases associated with mild symptoms suggest that HPVG does not always correlate with poor prognosis.[9] Relying solely on HPVG for assessing disease severity has its limitations, as hepatic/gastrointestinal surgeries and gastrointestinal endoscopy can also induce HPVG.[10] When HPVG is detected through clinical imaging examinations (such as CT or colour Doppler ultrasound), it indicates disease progression and warrants heightened vigilance.

Early identification of the underlying aetiology of HPVG is essential for developing targeted diagnostic and treatment strategies tailored to the specific cause, ultimately reducing mortality rates and improving patient prognosis.

PATIENT'S CONSENT:

The patient provided explicit written consent for the publication of all case-related data, including de-identified medical records, diagnostic images, and clinical details mentioned in this manuscript.

COMPETING INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

SW: Conceptualisation, project administration, writing, reviewing, and editing.
XL: Writing of the original draft and supervision.
SL: Data curation, formal analysis, and methodology.
SL: Investigation and resources.
All authors approved the final version of the manuscript to be published.

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