

Extensive hepatic-portal and mesenteric venous gas due to sigmoid diverticulitis

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Abstract

Hepatic portal venous gas is most often associated with extensive bowel necrosis due to mesenteric infarction. Mortality exceeds 75% with this condition. The most common precipitating factors include ischemia, intra-abdominal abscesses and inflammatory bowel disease. In this report, we present a 75-year-old woman with extensive hepatic portal and mesenteric venous gas due to colonic diverticulitis. She had a 10-year history of type II diabetes mellitus and hypertension. She was treated by sigmoid resection and Hartmann's procedure and discharged from the hospital without any complications.

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INTRODUCTION

Hepatic portal venous gas (HPVG) is a rare condition and traditionally regarded to be an ominous finding of impending death, with highest mortality reported in patients with underlying bowel with ischemia^[1-6]. Colonic diverticulitis is an inflammatory condition and in very rare cases can be associated HPVG^[6-9]. HPVG can be due to two mechanisms: gas under pressure in the bowel lumen or to an alteration of the mucosa, allowing the gas to enter the portal system through the mesenteric veins; or gas-forming bacteria in intra-abdominal abscesses with or without a related pylephlebitis^[3-6]. If there is an underlying intramesocolic abscess and perforation in complicated diverticulitis, the prognosis is favorable, but the prognosis of HPVG due to septic thrombophlebitis and gas-forming organisms is poor^[6]. Another factor affecting HPVG and its prognosis is the existence of a long-term chronic disease, such as chronic renal failure, diabetes mellitus and hypertension^[5]. It has been reported that long-term chronic diseases decrease immune functions and alter the intestinal microbial flora and tolerance capability of the HPVG patients, which might lead to fatality^[5,10].

We report the case of a 75-year-old woman with type II diabetes mellitus and hypertension presenting with extensive hepatic, portal and mesenteric venous gas due to sigmoid diverticulitis.

CASE REPORT

A 75-year-old woman was seen in the emergency department with a 4-d history of mild abdominal pain and fever. Except for her temperature (38.2°C), her vital signs were normal. She had a 10-year history of hypertension and type II diabetes mellitus, and antihypertensive drugs and insulin therapy had kept her blood pressure and blood sugar level within normal ranges. On physical examination, she had mild tenderness in the left lower quadrant but no localizing peritoneal signs. Her serum C-reactive protein level was 220 mg/L, her platelet count was 55 000/mm³, and other laboratory findings were normal. A computed tomography (CT) scan of the abdomen revealed multiple gas foci in the main hepatic-portal vein, portal vein branches (Figure 1) and superior mesenteric (Figure 2A), splenic (Figure 2B) and inferior



Figure 1 Extensive gas accumulation in the hepatic-portal vein branches.

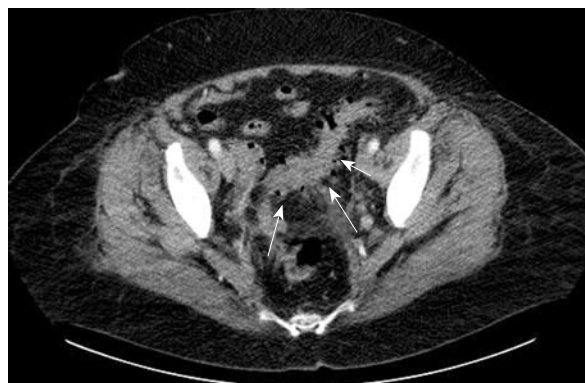


Figure 3 Diverticulosis in the sigmoid colon and mild inflammatory changes are present in the sigmoid mesocolon (arrows).

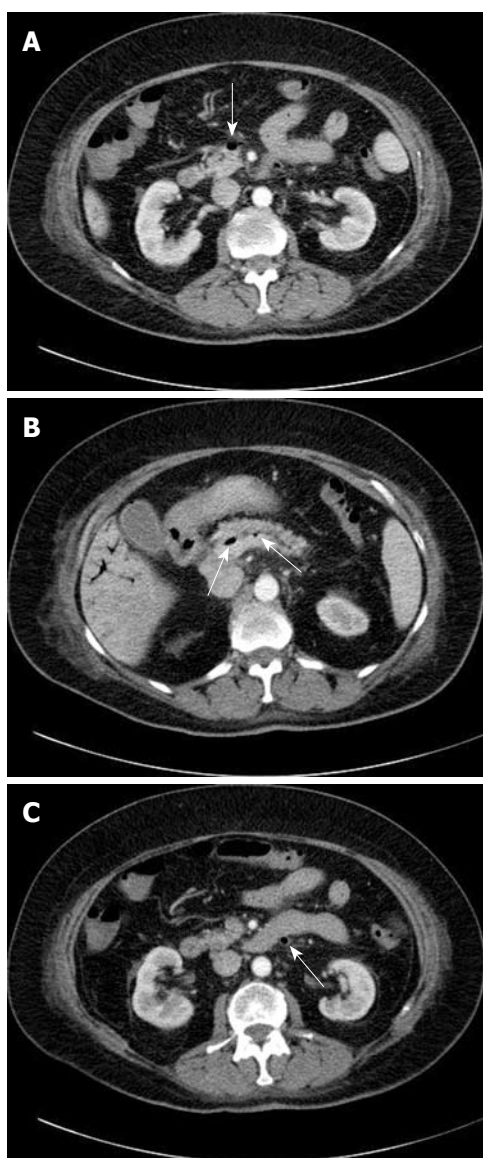


Figure 2 Gas observed. A: Superior mesenteric vein (arrow); B: Splenic vein (arrows); C: Inferior mesenteric vein (arrow).

mesenteric veins (Figure 2C). Sigmoid diverticulosis involved the mid and distal portions of the sigmoid colon. Slight wall thickening and inflammation suggesting localized sigmoid diverticulitis were observed (Figure 3).

The patient was transferred to the general surgery department, started on intravenous fluid therapy and antibiotics (ceftriaxone 2 g/d and ornidazol 1 g/d), and a laparotomy was planned for the same day. The emergency laparotomy revealed mild findings of diverticulitis in the sigmoid colon. A Hartmann's procedure and end colostomy were performed. The patient was discharged on the seventh postoperative day without further complications. Her colostomy was closed 8 wk after the first operation, and she reported no problems upon examination after 3 mo.

DISCUSSION

HPVG gas was first described by Wolfe and Evans^[11] in 1955 in fatal necrotizing enterocolitis in infants. This entity is most commonly associated with ischemic bowel disease. In the series of 64 patients reviewed by Liebman *et al*^[11], HPVG was mainly associated with necrotizing bowel disease (72%) and found to have a 75% mortality rate. HPVG venous gas has also been associated with such entities as bowel distention, perforated ulcer, acute hemorrhagic pancreatitis, corrosive ingestion and inflammatory bowel disease such as Crohn's disease^[6,12-15].

HPVG and thrombophlebitis is a rare complication of diverticulitis. Zielke *et al*^[3] reported that this entity was confined to nine patients with mesocolic abscesses. In 2007, Sellner *et al*^[6] reported a second review of 21 patients with complicated diverticulitis because of HPVG. In this report, 55% of cases had mesocolic abscesses and in the remaining 45%, mesocolic abscesses were absent and the patients presented with septic pylephlebitis. The authors suggested that patients with mesocolic abscess have better prognosis than patients with septic pylephlebitis. Despite our patient's symptoms, the clinical findings of diverticulitis were very slight. The CT findings revealed extensive multiple gas foci in the main portal vein, portal vein branches and superior mesenteric vein, and also revealed that the diverticulitis was milder than expected. We thought that this discordance might be due to virulence of pathogens or deficiency of the patient's immune system as a consequence of diabetes mellitus. Therefore, we decided

to perform a laparotomy on the same day. Operational findings of diverticulitis were quite limited and slight and we did not observe a mesocolic abscess. Nobili *et al*^[7] suggested that if medical conservative therapy could be resolute and the clinical status improves, the surgery could be delayed in these patients. Even though the clinical status of our patient was stable, we preferred to perform an urgent laparotomy because we thought that prognosis might be worse due to diabetes mellitus and gas-forming organisms. Chan *et al*^[5] have reported that long-term chronic diseases change the microbial flora in the intestine and decrease the immune function and tolerance capability of HPVG patients, which might lead to fatality. Our patient had type II diabetes mellitus and hypertension for 10 years, and extensive HPVG might be explained by increased aerobic and anaerobic microorganisms in the intestinal flora due to diabetes mellitus.

In conclusion, the clinical and radiological findings of HPVG associated with diverticulitis may be variable. We thought that, although the clinical status of our patient was stable, extensive HPVG in diverticulitis could be dangerous, due to a compromised immune system and alteration in intestinal flora, especially among elderly patients with chronic systemic diseases and no obvious mesocolic abscess or perforation, such as in our patient.

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