PNEUMATOSIS INTESTINALIS AND GAS IN THE SUPERIOR MESENTERIC VEIN AND HEPATIC PORTAL VEIN: A RARE PRESENTATION OF ACUTE PERFORATED APPENDICITIS

Kwok-Wan Yeung *,1 and Jun-Ping Shiau **

*Department of Radiology, Fooyin University Hospital, Taiwan, **Division of Surgery, Fooyin University Hospital, Taiwan

ABSTRACT

Purpose: Pneumatosis intestinalis in association with gas in the superior mesenteric and hepatic portal vein (portomesenteric venous gas) is typically due to bowel infarction and carries an ominous outcome. Much less commonly, it may be the result of nonischemic etiologies. We present a rare case of acute perforated appendicitis associated with pneumatosis intestinalis of the small bowel loop, and portomesenteric venous gas. Materials and methods: A 79-year-old male patient suffered from diffuse abdominal pain and cold sweating in the morning of the admission. Mild fever, tachycardia, diffuse abdominal distension and tenderness were noted. The blood analysis showed leukocytosis. A plain abdominal radiograph revealed distension of the bowel loop. CT of the abdomen showed swollen appendix with periappendiceal fluid, distension of small bowel loop, bowel wall gas in a segment of small bowel loop, and portomesenteric venous gas. Therefore, a CT diagnosis of acute perforated appendicitis, and probably bowel infarction was made. Results: Emergency exploratory laparotomy was performed. Fecal material was seen surrounding the cecum, and the appendix showed gangrenous change and perforation. Appendectomy was done. A 30 cm segment of ileum disclosed hyperemic change and marked distension, and decompression was performed. Two days later, second-look laparotomy showed no bowel ischemia. Due to persistent post-operative fever and turbid discharge from the surgical site, the patient’s family asked for a transfer to the other hospital on the 15th postoperative day. The patient was finally discharged after infection control at the other hospital for a 43-day period of the second hospitalization. No bowel ischemia was noted during the second course of the hospitalization. Conclusion: The mechanism of pneumatosis intestinalis and portomesenteric gas may be due to increased intraluminal pressure and associated mucosal disruption of the small bowel, leading to gas entering the bowel wall, through the superior mesenteric vein and subsequently to the hepatic portal vein. The clinical outcome is determined by the underlying disease and not just the presence of superior mesenteric and portal venous gas.

KEYWORDS acute appendicitis, pneumatosis intestinalis, portomesenteric venous gas
Figure 1 (A,B) (A) CT topogram of the abdomen showed distended small bowel loop. (B) Non-contrast-enhanced CT showed the swollen appendix (arrow) with a diameter of 0.9 cm and with periappendiceal infiltration and minimal fluid accumulation.

Introduction

Pneumatosis intestinalis in association with gas in the superior mesenteric and hepatic portal veins is typically due to bowel infarction and carries an ominous outcome [1-3]. Much less commonly, it may be the result of nonischemic etiologies, such as bowel trauma or surgery, inflammatory or infectious bowel disease [4-6]. Acute appendicitis is a rare cause leading to hepatic portal venous gas (HPVG) [7-9]. We present a rare case of acute perforated appendicitis associated with pneumatosis intestinalis of the small bowel loop, and gas in superior mesenteric vein and hepatic portal vein (portomesenteric venous gas).

Case report

A 79-year-old male patient suffered from constipation for 7 days, and diffuse abdominal pain and cold sweating in the morning of the admission. He was sent to the emergency room with mild fever (37.9°C), tachycardia (120/minute), diffuse abdominal distension and tenderness. A history of diabetes mellitus, hypertension and chronic obstructive pulmonary disease (COPD) was identified. The blood analyses showed elevated white blood cell count (13450/µL, with 7% in band form), C-reactive protein (41.45 mg/dL), creatinine (4.55 mg/dL), and random glucose (537 mg/dL). Plain abdominal radiograph revealed distension of the bowel loop. Due to the impaired renal function, only non-contrast-enhanced computed tomography (CT) of the abdomen was performed, which showed swollen appendix (with a diameter of 0.9 cm), periappendiceal fluid, distension of small bowel loop, bowel wall gas (pneumatosis intestinalis) in a segment of small bowel loop, and gas in the superior mesenteric vein and hepatic portal vein (portomesenteric venous gas). Therefore, CT diagnosis of acute perforated appendicitis, and probably bowel infarction was made. Emergency exploratory laparotomy disclosed thirty c.c. turbid ascites in right lower abdominal cavity, fecal material surrounding the cecum, and the appendix with gangrenous change and perforation. Appendectomy was performed.

A 30 cm segment of ileum showed hyperemic change. The whole small bowel loop was markedly distended, and decompression was performed. In fear of bowel ischemia, second-look laparotomy was carried out two days later. Fair bowel perfusion and mild distension of the small bowel loop and colon were found. No ischemia of bowel loop was identified. Bowel decompression was carried out. Despite aggressive treatment with antibiotics, the condition of the patient did not improve, with persistent post-operative off-and-on fever, turbid discharge from the surgical site, and much drainage amount from the nasogastric tube. The abdomen remained soft on palpation. The patient’s family asked for transfer to the other hospital for further management on the 15th postoperative day. During the 43-day period of the second hospitalization at the other hospital, the patient received medical treatment for the septic shock related to surgical wound, intraabdominal infection and urinary tract infection, and was then discharged. No bowel ischemia was noted during the second course of the hospitalization.

Discussion

Hepatic portal venous gas (HPVG) and mesenteric venous gas detected on imaging studies have been shown to be an ominous radiological sign in bowel ischemia and infarction [1], and carries a high mortality rate (75%) based on plain abdominal
radiograph before 1978, and a substantially lower mortality rate of only 39% at the beginning of 21th century, thanks to the development of computed tomography (CT), especially multidetector row CT with its high sensitivity to detect HPVG in more benign and non-life threatening disease entities [2-3]. The less common etiologies include gastrointestinal inflammation (such as enterocolitis, Crohn’s disease, ulcerative colitis), obstruction and dilatation, infection (such as diverticulitis and acute appendicitis), sepsis, injury due to iatrogenic procedures, abdominal trauma, gastrointestinal malignancies, and thrombophlebitis of the superior mesenteric vein. Other rare causes are respiratory tract infection, diagnostic and therapeutic gastrointestinal procedures, and drugs. In approximately 15% of cases, the HPVG is idiopathic [3].

Portal and mesenteric venous gases may be found together in some disease entities, such as the case presented here, or they may be present alone in the absence of the other [3]. Portal pylephlebitis and liver transplantation may cause portal venous gas without the presence of mesenteric venous gas; while in the intestinal disease process, mesenteric venous gas may be detected earlier before the gas reaches the portal venous system. Pneumatosis intestinalis (PI) is characterized by cystic or linear non-communicating accumulation of gas in the submucosal and subserosal regions of the intestinal wall. HPVG may be present with pneumatosis intestinalis in approximately 50% of cases [2] and results from the passage of bowel wall gas from the mesenteric venous drainage into the hepatic portal venous system. Concurrent presence of both the PI and HPVG has been shown to be associated with bowel ischemia and carries a high mortality rate [4-5].

The mechanism by which the gas enters the portomesenteric venous system is unclear. However, several mechanisms were proposed [3-8]. First, some disease processes, such as bowel ischemia, can cause bowel wall ulceration and necrosis, thus enhancing the intraluminal gas passage into the portomesenteric venous system. Second, bowel distention causes increased intraluminal pressure, thus leading to mucosal disruption and forcing intraluminal air into the mesenteric vein. The present case may be an example of this mechanism, because the mesenteric venous gas is present near the distended small bowel loop with or without the presence of PI, and absent near the site of the swollen appendix. Thirdly, intraabdominal sepsis related to infectious processes such as diverticulitis, appendicitis, cholecystitis, and intraabdominal abscess may produce portomesenteric venous gas from pylephlebitis of mesenteric and portal veins, bacterial degradation of carbohydrates and abscess-induced mesocolic gas perforation to the mesocolic vein. Fourthly, in 15% of cases, portomesenteric venous gas is idiopathic in such cases as organ transplantation, and pulmonary disease (e.g. COPD). In the case presented here, the combination of the bowel distension and intra-abdominal sepsis related to bacterial proliferation in acute appendicitis might cause increased intraluminal pressure and associated mucosal disruption of the small bowel, leading to gas entering the bowel wall, through the superior mesenteric vein and subsequently to the hepatic portal vein. The coexistence of COPD in the patient in the present case may be the other cause for the development of portomesenteric venous gas.

The diagnosis of HPVG is made using various imaging examinations. A plain abdominal radiograph may detect two-thirds of cases of PI, and only 12.5% of cases of HPVG [1, 6-8]; however, the detection is often difficult and is easily overlooked. US and CT are superior to the abdominal radiograph in identifying HPVG [1, 6]. The typical US features of HPVG include hyperechoic dot-like or streak-like patterns flowing within the portal vein and hyperechoic patches within the liver parenchyma in the non-dependent site, which is more easily detected on the real time phase. US can detect HPVG which is missed on CT [9].

CT is the gold standard for the detection of HPVG, mesenteric venous gas, PI, and the underlying causes [1, 6]. The HPVG appears as hypodense, tubular regions, predominantly in the left lobe of liver, and branching within 2 cm beneath the liver capsule, which is caused by the centrifugal flow of portal vein. This is in contrast to the centrally located pneumobilia related to the centripetal flow of the bile. CT can detect the underlying etiologies, both the ischemic bowel and other benign and non-life threatening causes of PI, mesenteric venous gas and HPVG.

The presence of HPVG alone cannot be an indication for emergency laparotomy. When CT reveals portomesenteric venous gas with concurrent signs of mesenteric ischemia, the operation is mandatory. However, the degree of portomesenteric venous gas does not necessarily imply a poor prognosis [3]. HPVG due to benign and straightforward etiologies can be treated in conservative management. The radiological detection of HPVG should not determine the clinical and surgical management [2]. The prognosis is related to the pathology itself, depends on the severity of the causal disease process, and is not influenced by the presence of HPVG [6-7].

Conclusion

In diseases with concurrent appearance of HPVG, the clinical outcome should be determined by the underlying disease, and not just the presence of superior mesenteric and portal venous gas. In this setting, CT is the modality of choice for evaluation. Decision making regarding diagnosis should be given with the context of both the clinical and imaging findings, and the most appropriate treatment should be applied to the patient to avoid unnecessary surgery in the benign disease processes.

Competing interests

The authors declare that they have no competing interests.

References


