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HEPATIC PORTAL VENOUS GAS

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Abstract

Hepatic portal venous gas (HPVG), an ominous radiologic sign, in most of the cases is associated with a severe abdominal disease that requires an urgent surgical intervention. In the medical literature, HPVG has been commonly associated with severe or lethal conditions.

The diagnosis of HPVG is usually made by plain abdominal radiography, ultrasonography, color Dopler flow imaging or computed tomography (CT) scan.

The increased use of CT scan in the inpatient setting allows early and highly sensitive detection and also recognition of an increasing number of benign and non-life threating causes of HPVG. The prognosis is related to the pathology itself and is not influenced by the presence of HPVG.

Key words: hepatic portal vein gas; bowel ischemia/necrosis; pneumatosis intestinalis; abdominal computed tomography scan; acute abdomen

Introduction

Hepatic portal vein gas (HPVG) is an uncommon feature of acute abdomen. Its radiological findings were first described by Wolfe and Evans in infants with necrotizing enterocolitis (NEC) in 1955 [1], and then an increasing frequency in adults was reported. The first case of HPVG in an adult was published in 1960 [2].

HPVG is associated with numerous underlying abdominal diseases, ranging from benign causes to potentially lethal diseases like an acute gastrointestinal catastrophe, such as mesenteric ischemia, with up to 80% of cases resulting in death [3]. In cases of mesenteric ischemia in which HPVG is found, abdominal distension, pain and fever are often present.

However, the extent of portal venous gas or pneumatosis intestinalis, gas seen on imaging in the small or large intestine, is not predictive of the extent of the disease itself or of the operative findings [4].

The mechanism for the appearance of gas in the portal vein is not well understood. The proposed factors predisposing the portal venous system to the accumulation of gas include the following: (1) escape of gas produced by gas-forming organisms in the bowel lumen or in an abscess which then circulates into the liver, or (2) the presence of gas-forming organisms in the portal venous system with passage of gas into the circulation [5].

Radiological signs of abnormal air density in the hepatic region, either with or without gastrointestinal disturbance are diagnostic clues for such urgent conditions. The diagnosis of HPVG is usually made by plain abdominal radiography, sonography, color Doppler flow imaging, or computed tomography (CT) scan.

The characteristic finding on abdominal plain film is a branching radiolucency extending to within 2 cm beneath the liver capsule. This is because of the centrifugal flow of portal venous blood, which carries portal venous gas peripherally in contrast to biliary gas, which tends to collect centrally as a result of the centripetal movement of the bile.

Although HPVG may be diagnosed by conventional radiography, detection is difficult and it is easily overlooked. Sonography, color Doppler flow imaging and CT scan have been reported to be superior to abdominal radiographs in identifying HPVG [6].

Among these imaging modalities, CT is the most sensitive and specific for detecting HPVG and for demonstrating associated intra-abdominal disorders and coexisting abnormal air.

The CT scan has a high sensitivity for detection of HPVG and can detect the underlying pathology. Also, a CT scan can disclose gas in the bowel wall (pneumatosis intestinalis) and in the extrahepatic portal vein or its splanchnic vasculature.

Demonstration of associated intra-abdominal conditions is essential for planning treatment. On the other hand, prediction of fatalities helps to avoid ineffective aggressive procedures. We present five fatal cases of enormous HPVG. These patients died within 2 d after initial presentation at the emergency department, despite intense medical treatment. We present these cases in order to better understand the clinical course and CT findings of HPVG [7].

Presentation of a case

A 58-year-old woman presented to the emergency department, complaining of constipation, crampy abdominal pain with diarrhea, nausea and bilious vomiting over the preceding 2 days. She denied fever, chills or night sweats. Her vital signs were within normal limits, but her abdomen was tense and rigid. Laboratory analysis was notable for leukocytosis, high CRP level (196.2 mg/L), LDH (858 U/L), AST (162 U/L), ALT (136 U/L).

A plain abdominal radiography demonstrated diffuse gaseous distension of the small and large bowel, and HPVG vas visible (Figure 1).

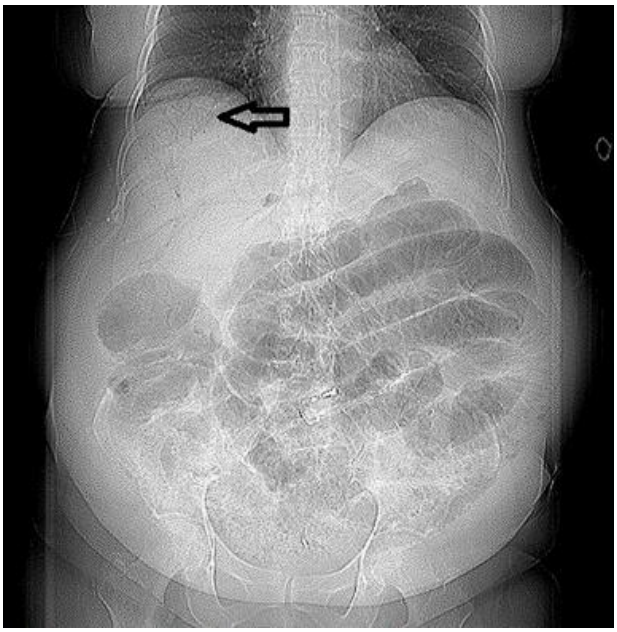
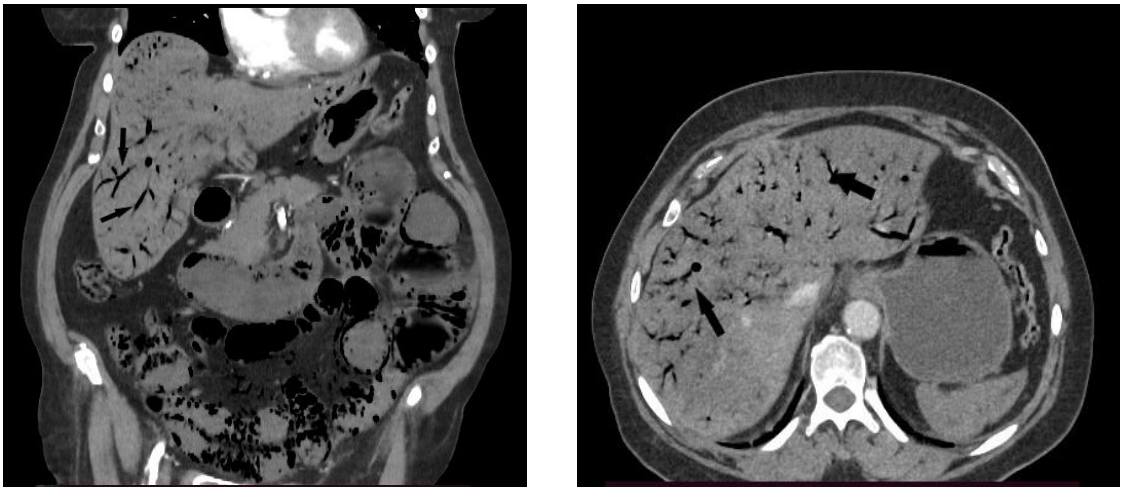


Figure 1. A frontal plain abdominal radiography obtained in the supine position demonstrates distended loops of bowel and extensive hepatic portal venous gas (arrow).

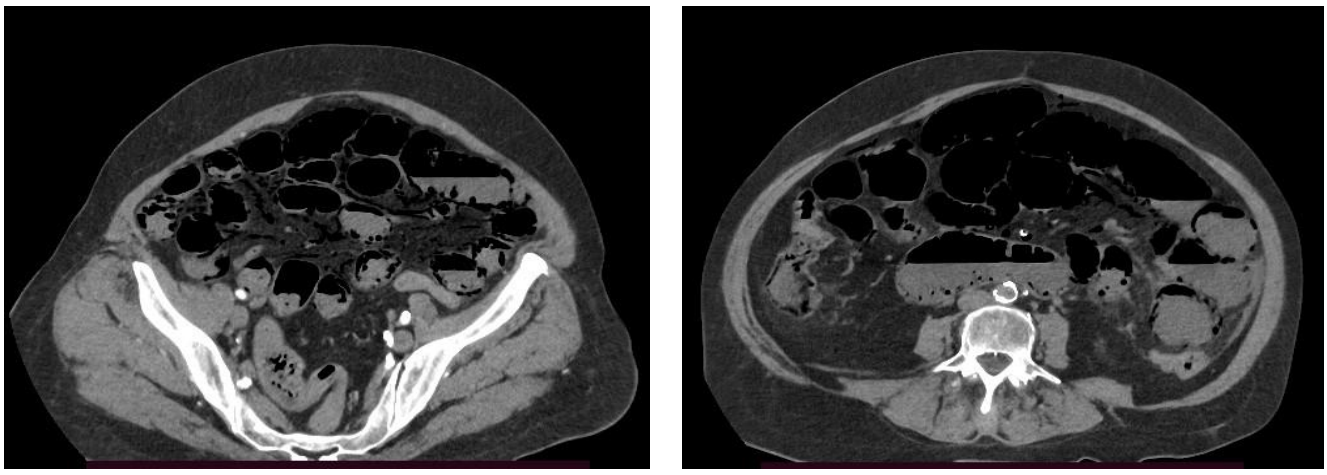


2.A 2.B

Figure 2. Coronal (A) and axial (B) views of contrast-enhanced computed tomographic (CECT) images of the liver with extensive hepatic venous gas (arrows).

A contrast-enhanced abdominal CT was performed and diffuse gaseous distension of the small bowel and colon with pneumatosis and portal venous gas was confirmed (Figures 2 and 3).

The initial procedure consisted of explorative laparotomy. Intraoperatively, thrombosis of superior mesenteric artery was found as well as a large gangrenous segment of the small bowel. Unfortunately, during the surgery procedure the patient died.



3.A 3.B

Figure 3. (A) and (B) Axial contrast-enhanced computed tomography (CECT) of abdomen from a case with extended pneumatosis intestinalis.

Discussion

Bowel ischemia and/or infarction is a common and dangerous abdominal condition, especially in elderly patients and is associated with a high mortality rate.

Bowel ischemia is produced by insufficient blood flow to or from the intestines. It may have an acute or chronic setting depending on the underlying disorder. The extent of bowel ischemia in the bowel wall is divided into three stages: Stage Ⅰ: the ischemic lesions are confined to the mucosa and are reversible (known as reversible ischemic enteritis); Stage Ⅱ: characterized by necrosis of the mucosal and submucosal tissues, which may lead to fibrotic stricture development; Stage Ⅲ: the entire wall is affected by ischemia. Intestinal ischemia results in damage to the mucosal barrier which, in association with over-distension of the bowel loops and gas-forming bacterial proliferation, leads to gas moving from the intestinal lumen to the mesenteric veins and flowing through it to the portal system and hepatic parenchyma [6].

Clinical scenarios leading to HPVG generally fall into three categories: bowel distention/obstruction, ischemia, and idiopathic. These mechanisms are coupled with the two main theories proposed for the pathophysiologic etiology of HPVG: mechanical versus bacterial. Firstly, mechanical disruption of mucosal integrity may result in dissection of gas into the intestinal wall and eventually the portal system. The breach of integrity of mucosa may be related to ulceration from ischemia, IBD, peptic ulcer disease (PUD), or from gastrointestinal neoplasms. Alternatively, the invasion or translocation of the intestinal wall by gas forming bacteria may result in the production of gas within the intestinal wall and portal system itself. Likely both mechanisms play a role in the development and propagation of HPVG [8].

HPVG is often associated with pneumatosis intestinalis, posing a grave prognosis, especially in the ischemic intestine. The CT scan alone cannot predict which patients are experiencing true intestinal ischemia and which simply have benign pneumatosis. The presence of HPVG does not provide any information concerning the extent of bowel necrosis. In all cases, CT findings should be correlated with the clinical signs and with laboratory parameters to reach a high sensitivity and specificity level for intestinal necrosis. When HPVG associated with ischemic bowel disease is encountered, coexisting other abdominal conditions should be considered pre- and intraoperatively. Intestinal resection is performed when bowel necrosis is found on laparotomy [6].

Assessment of the varied presentation of patients with HPVG has led to the suggestion of new algorithms in recent years to better identify patients who would benefit from operative intervention versus those who may not. Despite the small number of patients examined in these studies, all placed a similar emphasis on the clinical status of the patient, rather than on the CT findings alone, including physical exam findings, vital signs, and laboratory values [8]. Koami et al. found that in their sample of 33 patients with HPVG, using a criteria of lower blood pressure (<systolic BP 108 mmHg), higher lactate dehydrogenase (LDH) (>387 U/L), and the presence of pneumatosis intestinalis led to 100% of sensitivity and 78.9% of specificity for a necrotic bowel .

Conclusions

Our patient had a severe mesenterial thrombosis with gangrenous small bowel that caused an extensive HPVG. The aggressive surgical approach did not give the expected result and the patient ended fatally.

A radiologic finding of HPVG was considered as an indicator of the bad prognosis and was associated with a particularly high mortality rate. Nowadays, with the development of highly advanced imaging techniques, potentially severe pathologies, such as bowel ischemia, are diagnosed at much earlier stages, allowing prompt treatment and significantly reduced mortality rates. HPVG is not by itself a surgical indication and the treatment depends mainly on the underlying disease. The prognosis is related to the pathology itself and is not influenced by the presence of HPVG.

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