Hepatic Portal Venous Gas: A Case Report

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ABSTRACT

Cases of hepatic portal venous gas have been diagnosed more frequently because of the widespread use of advanced radiological imaging modalities. It is considered an ominous radiological finding, and is often associated with severe abdominal disease requiring urgent operative intervention. It is also associated with many benign and non-life-threatening conditions. Hence, detection of hepatic portal vein gas is not an indication for surgery by itself, and the treatment depends mainly on the underlying disease. Likewise, prognosis depends on the underlying pathology. In this article, we discuss a case of hepatic portal venous gas that we encountered in our hospital, together with a review of the literature.

Key words: Hepatic portal venous gas, Bowel ischemia

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ÖZET

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CASE REPORT

A 21-year-old female patient with no known comorbidities presented to the Accident and Emergency Department with the complaints of sudden onset of abdominal pain and distension of the abdomen for one day. The pain started periumbilically and became generalized. She did not have vomiting or fever. Her menstrual history was normal with no previous abdominal surgeries. On the general physical examination, the patient was conscious and oriented. Her pulse rate was 120 beats per minute and blood pressure was approximately 80/50 mmHg. The abdomen was distended with board-like rigidity throughout, and bowel sounds were negative. In view of her hemodynamic instability, she was resuscitated with crystalloids, and blood samples were drawn for grouping and cross-matching. With fluid resuscitation, her blood pressure increased, and she was immediately taken to the Radiology Department. Plain X-rays of the chest and abdomen were obtained, which revealed massive free intraperitoneal air. She underwent plain computed tomography (CT) scan of the abdomen, which showed massive gas in the intra- and extrahepatic portal vein, superior mesenteric vein, and splenic vein. Massive free intraperitoneal air and sub-diaphragmatic fluid collections were seen, which pushed the liver inferiorly and medially (Figures 1-3).

After returning from the radiological investigation, she once again became tachypneic and tachycardic, and her blood pressure dropped to 60/40 mmHg. Since there was massive distension of the abdomen, which was causing respiratory distress, a 22-gauge needle was inserted into the peritoneal cavity in the right hypochondriac region, and the free air was aspirated. Resuscitation measures were continued with crystalloids, colloids, vasopressors, and oxygen supplementation. Despite this, the patient died due to cardiorespiratory failure without a sufficient window for urgent surgical intervention.

LITERATURE REVIEW

Hepatic portal venous gas (HPVG) was first described in 1955 by Wolf and Evans. Air in the portal venous system is a relatively rare but ominous sign, usually indicating a serious intraabdominal pathology. Most cases are caused by mesenteric vascular occlusion and subsequent bowel necrosis. The other common causes are digestive tract dilatation, peptic ulcer, and inflammatory bowel disease\(^\text{[3]}\).

The mechanism by which the gas enters the portal circulation is unclear. The proposed hypotheses are: 1) the gas is forced into the venous system by mucosal

![Image 1](https://example.com/image1)

Figure 1. CT image showing branching pattern of air within 2 cm of the liver capsule. Also noted is massive pneumoperitoneum and subdiaphragmatic fluid, which has pushed the liver downward and medially.

![Image 2](https://example.com/image2)

Figure 2. Air is seen in the splenic vein and superior mesenteric vein (arrow).

![Image 3](https://example.com/image3)

Figure 3. Air is seen in the right and left branches of the portal vein.
barrier disruption or by tension in the distended bowel; 2) the gas is formed by the microorganisms inside the intestinal wall or venous system; and 3) there is an increase in the luminal gas pressure as in bowel obstruction or during a gastrointestinal endoscopic procedure[4].

The clinical presentation can be divided in three categories based on PVG pathophysiology. These are 1) ischemic pathology, 2) bowel obstruction, and 3) infectious processes. The commonest cause is ischemic bowel necrosis (43%). The other causes include acute digestive tract dilatation (12%), intraperitoneal abscesses (11%), ulcerative colitis, complication of endoscopic procedures, intraperitoneal tumors, and others (15%)[4]. The clinical presentation depends on the cause of HPVG, ranging from circulatory collapse at presentation to mild subtle peri-abdominal symptoms and signs.

Diagnosis

Diagnosis can be made with imaging studies such as plain film of the abdomen, ultrasonography and CT scan of the abdomen. Of these, the abdominal CT scan is the most sensitive. In the plain films, subtle changes with peripheral radioluencies may be seen. Plain X-ray can detect HPVG in only 20% of cases. On gray scale sonography, PVG can be seen as flowing echogenic bubbles. CT scan of the abdomen shows tubular areas of diminished attenuation in the liver, predominantly in the left lobe of the liver, as the left portal vein is more vertical in position[5].

This entity has to be differentiated from pneumobilia. In HPVG, the gas extends within 2 cm of the hepatic capsule due to centrifugal circulation of the blood, whereas the gas in the biliary system is primarily located centrally due to the centripetal circulation of bile. The amount of gas detected is not related to the severity of the underlying disease. The advantage of CT is that it can provide information regarding the underlying etiology of HPVG, e.g., bowel ischemia.

Treatment

The indications for surgery in portal mesenteric vein gas are based on the underlying cause of the disease rather than the HPVG per se. When intraabdominal sepsis and bowel necrosis are present, urgent surgical treatment is called for. Otherwise, broad spectrum antibiotics and other conservative measures are sufficient in benign conditions that cause HPVG[6].

The mortality rate varies depending on the underlying pathology. The mortality is highest if the HPVG is associated with bowel necrosis (75%), and the degree of bowel necrosis is shown to affect mortality. The overall mortality rate is about 39% considering both severe and less-severe cases of HPVG[7].

In conclusion, HPVG is being increasingly detected possibly due to the frequent use of CT scans due to its easy availability. The majority of patients have bowel necrosis due to mesenteric ischemia, but the proportion of patients with other benign conditions that cause HPVG are increasing. The latter subset of patients has a better prognosis. Close observations would be appropriate in stable patients without specific findings of intraabdominal catastrophe.

REFERENCES


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