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Hepatic Portal Venous Gas in Emergency Department: A Case Report

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Abstract

A 73-year-old man had vomiting for 15 days and severe abdominal pain for 2 days. Abdominal examination showed widespread tenderness and defense. Computed tomography showed gas images in the portal and mesenteric veins (porto-mesenteric venous gas), wall thickening in all intestinal loops and intramural gas image in the intestinal wall (pneumatosis intestinalis). The patient who had a high surgical risk died after the operation. In this article, we aimed to discuss the differential diagnosis, treatment and prognosis of HPVG in patients with current literature.

Keywords: Mesenteric ischemia, Pneumatosis intestinalis, Porto-mesenteric venous gas

Introduction

Hepatic portal vein gas (HPVG) is a rare radiological finding. It was initially noticed in patients with mesenteric ischemia or intestinal gangrene. Currently, it has been reported in cases with diverticulitis, necrotizing bowel diseases, blunt abdominal trauma, bowel obstruction, ulcerative colitis, intra-abdominal abscess, gastric ulcer, gastric cancer and colon surgery^{1,2}. 70-80% of pneumatosis intestinalis and HPVG cases can be seen together³. HPVG, in combination with pneumatosis intestinalis, is a condition requiring immediate surgical intervention.

Early diagnosis and rapid surgical intervention are necessary to prevent large bowel necrosis. Gas can be easily diagnosed by radiographic, ultrasonographic or computed tomography (CT) findings in the hepatic portal vein⁴.

In this article, we present a rare case with HPVG that caused by mesenteric vascular disease with the images.

Case

A 73-year-old male patient was admitted to the emergency department with complaints of abdominal pain especially in the epigastric region, nausea and vomiting, which started two days earlier. The patient had amyloidosis, congestive heart failure, chronic renal failure and dialysis three days a week. In the last 6 months, he has been lost 30 kilograms.

On physical examination, his body temperature was $37,6^{\circ}$ C, heart rate was $120 / \min$, respiratory rate was $28 / \min$ and blood pressure were 100/70 mmHg. The patient had cachectic, abdominal distended, defensive and extensive tenderness. Intestinal sounds were hypoactive. In laboratory examinations, leukocyte count was 19000 / uL, blood urea nitrogen was 61 mg / dL (normal value 15-40 mg / dL), creatinine was 4,2 mg / dL, potassium was 5,6 mEq / L. Coagulation parameters and C reactive protein tests were within normal limits. In arterial blood gas, Ph was 7.4, PaO_2 was 88 mmHg, $PaCO_2$ was 32 mmHg, SaO_2 was 97% and lactate was 3,5 mg / dL.

In the direct abdominal radiography, diffuse gas distension is observed in all bowel loops and pneumatosis intestinalis in intestinal walls (Figure 1). Abdominal tomography showed air values in portal veins and mesenteric veins within the liver (portomesenteric venous gas) (Figure 2). Severe dilatation was observed in intestinal loops. Non-contrast abdomen CT scan of the lower abdomen showed diffuse pneumatosis intestinalis in the intestinal walls (Figure 3).

He was operated on with a preliminary diagnosis of acute abdomen. Colon ischemia was seen in the intestinal exploration until full-thickness small intestine and splenic flexure, and no resection was performed. The patient was followed up in the post-op intensive care unit and was connected to a mechanical ventilator. On the same day, her blood pressure decreased gradually and did not improve despite inotropic support. Patient who did not respond to resuscitation died on postoperative first day.

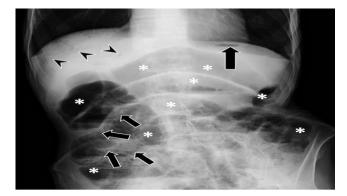


Figure 1. Direct abdominal X-ray image of the case. In all bowel loops, diffuse gas distension is noted (asterisks). Pneumatosis intestinalis in the intestinal walls is more prominent in small bowel loops, especially in the right lower quadrant (small arrows). The free air image in the subdiaphragmatic area on the left suggests perforation (large arrow). In addition, linear lucent areas of the liver are compatible with protal venous gas (arrowheads).

Written informed consent was obtained from the children of the patient for publication of this case report and accompanying images.

Discussion

Mucosal damage, transmural ischemia, intestinal distension especially in patients with mesenteric ischemia and increased intra-abdominal pressure in the trauma are the main causes of gas formation in the hepatic portal vein².

Acute intestinal ischemia is divided into four main clinical categories pathologically and histologically: acute mesenteric embolus, acute mesenteric thrombus, non-occlusive condition and mesenteric vein thrombosis⁵. The severity of intestinal ischemia can range from mild and transient intestinal mucosa damage to life-threatening transmural bowel in-

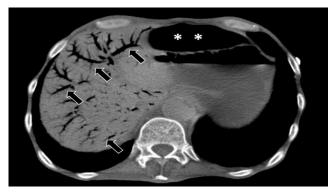


Figure 2. CT image of the abdomen without contrast material. There is widespread gas in the portal venous system in the liver (arrows). In addition, the image of free air in a large area of the stomach anterior is remarkable (asterisks).

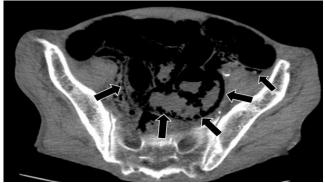


Figure 3. CT section of the non-contrast abdomen through the lower abdomen. Pneumatosis intestinalis is common in the intestinal walls (arrows).

farction⁶. Early diagnosis and treatment determine outcome. Increasing physician awareness and initiating timely diagnosis and treatment in patients with suspected mesenteric ischemia is important in saving the patient's life7. The absence of specific symptoms in the clinical examination may complicate an appropriate assessment. The clinical course of bowel ischemia occurs in three stages. In the first stage (0-6 hours), acute abdominal pain present and shock, accompanied by diarrhea. In the second stage (7-12 hours), there is a silent phase which shows a rapid clinical deterioration with intestinal paralysis, and in the last stage (12-24 hours) there is sepsis with ileus and bacterial peritonitis. At this stage, it usually develops to multi-organ damage. Satisfactory treatment results are only possible at an early stage $(0-12 \text{ hours})^8$. Our case was third stage, because of necrosis developed in all intestinal segments and increased lactateand blood urea nitrogen values.

Currently, biphasic contrast enhanced abdominal CT is the primary imaging modality in many medical centers. This technique provides high sensitivity imaging of the whole abdominal region in arterial and portal venous areas. Abdominal CT,fast and noninvasive technique, is available in most centers for 24 hours. Abdominal CT is an appropriate method for the diagnosis of acute mesenteric ischemia due to its sensitivity, specificity, availability and non-invasive⁹.

The presence of pneumatosis intestinalis and HPVG is a strong indicator of mesenteric infarction or ischemia, therefore urgent exploratory surgery is required³. Current data suggest that the incidence of intestinal ischemia associated with pneumatosis intestinalis and portal venous gas is approximately 70%¹⁰.

Patients with pneumatosis intestinalis and HPVG are more likely to show transmural infarction³. Kernagis et al. found that HPVG patients are more likely to show transmural infarction than pneumatosis intestinalis patients alone¹¹.

The presence of gas in the hepatic portal vein may also be helpful in deciding earlier surgical treatment¹. Detection of HPVG in our case helped in the planning of diagnosis and surgical treatment, but post-op loss was lost because of high risk factors.

Mortality in the presence of clinical and radiological signs of intestinal necrosis is 75-85% and in the presence of HPVG on plain x-ray is 75%. Emergency surgical intervention is indicated in this patient group. Mortality is 20-30% in patients with gastrointestinal distention, ulceration or abscess without peritonitis. Conservative treatments are recommended in this patient group who have low mortality. Conservative treatments combine antibiotic therapy, close observation, intravenous fluid, and naso-gastric decompression when required¹².

As a result, it is recommended to prevent the mortality rate with a rapid intervention in such rare cases with high mortality.

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