Portal Venous Gas Resulting from Enteritis in a Case with Portal Hypertension: Case Report

Portal Hipertansiyonlu Olguda Enterite Bağlı Portal Venöz Gaz

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ABSTRACT Hepatic portal venous gas is an uncommon radiographic finding. The mechanism for the appearance of gas in the portal vein is unknown. The most important predisposing factor is the damage to the intestinal mucosa combined with bowel distension or bacterial gas production. Our case presented with abdominal pain, fever, diarrhea and abdominal distension. We diagnosed portal vein thrombosis in computed tomography and saw gas image in the portal vein. It is important to know the underlying pathology for decision making on portal vein gas occurrence. If there is no intestinal ischemic injury requiring surgery, conservative follow up may be the therapeutic option.

Key Words: Hypertension, portal; portal vein

ÖZET Hepatik portal venöz gaz sık olmayan bir radyolojik bulgudur. Portal vende gaz görülmesinin mekanizması bilinmemektedir. Altta yatan en önemli neden barsak distansiyonu veya bakteriyel gaz üretimi ile beraber olan intestinal mukoza hasarıdır. Hastamız bize karın ağrısı, ateş, diyare ve distansiyon nedeniyle müracaat etti. Biz bilgisayarlı tomografide portal ven trombozunu ve ayrıca portal vende gaz görüntüsünü teşhis ettik. Portal ven gazı olan hastalarda tedavi kararı vermek için, altta yatan patolojiyi bilmek önemlidir. Eğer cerrahi müdahale gerektirecek intestinal iskemik hasar yoksa konservatif takip tedavi seçeneği olabilir.

Anahtar Kelimeler: Hipertansiyon, portal; portal ven

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Hepatic portal venous gas (HPVG) is an uncommon radiographic finding.[1] In 1955, Wolfe and Evans first described hepatic portal venous gas in neonates who died secondary to ischemic bowel disease.[2] The mechanism for the appearance of gas in the portal vein is unknown.[3] HPVG usually indicates an ominous prognosis, and it has a mortality rate of 75-85%.[4] The most important predisposing reason for development of HPVG is damage which is done to the intestinal mucosa due to bacterial gas production combined with bowel distension.[5] We report a patient with portal hypertension who was found to have HPVG on computerized tomography (CT).

CASE REPORT

A 17-year-old boy was admitted to hospital with fever, abdominal pain, diarrhea, weight loss and abdominal distension. His body temperature was

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38.4°C. On abdominal examination, there were abdominal distention, diffuse tenderness, increased bowel sounds and moderate degree of ascites. The liver was not palpable. The spleen was palpable 2 cm below the costal margin. Leukocyte count was 18,400/mm^3. C-reactive protein was 21.2 mg/dL (0-0.3 mg/dL). Other significant biochemical variables were as follows: Albumin: 2.6 g/dL, alkaline phosphatase: 892 U/L and gama-glutamyl transpeptidase: 119 U/L. Alanine aminotransferase, aspartate aminotransferase and total bilirubin levels were normal. Hepatitis markers were negative. Detailed laboratory tests are listed in Table 1. Doppler ultrasound revealed suspicious incomplete portal vein thrombosis and ascites. To explain portal vein thrombosis, multiplanar reconstruction of abdominal computed tomography scan (MDCT) was done. MDCT confirmed the presence of thrombus and gas in the portal vein (Figure 1). No CT evidence of mesenteric ischemia was identified. All pro-thrombotic workup including protein C, S, antithrombin, lupus anticoagulants, antiphospholipid antibodies were studied and only protein C activity deficiency was found. The activity level of protein C was 35% (75-150%). *Escherichia coli* species were identified in blood culture specimens as the etiology of fever. *Campylobacter jejuni* was also identified from the fecal cultures. Intravenous ceftriaxone 1 g was administered twice a day in order to treat *E. coli* and *C. jejuni* infections. The fever resolved and leukocyte count normalized on the fifth day. On endoscopy, grade II esophageal varices were observed. Abdominal paracenthesis was performed, and the serum-ascites albumin gradient was reported as 1.4 g/dL. Therefore, the patient was assumed to have portal hypertension. Analysis of the ascitic fluid revealed negative microbiology (total white blood cells of 18/mm^3) and cytology investigations. A beta blocker (propranolol) and a diuretic (aldactone) were started at a dose of 80 mg/day and 100 mg/day, respectively. Oral anticoagulant treatment was started for portal vein thrombosis, and patient was discharged to come back to hospital 3 months later.

TABLE 1: Laboratory values on admission.

Variable	Value
Hematocrit (%)	41.2
White blood cells (per mm/3)	18.400
Differential count (%)	
Neutrophils	75.3
Lymphocytes	18.4
Monocytes	5.7
Eosinophils	0.3
Basophils	0.3
Platelet count (per mm/3)	213 000
Glucose (mg/dL)	102
Aspartate transaminase (IU/L)	18
Alanine aminotransferase (IU/L)	24
Alkaline phosphatase (IU/L)	892
γ-glutamyl transpeptidase (IU/L)	119
Albumin (g/dL)	2.6
Globulin (g/dL)	3.4
Prothrombin time (seconds)	15.3
Hepatitis B and C	Negative
Autoimmune liver panel	Negative

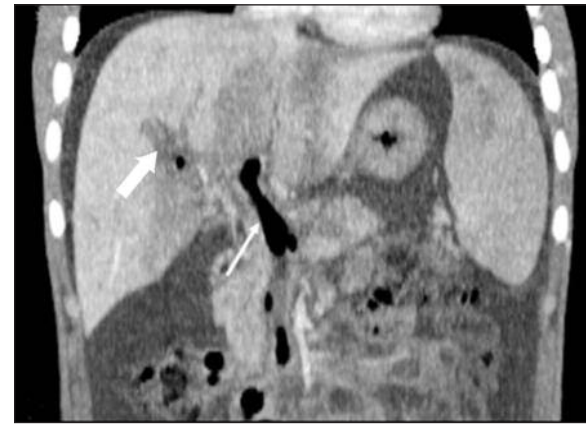

FIGURE 1: Multislice abdominal computed tomography scan showing portal venous gas (thin arrow) and portal thrombus (thick arrow).

DISCUSSION

Hepatic portal venous gas is an ominous finding with a potentially fatal outcome that warrants immediate emergency surgery.[6] The occurrence of gas in the portal venous system was first noted by Wolfe and Evans in 1955. HPVG has been stated in the literature as pneumoportogram, gas embolization of the portal vein or portal vein gas. Liebman et al. have first used the term hepatic por-

tal venous gas to describe this entity.[7] There are two hypotheses for gas accumulating in the portal venous system: 1) Microorganism-derived gas production, and 2) Absorbed intraluminal air (by an impaired epithelial barrier or increased intraluminal pressure).[8] However, in some cases, the pathogenesis for the appearance could not be well defined.[9] Today, increased utilization of advanced radiographic methods in emergency situations has led to increased detection of HPVG associated with various benign non-ischemic conditions that do not necessarily require surgery.[5]

HPVG has been reported in many non fatal conditions, such as bowel necrosis (72%), ulcerative colitis (8%), intra-abdominal abscesses (6%), small bowel obstruction (3%) and gastric ulcers (3%) since it was first described. Intra-abdominal infections associated with HPVG include diverticulitis, abdominal abscesses, cholecystitis, cholangitis, appendicitis, and colitis.[10,11]

Liebman et al. reviewed 64 cases of HPVG and reported a mortality rate of 75%.[7] In a recent survey of HPVG literature, Kinoshita et al. reviewed the clinical data from 182 cases of this entity in adults from the literature.[12] Patient management included conservative therapy in 79 (43%). The overall mortality rate was 39%. This is obviously a significant reduction from the 75% mortality rate seen in 1978. The reduction in mortality was driven by a decrease in the proportion of HPVG associated with mesenteric ischemia. The clinical features and prognosis of HPVG patients varies depending on the underlying disease. The treatment should be directed to the underlying disease. Kinoshita et al. noted an infectious etiology in the absence of other bowel diseases in 26 of 182 patients.[12] These data indicate that an infectious origin for HPVG may occur through a mechanism which is quite independent from the one in ischemic bowel diseases.

The gas itself requires no treatment, but the etiology of HPVG determines the choice for either conservative or surgical treatment. The use of modern CT has resulted in detecting HPVG more frequently in benign conditions. Together with improved surgical techniques, recent studies have reported a decreased mortality rate of 39%. The prognosis is related to the underlying pathology and not to the HPVG itself.[13,14]

The probable pathogenesis contributing to the presence of HPVG in our patient was severe enteritis. It was treated with high dose antibiotics and abdominal pain, fever and portal hypertension were resolved. The portal venous gas disappeared in our patient and it showed a relatively benign course. It is therefore important to identify the underlying diseases and administer the appropriate treatment.

In conclusion, HPVG is an important radiological finding associated with many pathological processes. HPVG itself is merely a clinical finding and not a prognostic factor. HPVG is not a specific disease entity but rather a diagnostic clue in patients with abdominal diseases. The approach to the patient with HPVG should focus on the underlying disease.

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