A rare case of transient portal venous gas

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A B S T R A C T
Hepatic portal venous gas, while a rare finding with a classically poor prognosis, is not always fatal. Mortality varies depending on the underlying etiology; bowel ischemia carries the highest mortality rate. Other etiologies include gastrointestinal obstruction, gastric ulcer, infectious processes (intraperitoneal abscess and gastroenteritis), inflammatory processes (ulcerative colitis, Crohn disease, chemotherapy-induced), and complications from endoscopic procedures. We report a case of a 68-year-old woman who presented with a week-long history of diminished intake, nausea, and vomiting, with unremarkable abdominal examination, who was found to have significant portal venous gas that completely resolved within 16 hours without surgical intervention.

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Case report

A 68-year-old woman with history of stage IIIA ER+/PR+ breast cancer with suspected brain metastasis status after chemotherapy with paclitaxel, doxorubicin, and cyclophosphamide, ischemic stroke with residual left-sided weakness, and atrial fibrillation presented with a week-long history of diminished oral intake, nausea, vomiting, near-syncpe, and altered mentation. She denied fever, abdominal pain, or diarrhea. On admission she was afebrile, hypotensive, and tachycardic. Cardiopulmonary and abdominal examination were unremarkable. Laboratory diagnostics demonstrated leukocytosis (24.5 k/μL), mild microcytic anemia (hemoglobin 9.4 g/dL, mean corpuscular volume 79.5 FL), mild thrombocytopenia (119 k/μL), elevated BUN (26 mg/dL), creatinine (1.03 mg/dL, baseline 0.46 mg/dL), lactic acid (3.4 mmol/L), AST (53 units/L), ALT (67 units/L), and alkaline phosphatase (133 units/L), low sodium (134 mmol/L), potassium (3.3 mmol/L), albumin 2.2 gm/dL, and total protein (6.1 g/dL), and normal total bilirubin (0.6 mg/dL) and bicarbonate (27 mmol/L). Computed tomography (CT) scan of the abdomen and pelvis without contrast revealed extensive hepatic peripheral branching radiolucency suggestive of portal venous gas with a small amount of air in the gastric wall, and air within the small and large bowel loops without abnormal bowel loops or pneumatosis intestinalis (Figs. 1 and 2). CT 2 weeks prior was normal except for a solitary gallstone without cholecystitis. Brain imaging excluded intracranial space occupying lesions. Patient was managed conservatively.

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with intravenous fluids, antiemetics, and nil per os. Repeat CT with IV contrast 16 hours later demonstrated mildly dilated bowel loops, nonspecific colonic wall thickening without inflammation, and complete resolution of hepatic portal venous gas (HPVG) and air in the gastric wall (Fig. 3). She remained clinically stable, tolerating oral intake, and was discharged.

**Discussion**

HPVG is an ominous radiologic finding that can be associated with a spectrum of disease processes ranging from bowel necrosis to gastroenteritis to ileus. While clinically rare, gas within the liver has 2 main differential diagnoses – HPVG and pneumobilia. While HPVG generally manifests as branching peripheral radiolucency extending to within 2 cm of the liver capsule, pneumobilia is more central in location. The nature of the distribution is in part due to the hemodynamics of portal venous flow and biliary flow, with centrifugal flow of portal venous blood to the periphery and centripetal flow of bile, with resultant peripheral or central distribution of accompanying air, respectively [1,2].

While the pathophysiology of HPVG remains unclear, hypotheses have focused on an escape of gas from increased bowel pressure (eg, Valsalva), gas formation in a hepatic abscess with escape to the portal circulation, or the presence of gas-forming bacteria within the portal venous system [2,3]. In this case report, our patient demonstrated the presence of transient HPVG with rapid resolution, suggesting either rapid resorption or bulk removal without persistent gas production. Unfortunately, the CT was done without intravenous or oral contrast, preferably neutral oral contrast (eg, water), to confidently exclude bowel ischemia, which remained on the differential. The lack of abdominal pain, only mild lactic acidosis and acute kidney injury, rapid resolution with crystalloid administration, and the lack of significant calcification at the origin of the mesenteric vasculature decreased the likelihood of this diagnosis. In contrast, the antecedent history of nausea and vomiting coupled with the presence of a small amount of air within the gastric wall and the ability for this air to dissect through tissue planes and enter the portal system, a feature noted in cases of recurrent emesis, suggested that increased intraluminal pressure remained the underlying etiology [2-4]. Indeed, CT scan with contrast, performed 16 hours later, confirmed the absence of bowel ischemia, noting only bowel dilation and nonspecific bowel wall thickening without associated inflammation.

In closure, the treatment of HPVG should be directed toward the underlying disease, after excluding a more fatal cause, such as bowel ischemia, requiring surgical evaluation. In the absence of alarming signs and symptoms, HPVG can be managed conservatively with the potential for complete resolution. Patients should be closely monitored for worsening clinical features and/or radiologic findings.
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