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Extensive portal venous gas: Unlikely etiology and outcome

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ABSTRACT

Portal venous gas or hepatic portal venous gas (HPVG) found on imaging portends grave outcomes for patients suffering from ischemic bowel disease or mesenteric ischemia. HPVG is more rarely seen with severe but treatable abdominal infection as well as multiple benign conditions, and therefore must be aggressively evaluated. We report a 70-year old female who developed extensive intra- and extra-hepatic portal venous gas, pneumatosis intestinalis and free air associated with a perforation of the jejunojejunostomy after a gastrectomy for gastric carcinoma.

1. Introduction

HPVG was first described in infants with necrotizing enterocolitis (NEC) by Wolfe and Evans in 1955,[1] and was then reported in increasing frequency in adults as well.[2] HPVG has been most commonly associated with an acute gastrointestinal catastrophe, such as mesenteric ischemia, with up to 80% of cases resulting in death.[3] Cases in which HPVG is found with mesenteric ischemia often present with abdominal distention, pain, fever, and possibly sepsis, however the extent of the 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] Initially noted in abdominal x-ray films as branching radiolucency within 2 cm of the hepatic edge, other modalities such as ultrasound, Doppler imaging, and computed tomography (CT) detect HPVG with far more sensitivity.[5,6] With increased use of advanced diagnostic imaging, HPVG has also been an incidental finding in many benign diseases, and therefore cannot on its own be used as a prognostic indicator.[7]

2. Presentation of Case

A 70-year old female patient underwent an elective gastrectomy and feeding jejunostomy for stage IIA gastric cancer, after biopsies of a 3 cm gastric ulcer showed moderately differentiated adenocarcinoma. The patient had initially presented with a pulmonary embolism, Factor V Leiden and subsequently developed an upper gastrointestinal hemorrhage for which she had an upper endoscopy revealing a uT3N0 gastric tumor (Fig. 1). She underwent neoadjuvant chemotherapy with epirubicin, oxaliplatin, and fluorouracil (EOF).

The initial procedure consisted of total gastrectomy with D2 lymph node dissection, Roux-en-Y esophagojejunostomy and placement of a feeding jejunostomy. The patient developed ARDS postoperatively and was treated with high dose steroids.

On post-operative day 14, the patient became hypotensive with new acute abdominal pain and peritonitis. CT scan showed extensive intra- and extra-hepatic portal venous gas, with an air-fluid level in the main portal vein, as well as small bowel pneumatosis, with free intraperitoneal air (Figs. 1 and 2. She was taken back to the operating room emergently for exploration.

Intraoperatively, two small perforations were found at the distal jejunojejunostomy with no ischemia or necrosis of any of the bowel was found. Red rubber catheters were placed in the two defects in the edematous anastomosis, proximally and distally along with intra-abdominal drains. She underwent a second look procedure after 48 h, and the abdomen was closed.

Post-operatively the patient was stabilized and was eventually discharged in stable condition to a rehabilitation facility. Now, ten months from her original procedure, the patient is living at home with a performance status of 1 and is currently on chemotherapy as she unfortunately developed a liver metastasis.

3. Discussion

Hepatic portal venous gas with the finding of pneumatosis intestinalis is most frequently associated with ischemic bowel, with

Abbreviations: HPVG, hepatic portal venous gas; NEC, necrotizing enterocolitis; CT, computed tomography; EOF, epirubacin, oxaliplatin, and fluorouracil; IBD, inflammatory bowel disease; COPD, chronic obstructive pulmonary disease; PUD, peptic ulcer disease; SBO, small bowel obstruction; LDH, lactate dehydrogenase.

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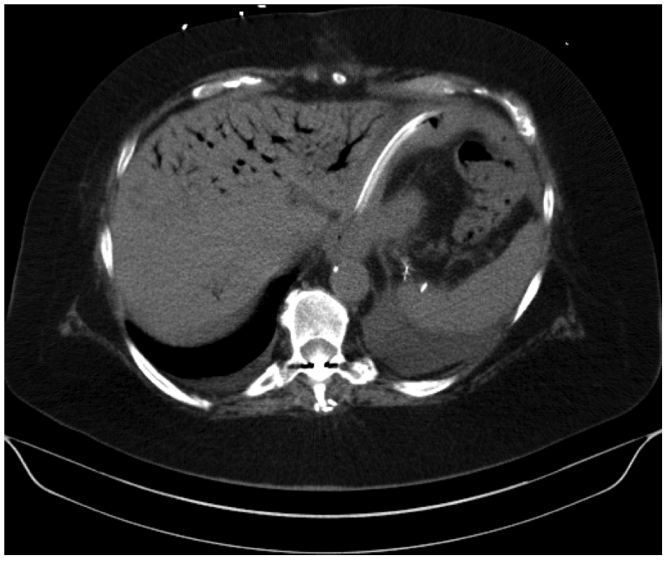

Fig. 1. Axial view of abdominal/pelvic CT scan showing extensive hepatic portal venous gas and pneumatosis intestinalis.

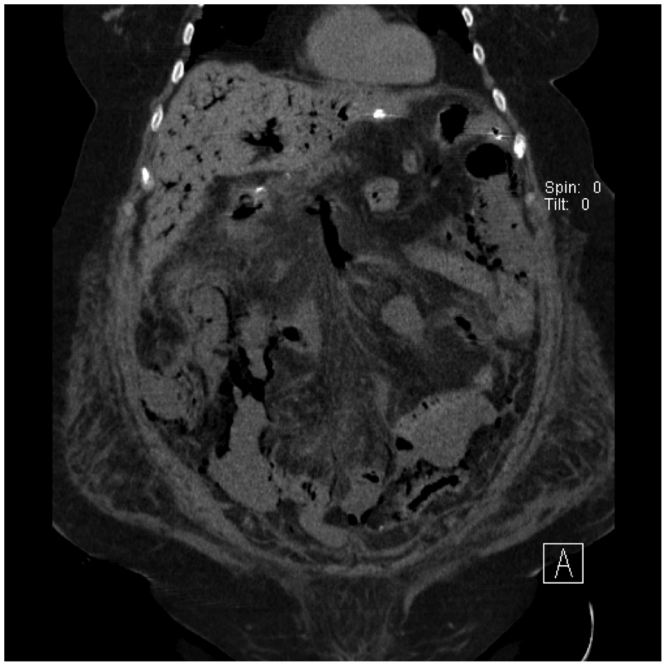

Fig. 2. Coronal view of abdominal/pelvic CT scan showing extensive gas in the superior mesenteric vein.

a reported mortality of 39–80% and up to 100% without operative intervention.[3,5,8–10] First described in 1955 associated in infants with necrotizing enterocolitis,[1] HPVG has since been described in numerous other clinical scenarios. Some less morbid causes of portal venous gas include ileus,[11] diverticulitis,[12,13] gastric distention,[14,15] inflammatory bowel disease (IBD),[16] hypotension post dialysis treatment,[17] decompression sickness,[18] trauma[19] and iatrogenic causes from instrumentation and recent surgery either with or without complications related the procedure.[8,20] Some benign causes of pneumatosis intestinalis, but not directly shown to cause HPVG, include high-pressure ventilation, chronic obstructive pulmonary disorder (COPD), and high dose steroids as well as numerous other conditions both organic and iatrogenic.[21] The clinical presentation in these cases is dependent on the etiology of the disease.[8,22]

Clinical scenarios leading to HPVG generally fall into three categories: bowel distention/obstruction, ischemia, and idiopathic.[8] These mechanisms are coupled with the two main theories proposed for the pathophysiologic etiology of HPVG: mechanical versus bacterial. First, 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.[8,23] Likely both mechanisms play a role in the development and propagation of HPVG.[24]

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 a small population sizes used for these studies, all placed similar in emphasis on the clinical status of the patient, rather than the CT findings alone, including physical exam findings, vital signs, and laboratory values.[8,23,25,26]

These algorithms will ideally help distinguish between patients with potentially benign disease, versus patients with necrotic bowel who may benefit from emergent surgery. Based on findings from a retrospective review of 26 patients from their institution, Iannitti et al.[27] suggest that surgery should be recommended with certain presentations (frank peritonitis), additional CT findings (complete small bowel obstruction—SBO, mesenteric ischemia), certain recent interventions (e.g. vascular surgery procedures), or with complicated medical diagnosis (complicated infectious or inflammatory process). Koami, et al., found that in their sample of 33 patients with HPVG, using a criteria of lower blood pressure (<systolic BP 108 mm Hg), higher lactate dehydrogenase (LDH) (>387 U/L), and the presence of pneumatosis intestinalis led to 100% sensitivity and 78.9% specificity for necrotic bowel.[26] Another retrospective review of 150 patients by Duron et al.[28] showed that abdominal distention (or CT finding of small bowel distention), peritonitis, and lactic academia were the only statistically significant predictors of positive operative findings. In one of the largest retrospective reviews, Bani Hani et al.[29] pooled data from four tertiary centers and evaluated 209 patient charts who had HPVG in order to better predict which patients require operative intervention. After assessing a range clinical findings, laboratory values, radiologic findings and comorbidities, they found that older age, peritoneal signs, and elevated BUN are most highly associated with ischemia and necrotic bowel.

One of the most detailed algorithms developed by Wayne et al. used a retrospective series of 88 patients. The algorithm incorporates many of the previously mentioned clinical findings, including abdominal exam, lactate (>3 mg/dL), and radiological findings of pneumotosis intestinalis, as well as, recent instrumention, and significant past medical history suggestive of vascular risk. Using these factors, the group tested the algorithm prospectively on 14 patients, ideally reducing the risk of nontherapeutic laparotomies, but with four patients not explored described as 'futile'. The decision-making process for futility was not discussed.[8]

4. Conclusion

Our patient had a variety of the potential causes of HPVG, including recent surgery, intestinal distention, ischemia related to hypotension and inotropes. The complicated postoperative course, along with the clinical picture and the finding of extensive HPVG and pneumatosis intestinalis led to frank discussions with the

family regarding the grave nature of this finding and the high risk of surgical intervention. Nonetheless, the operative findings and outcome of this case illustrate the inability to predict the extent of bowel compromise based on HPVG, suggesting that an aggressive surgical approach is still appropriate, even when the prognosis and radiographic findings appears dismal.

Ethical statement

Written informed consent was obtained from the patient for publication of this Case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

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