



Diagnostic yield of portal venous gas and pneumatosis intestinalis: Does it always mandate a trip to the OR?

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Etiology

There are namely 4 theories for the formation of PI & HPV: intestinal wall inflammation (from ischemia, inflammatory bowel disease, infectious enteritis etc.) leading to increased mucosal permeability allowing the passage of gas into the mesenteric veins and from there to the portal vein [1], bowel distension also altering the intestinal mucosal barrier and effectively pushing gas into intestinal wall capillaries [1]. gas forming bacteria leading to pylophlebitis, and finally, idiopathic.

Reported causes of PI & HPV include but are not limited to: occlusive and non-occlusive mesenteric ischemia, peptic ulcers, inflammatory bowel disease, bowel obstruction, ileus, toxic mega colon, diverticulitis, intra-abdominal abscess, infectious enteritis, endoscopy, barium enemas, feeding tubes, post-surgi-

Abstract

Hepatic portal venous gas (HPVG) and pneumatosis intestinalis (PI) are worrisome signs when seen on imaging as they are associated with intestinal ischemia. However, they have multiple etiologies, some benign and some not. It is important for the clinician to be able to differentiate which patient requires operative intervention and which does not. This article will aim to review the available literature regarding the HPV & PI with the purpose of aiding clinicians to differentiate pathological from benign HPV & PI.

cal anastomosis, organ transplantation, steroids, chemotherapeutic agents, colchicine toxicity, lactulose, connective tissue disorders, AIDS, asthma, COPD, cystic fibrosis and trauma [2,7-13].

Imaging Findings (Figures 1-6)

With regards to HPV, it is important to differentiate it from pneumobilia. HPV as seen on either abdominal radiography or CT is described as branching hypodensities that extend to within 2 cm of the liver capsule in contrast to pneumobilia which is seen more centrally [3,10]. This difference, is due to the centrifugal flow of the portal veins as compared to the centripetal flow of the bile ducts that drain from the periphery to the common hepatic duct [3,10]. Consistently, large bowel PI has been found to be more common than small bowel PI; however, small bowel PI is more likely to be due to transmural ischemia [2-6]. The



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presence of both PI and gas in the mesenteric veins or portal venous system (portomesenteric gas) vs. only PI has also been shown to be more likely due to transmural ischemia and thus have worse prognosis [2,6-9]. Ascites on imaging in the setting of HPVG and/or PI is associated with bowel ischemia [4,14].

While PI and HPVG are overwhelmingly identified on computed tomography (CT), they can also be seen on abdominal radiography, sonography, and MRI. More extensive PI & HPVG is needed for it to become apparent on abdominal radiography than what can be detected on CT, which correlates with a more severe disease. Indeed, in Liebman's 1978 review of 64 cases of HPVG that were seen on abdominal X-ray (CT scanning had not become widely available until about 1980), he noted a 75% mortality rate that goes up to 81% after excluding iatrogenic cases (barium enemas in patients with ulcerative colitis) [10]. More recent studies have shown overall mortality rates ranging between 22 and 42% reflecting the detection of less severe cases of PI & HPVG with CT-scans [4-6]. HPVG is more easily seen on X-rays taken in the left lateral decubitus position [10].

On ultrasonography, HPVG is seen as flowing echogenic bubbles in the portal vein, and PI as a linear or focal area of echogenicity within the bowel wall [7,11]. On MRI, air can be seen in the bowel wall more easily on gradient echo images [7].

Clinical Presentation

It is imperative in patients with PI on imaging to rule out abdominal emergencies such as acute mesenteric ischemia. PI and/or HPVG is a common and specific finding in all forms of acute mesenteric ischemia (thromboembolic, atherosclerotic, non-obstructive mesenteric ischemia, and mesenteric venous thrombosis) and are associated with a substantial increase in the in-hospital mortality for these patients. Therefore, in suspected patients with acute mesenteric ischemia, it is always advantageous to initially order a contrast-enhanced CT scan, with an arterial and venous phase, to evaluate the mesenteric vasculature concurrently [15].

The Pneumatosis Intestinalis Predictive Evaluation Study (PIPES), a multicenter retrospective study between 2001 and 2010 by The Eastern Association for the Surgery of Trauma, identified 500 patients with PI. Sixty percent (299) of the patients had benign disease and 40% (201) had pathologic PI. Pathologic PI was defined as full thickness bowel ischemia detected on endoscopy/surgery or withdrawal of life sustaining measures leading to subsequent death. The most common symptoms and signs on exam were abdominal distention (54%), constipation (19%) and peritonitis (17%) [2]. Twenty-one percent of patients could not be evaluated due to altered mental status or sedation. Abdominal distention, peritonitis, absent bowel sounds, and abdominal rigidity were significantly associated with pathologic PI [2]. In addition, lactate greater than 2 mmol/L, peritonitis, hypotension or vasopressor use, acute kidney injury, mechanical ventilation, and absent bowel sounds were found to be independent predictors of pathologic PI [2]. A classification and regression tree analysis looking three variables (lactate >2 mmol/L, hypotension/vasopressor use, and peritonitis) found that the presence of lactate >2 mmol/L and hypotension/vasopressor use had a predictive probability of 93% for pathologic PI while the absence of all three variables had a diagnostic accuracy of 22% for pathologic PI [2].

The same group designed a multicenter prospective observational study to confirm the findings of the latter study and

published it in March of 2017 [4]. During the 3 year study period, 127 patients with PI were identified; 79 benign (62.2%) and 48 pathologic (37.8%) [4]. Those with lactate > 2 mmol/L and/or peritonitis were significantly more likely to have transmural ischemia, similar to their previous study. Patients with anemia (mean hemoglobin of 10.3 g/dL in pathological PI vs 11.9 in benign PI) and higher international normalized ratio (1.4 vs 1.1) on lab work were more likely to have pathologic PI.

A retrospective study that included 87 cases of PI diagnosed via CT scanning between 1993 and 2001 found that serum lactic acid > 2.0 mmol/L was associated with an 80% mortality [5]. Another single-institution retrospective study of 52 PI cases found that the most common signs and symptoms were abdominal pain (59.6%), abdominal distention (50%), nausea/vomiting (32.7%), and peritonitis (30.8%) [14]. In this same study, peritonitis, abdominal pain, elevated lactate, elevated CRP, and elevated creatinine were significantly higher in the patients with bowel necrosis [14].

A retrospective case-control study of 208 PI patients who underwent surgery investigated whether pre-operative CT scans could determine the degree of bowel wall ischemia, partial thickness vs. transmural [16]. They looked at CT findings such as bowel wall thickening, bowel distention, pneumatosis, portomesenteric gas, and presence of thrombi or emboli but found no difference between their cases (121 cases with transmural ischemia) and controls (87 cases with partial bowel necrosis) [16].

A single-institution case-series of 88 PI patients was used to generate a treatment algorithm for PI & HPVG [17]. This study divided PI & HPVG into 3 major subgroups: mechanical causes (adhesion related bowel obstruction, hernia, post-endoscopy, intussusception, and diverticulitis), acute mesenteric ischemia, and benign idiopathic (cancer on chemotherapy, inflammatory bowel disease, chronic obstructive pulmonary disease on steroids, etc.) [17]. A vascular disease score and an algorithm was generated that distinguished the 3 subgroups with a sensitivity of 89%, specificity of 100%, and positive predictive value of 100% [17]. In this study, acute mesenteric ischemia was significantly associated with abdominal pain, elevated lactate (>3 mg/dL), small bowel PI, and elevated vascular disease score [15]. The benign idiopathic group had few vascular risk factors [17].

A multicenter retrospective study looking at 209 patients found 3 variables that were significantly associated with mesenteric ischemia: age \geq 60 years, peritonitis on physical exam, and blood urea nitrogen > 25 mg/dL [18]. Lactate levels were only available for 89 patients and were not used in their analyses. The presence of both PI & HPVG on imaging (vs. only one) was associated with increased odds of having mesenteric ischemia but it was not statistically significant [18]. This study found that CT findings could not reliably distinguish between ischemic and non-ischemic PI & HPVG [18].

To note, although the presence of elevated lactate levels is associated with increased disease burden and adverse events; normal lactate levels, especially early in the course, does not exclude intestinal ischemia [19]. Lactate produced by the ischemic bowels is primarily released into the porto-mesenteric venous system where it effectively metabolized by the liver, as suggested by experimental works. Therefore, plasma lactate levels do not necessarily reflect lactate produced by the ischemic bowels [19]. Plasma lactate has been reported to have high sensitiv-

ity but low specificity in the diagnosis of acute mesenteric ischemia, and lactate levels have been found to be normal in 50% of acute mesenteric ischemia [20,21].

As evident from the above studies, the clinical presentation, imaging findings, and laboratory values of patients with PI and/or HPVG can be rather variable and cannot reliably determine the degree of intestinal ischemia or discern between the patients who can be managed conservatively vs. operatively. The only absolute indication for laparotomy is the presence of peritonitis unless a palliative approach has been elected. Laparotomy remains the most accurate way to assess the severity and extent of intestinal ischemia [19].

Conclusion

As of yet, we do not have a clear-cut way to determine whether a patient with PI and/or HPVG has transmural bowel ischemia mandating operative intervention. This is likely because of the myriad of causes of HPVG and PI. However, there are some imaging characteristics, physical exam findings, and laboratory markers that have been shown consistently in multiple studies to be indicative of transmural bowel ischemia. These include the presence of both PI and portomesenteric gas (vs. only one), the presence of PI in the small bowel (vs. large bowel), peritonitis on physical exam, and lactate > 2 mmol/L.

Figures

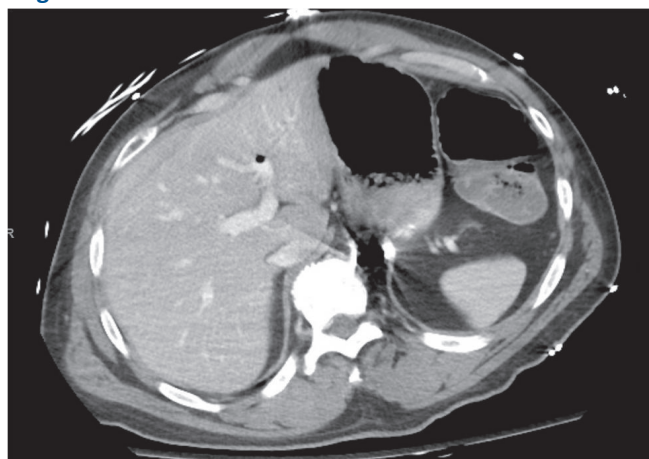


Figure 1: HPVG and Cecalpneumatosis in patient with STEMI and intra-aortic balloon pump

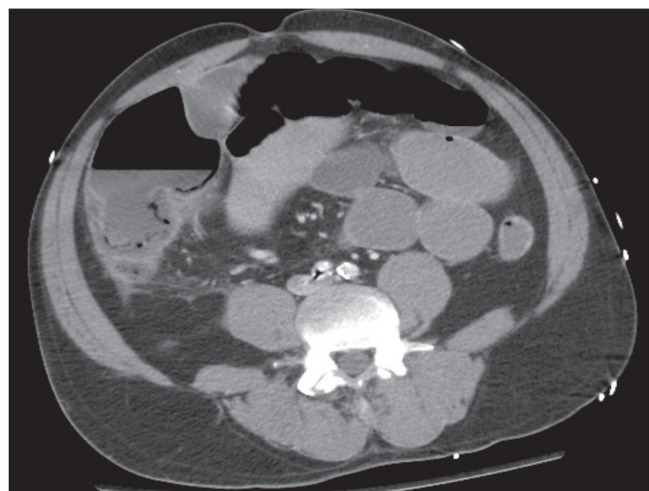


Figure 2: HPVG and Cecalpneumatosis in patient with STEMI and intra-aortic balloon pump



Figure 3: Patient with hx of liver transplant with chronic HPVG for many years

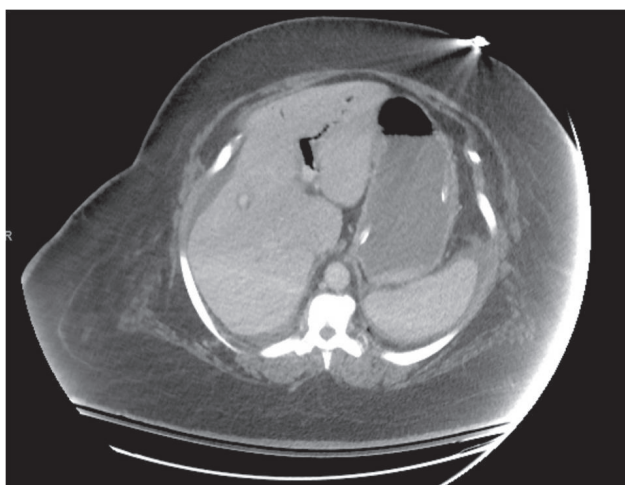


Figure 4: Patient with hypotension due to dialysis and abdominal pain. Found to have HPVG (figure 4), mesenteric venous gas (figure 5), and PI of small bowel (figure 6). Found to have necrotic small bowel on exploratory laparotomy.



Figure 5: Patient with hypotension due to dialysis and abdominal pain. Found to have HPVG (figure 4), mesenteric venous gas (figure 5), and PI of small bowel (figure 6). Found to have necrotic small bowel on exploratory laparotomy.



Figure 6: Patient with hypotension due to dialysis and abdominal pain. Found to have HPVVG (figure 4), mesenteric venous gas (figure 5), and PI of small bowel (figure 6). Found to have necrotic small bowel on exploratory laparotomy.

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