

# Benign Pneumatosis Intestinalis: A Case Report and Review of the Literature

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**Background:** Pneumatosis intestinalis is the presence of gas within the walls of the intestine on imaging and has variable clinical significance, ranging from an incidental finding to a harbinger of intra-abdominal catastrophe. There are multiple mechanisms by which this radiographic finding develops, including bacterial gas production, increased intraluminal pressure within the gastrointestinal tract, mechanical disruption of the gut mucosa, and tracking of pulmonary gas along the mesenteric vasculature.

**Case Presentation:** We present the case of a 61-year-old male veteran who had sustained a remote anoxic brain injury resulting in chronic dependence on a gastrostomy tube for enteral nutrition, found incidentally to have pneumatosis intestinalis without signs of intra-abdominal catastrophe.

**Conclusions:** Appropriate interpretation of this finding can lead to life-saving interventions and avoid unnecessary surgical procedures.

**P**neumatosis intestinalis (PI) is the finding of gas within the walls of the intestine on imaging. It is most commonly detected via radiograph or computed tomography (CT). The diseases leading to the accumulation of gas within the submucosal space of the gastrointestinal (GI) tract are heterogeneous, and the finding of PI itself has a wide range of clinical implications from impending clinical deterioration to an incidental finding of minimal consequence.

We present the case of a veteran who had sustained a remote anoxic brain injury resulting in chronic dependence on a gastrostomy tube for enteral nutrition, found incidentally to have PI without signs of intra-abdominal catastrophe. An exclusion of other, more life-threatening causes of PI led to a diagnosis of benign PI secondary to the presence of his gastrostomy tube. This case highlights the importance of interpreting the finding of PI in the clinical context of the specific patient and how conservative management may be appropriate in some cases.

## CASE PRESENTATION

A 61-year-old male patient was admitted for fever. The patient had a remote history of cardiac arrest complicated by anoxic brain injury requiring tracheostomy, gastrostomy tube, and a suprapubic catheter with recurrent catheter-associated urinary tract infections (CAUTI), secondary seizure disorder, atrial fibrillation off anticoagulation due to recurrent GI bleeding, and treatment naive chronic hepatitis C virus. His ability to provide a clinical history was limited by his

nonverbal status. He had no prior surgical history but had presented a month earlier for a high-grade small bowel obstruction (SBO) with pneumobilia that was managed conservatively as the surgical team deemed him a poor candidate for surgical intervention with his extensive comorbidities. A bioethics consultation at the time supported minimizing potential surgical risk in favor of conservative medical management; this was discussed with the patient's surrogate decision maker, who also wished to avoid surgery. The SBO resolved with conservative management. He had been residing in a nursing home and doing well until 24 hours prior to admission when he developed fevers.

Vital signs on admission showed a temperature of 100.8 °F, heart rate 100 beats per minute, blood pressure 116/85, respiratory rate 22 per minute, and oxygen saturation of 100% on 6 L of oxygen via tracheostomy collar. His initial examination was notable for clear lung sounds, a nondistended nonrigid abdomen with an indwelling percutaneous gastrostomy tube, and absence of areas of skin breakdown or erythema. Notable laboratory studies showed a leukocytosis and urinalysis suggestive of CAUTI (Table). His urinary catheter was exchanged, he was fluid resuscitated and started on empiric vancomycin and piperacillin-tazobactam for management of sepsis due to CAUTI.

For the first 3 days of his hospitalization, he demonstrated clinical improvement on vancomycin and piperacillin-tazobactam while awaiting results from his urine bacterial culture. On hospital day 3, he

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**TABLE** Laboratory Values on Presentation

Laboratory Studies	Results	Reference Ranges
WBC, k/ $\mu$ L	11.6	4.5-11.0
Neutrophils, %	74	41-85
Hemoglobin, g/dL	11.6	13.3-17.7
Platelets, k/ $\mu$ L	463	150-440
Sodium, mmol/L	137	136-146
Potassium, mmol/L	4.7	3.5-5.3
Chlorine, mmol/L	103	95-110
Carbon dioxide, mmol/L	26.4	21-31.0
Blood urea nitrogen, mg/dL	9	5-25
Creatinine, mg/dL	0.55	0.66-1.28
Glucose, mg/dL	115	70-110
Lactate, mmol/L	2.1	0.5-2.2
Urinalysis		
Color	Amber	-
Appearance	Cloudy	-
Specific gravity	1.026	1.003-1.030
pH	6	5.5-8.5
Protein, mg/dL	30	Negative
Glucose	Negative	Negative
Ketones	Negative	Negative
Bilirubin	Negative	Negative
Blood	Small	Negative
Nitrite	Positive	Negative
Urobilinogen	Negative	Negative-1 mg/dL
Leukocyte esterase	Large	Negative
RBC/HPF	14	0
WBC/HPF	> 182	0
WBC clumps	Many	None
Bacteria	Small	None

Abbreviations: HPF, high power field; RBC, red blood cell; WBC, white blood cell.

developed recurrent nonbloody, nonbilious emesis despite no change in the rate or formulation of his enteral nutrition. He also had 3 watery brown bowel movements. His vital signs remained within normal limits. His abdominal examination at this point showed mild distention and was hypertympanic to percussion, but there was no rigidity or involuntary guarding. On hospital day 4, he continued to have emesis with an unchanged abdominal examination. The differential diagnosis included recurrence of prior SBO, ileus, intestinal ischemia, enteral nutrition intolerance, *Clostridioides difficile*

(*C difficile*) colitis, and GI dysmotility because of his anoxic brain injury.

Testing for *C difficile* was negative. An abdominal radiograph was obtained and revealed no bowel obstruction but, alarmingly, showed extensive intramural bowel gas, suggestive of PI (Figure 1). His leukocyte count, serum bicarbonate, and serum lactate levels remained within normal limits. A CT with contrast of the abdomen and pelvis demonstrated no vascular obstruction but confirmed the presence of diffuse intramural gas in his stomach and proximal small bowel, as well as the presence of mesenteric and portal venous gas (Figures 2 and 3). Although his abdominal examination had not changed and did not suggest peritonitis, general surgery was consulted to discuss the need for surgical intervention. Given his overall clinical stability and high surgical risk due to his many comorbidities, surgery recommended a conservative approach.

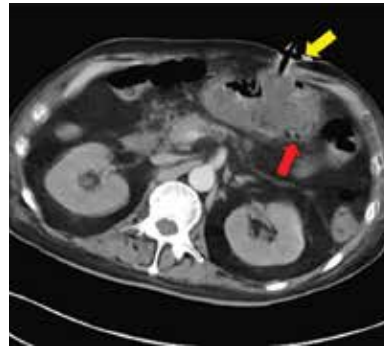
Through the following hospital days, his enteral nutrition was held and serial abdominal examinations were performed without change. Serial laboratory studies, including serum lactate and leukocyte count, remained reassuringly within normal limits. His urine culture eventually revealed multidrug-resistant *Pseudomonas aeruginosa*. Antimicrobial therapy was narrowed to piperacillin-tazobactam for a complete course. Enteral nutrition was gradually reintroduced at a low rate, ultimately reaching goal rate with return of bowel function by hospital day 9. Despite extensive workup, the etiology of his transient enteral nutrition intolerance remained uncertain, though an adverse effect of antibiotic therapy was thought possible. Follow-up abdominal radiographs demonstrated interval improvement of PI. He was discharged back to his skilled nursing facility on hospital day 11 without incident.

## DISCUSSION

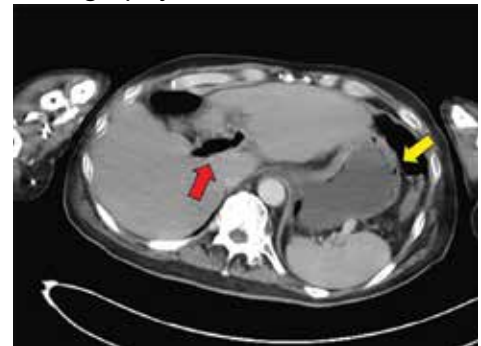
PI is an incompletely understood condition seen in multiple diseases. Patients may present with highly variable symptoms, often more attributable to the underlying disease causing the PI than the presence of PI, as patients may be entirely asymptomatic. When symptoms are attributed to PI, those most reported are abdominal pain, bloody stools, and diarrhea.<sup>1</sup> It is often

**FIGURE 1** Abdominal Radiograph

Yellow arrow indicates presence of extensive gas within walls of small intestine.

**FIGURE 2** Abdominal Computed Tomography

Gastro-jejunoscopy tube (yellow arrow) and presence of gas within the wall of the stomach (red arrow) can be seen.

**FIGURE 3** Abdominal Computed Tomography With Contrast

Presence of pneumatosis intestinalis in the wall of the stomach (yellow arrow) and portal venous gas (red arrow) are visible.

detected on abdominal plain films. Alternative methods of diagnosis include ultrasonography, barium enema, and endoscopy although the last method has been known to occasionally lead to bowel perforation.<sup>2-6</sup> The most sensitive method of detection is CT, which also provides additional information about abdominal pathology and may identify the underlying process responsible for the PI.<sup>7</sup>

While not fully understood, much information about PI and its pathogenesis is known. Understanding the mechanisms of PI is vital to direct the clinician's evaluation of the patient for reversible conditions that may cause PI. Early descriptions of PI in the literature documented an association with pyloric stenosis, leading to the theory that gas from the intestinal lumen is driven into the submucosal space during episodes of forceful vomiting with increased intraluminal pressure.<sup>8</sup> As PI was subsequently described in multiple other disease states not typically associated with increased intraluminal pressure such as inflammatory bowel disease, GI malignancy, cryptosporidiosis and CMV infection, additional theories about the pathogenesis of PI have arisen.<sup>9-24</sup> There is now experimental data to support multiple mechanisms of intramural gas accumulation. It has become accepted that PI represents a common pathway shared across various pathologic states and results from multifactorial mechanisms of gas entry into the intestinal wall.<sup>25-29</sup>

Factors leading to the development of PI include bacterial production of gas, intraluminal GI gas compositions, increased intra-

luminal pressure, pulmonary gas tracking through vessels communicating with the thorax, and mucosal disruption. PI has been linked to bacterial infections of the GI tract in humans including *C difficile*, *Klebsiella*, and Whipple disease.<sup>14-18</sup> In animal models, *C difficile* within the walls of rat intestine results in the appearance of pneumocysts, or discrete collections of submucosal gas, which are the hallmark feature of PI.<sup>30</sup> It is thought that direct invasion of bacteria into intramural spaces can cause PI in humans, although bacteria have yet to be directly isolated from the pneumocysts. Translocation of luminal gas into pneumocysts found in PI is theorized to be driven by differences in partial pressures.<sup>31</sup> The concentration of hydrogen within the intestinal lumen is high due to bacterial production. Hydrogen, diffusing along its partial pressure gradient between the lumen and blood, accumulates within the intestinal wall and causes the formation of pneumocysts. This phenomenon has been hypothesized to explain the tendency for pneumocysts to form around the mesenteric vasculature.

Gas from the lumen can also be forced into the intestinal wall during an abrupt increase in intra-abdominal pressure, such as that seen with forceful vomiting. The final possible origin of the gas is the lungs, as PI has been associated with lung disease. It was previously thought that gas from ruptured alveoli tracks along mediastinal vessels, below the diaphragm, and into the mesentery.<sup>30</sup> Newer theories argue that increased intra-abdominal pressure, typical of patients with obstructive lung disease and frequent coughing, is

the driver of PI by the mechanism previously described.<sup>32-34</sup> Additionally, mucosal disruption leads to increased permeability and allows accumulation of gas within the intestinal walls. Mucosal abnormalities have been described in histopathologic studies of patients with PI and associated with conditions known to compromise mucosal integrity, such as immunodeficiencies, inflammatory bowel disease, and the receipt of cytotoxic chemotherapy.<sup>10,12,19-23</sup>

Our patient likely had mucosal disruption due to his gastrostomy tube as well as increased intraluminal pressure from recurrent vomiting, contributing to translocation of otherwise normal intraluminal gas. The presence of portal venous gas, as seen in this case, has historically portended a worse prognosis, with 37% mortality in one series.<sup>7,35,36</sup> However, portal venous gas as well as pneumoperitoneum occur in benign etiologies of PI as well. It is thought that this occurs due to rupture of the submucosal pneumocysts through the wall opposite the intestinal lumen and thus does not result in a direct communication between the intestinal lumen and the peritoneal cavity.<sup>12</sup>

PI is not a diagnosis but a manifestation of an underlying disease. As such, the treatment of PI is targeted toward the underlying condition. Of note, the pattern and extent of PI seen on imaging has not been shown to correlate with the severity of the underlying pathologic process.<sup>35,37</sup> Instead, assessment of the patient and their clinical trajectory should determine the appropriate treatment. The decision facing the clinician when PI is discovered is whether urgent surgery is indicated, as is the case in mesenteric ischemia, bowel necrosis, or intestinal perforation, conditions known to be associated with PI. Otherwise, there is no definitive treatment for PI. Bowel rest is almost universally pursued. There are reports of treating with supranormal levels of supplemental oxygen, maintaining arterial partial pressure of oxygen above 300 mm Hg, with a face mask and 8 L/min flow rate.<sup>38,39</sup> The proposed mechanisms of benefit include establishing a favorable diffusion gradient for intramural gas to exit the pneumocysts as well as creating an inhospitable, aerobic environment for hydrogen-producing anaerobic enteric bacteria. A prudent approach for most cases of PI is conservative management with bowel rest and

supplemental oxygen unless there is a definitive indication for urgent surgical intervention, such as peritonitis, abdominal sepsis, or perforation.<sup>40,41</sup> Management recommendations suggest that up to 50% of cases can be successfully managed nonoperatively.<sup>42</sup>

## CONCLUSIONS

PI is the radiographic finding of gas within the walls of the intestinal tract and has variable clinical significance. It can represent a benign incidental finding or a sequela of intra-abdominal emergencies such as mesenteric ischemia or bowel necrosis. Because PI is seen in a variety of disorders, several proposed mechanisms are supported in the medical literature. These include bacterial production of gas, gas pressure gradients between the intestinal lumen and the blood, increased intraluminal pressure, pulmonary gas tracking from intrathoracic vessels, and mucosal disruption. The evaluation of a patient with PI must begin with an assessment for the need for urgent surgical intervention. Additional management measures include bowel rest, IV hydration, and supplemental oxygen administration. Because of its wide variety of etiologies of varying clinical urgency, placing the finding of PI in the context of the patient is paramount to selecting an appropriate management strategy.

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## Disclaimer

The opinions expressed herein are those of the authors and do not necessarily reflect those of *Federal Practitioner*, Frontline Medical Communications Inc., the US Government, or any of its agencies.

## Ethics and consent

Informed consent was not obtained from the patient or surrogate decision maker. Patient identifiers were removed to protect the patient's identity.

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