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Visual Case Discussion

Cecal pneumatosis intestinalis in necrotic colorectal carcinoma

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ABSTRACT

Pneumatosis Intestinalis (PI) while infrequent, can be fatal in some cases. An abdominal computed tomography (CT) scan is typically performed to aid in the diagnosis and in the rare cases that are lethal, an emergent exploratory laparotomy is necessary. We present a case of a 57-year-old male who was seen at the emergency department (ED) complaining of severe abdominal pain, fever, and shortness of breath with a past medical history significant for metastatic colon cancer. Through lab work and a CT scan, the patient was diagnosed with PI involving the ascending colon, cecum, and terminal ileum. A laparotomy as well as a resection of the terminal ileum and cecum was performed. The patient expired from septic shock the following day. This case demonstrates the need to distinguish between benign and lethal PI in a timely manner.

1. Introduction

Pneumatosis intestinalis (PI) is a rare clinical condition characterized by the presence of gas within the intestinal wall.[1] While the exact incidence of PI is unknown, it is estimated to be 0.03% based on an autopsy series.[1] The mean age of those afflicted is 45.3 years with a male predominance of 2.4 to 1.[2] Most patients are asymptomatic, however, it can co-occur with life-threatening mesenteric ischemia in rare cases. PI can involve the colon (46%), small intestine (27%), or both (7%).[3] Patients with severe intestinal ischemia or bowel obstruction commonly present with abdominal distention and peritoneal signs. Contrast-enhanced abdominal computed tomography (CT) scans are frequently used to establish the diagnosis. Gas in PI often assumes two distinct morphologies on CT image: 1) a cystic or bubbly pattern, commonly associated with benign causes; and 2) a linear pattern, often associated with lethal causes.[4] Life-threatening PI requires emergent exploratory laparotomy.

2. Case report

A 57-year-old male patient presented to the emergency department (ED) with acute onset of severe abdominal pain. His-past medical history was significant for metastatic colon cancer, hypertension, hyperlipidemia, diabetes mellitus, and an umbilical hernia. Ten months prior to presentation, he was diagnosed with stage IV colon cancer located in the cecum with metastases to the liver. He subsequently started FOLFOX chemotherapy regimen (folinic acid, 5-fluorouracil, and oxaliplatin) and bevacizumab. In the ED, the patient reported severe right lower quadrant pain with fever and shortness of breath. His-vitals were: blood pressure of 128/53 mm/Hg, pulse of 130 beats per minute, temperature of 35.7 °C, and respiratory rate of 20 breaths per minute. A physical examination showed that the patient had tachycardia, diffused abdominal tenderness, and was in respiratory distress. Despite this, his bowel sounds were normal and there was no abdominal distention. An arterial blood gas showed significant metabolic acidosis with a pH of 7.05 (reference value 7.35–7.45) and a HCO_3 of 12 mEq/L (reference value 21–26 mEq/L). Blood work revealed leukopenia with a white blood cell count of 1.1 × 10^3/μL (reference value 4.0–10.5 × 10^3/μL), absolute neutrophil count of 0.5 × 10^3/μL (reference value 1.8–7.8 × 10^3/μL), normocytic anemia with a hemoglobin of 10.6 g/dL (reference value 12.5–17.0 g/dL), MCV of 95 fL (reference value 80–100 fL), elevated BUN of 30 mg/dL (reference value 7–28 mg/dL), elevated creatinine of 2.69 mg/dL (reference value 0.5–1.30 mg/dL), severely elevated lactate of 14.9 mmol/L (reference value 0.5–2.1 mmol/L), elevated AST of 97 U/L (reference value <41 U/L) and ALT of 59 U/L

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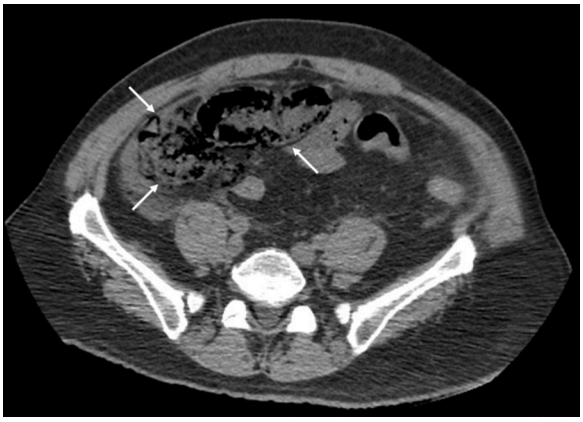

Fig. 1. Computed tomography of the abdomen showing pneumatosis intestinalis (arrows) of the terminal ileum, cecum, and proximal ascending colon.

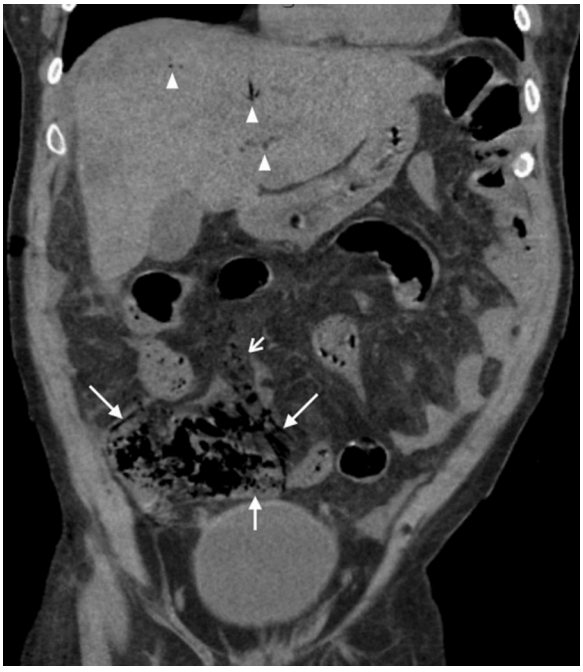

Fig. 2. Computed tomography of the abdomen showing pneumatosis intestinalis (long arrows) of the terminal ileum, cecum, and proximal along with intrahepatic portal venous gas (arrow heads) and mesenteric venous gas (open short arrow).

(reference value <56 U/L). A CT without contrast showed portal venous gas with PI involving the ascending colon, cecum, and terminal ileum, with surrounding intravascular air in addition to the preexisting cecal colon adenocarcinoma (Figs. 1,2,3) The patient underwent an emergent exploratory laparotomy and a resection of the terminal ileum and cecum. In the ICU thereafter, the patient remained in critical condition, requiring three vasopressors for hemodynamic support. His-lactate remained elevated at 8.6 mmol/L (reference value 0.5–2.1 mmol/L). Unfortunately, the patient passed away the next day due to septic shock. Histopathological examinations confirmed the presence of PI in the colon (Fig. 4).

3. Discussion

Two theories have been proposed to explain the pathogenesis of PI: the mechanical theory and the bacterial theory. The mechanical theory postulates that air may invade the intestinal wall through a break in the

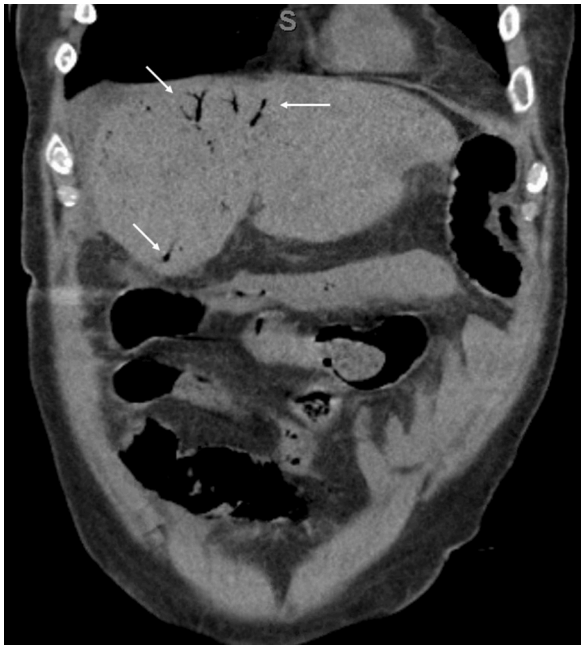

Fig. 3. Computed tomography of the abdomen showing a 57-year-old male with metastatic colon cancer presenting with pneumatosis intestinalis and consequent intrahepatic portal venous gas (arrows).

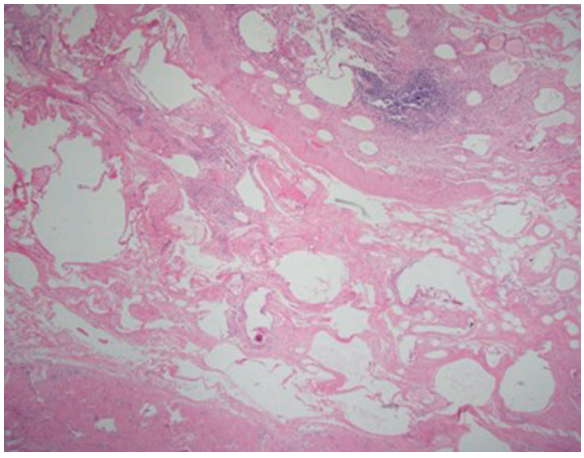

Fig. 4. Necrotic bowel wall showing empty cyst-like spaces involving the entire thickness of the wall including submucosa and muscularis propria (hematoxylin and eosin, 40X magnification).

mucosa or by tracking the mesenteric vessels on the serosa[5] while the bacterial theory states that gas produced by colonic flora via fermentation may enter the intestinal wall through areas of mucosal erosion. Excessive hydrogen gas may be forced into the mucosa and submucosa directly.[6] Our patient already had existing mucosal damage and a bowel obstruction from the colon carcinoma. The tumor may have served as a nidus for bacterial growth, and the excess gas produced may have entered the submucosa through areas of mucosal destruction. Although the patient was on bevacizumab, a target molecular chemotherapy agent known to cause PI before presentation, it is unclear what role the drug played in the development of PI. When PI occurs, it usually self-resolves after discontinuation of the drug.[7] The patient's history of colorectal cancer is the more likely culprit of his fulminant PI.

Distinguishing between benign and life-threatening PI remains a challenge to clinicians. Specific patient characteristics have been identified to be significantly associated with pathological PI, such as age ≥

60, hypotension/need for vasopressors, signs of peritonitis, serum lactate >2.0 mmol/L, creatinine > 2.0 mg/dL, white blood cell count <5 or >11 × 10^3/μL, small bowel pneumatosis, extensive pneumatosis, ascites, and portomesenteric venous gas.[8] Among these factors, elevated lactate is the strongest independent predictor of pathological PI.[8]

4. Conclusion

While benign and life-threatening PI can present in similar ways, there are key differences that emergency physicians should be aware of such as age, lab values, and CT results. Our case of lethal PI has demonstrated many of these characteristics, providing support to using the above model as a decision-making tool in the evaluation of PI.

Credit author statement

Su-Yuan Yu - Writing - Original Draft, Conceptualization
Christopher R Connell - Writing - Review & Editing, Visualization
Joseph J. Stirparo - Writing - Review & Editing, Supervision
Victor Longo III - Writing - Review & Editing, Supervision, Visualization

Author contributions

All authors provided substantial contributions to manuscript content. All authors gave final approval of the version of the article to be published.

Declaration of Competing Interest

None

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.visj.2022.101300.

References

1 Di Pietropaolo M, Trinci M, Giangregorio C, Galluzzo M, Miele V. Pneumatosis cystoides intestinalis: case report and review of literature. *Clin J Gastroenterol.* Feb 2020;13(1):31–36. https://doi.org/10.1007/s12328-019-00999-3. Epub 2019 Jun 3. PMID: 31161540.
2 Wu LL, Yang YS, Dou Y, Liu QS. A systematic analysis of pneumatosis cystoids intestinalis. *World J Gastroenterol.* Aug 14 2013;19(30):4973–4978. https://doi.org/10.3748/wjg.v19.i30.4973. PMID: 23946603; PMCID: PMC3740428.
3 Dibra R, Picciariello A, Trigiante G, et al. Pneumatosis intestinalis and hepatic portal venous gas: watch and wait or emergency surgery? A case report and literature review. *Am J Case Rep.* Jul 12 2020;21, e923831. https://doi.org/10.12659/ajcr.923831. PMID: 32653891; PMCID: PMC7377522.
4 Torres US, Fortes CDFM, Salvadori PS, Tiferes DA, D'Ippolito G. Pneumatosis from esophagus to rectum: a comprehensive review focusing on clinico-radiological differentiation between benign and life-threatening causes. *Seminars in Ultrasound, CT and MRI.* 2018;39(2):167–182. https://doi.org/10.1053/j.sult.2017.09.002. /04/01/ 2018Epub 2017 Sep 9. PMID: 29571553.
5 Pieterse AS, Leong AS, Rowland R. The mucosal changes and pathogenesis of pneumatosis cystoides intestinalis. *Hum Pathol.* Jul 1985;16(7):683–688. https://doi.org/10.1016/s0046-8177(85)80152-0. PMID: 4007844.
6 Yale CE, Balish E, Wu JP. The bacterial etiology of pneumatosis cystoides intestinalis. *Arch Surg.* Jul 1974;109(1):89–94. https://doi.org/10.1001/archsurg.1974.01360010067017. PMID: 4365449.
7 Shinagare AB, Howard SA, Krajewski KM, Zukotynski KA, Jagannathan JP, Ramaiya NH. Pneumatosis intestinalis and bowel perforation associated with molecular targeted therapy: an emerging problem and the role of radiologists in its management. *AJR Am J Roentgenol.* Dec 2012;199(6):1259–1265. https://doi.org/10.2214/ajr.12.8782. PMID: 23169717.
8 DuBose JJ, Lissauer M, Maung AA, et al. Pneumatosis intestinalis predictive evaluation study (PIPES): a multicenter epidemiologic study of the eastern association for the surgery of Trauma. *J Trauma Acute Care Surg.* Jul 2013;75(1):15–23. https://doi.org/10.1097/TA.0b013e318298486e. PMID: 23778433.