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ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Surgery Quiz – Case 39

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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2022, 39(3):426–427

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A 93-year-old woman was examined in the emergency department because of acute abdominal pain at the left iliac fossa. The pain initiated 8 hours ago and palpation revealed signs of peritoneal inflammation at the left part of the abdomen. Abdominal X-ray (fig. 1) and subsequent computed tomography (CT) depicted a large ovarian cyst and intramural intestinal gas (fig. 2). A diagnosis of acute abdomen was made and the possibility of intestinal ischemia made acute laparotomy imperative. A large ovarian cyst was found to obstruct sigmoid colon and the whole small intestine contained gas inside its wall. A small cyst in the final ileus was perforated as well (fig. 3 a–c). The ovarian cyst was resected and the perforation at the final ileus was sutured.

Comment

Pneumatosis intestinalis (PI) is a radiological and intra-operative finding of gas in the bowel wall. Its incidence has been calculated to be 0.03% but it may be higher as many patients remain undiagnosed due to the possible asymptomatic presence of this condition. It does not have a gender predominance and its mean duration is 6 months.

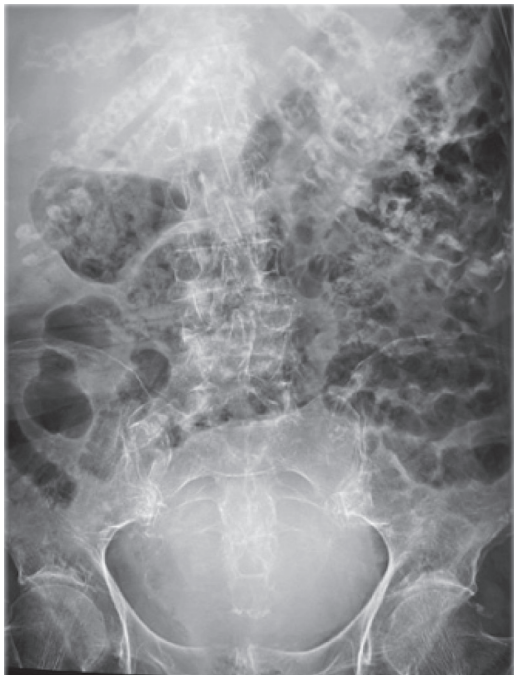

Figure 1. Abdominal X-ray, non-specific for pneumatosis intestinalis.

In PI, bowel subserous or submucosal layer contains gas, while the most commonly affected anatomical locations are the splenic fixture of transverse colon, the descending and the sigmoid colon. PI distribution may present a bubbly or a linear pattern.

PI can be distinguished as primary PI (15%) or secondary PI (85%). Secondary PI occurs as a result of different conditions such as gastrointestinal (GI) stenosis, inflammation or ischemia, chronic obstructive pulmonary disease (COPD), drugs, malnutrition and abdominal injury or surgery. A link of PI pathogenesis with COVID-19 has even been proposed by a case report, since the ability of SARS-CoV-2 to invade intestinal cells and provoke GI symptoms is established.

Three hypotheses exist about PI pathogenesis. According to the mechanical theory, when intraluminal pressure augments, the intestinal mucosa may rupture and gas is able to migrate from the normal GI cavity into the intestinal wall. Interventions, such as colonoscopy and biopsy may contribute in this procedure. On the other hand, pulmonary theory supports that chronic respiratory conditions, such as COPD, asthma and interstitial pneumonia, may provoke alveolar rupture. In that case, free gas may follow existing anatomical routes and may travel across mediastinum, aorta and

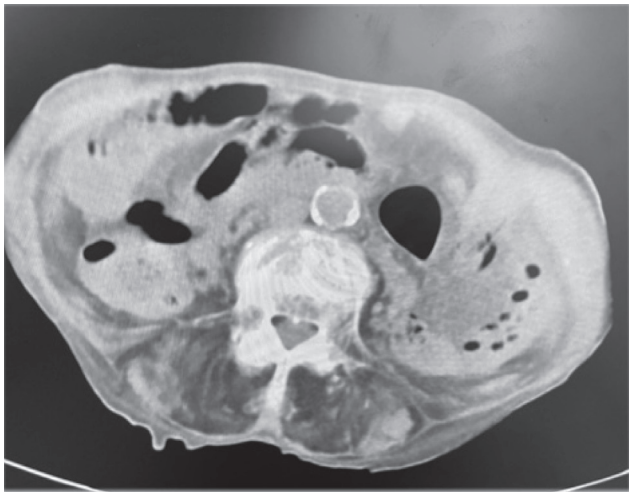

Figure 2. Abdominal computed tomography (CT) depicting intramural intestinal gas.

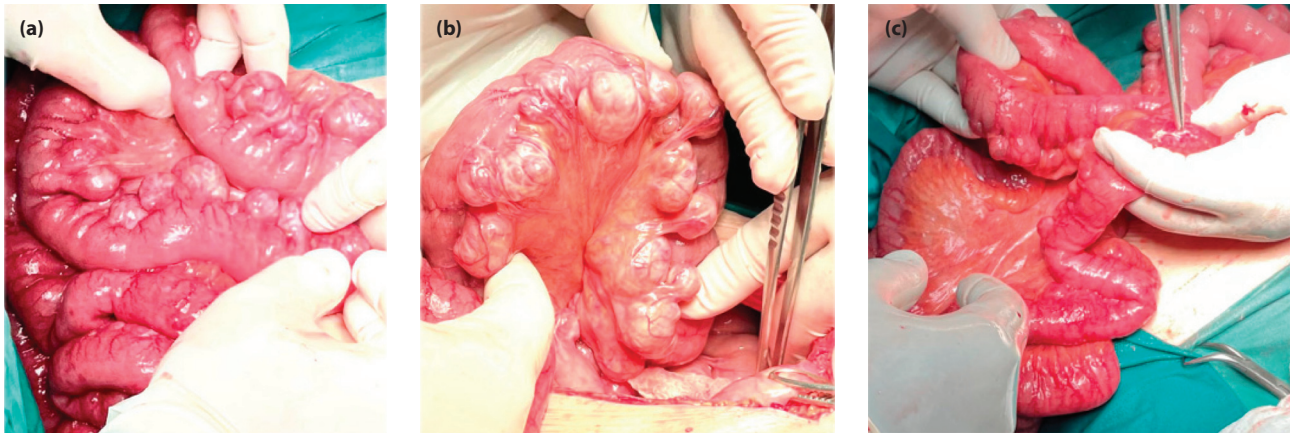

Figure 3. Laparotomy images, presenting multiple cysts of variable size in bowel wall, which contain gas.

mesenteric blood vessels before it positions itself inside the intestinal wall. Last but not least, the bacterial theory claims that bacteria, such as Escherichia coli and Clostridia, may penetrate intestinal mucosa and translocate inside intestinal wall, where they produce gas.

PI often remains asymptomatic. Primary PI may present with abdominal pain, diarrhea, nausea, vomiting, mucus in stool and hematochezia. Secondary PI may provoke all of the aforementioned symptoms but may also present with peritonism or manifestations of PI potential life threatening coexisting conditions such as pneumoperitoneum, volvulus, intestinal obstruction or intestinal ischemia in 3% of cases. In case of ischemia mortality is as high as 50 to 75%. It is noteworthy that neither the anatomical position nor the length of intestinal involvement correlates with the possibility and significance of intestinal ischemia.

PI is very often misdiagnosed as inflammatory bowel disease, intestinal polyps or tumors because most clinicians are not familiar with its existence. Even though radiography, CT, ultrasound (US) and colonoscopy may be implemented, PI diagnosis bases on CT findings. CT usually detects multiple grape-like or beaded low-density cystic transmission areas but may also reveal a linear or curvilinear pattern. CT also aids in the diagnosis or exclusion of intestinal ischemia or other potential coexisting conditions with high mortality. Elaborating, when PI is accompanied by portomesenteric venous gas or decreased mural contrast enhancement, intestinal ischemia must be suspected.

Laboratory finding are specific neither for PI nor for coexisting intestinal ischemia diagnosis. However, some indicative laboratory test results are white blood cells (WBCs) >12,000/mm^3, serum lactate >2.4 mmoL/L, arterial pH <7.34 and blood urea nitrogen (BUN) >7.7 mmoL/L.

Treatment of PI is usually successful with conservative measures such as observation, oxygen or hyperbaric oxygen therapy, antibiotics and endoscopic treatment. Initial conservative treatment proposes fasting, fluid replacement and potential discontinuation of alpha glycosidase inhibitors. Antibiotic therapy aims to inhibit bacterial infection and includes metronidazole and quinolones. Endoscopic fine needle aspiration can be both diagnostic and curative as it helps exhaust gas accumulation. Endoscopic resection of the PI cyst wall can also treat PI-induced bowel incomplete obstruction, since it can promote spontaneous cyst collapse. In case of intestinal incomplete obstruction relapse, endoscopic sclerotherapy of the cyst wall is indicated.

Surgical intervention can be avoided in most cases. However, it is necessary in case of severe or complicated PI. Operative indications include obstructive symptoms, WBCs >12,000/mm^3, CT findings of portal vein gas and signs of sepsis. Most alarming septic signs are arterial pH <7.3, serum lactate >2 mmoL/L, acute abdomen, acute kidney injury and hypotension.

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