Pneumatosis intestinalis: Ischemia vs non-ischemia

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Abstract

Pneumatosis intestinal is a common radiographic finding in Ischemic and Non-Ischemic bowel pathology. It is often a sign of the serious underlying illness and is associated with a poor prognosis. A case of pneumatosis intestinalis with mesenteric ischemia and rare association with abdominal tuberculosis in a young adult is presented here. In the setting of an acute abdomen, rapid evaluation is necessary to identify intra-abdominal processes that require emergent surgical intervention.

Keywords: Pneumatosis intestinalis, abdominal tuberculosis, mesenteric ischemia.

Introduction

Pneumatosis Intestinalis is the presence of gas bubbles inside the wall of the intestine. It has been reported to be associated with a variety of clinical conditions such as superior mesenteric ischemia, intestinal perforation, bowel necrosis, and infections. It is usually documented by radiological techniques such as X-ray, CT scan and endoscopy and is characterized by the typical pattern of gas in the wall of the intestine. The majority of cases may be managed conservatively. But intramural gas resulting from bowel necrosis is a surgical emergency.

After its diagnosis, its pathogenesis should be ascertained because the appropriate treatment is related to the underlying etiology. Surgical treatment should be considered in symptomatic patients presenting with an acute abdomen, signs of ischemia, or bowel obstruction. In asymptomatic patients with inconspicuous findings, the underlying disease should be treated first.

Mesenteric ischemia is classified into two forms, acute and chronic, which are differentiated on the timing of symptom onset and extent of decreased blood flow. Mesenteric ischemia is further subdivided by etiology: arterial, venous, and non-occlusive. In a general sense, intestinal ischemia frequently presents with nonspecific clinical symptoms. Pneumatosis of the small bowel mesentery is a pathological sign, characterised by gas within the mesenteric sleeves and it is likely associated with significant morbidity.

Two main theories of the pathophysiology of gas formation in the bowel wall have been proposed, based on a mechanical or a bacterial cause. The mechanical theory hypothesizes that the gas moves into the bowel wall from either the intestinal lumen of the lungs via the mediastinum by different mechanisms, leading to an increase in pressure. The bacterial theory proposes that the gas is produced by gas-forming organisms that enter the mucosal barrier through mucosal rents or increased mucosal permeability and produce gas within the bowel wall.

Case report

A 55 years old man presented to the emergency room with severe abdominal pain, which started a few days before the presentation. His medical problems included type 2 diabetes mellitus, hypertension, hyperlipidemia, and alcohol abuse. On examination, he had mild abdominal wall rigidity, guarding and tenderness. Otherwise, examination and laboratory studies were unremarkable.

Imaging radiography

Plain radiography demonstrated dilated fluid-filled bowel loops, suggesting a bowel obstruction. Pneumatosis noted in the bowel wall.
Ultrasound showed focal dilated small bowel loops with absent peristalsis and minimal interbowel fluid.

**Computed tomography**

A non-contrast CT scan of the abdomen showed 'pneumatosis intestinalis' involving focal dilated small bowel loop.

Contrast CT showed a filling defect in the distal segmental branches of the superior mesenteric artery. Mild atherosclerotic disease of the mesenteric arteries was noted.

Lack of bowel wall enhancement involving focal segment of the ileal loop.

Computed tomography angiogram demonstrates the nonopacification of the superior mesenteric artery at the level of occlusion.

Mesenteric ischemia was high on differential due to multiple risk factors. He was taken for surgery. A small segment of the ileal loop was found to be necrosed and it was resected.
Case 2: A twenty-eight-year young man, was admitted to our hospital, with a history of increasing abdominal distention, abdominal pain, weight loss, anorexia, and malaise. He had been seen and investigated by surgeons in our hospitals, including a diagnostic laparotomy. On examination, he looked ill. He was apyrexial with no lymphadenopathy. Abdominal examination revealed diffuse distension and tenderness. The patient appeared unstable with clinical evidence of abdominal sepsis. Physical examination revealed pallor and a slightly malnourished condition (a BMI of 20), a distended abdomen with diffuse abdominal pain and normal borborygmi. The cardiopulmonary examination was uneventful (blood pressure: 100/60 mm Hg, pulse rate: 120/min, respiratory rate 40/min). The laboratory tests showed mild elevated liver enzymes (AST: 76 IU/L; ALT: 33 IU/L; Alk. phos: 109 IU/L), blood urea 96 mg/dl, s.creatinine 2.3 mg/dl, and s.bilirubin 5.5 mg/dl.

IMAGING

Plain abdominal x-rays showed dilated loops of bowel with extensive intramural gas.

Ultrasound

Abdominal ultrasound demonstrated the presence of dilated intestinal loops and extensive echogenic foci within the bowel wall.

Computed tomography

CT demonstrated nonspecific dilated bowel with wall thickening, ascites, and mesenteric edema. An abdominal coronal and axial CT images confirmed extensive small bowel pneumatosis. Inflammatory bowel disease may produce a similar appearance. Concurrently, thoracic CT revealed mediastinal lymphadenopathy associated with left pleural effusions.

Despite radiological findings, the surgeons proceeded with emergency laparotomy because of perforative peritonitis. The stomach, jejunal and ileal loops were not separable due to adhesions. Multiple tubercles were present all over the visceral peritoneum, small bowel, and mesentery. Biopsy specimen was taken from the omentum and nodules. Histopathological diagnosis was reported as 'multiple caseating tubercles'.
Fig 12, 13: The surgical image shows multiple tubercles in the bowel wall, mesentery, and omentum

Fig 14, 15: Histopathological image shows features of caseating tubercles

Since radiological studies and laboratory tests may be non-diagnostic, tuberculosis must be always considered in the differential diagnosis of unusual gastrointestinal presentations. Unfortunately, he died suddenly on the third postoperative day, and the possible cause of death may be septicemia with gas embolism and multiorgan dysfunction. The development of PI may be caused by tuberculosis, resulting in the atrophy of Peyer’s patches, the impairment of mucosal integrity, and the transmigration of gas-producing bacteria through the mucosa. These processes finally result in submucosal and subserosal gas accumulation.

Discussion
Different etiologies of PI can be discussed in acute abdomen, including intestinal tuberculosis. PI does not represent a closed entity, but rather a radiological finding that can be caused by benign conditions up to fulminant gastrointestinal diseases with a lethal outcome. Arterial embolism is the most frequent cause of mesenteric ischemia, accounting for 40%–50% of all AMI cases. The SMA is the visceral arterial branch that is most vulnerable to embolism owing to its low branching angle from the aorta.

The most important prognostic factor in patients with AMI is intestinal viability. Those with peritonitis and signs of bowel infarction or perforation should undergo emergent open laparotomy, which allows for direct visualization of the bowel, reestablishment of blood flow to areas of ischemic bowel, and resection of all regions of non-viable intestine.

M. tuberculosis is a rare cause of the infectious conditions underlying Pneumatosis Intestinalis. Tuberculosis is still a considerable problem in developing countries. Diagnosis of abdominal tuberculosis is difficult because it mimics many other abdominal conditions, such as abdominal mass, pain, lymphadenopathy, fever, ascites, and weight loss. CT has been used to evaluate patients with clinical signs and symptoms of ischemic bowel disease. Depending on the severity of the disease, intestinal ischemia manifests on CT by aspectrum of findings, including dilatation of bowel, mural thickening, a mural stratification pattern, mesenteric edema, mural or mesenteric hemorrhage, ascites, pneumoperitoneum, mesenteric arterial or venous thrombi, and portomesenteric venous gas. In the clinical setting of intestinal ischemia, pneumatosis and portomesenteric venous gas have been considered to be signs of advanced disease, usually indicating irreversible injury and transmural necrosis. However, the use of CT improves our ability to detect even subtle cases of pneumatosis. Both of these studies refute the long-held concept that pneumatosis is a specific sign of bowel infarction in ischemic bowel disease. Presumably, gas can enter the ischemic bowel wall via a disrupted mucosa in the absence of transmural infarction. This observation explains our ability to detect pneumatosis on CT in patients with viable bowel in whom surgical resection is not required.

Conclusion
We present this case to raise awareness of the diagnosis of abdominal tuberculosis in patients with Pneumatosis Intestinalis in developing countries. Pneumatosis Intestinalis often have a poor prognosis. Abdominal tuberculosis with pneumatosis intestinalis may be an unusual presentation in...
adults. We recommend that cases with PI should be carefully observed. In conclusion, our data suggest that the CT finding of pneumatosis does not always indicate transmural infarction of the bowel in intestinal ischemia.

Fig. 1.a–c The initial CT scan of the abdomen. The scan detected linear collections of gas in the subserosal or submucosal layers of the ascending colon wall (arrow), accompanied by pneumoperitoneum and pneumoretroperitoneum (star). D–f On the follow-up CT scan, no signs of a pneumatosis were detectable.

References