Case Report

Esophageal pneumatosis in the setting of small bowel ileus with acute resolution after nasogastric tube decompression

Sanjit O. Tewari MD*, Allen R. Wolfe MD, Richard Seguritan MD, Raihan Faroqui BS, Michael Meshreki MD

Department of Radiology, Richmond University Medical Center, 355 Bard Avenue, Staten Island, NY 10310, USA

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ABSTRACT

Esophageal pneumatosis is a rare condition with diverse potential etiologies including traumatic, mechanical, ischemic, obstructive respiratory, autoimmune, immunodeficient, and infectious causes. Here, we present a case of esophageal pneumatosis in the setting of upper gastrointestinal and small bowel ileus, diagnosed on computed tomography (CT), with acute resolution after nasogastric tube decompression. A patient presented to the emergency department with epigastric discomfort. CT of the abdomen/pelvis demonstrated intramural air in the mid-to-distal esophagus, consistent with esophageal pneumatosis, and diffuse dilatation of the visualized esophagus, stomach, and small bowel, consistent with an ileus. Patient was managed with nasogastric tube decompression and bowel rest. Subsequent esophagram did not demonstrate any evidence of perforation and a repeat CT of the abdomen/pelvis, performed 11 hours after initial diagnostic CT, demonstrated interval resolution of patient's esophageal pneumatosis, and improvement of patient's ileus.

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Introduction

Esophageal pneumatosis is a rare condition with diverse potential etiologies including traumatic, mechanical, ischemic, obstructive respiratory, autoimmune, immune deficient, chemotherapy side effect, and infectious causes [1–5]. Here, we present a case of esophageal pneumatosis in the setting of upper gastrointestinal and small bowel ileus, diagnosed on computed tomography (CT) with subsequent acute resolution after nasogastric tube decompression, confirmed on interval CT just 11 hours after the initial diagnosis.

Case report

An 87-year-old man presented to the emergency department (ED) complaining of epigastric discomfort that woke him from...
sleep the night before. The patient described the discomfort as a burning sensation radiating to his chest and left upper quadrant, ranging from 5 to 7 of 10 and without any relieving or exacerbating factors. The patient also complained of nausea and had 1 episode of watery, nonbloody emesis after arrival to the ED with associated hiccupping. The patient denied any complaints of recent chest pain, fever, diarrhea, constipation, sneezing, coughing, or other Valsalva maneuvers. The patient’s past medical history was significant for coronary artery disease, angina, valvular heart disease, hypertension, hyperlipidemia, gout, gastro-esophageal reflux disease, and a recent hospital admission for constipation which resolved uneventfully after medical treatment. The patient denied any history of autoimmune disease, connective tissue disorder, or any history of upper gastrointestinal endoscopy or concomitant esophageal biopsy. His home medications included furosemide, atenolol, doxazosin, nifedipine, simvastatin, nitroglycerin PRN, omeprazole, and oxybutynin. Finally, he denied current smoking or alcohol usage.

On presentation, patient’s vital signs were remarkable only for hypertension (170/74 mm Hg). He was afebrile. Physical examination was remarkable for slight abdominal distention and sluggish bowel sounds; however, the abdomen was otherwise soft and nontender. Cardiac enzymes were negative, and there were no acute electrocardiogram changes. Remainder of physical examination and laboratory studies, including serum lactate and creatinine, were unremarkable.

With clinical concern for small bowel obstruction, contrast-enhanced CT of the abdominal and pelvis was performed. CT images demonstrated diffuse dilation of the small bowel, stomach, and mid-to-distal esophagus, without a discrete transition point, with associated esophageal pneumatosis, extending from the mid-to-distal esophagus into the proximal stomach (Fig. 1). No pneumomediastinum, pneumoperitoneum, or other sites of pneumatosis were identified in the abdomen/pelvis. Findings were considered consistent with an upper gastrointestinal and small bowel ileus with associated pneumatosis of the mid-to-distal esophagus.

At this time, an esophagram was attempted to evaluate for esophageal perforation, however, was nondiagnostic secondary to patient being unable to drink the water-soluble contrast. Patient subsequently began to decompensate and was admitted to the surgical intensive care unit. Nasogastric tube was placed for decompression which drained 1700 ccs of nonbloody fluid. The patient was then treated with bowel rest, intravenous fluids, and close observation. Four hours after the initial CT examination, an esophagram was performed through the patient’s indwelling NG tube and was found to be negative for perforation and otherwise unremarkable (Fig. 2). Additional nasogastric tube decompression was also performed under fluoroscopic guidance following completion of the diagnostic esophagram. Seven hours after the esophagram and 11 hours after the initial CT demonstrating esophageal pneumatosis, a repeat CT examination was performed which demonstrated complete resolution of the esophageal pneumatosis and interval improvement of patient’s upper gastrointestinal ileus (Fig. 2).

The patient was continued on bowel rest and initially managed expectantly. On hospital day 2, the patient became septic and was started on broad-spectrum antibiotics.
Tracheal aspirates eventually grew *Klebsiella pneumoniae*. Patient’s intravenous antibiotics treatment was complicated by acute kidney injury. Patient’s antibiotic regimen was subsequently adjusted, his acute kidney injury resolved, and he was discharged home tolerating a regular oral diet on hospital day 12.

**Discussion**

Here, we report a case of esophageal pneumatosis in the setting of upper gastrointestinal ileus, treated with NG tube decompression and bowel rest, with interval resolution of the esophageal pneumatosis, confirmed on cross-sectional imaging within just 11 hours of initial presentation. In light of patient’s CT diagnosis of ileus, we hypothesize that our patient developed esophageal pneumatosis secondary to mechanical rather than bacterial causes. According to mechanical theory, air present in the gastrointestinal tract lumen dissects into the walls of the gastrointestinal tract through a mucosal tear [1,5]. However, as in our case, even if a mechanical cause is suspected, ischemia of the intestine should be ruled out [2,6,7]. Although patient’s ileus was considered to be the primary precipitating mechanical factor, the episode of hiccupsing in the ED may have been an aggravating factor. Bacterial theory posits that the air infiltrating the esophageal wall is produced by gas-forming bacteria in the bowel such as *Escherichia coli* and/or *Clostridium* [2]. We consider this to be less likely in our case, although there are reports of gas-forming abscesses caused by *Klebsiella Pneumoniae* [8].

Multiple reports have demonstrated the efficacy of conservative management of pneumatosis [6,8–11], particularly in the absence of elevated serum lactic acid or creatinine [10] and lack of image findings to suggest ischemia such as mural nonenhancement [6], neither of which were present in our case. A recent case series involving pneumatosis intestinalis demonstrated similar efficacy for treatment by different modalities including endoscopy, surgery, and conservative approach [11,12]. Similarly, our case responded to conservative approach while also providing evidence of acute expedience of noninvasive decompressive therapy. To our knowledge, there are no reports in the literature confirming resolution of pneumatosis within the short time frame of 11 hours after noninvasive decompressive therapy.

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Fig. 2 – Fluoroscopic spot images of the esophagram study (A and B) demonstrate no evidence of extravasated contrast to suggest perforation. Follow-up CT images obtained 11 hours after the initial CT demonstrate resolution of esophageal pneumatosis (C and D) and gross improvement of previously seen gastric and small bowel dilatation (E). CT, computed tomography.
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