CASE REPORTS

An incidental finding of pneumatosis intestinalis: Conservative management without oxygen therapy

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ABSTRACT

Pneumatosis intestinalis can be identified radiographically incidentally in an asymptomatic patient, or it may be present in its fulminant form with peritonitis. Although multiple mechanisms have been postulated, most believe it arises from mechanical or infectious factors. Respiratory factors have also been described as possible causes for this condition. Clinically, it is important to differentiate among patients whom require surgical intervention from those who would benefit from conservative management, such as hyperbaric oxygen, changes in diet, and/or antibiotic administration. Although supplemental oxygen has become the standard of care for the treatment of benign pneumatosis intestinalis, we question whether all patients require oxygen therapy as a treatment. Although oxygen may be beneficial, the literature suggests there may be detrimental effects from oxygen toxicity and the free radicals formed during hyper-oxygenation. Furthermore, given the rising epidemic of antibiotic resistance and the various toxicities associated with usage of antibiotics, do all patients really require antibiotics? We present a case of a patient with complaints of hematuria, but no other gross abdominal complaints and was incidentally found to have pneumatosis intestinalis and pneumoperitoneum without any evidence of vascular compromise or ischemia. This patient was managed successfully with conservative treatment without oxygen therapy or antibiotics.

Key Words: Incidental pneumatosis intestinalis, Conservative management, Oxygen therapy

1. INTRODUCTION

Pneumatosis intestinalis, pneumatosis coli, and pneumatosis cystoides intestinalis, peritoneal pneumatosis, intestinorum abdominal gas cysts, intestinal emphysema or intestinal gas cysts are synonyms for the same disease process.[1–3] It is marked by air or gas-filled cysts within the wall of the GI tract, potentially involving the small bowel, large bowel, stomach, or esophagus, which may be subserosal and/or submucosal.[2,4] These cystic collections of gas have been recognized as early as 1730.[5] The most common site is the small bowel followed by the colon.[3] The incidence and prevalence of this disease process are difficult to ascertain because most patients are often asymptomatic, which brings us to the question of who needs treatment. Treatment is usually conservative in the form of hyperbaric oxygen, changes in diet, and/or antibiotics. There is a subset of patients that require surgery for pneumatosis intestinalis; generally, these patients are hemodynamically unstable and/or are peritonitic. Regarding asymptomatic patients, it is important to determine how aggressively to treat or not.

2. CASE PRESENTATION

We present a case of a 79-year-old male who presented to our community hospital's emergency department with a chief complaint of gross hematuria and dysuria. The patient reported these symptoms for two weeks prior to presentation.

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He was in contact with urology and was to undergo a cystoscopy and a computed tomography of the abdomen and pelvis scan for further evaluation as an outpatient. The patient reported that he presented to the emergency department because of worsening dysuria and hematuria. The patient denied any complaints of fevers, chills, abdominal pain, nausea, vomiting, hematochezia, melena, hematemesis, or any changes in his bowel habits. He underwent a colonoscopy in 2014 and was found to have nonthrombosed external hemorrhoids, diverticulosis, and a polyp in the mid-descending colon, which was removed and final pathology was benign. He underwent an upper endoscopy in 2012 and was found to have a small hiatal hernia in addition to gastritis. The only significant past medical/surgical history was greenlight laser therapy for benign prostatic hyperplasia and cardiac angioplasty with drug-eluting stent placement. Family history and social history were noncontributory to the case.

Upon physical examination, the patient was noted to be afebrile and hemodynamically stable upon arrival with the following vital signs: Temperature of 36.9 degrees Celsius, BP: 165/85, HR: 58, RR: 14, O_2 Saturation 98%. The patient appeared to be in no acute distress, and the abdomen was noted to be soft, nontender, and nondistended. He underwent a rectal exam, which revealed external hemorrhoids without evidence of masses. The remainder of his physical was benign.

The patient's labs revealed a WBC count of 7,000 $\times$ 10^6/L with neutrophils of 78% without bandemia. His hemoglobin and hematocrit were 13.5 gm/dl and 40.1%. Comprehensive metabolic panel, amylase, and lipase were normal. The patient also underwent a UA which was grossly positive for blood, but negative for underlying infection. The patient underwent a computed tomography abdomen and pelvis scan with oral and intravenous contrast (see Figure 1), which revealed several locules of free intraperitoneal air, pneumatosis intestinalis involving the ascending colon, and the stomach appeared thickened with no evidence of extravasation of contrast. Additionally, he was noted to have a 3.3 cm abdominal aortic aneurysm, enlarged heterogeneous prostate gland protruding into the floor of the bladder, and a stable liver cyst measuring 2.5 cm. Otherwise, the bladder appeared normal without any evidence of emphysematous cystitis, bladder wall thickening, or any associated fat stranding. The patient was admitted to the general surgery service and was managed conservatively: nothing by mouth, intravenous fluid resuscitation, protonix drip given thickened appearance of stomach, serial abdominal exams, and antibiotics and oxygen therapy were withheld. It was decided to continue with conservative measures and surgical intervention would follow if the patient developed a leukocytosis, became hemodynamically unstable, or if the patient became peritonitic. Conservative management was continued for the next three days, during which time the patient continued to deny any abdominal pain, hematuria had resolved, remained completely hemodynamically stable, physical exam remained benign, and labs revealed no evidence of a leukocytosis or left shift with a stable hemoglobin and hematocrit. By hospital day four, the patient was initiated on a clear liquid diet which he tolerated well and a repeat computed tomography abdomen and pelvis with oral and intravenous contrast (see Figure 2) was performed, which revealed resolution of the pneumoperitoneum, but the pneumatosis intestinalis of the ascending colon appeared grossly unchanged. By hospital day six, the patient continued to progress as expected without onset of abdominal pain, hemodynamically stable, with normal labs and a benign physical exam. His diet had been advanced to a regular diet and he had met all discharge criteria. He was discharged home in stable condition with planned repeat computed tomography abdomen and pelvis with oral and intravenous contrast as an outpatient and follow-up with urology for his hematuria.

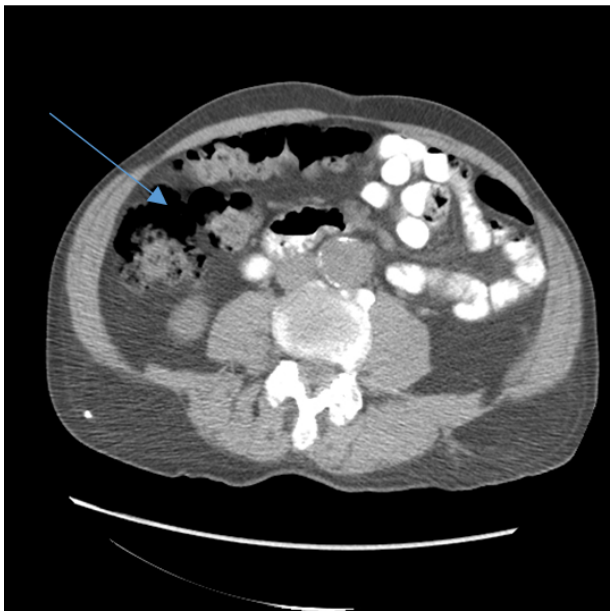

Figure 1. Arrow depicting pneumatosis intestinalis

Upon follow-up with urology, the patient underwent a cystoscopy, he was found to have large amounts of clots in his bladder and bleeding was found related to residual adenoma from greenlight laser prostatectomy. Bleeding was controlled with resection of prostate tissue. Repeat repeat computed tomography abdomen and pelvis with oral and intravenous contrast (see Figure 3) was obtained nine weeks after hospitalization, which revealed no further evidence of pneumatosis intestinalis of the ascending colon, pneumoperitoneum, or

free fluid. When seen and evaluated in the office, the patient continued to deny any complaints of abdominal pain. He was tolerating a regular diet and was having normal bowel movements. However, he did initially complain of symptoms related to his hemorrhoids, but these were medically managed and subsequently resolved.

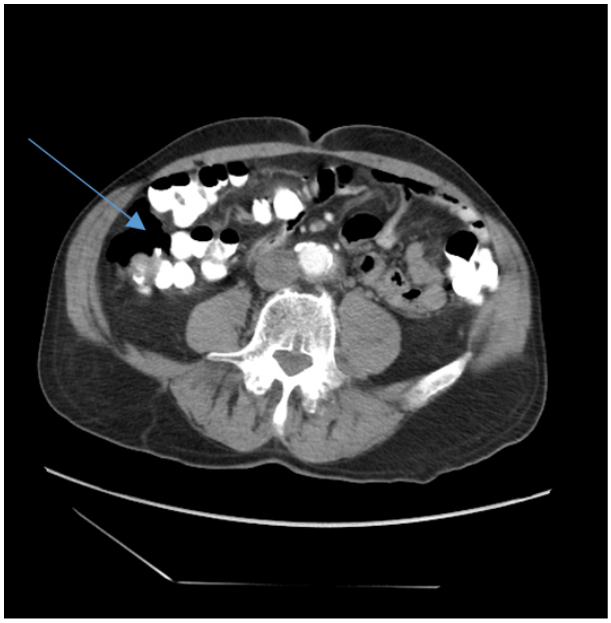

Figure 2. Arrow depicting unchanged pneumatosis intestinalis

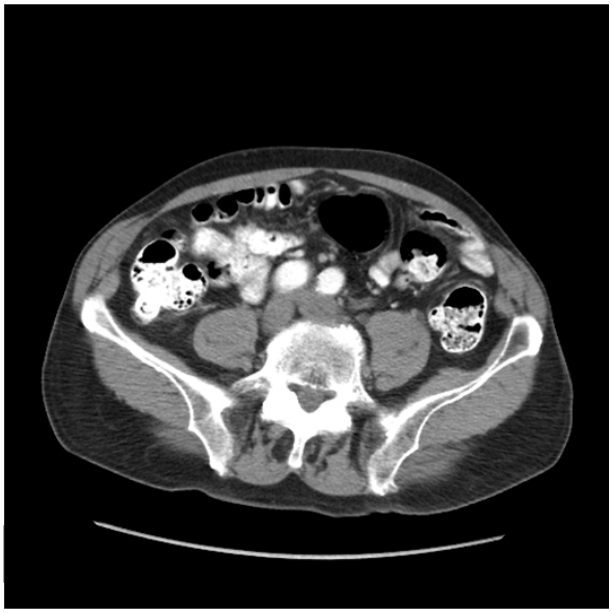

Figure 3. Resolution of pneumatosis intestinalis

3. DISCUSSION

Pneumatosis intestinalis is an interesting disease process that is often difficult to identify because many of the patients, including our patient, are asymptomatic at the time of diagnosis, which makes it difficult to identify the incidence and prevalence.[1] Given concerns for ischemia and the possibility of impending perforation, general surgery is often consulted early. However, there are multiple causes including obstruction, infarction, irritable bowel disease, intestinal neoplasms, enteritis, appendicitis, tuberculosis, adhesions, a prior end to end anastomosis, obstructive pulmonary disease, drug-induced, pyloric stenosis, immunosuppression related, trauma, or it may be idiopathic.[1,3,5] These patients may have a completely benign disease or they may be present in extremis. Patients may have the fulminant or the benign form of the disease; fulminant pneumatosis intestinalis is associated with an acute bacterial process, sepsis, and necrosis of the bowel.[2] Symptoms of pneumatosis intestinalis include diarrhea, constipation, rectal bleeding, rectal urgency, the passage of mucus per rectum, vague abdominal discomfort, abdominal pain, abdominal distention, urgency, weight loss, malabsorption, flatulence, or no complaints at all.[2,4,6] Pain and emesis are the most common presenting symptoms.[7] Other symptoms are related to complications of pneumatosis intestinalis, occurring in about 3% of patients, and include obstruction, pneumoperitoneum, tension pneumoperitoneum, hemorrhage, perforation and intussusception.[2,3,6]

There are multiple theories which attempt to explain the pathogenesis of pneumatosis intestinalis. One model suggests a mechanical breakdown in the mucosa, which is followed by extravasation of air into the GI tract wall and possibly into the peritoneum.[4,8] A sub group of patients with pulmonary disease may have pneumatosis intestinalis from a cough and rapid changes in intraabdominal pressure, which produce interstitial pulmonary emphysema and pneumomediastinum, which finally leads to a dissection of air through the posterior anatomic planes to the blood vessels or lymphatics of the intestinal wall.[4,6] A similar theory postulates that air may travel from ruptured alveoli to the vasculature to the bowel wall.[7] According to the bacterial model, when gas-producing bacteria such as Clostridia species gain access to the GI tract wall, they produce pockets of gas leading to pneumatosis intestinalis;[4,6] this may also occur after procedures such as sigmoidoscopy, colonoscopy, or mucosal biopsies.[2] Additional dietary and chemical theories exist, which hypothesize an increased lactic acid level due to a certain diet or an altered carbohydrate metabolism, which subsequently leads to decreased carbon dioxide and oxygen resorption with cyst formation.[2]

It is important to determine not only if and when to treat, but also how to successfully treat these patients. Treatment modalities have included high flow oxygen therapy, diet modification, or surgical intervention. Additionally, some

argue the importance of antibiotics in the treatment of all cases. The gas contained within these cysts is estimated to be 72.5%-90% nitrogen, 5%-16% oxygen, 10% hydrogen and 0.3%-4% carbon dioxide, noting free diffusion of gas between bowel lumen and capillaries.[1,6] Patients treated with high flow oxygen therapy have been noted to have radiographic resolution of their pneumatosis intestinalis in a matter of days; therapy may be administered via a head tent, hyperbaric oxygen, or a nonrebreather mask.[1,2,6] It is thought that the high concentrations of oxygen lead to denitrogenation of the blood, which inevitably eradicates the gas cysts.[1,2,6] These patients may still have a recurrence.[1,2] Our patient, without any oxygen therapy or the use of antibiotics, showed complete resolution of his pneumatosis intestinalis. Oxygen has been known to cause toxicity as early as the late 1800s.[9] The mechanism of oxygen toxicity is attributed to oxygen-free radicals, which have one or more free electrons, making them unstable; these may combine with other species and may directly or indirectly react with lipids, DNA, and proteins, causing cell signaling abnormalities to significant damage in the form of necrosis and apoptosis.[9] Studies in mice have revealed exposure to 100% O_2 result in mortality after 3-4 days, secondary to diffuse alveolar damage and alveolar edema leading to respiratory failure; also, on a cellular level damaged cells were noted to have signs of apoptosis and necrosis.[10] Hyperbaric oxygen therapy is thought to be safe, but can still cause myopia, cataract formation, rupture of the middle ear, cranial sinuses, barotrauma in general, seizures, tracheobronchial symptoms, and decrements in pulmonary function.[11] It is important to remember oxygen is a prescribable drug that affects the natural biochemistry and physiology; aside from its combustibility, oxygen can cause vasodilation of the pulmonary vasculature and vasoconstriction of the systemic circulation, decline in lung function, chronic pulmonary fibrosis, emphysema, visual changes, including tunnel vision, tinnitus, nausea, dizziness, and confusion, and ocular toxicity.[9] The risks of oxygen therapy may outweigh the benefits of treating pneumatosis intestinalis in asymptomatic patients. There are many proponents that advocate the use of antibiotics in these patients, however, we recommend a more selective approach. Antibiotics are not without risk in modern medicine we are dealing with rising resistance to antibiotics, toxicity associated with antibiotics, and Clostridial difficile infections; all of which can be decreased when antibiotics are used appropriately.

Regarding treatment, an elemental diet has also been shown to resolve pneumatosis intestinalis with symptomatic relief, but these patients may also have a recurrence.[2] Surgery is appropriate in fulminant cases of infants and adults where any delay may lead to a poor outcome with necrosis of the bowel, sepsis, and death. It is important to note, mortality is high even with surgery in fulminant cases.[2] However, given that all patients are not symptomatic, it is vital to determine who truly needs surgery. It is important to stratify patients into those who would benefit from surgery with a good prognosis for recovery, those who are severely ill with risks that outweigh the benefits (surgery may be futile), and those who have benign pneumatosis intestinalis.[7] A retrospective study was performed using the radiology database of the Mount Sinai Medical Center for cases of pneumatosis intestinalis between 1996-2006, and they concluded patients with pneumatosis intestinalis, a WBC > 12, +/- emesis, > 60 years of age were most likely to require a surgical intervention and those with sepsis were likely to have a significantly higher mortality.[7] Wayne et al. created a slightly different stratification and treatment plan, following 88 patients. They stratified the patients into patients with mechanical disease, acute mesenteric ischemia, and benign idiopathic groups. Those in the benign idiopathic group that underwent surgical intervention were noted to have negative laparotomies with benign pathology, patent vasculature, and viable tissues. Those patients that were managed nonoperatively were noted to be doing well and were alive 30 days after initial imaging. Per their results, early surgical intervention in patients with pneumatosis intestinalis +/- portal venous gas yielded a non-therapeutic laparotomy rate in 30%.[4] They attempted to use the Greenstein algorithm mentioned above, but were unable to find it clinically useful. Depending on their vascular disease score (assessing vascular risk factors, coronary artery disease, peripheral vascular disease, risk for low-flow state, vasculitis, abdominal pain or abnormal abdominal exam, Lactate $\geq$ 3, or a small bowel pneumatosis) the patients were most likely to have a diagnosis strongly suspecting mesenteric ischemia, possible mesenteric ischemia, or benign pneumatosis intestinalis/portal venous gas, and treatment was chosen appropriately.[4] In summary, patients with an acute abdomen, metabolic acidosis, elevated lactate, leukocytosis warrant surgery, while less severe cases of pneumatosis intestinalis may be treated conservatively and oxygen therapy may not always be necessary.[11] It is important to remember although oxygen therapy and antibiotics are readily available, they are not without toxicity. We believe that conservative management with careful observation and without oxygen therapy or antibiotics may be equally effective in select cases as in the case of our patient with an incidental finding of pneumatosis intestinalis and without any evidence of ischemia. Given the small sample size, further investigation is necessary.

CONFLICTS OF INTEREST DISCLOSURE

The authors declare they have no conflicts of interest.

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