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Gastric pneumatosis: An unexpected complication in multiple myeloma patient

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Abstract

Gastric pneumatosis is an unusual and rare condition with unclear management recommendations. We report a case of gastric pneumatosis in an adult patient with multiple myeloma on chemotherapy with concomitant fungemia. Patient presented with right lower abdominal pain and subsequently developed spinal cord compression and got treated with high dose dexamethasone and chemotherapy. Patient developed upper GI symptoms and diagnostic imaging was ordered. A CT scan of the abdomen and pelvis was done which showed intramural gas in the gastric and esophageal wall along with positive blood cultures for candida krusei. Our patient was managed conservatively with antifungal, antibacterial therapy along with decompression via nasogastric tube but failed to improve. General surgery team recommended operative management, but the patient and family opted for comfort measures.

Keywords: Multiple myeloma; Intramural gastric air; Candidemia.

Introduction

Pneumatosis intestinalis is an exceedingly uncommon condition which is characterized by the presence of air-filled cysts within the wall of gastrointestinal (GI) tract. Its pathogenesis and etiology are poorly understood, but several hypotheses propose mechanical, biochemical, or infectious etiology. It can involve any part of the gut from the esophagus up to the rectum. The incidence of PI is from 0.03 to 2% in the general population [1]. Gastric pneumatosis is a rare complication characterized by air in the gastric wall. Fraenkel first described it in 1889 [2]. Conservative management with early recognition and treatment of the underlying condition is preferred as surgical intervention has not shown to significantly improve mortality [3]. We describe a case of gastric pneumatosis that failed despite conservative management.

Case presentation

A 58-year-old female was admitted to our hospital with a complaint of right lower abdominal pain. Her medical history included relapsed IgG kappa multiple myeloma (MM) on Pomalidomide, Cytoxan and dexamethasone, Deep vein thrombosis, coronary artery disease, and diabetes mellitus on insulin pump. She had no history of abdominal surgery or connective tissue disorders. MM was diagnosed two years ago with last chemotherapy four weeks ago prior to ED (Emergency Department) presentation. In the ED, she had CT (computerized tomography) of the abdomen and pelvis that showed a right iliac bone lytic lesion concerning for myeloma relapse. She started having lower back pain associated with bilateral leg weakness. MRI (magnetic resonance imaging) of the spine showed thecal sac compression at the level of thoracic T10 vertebra. She received high-dose dexamethasone along with chemotherapy (daratumumab, vincristine, cisplatin, adriamycin, cyclophosphamide, and etoposide). One week later, she started having worsening upper

abdominal pain associated with nausea and vomiting. Physical exam revealed mild epigastric tenderness, slight abdominal distension, and sluggish bowel sounds. Repeat CT scan of abdomen and pelvis showed air in the esophageal wall extending from the mid esophagus to the posterior wall of the stomach (Figure 1). A complete blood count showed severe leukopenia and thrombocytopenia with white blood cell count (WBC) 0.1 K/uL, hemoglobin 10.4 g/dl, platelets 19 k/uL. Serum chemistry showed a serum sodium 136(135-145 mmol/L), hypokalemia with level 2.9(3.5-5.1 mm/L, acute kidney injury (AKI) with blood urea nitrogen (BUN) 43(6-20 mg/dl) creatinine 1.3 mg/dl (patient baseline is 0.8) calcium 8.5(8.6- 10.2 mg/dl) lactic acid 1.6(0.5 to 2.2 mmol/L) and normal liver function tests. Surgery team was consulted and evaluated her with recommendations for conservative management with nasogastric decompression and intravenous fluid, antibiotics, and antifungals. She received treatment with micafungin, vancomycin, meropenem, and acyclovir prophylaxis. Due to progressive worsening of abdominal distention and metabolic derangements, she was transferred to the intensive care unit. Blood cultures grew Candida krusei but was negative for any bacterial growth. Infectious disease was consulted, and micafungin was switched to amphotericin B. She failed conservative management with worsening abdominal distension. A family meeting was arranged, and patient and family opted for comfort care and refused surgical intervention. She was transitioned to inpatient hospice and passed away during hospitalization.

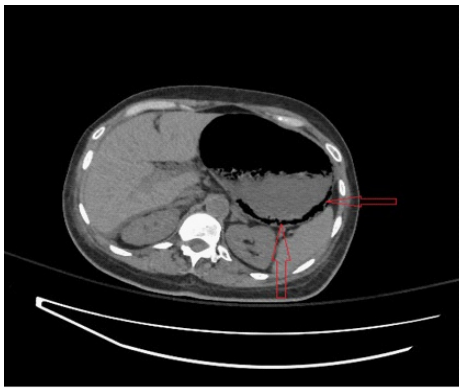

Figure 1: Arrow showing air in posterior wall of stomach

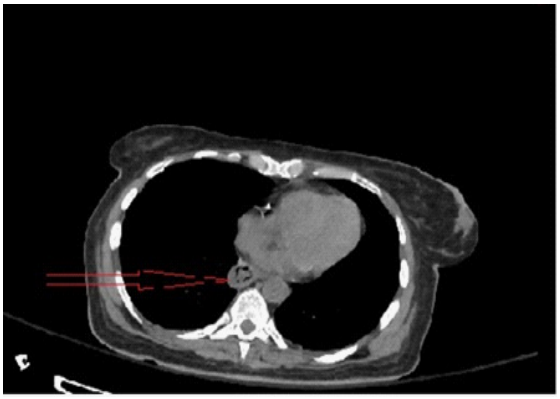

Figure 2: Arrow pointing air in wall of esophagus

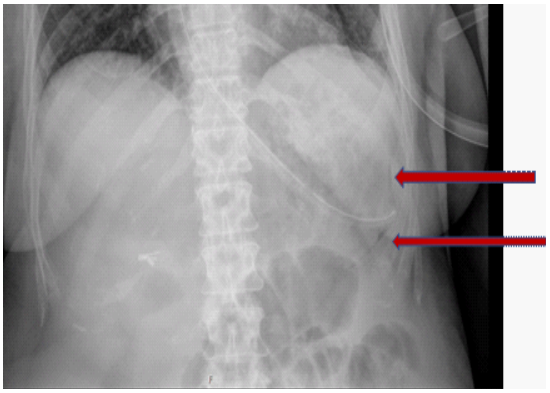

Figure 3: Arrows pointing air in the wall of stomach

Discussion

Gastric Pneumatosis can be divided into two main categories gastric emphysema and emphysematous gastritis. Emphysematous gastritis is an uncommon clinical entity with high mortality documented in up to 75% of patients who require surgery [4]. It is characterized by air in the stomach wall and portal vein due to gas-forming bacteria associated with systemic toxicity, differentiating it from gastric emphysema, a benign condition with an excellent clinical outcome [5]. Pneumatosis intestinalis (PI) affects the small intestine, followed by the large intestine, but rarely are both small and large intestines are involved at the same time [6]. It can be idiopathic (15%) or secondary type (85%) cases [7]. The secondary type is associated with gastrointestinal causes, pulmonary disease, infection, immunocompromised status, and endoscopic procedures in adults [8]. Medications specifically cancer chemotherapy is a well-recognized etiology of PI [9]. PI has been named as pneumatosis cystoides intestinalis pneumatosis coli, intramural gas intestinal emphysema, bullous emphysema of the intestine, pseudolipomatosis, and lymphopneumatosis in the literature [10]. Its etiology is poorly understood but has several proposed theories. According to the mechanical theory, gas dissects into the bowel wall through the luminal or serosal surface through breaks on the mucosa or via tracking along the blood vessels [11]. Once inside the gut wall, it can spread to the distant sites along with mesentery [12]. This mechanical theory is related to surgery, bowel obstruction, trauma, and endoscopy. The bacterial theory proposes that gas-forming bacteria enter through the mucosa and produce gas in the submucosa of the gut. The resolution of symptoms with antibiotics supports this bacterial theory [13]. The biochemical theory proposes intraluminal bacteria produce hydrogen gas from the fermentation of carbohydrates. This gas enters through the mucosa and is trapped in the submucosa [14]. Finally, there is mucosal damage theory; gas enters the gut wall due to mucosal damage caused by a peptic ulcer, pyloric stenosis, and malignancy [15].

Infection-related gastric pneumatosis may develop due to direct bacterial inoculation vs. the hematogenous spread [16]. The common risk factors are alcohol abuse, abdominal surgery, diabetes mellitus, and immunocompromised status and nonsteroidal anti-inflammatory abuse [17]. Commonly involved microorganisms are Escherichia coli, Enterobacter species, Pseudomonas aeruginosa, Clostridium perfringens, Staphylococcus aureus, and rare fungal infections [18]. Ischemia can also be a

rare cause of this conditions, although the stomach has a rich blood supply, but a dramatic increase in intragastric pressure >30 mm of Hg can decrease blood flow leading to necrosis [19]. The patients can present with abdominal pain, nausea or vomiting, diarrhea, and signs of peritonitis, volvulus, hematochezia, bowel necrosis, and pneumoperitoneum and perforation [20]. Immunocompromised patients have a less dramatic presentation [21].

Plain abdominal x-ray film can detect gastric pneumatosis in about two-thirds of affected patients [22,23] and show intramural gas in linear, curvilinear, or circular pattern [24,25]. X-ray abdomen can also detect pneumoperitoneum in 9% of cases [23]. CT scan of the abdomen is the gold standard for the diagnosis, showing air in the greater curvature of the stomach and free air under the diaphragm in case of perforation [26].

Treatment of gastric pneumatosis depends upon the severity and underlying etiology [27]. Most patients are managed conservatively and rarely need surgical intervention [28]. Due to infection, treatment involves intravenous fluid, antibiotics, hemodynamic support, and nasogastric decompression [29]. Endoscopy is helpful for non-infectious causes like trauma, rupture of the pulmonary bulla, or gastric outlet obstruction [30]. Surgery can be considered in patients with signs of peritonitis, portal venous gas, lactic acidosis, metabolic acidosis (PH<7.3, HCO_3< 20, and failure of conservative management [31,32].

Our patient had several risk factors for gastric pneumatosis including chemotherapy, high dose corticosteroids, and infection specifically fungemia [33].

Conclusion

Physicians should be aware of this rare clinical entity. Gastric pneumatosis can be differentiated from gastric emphysema by utilizing a combination of clinical presentation, biochemical testing, and diagnostic imaging. Self-limited gastric emphysema must be differentiated from fatal gastric pneumatosis for early appropriate management.

Declarations

Conflict of interest/financial relationship: All authors have declared that there are neither relationship nor activities that could appear to have influenced the submitted work.

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