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Case Report

A rare cause of acute abdomen in the ED: Chemotherapy-induced pneumatosis intestinalis

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ABSTRACT

Pneumatosis intestinalis (PI) and portomesenteric venous gas (PVG) refers to the presence of air within the intestinal wall and portomesenteric vessels. Most of the time, it is associated with mesenteric ischemia that requires immediate surgical intervention as it has high mortality rate. It may also be seen secondary to various conditions, including infections, surgeries, and some chemotherapeutic drugs. A 61-year old-male was admitted to our emergency department complaining of abdominal pain after chemotherapy. Radiological evaluation of the patient demonstrated massive PVG and PI. Patient underwent urgent surgery due to the possibility of intestinal ischemia and infarction, but no necrosis was identified Chemotherapeutic drug-induced PI and PVG was the final diagnosis. Although PI and PVG are signs of mesenteric ischemia and intestinal necrosis most the of time, chemotherapeutic drugs may also cause PI and PVG rarely. Recent history of chemotheraphy and absence of any mesenteric vascular occlusion may be the diagnostic clue.

1. Introduction

Pneumatosis intestinalis (PI) refers to a rare condition that is characterized by the presence of air in the submucosa or subserosa of the intestinal wall. It may be idiopathic or may be considered secondary to various conditions, including intestinal ischemia or infarction, infections, surgeries, and some chemotherapeutic agents.[1,2] As PI may have serious life-threatening complications, its timely diagnosis is crucial. In this case report, we present a rare case of a chemotherapeutic agent-induced PI in a neutropenic patient after chemotherapy.

2. Case presentation

A 61-year old-male patient was admitted to our emergency department (ED) with complaint of abdominal pain lasting for the last 10 hours. He had two episodes of bilious vomiting after the pain started. He denied any past abdominal surgery or any other medical history, but he had been diagnosed with nasopharyngeal cancer recently and he had received his first session of chemotherapy seven days before admission. The patient's chemotherapy regimen included 140 mg docetaxel and a total dosage of 7000 mg fluorouracil. Physical examination showed generalized abdominal pain, and rebound and tenderness which was predominantly located at the right lower quadrant. On admission, his white blood cell (WBC) count was 310/μL (Normal: 3700–9700/μL) with a neutrophil count of 110/μL. Other blood tests were unremarkable as the laboratory results showed a hemoglobin level of 13.9 g/dL; platelets, 192 × 10^3/mm; sodium, 138 mEq/L; potassium, 3.58 mEq/L; BUN, 21.8 mg/dL; creatinine, 1.1 md/dL. His vital parameters were within the normal limits. Urinalysis was unremarkable. An abdominal ultrasound examination demonstrated

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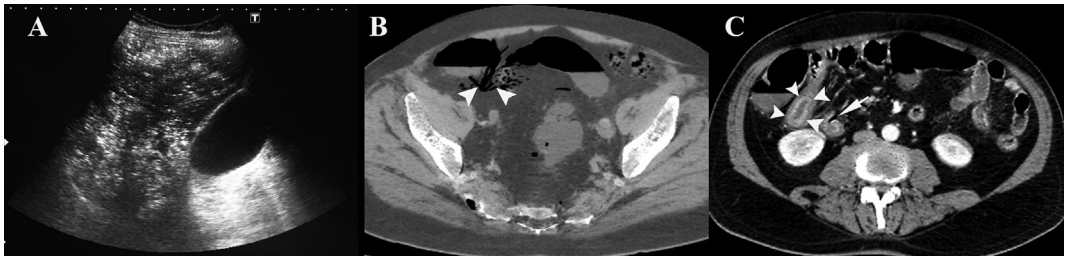

Fig. 1. Ultrasound of the liver demonstrates widespread millimeter-sized hyperechoic particles consistent with air bubbles (A). Abdominal CT images show air bubbles within the mesenteric veins (arrowheads) (B), pneumatosis intestinalis (arrowheads) and air bubbles in the thickened intestinal wall (arrow) (C).

moving hyperechoic air particles within the main portal vein and widespread air particles in the whole liver parenchyma (Fig. 1A). Computed tomography (CT) scan demonstrated small bubbles in the portal vein, at the periphery of the liver parenchyma, and in the mesenteric veins (Fig. 1B). Air bubbles were also observed within the thickened wall of the intestinal segments (Fig. 1C). No detectable arterial or venous mesenteric vascular occlusion was observed. There was no detectable free intraperitoneal air. As the patient had severe abdominal pain, he had an urgent diagnostic laparotomy to exclude intestinal necrosis and related perforation or peritonitis. However, no necrotic intestinal segment was observed. After surgery, the patient had a good response to nasogastric intubation with oral withheld, wide spectrum antibiotics, parenteral nutrition, and IV fluid therapy. Seven days later, the patient was discharged with full recovery. Chemotherapeutic drug-induced PI and PVG was the final diagnosis.

3. Discussion

PI is a radiologic term that defines the presence of small amount of air within the intestinal wall. Ultrasound and plain radiographs can demonstrate the presence of air within the intestinal wall, but cross-sectional imaging with CT is the best diagnostic technique.[3] PI appears as a linear or bubbly pattern of air along the intestinal wall.[4] Accompanying findings such as bowel wall thickening, absent or intense mucosal enhancement, dilated bowel, arterial or venous occlusion, ascites, and hepatic portal or portomesenteric venous gas can increase the possibility of an underlying life-threatening cause.[5] CT is also more sensitive than radiography in demonstrating hepatic portal and portomesenteric gas.[4,6] Kernagis et al.[7] and Weisner et al.[8] found that the presence of an accompanying hepatic portal or portomesenteric venous gas is strongly associated with a transmural infarction.

PI can occur in infants or adults. Most infant cases are secondary to necrotizing enterocolitis. In adults, 15% of PI is idiopathic and 85% is secondary to various conditions.[2,9,10] The most common and life-threatening cause is intestinal necrosis due to mesenteric ischemia or infarction, necrotizing enterocolitis, neutropenic colitis, volvulus, and sepsis.[11] Its exact cause remains speculative, but defective mucosal integrity is proposed to underlie PI. Defects in the mucosa or the gut's immune system may increase the diffusion of air and bacterial invasion into the wall.[2] Attenuation of the barrier may be the result of immunosuppression or some cytotoxic drugs. Several chemotherapeutic agents are associated with PI, including cyclophosphamide, cytarabine, vincristine, doxorubicin, etoposide, docetaxel, irinotecan, and cisplatin.[2] Treatment choices can vary from emergency surgery to conservative therapy. The presence of clinical signs of severe peritonitis, intestinal necrosis, or perforation may be accepted as a surgical indication. Asymptomatic patients and non-ischemic processes may be treated conservatively with broad-spectrum antibiotics and supportive therapy.

Chemotherapeutic agents may rarely cause PI. The underlying mechanism is thought to be the disruption of mucosal integrity by the damage of these agents to the intestinal mucosa. Because the intestinal mucosa is composed of rapidly proliferating cells, it may be very sensitive to chemotherapeutic agents. Decreased submucosal lymphoid tissue in patients receiving chemotherapy may be another underlying mechanism of PI. For this reason, neutropenia may also be an important risk factor for PI.[12] Fluorouracil and docetaxel-associated PI cases have been reported in the literature.[12–14] Cytotoxicity of taxanes is related to mitotic arrest and patients undergoing treatment with taxanes usually develop neutropenia.[15] For this reason, both the disruption of the mucosal integrity and the reduction of lymphoid tissue may be the underlying pathogenesis of PI. Fluorouracil is also thought to have multifactorial effect by reducing the blood flow of the mucosa with its vasospastic and thrombogenic effect.[12] Our patient's treatment regimen included these two drugs, and the patient was also neutropenic. The mechanism of PI was thought to be multifactorial.

The treatment of PI depends on the patient's presenting symptoms and comorbitidity. Clinical deterioration or signs of peritonitis and perforation are accepted surgical indications.[2] Chemotherapy induced PI can be managed with conservative treatment. To prevent any unnecessary surgical intervention, learning about the patient's medical history is crucial. Our patient had a history of recent chemotherapy, and his WBC count on admission was below 500/μL. Chemotherapy-related cytotoxicity and neutropenia could have interrupted the intestinal mucosal integrity. However, he had severe abdominal pain with signs of peritonitis. Therefore, we performed emergency laparotomy. The surgeons could not find any sign of perforation or intestinal necrosis. The patient went on conservative therapy after surgery and was relieved. We suggested to adjust the chemotherapy regimen with other more eligible agents or to follow the patient very closely during the therapy.

In conclusion, we emphasize that keeping in mind the possibility of chemotherapy-induced PI in patients presenting with abdominal pain after chemotherapy in the ED is essential. The patient's medical history has a vital role in the diagnosis. Both surgeons and clinicians should be more familiar with this rare entity, diagnostic clues, and management strategies to prevent unnecessary surgical interventions.

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Conflicts of interest

None declared.

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