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Pediatric

Perforated jejunitis in a child with acute lymphoblastic leukemia treated with pegaspargase

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ABSTRACT

Survival rates of children with acute lymphoblastic leukemia have improved since the incorporation of asparaginase in the treatment protocol, but the medication has potential serious complications, including vascular thrombosis. Here, we describe the case of a 13-year-old boy with pre-T-cell acute lymphoblastic leukemia whose treatment course was complicated by perforated jejunitis requiring resection of a portion of his small bowel. Pathologic assessment showed transmural ischemia, mesenteric venous and arterial thrombi, and scattered cytomegalovirus inclusion bodies. Pediatric mesenteric ischemia is rare, and its consideration in patients treated with asparaginase is discussed.

Introduction

Acute lymphoblastic leukemia (ALL) is the most common cancer diagnosed in children, with an incidence of approximately 3.9% among individuals younger than 19 years and a mortality rate of 0.3 per 100,000 in the United States [1]. Reported toxicities in patients undergoing chemotherapy for ALL are high [2], and gastrointestinal (GI) complications such as diarrhea and neutropenic colitis are particularly common [3]. The long-term survival of patients with ALL has improved substantially since the introduction of asparaginase therapy [4]. However, this therapy can be associated with many serious toxicities, including an increased propensity for thrombosis [5]. We present a case of an adolescent with pre-T-cell ALL who was found to have perforated jejunitis during induction chemotherapy. Pathologic assessment of the resected bowel demonstrated changes associated with transmural ischemia, as well as focal mesenteric venous and arterial thrombi and scattered cytomegalovirus (CMV) inclusion bodies. This case illustrates the complex

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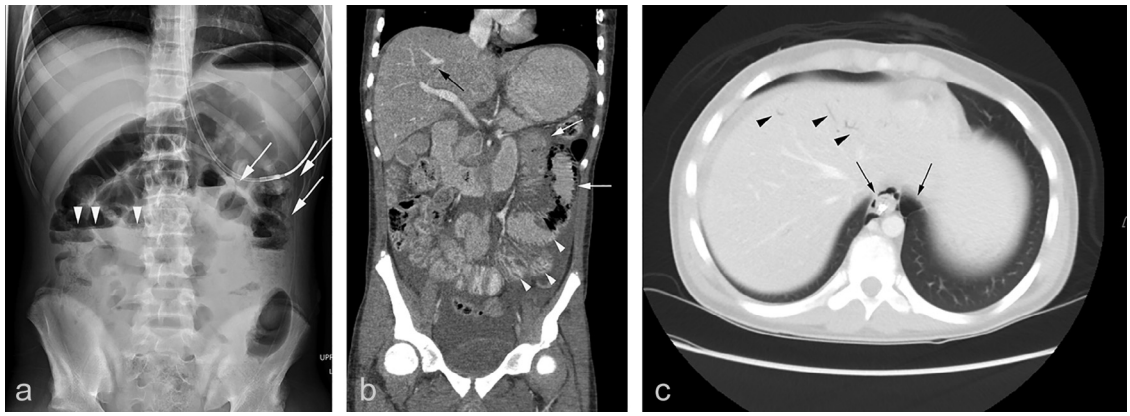

Fig. 1 – Abdominal imaging acquired on day 14 of induction chemotherapy after onset of abdominal pain showing jejunal pneumatosis and dissection of gas through the mesentery into the retroperitoneum and the portal venous system. (A) The upright anterior-posterior abdominal radiograph shows pneumatosis in the left upper quadrant (white arrows), as well as air-fluid levels in mildly dilated loops of bowel (white arrowheads). Contrast-enhanced computed tomography images in (B) the coronal plane with soft tissue algorithm and in (C) the axial plane with lung algorithm shows pneumatosis involving an abnormally dilated segment of jejunum (white arrows in B) and abnormally enhancing mucosa in the jejunum (white arrowheads in B). Abnormal retroperitoneal air is present at the diaphragmatic hiatus (black arrows in C), and portal venous gas is also observed (black arrow in B, black arrowheads in C).

pathophysiology of jejunitis in a child undergoing induction chemotherapy for ALL and allows for a discussion about both mesenteric ischemia and CMV enteritis in children.

Case report

A 13-year-old boy with a recent diagnosis of pre-T-cell ALL presented with severe worsening abdominal pain that began 2 weeks after initiation of chemotherapy. The boy was undergoing induction chemotherapy per the standard arm of Children's Oncology Group study AALL1231 that includes dexamethasone, daunorubicin, vincristine, and intensified pegaspargase (given on days 4 and 18). On day 14 of induction, the patient developed epigastric abdominal pain and was found to have lost 5 kg since his initial ALL diagnosis. Abdominal pain and nutritional status initially improved with optimization of proton pump inhibition and initiation of nasogastric feeds. However, on day 25 of induction, the patient presented to the emergency department with increasing epigastric pain and multiple episodes of nonbilious, nonbloody emesis.

On examination, the patient was afebrile and normotensive. He had left periumbilical tenderness, without peritoneal signs. Significant laboratory results included an absolute neutrophil count of 3100 cells/μL, elevated serum lactate, and worsening metabolic acidosis. Abdominal radiograph (Fig. 1A) revealed multiple air-fluid levels, suggestive of ileus or early bowel obstruction, as well as probable pneumatosis in the left midabdomen. Contrast-enhanced computed tomography (CT) performed in standard portal venous phase confirmed ileus and pneumatosis intestinalis in the jejunum, with associated bowel wall thickening and segments of mucosal hypoenhancement and hyperenhancement (Fig. 1B). Simple-appearing ascites was seen. There was portal venous gas, along with small amounts of ectopic air dissecting along the mesenteric root into the esophageal or periesophageal region of the lower posterior mediastinum (Fig. 1C). Mesenteric vasculature was normal, without obvious CT evidence of arterial or venous thrombosis, although CT angiography was not performed.

Two days after admission (day 26 of induction), the patient developed rebound tenderness, and the abdominal radiographs revealed pneumoperitoneum. A jejunal perforation was found during emergent and exploratory laparotomy. Two separate 30- to 35-cm segments of jejunum with gangrenous necrosis were resected, and the abdomen was left open. Re-exploration and washout of the abdomen were performed 2 days later, along with end-to-end anastomoses of 2 of the 3 jejunal segments and creation of mucus fistulae.

The resected bowel segments (Fig. 2) showed multiple foci of geographic transmural or mucosal necrosis with bile-stained bacteria-rich fibrinopurulent exudates borne between areas of residual mucosa. Several submucosal vessels near the ulcers were thrombosed. Few scattered CMV inclusions were identified in mucosal and submucosal endothelial and stromal cells. Occasional partially occlusive fibrin thrombi were seen within few mesenteric vessels; large feeding arteries were patent.

The patient was treated with postoperative broad-spectrum antibiotics for non-neutropenic enterocolitis and ganciclovir (6 weeks) for CMV colitis. Jejunostomy takedown was uncomplicated, and the patient was discharged from the hospital tolerating full enteral feeds. The end of induction assessment for minimal residual disease (<0.01%) was negative for residual leukemia in the bone marrow.

Discussion

We present a rare case of non-neutropenic jejunal necrosis and perforation, in the setting of immunosuppression by