The presented case was an elderly female, who despite previous history of Hodgkin's disease and treated early bladder cancer, could be considered immunocompetent. However she underwent hepatic resection for malignancy which combined with prolonged ICU stay might have rendered a relative immunocompromised state. Opportunistic viral infections are common and the portal of entry may be the respiratory tract in patients on ventilator leading to viraemia. The patient discussed here had CMV on tracheal aspirate as well as in the serum, quantified by PCR assay.

CMV enteritis may also mimic ischemic colitis and *Clostridium difficile* infection adding to the diagnostic dilemma in a postoperative patient. Negative diagnosis of *Clostridium difficile* should therefore raise the possibility of CMV ileitis in these patients, at least when there is proven infection with the virus elsewhere in the body. The CT images obtained in this patient are classic. In addition to pointing to the potential site of bleeding, concurrent appearance of thickened distal ileum is leading to the diagnosis of ileitis. The radiological images may mimic Crohn's disease, *Clostridium difficile* infection and rarely malignancy.[14] Though the primary treatment for clinically significant CMV infection is antiviral therapy, surgical intervention may be required in complicated cases, as in this case where despite 2 weeks of antiviral treatment with gancyclovir, she required resection for a rare but lethal complication. A high rate of mortality up to 44% is reported when CMV enteritis develops in an immunocompromised patient.[15]

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Late omental gangrene following corrosive ingestion

Introduction

Infarction of the greater omentum is a rare cause of acute abdomen with only few cases reported in the literature. Given

its low incidence and lack of awareness among clinicians, an accurate preoperative diagnosis is unusual and patients may be explored for peritonitis due to other causes. Omental gangrene presenting as a late complication following corrosive ingestion has not been reported in the literature. To our knowledge we report here the first case of omental gangrene occurring nearly three months after corrosive ingestion, presenting as an acute abdomen and requiring surgery.

Case Report

A 40-year old female had ingested corrosive acid 3 months ago, following which she developed abdominal pain associated with vomiting and was managed conservatively. She had no respiratory distress, hemoptysis or hematemesis. She developed dysphagia to solids and semisolids 1 month later. Barium study showed a long esophageal stricture (**Figure 1a**) with a small capacity contracted stomach, and passage of dye into the duodenum (**Figure 1b**). She was planned for an upper gastrointestinal endoscopy and possible dilatation of esophageal stricture. Esophagoscopy revealed a stricture just below the upper esophageal sphincter for which dilatation was attempted. A day after dilatation, patient came with acute onset severe pain abdomen. On examination her pulse rate was 120/min and BP 100/60 mmHg. Abdominal examination showed signs of peritonitis. She was resuscitated with intravenous fluids and intravenous antibiotics were started. Blood gases showed metabolic acidosis. With a pre-operative suspicion of lower esophageal perforation, the patient underwent exploratory laparotomy. Per operative findings revealed dark hemorrhagic foul smelling fluid with gangrenous omentum (**Figures 2a & 2b**) and no evidence of gastric or esophageal perforation. Stomach was small and contracted. Surgical procedure included omentectomy, feeding jejunostomy, lavage and drainage. She was kept on elective ventilation and was extubated after her acidosis was corrected. Postoperatively she developed chest infection which was treated with antibiotics. She was

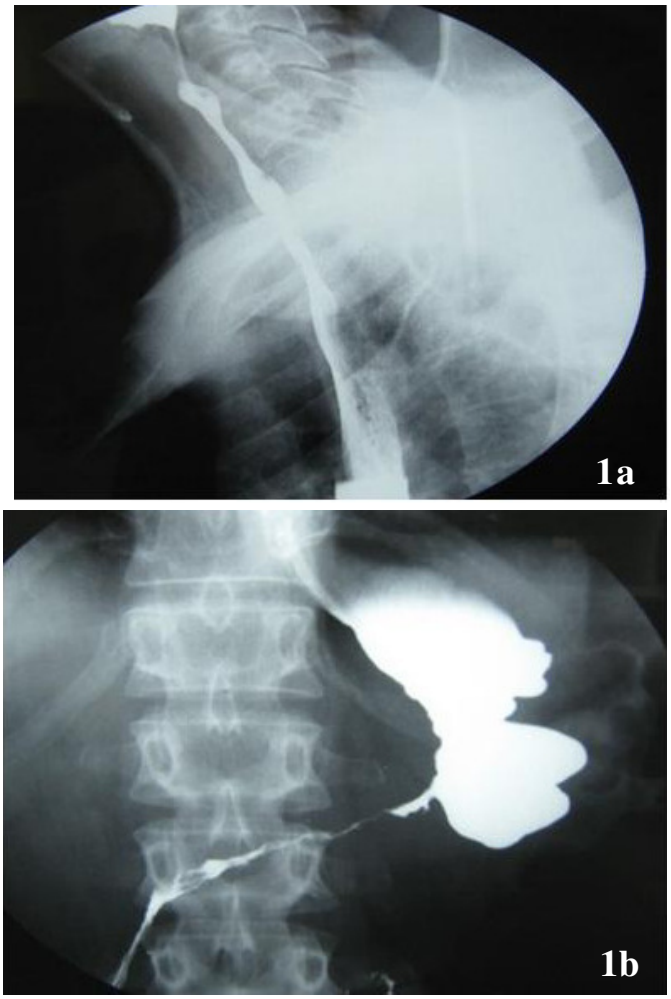

Figure 1: (a) Barium study shows long esophageal stricture (b) shows stomach is small and contracted with dye passing into the duodenum

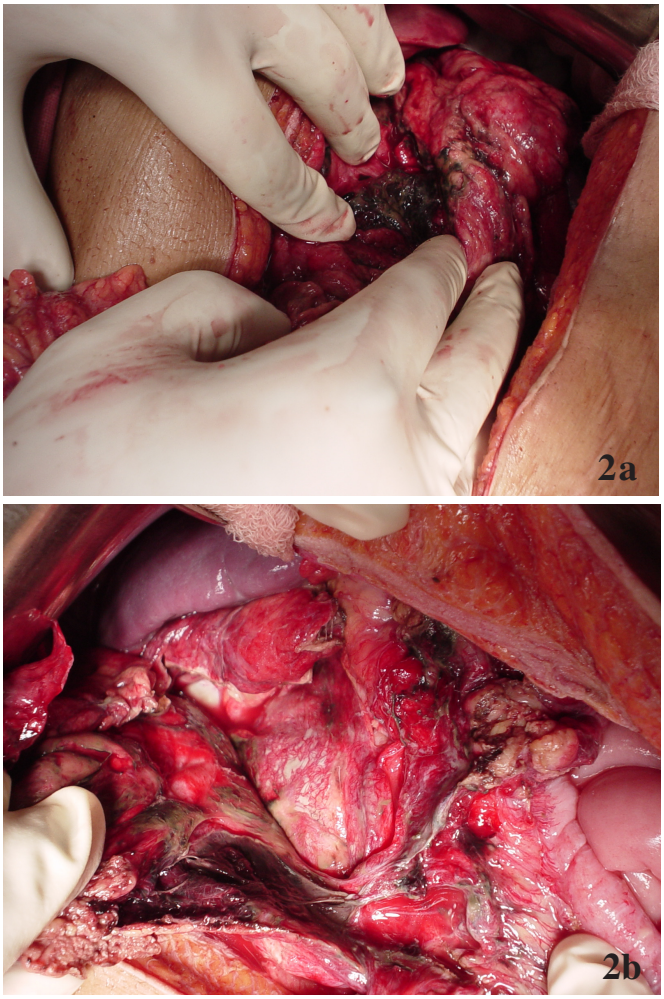

Figure 2: (a) & (b) Per-operative photograph showing omental gangrene

discharged after 2 weeks. Histopathology showed lobule of omental fat showing gangrene with fibrin exudates and few inflammatory cells (**Figures 3a & 3b**). At 3-months follow up she is taking feed through jejunostomy and is being worked up for definitive surgery.

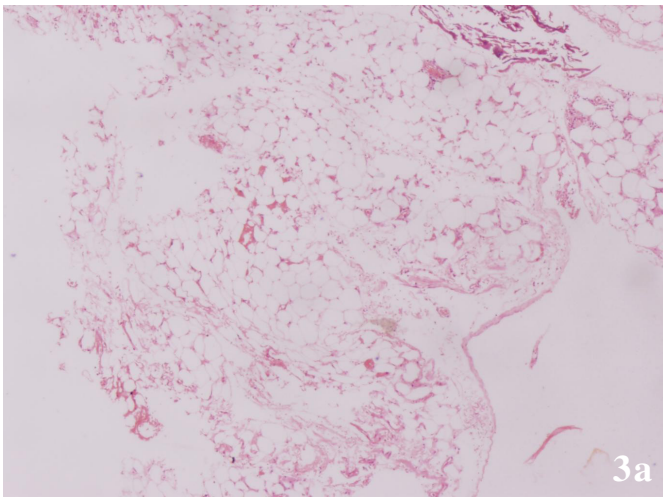

Figure.3a: 10x H & E shows lobules of omental fat showing gangrene. Fibrin exudates and scant inflammatory cells seen

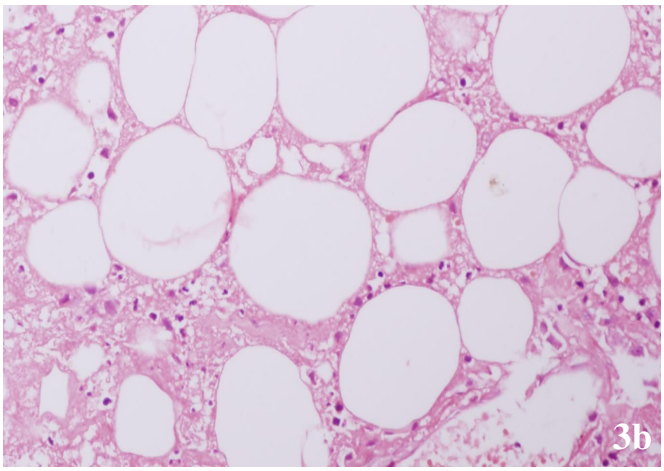

Figure 3b: 20x H & E high power shows dead smudge nuclei of fat cells seen along with sparse inflammation and fibrin

Discussion

Primary omental torsion was first reported by Eitel in 1899 in which a bulky and mobile greater omentum particularly on the right side twists around the distal right epiploic vessels with subsequent venous occlusion, edema and arterial thrombosis leading to gangrene. Johnson[1] in 1932 first described, primary omental infarction without torsion. The reported incidence is 0.1% of patients undergoing laparotomy for appendicitis.[2] The incidence has been rising attributed to increased awareness of the entity, technical advancements in imaging and utilization of CT scan for evaluation of abdominal pain.

The exact etiology is not known. Predisposing factors reported in the literature include variations in arterial supply to the right side of the greater omentum, excess omental fat associated with obesity (explaining its greater incidence in adults), hypercoagulability, trauma, straining, vascular congestion secondary to overeating, overexertion, and sudden positional change.[3] Omental infarction in adults is rarely described following intra-abdominal surgery, after marathon running, with right-sided heart failure, or as a complication of vasculitis[4].

To our knowledge this is the first case in which corrosive ingestion has been the pathophysiological mechanism for the etiology of late omental infarction. The reason for late development of omental gangrene following corrosive intake is not known, but could possibly be related to the development of thrombosis of the omental vessels resulting from inflamed contracted stomach following corrosive ingestion.

The right side of the omentum is affected in the majority of patients leading to right sided abdominal pain. The right-sided predilection is usually attributed to anomalous right omental vascular development.[5,6] It was predominantly left sided in our case, probably related to the etiology of gangrene. Other symptoms include low grade fever and occasional vomiting. Leukocyte count may be mildly elevated.

Characteristic features[7-8] of omental infarction on imaging may help in making a preoperative diagnosis in suspicious cases. The omental infarct on CT scan appears as an ovoid area of high attenuated fat containing hyperattenuated streaks just beneath the parietal peritoneum with thickening of the overlying anterior abdominal wall. Ultrasound scan shows the omental infarct typically appearing as a hyperechoic, non-compressible, ovoid intra-abdominal mass adherent to the anterior abdominal wall with localized point tenderness.

Omental infarction usually follows a benign course that allows conservative management. Spontaneous and complete resolution of symptoms typically occurs within 2 weeks. However non-operative treatment has a risk of leaving the necrotic tissue inside, resulting in prolonged convalescence due to gangrene or abscess formation. Omental abscess and bowel obstruction are the delayed complications of omental infarction.

Surgery is indicated in patients who have worsening of symptoms on conservative management, in whom omental gangrene result in sepsis and when there is diagnostic dilemma. Surgery usually results in early resolution of symptoms with little morbidity. Histology shows hemorrhagic infarction

associated with fat necrosis, followed by cellular infiltration, fibrosis and scar formation.

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Rectal redundancy leading to intestinal obstruction in a boy with Ehlers-Danlos syndrome

Introduction

Ehlers-Danlos syndrome (EDS) is a connective tissue disorder characterized by skin extensibility, abnormal wound healing and joint hyper mobility.[1-4] The striking clinical features are hyper elastic smooth skin that is prone to splitting with minor trauma, easy bruising, spontaneously resolving dislocations of joints, scoliosis and ocular features such as keratoconus depending on the type of EDS.[1-8] Multiple hernias are usually a rule rather than exception in these patients. However, the abdominal symptoms are very rare presenting features of this disease.

We have treated a boy suffering from EDS with multiple hernias and poor wound healing. The unique feature in this child was rectal redundancy that caused intestinal obstruction and generalized abdominal distension, both of which got resolved after partial resection of the redundant rectum. No similar case was found in the literature.

Case Report

A one month old baby presented with right inguinal hernia, left undescended testis with hernia and supraumbilical hernia. Ten days following the repair of right inguinal hernia and supraumbilical hernia, he presented with features of obstructed left inguinal hernia for which a left inguinal herniotomy with left orchidopexy (as he had left undescended testis as well) was done. It was a large sliding hernia with appendix and a floating caecum. Post-operatively, the baby developed bronchiolitis that progressed to pneumonia and had to be managed in pediatric ICU.

The baby developed scrotal wound dehiscence on the 7th post-operative day, without any gross evidence of predisposing factors. However, with supportive management, the wound healed with secondary intension. A week later the baby developed generalized abdominal distension. Plain abdominal X-rays showed an even distribution of the gas throughout the gastro-intestinal tract up till rectum, with no cut off of gas (**Figure 1**). There was no vomiting but the patient produced high bilious aspirate through the nasogastric tube. Septic screen was negative and bowel sounds were normal. Rectal examination was normal and on direct questioning, the mother gave past history of similar episodes of spontaneously resolving abdominal distension. There was no history of similar illness in the family. The distension decreased once the flatus tube was passed rectally.