Emphysematous gastritis causing gastric and esophageal necrosis in a young boy

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Abstract

Emphysematous gastritis is a rapidly fatal and rare type of infectious gastritis. It may lead to involvement of esophagus, and organ necrosis, in its severe form. A 16-year-old, previously healthy, boy presenting with acute abdomen was diagnosed to have emphysematous gastritis on CT scan. During laparotomy, there was complete necrosis of the stomach, with patchy esophageal involvement. Aggressive management in the form of total gastrectomy, and later, transthoracic esophagectomy was done. However, it failed to alter the course of the illness, and the patient succumbed to the illness. Emphysematous gastritis is rare in young patients without known risk factors. Also, only two previous cases have been reported with esophageal involvement. We have presented this case with a brief review of literature. (Acta gastroenterol. belg., **2009**, 72, **354-356**).

Key words : emphysematous, gastritis, esophagitis, necrosis, gangrene, empyema.

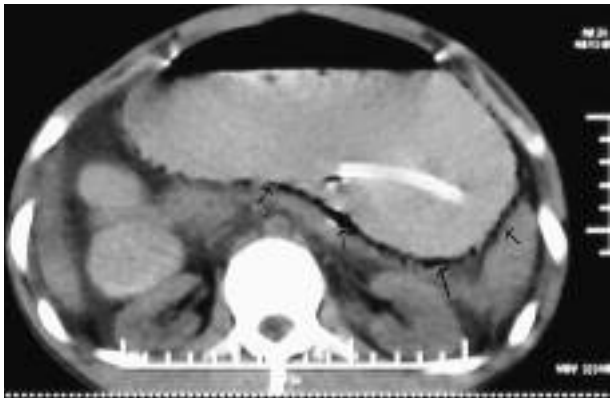

Fig. 1. — CT scan showing intramural gastric air (black arrows).

Introduction

Emphysematous gastritis is a rare, highly fatal, infectious condition, characterized by gas formation inside the gastric wall. In some cases, necrosis of the stomach may ensue (1,2,3). Esophageal involvement with subsequent necrosis is even rarer, and, has been reported in only two cases previously (1,4). We present the case of a young boy presenting with emphysematous gastritis and esophagitis, who succumbed to the illness despite aggressive treatment. The literature on the topic has also been reviewed. To the best of our knowledge, this is only the third such case reported so far.

Case report

A sixteen year-old boy, a motor workshop assistant, presented with severe upper abdominal pain, vomiting and fever of one day's duration. No previous history of significant illness, or substance abuse was elucidated. On examination, he had tachycardia and hypotension, with tenderness and guarding mainly in the epigastric region. Blood investigations revealed leucocytosis, with raised serum amylase (1890 U/L) and serum lipase (2540 U/L) levels. Other hematological and biochemical parameters were normal. Bilateral pleural effusion was evident on chest X-ray. Clear fluid was aspirated from bilateral pleural cavities. Contrast enhanced CT abdomen showed diffuse and mottled intramural gastric air with massive dilatation of the stomach (Fig. 1). There was no pneumoperitoneum, and the pancreas was normal. Supportive treatment was initiated, along with broad-spectrum antibiotics. The worsening clinical condition led to suspicion of visceral necrosis, and prompted to exploratory laparotomy, 20 hours after admission. The operative findings were huge dilatation and complete necrosis of the stomach, extending onto the anterior wall of the abdominal esophagus (Fig. 2). The pancreas and other viscera were grossly normal. Total gastrectomy and transhiatal abdominal esophagectomy was performed. Continuity of bowel was not restored due to the poor general condition of the patient. Postoperatively, the patient did not improve, and developed left-sided empyema, which was drained by thoracostomy. Bedside esophagoscopy revealed mucosal necrosis in the thoracic esophagus, distal to 25 cm from the incisors. Right posterolateral thoracotomy and esophagectomy till the level of carina was performed to remove the necrotic segment. However, the patient continued to deteriorate, and died on the 8th postoperative day following multi-organ failure. Histopathological examination of the resected stomach and esophagus showed a non-specific transmural necrosis with infiltration of neutophils. By the Sydney

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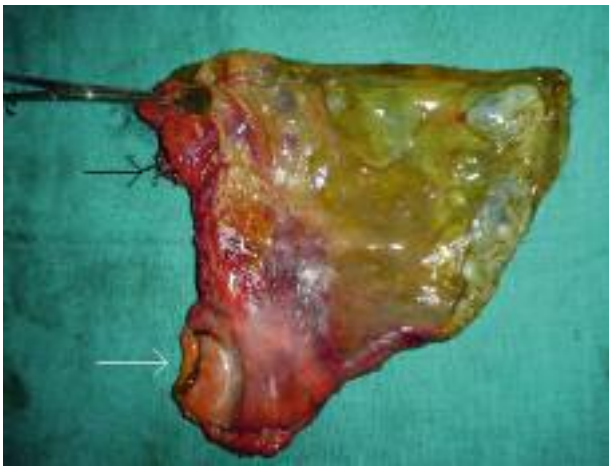

Fig. 2. — Dilated and necrosed stomach ; esophageal end (black arrow), antral end (white arrow) ; the resected specimen.

classification, there was acute gastritis (histomorphologic criteria), and Type B or bacteria-related gastritis (etiologic criteria). Fungal cultures form the viscera were negative.

Discussion

Emphysematous gastritis is a rare infection caused by gas-forming organisms which presents with acute systemic toxicity, and has a rapidly fatal course. Commonly implicated predisposing factors are corrosive ingestion, alcohol abuse, immunosuppressed states (including diabetes), abdominal surgery, acute pancreatitis, acid peptic disease, gastric outlet obstruction, neoplasms, renal failure, systemic mycosis, and, trauma (2,3,5). Invasion of the weakened gastric mucosal mechanisms by virulent bacteria, most notably *Clostridium* species is believed to be the immediate cause. The presentation is most often that of an elderly patient with an underlying predisposing factor, developing subtle or overt signs of abdominal sepsis. In children, gastric necrotizing infections have been reported in about 15 cases. Most have been seen in early infancy (1-5,6,7). The common predisposing factors in this subgroup are gastric outlet obstruction, and associated necrotizing enterocolitis. Only one healthy adolescent patient has been reported earlier with emphysematous gastritis, presumably by the ingestion of large quantities of aerated drinks (7). Our patient was also a healthy adolescent male with no obvious associated risk factor, who presented primarily with an acute abdomen. Despite raised biochemical markers like serum amylase and serum lipase in our patient, we cannot attribute acute pancreatitis as the causative factor, because the pancreas was found to be normal on CT scan and during the operation. Without pancreatic necrosis, it appears improbable that emphysematous gastritis with gangrene would develop so rapidly. Fungal studies were negative in our case, although mucormycosis has been implicated in a few cases (8,9).

The diagnosis of emphysematous gastritis is made after CT scan, by demonstration of intramural stomach gas bubbles. The clinical picture and the mottled gas pattern can readily differentiate this condition from benign gastric emphysema, which is characterized by the presence of air in the stomach wall following instrumentation (10,11). Endoscopy can aid the diagnosis by showing patchy or confluent mucosal necrosis. In patients presenting with signs of peritonitis due to gastric gangrene, diagnosis may be first evident only at laparotomy.

The treatment involves early institution of broad-spectrum antimicrobial therapy, with appropriate supportive measures for endotoxic shock. Many patients have been shown to respond to conservative treatment alone (2,5,12). Mortality, however, is greater than 60% (5). Laparotomy with gastrectomy and debridement, when indicated, seldom helps to halt the progression of septicemia to multi-organ failure. Concomitant esophageal involvement with the gas-forming process, though plausible, has been reported only twice earlier (1,4). In the first instance, a 76-year-old man with a history of alcohol intake and ingestion of nonsteroidal anti-inflammatory agents was diagnosed with diffuse emphysematous gastroesophagitis, and rapidly succumbed to the disease (4). Miller and colleagues reported the second case in a 57-year-old healthy male liver donor, who developed emphysematous gastritis with partial involvement of the lower esophagus in the postoperative period (1). Sud *et al*. (13), from Chandigarh, India, have reported a case of emphysematous gastritis with empyema. Empyema was also observed in our patient during the course of treatment. We believe that esophageal involvement may represent an advanced form of the disease. Also, empyema may herald the onset of, or, accompany the mediastinitis and esophageal necrosis in these patients. It may be inferred that the risk factors for concomitant esophageal involvement could be advanced age, ongoing septic process, or late diagnosis. But more cases will be needed for further study of this rare condition.

Emphysematous gastritis is a rare acute illness, which is difficult to diagnose and treat, due to its rarity and its presentation with the common symptoms of acute abdomen. The atypical features in our case were the occurrence in a young adolescent with no apparent predisposing factors, and the associated esophageal involvement.

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