

Emergency Operation for Phlegmonous Gastritis

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ABSTRACT

Phlegmonous gastritis is a rare inflammatory lesion in which bacterial infection occurs in the gastric wall. A case of phlegmonous gastritis producing an intramural filling defect in the stomach is presented. Endoscopy showed edematous and reddened gastric mucosa with a mass lesion in the gastric body and antrum. An abdominal CT scan showed diffuse and irregular thickening of the gastric wall. At emergency operation, a total gastrectomy with splenectomy was performed. The most important differential diagnosis is carcinoma, especially scirrhus-type gastric cancer. Radiographic findings of phlegmonous gastritis resemble those of scirrhus gastric cancer. More frequent recognition of this disease, early diagnosis and prompt institution of treatment is essential.

INTRODUCTION

Phlegmonous gastritis is a rare inflammatory lesion of the gastric wall. Although the disease usually shows characteristic macroscopic and histological findings¹, the diagnosis may contribute to problem and it is sometimes difficult to distinguish phlegmonous gastritis from scirrhus gastric cancer. In this report we describe a case of phlegmonous gastritis with an acute clinical course where emergency operation was necessary.

CASE REPORT

A 74-year-old woman was sent to the Sendai Medical Center in June 2002 because of abdominal pain and vomiting of one-week duration. On admission, the patient was drowsy. Physical examination revealed tenderness in her upper abdomen with sluggish bowel sounds. Blood pressure was 130/65 mmHg, and pulse rate was 55

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beats/min and regular. Her temperature was 38.5°C. Laboratory examination showed a white blood cell count of $28.3 \times 10^9/L$; hemoglobin of 129g/L; normal serum sodium, potassium and chloride levels; serum calcium level of 5.5 mmol/L; serum phosphorus level of 1.9 mmol/L blood urea nitrogen of 2.9 mmol/L; and a serum creatinine level of 0.8 mg/dl. Liver enzymes were normal. The patient was aggressively treated with fluid resuscitation. One day after the start of treatment, serum calcium level had decreased to 2.5mmol/L. An abdominal CT scan showed diffuse and irregular thickening of the gastric wall (Fig. 1a). An upper gastrointestinal series showed narrowing of the antrum and pylorus involving almost the entire gastric cardia, mimicking Borrmann type 4 gastric cancer of “linitis plastica” (Fig. 1b). At gastroscopy, the stomach mucosa had a thinned atrophic appearance, with numerous white nodules scattered irregularly throughout the body and fundus of the stomach. The nodules varied in size, with the largest being approximately 2-3 mm in diameter. A stricture was observed in the stomach body (Fig. 1c). The patient’s symptoms worsened during the next 5 days, and she developed nearly complete gastric outlet obstruction secondary to an annular constriction of the antrum. No ulceration was seen. Examination of a biopsy specimen from the gastric mucosa revealed severe inflammation. Since an upper GI series demonstrated typical features of Borrmann type 4, we suspected that it was cancerous lesions. An emergency operation was performed. On laparotomy, a total gastrectomy with splenectomy was performed. Adhesion between the stomach and spleen was very strong, and we could not separate them. It was easier to resect the stomach and spleen as one organ. The stomach wall was thickened and edematous with diffuse ulceration. Microscopic examination showed the gastric mucosa to be infiltrated by lymphocytes. The submucosa was edematous and infiltrated by inflammatory cells with a focal area of necrosis (Fig. 1d).

DISCUSSION

Very little is known about the pathogenesis of phlegmonous gastritis. In most cases, hemolytic streptococcus is reported to be the offending organism (1). There are three possible routes of spread: 1) direct injury to gastric mucosa due to sharp bones or other hard food, 2) hematogenous spread from some other septic focus such as endocarditis, and 3) lymphatic spread from a septic focus. Some researchers produced a submucosal abscess in dogs by introducing streptococci into the mucosal surface after induction of alcohol gastritis, but others failed to produce a similar condition¹. Until more is known about the mechanism of gastric defense in response to bacterial invasion, the pathogenesis of this disease will remain unclear. Patients with this disease present with an acute abdomen, with intense epigastric pain of relatively rapid onset associated with epigastric guarding (2). Leucocytosis and nausea and vomiting are present. In the most fulminant varieties, the patient develops severe toxemia and early peripheral circulatory collapse (3). There are few descrip-

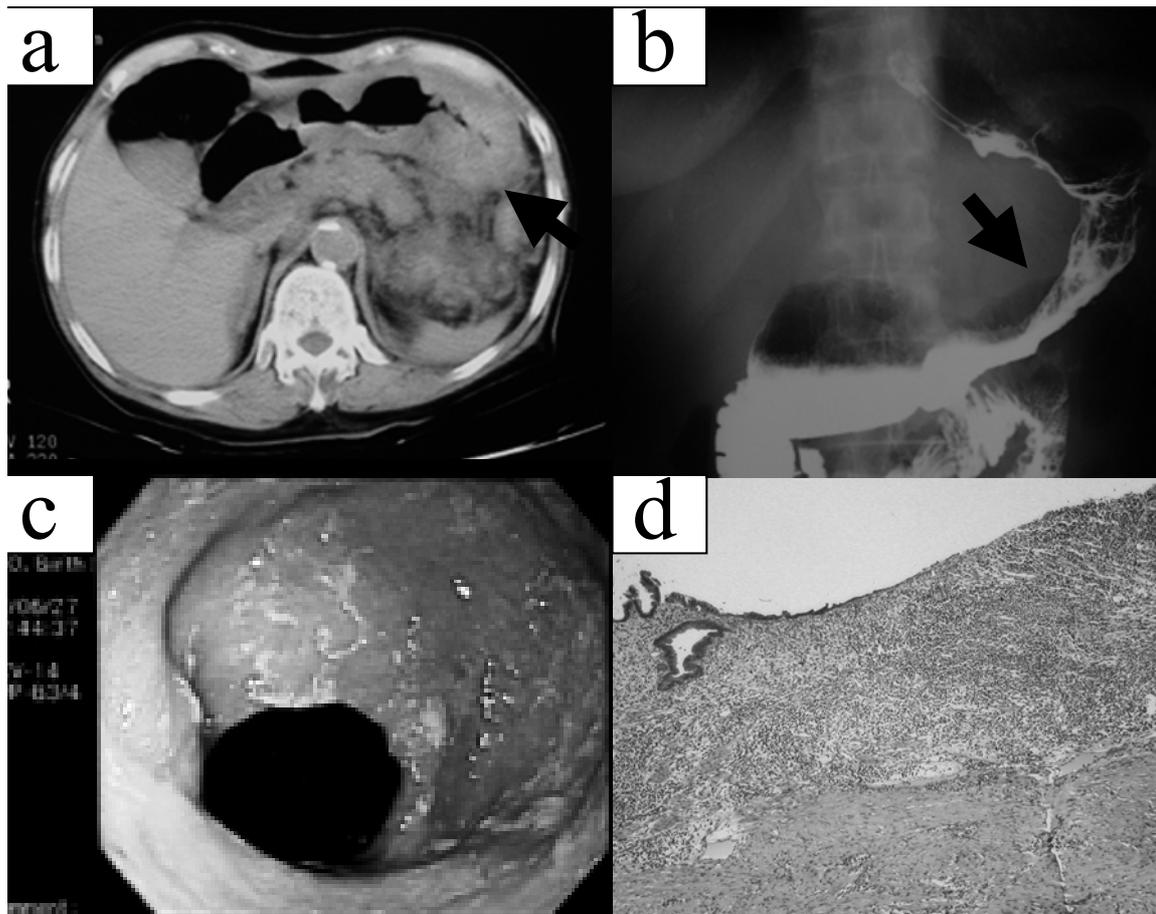


Figure 1a: Abdominal CT showed diffuse and irregular thickening of the stomach body wall (arrow). b: Barium examination of the upper gastrointestinal tract revealed a marked large irregular filling defect involving the entire gastric wall (arrow). c: Endoscopy showed edematous and reddened gastric mucosa with a stricture in the gastric body. d: A gastric section showing diffuse inflammatory swelling of submucosa with involvement of the muscularis. (original magnification, x100)

tions of radiographic findings of phlegmonous gastritis (4). The common features seem to be a relatively small intramural mass, often in the antrum of the stomach, usually without destruction of the overlying mucosa. The mucosal folds are completely effaced at the site of involvement. There is frequently gastric retention of barium, and complete pyloric obstruction has been reported. The most important differential diagnosis is carcinoma; actually, the preoperative diagnosis in our patient was scirrhous-type gastric cancer in spite of a “negative” biopsy. Scirrhous type carcinoma cells invade extensively under the mucosal layer, and results of biopsy examination are sometimes negative. Radiographic findings of phlegmonous gastritis resemble those of scirrhous gastric cancer. Scirrhous gastric cancer is clinically characterized by diffuse infiltration by cancer cells and shows significant thickening of the gastric wall with no prominently elevated or depressed lesions (5). The infiltrative carcinoma may grow either superficially over the surface of the mucosa or permeate the entire thickness of the wall, producing a pattern known as linitis plastica. A submucosal biopsy has potential diagnostic benefit.

There is a lack of agreement about the role of surgery in the management of acute phlegmonous gastritis, some authors recommending antibiotic therapy alone and others recommending gastrectomy or gastrostomy for drainage of submucosal abscesses¹⁻³. Early diagnosis and prompt institution of treatment with or without surgery are essential for this disease.

REFERENCES

1. Miller AI, Smith B, Rogers AI (1975) Phlegmonous gastritis. *Gastroenterology* 68: 231-238.
2. Iwakiri Y, Kabemura T, Yasuda D (1999) A case of acute phlegmonous gastritis successfully treated with antibiotics. *J Clin Gastroenterol* 28: 175-177.
3. Webster VJ (1980) Necrotizing gastritis and phlegmonous gastritis-are they separate entities? *Aust NZ J Surg* 50: 194-196.
4. Sood BP, Kalra N, Suri S (2000) CT features of acute phlegmonous gastritis. *Clin Imaging* 24: 287-288.
5. Yokota T, Teshima S, Saito T (1999) Borrmann type IV gastric cancer: Clinicopathological analysis. *Can J Surg* 42: 371-376.

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