Gastric wall abscess presenting as thoracic pain: rare presentation of an old disease

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Abstract
Acute phlegmonous gastritis, a suppurative infection of the gastric wall, has a high mortality rate. This disease remains a mystery to most clinicians. The rarity of this disease and its nonspecific presentation make early diagnosis difficult, especially in the emergency department. We describe a case of acute phlegmonous gastritis presenting as gastric wall abscess in a 55-year-old woman who visited the emergency department, illustrating the severity of this disease, the difficulty in its diagnosis and its unusual presentation.

Keywords Suppurative gastritis, gastric abscess, CT scan

Ann Gastroenterol 2013; 26 (4): 360-362

Introduction
Suppurative gastritis (SG) is an uncommon, often fatal condition characterized by suppurative bacterial infection of the stomach arising from a local or disseminated hematogenous infection. SG can occur in a diffuse, localized, or mixed form. The most common type, the diffuse form, is characterized by initial involvement of the gastric submucosa with posterior extension to all layers of the gastric wall, resulting in extensive gangrene of the stomach and the much rarer intramural localized gastric abscess. We report a case of acute phlegmonous gastritis and discuss the diagnosis and treatment of this rare disease.

Case report
A 55-year-old patient presented to the emergency department with a 48-h history of pain in the lower left hemithorax with pleuritic characteristics. Two days before she experienced vomiting and diarrhea. At arrival, she complained of moderate pain in the left lower hemithorax. The pain was characterized as dull, constant, radiating to the left upper abdominal quadrant, aggravated by body posture changes without any alleviating factors, and accompanied by nausea and low-grade fever. Her medical history was unremarkable except for previous appendectomy. The patient did not take any medications, and she was not a smoker or an alcohol consumer. She looked ill with a blood pressure of 123/88 mmHg, a pulse rate of 84 beats/min and a temperature of 38.5°C. A thorough physical examination revealed hypoventilation in the left lower base without other relevant findings. The laboratory examination was unremarkable except for polymorphonuclear leukocytosis (total leukocyte count, 18000/mL; neutrophils, 86%) and an elevated C-reactive protein (86 mg/dL; reference range, 0-5). An upright chest radiograph demonstrated elevation of the left hemidiaphragm and free gas under the diaphragm (Fig. 1).

Abdominal contrast-enhanced computer tomography (CT) scan showed thickening of the posterior wall of the

Figure 1 Chest X-ray showing elevated left hemidiaphragm and subdiaphragmatic air-fluid level
gastric fundus with a thick wall collection that measured 58x41 mm with intraluminal growth suggesting gastric abscess (Fig. 2 A).

The patient was treated by endoscopic drainage of the abscess. The lesion was opened with a cystotome following drainage of pus. Aspirate cultures were positive for *Streptococcus pyogenes*. Antibiotic treatment with amoxicillin / sulbactam (1,000 mg/125 mg q.i.d.) and amikacin (500 mg b.i.d.), both intravenously for 14 days was started. Investigations for fungi, mycobacteria and parasites were negative. Histopathologic diagnosis was acute phlegmonous gastritis. Blood cultures were positive for *Streptococcus pyogenes*. The clinical course was uneventful, and the patient showed a significant and prompt recovery. The patient was discharged the tenth day of admission. One month later, esophagogastroduodenoscopy, and abdominal CT (Fig. 2 B) were performed showing complete resolution of the process.

**Discussion**

SG is a rare bacterial infection of the submucosa and the muscularis propria of the stomach, a rare entity with approximately 500 cases reported in the literature. It is widely accepted that the stomach is relatively protected from infection due to vascular supply and the antimicrobial environment provided by gastric acidity. Acute phlegmonous gastritis can progress quickly, and can be fatal in two-thirds of patients. Two types of SG have been described in the literature: a diffuse or phlegmonous variant type of SG as opposed to a localized or intramural gastric abscess. The true prevalence of acute SG is unknown. The infection occurs primarily within the gastric submucosa and spreads horizontally. Suppuration progresses vertically into the muscularis and serosa, sparing the gastric mucosa until late in the disease. The localized abscess of the gastric wall is another variety of SG [1], a purulent inflammatory process involving the gastric wall that can occur in diffuse, localized, and mixed forms. A review of English language publications identified only 21 reported cases of gastric abscess [2,3]. The localized form accounts for 5 to 15% of all cases [3]. The exact pathogenetic mechanism of SG is unknown [4,5]. Risk factors can be identified in nearly 60% of patient and they include increased age, mucosal injury (chronic gastritis, peptic ulcer disease, and endoscopic injury), hypoacidity (post-vagotomy and gastric resection), malnutrition and immunosuppression (alcoholism, HIV infection). The SG is secondary to an infection (local or disseminated) of the stomach wall by gram-positive cocci (mostly alpha-hemolytic *Streptococcus*) [4], which are not always isolated from routine microbiological cultures [4,6]. Other etiologic organisms in decreasing order of frequency are *Staphylococcus* spp, *Escherichia coli, Haemophilus influenzae, Proteus, Clostridia*, while mixed bacterial infections are documented in 30% of cases. The classic clinical symptoms of SG are upper abdominal pain, high fever, epigastric pain, nausea and vomiting. In most cases, epigastric abdominal pain and nausea dominate the clinical picture. Unusual clinical presentations including purulent emesis and Deininger’s sign (decreased abdominal pain on changing from the supine to sitting position). The symptoms may be present all day long although they may take several days to develop [4-7]. SG progresses rapidly, often leading to a fatal infection unless recognized early. The SG must be differentiated from gastric carcinoma, MALT lymphoma, gastrointestinal stromal tumor, leiomyoma or carcinoid tumor [4,6].

Laboratory examination is not helpful, hemogram usually demonstrates leukocytosis with a left shift. Changes in acute phase markers (elevation in C-reactive protein and erythrocyte sedimentation rate; decrease in albumin) may also be seen. The rarity of this disease and its nonspecific presentation make early diagnosis difficult. Plain abdominal X-rays are abnormal in 50% of cases and findings include paralytic ileus, edematous gastric folds, elevation of the left hemidiaphragm and free gas under the diaphragm. Upper gastrointestinal series may show a filling defect suggestive of submucosal mass. On CT, the lesion appears as a localized...
area of mural thickening within the stomach wall, while fluid and air may also be seen within the mass [8]. Intramural gastric abscess is diagnosed with increasing frequency by endoscopic ultrasonography [2,3,9]. On ultrasound, it appears as a well-defined hypoechoic mass within the gastric wall with increased vascularity around the mass on the color Doppler images [1]. Although the ability of EUS to evaluate phlegmonous gastritis is not established it may be helpful in differential diagnosis as it is the best examination to delineate the gastric wall. The recommended therapy for an intramural gastric abscess is surgical drainage in combination with antibiotics. However, technical advances now allow both radiologic and endoscopic intervention. Antibiotics are the mainstay of treatment. Drainage, surgical or endoscopic, is helpful and occasionally necessary and may reduce hospitalization time [2,3,9,10].

In conclusion, the diagnosis of intramural gastric abscess requires a high index of suspicion because of its rarity. CT images and patient risk factors should make the clinician think of a gastric abscess as a cause of acute abdominal or even thoracic pain. Early diagnosis is important, as it may obviate a needless gastrectomy and even death.

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