Phlegmonous Gastritis: A Report of Three Cases with Clinical and Imaging Features

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Abstract:
Phlegmonous gastritis is a rare but often fatal acute pyogenic infection of the stomach. We herein report three cases of phlegmonous gastritis with different causes: the long-term placement of a nasogastric feeding tube, bacteremia associated with cellulitis in a diabetic patient, and an adverse reaction to paclitaxel/carboplatin chemotherapy for cancer of unknown primary cause, which were classified as primary, secondary, and idiopathic types, respectively. Coping with the increasing morbidity rate associated with the diverse background of such patients requires a thorough understanding of the clinical features and image findings associated with this entity.

Key words: phlegmonous gastritis, nasogastric feeding tube, cellulitis, paclitaxel/carboplatin chemotherapy

Introduction
Phlegmonous gastritis is an uncommon but often fatal acute pyogenic infection of the stomach. Although the mortality rate has been reduced with the improvement of antibi-otic therapy, a majority of patients are still diagnosed after gastrectomy or autopsy (1, 2). According to most previous reports, mucosal injury due to gastric cancer or peptic ulcer is regarded as the main etiology of phlegmonous gastritis. However, a diverse background of these patients has been noted in recent reports, suggesting that the range of clinical features and image findings of this entity should be noted. We herein report three cases of phlegmonous gastritis with different causes: the long-term placement of a nasogastric feeding tube, bacteremia associated with cellulitis in a diabetic patient, and an adverse reaction to paclitaxel/carboplatin chemotherapy for cancer of unknown primary cause (CUP).

Case Reports
Case 1
Early in November 2017, an 84-year-old woman with an advanced stage of dementia was transported to our emergency room because of the obvious flow of fresh blood from her nasogastric feeding tube. Her temperature was 37.4°C, and the laboratory findings showed an elevated serum C-reactive protein (CRP) level of 13.6 mg/dL. Emergency esophagogastroduodenoscopy (EGD) revealed extensive ulceration with necrotic tissue and an edematous mucosa (Fig. 1A and B). Subsequent abdominal computed tomography (CT) showed a thickened gastric wall with an intramural low-density area thought to be an abscess. Proteus mirabilis and α-streptococcus were isolated from a culture of gastric juice and biopsies. Following a diagnosis of phlegmonous gastritis, sulbactam/ampicillin (3 g/day) was started intravenously. One week later, EGD was performed again and showed a significant improvement (Fig. 1C and D). Four weeks later, we confirmed the complete healing by EGD (Fig. 1E and F), and the patient was discharged from our hospital.
Case 2

A 44-year-old man presented with a 4-day history of nausea and epigastric pain early in April 2017. He had a history of uncontrolled type 2 diabetes for 10 years and had undergone treatment for diabetic foot gangrene. As an increase in the white blood cell (WBC) count ($12.1 \times 10^9$ cells/L) and CRP level (8.93 mg/dL) was observed, chest-abdomen-pelvis CT was conducted, and a thickened gastric wall with an intramural low-density area were detected (Fig. 2A). Subsequent EGD revealed markedly thickened gastric folds and redness (Fig. 2B) that appeared to be normal after the 9-day intravenous administration of ceftriaxone (2 g/day). Notably, in this case, Staphylococci were isolated from a culture of both gastric biopsies and diabetic gangrene. The following month, he developed diabetic cellulitis of the leg and bacteremia caused by Staphylococci again (Fig. 2C), but abdominal pain did not recur.

Case 3

The patient was a 64-year-old man introduced to our hospital at the beginning of March 2017 due to CUP. His fam-
ily doctor initially found a brain tumor as the cause of his six-month history of gradually deteriorating numbness in the right hand. Whole-body CT in our hospital revealed multiple lesions in the lung, liver, ribs, and lymph nodes. The upper/lower gastrointestinal tract was intact at this time. On consideration of the histological findings, including immunohistochemistry from a right axillary lymph node biopsy, poorly differentiated lung adenocarcinoma was highly suspected. First-line treatment with paclitaxel (200 mg/m²) and carboplatin (area under the curve 5) every 3 weeks was started. Four days after the first administration of chemotherapy, nausea and hematemesis occurred. Emergency EGD showed a dark-purple, friable, and thickened mucosa (Fig. 3A) that improved after the 7-day intravenous administration of ceftazidime (2 g/day). Air in the thickened gastric wall with concomitant portal venous pneumatosis was seen on CT at onset (15, 16). The white arrow indicates abscesses in the thickened gastric wall. The arrowhead indicates portal venous pneumatosis.

EGD: esophagogastroduodenoscopy

Regardless of its etiology, the clinical features and image findings of phlegmonous gastritis shared by most cases are as follows: the sudden onset of a fever, epigastric pain, nausea and hematemesis with severe inflammation; a thickened gastric wall with an intramural low-density area on CT; and fold thickening or extensive ulceration on EGD. Given that it can often be misdiagnosed as peptic ulcer perforation, Borrmann type 4 gastric cancer or gastric malignant lymphoma from CT/EGD alone, it is important to judge all of these elements together to make an accurate diagnosis of phlegmonous gastritis (8, 9).

Streptococcus spp. is isolated as the causative agent in approximately 70% of all cases followed by Staphylococcus spp., Escherichia coli, and the other indigenous bacteria in the digestive tract (2, 10). In the present cases, indigenous bacteria in the digestive tract, Proteus mirabilis, α-streptococcus, Staphylococci, and Candida, were isolated from either gastric juice or biopsy specimens. An elevated gastric pH is known to be a risk factor of phlegmonous gastritis (1, 11). Interestingly, a recent study indicated that proton pump inhibitors (PPIs) alter the gut microbiota and increase the growth of pathogens of phlegmonous gastritis, such as Streptococcus spp., Staphylococcus spp., and Escherichia coli (12, 13). In our cases 1 and 3, PPIs had been prescribed for over a year. PPI usage is also reported to be a risk factor of spontaneous bacterial peritonitis in cirrhotic patients (14). In these previous reports, gastric acid was thought to play a crucial role in preventing the invasion of pathogens, and PPIs induce a dysfunction of the mucosal barrier (11-14). For this reason, phlegmonous gastritis can also occur as an adverse event associated with direct mucosal injury, such as that induced by endoscopic submucosal dissection and endoscopic ultrasound-guided fine-needle aspiration (15, 16).
In conclusion, phlegmonous gastritis is a rare condition which occurs due to common causes. Its morbidity may subsequently increase due to the following backgrounds: an aging society, increasing prevalence of diabetes mellitus, overuse of PPIs, more aggressive endoscopic techniques, and the development of new chemotherapies. Having a good knowledge of its clinical features and imaging findings is indispensable to make an early diagnosis and improve the treatment outcomes.

The authors state that they have no Conflict of Interest (COI).

References


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