Successful Treatment of Early-Diagnosed Primary Phlegmonous Gastritis

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Abstract

A 64-year-old man presented with epigastralgia and nausea after an acute exacerbation of chronic pancreatitis. Abdominal computed tomography revealed remarkable thickening of the gastric wall and intramural hypodense areas. Esophagogastroduodenoscopy showed a large gastric ulcer surrounded by an edematous mucosa and mucus. The results of a culture from a biopsy of the lesion indicated phlegmonous gastritis. The patient was successfully treated with an antibiotic without gastrectomy.

Key words: gastric infection, conservative therapy, phlegmonous gastritis


Introduction

Phlegmonous gastritis is an uncommon disease caused by a bacterial infection of the gastric wall. The infection predominantly occurs in the submucosa of the stomach. Therefore, it is difficult to accurately diagnose this disease in its early stage, and many patients often undergo surgical treatment in the absence of a definitive diagnosis of primary phlegmonous gastritis. The mortality rate of this disease is therefore thought to be high when appropriate treatment is delayed. Some patients are diagnosed with the disease in its chronic stage of advanced gastric cancer. We herein report a case of early-diagnosed primary phlegmonous gastritis that was successfully treated with an antibiotic.

Case Report

A 64-year-old man had been previously admitted to a hospital with severe epigastralgia and nausea. His medical history included chronic pancreatitis, type 2 diabetes mellitus (HbA1c 7.0%), and a subtotal gastrectomy with Billroth II gastroenterostomy to treat the perforation of a duodenal ulcer that had developed when he was 26 years old. Abdominal computed tomography (CT) revealed a single cyst, and calcifications were observed at the pancreatic body. Edematous change was also observed at parts of the body and tail of the pancreas. At this time, no thickening of the gastric wall was found (Fig. 1). The serum and urinary amylase levels were high at 689 IU/L and 878 IU/L, respectively. The patient’s white blood cell (WBC) count (7,600/mm$^3$) and C-reactive protein (CRP) level (0.08 mg/dL) were within the normal range. Hence, he was diagnosed with an acute exacerbation of chronic pancreatitis. Initial treatment with an antibiotic (sulbactam/ampicillin, 6 g/day) for severe clinical symptoms, including abdominal pain, led to the alleviation of his symptoms. However, on day 4 after admission, there was a recurrence of epigastralgia and nausea. Contrast abdominal CT revealed marked thickening of the gastric wall and intramural hypodense areas (Fig. 2). Laboratory findings showed that his WBC count (14,500/mm$^3$) and CRP level (17.8 mg/dL) were elevated, although his serum amylase level had decreased to 67 IU/L. A pancreatic pseudocyst infection was suspected; nevertheless, antibiotic administration was continued with imipenem (1.5 g/day) instead of sulbactam/ampicillin (6 g/day) because of the good transitivity of imipenem to the pancreas. The patient was referred to our hospital for further examination and treatment.
On admission to our hospital, his vital signs were stable: blood pressure, 150/90 mmHg; heart rate, 88 beats/min; and body temperature, 37.5°C. His nausea was relieved, but mild epigastralgia was still present. His laboratory findings were as follows: WBC count, 11,600/mm³; red blood cell (RBC) count, 310×10⁶/mm³; hemoglobin, 8.8 g/dL; platelet count, 17.4×10⁴/mm³; aspartate aminotransferase, 21 IU/L; alanine aminotransferase, 16 IU/L; total bilirubin, 0.3 mg/dL; amylase, 67 IU/L; and CRP, 14.5 mg/dL. Abdominal ultrasonography (EUS) during the procedure, a large gastric ulcer was detected at the lesser curvature of the gastric body. Marked redness and edema with mucopus were also detected in the gastric mucosa. This was thought to be one of the reasons behind the mild anemia (Fig. 4). EUS showed the presence of a pancreatic pseudocyst adjacent to the gastric wall. However, the border between the pancreatic cyst and the gastric wall was clear. Inner images of each layer of the stomach showed swelling and unclear structures. Furthermore, there was no stomal ulcer. These findings were suspected to be the cause of the symptomatic exacerbation, however, a pancreatic pseudocyst infection could not be completely excluded. To rule this possibility out in the differential diagnosis, EUS-guided aspiration of the pancreatic pseudocyst was performed. The fluid from the pseudocyst was clear and slightly brownish. The results of fluid culture were negative. The severity and time course of the clinical findings of the image examinations suggest that the main source of the patient’s symptoms may have been the stomach. Therefore, a final diagnosis of primary phlegmonous gastritis was made.

Transfusion therapy was administered and the patient was not allowed to consume either food or water. He underwent conservative treatment with the intravenous administration of a proton pump inhibitor (omeprazole, 40 mg/day) and an antibiotic (meropenem, 3 g/day). We believe that proton pump inhibitors are potent inhibitors of acid secretion, which can be administered before a definitive diagnosis of a lesion is made because they do not have any adverse effects.
Phlegmonous gastritis is a rare inflammatory disease of the stomach caused by suppurative bacteria. The incidence of this disease has decreased in recent years owing to improvements in antibiotics. Phlegmonous gastritis is caused by local or diffuse inflammation of the gastric wall (1). Local inflammation of the gastric wall often results in abscess formation in the stomach (2). Phlegmonous gastritis is classified into primary and secondary types. The primary type is usually idiopathic or occurs after the gastric wall is damaged due to trauma. The secondary type is associated with an infection of neighboring organs, such as infection due to transverse colon cancer, biliary infection, or a hepatic abscess (3). Furthermore, secondary phlegmonous gastritis may occur after endoscopic submucosal dissection (ESD) (4). Although a case of secondary phlegmonous gastritis has been reported to have occurred within one week of ESD, there are no reports of its onset a long period of time after ESD or surgery. While bacterial infection is often hematogenous, the spread of infection is sometimes lymphogenous and may also occur by direct spread from a neighboring infected site (5). In the present case, there was no evidence of abscess formation around the pancreas, and no obvious focus of bacterial infection, such as abscess formation, was detected on the abdominal CT examination, even when exacerbation of the symptoms was observed. Furthermore, we found no bacterial infection in the puncture liquid from the pancreatic pseudocyst on endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA), the puncture liquid was clear and not turbid. If phlegmonous gastritis is of the secondary type, then the infection must be considered to have originated from the infected pancreatic pseudocyst. However, even if a past infection in puncture liquid was suspicious, the puncture liquid might be dirty or turbid. Furthermore, no bacterium was detected in the puncture liquid from the pancreatic pseudocyst (probably due to the use of antibiotics). From these findings, we could not confirm that any infection occurred in the pancreas, pancreatic pseudocyst, or the connective tissues surrounding the pancreas. Our findings suggest that the direct infiltration of bacteria into the gastric wall occurred via the oral cavity rather than via the pancreas or a hematogenous infection. Together with these findings, the patient’s acute phlegmonous gastritis could be considered to be primary type - although the infection route and bacterial source of phlegmonous gastritis could not be identified throughout the clinical course of this disease.

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**Discussion**

Phlegmonous gastritis is a rare inflammatory disease of the stomach caused by suppurative bacteria. The incidence of this disease has decreased in recent years owing to improvements in antibiotics. Phlegmonous gastritis is caused by local or diffuse inflammation of the gastric wall (1). Local inflammation of the gastric wall often results in abscess formation in the stomach (2). Phlegmonous gastritis is classified into primary and secondary types. The primary type is usually idiopathic or occurs after the gastric wall is damaged due to trauma. The secondary type is associated with an infection of neighboring organs, such as infection due to transverse colon cancer, biliary infection, or a hepatic abscess (3). Furthermore, secondary phlegmonous gastritis may occur after endoscopic submucosal dissection (ESD) (4). Although a case of secondary phlegmonous gastritis has been reported to have occurred within one week of ESD, there are no reports of its onset a long period of time after ESD or surgery. While bacterial infection is often hematogenous, the spread of infection is sometimes lymphogenous and may also occur by direct spread from a neighboring infected site (5). In the present case, there was no evidence of abscess formation around the pancreas, and no obvious focus of bacterial infection, such as abscess formation, was detected on the abdominal CT examination, even when exacerbation of the symptoms was observed. Furthermore, we found no bacterial infection in the puncture liquid from the pancreatic pseudocyst on endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA), the puncture liquid was clear and not turbid. If phlegmonous gastritis is of the secondary type, then the infection must be considered to have originated from the infected pancreatic pseudocyst. However, even if a past infection in puncture liquid was suspicious, the puncture liquid might be dirty or turbid. Furthermore, no bacterium was detected in the puncture liquid from the pancreatic pseudocyst (probably due to the use of antibiotics). From these findings, we could not confirm that any infection occurred in the pancreas, pancreatic pseudocyst, or the connective tissues surrounding the pancreas. Our findings suggest that the direct infiltration of bacteria into the gastric wall occurred via the oral cavity rather than via the pancreas or a hematogenous infection. Together with these findings, the patient’s acute phlegmonous gastritis could be considered to be primary type - although the infection route and bacterial source of phlegmonous gastritis could not be identified throughout the clinical course of this disease.

Pathogens causing phlegmonous gastritis are identified from gastric tissue or fluid cultures. The pathogen most
commonly found to cause phlegmonous gastritis is *Streptococcus*, which is resistant to gastric acid (6). *Staphylococcus, Escherichia coli, Haemophilus influenza*, and endogenous bacteria of the oral cavity are also commonly involved. In addition, mixed infections are often reported (7). Nevertheless, in this case, the use of an antibiotic meant that the original pathogen was not clear. The bacterial culture of the gastric tissues showed the presence of *Peptostreptococcus spp.*, which is part of the normal bacterial flora. However, there are no reports of this bacterium alone causing gastric infection. Although the definitive bacterium was not proven in this case, we hypothesize that the phlegmonous gastritis in this case was most likely caused by a combined infection involving *Peptostreptococcus spp.*

Although the etiology of phlegmonous gastritis is unknown, alcohol consumption, immunosuppression, chronic gastritis, drugs, mucosal injury, and malignancy are considered to be possible causes (8). Alcohol addiction is often associated with the occurrence of phlegmonous gastritis. In this case, the patient had consumed alcohol (20 g/day) since the age of 20 years and had a history of alcohol abuse before the development of upper gastrointestinal symptoms. He was prescribed mecobalamin (1,500 mg/day), losartan potassium (50 mg/day), and hydrochlorothiazide (12.5 mg/day). He did not take oral hypoglycemic drugs or insulin, and his HbA1c level was 7.0% on admission. Therefore, we did not believe that diabetes mellitus and the medications were the main reason behind the occurrence of phlegmonous gastritis, although the association between these diseases cannot be completely excluded. It can therefore be said that the patient’s alcohol consumption most likely caused the phlegmonous gastritis.

The symptoms of phlegmonous gastritis are nonspecific. Typical clinical manifestations are reported to include epigastric pain, vomiting, and fever. Epigastric pain, which is sometimes relieved by sitting in an upright position (Deininger’s sign), and purulent emesis, although rare, are typical signs of this condition (9). In this case, we observed Deininger’s sign, in which abdominal pain becomes more severe in the supine position and is relieved with the sitting position, throughout the clinical course. The onset and progression of phlegmonous gastritis is usually rapid, such that accurate diagnosis is difficult in the early stage based on clinical features alone.

Imaging examinations are helpful in the diagnosis of phlegmonous gastritis; the disease is often associated with gastric intramural hemorrhage, necrosis, pus, and thrombosis of the submucosal blood vessels. All of these findings indicate that abdominal CT may show gastric wall thickening and intramural hypodense areas. EUS is also reported to be able to detect gastric wall thickening and anechoic areas (8).

The optimal treatment for phlegmonous gastritis remains controversial. The most typical treatment is suggested to be conservative management with antibiotics or surgical gastrectomy. In the past, total gastrectomy was performed for the treatment of phlegmonous gastritis; however, the success rate was low. Recently, local resection has been recommended for patients with septic conditions because of the high mortality rate and complications, such as perforation (10, 11). As in our case, if phlegmonous gastritis is diagnosed early, it can be treated with an appropriate antibiotic and supportive measures, including the administration of a proton pump inhibitor (12). The diagnosis of phlegmonous gastritis in the early stage and treatment with an appropriate antibiotic may therefore improve the prognosis and cure rate of this disease.

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

**References**