

Phlegmonous Esophagitis Treated with Internal Drainage and Feeding Jejunostomy

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We report the case of a 67-year-old woman presenting with epigastric pain. Computed tomography identified diffuse phlegmonous esophagitis. Esophagogastroduodenoscopy revealed multiple perforations in the mucosal layer of the esophagus. A large amount of pus was drained internally through the gut. The patient was treated with antibiotics and early jejunostomy feeding. Although phlegmonous esophagitis is a potentially fatal disease, the patient was successfully treated medically with only a minor complication (esophageal stricture).

Key words: 1. Phlegmonous esophagitis
2. Drainage
3. Esophagitis

Case report

A 67-year-old woman with epigastric pain presented to Gangnam Severance Hospital. Her symptoms had begun 2 weeks earlier, and she had been diagnosed with esophagitis at another hospital. After 2 weeks of fasting, she started oral feeding; however, epigastric pain recurred, and she was transferred to our hospital. Her medical history included type 2 diabetes mellitus and well-controlled hypothyroidism. She had a history of drinking 2 glasses of wine 3 times per week for 50 years. Her body temperature was 36.4°C and blood pressure was 143/69 mm Hg. Her abdomen was soft without tenderness. Laboratory tests showed a leukocyte count of 7,570/ μ L (neutrophils, 68.0%) and a C-reactive protein level of 79.7 mg/L. No bacterial growth was observed in blood or sputum cultures.

Esophagogastroduodenoscopy (EGD) was performed (Fig. 1). Although there were multiple esophageal perforations, the muscular layers were intact; thus, a

large amount of pus was drained internally, resulting in effective drainage without penetrating the muscular layer of the esophagus. Moreover, contrast-enhanced chest computed tomography (CT) revealed diffuse edematous wall thickening with submucosal areas of air density in the esophagus. Air bubbles were also seen in the thickened wall of the esophagus (Fig. 2).

On chest CT, bilateral pleural effusions were noted, but there was no evidence of fluid collection in the mediastinum, and the patient's heart function was normal. These findings suggested a diagnosis of acute phlegmonous esophagitis.

Intravenous antibiotics and other drugs to control the patient's diabetes were administered, and fasting was recommended. However, because early enteral nutrition was considered necessary for long-term medical management and L-tube insertion could induce a friable esophageal wall injury, a feeding jejunostomy and enteral feeding were started on the fifth hospital day. Thereafter, the patient gradually recov-

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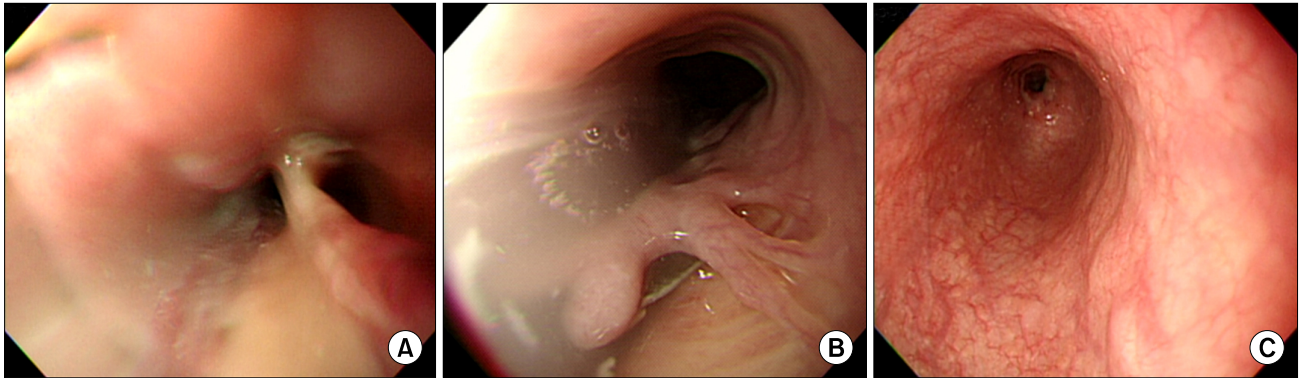


Fig. 1. EGD findings showing improvements of the esophageal perforation and erythema in the diffusely thickened mucosal wall. (A) Initial EGD. (B) 3 Weeks later. (C) 2 Months later. EGD, Esophagogastroduodenoscopy.

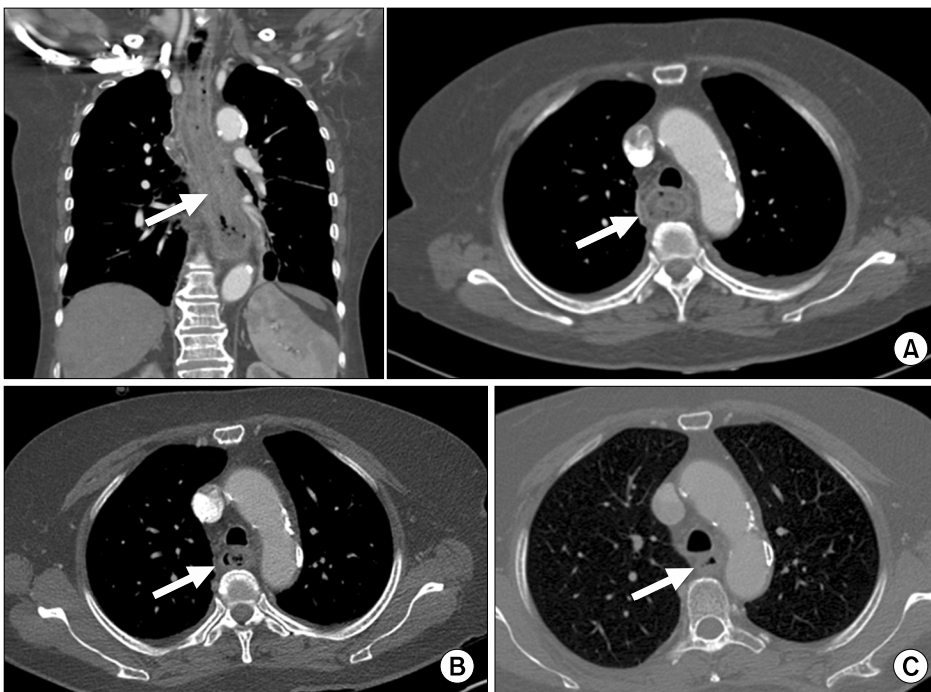


Fig. 2. Chest CT images showing improvements in esophageal inflammation and disappearing sub-mucosal areas of air density (arrow). (A) Initial CT. (B) 3 Weeks later. (C) 2 Months later. CT, computed tomography.

ered and was discharged on the 10th hospital day.

We then evaluated the patient with EGD and a chest CT scan by three weeks at our outpatient clinic. After 2 months, the phlegmonous esophagitis had significantly improved on EGD, but there was an esophageal stricture that could not be passed with a scope. Because the patient experienced symptoms of dysphagia, we performed esophageal ballooning and stent insertion. Two weeks after esophageal stenting, the dysphagia had resolved significantly; the stent was removed and the patient was able to tolerate oral feeding. She had no complaints of dysphagia and

showed no recurrence of the infection during 14 months of follow-up.

Discussion

The term 'phlegmonous' refers to pus formation through a diffuse inflammatory process. This kind of infection can spread through the gastrointestinal tract, but the stomach is most frequently involved [1]. Acute phlegmonous esophagitis is a rare, life-threatening infection of the esophagus. The inflammation infiltrates into the muscularis mucosa and serosa,

and can induce perforation or peritonitis.

The pathogenesis of acute phlegmonous esophagitis is unclear, but predisposing factors include immune suppression, peptic ulcer disease, alcoholism, chronic gastritis or other gastric mucosal injuries, achlorhydria, infection, connective tissue disease, and malignancy. Nevertheless, approximately 50% of patients have no significant risk factors [2].

In a histopathological examination, the submucosa is thickened and infiltrated by neutrophils and plasma cells with intramural hemorrhage, necrosis, and thrombosis of submucosal blood vessels. The most common pathogens are *Streptococcus*, *Staphylococcus*, *Escherichia coli*, *Haemophilus influenzae*, *Proteus*, and *Clostridia* [3].

Acute phlegmonous esophagitis is associated with specific findings on chest CT images. Typically, a low-density circumferential area is found within the thickened wall of the esophagus, with significant rim enhancement after the intravenous injection of contrast medium. Moreover, air bubbles formed by gas-producing organisms are seen in the esophageal wall. Intramural low attenuation represents severe inflammation and abscess localized to the submucosal and muscularis layers [4].

The overall mortality of phlegmonous gastritis in a review of 36 reported cases was 42%; the mortality rate for patients with localized disease was 17%, whereas that for patients with diffuse disease was 60%. Moreover, the mortality rate for cases requiring surgical drainage and resection was 50% (13 of 26). Because surgical and medical management are both high-risk, the most effective treatment for acute phlegmonous esophagitis should be chosen depending on the clinical circumstances [1,5-7].

The only risk factors of this patient were diabetes mellitus and chronic alcohol consumption; there were no infectious events in the esophagus. Moreover, we were able to observe the effective internal drainage of pus in the mucosal and submucosal layers by EGD; thus, surgical drainage was not considered at first. Because pus drainage is important, other institutions tend to attempt surgical drainage first; however, since the esophageal muscular wall was intact and internal drainage was confirmed through EGD, we thought that surgical management was not needed because it could induce mediastinitis by caus-

ing pus to drain from the esophageal wall, as well as because the surgical risk was high [6]. Furthermore, early antibiotic therapy is important, and enteral feeding through early jejunostomy is also critical for long-term medical management. Without enteral nutrition, the patient may not receive sufficient nutritional support and the natural immune system could be compromised [6]. Therefore, regardless of whether medical or surgical treatment is chosen, we believe that early enteral feeding should be considered in patients who need long-term fasting.

Although phlegmonous esophagitis is a severe illness with a high mortality rate, adequate antibiotic therapy, internal drainage of pus, and early enteral feeding could yield good clinical results. Moreover, regular EGD may be beneficial for evaluating clinical improvements and performing additional procedures to treat post-inflammatory esophageal stricture.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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