Acıbadem Üniversitesi Sağlık Bilimleri Dergisi
Cilt: 4 • Sayı: 2 • Nisan 2013

Acute Phlegmonous Gastritis During Steroid Treatment for Crohn's Disease

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ABSTRACT

Acute phlegmonous gastritis is an extremely rare and frequently fatal condition characterized by suppurative bacterial infection of the gastric wall. Since Cruveilhier first described the condition in 1862, the proportion of affected patients who survive remains no higher than 58%, despite important advances in diagnostic techniques and the availability of more potent broad-spectrum antibiotics. In this manuscript, we present a unique case of acute phlegmonous gastritis encountered during steroid treatment for Crohn's disease. We think that diagnosis at a very early stage and rapid treatment with a combination of broad-spectrum antibiotics, and sometimes early surgical removal of the infected tissues, may prove to be a life-saving strategy when treating such patients, as it was in the case discussed here.

Keywords: Phlegmonous gastritis, pancreatitis, steroid treatment, Crohn's disease

CROHN HASTALIĞI İÇİN UYGULANAN STEROİD TEDAVİSİ SIRASINDA GELİŞEN AKUT FLEGMONÖZ GASTRİT

ÖZET

Akut flegmanöz gastrit, mide duvarının süpüratif bakteriyel infeksiyonu ile karakterize, son derece nadir görülen ve sıklıkla ölüme neden olan klinik bir durumdur. Cruveilhier tarafından ilk kez tanımlandığı 1862 yılından bu yana, teşhis yöntemlerindeki önemli gelişmelerin olması ve elimizde çok daha etkin geniş spektrumlu antibiyotiklerin olmasına olmasına rağmen, hayatta kalabilen hasta oranı halen %58'i geçmemektedir. Bu yazıda, Crohn hastalığı nedeniyle steroid tedavisi almakta olan bir hastada gelişen akut flegmanöz gastrit vakası sunmaktayız. Sunduğumuz bu vakada olduğu gibi, bu tür bir hastayı tedavi ederken çok erken dönemde doğru tanıyı koyarak hızlı bir şeklide geniş spektrumlu bir antibiyotik kombinasyonunun başlanılmasının ve gereğinde erken dönemde enfekte dokuların cerrahi olarak uzaklaştırılmasının hasta açısından hayat kurtarıcı olabileceğini düşünmekteyiz.

Anahtar kelimeler: Flegmanöz gastrit, pankreatit, steroid tedavisi, Crohn hastalığı

Acute phlegmonous gastritis is an extremely rare and frequently fatal condition characterized by suppurative bacterial infection of the gastric wall. During the rapid course of the disease, progression of inflammation from the stomach to the adjacent tissues and systemic inflammatory reactions is frequently observed. The patient usually dies within a few hours or days (1). Cruveilhier first described the condition in 1862 (2), but the rate of affected patients who ultimately survive remains no higher than 58% today (3). This survival rate has not increased despite advances in diagnostic modalities, such as esophagogastroduodenoscopy (EGD) and endoscopic ultrasonography (EUS), as well as the availability of more potent broad-spectrum antibiotics. A high index of suspicion is an important issue for early diagnosis, as is the immediate start of life-saving medical or surgical therapies.

In this report, we present a unique complication, acute phlegmonous gastritis, which is related to steroid treatment for Crohn's disease.

Gönderilme Tarihi: 16 Kasım 2012 • **Revizyon Tarihi:** 08 Şubat 2013 • **Kabul Tarihi:** 09 Şubat 2013
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Case Report

A 33-year-old man with a history of Crohn's ileocolitis who had been receiving deflazacort (50 mg/day) and azathioprine (100 mg/day) for three weeks presented with sudden-onset severe upper abdominal pain, nausea, vomiting, and fever. Upon admission, the patient was very feeble and restless. On physical examination, blood pressure was 80/60 mm Hg and body temperature was 39.7 ^{0}C. The patient complained of upper abdominal tenderness, but there was no clinical evidence of peritonitis. Initial laboratory examinations revealed the following (normal values in parentheses): white blood cell count (WBC), 24.3 x 10^3/L (4–10 x 10^3/L); Hb, 13.9 g/dL (12–16 g/dL); hematocrit, 39.9% (37%–47%); platelet count, 292 x 10^3/L (150–400 x 10^3/L); and C-reactive protein (CRP), 129 mg/L (<5 mg/L).

Other laboratory results were within normal ranges. Contrast-enhanced computed tomography (CT) of the abdomen was immediately performed to exclude intra-abdominal abscess formation. This revealed a diffusely thickened antral gastric and bulbar wall and an edematous appearance of the head of the pancreas (Figure. 1A and 1B).

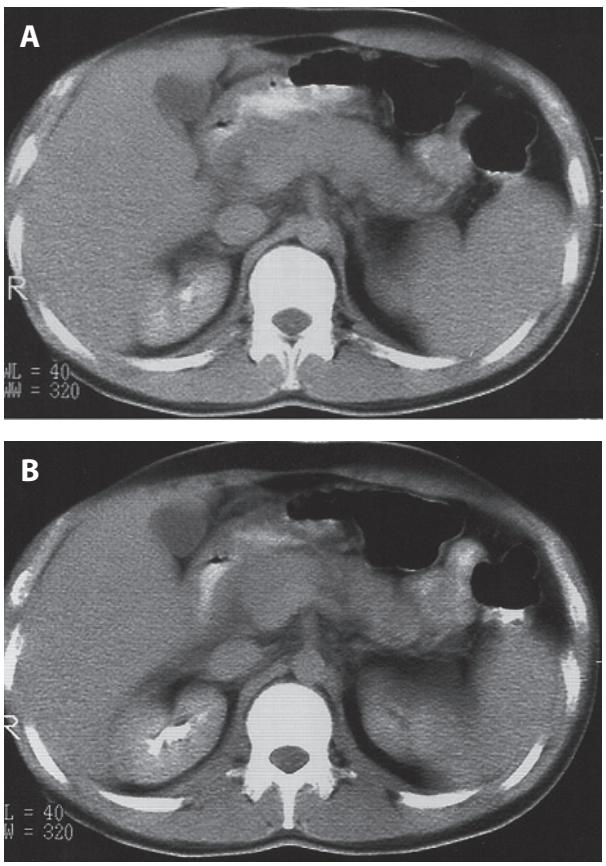

Figure 1. **A** and **B.** Abdominal CT scans at two different levels show marked thickening of antral and bulbar walls along with an edematous appearance of the head of the pancreas.

The EGD revealed diffuse swelling in the gastric folds and moderately inflamed mucosa with diffuse erythema and punctate submucosal inflammatory deposits in the distal part of the corpus and antrum (Figure. 2A and 2B). Beyond the gastric mucosa, the entire anterior wall and part of the posterior wall of the bulbus were extensively inflamed with a fibrinopurulent exudate overlying a friable, hyperemic, and highly edematous mucosa (Figure. 2C).

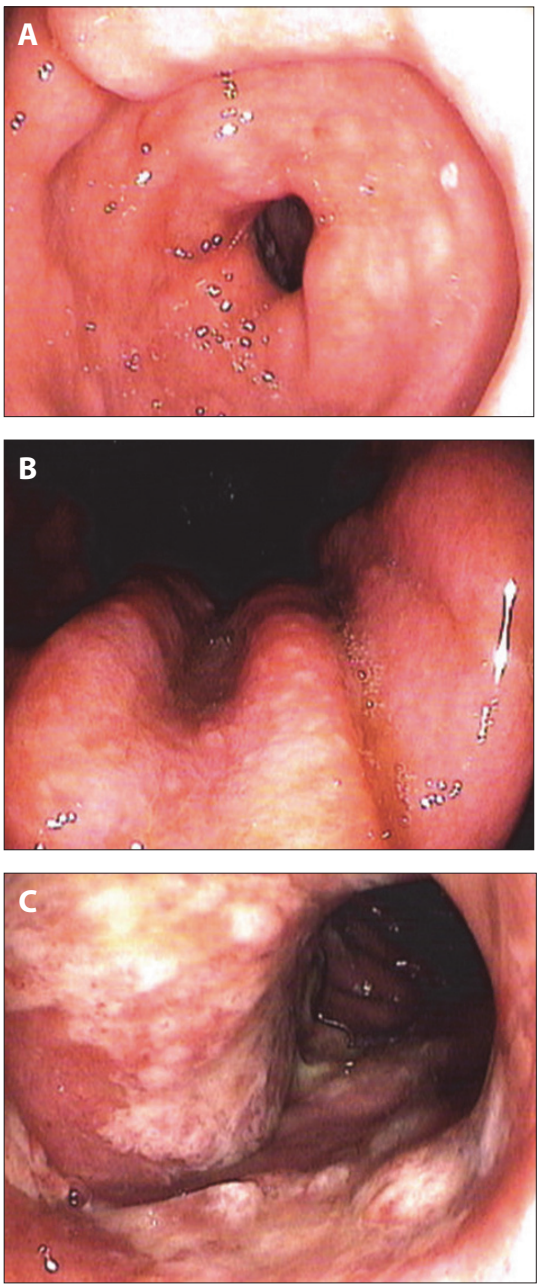

Figure 2. **A, B** and **C.** Endoscopic views from antrum **(A)** and angulus **(B)** show diffuse swelling in the gastric folds and moderately inflamed mucosa with diffuse erythema and punctate submucosal inflammatory deposits in the distal part of the corpus and antrum. At the bulbus **(C)**, the entire anterior wall and part of the posterior wall are extensively inflamed with a fibrinopurulent exudate on the surface of a friable, hyperemic, and highly edematous mucosa.

Biopsy specimens were taken from both the gastric and bulbar mucosa. Pathological examination revealed intense submucosal and partly mucosal neutrophil and plasma cell infiltration with micro-abscess formations (Figure. 3A and 3B). These pathological findings were all concordant with acute phlegmonous gastritis.

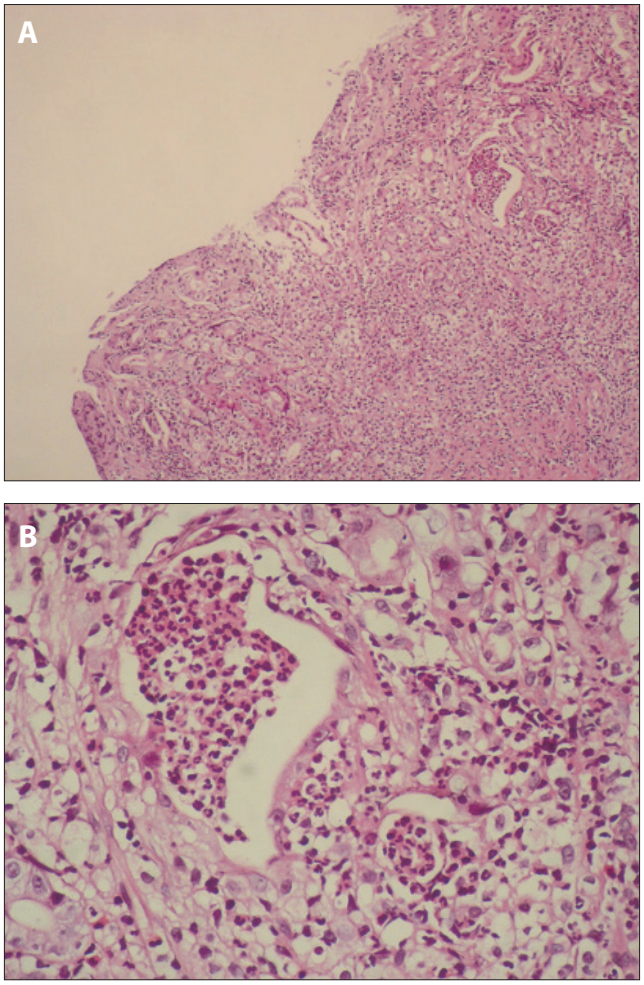

Figure 3. - A and B. Histopathologic examination of biopsy specimens from the mucosa of the angulus. A. Thickening of the submucosa because of the dense infiltration of neutrophils and plasma cells formed some micro-abscesses (H&E, 20x). B. Upon closer view, micro-abscess formations can be seen in more detail (H&E, 40x).

After the diagnosis, deflazacort and azathioprine therapies were stopped, and intravenous administration of broad-spectrum antibiotics was started immediately: ceftriaxone (4 g/day), metronidazole (1.5 g/day), and orally administered mesalazine (4 g/day). During this treatment, he had no oral intake.

On the third day of hospitalization, the patient's condition began to improve. His abdominal pain and fever decreased, and he reported feeling better. On the fifth day,the patient stopped reporting symptoms and all physical evidence of symptoms disappeared as well. Intravenous antibiotics were continued until the seventh day and then were changed to orally administered ciprofloxacin (400 mg/day) and metronidazole (1.5 g/day) for 10 days. The patient was discharged on the tenth day with the following laboratory results: WBC, 7.8 x 10^3/L; Hb, 12.8 g/dL; hematocrit, 38.8%; platelet count, 312 x 10^3/L; amylase, 56 IU/L; lipase, 121 IU/L; and CRP 3 mg/L. Other laboratory examinations were again within normal ranges.

One month after the patient was discharged, the EGD was repeated. Gastric and bulbar mucosa displayed a normal appearance, and the pathological examinations of the control biopsy specimens were nearly normal except for slight chronic antral gastritis. All abdominal CT findings were normal, and laboratory results were unremarkable.

Discussion

Acute phlegmonous gastritis is an extremely rare condition characterized by a suppurative bacterial infection of the stomach. Three different clinical presentations of phlegmonous gastritis have been described (4). In the fulminating form, the disease follows a rapidly progressive course with a sudden onset of severe upper abdominal pain, nausea, vomiting, and high fever with prominent systemic inflammatory signs, such as a marked leucocytosis with high CRP levels and erythrocyte sedimentation rate. The patient's health deteriorates rapidly, and the patient usually dies within a few hours, before a physician can correctly diagnose the condition (5). This may be related to endotoxemic shock because of Gram-negative or Gram-positive sepsis (6). However, the more usual presentation is the acute form. In this presentation, the course of the illness is slower than in the fulminating form, but it causes similar patient complaints and laboratory findings. The patient usually dies within a few days if a timely diagnosis is not made. In rare cases, the disease may follow a more chronic benign course, with only localized intramural abscess formation. The patient may be afebrile, complaining of only mild epigastric pain and anorexia.

A large number of predisposing factors have been proposed, such as an immunocompromised state (chronic alcoholism, HIV infection, Kaposi's sarcoma, pregnancy and coronary bypass surgery) (7-11), and loss of mucosal integrity (chronic gastritis, peptic ulcer disease, and endoscopic injury) (12-14). Interestingly, half of the patients who present with this condition have no history of serious illness.

The most common pathogens responsible for phlegmonous gastritis are *Streptococcus* spp. [3], although many other bacterial organisms have been implicated as causes (15,16). The possible routes of infection are hematogenic spread of bacteria from any infected site such as pneumococcal endocarditis, erysipelas, impetigo, puerperal infections (3,17), and possibly the gastrointestinal tract, such as was observed in our patient. In addition, infection can result from direct contamination of the gastric lumen with infected secretions from the throat and lungs.

Pathologically, the inflammatory process may be limited, resulting in gastric abscess (18), but more frequently the process is diffuse (15). The major site of the inflammation is the submucosal space, but inflammation frequently spreads outward and inward, and results in peritonitis and mucosal inflammation (19, 20). Furthermore, other than typical phlegmonous gastritis, emphysematous or necrotizing gastritis can be seen following, respectively, gastric submucosal invasion by gas-forming microorganisms or extensive thrombosis of the submucosal vessels (21,22).

When an EGD is performed during the early phase of this illness, only generalized erythema and edema of gastric folds can be seen. With progression of the disease, scattered submucosal inflammatory deposits or diffuse mucopurulent exudate are frequently seen on the surface of hyperemic, edematous, and sometimes necrotic mucosa. Upon EUS examination, diffuse thickening of the gastric wall is clearly visible, and this thickening is due mainly to widening of the submucosa (20) or localized gastric abscesses (23). CT of the abdomen is also a useful diagnostic technique for early detection of gastric or accompanying organ inflammation. The more typical CT finding in phlegmonous gastritis is the thickening of the gastric wall with low attenuation, and sometimes circumscribed intramural abscess formation (24,25).

Despite some older reports that conclude that inflammation in phlegmonous gastritis occurs only in the region of the stomach from the cardia to the pylorus (26), more recent reports document esophageal (27), small intestinal, and colonic involvements (28). In the present case, we observed bulbar and pancreatic involvement.

Due to the absence of broad-spectrum antibiotics in the past, the only treatment option for phlegmonous gastritis was surgical removal of the infected tissues from the body. This was achieved by either a total or subtotal gastrectomy for the diffuse form, or a gastrotomy with transmucosal incision and drainage for the localized form. Many of these patients died before or after the operation (1,29). However, once more potent broad-spectrum antibiotics became available, an increasing number of case reports documented the successful treatment of phlegmonous gastritis with medical therapy alone, such as in the case of our patient (20, 23). Nevertheless, a recent review analysing 37 cases reported from 1973 to 2003 concluded that the combination of antibiotic therapy with an early gastric resection might have a more favourable effect on the survival rate than medical therapy alone (3). However, we think that treatment options must be adapted to a patient's clinical situation: for example, as a function of the extent and location of the inflammation or the presence of co-morbid conditions.

In conclusion, acute phlegmonous gastritis is an extremely rare and frequently fatal condition, and clinical diagnosis may sometimes be challenging. A high index of suspicion is mandatory to differentiate acute phlegmonous gastritis from the gastric involvement of Crohn's disease, because the exact diagnosis is not possible with only EGD or abdominal CT findings. It is well-known that a large number of complications related to steroids can be seen during the treatment for Crohn's disease, but to the best of our knowledge, this is the first report of such a complication in the medical literature. We think that diagnosis at a very early stage and rapid treatment with a combination of broad-spectrum antibiotics, and sometimes early surgical removal of the infected tissues, may prove to be a life-saving strategy when treating such patients, as it was in the present case.

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