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Primary gastric actinomycosis: A case report

Primer gastrik aktinomikoz: Olgu raporu

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Gastric actinomycosis is an extremely rare disease. To date, about 20 cases have been reported in the literature. In most cases, diagnosis was made by histopathologic evaluation of an operative specimen. We report here a 68-year-old man with primary gastric actinomycosis who was admitted to the hospital with upper gastrointestinal bleeding and diagnosed as actinomycosis by microscopic examination of biopsy specimens obtained by endoscopy. This case is reported because of the rarity of endoscopically diagnosed primary gastric actinomycosis.

Key words: Gastric actinomycosis, GI bleeding

Gastrik aktinomikoz oldukça nadir bir hastalıktır. Literatürde şimdiye kadar yaklaşık 20 olgu bildirilmiş olup çoğunda tanı operasyon materyelinin histopatalojik değerlendirmesiyle konulmuştur. Burada üst gastrointestinal kanama ile hastaneye başvuran ve endoskopik biyopsi ile aktinomikoz tanısı konulan 68 yaşında bir erkek hasta sunulmaktadır. Bu olgu, endoskopik olarak tanı konulan primer gastric aktinomikozun çok nadir oluşu nedeniyle bildirilmektedir.

Anahtar kelimeler: Gastrik aktinomikoz, GI kanama

INTRODUCTION

Gastric actinomycosis is an extremely rare disease. To date, about 20 cases have been reported in the literature (1, 2). In most cases, the diagnosis was made by histopathologic evaluation of an operative specimen. Pre-operative studies, including endoscopy with biopsy specimens, frequently suggest malignant tumor or peptic ulcer disease (1). Here, we report a case with gastric actinomycosis diagnosed by microscopic evaluation of endoscopic biopsy specimens. Previously reported cases and the literature on diagnostic procedures are reviewed.

CASE REPORT

A 68-year-old man with hematemesis and melena was admitted to the emergency ward for evaluation of upper gastrointestinal (GI) bleeding. He had a history of three previous GI bleeding episodes, at 7, 4 and 1 year(s) prior to this admission. The most recent bleeding episode was associated with nonsteroid anti-inflammatory drug (NSAID) use. He had a smoking history of one pack of cigarettes per day over the last 25 years, without alcohol consumption. Past medical history revealed a laparoscopic cholecystectomy 2.5 years ago following a diagnosis of cholelithiasis.

Physical examination revealed a blood pressure of 100/60 mm Hg, pulse rate of 112/min and respiratory rate of 20/min. He was in moderate general condition, conscious and with good cooperation. His skin was pale and he had poor oral hygiene with many decayed teeth. He did not have jaundice, fever or palpable lymph nodes. The only abnormal finding in abdominal examination was mild epigastric tenderness during deep palpation. Laboratory test results revealed hemoglobin level: 7.1 g/dl, mean corpuscular volume (MCV): 88 fL, white blood cell (WBC): 10500/µl with a normal differential, platelet (PLT): 241,000/ml, fasting blood glucose 89 mg/dl, creatinine: 0.9 mg/dl, aspartate

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aminotransferase (AST): 25 IU/L, alanine aminotransferase (ALT): 20 IU/L, total protein: 7.4 g/dl, albumin: 3.5 g/dl, total bilirubin: 0.4 mg/dl, iron (Fe): 48 mg/dl, and unsaturated iron binding capacity (UIBC): 338 pg/dl.

Electrocardiography and chest X-ray findings were normal. Panendoscopy performed 24 hours following admission revealed a gastric ulcer with a wide base on a polypoid lesion showing approximately 1.5 cm protuberance into the gastric lumen associated with small erosions in the bulbus and antral region. When a remnant of hemorrhage in the center of the ulcer was removed by washing, a black colored base was observed (Figure 1). No active hemorrhagic focus was observed. Lack of gallbladder due to previous cholecystectomy was demonstrated by an abdominal ultrasound.

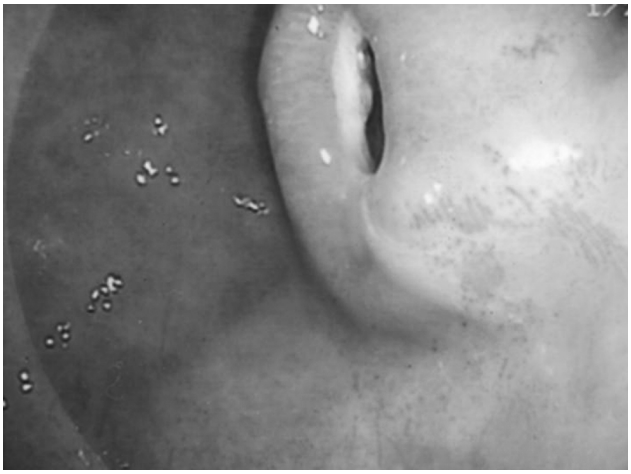

Figure 1. Endoscopic photograph showing gastric ulcer with a wide black colored base on a polypoid lesion in association with small erosions

Oral feeding was stopped and treatment with a parenteral proton pump inhibitor was initiated. Four units of packed red blood cells were transfused to increase hemoglobin levels to 10 mg/dl. After stabilizing the patient's hemodynamic status, a second panendoscopy was performed to reevaluate the previously observed lesion. At the second look, small sand-like black particles were observed in the base of the ulcer. Multiple biopsies were performed. Rapid urease test (CLO) performed on antral biopsy for *Helicobacter pylori* was total positive. Partial healing of previously observed erosions was noted. Microscopic examination of the endoscopic biopsy specimens revealed a diagnosis of actinomycosis (Figure 2).

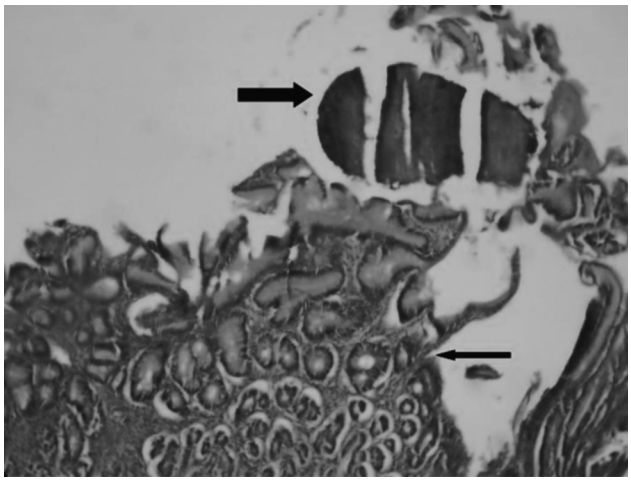

Figure 2. Microphotograph showing *Actinomyces* colonies in the gastric mucosa. Edema, congestion and active chronic inflammatory cells are prominent in the lamina propria. Thick arrow: *Actinomyces* colonies; thin arrow: gastric pits [hematoxylin and eosin, X 10]

Two-week treatment with crystallized penicillin G (24 million units per day) was administered during the patient's hospitalization. Operation was recommended by surgeons after completion of medical therapy. The patient was discharged to continue treatment with oral penicillin up to 12 months.

DISCUSSION

Actinomycosis is a rare, chronic, suppurative disease caused by *Actinomyces* species that is present in the normal flora of the oral cavity and GI tract, and characterized by formation of sulfur granules. *Actinomyces* can affect the cervicofacial, pulmonary, abdominal and pelvic area. The most common site of invasion has been reported as the cervicofacial region. Intra-abdominal actinomycosis is less common and has predilection for the terminal ileum, cecum, and appendix. Since low gastric pH is generally sufficient for killing or inhibition of microorganisms, primary gastric actinomycosis is an extremely rare condition. To date, about 20 cases have been reported in the literature. Factors that precipitate intra-abdominal actinomycosis include GI surgery, inflammation, perforation, foreign bodies and intrauterine devices (1). The time interval between suspected invasion and diagnosis is long. In our case, the suspected precipitating factor was cholecystectomy, and the interval between cholecystectomy and diagnosis was 2.5 years.

Clinical manifestations of gastric actinomycosis have been frequently confused with peptic ulcer or gastric malignancy-related symptoms, which are low-grade fever, epigastric pain, weight loss (3) and GI bleeding. Radiologic studies (4-6) and endoscopic findings of gastric actinomycosis usually suggest malignant tumor or peptic ulcer disease. In most cases, the diagnosis can be made by histopathologic evaluation of a biopsy specimen. Our patient was hospitalized with upper GI bleeding, and endoscopic biopsy specimens from the ulcer base revealed sulfur granules, confirming the diagnosis of actinomycosis.

Long-term and high-dose antibiotic therapy is required for a successful outcome. Penicillin is the drug of choice, administered at a dose of 18-24 million units per day for 2 to 6 weeks, followed by oral penicillin up to 12 months. Ampicillin, amoxicillin, tetracycline, macrolides, clindamycin, chloramphenicol, and cephalosporins were also reported to be effective (7).

In conclusion, although rare, gastric actinomycosis should be included in the differential diagnosis of gastric ulcers. Reporting of such cases may help increase the awareness of this important and curable disease.

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