

Biliary Esophagitis: A Case Report

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Abstract

Bile reflux esophagitis has been reported and discussed in the literature for many years. What makes our case unusual are the gross and microscopic photos of bile in the esophageal mucosa. The pertinent literature is discussed especially the deleterious and oncogenic effects of bile in the esophageal mucosa.

Keywords: Mucosa; Esophageal mucosa; Hiatal hernia; Biliary tract

Case Report

A 76 year old woman was admitted to Christ Hospital, Jersey City, New Jersey on March 27, 2004, with intractable bilious vomiting. On examination she was found to have severe dehydration, prostrations and atrial fibrillation with hypertension. She appeared to have continuous bilious vomiting, spilling on her bed.

Rales were present on the left lower chest. Past history revealed 20 prior hospitalizations with malnutrition, vomiting, anaemia and urgent and chronic diabetic care. Four months previously, she was admitted for removal of infected right pectoral pacemaker and replacement with a new one in the left pectoral area.

fundal esophagitis, severe malnutrition, left lower lobe atelectasis and pneumonia. Electrocardiogram revealed atrial fibrillation, sick sinus syndrome and a functioning pacemaker. Echocardiogram showed normal left ventricular size and contractility and mild left atrial enlargement. Bacterial culture revealed gram positive bacterium and urinary infection.



Figure 1: Barium esophagram; sliding hiatal hernia.



Figure 2: Endoscopic view of hiatal hernia and acute gastritis.

Work-up revealed a gastrointestinal series, oropharyngeal dysphagia, esophagogastric paresis, and hiatal hernia with severe gastroesophageal reflux (Figure 1). Other tests revealed severe anemia,

A left subclavian catheter and a nasogastric catheter were inserted. Nevertheless, she kept vomiting bile. On April 22, 2004, I was called on consultation and performed an upper gastrointestinal fiberoptic endoscopy. Photograph revealed pronounced gastritis and a large hiatal hernia (Figure 2).

The esophagogastric area revealed mucosal biliary staining and Barrett's esophagus (Figure 3). Biopsies confirmed the Barrett's ulcers of esophagus (Figures 4 and 5).

The black spots in these pictures were negative for melanin and hemosiderin. No chemical tests for bile were available. The patient's relatives refused surgical correction at this time and for the next two months.

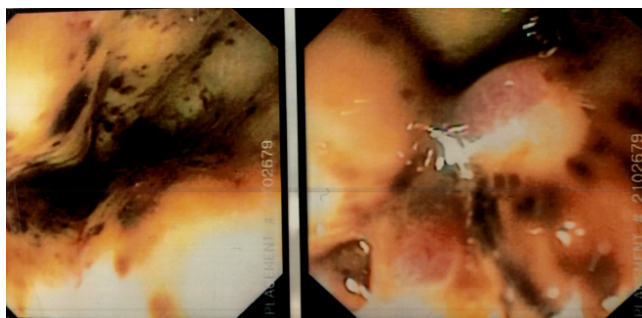


Figure 3: Esophagogastric area bile stains, Barrett's changes.

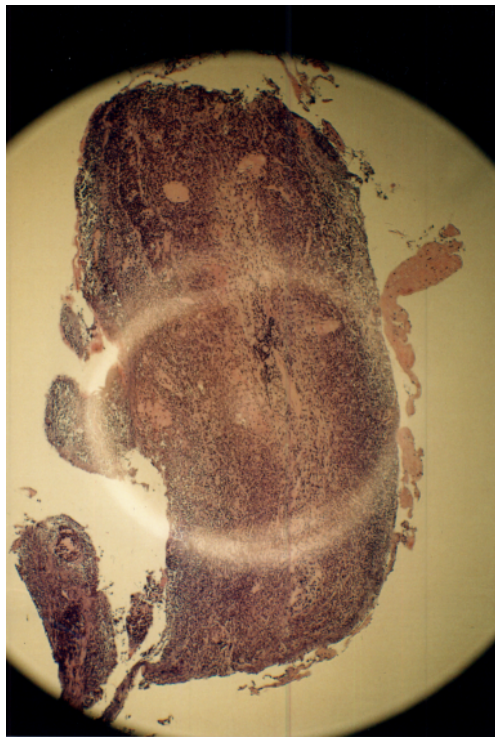


Figure 4: Low power biopsy of Barrett's area.

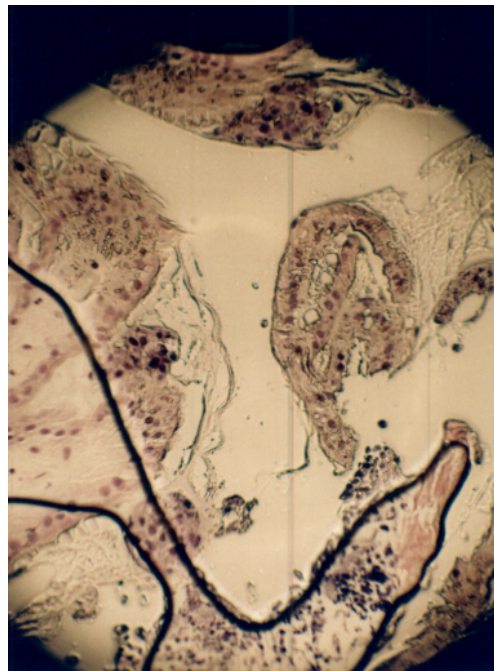


Figure 5: High power view: see bile particles in the esophageal mucosa.

The patient was treated for two months. A percutaneous fiberoptic gastrostomy was performed. Diabetic management, antibiotics, and antacids were given. Vomiting continued even though to a lesser extent. Two months after admission, repeat upper endoscopy was performed revealing clearing of bile staining but confirming the Barrett's metaplasia (Figure 6). She was discharged and two months later she was readmitted to a different hospital for a repeat percutaneous gastrostomy. On November 6, 2004, she died at the nursing home.



Figure 6: Barrett's metaplasia 2 months later: no bile stains.

Discussion

This case report emphasizes the destructive and fatal results of long standing gastroesophageal reflux. Bile esophagitis was present in our patient. The additional effects of bile on the esophageal mucosa are seen in Figures 3-5. Jiang has described esophagitis in children caused by bile and acid [1]. Eros described bile induced ATP depletion, vast cell degranulation and tissue damage in dogs, all of which could be prevented by choline antagonists. Severe inflammation in the submucosa but no bile was seen [2]. Aiyer also studied the bile changes in esophageal mucosa but no bile was seen in biopsies [3]. Mitros's Atlas of extensive study of gastrointestinal pathology mentions bile gastritis but no bile esophagitis [4].

Bile plays a role with refluxed acid in the development of Barrett's esophagus and its malignant transformation, according to Peters and colleagues [5]. In our case, visual evidence of bile staining had disappeared two months later (Figure 6). Perhaps the injury to the esophageal mucosa had occurred or it may have continued, since Barrett's metaplasia continued in the second endoscopy (Figure 6).

Refusal of operation was unfortunate. The patient's aspiration pneumonia cleared. The diabetes was under control. Echocardiogram revealed normal ventricular size and contractibility. The intended surgical correction carried no formidable risk. We had ample experience of surgical correction. In 1999, we published a 24 year follow-up of our stapled uncut gastroplasty in 161 patients. Only one

fatality occurred in an emergency operation [6,7]. Our patient expired seven months after the first endoscopy in a nursing facility.

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