Chest Pain in an Aspirin-Sensitive Asthmatic Patient*

**Eosinophilic Esophagitis Causing Esophageal Dysmotility**

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We describe a case of eosinophilic esophagitis in a 38-year-old man with aspirin-sensitivity asthma which presented as noncardiac chest pain. Manometric measurements demonstrated tertiary contractions. Biopsies showed a dense eosinophilic infiltrate in the mucosa. There was no response to therapy for reflux. Symptoms quickly resolved with corticosteroid therapy. Subsequent manometric values recorded after corticosteroid therapy showed resolution of the dysmotility. Biopsies showed normal mucosa. Adult asthmatic subjects with noncardiac chest pain should receive further investigation if reflux therapy fails to resolve the symptoms.  

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Key words: esophageal spasm

The most common esophageal symptoms in asthmatic subjects are due to gastroesophageal reflux. Reflux may worsen the asthma, and control of the asthma may not occur until the reflux is adequately managed. Many medications used to treat asthma decrease lower esophageal sphincter tone. This may contribute to the reflux symptoms. Therefore, reflux is commonly seen in both children and adults with asthma, both as a cause of the asthma and as a consequence of treatment.

In this case report, we describe an asthmatic patient with a long history of reflux who develops a new chest pain syndrome related to, but distinct from, the reflux. Initial attempts to manage the reflux did not decrease the pain. A diagnosis of dysmotility was made by esophageal manometric measurements. Mucosal biopsy demonstrated eosinophilic esophagitis. The patient promptly responded to corticosteroid treatment.

**Case Report**

A 38-year-old man with asthma was seen for a 1-month history of atypical chest pain. The pain was substernal, squeezing, and did not radiate. The pain often awoke the patient at night. Sometimes the pain was preceded by heartburn. It was partially relieved by antacids and swallowing cold liquids. The episodes of pain would last from 15 min to several hours. There was no shortness of breath or diaphoresis. There was no exertional component. The chest pain was preceded by a 2-week history of increased nasal discharge. There were no new medications, new foods, or changes in diet.

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Food had occasionally become painfully “stuck” while swallowing over a period of the past 7 years. This could be cleared by swallowing liquids. There were no other gastrointestinal complaints.

The past medical history was significant for lifelong asthma, presently controlled with inhaled steroids; chronic sinusitis treated with inhaled nasal steroids; a 25-year history of heartburn relieved by antacids; and a 10-year history of migraine headaches. There had been two instances of nasal polyectomy in the past. The patient enjoyed normal exercise tolerance and participated in bicycling, hiking, snowshoeing, and mountain climbing without limitation.

Current medications were triamcinolone acetonide (Azmacort), 4 puffs bid; triamcinolone acetonide nasal spray (Nasacort), 4 sprays in each nostril bid; and a tablet containing terfenadine and pseudoephedrine hydrochloride (Seldane-D), taken every morning. The patient was allergic to aspirin, which caused angioedema and bronchospasm. Sulfa drugs caused a rash. There was a family medical history of atopy and asthma in the maternal relatives. The occupational and the social histories were noncontributory.

The physical examination revealed a thin healthy man. There was no wheezing on auscultation of the chest. The heart sounds were normal. The abdomen was nontender with normal bowel sounds. Results of the remainder of the examination were also within normal limits.

The peripheral eosinophil count was normal. The ECG showed first-degree atroventricular block, but there were no ST- or T-wave abnormalities.

A clinical diagnosis of reflux esophagitis was made. The patient began receiving omeprazol, 20 mg qd, and antireflux measures were initiated. The painful episodes continued and the patient began to sleep in a sitting position in order to decrease the heartburn which seemed to trigger the painful episodes. A barium swallow showed mild esophagitis, no reflux, and no spasm. Esophagastroduodenoscopy was performed. Esophageal spasm and mild esophagitis were noted. Biopsy specimens demonstrated a dense eosinophilic infiltrate of the mucosa (Fig 1, left). Eosinophil infiltration was absent in the stomach biopsy specimens, but was present, to a lesser degree than in those of the esophagus, in the duodenum specimens (not shown). Esophageal manometric data (Fig 2, A) demonstrated decreased lower esophageal sphincter tone and impressive esophageal tertiary contractions. A Bernstein test was positive. Manometric measurements following acid perfusion demonstrated prolonged multiphasic contractions of the lower esophagus associated with pain (Fig 2, B). The omeprazol dose was increased to 20 mg bid, and ciaspride, 20 mg bid, was added to the regimen. Symptoms persisted. After 6 weeks without improvement in chest pain attacks, prednisone, 40 mg po qd for 10 days, was prescribed. By day 3, the chest pain and dysphagia resolved. Biopsies and manometric measurements were repeated 3 weeks after steroid treatment. The eosinophilic infiltrate resolved (Fig 1, right), and evidence of multiphasic contractions, detected through the manometric values, was absent (Fig 2, C). The decreased lower esophageal sphincter tone persisted (Fig 2, C). Dysphagia and chest pain did not return after 1 year of observation following the corticosteroid treatment. The reflux symptoms were completely controlled with ranitidine, 150 mg, and ciaspride, 20 mg, before bedtime.

**Discussion**

The patient described in this report has lifelong aspirin-sensitive asthma with a 7-year history of dysphagia and a 1-month history of chest pain. The biopsy specimens prior to steroid treatment showed a dense eosinophilia which resolved with corticosteroid therapy.

Mild eosinophilia often is associated with esophagitis, especially in children. However, dense eosinophilia, as reported here, is rare and usually is associated with eosino-
Figure 2. Esophageal manometric measurements. Esophageal manometric values with tracings obtained at 28, 31, 34, and 37 cm from the incisors. A: the tracings show peristaltic propagation of contraction from the mid-esophageal (top tracing) to the gastroesophageal junction (bottom tracing). Abnormal triphasic contractions are clearly demonstrated in the lower tracings (34 and 37 cm). B: multiphasic contractions of the lower esophageal (37 cm) during a positive Bernstein acid perfusion test. During infusion of the acid, 0.1N HCl, the patient developed chest pain. Several episodes of multiphasic lower esophageal contractions, consistent with esophageal spasm, were recorded. These continued during infusion of normal saline solution. C: normal esophageal manometric values following corticosteroid treatment. Normal peristalsis and waveforms are present. No triphasic or multiphasic contractions were seen. Lower esophageal sphincter tone remained low, 10 cm.

Eosinophilic gastroenteritis. Eosinophilic gastroenteritis predominantly affects children, is associated with peripheral eosinophilia and dense eosinophilic infiltration of the stomach and small bowel, and frequently is associated with a history of allergies. Isolated eosinophilic esophagitis is rare and often is associated with esophageal stricture.

Vitellas et al described 13 patients with eosinophilic esophagitis; 9 of the patients had eosinophilic gastroenteritis with esophageal involvement. Of the four patients with isolated esophageal disease, three had esophageal stricture. Twelve of the 13 patients had peripheral eosinophilia. Ten patients had a history of allergies (asthma, allergic rhinitis, urticaria, food allergy, atopic dermatitis, or medication allergy). Twelve patients were treated with steroids or cromolyn sodium, and all responded.

Berezin et al described 16 asthmatic children with asthma and chest pain. All had esophagogastroendoscopy, manometric measurements, and Bernstein testing. Eleven of these patients had endoscopically and histologically confirmed esophagitis; four had a positive Bernstein test. One had esophageal spasm. Seven of the patients with esophagitis had 24-h pH monitoring, and all had reflux. Nine of the 11 patients had resolution of symptoms with medical treatment for reflux. One refused fundoplication. One refused surgery and remained symptomatic. There was no description of eosinophilia in the histologic sections in the report. In each case, resolution of reflux esophagitis was associated with resolution of chest pain.

Attwood et al described 12 patients with isolated esophageal eosinophilia (defined as >20 eosinophils per high-power field). These patients presented with dysphagia, and none had anatomic obstruction. Ten had abnormal esophageal motility. Two of the patients had diffuse spasm, and two had nutcracker esophagus (typified by peristaltic but high amplitude contractions). Three of these patients had asthma, two had sinusitis, and two had a history of medication allergy. Of seven peripheral eosinophil counts, six were normal. Only one patient was treated with steroids, and symptoms improved.

We believe that our patient had eosinophilic esophagitis without gastric involvement presenting as chest pain and dysphagia. The prompt response to steroids is consistent with previous reports. What is unusual about this case is that aspirin-sensitivity asthma is not previously reported in association with eosinophilic esophagitis. Aspirin-sensitive asthmatic subjects tend to have "intrinsic" asthma with few allergic symptoms.

The patient in this report had no seasonal component to his asthma, no peripheral eosinophilia, and no known allergies which exacerbated his disease. We speculate that inflammatory mediators from his sinusitis may have contributed to the esophageal eosinophilia. Perhaps the esophageal reflux contributed by injuring the mucosa, allowing inflammatory mediators access to the submucosa.

This case report emphasizes that asthmatic subjects with chest pain may have eosinophilic esophagitis causing esoph-
ageal spasm. In this case, there was a dramatic and lasting response to a short course of corticosteroid therapy. Esophagogastroduodenoscopy with biopsies and esophageal manometric measurements are required to confirm the diagnosis. Our patient had sustained remission of symptoms on qHS ranitidine and cisapride.

REFERENCES

Upper Airway Obstruction Secondary to Acinic Cell Carcinoma of the Trachea

Use of Nd:YAG Laser

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Comprising 1 to 4% of all tumors and 7 to 15% of malignant neoplasms of the major salivary glands, acinic cell carcinoma (ACC) rarely occurs in the respiratory tract. There has been only one case of ACC of the trachea previously reported in the medical literature. A second case of ACC of the trachea associated with upper airway obstruction and its management by Nd:YAG laser and surgical resection is reported.

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Key words: acinic cell carcinoma; Nd:YAG laser resection; tracheal tumor

Abbreviations: ACC=acinic cell carcinoma

Acinic cell carcinoma (ACC) comprises 1 to 4% of all tumors and 7 to 15% of malignant neoplasms of the major salivary glands. However, ACC of the respiratory tract is extremely rare and there has been only one case of ACC of the trachea previously reported in the medical literature by Horowitz and Kronenberg in 1994. Here, we report a second case of ACC of the trachea associated with upper

Figure 1. Bronchoscopic view of the tracheal lesion obstructing the trachea by approximately 80% (top), and the patent trachea (bottom).