Candy Cocaine Esophagus*

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Reversible thermal injury to the esophagus from drinking boiling-hot liquids has been reported to produce alternating pink and white linear bands that impart a “candy-cane” appearance to the inner esophageal wall. This injury has been associated with chest pain, dysphagia, odynophagia, and abdominal pain. We describe a case of candy-cane esophagus caused by thermal injury from smoking freebase cocaine, associated with left shoulder and arm pain, diaphoresis, hypotension, and transient cardiac ischemia. This case illustrates the importance of considering candy cane esophagus in the evaluation of chest pain, even when this symptom is suspected to be of cardiac origin.

(CHEST 2002; 121:1701–1703)

Key words: angina pectoris, variant; burns; cocaine-related disorders; coronary angiography; “crack” cocaine; endoscopy; GI; esophageal diseases; esophagitis; myocardial ischemia; wounds, nonpenetrating

Dutta et al1 reported two cases of reversible thermal injury to the esophagus from drinking boiling-hot liquids, in which alternating pink and white linear bands imparted a “candy-cane” appearance to the mucosa. The injury produced chest pain, as well as dysphagia, odynophagia, and abdominal pain. We wish to share a case of candy-cane esophagus caused by thermal injury from smoking freebase (“crack”) cocaine, which also resulted in transient cardiac ischemia. This case illustrates the importance of considering candy cane esophagus in the evaluation of angina when in the proper clinical setting, as well as its converse.

Case Report

A 55-year-old African-American man ingesting two to four analgesics containing acetaminophen, aspirin, and caffeine per day for a toothache had sudden and unrelenting pain develop in the left shoulder and arm accompanied by diaphoresis while lifting boxes, 2 days after smoking freebase cocaine. His bowel movements had been normal. On presentation to the emergency department, he was in mild distress. His temperature was 36.8°C, pulse rate was 97 beats/min, BP was 138/77 mm Hg, respiratory rate was 18 breaths/min, and the oxygen saturation was 97% breathing room air. Physical examination of the cardiac, pulmonary, and abdominal regions was unremarkable. Digital rectal examination revealed melena stool. The hematocrit was 30%, BUN was 35 mmol/L, and creatinine level was 1.1 mmol/L. The chest radiograph appeared normal. The initial ECG showed normal sinus rhythm, left atrial enlargement, and borderline voltage criteria for left ventricular hypertrophy (Fig 1, left).

Two hours after presentation, the patient became acutely diaphoretic and hypotensive, with a BP of 85/50 mm Hg. Repeat ECG showed new biphasic T waves and T-wave inversions in leads V3 through V6 (Fig 1, right). Urgent cardiac catheterization demonstrated patent coronary arteries, with the exception of a nearly occluded small branch at the bifurcation between the left anterior descending and circumflex coronary arteries. Esophagogastroduodenoscopy within the hour revealed a candy-cane appearance to the distal 10 cm of esophagus (Fig 2), patchy erythema and erosions in the gastric antrum, and an 8-mm diodenal bulb ulceration with a red spot in its otherwise white base. Biopsies of the distal esophagus revealed parakeratosis, squamous hyperplasia with regeneration, and scant acute inflammatory infiltrate. Biopsies of the stomach revealed chronic gastritis and bacteria consistent with Helicobacter pylori.

Following his diagnostic studies, the patient was specifically questioned about possible thermal injuries to his esophagus. He recalled that when he had last smoked freebase cocaine and inhaled what he had expected to be vapor, he instead sucked boiling liquid into his mouth, which he spit out. After belching smoke, however, he had recognized that he must have swallowed at least a portion of this liquid.

The patient’s ECG changes resolved later that day, with no enzymatic evidence for myocardial necrosis. The hematocrit remained at 31 to 32% over 24 h. The day following presentation, the patient was discharged home receiving a proton pump inhibitor, which was continued for 1 month. Amoxicillin and clarithromycin were added for 2 weeks. He was instructed to avoid nonsteroidal anti-inflammatory drugs. The patient has remained asymptomatic for >1 year.

Discussion

The most likely cause of the patient’s angina equivalent was microvascular spasm of the epicardial coronary arteries induced by either thermal injury to the esophagus or the pharmacologic effects of cocaine.2 In patients with normal coronary arteries but with syndrome X, esophageal stimulation has produced symmetric decreased coronary blood flow.3 In patients who use cocaine, myocardial ischemia from coronary artery vasomotor constriction has been observed acutely4 and during withdrawal.5

Acute pulmonary injury from inhalation of “crack” cocaine or its by-products of pyrosysis was unlikely to have caused the patient’s left upper extremity pain, as he had no dyspnea, cough, alveolar infiltrate, or pneumomediastinum.2 Similarly, a vasovagal response, hypoxia or hypovolemia was unlikely to have explained his transient myocardial ischemia, since the patient had improvement in his
Figure 1. Left: initial ECG on presentation to emergency department. Right: ECG following episode of angina equivalent and hypotension. Note new T-wave changes in leads I, aVL, and V3-V6.

Figure 2. Candy-cane appearance of distal esophagus at esophagogastroduodenoscopy immediately following cardiac catheterization.
Delayed Closure of Persistent Postpneumonectomy Bronchopleural Fistula*

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A 73-year-old man with a history of postpneumonectomy empyema and a long-term chest tube since 1979 presented with fever, chills, leukocytosis, and purulent fluid from the left tube thoracostomy. CT scan and bronchoscopy demonstrated a right lower lobe pneumonia and a left mainstem dehiscence with direct communication to the left tube thoracostomy. He underwent primary closure of the bronchopleural fistula with latissimus dorsi muscle flap coverage after antibiotic therapy for right lower lobe pneumonia. (CHEST 2002; 121:1703–1704)

Key words: empyema; pneumonectomy; thoracostomy

Abbreviations: BPF = bronchopleural fistula; PNE = postpneumonectomy empyema

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The incidence of postpneumonectomy empyema (PNE) occurs in approximately 5% of pneumonectomy patients, and the occurrence of bronchopleural fistula (BPF) appears in > 50% of cases.1,2 The mortality of PNE with a BPF has been reported to be 11 to 13%. 2 Persistent BPFs often are associated with multiple operations and prolonged hospitalization.3 Occasionally, small, uncomplicated BPFs may heal spontaneously, and in 20% of patients, BPFs will close with drainage only. However, the remaining 80% of patients whose BPFs persist will require additional operative procedures.1–6

Case Report

We report a 73-year-old man with a significant history of postpneumonectomy BPF and PNE. The patient had a 90-pack-year history of smoking. In 1979, he underwent a thoracotomy and left upper lobectomy for removal of an aspergilloma. Three months postoperatively, he presented with pneumonia in the left lower lobe, which was resistant to antibiotic treatment. He underwent a second thoracotomy for a completion pneumonectomy. He developed postpneumonectomy BPF and PNE 2 weeks following his pneumonectomy. He underwent an additional thoracotomy to control massive bleeding and empyema in the left chest cavity. Four days later, the chest was closed and a tube thoracostomy was placed. Thereafter, the chest tube remained in place without sequelae for 22 years until the patient re-presented in April 2001 with cough, dyspnea, chills, anorexia, malaise, and new-onset purulent drainage from the tube thoracostomy. Chest radiography revealed fluid collection in the left pleural space and right lower lobe pneumonia. Candida albicans was identified in the chest cavity. A chest CT scan demonstrated BPF from the left pneumonectomy stump site and right lower lobe pneumonia. Flexible bronchoscopy revealed a wide-open left mainstem bronchus to the left tube thoracostomy with an 8-cm stump length from the carina.

The patient was taken to the operating room after a 1-week course of antibiotics to resolve his right lower lobe pneumonia. He underwent a double-lumen right-sided rapid sequence intubation, with right-lung ventilation only. The thoracotomy incision was reopened to the bronchial stump site. The long-term chest tube was removed. An extremely fibrotic, long, bronchial stump was excised and debrided. The pleural space was filled with antibiotic solution. The stump site was closed in a double-layer fashion with interrupted polypropylene (Prolene; Ethicon; Somerville, NJ) and interrupted vicryl. The latissimus dorsi muscle was sutured over the stump site, a chest tube was placed, and the incision was closed. He was extubated on the operating room table. The patient had an unremarkable postoperative course and was discharged on postoperative day 7. IV antibiotic therapy continued until discharge. Follow-up is limited to 7 months. Postoperative bronchoscopy and chest radiographs reveal no evidence of recurrent BPF.

Comment

Various systemic factors and therapeutic interventions often contribute to the risk of PNE and BPF, including advanced age in men (> 70 years), preoperative radiation, malnutrition, and prolonged steroid therapy. In addition, technical factors such as prior lung resection, infection at a long bronchial stump site, and residual sepsis in the pleural space may further contribute to the development of this complication.1,2 The treatment of early PNE includes antimicrobial therapy, adequate pleural drainage,