Candidiasis-Induced Esophageal Strictures

Farooq P. Agha
Department of Radiology, University Hospital and Medical Center, Ann Arbor, Michigan, USA

Abstract. Candidiasis of the esophagus progressing to hard fibrosed strictures of the esophagus in 2 patients is reported. Both patients had deficient immunologic systems and received extended courses of broad-spectrum antibiotic therapy for control of sepsis. The strictures were progressive despite adequate antifungal therapy and several attempts at dilatations and necessitated visceral esophageal substitution as definitive surgical therapy.

Key words: Esophagus, stricture – Candida esophagitis.

Esophageal strictures are rare sequelae of candidiasis of the esophagus. Since the first radiologic description of candidiasis of the esophagus by Andreén and Theander in 1956 [1], a wide spectrum of radiographic features have been described in the literature [2–10]. However, Candida-induced esophageal strictures have only been recorded in 4 patients previously [3, 10–12]. This report describes 2 immunologically deficient patients who received extended courses of broad-spectrum antibiotics for control of sepsis. They subsequently developed progressively disabling hard fibrosed esophageal strictures due to recurrent candidiasis. This unique and rare sequela of invasive candidiasis required visceral esophageal substitutes as definitive surgical therapy despite adequate antifungal therapy and many attempts at dilatation.

Case Reports

Case 1

A 29-year-old man complained of decreased sweating, dryness of the mouth and eyes, and generalized weakness of several months' duration. His work-up in 1977 suggested a Sicca complex and myasthenia gravis syndrome. His chest radiograph revealed a large lobulated anterior mediastinal mass which had been growing over the past 7 years. In September 1979, he underwent resection of the anterior mediastinal malignant thymoma measuring 17 × 9 cm in size which had invaded the pericardium and the lingula. Partial pericardectomy and lingular resection were also performed. The postoperative course was complicated by myasthenic crisis and purulent mediastinitis, which resolved with chest tube drainage and antibiotic therapy. He developed Candida infection involving the oropharynx, esophagus, and the inguinal and gluteal regions. A course of amphotericin B resolved the oral lesions. A barium swallow in October 1979 (Fig. 1A) showed monilial plaques in the esophagus. Antifungal therapy was continued. Over the next several months he experienced progressive dysphagia. In May 1980 a barium swallow (Fig. 1B) revealed an 8-cm-long stenosis of the proximal thoracic esophagus. Immunologic studies at that time revealed depressed T-cell function. The esophageal stricture was periodically dilated and the patient was maintained on antifungal therapy (nystatin and amoxicillin). A barium swallow in October 1980, carried out because of persistent dysphagia (Fig. 1C), revealed 2 areas of tight stricture formation at the site of previous stenosis. Attempts at dilation resulted in esophageal perforation necessitating transmediastinal esophagectomy and visceral esophageal substitution with gastric interposition. The postoperative course was uneventful. When last seen in May 1982 the patient's gastric interposition was functioning well and he was eating without dysphagia.

Case 2

A 49-year-old insulin-dependent diabetic man developed a sigmoid volvulus in November 1975 for which he underwent exploratory celiotomy and sigmoidopexy. In January 1976 he developed a small bowel obstruction. At the time of operation several adhesions were lysed and a 122-cm infarcted loop of small bowel was resected. Postoperatively he developed aspiration pneumonia which was treated with vigorous nasotracheal suction and broad-spectrum antibiotics. His slow recovery due to diabetes mellitus required long-term antibiotic therapy. This
increased his susceptibility to Candida infection, and he developed severe oropharyngeal, laryngeal, and esophageal involvement by Candida albicans. Antibiotics were discontinued and antifungal therapy was instituted. The Candida infection cleared from the oropharynx; however, the patient began to experience progressive dysphagia. A barium esophagogram (Fig. 2) in February 1976 revealed diffuse narrowing of the entire thoracic esophagus with few plaques and minor mucosal irregularities.

Endoscopy confirmed the presence of an esophageal stricture starting 30 cm from the incisors. During the next month the stricture was dilated several times. Bougienage became increasingly difficult, and on April 6, 1976, a #18 Hurst Maloney dilator could not be passed under general anesthesia beyond 31 cm. A high perforation of the esophagus occurred. The esophageal tear was repaired, and a gastrostomy tube was placed at the same time. A string was left in place from the mouth to the gastrostomy stoma for future retrograde dilatation. Over the next 2 months, the few attempts at retrograde dilatations were unsuccessful, therefore, on July 12, 1976 he underwent long-segment colonic interposition. His postoperative course was complicated by a cervical esophagocolic anastomotic leak and a pelvic abscess which resolved over the next 3 weeks. When last seen in October 1982, the patient’s colonic interposition was functioning well, and he was eating without dysphagia.

Discussion

Candida albicans, a saprophytic organism found in the oropharynx of normal persons, may progress to involve the esophagus in those with lowered host resistance. The usual predisposing factors for this opportunistic infection include leukemia, lymphoma, other malignant neoplasms, diabetes mellitus, chronic debilitating disorders requiring long-term steroid therapy, and extended use of broad-spectrum antibiotics, immunosuppressants, and chemotherapeutic agents [2, 3, 10]. In our case 1, Sicca complex, malignant thymoma, and depressed T-cell functions and in case 2 uncontrolled diabetes mellitus increased these patients’ susceptibility to candidal invasion of the esophagus. Extended use of broad-spectrum antibiotics in both patients further lowered their resistance and created ideal conditions for recurrent invasion of the esophagus by Candida.

The usual clinical presentation of acute candidiasis of the esophagus is dysphagia and odynophagia. About 50% of patients have concomitant oral lesions. Clinical diagnosis is straightforward when characteristic whitish plaques are seen in the esophagus at endoscopy. The definitive diagnosis, however, depends upon the demonstration of the fungal organisms within the esophageal tissue. The usual radiographic features and differential diag-