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Case report

Esophageal stricture secondary to candidiasis without underlying disease

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Abstract: Candidiasis of the esophagus progressing to stricture formation in a 74-year-old male is reported. Esophageal candidiasis develops in the presence of various predisposing conditions such as long-standing administration of antibiotics or corticosteroids, and malignancy. The first endoscopic examination of this patient revealed unusual multiple black plaques in the esophagus. Despite intensive exploration, no predisposing factors were found. The stricture was progressive, despite the administration of adequate antifungal therapy, and its presence necessitated several attempts at dilatation. Case reports of esophageal candidiasis without underlying disease are very rare.

Key words: Esophagitis, candidiasis, esophageal stricture

Introduction

Candida albicans is a fungus that is normally a common inhabitant of the human mouth, oropharynx, and lower gastrointestinal tract. Esophageal candidiasis is a complication of blood disease, cancer, diabetes, and treatment with broad-spectrum antibiotics, steroids, cytotoxic agents, and radiotherapy. Case reports of esophageal candidiasis in the absence of underlying disease are very rare. Moreover, esophageal strictures are rare sequelae of candidiasis of the esophagus. We wish to report this very rare case of candidiasis in the presence of esophageal obstruction without underlying disease.

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Case report

A 74-year-old male presented with chest pain and nausea. On examination, he was well-developed and well nourished. The epigastrium was soft and flat. His vital signs included body temperature of 36.7°C, pulse rate of 80 beats per min, respiratory rate of 18 per min, and blood pressure of 120/80 mmHg. The leukocyte count was 10300/mm^3, hemoglobin was 14.2 g/dl, and the platelet count was 17.6 × 10^4/mm^3. The patient was not being treated with steroids or antibiotics. The history was negative for diabetes and malignant disease.

The first endoscopic examination revealed multiple black plaques in the esophagus (Fig. 1a). These nodular elevated plaques spread with frank ulceration. Biopsy specimens showed focal erosions of the mucosa and inflammatory reaction. Periodic acid-Schiff (PAS)-staining fungus was present in the paraffin fixed tissue (Fig. 2). Culture of the black plaque on Sabouraud-glucose-agar was positive for a white colony of Candida. A barium swallow revealed a long, irregular constriction of the distal two-thirds of the esophagus, with multiple transverse ulcerations (Fig. 3a).

The patient was treated with fluconazole oral suspension (300 mg/day) without symptomatic relief. At the time of the second endoscopic examination, 2 weeks later, the black plaques had disappeared from the esophagus. The white plaques remained in the esophagus (Fig. 1b). Culture of the white plaque was also positive for Candida. A repeat barium swallow 4 weeks later demonstrated a persistent irregular stricture of the esophagus with a decrease in the depth of the transverse ulcerations (Fig. 3b). The patient was discharged from the hospital after several attempts at balloon dilatation of the esophagus.

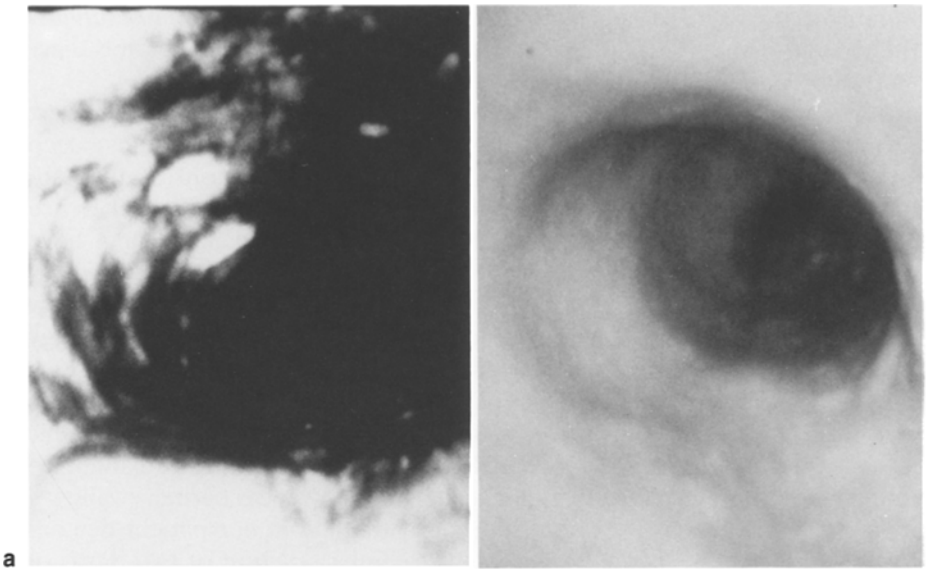

Fig. 1a,b. Endoscopic examination of the esophagus reveals **a** multiple black nodular elevated plaques with frank ulceration. **b** After antifungal therapy the black plaques are no longer apparent

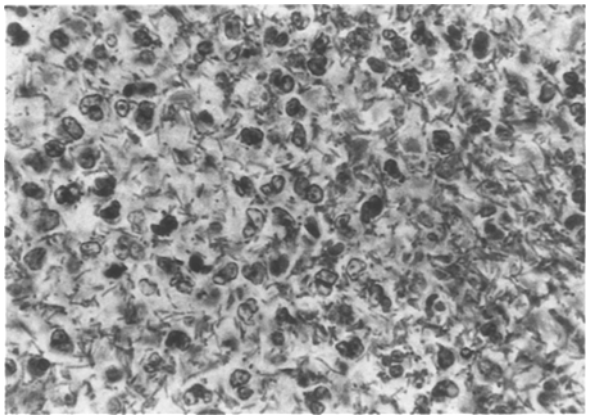

Fig. 2. Photomicrograph of biopsy specimen from the esophagus demonstrates fungal bodies. PAS, × 450

Discussion

Candida albicans is a normal flora of the mouth, oropharynx, and lower gastrointestinal tract. It becomes pathogenic when the immunity of the body is compromised.[1] The usual predisposing factors for opportunistic infection include leukemia; lymphoma; other malignant neoplasms; diabetes mellitus; and chronic debilitating disorders requiring long-term steroid therapy and extended use of broad-spectrum antibiotics, immunosuppressants, and chemotherapeutic agents.[2–4] There have been few previously documented cases of candidiasis-induced esophageal stricture. Ott and helfand[5] described patients in whom strictures of two-thirds of the esophagus developed after the resolution of acute candidiasis-induced esophagitis that occurred following the prolonged

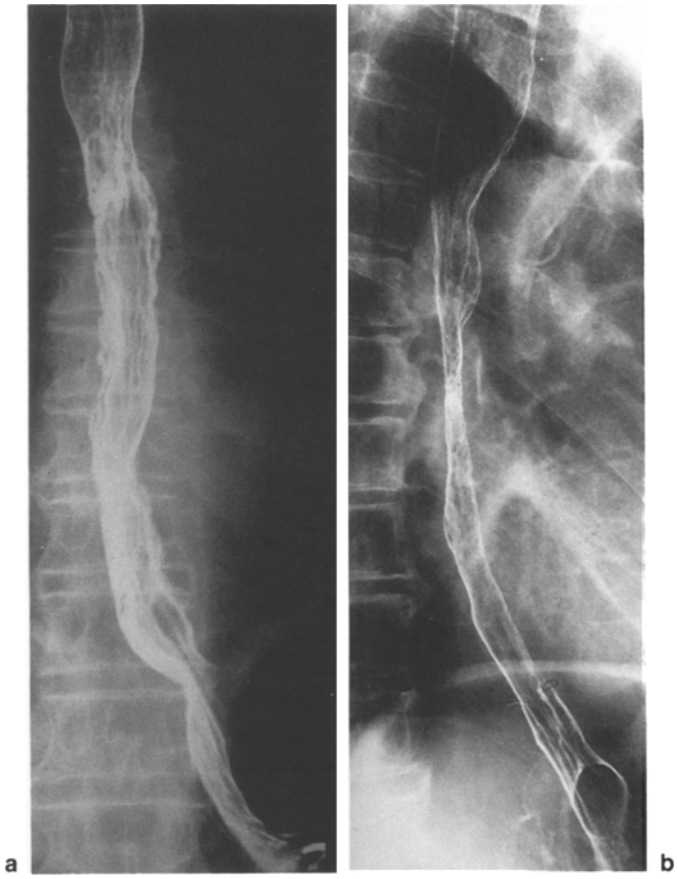

Fig. 3. a Initial examination reveals typical shaggy marginal contour and diffuse mucosal nodularity in the distal two-thirds of the esophagus. **b** Follow-up study a month later shows a persistent irregular stricture of the esophagus with proximal dilatation

administration of broad-spectrum antibiotics for pneumonia. After nystatin therapy, the caliber of the esophageal lumen increased sufficiently to permit chronic bougienage. Agha[6] reported two patients with candidiasis of the esophagus progressing to hard fibrotic strictures of the esophagus. Both patients were immunodeficient and had received extended courses of broad-spectrum antibiotics for the control of sepsis. The strictures were progressie, despite adequate antifungal therapy and several attempts at dilatation, and they necessitated visceral esophageal substitution as definitive surgical therapy. Case reports of esophageal candidiasis in the absence of underlying disease are very rare, but in out patient there was no history of any such disease.

In Candida esophagitis, the most common presenting symptoms are dysphagia and/or substernal pain or burning. These symptoms, particularly the chest pain, may initially be ascribed to other causes, such as heart disease. About 50% of patients have concomitant oral lesions.[3,4] The clinical diagnosis is straightforward when characteristic whitish plaques are seen in the esophagus at endoscopy. Based on the endoscopic picture, Kodsi et al.[7] classified four degrees of severity of esophageal candidiasis: grade I, a few raised white plaques up to 2 mm in size, with hyperemia but no edema or ulceration; grade II, multiple raised white plaques greater than 2 mm in size, with hyperemia but no edema or ulceration; grade III, confluent, linear, nodular elevated plaques, with hyperemia and frank ulceration; grade IV, findings of grade III with increased friability of the mucous membranes and occasional narrowing of the lumen. According to these criteria, the esophageal candidiasis in our patient can be classified as grade IV. The first endoscopic examination revealed multiple black plaques in the esophagus. Black rather than white or yellow plaques are uncommon. With regard to findings of black hairy tongue in the oral cavity, it has been reported that there is some relation between the black tongue and increased *Bacillus subtilis* varietas niger bacteriologically.[8] In our patient, *Candida albicans* was identified in the biopsy tissue treated with PAS stains, and culture of the black plaque was positive for Candida. Bacteriologically, *Bacillus subtilis* varietas niger was not identified, although, if the black color were really due to increased *Bacillus subtilis* varietas niger, this would support the presence of a compound infection with *Candida albicans* and *Bacillus subtilis* varietas niger in the esophagus. Makino and Amatsu[9] reported a rare case of Candida pharyngo-laryngo-esophagitis which suggested that *Bacillus subtilis* in the oral cavity may descend to the esophagus. *Bacillus niger* does not always result in a black pigmentation, especially in the presence of a low pH.[10] A repeat endoscopic examination in our patient revealed plaques of the usual white color. In this patient, *Candida albicans* was the predominant factor in comparison with *Bacillus subtilis*. Endoscopy is a very important method for the diagnosis of gastrointestinal candidiasis.

The radiographic features of esophageal candidiasis, such as shaggy outline, loss of mucosal folds, deep ulceration, nodular filling defect, spasm of the esophagus, diminished esophageal peristalsis, adherence of contrast medium, and segmental narrowing, have been described in detail by Guyer et al.[11] The findings ranged from edematous mucosal nodularity, shallow ulcers, and pseudomembrane formation to a shaggy appearance caused by deep ulceration and sloughing of the mucosa. As in our patient, several of these findings were demonstrated on the first radiographic examination. Obvious esophageal stricture cannot be detected in the acute stage. A repeat barium swallow 4 weeks after antifungal therapy in our patient demonstrated a persistent irregular stricture of two-thirds of the proximal thoracic esophagus.

In the acute stage, the mucosa is erythematous and is covered by a whitish, cheesy pseudomembrane.[7,12] This pseudomembrane consists of necrotic debris and fungi. As the disease progresses, the mucosa becomes granular and friable, with ulcerations. The submucosa and muscle layers become involved. Blockage of the submucosal gland ducts results in their dilatation. This gives the characteristic appearance of pseudodiverticulosis on barium swallow. In severe cases, esophageal disruption can occur.[6,13–17] The inflammation of the tissues around these glands later results in stricture formation. There is a predilection for involvement of the distal and middle thirds of the esophagus, although stricture formation usually occurs in the upper and middle thirds. This can be explained by the abundance of submucosal glands in this part of the esophagus.

In conclusion, our case was rare for the following reasons: First, the esophageal candidiasis developed in the absence of any predisposing condition, such as long-standing administration of antibiotics or corticosteroids, and malignancy. Second, the color of the plaques was unusual, in that they were not white or yellow, but black. Third, the patient developed progressively disabling hard fibrotic esophageal strictures due to candidiasis, despite the administration of adequate antifungal therapy.

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