Candida Esophagitis in Achalasia Cardia: Case Report and Review of Literature

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ABSTRACT

Candida albicans is by far the most common cause of infectious esophagitis. In most patients, this infection is secondary to an immuno-compromised state. In nearly 25% of the cases, underlying causes of esophageal stasis, e.g., achalasia and scleroderma, facilitate fungal colonization of the esophagus. Double contrast esophagography is a highly sensitive tool for diagnosing candida esophagitis. This report discusses the uncommon association of *Candida* esophagitis with achalasia cardia, their radiographic features and a short review of the available literature.

Key Words: Achalasia cardia, *Candida* esophagitis, double contrast esophagography, endoscopy

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INTRODUCTION

Candida esophagitis most frequently develops in immunocompromised patients but occasionally it complicates food stasis in patients with achalasia, scleroderma or peptic stricture.[1] The association of achalasia with *Candida* esophagitis is quite uncommon and an early diagnosis is mandated for adequate treatment of both these entities and for preventing the risk of metaplasia.[2] Real time barium swallow is the modality of choice to depict the manifestations of altered physiology in achalasia and supplement the findings of manometry.[3] In addition, a double contrast barium esophagogram can help assess the mucosal pathology caused by candidiasis and taper down the differential diagnosis.[3] Endoscopy with sampling for cytological / microbiological evaluation is however, the conclusive way to establish the diagnosis.

CASE REPORT

We report a 45 year old male who presented with dysphagia both to solids and liquids for the last two years with which occasional odynophagia, intermittent regurgitation and aspiration were associated. There was also a complaint of fever, chest pain and significant weight loss for the last one year. Past history of the patient was non-contributory and there was no history suggestive of immunosuppression. General physical examination was unremarkable except for pallor and mild hepatomegaly. Oral mucosa, skin and nails were healthy. Chest X-ray showed absent gastric fundal air bubble, widened superior mediastinum, straightened right mediastinal border with an air fluid level in the upper mediastinum. On the basis of clinical and chest radiographic findings, a provisional diagnosis of achalasia cardia was made. Single and double contrast barium esophagography using barium sulphate suspension (Microbar HD Eskay fine chemicals, Mumbai, India) was done under fluoroscopic control using a standard protocol. Filming was done in the erect position for the double contrast and in the prone position for single contrast studies. These studies showed grossly dilated esophagus almost along its entire length with gradual tapering at its lower end with slow and delayed passage of barium through the gastro-esophageal (GE) junction into the stomach, resembling a bird's beak or a rat's tail typical of achalasia cardia. Primary and secondary peristaltic waves were absent with tertiary contractions being the major peristaltic waves in esophagus. "Milking effect" was seen at the gastro-esophageal junction. A suspicion of mucosal irregularity was raised for which a double contrast study was done which revealed the presence of multiple small ulcers with mucosal nodularity [Figure 1]. On performing upper gastro-intenstinal endoscopy, the findings of the barium examination were confirmed. The GE junction was tight suggestive of motor dysphagia with white

patches along the entire length of the esophagus [Figure 2]. The esophageal mucosa had a friable, erythematous appearance with multiple small ulcers located in relation to the white plaques. Stomach and duodenum were normal. Microbiologic cultures of brushings obtained from the esophagus were positive for candidiasis. A manometeric examination followed by graded balloon dilatations of the esophagus for treatment of achalasia was conducted. The patient was put on antifungal therapy and was improving till the last outpatient attendance. Follow up clinical, imaging and endoscopic pictures revealed resolution of fungal etiology and reduction in severity of achalasia cardia.

DISCUSSION

Candida albicans is by far the most common cause of infectious esophagitis. Most patients with *Candida* esophagitis are immunocompromised as a result of underlying malignancy, AIDS or treatment with radiation, chemotherapy, steroids or other cytotoxic agents.[2] In nearly 75% of the cases, conditions like achalasia cardia, scleroderma etc. cause esophageal stasis that facilitate fungal colonization of the esophagus with subsequent esophagitis.[1] Prolonged untreated achalasia was the predisposing condition for candidial infection in the present report as discussed by Holt.[4]

Achalasia cardia is a motor disorder of esophagus generally occurring in the 35-50 year old age group. It is caused by degeneration of neurons in the Auerbach's plexus leading to failure of primary and secondary peristalses. Tertiary contractions subsequently develop with failure of relaxation of lower esophageal sphincter (LES). Both these factors together lead to stasis of food and secretions in the esophagus, which when prolonged may get complicated with growth of *Candida albicans* leading to *Candida* esophagitis.[2] The esophagus may undergo marked dilation with a tortuous course (i.e., sigmoid-shaped esophagus).[5]

Radiographically, as in our case, an esophagus with severe and long standing achalasia may form fluid levels and a foamy appearance in the mediastinum on plain chest film.[1] The right mediastinal border is straightened or even deviated laterally. Primary and secondary peristalses are absent on single contrast swallow with appearance of tertiary contractions in the lower half of esophagus. The incomplete opening of LES is depicted in the barium study as a sharp, smooth, tapered segment at the lower end of esophagus, described as a "rat tail" or "bird beak appearance".[2] A dilated esophagus is often seen and may have a "sigmoid appearance" in late phases.[5] Achalasia cardia must be differentiated from other causes of narrowing occurring at the lower end of esophagus such as peptic stricture, neoplasm, complicated

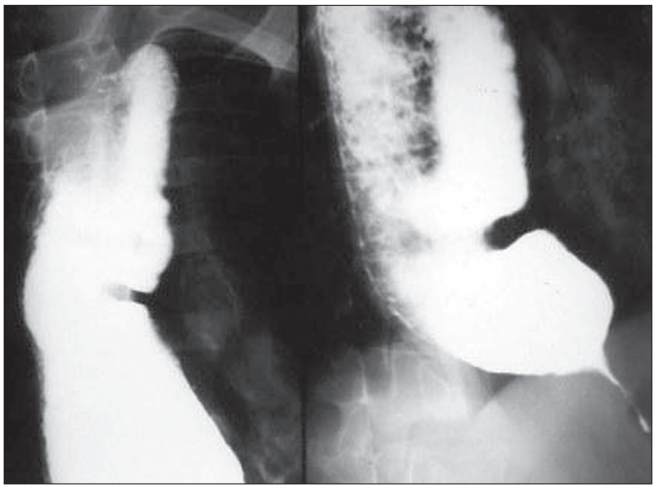

Figure 1: Double contrast barium esophagogram AP projection showing dilated esophagus with distal bird beak / rat tail taper, multiple ulcers and mucosal nodularity

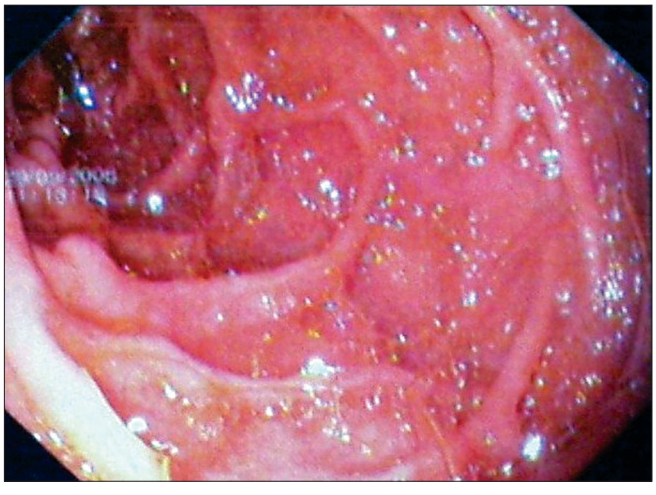

Figure 2: Upper GI endoscopy showing white patches with punctate ulcerations in the background of friable, erythematous mucosa

scleroderma, Chagas' disease and postvagotomy effect. In the present case, prolonged accumulation of food and secretions secondary to achalasia cardia, led to candidial colonization and esophagitis which was confirmed by a double contrast esophagogram and endoscopy.

A double contrast barium study has been shown to have sensitivity of 90% in the diagnosis of mucosal pathology including *Candida* esophagitis.[1,3] It is usually manifested as linear or irregular plaque-like lesions separated by segments of normal intervening mucosa. In more advanced disease, coalescent plaques may produce a "cobblestone" or "snake skin" appearance. There may be trapping of barium in between plaques and pseudomembranes.[1] Differentials of *Candida* esophagitis include herpes esophagitis, reflux

esophagitis, artifacts from air bubbles and residual adherent food particles.[2,4] The endoscopy of this patient showed the presence of a bubbly foam filling the esophagus adherent to its wall. The underlying mucosa was friable and erythematous with punctate ulcers at a few sites. These are the classical findings described in literature for *Candida* esophagitis.[1]

Although the development of candidiasis in the obstructed esophagus is uncommon, some literature reviews and infrequent case reports suggest such an association. This report reinforces the facts that long-standing stasis in the esophagus can lead to secondary opportunistic infective esophagitis, *Candida* being the most common. Single and double contrast esophagogram can diagnose these conditions accurately thus helping in early installation of antifungal therapy. We wish to highlight the occurrence of candidiasis in the presence of esophageal obstruction in our case which is consistent with many reported in literature and to illustrate the radiological findings in these cases.

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