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A case report of herpetic and candidal esophagitis in an immunocompetent adult

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ABSTRACT

Reports of combined candidal and herpetic esophagitis in immunocompetent states are rare and sporadic. A 44-year-old previously healthy lady presented with a one week history of progressive dysphagia, odynophagia and fever. Esophagogastroduodenoscopy (EGD) showed extensive desquamation of the entire esophagus except for distal 4 cm. Histopathological examination revealed ulcerated and inflamed squamous epithelium with the margin of ulcer showing a few overhanging squamous cells with dense eosinophilic cytoplasm, multinucleated and faceted nuclei with glassy chromatin, and an occasional Cowdry type A intranuclear inclusion bodies. Few candidal spores were seen in the underlying stroma. Intravenous acyclovir, fluconazole and pantoprazole were initiated. Oral analgesics were given for pain relief. She was treated for a total of 14 days. She showed significant improvement and was tolerating oral intake after discharge. The patient was asymptomatic with no evidence of recurrence at a 2-month follow-up.

1. Introduction

Esophageal infections, especially those caused by Candida and Herpes simplex, are common causes of dysphagia, more so in immunocompromised individuals^[1]. Reports of combined candidal and herpetic esophagitis in immunocompetent states are rare and sporadic[2,3,4].

2. Case report

We report a case of a 44-year-old previously healthy lady who presented with a one week history of progressive dysphagia, odynophagia and fever. There was no history of oral contraceptive use, no recent antibiotic or corticosteroid use and no risk factors for human immunodeficiency virus (HIV) infection. She had no history of recurrent infections or other findings suggestive of congenital and acquired immunodeficiency. There was no history suggestive of gastro esophageal reflux disease. On admission, her vital signs and

general physical examination were normal. Examination of the oropharynx, heart and lungs was unremarkable. Her complete blood count showed a hemoglobin of 11.7 g/dL, white blood cell count of 6800/mm³ with 41% lymphocytes and 53% neutrophils. She had a platelet count of 404000 mm³ and an ESR of 94 mm at the end of 1 hour. Her serum electrolytes, blood glucose and immunoglobulin levels were within normal limits. Serologic results of ELISA determination showed that HIV and P24 antigen were negative. Esophagogastroduodenoscopy (EGD) showed extensive desquamation of the entire esophagus except for distal 4 cm. Histopathological examination revealed ulcerated and inflamed squamous epithelium with the margin of ulcer showing a few overhanging squamous cells with dense eosinophilic cytoplasm, multinucleated and faceted nuclei with glassy chromatin and an occasional Cowdry type A intranuclear inclusion bodies. Few candidial spores were seen in the underlying stroma. ELISA titers of serum IgM antibody against HSV-1 were highly suggestive of an active HSV infection. Intravenous acyclovir, fluconazole and pantoprazole were initiated. Oral analgesics were given for pain relief. She was treated for a total of 14 days. She showed significant improvement and was tolerating oral intake after discharge. The patient was asymptomatic with no evidence of recurrence at a 2-month follow-up.

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Figure 1. Section of the esophagus showing the ulcerated squamous cells with Cowdry Type A inclusion bodies and candidal spores in the stroma.

3. Discussion

Infections of esophagus are most commonly seen in immunocompromised individuals^[1]. Common infectious agents include *Candida* spp., HSV and cytomegalovirus^[2]. Simultaneous esophageal infection with HSV and *Candida* spp. is rare and a few cases have been described in patients with sepsis or malignancies receiving radiation or chemotherapy^[5]. Herpetic esophagitis can be occasionally seen in immunocompetent hosts^[13–15]. Candidal esophagitis can also be seen rarely in immunocompetent individuals ^[16–18]. Dual infections in immunocompetent hosts are extremely uncommon^[2, 5]. Reports in adults with combined esophageal infections^[1] suggest that a possible mechanism includes injury to the esophageal epithelium by HSV first and disruption of the mucosal barrier creating a supportive environment for *Candida* spp^[6,7].

Severe odynophagia^[3] is reported to be the typical presentation for HSV esophagitis though history alone cannot clinch the diagnosis. Herpetic lesions of the esophagus are commonly seen as discrete ulcerations on an inflamed and friable mucosa at endoscopy mostly in the distal esophagus unlike in our patient where there was only desquamation with distal few centimeters of esophagus being spared^[8,9,11]. In candidal esophagitis, the esophageal mucosa is typically friable and erythematous with ulcers covered by thick adherent white exudates^[6,10].

Histologic demonstration of intranuclear inclusions in epithelial cells or multinucleated giant cells with ground– glass–appearing nuclei is occasionally found in the biopsies of patients with herpetic esophagitis^[12]. To confirm the presence of invasive candidal esophagitis, Candida spp. are seen along with squamous cells and invading hyphae on smears^[2,4].Viral culture, immunohistochemistry, PCR and serology have been used for the diagnosis^[11,19]. Herpetic esophagitis is usually regarded as a self–limited illness in otherwise healthy individuals^[9]. Treatment with acyclovir and fluconazole is recommended in severe cases and in immunocompromised patients to prevent strictures^[2, 5, 20].

Conflict of interest statement

We declare that we have no conflict of interest.

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