A rare case of dysphagia in a severely immunocompromised patient

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A 36-year-old Greek male patient presented with a 3-week history of dysphagia for both solids and liquids, anorexia, low-grade fever and fatigue. He had been diagnosed with HIV infection 13 years ago. His medical history included *Pneumocystis jirovecii* pneumonia and *Mycobacterium avium* complex infection, 20 and 14 months ago respectively, as well as a recent admission for esophageal candidiasis, currently treated with fluconazole. His last CD4 lymphocyte count was 6 cells/mm³ and the blood viral load reached 44,213 copies/ml. He was on a highly active antiretroviral therapy, but compliance was poor. He reported no illicit intravenous drug use.

On physical examination he was malnourished, afebrile, with oral thrush, hepatomegaly and splenomegaly.

Abnormal laboratory results included a leukocyte count of 1.58 K/ μ l (76% neutrophils), hemoglobin of 8 g/dl and platelet count of 110 K/ μ l.

The patient was referred for upper gastrointestinal endoscopy, since no symptomatic improvement was declared despite the antifungal treatment. Endoscopy revealed several raised whitish plaques on erythematous mucosa throughout the esophagus, while the stomach and duodenum appeared normal (Figure 1). Histology showed features of acute esophagitis. Many *Candida* hyphae and yeast were seen with both PAS and Grocott stains. The lamina propria contained aggregates of macrophages with intracytoplasmic, light blue (Giemsa), PAS-negative granules, interpreted as intracytoplasmic leishmaniae (Figure 2). Whereas titers of anti-leishmanial antibodies were equivocal, a PCR analysis performed in both bone marrow aspirate and peripheral blood smear confirmed the diagnosis of visceral leishmaniasis (VL).

The patient discontinued fluconazole and was placed on anidulafungin (100 mg *i.v.* on day 1, 50 mg *i.v.* on days 2-14) and amphotericin B liposomal (4 mg/kg *i.v.* on days 1-5, 10, 17, 24, 31 and 38). He experienced rapid resolution of dysphagia and subsequent increase in his appetite and body weight. Improvement in complete blood count (Hb: 12 g/dl, WBC: 3.27 K/µl, PLT: 268 K/µl), CD4 lymphocyte count (40 cells/mm³) and viral load (424 copies/ml) was achieved after treatment completion.

We hereby present a case of a stage IV HIV patient with persistent dysphagia secondary to leishmanial and candidal esophagitis. Combined

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Figure 1. Esophagoscopy: rised whitish plaques on erythematous mucosa

therapy resulted in symptomatic relief with hematologic and virologic improvement.

The VL is associated with bone marrow, spleen, liver and lymph node involvement. Infection of the digestive tract may occur, in the setting of profound immunosuppression. *Leishmania* species can invade any part of the digestive tract, with the duodenum being most commonly affected. Their presence is frequently accompanied by other pathogens, such as cytomegalovirus or *Candida* species [1].

Esophageal infection in HIV patients usually indicates the onset of the symptomatic stage. The majority of cases are due to *Candida* species, but herpes simplex virus and cytomegalovirus may participate. Patients who are not taking antiretroviral therapy or have not attained immune reconstitution despite treatment and those with extremely low CD4 lymphocyte count are at high risk [1].

The current standard of care is to treat an AIDS patient complaining of dysphagia and/or odynophagia with systemic antifungal agents on the basis of compatible oropharyngeal lesions. If symptoms do not improve within the first few days, endoscopy and biopsy should be performed, since it is likely that a disease other than or in addition to *Candida* esophagitis is present, as in our patient.

To date, there have been only a few published reports of esophageal leishmaniasis in HIV patients [2-5]. Our case emphasizes the importance of endoscopy, pathology and histochemistry in identifying uncommon causes of persisting dysphagia in immunocompromised individuals.



Figure 2. A – Candidal hyphae (PAS 20×). B – Macrophage aggregates with intracytoplasmic granules; squamous epithelium overlying (hematoxylin-eosin 40×). C – Same as B (Giemsa 20×). D – Macrophages with intracytoplasmic blue granules (Giemsa 100×)

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