

CANDIDA ESOPHAGITIS: AN UNUSUAL DIAGNOSTIC SAMPLE

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A 65-year-old-woman was diagnosed with AML (M5a according to the FAB classification¹) in April 1993.

She received standard remission-induction therapy with daunorubicin (106 mg daily for three days), cytarabine (178 mg every 12 hrs for five days) and etoposide (178 mg daily for three days). Severe neutropenia ensued one week thereafter, and she developed fever of undetermined origin. Imipenem (500 mg every 6 hrs iv) was started on an empirical basis. Blood cultures were negative for bacteria and fungi. Nevertheless, fever persisted over the following five days. At this point she referred retrosternal pain that impeded her from swallowing, as well as nausea and blood-stained vomiting.

During one of these episodes she expelled membrane-like, rectangular-shaped fibrinous material 14.5 cm in length, 5 cm in width and 0.4 cm thick, which was initially interpreted as being denuded esophageal mucosa by her attending physicians. Pathologic examination after appropriate processing disclosed a mesh-work of fibrin densely infiltrated by yeasts highly suggestive of *Candida* ssp (Figure 1). A clinical diagnosis of candida esophagitis was made and fluconazole (200 mg as a single daily oral dose) was given. Her general status progressively improved, and the fever abated 10 days later in coincidence with recovery of the neutrophil count.

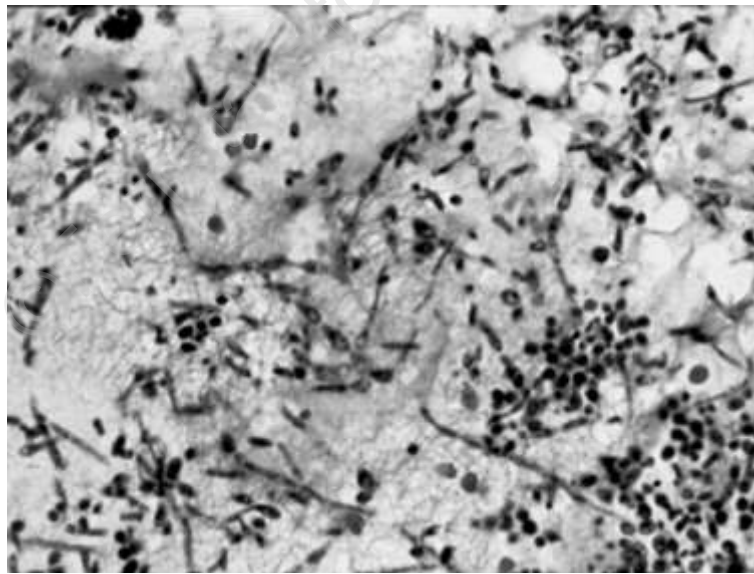


Figure 1. Yeastlike budding organisms and pseudohyphae.

Discussion

Candida esophagitis is a locally invasive infection of the esophagus produced by *Candida* ssp.² Although clinical and radiological findings may suggest this diagnosis (especially in HIV-infected patients), only endoscopic studies with culture and histologic tissue examination can establish it definitively. Patients who are not candidates for endoscopy are frequently treated with amphotericin B or, as in the present case, with an azole antifungal agent. In some cases acyclovir is also administered because of the possible coexistence of a herpetic esophagitis.

Factors predisposing to *Candida* infections include neutropenia,³ ulcerations of the oropharynx and along the gastrointestinal tract caused by cytotoxic agents, corticosteroid therapy and, probably, indwelling catheters, parenteral nutrition and the use of omeprazole.⁴

Chemotherapy-induced cytotoxicity and the lytic effects of the invasive fungal infection on the epithelium of the esophageal mucosa were probably the mechanisms responsible for the

appearance of this unusual sample, which allowed us to establish the diagnosis. A high index of clinical suspicion and accelerated pathologic processing of any suspicious material obtained from patients at risk⁵ may permit early treatment of candida esophagitis before systemic dissemination, chronic abscess formation or serious hemorrhagic complications can develop.

References

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