Esophageal actinomycosis – a rare cause of odynophagia in an HIV patient

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Abstract

Odynophagia is a common complaint among individuals living with HIV, with candidiasis being the predominant cause. We present a case of 38-year-old man with poorly controlled HIV who presented with odynophagia and oropharyngeal candidiasis that is refractory to antifungal therapy. Further investigations showed esophageal candidiasis with esophageal ulcers, which pathology revealed extensive filamentous bacteria in addition to fungal hyphae. Diagnosis of esophageal candidiasis superimposed with actinomycosis was made, and he responded well to amoxicillin in addition to fluconazole therapy.

Keywords: Esophageal actinomycosis, Actinomycosis, Actinomyces, HIV

Introduction

Odynophagia is one of the most common gastrointestinal complaints among poorly controlled HIV individuals. Candida esophagitis covers the majority of cases, with cytomegalovirus (CMV) and herpes simplex virus (HSV) infection responsible for most of the remaining infectious etiologies.[1] Other infectious causes apart from these are often overlooked by clinicians. We present a rare case of an individual with poorly controlled HIV infection with odynophagia and esophageal candidiasis not responding to fluconazole therapy. He was later found to have superimposed esophageal actinomycosis.

Case

A 38-year-old African American man with a past medical history of human immunodeficiency virus (HIV) infection with poor adherence to antiretroviral therapy (ART) and a history of neurosyphilis, methamphetamine abuse, and chronic anemia presented to the hospital with odynophagia for two weeks. He described throat pain that radiated down his chest, aggravated by swallowing, causing poor oral intake and a 20-pound weight loss. Regarding HIV infection, he was diagnosed ten years ago and had poor adherence to ART. The last ART regimen was Emtricitabine-Tenofovir-Dolutegravir, which was taken five months ago. On encounter, he was alert and oriented. Physical examination was remarkable for oral thrush with shotty bilateral cervical lymphadenopathy.

Initial investigations showed pancytopenia with WBC 3,630/μL, hemoglobin 8.9 g/dL (MCV 88.8 fL), and platelet 123,000/μL. Chest x-ray was unremarkable. Point-of-care Streptococcus Group A test was negative. Patient was found to have a viral load of 147,000 copies/mL and an absolute CD4 T cell count of 5 cells/μL. Other infectious screenings, including serum cryptococcal antigen, hepatitis B and C antibodies, *Neisseria gonorrhea* and *Chlamydia trachomatis* urine PCR, CMV IgM and IgG, serum 1–3 β-D-Glucan, and Coccidioides antibodies, were negative. RPR titer decreased from 1:1024 to 1:32 over the prior 18 months.

He was started on intravenous fluconazole for oral and suspected esophageal candidiasis. Atovaquone was started for PJP prophylaxis in the setting of pancytopenia. Active CMV retinitis was ruled out by an

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DOI: 10.12746/swjm.v13i57.1517

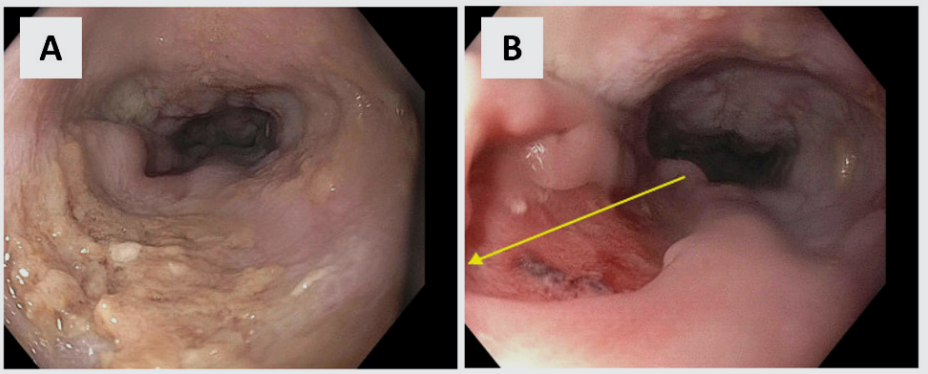

Figure 1. Esophagogastroduodenoscopy findings. **A:** Diffuse yellow plaque in the entire esophagus. **B:** Cratered non-bleeding esophageal ulcer at the middle third of the esophagus (yellow arrow).

ophthalmologist. EGD showed chronic gastritis with a diffuse yellow plaque in the entire esophagus, consistent with candidiasis, with two three-centimeter cratered non-bleeding esophageal ulcers (Figure 1).

Despite seven days of intravenous fluconazole therapy, he still complained of persistent chest pain after swallowing. Pathology from esophageal ulcers revealed budding yeast with hyphae and an extensive superimposed filamentous bacterial infection throughout the lesion (Figure 2). Staining for CMV and mycobacteria were negative. H. pylori was noted in gastric mucosa. Morphological characteristics of filamentous bacteria seen resembled *Actinomyces*. Unfortunately, bacterial culture was not sent. Given the extensivity and burden seen, treatment with amoxicillin 875 mg oral twice daily was started.

He reported improvement in symptoms after 7 days of amoxicillin initiation. He was discharged with three-week course of fluconazole and six-week course of amoxicillin. He was scheduled for a follow up and repeat EGD in three months but was lost to follow up.

Discussion

Actinomyces spp., a facultative anaerobic Gram-positive bacillus, is a slow-growing bacteria that resides commensally in the oral cavity, genitourinary tract, and

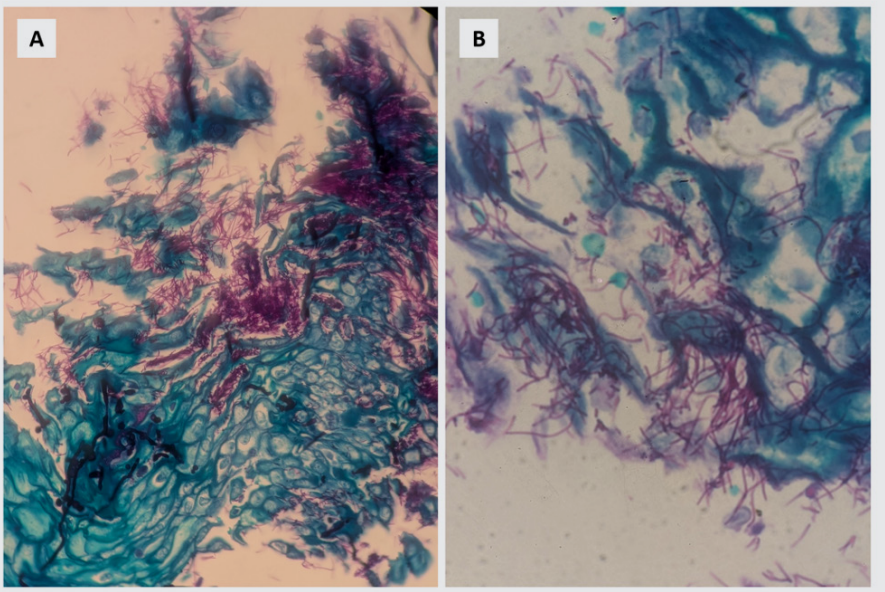

Figure 2. Pathology from the esophageal ulcer biopsy which filamentous bacterial colonies were identified along with fungal hyphae. **A:** Periodic acid–Schiff stain at 40x magnification. **B:** Periodic acid–Schiff stain at 100x magnification.

gastrointestinal tract. *Actinomyces israelii* is the most common species responsible for human illnesses.[2] Esophageal actinomycosis is an extremely rare etiology of esophageal infection, typically manifest as odynophagia and dysphagia. The literature has reported only 41 cases from 1953 to the present, predominantly occurring in immunocompromised individuals, accounting for around 60% of all cases.[3] For these organisms to be pathogenic, an initial disruption of the mucosal barrier is necessary, typically resulting from invasive surgical operations or trauma, which facilitates bacterial infiltration at the infection site.[2] Esophageal actinomycosis may present similarly to esophagitis, esophageal mass, and esophageal ulcer. It could potentially mimic clinical signs of neoplasia, as seen by the presentation of odynophagia accompanied by weight loss in our case.

Esophageal actinomycosis has been reported in all age demographics among both male and female, with a predominance in male – approximately 70% of all documented cases.[3] It was found in both immunocompetent individuals (40%) with a history of invasive procedures and immunocompromised patients (60%).[3] According to prior cases, among immunocompromised individuals diagnosed with esophageal actinomycosis, 43% had underlying HIV infection, 38% had underlying malignancy, 14% had systemic autoimmune disease, and one patient had a history of organ transplantation.[3] In our case, it was possible that candidiasis caused initial disruption of mucosal barrier and resulted in invasive actinomyces infection. EGD predominantly revealed erosive lesions with varied degrees of hyperemic borders or extensive ulceration covered by necrotic tissue.[3] EGD findings may resemble candidiasis, and patient was often treated with antifungal therapy, as in our scenario. This also underscored the necessity to further investigate for actinomycosis, which necessitates histological investigation and bacterial culture of biopsied tissues following failure of standard antifungal therapy. Histological examination typically shows inflammation of the mucous membranes characterized by the presence of aggregates of Gram-positive filamentous bacteria.

In HIV patients, cases of esophageal actinomycosis have predominantly been observed in patients with CD4 T-cell counts below 200 cells/μL, including our patient.[4–6] Despite being rare, invasive actinomycosis has been identified as a surrogate sign for poor prognosis in immunocompromised patients.[7] This further underscores the necessity for differential diagnosis of esophageal candidiasis in HIV patients with odynophagia, particularly following the failure of antifungal therapy. The clinical response and outcome of actinomycosis treatment with penicillin therapy, ranging from 6 to 12 months, correlated well with the good response in improving symptoms in our case and in reducing the size of esophageal mass in the previously reported cases.[6,8,9]

Conclusion

In conclusion, although rare, esophageal actinomycosis is a potential cause of odynophagia in individuals with HIV. It may present as a solitary infection or as a co-infection with other common pathogens. A high degree of clinical suspicion is required for diagnosis, as *Actinomyces spp.* is a commensal bacterium and a facultative anaerobe, making it challenging to culture.

Acknowledgments

The patient provided consent for this publication. The authors would like to thank Texas Tech University Health Sciences Center for its support and resources that made this case report possible. We are grateful for the institution's commitment to clinical education and research.

Article citation: Thiravetyan B, Sodsri T, Puchongmart C, Nichols J. Esophageal actinomycosis – a rare cause of odynophagia in an HIV patient. The Southwest Journal of Medicine 2025;13(57):49–52
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Submitted: 7/6/2025
Accepted: 9/10/2025
Conflicts of interest: none

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