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Case Report

Dual infection of the esophagus in an immunocompetent individual

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Abstract

Background: Infectious esophagitis is predominantly observed in immunocompromised individuals, with Candida albicans, herpes simplex virus, and cytomegalovirus being the most common pathogens. Dual infection involving both Candida and Herpes simplex is exceedingly rare, especially in immunocompetent individuals.
Case Presentation: 50-year-old male with type 2 diabetes mellitus and coronary artery disease presented with progressive dysphagia, odynophagia, chest pain, and weight loss for 6 months. Endoscopy revealed oesophageal ulcers and whitish mucosal plaques throughout the oesophagus. Initial histopathology confirmed Candida infection, and the patient was treated with fluconazole. Due to persistent symptoms and suspicion of other dual aetiology, a repeat endoscopy and biopsy of oesophageal ulcer was performed, which revealed histological features consistent with HSV esophagitis. Serum HSV IgG was positive, and the patient responded well to intravenous acyclovir.
Conclusion: This case underscores the importance of considering dual infections in patients with esophagitis, even in the absence of overt immunosuppression. Early endoscopic evaluation with repeat biopsies, when clinically indicated, is crucial for accurate diagnosis and appropriate treatment.

Keywords: Herpes simplex virus (HSV), Candida, Infectious esophagitis

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1. Introduction

Infectious oesophagitis is an inflammatory condition of the oesophageal mucosa, most commonly seen in immunocompromised individuals. The leading causes include Candida albicans, herpes simplex virus (HSV), and cytomegalovirus (CMV).[1] They can occasionally occur in immunocompetent individuals, often in the setting of chronic illnesses like uncontrolled diabetes mellitus, prolonged hospitalization, or use of certain medications.[2] Dual oesophageal infections with both Candida and HSV are rare, particularly in patients without overt immunodeficiency. Most reported cases occur in individuals with compromised immunity due to chemotherapy, radiotherapy, or critical illness.[3] We present a rare case of concurrent Candidal and HSV esophagitis in a middle-aged man with well-controlled diabetes mellitus and no apparent immunosuppression. This case highlights the importance of considering infectious aetiologies even in seemingly immunocompetent individuals presenting with persistent oesophageal symptoms, and it underscores the critical role of endoscopy and histopathology in establishing an accurate diagnosis.

2. Case Presentation

A 50-year-old gentleman with a 5-year history of diabetes mellitus and coronary artery disease, with a coronary artery stent placed one year ago, presented to our clinic with the chief complaints of chest pain for the past 6 months, intermittent low-grade fever over the last 4 months, and progressive dysphagia and odynophagia, more pronounced with solids than liquids. He also reported an 8 kg weight loss over the past 6 months, accompanied by decreased appetite.

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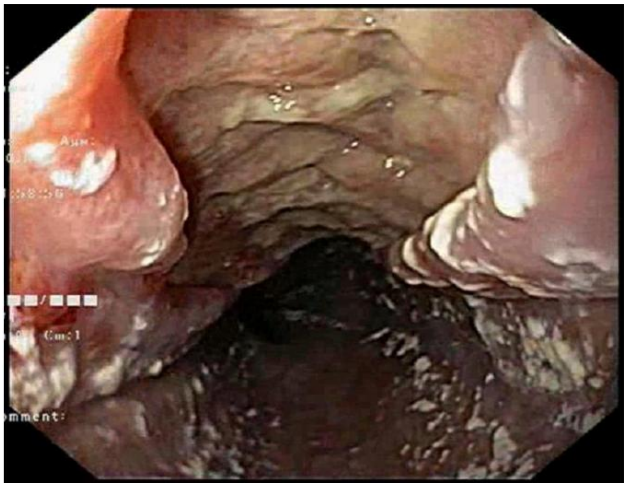

Figure 1: Endoscopic image showing longitudinally oriented deep ulcer with base of ulcer covered with thick yellowishwhite exudate and surrounding candidiasis

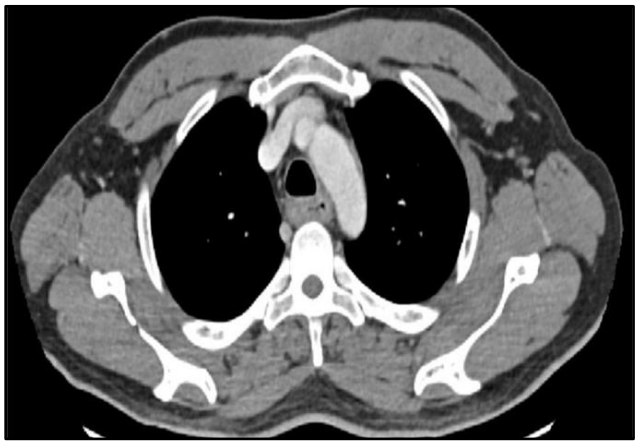

Figure 2: CECT chest showing thickened oesophageal wall

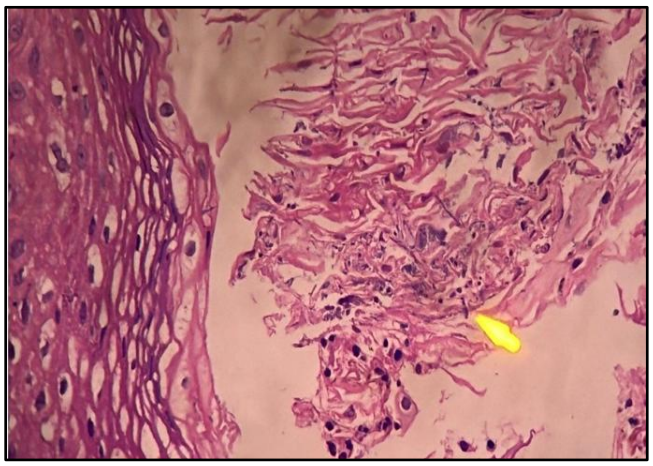

Figure 3: Low power haematoxylin and eosin-stained section showing ulcerated fragment of squamous mucosa with prominent candidal colonies (arrow)

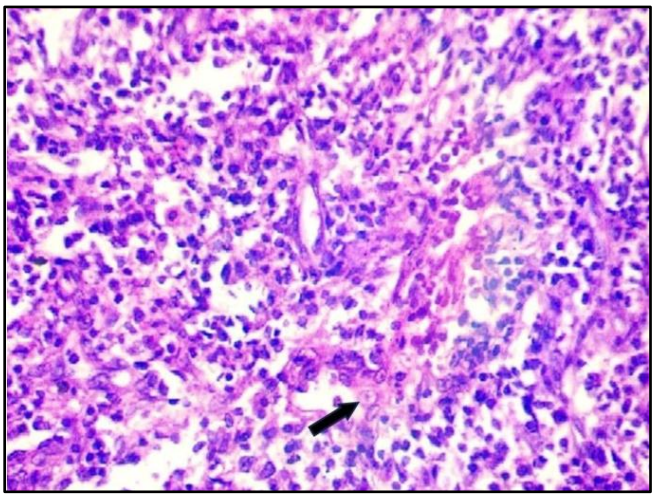

Figure 4: Low power haematoxylin and eosin-stained section showing acute inflammatory cell infiltrate and infected epithelial cells

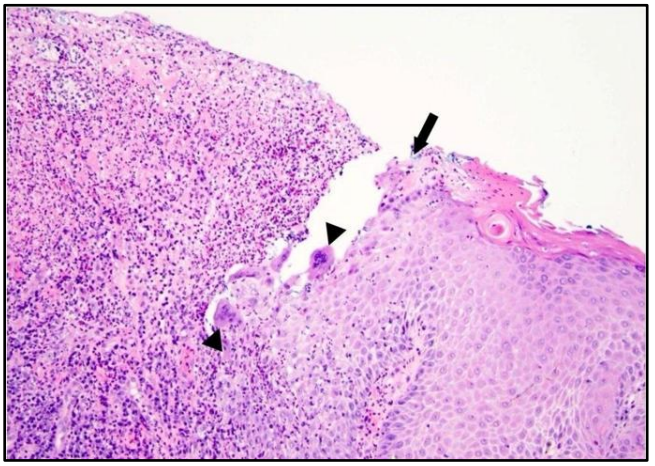

Figure 5: Low power haematoxylin and eosin-stained section showing ulcerated epidermis, acantholysis (arrow), nuclear margination, molding and multinucleation (arrow head)

There was no history of tuberculosis contact, cough with sputum production, joint pains, oral ulcers, diarrhoea, or skin lesions. He has been on antidiabetic medications (metformin and glimepiride), dual antiplatelet therapy (aspirin and clopidogrel), and atorvastatin. His HbA1c at the time of admission was 6.8%, compared to 7.2% ten months earlier. Initially, he consulted multiple practitioners and was treated with several courses of antibiotics, proton pump inhibitors, and antacid syrups, but experienced no improvement in his symptoms. His general physical examination was unremarkable. Routine investigations like hemogram, liver function test, kidney function test and viral screening (HBV/HCV/HIV) were normal.(**Table 1**)

Table 1: Summary of diagnostic investigations

Parameter(units)	Value	Normal range
Hemoglobin (gm/dL)	11.5	13-16
White blood cell count	10500	4000-11000
Differential count(N/L/M/E/B)	60/30/10/-/-	
Platelet count	3.5	1.5-4
ESR (mm/hr)	98	0-15
Creatinine(mg/dL)	1	0.6-1.2
Total bilirubin (mg/dL)	0.8	<1.2
Albumin (gm/dL)	3.4	<0.4
AST (IU/L)	34	20-40
ALT(IU/L)	28	20-40
ALP(IU/L)	112	30-120
Calcium (mg/dL)	8.8	8.8-10.4
PT	12	10-12
INR	1.1	0.9-1.1
HbA1c (%)	6.8	<5.6
HBsAg	Negative	
Anti HCV	Negative	
HIV	Negative	
IGRA	Negative	
Mantoux test	Negative (4mm)	
CMV-IgM	Negative	
CMV-IgG	Negative	
HSV-IgM	Negative	
HSV-IgG	Positive	
ANA	Negative	
Anti Ds DNA	Negative	
ANCA	Negative	

The Upper GI endoscopy was done which suggested multiple whitish plaques of variable sizes throughout esophagus along with longitudinally extended deep ulcer with base of the ulcer covered with thick yellowish white exudates, extending from 23cm to 28cm from upper incisors and another small round ulcer with surrounding friable mucosa noted at 20cm (**Figure 1**). Multiple biopsies were taken separately from both edge and base of the ulcer for HPE and gene Xpert. The CECT chest was done which suggested edematous wall thickening of thoracic esophagus with mild enhancing wall extending from carina to retrocardiac region along with few mediastinal lymph nodes largest measuring about 9x6mm (**Figure 2**).

The tissue gene Xpert from the ulcer came out to be negative. He started on oral fluconazole 400mg on day 1 followed by 200mg once daily for 14 days along with rabeprazole 20mg sachet twice daily and syrup sucralfate-oxetacaine 10ml every 6 hours. HPE suggests prominent ulceration and florid granulation tissue with candidal infection with no evidence of dysplasia, granulomas, and inclusion bodies (**Figure 3**). In view of persistent symptoms and suspicion of dual pathology, repeat endoscopy done after 12 days shows absent candidiasis and repeat biopsy taken from ulcer. In view of nutritional compromise of the patient due to dysphagia and odynophagia we placed a nasogastric tube under endoscopic guidance. Blood workup was sent for tuberculosis, vasculitis, connective tissue disorders, CMV and HSV.

Histopathological examination of repeat biopsy showing nuclear molding, margination and multinucleation of infected cells suggestive of viral (HSV) esophagitis (**Figure 4** & **Figure 5**). Tissue PCR and immunohistochemistry were not performed due to the unavailability of these diagnostic modalities at our centre. HSV IgG antibody test positive and other workup for TB, CMV and vasculitis turnout to be negative. Considering the commonality of HSV as a cause of infectious esophagitis, along with supportive histopathological findings and positive HSV antibody results, a diagnosis of HSV esophagitis was made. The patient was started on intravenous acyclovir at a dose of 5 mg/kg every 8 hours for 14 days. On day 4, the patient showed significant improvement in fever, chest pain and odynophagia. The nasogastric tube was removed, and oral intake was resumed. After completing 7 days of intravenous acyclovir, the patient was transitioned to oral acyclovir and subsequently discharged. Follow-up was advised after one week; however, the patient was lost to follow-up.

3. Discussion

Esophagitis is an inflammatory condition of the oesophageal mucosa. There are many causes of esophagitis and typically presenting with symptoms such as heartburn, chest pain, odynophagia and dysphagia. The most common cause of esophagitis is gastroesophageal reflux disease.[4] Other causes of esophagitis include infections, pill esophagitis, eosinophilic esophagitis, corrosive ingestion, radiation, Crohn's disease etc. History and examination are essential for making an accurate diagnosis. In the index case, the presence of fever along with the endoscopic findings raised a strong suspicion for an infectious aetiology. Infectious esophagitis is the third leading cause of esophagitis behind gastroesophageal reflux disease and eosinophilic esophagitis and can be caused by bacterial, viral, fungal, tubercular and parasitic infections.[5] Oesophageal candidiasis is the most common cause of infectious esophagitis. Among patients with infectious esophagitis, approximately 88% of cases are due to candida albicans, 10% to herpes simplex virus, and 2% to cytomegalovirus.[1] Infectious oesophagitis is common in immunosuppressed patients but has rarely been reported in immunocompetent individuals.

With the advent of organ transplantation, the use of immunosuppressive therapies for rheumatologic diseases and inflammatory bowel disease (IBD), and the emergence of acquired immunodeficiency syndrome (AIDS), oesophageal infections have become an increasingly common medical concern. Other risk factors for infectious esophagitis includes

uncontrolled diabetes, congenital immunodeficiencies (especially chronic mucocutaneous candidiasis), haematological malignancies, chronic antibiotic use etc.[6] The index patient is a known case of diabetes mellitus, currently on oral hypoglycaemic agents, with reasonably well-controlled blood glucose and HbA1c levels and not taking any immunosuppressive drugs.

Oesophageal candidiasis (EC) is the most common oesophageal infection caused mainly by candida albicans. Candida is a common commensal organism found on human skin and within the gastrointestinal tract. While it typically causes infections in immunocompromised individuals, it can also affect patients without evident immunodeficiency, especially those with renal or liver dysfunction or those who have prolonged stays in intensive care settings.[7] Esophagoscopy is the preferred diagnostic modality for candida esophagitis. It allows direct visualization of the oesophageal mucosa, typically revealing white plaques or exudates that adhere firmly and cannot be removed with water irrigation. In some cases, mucosal breaks or ulcerations may also be observed.[8] Biopsy or brushings from the lesions reveals yeasts and pseudo hyphae. Treatment consists of oral fluconazole, 200–400 mg (3–6 mg/kg) daily, for 14–21 days is recommended.[9] In our patient oesophageal candidiasis manifested as multiple whitish plaques of variable sizes scattered throughout the oesophagus, which showed improvement after oral fluconazole treatment.

HSV esophagitis is the second most common cause of infectious esophagitis, typically occurs in immunocompromised individuals, either as a primary infection or due to reactivation of a latent virus. Its occurrence in immunocompetent individuals is uncommon(2).[10] The gross appearance of lesions can vary based on the timing of endoscopy. In the early stages, vesicles may be observed, which typically rupture and evolve into well-defined, circumscribed ulcers. These ulcers may appear punched-out or volcano-like, and in some cases, may merge to produce a cobblestone or shaggy ulcerative pattern. The disease usually affects the distal or mid-oesophagus and, at times, the entire oesophagus. Microscopically, biopsies from the edge of ulcers provide the best diagnostic yield.[11] Histological features of HSV esophagitis on H&E staining typically include multinucleated giant cells, with nuclear molding, chromatin margination, and the presence of characteristic Cowdry type A inclusion bodies.[12] In the index patient, one ulcer was deep and longitudinally oriented, whereas the other was small, round, and superficial. Histopathological examination of biopsy from ulcer showed multinucleation with nuclear molding and chromatin margination. Although intranuclear inclusions typically specific for HSV infection were not observed, the diagnosis was supported by positive serum HSV-specific antibodies along with suggestive histological findings and prompt clinical response to antiviral therapy.

Concurrent oesophageal infection with both HSV and candida species is uncommon. Only a limited number of cases have been documented, mostly in adults receiving chemotherapy or radiotherapy for malignancies, as well as in those with sepsis.[13] Rarely, it may occur in healthy young immunocompetent individuals.[14-15] Reports of dual oesophageal infections in adults suggest that HSV may initially damage the oesophageal epithelium, compromise the mucosal barrier and thereby facilitate secondary colonization or infection by candida species.

4. Conclusion

Infectious esophagitis may also develop in immunocompetent individuals. This case illustrates a rare instance of dual oesophageal infection involving candida albicans and herpes simplex virus (HSV) in an otherwise immunocompetent adult. Early endoscopic evaluation with targeted biopsies remains essential for accurate diagnosis and effective management. Collaboration between gastroenterologist and pathologist was pivotal in achieving favourable outcomes especially in resource limiting setting where IHC and tissue PCR is not available.

5. Source of Funding

None.

6. Conflict of Interest

None.

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