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Two sides of the same coin: eosinophilic and herpetic esophagitis in an immunocompetent young adult

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Statement of ethics

Informed consent was obtained from the patient for the publication of her information and imaging in this case report.

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Keywords: Eosinophilic esophagitis. Herpetic esophagitis. Infectious esophagitis.

Dear Editor,

We report a case of a 30-year-old woman with an 8-year diagnosis of eosinophilic esophagitis (EoE) treated with swallowed fluticasone propionate throughout this period. She presented to the emergency room with a two-day history of severe odynophagia, aphagia, retrosternal pain, and fever. The patient was febrile and hemodynamically stable, with no visible oropharyngeal lesions. She presented with elevated C-reactive protein (37 mg/L). An esophagogastroduodenoscopy was performed, which revealed white plaque-like lesions with "volcano-like" shallow ulcerations with raised edges on the distal esophagus (Figure 1a&b). Multiple biopsies were taken from both the center and edges of the lesions. The patient was empirically started on intravenous fluconazole due to the suspicion of candida esophagitis. However, the patient's symptoms worsened over the next two days, and acyclovir at a dose of 5 mg/kg was started. The initial work-up showed a positive titer for Herpes

Simplex Virus (HSV)-2 IgM (1.6 U/L) and a negative titer for IgG (2.24 U/L), as well as a negative serological study for HSV-1, cytomegalovirus, and human immunodeficiency virus (HIV). Histological examination revealed multinucleated giant cells with nuclear molding and chromatin margination and cells with "ground glass" nuclei, along with typical Cowdry type A intranuclear inclusion bodies and immunohistochemical staining for HSV type 2, confirmed the diagnosis of herpetic esophagitis (Figure 1c&d). The patient experienced rapid improvement and was discharged on oral acyclovir therapy at 400 mg/day, completing a total of 14 days of treatment with a total resolution of symptoms.

Discussion

Herpetic esophagitis (HSE) is common in immunocompromised patients, those with HIV or immunosuppressive treatments. However, it is rare in immunocompetent hosts, even rarer when caused by HSV type 2. It should be suspected with the acute onset of odynophagia, heartburn, and fever[1]. Endoscopically, multiple coalescent ulcers with a "volcano-like" appearance are typical, most commonly in the distal esophagus[2]. As highlighted by this case, plaque-like lesions resembling candida esophagitis may also occur, making endoscopic diagnosis challenging. In our case, the diagnosis of HSE was suspected from endoscopic findings, the lack of response to azole therapy, and confirmed by histology and immunohistochemical staining[3].

EoE, through esophageal mucosal inflammation, and the use of swallowed fluticasone propionate can predispose individuals to infections by candida and viruses such as HSV[3–5]. This case highlights that HSE and EoE can coexist in the same patient. Therefore, clinical suspicion, endoscopic examination, and histopathological evaluation are fundamental for correct diagnosis and implementing targeted treatment.

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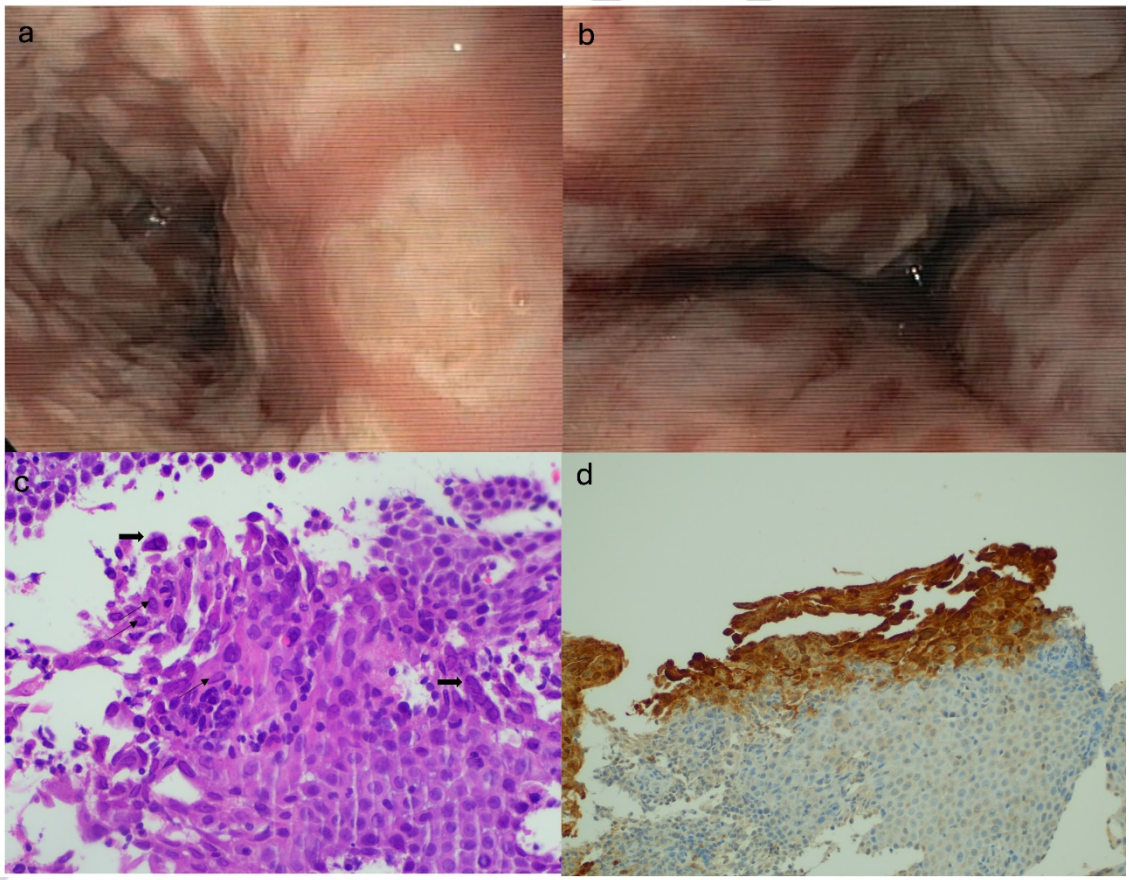

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Figure 1. **(a/b)** The distal esophagus exhibits the presence of white plaque lesions and multiple superficial ulcers; **(c)** Esophageal epithelial cells showing classic histological changes of HSV infection, namely nuclear chromatin margination, multinucleation, and nuclear molding (➡) . Cowdry type A nuclear inclusions (→) can also be observed. (H&E, 40x). **(d)** Immunohistochemical study revealing epithelial cell immunoreactivity for Herpes Simplex Virus (HSV). (H&E, 20x)