



## Eosinophilic Esophagitis induced by semaglutide use: A case report

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### Abstract

We report the case of a 46-year-old male patient undergoing treatment with a GLP-1 receptor agonist (semaglutide) for six months, who developed progressive dysphagia to solids and a retrosternal foreign body sensation. An upper endoscopy revealed impacted food at 40 cm from the dental arch, which was successfully advanced into the stomach. At the same location, three small fibrin-covered ulcers were identified. Histopathological analysis of esophageal biopsies confirmed the diagnosis of eosinophilic esophagitis. Follow-up evaluations at one and three months showed the patient remained asymptomatic, adherent to an allergen-free restrictive diet and proton pump inhibitor therapy, without resuming semaglutide and without requiring corticosteroid treatment. This case highlights the importance of recognizing potential adverse effects of pharmacological agents, even when they are rare or scarcely reported in the literature. Such awareness may improve patient outcomes and provide valuable insight for clinicians managing similar presentations.

**Keywords:** Eosinophilic Esophagitis; Semaglutide; GLP-1 Receptor Agonist; Drug-Induced Esophagitis

### Introduction

Eosinophilic esophagitis (EoE) is a chronic, immune-mediated inflammatory condition triggered by allergens. It is characterized by esophageal dysfunction accompanied by a predominant eosinophilic infiltration of the esophageal mucosa. EoE is now recognized as an adaptive immune disease mediated by type 2 helper T cells (Th2), arising when allergens interact with a compromised esophageal barrier—whether due to intrinsic or acquired defects. In this process, eosinophils serve as key effector cells, contributing to inflammation, tissue remodeling, and fibrosis, ultimately leading to the characteristic clinical manifestations of the disease [1].

In recent years, various glucagon-like peptide-1 receptor agonists (GLP-1 RAs), including lixisenatide, liraglutide, dulaglutide, albiglutide, and semaglutide, have been approved and widely implemented in clinical practice for the management of diabetes mellitus and certain forms of obesity. However, as their use has expanded, isolated cases of eosinophilic reactions associated with these agents have been reported. These include peripheral eosinophilia, bullous pemphigoid with eosinophilic infiltrate, acute interstitial nephritis, eosinophilic panniculitis, and eosinophilic hepatitis, among others [1].

The clinical presentation of eosinophilic esophagitis (EoE) varies depending on the patient's age. In adolescents and adults, the most common manifestations include dysphagia and food impaction, although heartburn and chest pain may also occur<sup>2</sup>. The presence of atopic conditions—such

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as immediate-type food allergies, asthma, eczema (atopic dermatitis), and allergic rhinitis—should be considered a risk factor for the development of EoE. It is estimated that 60% to 80% of patients with EoE have associated allergic disorders, and the likelihood of developing this condition increases in individuals with multiple atopic comorbidities [2].

This report describes the case of a 46-year-old male patient undergoing treatment with *Rybelsus* (semaglutide) for six months, who presented with an acute episode of food impaction. An upper gastrointestinal endoscopy was performed, during which the impacted food bolus was successfully dislodged. Endoscopic examination revealed findings suggestive of eosinophilic esophagitis, which was subsequently confirmed through histopathological analysis of esophageal biopsy specimens [2].

## Objective

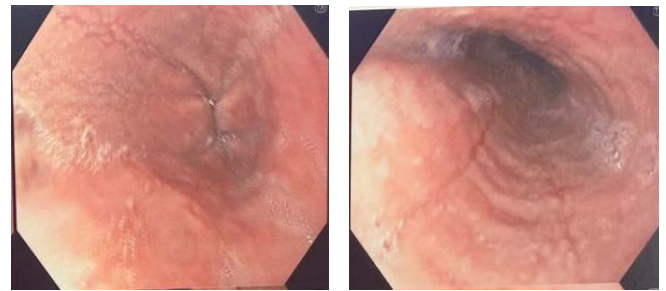
The purpose of this report is to highlight eosinophilic esophagitis as an uncommon adverse event following the administration of a glucagon-like peptide-1 receptor agonist (GLP-1 RA), and to describe its diagnostic and therapeutic approach.

## Case presentation

A 46-year-old male with a history of grade I obesity was undergoing treatment with oral semaglutide 7 mg daily (*Rybelsus*). Two months prior to admission, he experienced a self-limited episode of dysphagia to solids that did not prompt medical evaluation. Four hours before presentation, he developed food impaction (chicken with vegetables), accompanied by a retrosternal foreign body sensation. He denied drooling, cough, or vomiting. Physical examination revealed no pathological findings.

An upper endoscopy was performed with the following findings: a tubular esophagus with diffuse mucosal edema and trachealization that resolved with maximal insufflation. Longitudinal furrows were observed in the middle and lower thirds. At the esophagogastric junction, 40 cm from the dental arch, impacted food was visualized and successfully advanced into the stomach. At the same site, three small fibrin-covered ulcers were identified. The gastric mucosa appeared normal, with mild erosions in the antrum and a centrally located pylorus. The fundus was unremarkable, and the hiatal opening was closed, confirming the presence of the three ulcers (Figure 1). The duodenum was normal.

Three biopsies were obtained: (1) antral and incisural mucosa; (2) ulcers at the esophagogastric junction; and (3) distal and proximal esophageal mucosa. The presumptive diagnosis was resolved food bolus impaction in the distal esophagus, probable eosinophilic esophagitis (E1F1E0R1S0 classification), and Forrest III ulcers at the esophagogastric junction.



**Figure 1:** Upper endoscopy findings

Endoscopic images demonstrate esophageal mucosal edema, longitudinal furrows in the mid and distal esophagus, and three small fibrin-covered ulcers located at the esophagogastric junction. These findings are consistent with eosinophilic esophagitis.

Laboratory tests were obtained, revealing normal parameters except for hemoglobin of 13 g/dL, eosinophils at 9%, and serum potassium of 3.46 mmol/L. Semaglutide was immediately discontinued, and treatment was initiated with intravenous esomeprazole 40 mg every 12 hours, metoclopramide 10 mg every 8 hours, and acetaminophen 1 g every 8 hours. During hospitalization, oral intake was progressively resumed, beginning with clear liquids and advancing to a soft, allergen-free diet (excluding dairy, gluten, shellfish, nuts, and soy). Upon discharge, the patient was prescribed esomeprazole 40 mg once daily in the morning, famotidine 40 mg once daily in the evening, and metoclopramide 10 mg every 8 hours.

The final pathology report from esophageal biopsy described fragments of gastric-type mucosa lined by simple columnar, mucin-secreting epithelium forming foveolar structures and glands with reactive nuclear changes. These were associated with extensive areas of ulceration, fibrin deposition, recent hemorrhage, and a predominantly acute inflammatory infiltrate. The underlying lamina propria showed edema, vascular congestion, and a mixed inflammatory infiltrate composed of lymphocytes, plasma cells, eosinophils (15–20 per high-power field), and neutrophils [3–5].

At follow-up visits at 1 and 3 months, the patient remained asymptomatic, adherent to an allergen-free restrictive diet and proton pump inhibitor therapy. Semaglutide was not reintroduced, and topical corticosteroids were not required. A follow-up endoscopy is planned in three months.

## Discussion

GLP-1 receptor agonists (GLP-1 RAs) are associated with a range of adverse effects, the most commonly reported being nausea, vomiting, and diarrhea. Less frequently, serious adverse events such as pancreatitis, acute kidney injury, and hypoglycemia have been documented. Isolated cases of eosinophilic reactions during GLP-1 RA therapy have also

**Table 1.** Case Reports of Eosinophilic Disorders Associated with Semaglutide Use

Case	Age	Sex	Adverse effect	Improvement after drug withdrawal
1	61	F	Bullous pemphigoid with subepidermal vesicles and a mixed dermal infiltrate rich in eosinophils <sup>5</sup> .	Yes
2	83	F	Acute kidney injury with lymphoplasmacytic and eosinophilic interstitial infiltrate <sup>5</sup> .	Yes
3	30	M	Acute interstitial nephritis with irregular inflammatory infiltrate (lymphocytes, plasma cells, mild eosinophils) <sup>5</sup> .	Yes
4	42	F	Eosinophilic fasciitis with peripheral eosinophilia (absolute count 950/mm <sup>3</sup> ); lower limb MRI showed post-contrast enhancement of all superficial and deep fascial planes <sup>5</sup> .	Yes
5	75	F	Subepidermal vesicles on bullous lesions of the thighs, back, and chest with eosinophilic infiltrate and no evidence of vasculitis <sup>4</sup> .	Yes
6	74	M	Rash with erythematous plaques on flanks and abdomen with superficial and deep perivascular inflammatory infiltrate predominantly composed of eosinophils; no epidermal damage or vasculitis <sup>4</sup> .	Yes
7 (present case)	46	M	Esophageal lamina propria with edema, vascular congestion, acute and chronic inflammatory infiltrate composed of lymphocytes, plasma cells, eosinophils (15–20 per high-power field), and neutrophils.	Yes
Ref [5]				

been reported, including peripheral eosinophilia, bullous pemphigoid with eosinophilic infiltrate, acute interstitial nephritis, eosinophilic panniculitis, and eosinophilic hepatitis. Given the established link between GLP-1 RAs and eosinophilic responses, it is essential to further explore the full spectrum of immune-mediated manifestations, such as eosinophilic esophagitis, as illustrated in this case. As the use of GLP-1 RAs continues to rise, so does the likelihood of encountering eosinophilic adverse effects [2].

The diagnosis of eosinophilic esophagitis (EoE) is based on three fundamental criteria: (i) the presence of symptoms indicative of esophageal dysfunction; (ii) the identification of an eosinophilic infiltrate of at least 15 eosinophils per high-power field (eos/hpf) on esophageal biopsy; and (iii) the exclusion of other non-EoE conditions that could cause or contribute to esophageal eosinophilia [2].

## Conclusion

Eosinophilic esophagitis represents a rare but plausible immune-mediated adverse reaction to GLP-1 receptor agonists such as semaglutide. In this case, the temporal relationship between drug initiation and symptom onset, histological confirmation, and clinical improvement following drug discontinuation support a causal association. As the clinical use of GLP-1 analogs continues to expand, healthcare providers should maintain a high index of suspicion for eosinophilic complications in patients presenting with esophageal symptoms. Early recognition and appropriate management, including drug withdrawal and dietary modification, can lead to favorable outcomes without the need for corticosteroid therapy.

## Conflict of interest disclosure

None of the authors have any conflicts of interest to disclose

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## Ethics statement

- “Institutional review board approval was not required for a single-patient case report in our hospital Christus Muguerza Cumbres, Monterrey, México, and the report was prepared in accordance with institutional ethical guidelines.”
- “This case report was not required to be registered on a research registry.”
- “No experiments have been carried out on humans or animals for this research.”

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