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Case report

NSAID-Induced Esophagitis: An Uncommon Cause of Severe Eosinophilic Infiltration

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Abstract

Background: Drug-induced esophagitis is an underdiagnosed condition, largely due to the inadequate clinical assessment these patients often receive. A thorough medical history is essential for establishing a correct diagnosis. Early identification is crucial for proper management and counseling, helping to prevent recurrent episodes that may significantly impair quality of life. Upper endoscopy and histopathological examination are fundamental to clarifying the diagnosis and excluding other conditions with similar presentations. Severe eosinophilic infiltration, although uncommon in drug-induced esophagitis, may resemble other disorders such as eosinophilic esophagitis. **Case report:** We present the case of a young patient who developed severe dysphagia following exposure to nonsteroidal anti-inflammatory drugs (NSAIDs). Upper endoscopy revealed pronounced inflammation with marked eosinophilic infiltration on histopathological evaluation. Symptoms resolved fully after discontinuing NSAIDs and initiating proton pump inhibitor therapy, with no recurrence over the medium- to long-term follow-up. **Conclusions:** Drug-induced esophagitis should be recognized as a potential cause of severe eosinophilic infiltration and may even mimic the endoscopic and histologic profile traditionally associated with other conditions, such as eosinophilic esophagitis.

Keywords

Esophagitis, nonsteroidal anti-inflammatory agents, eosinophilic esophagitis.

INTRODUCTION

Several drugs have now been identified as responsible for the development of esophagitis of varying degrees of severity, the most common being antibiotics (primarily tetracyclines) and nonsteroidal anti-inflammatory drugs (NSAIDs). There is no consensus defining the clinical-epidemiological profile of this condition; however, most studies agree that an atopic component in genetically predisposed individuals is a frequent feature in this group of patients[1–9].

The most characteristic clinical presentation of this condition is retrosternal pain and dysphagia of recent onset following ingestion of the potentially causative drug. In most cases, the disease is self-limited and without serious complications; however, certain pharmacologic groups, such as NSAIDs, have been associated with more severe (gastrointestinal bleeding and/or esophageal stricture) and prolonged symptoms. Upper gastrointestinal endoscopy is currently considered the best diagnostic modality to characterize and analyze the esophageal lesions produced. The most frequent findings include superficial ulcers with regular margins and an inflammatory appearance, generally located in the middle and upper thirds of the esophagus, classically described in the literature as "kissing ulcers," often accompanied by erythematous and congestive areas. However, it is unusual to find whitish longitudinal furrows,

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which represent the endoscopic manifestation of severe eosinophilic infiltration of the esophageal mucosa[10–13].

Histologic examination is very useful because it helps exclude other entities such as malignant neoplasms. In most cases, it shows only an acute inflammatory reaction composed of a marked neutrophilic infiltrate, with a severe eosinophilic component being rarely observed in the examined samples[14–18].

Here, we present a case of NSAID-induced esophagitis that exhibited a severe eosinophilic inflammatory component with endoscopic representation and responded successfully to treatment with proton pump inhibitors in association with withdrawal of the offending drug.

CASE DESCRIPTION

A 23-year-old male patient with a history of allergic rhinitis presented with the sudden onset, following the use of NSAIDs (naproxen) prescribed for an odontogenic condition, of moderate-intensity retrosternal burning pain associated with dysphagia for both solid and liquid foods (Atkinson dysphagia scale: 4). The complete blood count showed peripheral blood eosinophils of 395.5 eosinophils/μL (normal range: ≤500 eosinophils/μL) and a serum immunoglobulin E (IgE) level of 189.8 IU/mL (normal range: 0–99.9 IU/mL).

Upper gastrointestinal endoscopy revealed a superficial ulcer with an inflammatory appearance and regular, congestive margins, located in the middle third of the esophagus and involving approximately 40% of the circumference (**Figure 1A**). Multiple whitish exudates were also observed in the lower third of the esophagus, associated with erosions and erythematous areas (**Figures 1B** and **1C**).

Histologic analysis showed a marked eosinophilic infiltrate (20 eosinophils/HPF), predominantly involving the epithelium, lamina propria, and submucosa (**Figure 2**). NSAIDs were discontinued, and treatment with proton pump inhibitors was initiated, with clinical, endoscopic, and histologic remission observed after eight weeks, without recurrence.

DISCUSSION

At present, there is limited scientific evidence demonstrating the true clinical impact of this condition in the affected population. Likewise, there are no international consensuses that clearly define diagnostic and therapeutic guidelines for drug-induced esophagitis. Our study consolidates the knowledge established to date, but with an atypical endoscopic-histologic profile, mimicking other clinical entities associated with esophageal eosinophilia.

A major challenge for the gastroenterologist is distinguishing drug-induced esophagitis from eosinophilic esophagitis when atypical variations are present in some cases. The most recent guidelines on the diagnosis and treatment of eosinophilic esophagitis have clearly established the endoscopic and histologic criteria for identifying this condition[1–5]. However, to date, there is no international consensus defining the parameters for drug-induced esophagitis, as most of the available scientific evidence is based solely on case reports[6–13]. Consequently, when a combination of similar endoscopic and histologic criteria exists for these two cli-

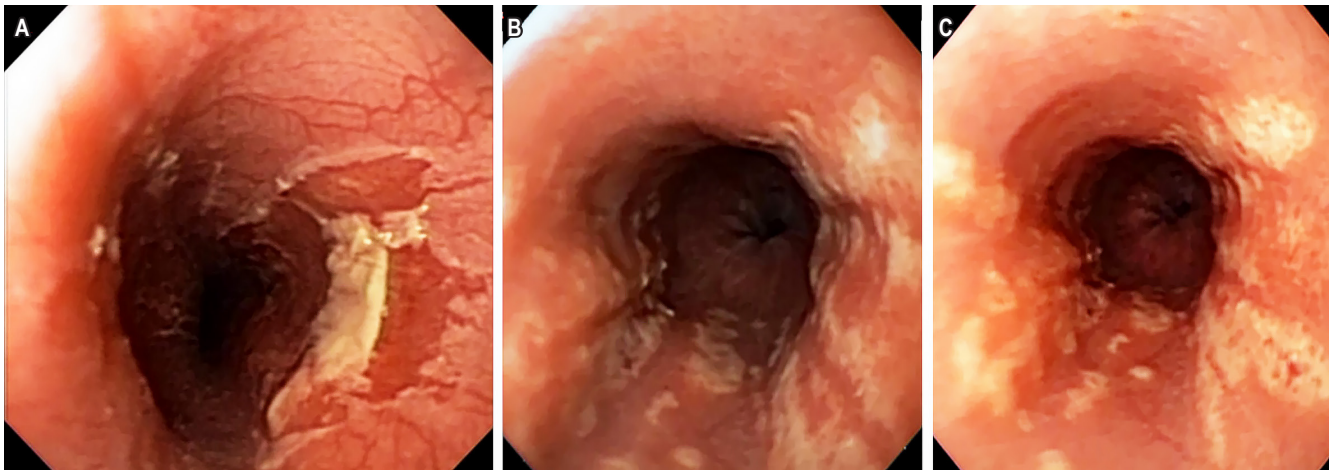


Figure 1. A. Esophageal ulcer. **B, C.** Whitish exudates. Author's file.

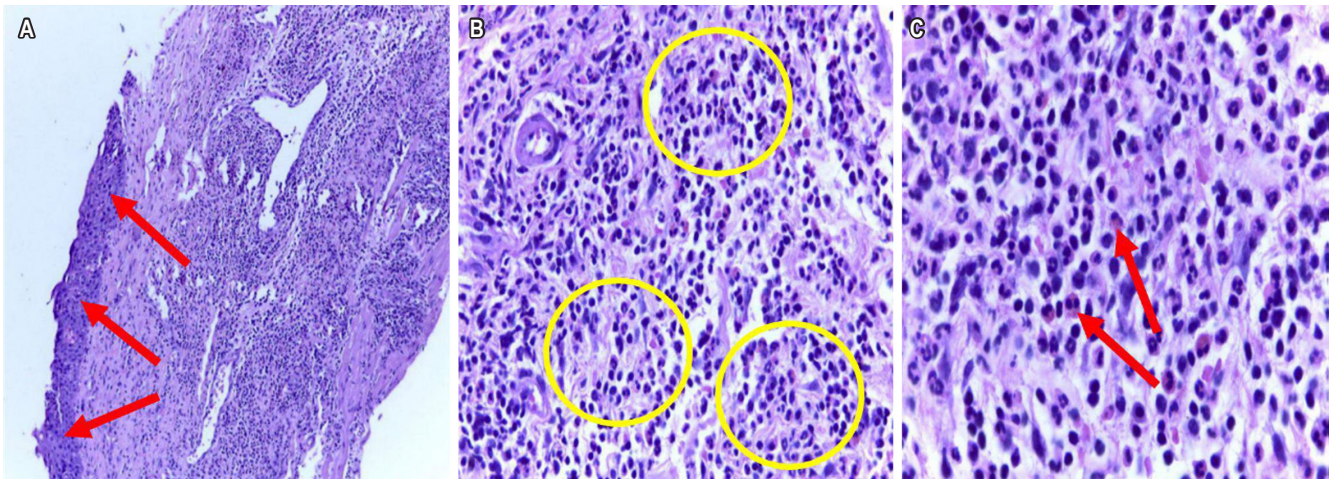


Figura 2. A. Red arrows: panoramic view showing involvement of the esophageal epithelium and mucosa by a severe eosinophilic infiltrate. **B.** Yellow circles: involvement of the stroma and submucosa by eosinophilic infiltration. **C.** Red arrows: close-up view of eosinophils with their granules. Author's file.

nical entities, it is essential to differentiate them accurately through a detailed epidemiologic history and a thorough clinical interview, as was done in the patient we present.

Currently, there are no studies that demonstrate the exact pathophysiology of this exaggerated eosinophilic infiltrate in certain patients with drug-induced esophagitis; however, this finding may be explained by a marked atopic component, mediated by hypersensitivity reactions to certain medications[14–18]. For this reason, it is important to evaluate specific factors in this group of patients, such as immunoallergic characteristics or inadequate fluid intake at the time of drug ingestion, as both may contribute to triggering new events in the medium and long term. Our patient had a history of allergic rhinitis, peripheral eosinophilia, and elevated serum IgE levels as markers of an underlying allergic component.

CONCLUSION

Drug-induced esophagitis is a clinical entity that should be considered among the etiologies capable of generating severe eosinophilic infiltration, which may even produce an endoscopic-histologic profile similar to that traditionally described in other conditions such as eosinophilic esophagitis. Likewise, it is necessary to evaluate the immunoallergic component and accurately identify the responsible drug in order to appropriately counsel this group of patients and prevent new events in the medium to long term.

Informed Consent

The authors declare that informed consent was obtained from the patient in this case.

Conflict of Interest

The authors declare no conflict of interest.

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Author Contributions

Study conception and design: all authors; data and information acquisition: all authors; article planning: all authors; critical revision of important intellectual content: all authors; final approval of the version to be published: all authors.

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