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CASE REPORT | STOMACH

Eosinophilic Gastroenteritis and Colitis After Intragastric Balloon Placement

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ABSTRACT

Eosinophilic gastrointestinal diseases are rare disorders characterized by infiltration of eosinophils in one or multiple segments of the gastrointestinal tract. Hypersensitivity to food or environmental allergens is believed to play an important role in the pathogenesis. In this case report, we describe a 61-year-old man who developed eosinophilic gastroenteritis and colitis with severe peripheral eosinophilia after intragastric balloon (IGB) placement for weight loss. His symptoms and peripheral eosinophilia improved rapidly after removal of the IGB without the need for immunomodulatory therapies or diet modifications. This case suggests a possible association between IGB and eosinophilic gastrointestinal diseases, which warrants clinicians' awareness and further studies.

INTRODUCTION

Eosinophilic gastrointestinal diseases (EGIDs) are a group of rare disorders characterized by infiltration of eosinophils in the gastrointestinal (GI) tract.[1] EGIDs can affect any part of the GI tract and include eosinophilic esophagitis (EoE), eosinophilic gastritis, eosinophilic gastroenteritis (EGE), and eosinophilic colitis (EC).[2] In recent years, EGIDs have been increasingly recognized.[2] Although the exact pathobiology of EGIDs has not been fully elucidated, it is believed that hypersensitivity to food or environmental allergens might play a pivotal role.[3]

Intragastric balloon (IGB) has been demonstrated as an effective and safe therapy for weight loss.[4] Interestingly, a previously published case reported the development of EoE after IGB placement, suggesting a possible association between IGB and EoE.[5] Otherwise, no report in the literature suggests a link between IGB and non-EoE EGIDs. In this article, we report a case of EGE and EC with peripheral eosinophilia after IGB placement, in whom rapid improvement and complete resolution of clinical symptoms and peripheral eosinophilia occurred after IGB removal without additional therapies. We hypothesize that IGB may be a trigger for EGIDs, which warrants clinicians' awareness.

CASE REPORT

A 61-year-old White man with a body mass index of 30 kg/m^2 and accompanying metabolic syndrome underwent endoscopic placement of an Orbera intragastric balloon (Apollo Endosurgery, Austin, TX) filled with 500 mL of saline for weight reduction. Forty days later, he was admitted with a 3-day history of watery and nonbloody diarrhea. He also reported nocturnal bowel movements, abdominal pain, nausea, nonbloody nonbilious emesis, and poor appetite. He had no fever, chills, or skin rashes and reported no sick contact, recent travel, or new pets. He was allergic to latex and penicillin; otherwise, he had no known allergies to any food or other medications. He denied any personal and family history of autoimmune diseases and inflammatory bowel disease. He was not on any special diet. There were no new medications started after IGB placement.

After admission, his symptoms persisted despite the use of loperamide and empiric ciprofloxacin and metronidazole. In addition, he developed severe peripheral eosinophilia over 15,000/mL (Figure 1). Infectious workup, including stool

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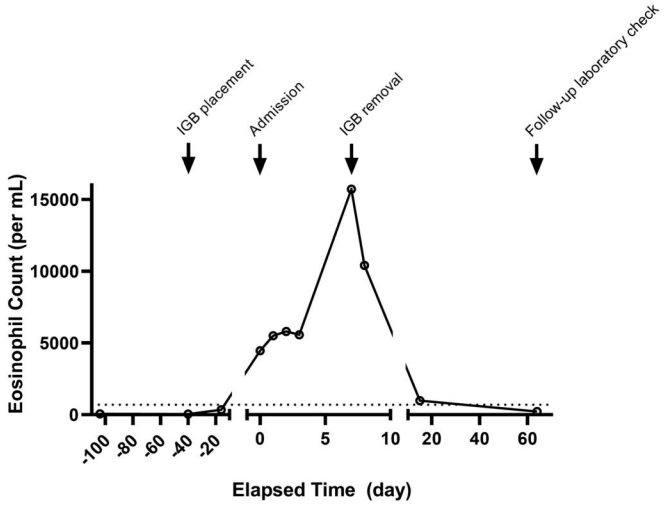

Figure 1. Blood eosinophil count over time. The dotted line represents the upper limit of normal eosinophil count (700/mL). The day of admission was set at day 0. IGB, intragastric balloon.

Clostridium difficile toxin, stool ova and parasite, stool culture, stool GI pathogen panel (BioFire Diagnostics, Salt Lake City, UT), Strongyloides IgG, and HIV, was negative.

Six days after admission, his IGB was removed because he had reached his goal weight. Then, he underwent esophagogastroduodenoscopy and flexible sigmoidoscopy with biopsy given persistent symptoms. Endoscopic examination was notable for subtle circumferential ridges in the esophagus, diffuse erythema in the stomach and duodenum, and congestion in the entire examined colon (Figure 2). Histologically, dense inflammatory infiltration of eosinophils and plasma cells in the lamina propria was observed in the stomach, duodenum, and colon, whereas biopsies of the distal and middle esophagus were unremarkable (Figure 3). In comparison, his previous esophagogastroduodenoscopy 4 years before showed mild reactive gastropathy in the antrum.

After removal of the IGB, his symptoms improved dramatically within 3 days and were completely resolved within 1 month. Peripheral eosinophilia was also resolved (Figure 1). He did not require corticosteroids, elimination diet, or biologic agents, which are commonly used for the treatment of EGIDs. There was no symptomatic recurrence at the 2-month follow-up.

DISCUSSION

EGIDs are rare disorders and can affect one or more segments of the GI tract. To date, EoE is the most studied subtype of EGID,[6,7] whereas the studies of non-EoE EGIDs are mostly limited to case reports and single-center cohorts.[3] In this article, we report a case with typical characteristics of EGE and EC including consistent GI symptoms and evidence of eosinophilic infiltration in the stomach, duodenum, and colon. Moreover, the patient in our case presented with severe peripheral eosinophilia, which is commonly seen in 80% of patients with EGE.[8] Other common causes of GI eosinophilic infiltration and peripheral eosinophilia, including drug and food allergies, infections, and IBD, were ruled out.

The pathogenesis of EGIDs is not fully understood. EGIDs are often associated with other allergic disorders.[9,10] It is believed that Th2-mediated hypersensitivity reaction, triggered by food or environmental allergens, plays an important role in the pathogenesis.[11] Although the trigger for the hypersensitivity reaction is not identifiable in all cases, elimination of certain food or other allergens may lead to symptomatic improvement and histological resolution.[12]

In our case, it is highly suspicious that the IGB triggered a hypersensitivity reaction that eventually led to an EGID. This is supported by the occurrence of symptoms and peripheral eosinophilia after IGB placement and rapid resolution after IGB removal without any immunomodulatory therapy or diet modification. In addition, this is supported by a previous case report suggesting an association between IGB and EoE.[5] It is less likely to be caused by other food or environmental allergens at home because the patient had persistent symptoms and eosinophilia after hospitalization. An unrecognized infection was also considered, given that fungal and bacterial colonization of IGB has been described in previous studies.[13] However, worsening eosinophilia, regardless of empiric antibiotics, and rapid improvement after IGB removal without additional antimicrobial therapies argue against this possibility.

The Orbera intragastric balloon has been demonstrated as an effective and well-tolerated therapy for weight control.[14]

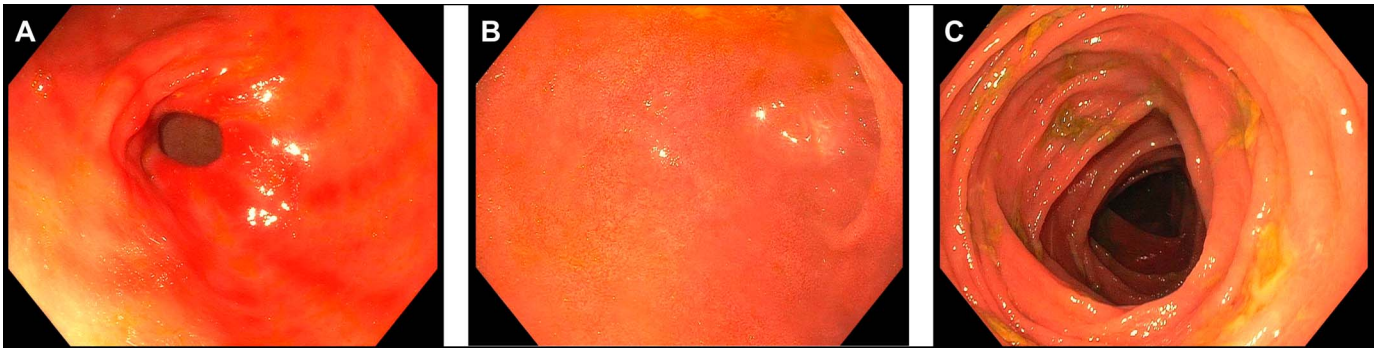

Figure 2. Endoscopic findings in the stomach (A, diffuse erythema), duodenum (B, diffuse erythema), and colon (C, congestion).

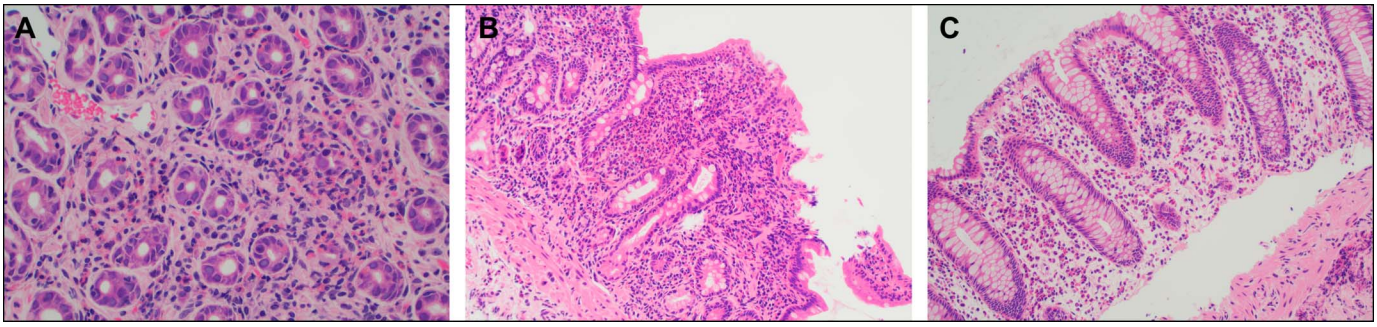

Figure 3. Representative histological findings in the stomach (A, 40× magnification), duodenum (B, 20× magnification), and colon (C, 20× magnification), notable for dense inflammatory infiltration of eosinophils and plasma cells in the lamina propria.

Accommodative symptoms are not uncommon within the initial weeks after placement, whereas major complications are rare. To the best of our knowledge, no report of allergic reaction to the Orbera IGB has been published. Of note, the Orbera IGB is made of nontoxic silicone, which has been reported as a potential cause of cerebrospinal fluid eosinophilia after intraventricular shunt placement.[15] In addition, there is a case of peripheral eosinophilia after rupture of silicone breast implants.[16] As such, further studies are merited to investigate potential allergic reactions to the Orbera IGB in selected cases, which could manifest as peripheral eosinophilia or EGIDs.

In summary, we describe a case of EGE and EC after IGB placement, presenting the first report in the literature that suggests a possible association between IGB and non-EoE EGIDs. We believe that this possible association warrants clinicians' awareness, and further studies are needed to confirm our observation and elucidate the underlying mechanism.

DISCLOSURES

Author contributions: J. Yin wrote the article and obtained the data. E. Wu and J. Jan obtained the data and searched the literature. T. Gray provided the pathology images. SL Shah and N. Enwerem revised the article for intellectual content. All authors approved the final article. N. Enwerem is the article guarantor.

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