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CASE REPORT

Eosinophilic gastritis - Case report

Bruno Carminatti da Silva[1], Jaqueline Cavalcanti de Albuquerque Ratier[2], Mônica Lisboa Chang Wayhs[2], Camila Marques de Valois Lanzarin[2], Renata Meirelles Gaspar Coelho Tomazzoni[2]

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Abstract

Eosinophilic gastritis is a rare condition characterized by a dense infiltration of eosinophils in the gastric mucosa. The suggested pathophysiological processes are multifactorial and involve mechanisms that lie between an immunoglobulin E-mediated response and a late T helper 2 response. The clinical presentation is variable and there is a strong association between allergic disorders and eosinophilic gastritis. In this report we describe the clinical and laboratory findings of eosinophilic gastritis in a 9-year-old boy treated with prednisolone and double-dose esomeprazole

[1] Universidade Federal de Santa Catarina, Curso de Medicina - Florianópolis - Santa Catarina - Brazil.
[2] Universidade Federal de Santa Catarina, Centro de ciências da saúde - Departamento de pediatria - Florianópolis - Santa Catarina - Brazil.

Correspondence to:
Bruno Carminatti da Silva.
Universidade Federal de Santa Catarina. R. Eng. Agronômico Andrei Cristian Ferreira, s/n - Trindade, Florianópolis - SC, 88040-900.
E-mail: carminatti94@gmail.com; ratierjaqueline@gmail.com

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INTRODUCTION

Eosinophilic gastritis (EoG) is a rare disease with limited evidence, mostly based on case reports or case series[1]. It is characterized by a dense infiltration of eosinophils in the gastric mucosa, in the absence of secondary causes for gastrointestinal eosinophilia[1]. EoG is typically part of a spectrum of eosinophilic gastrointestinal diseases (EGID), and it usually simultaneously affects other gastrointestinal segments in addition to the stomach, being the second most common presentation among eosinophilic diseases of the gastrointestinal tract[2,3]. The clinical manifestation is variable and nonspecific; and the diagnosis is made based on histopathological analysis of the gastric mucosa[1,2]. Considering the rarity of the presentation and the challenges for treatment, we present this case report. This study was approved by a human research ethics committee.

CASE REPORT A 9-year-old male patient, school-aged, asthmatic, undergoing treatment with budesonide and formoterol fumarate by inhalation. He attended a pediatric outpatient clinic on 10/7/2021, complaining of stabbing pain in the epigastric and thoracic regions associated with bad breath and nighttime awakening due to pain for 2 months, with worsening in the last month. He had been taking omeprazole 20 mg and ondansetron 4 mg for 15 days, with partial improvement of symptoms. He denied fever and changes in stool. His blood count, C-reactive protein and erythrocyte sedimentation rate were within normal limits on 09/06/2021. As he continued to have dyspeptic symptoms despite treatment, an upper digestive endoscopy was ordered, and omeprazole was discontinued. Upper digestive endoscopy (UDE) performed on 10/13/2021 showed endoscopic gastritis with moderate flat erosions of the antrum and negative H. pylori by the urease method, as described in Table 1.

On 10/15/2021, pantoprazole 40 mg once a day was prescribed. On 10/28/2021, the diagnosis of eosinophilic gastritis was defined based on the histopathological analysis of fragments of the gastric mucosa collected on 10/13/2021, in which eosinophilic invasion was identified with more than 30 eosinophils per high-power field, while the number of eosinophils in the esophagus and duodenum were normal, as described in Table 2. After 13 days of using pantoprazole, there was partial improvement of symptoms.

On 11/03/2021, there was a significant worsening of heartburn and epigastric pain. Pantoprazole was then discontinued and prednisolone 1.4 mg/kg started for 14 days. On 11/11/2022, the patient presented severe epigastric pain, with no improvement after ingesting 5 ml of aluminum hydroxide. A complementary investigation was then requested to exclude secondary causes of gastrointestinal eosinophilia, which resulted in a negative stool parasitological test, increased total immunoglobulin E (IgE), while specific IgE (casein, alpha-lactalbumin, beta-lactoglobulin, gluten, egg, egg white, peanut, oat, Brazil nut, shrimp, tuna, salmon, mussel) were normal, as described in Table 3. Transaminases, total proteins and fractions were within normal limits and there was no blood eosinophilia. Table 3 - Specific IgE levels on 11/08/2021

On 11/18/2021, he started esomeprazole 40 mg twice a day and gradually reduced the doses of corticosteroids. On 11/25/2021, he had a reduction in nocturnal awakenings, maintaining the other symptoms. He stopped taking prednisolone on 11/30/2021. On 12/16/2021, he completed 4 weeks of esomeprazole 40 mg twice a day, starting to take 40 mg once a day for another 4 weeks and then 20 mg for another 4 weeks, after which he stopped taking it. On 12/23/2021, he showed improvement in the epigastric pain and in his

Table 1. Results from the Upper digestive endoscopy

Date	
10/13/2021	ESOPHAGUSCOPY: Esophagus with preserved gauge and motility. The mucosa has a normal appearance throughout the organ. The esophagogastric epithelial transition is positioned at the level of diaphragmatic clamping. GASTROSCOPY: Gastric chamber with normal morphology. Antrum mucosa with edema, foci of enanthema and flat, shallow erosions with a red background. DUODENOSCOPY. Anatomical duodenal bulb with normal-looking mucosa. Second portion without alterations on examination with frontal viewing device. CONCLUSION: Endoscopic gastritis with moderate flat erosions of the antrum. The test for H. pylori by the urease method came back negative.
02/15/2022	ESOPHAGUSCOPY: Esophagus with preserved gauge and motility. The mucosa has a normal appearance throughout the organ. The esophageal-gastric epithelial transition is positioned at the level of diaphragmatic clamping. GASTROSCOPY Gastric chamber of normal morphology, containing clear residue in a small volume, easily aspirated. Good expansion of the gastric walls. The mucosal relief in the fundus, body and antrum does not present alterations. DUODENOSCOPY: Anatomical duodenal bulb, with normal-looking mucosa. Second portion without alterations on examination with a frontal viewing device. CONCLUSION: Endoscopically normal.

Table 2. Anatomopathological analysis of biopsies of the esophagus, stomach and duodenum

Date	
10/13/2021	1 - Small bowel biopsy specimen: finger-shaped mucosa of the small bowel (villus/crypt ratio of 3 to 5:1), with no particularities. The number of eosinophils and the usual lymphocyte/enterocyte ratio is 16% 2 - Gastric biopsy specimen: sample represented by pyloric gastric mucosa (antrum), with mild chronic inflammation, non-granulomatous, mild inflammatory activity and numerous eosinophils (more than 30 elements per high-power field in numerous histological fields). Helicobacter pylori was not detected in the sample. 3 - Esophageal biopsy specimen: detached fragments of squamous epithelium showing very few neutrophils in the intraepithelial region, with no other particularities, absence of eosinophils in the sample.
02/15/2022	1 - Small intestine biopsy specimen: duodenal finger-like mucosa (villus/crypt ratio of 3 to 5:1), with lymphoid follicles, without particularities. The number of eosinophils is normal, and the lymphocyte/enterocyte ratio is up to 16% (normal value). 2 - Gastric biopsy specimen: pyloric gastric mucosa (antrum), without particularities, fundic gastric mucosa (body), without particularities. Helicobacter pylori was not detected in the sample. 3 - Esophageal biopsy specimen: detached fragments of squamous epithelium showing rare neutrophils in the intraepithelial region, without other particularities, absence of eosinophils in the sample.

Table 3. Specific IgE doses on 08/11/2021.

IgE	Result	Reference value
f76 (alpha-lactalbumin)	0.17 < KUA/l	0.10 to 0.34 KUA/l (very low)
f77 (beta-lactoglobulin)	< 0.10 KUA/l	< 0.10 (undetectable)
f245 (full egg)	< 0.10 KUA/l	< 0.10 (undetectable)
f78 (casein)	< 0.10 KUA/l	< 0.10 (undetectable)
fp1 (peanut, oatmeal, almond, Brazil nut and coconut)	< 0.10 KUA/l	< 0.10 (undetectable)
f79 gluten	< 0.10 KUA/l	< 0.10 (undetectable)
fp2 (fish, shrimp, tuna, salmon, cod and blue mussel)	< 0.10 KUA/l	< 0.10 (undetectable)
f2 (cow milk)	< 0.10 KUA/l	< 0.10 (undetectable)
Total IgE	1,456.7 UI/ml	5 to 10 years (3.4 to 411.6 UI/ml)

chest pain, maintaining occasional episodes of heartburn in the morning. In January 2022, he was asymptomatic and using 20 mg of esomeprazole. In February 2022, he remained asymptomatic. On February 15, 2022, a new EDA was performed with biopsies of the esophagus, stomach, and duodenum, and in all fragments analyzed, the number of eosinophils was within normal limits, as described in Table 2.

DISCUSSION

Here, we describe an unusual presentation of EoG, with eosinophilia restricted to the stomach. We also demonstrate the importance of EoG as a differential diagnosis for diseases that present with dyspepsia and other nonspecific gastrointestinal symptoms, and the challenges involved in its treatment.

The clinical manifestation of EoG is variable and depends on the gastrointestinal layer involved (mucosa, muscularis, or serosal). The most frequent symptoms are abdominal pain, chest pain, nausea, vomiting, and diarrhea[2,4]. The prevalence of EoG in the pediatric population is estimated at 4.4/100,000, of which up to 59% have some allergic condition[5,6].

The suggested pathophysiological processes are multifactorial and involve responses mediated by IgE, cytokines, chemokines and T helper 2 (Th2) cells. The mechanisms underlying EGID are located between an IgE-mediated response and a late Th2-type response[7]. The result of this mixed IgE-mediated and Th2-dependent immune dysregulation is the recruitment, activation and prolonged survival of eosinophils with antigen-specific IgE production[7]. Upper gastrointestinal endoscopy with biopsies is the main examination for the diagnosis of EoG, although there is no consensus on the cutoff points for the number of gastric eosinophils, a mean density ≥ 127 eosinophils/mm² or ≥ 30 eosinophils per high-power field in at least 5 fields is suggested[7,8]. Additional investigation is usually necessary to exclude secondary causes of gastric eosinophilia, such as parasitic infections, drug allergies, and malignant neoplasms[4,9]. Food allergy tests, such as prick tests, patch tests, and serum specific IgE levels, are not very effective in identifying allergens associated with EGID. These tests are only useful in patients with symptoms of IgE-mediated food allergy[7].

Corticosteroids are the mainstay of treatment, although the type, duration, and dosage vary[7]. Prednisone is often the first choice, with doses of 20 to 40 mg/day, or in higher doses (0.5–1 mg/kg per day). The initial duration of treatment is 2 weeks, followed by a gradual reduction in dosage[1,7]. Another important medication is proton pump inhibitors (PPIs), whose proposed mechanism is that they act by decreasing the expression of eotaxin-3 (eosinophil chemoattractant) stimulated by interleukin-4 and interleukin-13. In addition, it reduces the transcription of messenger RNA and eotaxin-3 by decreasing the binding of signal transducer and activator of transcription 6 and RNA polymerase to the eotaxin-3 promoter.[10] In a prospective study of fifty-one children with eosinophilic esophagitis treated exclusively with esomeprazole at a dose of 1 mg/kg per dose, twice a day, there was an improvement in the histological pattern in 68.6% of the children. Clinical improvement was observed in 82% of the children and 47% reported complete improvement of symptoms[11]. However, despite the various treatments available, there are still no established guidelines for the clinical management of patients with EGID distal to the esophagus[7].

In this case, the complementary investigation did not find changes that would justify gastric eosinophilia. Specific IgE levels were not useful for identifying food allergies, although the amount of total IgE was increased. The corticosteroid doses used were higher than those usually used in cases of EoG, and there was still no satisfactory clinical improvement.

CONCLUSION

Although the use of esomeprazole in EoG in double and fractionated doses has not been well studied as a monotherapy, the results found for eosinophilic esophagitis suggest that there may be an important role for PPIs in EoG[11]. Finally, it is essential to maintain a high index of suspicion for EGID in atopic patients due to the high rate of association between allergic processes and EGID, thus enabling earlier diagnoses.

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