

## Duodenal-jejunal Stenosis as a Manifestation of Eosinophilic Gastroenteritis: A Case Report

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### ABSTRACT

**Introduction:** Eosinophilic gastroenteritis (EGE) is a rare disorder characterized by eosinophilic infiltration of the stomach and small intestine. Its clinical manifestations depend on the extent, location, and depth of the inflammatory infiltrate. The pathogenesis remains unclear, but it may be related to food allergy or other hypersensitivity reactions in some patients. Untreated cases can cause intestinal stricture or perforation, making timely diagnosis and intervention essential.

**Clinical case:** A 13-year-old male adolescent was hospitalized due to symptoms suggesting intestinal obstruction or alterations in intestinal motility. Medical imaging revealed significant gastric distension and delayed gastric emptying. An endoscopic study showed a “snake-skin” pattern, duodenitis due to food stasis, a 2 cm ulcer, and stenosis in the duodenum. The biopsy confirmed EGE with significant irregular eosinophilic infiltration of the lamina propria. A panendoscopy was performed after steroid treatment without being able to assess the site of stenosis and perform pneumatic dilation. After completing the immunomodulatory management, a contrast-enhanced abdominal tomography was performed showed a significant decrease in the intestinal lumen, therefore an exploratory laparotomy with a lateral to lateral jejunum-jejunal anastomosis was performed.

**Discussion:** EGE is a rare inflammatory disorder characterized by eosinophilic infiltration of the stomach and small intestine. Symptoms are based on the affected gastrointestinal tract segment. In this case, our patient presented intestinal obstruction symptoms with endoscopic biopsies and a histopathological study confirmed eosinophil infiltration. Treatment typically involves identifying potential triggers and corticosteroids to reduce inflammation and symptoms. However, in this case, a decrease in the intestinal lumen was observed, therefore an elective surgical resection was performed.

**Conclusion:** Eosinophilic gastroenteritis is a rare disorder that requires strong clinical suspicion to be diagnosed, which is why its knowledge is important. In this case, gastrointestinal symptoms, radiological and endoscopic images, in addition to the histopathological report, were key tools for integrating the diagnosis. Although management with steroids and immunomodulators was not successful, surgical management became an appropriate option as definitive treatment.

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### INTRODUCTION

Eosinophilic gastrointestinal diseases are a group of chronic inflammatory diseases of the gastrointestinal tract, characterized by symptoms that are related to the dysfunction of the affected gastrointestinal segments. Histologically, they are marked by an extensive eosinophilic infiltrate without any identifiable secondary cause. These diseases are classified according to the segment affected into eosinophilic esophagitis (EE), eosinophilic gastritis (EG), eosinophilic gastroenteritis (EGE), and eosinophilic colitis (EC). The most common and best described is EE <sup>(1)</sup>.

Eosinophilic gastroenteritis (EGE) is a rare inflammatory disorder, characterized by eosinophilic infiltration of the stomach and small intestine. <sup>(1)</sup> Although information on the prevalence of EGE is limited, it has been suggested that this disease may have an estimated prevalence between 28-30 per 100,000 inhabitants. <sup>(2)</sup>

This rare condition usually appears in children or young adults with symptoms that relate to the particular segment of the gastrointestinal tract affected with the disease. The stomach is the most common site of involvement, followed by the duodenum, jejunum, and ileum, which are affected approximately equally. Small intestine disease and gastric disease often occur together. In some patients, EGE presents with peripheral eosinophilia and frequently coincides with allergies or asthma. <sup>(3)</sup>

Clinical manifestations depend on the extent, location, and depth of the inflammatory infiltrate. The gastric antrum and small intestine are the most frequently affected areas. Three clinical forms are distinguished according to eosinophilic infiltration: mucosal form (the most common, presenting with abdominal pain, diarrhea, weight loss and malabsorption), muscular (thickening of the intestinal wall manifested by obstructive symptoms, characteristically at the pyloric region) and serous (transmural infiltrate with presence of eosinophilic ascites). <sup>(4)</sup> Approximately 50% of patients develop mucosal disease, followed by mural disease and, less frequently, serous disease, with a slight male predominance. <sup>(3)</sup>

The pathogenesis of eosinophilic gastroenteritis remains unclear. However, it may vary among the three types

described (mucosal, mural, and serous). Familial cases occur, but no specific genetic abnormalities or inheritance patterns have been identified. In some patients with mucosal disease, eosinophilic gastroenteritis appears to be related to food allergy or other hypersensitivity reactions. IgE is elevated in some patients. The degree of tissue damage and the severity of symptoms are related to the degree of eosinophilic infiltration and eosinophilic activation and degranulation. <sup>(3)</sup>

Because untreated GEE can cause intestinal stricture or, in severe cases, perforation, timely diagnosis and immediate intervention are essential <sup>(5)</sup>.

We present a clinical case of a 13-year-old adolescent who experienced hyporexia, vomiting, abdominal distension, and dyspepsia. Radiological and endoscopic studies were performed and confirmed the diagnosis of duodenojejunal stenosis. Biopsies were taken and histopathological reports showed eosinophilic enteritis, then it was decided to perform an elective intestinal resection plus anastomosis.

### CLINICAL CASE

A 13-year-old male adolescent, with a height of 143 cm and a weight of 63.6 kg, with a history of admission to the pediatric intensive care unit due to diabetic ketoacidosis and acute pancreatitis. During his stay, he underwent venodissection to place a right jugular catheter. He has no other surgical interventions and denies any allergies. History of Prader-Willi syndrome and multiple hospital admissions because of oral intolerance, with a previous diagnosis of gastroparesis.

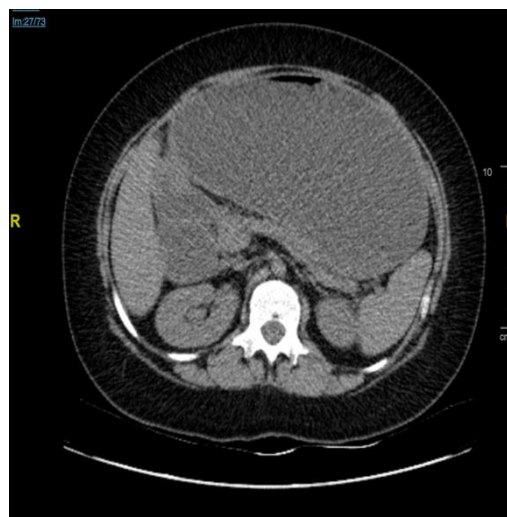
This time, he was readmitted to the hospital due to hyporexia, oral intolerance, episodes of vomiting with gastrobiliary content, abdominal distension, and dyspepsia, leading to suspicion of intestinal obstruction or alterations in intestinal motility.

On admission, a two-position abdominal X-ray was initially performed, showing significant gastric distension, therefore a CT scan was performed 24 hours after admission, which showed a stomach with an estimated volume of luminal content of 2 880 cc, associated with distension of the duodenal arch and proximal jejunum (**image 1, image 2**).

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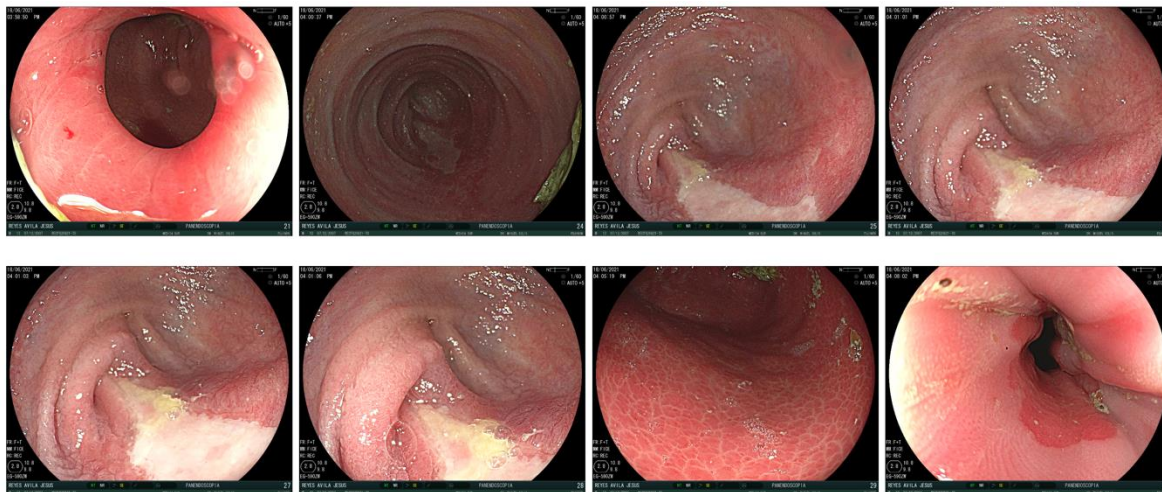
**Image 1.** The duodenal arch and proximal jejunum appear markedly distended at the expense of intraluminal liquid content material with similar characteristics to those of the stomach.



**Image 2.** Stomach at maximum filling, with predominantly liquid material in its lumen and with an estimated volume of 2 880 cc.

The symptoms of intestinal obstruction persisted, so a barium intestinal transit was performed 72 hours after admission, which reported delayed gastric emptying, dilation of the gastric chamber, duodenum and part of the jejunum, with diffuse thickening of the wall of the first and second portion of the duodenum. We decided to request an endoscopic study where an esophagus with long mucosal disruptions was

observed, the stomach with mucosa in a “snake-skin” pattern, antrum with reddened mucosa, with signs of duodenal-gastric reflux; the duodenum with duodenitis due to food stasis, the second portion with food residue, and in the third portion a 2 cm ulcer was visualized (**image 3**) with punctiform stenosis at this level, which conditioned a clinical picture of intestinal sub occlusion.

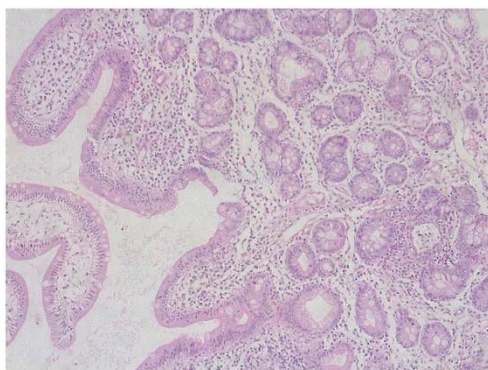


**Image 3.** The gastric mucosa is observed with a "snake-skin" pattern, gastric antrum with reddened mucosa. The pylorus is central, with the presence of signs of duodenal-gastric reflux. The duodenal bulb is observed with signs of duodenitis in relation to food stasis, the third duodenal portion with a long ulcer of approximately 2 cm.

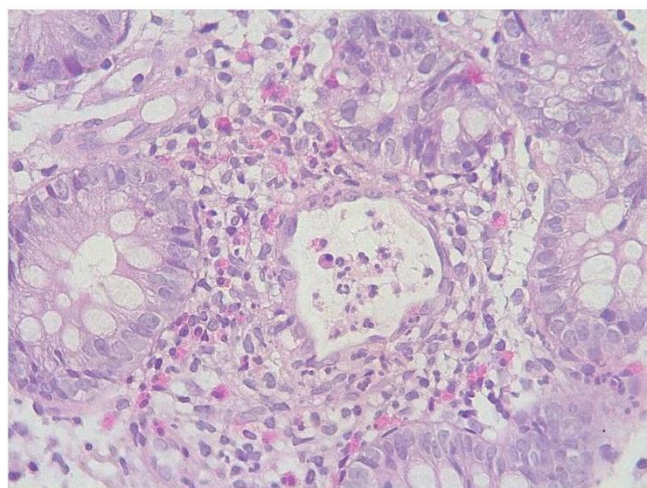
A biopsy on the area of stenosis and duodenal ulcer was performed; the histopathological report was obtained seven

days after the endoscopy, concluding: the presence of eosinophilic enteritis, with marked irregular eosinophilic infiltration of the lamina propria (>50 eosinophils / high power field in at least one focus).

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**Image 4.** Duodenal mucosa with expansion of the lamina propria due to inflammation, edema, and vascular congestion.

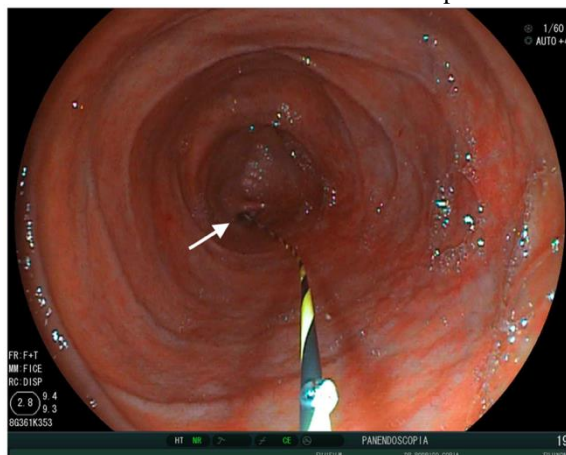


**Image 5.** Marked irregular eosinophilic infiltration of the lamina propria (>20 eosinophils/ HPF in at least one focus) with occasional prominent vessels.

The following laboratory studies were also performed: coproparasitoscopic series without evidence of parasites, immunoglobulin profile: IgG 1120 mg/dl, IgA 281 mg/dl, IgM 74.3 mg/dl, IgE 1520 IU/ml. Leukocyte count 4170/ $\mu$ l, Lymphocytes 38%, TCD3 lymphocytes 67%, TCD4+ CD3+ lymphocytes 47%, TCD8+ CD3+ lymphocytes 17%, CD4/CD8 index 2.85, Lymphocyte count 1590/ $\mu$ l, TCD3+ lymphocyte count 1340/ $\mu$ l, TCD4+ TCD3+ lymphocyte count 950/ $\mu$ l, TCD8+TCD3 lymphocyte count 333/ $\mu$ l. IgE 1,826 IU/ml. Food optigen profile (food-specific IgE): tuna and shrimp, class 1 (low level of IgE antibody). Anti-tissue

transglutaminase antibodies IgA 0.20 mg/dl, IgG 0.07 mg/dl. Anti-gliadin antibodies IgA 4.24 mg/dl, IgG 0.13 mg/dl.

Given the characteristics of the clinical picture, management with three pulses of intravenous methylprednisolone every 24 hours at 30 mg/kg/dose was initiated. After the steroid treatment (72 hours), a panendoscopy was performed to assess the site of stenosis and perform pneumatic dilation. However, during this procedure, a punctiform stenosis of 95% of the circumference was observed at 75 cm of the pyloric canal, without being able to reach it with the length of the phonendoscope (**Image 6**).



**Image 6.** Duodenum with cobblestone pattern, with a punctiform stenosis (arrow) involving 95% of the circumference 75 cm distal to the pyloric canal.

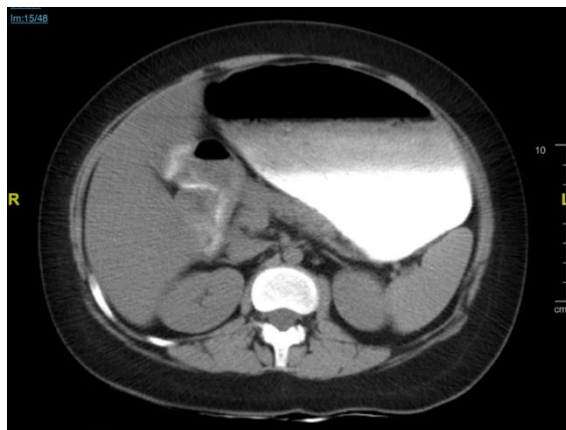
Through fluoroscopic control we observed that the stenosis was longer, with irregular edges, up to 13.5 cm in length, with filiform passage of contrast material, therefore it was not possible to perform hydropneumatic dilation. As the area of stenosis was larger than previously reported and in a more distal site, it was decided to continue outpatient management

with oral steroid (prednisone 50 mg PO every 24 hours) with a gradual decrease over 4 weeks, adding immunomodulator (azathioprine 50 mg PO every 24 hours for 6 weeks) and dietary allergen elimination therapy.

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After completing the immunomodulatory management, a contrast-enhanced abdominal tomography was performed (**image 7**) which reported a significant decrease in the intestinal lumen at the level of the third portion of the

duodenum, with subsequent dilation of the intestinal loops from the fourth duodenal portion to the proximal jejunum, with an abrupt change in caliber at that level (proximal jejunum), being located in the hypogastric area.



**Image 7.** Great distention of the gastric chamber is identified, with the passage of the filiform contrast through the first and second portions of the duodenum.

Therefore, it was decided to electively perform a resection of the intestinal stenosis site. In the exploratory laparotomy, the jejunum was found with a 5 cm long stricture, 40 cm from the angle of Treitz, with bands in the serous layer, and it was decided to resect 10 cm of the compromised intestine with subsequent lateral-to-lateral jejunum-jejunal anastomosis.

The resected tissue was sent for biopsy, whose histopathological report showed moderate chronic enteritis with the presence of up to 52 eosinophils per 40x field, with lymphoid follicular hyperplasia and villous shortening.

After the surgical event, the patient was kept under observation for five days and showed good recovery before being discharged. He continued outpatient immunomodulatory management with azathioprine 50 mg

PO every 24 hours until completing 6 additional weeks and was maintained on an allergen elimination diet.

Medical follow-up was provided in the pediatric outpatient clinic, observing a favorable evolution, and improvement concerning feeding tolerance, without recurrent vomiting episodes. The patient is currently in satisfactory general condition and has a good prognosis for life.

### DISCUSSION.

The manifestation of EGE has been described in the literature as symptoms of total or partial obstruction at various levels of the gastrointestinal tract that may require surgical treatment. Four criteria have been proposed for its diagnosis. <sup>(6)</sup>

Criteria for EGE diagnosis
Presence of gastrointestinal symptoms
Eosinophilic infiltration of the gastrointestinal tract
Exclusion of parasitic disease
Absence of other systemic involvement

Table 1. Criteria for EGE diagnosis.

We present the clinical case of a patient who had symptoms suggestive of intestinal obstruction characterized by dyspepsia, abdominal distension, and vomiting gastrobiliary content. A coproparasitoscopic study was performed where the presence of parasites was ruled out. It was demonstrated

with endoscopic biopsies and histopathological study, the infiltration of eosinophils at the intestinal level.

In eosinophilic diseases, imaging studies may demonstrate diffuse narrowing of the intestinal caliber and nonspecific data related to the level of obstruction. <sup>(7)</sup> In this case, the

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imaging studies (abdominal X-ray and CT scan) reported data of significant gastric distention. The barium intestinal transit reported delayed gastric emptying, dilation of the gastric chamber, duodenum, and jejunum partially, with diffuse thickening of the wall of the first and second duodenal portion.

Regarding endoscopic findings, they often appear to be non-specific and vary in appearance with nodules, polyps, erythematous or friable areas, and mucosal ulcers.<sup>(1)</sup> In this case, the esophagus was observed with long mucosal disruptions, the stomach with the mucosa in a “snake-skin” pattern, the gastric antrum with reddened mucosa, and signs of duodenal-gastric reflux. In addition, duodenitis was observed in relation to food stasis. Food remains were found in the second duodenal portion and an ulcer 2 cm in diameter was found in the third. Additionally, a punctate stenosis was observed.

In cases of suspected eosinophilic gastroenteritis, obtaining biopsies of the gastric antrum and duodenum is crucial for diagnosis and subsequent treatment.<sup>(8)</sup> EGE is a fundamentally histopathological diagnosis; however, as it is a rare disease, there is research that estimates the level of eosinophils in the gastrointestinal tract to consider the diagnosis, although without a consensus on formal cut-off points.<sup>(9)</sup> Excess eosinophils in the small intestine could be considered a multiple of the maximum count found in normal biopsies, such as 2x26/ HPF or 52/ HPF in the duodenal mucosa.<sup>(10)</sup> In our case, the biopsy of the area of stenosis and the duodenal ulcer reported eosinophilic enteritis, with marked irregular eosinophilic infiltration of the lamina propria (>50 eosinophils/ HPF in at least one focus).

The etiopathogenesis of EGE is not completely known, even though most authors recognize the intervention of an allergic etiology in most patients, which triggers an inflammatory reaction mediated by IgE and cytokines; therefore, reviews have been published suggesting various treatments, among which steroids as a cornerstone and the elimination diet stand out.<sup>(4,11,12)</sup>

The treatment of eosinophilic enteritis typically involves a multifaceted approach, including the identification of potential triggers and the use of corticosteroids to reduce inflammation.<sup>(13)</sup> Steroids, such as prednisone or methylprednisolone, are often employed as induction therapy to reduce eosinophilic inflammation and reduce symptoms in cases of eosinophilic enteritis<sup>(14)</sup>.

However, cases of eosinophilic duodenitis have been reported, where the severe abdominal pain and elevated eosinophil count, were resolved with a restrictive diet (removal of 1 to 6 of the following foods: dairy, wheat, egg, soy, peanuts and tree nuts, and fish and shellfish) and proton pump inhibitor (pantoprazole) without the use of corticosteroids. Indicating that dietary modification and acid suppression therapy alone can lead to symptom resolution in some cases of eosinophilic gastrointestinal disorders<sup>(15,16)</sup>.

This case highlighted the potential role of azathioprine use in the treatment of eosinophilic enteritis, particularly in tapering and discontinuing prednisone without disease relapse; to achieve long-term stabilization of the patient's condition, in case of adequate response to steroid management<sup>(17)</sup>.

However, our patient did not respond satisfactorily to the elimination diet, therapy with steroids (methylprednisolone, prednisone) and immunomodulators (azathioprine), persisting with gastro-biliary vomiting and being documented by a new contrast-enhanced abdominal tomography a significant decrease in the intestinal lumen to level of the third duodenal portion. Therefore, it was decided to electively perform surgical resection of the affected intestinal segment plus anastomosis at the same surgical time; the surgery was performed without complications.

Histopathology of the resected intestinal section reported: moderate chronic enteritis with the presence of up to 52 eosinophils per 40x field, with lymphoid follicular hyperplasia and shortening of the villi.

The patient had an adequate evolution in the postoperative period, and upon discharge from the hospital continued with follow-up by the pediatric service, reporting satisfactory evolution, without recurrence of gastrointestinal symptoms and with adequate tolerance to the oral route.

Nowadays recent advancements have led to the approval of *dupilumab* as the first treatment for eosinophilic esophagitis by the Food and Drug Administration, signifying progress in therapeutic options<sup>(18)</sup>.

The natural history of eosinophilic gastroenteritis has not been well documented; however, these diseases fluctuate chronically. If the patient has a disease induced by food antigens, abnormal levels of circulating IgE and eosinophils often serve as markers of tissue damage. Because these diseases often represent a manifestation of another primary process, systematic monitoring of the cardiovascular and respiratory systems for eosinophilic damage is advisable. If the disease occurs in infancy and sensitization to specific foods is detected, there is a high probability that it will remit at the end of childhood<sup>(19)</sup>.

### CONCLUSION

Eosinophilic gastroenteritis is a rare disorder that requires strong clinical suspicion to be diagnosed, which is why its knowledge is important. In this case, gastrointestinal symptoms, radiological and endoscopic images, in addition to the histopathological report, were key tools for integrating the diagnosis. Although management with steroids and immunomodulators was not successful, surgical management became an appropriate option as definitive treatment.

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### CONFLICT OF INTERESTS

The authors declare no conflict of interest.

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