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Gastrointestinal Eosinophilic Disorder in a Patient with Acute Pancreatitis. Case Report

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ABSTRACT

SUMMARY

Eosinophilic colitis is a rare disease. It is part of the eosinophilic gastroenteritis, characterized by eosinophilic infiltration of the tissues that affects any segment of the digestive tract, but more frequently the stomach and small intestine and rarely the colon in isolation. The clinical case of a 29year-old patient is described, who initially presented data of acute pancreatitis and later acute diarrhea with sluggishness and scant mucus. The analysis showed a complete blood count with eosinophilia 9.9% Immunoglobulin E with a positive result + 2500 IU/mL (<100IU/mL). Endoscopy and colonoscopies show diffuse inflammatory changes, confirming with the histopathological study infiltration of eosinophils throughout the gastrointestinal tract, conservative treatment was started with a good response. Eosinophilic gastroenteritis has a benign course, with significant infiltration of eosinophils in the wall of the digestive tract, which rarely involves only the colon. Its cause and pathogenic mechanisms are unknown; It classically presents in patients between the third and fifth decades of life, although it can affect any age group. The diagnostic criteria are: 1) gastrointestinal symptoms; 2) eosinophilic infiltration of one or more areas of the digestive tract demonstrated by biopsy; 3) absence of eosinophilic infiltration in organs outside the digestive tract; and 4) absence of parasitic infection. Eosinophilic gastroenteritis are pathologies that require a high index of suspicion for their diagnosis. It was observed that there was no need to use systemic corticosteroids, only suspension of previous treatments and free antigenic dietary adjustment.

KEYWORDS: Eosinophilic enteropathy, Pancreatitis, Eosinophilia

ARTICLE DETAILS

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The term gastrointestinal eosinophilic disorders (DEGI), described for the first time in 1980, refers to an accumulation of an abnormal number of eosinophils in a region of the gastrointestinal tract, including eosinophilic esophagitis and eosinophilic gastroenteritis, the latter being able to subclassify into gastritis, duodenitis, ileitis, or colitis, each with distinctive epidemiological, clinical, endoscopic, and histological characteristics; on some occasions, a more extensive inflammatory involvement may present that encompasses multiple sites of the gastrointestinal tract[1,2,3]. DEGI are characterized by a dense infiltration of eosinophils in the gastrointestinal tissues, giving morphological and functional alterations of the gastrointestinal tract. Considered to be the cause of allergic reactions to various allergenic mechanisms such as food, environmental antigens, dividing into eosinophilic esophagitis (EoE), and eosinophilic gastroenteritis (EGE)[4].

Analyzes have shown increased TH2 levels in the gastrointestinal mucosa. Based on the pathogenesis, EGE is believed to be a chronic allergic reaction to TH2-type eosinophils, caused primarily by food allergens[5,6].

There is no gender difference in this entity, as the gender ratio has been reported to be almost 1:1. Compared to the esophageal epithelium, the intestine, especially the small intestine, has higher permeability, with greater penetration of food antigens[7].

Clinically it is defined by the presence of abdominal symptoms, which include pain, diarrhea, as well as a dense infiltration of eosinophils, in the gastrointestinal wall. Unlike the esophagus, due to the physiological characteristics of the epithelium of the stomach and intestine, we can find eosinophilic infiltration. Without a pathological condition. The density of subepithelial tissues is not uniform, differing between the different segments of the gastrointestinal tract[8,9,10,11].

CLINICAL CASE

A 29-year-old woman with a history of failed low-intensity autolysis attempt at age 17 due to ingestion of an unspecified chemical with antidepressant treatment for 6 months and currently suspended, denies atopy, denies chronic diseases. It began with intense colicky epigastric pain, transfictive, progressive and associated with food intake accompanied by vomiting of 7 days of evolution, an initial diagnostic protocol was carried out where biochemically it presented an elevation of lipase 111 U/L (13-60u/L)) without evidence of metabolic cause and with negative antibodies, elevation of Leukocytes 12.78 thousand with eosinophilia in the differential count of 9.9%; Mild acute pancreatitis was staged by Atlanta computed tomography of the abdomen. After 3 days, it begins with diarrheal stools with lentieria and little mucus, frequently 6 to 10 times a day and biochemically showing a progressive increase in leukocytes and eosinophils; A diagnostic approach to acute diarrheal syndrome was carried out with a coproparacytoscopic test and fecal culture, with a negative report. Given the persistence of symptoms, an immunoglobulin E test was performed with a positive result of +2500 IU/mL. Endoscopy shows body mucosa with erythema and increased vascularity, colonoscopy with evidence of non-specific colitis (Fig.1A) and biopsy results that show eosinophilic infiltrates throughout the gastrointestinal tract(Fig.1B); 11 days after the onset of the acute diarrheal syndrome, leukocytes decrease, and bowel movements subside. The patient is discharged home due to improvement with an appointment at the outpatient clinic to assess the initiation of steroid therapy in the event of persistent symptoms.

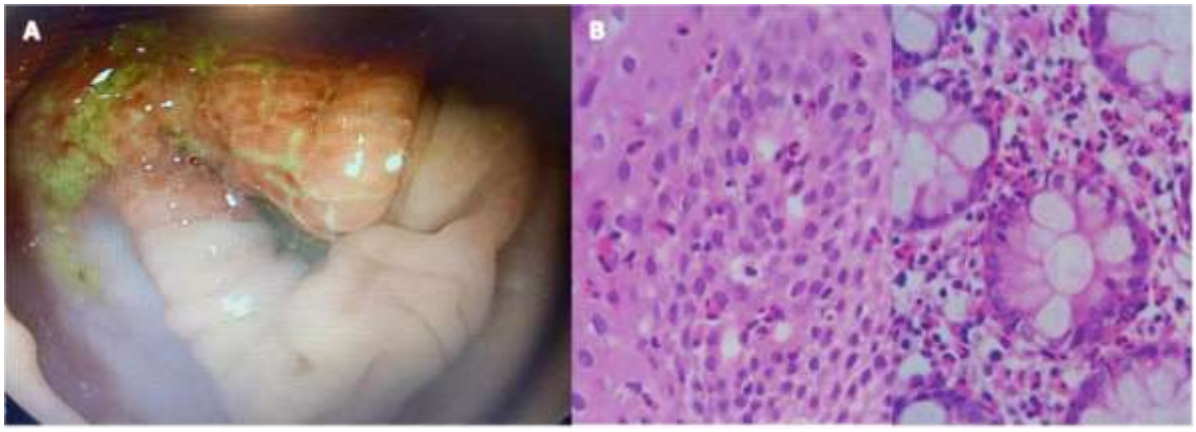

Figure 1.-A) Colonic Mucosa: congestive colonic mucosa is observed, dermatosis and presents segmental subepithelial hemorrhage. B) Stratified squamous epithelium is observed, with congestive vascular papillae and a transepithelial infiltrate of eosinophils, approximately 25 to 30 per high-power field. The gastric portion at the level of the cardia shows glands with a severe infiltration of leukocytes, among which abundant eosinophils are observed, more than 50 per high power field

DISCUSSION

DEGI are a diagnosis of exclusion, and it is necessary to rule out other pathologies that cause tissue eosinophilia such as bacterial and parasitic infections, inflammatory bowel disease, hypereosinophilic syndrome, connective tissue diseases, and myeloproliferative neoplasm[12]; In Western countries, a prevalence of 50 per 100,000 has been reported for eosinophilic esophagitis, and 2 to 8 per 100,000 for eosinophilic gastroenteritis, identifying an increase in the last 2 to 3 decades, as a result of greater recognition. of the disease as well as a true increase in its prevalence[13].

In 1985, oyaizu et al. first presented evidence for mast cell and IgE-mediated eosinophilic chemotaxis, although the pathophysiology is not exactly known, the predominant mechanism is considered to be an inappropriate and exaggerated Th2 cell immune response to exogenous antigens. , mainly food and environmental antigens; Risk factors such as the use of proton pump inhibitors and antibiotics have been identified, which have been associated with changes in the intestinal microbiota and alterations in the digestion of food antigens[14,15,16]. In addition, genes associated with increased risk have been identified, such as thymic stromal lymphopoietin and calpain 14, through the alteration of the Th2 immune response and increased epithelial permeability[17,18]. The diagnosis is made with the combination of clinical, biochemical, endoscopic, and

histopathological findings. In systematic reviews it has been reported that up to half of patients with gastrointestinal eosinophilic disorder have a history of atopic diseases, mainly bronchial asthma, allergic rhinitis, atopic dermatitis and immunoglobulin-mediated food allergy, commonly presenting with non-specific gastrointestinal symptoms, mainly abdominal pain. , nausea, vomiting, weight loss and diarrhea, although hematochezia or hidden blood in feces can occur in up to 50%, these manifestations will depend on the location and degree of eosinophilic infiltration [19]. Peripheral eosinophilia is found in approximately one third of patients with eosinophilic esophagitis, however, in eosinophilic gastroenteritis it can occur in up to 80%, with greater severity, additionally, IgE elevation is identified in up to 70%, being found in some cases IgE specific for antigen[20, 21,22].

Regarding endoscopic findings, these vary according to the specific eosinophilic gastrointestinal disease. In eosinophilic esophagitis, longitudinal grooves in the lower portion of the esophagus are a common finding, identified in approximately 90% of cases, together with concentric rings. and the whitish plaques constitute the three main findings; To the on the contrary, in eosinophilic gastroenteritis the endoscopic study is normal in 60-70%, in the rest non-specific findings such as edema and mucosal erythema, ulcers, erosions and nodularity can be identified, which can also be found in other gastrointestinal diseases[15,23,24]. Except for the esophagus, the presence of eosinophils in the gastrointestinal tract is a common finding, with variation in their number according to the site, in some studies identifying an increasing gradient of eosinophils that goes from proximal to distal, without having a determination of the value. normal For the histopathological diagnosis of eosinophilic esophagitis, an infiltration of eosinophils > 15 per high-powered field (x400) is required in the esophageal epithelial layer, other findings described are mast cell infiltration, basal hyperplasia, and dilation of the intercellular space[25,26]. On the contrary, for eosinophilic gastroenteritis there is no consensus on the optimal cut-off point regarding eosinophil infiltration according to the segment evaluated; however, based on the literature and clinical experience, a cut-off value of 25-30 for eosinophils has been suggested. by high power field for stomach and small intestine and more than 65 eosinophils for the colon, among other findings are intraepithelial eosinophils, eosinophils in Peyer's patches, granule protein deposition, villous atrophy, crypt hyperplasia and infiltration of mast cells[27].

Treatment includes strategies such as restriction of certain foods, use of corticosteroids. mast cell stabilizers, leukotriene receptor antagonists, and monoclonal antibodies, however, there are no randomized controlled trials or clinical guidelines to justify their use[28,29]. The recommendation of a diet with food restriction is based on retrospective studies and case reports, this can be guided through skin tests and RAST test, however, this measure usually has a variable effect, in the same way, in a clinical trial prospective study that included 9 patients with eosinophilic gastroenteritis identified improvement in symptoms and histological findings with the elimination therapy of 6 foods (milk, wheat, eggs, legumes, nuts, fish/shellfish) and elemental diet[30]. Corticosteroids are considered the basic pharmacological therapy, being recommended in those who do not respond to dietary therapy, even though there is no scheme evaluated by randomized controlled trials, a scheme based on prednisone doses of 20-40 mg/day is usually recommended. from 2 to 6 weeks, with subsequent reduction in a range of weeks to months, however, the requirement for long-term therapy has been reported, as in a retrospective study at the Mayo Clinic in which it was reported that in the group of patients with peripheral eosinophilia required maintenance therapy with an average duration of 52 weeks[20,31,32,33,34]. Among the latest therapeutic developments are monoclonal antibodies such as the anti-Il-5 agents mepolizuman and reslizumab, which, although initially approved for the treatment of eosinophilic asthma, have subsequently been identified as having a beneficial effect in patients with eosinophilic esophagitis through multiple trials. phase 2 randomized clinical trials; Similarly, anti Il13 agents such as RPC4046 have been used, in which through randomized clinical trials a significant histological and endoscopic improvement was identified[35,36,37].

CONCLUSION

DEGI are a rare class of chronic allergic diseases, often due to allergens food. The vital point of the diagnosis is due to the effectiveness of the medical treatment in the resolution of the symptoms, in addition to the reduction of sequelae and the impact on quality of life. The diagnosis must be made by a doctor who is an expert in the subject, since the low incidence of the disease, as well as the few cases reported to date, requires expert clinical judgment and hence the first-line treatment to be used.

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