Health Problems of Civilization
eISSN: 2354-0265, ISSN: 2353-6942

REVIEW PAPER

EOSINOPHILIC GASTROENTERITIS IN CHILDREN: A SYSTEMATIC REVIEW OF CASE REPORTS

Julia Plewniok[1(A,B,C,D,E,F)], **Maria Partyka**[1(B,C,D,E,F)], **Maksymilian Janeczek**[1(A,B,C,D,E,F)], **Wiktoria Maria Szymańska**[1(E,F)], **Karolina Kinga Kantor**[1(E,F)], **Julia Wójcik**[1(B,C)], **Kacper Wojciech Pamuła**[1(B,C)], **Karolina Jaglarz**[2(E,F)], **Maciej Kuca**[2(C,D)], **Marcin Cholewa**[3(C,D)]

[1]Faculty of Medical Sciences in Katowice, Medical University of Silesia in Katowice, Poland

[2]Department of Anesthesiology and Intensive Care, Katowice Oncology Center, Katowice, Poland

[3]Department of General and Oncological Surgery, St. Luke's Provincial Hospital, Independent Public Healthcare Institution, Tarnów, Poland

Plewniok J, Partyka M, Janeczek M, Szymańska WM, Kantor KK, Wójcik J, et al. Eosinophilic gastroenteritis in children: a systematic review of case reports. Health Prob Civil. https://doi.org/10.5114/hpc.2025.153689

Tables: 2

Figures: 1

References: 51

Submitted: 2025 Feb 2

Accepted: 2025 Aug 20

Address for correspondence: Julia Plewniok, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Poniatowskiego 15, 40-055 Katowice, Poland, e-mail: julkaplew19@gmail.com, phone: +48 509 339 331

ORCID: Julia Plewniok https://orcid.org/0009-0008-9728-7795, Maria Partyka https://orcid.org/0009-0003-0061-3122, Maksymilian Janeczek https://orcid.org/0009-0003-9854-4742, Wiktoria Maria

Szymańska https://orcid.org/0009-0005-4263-7565, Karolina Kinga Kantor https://orcid.org/0009-0005-0484-2883, Julia Wójcik https://orcid.org/0009-0007-6178-1532, Kacper Wojciech Pamuła https://orcid.org/0009-0003-5236-5298, Karolina Jaglarz https://orcid.org/0009-0009-7316-4042, Maciej Kuca https://orcid.org/0000-0002-6749-7360, Marcin Cholewa https://orcid.org/0009-0002-8520-8187

Summary

Among gastrointestinal disorders, to this day, many diseases are insufficiently studied. One of these under-researched conditions is eosinophilic gastroenteritis. The underlying cause of this rare disease is an inflammatory process affecting a large section of the alimentary tract – mainly the stomach and small intestine. This condition presents with non-specific clinical symptoms. Due to similarities between the initial symptoms and those of other prevalent gastrointestinal conditions, patients often initially receive an inaccurate diagnosis, leading to ineffective treatment and a significant challenge for physicians. Unfortunately, even if the diagnosis is made, the treatment process remains challenging. Due to the lack of precise recommendations regarding the medical approach, some individuals need to modify their therapy multiple times before the symptoms resolve. This systematic review is based on the

PubMed database and concentrates on articles published between 2014 and 2024, focusing on case reports of patients under 18 years of age. The main aim was to expand the knowledge regarding the etiology, symptoms, diagnostic methods, and treatment approaches for eosinophilic gastroenteritis .

Keywords: eosinophilic enteropathy, eosinophilia, pediatric, rare diseases, gastrointestinal disorder

Introduction

Eosinophilic gastroenteritis (EGE) is a rare inflammatory condition which affects a large section of the gastrointestinal tract – mainly the stomach and small intestine. This disorder affects all demographics groups, regardless of race, gender or age; however, a higher frequency is observed in patients between the ages of 30 and 50 [1]. That being said, children are also regularly affected.

Definition

EGE is a comprehensive term comprising a group of chronic, immune-mediated diseases characterized by eosinophil-rich inflammation affecting one or more segments of the gastrointestinal tract [2]. Using Klein's classification, which has been widely employed to define groups of patients with EGE, the disease can be divided into three subtypes (mucosal, muscular, and serosal) [3]. Clinical manifestations of EGE vary depending on several factors, including which intestinal segments and layers are affected by eosinophils.

History

The history of EGE dates back to the mid-20th century. In 1937, Swedish surgeon Albert Kaijser published an original description of the ailment in the context of an allergic gastrointestinal reaction [4]. Since then, knowledge about EGE has come a long way. Around 1970, Neil C. Klein published a case report that initiated the histological classification of patients based on the depth of eosinophil infiltration [5]. His work remains a cornerstone of the diagnosis to this day.

Medical information

Clinical presentation of EGE

EGE symptoms and signs depend on the layer depth and location of the affected tissue. The organ most commonly affected by EGE is the stomach. Other parts of the digestive system that are frequently affected by this condition include the small intestine and colon [6]. Some of these symptoms are nonspecific, which makes diagnosis more challenging. A large number of patients may experience nonspecific symptoms such as nausea, vomiting, abdominal pain, diarrhea, or hemorrhage [7]. Children may also often develop symptoms not directly associated with the digestive tract, such as delayed sexual development, amenorrhea, or growth disturbances [6]. The symptoms also depend on the age at which the disease manifests. It is observed that the most common indication in infants and toddlers is feeding difficulties, while in children it is vomiting and abdominal pain. At the same time, most adolescents complain of dysphagia and food impaction [8].

On the other hand, the mucosal form of the disease leads to significant disruptions in hematological parameters, as well as in the essential nutrients necessary for the proper functioning of the human body. These manifestations may include malabsorption (e.g. growth retardation, anemia, constipation, protein loss) or bleeding [6].

When the inflammation process in EGE affects the muscular layer, it may clinically present as a mass through thickening of the bowel wall, the appearance of a mass in its lumen, or obstruction. In rare cases, it may also affect other parts of the digestive system (appendix) or other organs like the pancreas, bile ducts, or spleen.

The last layer affected by the disease process is the serosal layer. Among all three, this is the most challenging to diagnose due to its atypical nature [9]. Moreover, in some cases, this form does not show eosinophil-rich inflammation on endoscopic examination, which further complicates establishing a correct diagnosis [10].

Diagnostic algorithm

It is widely known that the symptoms of EGE are non-specific, and as a result, it often takes many years from symptom onset to correct diagnosis. Unfortunately, to date, a detailed clinical management algorithm for enabling the accurate diagnosis of patients in the shortest possible time has not been developed.

Currently, the only diagnostic workflow is that established by Talley et al. in 1990, which includes three fundamental elements:

– non-specific gastrointestinal symptoms,

– eosinophilic infiltration of one or more areas of the GI tract,

– exclusion of other causes of tissue eosinophilia [6,7].

With advances in knowledge about EGE, certain factors that facilitate the diagnosis of the disease have been identified. These include a thorough medical history, laboratory and allergy tests, imaging studies (ultrasound, CT, endoscopy, colonoscopy), and histopathological examination.

Medical history

Factors that should guide a physician toward correct diagnosis include a properly conducted medical history. Many patients report the presence of allergic conditions, such as asthma, allergic rhinitis, allergic skin conditions, or IgE-mediated food allergies [11]. Additionally, it is important to determine whether the patient's family has a history of allergies, autoimmune disorders, or conditions affecting the digestive system.

Laboratory tests

In the case of suspected EGE, laboratory testing is indispensable. These tests help determine the levels of IgE antibodies, which are often elevated in patients with EGE. Additionally, a complete blood count (CBC) can provide valuable information regarding white blood cells (e.g. peripheral eosinophil count) and red blood cells (to assess potential bleeding). Other tests that should be conducted include: blood biochemistry tests, serum albumin level, evaluation of IgE antibodies directed against specific allergens, and stool tests (general stool analysis, stool culture, and parasite screening) [12].

Medical imaging tests

Basic imaging tests, such as ultrasound (US) or computed tomography (CT), can be performed in most hospitals and do not require significant patient preparation. In some cases, these studies may be helpful by revealing a thickening of the gastrointestinal wall, a mass narrowing the lumen, or enlarged lymph nodes. Additionally, they can help assess which section of the gastrointestinal tract is involved in the disease process [13]. However, the main role in differential diagnosis is played by histopathological examination, for which biopsies are taken during gastroscopy and/or colonoscopy – procedures that are critical for accurate diagnosis.

Histologic evidence

As a crucial diagnostic element of EGE, histopathological examination provides essential information. The best source of tissue samples, containing all layers, is obtained through surgical intervention; however, this option is not most commonly chosen. Samples can also be obtained during endoscopy and/or colonoscopy procedures. Biopsies should be provided from both normal and abnormal mucosa, as macroscopically normal tissue may appear diagnostic under microscope [14]. Currently, the most commonly used staining method is H&E (hematoxylin and eosin), which only detects intact eosinophils and cannot identify eosinophil degranulation. Therefore, novel methods are highly anticipated, e.g. the Hansan et al. method, which proposes semi-automated detection that simultaneously reduces the drawbacks of manual counting [11]. The eosinophil count, which is determined during histopathology, depends on various factors, including the intestinal segment from which the

biopsy was taken. Despite the lack of standardized criteria, most scientific studies set cutoff values at >10 eosinophils/HPF for children and >20 eosinophils/HPF for adults [14,15].

Differential diagnosis

The differential diagnosis for EGE includes several disease entities that manifest similar clinical symptoms (gastrointestinal signs and eosinophilia), namely: hypereosinophilic syndrome (HES), bacterial infections (e.g. *H. pylori*), viral or parasitic infections (e.g. *E. vermicularis, A. caninum, Ascaris, T. canis, S. stercoralis*), autoimmune diseases (e.g. eosinophilic granulomatosis with polyangiitis), inflammatory bowel disease (e.g. Crohn's disease and ulcerative colitis), celiac disease, hematological states, side effects of some medications (e.g. carbamazepine, rifampicin, azathioprine, or interferon), or malignancy [11,16].

Treatment

In a manner akin to the diagnostic process, no precise recommendations regarding treatment of EGE have been developed to date. Nevertheless, patients have access to a wide range of treatment options, from simple approaches like dietary modifications to more advanced methods including biological therapy.

Dietary therapy

As potentially the simplest treatment method, dietary modification is often chosen as the first step in managing EGE. Most patients with diagnosed food allergies can secure remission

with primarily elimination of the foods to which intolerance has been identified [17]. However, when a patient is allergic to a wide range of food items, an empiric elimination diet or elemental diet may be the optimal approach. The aforementioned diets, as well as the six-food elimination diet (6-FED) or 7-FED (red meat is additionally excluded), can be applied to patients in whom no allergens negatively affecting their health have been identified [14].

Although dietary therapy, based on published data, reduces or eliminates the presence of symptoms, it is primarily a treatment option for individuals whose mucosal layer is affected. Unfortunately, when it comes to muscular and serosal type of EGE, this therapy method may not demonstrate the most optimal outcomes [18]. Additionally, it is worth noting that as the duration of its use increases, the effects may become less pronounced. Combined with the significant sacrifices such a diet entails, this may lead to discontinuation of the regimen and a preference for pharmacological treatments.

First-line therapy for EGE

Corticosteroids are one of the primary foundations of EGE therapy [19]. Their mechanism of action is based on the suppression of transcription of eosinophil growth factors such as IL-3, IL-5, and GM-CSF, which leads to a reduction in the number of circulating eosinophils [16]. This also decreases the production of toxic mediators and other pro-inflammatory factors, directly impacting the reduction of disease activity. Despite the fact that most scientific sources identify corticosteroids as the most effective treatment for EGE, there is still a lack of information regarding the most effective doses, duration of therapy, or the most efficient corticosteroid analog. This highlights the need to consider the potential adverse effects of prolonged use and raises the question of whether the treatment benefits will outweigh the side effects [14].

Other pharmacologic agents employed as a first-line therapy are proton-pump inhibitors. They attenuate gastric acid secretion, which in turn helps mitigate clinical symptoms and reduce the severity of the pathological process. At the same time, they are hypothesized to be involved in the blockade of IL-4 and IL-13 activity. The combined effect of these two mechanisms leads to a reduction in eosinophil viability [14].

Second-line therapies for eosinophilic gastroenteritis

When corticosteroids no longer yield therapeutic effects, their adverse effects become intolerable for the patient, or the disease persists, second-line therapies may be considered. These include pharmacological agents such as leukotriene receptor antagonists, antihistamine drugs, or mast cell stabilizers.

Leukotriene receptor antagonists

This drug, customarily used for asthma treatment, acts by preventing or alternating pathological factors caused by ongoing inflammatory process induced by leukotrienes C4, D4, and E4 [6]. A majority of sources advocate the use of Montelukast sodium in the management of EGE [18].

Antihistamine drugs

H1 antihistamines, such as Ketotifen, have been employed in the therapeutic management of EGE. Ketotifen is classified as a first-generation H1 antihistamine, which may cause sedation due to its ability to cross the blood-brain barrier; however, it also exhibits

activity as a mast cell stabilizer [14]. An article by Melamed et al. demonstrated the clinical and histological efficacy of this medication in the management of EGE, indicating that Ketotifen is recommended for adjunctive therapy with corticosteroids or leukotriene receptor antagonists [18].

Mast cell stabilizers

The objective of this class of drugs is to inhibit mast cell degranulation, which secondarily leads to a reduced release of histamine and other mediators associated with EGE [14]. The proposed medication is Cromolyn sodium, which has documented partial efficacy in cases involving the mucosal and subserosal forms of the disease, when administered either as monotherapy or in combination [16].

Other therapies

While for some patients first-line and second-line treatment methods are sufficient and yield the expected results, as with any condition, a subset of patients may be resistant to treatment or exhibit inadequate tolerance to the side effects of medications. In such cases, alternative therapies may be considered, including immunomodulators (e.g. azathioprine or 6-mercaptopurine), which are mainly used as substitutes in corticosteroid-dependent patients [14].

Biological medications

Based on the underlying pathways of EGE, which involve, for instance, an increased number of eosinophils or excessive cytokine release, the use of advanced biologic agents is a viable option. Their mechanism of action, which targets specific cytokines or cellular components, is currently being explored in clinical trials for the therapeutic management of EGE [14]. The initial reports regarding the effectiveness of biological agents in EGE treatment date back to 2007. At that time, the effectiveness of omalizumab was demonstrated. Nowadays, treatment concentrates on the three most developed drugs: dupilumab, cendakimab, and lirentelimab [20]. Nevertheless, it is important to note that many biologic medications directly targeting eosinophils are currently being tested. Among these, for example, are mepolizumab and benralizumab, which hold significant potential to become cornerstone treatments for EGE patients in the future [21,22].

Aim of the work

The aim of the systematic review was to analyze and summarize case reports on EGE in children, published over the past decade, with a focus on recognizing the most frequently reported symptoms, their duration, diagnostic assessments performed, and treatment methods proposed.

Methods

Search Strategy

To identify the available scientific articles, a detail search related to EGE was conducted according to the PRISMA guidelines.

A systematic literature search was performed between October and November 2024 using the electronic database PubMed (328 results). The articles were retrieved using the following keywords: (eosinophilic gastroenteritis) AND (child). Additionally, during the database search, a restriction was applied: articles were limited to a publication date from 2014 to 2024. No other limitations were used.

Out of the 328 results retrieved, 28 of them were selected for review [23-50]. All of the included articles were case reports or case series.

Data extraction and quality assessment

All retrieved results about eosinophilic gastroenteritis in children that included various types of scientific articles, contain both basic and detailed information about the disease, diagnostic methods, treatment innovations, as well as descriptions of instructive patient cases were carefully read and evaluated to reach a decision regarding their qualification for our systematic review.

In the first phase, after deleted duplicates, 323 titles and abstracts were carefully analyzed by the authors to find adequate articles. In the second phase, the full texts of 52 qualified articles were carefully read. The inclusion criteria were: full text available in English, case report or case series, patients' age under 18, availability of detailed data on the patients' course of treatment. We excluded studies without the gender of the patient stated.

After completing the second phase of the work, 28 articles meeting all the requirements were selected for the systematic review (details of the article selection process are presented in Figure 1).

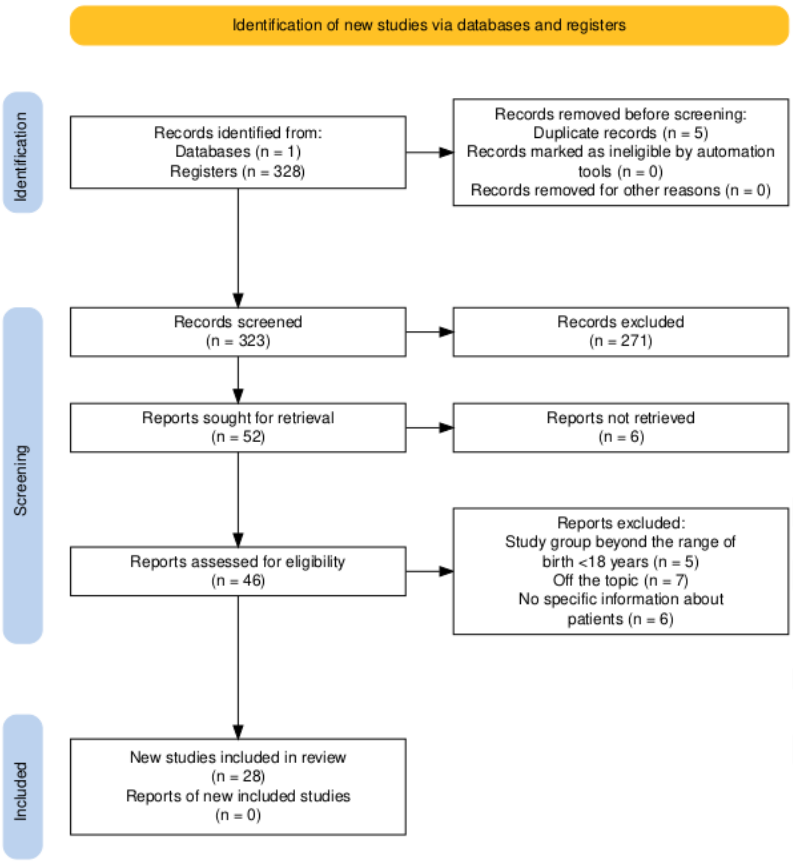

Figure 1. PRISMA 2020 flow diagram

We extracted items for the characteristics of the articles including article details, country, patient details such as gender, age, allergies, medical and family history, and EGE course, including first symptoms, duration of signs, patient examination, laboratory results, imaging, histopathology results, and treatment.

Assessment of the quality of studies

To assess the quality of the included studies, we used the JBI Critical Appraisal Checklist for Case Reports. We included details of the assessment in Table 1.

Table 1. Quality assessment

Article details	Were patient's demographic characteristics clearly described?	Was the patient's history clearly described and presented as a timeline?	Was the current clinical condition of the patient on presentation clearly described?	Were diagnostic tests or assessment methods and the results clearly described?	Was the intervention(s) or treatment procedure(s) clearly described?	Was the post-intervention clinical condition clearly described?	Were adverse events (harms) or unanticipated events identified or described?	Does the case report provide takeaway lessons?
Vengalil et al. 2020 [23]	Unclear	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Katiyar et al. 2016 [24]	Yes	Unclear	Unclear	Yes	Unclear	No	No	Unclear
Kakaje et al. 2020 [25]	Unclear	Yes	Yes	Yes	Yes	Yes	No	Yes
Di Mari et al. 2024 [26]	Yes	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Kimura et al. 2023 [27]	Yes	Unclear	Yes	Yes	Yes	Yes	N/A	Yes
Zhou et al. 2014 [28]	Unclear	Unclear	Yes	Yes	Unclear	Yes	No	Yes
Ming et al. 2015 [29]	Yes	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Kwon Ji Yoon et al. 2017 [30]	Unclear	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Nguyen et al. 2018 [31]	No	No	Yes	Yes	Unclear	Unclear	No	Unclear
van Hoeve et al. 2023 [32]	Unclear	Unclear	Yes	Yes	Yes	Yes	N/A	Yes
Peck et al. 2020 [33]	Yes	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Itoh et al. 2020 [34]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sasaki et al. 2020 [35]	Unclear	Yes	Unclear	Yes	Yes	Yes	N/A	Yes
Doi et al. 2020 [36]	Unclear	Unclear	Unclear	Yes	Yes	Yes	N/A	Yes
Shetty et al. 2017 [37]	Unclear	Yes	Yes	Yes	Yes	Unclear	N/A	Yes
Getsuwan et al. 2022 [38]	Unclear	Yes	Yes	Unclear	Yes	Yes	N/A	Yes
Mori et al. 2023 [39]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Unclear
Mizuo et al. 2020 [40]	Unclear	Yes	Yes	Yes	Yes	Yes	N/A	Unclear
Hagiwara et al. 2022 [41]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Avinashi et al. 2021 [42]	Unclear	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Stoecklein et al. 2021 [43]	Yes	Yes	Yes	Yes	Yes	Unclear	Yes	Yes
Fujita et al. 2022 [44]	Unclear	Unclear	Unclear	Yes	Yes	Yes	N/A	Yes
Manrriquez et al. 2020 [45] - case no. 1	Unclear	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Manrriquez et al. 2020 [45] - case no. 2	Unclear	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Pelizzo et al. 2018 [46]	Yes	Yes	Yes	Yes	No	N/A	N/A	Yes
Sasaki et al. 2019 [47]	Unclear	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Daidone et al. 2019 [48]	Yes	Unclear	Yes	Yes	Yes	Yes	N/A	Yes
Eren et al. 2020 [49] - case no. 1	Yes	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Eren et al. 2020 [49] - case no. 2	Unclear	Yes	Yes	Yes	Yes	Yes	N/A	Yes
Muir et al. 2018 [50]	Yes	Yes	Unclear	Yes	Yes	Yes	Yes	Yes

Notes: Own elaboration based on the JBI Critical Appraisal Tool for Case Reports.

Literature review results

Details of the study group

The research included 28 articles [23-50] published between 2014 and 2024. The largest number of articles regarding EGE was published in 2020, accounting for 32%. The articles originate from all parts of the world, with the largest proportion (36%) having been published in the United States.

After summarizing all the studies, a total of 30 cases of patients with EGE were gathered. Gender distribution: 19 patients were men (63%), and 11 were women (37%). The

age of the patients ranged from 10 weeks to 17 years, with an average of 9 years. The largest age groups were 11- and 12-year-olds, each comprising 4 cases (13%). The median age at diagnosis in the United States, the country of origin for the majority of reported patients, was 10 years. Moreover, the earliest diagnosis was made in the United States, at just 2 months of age.

Medical history of the patient and their family

Out of 30 selected cases, in 16.7% of the cases, information about medical history was not included, and in another 16.7% of the articles, medical history was defined as not relevant or unremarkable. Only one patient's medical history described in the selected cases had a previous diagnosis of EGE.

Among the discussed medical histories, the largest percentage was represented by allergic diseases: food allergies (16.7%), asthma (10%), and allergic rhinitis (6.7%).

In 13.3% of the medical histories, patients struggled with gastrointestinal diseases such as Crohn's disease, reflux esophagitis, or acute gastroenteritis. Additionally, 10% of the patients had been diagnosed with anemia. At the same time, 66.7% of the patients with anemia also presented with hypoalbuminemia.

In 6 cases, medical history was categorized under the "other" category and included conditions such as cerebral palsy, congenital congenital myotonic dystrophy type 1, thalassemia, hypereosinophilic syndrome, influenza A virus infection, or chronic pancreatitis with common bile duct dilation. Patients with more than one condition from the above-mentioned conditions constituted 13.3%.

Among the described patients, 11 were diagnosed with allergies. The protocols used to diagnose allergies in patients varied. In 7 cases, the diagnosis of an allergy was established on

a clinical basis. In 57% of these cases, serum-specific IgE testing was conducted; in 2 patients, skin prick tests were performed; and in one case, the diagnosis method was not specified. Additionally, in the remaining 4 patients, the diagnosis was based solely on information obtained during taking of the medical history. The majority of these patients (46%) exhibited both food and respiratory allergies. The most frequently observed food allergy was to egg, present in 5 patients. Other common food hypersensitivity included cow's milk (36%), fish, wheat, soy, peanuts, sesame (each affecting 18% of the patients), dairy (9%), yeast (9%), and food additives (9%).

Only 11% of the cases mentioned a family medical history. Furthermore, all of these cases referred to genetic allergies, with asthma being the most commonly mentioned.

Symptoms of EGE

The most prevalent symptom prompting patients to seek medical care was abdominal pain, reported by 63% of the patients. Vomiting emerged as the next most commonly observed symptom, noted by 47% of the patients, among whom bilious vomiting accounted for 14%. Additional symptoms reported by the patients included anorexia (23%), diarrhea (20%), manifestations of gastrointestinal bleeding (20%, e.g. melena or hematochezia), fatigue (13%), hematemesis (10%), anemia (10%), pyrexia (10%), nausea (10%), hypoproteinemia (7%), constipation (7%), dysphagia (3%), pruritus (3%), and lower extremity edema (3%).

The duration of symptoms was provided for 25 patients. The shortest duration was 1 day, while the longest persisted for 6 years. The mean duration of symptoms was approximately 6 months and 19 days.

Diagnostic process

The diagnostic approach demonstrated variability across the majority of cases. Notably, the essential procedure of physical examination was documented in only 50% of the cases. The most frequently reported abnormality was abdominal tenderness (20%), followed by edema (7%), growth/weight abnormalities (7%), ascites (7%), decreased bowel sounds (3%), and a palpable mass in the abdomen (3%).

Laboratory investigations were performed in 97% of the juveniles. The laboratory parameter that was most frequently elevated was eosinophil total count. In 53% of the patients, this parameter was elevated, with an average value of 9,273 cells/µl. Concurrently, a decrease in hemoglobin levels was observed in 12 patients, with a mean value of 8.95 g/dL and the lowest recorded level of 6.1 g/dL. Other laboratory parameters that were altered included: total IgE level (30%), albumin (23%), and C-reactive protein (13%). Additionally, stool tests were performed in 14 patients, of which 57% were within reference range, 21% showed elevated levels of calprotectin, and 14% had the presence of blood in the stool.

A consistent component for all patients involved the use of at least one imaging study, with endoscopy representing the predominant method (97%). During the endoscopic procedure, biopsy specimens were obtained from all patients except one. Thereafter, the samples were referred for histopathological examination, in the course of which, the most frequently observed result was eosinophilic infiltration (demonstrated in 86% of the findings, with an average value of 68.35 per high power field). Only one histopathological examination result did not demonstrate any abnormalities.

Other commonly selected imaging studies included: ultrasonography (37%), computed tomography (27%), radiography (10%), magnetic resonance cholangiopancreatography (7%), along with laparotomy (7%). Infrequently utilized methods (employed in only 3% of the

patients) include MR imaging, endoscopic ultrasound, barium contrast X-ray, and magnetic resonance enterography.

Treatment process

During the treatment process, a total of 15 patients were administered corticosteroids, which constituted the most commonly used class of medications in the management of EGE in the retrieved articles. Corticosteroids were administered across nearly all age groups, with the exception of patients younger than 1 year of age. In the largest age group (11-12 years), corticosteroids were employed in all of the described treatment approaches. The preferred drug was prednisone (47%). Treatment for 8 patients included dietary adjustments based on identified food allergies. Antihistamines (23%) and proton pump inhibitors (20%) were also commonly prescribed.

Some patients also received second-line therapies, including leukotriene receptor antagonists (17%) and biologic agents (10%).

Surgical intervention was required in 3 cases and included procedures such as resection of the small intestine, gastrectomy, and =percutaneous transhepatic biliary drainage (PTBD).

Other medications used in individual cases included methotrexate, metronidazole, suplatast tosilate, and antibiotics (gentamycyn, ceftriaxone, and ticarcillin-clavulanate).

Depending on the patients' test results, additional treatments were applied, including iron supplementation (10%), protein supplementation (7%), albumin infusions, fluid therapy, or red cell transfusion (each required in 3% of the patients). Detailed findings are provided in Table 2.

Table 2. Summary of the information collected from the articles [19-46]

Article	Place/nation	Gender	Age (years)	First symptoms	Duration (months)	Allergies	Family history	Medical history	Examination	Laboratory	Endoscopy	Eosinophilic infiltration	Imaging other	Treatment
Vengalil et al. 2020 [23]	England	Female	11	Abdominal pain, recurrent bilious vomiting, loss of appetite, ascites	2	Shellfish and other fish, yeast and food additives	N/A	Unremarkable	N/A	Leucocyte (27,500/ul), Absolute eosinophil count 9000, IgE 2633 IU/ml, stool examinations – negative	YES	N/A	Abdominal USG	Prednisone
Katiyar et al. 2016 [24]	Netherlands	Female	3	Vomiting, fever, hematemesis	2	N/A	N/A	N/A	N/A	Hemoglobin 10 g/dl	YES	80/HPF	Barium contrast X-ray	Distal gastrectomy with gastro-duodenal anastomosis
Kakaje et al. 2020 [25]	Netherlands	Female	3.5	Recurrent abdominal pain, fever and chills, bilious vomiting	0.25	N/A	Grand-mother asthma	Unremarkable	Tendreness over abdominal area	White cell count of 29,000 * 109 per L, hemoglobin level of 10.6 g/dL, CRP of 94.2 mg/L	YES	20/HPF	Ultrasonography	Metronidazole, ceftriaxone, urgent surgery with resection
Di Mari et al. 2024 [26]	Switzerland	Male	14	Upper abdominal pain, repeated nonbilious vomiting, occasional dysphagia	12	Inhalants	Allergic asthma and psoriasis	Recurrent aphthous stomatitis, allergic oculorhinitis from age 8	Upper abdominal tenderness	Eosinophilia (Eo 1,000/mm3), increased total IgE (341 kU/L), fecal calprotectin and parasitological examination resulted negative	YES	60/HPF	Ultrasound, magnetic resonance cholangio-pancreatography	Steroids (prednisone), proton pump inhibitors
Kimura et al. 2023 [27]	United States	Male	10	Abdominal pain fatigue, severe anemia with hypo-proteinemia	6	N/A	Father - *H. pylori*, anemia	N/A	Pale eyelid conjunctiva	Hemoglobin (9.7 g/dL), albumin (2.9 g/dL), fecal analysis elevated levels of human hemoglobin (2018 ng/mL) and calprotectin (510 µg/g)	YES	80/HPF	N/A	Red blood cell transfusion, iron supplementation, montelukast, elemental diet therapy

Zhou et al. 2014 [28]	Germany	Male	11	Recurrent abdominal pain, diarrhea	0.625	N/A	N/A	N/A	Tenderness at McBurney's point without rebound tenderness, no urinary symptoms	White blood cell count of 22×106/dL, eosinophil count of up to 45%, stool examination was negative for parasites or ova	YES	Increased	Sonographic, CT	Corticosteroids
Ming et al. 2015 [29]	India	Male	11	Abdominal pain	0.25	Egg	N/A	No relevant medical history presented	The patient looked pale, periumbilical tenderness with ascites	WBC 7.98×109/L eosinophil levels 31%, total IgE 354 mIU/mL, stool exam negative	YES	40/HPF	Ultrasonography, CT	Prednisolone, cetirizine
Kwon Ji Yoon et al. 2017 [30]	Korea	Female	9	Severe, colicky abdominal pain with bilious vomiting and loss of appetite, no urination for 12 hours	0.25	N/A	N/A	Influenza A virus infection, no history of abdominal surgery, allergic disease, or food sensitivity	Abdomen markedly distended, bowel sounds decreased, tenderness evident over the entire abdomen with shifting abdominal dullness, no rebound tenderness	White blood cell count of 12,870/mm3, hemoglobin level of 17.1 g/dL×103/mm3, albumin, 3.9 g/dL, C-reactive protein, 20.2 mg/dL, stool calprotectin level increased to 1,383.0 mg/kg	YES	20/HPF	Radiograph, CT	Intravenous fluid therapy, prednisolone
Nguyen et al. 2018 [31]	United States	Male	2	Fatigue, anorexia, iron deficiency anemia	N/A	N/A	N/A	N/A	N/A	Hemoglobin 8.5 g/dl, hemoccult positive stool, eosinophilia (880 cells/µl)	YES	72/HPF	N/A	Budesonide
van Hoeve et al. 2023 [32]	Belgium	Female	4	Progressive orbital and peripheral edema of the lower limbs, abdominal pain, non-bloody diarrhea and intermittent vomiting	2	N/A	N/A	N/A	Generalized edema with 10% weight gain.	Negative stool samples, albumin of 16.3 g/L, hemoglobulin (g/dL) 11.5	YES	120/HPF	N/A	Albumin infusions, loop diuretics

Peck et al. 2020 [33]	United States	Male	16	Progressive pruritus, jaundice, fatigue, abdominal pain, acholic stools, and dark-colored urine	1.5	N/A	N/A	Reflux esophagitis, duodenal ulcer (three years prior by upper endoscopy and treated with proton pump inhibitor therapy)	N/A	C-reactive protein (CRP) < 0.5 mg/L, absolute eosinophil count (AEC) 1,000 eosinophils per microliter (<500)	YES	N/A	Abdominal ultrasound, magnetic resonance cholangio-pancreatography (MRCP)	Intravenous antibiotic therapy with ticarcillin-clavulanate
Itoh et al. 2020 [34]	Australia	Female	4	Weight loss, persistent diarrhea, hematochezia	1	Egg and cow's milk, dairy	N/A	Urtucaria during weaning by CM and egg Ecterocolitis treated with Bigidobacterium probiotics	N/A	White blood cell count 24 700/μL, eosinophilis 9880/μl	YES	100/HPF	Computed tomography, Bone marrow examination	Fasting, parenteral nutrition
Sasaki et al. 2020 [35]	Japan	Male	9	Intermittent abdominal pain	2	Egg, seasonal allergic rhinitis	N/A	Acute gastroenteritis, he was prescribed probiotics.	N/A	Eosinophil count of 660 cells/μL, feces tested negative for occult blood and enteric pathogens	YES	50/HPF	N/A	Histamine H1 receptor antagonist, leukotriene antagonist
Doi et al. 2020 [36]	Australia	Male	8	Chronic diarrhea	48	egg, cow's milk, wheat, and soy	N/A	N/A.	N/A	Peripheral eosinophil count 2,362.5/μl, total IgE 5,570 IU/ml, white blood cell count 11,250/μL	YES	40/HPF	Ultrasonography, computed tomography	Fexofenadine, suplatast tosilate, montelukast
Shetty et al. 2017 [37]	United States	Female	0.2	Projectile hematemesis	0.033	N/A	N/A	No relevant medical history presented	Well-appearing on exam, stable vital signs, no evidence of hemodynamic instability	Hemoglobin 7.4 g/dL	YES	Increased	X-ray	H2 receptor antagonist

Getsuwan et al. 2022 [38]	United States	Male	12	Vomiting and abdominal pain	0.1	N/A	N/A	Hemoglobin E-b-thalassemia, matched unrelated male donor bone marrow transplantation (BMT) at the age of 5 years. Took immunosuppressive agents for 1 year after BMT without any documented history of GVHD	Distended abdomen and ascites without any signs of hepatosplenomegaly, edema, peritonitis or lymphadenopathy	Hemoglobin 14.2 g/dL, total white blood cells 19,300/mm3, absolute eosinophil count of 9,457/mm3, albumin of 3.0 g/dL, stool ova and parasites were unremarkable	NO	N/A	Ultrasound, computed tomography, abdominal paracentesis	Methylprednisolone
Mori et al. 2023 [39]	England	Male	14	Nausea, abdominal pain, poor appetite, recurrent vomiting, microcytic anemia, and weight loss	72	Seasonal/ perennial aeroallergens	N/A	In 2017 EGE diagnosed. treatment fail with PPI, corticosteroid, diet, clinical histological remission achieved with dupilumab	N/A	Eosinophil count of 2061 cells/μL	YES	60/HPF	N/A	Budesonide, proton pump inhibitors, prednisolone, empiric diet without eggs and milk
Mizuo et al. 2020 [40]	United States	Male	10	Protein-losing gastroenteropathy with persistent vomiting	1	N/A	N/A	Anemia and diarrhea, hypoalbuminemia.	N/A	White blood cell count (4,100/μL), eosinophils (164/μL), high IgE (1,793 IU/mL)	YES	Increased	N/A	Leukotriene receptor-1 antagonist, sodium cromoglicate
Hagiwara et al. 2022 [41]	Netherlands	Male	11	Persistent nausea and epigastric pain	0.25	N/A	N/A	Diagnosed with FIP1L1-PDGFRA fusion-negative HES with eosinophilic cystitis and had been	Epigastric tenderness	Eosinophilia (2,254 eos/μL)	YES	<20/HPF	Ultrasonography	Corticosteroid, tacrolimus

								taking a small dose of corticosteroids for 9 years						
Avinashi et al. 2021 [42]	United States	Male	12	Daily non-bilious, non-bloody vomiting	0.875	Allergic rhinitis, and food allergies (peanut, tree nuts, finned fish, sesame)	N/A	ASTHMA, OIT- minor nausea within an hour of the dose.	N/A	N/A	YES	100/HPF	N/A	Budesonide
Stoecklein et al. 2021 [43]	United States	Female	4	Pallor and decreased energy	N/A	N/A	N/A	Anemia and hypo-albuminemia treated with oral iron, in infancy - obstruction from volvulus complicating malrotation macrocephaly, asthma treated with montelukast, tonsillar adeno-idectomy	Diffuse area of swelling on the right thigh consistent with a lipoma, mild lower extremity pitting edema	Eosinophil count 540 cells/μL, albumin 2.3 g/dL, hemoglobin 6.6 g/dL, stool examination - calprotectin 544.1 μg/mg	YES	N/A	N/A	Budesonide
Fujita et al. 2022 [44]	Japan	Male	12	Epigastric pain	1	N/A	N/A	No relevant medical history presented	N/A	White blood cell count, 4,700/μL; eosinophil count, 150/μL (3.2%); albumin level, 4.5 g/dL; C-reactive protein level, < 0.01 mg/dL; total IgE, 151.6 IU/L	YES	100/HPF	Ultrasonography	Esomeprazole

Manrrique z et al. 2020 [45]	United States	Female	13	Acute-on-chronic abdominal pain, emesis	N/A	Sesame, wheat, milk, corn, soy, peanut, egg, atopic dermatitis	N/A	Asthma	Nontender, ill-defined mobile mass was palpable along the left lower quadrant on examination	Normal level of hemoglobin, platelets, undetectable C-reactive protein	YES	100/HPF	CT	*H. pylori* - triple therapy, elemental nutrition therapy, prednisone
		Female	13	Emesis, decreased oral intake, abdominal pain, constipation	12	N/A	N/A	Chronic pancreatitis, status post sphincterotomy for common bile duct dilation	N/A	Hemoglobin 8.5 g/dL, C-reactive protein 0.9 mg/dL, absolute eosinophil count 630 k/UL, percent eosinophils 8%, elevated total IgE 2808 kU/L	YES	85/HPF	MRI	Protein formula supplementation, soft mechanical diet, EEN therapy, prednisolone
Pelizzo et al. 2018 [46]	Turkey	Male	12	Symptoms of bowel obstruction, abdominal pain, vomiting, diarrhea, abdominal tenderness, absence of bowel sounds	0.07	N/A	N/A	Congenital MD type 1 (cytosine-thymine-guanine triplet expansion in the 3'-non-translated region of the dystrophia myotonica protein kinase gene), surgery for intestinal obstruction, no surgical or histological documentation	N/A	Increased C-reactive protein, mild elevation in hemoglobin level. The white blood cell count (including eosinophils), red blood cell count were within normal limits.	YES	Increased	X-ray, Explorative laparoscopy	N/A
Sasaki et al. 2019 [47]	Japan	Male	7	Continuous abdominal pain, vomiting	N/A	Milk, allergic rhinitis	N/A	Big elimination diet, treatment for rhinitis - cetrizine, pranlukastm suplast tosylate	No abnormal findings, except of short stature (H 107.8 cm)	White cell count 7600/mm2, eosinophils 2,052, hemoglobin 11.5, platelet count 17, CRP 0.08, albumin 3.1, total protein 6.6, IgE 20,982 IU/l	YES	20/HPF	CT	Diet, pranlukast, cetirizine, steroids

Daidone et al. 2019 [48]	England	Female	0.3	Melena, vomiting	1	N/A	N/A	N/A	Well-appearing, unremarkable physical examination, weight was 5,930 g (25°-50°). A rhinoscopy ruled out upper airway bleeding	WBC 12,780/mmc, Hb 10.1 g/dl, CPR 69.2 mg/L	YES	Increased	Ultrasonography	Feeding was stopped for 24 h, proton pump inhibitor treatment
Eren et al. 2020 [49]	Turkey	Male	9	Fever, melena, hematemesis	0.67	N/A	N/A	Cerebral palsy, percutaneous endoscopic gastrostomy and a tracheostomy tube	No sign of peristomal blood leakage or wound site infection	White blood cells (30,600 µL), albumin (2.5 g/dL), hemoglobin 6.9 g/dL	YES	80/HPF	N/A	Peptide-based enteral solution, lansoprazole, gaviscon treatment
		Male	17	Nausea, melena	0.1	Pollen, mite, cat allergy	N/A	Anemia - iron treatment for 3 months	N/A	Hemoglobin 6.1 g/dL, stool examination was negative	YES	40/HPF	N/A	Cow's milk elimination diet
Muir et al. 2018 [50]	United States	Male	10	Abdominal pain, diarrhea	N/A	N/A	N/A	Upper tract and ileocolonic Crohn's disease diagnosed at the age of 8 years	N/A	Eosinophilia, total count 5,120, elevated IgE, albumin 3 g/dl, ova and parasite testing was negative	YES	100/HPF	N/A	Prednisone, diet elimination, infliximab, methotrexate

Discussion of the review results

This systematic review highlights the importance of expanding knowledge regarding the etiology, clinical manifestations, diagnostic methods, and therapeutic approaches for EGE. Among the available scientific articles on the topic of our review, particularly among the 28 included studies, we observed a remarkable heterogeneity both in clinical presentation and in diagnostic and treatment protocols. Similarities, which were mainly observed in the part reporting patients' symptoms, confirmed the predominance of gastrointestinal discomfort in EGE. Abdominal pain was the most frequently reported symptom by over 60% of the patients, vomiting stood out as the second most common clinical manifestation (47%). Other symptoms such as anorexia (23%), diarrhea (20%), gastrointestinal bleeding manifestations (20%), and anemia (10%) illustrate a broad, nonspecific clinical spectrum, which substantially complicates early diagnosis.

The duration of symptoms ranged from one day to as much as six years, with an average of six months, reflecting a chronic and often insidious disease course. This wide variability contributed to diagnostic delays and underscored the need for greater clinical vigilance.

Laboratory investigations demonstrated elevated peripheral eosinophil counts in 53% of the patients, with a mean value of 9,273 cells/µl. Reduced hemoglobin levels were identified in 12 patients (mean value 8.95 g/dL), while hypoalbuminemia was reported in 23%. These findings suggest chronic inflammation and potential nutritional deficiencies. At the same time, increased total IgE levels were observed in 30% of the cases, correlating with the high prevalence of allergic comorbidities. Among patients with allergies, 46% exhibited both food and respiratory hypersensitivities. Meanwhile, the most common food allergy was to egg, occurring in 17% of the patients.

Significant variability was likewise noted in the diagnostic process. Physical examination findings were reported in only half of the cases, most commonly indicating abdominal tenderness. Laboratory testing was performed in nearly all patients (97%), emphasizing its fundamental role in the diagnostic assessment. Nevertheless, discrepancies in both diagnostic and therapeutic approaches reflect the absence of standardized protocols, a consequence of the disease's rarity and nonspecific clinical features.

Despite some similarities in symptoms, treatment strategies, and diagnostic approach, it is not possible to identify two identical medical protocols. These disparities are not dependent on the geographical location of the patients. The likely cause of these differences is the lack of characteristic symptoms and the rarity of the disease. As a result, physicians worldwide often first consider more common conditions, without associating abnormal white blood cell and eosinophil total count result with gastrointestinal symptoms.

As indicated by Li et al., awareness and recognition of EGE among clinicians is gradually improving, particularly the association between peripheral eosinophilia and gastrointestinal symptoms [51]. This forms a solid foundation for the continued advancement of awareness, the development of diagnostic methods, and treatment strategies based on medications with minimal side effects, aiming to enhance the quality of life for patients, particularly adolescents, with eosinophilic gastroenteritis.

The findings of this review emphasize the necessity of future research focusing on the identification of sensitive and specific biomarkers, as well as the establishment of standardized diagnostic criteria and treatment algorithms. These efforts are crucial for enhancing quality of life, especially for children and adolescents, who often face significant delays in diagnosis and variability in treatment outcomes.

Limitations

This article, summarizing the available knowledge on EGE, is subject to certain limitations. Although it offers valuable insights into the clinical presentation, diagnostic challenges, and treatment approaches in pediatric EGE, it is subject to several inherent biases. The primary constraint arises from the inherent heterogeneity across the included case reports, particularly in diagnostic approaches, therapeutic strategies, and the extent of clinical documentation. The absence of standardized protocols impedes meaningful comparative analyses and may introduce reporting bias due to inconsistencies in the description of symptoms, laboratory findings, and treatment outcomes. Moreover, the largely descriptive nature of the available data precludes quantitative synthesis and limits the ability to derive robust conclusions regarding treatment efficacy and prognostic indicators. The insufficient reporting of family history and incomplete characterization of allergic profiles further complicate the evaluation of potential etiological associations and genetic predispositions. Additionally, the wide variation in symptom duration among the cases hampers the ability to elucidate the true natural history of EGE and may obscure relevant prognostic factors. These limitations underscore the need for future prospective, multicenter studies employing standardized diagnostic criteria and uniform reporting frameworks to enable more precise phenotypic stratification and to advance the development of evidence-based clinical guidelines.

Conclusions

EGE is a rare gastrointestinal disorder. The nonspecific nature of its symptoms often significantly prolongs the time required to establish an accurate diagnosis. Regrettably,

prolonged diagnostic delays, particularly in pediatric patients, may result in severe outcomes, including profound malnutrition and significant systemic deterioration. It is imperative to develop standardized management protocols for EGE, which would significantly expedite and facilitate the diagnostic process for this condition. Further advancement of knowledge regarding EGE is highly warranted and may lead to the development of novel therapeutic approaches, offering substantial benefits for affected patients.

Disclosures and acknowledgements

The authors declare no conflicts of interest with respect to the research, authorship, and/or publication of this article.

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Artificial intelligence, ChatGPT, was used initially by the authors in the process of checking the grammatical and stylistic correctness when translating the text into English. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

References:

1. Al Maksoud AM, Ahmed AS, O'Donnell N. Obstructive eosinophilic gastroenteritis in a patient with rheumatoid arthritis. BMJ Case Rep. 2015; 2015: bcr2015210962. https://doi.org/10.1136/bcr-2015-210962
2. Dunn JLM, Spencer LA. Pathophysiology of non-esophageal eosinophilic gastrointestinal disorders. Immunol Allergy Clin North Am. 2024; 44(2): 299-309. https://doi.org/10.1016/j.iac.2024.01.003

3. Shih HM, Bair MJ, Chen HL, Lin IT. Eosinophilic gastroenteritis: brief review. Acta Gastroenterol Belg. 2016; 79(2): 239-244.
4. Pineton de Chambrun G, Gonzalez F, Canva JY, Gonzalez S, Houssin L, Desreumaux P, et al. Natural history of eosinophilic gastroenteritis. Clin Gastroenterol Hepatol. 2011; 9(11): 950-956.e1. https://doi.org/10.1016/j.cgh.2011.07.017
5. Havlichek D, Choung RS, Murray JA. Eosinophilic gastroenteritis: using presenting findings to predict disease course. Clin Transl Gastroenterol. 2021; 12(10): e00394. https://doi.org/10.14309/ctg.0000000000000394
6. Ingle SB, Hinge Ingle CR. Eosinophilic gastroenteritis: an unusual type of gastroenteritis. World J Gastroenterol. 2013; 19(31): 5061-5066.. https://doi.org/10.3748/wjg.v19.i31.5061
7. Waheed MFA, Bakhshi GD, Rangwala Z, Patel OA, Mohan A, Jain U. Eosinophilic enteritis with enteroliths: a diagnostic dilemma. Int J Surg Case Rep. 2021; 89: 106571. https://doi.org/10.1016/j.ijscr.2021.106571
8. Koutri E, Papadopoulou A. Eosinophilic gastrointestinal diseases in childhood. Ann Nutr Metab. 2018; 73(Suppl. 4): 18-28. https://doi.org/10.1159/000493668
9. Galere P, Gilbert A. Eosinophilic gastroenteritis with ascites: an enigmatic diagnosis. Lancet. 2022; 400(10346): 126. https://doi.org/10.1016/S0140-6736(22)01056-X
10. Melboucy-Belkhir S, Khentache R, André-Ledun L, Brihaye B. Eosinophilic ascites, a challending diagnosis. Int Arch Intern Med. 2018; 2(1): 009. https://doi.org/10.23937/iaim-2017/1710009
11. Li K, Ruan G, Liu S, Xu T, Guan K, Li J, Li J. Eosinophilic gastroenteritis: pathogenesis, diagnosis, and treatment. Chin Med J (Engl). 2023; 136(8): 899-909. https://doi.org/10.1097/CM9.0000000000002511

12. Conus S, Simon HU. General laboratory diagnostics of eosinophilic GI diseases. Best Pract Res Clin Gastroenterol. 2008; 22(3): 441-453. https://doi.org/10.1016/j.bpg.2007.09.003
13. Kuźmiński A, Rosada T, Przybyszewska J, Ukleja-Sokołowska N, Bartuzi Z. Eosinophilic gastroenteritis – a manifestation of an allergic disease in the gastrointestinal tract? Part 1. Epidemiology and diagnosis. Prz Gastroenterol. 2023; 18(1): 43-46. https://doi.org/10.5114/pg.2022.118634
14. Sunkara T, Rawla P, Yarlagadda KS, Gaduputi V. Eosinophilic gastroenteritis: diagnosis and clinical perspectives. Clin Exp Gastroenterol. 2019; 12: 239-253. https://doi.org/10.2147/CEG.S173130
15. Chen PH, Anderson L, Zhang K, Weiss GA. Eosinophilic gastritis/gastroenteritis. Curr Gastroenterol Rep. 2021; 23(8): 13. https://doi.org/10.1007/s11894-021-00809-2
16. Uppal V, Kreiger P, Kutsch E. Eosinophilic Gastroenteritis and colitis: a comprehensive review. Clin Rev Allergy Immunol. 2016; 50(2): 175-88. https://doi.org/10.1007/s12016-015-8489-4
17. Higuchi T, Tokunaga M, Murai T, Takeuchi K, Nakayama Y. Elemental diet therapy for eosinophilic gastroenteritis and dietary habits. Pediatr Int. 2022; 64(1): e14894. https://doi.org/10.1111/ped.14894
18. Abou Rached A, El Hajj W. Eosinophilic gastroenteritis: approach to diagnosis and management. World J Gastrointest Pharmacol Ther. 2016; 7(4): 513-523. https://doi.org/10.4292/wjgpt.v7.i4.513
19. Grandinetti T, Biedermann L, Bussmann C, Straumann A, Hruz P. Eosinophilic gastroenteritis: clinical manifestation, natural course, and evaluation of treatment with corticosteroids and vedolizumab. Dig Dis Sci. 2019; 64(8): 2231-2241. https://doi.org/10.1007/s10620-019-05617-3

20. Dellon ES, Spergel JM. Biologics in eosinophilic gastrointestinal diseases. Ann Allergy Asthma Immunol. 2023; 130(1): 21-27. https://doi.org/10.1016/j.anai.2022.06.015
21. Kliewer KL, Murray-Petzold C, Collins MH, Abonia JP, Bolton SM, DiTommaso LA, et al. Benralizumab for eosinophilic gastritis: a single-site, randomised, double-blind, placebo-controlled, phase 2 trial. Lancet Gastroenterol Hepatol. 2023; 8(9): 803-815. https://doi.org/10.1016/S2468-1253(23)00145-0
22. Dellon ES, Peterson KA, Mitlyng BL, Iuga A, Bookhout CE, Cortright LM, et al. Mepolizumab for treatment of adolescents and adults with eosinophilic oesophagitis: a multicentre, randomised, double-blind, placebo-controlled clinical trial. Gut. 2023; 72(10): 1828-1837. https://doi.org/10.1136/gutjnl-2023-330337
23. Menon J, Venkatesh V, Bhatia A, Rana SS, Lal SB. Ascites: an unusual presentation of eosinophilic gastroenteritis in a child. Trop Doct. 2020; 50(3): 277-279. https://doi.org/10.1177/0049475520911230
24. Katiyar R, Patne SC, Dixit VK, Sharma SP. Primary eosinophilic gastritis in a child with gastric outlet obstruction. J Gastrointest Surg. 2016; 20(6): 1270-1271. https://doi.org/10.1007/s11605-016-3074-6
25. Kakaje A, Hedar N, Alali Alahmad N. Eosinophilic gastroenteritis in small intestine in a child in a remote medical centre required surgery. Int J Surg Case Rep. 2020; 71: 209-212. https://doi.org/10.1016/j.ijscr.2020.05.029
26. Di Mari C, Pozzi E, Mantegazza C, Destro F, Meroni M, Coletta M, et al. Duodenal stenosis, an unusual presentation of eosinophilic gastroenteritis: a case report. Front Pediatr. 2024; 12: 1390946. https://doi.org/10.3389/fped.2024.1390946
27. Kimura K, Jimbo K, Arai N, Sato M, Suzuki M, Kudo T, et al. Eosinophilic enteritis requiring differentiation from chronic enteropathy associated with *SLCO2A1* gene: A case

report. World J Gastroenterol. 2023; 29(11): 1757-1764. https://doi.org/10.3748/wjg.v29.i11.1757

28. Zhou HC, Lai C, Yang L. Eosinophilic gastroenteritis with involvement of the urinary bladder. Pediatr Radiol. 2014; 44(11): 1454-1457. https://doi.org/10.1007/s00247-014-3012-2
29. Ming G, Bo Y, Li-Ping Y. Eosinophilic gastroenteritis with ascites in a child. Indian Pediatr. 2015; 52(8): 707-708. https://doi.org/10.1007/s13312-015-0703-1
30. Kwon JY, Huh JS, Je BK, Hong KD, Lee JH. Eosinophilic gastrointestinal disorder presenting as intractable vomiting and ascites in a young girl. Pediatr Gastroenterol Hepatol Nutr. 2017; 20(3): 198-203. https://doi.org/10.5223/pghn.2017.20.3.198
31. Nguyen N, Kramer RE, Friedlander JA. Videocapsule endoscopy identifies small bowel lesions in patients with eosinophilic enteritis. Clin Gastroenterol Hepatol. 2018; 16(6): e64-e65. https://doi.org/10.1016/j.cgh.2017.08.043
32. van Hoeve K, De Keukelaere M, De Hertogh G, Hoffman I. Child with protein losing enteropathy as presentation of collagenous duodenitis and eosinophilic gastroenteritis. Acta Gastroenterol Belg. 2023; 86(2): 363-366. https://doi.org/10.51821/86.2.9374
33. Peck J, Kimsey KM, Harris E, Monforte H, Wilsey M Jr. Solitary duodenal ulcer causing biliary obstruction requiring rendezvous procedure in a pediatric patient with eosinophilic gastroenteritis. Cureus. 2020; 12(7): e9377. https://doi.org/10.7759/cureus.9377
34. Itoh N, Murai H, Kawasaki A, Suzuki K, Ohshima Y. Eosinophilic gastroenteritis developed after remission of cow's milk allergy. Pediatr Int. 2020; 62(2): 233-234. https://doi.org/10.1111/ped.14078
35. Sasaki Y, Kajino H. Eosinophilic gastroenteritis with persistent abdominal pain: a case report. J Rural Med. 2020; 15(1): 44-46. https://doi.org/10.2185/jrm.2019-009

36. Doi M, Furuichi Y, Tsuji S, Takano T. Eosinophilic gastroenteritis treated with a targeted food elimination diet. Pediatr Int. 2020; 62(7): 866-868. https://doi.org/10.1111/ped.14217
37. Shetty V, Daniel KE, Kesavan A. Hematemesis as initial presentation in a 10-week-old infant with eosinophilic gastroenteritis. Case Rep Pediatr. 2017; 2017: 2391417. https://doi.org/10.1155/2017/2391417
38. Getsuwan S, Tanpowpong P, Hongeng S, Anurathapan U, Pakakasama S, Treepongkaruna S. Ruxolitinib treatment in an adolescent with chronic graft-versus-host disease mimicking eosinophilic gastrointestinal disorders: a case report. Transplant Proc. 2022; 54(6): 1675-1678. https://doi.org/10.1016/j.transproceed.2022.05.003
39. Mori F, Renzo S, Barni S, Scarallo L, Giovannini M, Villanacci V, et al. Dupilumab treatment of eosinophilic gastrointestinal disease in an adolescent. Pediatr Allergy Immunol. 2023; 34(6): e13973. https://doi.org/10.1111/pai.13973
40. Mizuo A, Kondo S, Kobara H, Nishiyama N, Kondo T, Ishikawa R, et al. Appearance of gastric polypoid lesions in eosinophilic gastroenteritis. J Pediatr Gastroenterol Nutr. 2020; 70(4): e84. https://doi.org/10.1097/MPG.0000000000002429
41. Hagiwara SI, Ueki S, Watanabe K, Hizuka K, Etani Y. Case of hypereosinophilic syndrome with gastrointestinal involvement showing tissue eosinophil cytolysis. Asia Pac Allergy. 2022; 12(4): e37. https://doi.org/10.5415/apallergy.2022.12.e37
42. Avinashi V, Al Yarubi Z, Soller L, Lam G, Chan ES. Oral peanut immunotherapy acutely unmasking eosinophilic esophagitis with an esophageal stricture. Ann Allergy Asthma Immunol. 2021; 127(6): 691-692. https://doi.org/10.1016/j.anai.2021.09.001
43. Stoecklein N, Ahmed AA, Lawson CE, Attard T. PTEN hamartoma syndrome in a child presenting with malrotation, panintestinal polyps, severe anemia, and protein-losing

enteropathy. JPGN Rep. 2021; 2(3): e092. https://doi.org/10.1097/PG9.0000000000000092

44. Fujita Y, Tominaga K, Ishida K, Masuyama H, Yoshihara S. proton pump inhibitor to treat an eosinophilic duodenal ulcer with esophageal involvement: a pediatric case. Tohoku J Exp Med. 2022; 257(4): 309-313. https://doi.org/10.1620/tjem.2022.J045
45. Manrriquez A, Alharbi O, Braskett M, Bhardwaj V. Mural eosinophilic gastrointestinal disease in 2 pediatric patients presenting as focal mass. Pediatrics. 2020; 145(3): e20191610. https://doi.org/10.1542/peds.2019-1610
46. Pelizzo G, Calcaterra V, Villanacci V, Mura GB, Bassotti G. Myotonic dystrophy type 1 and pseudo-obstruction in a child with smooth muscle α-actin deficiency and eosinophilic myenteric plexitis. Turk J Gastroenterol. 2018; 29(2): 226-229. https://doi.org/10.5152/tjg.2018.17582
47. Sasaki A, Sugimoto M, Tokaji N, Irahara M, Okamoto K, Uehara H, et al. Efficacy of an elimination diet in a patient with eosinophilic gastroenteritis: a pediatric case with multiple food allergies. J Med Invest. 2019; 66(1.2): 201-204. https://doi.org/10.2152/jmi.66.201
48. Daidone A, Barbi E, Villanacci V, Di Leo G. Severe anaemia after gastric biopsy in an infant with eosinophilic gastritis. Ital J Pediatr. 2019; 45(1): 69. https://doi.org/10.1186/s13052-019-0661-7
49. Eren M, Uluğ N, Aydemir Y. Eosinophilic gastroenteritis as a cause of gastrointestinal tract bleeding and protein-losing enteropathy. Turk Pediatri Ars. 2020; 55(3): 299-303. https://doi.org/10.14744/TurkPediatriArs.2018.48376
50. Muir A, Surrey L, Kriegermeier A, Shaikhkalil A, Piccoli DA. Severe eosinophilic gastroenteritis in a crohn's disease patient treated with infliximab and adalimumab. Am J Gastroenterol. 2016; 111(3): 437-438. https://doi.org/10.1038/ajg.2015.438

51. Li K, Ruan G, Liu S, Xu T, Guan K, Li J, et al. Eosinophilic gastroenteritis: pathogenesis, diagnosis, and treatment. Chin Med J (Engl). 2023; 136(8): 899-909. https://doi.org/10.1097/CM9.0000000000002511