REVIEW

Eosinophilic ascites and eosinophilic gastrointestinal diseases

David Longhino[1], Irene Spinelli[2], Francesca Fianchi[2], Federica Castri[3], Gianluca Ianiro[2], Francesca Romana Ponziani[2], Cristiano Caruso[1], Alessandro Buonomo[1], Gasbarrini Antonio[2], Arianna Aruanno[1] *

[1] Allergy and Clinical Immunology Unit, Fondazione Policlinico Universitario A. Gemelli IRCCS, Università Cattolica del Sacro Cuore, Rome, Italy

[2] Centro Malattie Apparato Digerente (CEMAD), Fondazione Policlinico Universitario A. Gemelli IRCCS, Rome, Italy

[3] Division of Anatomic Pathology and Histology, Fondazione Policlinico Universitario A. Gemelli IRCCS, Università Cattolica del Sacro Cuore, Rome, Italy

Summary

Background: Eosinophilic ascites (EA) is characterised by a high eosinophil count in the ascitic fluid and, although very rare, is mainly caused by eosinophilic gastrointestinal diseases (EGIDs), parasitic infections, and hypereosinophilic syndrome (HES). Symptoms caused by EGIDs are varied and depend on the gastrointestinal tract affected as well as the layer(s) involved; typically, EA implies a serosal involvement. Herein, we

report our single Centre experience of three cases of EA in patients suffering from EGIDs together with a systematic review of all the other cases described in the literature.

Methods: Patients selection was performed by querying the database containing patients suffering from EGIDs referred to our hospital and extracting all cases with eosinophilic ascites. In order to describe the reported cases of eosinophilic ascites, a literature review was carried out on Pubmed.

Results: We identified three patients with EA suffering from EGIDs at our Centre. In the systematic literature review, a total of 105 patients suffering from EA due to EGIDs were identified from 81 literature articles. For both groups, we described: demographic characteristics, other immunological diseases, gastrointestinal symptoms, how ascites was diagnosed, ascitic fluid characteristics, peripheral eosinophils, gastrointestinal tract affected, therapy, and relapses.

Conclusions: EA is a rare presentation of EGIDs and proper evaluations should be performed to make a proper diagnosis; it is crucial to exclude clonal and secondary causes of hypereosinophilia, but differential diagnosis with single organ-HES is often unclear. Steroid therapy is effective and monoclonal antibodies directed against IL-5 could be as well, but further studies are needed.

Key words: #Eosinophils #Ascites #Interleukin-5 #HypereosinophilicSyndrome

Impact statement

We report our experience of three cases of eosinophilic ascites in patients suffering from eosinophilic gastrointestinal diseases together with a systematic review of all other cases described in the literature.

Introduction

Ascites is defined as accumulation of fluid in the peritoneal cavity. Cirrhosis is the main cause of ascites, and other possible aetiologies include malignancy, heart failure, tuberculosis, pancreatic disease, and other miscellaneous conditions (1). Eosinophilic ascites (EA) is characterised by a high eosinophil count in ascitic fluid and, although rare, is mainly caused by eosinophilic gastrointestinal diseases (EGIDs), parasitic infections, and hypereosinophilic syndrome (HES) (2). EGIDs encompass a group of immune-mediated disorders marked by eosinophil-predominant inflammation in specific gastrointestinal tracts, in the absence of secondary causes of eosinophilia, and are associated with characteristic symptoms. EGIDs are currently classified into eosinophilic esophagitis (EoE), eosinophilic gastritis (EoG), eosinophilic enteritis (EoN), further subclassified into eosinophilic duodenitis (EoD), eosinophilic jejunitis (EoJ), and eosinophilic ileitis (EoI), and eosinophilic colitis (EoC) (3). Symptoms caused by EGIDs depend on the affected gastrointestinal tract as well as the layer(s) involved; typically, EA implies a serosal involvement (4).

Herein, we report our single-centre experience of three cases of EA in patients suffering from EGIDs, along with a systematic review of previously reported cases in the literature.

Methods

Patients selection was performed by querying the database containing patients suffering from EGIDs referred to the Rare Gastrointestinal Disease Department of Fondazione Policlinico Universitario A. Gemelli IRCCS between January 2018 and January 2024 and extracting all cases with eosinophilic ascites.

In order to describe the reported cases of eosinophilic ascites, a literature review was carried out on PubMed (MEDLINE). Primary screening was

performed using the following MeSH headings and keywords: “eosinophilic ascites”, “eosinophilia AND ascites”, “eosinophilic gastrointestinal disease AND ascites”, “eosinophilic esophagitis AND ascites”, “eosinophilic gastritis AND ascites”, eosinophilic enteritis AND ascites”, “eosinophilic colitis AND ascites” from inception to 31 May 2024.

Inclusion criteria were as follows: full-text case reports, abstracts from which the necessary information could be inferred (abstracts were analysed due to the lack of articles about this argument), classical articles, letters, clinical studies, review articles and observational studies.

The exclusion criteria were duplicate publications, unavailability of full text, and articles not written in English, Spanish, German, or French.

A total of 1328 articles were selected. We then excluded articles with EA not associated with EGIDs (e.g. neoplasia, parasitosis, HES, eosinophilic granulomatosis with polyangiitis (EGPA), drugs, amyloidosis, and autoimmune diseases), articles in which the ascitic fluid was not analysed or with non-eosinophilic ascites, and articles in which a bioptic diagnosis of EGID was not made. The final selection resulted in a total of 81 articles.

With regard to the literature review, we considered a population of 105 patients with EA, then the sample was described in its clinical and demographic characteristics through descriptive statistics techniques. Qualitative variables have been presented as absolute frequencies and percentages and have been summarised as means and standard deviations. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) statistical software (Released 2017, IBM SPSS Statistics for Windows, Version 25.0; IBM Corp., Armonk, NY).

Results

Case series

Salient features of our patients are summarised in table I.

Case 1

Female, 34 years old. At the age of 31, due to acute onset of dysphagia, vomiting, and watery diarrhoea (up to 20 discharges per day), she was admitted to a local hospital where she underwent colonoscopy, oesophagogastroduodenoscopy, abdominal computed tomography (CT), and blood tests.

A diagnosis of eosinophilic duodenitis (>60 eosinophils/high power field) with EA and peripheral hypereosinophilia (5100 eosinophils/microliter – 41% of total white blood cells) was made.

She was then treated with intravenous methylprednisolone 50 mg for five days, which was tapered at home down to prednisone 5 mg twice a day in eight weeks (reducing it 5 mg per week). Since the same symptoms recurred 6 months later, the patient started therapy with sustained-release budesonide 9 mg/day for ten days; as she had no relief from this therapy, she tried a six-food elimination diet that lacked benefits. Therefore, on her own initiative, she took prednisone 5 mg twice a day. Three years later, she tried to stop steroid therapy but again experienced watery diarrhoea (up to 9 discharges per day). Then she was evaluated in our centre: her vital parameters were within the normal range, and her abdomen was batrachian in appearance, with a dull sound upon percussion of all abdominal quadrants. A subsequent abdominal CT scan confirmed the suspicion of ascites. A diagnostic paracentesis revealed the presence of numerous granulocytes (predominantly eosinophils) in the absence of cyto-nuclear atypia (bacterial cultures were negative). She subsequently underwent oesophagogastroduodenoscopy and colonoscopy with biopsies, extensive blood tests (thyroid, glycaemic, hepatic, renal, coagulative, and electrolyte profiles, serum electrophoresis, ANA, ENA, C-reactive protein, LDH, uric acid, total IgG, total IgM, total IgA, total IgE, autoantibodies for celiac disease and for autoimmune gastritis, basal serum tryptase, vitamin B12, serology for parasites, HIV, HCV, peripheral blood smear), urine and stool examinations, and bone marrow biopsy. Blood exams revealed moderate peripheral hypereosinophilia (4000 eosinophils/microliter - 34% of total white blood cells). Reactive and clonal causes of hypereosinophilia were ruled out, and a diagnosis of EA in a patient suffering from eosinophilic duodenitis was made. Given her young age and

the previous prolonged steroid therapy, the patient started mepolizumab 300 mg per month, allowing her to spare steroids, without presenting other relapses.

Case 2

Male, 62 years old, with a history of recurrent watery diarrhoea, abdominal pain, and mild food dysphagia for 16 years came to our centre for evaluation. His past medical history included ischaemic heart disease (two coronary stents), dyslipidaemia, Berger's disease, and surgery for abdominal hernia. His medical therapy included bisoprolol 2.5 mg/die, clopidogrel 75 mg/die, acetylsalicylic acid 100 mg/die, rosuvastatin/ezetimibe 5/10 mg/die. Oesophagogastroduodenoscopy and colonoscopy with biopsies diagnosed eosinophilic oesophagitis (>25 eosinophils/high power field) and eosinophilic duodenitis (>60 eosinophils/high power field). Blood count showed 7000 eosinophils/microliter (39.8% of total white blood cells), indicating severe hypereosinophilia. He also underwent an abdominal CT which revealed free abdominal effusion. Paracentesis showed a fluid with rare mesothelial cells, negative for neoplastic cells and infectious diseases, with an enormous prevalence of eosinophilic granulocytes. He also underwent bone marrow biopsy which showed no clonal aetiologies of hypereosinophilia. Other examinations (thyroid, glycaemic, hepatic, renal, coagulative, and electrolyte profiles, ANA, ENA, C-reactive protein, LDH, uric acid, IgG, IgM, IgA, autoantibodies for celiac disease, serum tryptase, vitamin B12, serology for parasites, HIV, HCV, peripheral blood smear, echocardiogram, and bone marrow needle aspiration) were negative. The patient underwent skin prick tests for aeroallergens and food which showed a pellitory sensitivity. A diagnosis of EA in a patient with eosinophilic oesophagitis and eosinophilic duodenitis was made. He then started therapy with esomeprazole 40 mg 2 tablets daily and sustained-release budesonide 9 mg/day. Subsequently, due to the absence of response to PPIs, we replaced esomeprazole with budesonide 1 mg 2 orodispersible tablets daily, with clinical benefits.

Case 3

Female, 48 years old, with a history of abdominal pain, emesis, and abdominal bloating that began at the age of 18 years. At that time, the symptoms regressed spontaneously. Six years later, due to recurrence of the same symptoms, she was admitted to a local hospital where she was diagnosed with eosinophilic colitis (>90 eosinophils/high power field in transverse and descending colon) and ascites (paracentesis wasn't performed at the time). Blood tests showed peripheral eosinophilia (2100 eosinophils/microliter - 28.1% of total white blood cells). The patient was then treated with steroids, with remission of her symptoms. At the age of 26 years, she was again admitted to hospital for recurrence of the same symptoms; a diagnostic paracentesis was performed which showed eosinophilic ascites. Peripheral eosinophilia was also observed. The patient was treated with intravenous methylprednisolone with relief, and she was discharged with deflazacort 6 mg/day per os, which was later replaced with budesonide 3 mg/day per os. The patient always experienced flare-ups of abdominal symptoms in the spring and summer months, which were treated with deflazacort 30 mg/day per os.

When the patient was first evaluated at our centre, she was asymptomatic for abdominal pain. Her bowel movements were regular. She did not complain of any other symptoms. Her medical therapy included omeprazole 20 mg/day, sustained-release budesonide 3 mg/day, and vitamin D supplementation. Her past medical history included appendectomy, breast augmentation, lactose intolerance, surgery for removal of a subcutaneous lipoma, previous Helicobacter pylori infection eradicated with antibiotic therapy. Abdominal ultrasound performed two months earlier showed no evidence of ascites. The tests requested subsequently showed a mild absolute-relative peripheral eosinophilia (500 eosinophils/microliter - 7.4% of total white blood cells), while thyroid, glycaemic, hepatic, renal, coagulative and electrolyte profils were normal, as well ANA, ENA, C-reactive protein, LDH, uric acid, IgG, IgM, IgA, autoantibodies for celiac disease serum tryptase, vitamin B12, serology for parasites (Strongyloides, Toxocara, Toxoplasma, Trichinella, Ascaris, Echinococcus, Scabies, Microfilaria), HIV, HCV, peripheral blood smear

and bone marrow biopsy. Subsequent colonoscopy (under steroid therapy) did not show the presence of eosinophils. Due to the stability of the eosinophilic colitis, she was advised to continue therapy with sustained-release budesonide 3 mg/day.

Literature review

A total of 105 patients suffering from EA due to EGIDs were identified from 81 literature articles. Their mean age was 29.64 (SD 13.87); 48 (45.7%) were male and 53 (50.47%) were female (in four patients, demographic data could not be traced back). With regard to their medical history, the most common diseases affecting their immune system were: asthma in 18 patients, food sensitization/allergy in 11, allergic rhinitis in 10, atopy/atopic dermatitis in 9, drug allergy in 5, 7 patients had a non-specified "allergy", 2 patients suffered from coeliac disease, 3 patients from chronic spontaneous urticaria and 37 patients had a negative medical history (in 20 patients this data was omitted or could not be traced back).
The mean blood eosinophils value was 6554.47/microliter (SD 5373.72 eosinophils/microliter); in six patients this data was not recorded or could not be traced back, in twelve patients was reported a non-specified "peripheral eosinophilia" and only 1 patient (0.95%) had no peripheral eosinophilia.
48 (45.71%) patients were diagnosed with eosinophilic gastroenteritis (EGE), three (2.86%) patients with EoE, five (4.76%) with EoG, two (1.90%) with EoN, 7 (6.66%) with EoD, one (0.95%) with EoJ, three (2.86%) with EoI and eight (7.62%) with EoC; fifteen (14.29%) with EoG + EoD, two (1.90%) with EoN + EoC, one (0.95%) with EoD + EoC, two (1.90%) with EoI + EoC, one (0.95%) with EoE + EoG, one (0.95%) with EoD + EoJ, one (0.95%) with EoD + EoI, one (0.95%) with EoE + EoG + EoN + EoC, 2 with EoG + EoD + EoI + EoC, 2 with EoE + EoG + EoD + EoC, 1 with EoE + EoG + EoD + EoJ, 1 with EoE + EoG + EoD.
73 patients (69.52%) were treated with steroids, one with steroids and total gastrectomy, one with steroids and clarithromycin, nine with steroids and an exclusion diet, three with steroids and antihistamines, two with steroids and montelukast, three with an exclusion diet, one with steroids and

sodium cromoglycate, one with azathioprine, one with benralizumab, one with Vitamin D; six patients had a spontaneous remission and in two patients the therapy performed was not reported.

In 60 patients (57.14%), the symptoms did not recur, while 14 patients experienced at least one recurrence of ascites (six patients had one relapse, one patient had two relapses, three patients had three relapses, and one patient had four relapses), and in 31 (29.52%), these data were not reported. Salient features of patients from Literature are summarised in table II.

Discussion and conclusions

The concept of eosinophilic gastroenteritis (EGE) was first described by Kaijser in 1937 (5), but only in 1970, Klein et al. proposed a classification based on the degree of eosinophilic infiltration: predominant mucosal, muscular, and subserosal layer disease; the latter is the least common and most associated with ascites (6). In 1990, Talley et al. defined EGE by three criteria: 1) the presence of gastrointestinal symptoms, 2) biopsies showing eosinophilic infiltration of one or more areas of the gastrointestinal tract from the oesophagus to the colon, or characteristic radiological findings with peripheral eosinophilia (such as a high eosinophil count in ascites fluid in serosal type), and 3) no evidence of parasitic or extraintestinal disease (4). The latest International Consensus for EGID nomenclature recommends using the term “eosinophilic gastrointestinal disease” (EGID) instead of “eosinophilic gastroenteritis” (EGE), and specifying the digestive tract involved (3). This terminology is crucial for two reasons: the number of eosinophils normally varies along the gastrointestinal tract (see later) and, depending on the tract concerned, the symptomatology will be different; unfortunately, in many articles we analysed, EGE was diagnosed regardless of the tract involved (classified as ‘EGE’ in table II).

EoE is by far the most common EGID, with a reported incidence between 2.1 and 12.8/100,000 per year in the Netherlands and Ohio, USA,

respectively, and a prevalence between 0.5 and 1 case per 1000, with a male predominance (7). The estimated prevalence of EoG, EGE, and EoC is 6.3/100,000, 8.4/100,000, and 3.3/100,000, respectively, with an almost equal male-female ratio (8,9).

Diagnosis of EoE requires oesophageal dysfunction symptoms, >15 eosinophils per high-power field (eos/hpf) on esophageal biopsy, and exclusion of other causes of esophageal eosinophilia (3). Atopic comorbidities, family history of EoE or dysphagia, and typical endoscopic findings (e.g., circular rings, linear furrows, exudates, oedema, strictures, narrowing, and crepe-paper mucosa) raise suspicion. Eosinophilic infiltration should be isolated to the oesophagus, with biopsies taken from upper, middle, and lower oesophageal mucosa (10). Non-EoE EGID should be suspected in patients complaining of typical symptoms (abdominal pain, nausea, vomiting, diarrhoea, weight loss, or ascites), usually associated with peripheral eosinophilia. As there is no unanimous consensus on the diagnostic criteria for non-EoE EGIDs, their diagnosis should take into account, as with EoE, typical symptoms, the exclusion of other causes, and of course the presence of eosinophils in the digestive tract; interestingly, the presence of eosinophilic ascites, even in the absence of eosinophils in the gastrointestinal tract, according to Talley's diagnostic criteria, falls within the EGID classification. The presence of eosinophils in the gastrointestinal tract is crucial for the diagnosis of EGIDs, and their presence should be demonstrable and quantifiable by an experienced pathologist.

Collins described EGIDs histopathologic features (11); it is interesting to note how the number of eosinophils/high power field varies along the digestive tract (at least 30 for EoG, more than 52 for EoD, more than 56 for EoI, more than 100 for right colon, more than 84 for transverse and descending colon, more than 64 for rectosigmoid colon) and that eosinophils are normally present in our digestive tracts, although the cut-off varies for other Authors. For this reason, even if Talley’s criteria did not require a histopathological diagnosis, many articles that classify a patient as suffering from EGID solely due to the presence of eosinophilic ascites, without excluding further causes, have been ruled out; indeed the mere presence of peripheral eosinophilia associated with eosinophilic ascites could not be sufficient to classify a patient as suffering from EGID,

reiterating the importance of histological diagnosis.

The pathophysiology of EGID remains poorly understood. EoE is a chronic, inflammatory, allergen-driven oesophageal disease, with a significant genetic predisposition, in which a damaged epithelial barrier and a dysregulated immune cell responses create a feed-forward loop, causing loss of immunologic tolerance to exogenous allergens and chronic eosinophilic inflammation (12, 13). The pathophysiology of non-EoE EGIDs remains unclear. Under the influence of IL-5, adhesion molecules, and eotaxin-5, eosinophils, after being formed in the bone marrow, migrate into the peripheral circulation and then localise to specific organs, mainly the gastrointestinal tract, thymus, haematopoietic organs, and, during puberty, mammary glands; thus, there is a constitutive resident eosinophilic population in the digestive tract (this is relevant, for example, to protect against helminthic infections) (14). Shoda et al. carried out molecular analyses of EoG and EoC, finding out an increased expression of eosinophilic chemokines; comparing the results with EoE, they found an overlap of T2 signature for EoG, but not for EoC (15,16). As shown by a recent Italian multicentre study, EoC seems to be associated with a personal history of atopy (17).

In many excluded articles, an analysis of the ascitic fluid was not carried out; interestingly, 74% of the EA analysed by Pinte et al. was associated with EGID (2). Cytological analysis of the ascitic fluid in order to exclude other causes (mainly infectious such as tuberculosis or neoplasms) is essential to make a correct diagnosis, and it should be performed both in patients with a known history of EGID who present with ascites, especially in those cases in which the first manifestation is EA.

Interestingly, we found three cases of EA arising during the puerperium (18-20), which could be related to the type-2 switch that occurs during pregnancy (21).

A correct diagnosis is also important to set up a correct therapy; our patients, and most patients from the Literature, were treated with steroid therapy. Although there are sustained-release forms with low systemic absorption, the risk of side effects remains high, especially in patients with

many relapses or those who are unable to discontinue therapy. Anti IL-5 therapies seem to be promising (22), as we noticed in our patient on mepolizumab therapy, since peripheral eosinophilia is a constant finding in EA in non-EoE EGIDs; this could also lead to classifying these patients as suffering from HES. Indeed, as shown by the latest diagnostic criteria proposed by Valent et al., the involvement of only one organ associated with increased peripheral eosinophils is sufficient to make a diagnosis (although the same authors recognise that EGIDs and other conditions in which only one organ is affected should be classified as reactive HES and that HES typically involves several organs) (23). We believe that it is more appropriate to include all conditions in which hypereosinophilia and eosinophilic involvement of a single organ is present under the term 'hypereosinophilia (HE) with single organ involvement', in accordance with Italian Guidelines (24). In addition to HES patients, those with EGPA were also excluded. While asthma and chronic rhinosinusitis are prevalent in these patients, their presence alone does allow the diagnosis of vasculitis. Most reviewed articles are case reports lacking follow-up information. Longitudinal follow-up is crucial, as we noted an EA recurrence coinciding with increased peripheral eosinophils in our patients. Furthermore, we noticed that in the patient suffering from wall-pellitory allergy, the gastrointestinal symptoms worsened during the pollen period.

In conclusion, EA is a rare EGID presentation requiring thorough evaluation for accurate diagnosis. Excluding clonal and secondary hypereosinophilia causes is critical, but differentiating from single-organ HES is often challenging. Steroid therapy is effective, and monoclonal antibodies targeting IL-5 seem to be promising, though further research is needed.

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Author contributions

DL: Conceptualization, Investigation, Methodology, Data curation, Formal Analysis, Project administration, Software, Writing – original draft. **IS, AA**: Supervision, Investigation, Data curation, Validation, Visualization, Writing – review & editing. **FF, FC, GI, FRP, CC, AB, GA**: Supervision, Validation, Visualization.

Conflict of interest statement

The authors declare not having any conflict of interest.

References

1. European Association for the Study of the Liver. EASL clinical practice guidelines on the management of ascites, spontaneous bacterial peritonitis, and hepatorenal syndrome in cirrhosis. J Hepatol. 2010 Sep;53(3):397-417. doi: 10.1016/j.jhep.2010.05.004.
2. Pinte L, Baicuş C. Causes of eosinophilic ascites - A systematic review. Rom J Intern Med. 2019 Jun 1;57(2):110-124. doi: 10.2478/rjim-2018-0041.
3. Dellon ES, Gonsalves N, Abonia JP, Alexander JA, Arva NC, Atkins D, et al. International Consensus Recommendations for Eosinophilic Gastrointestinal Disease Nomenclature. Clin Gastroenterol Hepatol. 2022 Nov;20(11):2474-2484.e3. doi: 10.1016/j.cgh.2022.02.017.
4. Talley NJ, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. Gut. 1990 Jan;31(1):54-8. doi: 10.1136/gut.31.1.54.

5. Kaijser R (1937) Allergic diseases of the gut from the point of view of the surgeon. Arch Klin Chir 188:36–64.
6. Klein NC, Hargrove RL, Sleisenger MH, Jeffries GH. Eosinophilic gastroenteritis. Medicine (Baltimore). 1970 Jul;49(4):299-319. doi: 10.1097/00005792-197007000-00003.
7. Dellon ES, Hirano I. Epidemiology and Natural History of Eosinophilic Esophagitis. Gastroenterology. 2018 Jan;154(2):319-332.e3. doi: 10.1053/j.gastro.2017.06.067.
8. Jensen ET, Martin CF, Kappelman MD, Dellon ES. Prevalence of Eosinophilic Gastritis, Gastroenteritis, and Colitis: Estimates From a National Administrative Database. J Pediatr Gastroenterol Nutr. 2016 Jan;62(1):36-42. doi: 10.1097/MPG.0000000000000865.
9. Pesek RD, Reed CC, Muir AB, Fulkerson PC, Menard-Katcher C, Falk GW, et al.; Consortium of Eosinophilic Gastrointestinal Disease Researchers (CEGIR). Increasing Rates of Diagnosis, Substantial Co-Occurrence, and Variable Treatment Patterns of Eosinophilic Gastritis, Gastroenteritis, and Colitis Based on 10-Year Data Across a Multicenter Consortium. Am J Gastroenterol. 2019 Jun;114(6):984-994. doi: 10.14309/ajg.0000000000000228.
10. Nielsen JA, Lager DJ, Lewin M, Rendon G, Roberts CA. The optimal number of biopsy fragments to establish a morphologic diagnosis of eosinophilic esophagitis. Am J Gastroenterol. 2014 Apr;109(4):515-20. doi: 10.1038/ajg.2013.463.
11. Collins MH. Histopathologic features of eosinophilic esophagitis and eosinophilic gastrointestinal diseases. Gastroenterol Clin North Am. 2014 Jun;43(2):257-68. doi: 10.1016/j.gtc.2014.02.007.
12. Allen-Brady K, Firszt R, Fang JC, Wong J, Smith KR, Peterson KA. Population-based familial aggregation of eosinophilic esophagitis suggests a genetic contribution. J Allergy Clin Immunol. 2017 Oct;140(4):1138-1143. doi: 10.1016/j.jaci.2016.12.979.

13. Underwood B, Troutman TD, Schwartz JT. Breaking down the complex pathophysiology of eosinophilic esophagitis. Ann Allergy Asthma Immunol. 2023 Jan;130(1):28-39. doi: 10.1016/j.anai.2022.10.026.
14. Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID). J Allergy Clin Immunol. 2004 Jan;113(1):11-28; quiz 29. doi: 10.1016/j.jaci.2003.10.047.
15. Shoda T, Wen T, Caldwell JM, Collins MH, Besse JA, Osswald GA, et al.; Consortium of Eosinophilic Gastrointestinal Disease Researchers (CEGIR). Molecular, endoscopic, histologic, and circulating biomarker-based diagnosis of eosinophilic gastritis: Multi-site study. J Allergy Clin Immunol. 2020 Jan;145(1):255-269. doi: 10.1016/j.jaci.2019.11.007.
16. Shoda T, Collins MH, Rochman M, Wen T, Caldwell JM, Mack LE, et al.; Consortium of Eosinophilic Gastrointestinal Diseases Researchers (CEGIR). Evaluating Eosinophilic Colitis as a Unique Disease Using Colonic Molecular Profiles: A Multi-Site Study. Gastroenterology. 2022 May;162(6):1635-1649. doi: 10.1053/j.gastro.2022.01.022.
17. Rossi CM, Lenti MV, Merli S, Lo Bello A, Mauro A, Anderloni A, Ribaldone DG, Marabotto E, Vernero M, Sheijani SD, Maniero D, Vanoli A, Klersy C, Savarino EV, Di Sabatino A. Clinical and atopic features of patients with primary eosinophilic colitis: an Italian multicentre study. Intern Emerg Med. 2024 Jun;19(4):993-1005. doi: 10.1007/s11739-024-03568-w. Epub 2024 Mar 10. PMID: 38461469; PMCID: PMC11186925.
18. Hepburn IS, Sridhar S, Schade RR. Eosinophilic ascites, an unusual presentation of eosinophilic gastroenteritis: A case report and review. World J Gastrointest Pathophysiol. 2010 Dec 15;1(5):166-70. doi: 10.4291/wjgp.v1.i5.166.
19. Milić S, Poropat G, Malić D, Stimac D. A case of postpartum eosinophilic gastroenteritis and review of the literature. Dig Dis. 2012;30(2):232-5. doi: 10.1159/000336711.

20. Gonçalves I, Pinho AFS, Antunes A, Carvalho S, Pinto L. Postpartum Eosinophilic Ascites: A Case Report. Cureus. 2022 Mar 18;14(3):e23301. doi: 10.7759/cureus.23301.
21. Mor G, Aldo P, Alvero AB. The unique immunological and microbial aspects of pregnancy. Nat Rev Immunol. 2017 Aug;17(8):469-482. doi: 10.1038/nri.2017.64.
22. Belfeki N, Ghriss N, Zayet S, El Hedhili F, Moini C, Lefevre G. Successful Introduction of Benralizumab for Eosinophilic Ascites. Biomedicines. 2024 Jan 6;12(1):117. doi: 10.3390/biomedicines12010117.
23. Valent P, Klion AD, Roufosse F, Simon D, Metzgeroth G, Leiferman KM, et al. Proposed refined diagnostic criteria and classification of eosinophil disorders and related syndromes. Allergy. 2023 Jan;78(1):47-59. doi: 10.1111/all.15544.
24. Caminati M, Brussino L, Carlucci M, Carlucci P, Carpagnano LF, Caruso C, et al. Managing Patients with Hypereosinophilic Syndrome: A Statement from the Italian Society of Allergy, Asthma, and Clinical Immunology (SIAAIC). Cells. 2024 Jul 11;13(14):1180. doi: 10.3390/cells13141180.
25. McNabb PC, Fleming CR, Higgins JA, Davis GL. Transmural eosinophilic gastroenteritis with ascites. Mayo Clin Proc. 1979 Feb;54(2):119-22.
26. Harmon WA, Helman CA. Eosinophilic gastroenteritis and ascites. J Clin Gastroenterol. 1981 Dec;3(4):371-3. doi: 10.1097/00004836-198112000-00013..
27. Kravis LP, South MA, Rosenlund ML. Eosinophilic gastroenteritis in the pediatric patient. Clin Pediatr (Phila). 1982 Dec;21(12):713-7. doi: 10.1177/000992288202101202.
28. Trounce JQ, Tanner MS. Eosinophilic gastroenteritis. Arch Dis Child. 1985 Dec;60(12):1186-8. doi: 10.1136/adc.60.12.1186.

29. Pilati G, Villecco AS, Allegro G. La gastroenterite eosinofila ad espressione esclusiva di ascite (ascite eosinofila). Presentazione di un caso e revisione della letteratura [Eosinophilic gastroenteritis simulating ascites (eosinophilic ascites). Presentation of a case and review of the literature]. Minerva Med. 1985 Dec 22;76(49-50):2319-21. Italian.
30. Jenss H, Plauth M, Gärtner HV. Eosinophile Gastroenteritis mit Aszites [Eosinophilic gastroenteritis with ascites]. Dtsch Med Wochenschr. 1986 Jun 20;111(25):984-7. German. doi: 10.1055/s-2008-1068569.
31. San José Díez J, González Hernández JM, Sánchez Palacios A, Velicia Llames R, Martín Escudero JC, Anllo F, et al. Eosinophilic gastroenteritis: a description of a new case with serous affectation. Allergol Immunopathol (Madr). 1987 Nov-Dec;15(6):383-7.
32. Solis-Herruzo JA, de Cuenca B, Muñoz-Yagüe MT. Laparoscopic findings in serosal eosinophilic gastroenteritis. Report of two cases. Endoscopy. 1988 Jul;20(4):152-3. doi: 10.1055/s-2007-1018162.
33. Tai YG, Liu JD, Lin KY, Chang JG, Wang CK, Siauw CP, et al. Eosinophilic gastroenteritis with eosinophilic ascites: report of a case. J Formos Med Assoc. 1990 Oct;89(10):901-4.
34. Ottobrelli A, Lagget M, Arrigoni A, Gindro T, Bosio C, Balbo G, et al. Gastroenterite eosinofila ed ascite. Caso clinico [Eosinophilic gastroenteritis and ascites. Clinical case]. Minerva Gastroenterol Dietol. 1991 Apr-Jun;37(2):131-4. Italian.
35. Wächter B, Jäger-Arand E, Engers R, Meyer zum Büschenfelde KH, Manns M. Eosinophile Gastroenteritis mit Serosabefall. Seltene Differentialdiagnose des Aszites [Eosinophilic gastroenteritis with serosa involvement. A rare differential diagnosis of ascites]. Z Gastroenterol. 1992 Jul;30(7):469-72. German.
36. Durieu I, Nove-Josserand R, Cathebras P, Durand DV, Rousset H, Levrat R. Ascites à éosinophiles. A propos de deux nouvelles observations [Eosinophilic ascites. 2 new case reports]. Rev Med Interne. 1992 Nov;13(6):446-8.

37. KURI, K., & LEE, M. (1994). Eosinophilic Gastroenteritis Manifesting With Ascites. Southern Medical Journal, 87(9), 956–957. doi: 10.1097/00007611-199409000-00021.

38. Salazar F, Salazar F. Gastroenteritis Eosinofilica [Eosinophilic gastroenteritis]. Rev Gastroenterol Peru. 1995 May-Aug;15(2):176-80. Spanish.

39. Santos J, Junquera F, de Torres I, Molero X, Vilaseca J, Malagelada JR. Eosinophilic gastroenteritis presenting as ascites and splenomegaly. Eur J Gastroenterol Hepatol. 1995 Jul;7(7):675-8.

40. Pfaffenbach B, Adamek RJ, Bethke B, Stolte M, Wegener M. Eosinophile Gastroenteritis bei Nahrungsmittelallergie [Eosinophilic gastroenteritis in food allergy]. Z Gastroenterol. 1996 Aug;34(8):490-3. German.

41. Tan AC, Kruimel JW, Naber TH. Eosinophilic gastroenteritis treated with non-enteric-coated budesonide tablets. Eur J Gastroenterol Hepatol. 2001 Apr;13(4):425-7. doi: 10.1097/00042737-200104000-00021.

42. Amirav I, Rosenthal E, Bentur L, Gleich GJ, Naveh Y. Coexistence of celiac disease and eosinophilic gastroenteropathy. J Pediatr Gastroenterol Nutr. 2001 Aug;33(2):200-1. doi: 10.1097/00005176-200108000-00020.

43. Hsu YQ, Lo CY. A case of eosinophilic gastroenteritis. Hong Kong Med J. 1998 Jun;4(2):226-228.

44. Kobayashi TK, Ueda M, Nishino T, Muramatsu M, Moritani S, Shigematsu T, et al. Eosinophilic ascites. Cytopathology. 2003 Apr;14(2):84-6. doi: 10.1046/j.1365-2303.2003.00029.x.

45. Fenoglio LM, Benedetti V, Rossi C, Anania A, Wulhfard K, Trapani M, et al. Eosinophilic gastroenteritis with ascites: a case report and review of the literature. Dig Dis Sci. 2003 May;48(5):1013-20. doi: 10.1023/a:1023080419660.

46. Barabino AV, Castellano E, Gandullia P, Torrente F, Guida A, Magnano GM. Chronic eosinophilic ascites in a very young child. Eur J Pediatr. 2003 Oct;162(10):666-8. doi: 10.1007/s00431-003-1279-7.

47. Bouhmidi A, Lorente Poyatos R, Romero Cara P, Ibáñez Martín JJ, Casado Caballero F, Quintero Fuientes D, et al. Enteritis eosinofílica como causa infrecuente de ascitis [Eosinophilic enteritis as a rare cause of ascites]. Gastroenterol Hepatol. 2003 Oct;26(8):480-1. doi: 10.1016/s0210-5705(03)70398-7.

48. Chen MJ, Chu CH, Lin SC, Shih SC, Wang TE. Eosinophilic gastroenteritis: clinical experience with 15 patients. World J Gastroenterol. 2003 Dec;9(12):2813-6. doi: 10.3748/wjg.v9.i12.2813.

49. Quack I, Sellin L, Buchner NJ, Theegarten D, Rump LC, Henning BF. Eosinophilic gastroenteritis in a young girl--long term remission under Montelukast. BMC Gastroenterol. 2005 Jul 18;5:24. doi: 10.1186/1471-230X-5-24.

50. Yassin MA, Khan FY, Al-Ani A, Fawzy Z, Al-Bozom IA. Ascites and eosinophilic colitis in a young patient. Saudi Med J. 2005 Dec;26(12):1983-5.

51. Méndez Sánchez IM, Rivera Irigoín R, Ubiña Aznar E, Vera Rivero FM, Fernández Pérez F, Navarro Jarabo JM, et al. Diferentes formas de presentación de una misma entidad clínica: gastroenteritis eosinofílica [Distinct clinical presentations of a single medical entity: eosinophilic enteritis]. Gastroenterol Hepatol. 2007 Jan;30(1):19-21. doi: 10.1157/13097445

52. Gallagher TK, Winter DC. Diarrhoea, ascites and eosinophilia: an unusual triad. Scand J Gastroenterol. 2007 Dec;42(12):1509-11. doi: 10.1080/00365520701435873.

53. Doggui MH, Ben Yaghlène L, Mohsni S, Mestiri A, Bouguassas W, Ksantini I. La gastroenterite avec ascite a eosinophile: a propos d'un cas [Eosinophilic gastroenteritis with ascites: a case report]. Tunis Med. 2008 Jun;86(6):600-3. French.

54. Sánchez AG, Berjano CC, Vallejos PL, Rodríguez GV, Herrera GR, Díez NR, et al. Gastroenteritis eosinofílica con manifestación inusual [Eosinophilic gastroenteritis with atypical manifestation]. Gastroenterol Hepatol. 2009 May;32(5):384-6. Spanish. doi: 10.1016/j.gastrohep.2008.12.009.

55. Shiraishi E, Yasunaga Y, Nagai K, Matsuura N, Yamai T, Ikezoe M, et al. A case of eosinophilic gastroenteritis with ascites and protein-losing gastroenteropathy. Nihon Shokakibyo Gakkai Zasshi. 2009 Nov;106(11):1625-35.

56. Cha JM, Lee JI, Joo KR, Shin HP. Eosinophilic gastroenteritis with eosinophilic dermatitis. Yonsei Med J. 2010 Jan;51(1):145-7. doi: 10.3349/ymj.2010.51.1.145.

57. Setia N, Ghobrial P, Liron P. Eosinophilic ascites due to severe eosinophilic ileitis. Cytojournal. 2010 Sep 17;7:19. doi: 10.4103/1742-6413.70408.

58. Jarry J, Peycru T, Shekher M. A rare cause of ascites. Gastroenterology. 2011 Apr;140(4):1149, 1364. doi: 10.1053/j.gastro.2010.05.095.

59. Lim KC, Tan HK, Rajnakova A, Venkatesh SK. Eosinophilic gastroenteritis presenting with duodenal obstruction and ascites. Ann Acad Med Singap. 2011 Aug;40(8):379-81.

60. Bleibel F, Fragoza K, Faller GT. Acute eosinophilic ascites in a middle-aged man. Case Rep Gastrointest Med. 2012;2012:896523. doi: 10.1155/2012/896523.

61. Liao WH, Wei KL, Po-Yen-Lin, Wu CS. A rare case of spontaneous resolution of eosinophilic ascites in a patient with primary eosinophilic gastroenteritis. Chang Gung Med J. 2012 Jul-Aug;35(4):354-9. doi: 10.4103/2319-4170.106134.

62. Antonini F, Saltarelli P, Frieri G, Latella G. Education and Imaging: gastrointestinal: eosinophilic ascites. J Gastroenterol Hepatol. 2012 Nov;27(11):1759. doi: 10.1111/j.1440-1746.2012.07254.x.

63. Ohe M, Hashino S. Successful treatment of eosinophilic gastroenteritis with clarithromycin. Korean J Intern Med. 2012 Dec;27(4):451-4. doi: 10.3904/kjim.2012.27.4.451.

64. Teng X, Xu L, Wu J, Sun M, Guo J, Mao Z. Clinical and morphological features of serosal form of eosinophilic gastroenteritis in a retrospective study of 10 children. Fetal Pediatr Pathol. 2013 Jul;32(4):276-83. doi: 10.3109/15513815.2012.754524.

65. Lim DN, Wilkins A, Horton LE, Ahmad I, Lo Polito C, Phillips C. Eosinophilic gastrointestinal disorder in coeliac disease: a case report and review. Case Rep Gastrointest Med. 2012;2012:124275. doi: 10.1155/2012/124275.

66. Salkić NN, Mustedanagić-Mujanović J, Jovanović P, Alibegović E. Enhanced therapeutic response with addition of loratadine in subserosal eosinophilic gastroenteritis. Med Glas (Zenica). 2013 Feb;10(1):178-82.

67. Salgueiro P, Magalhães R, Lago P. Cramping pain and eosinophilic ascites: what is the diagnosis? Gastroenterology. 2013 Jun;144(7):1353, 1577. doi: 10.1053/j.gastro.2013.01.008.

68. Taş A, Çelık H. An unusual cause of ascites in a young patient. Turk J Gastroenterol. 2013;24(1):79-80.

69. Liu L, Liang XY, He H, Huang H, Jia LP. Clinical features of eosinophilic gastroenteritis with ascites. Z Gastroenterol. 2013 Jul;51(7):638-42. doi: 10.1055/s-0033-1335326.

70. Elliott JA, McCormack O, Tchrakian N, Conlon N, Ryan CE, Lim KT, et al. Eosinophilic ascites with marked peripheral eosinophilia: a diagnostic challenge. Eur J Gastroenterol Hepatol. 2014 Apr;26(4):478-84. doi: 10.1097/MEG.0000000000000037.

71. Alhmoud T, Arif H, Auyang E, Samedi V, Parasher G. Chronic abdominal pain, ascites, and diarrhea: seeing red. Dig Dis Sci. 2014 Apr;59(4):740-3. doi: 10.1007/s10620-014-3095-5.

72. Maleki N, Kalantar Hormozi M, Bahtouee M, Tavosi Z, Mosallai Pour H, Taghiyan Jamaleddin Kolaii SS. Eosinophilic ascites and duodenal obstruction in a patient with liver cirrhosis. Case Rep Gastrointest Med. 2014;2014:928496. doi: 10.1155/2014/928496.

73. Saraiva N, Filipe C, Simão A, Carvalho A. A 22-year-old male patient with ascites. BMJ Case Rep. 2015 Jun 8;2015:bcr2014209265. doi: 10.1136/bcr-2014-209265.

74. Ming G, Bo Y, Li-Ping Y. Eosinophilic gastroenteritis with ascites in a child. Indian Pediatr. 2015 Aug;52(8):707-8. doi: 10.1007/s13312-015-0703-1.

75. Alsulaiman RM. Eosinophilic ascites: A case report and literature review. J Family Community Med. 2015 Sep-Dec;22(3):183-5. doi: 10.4103/2230-8229.163042.

76. Khalil H, Joseph M. Eosinophilic ascites: a diagnostic challenge. BMJ Case Rep. 2016 Sep 6;2016:bcr2016216791. doi: 10.1136/bcr-2016-216791.

77. Agrawal S, Vohra S, Rawat S, Kashyap V. Eosinophilic ascites: A diagnostic and therapeutic challenge. World J Gastrointest Surg. 2016 Sep 27;8(9):656-659. doi: 10.4240/wjgs.v8.i9.656.

78. Nehme F, Kisang G, Green M, Tofteland N. Abdominal Pain and Ascites: Not Always Related to Portal Hypertension. Cureus. 2016 Oct 3;8(10):e812. doi: 10.7759/cureus.812. doi: 10.7759/cureus.812.

79. Martín-Lagos Maldonado A, Alcazar Jaén LM, Benavente Fernández A. Eosinophilic ascites: A case report. Gastroenterol Hepatol. 2018 Jun-Jul;41(6):372-374. doi: 10.1016/j.gastrohep.2017.08.003.

80. Lourenço LC, Horta DV, Reis J. Eosinophilic Ascites: Uncommon Presentation of Eosinophilic Gastroenteritis in a Young Adult Male. GE Port J Gastroenterol. 2017 Jul;24(4):206-208. doi: 10.1159/000453363.

81. Santos C, Morgado F, Blanco C, Parreira J, Costa J, Rodrigues L, et al. Ascites in a Young Woman: A Rare Presentation of Eosinophilic Gastroenteritis. Case Rep Gastrointest Med. 2018 May 10;2018:1586915. doi: 10.1155/2018/1586915.

82. Lopes Azevedo RJ, Pinto J, Ribeiro H, Pereira F, Leitão C, Caldeira A, et al. Eosinophilic Enterocolitis: An Exceedingly Rare Entity. GE Port J Gastroenterol. 2018 Jun;25(4):184-188. doi: 10.1159/000481915.

83. Letrán A, Hueso S, González V, Ramos C, Amaya A. Eosinophilic Ascites: Singular Presentation of Eosinophilic Gastrointestinal Disorder. J Investig Allergol Clin Immunol. 2018 Aug;28(4):275-276.

84. Shi L, Jia QH, Liu FJ, Guan H, Jiang ZY. Massive hemorrhagic ascites: A rare presentation of eosinophilic gastroenteritis. World J Clin Cases. 2018 Jul 16;6(7):156-160. doi: 10.18176/jiaci.0260.

85. Salah HT, Al-Hussaini HF, Alqaraawi AM, Alanazi KM. A Case of Eosinophilic Gastroenteritis Associated with Eosinophilic Ascites Diagnosed by Full-Thickness Biopsy of the Small Intestine. Am J Case Rep. 2019 Feb 13;20:189-193. doi: 10.12659/AJCR.913319.

86. Šaban J, Milošević V. A rare case of eosinophilic esophagitis and eosinophilic subserosal gastroenteritis with ascites. Turk J Gastroenterol. 2019 Sep;30(9):851-853. doi: 10.5152/tjg.2019.18680.

87. Kim MJ, Oh MJ. Eosinophilic Enteritis Presenting as Massive Ascites after Influenza A Virus Infection in a Young Female. Korean J Gastroenterol. 2019 Sep 25;74(3):163-167. doi: 10.4166/kjg.2019.74.3.163.

88. Feng W, Zheng K, Shen H. Eosinophilic ascites: an unusual manifestation of eosinophilic gastroenteritis. Int J Colorectal Dis. 2020 Apr;35(4):765-767. doi: 10.1007/s00384-020-03510-4.

89. Pereira F, Ferreira AI, Linhares M, Paulo S, Tristan J, Pereira E, et al. A rare case of diarrhea and ascites. Int J Colorectal Dis. 2020 Apr;35(4):769-773. doi: 10.1007/s00384-020-03523-z.

90. Liang M, Liwen Z, Bingfang C, Yanbo D, Jianping C. Eosinophilic gastroenteritis with multiple serous membrane effusion as the first sign: a case report and literature review. J Int Med Res. 2020 Apr;48(4):300060520917274. doi: 10.1177/0300060520917274.
91. Fonseka CL, Nanayakkara S, Singhapura SDAL, Herath HMM, Bodinayake CK. Eosinophilic Gastroenteritis with a Relapsing and Remitting Course with Presence of Autoimmune Antibodies. Case Rep Med. 2020 Aug 18;2020:1745834. doi: 10.1155/2020/1745834.
92. Huang X, Liao X, Xiao Z, Huang Z. Halo sign and araneid limb-like sign in eosinophilic enteritis. Lancet Gastroenterol Hepatol. 2020 Oct;5(10):954. doi: 10.1016/S2468-1253(20)30115-1.
93. Qua CS, Peh KB, Saravannan K, Goh KL. Vitamin D deficiency causing eosinophilic esophagogastroenteritis and ascites: a rare association. BMJ Case Rep. 2021 Feb 4;14(2):e240039. doi: 10.1136/bcr-2020-240039.
94. López-Sáez MP, Magdalena Bethencourt K, Muñoz Tornero M, Alajarin Cervera M, Serrano Santos E, Sánchez-Guerrero Villajos I, et al. Ascites as a Rare Presentation of Eosinophilic Enteritis in a Pollinic Patient. J Investig Allergol Clin Immunol. 2021 Feb 21;32(1):56-58. doi: 10.18176/jiaci.0697.
95. Amado C, Silva Leal M, Neto P, Ferreira G. Subserous Type of Eosinophilic Colitis: A Rare Disease. Eur J Case Rep Intern Med. 2021 Jul 2;8(7):002671. doi: 10.12890/2021_002671.
96. Yang K, Luo RH, Sun YL. Recurrent abdominal pain, peritoneal effusion, and eosinophilia in a boy aged 17 years. Zhongguo Dang Dai Er Ke Za Zhi. 2021 Nov 15;23(11):1169-1173. doi: 10.7499/j.issn.1008-8830.2107109.
97. He YJ, Sun JY, Wang P, Wang JK, Li B, Yu XH. Plasma membrane lesion type total intestinal eosinophilic enteritis: a case report. Transpl Immunol. 2022 Apr;71:101547. He YJ, Sun JY, Wang P, Wang JK, Li B, Yu XH. Plasma membrane lesion type total intestinal eosinophilic enteritis: a case report. Transpl Immunol. 2022 Apr;71:101547.

98. Kamath SD, Rana F, Kumar K, Sunder A. An Unusual Case of Eosinophilic Ascites with Pleural Effusion - A Rare Manifestation of Eosinophilic Gastroenteritis (EGE). Niger Med J. 2022 Apr 28;62(4):208-211. doi: 10.60787/NMJ-62-4-38.

99. Galere P, Gilbert A. Eosinophilic gastroenteritis with ascites: an enigmatic diagnosis. Lancet. 2022 Jul 9;400(10346):126. doi: 10.1016/S0140-6736(22)01056-X.

100. Silva Mendes S, Gonçalves B, Soares JB, Caetano AC, Gonçalves R. Eosinophilic Gastroenteritis: Still a Diagnostic Challenge. GE Port J Gastroenterol. 2022 Sep 6;30(Suppl 2):52-56. doi: 10.1159/000525809.

Table I. Main characteristics of our patients.

Age	**Sex**	**Other immunological diseases**	**Gastrointestinal symptoms**	**Ascites diagnosis**	**EGIDs**	**Ascitic fluid analysis**	**Peak peripheral eosinophils (cells/mcl - % of total white blood cells)**	**Therapy**	**Relapses**
34	F	No	Dysphagia, vomiting and watery diarrhea	Abdominal US and abdominal CT	EoD	Numerous granulocytes (predominantly eosinophils) in the absence of cyto-nuclear atypia (also negative for	5100/mcl - 41%	Mepolizumab 300 mg per month	Yes

						bacterial cultures)			
62	M	Wall pellitory allergy	Watery diarrhea, abdominal pain and mild food dysphagia	Abdominal CT	Eo E + Eo D	Fluid with rare mesothelial cells, negative for neoplastic cells and infectious diseases, with an enormous prevalence of	7000/mcl - 39.8%	Orodispersible budesonide 1 mg 2 tablets/day PO + sustained-release budesonide 9 mg/day PO	Yes

						eosinophilic granulocytes			
48	F	No	Abdominal pain, emesis and abdominal bloating	Abdominal US	Eo C	Negative for neoplastic cells and infectious disease; numerous granulocytes (mostly eosinophils)	2100/mcl - 28.1%	Sustained-release budesonide 3 mg/day PO	Yes

F = female; M = male; US = ultrasound; CT = computed tomography.

Table II. Features of patients from the retrieved studies.

Author	Age	Sex	Other immunological diseases	Gastrointestinal symptoms	Ascites Diagnosis	EGIDs	Salient features of ascitic fluid analysis reported by the Authors	Peak peripheral eosinophils (eos/mcl - % of total white blood cells)	Therapy	Relapses
McNabb PC et al. (25)		/	Atopy	Recurrent abdominal pain, diarrhoea	/	EGE	Eosinophilia	/	CCS	/
Harmon WA et al. (26)	53	M	Atopy	Epigastric burning, cramping abdominal pain	/	EGE	99% eosinophilic cells	4420/mcl	CCS	/
Kravis LP et	2	F	/	Abdominal pain, nausea, vomiting	/	EGE	Eosinophilia	27000/mcl	Intermittent prednisone	/

al. (27)										
Trounce JQ et al. (28)	9	M	No	Abdominal pain, distension, diarrhea	Abdominal US	EGE	Bloody, rich in eosinophils and protein (44 g/l), sterile on bacterial, tuberculous, and fungal culture	13900/mcl	Prednisolone 2 mg/kg/day on induction, prednisolone 5 mg on alternate days on maintenance	Yes
Pilati G et al. (29)	/	/	/	/	/	EGE	Eosinophilia	Peripheral eosinophilia	Spontaneous remission	Yes
Jenss et al. (30)	28	F	Asthma	Abdominal pain, diarrhoea	/	EGE	Eosinophilia	/	Spontaneous remission	2

San José Díez J et al. (31)	51	F	/	Periodic episodes of abdominal pain, nausea and vomiting	/	EGE	Eosinophilia	Peripheral eosinophilia	/	/
Solis-Herruzo JA et al. (32)	41	F	Asthma, CSU, multiple drugs allergy	Recurrent abdominal pain	Abdominal CT and US, laparoscopy	EGE	Protein 4.4 g/dL, 2400 white blood cells (WBCs)/ml (70% eosinophils), sterile on bacterial and tubercule culture	5202 /mcl - 44.46%	Prednisolone 1 mg/kg	No
Solis-Herruz	22	F	No	Abdominal pain	Laparoscopy	EGE	Protein 4.3 g/dl, 4560/ml	11300 /mcl - 46.50%	Prednisolone	/

o JA et al. (32)							WBCs with 83% eosinophils, sterile on bacterial and tubercule culture				
Talley NJ et al. (4) (5 patients)	42 (mean)	4 M, 1 F	4 had atopy	4 had abdominal pain, 2 nausea/vomiting, 3 bloating, 3 diarrhea, 1 steatorrhea	/	EGE	Eosinophilic ascites	8413/mcl (mean)	CCS		/
Tay YG et al. (33)	30	M	Asthma, rhinitis, CSU	Abdominal pain, diarrhea	Abdomen CT	EGE	Exudative ascites rich in eosinophils	Peripheral eosinophilia	CCS		/

Ottobrelli A et al. (34)	/	/	/	30 years history of sub-occlusive episodes and diarrhea	Abdomen CT	EGE	Eosinophils	Peripheral eosinophilia	CCS		
Wächter B et al. (35)	21	M	/	14 days from cramping abdominal pain, associated with nausea and vomiting	Laparoscopy	EGE	Marked eosinophilia in protein rich ascitic fluid	Peripheral eosinophilia	Low dose prednisolone therapy	/	
Durieu I et al. (35)	50	M	Asthma		Laparoscopy	EGE	Exudate rich in eosinophils (1900/ml)	/	Spontaneous remission	/	
Durieu I et al. (36)	50	F	/	Diarrhea	Abdominal US	EGE	Exudate rich in eosinophils (3800/ml)	10000/mcl	Spontaneous remission	/	

Kuri K et al. (37)	41	F	Asthma, Atopic dermatitis	Nausea, vomiting, constipation, abdominal pain and distention	Abdominal CT	EGE	Protein 5.1 g/dl, WBCs 17900/ml (94% eosinophils), negative culture	1615,5/mcl - 45%	CCS	No
Salazar F et al. (38)	/	/	/	Gastric outlet obstruction	/	EGE	Eosinophilia	Peripheral eosinophilia	Total gastrectomy + CCS	Yes
Santos J et al. (39)	25	M	/	1 year history of episodic abdominal pain	/	EGE	Eosinophilic ascites	Peripheral eosinophilia	CCS	No
Pfaffenbach B et	23	F	Food allergies	Abdominal pain, nausea and diarrhea	CT	EGE	Exudative ascites with eosinophils	Peripheral eosinophilia	Elimination diet	No

al. (40)										
Tan AC et al. (41)	32	F	No	Abdominal pain, nausea, diarrhea, weight loss	Abdominal US	EGE	Eosinophils	20010/mcl	Prednisone, then budesonide as long-term maintenance	No
Amiral I et al. (42)	14	M	Asthma, celiac disease	Diarrhea	Paracentesis	EGE	Eosinophils	No	CCS	/
Hsu YQ et al. (43)	34	F	No	Abdominal distension, frequency of bowel motion, and tenesmus	Abdomen CT, laparotomy	EGE	Abundant WBCs counts which were predominantly eosinophils. Cytology, culture, and smear tests for acid-fast	22200 /mcl - 47%	Prednisolone 10 mg	No

							bacilli in the fluid gave negative results			
Kobayashi TK et al. (44)	24	F	No	Recurrent abdominal pain	abdomen CT	EoG	80% eosinophils (many mature eosinophils, some with nuclear hypersegmentation)	819/mcl - 6.5%	Methylprednisolone 75 mg iv (then tapered)	No
Fenoglio LM et al. (45)	29	F	No	Abdominal swelling, body weight increase, and diarrhea	Paracentesis	EGE	Exudative effusion with normal LDH, triglycerides, and amylase; WBCs were	8730/mcl	Methylprednisolone 50 mg iv (then tapered with deflazacort 6 mg/die)	3

							5800/ml with more than 90% eosinophils; cultures for aerobic and anaerobic bacteria were negative and no atypical cells were found			
Barabino AV et al. (46)	2	M	No	Pallor, fatigue	Abdominal US, paracentesis	EoG + EoD	5000 WBCs/ml with 32% eosinophils. C-reactive protein,	820/mcl	Oral prednisone (2 mg/kg per day) + multiple therapeutic paracentesis + spironolactone	No

							cultures, parasites, Koch bacillus and neoplastic cells were all normal or negative.				
Bouhmidi A et al. (47)	18	F	NO	Abdominal distention, fatigue, loss of weight	Abdominal CT, laparotomy	EGE	Eosinophilic ascites; neoplastic and infectious causes were excluded	1190/mcl - 14%	CCS		No
Chen MJ et al. (48)	17	M	Allergy	Abdominal pain, diarrhea, bloating, nausea,	Abdominal CT and US	EGE	Eosinophilic ascites	11016/mcl	CCS		1

				vomiting, hypoalbuminemia, fecal blood loss							
Chen MJ et al. (48)	38	F	Allergy	Abdominal pain, diarrhea, bloating, nausea, vomiting, hypoalbuminemia, fecal blood loss	Abdominal CT and US	EGE	Eosinophilic ascites	1008/mcl	CCS		No
Chen MJ et al. (48)	20	F	Allergy	Abdominal pain, diarrhea, bloating, nausea, vomiting, hypoalbuminem	Laparotomy	EGE	Eosinophilic ascites	3984/mcl	CCS		3

				ia, fecal blood loss							
Chen MJ et al. (48)	35	M	No	Abdominal pain, diarrhea, bloating, nausea, vomiting, hypoalbuminemia, fecal blood loss	Abdominal CT and US	EGE	Eosinophilic ascites	621/mcl	CCS		No
Chen MJ et al. (48)	53	M	No	Abdominal pain, diarrhea, bloating, nausea, vomiting, hypoalbuminem	Abdominal CT and US	EGE	Eosinophilic ascites	14500/mcl	CCS		No

				ia, fecal blood loss						
Chen MJ et al. (48)	3	F	Allergy	Abdominal pain, diarrhea, bloating, nausea, vomiting, hypoalbuminemia, fecal blood loss	Abdominal CT and US	EGE	Eosinophilic ascites	264/mcl	CCS	4
Quack I et al. (49)	17	F	Allergic rhinitis	Recurrent abdominal cramps, nausea, vomiting	Laparoscopy, abdominal US and CT	EoE + EoG + EoN + EoC	Eosinophilic ascites	7714/mcl - 58%	40 mg prednisone/day, then montelukast 10 mg/day	No

Yassin MA et al. (50)	32	M	/	Abdominal pain and distention	/	EoC	Eosinophilic ascites	Peripheral eosinophilia	Spontaneous remission	/
Méndez Sánchez IM et al. (51)	45	F	Asthma	Abdominal pain	Abdominal US and CT	EoN + EoC	Negative for neoplastic cells and infectious disease; numerous granulocytes (mostly eosinophils)	4690/mcl	CCS, then CCS + azathioprine	1
Gallagher TK et al. (52)	37	F	No	Diarrhea	Abdominal US and CT	EoD + EoC	Abundant macrophages with a few lymphocytes	5700/mcl	CCS, then disodium cromoglycate	No

							and reactive mesothelial cells. No malignant cells were seen. Ziehl-Neelsen staining was negative.				
Doggui MH et al. (53)	65	M	Allergic asthma	Diarrhea, vomiting and loss of weight	Paracentesis	EoD	Exudative effusion with high level of eosinophils	Peripheral eosinophilia	CCS		/
Sánchez AG et al. (54)	28	M	No	Vomiting and diarrhea	Abdominal US and CT, paracentesis	EoD	85% of total white blood cells were eosinophils; negative for	5300/mcl	CCS		No

							cytological abnormalities and for infectious diseases.			
Shiraishi E et al. (55)	24	M	/	Abdominal distension, diarrhea, and nausea	Abdominal US and CT	EGE	Massive ascites (WBCs 11500/ml with 95% eosinophils)	5184/mcl	CCS	/
Cha JM et al. (56)	24	F	No	Abdominal pain and vomiting	Laparotomy	EoG + EoD	Exudative ascites with predominant eosinophils (WBCs 3150/ml with 75%	1729/mcl	Prednisolone 40 mg/day	No

							eosinophils) and no evidence of malignant cells.				
Setia N et al. (57)	17	M	Asthma	Intermittent abdominal pain, nausea, bilious emesis, bloody diarrhea	Abdominal US and CT	EoI	Fluid analysis was remarkable for 65% eosinophils. The peritoneal fluid revealed an abundance of mature eosinophils present in a bloody	3600/mcl - 31%	CCS		No

							background. Malignant cells or microorganisms were not identified. Microbiology cultures of the ascitic fluid were negative for bacteria, mycobacteria, and fungal organisms.				
Jarry J et al. (58)	40	F	Asthma	Abdominal pain and vomiting	Abdominal CT	EGE	Sterile exudative peritoneal effusion with	3672/mcl - 34%	/		/

							up to 95% eosinophils					
Hepburn IS et al. (18)	20	W	No	Nausea, non bloody vomiting, diarrhea, abdominal pain, distention, leg edema, weight gain	Abdominal US	EGE	Hazy dark fluid with no cytological signs of malignancy, with protein level 4.5 g/dl, albumin 2.4 g/dl, WBCs 1780/ml with significant eosinophilia of 82%. Ascitic fluid for bacterial	4864/mcl - 38%	CCS			No

							culture and for tuberculosis had no growth.			
Lim KC et al. (59)	31	M	No	Epigastric discomfort, vomiting and diarrhea	Abdominal CT	EoD	Sterile exudative ascites with predominant eosinophils. There was no evidence of malignant cells.	12510/mcl - 51.9%	Oral prednisolone (30 mg daily)	No
Bleibel F et al. (60)	55	M	No	Abdominal pain and distention	Abdominal US and CT	EoJ	WBCs count of 6600/mL, 95% of which were	12141/mcl - 71%	Oral prednisone (20mg/day)	No

							eosinophils, LDH 284 mg/dl, albumin 3.2 g/dl (simultaneous serum albumin 4.1 g/dl)			
Milić S et al. (19)	30	F	Chronic rhinosinusitis, asthma	Epigastric pain, vomiting, diarrhea	Paracentesis	EoE + EoG + EoN + EoC	Exudative ascites with high protein amount (39 g/l), and a high cell count consisting predominantl	1344/mcl - 12%	40 mg of prednisone and 10 mg montelukast daily	No

							y of eosinophils (up to 40%)			
Liao WH et al. (61)	43	M	No	Intermittent crampy epigastralgia	Abdominal US and CT	EoC	WBCs were 1700/ml with 99% eosinophils. There were no malignant cells on cytological examination of the ascites. The gram stain and culture of the ascites were negative.	5390/mcl - 49%	Spontaneous remission	No

Antoni ni F et al. (62)	29	F	Drug allergy	Diarrhea, nausea, vomiting	Abdominal US and CT	EoG + EoD + EoI + EoC	Sterile with > 90% of eosinophils	4950/mcl - 43%	Prednisone 25 mg/day per os	No
Ohe M et al. (63)	52	M	Food allergens sensitization (no history of food allergies)	Abdominal pain, abdominal distension, and diarrhea	Abdomen CT	EoI + EoC	Abundance of mature eosinophils against a bloody background; no malignant cells or microorganisms were identified.	8674/mcl - 62%	Prednisolone 40 mg/die (tapered to 20 mg/die, then to 3 mg/die), then prednisolone 20 mg/die (tapered to 5 mg/die), then prednisolone 10 mg/day + clarithromycin (CAM) 400 mg/die, then only CAM 400 mg/die, then prednisolone 5 mg/day + CAM 800 mg/day, then prednisolone 4 mg/day + CAM 600 mg/day	3

Teng X et al. (64)	13	M	Egg allergy	Abdominal pain	Abdominal US	EoG + EoD	62% eosinophils in ascitic fluid	5490/mcl - 46.9%	CCS	/
Teng X et al. (64)	13	F	Corn/cod allergy	Abdominal pain	Abdominal US	EoG + EoD	89% eosinophils in ascitic fluid	8310/mcl - 55.4%	CCS	/
Teng X et al. (64)	8	F	Egg allergy	Abdominal pain, vomiting	Abdominal US	EoG + EoD	78% eosinophils in ascitic fluid	25580/mcl - 67.5%	CCS	/
Teng X et al. (64)	13	M	Egg allergy	Abdominal pain, diarrhea	Abdominal US	EoG + EoD	72% eosinophils in ascitic fluid	8960/mcl - 48.7%	CCS	/
Teng X et al. (64)	8	M	Corn/cod/egg allergy	Abdominal pain, diarrhea	Abdominal US	EoG + EoD	96% eosinophils in ascitic fluid	3980/mcl - 28.2%	CCS	/

Teng X et al. (64)	12	F	Milk/egg/tomato allergy	Abdominal pain, diarrhea, vomiting	Abdominal US	EoG + EoD	68% eosinophils in ascitic fluid	2800/mcl - 30.1%	CCS	/
Teng X et al. (64)	13	F	No	Abdominal pain	Abdominal US	EoG + EoD	86% eosinophils in ascitic fluid	850/mcl - 13.7%	CCS	/
Teng X et al. (64)	8	M	No	Abdominal pain	Abdominal US	EoG + EoD	90% eosinophils in ascitic fluid	730/mcl - 9.4%	CCS	/
Teng X et al. (64)	13	M	No	Abdominal pain	Abdominal US	EoG + EoD	84% eosinophils in ascitic fluid	590/mcl - 6.6%	CCS	/
Teng X et al. (64)	7	F	No	Abdominal pain	Abdominal US	EoG + EoD	79% eosinophils in ascitic fluid	1040/mcl - 6.9%	CCS	/
Lim DN et al. (65)	30	F	Coeliac disease	Abdominal pain, distension, vomiting	Abdominal CT	EoD + EoJ	Total protein 4.5 g/dL, albumin 2.4	860/mcl	CCS	No

						g/dl, serum to ascites albumin gradient (SAAG) 0.1 g/dl, eosinophil count 6030/ml. No evidence of malignant or lymphoma cells				
Salkić NN et al. (66)	45	M	/	Abdominal obstruction	/	EGE	Eosinophilic ascites	Peripheral eosinophilia	Parenteral methylprednisolone and oral loratadine	/

Salguero P et al. (67)	37	F	/	Abdominal pain, increased abdominal size	Abdominal CT	EoE	Exudate with marked eosinophilia without signs of malignancy (WBCs 6988/ml; eosinophils 6777/ml; total protein 4.8 g/dl; albumin 3.42 g/dl; no malignant cells on cytologic analysis)	2330/mcl - 13.7%	CCS, then CCS + ketotifen	1

Taş A et al. (68)	24	M	No	Abdominal distension	Abdominal US and CT	EoC	45% eosinophils. SAAG was < 1.1 g/dl. Microbiology cultures of the ascitic fluid were negative for bacteria, mycobacteria, and fungal organisms.	1575/mcl - 15%	Budesonide 9 mg	No
Liu L et al. (69)	24	F	/	Abdominal pain, nausea and vomiting	Abdominal US	EGE	Large number of eosinophils	12900/mcl - 69%	CCS	No
Liu L et al. (69)	15	M	Allergy	Abdominal pain, diarrhea	Abdominal US	EGE	Large number of eosinophils	2740/mcl - 22.7%	CCS	No

Liu L et al. (69)	20	M	Allergy	Abdominal pain, diarrhea	Abdominal US	EGE	Moderate number of eosinophils	6410/mcl - 60.9%	CCS	No
Liu L et al. (69)	27	F	/	Vomiting, diarrhea, bloating	Abdominal US	EGE	Large number of eosinophils	3420/mcl - 18.2%	CCS	No
Liu L et al. (69)	28	M	Allergy	Abdominal pain, nausea and vomiting	Abdominal US	EGE	Large number of eosinophils	3330/mcl - 33.1%	CCS	No
Elliott JA et al. (70)	41	F	Atopy, penicillins and macrolides allergy	Abdominal pain, bloating, nausea, vomiting, diarrhea, dysphagia	Abdominal US and CT	EoE + EoG + EoD + EoC	Abundant eosinophils	10200/mcl - 56%	Prednisolone 60 mg + swallowed fluticasone and oral nonenteric-coated budesonide, then corticosteroid wean to 15 mg, then exclusion diet + prednisolone 5 mg	1

Alhmoud T et al. (71)	38	M	No	Abdominal pain and diarrhea	Paracentesis	EoI	Grossly yellow and cloudy ascitic fluid, with a total of 3620 nucleated cells/ml, 78% of which were eosinophils. Ascitic fluid albumin was 3.2 g/ dl, protein concentration was 5.6 g/dl, and SAAG was 0.7 g/dL. Bacterial	4592/mcl - 28%	Prednisone 40 mg	No

	culture of ascitic fluid was negative, and cytologic examination revealed abundant eosinophils and reactive mesothelial cells, but no acid-fast organisms or malignant cells.

Maleki N et al. (72)	50	F	No	Abdominal pain, abdominal distension, nausea, bloating, and constipation	Abdominal CT	EoC	Ascitic fluid revealed 3840 WBCs/ml (8% neutrophils, 10% lymphocytes, and 82% eosinophils), glucose 110 md/dl, high protein content (4.15 g/dl), SAAG 1.6 g/dl, and an adenosine deaminase 4 IU/l. There	5049/mcl - 27%	Prednisone 40 mg/day		No

							were no malignant cells on cytological examination of the ascites.				
Saraiva N et al. (73)	22	M	No	Abdominal pain, diarrhea, nausea and vomiting	Abdominal US and CT	EoG + EoD	SAAG of 0.8. Cytology identified reactive mesothelial cells and inflammatory cells with a predominance of polymorphonuclear cells	3400/mcl-24.8%	Prednisolone 40 mg/day		No

							(eosinophils) with no neoplastic cells			
Ming G et al. (74)	11	M	Egg allergy	Abdominal pain	Abdominal US and CT	EoG	Elevated WBCs with 93% eosinophils, SAAG 0.8 g/l and LDH 608 IU/l	2473/mcl-31%	Prednisolone (20 mg/day) and cetirizine (10 mg/day)	No
Alsulaiman RM (75)	28	F	No	Abdominal pain and distention	Abdominal US and CT	EGE	WBCs count of 1638/ml, 97% of which were eosinophils, LDH 481 mg/dl,	11492/mcl - 61%	Prednisone (40 mg/day)	No

							albumin 2.7 g/dl (simultaneous serum albumin 2.2 g/dl)				
Khalil H et al. (76)	25	F	Asthma, allergic rhinitis; anaphylaxis due iodinated contrast media	Abdominal pain, nausea, vomiting, diarrhoea and progressive gener- alised abdominal swelling	Abdominal US and CT	EoE + EoG + EoD + EoC	Exudative ascites with SAAG of 8. Amylase, triglycerides, glucose and lactate dehydrogenase in the ascitic fluid were normal. Ascitic fluid	2670/mcl - 30%	Oral CCS + six-food elimination diet		No

		bacterial and tuberculous enrichment cultures did not grow any organisms. WBCs in the ascitic fluid was 2558/ml. Cytological examination revealed numerous eosinophils, scattered histiocytes, neutrophils, lymphocytes

							and occasional normal mesothelial cells. No evidence of malignant cells was noted			
Agrawal S et al. (77)	35	F	Asthma	Abdominal distension and diarrhea	Abdominal US and CT	EoG + EoD	Moderately cellular with 100% eosinophils, negative for malignant cells and sterile	11908/mcl - 52%	prednisone 25 mg	No

Nehme F et al. (78)	22	F	No	Abdominal pain, abdominal distention, constipation, and bloating	Abdominal CT	EoE + EoG + EoD + EoJ	Low SAAG of 0.3 g/dL with significantly elevated WBCs (6900/ml) and > 90% eosinophils.	8800/mcl - 47%	Prednisone 40 mg tapered over six weeks and an empiric six-food elimination diet	No
Martín-Lagos Maldonado A et al. (79)	35	M	Allergic rhinitis	Abdominal pain and abdominal distention	Abdominal CT	EGE	95% eosinophils, with no evidence of malignant cells; adenosine deaminase <40 U/l;	2969/mcl - 29.9%	Prednisone 25 mg	No

							culture negative; SSAG <1.			
Lourenço LC et al. (80)	27	M	Allergic rhinitis and asthma (due to house dust mites sensitization)	Abdominal pain and distention	Abdominal US	EoG	SAAG <1.1 g/dL and significant eosinophilia	4100/mcl - 28%	Prednisolone (40 mg/day for 7 days, then tapered by 5 mg/week)	No
Santos C et al. (81)	32	F	No	Abdominal pain, nausea, postprandial infarction, diarrhea	Abdominal CT	EoC	6912 WBCs/ml, of which 93.3% were eosinophils (6450/ml),	4880 /mcl- 36.5%	Prednisolone 40 mg/day	No

							without malignancy; laboratory testing of the ascitic fluid for bacterial culture and tuberculosis was negative.			
Lopes Azevedo RJ et al. (82)	30	M	No	Abdominal pain, abdominal distention, and watery diarrhea	Abdominal US	EoI + EoC	5182 WBCs/ml with 83% eosinophils (4301 cells/ml). Bacterial and mycological	1710/mcl - 14%	Six-food elimination diet for 4 weeks + 40 mg/day of prednisolone for 2 weeks, followed by a 2-week taper	No

							cultures were all negative.			
Letrán A et al. (83)	36	M	Allergic rhinitis and asthma (HDM sensitization); wheat allergy	Epigastric pain, abdominal distention, nausea, and vomiting	Abdominal US	EoE + EoG + EoD	Total protein 5.50 g/dl; lactate dehydrogenase 158 mg/dl; adenosine deaminase 0.20 U/l; WBCs 9100/ml (45% eosinophils)	4940/mcl - 32%	Prednisone (60 mg/day) and a diet excluding wheat	No

Shi L et al. (84)	57	F	No	Abdominal pain and distention	Abdominal US	EoD	Hemorrhagic peritoneal fluid with a low SAAG; microscopy showed abundant WBCs counts in the fluid, which were predominantly eosinophils. Cytology was negative for malignancy, and cultures were negative for acidfast	8397/mcl-65.6%	Ketotifen 1 mg bid (patient refused steroid treatment)	No	

							bacilli, and bacterial and fungal infections.			
Salah HT et al. (85)	28	M	Asthma	Abdominal pain, nausea, vomiting, diarrhea, loss of appetite, unintentional weight loss of 32 kg	Abdominal CT	EGE	Ascitic fluid was negative for malignant cells, but showed numerous eosinophils mixed with reactive histiocytes	22.6 %	Prednisone 40 mg/die	No

Šaban J et al. (86)	30	F	/	Abdominal pain, nausea, vomiting, abdominal distension, and weight loss of 5 kg	Paracentesis	EoE	Large number of eosinophilic granulocytes without malignant cells	67.7%	Oral prednisone, then topical fluticasone (440 mcg twice daily, then 220 mcg twice daily after 8 weeks) + six-food elimination diet	No
Kim MJ et al. (87)	29	F	Allergic rhinitis (cat epithelium)	Abdominal pain, and diarrhea	Abdominal US and CT	EoN	Ascitic color was orange for increased turbidity; WBCs 6400/ml with 97% of eosinophils; protein level 47 g/dl; albumin level	6351/mcl - 44.8%	Prednisolone 30 mg/die tapered	No

	2.9 g/dl; and adenosine deaminase level 20.2 U/l. Acid fast bacilli stain, bacterial culture and cytology of ascitic fluid were performed, but no specific findings were noted.

Feng W et al. (88)	26	M	No	Diarrhea, abdominal pain, and distention	Abdominal CT	EGE	Massive hemorrhagic ascites with a high eosinophil count. The cultures for bacterial and tuberculosis were negative	8200/mcl	Prednisone 40 mg/die	No
Pereira F et al. (89)	22	M	No	Watery diarrhea, abdominal distension, abdominal pain, vomiting, loss of appetite with weight loss	Abdominal US	EoC	Increased total cell count (4214 WBCs/ml), with 86% of eosinophils	9880/mcl - 54%	Six-food elimination diet + prednisone 40 mg/day for 1 week, followed by a tapering regimen of 5 mg every week	N0

Liang M et al. (90)	44	F	Asthma	Nausea, vomiting, abdominal distention	Abdominal US	EoG	Exudative with 82.7% eosinophils	1310/mcl - 15%	Prednisolone 30 mg	No
Fonseka CL et al. (91)	38	M	No	Abdominal pain	Paracentesis	EoI	Protein 5.4g/dl and WBCs of 420/ml of which 95% were eosinophils.	5580/mcl - 39.3%	Six-food elimination diet	No
Huang X et al. (92)	36	F	/	Abdominal pain and distention	Abdominal CT	EoC	Large quantity of eosinophils	2830/mcl - 30.8%	Methylprednisolone iv 30 mg/die + six-food elimination diet	No
Qua CS et al. (93)	54	M	/	Abdominal distention and dysphagia	Paracentesis	EoE	Turbid fluid removed with a protein level of 45 g/l and	4500/mcl - 26.5%	Vitamin D3 8000 IU/day	No

							the presence of 5580/ml of WBCs, predominantly eosinophils (90%). There was no microorganism or malignant cells present.			
López-Sáez MP et al. (94)	36	F	Allergic rhinoconjunctivitis and bronchial asthma (due to	Abdominal distension, colic and diarrhea	Abdominal US and CT	EoD	Clear ascitic fluid, with 95% polymorphonuclear eosinophils	2200/mcl - 23%	Avoiding the food for which she had reported abdominal discomfort (corn, nuts, legumes, fruits, and various vegetables)	Yes (seasonal according to her respiratory allergies)

			house dust mites, cat and dog epithelia, pollens sensitizations); non structural Lipid Transfer Protein (nsLTP) sensitization				and negative cultures			
Amado C et al. (95)	55	F	Allergic rhinitis and	Abdominal pain, nausea, diarrhea and	Abdominal US	EoC	Moderately cellular (7911 cells/ml) with 87%	8730/mcl	Prednisolone 30 mg/day	No

			penicillin allergy	abdominal distension			eosinophils, sterile and negative for malignant cells				
Yang K et al. (96)	17	M	/	Abdominal pain, diarrhea	Abdominal US	EoG	Large number of eosinophils	Peripheral eosinophilia	CCS		No
He YJ et al. (97)	20	M	/	Abdominal distension	Abdominal US	EoN	Yellow and turbid. WBCs count in the ascites fluid was 1030/ml, of which eosinophils accounted for 56%, and	16056/mcl - 78.4%	Prednisone		No

	lymphocytes for 44%. A large number of eosinophils and a small number of lymphocytes were observed in the ascites smear, while mesothelial cells were occasionally observed, and no tumor cells were recorded.

Gonçalves I et al. (20)	26	F	No	abdominal pain and distention, nausea, vomiting	Abdominal US	EoG + EoD	Hazy dark yellow, with low SAAG and significant eosinophilia (85%). Mycobacterial and microbiological cultures were negative. No cytological signs of malignancy were found.	8400/mcl - 44%	Prednisone 0.5 mg/kg/die		No

Kamath SD et al. (98)	21	F	/	Fever, watery stools, abdominal distension	Abdominal US	EoG + EoD + EoI + EoC	Ascitic fluid was straw colored. Analysis showed total 1040 WBCs - 60% neutrophils, 25% eosinophils and 15% lymphocytes; glucose 93 mg/dl, LDH 131.6U/dl, proteins 4.3g/dl, albumin	7395/mcl - 29%	Hydrocortisone 100 mg iv three times/day, then oral prednisolone 40 mg /day for 2 weeks tapered by 5 mg every week	No

	2.45g/dl, adenosine deaminase 2.6U/l. Smear revealed neutrophils and eosinophils. Malignant cells and parasites were not found. Cultures for bacteria and fungi were negative.

Galere P et al. (99)	41	F	No	Abdominal pain, nausea, vomiting	Abdominal CT	EGE	Increased eosinophils; no viral, bacterial, and parasitic infections.	8940/mcl - 46%	Oral methylprednisolone	No
Silva Mendes S et al. (100)	36	F	Chronic spontaneous urticaria, asthma	Diarrhea, abdominal discomfort, abdominal swelling, early satiety, asthenia	Abdominal US	EoD + EoI	89% of eosinophils and SAAG < 1.1	7000/mcl - 45%	Prednisolone 40 mg per day + six-food elimination diet	No
Belfeki N et al. (22)	16	F	No	Abdominal pain, diarrhea, vomiting	Abdominal CT	EoD	Low SAAG (23.3 g/L) with elevated eosinophil count (WBCs 1000/ml with	13000 eos/mcl (65.7%)	Prednisone 30 mg/day for two weeks with progressive tapering, then prednisone 30 mg/day + leukotriene receptor antagonists, then benralizumab 30 mg/4 weeks for 3 months, then 30 mg/8 weeks for 18 months	Yes

	890 eosinophils). Mycobacbacterial and microbiological cultures were negative, and no malignant cells were detected	

F = female; M = male; US = ultrasound; CT = computed tomography; EGE = eosinophilic gastroenteritis; EoE = eosinophilic esophagitis; EoG = eosinophilic gastritis; EoN = eosinophilic enteritis; EoD = eosinophilic duodenitis; EoJ = eosinophilic jejunitis; EoI = eosinophilic ileitis; EoC = eosinophilic colitis; CCS = corticosteroids (unspecified dosage); SAAG = serum-ascites albumin gradient; CAM = clarithromycin; nsLTP = non structural Lipid Transfer Protein.