Medhat *et al. Egyptian Liver Journal* (2024) 14:24
https://doi.org/10.1186/s43066-024-00329-4

Egyptian Liver Journal

ORIGINAL RESEARCH ARTICLE

Open Access

Clinical and epidemiological characterization of eosinophilic ascites in Egypt: a single center experience

Mohammed Ahmed Medhat[1], Mohamed O. Abdelmalek[1], Mohamed A. Mekky[1], Sherief Abd-Elsalam[2], Mohamed Ahmed Yehya Abdel-Malek[3], Sayed Hassan Ahmed[4], Yusuf S. Amry[1] and Waleed Attia Hassan[1*]

Abstract

Background & aims Eosinophilic ascites is non-common and challenging disease. This study aimed to describe the clinical presentation and treatment of eosinophilic ascites.

Methods This was a prospective single-center study that included cases with eosinophilic ascites who were admitted to Tropical Medicine and Gastroenterology Department, Assiut University Hospital, Assiut, Egypt, during the period between May 2020 to May 2023. The clinical presentation, investigations, treatment, and follow-up data of the included patients were collected and analyzed.

Results Seventeen cases of eosinophilic ascites were included in the study. The main presenting manifestations were abdominal pain (47.1%), and abdominal pain with distension (29.4%). Two patients presented with a picture of intestinal obstruction. Moderate ascites was found in 10 patients (58.8%) by ultrasound. Eosinophilia in the peripheral hemogram was detected in 76.5% of the study population. Endoscopic examination showed gastro-duodenitis in 9 patients (52.9%) and duodenitis in 4 patients (23.5%). All patients showed complete improvement and disappearance of ascites after starting steroids within two weeks. Eleven patients (64.7%) relapsed after discontinuation of steroids.

Conclusion Eosinophilic ascites is an uncommon cause of ascites that is often underdiagnosed. The relapse rate after stopping treatment is high but with excellent response to retreatment.

Keywords Ascites, Relapse, Intestinal obstruction, Eosinophilia, Steroids, Gastroenteritis

Introduction

Eosinophilic gastroenteritis (EGE), first described by Kaijser in 1937, is a rare disorder without a clear etiology characterized by eosinophilic infiltration of gastrointestinal wall layers [1]. The etiology and pathogenesis of EGE remain ambiguous. Allergy may play a role in the recruitment of eosinophils to the digestive tract, as several studies showed that about 50% of the patients with EGE have a preexisting history of atopy [2]. Following an initial trigger, activated tissue eosinophils release various chemo-attractive cytokines resulting in the recruitment of more eosinophils into the affected tissues [3–5].

Eosinophils could infiltrate any layer of the digestive tract. The symptoms of EGE vary according to the

*Correspondence:
Waleed Attia Hassan
wallo403a@aun.edu.eg
[1] Department of Tropical Medicine and Gastroenterology, Faculty of Medicine, Assiut University, Assiut 71515, Egypt
[2] Tropical Medicine Department, Faculty of Medicine, Tanta University, Tanta, Egypt
[3] Clinical Pathology Department, Faculty of Medicine, Assiut University, Assiut, Egypt
[4] Radiology Department, Faculty of Medicine, Assiut University, Assiut, Egypt

affected layer. The available data about the true prevalence of EGE and each of its subtypes are scarce. Nonetheless, published data reported that the mucosal layer is the most frequently affected one, followed by muscular and lastly sub-serosal [4].

EGE's mucosal subtype often presents with abdominal pain, nausea, vomiting, and/or diarrhea. Eosinophilic infiltration of the muscular layer leads to increased gut wall thickness that can produce symptoms of intestinal obstruction [4, 6]. Ascites with a high eosinophilic count in the ascitic fluid are usually the main clinical manifestations in patients with sub-serosal EGE [4, 7–9]. Furthermore, this subgroup is clinically distinct in having abdominal bloating, higher eosinophil counts [2, 10]. Mucosal biopsies may not be helpful in diagnosing serosal and sub-serosal EGE, as 10% of them don't show eosinophilic infiltrations [2, 11]. The radiographic picture for cases with EGE has no characteristic appearance [12]. Treatment includes the elimination of allergic triggering factors, oral steroids, especially for the patient who presents with the obstructive symptoms and eosinophilic ascites [13]. Herein, we try to collect all cases of eosinophilic ascites in our locality and to follow them up. In this study, we aimed to summarize the clinical presentation and management of eosinophilic ascites in our locality.

Methods

This was a prospective study to evaluate the clinical presentation, treatment, and follow up results for this rare disease. Cases with eosinophilic ascites who were admitted at Tropical Medicine and Gastroenterology Department, Assiut University Hospital, Assiut, Egypt, during the period between May 2020 to May 2023 were included in the study.

Detailed history taking, including a history of allergic diseases and data of the full clinical examination, were reviewed. Laboratory tests done for the patients including Complete Blood Count (CBC) with eosinophilic count and percentage, liver function tests (ALT, AST, alkaline phosphatase, serum albumin), serum level of IgE (immune CAP; Phadia AB, Uppsala, Sweden), blood urea and serum creatinine, anti-fasciola Ab, urine analysis, stool analysis for parasites and ova, and ascitic fluid studies including Serum-Ascitic Albumin Gradient (SAAG), the protein level in ascitic fluid and ascitic fluid cytology were recorded and analyzed.

Data of the imaging methods, including chest X-ray, abdominal ultrasonography, abdominal computed tomography for evaluation of small intestinal affection, and exclusion of non-gastrointestinal diseases, were collected and analyzed. Upper endoscopy was done for all patients, and multiple biopsies were taken for assessment of upper gastrointestinal tract pathology.

The diagnosis of eosinophilic ascites was based on the following criteria: any patient presented with gastrointestinal symptoms and ascites, ascitic fluid study showed high eosinophilic count, with or without raised serum IgE level, eosinophilic mucosal infiltration in GI endoscopic biopsies, after exclusion of other causes of ascites, and with or without eosinophilia in peripheral blood [14].

After confirming the diagnosis of eosinophilic ascites, treatment was started with oral prednisone 1 mg/kg for one month (60 mg maximum) [14]. A good response was indicated by the improvement of symptoms, the disappearance of ascites by ultrasound, and the decrease or normalization of eosinophilic count in CBC. After a good response, the dose of steroids was gradually reduced up to discontinuation (within 3 months) [14].

All patients were followed for the occurrence of relapse. Relapse was considered when patients developed ascites again after stopping steroids, and ascitic fluid study and CBC showed high eosinophilic count. Relapsed patients were treated again with oral prednisone 1 mg /kg for one month. After the improvement of their symptoms, they continued the lowest dose of steroids that maintain the patient in remission. The study was approved by the institutional ethical committee of Assiut University in May 2020, and the IRB number is 17,300,385. Informed consent was obtained from included patient or their parents if they are < 18 years old. We followed the guidelines of the Agency for Healthcare Research and Quality (AHRQ) [10] to report included cases.

Statistical analysis

The data were analyzed by SPSS V. 23 (SPSS Inc. Released 2015. IBM SPSS statistics for windows, version 23.0, Armnok, NY: IBM Corp.). Data were expressed in Number (No), percentage (%) mean ($\bar{x}$), and standard deviation (SD). The Chi-square test was used to determine if there is a relationship between two categorical variables. Two-sided *P*- value of < 0.05 was considered statistically significant.

Results

Seventeen cases were diagnosed as eosinophilic ascites (9 males) with median age 37-year-old (range 5—54 years old). Regarding atopy, one patient was known to have bronchial asthma, and another patient had eczema. The main presenting manifestations were abdominal pain in 8 patients (47.1%), abdominal distension in 4 patients (23.5%), and abdominal pain with distension in 5 patients (29.4%). Two patients (11.8%) presented with a picture of intestinal obstruction, which was treated conservatively (Table 1).

Eosinophilia in peripheral hemogram was detected in 13 patients (76.5%). The ascitic fluid analysis showed high

Table 1 Sociodemographic data of the patients

	Number	Percent or IQR
Male[a]	9	52.9
Age[b]	37	22.5
Urban[a]	8	47.1
Smokers[a]	4	23.5
DM[a]	1	5.9
Hypertension[a]	1	5.9
Eczema[a]	1	5.9
Bronchial asthma[a]	1	5.9
Symptoms		
Vomiting[a]	9	52.9
Diarrhea[a]	4	23.5
Intestinal obstruction[a]	2	11.8
Abdominal pain[a]	8	47.1
Abdominal distension[a]	4	23.5
Abdominal pain and distension[a]	5	29.4

[a] Percent

[b] Median and IQR

Table 2 Laboratory investigations of the patients

Laboratory investigations	Result	Percent or IQR
WBCs[b]	14	8
Eosinophilia %[b]	45	16
Elevated ESR[a]	14	82.4
ESR[b]	12	9
CRP[b]	12	8
Elevated IgE level[a]	13	76.5
IgE levels (IU/ml)[b]	234	443
Serum albumin[b]	39	6

[a] Percent

[b] Median and IQR

eosinophilic count in all patients (Fig. 1), and the serum IgE level was high in thirteen patients (76.5%) (Table 2).

Ten patients (58.8%) had moderate ascites, and 7 had mild ascites by ultrasound. Fifteen patients out of 17 (88.2%) had thick intestinal wall by ultrasound and computed tomography of the abdomen (Table 3 and Fig. 2).

Only one patient showed trachealization of the esophagus with gastritis, while 9 patients (52.9%) showed gastroduodenitis. Gastric and duodenal biopsies showed high eosinophilic count in 15 patients out of 17 (88.24%) (Figs. 3 and 4, Table 4). Despite having normal endoscopy, 2 patients out of 3 (66.7%) showed eosinophilia in gastric and duodenal biopsies.

All patients showed a response to steroid therapy. All patients showed complete improvement of the gastrointestinal manifestations and disappearance of ascites within two weeks of therapy. The median duration of follow up was 32 months (IQR: 27). Eleven patients (64.7%) relapsed after discontinuation of steroids. The main manifestations after relapse were abdominal pain and ascites. The occurrence of relapse ranged from one month to one year after the stoppage of steroids. All of them showed improvement of manifestations after re-administration of steroids, and they are on a maintenance dose (5–10 mg) of prednisolone daily.

Table 5 shows the independent predictors of relapse among the studied cohort. In the final multivariable regression model, there were ten predictors: age, sex, residence, three symptoms (vomiting, diarrhea and main symptom), ascites, eosinophilic%, IgE level and serum albumin.

With one-year increase in age, there was 2% (Adjusted Odds Ratio (AOR) = 0.98, 95% CI: 0.81 – 1.0, $p = 0.041$) decrease in the chance of relapse. Also, female patients had triple the risk of relapse (AOR = 2.95, 95% CI: 1.004 – 9.293, $p = 0.044$) compared with males. As well, rural

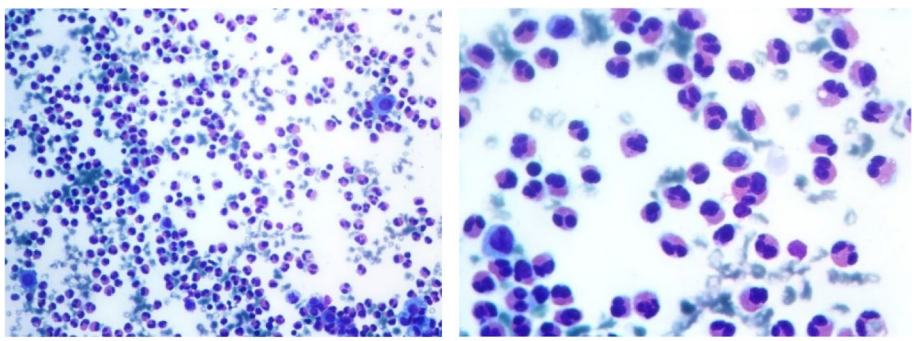

Fig. 1 Smears for cytological examination of the centrifuged ascitic fluid revealed eosinophilic cells by low power (**A**) and by high power (**B**). No evidence of cytological atypia or malignancy. Stained by H& E. in 33 years old female patient

Table 3 Imaging findings of the patients

	Number	Percent
Amount of ascites		
Mild	**7**	**41.2**
Moderate	**10**	**58.8**
Hepatomegaly	**4**	**23.5**
Thick intestinal wall	**15**	**88.2**

residents had 33% less liability for relapse (AOR = 0.67, 95% CI: 0.06 – 0.96, $p = 0.046$) compared with urbans.

For disease symptoms, patients with vomiting had 80% more possibility for relapse (AOR = 1.8, 95% CI: 1.05 – 8.8, $p = 0.031$). Also, those with diarrhea had 44% increase in the risk of relapse (AOR = 1.44, 95% CI: 1.05 – 4.37, $p = 0.037$). Further, cases with pain/distension as the main symptom had 3.5 times the risk of relapse (AOR = 3.5, 95% CI: 1.76 – 9.16, $p = 0.047$).

Moreover, patients with moderate ascites had 84% more likelihood of relapse (AOR = 1.84, 95% CI: 1.002 – 5.114, $p = 0.040$) than those with mild ascites. Additionally, with one-percentage increase in the eosinophilia, there was 5% (AOR = 1.05, 95% CI: 1.001 – 1.113, $p = 0.015$) increase in the chance of relapse. Similarly, with one-IU/ml increase in the IgE level, there was 1.4% (AOR = 1.014, 95% CI: 1.001 – 1.029, $p = 0.024$) increase in the chance of relapse. Likewise, with one-mg/dl increase in the s. albumin, there was 11% (AOR = 0.893, 95% CI: 0.499 – 0.989, $p = 0.044$) reduction in the relapse risk.

Discussion

To our knowledge, this is the first paper that describes cases with EGE in Egypt. Little is known about the prevalence of EGE; however, it is sporadic in distribution, and

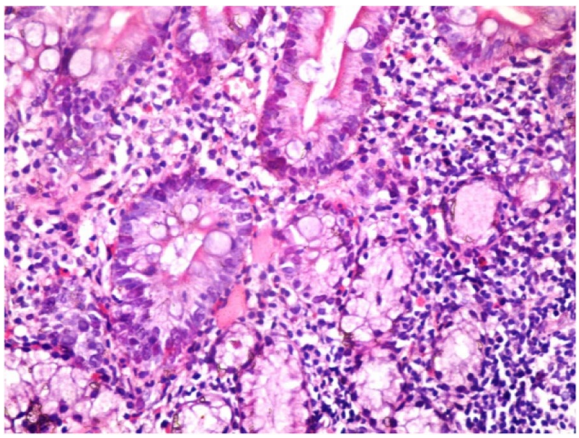

Fig. 3 Show gastric mucosa infiltrated with heavy (Eosinophils) 48 years old male, Stain: H&E

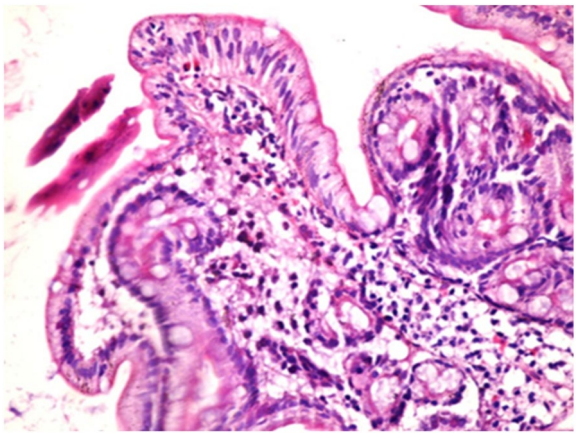

Fig. 4 Show duodenal mucosa infiltrated with heavy (Eosinophils) 31 years old Female patient, Stain: H&E

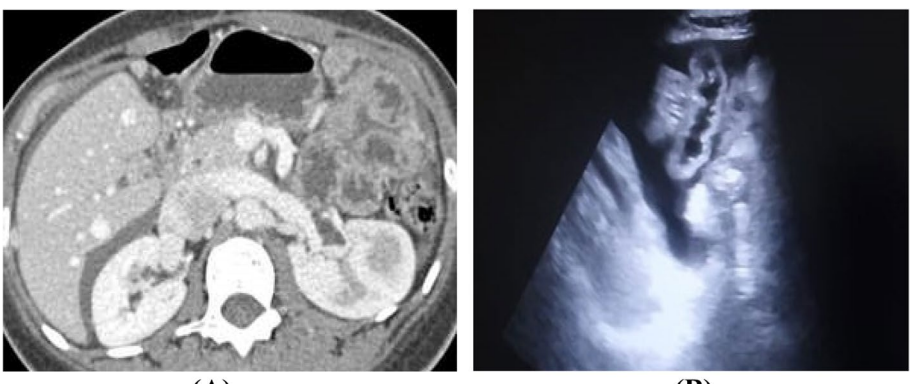

Fig. 2 CT scan of the abdomen (**A**) and Abdominal Ultrasound (**B**) showing the presence of ascites with thick edematous intestinal loops in 50 years old male patient

Table 4 Endoscopic and histopathological findings of the patients

Findings of upper endoscopy	Number	Percent
Normal	**3**	**17.6**
Gastroduodenitis	**9**	**52.9**
Duodenitis	**4**	**23.5**
Trachealization of the esophagus and gastritis	**1**	**5.9**
Eosinophilic mucosal infiltrate	**15**	**88.2**

Table 5 Independent predictors of relapse: multivariable logistic regression

	OR (95% CI)	*P* -value
·Age/years	0.982 (0.806 – 0.999)	**=0.041**
·Sex (Female)	2.945 (1.004 – 9.293)	**=0.044**
·Residence (Rural)	0.667 (0.060 – 0.957)	**=0.046**
·Symptoms		
✓Vomiting	1.801 (1.046 – 8.799)	**=0.031**
✓Diarrhoea	1.444 (1.045 – 4.374)	**=0.037**
✓Pain/Distention	3.498 (1.758 – 9.156)	**=0.047**
·Ascites (Moderate)	1.842 (1.002 – 5.114)	**=0.040**
·Eosinophilic%	1.049 (1.001 – 1.113)	**=0.015**
·IgE Level	1.014 (1.001 – 1.029)	**=0.024**
·S. Albumin	0.893 (0.449 – 0.989)	**=0.044**

OR Odds Ratio, *CI* Confidence Interval

familial occurrence has been reported [15]. It can affect both sexes, although it seems to be more common in men [16].

The median age at presentation in our series was 37 years, with one child out of 17 patients, and 52.9% of our study population were males. To date, the published data about EGE suggests that it can affect any age group; however, it occurs most commonly between the 3rd and 4th decade, with a slightly male predominance [1, 2, 6, 17–19].

In association with peripheral eosinophilia, [20, 21] abdominal pain was the main clinical presentation in this series (47.1%), followed by abdominal distension (23.5%), and abdominal pain with distension (29.4%). All patients had ascites at presentation, and peripheral eosinophilia > 30%. Although the manifestations of EGE vary according to the affected gastrointestinal layer, abdominal pain remains the main symptom [18]. Mucosal layer affection is usually presented by vomiting, diarrhea, and protein-losing enteropathy, while muscular layer affection can lead to partial or total intestinal obstruction. Eosinophilic ascites results from serosal layer affection and may be associated with peritonitis and perforation in severe cases, [22] Infiltration of the duodenal papilla by eosinophils may result in biliary obstruction and pancreatitis [23, 24].

Peripheral eosinophilia is present is about 70% of cases with EGE, and the level of eosinophilia increases with deeper layers of affection [2, 25]. Elevated ESR and IgE can be detected in about two-thirds of EGE cases [18]. In our study, peripheral eosinophilia and IgE were elevated in 76.5%, and ESR was positive in 82.4% of patients.

Endoscopic findings in EGE are neither sensitive nor specific. They include hyperemic gastric and duodenal mucosa, trachealization of the esophagus, thickening of gastric folds, friable or nodular mucosa, rough areas, whitish specks, erosions, or superficial ulcers. Normal endoscopy is also reported in cases with EGE [26–28]. Endoscopy findings in our study included gastroduodenitis in 52.9%, duodenitis 23.5%, and normal findings in 17.6%.

Endoscopic biopsies are mandatory for the diagnosis of EGE [19]. Multiple biopsies are required from normal and abnormal mucosa due to the patchy distribution of the disease [29]. Our study showed eosinophilic infiltrations in the gastroduodenal mucosa in 88.2% of the study population. The standardized diagnostic cut-off value, above which EGE is diagnosed, is an eosinophilic count of 20/ high power field (HPF) [4, 6, 27, 30]. Other histopathological features of EGE include intraepithelial eosinophils and eosinophils in the Peyer's patches [31]. Extracellular deposition of eosinophil major basic proteins (MBPs) was also reported in cases with EGE [32] and may indicate more significant structural damage [33]. Villous atrophy, crypt hyperplasia, or abscesses and epithelial degeneration/ regeneration are also documented in cases with EGE [34].

Imaging methods are useful in the diagnosis of EGE. Ultrasound can detect ascites and intestinal wall thickening and can help in ascitic fluid aspiration for examination [35, 36]. Thickening of mucosal folds, intestinal wall thickening, ascites, and obstruction can be detected by a Computed Tomography (CT) scan [37, 38]. Intestinal wall thickening could be detected by abdominal ultrasound and CT in 88.2% of this case series.

In our study, all patients responded to steroid therapy with complete improvement and disappearance of ascites within two weeks of therapy. Relapsed patients responded well for reinitiated treatment with steroids. Predictors of relapse were larger amount of ascites, younger female patients, vomiting or diarrhea in the presentation.

This was the most extensive prospective study that summarized the clinical presentation and management of this rare disease in Egypt.

Conclusions

Eosinophilic ascites is a rare disease and needs a high index of suspicion for its diagnosis. Oral corticosteroids are the mainstay of treatment. Relapse after initial treatment is common with good response to retreatment. Additional studies are required to follow the natural history of this disorder.

Limitations

The relatively small sample size of our study is a limitation. But eosinophilic ascites is a non-common disease, and further larger cohorts can be done in the future.

Abbreviations

EGE Eosinophilic Gastroenteritis
MBPs Eosinophil Major Basic proteins
SAAG Serum-Ascitic Albumin Gradient

Acknowledgements

Not applicable.

Authors' contributions

MAM participated in the design of the study, coordination of the work, and drafted the manuscript. MOA participated in the design of the study, curation of patients, and critical revision of the manuscript. MM participated in the design of the study, revised the statistical analysis, and helped in drafting the manuscript. SA Helped in revision of the manuscript. MAY participated in the study design and performed the laboratory work and revision of the manuscript. SHA performed the radiological investigations and revised the manuscript. YSA, WAH participated in the design of the study, curation of the patients, data entry and sharing in analysis of the results. All authors read and approved the final manuscript.

Funding

We did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit organizations.

Availability of data and materials

All datasets, on which the conclusions of the manuscript rely are presented in the main paper.

Declarations

Ethics approval and consent to participate

Our study was approved by the Medical Ethics Committee of the Institutional Review Board of the Faculty of Medicine, Assiut University, Egypt. IRB number: 17300385.

Consent for publication

We obtained written consent from all participants according to the declaration of Helsinki.

Competing interests

The authors declare no conflict of interest.

Received: 5 March 2024 Accepted: 26 March 2024
Published online: 03 April 2024

References

1. Kaijser R (1937) Allergic disease of the gut from the point of view of the surgeon. Arch Klin Chir 188:36–64
2. Talley N, Shorter R, Phillips S, Zinsmeister A (1990) Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. Gut 31:54–58. https://doi.org/10.1136/gut.31.1.54
3. Kim Y-J et al (2004) Rebound eosinophilia after treatment of hypereosinophilic syndrome and eosinophilic gastroenteritis with monoclonal anti–IL-5 antibody SCH55700. J Allergy Clin Immunol 114:1449–1455
4. Rothenberg ME (2004) Eosinophilic gastrointestinal disorders (EGID). J Allergy Clin Immunol 113:11–28. https://doi.org/10.1016/j.jaci.2003.10.047
5. Lee CM, Changchien CS, Chen PC, Lin DY, Sheen IS, Wang CS, Tai DI, Sheen-Chen SM, Chen WJ, Wu CS (1993) Eosinophilic gastroenteritis: 10 years experience. Am J Gastroenterol 88(1):70–74
6. Chen M-J, Chu C-H, Lin S-C, Shih S-C, Wang T-E (2003) Eosinophilic gastroenteritis: clinical experience with 15 patients. World J Gastroenterol 9:2813. https://doi.org/10.3748/wjg.v9.i12.2813
7. Wächter B, Jäger-Arand E, Engers R, Manns M (1992) Eosinophilic gastroenteritis with serosa involvement. A rare differential diagnosis of ascites. Z Gastroenterol 30:469–472
8. Sánchez-Fayos M, Miranda R, Renedo L, Porres J, Hernández CG (1992) Eosinophilia and ascites as an expression of a subserous form of eosinophilic gastroenteritis. Rev Clin Esp 191:30–34
9. Santos J, Junquera F, Molero X, Vilaseca J, Malagelada J (1995) Eosinophilic gastroenteritis presenting as ascites and splenomegaly. Eur J Gastroenterol Hepatol 7:675–678
10. Feng W, Zheng K, Shen H (2020) Eosinophilic ascites: an unusual manifestation of eosinophilic gastroenteritis. Int J Colorectal Dis 35(4):765–767. https://doi.org/10.1007/s00384-020-03510-4
11. Katz AJ, Goldman H, Grand RJ (1977) Gastric mucosal biopsy in eosinophilic (allergic) gastroenteritis. Gastroenterology 73:705–709
12. Wiesner W, Kocher T, Heim M, Bongartz G (2002) CT findings in eosinophilic enterocolitis with predominantly serosal and muscular bowel wall infiltration. J Belge Radiol 85:4–6
13. Khan S, Orenstein SR (2008) Eosinophilic gastroenteritis. Gastroenterol Clin North Am 37:333–348, v. https://doi.org/10.1016/j.gtc.2008.02.003
14. Ingle SB, Hinge CR (2013) Eosinophilic gastroenteritis: an unusual type of gastroenteritis. World J Gastroenterol 19:5061
15. AHRQ Evidence Report Summaries (1998–2005) Agency for Healthcare Research and Quality, Rockville. Available from: https://www.ncbi.nlm.nih.gov/books/NBK11854/
16. Min K-U, Metcalfe D (1991) Eosinophilic gastroenteritis. Immunol Allergy Clin North Am 11:799–813
17. Durieu I et al (1992) Eosinophilic ascites. 2 new case reports. Rev Med Interne 13:446–448
18. Klein NC, Hargrove RL, Sleisenger MH, Jeffries GH (1970) Eosinophilic gastroenteritis. Medicine 49:299–319. https://doi.org/10.1097/00005792-197007000-00003
19. ChangJY et al (2010) A shift in the clinical spectrum of eosinophilic gastroenteritis toward the mucosal disease type. Clin Gastroenterol Hepatol 8:669–675; quiz e688. https://doi.org/10.1016/j.cgh.2010.04.022
20. Uppal V, Kreiger P, Kutsch E (2016) Eosinophilic gastroenteritis and colitis: a comprehensive review. Clin Rev Allergy Immunol 50:175–188. https://doi.org/10.1007/s12016-015-8489-4
21. Lee M, Hodges WG, Huggins TL, Lee EL (1996) Eosinophilic gastroenteritis. South Med J 89:189–194
22. Tien F-M et al (2011) Clinical features and treatment responses of children with eosinophilic gastroenteritis. Pediatr Neonatol 52:272–278. https://doi.org/10.1016/j.pedneo.2011.06.006
23. Shin WG et al (2007) Eosinophilic enteritis presenting as intussusception in adult. Korean J Intern Med 22:13–17. https://doi.org/10.3904/kjim.2007.22.1.13
24. Maeshima A et al (1997) Eosinophilic gastroenteritis presenting with acute pancreatitis. J Med 28:265–272
25. Madhotra R, Eloubeidi MA, Cunningham JT, Lewin D, Hoffman B (2002) Eosinophilic gastroenteritis masquerading as ampullary adenoma. J Clin Gastroenterol 34:240–242. https://doi.org/10.1097/00004836-200203000-00009

26. Pineton de Chambrun G et al (2011) Natural history of eosinophilic gastroenteritis. Clin Gastroenterol Hepatol 9:950–956.e951. https://doi.org/10.1016/j.cgh.2011.07.017
27. Chehade M, Sicherer SH, Magid MS, Rosenberg HK, Morotti RA (2007) Multiple exudative ulcers and pseudopolyps in allergic eosinophilic gastroenteritis that responded to dietary therapy. J Pediatr Gastroenterol Nutr 45:354–357. https://doi.org/10.1097/MPG.0b013e31803219d5
28. Manatsathit W, Sermsathanasawadi R, Pongpaiboon A, Pongprasobchai S (2013) Mucosal-type eosinophilic gastroenteritis in Thailand: 12-year retrospective study. J Med Assoc Thai 96(Suppl 2):S194–202
29. Zhang L et al (2011) Eosinophilic gastroenteritis: clinical manifestations and morphological characteristics, a retrospective study of 42 patients. Scand J Gastroenterol 46:1074–1080. https://doi.org/10.3109/00365521.2011.579998
30. Wong GW, Lim KH, Wan WK, Low SC, Kong SC (2015) Eosinophilic gastroenteritis: clinical profiles and treatment outcomes, a retrospective study of 18 adult patients in a Singapore Tertiary Hospital. Med J Malaysia 70:232–237
31. Reed C, Woosley JT, Dellon ES (2015) Clinical characteristics, treatment outcomes, and resource utilization in children and adults with eosinophilic gastroenteritis. Dig Liver Dis 47:197–201. https://doi.org/10.1016/j.dld.2014.11.009
32. Rothenberg ME, Mishra A, Brandt EB, Hogan SP (2001) Gastrointestinal eosinophils. Immunol Rev 179:139–155. https://doi.org/10.1034/j.1600-065x.2001.790114.x
33. Torpier G et al (1988) Eosinophilic gastroenteritis: ultrastructural evidence for a selective release of eosinophil major basic protein. Clin Exp Immunol 74:404–408
34. Keshavarzian A et al (1985) Activated eosinophils in familial eosinophilic gastroenteritis. Gastroenterology 88:1041–1049. https://doi.org/10.1016/s0016-5085(85)80026-3
35. Hurrell JM, Genta RM, Melton SD (2011) Histopathologic diagnosis of eosinophilic conditions in the gastrointestinal tract. Adv Anat Pathol 18:335–348. https://doi.org/10.1097/PAP.0b013e318229bfe2
36. Esola CC, Chopra S, Dodd GD (1997) Sonographic guidance in biopsies and drainages: techniques and applications. Semin Interv Radiol 14:343–369
37. Anuradha C et al (2012) Eosinophilic disorders of the gastrointestinal tract: imaging features. Diagn Interv Radiol 18:183
38. Zheng X et al (2008) Eosinophilic enteritis: CT features. Abdom Imaging 33:191–195

Publisher's Note