Recent Advances in Gastroenterology

Edited by **Her Hsin Tsai**

Contents

1. Advances in the Treatment of Moderate-to-severe Ulcerative Colitis: Anti-TNFs and Beyond 1
Her Hsin Tsai

- Optimizing Conventional Therapies *2*
- Optimizing Anti-TNF Treatments *5*
- New Drugs *7*

2. Surgical Management of Ulcerative Colitis 19
Christopher Harmston

- The Surgeon as Part of the Multidisciplinary Team *19*
- Indications for Surgery *19*
- Surgical Technique *22*

3. Microscopic Colitis 29
Catriona Scicluna, John Schembri, Pierre Ellul

- Epidemiology *29*
- Pathophysiology *29*
- Genetic Factors and Microscopic Colitis *30*
- Environmental Factors *31*
- Clinical Features *33*
- Diagnosis *34*
- Management *38*

4. Eosinophilic Gastroenteritis: Eosinophilic Gastrointestinal Disorders Distal to the Esophagus 49
Her Hsin Tsai

- Epidemiology *50*
- Pathogenesis *50*
- Clinical Features *50*
- Clinical Assessment *50*
- Diagnosis *55*
- Differential Diagnosis *55*
- Natural History and Clinical Course *58*
- Management *58*

5. Eosinophils and the Gut: Eosinophils in the Human Intestinal Tract in Normal Conditions and Major Colorectal Diseases 65
Alexandre Loktionov

- Structural and Functional Characteristics of Mature Eosinophils *65*
- Migration of Eosinophils to the Gut *67*
- Functions of Eosinophils in the Normal Gut *68*
- Eosinophils in the Major Colorectal Diseases *70*

6. Gut Mucus and its Functional Significance in Health and Disease 86
Alexandre Loktionov

- Mucus Composition and Structure Throughout the Gastrointestinal Tract *86*
- Mucus as a Component of the Intestinal Protective Barrier *88*
- Gut Mucus and Microbiome *90*
- Host Cell Exfoliation and Migration from the Epithelial Surface to Mucus Layers in Normal Physiological Conditions *91*
- Gut Mucus Changes Associated with Inflammatory Bowel Disease *92*
- Gut Mucus Changes Associated with Colorectal Cancer *95*

7. Ten Common Errors in the Treatment of *Helicobacter Pylori* Infection..... 106
Javier P Gisbert, Olga P Nyssen

Common Errors in the Treatment of *Helicobacter Pylori* Infection *107*

8. Artificial Intelligence in Gastroenterology 151
Corinna Hauff, Her Hsin Tsai

- Application of Artificial Intelligence in Gastrointestinal Radiology *155*
- Artificial Intelligence in Gastrointestinal Endoscopy *159*
- Key Points for Clinical Practice *164*

9. Improving Polyp Detection at Colonoscopy 167
Sreedhari Thayalasekaran, Pradeep Bhandari

- Simple Measures *168*
- Water-assisted Colonoscopy *170*
- Colonoscopy Technology *170*
- Artificial Intelligence *177*

10. Post-endoscopic Retrograde Cholangiopancreatography Pancreatitis 184
Arjun Sugumaran

- Complications Following an ERCP *185*
- Post-ERCP Pancreatitis *185*

11. Management of Nonalcoholic Fatty Liver Disease 196
Stephen Malnick, Ali Abdullah

- Dietary Changes *197*
- Lifestyle Changes *198*
- Bariatric Surgery *199*
- Medical Therapy of Nonalcoholic Fatty Liver Disease *199*
- Where do we Go from Here? *206*

12. Management of Hepatitis C..... 223
Soe Thiha Maung, Aung Hlaing Bwa, Si Thu Sein Win, Khin Maung Win

- Epidemiology *223*
- Pathogenesis *224*
- Clinical Presentations and Natural History of Diseases *225*
- Diagnosis *227*
- Treatment *229*

13. Neurological Complications in Liver Diseases 238
J Zizzo, Z Rahaman, AR Jayakumar

- Hepatic Encephalopathy *239*
- Hepatitis *241*
- Hepatic Myelopathy *249*

14. The Impact of COVID-19 on Gastroenterology 261
Her Hsin Tsai

- The Structure of SARS-CoV-2 *261*
- Epidemiology *262*
- The Effect of SARS-CoV-2 on the Gastrointestinal Tract *263*
- Effect of COVID-19 on Gastrointestinal Endoscopy *264*
- Effect of COVID-19 on Patients with Pre-existing Gastrointestinal Diseases *265*
- Effect of COVID-19 on Delivery of Gastrointestinal Services *265*

Index 269

CHAPTER

4

Eosinophilic Gastroenteritis: Eosinophilic Gastrointestinal Disorders Distal to the Esophagus

Her Hsin Tsai

INTRODUCTION

Eosinophilic gastrointestinal disorders (EGID) are a group of gastrointestinal (GI) conditions that are characterized by often dense eosinophilic infiltration of the GI tract. Depending on the site involved, it may be variously referred to in the literature as eosinophilic esophagitis (EoE), eosinophilic gastritis (EG), eosinophilic gastroenteritis, enteritis (EGE), and eosinophilic colitis (EC). While gastric, small and large bowel involvement have been recognized for over 80 years, EoE is a more recently recognized clinical entity and is well reviewed in the literature. This review will thus focus on gastrointestinal eosinophilic diseases distal to the esophagus. For the purpose of this review, EGID is the umbrella term covering all the different parts of the GI tract affected.

CLASSIFICATION

Eosinophilic gastroenteritis was first described by Kaijser in 1937 on surgical resection specimens who correctly referred to it as an "allergic affection".[1] Attempts to classify it were made by Klein et al. in 1970.[2] Aside from the site of disease, they classified the condition into three distinct groups:

- *Type 1* has a predominantly mucosal disease only without muscularis infiltration or serosal disease
- *Type 2* has infiltration of the muscularis but without serosal disease or ascites
- *Type 3* involves the serosa as well as presence of ascites.

There is some degree of correlation between the histological type and clinical manifestation of the disease. Mucosal disease might result in diarrhea but does not result in obstructive symptoms. Type 2 would result in narrowing of the gut and obstructive symptoms while type 3 would have both obstructive symptoms and ascites. The exact symptoms would also depend on site—dysphagia in esophageal disease, vomiting if gastric outlet narrowing occurs, and more classical obstructive symptoms if the small bowel is affected. Mucosal disease in colonic involvement would manifest itself as diarrheal disease.

EPIDEMIOLOGY

Eosinophilic gastrointestinal disorder is relatively rare. Based on pediatric survey, it is estimated to have a prevalence of around 22–28 per 100,000 of the population.[3] This figure is likely to be underrepresented and if we include adult cases, the figure is likely to be higher. It still remains a rare disease. Another study estimated the prevalence of EG, EGE, and colitis to be 6.3/100,000, 8.4/100,000, and 3.3/100,000, respectively in United States.[4] EG/EGE can affect patients of any age, but in adults typically presents in the third through fifth decades and has a peak age of onset in the third decade.[5]

PATHOGENESIS

The etiology and pathogenesis of EGID are not fully understood. However, there is good epidemiologic evidence of an allergic component. As with many allergic conditions like asthma, there are raised IgE levels and peripheral eosinophilia.[6,7] In the GI tract, the role of food as possible allergens is based on reports of elimination diets being of benefit in patients with EGID. There is involvement of interleukin-5 (IL-5) which is pivotal in expressing food allergen-specific T-helper 2 (Th2) response.[8] A detailed discussion of eosinophils in the gut in health and disease is available in Chapter 5.

CLINICAL FEATURES

A history of atopic conditions is frequently encountered. There may be defined food allergies, eczema, asthma, and rhinitis.[8]

The clinical manifestation of eosinophilic GI disorders will depend on site affected and class (depth) of eosinophilic infiltration. Hence in the esophagus, the main feature is dysphagia. If the inflammation affects the body and antrum of stomach, it may result in epigastric symptoms of pain and nausea/vomiting. If the small bowel is affected, there would be pain and perhaps small bowel obstruction as well. Colonic involvement would typically result in diarrhea. Mucosal involvement may result in malabsorption while muscularis involvement may lead to dysmotility of the area affected. In the esophagus, it will result in dysmotility, reflux, and dysphagia. Thickening of the antrum of the stomach or wall of the small bowel may result in gastric outlet obstruction or small bowel obstruction. One study suggested a predominance of duodenal, ileal, and colonic involvement with gastric involvement less frequently encountered.[9] If the serosa is affected then there may be ascites. Very occasionally, pleural effusion may occur. In both abdominal ascitic fluid and pleural effusion, the aspirate will be teeming with eosinophils.[10]

CLINICAL ASSESSMENT

As with any patient with a GI complaint, history is paramount in elucidating the problem. A drug history is essential (both prescription and

nonprescription medication). Drug-induced eosinophilia syndromes are not uncommon. There may be a rash associated. History of atopy and any food intolerances should be evaluated. Ingestion of poorly cooked meats from endemic areas should raise concern of parasitic infestations and history of travel to areas where parasitic infestations are prevalent should be noted.

A full physical examination should focus on the abdomen. There may be palpable mass which may or may not be tender. If there is gastric obstruction, a succession splash may be heard and bowel sounds may be active in small bowel hold-up. Look for signs of other organs being involved (skin, eyes, and lymph glands) that may suggest an alternative diagnosis.

Laboratory Findings

Table 1 lists the number (not exhaustive) of useful laboratory investigations. The most common finding is raised peripheral eosinophil count found in over 70% of presentations. The eosinophil count can fluctuate and historic results if available could also be helpful. Counts are usually over 500/μL, typically 500–3,500/μL. On average, levels of 1,000/μL are usual.

IgE levels are raised in children but less often in adults.[6] Stool examination is essential to exclude a variety of parasitic infestations. Serology for HIV, *Strongyloides, and Toxocara* may be useful. Iron deficiency is a common feature. Celiac antibody may be helpful. In patients presenting with diarrheal illness, fecal calprotectin is frequently requested. It is helpful in distinguishing between function bowel problems and organic ones. There are few studies in EGID but calprotectin levels are modestly raised, to levels of 50–100 mg/kg but not to the very high levels encountered in inflammatory bowel diseases which tend to be over 500 mg/kg in active disease.[11]

Imaging

A plain abdominal film is often performed if the patient presents to an emergency department. This may show some mucosal thickening in the form of "islands" or "nodules". These reflect areas of the gut with infiltrative disease. Occasionally there is evidence of subacute small bowel obstruction with dilated loops of bowel with fluid levels. Perforation is extremely rare but has been documented.

Table 1: Laboratory investigation.

Full blood count film examination	*Biochemistry profile*
Immunoglobulins, IgE	Iron studies
HIV serology	Tryptase (mastocytosis/hypereosinophilia syndromes)
Stool examination (cysts, ova)	Parasite serology (strongyloides, toxocara)
C-reactive protein (CRP)	Fecal calprotectin

Computed tomography (CT) scan is widely performed in patients with abdominal symptoms. Magnetic resonance imaging (MRI) is also very helpful in investigating these patients. These imaging modalities will show the affected part of the GI tract with gut wall thickening. This can be very dramatic as seen in the antrum of the stomach in **Figures 2 and 3**. The appearance will depend on the depth of eosinophilic involvement. Mucosal disease may show thickening and nodularity of antrum and duodenum **(Fig. 4)** or a saw-tooth mucosa **(Fig. 7)**. Muscular involvement would reveal luminal narrowing especially in the antrum **(Figs. 2 and 3)** with marked thickening and signs of gastric hold-up with food remnants. In the small bowel, there may be irregular thickening and signs of small bowel hold-up.

Endoscopy

If the site of EGID involvement is accessible to an endoscope, it should be performed and mucosal biopsy specimen obtained. Thus esophageal, gastric, and duodenal diseases are amiable to an upper GI endoscopy and colonic and terminal ileal disease can be accessed with a colonoscope. The appearances can vary from mucosal edema to frank ulceration, but is usually more subtle **(Figs. 1, 5 and 6)**. In the stomach there may be thickening of the folds and in the antrum, mild reddening and thickening. The appearances may mimic *linitis plastica* which is caused by infiltrative tumor of the stomach. Biopsy (sometimes deep mucosal biopsies) will be required to differentiate the two. In the duodenum, there may be raised nodular lesions. In the colon, the inflammation is often patchy and subtle, frank ulceration and spontaneous bleeding or contact bleeding is uncommon.[12] Biopsies should be obtained from any inflamed looking mucosa. In general, mucosal biopsies would suffice. Deep mucosal biopsies are rarely needed and fine-needle aspiration (FNA) is seldom required to establish diagnosis.

Laparoscopy

It is rare to require laparoscopic biopsy. If there is lymph node involvement or suspicion of lymphoma then a laparoscopic examination and a full-thickness biopsy can be performed.

Ascitic Tap

In patients with ascites, ascitic fluid analysis should include cell count with differential, Gram stain, culture, acid-fast bacillus stain, fungal and mycobacterial cultures, and cytology. Although there are no established criteria for ascitic fluid eosinophilia, studies have reported markedly elevated eosinophil counts in patients with EGID with ascites.

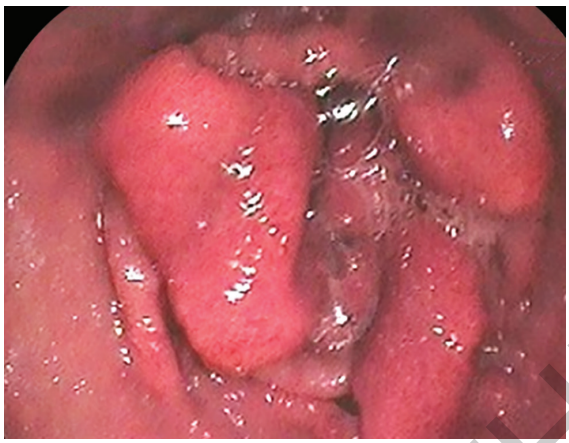


Fig. 1: Eosinophilic gastritis with thickened pylorus.

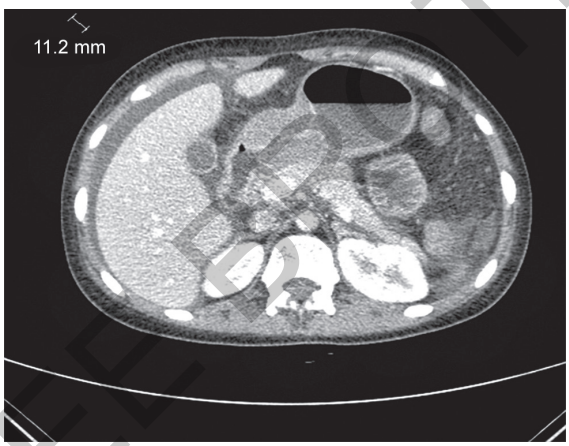


Fig. 2: CT scan showing marked antral thickening of the stomach.

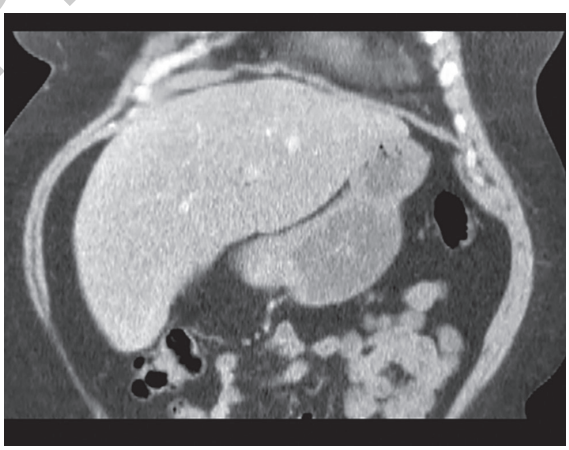


Fig. 3: CT scan of the above coronal view.

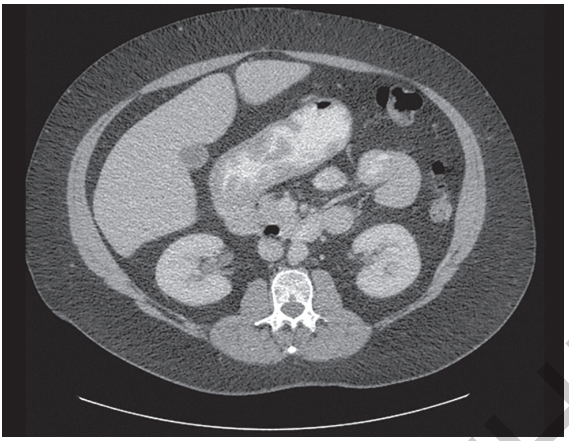


Fig. 4: Duodenal involvement with nodular appearance on CT.

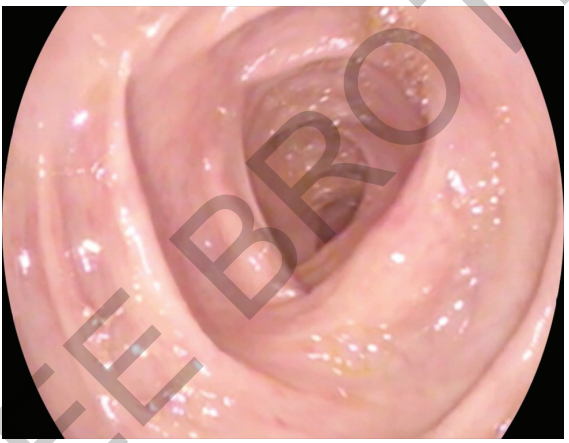


Fig. 5: Eosinophilic colitis affecting the transverse colon.

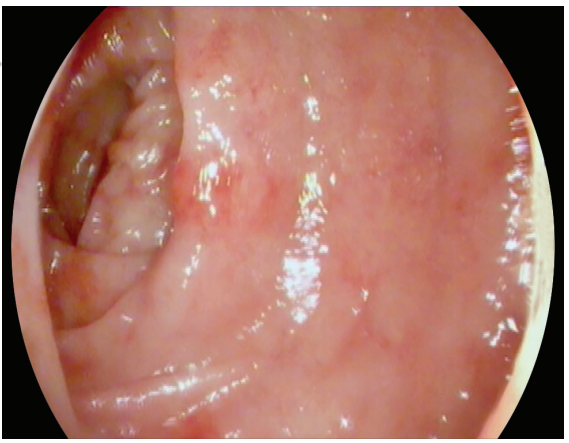


Fig. 6: Colonoscopic appearance of sigmoid eosinophilic colitis.

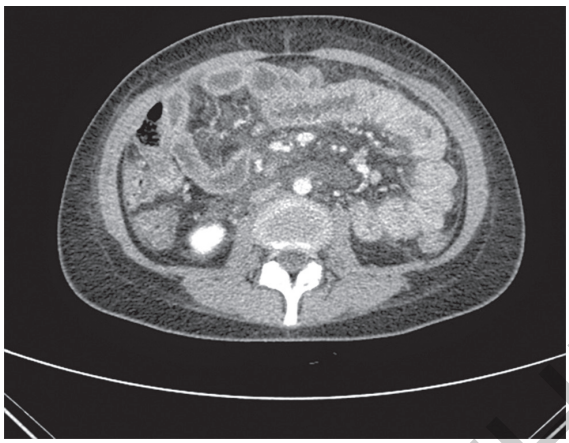


Fig. 7: Small bowel involvement of eosinophilic gastroenteritis.

DIAGNOSIS

Diagnosis is made on histological examination of affected tissue. With the exception of the esophagus, eosinophils can be present in normal physiologic states throughout the rest of the GI tract. Thus the diagnosis of mucosal EGID is established by the presence of more than the number of expected eosinophils on microscopic examination of biopsies of the GI tract. There is no agreed number of eosinophils per microscopic field to make the diagnosis. It depends largely on an experienced histopathologist's call. Many other inflammatory conditions such as inflammatory bowel disease and drug-induced changes may show an excess of eosinophils in the biopsy.

Conventional staining is with hematoxylin and eosin stain (HE) staining **(Fig. 8A to C)** with the eosinophils staining bright pink. It is readily recognized by cell morphology and staining counts made per high power field. Collins had suggested cut-off numbers of eosinophils that would suggest a diagnosis of EGID **(Table 2)**.[13]

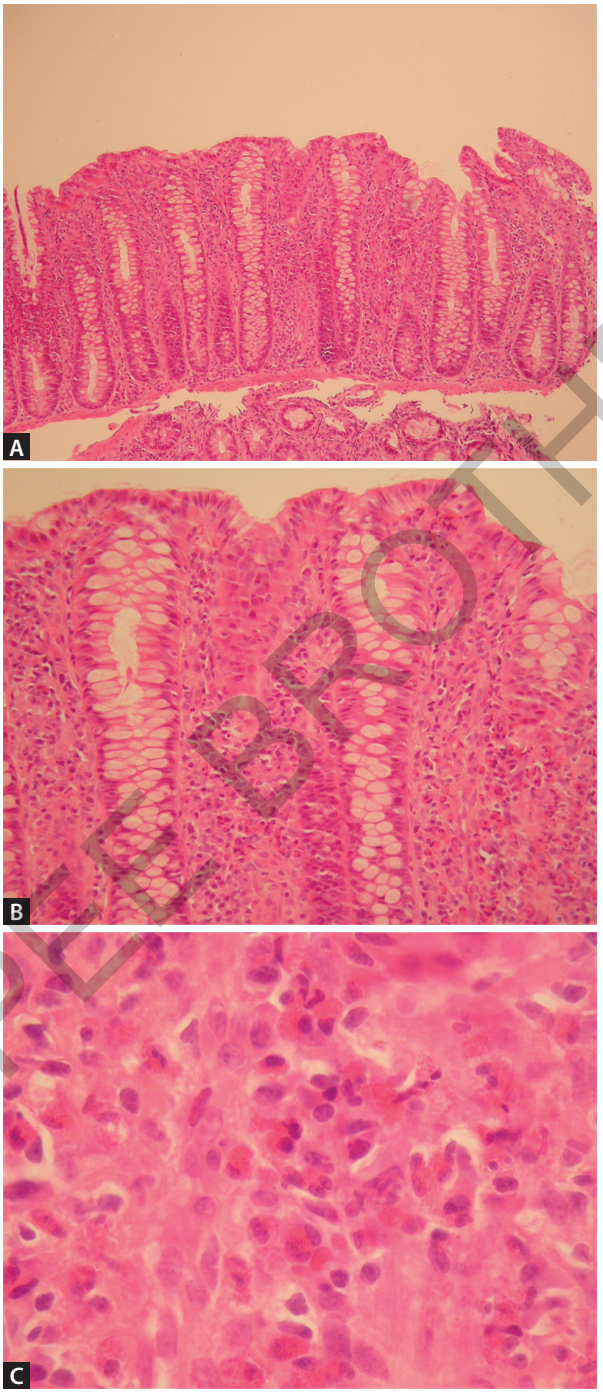
Ultimately the diagnosis is made on the combination of clinical presentation, radiological features, and endoscopic and histological correlation. This is best achieved in a multidisciplinary team meeting and presentation of all the evidence together and reaching the most appropriate diagnosis.

DIFFERENTIAL DIAGNOSIS

Many conditions can mimic eosinophilic GI disorders. These include conditions where peripheral eosinophilia is present.

Malignancy

Many malignant conditions are associated with raised peripheral eosinophil count.[14] In addition, the radiological features may show thickening of the



Figs. 8A to C: Histology of EGID. (A) Low magnification power; (B) Higher magnification; (C) High magnification.
Courtesy: Dr L Karsai.

Table 2: Suggested cut-off levels of eosinophils in biopsies.

	Eosinophils per high-power field (HPF) in 5 HPF
Stomach	≥30 eosinophils
Duodenum	≥30 eosinophils
Ileum	>56 per HPF in the ileum
Right colon	>100 per HPF
Transverse and descending colon	>84 per HPF
Rectosigmoid colon	>64 per HPF

gut wall. They may also present with obstructive symptoms. In the stomach, infiltrative gastric cancer (*linitis plastica*) can have very similar appearance on CT. Obstruction or localized thickening of colon may more likely be cancer than an esoteric condition like EGID. Endoscopy should be possible in most cases and biopsy readily establishes the diagnosis. Lymphoma may be more difficult and may require laparoscopy node or full thickness biopsy of affected bowel.

Inflammatory Bowel Disease

Symptoms of abdominal pain and diarrhea similarly occur in initial presentation of inflammatory bowel diseases. Crohn's disease can affect the upper GI tract but most commonly the terminal ileum. Ulcerative colitis will tend to produce more confluent inflammation and biopsy obtained at colonoscopy should show crypt abscesses and distortion suggesting a chronic inflammatory bowel disease and granuloma may be seen in Crohn's disease biopsies. Occasionally, there may be an excess of eosinophils in histological samples of the colonic biopsies and it is important to consult an experienced histopathologist.

Parasitic Infestations

Infection with *Ancylostoma, Anisakis, Ascaris, Basidiobolomycosis, Capillaria, Strongyloides, Toxocara, Trichiura*, and *Trichinella* can all cause GI symptoms and peripheral eosinophilia. Infection with the dog hookworm, *Ancylostoma caninum*, can mimic EGID clinically and pathologically with eosinophilic infiltration of the gut wall and ascites.[15] However, a parasitic infection can be excluded by examination of the stool for ova or parasites and/or serologic testing. In addition, stool examination in patients with a parasitic infection may reveal Charcot-Leyden crystals, which are the product of eosinophil granules.

Hypereosinophilic Syndrome

Hypereosinophilic syndrome (HES) is an idiopathic condition associated with marked peripheral eosinophilia and may rarely present with GI

symptoms as well. Many EGID patients may fulfill the diagnostic criterion for HES (absolute eosinophil count ≥ 1,500 cells/mL present for over 6 months). However, in contrast with EGID, HES involves multiple organ systems (e.g., heart, lungs, brain, and kidneys).[16]

Other Rare Conditions

Polyarteritis nodosa (PAN) is associated with peripheral eosinophilia and abdominal pain. Nodular masses may also be visualized in the stomach, but, in contrast with EGID, patients with PAN have systemic manifestations, a markedly elevated erythrocyte sedimentation rate, and on biopsy, the eosinophilia is perivascular. Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome) is a vasculitis condition which can affect the bowel with abdominal pain, diarrhea, gastrointestinal bleeding, and colitis. Asthma is the cardinal feature of this disorder (occurring in >95% of patients) and usually precedes the vasculitic phase by approximately 8 to 10 years. Eosinophilic granuloma (Langerhans cell histiocytosis), such as EG, can present as a gastric antral mass. However, it can be differentiated from EGID by its typical granulomatous appearance on biopsy.[17]

NATURAL HISTORY AND CLINICAL COURSE

The natural history and clinical course of EGID are not fully elucidated. However, there is a French study of 43 patients whom they followed up for a mean of 13 years. They found that disease phenotype can be classified as mucosal, subserosal, or muscular in 44%, 39%, and 12%, respectively. Disease location was mostly duodenal (62%), ileal (72%), or colonic (88%); it was less frequently esophageal (30%) or gastric (38%). Blood eosinophilia (numbers > 500/mm^3) was observed in 74% of cases. Spontaneous remission occurred in 40% of patients; the majority of treated patients (74%) received oral corticosteroids, which were effective in most cases. After a median follow-up period of 13 years (0.8–29 years), they identified three different courses of disease progression—18 patients (42%; 9 with subserosal disease) had an initial flare of the disease without relapse, 16 (37%) had multiple flares that were separated by periods of full remission (recurring disease), and 9 (21%) had chronic disease.[9]

MANAGEMENT

There are few controlled trials to base our evidence of best treatment practice. The few trials that are available are on EoE and many of the recommendations below are based on these studies and extended to cover other sites. Treatments that work for EoE may not necessarily apply to other sites. Efforts are now gathering pace to have a consortium to tackle the critical issues of best treatment for these patients. One such consortium is the Consortium of Eosinophilic Gastrointestinal Disease Researchers.[18] One of their projects

"The Outcome Measures for Eosinophilic Gastrointestinal Diseases across Ages (OMEGA)" is an observational, prospective, multicenter study of the clinical, endoscopic, histologic, molecular, and patient-reported outcomes (PROs) in pediatric and adult patients with EGIDs, namely, EoE, EG, and EC, and is focused on defining the natural history of EGIDs. The results of this study are eagerly awaited.

Dietary Therapy

The mainstay of treatment is either dietary or drugs. The rationale behind dietary therapy is the hypothesis that food is the main trigger of the allergic response. However specific testing for food allergens such as skin testing has consistently been less than useful with fewer than 20% having any proven allergies. However, trials have shown benefit from dietary therapy.[19] There are three different dietary approaches to the management of EGID—(1) the elemental diet, (2) the removal of foods based on allergy testing, and (3) the removal of the foods that are most commonly blamed for eosinophilic gastroenteritis.

Elemental diet in its complete form consists of amino acids, short chain fatty acids, and carbohydrates. As such it has no food allergens. However, to have a complete diet of elemental feeds is expensive and unpleasant to take. In most cases and in pediatric practice, a nasogastric tube is usually required to deliver the feeds. The second strategy does not appear to work. Food testing rarely identifies the incriminating allergen or food. The most practical approach is the removal of foods most commonly associated with allergic conditions. A combination approach may also be practical, with the use of elemental diets and transitioning or weaning to an exclusion diet.

There had been a few small studies of dietary therapy in EoE. A retrospective observational study assessed the short-term clinical and histologic responses of two cohorts of children with EE evaluated during two different time periods—one was treated with the standard six-food elimination diet (SFED) and the other was treated with elemental diet (ELED). Of the 60 children who met the inclusion criteria and were compliant with the dietary protocol, 35 were treated with a diet excluding cow-milk protein, soy protein, wheat, egg, peanut, and seafood while allowing all other table foods and 25 were treated exclusively with ELED. Repeat esophageal biopsy specimens were obtained at least 6 weeks later. 26 of 35 (74%) in the SFED group and 22 of 25 (88%) in the ELED group achieved significant improvement of esophageal inflammation (≤10 eosinophils/high-power field). The pretreatment and post-treatment peak eosinophil counts for the SFED were 80.2 ± 44.0 and 13.6 ± 23.8 ($p < 0.0001$) and 58.8 ± 31.9 and 3.7 ± 6.5 ($p < 0.001$) for the ELED group, respectively.[20]

Compliance is the main limitation of dietary therapy. Even highly motivated patients may find elemental diets unpleasant and exclusion diets hard to adhere to. Careful coaching from a dietitian would be necessary

to consolidate patient education. Avoidance of proscribed foods while maintaining adequate nutrition may be difficult, especially in children. Supplementation with vitamins and other micronutrients may be necessary.

If the patient is adherent to the dietary therapy, it has to be continued for at least 6 weeks before reintroducing foods. The patient can be monitored using peripheral eosinophil counts. A response is a reduction of eosinophils by 50%. In patients without a peripheral eosinophilia, the clinical response may be based on symptom improvement. A simple PRO may be all that is necessary which may vary according to disease site. In some cases, histological monitoring may be justified. If endoscopic samples are easily obtained, then a follow-up biopsy at 6 weeks may be obtained. There is a disconnect between reported symptom severity and histological appearance, so such an approach has its merits.

In another study, 50 adults with EoE underwent esophagogastroduodenoscopies (EGDs), biopsies, and skin-prick tests for food and aeroallergens. After 6 weeks of SFED, patients underwent repeat EGD and biopsies. The mean peak eosinophil counts in the proximal and distal esophagus were reduced significantly after the SFED ($p < 0.0001$). After the SFED, 64% of patients had peak counts ≤5 eosinophils/high-power field and 70% had peak counts of ≤10 eosinophils/high-power field. Symptom scores decreased in 94% ($p < 0.0001$). After food reintroduction, esophageal eosinophil counts returned to pretreatment values ($p < 0.0001$). Based on reintroduction, the foods most frequently associated with EoE were wheat (60% of cases) and milk (50% of cases). Skin-prick testing predicted only 13% of foods associated with EoE.[21]

The success of dietary therapy depends on motivation of the patient, good dietetic support, and careful individualizing of therapy.

Adrenocorticosteroids

Corticosteroids have been extensively used in eosinophilic and allergic disorders like asthma. Its use in EGID is logical and intuitive. However, there are few trials to base our evidence on. It is usual to start steroids if dietary therapy fails or compliance is difficult. A dose of prednisolone 20–40 mg/day should suffice. Response usually is apparent within 2 weeks and then a rapid taper over the following 2 weeks can be initiated. The goal of therapy is to induce clinical response while avoiding the toxic effects of corticosteroids.[22]

Monitoring of response is usually by symptoms as peripheral eosinophilia does not accurately predict tissue response. Steroids can quite rapidly reduce peripheral eosinophilia. If response is poor, then further clinical evaluation may be necessary.

In the majority of cases, the patient responds to therapy, and successfully tapered. If they do flare up again, then the same therapy should work. Many patients stay in remission for many months or even years so this strategy of intermittent therapy is sound.

Recent Advances in Gastroenterology

14

With the rapid speed of development in gastrointestinal (GI) medicine, the average practicing gastroenterologists will find it difficult to keep pace. A single volume digest of some of the critical developments in gastroenterology is clearly required, and this volume of *Recent Advances in Gastroenterology* will hopefully fill that need. Major developments in the past 5 years are highlighted, including advances in inflammatory bowel disease (IBD), artificial intelligence in GI medicine, and liver topics including hepatitis C, nonalcoholic fatty liver disease (NAFLD) and the impact of COVID-19 on gastroenterology written by an international team of expert authors.

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