symptomatic 44-year-old woman in whom gastric biopsies showed both *H pylori* infection and dense eosinophil infiltration (29 eosinophils/hpf) of the gastric mucosa. Treatment of erosive *H pylori* gastritis resulted in resolution of symptoms and normal repeat gastroscopy and biopsies 2 months later (120). In the study by Lee et al (24), eosinophil counts in the gastric antrum and body were significantly higher in children with *H pylori* infection than in children with AP-FGID ($P < 0.001$ in the antrum, $P = 0.002$ in the gastric body). However, other case reports have not demonstrated a clear association between EoG and *H pylori* infection (121).

Inflammatory Bowel Disease

Although the exact relationship has not yet been established, *non-EoE EGIDs* have been described in association with IBD (122,123). Gastric eosinophil counts (both gastric antrum and gastric body) were significantly higher in children with Crohn's disease than in children with FAPD or normal controls, although in the latter comparison, eosinophil counts were higher at almost all levels of the GI tract from stomach to rectum (25).

Connective Tissue Diseases with Vasculitis and Collagenous GI Disorders

Gastric eosinophil infiltration has been described in several connective tissue diseases (CTD) including Churg-Strauss syndrome (124–127), as well as in collagenous colitis (128). Benchimol et al (128) reported a 4-year-old girl with EoG associated with collagenous colitis. Arnason et al (129) performed a multi-institutional series of 40 patients [26 females, 14 males; mean age 16 years (range 3–89)], including 24 patients (60%) younger than 18 years. Twelve patients (30%) had celiac disease, collagenous sprue, or collagenous colitis. An eosinophil-rich pattern (≥30 eos/hpf) was noted in 21/40 (52%) patients.

Lecouffe-Desprets et al (130) performed a systematic literature review and identified 20 cases of autoimmune CTD associated with *non-EoE EGIDs*, specifically systemic lupus erythematosus (35%), followed by rheumatoid arthritis (20%), systemic sclerosis or inflammatory myopathies (15% each), and Sjögren syndrome, scleromyositis, or other overlapping CTD (5% each). No patient had a history of atopy. In contrast to classic *non-EoE EGIDs*, EoG and/or EGE occurred in 95% of cases. GI symptoms were often nonspecific. Peripheral eosinophilia was noted in 67% of cases. Upper and lower GI endoscopy showed abnormal findings in only 40% and 30% of cases, respectively. The diagnosis of *non-EoE EGIDs* was confirmed by evidence of eosinophilic infiltration mainly in the mucosa or submucosal layer.

Idiopathic Hypereosinophilic Syndrome (HES)

HES is a term to indicate a more aggressive form of hypereosinophilia (absolute eosinophil count of over 1500/mm^3) resulting in complications such as cardia morbidity which is not the case in primary *non-EoE EGIDs*. HES has been described in a number of case reports in both children and adults aged 8–71 years with eosinophilic infiltration and wall thickening of the stomach (131–134). Kuang et al (135) reported 56 patients with *non-EoE EGIDs* and HES, of whom 34 were categorized as HES/*non-EoE EGIDs* overlap and 22 as multisystem HES. Eosinophilia in multiple GI segments was present in 20 of 30 (67%) patients in whom tissue samples were obtained from all 4 (esophagus, stomach, small bowel, and colon) GI segments. Tissue eosinophilia in all 4 GI segments was found in 5 of 30 (17%) patients. Isolated eosinophilia of the gastric mucosa was not described. Based on the findings of the above study there are certain similarities between the 2 entities, but more studies are needed to draw strict conclusions.

Other

Other entities which should be considered in the differential diagnosis of EoG include autoimmune gastritis (136), invasive gastric adenocarcinoma (137), congenital GI obstruction (79), and adrenal insufficiency (138). When evaluating for EoC, one should consider the above mentioned conditions as well as allergic conditions, infections, hypereosinophilic syndrome, IBD, autoimmune diseases, malignancy, chronic graft-versus-host disease, immune dysregulation such as polyendocrinopathy enteropathy X-linked syndrome and other immunodeficiencies (5,139–143), as well as mastocytic enterocolitis (144,145) or systemic mastocytosis (146,147). Oral immunotherapy (OIT) is currently used to treat food allergies. During desensitization, many patients develop GI symptoms and few develop *non-EoE EGIDs* (148), although this is not universally the case. It is possible that in such cases, unmasking of preexisting *non-EoE EGIDs* may occur, and the occurrence is likely multifactorial. Thus, additional data are clearly needed to draw conclusions about the associations between OIT and *non-EoE EGIDs*.

Recommendation 18 We recommend that other clinically relevant diseases associated with mucosal eosinophilia be evaluated prior to making a non-EoE EGIDs diagnosis (See Table 8).

TABLE 9. Minimum evaluation of patients with symptoms suggestive of eosinophilic gastrointestinal disorders beyond eosinophilic esophagitis (*non-EoE EGIDs*)

In all patients	Only if clinically indicated
• Complete blood count with differential	• Ferritin
• Erythrocyte sedimentation rate	• Coagulation panel
• C-reactive protein	• Celiac serology
• Serum electrolytes, urea nitrogen and creatinine, liver function tests, albumin, total protein	• Thyroid testing • Adrenocorticotropic hormone stimulation testing (ACTH) • Serology testing for Anisakiasis and Basidiobolomycosis
• Microscopic examination of stool for ova and parasites and serologic testing for *Strongyloides* and *Toxocara* species should be directed by local epidemiology and incidence*	• Assessment of GI protein loss (alpha-1 anti-trypsin), steatorrhea (stool steatocrit, fecal elastase and/or tryptase) • Referral to/for: - hematology consultation for evaluation of HES for excessive peripheral eosinophilia; - rheumatology or immunology; - diagnostic paracentesis for ascites;
• Upper with or without lower endoscopy with biopsies of all segments depending on symptoms	- cross-sectional imaging for obstructive symptoms; - full thickness biopsy in case of obstruction; - fecal calprotectin in patients with symptoms suggestive of IBD; - capsule endoscopy/double balloon enteroscopy with biopsies of affected segments.

ACTH = adrenocorticotropic hormone; GI = gastrointestinal; HES = hypereosinophilic syndrome; IBD = inflammatory bowel disease. *Patients who have recently travelled to or stayed in endemic areas should also be screened for antibodies to fungi and parasites such as *Coccidioides*, *Echinococcus*, *Schistosoma*, and *Trichinella spiralis*.