Eosinophilic gastrointestinal disorders (EGID) are increasingly recognized, and the diagnosis requires correlation of clinical and pathologic findings (1). EGID may be associated with numerous disorders such as hypereosinophilic syndrome, may be related to food allergy, or may occur without any identifiable etiology. Currently, with few exceptions (eg helminth infections) histopathology does not identify a specific etiology for eosinophil infiltrates in GI mucosal biopsies.

Eosinophils are normally found distributed as single cells throughout the GI tract, mostly in the deep lamina propria. An important criterion for histologic diagnosis of EGID is increased numbers of eosinophils in mucosal GI biopsies. However, few studies quantitate eosinophils in normal biopsies, and the reporting methods vary. In clinical practice, generating a peak eosinophil count per high power field is the most practical method to quantitate eosinophils in mucosal biopsies. Some studies report either peak eosinophil count, or mean count obtained from several or all high power fields, as number per unit area. Few studies specify the size of the high power field used for eosinophil quantitation, hampering the ability to compare results among studies.

Only one study, of pediatric GI mucosal biopsies, documents that few eosinophils are found in normal esophageal epithelium (2). Two studies of normal pediatric biopsies report numbers of eosinophils in normal gastric and small and large bowel biopsies that appear comparable even considering differing reporting methods (2, 3). Each study identifies the greatest concentration of eosinophils in the right colon. One study of eosinophils in normal colon biopsies from adults reports an increasing gradient of eosinophils in biopsies obtained in the southern U.S. compared to biopsies obtained from the northern U.S (4). A study of dyspepsia in adults includes eosinophil quantities in gastric and duodenal biopsies of patients without dyspepsia, and a study of histologic eosinophilic gastritis in adults includes eosinophil numbers in normal gastric biopsies (5, 6). Reporting methods differ significantly among the studies of normal pediatric and adult upper tract GI biopsies. Eosinophil counts in normal pediatric GI biopsies do not differ between males and females or between atopic individuals compared to nonatopic individuals (2).
Another important criterion to evaluate for a histologic diagnosis of EGID is the distribution of eosinophils. Only one study, of normal pediatric GI mucosal biopsies, documents that small numbers of intraepithelial eosinophils are found in crypt and surface epithelium in normal biopsies (2).

A histologic criterion for EGID of uncertain significance is degranulation, or the presence of extracellular eosinophil granules in tissue sections. Two studies provide data that at least some extracellular granules may result from mechanical disruption of eosinophils rather than active cellular granule extrusion (2, 7).

Most GI mucosal biopsies that exhibit large numbers of eosinophils show other pathologic alterations, such as glandulitis or cryptitis, eosinophil gland or crypt abscesses, etc. However, biopsies that do not exhibit excess eosinophils may be considered consistent with EGID if changes such as eosinophil cryptitis are present, or if eosinophils are found in groups and aggregates, especially in the superficial lamina propria (8, 9).

Consensus recommendations for the diagnosis of eosinophilic esophagitis (10, 11) include recommendations for histopathologic evaluation but consensus recommendations for the clinicopathologic diagnosis of EGID in the remainder of the GI tract do not exist.

In a recent study of gastric biopsies containing numerous eosinophils from adults (mostly) and children, epigastric pain is the most common reported symptom (6). The gastric mucosa in most children in that study is reported as normal endoscopically. In contrast, gastric mucosa in most adults in that study exhibits gastritis/erythema and ulcers/erosions. Criteria for a histopathologic diagnosis of eosinophilic gastritis identified in this study are sheets of eosinophils in the lamina propria, including superficial lamina propria, increased intraepithelial eosinophils, eosinophils in the muscularis mucosa, and ≥30 eosinophils in five high power fields. These are reasonable diagnostic criteria and should be useful for a clinicopathologic diagnosis of eosinophilic gastritis. The differential diagnosis includes Anisakis infection (12, 13).

Eosinophilic enteritis refers to abnormal eosinophil infiltrates in any part of the small bowel and may be subdivided into eosinophilic duodenitis, eosinophilic jejunitis, and eosinophilic ileitis. Numerous eosinophils, in epithelium and in the muscularis mucosa, and blunt villi are useful diagnostic criteria (8, 9). A high index of suspicion for parasitic infections, including reactivated infections, should be maintained for patients whose small bowel biopsies are consistent with eosinophilic enteritis. If the parasite load is particularly heavy, organisms may be seen in the tissue sections (14). Excess eosinophils in the duodenum correlate with early satiety, retching and retrosternal pain (5).

Eosinophilic colitis may affect the entire colon or be more restricted. Useful diagnostic criteria include excess eosinophils in the lamina propria and epithelium, eosinophil cryptitis, eosinophil crypt abscesses and eosinophils in the muscularis mucosa (8, 9). The differential diagnosis includes helminth infections, allergic colitis of infancy (15, 16), allergic colitis
following liver transplantation (17, 18), mastocytosis (19, 20), etc. The presence of acute inflammation should raise suspicion for inflammatory bowel disease (21).

REFERENCES