Eosinophilic esophagitis (EE) is a disease whose presence has exploded into clinical practice. Whereas, this disease was isolated to case reports as recently as 5 years ago, now studies that examine many more patients regularly appear in the medical literature. This rise may stem from two sources: previous underrecognition and a likely increase in prevalence. Underrecognition most likely results from the similarity of the symptoms of EE to gastroesophageal reflux. Overlap and common symptoms include heartburn and dysphagia. Similarly, stricture formation also is common in EE and, although commonly proximal, also may be distal in the esophagus, easily confused with peptic stricture or a Schatzki’s ring. On the other hand, the suggestion that EE is a disease truly on the rise is in keeping with the increasing prevalence of other allergic diseases putatively a result of our self made sterile and thus antigen underexposed society. Indeed, the prevalence of such problems as atopic dermatitis and asthma have increased significantly over time in Western civilization. Furthermore, whereas, EE was first recognized as predominantly a disease of children, several previous reports make it quite clear this also is a disease of adults, generally under the age of 40. That EE represents an allergic esophagitis has been demonstrated through elegant work, showing an increase is CD8, interleukin 5, and eotaxin, cells, and substances typically associated with histologic allergic reactions. The specific antigens that initiate EE are still unclear. Evidence exists for both food and aeroallergens. The former is suggested by resolution of EE in patients placed on an elemental diet or with the avoidance of specific foods that test positive on radioallergosorbent (RAST) or skin prick testing in patients. The latter is given by animal model data. Most likely, both may be involved.

As with any newly recognized disease, each additional study brings valuable information to our still sketchy picture of EE. This is true with this important study by Desai et al. which emphasizes a common presenting symptom of EE: food impaction. Although solid-food dysphagia and impaction has been well recognized in prior studies of EE, this report takes it a step further and demonstrates that EE is perhaps the common cause of food impaction in a community gastroenterology practice. How much we can generalize this to the general population is unclear but will depend on several demographic factors, knowing that EE is found most commonly in white, male, young adults and children. At the very least, this study suggests that it is a common cause of food impaction not previously appreciated. This is an important point, because the medical treatment of EE is far different from gastroesophageal reflux, which uses combinations of inhaled or systemic steroids, leukotriene inhibitors, and identification and avoidance of possible food allergens. Without these therapies, one would expect repeat impactions and progression of esophageal injury to further fibrotic change and stricture formation. Once EE reaches this stage, it becomes particularly problematic for the endoscopist, because the risk of mucosal tear and perforation is higher than one would expect in other esophageal diseases. This study goes on to add several more insights to EE and key points for the endoscopist in its recognition. First is the minimal number of symptoms that these patients had other than their presentation of food impaction. Second is the minimum of obvious endoscopic and barium findings in these patients with EE. Specifically, the only consistent finding among all 17 patients with EE were longitudinal furrows, with a distinct absence of the more expected and obvious findings in food impaction, such as rings or strictures. The importance of noting such subtle symptoms or findings is that it compels the gastroenterologist to be suspicious enough of the diagnosis of EE such that in the absence of an obvious explanation for food impaction, a biopsy specimen of both the proximal and the distal esophagus must be taken either during the initial endoscopy or within a few weeks after. Third, is the upper range of adult patients noted in this report. Whereas, most studies emphasize this disease in young adults in their teens, 20s, and perhaps 30s, 5 of 17 patients with EE were...
between the ages of 40 and 55 years. This study also offers a potential mechanism, other than the number of eosinophils per high power field, of differentiating eosinophilic infiltration of the esophageal mucosa in EE from GERD, specifically, the finding of extracellular major basic protein in the mucosa. Unfortunately, we do not know how much this finding is a manifestation exclusively of the eosinophil count as opposed to a specific finding of eosinophil action in EE. One might hope in those patients where the eosinophil count is intermediate between GERD and EE, it would be helpful. Finally, it is noteworthy that 4 patients had peripheral eosinophilia. Although we do not know whether this represents a more systemic disorder in these 4 patients (no mention of gastric or duodenal biopsies specimens taken), this is in contrast to several prior studies either not noting or excluding patients with peripheral eosinophilia. One then might generally ask why this study offers several differences from other EE studies, including a lack of symptoms other than impaction, a paucity of more obvious endoscopic findings and, somewhat paradoxically, a finding of ringed esophagus in 4 patients presumably without EE. This does not so much indicate discrepancies among these studies but instead the evolving spectrum of this disease as we assemble more data and perhaps the overlap between patients with EE and GERD, some perhaps still misclassified or with features of both.

EE is not only a disease that is here to stay but has become one that must be recognized by the gastroenterologist. This study takes it a step further and suggests it is the most common cause of food impaction. One has to wonder how many other clinical scenarios in our practices will push this heretofore rare pediatric disease to the forefront of adult gastroenterology.

DISCLOSURE

D. A. Katzka is a member of the Speakers Bureau for AstraZeneca, Novartis.

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