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The Challenge of Eosinophilic Esophagitis

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Eosinophilic esophagitis (EoE) is a chronic immune-mediated disorder characterized by eosinophilic inflammation of the esophagus. Although the underlying etiology is unknown, EoE is thought to be a TH2 response to environmental allergens in a genetically predisposed individual [1]. The inflammation in EoE is associated with tissue remodeling and fibrosis and can progress to esophageal strictures. The pathogenesis of EoE is thought to occur by activation of antigen-specific T-cells with subsequent production of pro-inflammatory cytokines. These cytokines (especially eotaxin-3) recruit eosinophils with subsequent activation and release of eosinophilic granules. These granules are thought to be responsible for the clinical symptoms including dysmotility and fibrosis [2].

Treatment objectives for patients with EoE include improvement of symptoms and prevention of complications. Ideally, resolution of esophageal inflammation should be the ultimate goal. Currently, standard treatment regimens include topical corticosteroids and elimination diets [3]. Although many studies have demonstrated the benefits of such therapy, most patients have recurrence of inflammation (and symptoms) once the treatment has been stopped. Therefore, the most difficult decision faced by those caring for patients with EoE is what is the most ideal maintenance therapy. As part of the discussion regarding long-term treatment, physicians need to decide what is the goal of maintenance therapy; specifically, is it resolution of symptoms and prevention of complications or mucosal healing. Although mucosal healing would seem the best option, long-term corticosteroid treatment is associated with complications and compliance with strict dietary therapy is difficult, especially in children. We have previously demonstrated that long-term proton-pump inhibitor (PPI) therapy is effective in improving symptoms and was not associated with increased complications [4]. Zheng et al. [5] has demonstrated that PPI's effect eotaxin-3 release, thereby potentially preventing eosinophilic recruitment and activation. We have recently demonstrated that long-term PPI monotherapy leads to decreased eosinophil degranulation in children with EoE [6]. However, PPI therapy does not decrease esophageal inflammation.

Therefore, the challenge remains as to whether one should strive for mucosal healing and expose the patient to the potential consequences of long-term steroid exposure, continue dietary therapy knowing that most patients are not completely compliant and therefore at risk for under-treatment or consider PPI monotherapy, knowing that, although most patients have improved symptoms, the esophageal inflammation persists and therefore close follow-up is needed to screen for the development of progressive fibrosis and stricture formation.

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