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Review Article

An overview of eosinophilic esophagitis in children

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ABSTRACT

Based on the patient's age, eosinophilic esophagitis (EE) has different clinical manifestations. Infants and young children frequently have vague manifestations such difficulty eating, nausea, and discomfort in the abdomen. Despite having symptoms that resemble gastroesophageal reflux, children with EE, do not respond to intensive ant reflux medication. Dysphagia and food impaction became the most common symptoms in adults and adolescents. EE should also be considered when treating adults and children who continue to have heartburn. Pediatric and adult patients frequently have concurrent allergy disorders such asthma, rhinitis, and eczema, in addition to peripheral eosinophilia and increased total blood IgE levels. Mucosal oedema, furrows, exudates, corrugated bands, strictures, and the alleged "crepe paper sign" are some of the endoscopic characteristics of EE. EE is widely seen as a distinct condition from reflux disorder. According to current understanding, the former could be a symptom of eosinophilic gastroenteritis or be induced by cell-mediated food hypersensitivity. Reports back up the effectiveness of steroid treatment or food restriction. To ascertain the cause, enable early clinical identification, and enhance treatment, more research is required.

Keywords: EE, Pediatric allergy, Gastro-esophageal reflux

INTRODUCTION

A set of symptoms known as EE is commonly confused for refractory gastroesophageal reflux disease (GERD). Patients most typically experience difficulty swallowing solid foods and typically have a record of food impaction. Substernally or abdominally felt pain, as well as vomiting or regurgitating, are further frequent complaints.[1-4] The previous research on EE reported a unique clinical and pathological entity with discrete radiological, endoscopic, and histological results, despite the fact that many EE manifestations coincide with those of reflux.[5-8] In 1993, Attwood et al. evaluated the histological study of tissue samples and found that individuals with dysphagia comprise a somewhat distinct patient base with significant intraepithelial eosinophil levels.[9] Compared to what they observed in biopsy samples derived from

GERD patients, this eosinophilic infiltration was substantially more pronounced. They concluded that high-grade intraepithelial eosinophilic infiltrates appeared to reflect a unique disease process, despite low-grade eosinophilia occasionally being linked to GERD (averaging 3.3 eosinophils per high-power field).[9]

Since then, several researchers have looked into the clinical traits of small populations of people who have this symptom complex but do not react to conventional antireflux medications (including fundoplication).[1-4] Kelly et al started 10 young dysphagic and recurrent emesis patients on an amino acid-based formulation after excluding all meals from their diets in order to test their theory that these persisting problems were caused by a hypersensitivity to foods.[10] The intraepithelial eosinophil count was significantly decreased in follow-up specimens, but the children's complaints had returned in response to open meal challenges.[10] Pediatric gastroenterologists started to search for and identify trends in these children with refractory reflux symptomatology after receiving this fresh perspective.

LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on December 10, 2022. in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the information about EE in children. There were no restrictions on date, language, participant age, or type of publication

DISCUSSION

Eosinophilic infiltrate into the mucous membrane is the hallmark of EE, and multiple investigations have shown that this is associated with an inflow of Th2 dominant lymphocytes, basophils, mast cells, and iNKT cells.[11-13] Important involvement for IL-4, IL-5, STAT6, and CCR3 (the receptor for eotaxin-3) have been shown in murine models of EE.[14,15] Numerous research has looked into how genetic inheritance affects the etiology of EE. A first-degree relative with EE increases an individual's probability of developing the disease, and monozygotic twin concordance of inheritance of EE is greatly raised at about 40%.[16,17] Eotaxin-3 (CCL26) and filaggrin (FLG) have been linked to EE inheritance in analyses of single candidate genes.[14,18] Furthermore, multiple potential genes have been found in EE cohorts in the USA through genome-wide association analyses. In a preliminary, smaller cohort analysis, a TSLP variation was discovered that was associated with TSLP overexpression in the esophageal mucosa.[19] An additional CAPN14 version was found after this cohort grew.[20] The results of the TSLP and CAPN14 variations were repeated in a different cohort, and new relationships at c11orf30, STAT6, and ANKRD27 were also discovered.[21] A heightened incidence of EE with partial penetrance has been linked to a number of monogenic illnesses. As a result, it is now understood that EE follows a complicated, multifactorial inheritance pattern. As previously mentioned, atopy has been closely associated with EE. Food allergens have been shown to be the most frequent cause of mucositis in EE, and several investigations have looked at the advantages of eliminating allergenic food items from the diet for the management of EE.[22,23] In some EE patients, external allergens are also linked to inflamed esophagus, which can cause seasonal flare-ups of the condition.[24-26] Therefore, identifying the aeroallergen sensitivities of EE patients and managing their allergic rhinitis effectively may be a key action in avoiding flare-ups of the condition. Many clinical trends could be useful in bolstering the identification. Up to 81% of the patients reported by various investigators are male.[2,3,27,28] Additionally, observations of a bimodal distribution in children with two peaks—one in infancy and the other in adolescence-have been made.[1,2] Esophagogastroscopy with proximal and distal esophageal biopsy is the most important diagnostic procedure for EE. Esophageal mucosal granularity, adhering whitish exudate, concentric circles, vertical linear furrowing, and low esophagus strictures are only a few of the unusual signs seen during esophoscopy in EE.[2,4,28-30] The most frequent aberrant discoveries include erosive lesions, striated lesions, and furrowed lines. Additionally, EE-related strictures are said to be more delicate than peptic disease-related ones, putting individuals at greater risk for profound mucosal rupture and perforation during dilatation procedures.[2] Orenstein et al. emphasized the connection between EE and airway symptomatology by stating that 62% of EE patients experienced airway problems, such as wheezing, pneumonia, sinusitis, or congestion.[4] Asthma is the most prevalent issue, however 25% of individuals also experience rhinosinusitis. The signs of EE and reflux esophagitis are very similar to one another. It is discovered that many patients have already been given a GERD diagnosis. Studies have stressed that when individuals do not react to traditional GERD treatment, EE should be taken into account.[3] Although EE and reflux may coexist due to aberrant tone of the lower esophageal sphincter from extreme inflammation, pH study results are frequently normal in individuals with EE.[2] Since 41% of the individuals evaluated had aberrant pH probe values, we were unable to identify any consistent pattern. Individuals who have undergone several esophageal dilations for dysphagia and food impaction should also be believed to have this condition. When a significant eosinophilic infiltration of the esophageal epithelial lining is seen histologically, the diagnosis is then established.[28] The output of diagnostic information is greater in tissue biopsies taken from visibly aberrant regions of the esophagus, especially in regions with whitish exudate or plaque.[2] Although the etiology of EE is not fully known, the link to other atopic

illnesses raises the possibility that it is an immune dysregulation condition.[31] In various case series, RAST, skin prick, or skin patch testing revealed food allergies in more than half of the individuals with EE.[4] Sensitized individuals can experience problems up to eight hours after consuming provoking foods in open food challenges.[32] Since peripheral eosinophils and IgE are typically raised in EE individuals, they may provide some hints in the diagnostic approach.[28] From 1998, when Faubion et al published four younger patients with EE who were effectively treated with this therapeutic intervention, ingested topical corticosteroids have become a staple of management.[33] The safety and effectiveness of this medication were further shown in a second prospective investigation of children with EE.[31] The presenting manifestations of all nine of the eleven EE children whose food restriction failed were managed with fluticasone propionate that was ingested.[31] According to this course of therapy, ingested fluticasone was administered to 79% of the individuals examined in this series, and the bulk of them improved. The substance most frequently used in ingested topical corticosteroid treatment is fluticasone propionate, which is administered by a typical metered-dose inhaler. The purpose of teaching the individual a "poor" inhaler approach is to have them ingest at least 80% of the drug without using a spacer. The typical dosage ranges from 44 ug to 220 ug each puff, two to four puffs two times daily for six weeks, based on the child's bodyweight and the extent of the illness. With little gastrointestinal absorption and quick first-pass liver metabolism, this method enables direct delivery of the active substance to the site of inflammation while preventing the negative impact of oral steroids. Topically administered steroids that are taken occasionally cause resistance, oral thrush, or esophagus candida infection, and their effectiveness is insufficient if deeper tissue layers are affected (in more serious conditions). In order to reduce the risk of thrush, individuals are advised to rinse their mouths with water after consuming a dose. They are also advised to refrain from any additional oral consumption for 30 minutes to ensure that the drug is not diluted. Children with EE who are resistant of or insensitive to ingested steroids may benefit from more conventional oral steroids, which have played a major role in the therapy of many immunological dysregulation disorders. Dramatic histological progress (from a mean of 34 to 1.5 eosinophils per high-power field) was observed in 19 of 20 EE cases in a recently published trial of a four-week regimen of methylprednisolone.[34]

In keeping with the idea that food antigens may be the major instigating factors, elimination diets can be an alternate form of therapy, especially for people who have food intolerances. Orenstein et al. showed that 10 of 12 individuals with food allergies who were put on elimination menus improved from the treatment, despite contradicting data on the efficacy of diet restriction.[4] Elimination diets can be combined with other forms of therapy such oral corticosteroids or fluticasone ingested as a medicine. With the successful step-by-step return of questionable meals, elemental solutions like Neocate have been employed for very resistant cases.[10] Patients may also be given the formulation through a transnasal or gastric feeding tube as an option. Last but not least, anti-interleukin-5 treatment (mepolizumab) has lately come to light as a viable therapy for instances that are extremely resistant. In a pilot research, injectable anti-interleukin-S was administered for six weeks to patients with hypereosinophilic conditions, which include EE, and it was effective with no significant side effects.[35] To support these findings, further research involving greater numbers of patients would be required. Regardless of the technique of treatment, symptom relapse may happen in more than half of patients after treatment is finished, necessitating appropriate child and parent counseling as well as active follow-up.[28]

Biologics

There has been enthusiasm in figuring out whether there is a tailored treatment for EE, and monoclonal antibody therapy for EE sufferers is currently being actively investigated. Anti-IL-5 is the only biologic that has been studied thus far in pediatric patients. Mice lacking in IL-5 are resistive to developing EE, which is evidence that IL-5 is essential for eosinophil transport to the esophagus.[15] The FDA has granted mepolizumab orphan medicinal status for the therapy of hypereosinophilic syndrome and eosinophilic granulomatosis with polyangitis, and it is now recommended for the therapy of serious eosinophilic asthma in people aged more than or equal to 12 years old.[36] Preliminary findings in adult populations showed that mepolizumab reduced eosinophil average and peak levels in the mucous membrane and reduced serum eosinophil numbers without suppressing other peripheral cell numbers.[37,38] The fact that patient symptom grades did not enhance, however, was a significant concern raised by these trials. Two different studies in juvenile groups have looked into the anti-IL-5 monoclonal antibodies mepolizumab and reslizumab. Reslizumab showed histological advancement but no clinical improvement in the EE main symptom evaluation of the Children's Health Questionnaire in a prospective controlled trial by Spergel et al. in cases between 5-18 years of age with PPInonresponsive EE.[39] In a multicenter, randomized, double-blind study of mepolizumab in EE patients aged 2 to 17 who had not responded to prior treatments, Ass'ad et al. reported similar outcomes. Mepolizumab therapy was risk-free and reduced mucosal eosinophilia.[40] When using a pediatric-specific complaint assessment, there was no change in clinical symptom levels, nevertheless. Further monoclonal antibody trials have been conducted in adults that have not yet been conducted in pediatric patients. Children have not been tested with anti-IL13 monoclonal antibodies, although they have been tested in adults with PPI-resistant EE.[41] While there was a significant reduction in esophageal mucosal eosinophils, medical outcomes were not significant, similar to the

outcomes reported with anti-IL5 treatments. Individuals with EE and eosinophilic gastroenteritis have participated in a number of minor investigations using the anti-IgE antibody omalizumab; the bulk of these cases showed no progress in their EE, demonstrating that EE is not an IgE-mediated condition.[42]

Esophageal dilation

Esophageal strictures, the most serious sequela of EE, are only treated through esophageal dilatation. In large published studies, a rise in age by a decade is linked to a 2.1 (95% CI, 1.7-2.7) rise in the odds ratio of esophageal fibrostenosis.[43] The probability of stricture generation increases with increasing diagnostic lag, while the risk of food impactions decreases over time with EE management.[44] In children with EE, there is an uncertain specific susceptibility of stricture generation. While pediatric EE patients' esophageal constriction has been demonstrated to have reversible element that is connected to level of inflammation present, it is commonly accepted to be lower than in adult patients.[45] Constant dysphagia, odynophagia, slowed food passage, and food occlusion are all signs of stricture. Individuals may start to chew food extremely slowly and carefully, sip liquid frequently, and cut food into very little pieces as compensatory dietary habits. At every session, it is crucial to check EE cases for these symptomatology and actions. It is advised to acquire an esophogram to assess extent of esophageal constriction if permanent stricture is presumed before doing an upper endoscopy because this method is not the best one for detecting strictures.[46]

CONCLUSION

In the past decade, EE has gradually garnered attention, but it is still not well understood in the domain of otolaryngology. Since many individuals have concurrent pharyngolaryngeal and airway problems, otolaryngologists should be acquainted with presentation. Diagnosis requires an esophagoscopy with distal and proximal esophageal area biopsies targeted at obvious irregularities. RAST, skin prick, or patch diagnostics for food allergies may be useful in detecting potential causal factors that need to be limited or eradicated. While typically high, peripheral eosinophil levels and IgE are less particular for the illness. Inhaled steroids that are ingested have been demonstrated to be efficient and well-tolerated, although there are additional treatments available for instances that are resistant to these.

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