Introduction

As an otolaryngologist we have a plethora of patients come through the office each day with complaints of dysphagia. Many times we chalk the diagnosis up to the reflux gods and place patients on proton pump inhibitors. Rightfully so, reflux has been associated with many other disease processes such as rhinitis, rhinosinusitis, otitis media, laryngitis, lymphoid hypertrophy, and even asthma. Although reflux is at least a contributor to head and neck disease, it is a requirement of the profession to keep an open mind and always be thinking, “What else could this be?”, rather than falling into the trap of cookbook medicine. In this article a relatively new entity, eosinophilic esophagitis (EoE), will be presented as a disease process to look out for, especially in our population of patients who are resistant to standard therapies for dysphagia.

History

EoE was first described in 1978 based off of biopsies from the esophagus, but reflux was thought to be the driving force behind the disease (1). This notion was supported by several studies in the 1980’s. Winter et al. correlated intraepithelial eosinophils (>1 eos/hpf) with pH, manometry, endoscopy and histologic findings all suggestive of reflux disease (2). Lee et al. presented a case series of 11 patients, showing 91% with >5 eos/hpf had reflux esophagitis (3).

In the early 1990’s, however, work was being done that would revolutionize the thinking behind esophageal eosinophilia. In 1993, Attwood et al., performed a retrospective review of 12 patients who had all undergone esophagram, PH monitoring, esophageal manometry, and endoscopy with biopsy. They characterized patients as having either high grade (≥20 eos/hpf) and low grade (<20 eos/hpf) eosinophilia. On average the patients in this study had 56 eos/hpf. Ninety-two percent of patients had high grade eosinophilia but none of these patients had reflux based on pH studies. Interestingly, 58% of patients have allergies, asthma, or other atopic disease. The authors then compared this group with 90 controls that had pH proven GERD, and only 48% had intraepithelial eosinophilia with an average of only 3.3 eos/hpf (4). This study clearly paved the way for other studies to refute GERD as the sole cause for eosinophilia.
In 1995 Kelly et al. suggested a different cause of the disease. They evaluated a cohort of 75 pediatric patients with finding of reflux who were resistant to therapy and found that 31% had persistent eosinophilia on biopsy. Of the 75 patients evaluated, 12 agreed to proceed with a prospective trial of an elemental diet for 6 weeks. Upon treatment with the elemental diet, 80% showed complete resolution of symptoms and 60% showed complete resolution of eosinophilia on repeat biopsy. Average eosinophilia dropped from 41 to 0.5 eos/hpf over the study period, and again, 70% of patients also had atopic dermatitis or asthma as a co-morbidity. Ten of the twelve patients had foods reintroduced and within a median of 1 hour, symptoms returned with the most common foods causing problems being cow’s milk, soy, wheat, peanut, and egg (5). Their conclusion, then, was that EoE may be due to more of a food allergy than reflux and that elemental diets are very effective in treating symptoms in patients resistant to medical therapy for dysphagia.

Other studies began to look at the possibility of allergy playing a role in the disease process. One patient was reported to have relapsing disease visualized on endoscopy when pollen counts were high, with resolution of symptoms and endoscopic findings in the winter months were reported (6). Ruchelli et al found that 56% of patients failing anti-reflux therapy had wheezing, atopic disease, or rhinitis. Of 16 patients in the failure group, 8 completely resolved with corticosteroids and 4 improved with elemental diet (7). Had this been due to reflux patients would have theoretically worsened with corticosteroid therapy given the side effects of the medication.

By 2003 the elemental diet had really caught on as a valid treatment in patients with esophageal eosinophilia that were resistant to medical therapy. Markowitz et al. looked at 51 children and treated then with 4 weeks of an elemental diet. Ninety-six percent showed symptomatic improvement in vomiting, abdominal pain, and dysphagia. The mean time to improvement was only 8.5 days and a drop of 33.7 to 1.0 eos/hpf was observed in those treated (8). In contrast to earlier studies with small numbers this study showed that in a large population earlier trends could be repeated on a larger scale. Therefore in 2007 the first consensus statement for EoE was published with diagnostic criteria for the disease. Diagnostic criteria can be found in Table 1 below.
Eosinophilic Esophagitis

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<tr>
<th>Presence of Symptoms</th>
<th>Adults</th>
<th>Children</th>
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<tbody>
<tr>
<td></td>
<td>Dysphagia</td>
<td>Heartburn</td>
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<td></td>
<td>Heartburn</td>
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<td>Retrosternal chest pain</td>
<td>Nausea/Vomiting</td>
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<td>Odynophagia</td>
<td>Abdominal pain</td>
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<td>Regurgitation</td>
<td>Regurgitation</td>
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<td></td>
<td>Food Impaction</td>
<td>Failure to Thrive</td>
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<tr>
<th>Histologic Findings</th>
<th>1. Patients must have biopsies after 6–8 weeks of twice daily acid suppression with proton pump inhibitors or have a documented negative pH study.</th>
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<td>2. Normal gastric and duodenal biopsies</td>
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<td>3. Esophageal biopsies ≥15 eos/HPF</td>
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Table 1. Showing diagnostic criteria for EoE determined in the 2007 consensus statement (9).

Epidemiology and Characteristics of Disease

At this point in the evolution of defining the disease we had ruled out reflux as the primary culprit, assumed allergy as the new target for disease development, and set diagnostic criteria, but there was still a lot more to learn about the disease.

EoE seems to be relegated to the westernized world. Prevalence studies have been performed to look at just how many of us are affected. A random population survey was performed on 3000 adults, and 1563 were invited to have an endoscopy performed based on survey findings suggestive of upper gastrointestinal disease. Of the 1563 patients, 100 had endoscopies performed with biopsy. Eosinophilia was present in 5% of patients but only 1% met histologic criteria for EoE (10). One study showed that prevalence is actually on the rise and that it mirrors atopic or allergic disease increases over the same time period. In 1995 the prevalence was only 0.5/100,000, but in 2004, just 10 years later the prevalence was nearly 8.9/100,000 (11). Part of these increases can be attributed to better awareness of the disease as an entity, but there is a clear increase over the 10 year period.

Males represent approximately 60-80% of cases and this is thought to be in part to the cytokine IL-13 being present on the X chromosome (11). Since males have only one copy they are more susceptible to mutations. The age at presentation is bimodal with adults presenting within the 4th to 6th decade and children presenting between 5 and 10 years of age. It is still unclear whether childhood disease progresses into adulthood given the lack of longitudinal studies. Genetics plays an important role with 7% of patients having a parent with EoE or a stricture and 5% having a sibling with EoE.

Children with EoE tend to present with more systemic symptoms such as nausea and vomiting, abdominal pain, and failure to thrive (1). More children are affected by foods than adults by comparison (70% vs 30% respectively). Those with IgE mediated forms tend to be less responsive to
long term corticosteroid therapy and strictures are rarely present in children supporting the idea that this is a chronic disease. Again it is common for children with EoE to have parents with longstanding dysphagia or documented eosinophilia/strictures on endoscopy.

The natural history of disease has shown that most patients with EoE have a lag of 4.3 years between symptom onset and the diagnosis of disease. It is restricted to the esophagus and is a chronic disorder. The chronicity can be either chronic persistent with no intermittent relief or chronic relapsing with spontaneous resolution of symptoms at any time during the course of disease followed by recurrence. Prolonged disease leads to remodeling of the esophagus with fibrosis and stricture formation, however unlike reflux disease and Barrett’s esophagus, EoE is not associated with any premalignant or malignant transformation to date. A 17 year database has also shown no deaths related to EoE. One study looked at quality of life issues and showed that about 50% of people with disease just learn to deal with the symptoms however the other 50% require diet and lifestyle modifications. Some patients will actually refuse solid foods unless they have water or other liquids to help wash the food down (12).

Several conditions have been shown to be implicated with EoE in otolaryngology. Clearly allergies are implicated in anywhere from 60-80% of patients, and rhinosinusitis is present in about 19-25% of patients. Other diseases found to be associated have been laryngitis, subglottic stenosis, and recurrent croup (13).

**Pathophysiology**

While there are still many facets to the disease process to be worked out it is clear that EoE is a multifactorial disease. Genetics, the immune system, and environmental or external allergens all seem to play a role, but how do we know that there are multiple pathways involved. First, allergic disease is present in some but not all patients with EoE, so allergy cannot be the only cause. Second, only a minority of patients present with anaphylaxis. This argues that the classical IgE mediated response is not the primary player in the immune system. Third, seasonal variation disease has led to the study of aeroallergens. Allergens have been shown to increase IL-5 and IL-13, both of which are cytokines from the Th-2 cell mediated hypersensitivity pathway (14).

As the Th-2 cell mediated hypothesis gained ground, several studies were performed to determine how the process actually unfolds. Two studies looked at epicutaneous exposure of antigens and showed that despite a strong systemic Th-2 response, chronic cutaneous exposure of antigen, bone marrow eosinophilopoiesis, and circulating eosinophilia; EoE and lung inflammation could not be obtained. In the presence of a nasal challenge of antigen, however, a robust inflammatory reaction and episode of EoE could be produced (15, 16). Another study looked at knockout mice deficient in IL-4, IL-5, and IL-13 who were unable to amount a response to antigenic stimulation unless intratracheal IL-13 was administered. Upon administration both airway and esophageal eosinophilia ensued (17,18). Taking these studies into consideration it was proven that both external triggers as well as a strong intrinsic Th-2 response was required thus making the argument that this disease was in fact multifactorial.

As I discussed earlier, genetics plays a healthy role in the development of the disease as well. Certainly atopic disease can be passed on from parents to child. Also males are more affected than
females potentially due the two changes for the IL-13 receptor being located on the X chromosome (14). More importantly is the presence of eotaxin-3. This substrate/gene has been found to be the signature gene for EoE. It is a powerful eosinophil activator and chemo attractant and is induced in all patients with EoE regardless of allergy, gender, or in patients with associated reflux. IL-13 actually induces eotaxin-3 in keratinocytes through the STAT-6 pathway leading to epithelial proliferation and fibrosis. Mice with genes targeted against the eotaxin-3 receptor, CCR3, are protected from the development of the disease. One single nucleotide polymorphism of the gene has been found in EoE patients and is 50 times more prominent compared to normal controls (18).

The esophageal epithelium is the only GI segment devoid of eosinophils. Therefore any eosinophilia is pathologic. The epithelium of the esophagus is squamous but not keratinized and therefore the esophageal lining is directly exposed to esophageal contents as well as their antigens. Therefore the esophageal epithelium is though to play a role in the induction of esophageal inflammation (19).

Cytokines involved in the Th-2 response are mainly IL-5 and IL-13. IL-5 is the most specific cytokine for the eosinophil. It is involved in the growth, differentiation, activation, survival and maturation of the cell and has been show to be overexpressed in esophageal biopsies of patients with EoE. Interestingly, studies have also shown that mice deficient in IL-5 do not produce an esophageal response to antigens, thus proving that IL-5 is critical in the development of the disease. IL-13 is a more no-specific cytokine that is more involved with the systemic aspect of atopic disease. Overexpression of IL-13 leads to eosinophilia, mucous hyper production, and airway hyper responsiveness, and has been implicated in the development of asthma, atopic dermatitis, and allergic rhinitis. Again, studies looking at patients treated with intratracheal IL-13 show inflammation of both the lungs and esophagus (19).

Once all the above players have been assembled it is up to the mast cells and eosinophils to produce their effects locally. Mast cells represent the IgE mediated pathway of disease with preformed antibodies and eosinophils represent the non-IgE cell mediated response, both of which are implicated in the disease process. Both cells release lipid mediators that induce smooth muscle contraction, vascular permeability, and mucous secretion, but they also release leukotrienes and TGF-β which lead to the recruitment of inflammatory cells and tissue fibrosis. Below is a schematic describing the pathogenesis of the disease process in Figure 1 (19).
Figure 1. This figure shows the pathogenesis of EoE. First allergens are engulfed by antigen presenting cells (APC’s) near the esophageal lumen or skin. The APC then presents the antigen to a T cell which then goes on to induce both IgE mediated and Th-2 cell mediated pathways. B cells that have been activated produce preformed antibodies that attach to mast cells through the IgE mediated pathway. T cells that have been activated produce cytokines that attract eosinophils to the esophageal lumen through the non-IgE mediated pathway. When an antigen is encountered both eosinophils and mast cells degranulate, increasing inflammation and vascular permeability, thus increasing the influx of other inflammatory cells. Once the inflammatory reaction is present eotaxin-3 and TGF-β produce epithelial hyperplasia and fibrosis and the reaction continues until an intervention is made. Mishra A. Mechanism of eosinophilic esophagitis. Immunol Allergy Clin North Am 2009; 29(1): 29-40

Making the Diagnosis

At this point we have talked a lot about the disease process, but have neglected the disease findings. For patients that come to the office complaining of dysphagia, the differential diagnosis can be vast. Allergy and reflux are often diagnoses that come to mind first, but it is entirely possible for the problem to be infectious. Candida, CMV, HSV, parasites, and bacteria may all cause similar complaints. Autoimmune disease such as celiac, inflammatory bowel disease, scleroderma, and autoimmune enteropathy are also possibilities. Therefore it is important to keep an open mind especially for patients complaining of residual disease despite adequate therapy.

Pointed historical questions are important since patients with EoE have very specific complaints. If you suspect EoE you should elucidate if there is any food avoidance, if they eat very slowly, if they chew excessively, if they have to drink water after each bite of food, or if they have other gastrointestinal complaints to suggest another etiology. Although physical exam is important in all patients it is likely going to be very benign in the patient who has EoE or other esophageal disorders. The esophagus is buried deep in the neck and chest and therefore we rely on endoscopy to actually appreciate any pathology.
In the consensus statement on EoE from 2007, many recommendations were made regarding the ancillary tests used to evaluate patients with EoE (9). Below these tests will be discussed individually.

1. Laboratory testing has been evaluated in its ability to identify severity or progression of disease. Peripheral eosinophilia has been found in approximately 5-50% of patients and IgE levels are elevated in up to 70% of patients with eosinophilia (20). Although these numbers are impressive, remember that many patients with EoE have other atopic diseases and good reasons to have elevated eosinophil and IgE levels. Therefore it is recommended that neither test be used to diagnose or predict progression of the disease.

2. pH studies are required for the diagnosis of EoE unless a patient has been treated with 6 weeks of PPI therapy in order to rule out GERD. Twenty studies have reported on patients with EoE who have had pH studies performed. In 228 adults 40% were studied with pH probes and 82% were normal. Of 223 children 78% were evaluated with pH probes and 90% were found to be normal (9). Therefore the recommendation is that pH testing is required only initially to rule out reflux disease and then only if the provider believes that reflux has developed later in the disease process.

3. Manometry has been reported in 10 studies of patients with EoE. The lower esophageal sphincter (LES) is normal in 66/77 patients however 10 have shown hypotensive LES and 1 was shown to have hypertensive LES. Peristaltic abnormalities were found in 30/77 patients but the majority show nonspecific peristaltic abnormalities. Of the 14 children that have been reported on, none have shown any abnormalities. Clearly this shows that manometry studies can show abnormalities in over half of the patients who were studied but it provides no diagnostic value and is therefore not required unless patients have specific complaints.

4. Upper gastrointestinal series with contrast have been shown to be discordant with endoscopy findings. Many times a stricture will be seen on plain films however this is often due to intermittent contraction of the esophagus appearing like a stricture. Nonetheless contrasted studies can help identify strictures, small caliber esophagus, malrotation, and hiatal hernia and are recommended as ancillary tests in pediatric patients who have a negative endoscopy to help identify other sources of the problem.

Because these other tests are not recommended for routine evaluation of the dysphagia patient, and because physical exam is not reliable as a diagnostic tool, we rely on endoscopic findings. Unfortunately there are no “cardinal signs” of EoE (21). Thus making the diagnosis with endoscopy alone is difficult and this is why we rely on biopsy specimens to show us what is going on beneath the surface.

Common finding on endoscopic examination can include: crepe paper mucosa, friability, shearing, edema, white plaques, linear furrowing, small caliber esophagus, concentric mucosal rings, and strictures. Thirty percent of children and 9% of adults will have a visually normal endoscopy (22,23). The normal endoscopic exam will show smooth mucosa, prominent submucosal vessels, and a whitish pink color. Linear furrowing will be present in 25 to 100% of patients and loss of vascularity occurs in about 93% of patients. White exudates can be challenging to determine whether or not they are due to microabscesses of eosinophils, or if they are candida infections. The sensitivity of white exudates is only 30-50% for EoE, however the specificity is 95% (21). It is important to differentiate
between GERD and EoE. GERD may present with linear furrowing similar to that seen in EoE however there will also likely be a distal component of ulceration near the gastroesophageal junction. Also, strictures in EoE tend to occur both proximally and distally whereas isolated distal strictures are more commonly seen in reflux.

As stated previously the need for biopsy is paramount. If the distal esophagus is the only place biopsied, nearly 20% of the time you will miss the diagnosis (24). In order to achieve a sensitivity of 10% you must take 4 biopsies in children and 5 biopsies in adults (24,25). Therefore the recommendation is to take 4 biopsies distally (5cm above GE junction) and 4 biopsies proximally (10cm above distal site), in order to have adequate samples. It is important to remember that biopsies of the stomach and duodenum are also required as part of the diagnostic criteria for EoE.

After biopsies are performed there are several histologic findings that may be seen under standard H&E staining. Basal layer thickening and increase rete papillae will be present as will the classic >15 eos/hpf. Eosinophils are often scattered superficially in the esophageal epithelium and microabscesses can form when there are more than 4 eosinophils clustered together at the surface. These microabscesses produce the white exudate seen on endoscopy. Finally eosinophilic degranulation can occasionally be seen as well.

The Role of Allergy Testing

It has been shown that allergy is involved in the pathogenesis of EoE. As any allergist worth their salt will tell you, avoidance is the key to any successful anti-allergy regimen. There are two types of testing commonly performed in patients with EoE: skin prick testing (IgE mediated) and atopy patch testing (non-IgE mediated). Skin prick testing is standardized and has been show to produce very consistent results. Atopy patchy testing is much more variable in it preparations and methodologies but because it is able to associate Th-2 type responses, it is helpful in the evaluation of EoE patients.

A study by Penfield et al looked at allergy testing in a group of patients with EoE. 50% of patients testing had ≥1 food allergy, 93% of those tested had ≥1 aeroallergen, and overall 81% of patients had ≥1 allergy (26). The most common allergic foods were peanut, egg white, soy, cow’s milk, and tree nuts.

Complications of Disease

EoE is a chronic disease that progresses to tissue remodeling and fibrosis within the esophagus. Therefore many structural changes can lead to complications. Strictures for instance are present in a bout 57% of patients on endoscopy, however they are much less common in children at only 6% (6). Because strictures form narrow regions within the esophagus they predispose patients to the ever dreaded food impaction. Amazingly, food impaction occurs in nearly 60% of patients with EoE and about 50% of patients with food impaction will eventually be diagnosed as having EoE (1). Food impaction is characterized by retrosternal discomfort, delayed passage of the bolus, and followed by hypersalivation. Meats and dry rice are often the culprits, and obstruction can persist for hours.

Secondary reflux is a common problem as chronic inflammation can lead to hypotension of the LES. Remedios looked at 26 patients with chronic EoE and found that 10/26 had reflux on pH probe testing and 8/10 showed reduced LES pressures on manometric studies (27). Therefore it is important
to consider that although initially EoE patients are reflux free, they may eventually become symptomatic and require therapy.

Candida infections are a common complication of steroid therapy but can also occur spontaneously in EoE. It is important to get biopsies of the esophageal mucosa to decipher infection from eosinophil microabscess.

Finally, the most feared complication is esophageal rupture or Borhaave’s disease. Three cases have been reported in the literature and all have involved severe/prolonged retching and vomiting during a gastrointestinal infection or impacted food bolus (1). Although this is an uncommon complication it is one to be aware of due to its dreaded consequences.

**Treatment**

Because of the multifactorial process of EoE, providers have attempted several therapies for the disease process. Dietary modifications, steroids, anti-inflammatory drugs, and supportive therapies have all been evaluated and several novel therapies are still under evaluation.

Although there has been no randomized control trial looking at the effects of an elemental diet on EoE, several case reports have been studied. Case series data have shown 92-98% response rate to elemental diets both symptomatically and histologically. Patients are usually treated for 6 weeks with reintroduction of the least antigenic foods first (vegetables and fruits) and going to higher protein foods last. Foods are reintroduced with about 1 new food per week and patients are re-evaluated with endoscopy and biopsy after every 5-7 new foods to look for recurrent disease (28). This process can be very psychosocially daunting and is often very difficult to follow. Because these diets are so difficult to follow others have attempted elimination diets where the most antigenic foods are removed from the diet rather than all food altogether. Dairy, soy, egg, wheat, beef, peanut, and corn are the usual offenders. Reports have shown that 74% of patients will respond symptomatically and histologically to this diet (28).

Because this is an inflammatory condition steroids were often attempted as a first line agent. Prednisone at 1mg/kg/day up to a maximum of 60mg/day has been shown to improve both symptoms and histologic findings in 95% of patients, however upon stopping therapy, 90% of patients will show recurrence (28). Because of the long term consequences of systemic steroid therapy we consider this to be used only for acute exacerbations or for refractory cases. In contrast to systemic steroids topical steroids have been shown to be just as effective. In a randomized control trial of 80 patients on prednisone versus topical fluticasone for 4 weeks with an 8 week weaning period. Ninety-eight percent showed complete symptom relief and 94% showed complete histologic response with no significant differences between the groups studied. Forty-five percent of patients relapsed by the 24 week follow up just 12 weeks after weaning off the steroids (29). Therefore it is argued that topical steroids with long term management is the first line therapy and treatment of choice. Since this study budesonide has also shown promising results. Oral candidiasis has been found to occur in about 15% of patients treated with topical steroid therapy (29).

Anti-inflammatory drugs have been evaluated and cromolyn has not been shown to be an effective agent at all. Montelukast, a leukotriene inhibitor on the other hand, has been evaluated with
minimal success. Attwood et al. in 2008 looked at a small number of patients (n=8) and treated them with 100mg initially with a maintenance dose of 20-40mg daily for up to 14 months. All but one showed symptom relief but upon stopping the medication, 6/8 had recurrence of symptoms (30). In addition to small sample size, another weakness of this study was that they did not evaluate patients with repeat endoscopy and therefore eosinophilia/histologic response could not be evaluated.

Several novel therapies are currently under clinical trials. Mepolizumab is a monoclonal antibody against IL-5 and has shown some good results in early clinical studies. Anti-TNF-β has also been evaluated to try and decrease hyper vascularity and fibrosis. Both are awaiting further clinical trial data prior to large scale therapy, but will probably be reserved for refractory cases due to the expense of the medication.

Supportive therapy include stricture dilatation and food disimpaction as indicated. Stricture dilatation is looked at as a last resort when medical treatment has failed or patients develop a food impaction. There have been reports of esophageal tears or perforations upon instrumentation of the esophagus. In 2008 Schoepfer et al. looked at a small series of patients that were resistant to medical therapy. All patients experienced prompt relief after dilatation, with mild odynophagia post-procedure, no complications and sustained response for 6 months (31). A systematic review of all papers describing esophageal dilations was reported in 2010 looking at esophageal dilations in 468 patients with 671 dilations performed. Most authors reported mucosal tears but only one perforation was described leaving a perforation rate of 0.1% (32).

**Conclusion and Future**

EoE is a new disease that is becoming increasingly common in the westernized world. It is a diagnosis of exclusion, the pathogenesis is multifactorial, and the keys to diagnosis are history and endoscopy with biopsy. Much is still left to be learned about the disease however. The future requires that we answer questions about non-invasive markers of disease, long term management strategies, developing novel therapies, whether we treat the symptoms or the eosinophilia, and finally should we be empirically treating all patients with PPI’s. The past 10 years has brought many new ideas to the table and the next 10 will be just as important.

**DISCUSSION: HAROLD PINE, MD**

That was a nice review Dr. Coughlin and certainly one of those interesting problems that we need to be aware of in ENT. A couple of things strike me. Number one, all the great pictures you showed were clearly from flexible endoscopes with the ability of insufflation. It’s hard to get those nice views with the rigid endoscopes so commonly used in ENT practices. That said, more and more ENT surgeons are becoming proficient with the flexible TNE scope. (Transnasal esophagoscope). I encourage the residents to see out opportunities to learn this technique while in training.

Number two, based on the criteria you showed to make the diagnosis, you have to have biopsies from the stomach and duodenum which we’re not doing. We are down there in the esophagus and certainly are capable of doing esophageal biopsies but I get the sense that most ENT surgeons would not be doing the multiple level, multiple quadrant technique. Every once in a while during a microlaryngoscopy and bronchoscopy in children I’ll do a postcricoid biopsy, more to just look for changes of reflux versus this eosinophilia esophagitis. But the thing I wonder about, and it would
make a really nice prospective study, is why not for any patient that does come in with a food bolus stuck in their esophagus, as part of bringing them to the operating room, sign them up to do some biopsies to rule this out. If that statistic is right we’re going to have a pretty good hit rate and not only are we going to make them better, we can perhaps figure out why they got it in the first place.

I suppose the best take home point for me would be if you suspect this issue or are having kids with presumed reflux not getting better with medical management, refer to a gastroenterologist and perhaps an allergist as well.

REFERENCES


