implant and underwent successful repair. The remaining ten patients are continuing with the LES-EST; 6 have completed their 3 month and 3 have completed their 6 month evaluation. The median (IQR) off-PPI GORD-HRQL scores at baseline were 32 (25-38), which improved to 9 on EST at months 3, (n=6; p<0.001) and remained stable at 9 at month 6 (n=3; p=0.05). There was improvement compared to the on-PPI GERD-HRQL scores of 22 (26-9) at baseline. Patients median oesophageal acid exposure was 11.8% (8.8-15) at baseline and improved to 7.8% (2.4-12, n=6) % at 3 months and 7.3% (0.2-15.3; n=3) at 6 months. 91% (10/11) patient were able to discontinue PPI. Thirteen AEs including 1 SAE were reported in 4 patient; 9 related to the device or procedure, 7 non-serious AE were pain at the implant site and 1 was post-op nausea.

Conclusion: Preliminary results show that LOS-EST is effective in treating refractory GORD. There was a significant improvement in patient’s symptom, PPI usage and trend in improvement in their oesophageal pH. LOS-EST was safe with no GI or cardiac side effects. Long-term results in a larger group of patients are being collected to establish safety and efficacy of LOS-EST in refractory GORD.


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Methods: Data from the 24 hour ambulatory pH monitoring from 398 patients on proton pump inhibitor therapy for GERD symptoms were analyzed using ambulatory pH monitoring. PPIs were taken once daily (n=92), twice daily (n=218), or in combination with H2RA (n=88). Data were recorded on the total percentage time of gastric pH less than 4, the total percentage time of esophageal acid exposure, the upright percentage time of gastric pH less than 4 and upright percentage time of esophageal acid exposure, the recumbent percentage time of gastric pH less than 4 and the recumbent time of esophageal acid exposure. pH electrodes were located 5 cm above the lower esophageal sphincter and 10 cm below the lower esophageal sphincter. The data for individual patients was analyzed and plotted for total, recumbent and upright to determine if there was a correlation between gastric acid control and esophageal acid exposure.

Results: Results of the analyses of all groups showed that there was a relatively weak (r=0.38, 0.33, 0.34) although significant (p<0.0001) correlation between intragastric pH < 4 and esophageal acid exposure, whether upright, recumbent, or throughout the duration of the testing.

Conclusion: The results of this analysis suggest that control of gastric acid as a primary goal for the control of gastroesophageal reflux disease is not sufficient for all patients. The lack of a strong correlation between gastric pH < 4 and esophageal acid exposure underscores the fact that reflux is a multifaceted problem.

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Tryptase: A New Measure (or Predictor) of the Extent of GI Involvement in Eosinophilic Esophagitis (EoE)?

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Purpose: Several investigators have noticed increased levels of tryptase positive mast cells in the esophagi of patients with EoE. Serum tryptase is elevated in patients suffering from anaphylaxis and mastocytosis, but has not been reported in EoE patients. Since elevations in tryptase levels have been shown to be predictive of the severity of allergic reactions, we measured baseline tryptase levels in a cohort of patients with EoE and observed whether elevated levels correlated with disease extent and severity.

Methods: Retrospective study involving 46 patients (27 male, 19 female) diagnosed with eosinophilic esophagitis on the basis of biopsy. Additionally, 8 patients in this cohort were eventually found to have eosinophils on gastric and small bowel biopsies. The tryptase levels (ImmunoCap; Phadia) were available for review in 42 of the patients. Univariate analysis was performed to compare tryptase levels with associated symptoms and findings on chart review.

Results: 8 out of 42 patients (19%) had elevated tryptase levels (> 10.4 serum value). The mean tryptase level was 7.7±4.42. The mean peripheral blood eosinophil count was 481±295. On univariate analysis, 5 of the 8 (63%) of those with elevated tryptase had evidence of eosinophilic gastroenteritis as compared to 6 out of 34 (11%) with normal tryptase levels (p=0.02). The mean tryptase level for patients with eosinophilic gastroenteritis was 10.3 with a standard deviation of 6.5. There were no differences in reported seasonal or food allergies between those with elevated tryptase as compared to normal tryptase values (p=NS). There were significant differences in reported abdominal pain in patients with elevated tryptase levels (p=0.01). However, no differences in cramping (38% versus 12%) or loose stools (63% versus 26%) occurred in patients with elevated versus normal tryptase. In those with elevated tryptase, resolution of eosinophilic gastroenteritis correlated with normalization of the serum tryptase.

Conclusion: The role of tryptase in patients with EoE and food allergies is not completely understood but significant elevations in serum tryptase may indicate more diffuse disease such as eosinophilic gastroenteritis. Patients with elevated levels also appeared to have more abdominal pain. Gastric and small bowel biopsies should be performed in EoE patients with elevated serum tryptase as such testing may reveal more diffuse involvement of gastrointestinal eosinophilia and tryptase levels may serve as a non invasive marker to assess response to therapy.