

The Lower Esophageal Sphincter: A New Paradigm in Gastroesophageal Reflux Disease

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DESCRIPTION

Gastroesophageal Reflux Disease (GERD) is a digestive disorder that affects the lower esophageal sphincter muscle which is present in between esophagus and stomach. This is a chronic disease that occurs when stomach acid or bile flows into the food pipe and irritates the lining. *Helicobacter pylori* infection plays a vital role in the pathogenesis of GERD and other digestive disorders as it is the most common chronic bacterial pathogen in humans. Acid reflux and heartburn occurring more than twice a week may indicate GERD.

Other symptoms include dysphagia, odynophagia, regurgitation, belching, cough, wheezing, hoarseness, sore throat, globus sensation, epigastric pain, non-cardiac chest pain, etc.

Pathophysiology

Decreased lower esophageal sphincter pressure: The primary barrier to gastroesophageal reflux is the Lower Esophageal Sphincter (LES). It normally works in conjunction with the diaphragm. If barrier gets disrupted, acid goes from stomach to esophagus. It may be due to spontaneous transient LES relaxations and transient increase in intra-abdominal pressure.

Disruption of anatomical barriers: It is associated with hiatal hernia. The size of hiatal hernia is proportional to the frequency of LES relaxations. Hypotensive LES pressures and large hiatal hernia leads to more chances of GERD following abrupt increase in intra-abdominal pressure.

Esophageal clearance: The Gastrointestinal (GI) acid produced will be in contact with the esophageal mucosa for long time. Normally swallowing contributes to esophageal clearance by increasing salivary flow. Saliva decreases with increasing age, so more often seen in elder people.

Mucosal resistance: The mucus secreted by the mucus secreting glands involves in the protection of esophagus. The bicarbonates which are moving from the blood to lumen can neutralize acidic refluxate in the esophagus. Due to some defect in normal mucosal defenses, hydrogen ions diffuse into the mucosa leading

to cellular acidification and necrosis leading to esophagitis.

Delayed gastric emptying: An increase in gastric volume may increase both the frequency of reflux and the amount of gastric fluid available to be refluxed. Physiologic postprandial gastroesophageal reflux occurs.

Diagnosis

The diagnostic tests for GERD include barium swallow test, endoscopy, ambulatory pH monitoring and impedance pH monitoring, esophageal manometry.

Barium swallow test: An esophagram or barium swallow is an x-ray imaging test used to visualize the structures of the esophagus. The patient swallows barium while x-ray images are obtained. The barium fills and then coats the lining of the esophagus which diagnoses the anatomical abnormalities such as tumours. It is first performed in patients suffering with dysphagia.

Endoscopy: Upper endoscopy is also known as Esophago-Gastro-Duodenoscopy (EGD). It is a procedure in which a thin scope with a light and camera at its tip is used to look inside the upper digestive tract. Endoscopy with biopsy is needed for those patients who fail in a medication trail. This allows for detection, stratification and management of esophageal manifestations or complications of GERD.

Treatment

Over the counter acid suppressants and antacids are appropriate for initial therapy. Approximately 1/3rd of patients with heartburn related symptoms use antacids twice in a week. These are more effective than placebo in relieving GERD symptoms. Histamine H₂ receptor antagonists competitively block the histamine receptors in gastric parietal cells, and prevent the acid secretion. GERD can also be treated by using probiotics such as *Bifidobacterium bifidum*.

Proton pump inhibitors: These acts by decreasing the basal and stimulated gastric acid secretion through inhibition of the acid secretion by parietal cells i.e., H⁺/K⁺ ATPase proton pump.

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These are effective not only in patients having erosive esophagitis but also with non-erosive GERD who have moderate to severe symptoms.

CONCLUSION

GERD is the frequent entity in pediatrics, which is still under diagnosed. The initial drugs have been lately replaced by proton-

pump inhibitors as they are considered as the preferred medication which is able to determine reflux symptom disappearance. The evolution is favourable for the physiological GERD, while the pathological GERD may be complicated by peptic esophagitis, stenosis, upper digestive haemorrhage or barrett esophagus. GERD is a prognosis condition with complete and sometimes spontaneous healing.