Gastroesophageal Reflux, Eosinophilic Esophagitis, and Foreign Body

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KEYWORDS

• GERD • Foreign body • Chest pain • Pediatrics

Chest pain as a presenting symptom is more common in older children and is usually benign without a life-threatening cause. Infants, toddlers, and children with neurodevelopmental delays (NDD) are often unable to communicate clearly and may therefore present with nonspecific symptoms such as fussiness or other behavioral changes. The causes of chest pain include musculoskeletal, cardiovascular, pulmonary, gastrointestinal (GI), psychogenic, and idiopathic. Only about 0.2% to 0.6% of pediatric emergency room visits are for chest pain[1] and the most common cause (up to 60%) reported in children over 4-years-old is "idiopathic."[2] Prevalence of identified GI diagnosis as a cause of chest pain in children is low (5%–8%).[2,3] Although history and associated symptoms are helpful in revealing the underlying cause for the chest pain, it may often be misleading. As an example, even though epigastric tenderness associated with chest pain is usually indicative of a GI pathology,[4] exertion-associated chest pain does not necessarily rule out a GI cause.[5] It has, therefore, been suggested that children with chest pain be evaluated for upper GI disorders even if associated symptoms may or may not suggest a GI diagnosis.[6] The most consistent symptom of an esophageal disorder is pain localized alongside the course of the esophagus (retrosternal).[7]

In pediatrics, common esophageal causes of chest pain include eosinophilic esophagitis (EoE), gastroesophageal reflux disease (GERD), and motility disorders.[8,9] Accidental ingestion of foreign bodies that are lodged in the esophagus may also present with chest pain. In a study of 19 children, aged 10 to 17 years, complaining of substernal chest pain, 42% had replication of chest pain symptoms with acid infusion and 3 of these 8 patients exhibited abnormal esophageal motility during infusion. All of these patients showed excellent response to acid suppression.[10] This study underscores

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the fact that acid contact with the esophageal mucosa and abnormal esophageal motility can both cause chest pain.

GERD

Gastroesophageal reflux (GER) is a physiologic phenomenon that occurs at all ages but more frequently in infants. When this retrograde movement of gastric contents into the esophagus causes troublesome symptoms or complications it is referred to as GERD. The refluxed material may be air (belch), liquid, solid, or mixed; and, depending on the pH, may be acid (pH $\leq$4) or nonacid. The relative proportion of acid and nonacid reflux episodes was not known until the advent of the combined pH-impedance technology. This was mainly because the conventional pH probe study only measured episodes that were acid. We now know that nonacid reflux episodes are at least as frequent as acid episodes and are capable of producing symptoms as well.

The lower esophageal sphincter (LES) is the most important physiologic antireflux barrier. The LES is a high-pressure zone maintained by two muscular systems that help keep gastric contents from refluxing into the esophagus. The first is the circular (smooth) muscle layer of the lower end of the esophagus primarily innervated by the intrinsic enteric nervous system. The second is the sling (skeletal) muscle of the diaphragmatic crura that envelops this area and is supplied by the phrenic nerve as well as inhibitory (nitrenergic) motor fibers from the myenteric neurons. The LES is normally situated in the abdominal cavity below the diaphragm and the relative higher pressure in the intra-abdominal cavity over the intrathoracic pressure adds to the sphincter mechanism of the LES. Displacement of the LES into the thoracic cavity in a patient with hiatal hernia therefore disrupts proper functioning of the LES and predisposes to GERD.

Once the refluxed material enters the esophagus, the bolus causes distension of the wall and stimulates the receptors which induce a neurally mediated peristaltic contraction that moves the refluxate back into the stomach. This secondary peristalsis is often supplemented by a swallow-induced primary peristaltic wave that helps clear the esophagus. These clearance or “stripping” waves are an important mechanism for preventing damage to the esophageal mucosa from prolonged contact with the refluxed material. Another physiologic barrier to reflux-induced esophageal damage is reflex swallowing of alkaline saliva in response to a GER episode that helps neutralize acid content of the refluxate. GER-induced swallowing as well as clearance peristaltic waves are inhibited during sleep such that increased nocturnal reflux episodes tend to be more harmful to the mucosal lining.

The physiologic basis for most GER events is a phenomenon referred to as transient lower esophageal sphincter relaxation (TLESR).[11] This brief relaxation of the LES can be triggered by distension of the gastric fundus and is mediated through the vagus nerve.[11] TLESR can result in reflux of air (belch), liquid, solid, or mixed gastric contents into the esophagus. In infants, reflux episodes occur more often and are likely due to frequent feeds that distend the fundus causing more TLESR events. Anatomy of the stomach and gastroesophageal junction and the more frequent recumbent posture also predispose infants to more reflux episodes. When the refluxed material enters the short esophagus of an infant, it usually travels the entire length up to the pharynx and presents as regurgitation or vomiting. Full-column reflux episodes, and GER episodes in general, decrease as the child grows owing to esophageal lengthening and transition of the anatomy of the stomach and gastroesophageal junction to a more adult-like configuration.