Pediatric Gastroesophageal Reflux Disease

Regina F. Prusinski
Otterbein University, rprusinski@otterbein.edu

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn

Part of the Nursing Commons

Recommended Citation
Prusinski, Regina F., "Pediatric Gastroesophageal Reflux Disease" (2016). Nursing Student Class Projects (Formerly MSN). 199.
https://digitalcommons.otterbein.edu/stu_msn/199

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Nursing Student Class Projects (Formerly MSN) by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact digitalcommons07@otterbein.edu.
Pediatric Gastroesophageal Reflux Disease

Regina Prusinski
Otterbein University, Westerville, Ohio
Nationwide Children’s Hospital, Columbus, Ohio

Introduction

Gastroesophageal reflux is the return of stomach contents into the esophagus. It is normal in the newborn due to an immature gastroesophageal sphincter. Gastroesophageal reflux disease (GERD) is a common condition found in 33% of the pediatric population that occurs when the physiological barrier of the esophageal sphincter opens during a transient lower esophageal sphincter relaxation (TLESR) (Rimsa et al., 2016) resulting in complications like mucosal erosions, bleeding, dysphagia, or failure to thrive (Quitate, Ummatino, & Saiano, 2015). GERD in children can be directly related to the maturation of the gastroesophageal (GE) sphincter or an impaired hormonal or neural response. A high pressure gradient surrounding the GE sphincter aids in maintaining forward flow of food and stomach contents. When either the position of the sphincter or the thick mucosal lining of the GE sphincter are affected, GERD is likely to occur. Recurrent reflux results in inflammation of the esophageal epithelium or esophagitis. It has also been linked to reactive airway disease and otitis media with effusion (Gorecka-Tutega, Jastrzebska, & Skladzien, 2016).

Epidemiology and Etiology

- Infant reflux shows up in the first few months of life, peaks at four months, and resolves in almost all children by the age of 2.
- One out of 300 children will have reflux and associated complications, and it is the most common esophageal disorder for all pediatric patients.
- Symptoms in children are more likely to be chronic and decreased frequencies of symptoms (Khan & Orenstein, 2016).

Physiologic GER is the regurgitation that occurs without effort or pain while pathologic GERD in infants and children have frequent and/or persistent symptoms that affect their nutritional or respiratory status.

Signs and Symptoms

Most clinical manifestations of GERD relate to the pathologic effects of acid found outside of the stomach. Symptoms of heartburn and regurgitation are the classic findings (Falk & Vivian, 2015). Infantile reflux happens most commonly after meals as simple regurgitation. Infants have regurgitation in the early years or complaints of abdominal pain as they age. Sleep has been found to be interrupted with or without obstructive sleep apnea when they have GER (Machado et al., 2016). Infants with chest pain presentation had a low prevalence of cardiac disorders, and should be evaluated by a pediatric gastroenterologist for GERD. Children can also have sleep disturbances and other behavioral symptoms. Some older children may have neck contortions or refuse food with GERD. This is called Sanderizer syndrome.

Respiratory symptoms are also age specific. Infants can present with obstructive sleep apnea, stridor or lower airway disease where reflux has worsened primary airway disease like laryngomalacia or bronchopulmonary dysplasia (Khan & Orenstein, 2016). Older children are more likely to have asthma or obstructive sleep apnea issues such as laryngitis or sinusitis related to GERD.

Pathophysiology

Multiple factors determine whether reflux occurs or not: duration of esophageal exposure to reflux episodes, causality of the reflux material, and the susceptibility of the esophageal tissue to harm (Khan & Orenstein, 2016). The lower esophageal sphincter (LES) is anatomically supported by the crura of the diaphragm and the gastroesophageal junction (GEJ). This is the valve-like mechanism that stop the return of gastric contents. When the LES is relaxed or when hiatal herniation prevents the LES from being propitiously pressurized, reflux is more likely to occur during events of strain. The length of a reflux episode is increased when the swallowing reflex is decreased, and if any disease state that results in defective esophageal peristalsis. The pathological disease state is cyclic. The more chronic the episodes of reflux are, the more likely esophageal peristalsis will be defective, and the greater the decrease in LES tone, and inflammation to the esophagus shortens its structure and induces hiatal herniation (Khan & Orenstein, 2016).

1. TLESR is the mechanism that allows reflux to happen. It is described as the simultaneous relaxation of both the LES and the surrounding crura, a pressure drop of up to 2 mm Hg and lasts up to a minute in duration. The lower esophageal sphincter (LES) is anatomically supported by the crura of the diaphragm and the gastroesophageal junction (GEJ). This with the valve-like mechanism that stop the return of gastric contents. When the LES is relaxed or when hiatal herniation prevents the LES from being propitiously pressurized, reflux is more likely to occur during events of strain. The length of a reflux episode is increased when the swallowing reflex is decreased, and if any disease state that results in defective esophageal peristalsis. The pathological disease state is cyclic. The more chronic the episodes of reflux are, the more likely esophageal peristalsis will be defective, and the greater the decrease in LES tone, and inflammation to the esophagus shortens its structure and induces hiatal herniation (Khan & Orenstein, 2016).

2. TLESR is the mechanism that allows reflux to happen. It is described as the simultaneous relaxation of both the LES and the surrounding crura, a pressure drop of up to 2 mm Hg and lasts up to a minute in duration. The lower esophageal sphincter (LES) is anatomically supported by the crura of the diaphragm and the gastroesophageal junction (GEJ). This with the valve-like mechanism that stop the return of gastric contents. When the LES is relaxed or when hiatal herniation prevents the LES from being propitiously pressurized, reflux is more likely to occur during events of strain. The length of a reflux episode is increased when the swallowing reflex is decreased, and if any disease state that results in defective esophageal peristalsis. The pathological disease state is cyclic. The more chronic the episodes of reflux are, the more likely esophageal peristalsis will be defective, and the greater the decrease in LES tone, and inflammation to the esophagus shortens its structure and induces hiatal herniation (Khan & Orenstein, 2016).

3. Infants with congenital disorders that require surgical management may have long-term sequelae, particularly GER. Though the majority of GERD diagnosis are made to patients who do not have a congenital gastrointestinal malformation, GER is most common in infants and children with gastrointestinal malformations (Mareglia, et al., 2015). Being mindful of this high risk population can improve the speed of diagnosis and better individualize care.

Treatment

Medications

- **Antacids:** most often used non-prescribed medication treatment for GERD. They act over the counter and with their acid neutralization action directly affect the pathophysiology of GERD.
- **Histamine-2 receptor antagonists (H2RAs):** inhibit histamine receptors on the gastric parietal cells. They are very safe for the pediatric population and work well for all age groups.
- **Proton pump inhibitors (PPIs):** block the hydrogen-potassium adenosine triphosphatase channels of the final common pathway in gastric acid secretion.

Complications

Children need larger doses on a dose per weight basis then adults. PPIs are used over GERD therapies (Khan & Orenstein, 2016). A recent systematic review found that proton pump inhibitors fail to improve symptoms or for dysphagia, oesophagitis, heartburn or pain and diarrhea (Falk & Vivian, 2015).

Surgery

Fundoplication may be necessary when GERD is intractable to medical management and complications of esophagitis, strictures, or risk for morbidity from chronic pulmonary disease is significant (Lightdale & Gremse, 2013; Khan & Orenstein, 2016). A gastropathy in combination for feeding and vomiting as impaired nutrition is a common complication (Falk & Vivian, 2015).

Diagnosis

Esophagitis, particularly prolonged or chronic esophagitis, can result in strictures located in the distal esophagus that can need repeated dilatations and ultimately fundoplication. Over time, metabolic changes in the esophageal tissue can lead to Barrett’s esophagus which is a precancerous to esophageal adenocarcinoma. This is rare in children, but progressive Barrett lesions should be monitored closely regardless of the patients age (Khan & Orenstein, 2016). Complications can start with infants failure to thrive (FTT) and progress FTT may be directly caused by the lack of calories due to frequent regurgitation. As many as half of infants in cases are attributed to GERD and improve with treatment of GERD. Apparent life-threatening events (ALTE) caused by GERD are obstructive from a laryngospasm. Current evidence shows that GERD is not causal for ALTEs. Infants who already have laryngomalacia or micrognathia have stridor under GERD conditions (Khan & Orenstein, 2016).

Nursing Implications

Pediatric feeding complications like GERD can lead to serious complications and longstanding issues. Only by working as an interdisciplinary team can the patient best be treated. As in all pediatric patients, nutrition is closely monitored and growth is followed to monitor for short stature or inadequate weight gain.

References


