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The surgical treatment of gastro-esophageal reflux in neonates and infants

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Gastro-esophageal reflux (GER) is a physiological process characterized by the involuntary passage of gastric contents into the lower esophagus not induced by noxious stimuli. It represents a common condition in preterm infants and may occur in healthy neonates. The phenomenon is only considered as GER *disease* when it causes the patient to be symptomatic or results in pathological complications. Fundoplication is recommended in symptomatic neonates and infants with GER that does not respond to medical treatment. The presence of respiratory symptoms related to GER is the primary indication for fundoplication in this selected population. The Nissen fundoplication is the antireflux procedure of choice and the experience concerning other procedures, including laparoscopic techniques, is limited in this age group. The best results are achieved in newborn infants with isolated GER, as the recurrence rate of GER in infants with associated anomalies is high. Further studies are necessary to evaluate the benefit of laparoscopic fundoplication in this age group.

Gastro-esophageal reflux (GER) is a physiological process characterized by the involuntary passage of gastric contents into the lower esophagus not induced by noxious stimuli. The phenomenon is only considered as GER *disease* when it causes the patient to be symptomatic or results in pathological complications. GER represents a common condition in preterm infants and may occur in healthy neonates. In the former, the incidence of GER can be as high as 85%[1] with male to female preponderance of 1.6.[2] In the majority of cases GER resolves spontaneously, with its prevalence decreasing to 18% in childhood.[3] The incidence of GER is highest in neurologically impaired children (70%), who comprise 44 to 67% of children undergoing antireflux surgery.[4-6]

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The newborn infant with GER typically presents with vomiting or feeding intolerance, aggravated at night during the supine position. If GER is left untreated, failure to thrive from calorie deprivation may ensue. Reflux of gastric contents into the airways may result in coughing and choking, and chronic aspiration may cause the infant to present with complications of GER, including laryngospasm with apnoic and bradycardia spells (particularly during sleep), stridor or pneumonia.

Antireflux surgery is required in the neonatal period and during early infancy in the presence of severe GER-related symptoms that persist despite medical treatment. Although several reports have documented the effectiveness of fundoplication in older children and adolescents,[7-10] few studies have evaluated the results of fundoplication in infancy and the benefits of surgery in this population are not clearly defined. This article will focus on the surgical treatment of GER in neonates and infants. We have reviewed the liter-

ature on the topic and appraised the results of surgery for GER in these age groups.

Pathophysiology

A number of physiological and anatomical factors normally contribute to prevent chronic reflux of gastric contents into the lower esophagus. The combination of esophageal motility and gravity facilitates esophageal clearance of refluxed material as well as of saliva, which is rich in bicarbonate that coats the esophagus. These esophageal clearance mechanisms are usually developed by 31 weeks gestation.[11] Other physiological barriers to GER include antral contractions facilitating gastric emptying, and the production of mucous, prostaglandin and epithelial growth factors, which help to prevent damage to the esophageal mucosa.

Anatomically, the length of the intraabdominal esophagus, the phreno-esophageal ligaments, the gastric mucosal "rosette," and the esophageal hiatus, which is a sling formed by the crura of the diaphragm causing a pinchcock effect, all contribute to a higher-pressure zone in the lower esophagus. This high-pressure zone forms the lower esophageal sphincter (LES), a physiological rather than a true anatomical sphincter. Pressures at this gastro-esophageal junction (10-30 mmHg) are greater than gastric luminal pressure (5 mmHg), thereby preventing retrograde passage of gastric contents. In addition, the acute angle of His (made by the esophagus and the axis of the stomach) and the above physiological factors cumulatively contribute to limit the volume and frequency of gastric contents refluxing into the lower esophagus. Much of these anatomical features however are poorly developed in the first weeks of an infant's life, predisposing it to the higher risk of GER within this period. For instance the angle of His is obtuse in newborns and only decreases as the infant grows while the length of intraabdominal esophagus is shorter, only 1 cm at birth, compared with 3 cm by 3 months of age. Other abnormalities that predispose to GER include disruption of the gastro-esophageal junction (with resulting hiatus hernia), weakness or incompetence of the LES, and poor clearance of acid from the esophagus.[12]

A mean intraabdominal pressure of less than 10 mmHg is necessary for the LES to remain competent. GER is made more likely in groups with raised intraabdominal pressure, eg, following repair of omphalocele (43%),[13] congenital diaphragmatic hernia[14] and chronic respiratory infections.

Previous studies indicated that GER is a temporary condition and that symptoms resolve spontaneously without medical intervention.[15-17] Indeed, physiological antireflux mechanisms, such as increasing length of the intraabdominal esophagus and maturation of the LES, occur in the first few months after birth.[18] However, some neonates and infants develop serious symptoms related to GER.

These symptoms are more frequent in patients with neurological disorders[8-10,19-23] and following repair of esophageal atresia and/or tracheo-esophageal fistula.[19,21,24-27] Neurologically impaired patients have the highest incidence of GER (65-70%).[28] This is due to a combination of poor esophageal and gastric motility (due to vagal nerve dysfunction), chronic supine positioning, abdominal spasticity, diaphragmatic flaccidity, scoliosis, retching and increased use of gastrostomy for feeding. GER occurs in 30 to 80% of children treated for esophageal atresia, the incidence being related to the length of the atresia gap. The GER is attributed partly to poor esophageal motility in these patients and partly to a shortened esophagus. The shortened esophagus, from the original anomaly and compounded by the surgical repair, results in upward displacement of the gastro-esophageal junction.

Insertion of gastrostomy tubes has been reported to be associated with the development of or worsening of preexisting GER. The gastrostomy, which fixes the stomach to the anterior abdominal wall, potentially opens the angle of His[29] and lowers the LES pressure[30] thereby predisposing to GER.

Diagnosis

Investigations used for diagnosis of GER in newborn infants include 24-hour pH monitoring, upper gastro-intestinal contrast studies and esophagoscopy. Gastric isotope scintiscan and esophageal manometry are rarely used in neonates. The 24-hour pH monitoring is currently the most sensitive and specific test available for diagnosing GER. Monitoring is performed for 24 continuous hours, during which time the patient is only fed breast-milk, formula or apple juice. The latter is preferable as the alkaline content of milk feeds may neutralize the gastric acid reflux and thereby potentially produce a false negative result. Acid reflux is defined by pH < 4.0 in the lower esophagus. Esophageal exposure to gastric acid is assessed in terms of the cumulative time during which the esophageal pH is below 4.0, expressed as the percentage of the total 24 hours. A positive test for GER is indicated by a pH below 4.0 for more than 5% of the duration of the study. Upper gastro-intestinal contrast studies may diagnose active episodes of GER. However, they are more useful for detecting anatomical abnormalities, eg, hiatus hernia, stricture, esophageal motility, and may rule out the presence of malrotation of the bowel or gastric outlet obstruction as a cause of vomiting. Esophagoscopy allows visualization of the gastro-esophageal mucosa. However, only 40% of GER will demonstrate unequivocal esophagitis. Endoscopy is therefore a poor tool for diagnosis of GER, and is more useful in the assessment of complications of reflux, eg, esophagitis, stricture and in obtaining biopsies (eg, *Helicobacter pylori* infections, development of Barrett's esophagus). The presence of lipid laden alveolar macrophages in tracheal aspirates/broncho-alveolar lavage may indicate aspiration secondary to GER. However, its sensitivity and specificity for detecting GER is as low as 38% and 59%, respectively,[31]