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Hiatal and paraesophageal hernia repair in pediatric patients

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ABSTRACT

Hiatal and paraesophageal hernia (HH/PEH) can be congenital, resulting from embryologic abnormalities/genetic predisposition, or acquired, most commonly after gastroesophageal surgery such as fundoplication. Minimizing circumferential esophageal dissection at the time of Nissen fundoplication has been shown to decrease the risk of acquired HH/PEH from 36.5% to 12.2%. Gastrointestinal, respiratory, and constitutional symptoms, including anemia and failure to thrive, are common with high rates of associated gastroesophageal reflux. Chest x-ray is often abnormal and upper GI confirms the diagnosis. Treatment is surgical with the goal of reducing the hernia contents, excising the hernia sac, closing the crura, and performing an antireflux procedure. The laparoscopic approach is safe and effective.

Introduction

In 1853 review of 88 postmortem cases of diaphragmatic hernia, Henry Ingersoll Bowditch noted three cases wherein there was dilation of the esophageal opening and described what is thought to be the first report of a type II paraesophageal hernia.[1] A hiatal hernia (HH), also known as a type I or sliding hernia, occurs when the gastroesophageal junction (GEJ) abnormally protrudes cephalad into the chest through the esophageal hiatus. Types II–IVn HHs are known as paraesophageal hernias (PEH) and are the least common congenital diaphragmatic hernias. A type II PEH occurs when the gastric fundus herniates through the esophageal hiatus alongside the lower thoracic esophagus while the GEJ remains in the appropriate intra-abdominal location. Type III PEHs represent a combination of types I and II with an intrathoracic position of the GEJ and herniation of the gastric fundus. The term giant PEH is used when $>30\%$ of the stomach is herniated into the chest. Type IV PEHs involve herniation of other intra-abdominal contents, in addition to the stomach, such as colon, small intestine, spleen, or omentum.[2] This review details the historical perspective, epidemiology, presentation, diagnosis, treatment and outcomes of HH/PEH in the pediatric population.

History

Well before Bowditch's review in 1853, there were a number of descriptions of congenital and post-traumatic diaphragmatic hernias including those by Batista Morgagni and Vincent Alexander Bochdalek. However, it was not until 1900 that a HH was diagnosed by means of X-ray, first by Hirsch prior to autopsy and later by Eppinger in live patients.[1,3] Shortly thereafter, in 1925, Julius Friedenwald and Maurice Feldman correlated the symptom of heartburn to the presence of a HH.[4] The first report on elective surgical repair of HH was published by Angelo Soresi in 1919 detailing an abdominal approach, reduction of the hernia and closure of the hiatus for which he described “the suture has to close in the most perfect manner the opening of the diaphragm especially around the organs that pass through it, esophagus, vena cava, aorta, but without compressing the important organs just mentioned”—surgical principles that are still pertinent today.[5] The current surgical approach to the hiatus is overwhelmingly laparoscopic, a technique first described by Cuschieri et al.[6] The present day focus continues to be on the perfect manner by which to protect and close the hiatus with variations in degree of dissection, suture choice and placement, and mesh reinforcement of the crural closure.

Epidemiology

Etiology and embryology

HH/PEH are either congenital or acquired, the latter of which can be the post-operative consequence of gastroesophageal surgery or, less commonly, the result of traumatic injury. There are a number of embryological hypotheses as to why these hernias form; however, the true etiology is not fully understood. During development, the pneumoenteric recesses are two small coelomic spaces, which evolve on either side of the midline of the mediastinum, and persistence of the right recesses results in a flattened and elongated mesothelial lined space within the esophageal

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hiatus.[7,8] This space has been measured as approximately 1 cm at birth and likely obliterates during normal development into adulthood suggesting persistence of this right recess may predispose to HH/PEH development. Other hypotheses include laxity in gastric attachments, deficient diaphragmatic hiatus, and aberrant development of the lumbar component of the diaphragm origination from mesoderm cells around the aorta.[9–11]

Any surgical dissection at the esophageal hiatus resulting in disruption of the phrenoesophageal membrane can predispose to the development of HH/PEH. The most common pediatric procedure during which this could occur is fundoplication for gastroesophageal reflux, and the risk of acquired HH/PEH has been shown to be related to the extent of peri-esophageal dissection. A prospective randomized controlled trial during Nissen fundoplication comparing circumferential esophageal mobilization to minimal dissection leaving the phrenoesophageal membrane intact found a post-operative herniation rate at 1-year minimum follow-up of 22.7% for the circumferential dissection compared to 2.8% for the minimal dissection group.[12] The 5-year minimum follow-up study to the original trial showed an increase in post-operative hernia rates for both groups (36.5% for the circumferential dissection group versus 12.2% for the minimal dissection group).[13] The importance of minimizing esophageal dissection at the time of initial Nissen fundoplication to decrease the risk of post-operative, acquired HH/PEH cannot be over emphasized. HH/PEH has also been reported after surgical intervention for esophageal atresia and tracheoesophagel fistula.[14] As for post-traumatic HH/PEH, in his review of 216 adult cases, Marchand[15] determined 5% of hiatal hernias were the result of a traumatic incident, most commonly involving a crush injury, but there is no pediatric literature regarding acquired traumatic HH/PEH.

Incidence and patient population

In 370 patients who underwent laparoscopic Nissen fundoplication over a six year period, 8.1% required a second operation for repair of an acquired HH/PEH with fundoplication transmigration, and the incidence of post-operative herniation at 5-year follow-up from the prospective trial described above ranged from 12.2 to 36.5% depending on the extent of esophageal dissection performed.[13,16] The true incidence of congenital PEH, on the other hand, is unknown, but this diagnosis is felt to be uncommon. In the Montreal series of 14 patients of congenital PEH, type III PEHs were the most common at 57% versus 29% for type II and 14% for type IV PEH.[18] There have only been a few case reports of type IV PEH in the pediatric population.[18] A summary of the larger case series examining congenital PEH is shown in Table 1. There was no gender predilection from these studies, and patients were most often diagnosed within the first 2 years of life. Other congenital abnormalities including malrotation, trisomies, and Marfans syndrome have been associated with PEH.[9,18-20] In keeping with the embryological hypotheses above, the question of a familial mode of inheritance has also been raised as there have been a number of case series of HH/PEH within families suggesting a genetic predisposition.[9,20,21]

Clinical presentation

Patients with either congenital or acquired HH/PEH can be asymptomatic or, more commonly, present with a variety of gastrointestinal, respiratory, and/or constitutional symptoms. Not surprisingly, symptomatic gastroesophageal reflux was present in 50–68% of patients with PEH. Recurrent respiratory tract infections, emesis, anemia, and failure to thrive were other common presenting symptoms. In the Montreal series, emesis was the most common symptom for Type II PEH, while respiratory distress was most commonly seen in type III and IV PEHs, with failure to thrive and anemia also seen in type III. The feared complication of volvulus was reported in three of five patients in a series from Turkey. In all three of these patients, the volvulus was all organoaxial rotation where the greater curve of the stomach rotates horizontally along the longitudinal axis between the pylorus and the gastroesophageal junction.[2,19] Given the rarity of HH/PEH, clinicians must maintain an index of suspicion to proceed with radiographic evaluation in pediatric patients who present with these sometimes vague symptoms. Physical examination is often unrevealing, but one may hear borborygmi (bowel sounds) on auscultation of the chest.

Diagnostic evaluation

Evaluation often begins with an anteroposterior and lateral chest x-ray (CXR). One series reported seeing a cystic mass on all CXRs corresponding to the hernia diagnosis in all cases, while another series reported a correct presumptive diagnosis was only made in 29% of cases based on clinical symptoms and CXR alone.[11,18] Regardless, CXR is an appropriate first test to evaluate for other pulmonary pathology in addition to HH/PEH, the latter of which can appear as a cystic mass in the posterior mediastinum with an air-fluid level (Figure 1), or demonstrate esophageal dilation or abnormal intrathoracic location of a nasogastric tube.[11,19] The differential diagnosis of a fluid-filled posterior mediastinal cystic

Table 1
Summary of congenital paraesophageal hernia studies.

Author	Years (range)	No. of patients	Mean age at repair (range)	Surgical approach Lap = laparotomy Thor = thoractomy MIS = laparoscopy	Recurrence
Karpelowsky	1988–2013	59	23.4 mo (1 d–11 y)	Lap n = 56 (95%) Thor n = 3 (5%)	12/20 (60%) recurrent reflux Sx (no fundo group)
Yazici	1979–1999	19	32 mo (8 d-14 y)	Lap n = 12 (63%) Thor n = 4 (21%) Thor + Lap n = 3 (16%)	n = 2 (11%)
Yousef	1988–2013	14	35 d (0–500 d)	Lap n = 11 (79%) MIS n = 3 (21%)	n = 1 (7%)
Jetley	1997–2007	9	Unknown (8 d–3 mo)	Lap n = 8 (89%) Thor n = 1 (11%)	n = 2 (22%)
Al-Salem	1989–1997	6	1.3 y (2 d-2.5 y)	Lap n = 6 (100%)	n = 0
Imamoglu	1992–2004	5	7 mo (4–11 mo)	Lap n = 5 (100%)	n = 0