PEDIATRIC SURGERY *Update* ©
Vol. 48 No. 03 MARCH 2017

Petersen Hernia

Petersen hernia (PH) is a specific type of internal hernia where the small bowel migrates into the space between the caudal surface of the transverse mesocolon and the mesentery of the gastrojejunostomy limbs when either open or laparoscopic Roux-en-Y gastric bypass are performed for morbid obesity and biliopancreatic diversion. Clinical presentation is characterized by nonspecific symptoms of bowel obstruction such as postprandial abdominal pain, nausea and vomiting leading to delayed diagnosis and producing small bowel ischemia and even death. Some patients may have recurrent transient herniation and intermittent abdominal pain. The Petersen's space was initially described in 1900 as a space between the Roux limb and the transverse mesocolon formed after gastrectomy with Roux-en-Y reconstruction. Body weight loss is considered to be a risk factor for an internal hernia to develop. A greater loss of weight such as it occurs in bariatric surgery can induce an increase in the size of Petersen defect increasing the risk of an internal hernia. Antecolic reconstruction procedures may tend to specifically lead to Petersen hernia. Three types of Petersen hernia have been described: Type A involves the alimentary (Roux) limb, Type B involves the bilio-pancreatic limb and Type C involves the common channel. The diagnosis of Petersen hernia is confirmed using oral and intravenous contrast CT-Scan with findings of whirl sign, target sign, small bowel obstruction, clustered loop, retraction of the mesentery, congestion of mesenteric fat and vessels, mushroom sign, hurricane sign, small bowel behind SMA and right-sided anastomosis. The whirl signs of mesenteric fat or vessels have been reported to be the best single predictors of Petersen hernia with sensitivity of 80% and specificity of 90%. A high index of suspicion should be maintained to diagnose a Petersen hernia. Management is surgical reduction of the internal hernia on an emergency basis.

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Multiple Intestinal Atresias

Bowel atresia is a common cause of surgical intestinal obstruction in newborns. Most bowel atresia occurs in the jejunoileum and are single in nature. Pathogenesis of an intestinal atresia is a late intrauterine vascular accident in the mesentery causing loss and discontinuity of a segment of bowel. Bowel atresias are classified into: Type I: an intraluminal diaphragm with seromuscular continuity. Type II: cord-like segment between the bowel blinds ends. Type IIIA: atresia with complete separation of blind ends and V-shaped mesenteric defect. Type IIIB: jejunal atresia with extensive mesenteric defect and distal ileum acquiring its blood supply entirely from a single ileocolic artery. The distal bowel coils itself around the vessel, giving the appearance of an "apple peel"deformity. Type IV: multiple atresias. Multiple intestinal atresias (MIA) are the rarest of them all often associated with absence of significant bowel length resulting in short bowel syndrome. Though multiple anastomosis may suggest a higher risk of complications such as stricture or leak, they are the most effective treatment to preserve the maximum intestinal length in children with MIA. The proximal bowel can be amenable to tapering or the serial transverse enteroplasty (STEP) procedure. This proximal dilated bowel can be taken out as a jejunostomy. To accomplish the multiple segmental anastomosis in the affected bowel a soft silastic catheter can be used as stent of each anastomosis and exteriorize as a proximal mucous fistula while the distal end of the catheter can be brought out of the abdomen through the appendix. Intestinal continuity can be established at a later operation. There is a syndrome of hereditary MIA with multiple intestinal atresias from the stomach to the rectum in association with immune deficiency. The most common congenital malformation associated with MIA is Meckel diverticulum.

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First Branchial Cleft Anomaly

First branchial cleft anomalies (FBCA) are very rare frequently overlooked and mismanaged. First branchial cleft anomaly is the result of incomplete closure of the cleft formed in the development of the lower face and neck during the 4th to 7th weeks of human development. FBCA has a closed relationship to the parotid and facial nerve.

Anatomically two types of FBCA are described: Type 1 with defect in the parotid region, of ectodermal origin arising from duplication of the membranous external auditory canal appearing as sift cysts lined by squamous epithelium. Type 2 more commonly found in children is a defect in the anterior cervical triangle communicating with the external auditory canal, ectodermal and mesodermal in origin, containing skin with adnexal structures as well as cartilage. They present as cyst, sinus, fistula or combinations with opening in the region of the submental triangle. FBCA can present later in life. Recurrent and chronic otorrhea or otitis externa is the most frequent symptom. Other presentations include recurrent periauricular swelling, a sinus in the neck, sinus in external auditory meatus presenting with discharge or fistula below the angle of the mandible. Some are associated with a myringeal web, an epidermal structure that extends from the floor of the external auditory canal to the umbo of the tympanic membrane. Type 1 cysts can be removed via a retroauricular incision. Type 2 excision needs early identification of the facial nerve at the stylomastoid foramen or proximally in the temporal bone. Many cases present as an infected abscess in the region of Pochet's triangle where they are recurrently incised and drained sometimes not considering the diagnosis of a FBCA. CT Scan can confirm the diagnosis showing the wide tract near the external auditory canal. If there an opening sinus a fistulogram can be performed. Aim of management is complete removal of the lesion with preservation of the facial nerve. Recurrence occurs with infection, incomplete resection and non-curative interventions.

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ISSN 1089-7739