PEDIATRIC SURGERY *Update* ©
Vol 19 No 02 AUGUST 2002

Ovarian Hernia

Inguinal hernias are the most common surgical pathology seen in infants and children. A lump in the inguinal canal area of a newborn or infant female is most probably an ovarian incarcerated inguinal hernia. On very rare occasion the lump is a testis in a child with testicular feminization syndrome. Clinically the irreducible ovarian lump is usually asymptomatic, movable and non-tender mass within the labia majora. Ultrasound can determine the nature of the gonad present. The main problem with an ovarian hernia in infants is the incidence of ovarian torsion associated before repair. Ovaries trapped within inguinal hernias undergo torsion far more commonly than ovaries and tubes in the normal pelvic position increasing the chances of infarction. Torsion can occur at any time after diagnosis of the hernia. The incarcerated ovarian pedicle is narrowed and lengthened within the defect and the internal ring serves as a fixed point around which a twist can occur. The risk of torsion and infarction creates the view that ovarian hernias should be repaired at the earliest elective opportunity if they can be reduced manually. Children with edema, tenderness or skin discoloration in the inguinal area should be repaired immediately. Early recognition and management of this condition reduce the risk of gonadal infarction. During repair surgeons must be aware that in 20% of girls with inguinal hernia, the fallopian tubes occasionally with the ovary or uterus comprise the wall of the hernial sac (sliding component).

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Diversion Colitis

Diversion colitis is an inflammatory state resulting from a nutritional deficiency in the lumen of the colonic epithelium. The colitis develops in segments of the colon and rectum after surgical diversion of the fecal stream persisting indefinitely unless the excluded segment is reanastomosed. Diversion colitis is characterized by rectal discomfort, pain, discharge, tenesmus and bleeding. Symptoms occur three months or more after bowel diversion. Diagnosis is established by colonic or rectal biopsy. Histologic abnormalities included

aphthous ulcers, crypt distortion, atrophy and abscesses, a villous colonic surface, and a mixed acute and chronic inflammatory infiltrate with patchy lymphoid hyperplasia. This condition is caused by the absence of luminal short-chain fatty acids, the preferred metabolic substrates of colonic epithelium. In children diversion colitis can be seen in bowel derived Hirschsprung's disease, imperforate anus, ulcerative colitis and Crohn's disease. It has also been reported to also occurs after sigmoid neovagina reconstruction. Adjunctive management includes the use of a topical mixture of short-chain fatty acids (propionate, acetate, butyrate) and/or 5-Aminosalicylic acid (5-ASA) to control symptoms. Definitive treatment consists of excision of rectum or stomal closure.

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Duodenal Atresia

Duodenal atresia (DA) is the most common congenital anomaly associated with the duodenum and the most common atresia found in the GI tract. The double-bubble appearance of the dilated stomach and duodenal bulb seen prenatally (US) as two anechoic cysts or after birth (simple abdominal films) is characteristically diagnostic. DA is associated one-third of the time with Down's syndrome followed by cardiac malformations (20%). Prenatal karyotyping and fetal echocardiogram of suspected DA cases will establish the association. Clinically, the child with DA presents with bilious vomiting and epigastric distension (dilated stomach) depending whether the atresia occurs proximally (10%) or distally (90%) to the papilla of Vater. Bowel obstruction from DA needs urgent differentiation from malrotation which carries the risk of midgut volvulus. If in doubt a small upper GI series using a water soluble contrast is recommended. After correction of electrolytes imbalances, management of DA consists of diamond-shaped duodeno-duodenostomy. If the proximal duodenum is massively dilated a tapering duodenoplasty (imbrication or stapler resection) will help reduce the possibilities of a functional anastomotic obstruction. Late complications include motility disorders, megaduodenum, gastroesophageal reflux, duodenal-gastric reflux, gastritis, peptic ulcer disease, blind loop syndrome and biliary-pancreatic conditions which may be observed months to years after surgical management.

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* Edited by: **Humberto L. Lugo-Vicente, MD, FACS, FAAP**
Professor/Associate Director of Pediatric Surgery, University of Puerto Rico School of Medicine and University Pediatric Hospital, Rio Piedras, Puerto Rico.
Address: P.O. Box 10426, Caparra Heights Station, San Juan, Puerto Rico USA 00922-0426.
Tel (787)-786-3495 Fax (787)-720-6103 E-mail: *titolugo@coqui.net*
Internet: http://home.coqui.net/titolugo

ISSN 1089-7739