



PEDIATRIC SURGERY Update © **Vol. 48 No. 02 FEBRUARY 2017**

Barrett Metaplasia in Esophageal Atresia

The prevalence of gastroesophageal reflux is increased significantly in children born and managed for esophageal atresia (EA). Chronic untreated gastroesophageal reflux can lead to malnutrition, esophagitis, esophageal strictures and intestinal metaplasia of the esophagus epithelium known as Barrett esophagus (BE). BE is defined as a change in the esophageal epithelium of any length that can be recognized at endoscopy and is confirmed to have intestinal columnar metaplasia by biopsy. The gastric type of metaplasia resembles the epithelium found in the gastric fundus and cardia, whereas the intestinal type of metaplasia (which is also called specialized columnar epithelium) has goblet cells as seen in intestinal mucosa. BE associated with intestinal metaplasia is a well-known risk factor for development of adenocarcinoma of the esophagus with a 30- to 125-fold increase compared with the general population. Long term results have found that heartburn, dysphagia and retrosternal pain, symptoms of gastroesophageal reflux might be present in almost one-third of all children repaired of EA as infant. Dysphagia occurs due to impaired motility, esophagitis, and anastomotic or peptic structure formation. BE is rare in children without neurodevelopmental delay or tracheoesophageal anomalies like esophageal atresia. Duration of symptoms and/or age related effects are important risk factors for BE development. The lag time to developing metaplasia from the time of initial surgical correction is about ten years. Endoscopy and biopsies are the best way of detecting such mucosal changes. Recently prevalence rates of 42% for gastric metaplasia and 1% for intestinal metaplasia has been found in adolescent and young adults with repaired EA. Characteristics of these patients include peptic esophagitis, previous multiple antireflux surgery, type I atresia and esophageal dilatation. Nissen fundoplication was found not to prevent BE in EA patients. Systematic upper GI endoscopy and multistaged biopsies should be performed before the transition to adulthood in all patients with EA, even if asymptomatic. If no BE is found, endoscopy should be repeated every five to 10 years through adulthood.

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Intrapericardial Teratoma

Tumors of the heart and pericardium are rare causing a variety of cardiac and systemic symptoms depending on the size and anatomic location. Growth rate, friability and ability to invasiveness can determine clinical features and outcome. Intrapericardial teratoma is a very rare congenital tumor which can be diagnosed in-utero or soon after birth due to its association with massive pericardial effusion. Intrapericardial teratoma is a germ cell origin tumor composed of the three primitive germ layers, namely endoderm (gastric and intestinal mucosa), ectoderm (neuroglia) and mesoderm (bone, cartilage, fatty or fibrous tissue) arising from the pericardium. Patient age ranges from intrauterine life to adulthood with most cases occurring in infants. More than 75% of intrapericardial teratomas occur in children under the age of fifteen. Main clinical symptoms include respiratory distress, pericardial tamponade and cyanosis. Most are mature type teratomas followed by immature cases. They can be diagnosed intrauterine using prenatal ultrasound with findings of a large pericardial effusion and intrapericardial multilobulated and cystic mass with calcifications. Diagnosis is confirmed using MRI and fetal echocardiogram. In cases of fetal cardiac tamponade or hydrops intrauterine pericardiocentesis can be performed to permit near full-term birth. Intrapericardial teratomas are usually located in the right side of the heart causing displacement and left-side rotation. The arterial supply is from a pedicle to one of the great vessels or directly from the aorta. Surgical excision is the only effective management for intrapericardial teratoma. Since most tumors are benign the prognosis is usually good after resection. The presence of immature neuroepithelium carries a poor prognosis needing adjuvant radio- and chemotherapy. Surgical resection of the teratoma in the fetus through EXIT strategy or open fetal surgery is feasible if the tumor is growing fast and causing significant hemodynamic changes including hydrops or impending death.

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Tubo-ovarian Abscess

Tube-ovarian abscess (TOA) is a well known complication of pelvic inflammatory disease (PID) in young sexually-active women during reproductive years, including adolescents. TOA is an ascending infection from the cervix and/or vagina through the uterus to the fallopian tubes and ovaries. The infection is usually the result of sexually transmitted disease or after instrumentation of the female genital tract. Patients with TOA usually present with low abdominal/pelvic pain, vomiting and fever. Pelvic examination shows adnexal mass or tenderness. White blood cell count is elevated. The diagnosis is made with the help of US, CT-Scan or MRI. In occasion the diagnosis cannot be separated from symptoms of appendicitis. Appropriate management of PID complicated with a tubo-ovarian abscess requires prompt initiation of empiric broad spectrum antibiotics effective against both *Neisseria gonorrhoea* and *Chlamydia trachomatis*. In the majority of patient antibiotics is all the treatment that is needed. At least 24 hours of inpatient observation is recommended during therapy. It is estimated that almost 40% of women with TOA fail to respond within 48-72 hours of therapy needing drainage of the abscess either percutaneously or via laparoscopic approach. PID and TOA are extremely rare in non-sexually active or amenorrheic adolescent females. The etiology in such cases includes inflammatory bowel disease with hematogenous seeding of bacteria, recurrent urinary tract infection with urinary vaginal reflux, poor hygiene, obesity with vulvar adiposity and müllerian abnormalities. The organism most commonly identified in such cases of virginal adolescents is *Escherichia Coli*, alpha hemolytic streptococcus and *Pasteurella multocida*. Though most cases of TOA are due to PID, laparoscopy can elucidate the correct diagnosis in atypical cases such as virgins, postmenopausal or those that do not respond to antibiotherapy.

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