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Fetal Neuroblastoma

Routine use of prenatal sonography will increase the incidental diagnosis of fetal neuroblastoma. Most are detected during the third trimester of pregnancy as cystic/solid suprarenal mass. The tumor does not cross the placenta but can metastize in utero to the fetal liver or placenta. After birth 50% of babies have elevated HMA/VMA levels. Most enjoy improved survival due to: lower stage of disease, cystic variety (in -situ), and higher stage IV-S (which has been associated with spontaneous inmuno-regression. Adverse biologic features are: diploid tumor karyotype (cytometry) and amplify N-myc oncogene. They can be very difficult to differentiate from neonatal adrenal hemorrhage; T2 of MRI can be of help. Are they neuroblastoma in-situ, and will they regress spontaneously without treatment are question waiting answer in the near future.

Prenatal CCAM

Congenital cystic adenomatoid malformation is a lung bud lesion characterize by dysplasia of respiratory epithelium caused by overgrowth of distal bronchiolar tissue. Prenatally diagnosed CCAM prognosis depends on the size of the lung lesion and can cause: mediastinal shift, hypoplasia of normal lung tissue, polyhydramnios, and fetal hydrops (cardiovascular shunt). Classified in two types based on ultrasound findings: macrocystic (lobar, > 5 mm cysts, anechoic, favorable prognosis) and microcystic (diffuse, more solid, echogenic, lethal). Occurs as an isolated (sporadic) event with a low rate of recurrence. Survival depends on histology. Hydrops is caused by vena caval obstruction, heart compression and mediastinal shift. The natural history is that some will decrease in size, while others disappear. Should be follow with serial sonograms. Prenatal management for impending fetal hydrops has consisted of thoraco-amniotic shunts (dislodge, migrate and occlude), and intra-uterine fetal resection (technically feasible, reverses hydrops, allows lung growth). Post- natal management consist of lobectomy.

Fetal Intestinal Obstruction

The fetal gastrointestinal tract (foregut, midgut and hindgut) undergoes ventral

folding between 24-28 days' gestation. By the 5- 6th wk the stomach rotates to the right and the duodenum occludes by cell proliferation. Recanalization of the duodenum occurs around the 8th wk. The midgut rotation takes place during the 6-11th wk and the final peritoneal closure by 10th wk. The fetal GI tract begins ingestion and absorption of amniotic fluid by the 14th wk. This fluid contributes to 17% effective nutrition; proximally obstructed gut can cause growth retardation. Fetal intestinal obstruction is caused by: failure of recanalization (duodenal atresia), vascular accidents (intestinal atresias), intrauterine volvulus, intussusception, or intraluminal obstruction (meconium ileus). Esophageal obstruction causes polyhydramnios, absent visible stomach and is related to tracheo-esophageal anomalies. Duodenal obstruction seen as two anechoic cystic masses is associated to aneuploidy (trisomy 21) and polyhydramnios. Jejuno- ileal obstruction produces dilated anechoic (fluid-filled) serpentine masses and bowel diameter of 1-2 cm. Large bowel obstruction is most often caused by meconium ileus, Hirschsprung's disease or imperforate anus. The colon assumes a large diameter and the meconium is seen echogenic during sonography. In general the method of delivery is not changed by the intrauterine diagnosis of intestinal obstruction. Timing can be affected if there is evidence of worsening intestinal ischemia (early delivery recommended after fetal lung maturity).

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