

Basic Pathology: pp. 701-705

- What is the most common site for an ectopic pregnancy?
- How does a baby get a congenital aspiration pneumonia?
- Describe preclampsiaBclinical and pathologic findings.
- Which cells are responsible for the common epithelial tumors of the ovary?
- What is the most common ovarian carcinoma?
- What is the most common germ cell tumor?
- What serum markers are drawn for germ cell tumors of the ovary?

Ectopic pregnancy:

Extrauterine implantation occurs in as many as 1% of all pregnancies, may involve one of multiple sites: fallopian tube (most common), ovary, cervix, abdomen (very rare).

Ectopics associated with rupture, hemorrhage, constitute an obstetric emergency. Risk factors: previous pelvic inflammatory disease, tubal surgery, previous ectopic pregnancy.

Placental Infections:

Acute chorioamnionitis

Ascending bacterial infection. β - hemolytic strep. Coliforms. Vaginal flora. Associated with premature rupture of membranes, premature labor, and prematurity.

Path: neutrophils migrating through placental membranes. Baby gets infected by breathing in or swallowing infected amniotic fluid. Gets pneumonia, then sepsis. May be lethal in utero.

Chronic villitis: Hematogenous spread of infection from mother's blood stream.

TORCH infections (toxoplasmosis, other, rubella, cytomegalovirus, herpes virus).

Congenital CMV infection may be lethal. Common cause of hearing loss in US. Path: maternal lymphocytes and organisms in membranes/placental villi.

Pre-eclampsia:

Also called toxemia of pregnancy. Condition affecting young gravidas and older gravidas. Results in hypertension in pregnancy, proteinuria and edema. Involves 5-10% of pregnancies. May develop superimposed seizures-> then called eclampsia.

May involve liver, HELLP syndrome-hemolysis, elevated liver function tests, low platelets. May develop DIC (disseminated intravascular coagulation) and die of ischemic organ injuries. Decreased trophoblastic production of PGI₂, PGE₂, and nitric oxide (which normally help dilate maternal blood vessels) thus allowing hypertension to develop.

Associated with failure of normal trophoblastic invasion of uterine blood vessels which allows for maximal perfusion of the placenta by dilating the vessels and increasing blood flow. Thus, the placentas may be small, growth retarded, with infarcts. Path: decidual vasculopathy of maternal blood vessels narrows lumen -> decreased placental perfusion B> placental infarcts.

Placental abruption:

Premature separation of the placenta may be partial or complete. In complete abruption the placenta shears off and the fetus subsequently dies. In partial abruption, only a portion of the placenta becomes detached and if not extensive, fetal life continues. Abruptions may be overt, i.e. with vaginal bleeding; or concealed, i.e. without vaginal bleeding.

Abnormal placental adherence:

Normally, the mature placenta detaches from the uterine wall within 20 minutes of delivery of the infant. When the placenta does not detach, it is called placenta accreta. This means abnormal placental adherence. When the placental villi invade partially into the uterine wall it is called placenta increta, when thru the wall, placenta percreta. The latter two conditions usually require hysterectomy.

Gestational trophoblastic disease:

Complete hydatidiform mole: 46,XX or 46,XY all paternal chromosomes. Essentially, no fetus forms. Placental villi form with exuberant trophoblastic proliferation involving all 3 types of trophoblast: cytotrophoblast, intermediate trophoblast and syncytiotrophoblast. Tumor secretes β human chorionic gonadotropin (hCG) which serves as a tumor marker. Follow-up levels are assayed to determine efficacy of treatment. In the US, about 20-30% develop persistent gestational trophoblastic disease requiring chemotherapy. A very few of these patients develop choriocarcinoma, a highly malignant trophoblastic tumor which is exquisitely sensitive to chemotherapy.

Partial hydatidiform mole: 69 chromosomes, 2/3 paternal, 1/3 maternal. Abnormal placenta with some degree of trophoblastic proliferation. Forms a fetus which is malformed, occasionally making it well into the 2nd trimester or even the 3rd. Rarely progresses to persistent gestational trophoblastic disease.

Invasive mole: Complete hydatidiform mole invades into uterine wall. Requires chemotherapy for ablation.

Choriocarcinoma: A highly malignant tumor of cytotrophoblast and syncytiotrophoblast occurs in 1 in 30,000 pregnancies in US and in as many as 1 in 2,000 pregnancies in Asia and Africa. Increased risk in very young and in older women. Presents with vaginal bleeding or other organ involvement in disseminated disease. 50% follow complete mole, 25% follow abortion and the remainder follow normal pregnancy. Does not form placental villi. No fetus formed. Kills patient quickly if not given chemotherapy.