Placental and Gestational Disorders

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Modified from original lecture by Theresa M. Kristopaitis, MD
What Do You Need to Know?

• Per USMLE guides
  – Twinning
  – Preeclampsia – eclampsia
  – Abruptio placentae
  – Placenta accreta
  – Placenta previa
  – Ectopic pregnancy
  – Retained placental tissue
  – Gestational Trophoblastic Disease

• Key Points
  – Basic understanding of the placental anatomy
  – Placental cell types
  – FACT: there is no mixing of maternal/fetal blood.
  – Abnormal placentation - previa and accreta
  – Gestational trophoblastic disease
  – Complete versus incomplete mole

Placenta

• Function of the placenta?
  • Establish effective communication between mother and developing fetus
    – maintaining immune and genetic integrity of both individuals.
  • Allows intimate apposition of maternal and fetal circulations
    – exchange of nutrients, oxygen and waste products.
  • Secretes a variety of hormones
    – including human chorionic gonadotropin.
Placenta

- A membrane surrounds the developing fetus and forms the amniotic cavity
  - Derived from fetal tissue
  - Composed of two layers
    - amnion (inner layer)
      - Chorion (outer layer)
    - Chorion attaches to the decidual
  » decidual = endometrium of pregnancy

Chorionic Villi

- Chorionic Villi
  - Placenta composed of chorionic villi that sprout from the chorion to provide a large contact area between fetal and maternal circulations
- Central stroma
- Two layers of epithelium (trophoblast):
  - syncytiotrophoblast and cytotrophoblast

Circulation

Under normal circumstances maternal and fetal blood do not “mix”.
SPONTANEOUS ABORTION (MISCARRIAGE)

- Pregnancy loss before 20 weeks
- 1/3 of all pregnancies lost (10-15% of recognized pregnancies)
  - More than half due to chromosomal abnormalities
  - Defective implantation
  - Fetal abnormalities
  - Maternal causes
    - inflammation, uterine deformity, DM, luteal-phase defects
  - Unknown

Ectopic Pregnancy

Implantation occurs outside uterus
- 1:150 of pregnancies
- 90% in the fallopian tubes
  - 10% ovary and abdominal cavity
- Predisposing factors:
  - inflammation and scarring
  - intrauterine Device
  - Presentation – abdominal pain, acute abdomen
- Clinical complications: rupture or hemorrhage, high mortality unless removed surgically
Twin Placentas

- Dizygotic
  - Fertilization of 2 ova
- Monozygotic
  - Division of one fertilized ovum
- 3 types of twin placentas:
  - Diamnionic, dichorionic
  - Diamnionic, monochorionic
  - Monoamnionic, monochorionic
- Monochorionic placentas imply monozygotic (identical twins)
- Number of amnions determined by the time of splitting of the ovum

Access Medicine

What is TTTS – Twin to Twin Transfusion Syndrome?

Twin-to-twin transfusion syndrome (TTTS), also known as Feto-Fetal Transfusion Syndrome (FFTS) and Twin Oligohydramnios Polyhydramnios Sequence (TOPS), is a complication of monochorionic diamniotic (MCDA) pregnancies in which the presence of OLIGOHYDRAMNIOS in one sac and POLYHYDRAMNIOS in the other sac results from intratwin vascular connections within the placenta.

Placentation Abnormalities

A - Normal Placenta
B - Placenta Previa
C - Placenta Accreta
D - Abruptio Placenta
Placenta Previa

- Attachment of placenta to lower uterine segment or cervix
- Serious 3rd trimester bleeding
- Dilatation of cervix disrupts placenta

Placenta Accreta

- Partial or complete absence of decidua with adherence of placental villous tissue directly to myometrium
- Failure of placental separation
- Cause of postpartum bleeding
- Predisposing factors:
  - Placenta previa (60%)
  - Hx previous cesarean section

Placental villi

Uterine myometrium
Abruptio Placentae

• Premature separation of placenta prior to delivery
• Formation of retroplacental blood clot
  — Blood supply of oxygen and nutrients to fetus compromised to greater degree with increasing size of abruption
  — Painful maternal bleeding
  — Potential fetal death

Abruptio Placenta (Placental Abruption)

Abruptio Placentae
## Retained Placental Tissue

- May cause postpartum hemorrhage
  - Potential infection

## Preeclampsia-Eclampsia

“Systemic syndrome characterized by widespread maternal endothelial dysfunction presenting clinically with hypertension, edema and proteinuria during pregnancy”

### Demographics
- 3-5% pregnancies
- Usually last trimester
- More common with 1st pregnancy

## Pathogenesis

- Placenta plays key role
  - Symptoms rapidly disappear after delivery of placenta
  - Principal theories
    - Abnormal placental vasculature
    - Endothelial dysfunction and imbalance of angiogenic and anti-angiogenic factors
    - Coagulation abnormalities
Generalized (Multisystem) Process

- Liver: fibrin thrombi, hemorrhage, necrosis
- Kidney: fibrin in glomeruli and capillaries, renal cortical necrosis
- Brain: hemorrhage and thrombosis
- Heart and anterior pituitary
Placenta Morphology

- Malperfusion, ischemia, vascular injury
  - Infarcts
  - Retroplacental hematoma
  - Villous ischemia
  - Acute atherosis of uterine vessels
    - fibrinoid necrosis, macrophages inflammation

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Infarct

Villous ischemia

Acute atherosis

Distinctions

- HTN, edema, proteinuria = **preeclampsia**
- Preeclampsia + headaches and vision changes = **severe preeclampsia**
- Preeclampsia + convulsions = **eclampsia**
- Severe preeclampsia + hemolysis, elevated liver enzymes, low platelets = **HELLP syndrome**
- Other complications – hypercoagulability, acute renal failure, pulmonary edema

Management/Outcome

- **Term**= delivery
- **Preterm**
  - Mild – expectant management
  - Severe – delivery regardless of fetal age
- **Long thought no long-term maternal sequelae**
  - 20% develop HTN and micro albuminemia within 7 years
  - 2x increased heart and brain vascular disease

Placental Infections

- Two pathways
  - **ASCENDING** (more common)
    - Through birth canal
    - Usually bacterial
    - Result - premature rupture of membranes, pre-term delivery
  - **Hematogenous**
    - Transplacental
    - **TORCH**
Acute Chorioamnionitis

PMNs
Green (purulent) membranes
Amnionchorion

TORCH infections

- Toxoplasma gondii
- O – Others Parovirus B 19, Syphilis, TB, listeria
- Rubella
- CMV
- Herpes Simplex virus, HIV

- All may evoke neonate fever, encephalitis, chorioretinitis, hepatosplenomegaly, pneumonitis, myocarditis, hemolytic anemia and vesicular or hemorrhagic skin lesions.

Gestational Trophoblastic Disease

- “Tumors”
- Proliferation of placental tissue
  – Villous or trophoblastic
- Hydatiform mole (complete and partial)
- Invasive mole
- Choriocarcinoma
- Placental-site trophoblastic tumor
Hydatiform Moles
(complete and incomplete)

- Cystic swelling of chorionic villi with trophoblastic proliferation
- Infrequent in US (1:1000-2000 pregnancies)
- Most women present with miscarriage & undergo D&C based on US/HCG findings
- BENIGN but we want to know and distinguish them with regard to increased risk of invasive mole or choriocarcinoma

Complete Mole

- MOST villi enlarged, edematous
- Diffuse trophoblast hyperplasia
- Androgenic (empty ovum)
- Embryo dies very early (fetal parts rarely seen)
- 2.5% risk of choriocarcinoma
COMPLETE MOLE

GROSS:
• Delicate friable mass of thin-walled, translucent, cystic, grape like structures
DIFFUSE TROPHOBLASTIC HYPERPLASIA

COMPLETE MOLE

COMPLETE MOLE CLINICAL COURSE

• Abnormal uterine bleeding
• Passage of fluid and tissue
• Ultrasound diagnostic (snow storm pattern)
• Serum HCG↑
• Removed via curettage; serum HCG levels monitored
• 10% develop into invasive moles
• 2.5% risk of choriocarcinoma

Normal pregnancy Source: LUMEN

Uterine cavity filled with multiple sonoluscent areas of varying size and shape, "snowstorm pattern." No embryonic or fetal structure.
Partial Mole

- Some villi are edematous
- Minimal Trophoblastic proliferation
  - Minimal Triploid (69,XXY or 69,XXX)
  - Occasionally tetraploid (92,XXXY)
- One egg, two/three sperm
- Fetus, although abnormal, mostly present
- NOT increased risk for choriocarcinoma

**FEATURES OF COMPLETE VERSUS PARTIAL HYDATIDIFORM MOLE**

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>COMPLETE MOLE</th>
<th>PARTIAL MOLE</th>
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<tbody>
<tr>
<td>Karyotype</td>
<td>46,XX(46,XY)</td>
<td>Triploid</td>
</tr>
<tr>
<td>Villous edema</td>
<td>All Villi</td>
<td>Some Villi</td>
</tr>
<tr>
<td>Trophoblast proliferation</td>
<td>Diffuse; circumferential</td>
<td>Focal; slight</td>
</tr>
<tr>
<td>Atyopia</td>
<td>Often present</td>
<td>Absent</td>
</tr>
<tr>
<td>Serum HCG</td>
<td>Elevated</td>
<td>Less elevated</td>
</tr>
<tr>
<td>Fetal tissue</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Behavior</td>
<td>2.5% choriocarcinoma</td>
<td>Rare</td>
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Images: UHP Lab Web Path
INVASIVE MOLE

- Mole that penetrates uterine wall
- Hydropic chorionic villi invade myometrium
  - May embolize to distant sites
- Vaginal bleeding, persistently elevated HCG
- Risk of uterine rupture
- Chemotherapy
**Gestational Choriocarcinoma**

- **Malignant**
  - Rapidly invasive, widely metastatic
- Neoplasm of trophoblast derived cells
- Uncommon 1:20,000-30,000 US pregnancies
  - 50% from complete moles
  - 25% previous abortion
  - 22% normal pregnancy (intraplacental choriocarcinoma)
  - Ectopic pregnancy

**Choriocarcinoma**

- Rapidly growing
- Necrotic
- Hemorrhagic

**Choriocarcinoma**

- Proliferation of neoplastic cystotrophoblasts and syncytiotrophoblasts
- NO chorionic villi
Choriocarcinoma

• Presents as vaginal blood, brown fluid spotting
  – During pregnancy, after miscarriage, after curettage
  – Can be months delay

Choriocarcinoma of the ovary

• Ovarian choriocarcinoma is non-gestational
• Result of extra-embryonic differentiation of malignant germ cells
• Ovarian choriocarcinomas are generally poorly responsive to chemotherapy, poor prognosis
• Rare - Choriocarcinoma of the testis

FIN

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