PLACENTA – GROSS PATHOLOGY
ASPEN 2014
Michael K Fritsch MD, PhD
Northwestern University and Ann and Robert H. Lurie Children’s Hospital of Chicago

Maternal-Fetal-Placental Unit

From DB Singer
GOALS –

Review normal histology.

Review gross pathology of placenta, membranes, and umbilical cord.
NORMAL PLACENTAL HISTOLOGY
Membranes

- Amnion
- Chorion
- Extravillous Trophoblast
- Decidua
Chorionic Plate

- Amnion
- Epithelium
- Compact layer
- Amnion mesoderm
- Chorion with fetal vessels
- Few trophoblast stem cells
- Langhans fibrinoid
- Invasive cytotrophoblast
- Syncytiotrophoblast
- Intervillous space

- Villi and Intervillous space

- Chorion
Basal Plate

Nitabuch’s Fibrinoid

Rohr’s Fibrinoid

Villi

Decidua

Extravillous Trophoblast
Term Villi
PLACENTAL PATHOLOGY
Why examine the placenta?

**Pros:**
Many neonatal diseases are associated with placental pathology.

Placental pathology can give some insight into outcome.

Prediction for future pregnancy outcomes.

**Cons:**
High false positive findings in placenta. Many normal neonates may have pathology in their placentas.

Pathology findings are not necessarily disease specific.

False negative findings not as great, but still significant. Fetuses with pathology may have a normal placenta.

Skill and efforts of pathologists vary tremendously.
Fetal Outcomes Associated with Singleton Placental Pathology

1) Normal

2) Preterm delivery (spontaneous abortion)

3) Fetal growth restriction (SGA - IUGR)

4) Hypoxic/ischemic CNS injury

5) Infection

6) Death (stillbirth)

7) Others (syndromes, tumors, gestational trophoblastic disease, recurrence, etc)
When does a placenta get sent to a pathologist?

<table>
<thead>
<tr>
<th>Maternal</th>
<th>Placenta and umbilical cord</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>Infarcts</td>
</tr>
<tr>
<td>Pregnancy-induced hypertension</td>
<td>Abruptio</td>
</tr>
<tr>
<td>Premature rupture of membranes</td>
<td>Vasa previa</td>
</tr>
<tr>
<td>Preterm delivery before 36 weeks</td>
<td>Placenta previa</td>
</tr>
<tr>
<td>Post term delivery greater than or equal to 42 weeks</td>
<td>Abnormal appearance of placenta or cord</td>
</tr>
<tr>
<td>Unexplained fever</td>
<td></td>
</tr>
<tr>
<td>Poor previous obstetric history</td>
<td></td>
</tr>
<tr>
<td>Oligohydramnios</td>
<td></td>
</tr>
<tr>
<td>History of drug abuse</td>
<td></td>
</tr>
<tr>
<td><strong>Fetus/newborn</strong></td>
<td></td>
</tr>
<tr>
<td>Stillborn</td>
<td></td>
</tr>
<tr>
<td>Neonatal death</td>
<td></td>
</tr>
<tr>
<td>Multiple gestation</td>
<td></td>
</tr>
</tbody>
</table>

CAP guidelines (1997) and each hospital to establish their criteria based on above.

From Embryo and Fetal Pathology by E. Gilbert-Barness
SUMMARY

Figure 2-1
ANATOMIC LOCALIZATION OF PLACENTAL LESIONS

AFIP Placental Pathology by Kraus et al. 2004
GROSS PLACENTAL PATHOLOGY
Normal:

Size (at term) – 450-630 gm, 15-25 cm in diameter, up to 3 cm thick, ovoid to round, single lobe with 15-20 cotelydons.

Membranes – Clear and inserted at margins.

Placenta – Parenchyma beefy red without lesions. Chorionic plate clear with uniformly sized surface vessels.

Umbilical cord – Eccentric insertion of 3 vessel cord, 1-2 cm in thickness. Uniformly white surface and Wharton’s jelly.
SIZE AND SHAPE
SHAPE

ACCESSORY LOBES
Accessory Lobe
Succenturiate (Multilobed) Placenta

1 – 5%

2 – membrane vessels

3 – increased risk for:
   - bleeding
   - placenta previa
   - retained placenta

From DB Singer
SIZE

LGA (>10%) due to diabetes, hydrops, mesenchymal dysplasia, infections (syphilis), maternal obesity, genetic, others.

SGA (<10%) due to MVU (HTN, preeclampsia, infarcts), MPVFD/MFI, maternal chronic disease, chronic villitis, severe fetal thrombotic vasculopathy, genetic, others.
MEMBRANES & CHORIONIC PLATE
Circumvallate Membrane Insertion

Circumvallate – ridge present – associated with increased risk of bleeding and premature delivery. Circummarginate – no ridge – (common 25%) – significance uncertain.

We report both as % of circumference involved and widest amount of extrachorialis (cm).

From DB Singer
Membranes with remote parietal hemorrhages with hemosiderin.

When extensive consider the diagnosis of diffuse chorioamniotic hemosiderosis.
Subchorionic Hemorrhages and Fibrin Thrombi
Subchorionic Fibrin Thrombus

Common – 60%
Associated with preterm birth, abortion, vaginal bleeding, IUGR, fetal demise. Frequent in placentas from mothers with severe heart disease or thrombophilia.
Subchorionic Acute Hemorrhage Associated with Amniotic Fluid Infection
Squamous Metaplasia of Amnion
Squamous Metaplasia

From DB Singer
Amnion Nodosum
(OLigohydramnios & Decreased Movement)

From DB Singer
Amnion Nodosum

From DB Singer
Fetus Papyraceous
Fetus Papyraceous
BASAL PLATE & PARENCHYMA
Intervillous Thrombus

Intervillous Thrombi
Villous Infarcts

Due to diminished maternal perfusion with ischemic necrosis of affected villi. Associated with HTN and preeclampsia (MVU).

Histologic findings:
Early – Loss of intervillous space and villous crowding.
   Increased perivillous fibrin.
   Acute inflammation.

Later - Loss of nuclear basophilia.
   Ghosted villi +/- surrounding fibrin.
   Calcification.
   Surrounding villi with DVH and increased syncytial knots.

Adverse outcomes: IUGR, small placenta, death (>50% placenta infarcted).
Placental Infarcts

From DB Singer
Acute Infarct with Inflammation
Remote Infarct with DVH and SK
Chorangioma

Benign neoplasm of fetal capillaries.
Associated with multiple gestations and congenital anomalies.

Grossly: a bulging white or red mass.

Histology:
Proliferating fetal blood vessels (capillaries) with a cellular stroma.

Adverse outcomes: Rare unless large. Fetal hydrops, stillbirth, IUGR, anemia, thrombocytopenia, CHF, abruption, premature delivery, preeclampsia.
Chorangioma with fibrosis

From DB Singer
UMBILICAL CORD PATHOLOGY
**Insertion Pathology:**

**Marginal (<1cm from margin):** 7%; ? Clinical significance – associated with preterm labor, neonatal asphyxia, abortions, malformed infants.

**Velamentous (into membranes):** 1% singleton (> in twins); prone to trauma, rupture, compression, thrombosis; associated with fetal thrombotic vasculopathy, low birth weight, low Apgar, abnormal fetal heart rate patterns, prematurity, cerebral palsy, early abortion, congenital anomalies, and death.

**Furcate (vessels leave Wharton’s jelly before insertion):** most normal, but weak association with stillbirth, thrombosis of fetal vessels, IUGR & hemorrhage.
Marginal and Velamentous Insertion
Velamentous Cord Insertion

From DB Singer
Velamentous Cord with Ruptured Vessel
**Coiling Pathology:**

**Normal:** 1-3 twists (coils) per 10cm of cord.

**Hypercoiled:** >3 twists/10cm

**Undercoiled:** <1 twist/10cm

Both associated with increased risk of IUGR, fetal distress and perinatal death. Recent studies question whether hypercoiled cords are truly associated with poor outcomes!
Marginal Insertion of Hypercoiled UC with Meconium
Hypercoiled UC with Partial Loss of Wharton’s Jelly
UNDERCOILED 2 VESSEL UC
**Length:**

Normal at term: 60 +/- 13 cm.

Excessively long cords (5%) are associated with cord accidents (stillbirth), entanglements, cord prolapse, true knots, excessive coiling, constricture, thrombi. Associated adverse outcomes include fetal distress, neurologic impairment, IUGR and IUFD.

Abnormally short cords (1-2%) are associated with cord hemorrhages, abruption, failure of descent, fetal distress, low Apgar, & congenital anomalies. Difficult to assess as usually entire cord not sent to Pathology.
Long and Hypercoiled Umbilical Cord
**Number of UC vessels:**

Single umbilical artery (2 vessel cord) occurs in 1% of singleton pregnancies.

Most outcomes completely normal.

Associated increased risk of IUGR, antepartum hemorrhage, polyhydramnios and oligohydramnios.

Increased SUA in mothers with diabetes.

In autopsy studies SUA is associated with increased likelihood of other congenital anomalies.

**Accessory vessels:** aberrant right umbilical vein or vitelline vessels.
**Knots:**

Rare < 1%.

Classify as tight or loose.

Tight knots can result in umbilical vein compression. Tight knots associated with increased risk of IUFD and intrapartum demise (up to 10%) and poor neurologic outcome.
False Knots (varices)