PLACENTA – PART 2
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 – Placenta Lecture 2

- Review pathologic criteria and clinical implications of miscellaneous placental lesions including:

  Hemorrhages (abruption).

  Decidual laminar necrosis and hypoxia.

  Increased villous vascularity (chorangiosis and chorangiomatosis).

  Increased circulating nucleated fetal red blood cells.

  Meconium.

  Chronic deciduitis with plasma cells.

  Villous edema.

  Eosinophilic/T-cell vasculitis.

  Accreta.
PLACENTAL HEMORRHAGES (ABRUPTION)
Placental hemorrhages are described by their locations: marginal, retroplacental, intraparenchymal, intervillous.

Abruption defined as clinically significant antenatal detachment of the placenta, usually associated with retroplacental hemorrhage.

Not all retroplacental hemorrhages are abruptions.
Abruptions (1-4%) are clinically significant retroplacental/marginal hemorrhages. Clinical triad: vaginal bleeding, pain, rigid abdomen.

From DB Singer
Marginal Abruption

From DB Singer
Marginal Abruptio Placentae
Marginal Abrupton
Low Implantation- Praevia
Central Abruption

From DB Singer
Central Abruption
Adherent central clot 3.4 cm
Adherent marginal clot 2.4 cm
Multiple Abruptions
HISTOLOGIC FEATURES SUPPORTING THE CLINICAL HISTORY OF POSSIBLE ABRUPTION:

< 1h maybe nothing, if suspected we add this NOTE
“While there is no gross or histologic evidence of abruption, it should be noted that acute abruption may show no pathologic findings on placental examination as the clot forms acutely without time to attach to the maternal surface, indent or infarct the placental parenchyma.”

*Acute to subacute (hours to a few days) – compression of villi and loss of intervillous spaces, increased perivillous fibrin focally, acute hemorrhage with neutrophils, associated decidual necrosis (maternal arterial bleeding).

Chronic (days to weeks) – above with infarcted villi and hemosiderin (maternal venous bleeding).
Associated with multiparity, smoking, oligohydramnios, and deep implantation, circumvallate insertion, preterm delivery, CP, neurologic impairment.
Retroplacental & marginal acute to subacute hemorrhages with intraparenchymal extension consistent with the clinical history of abruption.
Compression of intervillous space near hemorrhages.
MARGINAL HEMORRHAGE/ABRUPTION IN AFI
Chronic Abrupton

From DB Singer
Summary Retroplacental and Marginal Hemorrhages:

1 – Do not correlate well with abruption (separation of basal plate from uterine wall due to intervening hemorrhage).

2 – Clinically significant (true abruption) acute hemorrhages associated with adverse outcomes: preterm delivery, IUGR, stillbirth, CNS injury.

DECIDUAL LAMINAR NECROSIS
Membrane decidual laminar necrosis is a band of decidual, trophoblastic or mixed coagulative necrosis occupying greater than 10% of membranes. Neutrophils can be associated with this lesion.

Significance and etiology is controversial but is proposed to be associated with fetal growth restriction, maternal hypertensive disorders (including preeclampsia), and in utero hypoxia. Not reproducible in all studies.

Incidence 6.6 to 18% of placentas.

While I acknowledge that membrane decidual LN is associated with various other placental pathologies and with fetal hypoxia, I consider it relatively nonspecific. I do report the presence of membrane decidual laminar necrosis, unless >30% of membranes are affected.

Papers discussing various placental findings associated with fetal hypoxia are listed at the end of the notes.
CHORANGIOMATOSIS AND CHORANGIOSIS
**Chorangiosis (hypervascularization)** – diffuse increase in number of capillaries in terminal villi.

Occurs in association with chronic hypoxia: diabetes, Beckwith-Wiedemann, Smith-Golabi-Behmel, severe maternal anemia, hypoxic maternal heart disease, maternal vascular underperfusion, fetal thrombotic vasculopathy, chronic villitis, in high altitude pregnancies, and placentas from smokers.

Incidence – 7%.

Chorangiosis is associated in some studies with perinatal cerebral palsy and is a marker for chronic hypoxia. Association with other adverse outcomes is weak.

Pathology:
Increased capillaries in terminal villi: >10 capillary cross-sections in 10 villi viewed in several fields (10) at 10X power in 3 different noninfarcted areas of placenta (diffuse). Often see some with 15-20 capillary cross-sections. Terms such as borderline or focal chorangiosis can be used. If strict criteria are not met we use the term “focal villous hypervascularity.”

Difficult in congested placentas!!! CD31 or CD34 may give falsely increased # of capillaries.
Chorangiomatosis is a lesion with features of both chorangiomas and chorangiosis.

**Chorangioma** – can be focal or multifocal. **Localized chorangiomatosis** is related to chorangiomas. Defined as chorangiomas extending into stem villi. Associated with preeclampsia, multiple gestation and late preterm birth.

**Multifocal (diffuse) chorangiomatosis** is a distinct lesion. Multifocal chorangiomatosis is characterized by a network of small anastomosing capillaries at the margins of placental stem and immature intermediate villi.

Incidence of diffuse chorangiomatosis is 0.23% (rare).

Diffuse chorangiomatosis is frequently seen in VLBW premature infants (<32w). Associations include IUGR, prematurity, preeclampsia, large placentas (Beckwith-Wiedemann), and other congenital anomalies.