Chorangiosis
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Focal Chorangiomatosis
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INCREASED NUCLEATED FETAL RED BLOOD CELLS
**Increased circulating nucleated fetal red blood cells**

Indicative of non-specific hypoxic/ischemic stress for any cause including fetal anemia.

Due to premature release of red cell precursors from BM or increased fetal erythropoiesis.

Large number of associations: MVU, FTV, MPVFD, CV, anemia (ABO, Rh, or other blood group incompatibility, fetomaternal hemorrhage).

**Pathology:**
Distinguish from lymphocytes.

Quantitate:
Mild – at 40X 2-3 unequivocal nRBCs in one or more terminal villi in >3 fields.
Moderate - >3 nRBCs in one or more terminal villi in 3 or more 40X fields.
Severe – clusters of nRBCs and immature erythroblasts in most 40X fields.

*Critical cut-off of increased circulating nfRBC is 10/10HPF.*

Timing after stress event remains controversial (2-24h minimum). Probably 6-12h reasonable estimate.

Fetomaternal hemorrhage – requires flow cytometry or Kleihauer-Betke; pathology not very helpful – associated findings: intervillous thrombi, villous edema, and increased nFRBCs.
Mild increased nFRBCs
Moderate increased nFRBCs
Moderate increased nFRBCs
Severe increased nFRBCs
CONCLUSION:
Following fetal hypoxia, transcription and translation of the EPO gene result in an elevation in plasma EPO concentration. Previous fetal studies suggest this process requires 4 to 5 h. The present studies suggest that, following the increase in plasma EPO, NRBC emerge into the circulation in $\geq 24$ h. If this model serves as a reasonable estimate, it suggests that neonates with an elevated NRBC count at birth had the onset of hypoxia at least 28 to 29 h before birth.

Potential weaknesses:
Is EPO expression the only thing driving rapid release of nfRBC?
Very artificial setting (sick liveborn neonates) injected with darbepoetin.
MECONIUM
Meconium is fetal stool composed of bile acids, phospholipids, and other components.

In late third trimester hypoxic/ischemic stress can elicit meconium passage into amniotic fluid. (a marker for stress and detrimental effects) Common 15-20% term placentas.

Toxic effects include necrosis of amnion and myonecrosis of muscular walls of umbilical cord vessels.

Factors influencing toxic effects include:
Amount passed
Duration of in utero exposure
Amount of amniotic fluid

Meconium passage and chorioamnionitis often coexist.
Meconium

Pathology:
Gross – deep green to mild opacification/discholoration.

Toxic effects on amnion – vacuolization, reactive changes, edema, separation from chorion, amniocyte necrosis.

Meconium is ingested by macrophages (red to tan-brown, often foamy) in amnion, chorion, EVT layer, and decidua.

Duration of meconium passage relatively inexact:
1h to appear in amnion
>3h to appear in upper chorion
12-16h deeper layers
I do not agree with these estimates.

Myonecrosis of smooth muscle cells composing the walls of umbilical cord vessels. Initially cells round up, cytoplasm becomes hypereosinophilic, and pyknosis of nuclei. Longer exposure results in coagulative necrosis of smooth muscle cells with complete loss of nuclear basophilia. Associated with adverse CNS outcomes.

Possible meconium aspiration syndrome.
Meconium Staining
Meconium Macrophages

From DB Singer
Meconium laden macrophages in chorion
Meconium and iron laden macrophages in chorion (Prussian Blue iron stain)
Early myonecrosis of umbilical vein
MYONECROSIS ARTERY
CHRONIC DECIDUITIS WITH PLASMA CELLS
Chronic Deciduitis with Plasma Cells

Plasma cells in decidua are pathologic. Lymphocytes and macrophages in decidua are not necessarily pathologic.

Need to pass on to the obstetrician that the presence of plasma cells raises concern for possible chronic maternal infection (endometritis). Association is about 30%.
What do the plasma cells in the other 70% represent?

Chronic deciduitis with plasma cells is frequently seen in association with chronic villitis.

Focal lymphoplasmacytic deciduitis proposed to take weeks to develop. Chronic deciduitis with plasma cells is associated with recurrent pregnancy loss and preterm delivery.

How many plasma cells are pathologic?
Chronic deciduitis with plasma cells