VILLOUS EDEMA - HYDROPS
**Villous Edema - Hydrops**

Villous edema is seen in association with numerous other placental pathologies: amniotic fluid infection, MVU, cytogenetic disorders, maternal disease states, fetal anemia, moles, and many more.

It is a nonspecific finding.

**Pathology:**
Grossly the placenta may be heavy, normal or small. Villi show diffuse or focal widening with increased clear spaces (edema). Villous capillaries may be small (compressed) by the edema. May be syncytiotrophoblast necrosis, nFRBCs, and increased Hofbauer cells.

Score as mild, moderate, severe.

Always think about metabolic disorders (look at amnion and stromal cells for cytoplasmic clearing in metabolic disorders).

In first trimester and early second trimester placentas many villi have stromal clearing that can mimic edema.

**Etiology** often difficult to establish from placenta alone, if no chorioamnionitis is present.
EOSINOPHILIC/T-CELL CHORIONIC VASCULITIS
Eosinophilic/T-cell Chorionic Vasculitis

**Background:**
Recently described placental lesion (2002).
Rare (0.2-0.6% placentas)***
Most cases involve term placentas – 94%– (virtually all >34 weeks).
Associated with VUE (43% of cases vs. 13% controls).
No association with other placental lesions (recently shown to be associated with chronic villitis in 32% of cases).

**Defined histologically:**
Fetally derived chronic inflammatory infiltrate composed of eosinophils and CD3 positive T lymphocytes focally involving a single chorionic artery or vein.
In first study, additional sections failed to reveal additional lesions.
Most recent study found 78% of cases involved a single chorionic plate vessel.
Other cases have more than one vessel involved.
(I recently had a case with multiple vessels involved with thrombi).
25% of involved vessels showed mural thrombi.

Neutrophils are not a component.
Eosinophilic/T-cell Chorionic Vasculitis
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Association with adverse outcomes unknown at this point.

Single case report of IUFD at 34w with placental findings of multivessel Eosinophilic/T-cell vasculitis without chorioamnionitis.

Related citations
Related citations
PLACENTA ACCRETA
Separation zone usually at Nitabuch’s layer/decidua.
ABNORMAL PLACENTAL IMPLANTATION.

Lack of proper decidualization can result in abnormal placental implantation – invasion into the uterine wall. Molecular/biochemical etiology unknown. Highly associated with placenta previa, *prior C-section* and prior endometrial curettage. Other risk factors include leiomyomas and uterine anomalies.

Classified by depth of placental invasion into the uterus:

**Placenta accreta** – superficial implantation of chorionic tissue (not just villi) onto myometrium with no intervening decidua. Histologically can have villi, fibrinoid, or EVT in direct contact with myometrium. **NO INTERVENING DECIDUA!!!**

**Placenta increta** – deep implantation of placental tissue into myometrium with no intervening decidua. Same histologic criteria.

**Placenta percreta** – penetration of placental tissue through the uterine wall serosa. This is primarily a gross diagnosis made by the surgeon and confirmed by the pathologist.
Focal Placenta Accreta

**Incidence:** Markedly increasing: likely due to repeat C-sections and newer definition.
1970s 0.25 to 0.4:1000; 1980s 0.8/1000; 2000s 3/1000.

Decidua prevents deep invasion into the myometrium by the EVT. Trophoblast giant cells do invade deep into the myometrium! Common to find myometrial fibers attached to the basal plate with intervening decidua (non-pathologic).

**Clinical outcomes of accreta:**
Retained placenta, pre- or post-partum hemorrhage, uterine rupture (increta/percreta).
Old studies of placenta accreta (stricter definition – villi in direct contact with myometrium) indicated 9-10% risk of maternal or fetal death.

Treatment for increta and percreta is C-section followed by hysterectomy.
Basal Plate with Attached Myometrial Fibers (Non-pathologic)
PLACENTA INCRETA
Patient – clear focal placenta accreta
Placenta Percreta