Excerpts from

THE PLACENTA: TO KNOW ME IS TO LOVE ME

A reference guide for gross placental examination

Placental Indication: Amnion Nodosum

Maternal Indication: Oligohydramnios

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First described in Germany in 1889, "amnion knots" on the placental fetal surface were later correlated to pregnancies complicated by severe oligohydramnios. However, it wasn't until 1961 that vernix caseosa was discovered to be the material forming the "knots," which are seen as granules on the fetal surface. Vernix caseosa is the white cheesy material composed of superficial cornified squamous cells and sebaceous secretions which covers the fetal skin in increasing amounts as term approaches (decreasing at term and possibly completely absent postdates) and protects the fetal skin from the amniotic fluid.

The granules of amnion nodosum are composed of hair intermingled with sebum and desquamated fetal squamous cells from the vernix caseosa. Initially it was thought contact between the vernix-covered fetus and the amnion was necessary to produce the granules—from the trauma of scratching or merely close fetal contact. However, the granules are too finely distributed and are now thought to form by apposition of vernix to regions of amnion dying due to deficient amniotic fluid (oligohydramnios) no longer supporting the nutrition and oxygen demands of the amnion. The vernix is eventually covered by regenerating amniotic epithelial cells without either inflammatory or other reaction by the amnion to the vernix. There is no correlation between the severity of oligohydramnios and the amount of amnion nodosum.

Amnion nodosum is a lesion of later gestation because there is not enough vernix during early gestation to produce the granules. Even in cases of severe oligohydramnios, amnion nodosum typically doesn't develop before 28 weeks gestation.

Amniotic Fluid

In very early pregnancy the fluid within the amniotic sac, amniotic fluid, is derived from extracellular fluid, with transfer of water and other small molecules across the amnion and then fetal skin later in the first half of gestation. After about the fourth month, amniotic epithelial secretions and fetal urine are the main sources of amniotic fluid, a time when the fetus also begins to swallow and breathe the amniotic fluid. At mid gestation there is about 400 ml of amniotic fluid, peaking at 1000 ml at 33-34 weeks, subsequently decreasing to 600-800 ml at term. The cushioning effect of the amniotic fluid allows free fetal movement without injury.

Oligohydramnios may have significant fetal/neonatal consequences. It may be related to fetal malformations producing less than expected amniotic fluid (renal agenesis), fetal chromosomal abnormalities (trisomy 18, triploidy, Turner's syndrome), congenital anomalies (Tetralogy of Fallot, meningocele, sirenomelia), hypertensive states of pregnancy, premature or prolonged rupture of membranes or even postdates gestation.

Early pregnancy oligohydramnios is often associated with fetal damage, including pressure and positional deformities, dry, leathery and wrinkled skin, and pulmonary hypoplasia. Pulmonary hypoplasia is a serious consequence of oligohydramnios. Studies have tried to decide if pulmonary hypoplasia is the result of the primary inability of the lungs to inhale normal amounts of amniotic fluid or if it is due to thoracic compression in a less-than-normally-distended (i.e. too tight) uterus. Breathing motions alone don't determine lung development; amniotic fluid is necessary for useful breathing expansion of the lungs.

Late pregnancy oligohydramnios is a sign of fetal distress. It can lead to umbilical cord compression with obstruction o blood flow and fetal hypoxia and meconiun discharge into minimal amniotic fluid resulting in concentrated meconium. Thi concentrated meconium increases the risl for fetal meconium aspiration and possibl bronchiolar obstruction, risks which ar increased in postdates gestations.

Gross Placental Appearance

The granules of amnion nodosum ar typically confined to the fetal surfac amnion and are diffuse, fine, brown yellow dull granules best seen in obliqu light. These granules can be rubbed off Amnion nodosum rarely involves th umbilical cord.

In contrast, squamous metaplasia typi cally presents on the fetal surface amnio at the base of the umbilical cord as a patcl of 0.1 - 0.2 cm plaque-like, shiny, hydro phobic, concentric elevations, whicl cannot be rubbed off. Seen on at least 25% of term and near-term placentas, these foc of keratinized squamous cells are no etiologically metaplastic, rather they ar believed to merely reflect placental matu rity. The subamniotic wedge-shape abscesses within Wharton's jelly seen i Candidiasis are confined to the umbilica cord as granular, green-yellow clusters o acute inflammatory cells. Fungal hypha are demonstrable on special stains

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