

Excerpts from
THE PLACENTA: TO KNOW ME IS TO LOVE ME

Placental Indication: Meconium
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During gestation the fetus swallows amniotic fluid, which passes rapidly through the stomach into the intestines. Most of the water is reabsorbed, leaving behind the nondigestible material, such as lanugo hairs and desquamated cells, which mixes with mucus, gastrointestinal secretions, bile and pancreatic secretions as it moves through the small and large intestines. The resultant viscid, slippery green material is meconium.

Peristaltic movement through the fetal intestinal lumen is regulated by a variety of hormones, including a recently discovered protein moiety called *motilin*. Because immature fetuses have significantly lower levels of motilin than mature fetuses, it is easy to understand why meconium discharge is rare in premature infants and increasingly common after 35 weeks gestation, particularly near term and, with increased frequency, postdates. The overall incidence of prenatal meconium discharge is about 5%.

Why is prenatal meconium discharge important? Meconium is known to have toxic components which cause necrosis and erosion with reparative dysplastic changes of the extraplacental membranes and fetal surface amnion. Repeated meconium discharge causes epithelial erosion with ballooned and vacuolated amniotic cells. It is also thought to be toxic to myocytes, particularly endothelial myocytes, causing vasoconstriction and myocyte necrosis. And what structures cover the entire placental fetal surface? Are protected by Wharton's jelly in the umbilical cord? Arteries and veins! Therefore, meconium's vasotoxic effects may result in fetal hypoperfusion with resultant fetal hypoxia.

Vascular necrosis/myonecrosis of the umbilical cord vessels associated with prolonged meconium exposure (over 18 hours) has been linked with perinatal asphyxia, intrauterine death, neonatal

death and cerebral palsy. The normally spindle-shaped myocytes of one, two or all three umbilical vessels may "ball up," with distinctive orangophilic cytoplasmic degeneration in a wedge-shaped zone around the vessel(s). This is a distinct change, different from sectioning artifact where there is no "balling up" or orangophilic change, and different from autolysis where the myocytes degenerate but don't "ball up."

If prenatal meconium discharge is not uncommon near term and postdates, what causes it to be released preterm? Research suggests that, in some cases, meconium is discharged preterm in response to fetal distress and hypoxia, and has been correlated with an increased incidence of cerebral palsy. Hence, meconium has become a "red flag" for obstetricians and attorneys. Fetal distress is thought to cause relaxation of the anal-rectal sphincter (which normally remains closed, preventing meconium discharge) and increased peristalsis in response to the fetal hypoxia/decreased oxygenation. However, the relationship between meconium and stress is not absolute, as many infants with documented distress during labor do not discharge meconium and not all stillborns, implied to have suffered severe distress before death, show meconium staining at delivery. Therefore, the significance of meconium-stained amniotic fluid should not be interpreted to be due to prenatal or labor/delivery hypoxia without conclusive evidence of fetal blood/umbilical artery blood hypoxemia and acidosis.

In addition, in breech presentation meconium is discharged due to uterine contractions bearing down on the fetal intestines and is not considered to be a sign of anoxia or fetal distress. Meconium discharge is also seen more commonly in pregnancies in which the mother used cocaine during gestation.

Keep in mind, not all green placental staining is due to meconium, particularly

before term. Hemosiderin from chronic hemorrhage may cause green discoloration, as may some infections, such as Listeriosis which colonizes the amniotic surface causing the opacity associated with chorioamnionitis to have a green hue. For green placentas of less than 35 weeks gestation, when meconium discharge is less likely, consider old hemorrhage or infection as possible causes of the discoloration. Iron stains easily differentiate between the two.

Gross Placental Appearance

Timing of histologically-apparent meconium has not been clearly established. Studies suggest gross meconium staining is evident 1-2 hours after discharge, with maximal staining by 3-6 hours. The placenta and umbilical cord may only be slippery due to meconium's mucus or only the amnion may be slightly green if meconium was discharged less than 1 hour before delivery. After 1 hour, the amnion may be intensely green, and after 3 hours the chorion may also be green.

The transfer of meconium from the amnion to the chorion may take place *after* delivery of the placenta (i.e. postpartum macrophage transfer). Therefore, it is important to promptly examine the placenta to accurately assess the distribution of meconium, which relates to the timing of prenatal discharge, or to refrigerate it until the gross examination can be done.

Microscopic Features

Meconium, within amnion and chorion macrophages, is diagnosed as bland, non-birefringent brown clumps, versus the birefringent hematoidin crystals associated with old hemorrhage. At low power, the usually poorly-cellular regions of the membranes show increased cellularity due to pseudostratification; at high power the brown pigment can be seen. With meconium discharge less than one hour prior to delivery, even with green stained amnion,

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the amnion macrophages may not show meconium ingestion. However, with meconium exposure longer than 1 hour before delivery, the amnion macrophages may show meconium ingestion, and with exposure longer than 3 hours, the chorion macrophages may also show meconium ingestion.

Meconium and Chorioamnionitis

Meconium staining is often accompanied by chorioamnionitis. In term and postdates gestations particularly, meconium may contribute to the severity of chorioamnionitis but it *does not* cause chorioamnionitis. It is hypothesized meconium alters the antimicrobial activity of amniotic fluid, creating an environment more favorable to infection, therefore creating an increased risk for chorioamnionitis and all of its complications.

Meconium Aspiration Syndrome

With meconium-stained amniotic fluid, the fetus is also at risk for meconium aspiration, which presents in the neonate as meconium in the trachea below the vocal cords and is accompanied by tachypnea. Most such infants are born at or near term and many have low Apgar scores. Meconium aspiration usually

improves within 48 hours of birth. However, if severe, it is associated with high mortality due to meconium's inhibition of pulmonary surfactant function and its toxicity which may result in pulmonary hemorrhage compounded by associated chorioamnionitis, major congenital anomalies and disorders causing low uteroplacental blood flow.

ACUTE MECONIUM STAINING

Gross Appearance: blue-green, glistening placenta covered with slimy, green meconium.

Clinical Outcome: typically normal.

SUBACUTE MECONIUM STAINING

Gross Appearance: slippery, edematous, dark discoloration of the placental membranes.

Clinical Outcome: high risk association with asphyxia and cerebral palsy; meconium aspiration syndrome.

CHRONIC MECONIUM STAINING

Gross Appearance: dull, diffuse, muddy brown-green discoloration of placenta and membranes; cord sometimes stained throughout.

Clinical Outcome: some of the infants later manifest cerebral damage, assumed to be secondary to prenatal asphyxia.

References

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