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Pathological Case of the Month

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THE INFANT pictured in **Figure 1** was a 415-g female born at 22 weeks' gestational age to a 17-year-old gravida 2, para 1 mother. The mother's previous pregnancy produced a healthy child. This pregnancy was complicated by a foul-smelling vaginal discharge and abdominal cramps 2 days prior to admission to the hospital. At admission she had a high leukocyte count and fever. After amniocentesis, she was delivered of this female infant, who lived for 8 hours. The placenta weighed 193 g and had necrotizing acute chorioamnionitis (**Figure 2** and **Figure 3**).

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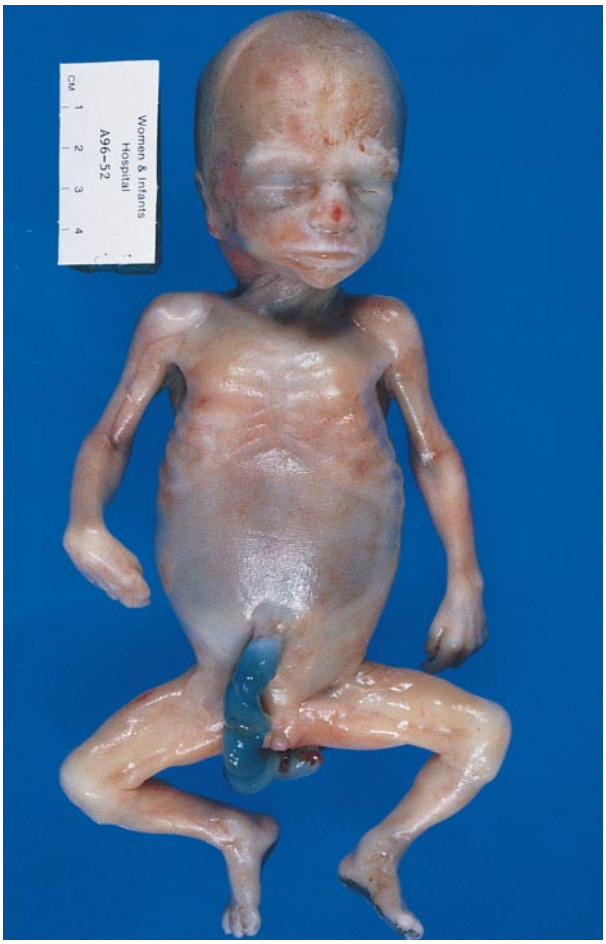

Figure 1.

All 3 umbilical vessels had vasculitis and funisitis. Airspaces were filled with polymorphonuclear leukocytes (**Figure 4**). Amniotic fluid and postmortem cultures grew *Haemophilus influenzae* biotype 1.

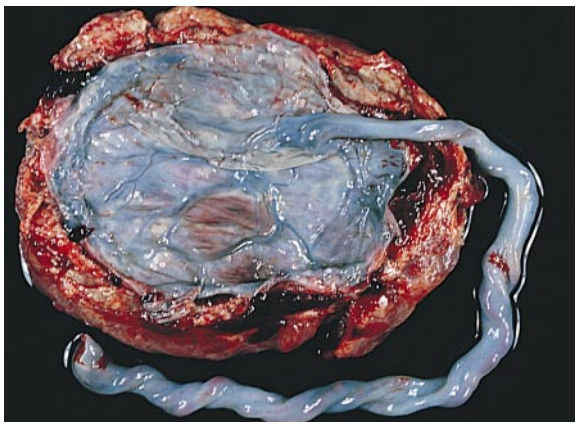

Figure 2.

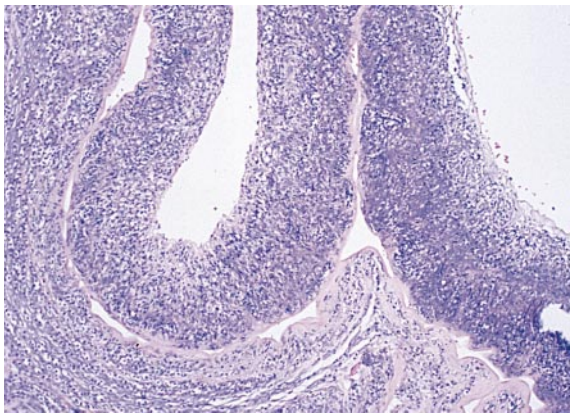

Figure 3.

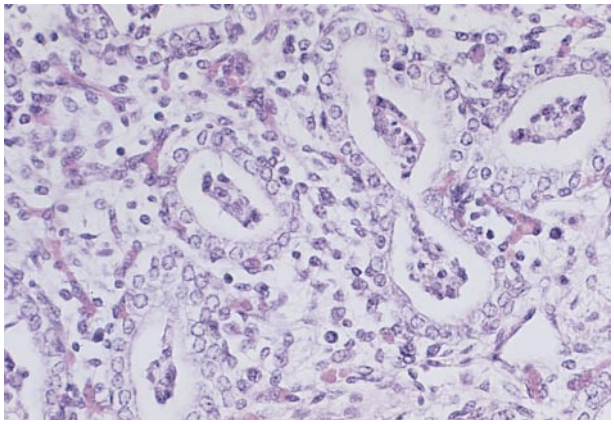

Figure 4.

Diagnosis and Discussion

Turquoise Discoloration of the Umbilical Cord and Membranes After Intra-amniotic Injection of Indigo Carmine Dye for Premature Rupture of Membranes

Figure 1. *Turquoise discoloration of the umbilical cord.*

Figure 2. *Turquoise discoloration of the fetal surface of the placenta.*

Figure 3. *Histological appearance of the necrotizing acute chorioamnionitis (hematoxylin-eosin, original magnification ×100).*

Figure 4. *Clusters of polymorphonuclear leukocytes in the airspaces are suggestive of amniotic fluid infection syndrome (hematoxylin-eosin, original magnification ×100).*

Diagnosis of premature rupture of membranes (PROM) must be considered in pregnant patients who complain of watery vaginal discharge. A vaginal pool or obvious leakage of fluid from the cervix is strong evidence supporting PROM. The fluid can be tested for "ferning" or its neutral or alkaline pH checked with phenaphthazine (Nitrazine Paper). The diagnosis is more complicated when amniotic fluid is absent. Some instances call for invasive techniques such as transabdominal injection of a dye (indigo carmine, Evans blue, fluorescein) into the amniotic cavity. A tampon in the vagina can be used to document subsequent leakage in cases of PROM. Methylene blue should not be used because it may cause fetal methemoglobinemia.[1,2] Indigo carmine was used in this patient and produced the turquoise discoloration in the surface membranes of the umbilical cord and placenta. Inflammation may have enhanced the adsorption of the dye to these surfaces.

Spontaneous rupture of membranes often leads to the onset of labor. If labor begins shortly after PROM, management issues are simple. "Prolonged PROM" implies a latency period longer than 24 hours. The prevalence of PROM is about 2% to 3.5% before 37 weeks' gestation. Despite this apparently low prevalence in preterm gestation, 30% to 40% of preterm neonates are born to women who have PROM—making PROM the leading identifiable cause of preterm delivery.[3]

The overall prevalence of positive bacterial amniotic fluid cultures in women who have PROM is 28.5%.[3] Patients with microbial invasion of the amniotic cavity are more likely to have chorioamnionitis, endometritis, and neonatal sepsis than patients who have a negative amniotic fluid culture on admission. Microorganisms isolated from the amniotic fluid of women with PROM are similar to those normally found in the lower genital tract. Mycoplasmas (*Ureaplasma urealyticum* and *Mycoplasma hominis*) are the most frequent isolates, followed by *Streptococcus agalactiae* (group B streptococcus), *Fusobacterium*, and *Gardnerella vaginalis*.[3]

Haemophilus influenzae organisms are uncommon in the female genital tract. An estimated 0.8% to 1% of all pregnant women harbor *H influenzae*. Serotyping has disclosed that from 6% to 20% of perinatal *H influenzae* infections are due to type b and rarely type c.[4] The scarcity of fetal and neonatal infections due to *H influenzae* types b and c has been attributed to the protection provided by the mother's passively transferred anticapsular antibody. Most of the remaining *H influenzae* are nonencapsulated organisms and therefore "nontypable" by serologic tests. Various chemical reactions provide separation into biotypes. All the biotypes (1-8) are capable of producing maternal and perinatal infections.[5] Among 45 cases of neonatal *H influenzae* sepsis reviewed by Friesen and Cho,[6] the overall mortality rate was 5.5%; it was 90% for 20 infants of gestational age younger than 30 weeks. In most infants with *H influenzae* sepsis, acute chorioamnionitis is present but it may be mild even in the fatal cases. Funisitis and villitis are not consistently seen, but these lesions are associated with fulminant and fatal cases.[5]

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