Infectious Diseases

Chorioamnionitis

Macroscopic Appearance

Typically, the placenta of the amnionic sac infection syndrome is premature. It lacks the blue sheen of the normal immature organ, and the membranes are obscured by an inflammatory exudate of polymorphonuclear leukocytes (PMNLs, neutrophils) (Figs. 20.1 to 20.3). The surface becomes yellow when much leukocytic exudate has accumulated and when the process has been of long duration. The amnion may be roughened or have lost the luster it normally possesses. The placenta is also frequently malodorous, and the astute observer may identify the prevailing organism by the odor. Thus, the fecal odor of fusobacterial and \textit{Bacteroides} infections, and the sweet odor of \textit{Clostridium} and \textit{Listeria} infections are useful identifiers. The membranes are typically more friable, and the decidua capsularis is frequently detached and hemorrhagic and may even be absent in the placental specimen having been left in the uterus to be later discharged as lochia. These prematurely delivered placentas are often accompanied by an acute marginal hemorrhage that undermines the edge of the placenta and that originates from deciduitis. Harris (1988) has described this in greater detail. Although this mimics abruptio placentae, this hemorrhagic process (Figs. 20.4 and 20.5) markedly differs from the typical abruptio placentae of preeclampsia, the retroplacental hematoma. Vintzileos et al. (1987) believed that true abruptio occurs after premature rupture of the membranes. They found an incidence of 6.3% (control 2.7%) of normally implanted placentas with “indentations in the placental substance.” Unfortunately, they did not discuss inflammatory reactions. Gonen et al. (1989) also provided evidence that abruptio (loosely defined) is frequently preceded by prolonged rupture of membranes. Darby et al. (1989) compared severe preterm abruptions with control women requiring preterm delivery. The former group had significantly more frequently chorio-
Figure 20.1. Near-term placenta with severe chorioamnionitis. Note the marginal hemorrhage at left, caused by deciduitis. The surface of the placenta is obscured by a whitish exudate that obscures the normal underlying blue color; the vasculature is also indistinct. Neonatal death.

Figure 20.2. Immature diamnionic, dichorionic twin placenta from a cesarean section. Twin A (left, one cord clamp) was located higher in the uterus; twin B (right), with a marginally inserted cord, was near the lower uterine segment and had significant chorioamnionitis. Compare the luster of the normal placenta (left) with the indistinct features of the abnormal placenta (right), which are due to inflammation. Neonatal deaths.

Figure 20.3. Immature twin placenta (19 weeks’ gestation) from which twin A (right) had been delivered 1 week prior to twin B (left). Both twins had severe chorioamnionitis and fatal aspiration pneumonia. The placental surface was yellow, purulent, and malodorous.