Excerpts from

# TO KNOW ME TO IS LOVE ME: THE PLACENTA

## Placental Indication: Rule out chorioamnionitis

By Doris Schuler-Maloney, M.S.

Placentas are often submitted to Surgical Pathology with a clinical history of incompetent cervix with or without cerclage, neonate temperature and the request to "rule out chorioamnionitis." Simply put, chorioamnionitis is the inflammation of the placental membranes (extraplacental and fetal surface) secondary to infection of the membranes themselves or in response to infection in the amniotic fluid. It is seen in up to 4% of term placentas and 25% of preterm placentas, and is an indication of amniotic fluid infection, usually of mixed flora.

Prenatal infections are common and varied, and many cause placental changes. Infections may ascend from the cervicovaginal canal, breaching the protective cervical mucus plug and infecting the extraplacental membranes, or they may reach the placenta parenchyma hematogenously from the maternal blood infecting the intervillous space. Most infections cause gross and microscopic placental changes, while others (like Coxsackie virus and Parvovirus) cause few characteristic or recognizable lesions.

Most commonly, bacteria reach the extraplacental membranes by transcervical passage after the bacteria degrade the cervical mucus plug via liberation of proteolytic enzymes (which are also liberated by the inflammatory cells which respond to the infection), with subsequent significant loss of elasticity and tensile strength of the extraplacental membranes at and around the os. They may also reach the extraplacental membranes via contiguous spread from endometritis or salpingitis, or by iatrogenic introduction to the amniotic cavity, membranes or placenta during surgery, chorionic villus sampling or amniocentesis.

Transcervical passage of bacteria may occur across previously ruptured membranes or intact membranes, through an area of devitalized extraplacental membrane which covers the internal cervical os. Before 30 weeks gestation, chorioamnionitis/amniotic sac infection more often cause premature rupture of membranes due to the proteolytic activity described above. When premature rupture of membranes precedes chorioamnionitis/amniotic sac infection, the cervicovaginal bacteria have unimpeded access to the amniotic cavity because the endocervical mucus plug is gone; in such cases the amniotic cavity is quickly colonized by the organisms.

Grossly, because typically delivery is premature, the placenta lacks its normal blue sheen and has gray-tan and opaque extraplacental membranes and fetal surface which obscure the fetal surface vasculature (due to the presence of acute inflammatory cells, particularly the myeloperoxidase from the polymorphonuclear cells). In cases of long standing inflammation, the fetal surface becomes more yellow from accumulated leukocyte exudate and there may be mural thrombi in the chorionic vessels. The amnion may be roughened and dull; the membranes are often friable. The decidua capsularis is frequently detached or hemorrhagic. Also, a particular pathogen may liberate a recognizable odor.

Microscopically, acute (leukocytic, granulocytic) or chronic (monocytic, usually seen in association with acute) chorioamnionitis may be diagnosed. Most commonly, acute inflammation, principally polymorphonuclear, involves one or more of the following: extraplacental membranes, chorionic plate and its under surface (subchorionic space), or blood vessels in the chorionic plate and umbilical cord. Inflammation is most consistently found at the margin of the membrane point of rupture and the under surface of the chorionic plate. The leukocytes emigrate from the intervillous space (therefore are of maternal origin) and from the fetal surface blood vessels, always toward the amniotic cavity (presumed antigenic source). Plasma cells are usually absent, but may be seen in some chronic inflections. Inflammation may be absent in some microscopic sections of one or more of these areas; therefore sections should be submitted from all of these areas - don't forget the margin of the point of rupture in the membrane roll.

Before 20 weeks gestation, the fetus is not capable of producing leukocytes and the polymorphonuclear cells present in the membranes are mainly of maternal origin - having reached the membranes from the parenchymal intervillous space (maternal circulation) and from the decidua of the extraplacental membranes. After 20 weeks, when the fetus is producing leukocytes, the fetal surface and umbilical cord blood vessels participate in the inflammatory response.

Chorioamnionitis is strongly associated with prematurity, due to preterm delivery after premature rupture of membranes. It is also associated with stillbirth and infant sepsis. However, chorioamnionitis - predominantly a maternal response so it can occur after fetal death - may only be implicated as the cause of intrauterine death if there is a demonstrable fetal response such as funisitis or if inflammatory cells are present within placental chorionic vessels or in the fetal lung or stomach.

Fetal sepsis, which develops in less than 1% of cases of chorioamnionitis, does not correlate with placental histopathology; therefore classification of chorioamnionitis based upon location of the inflammatory infiltrate has little clinical value. Fetal sepsis may develop subsequent to normal swallowing and inhalation of infected amniotic fluid; it may be overwhelming and fatal. Chorioamnionitis may also lead to fetal vascular reactivity and subsequent hypoxia, fetal bradycardia and variable and late heart rate decelerations.

Because chorioamnionitis develops in spite of ongoing medical care and results in labor which often cannot be stopped with subsequent premature delivery, it remains a major public health issue, particularly for women of low socioeconomic status and those in populations with impaired amniotic fluid antimicrobial systems. This explains, at

least in part, why the frequency of preterm births has not declined significantly over the last 30 years.

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