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MORPHO-HISTOLOGICAL FEATURES OF THE UMBILICAL CORD AND FETAL MEMBRANES IN PREGNANCIES WITH MATERNAL CARDIAC DEFECTS: CLINICAL AND PERINATAL IMPLICATIONS

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Annotation. *This thesis investigates the morpho-histological characteristics of the umbilical cord and fetal membranes in pregnant women with cardiac defects. Structural abnormalities including Wharton's jelly edema, umbilical vascular wall thickening, and chorioamniotic membrane inflammatory infiltration are identified as specific histological signatures reflecting chronic hemodynamic and hypoxic compromise in these high-risk gestations.*

Keywords: *cardiac defects in pregnancy, umbilical cord morphology, Wharton's jelly, umbilical vascular wall, chorioamniotic membranes, funisitis, fetal membrane histology, perinatal outcome, placental components, hemodynamic hypoxia*

Today, congenital cardiac defects, affecting approximately one percent of all live births and representing the most prevalent congenital structural anomaly diagnosed in contemporary obstetric practice, exert demonstrable hemodynamic and hypoxic effects not only upon the placental parenchyma but also upon the structural integrity of the umbilical cord and fetal membranes, components of the placental organ that remain systematically undercharacterized in the existing scientific literature despite their direct

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functional relevance to fetal oxygenation, immune defense, and amniotic fluid homeostasis. Abnormal umbilical cord insertion is documented in a significantly elevated proportion of pregnancies affected by cardiac defects, with conotruncal anomalies conferring a relative risk of 3.08 for abnormal cord insertion compared to normocardiac pregnancies.

The umbilical cord and fetal membranes, as integral anatomical and functional components of the human placental organ, undergo characteristic morpho-histological alterations in pregnancies complicated by maternal cardiac defects that reflect the combined hemodynamic and metabolic consequences of reduced cardiac output, altered systemic and fetoplacental vascular resistance, and chronic tissue hypoxemia. The umbilical cord, composed of two umbilical arteries and a single umbilical vein embedded within the gelatinous extracellular matrix designated Wharton's jelly and invested by a single-layered amniotic epithelium, exhibits several reproducible histological changes in the setting of maternal cardiac disease. Edematous expansion of Wharton's jelly, arising from impaired interstitial fluid drainage secondary to elevated fetoplacental venous pressure in the context of reduced right ventricular compliance or pulmonary hypertension, is among the most commonly documented findings and correlates with increased umbilical cord diameter on antenatal ultrasonographic assessment. Histopathological examination of the umbilical vessel walls in these pregnancies reveals medial smooth muscle hypertrophy of the umbilical arteries, reflecting the chronically elevated resistance within the fetoplacental vascular bed documented by elevated umbilical artery pulsatility index on Doppler velocimetry, as well as intimal thickening and subintimal fibromuscular proliferation indicative of remodeling responses to abnormal blood flow patterns. The single umbilical vein demonstrates venous wall thickening with fibrous adventitial expansion in proportion to the severity of venous hypertension imposed by diminished fetal

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cardiac compliance. These vascular wall changes directly reduce the cross-sectional luminal area available for umbilical blood flow and contribute to the progressive deterioration of fetoplacental circulatory efficiency documented over the third trimester in affected pregnancies.

Examination of the fetal membranes, encompassing the innermost amnion, the intermediate spongy layer, the chorionic membrane, and the decidua capsularis, reveals membrane thickening, reduced amnion epithelial cell height, subamniotic fibrous tissue accumulation, and increased rates of subacute chorioamnionitis reflecting inflammatory changes secondary to chronic hypoxemia and altered immune surveillance at the maternal-fetal interface. The amnion epithelium, under conditions of chronic reduced oxygen tension, exhibits cellular hydropic degeneration, nuclear pyknosis, and loss of surface microvilli responsible for amniotic fluid ion transport regulation, potentially contributing to the oligohydramnios frequently observed in pregnancies with severe cardiac compromise. The correlation between the histological severity of membrane abnormalities, the degree of maternal cardiac decompensation as assessed by functional classification, and adverse perinatal outcomes including preterm delivery, intrauterine growth restriction, and low five-minute Apgar scores underscores the clinical relevance of comprehensive placental component pathological analysis in this obstetric population.

Morpho-histological abnormalities of the umbilical cord and fetal membranes constitute specific and clinically informative structural signatures of hemodynamic and hypoxic compromise in pregnancies with cardiac defects. Their systematic pathological characterization represents an essential and currently underutilized dimension of perinatal risk assessment in this high-risk group.

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