ANGIOGENIC BIOMARKER PROFILE AND VASCULAR MORPHOLOGY OF THE PLACENTA IN PREGNANCIES COMPLICATED BY MATERNAL CARDIAC DEFECTS

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Annotation: *This thesis characterizes the angiogenic biomarker profile and vascular morphology of the placenta in pregnant women with cardiac defects. Reduced placental growth factor expression, elevated antiangiogenic mediators, and quantifiable fetoplacental vascular rarefaction are identified as the molecular and structural determinants of placental dysfunction in this population.*

Keywords: *cardiac defects in pregnancy, placental growth factor, soluble fms-like tyrosine kinase 1, fetoplacental vascular morphology, angiogenic imbalance, umbilical artery Doppler, placental microstructure, villous capillary density, syncytiotrophoblast stress, placental gene expression.*

Today, the biological interrelationship between the cardiac system and placental vascular development is increasingly recognized as a bidirectional and mechanistically complex axis, designated the cardiac-placental developmental axis, in which normal placentation is contingent upon adequate fetal cardiac output and normal fetoplacental blood flow, while placental angiogenic signaling simultaneously influences the trajectory of embryonic cardiac morphogenesis. Population-based epidemiological evidence demonstrates that cardiac defects affecting approximately one percent of all live births are associated with measurable alterations in placental angiogenic biomarker concentrations in maternal serum and umbilical cord blood,

including significantly reduced placental growth factor and elevated soluble fms-like tyrosine kinase 1, indicating a state of functionally relevant antiangiogenic imbalance throughout pregnancy.

The molecular and vascular characteristics of the placenta in pregnant women with cardiac defects reflect a state of chronic angiogenic insufficiency that begins during the critical window of uteroplacental establishment in the first trimester and persists throughout gestation. Under physiological conditions, placental angiogenesis is driven by the coordinated expression of vascular endothelial growth factor, placental growth factor, and their cognate tyrosine kinase receptors, which together orchestrate the formation of the richly branched terminal villous capillary network essential for efficient gaseous exchange between the maternal and fetal blood compartments. In pregnancies affected by cardiac defects, this coordinated angiogenic program is demonstrably disrupted, with systematic reviews confirming that reduced levels of pro-angiogenic biomarkers in maternal serum and umbilical cord blood are consistently documented across multiple independent study cohorts, independent of the specific cardiac defect subtype.

Histomorphometric quantification of the placental vascular bed in cardiac defect-affected pregnancies reveals statistically significant reductions in terminal villous capillary density, impaired branching angiogenesis, and increased mean oxygen diffusion distance from the intervillous space to the nearest fetal capillary endothelium. These structural changes directly reduce the theoretical oxygen transfer capacity of the placenta and provide the morphological explanation for the elevated rates of intrauterine growth restriction and Doppler velocimetric abnormalities documented clinically in these pregnancies. Advanced magnetic resonance imaging studies using combined diffusion-relaxation acquisition protocols have demonstrated significant differences in placental microstructural tissue components and perfusion characteristics between cardiac defect-affected and control

pregnancies, with changes most pronounced after thirty weeks of gestation, corresponding to the period of maximal fetal oxygen demand. Altered placental gene expression in cardiac defect-affected pregnancies encompasses transcriptional programs governing trophoblast invasion, angiogenesis, nutrient transport, and oxygen sensing, with differentially expressed genes documented in both placental tissue specimens and maternal serum cell-free ribonucleic acid fractions. These molecular alterations provide mechanistic links between fetal hemodynamic compromise and placental structural abnormality, suggesting that the reduced combined ventricular output characteristic of complex cardiac defects may itself impair fetoplacental vascular development through reduced mechanical shear stress within the villous capillary network, a recognized driver of endothelial angiogenic gene activation under normal developmental conditions.

Antiangiogenic imbalance and fetoplacental vascular rarefaction are central molecular and morphological determinants of placental dysfunction in pregnancies with cardiac defects. Serial biomarker monitoring and advanced placental imaging offer promising avenues for early identification and management of placental failure in this vulnerable obstetric population.

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