

**VILLOUS ARCHITECTURE AND MATURATION
ABNORMALITIES OF THE PLACENTA IN PREGNANT
WOMEN WITH CARDIAC DEFECTS: A
HISTOPATHOLOGICAL ASSESSMENT**

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Annotation: *This study examines characteristic villous architectural and maturation abnormalities of the placenta in pregnant women with cardiac defects. Systematic histopathological analysis reveals delayed villous maturation, increased maternal vascular malperfusion lesions, and reduced terminal villous branching as the primary morphological substrate of placental dysfunction in this high-risk obstetric population.*

Keywords: *cardiac defects in pregnancy, placental histopathology, villous maturation defect, maternal vascular malperfusion, terminal villous branching, syncytiotrophoblast, placental morphology, uteroplacental insufficiency, fetoplacental circulation, angiogenic imbalance*

Today, congenital and acquired cardiac defects represent one of the most significant categories of extragenital pathology complicating pregnancy, affecting approximately one percent of all live births and constituting the most prevalent structural congenital malformation encountered in obstetric practice worldwide. Epidemiological data from a nationwide Danish cohort study encompassing 924,422 liveborn singletons demonstrate that cardiac defects are independently associated with significantly reduced placental weight at birth and elevated rates of microscopic placental abnormalities, including pathological villous maturation patterns and increased maternal vascular

malperfusion lesions, underscoring the profound impact of maternal and fetal cardiac hemodynamic compromise on placental morphological integrity.

The placenta in pregnancies complicated by cardiac defects exhibits a reproducible and clinically significant pattern of histopathological abnormalities that reflects the chronic hemodynamic and hypoxic stress imposed by impaired cardiac output and altered systemic vascular resistance. Under physiological conditions, placental villous development proceeds through an orderly sequence of mesenchymal, immature intermediate, mature intermediate, and terminal villous stages, culminating in a richly vascularized exchange surface optimized for gaseous and nutrient transfer. In pregnant women with cardiac defects, this developmental trajectory is characteristically disrupted, with delayed villous maturation representing the most frequently documented microscopic abnormality. Delayed maturation manifests histologically as the persistence of large, pale, poorly vascularized immature intermediate villi in regions of the placenta where terminal villi with vasculosyncytial membranes should predominate by the third trimester, reflecting the impaired angiogenic signaling associated with chronic hypoxemia.

A systematic review of published literature encompassing twenty-one studies on placental morphology in pregnancies with cardiac defects confirmed that abnormal villous maturation and increased maternal vascular malperfusion lesions occur significantly more frequently in this population compared to normocardiac controls. Maternal vascular malperfusion lesions, including villous infarction, decidual arteriopathy, and accelerated villous maturation with syncytial knot formation, indicate inadequate remodeling of uterine spiral arteries and impaired intervillous perfusion, which are mechanistically linked to the reduced cardiac output and increased systemic vascular resistance characteristic of decompensated cardiac disease. The resulting reduction in uteroplacental blood flow precipitates ischemic injury to villous trophoblast

cells, progressive fibrinoid deposition at the villous surface, and ultimately a quantifiable reduction in the functional exchange surface available for fetal oxygenation. Histomorphometric analysis further demonstrates that placentas from pregnancies affected by congenital cardiac defects display a significantly reduced density of terminal villous capillaries, widened mean oxygen diffusion distances from maternal intervillous blood to fetal erythrocytes, and elevated rates of thrombotic vasculopathy within fetal stem vessels. Immunohistochemical characterization of angiogenic mediators in placental tissue from these pregnancies reveals reduced expression of vascular endothelial growth factor and placental growth factor alongside elevated levels of antiangiogenic soluble fms-like tyrosine kinase 1, mirroring the angiogenic imbalance documented in preeclampsia and providing a molecular explanation for the observed structural rarefaction of the villous vasculature.

Delayed villous maturation and maternal vascular malperfusion constitute the principal histopathological signatures of placental dysfunction in pregnancies with cardiac defects. These morphological findings provide an essential scientific basis for targeted surveillance and timely obstetric intervention.

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