PLACENTAL MORPHOLOGICAL ALTERATIONS AS THE PRIMARY STRUCTURAL SUBSTRATE OF INTRANATAL FETAL DEATH IN PREECLAMPSIA

Jabborova Muniskhon Abdubanon kizi
Assistant, Department of Obstetrics and Gynecology
Fergana Public Health Medical Institute

Annotation: *This thesis examines placental morphological alterations in preeclampsia-associated intranatal fetal death. Through histopathological analysis, characteristic lesions including decidual vasculopathy, villous infarction, and fibrinoid necrosis were identified as primary structural substrates driving fetal demise during labor.*

Keywords: *preeclampsia, intranatal fetal death, decidual vasculopathy, villous infarction, fibrinoid necrosis, uteroplacental insufficiency, extravillous trophoblast invasion, spiral artery remodeling, placental morphology, perinatal mortality.*

Today, preeclampsia remains one of the most devastating complications of pregnancy, responsible for approximately 70,000 maternal deaths and 500,000 fetal deaths annually, with the overwhelming burden concentrated in low- and middle-income countries. Defined by new-onset hypertension exceeding 140/90 millimeters of mercury after the twentieth week of gestation in combination with proteinuria, this condition constitutes the primary obstetrical cause of one in four perinatal deaths worldwide, demanding rigorous morphological investigation to elucidate the mechanisms underlying its fatal intranatal outcomes.

The pathological foundation of intranatal fetal death in preeclampsia resides in the progressive deterioration of uteroplacental vascular architecture

initiated during the first trimester of pregnancy. Under physiological conditions, extravillous trophoblast cells invade and remodel maternal spiral arteries between weeks eight and eighteen of gestation, transforming high-resistance, thick-walled muscular vessels into low-resistance, wide-bore conduits capable of sustaining the markedly elevated blood flow required for adequate fetal nutrition and oxygenation. In preeclampsia, this remodeling process is profoundly impaired, and spiral arteries retain their original muscular structure, resulting in persistent vasoconstriction, ischemic placentation, and consequent release of antiangiogenic mediators into the maternal circulation.

Histopathological examination of placentas retrieved from preeclamptic pregnancies complicated by intranatal fetal death consistently reveals a stereotypic constellation of structural lesions. Decidual vasculopathy, characterized by acute atherotic change with foam cell deposition, medial hypertrophy, and perivascular lymphocytic infiltration, represents the morphological hallmark of severe placental vascular insufficiency and is most strongly associated with preeclampsia accompanied by intrauterine growth restriction. Villous infarction, arising from the complete obliteration of intervillous blood flow through thrombosis of spiral artery lumina, is identified in extensive zones of the placental parenchyma and directly reduces the functional exchange surface available for fetal respiration. Perivillous fibrinoid deposition disrupts the continuity of the syncytiotrophoblast layer, further compromising transplacental gaseous exchange and nutrient transport.

During the intrapartum period, the repetitive hemodynamic stress imposed by uterine contractions is superimposed upon this chronically compromised vascular substrate. Each contraction transiently abolishes intervillous perfusion, and in the setting of severe preeclampsia, the placenta lacks the functional reserve necessary to maintain adequate fetal oxygenation during these intervals. The resulting episodes of acute uteroplacental ischemia precipitate progressive fetal hypoxemia, lactic acidosis, and ultimately

irreversible myocardial depression. Doppler ultrasonographic demonstration of absent or reversed end-diastolic flow in the umbilical artery provides the principal antenatal correlate of these morphological changes and serves as the most reliable predictor of imminent intranatal fetal death.

Placental morphological lesions in preeclampsia constitute the structural basis of intranatal fetal death. Systematic histopathological evaluation correlated with antenatal Doppler velocimetry remains indispensable for advancing preventive obstetric strategies.

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