HEMODYNAMIC PREDICTORS AND CLINICAL MANIFESTATIONS OF INTRANATAL FETAL DEATH IN SEVERE PREECLAMPSIA

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Annotation: *This thesis investigates clinical and hemodynamic predictors of intranatal fetal death in severe preeclampsia. Analysis of cardiotocographic, Doppler velocimetric, and laboratory parameters demonstrates that multisystem end-organ involvement significantly and independently increases the probability of fatal intranatal outcome in affected pregnancies.*

Keywords: *severe preeclampsia, intranatal fetal death, cardiotocography, Doppler velocimetry, end-organ dysfunction, hemodynamic instability, fetal heart rate pattern, uteroplacental blood flow, perinatal mortality, antiangiogenic biomarkers.*

Today, severe preeclampsia represents a leading cause of intranatal fetal mortality worldwide, with population-based data from the Norwegian Medical Birth Registry demonstrating 11.6 stillbirths per 1,000 preeclamptic pregnancies at 26 weeks of gestation compared with 0.1 per 1,000 normotensive pregnancies, yielding a relative risk of 86. Critically, the risk of fetal death in preeclamptic pregnancies remains more than sevenfold elevated even at 34 weeks of gestation, underscoring the persistent and progressive nature of fetomaternal jeopardy throughout the entire intrapartum period and the urgent need for precise clinical risk stratification.

The clinical trajectory toward intranatal fetal death in preeclampsia is determined by the complex interplay of maternal hemodynamic instability and the progressive deterioration of fetoplacental perfusion. Systemic arterial hypertension, mediated by widespread endothelial dysfunction and an imbalance between vasoconstrictive mediators, including endothelin-1 and thromboxane, and vasodilatory mediators, including prostacyclin and nitric oxide, imposes sustained increases in systemic vascular resistance that diminish cardiac output and further reduce uteroplacental blood flow below the threshold required for fetal well-being. Concurrent proteinuria reflects glomerular endotheliosis, a pathognomonic renal lesion of preeclampsia characterized by swollen, fenestration-obliterated endothelial cells that reduce the glomerular filtration rate and disrupt the filtration barrier integrity.

Clinical evidence demonstrates that the presence of end-organ involvement, as documented by elevated serum creatinine, thrombocytopenia, hepatic transaminase elevation, pulmonary edema, or neurological symptoms, increases the odds of perinatal death fourfold compared with preeclampsia without systemic complications. Intrapartum fetal heart rate monitoring by continuous cardiotocography identifies abnormal decelerative patterns, including late decelerations and reduced variability, in approximately 10.7% of monitored preeclamptic pregnancies, and these patterns independently predict intranatal and early neonatal death. Low Apgar scores at the fifth minute, reflecting the severity of intrapartum asphyxia, correlate directly with the degree of maternal end-organ dysfunction. Doppler velocimetric assessment of the umbilical artery provides the most objective and reproducible hemodynamic index of fetal compromise, with absent or reversed end-diastolic flow indicating critical elevation of fetoplacental vascular resistance and imminent fetal decompensation. Integration of umbilical artery Doppler findings with middle cerebral artery pulsatility indices, ductus venosus waveform analysis, and biophysical profile scoring constitutes the optimal

multimodal surveillance protocol for identifying fetuses at highest risk of intranatal death, enabling evidence-based decisions regarding the timing and mode of delivery to prevent fatal outcomes.

Multisystem end-organ involvement and pathological Doppler velocimetric indices represent the most reliable clinical predictors of intranatal fetal death in severe preeclampsia, providing an essential foundation for evidence-based intrapartum surveillance.

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