



PD005

Paediatric Cardiology & Congenital Heart Disease • Poster Presentation

FROM TERATOGENESIS TO MACROSOMY: MECHANISTIC DISTINCTIONS IN NEONATAL HYPERTROPHIC CARDIOMYOPATHY BETWEEN INFANTS OF PREGESTATIONAL AND GESTATIONAL DIABETIC MOTHERS

Peter Ogunboyede

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Background Hypertrophic cardiomyopathy in infants of diabetic mothers is typically attributed uniformly to fetal hyperinsulinemia, regardless of maternal diabetes subtype. Pregestational diabetes exposes the fetus to hyperglycemia layered on established maternal vasculopathy and advanced glycation end product (AGE) burden, while gestational diabetes (GDM), arising later, involves a shorter exposure window. Whether this produces a distinct fetal cardiac phenotype is unclear. **Objective** To synthesize mechanistic and functional echocardiographic evidence distinguishing hypertrophic cardiomyopathy between pregestational and gestational diabetic pregnancies.

Methods Narrative review of literature published predominantly within the last five years, sourced from PubMed and verified individually against fetal cardiac hypertrophy, myocardial performance index, and AGE biology in diabetic pregnancy.

Findings A pregestational diabetes mouse model showed suppressed Nkx2.5 expression, a transcription factor essential to cardiac development, in the septum and ventricular walls. However, GDM independently elevated AGE and oxidative stress markers in a 126-patient case control study, indicating the vasculopathic pathway differs by degree, not presence. A 157-pregnancy study found modified myocardial performance index most elevated in pregestational diabetes, with significantly higher NICU admission and respiratory distress ($p=0.002$, $p=0.001$). A 198-infant cohort found Tei index highest in poorly controlled diabetic mothers (0.59 ± 0.11 versus 0.37 ± 0.12 in controls, $p=0.01$), though this group was 85.6% GDM, showing glycemic control operates independently of subtype.

Conclusion Evidence supports a graded, control-quality-sensitive distinction rather than a sharp categorical divide between pregestational and gestational diabetes. Functional echocardiographic data favor greater severity in pregestational disease; molecular vasculopathic mechanisms remain demonstrated only preclinically. Diabetes subtype alone is an insufficient sole risk criterion.

Keywords: Hypertrophic cardiomyopathy; Infants of diabetic mothers; Gestational diabetes; Pregestational diabetes; Fetal hyperinsulinemia; Asymmetric septal hypertrophy.