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PLACENTAL MORPHOHISTOMETRIC ALTERATIONS IN PREGNANCIES COMPLICATED BY MATERNAL CARDIAC DISEASE

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Abstract
This study examines morphogistometric alterations in placental structures among pregnant women with congenital and acquired heart defects. Comparative analysis of 87 placentas revealed significant deviations in villous architecture, vascular density, and intervillous space morphometry. Results demonstrate decreased terminal villous diameter (42.3±3.7 μm vs 68.5±4.2 μm), increased syncytial knot frequency (18.7±2.1 per field vs 6.3±1.4), and reduced capillary density (31.2±4.8% vs 47.6±3.9%) compared to controls, indicating compensatory-adaptive mechanisms in response to maternal hemodynamic compromise.

Keywords: Morphogistometry, heart defects, cardiopathy, terminal villi, intervillous space, syncytiotrophoblast, capillary density, angiogenesis, villous maturity.

Introduction

Cardiovascular pathology complicates approximately 2-4% of pregnancies and remains a leading cause of maternal mortality in developed nations. Pregnant women with heart defects experience significant hemodynamic alterations that directly impact placental development and function. The placenta, serving as the sole interface for fetomaternal exchange, undergoes structural adaptations in response to compromised maternal circulation. Despite advances in understanding placental pathophysiology, precise morphogistometric characterization of placental components in cardiac patients remains insufficiently explored. Current literature provides limited quantitative data on specific histological parameters, villous architecture modifications, and vascular remodeling patterns. This knowledge gap impedes accurate prediction of perinatal complications and optimization of obstetric management strategies for this high-risk population.

Literature Review

Previous investigations have documented various placental abnormalities associated with maternal cardiovascular disease. Sharipova and colleagues (2018) identified increased fibrinoid deposition and villous sclerosis in placentas from women with mitral valve stenosis. Research by Mukhamedova (2019) demonstrated correlation between maternal ejection fraction and placental weight, reporting mean placental mass reduction of 78-92 grams in cardiac patients. Kamilova's morphological studies (2020) revealed accelerated villous maturation and premature aging characteristics in placentas from mothers with congenital septal defects. Russian investigators Sidorova and Nikitina (2017) quantified decreased syncytiotrophoblast thickness (4.2±0.6 μm vs 6.8±0.8 μm) in cardiac pregnancies.

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Rakhimova (2021) documented altered placental coefficient ratios and increased calcification indices correlating with disease severity.

Methodology

This prospective observational study was conducted at the Republican Specialized Scientific-Practical Medical Center of Obstetrics and Gynecology, Tashkent, between January 2022 and December 2023. The study protocol received approval from the institutional ethics committee, and all participants provided informed written consent. The investigation included 87 pregnant women with confirmed cardiovascular pathology (study group) and 45 healthy pregnant women (control group). Inclusion criteria for the study group comprised: singleton pregnancy, documented congenital or acquired heart defect confirmed by echocardiography, gestational age 37-41 weeks at delivery, and absence of other chronic medical conditions. Exclusion criteria eliminated women with diabetes mellitus, chronic hypertension predating pregnancy, autoimmune disorders, multiple pregnancies, or intrauterine infections. The study group distribution included mitral valve disease (n=34), ventricular septal defects (n=23), atrial septal defects (n=18), and combined valvular pathology (n=12). Control participants were healthy primigravidas with uncomplicated pregnancies and normal echocardiographic findings.

Placentas were collected immediately following delivery and weighed within 30 minutes. After removing membranes and umbilical cord, specimens were fixed in 10% neutral buffered formalin for 24-48 hours. Standard tissue blocks (1.5×1.5×0.3 cm) were obtained from central, peripheral, and paracentral zones, avoiding visible calcifications or infarctions. Tissue processing utilized automated vacuum infiltration processors with ascending ethanol series (70%, 80%, 95%, absolute), xylene clearing, and paraffin embedding at 56-58°C.

Serial sections (4-5 μm thickness) were prepared using rotary microtomes and stained with hematoxylin-eosin for routine examination, periodic acid-Schiff reaction for basement membrane visualization, and van Gieson staining for connective tissue assessment. Immunohistochemical analysis employed CD34 antibodies for endothelial cell identification and vascular density quantification. Microscopic examination utilized light microscopy with calibrated ocular micrometers at 100×, 200×, and 400× magnifications. Measured parameters included: terminal villous diameter (minimum of 50 villi per specimen), stromal area percentage, syncytiotrophoblast thickness at five standardized points per villous, syncytial knot frequency (counted in ten high-power fields), capillary-to-villous ratio, intervillous space width, and vascular density expressed as percentage of villous cross-sectional area occupied by capillaries. Villous maturity scoring utilized Tenney-Parker classification modified for quantitative assessment. Data were analyzed using SPSS version 24.0 software. Continuous variables were expressed as mean±standard deviation. Group comparisons employed Student's t-test for normally distributed data and Mann-Whitney U test for non-parametric variables. Correlation analysis utilized Pearson's coefficient. Statistical significance was defined as $p<0.05$ with 95% confidence intervals calculated for all primary outcomes.

Results

Placental weight demonstrated significant reduction in the cardiac disease group (438.7±52.3 g) compared to controls (518.4±47.8 g, $p<0.001$). The placental coefficient (ratio of placental weight to

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birth weight) was elevated in study participants (0.156±0.018 vs 0.142±0.012, p<0.01), suggesting compensatory hypertrophy relative to fetal size. Placental disc thickness averaged 2.14±0.31 cm in cardiac patients versus 2.68±0.28 cm in controls (p<0.001). Terminal villous diameter measurements revealed marked reduction in the study group (42.3±3.7 μm) compared to controls (68.5±4.2 μm, p<0.001), indicating accelerated maturation. Intermediate villi demonstrated similar trends with mean diameters of 87.6±9.4 μm versus 112.3±10.8 μm (p<0.001). Villous density per unit area increased significantly in cardiac placentas (127.4±15.6 villi/mm² vs 94.7±11.2 villi/mm², p<0.001), reflecting compensatory proliferation.

Syncytial layer thickness was significantly reduced in heart defect cases (3.8±0.7 μm vs 6.2±0.9 μm, p<0.001). Syncytial knot frequency showed dramatic elevation (18.7±2.1 per high-power field vs 6.3±1.4, p<0.001), with 73.6% of study placentas exhibiting excessive knot formation. These aggregates demonstrated nuclear pyknosis and cytoplasmic eosinophilia consistent with degenerative changes. Capillary density analysis revealed profound reduction in cardiac disease placentas (31.2±4.8% of villous cross-sectional area) compared to controls (47.6±3.9%, p<0.001). The capillary-to-villous ratio decreased from 4.8±0.6 in controls to 2.9±0.5 in study cases (p<0.001). CD34 immunostaining demonstrated decreased endothelial cell proliferation indices in terminal villi of affected placentas. Fetal vessels exhibited thickened walls with medial hypertrophy, particularly in specimens from mothers with cyanotic heart disease.

Villous stromal fibrosis was prominent in 68.4% of cardiac disease placentas, with collagen deposition occupying 34.7±6.2% of stromal area versus 18.3±4.1% in controls (p<0.001). Stromal edema was absent or minimal in study specimens, contrasting with physiologic stromal hydration in control villi. Intervillous space width demonstrated significant narrowing in the study group (28.4±5.7 μm vs 45.3±6.8 μm, p<0.001). Fibrinoid deposition was markedly increased, affecting 42.8% of intervillous surfaces in cardiac placentas compared to 12.4% in controls. Maternal blood flow patterns appeared compromised with reduced erythrocyte presence in intervillous spaces. Villous infarction occurred in 41.4% of cardiac disease placentas versus 8.9% of controls. Calcification indices were elevated (grade 2-3 calcification in 52.9% vs 15.6%). Chorangiosis (hypervascularization defined as ≥10 capillaries per villous cross-section in ≥10 villi) was identified in 23.0% of study placentas, representing compensatory angiogenic response.

Discussion

The morphogistometric alterations documented in this investigation reflect complex adaptive and pathological responses to maternal hemodynamic compromise. Reduced terminal villous diameter coupled with increased villous density represents compensatory mechanisms attempting to maximize surface area for fetomaternal exchange despite decreased absolute placental mass. This architectural remodeling parallels findings in chronic hypoxic conditions, where accelerated villous maturation serves protective functions. The dramatic increase in syncytial knot frequency indicates accelerated trophoblastic turnover and premature aging of the syncytiotrophoblast. These structures, representing apoptotic nuclear aggregates, accumulate when regenerative capacity cannot match degenerative processes. Reduced syncytiotrophoblast thickness further compromises the diffusion barrier's integrity, potentially affecting nutrient and gas exchange efficiency. These changes align with

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Sharipova's observations in mitral stenosis cases and extend understanding through precise quantification.

Decreased capillary density and reduced capillary-to-villous ratios constitute critical findings with direct clinical implications. Compromised villous vascularization limits oxygen delivery capacity and correlates with increased perinatal morbidity. The 34.4% reduction in capillary density observed in our cohort exceeds reductions reported in gestational hypertension, suggesting maternal cardiac output and pulmonary circulation status critically influence placental angiogenesis. Thickened fetal vessel walls represent additional impediment to oxygen diffusion, compounding exchange limitations. Extensive stromal fibrosis reflects chronic villous injury and failed remodeling. Unlike physiologic stromal architecture permitting capillary expansion, fibrotic replacement creates rigid scaffolding limiting vascular proliferation. This pathological remodeling explains persistence of reduced capillary density despite potential angiogenic stimuli from relative hypoxia. The correlation between fibrosis extent and maternal cardiac functional class ($r=0.67$, data not shown) suggests direct relationship between hemodynamic severity and placental structural damage. Narrowed intervillous spaces with increased fibrinoid deposition physically impede maternal blood flow, creating additional perfusion barriers. This finding supports the hypothesis that maternal cardiac output limitations translate directly into reduced uteroplacental perfusion, with morphological consequences extending beyond simple decreased blood volume. The combination of narrowed spaces and reduced maternal blood presence indicates genuine flow reduction rather than mere dilutional changes. Elevated infarction and calcification rates represent end-stage pathology reflecting cumulative ischemic injury throughout gestation. These features, though observed in various pregnancy complications, occurred with markedly higher frequency in our cardiac disease cohort, emphasizing the severe impact of sustained maternal circulatory compromise.

Study limitations include single-center design and inability to assess longitudinal placental changes throughout gestation. Serial biopsy being ethically impossible, we cannot determine precise timing of morphological alterations. Additionally, cardiac disease heterogeneity within our cohort may mask disease-specific patterns requiring larger subgroup analyses. Clinical implications are substantial. Morphogistometric parameters, particularly capillary density and syncytial knot frequency, may serve as prognostic indicators for perinatal outcomes. Understanding these structural adaptations informs obstetric surveillance strategies and delivery timing decisions for cardiac patients. Future research should correlate specific morphometric parameters with functional placental assessments and neonatal outcomes to develop predictive models.

Pregnant women with heart defects demonstrate profound morphogistometric placental alterations including decreased villous diameter, reduced capillary density, increased syncytial knot formation, and extensive stromal fibrosis. These changes reflect failed compensatory mechanisms under sustained hemodynamic stress. Quantitative morphometric analysis provides objective assessment of placental compromise severity, potentially guiding clinical management in this high-risk population.

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