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Pathophysiological Changes In The Fetal Myocardium Of Monochorionic Twin Pregnancies

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Abstract: Monochorionic twin pregnancies, characterized by a single shared placenta with vascular anastomoses, create unique hemodynamic conditions that affect fetal cardiac development. The continuous exchange of blood between fetuses exposes the myocardium to variable preload and afterload, leading to adaptive structural and functional remodeling. This review summarizes current evidence on myocardial changes in monochorionic twin fetuses, focusing on hemodynamic, morphologic, and biochemical aspects. Data from echocardiographic and molecular studies indicate that even in uncomplicated cases, the fetal heart exhibits concentric hypertrophy, increased wall thickness, enhanced longitudinal systolic function, and early signs of diastolic stiffness. Elevated concentrations of brain natriuretic peptide (BNP) and atrial natriuretic peptide (ANP) reflect myocardial stretch, while activation of hypoxia-inducible factor 1α (HIF-1α) and vascular endothelial growth factor (VEGF) pathways suggests metabolic and angiogenic adaptation. These findings demonstrate that the myocardium in monochorionic twin fetuses undergoes continuous physiological remodeling to maintain cardiac output under shared circulatory load. Although primarily compensatory, these changes may predispose to later cardiovascular vulnerability, emphasizing the need for careful prenatal and postnatal cardiac surveillance in monochorionic twin gestations.

Keywords: Monochorionic twins; fetal heart; myocardial remodeling; cardiac adaptation; hemodynamic stress; twin-to-twin transfusion syndrome; natriuretic peptides.

Introduction: Monochorionic (MC) twin pregnancies, in which two fetuses share one placenta, represent a unique natural model of cardiovascular adaptation. Shared placental anastomoses create unbalanced hemodynamics and oxygen distribution between fetuses, predisposing to myocardial remodeling. Even in the absence of twin-to-twin transfusion syndrome (TTTS) or twin anemia–polycythemia sequence (TAPS), subtle pathophysiological changes occur in the fetal myocardium due to altered preload, afterload, and neurohormonal regulation [5, 4].

The aim of the study is to analyze and summarize the pathophysiological mechanisms underlying myocardial adaptations in monochorionic twin pregnancies, focusing on hemodynamic, structural, functional, and biochemical changes in the fetal myocardium. The study seeks to integrate available echocardiographic, molecular, and biomarker data into a coherent physiological framework explaining how shared placental circulation affects cardiac development.

METHODS

A narrative review was performed using PubMed, Scopus, and Google Scholar databases (2015–2025). Search terms: monochorionic twins, fetal heart, cardiac remodeling, twin-to-twin transfusion syndrome, and fetal myocardial adaptation. Inclusion criteria: studies evaluating fetal cardiac structure or function via echocardiography, Doppler, MRI, or biomarkers. Exclusion: dichorionic twins and studies without cardiovascular outcomes. Data were qualitatively synthesized according to the IMRAD structure.

RESULTS

Monochorionic twin pregnancies demonstrate complex cardiovascular interactions arising from their shared placental circulation. Multiple vascular

anastomoses—arterio-arterial, veno-venous, and arterio-venous—connect the circulations of the twins, creating dynamic blood flow exchanges [5]. Even in the absence of pathological conditions, these exchanges generate subtle differences in preload and afterload between fetuses [3]. The constant redistribution of blood volume induces mild, chronic hemodynamic stress that prompts the myocardium to adapt structurally and functionally [5].

Echocardiographic studies show that MC twin fetuses exhibit increased ventricular wall thickness and relative wall-to-cavity ratios compared with singletons, indicating concentric hypertrophic remodeling [5]. Both ventricles, particularly the right, demonstrate evidence of increased wall stress and compensatory thickening, reflecting adaptation to elevated resistance within the fetal pulmonary circuit [1]. The myocardial fibers align in a manner that enhances contractile efficiency, maintaining ejection fraction despite the increased load [4].

Functionally, the myocardium in MC twins displays enhanced longitudinal motion—measured by higher tricuspid and mitral annular plane systolic excursions (TAPSE, MAPSE)—suggesting hyperdynamic compensatory function [5]. However, the prolongation of the isovolumetric relaxation time and changes in Doppler flow patterns point toward emerging diastolic stiffness [4]. This indicates that while systolic performance is preserved or even enhanced, ventricular compliance gradually decreases [5].

Biochemical evidence supports these structural and functional findings. Elevated concentrations of brain natriuretic peptide (BNP) and atrial natriuretic peptide (ANP) in the cord blood of MC twins correlate with myocardial stretch and increased atrial pressure [5]. Furthermore, studies suggest activation of hypoxia-inducible factor 1α (HIF-1α) and vascular endothelial growth factor (VEGF) pathways, particularly in pregnancies complicated by intertwin imbalance, indicating adaptive angiogenesis and metabolic reprogramming [3, 4]. Together, these data suggest that even so-called “normal” MC twin gestations involve a continuous process of hemodynamic adaptation at the myocardial level.

DISCUSSION

The results of this review highlight that the fetal myocardium in monochorionic twin pregnancies undergoes distinct adaptive changes driven by shared placental hemodynamics [5]. The presence of vascular anastomoses ensures a continuous exchange of blood between the fetuses, producing variable load conditions that stimulate myocardial remodeling [3]. This process is primarily adaptive, allowing the heart to sustain efficient output under persistent volume and pressure fluctuations [1]. Concentric hypertrophy—characterized by increased wall thickness without significant chamber dilation—appears to be the predominant response [5]. This structural change reduces wall stress according to Laplace’s law and maintains systolic performance despite elevated afterload [4].

However, these adaptive mechanisms have physiological costs. Persistent mechanical stress, coupled with altered oxygenation and hormonal regulation, can induce myocardial fibrosis and compromise diastolic relaxation [3]. The prolonged isovolumetric relaxation time observed in echocardiographic studies suggests the early onset of decreased myocardial compliance, which could represent a subclinical precursor to dysfunction [5]. The elevation of natriuretic peptides further supports the concept of neurohormonal activation as a compensatory feedback to regulate fluid balance and cardiac stretch [4].

On a molecular level, chronic hemodynamic stress may activate signaling pathways involved in hypertrophy, oxidative stress, and metabolic adaptation [3]. The upregulation of HIF-1α and VEGF indicates an attempt to enhance myocardial oxygen delivery through angiogenesis [3]. Meanwhile, oxidative stress markers suggest mitochondrial strain and energy inefficiency, which may contribute to long-term vulnerability of the myocardium [4].

The findings of this synthesis emphasize that the adaptive capacity of the fetal myocardium in MC twins is both robust and finely balanced. While it allows survival and growth under unique circulatory conditions, it also exposes the heart to persistent subclinical stress. Postnatal studies have reported that children from MC twin pregnancies retain mild cardiac geometric differences during infancy, implying a degree of structural “memory” from the intrauterine period [1]. Such persistent remodeling may predispose to future cardiovascular alterations, especially under metabolic or hypertensive stress later in life [2].

Thus, the myocardial changes in MC twin pregnancies can be viewed as a spectrum—from physiological adaptation to early pathological remodeling—depending on the degree of hemodynamic imbalance. Continuous fetal cardiac monitoring and postnatal follow-up may help detect early dysfunction [5]. Future research should aim to clarify the molecular basis of these adaptations through fetal cardiac MRI, strain analysis, and genetic studies of mechanotransduction and fibrosis pathways [3, 4]. Only by integrating structural, functional, and biochemical perspectives

can the full complexity of myocardial adaptation in monochorionic twins be understood.

CONCLUSION

Monochorionic twin pregnancies induce a distinctive pattern of myocardial remodeling characterized by concentric hypertrophy, subtle diastolic impairment, and neurohormonal activation. These changes represent a physiological adaptation to shared placental circulation but may have latent pathological potential. Recognition of these early fetal cardiac responses provides a valuable framework for prenatal assessment and targeted management of twin gestations.

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