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Name of journal: World Journal of Diabetes
ESPS Manuscript NO: 13560
Columns: Review

Contractile apparatus dysfunction early in the pathophysiology of diabetic cardiomyopathy

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Abstract

Diabetes mellitus significantly increases the risk of cardiovascular disease and heart failure in patients. Independent of hypertension and coronary artery disease, diabetes is associated with a specific cardiomyopathy, known as diabetic cardiomyopathy. Four decades of research in experimental animal models and advances in clinical imaging techniques suggest that diabetic cardiomyopathy is a progressive disease, beginning early after the onset of type 1 and type 2 diabetes, ahead of left ventricular remodeling and overt diastolic dysfunction. Although the molecular pathogenesis of early diabetic cardiomyopathy still remains largely unclear, activation of protein kinase C appears to be central in driving the oxidative stress dependent and independent pathways in the development of contractile dysfunction. Multiple subcellular alterations to the cardiomyocyte are now being highlighted as critical events in the early changes to the rate of force development, relaxation and stability under pathophysiological stresses. These changes include perturbed calcium handling, suppressed activity of aerobic

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