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Diabetes Mellitus as a Cause or Consequence of Diabetic Cardiomyopathy

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Introduction

Diabetic Cardiomyopathy (DC) is a pathological condition that raises the risk of death and morbidity associated with diabetes. Hyperglycemia, hyper insulinemia, and oxidative stress all contribute to the underlying processes of DC that cause cardiac injury and cardiomyopathy, resulting in an increase in advanced glycation end products, inflammation, fibrosis, hypertrophy, and apoptosis, among other things.

Diabetes Mellitus (DM) is considered a public health problem. The multiple complications linked with diabetes increase the incidence, morbidity, and mortality in diabetics, with heart disease being the leading cause of death. Coronary Artery Disease (CAD) is one of the primary causes of heart failure and death in people with diabetes. Even when CAD and hypertension are taken into consideration, the risk of heart failure continues to climb. Rubler et al. described ventricular dysfunction in the absence of CAD and hypertension as "a cardiac entity." At the clinical level, DC is defined by ventricular hypertrophy and diastolic dysfunction, resulting in HC with an intact ejection fraction (HFpEF).

Some sources indicate that DC causes HFrEF (systolic heart failure), although further evidence is needed to support this assertion. Diabetes is estimated to reach 300 million people by 2015, owing to an increase in obesity and physical inactivity. CD is responsible for 50% of diabetes deaths, leading in an annual mortality rate twice that of the non-diabetic population and a 5-to-10-year reduction in life expectancy. Diabetes damages the heart at multiple levels, including the epicedial arteries, autonomic dysfunction, DC, and micro vascular coronary disease, with the combination of multiple illnesses being common.

In diabetic patients, there is a growing prevalence of asymptomatic ventricular (systolic, diastolic, or mixed) malfunction that is unrelated to cardiovascular disease, hypertension, alcoholism, valve disease, or congenital, the so-called entity "diabetic cardiomyopathy," which determines a major adversity prognosis. DM is a predictive factor for cardiovascular death when only ischemic people are evaluated.

In diabetics with non-ischemic cardiomyopathy, both the SOLVD and BEST investigations indicated a substantial increase in mortality. In comparison to a non-diabetic population of the same age and gender, several epidemiological studies from the 1970s to 1979 support the high prevalence of HF in people with diabetes, with a 2-fold risk in males and a 5-fold risk in women.

Clinical Diabetic Cardiomyopathy

Despite the fact that more studies on DC have been published and more clinical evidence is available, the presence of this condition is still debatable, as DC lacks traditional cardiomyopathy markers such as ventricular dilatation and significant systolic dysfunction. DC refers to a relationship between molecular cardiac abnormalities and myocardial dysfunction, particularly when combined with other stresses such as hypertension and Coronary Artery Disease (CAD). It's difficult to distinguish the cardiac damage caused by these two disorders because CI in diabetic individuals usually coexists with hypertension and CE; the definition of Diabetic Cardiomyopathy (DC) is still up for controversy.

According to Bell, ETS or EC do not cause ventricular malfunction if diabetes is not complicated by neuropathy, nephropathy, or retinopathy, and only the myocardial one accepts the structural damage caused by diabetes associated with the AHT, which would take the ventricular malfunction as the first statement and the IC as the last statement.

The typical light subclinical cardiomyopathy of diabetes can quickly advance to a diastolic malfunction and, more recently, a systolic malfunction when untreated HTA and/or ischemic heart disease are coupled. Pathologic changes in the myocardial interstitial occur during DC, including the production of AGE, reduced compliance, and ischemia caused by sickness in the vasa vacuum. Although the structural features of myocardial cells and coronary arteries are preserved, these modifications result in a deregulation of cardiac contractility. When DC begins, LV hypertrophy develops as a result of cardiac cell enlargement, interstitial and perivascular fibrosis, thickening of the capillary basement membrane, and the formation of micro aneurysms in small capillary arteries.

The deleterious consequences of LV hypertrophy and diastolic rigidity reported in DC are exacerbated by diabetic patients' systemic pro-inflammatory state, which includes vascular inflammation and endothelial dysfunction. Endothelial dysfunction in the coronary vasculature and central cardiac endothelium reduces NO bioavailability to adjacent cardiomyocytes, lowering Cyclic Guano Sine Monophosphate (cGMP) production and Protein Kinase G (PKG) activity in cardiomyocytes, and causing DC histological and functional changes.

Other pathogenic mechanisms are produced to impair cardiac function and promote cardiomyocyte injury in diabetes: altered calcium homeostasis, altered signal transduction (insulin signaling and up regulation of the renin-angiotensin system), altered cell homeostatic processes such as apoptosis and autophagy, changes in gene regulation (activation of transcription factors, microRNAs, and epigenetic mechanisms), and post-translational modifications of structural and signaling proteases.

A DC may be suspected in diabetic patients who have ventricular dysfunction but neither atherosclerosis nor hypertension. In any case, DC may go unnoticed for a long time before clinical signs or symptoms appear. The most prevalent cardiac abnormalities identified

in asymptomatic diabetic patients are diastolic cardiac dysfunction and Left Ventricular Hypertrophy (LVH).

The initial step in the progression of cardiomyopathy is the formation of time-dependent heart muscle disease, which includes a subclinical period in which the disease's symptoms and frequent signs are missing. As a result, proof of ventricular dysfunction in asymptomatic young diabetic persons with no other illnesses capable of impacting the heart muscle and whose cardiac abnormalities are purely due to adequate diabetes is the most crucial evidence for diagnosing DC.

Marcinkiewicz et al. defined DC as a long-term process that damages the myocardium and develops at a very early stage of metabolic changes, even before diabetes is diagnosed (such as insulin resistance or overexpression of resisting). The onset of myocardial ischemia is hastened as the condition progresses. In addition to cardiac dysfunction induced by ischemia, studies reveal a relationship between heart failure and diabetes in both type 1 and type 2 diabetes mellitus. Non-Ischemic Diabetic Cardiomyopathy (NIDC) has unknown molecular and physiological processes, and there are few research on myocardial mechanics in the early stages of the disease.

Throughout the course of the disease, however, early myocardial hyper dynamics have been seen in both humans and animal models in diverse investigations. NIDC is now thought to be nonlinear, displaying an asymptomatic subclinical period of myocardial hyper contractility prior to the long-term development of cardiac dysfunction associated with diabetes and, finally, HF. Diabetes-related metabolic alterations can cause an inotropic increase and mechanical deregulation of the myocardium, leading to a gradual loss of cardiac function. As a result, diabetic patients should undergo regular tests throughout their treatment, including ultra-sensitive imaging of myocardial deformation, to identify patients who are at risk for diabetes-related heart failure.

In addition, hyper dynamic myocardial deformation may help distinguish between non-ischemic and ischemic diabetic cardiomyopathies. More research is needed to fully comprehend the pathophysiological mechanisms at play, as well as the spatiotemporal evolution of DC and its long-term relationship with clinical outcome markers.