COMMENTARY ARTICLE

Diabetic related cardiomyopathy: Commentary

Sai Sravani Botcha Venkata

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ABSTRACT

Diabetic cardiomyopathy is a cardiac muscle condition that affects diabetics. It can cause heart failure, which is defined as the inability of the heart to adequately circulate blood throughout the body, as well as fluid accumulation in the lungs (pulmonary edoema) or legs (leg edoema) (peripheral edema). Patients with diabetes mellitus show an increased incidence of Heart Failure (HF) even after adjustment for well-established risk factors for HF such as hypertension and ischemic heart disease. The resulting specific form of cardiomyopathy is known as "Diabetes Mellitus Related Cardiomyopathy" (DMCM). Pathogenic mechanisms underlying DCM are likely to be multifactorial from altered myocardial metabolism (hyperglycemia, hyperinsulinemia, increased circulating fatty acids and triglycerides) to microvascular disease, and altered myocardial structure with fibrosis. Current guidelines recommendations for medical treatment on HF in patients with Diabetes Mellitus (DM) do not differ from those for patients without DM.

Key Words: *Diabetic cardiomyopathy; Cardiac muscle; Peripheral edema; Hyperglycemia; Hyperinsulinemia*

INTRODUCTION

A cardiomyopathy is defined as a heart muscle disease in which the myocardium is structurally and functionally abnormal in the absence of coronary artery disease as well as hypertensive, valvular, or congenital heart disorders [1]. Diabetes Mellitus-Related Cardiomyopathy (DMCMP) was initially identified as a cardiomyopathy with a dilated/HFREF (heart failure with reduced ejection fraction) phenotype. Currently it manifests itself mainly as a cardiomyopathy with a restrictive/HFPEF (heart failure with preserved ejection fraction) phenotype. Diabetes mellitus also appears to be strongly linked to Heart Failure (HF).

But it has also been shown that diabetes can cause heart failure independently of Ischemic Heart Disease (IHD) by causing diabetic related cardiomyopathy. The prognostic impact of diabetes mellitus in patients with heart failure is markedly influenced by the underlying etiology and is particularly deleterious in those with Diabetic Ischemic Cardiomyopathy (DMICM) [2]. It is also reported that there are poor prognosis and high-risk mortality associated with diabetes and ischemic cardiomyopathy, while there was no association between diabetes mellitus and mortality risk in those with non-ischemic cardiomyopathy. However, two studies found diabetes to be associated with an increased mortality rate only in the non-ischemic heart failure subgroup [3-4].

Increased oxidative stress

Increased ROS production in the diabetic heart is a contributing factor in the development and the progression of diabetic cardiomyopathy [5]. Increased ROS generation and impaired antioxidant defenses could both contribute to oxidative stress in diabetic hearts. Increased mitochondrial ROS generation has been shown in various tissues such as endothelial cells that are exposed to hyperglycemia [6]. Increased ROS generation may activate maladaptive signaling pathways, which may lead to cell death, which could contribute to the pathogenesis of diabetic cardiomyopathy [7]. Increased ROS production was associated with increased apoptosis as well as with increased DNA damage and loss of activity of DNA repair pathways.

Diagnosis of diabetic cardiomyopathy

Department of Pharmaceutical Sciences, Vignan Institute of Pharmaceuticals Technology, Vizag, AP, India

Correspondence: Sai Sravani Botcha Venkata, Department of Pharmaceutical Sciences, Vignan Institute of Pharmaceuticals Technology, Vizag, AP, India, E-mail sravanibotcha05@gmail.com

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There are 2 important components for the clinical diagnosis of diabetic cardiomyopathy: the detection of myocardial abnormalities and the exclusion of other contributory causes of cardiomyopathy. An important challenge in the clinical diagnosis of diabetic cardiomyopathy has been the lack of any pathognomonic histologic changes or imaging characteristics associated with the diagnosis. The diagnosis of diabetic cardiomyopathy currently rests on noninvasive imaging techniques that can demonstrate myocardial dysfunction across the spectra of clinical presentation. Endomyocardial biopsies are not indicated because of their invasiveness. In patients with overt heart failure, the presence of echocardiographic features of cardiac dysfunction or structural abnormalities is often confirmatory. However, in the absence of overt symptoms (so-called Stage B heart failure in the American College of Cardiology/American Heart Association staging of chronic heart failure), an imaging diagnosis is warranted. It is important to emphasize that there is still no consensus in the precise imaging definition of diabetic cardiomyopathy, but evidence of hypertrophy or diastolic dysfunction is likely crucial to support a diagnosis of diabetic cardiomyopathy, but is not specific to it. A proposed Imaging definition for diabetic cardiomyopathy includes either or both features listed as follows:

- Evidence of cardiac hypertrophy determined by conventional echocardiography or cardiac magnetic resonance imaging
- Evidence of LV diastolic dysfunction (with or without LV systolic dysfunction), either clinically by Trans mitral Doppler or tissue Doppler imaging (TDI), or evidence of left atrial enlargement; or sub clinically by novel imaging techniques or provocative testing (eg, strain imaging or stress imaging)

Evidence of left ventricular diastolic dysfunction

Early studies demonstrated that abnormalities in trans mitral Doppler inflow patterns were associated with poor glycemic control and presence of cardiac structure abnormalities [8]. Also, improvement in glycemic control has demonstrated the return to a more normal profile, suggesting that the process might be reversible in its early stages [8,9]. TDI uses the ability of detecting changes in the movements of the mitral valve by Doppler imaging signals at specified myocardial locations adjacent to the mitral annulus. By using a combination of trans mitral Doppler (E) and TDI indices (E'), the ratio of mitral E/E' has been used to detect the presence of impaired LV compliance (and to some extent an estimate of LV end-diastolic pressure). In several surveys of diabetic patients without overt signs and symptoms of heart failure, TDI studies have helped uncover subtle abnormalities and have identified diastolic dysfunction in a significantly higher number of asymptomatic subjects than conventional Doppler echocardiography.

Glycemic control medications

Although the effect of glycemic control on diabetic cardiomyopathy has been studied in only a limited fashion, evidence suggests that good glycemic control is beneficial, at least in the early stages of myocardial dysfunction [9]. Evidence also suggests that diabetic cardiomyopathy does not develop in patients with tightly controlled type 1 diabetes, supporting an important role for hyperglycemia in the pathogenesis of diabetic cardiomyopathy [10]. Hyperglycemia is responsible for microvascular complications in diabetes, and because microvascular alterations are thought to contribute significantly to the pathogenesis of diabetic cardiomyopathy, good glycemic control is perhaps the most important component in the overall management of diabetic cardiomyopathy.

Firm recommendations regarding the choice of current glucose-lowering therapies in patients with diabetic cardiomyopathy cannot be made because of a lack of evidence. However, glucagon-like peptide-1 analogues have demonstrated improved hemodynamic variables in diabetic patients without overt heart failure. Improved cardiac parameters also have been noted with this agent class in post infarction and in populations with advanced heart failure [11,12]. In general, the choice of antidiabetic therapy in diabetic cardiomyopathy should be dictated by clinical characteristics, such as the presence or absence renal dysfunction, risk of hypoglycemia, age, volume status, and concomitant drug therapy. Only some glucose-lowering medications have specifically been studied in patients with HF.

CONCLUSION

The risk of HF in patients with DM is significantly increased, and it is one of the most common initial manifestations of CVD in patients with T2D. Diabetes is associated with worse outcomes in patients with HF. In diabetic cardiomyopathy, the disease course consists of a silent asymptomatic subclinical and usually long period, during which cellular structural damage initially leads to diastolic dysfunction, thereafter to systolic dysfunction, and eventually to HF. The pathogenic mechanisms underlying DCM are likely to be multifactorial, ranging from altered myocardial metabolism to endothelial dysfunction, microvascular disease, autonomic neuropathy, and altered myocardial structure with fibrosis.

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