Diabetic Cardiomyopathy: Understanding the Independent Relationship Between Diabetes and Heart Failure

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Diabetic Cardiomyopathy: Understanding the Independent Relationship Between Diabetes and Heart Failure.

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Introduction

Type 2 diabetes mellitus (DM) and heart failure (HF) are common chronic disorders that affect 29 million and 6.5 million Americans respectively. While both often occur individually, there is an increase rate of the two diseases occurring concomitantly. It is estimated that 19-28% of patients with DM having clinically significant HF and 10-47% of patients with HF having evidence of DM, both with independent or other risk factors. This association between DM and HF is well documented, but the evidence supporting diabetic cardiomyopathy is less understood. This is due to the presence of other comorbid conditions such as hypertension (HTN) and coronary artery disease (CAD), both of which are independently associated with the development of HF.

Currently, Diabetic Cardiomyopathy (DCM) is defined as a clinical condition of ventricular dysfunction that occurs in the absence of significant CAD, HTN, or valvular disorders. Currently, Diabetic Cardiomyopathy (DCM) is defined as a clinical condition of ventricular dysfunction that occurs in the absence of significant CAD, HTN, or valvular disorders. While both often occur individually, there is an increase rate of the two diseases occurring concomitantly. It is estimated that 19-28% of patients with DM having clinically significant HF and 10-47% of patients with HF having evidence of DM, both with independent or other risk factors. This association between DM and HF is well documented, but the evidence supporting diabetic cardiomyopathy is less understood. This is due to the presence of other comorbid conditions such as hypertension (HTN) and coronary artery disease (CAD), both of which are independently associated with the development of HF.

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Case Report

History: A 34-year-old African American female with a past medical history of type 2 diabetes was admitted to our hospital from an outside emergency department due to anemia. History was obtained from the patient and her partner who was at the bedside. The patient reported feeling increasing malaise and lower extremity edema for 1 month with associated shortness of breath and chest pain (CP). She described the CP as a substernal pressure and like someone was “sitting on her chest.” She also complained of orthopnea and heartburn when lying flat, a dry cough that was now productive of white sputum, and sinus congestion. She denied any nausea, vomiting, diarrhea, constipation or abdominal pain, but did have 1 episode of posttussive emesis. She had previously been worked up for anemia without any diagnosis being made. She denied any history of epistaxis, menorrhagia, menopause, or any history of GI bleeding. She had been uncontrolled recently and were running 200-500 on her glucometer due losing her insurance and no longer having a primary care physician.

Past Medical History: Type 2 Diabetes, Hypertension, Hypertoproteinemia

Family History: Sickle Cell Trait

Medications: Atenolol, Hydrochlorothiazide, Insulin Levenir and Novolog, and Gabapentin.

Vital Signs: BP 184/91, HR 120, RR 20, T 37.1°C, SpO2 96% RA, BMI 38.61

Physical Exam: On exam, the patient was an awake and alert obese female in mild distress. She had crackles noted in the lower lung lobes bilaterally and a tachycardic pulse. She denied any history of epistaxis, menorrhagia, or melena. Blood sugars had been uncontrolled recently and were running 200-500 on her glucometer due losing her insurance and no longer having a primary care physician.

Emergency Department paddles were set at 100, and she received epinephrine and norepinephrine. The patient complained of substernal chest pain and was diaphoretic. She had previously been worked up for anemia without any diagnosis being made. She denied any history of epistaxis, menorrhagia, menopause, or any history of GI bleeding. She had been uncontrolled recently and were running 200-500 on her glucometer due losing her insurance and no longer having a primary care physician.

C. D-ECHO report documenting a normal size left ventricle with an ejection fraction of 51%. There is mild thickening of the left wall. Segments of the basal inferior and mid inferior left ventricular wall are akinetic. Findings were consistent with a pseudonormal left ventricular filling pattern, with consistent abnormal relaxation and increased filling pressure (Grade 2 Diastolic Dysfunction). There was normal ventricular size and function of the right ventricle.

D. ECHO report documenting a normal size left ventricle showing no evidence of acute cardiovascular disease.

Figure 2A. From admission showing sinus tachycardia (Rate 103) with nonspecific T wave abnormalities.

Figures

Discussion

The development of DCM is related to the effects of DM systemically, on the myocardium, and directly on cardiomyocytes. Hyperglycemia, hyperinsulinemia, and insulin resistance are the major contributors to the pathogenesis of DCM, causing autonomic dysfunction, oxidative stress, and altered cellular metabolism (Figure F). This results in cardiac hypertrophy, atherosclerosis, and dysfunction of the renin-angiotensin-aldosterone system (RAAS), and the formation of advanced glycation end products (AGEs), all of which contribute to the eventual development of HF. In addition, DCM is also a major risk factor for CAD by increasing the arteriogenicity of lipids and accelerating the formation atherosclerosis; therefore also contributing indirectly to the development of HF.

Functionally, one of the earliest manifestations of DCM is left ventricular hypertrophy (LVH), primarily affecting the ventricular septal and left posterior myocardial walls, along with associated diastolic dysfunction. These early cardiac changes occur subclinically and are believed to be present in 40-70% of patients with DM. This is important because cardiac damage and disease progression begin before patients ever develop clinical symptoms, which highlights the challenge of how to effectively identify and treat patients with DCM.

Multiple studies have sought to use markers and tests such as B-type natriuretic peptide (BNP), exercise stress testing, echocardiography, and nuclear imaging to identify patients with subclinical disease, but none have proved to have sufficient sensitivity or specificity.

As a result, an understanding and clinical awareness of diabetic cardiomyopathy by providers when managing diabetic patients is imperative. The UK Prospective Diabetes Study found that a 1% reduction in hemoglobin A1c (HbA1c) was associated with a 16% risk reduction for the development of HF. In patients with diagnosed HF though, DM management had a more U-shaped effect, with the lowest mortality among patients with a HbA1c of 7-8.0.

Another important decision in the management of patients with DM is medication selection. Research has shown that SGLT2 inhibitors are the most beneficial for DM patients both at risk of developing HF or with established HF, while GLP-1 receptor agonists are beneficial in patients with established HF.

References


11. SGLT-2 inhibitors are the most beneficial for DM patients who are at risk of developing HF or with established HF, while GLP-1 receptor agonists are beneficial in patients with established HF. Insulin, metformin, and sulfonylureas are acceptable alternatives and thiazolidinediones and dipeptidyl peptidase-4 inhibitors (DPP-4) are not recommended in patients at risk or with established HF.

Understanding the development of DCM and how best to identify and treat this condition is vital to improving patient morbidity and mortality.

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