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 19-26% of patients with DM having clinically significant HF and 10-47% of patients with HF having evidence of DM with both independent of 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. This is due to the presence of other comorbid conditions such as hypertension and coronary artery disease (CAD), both of which are independently associated with the development of HF. 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.

Diabetes and Heart Failure

Type 2 diabetes mellitus (DM) and heart failure (HF) are common chronic disorders that affect 19 and 47% of all Americans respectively. 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 1 month with associated shortness of breath and chest pain (CP). She described her 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, melorrhagia, or menorrhagia. She had been controlled recently and were running 700-500 on her glucose due losing her insurance and no longer having a primary care physician.

Past Medical History: Type 2 Diabetes, Hypertension, Hypertension.

Family History: Sickle Cell Trait

Medications: Alovastatin, Hydrochlorothiazide, Insulin Levemir and Novolog, and Gabapentin.

Vital Signs: BP 184/91, HR 20, T 37.1C, Sp02 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 heart rate with a 2/6 sистolic murmure. Jugal venous pulse was elevated. The abdomen was soft, nontender, and nondistended. The lower extremities were tense and edematous with nonpitting edema. Small purple lesions were scattered over the lower abdomen was soft, nontender, and nondistended. The lower extremities were tense and edematous with nonpitting edema. Small purple lesions were scattered over the lower extremities. She had crackles noted in the lower lung lobes bilaterally and a tachycardic heart rate with a 2/6 systolic murmur. Jugal venous pulse was elevated. The abdomen was soft, nontender, and nondistended. The lower extremities were tense and edematous with nonpitting edema. Small purple lesions were scattered over the lower extremities.

A. EKG from admission showing sinus tachycardia (Rate 105) with nonspecific T wave abnormalities.

B. Initial CMP showing no evidence of acute cardiocirculatory disease.

C. EKG from admission showing sinus tachycardia (Rate 105) with nonspecific T wave abnormalities.

D. EKG CHF showing a normal size left ventricle with an ejection fraction of 50%. There is mild thickening of the left wall. Segments of the basal inferolateral and mid inferoapical walls are hypokinetpic. The left posterior and septal wall segments are akinetic. Findings are consistent with a pseudonormal left ventricular filling pattern, with concomitant abnormal relaxation and increased filling pressure (Grade 2 Diastolic Dysfunction). There were normal ventricular size and function of the right ventricle.

Figure 1: EKG from admission showing sinus tachycardia (Rate 105) with nonspecific T wave abnormalities.

Case Conclusion

During her admission, the patient underwent extensive evaluation for her complaint of malaise. Her ECHO (Figure 2) showed evidence of heart failure with abnormal ventricular relaxation, which was consistent with the fibrosis/scarring noted on her cardiac MRI (Figure E). The differential diagnoses were whether the cardiomyopathy was primarily related to her DM or due to ischemia. Due to the elevated troponin, the patient eventually underwent a coronary catheterization which showed partial occlusion of the right coronary artery. While this finding was evidence of the presence of CAD, discussion amongst the care team felt that the cardiomyopathy was primarily related to the patients long standing and markedly uncontrolled DM, while this incident of acute decompensation was likely related to acute ischemia.

Final Diagnosis: Diabetic Cardiomyopathy

Discussion

The development of DCM is related to the effects of DM systemically, on the myocardium, and directly on cardiomyocytes. Hyperglycemia, hyperinsulinaemia, and insulin resistance are the major contributors to the pathogenesis of DCM, causing autonomic dysfunction, oxidative stress, and altered cellular metabolism (Figure 2). This results in cardiac hypertrophy, fibrosis, and remodelling of the renin-angiotensin-aldo.sterone system (RAAS), and the formation of advanced glycation end products (AGEs), all of which contribute to the eventual development of HF. Additionally, 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%. Another important decision in the management of patients with DM is medication selection. Research has shown that SGLT-2 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 establish HF. Insulin, metformin, and sulfonylureas are acceptable alternatives and thiazolidinediones and dipeptidyl peptide-4 inhibitors (DPP-4) are not recommended in patients at risk or with established HF.

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

References