Uncontrolled Diabetes Resulting in Diabetic Cardiomyopathy in a Young Male Patient and Eventually Presenting With a Stroke

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ABSTRACT

Diabetic cardiomyopathy (DCP) is defined as the cardiovascular damage present in diabetes patients, which is characterized by myocardial dilatation and hypertrophy, as well as a decrease in the systolic and diastolic function of the left ventricle, and its presence is independent of the coexistence of ischemic heart disease or hypertension. As in the case of the patient that we present here, DCP may be subclinical for a long time, before the appearance of serious clinical symptoms, signs and complications. DCP is poorly recognized by most physicians. Currently, there is no specific treatment for this pathologic entity. However, proper treatment of diabetes and its metabolic abnormalities in the primary care setting reduces the rates of this serious metabolic complication of diabetes. As this high-risk diabetic population is constantly rising, increasing the awareness of physicians for the serious metabolic complications of diabetes, especially in the primary care setting, will help in taking appropriate and early action towards the prevention of full-blown disease and decreasing disability and mortality.

Keywords: Diabetes, diabetic cardiomyopathy, stroke

Introduction

Diabetes affects almost every tissue in the body and causes significant organ dysfunction that results in diabetes-related morbidity and mortality. Cardiovascular diseases account for about 65% of diabetes-related mortality. Coronary artery disease is the leading cause for the increased cardiovascular morbidity and mortality in diabetes, and atherosclerosis of the coronary vessels is its initial pathogenetic mechanism. Coronary artery disease and hypertension can account for most of the myocardial abnormalities that occur in diabetes. However, postmortem, experimental, and observational studies also provide evidence for a specific cardiomyopathy in diabetes, which may contribute to myocardial dysfunction in the absence of coronary artery atheroma. This is also sustained by the fact that patients with diabetes, independently of the severity of coronary artery disease, have increased risk of heart failure in comparison with subjects without diabetes.

In general diabetes affects the heart in 3 ways: (1) coronary artery disease due to accelerated atherosclerosis; (2) cardiac autonomic neuropathy; and (3) diabetic cardiomyopathy (DCP). Although there is high awareness among clinicians about the first two disease entities, diabetic cardiomyopathy is poorly recognized by most physicians. DCP, first defined by Rubler in 1972, is characterized by the myocardial dysfunction in the absence of coronary artery disease, hypertension, or valvular heart disease. It is associated with both type 1 and type 2 diabetes mellitus (T1DM and T2DM). As in the case that we report here, DCP may be subclinical for a long time, before the appearance of clinical symptoms or signs.

Case Report

We present the case of a 39-year-old man that was admitted in our clinic due to a stroke. According to its past medical history, the patient was diagnosed in the primary care setting with elevated blood glucose levels, about 8 months before the stroke. The rest of his past medical history was free of any other diseases. According to the information we have, at that time the patient was given lifestyle advice on dietary and physical activity, by the
primary care physician. He was also strongly encouraged to lose weight as the patient had body mass index of 34 (BMI = 34). Unfortunately, there is no record of the specific glucose values identified at that time. We don't know also, the time period that this patient had deranged blood sugar levels, since, according to what he reported, he didn't have any medical blood tests done over the past 10 years.

During his admission to the hospital, the patient was afebrile, well-oriented in time and space, with paresis of the right upper and lower limbs. His arterial blood pressure was 140/90 mmHg, electrocardiogram (ECG) with SR and his pulse was regular. The rest of the physical examination was unremarkable. His BMI at that time was the same with the BMI originally calculated about 6–8 months ago. His medical history was free of any other diseases, apart from the hyperglycemia mentioned earlier.

The computed tomography (CT) brain scan revealed an extensive ischemic infarct of the left parietal lobe in the supply zone of the left middle cerebral artery. His biochemical tests results were: White Blood Cell count (WBC) =9000/μL, hematocrit value (HCT) =44.8%, serum glutamic oxaloacetic transaminase (SGOT) =33U/L, serum glutamic-pyruvic transaminase (SGPT) =21U/L, γ-GT: 53U/L, blood glucose level: 187 mg/dl.

During the 2nd day of hospitalization we performed an ECG. This revealed heart failure and a large thrombus in the left ventricle. We found an enlarged left atrium, an enlarged hypokinetic left ventricle with an ejection fraction of 30% and a large thrombus in it as well as an enlarged right ventricle. As already mentioned, the patient had no specific symptoms of heart failure. Warfarin was added to his drug therapy, that already included antiplatelet agents.

During his stay in the hospital, the diagnosis of DM for the first time was made and his glycosylated hemoglobin level (HbA1c) was 10.42%. Insulin therapy was initiated. The patient followed a stable clinical course with a progressive improvement in his motor functions. He is now on motor physiotherapy and a regular medical follow-up for his diabetes, his heart failure and the stroke.

**Discussion**

DCP is defined as the cardiovascular damage present in diabetes patients, which is characterized by myocardial dilatation and hypertrophy, as well as a decrease in the systolic and diastolic function of the left ventricle, and its presence is independent of the coexistence of ischemic heart disease or hypertension. As in the case of our patient, DCP may be subclinical for a long time, before the appearance of clinical symptoms or signs.\(^{3}\)

The pathogenesis and pathophysiology of DCP is not yet fully defined. Various proposed mechanisms include metabolic disturbances, insulin resistance, microvascular disease, alterations in the renin-angiotensin system (RAS), cardiac autonomic dysfunction and myocardial fibrosis.

Chronic hyperglycemia is thought to play a central role in the development of DCP, although multiple complex mechanisms and an interplay of many molecular and metabolic events within the myocardium and plasma contribute to the pathogenesis. The main metabolic abnormalities in diabetes are hyperglycemia, hyperlipidemia and inflammation, all of which stimulate generation of reactive oxygen or nitrogen species that cause most of the diabetic complications, including diabetic nephropathy and DCP. Several adaptive responses caused by these metabolic abnormalities finally result in cardiac dysfunction and heart failure.\(^{5-8-10}\)

Currently, there is no specific treatment for this pathologic entity. However, proper treatment of diabetes and its metabolic abnormalities in the primary care setting reduces the rates of DCP, myocardial infarction, and associated cardiovascular death. DCP is poorly recognized by most physicians. Since DCP is now known to have a high prevalence in the asymptomatic diabetic patient, screening for its presence at the earliest stage of development would be appropriate in order to prevent the progression to congestive heart failure and further significant complications.

As this high-risk diabetic population is constantly rising, increasing the awareness of physicians for the serious metabolic complications of diabetes, especially in the primary care setting, will help in taking appropriate and early action towards the prevention of full-blown disease and decreasing disability and mortality.

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