Original Article

Case Report on Dilated Cardiomyopathy in Type 2 Diabetes Mellitus Patient with Hypothyroidism

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ABSTRACT

Dilated cardiomyopathy (DCM) is an idiopathic condition manifesting decreased contractility of heart, which is irreversible and slowly progressive. DCM, thyroid disorder and diabetes mellitus are the most interlinked symptomatology. Thyroid disease is a common endocrine disorder associated with diabetic cardiomyopathy. The complications which have been exhibited are contractile dysfunction, systolic dysfunction, ventricular dysfunction and diastolic dysfunction, ventricular diastolic and systolic dysfunction. A case study of a 75-year-old female patient visited the general medicine OPD with chief complaints of shortness of the breath, swelling of legs, chest discomfort, severe constipation, fatigue and palpitation. The patient was advised to have electrocardiogram which showed atrial fibrillation with ventricular tachycardia, and the chest X-ray which revealed cardiomegaly. The patient was also undergone echocardiogram which showed dilated cardiomyopathy with severely decreased left ventricular systolic function and a reduced left ventricular ejection fraction of 39%. Thyroid function test revealed a low hormone level. A regular treatment and continued patient's follow up care were suggested to the patient, and she manifested a progressive development of her condition. The patient was prescribed with discharge medications, and advised for a follow-up care.

Keywords: Dilated cardiomyopathy, thyroid diseases, diabetes mellitus, diastolic and systolic dysfunction.

Introduction

Cardiomyopathy and hypothyroidism were first reported in the late 1918. Dilated cardiomyopathy is a heart muscle disorder with dilated and poor left ventricular function [1-5]. Dilated cardiomyopathy (DCM) is an idiopathic cardiac disorder, and its identifiable cause is still remaining unknown. DCM associated hypothyroidism causes elevated cardiac output, reduced peripheral vascular resistance, narrowed pulse pressure, increased mean arterial pressure, reduced left ventricular systolic function, and ejection fraction substantially projects the complicated cardiac events [2-4,6-13].

Case report:

A 75-year-old female patient was presented to the general medicine with chief complaints of shortness of the breath, swelling of legs, chest discomfort, severe constipation, fatigue and palpitation. The patient had the past medical history of type 2 diabetes mellitus and hypothyroidism. She had food habits of consuming fatty foods and sea foods regularly. The patient was subjected to the general examination which revealed that she was conscious, oriented and afebrile, having the pulse rate of 80 bpm, respiratory system of NVBS, blood pressure of 150/90 mm hg, cardiovascular sounds of S1, S2 which were normal, the soft P/A, and a normal CNS examination.

Chest X-ray of the patient detected the cardiomegaly, and the electrocardiogram reports showed the atrial fibrillation with fast ventricular rate. Echocardiogram investigations revealed the presence of dilated cardiomyopathy with severely dilated LA/LV/RA with decreased left ventricular systolic function, reduced left ventricular ejection fraction (LVEF) of 39% and left ventricular dysfunction. Aortic value sclerosis and atrial fibrillation were also present.

Clinical laboratory examinations exhibited Hb 9.5g/dl, PCV 28%, total cells 6800/cum, neutrophils 78%, eosinophils 4%, basophils 2%, monocytes 4%, red blood cells 4.8 x 10^6/mm^3, ESR 29/hr, MCV 82 fl, MCH 25 pg/cell, MCHC 31 g/dl and platelets count was 2,45,000 cells/cum. Her blood sugar levels reported FBS 140 Mg/dl, RBS 160 mg/dl, PPBS 180 mg/dl and HbA1C was found to be 7.2. Liver function test revealed elevated alkaline phosphatase 89 mu/ml, SGPT 33u/l, SGOT 42 u/l, total bilirubin 1.28 mg/dl, direct bilurubin 0.2 mg/dl, albumin 4.1 g/dl and globulin 2.9 mg/dl. Serum electrolytes include [Na^+] = 133 meq/l suggesting hyponatremia and [K^+] = 4.2 meq/l. [cl^-] = 106 mmol/l, HCO_3 = 22 mmol/l. Thyroid

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function test detected elevated thy¬roid stimulating hormone (89 µIU/ml), decreased serum levels of T3 (42 ng/dl) and decreased levels free T4 (0.049 ng/dl). Renal function test showed serum creatinine 0.9 mg/dl, blood urea nitrogen 29 mg/dl and uric acid 4.2 mg/dl. Her lipid levels were total cholesterol 220 mg/dl, HDL 29 mg/dl, triglyceride 188 mg/dl, very low density lipoprotein 120 mg/dl and low density lipoprotein 110 mg/dl.

From the above clinical investigations, the diagnoses confirmed that she was suffering from dilated cardiomyopathy associated type 2 diabetes mellitus and thyroid dysfunction. The patient was admitted to the cardiology unit for improvement of her condition and management of her diabetes and thyroid functions. She was advised to take low fat diet to control her blood cholesterol levels.

The patient underwent the treatment, and was prescribed with medications for her DCM and for the other complications. She was advised to physical exercises and walking regularly. She was sent to a clinical pharmacist to get counseling on her laboratory results, prescribed medications, food habits and regular aerobics to keep her condition under control. The patient was discharged from the hospital with discharge medications after her condition showed improvements. She was advised to a regular follow-up care on a scheduled basis.

Table 1: Patient's discharge medications

S.no.	Name of the Medication	Dose	Indications
1.	Tablet Digoxin	0.25 mg	Heart failure
2.	Tablet Eltroxin	50 mg	Hypothyroidism
3.	Tablet Atenolol	25 mg	High blood pressure
4.	Tablet Furosemide	20 mg	Edema
5.	Tablet Acitrom	2 mg	Blood clot treatment
6.	Tablet Metformin	500 mg	Diabetes mellitus
7.	Tablet Livogen	50 mg	Anemia
8.	Tablet Pantop	40 mg	Gastric problems
9.	Inj. Deriphylline Iv	2ml	Breathing problem
10	Syrup duphalac	15 ml	Constipation
11	Syrup Haem up	5ml	Iron deficiency Anemia

Discussion

Diabetes is a well-established risk factor for cardiovascular diseases. The progression of diabetes leads to systolic dysfunction, thereby causes the idiopathic cardiomyopathy [5, 6, 14-27]. The development of diabetic cardiomyopathy is linked to the hyperglycemia, insulin resistance, abnormal fatty acid metabolism, increased apoptosis and local renin angiotensin aldosterone system's excessive activation. Diabetes impairs the calcium ions' contractile functions, and prolonged action potential causes the slow decay of calcium from the cardiomyocytes [7,28-35]. The changes in the Ca^{2+} transient are responsible for the origin of the systolic ventricular dysfunction. Slowed decay in Ca^{2+} transient in diabetic cardiomyocytes initiates the reduced rate of Ca^{2+} removal from the cytosol and the reduced affinity of troponin C, as for the effect of diabetes on affinity of troponin C to Ca^{2+} which was shown by a study done by [8,36-42]. The Increased leakage of Ca^{2+} from the sarcoplasmitc reticulum has been reported as an abnormality in diabetic hearts by [9,43-44]. Diastolic dysfunction is a common finding in healthy and asymptomatic diabetic patients [10-13] and is stated to be the earliest identification of functional abnormality in diabetic cardiomyopathy suggested by [14]. In a study of normotensive, asymptomatic Type 2 diabetic patients with good glycaemic control, 47% were found to have diastolic dysfunction suggested by [15]. Other studies using more sensitive diagnostic methods have reported that as many as 75% of diabetic patients demonstrate abnormalities of diastolic function suggested by [16].

Hyperglycemia increases in oxidative stress by the mitochondrial generation of reactive oxygen species which damage DNA that contributes to apoptosis. Oxidative stress occurs due to the production of elevated reactive oxygen species, that leads to cellular damage by oxidation, disruption of vascular homoeostasis through interference with NO, and alters the intracellular signaling pathways. These reactive oxygen species were identified as the root cause of cardiac contractile dysfunction. A study done by [17] showed that the activation of renin angiotensin system promotes the release of Angiotensin II which acts on cardiomyocytes through AT1 receptors, and induces functional abnormalities in ventricular myocytes, and produces an abnormal contraction which can be prevented by prescribing Angiotensin II blockade as suggested by [18]. Advanced glycation end-products (AGE) contribute to endothelial dysfunction and atherosclerosis plaque formation which further increase the risk in diabetic patients. AGEs can easily make covalent cross-linkage with proteins, by this way they change the cardiac functions and exhibit the myocytes necrosis.

Dilated cardiomyopathy associated with decreased overall contraction properties of heart is irreversible and slowly progressive. The evidence suggests that the effects of thyroid hormone on heart are primarily via a change in protein synthesis. Thyroid hormone plays a significant role in modifying the cardiovascular system functions, and exerting several effects including the bradycardia, reduced chronotropy and inotropy with an increase in systemic vascular resistance, resulting in the increase in the afterload. Consequently, it will reduce the stroke volume and cardiac output, and the hypothyroidism condition can reduce oxidative metabolic pathways. These events may associate with the development of coronary artery disease [19]. Calcium ions reuptaken from the sarcoplasmic reticulum can be the determinant of the myocardial contraction and relaxation. It has been proposed that thyroid hormones can regulate the transcription of cardiac genes, including myosin heavy chain α, sarcoplasmic reticulum calcium ATPase, Na-K-ATPase, thereby modify the physiochemical properties of the myocardium. Thyroid hormone modifies the functions of ion channels and controls the mechanical events by activating the Na^{+}/K^{+}-activated ATPase enzyme. Clinically dilated cardiomyopathy and inversion of T wave may reflect the cardiac functions [20]. Non-genomic actions of thyroid hormone affect the cardiac myocytes which can regulate physiological functions of cells. Few evidence have suggested an association of hypothyroidism with increased oxidative stress secondary to reduced glutathione levels in the myocardial tissue, leading to direct myocardial damage. These effects can alter serum T3 levels, as cardiac myocytes are known to be unresponsive to changes in serum T4levels.This low T3 levels found to be a risk factor for the progression of cardiomyopathy [21, 22].

The prevention and management of diabetic cardiomyopathy with maintaining glycemic control, lifestyle modification and avoiding exposure to the risk factors and pharmacotherapeeutic strategies can help improve the treatment outcomes.

Conclusion

Diabetes is a known risk factor for developing cardiovascular diseases. The hyperglycemia is identified as a cause of pathogenesis of diabetic cardiomyopathy [23, 24]. The poor

glycemic control and improper medication adherence increase the disease complications. Early detection of the causative factors, treatment care initiation, patient follow-up care services and screening procedures can help reduce the mortality rate. Regular glycemic control, adherence to the medications and regular health care visits can reduce the hospital readmissions. Approaching novel diagnostic techniques and therapeutic strategies may improve the outcome in patients with diabetic cardiomyopathy [25].

The patient diagnosed with dilated cardiomyopathy with type II diabetes mellitus and thyroid dysfunctions should be given a clear knowledge on the complications of the disease [26]. Physician's care and clinical pharmacist's support can ultimately execute such services to the best of the treatment following an advanced health care guidelines of clinical practices could ultimately reduce the future occurrences and disease burden in the society.

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Conflict of interest

Authors declare no conflict of interest

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