The Egyptian Journal of Hospital Medicine (April 2018) Vol. 71 (6), Page 3390-3392

Dilated Cardiomyopathy and Hypothyroidism: A Case Report

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ABSTRACT

The exact mechanism of developing dilated cardiomyopathy DCM in hypothyroidism is still unclear.

The possible influence of hypothyroidism on the etiology and progression of DCM is controversial. We have experienced a case of a 32-year-old female with DCM secondary to hypothyroidism that was improved after hormone replacement therapy.

INTRODUCTION

Accelerated atherosclerotic changes and cardiovascular diseases are caused by thyroid dysfunction have been reported [(1).] It is well established that thyroid hormones alternate cardiac functions.

Hemodynamic changes in hypothyroidism includes bradycardia, decreased cardiac output by 30% to 50%, mild diastolic hypertension, narrow pulse pressure, slightly increased mean arterial pressure and an incensement in serum cholesterol and homocysteine [(2,3).] Fortunately, most of these cardiovascular changes are reversible if hypothyroidism was diagnosed and treated early; although rare, but dilated cardiomyopathies (DCM) has been well documented in patients with hypothyroidism.

A link between CMD and hypothyroidism was first reported in 1918 in four patients with heart failure refractory to digitalis and diuretics treated successfully with substitutive hormonal therapy [(4)].

CASE REPORT

A 32 years old female who is known as a case of sickle cell trait presented to the emergency room complaining about the difficulty of breathing for 1 month. The dyspnea, according to NYHA, it began with grade III but progressed to grade IV making the patient in a symptomatic case even at rest. Moreover, her dyspnea was associated with a dry cough, orthopnea, and PND over the last month as well.

To explain, she had a history of a dry skin, puffiness of the face, changing of voice tone, with increasing of weight and she also gave history of amenorrhea for the last 18 months. The patient had a history of irregular menstruation before 18\12 so she went for a medical advice when she discovered to have hypothyroidism. She was given thyroid replacement therapy for 3 months; after that, she stopped taking the medications on her own and she didn't go for following up since then.

On physical examination, the patient was on semi-setting position with puffy face, loss of lateral third of the eye brow, and coarse features. Her blood pressure was 101\95 mmHg, her pulse was 95/minute, her O_2 saturation was 98% on room air and respiratory rate was 24 per minute. Juglar venous pressure JVP was elevated measuring 7 cm from sternal angle, no goiter was appreciated. Chest examination revealed bilateral basal crackles. Apex was felt in the 6th Intercostal space anterior axillary line with pansystolic murmur grade 4/6 at the apex radiating to axilla, blowing in character. Abdominal examination was normal. There was a reduced deep tendon reflexes at the knee with delayed relaxation phase. Skin examination was shown as dry and rough skin with bilateral lower limb edema.

ECG showed low voltage, with poor R progression V1-V5. Chest x-ray reveled cardiomegaly with mild hilar congestion. Echocardiogram showed a significant dilation and global hypokinesia of left ventricle (LV) with severely impaired systolic function and ejection fraction (EF) of 20-25%. Right atrium RA, and left atrium LA were markedly dilated with severe mitral and tricuspid regurgitation were present with normal valves morphology. Right ventricular systolic pressure RVSP was about 40-45 mmHg. However, moderate pericardial effusion was noted.

Hematologic and biochemical profile showed leucopenia of 2.32 $x10^9$ /L. Hemoglobin was 11.4 g/dl. In peripheral blood film, there was a microcytosis and target cells. Her serum creatinine was raised 94 umol/L, but blood urea nitrogen BUN was normal. Serum calcium was low as 2.07 mmol/L. Furthermore, she had normal lipid profile, LDH of 235 U/L, Creatine Kinase (CK) of 333 U/L, and her ALT was elevated with 79,32 U/L. The patient had significant elevated level of thyroid stimulating hormone (TSH) of (100 µIU/mL), decreased serum levels of free T4, free T3 (0.436 pmol/L) and (1.56 pmol/L) respectively, establishing a diagnosis of primary hypothyroidism.

Received:17 /2 /2018
Accepted:27 /2 /2018

DOI**: 10.12816/0047290**

In addition, vitamin D level was low as 3 ng/ml. Since the patient was known for sickle cell trait, Hb electrophoresis showed decreasing levels of HbA: 68.7 and elevation of HbS: 27.5 confirming the patient condition of sickle cell trait(HbAS).

Sickling test was positive as well, other hormonal and serological tests were negative. The patient was treated with substitutive hormonal treatment with thyroxin at an initial dose of 50 mcg then it progressively increased until a dosage of 100 mcg/day. Following up after one month showed a significant improvement of her thyroid profile, her TSH dropped from 100 µIU/mL to 40 µIU/mL, Free T3 and free T4 levels were normalized up to 2.62 pmol/L and 16.46 pmol/L respectively. After a period of 6/12 months, mild improvement of LV systolic function EF 30-35%, no pericardial effusion was noted and patient became symptoms free.

DISCUSSION

Dilated cardiomyopathy is defined as a progressive condition affecting the myocardium leading eventually to impaired ventricular contractility. Although, it is considered as an idiopathic condition but it was found that the factors like genetic, infections, pregnancy, alcohol consumption, and endocrine disorders in particular " thyroid disorders" are some of the causes in developing reversible DCM [6].

The impact of thyroid disorders upon cardiovascular changes has been widely studied. It is believed that thyroid hormones have an anti-atherogenesis effect. They have a direct vascular effect by promoting vasodilatory substances as nitric oxide (NO), and inhibiting the expression of angiotensin II receptor which plays a role in vasoconstriction [7]. Moreover, they stimulate the breakdown of LDL and regulate cholesterol level by activating the catalytic enzyme HMG-CoA reductase [1]. Consequently, patient with hypothyroid may suffer from premature atherosclerosis. Indeed, secondary hyperlipidemia, hypertension and coronary artery diseases have been reported in hypothyroid patients. A study on the correlation of hypothyroidism and secondary dyslipidemia was done in mayo clinic on 268 patients and it showed that 91.4% of whom were hypothyroid had secondary dyslipidemia [8]. Cardiovascular risk in hypothyroidism is not only due to dyslipidemia, but other metabolic factors as some increased levels of homocysteine, decreased fibrinolytic activity, endothelial dysfunction and hemodynamic changes as bradycardia, decreased cardiac output by 30% to 50%, mild diastolic hypertension, narrow pulse pressure [2]. All of these factors carry a great risk thrombotic event and cardiovascular diseases [1]. It has been proposed that there is a direct effect of thyroid hormones on the heart. They exert their effect by alternating the synthesis and transcription of the protein responsible for relaxing the myocardium, and regulation of electromechanical properties by activating ionic channels of the heart. Hence, patients with hypothyroidism have an impaired left ventricular (LV) diastolic function and an increase in systemic vascular resistance [9,10]. **Fazio's** studied the effect of thyroid hormones on the heart and mentioned that the heart is sensitive for any changes in the levels of thyroid hormones, and it responses to even minimal but persistent changes in the levels of thyroid hormones as in subclinical hypothyroidism [11]. This suggests that deficiency in thyroid hormones alters cardiovascular functions. **Kumar** considers DCM as a rare presentation of hypothyroidism [12]. On the other hand, **Marovic** found that 18%-49% of patients with dilated cardiomyopathy had an associated disorder in their thyroid gland [13].

In our current case, the problem began after a year and a half for the history of amenorrhea. Then the diagnosis of hypothyroidism was made and she was given hormonal replacements but she was not compliment to it. Three months after stopping the treatment, she began experiencing the classical hypothyroid signs and symptoms plus dyspnea. The patient did not have any family history of DCM or thyroid disease but she had a positive history of sickle cell disease. Although, Hemosiderosis-induced cardiomyopathy has been observed in older sickle cell affected patients but our patient is a sickle cell trait. There was no history according to alcohol addiction nor substance abuse. Our patient's clinical, laboratory and radiological findings showed a dilated LV with hypokinesis, giving the diagnosis of DCM, which is attributed to her thyroid hormonal deficiency.

Most of the reported cases had a similar clinical presentation consisting of progressive dyspnea which was the main chief complain. In addition to the hypothyroidism changes and high level of TSH, Echocardiogram showed similar changes in all patients consisting of reduced ejection fraction, global hypokinesia and LV enlargement. Surprisingly, we found out that if the treatment of hypothyroid state was initiated, a substantial improvement in the overall cardiac functions is achieved.

Singhai reported a case of a 40 years female who was diagnosed with DCM secondary to

her severe hypothyroidism after presenting with dyspnea and lower limb edema [16]. Her EF was as low as 15% and TSH was >100mU/l. but only after six months of starting thyroxine, her EF increased to 40% and she had an excellent improvement. Similarly, **Bhardwaj** reported a 36 years old female who presented with featured of severe hypothyroidism in addition to progressive dyspnea. EF was 20% with LV enlargement, and TSH>60mU/I and was diagnosed with DCM. Thyroxine was given and the patient became free from the symptoms [10].

Even though hypothyroidism is common in females but **Seol** reported a 36 years old male who presented with classic features of congestive heart failure with EF 16%. His thyroid panel showed TSH>100mU/I. He was diagnosed with Hashimoto's thyroiditis and was treated with thyroxine. The patient improved and had a normalization of clinical and laboratory findings [14].

The reported cases did not only vary in gender, but age as well. Cases of young adults presented with DCM secondary to hypothyroidism were reported. Moreover, **Ladenson** reported a 22 years old male who presented with a picture of heart failure, his investigation revealed severe hypothyroidism, hormonal replacement therapy that were given in less than one year, after this period, the patient had a remarkable improvement [15]. Similarly, **Kumar** reported a 14 years old female who had similar cardiac presentation; she was diagnosed with Hashimoto thyroiditis [12].

Since restoration of euothyrodism state by levothyroxine therapy proved its ability to reverse the cardiovascular abnormalities in the previously mentioned report cases, our patient as well had an excellent response to the hormonal therapy. Indeed, this study was done by **Fazio**, who mentioned that the treatment by levothyroxine, especially among those with high serum TSH levels, might correct the dyslipidemia and overall cardiac functions [11].

Our patient's presentation and course strongly indicates that the etiology of her heart muscle disease was secondary to her hypothyroidism disease.

CONCLUSION

In conclusion, DCM is a progressive dilation of the heart with impairment of contraction. When it is idiopathic, it's usually an irreversible condition except if the cause is known as our case, which showed a reversible form of DCM that is secondary to the thyroxine deficiency. Thus, we highly recommend healthcare workers to have a strong clinical subsection of a secondary cause in patients who present with a picture of dilated cardiomyopathy.

REFERENCES

1- **Neves M (2008):** Thyroid Diseases, Dyslipidemia and Cardiovascular Pathology, Rev Port Cardiol., 27 (10): 1211-1236

2- **Danzi S, and Klein I (2004):** Thyroid hormone and the cardiovascular system. Minerva Endocrinologica, 29: 139–150.

3- **McAllister RM, Delp MD, and Laughlin MH (1995):** Thyroid status and exercise tolerance. Cardiovascular and metabolic considerations. Sports Med., 20:189–198

4- **Pöppl (2015):** Dilated Cardiomyopathy in a Fila Brasileiro with Hypothyroidism Secondary to Lymphocytic Thyroiditis, Acta Scientiae Veterinariae, 43(1): 102

5- **Zondek H (1918):** Das myxodemherz. Munch Med Wochenschr, 65:1180-83

6- **Singhai (2014):** A Case Report of Reversible Dilated Cardiomyopathy, Heart India, http://cutt.us/3YjQM

7- **Ichiki T (2010):** Thyroid hormone and atherosclerosis. https://www.ncbi.nlm.nih.gov/pubmed/19808101

8- **Hoefler G, Harnocourt F, Paschke E, Mirtle , Pfeiffer KH, Kostner GM and Lipoprotein Lp (1988):** A risk factor for myocardial infarction. Arteriosclerosis, 8:398-401.

9- **McAllister RM, Delp Mand Loughlin MH (1995):** Thyroid status and exercise tolerance. cardiovascular and metabolic consideration. Sport Med., 98-20:189

10- **Bhardwaj** A **(2009):** Hypothyroidism Presenting as Dilated Cardiomyopathy, MJAFI., 65: 284-286

11- **Fazio E(2004):** Effects of thyroid hormone on the cardiovascular system, Recent Prog Horm Res., 59:31-50.

12- **Kumar K, Tripathy PR, Krishna K, Jammula S, Kumar M and Modi K (2012):** Primary hypothyroidism: uncommon presentation with reversible dilated cardiomyopathy in a young subject. Int J Endocrinol Metab., 10:440-3

13-**Marković (2005):** đurica, dilated cardiomyopathy associated with autoimmune disease of thyroid gland. BIBLID., 133(1):46-51

14- **Seol R(2014):** Dilated Cardiomyopathy Secondary to Hypothyroidism: Case Report with a Review of Literatures, J Cardiovasc Ultrasound, 22(1):32-35

15-**Ladenson A(1992):** Reversible alterations in myocardial gene expression in a young man with dilated cardiomyopathy and hypothyroid, Medical Science ,89:5251-5255.