Case Report

Thyrotoxic cardiomyopathy - A case report

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Abstract

Cardiomyopathy is an uncommon presentation in hyperthyroid patients. There are very few case reports of thyrotoxic cardiomyopathy. The mechanism due to which cardiomyopathy occurs in hyperthyroid patients is not very well understood. After extensive literature search, it was found that some of the mechanisms described which include genomic, non-genomic and direct action of Thyroid hormone on the cardiac muscle may cause cardiomyopathy. In this case report, a case of Multi-nodular goitre with cardiomyopathy is described.

Key words

Thyrotoxic cardiomyopathy, Multi-nodular goitre, Thyrotoxicosis, Genomic, Non-Genomics.

Introduction

There are very few cases of documented thyrotoxic cardiomyopathy. Cardiomyopathy as an initial presentation has been reported in 6% of hyperthyroid patients [1] while less than 1% of them developed dilated cardiomyopathy with severe left ventricular dysfunction [2]. The mechanism due to which cardiomyopathy occurs in hyperthyroid patients is not very well understood. After extensive literature search, it was found that some of the mechanisms described below which include genomic, non-genomic and direct action of Thyroid hormone on the cardiac muscle may cause cardiomyopathy. A case of thyrotoxic multi-nodular goiter with cardiomyopathy is described in this case report.

Case report

A 46 year-old non-diabetic, non-hypertensive female presented with history of chest pain associated with sweating for twenty days prior to admission. Patient was apparently asymptomatic twenty days ago, and then developed left sided chest pain, which was non-radiating and associated with sweating. The chest pain was not

associated with shortness of breath, paroxysmal nocturnal dyspnea, pedal edema, cough or fever. She also complained of swelling over neck of 1 year duration, loss of weight of 4 month duration, and generalized weakness of 20 days duration. She had no similar complaints in the past and had no history of coronary artery disease/Bronchial Asthma/Tuberculosis/Chronic obstructive pulmonary disease. She had never smoked and never consumed alcohol. She has three children and attained menopause three years ago.

On examination, she was thin built; pale, with no icterus, no pedal edema. Among vital signs; Temperature was 98.6, pulse rate (PR) was 90/min, regular, blood pressure (BP) was 130/80mm Hg, respiratory rate (RR) was 16/min. Cardiovascular system examination showed jugular venous pressure (JVP) was not elevated. Cardiomegaly was present, apex in 6th left inter costal space (ICS), in anterior axillary line, forceful, S1, S2 heard normal, Ejection Systolic Murmur heard over pulmonary area, no other murmurs or rub, Respiratory System was normal, Abdomen was soft and there were no masses. Central nervous system showed generalized wasting of muscles, no tremor, deep tendon reflexes (DTR): 3+ bilaterally. Thyroid examination showed swelling in the neck - moving with deglutition, multinodular, Right lobe: 7 x 4 x 4 cm, Left lobe: 6 x 3 x 2 cm, small swelling over isthmus size 1x1 cm, and no bruit heard over the swelling (**Figure - 1**). All other systems were normal.

Her investigations revealed as below. Among Complete blood picture (CBP), Hemoglobin was 8.5 gm%, Red Blood Cell (RBC) count was 3.71 million/cumm, Packed Cell Volume (PCV) was 27.4, Total Platelet Count (TPC) was 1.9 Lakh/cumm, Total leukocyte count (TLC) was 7500/ cumm, N_{60}, L_{35}, M_3, E_3, $B_{0,}$ ESR was 20 mm, Blood Urea Nitrogen (BUN) was 9 mg%, Serum Creatinine was 0.6 mg, Complete Urine Examination (CUE) – NAD, Random Blood Sugar (RBS) was 103 mg/dl, Serum Electrolytes: Na – 143 mmol/L, K - 3.6 mEq/L, CL – 109 mEq/L, X-ray Chest PA view showed cardiomegaly, Right Ventricular type, ECG (**Figure - 2**) showed LBBB with LVH, 2D ECHO showed Global hypokinesia of Left Ventricle with EF – 62%, mild TR/MR + mild PAH, LV dysfunction+, no PE/Clot, Viral Markers showed HIV – negative, HbsAg - negative, HCV - negative. Coronary angiogram revealed Normal Coronaries. T3 - 188.40 ng/dl (60-180), T4 - 13.6 µg/ dl (7.3-15), TSH – 0.01 µIU/L (0.55-4.78). Ultrasonography of thyroid showed right lobe was 6.9 x 3.3 x 3.3 cm, enlarged and heterogeneous echotexture, Left lobe was 6.0 x 2.6 x 2.1 cm, enlarged and heterogenous echotexture. Evidence of non-homogenous hyperecohic lesion with micro-calcification and increased vascularity seen in both lobes. Isthmus - 10 mm, no evidence of lymph nodes seen, Submandibular and Parotid gland appear normal, Great vessels appear normal. Final impression was inhomogeneous hyperecohic lesions with microcalcifications and increased vascularity seen in both lobes.

Figure - 1: View of thyromegaly.

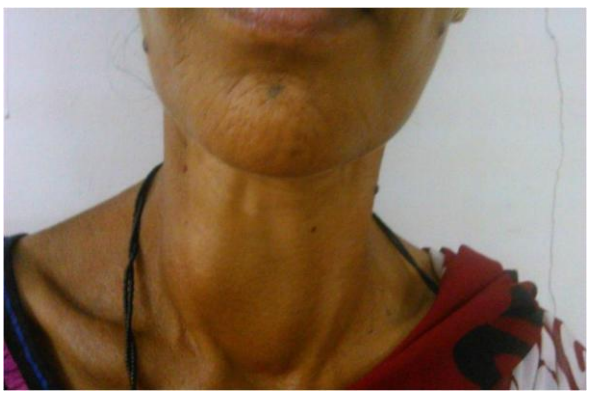
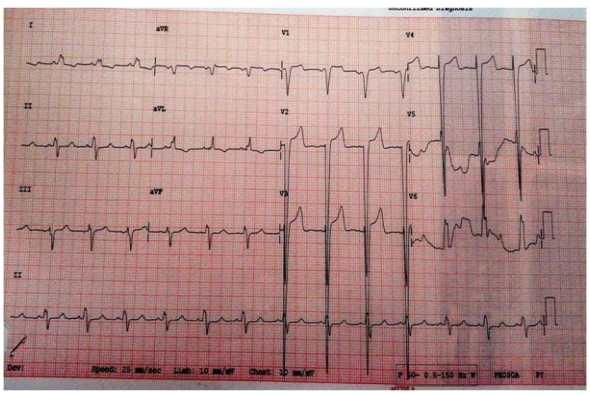


Figure - 2: ECG changes.



Fine Needle Aspiration Cytology (FNAC) of thyroid revealed moderate cellularity comprising of thyroid follicular cells, arranged in monolayered sheets, clusters and follicles against a background of hemorrhage and thick colloid. Numerous hemosiderin laden foamy cystic macrophages are seen in cohesive clusters. Hyperplastic and involutional follicular cells are seen. Anisonucleosis is seen in hyper follicuar cells. Final impression was features suggestive of Multi nodular goiter.

Patient was kept on Tab. Propythiouracil 100 mg/day.

Discussion

Hyperthyroidism causes cardiac complications in structurally normal hearts, in patients with pre-existing cardiac disease and may unmask the silent CAD or compensated heart failure. Cardiomyopathy and CHF due to hyperthyroidism are not common [3]. The reason why some patients develop Hyperthyroid Cardiomyopathy and advanced heart failure remains unknown.

Some of the mechanisms described below which include genomic, non-genomic and direct action of Thyroid hormone on the cardiac muscle may cause cardiomyopathy [4, 5, 6, 7].

Molecular and cellular mechanism of thyroid hormone action on the heart

Dual mechanism

- **Direct effect on the transcription of [4]**:
  - Specific genes
  - Non-specific genes
- **Non-Genomic action on**
  - Plasma membrane
  - Mitochondria
  - Sarcoplasmic reticulum

V1 – Isoform: It is activated by Thyroid hormone and causes:

- Increased ATPase activity
- Increased velocity of muscle fiber shortening
- Increased muscle contractility, small heart,
- Increased heart rate in hyperthyroidism

Non-specific genes

Thyroid hormone

- Upregulates SERCA gene (Sarcoplasmic Reticulum Calcium ATPase) [8]. Regulates rate of myocardial contraction and relaxation.
- Downregulates Phospholambam protein expression [9]. Allows an accelerated reuptake of calcium by the sarcoplasmic reticulum resulting in increase in cardiomyocyte peak tension development, shortening of the duration of contraction in ventricular muscle.

This mechanism explains the improvement in diastolic relaxation properties of the hyperthyroid heart .Thyroid hormone does not increase the sensitivity of left ventricular contractility to beta-adrenergic stimulation. Hence, beta-blockade by beta- blockers as treatment is useless.

Non-genomic action on plasma membrane

Na^{++} channel

- Prolongs inactivation of Na^{++} channels
- Increases intracellular uptake of Na^{++}
- Increases secondary activation of the sarcolemmal Na^{++} - Ca^{++} exchange
- Increases inotropic activity of thyroid hormone

Ca^{++} channel

Inj. T3 has direct effect on L-type of Ca^{++} channels, enhances the Ca^{++} entry into myocytes, and causes rapid onset of ↑ cardiac output.

All diastolic functions are decreased

- Left ventricular relaxation time is decreased
- Diastolic flow velocity is decreased
- Isovolumetric relaxation time is decreased

All the above diastolic parameters become normal when the hyperthyroid patient becomes euthyroid with treatment.

Specific genes controls

Sr. No	Myosin isoforms	Controlling gene	Effect of thyroid hormone
1	V - 1	MHC α / α (↑ Rate of myosin synthesis)	Activates
2	V - 2	MHC α / β	
3	V - 3	MHC β / β (↓ Rate of myosin synthesis)	Represses

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