Hypothyroid Cardiomyopathy: A Case Report

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Introduction:

Dilated Cardiomyopathy in the young patient poses an aetiological diagnostic challenge, since detection of a treatable lesion is always a priority. With tremendous improvement in the Cardiac imaging techniques, structural heart disease is less of a problem these days as compared to metabolic and biochemical factors leading to cardiac decompensation, the identification of these is vital for timely treatment, which may salvage myocardial function.

Abstract:

A young lady with symptoms of partially treated postpartum pitutary failure over a period of three years and intermittent paroxysmal nocturnal dyspnoea and ankle swelling for six months presented with a worsening of her symptoms due to a pneumonic illness complicated by disseminated intravascular coagulation. She was found to be in biventricular failure and with treatment of her acute illness her cardiac status improved. However, subsequent search for the cause of her dilated cardiomyopathy was unrevealing, but since she was overtly hypothyroid secondary to her postpartum failure it was reasonable to ascribe her cardiac status to Hypothyroidism, for which she received treatment and showed signs of improvement initially. A review of the literature on the subject is presented.

Case Report:

A 34 years old lady presented to the medical unit of this hospital in May 1997. She had delivered a female baby in May 1994 with significant postpartum haemorrhage and subsequently remained ammenohric, failed to lactate and had scant growth of pubic and axillary hair. A year later she became lethargic, intolerant to cold with infrequent bowel movements, a change in her voice was noticed. Despite the obvious presentation a diagnosis of postpartum pitutary failure was not made. Six months prior to presentation she became progressively dyspnoeic NYHA 11-111 and complained of swollen feet. She was treated by a "Hakim" during this time with no record of medication and was put on 100mg of Thyroxine a month prior to presentation, without a baseline profile, following which she experienced palpitations recurrently. A week prior to presentation she became febrile, and had cough productive of purulent sputum with a tendency to bruise easily. At presentation to the hospital she was distressed and pale with widespread ecchymois, a regular pulse of 80 per minute, blood pressure of 90/60 mmhg with no postural drop, a normal wave form jugular venous pressure upto the angle of the jaw, she was not cyanosed but had demonstrable ankle oedema. Physical findings other than that were suggestive of congestive failure as evidenced by an S3 gallop, apical ejection systolic murmer, bilateral basal crackles and a smoothly enlarged liver. Neurological examination revealed the classical slow relaxation of the ankle jerk. The baseline lab profile is shown in the table. Her chest X-ray showed a patch of consolidation in the right lower zone with an enlarged cardiac silhouette, ECG showed poor R wave progression across the chest leads. Trans thoracic 2D echocardiogram showed a globally hypokinetic left ventricle with an ejection fraction of 20%.

An initial diagnosis of peripartum Pitutary failure with low grade disseminated intravascular coagulation secondary to pneumonia and dilated Cardiomyopathy of undetermined nature was made, she was treated with

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third generation Cephalosporins and did not require any replacement therapy for her DIC. In view of her persistent hypotension and cardiac failure she was subsequently shifted to the Coronary Care Unit for inotropic support, where she improved over the ensuing week. Her echo 15 days later showed a marked improvement in her ejection fraction which increased to 43%, however subsequent echocardiograms done at the intervals of 1 and 2 months did not show this trend in the improvement of the ejection fraction.

The patient is currently on Thyroxine and Prednisolone replacement, an ACE inhibitor and a small dose of a diuretic. Subsequent investigations to define the cause of the dilated cardiomyopathy were exhaustive and unrevealing. Valvular disease was ruled out with 2D and Doppler echo. Rest thallium-201 scan showed no segmental abnormalities. Toxic and infective screen was negative, likewise serum ACE and ferratin levels and collagen profile were normal.

In our opinion this lady suffered from Hypothyroid Cardiomyopathy, the stress of the chest infection and DIC tipped her into imbalance and she presented with acute gross left ventricular decompensation, which improved as her acute infective state came under control. She subsequently remained in a state of partially compensated cardiac failure, which we attribute to her Hypothyroid state.

Discussion:

The heart is a major target organ for thyroid hormone action and marked changes in cardiac function occur in patients with hyper or hypothyroidism. Cardiac contractility is increased in the hyperthyroid state and decreased in the hypothyroid state with changes in the specific proteins mediating cardiac contraction accompanying these alterations. Changes in thyroid status mediate their influence on cardiac function by a combination of direct thyroid hormone effects on the heart, alteration in the responsiveness of the cardiac sympatho-adrenal system and hemodynamic effects generated in the periphery.[1] Thyroid hormone effects on myocardial gene expression have been well defined in animal models, but their relationship to the pathogenesis of myocardial dysfunction in the hypothyroid state in humans remains uncertain. Recent studies on profoundly hypothyroid men with dilated hearts has shown that the alteration in gene expression in the dilated myopathic heart maybe correctable when a treatable cause is sought.[2]

Cardiovascular involvement in Hypothyroidism manifests in many ways, amongst them pericarditis, electrocardiographic abnormalities, conduction abnormalities and hypertension are well known. Hypothyroidism also leads to cardiac decompensation in the form of a reversible hypertrophic or dilated cardiomyopathy,[3,4] dilated cardiomyopathy may be asymptomatic or manifest itself as a compromise of systolic function and hypertrophic cardiomyopathy as an asymmetrical septal hypertrophy which is usually asymptomatic. Contrary to general belief ASH is not invariably present in hypothyroidism, but its presence in certain cases of hypothyroidism suggests that the application of positive pressure inotropic and afterload reducing agents may invoke deleterious effects and should be considered hazardous in the treatment of hypothyroidism even when indicated by concurrent other heart diseases.[5] This may have important therapeutic implications regarding the usage of inotropes in cases of dilated cardiomyopathy in the setting of hypothyroidism, and therefore echo can give very useful information regarding the morphological type of the underlying cardiomyopathy.

The increased incidence of angina in the setting of hypothyroidism is attributed to the underlying Coronary Artery disease, which is related to the accelerated atherosclerosis and the tendency to hypertension, it is also attributed to the reversible anatomical narrowing of the coronary arteries and the reversible endocrine cardiomyopathy. In severe hypothyroidism thyroid replacement therapy precipitates or aggravates Angina Pectoris, whereas in others Angina Pectoris is ameliorated or even disappears. The reason for this paradox is not known, it has been attributed either to the reversible nature of the endocrine cardiomyopathy in the form of Asymmetric septal hypertrophy (ASH) or reversible anatomical narrowing of the coronary arteries.

Studies with radionucleotide ventriculography and spect nuclear imaging assessing myocardial perfusion and performance conclude that reversible coronary dysfunction or reversible anatomical narrowing of the coronary vessels is not an infrequent manifestation of

severe hypothyroidism.[6]

Diagnosis is based on the morphological identification of the cardiomyopathy whether ASH or dilated in the setting of hypothyroidism and by the exclusion of other causal factors. Studies have shown that whereas myosin light chain 1 assessment is not helpful in clinical assessment of patients with idiopathic dilated cardiomyopathy, it is useful in the detection of secondary cardiomyopathy specifically in hypothyroid cardiomyopathy, where it is related to decreased clearance of normal LC-1 concentration or increased myosin light chain in the skeletal muscle, it may therefore be useful in the detection of hypothyroid cardiomyopathy.[7]

Treatment in addition to Thyroxine replacement is largely supportive and symptomatic with the hope and knowledge that the endocrine cardiomyopathy will reverse.

Investigations:

Test	Result
Hemoglobin	13g/dl
WBC	17000/mm^3(admission) 9000/mm^3(discharge)
Platlets	27,000/mm^3(admission) 135,000/mm^3(discharge)
Urea	45 mg/dl
Creatinine	1.5 mg/dl
Bilirubin	2.9 mg/dl
SGOT	209 IU/1
Creatinine Kinase	351 IU/1
LDH	415 IU/1
Prothrombin time	25 (admission) 14 (discharge) (control:13)
APTT	136(admission) 40 (discharge) (control:32)
FDP	40 IU/1 (admission) 10 (discharge) (normal: 10-40)
Serum T3	0.90 (66-1.8)
Serum T4	7.1(3.2-12.6)
Serum TSH	38 (.35-5.50)
Serum FSH	0.02 miu/ml (3-15)
Serum LH	.4 miu/ml (2.5-9)
Serum cortisol	64.73 4 pm (3.09-16.60)

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