Case report

Reversible Dilated Cardiomyopathy in Hypothyroidism

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ABSTRACT

Introduction:

The concept of reversible dilated cardiomyopathy is rare in hypothyroidism is yet a matter of debate although many theories have been postulated with this regard. We report overt heart failure due to dilated cardiomyopathy in an elderly lady with nonsignificant coronary artery disease and hypothyroidism. Early suspicion, evaluation and judicious use of thyroxine with appropriate anti ischaemic measures proved beneficial with a better prognostic outcome in this patient

Introduction:

Thyroid hormone has many effects on the heart and vascular system.[1] The heart is very sensitive to alterations in serum thyroid levels. Many of the clinical manifestations of hyperthyroidism and hypothyroidism are due to the ability of thyroid hormone to alter cardiovascular hemodynamics.[2]

Cardiac manifestations of thyroid hormones are due to dyslipidemia, accelerated atherogenesis, reduced heart rate, contractile states of myocardium and pericardial effusion. Nonspecific histological abnormalities have been demonstrated repeatedly in the hearts of myxoedema patients since first reported in1888 in report of a committee of the Chemical Society of London.[3]

The structural changes together with haemodynamic changes in heart of a hypothyroid patient termed as hypothyroid cardiomyopathy [4,5,6] has shown a good response to thyroxine replacement.

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Ischaemic cardiac events have also been implicated in causing transient thyroid dysfunction. But whether the cardiomyopathy associated with both ischemic heart disease and hypothyroidism are interrelated is still a matter of debate as significant improvement has been seen in patients treated concurrently for the two different conditions

Case report:

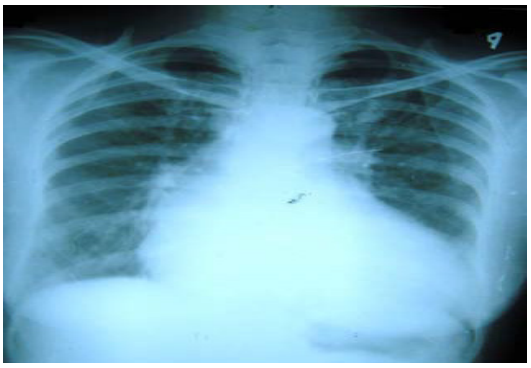
A 61 year old lady presented to the emergency department of a tertiary institution with exertional dyspnoea since 1 week with no documented medical history. On examination the patient was mildly cyanosed, pulse rate 120 beats per minute; regular rhythm, blood pressure 110\90 mmHg, respiratory rate 32 cycles/min, saturation of O2 85% with elevated JVP. Cardiovascular examination showed tachycardia with gallop rhythm and bilateral basal crepitations. A clinical diagnosis of heart failure was made. All preliminary investigations were within normal range except the lipid profile which was altered with total cholesterol-220mg /dl, LDL cholesterol - 120 mg/dl, HDL- 40 m/|dl, TG -180 mg \dl with normal values of CPK and Troponin-I. Electrocardiogram showed sinus tachycardia with no ST-T changes. Chest x-ray showed cardiomegaly (Figure 3 a) with pruning of upper lobar veins and peri hilar congestion. 2D

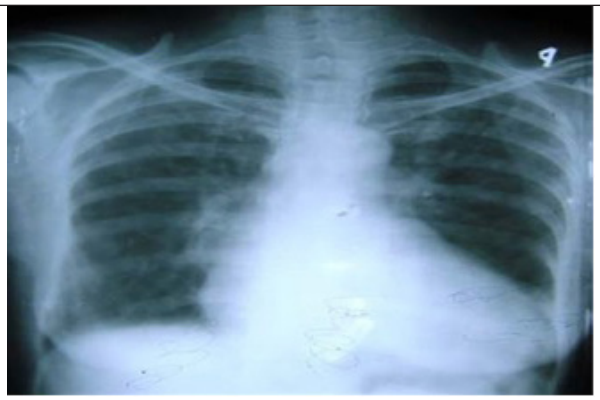
Echocardiography showed apical hypokinesia with an inter ventricular septal thickness of 8.1 mm, mild mitral regurgitation, minimal pericardial effusion and an left ventricular ejection fraction (LVEF) of 37%. She was decongested with diuretics and recovered symptomatically. An emergency coronary angiography was performed, which revealed a single vessel disease with blocks of–left anterior descending (LAD) 30 %. Respecting her LVEF of 37% she was subjected to conventional treatment. Myocardial perfusion study showing mild perfusion defects in anterior wall. As there is no correlation between her symptoms and severity of CAD, we are evaluating for other medical disorders and later she was diagnosed as hypothyroid and she was treated with antiplatelets, diuretics, ACEI, thyroxine.

She was started cautiously with Levothyroxine 0.25 micro gram per day which was gradually built upto a dose 0.1 mg over 3 weeks duration. Following this treatment she showed significant improvement in her symptoms and was later discharged after fixing the dose of thyroxine at 0.1mg/ day.

During serial follow ups, 4 months after her discharge, she was asymptomatic, active and able to carry out her routine activities She was reinvestigated to study her present status and therapeutic response. Her chest x-rays (figure 3 b, c) taken then showed significant reduction in cardiac size. Echocardiography showed improved LVEF to 55%.

Her lipid profile and thyroid function test were also within normal ranges. She was continued on her medications and has been doing well till date.





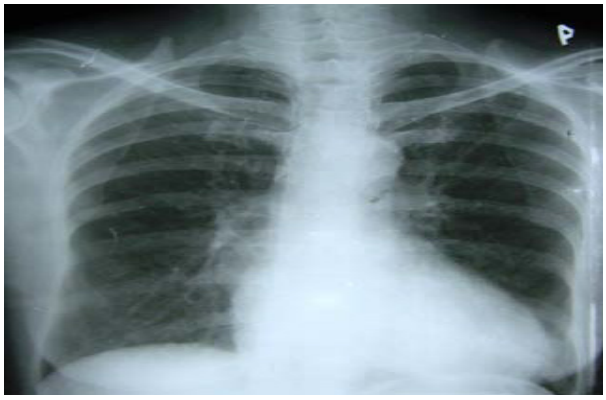


Fig a,b,c :Serial chest radiographs of patients of patients at time of admission and serial follow-ups.

Discussion

Thyroid hormone has many effects on the heart and vascular system. Many of the clinical manifestations of hyperthyroidism and hypothyroidism are due to the ability of thyroid hormone to alter cardiovascular hemodynamics. The hemodynamic effects of hypothyroidism are opposite to those of hyperthyroidism, although the clinical manifestations are less obvious. Prompt evaluation here made the diagnosis of congestive cardiomyopathy. We believed this to be related to the underlying coronary pathology with respect to her altered lipids, low ejection fraction, poor LV systolic function and apical hypokinesia supported with coronary angiogram. Radionuclide tech 99 resting myocardial perfusion scan revealed significant perfusion defects with viable myocardium. PET studies of O2 consumption in patients with hypothyroidism have revealed that myocardial work efficiency is lower than in normal subjects.[7] Significant dyslipidemia in a slim elderly

patient (BMI-20) prompted us to investigate her thyroid status as such an accelerated coronary atherosclerosis due to hypercholesterolemia in hypothyroidism and post menopausal ladies has already been postulated Some theories have explained overt hypothyroidism to occur following an acute coronary event or acute myocardial infarction, but the phenomena is a sub clinical state of hypothyroidism and in heart failure, patients have low serum T3 concentration and the degree is proportional to severity of heart failure as per NYHA functional classification.[8] We were in a dilemma as to whether the heart failure has depressed the thyroid hormones or hypothyroidism per se is only the true cause for this cardiomyopathy. Here significant elevation of TSH more than 150 and significant reduction in T3 and T4 made the diagnosis of hypothyroid cardiomyopathy. We had initially thought of IV T3 as an immediate therapy to tide over this crisis but due to its non availability we treated this patient cautiously with thyroxine initiating with the lowest possible dose, gradually building up the dose to a maximum of 0.1 mg within 6 weeks. In fact risk versus benefits with thyroxine therapy in elderly patients with concomitant coronary artery disease were thought seriously as thyroxine is known to improve the cardiac contractility and reduce the peripheral vascular resistance and has no effect in improving the LVEF .Theories have explained maximum beneficial effects of thyroxine in patients who were diagnosed to have heart disease in long standing hypothyroidism*but in our case it was a risk as patient was tachycardic. Many of the patients with severe heart failure in hypothyroidism with significantly compromised LVEF, poor LV systolic function and a jeopardized myocardium are expected to have prolonged QT interval and abrupt initiation of thyroxine therapy in them may culminate with torse de pointes, ventricular arrhythmias and a premature sudden cardiac death. Whether the thyroid condition in this case was a separate preexisting entity precipitating the underlying cardiac events or whether it was precipitated by the cardiac event was yet to be explained.

Conclusion

Here we reported a rare case of reversible dilated cardiomyopathy of hypothyroidism .

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