

# Restoration of sinus rhythm following levothyroxine treatment in a case of primary hypothyroidism presenting with atrial fibrillation and pericardial effusion

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### ABSTRACT

A 72-year-old man presented with palpitation, dyspnea, and chest discomfort. Initial investigations revealed atrial fibrillation (AF) and pericardial effusion, further investigations unraveled primary hypothyroidism (thyroid stimulating hormone) of 34.7  $\mu$ IU/ml and total thyroxine (T4) of 5.57  $\mu$ g/dl). Treatment with levothyroxine led to resolution of symptoms, AF, and pericardial effusion.

**Key words:** Atrial fibrillation, primary hypothyroidism, pericardial effusion

## INTRODUCTION

Hyperthyroidism is commonly associated with atrial fibrillation (AF), particularly in the elderly. Association of hypothyroidism with AF is less well recognized. Association of AF, pericardial effusion, and hypothyroidism have not been reported.<sup>[1]</sup> We hereby report a case of patient with primary hypothyroidism presenting with AF and pericardial effusion which resolved with levothyroxine therapy.

## CASE

A 72-year-old male presented with palpitations, dyspnea, and chest discomfort. He had episodes of “fast heart

beats” for 3 months. He complained of chest pain which was substernal, nonradiating, and throbbing in nature. He had no other positive history suggestive of any other disorder. He smoked 10 cigarettes per day for past 25 years, and denied use of alcohol, caffeine, or drugs. Physical examination was unremarkable except for the irregular heart rate (approximately 130 beats per min) and muffled heart sounds on auscultation. Electrocardiogram [Figure 1] confirmed AF with rapid ventricular rate, which responded to initial treatment with metoprolol [Figure 2].

Thyroid function tests revealed a sensitive thyroid stimulating hormone (TSH) concentration of 34.70  $\mu$ IU/ml (normal: 0.40-4.70  $\mu$ IU/ml), and thyroxine (T4) of 5.57  $\mu$ g/dl (normal: 8-12  $\mu$ g/dl) confirming primary hypothyroidism.

Anti-thyroperoxidase antibodies were negative. His blood counts, serum electrolytes, and serial cardiac enzymes were normal. Chest X-ray revealed mild cardiomegaly. Transthoracic echocardiography showed normal left ventricular systolic function (ejection fraction of 63%), decreased left ventricular diastolic compliance, and massive pericardial effusion (posterior 17 mm, anterior 13 mm). Right atrial and right ventricle showed 30% collapse during

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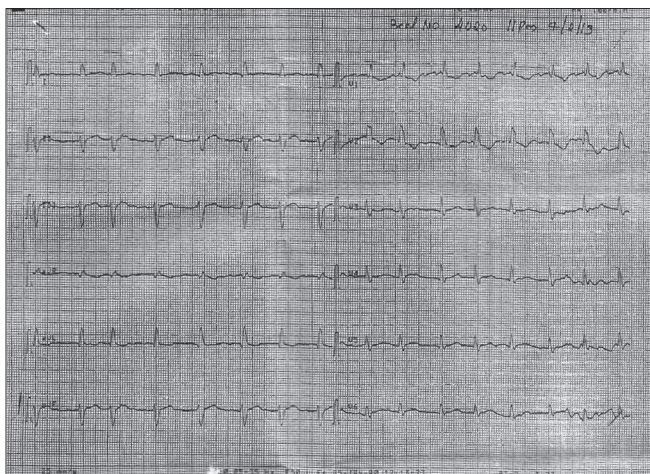
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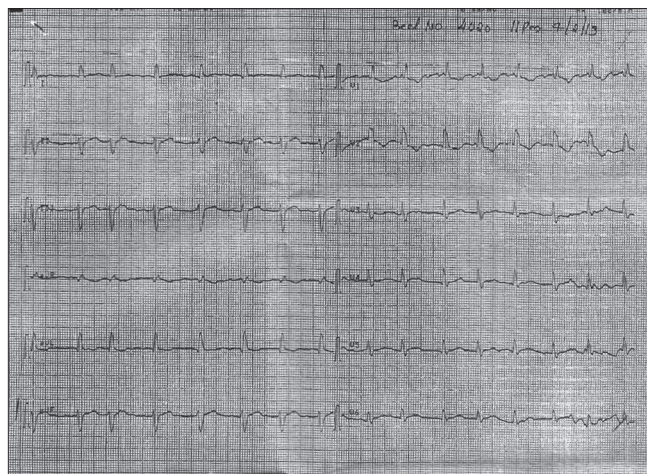
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**Figure 1:** AF with rapid ventricular rate



**Figure 2:** Normalization of AF with metoprolol

diastole, mild left ventricular hypertrophy was present with no regional wall motion abnormalities.

Patient was treated with levothyroxine which lead to resolution of symptoms and restoration of normal sinus rhythm. Repeat echocardiography showed no collapse of right atrium and right ventricle during diastole) and mild pericardial effusion (11 mm circumferential).

## DISCUSSION

The most common cardiovascular signs and symptoms of hypothyroidism include bradycardia, mild hypertension (diastolic), narrow pulse pressure, cold intolerance, pericardial effusion, cardiomyopathy and fatigue.<sup>[2]</sup> Hypothyroidism is often associated with electrocardiographic changes like bradycardia, right bundle branch block, flat or inverted T wave, QRS prolongation, QT prolongation, and infrequently ventricular arrhythmia, torsades de pointes. Treatment of hypothyroidism is well-documented to cause AF due to inadvertent overdosing of levothyroxine. Although atrial arrhythmias are common and ventricular ectopy is rare in patients with hyperthyroidism, it is rarely associated with hypothyroidism. AF is seen in 5-15% patients of hyperthyroidism and may be the presenting feature. Increased chronotropic activity due to increased thyroid hormones and increased sympathetic tone are the proposed underlying mechanisms. The physiological chronotropic response and normal tension of the heart muscle in diastolic phase depend on the action of triiodothyronine in the heart cells and its stimulating influence on  $\text{Na}^+\text{-K}^+$  ATPase and  $\text{Ca}^{2+}$  ATPase in endoplasmic reticulum. Normal contractility is also related to triiodothyronine stimulated transcription of the myosin heavy-chain alpha gene and inhibition of the heavy-chain beta gene. Moreover, triiodothyronine

acts on the cardiac muscle affects the number of beta adrenergic receptors and their sensitivity to catecholamines. Few case reports have demonstrated that hypothyroidism may cause a prolongation of the QT interval which predisposes the patient to ventricular irritability.<sup>[3]</sup> Rarely, torsades de pointes may result which is reversed with treatment. Patients with AF, rarely have been reported to have hypothyroidism.<sup>[1,4]</sup> The Canadian Registry of Atrial Fibrillation Investigators reported that 1.5% of 726 patients with AF had hypothyroidism over a period of 1.7 years.<sup>[5]</sup> Studies have also reported that up to 8% of patients with atrial fibrillation were hypothyroid.<sup>[6]</sup> Hypothyroidism is known to cause nodal defects, but the mechanism of AF in hypothyroidism is not known. It is also not clear whether its presence is causally related or is a mere manifestation of an underlying unrecognized structural heart disease. However in our case, reversal of AF with levothyroxine treatment negates underlying structural damage of the heart mans suggests causal relationship. Pericardial effusion is a well-recognized feature of hypothyroidism. Previous case reports have shown association of AF with hypothyroidism but pericardial effusion was absent in those cases. This is possibly the first reported case of AF and pericardial effusion in a case of hypothyroidism. Thyroxine therapy usually reverses all the cardiovascular changes associated with hypothyroidism. Young patients with no evidence of organic heart disease may be started on a full replacement dose of thyroxine. Older patients or those with known or suspected ischemic heart disease, should initially be given about 25% of the anticipated replacement dose, and the dose should be increased gradually at 6-8 week intervals.<sup>[7]</sup>

## CONCLUSION

Hypothyroidism may present with AF. However, AF is more commonly a presenting feature of hyperthyroidism.

Pericardial effusion is also common in hypothyroidism. Both are easily amenable to treatment.

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