

Case Report

An Unusual Cause of Completely Reversed Complete Heart Block “Uncontrolled Hypothyroidism”

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Received: January 21, 2018; Accepted: February 21, 2018; Published: March 26, 2018

Abstract

Background: Hypothyroidism is a common endocrine disorder with multiple system involvement and distinct clinical presentation. Its common presenting cardiovascular manifestations are sinus bradycardia and pericardial effusion usually associated with other symptoms of hypothyroidism. We report a case of a patient presenting with the uncommon manifestation of complete atrioventricular (AV) block which subsequently completely resolved with the thyroxin replacement alone suggesting hypothyroidism as the underlying aetiology.

Case Presentation: A 49yr old diagnosed patient with primary hypothyroidism who was defaulted thyroxine replacement therapy presented with an attack of fainting lasted for one hour with reduced exercise tolerance, episodes of lightheadedness associated mild leg swelling and constipation for one month. On examination she had peripheral stigmata of hypothyroidism with a regular heart rate of 56 beats per minute with normal blood pressure and rest of the systemic examination. Her electrocardiogram (ECG) showed evidence of complete heart block with thyroid function tests revealing a severe hypothyroidism without any other apparent cause for AV block. The complete heart block was subsequently completely resolved with the thyroxin replacement alone without need for pacing.

Conclusion: This case demonstrates an uncommon presentation of hypothyroidism with complete atrioventricular block. With prompt recognition and treatment, we were able to avoid invasive procedures and to minimize morbidity and mortality. The AV block was completely resolved with the correction of hypothyroidism.

Keywords: Hypothyroidism; Complete heart block

Introduction

Hypothyroidism can cause a variety of cardiovascular manifestations including sinus bradycardia, reduced contractility of heart, reduced stroke volume, hypertension due to increased total peripheral resistance and pericardial effusion [1-3]. The more common presenting cardiovascular manifestations are sinus bradycardia and pericardial effusion [1]. The affected patients usually have severe hypothyroid symptoms. Hypothyroidism rarely causes complete atrioventricular (AV) block [2]. In this paper, we report a patient with a main presenting feature of hypothyroidism had been complete heart block. The complete heart block was subsequently completely resolved with the thyroxin replacement alone suggesting hypothyroidism as the underlying aetiology.

Case Presentation

A 49yr old Sri Lankan female patient presented with an attack of acute onset fainting lasted for about one hour. She was found to be bradycardic with a heart rate of 40/min at that time and had a blood pressure of 90/60 mmHg. Her electrocardiogram (ECG) with a rhythm strip showed evidence of complete heart block (Figure 1) and she was transferred from local hospital where she first presented, to Teaching Hospital Karapitiya for further management including

acardio electrophysiologist's opinion. On further questioning we found out that she was diagnosed to have primary hypothyroidism in 2004 with a thyroid stimulating hormone (TSH) value of 60 mIU and was started on levothyroxine 75 ug daily. Apparently she had defaulted follow-up and was taking over the counter thyroxine on her own without satisfactory compliance. She was off treatment for last several months. She complained of reduced exercise tolerance and episodes of lightheadedness for past one month and also noted associated mild leg swelling and constipation. She did not experience orthopnea, paroxysmal nocturnal dyspnea, chest pain, syncope, urinary symptoms or unusual weight gain. She did not have any history of ischaemic heart disease; and irradiation or surgery to the neck or chest.

On detailed physical examination, she was conscious and rational. Her temperature was 36.7 °C and respiratory rate 18 breaths/minute. She had mild bilateral pitting ankle oedema. Examination of her extremities revealed dry cracked skin with no apparent yellowing of palms. She had coarse scalp hair, facial puffiness and thinning of the lateral third of the eyebrows. She had no macroglossia. The thyroid gland was normal on palpation. There was no lymphadenopathy. Careful examination of the cardiovascular system revealed bradycardia with a regular heart rate of 60/min blood pressure 110/70

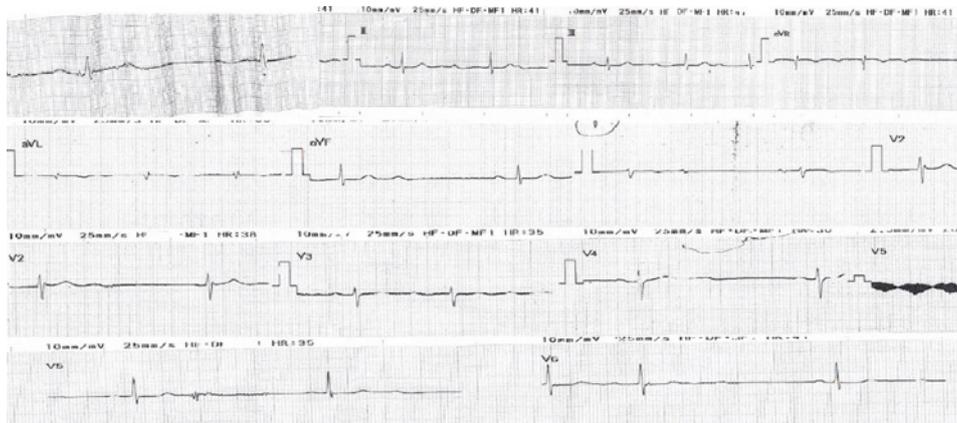


Figure 1: ECG with complete heart block before starting treatment.

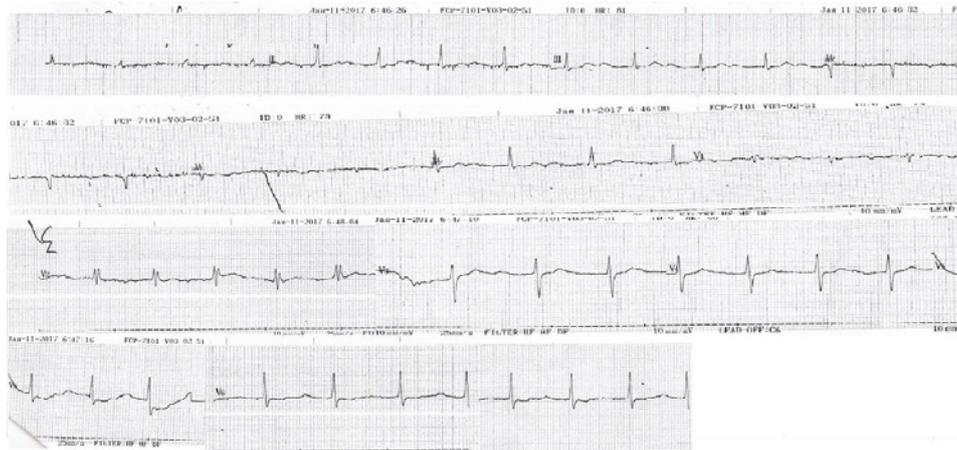


Figure 2: ECG with the first degree heart block while on treatment.

mmHg; normal S1 and S2; and no cardiac murmurs. Neurological examination revealed slow relaxation of both ankle reflexes.

Laboratory results were as follows: complete blood count with differential count showed hemoglobin of 11.3 g/dL with normal white cell and platelet counts. Blood urea nitrogen 19 mg/dL and serum creatinine 1.05 mg/dL. Electrolyte profile revealed Serum sodium 140 mmol/L, Potassium 3.9 mmol/L, Ionized calcium 1.2 mmol/L (1.1-1.35 mmol/L) and Serum Magnesium of 0.8 mmol/L (0.7-1 mmol/L). Fasting blood glucose was 98 mg/dL and liver transaminases were within normal limits.

Her thyroid function tests revealed thyroid stimulating hormone (TSH) > 100 mIU/L (0.47-4.7) with low free T3 0.9 pg/mL (2.0-4.0 pg/mL), and low free T4 < 0.30 ng/dL, (1.0 - 2.0 ng/dL). Her lipid profile revealed a total cholesterol level of 180 mg/dL, low-density lipoprotein cholesterol (LDL-C) of 114 mg/dL, high-density lipoprotein (HDL-C) of 31 mg/dL and triglyceride level of 154 mg/dL. Troponin-T level was within normal limits.

Her chest x-ray demonstrated normal lung parenchyma, thoracic cage, and mild cardiomegaly. Transthoracic echocardiogram showed no pericardial effusion or regional wall motion anomalies.

With these investigations she was seen by the cardio electro physiologist and since the patient was hemodynamically stable it was decided to correct hypothyroidism and to reassess with response to treatment and to consider the need of pacing. Therefore patient was started on levothyroxine 150 ug/day and was carefully monitored. On fifth day of hospital admission her ECG showed conversion of complete heart block to a First degree heart block (Figure 2).

Since the patient was completely asymptomatic with regard to cardio vascular status, she was discharged from hospital and careful follow up was arranged. On follow-up visit at 2 weeks after discharge she was symptom free without any dizziness, dyspnea or syncope and her pulse rate was found to be 80 per minute. Her ECG showed resolution of complete heart block.

Finally at 2 month review she was completely asymptomatic and clinically euthyroid. Her TSH was normal and her ECGs showed complete restoration of sinus rhythm (Figure 2,3).

Discussion

This case report describes a patient with hypothyroidism where the main presenting feature had been complete heart block with an episode of syncope, which got completely reversed with

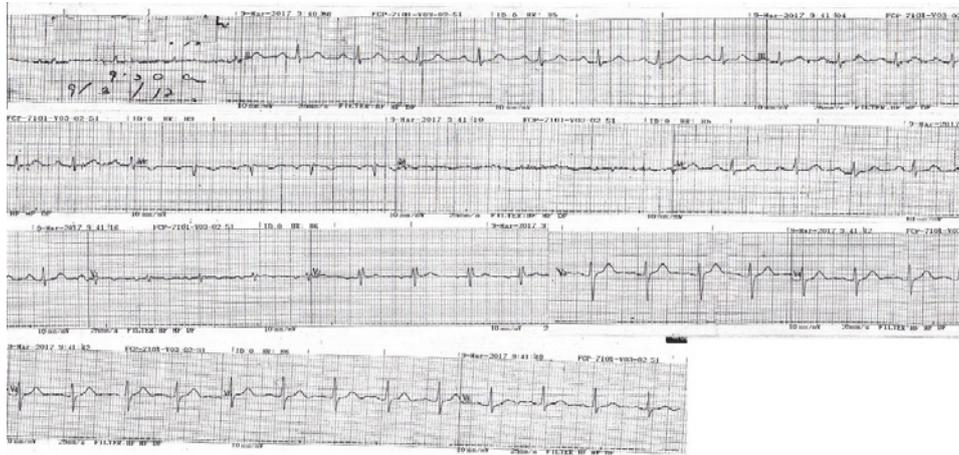


Figure 3: ECG showed restoration of sinus rhythm after 2 months of treatment.

thyroxin replacement therapy alone. Thyroid hormones also play an important role in electrical current generation and conduction in the myocardium. Changes in the cardiovascular system may give rise to the presenting manifestation in patients with both hypothyroidism and hyperthyroidism [1]. The estimated prevalence of hypothyroidism varies, depending on the population studied. In the United States, hypothyroidism affects approximately 0.3% of the general population [1]. Triiodothyronine increases systolic depolarization and decreases diastolic repolarization [2]. It shortens the duration of the action potential, the refractory period of the atrial myocardium and the atrioventricular nodal period. These changes are caused by increase in sodium pump density and sodium and potassium permeability. For the electrical current generation, T3 causes an increase in the L-type calcium channel at the sino-atrial node [2].

The cardiac electrical activity changes that can be observed in hypothyroid patients are sinus bradycardia, QT interval prolongation, T wave inversion and AV block [2,3]. Other cardiovascular effects include increased peripheral vasoconstriction; decreased cardiac contractility; and prolonged diastolic relaxation. Subsequently, cardiac output decreases due to a reduction in stroke volume and heart rate. Changes ensue as a result of the decrease in inotropy and chronotropy, such as bradycardia, narrowing of pulse pressure, prolongation of circulatory time and decrease in peripheral blood flow [3-5].

Among healthy elderly individuals, prevalence of hypothyroidism ranges from 0.55 to 1.5% [6,7]. Moreover, asymptomatic hypothyroidism rarely causes cardiovascular morbidity and mortality. Although the AV node is the first part of the AV conduction system to be affected by aging, the prevalence of second and third degree blocks in the general population is only approximately 1% [8,9].

In this case, the patient had been well until a few weeks before admission, and had never had any prior arrhythmic event causing syncope. Most case reports of hypothyroid patients with conduction abnormalities describe severe accompanying symptoms, such as myxedema coma [10,11]. In the absence of these symptoms, conduction abnormality is usually accompanied by other cardiac conditions, such as cardiomegaly or pericardial effusion [10,12].

In contrast, our patient admitted to hospital mainly because of the syncopal episode secondary to complete heart block.

Conclusion

This case demonstrates an uncommon presentation of hypothyroidism with complete atrioventricular block. With prompt recognition and treatment, we were able to avoid invasive procedures and minimize morbidity and the AV block was completely resolved with the correction of hypothyroidism.

Consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

References

- Brent G, Davies TF and Larsen PR. Hypothyroidism and thyroiditis. In: Kronenberg HM, Melmed S, Polonsky KS, Larsen PR, eds. *Williams Textbook of Endocrinology*. 11th ed. Philadelphia: Saunders Elsevier; 2008: 377-409.
- Freedberg AS, Papp JG, Williams EMV. The effect of altered thyroid state on atrial intracellular potentials. *J Physiol*. 1970; 207: 357-369.
- Kahaly GJ, Dillmann WH. Thyroid hormone action in the heart. *Endocr Rev*. 2005; 26: 704-728.
- Klein I, Ojamaa K. Thyroid hormone and the cardiovascular system. *N Engl J Med*. 2001; 344: 501-509.
- Kahaly GJ. Cardiovascular and atherogenic aspects of subclinical hypothyroidism. *Thyroid*. 2000; 10: 665-679.
- Ceresini G, Lauretani F, Maggio M, Ceda GP, Morganti S, Usberti E, et al. Thyroid function abnormalities and cognitive impairment in the elderly: Results of the Invecchiare in Chianti study. *J Am Geriatr Soc*. 2009; 57: 89-93.
- Cappola AR, Fried LP, Arnold AM, Danese MD, Kuller LH, Burke GL, et al. Thyroid status, cardiovascular risk, and mortality in older adults. *JAMA*. 2006; 295: 1033-1041.
- Schoenmakers N, de Graaff WE, Peters RH. Hypothyroidism as the cause of atrioventricular block in an elderly patient. *Neth Heart J*. 2008; 16: 57-59.
- Fragakis N, Katsaris G. Arrhythmias in the elderly: Modern management. *Hellenic J Cardiol*. 2006; 47: 84-92.
- Singh JB, Starobin OE, Guerrant RL, Manders EK. Reversible atrioventricular block in myxedema. *Chest*. 1973; 63: 582-585.

11. Schantz ET, Dubbs AW. Complete auriculoventricular block in myxedema with reversion to normal sinus rhythm on thyroid therapy. *Am Heart J.* 1951; 41: 613-619.
12. Nakayama Y, Ohno M, Yonemura S, Uozumi H, Kobayakawa N, Fukushima K, et al. A case of transient 2:1 atrioventricular block, resolved by thyroxine supplementation for subclinical hypothyroidism. *Pacing Clin Electrophysiol.* 2006; 29: 106-108.