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Case Report,

Refractory Complete Atrioventricular Block Secondary to Hypothyroidism Requiring Permanent Pacemaker

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Abstract:
Thyroid hormone regulates the body's metabolism, and its insufficiency can result in a wide variety of manifestations, including fatigue, lethargy, constipation, cold intolerance, and change in voice. Hypothyroidism also affects the cardiovascular system and can cause decreased cardiac output, increased systemic vascular resistance, pericardial effusion, bradycardia, and heart block. Hypothyroidism is a rare cause of complete atrioventricular block (AVB), which is generally thought to be reversible after thyroid replacement therapy. Here, we report a case of a 74-year-old female who presented to our hospital with lower extremity swelling and fatigue. She was diagnosed with hypothyroidism based on her thyroid function test. An electrocardiogram (ECG) revealed a complete AVB, and she was started on thyroid replacement therapy. During the second week of thyroid replacement therapy, she experienced episodes of bradycardia, and ECG still showed a complete AVB; despite normalization of her thyroid function, prompting placement of a pacemaker. The patient wasdischarged with endocrinology and electrophysiologist (EP) follow-up

Keywords: Hypothyroidism, Complete atrioventricular block, Permanent pacemaker

Introduction:

Hypothyroidism is a common condition and affects approximately 5% of the general population [1]. Patients with hypothyroidism are prone to cardiovascular manifestations, with sinus bradycardia and pericardial effusion being the most common, while complete atrioventricular (AVB) block is rare [2].

Complete AVB secondary to hypothyroidism is generally thought to be reversible with adequate thyroid hormone supplementation. Here, we report a case of persistent complete AVB secondary to hypothyroidism despite achieving euthyroid status.

Case Description:

A 74-year-old female with a past medical history of coronary artery disease status post a coronary stent, hyperthyroidism status post radioactive ablation, and hypertension presented to our hospital with poor appetite, decreased energy, and leg swelling. The patient denied shortness of breath, orthopnea, or paroxysmal nocturnal dyspnea. Vital signs were notable for blood pressure of 197/81 mmHg, heart rate of 56 beats per minute, and oxygen saturation of 98% on room air with a respiratory rate of 17 breaths/minute. Physical exam was positive for bradycardia and bilateral 2+ pedal edema;

otherwise, unremarkable. An electrocardiogram (ECG) demonstrated a third-degree heart block with complete atrioventricular (AV) dissociation (Figure*1*).

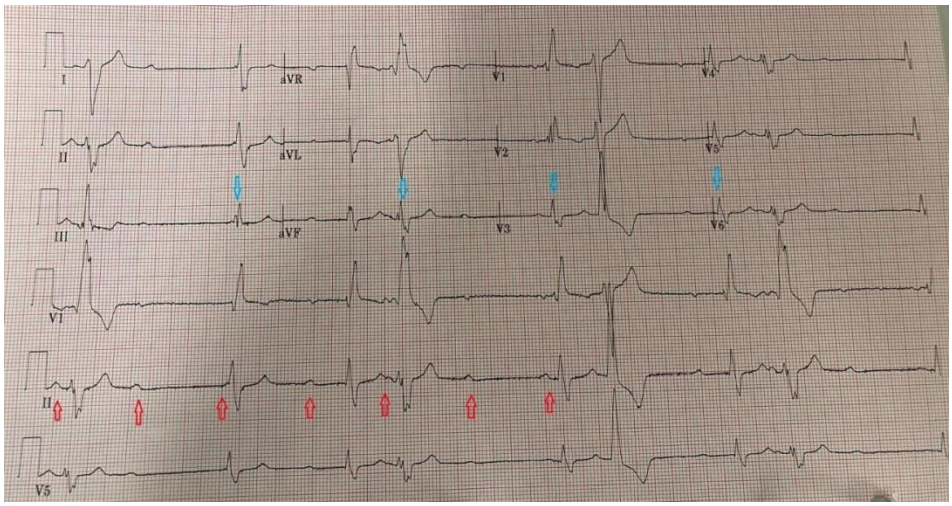

Figure 1. ECG on admission revealed complete atrioventricular dissociation, with constant PPinterval (red arrow) and RR interval.

The laboratory workup is summarized in Table 1.

Test	Results	Reference Range
White Blood Count	4.89	5.00 - 11.00 x10E3/uL
Hemoglobin	142.7	12.0 - 15.0 G/DL
Platelet	228	150 - 400 x10E3/uL
Sodium	135	135 - 145 mmol/L
Potassium	3.1	3.5 - 5.2 mmol/L
Creatinine	1.01	0.5 - 1.1 MG/DL
Blood Urea Nitrogen	11	6 - 23 MG/DL
Brain Natriuretic Peptide	3887	<101 pg/mL
Troponin	0.06	<0.031 mg/dl
Aspartate aminotransferase	58	1 - 35 U/L
Alkaline phosphatase	58	38 - 126 U/L
Bilirubin Total	0.9	0.1 - 1.2 mg/dL
Free Thyroxine(T4)	0.2	0.8-1.7 ng/dl
Thyroid stimulating hormone(TSH)	0.37	0.40 - 4.20 uIU/mL

Table 1: Laboratory workup on admission

Chest X-ray showed cardiomegaly with mild basilar interstitial edema (Figure *2*). A transthoracic echocardiogram revealed a severely increased left ventricular wall thickness, normal systolic function with an ejection fraction of 60 to 65%, and a moderately dilated left atrium, otherwise unremarkable.

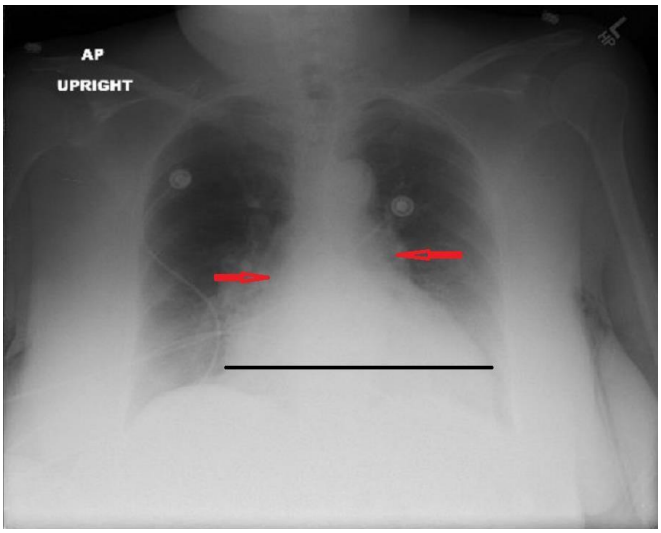

Figure 2. Chest X-ray on admission revealed cardiomegaly (horizontal black line) andpulmonary congestion (red arrow)

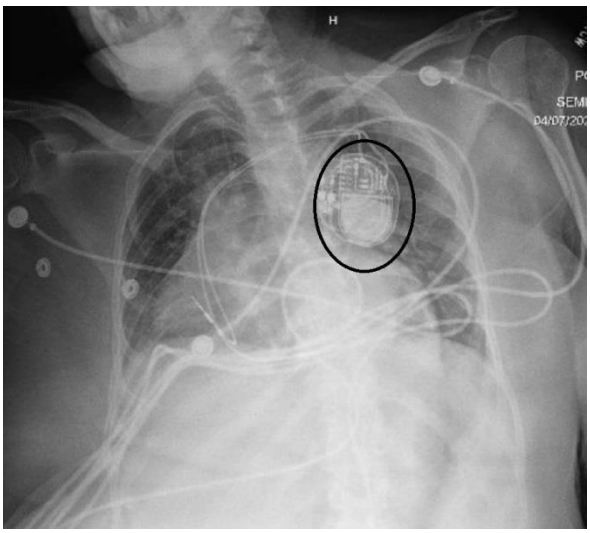

Figure 3. Chest X-ray after dual-chamber pacemaker placement

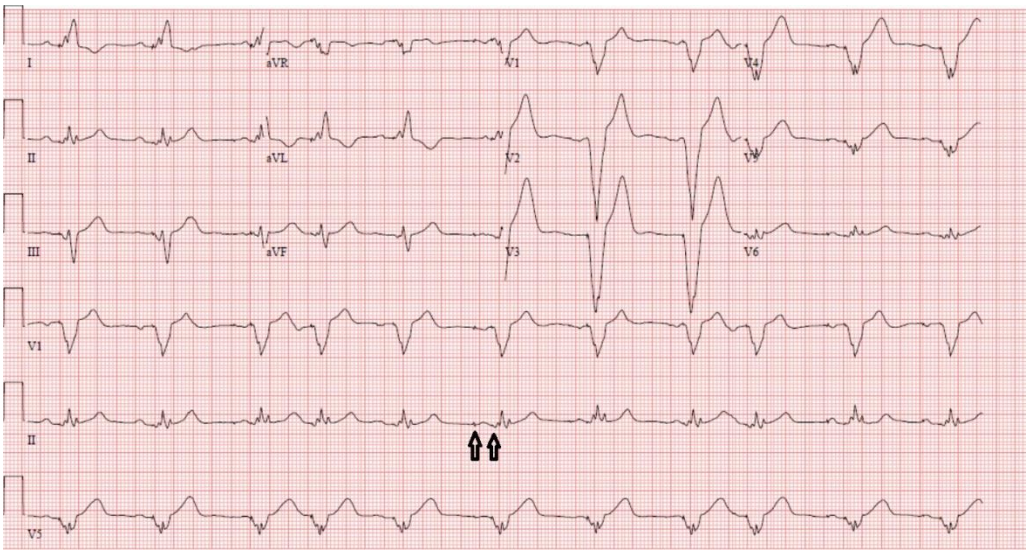

Figure 4. Dual-chamber pacing. Note that the pacing detector is turned on (black arrows)

Discussion:

Hypothyroidism is a common endocrine pathological condition of the thyroid gland defined by an increase in thyroid-stimulating hormone (TSH) concentrations above the reference range and free thyroxine (T4) concentrations below the reference range, making the diagnosis predominantly paraclinical. Hypothyroidism can affect all the major/organ systems like the neurological, hematological, and gastrointestinal, including the cardiovascular system, which is the most studied [1]. Clinical presentation varies, and certain age groups like the elderly can have nonspecific symptoms. Some common presenting symptoms are; hoarseness of voice, bradycardia, muscle weakness, lethargy, cold intolerance, depression, weight gain, constipation, thinning of hair, and dry skin [1-2].

Hypothyroidism causes a wide array of cardiovascular manifestations since the thyroid hormone regulates the heart's electrical activity by a couple of mechanisms, mainly by playing a role in the electrical current generation and myocardial conduction. The most common manifestations are; low cardiac output, reduced cardiac contractility, decreased heart rate, diastolic hypertension,

increased peripheral vascular resistance, and, less commonly, pericardial effusion and complete AVB [3-5].

Multiple causes can impair the cardiac conduction system resulting in AVB. They can be dividedinto idiopathic (50%), cardiac such as ischemic heart disease (40%) [6], infectious such as Lymecarditis and endocarditis, toxic such as drug side effects, and less commonly metabolic, which involve hypothyroidism, hyperthyroidism, acid-base disorders, electrolyte abnormalities, and adrenal disease.

AVB is classified as; first degree, second degree, and third (complete) degree. Complete heart block has a prevalence of 0.04 % in the general population [7]. It is defined by the lack of conduction of atrial impulses to the ventricle. As previously mentioned, hypothyroidism-induced complete AVB is an uncommon etiology, and there is a paucity of data and reports documenting this phenomenon. Generally, conduction abnormalities like AVB in hypothyroidism often occur in patients with severe accompanying features like myxedema coma [2,8]; however, our case hadno profound signs of hypothyroidism.

Complete AVB manifests mostly with dyspnea and chest pain, presyncope and syncope beingthe most common symptoms. It is diagnosed through ECG by recognizing severe bradycardia with independent atrial and ventricular activity, also referred to as AV dissociation [2,5].

Treatment generally involves the identification of reversal causes such as Lyme carditis in endemic areas, digoxin toxicity though uncommon, an overdose of antiarrhythmic drugs, beta- blockers, calcium channel blockers, electrolyte abnormalities, particularly hyper and hypokalemia, and hypothyroidism with some patients ultimately requiring permanent pacing if complete AVB persists despite treating the underlying cause [5].

In this case, we ruled out the other reversible causes of complete AVB, and this left us with hypothyroidism as the most likely etiology. The reversal of complete AVB back to sinus rhythm in the setting of hypothyroidism is generally seen with adequate thyroid hormone supplementation back to a euthyroid state [9]. This is contrary to our patient, who had a complete refractory AVB with worsening bradycardia despite adequate thyroid hormone replacement for several days with normalization of her free T4 and TSH levels, prompting the implantation of a dual-chamber pacemaker (Figure *3*,4).

The management is still quite complex and should be tailored to individual patients as to the need for pacemaker vs. thyroid hormone replacement therapy only. This calls for our attention to the need for more randomized control trials regarding the need for permanent pacemakers by default in patients with complete AVB secondary to hypothyroidism so as not to delay patient care.

Endocrinology was consulted because of the new onset of post-ablative hypothyroidism. The patient was started on oral levothyroxine 50 mcg daily. Magnetic resonance imaging (MRI) of the brain was obtained to rule out pituitary dysfunction and was unremarkable. The patient received thyroid replacement therapy for 12 days while inpatient, but she had frequent episodes of significant bradycardia and multiple episodes of nonsustained ventricular tachycardia on telemetry. At that point, an electrophysiologist (EP) was consulted, and a dual-chamber pacemaker was placed. This patient was discharged to the rehab on levothyroxine with endocrinology and EP follow-up.

Conclusion:

Rarely hypothyroidism can present as a complete atrioventricular block; therefore it is imperative to rule out hypothyroidism in a patient presenting with a complete heart block. The heart block secondary to hypothyroidism may not be reversible after achieving euthyroid statusand may require a permanent pacemaker. We need large randomized control trials to better understand the pathophysiology of heart block in patients with hypothyroidism and the need for permanent pacemaker placement.

Conflicts of Interest:

None **Funding:** None

Acknowledgment:

None

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