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Severe Hypothyroidism Causing Reversible Pacing Failure: A Case Report and Literature Review

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Abstract

Pacemakers are implantable electronic devices used in patients with conduction disorders to deliver electrical stimuli, configured with predetermined settings. These devices facilitate effective myocyte contraction and maintain the heart's electrical excitability. Pacemaker malfunctions can arise from sensing or pacing issues and manifest in various clinical presentations. Although malfunctions often involve anomalies in the pulse generator or leads, underlying conditions and diseases affecting the conduction system may be principal causes; thus, their management can lead to rectification of the malfunction. We present a case of a 66-year-old woman with asymptomatic profound hypothyroidism revealed through pacemaker malfunction, with restoration of normal pacemaker function following thyroid hormone replacement.

Categories: Cardiology
Keywords: autoimmune thyroiditis, hypothyroidism, pacemaker malfunction, thyroid hormone replacement, ventricular noncapture

Introduction

Thyroid hormones are essential for human life, exerting significant effects on various metabolic processes, notably within the cardiovascular system [1,2]. Hypothyroidism induces a low metabolic state, leading to bradyarrhythmias, reduced cardiac contractility (inotropism) and excitability (bathmotropy), decreased cardiac output, and increased peripheral vascular resistance [3]. Although severe bradycardia and high-degree atrioventricular blocks secondary to hypothyroidism are well-documented, reports of pacemaker malfunction due to hypothyroidism remain scarce [4-6]. We present a case of severe hypothyroidism manifesting as pacemaker failure, which was successfully reversed with thyroxine replacement therapy, obviating the need for pacemaker reimplantation.

Case Presentation

A 66-year-old female patient presented for a routine follow-up of her pacemaker. Her medical history was notable for atrial fibrillation and the implantation of a single-chamber pacemaker several years before the current presentation for a high-grade atrioventricular block, which was deemed degenerative as no underlying cause was identified. The patient denied experiencing chest pain, palpitations, shortness of breath, or headaches at the time of the consultation. Physical examination revealed an irregular pulse rate of 50 beats per minute, respiratory rate of 16 breaths per minute, blood pressure of 135/70 mmHg, and an oxygen saturation of 99% on room air.

Initial ECG revealed an irregular rhythm and ventricular noncapture (Figure *1*). Pacemaker interrogation demonstrated a markedly elevated ventricular capture threshold of 4.7 V, exceeding the programmed output of 2.5 V. Ventricular lead impedance was 850 Ω, and the ventricular sensing amplitude was 5 mV. The patient was not on any antiarrhythmic medications, such as beta-blockers, calcium channel blockers, amiodarone, or digoxin, which could account for the ECG abnormalities. The echocardiography showed proper lead placement of the pacemaker, a left ventricular ejection fraction (LVEF) of 70%, normal diastolic function, and preserved right ventricular function (Figure *2*).

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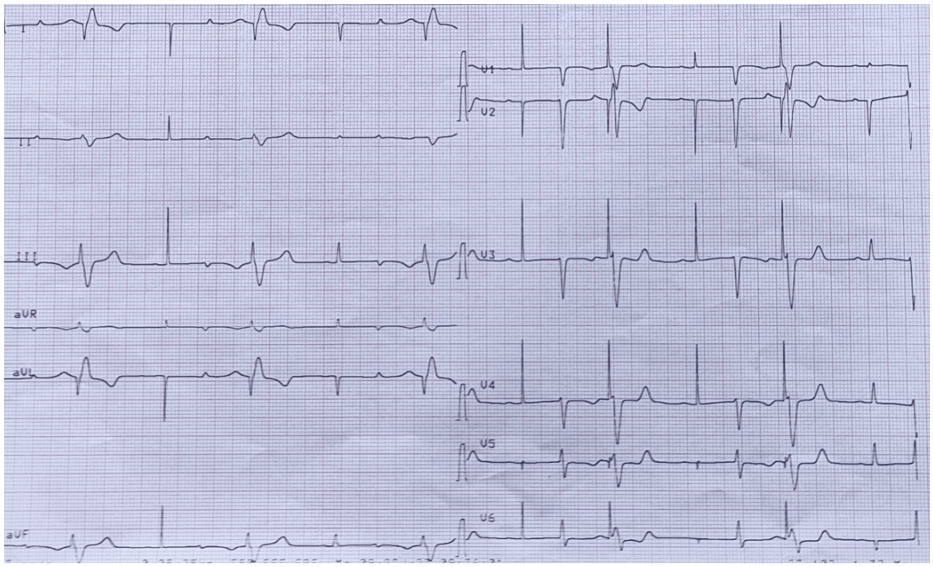


FIGURE 1: Initial ECG revealing an irregular rhythm and ventricular noncapture

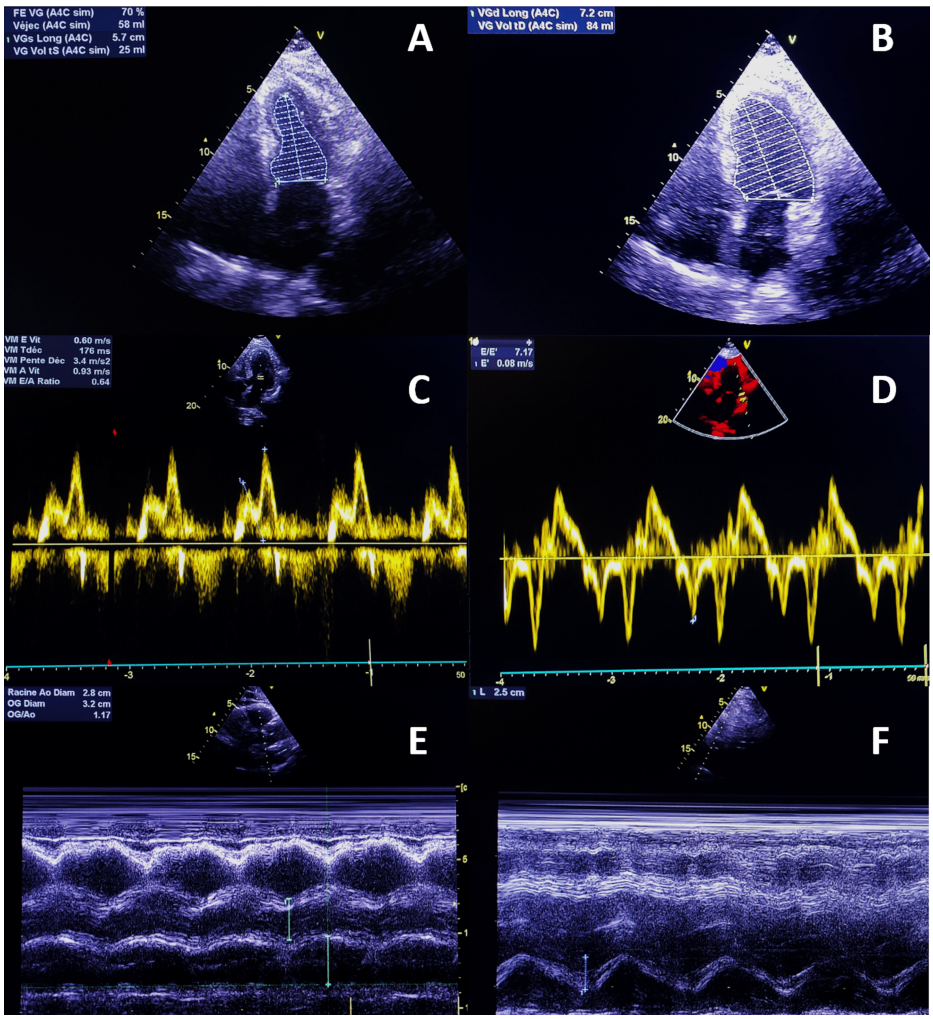


FIGURE 2: Transthoracic echocardiographic assessment on Day 1 of admission

(A) and (B) Apical four-chamber views, in systole and diastole, respectively, demonstrating preserved left ventricular systolic function with a left ventricular ejection fraction of 70%. (C) Pulsed-wave Doppler assessment of transmitral flow demonstrating a normal grade I diastolic filling pattern. (D) Tissue Doppler imaging demonstrating a normal early diastolic mitral annular velocity E′. (E) Parasternal long-axis view demonstrating normal cardiac chamber dimensions. (F) M-mode assessment demonstrating preserved right ventricular systolic function

Initial laboratory tests revealed normal pH and an unremarkable basic metabolic panel; the complete blood count was within normal limits for age and gender. Thyroid-stimulating hormone (TSH) was significantly elevated at 190.2 mIU/L (reference range: 0.5-5 mIU/L), while free thyroxine (fT4) was decreased at 0.42 ng/dL (reference range: 0.7-1.7 ng/dL). Given this abnormal thyroid function test, additional thyroid studies were requested, which revealed significantly elevated anti-thyroid peroxidase (anti-TPO) antibodies at 241 IU/ml (less than 9.0 IU/ml) and anti-thyroglobulin (anti-TG) antibodies at 560 IU/ml (less than 116 IU/ml) (Table *1*). The thyroid ultrasound revealed a diffuse enlargement of the thyroid gland, without any identifiable nodules or other abnormalities (Figure *3*).

Variable	Result	Reference range
Sodium (mmol/L)	135	135-145
Potassium (mmol/L)	5.3	3.5-5.3
Chloride (mmol/L)	111	96-106
Calcium (mg/dL)	9.3	8.5-10.5
Blood glucose (mg/dL)	90	70-99
Serum bicarbonate (mmol/L)	23	22-29
Urea (g/L)	0.41	0.15-0.45
Creatinine (mg/L)	14	7-13.5
ASAT (U/L)	19	<35
ALAT (U/L)	6	<55
GGT (U/L)	35	5-40
Lactates (mmol/L)	1.2	0.5-2.2
pH	7.4	7.35-7.45
pO2 (mmHg)	82	80-100
pCO2 (mmHg)	37	35-45
CRP (mg/L)	2.7	<6
TSH (mIU/L)	190.2	0.4-4
fT4 (ng/dL)	0.42	0.8-1.8
Anti-TPO (IU/mL)	241	9-35
Anti-TG (IU/mL)	560	<115
White blood cells (/µL)	4980	4000-10000
Neutrophils (/µL)	1240	1500-7000
Hemoglobin (g/dL)	11.8	11.6-15
Platelets (/µL)	270000	250000-450000

TABLE 1: Biological parameters on admission

ASAT: aspartate aminotransferase; ALAT: alanine aminotransferase; GGT: gamma-glutamyl transferase; TSH: thyroid-stimulating hormone; anti-TPO: anti-thyroid peroxidase; anti-TG: anti-thyroglobulin; CRP: C-reactive protein; fT4: free thyroxine

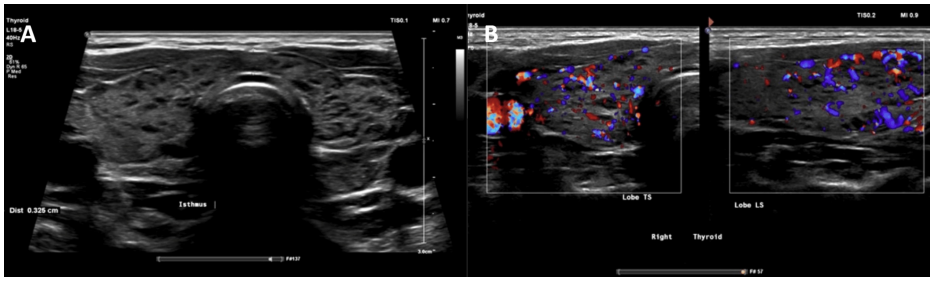


FIGURE 3: Thyroid ultrasonography demonstrating diffuse enlargement of the thyroid gland (A) with increased vascularity on color Doppler imaging (B)

Given the clinical presentation and supplementary examination findings, a diagnosis of autoimmune thyroiditis was established. Levothyroxine replacement therapy was initiated at a dose of 25 µg/day. During the first week of treatment, the patient's heart rate improved, and pacemaker interrogation on Day 8 demonstrated a decrease in the ventricular capture threshold from 4.7 V to 1.8 V, while the programmed output remained at 2.5 V. Lead impedance and ventricular sensing remained stable at 840 Ω and 5 mV, respectively. Follow-up ECGs confirmed restoration of normal pacemaker function (Figure *4*), and pacemaker replacement was therefore not required. By Day 13, thyroid function tests showed an initial trend toward normalization. The patient was subsequently referred to the endocrinology department for continued management of her thyroid disease and follow-up of thyroid function.

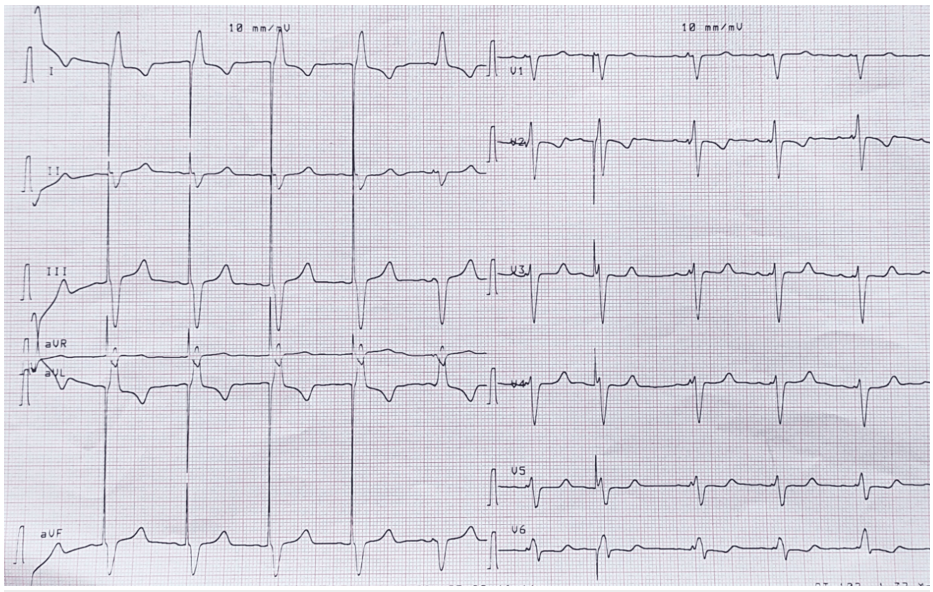


FIGURE 4: Restoration of normal pacemaker function following thyroid hormone replacement

Discussion

This case highlights a rare but important manifestation of pacemaker failure secondary to profound hypothyroidism. Pacemaker malfunctions are generally attributed to device-related issues such as lead displacement, battery depletion, or improper sensing/pacing algorithms. However, underlying systemic conditions such as thyroid dysfunction can also significantly impact pacemaker performance, as illustrated in this patient with severe hypothyroidism [7,8].

Thyroid hormones play a crucial role in maintaining cardiovascular homeostasis. Hypothyroidism is well-known for its deleterious effects on cardiac function, inducing bradycardia, decreased myocardial contractility, and impaired electrical conduction. These physiological alterations stem from a reduced metabolic state, which diminishes the heart’s excitability and conductivity [2,9]. As seen in this case, the marked hypothyroidism led to a ventricular pacing failure, despite the absence of structural issues with the pacemaker itself. The patient's irregular heart rate and failure to capture were reversible once thyroid

hormone levels were restored, obviating the need for surgical pacemaker revision or replacement.

The association between hypothyroidism and abnormal cardiac pacing parameters has been described in isolated reports. Basu and Chatterjee reported an unusually high pacemaker threshold in a patient with severe myxedema that progressively decreased following thyroid hormone replacement [10]. Similarly, Schlesinger et al. described exit block developing in a patient with an implanted pacemaker during severe myxedema, with normalization of the pacing threshold after thyroid hormone therapy [11]. Other reports have subsequently described elevated stimulation thresholds and reversible atrial lead dysfunction in the setting of hypothyroidism [12,13].

In our case, the marked elevation of TSH and low fT4 levels confirmed the presence of severe hypothyroidism, with positive anti-TPO and anti-TG antibodies supporting a diagnosis of autoimmune thyroiditis. While autoimmune thyroiditis is a well-established cause of hypothyroidism, its role as a trigger for pacemaker malfunction is exceedingly rare. A review of the existing literature revealed a case reported by Waseem et al., describing high-grade AV block and symptomatic bradycardia necessitating pacemaker implantation; however, the correction of hypothyroidism in that instance resulted in complete resolution of the AV block, thereby obviating the need for pacemaker placement [14].

Our case differs from reports in which hypothyroidism was identified before or during initial pacing because the patient had a previously implanted and functioning pacemaker and subsequently presented with ventricular noncapture. The thyroid disorder was clinically silent, and severe biochemical hypothyroidism was identified only during the evaluation of pacemaker dysfunction. Importantly, pacemaker function improved after thyroid hormone replacement, allowing device revision to be avoided.

Conclusions

Pacemaker malfunction is not always attributable to a primary device abnormality. Severe hypothyroidism represents a rare but potentially reversible systemic cause of pacing dysfunction and should be considered when common mechanical and device-related causes have been excluded. Early recognition and treatment of thyroid dysfunction may restore pacemaker function and avoid unnecessary device revision or replacement.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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