

Severe primary hypothyroidism presenting as polymorphic ventricular tachycardia

Hipotiroidismo primario grave que se presenta como taquicardia ventricular polimórfica

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Abstract

Introduction: Hypothyroidism may have various cardiovascular manifestations due to morphological, functional and electrical alterations in the heart. The usual electrocardiographic findings being sinus bradycardia, low voltage complexes, and slowed intraventricular conduction. Hypothyroidism manifesting as polymorphic ventricular tachycardia has only been reported in a few case reports. **Clinical case.** A 60-year-old lady presented to us in the emergency department in an unresponsive and unconscious state and electrocardiogram showed a polymorphic ventricular tachycardia. After initial resuscitation with direct current cardioversion and supportive care, she found to have severe hypothyroidism and responded well to thyroid replacement therapy. **Conclusion.** Polymorphic ventricular tachycardia is a life threatening emergency that can have various etiologies. Polymorphic ventricular tachycardia secondary to primary hypothyroidism is a rare presentation but it is treatable and reversible with thyroid replacement therapy. In patients presenting with QT interval prolongation and ventricular tachycardia, hypothyroidism should be one of the differential diagnosis.

Keywords: Hypothyroidism. Polymorphic ventricular tachycardia. QT interval. Torsades de pointes. Cardiogenic shock.

Resumen

Introducción: El hipotiroidismo puede presentar diferentes manifestaciones cardiovasculares dadas por alteraciones morfológicas, funcionales y eléctricas en el corazón, siendo los hallazgos electrocardiográficos usuales son la bradicardia sinusal, los complejos de bajo voltaje y la conducción intraventricular lenta. El hipotiroidismo manifestado como taquicardia ventricular polimórfica solo se ha descrito en unos pocos reportes de caso. **Caso clínico:** Se trata de una mujer de 60 años que acudió que acurdió al servicio de urgencias en un estado inconsciente y sin respuesta a estímulos, y el electrocardiograma reveló taquicardia ventricular polimórfica. Luego de la reanimación inicial con cardioversión con corriente directa y tratamiento sintomático se le encontró un hipotiroidismo grave, el cual se trató con terapia de reemplazo con hormona tiroidea. y se obtuvo una buena respuesta **Conclusión.** La taquicardia ventricular polimórfica es una emergencia vital que puede tener varias etiologías. La taquicardia ventricular polimórfica secundaria a un hipotiroidismo primario es una presentación poco común, pero es tratable y reversible con la terapia de reemplazo con hormona tiroidea. En los pacientes que presentan una prolongación del intervalo QT y taquicardia ventricular, es pertinente incluir el hipotiroidismo en el diagnóstico diferencial.

Palabras clave: Hipotiroidismo. Taquicardia ventricular polimórfica. Intervalo QT. Torsades de pointes. Choque cardiogénico.

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Introduction

The effects of thyroid disorders on cardiovascular systems have been known for many decades. Both hypothyroidism and hyperthyroidism may cause exacerbation of various cardiovascular diseases and symptoms like atherosclerotic heart disease, dyslipidemia, arrhythmias, and heart failure, etc. Hypothyroidism may have various functional, structural, and electrical effects on the heart¹. Hypothyroidism can have a myriad of electrocardiographic manifestations that may include sinus bradycardia, low voltage complexes, prolongation of PR and QT interval, T wave inversion, and nonspecific ST-segment changes². Hypothyroidism is a known reversible cause of prolonged QT interval, which is due to prolonged ventricular activation and may lead to torsades de pointes³. Hypothyroidism and its incidence increases in age and may particularly affect patients who are not on treatment or who are elderly⁴. We report one such case of a 60-year-old lady who presented to the emergency in an unconscious state having torsades de pointes and upon further investigations found to be diagnosed with severe hypothyroidism and subsequently improved with thyroid replacement therapy.

Case report

A 60-year-old lady presented to us in the emergency department in an unresponsive and unconscious state for the last one hour. On presentation, she had cold extremities with an irregular pulse with a heart rate of 40/min, and blood pressure was unrecordable at the time of presentation. She had a history of hypertension for the last 5 years for which she was on tablet amlodipine 5 mg once daily. The patient also had a history of generalised fatigue and weight gain for the last 6 months. She had no history of diabetes, chronic illness, any malignancy, smoking, and drug abuse. Differential diagnosis considered by the emergency team were acute coronary syndrome, myocarditis, acute pulmonary embolism, and acute metabolic dysfunction. On presentation, her electrocardiogram (ECG) revealed polymorphic ventricular tachycardia (Fig. 1). The patient was given prompt resuscitation and tachycardia was reverted with direct current (DC) cardioversion with 200 Joules. After initial resuscitation, she was transferred to the intensive care unit (ICU), where she again had two episodes of ventricular tachycardia which were reverted with DC cardioversion. After resuscitation, her ECG (Fig. 2A) revealed sinus rhythm with T wave

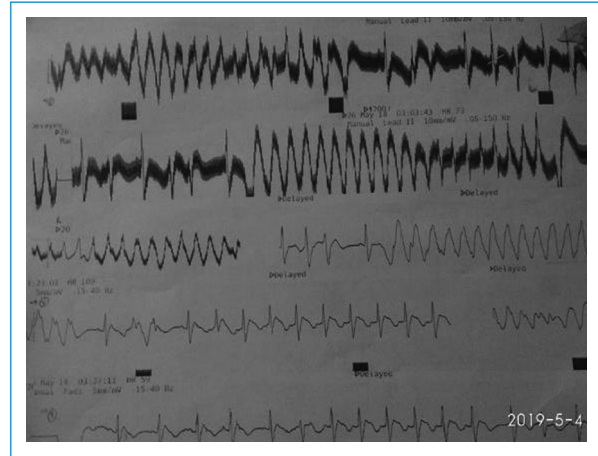


Figure 1. On presentation, Electrocardiogram (ECG) showing polymorphic ventricular tachycardia (Torsades de pointes).

inversion in anterior leads, prolongation of QT interval with corrected QT interval (QTc) of 625 milliseconds (ms). Her initial laboratory workup was unremarkable except for increased serum troponin levels of 1.86 ng/ml and brain natriuretic peptides (BNP) levels of 1784 pg/ml. Her echocardiography revealed a dilated left ventricular cavity with global hypokinesia, mild mitral regurgitation, and severe left ventricular dysfunction (ejection fraction of 20%). She was subsequently planned for a diagnostic coronary angiogram to rule out significant coronary artery disease, however, it revealed normal left and right epicardial coronary arteries (Fig. 3A-3B). Thyroid function tests revealed a high thyroid-stimulating hormone (TSH) level of 51.2 mill units per litre (mU/l) with decreased free levothyroxine levels (fT4) of 0.28 nanogram/decilitre (ng/dl) [normal range 0.89-1.76 ng/dl] and decreased free tri-iodothyronine (fT3) levels of 1.5 picograms/millilitre (pg/ml) [normal range, 2.00-4.40 pg/ml]. Serum electrolytes like potassium, calcium, and magnesium were within normal limits.

After initial resuscitation and successful reversion of ventricular tachycardia, she received magnesium sulphate and supportive care. After confirmation of hypothyroidism, she was started on thyroid replacement therapy with levothyroxine 100 microgram/day, the QTc interval got normalised to 437 ms on the fifth day of hospitalisation. The patient was discharged in a stable condition on thyroid replacement therapy. At 3 months follow up, she remained asymptomatic and her ECG showed sinus rhythm (Fig. 2B), and echocardiogram revealed normal left ventricular dimensions and ejection fraction.

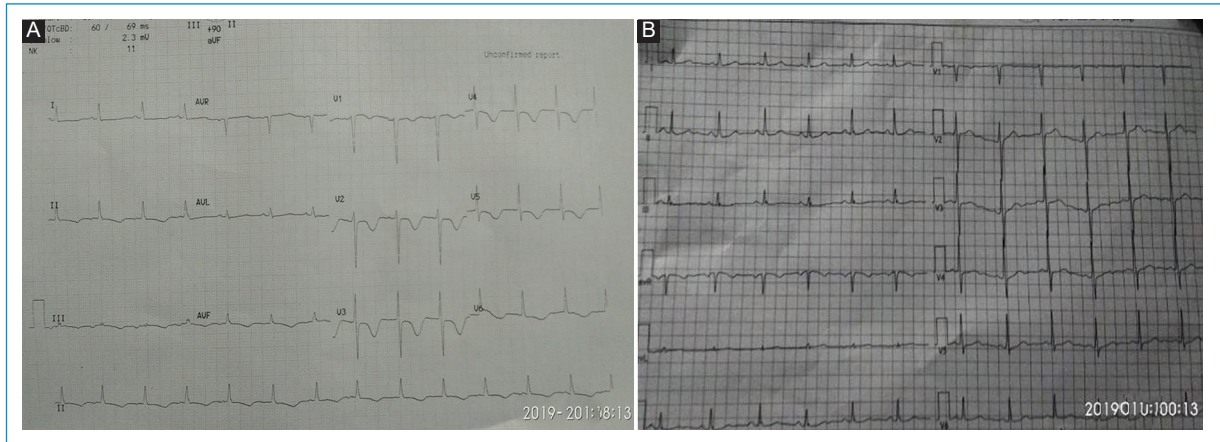


Figure 2. A: after initial resuscitation, Electrocardiogram (ECG) showing prolongation of QT interval. **B:** at 3 months follow up, ECG showing sinus rhythm and normal QT interval.

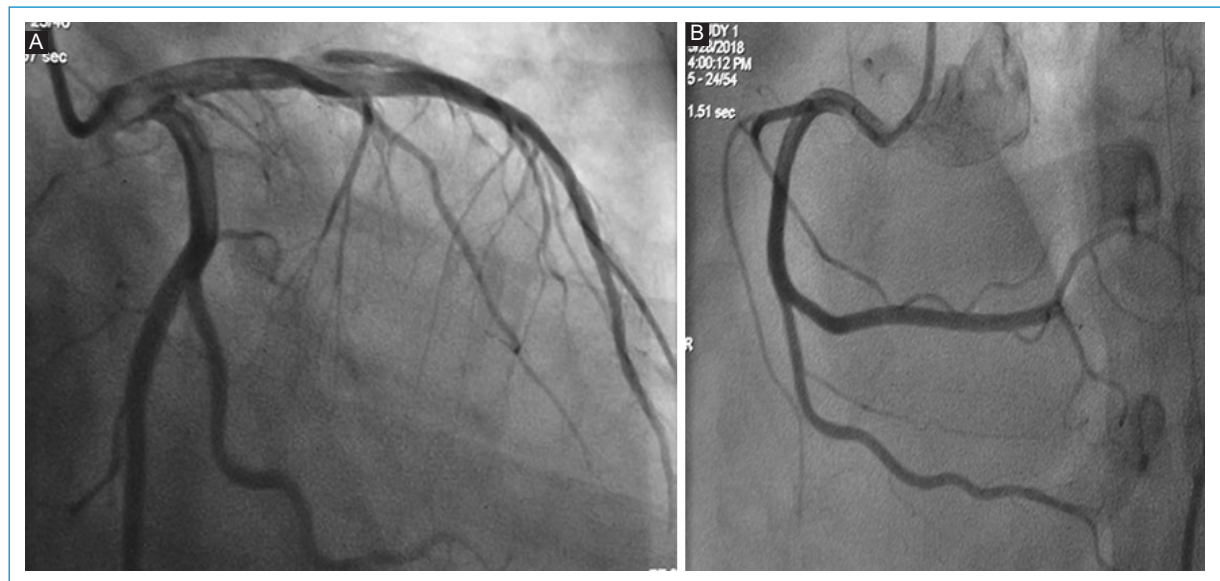


Figure 3. Coronary angiogram showing normal circulation. **A:** in the left coronary artery. **B:** right coronary artery.

Discussion

The thyroid hormone has both inotropic and chronic effects on the heart. In hypothyroidism, as a result of decreased inotropic and chronotropic effects, a patient may have reduced heart rate and contractility which when prolonged may further lead to a reduction in ejection fraction, dilated cardiomyopathy, and heart failure³. Thyroid hormone affects the cardiac electrophysiology and rhythm by its action on cardiac membrane ion channels, membrane transporters, and ion exchangers that regulate the electrical activity of the heart⁵. In

hypothyroidism, a patient may present with an abnormal rhythm like atrial fibrillation (AF), sinus bradycardia, heart block, supraventricular and ventricular tachyarrhythmias, and very rarely polymorphic ventricular tachycardia. There are only a few case reports published with primary hypothyroidism manifesting as torsades de pointes, and successfully managed by thyroid replacement therapy^{4,6-7}.

Our patient presented in emergency with shock and ECG showed torsades de pointes, she responded initially to cardioversion and supportive care. The usual predisposing factors for torsades de pointes like hypokalemia,

hypomagnesemia, and hypocalcemia were checked, which were within normal limits. On further evaluation, we found that the cause of the torsades de pointes was severe primary hypothyroidism that was undetected for a long time. She responded well to thyroxine replacement therapy and QT interval normalised in 5 days of thyroxine therapy and ECG was normal at 3 months follow up (Fig. 2B). We are reporting this case due to a very rare presentation of primary hypothyroidism as a polymorphic ventricular tachycardia that was managed successfully by thyroxine replacement therapy. (Fig. 3)

Conclusion

Torsades de pointes is a rare but life-threatening complication of primary hypothyroidism. In patients presenting with QT interval prolongation and ventricular tachycardia, primary hypothyroidism should be kept as one of the differential diagnosis.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical responsibilities

Protection of humans and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality of data. The authors declare that they have followed their center's protocols on the publication of patient data.

Right to privacy and informed consent. The authors have obtained the informed consent of the patients and/or subjects referred to in the article. This document is in the possession of the corresponding author.

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