Acquired Long QT Syndrome Secondary to Hypoparathyroidism

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ABSTRACT. We report a case of a patient who presented with history of recurrent short episodes of palpitations associated with lightheadedness and syncope. He was found to have prolonged QT interval on his 12-lead electrocardiogram (corrected: 582 ms). His serum electrolytes showed normal potassium and magnesium levels, severe hypocalcaemia, and low parathyroid hormone and vitamin D levels. After correction of the calcium level, the QT interval normalized and there were no further episodes of syncope, lightheadedness, or palpitations.

KEYWORDS. Hypocalcaemia, hypoparathyroidism, long QT syndrome.

Introduction

Long QT syndrome (LQTS) is a disorder characterized by a prolongation of the QT interval on electrocardiograms (ECGs) and a propensity to ventricular tachyarrhythmias, which may lead to syncope, cardiac arrest, or sudden death.1,2 The LQTS may be either congenital or acquired.3 Acquired LQTS usually results from drug therapy, electrolyte disturbances such as hypokalemia, hypomagnesaemia, and hypocalcaemia as well as bradycardia, which can increase the risk of drug-induced LQTS. Hypocalcaemia is known to prolong the cardiac repolarization by prolonging the phase 2 action potential (AP).4-6 This can expose the AP to repetitive triggered activity or achieving a sustained functional re-entry, which may propagate and lead to polymorphic ventricular tachycardia.7

Case report

A 55-year-old man, with no family history of cardiac disease or sudden cardiac death, presented with a history of recurrent episodes of syncope accompanied by inability to speak, and abnormal movement of his mouth. These episodes lasted for a few minutes and were preceded by palpitations and chest pain. The patient’s past medical history was unremarkable except for well-controlled hypertension with amlodipine. On presentation to the hospital, the patient’s ECG showed prolonged QT/QTc interval of 582/582 ms (Figure 1). The serum electrolytes were sent and revealed normal potassium and magnesium levels. His serum calcium level was low 5.2 mg/dl (8.5–10.2 mg/dl), and his phosphorus level was high 7.8 mg/dl (2.5–4.5 mg/dl). Further work-up showed low vitamin D and low parathyroid hormone levels. The patient was treated with intravenous calcium supplementation and vitamin D. He was observed closely in the intensive care unit with serial ECGs and calcium readings. His symptoms as well as the QT interval returned to normal after correction of his calcium level (Figure 2).

Discussion

Acquired LQTS is a disorder of cardiac repolarization most often preceded by drug administration or electrolyte disturbances. Other possible causes include heart disease, ischemia, marked bradycardia, stroke, and brain injury. The cornerstone of the management of acquired LQTS includes the identification and discontinuation of any precipitating drug, and the aggressive correction of any metabolic abnormalities.8
Figure 1: The patient’s first 12-lead electrocardiogram (ECG), showing marked prolongation of the QT interval (QT/QTC 582/582 ms). Note the delayed, pointed T wave and the clear ST segment prolongation that resemble the ECG morphology of long QT syndrome type 3.

Figure 2: The patient’s 12-lead electrocardiogram after correction of the patient’s calcium level. Note the complete normalization of the QT intervals.

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In this patient who presented with recurrent palpitations and syncope, the initial ECG showed marked prolongation of the QT interval. His ECG was highly suggestive of congenital LQTS type 3 (Figure 1), given the prolonged ST segment and normal pointed T-wave morphology. This presentation alone is enough to stratify the patient as a high risk for sudden cardiac death and to consider implantation of an automated internal defibrillator. These interventions were unnecessary because the calcium level had been corrected, the QT interval had normalized after correction of the calcium level, and the patient’s condition had improved clinically.

Conclusion

LQTS is congenital or acquired. Acquired causes should be ruled out first as they are more common than congenital causes, and they are reversible. Their identification avoids unnecessary diagnostic and therapeutic measures.

References