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Brief Report

Electrolyte screening in the evaluation of prolonged QTc interval

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Abstract We present a case of a previously healthy adolescent male for outpatient evaluation of prolonged QT interval. He was ultimately found to have acquired QT interval prolongation secondary to hypocalcaemia related to undiagnosed hypoparathyroidism. This case report highlights the importance of routine electrolyte analysis, even in the outpatient setting, during initial diagnostic workup for QT interval prolongation.

Keywords: Long qt syndrome; electrocardiography; electrolytes; pediatrics

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The QT interval of an electrocardiogram correlates with repolarisation of the ventricular tissue.[1] A normal QT interval in the paediatric population can be as high as 460 ms.[2] Abnormal ventricular repolarisation, as in long QT syndrome, can generate life-threatening arrhythmias such as torsades de pointes. Evaluation of patients with QT prolongation requires the clinician to distinguish between acquired and congenital long QT syndromes. Acquired QT prolongation accounts for the majority of cases and is potentially reversible.[1] We present a case of an adolescent male with prolonged QT interval secondary to hypocalcaemia associated with undiagnosed primary hypoparathyroidism. Of note, corrected QT (QTc) intervals were calculated using Bazett's formula and will be reported to adjust for heart rate variability.

Case report

A healthy 13-year-old boy with a history of prematurity complained of chest pain without associated palpitations, dizziness, or syncope. He reported increased fatigue, weakness, and bilateral hand paresthesias. Electrocardiogram obtained by his primary care physician revealed a prolonged QTc interval of 495 ms. He was referred for cardiology outpatient evaluation. He had a normal physical examination, and there was no family history of long QT syndrome, sudden death, hypoparathyroidism, or thyroid disease. The patient's prolonged QTc was confirmed on repeat electrocardiogram (Fig 1). In addition, his electrocardiogram revealed a prolongation of the onset of QRS complex to the onset of the T-wave (Q-oT interval). The prolonged Q-oT interval prompted the collection of basic laboratory studies, including a complete blood count, basic metabolic panel, and thyroid-stimulating hormone. The results were notable for hypocalcaemia (calcium 1.28 mmol/L; normal reference range 2.20–2.70 mmol/L), hyperphosphatemia (phosphorus 2.65 mmol/L; normal reference range 1.45–1.78 mmol/L), and normal-thyroid stimulating hormone. The patient had a normal echocardiogram.

The patient required hospital admission for management of his hypocalcaemia. His admission physical examination was notable for hyperreflexia. Further testing revealed a significantly decreased parathyroid hormone (<3 ng/L; normal reference range 11–74 ng/L), normal albumin, normal Vitamin D assays, and negative fluorescence in situ hybridisation for DiGeorge Syndrome. He was diagnosed with primary hypoparathyroidism, most likely autoimmune. While in the hospital, he received calcium replacement with intravenous calcium gluconate and oral calcium carbonate. His calcium level and QTc interval returned to normal range (442 ms) before discharge.

He was discharged home on calcitriol, calcium carbonate, magnesium chloride, and ergocalciferol. The patient was temporarily lost to follow-up. He

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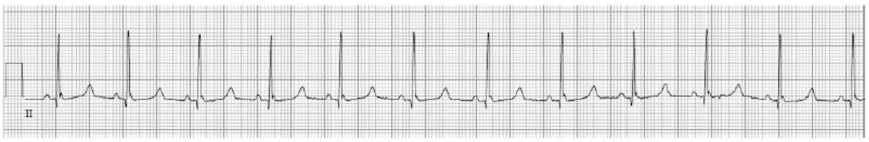

Figure 1.
Electrocardiogram lead II at initial cardiology evaluation. Sinus rhythm with QTc = 536 ms.

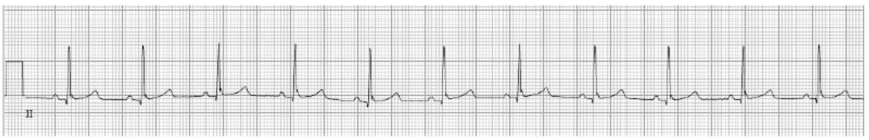

Figure 2.
Electrocardiogram lead II from 2-year follow-up. Sinus rhythm with QTc = 424 ms.

returned 15 months after discharge with a prolonged QTc interval (460 ms) and recurrence of fatigue and paresthesias. He was found to be hypocalcaemic (calcium 1.13 mmol/L), secondary to non-compliance with calcium supplementation. The patient restarted calcium supplementation. At the 2-year follow-up, he was asymptomatic with normal QTc interval (Fig 2) and calcium level (2.23 mmol/L).

Discussion

Causes of acquired QT prolongation include medications, electrolyte abnormalities, and medical conditions such as myocarditis.[1] Electrolyte abnormalities can be the primary cause of acquired QT prolongation, compound an underlying long QT syndrome, or herald the diagnosis of a medical condition such as anorexia nervosa. Whereas family history and medication review are standard while evaluating QT prolongation, basic electrolyte screening may not be routine, especially for otherwise healthy outpatients. In the above case, the patient's prolonged Q-oT interval prompted electrolyte analysis. Hypocalcaemia characteristically elongates the ST segment, resulting in a prolonged Q-oT interval.[3] This basic electrolyte analysis at an outpatient cardiology evaluation for QT prolongation led to a diagnosis of hypoparathyroidism. Hypocalcaemia associated with hypoparathyroidism is a reversible aetiology of acquired QT prolongation that has been referenced in previous case reports.[4,5]

QT prolongation can be congenital or acquired, and it mandates careful evaluation. Some authors have advocated electrolyte screening as part of the evaluation,[6] but this is not uniformly performed in seemingly healthy outpatients. When evaluating patients with long QT intervals, a high index of suspicion should be present for electrolyte abnormalities. A thoughtful history and physical examination may reveal signs and symptoms of electrolyte aberrancy; however, such history and physical examination findings can be subtle. This case highlights the need to consider routine electrolyte assessment in all patients with QT prolongation. The authors propose electrolyte screening for individuals undergoing evaluation for QTc $\geqslant$480 ms.

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Conflicts of Interest

None.

Ethical Standards

This case report does not involve human and/or animal experimentation. In compliance with our institutional protocol, parental consent was obtained and authorised the child to be a subject in a case report for possible publication in a professional journal.

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