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Seizure with Long QT Syndrome Elicited by Hypocalcaemia: Case Report

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Abstract

This is a case about a 60 years old female that presented with seizures, troponins leakage and prolonged QT on Electrocardiogram (ECG). On further investigation, she turned out to be hypocalcaemia secondary to hypoparathyroidism. She was administered calcium and vitamin D supplementation orally, which saw the QT segment returning to normal with cessation of any further episodes of seizures and troponins leakage. It is emphasized through this case report to look at all causative factors for QT prolongation and troponitis.

Introduction

Seizures may be either provoked or unprovoked. Provoked seizures, also known as acute symptomatic seizures, may result from electrolyte disorders, toxins, head injury, infectious processes, vascular anomalies, tumors or other mass lesions, and many other causes. In the year 2000, the European Society of Cardiology and the American College of Cardiology Committee jointly redefined Myocardial Infarction (MI) by an elevation of Cardiac Troponin T (cTnT) or I (cTnI) in conjunction with clinical evidence of myocardial ischaemia. Troponitis is the colloquial term used by many clinicians to describe a falsely elevated (false positive), Cardiac Troponin (cTn) result. Hypoparathyroidism is a condition of Parathyroid Hormone (PTH) deficiency. Primary hypoparathyroidism is a state of inadequate PTH activity. In the absence of adequate PTH activity, the ionized calcium concentration in the extracellular fluid falls below the reference range. This case stresses the value of Long QT Syndromes (LQTS) screening.

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Case presentation

The patient is a 60 years old female who came to the hospital with complains of loss of consciousness with seizures of about 15-20 minutes sans urinary incontinence and tongue bite. The patient has no previous history of seizures. She recovered spontaneously and consciousness was regained without dizziness or headache. The patient doesn't state any aura, palpitations, dizziness or sweating before the collapse. A diagnosis of prolonged QTc was made on resting ECG. MRI brain was negative for any organic causes of seizures. There were no further episodes of seizures. The ECG on admission showed a QTc of 600 Milliseconds (ms) according to the Bazzett's formula: QT interval/square root of the R-R interval (Figure 1). She had raised Cardiac Troponin I (cTnI) of 0.133 -> 0.386 -> 0.420. A Lumbar Puncture (LP) was done to rule out meningitis (Table 2). She was kept on valproate 500 mg twice daily with consult from neurology. The examination was unremarkable, pulse rate of 101 beats per minute and blood pressures of 148/77 mmHg. She had normal cardiac examination with normal heart sounds.

On admission her corrected calcium levels were <1.27 mmol/l (<5 mg/dl). Her blood results are shown in Table 1. She was kept on oral calcium and vitamin D supplements and within 2 days her serum calcium levels increased to 2.17 mmol/l (8.7 mg/dl) and the QTc was reduced to 480 ms. Her parathyroid levels turned out to be 6.24 Pg/ml.

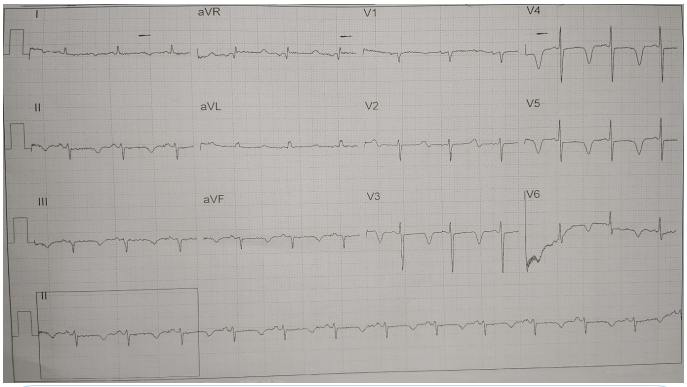

Figure 1: 12-Lead Electrocardiogram (ECG).

Table 1: Serum Hormone and Electrolytes.

Test Name	Test Result	Normal Value
Paratharmon	6.24 Pg/ml	(10-65 Pg/ml)
Electrolytes		
Sodium	140	(136-145) M.Eq/L
Potassium	3.6	(3.5-5.1) M.Eq/L
Chloride	99	(98-107) M.Eq/L
Bicarbonate	27	(23-29) M.Eq/L
Calcium	<5.0	(8.6-10.0) mg/dl

Table 2: Cerebrospinal Fluid Detailed Report.

CSF FORD/R			
Volume	**Color**	**Appearance**	**Coagulum**
6 ml	Colorless	Clear	Absent
Chemical Examination	**Results**	**Reference Ranges**	
Protein	36 mg /dl	New born: up to 120 mg/dl	-
		<1 month: 20-80 mg / dl	-
		>1 month: 20-40 mg/dl	-
Sugar	85 mg/dl	60 % of plasma glucose	-
Microscopic Examination			-
RBC	3/cumm	Nil	-
WBC	2/cumm	0-5/cumm	-

CSF: Cerebrospinal Fluid; D/R: Detailed Report.

Discussion

The QT segment on ECG reflects both the period of depolarization (influx of Na+, Ca2+, efflux of K+) and repolarization (extraction of Na+, Ca2+) of cardiac myocytes. Lengthening QTc is associated with a higher risk of torsades de pointes, Ventricular Tachycardia (VT) and Ventricular Fibrillation (VF) and this ECG finding can be acquired, congenital (LQTS) or due to a combination of both. LQTS has over recent years been proposed as a common cause of arrhythmia induced sudden cardiac death [1-4]. It was first described in the late sixties by Jervell and Lange-Nielson in association with sensorineural l deafness and an autosomal recessive inheritance pattern [5]. Soon after, Romano and Ward described similar families, this time with an autosomal dominant inheritance pattern and no deafness [6,7]. The distinction between the groups based on inheritance pattern is no longer as clear, as some autosomal recessive cases of Romano-Ward have been reported [8]. There is now growing data linking LQTS genes to specific cardiac ion channel defects, and in turn to specific triggers and ECG findings. Eight genetic LQT subtypes have been identified to date with 471 different mutations and 124 polymorphisms [2]. These mutations effect cardiac ion channels with the net result of reducing passage of K+ ions or increasing passage of Na+ ions. It is beyond the scope of this article to define each subtype; we recommend the recent review by Modell and Lehmann, which includes links to LQTS databases and registries [2]. The main stay of therapy for patients with LQTS are beta blockers and, dependent on risk profile, implantation of an ICD [1,3]. There is evidence suggesting individuals may have a genetic predisposition to acquired forms of long [2]. When assessing a child with history of syncope excluding LQTS is important. We recommend using the scoring system initially described by Schwartz as a guide to predicting the likelihood of LQTS [9,10]. To summarize this scoring system, patients with a QTc greater than 450 ms are considered abnormal, while those with a QTc greater than 460 ms score more highly. Similarly, family history, syncope, previous torsades de pointes, relative bradycardia for age and ECG findings of T-wave alternans/notched T wave are all included in the scoring tool (our patient would have scored five points on diagnostic criteria making LQTS likely). Whilst the congenital forms are more common in childhood, acquired causes must be considered. These include hypokalaemia, hypomagnesemia, hypocalcaemia and the use of drugs that are known to potentiate the QT (macrolides, antifungals, antiarrhythmics, antihistamines, proton pump inhibitors and psychotropic drugs) [1].

Calcium regulates and carries ionic currents that are responsible for the myocardial action potential and its chronicity [11]. Low extracellular [Ca2+] is thought to shift the activation of the action potential to a lower membrane electro-potential thereby increasing excitability.

The commonest cause of hypocalcaemia in childhood is nutritional vitamin D deficiency, and rarer causes include pseudohypoparathyroidism, hypoparathyroidism, vitamin D receptor abnormalities and calcium sensing disorders [12]. The aetiology of hypoparathyroidism in this patient is not clear, although normal calcium in infancy suggests that it may be acquired. A number of inherited forms of hypoparathyroidism are described, some as part of dysmorphic syndromes, including velocardiofacial syndrome (associated with 22q deletions). Autoimmune hypoparathyroidism can develop during childhood. This patient had no other features of a multiple autoimmune endocrinopathy syndrome and parathyroid antibodies were negative.

Conclusion

A long QT interval is caused by a number of genetic syndromes; however, we report an unusual presentation of hypocalcaemia. The case highlights the importance of excluding an acquired aetiology in patients with a long QT interval.

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