Unusual case of seizures due to prolonged QT syndrome - A Case Report

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ABSTRACT

Long QT syndrome (LQTS) is a cardiac electrical disorder. One of the rare symptoms of long QT caused by ventricular arrhythmia is seizure. Patients with LQTS may develop seizures due to an acute hypoxic-ischemic event associated with a ventricular arrhythmia. We present a case of a 47-year-old male who came to the Emergency Department (ED) with seizures and was diagnosed as LQTS. The cardiac cause of seizures was suspected because the patient was pulseless during the episode of seizures. The patient developed refractory ventricular tachycardia in the ED and was cardioverted (synchronized) multiple times. He was also put on amiodarone infusions. The patient was then urgently shifted to the Coronary Cath Lab for temporary pacemaker insertion with overdrive pacing. He was advised for an automated implantable cardioverter-defibrillator. This case illustrates that prolonged QT syndrome can masquerade as seizure. Therefore, a careful examination should be done in the patient presenting with the same and a cardiac cause should be excluded. Delays in recognition and treatment may expose the patient to a high risk of sudden cardiac death.

Key words: *Arrhythmias, Prolonged QT, Seizures*

Long QT syndrome (LQTS) is a cardiac electrical disorder caused by prolonged ventricular repolarization which can be congenital or caused by drugs [1]. It has been found to affect approximately 1 in 2000 people worldwide [2]. Two entities of idiopathic LQTS have been described: The first by Jervell and Lange-Nielsen [3] in 1957; the second by Ward [4] in 1963. LQTS has a propensity to ventricular tachyarrhythmia's, which may lead to syncope, cardiac arrest, or sudden death [5,6]. Athough very rare, patients with LQTS may develop seizures related to an acute hypoxic-ischemic event associated with a ventricular arrhythmia. Alternatively, patients with recurrent and unprovoked seizures may have epilepsy [7]. We describe a case of electrical storm secondary to LQTS presenting with seizures.

CASE REPORT

A 47-year-old diabetic and hypertensive male came to the Emergency Department (ED) with complaints of up rolling of eyes with 3–4 episodes of seizure-like activities and 2 episodes of vomiting. The history of seizure-like activity was elicited from his wife as the patient had no recollection of the same. On arrival to the ED, the patient was conscious and oriented with a pulse of 90/min, blood pressure of 100/70 mmHg, respiratory rate of 20/min. patient's temperature was 98.6 F, and saturating at 98% on room air. His blood glucose was 299 mg/dL. His secondary survey was unremarkable. He was taking regular medications for diabetes and hypertension and gave no history of similar events in the past. He gave no history of antidepressants or use of any recreational drug use. There was no significant family history noted.

The patient was being evaluated for suspected seizures, neurology consult sought, and the patient was administered a loading dose of levetiracetam. Shortly during the stay in the ED, the patient had an episode of seizures during which time he became pulseless and the cardiac monitor showed ventricular tachycardia (VT). The first episode reverted spontaneously, and subsequently, the ECG showed long QT with corrected QT (QTc) of 621 ms (Fig. 1). Two dimensional echocardiography revealed mild left ventricular dysfunction with an ejection fraction of 40% and associated global hyperkinesias. Complete blood counts, renal profile, electrolytes, liver function test, and thyroid profile were largely unremarkable. His serum calcium was 9.7 mg/dl, potassium - 4.2 mEq/l, and magnesium - 2.35 mEq/l. Cardiac enzymes revealed slightly elevated troponin I - 0.41 ng/mL (0–0.04).

The patient then developed refractory VT in the ED (Fig. 2) and was cardioverted (synchronized) multiple times. He was also put on amiodarone infusions. The patient was then urgently shifted to the Coronary Cath Lab under the cardiology team for temporary pacemaker insertion with overdrive pacing and coronary angiogram which revealed normal coronaries. The patient was kept in the cardiac care unit and recovered well. He was advised for an automated implantable cardioverter-defibrillator (AICD) device implantation to prevent sudden cardiac death, but the patient did not consent for the same and left the hospital against medical advice after 3 days with a final diagnosis of LQTS with electrical storm. The patient got the implant from another hospital and is now doing better.

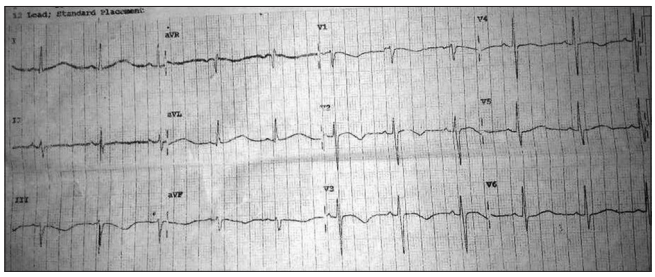

Figure 1: ECG shows prolonged QT

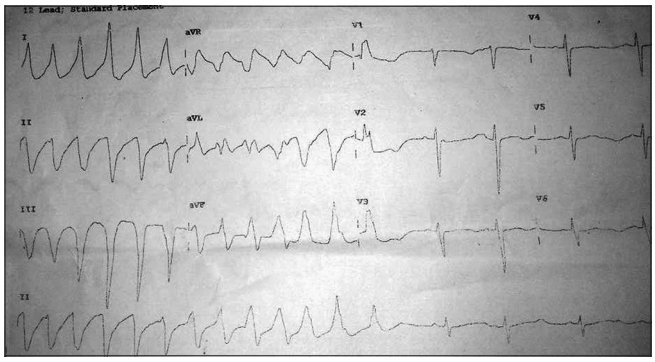

Figure 2: ECG shows ventricular tachycardia

DISCUSSION

The prolonged QT syndrome represents a variety of congenital and acquired disorders of ventricular repolarization characterized by a prolonged-corrected QT interval on the electrocardiogram [8]. A normal QT is defined when it is <440 ms, and if QT >450 ms, it produces prolonged ventricular repolarization predisposing to malignant ventricular arrhythmias. The QT interval on the ECG reflects the length of ventricular action potential and an interval over 0.44 seconds should be considered prolonged [9,10]. There are medications that can lengthen the QT interval and upset heart rhythm including certain antibiotics such as chloroquine and erythromycin; antihistamines such as terfenadine, diphenhydramine, and loratadine; tricyclic antidepressants such as imipramine, citalopram, and venlafaxine; diuretics; certain cholesterol; and sugar medications. Hypocalcemia, hypomagnesemia, hypokalemia, and hypothermia are established causes as well. Treatment is mainly treating the underlying cause.

Congenital QT syndrome patients will give a history of similar episodes in the past or syncope episode. Acquired are usually incidentally diagnosed when patients show symptoms. Thus, a thorough history is imperative in the identification of these patients. A family history of sudden unexplained death or deafness also suggests congenital LQTS, and the recent introduction of new drugs, in particular, antiarrhythmics such as stall and flecainide may be an acquired cause of QT prolongation. Treatment of the patient with LQTS includes medical management (usually beta-blocker therapy), left cardiac sympathetic denervation, and AICD placement [7].

Our case depicts how LQTS camouflaging as seizures convinced us to treat the former as a seizure disorder, causing a delay in both the diagnosis and treatment, exposing the patient to risk of sudden cardiac death. It is therefore essential that whenever the patient comes to emergency with features/ symptoms or history of seizures, our point of care testing in such patients should invariably include 12 lead ECG to rule out the diagnosis of LQTS. Patients with symptomatic idiopathic LQTS have erroneously been considered to be "epileptic" patients and treated with anticonvulsants [11].

CONCLUSION

This case illustrates that prolonged QT syndrome can masquerade as seizure. Therefore, a careful examination should be done in the patient presenting with the same and a cardiac cause should be excluded. Delays in recognition and treatment may expose the patient to a high risk of sudden cardiac death.

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