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Case Report

QT INTERVAL PROLONGATION AFTER PREMATURE VENTRICULAR CONTRACTIONS (PVCS)

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ABSTRACT:
Long QT syndrome (LQTS) is an inherited ion channelopathy resulting in abnormal ventricular repolarization and abnormal prolongation of QT interval on the ECG. Syncope, fainting, cardiac arrest, and sudden death are common manifestations of LQTS. We present a case report that describes a patient with prolonged QT interval after extrasystoles and a family history of sudden cardiac deaths.
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Keywords: Long QT syndrome; Sudden cardiac death; Extrasystoles

INTRODUCTION

The long QT syndrome (LQTS) is a disorder of myocardial repolarization characterized by a prolonged QT interval on the electrocardiogram (ECG) and an increased risk of sudden cardiac death. This syndrome is associated with an increased risk of a characteristic life-threatening cardiac arrhythmia, known as Torsade de Pointes or "twisting of the points" (TdP). Despite the availability of genetic analysis, ECG is still very important tool to establish a diagnosis of LQTS. We present the case of a young lady with a strong family history of sudden cardiac death and remarkable prolongation of QT interval after PVCs, documented on a 24-hour ECG record.

CASE PRESENTATION

The patient, a 36-years-oldwoman, had a history of frequent syncopal attacks and palpitations during the previous two years. She was referred from the department of neurology to our clinic for evaluation of syncope and run of wide QRS tachycardia on ECG.

Her family history was remarkable, including a recent sudden death of her sister at 38 while sleeping and unexplained sudden deaths of her aunt and cousin (in a traffic accident) at ages 43 and 38 respectively. Her routine ECG, echocardiography, and blood workup were unremarkable and her 24-hour ECG revealed short run of non-sustained ventricular tachycardia (two or three episodes). Based on the ECG, Holter (Figs. 1 and 2), and her clinical history, a presumed diagnosis of LQTS type 2 was made. A beta blocker (Metaprolol 200 mg) was prescribed and an ICD was implanted for the primary prophylaxis of sudden cardiac death.

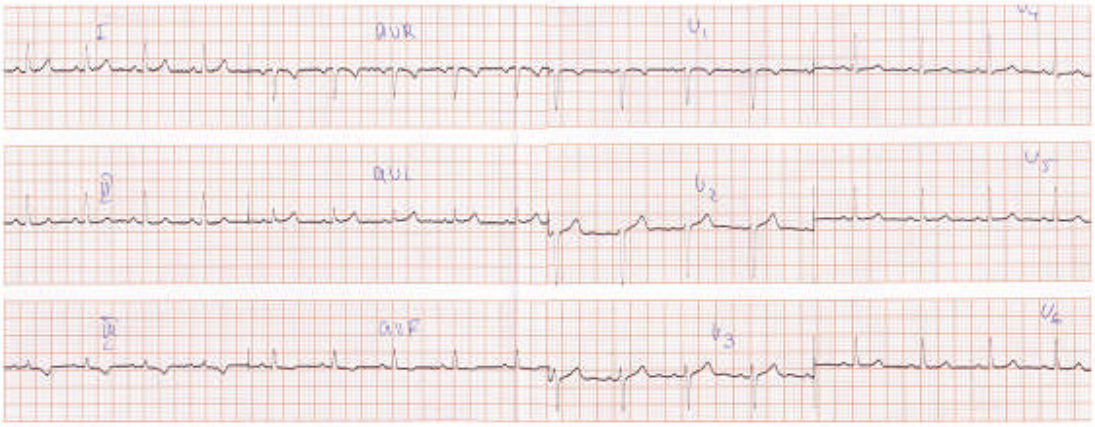
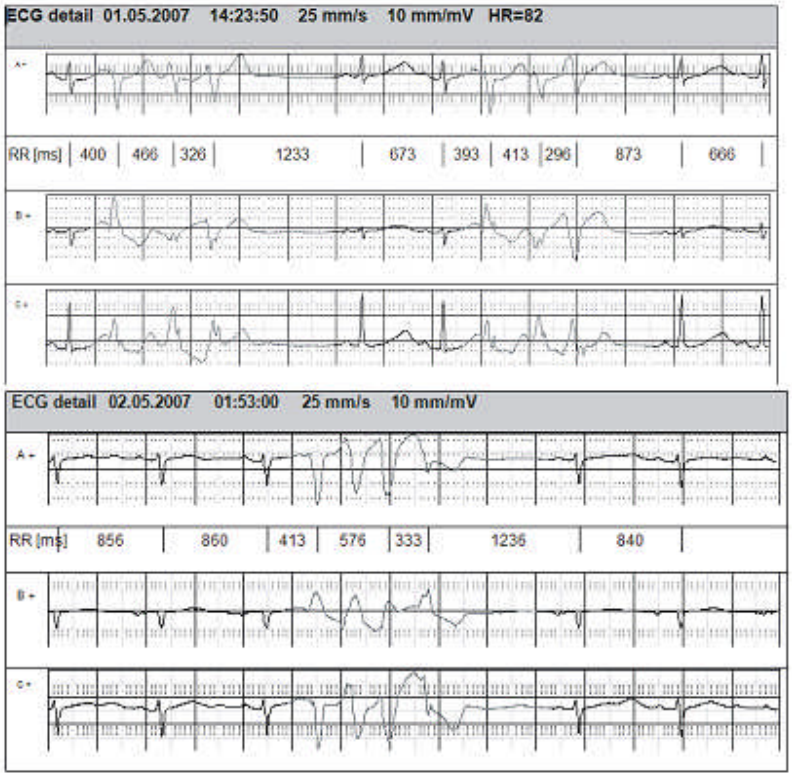


Figure 1. Routine ECG showing normal QT/QTc intervals



Fifure 2. 24hour Holter ECG strip recorded in our patients shows sudden prolongation of QT interval after PVCs (arrow).

DISCUSSION

Instances of QT prolongation after PVC were first reported in 1915 and are not frequently observed in either cardiac patients or healthy individuals. The mechanism is unknown, but it is considered to be related to cardiac memory, similar to the T-wave changes following intermittent ventricular pacing, LBBB, or ventricular pre-excitation (1).

The clinical significance of this is unknown. Extrasystoles are common in otherwise healthy young patients. For most, this is a harmless condition that does not require a specific therapy. However, the rare patient who is at risk for lethal arrhythmias must be identified (2). In this particular case, structural heart disease was excluded by echocardiography. Nevertheless, we focused on symptoms suggestive of tachyarrhythmias (such as syncope and rapid heartbeat that terminates abruptly), and her evident family history of early-age sudden deaths. Arrhythmic syncope in patients with long QT syndrome is often misdiagnosed as vasovagal or hysterical. The patient's family members' early deaths were consistent with the presence of a familial long QT syndrome. The long QT syndrome may be congenital or acquired. Some pharmacological agents can cause acquired long QT syndrome. Agents and conditions that reduce net repolarizing current amplify the intrinsic spatial dispersion of

repolarization, creating the substrate for the development of re-entry. The result is a prolongation of the QT interval, abnormal T waves, and development of polymorphic re-entrant ventricular tachycardia displaying characteristics of Torsades de Pointes (TdP) (3). These conditions also predispose M-cells and Purkinje fibers to develop early afterdepolarization-induced extrasystoles that are thought to trigger episodes of TdP. Agents that prolong the QT interval but do not increase transmural dispersion of repolarization are not capable of inducing TdP. The available data suggest that the principal problem with the long QT syndrome is the dispersion of repolarization that often accompanies prolongation of the QT interval (4).

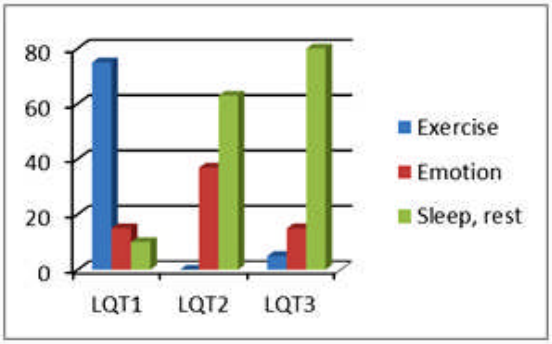


Figure3. Fatal cardiac events classified according to the three main triggers in the genotypes (Circulation 103:89, 2001)

As an inheritable channelopathy, family screening in LQTS is clearly important. LQTS has been classified based on LQTS genes and 10 previously identified types of this pathology. But there is agreement that LQT1, LQT2 and LQT3 variants account for greater than 90 percent of all genotyped patients (5). Triggers for cardiac events differ strikingly among the genetic variants of LQTS (6). We have classified our patient as probable LQT2 type (7) and hence beta blockers (8, 9) and ICD use was considered the best course of primary prevention in management (9) of our patient. The basis of our diagnosis was the evidence given by Peter J, Schwartz et al. in their landmark study "Geno-type-Phenotype Correlation in Long QT Syndrome" (10). Fatal cardiac events are also classified according to the three main triggers in the genotypes (Fig. 3).

CONCLUSIONS

QT interval prolongation after PVCs is a rare clinical manifestation of LQTS. In our particular case, the risk of sudden cardiac death was stratified as high and, according to the guidelines and experts consensus, we managed the patient with beta blockers and ICD insertion. On follow-up patient has clinically benefited.

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