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Case Report Open Access

Hidden Reason for Long QT

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Abstract

Long QT syndrome either congenital or acquired is a fatal condition which unfortunately results in 'torsade de pointes' (TdP) type ventricular arrhythmia, recurrent syncopes and sudden cardiac deaths. The definite diagnosis is lifesaving in order to understand if there is predisposing factor or not. In acquired long QT syndrome ion channel disorder is secondary to metabolic disorder or drugs. Here we present 36 years-old woman patient hospitalized with congestive heart failure secondary to dilated cardiomyopathy. She experienced ventricular tachycardia (VT) episode under medical control, hypokalemia was thought to be the underlying cause. Her anamnesis deepened, surprisingly primary hyperaldosteronism appeared which was first diagnosed eight years ago. Mexsiletine treatment (group IB anti-arrhythmic drug) started with the purpose of preventing recurrent VT episodes after potassium replacement. Thoracoabdominal angiographic computerized tomography was performed to see development of the adenoma, subsequently patient was referred to general surgery department for advanced treatment.

Introduction

In routine clinical practice ventricular tachycardia was observed which is an absolute emergent situation should be treated immediately. Even though VT is generally due to ischemic heart diseases, rare causes should be kept in mind.

Here we discuss an unusual case of a patient defibrillated because of ventricular tachycardia, investigated thoroughly as a result primary hyperaldosteronism diagnosis appeared. Diuretic usage and primary hyperaldosteronism were thought to be the causes of the hypokalemia which is responsible for the VT.

Case Report

A 36 years-old woman with a diagnosis of dilated cardiomyopathy (coronary angiography was performed before) presented to the emergency with palpitation, dyspnea and nausea. On physical examination she was tachypneic and agitated. Her blood pressure was 138/54 mmHg and oxygen saturation was 94% with pulse oximetry. Auscultation of the patient revealed gallop rhythm due to tachycardia. Bilateral basilar rales were heard on pulmonary auscultation. The electrocardiogram (ECG) revealed sinus tachycardia, no ST segment changes were seen. QTc duration was 631ms in the first ECG (Figure 1). In the laboratory tests parameter that were abnormal: troponin level 0,2 ng/ml (troponin normal level 0-0,06 ng/ml) thought to be secondary to tachycardia and volume load; potassium level: 3,3 mEq/L (normal level 3,5-5,5 mEq/L) despite usage of spironolactone.

Patient was diagnosed with decompansated congestive heart failure based on her continuing complaints despite usage of almost maximal heart failure medical therapy. She was hospitalized to cardiology service for further evaluation after first intervention in the emergency. Under medical therapy she had an episode of ventricular tachycardia, patient was defibrillated successfully and arterial blood gas showed us hypokalemia (K: 2,1 mEq/L). After potassium replacement non-sustained ventricular tachycardia episodes stopped. Mexiletine treatment was started in order to stop the VT episodes. Shortly after defibrillation QTc duration was 680ms in the ECG (Figure 2). After potassium replacement QT duration was dropped to 529 msn in the ECG (Figure 3).

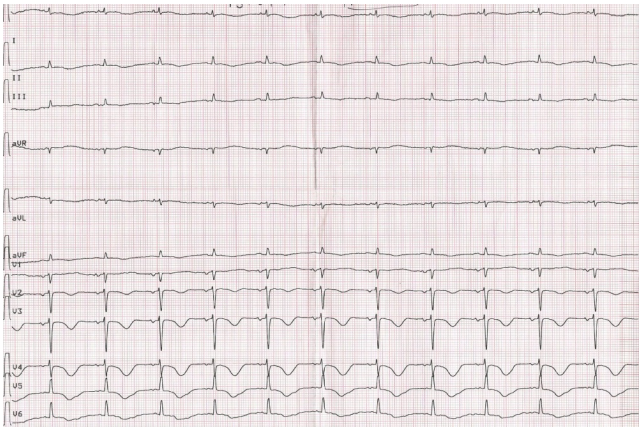

Figure 1: First ECG showing the QTc duration as 631 ms.

Before hospitalization she was using furosemide 40 mg tablet once a week. After she was hospitalized intravenous (iv) diuretics (furosemide 100 mg iv+spironolactone 25 mg) was started. The VT episode was thought to be result of intense diuretic usage nevertheless her medical anamnesis deepened, nevertheless primary hyperaldosteronism was diagnosed eight years ago. The laboratory test done in 2007, showed plasma aldosteron level/plasma renin level: 60,4 (plasma aldosteron level: 29 ng/ml-plasma renin activity 0,48 ng/ml). Captopril suppression verified the diagnosis and computerized tomographic angiography detected the ipsilateral adrenal adenoma. Operation to the adrenal adenoma was recommended in 2007, she refused L/S operation just due to fear.

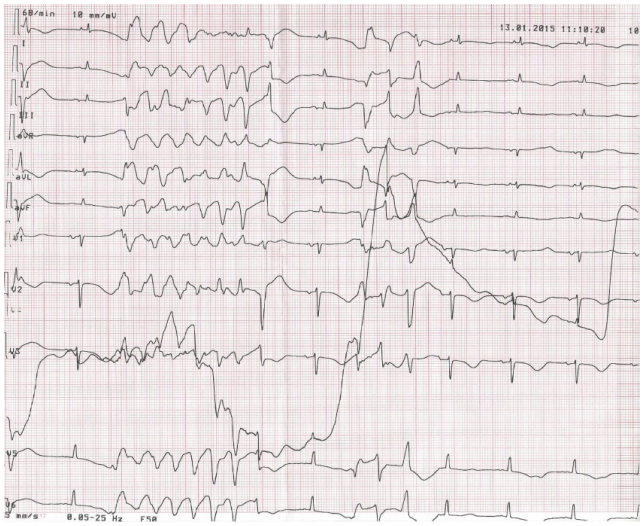

Figure 2: ECG showing the QTc duration as 680 ms after defibrillation.

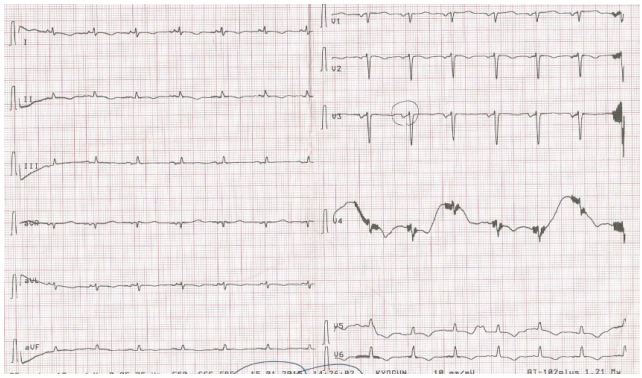

Figure 3: ECG showing the QTc duration as 529 ms after potassium replacement.

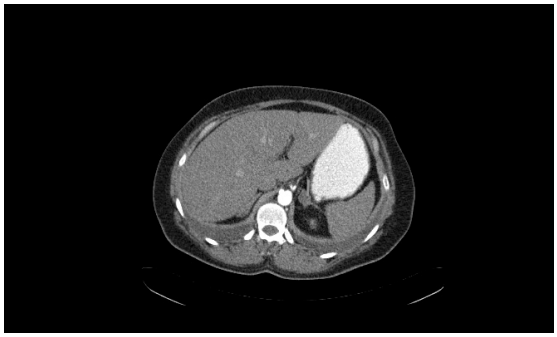

Figure 4: Thoracoabdominal angiographic computerized tomography showing the adrenal adenoma's final spread.

Once again thoracoabdominal angiographic computerized tomography with contrast was performed in order to see the adrenal adenoma's final spread (Figure 4). She was referred to the general surgery department for advanced treatment. We planned to use 24-h Holter to understand if the ventricular tachycardia episodes would continue after the surgical removal of the adrenal adenoma.

Discussion

The long QT syndrome, caused by delayed cardiac repolarisation, is associated with fatal cardiac arrhythmias such as ventricular tachycardia and ventricular fibrillation. We classify long QT syndrome as congenital and acquired which is more common [1].

In congenital long QT syndrome there is a mutation in genes that regulate ion movement in the channels of the heart myocytes. Patients are especially sensitive to ventricular arrhythmias when sympathetic activity increased such as exercise and emotional stress. Nevertheless heart rate takes part an important role and it has a great influence on the occurrence of TdP in some congenital form of long QT (LQT1) syndrome. Hyperpolarization and Cyclic Nucleotide (HCN)- gated channels represent the molecular correlates of the funny pacemaker current (If) considered to influence heart rate [2]. From that idea beta-blockers, calcium channel blockers, ivabradine (If channel blocker) may have a role in anti-arrhythmia beyond rhythm control [3].

Acquired long QT syndrome is usually due to drugs prescribed by doctors [4]. Clinicians should be aware of the drugs side effect used in the therapy. Unconsciously it might harm patients seriously. First goal in drug related long QT is discontinuation of the drug, magnesium and potassium replacement is also effective to stop the episodes. Anti-arrhythmic drugs should be avoided in addition to overdrive cardiac pacing is highly effective [5].

The other cause of acquired long QT is metabolic which includes electrolyte disorders (hypokalemia, hypomagnesemia and hypocalcemia), starvation, endocrine disorders (hypothyroidism) and pheochromacytoma [6]. In our patient the reason for the long QT was hypokalemia. Fortunately primary hyperaldosteronism diagnosis was not missed in order to treat patient accurately. QT interval is prolonged but QT dispersion is maintained in primary hyperaldosteronism patients. Unchanged QT dispersion will be responsible infrequency of VT among primary hyperaldosteronism patients [7]. In the literature there is only one case describing primary hyperaldosteronism related to long QT resulted in ventricular tachycardia [8].

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