Acquired long QT Syndrome: Case report

Síndrome de QT longo adquirido: Relato de caso

António Filipe Pinto Rodrigues
University Hospital Center of Cova da Beira - Covilhã, Portugal
Dr. Lopes Dias Higher School of Health, Polytechnic Institute of Castelo Branco, Portugal
E-mail: afrodrigues6@gmail.com

Bruno Tiago Madeira Morais Valentim
University Hospital Center of Cova da Beira - Covilhã, Portugal
Dr. Lopes Dias Higher School of Health, Polytechnic Institute of Castelo Branco, Portugal
E-mail: bruno.valentim81@gmail.com

ABSTRACT
Long QT syndrome, whether genetic or acquired, is closely associated with arrhythmogenic potential and consequently with syncope or even sudden death. This case report concerns a 77-year-old female who attended the emergency department for right-sided hemiparesis, diagnosed with ischemic stroke. She underwent Holter monitoring for paroxysmal atrial fibrillation, where an increase in the QT interval was found, which triggered a Torsades de Pointes by R on T phenomenon. After investigating the cause, it was found that the origin of the increase in the QT interval was iatrogenic. The drug that was influencing this change was Sotalol, which after being removed from the therapeutic table, reverted the QTc interval to within normal values.

Keywords: Long QT Syndrome, Stroke, *Torsades de Pointes*.

1 INTRODUCTION

Long QT syndrome (LQTS), whether genetic or acquired, may be associated with high arrhythmogenic potential. The QT interval constitutes ventricular electrical systole and is defined as the time from the onset of the QRS complex to the end of the T wave. On surface ECG, this syndrome is characterized by an increased QT interval and is often associated with morphological changes in the T wave. (1). Identifying pathological changes in the QT interval is challenging but essential to avoid complex malignant ventricular dysrhythmias that can lead to syncope, cardiac arrest and even sudden death (1) . Congenital increase in QT interval is caused by mutations associated with ion channels. On the other hand, the acquired form can be caused by iatrogenesis, electrolyte disturbances, central nervous system diseases, among others.(2).

The aim of this case report is to show a patient with acquired LQTS and discuss possible etiologies.

2 CASE REPORT

Female patient, 77 years old.

Personal history of atrial fibrillation (AF), hypertension, concentric left ventricular hypertrophy, diabetes *mellitus* and glaucoma.

Medicated with dabigratan, humalog, janumet, omeprazole, atorvastatin+perindopril+amlodipine, sotalol 160 mg, nebivolol 5 mg, levothyroxine, monitrate, timolol, furosemide and alprazolam.

On January 16, 2019, the patient was admitted to the emergency department (ER) of the Centro Hospitalar e Universitário da Cova da Beira, due to neurological changes with right hemiparesis.

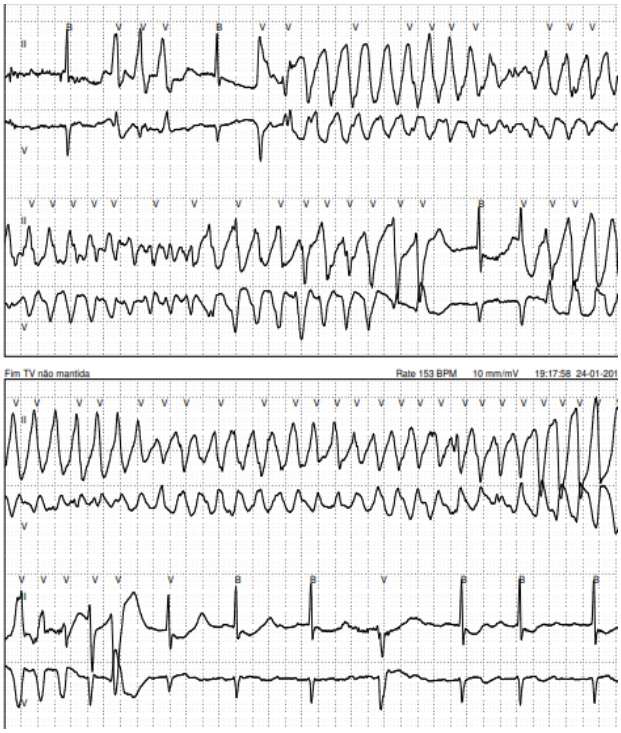
A Via Verde stroke pathway was activated, and a computed tomography scan of the skull revealed acute ischemic injury. The electrocardiogram (ECG) in the service showed sinus bradycardia, 50 bpm and nonspecific changes in ventricular repolarization.

The patient was admitted to the stroke unit for stabilization and a protocol cardiovascular study, and on the second day of admission a 24-hour Holter was performed. The 24-hour electrical study showed AF throughout the recording, frequent polymorphic ventricular ectopy with 36 pairs, 8 runs, 2 episodes of polymorphic non-maintained ventricular tachycardia of the *torsade de pointes* type, initiated by an R on T phenomenon, and a maximum QT of approximately 600 ms.

After alerting the patient of the finding, an analytical collection was performed, namely an ionogram to screen for electrolyte disturbances. The analytical results did not show altered values. For iatrogenic study, medication was reviewed and it was found that the patient was taking Sotalol, a long QT inducing drug.

During hospitalization, sotalol was discontinued and replaced by propafenone hydrochloride. On the 19th day of hospitalization, the patient presented with hypomagnesemia, and an ECG showed AF with +/- 90 bpm and nonspecific changes in ventricular repolarization (QT 410 ms - normal).

Figure 1: Electrocardiographic tracing of Holter recording showing torsade de pointes.



3 DISCUSSION

Holter recording contributed to the dynamic analysis of the QT interval, T wave morphology and detection of arrhythmic phenomena.(3). *Torsade de Pointes* (a ventricular arrhythmia often associated with increased QT interval) is a polymorphic ventricular tachycardia with cyclic oscillation of polarity, morphology and duration of QRS complexes, which may be self-limiting or evolve to ventricular fibrillation.(2).

Acquired LQTS has numerous etiologies, ranging from pharmacological interactions to electrolyte disturbances (hypopotassemia and hypomagnesemia), which makes its clinical management challenging.

The healthcare professional should be aware of the electrocardiographic patterns of this syndrome, which may be essential for correct diagnosis and treatment, since it is an entity strongly associated with a high risk of sudden death.

4 CONCLUSION

In this case, although the patient had hypomagnesemia and a history of hypopotassemia, the origin of the long QT was proven to be iatrogenic, since the withdrawal of sotalol decreased the QT interval and the health professional played an important role in alerting the UAVC.

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