Case Reports

Acquired Long QT Syndrome after Acute Myocardial Infarction:

A Rare but Potentially Fatal Entity

Samuel S. Gordon, MD
John Hollowed, MD
Justin Hayase, MD
Carlos Macias, MD
Jessica Wang, MD, PhD
Holly R. Middlekauff, MD

Acquired long QT syndrome is typically caused by medications, electrolyte disturbances, bradycardia, or catastrophic central nervous system events. We report a case of myocardial infarction–related acquired long QT syndrome in a 58-year-old woman that had no clear cause and progressed to torsades de pointes requiring treatment with isoproterenol and magnesium. Despite negative results of DNA testing against a known panel of genetic mutations and polymorphisms associated with long QT syndrome, the patient's family history of fatal cardiac disease suggests a predisposing genetic component. This report serves to remind clinicians of this potentially fatal ventricular arrhythmia after myocardial infarction. ***(Tex Heart Inst J 2020;47(2):163-4)***

Acquired long QT syndrome (LQTS) is typically attributed to recognized causes such as medications, electrolyte disturbances, bradycardia, and catastrophic central nervous system events.[1] Heralded by sudden and frequent ventricular ectopic beats, acquired LQTS must be promptly recognized and treated to prevent it from progressing to polymorphic ventricular tachycardia, a potentially fatal condition, also known as torsades de pointes. We report the case of a 58-year-old woman who had a myocardial infarction (MI) and severe arrhythmic complications consequent to acquired LQTS.

Case Report

A 58-year-old woman presented at the emergency department 30 minutes after the sudden onset of crushing substernal chest pain. Her medical history included hypertension, hyperlipidemia, and chronic tobacco use. Her family history included the deaths of her mother of MI at 42 years of age, her sister of heart failure at 57 years, and her sister's niece of heart failure in her 20s.

The patient's electrocardiogram (ECG) at presentation revealed an anterior ST-segment-elevation MI, sinus rhythm, and a corrected QT interval (QTc) of 509 ms (Fig. 1A). While being evaluated in the emergency department, the patient experienced cardiac arrest due to ventricular fibrillation, and she was treated with electrical defibrillation, 1 mg/mL of intravenous (IV) epinephrine, and a 150-mg bolus of IV amiodarone. The patient was then taken urgently to the catheterization laboratory, where an angiogram revealed an acute thrombus completely obstructing the mid left anterior descending coronary artery (LAD). The LAD was immediately and successfully stented. The next day, in the cardiac care unit, a contrast-enhanced echocardiogram revealed a left ventricular ejection fraction of 0.40 to 0.45 and a regional wall-motion abnormality in the LAD territory.

On hospital day 3, telemetric ECG tracings revealed a markedly increased burden of ventricular ectopy, thought to be due to recurrent ischemia (Fig. 1B). The patient was treated with a 150-mg bolus of IV amiodarone and scheduled for repeat angiography. However, reinspection of the ECG tracings revealed no evidence of ischemia, but a QTc of 757 ms (Fig. 1C), so repeat angiography was deferred. On the basis of the patient's marked QTc prolongation and polymorphic ventricular tachycardia, the diagnosis of acquired LQTS was made. Treatment with 2 mg of IV magnesium and with IV isoproterenol titrated to increase the heart rate to >100 beats/min promptly resolved the ventricular arrhythmia. Review of the patient's chart for electrolyte levels,

Key words: *Long QT syndrome/etiology/genetics; torsades de pointes; myocardial infarction*

From: *Department of Medicine (Dr. Gordon) and Division of Cardiology (Drs. Hayase, Hollowed, Macias, Middlekauff, and Wang), David Geffen School of Medicine at UCLA, Los Angeles, California 90025*

Address for reprints: *Holly R. Middlekauff, MD, Department of Medicine, Division of Cardiology, David Geffen School of Medicine at UCLA, A2-237 CHS, 650 Charles Young Dr. S., Los Angeles, CA 90025*

E-mail: *hmiddlekauff@mednet.ucla.edu*

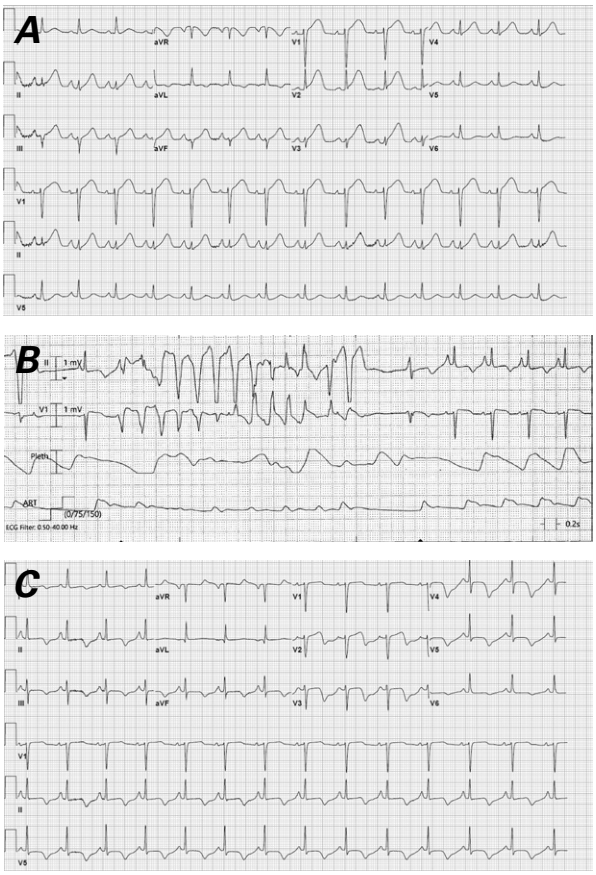

Fig. 1 ***A****) Electrocardiogram (ECG) on admission shows a QT interval of 420 ms and corrected QT interval (QTc) of 509 ms.* ***B****) Telemetric ECG tracing on hospital day 2 shows polymorphic ventricular tachycardia (torsades de pointes).* ***C****) Six hours later, the ECG tracing shows a QT interval of 640 ms and QTc of 757 ms.*

medications, and heart rates revealed no clear cause of the QTc prolongation.

The patient's QTc interval gradually decreased, and isoproterenol was discontinued. On hospital day 6, she was discharged with a QTc of 461 ms (Fig. 2). A DNA sample obtained while she was in the cardiac care unit was tested against a panel of known genetic polymorphisms associated with LQTS.[2] The results were negative. The patient was lost to follow-up 2 months later.

Discussion

After an MI occurs, the QTc is minimally prolonged, but typically remains within the normal range.[3] This QTc prolongation often correlates with infarct size and predicts a poorer prognosis, but does not mandate any specific therapy and is not associated with torsades de pointes.[3] However, this was not true in our case.

Our patient's acute MI led to torsades de pointes. To our knowledge, fewer than 2 dozen previous reports of infarction-related acquired LQTS have been reported in the literature.[2,4] The QTc prolongation in this case followed a time course consistent with that described

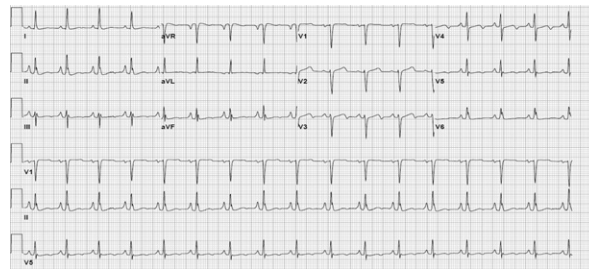

Fig. 2 *Electrocardiogram on day of discharge (hospital day 6) shows a QT interval of 360 ms and QTc of 461 ms.*

in the first reported case,[4] reaching maximal prolongation 48 to 72 hours after the initial ischemic event, then returning to normal within several days. One proposed mechanism for this MI-related acquired LQTS is amplification of the inherent dispersion of refractoriness within the different layers of the normal myocardium.[5,6]

Interestingly, in a series of 13 patients with infarct-related LQTS, Crotti and colleagues[2] found that 11 (85%) carried either an LQTS-causing genetic mutation (2 patients) or a common polymorphism called K897T (9 patients) that may have predisposed them to the torsades de pointes associated with their acquired LQTS.[2] Genetic testing of our patient's DNA revealed no known LQTS-associated mutation or polymorphism. Yet, despite the limited current knowledge of predisposing genetic factors, we think that our patient's family history of fatal cardiac disease was a factor in her case.

We quickly recognized and diagnosed our patient's acquired LQTS. Recatheterization and further amiodarone administration were unnecessary and potentially harmful, and treatment with isoproterenol and magnesium was successful. This case serves to remind clinicians of this rare, life-threatening ventricular arrhythmia after MI.

References

1. El-Sherif N, Turitto G, Boutjdir M. Acquired long QT syndrome and torsade de pointes. Pacing Clin Electrophysiol 2018;41(4):414-21.
2. Crotti L, Hu D, Barajas-Martinez H, De Ferrari GM, Oliva A, Insolia R, et al. Torsades de pointes following acute myocardial infarction: evidence for a deadly link with a common genetic variant. Heart Rhythm 2012;9(7):1104-12.
3. Ahnve S. QT interval prolongation in acute myocardial infarction. Eur Heart J 1985;6 Suppl D:85-95.
4. Halkin A, Roth A, Lurie I, Fish R, Belhassen B, Viskin S. Pause-dependent torsade de pointes following acute myocardial infarction: a variant of the acquired long QT syndrome. J Am Coll Cardiol 2001;38(4):1168-74.
5. Antzelevitch C, Sicouri S. Clinical relevance of cardiac arrhythmias generated by afterdepolarizations. Role of M cells in the generation of U waves, triggered activity and torsade de pointes. J Am Coll Cardiol 1994;23(1):259-77.
6. Rozanski GJ, Witt RC. Early afterdepolarizations and triggered activity in rabbit cardiac Purkinje fibers recovering from ischemic-like conditions. Role of acidosis. Circulation 1991;83(4):1352-60.