EP CASE REPORT

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A heartless brain

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An 81-year-old woman treated with sotalol developed torsades de pointes 2 days after her admission for right-sided hemiplegia. There was no mutation in LQT2, KCNE1, KCNE2, KCNQ1, and SCN5A. Plasma sotalol concentration was normal. This case illustrates the influence of cerebral infarction on ventricular repolarization and suggests that antiarrhythmic drugs should be withdrawn in patients hospitalized in stroke units.

Case report

A 81-year-old woman presented to our hospital for sudden onset right-sided hemiplegia. She had a one-month history of palpitations related to atrial fibrillation. An electrocardiogram (ECG) obtained 10 days prior to admission showed atrial tachycardia with a corrected QT interval (QTc) of 420 ms (*Figure 1A*). Echocardiography revealed no evidence of structural heart disease. Treatment with a vitamin K antagonist and sotalol (80 mg twice daily) were initiated. There was no family history of sudden death or long QT syndrome. Brain magnetic resonance imaging confirmed left middle cerebral artery territory infarction affecting the insular region (*Figure 1B*). On admission, the ECG showed sinus rhythm with a QTc interval of 460 ms (*Figure 1D*). On day 2, the patient suffered from syncope related to torsades de pointes (TdP) (*Figure 1C*). An ECG after the arrhythmic event demonstrated sinus

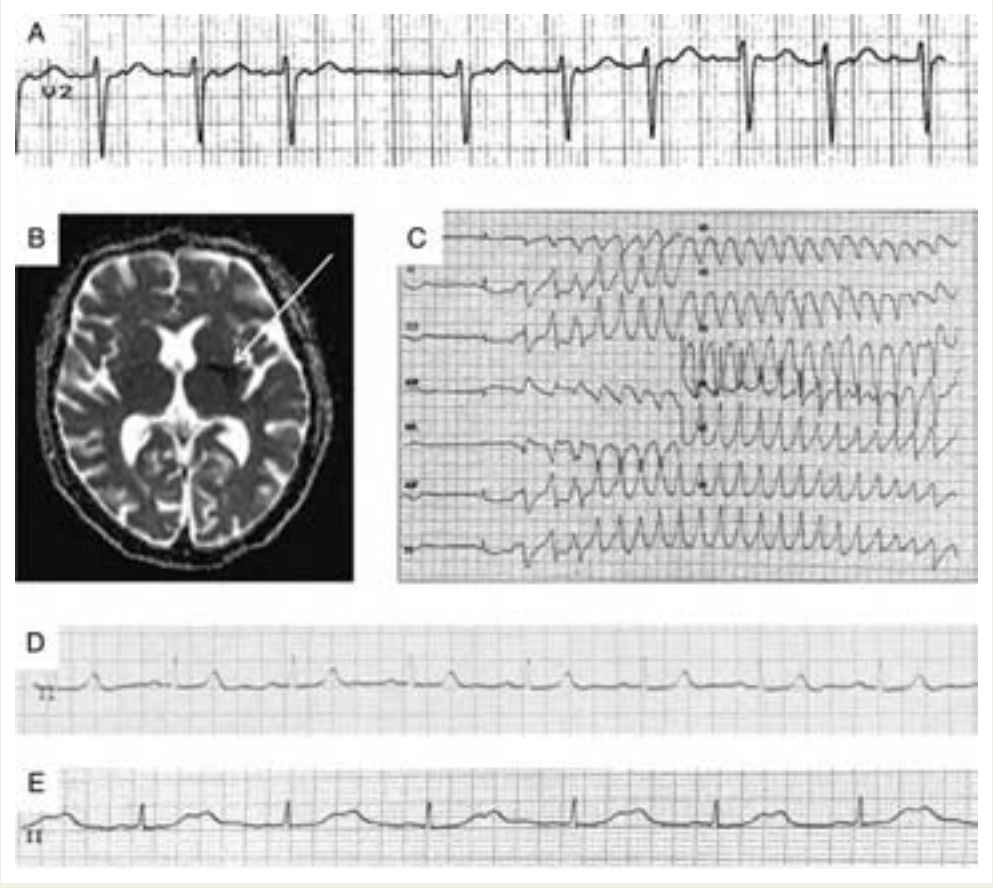

Figure 1 (*A*) Atrial tachycardia recorded 10 days before stroke occurrence. Corrected QT interval: 420 ms. (*B*) Brain magnetic resonance imaging demonstrated left middle cerebral artery territory infarction affecting the insular region (arrow). (*C*) Initiation of TdP after a short coupling PVC. (*D*) Electrocardiogram on admission. Corrected QT interval: 460 ms. (*E*) Corrected QT interval: 640 ms. There is a distorted T-U wave and a sinus bradycardia is present.

bradycardia (46 b.p.m.), QT interval prolongation to 640 ms, and a distorted T-U pattern (*Figure 1E*). Emergency management consisted of magnesium administration, discontinuation of sotalol and transfer to the intensive care unit. Biological tests and transthoracic echocardiography were normal, including plasma sotalol concentration (2.88 mg/L, reference range: 0.5–3 mg/L). Genetic testing for LQT2, KCNE1, KCNE2, KCNQ1, and SCN5A mutations were negative. On day 5, the ECG returned to normal. Sotalol was replaced by bisoprolol with a gradual dose increase under continuous ECG monitoring. At 10 months, she was doing well with no recurrence of the arrhythmia.

Discussion

It is well established that a combination of different parameters, both acquired and congenital, are required to facilitate the occurrence of long QT (LQT) syndrome.[1] Following acute ischaemic stroke, QT interval prolongation and increased QT dispersion are common ECG findings, but cases of acquired LQT syndrome complicating neurological disorders have not been documented.

QT interval prolongation and other changes in ventricular repolarization have been reported in association with ischaemic stroke and intracerebral or subarachnoid haemorrhage. It is known that the autonomic nervous system which has a critical role in cardiac electrical stability is mainly dependent on the insula area of the brain.[2] Oppenheimer[3] demonstrated that stimulation of the rat insular cortex yields to frank myocardial repolarization changes. In an animal model, middle cerebral artery ischaemic stroke was consistently associated with QT prolongation. Although cardiac arrhythmias are presumed to be the most common cause of sudden death in this setting, evidence remains weak and clinical observations are scarce.

In the present report, polymorphic ventricular tachycardia occurred 10 days after initiation of antiarrhythmic therapy with low-dose sotalol and 2 days after left middle cerebral artery territory infarction. Since the proarrhythmic potential of sotalol is dose-dependent, it is unlikely to be the sole culprit in the present case as the insular stroke may have facilitated the occurrence of TdP. This hypothesis is further strengthened by the time course of QT interval changes. This observation suggests that all potentially QT prolongation drugs may need to be discontinued in patients with acute stroke.

Conflict of interest: none declared.

References

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