

Ventricular tachycardia following ingestion of a commonly used antihistamine

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This report describes ventricular tachycardia, likely to have been torsade de pointes, following ingestion of the non-sedating antihistamine loratadine. Documentation of the arrhythmia was made possible by the automatic electrogram storage facility of an implanted defibrillator in a patient with no prior history of cardiac arrhythmia. (MJA 2002; 176: 429-430)

NON-SEDATING ANTIHISTAMINES are available as “over-the-counter” preparations and have been widely promoted in public advertisements. However, two such antihistamines, terfenadine and astemizole, have been withdrawn in Australia and overseas. Their use was associated with ventricular arrhythmias — QT-interval prolongation on the surface electrocardiogram (ECG) and polymorphic ventricular tachycardia (“torsade de pointes”).¹⁻³

We report a case of ventricular tachycardia (VT) which was probably “torsade de pointes” following ingestion of a single tablet of loratadine.

Clinical record

A 43-year-old woman presented after her identical twin died suddenly, presumably from cardiac arrhythmia. The patient was asymptomatic, had no other risk factors and was not taking any regular medication. Clinical examination showed evidence of mitral valve prolapse, with normal left ventricular function and trivial mitral regurgitation on echocardiography. Several ECGs (Box 1a) and her biochemical profile were normal. An electrophysiological study did not show inducible ventricular arrhythmias with standard stimulation testing or adrenaline provocation. Atrial fibrillation was induced during catheter introduction into the right atrium, with ventricular rate less than 130/minute. A prophylactic automatic implantable defibrillator (Medtronic Micro Jewel 7221) was subsequently inserted. Transient prolongation of the QT interval was noted on ECG monitoring in association with non-sustained VT (Box 1b) within 48 hours of the implant.

Two years later, the patient had an episode of presyncope, interrupted by a spontaneous defibrillator shock, about 90 minutes after taking a single 10 mg tablet of loratadine for minor symptoms of nasal congestion; this was the patient’s first exposure to this drug. She was reviewed on the

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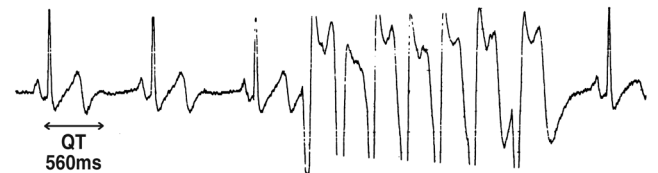
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1: Electrocardiograms from the patient before (a) and after (b) implantation of an automatic implantable defibrillator



(a) ECG strip showing sinus bradycardia (rate, 50/min) and a normal QT interval (420 ms).



(b) ECG monitor strip showing QT prolongation (560 ms) and a run of non-sustained ventricular tachycardia (arrow).

following day, when the defibrillator device history revealed a rapid ventricular rhythm with changing axis of the intracardiac ventricular electrogram, with a cycle length of 320–230 ms (average rate, 250/minute; Box 2), successfully terminated by a 31-Joule biphasic discharge. The patient was not taking other medications at the time.

Discussion

The term “torsade de pointes” describes polymorphic ventricular tachycardia where the QRS axis appears to be twisting around a baseline. It is associated with prolongation of the QT interval, usually greater than 500 ms (normal is less than 440 ms). A pattern of long-short cycle length in the beats immediately before tachycardia is typical of torsade de pointes (Box 3). It is only possible to diagnose probable “torsade de pointes” in our case, as the defibrillator placed in the patient recorded only a brief snapshot after the device had made the diagnosis of a treatable arrhythmia, so the initiating beats were not recorded. Further, the tracing that the device makes is a ventricular electrocardiogram without any atrial tracing, and the intracardiac QT is not generally accepted as a valid measure of the QT interval. However, the rate of the arrhythmia and its twisting morphology, together with the previous brief episode of prolonged QT interval, make any other diagnosis unlikely.

