A dangerous “notch”

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Case report

A 72-year-old woman was admitted to the hospital due to cardiac decompensation and pre-syncopes. She had a history of mitral valve replacement for rheumatic disease with a mechanical valve in 1995, severe left ventricular systolic dysfunction, left bundle branch block and tachycardia-bradycardia syndrome for which a dual chamber pacemaker was implanted one year ago (programmed in VVI 40 bpm). She also suffered from schizophrenia. Her treatment consisted of enalapril, atenolol, furosemide, amiodarone, acenocumarol and haloperidol. The ECG recorded on admission is illustrated in figure 1. What are the “unusual waves” in this trace (arrows)? What is the cause of the pre-syncope?

Discussion

The ECG in figure 1 shows a relatively slow sinus rhythm with left bundle branch block and premature atrial beats. There are some artifacts on V2 and V3, 

Figure 1

ECG showing a sinus bradycardia (40–60 bpm) with left bundle branch block and frequent premature atrial beats (in precordial leads). The T-waves, best seen in V4, are unusual with a double component (arrows) varying in amplitude inversely with the preceding cycle length. The abnormal T-waves result in a prolonged QT interval reaching 700 ms.

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but the “unusual waves” at the end of the T-waves (arrows), best seen in V4, are not artifacts as one could think at first glance. These waves are indeed part of the double peak T-waves, which are also called “notched T-waves” and result in a very long QT interval reaching 700 ms. Notched T-waves are clearly augmented with long R-R intervals after premature atrial beats.

Shortly after admission the patient again had a pre-syncope. The ECG strip (fig. 2) documented an episode of self-terminated torsade de pointes preceded by a long interval (1580 msec) ended with a paced beat (VVI 40 bpm). The serum potassium concentration was 3.3 mmol/l (3.5–5.0 mmol/l), the magnesium 0.60 mmol/l (0.65–1.05 mmol/l).

The pacemaker was reprogrammed in DDD 70 bpm, an infusion of magnesium sulphate was administered, and potassium was replaced. Moreover, we optimised treatment of heart failure, and withdrew haloperidol. Clinical conditions of the patient progressively improved, and heart rhythm stabilisation was achieved. 48 hours after admission the ECG (fig. 3) showed the disappearance of the notched T-waves and the shortening of the QT interval to 500 ms.

This case illustrates an example of acquired long QT syndrome complicated by torsade de pointes. Long QT interval was caused by a combination of electrolyte abnormalities (hypokalaemia and hypomagnesaemia), drugs (haloperidol and amiodarone), underlying decompensated heart failure and pauses due to relative bradycardia with premature atrial beats. As illustrated in figure 1, the T-waves are very abnormal with two components (notched T-waves, arrows). The second component represents early after-depolarisation that can lead to ventricular ectopic beats and can degenerate into torsade de pointes, the polymorphic ventricular tachycardia typically associated with long QT syndrome [1]. Tor-
The interesting ECG


The terminal component of the T-wave, represent signs of imminent danger for developing malignant ventricular arrhythmias and deserve, therefore, close rhythm monitoring [2].

References
