Resuscitation with a twist

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Answer to the rhythm puzzle

The ECG on admission to the coronary care unit showed sinus rhythm, QT prolongation and disappearance of previous ST depression. It was mentioned that the patient was under continuous cardiac monitoring during his stay on the surgical ward. Review of these tracings (Fig. 1), corresponding with extremity leads II and III, demonstrated the occurrence of torsade de pointes (TdP). At first, a typical short–long–short sequence of R-R cycles is seen, consisting of a premature ventricular extrasystole (VES), followed by a compensatory pause and another VES which, because of the underlying long QT, does not have the short coupling interval typical of idiopathic ventricular fibrillation. This initiates a polymorphic ventricular tachycardia at a rate of approximately 240 beats/min (Fig. 1, arrow), which finally degenerates into ventricular fibrillation (VF; Fig. 1, dotted arrow). After DC shock by the resuscitation team (Fig. 1, single arrowhead), there is a short period of asystole with a few non-captured pacemaker spikes (Fig. 1, striped arrows), followed by the onset of atrial fibrillation (Fig. 1, double arrowhead), resulting in haemodynamic recovery. At the time of his collapse, the patient was receiving treatment with fluconazole and haloperidol, both known to cause QT prolongation. After these medications were stopped, the

QT interval normalised and the patient made a full recovery.

The American Heart Association/American College of Cardiology Foundation (AHA/ACCF) recently published a statement to raise awareness on the prevention of in-hospital TdP [1]. We have presented a case of in-hospital VF thought to be secondary to cardiac ischaemia. In retrospect, however, it was shown that the VF was caused by drug-induced QT prolongation and TdP. TdP, literally meaning ‘twisting of the points’, is a polymorphic ventricular tachycardia, typically at a rate of 160 to 250 beats/min, occurring in the setting of QT prolongation and characterised by frequent variations of QRS morphology and amplitude around the iso-electric line. The QT prolongation can be inherited (as part of a ‘long QT syndrome’) or acquired, mostly due to certain drugs or electrolyte disturbances. Since hospitalised patients frequently combine several risk factors for the development of TdP (e.g. advanced age, diuretic treatment, hepatic dysfunction, heart disease, electrolyte abnormalities), one needs to be vigilant about the prevention of this life-threatening arrhythmia [1, 2].

References

1. Drew BJ, Ackerman MJ, Funk M, et al. Prevention of torsade de pointes in hospital settings: a scientific statement from the American Heart Association and the American College of Cardiology Foundation. *Circulation*. 2010;121(8):1047–60.
2. De Bruin ML, Langendijk PNJ, Koopmans RP, et al. In hospital cardiac arrest is associated with use of non-antiarrhythmic QTc prolonging drugs. *Br J Clin Pharmacol*. 2007;63:216–23.

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Fig. 1 Cardiac tracings whilst the patient was on the ward, corresponding with leads II and III. A short–long–short sequence of R-R cycles initiates TdP (arrow) which degenerates into VF (dotted arrow), terminated by DC shock (single arrow-head). A brief period of asystole (striped arrow) is followed by the onset of atrial fibrillation (double arrowhead), resulting in restoration of cardiac output