**Case report**

An 81-year-old patient was admitted to the coronary care unit due to unstable angina and respiratory distress after urgent eye surgery for retinal detachment. He had a medical history of hypertension, diabetes mellitus, coronary artery disease, and valvular heart disease. He underwent coronary artery bypass grafting and aortic valve replacement in 1998 and received a dual chamber pacemaker (Pulsar Max DR Guidant/Boston Scientific Natick, MA, USA) for third-degree heart block in 1999. The diagnosis of myocardial infarction complicated by acute pulmonary oedema was withheld, in view of a rise in troponin I level to a maximum of 14.66 μg/L (normal value < 0.14 μg/L). ST-segment elevation could not be assessed because of ventricular pacing (Figure 1A). He developed respiratory failure for which mechanical ventilation was initiated. His echocardiography showed a depressed left ventricular function with an ejection fraction of 30% due to a large antero-lateral myocardial infarction. An urgent coronary angiography showed a critical stenosis of the left coronary artery, and a percutaneous coronary intervention of the native left anterior descending and circumflex artery was performed.

During the night, he developed sustained runs of a regular wide QRS complex tachycardia (WCT) of 110 bpm, with a right bundle branch appearance and a left axis (Figure 1B). During these runs that could last up to 10 min, the patient was mildly haemodynamically compromised.

What is your diagnosis?

**History and 12-lead ECG**

The previous cardiac history of the patient and the setting of an acute coronary syndrome make the diagnosis of ventricular tachycardia highly likely.1 Applying the classical diagnostic criteria of WCT on the 12-lead ECG, there is a regular, monomorphic (152 ms) WCT with right bundle branch morphology, superior axis, qR in V1 lead, and rS in V6 lead. There is no concordant pattern and are no clear signs of atrioventricular (AV) dissociation, favouring the diagnosis of ventricular tachycardia. When using the Brugada criteria, there is no absence of an RS complex in the precordial leads, the ‘R-to-S interval > 100 ms’ criterion is borderline, and there are no clear signs of AV dissociation, so again the morphology criteria in V1-2 and V6 leads must be used, favouring the diagnosis of ventricular tachycardia.2

However, a newer algorithm proposed by Vereckei et al. raises the suspicion of supraventricular tachycardia: there are no clear signs of AV dissociation, no initial R wave in aVR, and there is a right bundle branch appearance. This obliges us to use the fourth criterion, the estimation of initial (vi) and terminal (vt) ventricular activation velocity ratio (vi/vt): the initial ventricular activation is the fastest in leads V3 to V5, showing a v/vt ratio > 1, favouring supraventricular tachycardia.3,4 Older ECGs all showed a paced rhythm, and there was no documentation of an underlying escape rhythm available. The cardiologist on call reprogrammed the pacemaker from DDD 60–120 bpm (AV delay 200 ms) to VVI 80 bpm to rule out pacemaker-mediated tachycardia. This however did not influence arrhythmia occurrence. Therefore, he judged it to be prudent to consider and treat the tachycardia as ventricular tachycardia, and intravenous amiodarone was started.

Pacing manoeuvres using the dual chamber pacemaker

The following day we used the pacemaker programmed to further differentiate this tachycardia. The tachycardia could be entrained both from the atrium and the ventricle. Atrial pacing at 125 bpm slowed down tachycardia, but it often resumed when pacing was stopped (Figure 2A), making a ventricular origin highly unlikely. Pacing in the ventricle at 125 bpm could stop tachycardia reliably (Figure 2B), arguing against an ectopic atrial tachycardia. Furthermore, the tachycardia could be stopped with iv administration of adenosine 6 mg (Figure 2C), suggestive of a re-entrant mechanism involving the AV node. The intracardiac EGM showed tachycardia with a short VA (40 ms) and a long AV (440 ms) interval (Figure 2D), diagnostic of AV nodal tachycardia.5 The pacemaker was reprogrammed to VVI 40 bpm to document tachycardia initiation. An underlying slow sinus rhythm with a QRS morphology similar to tachycardia was seen along with gradual AV prolongation. When a critical AV delay was reached, retrograde conduction to the atrium was seen, thus initiating tachycardia (Figure 3). This phenomenon was repeatedly seen, confirming the diagnosis of typical slow–fast AV nodal tachycardia.

Amiodarone was stopped, and beta-blocker therapy was intensified. The pacemaker was temporarily reprogrammed to DDD 80–120 bpm, with a short AV delay of 150 ms. These measures controlled the WCT, but despite maximal supportive therapy, the patient died due to septic shock because of pneumonia.

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Figure 1  (A) ECG admission and (B) ECG tachycardia.
Figure 2 (A) EGM temporary AAI 125 bpm (permanent VVI 80 bpm), (B) EGM temporary VVI 125 bpm (permanent VVI 80 bpm), (C) EGM after adenosine 6 mg iv (permanent VVI 80 bpm), and (D) EGM tachycardia (permanent VVI 80 bpm).
Conflict of interest: none declared.

References