

Wide-complex tachycardia with heart failure

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Case presentation

A 67-year-old male patient presented with dyspnoea and chest discomfort during mild physical activity. The medical history was unremarkable, apart from a tick bite five years ago.

The patient presented stable findings, but the physical examination showed signs of heart failure. Transthoracic echocardiography revealed severely depressed left ventricular function (LVEF 30%) without regional wall motion abnormalities. The 12-lead ECG is shown in figure 1.

Questions

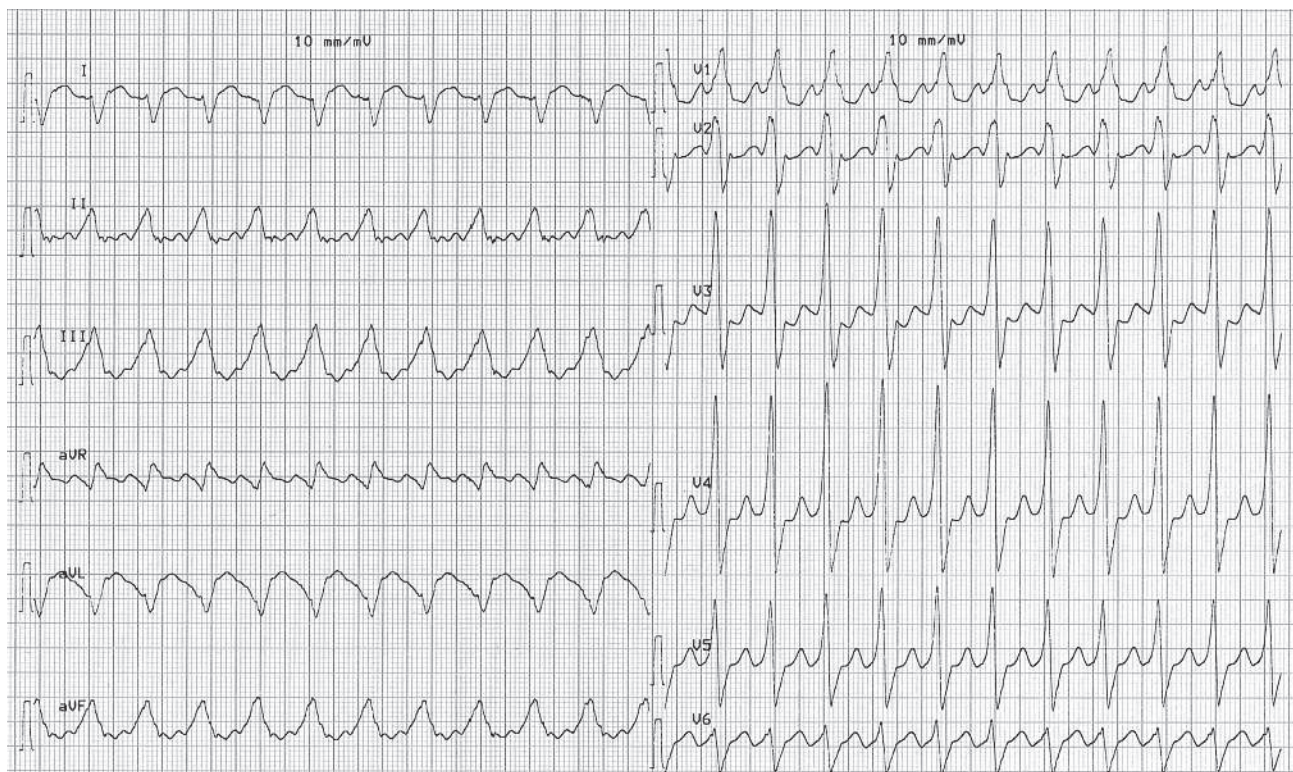
1. What is the differential diagnosis of the 12-lead ECG (fig. 1)?
2. How would you manage this patient?

Commentary

Figure 1 shows regular wide-complex tachycardia (WCT) with an indeterminate axis and a right bundle branch block pattern in the precordial leads with a ven-

Figure 1

12-lead ECG showing wide-complex tachycardia with right axis deviation and right bundle branch block morphology with a rate of 125 bpm.



Funding / potential competing interests:

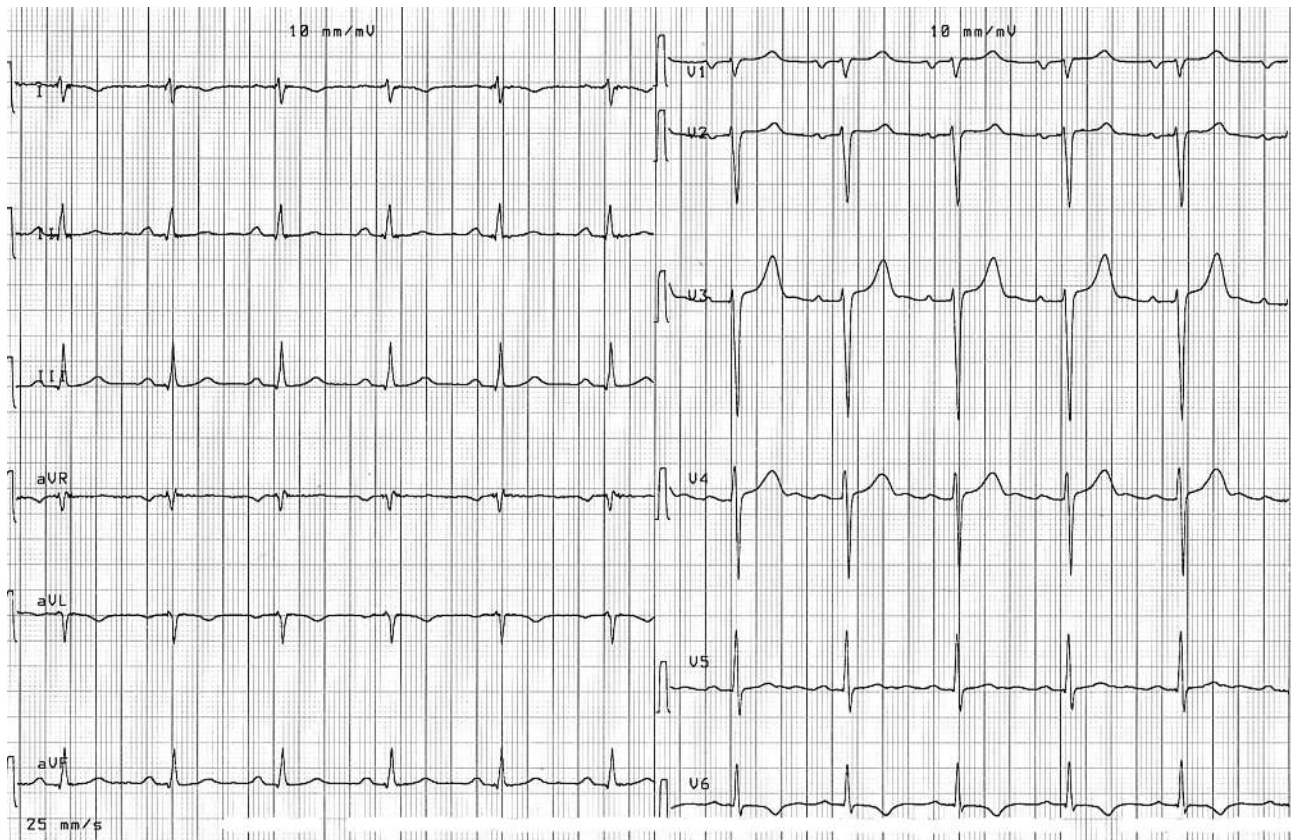
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Figure 2

12-lead ECG after cardioversion showing normal sinus rhythm with first-degree AV block.



tricular rate of 125 bpm. A widened QRS complex (≥ 120 ms) occurs when ventricular activation is slow. The arrhythmia may originate outside the specific conduction system (ventricular tachycardia [VT]), or abnormalities within the His-Purkinje system may be present (supraventricular tachycardia [SVT] with aberrancy). Much less common are SVTs with antegrade conduction over an accessory pathway (AP). In other rare cases, recognition that a wide QRS complex is caused by a pacing spike may constitute a challenge. VT accounts for up to 80% of WCTs [1, 2], and differential diagnosis is often difficult. ECG algorithms have been developed that evaluate QRS axis, duration, concordance in the precordial leads, and morphology of the QRS complex to distinguish VT from SVT with aberrancy [3]. However, all these algorithms are complex and imperfect with low specificity to rule out VT. The presence of AV dissociation in WCT strongly suggests VT, although its absence is less helpful.

In our case the QRS is approx. 200 ms. Close inspection of lead II is suggestive of P waves. There is no sign of AV dissociation. The axis and the very wide QRS are rarely seen in SVT with aberrancy. The most likely arrhythmic mechanism, therefore, is VT or SVT with antegrade conduction over an AP with direct activation of the ventricular myocardium, bypassing the

normal His-Purkinje system, which cannot be distinguished from the surface ECG.

The patient was monitored and emergent synchronised cardioversion was performed under conscious sedation, which restored sinus rhythm with first-degree AV block (fig. 2). Arrhythmia recurred a few days later and the patient elected to undergo an electrophysiological study (EPS) during tachycardia. EPS revealed common counterclockwise atrial flutter with an atrial cycle length of 240 ms and 2:1 antegrade conduction over a left-lateral AP with a surface ECG identical to the ECG in figure 1. During catheter placement the ablation catheter inadvertently entered the left atrium via a patent foramen ovale. The AP was therefore mapped and ablated. After ablation of the AP, the ECG showed the typical sawtooth flutter waves with 2:1 to 4:1 conduction over the His-Purkinje system during common atrial flutter (fig. 3). Catheter ablation of the cavotricuspid isthmus with termination of the arrhythmia and proof of bidirectional block was achieved. Ventricular stimulation showed retrograde conduction over the AV node. During follow-up, functional status improved and the patient remained symptom-free. Echocardiography a few weeks after the ablation procedure showed normal left ventricular function, suggesting tachycardia-mediated cardiomyopathy (tachy-

Figure 3

12-lead ECG during electrophysiological study. After ablation of the left-sided accessory pathway, typical flutter waves (negative in leads II, III, and aVF, positive in V₁) are clearly discernible. There is 2:1 to 4:1 conduction over the AV node, resulting in a normal QRS complex.



myopathy) as the cause of ventricular impairment and heart failure.

SVT with antegrade conduction over an AP is an uncommon cause of wide-complex tachycardia. Differentiation of this arrhythmia from VT is particularly difficult. Due to the abnormal ventricular activation in both arrhythmias, many of the ECG criteria are unhelpful. The patient showed clinical signs of heart failure, severely depressed left ventricular function and diagnosis of the WCT remained uncertain after careful evaluation of the ECG. Synchronised electrical cardioversion was therefore the therapy of choice. EPS revealed the arrhythmia mechanism. Catheter ablation of recurrent and/or poorly tolerated atrial flutter is a class I indication. Ablation of the AP in our case is debatable, since it was not part of the arrhythmia mechanism and the patient had no documented AV reentrant tachycardia. However, in the 2010 ESC guidelines on AF, catheter ablation of an overt but asymptomatic AP is recommended in patients at high risk of developing AF (i.e. patients with common atrial flutter) [4].

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