

## A reminder of the importance of the 12-lead ECG in daily practice

# A simple case of atrial fibrillation?

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### Case presentation

A 70-year-old female patient with a history of controlled arterial hypertension presented to her cardio-

logist for palpitations, chest pain and dyspnoea of progressive onset over the last few days. She had suffered intermittent palpitations for the past 4 weeks, some episodes lasting for several hours. Her cardiologist

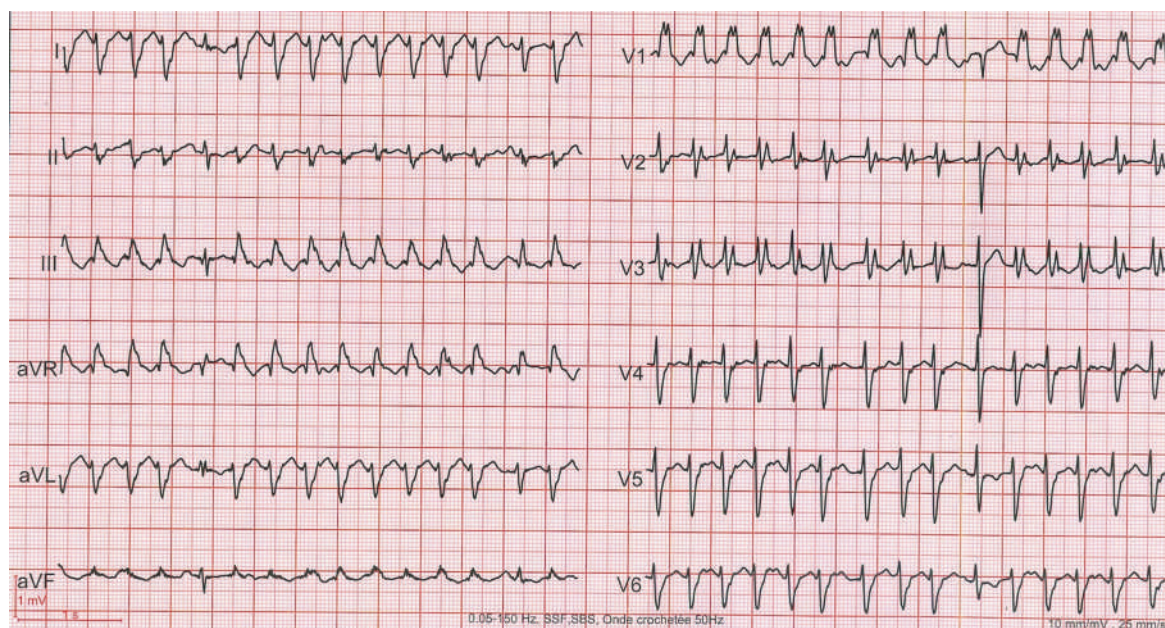


Figure 1a: Standard 12-lead ECG at admission.

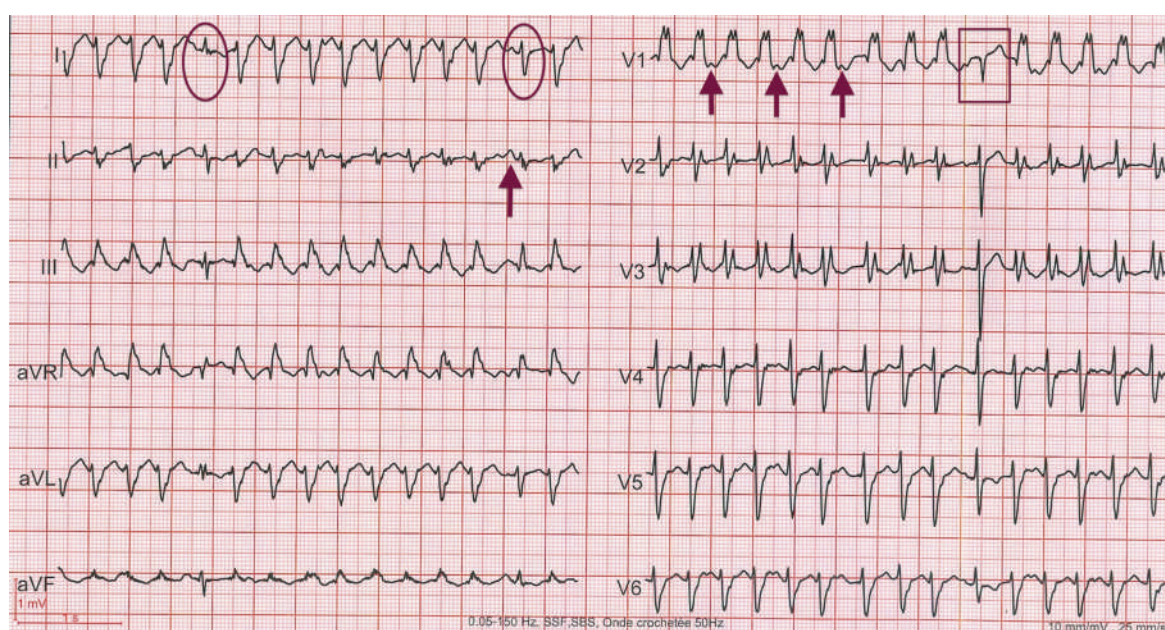


Figure 1b: Standard 12-lead ECG at admission showing irregular wide-complex tachycardia with a QRS duration of 140 ms and an atypical RBBB morphology in V1. There is a right axis deviation, atrioventricular dissociation (P waves shown with arrows), two ventricular fusion beats (shown with red circles) and one capture beat (shown with red rectangle).



diagnosed atrial fibrillation (AF) with global left ventricular (LV) dysfunction, and prescribed oral anticoagulation and metoprolol.

However, symptoms increased despite drug treatment, prompting an emergency room admission. At presentation she was tachycardic at 170 bpm, without signs of peripheral hypoperfusion; her blood pressure was normal. Lung auscultation revealed mid-lung crackles. The rest of the physical examination was unremarkable.

The initial ECG is shown in figure 1a; it is similar to the ECG performed by her physician 24 hours earlier.

### Question

Do you agree with the diagnosis of AF?

### Commentary

The ECG (fig. 1a) shows an irregular wide-complex tachycardia with a QRS duration of 140 ms and an atypical right bundle-branch block (RBBB) morphology with a qRR' in V1, rS in V6, right axis deviation and qR in lead aVR [2]. Furthermore, there are visible sinus P waves (positive in leads I and II, negative in aVR) with atrio-ventricular dissociation (fig. 1b).

Note the presence of captured beats (when an atrial impulse depolarises the ventricles through the normal conduction system, producing a narrow native QRS complex) and fusion beats [when a sinus impulse par-

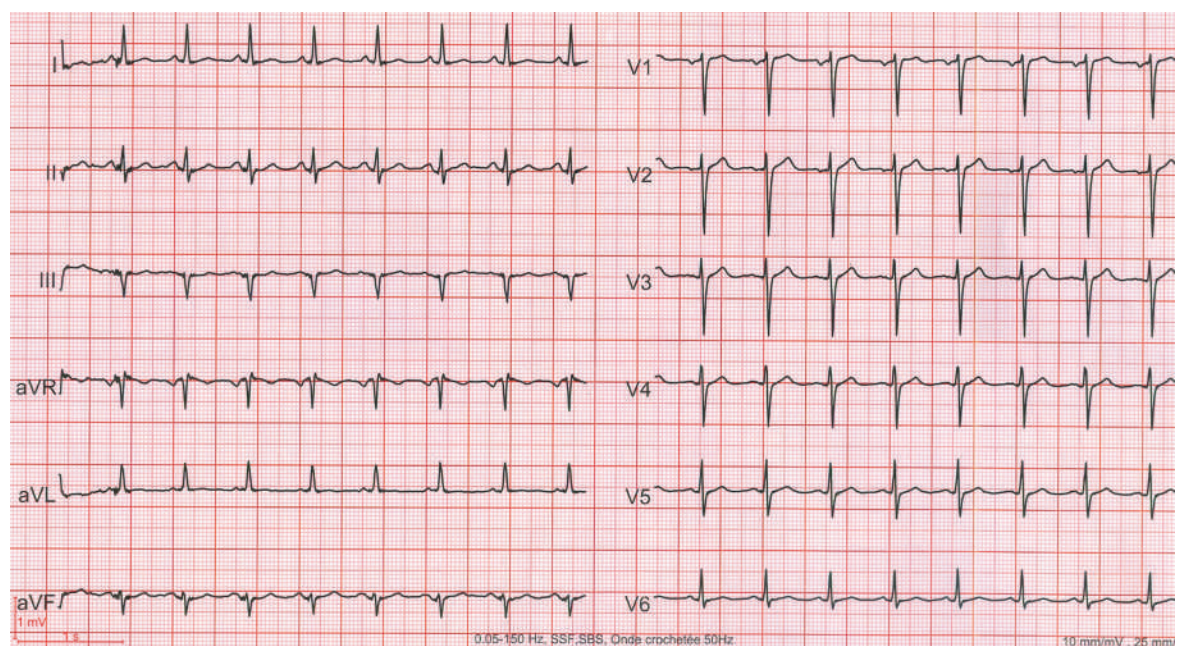
tially depolarises the ventricle and fuses with a ventricular tachycardia (VT) QRS complex producing a mixed and relatively narrow QRS] (fig. 1b).

These features are diagnostic of VT with a RBBB pattern [1, 2].

The laboratory workup was unremarkable. A transthoracic echocardiogram showed a severely globally depressed ejection fraction of 25%. A coronary angiogram was normal.

The irregularity of the arrhythmia cycle length speaks against a reentry mechanism but supports an automatic focus as the origin of the VT. In the absence of underlying disease incessant idiopathic left VT was suspected.

ECG morphology was atypical for a fascicular or an outflow tract VT and compatible with an anterolateral papillary muscle origin; however, papillary muscle arrhythmias are rarely expressed as incessant VT, but more often as ventricular premature beats [3–5]. The LV was endocardially mapped and successful ablation was performed in the antero-apical wall of the LV where early polyphasic potentials were recorded. Cardiac function improved progressively after the ablation and normalised within 3 months after the procedure. The ECG in sinus rhythm showed no major abnormality (fig. 2). During follow-up, a cardiac magnetic resonance imaging scan with intravenous gadolinium contrast was normal, allowing us to exclude myocardial scar and giving extra weight to the diagnosis of idiopathic VT.



**Figure 2:** Standard 12-lead ECG registered at discharge, showing normocardic sinus rhythm without significant abnormality.

The patient was uneventfully discharged following arrhythmia ablation. During 6 months of follow-up, Holter recordings confirmed the absence of significant ventricular arrhythmia recurrence.

This case reminds us of the importance of the 12-lead ECG in daily practice; misdiagnosis of arrhythmia can lead to unnecessary prescription of potentially dangerous medication.

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#### Disclosure statement

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