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# A nearly misdiagnosed wide complex tachycardia: all algorithms have flaws

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**Abstract.** Wide complex tachycardia is a common presentation in the Emergency Department. Although differentiating between supraventricular and ventricular tachycardia is critical for an appropriate treatment, it can still prove quite challenging. Clinicians usually make this distinction based on clinical context and several electrocardiographic criteria and algorithms. We report a case of wide complex tachycardia where these criteria were conflicting and where the correct diagnosis was only established through the careful analysis of serial electrocardiograms during the treatment of the arrhythmia. It therefore highlights the importance of performing electrocardiograms before and after treatment and of meticulously analysing them, beyond the diagnostic algorithms, before making a final diagnosis. Fig. 3, Ref. 10, on-line full text (Free, PDF) www.cardiologyletters.sk

**Keywords:** atrial flutter – ECG – supraventricular tachycardia – ventricular tachycardia

Wide QRS complex tachycardia (WCT) is a common presentation in the Emergency Department (ED) but can still prove to be a diagnostic challenge even to the cardiologist. Nevertheless, differentiating between supraventricular and ventricular tachycardia (VT) is extremely important since it has significant therapeutic and prognostic implications.

## Case report

We present the case of a 55-year-old man referred to a Cardiology consultant from the ED for VT. He had presented with an inaugural episode of palpitations and light-headedness. He denied syncope, chest pain or dyspnea but mentioned work-related stress and increased coffee consumption that day. There was no relevant personal or family history and he was under no medication. Blood pressure was normal (108/63 mmHg), he was apyretic and physical examination was unremarkable except for the presence of tachycardia. 12-lead electrocardiogram (ECG) showed a wide QRS – 140 ms – regular tachycardia with a ventricular rate of approximately 250 beats per minute (bpm) and right bundle branch block (RBBB) morphology with extreme axis deviation and no clear atrioventricular (AV) dissociation (**Figure 1**). Since the tachycardia was hemodynamically well tolerated, the physician in the ED tried vagal maneuvers and administration of adenosine (6+12+18 mg) followed by rapid saline flush, with no response, including no change

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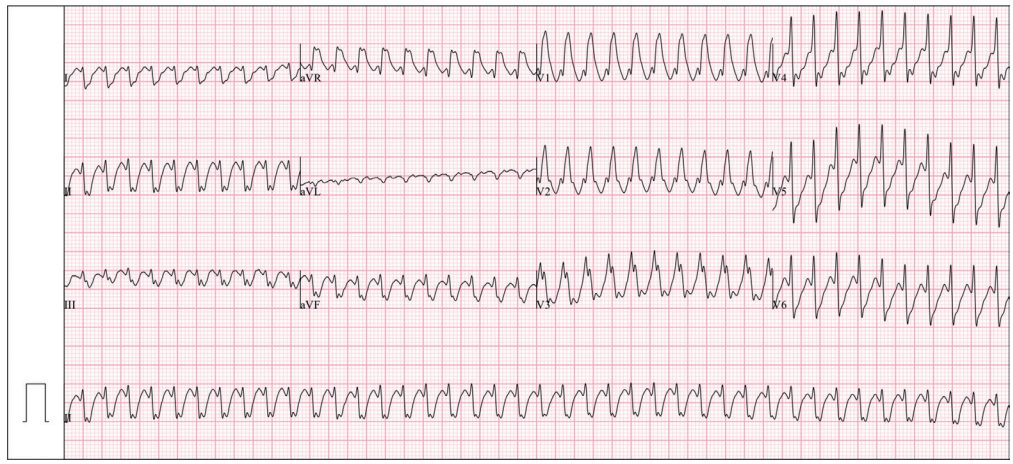


**Figure 1 Admission ECG: wide QRS regular tachycardia with right bundle branch block morphology, at 250 bpm**

in ventricular rate. Blood tests were unremarkable, excluding acute coronary syndrome, electrolyte disturbances or thyroid dysfunction. A cardiologist assessed the patient and performed a transthoracic echocardiogram, which showed a dilated and diffusely hypokinetic left ventricle (LV) with ejection fraction (LVEF) of 30% and a dilated left atrium.

Based on the context of LV dysfunction, the unresponsiveness to adenosine and several ECG criteria, the diagnosis of VT was assumed. As the tachycardia was hemodynamically stable, a perfusion of amiodarone was initiated and, after 30 minutes, the tachycardia displayed in the cardiac monitor changed to a narrow QRS tachycardia at exactly half the rate of the previous one, and the patient became asymptomatic. An ECG was performed and revealed an atrial flutter with an atrial rate of 250 bpm and ventricular rate of 125 bpm with narrow QRS (**Figure 2**). Two hours later, he converted to sinus rhythm, with normal AV and intraventricular conduction and no evidence of pre-excitation.

Comparing the ECGs, we noticed that the WCT had exactly twice the ventricular rate of the atrial flutter and, when reviewing the first ECG, we observed the presence of flutter waves at a rate of 250 bpm without AV dissociation (**Figure 3**), which established the diagnosis of atrial flutter with 1:1 AV conduction.

An electrophysiologic study (EPS) was then conducted and no sustained ventricular arrhythmias were

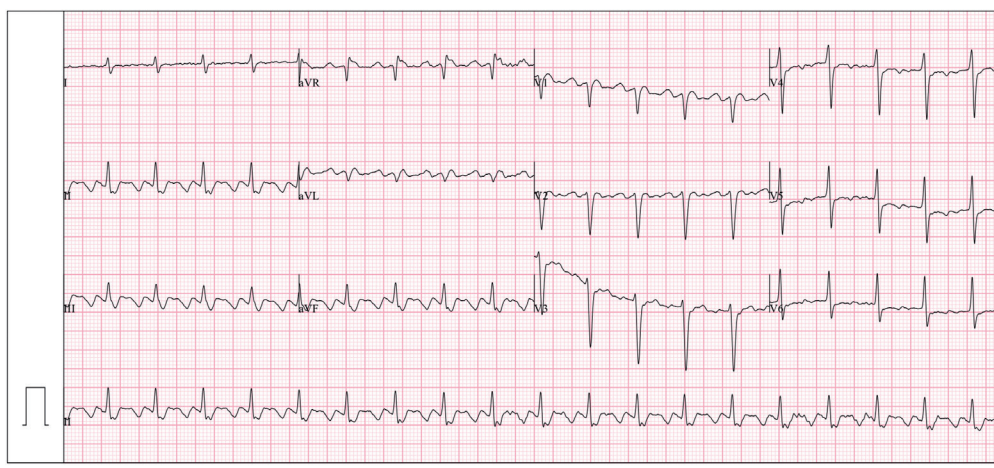


**Figure 2 ECG after 300mg of amiodarone: typical counterclockwise atrial flutter with 2:1 conduction, at 125 bpm**

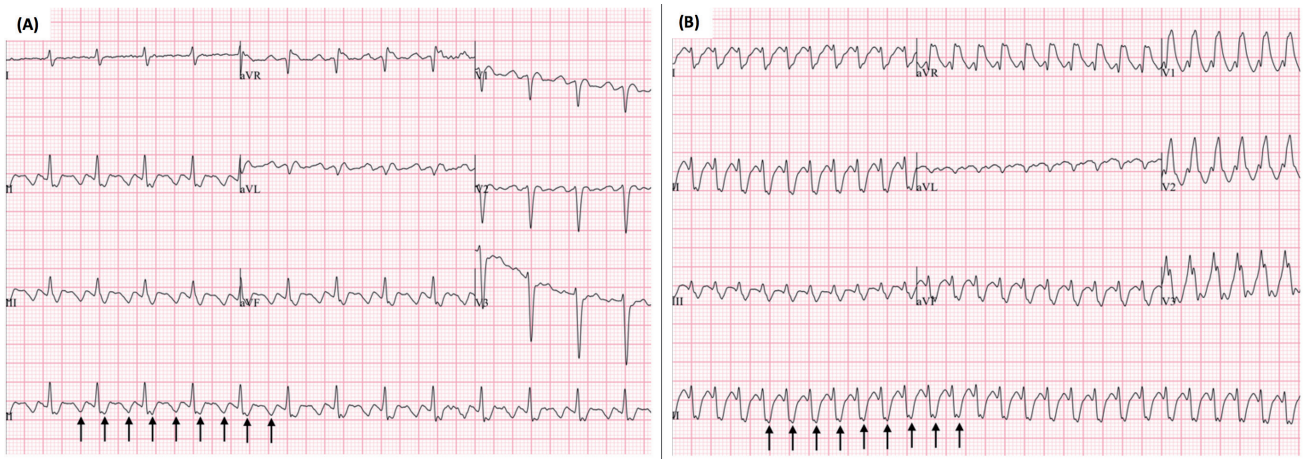


**Figure 3 Flutter waves (black arrows) after 300mg of amiodarone (A) and in the admission ECG (B)**

induced nor was there any evidence of an accessory pathway. A typical atrial flutter was elicited, and the patient underwent a successful cavotricuspid isthmus ablation.

Cardiac MRI was performed, confirmed the presence of LV dilation and severe biventricular systolic dysfunction and excluded fibrosis. Coronary angiography was also performed and excluded coronary disease. The systolic dysfunction was assumed to be secondary to tachycardia--induced cardiomyopathy and the patient was discharged on direct oral anticoagulants, sacubitril-valsartan, beta-blockers and spironolactone. Echocardiographic reassessment three months after ablation demonstrated a recovered biventricular systolic function.

## Discussion

WCT can be challenging to diagnose. Its differential diagnosis includes VT – estimated to represent around 80% of the cases – and supraventricular tachycardia (SVT) with anomalous conduction (either secondary to bundle branch block or other intraventricular conduction disturbances or to conduction over an accessory pathway). When structural disease is present, VT becomes even more likely (1, 2).

In this case, the ECG showed a WCT that was initially interpreted as VT in the ED. Its conversion to atrial flutter (before sinus rhythm) under amiodarone later led to a differential diagnosis between VT in a patient with atrial flutter as baseline rhythm or atrial flutter with 1:1 AV conduction and aberrancy at presentation.

The patient's age and the LV dilation and dysfunction increased the likelihood of VT. Many of the electrocardiographic features also suggested this diagnosis, such as the extreme axis deviation and the RBBB morphology with R/S ratio < 1 in V6 (3). In fact, applying the limb lead (4) and D12V16 algorithms (5), this tachycardia would also be diagnosed as VT (negative QRS in DI, DII and DIII, and R/S ratio < 1 in DI, DII and V6, respectively). Given its axis and RBBB morphology, VT originating in the LV or posterior fascicular tachycardia would be most likely. On the contrary, the absence of visible AV dissociation and of chest lead QRS concordance, the borderline QRS duration and the rapid initial QRS deflections (including R-wave peak time < 50 ms in DII) (6) were in favour of SVT (particularly atrial flutter), which was also supported by Brugada (7) and Vereckei aVR (8) algorithms.

Vagal maneuvers and adenosine's AV blocking action would have been expected to slow atrial flutter and reveal flutter waves. However, in our patient's case there had been no change in ventricular rate despite a bolus of 18 mg of adenosine, which further pointed towards a diagnosis of VT.

In the face of a WCT with discordant ECG criteria for VT *vs* SVT, unresponsive to adenosine, in a patient with LV dysfunction, VT became the most likely diagnosis. It was only the detailed comparison of the admission ECG with the intermediate one (showing the 2:1 atrial flutter) that revealed the presence of flutter waves without AV

dissociation and allowed us to diagnose the WCT as an atrial flutter.

Complete unresponsiveness of the AV node to bolus of 18 mg of adenosine is rare (a 97% efficacy rate in terminating SVTs is reported for bolus of 12+18 mg) (9). Anomalous conduction of this flutter over an accessory pathway could have explained the lack of response to adenosine (as well as the wide QRS and extreme axis) but was excluded in the EPS. Therefore, the final diagnosis was atrial flutter with 1:1 AV conduction and rate-related aberrancy. Our patient's case is a rare example of how an AV node with apparently normal baseline conduction can have increased capacity for rapid conduction, spontaneously allowing 1:1 conduction at 250 bpm. We speculate that emotional stress, by increasing sympathetic activity, might have facilitated AV conduction and allowed AV 1:1 conduction at such high rates. Additionally, the caffeine the patient had ingested might have reduced adenosine efficacy due to blockage of adenosine receptors (10).

Since the patient was asymptomatic after conversion to atrial flutter at 125 bpm, we believe that he might have previously been in atrial flutter with 2:1 conduction for a prolonged (and asymptomatic) period, leading to tachycardia-induced cardiomyopathy; and it was only when AV conduction became 1:1, increasing ventricular rate to 250 bpm, that he developed palpitations and light-headedness.

## Conclusion

This case reminds us that, even with LV dysfunction, not all WCTs are VT. Sometimes, LV dilation and dysfunction are not the origin of the tachycardia but rather its consequence. It also shows that ECG diagnostic algorithms have flaws, and that a meticulous analysis of the ECG before and after treatment is essential since subtle features can be clues to the right diagnosis that would otherwise be missed. Finally, while assuming VT as the working diagnosis when in doubt is a valid (and the safest) approach in the emergency context, in these cases an EPS should be performed afterwards, before establishing a final diagnosis.

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