

A tale of two tachycardias

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ABSTRACT A patient with non-ischaemic cardiomyopathy, and pre-existing atypical atrial flutter and left bundle branch block, developed broad complex tachycardia. In this unique and uncommon case of double tachycardia, we discuss the diagnostic approach of ventricular tachycardia in patients with broad complex tachycardia, and the use of different contemporary algorithms to help diagnose ventricular tachycardia and differentiate it from supraventricular tachycardia with aberrant conduction.

Keywords: atrial flutter, atypical atrial flutter, electrocardiography, ventricular tachycardia

INTRODUCTION

Accurate electrocardiographic diagnosis of broad complex tachycardia is important for both treatment during acute presentation and long-term treatment, and prognostication. Over the years, various algorithms have been proposed to help differentiate ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrant conduction. Although individual algorithms are not perfect and risk oversimplifying the clinical condition, it is important that every physician has an initial strategy when approaching a case of broad complex tachycardia. We herein report the case of a patient with non-ischaemic cardiomyopathy, and pre-existing atypical atrial flutter and left bundle branch block (LBBB), who developed broad complex tachycardia. We also discuss the diagnostic approach of VT in such patients.

CASE REPORT

A 58-year-old man presented with shortness of breath and broad complex tachycardia. He had a significant history of diabetes mellitus, hypertension, hyperlipidaemia, atrial flutter and non-ischaemic cardiomyopathy, with an ejection fraction of 22%. He had previously refused implantation of a cardioverter defibrillator.

A 12-lead electrocardiography (ECG) performed during tachycardia demonstrated regular broad complex tachycardia with flutter waves marching across the screen, a QRS complex duration of 172 ms, and a right bundle branch block (RBBB)-like morphology of 167 beats/min (Fig. 1). In contrast, the baseline 12-lead ECG showed an irregular rhythm, with a QRS complex duration of 128 ms and typical LBBB morphology (Fig. 2). This development of wider contralateral morphology of the QRS complex, which differed from the baseline LBBB, is suggestive of VT. The presence of fusion beat, best seen on the 7th QRS complex on the rhythm strip, further supported the diagnosis of VT. The morphology of the QRS complex with a monophasic R wave, which is not typical in RBBB, also suggested the presence of VT. The S wave in the V6 and inferior

limb leads, and a dominant R wave in lead I localised the VT to the inferior apical region of the left ventricle. Alternatively, by just restricting the analysis to the aVR, the diagnosis of VT can also be reached rapidly in the presence of a dominant R wave without complicated morphological analysis of the QRS complexes.

Intermittently, atypical atrial flutter waves with pointed appearances at cycle lengths of 160 ms were seen indenting the diastolic intervals between the regular RR intervals in V1 (Fig. 1). These flutter waves bore no fixed relation to the ensuing QRS complex, sometimes even indenting the QRS complex, accounting for the slight variation in the QRS morphology in lead V1 and atrioventricular dissociation, which was most evident in lead V1. In contrast, the RR intervals (Fig. 2) were irregular, reflecting a variable ventricular response probably due to rapid bombardment of the atrioventricular node (AVN) by SVT with resultant physiological AVN response and variable conduction. The nature of SVT was best seen at intervals bracketed by longer RR intervals, such as those between the 5th and 6th QRS complexes in lead V1; sharp flutter-like waves were seen occupying this diastolic interval. These flutter waves were similar in morphology to those in Fig. 1 and occurred at the same cycle length (i.e. 160 ms). Fig. 2 shows atypical atrial flutter with rapid and variable atrioventricular conduction, while Fig. 1 shows the presence of dual tachycardia with VT and atypical atrial flutter occurring simultaneously and spontaneously.

DISCUSSION

Despite the presence of a wide variety of algorithms for differentiating broad complex tachycardia, the ability to make a rapid and accurate diagnosis of the condition remains a clinical challenge. Previously, Isenhour et al⁽¹⁾ and Lau et al⁽²⁾ separately demonstrated that real-life application of the Brugada algorithm did not achieve sensitivity and specificity values as high as that reported by the authors of the algorithm.⁽³⁾ Miller et al recently re-evaluated some of the ECG differentiating criteria and found

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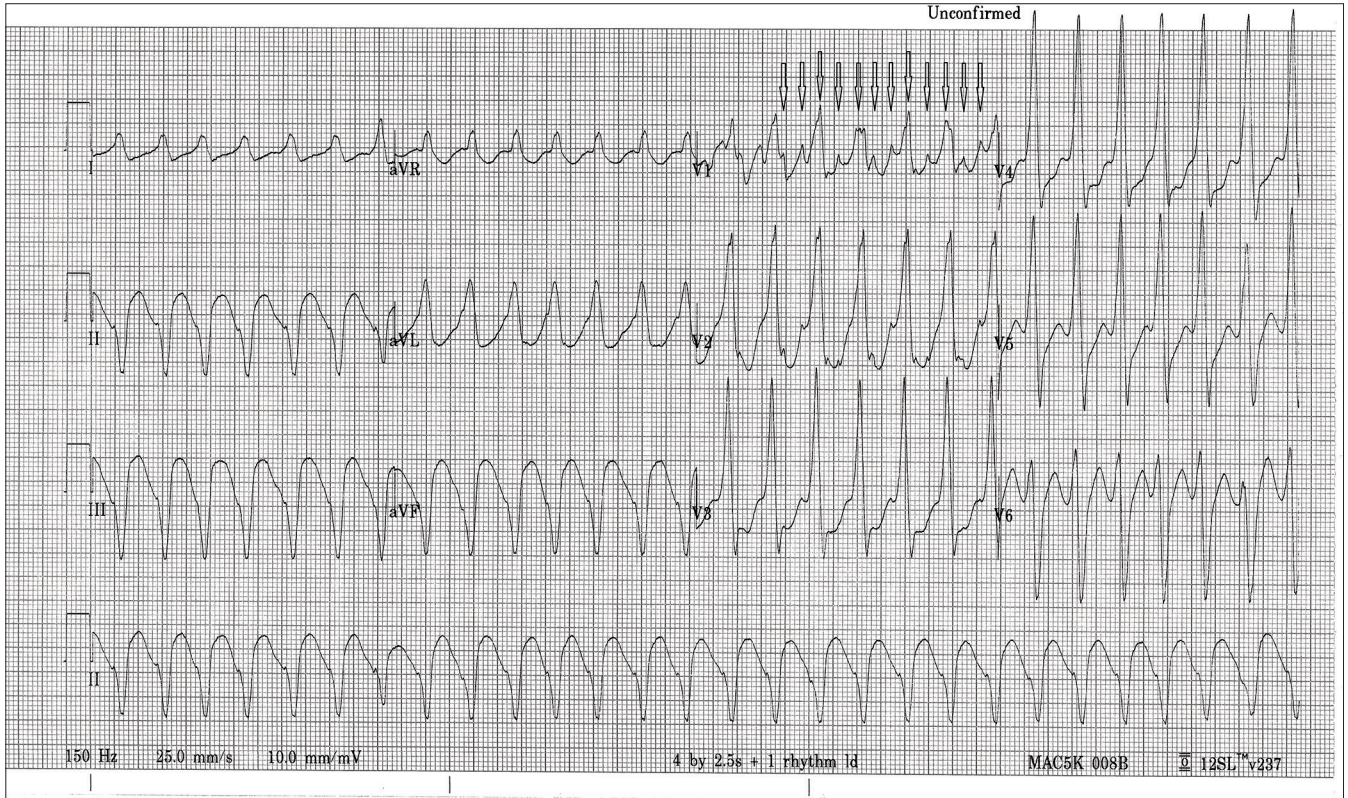


Fig. 1 12-lead ECG shows regular broad complex tachycardia of atypical right bundle branch block morphology, with underlying atypical atrial flutter waves (arrows) and no fixed relationship to QRS complexes.

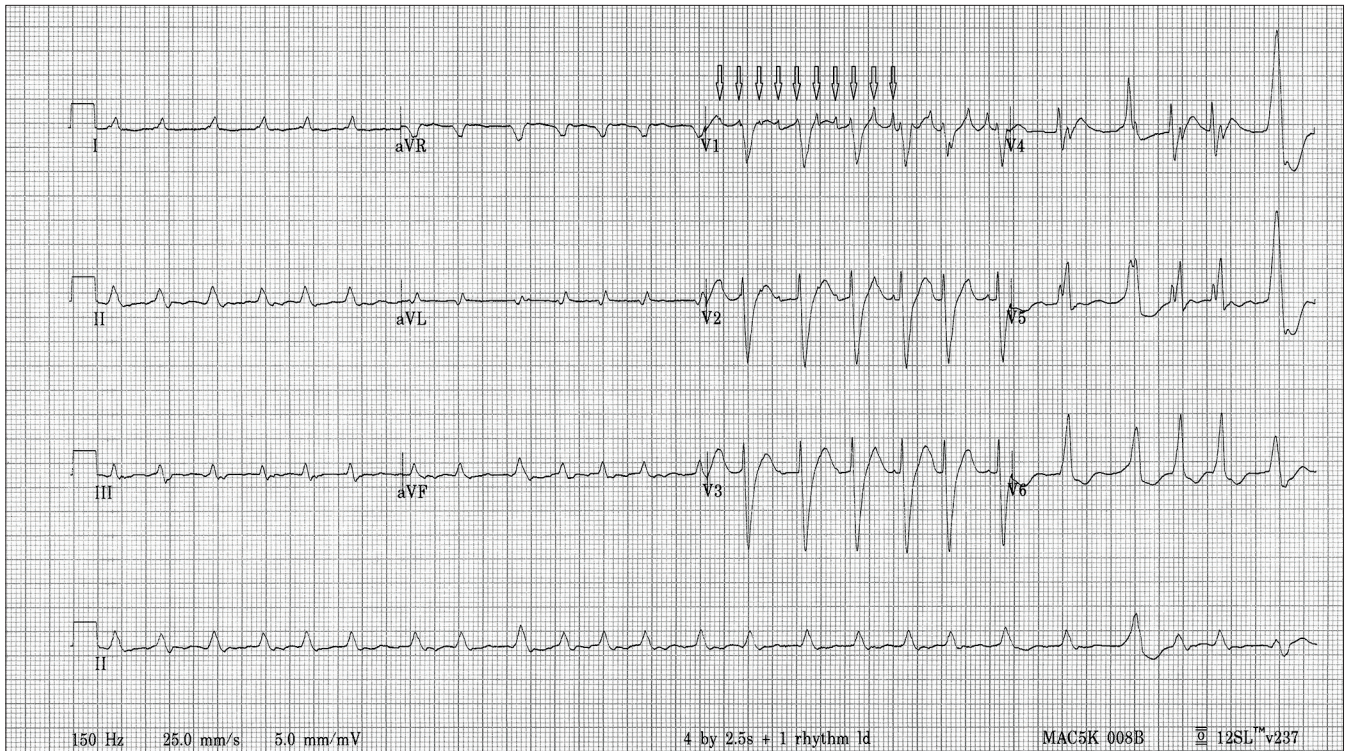


Fig. 2 Baseline 12-lead ECG shows irregularly irregular RR intervals with typical left bundle branch block morphology and atypical flutter waves (arrows) with no fixed relationship to QRS complexes.

no statistical difference in using this criteria or the criteria of negative concordance to differentiate between patients with VT and those with SVT.⁽⁴⁾ It has been postulated that since patients with heart failure live longer, they may be more prone

to experience atrial fibrillation.⁽⁵⁾ The use of classes IA and IC antiarrhythmic agents for the rhythm control of paroxysmal episodes may account for progressive widening of the QRS complex.⁽⁶⁾ When SVT occurs in patients who are using these

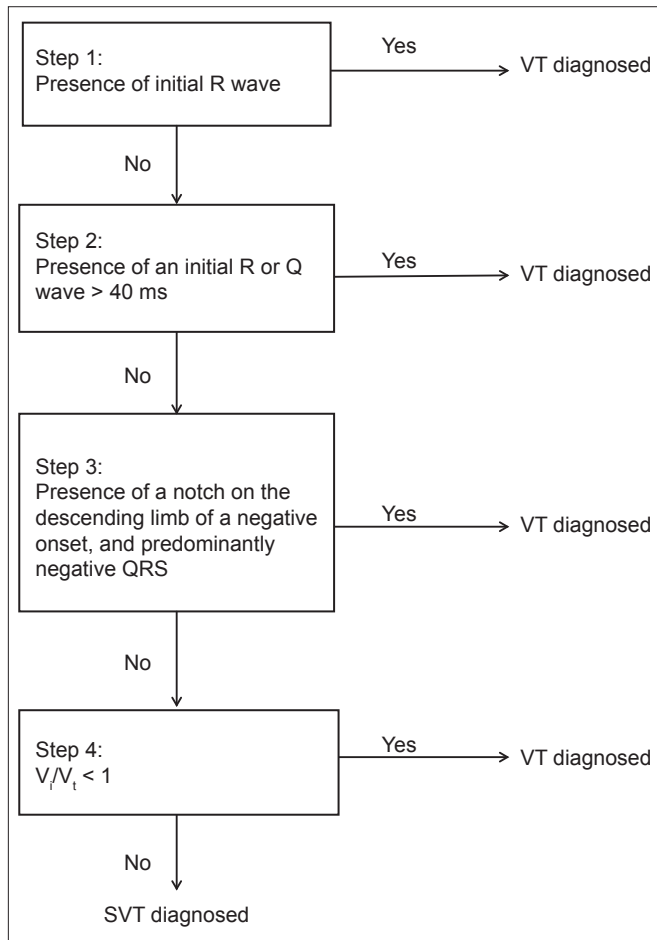


Fig. 3 The aVR criteria⁽⁷⁾ used for the diagnosis of ventricular tachycardia. SVT: supraventricular tachycardia; VT: ventricular tachycardia

antiarrhythmic agents, the rhythm may be conducted with a bizarre pattern that is similar to the baseline ECG, but may be erroneously labelled as VT in the absence of a baseline ECG for comparison.

A more robust criteria that has been tested in recent times is the aVR criteria developed by Verecke et al (Fig. 3).⁽⁷⁾ This algorithm is simplified as it restricts the analysis to just one lead and omits the morphological analysis in the precordial leads. Verecke et al's study included a significant number of patients with dilated cardiomyopathy and the algorithm proved to have greater sensitivity and negative predictive values for diagnosing VT, and greater specificity and positive predictive values for diagnosing SVT compared with the Brugada algorithm (Fig. 4).^(3,4,7)

In Verecke et al's algorithm,⁽⁷⁾ diagnosis of VT is made when any one of criteria 1 to 3 is present. In criterion 1, VT is diagnosed in the presence of initial R wave in lead aVR, as was found in our patient. If an initial R wave is absent, criterion 2 is invoked; it looks for the presence of initial R or Q wave > 40 ms. If this is absent, then the presence of notching on the initial downstroke of predominantly negative QRS complex in aVR is looked for (i.e. criterion 3). If all three criteria are absent, the initial (V_i) and terminal (V_t) ventricular activation velocity ratio (V_i/V_t) is calculated. This is done by measuring (in mV) the vertical excursion during V_i and V_t 40 ms of QRS complex. The

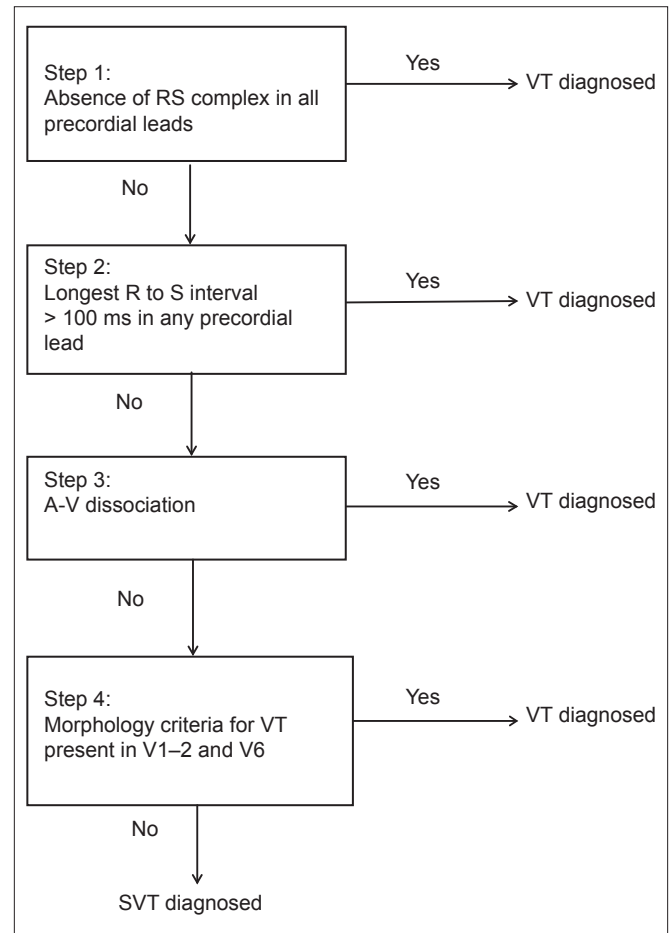


Fig. 4 The Brugada algorithm⁽³⁾ used for the diagnosis of ventricular tachycardia. SVT: supraventricular tachycardia; VT: ventricular tachycardia

sum of their absolute values (regardless of polarity) is used for the values of V_i and V_t .

The Brugada algorithm also comprises four steps (Fig. 4).⁽³⁾ The first step is to look for the presence of concordance in the precordial leads, which was lacking in our case (Fig. 1). In the second step, if the duration from onset of R wave to nadir of S wave is > 100 ms and R wave is wider than S wave in at least one precordial lead, the diagnosis of VT is made. In our case, the RS duration is < 100 ms in the precordial leads. Hence, we were unable to use the second step to diagnose VT. In the third step, the presence of atrioventricular dissociation allows the diagnosis of VT to be made; our case showed double tachycardia of both VT and atrial flutter, with rapid variable atrioventricular conduction. In the fourth step, the diagnosis of VT is made based on the morphological analysis of the patient. The presence of dominant R wave in lead V1, leading to a monophasic appearance of the QRS complex in V1 and R/S ratio < 1 in lead V6 suggests VT. The American College of Cardiology had also published its own VT criteria.⁽⁸⁾ However, the criteria would not have been useful in our patient because of his concomitant atrial flutter. A schema of the criteria is seen in Fig. 5.

In summary, we presented a case of atrial flutter and VT arising spontaneously and simultaneously in a patient with non-ischaemic cardiomyopathy.

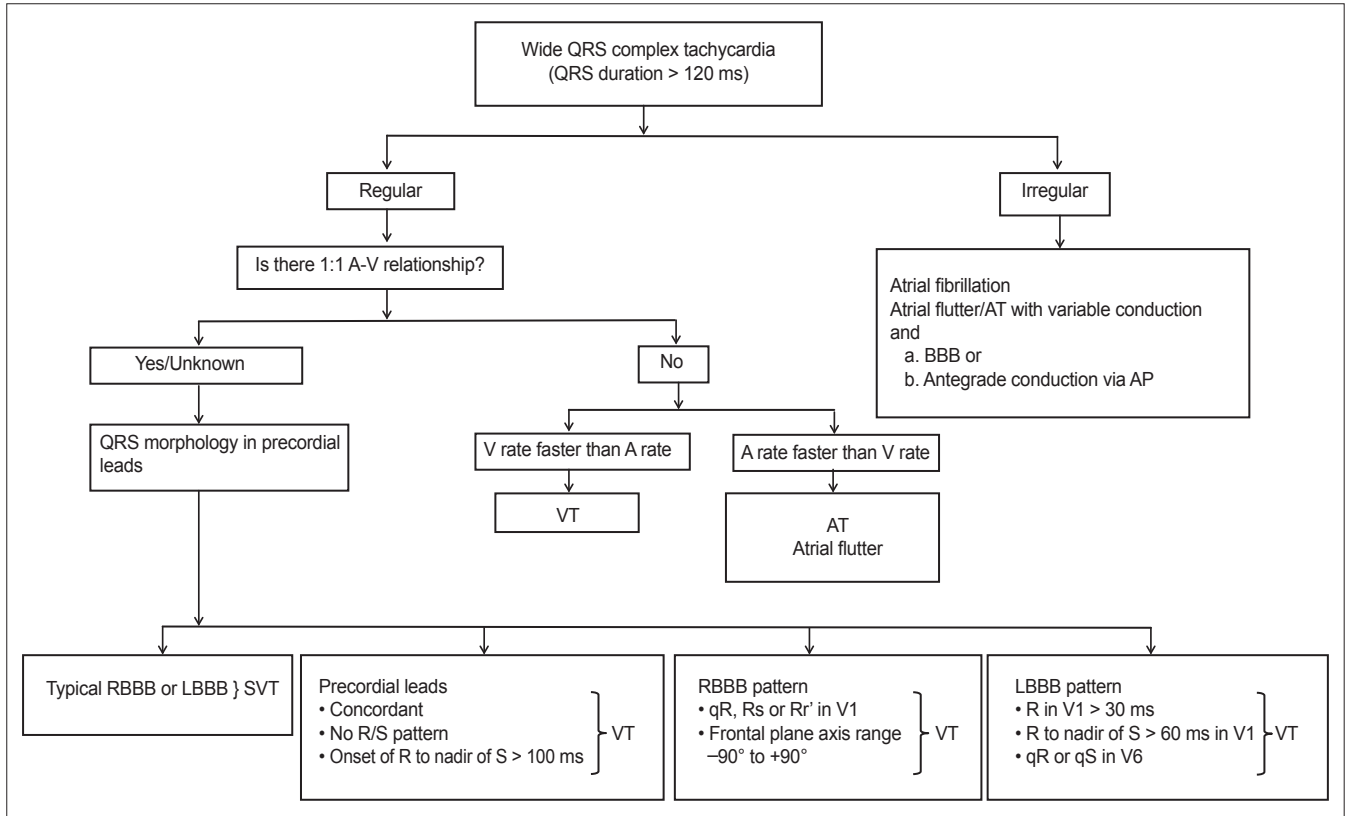


Fig. 5 The ventricular tachycardia criteria published by the American College of Cardiology.⁽⁶⁾ AP: accessory pathway; AT: atrial tachycardia; BBB: bundle branch block; LBBB: left bundle branch block; RBBB: right bundle branch block; SVT: supraventricular tachycardia; VT: ventricular tachycardia

REFERENCES

1. Isenhour JL, Craig S, Gibbs M, et al. Wide-complex tachycardia: continued evaluation of diagnostic criteria. *Acad Emerg Med* 2000; 7:769-73.
2. Lau EW, Ng GA. Comparison of the performance of three diagnostic algorithms for regular broad complex tachycardia in practical application. *Pacing Clin Electrophysiol* 2002; 25:822-7.
3. Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation* 1991; 83:1649-59.
4. Miller JM, Das MK, Yadav AV, et al. Value of the 12-lead ECG in wide QRS tachycardia. *Cardiol Clin* 2006; 24:439-51, ix-x.
5. Anter E, Jessup M, Callans D. Atrial Fibrillation and Heart Failure. *Circulation* 2009;119:2516-2525.
6. Bhardwaj B, Lazzara R, Stavrakis S. Wide Complex Tachycardia in the Presence of Class I Antiarrhythmic Agents: A Diagnostic Challenge. *Ann Noninvasive Electrocardiol* 2014;19:289-292.
7. Verecke A, Duray G, Szenasi G, Altemose GT, Miller JM. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. *Heart Rhythm* 2008; 5:89-98.
8. Blomström-Lundqvist C, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias—executive summary. a report of the American college of cardiology/American heart association task force on practice guidelines and the European society of cardiology committee for practice guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol* 2003; 42:1493-531.