A misleading wide complex tachycardia with unusual features after carotid sinus pressure: what is the diagnosis?

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Electrocardiographic differential diagnosis of a wide complex tachycardia (WCT) is challenging. During the last years different algorithms have tried to overcome these difficulties. The present article presents a case of a WCT in which traditional algorithms fail to give a definitive diagnosis that can be facilitated by a simple manoeuvre: carotid sinus pressure (CSP). An unusual response of the tachycardia after CSP is also discussed.

Case presentation

A 54-year-old man presented to the emergency room (ER) complaining of palpitations. He had history of hypertension, hypercholesterolaemia, diabetes mellitus, and a previous episode of paroxysmal atrial flutter that had been treated with electrical cardioversion 1 year before. For that reason he was taking 100 mg per day of oral flecainide. The physical examination and laboratory workup were unremarkable except for the presence of tachycardia. Initial 12-lead ECG obtained at the ER showed a regular wide complex tachycardia (WCT) at 190 bpm (Figure 1). What is the diagnosis based on the 12-lead ECG features? Figure 2 shows the response to carotid sinus pressure (CSP). What is the diagnosis?

Discussion

Figure 1 shows a WCT at 190 bpm with left bundle branch morphology and right superior axis. Possible differential diagnoses include: ventricular tachycardia (VT); supraventricular tachycardia (SVT) conducted with aberrancy; and pre-excited tachycardia. Although apparent negative concordance pattern in precordial leads seems to be present, close inspection to the tracing drawing a vertical line from the onset of the QS complex in V1 reveals that an initial r wave is present in V6. However, the QRS width (≈190 ms), the QRS axis in the frontal plane and an R to S interval >100 ms in precordial leads argue against the diagnosis of SVT with aberrancy.1–2 The ECG pattern is also inconsistent with conduction through an accessory pathway (left bundle branch morphology with negative QRS in leads I, aVL, and activation from apex to base in precordial leads), rendering VT as the most plausible diagnosis based on ECG criteria. Moreover, the presence of a monophasic R wave in aVR also favours VT according to the new algorithm recently described by Vereckei et al.3 No clear atrio-ventricular (AV)-dissociation and no capture/fusion beats can be seen in the tracing. The presence of multiple cardiovascular risk factors also favours VT although no previous history of myocardial infarction and no Q complexes can be identified during the WCT.

At the time of evaluation in the ER, the tachycardia was haemodynamically well tolerated (BP 110/85 mmHg). Transthoracic echocardiography showed no structural heart disease. Results of CSP can be seen on Figure 2. During CSP the ventricular rate changes from 190 to 105 bpm and the QRS complexes become narrow. A continuous fluctuation of the baseline can be identified as the atrial rhythm at ~210 bpm. This response to CSP favours SVT although some VTs can also be terminated by vagal manoeuvres. The morphology of the atrial activity with a continuous oscillation of the baseline suggest the presence of atrial flutter as the underlying arrhythmia mechanism with 1:1 conduction to the ventricles and left bundle branch abnormality (LBBB) aberrancy during WCT, and with 2:1 conduction to the ventricles after CSP during narrow QRS tachycardia.

Close inspection of the tracing shows that the wide QRS complex immediately posterior to the arrow pointing the onset of CSP in Figure 2 is followed by a longer RR interval ended by a narrower QRS complex. The later is followed by a shorter RR interval ended by a wide QRS complex similar to those observed during the WCT. This same sequence is repeated once again until the QRS complexes finally become persistently narrow. These observations could represent the transition from a SVT with 1:1 AV conduction to a 3:2 and, finally, a 2:1 AV conduction. Alternation of wide and narrow QRS complexes depending on the preceding RR intervals could be explained by the Ashman phenomenon. However, the hypothetical possibility of a double tachycardia, that is, VT coexisting with atrial flutter during the WCT and termination of the VT with CSP, although unusual in the absence of structural heart disease, cannot be completely excluded.

Of note, there is an increase in the atrial rate after CSP (from 190 bpm during WCT to 210 bpm during narrow complex tachycardia). This could be attributed to the cholinergic effects of this manoeuvre on the atrial myocardium. Cholinergic stimulation is
**Figure 1** Twelve-lead ECG of the wide complex tachycardia registered at the emergency room.

**Figure 2** Twelve-lead ECG showing the effect of carotid sinus pressure.
known to induce a shortening of the atrial refractory period without affecting atrial conduction velocity. The acceleration in the rate of the tachycardia observed in this case could be hypothetically explained by a tachycardia circuit with a wavelength almost equal to the relative refractory period of one of its components. In this scenario the shortening of the refractory period would reduce the wavelength of the circuit and the tachycardia will then be accelerated. However, the effect of CSP would be expected to be short-lasting and in this case it persisted overtime in the following ECG (Figure 3). An alternative hypothesis could be that the initial fast WCT induced a sympathetic discharge which accelerated the atrial flutter rate favouring the transition from 1:1 to 3:2 and 2:1 AV ratio. In that case the application of the CSP was by mere chance ‘coincidental’ with the aforementioned electrocardiographic changes.

This case highlights the limitations of surface 12-lead-ECG in the diagnosis of WCT and shows an unusual response of the tachycardia after CSP. Intraventricular conduction slowing induced by flecainide could explain the QRS width of ≈ 190 ms registered during the WCT tachycardia. The apex to base ventricular activation in precordial leads is not consistent with a typical LBBB aberrant conduction. However, Rhee et al. postulated that patients with laterally directed cardiac apices can exhibit negative concordance patterns during SVT with functional LBBB. The presence of deep S waves in lead V5 and V6 during narrow QRS atrial flutter are also consistent with this hypothesis (Figure 3).

When confronted with a WCT it is crucial to integrate both the clinical and the electrocardiographic information and not rely exclusively on ECG algorithms. The medical history (presence or absence of structural heart disease) and the ECG in sinus rhythm, when available, can be of great value in this situation. Moreover, in the presence of haemodynamic stability CSP should be routinely performed. Unfortunately, CSP during WCT is of diagnostic value only when it affects the tachycardia, either terminating it or modifying AV conduction.

An electrophysiological study was performed after discontinuation of flecainide revealing the presence of a typical counterclockwise atrial flutter. After successful ablation of the cavotricuspid isthmus, VT could not be induced with programmed ventricular stimulation.

Conflict of interest: none declared.

References