**ECG for Students and Associated Professionals**

**Wide QRS complex tachycardia responsive to both ATP and verapamil**

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1. Case presentation

   A 38-year-old female with frequent episodes of palpitation was referred to our hospital for the diagnosis and management of tachycardia. Her annual medical check-ups did not reveal any abnormalities, and she had no history of cardiovascular diseases or medications. Her family history was negative for cardiac events and sudden death. No obvious structural heart disease was detected during physical examination, resting electrocardiography, chest radiography, or echocardiography. She generally had palpitations during exercise and when she was under emotional stress, and they terminated spontaneously within 2 h. Surface 12-lead electrocardiogram (ECG) obtained during a lasting episode of palpitation showed wide QRS complex tachycardia with a right bundle-branch block (BBB) pattern and inferior axis (Fig. 1). This tachycardia transiently developed from sinus tachycardia occurring after psychological stress and disappeared spontaneously, with the restoration of sinus rhythm (Fig. 2). A small dose of ATP temporarily suppressed the tachycardia, which was then completely eliminated after the administration of a 5.0-mg dose of verapamil (Fig. 3).

   What is your diagnosis of the tachycardia and its mechanism?

2. Commentary

   Her ECG showed a regular wide QRS complex tachycardia at a rate of 130 beats per minute. The morphology of the QRS complex exhibited the right BBB (RBBB) pattern and inferior axis. The duration of the QRS complex was 0.16 s. P waves of sinus origin were not seen, presumably hidden in the QRS complex, during the tachycardia.

   Wide QRS tachycardia can be divided into 3 different types: (1) supraventricular tachycardia (SVT) with intraventricular aberrant conduction; (2) SVT with atrioventricular conduction over an accessory pathway; and (3) ventricular tachycardia (VT) [1–3]. Width of the QRS complex is a classical parameter used to differentiate VT from SVT with aberrancy. A QRS width of more than 0.14 s in RBBB tachycardia and 0.16 s in left BBB (LBBB) is highly suggestive of VT [1–3]. On analyzing the QRS width, this tachycardia appeared to be VT rather than SVT with aberrancy. However, the QRS width is not helpful in differentiating VT from SVT conducting to the ventricle over an accessory pathway. Configurational characteristics of the QRS complex can also be very helpful in correctly differentiating RBBB pattern tachycardia. An R, qR, QR, RS, or Rs in lead V1 is more often observed in VT, and the presence of an initial R wave in lead aVR has been proposed to be suggestive of VT [1,2,4]. In this case, Rs was distinctively seen in lead V1, but an initial R wave in lead aVR was not observed. Moreover, the pharmacological responses of this tachycardia to the administration of both ATP and verapamil are more consistent with SVT than VT, although VT due to triggered activity is also occasionally terminated with these agents [5].

   Atroventricular (AV) dissociation (more QRS complex than P waves) during tachycardia is a hallmark of VT. P wave was difficult to identify in this case, as shown in Fig. 1. However, in wide QRS complex tachycardia (Fig. 2), the ventricular rate becomes transiently higher than the atrial rate. In the figure, the QRS complex is recorded after the P wave (arrow) in the 5th and 6th beats, and P wave in the 7th, 8th, and 9th beats is hidden within the QRS complex. Subsequently, the atrial rate again was higher than the ventricular rate. These findings suggest that AV dissociation was transient, which leads to the diagnosis of...
VT. The QRS complex (asterisks) following the P wave in the 5th, 11th, 12th, and 13th beats indicate fusion, in which one impulse from the ventricle and a second supraventricular impulse simultaneously activate the ventricular myocardium.

Consequently, in view of the abovementioned diagnostic features, this tachycardia was diagnosed as idiopathic VT. The clinical characteristics of idiopathic VT in subjects without structural heart disease have been explored. In particular, VT exhibiting QRS morphologies of RBBB with inferior axis and tall R in V5/V6 is believed to originate in the left ventricular outflow tract (LVOT). VT from LVOT is not as common as that from the right ventricular outflow tract (RVOT) and comprises 10–18%
of wholly idiopathic, adenosine-sensitive VTs. Most VTs arising from the LVOT, as seen in the present case, are sensitive to catecholamine and easily suppressed by ATP and vagal stimulation [5]. Moreover, β-blockers and verapamil are also sometimes effective [5]. Therefore, the mechanism of VTs originating from the LVOT is mostly thought to be cAMP-mediated triggered activity similar to that of VTs originating from the RVOT since the former suppress the slow-inward current directly by modulating the L-type calcium channel or indirectly by inhibiting the production of cellular cAMP [5].

Catheter ablation has been proven effective and has a high success rate in the elimination of VTs originating from the LVOT as well as those originating from the RVOT, by targeting the earliest activation site or the site of perfect-pulse mapping [5–7]. This was also verified in this case where VT was successfully eliminated by the application of radiofrequency energy at the infravalvular sites around the aortomitral continuity. The patient has remained free of VT during a follow-up period of 5 years.

References