Post-exercise Paroxysmal Tachycardia with QRSs Changing from Wide to Narrow Complexes

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1. Case presentation

A 33-year-old man visited the outpatient clinic complaining of episodic palpitations, which began to develop 3 years earlier but increased frequency lately. Characteristically, the palpitations develop immediately after vigorous exercise and terminate spontaneously within 5 min. Each episode was accompanied by chest tightness and dizziness, with cold sweat.

Physical examination was unremarkable, and his resting 12-lead EKG showed normal sinus rhythm with normal P, QRS and T morphologies. He then underwent treadmill exercise testing where he walked for 9 min and completed stage 3 of the Bruce protocol with the heart rate (HR) reaching 164/min, the 85% predicted maximal HR, without having chest pain or ischemic ST changes.

One minute after termination of exercise, the monitoring EKG showed a sudden onset of wide QRS tachycardia with LBBB morphology (Figure 1). He experienced abnormal palpitations and the BP decreased from 160/43 to 96/77 mmHg. Twenty seconds later, the wide QRS complexes suddenly changed into narrow QRS complexes associated with a HR increase from 200 to 240/min (Figure 2). The tachycardia soon terminated spontaneously, and the BP returned to 149/89 mmHg.

What is your diagnosis of the tachycardia?

2. Commentary

Exercise related tachycardia is either catecholamine-dependent or just HR-dependent. In contrast, tachycardia developing after exercise may reflect a delicate mixture of autonomic tones comprising sympathetic withdrawal and reflex parasympathetic activation, which would create underlying substrates for abnormal automaticity and/or reentry. The mechanism of the tachycardia will be explored through detailed analysis of the EKG. Let us first look at Figure 1.

Although what is most impressive in Figure 1 is a wide QRS tachycardia for which we may suspect ventricular tachycardia (VT), one has to be reminded to read every nook and cranny of the EKG. The rhythm just before the onset of tachycardia was sinus tachycardia at HR of 130/min. Make sure this was recorded one minute after exercise was completed, and the absence of ST depression at this HR almost always rules out background pathology of obstructive coronary artery disease. This is important since it indicates that the development of subsequent wide-QRS tachycardia was not induced by myocardial ischemia. In addition, the fact that this patient completed the 10 METS exercise would also exclude the possibility of heart failure. If there were signs of ischemia or heart failure at the initiation of wide-QRS tachycardia, one would naturally suspect VT as the diagnosis, but this was not the case.

If the wide-QRS tachycardia was of ventricular origin, it would more likely be idiopathic VT developing in the absence of structural heart disease. And if idiopathic VT is exercise related and of LBBB morphology, it is most typically of right ventricular (RV) outflow tract origin showing an inferior axis. On the contrary, however, the tachycardia of this case shows a superior axis, suggesting the origin of the wide-QRS complexes is most likely to be located near the apex of the right ventricle.

Here, one would speculate another mechanism of wide-QRS tachycardia, which is supraventricular
tachycardia (SVT) with functional LBBB. Although the majority of aberrant conduction during SVT show RBBB morphology, aberrant conduction with LBBB morphology can also be seen, in which case, the atrioventricular (AV) conduction takes place through the right bundle branch (RBB) and ventricular myocardial conduction typically starts near the RV apex, showing a similar QRS morphology to that of the present case.

A possibility of VT is further excluded when one looks at the initiation of tachycardia. The tachycardia is induced by a single atrial premature excitation characterized by a premature P wave on top of the preceding T wave, making the T wave taller than those in the preceding beats during sinus tachycardia. Without much PR prolongation, this premature P wave is followed by a single premature beat with incomplete LBBB morphology, which itself would exclude a possibility of VT. This was then followed by tachycardia with complete LBBB morphology. At this point, the tachycardia is diagnosed as paroxysmal SVT with aberrant conduction. In view of the relatively short PR interval at the onset, the diagnosis of AV nodal reentrant tachycardia is least plausible, but the mechanism of the tachycardia can be further clarified by analyzing Figure 2.

Here, a sudden transition of QRS morphology from wide complexes of LBBB pattern to narrow complexes is seen. This type of transition is not rare during SVT and can be interpreted as the effective refractory period of the left bundle branch (LBB) becoming shortened enough to eventually allow conduction through LBB as the tachycardia is sustained. Typically this transition would occur when the HR slows down due to decremental AV nodal conduction and the RR interval is prolonged enough to allow conduction through previously refractory tissues (LBB in this case). What is unusual and surprising in this tracing is that the HR rather increased suddenly from 200 to 240/min as aberrant conduction disappeared. This paradoxical acceleration can only be explained by introducing another unique mechanism involved in this case.

The paradoxical acceleration from 200 to 240/min means that there was a shortening of conduction time by 50 msec with tachycardia cycle length decreasing
from 300 msec to 250 msec. To put it another way, the conduction time for the reentrant pathway was 50 msec shorter when the LBB was used than when the RBB was the only pathway used for AV conduction. The sole possible explanation for this phenomenon is the utilization of the left-sided accessory pathway (AP) for the retrograde ventriculo-atrial conduction. With this left-sided AP, the reentrant circuit is shorter when the LBB is available for AV conduction compared to the circuit when the RBB, but not LBB, is available. Therefore, one should be proud of being able to diagnose the presence of a concealed left-sided AP contributing to AV reentrant tachycardia based on a single EKG tracing. This interesting and useful sign of tachycardia acceleration concomitantly with disappearance of aberrant conduction in the presence of AP ipsilateral to the blocked bundle branch has been first described by Phillip Coumel and has often been referred as the Coumel’s sign. Ablation of the culprit AP is apparently the next strategy to cure this patient’s problem.

Figure 2  Transition from wide- to narrow-QRS morphology during tachycardia