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CASE REPORT

Presence of Septal Q Waves in a Patient with WPW and Manifest Preexcitation

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Wolff-Parkinson-White syndrome (WPW) is characteristically diagnosed by the presence of a short PR interval, a delta wave, and a wide QRS wave on the surface ECG. In the absence of these clear criteria, absent septal Q waves have been used as additional evidence suggestive of subtle preexcitation. We report a patient with WPW and manifest anteroseptal (AS) accessory pathway who had prominent septal Q waves on the surface ECG. This case highlights that physicians should be careful not to dismiss preexcitation in the presence of septal Q waves.

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electrophysiology; AV re-entry/WPW; noninvasive techniques; electrocardiography

CASE REPORT

A 46-year-old man presented to the emergency room with wide-complex tachycardia at 240 bpm. He was given intravenous procainamide that successfully terminated the palpitations. The patient had a history of palpitations that usually terminate when he bears down or puts cold water to his face. The patient denied chest pain or recent changes in exercise tolerance. An electrocardiogram (ECG) was obtained in sinus rhythm after termination of tachycardia (Fig. 1). The ECG analysis shows Wolff-Parkinson-White syndrome (WPW) pattern with positive delta waves in V_1–V_6 and QRS transition from V_1 to V_2, suggestive of a septal accessory pathway. Patient is noted to have Q waves in the lateral leads, V_5 and V_6. Echocardiography showed normal LV function and stress test was normal. Electrophysiology study revealed that patient had an anteroseptal (AS) accessory pathway (consistent with past history of WPW) that was successfully ablated with cryoablation. This postablation ECG (Fig. 2) shows RBBB with septal Q waves in the lateral leads. Patient did not have symptoms or WPW ECG pattern in long-term follow-up.

DISCUSSION

In a normally functioning conduction system, ventricular activation only begins when the left septal fascicle activates a portion of the septal myocardium in the left to right direction.[1] This causes an initial negative deflection, septal Q wave, in leads I, aVL, V_5, and V_6.[2] Individuals with WPW have an accessory pathway that allows for an alternate means of conduction from the atria to the ventricles.[3] This accessory pathway bypasses the AV node to allow faster ventricular conduction during atrial fibrillation, putting the patient at risk for ventricular fibrillation.[3] There is some evidence that WPW with ventricular preexcitation can obscure the septal Q wave incurred on an ECG. The septal Q is not visualized because ventricular preexcitation via the accessory pathway occurs before the left bundle activates the ventricular.[1,4,5] Bogun et al. showed that none of 37 patients with minimal preexcitation had a septal Q wave in lead V_6. Eighty percent of these 37 patients recovered the septal Q wave postablation.[1] The authors concluded that the presence of a septal Q wave on the surface ECG in lead V_6 excludes manifest preexcitation.[1]

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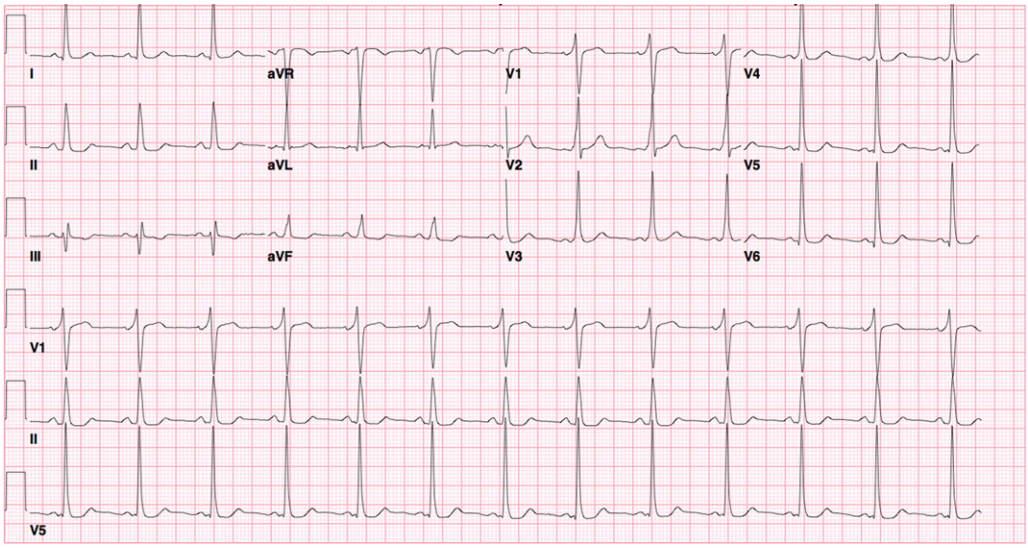

Figure 1. Preablation ECG taken December 7, 2009.

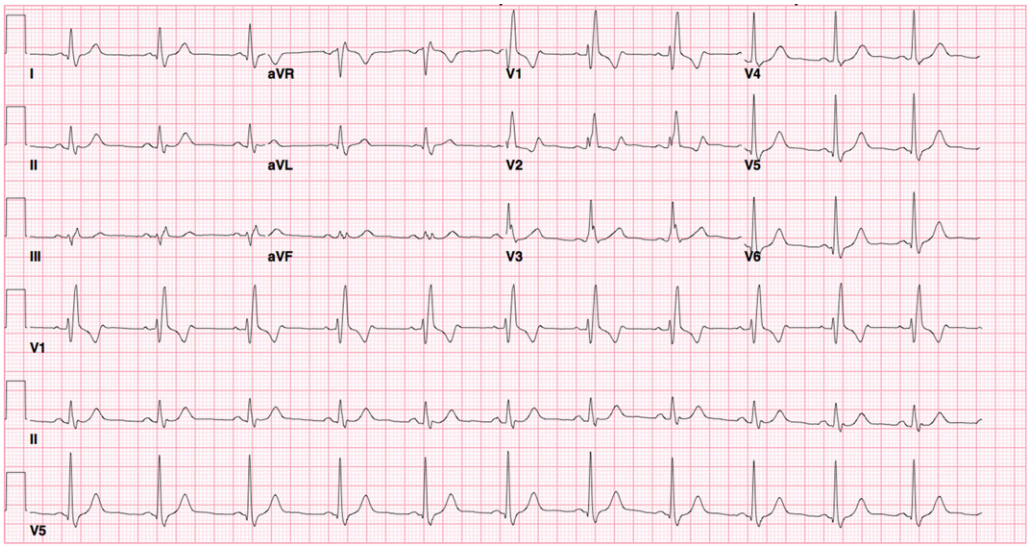

Figure 2. Postablation ECG taken May 24, 2010.

There are exceptions to the use of the septal Q wave as an exclusion criterion for ventricular preexcitation. In preexcitation patients with a left-sided accessory pathway, the rSR′ complex can be misinterpreted to give an impression of a septal Q wave.[1] In preexcitation patients with atriofascicular pathways inserting close to the apex, the apex to base ventricular activation can give a septal Q wave in V_6.[1] Patients with structural heart disease or multiple accessory pathways could have septal Q waves despite ventricular preexcitation.[1] Another study showed that 15% of WPW patients who had a left accessory pathway and longer than average HV (His Ventricular) interval (15 ± 11 ms) had septal Q waves.[4]

Preablation, our patient with WPW had a prominent septal Q wave in lead V_6. The patient did not have structural heart disease or multiple accessory pathways. He had an AS atrioventricular pathway (not a left-sided accessory pathway or an atriofascicular pathway as reported in previous studies). The patient had an HV interval of 20 ms that is longer than average HV interval reported in previous studies in patients with WPW.[1,4]

The longer HV interval indicates relatively faster conduction in the AV node or relatively slower accessory pathway conduction. Hence the normal left to right septal conduction is likely not masked, giving rise to a septal Q wave on the ECG. Another possible explanation for this finding is that the distal insertion of the accessory pathway is into ventricular tissue that is proximal to the right bundle. The patient had RBBB as noted on the postablation ECG and the ECG during orthodromic atrioventricular reentrant tachycardia (AVRT) preablation. This could have delayed ventricular activation by the accessory pathway. Hence faster activation of the septal myocardium by the left septal fascicle could result in a septal Q wave on the ECG. There are no reported cases of patients with AS accessory pathway with septal Q waves in the lateral leads.

CONCLUSION

We present a patient with WPW and an AS accessory pathway who had septal Q waves on his ECG. In this case, the Q wave was likely a result of either a slower pathway conduction (relative to the AV node) or distal insertion of pathway proximal to right bundle in this patient with RBBB. Therefore, while the absence of septal Q wave argues for WPW, its presence should not exclude a diagnosis of WPW.

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