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Unusual manifestation of left ventricular electrical conduction delay on the surface 12-lead electrocardiogram in a patient with prior myocardial infarction☆

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ABSTRACT

We describe a 41-year-old man with a prior history of myocardial infarction, whose surface 12-lead electrocardiogram did not show typical left bundle-branch block pattern or wide QRS complex. However, electrophysiological study showed distinct left ventricular electrical conduction delays. The surface 12-lead electrocardiogram modified to the paper at 50 mm/s and double standard (20 mm equals 1 mV) revealed obvious notches of the terminal forces of the QRS in leads II, III, aVL, aVF, V3, V4, V5, and V6, these might be partially consistent with left ventricular electrical conduction delay in the scar lesion of the infero-posterior of the ventricle.

Case presentation

A 41-year-old man was referred to our institute with dizziness. He had a prior history of myocardial infarction (MI) involving occluded left circumflex artery (LCX). A 12-lead electrocardiogram (ECG) on admission revealed wide QRS regular tachycardia at a heart rate of 189 beats/min with evidence of atrioventricular dissociation (Fig. 1A). Therefore, monomorphic ventricular tachycardia (VT) was diagnosed. A wide QRS complex with a right bundle-branch block and superior axis during VT was suggestive of its exit at the LCX lesion. VT spontaneously terminated and converted to sinus rhythm (SR). The QRS configuration during SR was characterized by a width of 108 ms with initial r wave >1 mm in lead V1, and absence of mid-QRS notching in leads I, V5, and V6 (Fig. 1B). Echocardiography revealed akinesis of the infero-posterior wall of the left ventricle (LV) with an ejection fraction of 48%. Cardiac MRI showed a late gadolinium enhancement in the infero-posterior wall of the LV. These findings were suggestive of myocardial fibrosis, which could lead to the underlying arrhythmogenic substrate of VT. Therefore, the scar-related monomorphic VT was diagnosed and electrophysiological study was performed. The electrode catheters deployed at the coronary sinus (CS) recorded sequential left ventricular electrical conduction delay (LVCD) during SR (Fig. 2). Although monomorphic sustained VT was induced during the procedure, VT was hemodynamically unstable. Therefore, radiofrequency catheter ablation (RFCA) targeting diastolic fragmented potentials in the LCX lesion during SR was performed. After the RFCA, an implantable cardioverter-defibrillator was implanted.

Discussion

Previous studies have reported that prolonged QRS duration with left bundle-branch block (LBBB) patter predicts longer LVCD and this electrophysiological criterion could select appropriate candidates for

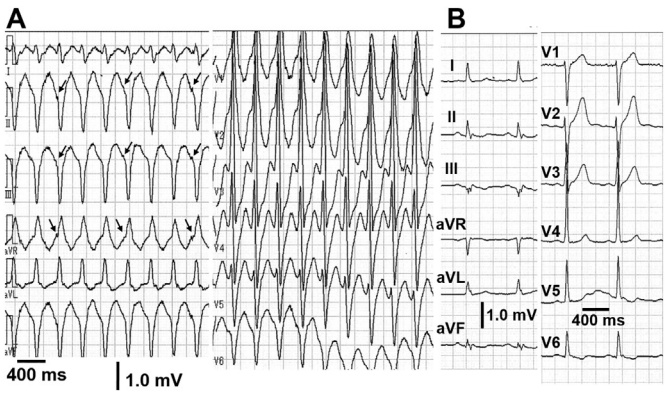

Fig. 1. (A) The 12-lead ECG on admission. Wide QRS regular tachycardia with evidence of atrioventricular dissociation (arrows: P waves) is indicative of VT. (B) The 12-lead ECG during SR.

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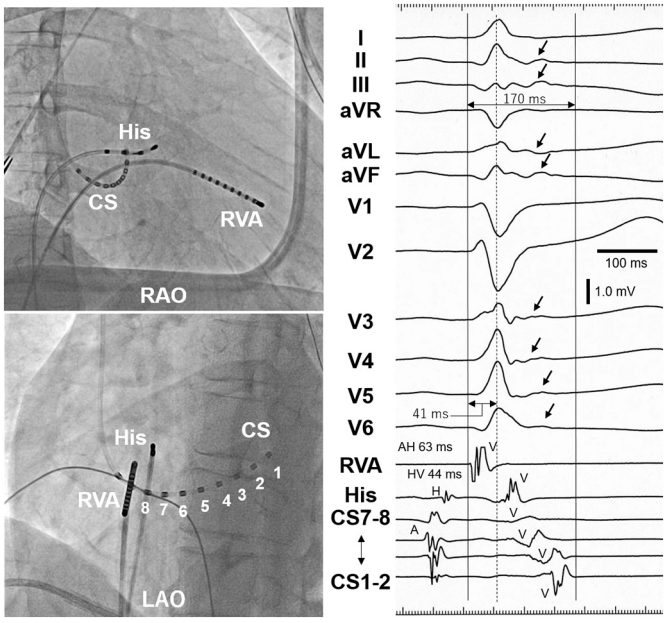

Fig. 2. Demonstration of surface and intracardiac electrocardiograms. Intrinsicoid deflection, defined as time from the beginning of QRS to peak of the R wave (a dotted line), was 41 ms in lead V6. Note that ventricular activations recorded at the coronary sinus are sequentially delayed after the peak of the R wave. The interval from the beginning of the QRS to the end of the late ventricular potentials is 170 ms (vertical lines). Also note that these left ventricular conduction delay are partially correlated with the obvious notches of the post-QRS in leads II, III, aVL, aVF, V3, V4, V5, and V6 on the surface ECG (arrows). AH, atrio-His; CS, coronary sinus; HV, His-ventricular; LAO, left-anterior oblique; LV, left ventricle; RAO, right-anterior oblique; and RVA, right ventricular apex.

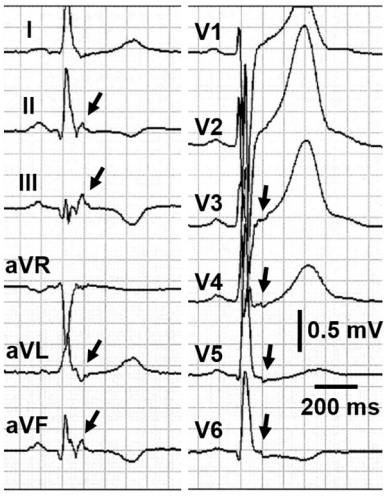

Fig. 3. The same 12-lead ECG as Fig. 1B in which the paper speed was increased to 50 mm/s and double standard was used (20 mm equals 1 mV). Note that obvious notches of the terminal forces of the QRS in leads II, III, aVL, aVF, V3, V4, V5, and V6 (arrows).

cardiac resynchronization therapy (CRT) [1–3]. In the present case, standard precordial leads did not show typical LBBB (Fig. 1B); consequently, LVCD was not predictive. However, electrode catheters depicted sequential marked LVCD in the scar lesion (Fig. 2). When the same ECG was modified to the paper at 50 mm/s and double standard (20 mm equals 1 mV; Fig. 3), notches of the terminal forces of the QRS in leads II, III, aVL, aVF, V3, V4, V5, and V6 were obvious, these were partially consistent with the scar lesion of the infero-posterior of the ventricle (arrows in Fig. 3). Although activation mapping during SR was not obtained from the whole ventricle, these post-QRS notches might be partially correlated with the LVCD in the scar lesion.

Some patients with heart failure present with a wide QRS complex, that is neither an LBBB nor a right bundle branch block (RBBB): nonspecific intraventricular conduction delay (NIVCD). Few studies have investigated the relationship between the NIVCD and its electrophysiological findings [4].

Future study should investigate the relationship between the ECG and intraventricular electrical conduction obtained from the EPS, that may be helpful to identify patients with NICD who will be appropriate candidates for CRT.

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