CASE REPORT

Ventricular tachycardia with a myocardial fibre travelling from the origin in the right aortic sinus cusp to the epicardial breakout site of the right ventricular outflow tract

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This report describes a case of ventricular tachycardia (VT) with a possible myocardial fibre travelling from the origin in the aortic sinus cusp (ASC) to the epicardium of the ventricular outflow tract. This case may provide a clinical implication for catheter ablation of VT originating from the ASC.

Case report

A 74-year-old man with symptomatic idiopathic VT underwent electrophysiologic testing. Programmed electrical stimulation during an isoproterenol infusion induced two different VTs. One VT had a left bundle branch block and right inferior axis QRS morphology (VT1) (Figure 1) and the other had a right bundle branch block and right inferior axis QRS morphology (VT2). In VT1, mapping in the right ventricular outflow tract (RVOT) was first performed using the femoral approach, but it revealed neither an early ventricular activation preceding the QRS onset nor an excellent pace map. Finally, successful radiofrequency (RF) ablation was achieved in the right aortic sinus cusp (RCC) (Figure 2). An excellent pace map with a long stimulus-to-QRS interval (St-QRS) (85 ms) was obtained at the successful ablation site in the RCC (Figure 1). In VT2, successful ablation was achieved on the epicardial surface of the anterior wall of the left ventricle using an epicardial access by a pericardial puncture. After elimination of VT2, pace mapping was performed on the epicardial surface around the ventricular outflow tract. Although an excellent pace map of VT1 was also obtained on the epicardial surface of the RVOT ~1 cm away from the successful ablation site in the RCC, the St-QRS was 40 ms (Figures 1 and 2).

Discussion

It has been demonstrated that RF catheter ablation is a safe and reliable technique for curing ventricular arrhythmias originating from the ASC.1,2 Although there have been a few reports describing VTs with ASC origins and endocardial breakout sites in the left ventricular outflow tract (LVOT)3 or RVOT,4,5 the details about the myocardial network around the ventricular outflow tract remain unknown. In this case, an excellent pace map was obtained both in the RCC and on the epicardial surface of the RVOT away from the RCC. However, the St-QRS was much greater during pacing in the RCC than on the epicardial surface of the RVOT. These findings suggest that an insulated myocardial fibre travelling from the origin in the RCC to the epicardial breakout of the RVOT might exist. Such a myocardial fibre may cause VTs

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The pace map in the RCC actually exhibited a little narrower QRS complex than VT1 and the epicardial pace map. That finding may be explained by a mechanism in which the pacing in the RCC might also have captured another part of the myocardium. Of course, such a musculature would not have been the local myocardium surrounding the origin in the RCC, but an insulated fibre from near the origin of VT1 in the RCC to the endocardial breakout site close to the epicardial breakout site, because otherwise, the ST-QRS would have been much shorter. If the pacing in the RCC had captured both myocardial fibres to the endocardial and epicardial breakouts simultaneously, it might have produced a narrower QRS than when the activation from the origin in the RCC propagated over only the myocardial fibre to the epicardial breakout. The ST-QRS during pacing in the RCC was actually longer than expected when the activation from the origin to the epicardial breakout. Therefore, the myocardial fibre may have been diseased and associated with slow conduction.

References