A case of ridge-related re-entrant atrial tachycardia utilizing the vein of Marshall to span a conduction gap at the mitral isthmus scar

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A 41-year-old man experienced atrial tachycardia (AT) after multiple ablative procedures for atrial fibrillation. Electroanatomical mapping revealed the apparent impulse propagation through the scar at the mitral isthmus. An electrode in the vein of Marshall (VOM) showed the VOM was on the circuit of AT. Radiofrequency energy application at the connection between the VOM and left atrium terminated the AT.

A 41-year-old man had undergone pulmonary vein isolation and linear ablation at the roof and mitral isthmus (MI) for persistent atrial fibrillation. He subsequently experienced atrial tachycardia (AT) with a cycle length of 310 ms. Therefore, an electroanatomical mapping system (Ensite NavX; St Jude Medical, Minneapolis, MN, USA) was used to construct an activation map during the AT. This map revealed

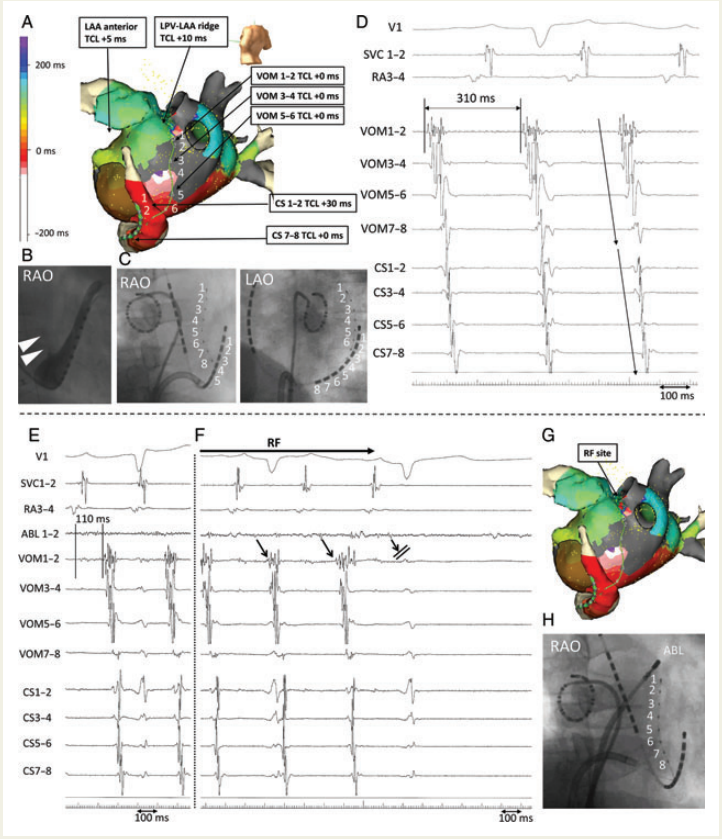

Figure 1 (*A*) The scar tissue was defined as an area with a local voltage of ≤0.1 mV. Post-pacing intervals along anterior LAA, LPV–LAA ridge, the VOM, and proximal CS are equal to the TCL, although the PPI at the distal CS (CS 1–2) is not comparable with the TCL. The PPI map indicated that the AT propagated around the MA and LAA. (*B*) Venography of CS revealed proximal portion of the VOM (white arrow head). (*C*) Fluoroscopy indicates the placement of the VOM and CS catheters. The CS was cannulated with a duo-decapolar electrode catheter. (*D*) The intracardiac electrograms during AT. (*E*) Intracardiac electrograms during AT, when the ablation catheter (ABL 1–2) at the LPV–LAA ridge recorded fractionated potentials of 110 ms. (*F*) Intracardiac electrograms during RF ablation, which terminated AT within 4 s of producing conduction block between the LPV–LAA ridge and the VOM. (*G*) Activation map and (*H*) fluoroscopic view of the successful ablation site. PPI, post-pacing interval; LAA, left atrial appendage; LPV–LAA, left pulmonary vein–left atrial appendage; VOM, vein of Marshall; TCL, tachycardia cycle length; CS, coronary sinus; RAO, right anterior oblique position; LAO, left anterior oblique position; SVC, superior vena cava; RA, right atrium; AT, atrial tachycardia; ABL, ablation catheter; RF, radiofrequency.

macro-re-entrant AT with impulse propagation in a clockwise direction around the mitral annulus (MA) and the left atrial appendage (LAA), and through the scar tissue bundle at the MI (*Figure 1A*). We cannulated the vein of Marshall (VOM) with a 2-Fr electrode. The impulse from the ridge area was conducted along the VOM and to the coronary sinus (CS) (*Figure 1D*), and the post-pacing intervals at multiple points along the VOM were equal to the tachycardia cycle length (*Figure 1A*). The fractionated potentials at the left pulmonary vein–LAA (LPV–LAA) ridge expanded to a duration of 110 ms, and preceded the electrogram of the distal VOM (VOM 1–2) (*Figure 1E*). Therefore, radio-frequency (RF) ablation was applied at the LPV–LAA ridge (*Figure 1G* and *H*) using an irrigated tip ablation catheter, which successfully terminated AT after 4 s of application (*Figure 1F*). Differential site pacings were subsequently performed at the CS and left atrium to confirm bidirectional block across the MI, distal VOM, and the LPV–LAA ridge.

Discussion

Valderrabano *et al.*[1] established the technique to cannulate the VOM. After that, it was demonstrated that the VOM plays a role in the re-entry circuit in some case reports.[2] In this case, the VOM bypassed the epicardial impulse propagation across the scar tissue at the MI and formed a re-entrant circuit. Similarly, Takatsuki *et al.*[3] have reported that AT after ablation of the pulmonary vein and MI was caused by a ridge-related re-entry (RRR). The present case had similarities to certain aspect of their cases, with the exception of apparent conduction across the previous linear lesion scar at the MI.

To the best of our knowledge, this is the first case of RRR in which the VOM has played a role in the epicardial conduction across the low-voltage area. This type of re-entrant tachycardia is caused by slow impulse conduction via the contact site between the LPV–LAA ridge and the VOM. When a conduction gap is apparent at the MI lesion and RF ablation at the MI does not produce a complete bidirectional block, we suggest that RF ablation be applied to the connection between LPV–LAA ridge and VOM area guided by the electrode in the VOM.

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