Limb lead reversal giving a potentially misleading ECG

Topsy-turvy ventricular tachycardia

Federico Moccetti[a,b], Jan Novak[c], Aline Mühl[a], Michael Kühne[a]

[a] Cardiology/Electrophysiology, University Hospital Basel, Switzerland; [b] Knight Cardiovascular Institute, Oregon Health and Science University, Portland, Oregon, USA; [c] Cardiology/Electrophysiology, Bürgerspital, Solothurn, Switzerland

Case presentation

We present the case of a 72-year-old male with a history of coronary artery disease and an inferior myocardial infarction 19 years previously, when the occluded right coronary artery was percutaneously revascularised. A DDD pacemaker had been implanted 3 months before because of complete atrioventricular (AV) block. The chief complaint upon presentation was fatigue, and the patient denied chest pain or dyspnoea. No clinical signs of congestive heart failure were noted on physical examination. Haemodynamic parameters were stable. The ECG on admission is shown in figure 1A.

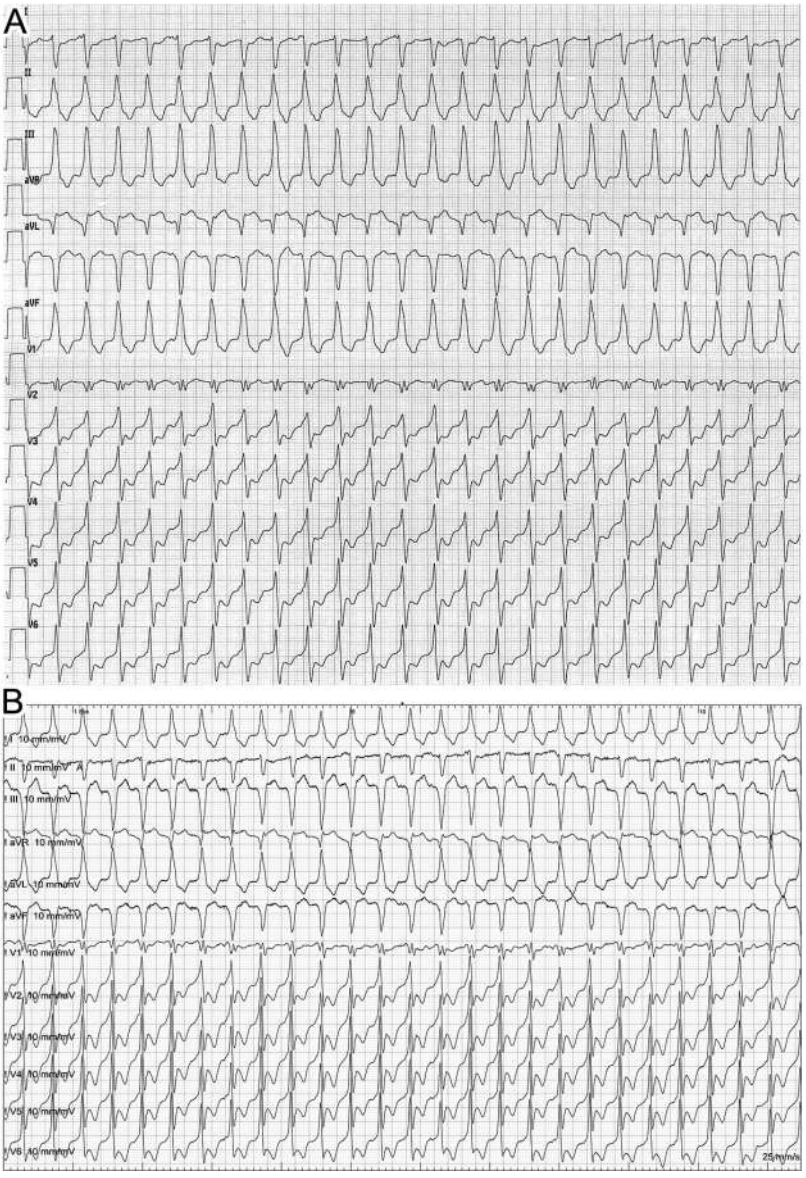

Figure 1: (A) 12-lead ECG on admission.
(B) Easily and reproducibly inducible VT during electrophysiological study. Note the almost identical cycle length and QRS morphology in the precordial leads. However, there was now a superior axis based on the limb leads. Note that all limb leads are different except aVR suggesting limb lead reversal between the left arm and the left leg.

Questions

1 What is the rhythm?
2 Where is the origin of the arrhythmia?

Comment

Figure 1A shows a regular wide complex tachycardia with a heart rate of 150 bpm. A diagnosis of ventricular tachycardia (VT) was made, based on the known persistent complete AV block and a QRS morphology compatible with VT with a Q in V1. Owing to the transition between V1 and V2 and the inferior axis, an anterior origin of the VT in the left ventricle was suspected, not compatible with the location of the scar after inferior myocardial infarction.

As a result of the recurrent VT documented on telemetry at the given rate, catheter ablation of the VT was attempted. At the beginning of the electrophysiological study, spontaneous premature ventricular contractions (PVCs) were present. In the precordial leads, the PVC morphology was almost identical to the VT on the initial ECG, but the limb leads showed a superior axis. A VT with the morphology of the PVCs was easily and reproducibly inducible using isoprenaline. The cycle length and QRS morphology in the precordial leads were almost identical to the VT at presentation, but the VT had a superior axis (fig. 1B). Haemodynamic parameters were stable. The VT terminated spontaneously and was often nonsustained.

After transseptal puncture, electroanatomical voltage mapping with a 3D mapping system and remote magnetic navigation (CARTO 3, Stereotaxis) showed a local-

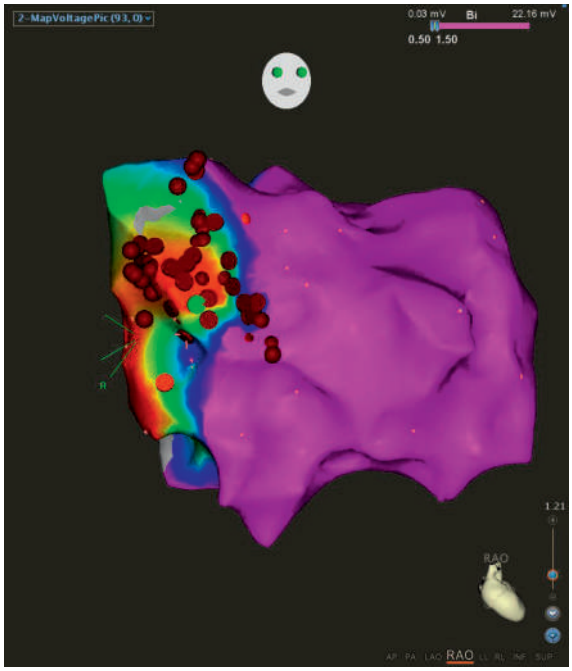

Figure 2: 3D electroanatomic voltage map of the left ventricle showing an infero-septo-basal scar. The purple areas correspond to areas with normal endocardial voltage, the red areas correspond to sites of endocardial scar, and the remaining colors correspond to the transition zone. The green tag denotes the site with a perfect pace-map (12/12). The dark red dots denote ablation sites. The bright red dots denote sites with no matches based on pace-mapping.

ised endocardial infero-septo-basal scar. Pacing at that site accurately reproduced the morphology of the VT. Ablation at that site eliminated the VT. After ablation, no further VT could be induced with programmed ventricular stimulation.

With the given history, a left ventricular ejection fraction of 35–40% and optimised medical treatment (angiotensin converting-enzyme inhibitor, β-blocker and aldosterone receptor antagonist) the patient subsequently underwent an upgrade to a DDD implantable cardioverter defibrillator. He was arrhythmia-free during a follow up of 6 months.

Discussion

This case nicely illustrates the potential implications of incorrectly placed leads in the surface 12-lead ECG. Incorrect lead placement may have clinically relevant consequences [1]. A large retrospective study of 11 432 ECGs showed a rate of incorrect lead placement of 2% [2]. In a study analysing the detection capability of cardiologists with regard to incorrect lead placement, the right arm lead was switched with the right leg on purpose. Of the 25 experienced cardiologists, none was able to detect the ECG abnormality [3]. Incorrectly placed leads may lead to a bizarre QRS-axis, low voltage in leads I, II or III, or generally findings that do not correlate with the patient's history, clinical or echocardiographic examination. Based on the number of electrodes used during electrocardiography, a plethora of possible displacement "options" exist (contralateral interchange, homolateral interchange, cross-over interchange, clockwise and counterclockwise interchange). A helpful overview on different ECG patterns and how to recognise the most common lead reversals is provided elsewhere [4, 5].

In our patient, the suspected localisation of the clinical VT was in the anterolateral aspect of the left ventricle, but this was not in accordance with the patient's history of inferior myocardial infarction. Since lead reversal (reversal of left arm and left leg) was suspected because of spontaneous PVCs with a nearly identical QRS morphology in the precordial leads but a superior axis based on the limb leads, the subsequently induced VT (with morphology identical to the spontaneous PVCs) could be found and eliminated in an infero-septo-basal scar zone.

The presented case shows that limb lead reversal may confuse the clinician not only when occurring during sinus rhythm but also, and probably even more so, when an arrhythmia is present. Failure to recognise limb lead reversal in this case could lead to several consequences including a prolonged ablation procedure with mapping focusing on and possibly even ablation of clinically irrelevant sites in the left ventricle.

Correspondence:
Federico Moccetti, MD
Knight Cardiovascular Institute, OHSU
3181 S.W. Sam Jackson Park Rd
Portland, OR 97239, USA
moccetti[at]ohsu.edu

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