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Case Report

Epicardial Ablation: Prevention of Phrenic Nerve Damage by Pericardial Injection of Saline and the Use of a Steerable Sheath

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Abstract

Because of the close proximity of the phrenic nerve to the pericardium, phrenic nerve damage caused by epicardial ablation can easily occur. We report two cases of epicardial VT ablation where pericardial injection of saline, combined with the use of a steerable sheath, successfully prevents the phrenic nerve from being damaged.

Key Words: Ablation, epicardium, pericardium, phrenic nerve, saline, steerable sheath

Introduction

In recent years epicardial ablation has become an increasingly used treatment modality for ventricular tachycardia (VT) ablation.[1] Because of the close proximity of the phrenic nerve (PN) to the pericardium, PN damage caused by ablation can easily occur, resulting in PN palsy.[2] The exact course of the PN may be identified by pace-mapping with high-voltage output and use of a 3-dimensional imaging system.[3]

We report two cases of epicardial VT ablation where pericardial injection of saline, combined with the use of a steerable sheath, successfully prevents the PN from being damaged.

Methods

Electrophysiologic study and identification of epicardial ablation site

The CARTO electroanatomic mapping system (Biosense Webster, Inc., Diamond Bar, CA, USA) was used to generate epicardial voltage maps of the ventricles. Electrograms with delayed components (E-DCs) in the epicardial scar area were identified. The E-DCs had an activation sequence from the edge to the center of the scar. Therefore, radiofrequency (RF)

applications were delivered at the edge of the scar, over E-DCs with the shortest delayed component lateness (i.e., entrance of late potential channels during sinus rhythm), as described previously.[4] Finally, a remap was obtained to establish complete elimination of E-DCs.

Phrenic nerve mapping

In order to prevent PN injury, after determination of the target ablation site the course of the PN was identified by provoking diaphragmatic stimulation with bipolar pacing at 10 mA and 2 ms pulse width from the distal electrode of the ablation catheter. The locations where the PN could be captured were then marked on the electroanatomic map.

Injection of saline to separate the phrenic nerve from the epicardium

To increase the distance between the epicardium and the PN, saline was introduced into the pericardium in steps of 20 ml until PN capture was lost. Arterial pressure and fluoroscopy were carefully monitored during and after each injection. After each bolus of saline, pacing was repeated to evaluate diaphragmatic stimulation and therefore PN capture.

Radiofrequency catheter ablation

RF was delivered using a 3.5 mm Thermo-Cool Navistar catheter (Biosense Webster) and an Agilis steerable epicardial sheath (St. Jude Medical, St. Paul, MN, USA). Energy settings were 40 watt, 45ºC maximum temperature, 17 ml/min ablation catheter flow rate. Successful RF ablation was defined as the complete elimination of all E-DCs and the inability to induce VTs with programmed stimulation. After completion of the RF ablation all fluids were aspirated using a pigtail catheter. Following the procedure, the absence of fluid in the pericardial space was confirmed by transthoracic ultrasound.

Case report

The first patient was a 55-year-old man with no previous cardiac history who suffered three times from syncope. The ECG suggested presence of a VT originating from the LV (**Figure 1b**). After intravenous administration of amiodarone the tachycardia converted to sinus rhythm with frequent ventricular premature complexes (PVC) of the same morphology as the clinical VT. Echocardiography showed global hypokinesia with LV ejection fraction (LVEF) of 45% and LV end-diastolic diameter (LVEDD) of 57 mm. A coronary angiogram showed no significant coronary artery disease. An electrophysiologic study was performed and the clinical VT was induced. The VT met ECG criteria for an epicardial origin. No ablation was performed. Cardiac magnetic resonance imaging (MRI) showed LVEF of 41% and LVEDD of 58 mm, confirming the results of the echocardiogram. An epicardial scar at the posterolateral basal part of the LV also was identified. Subsequently, a single-chamber implantable cardioverter defibrillator (ICD) was implanted. A second electrophysiologic study was performed as described above and a posterolateral basal scar on the epicardium of the LV was identified, with evidence for E-DCs and two channels going through the scar tissue: one close and parallel to the mitral annulus and the other more apical (**Figure 2**). The RF ablation of the first channel was successful and uneventful as there was no PN capture. Before starting RF ablation on the site of the second channel, the PN could be captured along the whole trajectory of the identified channel (**Figure 2**). Without extra precautions RF ablation would have been impossible at these sites. Therefore saline was injected as described above and after injection of 200 ml PN capture was lost. The patient was in sinus rhythm before, during and after the injection, invasive systolic arterial blood pressure remained above 80 mmHg (**Figure 3**). Then all E-DCs could be eliminated by targeting the late potential channel entrance without any collateral damage. After successful completion