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Case Report

Refractory ventricular tachycardia caused by inflow cannula mechanical injury in a patient with left ventricular assist device: Catheter ablation and pathological findings

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ABSTRACT

In patients with left ventricular assist device (LVAD), a minority of post-operative ventricular tachycardias (VTs) is caused by contact between the inflow cannula and the endocardium. Currently, electrophysiologic characteristics and pathologic features of this condition are lacking. We report on a case of a successfully ablated mechanical VT. After VT recurrence, heart transplantation took place. Pathologic observations were consistent with direct tissue injury and inflammation, eventually contributing to persisting arrhythmias. Radiofrequency catheter ablation can be a safe and effective option to treat arrhythmias caused by inflow cannula interference in the short term, although a high recurrence rate is expected.

1. Case report

A 58-year-old man affected by end-stage non-ischemic dilated cardiomyopathy received a left ventricular assist device (LVAD) (HeartMate II, Thoratec Corp., Pleasanton, CA, USA) with a "bridge to candidacy" indication in January 2015. Recurrent episodes of monomorphic ventricular tachycardia (VT) occurred from post-operative day 11, triggered by orthostatism and Valsalva maneuvers. Electrolytic imbalance, LVAD suction events, and inflow cannula malposition were excluded. Initial therapeutic attempts with preload adaptation, LVAD rotation speed reduction, and intravenous amiodarone and lidocaine failed to control the VT recurrences. An electrophysiologic procedure was set up.

1.1. Electrophysiology procedure

The procedure was conducted under conscious sedation, with unchanged LVAD rotating speed. A 3.5 mm bidirectional open irrigated mapping catheter (ThermoChool SmartTouch®, Biosense Webster, Johnson & Johnson, Diamond Bar, CA, US) was inserted into the LV after transseptal access to the left atrium.

When the mapping catheter came in contact with the endocardium facing the upper border of the inflow cannula (Fig. 1A), the clinical tachycardia (right bundle, right inferior morphology) started immediately, and persisted despite contact withdrawal. A quick activation map was obtained, showing centrifugal activation from the site described above. Locally, the bipolar signal did not show diastolic potentials or continuous electrical activity, and the bipolar signal was maximally anticipated on the surface QRS. The unipolar potential showed a steep Q wave without any preceding far-field. Simultaneously, contact artifacts with the inflow cannula reproducibly appeared (Fig. 1C, and D). None of the neighboring sites showed diastolic activity.

A first radiofrequency (RF) pulse application (40 W, T cutoff 41 °C) was able to interrupt the VT after about 30 s. We extended the treatment to all possible cannula-endocardium contact sites. Notably, repetitive contacts with the cannula caused sudden impedance drops (from about 110 to 70 Ω).

At the end of the procedure, the clinical VT was not inducible either with aggressive programmed ventricular stimulation or with induced suction. The procedure was concluded without any complications.

During the subsequent two weeks, no VT recurrence was noted. On postoperative day 14, however, short runs of VT appeared

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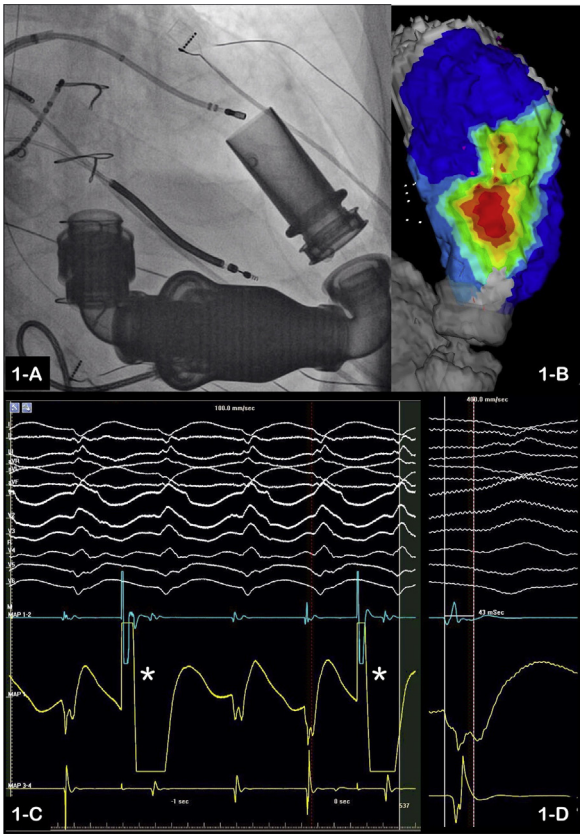

Fig. 1. Ventricular tachycardia activation mapping, (A) Fluoroscopic 30° right anterior oblique view of the mapping catheter at the effective radiofrequency ablation site. (B) Electroanatomic activation map merged with the left ventricle-cannula shield obtained from angio-computed tomography (isochrone set at 5 ms). (C) Mapping catheter at the effective radiofrequency site. Contact artifacts with the inflow cannula are indicated with white asterisks. (D) Maximal anticipation of bipolar potential on surface electrocardiogram and initial downslope of the unipolar potential.

again, with the same features. While a second procedure was being planned, heart transplantation took place 5 weeks after the first ablation procedure.

1.2. Pathology observations

A pathological gross specimen of the explanted heart, as well as histological preparations, were reviewed. The endocardium corresponding to the upper lateral wall of the inflow cannula appeared whitish, with multiple ecchymotic discontinued lesions, corresponding to RF application sites (Fig. 2). The histology revealed dense subendocardial fibrosis with diffuse macrophage infiltration, both at the interface with areas of RF-induced coagulative necrosis and granulation tissue, and at the watershed between vital cardiomyocytes and fibrotic tissue, far from RF lesions. Remote vital myocardium showed cardiomyocyte disarray, with mild interstitial fibrosis and no infiltrates.

2. Discussion

VT occurrence after LVAD implantation has been associated with prior myocardial fibrosis as a substrate for reentry in more than 90% of cases [1]. Reentry involving apical scarring around the inflow cannula or mechanical contact of the latter with the endocardium have rarely been described [2].

In our case, a cardiovascular magnetic resonance performed before the implantable cardioverter-defibrillator implant did not show myocardial enhancement. The implantable cardioverter-defibrillator was implanted for primary prevention of arrhythmic cardiac death, and no ventricular sustained arrhythmia was detected by the device before the LVAD implant.

We observed distinctive tissue alterations, characterized by inflammation and reparative fibrosis, all confined to the subendocardium. Considering the time course of myocardial healing process, we can almost exclude that reparative fibrosis had been

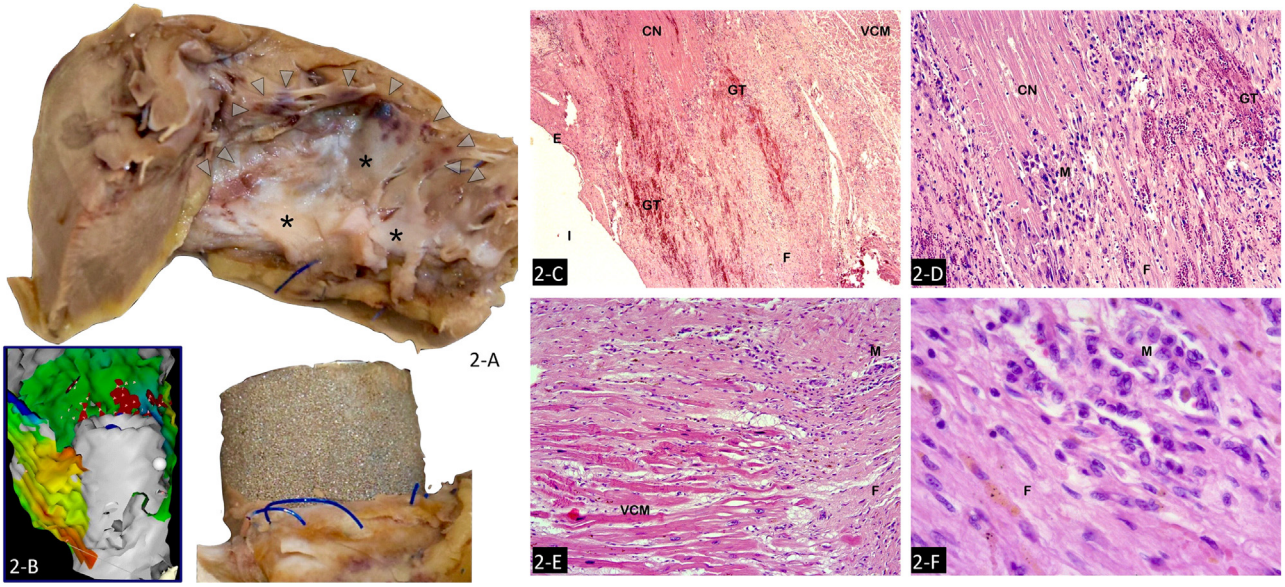

Fig. 2. (A) Gross view of the endocardial aspect of the mid-lateral left ventricular wall from the explanted heart with the inflow cannula; black asterisks: subendocardial thickening and fibrosis reproducing the shape of the cannula; grey arrowheads: confluent, yet discontinue ecchymotic lesions ascribable to radiofrequency ablation lesions. (B) Electroanatomic internal view of the ablation sites. (C–F) Histologic preparations of the ablation sites and surrounding tissue (hematoxylin and eosin). (C) Endocardial disruption with underneath coagulative necrosis of myocardial cells and granulation tissue characterizing an ablation site; adjacent dense fibrotic tissue; vital cardiomyocytes visible at the upper right site of the picture (25 × magnification). (D) A magnified image from C, showing macrophage infiltration at the edge of necrotic myocardial tissue (100 × magnification). (E) Macrophage infiltration at the interface between viable myocardium and fibrotic tissue (100 × magnification). (F) Macrophage infiltration network interspersed into fibrotic reparative tissue (400 × magnification). E: endocardial layer. C: coagulative necrosis of myocardial cells. G: granulation tissue. F: fibrotic reparative tissue. VC: vital cardiomyocytes. M: macrophage infiltration.

set up as soon as the VT initially appeared, as the dense fibrotic network needs about 8 weeks to be completed [3]. Conversely, the presence of macrophage infiltration demonstrates that some tissue injury occurred within 3 weeks [4] before the transplant, well beyond the RF tissue lesions had been placed. Therefore, it is conceivable that a repetitive physical damage caused by the LVAD cannula perpetuated a tissue injury, triggering an inflammatory mediated cellular infiltration.

The clinical aims of the procedure did not give us the opportunity to perform a rigorous evaluation of the electrophysiologic mechanism of the VT. However, the mechanism of induction, the acute success of endocardial ablation, and the pathological evidence strongly suggest that the VT substrate was in the subendocardium.

Although we cannot exclude a localized structural or functional reentry, observations from the intracavitary signals and the time course of the arrhythmia are in favor of a focal origin. In our opinion, the most likely mechanism was arrhythmogenesis associated with tissue inflammation.

Catheter ablation of VT seems promising in LVAD patients [5], even in cases of suction-related arrhythmias [6]. Our report reinforces the evidence that ablation of a mechanical contact site may be safe and effective to prevent arrhythmias recurrences. However, the histologic feature of the lesion, RF power dispersion during ablation, as well as ongoing changes of anatomical contact sites following LV unload and reverse remodeling might affect the long-term efficacy of the procedure. Whether repeated procedures would be able to ensure more reliable results in the medium term should be hopefully proven in the future.

Conflict of Interest

Authors declare no conflict of interest.

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