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Surgical linear ablation for ventricular tachycardia with postinfarction ventricular aneurysm

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ABSTRACT

Background: Left ventricular aneurysm (LVA) might be an arrhythmogenic substrate. Endocardiectomy and cryoablation for ventricular tachycardia (VT) with LVA can cause extensive myocardial damage. We aimed to evaluate the feasibility of surgical radial linear ablation for VT with LVA guided by electrophysiological mapping.

Materials and methods: Porcine models of VT with LVA were developed. Endocardial and epicardial substrate mapping during sinus rhythm were performed under thoracotomy. Surgical radial linear ablation was achieved by a bipolar radiofrequency ablation device. Outcomes, including procedural success and acute freedom of VT, were analyzed.

Results: Fifteen of 20 pigs developed LVA in a 6-wk survival period. A total of 28 sustained monomorphic VTs were initiated in 13 of 15 pigs (86.67%). The number of potential points captured from the endocardium and epicardium were 319 ± 45 and 358 ± 52 per animal, respectively. The ablative targets containing abnormal potentials were located largely on the border zone of LVA. Eight linear lesions from core to border zone of LVA were achieved per animal in a radial and even manner continuously, and ablation was repeated three times to transect border zone. The acute freedom of VT was 84.62%, $P < 0.05$.

Conclusions: Surgical linear endo-epicardial ablation seemed to be feasible in a porcine model with VT and LVA.

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Introduction

Ventricular tachycardia (VT) is a commonly adverse factor in patients with a postinfarction left ventricular aneurysm (LVA).[1] Mapping-guided catheter ablation is an option for scar-related VT, and linear ablation[2] and endo-epicardial ablation[3] has been proven to improve VT ablation prognosis. However, these catheter ablation techniques are challenging for applying to VT with postinfarction LVA, because LVA has complicated arrhythmogenic substrates with VT, probably presenting hemodynamic instability, noninducibility, or multiple morphology.[4,5] Furthermore, additional LVA repair and coronary artery bypass grafting (CABG) are commonly required for most patients with postinfarction LVA and VT.

Surgery, including endocardiectomy and cryoablation, CABG, and LVA resection is considered a valid treatment for LVA and VT. Although this scar resection and surgical ablation has excellent results, with more than 90% long-term freedom from VT,[6] clinical application of this procedure was limited, due to its associated extra myocardial damage and high perioperative mortality.[7-9]

Off-pump CABG and LVA repair without ventriculotomy is a less invasive procedure with good outcomes.[10] To reduce myocardial injury during surgical VT ablation, we hypothesized that radial linear endo-epicardial radiofrequency ablation for VT with LVA is feasible during off-pump CABG and LVA repair.

Therefore, we developed human-like models of VT with LVA and sought to assess the feasibility of off-pump radial linear endo-epicardial radiofrequency ablation for VT with LVA under thoracotomy. These swine models may contribute to improved methodologies of surgical treatment for LVA and VT in the pre-clinical setting.

Methods

The experimental protocol was approved by the Animal Care and Use Committee of Beijing Anzhen Hospital, Capital Medical University. All animals were given human-like care and were performed according to the institutional guidelines (NIH Publications No. 8023, revised 1978).

Developing swine model with LVA

As previously described, a minimally invasive ligation of coronary arteries through the left chest was performed in 35- to 45-kg pigs.[11] After an overnight fast, sedation was initiated with intramuscular injection of 0.25 mL/kg xylazine and 0.5-mg atropine. Tracheal intubation was then performed with ventilation. General anesthesia was maintained with inhaled 1.5%-2.5% isoflurane. Hemodynamic signs including heart rate, electrocardiograph, arterial blood pressure, and oxygen saturation were continuously monitored. The heart was revealed by pericardiotomy. Using the whole length of the artery as a coordinate, the left anterior descending artery, posterior descending artery, and diagonal and obtuse marginal branches were ligated totally and permanently at one-third of the distance from the apex, respectively (anatomical location by a trisection suture expanding from onset of the target artery to the apex). Before ligating coronary arteries, pretreatment with intravenous 100 mg bolus injection and ensuing 1 mg/min continuous drip lidocaine via an auricularly venous access was conducted to reduce the incidence of malignant ventricular arrhythmias. After extubation, the pigs were observed and monitored for 1-3 h until they could ambulate freely. After a 6- wk period following acute myocardial infarction, the morphology and function of the surviving pigs' left ventricles (LVs) were assessed by left ventriculography (LVG) and transthoracic echocardiography.

LVA was diagnosed angiographically if all the following three criteria were found: (1) protrusion of the involved segment, displaying either akinetic or dyskinetic motion; (2) absence of trabeculation in the involved segment; and (3) well-defined demarcation of the infarcted segment according to the Coronary Artery Surgery Study protocol.[12]

After the end of LVG, the pigs with postinfarction LVA underwent median sternotomy and pericardiotomy following electrophysiological study.

Electroanatomic reconstruction of LV

The 8F and 6F intravascular sheaths were placed in the femoral artery and vein, respectively, and 5000 units of unfractionated heparin were administered via the venous sheath to maintain an activated clotting time of 250–350 s. Then, three-dimensional (3D) electroanatomic mapping was performed using the Carto3 system (Biosense Webster, Johnson & Johnson, Diamond Bar, CA) with an 8F 3.5 mm irrigated-tip catheter (Navistar-ST, Biosense Webster, Johnson & Johnson).

Endocardial maps were made with bipolar electrograms (EGMs) filtered at 10–400 Hz and displayed at a sweep speed of 100 mm/s. The validated bipolar voltage (BV) cut-off values of $<$0.5 mV (dense scar), 0.5–1.5 mV (border zone), and $>$1.5 mV (normal tissue) were used for mapping.[13] Using a fill threshold $<$10 mm, sufficient mapping density was achieved, allowing for accurate electroanatomic reconstruction.

Inducing VT

After LV reconstruction, programmed electrical stimulation (PES) was performed in pigs with LVA. A 6 F quadrupole catheter (Bard EP, Lowell, MA) was placed in the apex of the right ventricle (RV) or the right ventricular outflow tract (RVOT) to allow recording and pacing.

PES was first performed from the RV apex. The stimulated protocol included pacing at basic cycle length (400-500 ms) with up to three extrastimuli, until pacing decreased to the refractory period using 10 mA current strength and 2 ms pulse width, burst pacing, and intravenous isoproterenol (up to 5 μg/min).[14] Sustained VT is defined as tachycardia lasting more than 30 s or requiring countershock due to hemodynamic intolerance.[15] If RV apex stimulation failed to induce VT, the same protocol was repeated from the RVOT. If VT was hemodynamically nontolerated, it was terminated by pacing or electrical cardioversion.