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Concomitant Right Ventricular Outflow Tract Cryoablation during Pulmonary Valve Replacement in a Patient with Tetralogy of Fallot

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A 38-year-old female patient with a history of tetralogy of Fallot repair at 10 years of age underwent pulmonary valve replacement with a mechanical prosthesis, tricuspid annuloplasty, and right ventricular outflow tract cryoablation due to pulmonary regurgitation, tricuspid regurgitation, and multiple premature ventricular contractions with sustained ventricular tachycardia. After surgery, she had an uneventful postoperative course with arrhythmia monitoring. She was discharged without incident, and a follow-up Holter examination showed a decrease in the number of ventricular ectopic beats from 702 to 41.

Key words: 1. Right ventricular outflow tract
2. Pulmonary valve replacement
3. Cryosurgery

Case report

A 38-year-old female patient with a history of tetralogy of Fallot (TOF) repair at 10 years of age presented with arrhythmia. Electrocardiography showed atrial fibrillation with a QRS duration of 106 ms. Holter monitoring indicated 702 ventricular ectopic beats and 633 supraventricular ectopic beats, each representing 1% of the total QRS complexes. Of the 702 ventricular ectopic beats, there were 276 (39.3%) bigeminal cycles (Fig. 1A). Echocardiography revealed moderate pulmonary insufficiency, tricuspid regurgitation, and right atrial dilatation with decreased systolic function and a left ventricular ejection fraction of 45%. A cardiopulmonary exercise test revealed a decreased maximum oxygen consumption (VO_{2max}) of 23.6 mL/kg/min. Computed tomographic imaging showed right ventricular enlargement with a right ventricular end-diastolic volume index of 216.5 mL/m^2. Thus, we determined that pulmonary valve replacement (PVR), tricuspid valve repair, and arrhythmia surgery were necessary. An electrophysiological study was not performed because of an unstable heart rhythm, mainly pause-dependent torsades de pointes; therefore, an emergency operation was required. A maze procedure was not planned due to advice of arrhythmia part because of chronic atrial fibrillation, and right ventricular outflow tract (RVOT) ablation was planned because it can help reduce ventricular arrhythmia. The surgical approach

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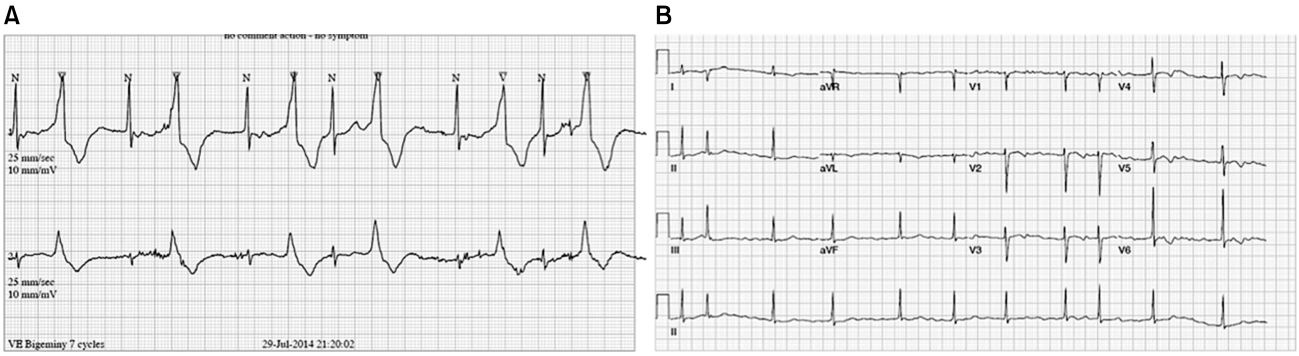

Fig. 1. (A) Preoperative EKG shows multiple bigeminal cycles, and (B) postoperative EKG shows atrial fibrillation with a narrow QRS. EKG, electrocardiography.

was via median sternotomy, during which there were multiple premature ventricular contractions with sustained ventricular tachycardia that required lidocaine injection. During cardiopulmonary bypass, a meticulous dissection was performed. After aortic cross-clamping, right atriotomy and tricuspid annuloplasty were performed using the DeVega method to correct moderate tricuspid regurgitation. Then, the aortic cross-clamp was released, and a vertical incision was made in the main pulmonary artery with the heart contracting. After the previous thickened bovine pericardium was detached, RVOT cryoablation was performed using a Frigitronics (Cooper Surgical Inc., Trumbull, CT, USA) cryoprobe (Fig. 2). A cryoablation lesion was located around the RVOT-free wall margin and near the ventricular septal defect patch repair site, including the pulmonary annulus. The cryoablation was carried out for 60 seconds at $-60^{\circ}C$ with a 15-mm probe. After RVOT cryoablation, PVR with an On-X 25 mm (On-X Life Technologies Inc., Austin, TX, USA) and left pulmonary angioplasty for the correction of left pulmonary artery stenosis and kinking were performed. The durations of cardiopulmonary bypass and aortic cross-clamping were 265 minutes and 37 minutes, respectively. The patient had an uneventful postoperative course with arrhythmia monitoring and was discharged on postoperative day 30. A follow-up Holter examination 28 days after surgery revealed 41 ventricular ectopic beats (<1% of the total QRS complexes) and no supraventricular ectopic beats. A follow-up electrocardiogram 14 months later showed atrial fibrillation with a decreased QRS duration of 92 ms (Fig. 1B). The patient was well at the most recent follow-up, with no signs of dyspnea or chest discomfort.

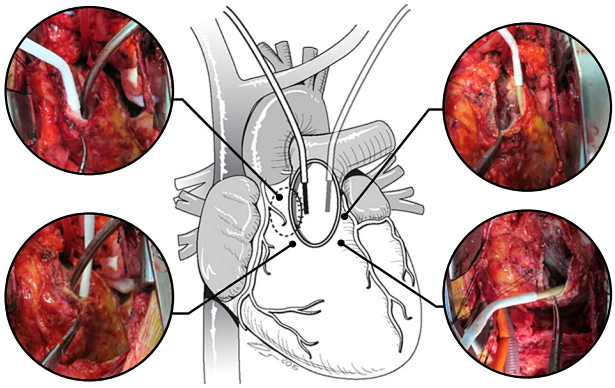

Fig. 2. The cryoablation lesion was located around the previous suture margin of the bovine pericardium, consisting of the right ventricular free wall, excluding the main pulmonary artery tissue. Another cryoablation was performed vertically from the ventricular septum next to the ventricular septal defect patch to the pulmonary annulus. Cryoablation was performed for 60 seconds at $-60^{\circ}C$ with a 15-mm cryoprobe.

Discussion

Pulmonary insufficiency after surgical or interventional relief of pulmonary stenosis can lead to progressive right ventricular dilatation, arrhythmia, exercise intolerance, or even sudden death. If the duration of pulmonary regurgitation is prolonged, ventricular arrhythmia usually develops; however, the evaluation and management of ventricular arrhythmia is not currently standardized. TOF patients with a severely prolonged QRS duration (>180 ms) are at risk for adverse events [1], but other factors related to ventricular arrhythmia are not well known. Pulmo-

nary insufficiency after TOF repair, loading volume, and right ventricular dysfunction often lead to the development of ventricular arrhythmia; thus, PVR itself can reduce the incidence of ventricular arrhythmia.

However, some patients have poor outcomes even after PVR [2]. At present, electophysiologic studies including intracardiac mapping and ablation have confirmed that ventricular tachycardia is typically of a macro-re-entrant nature, using cardiac valves and ventricular scars as boundaries [3,4]. Therefore, surgical ablation of the RVOT is a logical approach for isolating diseased substrates [2,5,6]. On the basis of this knowledge, we performed concomitant PVR and RVOT cryoablation. Although RVOT cryoablation has been used since the early 2000s [7], there are no standard surgical ablation guidelines. A major concern with the cryoablation procedure is the conduction block. Additionally, if the lesion is not completely ablated, a remnant lesion can serve as a substrate, resulting in slow cardiac conduction and re-entrant arrhythmia. In such cases, we expect the RVOT suture margin and the ventricular septal defect patch repair site to cause arrhythmia, and these sites should therefore be ablated. Whether this patient received a rhythmic advantage from PVR, which reduces the hemodynamic burden, or RVOT cryoablation, which eliminated the arrhythmic substrate, is still to be determined. However, surgical cryoablation of the RVOT itself appears to be safe and reduces the risk of arrhythmic events after PVR.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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