Polymorphic ventricular arrhythmia triggered by temporary epicardial right ventricular stimulation after cardiac surgery

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Introduction

Temporary epicardial pacing is commonly used for rhythmic support in the acute postoperative period after cardiac surgery. Clinical studies suggest that pacing in the left ventricular epicardium during resynchronization therapy may be proarrhythmic in a minority of patients. The epicardial pacing increases transmural heterogeneity of repolarization. This could prolong the QT and JT intervals on the electrocardiogram (ECG), with subsequent proarrhythmic effects.

The following case describes polymorphic ventricular arrhythmias after temporary epicardial ventricular stimulation.

Case report

A 76-year-old woman with a history of severe degenerative mitral valve regurgitation was admitted for mitral valve replacement. She previously underwent an implant of a VVIR pacemaker with an endocardial right ventricular lead for chronic atrial fibrillation and complete atrioventricular block (Figure 1A and B). The transthoracic echocardiography showed severe tricuspid regurgitation, severe pulmonary arterial hypertension, and preserved left ventricular ejection fraction. She had no coronary artery disease. In the operating room, it is a common practice to program the device in VOO mode for pacemaker-dependent patients. When extracorporeal support starts, the device is switched to OOO mode, since risks from electrocautery or from eventual cardioversion are not precluded.

Tricuspid valve annuloplasty together with mitral valve surgery was performed. A temporary epicardial pacing lead was placed in the right ventricular epicardium and immediately initiated after the cardiac surgery was complete (Figure 1C). Before chest closure, the patient developed ventricular fibrillation preceded by frequent premature ventricular complexes (PVCs) falling on the T wave; it was treated by external defibrillation. At that time, both the electrolyte levels and acid–base balance were within the allowable range.

The patient was transferred to the postoperative care unit. The ECG showed ventricular pacing with a corrected QT interval of 646 ms (Bazett) (Figure 1D). She was not under any prolonging QT drug. Forty-eight hours later she experienced another torsades de pointes (TdP) episode preceded by R-on-T PVCs (Figure 1E). She was given an intravenous loading dose of amiodarone followed by a continuous infusion. A few days after the cardiac surgery, she suddenly developed an episode of polymorphic ventricular tachycardia requiring an external shock and she then had 2 more episodes of polymorphic ventricular fibrillation requiring more external cardioversion (Figure 1F). Repeated laboratory tests showed normal values for electrolyte levels.

After those episodes, the permanent endocardial pacemaker was switched on, amiodarone was discontinued, and a low dose of beta-blockers was initiated. During endocardial stimulation, ECG showed a normal corrected QT interval (480 ms, Bazett) (Figure 1B). Once epicardial pacing was turned off, the patient remained asymptomatic with no more episodes of ventricular arrhythmias.

Discussion

To our knowledge, this is the first case describing ventricular arrhythmias triggered by temporary epicardial stimulation. Under baseline conditions and during depolarization, the impulse is carried from endocardium to epicardium, but the first structure to repolarize is the last to depolarize. The early repolarization of epicardium provokes a T wave displaying the same polarity as that of the QRS. Therefore, the time interval between the peak and the end of the T wave represents the dispersion of repolarization across the ventricular wall.

There is clinical and experimental evidence that a pacing site–dependent change in ventricular activation may lead to an augmentation of transmural heterogeneity of repolarization,

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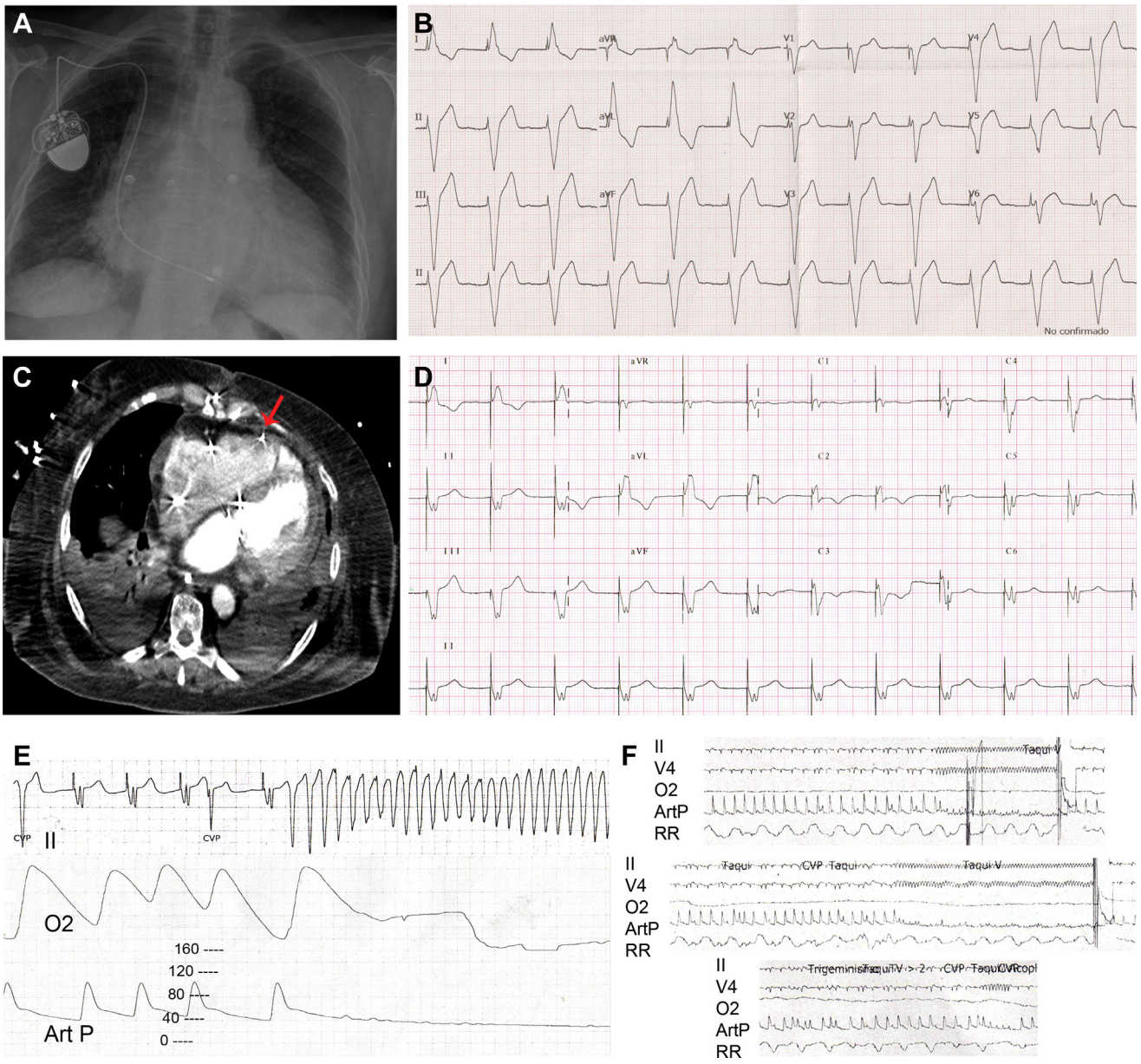

Figure 1 **A:** Chest radiograph shows transvenous endocardial pacing electrode into the right ventricle and single-chamber pacemaker. **B:** Electrocardiogram (ECG) illustrates wide QRS complexes with left bundle branch block morphology, indicating the stimulation from the right ventricle, with a corrected QT interval of 480 ms. **C:** Chest computed tomography scan. Red arrow indicates the temporary epicardial lead in the right ventricle free wall. In this slice we can also see the definitive pacemaker lead across the right atrium and the mitral prosthesis and tricuspid ring. **D:** During temporary epicardial stimulation, the ECG shows a more prominent R wave in V1-V2 derivations and a corrected QT interval of 646 ms. **E:** The external telemetry system illustrates early afterdepolarizations, manifested as short-coupled premature ventricular contractions with "R on T" phenomenon, causing torsades de pointes with immediate loss of pulse and respiratory arrest. **F:** Reiterative torsades de pointes episodes were recorded at 24 hours heart rhythm monitoring. Art P = arterial pressure; RR = respiratory rate.

prolonging the QT interval, which is associated with an increased risk of TdP and sudden cardiac death.[1,2] Early afterdepolarizations can be seen on the ECG as tall U waves; if these reach threshold amplitude they may manifest as PVCs, being an indicator for imminent risk of TdP.[3]

The proarrhythmic effect of biventricular pacing in patients without prior history of ventricular arrhythmias, as in this case, is considered when it occurs in the first month after the implant. However, most of the cases present within the first hours or the same day.[4,5]

Distinct mechanisms have been proposed to explain the proarrhythmic effect of cardiac resynchronization through left ventricular epicardial pacing. Reentrant ventricular arrhythmias can be favored by the left ventricular lead positioned within an epicardial scar[1]; those cases are well managed with epicardial ablation allowing the maintenance of biventricular pacing.[5] On the other hand, the change of the activation pattern from epicardium to endocardium may alter the transmural dispersion in the repolarization pattern and prolong the QT and JT intervals on the

KEY TEACHING POINTS

- The proarrhythmic effect of epicardial pacing is rare but needs to be taken into consideration, in the setting of either monomorphic reentrant tachycardia or polymorphic arrhythmias, depending on the clinical situation.
- Short-coupled premature ventricular complexes are the manifestation of early afterdepolarization in patients predisposed to QT prolongation and may lead to torsades de pointes.
- Amiodarone is the first-line antiarrhythmic drug in structural heart disease, but its proarrhythmic effects are usually underestimated, particularly in women and in the presence of concomitant factors that could prolong the QT interval.

ECG, posing a risk for polymorphic ventricular arrhythmias.[2]

Both situations are very rare and, consequently, difficult to prevent or suspect. The management depends on the causal mechanism but, if resynchronization is not the target and when another option for pacing is available, the QT prolongation effect of the epicardial pacing needs to be taken into consideration, especially in the subset of patients with nonischemic etiology or under prolonging QT drugs.

We must consider that the most important principle in the treatment of TdP is to recognize the arrhythmia. All prolonging QT drugs, such as amiodarone (which was unfortunately the first antiarrhythmic drug selected in our case), should be withdrawn and even modest hypokalemia should be corrected. Moreover, shortening the action potential will prevent both early afterdepolarizations and triggered activity. The most effective way to do this is cardiac pacing at 90–100 beats/min. A transvenous temporary lead should be considered to increase heart rate. Isoproterenol can also be effective if pacing is not available. Furthermore, aggressive magnesium supplementation and even beta-blockers, if atrioventricular conduction is preserved or the rhythm is paced, could eliminate triggered activity, preventing recurrences of TdP in most patients.

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