Left ventricular (LV) myocardial perforation is a rare complication following ventricular tachycardia (VT) ablation with radiofrequency (RF); this complication should be diagnosed and treated promptly. LV free wall rupture after elective RF ablation for sustained VT refractory to medical treatment is rarely reported in the medical literature. Herein we discuss an interesting case which contributes to the ongoing literature, regarding a patient who developed LV perforation due to RF ablation for VT which was resistant to pharmacotherapy and repeated cardioversion attempts after acute myocardial infarction.

**Key Words:** Cardiac perforation • Catheter ablation • Left ventricle • Ventricular tachycardia

**INTRODUCTION**

Catheter-based radiofrequency (RF) ablation procedure has been increasingly used by medical professionals in surgical facilities for the treatment of ventricular tachycardia (VT).\(^1\) RF ablation performed for VT refractory to medical treatment following acute myocardial infarction (MI) is a new technique. Catheter ablation can cause complications such as atrioventricular block, right atrial thrombus, pericarditis, coronary spasm, and cardiac tamponade.\(^2\) Here we have reported a case who developed left ventricular (LV) perforation through RF ablation for the VT, which was resistant to pharmacotherapy and repeated cardioversion after acute MI.

**CASE REPORT**

A 45-year-old male without a relevant cardiac history presented to the emergency department complaining of chest pain three hours in duration. The patient’s physical examination revealed a blood pressure of 110/70 mmHg and normal heart sounds, and his admission electrocardiogram (ECG) indicated acute inferior MI. He underwent successful primary percutaneous coronary intervention via stenting of the totally occluded left circumflex artery (LCX) (Figure 1A-B).

On hospital day four, the patient developed a sudden onset of respiratory distress, palpitation and chest pain. His electrocardiogram showed ventricular tachycardia, and VT persisted despite continued antiarrhythmic drug (intravenous amiodarone and lidocaine); therefore, direct current (DC) cardioversion with 50 joules under sedation was performed and tachycardia was terminated. Following coronary angiography (CAG) emergency CAG, 70% stenosis was noted in the LCX proximal to the implanted stent, which was successfully treated by placement of a bare metal stent. On the 7th
day of hospitalization, the patient re-experienced incessant VT (Figure 1D) under IV antiarrhythmic drugs that persisted despite DC cardioversion and overdrive pacing via temporary pacemaker. During the VT period, the patient’s arterial blood pressure was almost 102/56 mmHg. Additionally, transthoracic echocardiography (TTE) showed LV segmentary hypokinesia compatible with MI without pericardial effusion. Repeat CAG revealed patent LCX artery stents (Figure 1C).

The patient was referred to the electrophysiology laboratory for catheter-based RF ablation for incessant VT. Thereafter, a 7F electrode catheter was positioned at the His bundle area through the left femoral vein. Subsequently, another 7F electrode catheter was introduced percutaneously through the left femoral vein and advanced into the coronary sinus. Left femoral arterial access was obtained to record arterial blood pressure and provide access for 7F multi-curve ablation catheter with a 4-mm tip (RF Marin®; Medtronic, Inc., Minneapolis, MN, USA) introduced retrogradely into the LV. Systemic heparin was given with an activated clotting time ranging from 250-350 seconds. Ablation was aided by electrogram characteristics, activation mapping and entrainment mapping; unfortunately, 3D electroanatomic mapping system could not be used due to hospital equipment deficiency. RF ablation consisted of a max heat of 60°C, mean strength of 15 watts (max 50 watt), and impedance 109 Ω (Ohm) was performed to the LV posterior wall. RF current was applied repeatedly for ten applications, which took a total of six minutes. The patient’s tachycardia rate slowed after RF ablation, but was not terminated. The ablation procedure was terminated without performing DC shock, after which our patient was normotensive with a rhythm of slow VT. Overall, the total procedure time was 150 minutes (the exact fluoroscopy time was 50 minutes). The patient was subsequently transferred to the intensive care unit for electrocardiographic and hemodynamic monitoring.

Due to the persistence of slow VT, repeated cardioversion with 50 joules was performed, and tachycardia

Figure 1. Coronary angiography revealed total occlusion of the left circumflex coronary (LCX) artery at midportion, and diffusely stenotic right coronary artery and normal left anterior descending artery (A, B). Earlier radiofrequency ablation coronary angiography revealed LCX artery stents were patent (C). Electrocardiogram (ECG) showed ventricular tachycardia (D).
was terminated (Figure 2A). Post-cardioversion, the patient became hypotensive with SBP of 70 mmHg 45 minutes after the procedure. Thus, IV fluids and vasopressor agents were administered, yet the patient’s hypotension persisted. TTE revealed approximately 18 mm pericardial effusion (Figure 2B), with collapse of the right heart suggesting possible perforation of the ventricle due to ablation procedure. Protamine was given intravenously. Following emergency cardiovascular surgery consultation, a pericardial drain was placed via the subxiphoid approach before transfer to the operating room, but only 100 cc of blood could be withdrawn without any improvement in hemodynamics. Insufficient withdrawal was thought to be secondary to clotting. In addition, pericardiocentesis was not preferred due to the emergency surgical intervention. Thereafter, emergency surgery with median sternotomy was performed, wherein the pericardium was opened revealing a large volume of hemorrhagic fluid inside the pericardial cavity. After removal of 500 cc hemorrhagic fluid, the perforation site with active bleeding was discovered at the posterior wall of the LV (Figure 2C); the perforation site was closed with pledgeted sutures. After 12 hours, the patient was successfully extubated and mediastinal chest tubes were removed on post-operative day 2. Throughout the entire post-operative follow-up, VT episode was not reported under beta-blocker treatment. Ultimately, the patient was discharged on the 12th day.

**DISCUSSION**

Radiofrequency catheter ablation is an emerging treatment modality used for patients with VT, especially for those resistant to antiarrhythmic therapy, in cases of generally nonacute coronary syndromes. In our situation, RF ablation was used as a last treatment option,

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**Figure 2.** Post-ablation and post-cardioversion electrocardiogram (ECG) (A). About 18 mm pericardial effusion near right ventricle in transthoracic echocardiography (B). Perforation site in left ventricle posterior wall (C).
due to VT resistance to intensive antiarrhythmic therapy, and DC cardioversion in patients with acute myocardial infarction.\textsuperscript{3}

A steam pop, which can be described as an audible pop generated by the mini-explosion of a gas bubble, occurs during RF catheter ablation, when the subendocardial tissue temperature increases over the boiling point, leading the blood to vaporize. An RF temperature control mode is utilized and titrated to an electrode temperature based on the catheter used, or decreased impedance between 10-15\textsuperscript{\textOmega} is created to avoid excessive tissue damage. With an irrigated-tipped catheter, the aim is to minimize steam pop occurrence by terminating RF energy delivery at the time of sudden catheter tip temperature increase or impedance decrease.\textsuperscript{3,5} Tissue damage arises from the pathophysiology of cardiac tamponade after a steam pop. In our case, we didn’t observe any dramatic fluctuations in both impedance and tip temperature levels.

Cardiac tamponade is a rare complication following RF ablation. During post-ablation follow-up when cardiac tamponade is suspected, TTE should be performed for immediate diagnosis. If pericardial bleeding continues after initial pericardiocentesis, the patient should be urgently moved to the operating room for surgical repair. Even if the pericardial bleeding has stopped after pericardial drainage, the pericardial catheter should be left in place until the clinical situation of the patient has stabilized or no significant residual effusion has been observed via TTE follow-up.\textsuperscript{4} In our case, as there was not any improvement in the patient’s clinical situation after pericardiocentesis, and he was urgently transferred to the operating room.

In reported cases, the majority of perforations happened during either RF ablation or even placement of diagnostic catheters at the right ventricle, especially at the outflow tract. It is rare to see perforation at the LV because of heavy muscle character.\textsuperscript{4}

In this case, we identified that the first LV perforation developed through ablation for the VT, which was resistant to pharmacotherapy and cardioversion after acute MI. Ultimately, clinicians should proceed with caution to avoid LV perforation due to ablation execution in patients experiencing ventricle thickness decrement and fragility in the acute phase of MI.

REFERENCES