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CASE REPORT
Inherited cardiac conditions/cardiomyopathies

Two severe complications post-percutaneous intramyocardial septal radiofrequency ablation in a patient with failed alcohol septal ablation: pulseless electrical activity cardiac arrest and pericardial tamponade—a case report

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Background Alcohol septal ablation (ASA) can be recommended for patients with drug-refractory hypertrophic obstructive cardiomyopathy (HOCM). Recently, percutaneous intramyocardial septal radiofrequency ablation (PIMSRA) was reported as a safe and effective treatment for HOCM.

Case summary We present a case report of pulseless electrical activity (PEA), cardiac arrest, and pericardial tamponade occurring post-PIMSRA. We performed PIMSRA for the patient with HOCM after failed ASA. Two hours post-PIMSRA, transthoracic echocardiography (TTE) revealed that the hypokinetic basal intraventricular septal (IVS) thickness increased with aggravation of systolic anterior motion of the mitral valve. After the occurrence of subsequent PEA cardiac arrest, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) support was provided. With sinus rhythm restoration and blood pressure stabilization after ECMO removal, the patient had pericardial tamponade on Day 3 post-PIMSRA. After excluding apparent myocardial perforation and draining haemorrhagic effusion under TTE guidance, her symptoms and haemodynamic status improved. She was asymptomatic at her one-year follow-up. The left ventricular outflow tract gradient (LVOTG) at rest and the thickness of the basal IVS reduced to 5 mmHg and 12 mm, respectively.

Discussion We assumed that the main causes of PEA cardiac arrest and pericardial tamponade in our case were ablation-related tissue oedema at the basal IVS and blood leakage possibly related to puncture haemorrhage, respectively. While waiting for myocardial oedema to resolve, ECMO was applied as a bridge-to-recovery therapeutic approach. Pericardiocentesis is a strategy for the emergency drainage of pericardial effusion. It is essential to distinguish life-threatening complications with TTE for management planning post-PIMSRA.

Keywords Case report • Pulseless electrical activity cardiac arrest • Pericardial tamponade • Percutaneous intramyocardial septal radiofrequency ablation

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Learning points

- Post-percutaneous intramyocardial septal radiofrequency ablation, aggravated obstruction of left ventricular outflow tract secondary to IVS oedema and severe systolic anterior movement can cause pulseless electrical activity cardiac arrest, and puncture haemorrhage can cause pericardial tamponade.
- Veno-arterial extracorporeal membrane oxygenation was an effective bridge-to-recovery therapeutic approach, but severe complications occurred as a result of IVS oedema. Routine pericardiocentesis was a successful treatment for pericardial tamponade associated with puncture haemorrhage.

Introduction

Alcohol septal ablation (ASA) is considered the best treatment for drug-refractory hypertrophic obstructive cardiomyopathy (HOCM).[1] Regarding peri-ASA-related complications, conduction abnormalities are common (39.6%), while other complications, including coronary artery dissection, coronary spasm, ventricular fibrillation, cardiac tamponade, pulmonary embolism, and cardiogenic shock, are rare.[1] Recently, a single-centre study demonstrated that transthoracic echocardiography (TTE)-guided percutaneous intramyocardial septal radiofrequency ablation (PIMSRA) was a safe, effective, and invasive treatment for HOCM. The PIMSRA might be an alternative therapeutic option for HOCM patients.[2,3] We present a HOCM patient with a history of ASA failure suffering from pulseless electrical activity (PEA) cardiac arrest and pericardial tamponade post-PIMSRA. Symptoms of both complications were alleviated after effective treatment, and the patient's long-term prognosis improved.

Summary figure

Time	Event
6 months prior to PIMSRA	The patient had underwent ASA.
10 days prior to PIMSRA	The patient complained of increasing dyspnoea that prompted repeat TTE and showed recurrence of left ventricular outflow tract obstruction (LVOTO).
Time to PIMSRA	We performed PIMSRA, and five regions of anterior and posterior IVS were ablated.
3 h post-PIMSRA	Pulseless electrical activity cardiac arrest occurred, and we performed CPR.
4 h post-PIMSRA	Extracorporeal membrane oxygenation was applied.
3 days post-PIMSRA	The obstruction was relieved and we stopped ECMO. 10 h later, pericardial tamponade occurred and then, drainage of the effusion was performed.
15 days after PIMSRA	The patient was discharged.
One-year follow-up	The patient was asymptomatic without obstruction of the LVOT.

Case presentation

A 72-year-old woman with HOCM presented with exertional dyspnoea and syncope that had lasted for 5 years despite receiving beta-blocker therapy. The TTE showed a basal intraventricular septal (IVS) thickness of 17.6 mm and a resting left ventricular outflow tract gradient (LVOTG) of 163 mmHg with systolic anterior movement (SAM) of the mitral valve before septal reduction therapy. Then, ASA was performed due to her rejection of myectomy, but the reduction in the LVOTG was unsatisfactory after ASA (*Figure 1A*).

As a result of failed ASA that was reconfirmed by cardiac magnetic resonance (CMR) and electrocardiogram (ECG) (see Supplementary material online, *Figure S1C* and *D*, *Figure S2A*), the decision was made to proceed to PIMSRA. The PIMSRA was mainly performed according to a previously reported protocol.[2,3] In brief, under real-time TTE guidance, a radiofrequency electrode needle (17G, Cool-tip RF Ablation System and Switching Controller, Medtronic Minimally Invasive Therapies, Minneapolis, MN, USA) was inserted into the basal segment of hypertrophic IVS through the left ventricular (LV) apex via the percutaneous intramyocardial approach. The initial ablation power was initially delivered at 20 W and then gradually increased up to 40 W for 5 min. The ablation needle was then withdrawn by 1 cm to prepare for the next application. From the basal to mid segments, both the hypertrophic anterior and posterior IVS were ablated. The total ablation time was 30 min. The thickness of the basal IVS and LVOTG at rest did not significantly nor quickly decrease at the end of ablation. Her blood pressure dropped precipitously 2 h post-PIMSRA despite receiving active rehydration and intravenous injections of noradrenaline and methylprednisolone. The TTE revealed that the thickness of hypokinetic basal IVS had increased to 21 mm with aggravation of SAM of the mitral valve, and we failed to measure the LVOTG because no blood flow was observed in the left ventricular outflow tract (LVOT). The extreme obstruction in the LVOT caused refractory cardiac shock and the left ventricular ejection fraction (LVEF) to significantly decrease to 30% without pericardial tamponade. The patient became bradycardic with ventricular escape beats and went into PEA cardiac arrest (see Supplementary material online, *Figure S2B*). We performed cardiopulmonary resuscitation (CPR) for 20 min, but the arrest was not resolved. As a bridge-to-recovery approach to stabilize systemic circulation, reduce preloading, and gain time for swollen IVS recovery, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) support was applied. The activated clotting time of whole blood (ACT) was maintained at 160–200 s under the application of heparin. After sinus rhythm restoration and blood pressure stability, ECMO was withdrawn on Day 3 post-PIMSRA. Heparin was discontinued, and the ACT was 123 s. Unfortunately, severe dyspnoea recurred approximately 10 h post-ECMO removal. The TTE showed an increasing pericardial effusion (*Figure 1B*) that confirmed by ECG (see Supplementary material online, *Figure S2C*) while myocardial contrast echocardiography

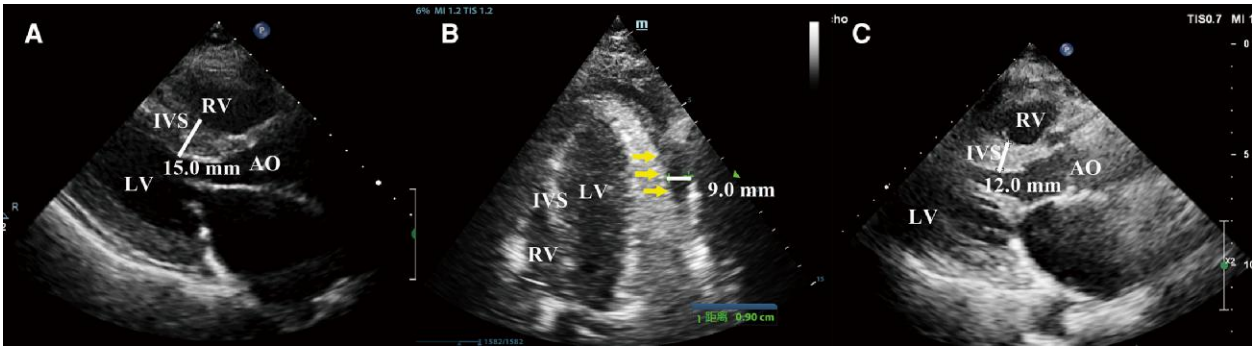

Figure 1 Thickened intraventricular septal (IVS) of 15 mm in diastole and systolic anterior movement (SAM) of the mitral valve was confirmed by transthoracic echocardiogram (TTE) before percutaneous intramyocardial septal radiofrequency ablation (PIMSRA) (*A*). The TTE showed pericardial effusion (up to 9 mm) accumulated behind the left apical lateral ventricle (arrows) (*B*). The IVS thickness and peak left ventricular outflow tract gradient (LVOTG) decreased to 12 mm and 5 mmHg at the one-year follow-up, respectively (*C*). IVS, intraventricular septal; SAM, systolic anterior motion; TTE, transthoracic echocardiogram; PIMSRA, percutaneous intramyocardial septal radiofrequency ablation; LVOTG, left ventricular outflow tract gradient.

(MCE) found no visible perforation foci. After draining 190 mL of haemorrhagic pericardiac effusion, her symptoms and haemodynamic status rapidly improved. The IVS, LVOTG, and LVEF returned to normal 15 days post-PIMSRA. ST-segment elevation and Rv5+Sv1 were reduced at follow-up (see Supplementary material online, *Figure S2D*). She was discharged and prescribed metoprolol succinate 47.5 mg once daily.

During the one-year follow-up, she remained asymptomatic. Her New York Heart Association classification had changed from class III to class I, which was an improvement. The TTE showed that the peak LVOTG and IVS thicknesses were 5 mmHg and 12 mm (*Figure 1C*), respectively. Follow-up CMR was not performed because a steel plate had been implanted in the treatment of an incident spinal fracture.

Discussion

Alcohol septal ablation, as a traditional nonsurgical invasive therapy, is especially suitable for medicine-refractory and surgery-intolerant HOCM patients.[4] However, nearly 20% of patients required reintervention.[1,5] The efficacy of ASA could be limited by the size and distribution of the septal perforator branches. A depot of alcohol may localize on the region not contributing to obstruction.[6] In addition, more severe septal hypertrophy[7] and higher baseline LVOTG[8] may also play a major role in unsuccessful outcome. The reason for ASA failure in this case could be that most alcohol delivery was targeting to the basal right ventricular septal region, while the hypertrophic basal LV septum still encroached into the LVOT (see Supplementary material online, *Figure S1A* and *B*). As an alternative approach for ASA, PIMSRA has been reported to effectively reduce the LVOTG and improve the long-term prognosis of patients with HOCM.[2,3] The 30-day major adverse clinical events included pericardial effusion (9.5%) and death (1%) and may have been due to cardiac shock and malignant arrhythmia.[3]

To our knowledge, this was the first case in which PEA cardiac arrest and pericardial tamponade occurred successively post-PIMSRA. We postulate that PEA cardiac arrest was secondary to cardiac shock caused by aggravation of tissue oedema post-ablation and exacerbated SAM of the mitral valve, as confirmed by TTE. Of note, even though the ablation power used in this case was lower than that used in the standard protocol,[2,3] the ablation still resulted in malignant myocardial tissue oedema. Due to radiofrequency ablation conducting thermal heat to deeper tissue,[9] expansion of oedema could potentially increase the risk for mortality. Considering that the extreme obstruction of LVOT mainly contributed to PEA cardiac arrest and would relieved after myocardial oedema subsides, ECMO should be considered to provide temporary circulation support. Moreover, short-term treatment with glucocorticoids might protect the stability of the vascular endothelial barrier and decrease tissue oedema.[10–12] Regarding delayed pericardial tamponade, the haemorrhagic point was too small to be recognized by MCE or TTE. We assumed that it was transapical puncture haemorrhage, which could be related to anticoagulation during the application of ECMO, myocardial contractility recovery, glucocorticoid administration,[8–10] or myocardial contusions following CPR.[11] Because of its high success rate and low procedural morbidity, pericardiocentesis has become the standard intervention for eliminating pericardial effusion. Therefore, although PIMSRA is expected to become a promising treatment for HOCM patients, a tailored PIMSRA approach should be taken into consideration to improve safety and efficacy. Changes in ablation strategy changes, such as the consideration of changes in impedance, current densities and temperature-controlled mode, replacement of electrical pulse energy, and guidance of intracardiac echocardiography, might help to optimize the PIMSRA procedure.[9,13]

Conclusion

Percutaneous intramyocardial septal radiofrequency ablation may be a new and promising treatment for symptomatic HOCM patients after ASA failure despite the risk of life-threatening complications. Interventional cardiologists should be aware of the possibility of PEA cardiac arrest and tardive pericardial tamponade. Extracorporeal membrane oxygenation and haemorrhagic pericardial effusion drainage are effective treatments. TTE should be routinely performed in the perioperative period.

Lead author biography

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Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports*.

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Data availability

The authors confirm that the data underlying this article are available within the article [and/or its *supplementary material*].

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