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CASE REPORT
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A case report: Amplatzer occluder device closure of an iatrogenic ventricular septal defect following radiofrequency ablation

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Background Interventricular septal perforation is an extremely rare complication of radiofrequency ablation (RFA), with an incidence of 1%. The most common mechanism is a 'steam pop', which can be described as 'mini-explosions' of gas bubbles. Data for percutaneous repair of cardiac perforations due to RFA are limited.

Case summary A 78-year-old female patient was referred to our department for the treatment of two iatrogenic ventricular septal defects (VSDs) following radiofrequency ablation (RFA) of premature ventricular contractions. One week post-ablation, chest pain and progressive dyspnoea occurred. Transthoracic echocardiography detected a VSD, diameter 10 mm. Hence, iatrogenic, RFA-related myocardial injury was considered the most likely cause of VSD, and the patient was referred to our tertiary care centre for surgical repair. Cardiovascular magnetic resonance (CMR) imaging demonstrated border-zone oedema of the VSD only and confirmed the absence of necrotic tissue boundaries, and the patient was deemed suitable for percutaneous device closure. Laevocardiography identified an additional, smaller muscular defect that cannot be explained by analysing the Carto-Map. Both defects could be successfully closed percutaneously using two Amplatzer VSD occluder devices.

Discussion In conclusion, this case demonstrates a successful percutaneous closure of a VSD resulting from RFA using an Amplatzer septal occluder device. CMR might improve tissue characterization of the VSD borders and support the decision if to opt for interventional or surgical closure.

Keywords Radiofrequency ablation • Ventricular septal defect • Occluder device • Premature ventricular contractions

Learning points

- Percutaneous ventricular septal defect closure could be favoured as a successfully treatment for radiofrequency-induced myocardial ablation lesions with basal location and without surrounding necrotic tissue.
- Cardiovascular magnetic resonance can perform myocardial tissue characterization of radiofrequency-induced myocardial defects (malacia, oedema, necrosis etc.) and can assist in the treatment decision regarding surgical vs. percutaneous closure.

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Introduction

Interventricular septal perforation is a potential complication of radiofrequency ablation (RFA), with an incidence of 1% after ventricular RFA.[1] The most common mechanism is a 'steam pop', which can be described as 'mini-explosions' of gas bubbles.[2] Data for percutaneous repair of cardiac perforations due to RFA are limited.

Timeline

Day 0	*Symptoms/medication*: heart failure, paroxysmal atrial fibrillation, anticoagulation with apixaban, arterial hypertension
	Diagnosis: high premature ventricular contraction (PVC) burden and restricted cardiac function
	Treatment: electroanatomical mapping and radiofrequency ablation (RFA) using irrigated tip catheter
Day 3	*Echocardiography*: exclusion of pericardial effusion and good cardiac function
	Discharge at home
Day 7	*Symptoms*: progressive dyspnoea, new cardiac murmer
	Diagnosis: transthoracic echocardiography detected iatrogenic ventricular septal defect (VSD) following RFA
	Treatment: transfer for surgical repair
Day 8	*Diagnosis*: cardiovascular magnetic resonance imaging was performed and indication for percutaneous repair
Day 9	*Treatment*: Laevocardiography identified an additional, smaller muscular defect that cannot be explained by analysing the Carto-Map; successfully percutaneous closure of two iatrogenic VSDs
Day 10	*Echocardiography*: proper position and function of occluder devices, small residual shunt
	Holter-electrocardiogram: isolated PVC, no bundle branch block, intermittent AV- block I°
Day 15	Anticoagulation with Apixaban and discharge on the 6th postinterventional day

Case presentation

A 78-year-old female patient was referred to our department for the treatment of an iatrogenic ventricular septal defect (VSD) following RFA of premature ventricular contractions (PVCs) (*Figure 1*).

RFA of PVCs was conducted due to a high PVC burden considered to represent the cause of new-onset heart failure (New York Heart Association III) with only moderately impaired left ventricular (LV) ejection fraction. The patient was anticoagulated with Apixaban for pre-existing symptomatic paroxysmal drug-refractory atrial fibrillation. Except for arterial hypertension, no further relevant previous diseases are known. Electroanatomical mapping identified clinical PVC origin in the basal right ventricular septum. Both sided endocardial ablation of the interventricular septum was performed with an irrigated tip 4 mm catheter (Smarttouch SF F curve, Biosense Webster, Diamond Bar, CA, USA). Ablation of the basal right ventricular septal region did not abolish PVC occurrence. Still, with continued ablation in the LV basal septal/para-Hisian region, complete PVC elimination could be achieved (*Figure 2*).

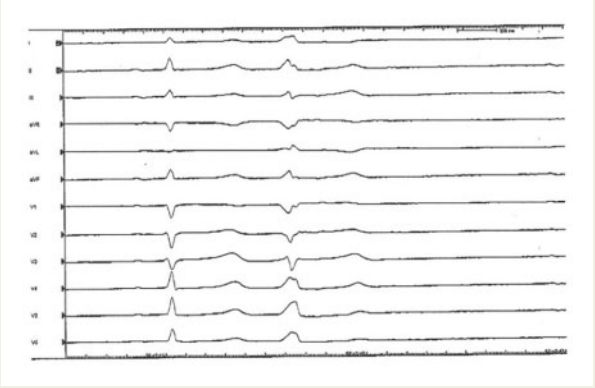


Figure 1 A 12 lead electrocardiogram showing premature ventricular contraction morphology, 100 mm/s.

While ablation of the clinical PVCs was successful, two other PVC morphologies were documented but not considered a suitable ablation target. Consequently, amiodarone was initiated successfully. The intraprocedural course appeared uneventful; no steam pops have been observed. A routine follow-up transthoracic echocardiography 3 days later demonstrated normal systolic LV function and the absence of pericardial effusion.

One week post-ablation, chest pain and progressive dyspnoea occurred, and a 4/6 systolic cardiac murmur was noted. Transthoracic echocardiography detected a VSD, (diameter 10 mm). X-ray coronary angiography excluded coronary arterial disease as a potential ischaemic origin of the VSD. Hence, iatrogenic, RFA-related myocardial injury was considered the most likely cause of VSD. The patient was referred to our tertiary care centre for surgical repair.

Cardiovascular magnetic resonance (CMR) imaging was employed to perform adequate radiofrequency-induced myocardial lesion characterization as part of the pre-procedural diagnostic work-up. CMR-based myocardial tissue characterization of RFA-induced VSDs with detection of myocardial malacia including necrosis, oedema, microvascular obstruction, or intramyocardial haemorrhage can assist in clinical decision making regarding the appropriateness of either percutaneous VSD closure or direct surgical repair. CMR-cine imaging depicted a 10 mm VSD of the basal anteroseptal segment with significant left-to-right shunt (Qp/Qs = 2.1; shunt fraction 52%) (*Figure 3*, *Video 1*); left and right ventricular dimensions and function were normal. The VSD exhibited a marked, semi-lunar appearance on the LV endocardial border site as typically seen following the so-called 'steam pop' phenomenon during high energy radiofrequency delivery, mostly resulting in complete evaporation of myocardial tissue. Besides, CMR imaging demonstrated border-zone oedema of the

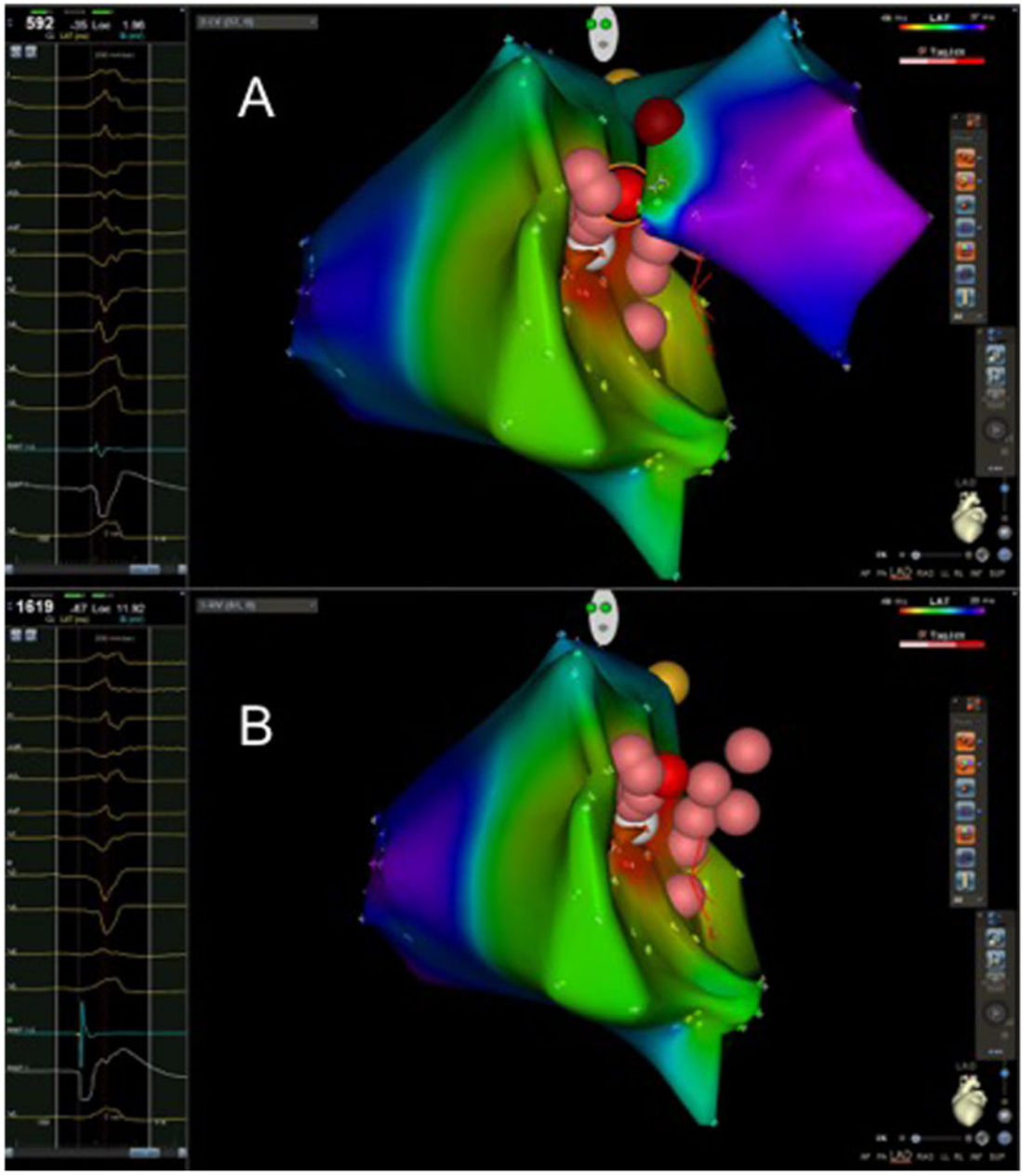


Figure 2 Electro-anatomical map. (*A*) Left anterior oblique position and (*B*) right anterior oblique position.

ventricular defect only and confirmed the absence of necrotic tissue boundaries. The patient was deemed suitable for percutaneous device closure.

Following percutaneous femoral arterial and venous access (7- and 8-Fr catheters, respectively), laevocardiography identified two VSDs: one mid-ventricular defect (diameter 10 mm) and an additional, smaller muscular defect (4 mm; *Figure 4*, please see also *Video 2*); significant left-to-right shunt was demonstrated (Qp/Qs 2,2:1). After the transseptal puncture, an Amplatzer muscular VSD occluder (14 mm) was deployed in the mid-ventricular defect and a second one (12 mm) in the muscular defect. On repeat laevocardiography, only a small residual shunt was recognized (*Video 3*, Supplementary material online, *Figure S5* and *Video S1*). It has to be mentioned that the more apical VSD cannot be explained by analysing the CartoMap. Therefore, it can only be assumed that the second more apical VSD could be pre-existing. No intra- or post-procedural complications were recorded. Because of pre-existing atrial fibrillation, we recommended Apixaban for oral anticoagulation. The patient was discharged 6 days after successful VSD closure in a good clinical condition.

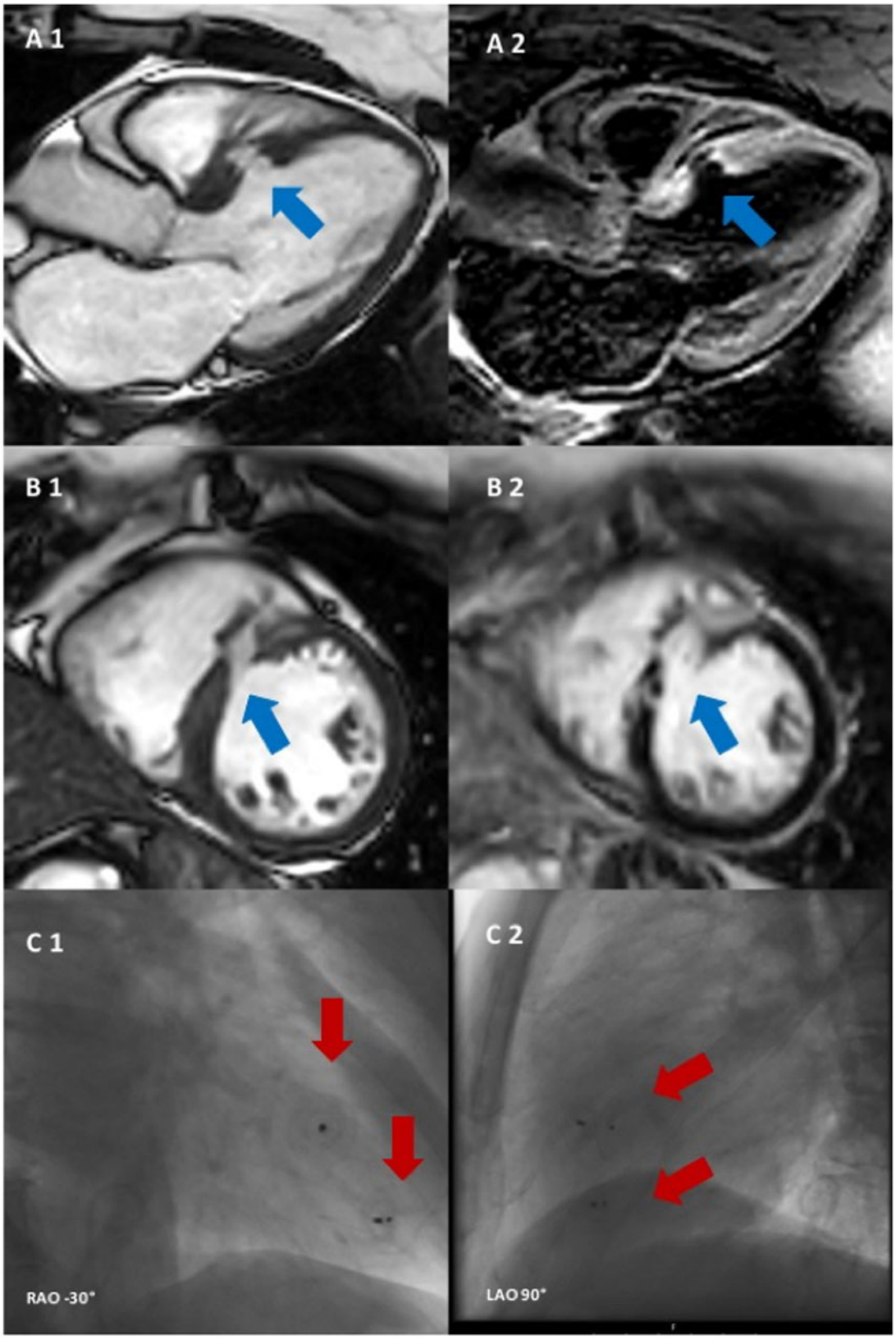


Figure 3 Cardiovascular magnetic resonance imaging (*A*, three-chamber geometry; *B*, short-axis geometry); blue arrows indicate ventricular septal defect location. Cardiovascular magnetic resonance demonstrated a 10 mm ventricular septal defect in the anteroseptal (basal) region (*A1*, cine still frame; *A2*, T2-weighted blackblood turbo-spin echo with ventricular septal defect surrounded by brightly appearing, oedematous myocardial tissue). Conventional X-ray fluoroscopy for demonstration of final occluder device position (red arrows): *C1*, right anterior oblique projection; *C2*, left anterior oblique projection.

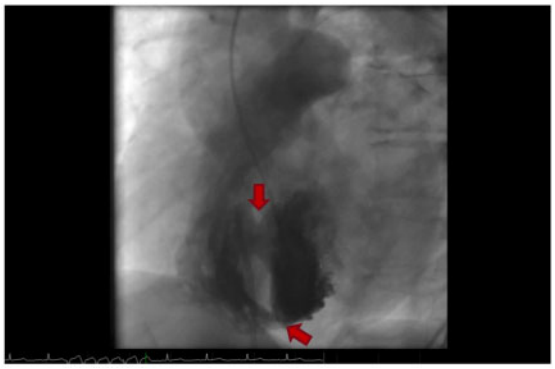
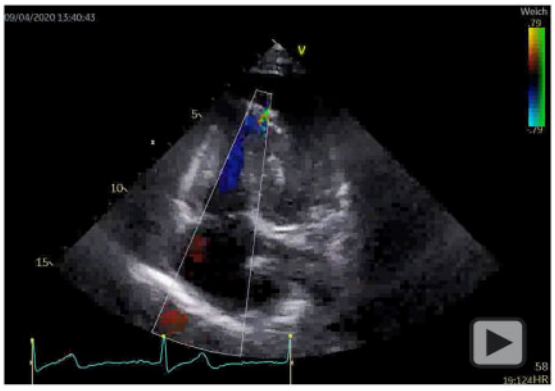
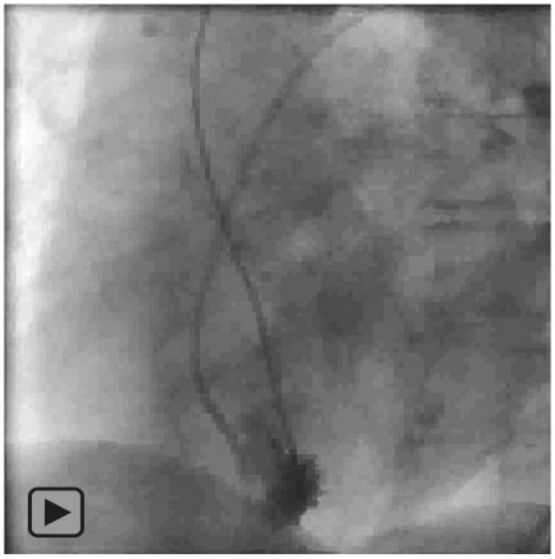


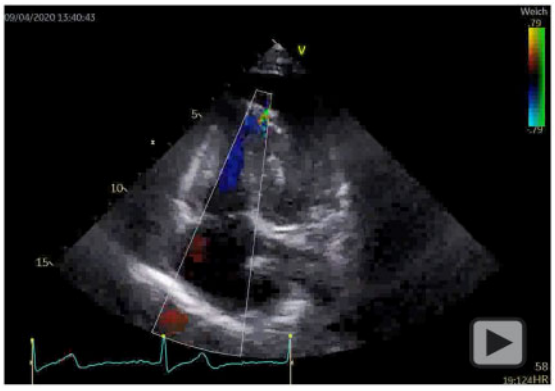
Figure 4 Laevocardiography (still-frame, left anterior oblique projection): the site/location of two ventricular septal defects could clearly be depicted (red arrows).



Video 2 Laevocardiography showing the two ventricular septal defects (left anterior oblique position 60°).



Video 1 Cardiovascular magnetic resonance imaging showing the mid-trabecular ventricular septal defect (three-chamber view and short-axis view).



Video 3 Echocardiography of the mid-ventricular septal occluder after percutaneous repair, modified four-chamber view (apical four-chamber view).

Discussion

Cardiac perforation due to ventricular RFA is reported with an incidence of 1%.[1,3] The power of RFA is particularly limited by the formation of thrombus at the tissue-electrode interface with increasing temperature. Irrigated tip catheters can cool the ablation electrode and thus create larger and deeper lesions with the potential for 'steam pops' that may lead to cardiac perforation.[2,4,5] In a retrospective study by Tokuda *et al.*,[3] more than half of perforations due to RFA were associated with the occurrence of steam pops. Patients received surgical repair in 55% of cases. A retrospective review of Seiler *et al.*[5] also showed a higher risk for cardiac surgery in perforations caused by steam pops, mainly influenced by the position of the VSD.[6]

However, data for percutaneous repair of cardiac perforations due to RFA are limited. Zipse *et al.*[7] reported a male patient with non-ischaemic cardiomyopathy with perforation of the right ventricular free wall and successful defect closure using an Amplatzer septal occluder. In our case, the basal position of the large, most likely ablation-induced VSD and the presence of a clearly marked radiofrequency-induced lesion without surrounding necrotic tissue favoured percutaneous VSD closure.

In conclusion, this case demonstrates a successful percutaneous closure of a VSD resulting from RFA using an Amplatzer septal occluder device. CMR might improve tissue characterization of the VSD borders and support the decision if to opt for interventional or surgical closure.

Lead author biography

Anna Michaelis studied human medicine at the Medical Faculty in Leipzig and successfully completed her studies in 2017. She is currently employed as a resident at the Leipzig Heart Centre department of paediatric cardiology.

Supplementary material

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

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patients in line with COPE guidance.

Conflict of interest: None declared.

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