Constrictive pericarditis following atrial fibrillation ablation – A rare complication

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Introduction
Catheter ablation is a well-established treatment for patients with symptomatic and drug-refractory atrial fibrillation (AF). Over the last years, outcomes have improved, with a concomitant progressive reduction in the complication rates. The updated worldwide survey on the methods, efficacy, and safety of AF ablation between 2003 and 2006 reported a major complication rate of 4.54% with tamponade occurring in 1.31%. In the recent CABANA trial, cardiac tamponade occurred in 0.8%, pericardial effusion in 2.2%, and severe pericardial chest pain in 1.1%. Right-sided heart failure (HF) syndrome and dyspnea following left atrial ablation are rarely reported; therefore, differential diagnosis of these complications is challenging.

Here, we report a case of progressive dyspnea that developed constrictive pericarditis (CP) following catheter ablation of persistent AF.

Case report
A 61-year-old man with no previous comorbidities or history of prior hospitalizations underwent catheter ablation at our hospital because of increasing symptoms of persistent AF, despite antiarrhythmic drug therapy. At the time of ablation, electrocardiogram revealed normal sinus rhythm with mildly dilated left atrium (volume index 29 mL/m²). The patient was on dabigatran 150 mg twice daily and the last dose was withheld before ablation. He successfully underwent pulmonary vein isolation (PVI) along with cavotricuspid isthmus and roofline ablation.

Briefly, electroanatomic mapping data were collected using a 3D mapping system (CARTO3, Biosense Webster, Inc, Diamond Bar, CA). Bipolar voltage mapping during sinus rhythm revealed patchy or low-voltage areas on the roofline using a conventional cut-off criterion (bipolar voltage value <0.5 mV: abnormal myocardial regions; bipolar voltage value <0.2 mV: dense scar). Subsequently, a wide-area circumferential ablation was performed with point-by-point encirclement using a Thermocool SmartTouch irrigated-tip contact force–sensing radiofrequency ablation catheter (Biosense Webster, Inc). The power control mode of 25–35 W was based on the target region of left atrium and was limited to 25 W at the posterior and inferior walls (temperature limit, 43°C; saline irrigation flow rate, 17 mL/min; target force value range, 8–30 g). Automated lesion tagging (VisiTAg, Biosense Webster, Inc) was used to mark the location of each lesion (lesion tag size, 3 mm; catheter position stability: minimum time, 6 s; maximum range, 3 mm; force over time, 30%; minimum contact force, 5 g) (Supplementary Figure 1).

Following bidirectional PVI, roofline ablation was performed given the location of the low voltage beyond PVI. Further, cavotricuspid isthmus line ablation was carried out after a typical atrial flutter was induced. Posttreatment TTE demonstrated resolution of pericardial effusion. The patient was reinitiated on dabigatran 3 hours after the procedure and was discharged on the following day.

Approximately 3 months after the procedure, the patient presented to the emergency department owing to dyspnea. Electrocardiogram revealed low QRS voltage in the limb leads. Further, TTE showed preserved biventricular function with mild pericardial effusion (8 mm). Several examinations were performed for exclusion of phrenic nerve palsy, pulmonary vein stenosis, and anemia. Because of the possibility of effusive pericarditis, the patient was put on colchicine and ibuprofen. Since the effusion was only mild and did not cause any hemodynamic compromise, he remained under oral anti-coagulation.
Meanwhile, there was a progressive worsening of symptoms associated with right HF. TTE performed 9 months post catheter ablation displayed paradoxical interventricular septum movement and restrictive transmitral flow, characteristic of CP (Figure 1). Cardiac magnetic resonance (CMR) showed circumferential pericardial thickening (8 mm) with inspiratory septal flattening and no sign of delayed gadolinium enhancement (DGE) or pericardial calcification (Figure 2). Right heart catheterization (RHC) revealed equalization of the right and left ventricular diastolic filling pressures and a “dip and plateau” sign in the right ventricular pressure tracing (Figure 3), suggestive of CP.

Several months after the diagnosis, and despite the absence of DGE on the CMR, a corticosteroid trial (oral prednisolone 0.5 mg/kg/day) was attempted. Subsequently, the patient showed significant clinical improvement in right HF with no restrictive transmitral flow or abnormal interventricular septum movement as revealed by TTE; therefore, a progressive reduction in corticosteroid therapy was considered. Unfortunately, the patient was rehospitalized with septic arm and knee arthritis owing to the progressive spread of infection, with worsening renal function and deterioration of the functional status. Considering his clinical condition, pericardiectomy was postponed and the patient eventually died 31 months postablation.

Discussion

This case highlights several important facts. First, it highlights the need for a quick differential diagnosis; second, the importance of considering CP as a potential cause of (predominantly right-sided) HF after AF catheter ablation. Finally, it emphasizes the importance of weighing the risks vs benefits of corticosteroid therapy.

Following AF catheter ablation, dyspnea can occur and a differential diagnosis should be promptly carried out.4 In our case, all potential complications related to catheter ablation; only at 3 months postablation did we exclude cardiac tamponade, pulmonary vein stenosis, and phrenic nerve palsy. This delayed investigation led to a late start of anti-inflammatory therapy, allowing persistent pericardium inflammation, which probably contributed to the CP. Although we could speculate that a pericardiocentesis could have been considered 3 months postablation, it would have been difficult to justify it. In fact, the pericardial effusion was only mild, and the patient had no signs of hemodynamic instability. Moreover, the pericardiocentesis would involve a small risk of complications5 and it is not clear that it would have solved the problem.

Even though complications after AF catheter ablation have steadily decreased over the years,2–4 physicians should be aware of possible complications associated with this procedure. CP is a relatively well-documented condition, but reduction in corticosteroid therapy was considered. Unfortunately, the patient was rehospitalized with septic arm and knee arthritis owing to the progressive spread of infection, with worsening renal function and deterioration of the functional status. Considering his clinical condition, pericardiectomy was postponed and the patient eventually died 31 months postablation.

**Discussion**

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**KEY TEACHING POINTS**

- Although constrictive pericarditis is a very rare complication of an atrial fibrillation ablation, it should be included in the differential diagnosis of dyspnea after this procedure.
- Pericardiectomy is the last resort for treating constrictive pericarditis but is associated with a nonnegligible mortality rate.
- Prolonged corticosteroid therapy is an alternative but can be associated with several adverse effects.
its occurrence following AF catheter ablation has rarely been reported. In fact, to the best of our knowledge, there has only been 1 case report describing this complication after AF ablation. We sought to demonstrate that CP can occur after catheter ablation and should be a relevant part of the differential diagnosis for (predominantly right-sided) heart failure after catheter ablation. After suggestive findings in the TTE, confirmation was possible with the CMR and right heart catheterization. The latter may be necessary when noninvasive evaluation is inconclusive. The classic findings characteristic of CP, including near equalization of right and left ventricular diastolic filling pressures, prominent y-descent in the right ventricular pressure tracing, and a “dip and plateau” sign in the right ventricular pressure tracing are likely to be present in restrictive cardiomyopathy. However, a diagnosis of restrictive cardiomyopathy was not a consideration in this case, as there was no such diagnosis prior to the ablation. CMR imaging offers information about cardiac anatomy, including pericardial thickening and calcification and presence of a pericardial effusion. CMR also informs about pericardial-myocardial adherence, enhanced ventricular interaction and respiratory variation in flows across the atrioventricular valves, and the tissue characteristics of the pericardium.

In our case, a late trial of corticosteroid was attempted despite the fact that there was no pericardial DGE, which is
normally present when the constrictive process is more likely to be inflammatory and modifiable with medical therapy. On the other hand, the alternative would be performing a pericardiectomy, which is associated with a significant complication rate and, most importantly, a 1-month mortality rate of 2.5%.

One study including 70 patients with suspected CP found that 25% of the patients could be assigned either to transient CP secondary to acute pericarditis or to effusive CP. The former was managed medically, whereas patients with effusive CP underwent pericardiocentesis. None of the patients underwent pericardiectomy and almost all had resolution of symptoms. Conversely, prolonged treatment with a corticosteroid can cause several adverse effects, particularly owing to their immunosuppressive effect, with renal toxicity, possible reactivation of viral infections, and exacerbation of bacterial infections, as was the case of our patient, which unfortunately culminated in a fatal outcome.

Conclusions
Although CP is a very rare complication of an AF ablation, it should be included in the differential diagnosis of dyspnea after this procedure. Prompt diagnosis is of paramount importance to allow adequate therapy at an earlier stage of this condition.

Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrrc.2019.09.005.