Constrictive pericarditis following atrial fibrillation catheter ablation with cardiac tamponade

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Introduction

Radiofrequency catheter ablation (CA) has become an increasingly common procedure to treat atrial fibrillation (AF) because of the efficacy and safety. In a recent trial, the complication rate has gradually decreased, but cardiac tamponade is still a serious complication, occurring in 0.8%–1.0% of patients.[1–3] Even with a lower rate of procedure-related complications, operators should be familiar with the complications and how to treat them. In the midterm evaluation of patients with CA-related cardiac tamponade, acute pericarditis occurred in 34%–53% of the patients,[2,4] but during the follow-up period constrictive pericarditis was not reported. Only 2 case reports described CA-related constrictive pericarditis.[5,6] We present a case of constrictive pericarditis following CA to treat AF and cardiac tamponade. This is the first case report associated with cardiac tamponade. CA-related constrictive pericarditis is a very rare complication; however, to treat CA-related constrictive pericarditis appropriately, clinical information must be available.

KEY TEACHING POINTS

- Radiofrequency ablation for atrial fibrillation-related constrictive pericarditis is a very rare, but possible complication.
- Several symptoms of constrictive pericarditis after catheter ablation and cardiac tamponade appear a few months after the procedure.
- The correct diagnosis of constrictive pericarditis enables surgical treatment, which is the most effective therapy to improve the patient's deteriorated state.

Case report

A 69-year-old man with paroxysmal AF was referred for pulmonary vein isolation. He had a history of bronchial and eosinophilic asthma for >20 years and mild hypertension and diabetes mellitus for several years. A chest radiography was normal (Figure 1A), and a computed tomography demonstrated no significant abnormalities (Figure 1E). Laboratory testing revealed a C-reactive protein (CRP) level of 0.45 mg/dL and an estimated glomerular filtration rate (eGFR) of 51 mL/min/1.73 cm^2. Echocardiography demonstrated normal ventricular structure and function with mild left atrial dilatation; the left ventricular ejection fraction was 61%, and the left atrial diameter was 41 mm. A routine single transseptal puncture was performed under bidirectional atrial contrast images, and 3 long sheaths were inserted into the left atrium via 1 puncture site. We did not use intracardiac echocardiography to guide the atrial septal puncture. A wide-area circumferential bilateral pulmonary vein and superior vena cava (SVC) isolation, and right atrial cavotricuspid isthmus ablation, were successfully performed using a ThermoCool SmartTouch irrigation-tip contact force radiofrequency ablation catheter (Biosense Webster Inc, Irvine, CA). An electroanatomical mapping system was used and ablation index (AI) guide ablation was performed (CARTO3 version 6; Biosense Webster Inc). The power was controlled between 35 and 40 W and the contact force was controlled between 10 and 20 g. AI was controlled based on the target region as follows: 450 at the right side of the left atrial posterior wall and roof; approximately 500 at the anterior wall; 500–550 at the left pulmonary vein anterior ridge; and 300–350 around the esophageal region. Bilateral pulmonary vein isolation was completed in a single pass by point-by-point ablation. Cavotricuspid isthmus linear ablation was performed at 35 W using the dragging technique. In this case the SVC sleeve length was 35 mm; we isolated the SVC using point-by-point ablation.[7] The power was 25–30 W and the AI was 350–400. The SVC was electrically isolated by 5-point radiofrequency delivery. Under deep sedation, noninvasive positive pressure ventilation was used. During the procedure, the systolic blood pressure (BP) was 100–120 mm Hg and the activated coagulation time (ACT) was 353–393 seconds (measured every 30 minutes). The procedure time was 150 minutes and

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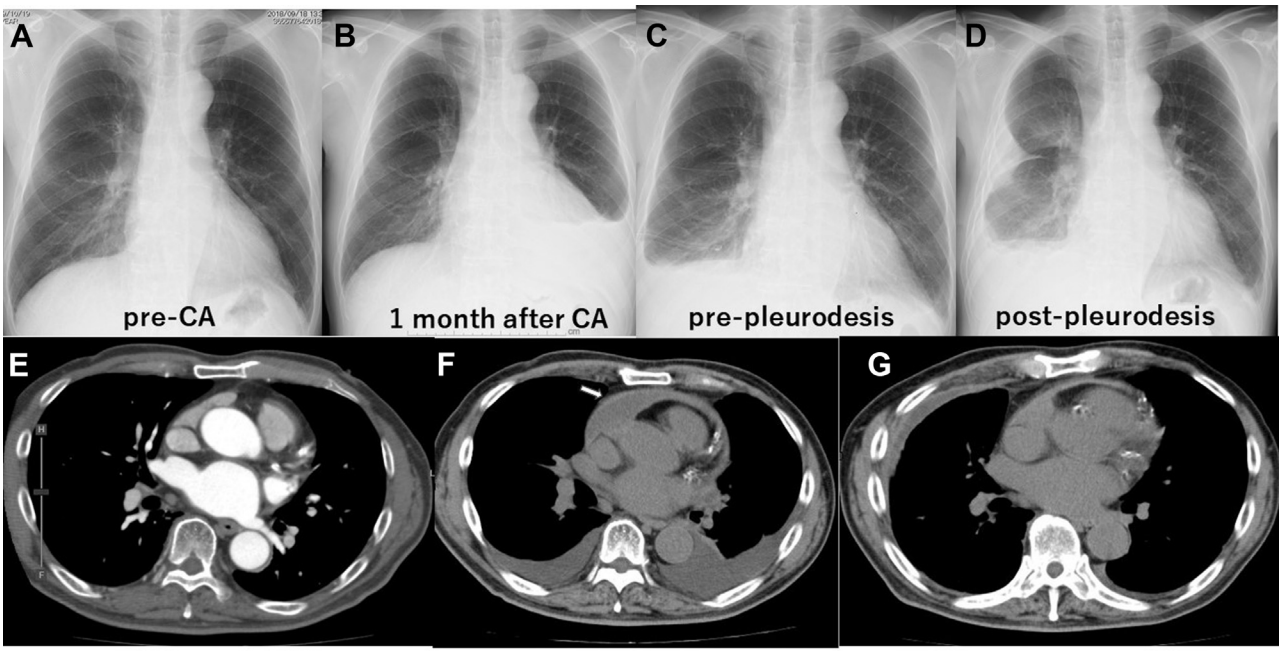

Figure 1 Chest radiograph and computed tomography during the clinical course. **A:** Before ablation. **B:** One month after ablation. A left pleural effusion is presented. **C:** Before pleurodesis 14 months after ablation. Treatment-resistant right pleural effusion was increased. **D:** After pleurodesis. Pleural effusion was controlled. **E:** Before ablation. Pericardial thickening and pericardial effusion were not observed. **F:** Three months after ablation. Pericardial effusion was decreased, but the bilateral pleural effusion was increased. The white arrow indicates a thickened pericardium. **G:** Nineteen months after ablation and after pleurodesis. Thickened pericardium anterior to the right ventricle was observed.

the fluoroscopy time was 38 minutes. Scheduled procedure was completed without complications. The vital signs were stable during ablation. At the end of the procedure the BP was 106/66 mm Hg and the ACT was 293 seconds. Three milliliters of protamine hydrochloride was administered intravenously at the end of the procedure.

When the patient returned to the cardiology ward, he complained of a cold sweat and chest discomfort. The systolic BP had dropped to 70 mm Hg and the heart rate was 70 beats/min. Bedside echocardiography revealed a 10 mm circumferential pericardial effusion; thus we made the diagnosis of cardiac tamponade. A percutaneous pericardial puncture was performed immediately at the bedside. After dark red venous blood–like fluid was drained, the BP increased to >90 mm Hg. The patient was transferred to the intensive care unit. Four hours after the pericardiocentesis, the measured ACT was still 180 seconds and an additional 2 mL of protamine hydrochloride was administered intravenously. The cause of the cardiac tamponade was not clear. The total drained pericardial fluid was 1890 mL in 10 hours. After pericardial drainage, the vital signs were stabilized. The hemoglobin dropped from 12.9 g/dL to 8.2 g/dL, and 4 units of red blood cells were transfused. The next day, the total amount of drained fluid decreased to 260 mL, and the drainage tube was removed 2 days after the procedure. Edoxaban (60 mg/day) was discontinued the day before ablation. Unfractionated heparin was used to prevent thrombosis during the procedure. Edoxaban was resumed 8 days after the procedure. Sinus rhythm was maintained after CA. The patient was discharged 9 days after the CA. The electrocardiogram did not show ST-segment elevation indicating acute pericarditis.

One month after discharge, he complained of shortness of breath, dyspnea, and leg edema. A chest radiography showed left costophrenic angle dullness (Figure 1B). An oral diuretic was prescribed, which improved the symptoms. A focal pericardial effusion was still present anterior to the right ventricle. Three months postoperatively a right pleural effusion was noted that increased in size and was resistant to medical treatment. As shown in Figure 1F, a bilateral pleural effusion, a small amount of pericardial effusion, and a thickened pericardium were observed. An evaluation of the pleural effusion did not reveal evidence of a malignancy, infection, or other disease process. After CA, sinus rhythm was maintained and a left ventricular ejection fraction >60% was preserved. Pericarditis or pleurisy were not ruled out owing to the prolonged elevated CRP (>1.0 mg/dL; Figure 2). The patient was started on colchicine (0.5 mg/day). Seven months later, the CRP had decreased to <0.5 mg/dL and the eGFR was 35–40 mL/min/1.73 cm^2; therefore, colchicine was discontinued. Ten months later, however, dyspnea and leg edema recurred and the right pleural effusion increased again. The cause of the right pleural effusion was evaluated again in Respiratory Department (Figure 1C). Blood and pleural effusion samples were evaluated and a pleural biopsy was obtained. The effusion had an exudative pattern, and clinical findings suggestive of malignancy, infection, or other disease process were not observed. Cardiovascular magnetic resonance imaging excluded pulmonary vein stenosis but demonstrated circumferential pericardial thickness (maximum 4–5 mm). To treat

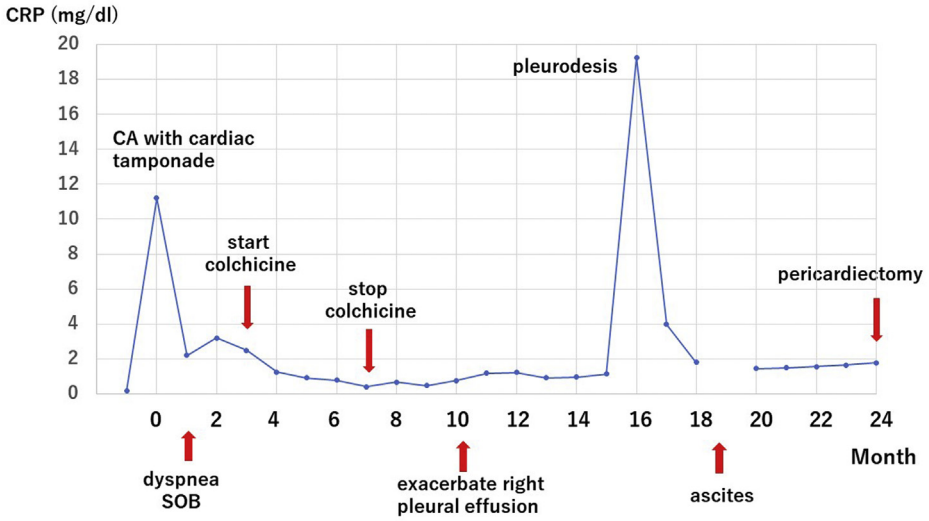

Figure 2 Clinical course and the C-reactive protein (CRP) trend. Continuously elevated CRP after catheter ablation (CA) was observed. SOB = shortness of breath.

the resistant right pleural effusion that was refractory to medical treatment, pleurodesis was performed (Figure 1D). At this time, we did not strongly suspect constrictive pericarditis as the cause of resistant pleural effusion.

After pleurodesis, ascites accumulated that was resistant to diuretics. The progressive worsening of dyspnea, ascites, and leg edema were consistent with right heart failure. Renal function worsened, and the eGFR was approximately 20 mL/min/1.73 cm^2. These findings suggested severe venous congestion. As shown in Figure 1G, a thickened pericardium without calcifications was observed that was prominent in computed tomography. We performed a right heart catheterization, which revealed equalization of the right and left ventricular diastolic filling pressure with a "dip and plateau" pattern (Figure 3), suggestive of constrictive pericarditis.

Twenty-four months after the CA, a pericardectomy was performed that revealed a thickened pericardium without calcifications, which was adherent to the right ventricular free

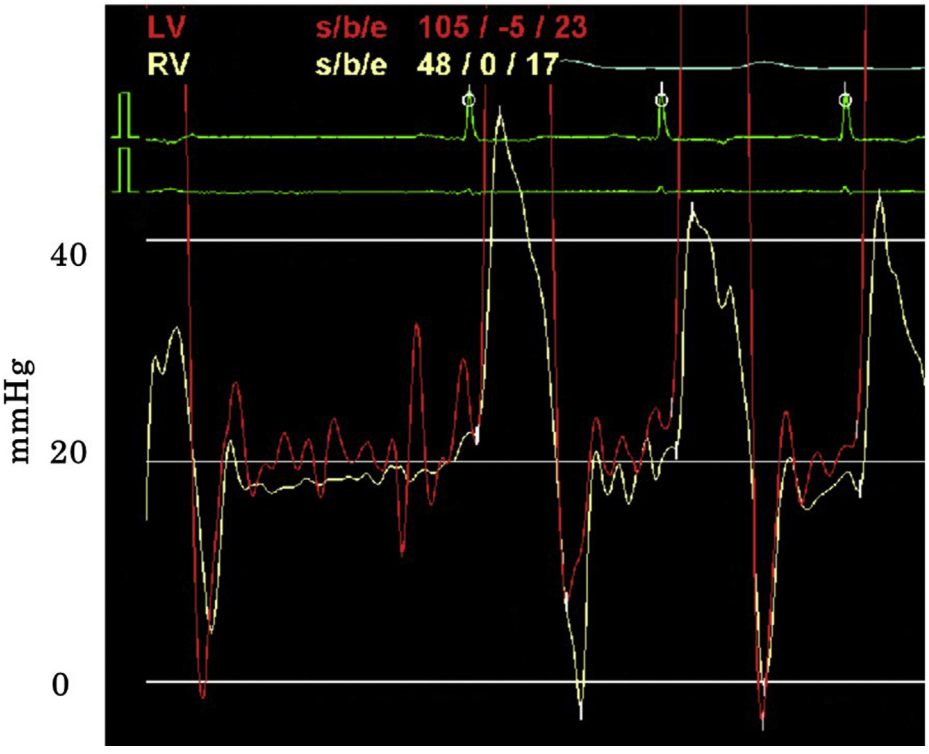

Figure 3 Right and left catheterization derived pressure curve showing elevated and equalization of the right and left ventricle, diastolic filling pressure, and the "dip and plateau" sign.

wall. The pathologic finding was fibrous thickening of the pericardium with a small number of inflammatory cells. After the pericardectomy was performed, ascites, a pleural effusion, and leg edema were controlled and the dyspnea decreased. He was discharged and followed as an outpatient.

Discussion

This case highlights the clinical information suggestive of a CA complication. First, constrictive pericarditis is a very rare, but possible, CA complication. Second, several symptoms related to constrictive pericarditis appear relatively early in the period after the CA. In this case the symptoms arose 1 month after the CA; therefore, differential diagnosis including constrictive pericarditis and presenting dyspnea is needed to establish the correct diagnosis.

Constrictive pericarditis after CA for AF is a very rare complication.[2,4] Only 2 case reports have described this complication.[5,6] In both cases, the initial symptom was dyspnea, which occurred approximately 3 months after the procedure. In the current case, dyspnea appeared 1 month after CA, and the symptoms deteriorated thereafter despite medical treatment. Several symptoms of constrictive pericarditis associated with CA may occur early after the procedure. The clinical course overlapped with other diagnosis in our case. After the correct diagnosis of constrictive pericarditis was established, surgical pericardiectomy was the only effective treatment. In our case, the appearance of resistant ascites after pleurodesis and progressive prominent symptoms of congestion were consistent with a diagnosis of constrictive pericarditis. We established the correct diagnosis after the right heart catheterization. The first reported case showed that clinical improvement is sustained after pericardectomy.[5] In the second reported case, the patient died 31 months after ablation without a pericardectomy.[6] Prolonged corticosteroid therapy may be related to missing the time for surgical therapy. A correct diagnosis is necessary to select surgical treatment, because of its invasiveness.

Constrictive pericarditis is reported to be the end stage of an inflammatory process involving the pericardium. Conventional thinking is that the clinical course of constrictive pericarditis takes time to manifest clinical symptoms. However, the initial symptoms in the current case appeared 1 month after the procedure. Acute pericarditis complicated with CA for AF occurred in 0.8% of patients.[8] With respect to cardiac tamponade following CA for AF, pericarditis occurred in 34%–53% of the patients.[2,4] Cardiac tamponade induces pericarditis and might provoke constrictive pericarditis. The mechanisms underlying constrictive pericarditis, which was exacerbated early and progressively, was not evident. The early-onset clinical symptoms were possibly related to acute pericarditis and gradually changed to the symptoms of constrictive pericarditis. An inflammatory process following radiofrequency CA and cardiac tamponade is an important factor leading to progression of constrictive pericarditis. A sustained elevated CRP after the CA procedure indicated a prolonged inflammatory process, and supports this hypothesis. We did not use anti-inflammatory drugs in the acute phase in this case. Although there are no approved treatments for pericarditis after cardiac tamponade, prophylactic nonsteroidal anti-inflammatory drugs or corticosteroids have been reported to decrease the incidence of pericarditis.[2] Indeed, the earlier administration of these drugs may prevent the occurrence of constrictive pericarditis.

Conclusion

Constrictive pericarditis after CA for AF is a very rare but possible complication. Symptoms occur early after the procedure. To achieve the correct diagnosis and surgical treatment, this complication needs to be understood.

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