Two Cases of Delayed Cardiac Tamponade due to Pericarditis after Pulmonary Vein (PV) Isolation for Atrial Fibrillation

Sadayoshi Torihashi, Hirokazu Shiraishi, Tetsuro Hamaoka, Mikimasa Imai, Akira Kuroyanagi, Naohiko Nakanishi, Takeshi Nakamura, Tetsuhiro Yamano, Akiyoshi Matsumuro and Takeshi Shirayama

Abstract

Catheter ablation is an established treatment for atrial fibrillation (AF). The incidence of major complications related to the procedure is reported to be 4.5%, and delayed cardiac tamponade (DCT) is a rare, although recently recognized, complication. However, the mechanisms underlying the development of DCT remain unclear. We herein report the cases of two men, both 49 years of age, who developed cardiac tamponade requiring pericardiocentesis a few weeks after undergoing pulmonary vein isolation for persistent AF. Physicians should explain to the patient the potential for DCT as a complication prior to performing catheter ablation and provide careful follow-up for at least a few weeks after the session.

Key words: pericarditis, delayed cardiac tamponade, atrial fibrillation, catheter ablation

(DOI: 10.2169/internalmedicine.54.2537)

Introduction

Catheter ablation is an established treatment for atrial fibrillation (AF). Generally, the procedure is safe and effective, although the incidence of major complications is reported to be 4.5% (1). Cardiac tamponade may occur during or immediately after pulmonary vein (PV) isolation, at a rate of between 0.6% and 1.3% (2, 3). Delayed cardiac tamponade (DCT) is a rare, although recently recognized, major complication (4, 5). In a recent worldwide survey (6), the incidence of DCT was reported to be 0.2%, with a mortality rate of 5%. However, the mechanisms underlying the development of DCT remain unclear. We herein report the cases of two patients who developed cardiac tamponade requiring pericardiocentesis a few weeks after undergoing PV isolation for persistent atrial fibrillation.

Case Reports

We herein report two similar cases of DCT following PV isolation. Both patients (case 1 and 2) were 49-year-old men admitted for the treatment of persistent drug-refractory atrial fibrillation. In case 1, the patient was admitted to our affiliated hospital for the treatment of heart failure due to dilated cardiomyopathy accompanied by atrial fibrillation and had undergone cardiac resynchronization therapy (CRT)-D implantation three years previously. In case 2, a diagnosis of lone atrial fibrillation was made on an echocardiogram. We performed Lasso-guided circumferential PV isolation and linear ablation between the tricuspid-inferior vena cava using the three-dimensional mapping system (CARTO, Biosense Webster, Diamond Bar, USA) for navigation based on fusion images with cardiac computed tomography. PV isolation was attained via point-by-point ablation with a 3.5-mm irrigated-tip catheter (Navistar ThermoCool Irrigated Tip).
Catheter, Biosense Webster, Diamond Bar, USA). A steer-
able sheath (Agilis, St. Jude Medical, St. Paul, USA) was
used to achieve catheter stability. A total of 94 and 100 ra-
diofrequency applications were applied in case 1 (Fig. 1)
and case 2 (Fig. 2), respectively. The ablation settings con-
sisted of a catheter tip temperature of 42°C, delivery dura-
tion of 25 seconds, power of 25-30 W and flow rate of 17
mL/min. A transesophageal thermometer was also inserted
to avoid injury to the esophagus. The activated clotting time
(ACT) was controlled at approximately 300 seconds during
the procedure with the transvenous administration of unfrac-
tionated heparin sodium. Oral anticoagulation with warfarin
potassium was continued during each patient’s hospital stay
with an international normalized ratio (INR) value of ≥2.
The antiarrhythmic regimen included amiodarone (100 mg)
and carvedilol (5 mg) once a day in case 1 and flecainide
(200 mg) and atenolol (37.5 mg) once a day in case 2. After
the completion of the procedure, atrial fibrillation was con-
verted to sinus rhythm, and the transthoracic echocardio-
gram showed no pericardial effusion in either case. Both pa-
tients complained of mild chest discomfort one day after the
session, although it was controllable with the administration
of non-steroidal anti-inflammatory drugs (NSAIDs) (loxo-
profen sodium hydrate). The patients’ vital signs during
their hospital stay were stable, without hypotension or pul-
sus paradoxus.
In case 1, the patient was discharged on the second day after the session and then readmitted on the 16th day after the procedure due to shortness of breath and chest pain. A physical examination performed on readmission showed the following findings: blood pressure = 152/86 mmHg, heart rate = 87 bpm, regular rhythm and body temperature = 37°C. Pericardial friction rub was heard, although there was no rales over the lung fields. Ultrasonic cardiography (UCG) showed massive pericardial effusion (left panel in Fig. 3), and a chest X-ray demonstrated cardiomegaly (Fig. 4). A 12-lead electrocardiography (ECG) disclosed a sinus rhythm, decrease in R-waves and non-ischemic ST elevation in leads II, III, aVF and V4-V6 compared with the preoperative ECG findings. Furthermore, biochemistry revealed an elevated C-reactive protein (CRP) level (Table 1). After performing serial UCG follow-up, a diagnosis of cardiac tamponade was
made based on the increase in pericardial effusion and right ventricular collapse accompanied by the gradual development of hypotension and tachycardia (blood pressure = 100/60 mmHg, heart rate = 100 bpm, irregular rhythm). Pericardiocentesis was performed three days after readmission, and 500 mL of hemorrhagic fluid with leukocytosis was drained. Following pericardiocentesis, the patient’s symptoms quickly improved and the CRP level normalized. A cytologic analysis and bacterial culture of the pericardial fluid were both negative. A drainage tube was inserted into the pericardial space and subsequently removed after four days, without signs of persistent bleeding. During the follow-up period of one year, the patient remained free of atrial fibrillation and pericardial effusion.

In case 2, the patient was discharged on the fourth day after the session and then readmitted to our hospital on the 26th day after the procedure due to chest discomfort. A physical examination performed on readmission showed the following findings: blood pressure = 137/96 mmHg, heart rate = 135 bpm, irregular rhythm, body temperature = 37.1°C and a dilated jugular vein. Pericardial friction rub was heard, although there was no rales over the lung fields. UCG revealed massive pericardial effusion (Fig. 5), and a chest X-ray disclosed cardiomegaly (Fig. 6). A 12-lead ECG demonstrated atrial fibrillation with a rapid response as well as a decrease in the R-wave amplitude in all precordial leads. Biochemistry revealed an elevated CRP level (Table 2). Furthermore, UCG showed right ventricular collapse, and pericardiocentesis was thus urgently performed on the day of readmission due to apparent cardiac tamponade (right ventricular collapse).
Figure 6.  Serial follow-up on chest X-rays in case 2. The left panel was recorded on the day after the session, and the middle panel was recorded on the eighth day after the session. The left panel shows slight congestion with cardiomegaly possibly due to long lasting atrial fibrillation and overload with the drip infusion during the operation. The middle panel shows an almost normalized cardiothoracic ratio. The right panel was recorded on readmission, showing apparent cardiomegaly due to massive pericardial effusion with left pleural effusion.

Table 2.  Serial Laboratory Data in Case 2 Showing Elevation of the CRP Level and an Increased INR on Readmission

<table>
<thead>
<tr>
<th>Lab data</th>
<th>One day</th>
<th>4 days after session</th>
<th>8 days after session</th>
<th>26 days after session</th>
<th>29 days after session</th>
<th>34 days after session</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC(µL)</td>
<td>10,900</td>
<td>4,900</td>
<td>5,100</td>
<td>7,100</td>
<td>6,700</td>
<td>4,000</td>
</tr>
<tr>
<td>Hb(g/dL)</td>
<td>14.1</td>
<td>14.1</td>
<td>13.9</td>
<td>12.4</td>
<td>12.4</td>
<td>13.9</td>
</tr>
<tr>
<td>Ht(%)</td>
<td>42.9</td>
<td>41.4</td>
<td>41.3</td>
<td>38</td>
<td>37.5</td>
<td>43.3</td>
</tr>
<tr>
<td>CRP(mg/dL)</td>
<td>0.89</td>
<td>5.69</td>
<td>0.92</td>
<td>11.8</td>
<td>7.9</td>
<td>0.97</td>
</tr>
<tr>
<td>CKI(IU/L)</td>
<td>109</td>
<td>146</td>
<td>169</td>
<td>48</td>
<td>48</td>
<td>53</td>
</tr>
<tr>
<td>AST(IU/L)</td>
<td>23</td>
<td>21</td>
<td>43</td>
<td>19</td>
<td>19</td>
<td>29</td>
</tr>
<tr>
<td>LDH(IU/L)</td>
<td>299</td>
<td>263</td>
<td>578</td>
<td>333</td>
<td>265</td>
<td></td>
</tr>
<tr>
<td>Cre(mg/dL)</td>
<td>0.83</td>
<td>0.82</td>
<td>0.96</td>
<td>0.84</td>
<td>0.86</td>
<td>0.96</td>
</tr>
<tr>
<td>PT-INR</td>
<td>2.86</td>
<td>3.13</td>
<td>2.71</td>
<td>5.88</td>
<td>1.32</td>
<td>1.08</td>
</tr>
</tbody>
</table>

These parameters immediately recovered after pericardiocentesis. The increased INR was caused by liver congestion due to cardiac tamponade.

Panel in Fig. 5). After draining 1,300 mL of hemorrhagic fluid with leukocytosis, the patient’s symptoms quickly improved, accompanied by the restoration of a sinus rhythm and normalization of the CRP level. A bacterial culture and cytologic analysis of the pericardial fluid were both negative. The drainage tube was removed after 48 hours, with no signs of persistent bleeding. During the follow-up period of one year, the patient remained free of atrial fibrillation and pericardial effusion.

Discussion

Catheter ablation (pulmonary vein isolation) is an established therapy for AF, although numerous complications (1) are associated with this procedure, including pericardial effusion. In addition, acute cardiac tamponade has been reported in 0.6-1.3% of cases (2, 3). Cappato et al. reported that the onset of acute cardiac tamponade was clearly associated with transseptal puncture or mechanical injury to the left atrium in addition to a high level of anticoagulation (7).

DCT is a rare, although recently recognized, major complication of catheter ablation. A recent study (6) by Cappato et al. reported 45 cases of delayed tamponade among 27,921 sessions performed in 21,478 patients (0.2%), with a mortality rate of 5%. The authors defined DCT as the development of hypotension or cardiogenic shock requiring pericardial drainage or causing death due to documented pericardial effusion occurring at least one hour after the procedure and
found that their patients presented with DCT a median of 12 days (range: 0.2 to 45 days) after ablation. In our cases, there were no marked episodes of hypotension or cardiogenic shock. However, we diagnosed both patients with cardiac tamponade because UCG showed massive pericardial effusion with right ventricular collapse. Cappato et al. (6) reported predictors of at least one DCT event to be as follows according to a multivariate analysis: a large number of treated patients (odds ratio: 5.03), the use of irrigation catheters (odds ratio: 2.77) and treatment of paroxysmal AF only (odds ratio: 3.97). In that study, the majority of patients showed gradual progression to tamponade accompanied by preceding symptoms, including chest pain, pain while breathing, shortness of breath, general malaise and fever, similar to that observed in our cases. Although the mechanisms underlying the development of DCT remain unclear, several causes can be speculated. For example, Goossens et al. reported that potential mechanisms include sealed ablation-induced left atrial rupture or small pericardial hemorrhage due to the application of post-procedural anticoagulation therapy (4). According to Cappato’s report (6) of 45 patients with DCT, hemorrhagic pericardial fluid was reported upon visual inspection in 36 patients, whereas serous effusion was the only finding in the remaining nine patients. The author reported that the mode of presentation of hypotension varied, with 39 patients exhibiting gradual progression to cardiac tamponade. Our two patients also exhibited a gradual progression to tamponade, with a massive volume of pericardial effusion. This indicates the gradual accumulation of pericardial fluid accompanied by the extension of the pericardium. Therefore, we speculate that the mechanisms of DCT were similar to those of subacute pericarditis, such as Dressler’s syndrome, in our cases, because the pericardial effusion demonstrated leukocytosis in association with an elevated serum CRP level based on the biochemistry data. Nevertheless, sealed microperforations in the atrium may have also played a role in the progression of tamponade in our patients due to the administration of relatively intensive anticoagulation treatment after ablation. In case 2, the prothrombin time-international normalized ratio (PT-INR) measured four days after the session was elevated at 3.13. We did not consider the INR at that time to be high, although the PT-INR value observed 26 days after the session, when the patient was readmitted, was extremely high at 5.88. We therefore believe that the prolonged INR was caused by congestive liver due to massive pericardial effusion, although the high INR may have also exaggerated the microbleeding into the pericardial space. The use of persistent oral anticoagulation with warfarin potassium may also be a cause of the hemorrhagic reaction in the pericardial space. The onset of Dressler’s syndrome following catheter ablation has been previously reported (4, 8, 9); corticosteroids were used for treatment in several cases (4, 8). Fortunately, our patients exhibited good clinical courses, with only treatment with NSAIDs and pericardiocentesis. Otherwise, the administration of corticosteroids should be considered.

We herein reported two cases of DCT in which cardiac tamponade developed a few weeks after PV isolation. Although DCT is rare, physicians should remember it as a potential major complication of catheter ablation. In addition, clinicians should explain to patients who undergo PV isolation the potential for DCT as a complication prior to performing catheter ablation and provide careful follow-up for at least a few weeks after the session.

The authors state that they have no Conflict of Interest (COI).

References