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Case Report

Post-cardiac injury syndrome after a concealed accessory pathway ablation

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ABSTRACT

The incidence of pericardial effusion for supraventricular tachycardias is less than 1%, and its combination with pleural effusion is rare. We present a case of severe pericardial and pleural effusion after a left-sided concealed accessory pathway ablation. The 480 cc of pericardial fluid was drained with the pericardial drainage system due to cardiac tamponade with hemodynamic compromise. The chest X-ray and thorax computed tomography showed moderate left-sided pleural effusion after pericardiocentesis. We considered the inflammatory response as the pathophysiology of the situation; we started ibuprofen 800 mg t.i.d. and colchicine 0.5 mg o.d. At a 3-week follow-up, her X-ray revealed the resolution of pleural effusion, and the echocardiography showed no pericardial effusion.

<**Learning objective:** Pericardial effusion is rarely seen after cardiac ablations. Despite the mechanism not being well-established, it seems that inflammation plays a critical role. A trial of non-steroidal anti-inflammatory drugs and colchicine can be used safely and seems reasonably effective in this setting.>

Introduction

The incidence of the combination of pericardial and pleural effusion after catheter ablation is rare. We present a case of successful management of post-cardiac injury syndrome after catheter ablation with anti-inflammatory medications.

Case report

A 34-year-old female patient presented with recurrent short-RP narrow-QRS tachycardias. The sinus electrocardiogram (ECG) showed sinus rhythm without pre-excitation. The patient had no history of chronic disease. The echocardiogram revealed no structural abnormality. After discussing the treatment options, she accepted the catheter-based therapy. During the electrophysiologic study, there was a narrow-QRS tachycardia of cycle length 250 ms while checking ventriculoatrial (VA) conduction. The VA conduction was revealed during the tachycardia, and a his-refractory premature ventricular contraction terminated tachycardia. The ventricular overdrive pacing from the posterolateral annulus was performed, the VAV response was seen, the difference between post-pacing interval and cycle length was 83 msn, and the stimulus-atrial interval minus VA interval was 58 msn. During the tachycardia, the radiofrequency catheter was introduced into the left ventricle via a retrograde aortic approach to map the earliest ventricular activation at the posterior part of the mitral annulus near the CS 3,4 after administering 6500 IU of intravenous heparin (100 IU per kg). The radiofrequency generator was set to a temperature limit of 43°C and energy of 35 Watts. The tachycardia was terminated in 2 seconds of radiofrequency ablation (RFA) with non-irrigated ablation catheter at this site, and the RFA was continued around 60 seconds (20 Watts, 43°C). After a post-ablation waiting period of 30 minutes, there was no inducible tachycardia with/without isoproterenol infusion. The total procedure time was 25 minutes, except for the waiting period. The activated clotting time (ACT) was >450 seconds at the end of the procedure. After 8 hours of the procedure, the patient became tachycardic and hypotensive (80/50 mmHg). The bedside echocardiographic examination revealed cardiac tamponade, which is shown in Fig. 1A. An emergency pericardiocentesis was performed, with immediate drainage of 480 mL of hemorrhagic fluid. Because of the emergency setting, we did not send the fluid obtained from the pericardial space for blood gas analysis. She became stable with a blood pressure of 110/70 mmHg, and she was transferred to the coronary care unit without inotropic support. At a 1-day follow-up, there was no increase in the pericardial fluid with a maximal diameter of 15 mm in the subcostal view. The complete blood count (CBC)

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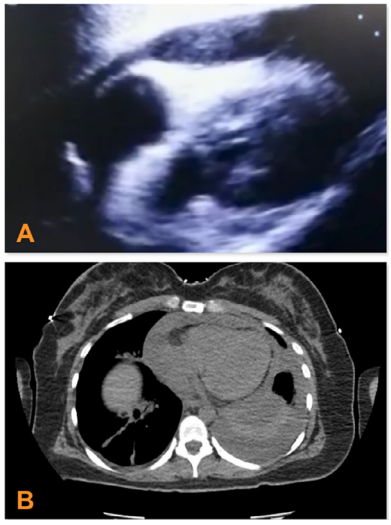

Fig. 1. (A) The bedside echocardiographic examination reveals cardiac tamponade physiology. (B) The thoracic computed tomography shows diffuse pericardial effusion and a left-sided pleural effusion.

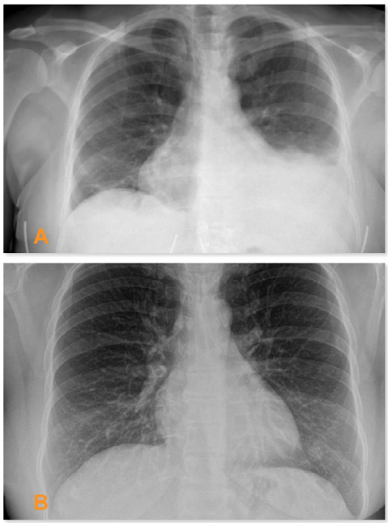

Fig. 2. (A) The chest X-ray shows a left-sided pleural effusion. (B) The chest X-ray shows resolution of the plural effusion after anti-inflammatory treatment.

revealed a mildly elevated white blood cell (WBC) of 10.8 $\times$ 10^9/L (normal 4.0–10.0 $\times$ 10^9/L) and C-reactive protein (CRP) level was 159 mg/dL (normal 0.0–5.0 mg/L). The patient was hemodynamically stable (blood pressure of 110/65 mmHg, heart rate 80 bpm). The ECG revealed no abnormalities with a heart rate of 80 bpm and she was discharged in five days. The patient presented with fatigue, shivering, dyspnea, and pleuritic chest pain after 12 days of discharge. The physical examination showed pericardial friction rubs, and the absence of breath sounds in the left lower lobe. The body temperature was 37.8 °C, and chest X-ray and thoracic computed tomography (CT) were ordered, and it showed that there was a left-sided pleural effusion with diffuse pericardial effusion (Fig. 1B). The mechanism of the pleural effusion due to post-cardiac injury syndrome was considered, and anti-inflammatory non-steroidal anti-inflammatory drug (NSAID) (ibuprofen (800 mg t.i.d.) with the adjunctive use of colchicine (0.5 mg o.d.) were administered. After three days of anti-inflammatory treatment, she had no chest pain with mild dyspnea. At a 18-day follow-up, there was mild pericardial effusion with a maximal diameter of 10 mm in the subcostal view. The chest X-ray (Fig. 2A) and thoracic CT showed mild pleural effusion. The complete blood count (CBC) revealed a WBC of 8.800 cells/μL, and the CRP level was 4.5 mg/dL. At a 24-day follow-up, the complete resolution of pericardial and pleural effusion was achieved, which is shown in Fig. 2B. The ibuprofen therapy was stopped at the 24-day follow-up visit and the colchicine therapy was continued for three months to reduce the risk of recurrence. The 6-month follow-up visit showed no pericardial or pleural effusions on echocardiography and chest X-ray, and the patient had no symptoms.

Discussion

Catheter-based therapy is the standard of care for most supraventricular tachycardias [1]. The low complication rates with high success rates make these procedures become the first-line therapy in the setting of narrow-QRS tachycardias [2]. Post-cardiac injury syndrome (PCIS) is rarely seen after catheter ablations [3]. PCIS was described in patients with myocardial infarction in 1956 [4]. The autoimmune-mediated conditions of pericardial and myocardial inflammation play a role in PCIS, which could result from direct damage to pericardial cells or a presence of blood in the pericardial space; it is thought that the release of cardiac antigens and injury-related immune response stimulates a systemic immune response [5,6].

A recent case series of PCIS following catheter ablation showed that PCIS is mainly seen in atrial fibrillation (AF) [3]. The common findings include chest pain (>80%), low-grade fever (50-60%), and dyspnea (50-60%), which were also seen in our case [7]. Mainstays of treatment of PCIS are anti-inflammatory drugs, including NSAIDs and colchicine. Steroid administration is reserved for patients with contraindications for NSAIDs or in case of refractory symptoms [3,7]. Some centers may give a short-term steroid regimen in the setting of PCIS [8]. In our case, we have seen the same complete resolution of pericardial and pleural involvement within 2-3 weeks of NSAIDs and colchicine regimen.

The small amount of pericardial effusion after cardiac ablations can be seen in patients on anticoagulants undergoing an RFA of AF using a double transseptal technique [9]. Uninterrupted periprocedural novel oral anticoagulants are recommended in patients undergoing AF catheter ablation, but heparin bridging increases the bleeding and should be avoided [10]. The increase in procedure time, heparin use, and catheter ablation of large areas are thought to be some reasons for pericardial effusion. In our case, the catheter ablation time was less than 120 seconds, and the ablated area was less than 1 cm^2, but the ACT time was still >450 sec one hour after the procedure. Even though the total time of mapping was 15 minutes, it was difficult to map the left ventricular annulus, because of the short stature of the patient and retrograde aortic approach, which might be one of the possible reasons for cardiac tamponade. In addition to that other probable cause of pericardial tamponade could be excessive anticoagulation, and then a large amount of blood leakage in the pericardial sac must have triggered the inflammatory response.

Even though PCIS is rare after cardiac ablations, it should be kept in mind, particularly in long procedure time, ablation of large areas, and excessive anticoagulation. A trial of anti-inflammatory medications consisting of NSAIDs and colchicine seems reasonably effective.

Declaration of Competing Interests

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