



Case report

Postcardiac injury syndrome following vascular interventional radiofrequency ablation for paroxysmal atrial fibrillation



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ABSTRACT

Postcardiac injury syndrome (PCIS) occurs following a pericardial or myocardial injury. On the other hand, PCIS following cardiac catheter intervention is rare and can be difficult to diagnose because of its delayed onset. A 24-year-old man underwent radiofrequency ablation (RFA) for paroxysmal atrial fibrillation and suffered from general fatigue and left-sided pleural effusion three months after the procedure. His symptoms and effusion were effectively treated within a month by administering nonsteroidal anti-inflammatory drugs. However, seven months later, he developed left-sided chest pain and low-grade fever. Computed tomography showed a thickening of the parietal pleura and recurrence of the pleural effusion. Pleural biopsy by video-assisted thoracoscopy demonstrated chronic pleuritis with a non-necrotizing granulomatous reaction. Given the previous RFA, and in the absence of infection or malignant disease, he was diagnosed with PCIS and treated with colchicine.

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1. Introduction

Postcardiac injury syndrome (PCIS) occurs following a pericardial or myocardial injury [1]. The term PCIS includes Dressler's syndrome and postpericardiotomy syndrome (PPS), which occur following myocardial infarction or cardiac surgery. Typically, the patient demonstrates fever elevation, irritability, pericardial friction rub, and pericardial effusion with or without pleural effusion [2]. Recently, the development of PCIS after catheter ablation has been reported in the literature [3,4], although it is considered to be rare. We describe a case of PCIS following a radiofrequency ablation (RFA) for paroxysmal atrial fibrillation.

2. Case report

A 24-year-old man consulted his primary care physician with general fatigue and chest and back pain. Chest radiography demonstrated a left-sided pleural effusion that was diagnosed as a parapneumonic effusion, and he was treated with oral levofloxacin

500 mg per day. However, neither the pain nor effusion improved; therefore, his primary care physician referred him to our institution.

He had low-grade fever without any symptoms of either respiratory disease or heart failure, but he had a history of interventional RFA for paroxysmal atrial fibrillation three months prior to the onset of symptoms. On physical examination on admission, his temperature was 37.4 °C, heart rate 93 beats per min, blood pressure 110/50 mmHg, respiratory rate 16 breaths per min, and pulse oximetry 97% oxygen saturation in room air. Radiography and computed tomography (CT) of the chest demonstrated a left-sided intermediate pleural effusion and slightly thickened parietal pleura. A transthoracic echocardiogram revealed 75% of left ventricular ejection fraction without regional wall motion abnormalities and normal level of pericardial effusion. The result of laboratory examinations were unremarkable except for a trivial increase of C-reactive protein (CRP) level of 0.46 mg/dL. QuantiFERON-TB Gold In-Tube assay was negative. Thoracentesis revealed a nonbloody exudative effusion with lymphocytosis. There was no evidence of infection or neoplasia in either the culture or the cytology results.

He was diagnosed with pleuritis and was prescribed oral loxoprofen in addition to original therapy. Both his symptoms and pleural effusion gradually improved over the following month.

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However, seven months after his initial treatment, he complained of intense chest pain. Chest radiography and CT showed the recurrence of the left-sided pleural effusion accompanied by a thickening of the parietal pleura (Fig. 1). He had a slightly elevated CRP level at 1.72 mg/dL and a normal peripheral white blood cell count of 7350 cells/ μ L.

Given his previous RFA for paroxysmal atrial fibrillation three months prior to the onset of his symptoms, we believed that the series of events were highly indicative of PCIS. However, further examination was required to exclude pleural tuberculosis and malignancy. Pleural biopsy by video-assisted thoracoscopic surgery (VATS) under general anesthesia was performed, and the histological specimens from the left parietal pleura demonstrated chronic pleuritis with a non-necrotizing granulomatous reaction (Fig. 2). Furthermore, cultures and polymerase chain reaction analysis of the parietal pleura were negative for mycobacterium. He was clinically diagnosed with PCIS and was successfully treated with colchicine 1 mg and Celecoxib 200 mg daily. The dose of colchicine was reduced to 0.5 mg per day after three weeks and discontinued after one month because of the development of diarrhea.

3. Discussion

PCIS is a condition that involves pleuropericarditis and follows cardiac or pericardial injury from surgery, catheter procedures, or trauma [3]. Although it is believed to be an autoimmune phenomenon, the precise mechanism remains unknown [5,6]. Putative etiological factors include traumatic events such as myocardial infarction [1], cardiac surgery [7], a repair of pectus excavatum [8], thymectomy [9] and cardiac catheter intervention [3,4,10]. On the other hand, only a few reports demonstrated PCIS following RFA.

The reported symptoms of PCIS in the manuscripts are low-grade fever associated with pleural or pericardial inflammation. Sometimes patients develop persistent symptomatic pleural effusions [11–13]. In addition, these symptoms generally persist within a few days to several months. The diagnosis of the PCIS is difficult because the onset may be delayed following the initial traumatic event as presented case. In addition, lacking of characteristic

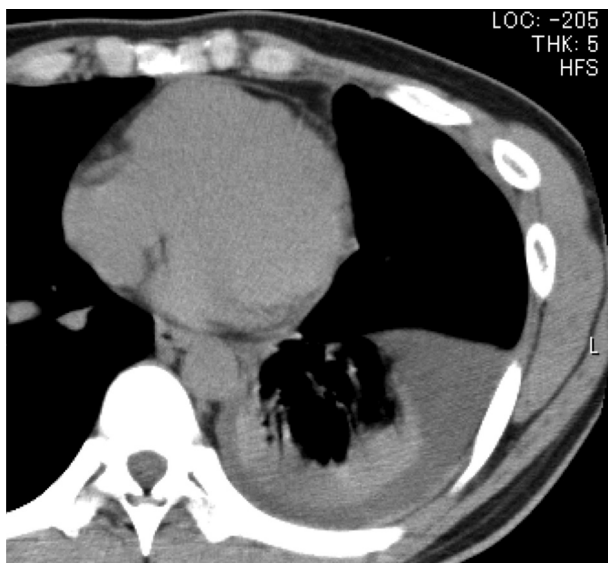


Fig. 1. Chest computed tomography showed a relapse of the left-sided pleural effusion associated with a thickening of the parietal pleura seven months after the first event.

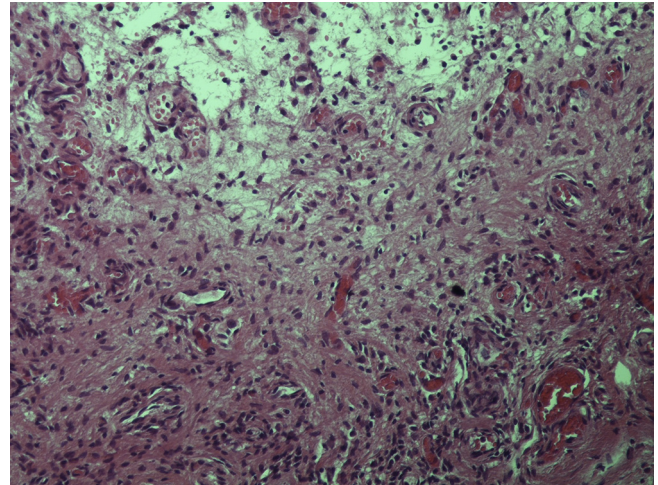


Fig. 2. Hematoxylin and eosin stain of a specimen from the left parietal pleura revealed chronic pleuritis with a non-necrotizing granulomatous reaction.

symptoms or laboratory findings in PCIS makes the diagnosis more difficult. Therefore, the diagnosis is generally made by exclusion.

Although the etiology of PCIS is poorly understood, it is thought to be related to autoimmune disturbance [4,5]. RFA is sometimes utilized for tumor destruction in cancer treatment, and it is proposed that ablated tumor debris could be taken up by the immune system to induce or enhance immune responses [14]. We speculate that RFA of the atrium influences the surrounding pericardium or other structures and triggers PCIS.

The treatment of PCIS is based on nonsteroidal anti-inflammatory drugs and steroids. Colchicine has been effectively used to prevent PCIS, but its efficacy in the treatment of established PCIS is unclear. In a recent report, colchicine administration for pericarditis, including patients with PCIS, proved to be effective in reducing the rate of incessant or recurrent pericarditis and in prolonging the time to recurrence [15]. Although the exact mechanism of colchicine in PCIS remains unclear, it is still a key therapeutic option.

The histological findings of pleura in cases of persistent pleural effusion after coronary artery bypass grafts have been well documented [16–18]. Although these cases were not diagnosed as PCIS, their clinical features are consistent with PCIS. In these reports, pleural biopsy by VATS revealed histological findings of chronic pleuritis with inflammatory cell infiltration. In addition, some formed non-necrotizing granulomatous changes, which are consistent with the present case.

In conclusion, the diagnosis of PCIS in a case following cardiac catheter intervention is difficult because patients no longer suffer from symptoms of the heart disease and general practitioner may undertake the follow up role. Therefore, it would be a matter of importance for clinicians to be aware of PCIS.

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