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Case Report

Uncommon presentation of postcardiac injury syndrome induced by radiofrequency catheter ablation for atrial fibrillation: Only pulmonary parenchymal inflammation

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ABSTRACT

Postcardiac injury syndrome (PCIS) is characterized by the appearance of pericardial, pleural, or pulmonary parenchymal inflammation following a cardiac operation. The autoimmune disorder is characterized by eosinophilia, pleuritic chest pain, pleural, and pericardial effusion. It is a troublesome complication, which may prolong hospital stay and lead to severe and life-threatening conditions. Recently, its incidence appears to be declining, owing to modern immunomodulatory drug therapies. Here we report an uncommon case of PCIS, which occurred following a pulmonary vein isolation for atrial fibrillation. The patient suffered from pulmonary parenchymal inflammation but no pleuropericarditis after the operation. We describe with detailed clinical information, laboratory, and imaging tests, treatment processes, and a favorable prognosis in the content. This case illustrates that even after initially uneventful radiofrequency catheter ablation, careful, long-term follow-up is necessary to recognize the potential development of postcardiac injury syndrome.

<**Learning objective:** Although uncommon, physicians should keep postcardiac injury syndrome in mind following radiofrequency catheter ablation. The pattern does not always resemble the classical picture described by Dressler, but may take many forms and should be sought. Diagnosis is based on the clinical signs and symptoms and is mostly confirmed after excluding other diseases. Prognosis is usually benign, and therapeutic response to anti-inflammatory drugs and corticosteroids can aid in the diagnosis.>

Introduction

The postcardiac injury syndrome (PCIS) is characterized by inflammation of the pericardium, pleura, and pulmonary parenchyma following myocardial infarction, cardiac surgery, blunt chest trauma, percutaneous left ventricle puncture, implantation of a pacemaker, and catheter ablation [1]. The literature contains information regarding the pericardium, pleural, and pulmonary manifestations of PCIS [2]. We report a rare case in which radiofrequency ablation of atrial fibrillation led to the development of pulmonary parenchymal inflammation but no pleuropericarditis.

Case report

A 68-year-old woman with paroxysmal atrial fibrillation that was not controlled by antiarrhythmic drugs was referred for electrophysiology evaluation and treatment (Figs. 1A and 2A). The ablation procedure consisted of a computed tomography (CT)-, CARTO-, and Lasso-guided circumferential pulmonary vein isolation. A three-dimensional anatomy of the left atrium and the pulmonary veins were created by CARTO Sound and ICE (Intra Cardiac Echocardiogram) catheter with an incorporated navigation sensor (SoundStar™ catheter, Biosense Webster, Diamond Bar, CA, USA) (Fig. 3). After the anatomical three-dimensional model of the left atrium was prepared, ablation was performed to encircle the right and left pulmonary veins in pairs, and no atrial fibrillation was induced after ablation. The radiofrequency generator was set to a temperature limit of 43 °C and energy of 30 W, and the total procedure time was 3 h. The trauma experienced by the heart during the ablation resulted in an inflammation of the myocardium.

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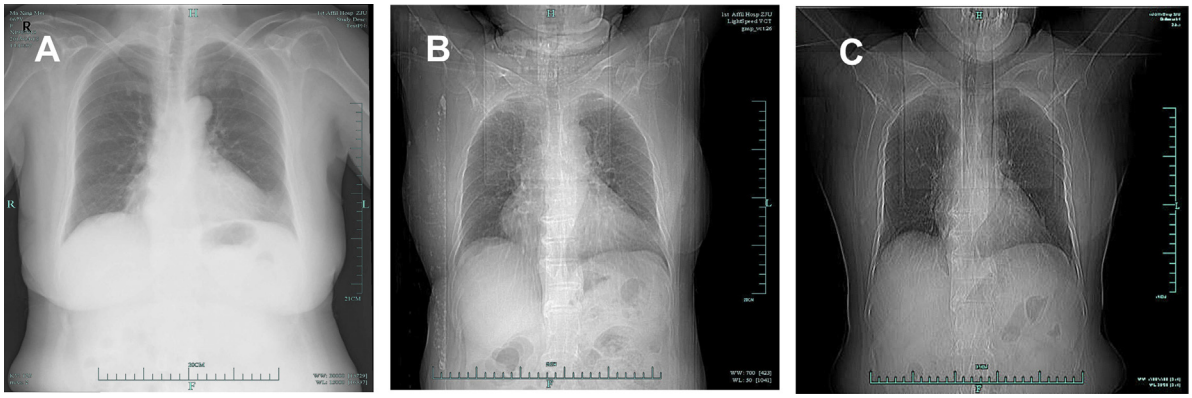

Fig. 1. Posteroanterior chest X-ray films before pulmonary vein isolation, during postcardiac injury syndrome, and after improvement. Enlarged cardiac silhouette (A, C) and ground-glass opacity (B) were present.

On the second day post ablation, the patient had a body temperature of 37.6 °C. The blood leukocyte count was 10.9×10^9/L (normal $4.0–10.0 \times 10^9$/L). C-reactive protein (CRP) level was slightly elevated to 11 mg/L (normal 0.0–8.0 mg/L). These values normalized in 4 days and the patient was discharged. Three weeks later, the patient developed cough and mild chest discomfort. On the day of readmission, she complained of increased shortness of breath. Physical examination revealed scattered rales and strengthened respiratory voices but no heart murmur and S3 were heard on auscultation. Jugular venous pressure was not elevated. No peripheral edema was noted. The blood test showed that the leukocyte count was 6.6×10^9/L, along with an increased percentage of eosinophilic granulocytes of 7.8% (normal value 0.5–5.0%). Erythrocyte sedimentation rate (ESR, 51 mm/h, normal value 0–20 mm/h) and CRP concentration (32.1 mg/L, normal value 0–8 mg/L) were both elevated. The value of D-dimer, B-type

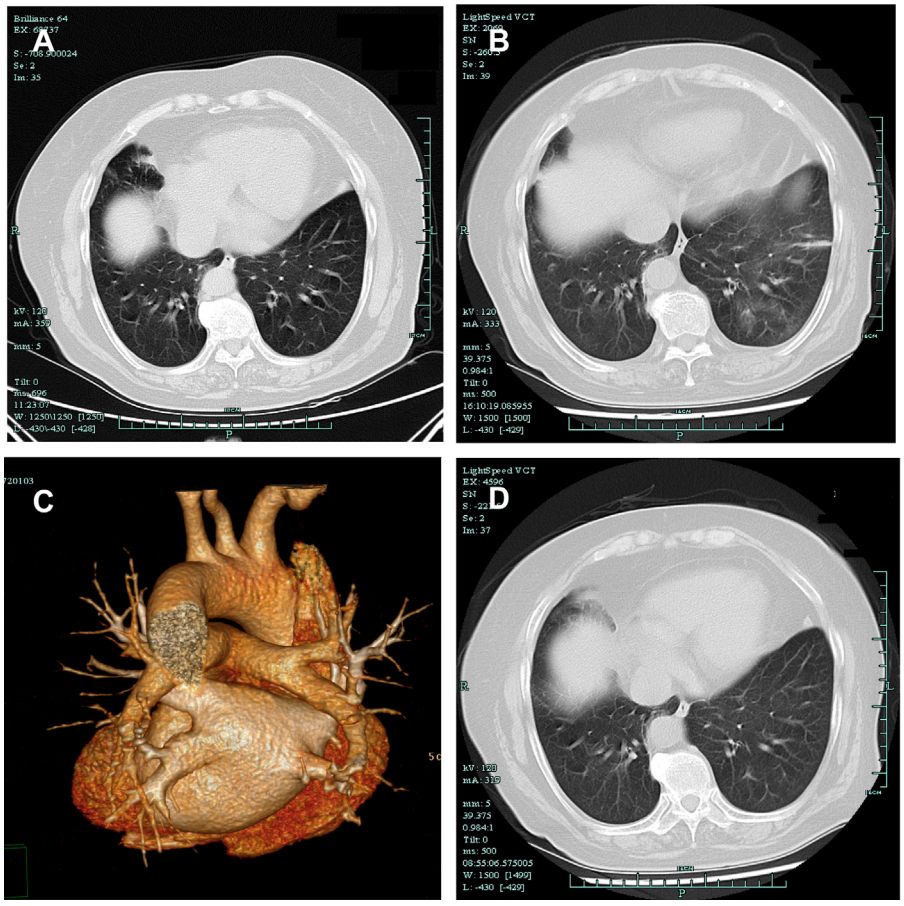

Fig. 2. Representative images from the chest computed tomography (CT) and pulmonary vein angiography. (A) Preoperation: CT images showed that no lesions were found on bilateral lung fields. (B) Postoperation: CT images showed bilateral patchy areas of ground-glass opacity, which were distributed mainly in the inferior lobe of left lung close to heart. (C) Pulmonary vein angiography: No obvious pulmonary vein stenosis was detected and anatomic structures were in normal places. (D) Follow-up: CT scan performed 11 days later showed near-complete resolution of the lesions.