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Case Report

Delayed cardiac tamponade 8 months after pulmonary vein isolation

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ABSTRACT

We herein report the case of a 55 year-old male who underwent pulmonary vein isolation (PVI) for paroxysmal atrial fibrillation. From 8 months after PVI, exertional dyspnea rapidly appeared. When he was referred to our hospital, massive pericardial effusion was observed by transthoracic echography. The pericardiocentesis revealed bloody pericardial effusion, and improved symptoms. Although aortic dissection, autoimmune disease, infection, metastatic pericardial tumor, primary pericardial tumor, and malignant neoplasm were considered as differential diagnosis, the cause of pericardial effusion failed to be found. From these findings, the cause of hemorrhagic pericardial effusion was considered delayed cardiac tamponade induced by PVI performed 8 months earlier.

<**Learning objective:** Cardiac tamponade occurring after pulmonary vein isolation (PVI) procedure called delayed cardiac tamponade (DCT) is not widely recognized. However, mortality from DCT is far less than 5%, and we believe that it is a complication that requires due care. This report describes that DCT can occur even after 8 months of PVI.>

Introduction

Pulmonary vein isolation (PVI) for atrial fibrillation (AF) has been shown to be generally safe and effective treatment and is being performed in an increasing number of patients [1]. However, complications associated with the procedure have been observed [1]. Most of such complications including cardiac tamponade occur during procedure [1,2], and the incidence of cardiac tamponade during PVI is reported to be approximately 0.6%–1.3% [3,4]. On the other hand, cardiac tamponade occurring after PVI procedure called delayed cardiac tamponade (DCT) is not widely recognized. According to several reports [1,5–10], DCT occurs between 0.2 and 55 days after PVI. However, we experienced a case in whom DCT occurred 8 months after PVI.

Case report

The case was a 55-year-old man with diagnosis of hypertension at the age of 54 years. Paroxysmal AF (PAF) was diagnosed and oralapixaban was started at a dose of 5 mg twice daily, because CHADS2/CHA2DS2-VASc score was 1/1 respectively, and his creatinine clearance was 76 mL/min. Because of frequent observation of severe palpitations, PVI was performed for PAF without interrupting apixaban. PVI was achieved via point-by-point ablation with the irrigated-tip catheter (Therapy™ Cool Flex™ Ablation Catheter, St. Jude Medical, St. Paul, MN, USA). The ablation settings consisted of a catheter tip temperature of 43 °C, power of 20–25 W. A total of 31 radio frequency applications were applied. There were no marked perioperative complications, and the patient subsequently progressed to stable with a sinus rhythm. Three months after PVI, apixaban was discontinued. Thereafter, the subject patient continued to be stable with no recurrence of AF. However, 8 months after PVI, the patient suffered from dyspnea; therefore, the patient was referred to our hospital. Physical findings included a noninvasive blood pressure of 124/87 mmHg, and paradoxical pulse was observed. Body temperature was 36.6 °C. The pulse was irregular at 105 bpm, and the SpO_2 was reduced to 92% in room air. Auscultation revealed no pericardial friction rub, but cardiac sound were slightly weak, and jugular venous distension was observed. Laboratory tests revealed white blood cell count of 8700/μL and C-reactive protein level of 0.44 mg/dL, both of which were slightly elevated. The soluble interleukin-2 receptor (sIL-2R), carbohydrate antigen19-9 (CA19-9), carcinoembryonic antigen (CEA), squamous cell cancer, sialyl

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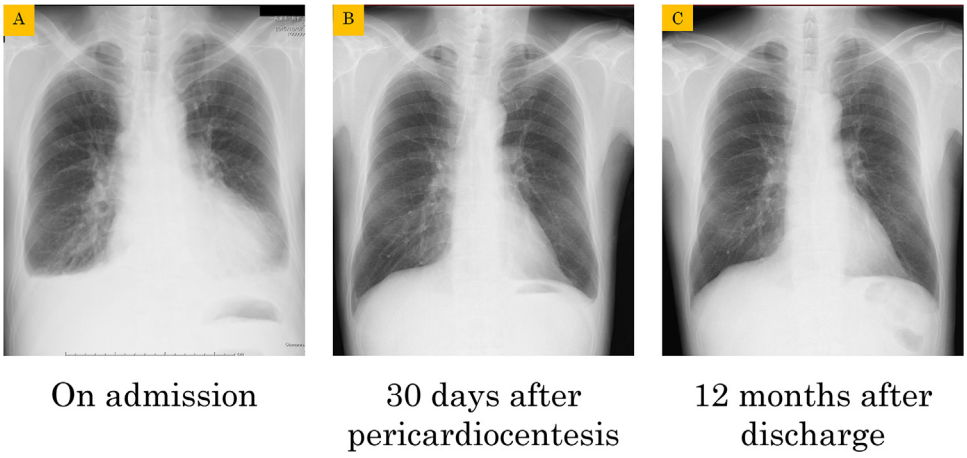

Fig. 1. (A) Chest radiography on admission revealed bilateral pleural effusion and an enlarged cardiac shadow with a cardiothoracic ratio of 64%. Chest radiography after the 30 days following the pericardiocentesis procedure (B) and 12 months after discharge (C). There was no re-accumulation of pericardial effusion.

Lewis x, neuron-specific enolase, cytokeratin-19 fragments, and pro-gastrin-releasing peptide level was 424 U/mL, 8.6 U/mL, 2.4 ng/mL, 0.8 ng/mL, 26.7 U/mL, 9.0 ng/mL, 0.9 ng/mL, and 27.2 pg/mL, respectively, all of which were within normal range. The T-SPOT®TB (Oxford Immunotec, Oxford, UK) was negative. Chest radiography revealed bilateral pleural effusion and an enlarged cardiac shadow with a cardiothoracic ratio of 64% (Fig. 1A). The electrocardiogram (ECG) revealed a heart rate of 120 bpm and AF rhythm. No marked ST–T wave changes were observed. Transthoracic echography (TEE) revealed left ventricular

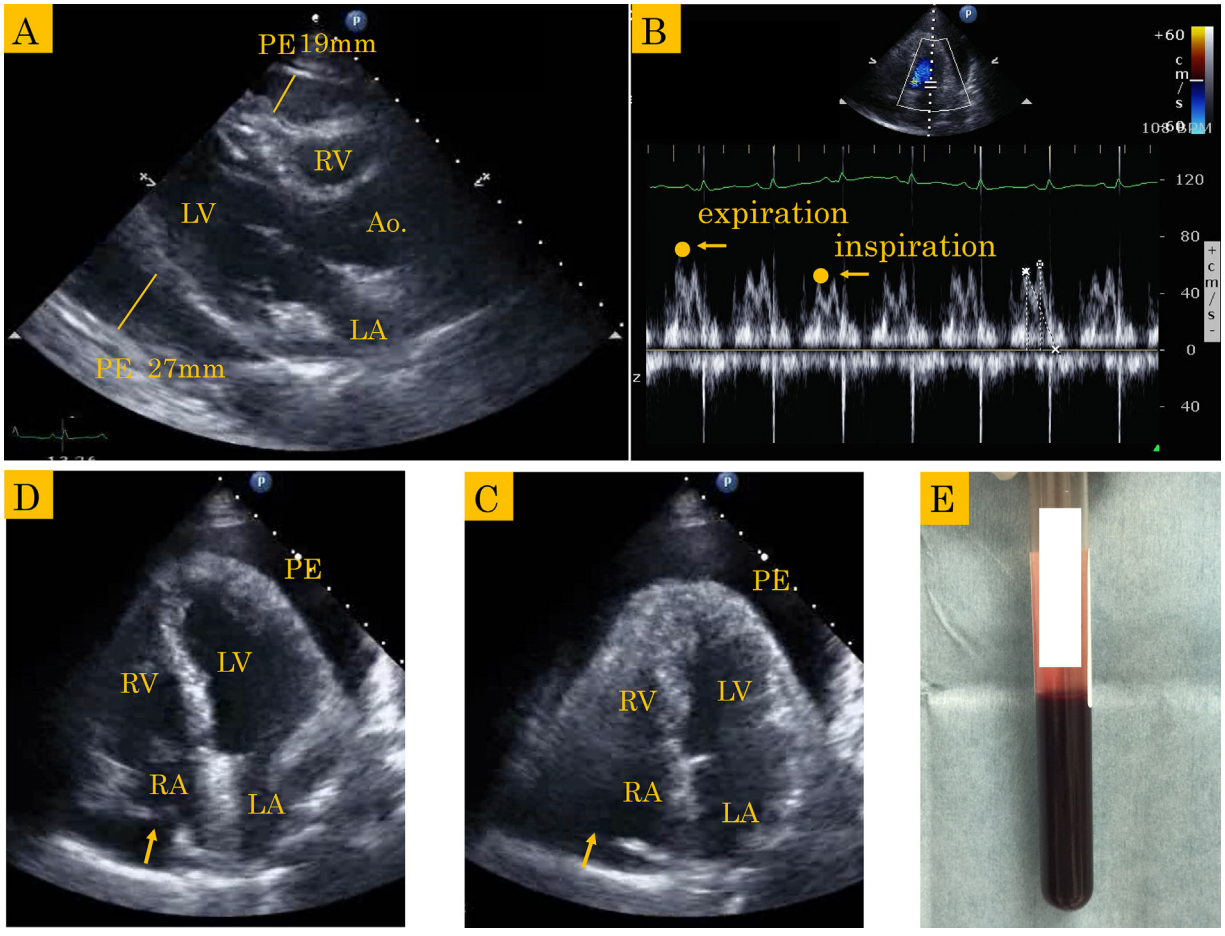

Fig. 2. (A) Transthoracic echocardiography revealed that in the end-diastolic phase on parasternal long-axis view, massive pericardial effusion was observed anteriorly and posteriorly (19 and 27 mm, respectively). (B) The transmitral E wave was 68/49 cm/sec (expiration/inspiration), and the respiratory fluctuation rate was greater than 25%. (C) During the diastolic phase, RA collapse sign was observed. On the other hand, (D) during the systolic phase, RA collapse sign was not observed. (E) Pericardiocentesis was performed for diagnosis and treatment, and bloody pericardial effusion was observed.
PE, pericardial effusion; RV, right ventricular; LV, left ventricular; Ao, ascending aorta; LA, left atrial; RA, right atrial.

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