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Case Report

Late complications of an atrial septal occluder provoked by anticoagulant therapy

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ABSTRACT

Late complications of an atrial septal occluder device (ASO) are rare but may be serious. We report a case with extensive hemopericardium five years after ASO implantation most likely triggered by anticoagulant therapy. Although not surgically confirmed, indirect clues for erosion of the atrial wall by the device were the exclusion of other etiologies, lack of recurrence after pericardial drainage and withdrawal of anticoagulants. In addition, multimodality imaging using echocardiography, computed tomography, and cardiac magnetic resonance imaging were helpful to elucidate this unusual cause. Initiation of anticoagulant treatment in patients with an ASO should be carefully balanced and may warrant more frequent echocardiographic follow-up.

<**Learning objective:** Late complications of an atrial septal occlude device (ASO) are rare. Initiation of anticoagulant therapy in patients with an ASO may lead to late hemopericardium, suggesting that more frequent echocardiographic follow-up is warranted.>

Introduction

Transcatheter closure of atrial septal defects using an atrial septal occluder (ASO) was developed almost 20 years ago as a successful minimally invasive alternative to surgical repair, leading to significant improvement in symptoms and daily activities [1,2]. Late complications of the Amplatzer ASO (St. Jude Medical, St. Paul, MN, USA) are rare and the vast majority occurs within twelve months after device implantation [2]. We report a case with extensive hemopericardium after initiation of anticoagulant treatment five years after ASO implantation.

Case report

A 51-year-old woman with obesity, inferior wall myocardial infarction, Ebstein's anomaly, and closure of an atrial septal defect type II with a 22 mm ASO device five years earlier, presented to the emergency department with progressive dyspnea and postural-independent chest pain. Because of a first episode of atrial fibrillation several months earlier, rivaroxaban 20 mg q.d. was initiated in the outpatient setting.

Physical examination revealed a normal blood pressure without paradoxical pulse, normal core temperature, soft and irregular heart sounds, no murmurs or rubbing, diminished breathing sounds over the basal lung fields, and normal abdominal findings. The electrocardiogram showed atrial fibrillation with a ventricular rate of 140 bpm, a pre-existent right bundle branch block, but new-onset low voltage amplitudes (Fig. 1A). Leucocyte count, C-reactive protein, and high-sensitivity troponin-T values were within normal limits. Transthoracic echocardiography (TTE) showed 3–4 cm circular pericardial effusion (Fig. 1B) and no signs of tamponade, a preserved left ventricular systolic function, a dilated right ventricle (RV) with impaired systolic function and massive

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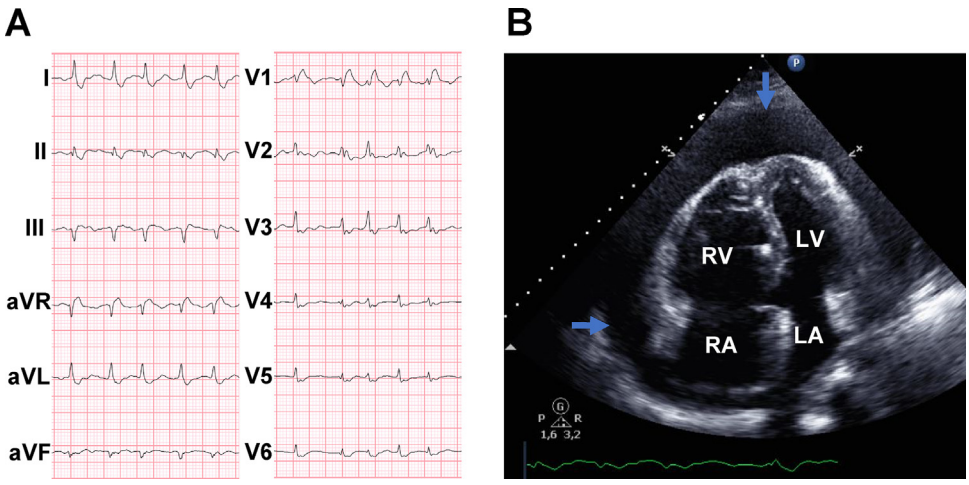

Fig. 1. Electrocardiogram showing atrial fibrillation with fast ventricular rate, pre-existing right bundle branch block, and low voltage amplitudes (panel A). Transthoracic echocardiography at the cardiac emergency department before pericardiocentesis demonstrating 3–4 cm circular pericardial effusion (blue arrows, panel B).
LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

tricuspid regurgitation that was previously considered inoperable. Via pericardiocentesis 2130 mL hemorrhagic fluid (hemoglobin level 3.0 mmol/L) was evacuated. The internationalized normalized ratio at time of first observation of the hemopericardium was 1.25. Importantly, pericardial effusion was absent on TTE three months earlier and before initiation of anticoagulant treatment.
The patient was admitted for further diagnostic work-up and rivaroxaban was terminated. An infectious cause was considered unlikely because core temperature and infectious parameters remained normal. Investigation of the evacuated hemorrhagic pericardial fluid ruled out a tuberculous, bacterial, viral, and malignant etiology. Computed tomography and cine cardiac magnetic resonance imaging showed that the ASO edges were close to the aortic (Fig. 2A) and left atrial wall (Fig. 2B), which was confirmed by transesophageal echocardiography (Fig. 3A). Although there was no proof of cardiac perforation (Fig. 3B), erosion of the atrial wall by the ASO device edges in combination with anticoagulant treatment was considered as the most likely explanation for slow onset hemopericardium. In addition to terminating anticoagulant therapy, surgical removal of the ASO was considered. However due to her co-morbidity and severe right ventricular and tricuspid valvular dysfunction, the risk-benefit ratio of surgical intervention was considered too high. She was treated conservatively without re-initiation of anticoagulant treatment. Close echocardiographic monitoring one, three, six and twelve months after discharge showed no recurrence of hemopericardium. A time course of the patient's history is summarized in Fig. 4.

Discussion

Transcatheter closure of an atrial septal defect using an ASO is increasingly being used in adult patients as a minimally invasive alternative to surgical repair and is considered a safe procedure with surgical back-up [1,3,4]. With an incidence of 0.1–0.3%, erosion of cardiac structures by an ASO is a rare but potentially

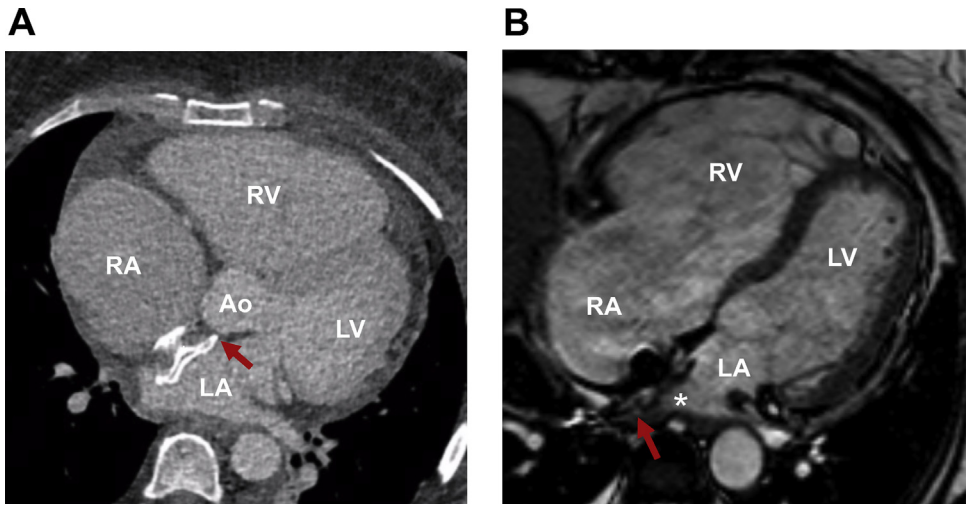

Fig. 2. Edges of the atrial septal occluder (*) impinging into the aortic and left atrial wall (red arrows) on computed tomography (panel A) and still frame cardiac magnetic resonance cine image acquired after pericardiocentesis (panel B).
Ao, aorta; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

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