Bi-atrial thrombus after occlusion of atrial septal defect with acute cerebral infarction and pulmonary embolism

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Abstract

A 49-year-old male presented to hospital with symptoms of acute cerebral infarction and pulmonary embolism who underwent transcatheter closure of atrial septal defect a year ago. Transthoracic echocardiography showed a 13×9 mm hypoechoic mass attached to the left-atrial side of the device, which was suspected to be neoplasm or thrombus. The patient was indicated for surgery after multidisciplinary discussion due to ineffective medical therapy and typical stroke and pulmonary embolism symptoms. Three-dimensional transesophageal echocardiography (3D-TEE) revealed left-atrial vegetation (21×16 mm) and right-atrial vegetation (8×6 mm) attached to the device, which were confirmed as thrombus by surgical separation and laboratory examination. This case highlights the importance of 3D-TEE and a multidisciplinary team in the diagnosis and therapy of device-related thrombus.

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Abstract

A 49-year-old male presented to hospital with symptoms of acute cerebral infarction and pulmonary embolism who underwent transcatheter closure of atrial septal defect a year ago. Transthoracic echocardiography showed a 13×9 mm hypoechoic mass attached to the left-atrial side of the device, which was suspected to be neoplasm or thrombus. The patient was indicated for surgery after multidisciplinary discussion due to ineffective medical therapy and typical stroke and pulmonary embolism symptoms. Three-dimensional transesophageal echocardiography (3D-TEE) revealed left-atrial vegetation (21×16 mm) and right-atrial vegetation (8×6 mm) attached to the device, which were confirmed as thrombus by surgical separation and laboratory examination. This case highlights the importance of 3D-TEE and a multidisciplinary team in the diagnosis and therapy of device-related thrombus.

KEYWORDS: Bi-atrial thrombus, Atrial septal defect, Transcatheter closure, Acute cerebral infarction, Pulmonary embolism

1 – INTRODUCTION

With the improvement of echocardiography, cardiac catheterization, and device materials, transcatheter closure of atrial septal defect (ASD) has gradually replaced traditional thoracotomy surgery as the optimal treatment for patients with indications. However, long-term complications of catheter-based closure, especially device-related malposition, embolization, and thrombus formation, seriously threaten the life and health of patients [1]. Bi-atrial thrombus formation-induced cerebral infarction and pulmonary embolism are rare but life-threatening late complications. Left atrial thrombus dislodges to the arteries of the brain, mesentery, and extremities, which can cause stroke, intestinal necrosis, and extremity ischemia; meanwhile, right atrial thrombus dislodges to the pulmonary artery and causes pulmonary embolism. Both conditions are common causes of acute cardiac arrest and sudden death.

2 – CASE PRESENTATION

A 49-year-old male underwent transcatheter ASD closure one year ago and was continuously treated with aspirin for long-term antiplatelet therapy. Six months ago, the patient was hospitalized for dry cough and pleural effusion and was unfortunately misdiagnosed as pneumonia. One week after the previous admission, the patient was admitted to a high-level hospital for hemoptysis and right-limb weakness and was diagnosed with pulmonary embolism. After antithrombotic and symptomatic treatment, he recovered and was discharged home with long-term rivaroxaban therapy.

At the latest admission, the patient presented with dizziness and pectoralgia and was diagnosed with acute cerebral infarction and pulmonary embolism. Physical examination revealed 3/5 strength in the right-limb without any other central nervous system abnormalities. Electrocardiogram (ECG) was normal sinus rhythm. Blood tests showed a normal complete blood count and normal coagulation function.

Brain computed tomography (CT) manifested as post-infarction of cerebral lacunar with no significant abnormalities (FIGURE 1A). Pulmonary CT angiography presented embolism in the distal of the right pulmonary artery and its branches (FIGURE 1B-C). Transthoracic echocardiography (TTE) showed a 13x9 mm hypoechoic mass attached to the left-atrial side of the device, which was suspected to be either neoplasm or thrombus (FIGURE 1D-F). There was no shunt at the atrial level and left ventricular contraction was normal. Then transesophageal echocardiogram (TEE) was recommended to further examine this mass. Three-dimensional TEE revealed fan-like vegetation (21x16 mm) attached to the left-atrial side of the device and short rod-like vegetation (8x6 mm) attached to the right-atrial side (FIGURE 2, VIDEO 1-3).

The patient was treated with low-molecular heparin anticoagulation and aspirin antiplatelet under ECG and coagulation (APTT ratio) monitoring, followed by device removal and atrial defect repair under cardiopulmonary bypass. Following the operation, double-sided vegetations were found on the device, which were confirmed to be thrombosis by laboratory tests. TTE re-examination at 5th day after operation showed the atrial septum was continuous and complete without shunts and thrombus. The patient was fully recovered and discharged home with long-term anticoagulation therapy.

3 – DISCUSSION

In this report, we present an unusual case of bi-atrial thrombus after device-based closure of ASD with acute cerebral infarction and pulmonary embolism. Bafflingly, there are no definitive guidelines for cardiac thrombus management in patients with acute cerebral infarction and pulmonary embolism. A recent paper reported a case of intracardiac thrombus that crossed the patent foramen ovale (PFO) and caused ischemic stroke, and was treated with intravenous unfractionated heparin under close neurological monitoring [2]. The patient in our case was decided to undergo surgical resection and atrial septal repair by a multidisciplinary team, including cardiology, neurology, respirology and hematology specialists. During the surgery, rivaroxaban antithrombotic therapy was switched to low-molecular heparin anticoagulation and aspirin antiplatelet treatment. Postoperative ultrasound found that there were no thrombi in the heart and limbs, except a few floating in the right internal jugular vein and subclavian artery.

Pulmonary embolism occurred 6 months after aspirin antiplatelet therapy in this patient, which may be related to incomplete endothelialization of the device, causing right atrial thrombus formation and detachment

into the right pulmonary artery. Followed by rivaroxaban antithrombotic therapy for 6 months, acute cerebral infarction still occurred, which may be due to the patient's lack of strict compliance during medication, resulting in multiple systemic thrombosis and vital organ infarction. As noted above, these complications are most likely due to the non-adherence to antiplatelet and antithrombotic therapy.

Although TTE and CT has been widely used to detect intracardiac thrombus, 3D-TEE is considered to be more superior than both, thanks to its advantages, including real-time, clarity and vividness[3]. Compared with TTE, TEE can closely observe cardiovascular system *via* esophagus, which overcomes the interference of edema, emphysema, obesity, dressings, and mechanical ventilation. Meanwhile, TEE is more suitable for intraoperative guidance and timely evaluation as compared with CT. In addition, real-time 3D-TEE provides patients with a virtual reality heart model with high temporal and spatial resolution, allowing cardiac surgeons to identify structure changes and valvular lesions.

Notably, this case needs to be differentiated from paradoxical embolization and atrial myxoma. As far as we know, acute cerebral infarction and pulmonary embolism can also be present in patients with paradoxical embolism and atrial myxoma. One distinguished feature of paradoxical embolism is the significant increase of right atrial pressure in patients with ASD or PFO, resulting in right-to-left shunt at the atrial level [4]. Moreover, the most common site of atrial myxomas is on the atrial septum, of which 75–80% located in the left atrium [5]. The usual complications of left atrial myxoma are mitral valve obstruction and left heart failure, but acute stroke is relatively rare. Pathological examination plays a key role in differentiating atrial myxoma from thrombus. Specifically, myxomas are composed of ovoid, spindle, or stellate mononuclear myxoma cells, whereas thrombi are predominantly composed of blood cells.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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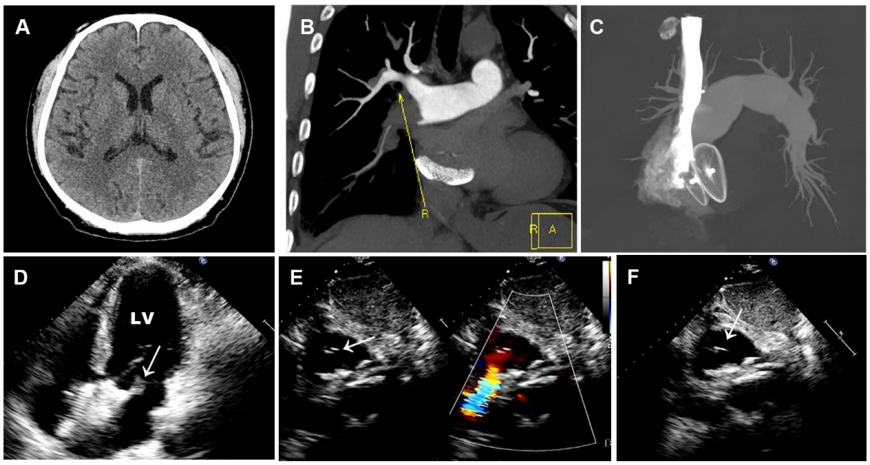

FIGURE 1 Preoperative imaging examination. (A) Brain CT manifested as post-infaction of cerebral lacunar with no other significant abnormalities. (B-C) Pulmonary CT angiography presented embolism in the distal of the right pulmonary artery and its branches. (D-F) Preoperative TTE showed a 13×9 mm thrombus floating in the left atrium without shunt. White arrows represent thrombus.

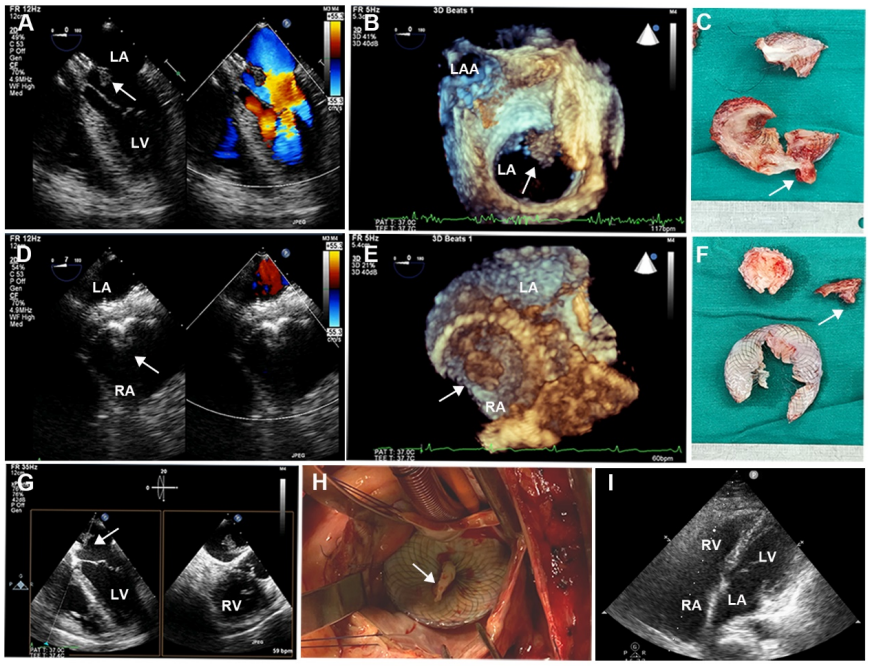

FIGURE 2 Intraoperative TEE, postoperative TTE, and surgical images. (A) and (D) 2D-TEE color Doppler showed an ASD device with double-sided thrombus. (B) and (E) 3D-TEE demonstrated a fan-like left-atrial thrombus attached to the device and a short rod-like right-atrial thrombus attached to the device. (C) and (F) Surgical resection of ASD device fragments with bilateral thrombus. (G) Biplanar TEE showed a giant thrombus floating in the left atrium. (H) Left atriotomy showed white thrombus attached to the device. (I) Postoperative TTE showed continuous atrial septum without shunt and thrombus. TEE, transesophageal echocardiography; TTE, transthoracic echocardiography; ASD, atrial septal defect. White arrows represent thrombus.

VIDEO 1 Mid-esophageal four chamber biplanar view demonstrating a giant thrombus floating in the left atrium.

VIDEO 2 Three-dimensional TEE left atrial view showing a giant thrombus attach to the left-side of the device.

VIDEO 3 Three-dimensional TEE showing a right atrial thrombus attach to the device.