



Surgical embolectomy for paradoxical cerebral embolism with massive pulmonary embolism

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Abstract

The patient was a 68-year-old woman with a history of multiple cerebral emboli. The patient presented with dysarthria, dysphagia, and left facial paralysis. Emergent MRI demonstrated multiple emboli in the brain stem and left occipital lobe. Echocardiography and chest CT demonstrated floating emboli entrapped in the foramen ovale and pulmonary emboli in both main pulmonary arteries. Emergent pulmonary embolectomy and extraction of the emboli on the foramen ovale were successfully performed without neurological exacerbation.

Keywords Pulmonary embolism · Cerebral embolism · Paradoxical embolism · Pulmonary embolectomy · Venous thromboembolism

Introduction

Pulmonary embolism (PE) is one of the major causes of cardiovascular mortality in Western countries. Right heart thrombi are present in 4–8% of patients having PE, with a high mortality of 20–25% [1–3]. The reason for the high mortality is that there is frequently a secondary episode of fatal and massive PE. Paradoxical embolism is an unusual cause of systemic embolism observed in patients having a patent foramen ovale (PFO). A successfully treated case of paradoxical embolism with massive PE plus saddle embolism on the PFO managed by surgical embolectomy is presented.

Case report

The patient was a 68-year-old woman with a history of multiple cerebral emboli 2 years prior to admission. She had been taking prednisolone sodium for 39 years to control systemic lupus erythematosus. The patient developed sudden onset of dysarthria, dysphagia, and left facial paralysis. Emergent MRI demonstrated multiple emboli in the brain stem and left occipital lobe (Fig. 1). There was no swelling of the brain. The patient showed hypoxemia and tachycardia with normal blood pressure after admission. Echocardiography demonstrated a flail, worm-like thrombus straddling the atrial septum (video 1). Moderate right ventricular dysfunction with moderate tricuspid regurgitation, pulmonary hypertension, and normal left ventricular contraction was also demonstrated. Contrast-enhanced computed tomography showed multiple emboli in bilateral main pulmonary arteries (Fig. 2) and venous thrombosis of the right superficial femoral vein. Blood tests showed a normal platelet count, elevated D-dimer (100.6 µg/ml), and elevated fibrinogen degradation products (421 µg/dl). Lupus anticoagulant was positive. A diagnosis of PE and multiple cerebral emboli was made. The patient underwent emergent pulmonary embolectomy on day 2 after stroke onset. Under bicaval venous drainage, aortic perfusion, and cardioplegic arrest, the right atrium was incised. Fresh, sausage-shaped thrombi (10 mm erythematosus × 300 mm) were entrapped in the PFO. The thrombi were extracted from the interatrial

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Fig. 1 MRI of the brain shows multiple emboli to the right occipital lobe (a) and brain stem (b)

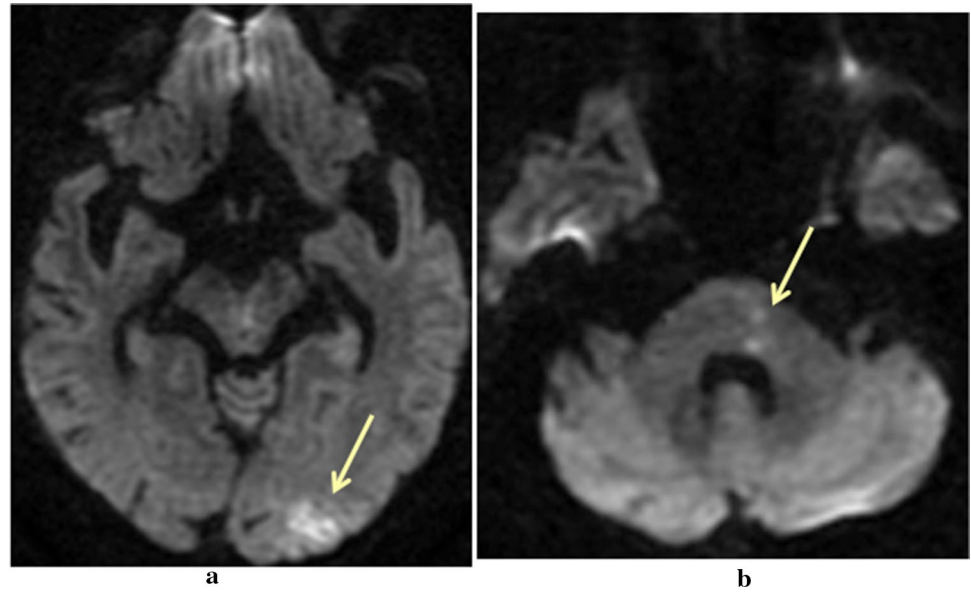


Fig. 2 a Enhanced CT of the chest shows pulmonary emboli to bilateral main pulmonary arteries. b Leg CT shows floating thrombus in the right femoral vein

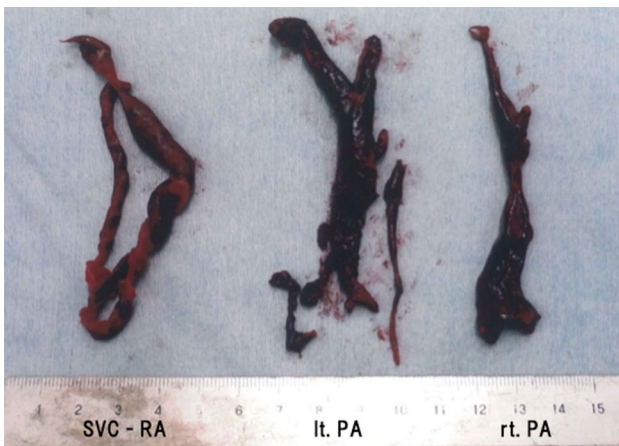
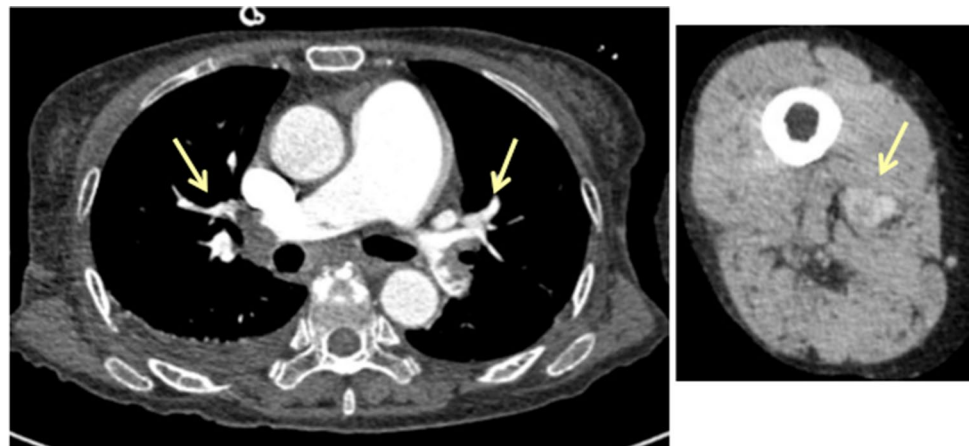


Fig. 3 Extracted thromboemboli

septum through a septal incision (Fig. 3, Video 2). The left atrium was thoroughly inspected through the septal incision to confirm no residual thrombi in the left atrium. The foramen ovale and septostomy were closed with continuous 4-0 polypropylene suture. Pulmonary emboli were extracted from the pulmonary trunk and the right main pulmonary artery. Aortic crossclamp time, cardiopulmonary bypass time, and operation time were 33 min, 64 min, and 217 min, respectively. The patient underwent placement of a retrievable caval filter for residual deep venous thrombosis on post-operative day 2, which was retrieved 2 weeks after operation. There were no complications related to pulmonary embolectomy. The neurological deficit improved with rehabilitation, and the patient was discharged on anticoagulant therapy.

Discussion

The prevalence of PFO in healthy adults is 25%, and it may cause recurrent cerebral emboli due to a transient reverse shunt by coughing or the Valsalva maneuver. When the patient with a PFO has acute massive or sub-massive PE, elevated right ventricular end-diastolic pressure may increase right atrial pressure, causing a persistent right-to-left shunt through the PFO. It also exacerbates hypoxemia. The reverse shunt may induce secondary systemic embolization. Management of PE with a right heart thrombus is controversial. Some physicians regard it as a transitory form of PE; therefore, they insist that surgical intervention is not necessary. Rose et al. reviewed 177 reported cases with right heart thrombi, 98% of which had associated PE. Among them, 9% of patients received no treatment, 20% received anticoagulation, 35% received thrombolytic agents, and 36% underwent surgical embolectomy, with respective mortalities of 100%, 28.6%, 11.3%, and 23.8% [4]. A thrombus entrapped on the PFO is more complicated. According to a review of 88 patients with right heart emboli by Fuaveau et al., surgical embolectomy was the most common treatment for saddle embolism on a PFO; however, thrombolysis is generally performed in unstable patients with severe PE [5]. PE was present in 94% of their cases, including severe PE in 34%. Paradoxical systemic embolism was found in 44% of cases. Fifty-five patients underwent pulmonary embolectomy, 21 were treated with heparin, and 11 received thrombolysis, with respective mortalities of 13%, 14%, and 36%. Of 26 patients with stroke, 14 underwent pulmonary embolectomy. Dzudovic et al. reported a case of saddle embolism entrapped on the PFO. The patient underwent full heparinization; however, the patient developed an ischemic stroke on the 5th day of presentation, just a few hours before the scheduled surgery [6]. Because the outcome of medical therapy is uncertain, urgent embolectomy is a reliable treatment for the patient having entrapped embolus with systemic and massive pulmonary embolism.

Recent literature showed that thrombolytic therapy does not improve survival in patients having massive PE, because hemorrhagic complications offset the release of right ventricular afterload. Thrombolytic therapy may also lyse entrapped thrombus into small fragments, causing secondary arterial embolism. We regarded thrombolytic therapy had a risk of cerebral hemorrhage or recurrence of systemic embolism in the presented case. Anticoagulation therapy is an alternative to these treatments if patients have comorbidities or stroke. Because the present case had a previous history of stroke suggesting recurrent episodes of paradoxical emboli, surgical embolectomy was performed. Surgical pulmonary embolectomy is safe for

patients having a minor stroke, because the anticoagulant burden for the operation is short. Minakawa et al. analyzed 355 patients undergoing pulmonary embolectomy and showed that a history of stroke is a favorable predictor for survival after pulmonary embolectomy; they also showed that independent risk factors for death were poor left ventricular function and respiratory failure. Poor left ventricular function, preoperative cardiopulmonary resuscitation, and respiratory failure were independent risk factors for either death or neurological deterioration [7]. Myers et al. showed that the 30-day mortality of biatrial thromboemboli entrapped in the foramen ovale was 18.4%. They showed that age, coma, and systemic embolization were risk factors for death on univariate analysis. They also showed favorable outcomes following surgical embolectomy [8].

Paradoxical arterial embolism accompanying acute PE is sometimes asymptomatic or shows subtle neurological symptoms, as in the present patient [9]. However, hemorrhagic transformation of cerebral infarction by thrombolytic therapy may exacerbate neurological symptoms.

Conclusion

Pulmonary embolectomy is an effective treatment and should be considered for paradoxical embolism with massive PE.

Compliance with ethical standards

Conflict of interest The authors declare they have no conflict of interest.

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