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aortic paradoxical embolus with subclavian artery occlusion**

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Pseudo-high risk acute pulmonary embolism resulting from a large saddle aortic paradoxical embolus with subclavian artery occlusion

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A 75-years-old woman with acute dyspnea was diagnosed for suspected acute pulmonary embolism (APE). Initially, she was stable with blood pressure of 135/80mmHg, tachycardia of 105bpm and oxygen blood saturation of 84%. Computed tomography pulmonary angiography (CTPA) showed a bilateral central pulmonary embolism and an enlarged right ventricle (RV). After this examination, a sudden drop in systolic blood pressure to 80mmHg was reported. She was classified as a high-risk APE and referred immediately to a tertiary cardiology care unit.

On admission to our department, the patient was in good general condition without peripheral hypoperfusion. Her left hand was colder without a palpable pulse, however without signs of acute ischemia. A difference of 60mmHg in systolic blood pressure between upper extremities

was found. Reassessment of previously performed CTPA identified a large (14cm) aortic thrombus lodged into the left subclavian artery, with the free part extending to the distal aortic arch. (Figures A, B). Echocardiography showed not only RV overload typical of APE but also massive right-to-left shunting via patent foramen ovale (PFO) following intravenous injection of agitated saline (Figures C-E).

The patient was consulted by the pulmonary embolism response team (PERT). Parenteral anticoagulation was continued. To prevent arterial embolization in case of aortic thrombus fragmentation, she was submitted for urgent surgery. The procedure was performed in hypothermia of 28 degrees C, bilateral pulmonary embolectomy was performed with complete circulatory arrest 7 minutes long. Additionally, a large thrombus, probably a venous cast lodged in the left subclavian artery, was removed from the aortic arch, which required circulatory arrest for 3 minutes with selective cerebral perfusion through the right carotid artery (Figure F). Eventually PFO was sutured. The patient was discharged home in good general condition on long-term oral anticoagulation with no clinical signs of peripheral embolization 10 days after successful surgery.

Paradoxical embolism is a well-known complication of APE resulting from right-to-left intracardiac shunt mostly via PFO leading to systemic embolization by thrombi originating from the venous system.[1] Of note, ischemic stroke due to paradoxical embolism occurred in patients with acute APE and RV dysfunction.[2] This considerable complexity requires multidisciplinary approach, established within PERT.[3]

We report a patient with intermediate-high APE and very large aortic thrombus successfully treated with simultaneous pulmonary and aortic surgical embolectomy. The optimal management of saddle emboli located in the aortic arch is still to be determined. Although such lesions can be successfully treated conservatively, anticoagulation may result in displacement of fragments and further complications, e.g. ischemic stroke or peripheral

embolization.[4] Despite finding only one report of successful aortic arch thrombolectomy with simultaneous pulmonary artery embolectomy, we think that surgical management should be preferred.[5]

Our case demonstrates the need for awareness of the risk of systemic embolism in patients with APE and RV overload. Hence, the crucial role of early echocardiography in screening for thrombi in transit, particularly in patients who may require percutaneous interventions. It also emphasizes the role of a thorough physical examination, which revealed the cause of low blood pressure values and led to a change in treatment. Eventually, it shows that surgical treatment of APE is an effective method and should be considered in the presence of additional indications.

Article information

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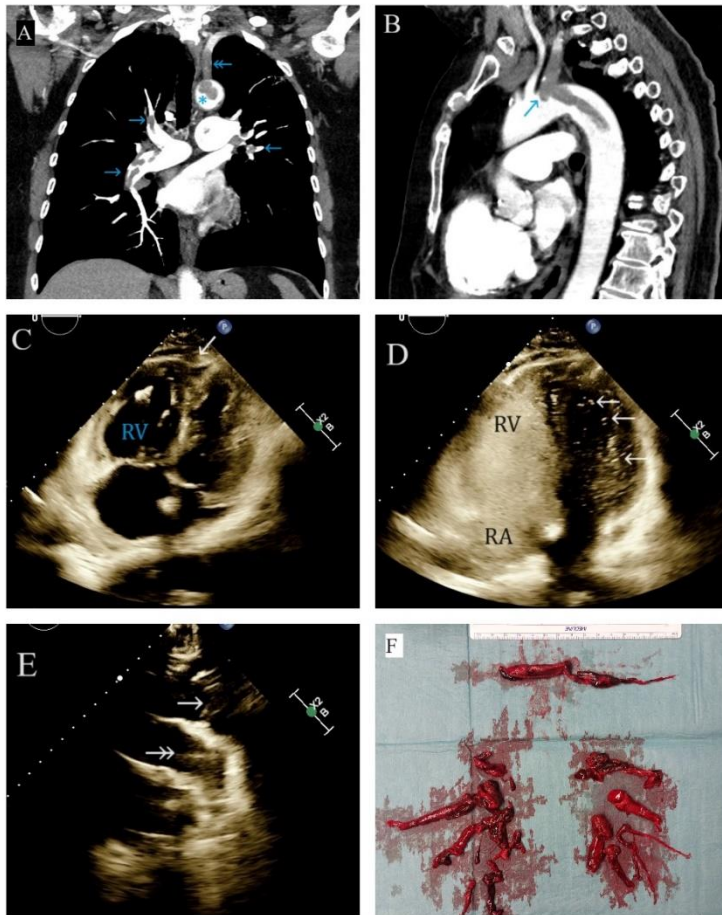


Figure 1 A – Computed tomography pulmonary angiogram, frontal reconstruction showing multiple emboli within pulmonary arteries (arrows) and aortic arch (asterisk), with contrast filling defect of left subclavian artery (double arrow); B – computed tomography pulmonary angiogram, sagittal reconstruction showing large saddle embolus lodged in aortic arch (arrow); C – right ventricle (RV) dilatation with prominent apical contraction (arrow) as in the McConnell sign; D – agitated saline filling the right atrium (RA) and RV, with bubbles in the left ventricle (arrows) during the second heart cycle; E – suprasternal view of aortic arch with a thrombus (arrow) propagating to the left subclavian artery, and left pulmonary artery with another clot within its lumen (double arrow); F – surgical specimens, demonstrating multiple thrombi from the pulmonary arteries (below) and the 15 cm long thrombus from the aortic arch (top)

Short title: Acute pulmonary embolism with large saddle aortic paradoxical embolus