Thrombus-in-transit

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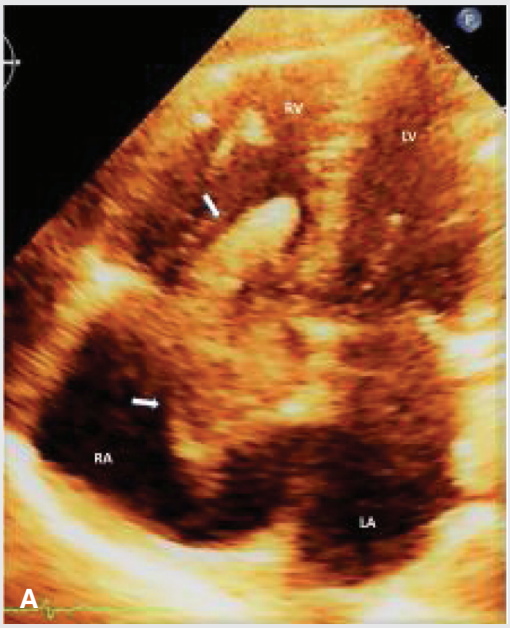
CASE

An 83-year-old woman with a past medical history of atrial fibrillation, rheumatoid arthritis, and obesity presented to the emergency center (EC) with complaints of generalized weakness, dyspnea on exertion, and dizziness for approximately two weeks. The patient was hemodynamically stable with an oxygen saturation of 93-97% on 3 L/min oxygen per nasal cannula. On initial laboratory tests, a D-dimer level was elevated at 8,815 ng/mL. Computed tomography with angiography of the chest identified pulmonary embolism (PE), and a venous duplex scan of the left lower extremity identified acute-on-chronic venous thromboembolism in both deep and superficial veins. A transthoracic echocardiogram (Video) showed paradoxical interventricular septal motion, right ventricular overload, and a large mobile structure primarily located in the right atrium with projection into the right ventricle (Figure 1A). Left ventricular diastolic and systolic function was normal. The patient underwent emergent atriotomy to remove a tubular clot measuring approximately 16.5 cm in length (Figure 1B). Post-surgical abdominal imaging consisted of a renal ultrasound which showed small bilateral renal cysts.

Three weeks prior to the EC presentation, the patient had undergone right-sided parotid pleomorphic



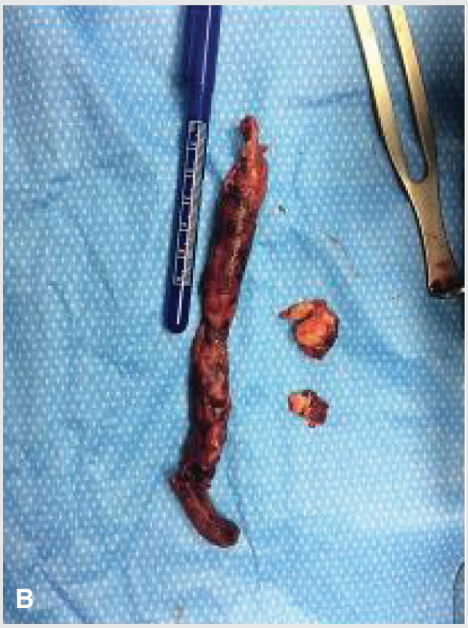


Figure 1A. Echocardiogram image of clot present in the right atrium with projection into the right ventricle. White arrows indicate thrombus. RV- right ventricle, RA- right atrium, LV- left ventricle, LA- left atrium

Figure 1B. Tubular clot approximately 16.5 cm × 2.0 cm after surgical removal from the right atrium

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adenoma resection. Following this surgical procedure, the patient spent her time mostly resting in bed due to fatigue. On further review of the medical history, the patient shared that she had been off warfarin for atrial fibrillation for over one year due to her INR's being too difficult to manage. She recovered well from the thrombectomy and was discharged home on apixaban.

Discussion

Three classes of cardiac thrombi, based on morphology, have been described: type A, a mobile, tubular structure; type B, a less mobile, mural structure; and type C, a mobile, amorphous structure.[1,2] Type A thrombi, also referred to as thrombi-in-transit, arise from deep venous thromboses and are associated with severe pulmonary embolisms and high mortality rates.[1,3,4] The actual prevalence of right heart thrombi in patients with PE remains an open question. However, one study suggests it may be as high as 20%.[3] When left untreated, the mortality of PE associated with type A thrombi approaches 100%.[3,4] Additionally, in the setting of a patent foramen ovale, type A thrombi can cross to the left heart causing arterial complications.[5,6] Treatment options include anticoagulation, thrombolytics, and surgical thrombectomy. There are no clear guidelines recommending treatment; physicians must act quickly in these cases.

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