CASE REPORTS/CLINICAL VIGNETTES

The Simultaneous Occurrence of Deep Vein Thrombosis and Pulmonary and Arterial Embolization

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INTRODUCTION: A 45-year-old female presented with 8 hours of right lower extremity pain and dyspnea. She was tachycardic and her right lower extremity was dusky, cold, and pulseless.

DISCUSSION: Computerized tomography of the venous and arterial systems revealed massive pulmonary embolism and right lower extremity arterial and left lower extremity venous thromboses. Management included intra-arterial thrombolytics, right lower extremity arterial embolectomy, and anticoagulation. Echocardiogram revealed a patent foramen ovale (PFO) with a right-to-left shunt.

CONCLUSION: We hypothesize that our patient developed deep venous thrombosis that led to major pulmonary embolization and increased right-sided pressures. In the setting of a patent foramen ovale, a later venous embolus transversed the PFO and lodged in the femoral artery. Our case illustrates the importance of a high index of suspicion for a paradoxical embolus in patients with arterial thrombosis.

KEY WORDS: paradoxical embolism; deep venous thrombosis; patent foramen ovale.

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INTRODUCTION

In adults, a patent foramen ovale (PFO) has been implicated in the etiology of migraine headaches, decompression sickness in underwater divers, cryptogenic stroke, and other arterial embolization¹. These phenomena are thought to be related to chemicals, bubbles, or thrombotic material escaping to the arterial circulation across this pathway. The presence of a paradoxical embolus signifies the need for a thorough diagnostic evaluation, including consideration of a PFO². We present a case which exemplifies in unusual detail the mechanism of the traversal of a clot from the lower venous system to the lower arterial system.

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CASE DESCRIPTION

A 45-year-old obese female presented to the emergency department with 8 hours of right lower extremity pain and shortness of breath after having been hospitalized 4 days previously for an asthma exacerbation. She described the pain in her right leg as initially burning in character, which then progressed to cramping and numbness. She admitted to being relatively non-ambulatory after her discharge from the hospital. Her past medical history was significant for asthma for which she used an albuterol inhaler. She denied pregnancy, had normal menstrual cycles, and denied recurrent abortions or a family history of blood clots. She was currently not smoking although she quit 18 years ago. She denied the use of oral contraceptives and over-the-counter or herbal medications.

She was tachycardic (pulse: 126 beats per min) with an oxygen saturation of 92% on room air, a body temperature of 97.1°F, a respiratory rate of 18 per minute, and a blood pressure of 108/86 mmHg. Her body mass index was 32 kg/m². She had no cardiac murmurs, and her lungs were clear to auscultation. Her right leg was very painful, mildly swollen, dusky, cold, and pulseless. Her left leg was mildly tender but had normal pulses. Her complete blood count and comprehensive metabolic profile were normal.

Chest radiography showed no acute cardiopulmonary disease, and the electrocardiogram was significant for sinus tachycardia without ST-T wave changes. Contrast-enhanced computed venous tomography showed both a left popliteal vein thrombosis and extensive pulmonary embolization, including filling defects in both lower lobe pulmonary arteries extending into the segmental branches, filling defects in both upper lobe pulmonary arteries, and a saddle embolus. (Fig. 1) A computed tomography (CT) arteriogram of the lower extremities showed diminished arterial perfusion in the right common femoral artery. Initial management included intravenous fluids, inferior vena cava filter, and intra-arterial thrombolytic therapy for the arterial embolus. Because of lack of improvement, an embolectomy was performed which retrieved a 6.5-cm long clot; she was anticoagulated.

A transthoracic echocardiogram with bubble study showed a patent foramen ovale with a right-to-left shunt. The right ventricle was mildly dilated, and the mean pulmonary artery pressure was estimated to be 19 mmHg (normal <18). Hypercoagulability studies demonstrated a prolonged partial thromboplastin time (PTT) that did not correct with the mixing study, a positive hexagonal phase neutralization assay (normal: negative), and a positive dilute Russell Venom Viper test

Figure 1. Computerized tomographic image demonstrating saddle pulmonary embolus

(normal: negative) (latter two studies performed at Esoteric Laboratory Services Inc., Brentwood, TN). Factor II mutation, Factor V Leiden, Protein C, Protein S, and Cardiolipin studies (IgA, IgG, IgM) were normal. Homocysteine concentration was mildly elevated at 15 $\mu\text{mol/L}$ (normal <10), and D-dimer was elevated to 3.6 $\mu\text{g/mL}$ (normal <0.5). The patient was discharged to a rehabilitation facility on oral anticoagulation. She received physical therapy and regained pulses in her right lower extremity.

DISCUSSION

The triad of PFO, deep venous thrombosis, and increased right-sided pressure is a milieu that enables the occurrence of a paradoxical embolism¹. A pulmonary embolus, especially a large one, may be a proximate cause of increased pulmonary artery pressure that propagates in a retrograde fashion to the right ventricle and the right atrium, causing a gradient of pressure across the PFO that leads to a right-to-left shunt. In this setting, all that is needed is for another bit of venous thrombus to dislodge and follow this pressure gradient, and the clot will reach the left circulation rather than the lung. This is the presumed mechanism for the occurrence of the large femoral embolus recovered from our patient. Our case is unusual in that all the stigmata consistent with this mechanism were present at the time of diagnosis: (1) the venous clot, (2) the extensive pulmonary embolization, (3) the arterial thrombus, (4) the PFO, and (5) the accompanying estimate of an increased pulmonary artery pressure on echocardiography.

In most situations, the pathological quintet that we saw in our patient will not be present. The venous embolus may entirely dislodge, and there may be no remaining clot in the venous system at presentation. In addition, a pulmonary embolus need not be the mechanism of the increased right-sided pressure. For example, the patient may have chronic

elevation of right-sided pressure related to chronic obstructive pulmonary disease—a known risk factor for venous thrombosis³. In fact, elevation of right-sided pressure may not be demonstrable after arterial embolization: the pressure may have only temporarily increased at the crucial time that the embolus reached the right atrium (for example, secondary to an ill-timed valsalva maneuver), or the increased pressure may be resolved by the time that the echocardiogram is performed (for example, after treatment of pulmonary embolization). In our case, the tip off to the pulmonary embolization was the patient's complaint of dyspnea.

Our patient had at least two risk factors for deep venous thrombosis. She had been recently hospitalized and had been at bed rest for most of the time between discharge and rehospitalization, and she was obese. In addition, she may have an underlying hypercoagulable state, as suggested by abnormalities in her hospital laboratory studies. Even if these abnormalities are not confirmed, many would argue that this patient with a known PFO and a large paradoxical embolus should be on life-long anticoagulation unless the PFO is closed^{4,5}. A finding of a hypercoagulable state might have implication for thromboembolism prophylaxis for family members, however.

CONCLUSION

This case is quite unusual in that it demonstrates in remarkable detail the pathological mechanism underlying paradoxical embolization. Most cases will not have such a complete Hansel-and-Gretel trail. Paradoxical embolization should be considered in all cases of arterial embolization and strokes, especially in younger adults.

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