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Paradoxical Embolism After a Traffic Accident: A Rare Case of Thrombus Entrapped in a Patent Foramen Ovale

Trafik Kazası Sonrası Gelişen Paradoks Emboli: Nadir Görülen Bir Olgu, Patent Foraman Oavalede Trombüs Tuzaklanması

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ABSTRACT

Paradoxical embolism is the passage of a thrombus formed in the venous system through shunts in the lung or heart into the systemic circulation. The most common intracardiac shunt is a patent foramen ovale (PFO). Since the transition of a thrombus formed in the right heart to the left heart and systemic circulation through the PFO is a temporary situation, the entrapped thrombus in the PFO is extremely rare. Herein, we present a case, including the diagnosis and treatment, of a paradoxical embolism in a 53-year-old female with a non-vehicle traffic accident who developed a pulmonary embolism and acute cerebral infarction.

Keywords: Paradoxical embolism, entrapped thrombus in PFO, surgical treatment in paradoxical embolism

ÖZ

Paradoks emboli akciğer veya kalpteki şantlar aracılığıyla venöz sistemde oluşan bir trombüsün sistemik dolaşıma geçmesi olarak bilinir. En sık görülen intrakardiyak şant patent foramen ovaledir (PFO). Sağ kalpte oluşan bir trombüsün PFO aracılığıyla sol kalbe ve sistemik dolaşıma geçişi geçici bir durum olduğundan PFO'daki trombüs tuzaklanması son derece nadir görülür. Burada araç-dışı trafik kazası sonrası pulmoner emboli ve akut serebral infarktüs gelişen 53 yaşındaki bir kadın hastadaki paradoksal embolinin tanı ve tedavisiyle ilgili bir olgu sunuyoruz.

Anahtar Kelimeler: Paradoks emboli, PFO'da trombüs tuzaklanması, paradoks embolide cerrahi tedavi

Introduction

Paradoxical embolism is a clinical situation in which a thrombus originating from the venous system passes into the systemic circulation through an intracardiac or pulmonary shunt (1). The two most common causes of paradoxical embolism are a patent foramen ovale (PFO) and arteriovenous malformation (2). The clinical diagnosis is based on detecting an arterial embolism due to an intracardiac defect or pulmonary arteriovenous shunt with the source of the venous embolism (3). Depending on the location of the embolism, neurological deficits due to ischemic stroke, chest pain, and electocardiographic changes due to myocardial infarction, acute abdominal pain due to gastrointestinal ischemia, back pain and hematuria due to renal infarction, and pain and coldness in the extremities due to peripheral arterial occlusion may occur.

A PFO is a cardiac anomaly that can be detected in approximately 25% of the general population. It occurs due to septum primum and septum secundum not fusing after birth and is a strong risk factor for paradoxical embolism. On the other hand, an atrial septal aneurysm (ASA) is an anomaly in the fossa ovalis region. The atrial septum protrudes into the right or left atrium by 1 cm or more. It is rarely seen in the general population, with a frequency of 2% compared with that of a PFO (3,4). The coexistence of PFO and ASA is associated with a significantly increased risk of cerebrovascular events compared with the risk of either seen alone (5). Since the transition of the thrombus from the right heart to the left heart through a PFO is a temporary situation, the image recording of a thrombus trapped in a PFO is extremely rare.

A PFO is closed passively under normal physiological conditions due to the pressure difference between the left and right atria. In Valsalva maneuvers (coughing, sneezing, straining, urination, others), the right atrial pressure can surpass that of the left atrial pressure, temporarily creating a shunt from right to left and creating a paradoxical embolism through the PFO. A pulmonary embolism and other causes of pulmonary hypertension may create a paradoxical embolism by making a shunt temporarily and permanently in the presence of a PFO.

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Herein, we present a case, including diagnosing and treating a paradoxical embolism in a 53-year-old female patient with a non-vehicle traffic accident who developed a pulmonary embolism and acute cerebral infarction.

Case Report

The consent of the patient and her relatives was obtained provided that their medical data would be used only for scientific purposes, and their identity information was kept confidential.

A 53-year-old female patient with diabetes was brought to the emergency room by the emergency team due to a non-vehicle traffic accident. Her first evaluation revealed a large hematoma in her right occipital region, a non-displaced pelvic fracture, and a left humerus fracture. Her physical examination showed that her blood pressure was 130/80 mmHg, pulse was 85/min, her consciousness was clear, central imaging was normal, and her respiratory system examination was normal. The patient had stable vital signs and was directed to orthopedic surgery for the humerus fracture.

We were consulted after the patient was operated on for the left humerus fracture because she experienced sudden shortness of breath, tachypnea, and hypotension during the orthopedic service follow-ups. The patient had previously consulted neurology due to short-term loss of consciousness and speech disorder, and a transient ischemic attack was considered according to her brain imaging. The patient's ECG was taken at 120/min sinus tachycardia, blood pressure was 100/50 mmHg, and her bedside echocardiography showed that the ejection fraction was normal. However, the right heart cavities were enlarged, and the right ventricular functions regressed. Pulmonary computed tomographic pulmonary angiography (CTA) was requested, with a preliminary diagnosis of a massive pulmonary embolism. Her CTA showed that a filling defect compatible with an acute embolism in the right main pulmonary artery, pulmonary branches leading to the right upper lobe, branches leading to the bilateral lower lobe, and segmental-subsegmental branches. The patient was taken to the cardiology intensive care unit. Thrombolytic therapy was not considered because the patient had a recent history of surgery and cerebrovascular events. Percutaneous mechanical intervention was not considered because of her elevated blood pressure after intravenous fluid administration, and the patient was hemodynamically stable. So, low molecular weight heparin was administered to the patient.

In the control echocardiography of the patient, the ejection fraction was normal, and the right heart spaces were wide, the right ventricle apical akinetic, and the trapped thrombus appearance was observed in the PFO extending from the right atrium to the left atrium. In transesophageal echocardiography (TEE), which was later performed to detect the thrombus, the thrombus was seen in both atria trapped in the PFO. The fragment in the right atrium was in two parts, and the longest length was 1.7 cm, and the one in the left atrium extended into the left ventricle was 9 cm (Figure 1, 2; Video 1, 2).

Whether to have the surgery was left to the patient, who was considered high risk for thrombolytic therapy. Although the patient and her relatives were told about the embolic risks that the patient might experience, they did not accept the surgical procedure. Afterward, low molecular weight heparin treatment was stopped, and a heparin infusion was started. The surgical option was re-explained upon recurrent consciousness and speech impairment in the patient's follow-up and general diffusion restrictions in both brain parenchyma suggesting an embolic infarction in central imaging. When the patient and her relatives accepted the surgical procedure, the patient was transferred to cardiovascular surgery. Surgery was performed to close the PFO, remove the intracardiac thrombus, and install an inferior vena cava.

After surgery, the patient's hemodynamics improved rapidly, and no complications developed. The patient was evaluated one month after discharge. Her control echocardiography showed no intracardiac thrombus image, her right heart cavities regressed, and her RV contraction had improved.

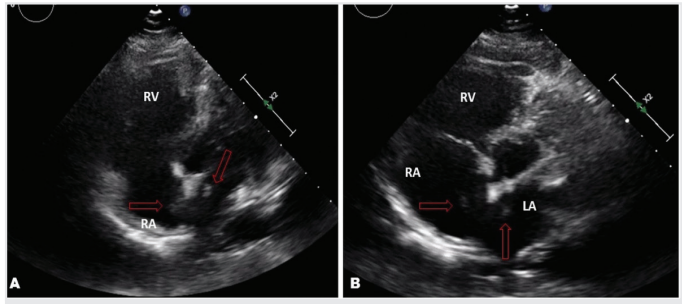

Figure 1. Transthoracic echocardiography demonstrating hyperechogenic density images of a trapped thrombus image in the interatrial septum with enlargement of the right ventricle and the right atrium (A, B)

RV: Right ventricle, RA: right atrium, LA: left atrium

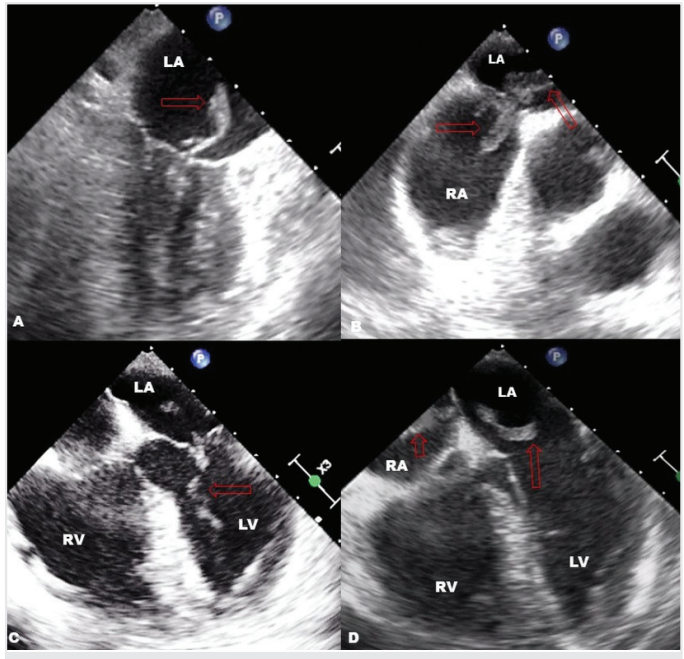

Figure 2. Transesophageal echocardiography demonstrating a large and long trapped thrombus image in the interatrial septum with an enlargement of the right ventricle and the atrium that extends from the mitral valve to the left ventricle (A-D)

LA: Left atrium, RA: right atrium, RV: right ventricle, LV: left ventricle

Discussion

Interest has increased in diagnosing PFO, which is a common condition found in 25% of the general population, after its association with paradoxical embolism. In particular, advances in TEE have played an essential role in the diagnosis of PFO.

An entrapped thrombus in the PFO is rarely seen. TEE is a highly reliable and excellent imaging method for diagnosing a trapped thrombus. Computed tomography is essential for evaluating the heart and examining other organs that can be reached by an embolism, such as the lungs and the brain.

No clear consensus exists in PFO regarding patient management and treatment planning for a trapped thrombus. Thrombolytic therapy, anticoagulant therapy, surgical treatment, and percutaneous treatments were applied individually or in combination in various clinics in different patient groups. An inferior vena cava filter is generally recommended when a high risk of mortality exists due to a massive pulmonary embolism with contraindications to anticoagulant therapy and inadequate anticoagulant therapy (6).

In a study, 84 patients with a trapped thrombus in a PFO were investigated. Of these, 55 were treated surgically, 21 were treated with heparin, and 11 were treated with thrombolytic therapy. The mortality rates for the treatments were 13%, 14%, and 36%, respectively (7).

Consequently, heparin treatment may be an option in patients with high comorbidity and cerebrovascular events and has mortality rates similar to surgical treatment. Thrombolytic therapy is chosen more frequently in the high-risk group but is associated with higher mortality (7). Also, systemic thrombolytic therapy and systemic anticoagulant therapy may present a high risk by causing rupture of the thrombus and hemorrhagic transformation of large ischemic strokes. Closure of the PFO following surgical thrombectomy for paradoxical embolism and entrapped thrombus has become the favored method in high-risk patients. More extensive studies with randomized clinical trials comparing acute thrombectomy with conservative medical therapy are required.

In this case, we evaluated thrombolytic treatment as a high risk in the patient's recent history of cerebrovascular events and multiple traumas. Therefore, the surgical treatment option was considered in the foreground. Another risk factor for thrombolytic therapy was the breakdown of the intracardiac thrombus and its embolism to the lung and systemic circulation. The inferior vena cava filter was applied because of the high mortality of the pulmonary embolism that the patient would experience due to recurrent venous thromboembolism.

Conclusion

Paradoxical embolism can lead to life-threatening situations by causing a pulmonary and systemic embolism. An elevated pressure gradient between the right and left atria increases the risk of systemic embolism. Clinical suspicion is the most important step in making a diagnosis. Since it mostly progresses with pulmonary embolism, paradoxical embolism must be included in the differential diagnosis of pulmonary embolism and systemic embolism. Since a missed diagnosis typically leads to a fatal outcome, early diagnosis and treatment are essential to prevent mortality and morbidity.

Ethics

Informed Consent: The consent of the patient and her relatives was obtained provided that their medical data would be used only for scientific purposes.

Peer-review: Externally and internally peer-reviewed.

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