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Paradox Embolism through Patent Foramen ovale with Acute Peripheral Arterial Occlusion and Severe Pulmonary Embolism

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Introduction

Pulmonary embolism (PE) is a common disease, especially after orthopedic or trauma surgery. It is associated with a considerable mortality rate. Early diagnostic and the use of therapeutically anticoagulation are essential. In cases of compromised circulation due to fulminant PE, thrombolytic medication or surgical and interventional thrombectomy are possible treatments that can reduce mortality and improve patients' outcome. Transthoracic echocardiography (TTE) is the diagnostic method of choice to discriminate right ventricular function as indicator for which treatment is needed [1–5].

In cases of cryptogenic embolism, further peripheral or cerebral diagnostics in form of transesophageal echocardiography (TEE) should be performed [6].

With this report we want to present a case of fulminant PE combined with a paradox embolism of the brachial artery sourced in a thrombotic embolus with echocardiographic verification of reaching through a patent foramen ovale (PFO) from the left atrium into the right ventricle.

Case Report

A 63-year old female patient introduced herself to our emergency room (ER) due to sensory miss sensation in her left arm. The recent medical history included a medial femoral neck fracture (Pouwels II) 2 weeks ago after a bicycle accident, that successfully underwent surgery in our trauma surgery department. During the stay in our ER the patient was in a stable cardiac and respiratory state at all time.

Clinically the left arm imposed with reduced skin color with poorly palpable pulses in the lower extremity. Capillary refilling was greatly prolonged. The white blood cell count was 8.1/nl, CRP was 47.4 mg/l, LDH was 398 U/l and CK was 66 U/l. All other laboratory data, including serum amylase and lipase, urine analysis, hepatic and renal function tests, and coagulation profile were within reference values while D-dimer was 9.01 mg/l-FEU with a reference < 0.5.

A Doppler sonography showed a thromboembolic subtotal occlusion of the left arteria brachialis, so the patient was introduced to our vascular surgery department for thrombectomy immediately. The needed procedure was performed successfully on the same day. Postoperative the patient continuously showed to be tachycard and with oxygen dependable dyspnea (NYHA IV). For further diagnostics we performed a computed

tomography (CT) of the patients' thorax to eliminate PE as the dyspnea cause.

The radiological diagnostics revealed a bipulmonal fulminant PE in the central arteries with yet no CT-morphological evidence of right ventricular stress. In a following CT phlebography we found a deep vein thrombus in the *Vena femoralis profunda* as likely source of the PE.

After diagnosing the severe embolism, we performed a TTE for detailed evaluation of right ventricular function that showed right ventricular enlargement combined with a low degree of dysfunction as well as a dilatated *Vena cava inferior*. Within the right ventricle we could see a mobile echogenic structure that seemed to be coming from the right atrium. Seeking for the structures origin the transthoracic ultrasound showed the thrombotic structure to be passing the atrial septum through a PFO, reaching into the left atrium and being in touch with the mitral valve (Fig. 1). In the TEE we could confirm the initial prognosis of a thrombotic structure that reached from the right ventricle into the left atrium (Fig. 2, 3).

Since the patient was still in a circulation compromising condition and dependable to oxygen supply, we performed an intra-



Figure 1. Trans-thoracic echocardiography 4-chamber view: free floating structure mainly seen in the right atrium reaching past the tricuspid valve into the right ventricle. Also, the structure seems to be passing the atrial sept into the left atrium.



Figure 2. Transesophageal echocardiography LA+RA: echogenic structure starting in the left atrium and reaching throughout the whole right atrium.

thoracic thrombectomy recovering the intracardial thrombus as well as the bipulmonal central artery embolisms under extracorporeal circulation.

After complication-free surgery the patients' recovery was timely and intubation was removed shortly after having the patient transferred to our intensive care unit. After a short surveillance time a cardiopulmonary stable patient was transferred to the departments general ward and discharged a few days later with therapeutically anticoagulation.

Discussion

PFO is a rare cardiac situation but is related to increased rates of cryptogenic strokes and peripheral embolism. Homma et al. reported that it occurs in more than 25% of the adult population [7, 8].

We assess the initial cause of introduction in our ER as a paradox embolism due to the huge intracardial thrombotic structure probably initially a deep vein thrombus, whose remains we were able to detect in phlebographic tomography, due to reduced mobility after trauma surgery that embolized paradox and got stuck in the PFO. Unfortunately, searching for reasons for a spontaneous total arterial embolization was planned for post-surgery of brachial thrombectomy. We assume that under surgery a right ventricular part of the intracardial thrombus embolized into the pulmonary arteries.

PE is a high-risk disease, missing the diagnose or its severity still causes a high mortality rate. Marshall et al. demonstrated that PE is estimated to cause 200 000 to 300 000 deaths in the United States annually [9]. Clinical indicator to discriminate between light and severe embolisms is the right ventricular function. Massive right ventricular congestion can often already be seen in radiological diagnostic procedures such as CT methods and



Figure 3. Transesophageal echocardiography: the thrombotic structure reaches far into the right ventricle.

thoracic x-ray, but echocardiography proved to be more sensitive in detecting sub-massive right ventricular dysfunction and is useful for further treatment decisions. Echocardiographic signs of severe PE often include right ventricular dilatation, abnormal septal movement and dilated Vena cava with reduced breath dependent caliber variation [4, 5, 10–12].

In our case the patient showed severe bipulmonal embolism in the CT but radiological not a massive right ventricular congestion. With further echocardiographic diagnostic we could confirm a compromised circulation due to the fulminant increase in afterload. With diagnosing the thrombus *in situ*, we were not just able to clarify the further treatment path with choosing surgical thrombectomy to reduce the risk of another paradox embolism or even a stroke under medical thrombolysis [13], but also to find the source of the pulmonal artery occlusion as well as of the brachial arteries embolism.

Since patients with PFO exhibit a higher risk of paradoxical embolism, transesophageal diagnostics is an important tool in evaluating the reasons of arterial embolism of unknown source [14].

The report shows the importance of echocardiographic diagnostics after radiological confirmation of PE to measure the impact on right ventricular circulation, to choose necessary treatment options especially decide whether a thrombolytic therapy or a surgery option is the treatment of choice.

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