



Pulmonary Thromboembolism in a Patient With Patent Foramen Ovale



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Citation Pazooki B, Radkhah H, Sherafati A. Pulmonary Thromboembolism in a Patient With Patent Foramen Oval. Case Reports in Clinical Practice. 2018; 3(4):128-131.

Running Title Pulmonary Thromboembolism in Patent Foramen Ovale



Article info:

Received: 06 October 2018

Revised: 09 November 2018

Accepted: 18 December 2018

Keywords:

Patent foramen ovale (PFO);
Pulmonary thromboembolism (PTE); Stroke

ABSTRACT

During the recent years, paradoxical embolism and Patent Foramen Ovale (PFO) have drawn great attention because of their strong correlation with cryptogenic stroke in young patients. The coexistence of pulmonary and paradoxical embolism is even rarer. Awareness of this complication with prompt recognition and treatment could prevent significant disability and death. We presented a 51-year-old man with evidence of concomitant pulmonary embolism and overload due to Pulmonary Thromboembolism and stroke, which suggests the presence of Patent Foramen Ovale. He was successfully treated with anticoagulation and PFO closure was performed.

Introduction

Patent Foramen Ovale (PFO) is a congenital cardiac lesion that frequently persists until adulthood [1-3]. In fact, up to 25% of the general population have PFO. Although most patients with PFO are asymptomatic, a variety of clinical manifestations may be associated with PFO, most importantly cryptogenic stroke. Diag-

nostic evaluation for PFO is indicated in patients with a cerebral ischemic event of uncertain origin or other clinical manifestations of PFO [3]. Pulmonary Thromboembolism (PTE) causes a dramatic pressure overload to the right side of the heart, which theoretically may lead to a right to left shunt at PFO level, resulting in increased risk of paradoxical emboli.

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Case Presentation

A 51-year-old heavy smoker (50 pack/year) man was admitted in the emergency room with anasarca, dyspnea, and confusion. Initial assessment revealed hypoxia (O₂ saturation was equal to 82%), crackles in lung auscultation, and generalized edema. Physical examination was otherwise normal. After initiation of oxygen therapy, renal and liver function tests, TSH and albumin for investigating the cause of edema, VBG, abdominopelvic sonography, and brain CT scan were requested (Table 1). Respiratory acidosis, severe ascites (in sonography), and multiple ischemic lesions (in brain CT-scan) were detected.

In the next step, abdominal paracentesis and echocardiography were performed. Ascites fluid analysis

revealed a high SAAG (serum ascites albumin gradient) and high protein fluid (Table 2).

Echocardiography showed severe Right Ventricular (RV) enlargement accompanied by significant RV systolic dysfunction. An elevated Pulmonary Artery Pressure (PAP) was noted, too. Mild pericardial effusion was also detected. Neurologic consultation suggested brain MRI to confirm ischemic lesions, and cardiologic consultation suggested performing Transesophageal Echocardiography (TEE) due to high suspicion of Patent Foramen Ovale (PFO) in the patient.

Brain ischemic lesions were detected in MRI, and aspirin and atorvastatin were initiated for the patient. PFO was detected in TEE. Because of elevated Pulmonary Arterial Pressure (PAP) and patient's complaint of dyspnea, pulmonary CT angiography was performed which

Table 1. Laboratory data

Test	Result	Normal Range
pH	7.30	7.35-7.45
PCO ₂	54 mmHg	38-42 mmHg
HCO ₃	30 mEq/L	23-30 mEq/L
Albumin	3.2 g/dL	3.5-5.5 g/dL
Total Protein	5.6 g/dL	6-8 g/dL
Cr	1.1 mg/dL	0.6-1.2 mg/dL
NT-pro-BNP	3678 ng/L	>450 ng/L
AST	30 U/L	<37 U/L
ALT	38 U/L	<41 U/L
TSH	1.2 mIU/L	0.3-5 mIU/L



NT-pro-BNP: N-Terminal pro B-type Natriuretic Peptide, AST: Aspartate Transaminase; ALT: Alanine Transaminase; TSH: Thyroid Stimulating Hormone; Cr: Creatinine.

Table 2. Ascites fluid analysis

Test	Result
WBC	10
Albumin	1.7 g/dL
Total protein	3.5 g/dL
Serum albumin	3.2 g/dL



revealed acute Pulmonary Thromboembolism (PTE) in most segmental branches of the bilateral pulmonary artery. Anticoagulation was initiated, and the patient was referred for PFO closure due to paradoxical emboli (brain lesions).

Discussion

Patent Foramen Ovale (PFO) is a common echocardiographic finding which is detected in around 25% of the general population. PFO prevalence is reported higher in cryptogenic stroke, and it is more likely to play a role in a subgroup of these patients younger than 55 years old [4, 5]. PFO has no symptoms in most of the cases; however, it can be associated with clinical syndromes like air embolism, platypnea-orthodeoxia syndrome, and most importantly cryptogenic stroke due to its potential for causing paradoxical emboli.

PFO can be missed in transthoracic echocardiography. TEE with contrast at baseline, and following the Valsalva maneuver or coughing to increase right atrial pressure and intensifying right to left shunt is considered as a diagnostic test for detection of PFO [6-8]. Thus, TEE is necessary for definite evaluation in certain cases. Not all patients need evaluation for PFO. In fact, patients with cryptogenic stroke or other embolic events or patients presenting with other manifestations of PFO need diagnostic evaluation with echocardiography.

On the other hand, it must be mentioned that the identification of PFO in a patient with ischemic stroke does not definitely make PFO the cause of that event. PFO is common thus it may only be an accidental finding in some patients with ischemic stroke. In fact, before determining PFO as the cause of the ischemic event, careful assessment of other more common causes of brain ischemia, including embolic events from cardiac or vascular sources should be considered [9-11].

Initial evaluation of ischemic stroke includes vascular imaging via magnetic resonance angiography, CT angiography, Doppler ultrasonography of the carotid and intracranial arteries, and transthoracic echocardiography to rule out the cardioembolic source. If the initial assessments were unremarkable and there is no alternative explanation for ischemic stroke, in patients with age ≤ 60 years, TEE is indicated for evaluation of left atrial appendage clot and PFO [12].

There is controversy over the best treatment for patients with cryptogenic stroke and PFO. Strategies for lifestyle modification, including physical activity and

healthy diet, control of hypertension, and statin therapy are indicated for risk reduction and secondary prevention. There is an inconsistency about using antiplatelet therapy or anticoagulation for patients with PFO and cryptogenic stroke.

There is no strong recommendation based on the available data to support PFO closure for patients with a cryptogenic stroke without evidence for deep vein thrombosis [13-15]. However, challenges continue in this era and new trials are running which their results will give a better understanding of future decision makings.

Our patient had a recent stroke accompanied by a submissive pulmonary embolism resulting in right heart side pressure overload which could facilitate the right to left shunt and occurrence of paradoxical emboli. Because of his stable blood pressure and lack of organ hypoperfusion and regarding the patient's recent stroke, systemic thrombolytic therapy was not delivered for pulmonary embolism and just anticoagulant therapy was initiated. Furthermore, because of the presence of PFO and evidence of thrombosis in systemic venous circulation, suspicion for paradoxical emboli as the reason for ischemic stroke was high, it was decided to close PFO. It seems that the decision for PFO closure must be based on each patient's situation.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles were considered in this article.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of interest

The authors declared no conflict of interest.

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