Case Report





Pulmonary Thromboembolism and Thrombus in Patent Foramen Ovale

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Introduction

Patent Oval Foramen (FOP) is the persistence of the opening between the atrial *primum* and atrial *secundum* in the location of the oval fossa present in about 25% of the adult population¹. In generally, it is a casual finding and without hemodynamic repercussions. However, the paradoxical embolism as a cause of ischemic events due to the patency of this foramen is documented since the nineteenth century. Recent studies have shown the association between FOP and various medical conditions, some potentially serious, such as ischemic stroke (AVCi) and pulmonary embolism (TEP).

Despite well-documented consequences of paradoxical embolism through the FOP, the true passage of venous clot through a patent foramen was rarely described in the literature². In this report, the case of a patient is described with a complicated TEP picture after having been shown the presence of a thrombus lodged in FOP and the conduct adopted is discussed.

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This is a female patient, 89 years old, suffering from systemic hypertension, chronic coronary insufficiency, severe asthma, deep vein thrombosis, admitted to the emergency department with dyspnea picture in evolution for three days. On physical examination, tachydyspneic was presented without adventitious respiratory auscultation, with a heart rate of 112 beats per minute in an irregular rhythm and blood pressure of $160 \times 85 \text{ mm}$ Hg.

The resting electrocardiogram showed atrial fibrillation. A transthoracic echocardiogram performed with equipment Hewlett-Packard/Phillips SONOS 5500 with transducers of 2.5 and 5 to 7.5 mHz showed a thrombus in the left atrium. Then we performed a transesophageal echocardiogram, which permitted visualization of a threadlike thrombus in the right atrium, crossing the FOP and protruding into the left atrium (Figure 1).

As a conduct, the patient remained on conservative treatment with low weight heparin associated with oral anticoagulation to reach the therapeutic range. The transthoracic echocardiogram was performed two weeks after

Keywords

Embolia Pulmonar; Forame Oval Patente; Ecocardiografia/diagnóstico.

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Manuscript received on July, 30th, 2013; accepted October, 23th, 2013.

DOI: 10.5935/2318-8219.20140006

the initial observation and revealed the complete absence of thrombus (Figure 2). The patient was discharged on the 16th hospitalization day, under oral anticoagulation and with ambulatory follow-up.

Discussion

The presence of FOP is crucial during fetal life by allowing oxygenated blood to pass directly into the systemic circulation, avoiding collapsed lungs at this phase of life. With lung expansion at birth, the *septum primum* moves against the left atrial side of the *septum secundum*, causing the fusion of septa and closure of oval foramen by the first month of life. However, about a quarter of the general population presents no such merger and patent oval foramen may remain throughout life or to close initially to open again in situations of right pressure overload¹.

In most cases finding the FOP is a casual finding of the echocardiogram operator and that does not generate a significant impact on patient's life. However, studies have highlighted the association of FOP with different pathologies. Steiner et al³ and Webster et al 4 found FOP in 45% and 50% of patients with cryptogenic ischemic stroke, respectively, demonstrating the role of FOP as one of the main factors involved in the pathogenesis of this disease, especially in young adults without other risk factors. There have also been reported that patients with classic migraine have a higher incidence of FOP and right-left shunt could explain the pathophysiology of aura and increased risk of stroke, especially below 45 years.^{5, 6}.

Furthermore, the FOP may appear associated with the occurrence of TOP, a disease often with 5/10.000 estimated incidence in the general population, and high mortality rate (around 30-35% when treatment is not instituted properly) ⁷. It is known of the concomitant presence of FOP and TEP to be associated to increased risk of systemic paradoxal emboli and, therefore, a higher severity of the clinical picture. In our case, due to the condition of bed constraint in which the patient lived, the deep venous system was the likely source of thromboembolism, consequent to venous stasis. Pulmonary arterial hypertension caused by TEP by increasing right atrial pressure, stimulates the right-left interatrial *shunt* and thrombus in the FOP lodge, as visualized⁷⁻⁸.

As for the therapy to be adopted, there is the option of drug treatment with anticoagulant drugs, percutaneous FOP closure and surgical embolectomy⁹. In spite of the predominance of cryptogenic ischemia being higher in patient with FOP than in the general population, one does not know if percutaneous closure, although usually recommended, is capable of reducing the risk of recurring infarcts ⁹⁻¹². Meier et al.² and Furlan et al.⁹ have recently shown that the intervention to close

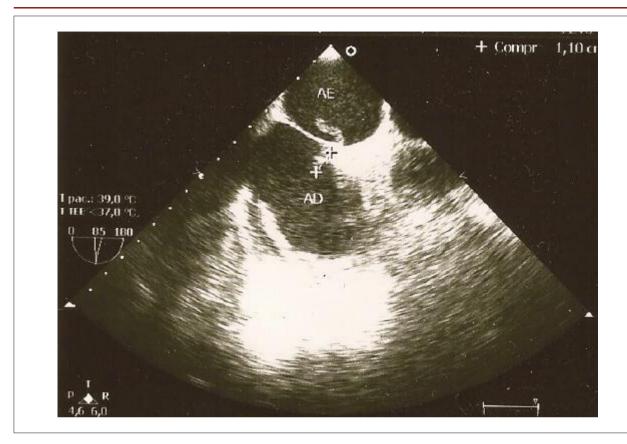


Figure 1 - Visualization, through transesophageal echocardiogram, of a threadlike thrombus in the right atrium, crossing the FOP and protruding into the left atrium.

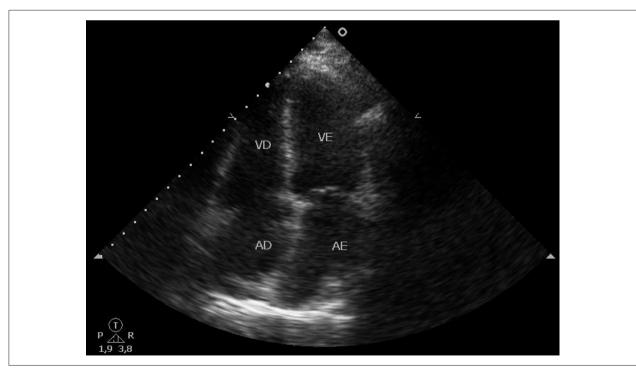


Figure 2 - Transthoracic control after two weeks on conservative treatment with low weight heparin associated with oral anticoagulation to reach the therapeutic range showing complete absence of thrombus.

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FOP did not offer higher benefits for the prevention of new ischemic events or death when compared to isolated drug therapy. Regarding surgical therapy, Erkut et al ¹¹ performed the removal of thrombus impacted in FOP with its subsequent closure by direct suture. Surgical therapy appears to have potential benefit to the complete removal of thrombi that may eventually move and cause paradoxical embolism. However, these authors argue that there is no consensus regarding the

treatment to be adopted and that the choice of procedure may be difficult in patients without evident contraindications^{11, 12}. In the case presented, the application of low weight heparin and anticoagulation therapy allowed complete resolution of intracardiac and pulmonary thrombi, corroborating other literature reports^{9,12}. It is therefore suggested that drug therapy can be an effective alternative in high risk patients with FOP and TOP.

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