Journal of Clinical & Experimental Cardiology

Facciorusso et al., J Clin Exp Cardiolog 2012, 3:12
DOI: 10.4172/2155-9880.1000226

Case Report Open Access

Pulmonary and Paradoxical Embolism after Fracture of the Big Toe in a Patient with Patent Foramen Ovale

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Abstract

The coexistence of pulmonary and paradoxical embolism is very rare. We report the case of a patient with evidence of both concomitant pulmonary and paradoxical embolism (cerebellar stroke), consequent to left foot immobilization after left big toe fracture. The patent foramen ovale and the increase in right atrium pressure due to pulmonary embolism accounted for the paradoxical embolism.

Keywords: Paradoxical embolism; Patent foramen ovale; Pulmonary embolism

Background

The association of pulmonary and paradoxical embolism is very rare [1,2]. The patent foramen ovale (PFO) is the heart defect most commonly responsible for paradoxical embolism [3]. Usually, patients with PFO are long-life asymptomatic. However, if right atrial pressure increases, a right to left shunt arises with risk for paradoxical embolism. This condition is caused by a venous embolus that crosses the PFO or that is trapped in PFO [4-6]. Brain arteries are the most frequent localization of paradoxical embolism [4].

We report a case of a patient with pulmonary embolism and cerebellar stroke due to paradoxical embolism.

Case Report

A 73 years old woman with history of hypertension, obesity and rheumatoid arthritis, was admitted to our department of cardiology with the diagnosis of pulmonary embolism associated with ischemic stroke.

In the patient's late medical history was unremarkable except for a left internal saphenectomy in youth, a right bundle branch block (RBBB) dating from several years and a urinary calculosis for which she had surgery some years before.

At home she was on methylprednisolone 4 mg/day and telmisartan/hydroclorithiazide 40/12.5 mg/day.

The patient reported a fracture of the left big toe that was treated with immobilization and limitation of walking. No antithrombotic prophylaxis was started. Two weeks after the fracture, the patient developed swelling, cyanosis and pain in her left foot and leg. Almost simultaneously she complained of dyspnea for mild efforts. Four further weeks later the patient started to complain dizziness and equilibrium disturbances. Due to the persistence of the symptomatology, the patient came to the Emergency department of our Hospital. At admission, at physical examination no other significant disorders were detected, particularly no pulmonary edema was found, in the Emergency department. Blood pressure resulted 120/70 mmHg. Lab tests documented an increase in D-dimers 17.24 mg/l (n.v<0.50). The electrocardiogram (ECG) showed sinus rhythm 70/min with RBBB. Contrast Computed Tomography (CT) scan of the head showed the presence of subacute infarction area of 4 cm × 3 cm in the right cerebellar hemisphere (Figure 1). Contrast CT scan of the chest documented the bilateral presence of multiple emboli in the branches of the pulmonary arteries (Figure 2). No Deep Vein Thrombosis (DVT) was found at lower limbs venous echography, but venous insufficiency was demonstrated at the left popliteal level. The carotid echography was normal. At transthoracic echocardiography mild concentric left ventricular hypertrophy and ejection fraction of 54% were found. Dilation of the right ventricle with dyskinesia of the free wall and pulmonary pressure of 40 mmHg were observed. These data were consistent with the hypothesis that the patient, after the fracture, had a DVT followed by pulmonary embolism and cerebellar infarction. Due to the suspicion of a paradoxical embolic origin of the cerebellar stroke, a heart transesophageal echocardiography was programmed.

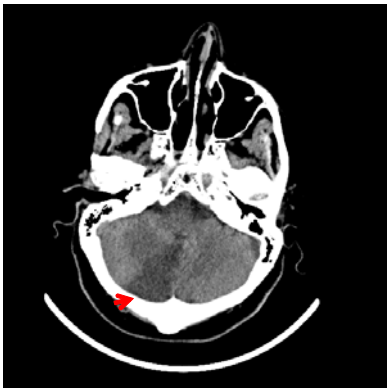

Figure 1: Contrast Computed Tomography scan of the head. Presence of subacute infarction area of 4 cm x 3 cm in the right cerebellar hemisphere (red arrow).

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Received October 21, 2012; **Accepted** November 27, 2012; **Published** November 30, 2012

Citation: Facciorusso A, Stanislao M, Pacilli MA, de Luca G, Stolfo GD, et al. (2012) Pulmonary and Paradoxical Embolism after Fracture of the Big Toe in a Patient with Patent Foramen Ovale. J Clin Exp Cardiolog 3:226. doi:10.4172/2155-9880.1000226

Intravenous heparin was started immediately after admission together with amoxicillin 1 g bid, methylprednisolone 4 mg/day and telmisartan/hydrochlorothiazide 40/12.5 mg/day. From the third day, intravenous heparin was replaced with subcutaneous enoxaparin 8000 IU bid. Baseline contrast transcranial color-coded duplex (TCCD) was normal, but at Valsalva maneuver a high grade right to left shunt was detected (Figure 3). At transesophageal echocardiography a PFO was observed (Figure 4). All these data, taken together, supported the hypothesis that the cerebellar infarction was consequent to a paradoxical embolism.

A brain magnetic resonance imaging (MRI), performed on 5th day from admission, confirmed the presence of a subacute ischemic stroke in the middle cortical-subcortical right cerebellar hemisphere together with other small ischemic stroke areas in the right optical radiation and right semioval centre (Figure 5). Serum markers (CEA, CA 15-3, CA 125, CA 19-9) resulted in the normal range. The blood coagulation screening showed presence of lupus anticoagulant, normal anticardiolipin IgG antibody 1.2 U/ml (n.v<15), slightly increased of anticardiolipin IgM antibody 20.1 U/ml (n.v<15), normal levels of the C protein, S protein, antithrombin and homocysteine, absence of mutation in the Leiden V factor and prothrombin.

In the following days the patient showed progressive improvement of the dyspnea and dizziness.

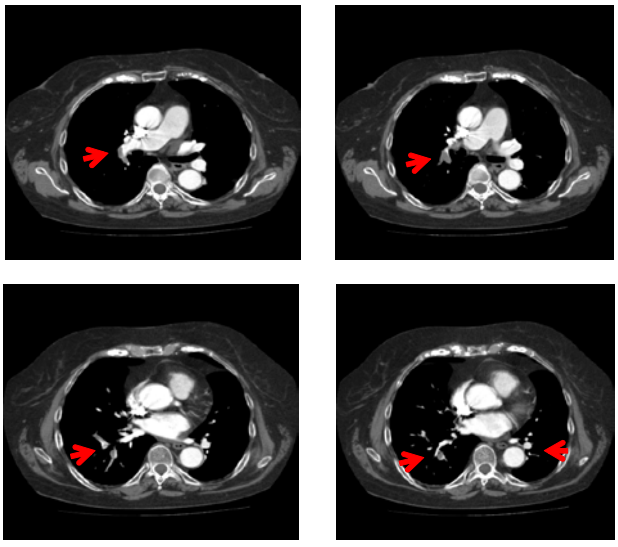

Figure 2: Contrast Computed Tomography scan of the chest. Bilateral presence of multiple emboli in the branches of the pulmonary arteries (red arrows).

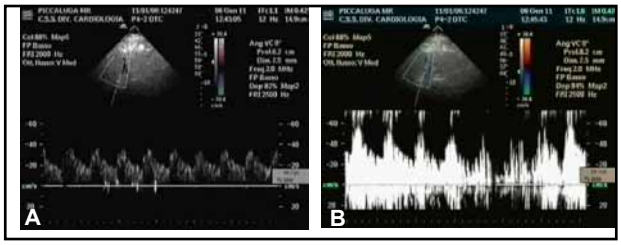

Figure 3: Transcranial Colour Coded Duplex (TCCD). (A) Baseline contrast TCCD was normal. (B) A high grade right to left shunt at contrast TCCD with Valsalva maneuver was found.

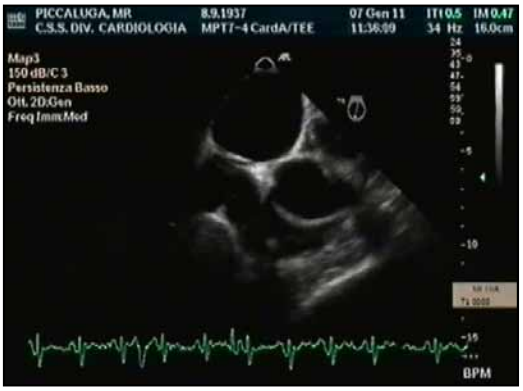

Figure 4: Transesophageal echocardiography. A patent foramen ovale (PFO) was observed.

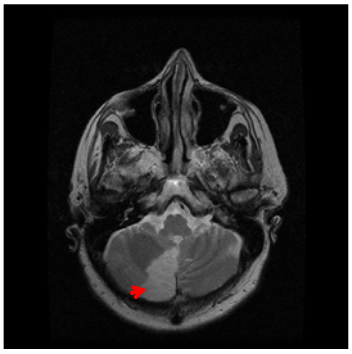

Figure 5: Brain Magnetic Resonance Imaging (MRI). Presence of a subacute ischemic stroke in the middle cortical-subcortical right cerebellar hemisphere (red arrow).

According to guidelines [7], oral anticoagulation therapy (OAT) with warfarin (5 mg) was started on 5th week, in order to keep International Normalized Ratio (INR) values between 2 and 3. The patient refused the percutaneous PFO closure.

Fortunately, an 8 months clinical follow-up, the patient's clinical conditions were good without recurrence of thrombotic/embolic events.

Discussion

The association of pulmonary and paradoxical embolism is very rare [1,2]. The PFO is a frequent congenital anomaly, with a prevalence ranging between 25-35%, and is the heart defect most commonly responsible for paradoxical embolism [3].

Usually, patients with PFO are long-life asymptomatic. However, if right atrial pressure increases, as in the case of pulmonary embolism or chronic obstructive pulmonary disease, a right to left shunt arises with risk for paradoxical embolism [1]. This condition is caused by a venous embolus that crosses the PFO or that is trapped in PFO [4-6]. Brain arteries are the most frequent localization of paradoxical embolism [4]; less commonly paradoxical embolism has been described in the upper limb [1], coronary [8], renal or retinal arteries [9].

Patients with pulmonary embolism and PFO diameter >4 mm have 10 fold higher risk of death and 5 fold higher risk of systemic embolism than patients without PFO [10].

The diagnosis of paradoxical embolism requires the simultaneous presence of venous thrombosis or pulmonary embolism without thrombi in the left section of the heart and/or in aorta, PFO and systemic arterial embolism [11]. In the reported case all elements were present.

In our patient, the fracture of the left big toe, treated with immobilization and limited walking without heparin prophylaxis, had caused a DVT as shown by swelling, pain and cyanosis of the left foot and leg. Left leg thrombosis was not detected at venous ultrasound study probably due to the complete embolization of thrombus, but venous insufficiency of the left popliteal level was suggestive for recent DVT. Probably, the patient's thrombophilic state, due to the positivity of lupus anticoagulant, had also contributed to the DVT.

The detachment of the thrombus from left leg vein caused pulmonary embolism as demonstrated by dyspnea. At its time, the pulmonary embolism caused elevation in right atrial pressure with consequently increase in the right to left shunt through the PFO as documented at the TCCD and transesophageal echocardiography. The venous embolism had migrated into the arterial circulation where it had caused the right cerebellar infarction as demonstrated by sudden onset of dizziness and by brain CT and MRI imaging.

In our case, a starting heparin and following warfarin is treatment of choice. In fact, as reported, this therapy is effective for the pulmonary and paradoxical embolism and prevents recurrent DVT [7].

The PFO closure after the first cerebral embolic event is a still debated topic. The guidelines on the treatment of stroke recommended the interventional procedure only in cases of recurrent embolic during optimal OAT [12]. In our case, we recommend PFO closure for several reasons: the high relevance of right to left shunt, the patient's thrombophilic status and the possible further neurological injuries. In fact, the brain MRI was consistent with multiple minor cerebral embolic episodes together with the symptomatic cerebellar one.

The patient refused the closure of PFO. Notwithstanding 8 months eventless follow-up confirmed the effectiveness of OAT. Further and larger experience is however needed, in our opinion, to understand the most valid therapeutic approach in this uncommon condition.

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