Case Report

Limb Ischemia and Dyspnea; A Plausible Indication of Paradoxical Embolism

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Coexistence of pulmonary embolism and systemic arterial embolism can be one of the most life threatening conditions, diagnosed as paradoxical embolism which suggests the presence of intra-cardiac defects. The case presented herein, is a 46-year old male with paradoxical emboli. The patient was diagnosed as having pulmonary embolism and unilateral lower extremity arterial emboli through a previously undetected Patent oval foramen oval (PFO). This case shows the importance of evaluating paradoxical embolism in unexplained cases of acute limb ischemia.

Keywords: Paradoxical Embolism, Limb Ischemia, Patent Foramen Oval, Pulmonary Embolism

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Introduction

Paradoxical Emboli (PDE) was described by Cohnheim in 1877, as passage of venous thrombus through a right to left shunt in cardiac defects into systemic circulation.[1] The intra cardiac shunts can be atrial septal defect (ASD), patent foramen ovale (PFO), patent dactus arteriosus (PDA), ventricular septal defect (VSD), tetralogy of fallot (TOF), aortopulmonary septal defect, coronary arterio-pulmonary artery fistula, and pulmonary arteriovenous malformation. PDE is apparently found in only 2% of general population.[2,3] However, on the one hand, PFO seems to be one of the most important risk factors of PDE and on the other hand, the prevalence of PFO is estimated about 25-30% in normal population.[3]

Case Presentation

The present case is a 46-year-old salesman admitted to the Emergency Department (ED), with an acute onset and severe pain in left lower limb distal to his knee. He described his pain as excruciating and accompanied by parasthesia and chilling sensation. He first felt the pain around 12:00 PM and tried to combat it by taking Ibuprophen and other over the counter analgesics. However, when his pain deteriorated with time, his family had to take him to our hospital. In the ED, the medical crew detected blood pressure 120/60, pulse rate 86 beat/min, respiratory rate 16/min and oral temperature 36 degrees Celsius. The left lower limb was pale, cold and numb and no pulse was detected in the left popliteal and dorsalis pedis arteries. The force was reduced to 2/5 in the affected leg. Cardiac examination demonstrated normal S1 and S2 without any murmur and additional sound but with mild tachycardia. The lungs were clear with no wheeze or crackle. The abdomen examination was unremarkable, active bowel sounds were heard; without any distention or gastrointestinal symptoms like constipation, diarrhea, or bleeding.

The patient did not have any history of medical situations, recent acute diseases, chronic illness, or any episode of immobilization, and hyper coagulability state. He did not smoke and was not alcohol or opioid consumer, as he stated.

A complete laboratory evaluation including ar-

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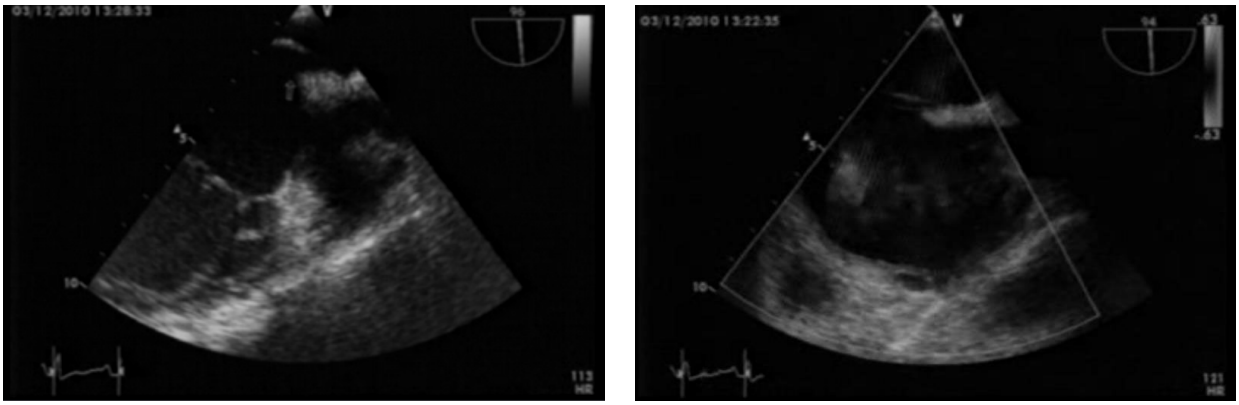

Figure 1: Transesophageal Echocardiography in longitudinal axis showing large size PFO with right to left shunt

terial blood gas, urinalysis, coagulability tests and serum electrolytes and glucose determinations was performed prior to any kind of intervention (table 1). All of the results were normal and unremarkable except a random sugar 192 mg/dl. The electrocardiogram revealed sinus tachycardia with right axis deviation and S1Q3T3 pattern with RV strain in V1-V4. A left leg Doppler sonography was performed before the surgery for confirming the diagnosis. The test revealed no significant arterial flow in femoral, popliteal, posterior tibialis and proneal arteries suggesting arterial emboli.

The first diagnosis was Ileo-femoral Artery emboli with unknown cause and our first act was an emergent vascular surgery to remove the clot and reopen the path of the arteries.

The operation was done successfully and an emergency fugarty catheter Thrombectomy was done. All of the symptoms were diminished. After the surgery, due to suspicious source of cardiac embolism, an echocardiography and Color Doppler test was performed and the underlying results were obtained.

Echocardiography

2D study showed normal LV size and function with dilated Right Ventricle and atrium (RV and RA) with moderate RV systolic dysfunction. There was mild to moderate Tricuspid Regurgitation (TR) with moderate to severe Pulmonary artery hypertension (SPAP: 65mmHg). Contrast study showed passage of many bubbles suggestive of right to left shunt. Trans-esophageal echocardiography (TEE) showed large size (5mm) PFO with significant right to left shunt. Due to RV enlargement and significant pulmonary hypertension, pulmonary thrombo em-

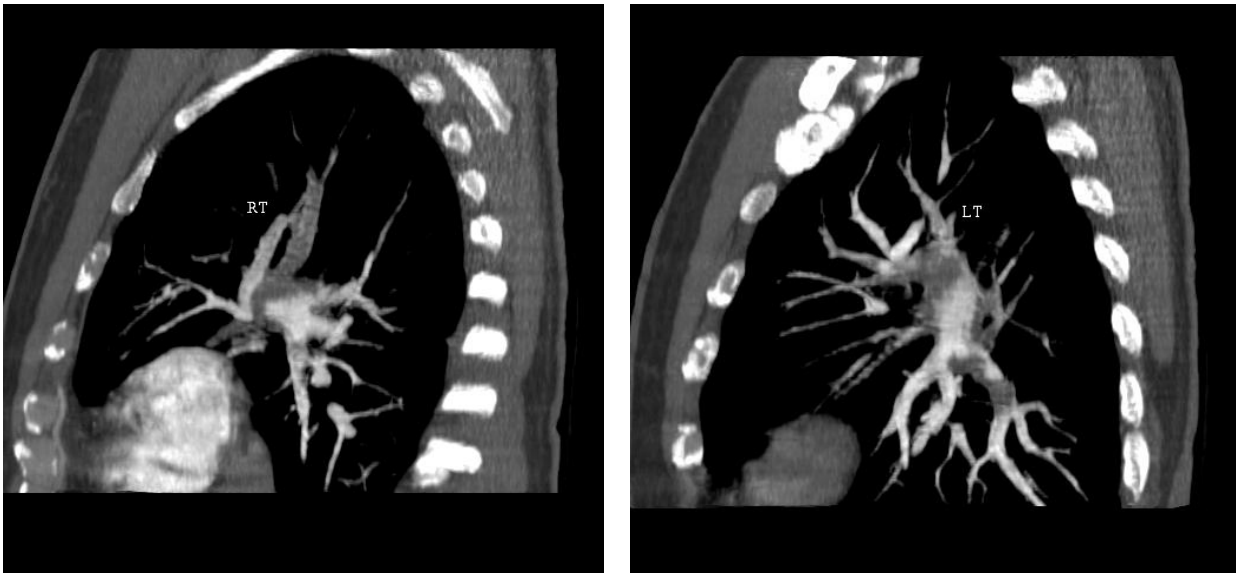

Figure 2: Pulmonary CT Angiography demonstrates shower emboli as a massive tubular defect in lobar and segmental branches of upper, middle, and lower segments.

bolism (PTE) was suspected and pulmonary angiography was recommended. (Fig. 1)

Doppler of both lower limb venous systems

External iliac, femoral, and popliteal veins showed pulsatile venous flow with no evidence of thrombotic obstruction and absence of deep vein thrombosis and popliteal cyst. Slight spheno-femoral reflux was detected under valsalva maneuver.

The patient was admitted to the coronary care unit of the hospital under heparin medication. During the night, the patient complained of short breath which was soon followed by a productive cough. An emergency team presently evaluated him. The results of arterial blood gas examinations were as follows: pH=7.47, PaCO=2 35 mmHg; PaO2= 42 mmHg on room air.

In the meantime, the patient underwent pulmonary CT-angiography and the result demonstrated "A shower emboli" as a massive tubular defect in lobar and segmental branches of upper, middle, and lower segments of both lungs. (Fig. 2)

His anticoagulation work up showed no evidence of factor V Leiden deficiency; furthermore, antiphospholipid antibody screening was negative and levels of factor VIII ,protein C and S and antithrombin III were all normal. In further evaluation, homocysteine level was mildly elevated (18umol/lit).

In conclusion, the patient was discharged after 10 days having normal general condition with warfarin medication and follow up appointment in a month. The PFO closure was planned in the near future.

Discussion

Typical clinical presentation of PDE includes peripheral embolism, brain abscess, cryptogenic stroke, renal and myocardial infarction, retina artery occlusion and decompression sickness in under water divers.[4,5] The diagnosis of PDE based on Johnson criteria is as follows:

1-Evidence of venous embolism like deep vein thrombosis

2-Right to left intracardiac shunting

3-Systemic arterial thrombosis while other sources such as left cardiac and proximal arterial Thrombosis have been ruled out.[6]

Furthermore right-to-left shunt during valsalva maneuver with "contrast transesophageal echocardiography" can confirm the definite diagnosis of PDE.[7] An increase in pulmonary artery pressure and also pulmonary resistance caused by Pulmonary thromboembolism will result in passage of the clot from right to left through PFO as a consequence of inversion shunting which can justify the simultaneous incidence of arterial and pulmonary embolism. PDE may complicate Pulmonary thromboembolism which is accompanied by PFO, especially if the patient is hemodynamically unstable.[8,9] Pulmonary Thrombo Emboli (PTE) has been established in 60% of patients with definite diagnosis of PDE.[4,10] Similar to our case, Inoue et al described a 67-year-old woman with paradoxical peripheral embolism in the bilateral external iliac arteries associated with PFO and complicated by acute pul-

Table 1. complete blood count (CBC) and routine biochemical assays of the patient.

Complete blood count		**Biochemistry evaluations**	
WBC (/mm³)	9700	Urea (mg/dl)	42
RBC(10^6/mm³)	5.02	Cratinine (mg/dl)	1
Hb (mg/dl)	14.1	sodium (meq/l)	141
MCH	82.3	potassium (meq/l)	4.2
MCV (fl)	28.3	Glucose (mg/dl)	191
MCHC	34.1	AST	19
Platletes (10^3/mm³)	148	LDH	492
Neurophills	72%	CK-MB	16
Lymphocytes	24%		

Hb:hemoglobin; MCH: mean corpuscular hemoglobin; MCV: Mean corpuscular volume; MCHC: mean corpuscular hemoglobin concentration; AST: Aspartate transaminase; LDH: lactate dehyroginase; CK-MB: Cratine kinase cardiac subtype; Fl: femto-litre

monary thromboembolism. The patient had severe peripheral ischemia due to a massive thrombus.[5] Caretta et al have also introduced a 79-years-old woman presented with dyspnea and lower left limb pain admitted to hospital in Italy, in 2009. Diagnostic workups revealed multiple thrombi in kidney, lower limb, and superior mesenteric artery during acute pulmonary embolism. Furthermore, Echocardiogram documented a PFO with a right-to-left shunt.[11] Similarly, Guo et al introduced a case of paradoxical emboli in 42 years-old man brought to hospital, who complained of loss of sensation in his legs, inability to stand, and 1-week history of short breath.[12]

Since the diagnosis of PDE is sometimes easily missed, it is vital to have a precise and focused examination on lower extremities such as evaluation of pulses, color, and performing doppler sonography in patients represented with pulmonary thromboembolism. In addition, if a patient presented conversely with acute onset of severe pain in lower limbs (likewise our case), coexistence of pulmonary embolism and systemic arterial embolism should be ruled out through As our patient seemed to have no specific Embolus origin, it is important to find the origin and cause of embolism.

Intracardiac shunt and PDE, although it is an uncommon condition, deserves immediate attention in a patient with PTE and systemic arterial embolism. Despite the fact that the cost-effective treatment and diagnostic method is still controversial, Doppler ultrasound, CT angiography, aortography and echocardiography appear to be the viable options. The treatment in such cases includes thrombolysis, catheter thrombectomy, an inferior vena cava filter if indicated, and instituting anticoagulation therapy in due course.

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