

When a Hole in the Heart Sends Clots the Wrong Way

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Abstract

Simultaneous presentation of ST-elevation myocardial infarction (STEMI) and pulmonary embolism (PE) is rare and should prompt consideration of paradoxical embolism (PDE) through a patent foramen ovale (PFO). We report the case of a 48-year-old lifelong athlete presenting with sudden cardiac arrest due to acute inferior STEMI, without angiographic evidence of atherosclerotic disease. Initial computed tomography angiography revealed bilateral pulmonary emboli. Coronary angiography demonstrated thrombotic occlusion of the mid-right coronary artery, successfully treated with mechanical thrombectomy, revealing normal coronaries. Subsequent echocardiography confirmed a PFO with right-to-left shunting. The patient was stabilized on anticoagulation and later underwent successful percutaneous PFO closure. Three-year follow-up showed no recurrence. This case underscores the importance of considering PDE in patients with acute coronary occlusion and concurrent PE or systemic emboli, particularly in the absence of atherosclerosis. Early recognition and targeted intervention, including PFO closure, can prevent recurrence and improve outcomes.

Keywords

ST-Elevation Myocardial Infarction (STEMI), Pulmonary Embolism (PE), Paradoxical Embolism (PDE), Patent Foramen Ovale (PFO)

1. Introduction

If a patient presents with STEMI without any evidence of atherosclerotic plaque in the coronaries, it raises the suspicion of an embolic coronary occlusion. Alt-

though the simultaneous occurrence of STEMI and Pulmonary Embolism (PE) is infrequent, when present, it suggests Paradoxical Embolism (PDE) through a PFO. Our case highlights this unique scenario.

2. Case Presentation

Our patient is a 48-year-old female, lifelong athlete, who was not expected to have atherosclerotic heart disease; she presents with cardiac arrest secondary to an acute inferior ST elevation myocardial infarction. The patient was in her usual state of health, physically very active, performing daily exercise; on the eventful day, she complained to a family member of the sudden onset of severe chest and back pain, followed by a witnessed cardiac arrest at home. When EMS arrived, no shockable rhythm was noted, CPR was initiated, and eventually she was successfully resuscitated. A 12-lead electrocardiogram (ECG) in the field showed inferior ST-segment elevation (**Figure 1**), consistent with myocardial infarction. After being transported to the emergency room, she underwent urgent computer tomography angiography (CTA) to rule out aortic dissection, which revealed bilateral pulmonary emboli (**Figure 2**) without right ventricular strain.

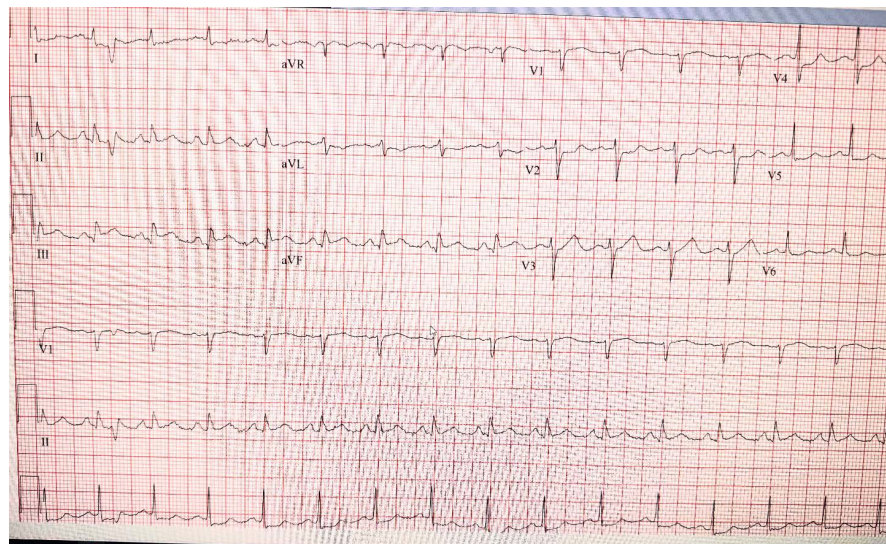


Figure 1. 12-lead electrocardiogram (ECG) in the field showed inferior ST-segment elevation.

As pulmonary embolism was not a saddle and no evidence of RV strain, likely this cardiac arrest related to acute myocardial injury. She was emergently taken to the cardiac catheterization lab to undergo coronary angiography, which revealed thrombotic occlusion of the mid-right coronary artery (RCA) (**Figure 3**). After successful mechanical aspiration of the thrombus, the angiography exhibited no coronary plaque (**Figure 4**). This raised the question of an embolic phenomenon in the coronary artery, prompting a transthoracic echocardiogram that demonstrated right-to-left shunting of saline bubbles, suggesting a patent foramen ovale (PFO). Our patient had right to left shunt without any evidence of right ventricular RV strain but probably patient had intermittent high right atrial pressure es-

pecially during Valsalva maneuver. Lower extremities doppler study was also performed which was negative for DVT. A transesophageal echocardiogram confirmed this (**Figure 5**). The patient was subsequently discharged on anticoagulation therapy Apixaban along with aspirin and Prasugrel in a stable condition. Apixaban was discontinued after 6 months. After a few months, the patient underwent a successful percutaneous closure of the PFO (**Figure 6**). Patient remains on DAPT (Dual antiplatelet therapy) for total 12 months from initial event. Three years after her initial event, she remains free from any recurrent episodes.

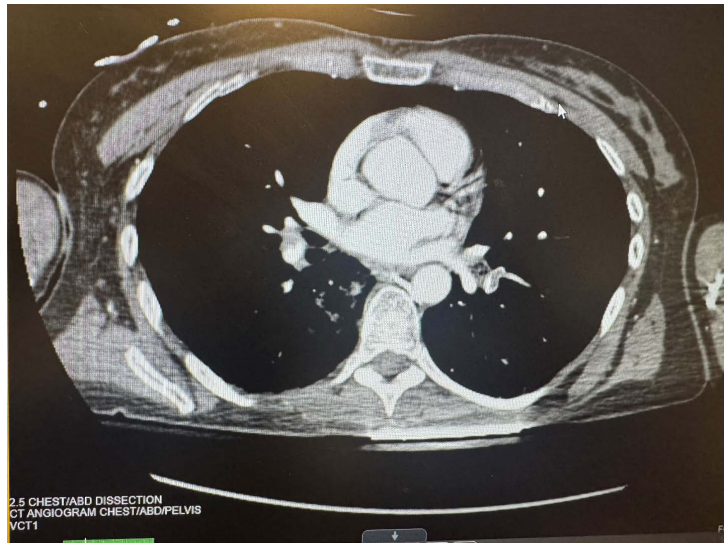


Figure 2. Computer tomography angiography showing bilateral pulmonary emboli.



Figure 3. Thrombotic occlusion of the mid-right coronary artery (RCA).

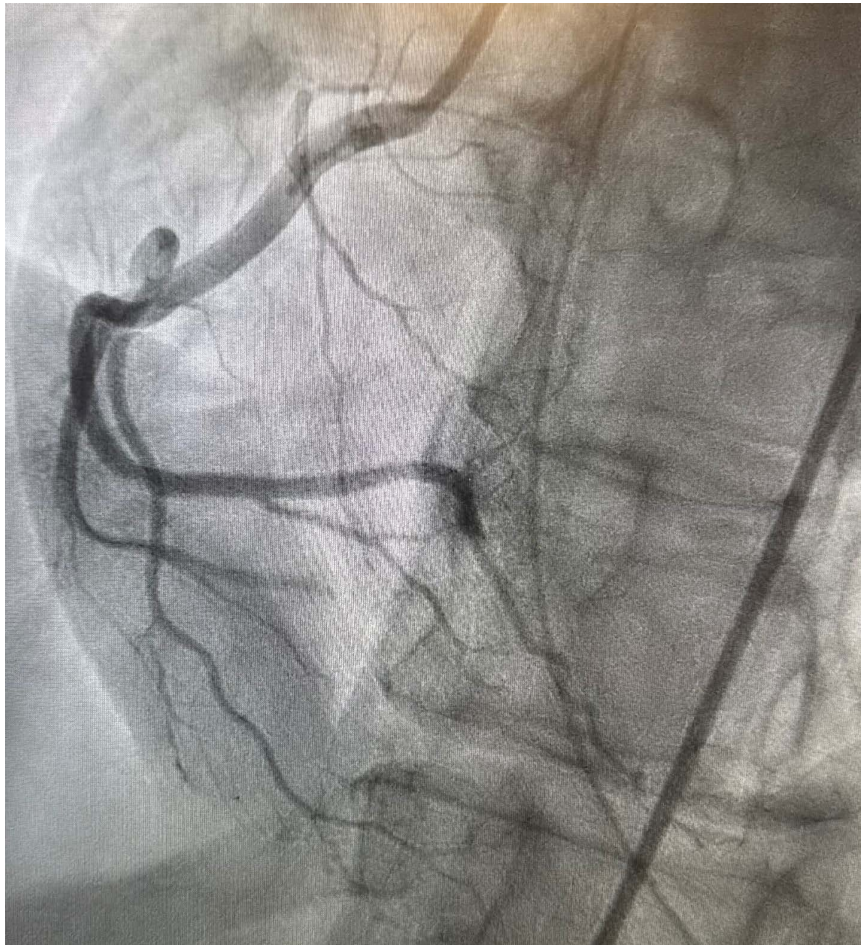


Figure 4. Normal coronaries after mechanical aspiration of the right coronary artery.

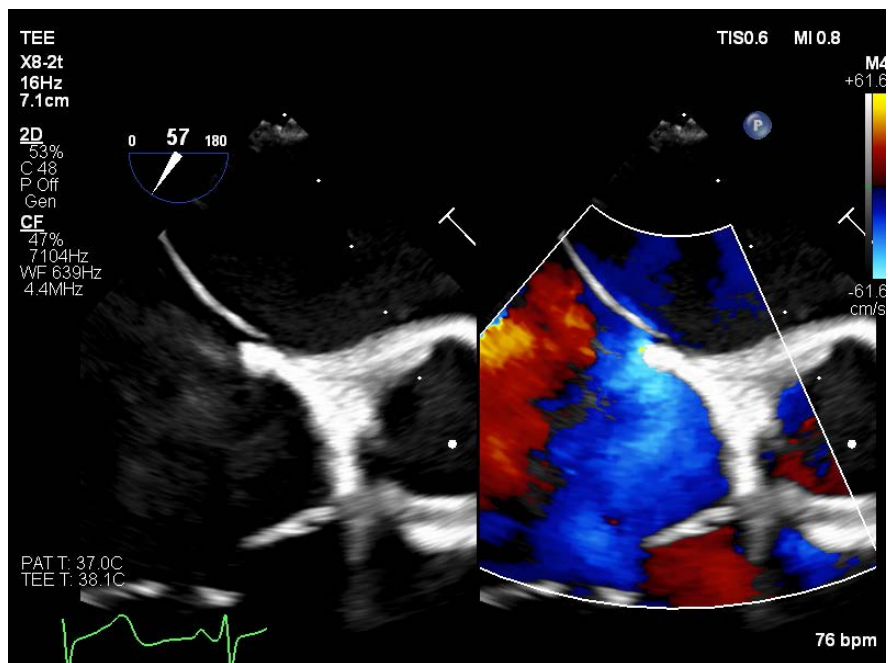


Figure 5. Transesophageal echocardiogram confirming patent foramen ovale.

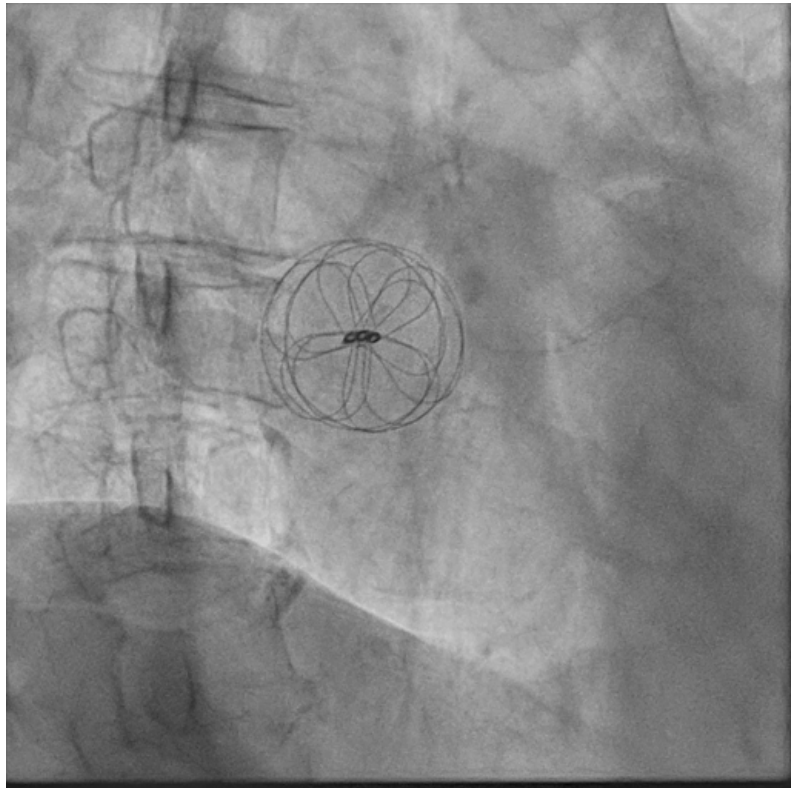


Figure 6. Percutaneous closure of the PFO angiography.

3. Discussion

3.1. Patent Foramen Ovale: Anatomical Origin

The foramen ovale is a fetal cardiac structure that permits oxygenated blood from the placenta to bypass the non-functioning fetal lungs by shunting from the right atrium to the left atrium. After birth, increased left atrial pressure with the onset of respiration typically results in functional closure, followed by fusion of the septum primum and septum secundum during infancy [1]. In approximately 25% of adults, complete anatomical closure does not occur, resulting in a patent foramen ovale (PFO) [2].

3.2. Types of PFO

PFOs come in varying sizes and morphology. A simple PFO is a small, slit-like opening that remains closed at rest and open transiently when the right atrial pressure rises, such as during coughing or the Valsalva maneuver [3]. High risk PFOs are those that are greater than 2 mm, have long tunnel (>10 mm), associated with low-angle PFO, atrial septal aneurysm, prominent Eustachian valve or Chiari's Network and larger number of bubbles crossing during bubble study [3]. Our patient has a high risk PFO with more than 2 mm

3.3. PFO Associated with an Atrial Septal Aneurysm

An atrial septal aneurysm (ASA), defined as a redundant and mobile interatrial

septum with excursion greater than 10 - 15 mm, is frequently associated with PFO and is considered a very high-risk substrate for paradoxical embolism (PDE) due to its increased mobility directing flow through the PFO [3] [4].

3.4. Diagnosing a PFO

Diagnosis of PFO is most reliably established with transesophageal echocardiography (TEE) and agitated saline contrast, which allows direct visualization of right-to-left shunting. Transthoracic echocardiography (TTE) with contrast serves as a noninvasive screening method, while Color-Doppler can also detect shunting [5].

3.5. Pathophysiology of Paradoxical Embolism

Pulmonary embolism (PE) most often arises as larger thrombi in the deep veins of the lower extremities or pelvis. Once dislodged, emboli travel through the venous system to the right atrium, right ventricle, and into the pulmonary arteries, causing partial or complete obstruction. In patients with a PFO and elevated right atrial pressure, as can occur in acute PE, the pressure gradient further permits right-to-left shunting, enabling the following emboli to pass directly into the systemic arterial circulation through the PFO [6]. This process, known as paradoxical embolism, can result in embolic events in the cerebral arteries leading to stroke, the coronary arteries causing a heart attack, or systemic arteries with regional consequences. Clinical presentation in case of heart attack and acute PE may have similar presentations, including chest pain, acute dyspnea, low blood pressure sometimes leading to syncope and ECG changes which may cause a conundrum in early diagnosis [7].

3.6. Closing a PFO

With the advent of percutaneous technologies for closing a PFO, surgical intervention has become largely obsolete [8]. These PFOs can be successfully closed using patch-like devices [Amplatzer™ PFO Occluder (Abbott Cardiovascular) and the Gore® Cardioform Septal Occluder (WL Gore & Associates)], which are delivered through the femoral vein via a catheter and deployed with patches on either side of the PFO, connected in between to create a secure closure. The device eventually undergoes endothelialization, effectively closing the PFO. This is an outpatient procedure, with same-day discharge in most hospitals.

4. Conclusion

Acute coronary occlusion due to paradoxical embolism is rare, but an astute physician can arrive at the correct diagnosis or have a high suspicion if the following scenarios are present. If no atherosclerotic disease is noted after clearing the thrombus in the coronaries, and 1) there is a concomitant diagnosis of pulmonary embolus, or 2) if the patient presents with concomitant stroke, or systemic embolus. Our patient presented with PE and heart attack, leading to suspicion for paradoxical embolism (PDE) through a PFO, which was promptly diagnosed and

closed successfully.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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