

# **A large snake-like thrombus in Transit across a Patent Foramen Ovale in a rare case with massive pulmonary embolism**

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## **ABSTRACT**

Reports of thrombus straddling the patent foramen ovale are extremely rare. Thrombus development in cardiac chambers increases the risk of mortality, compared to pulmonary embolism alone, and can require change in therapy.<sup>(1)</sup> We here describe a rare case of a 53-year-old patient with massive pulmonary embolism in which a large thrombus entrapped in patent foramen ovale and prolapsed into the left heart chambers with increased pulmonary artery pressure was found by echocardiography, which is a very rare and critical condition. Echocardiography is a readily available and safe tool for demonstrating the size, location, and extent of a thrombus, and it plays an important role in the early diagnosis and treatment evaluation for patients with a thrombus trapped in a PFO with concurrent pulmonary embolism. Our patient was treated with thrombolysis and anticoagulation and discharged from the hospital with an uneventful recovery. Our treatment of the present patient achieved a satisfactory result, but it may not be applicable to every patient. Because this is a rare diagnosis, with insufficient data, there is no formally established treatment guideline. However, in patients who are good surgical candidates, studies have shown better outcomes with surgical embolectomy as compared to anticoagulation alone or thrombolysis. However, anticoagulant treatment appears to be an acceptable therapeutic alternative to surgery, particularly in patients with comorbidities who are at high surgical risk and for patients with small PFO. Thrombolysis is linked to the highest mortality, which could be explained by the severity of the patient's initial presentation.

**Keywords:** Patent foramen ovale; massive pulmonary embolism; thrombus.

## **Introduction**

Thrombus straddling the patent foramen ovale is a rare and critical condition.<sup>(2)</sup> Thrombi in cardiac chambers appear to increase the risk of mortality, compared to PE alone, and can require a change in therapy.<sup>(3)</sup> In the presence of patent foramen ovale (PFO), paradoxical systemic embolization can occur, particularly when the pulmonary arterial and right atrial pressures are elevated which facilitate passage of the transitional thrombus through a patent foramen ovale (PFO), thereby increasing the incidence of embolic complications such as ischemic stroke and even sudden death.<sup>(4,5)</sup> As this finding remains extremely rare, the clinical features and therapeutic management of the condition remains unclear. The treatment of a transitional thrombus in patients with PE is controversial. Therapeutic options include surgical treatment with thrombectomy or medical treatment with heparin or thrombolysis. Surgical embolectomy has shown a trend toward improved survival, but the postoperative mortality rate is high.<sup>(6)</sup> Herein, we present a rare case involving a 53-year-old patient with right atrial huge thrombus entrapped in PFO and prolapsed into the left heart chambers and extensive bilateral PE. The patient underwent thrombolytic and anticoagulation therapy. After 30 days, she was discharged from the hospital with an

uneventful recovery. This case is being reported to illustrate that thrombolytic and anticoagulant therapy can be used as an alternative treatment in this condition.

## Case presentation

A 53-year-old woman was brought by non-medical paramedics to the emergency department with a 1-week history of chest tightness, unspecified chest pain, and shortness of breath. Of note, she lives a sedentary lifestyle since 26 days following to a second degree burn of the 2 legs (Figure 1). She has no notable family history, takes no treatment and is not followed for neoplasia. She reported the spontaneous onset of recurrent unspecified chest pain associated with stage 3 NYHA dyspnea. The patient had no palpitations or neurological symptoms prior to the discomfort. Upon arrival, the patient was conscious and oriented with a Glasgow score of 15/15. She had a body temperature of 38°C, the systolic blood pressure was measured at 120 mmHg, and the diastolic blood pressure at 60 mmHg, the heart rate at 100 beats per minute. She was breathless, with a respiratory rate of 31 cycles per minute with a capillary oxygen saturation in ambient air of 85%. On cardiac examination, she had normal heart sounds with regular rhythm and no murmurs, rubs, or gallops. Breath sounds were vesicular on respiratory exam and no wheezes or crackles were appreciated. Extremities were without edema and the left foot wound was well healed. The peripheral pulses are all perceived and symmetrical. The abdomen was soft, painless, without palpable hepatosplenomegaly, and the superficial lymph nodes were free. The electrocardiogram showed a sinus tachycardia at 100 beats per minute, with no anomaly in the PR space, negative T waves in the antero-septal leads (Figure 2). The initial biological assessment finds a hemoglobin at 12,1 g/dl, platelets at 267000/ $\mu$ l, serum creatinine at 7.5 mg/l, D-dimers at 41560 ng/ml, highly sensitive cardiac troponin marker at 4510 ng/l and C-reactive protein 35 mg/l. The hemostasis assessment was without abnormality. The performed transthoracic echocardiography (TTE) showed a large serpentine, free-floating echogenic mass in the right atrium (Figure 3) and extending to the left atrium (Figure 4) through a PFO (Figure 5) part of the mass moved to the left ventricle during diastole. The right heart cavities were dilated, and moderate right ventricular dysfunction was present. No emboli were visible in the central pulmonary artery. The systolic pulmonary artery pressure was 57 mmHg. Left ventricular function was normal. Based on the TTE findings, we decided to perform transesophageal echocardiography (TEE). However, the patient had an esophageal diverticulum; therefore, TEE was not carried out.

The angio-computed tomography scan confirmed a massive bilateral proximal pulmonary embolism with acute severe pulmonary hypertension without parenchymal lesions of the lungs (Figure 6). Lower limb venous echo-Doppler showed a right femoral thrombosis.

The patient was hemodynamically stable. However, she reported that her chest tightness and shortness of breath had worsened. After a discussion with the patient and her family and surgeons, the patient underwent thrombolytic and anticoagulant therapy. Recombinant tissue plasminogen activator was intravenously, and heparin was administered by a continuous pump from 24 to 48 hours. This was followed by subcutaneous injection of low-molecular-weight heparin and oral warfarin for 3 days, and the oral warfarin was sustained thereafter. After 30 days of treatment, the serum D-dimer

level had decreased to normal. Pulmonary computed tomography angiography showed that the PE had disappeared (Figure 4(a)). Follow-up TTE revealed no thrombus in the heart cavities (Figure 4(b)). The right heart cavities had recovered to normal size. The pulmonary artery systolic pressure decreased to 25 mmHg. No left-to-right shunt was present through the foramen ovale. The patient was discharged from the hospital with an uneventful recovery.

## Discussion

The visualization of a right atrial thrombus is referred to as a thrombus in transit and is a very rare echocardiographic finding, carrying a high mortality rate of up to 29% (7,8). It is even more rare to visualize a thrombus across PFO (9,10). Death due to cardiogenic shock and/or right heart failure has been noted in 44% of patients who presented with an embolus straddling the PFO and death due to stroke in nearly 16% (11).

In the present case, the foramen ovale is a remnant from the embryonic period and acts as a potential door capable of opening from the right to left atrium. The foramen ovale closes at 5 to 7 months after birth because of fusion of the first and second atrial septa. However, in up to 25% to 34% of normal adults, the foramen ovale is not completely fused and forms a potential space or separation between the septum primum and secundum; this is termed a PFO (12). Under normal conditions, because the pressure of the left atrium is higher than the right, there may be no left-to-right shunt through the atrial septum. However, the interatrial pressure gradient can be reversed, which can occur in the setting of pulmonary hypertension induced by coughing, laughing, and the Valsalva maneuver. It can also occur in patients with pulmonary arterial hypertension secondary to PE and congenital heart disease. Once the pressure of the right exceeds that of the left, the foramen ovale may open from the right to left atrium. A thrombus from the venous system into the right atrium may easily cross into the left through a PFO.

The first reported case diagnosed by echocardiography was by Nellessen et al. in 1985. With less than hundred cases being reported so far in the literature. In previous studies, the mortality rate associated with a paradoxical embolism secondary to a thrombus-in-transit was 18%, and 66% of patients died within 24 hours (12). Considering the high mortality of a thrombus-in-transit (13), early diagnosis and treatment is vitally important. Echocardiography may not only clearly identify the location and size of emboli, but it may also be helpful for making prompt management decisions, and, when necessary, TEE can be performed successfully. Our patient presented with acute shortness of breath and was discovered to have saddle PE with massive clot burden and thrombus-in-transit straddling PFO: a dangerous diagnosis, most probably due to DVT, but without systemic embolization. Doppler examination revealed a thrombosis of the right femoral thrombosis.

The best management of PE with an entrapped thrombus in PFO is still controversial. Anticoagulation, thrombolytic therapy or surgical intervention are the treatment options; however, there is no consensus on the optimal treatment of this clinical situation.

Anticoagulation and thrombolysis therapy seem to be hazardous in patients with a large thrombus-in-transit within a PFO because of the high risk of either fragmentation or complete embolization (13). However, Rose et al. (14) reviewed many such cases during a 34-year period and showed that the mortality rate was lower after thrombolytic therapy than surgery, and based on a previous review, anticoagulant treatment appears to be an acceptable therapeutic alternative to surgery, particularly in patients with comorbidities (e.g., increased age, stroke, and progressive cancer) who are at high surgical risk and for patients with small PFO. (15) Surgical treatment appears justified in the prevention of paradoxical embolism and must be done without delay if it is the preferred treatment strategy. In a recent review, surgical thromboembolism improved survival rates and reduced ischemic stroke, compared to anticoagulation therapy. However, there was a very slight difference in mortality between patients treated with heparin and the surgical group (14% and 12%, resp.). Thrombolysis is linked to the highest mortality (36%), which could be explained by the severity of the patient's initial presentation (15). Myers et al. (2) reviewed in 2010 the observational studies on this subject to identify prognostic factors and to compare mortality and systemic embolism between treatments. Surgical thromboembolism showed a nonsignificant trend toward improved survival (odds ratio (OR), 0.65 (0.24–1.72);) and significantly reduced systemic embolism and composite of mortality and systemic embolism, compared with anticoagulation alone. Thrombolysis, on the other hand, had the opposite effect, although not to a significant level (OR, 1.62 (0.43–5.97)). Our patient was hemodynamically stable, but she reported that her chest tightness and shortness of breath were worsening. In addition, the patient's PE showed extensive bilateral involvement, including the segmental arteries. Moreover, lower extremity vascular ultrasound showed a right femoral thrombosis. Therefore, we chose thrombolytic and anticoagulation treatment, and the patient achieved a good curative effect.

## **Conclusion**

TSFO is a complication of severe thromboembolic disease, with the migration of thrombus in left-heart cavities in favour of pulmonary hypertension. Diagnosis is easy using echocardiography but it is often necessary to complete TTE with TEE to show it clearly. In addition, echocardiography is very helpful to make early management decisions and it may give valuable information regarding the response to anticoagulation during the follow-up process. We used thrombolytic and anticoagulation therapy to treat the patient and achieved complete resolution of the thrombus and the embolism. However, this treatment may not be applicable to every patient because of considerations regarding age and comorbidities.

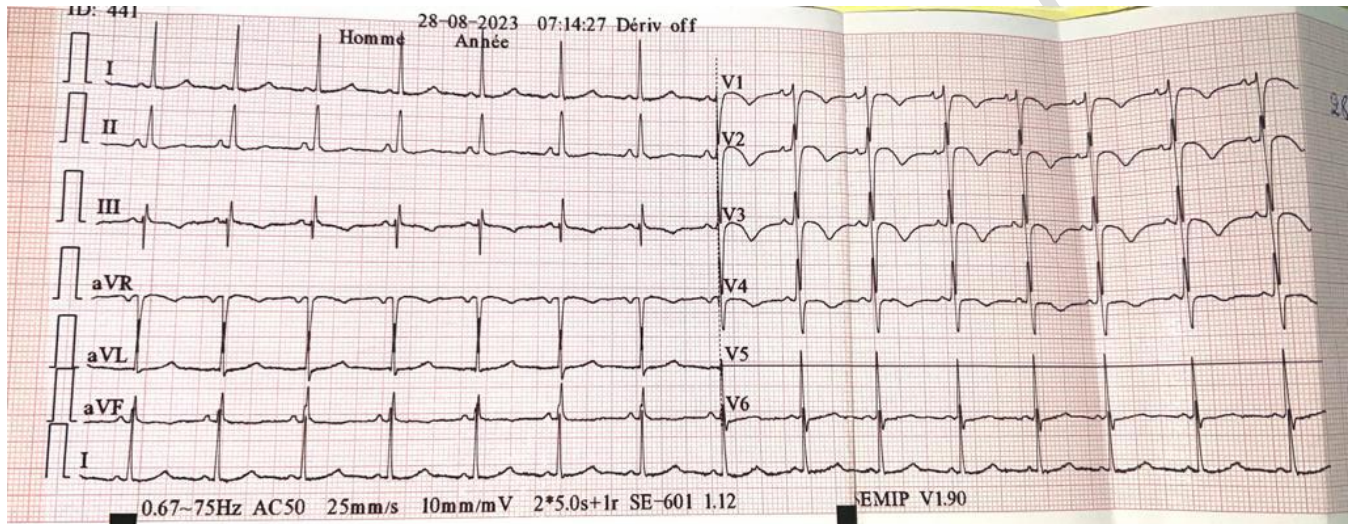
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**Figure1.** A second degreeburn of the 2 legs.



**Figure 2.** ECG shows relative negative T waves in the antero-septal leads.



**Figure 3.** Echocardiogram showing a large snake-like thrombus in the right atrium.



**Figure 4a .** Echocardiogram showing a serpentine thrombus in the left atrium.



**Figure 5.** Echocardiography showed a large thrombus entrapped in the patent foramen ovale.



**Figure 6.** Thoracic angio-CT scan: shows massive bilateral pulmonary embolism.