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CASE REPORT
Cardiac imaging

Case report: Thrombus in transit—a cause of impending paradoxical embolism

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Background A thrombus in transit (TIT) is a life-threatening condition associated with pulmonary embolism (PE). While TIT was once considered a rare diagnosis, its emergence has risen in recent years mainly through advancement in medical technology. Rare cases of a thrombus in transit crossing a patent foramen ovale in the context of pulmonary embolism have been reported. The appropriate treatment of patients in this setting remains unclear.

Case summary We describe a 64-year-old man who presented with syncope to the emergency room secondary to acute pulmonary embolism. Initial transthoracic echocardiogram revealed a large intracardiac thrombus in transit across a patent foramen ovale, verified by transoesophageal echocardiogram. He underwent anticoagulation and urgent surgical thrombectomy with a favourable outcome.

Discussion Risk stratification of patient with acute PE is mandatory for determining the appropriate therapeutic management. Initial risk stratification is based on clinical symptoms and signs of haemodynamic instability which indicate a high risk or early death associated massive PE. Thrombolytic therapy is indicated in high-risk patients (Grade 1B), while anticoagulation alone is recommended for intermediate-high- to low-risk patients. Assessment for intracardiac thrombi in PE modifies the treatment strategy in case of a thrombus in transit.

Keywords Pulmonary embolism • Thrombus in transit • Patent foramen ovale • Transoesophageal echocardiogram • Case report • Syncope

Learning points

- Pulmonary embolism is recognized as a cause of syncope although its actual prevalence among patients admitted to hospital was until recently unknown. Pulmonary embolism will be identified in nearly one of every six patients hospitalized for the first episode of syncope.
- Paradoxical embolism is a potential complication of a thrombus in transit. An entrapped embolus through a patent foramen ovale is an extremely rare finding.
- Assessment for intracardiac thrombi in pulmonary embolism modifies the treatment strategy especially in the scenario of a thrombus in transit. Concerns pertaining to thrombus dislodgement usually favours emergency surgical thrombectomy.

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Introduction

A thrombus in transit (TT) is a life-threatening condition associated with pulmonary embolism (PE). While TT was once considered a rare diagnosis, its emergence has risen in recent years mainly through advancement in medical technology. Rare cases of a thrombus in transit crossing a patent foramen ovale in the context of pulmonary embolism have been reported, the appropriate treatment of patient in this setting remains unclear. We describe a 64-year-old man who presented with syncope to the emergency room secondary to acute pulmonary embolism.

Timeline

Timeline	Description
Day 0	A 64-year-old male patient presented to the emergency room following sudden loss of consciousness.
Day 0–10 min after admission	Initial electrocardiogram showed sinus rhythm, incomplete right bundle branch block and S1Q3T3 pattern
Day 0–15 min after admission	Initial echocardiogram revealed non-dilated right ventricle with reduced right ventricular systolic function with right and left atrial thombi protruding through A-V valves. Probable diagnosis was made: intermediate-high-risk (submassive) pulmonary embolism with a Thrombus in transit
Day 0–30 min after admission	A triple rule-out protocol computed tomography angiography revealed multiple filling defects involving subsegmental branches of right and left pulmonary arteries
Day 0–2 h after admission	Urgent transoesophageal echocardiography confirmed the diagnosis of thrombus in transit across patent foramen ovale. Initial anticoagulation with low molecular weight heparin was initiated.
Day 0–8 h after admission	Patient underwent surgical thrombectomy with removal of thrombus from patent foramen ovale as well as small thrombus in both right and left main pulmonary arteries.
Day 0–14 h after admission	Patient enters post-surgical therapy intubated with inotropic and vasopressor support.
Day 1	Optimization of fluid therapy and removal of vasopressor and inotropic support, weaning and removal of mechanical ventilation.
Day 2	Respiratory therapy is started with incentive inspirometer.
Day 3	Removal of mediastinal tubes with subsequent mobilization. Oral anticoagulation with rivaroxaban 15 mg b.i.d. is started.
Day 6	Patient is discharged from post-surgical therapy to hospitalization. Post-surgical control echocardiogram is performed.
Day 8	Phase 1 of cardiac rehabilitation begins
Day 12	Coagulopathy workup is started.
Day 14	Patient is evaluated by pulmonary hypertension clinic without further changes in treatment. Patient undergoes abdomino-pelvic computed tomography scan without finding evidence of cancer with a normal coagulation panel.
Day 18	Patient is given a control appointment for phase 2 of cardiac rehabilitation. Patient is classified as provoked pulmonary thromboembolism do to antecedent of significant immobility (spends a substantial proportion of the day in bed or in a chair).
Day 20	Hospital discharge.

Case presentation

A 64-year-old male patient presented to the emergency room following sudden loss of consciousness witnessed by his wife. The patient was unresponsive for 1 min and regained consciousness spontaneously. He described 1 week of resting and exertional angina while performing usual daily activities followed by progressive dyspnoea 3 days prior to admission.

The patient had a history of diabetes mellitus, hypertension, and coronary artery disease due to a history of an acute non-reperfused anterior myocardial infarction 10 years prior to presentation as well as deep venous thrombosis 3 years prior to current admission. The patient had abandoned medical treatment by choice 1 year prior. Significant immobility within 3 months was identified (substantial proportion of the day in bed or in a chair). There was no family history of heart disease.

Initial assessment revealed blood pressure 130/70 mmHg, heart rhythm regular, tachycardia with a rate of 107 beats/min, respiratory rate of 28 breaths/min, and a room oxygen saturation of 88%. Physical examination of his head and neck was normal. Chest wall examination was normal without any abnormal movement or tenderness. Patient's lungs were clear to auscultation bilaterally, and no wheezing or crackles were appreciated. Cardiac and abdominal examinations were unremarkable. Examination of extremities was normal without any oedema or signs of a deep venous thrombosis (DVT).

Given the initial exertional angina and progressively worsening dyspnoea in a patient with cardiovascular risk factors, there were initial

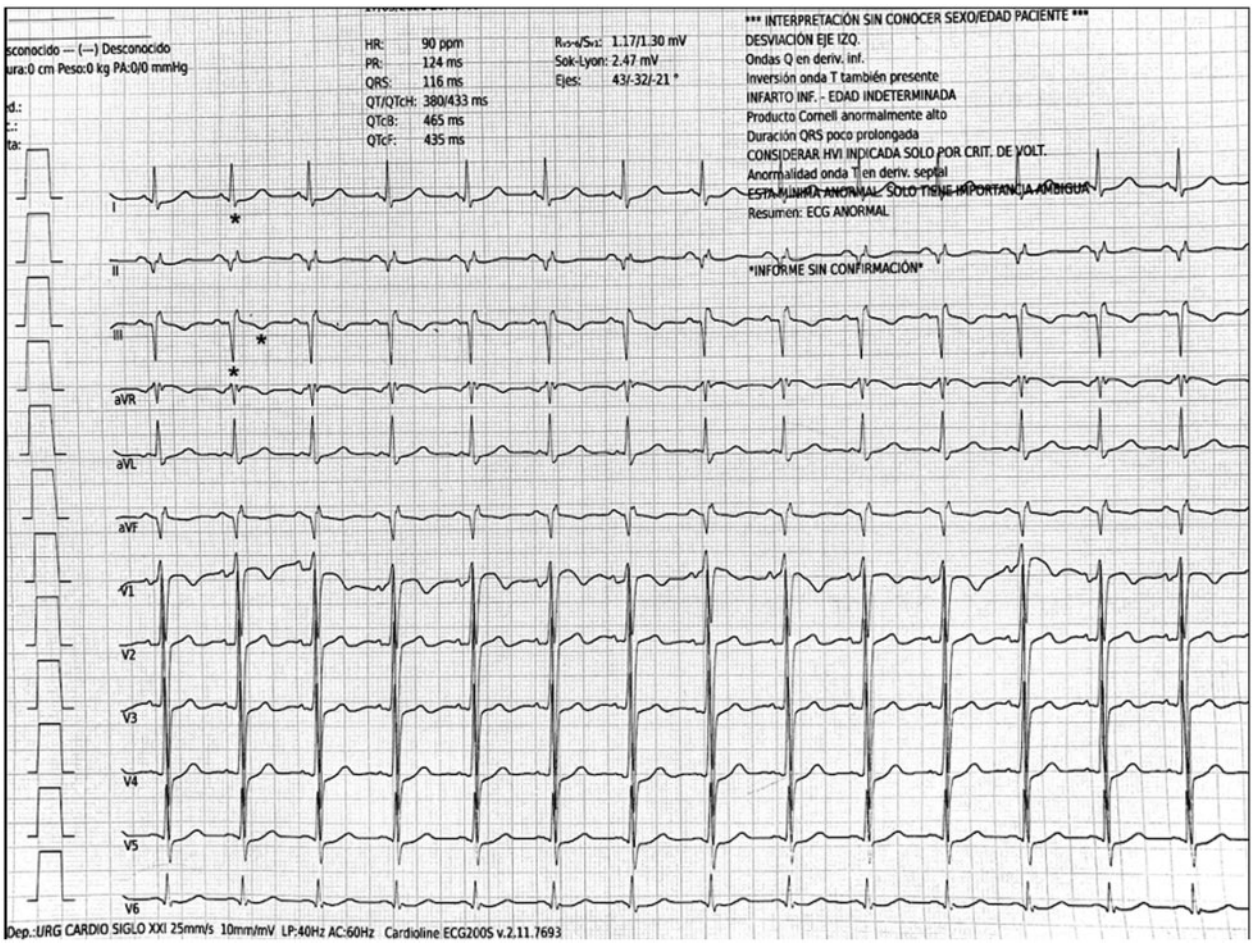


Figure 1 The electrocardiogram showed sinus rhythm, incomplete right bundle branch block and S1Q3T3 pattern (asterisk).

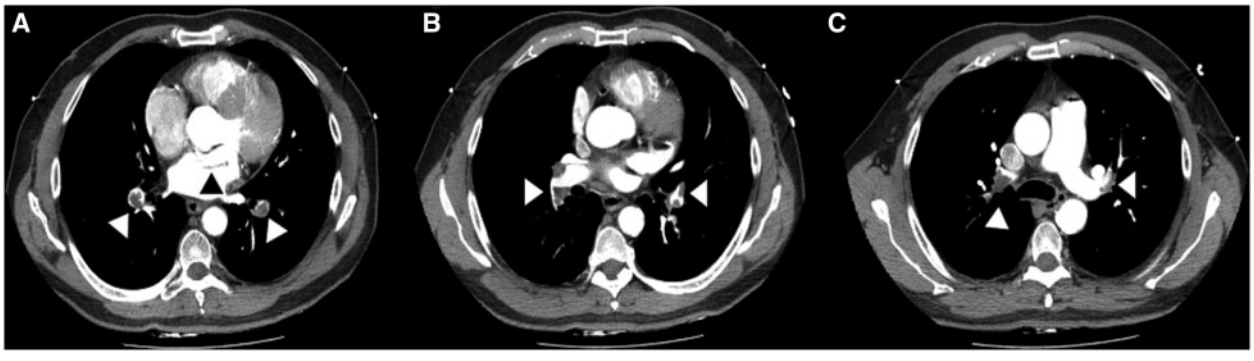


Figure 2 (*A*) Axial images showing left atrial thrombi (black arrow) as well as subsegmental emboli (white arrows). (*B*, *C*) Axial images through right and left pulmonary arteries revealing subsegmental emboli (white arrows).

concerns for an acute coronary syndrome vs. heart failure. Pulmonary embolism was also in the differential diagnosis [Wells Score 3 points, calculated from summation of tachycardia and previous, objectively diagnosed pulmonary embolism (PE) or DVT; Geneva Score 8 points, calculated from summation of tachycardia and previous DVT].

Initial electrocardiogram showed sinus rhythm with S1Q3T3 pattern (McGinn–White sign) with incomplete right bundle branch block (*Figure 1*). Laboratory results were as follows: troponin T hs, 94.6 pg/mL (reference 12.7–24.9 pg/mL); platelets 179 000 (reference 150 000–450 000 platelets/μl); prothrombin time, 10.8 s (reference

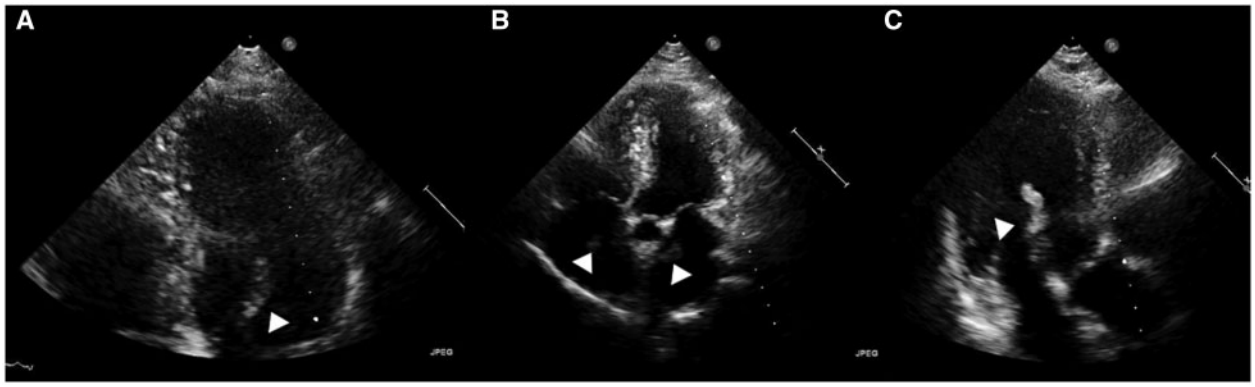


Figure 3 (*A*) Apical four-chamber view left ventricular-focused revealing left atrial mass protruding through mitral. (*B*) Apical five-chamber view revealing right and left atrial masses. (*C*) Apical 3 Chamber revealing left atrial mass observed in four-chamber view. See *Video 1*.

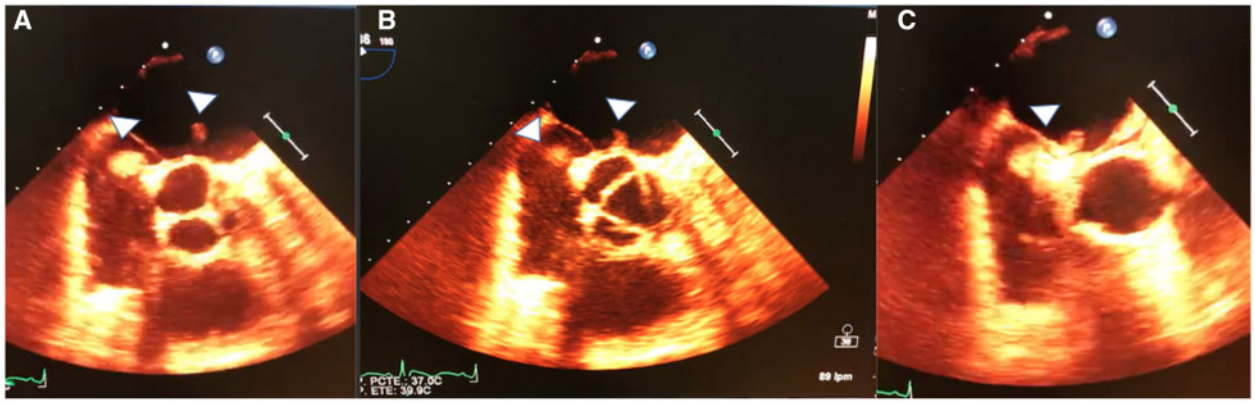


Figure 4 (*A,B*) Mid-oesophageal right ventricular inflow–outflow view revealing a thrombus in both right and left atria. (*C*) Modified mid-oesophageal right ventricular inflow–outflow view revealing a thrombus in transit though patent foramen ovale. See *Video 2*.

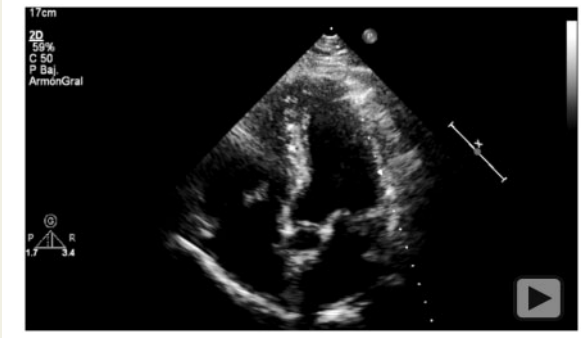
9.4–12.5 s); activated partial thromboplastin time, 29.2 s (reference 25.4–36.9 s). Chest X-ray was unremarkable. Venous ultrasound Duplex showed deep vein thrombosis of right common femoral vein. A triple rule-out protocol computed tomography angiography revealed multiple filling defects involving subsegmental branches of right and left pulmonary arteries as well as a thrombus in left atria (*Figure 2*).

Bedside transthoracic echocardiography demonstrated reduced left ventricular systolic function (27%) with impaired segmental mobility in the territory of the anterior descending artery, non-dilated right ventricle with reduced right ventricular systolic function (tricuspid annular plane systolic excursion 13 mm, Tricuspid S wave 6.8 cm/s). Right and left atrial thrombi protruding through A-V valves was identified with a high suspicion for thrombus in transit though a patent foramen ovale (*Figure 3*, *Video 1*). Urgent transoesophageal echocardiography confirmed the diagnosis of thrombus in transit across patent foramen ovale (*Figure 4*, *Video 2*).

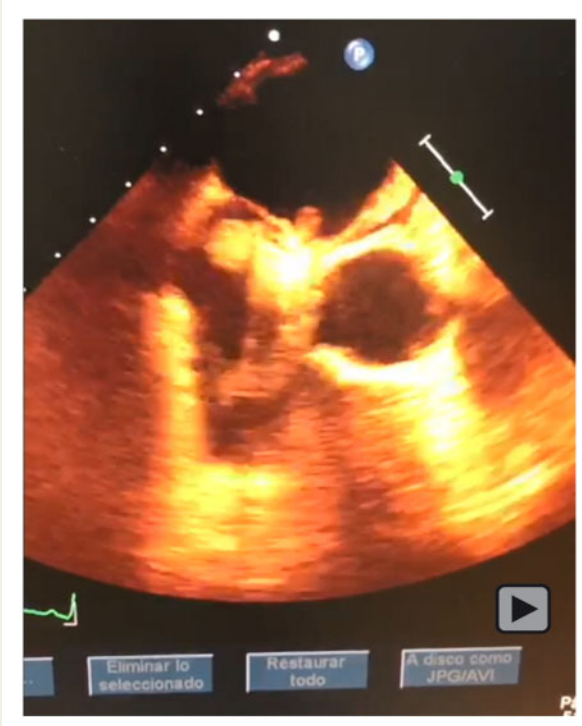
Heart team advice was sought taking into consideration the potential risk of thrombus dislodgement that could risk systemic embolization. The patient was classified as an intermediate-high-risk case. The patient scored 98 points on the Pulmonary Embolism Severity Index (PESI) (Class III). There was right ventricular dysfunction on transthoracic echocardiography as well as elevated cardiac troponin levels without haemodynamic instability. Initial anticoagulation with low molecular weight heparin was initiated at a dose of 1 mg/kg every 12 h. The patient underwent surgical thrombectomy with removal of thrombus from the patent foramen ovale, as well as a small thrombus in both right and left main pulmonary arteries.

Discussion

Risk stratification of patients with acute PE is crucial for determining the appropriate therapeutic strategy (Grade 1B). Initial risk



Video 1 Transthoracic echocardiography in an apical four chamber view revealing right and left atrial thombi protruding through A-V valves with a high suspicion for thrombus in transit though a patent foramen ovale.



Video 2 Transoesophageal echocardiography confirming diagnosis of thrombus in transit across patent foramen ovale.

stratification is based on clinical symptoms and signs of haemodynamic instability which indicate a high risk or early death associated massive PE. Thrombolytic therapy is indicated in high-risk patients (Grade 1B), while anticoagulation alone is recommended for intermediate-high- to low-risk patients.[1] Among patients with submassive PE, namely those who are haemodynamically stable but have signs of RV dysfunction, the selection of patients who may benefit from thrombolytic therapy remains to be determined. Anticoagulation remains the initial preferred choice. Thrombolytics are considered in cases with clinical haemodynamic deterioration (Grade 1B).[2]

Pulmonary embolism is recognized as a cause of syncope although its actual prevalence among patients admitted to hospital was until recently unknown.[3] The Pulmonary Embolism in Syncope Italian Trial (PESIT) was a cross-sectional study aimed at determining the prevalence of PE in patients admitted to the hospital with first episode of syncope regardless of clinical suspicion. In patients admitted for first episode of syncope, nearly one of every six patients will be diagnosed with pulmonary embolism after standardized, guideline-based inpatient evaluation.[3]

Patent foramen ovale (PFO) has an estimated prevalence of 27.5%.[4] It is an increasingly investigated cause of cryptogenic embolic stroke. An entrapped embolus through this foramen is an extremely rare finding. Fauveauau *et al.*[5] reviewed all reported cases of thrombus straddling the PFO published between 1985 and 2007. Thrombolysis was more frequently chosen in the higher risk group but was associated with the greatest mortality. Surgery seems to be justified in the prevention of paradoxical embolism.

A 64-year-old man admitted to the hospital for syncope was diagnosed with acute submassive PE with a thrombus in transit across a PFO. The patient was classified as an intermediate-high-risk patient, a category in which the benefit of systemic thrombolytics is limited.[2] Surgical thrombectomy was performed, favoured over catheter-based thrombolytic therapy and systemic thrombolysis due to concerns of thrombus dislodgement. The patient had a favourable post-operative outcome. The patient was discharged to cardiac rehabilitation with oral anticoagulation for 3 months in the context of provoked venous thromboembolism with rivaroxaban 15 mg twice daily for 21 days followed by 20 mg once daily. This case highlights the importance of non-invasive tests for identifying thrombus in PE as well as the role of imaging during decision-making for determining the appropriate therapeutic strategy.

Lead author biography

Rodrigo Núñez Méndez was born in Mexicali, Baja California on 5 December 1990. He studied Medicine at the Autonomous University of Baja California. Since 2019, he is Cardiology resident at UMAE Cardiology Hospital IMSS with interest in Cardiac Intensive Care.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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References

1. Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing G-J, Harjola V-P *et al.* ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS): the Task Force for the diagnosis and management of acute pulmonary embolism of the European Society of Cardiology (ESC). *Eur Heart Journal* 2020;**41**: 543–603.
2. Meyer G, Vicaut E, Danays T, Agnelli G, Becattini C, Beyer-Westendorf J *et al.* Fibrinolysis for patients with intermediate-risk pulmonary embolism. *N Engl J Med* 2014;**370**:1402–1411.
3. Prandoni P, Lensing AWA, Prins MH, Ciammaichella M, Perlati M, Mumoli N *et al.* Prevalence of pulmonary embolism among patients hospitalized for syncope. *N Engl J Med* 2016;**375**:1524–1531.
4. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. *Mayo Clin Proc* 1984;**59**:17–20.
5. Fauveau E, Cohen A, Bonnet N, Gacem K, Lardoux H. Surgical or medical treatment for thrombus straddling the patent foramen ovale: impending paradoxical embolism. Report of four clinical cases and literature review. *Arch Cardiovasc Dis* 2008;**101**:637–644.