ESC European Society of Cardiology

European Heart Journal - Case Reports (2021) **5**(8), 1–6
doi:10.1093/ehjcr/ytab282

CASE REPORT
Heart Failure

Case report of subacute presentation of tricuspid valve thrombus complicated by widespread bilateral pulmonary emboli: a multifactorial aetiology

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Received 21 December 2020; first decision 27 January 2021; accepted 30 June 2021

Background Right heart thrombus (RHTh) complicated by pulmonary embolism (PE) usually presents as a medical emergency with significant haemodynamic instability. However, less is known about subacute presentations.

Case summary We present a 74-year-old haemodynamically stable gentleman with a 3-week history of mild pleuritic chest pain and exertional dyspnoea preceded by lower respiratory tract infection. Early trans-thoracic echocardiogram (TTE) revealed a 3 cm elongated tricuspid valve thrombus with right ventricular dysfunction, new-onset atrial fibrillation, and new-onset severe left ventricular impairment. Subsequent computed tomography pulmonary angiogram showed widespread bilateral pulmonary emboli with retrograde opacification of the hepatic veins. The RHTh successfully resolved with warfarin therapy with no further complications, and the patient was discharged on Day 8 of hospitalization.

Discussion An early TTE is crucial in detecting the RHTh in patients suspected of PE and can significantly change the management compared with uncomplicated PE. The index of suspicion for PE and RHTh should remain high even in subacute cases.

Keywords Right heart thrombus • Tricuspid valve thrombus • Pulmonary embolism • Trans-thoracic echocardiogram • Case report

Learning points

- Right heart thrombus (RHTh) carries a worse prognosis and is an important differential diagnosis in patients suspected of pulmonary embolism (PE).
- Early trans-thoracic echocardiogram in patients suspected of PE is crucial in ruling out RHTh and can alter the management early.
- Recognition of trigger and prothrombotic factors are important as they have an impact on the duration of anticoagulation of this high-risk pathology.

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Handling Editor: Pierpaolo Pellicori
Peer-reviewers: Danny van der Sande; Abdullah Sayied Abdullah
Compliance Editor: Edwina McNaughton
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Introduction

Right heart thrombus (RHTh) is an infrequent finding in the presence of pulmonary embolism (PE) and has higher mortality than PE alone.[1,2] Two types of RHTh are commonly described. First, type A, highly mobile and serpiginous thrombi, usually arise from the peripheral venous system and are thought to be incidentally caught-in-transit in the right heart chambers.[3] Second, type B thrombi are less mobile, attached to the cardiac walls, and with morphology similar to that of left ventricular thrombi.[4] They are thought to form *in situ* secondary to atrial fibrillation (AF), intracardiac abnormalities or devices, and prosthetic valves.[3]

Currently, no guidelines on the optimal treatment of RHTh are available due to the lack of randomized clinical trials. Cases of RHTh in the presence of PE are commonly reported in an emergency setting and are treated with either anticoagulation, thrombolysis, surgery, or percutaneous removal.[5] We present a case of subacute bilateral pulmonary emboli and concurrent type A RHTh in a patient with new-onset AF and left ventricular (LV) dysfunction, who was successfully treated with oral anticoagulation therapy.

Timeline

Timeline	Description
Four weeks	Start of productive cough
Three weeks	Patient recalls the start of mild pleuritic chest pain, shortness of breath on exertion, and mild bilateral ankle swelling
Admission Day 0	Patient presents to the Emergency Department with worsening dyspnoea. Bedside trans-thoracic echocardiogram (TTE) reveals a large mobile structure 3 cm in length, attached to the tricuspid valve. Right ventricle is dilated with impaired function. Left ventricular ejection fraction—25–30% Computed tomography pulmonary angiogram shows widespread bilateral pulmonary emboli Therapeutic dose of enoxaparin and loop diuretics started, angiotensin-converting enzyme inhibitor held due to the mild acute kidney injury
Day 1	Warfarin therapy started with a bridging enoxaparin therapy
Day 3	Computed tomography of the abdomen and pelvis demonstrated no definite evidence of visceral malignancy
Day 6	Repeated TTE shows no evidence of any mass on his tricuspid valve or free-floating thrombus in his right ventricle
Day 7	Spironolactone started
Day 8	Patient discharged with an INR of 3.1. General practitioner to check INR in 1 week. Telephone follow-up in 1 and 3 months. Outpatient echocardiogram will assess the need for coronary angiography

Case presentation

A 74-year-old Caucasian male presented to the emergency department with a 1-month history of mild pleuritic chest pain, exertional dyspnoea, orthopnoea, and productive cough unresponsive to antibiotic therapy. His medical history was notable for chronic kidney disease, hypertension, and ischaemic heart disease. His regular medications on admission included bisoprolol 5 mg once daily, atorvastatin 40 mg once daily, lercanidipine 20 mg once daily, losartan 50 mg once daily, and aspirin 75 mg once daily. The patient was haemodynamically stable with a heart rate of 90 b.p.m. and blood pressure of 129/103 mmHg. His temperature was 35.8°C, and the respiratory rate was 19 breaths per minute with oxygen saturation of 94% on room air. Cardiovascular examination was remarkable for an irregular pulse and mild bilateral lower limb oedema, with no clinical evidence of deep venous thrombosis. An electrocardiogram (ECG) showed new-onset AF with a ventricular response of 95 b.p.m., with inferior and anterior T-wave inversions, indicating right heart strain (*Figure 1*).

Blood results were: haemoglobin 153 g/L (130–170 g/L), white cell count 9.4 × 10^9/L (4–9 × 10^9/L), C-reactive protein 20 mg/L (0–5 mg/L), eGFR 34 mL/min (baseline: 48 mL/min), creatinine 175 µmol/L (59–104 µmol/L), serial troponin T: 58–46 ng/L (0–14 ng/L). Chest X-ray was normal. Blood cultures remained negative for 7 days. An early point-of-care ultrasound performed in the emergency department demonstrated a right ventricular (RV) thrombus attached to the tricuspid valve, a hypertrophied left ventricle with an ejection fraction of 25–30% (50% in 2017) and a globally hypokinetic left ventricle (*Figure 2A* and *Video 1*). The apical four-chamber view showed a 3 cm elongated (type A) mobile structure attached to the tricuspid valve and dilated RV (*Figure 2B* and *C*; *Videos 2* and *3*). Subsequent computed tomography pulmonary angiogram (CTPA) revealed bilateral pulmonary emboli with thrombotic material in all lobar arteries (*Figure 3A*) and the right pulmonary artery (*Figure 3B*). Retrograde opacification of the hepatic veins was present, indicating right heart strain (*Figure 3C*). Computed tomography of the abdomen and pelvis did not identify any evidence of visceral malignancy. Carcinoembryonic antigen, alpha-fetoprotein, and carbohydrate antigen 19-9 were all within a normal range.

Given his haemodynamic stability on admission, we decided not to proceed with thrombolysis. Instead, he was promptly initiated on a bridging regime of low-molecular-weight heparin (1 mg/kg twice daily) and warfarin therapy. On Day 6, the previously seen mobile structure was no longer visible (*Figure 2D*). Furosemide 40 mg once daily was started on admission for his mild peripheral oedema. Considering his severe left ventricular systolic dysfunction (LVSD), spironolactone 12.5 mg once daily was started, and his bisoprolol up-titrated to 7.5 mg once daily. The angiotensin receptor blocker, initially held due to the acute kidney injury, was re-started after his renal function returned to baseline with the aim of up-titration in the future. Due to the patient's improvement on anticoagulation, the absence of cardiac chest pain and specific ECG changes, coronary angiography was cancelled and will instead be done after a follow-up echocardiogram. Due to the widespread nature of the PE, the presence of RHTh, and the lack of any evidence of malignancy, haematology was consulted. After discussion, the thrombophilia screen was deemed unnecessary due to his recent immobility, AF, and congestive

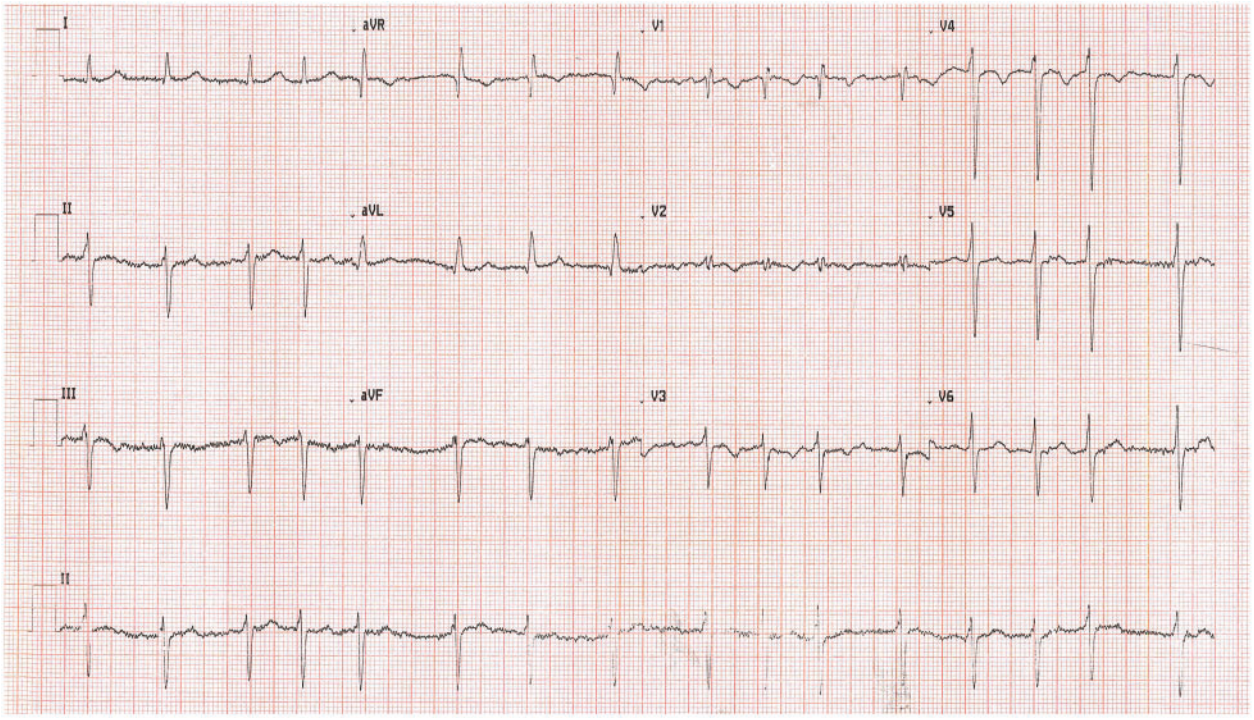


Figure 1 A 12-lead electrocardiogram showing atrial fibrillation with T-wave inversions in leads III, aVF, and in precordial leads V1, V2, V3, and V4.

heart failure. Follow-up was performed at 1 and 3 months, where the patient reported diminished breathlessness, resolved orthopnoea and increased exercise tolerance.

Discussion

Whilst RHTh complicated by PE usually presents as an emergency, this case provides evidence of subacute presentation further complicated by new-onset AF and LVSD. Several important points emerge from this case.

The subacute presentation complicated by new-onset AF and LVSD highlights the difficulty in establishing the exact aetiology. The preceding lower respiratory tract infection (LRTI) could have acted as a trigger for new-onset AF. Being a prothrombotic state, AF, coupled with reduced mobility from exertional breathlessness, may have precipitated a peripheral thrombus formation.[6] The elongated shape of the RHTh supports this, i.e. the thrombus dislodged from the periphery and was captured in-transit by the tricuspid valve.[3] Alternatively, the LRTI could have contributed to venous stasis and blood procoagulability and, in turn, increase his risk of peripheral venous thromboembolism. The widespread PE could have subsequently triggered the new-onset AF and, in turn, led to congestive cardiac failure, although it is difficult to determine their temporal relations.[7]

Early trans-thoracic echocardiograms (TTEs) is a useful screening test and should be considered in patients with suspected or confirmed PE to assess the right ventricular function and rule out RHTh, whose presence can significantly change the management.[8,9] Previous reports suggest that RHTh might be identified in up to 4.5% of patients.[2,10] However, the true incidence is likely to be underreported due to the low sensitivity of TTEs compared with trans-oesophageal echocardiograms.[1,11,12]

Describing the specific shape of the RHTh can help estimate its origin and the likelihood of causing PE. The mobile type A RHTh carries a high risk of severe PE with an early (≤8 days) mortality of 28–42%, compared with a mortality rate of 2.5% in acute PE alone.[1,3,13] In contrast, the less mobile type B thrombus likely originates within the cardiac chambers, is less likely to embolize and is thought to carry better outcomes.[3]

With no available guidelines, treatment of RHTh must be individualized. The main treatment options are anticoagulation, systemic or catheter-directed thrombolysis, and surgical or percutaneous embolectomy.[5] Thrombolysis has been described to have better outcomes than anticoagulation alone or surgery,[1] but a report of sudden near-catastrophic embolization post-thrombolysis has been described.[14] Thrombolysis should be particularly cautioned in type B thrombus in fear of dissolving the stalk connecting the thrombus to the cardiac wall.[4] In contrast, Barrios *et al.*[15] showed no significant difference in mortality in patients receiving anticoagulation therapy alone vs. anticoagulation with reperfusion therapy. Only limited data are available on the time it takes to dissolve RHTh. In this case, the mobile clot seen on admission was no longer visible on Day 6. Therefore, the RHTh has either dissolved secondary to the anticoagulation therapy or embolized without causing any further symptoms. Ferrari *et al.*[16] reported the disappearance of RHTh 2 h after thrombolysis in 50% of patients. For one patient on heparin infusion, the clot disappeared on Day 6 with an improvement of RV haemodynamics. Surgery is often the treatment of choice in very large RHTh that is not amenable to thrombolysis or is complicated by structural

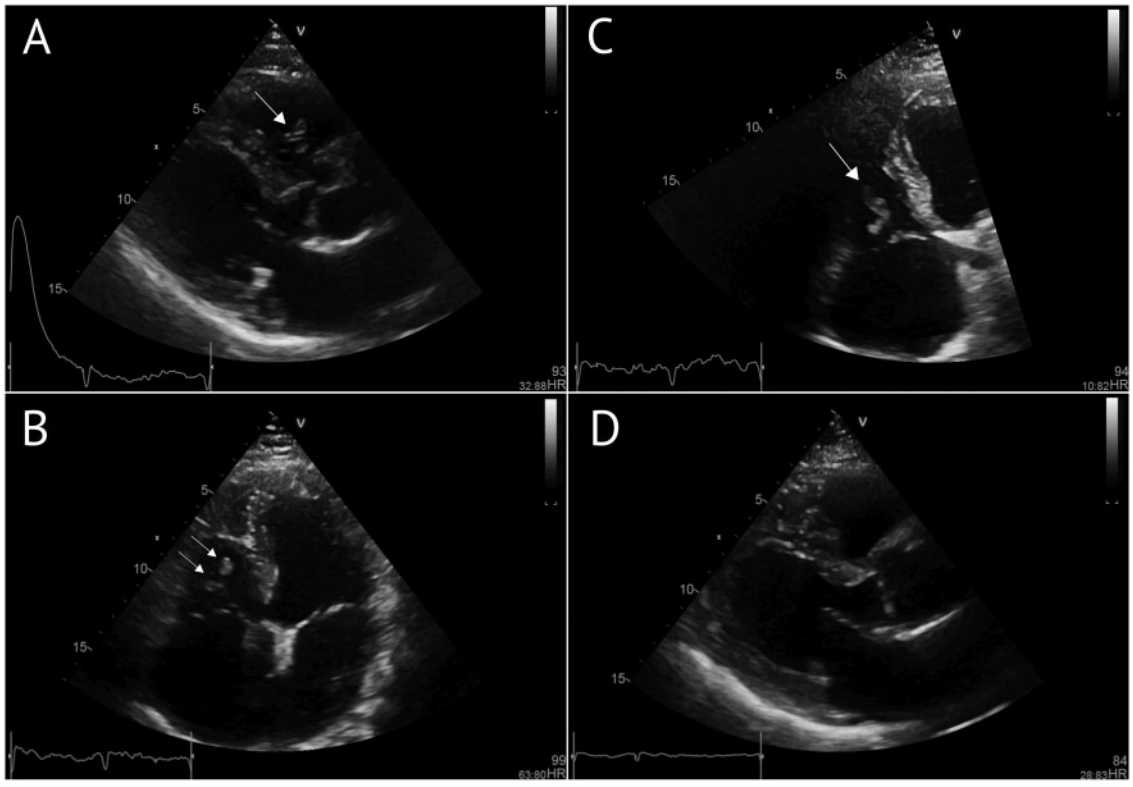
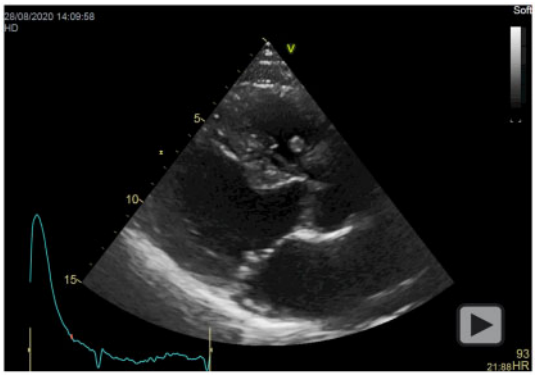
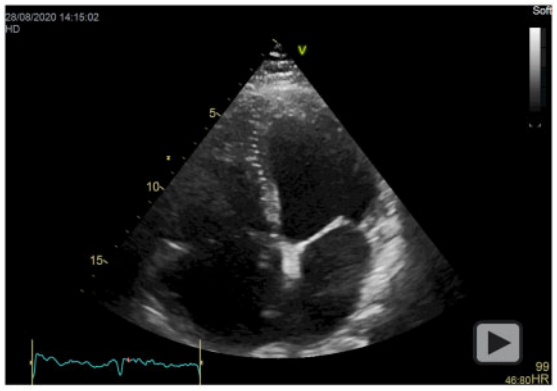


Figure 2 (*A*) Parasternal long axis of the heart demonstrating a right ventricular thrombus attached to the tricuspid valve and severe left ventricular impairment; (*B*) Apical four-chamber view of the heart demonstrating a 3 cm long right ventricular thrombus attached to the tricuspid valve, enlarged right ventricle with impairment and deviation of the interventricular septum towards the left ventricle; (*C*) Apical four-chamber view focused on the right ventricle. This view shows a much clearer outline of the elongated tricuspid valve thrombus; (*D*) Day 6 after admission: Parasternal long-axis view of the heart showing the disappearance of the right ventricular thrombus.



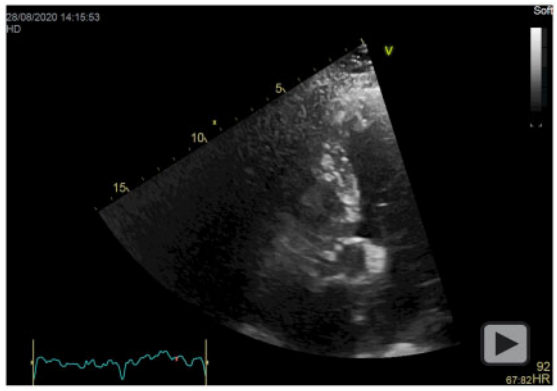
Video 1 Parasternal long axis of the heart demonstrating a right ventricular thrombus attached to the tricuspid valve and severe left ventricular impairment.



Video 2 Apical four-chamber view of the heart demonstrating a 3 cm long right ventricular thrombus attached to the tricuspid valve, enlarged right ventricle with impairment and deviation of the interventricular septum towards the left ventricle.

heart defects.[17] Percutaneous embolectomy offers a less invasive approach than surgery but carries a risk of cardiac tamponade, displacing the thrombotic material and pulmonary haemorrhage.[4]

Lastly, this case raises a few interesting management dilemmas. After prompt diagnosis and treatment of the RHTh with the bilateral



Video 3 Apical four-chamber view focused on the right ventricle. This view shows a much clearer outline of the elongated tricuspid valve thrombus.

PE, the severe LVSD, and new-onset AF must be addressed. Given the patient's previous history of mild coronary artery disease, we had a low threshold for coronary angiography after his CTPA. However, this was eventually cancelled due to the rapid improvement with anticoagulation, the absence of cardiac chest pain and specific ECG signs. Furthermore, despite this being the patient's first PE, his new-onset AF with a CHA_2DS_2-VASc score of 6 requires lifelong anticoagulation. In cases without AF, anticoagulation would be typically continued for a minimum of 3–6 months.[18]

Lead author biography

Dr Libor Myslivecek obtained his MBChB degree from University of Bristol in 2019. He is currently working as a foundation year trainee in Wye Valley NHS Trust.

Supplementary material

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

Slide sets: A fully edited slide set detailing this case 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.

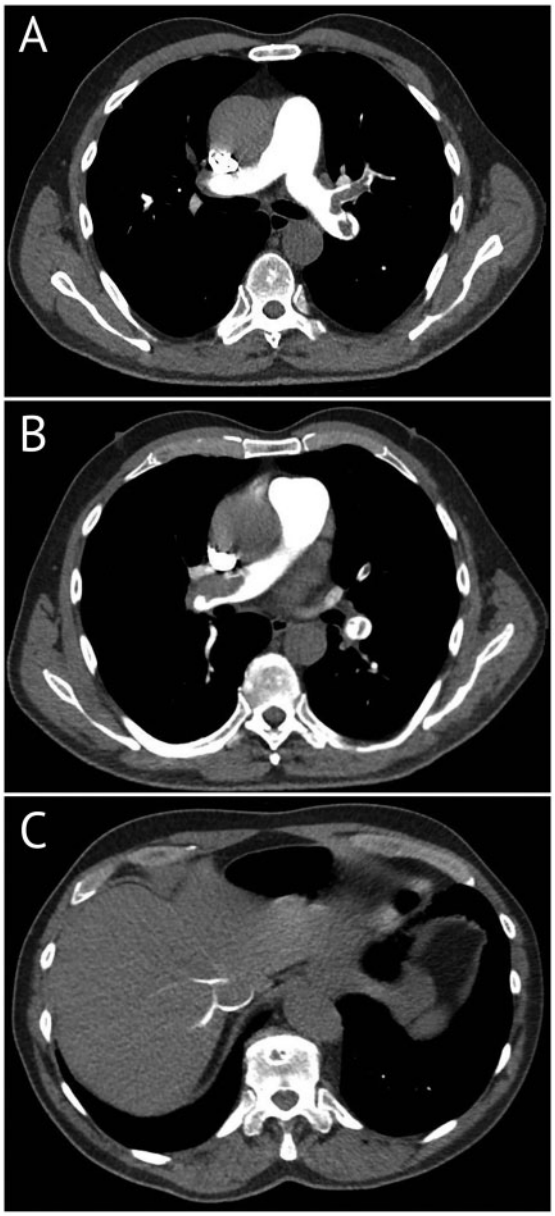


Figure 3 (*A*) Computed tomography pulmonary angiogram demonstrating multiple pulmonary emboli in the left lobar arteries; (*B*) computed tomography pulmonary angiogram demonstrating a large thrombus in the right pulmonary artery; (*C*) computed tomography pulmonary angiogram demonstrating retrograde opacification of the hepatic veins indicating right heart strain.

Conflict of interest: None declared.

Funding: None declared.

References

1. Rose PS, Punjabi NM, Pearse DB. Treatment of right heart thromboemboli. *Chest* 2002;**121**:806–814.
2. Barrios D, Rosa-Salazar V, Jiménez D, Morillo R, Muriel A, Toro J et al. Right heart thrombi in pulmonary embolism. *Eur Respir J* 2016;**48**:1377–1385.

3. Kronik G; The European Working Group on Echocardiography. The European Cooperative Study on the clinical significance of right heart thrombi. *Eur Heart J* 1989;**10**:1046–1059.
4. Thompson CA, Skelton TN, Jackson M. Thromboembolism in the right side of the heart. *South Med J* 1999;**92**:826–830.
5. Dincer HE. Right heart thrombus. *Clin Pulm Med* 2012;**19**:226–231.
6. Enga KF, Rye-Holmboe I, Hald EM, Løchen ML, Mathiesen EB, Njølstad I et al. Atrial fibrillation and future risk of venous thromboembolism: the Tromsø study. *J Thromb Haemost* 2015;**13**:10–16.
7. Wang TJ, Larson MG, Levy D, Vasan RS, Leip EP, Wolf PA et al. Temporal relations of atrial fibrillation and congestive heart failure and their joint influence on mortality: the Framingham Heart Study. *Circulation* 2003;**107**: 2920–2925.
8. Bikdeli B, Lobo JL, Jiménez D, Green P, Fernández-Capitán C, Bura-Riviere A et al. Early use of echocardiography in patients with acute pulmonary embolism: findings from the RIETE registry. *J Am Heart Assoc* 2018;**7**:1–7.
9. Jammal M, Milano P, Cardenas R, Mailhot T, Mandavia D, Perera P. The diagnosis of right heart thrombus by focused cardiac ultrasound in a critically ill patient in compensated shock. *Crit Ultrasound J* 2015;**7**:6.
10. Casazza F, Becattini C, Guglielmelli E, Floriani I, Morrone V, Caponi C et al. Prognostic significance of free-floating right heart thromboemboli in acute pulmonary embolism. *Thromb Haemost* 2014;**111**:53–57.
11. Obeid A, Mudamgha AA, Smulyan H. Diagnosis of right atrial mass lesions by transesophageal and transthoracic. *Chest* 1993;**103**:1447–1451.
12. Schwartzbard AZ, Tunick PA, Rosenzweig BP, Kronzon I. The role of transesophageal echocardiography in the diagnosis and treatment of right atrial thrombi. *J Am Soc Echocardiogr* 1999;**12**:64–69.
13. Carson JL, Kelley MA, Duff A, Weg JG, Fulkerson WJ, Palevsky HI et al. The clinical course of pulmonary embolism. *N Engl J Med* 1992;**326**:1240–1245.
14. Shankarappa R, Math RS, Papaiah S, Channabasappa YM, Karur S, Nanjappa M. Free floating right atrial thrombus with massive pulmonary embolism: near catastrophic course following thrombolytic therapy. *Indian Heart J* 2013;**65**:460–463.
15. Barrios D, Chavant J, Jiménez D, Bertoletti L, Rosa-Salazar V. Treatment of right heart thrombi associated with acute pulmonary embolism. Am J Med 2016;130; 588–595.
16. Ferrari E, Benhamou M, Berthier F, Baudouy M. Mobile thrombi of the right heart in pulmonary embolism. *Chest* 2005;**127**:1051–1053.
17. Kumar USD, Nareppa U, Shetty SP, Wali M. Right ventricular thrombus in case of atrial septal defect with massive pulmonary embolism: a diagnostic dilemma. Ann Card Anaesth. 2016;**19**:173–176
18. Konstantinides SV, Germany C, France MH, Sian C, United J, Jime D. 2019 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 J 2020;**41**:543–603.