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CASE SERIES
Cardiac Imaging

Right ventricular thrombus, a challenge in imaging diagnostics: a case series

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Background Presence of right ventricular thrombus (RVT) is a rare but life-threatening condition, thus immediate diagnosis and therapy are mandatory. Unfortunately, detection and distinction from intraventricular tumour masses or vegetations represent a complex task. Furthermore, consecutive therapy is principally led by clinical presentation without considering morphological features of the thrombus. Current literature suggests a multimodal non-invasive imaging approach. In this article, we discuss the role of cardiac magnetic resonance imaging (CMR) for the detection of RVT in patients with pulmonary embolism (PE). We consider the relatively expensive and not broadly available imaging procedure and weigh it up to its assumed high sensitivity, specificity, and importance for differential diagnosis and therapeutic decision-making.

Case summary In this case series, we report three cases of RVT with concomitant PE, whereof two were missed during routine cardiac workup by transthoracic echocardiography and computer tomography. Cardiac magnetic resonance imaging led to detection and further characterization of the thrombi in both cases.

Conclusions Cardiac magnetic resonance imaging reliably detects and characterizes RVT, even under unfavourable conditions for echocardiography such as arrhythmia, adiposity, or in posterior position of RVT. Obtained information could facilitate the choice of therapeutic approach (anticoagulation vs. systemic lysis vs. surgical thrombectomy). Future risk stratification scores will promote cost-effective use of CMR.

Keywords CMR • RVT • RHT • Thrombus • Cardiac imaging • Case series

Learning points

- Cardiac magnetic resonance imaging (CMR) detects intraventricular thrombus with higher sensitivity and specificity compared to the current standard with transthoracic echocardiography (TTE) and or transoesophageal echocardiography (TOE), especially in challenging clinical conditions such as arrhythmia, adiposity, or posterior localization of thrombus in the right ventricle.
- In case of elevated risk of right ventricular thrombus, e.g. concomitant presence of pulmonary embolism, younger age (<65 years of age), bleeding events, congestive heart failure, cancer, syncopal events, transient systolic blood pressure <100 mmHg, or oxygen saturation <90% as well as inconclusive TTE and/or TOE, CMR should be considered in patients with sufficient haemodynamic stability.
- Additional detailed information about position and stability of intraventricular thrombus obtained by CMR might guide consecutive therapy.

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Introduction

Right ventricular thrombus (RVT) specifically or right heart thrombus (RHT) is an infrequent and most likely underdiagnosed life-threatening condition.[1] Current literature provides little data on prevalence, predictors, and prognosis of RHT. Several observational studies report concomitant presence of RHT in 2.6–18% of patients with pulmonary embolism (PE).[2–4] Several risk factors associated have been identified: younger age, previous bleeding events, congestive heart failure, cancer, episodes of syncope, transient systolic blood pressure <100 mmHg, and arterial oxyhaemoglobin saturation <90%.[2]

Current literature suggests a multimodal non-invasive imaging approach consisting of transthoracic echocardiography (TTE), contrast-enhanced echocardiography, respectively, and cardiac magnetic resonance imaging (CMR).[3–6]

While TTE and transoesophageal echocardiography (TOE) reach high sensitivity and specificity for detection of left ventricular thrombus,[7] sensitivity and specificity for RHT appear to be inferior. Underlying reasons may consist in disadvantageous settings of first assessment, frequently in the setting of an emergency, with arrhythmia, adiposity, and posterior position of the right ventricle.[6] Finally, echocardiography is not able to provide additional information for the differential diagnosis of cardiac masses.[5,6]

In contrast, CMR offers accurate and non-examiner dependent images. Considering T1 weighted (T1w), T1w with fat saturation and T2 weighted (T2w) sequences, first-pass perfusion, early gadolinium enhancement (EGE), and late gadolinium enhancement (LGE), additional tissue characterization and detection of vascularization of cardiac masses is possible (*Table 1*). Benefits are also reflected in high sensitivity and specificity for detection of intracavitary cardiac thrombi.[7]

We report the diagnostic procedures of three patients with PE, deep vein thrombosis (DVT), and concomitant RVT. In the first two cases, RVT's were missed during routine cardiac diagnostic but detected using CMR. In contrast, Case 3 with its prolapsing thrombus into the right ventricle, illustrates conditions in which TTE successfully detects RVT.

We discuss the role of CMR for detection of RHT and in which circumstances this costly and time-intensive procedure might be indicated.

Case Presentations

Patient 1

A 54-year-old male patient without relevant medical history was referred to the emergency department (ED) due to dyspnoea with an oxygen saturation of 85%, tachypnoea, and tachycardia. He reported a general fatigue, increased stress at work as a salesman, a single episode of angina, and a flight journey of 1.25 h 7 days prior to admission. The electrocardiogram (ECG) showed new T-wave inversions in the anterior leads (V1–V5). Laboratory analysis indicated global respiratory insufficiency, troponinemia (high sensitive troponin T of 77.3 ng/L, cut-off <14.0 ng/L), elevation of the N-terminal fragment of pro-brain natriuretic peptide (NT-proBNP), and D-Dimer. Consecutive thoracic computer tomography (CT) revealed the presence of subsegmental PE, but no pathological masses. Aetiological clarification revealed multiple DVT and elevated factor VIII in thrombophilia screening.

For cardiac assessment and additional clarification of newly observed T negativities, TTE showed a left ventricular ejection fraction of 50%, with hypokinesia, but no signs of intraventricular masses. Coronary angiography showed normal coronary arteries.

To clarify ECG and laboratory abnormalities and considering also inflammatory causes and myocarditis as aetiology, we conducted a CMR, which did not show any Gadolinium enhancement nor oedema or other abnormalities in the myocardium. As an incidental finding, a partially fixed and partially floating mass measuring 32 mm × 11 mm (*Figure 1*) at the lateral wall of the right ventricle was detected. It showed hyperintense characteristics in T1w and T2w compared to the myocardium, absent mass perfusion at first-pass perfusion and no uptake of contrast with homogeneous signal suppression within the mass at EGE and LGE (*Figure 2*), thus, according to the criteria of standardized CMR tissue characterization, indicating thrombotic nature of the mass.[8,9] Hyperintense appearance of this thrombus in T1w and T2w sequences indicated its recent character.

Considering the stable cardiopulmonary condition and morphology of the thrombus, we waived surgical embolectomy, thrombolysis or percutaneous retrieval, and continued anticoagulation instead. Follow-up CMR after 3 months showed complete resolution of the thrombus.

Timeline

Patient 1	
Day 0 (admission)	A 56-year-old man was referred to the emergency room due to pathological saturation and dyspnoea
	Computer tomography (CT) scan of the thorax: subsegmental pulmonary embolism (PE)
	Transthoracic echocardiography (TTE): Heart Failure with preserved Ejection Fraction (HFpEF ,left ventricular ejection fraction 50%) Normal right ventricular function and dimension
	Coronary angiography: no sign of coronary sclerosis
	Start of the anticoagulation with rivaroxaban
Day 5	Cardiac magnetic resonance imaging (CMR): detection of a mass in the right ventricle with slight elevated T1 and T2 signals
Day 9	Discharge from hospital
Day 111	Thrombophilia screening [elevated factor VIII activity (188%)]
Day 205	Follow-up CMR: no sign of masses in the right heart

Continued

Patient 2	
Day 0 (admission)	Hospitalization due to macro-haematuria while continuously anticoagulated with rivaroxaban (since Day–13)
Day 1	CT scan of the abdomen: showing calculus in the left renal pelvis
	Duplex sonography of the lower right leg showing deep vein thrombosis
Day 9	CT scan of the thorax showing isolated, spiculated mass of the right upper lobule
Day 10	Bronchoscopy with detection of tumoural masses in the upper right lobulus. Histological examination showing cells of an adenocarcinoma (Day 26)
Day 17	Fluordesoxyglucose (FDG) PET-CT scan indicating advanced disease
Day 23	New onset of aphasia and facial palsy on the left side
	Cranial MRI showing acute multiple bi-hemispheric and cerebellar infarcts
Day 26	Abdominal sonography showing pathological mass within the right ventricle
	CMR showing pathological multi-lobular mass in the right ventricle located between septum and moderator bundle
Day 36	Re CT scan of the thorax–abdomen due to increasing thoracal pain, showing progression of the PE
Day 131	Follow-up CMR: complete resolution of the right ventricular thrombus
Patient 3	
Day 0 (admission)	A 60-year-old man with angina and dyspnoea while on therapy with rivaroxaban
Day 1	CT scan of the thorax: bilateral central PE
	TTE: signs of right ventricular dysfunction (McConnel, D-Shaping RV/RA 53 mmHg). Floating mass between right atrium and ventricle, measuring 5 cm × 1 cm
	Anticoagulation with Fondaparinux and transfer to a tertiary centre for local lysis of the thrombus
Day 2	Local lysis with 50 mg Alteplase
Day 3	TTE: no sign of intraventricular masses. Normal cardiac function
Day 5	Start anticoagulation with Phenprocoumon
Day 7	Termination of anticoagulation with Fondaparinux
Day 12	Hospital discharge
Day 164	TTE: no sign of intraventricular masses. Normal cardiac function

Patient 2

A 53-year-old male patient presented to the ED with macroscopic haematuria during therapeutic anticoagulation with rivaroxaban after multiple unprovoked DVT's of the right leg in the medical history and no other known diseases. The patient did not report any recent flight travel, surgeries, PE, or malignant tumours. Family history was negative for malignant tumours. On clinical examination, residual swelling oedema of the right lower limb was observed. For further clarification, an abdominal CT scan showed a staghorn calculus in the left kidney. Despite therapeutic anticoagulation, the patient developed multiple left-sided DVTs. Consecutively, onset of fever and concomitant atrial fibrillation (AF) was reported. To detect possible pulmonary infection, a chest radiograph was performed which showed a transparency reduction in the right upper lobe. A CT scan of the chest confirmed a lung tumour and subsegmental PE. Histological workup of an endobronchial biopsy revealed the presence of a bronchial adenocarcinoma.

For exclusion of structural cardiopathy as aetiology for the newly diagnosed tachycardic AF, we performed a TTE, showing ventricular hypertrophy but otherwise no sign of intraventricular masses. Nevertheless, the quality of the examination was limited due to tachycardic AF.

After successful cardioversion into sinus rhythm, abdominal sonography for assessment of possible hepatic metastases revealed an abnormal mass within the right ventricle. Since it was unclear whether the mass was the result of another thrombotic event or a metastasis of the adenocarcinoma, we performed CMR for further clarification. Cardiac magnetic resonance imaging detected a highly mobile intraventricular, multi-lobular mass in the right ventricle measuring 21 mm × 18 mm, partly fixed on the septum and on the moderator bundle (*Video 1*). The mass showed an isointense signal in T1w and T2w without EGE and LGE, thus indicating thrombotic nature of the mass.

Due to the complex situation with PE and bronchial adenocarcinoma, surgical removal of the RVT was initially considered but due to cardiopulmonary instability and poor prognosis, finally declined. Therapy was switched to therapeutic dose of unfractionated heparin, under which we observed a progression and development of new bilateral PE. Finally, the anticoagulation was changed to therapeutic dose low molecular weight heparin (Dalteparin), whereafter no further thrombotic events were observed. Follow-up CMR after 3 months showed complete resolution of the RVT.

Patient 3

A 59-year-old male patient was referred to the ED with chest pain and dyspnoea. Medical history was positive for immobilizing multiple sclerosis, PE, and intermittent AF, for which anticoagulation with rivaroxaban had been implemented. Upon admission, the patient presented pathological oxygen saturation of 84%. Blood analysis

Table 1 Cardiac magnetic resonance imaging in the differential diagnosis of cardiac masses based on findings of Motwani *et al.* and Patnaik *et al.*

Cardiac mass	T1w*	T1w fat saturation	T2w	LGE
Pseudotumours				
Thrombus	Low (high if recent)	Low	Low (high if recent)	No uptake
Pericardial cyst	Low	Low	High	No uptake
Benign tumours				
Myxoma	Isointense	Isointense	High	Heterogeneous
Lipoma	High	Low	High	No uptake
Fibroma	Isointense	Isointense	Low	Hyperenhanced
Rhabdomyoma	Isointense	Isointense	Isointense/high	No/minimal uptake
Malignant tumours				
Angiosarcoma	Heterogeneous	Heterogeneous	Heterogeneous	Heterogeneous
Rhabdomyosarcoma	Isointense	Isointense	Hyperintense	Homogeneous
Undifferentiated sarcoma	Isointense	Isointense	Hyperintense	Heterogeneous/variable
Lymphoma	Isointense	Isointense	Isointense	No/minimal uptake
Metastasis	Low	Low	High	Heterogeneous

See different signal intensities for different cardiac masses.
LGE, late gadolinium enhancement; T1w*, T1 weighted; T2w, T2 weighted.

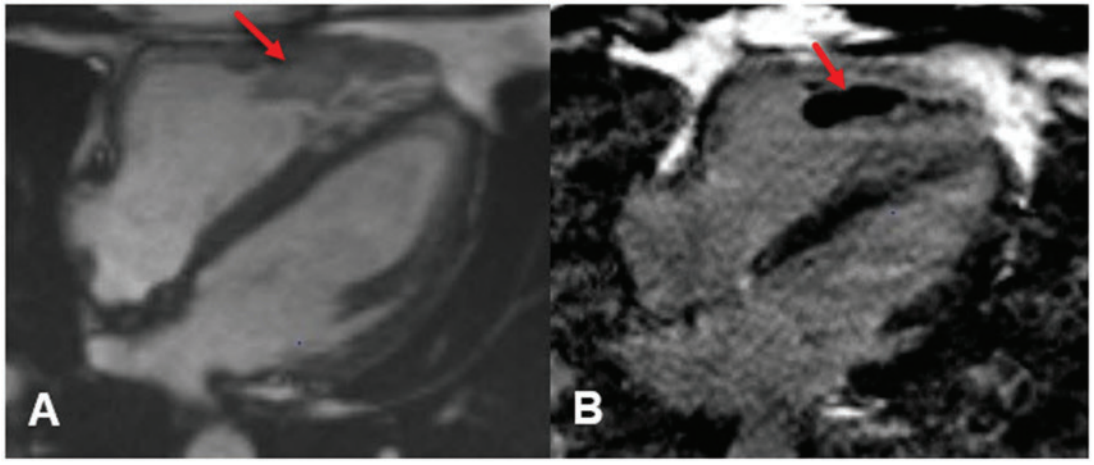


Figure 1 (*A*) Cine four-chamber view: mural thrombus fixed at the lateral wall of the right ventricle (indicated by the arrow). (*B*) Late gadolinium enhancement four-chamber view: no contrast uptake, with homogeneous signal suppression within the mass (arrow).

indicated elevation of NT-proBNP levels and troponinemia. Electrocardiogram showed sinus tachycardia without noticeable differences to previous ECGs. Since blood analysis indicated subtherapeutic rivaroxaban levels (<10.0 ng/mL measured by STAR MAX 2 ®, Hyphen Biomed SA, Neuville-sur-Oise, France) and concomitant high pre-test probability for PE, a thoracic CT scan showed bilateral paracentral PE. Due to positive history of type II heparin-induced thrombocytopenia, we started anticoagulation with Fondaparinux. Considering the presence of paracentral PE, increasing troponinemia, and elevated NT-proBNP levels, we performed routine TTE for assessment of cardiac function, in which we saw right ventricular dysfunction with positive McConnel sign, D-shaping, congested vena cava, and reduced right ventricular longitudinal function (*Videos 2 and 3*). In addition, a filiform and highly mobile mass measuring 5 cm × 1 cm, floating between right atrium and right ventricle and trespassing tricuspid valve was detected. Considering high mobility, acute onset, and absence of fever or increased inflammatory parameters, we interpreted the mass as a thrombus. Consequently, a systemic lysis was performed. In a follow-up TTE the day after, no mass could be detected. For aetiological clarification, a duplex sonography of the legs detected DVT's, while screening for thrombophilia was inconspicuous.

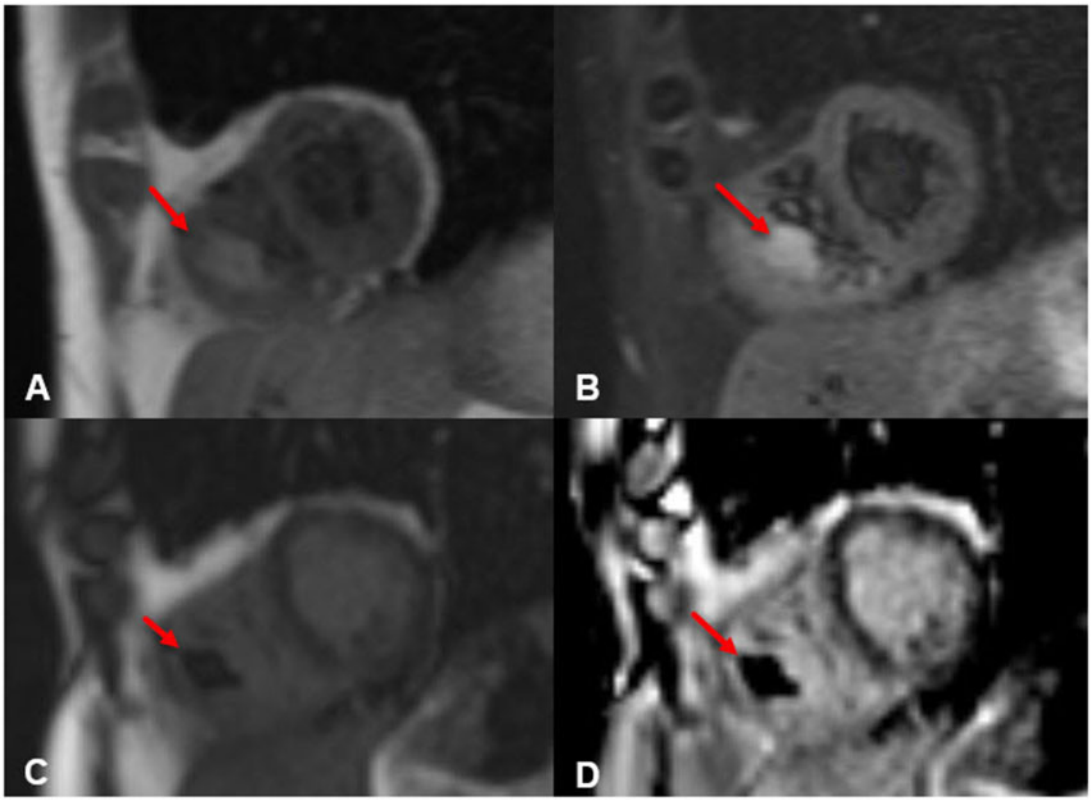


Figure 2 Tissue characterization of right ventricular thrombus: (*A*) T1-weighted and (*B*) T2-weighted short-axis view: mural mass with high signal intensity (indicated by the arrow) indicating the recent character of the thrombus. (*C*) Early gadolinium enhancement and (*D*) late gadolinium enhancement short-axis view: no uptake of contrast agent in the mass (indicated by the arrow) suggesting no vascularity within the mass.

Discussion

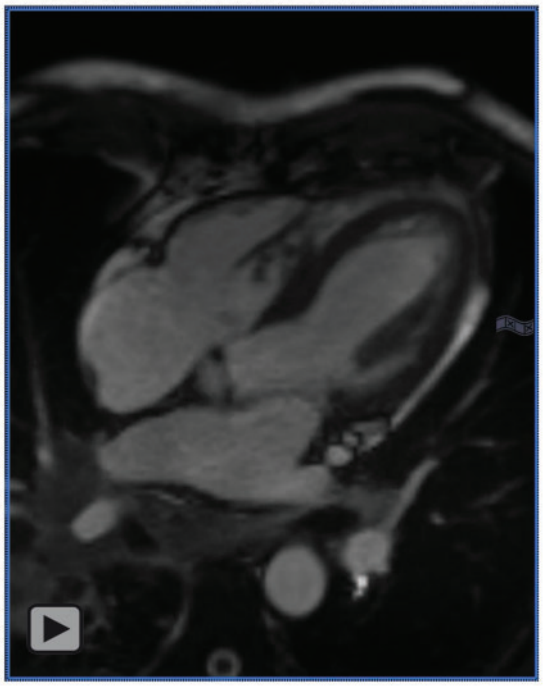
We report three cases of RHT with concomitant PE, all with a complex medical history and in two cases haemodynamical instability. Considering high mortality of RHT, which is estimated between 27% and 100%,[1,2] fast diagnosis and targeted treatment is essential. Several case reports attributed aetiology to Chagas infections,[10] takotsubo cardiomyopathy,[11] complication of myocardial infarction,[12] or abruption of DVT,[13] the latter indicating high coincidence of RHT and PE.[2–4] Furthermore, three different RHT types (A, B, and C) were described, suggesting specific aetiologies.[13] Type A describes a highly mobile serpiginous thrombus, often trapped in right heart cavities representing the result of a migration of thrombi. Hence, type A thrombi are associated with DVT and PE. Type B thrombi are fixed, *in situ* formed and associated with cardiac abnormalities. Type C assumes intermediate characteristics.[13]

Pathophysiology

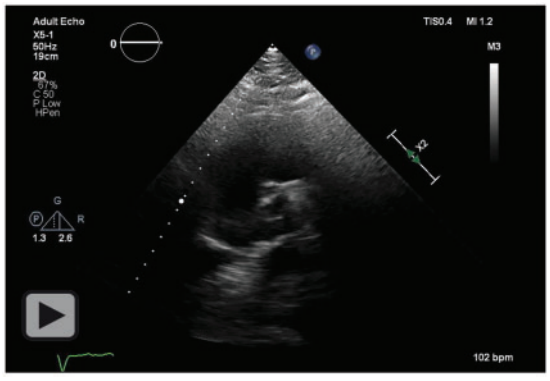
Our first case showed on the one hand generalized thrombophilia with elevated factor VIII level. On the other hand, negative T-waves, elevated troponin and NT-proBNP levels, and slightly impaired left ventricular function indicated a possible transient cardiac impairment and a takotsubo cardiomyopathy was considered as potential explanation, considering elevated stress in the patient history a week before admission. Cardiac magnetic resonance imaging findings with mural and non-mobile thrombus corresponded to above-mentioned type B thrombus. Second patient suffered generalized thrombophilia with multiple and multilocular thrombi. Considering the new diagnosis of bronchial adenocarcinoma, thrombi are most probably of paraneoplastic nature resulting from cancer-associated thrombosis. Cancer-associated thrombosis increases risk for venous thromboembolism (VTE) severalfold.[14] This RVT assumed intermediate characteristics hence corresponding to type C. In our third case, we observed subtherapeutic levels of rivaroxaban and concomitant DVT. The filiform mass detected by TTE examination corresponds to previously described type A thrombus, accompanied by multiple DVT and PE.[13]

Imaging

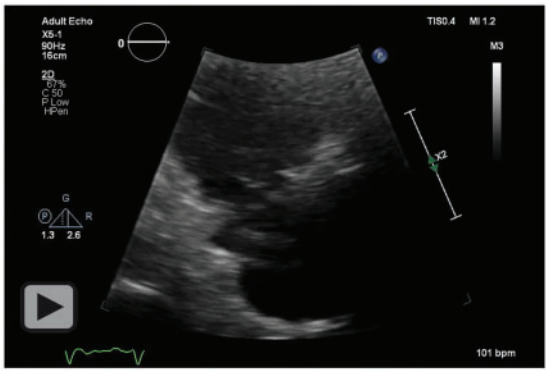
The first patient provided several disadvantageous conditions for initial assessment with unfavourable lateral mural position of the thrombus, mimicking myocardial tissue. Since thrombus and myocardium may have similar echogenicity, TTE alone is not suitable to distinguish



Video 1 Cine four-chamber view: Thrombus fixed in the right ventricle between moderator band and septum, highly mobile.



Video 2 Parasternal short-axis view with floating thrombus in the right atrium prolapsing into the right ventricle.



Video 3 Parasternal pseudo short-axis view (with right atrium and ventricle) with floating thrombus in the right atrium prolapsing into the right ventricle.

Table 2 Summary of high-risk findings according to Barrios *et al.* for right heart thrombus within reported cases

	Patient 1	Patient 2	Patient 3
Younger age (<65)	+	+	+
Bleeding events	-	-	+
Congestive heart failure	+	+	-
Presence of cancer	-	-	+
Syncopal events	-	-	-
Systolic blood pressure <100 mmHg	-	-	+
Oxygen saturation <90%	+	+	+

different types of tissues. Similarly, the second case provided unfavourable assessment conditions with tachycardic AF and haemodynamic instability. Despite floating character and distance from mural structure, RVT was missed by TTE likely because of the tachycardia. After normalization of heart rate, sonography of the upper abdomen was able to detect, though not fully characterize the mass.

In contrast, the third case shows a highly mobile and filiform thrombus, which ranged from the right atrium to the right ventricle. Floating character without broad fixation to the myocardium made it more distinguishable from surrounding structures, thus easier detectable by TTE.

In summary, unfavourable circumstances such as arrhythmia, variable posterior position of RVT, or an obese habitus can significantly decrease sensitivity and specificity of TTE and TOE. In some cases, RHT might be missed due to its proximity to myocardium and their similar echogenicity.

In contrast, CMR outperforms TTE and TOE in sensitivity and specificity for detection of the more frequent left ventricular thrombus.[7] Next to examiner independent images, CMR provides detailed information of intracardial structures using different imaging sequences like T1w with and without fat saturation, T2w, first-pass perfusion, EGE, and LGE. The relative signal intensity from a particular tissue depends principally on its proton density and the T1 and T2 relaxation times. Different tissues have different T1w and T2w relaxation times owing to different internal biochemical environments surrounding protons. By weighting images to emphasize either T1w- or

T2w-based contrast, CMR can exploit differences in signal intensity to discriminate between different tissue types.

This allows additional characterization of detected masses, therefore providing additional information for differential diagnosis.[9] Details about location, shape, and stability of the thrombus might predict risk of further embolic events[15] and facilitate the choice of must suitable therapy (anticoagulation, thrombectomy, or surgical removal). This was shown exemplary by our second case, in which CMR findings indicated a multi-lobular, stalked and therefore highly instable thrombus. Due to haemodynamic instability surgical removal was not possible, which is why oral anticoagulation was implemented. We also note that progression to PE during anticoagulation appears highly likely in presence of a highly unstable stalked thrombus.

On the other hand, CMR is a cost intensive imaging modality and not broadly available. The development of a pre-test probability score that helps to identify high-risk patients and stratifies risk for RHT would be desirable. Thus, over- or underdiagnosis with resulting economic burden could be prevented. Findings derived from the observational study of Barrios *et al.* could offer a basis for such a scoring system (*Table 2*). As additional risk factors, clinicians should also consider the presence of haematological disorders and malignant disease.

Lead author biography

Massimo Barbagallo was born in 1992 in Baden, Switzerland. He accomplished medical school at the University of Zurich in 2018, in which he started to pursue his passion for research. His great interests include cardio- and cerebrovascular diseases. Currently, he is in his second clinical year in the Department of General Medicine.

Supplementary material

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

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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 patients in line with COPE guidance.

Conflict of interest: none declared.

References

1. Chartier L, Béra J, Delomez M, Asseman P, Beregi JP, Bauchart JJ et al. Free-floating thrombi in the right heart: diagnosis, management, and prognostic indexes in 38 consecutive patients. *Circulation* 1999;**99**:2779–2783.
2. Barrios D, Rosa-Salazar V, Jiménez D, Morillo R, Muriel A, del Toro J et al. Right heart thrombi in pulmonary embolism. *Eur Respir J* 2016;**48**:1377–1385.
3. Nkoke C, Faucher O, Camus L, Flork L. Free floating right heart thrombus associated with acute pulmonary embolism: an unsettled therapeutic difficulty. *Case Rep Cardiol* 2015;**2015**:364780.
4. Ferrari E, Benhamou M, Berthier F, Baudouy M. Mobile thrombi of the right heart in pulmonary embolism: delayed disappearance after thrombolytic treatment. *Chest* 2005;**127**:1051–1053.
5. Patnaik S, Shah M, Sharma S, Ram P, Rammohan HS, Rubin A. A large mass in the right ventricle: tumor or thrombus? *Cleve Clin J Med* 2017;**84**:517–519.
6. Tsang BKT, Platts DG, Javorsky G, Brown MR. Right ventricular thrombus detection and multimodality imaging using contrast echocardiography and cardiac magnetic resonance imaging. *Heart Lung Circ* 2012;**21**:185–188.
7. Srichai MB, Junor C, Rodriguez LL, Stillman AE, Grimm RA, Lieber ML et al. Clinical, imaging, and pathological characteristics of left ventricular thrombus: a comparison of contrast-enhanced magnetic resonance imaging, transthoracic echocardiography, and transesophageal echocardiography with surgical or pathological validation. *Am Heart J [Internet]* 2006;**152**:75–84.
8. Maceira AM, Prasad SK, Khan M, Pennell DJ. Normalized left ventricular systolic and diastolic function by steady state free precession cardiovascular magnetic resonance. *J Cardiovasc Magn Reson* 2006;**8**:417–426.
9. Motwani M, Kidambi A, Herzog BA, Uddin A, Greenwood JP, Plein S. MR imaging of cardiac tumors and masses: a review of methods and clinical applications. *Radiology* 2013;**268**:26–43.
10. Las Casas AAJ, Las Casas AA, Borges MAF, Melo-Souza SE. Rivaroxaban for treatment of intraventricular thrombus in Chagas disease. *J Cardiol Cases* 2016;**13**:75–77.
11. Nonaka D, Takase H, Machii M, Ohno K. Intraventricular thrombus and severe mitral regurgitation in the acute phase of takotsubo cardiomyopathy: two case reports. *J Med Case Rep* 2019;**13**:152.
12. Fu W, Wang F, Han J, Li Z, Guo L, Gao W. Recurrent intraventricular thrombus following myocardial infarction. *Int J Cardiol* 2015;**181**:315–316.
13. Charif F, Mansour MJ, Hamdan R, Najjar C, Nassar P, Issa M et al. Free-floating right heart thrombus with acute massive pulmonary embolism: a case report and review of the literature. *J Cardiovasc Echogr* 2018;**28**:146–149.
14. Dipasco PJ, Misra S, Koniaris LG, Moffat FLJ. Thrombophilic state in cancer, part I: biology, incidence, and risk factors. *J Surg Oncol* 2011;**104**:316–322.
15. Barkhausen J, Hunold P, Eggebrecht H, Schüler WO, Sabin GV, Erbel R et al. Detection and characterization of intracardiac thrombi on MR imaging. *AJR Am J Roentgenol* 2002;**179**:1539–1544.