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REVIEW ARTICLE

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Right heart thrombi (RHT) and clot in transit with concomitant PE management: Approach and considerations

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Abstract

Right heart thrombi (RHT) continues to pose a clinical dilemma for multiple specialties and is especially concerning when present with concomitant pulmonary embolism (PE). Patients with PE and RHT are at an increased risk of poor outcomes compared to PE without RHT. Although the exact incidence of RHT is unknown, the increasing use of point-of-care ultrasound may lead to an increased detection and frequency of RHT. There are multiple treatment strategies available for RHT, including anticoagulation, systemic thrombolysis, and endovascular and surgical therapies. Given that these treatment strategies involve multiple medical specialties, the management of RHT with concomitant PE can be complex. Currently, there is limited clinical data and guidelines on the treatment and management of RHT. We aim to provide a review on RHT with concomitant PE, including risk stratification, treatment considerations, and our approach to the management of RHT.

KEYWORDS

pulmonary embolism (PE), right heart thrombi (RHT), thrombectomy, thrombolytic therapy

INTRODUCTION

Right heart thrombi (RHT) are defined as any thrombi visualized in the right atrium (RA), right ventricle (RV) or impending thrombi in the RA such as proximal inferior vena cava (IVC). RHT includes mobile thrombi, immobile thrombi, and thrombi adherent to pacemakers/leads and central venous catheters. RHT that is mobile (also known as clot in transit or RHT in transit) is commonly associated with concomitant acute pulmonary embolism (PE) and should raise suspicion for acute PE if not diagnosed already. The exact incidence of RHT is

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unknown (estimated to be around 2%–5% of all PE cases) but with possible greater incidence in patients with more severe PE.[1] Detection of RHT may be increasing, given the use of point-of-care ultrasound and formal transthoracic echocardiography (TTE) to help diagnose and risk stratify patients with PE. Patients with RHT and concomitant PE may be at an increased risk for poor outcomes including higher rates of mortality compared to patients without RHT.[1,2] Given the frequency and poor outcomes associated with RHT, there is a need for a framework and guidance to approach RHT. There are currently no society or expert guidelines and limited data on how to manage RHT. The aim of this article is to provide a framework of the understanding of RHT, review potential treatment options and provide guidance on the approach of RHT with concomitant PE.

PATHOPHYSIOLOGY

RHT may develop secondary to embolism/propagation of deep vein thrombosis (DVT), low-flow states (atrial fibrillation, severe RV dysfunction, or pulmonary hypertension), hypercoagulable states or from primary cardiac tumors or metastatic tumors such as renal cell carcinoma.[3] In addition, patients with mechanical valves, pacemakers, central-venous catheters and endocarditis are at an increased risk for RHT.[4] The treatment and prevention of RHT associated with mechanical valves, pacemakers, and central venous catheters is beyond the scope of this study; however, future recommendations on this topic are needed, as there is currently no formal guidelines addressing this issue. Previous studies have found that patients with RHT and PE were more likely to have a higher prevalence of chronic heart disease, renal insufficiency, immobility, and were more likely to have a greater incidence of RV hypokinesis and increased cardiac biomarkers.[1,2] This may suggest that increased severity of PE is associated with the development of RHT, possibly from reduced RV function and low-flow through the RV resulting in flow stagnation and formation of RHT.

RHT may be seen on computed tomography angiograpy-PE (CTA-PE) scan as a filling defect in the IVC, RA, or RV; however, small filling defects may be difficult to appreciate on CTA-PE and careful attention should be given in assessing for RHT within the RV and RA. More commonly, RHT is diagnosed by TTE or transesophageal echocardiography (TEE). Appearance on echocardiography may help characterize RHT into three main type as illustrated in Figures 1 and 2.[5]

Type A: Thin, highly mobile, serpentine

Type B: Immobile, ovoid shape, broad based, may be adherent

Type C: Features of both Type A and Type B

In addition to CTA-PE and echocardiography, cardiac magnetic resonance imaging (CMR) can play a valuable role in further identifying and characterizing RHT.[6]

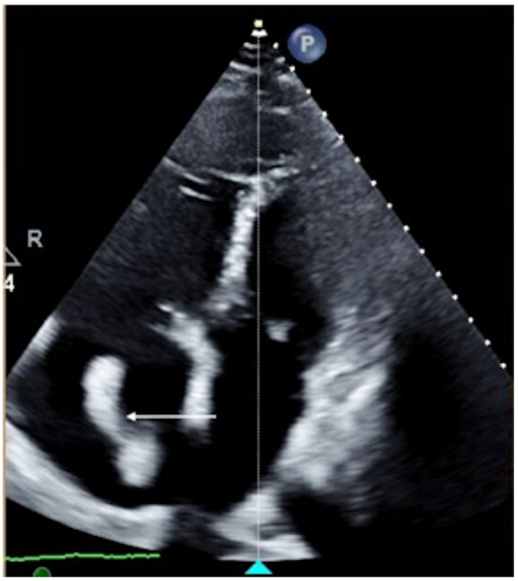

FIGURE 1 Transthoracic echocardiography with evidence of Type A or freely mobile (arrow) right heart thrombi (RHT), located within the cavity of the right atrium. This RHT is at high risk of embolization given that it is freely mobile.

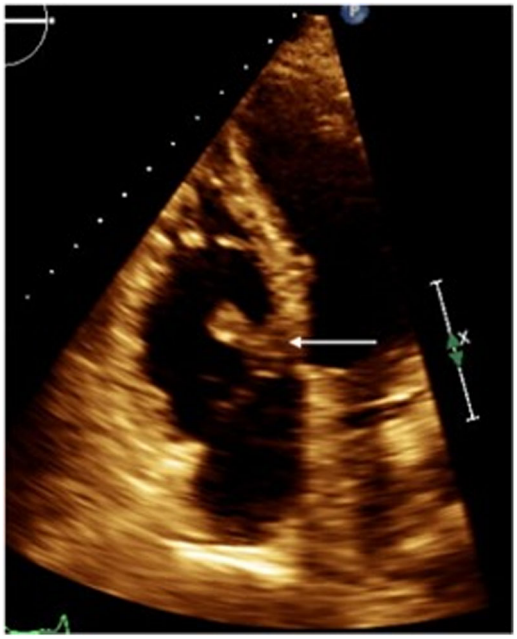

FIGURE 2 Transthoracic echocardiogram revealing the right heart thrombi with broad-based, adherent stalk (arrow) to the wall of the right ventricle, representative of Type B right heart thrombi.

By assessing early and late gadolinium levels CMR can help determine if the RHT is related to thrombus or an alternative diagnosis such as tumor or infective endocarditis.[7,8] CMR has been shown to be more sensitive and specific for LV thrombus when compared with TTE or TEE, this likely is true for RV thrombus; however, additional studies are required to confirm this.[9]

RHT may be acute, subacute, or chronic depending on the underlying etiology, and direct comparison with prior imaging can be helpful to determine if the RHT was previously present. Acute, mobile RHT may have a greater risk of embolization, whereas chronic, adherent-based clot may be less likely to embolize. The distinction between acute and chronic clot may play an important role in treatment as systemic thrombolytics are less likely to be effective against chronic organized clot. It is unknown if thrombi location increases risk of embolization such as thrombi in the RA versus RV.

TREATMENT

The optimal treatment for RHT with concomitant PE is unclear and ranges from anticoagulation alone to advanced therapies, which include systemic thrombolysis, endovascular therapies such as mechanical thrombectomy and surgical embolectomy. Limited studies have evaluated the optimal treatment with conflicting results. For example, Barrios et al.[2] reviewed patients with documented RHT in a large international patient registry and found no difference in all cause or PE-related mortality between patients treated with systemic thrombolysis versus patients treated with anticoagulation alone. In contrast to the above, several retrospective studies found that patients treated with anticoagulation alone appear to have higher rates of mortality compared to patients treated with systemic thrombolysis or surgical embolectomy.[1,10–12] Torbicki et al.[1] analyzed data from patients in the International Cooperative Pulmonary Embolism Registry and found that patients treated with heparin alone had a significantly increased mortality at 14 days. Athappan et al.[10] retrospectively reviewed RHT from 1992 to 2013 and compared efficacy of treatment, and found that mortality rates with anticoagulation alone were significantly higher compared to surgical embolectomy or systemic thrombolysis. Rose et al.[12] retrospectively reviewed RHT between 1966 and 2000, and also found that thrombolysis had a significantly lower mortality rate compared with anticoagulation alone. No large studies have compared the use of endovascular therapies to systemic thrombolysis or surgical embolectomy for treatment of RHT. Therapy for RHT is often based on local expertise and multiple patient specific factors including hemodynamic stability, presence of patent foramen ovale (PFO), location and characterization of RHT, and patient comorbid conditions.

Therapeutic options

Systemic thrombolysis

Alteplase (rt-PA, Activase, Genentech) is a common systemic thrombolytic agent used for the treatment of acute PE and can be considered for RHT. The optimal dose is unknown but ranges from full-dose 100 mg, to half dose 50 mg, administered over 2 h with or without a loading dose. Advantages of systemic thrombolysis include potential wider availability, rapidity of administration, and no need for additional expertise and specialized equipment required with endovascular or surgical approaches. There are however numerous relative and absolute contraindications to systemic thrombolysis listed in Table 1.[13–15] In addition, systemic thrombolysis may be less effective for chronic clot and have potential for significant bleeding.

Endovascular therapies

Triever catheter (Inari FlowTriever System, Inari Medical Inc.)

Endovascular procedure that can be performed in an angiography suite. The FlowTriever suction cannula is guided by using imaging such as TEE or intracardiac echocardiography (ICE). If available, imaging with ICE is an attractive option, as it negates the need for TEE, which may carry hemodynamic consequences in patients with RV dysfunction. The Triever Catheter comes in either 20Fr or 24Fr, and both are flexible catheters making them suitable for IVC, RA, and RV RHT. In addition, it can also be used to treat concomitant acute PE. There is no reinfusion cannula required, but there may be procedural blood loss associated with suction/aspiration. To minimize procedural blood loss, Inari recently released the FlowSaver device that allows aspirated blood from the procedure to be reinfused into the patient. This device may not be available at all centers and careful attention to volume status and procedural blood loss must be maintained. Patients must be able to tolerate anticoagulation as the procedure does require a therapeutic activated clotting time (ACT) to be completed. The

TABLE 1 Absolute and relative contraindications to systemic thrombolysis, adopted from Rivera-Lebron et al.[15]

Absolute contraindications to systemic thrombolysis	Relative contraindications to systemic thrombolysis
Active bleeding	Age > 75 years
Prior intracranial hemorrhage	Total body weight < 60 kg
Ischemic stroke within 3 months	Known bleeding diathesis or acquired coagulopathy
Suspected or confirmed aortic dissection	Platelet count < 100,000
Recent brain or spinal surgery	Coagulopathy (INR > 1.7)
Recent head or facial trauma	Uncontrolled hypertension (SBP > 180 mmHg/ DBP > 110 mmHg)
Intracranial neoplasm, vascular malformation, aneurysm, or any other structural brain disease	Recent significant non-intracranial bleeding (within 1 month)
	Recent major surgery, invasive procedure, and/or trauma (within 1 month)
	Current pregnancy or childbirth (within 1 week)
	History of remote ischemic stroke (>3 months)

Abbreviations: DBP, diastolic blood pressure; INR, international normalized ratio; PE, pulmonary embolism; SBP, systolic blood pressure.

Inari FlowTriever System recently received US Food and Drug Administration (FDA) approval for use of RHT in addition to already having FDA approval for treatment of PE and DVT.

AngioVac system (AngioDynamics Inc.)

Endovascular procedure that requires veno-venous extracorporeal bypass circuit. Traditionally, AngioVac was performed with guidance of TEE; however, there are increasing case reports of using ICE rather than TEE to perform the procedure. General anesthesia and a perfusionist is needed to help with the setup and operation of the device as the system requires the insertion of both a suction and reinfusion cannula to complete the veno-venous bypass circuit. The AngioVac cannula is rigid and there may be difficulty guiding the cannula into the RV and pulmonary artery, as well as the potential for tricuspid valve injury. It is therefore preferred that AngioVac be used for treatment of thrombi isolated/localized to the IVC and RA. The device can be spliced into an extracorporeal membrane oxygenation (ECMO) circuit allowing for treatment of thrombi related to the ECMO cannula, which are usually positioned near the c junction. AngioVac access sheath is 24Fr with a 22Fr inflow cannula and the reinfusion cannula is 18Fr. Patients must be able to tolerate anticoagulation as the procedure does require a therapeutic ACT to be completed. In addition, Angiodynamics has also created the AlphaVac System. This is a percutaneous aspiration device that is 22Fr in size and does not require a reinfusion cannula, allowing for faster setup compared with the AngioVac System, but may be associated with procedural blood loss during aspiration attempts. It can be used as an alternative percutaneous device for RHT, but similar to the AngioVac System, should be limited to the IVC and right heart. A comparison between the FlowTriever and AngioVac system can be seen in Table 2.

TABLE 2 Comparison of FlowTriever and AngioVac

	FlowTriever	AngioVac
Catheter size	T20 or T24 (20Fr or 24Fr)	24Fr access sheath 22Fr catheter
Return cannula required	No	Yes (18Fr)
VV ECMO circuit	No	Yes
General anesthesia	Optional	Yes
Perfusionist	No	Yes
TEE	No	Historically
ICE	Yes	Yes
Anticoagulation	Yes	Yes
Access location	Fem	IJ or Fem
Clot location	IVC, RA, RV, PA	IVC, RA

Abbreviations: ICE, intracardiac echo; IVC, inferior vena cava; PA, pulmonary artery; RA, right atrium; RV, right ventricle; TEE, transesophageal echocardiography; VV ECMO, venovenous extracorporeal membrane oxygenation.

Indigo Aspiration System (Penumbra)

Endovascular procedure currently FDA approved for use in the peripheral vasculature system and pulmonary arteries. The device features a smaller cannular size (12Fr or 8Fr) compared with the previous mentioned endovascular therapies. It does not require a reinfusion cannula and there may be associated procedural blood loss with aspiration. There are emerging case reports of the use of the Indigo Aspiration System for RHT; however, it is currently not FDA approved the use of RHT.

Surgical thromboembolectomy

Definitive therapy for RHT but most invasive. Patients must be appropriate surgical candidates without significant comorbid conditions. This may be best suited for cases where there is planned surgical pulmonary embolectomy, an open PFO, RHT trapped in a PFO (impending paradoxical embolism), prior PFO closure devices, or concern for cardiac tumors. It is also the optimal treatment for cases with chronic thrombi interfering with the tricuspid valve, where a concomitant tricuspid valve repair may be indicated. In certain cases where the clot is adherent to the IVC wall, deep hypothermia and a brief period of circulatory arrest may be required for complete clot removal.

Treatment guidelines for RHT/clot in transit with concomitant PE: Our approach

Given the lack of guidelines and studies on treatment of RHT with and without concomitant PE, we have developed a treatment pathway that is used at our institution as illustrated in Figure 3. We have the ability to provide a variety of treatment modalities including anticoagulation alone, systemic thrombolysis, endovascular approaches with FlowTriever and AngioVac, surgical embolectomy, and mechanical support with venoarterial extracorporeal membrane oxygenation (VA-ECMO). At our institution patients with a diagnosis of acute PE and concomitant RHT, trigger an immediate Pulmonary Embolism Response Team (PERT) activation, as the use of PERTs for treatment of PE may be associated with improved outcomes and decreased mortality.[16] A formal TTE is completed by a trained echo-sonographer and interpreted as soon as possible to confirm the diagnosis, characterize the RHT, and help

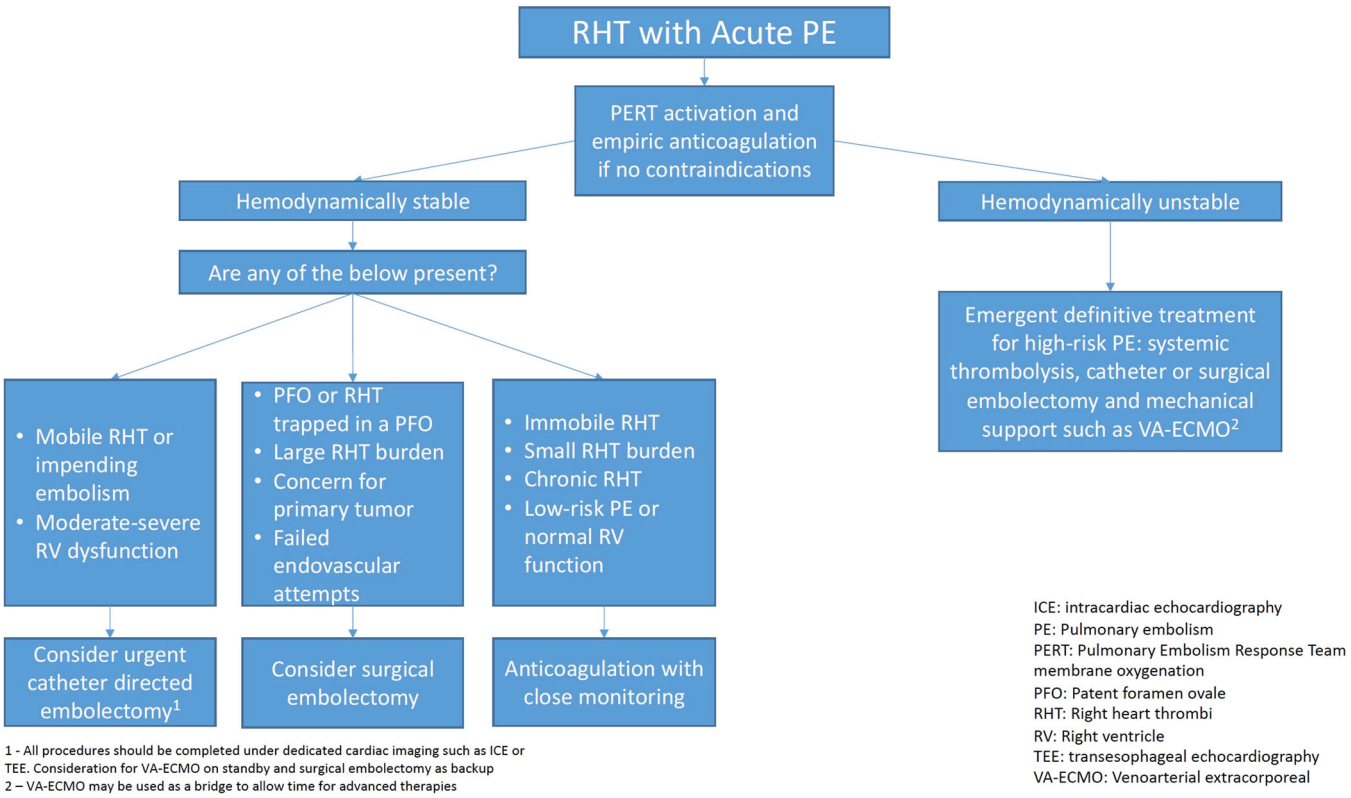

FIGURE 3 Current pathway for right heart thrombi with concomitant pulmonary embolism (PE) at Yale our local institution.

guide therapy. If RHT is diagnosed incidentally via echocardiography, we obtain a CTA-PE study to determine whether there is concomitant PE and to assess the embolic burden and location in the pulmonary vasculature. For patients with chronic clot, equivocal findings on TTE, CT, or with concerns of potential tumor, we recommend CMR for additional evaluation. For RHT that may be secondary to tumor (Type B clot), we recommend considering additional imaging of the abdomen and pelvis to rule out intra-abdominal or genitourinary malignancy. In addition, lower extremity ultrasound can help determine if there is DVT. The presence of DVT may lead one to believe that the RHT is related to thrombus rather than alternative differentials (tumor or infective endocarditis), it may also serve as a risk factor for additional embolization and further RHT.

We recommend all cases of RHT and concomitant PE to be discussed as a multidisciplinary team consisting of PERT members such as Pulmonary Critical Care, Interventionalist, Cardiothoracic Surgery, and Cardiology. Representation from specialists in endovascular therapies and surgical approaches are important for RHT given the potential use of advanced therapies. All recovered RHT samples should be sent to pathology for review to help assess etiology of RHT and to rule out primary cardiac tumors or metastatic disease.

Current guidelines do not take into account RHT as part of risk stratification for acute PE. We therefore continue to use the European Society of Cardiology Clinical Practice Guidelines on Acute PE for risk stratification and consider concomitant RHT as a marker of more severe disease and have a lower threshold to consider advanced therapies in addition to AC. Exceptions to this are for patients with very small RHT, small PE, and preserved RV function (low-risk or intermediate-low risk patients with concomitant small RHT), patients with limited life expectancy, and patients unable to tolerate anticoagulation. We consider patients who have underlying RV dysfunction and concomitant RHT to be at risk for transitioning to massive/high-risk PE with hemodynamic compromise given that additional embolization can overwhelm an already strained RV. Careful attention to the characteristics of the RHT can help determine the urgency and aggressiveness of treatment; however, there are currently no studies or predictive tools to assess when or if a RHT may embolize. Given the limited data, we feel that thrombus that is completely mobile (Type A) or prolapsing through a valve with each cardiac cycle, is at a high risk for embolization and could lead to potential abrupt RV decompensation. For thrombus that is adherent, examining the "stalk" of the thrombus could help determine the risk of embolization. For example, large thrombus adherent to the wall of the RA/ventricle via a small or thin "stalk" may be at higher risk of embolizing compared with thrombus with a broad-based attachment to the RA/ventricle. Additional studies need to be completed to determine if certain characteristics of RHT such as size, location, and mobility can predict risk of embolization.

Given the higher rates of mortality in RHT with concomitant PE treated with anticoagulation alone, we favor advanced therapies in addition to anticoagulation[10,11] for select patients. Advanced therapies for RHT with concomitant PE include systemic thrombolysis, endovascular approaches, or surgical approaches. Prior clinical trials comparing systemic thrombolysis versus surgical versus endovascular therapies for RHT and PE are currently limited. However, from the advanced therapies currently available, we tend to favor endovascular approaches for hemodynamically stable patients given the favorable safety profile, minimally invasive technique, and fewer contraindications compared with systemic thrombolytics or surgical approaches. AngioVac and FlowTriever are the two currently FDA-approved endovascular devices for RHT; however, at this time there are no studies comparing the two devices. We perform all endovascular cases for RHT with real-time imaging guidance such as ICE or TEE, to help assess RHT and guide catheter placement as illustrated in Figure 4. We prefer ICE compared with TEE given the potential need for increased use of sedation or possible need of intubation to facilitate TEE, which may impair hemodynamics in patients with underlying RV dysfunction. FlowTriever has a large catheter size (24Fr), which is flexible allowing easy entry into the RV and pulmonary arteries. There is also no requirement of a reinfusion cannula, bypass circuit, TEE, general anesthesia, or perfusionist. In addition, if there is concomitant PE, FlowTriever can also be used to perform catheter directed pulmonary embolectomy simultaneously. For cases of both RHT and PE treated with FlowTriever simultaneously, careful attention to volume status and blood loss with ongoing suction and aspiration is crucial as this may affect hemodynamics. Blood loss may be minimized by using the FlowSaver device that allows aspirated blood to be reinfused into the patient. AngioVac is an alternative method of treatment for RHT depending on the location and characteristics of the clot. We generally reserve AngioVac for large IVC and RA clot without concomitant PE given the rigid catheter and difficulty with maneuvering the catheter into the RV and pulmonary vasculature. Given its ability to be spliced in to the ECMO circuit we also consider the use of AngioVac for patients who develop thrombi associated with ECMO. AngioVac is more invasive than FlowTriever in the sense that it requires a reinfusion cannula to be

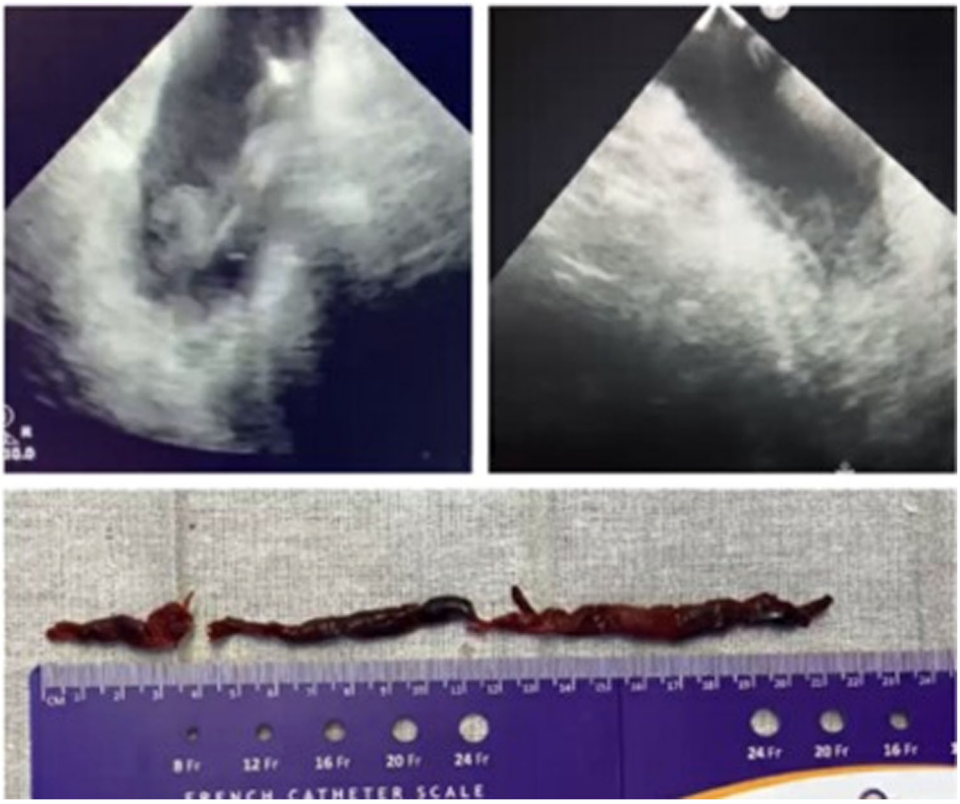

FIGURE 4 Use of the Inari FlowTriever under guidance of intracardiac echocardiography (ICE) for treatment of the right heart thrombi. Top left: ICE images revealing large right heart thrombi. Top right: ICE images post FlowTriever embolectomy revealing resolution of right heart thrombi. Bottom: Thrombus extracted during FlowTriever embolectomy.

inserted for the bypass circuit and requires a perfusionist for set-up and operation. AngioVac historically required a TEE and general anesthesia to complete the procedure, which may precipitate hemodynamic compromise in patients with underlying RV dysfunction, although there are increasing case reports of using AngioVac with ICE.

Given the risk of potential embolization of RHT and hemodynamic compromise with the use of endovascular therapies, a plan for rescue therapy should be discussed before the procedure. This may include the use of mechanical circulatory support in the form of VA-ECMO, vasopressors readily available, a plan for general anesthesia and the potential use of systemic thrombolysis or converting to a surgical approach. In particularly high-risk cases, VA-ECMO should be considered before the procedure or the empiric placement of wires and sheaths for cannulation to VA-ECMO before the procedure to help expedite the time it takes to place on VA-ECMO in the event of hemodynamic compromise.

For patients who have a known PFO, RHT trapped across a PFO (impending paradoxical embolism), prior PFO closure devices, or echocardiographic features concerning for a primary cardiac tumor, a surgical approach is favored if the patient is an appropriate surgical candidate.[7] A surgical approach may also be considered for large RHT burden, or previously failed endovascular attempts.

For patients who are hemodynamically unstable (high-risk/massive PE), both hemodynamics and the RHT should be treated. This is considered a medical emergency and expedited treatment is mandatory. There is limited data on what the optimal treatment is for high-risk/massive PE with concomitant RHT but options include systemic thrombolysis and endovascular and surgical approaches. Full-dose systemic thrombolysis could be considered for patients without relative or absolute contraindications, as the therapy can be administered quickly. If there are relative or absolute contraindications to systemic thrombolysis or for patients who do not improve with systemic thrombolysis, urgent evaluation for endovascular or surgical approaches could be considered. In addition, VA-ECMO could be considered as a bridge to allow for controlled definitive therapy (endovascular or surgical approach) to take place. These patients can present in shock and ECMO is a very effective modality to reverse the shock to allow for time to further evaluate additional treatment options. Femoral vein–femoral artery configuration can allow for

fairly quick cannulation. Patients with a large amount of clot in the IVC may complicate the decision to insert a femoral venous cannula and an alternative venous site may need to be selected. FlowTriever has been used in case reports for massive PE and the FlowTriever for Acute Massive PE trial (clinicaltrials.gov NCT04795167) may provide additional data for this patient cohort and allow for the device to be considered for high-risk/massive PE cases if the procedure can be completed in a timely fashion. In centers with expertise in surgical pulmonary embolectomy, surgical approaches could be considered rather than systemic thrombolysis or in addition to systemic thrombolysis if there is no improvement. In centers that do not have endovascular or surgical options available, urgent consultation and transfer to a tertiary center should be considered. In this scenario, potential treatment with systemic thrombolysis may be the only treatment option available to temporize hemodynamics or prevent hemodynamic collapse. Alternatively, a mobile ECMO unit could be deployed to retrieve the patient and transfer to a tertiary center on ECMO if possible. These transfers are high risk and require significant coordination and planning between the transferring hospital, receiving hospital and transport team to be completed safely.

CONCLUSION

RHT is associated with significant morbidity and mortality The optimal management strategy for RHT remains uncertain. Future research is needed to help guide treatment decisions around RHT and concomitant PE given the limited current evidence regarding treatment, the high incidence of RHT, and associated increase in mortality. With our current understanding and treatment options for RHT, we have created a framework for hospitals and PERTs to treat RHT with concomitant PE. We believe rapid consultation with a PERT should be considered for treatment of RHT and concomitant PE given the complexity of treatment and potential need for advanced resources.

Recent studies have revealed that there may be an association with better outcomes (lower cost, length of stay, readmission rates, and mortality) for patients with PE who are treated at high-volume centers.[17,18] This leads us to believe that similar outcomes for RHT are likely to be improved if patients are treated at more experienced, higher-volume centers, and transfer to a high-volume center with expertise of RHT should be considered. It is advantageous to have experience with multiple treatment modalities across different specialties, an understanding of what treatment options are available at an institution and how to transfer these high-risk patients to a higher level of care if needed.

Future research is needed to better understand the treatment and management of RHT especially with emerging endovascular therapies. A randomized control trial assessing the different therapeutic options for RHT would likely be difficult to complete given the heterogenous patient population and variation in care across treatment centers. Alternatively, a cohort study using a large registry of RHT and/or PE patients could potentially allow for a variety of therapies to be compared across different patient populations and health centers to assess important outcomes such as mortality, hospital and ICU length of stay, complications from treatment (systemic thrombolysis, and surgical and endovascular complications), hemodynamic decompensation and complications of RHT. This could be accomplished by reviewing data from ongoing PE registries such as the National PERT Consortium registry or the Registro Informatizado de Enfermedad TromboEmbólica (RIETE Registry).

AUTHOR CONTRIBUTIONS

All authors contributed to the conception and design of the work, critical revision of key intellectual content and approval of the final version to be published.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

ETHICS STATEMENT

Research ethics board's approval not required due to nature of the analysis.

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REFERENCES

1. Torbicki A, Galié N, Covezzoli A, Rossi E, De Rosa M, Goldhaber SZ, ICOPER Study G. Right heart thrombi in pulmonary embolism: results from the International Cooperative Pulmonary Embolism Registry. J Am Coll Cardiol. 2003;41(12):2245–51.
2. Barrios D, Rosa-Salazar V, Jiménez D, Morillo R, Muriel A, Del Toro J, López-Jiménez L, Farge-Bancel D, Yusen R, Monreal M, RIETE i. Right heart thrombi in pulmonary embolism. Eur Respir J. 2016;48(5):1377–85.
3. Kukla P, McIntyre WF, Koracevic G, Kutlesic-Kurtovic D, Fijorek K, Atanaskovic V, Krupa E, Mirek-Bryniarska E, Jastrzębski M, Bryniarski L, Pruszczyk P, Baranchuk A. Relation of atrial fibrillation and right-sided cardiac thrombus to outcomes in patients with acute pulmonary embolism. Am J Cardiol. 2015;115(6):825–30.
4. Benjamin MM, Afzal A, Chamogeorgakis T, Feghali GA. Right atrial thrombus and its causes, complications, and therapy. Proceedings. 2017;30(1):54–6.
5. Momose T, Morita T, Misawa T. Percutaneous treatment of a free-floating thrombus in the right atrium of a patient with pulmonary

embolism and acute myocarditis. Cardiovasc Interv Ther. 2013; 28(2):188–92.
6. Plodkowski AJ, Chan A, Gupta D, Lakhman Y, Kukar N, Kim J, Perez-Johnston R, Ginsberg MS, Steingart RM, Weinsaft JW. Diagnostic utility and clinical implication of late gadolinium enhancement cardiac magnetic resonance for detection of catheter associated right atrial thrombus. Clin Imaging. 2020;62:17–22.
7. Barkhausen J, Hunold P, Eggebrecht H, Schüler WO, Sabin GV, Erbel R, Debatin JF. Detection and characterization of intracardiac thrombi on MR imaging. Am J Roentgenol. 2002;179(6):1539–44.
8. El Ouazzani J, Jandou I, Thuaire IC. Thrombus or vegetation? Importance of cardiac MRI as a diagnostic tool based on case report and literature review. Ann Med Surg. 2020;60:690–4.
9. Srichai MB, Junor C, Rodriguez LL, Stillman AE, Grimm RA, Lieber ML, Weaver JA, Smedira NG, White RD. 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. 2006;152(1):75–84.
10. Athappan G, Sengodan P, Chacko P, Gandhi S. Comparative efficacy of different modalities for treatment of right heart thrombi in transit: a pooled analysis. Vasc Med. 2015;20(2):131–8.
11. Burgos LM, Costabel JP, Galizia Brito V, Sigal A, Maymo D, Iribarren A, Trivi M. Floating right heart thrombi: a pooled analysis of cases reported over the past 10years. Am J Emerg Med. 2018;36(6):911–5.
12. Rose PS, Punjabi NM, Pearse DB. Treatment of right heart thromboemboli. Chest. 2002;121(3):806–14.
13. Kearon C, Akl EA, Ornelas J, Blaivas A, Jimenez D, Bounameaux H, Huisman M, King CS, Morris TA, Sood N, Stevens SM, Vintch J, Wells P, Woller SC, Moores L. Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report. Chest. 2016;149(2):315–52.
14. Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galiè N, Gibbs JS, Huisman MV, Humbert M, Kucher N, Lang I, Lankeit M, Lekakis J, Maack C, Mayer E, Meneveau N, Perrier A, Pruszczyk P, Rasmussen LH, Schindler TH, Svitil P, Vonk Noordegraaf A, Zamorano JL, Zompatori M, Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2014; 35(43):3033–69.
15. Rivera-Lebron B, McDaniel M, Ahrar K, Alrifai A, Dudzinski DM, Fanola C, Blais D, Janicke D, Melamed R, Mohrien K, Rozycki E, Ross CB, Klein AJ, Rali P, Teman NR, Yarboro L, Ichinose E, Sharma AM, Bartos JA, Elder M, Keeling B, Palevsky H, Naydenov S, Sen P, Amoroso N, Rodriguez-Lopez JM, Davis GA, Rosovsky R, Rosenfield K, Kabrhel C, Horowitz J, Giri JS, Tapson V, Channick R, PERT C. Diagnosis, treatment and follow up of acute pulmonary embolism: consensus practice from the PERT Consortium. Clin Appl Thromb Hemost. 2019;25: 1076029619853037.
16. Chaudhury P, Gadre SK, Schneider E, Renapurkar RD, Gomes M, Haddadin I, Heresi GA, Tong MZ, Bartholomew JR. Impact of multidisciplinary pulmonary embolism response team availability on management and outcomes. Am J Cardiol. 2019;124(9):1465–9.
17. Kobayashi T, Young MN, Giri J. Volume, outcomes, and 'Centers of Excellence' for pulmonary embolism care. Vasc Med. 2021;26(1):47–9.
18. Finkelstein M, Cedillo MA, Kestenbaum DC, Shoaib OS, Fischman AM, Lookstein RA. Relationship of hospital volume on outcomes in patients with acute pulmonary embolism: analysis of a 70,000 patient database. Vasc Med. 2021;26(1):38–46.

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