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Right Atrial Thrombi, the Management Conundrum: 2 Case Reports

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Manuscript Preparation E
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Case series
Patients: Male, 62-year-old • Male, 66-year-old
Final Diagnosis: Right atrial floating thrombus • right heart thrombus-in-transit
Symptoms: Shortness of breath
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual clinical course

Background: There are no guidelines providing an algorithmic approach for the management of right atrial thrombi, to date, owing to a lack of strong supporting studies. In this case series, we describe 2 cases of high-risk patients with massive right atrial thrombi who had different outcomes.

Case Reports: Case 1: A 62-year-old man with ischemic cardiomyopathy and atrial fibrillation, who was on a permanent pacemaker for sick sinus syndrome and was noncompliant with medication for 2 years, presented to the Emergency Department for evaluation of a 2-month history of progressive shortness of breath and swollen neck veins. A cardiac ultrasound confirmed a large right atrial thrombus, and a computed tomography (CT) pulmonary angiogram was negative for pulmonary emboli. He was managed with a heparin infusion and thrombolytic therapy with favorable evolution.

Case 2: A 66-year-old man, with a past medical history of nonischemic cardiomyopathy, atrial fibrillation, deep venous thrombosis, and pulmonary emboli a year earlier, presented to an urgent care unit with sudden onset of shortness of breath. A cardiac ultrasound confirmed a large right atrial thrombus, and a CT pulmonary angiogram confirmed bilateral pulmonary emboli. He was managed with a heparin infusion and EkoSonic endovascular system therapy. He subsequently needed venoarterial extracorporeal membrane oxygenation for cardiopulmonary resuscitation and underwent mechanical aspiration thrombectomy. The patient's evolution was unfavorable.

Conclusions: In the absence of an evidence-based guideline to approach right atrial thrombi, management should be individualized for each patient, based on the type of thrombi, hemodynamic status, and presence or absence of associated pulmonary emboli.

Keywords: Case Management • Heart Atria • Thrombosis

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/933427

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Background

Right heart thrombosis is a rare and life-threatening condition, with an almost 100% mortality rate if not treated [1,2]. The European Working Group on Echocardiography classified right atrial thrombi (RAT) into 3 groups; type A, RAT in transit, which are typically large clots that are free floating, originating from the deep venous system; type B, RAT in situ, typically small clots attaching to the atrial wall or intracardiac device; and type C, mobile in situ thrombi, which have a stalk and a thin point of attachment to the atrial wall, similar to atrial myxoma [3]. The risk of embolization and prognosis varies by type [3]. Management of RAT decreases mortality by at least 3- to 4-fold, depending on the treatment option. Treatment options for RAT include anticoagulation, thrombolytic therapy, and surgical thrombectomy/embolectomy with vacuum extraction [4,5]. Previous meta-analyses on different treatment modalities thus far have had differing recommendations, with thrombolytic and surgical options reported to have better outcomes. However, there is no definitive algorithm to follow in the management of RAT [4-6]. Moreover, most of the reported cases were of patients with RAT in transit and RAT in situ. In this case series, we describe 2 high-risk patients: the first with type C massive right atrial thrombus that was successfully managed with tissue plasminogen activator (tPA) therapy, and the second with type A that was unsuccessfully managed with vacuum extraction.

Case Reports

Case 1

A 62-year-old man presented with a past medical history of several comorbidities, including hypertension, diabetes mellitus, ischemic cardiomyopathy, atrial fibrillation on anticoagulation therapy, and a permanent pacemaker for sick sinus syndrome in 2016 (4 years earlier). He was lost to follow-up since 2018 and was noncompliant with his medication owing to a lack of insurance and an inability to pay. He presented to the Emergency Department (ED) for evaluation of shortness of breath. He reported progressive shortness of breath of a 2-month duration. The shortness of breath was initially mild and exertional. More recently, however, he became short of breath at rest, and it was worse on lying down and relieved when sitting up. He also had concerns of distended neck veins and a swollen neck and upper limbs, and reported a weight loss of 13.6 to 18.1 kg, accompanied by a decreased appetite over the last 2 months. He denied having fever, cough, chest pain, palpitations, nausea, vomiting, change in bowel habit, and urinary symptoms. In the ED, he was hemodynamically stable with a blood pressure of 123/58 mmHg, heart rate of 71 beats per min, respiratory rate of 18 breaths per min, temperature of

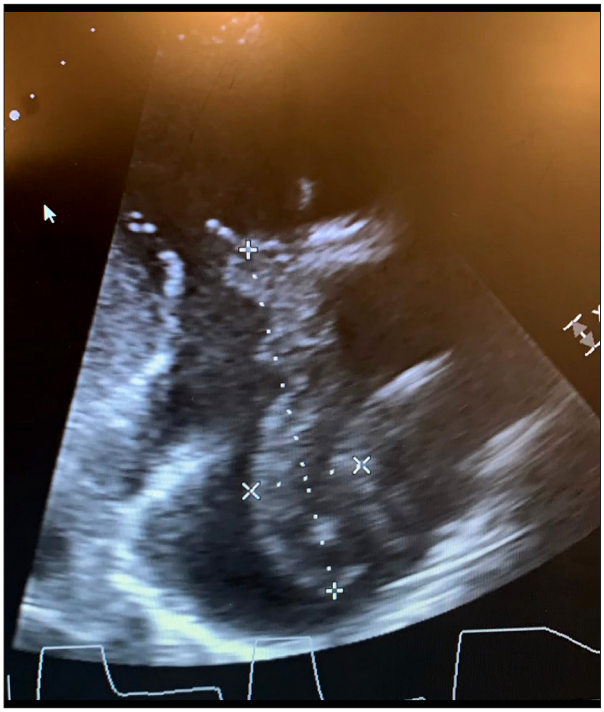

Figure 1. Case 1: Cardiac echography showing large right atrial thrombi.

36.3°C, and oxygen saturation of 98% on room air. He had distended neck veins and distended veins in the upper and lower extremities. A bedside cardiac echography performed in the ED revealed a mass in the right atrium. An official echocardiography confirmed a multi-lobular mass of 8.5×2.8 cm in the right atrium, consistent with fresh thrombi/vegetation (**Figure 1**). The mass seemed to be attached to the pacemaker wire and to be protruding through the tricuspid valve to the right ventricle in certain beats. A CT pulmonary angiogram showed a large thrombus in the right atrium but no evidence of pulmonary emboli. The patient was started on a heparin infusion. Then, the cardiology and cardiothoracic surgery teams were consulted, and the patient was admitted to the Cardiac Care Unit (CCU). His blood work was remarkable for a normocytic anemia of 7.5 g/dL. Based on the patient's severe anemia, for which internal bleeding could not be ruled out, thrombolytic therapy with tissue plasminogen activator (tPA) was not considered as an initial treatment option, and the patient was being planned for clot evacuation by a vacuum-based device (AngioVac). While in the hospital pending the AngioVac procedure, the patient developed tachypnea up to 40 breaths per min, tachycardia up to 110 beats per min, and hypoxia, with an oxygen saturation level of 88%. A bedside echocardiogram showed a thrombus in the right atrium that was more mobile than it had been in the morning. Due to the patient's high risk of embolization and clinical picture, the decision was made to administer tPA. A CT pulmonary angiogram confirmed segmental pulmonary emboli of the right middle lobe, and a bedside

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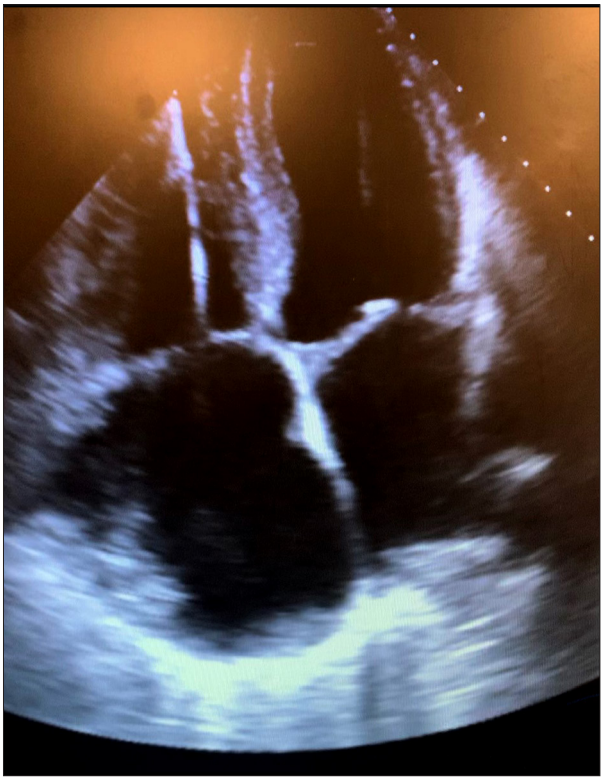

Figure 2. Case 1: Cardiac echography showing resolution of right atrial thrombi after thrombolytic therapy.

echocardiogram confirmed the complete dissolution of the atrial thrombus after the tPA (**Figure 2**). The heparin infusion was continued. A venous duplex scan of the upper and lower extremities subsequently performed was negative for thrombi. The evolution thereafter was favorable, and the patient was discharged with direct oral anticoagulants.

Case 2

A 66-year-old man with a past medical history of nonischemic cardiomyopathy, atrial fibrillation, and deep vein thrombosis/pulmonary emboli was referred to our facility for better management of a clot in his heart and lungs. He was walking his dog in the morning as usual and felt severely short of breath. He went to his urgent care facility and was told he had bronchitis; however, he recounted that he had had a pulmonary embolism a year ago and said that he knew the symptoms and felt he was having another clot. He was thus referred to a nearby hospital for further evaluation. At the ED, an echocardiogram and CT pulmonary angiogram showed a large clot in the right atrium and bilateral submassive pulmonary emboli. Given the risk of a massive RAT breaking off and obstructing flow through the pulmonary artery, the decision was made to not give tPA. A heparin infusion was started, and the patient was transferred to our facility for better management. On arrival at our facility, he had a blood pressure of 96/52 mmHg, heart rate of 112 irregular beats per min, respiratory rate of 23 breaths per min, temperature of 36.4°C, and 97% oxygen saturation on 4 L of oxygen by nasal canula. He was awake, alert, oriented, pleasant, appropriate, and anxious. There was no jugular venous distention. His heart sounds were irregularly irregular, with no murmurs. His electrocardiogram showed atrial fibrillation with a right bundle branch block and anterior fascicular block. Bilateral arterial and venous duplex scans of the upper and lower extremities were negative for thrombi. The patient was admitted for a hemodynamically significant acute-on-chronic massive pulmonary embolism with a significant clot in transit through the right atrium and underwent venoarterial extracorporeal membrane oxygenation (VA ECMO) cannulation. During the same procedure, the patient subsequently underwent AngioVAC mechanical aspiration to resolve the significant clot burden in transit in the right atrium. After the AngioVAC, 30% to 40% resolution of the clot was noted; however, there was a significant residual clot. He was then managed in the CCU on VA ECMO. The patient's course in the hospital remained uneventful, and, on hospital day 4, a subhepatic inferior vena cava filter was placed to allow for decannulation of the VA ECMO. Through day 1 to day 5 of hospitalization, an oxygenator change was required because of significant intravascular hemolysis, with white platelet plaques noted on the arterial side of the VA ECMO oxygenator, corroborated by cola-colored urine and elevated free hemoglobin. The patient subsequently developed acute kidney injury requiring continuous veno-venous hemofiltration initiation on day 5. With the above, the patient was considered for decannulation of the VA ECMO, as he was tolerating 1.5 L of support with the use of an intra-aortic balloon pump. After an elaborate multidisciplinary discussion on ECMO, consent was obtained from the next of kin for ECMO decannulation. ECMO decannulation was completed according to protocol; however, 15 min into decannulation, significant hypotension with worsening bradycardia was noted. The patient subsequently went into pulseless electrical activity cardiac arrest. All advanced cardiac life support measures were futile, and the patient was pronounced dead. A repeat acute-on-chronic pulmonary embolism with migration of the clot after VA ECMO removal was thought to be the cause of death.

Discussion

The present cases exemplify 2 types of RAT in high-risk patients, which were managed differently and had different treatment outcomes. RAT in transit is thought to be related to a dislodged clot from the deep venous system. Generally, the Case 2 patient is best described as the presence of large clots with a high risk of embolization. RAT in situ, or type B, is often in situ from structural or functional heart defects, such as atrial fibrillation and intracardiac devices. RAT in situ typically presents with a small immobile thrombus, with minimal

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risk for embolization. Our Case 1 patient had the above risk factors for RAT in situ; however, he had a large mobile clot attached to the pacemaker wire in the right atrium. The mobile in situ clot in this patient classified him as having type C RAT.

The guidelines suggest 3 treatment options for thromboembolic diseases. Several earlier reports on consecutive cases have suggested any 1 of the 3 treatment choices (anticoagulation, thrombolysis, or surgical embolectomy) can be effective in patients with RAT [1,7-10]. However, limitations of these reports are small sample sizes, selection bias, and a lack of control groups. In addition, the few meta-analyses reported to date have suggested different treatment recommendations [1-3]. Kinney and Wright, in an analysis of 119 patients from 1966 to 1989, demonstrated a slight increase in survival with anticoagulation (70%) when compared with thrombectomy or thrombolytic therapy (62%). However, in their group, not all patients had pulmonary emboli, and the representation of each therapy was unclear. The patients in the present report were started on unfractionated heparin therapy at presentation, prior to the development of pulmonary embolism in Case 1 and after the patient had been diagnosed with a pulmonary embolism in Case 2. It is unclear in Case 1 whether the development of pulmonary embolism in our patient following the initiation of unfractionated heparin was a natural progression of the massive mobile RAT or partial fractionation of the thrombus by heparin therapy. Previous authors reported that for all treatment options, prognosis was worse if pulmonary emboli were present.

Surgical management includes traditional open thrombectomy and percutaneous thrombectomy. Percutaneous aspiration thrombectomy through the AngioVac vacuum system is a novel and less invasive management option of RAT and is commonly more successful than other management options in select patients [11]. This innovation has also been utilized for the extraction of deep vein thrombi, including thrombi associated with inferior vena cava filters as well as vegetations, tumors, and foreign bodies [12,13]. The AngioVac system essentially functions as a veno-venous ECMO circuit connected to a thrombus filter and centrifugal pump. The aspirated thrombus is passed through a filter and trapped in a reservoir, then thrombus-free blood is returned to the body [13]. Our patient was a surgical-risk patient with severe anemia and was thus being considered for percutaneous aspiration; however, he became hemodynamically unstable prior to receiving thrombolytic therapy, which resolved the thrombi.

Thrombolytic therapy was suggested by Rose et al in a meta-analysis to be superior to anticoagulation and embolectomy in decreasing mortality [2]. The study noted anticoagulation by itself to be insufficient in the management of RAT. The study further suggested thrombolytic therapy was the preferred option in the absence of absolute or relative contraindications. Our patient in Case 1 ideally should have been treated with tPA at initial presentation; however, the patient was severely active with imminent active bleeding; thus, tPA was held at presentation and was administered only as a lifesaving treatment while the patient was being carefully monitored in the CCU. The option of thrombolytic therapy in our patient was also preferred because it can be administered quickly. Thrombolytic therapy is also advantageous because systemic thrombolytics can dissolve existing thrombi in various areas, including the pulmonary arteries, intracardiac chambers, and venous circulation. Three thrombolytic drugs have been approved by the FDA for severe pulmonary embolism, including urokinase, streptokinase, and alteplase; a fourth drug, tenecteplase, is currently under review [14]. Alteplase was given to our patient because of its benefits of accelerated thrombus lysis and pulmonary reperfusion, reduced pulmonary hypertension, and improved right ventricular function and overall cardiac function. Following alteplase administration, a repeat bedside echography showed total resolution of the clot.

Conclusions

There is an absence of guideline algorithms for the management of RAT; however, previous studies have shown thrombolysis, embolectomy, and anticoagulation to be effective in the treatment of patients with RAT types A and B. Our case series showed thrombolytic therapy to be equally useful in the acute management of type C RAT and vacuum aspiration to be suboptimal for the management of high-risk patients with type A RAT. Additional reports and randomized clinical trials are needed to substantiate these findings and those of previous reports.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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