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A Troika of Tricuspid Valve Thrombus, Bilateral Upper Extremity Deep Venous Thrombosis and Pulmonary Embolism

Farid Khan[1], Keyoor Patel[2], Muhammad Imran Ali[1*]

[1]Internal Medicine Residency Program, UHS Wilson Medical Center, Johnson City, NY, USA
[2]UHS Heart & Vascular Institute, Johnson City, NY, USA
Email: drfaridkhan@hotmail.com, Keyoor.Patel@nyuhs.org, *Muhammad.Ali@nyuhs.org

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Abstract

Background: Tricuspid valve thrombus with concomitant bilateral pulmonary embolism (PE) and right heart strain poses a significant risk of hemodynamic instability and increased mortality. **Case Report:** We report the unique case of a female who presented with dyspnea and tachycardia, and was subsequently found to have a structure attached to the tricuspid valve. Concomitantly, she also had bilateral upper extremity deep venous thrombosis (UEDVT) and bilateral sub-massive PE. Thorough clinical assessment, and diagnostic and risk stratification tools were applied to guide the management and disposition. Tricuspid valve thrombus resolved after unfractionated heparin therapy followed by oral anticoagulation as seen on repeat transthoracic echocardiography. We think the readership will benefit from our experience of managing an uncommon and critical clinical presentation of tricuspid valve thrombus in the setting of extensive venous thromboembolism. **Conclusion:** Careful clinical assessment, risk stratification tools, and close monitoring are needed to guide the management of tricuspid valve thrombus with concomitant bilateral PE and UEDVT.

Keywords

Pulmonary Embolism, Tricuspid Valve Thrombus, Deep Venous Thrombus, Heparin

1. Background

Tricuspid valve thrombus in the setting of upper extremity deep venous thrombosis (UEDVT) is an uncommon complication. It is usually associated with venous thromboembolism (VTE) due to hormonal therapies, structural heart

abnormalities, coagulative disorders, antiphospholipid syndrome, and catheter-associated thromboembolic causes [1] [2] [3] [4] [5].

Prevalence of right heart thrombi (RHT) varies from 4% to 18% in cases of massive pulmonary embolism (PE) [6] [7]. RHTs are either type A, highly mobile, originating from DVTs and caught-in-transit in the right chambers, or type B, less mobile, associated with structural abnormalities or atrial fibrillation [8]. Tricuspid valve thrombus (TVT) typically embolizes from the DVT of lower extremities or pelvic veins [9]. RHTs increase mortality up to 40% as compared to PE alone [8] [10]. The first 24 hours of the presentation are considered to be crucial due to the same reasons. [1] [11]. A rapid workup to establish a diagnosis and to exclude other causes of the mass attached to the tricuspid valve is required [12] [13].

We herein report the case of a patient with tricuspid valve thrombus presenting concomitantly with bilateral sub-massive PE and bilateral UEDVT.

2. Case Report

A 39-year-old female was admitted to our intensive care unit (ICU) after a near-syncopal episode with two days of dyspnea, pleuritic chest pain and palpitations. The patient did not have a fever or cough. She had a slight abdominal discomfort due to ascites secondary to malignancy. There were no recent travels, sick contacts, history of smoking or oral contraceptive use. In the past, she was treated with surgery and chemotherapy for ovarian cancer. Her family history was unremarkable for thromboembolic diseases.

On presentation, she was a febrile, tachycardiac and mildly hypoxic. Clinically, she was in moderate respiratory distress. Initial systolic blood pressures trended in the range of 95 - 105 mmHg lower than her usual baseline of 110 mmHg or more. Precordium examination revealed a rapid and regular heart rate with normal heart sounds and no jugular venous distension. Lungs were clear to auscultation with a non-tender chest wall. The abdomen was slightly distended and dull to percussion suggesting mild to moderate ascites. There was mild swelling and tenderness over the upper extremities while the lower extremities had no edema, erythema or tenderness.

Electrocardiogram revealed a heart rate of 145 beats/minute with nonspecific old T-wave inversions in leads II, III and AVF. Laboratory workup revealed leukocytosis, elevated C-reactive protein, mildly elevated troponin and a negative swab for COVID-19 (coronavirus disease 2019) (**Table 1**). Blood and sputum cultures were sent to microbiology. Computed tomography pulmonary angiogram

Table 1. Pertinent laboratory results with laboratory reference ranges.

Lab	Results	Ref. Range
Troponin I	0.423	<0.03
C-reactive protein	9	≤0.9 mg/dL
WBC	15.2	4.0 - 10.5 10*3/uL

(CTPA) revealed severe bilateral PE involving lobar arteries extending into the distal branches (Figure 1). Straightening of the ventricular septum was observed indicating right ventricular (RV) strain.

Doppler ultrasound of upper extremities revealed DVT in bilateral cephalic and basilic veins (Figure 2). A two-dimensional transthoracic echocardiography (TTE) revealed dilated RV with decreased systolic function and flattened septum. A large mobile echogenic structure [1.0 cm × 0.95 cm] was attached to the atrial side of the tricuspid valve anterior leaflet with moderate tricuspid regurgitation (Figure 3).

Based on the clinical presentation and evidence from the workup, our patient had acute unprovoked sub-massive PE with RV strain along with a structure entrapped on the tricuspid valve. She was initially treated in the ICU within travenous unfractionated heparin with a loading dose of 80 units/kg and continued on heparin infusion as well as oxygen therapy via nasal cannula. She remained hemodynamically stable with no further hypotensive or hypoxic episodes in the first 24 hours. Activated partial thromboplastin time goal of 60 - 100 was achieved within 12 hours of heparin administration.

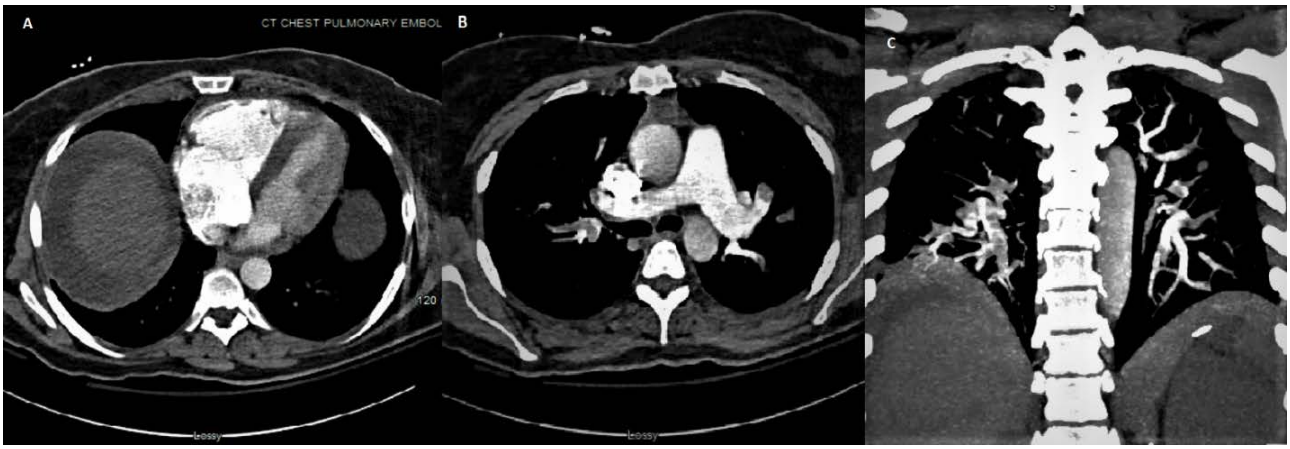

Figure 1. CT pulmonary angiogram reveals bilateral PE of lobar arteries (B) extending into distal branches (C) along with straightened interventricular septum indicating right ventricular strain (A).

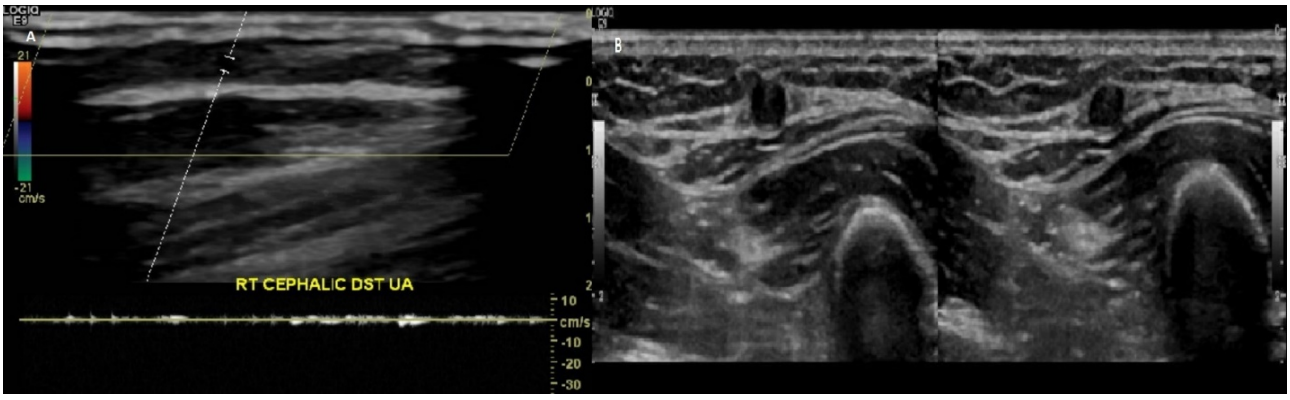

Figure 2. Doppler ultrasound of upper extremities showing thrombosis in right cephalic mid and distal arm (A) with dampened waveform, left cephalic vein mid are not compressible (B).

In view of clinical improvement, the patient was transferred to general medicine telemetry after one day in the ICU. On the 4th day of hospitalization, heparin was transitioned to oral apixaban 10 mg twice daily, and she was ambulated out of bed. The next day patient was weaned off of oxygen inhalation. Leukocytosis down trended, and blood and sputum revealed no growth in the final results. On the 7th day, she was discharged on oral anticoagulation. The mobile echogenic structure seen in the previous study was not appreciated in the repeat TTE in three weeks of discharge (Figure 4).

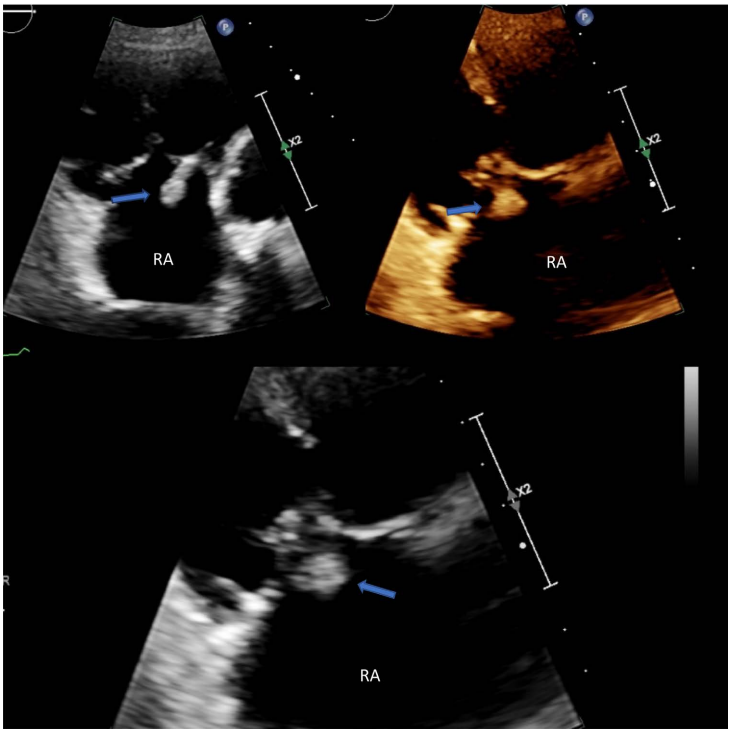

Figure 3. First transthoracic echocardiography view displays thrombus (arrows) entrapped on the atrial aspect of tricuspid valveanterior leaflet. RA: Right atrium.

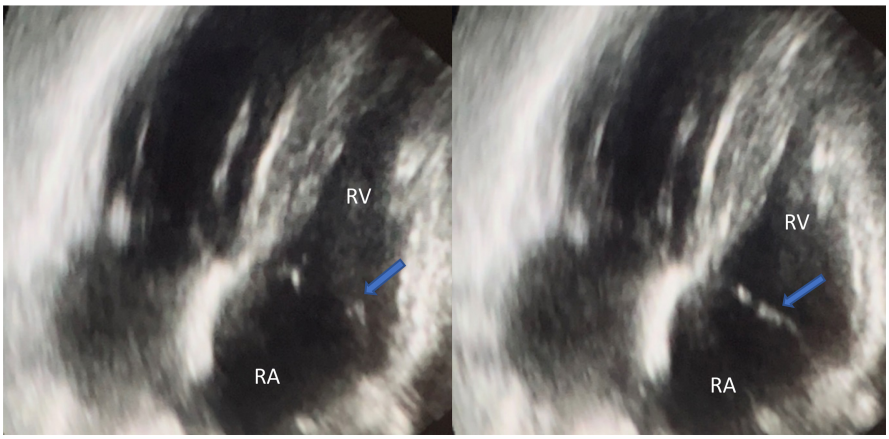

Figure 4. Repeat transthoracic echocardiography view displays tricuspid valves (arrows) with absence of the echogenic structureseen in Figure 3. RA: Right atrium. RV: Right ventricle.

3. Discussion

Our patient had several risk factors that predisposed her to develop significant VTE involving multiple vascular systems. Historically, Virchow's Triad three critical factors including venous stasis, vascular endothelial injury and hypercoagulable states contribute to thrombosis [14]. Preexisting hypercoagulability due to malignancy and sedentary lifestyle accumulatively may have led to the UEDVT in the beginning.

In our patient, tricuspid thrombus most likely originated from UEDVT and got entrapped on the anterior leaflet of the tricuspid valve (**Figure 3**). This was observed on the TTE as a mobile echogenic structure suggesting type A thrombus [8]. In the recent case report by Myslivecek *et al.*, thrombus was observed on the RV aspect of the tricuspid valve accompanied by bilateral PE [15]. TTE was utilized immediately to make a prompt diagnosis.

Tricuspid valve biomechanics have been extensively researched in the last decade. Anterior leaflet not only has the largest surface area but it is also under greater strain as compared to posterior or septal leaflets [16] [17]. These characteristics of the rapidly changing dynamic environment could have resulted in thrombus entrapment or thrombus formation on the tricuspid valve in a preexisting hypercoagulable state.

In light of the reported literature, about 6% cases of UEDVT are associated with pulmonary embolism [2]. Clinical manifestations such as respiratory symptoms and pleuritic chest pain with the ultimate diagnoses of sub-massive PE and tricuspid thrombus were most likely subsequent complications of the first VTE insult in the upper extremities.

The patient's clinical presentation guided a multidisciplinary workup that led to a timely diagnosis. Objective evidence obtained from CTPA revealed RV to LV ratio of >1 with straightening of the interventricular septum suggesting RV strain (**Figure 1**). These intracardiac changes due to PE were confirmed on TTE along with the revelation of TVT. The pulmonary embolism severity index score (PESI) <65 with elevated troponin put the patient at intermediate-high risk for clinical deterioration [18]. Based on these parameters, the decision was made to observe her in the ICU.

Regarding the ultimate therapeutic modalities, there is no clear evidence for the choice of therapy in this particular group of patients due to the absence of large studies on tricuspid thrombus [8] [10] [19]. Reported therapeutic strategies include anticoagulation, thrombolysis or surgical embolectomy. In our patient, management was guided by clinical manifestation, risk stratification based on workup and continuous monitoring. In the absence of shock on presentation and subsequent hemodynamic improvement, no further escalation such as thrombolysis was required.

Repeat TTE within three weeks revealed an absence of the structure attached to the anterior tricuspid leaflet suggesting resolution of the thrombus (**Figure 4**). As compared to the first study, RV size was found to be decreased and systolic

function improved suggesting improvement in the PE load.

A concomitant triad of TVT with UEDVT and PE is often underdiagnosed. This may be particularly due to the fact that a right-sided thrombus would have already migrated to the pulmonary arteries or resolved with initial therapies before an echocardiogram is performed [20] [21] [22] [23] [24]. However, as much as these make it an interesting and unique presentation, the presence of TVT is no less than the sword of Damocles [25]. Previous studies reported a strong association of RHTs with more hemodynamic instability and increased mortality in the early days [6] [8] [10] [20] [26]. We anticipated hemodynamic instability within the first 24 hours of presentation. Due to this impending danger, the patient was closely monitored in the critical care setting.

It is noteworthy that leukocytosis observed in initial labs down trended during the course of hospitalization. Blood and sputum cultures revealed no growth in the final results. Clinically patient remained non-septic with progressive improvement in hemodynamics after initiation of anticoagulation. These make infective endocarditis an unlikely possibility for the mass seen on the tricuspid valve. This was further confirmed by the absence of the structure attached to the tricuspid valve on repeat echocardiography.

Although our report has significant inherent limitations by design, we ponder that tricuspid thrombus is an infrequent complication of UEDVT that carries a significant risk of mortality. Therefore, the readership will benefit from our experience with this interesting presentation.

4. Conclusion

We reason that tricuspid valve thrombus, UEDVT, and PE with right heart strain make for an interesting presentation, but their simultaneous presence is no less than an impending danger considering associated hemodynamic instability and increased mortality. Prompt clinical assessment, diagnostic workup, application of risk stratification tools, and close monitoring can guide the management and improve outcomes. A repeat transthoracic echocardiography within 2 - 3 weeks may be performed to assess for tricuspid thrombus resolution. This is particularly important as unresolved tricuspid thrombi may cause recurrent PE.

Informed Consent

The case was reviewed by the Institutional Review Board and informed consent was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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