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Case Report

Migratory Massive Venous Thrombi in the Right Atrium without Pulmonary Thromboembolism: A Rare Case Presentation

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ABSTRACT:

Background: A dangerous condition known as right heart thrombi (RHT) in transit occurs when a clot from deep vein thrombosis becomes lodged in the right heart while it travels to the pulmonary arteries (PAs). The clot may migrate and cause further embolic complications. When there are significant bilateral pulmonary embolisms (PEs), RHT are frequently observed.1. When comparing patients with PE who do not have RHT in transit, those with RHT in transit frequently had greater heart rates, lower blood pressure, and more right ventricular dysfunction. The patients experienced cardiopulmonary collapse and ultimately passed away despite receiving intravenous thrombolysis and inotropic help. While less common than left atrial thrombi, right atrial thrombi is nevertheless a significant side effect of central venous cannulation. Anastomotic foci, damaged endothelium, implanted devices, or foreign entities, such as tumors, pacemakers, and indwelling right atrial catheters, can all cause right atrial thrombi. They have been linked to hemodialysis, pulmonary artery catheters, implantable venous access devices, triple-lumen catheters for chemotherapy, intravenous fluids, or parenteral nutrition2. If there is an atrial septal defect or patent foramen ovale, right atrial thrombi can have serious effects such as pulmonary embolism, septic emboli, mechanical issues with heart function, or even systemic embolization. Incident rates of central venous catheter (CVC)related thrombosis reported in the literature are inconsistent and vary according to host factors, catheter characteristics, cannulation site and the infusion administered. Thrombi within cardiac chambers are associated with an increased risk of mortality due to their propensity for embolization to the pulmonary vasculature[2]. Our case presents with pedal edema, bilateral pleural effusion, ascites with prominent dilated abdominal veins with thrombosis hepatic vein, inferior vena cava and massive thrombi in right atrium.

Keyword- thrombus, right atrium, pulmonary thromboembolism

CASE REPORT:

A 26 year old male patient presented with bilateral pedal edema,diffuse abdomen pain and abdomen distension from 15 days and shortness of breath and chest pain from 10 days . patient also complaints of fever with chills from 7 days . there was no history of vomiting diarrohea and jaundice. Past history non significant . General physical examination suggestive of bilateral pedal edema(pitting), febrile (101^0F) Per abdomen examination revealed dilated veins with flow below upward noted on anterior abdomen wall, Tender hepatomegaly 6cm below right costal margine, shifting dullness present . Respiratory system examination revealed dull note on chest percurssion bilateral infrascapular and infra axillary area, reduced air entery below bilateral infrascapular and infra axillary areas. Cardiovascular examination revealed tachycardia, normal blood pressure in all four limbs. on auscultation of heart was unremarkable. On Investigations-routine haemograme Hb 13gm% ,TLC -12400/cumm, Platlets counts 1.67 lac, ESR-25mm/1hr , urine complet microscopy examination was normal. hepatic enzyme SGOT/SGPT was mildly elevated , PT/INR levels was in normal range. D-dimer was in normal range. fasting blood sugar and renal function test was normal.

VDRL, HIV & hepatitis serology was negative. Covid-19 RTPCR was negative. Then we planned CTD profile, that suggestive ANA positive, anticardiolipin antibodies IgM Equivocal and Rheumatoid factor was positive, increased c reactive protein levels. ds DNA &

antiphospholipid antibody IgM &IgG ELISA test were negative.

Xray Chest PA: suggestive of bilateral pleural effusion. ECG revealed sinus tachycardia.

USG: abdomen suggestive of moderate hepatomegaly mild ascites with moderate pleural effusion bilateral.

Colour Doppler Study: reveals echogenic material in hepatic part of IVC and right hepatic vein suggestive of thrombus. no thrombus seen in portal vein and renal veins.

2D ECHO: (Fig.1) study reveals pedenculated mass in right atrium . IVC dilated around 23mm. Trans esophageal echocardiography(TEE) show a right atrial mass attached with inter atrial septum suggestive of thrombus. CT thorax (fig.2) show hypodense filling defect in right atrium which is seen entring into IVC, CT abdomen suggestive of filling defect in hepatic vein,ivc and bilateral external iliac veins suggestive of thrombus.

CARDIAC MRI: (fig.3) reveals non-enhancing leasion measuring approx(22×7mm) in right atrium on to septal leaflet of tricuspid valve with non-enhancing margins on late gadolinium enhancement image likely- thrombus .

So our case presented with polyserositis(ascites,b/l pleural effusion), hepatomegaly , venous thrombosis, right atrial thrombus , ANA & RA factor positive with anticardiolipin antibody IgM equivocal , raised C-reactive protein suggestive of overlap syndrome.

This patient remain hospitalized for 2 weeks and treated with low molecular weight heparine(LMWH), antibiotics,prednisolon and supportive treatment ,oral anticoagulant warfarin(target INR 2.5-3) and advised regular followup with PT/INR monitoring every 3-4 weeks . signs of venous congestion i.e pedal edema , ascites , hepatomegaly was subsided with warferin therapy .

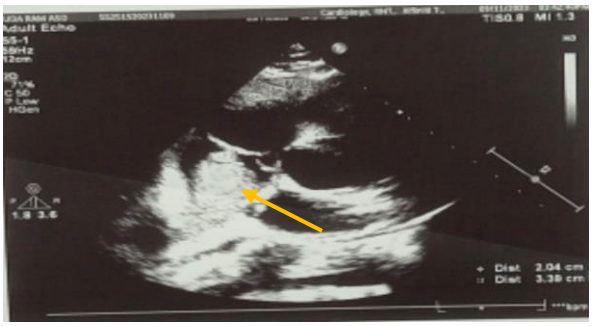

Figure1: Echocardiography suggestive of around 45×21mm mass in right atrium

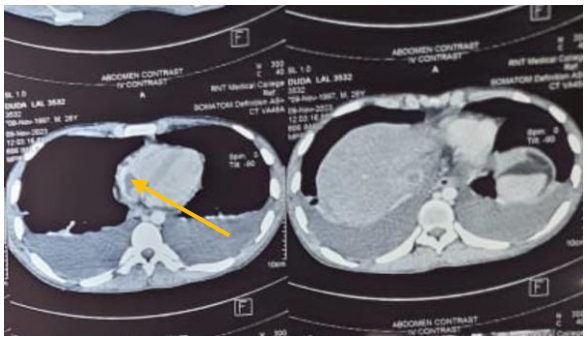

Figure2:CECT thorax and abdomen suggestive of filling defect in right atrium, ivc and b/l pleuraleffusion

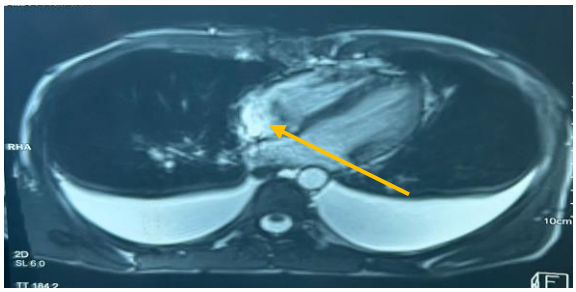

Figure3: cardiac MRI:hypodense filling defect seen in right atrium and ivc

DISCUSSION:

RHT has a strong propensity to embolize and typically originates from the deep leg veins. Between 4% and 18% of patients with acute PE have thrombi in the right heart that may be seen with echocardiography, and having RHT is linked to a high death rate3. In the largest study chartier et al reported on symptomatic free-floating RHT, 79% of RHT were in the right atrium, 16% were in the right ventricle, and 5% were in both right heart chambers.[4] consequences of PE are not RHT characteristics such as size, morphology, or mobility.[5] Pelaez et al. reported that a migratory thrombosis in the right heart became a PE within 24 hours, resulting in massive PE in 40% of cases, which eventually resulted in a 45% mortality rate[6]. The prognosis for trompi connected to the right atrium or right ventricle is often benign. Free-floating, "snakelike" thrombi, however, are typically connected to massively unstable PE. It's common to refer to these mobile RHT as "embooli in transit."7.

The diagnosis of RHT in transit is usually made by echocardiography or computed tomographic angiography. Since every mortality happens during the first 24 hours, more active and urgent care is needed when there is a right heart thrombus in progress. After receiving thrombolytic therapy, the right atrial thrombus most likely moved to the lungs8. It remained unclear, therefore, if the thrombolytic agent's disintegration or migration to the PAs was the cause of the RHT's absence after thrombolysis. Such fragmentation and migration

have been reported when thrombolytic therapy is administered to treat extensive deep venous thrombosis.[9] Anticoagulation, systemic or catheter-directed thrombolysis, and surgical embolectomy are the recommended management options for patients with PE and RHT.[10] As another option, there have been case reports documenting the use of percutaneous catheterization to retrieve RHT in transit. In three reports, there was one successful removal of RHT[11] in four attempts. It is important to remember that percutaneous extraction carries an unknown risk of dislodging or fragmenting the thrombus, either totally or partially, which could result in PE that is deadly. this procedure may represent a potential therapeutic alternative, particularly in patients with contraindications to thrombolysis and surgery.

Even though RHT in travel are typically linked to PE, the best course of treatment has not yet been determined, and the choice of treatments is still up for discussion. Since thrombolysis treats concomitant deep vein thrombosis, PE, and RHT all at the same time, it is a superior therapy option, particularly for patients who are hemodynamically unstable.12 Intravenous thrombolysis, however, has the ability to disintegrate clots in multiple places at once, with the possibility of pieces migrating into the PA and obstructing some of the remaining branches.

Barrios et al reported no significant difference between reperfusion therapy and anticoagulant therapy for mortality and bleeding. The RHT contained an unknown quantity of connected thrombi, which have a lower mortality than free-floating thrombi, which could account for this outcome. Reperfusion therapy was, interestingly, linked to a higher risk of recurrent venous thromboembolism than was the case with pure anticoagulant medication. They came to the conclusion that, regardless of the existence or lack of concurrent RHT, individuals with acute symptomatic PE and related hypotension or shock may be eligible for reperfusion therapy. Even in patients who seem to be clinically stable, heparin alone may not be sufficient treatment for RHT, despite reports of successful treatment of the condition with anticoagulation alone.[13]

However, other writers advise surgical thrombectomy because of the erratic systemic embolism risk that comes with thrombolysis or anticoagulation, which is deemed to be excessively high. Very significant RHT, tricuspid blockage, paradoxical embolism by patent foramen ovale transit, thrombolytic failure, or contraindications to thrombolytic therapy may all be treated surgically. In our presenting case we treated the patient with LMWH followed by oral anticoagulant warfarin overlape with target INR2.5-3 . and patient discharged from MB hospital after 2 week .

CONCLUSION:

RHT should be considered a therapeutic emergency because of the risk for sudden death. Since right heart thrombuses are uncommon, it is unlikely that two or three distinct therapy arms will be included in a randomized trial very soon. For the treatment of PE made more difficult by snakelike RHT, there are no evidence-based recommendations. The best course of action for managing this serious ailment is still up for debate. It appears that conventional anticoagulation should be chosen over routine reperfusion therapy in hemodynamically stable patients with PE and RHT, while thrombolysis should be reserved for patients who are unstable or who are developing hemodynamic instability. Primarily, before to initiating any therapeutic regimen, it is crucial to evaluate the possible short- and long-term adverse effects versus alternative approaches and the dangers associated with not receiving treatment.

REFERENCES:

1. Torbicki A., Galie N., Covezzoli A., Rossi E., De Rosa M., Goldhaber S.Z., ICOPER Study Group Right heart thrombi in pulmonary embolism. J Am Coll Cardiol. 2003;41:2245–2251.

2. Casazza F., Becattini C., Guglielmelli E., Floriani I., Morrone V., Caponi C. Prognostic significance of free-floating right heart thromboemboli in acute pulmonary embolism: results from the Italian Pulmonary Embolism Registry. Thromb Haemost. 2014;111:53–57.

3. Chapoutot L., Nazeyrollas P., Metz D., Maes D., Maillier B., Jennesseaux C. Floating right heart thrombi and pulmonary embolism: diagnosis, outcome, and therapeutic management. Cardiology. 1996;87:169–174.

4. Chartier L., Bera J., Delomez M., Asseman P., Beregi J.P., Bauchart J.J. Free-floating thrombi in the right heart: diagnosis, management, and prognostic indexes in 38 consecutive patients. Circulation. 1999;99:2779–2783.

5. Koc M., Kostrubiec M., Elikowski W., Meneveau N., Lankeit M., Grifoni S. RiHTER Investigators. Outcome of patients with right heart thrombi: The Right Heart Thrombi European Registry. Eur Respir J. 2016;47:869–875.

6. Pelaez J.B., de Miguel E.M., Moreno R.S., Gómez L.R., Etxebeste J., Martín R.G. Right-atrial floating thrombus attached to the interatrial septum

with massive pulmonary embolism diagnosed by echocardiography. Int J Cardiol. 2008;131:125–127.

7. Rose P.S., Punjabi N.M., Pearse D.B. Treatment of right heart thromboemboli. Chest. 2002;12:806–814.

8. Thery C., Bauchard J.J., Lesenne M., Asseman P., Flajollet J.G., Legghe R. Predictive factors of effectiveness of streptokinase in deep venous thrombosis. Am J Cardiol. 1992;69:117–122.

9. Goldsmith J.C., Lollar P., Hoak J.C. Massive fatal pulmonary emboli with fibrinolytic therapy. Circulation. 1990;64:1068–1081.

10. Kinney E.L., Wright R.J. Efficacy of treatment of patients with echocardiographically detected right-sided heart thrombi: a meta-analysis. Am Heart J. 1989;118:569–573.

11. Davies R.P., Harding J., Hassam R. Percutaneous retrieval of a right atrioventricular embolus. Cardiovasc Intervent Radiol. 1998;21:433–435.

12. Mahajan K., Negi P., Asotra S., Dev M. Right atrial free-floating thrombus in a patient with acute pulmonary embolism. BMJ Case Rep. 2016;2016

13. Barrios D., Chavant J., Jiménez D., Bertoletti L., Rosa-Salazar V., Muriel A. Registro Informatizado de la Enfermedad TromboEmbólica Investigators. Treatment of right heart thrombi associated with acute pulmonary embolism. Am J Med. 2017;130:588–595.