“Iceberg in Titanic?”: A Case of a Huge RV Thrombus in a Desperately Sinking (Failing) Heart

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Introduction

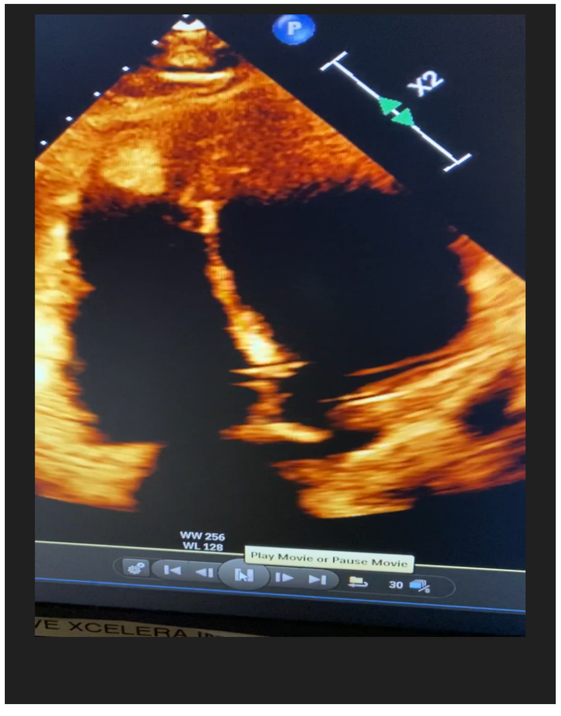
The incidental finding of cardiac chamber clots in patients with heart failure, MI or ischemic stroke is a harbinger of management and therapeutic challenges. While left sided cardiac thrombi are not unheard of, right sided thrombi are relatively rare. Patient approach and clinical decision-making needs to be tailored to each individual case given the high interdependence of major systems functioning. Here we describe a case of Stage C biventricular heart failure with an RV thrombus in the setting of thrombocytopenia with liver cirrhosis.

Case Summary

67 years old female with a PMH of DM-II, HTN, COPD, CAD in 2019 with diastolic dysfunction, Hepatitis C and liver cirrhosis presented to hospital with complaints of NYHA IV dyspnea, peripheral edema and no chest pain. On examination, she was alert and appropriately oriented but extremely dyspneic, with regular heart rate and rhythm, maintaining BP well, audible bilateral basal lung crackle and grade III peripheral pitting edema. Initial lab work up revealed a hemoglobin of 9.0 with normal MCV, platelet count of 57, serum sodium 135 mEq/L, NT pro BNP of 24000, cardiac tropnonins not elevated. EKG showed a heart rate of 112 per minute, regular rhythm, normal axis, no ST-T wave changes, CXR suggestive of cardiomegaly with bibasilar airspace disease and pleural effusion.

Case Summary continued….

2D transthoracic echocardiography revealed a severely reduced ejection fraction of 10-15%, mild LV dilation with severe global hypokinesis, RV moderate to severely dilated with a discrete echo dense mass with well-defined borders seen throughout systole and diastole, attached to the RV apex measuring 2.8 x 1.5 cm. Venous doppler legs was negative for DVT bilaterally. Given patient’s history of severe allergic reaction to IV contrast, we could not perform CT-PA to rule out pulmonary embolism. She had low anti thrombin III, Protein C and S levels. She tested negative for anti- phospholipid antibodies, Prothrombin gene and Factor V Leiden mutations.



2DTTE: Apical 4 chamber view showing thrombus in RV cavity attached to apex

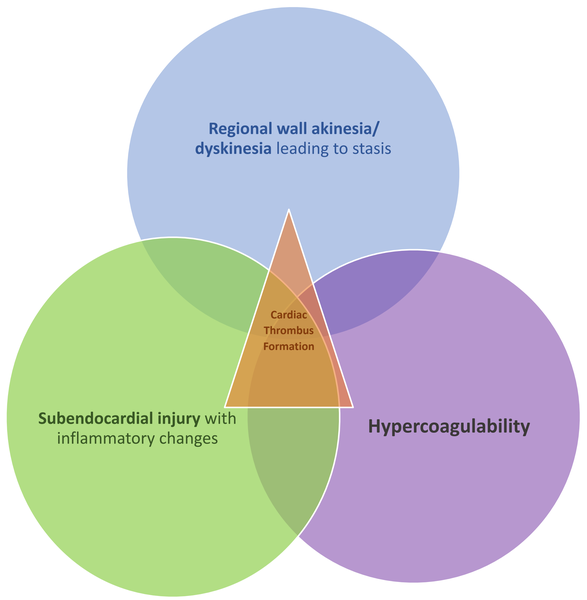
Case Summary continued….

She was managed for heart failure with IV diuretics and fluid restriction. Given patient’s clinically precarious situation in the setting of severe biventricular failure, thrombocytopenia with cirrhosis and anemia, it was deemed best to manage conservatively with IV heparin instead of EKOS. Of note, she also developed unilateral upper limb DVT secondary to IV line trauma during hospital stay. She was eventually transitioned to oral anti coagulants and discharged to rehab in a stable condition. She is to have 6 months follow up ECHO with primary cardiologist at which time, cardiac cath will also be planned to further evaluate her new low EF.

Discussion

- Intracardiac thrombi are one of the three common differentials of intracardiac masses, the other two being vegetations and tumors. Right sided cardiac thrombi are classified as either thrombi in transit i.e. originating from deep veins peripherally and embolizing to heart and lungs or immobile/ thrombi in situ[1]. Our patient did not have any prior or current h/o DVTs before developing RV thrombus and echocardiographically it seemed immobile and attached to RV wall.

- Commonly, in situ cardiac thrombi originate after MI, like anterior wall MI and development of LV thrombus, or with AF, indwelling catheter, prosthetic valves, anti-phospholipid antibody syndrome. Advancing age, smoking and HTN also predispose to cardiac thrombi[2]. Our patient developed RV thrombus presumably secondary to underlying enlarged RA and RV with decreased cardiac output predisposing to stasis of blood in the setting of a procoagulant state i.e. liver cirrhosis. Her thrombophilia screen was positive for abnormal Anti Thrombin III, Protein C and S only which are expected to be low in acute thrombosis. These need to be checked again once she is off anticoagulation for at least 4 weeks.



- Oral anti coagulation, thrombolysis and surgical embolectomy have similar efficacy. Given the convenience of NOACs, oral drug therapy should probably be chosen[3]. Thrombolysis should be performed if compelling indications exist like mobile thrombus migrating to lungs, cardiogenic shock, thrombus protruding into LA via PFO[1].

REFERENCES

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