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Case Report

Right Ventricular Myxoma Obstructing RV Inflow and Causing Pulmonary Embolism: Management and Brief Review

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Abstract

A rare case of RV myxoma causing RV inflow obstruction is being discussed. It was associated with recurrent pulmonary embolism leading to a clinical picture of pulmonary disease. The patient was managed with resection of tumor and tricuspid valve replacement.

Keywords: *Myxoma; Right Ventricle; Pulmonary Embolism; Inflow Obstruction; Cardiac Tumor*

Abbreviations

RV: Right Ventricular; CT: Computerised Tomographic

Case Presentation

32 Years old male was referred to our facility with history of cough, mucoid expectoration and fever for last 7 days. There was history of recurrent lower respiratory tract infections and pleural effusions. Patient had received anti-tubercular therapy twice in the past. Examination revealed bilateral crepitations with reduced breath sound in right infra-scapular, infra-axillary and infra-mammary regions.

X-ray chest revealed right-sided pleural effusion with haziness in right lower zone. Inhomogeneous opacities were seen in left middle and lower zone. Computerised tomographic (CT) scan of chest and CT pulmonary angiogram were performed. They revealed moderate right-sided pleural effusion and mild left-sided pleural effusion. Volume of right hemithorax was reduced with fibro-atelectatic lesions in right upper lobe and consolidation collapse of right lower lobe. 'Tree-in-bud' nodules were seen in both lung fields. There was mildly dilated main pulmonary artery and an eccentric filling defect in right pulmonary artery. Poor opacification of right lower lobe pulmonary artery branches was noted.

An echocardiogram was performed. Right sided chambers were difficult to image from standard views as heart was shifted to right. The leaflets of tricuspid valve were not clearly visible and there was a large mass of size 2.7 x 3.2 cm which appeared to be attached to anterior tricuspid leaflet (Figure 1 and 2). It was mildly hyperechoic and of uniform echogenicity with regular margins. The mass was ob-

structing the right ventricular (RV) inflow causing a mean diastolic gradient of 14 mmHg across the tricuspid valve (Figure 3). Right atrium was severely enlarged and there was mild RV systolic dysfunction. Mild tricuspid valve regurgitation with calculated right ventricular systolic pressure of 30 mmHg was noted. Spontaneous echo contrast was seen in right atrium. Left atrium and left ventricle were normal.

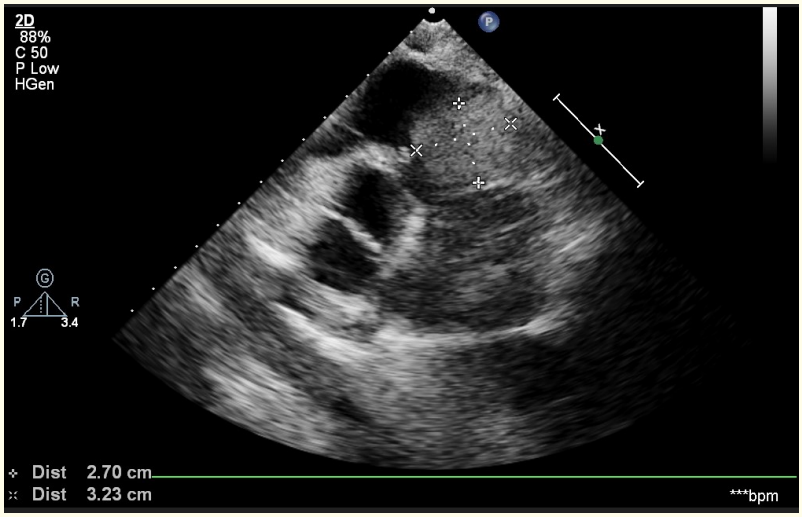

Figure 1: *Modified parasternal long axis view of transthoracic echo showing right ventricular inflow with mass lesion of 2.7 x 3.2 cm in right ventricle abutting anterior tricuspid leaflet.*

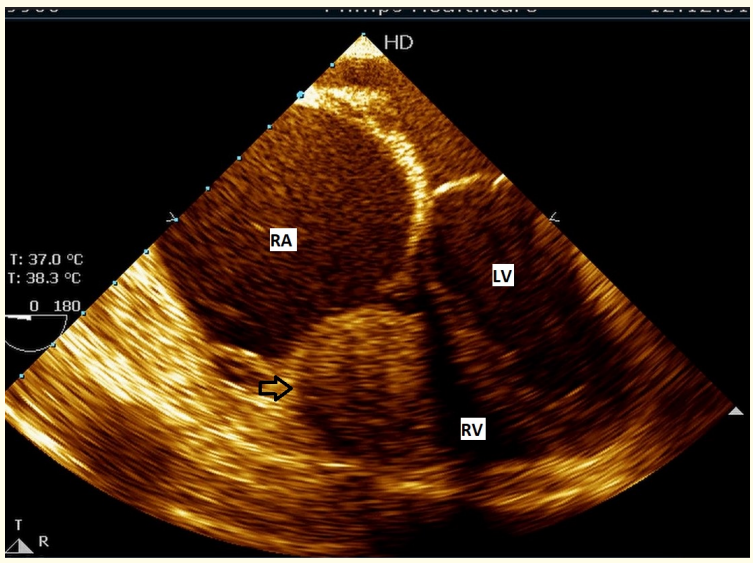

Figure 2: *Four-chamber view of transoesophageal echo showing mass lesion (black arrow) in right ventricle. RA: Right Atrium, RV: Right Ventricle, LV: left Ventricle.*

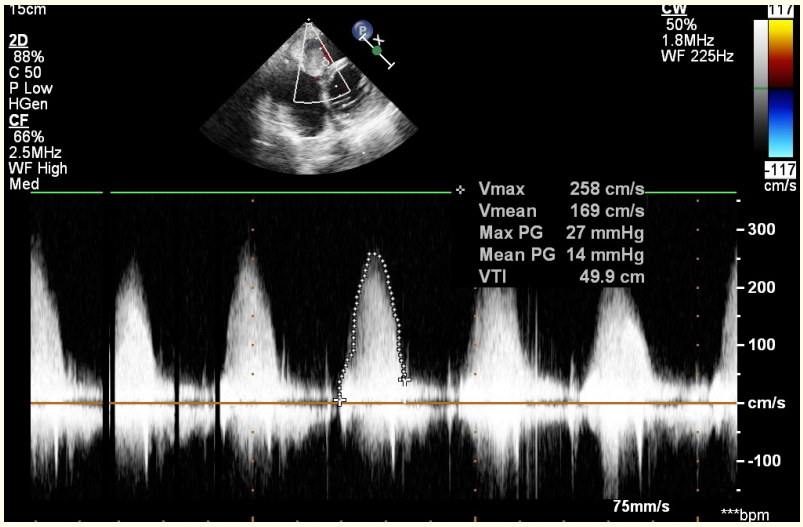

Figure 3: *Continuous wave Doppler across tricuspid valve showing a mean diastolic gradient of 14 mmHg.*

Surgical resection of mass was planned. After RA incision, it was found that it is difficult to approach the RV mass through tricuspid valve, so an RV incision was made. It revealed two masses attached to RV free wall abutting tricuspid valve and involving the lower surface of anterior tricuspid leaflet. Both masses were excised and 27 mm bioprosthetic valve was sutured to tricuspid valve annulus (Figure 4 and 5).

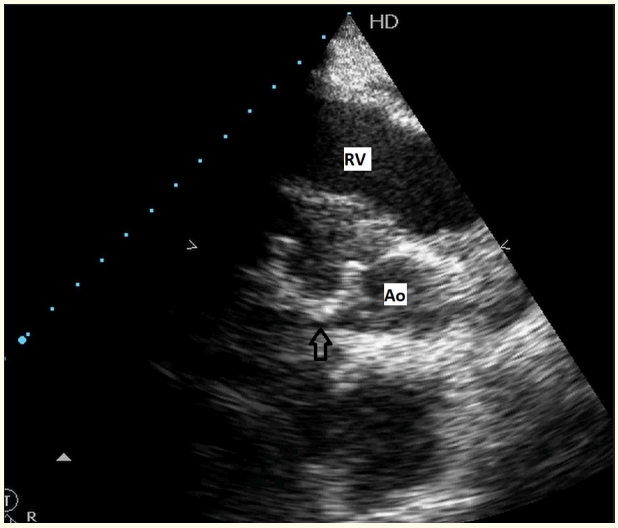

Figure 4: *Short axis view at aortic valve level of transthoracic echo showing bioprosthetic valve at tricuspid position (black arrow). Ao: Aortic Valve, RV: Right Ventricle.*

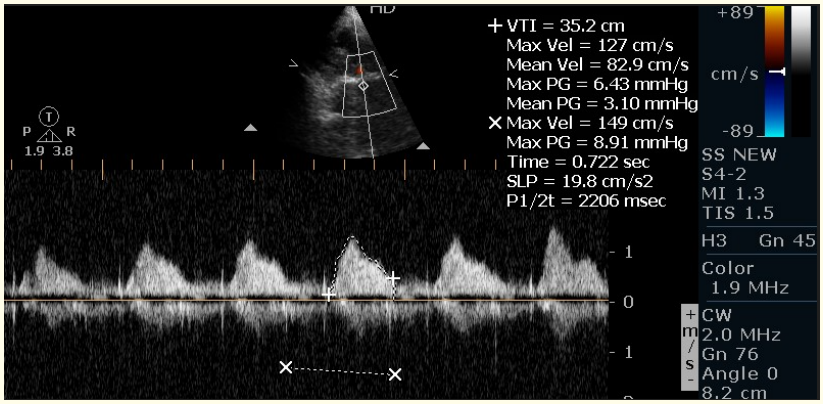

Figure 5: *Continuous wave Doppler across bioprosthetic valve at tricuspid position showing a mean gradient of 3 mmHg after resection of right ventricular mass.*

Histopathological examination of the masses revealed predominant areas of infarct necrosis with karyorrhectic debris. Small foci of viable areas revealed cellular lesions embedded in myxoid stroma, comprising of stellate cells with abundant eosinophilic cytoplasm, indistinct cells border, hyperchromatic nuclei and indistinct nucleoli. Possibility of cardiac myxoma was suggested.

The patient was discharged in stable condition.

Discussion

Three-fourth of cardiac tumors are benign. Myxomas are commonest benign tumors of heart. Most of myxomas occur in left atrium (75 - 80%). Exact incidence of RV myxomas is not known, however, they constitute about 2.5 - 4% of all cardiac myxomas [1]. In fact, in one of the studies with a duration of 50 years, ventricular myxoma was present in only 8 patients out of total 197 cases [2].

RV myxomas are possibly more common in outflow tract [1,3]. Rarely, large myxomas obstructing RV inflow have also been reported [4]. Our patient had significant inflow obstruction with mean gradient of 14 mmHg across tricuspid valve. This led to systemic venous hypertension and contributed to recurrent pleural effusion. Additionally, there was evidence of recurrent pulmonary embolism of part of tumor leading to lung necrosis and fibrosis. Embolization to pulmonary arteries is a rare but clinically significant feature of RV myxomas. In one of the recent reviews published in 2018, 12 cases of RV myxoma associated with embolization to pulmonary arteries were detailed [2]. One more case of this phenomenon was published in 2019 [5]. The propensity to embolize increases with villous myxomas, small size and presence of atrial fibrillation. At times, large tumor may become source of large fragment embolization. Tumor emboli have a propensity to lodge in more peripheral subsegmental arteries [5].

These events led to a clinical picture which was suggestive of a pulmonary disease rather than a cardiac pathology and the correct diagnosis remain hidden for long. As in this case and reported previously also, the diagnosis of RV myxoma may remain elusive for long because of rarity of occurrence, atypical presentation, overlap with pulmonary symptoms and low degree of suspicion. These features make the overall prognosis relatively poor [6].

Beside RV inflow obstruction and pulmonary embolization, another distinctive feature in this case was tricuspid valve replacement necessitated by involvement of anterior tricuspid leaflet by myxoma. This was possibly one of the rare occasions when RV myxoma resection was done with replacement of tricuspid valve.

Conclusion

This was a rare case of RV myxoma causing RV inflow obstruction with recurrent pulmonary embolism mimicking pulmonary pathology for long. The case was managed with resection of myxoma and tricuspid valve replacement.

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