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Wrong at Right-A Rare Presentation of Right a Trial Thrombus

Nithin Bharadwaj[1], Dr. Chandra Shekara Reddy[2], Dr. Arun Sriniivas[3], Dr. Siddarth Kumar Chawath[4], Dr. Lakshminarayan Achar[5]

[1]Senior Sonographer, Apollo BGS Hospital
nithinbharadwaj1997[at]gmail.com
+91 8904476838

[2]G. B., Apollo BGS Hospital, Senior Registrar
drcsreddycardio[at]gmail.com
+91 9900130723

[3]HOD, Department of Cardiology, Apollo BGS Hospital (Corresponding Author)
arunsrinivascpubs[at]gmail.com
+91 9845028387

[4]Apollo BGS Hospital Senior Registrar
siddarthchawath[at]gmail.com
+91 9901174111

[5]Apollo BGS Hospital, HOD-CTVS Surgeon
drlnachar[at]rediffmail.com
+91 9845551469

Running Title: *A Rare Presentation of Right a trial Thrombi in RHD Sequalae*

Abstract: ***Background*: Rheumatic Heart Disease is one of the common Valvular Heart Disease. Associated Atrial Fibrillation may result in Cardio Embolic Stroke and related complications. *Case Presentation*: Sixty-year old male admitted with palpitation and breathlessness since 2 weeks and aggravated since 3 days. He had past history of CVA and Seizure Disorder. He underwent PTMC for RHD-Severe MS (2009) and Craniotomy for SDH drainage (2009). Currently had Severe MS, AF with FVR, Large LA Clot, Severe TR, Severe LV Systolic Dysfunction, Cardiogenic Shock and Respiratory failure. Managed with Anti Arrhythmics, Inotropes and Mechanical Ventilation supports. His Coronary Angiogram was Normal. After CTVS consultation with high-risk consent under GA and CPB support, underwent successful removal of Bi Atrial Clots (RA Clot was an incidental finding) and MVR with 27 mm TTK Chitra Heart Valve. Clinical course was complicated by AKI & deranged LFT (symptomatically treated) Bilateral Pleural Effusion (drained) and Rt. Vocal Card Palsy (Tracheostomy done). Gradients across MV improved at discharge. *Discussion*: RA thrombus is rare.[1] Clinical presentation varies from Asymptomatic to PE to SCD.[2] Echocardiography, CT and CMR are useful noninvasive diagnostic tools.[2] Treated with Anticoagulants, Thrombolysis or Surgical thrombectomy.1 Multidisciplinary approach is the cornerstone for successful diagnosis, treatment and follow up.[3] *Conclusion*: Early diagnosis and management of Intra Cardiac Thrombi are key to prevent systemic complications.[2] Systemic Anticoagulation should be considered carefully on case to case basis.[2] Mortality in Right Heart Thrombi remains high regardless of the cause or chosen treatment.[4]***

Keywords: Intra Cardiac Thrombi, Pulmonary Embolism, Severe Mitral Stenosis, Thrombo Embolism, Percutaneous Transvenous Mitral Commissurotomy, Systemic Anticoagulation

1. Introduction

Intra Cardiac Thrombi, a rare and life threatening condtion. Traditionally managed by Surgical excision followed by use Oral Anticoagulants (OACs) is advised, but this is limited by elderly age, comorbids and unexpected responses from OACs. To the best of our knowledge, there is no other alternative safe and feasible method recommended.

2. Case Report

A Sixty year elderly Male was admitted with history of palpitation and breathlessness since 3 weeks and worsened since 3 days. Associated with tiredness, decreased appetite and vomiting for 1 day. With known case of RHD-MS and AF, CVA and Seizure Disorder, he underwent PTMC in 2004 (on Oral Anti Coagulation) and Craniotomy (for Sub Dural Hematoma Drainage in 2009). On arrival at our ER visit, he had Tachycardia of 160/min and BP of 110/70 mm of Hg. Cardiac Systemic Examination revealed Tachycardia and Neurologically he was drowsy, restlessness and without any focal neurologic deficits. Initially evaluated with **ECG** which showed AF with Fast Ventricular Rate.2**D Echocardiography** revealed, RHD-Severe MS and Large LA Clot with Severe LV Systolic Dysfunction EF-30% in AF (**Fig. A**). **Chest X Ray** showed Patchy opacities in Right Lower Zone. Stabilized with Antiplatelets, Inotropes (as later developed Cardiogenic shock with BP of 70/40 mm of Hg), Anticoagulants, Antiarrhythmics, Diuretics, Bronchodilators and other supportive measures. In view of drowsiness and hemodynamic instability, he was electively Intubated in ER iself for Mechanical Ventilation support and then gradually extubated after 8 days. Other important Investigations are-**Doppler Study of Carotids**, which

reports Right ICA of 10-20% lesion, **USG Abdomen** shows Single Hepatic Cyst in Segment IV of Left Lobe of Liver and **CT Brain**-Chronic Right Fronto-Parietal Subdural Collection with Dural calcification. Advised high-risk Surgical excision of large LA clot and MVR. **Coronary Angiogram** was Normal. Referred to Cardiothoracic Surgical team, relatives were counselled and consented for the same. We **regularly monitored** few more important investigations that includes LFT (for Ischemic Hepatitis), S. Albumin (for Hypoalbuminemia), ABG (for Respiratory and Metabolic status), RFT (for AKI and Electrolyte Imbalances), Urine Routine (for early Ketosis and UTI) and PT (for Bleeding tendencies). CXR and Screening 2D Echo was also done on regular basis. Hemodynamic parameters including Intake / Output were monitored with CVP, Arterial lines and Foley's cathetheterization.

Mitral Valve Replacement Surgery with Clots extraction: After high risk consent, the procedure was done under General Anesthesia with Mechanical Ventilation and CPB support, in Supine position with Median Sternotomy incision. We observed Dilated RA, RV and PA with Severe MS-which has heavily calcified both MV and sub mitral apparatus. Big LA clot was present extending upto LAA and Pulmonary Veins. IV Heparin given. Went on CPB with Aortic 22 Fr Aortic and Bicaval Cannulation. Aorta cross-clamped. Deinido CP given. Heart arrested in diastole. LA opened. MV inspected and excised. LA Clot was removed and MV Replaced with 27 mm TTK Chitra Heart Valve using 2.0 Ethibond, pledgeted interrupted sutures. LA closed with 4-0 Prolene. De-airing done. RA opened and RA clot was removed. All the clots were removed by piecemeal approach (**Fig. B**). Incidentally found to have RA Clot, which was also removed successfully. Mild TR was present with Saline test. Cross-clam released. Came-off CPB in NSR. De-cannulation done. Heparin reversed with IV Protamine. Hemostasis achieved. Mediastinal and Left & Right pleural tubes were placed. RV and RA pacing wires were placed. Sternum closed using No.6 wire. Wound closed in layers.

Post Procedure: He was gradually weaned off from CPB, Inotropes and Mechanical Ventilation supports. Received total of 6 units of Platelet Concentrations (for thrombocytopenia) and 4 units PRBC's transfusions (for surgery). His **In-Hospital complications** are: Bilateral Pleural Effusion (due to Hypoalbuminemia secondary to Chronic Alcoholism and drained about 2 liters of transudative fluid), AKI (due to initial Cardiogenic Shock, managed conservatively with Nephrologist's advise), Deranged LFT (due to congestive hepatopathy from Severe MS → Dilated LA → Severe TR → Plethoric IVC, managed with Percutaneous Endoscopic Gastrostomy (PEG) by Gastroenterologist and Dysphagia with Right Vocal Card Palsy (due to local damage or CVA, managed with Tracheostomy done by ENT Surgeon) for nearly 3 weeks. But Tracheostomy recanulated later because of desaturation. Gradually stabilized. Both Tracheostomy tube and PEG care were continued at discharge. Also received High Protein diet. Physiotherapy with Chest Physiotherapy, Deep breathing exercises, Thoracic expandable exercises, Inspiratory Spirometry and Positioning. At discharge, **2D Echocardiography** (**Fig. D**) showed S/P MVR-Normally functioning MV prosthesis with LVEF of 40%. Currently on clinical follow up for 3 years with stable Cardiac status.

3. Discussion

Right Atrial Thrombus (RAT) is rare and its incidence in a review of Sweden was 7% from 23796 autopsies with similar incidence Left Atrial Thrombus (LAT) noted.[1] (*Benjamin, 2017*) Clinical presentation varies from Asymptomatic to Pulmonary Embolism (PE) to Sudden Cardiac Death (SCD).[2] *(Prudhvi, 2020)* The prevalence of Right Heart Thrombus (RHT) in the setting of PE is 4.18%.[3] (*Selwanos, 2020)* Clinical consequences depends on clot size and overall clot burden.[4] (*Case Reports in Pulmonology, 2012)*

Rarity of RAT probably due to less platelet reactivity of Right Atrial (RA) Wall, Wider size, lack of Anatomical Remodelling and shallow anatomy of Right Atrial Appendages (RAA) with lower RAA emptying velocities.[1, 2] Thrombus formation is caused by altering **Virchow's Triad (**Intra Cardiac Chamber wall, Blood Flow and Blood component) as mentioned in the (**Table A**).[2] Paradoxical emboli is one of the dreadful complications of RHT via Patent Foramen Ovale (PFO).[3] RHT forms either directly due to Atrial Fibrillation (AF) or embolized from Deep Vein Thrombosis (DVT).[3] **RHT in Transit** (RHThIT)-is a rare phenomenon in the absence of structural heart disease, AF or Device located in Superior Ven Cava or Heart chambers.[4] But it is associated with Thrombolysis, Malignant Tumors, Crohn's disease or Behcet's Disease.[4] Clinical consequences depends on clot size and overall clot burden.[4]

Echocardiography, Computarized Tomography (CT) and Cardiac Magnetic Resonance (CMR) are useful noninvasive tool for the diagnosis of Intra cardiac thrombi.[2] RA thrombus most commonly diagnosed by Trans Thoracic Echocardiography (TTE), where as its site, origin and mobility were better defined by Trans Esophageal Echocardiography (TEE).[1] For diagnosis of Intra Cardiac Thrombi-TEE is better than TTE.[2] CT has better sensitivity & specificity than Echo, but risked by Intra Venous Contrast.[2] CMR (T1 delayed enhancement CMR) has highest diagnostic accuracy (99.2%), Sensitivity (100%) and specificity (99.2%).[2] The **European working group of Echocardiography** (in 1989) proposed morphological classification and described 3 patterns of RHT (**Table B**).[3] Those with Mechanical Valves, Right sided Pacemaker Leads, Shunt (ASD / VSD) Closure Devices and any indwelling venous lines will carry high risk of formation of RA thrombus.[1] In our case, the **probable mechanism of** RA clot may be migration of LA Clot from stretched and dilated LA either across PFO or through previous IAS puncture for any interventions (Note: Past h/o PTMC, 18 years ago) (**Fig. C**). **Differential Diagnosis** includes RA masses from Benign or Malignant / Primary or Metastatic tumors, TV Vegetations, Thrombi in Transit or Trans Venous embolized thrombi.[1] Right sided prosthetic valve thrombosis is more common than left.[2] Echo provides details of thrombus, reduced effective orifice area, elevated gradients across prosthetic valves, decreased mobility or immobile leaflets.[2]

The optimal therapeutic approach is still a subject of debate.[3] As a general rule, an individualized approach based on characteristics of the patient and thrombus needed.[4] Treated with Anticoagulants, Thrombolytic agents or Surgical thrombectomy (depending on its morphology and risk of PTE).[1] (*Benjamin, 2017)* Unfractionated Heparin, LMWH, VKAs are commonly used anticoagulants.[2] (*Prudhvi, 2020)* Initial bridging with Heparin or LMWH with VKAs to reach standard INR goal of 2 to 3 is commonly used Anticoagulation regimen.[2] Thrombolysis is simple, rapid and readily available therapy.[3] (*Selwanos, 2020)* Fate or Complications of Thrombolysis includes Major bleeding, Clot Fragmentation and Migration may results in either Complete PE or Recurrent PE for partial dissolution of the venous thrombus.[3] Surgery is the treatment of choice for Prosthetic valve obstruction by thrombus or thrombus size > 0.8 cm^2.[2] Thrombolysis is preferred as second line of treatment.2 RHThIT is considered to be an extreme therapeutic emergency (along with DVT), it should be sought in patients with massive PE.[4] Echocardiography is essential to rule out PFO.4 Although there is no consensus for preferred treatment of RHThI, rapid diagnosis and management is essential.[4] Other modalities of treatment are: Catheter based interventions (Aspiration thrombectomy, Thrombus Fragmentation and Rheolytic Thrombectomy) and recurrences can be prevented by Retrievable IVC Filters.[4] (*Case Reports in Pulmonology, 2012)* The European Cooperative study suggests that the mortality rate of risk was 60% with Anticoagulation, 40% with Thrombolysis and 20% after Surgery.[3] Surgical thrombectomy is warranted in those with large and mobile thrombi and recurrent thromboembolization, while pts are already on optimal anticoagulation.[2] Surgery itself has many limitations (**Table C**).[3] If there is massive PE, needs **Pulmonary Embolism Response Team** (PERT)-includes Multidisciplinary team involving Cardiologist, Radiologist and Cardiothoracic Surgeons.[3]

4. Conclusion

Early diagnosis and management of Intra Cardiac Thrombi will be key to prevent systemic complications.[2] The diagnostic accuracy of various cardiac imaging modalities varies with location of thrombi.[2] RHT represent a challenging diagnosis that requires determination of its source, morphology, anatomical extent and clinical presentation.[3] Systemic Anticoagulation should be considered carefully on case to case basis.[2] Surgical Embolectomy remains standard mode of treatment.[3] Multidisciplinary approach is the cornerstone for successful diagnosis, treatment and follow up.[3] Mortality in patients with RHT remains high regardless of the cause or chosen treatment.[4]

Leaning Points:

a) Extent of presence of thrombi will be accurately defined only during surgery.
b) Primary Intra Cardiac thrombi of RA are rare.
c) RA clots were probably migrated from LA either across PFO or through previous IAS puncture for any intervention.
d) Management Delimas:
- Use of Platelet Cell Transfusions for Thrombocytopenia and OAC (Oral Anti Coagulation)-Its excess use may results in more Thrombus formation and less use increases bleeding tendencies.

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Nithin Bharadwaj contributes to complete conceptualization and data collection, Chandra Shekara Reddy creates complete manuscript preparation and editing, Arun Srinivas is corresponding author and provides complete guidance and proof correction of manuscript, Siddarth Kumar Chawath guides complete journal search and publication guidelines and Lakshminarayan Achar permits to access complete surgical notes and figures / photos.

Affiliations / email address / Role:

1) Mr. Nithin Bharadwaj-[1]nithinbharadwaj1997[at]gmail.com-First Author, Senior Sonographer-Complete conceptualization and Data collections
2) Dr. Chandra Shekara Reddy G. B-[2]drcsreddycardio[at]gmail.com-Second Author, Senior Registrar-Complete Manuscript Preparation and Editing
3) Dr. Arun Srinivas-[3]arunsrinivascpubs[at]gmail.com-Third Author, Corresponding Author, HOD-Dept. of Cardiology, Interventional Cardiologist-Complete guidance and proof correction of Manuscript
4) Dr. Siddarth Kumar Chawath-[4]siddarthchawath[at]gmail.com-Fourth Author, Senior Registrar-Complete Journal search and publication guidance
5) Dr. Lakshminarayan Achar-[5]drlnachar[at]rediffmail.com-Fifth Author, HOD, Department of CTVS-Complete surgical notes and figures

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Tables and Figures

Table A: Components of Virchow's Triad: [2] *(Prudhvi, 2020)*

Cause	Description
Chamber Wall causes	Myocardial Infarction, Dilated LA / RA, Ventricular Aneurysms, Dilated Cardiomyopathies (Dilated, Peripartum, Takosubo, Stress related, Non Compaction), Endocardial Injury due to Central Venous Catheters, Pacemakers, Defibrillator leads, Left Ventricular Assist Devices (LVAD) and Atrial Septal Aneurysm
Abnormal Flow States	Heart Rate or Rhythm disturbances (Atrial-Fibrillation or Flutter, Ventricular-Fibrillation or Tachycardia), Increased turbulence due to Prosthetic Valves, Valve Stenosis (MV, AV, TV) and Mitral Annular Calcification
Blood Component Causes	Hypercoagulable states, Protein C and / or S deficiency, Antiphospholipid Antibody Syndrome and Paraganglioma due to Catecholamine excess

Table B: Morphological Classification of RHT by European working group of Echocardiography: [3] *(Selwanos, 2020)*

Class	Features
Type A Thrombi	• Serpiginous worms like appearance • Freely mobile • Associated with Pulmonary Embolism
Type B Thrombi	• Non Mobile • Ovoid in shape • Finally attached to chamber wall • Formed in-situ associated with cardiac abnormalities.
Type C Thrombi	• Similar appearance to Atrial Myxoma • Highly mobile

Table C: Surgery in RHT is limited by: [3] *(Selwanos, 2020)*

❖	Lack of availability of Surgical expertise
❖	Inherent delay while preparing for surgery
❖	Depressant effects of Anesthetic agents and Cardioplegia
❖	Inability to remove coexisting Peripheral-Pulmonary Emboli

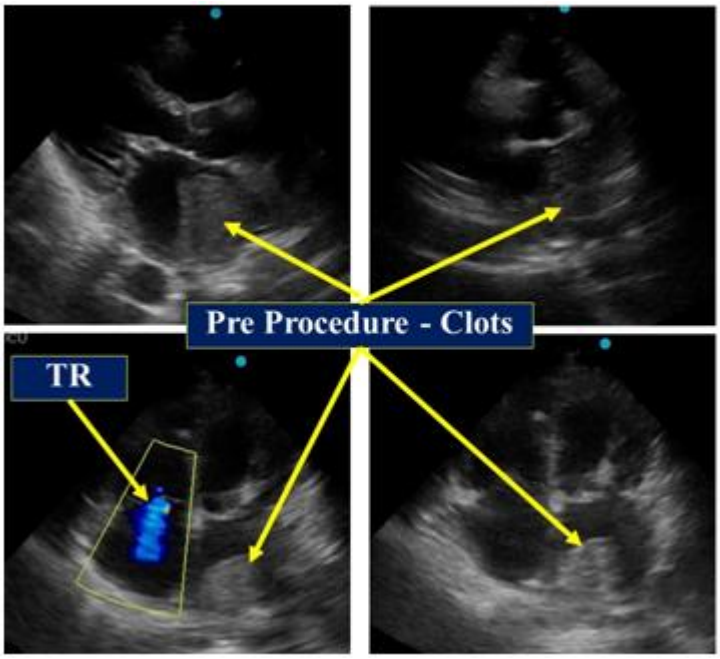

Figure A: Pre Procedural 2D Echocardiographic Stills

2D Echocardiography revealed, RHD-Severe MS, MVA-0.6 cm^2, PPG / MPG was 14/8 mm of Hg, Mild MR, Severe TR with PAH RVSP-60 mm of Hg, RV dysfunction, Large LA Clot, Diameters of Mitral & Tricuspid Annulus are 36 and 35 mm, Global LV Hypokinesia with Severe LV Systolic Dysfunction EF-30%, and Trace Pericardial Effusion

Both LA and RA clots were removed.
All clots were removed by piecemeal approach
Figure B-Clots extracted

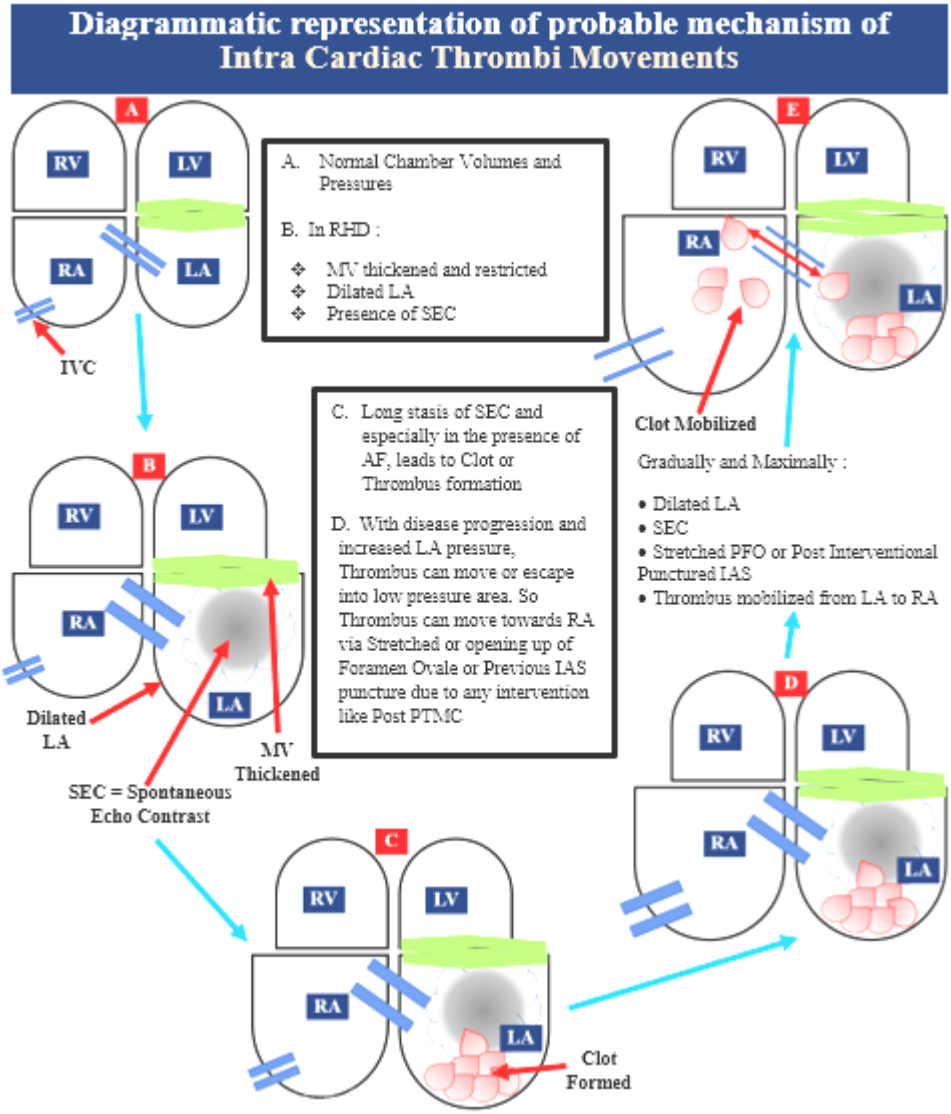

Figure C: Probable mechanism of Clot migration from LA to RA

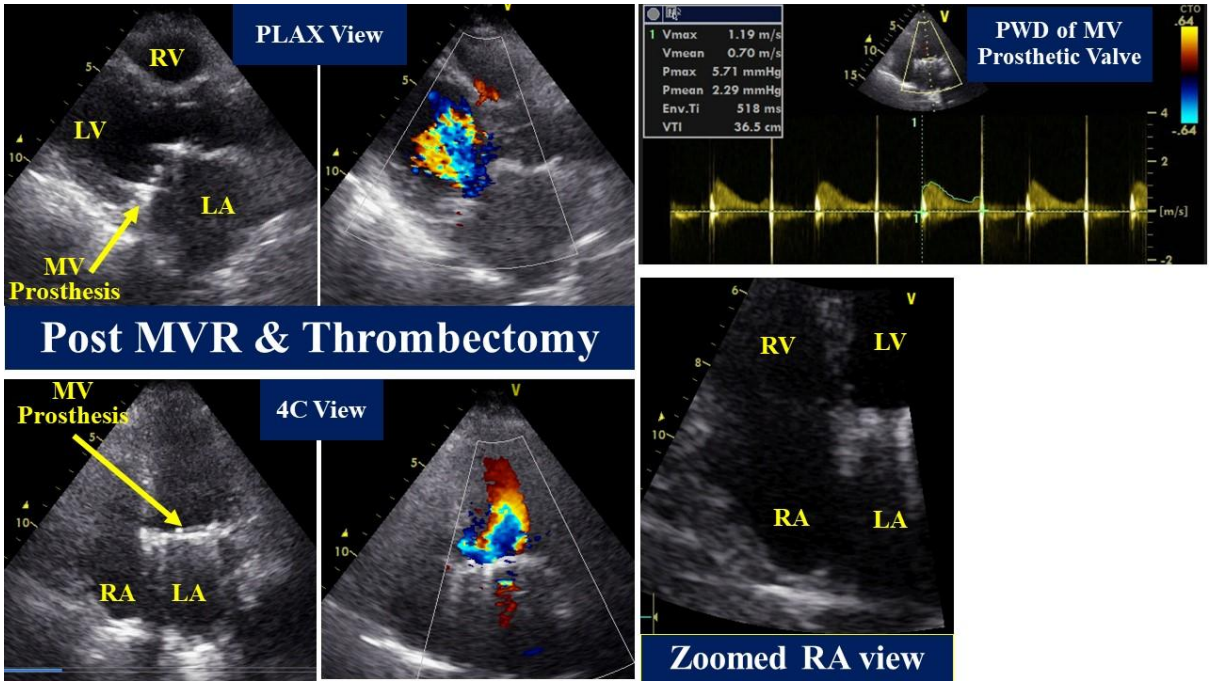

Figure D: Post Procedural 2D Echocardiographic Stills

Post Procedure 2D Echocardiography showed S/P MVR-Normally functioning MV prosthesis, PPG / MPG was 12/3 mm of Hg, Mild Valvular leak, Mild LV Hypokinesia with Mild LV Systolic Dysfunction EF-40%, No PE or Vegetations.