Percutaneous balloon mitral valvotomy and <mark>percutaneous coronary intervention</mark> of left anterior descending artery <mark>in combined procedure</mark>

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6 Abstract: In countries like India rheumatic heart disease (RHD) is still a common problem, 7 and with improvements in diagnosis and treatment, the lifespan of these patients is increased. With increase in the lifespan, these patients may develop coronary artery disease (CAD) and 8 9 present as acute coronary syndrome (ACS). In some cases especially RHD with severe mitral stenosis (MS), thrombus that develop in left atrium may embolize in one of the coronary 10 arteries, leading to acute coronary syndrome. We report a case of 51 year old female who was 11 a known case of rheumatic heart disease and now presented with acute coronary syndrome. 12 13 Patient was hemodynamically unstable and underwent percutaneous balloon mitral valvotomy (PBMV) and percutaneous coronary intervention (PCI) simultaneously. We here 14 discuss the possible complications that need to be addressed in such scenario and how can we 15 approach such cases. This is first of such intervention at our institute and also there are very 16 few such records available online. Patient tolerated the procedure well with significant 17 clinical improvement. 18

Keywords: Percutaneous Balloon Mitral Valvotomy, Percutaneous Coronary Intervention,
 Severe Mitral Stenosis, Left Anterior Descending Artery Stenosis, Rheumatic Heart Disease.

Abbreviations: RHD: Rheumatic heart Disease; MS: Mitral stenosis; PCI; percutaneous
 coronary intervention; BMV: Balloon Mitral Valvotomy; ACS: Acute coronary Syndrome

Introduction: RHD remains one of the leading cardiac diseases in tropical developing 23 countries like India. Around 25%–30% of all cardiac visits to hospitals are related to RHD¹. 24 Patient with RHD can present with left ventricular (LV) dysfunction due to multiple causes 25 including primary rheumatic myocarditis, secondary to LV remodelling due to altered 26 27 hemodynamics in valvular pathology, cardio-embolic phenomenon involving coronaries, or co-existing coronary artery disease (CAD) itself. Mitral stenosis (MS) is most common 28 valvular pathology in RHD. In countries like India where rheumatic heart disease is still a 29 common problem, with improvements in diagnosis and treatment, the lifespan of these 30 patients is increased. With increase in the lifespan, these patients may develop coronary 31 artery disease and present as acute coronary syndrome. In the study by Lacy et al.², the 32 patients were evaluated for the presence of concurrent CAD (50% or greater occlusion in at 33 34 least one major coronary artery), they found that 31.3% of the patients with MS had CAD and 19% had occlusive CAD, and 36.3% of the patients with Mitral Regurgitation (MR) had 35 CAD and 18% had occlusive CAD, and 58.9% of the patients with Aortic Stenosis (AS) had 36 CAD and 21% had occlusive CAD, and 36.6% of the patients with Aortic Regurgitation (AR) 37 had CAD and 30% had occlusive CAD. In some cases especially RHD with severe MS, 38 thrombus that develop in left atrium may embolize in one of the coronary arteries, leading to 39 acute coronary syndrome. Management of RHD patient with significant valve dysfunction 40

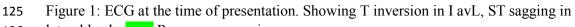
and significant coronary artery disease is primarily revascularisation of affected vessels by
coronary artery bypass graft (CABG) Surgery and valve replacement. In selected patients
with pliable mitral valve and single vessel disease, percutaneous interventions are possible,
like PBMV and PCI. However, there are limited guidelines about management of overtly
symptomatic patients with ACS in cases of RHD who may not tolerate major surgical
intervention.

47 Case Report:

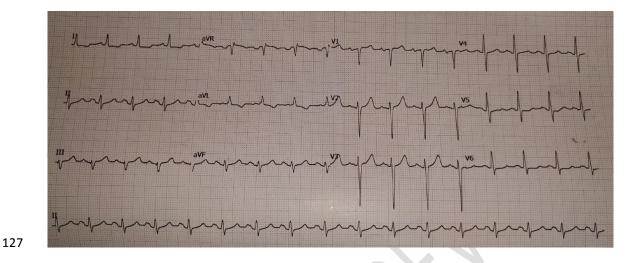
51 year old female, known case of RHD with MS, admitted in peripheral hospital with 48 49 multiple episodes of paroxysmal nocturnal dyspnea in last 1 month. She developed acute onset chest pain and New York Heart Association (NYHA) functional class IV breathlessness 50 a day before she was transferred to our hospital. Patient was transferred to our hospital in the 51 52 night on oxygen support and ionotropic support. On admission- her pulse rate was 110/min, blood pressure was 100/80 mmHg on inotropes and bilateral crepts were present on 53 54 auscultation. Routine blood investigations were normal, arterial blood gas (ABG) showed 55 hypoxia. Her electrocardiogram (ECG) showed sinus rhythm with poor R wave progression with ST segment downsloping depression in I avL V4 V5 V6 and T inversion in I avL (Fig 56 1). Old ECG obtained from the patient's previous records showed normal progression of R 57 wave with no significant ST-T changes (Fig 2). Echocardiography showed severe MS (Mitral 58 valve area (MVA) by planimetry - 0.67 cm², Wilkins score 7/16, MV gradient- 15/10 mmHg) 59 (Fig 3) with Left ventricular ejection fraction (LVEF) 35%- basal, mid, distal, anteroseptal, 60 anterior and anterolateral segments hypokinetic. Troponin T was significantly raised 61 62 (50ng/ml). Patient didn't respond well to the medical management, so she was taken in cath lab for percutaneous balloon mitral valvotomy (PBMV) and urgent coronary angiography 63 64 (CAG). Right femoral venous and arterial access obtained. Pulmonary artery pressure was 65 54/22 mmHg, aortic pressure was 138/80 mmHg, Pulmonary capillary wedge pressure (PCWP) was 23 mmHg (mean) and LV-end diastolic pressure was 12 mmHg (Fig 4). 66 Gradient across mitral valve was 11 (Fig 4). CAG showed LAD (Left Anterior Descending) 67 artery mid segment thrombotic 90% stenosis (Fig 5). In view of the general condition of the 68 69 patient, BMV was planned first, because PCI requires injectable heparin as anticoagulation 70 and transeptal puncture is usually done without giving any anticoagulation. ACCURA balloon No.26 was used and serial inflation of 26 mm was given for 1 sec (Fig 6). Post 71 balloon dilatation gradient reduced to 2 mmHg and the MVA improved to 1.54cm² (Fig 7). 72 Patient was taken up for PCI to LAD. Then LAD was stented with Drug Eluting Stent (DES) 73 74 2.75 x 24 mm after predilatation. Stent was post dilated with NC balloon 2.75 x 13 mm. Post 75 stenting check showed TIMI III flow with no residual lesion (Fig 8). Patient tolerated 76 procedure well. Patient was shifted to post operative monitoring and care. Post procedure 77 patient improved clinically. Over next few days she was off oxygen support and ionotropes 78 were tapered. Gradually she became ambulatory without symptoms during routine activities. 79 Patient was discharged after 5 days of procedure. Patient is asymptomatic on subsequent 80 weekly follow ups in last 1 month.

82 **Discussion:**

In countries like India, patients with RHD are commonly found to have CAD, more often, if 83 presentation is in late middle age or in elderly age group. There is limited data of incidence of 84 primary coronary artery disease in patients of RHD. In study by Jose et al³, the overall 85 prevalence of CAD in RHD patients undergoing valve surgery was 12.2%. In another study, 86 done by Dinesh et al⁴ showed that 9.05% of RHD patients (above 40 years of age) have 87 significant CAD. The Left Anterior Descending (LAD) artery is the most common vessel 88 involved. Acute coronary syndrome in RHD patients can be secondary to cardioembolic 89 phenomenon involving coronaries or due to atherosclerotic disease involving native vessels. 90 91 Acute coronary syndrome secondary to thrombo-embolic phenomenon was reported in 92 studies by Radhakrishnan et al⁵, Niniek Purwaningtyas et al⁶, Cardoz J et al⁷, Ideally transesophagial echocardiography should be done to rule out thrombus in left atrial 93 94 appendage even if the patient is in sinus rhythm. Differentiating these two etiologies, denovo 95 thrombosis or thromboembolic phenomenon, needs intravascular imaging with IVUS 96 (Intravascular Ultrasound) or OCT (Optical Coherence Tomography), either of which was not 97 done in our patient due to hemodynamic instability and financial constraints. Coronary 98 embolism appears to be the most reasonable explanation for acute myocardial infarction with angiographically normal coronary arteries⁸. In our patient, the coronaries were otherwise 99 normal except for a thrombus in mid LAD which makes embolic phenomenon more likely as 100 101 a cause. Transesophageal echocardiography (TEE) was not done initially as patient was 102 hemodynamically unstable. However, after hemodynamic stabilization TEE was done which showed no appendigeal/atrial thrombus in left atrium. Underlying diseases predisposing to 103 coronary emboli includes valvular heart disease (40%), cardiomyopathy (29%), coronary 104 atherosclerosis (16%), chronic atrial fibrillation (24%) and mural thrombi in 18 (33%)⁹. 105 There are limited available records of patients undergoing PCI and BMV in the same setting. 106 107 Patients undergoing PCI needs to be heparinised and loaded with dual antiplatelets and in setting of BMV where septal puncture carries inherent risk of pericardial effusion and cardiac 108 109 tamponade, PCI carries high risk to the patient. Most of the patients with coexisting CAD and 110 RHD are referred for valve replacement with CABG. However, sometimes patients may not 111 be stable enough or willing for major operative intervention. In our patients we first started 112 with BMV as mitral valve was pliable and there was no significant Mitral regurgitation. 113 Patient was heparinised after septal puncture and after echo confirmation of no pericardial effusion. Patient was given loading dose of clopidogrel during the procedure, chewed and 114 115 kept sublingually, although she was on dual antiplatelets for her ischemic event before procedure as well. The overall procedure was done with due care under fluoroscopic 116 guidance and patient stood procedure well. Similar report of BMV and PCI done in same 117 sitting simultaneously done by Paul G J et al¹⁰ was also safe. Paul G J et al¹⁰ also initiated 118 with BMV and later PCI was done. With our experience and limited available online records 119 120 it is understood that patients with ACS and RHD can undergo both transluminal valvular and 121 coronary intervention in the simultaneously with acceptable safety under adequate caution 122 and care.

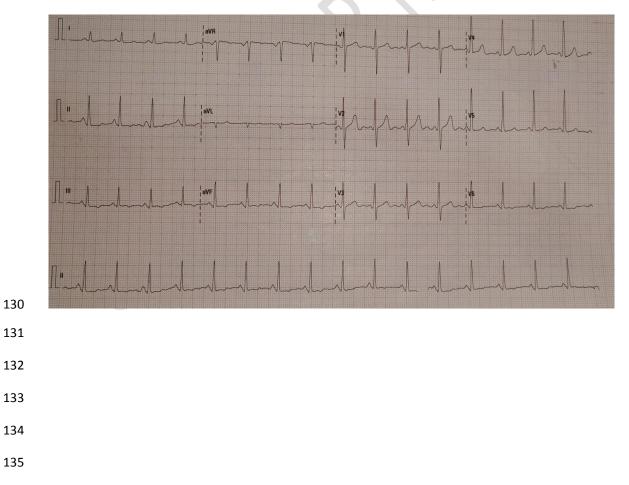


126 lateral leads, poor R wave progression.



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129 Figure 2: ECG from old records of patient. No significant ST-T changes and sinus rhythm.



137 Figure 3: ECHO image showing pre BMV MVA of 0.67 cm^2



- 140 Figure 4: Pressure tracing. Blue represent LV pressures and red represents PCWP (LA
- 141 pressure).



150 Figure 5: CAG showing LAD mid thrombotic lesion causing significant stenosis.

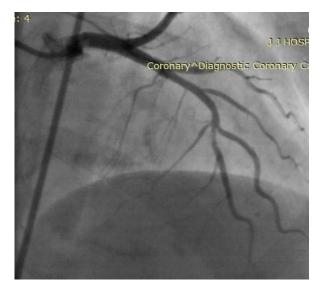
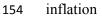


Figure 6: Balloon inflation with ACCURA Balloon No. 26 placed across mitral valve and its





161 Figure 7: ECHO image showing post BMV MVA of 1.54 cm^2



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164 Figure 8: CAG post PCI with drug eluting stent in mid LAD. Good result. TIMI III flow



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167 Conclusion:

- 168 In selected patients with pliable mitral valve and single vessel disease presenting with ACS
- 169 who are hemodynamically unstable, percutaneous intervention with BMV and PCI may be
- done to stabilize the patient hemodynamically and symptomatically.

171 Consent Disclaimer:

- 172 As per international standard or university standard, patient's consent has been collected and
- 173 preserved by the authors.

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References:

177	1.	Padmavati S. Rheumatic fever and rheumatic heart disease in India at the turn of the
178		century. Indian Heart J 2001;53:35-7.
179	2.	Lacy J, Godin R, McMartin D: Coronary Atherosclerosis in Valvular Heart Disease,
180		The annals of thoracic surgery. The Annals of Thoracic Surgery. 1977, 23: 429-435.
181		10.1016/S0003-4975(10)64162-8.
182	3.	Jose VJ, Gupta SN, Joseph G, Chandy ST, George OK, Pati PK, et al. Prevalence of
183		coronary artery disease in patients with rheumatic heart disease in the current era.
184		Indian Heart J 2004;56:129-31
185	4.	Choudhary D. et al (2016) "Prevalence of coronary artery disease in rheumatic heart
186		disease and comparison of demographic and coronary artery disease profile with
187		atherosclerotic coronary artery disease", Advances in human biology 2016; 6:76-83.
188	5.	Radhakrishnan S, Alagesan M, Kaliappan T, Gopalan R. Therapeutic dilemma –
189		Acute coronary syndrome in the presence of severe mitral stenosis. JICC. 2014; 4(2):
190		128-131
191	6.	Niniek Purwaningtyas. Acute Myocardial Infarction in Patient with Mitral Stenosis: A
192		Rare Case J Cardiovasc Dis Diagn 2018;6(5):333
193	7.	Cardoz J, Jayaprakash K, George R .Mitral stenosis and acute ST elevation
194		myocardial infarction. Proc (Bayl Univ Med Cent).2015;28(2):207-209
195	8.	Roberts WC. Coronary embolism: a review of causes, consequences and diagnostic
196		considerations. Cardiovasc Med. 1978;3(7):699-710.
197	9.	Prizel KR, Hutchins GM, Bulkley BH. Coronary artery embolism and myocardial
198		infarction. Ann Intern Med. 1978;88(2):155–161.
199	10	Paul GJ, Elangovan C, Gnanavelu G. Percutaneous transvenous mitral
200		commissurotomy and coronary intervention in kyphoscoliosis. IHJ interventions.
201		2018; 1(2):151-154.