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Acute severe mitral regurgitation with cardiogenic shock following balloon mitral valvuloplasty: echocardiographic findings and outcomes following surgery

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Abstract

Acute severe mitral regurgitation (MR) is the commonest indication for emergency surgery following a balloon mitral valvuloplasty (BMV). It results in hemodynamic compromise with cardiogenic shock and or acute pulmonary edema. These patients deteriorate fast and often require respiratory and critical care support, followed by urgent mitral valve replacement (MVR). We analyzed the data of 1224 BMV procedures done over the 18-year period. We had 85 patients (6.9%) with acute severe MR and cardiogenic shock. The clinical profile, echocardiographic features and operative findings were studied. The echocardiography scores were compared for association with occurrence of MR. The immediate and long-term clinical outcomes of these acutely sick patients were studied. Of the 85 patients, 84 underwent MVR. Anterior mitral leaflet tear was observed in 65 (75%) cases, para-commissural with annular tear in 8 (9.4%), Chordal injury in 7 (8%) and torn posterior leaflet in 5 (5.8%). We documented severe MR in 88 patients (7.1%), with 85 (6.9%) among them developing features of cardiogenic shock. None of the echocardiographic scoring systems were predictive of the occurrence of MR. The 30-day mortality was 4.7%. The mean clinical follow-up period after discharge was 9.3 ± 0.9 years (range 2.2–17.8) with no late mortality. Acute severe MR had an incidence of 7% in this study. Injury to the anterior mitral leaflet was the commonest cause. The long-term outcomes were good with timely intervention and valve replacement surgery despite the fact that the majority (96%) presented with cardiogenic shock. None of the present valve scoring systems could predict the occurrence of severe MR.

Keywords Mitral stenosis · Mitral valvuloplasty · Mitral regurgitation · Cardiogenic shock

Introduction

Acute severe mitral regurgitation (MR) is the commonest indication for emergency surgery following a balloon mitral valvuloplasty (BMV) [1, 2]. The incidence of severe MR following BMV has been reported between 0.9 and 7.5% in various small and large series of registry of cases published [1–3]. Acute severe MR often results in hemodynamic disturbances with hypotension and cardiogenic shock with or without pulmonary edema [1, 3, 4]. These patients deteriorate fast and often require urgent surgical mitral valve replacement (MVR) [5, 6].

The structure of the mitral valve apparatus is a complex one and BMV can sustain injury to different components of the valve apparatus, i.e., valve leaflets, annulus, chordae and papillary muscles [5–9]. Mitral valve pathology in rheumatic heart disease has been stratified by different classification systems [3, 4, 9–14] for selecting patients for BMV depending on the extent of involvement of these components. The Wilkins system of classification is a popular one in this regard [3, 4, 8]. Previous studies have shown Wilkins score to have good predictive accuracy for success of the procedure (in terms of commissural spilt and improved valve areas), but seems to be a poor predictor of the occurrence of MR [1, 7, 11, 15]. Alternative scoring systems Such as the Padial score [11] proposed by the Massachusetts group which gives more weightage to valve thickening and commissural calcium and the Nobuyoshi score [12] proposed

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from Japan were shown to predict the occurrence of MR. However, no scoring system is perfect [6, 7, 9]. We studied the echocardiographic, operative findings and outcomes following the occurrence of acute severe MR with cardiogenic shock following BMV in our institution in the last 18 years.

Methods

The study involved patients in whom acute MR and cardiogenic shock resulted from BMV. The study design was retrospective and data involving patients who underwent treatment between January 2000 and July 2017 were included. BMV was performed in symptomatic patients with moderate to severe mitral stenosis (MS) (mitral valve area ≤ 1.5 cm^2) during this period. Patients were enrolled after BMV if they developed new onset severe MR with clinical features consistent with cardiogenic shock. The occurrence of a significant degree of acute MR was confirmed by echocardiography or left ventricular angiography. Cardiogenic shock in this study was defined as systolic blood pressure of < 90 mmHg with or without pulmonary edema and associated with clinical signs of hypoperfusion such as tachycardia, altered sensorium, decreased urine output and cool extremities. The presence of more than grade 2 (moderate or severe) MR at baseline, dense bi-commissural calcification of the mitral valve and left atrial (LA) clot excluded patients from BMV as per the existing hospital protocol. In borderline cases, the decision to proceed with intervention was made after the heart team's decision. Informed written consent was obtained in all the cases. Data analyzed included baseline characteristics, mitral valve morphology by transthoracic echocardiography prior to BMV, pre-operative clinical profile, operative findings and surgical outcomes. Patients in atrial fibrillation or recent ischemic neurological events underwent transesophageal echocardiography (TEE). Other indications for TEE were the presence of LA thrombus on transthoracic echocardiography and suboptimal transthoracic imaging. All cases of acute severe MR with features consistent with cardiogenic shock after BMV were included for analysis.

Echocardiography

All patients underwent a detailed pre-procedure transthoracic echocardiography for assessment of the mitral valve anatomy, calcification, degree of sub valve disease and associated MR. All patients with baseline moderate degree or more MR were excluded as explained before. MS was defined to be intervened according to the mitral valve area (MVA). A valve area of (≤ 1.5 cm^2) was defined as severe and required intervention as per the existing hospital protocol. MVA was assessed with planimetry and pressure half

time (PHT). A PHT value ≥ 150 ms was defined as indicative of severe MS. A mean gradient of ≥ 10 mmHg was considered severe MS. All 4 components of the Wilkin's score were identified and noted in all cases as a standard measure. In addition, Padial score and Nobuyoshi score were calculated post hoc for all patients who developed MR for comparative analysis from the recorded images. All patients underwent echocardiography immediately after BMV to assess the success of the procedure and severity of MR. Success of BMV was quantified by Doppler measurement of transvalvular gradients and estimation of valve area by the pressure half-time method and planimetry. MR severity was evaluated by integrating data from the color flow image. Color flow occupancy $\geq 40\%$ of the left atrium, any wall hugging swirl, analysis of the vena contracta (≥ 0.7 cm), and the presence of the pulmonary venous systolic reflux were determinants of severity. The continuous-wave doppler tricuspid regurgitation jet velocity was used to determine the systolic pulmonary artery pressure (SPAP) using the simplified Bernoulli equation assigning a value of 10 or 20 mmHg to account for right atrial pressure considering inferior vena caval diameters. All results were based on the average of three measurements for patients in sinus rhythm and five measurements for patients in atrial fibrillation. All recordings were done with Philips iE 33 echocardiography machine.

Intervention

All valvuloplasty procedures were preceded by hemodynamic measurements of mean left atrial pressures, a and v wave heights, left ventricular systolic and end diastolic pressures, aortic systolic pressure, and peak and mean pulmonary artery pressures. Valve area calculations were done off-line by Gorlins method. The values were repeated after intervention. BMV was done (with Inoue triple lumen balloon from Toray or Accura dual lumen balloon from Vascular Innovations) according to the standard operating protocol. All procedures were done by experienced operators who had done a minimum of 100 BMV procedures. The sizing of the balloon was according to patient's height by the (height in cm/10) + 10 formula. An undersizing of − 1 to − 4 mm was done from the calculated measurement according to the operator's discretion. A successful procedure was defined as 50% reduction in the mean gradient and opening of one or both commissures. Any reduction in the mean pulmonary artery pressure was also documented. The procedure was declared a failure if there was < 50% reduction in the gradient. Severe MR was suspected in the cath lab from persistently elevated LA pressure after BMV with prominent V wave measuring more than 25 mm in LA pressure tracing. This was confirmed by echocardiography, based on the following parameters: large central MR jet (area $\geq 40\%$ of LA

area), a wall impinging jet of any size, swirling into LA and Doppler vena contracta width of ≥ 0.7 cm [8]. The severity of MR was substantiated by left ventricular angiography in cases with suboptimal echocardiography images with elevated V waves and left atrial pressures.

Surgical valve replacement

Patients with acute severe MR with cardiogenic shock were advised mitral valve replacement (MVR). Patients who required respiratory support were immediately intubated and shifted to the surgical room for emergency MVR. Patients in whom a delay in surgery was experienced were shifted to the critical care unit for monitoring before surgery. The waiting period for surgery if any and the reasons were documented. The valve morphology was inspected during surgery and the exact cause of MR ascertained. Operative findings noted include the etiology of MR, subvalvular pathology (scaled from 1 to 4 in severity: 1, none; 2, mild; 3, moderate; 4, severe), and calcification [scaled from 1 to 4 in severity: (1) a single area of calcific speck; (2) few specks confined to leaflet margins; (3) calcification involving mid-portion of the leaflets; (4) diffuse involvement of leaflet tissue]. The posterior chordal apparatus was always attempted to be preserved except in cases of severe subchordal fusion. The anterior mitral leaflet and chord were fully excised before prosthesis implant. MVR was done with standard sternotomy and crystalloid or blood cardioplegia (antegrade or combined antegrade and retrograde). Mitral valve is exposed by the "classic left atrial" approach, wherein the Sondergaard's plane is created near the interatrial groove which allows the left atrial incision to be as close to the interatrial septum as possible, aiding in the wide exposure of the mitral valve. Since all patients had significant sub valve disease, no attempt was done for valve repair. Native valve tissue was excised with possible preservation of the valve leaflet (predominantly mono-leaflet preservation). The decision for mechanical or bio prosthesis was as per patient characteristics and related clinical scenario. Mitral valve anatomy is analyzed for any ruptured chordae or leaflet tear and para-commissural or annular damage which has resulted in severe mitral regurgitation. An incision is made at the 12'o clock position in the anterior mitral leaflet and it is carried on to both commissures either with scissors or knife, leaving 2 mm of leaflet tissue from the annulus. Then the anterior leaflet is excised with underlying chordae and papillary muscle. Usually, the posterior leaflet if already rolled up or pliable is left in place with its chordae. If the annulus is wide enough and the chordae are not fused and shorter, the anterior leaflet chordae is also preserved by splitting the anterior leaflet in the center and reattaching it laterally to either commissure. But in case of a smaller annulus, preservation of leaflet tissue is likely to compromise the size of the valve to be used, resulting in

patient prosthesis mismatch and hence removed. After the valve is excised, a thorough saline wash is given. As the annulus will be mostly thin with not much fibrosis in this setting, multi-braided polyester interrupted horizontal mattress sutures with pledgets on the atrial side are performed on the annulus. Annular repair was done in cases of balloon-induced annular tear. Appropriate sized valve is chosen after matching the annulus with the valve sizer and the sutures are passed through the sewing ring. The valve is lowered on to the annulus level and the sutures are tied. The disc opening is checked especially in case of tilting disc valve whether any preserved chordae are hindering the disc opening or closure, if so the valve is rotated and the disc opening is confirmed.

Statistical methods

The distribution of categorical variables was expressed as frequency and percentages. The continuous variables were expressed as mean with 95% confidence intervals. The comparison of change in hemodynamic variables was carried out by using paired t test. The different etiologies of MR were described in percentages and comparison of different valve scores and etiology of MR was carried out by using Chi square test. Chi square test was used to find out significant associations between discrete variables. A p value of < 0.05 is considered to be significant. Pearson correlation coefficient (r) was used to compare the echocardiographic and surgical findings. The statistical analysis was carried out using IBM PASW statistics (SPSS) Version 19.0.

Results

A total of 1224 BMV procedures were done during the study period in our institution. The procedure was successful in 1057/1224 patients (86%). The mean Wilkin's score was different between success and failure group (9.29 ± 3.2 versus 10.09 ± 2.10, $p = 0.02$). When the individual component scores were analyzed, sub valve score (2.52 ± 0.48 vs. 3.02 ± 0.65) and calcium score were significantly lower [2.02 ± 0.76 vs. 2.87 ± 0.84) $p = 0.026, 0.04$), respectively]. We documented severe MR in 88 patients (7.1%), with 85 (6.9%) among them developing features of cardiogenic shock. The baseline clinical and echocardiographic features of the study population group are as shown in Table 1. The mean age of the study group was 24.62 ± 11.93 years and 76% were females. More than half of the patients were in the New York Heart Association class III at the time of admission. Mean Wilkin's score was 9.71 ± 2.1 and the mean sub valve score was $2.76.3 \pm 0.12$. There was no difference in the baseline characteristics between patients who developed severe MR and those who did not. The mean balloon size in this group ($n = 1136$) was 23.56 ± 2.34 (95% CI

Table 1 Baseline characteristics

Baseline variables	$n=85$
Age in years (mean ± SD) range	24.62 ± 11.93 (19–36)
Female gender (%)	65 (76)
NYHA class	
I	0
II	0
III	15 (18)
IV	70 (82)
Pulmonary artery hypertension (%)	77 (91)
Previous BMV (%)	13 (16)
Atrial fibrillation (%)	12 (14)
Pregnant females, n (%)	2 (2.3)
Mild mitral regurgitation (%)	17 (18.2)
Moderate mitral regurgitation (%)	10 (9.4)
Pulmonary hypertension by echo/cath (%)	76 (90%)
Mitral valve area (mean ± SD) planimetry, 95% CI	0.98 ± 0.12 (0.81–1.02)
Mitral valve area (mean ± SD) pressure half time, 95% CI	1.02 ± 0.45 (0.98–1.43)
Mean gradient (mean ± SD), 95% CI	23.4 ± 5.3 (18.5.8–30.9)
Overall Wilkins score (mean ± SD), 95% CI	9.71 ± 2.1 (7.6–11.7)
Leaflet mobility score (mean ± SD), 95% CI	2.5 ± 0.8 (2.1–3.5)
Valve thickening score (mean ± SD), 95% CI	2.4 ± 1.01 (1.89–3.8)
Valve calcification score (mean ± SD), 95% CI	2.14 ± 0.92 (1.34–3.61)
Sub valve disease score (mean ± SD), 95% CI	2.76 ± 0.12 (1.04–3.78)
Pre-procedure MR grade (mean ± SD)95% CI	1.18 ± 0.34 (0.78–2.01)
Mean LA dimension (cm) (mean ± SD), 95% CI	5.12 ± 0.76 (4.34–6.09)

20.67–26.34) without MR and 24.16 ± 2.45 mm (95% CI 20.8–26.72) with MR ($n=88$) ($p=0.1$).

Immediate clinical and echocardiographic findings

In three patients, the systolic blood pressure was ≥ 90 mmHg and all these patients developed acute pulmonary edema requiring immediate ventilator support. All except one among 85 patients with cardiogenic shock were managed with surgical valve replacement. One patient developed severe bradycardia and asystole on the table, which could not be reverted after resuscitation and respirator support. This patient had baseline severe right ventricular dysfunction and pulmonary hypertension. The clinical profile in our series after development of acute MR with cardiogenic shock was as follows: hypotension in 72 (84%), hypoxia in 33 (38%), orthopnea in 17 (20%) and pulmonary edema in 16 (18%). The echocardiographic findings immediately after developing acute severe MR are given in Table 2. There was significant improvement in valve area in these patients with persistent elevated mean gradient and calculated pulmonary artery pressure. The echocardiographic and operative findings on the etiology of MR are given in Table 3. Anterior mitral leaflet tear was the commonest cause in about 75% of the cases (Figs. 1, 2). In 3 patients without shock, the distribution of

valve damage was as follows (anterior leaflet tear in 2 and chordal rupture in 1). Figure 1a–d shows the echocardiographic and operative images of a case of anterior mitral leaflet tear. Figure 2 shows a case of para-commissural tear extending into the annulus. Figure 3 depicts the echocardiographic and operative images of chordal injury.

The immediate catheterization hemodynamic data in acute MR showed no significant reduction in the mitral valve gradient and the mean left atrial pressure despite significant improvement in valve area (Table 4). The pulmonary artery systolic and mean pressures did not show much change either. There was significant elevation of left atrial v wave height and drop in left ventricular systolic pressures ($p=<0.001$). The different components of mitral valve scores were analyzed in patients who developed severe MR. A comparative analysis was done in patients who developed severe MR and those who developed mild or moderate MR. The results are shown in Table 5. The mean sub valve score and calcification scores were also not different in patients with severe MR. Comparison of different echocardiographic scoring systems and components of scores in different etiologies of severe MR in this study are shown in Table 6. There was no difference in the distribution of various score components with respect to the etiology of MR. The mean balloon diameter was also not different. None of the clinical and echocardiographic parameters such as the total Wilkins/
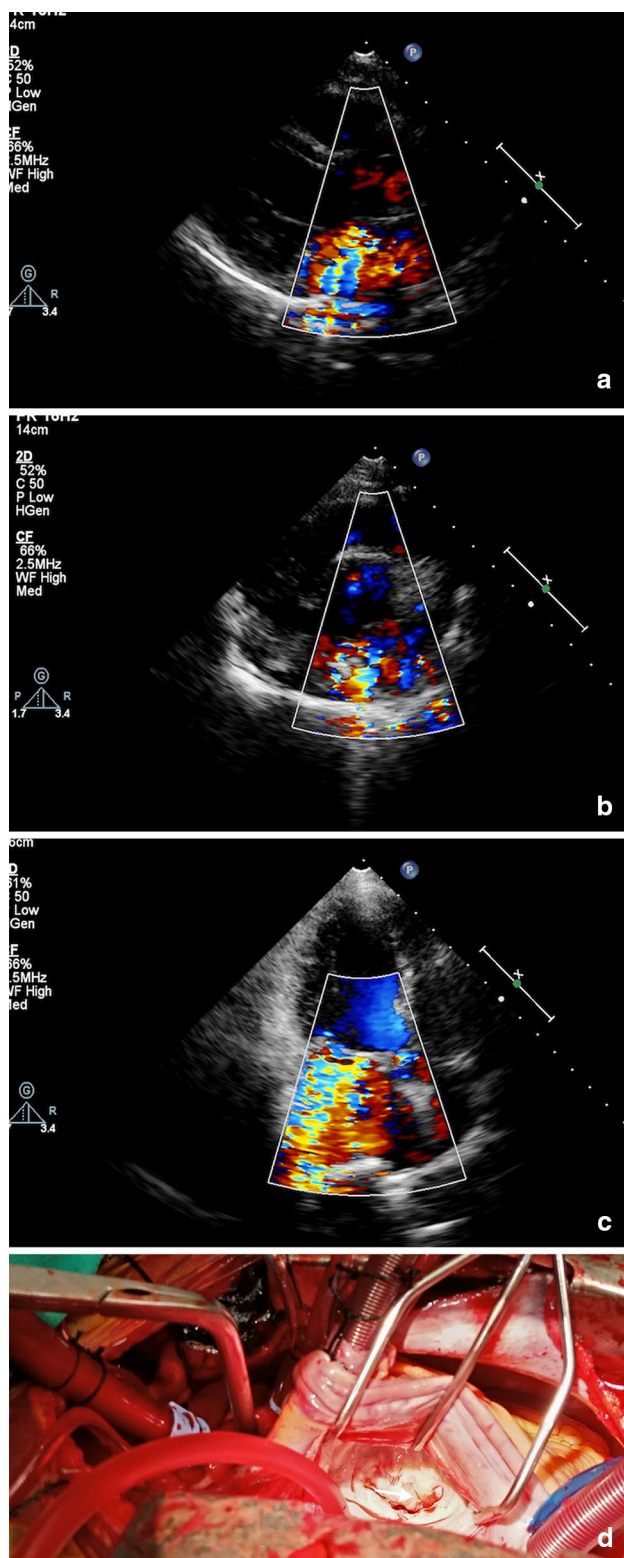
Fig. 1 Echocardiographic and operative images of torn anterior mitral leaflet after balloon mitral valvuloplasty. **a** Long axis para sternal trans thoracic echo image showing severe mitral regurgitation. **b** Short axis para sternal view showing valve leak from the torn middle portion of anterior leaflet. **c** Apical view showing severe regurgitation occupying > 40% of left atrium. **d** Surgical view from opened left atrium showing torn anterior mitral leaflet. Arrow points to the injured leaflet
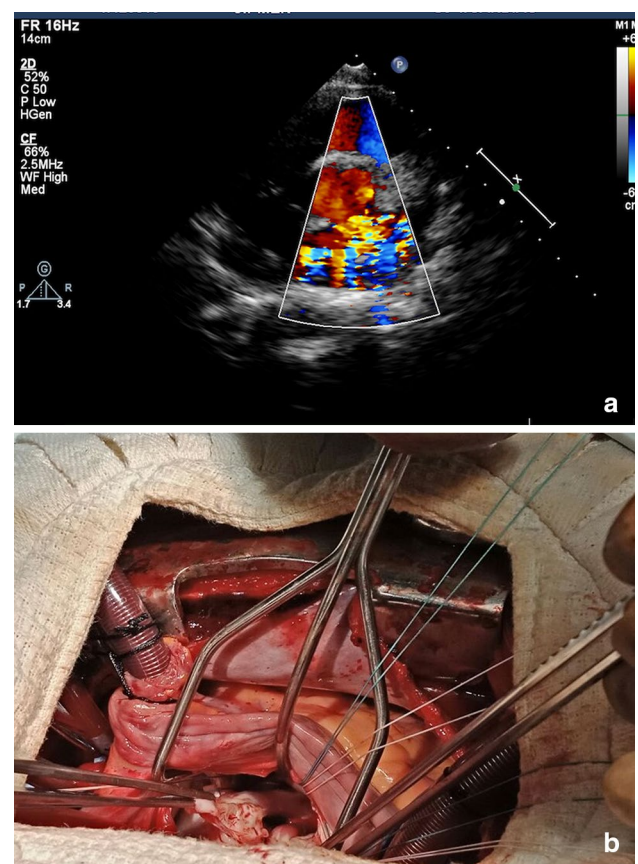

Fig. 2 Images of Para commissural tear with severe mitral regurgitation. **a** Short axis para sternal echo view showing severe MR from damage to the base of anterior mitral leaflet and lateral commissure. **b** Surgical view from left atrial side showing torn para commissural annular tissue

Padial/Nobuyoshi scores, sub valve score or calcification scores were predictive of severe MR.

Surgical treatment

The time interval to surgery for these cases ranged from 6 to 43 h (mean 12.8 ± 3.4 h). All patients underwent urgent mitral valve replacement under cardiopulmonary bypass. The correlation coefficient for echocardiography and surgical findings for etiology of MR was excellent ($r = 0.976$). All patients received metallic prosthesis.

Post-operative period

The mean duration of intensive care unit stay was 3.04 ± 1.32 days (range 2–5 days); the mean hospital stay was 9.12 ± 3.47 days (range 5–12 days). Five patients required hemodialysis support for delayed recovery of renal function. There were 3 cases of in-hospital post-operative deaths (3.6%). The cause for mortality was multiorgan
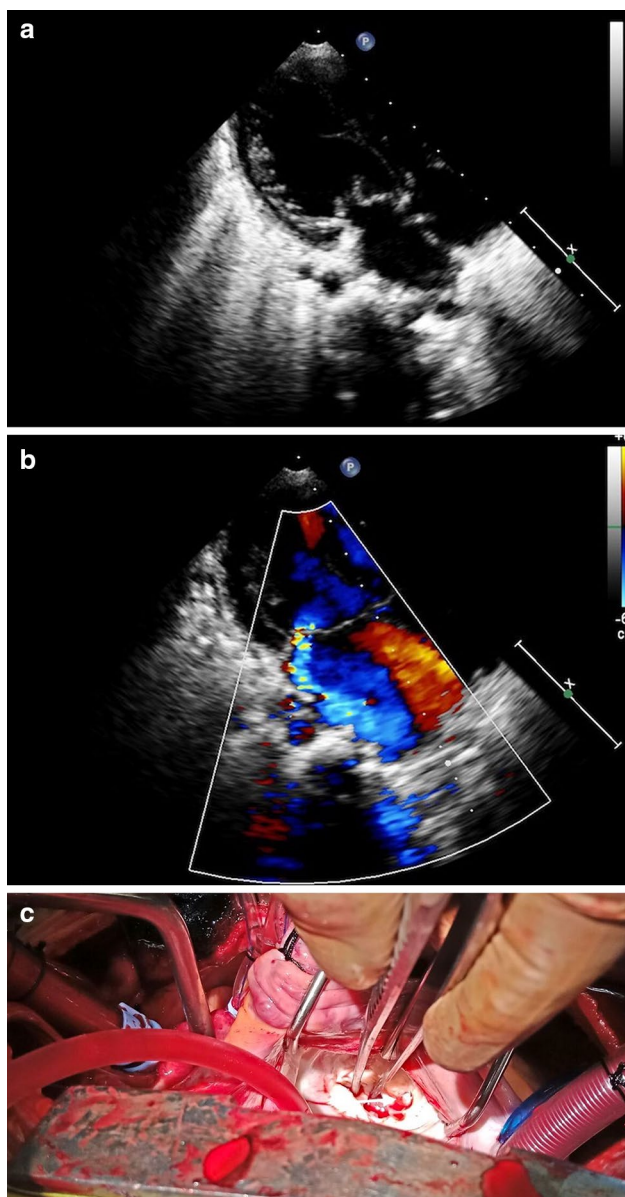
Fig. 3 Chordal injury after BMV. a Long axis echocardiographic view showing flail AML and prolapsing leaflet after anterior chordal injury. b Long axis echo image showing eccentric posterior wall swirling jet of MR. c Operative image showing torn chordae and flail leaflet in the same patient. Arrow points to the ruptured chordal head

dysfunction syndrome in two cases and bleeding with dissemination coagulopathy in one case. There was no statistically significant association between any clinical variable or Wilkins score and in-hospital mortality ($p = 0.653$).

Long-term follow-up

The mean clinical follow-up period after discharge was 9.3 ± 0.9 years (range 2.2–17.8). There was no late mortality recorded for any of these patients. All patients were given

infective endocarditis prophylaxis. Patients with mechanical valve were treated with acenocoumarol and monitored for prothrombin time (INR) every month. MVR resulted in event-free survival in 80/81 (98%) patients. One patient had prosthetic valve thrombosis in the first year of follow-up successfully managed with thrombolysis with streptokinase. The echocardiographic follow-up showed good functional prosthesis at 5-year follow-up.

Discussion

We present herewith our experience on acute severe MR following BMV of patients who were in cardiogenic shock. We had very sick acutely ill patients in this group who had hemodynamic compromise after the intervention. The heart team and critical care unit were put on emergency code for early respiratory support and urgent surgery. The majority of patients (85/88) had shock as the initial presentation after developing severe MR with balloon-related injury to the valve apparatus. Generally, with acute MR, left atrial and pulmonary capillary wedge pressures increase abruptly because of the noncompliant left atrium, and pulmonary edema develop [5–7]. In previously stenosed mitral valve with rheumatic involvement, the chronically elevated left atrial and pulmonary venous pressures result in elevated pre-capillary chronic pulmonary arteriolar vasoconstriction [7]. This makes pulmonary edema less frequent, and more often patients, after developing severe MR, have clinical presentation of cardiogenic shock [1, 7]. The effective forward stroke volume is decreased here and a portion of the systolic ejection is diverted to the left atrium. Compensatory tachycardia may initially preserve cardiac output, due to reduced effective stroke volume; nonetheless, hypotension, end-organ failure, and features of florid cardiogenic shock eventually develop [1, 7, 8].

We studied the popular Wilkins score parameters and found that the score components were not related to or predictive of the occurrence of MR. The Wilkins score was significantly different in success versus failure groups, but was not able to relate to the occurrence of MR as did other systems of valve assessment. We looked at Padial score and Nobuyoshi scores, as these scores were predictive of MR in the previous studies [11, 12]. But we could not replicate these results in our series. The anterior leaflet tear was the commonest mechanism of severe MR in this study (76%). This was similar to the observation made by Nanjappa et al. [1]. However, in our series there was good correlation between the echocardiography and surgical findings ($r = 0.97$). We had 8 patients documented with paracommissural MR which was severe in nature and associated with annular damage. The annular injury in 7 cases was confirmed during surgery (see Fig. 2b). None of the score

Table 2 Echocardiographic features of mitral valve before and after intervention in acute MR

Variable	Parameter before Mean ± SD 95% CI	Parameter after Mean ± SD 95% CI (post)	*p* value
Mitral valve area cm^2 (echo planimetry)	0.98 ± 0.12 (0.81–1.02)	1.22 ± 0.06 (1.12–1.56)	< 0.001
Mitral valve area cm^2 (echo pressure half time)	0.92 ± 0.06 (0.91–1.23)	1.25 ± 0.10 (1.02–1.43)	< 0.001
Mean valve gradient	23.4 ± 5.3 (18.5.8–30.9)	26.9 ± 15.2 (23.45–33.6)	0.10
Calculate right ventricular systolic pressure	80.3 ± 5.6 (54.34–98.4)	79.45 ± 7.9 (72.1–89.5)	0.32
Degree of mitral regurgitation	1.18 ± 0.34 (0.78–2.01)	3.76 ± 0.47 (3.23–4.12)	< 0.001

The absolute valve area improvement was significant with no change in the mean gradient. The patients had no change in the calculated right ventricular systolic pressures

Table 3 Etiology of mitral regurgitation with cardiogenic shock after BMV (*N* = 85)

Etiology	Echocardiography	Surgical
Anterior mitral valve leaflet body tear (%)	65 (76.4)	64 (75.2)
Para-commissural tear with annular extension (%)	8 (9.4)	7 (8.2)
Chordal/papillary muscle injury (%)	7 (8)	8 (9.4)
Posterior leaflet body tear (%)	5 (5.8)	6 (7)

Table 4 Hemodynamics before and after the development of acute MR (*N* = 85)

Hemodynamic variable	Mean ± SD (95% CI) Before	Mean ± SD (95% CI) After	*p* value[#]
Mean valve gradient	26.7 ± 1.3 (21.8–36.9)	29.6 ± 1.12 (17.6–32.8)	0.1
Mean valve area (cm^2)	1.02 ± 0.34 (0.92–1.23)	1.32 ± 0.54 (1.10–1.67)	< 0.001
Mean left atrial pressure	35.8 ± 3.56 (21.3–49.3)	42.5 ± 4.7 (31.9–52.7)	0.4
Left atrial "v" wave height mean ± SD (95% CI)	38.7 ± 4.7 (34.5–43.6)	49.34 ± 3.7 (43.78–59.09)	< 0.001
Left ventricular systolic pressure	107 ± 4.12 (96.3–128.2)	82.4 ± 7.8 (73.2–109.1)	< 0.001
Pulmonary artery pressure (peak)	77.4 ± 10.4 (48.8–99.4)	76.32 ± 8.1 (62.5–88.2)	0.9
Pulmonary artery pressure (mean)	37.4 ± 13.4 (24.6–48.9)	35.5 ± 9.3 (26.7–49.7)	0.2

The absolute valve area improvement was significant. Concomitant significant raise in left atrial mean pressure and v wave height was noted with no change in the mean gradient. The patients had no change in pulmonary pressures

[#]Paired *t* test

Table 5 Comparison of patients with acute severe MR (*n* = 85) and no/mild or moderate MR (*N* = 1027) after balloon mitral valvuloplasty

	Severe MR (85)	Moderate MR (217)	Mild or no MR (892)[a]	*p* value[#]
Baseline valve gradient (mean ± SD), 95% CI	23.4 ± 5.3 (18.5.8–30.9)	22.98 ± 6.2 (16.57–32.23)	21.34 ± 7.9 (15.78–42.31)	0.78
Baseline pulmonary artery pressure (mean) (mean ± SD), 95% CI	77.4 ± 10.4 (48.8–99.4)	74 ± 8.54 (54.8–91.56)	69.24 ± 2.24 (53.24–87.43)	0.62
Wilkins score (mean ± SD), 95% CI	9.71 ± 2.1 (7.6–11.7)	9.61 ± 1.8 (7.45–10.3)	9.32 ± 1.12 (6.98–12.03)	0.83
Valve thickening score (mean ± SD) 95% CI	2.4 ± 1.01 (1.89–3.8)	2.31 ± 1.10 (1.78–3.45	2.45 ± 1.04 (1.76–3.57)	0.7
Sub valve disease score (mean ± SD), 95% CI	2.76 ± 0.12 (1.04–3.78)	2.54 ± 0.32 (1.23–3.02)	2.63 ± 0.51 (1.32–3.42)	**0.4**
Valve calcification score (mean ± SD), 95% CI	2.74 ± 0.92 (1.34–3.61)	2.69 ± 0.72 (1.21–3.34)	2.59 ± 0.89 (1.32–3.46)	**0.2**
Size of balloon in mm (mean ± SD), 95% CI	24.42 ± 2.4 (20.8–26.72)	24.56 ± 2.1 (20.32–27.10)	24.34 ± 3.1 (22.34–26.2)	0.1

Excluding patients who did have baseline mild MR

Bold values indicate none of the different score parameters were significant

[#]Paired *t* test

Table 6 Comparison of different valve scores in patients with severe MR of various etiologies

	Torn leaflet (AML/PML tear)	Torn chordae	Para-commissural and annular tear	p value[#]
Wilkins total score mean ± SD (95% CI)	9.62 ± 2.78 (7.56–10.89)	9.43 ± 2.67 (8.34–10.56)	9.69 ± 2.39 (8.45–10.75)	0.9
Wilkins valve calcification score[a] (mean ± SD), 95% CI	2.62 ± 0.92 (1.34–3.61)	2.57 ± 0.65 (1.29–2.97)	2.55 ± 0.59 (1.30–3.01)	0.1
Wilkins sub valve score (mean ± SD), 95% CI	2.76 ± 0.12 (1.04–3.78)	2.56 ± 0.45 (1.45–3.02)	2.64 ± 0.63 (1.43–3.02)	0.1
Padial total score mean ± SD (95% CI)	9.42 ± 1.78 (6.96–9.73)	9.45 ± 1.03 (8.21–9.56)	9.69 ± 2.39 (8.45–10.75)	
Padial valve commissural score (mean ± SD), 95% CI	3.01 ± 0.87 (2.45–3.21)	2.96 ± 0.76 (2.37–3.12)	2.98 ± 1.01 (2.32–3.12)	0.1
Nobuyoshi total score mean ± SD (95% CI)	8.96 ± 1.21 (6.78–9.65)	9.02 ± 1.32 (7.01–9.89)	9.13 ± 1.34 (7.89–10.24)	0.1
Nobuyushi valve commissural score (mean ± SD), 95% CI	2.96 ± 0.34 (2.13–3.23)	3.02 ± 0.47 (2.76–3.43)	2.91 ± 0.87 (2.09–3.12)	0.1
Size of balloon in mm (mean ± SD), 95% CI	24.42 ± 2.4 (20.8–26.72)	23.97 ± 2.23 (21.2–25.98)	24.23 ± 2.3 (21.6–26.4)	0.1

Bicommissural calcium was an exclusion criterion in this study

[#]Chi square test

components including the commissural score of the Nobuyoshi and Padial algorithms were significantly different with regard to the etiology of MR in this series. The incidence of severe MR in this study was 7.1%. Registry data published two decades earlier reported this complication in 6–7.5% of patients who underwent BMV, 12.4% by Kim et al. and 17% by Essop et al. In a large series from India comprising 3650 patients, severe MR occurred in 3.3% patients of whom 1.8% required mitral valve replacement [7]. Echocardiography showed leaflet rupture as the most common cause. The development of severe MR following BMV is an unpredictable event, with adverse consequences. There was no relationship between Wilkins score and in-hospital mortality.

The splitting of mitral commissures is the mechanism of increase in mitral valve area following BMV [8, 10, 16, 17]. Rheumatic process does not uniformly affect the mitral valve and areas with least resistance give way during balloon dilation, giving rise to severe MR [9, 15]. Significant submitral pathology may also prevent proper positioning of balloon and this will also lead to mechanical disruption of the valve with MR [10, 15]. Commissural MR is usually mild and of no significance [7, 15], whereas para-commissural MR actually represents damage to the base of the leaflet body with annular extension which usually causes severe MR [2, 11, 12]. Stretching of the mitral valve apparatus during BMV is never a cause of MR, as the size of the annulus never varies before and after BMV. We did not find any difference in balloon diameter with respect to the severity of MR and or different mechanisms of MR and none was statistically significant. However, the mean balloon size was marginally higher with acute MR compared to no MR by about 0.5 mm, which could be a clinically significant factor. Data on the

effect of balloon diameter on mitral regurgitation are conflicting, with some showing no correlation of mitral regurgitation with either absolute effective balloon dilating area or ratio of balloon dilating area to body surface area [13, 14].

The long-term outcomes following emergency valve replacement were good in this series, with no late mortality. This underscores the fact that early identification and surgical replacement is crucial even with severely ill patients with shock.

Limitations of the study

This is a retrospective case series from a single center. Three-dimensional (3D) echo was not part of the assessment. The 3D protocol was started in the last 5 years and the protocol for mitral valve assessment for BMV is still evolving in our hospital.

Conclusions

Acute severe MR had an incidence of 7% in this study. Injury to anterior mitral leaflet was the commonest cause of balloon-related injury. The long-term outcomes were good with timely intervention and valve replacement surgery despite the fact that the majority (96%) present with cardiogenic shock. None of the present valve scoring systems could predict the occurrence of severe MR.

Compliance with ethical standards

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