PERCUTANEOUS TRANSVENOUS BALLOON MITRAL VALVULOPLASTY: MID – TERM RESULTS IN ADOLESCENTS*

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Six patients with mitral valve stenosis underwent percutaneous balloon mitral valvuloplasty (PBMV) in our department between November 1992 and December 1997. Five patients had rheumatic mitral valve stenosis and one had congenital mitral valve stenosis and Eisenmenger's syndrome with patent ductus arteriosus (PDA). Functional status before PBMV was class IV in two patients, class III in two patients, and class II-III in two patients, as classified by the New York Heart Association (NYHA).

The mean diastolic pressure gradient across the mitral valve measured during heart catheterization before and immediately after PBMV was 18.8 ± 10.42 and 9.4 ± 7.7 mmHg, respectively ($p < 0.01$). The patients were followed for a mean period of 36.6 ± 8.5 months (range 12 to 72 months) after the procedure. During follow-up, post PBMV mean diastolic transmitral gradient measured by color Doppler echocardiography decreased from 19.3 ± 11.16 to 7.43 ± 7.3 mmHg ($p < 0.01$) and the mitral valve area increased from 1.09 ± 0.7 to 3.1 ± 0.9 cm^2 ($p < 0.002$). Functional capacly showed improvement to NYHA class I in four patients, to class II-III in the paient with congenital mitral valve stenosis and Eisenmenger's syndrome with PDA and to class II in one patient with severe mitral valve calcification in whom restenosis occurred three years after PBMV.

Percutaneous balloon mitral valvuloplasty PBMV can achieve very good short- and mid-term results in relieving symptomatic rheumatic mitral valve stenosis.
Key words: mitral stenosis, percutaneous balloon mitral valvuloplasty.

Stenotic mitral valve diseases are relatively uncommon in childhood. Congenital mitral valve stenosis accounts for 0.2 percent of all congenital heart diseases. Mitral stenosis is mostly secondary to deformity of mitral valve leaflets and chordae tendineae following recovery from rheumatic pancarditis. The most common finding is commissural union with increased fibrosis in leaflets.

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Calcifications may be found together with fusion and fibrosis. Not uncommonly, mitral insufficiency may accompany these valvular degenerative changes. Although mitral valve stenosis may occur as early as two or three years after an attack of acute rheumatic fever, a patient usually lives 10-20 asymptomatic years after the first rheumatic attack. Sometimes patients with severe mitral valve stenosis develop early symptoms[1-3].

After the appearance of symptoms related to mitral valve stenosis (New York Heart Association [NYHA] functional capacity class II), the disease follows a rapid downhill course. Symptomatic rheumatic mitral stenosis requires various interventions. Basically, two different approaches are available for palliation of mitral stenosis: surgical mitral commissurotomy using either cardiopulmonary bypass (open) or transvalvar dilation (closed), or percutaneous catheter balloon mitral valvuloplasty (PBMV)[2]. Percutaneous mitral valvuloplasty is of particular importance. PBMV is increasingly being performed more often for the treatment of acquired isolated mitral valve stenosis as an altrenative to surgical closed mitral commissurotomy[4-7]. Previous studies have demonstrated marked immediate improvement in hemodynamic measures and symptoms after PBMV[8-10], even in patients with calcification of the mitral valve[8,11] or severe pulmonary hypertension[8,11,12]. Although most of these studies are from elderly patients, PBMV has also emerged as a viable option in childhood, especially in adolescents. In this study, we present the short- and mid-term results of the first six patients in our department who underwent PBMV for mitral stenosis.

Material and Methods

Between November 1992 and December 1997, six patients (3 girls and 3 boys) aged 14 ± 2.8 years (range 10 to 17 years) underwent PBMV in our department. PBMV candidates were selected based on history, physical examination, ECG, chest x-ray and echocardiographic findings. Five patients had rheumatic mitral valve stenosis, one patient (Case 5) had congenital mitral valve stenosis (hypoplastic mitral valve with asymmetric papillary muscle) with patent ductus arteriosus (PDA) and Eisenmenger's syndrome (Table I). Two patients were in NYHA functional class I-III, two were in class III and two in class IV (Table II). Echocardiographic evaluations were done with Acuson 128 XP with 3-5 MHz transducers. Two-dimensional (2-D) and color Doppler echocardiography were performed 24 hours before valvuloplasty to exclude the presence of left atrial thrombus, to determine the degree of mitral regurgitation and mitral valve area, and to evaluate valvular structure. Echo score of each paient was determined with 2-D echocardiography considering valve thickness, calcification, mobility, and subvalvular apparatus[13]. Patients were selected according to the following criteria[12]: i) severe symptomatic mitral valve stenosis (NYHA clases ≥ II), mitral valve area ≤ 1 cm^2; ii) no previous history of thromboembolic complications; iii) having sinus rhythm; iv) mitral regurgitation ≤ 1st degree; v) no detectable left atrial thrombi on 2-D echocardiography; and vi) an echo score < 8. Cardiac catheterization and PBMV were performed simultaneously during the same study.

Table I: Demographic Features of Patients and Etiologies of Mitral Valve Stenosis

N	Age (Years)	Sex	Etiology
1	17	F	RF*
2	14	M	RF
3	12	F	RF
4	17	M	RF
5	10	F	Congenital
6	14	M	RF
Mean ± SD	14 ± 2.8		

* RF: Rheumatic fever.

Table II: Functional and Hemodynamic Status Before and After PBMV[1]

	Before PBMV					After PBMV				
N	FS[2]	MVA[3]	MG[4]	LAP[5]	PVR[6]	FS	MVA	MG	LAP	PVR
1	IV	2.0	12.2	26	4.66	I	3	2.7	17	2.09
2	II-III	0.5	7.4	25	23.64	I	3.8	7.3	17	18.22
3	II-III	0.5	25	20	7.73	I	2.06	3.7	12	4.63
4	III	1.7	10	15	2.35	I	3.7	0.9	8	1.65
5	IV	0.25	36	28	14.62	II-III	1.53	21.2	15	14.15
6	III	1.6	25	35	2.79	II	3.7	8.4	20	2.79
Mean±SD		1.09±0.7	19.3±11.16	24.8±6.8	9.30±8.35		3.17±0.97	7.43±7.3	14.83±4.26	7.26±7.11
						p	.002	.001	.001	.06

1 PBMV : Percutaneous balloon mitral valvuloplasty.
2 FS : Functional status (NYHA class)
3 MVA : Mitral valve area (cm^2)
4 MG : Transmitral valve mean gradient (mmHg)
5 LAP : Left atrial mean pressure (mmHg)
6 PVR : Pulmonary vascular resistance (Wood unit)

After premedication with lytic cocktail (demerol, Phenergan, Thorazine), or after sedation with ketamine and midazolam, diagnostic catheterization was performed. Serial oxygen and hemodynamic measurements and left and right ventricular angiograms were obtained before and after the valvuloplasty procedure. All patients were anticoagulated with heparin during catheterization. Dilation of the mitral valve was performed by the transvenous transseptal approach using properly sized monofoil or trefoil single balloon. After the diagnostic study, a Mullins sheath was inserted over the guide wire to the right atrium, and a Brockenbrough needle was used to make a transseptal puncture in the interatrial septum. After introducing the needle to the left atrium, confirmation was done by monitoring high pressure in the left atrium, and a back-up guide wire was then inserted in the left atrium. The interatrial septum was dilated with a 5-6 mm single balloon and the Mullins sheath was safely placed in the left atirum. A flow-directed balloon-tipped catheter was sent to the left ventricle through the mitral valve. A 260 cm x 1.10 cm J guide wire was placed at the apex of the left ventricle. The balloon-tipped catheter and Mullins sheath were then removed. A mitral valvuloplasty balloon of appropriate size for body surface area was positioned in the valve and inflated with diluted contrast media either by means of an indeflator at 4-6 atmosphere pressure or manually until the impression of the commissures disappeared (or for a maximum of 30 seconds). This procedure was repeated three times. Post procedure pressure tracings and left ventricular contrast injection for mitral regurgitation were obtained.

The clinical and echocardiographic findings were evaluated one day, two weeks and six months after PBMV (Figs. 1, 2). NYHA functional class was assessed

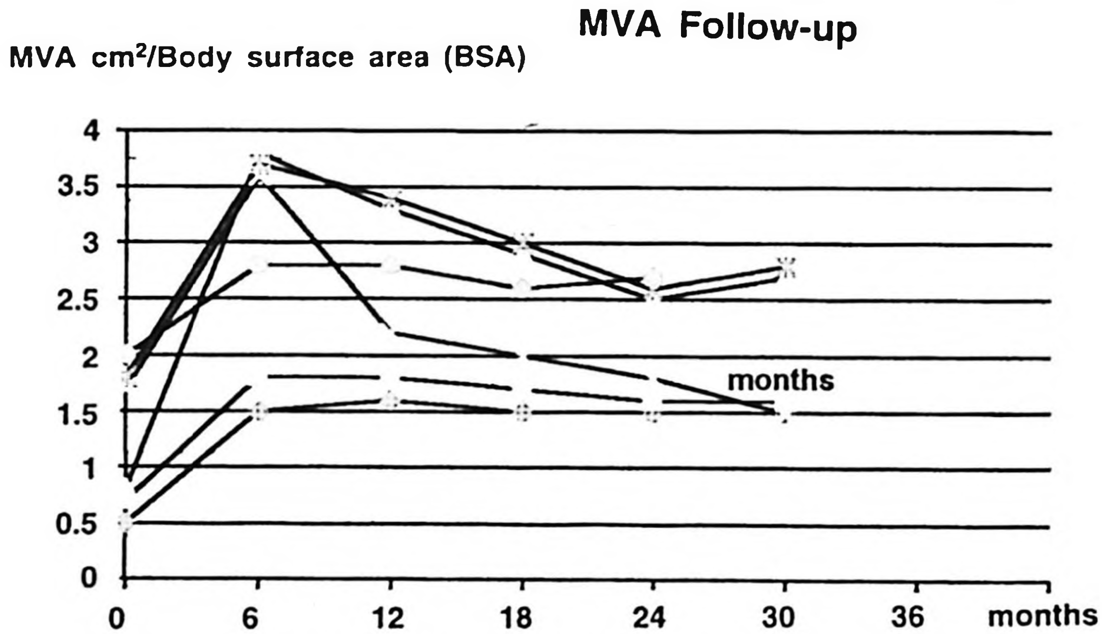


Fig. 1: Mitral valve area follow-up.

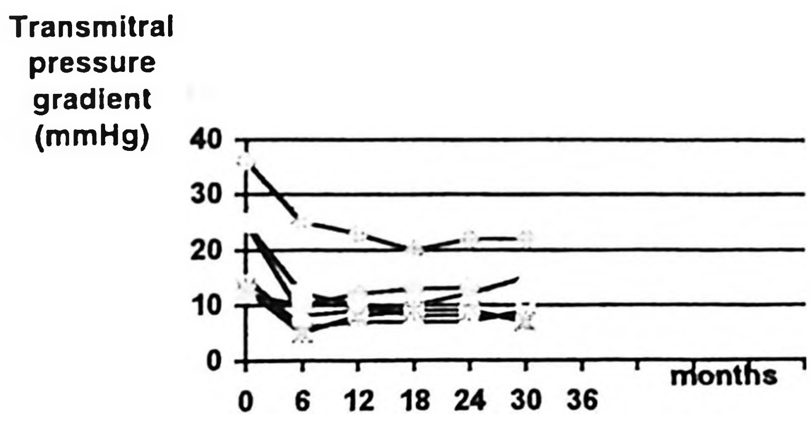
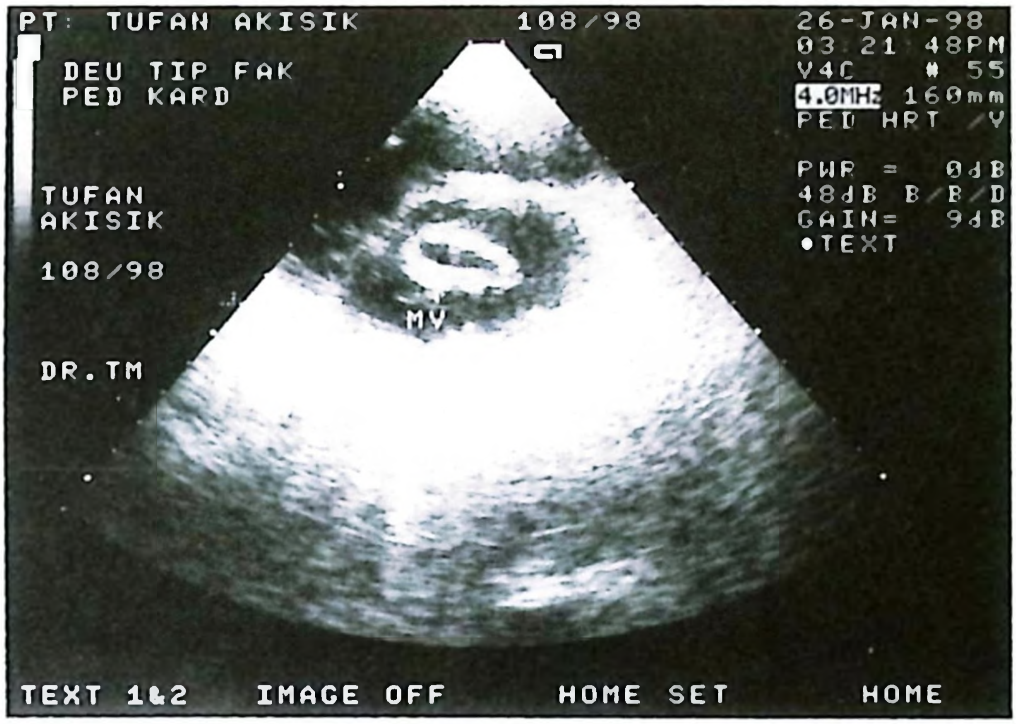
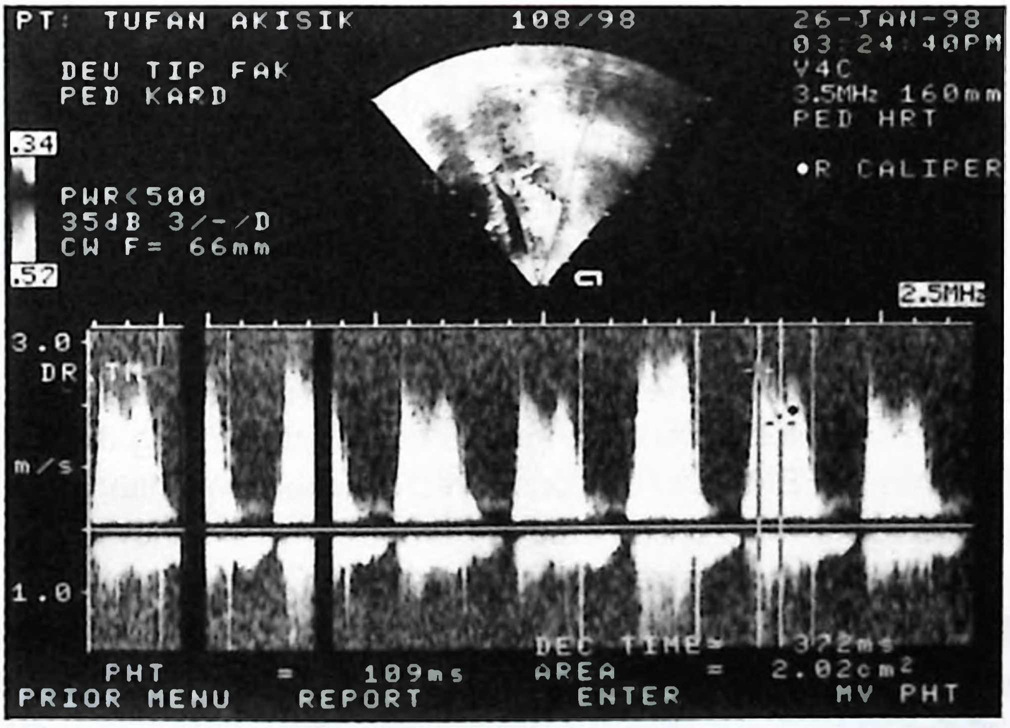


Fig. 2: Transmitral gradients measured by echocardiography.

and a complete echocardiographic study was carried out, including mitral valve area calculations using planimetric and pressure half time (PHT) methods (Fig. 3 a, b), presence and degree of mitral valve regurgitation, subvalvar and mitral valve abnormalities and left atrial thrombus formation.



(a)



(b)

Fig. 3 a, b: Mitral valve area calculations using planimetric and pressure half time (PHT) methods.

Wilcoxon rank sum test in SPSS software was used for statistical analysis. All data were expressed as mean ± 2 SD.

Results

Before PBMV, four of the patients with rheumatic mitral stenosis had mobile, pilable mitral valves. In one patient (Case 6), the mitral valve showed severe calcification (Fig. 3a). None of our patients had mitral valve regurgitation or atrial fibrilation. PBMV was performed without complications in all candidates and the patients were discharged 48 hours following the procedure.

After PBMV, cardiac catheterization findings revealed a decrease in the mean transmitral pressure gradient from 18.8 ± 10.42 mmHg to 9.4 ± 7.7 mmHg ($p < 0.01$), and the left atrial mean pressure decreased from 24.8 ± 6.8 mmHg to 14.8 ± 4.3 mmHg ($p < 0.001$). Pulmonary vascular resistance decreased from 9.3 ± 8.35 Wood units to 7.26 ± 7.11 Wood units ($p < 0.06$). The mitral valve orifice area measured by echocardiography increased from 1.09 ± 0.8 cm^2 to 3.1 ± 0.9 cm^2 ($p < 0.02$) (Table II). The patients were followed for 12-72 months (mean follow-up duration 36.6 ± 8.5 months). In Case 6 with the calcified mitral valve, restenosis developed. Although his functional status did not deteriorate (NYHA class II), a repeat PMBV was performed 36 months later. Functional status in Case 5 with congenital mitral stenosis with PDA and Eisenmenger's syndrome improved from class IV to II-III after PMBV. The remaining four patients (Cases 1-4) who were in NYHA class II-IV before PBMV showed good progress and are currently in NYHA class I.

Discussion

Percutaneous balloon mitral valvuloplasty PBMV was originally performed in 1984 by Inoue et al.[4] Following the first attempts, many other institutions used different techniques for mitral valvuloplasty with single or double balloon[4,11,12,14,15]. These techniques used either the retrograde route from the femoral artery[8,16] or the antegrade route from the femoral vein with transseptal puncture[4-7,17,18]. Insertion of the balloon catheter into the valve without the need for transseptal puncture has also been described[8]. There is no significant superiority of these techniques[8-10,12,15]. We preferred the monofoil or trefoil single balloon method using the transvenous transseptal approach. PBMV can be considered in patients with functional capacity NYHA class-II, a mitral valve orifice area ≤ 1 cm^2/m^2, mitral transvalvular pressure gradient ≥ 10 mmHg, and mitral regurgitation grade 1. Patients with higher degrees of mitral regurgitation are candidates for mitral valve replacement surgery.

Body surface area (BSA) is the main domain in choosing the right balloon. Balloon area/BSA ratio must be 3.5-4. With ratios lower than 3.5 successful results cannot be achieved, and with ratios greater than 4.5 complications are more frequent. Criteria for classification of a successful procedure are complete separation of both commisures (very well), complete separation of one commissure and partial separation of the other (well) complete separation of one commissure with the other still fused (good), both comissure partially separated or still fused (unsuccessful)[19]. According to these criteria, all of our patients were classified very well after PBMV except for the patient with congenital mitral stenosis. PBMV for congenital mitral valve stenosis is a matter of conflict, since the results are often suboptimal. Yet, some authors suggest a PBMV trial in certain forms of congenital mitral valve stenosis[20,21]. The rationale of this opproach in congenital mitral valve stenosis is to provide symptomatic relief and postponement of valve replacement. Because our patient was not eligible for open heart surgery due to Eisenmenger's syndrome with PDA, we performed PBMV.

After PBMV, complications such as restenosis, mitral regurgitation, iatrogenic secundum atrial septal defect (ASD), thromboembolic phenomena, cardiac perforation and tamponade, dysrhythmias, bleeding, vasoocclusion in the femoral artery or vein, cardiac arrest and death have been reported[8,9,12,14,16-19,22,23]. Ventricular dysrhythmias (ventricular premature beats, non-sustained ventricular tachycardia) were recorded in all our patients while the exchange guide wire was being positioned in the left ventricle, but none required treatment. Reported complication rate for mitral regurgitation is 20 percent and for iatrogenic ASD is 63 percent in short-term 20 percent after six months and four percent after one year[8,12,14,19]. All our patients developed minimal mitral regurgitation after PBMV. Four patients had interatrial left-to-right shunt on echocardiography soon after the procedure, but as of three months following PBMV, none has interatrial shunting Christodoulos et al.[8] reported 15.4 percent restenosis at two years follow-up. Should restenosis occur, it is easier, safer and less expensive to redilate with a percutaneously introduced balloon than it is to operate[8]. Only one of our patients, with heavy calcifications, developed restenosis 30 months after PBMV, and a second PBMV was performed successfuly.

Rheumatic mitral stenosis during childhood and adolescence is still not uncommon in developing countries. Adolescents with pure mitral valve stenosis are suitable candidates for PBMV as an alternative for surgery. PBMV should be considered the procedure of first choice for most patients with mitral stenosis, particularly for those whose mitral valve is mobile, pliable and not heavily calcified. PBMV can achieve very good short-term and mid-term results in relieving symptomatic rheumatic mitral stenosis.

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