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ORIGINAL ARTICLE

Initial Results and Long-Term Follow-up of Percutaneous Mitral Valvuloplasty in Patients with Pulmonary Hypertension

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Background Percutaneous balloon mitral valvuloplasty (PMV) is an attractive therapeutic approach in patients with mitral stenosis. The aim of this study was to assess the immediate and long-term clinical, echocardiographic and haemodynamic outcomes of PMV in patients with severe pulmonary hypertension (PAH).

Methods Percutaneous balloon mitral valvuloplasty was performed in 157 consecutive patients; 60 patients (38.2%) had significant PAH defined as baseline pulmonary artery mean pressure (PAMP) $\geq$ 30 mm Hg (Group 1) and 97 patients (61.8%) had PAMP $\leq$ 30 mmHg (Group 2).
Pulmonary artery systolic pressure (PASP), mortality, need for mitral valve replacement or new PMV, and valve restenosis were evaluated during follow-up.

Results Mean age was 44.2 years and 88.5% (139 patients) were women. Primary success was achieved in 79.6% of the patients (125 patients) without differences between the groups.
Mitral valve area increased from 0.90 cm^2 to 1.76 cm^2, PASP fell from 57 mmHg to 35 mmHg in Group 1 and from 38 mmHg to 30 mmHg in Group 2.
Median PASP in Group 1 was 35, 32, 36, 38 and 34 mmHg at 12, 24, 36, 48 and 60 months. There were no significant differences in mitral valve area, PASP and clinical status between the groups.

Conclusion Percutaneous balloon mitral valvuloplasty is a safe and effective technique for the treatment of patients with mitral stenosis and PAH. A significant decrease in pulmonary pressure was observed after valvuloplasty. Although there was a gradual decrease of MVA at long-term follow-up, most patients remained asymptomatic and PASP was stable.

Keywords Valvuloplasty • Mitral valve stenosis • Haemodynamics • Pulmonary hypertension

Introduction

Mitral stenosis (MS) of rheumatic aetiology is still a common valve disease in developing countries [1,2]. It is characterised by a limitation to left atrial emptying which causes a progressive increase in mean left atrial pressure and pulmonary vascular bed pressures with development of pulmonary hypertension (PAH) and increased right ventricular (RV) pressure which influences the clinical manifestations and prognosis of the disease.

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Increased pulmonary arterial pressure is often disproportional to left atrial pressure and is the result of significant increase in pulmonary vascular resistance [3–5].

Surgical decompression of the left atrium, either through mitral commissurotomy or valve replacement generally produces a marked reduction in pulmonary artery pressure [6,7]. However, as surgical mortality in this group of patients is high, percutaneous balloon mitral valvuloplasty (PMV) has emerged as a particularly attractive therapeutic approach in this population [8–12]. Up to the present time, there are a few publications on the long-term clinical outcome and pulmonary artery pressure response after PMV.

The aim of the study was to evaluate the immediate and long-term clinical outcomes and the changes in echocardiographic and haemodynamic parameters of patients with MS with and without PAH undergoing PMV.

Material and Methods

From January 1991 to December 2013, 157 patients with moderate to severe MS who underwent PMV were retrospectively enrolled. Patients were included if they met the following criteria: a) New York Heart Association (NYHA) functional class II or greater, in spite of optimal medical treatment; b) favourable anatomy by echocardiography (those patients with an unfavourable echocardiographic score were considered candidates for PMV according to their personal risk/benefit ratio); c) absence of contraindications for transseptal catheterisation; d) lack of grade $\geq$ II mitral regurgitation according to Sellers classification [13], and absence of concomitant valve disease requiring surgical treatment.

All patients gave their written informed consent to undergo the procedure and the study protocol. The study was conducted following the recommendations of the Declaration of Helsinki and was approved by the Committee on Ethics of our institution.

All the patients underwent transthoracic echocardiography before PMV, to evaluate mitral valve anatomy according to the Wilkins scoring system [14]. The mitral valve area (MVA) was determined by the pressure half-time method described by Hatle et al. [15] In patients with atrial fibrillation five cycles were averaged. After measuring the maximal velocity of the tricuspid regurgitation jet, pulmonary artery systolic pressure (PASP) was estimated using Bernoulli's equation in which right ventricular-right atrial gradient is added to mean right atrial pressure [16,17].

Transoesophageal echocardiography was performed within 72 hours before PMV to exclude left atrial thrombi.

Percutaneous balloon mitral valvuloplasty was performed using the INOUE device [18] (Toray Industries, Inc., Tokyo, Japan) in 153 patients and with the double balloon Multitrack device (NuMed, Inc., Hopkinton, NY, USA) in 4.

Right heart catheterisation was performed with balloon flow directed Swan-Ganz catheter and the following parameters were determined before and after the procedure: PASP, mean pulmonary artery pressure (PAMP), pulmonary artery diastolic pressure (PADP), pulmonary capillary wedge pressure (PCWP), right atrial pressure, RV pressure, cardiac output, trans-pulmonary pressure gradient (TPG, defined as the difference between PAMP and PCWP) and diastolic pulmonary vascular pressure gradient (DPG, defined as the difference between PADP and PCWP). Oxygen content was analysed from blood samples.

Patients were divided in two groups according to the PAMP measured by right heart catheterisation immediately before PMV: Group 1, patients with severe pulmonary artery hypertension (PAH), defined as PAMP $\geq$ 30 mm Hg, and Group 2: patients with PAMP $\leq$ 30 mmHg.

After PMV, left ventriculography was carried out to determine the presence and degree of mitral regurgitation.

Mitral valve area was assessed by Doppler echocardiography using the Gorlin method in the catheter laboratory immediately after the procedure, 72 hours later and during follow-up. Restenosis (Rs) was defined as loss of >50% of the initial gain of MVA by the preceding PMV with a final MVA <1.5 cm^2 [19,20].

Clinical and echocardiographic follow-up was performed during hospitalisation, at 1, 6 and 12 months and annually thereafter. Median follow-up was 48 months (24-84 months). Pulmonary artery pressure values were obtained only by echocardiography to avoid repeating catheterisations, as there was a very close correlation between the values obtained by this method and the baseline right heart catheterisation.

The primary end-points of the study were: 1) procedure success, defined as achievement of MVA $\geq$1.5 cm^2 without major complications (mortality, mitral regurgitation >II according to Sellers classification, systemic embolism or cardiac tamponade); 2) in-hospital mortality; 3) clinical improvement defined as $\geq$1-point New York Heart Association functional class improvement; 4) cardiovascular mortality.

Secondary end-points were: 1) development or worsening of mitral valve regurgitation after the procedure or during follow-up; 2) procedure-related complications as needed for mitral valve replacement (MVR); 3) need for new PMV or deterioration to functional class to III or IV; and, 4) restenosis.

Statistical Analysis

Categorical variables are expressed as frequencies and percentages and were analysed by the chi square test or Fisher exact test. Continuous variables are expressed as mean $\pm$ standard deviation (SD) or median and interquartile range (IQR 25-75) and comparison of parametric and no parametric values between the two groups was performed by means of two-tailed Student t-test or Kruskal-Wallis test, as appropriate. The association between demographic, clinical and haemodynamic variables with PMV success and with the presence of PAH was evaluated. The cumulative survival curves were constructed with the Kaplan-Meier method and the Cox method was used to compare time-dependent covariates. All the calculations were performed using the Statistix® 7.0 software package and a p value < 0.05 was considered statistically significant.

Results

The cohort was made up of 157 patients with MS considered candidates for PMV. Mean age was 44.2 ± 13.3 years and 139 patients (88.5%) were women. Ninety-eight patients (62.4%) were in NYHA class II, 53 patients (33.7%) in class III and 6 patients (3.8%) in class IV. Atrial fibrillation was present in 31.8% of the patients; 10 patients (6.4%) had a history of mitral commissurotomy. The clinical, echocardiographic and haemodynamic characteristics of patients are summarised in Table 1. Sixty patients (38.2%) had severe PAH (Group 1), and 97 patients (61.8%) had PAMP ≤ 30 mmHg (Group 2).

In the group of patients with PAH, mean age was 43.5 ± 14 years, 85% (51 patients) were women, 36.6% were in NYHA class III-IV and 25% presented AF. There were no differences in the clinical and demographic characteristics between groups 1 and 2.

Median MVA in Group 1 was 0.90 cm^2 (0.80 to 0.97 cm^2), PASP assessed by right heart catheterisation was 62 mmHg (55-67), PAMP 38 mmHg, PADP 27 mmHg, mean CWP 30 mmHg, TPG 12 mmHg (7-19) and DPG 2 mmHg (1-4); PASP assessed by Doppler echocardiography was 57 mmHg (51 to 65 mmHg); 30% of patients showed an echocardiographic Wilkins score greater than 8 and 58.3% (35 patients) had mitral regurgitation, which was mild in 97.1% of them (34 patients). There were no differences in the echocardiographic characteristics between both groups.

Percutaneous balloon mitral valvuloplasty was achieved in 125 patients (79.6%) with a median hospital stay of three days (2-8). The median MVA after PMV was 1.71 cm^2. The achieved MVA in the 32 patients with an unsuccessful procedure was 1.26 cm^2 (1.13 to 1.30).

In Group 1, the success rate was 76.6% (46 patients), with a MVA after PMV of 1.76 cm^2 (1.5 to 2.1). Pulmonary artery systolic pressure decreased from 62 to 35 mmHg and PCWP dropped from 25 to 16 mmHg. After PMV, 30 patients (50%) had mild (angiographic grade I) and 9 patients (15%) had moderate (angiographic grade II) mitral regurgitation. There were no statistically significant differences between both groups regarding the success rate, PASP, MVA or development of mitral regurgitation after PMV (Table 2).

Three hospital deaths (1.9%), unrelated to the presence of PAH occurred due to non-cardiac causes.

Clinical and echocardiographic follow-up was achieved in 139 patients (88.5%) with a median follow-up of 48 months (24-84 months). The median PASP in Group 1 was 35 (30-38), 32 (30-40), 36 (28.5 to 37.5), 38 (30-40) and 34 (30-41) mmHg at 12 24, 36, 48 and 60 months, respectively (Figure 1). No significant differences were observed between both groups in the outcome of MVA and PASP. No differences were observed in PHT progression in all patients with regard to

Table 1 Baseline demographic, clinical, echocardiographic and haemodynamic characteristics

	Group 1 PAMP > 30 mmHg (n = 60)		Group 2 PAMP ≤ 30 mmHg (n = 97)		p
Age (years) (mean ± SD)	43.5 ± 14		45.4 ± 13		0.52
Female gender	51	85%	88	90.7%	0,31
Atrial fibrillation	15	25%	35	36%	0,16
NYHA II	34	56.7%	64	65.9%	0,30
NYHA III	25	41.7%	28	28.9%	0,12
NYHA IV	1	1.6%	5	5.2%	0.40
Previous PMV	4	6.6%	7	7.2%	1,00
Previous commissurotomy	3	5%	7	7.2%	0,74
MVA before PMV (cm^2) Median (IQR)	0.90 (0.80-0.97)		0.90 (0.82-1.02)		0.29
PASP (echo) before PMV (mmHg) median (IQR)	57 (51- 65)		38 (30- 43)		<0.01
PASP - median (IQR)	62 (55-67)		40 (26-45)		<0.01
dPAP - median (IQR)	27 (21-32)		18 (14-19)		<0.01
PAMP - median (IQR)	38 (32-40)		25 (21-27)		<0.01
PCWP (mmHg) median (IQR)	30 (24-33)		20 (15-26)		<0.01
TPG (mmHg) median (IQR)	12 (7-19)		7 (3-9)		<0.01
MR before PMV	35	58.3%	54	55.6%	0.87
Wilkins score median (IQR)	8 (6-9)		7 (5-8)		0.06
Wilkins score >8	18	30%	29	29.8%	1.00

PAMP: pulmonary artery mean pressure; PASP: pulmonary artery systolic pressure; NYHA: New York Heart Association functional class; PMV: percutaneous balloon mitral valvuloplasty; MVA: mitral valve area; PCWP: pulmonary capillary wedge pressure; MR: mitral regurgitation; PADP: pulmonary artery diastolic pressure; TPG: transpulmonary pressure gradient; IQR: interquartile range.

Table 2 Echocardiographic and haemodynamic results

	Group 1 PAMP > 30 mmHg (n = 60)		Group 2 PAMP ≤ 30 mmHg (n = 97)		p
MVA after PMV (cm^2) Median (IQR)	1.76 (1.52 - 2.07)		1.71 (1.50 - 1.99)		0.23
Success (MVA >1,5 cm^2)	46	76.6%	79	81.4%	0.54
PASP after PMV (mmHg) (Doppler) median (IQR)	35 (30 - 49)		30 (27.7-35)		0.11
PASP after PMV (catheterisation) median (IQR)	35 (26-38)		27 (18-29)		0.35
PCWP after PMV (mmHg) median (IQR)	16 (12-18)		16 (11-17)		0.66
Mitral regurgitation after PMV	39	65%	68	86.1%	0.59
Mild MR	30	50%	56	57.7%	0.41
Moderate MR	9	15%	12	12.3%	0.81

MVA: mitral valve area; PMV: percutaneous balloon mitral valvuloplasty; PASP: pulmonary artery systolic pressure; PCWP: pulmonary capillary wedge pressure; MR: mitral regurgitation; IQR: interquartile range.

angiographic grades of mitral regurgitation following PMV (Figure 2).

In patients with PAH, there was a very gradual reduction in median MVA over time: 1.61 cm^2 (1.34 - 1.89); 1.60 cm^2 (1.33 - 1.89), 1.59 cm^2 (1,40 - 1,90), 1.56 cm^2 (1.32 - 1.84) and 1.50 cm^2 (1.34 - 1.86) at 12, 24, 36, 48 and 60 months, respectively (Figure 3). There was no association between the presence of PAH and the outcome of MVA or survival free of restenosis at follow-up (group 1: 0,87 vs. group 2: 0,76; p=0,21).

After four years of follow-up 109 patients remained asymptomatic (86.5%), 8 patients (6.3%) were on NYHA class II or III and 4 patients (3.1%) had palpitations. The number of asymptomatic patients was similar in both groups (Figure 4).

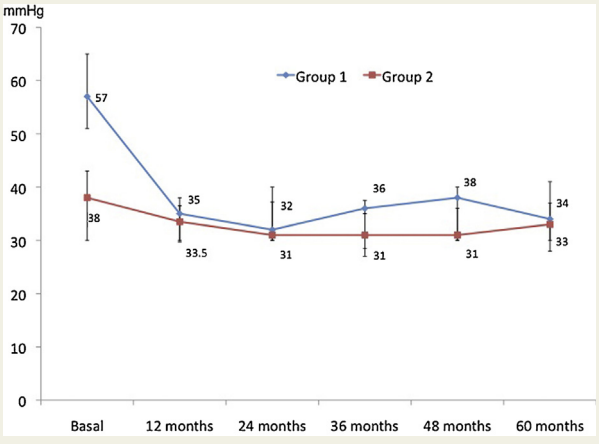

Figure 1 Evolution of the PASP after PMV and during follow-up according to the presence (Group 1) or absence (Group 2) of severe PAH.

Discussion

In this study, we found that a high proportion of patients with severe MS have associated PAH. This prevalence was similar to those observed in other studies analysing the outcome of patients with PAH after PMV or surgical replacement [21–23]. Although most reports do not describe the percentage of patients with PAH, some registries with a larger number of patients showed mean or median PASP values similar to those of this study [19,24,25].

The success rate in the total population was 79.6%, a result that fits within the reported range (73 to 99%) [19–21,26].

An inverse relation has been described between the echocardiographic Wilkins score and outcome. The best immediate results were obtained in young patients with an

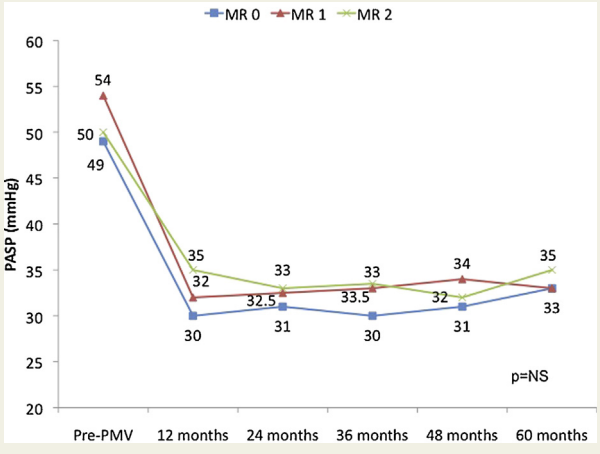

Figure 2 Evolution of PASP in all the patients, according to the presence of mitral regurgitation (MR). MR 0: no mitral regurgitation; MR 1: mitral regurgitation grade I; MR 2: mitral regurgitation grade II.

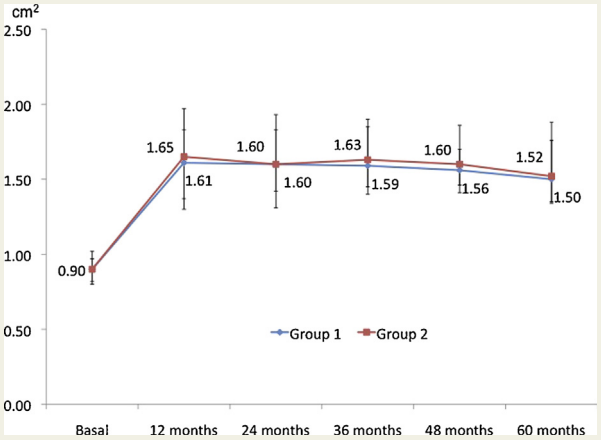

Figure 3 Evolution of the mitral valve area after PMV and during follow-up according to the presence (Group 1) or absence of severe PAH (Group 2).

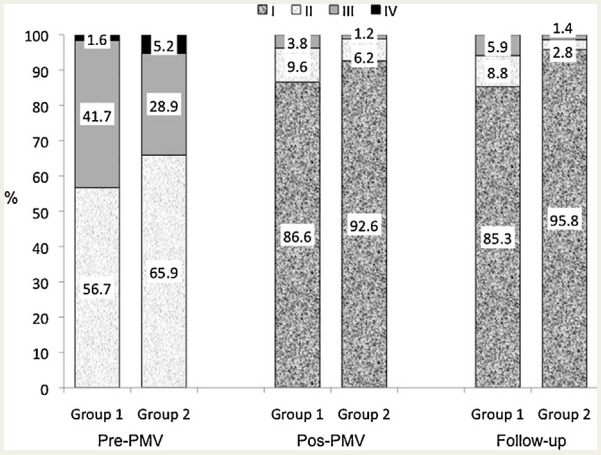

Figure 4 Evolution of NYHA class between baseline, pos PMV and follow-up according to the presence (Group 1) or absence of severe PAH (Group 2).

echocardiographic score <8, large MVA before PMV, mild to moderate mitral regurgitation, male gender and absence of previous commissurotomy [19]. In the present study, both the Wilkins score >8 and the presence of AF were associated with a lower rate of periprocedural success. However, we did not find a significant relationship between the presence of PAH and success rate as has previously been described [20,23].

We observed a decrease of PASP to normal values after PMV that persisted over time. Both the immediate and long-term results were similar among patients with or without high PASP, consistent with other reports [22,24]. Several studies evaluating patients treated with surgical valve replacement, have observed a significant decrease in PASP, achieving near normal levels [27,28]. Braunwald et al. reported 31 surgical patients with PAH who underwent a postoperative decrease from 75 to 39 mmHg of PASP [6]. Zener et al., analysing 27 patients with MS and PASP >100 mmHg, also have observed a decrease from 115 to 50 mmHg after surgery [28]. Although the passive component of pulmonary hypertension decreased immediately after the procedure, the regression of pulmonary vascular resistance took more time [29]. The decline of PASP in our patients with PAH was significant at six months reaching normal values at one year, and remained stable until end of follow-up; however, some studies found a gradual increase in pulmonary pressure in patients who developed restenosis [28].

We did not observe a significant relation between PAH before PMV and mortality during long-term follow-up.

The MVA showed a gradual decrease over time, which was similar in patients with and without PAH. Most registries showed a decrease of MVA in the five-year follow-up period between 0.12 to 0.20 cm^2, related to an increase in the rate of restenosis. Several reports showed a wide variability in the incidence of restenosis (between 3% and 70%) from one to three years of follow-up [19–22,25,30,31]. The rate of Rs in our study was 29.4% at 60 months follow-up, and was not associated with the presence of PAH. This rate is similar to that reported by Hernández et al. who observed a rate of Rs of 39% after seven years of follow-up [31]. Fawzy reported that patients with a PASP >60 mmHg before PMV, had a slight increase in the rate of Rs and events during follow-up, compared with patients with PASP <60 mmHg; however, only an echocardiographic score > 8 and female gender resulted in independent predictors of Rs in this investigation [22].

Study Limitations

The main limitation of this study is its retrospective nature, however, complete data were obtained in all patients, and 91% completed long-term follow-up.

Pulmonary artery pressures were determined by echocardiography during follow-up and not by direct measurement due to the excellent concordance of echocardiography with the pressures recorded at the initial right heart catheterisation.

Conclusions

Based on the results observed, PMV is a safe and effective procedure in patients with severe PAH. The significant reduction in PASP after PMV was maintained at long-term follow-up with a significant improvement in NYHA functional class without differences between groups. Despite the gradual reduction in MVA, most patients remained asymptomatic at the end of follow-up.

Disclosures

No sources of external funding to declare.

References

[1] Rheumatic fever and rheumatic heart disease. Report of a WHO Study Group. World Health Organ Tech Rep Ser. 1988;764:1-58

[2] Marcus RH, Sareli P, Pocock WA, Barlow JB. The spectrum of severe rheumatic mitral valve disease in a developing country. Correlations among clinical presentation, surgical pathologic findings, and hemodynamic sequelae. Ann Intern Med 1994;120:177–83.
[3] Wood P. An appreciation of mitral stenosis. I. Clinical features. Br Med J 1954;1:1051–63. contd.
[4] Horstkotte D, Niehues R, Strauer BE. Pathomorphological aspects, aetiology and natural history of acquired mitral valve stenosis. Eur Heart J 1991;(12 Suppl B):55–60.
[5] Walston A, Peter RH, Morris JJ, Kong Y, Behar VS. Clinical implications of pulmonary hypertension in mitral stenosis. Am J Cardiol 1973;32: 650–5.
[6] Braunwald E, Braunwald NS, Ross Jr J, Morrow AG. Effects of Mitral-Valve Replacement on the Pulmonary Vascular Dynamics of Patients with Pulmonary Hypertension. N Engl J Med 1965;273:509–14.
[7] Austen WG, Corning HB, Moran JM, Sanders CA, Scannell JG. Cardiac hemodynamics immediately following mitral valve surgery. J Thorac Cardiovasc Surg 1966;51:468–73.
[8] Ward C, Hancock BW. Extreme pulmonary hypertension caused by mitral valve disease. Natural history and results of surgery. Br Heart J 1975;37:74–8.
[9] Camara ML, Aris A, Padro JM, Caralps JM. Long-term results of mitral valve surgery in patients with severe pulmonary hypertension. Ann Thorac Surg 1988;45:133–6.
[10] Lefevre T, Bonan R, Serra A, Crepeau J, Dyrda I, Petitclerc R, et al. Percutaneous mitral valvuloplasty in surgical high risk patients. J Am Coll Cardiol 1991;17:348–54.
[11] Alfonso F, Macaya C, Hernandez R, Banuelos C, Iniguez A, Goicolea J, et al. Percutaneous mitral valvuloplasty with severe pulmonary artery hypertension. Am J Cardiol 1993;72:325–30.
[12] Bahl VK, Chandra S, Talwar KK, Kaul U, Sharma S, Wasir HS. Balloon mitral valvotomy in patients with systemic and suprasystemic pulmonary artery pressures. Cathet Cardiovasc Diagn 1995;36:211–5.
[13] Sellers RD, Levy MJ, Amplatz K, Lillehei CW. Left Retrograde Cardioangiography in Acquired Cardiac Disease: Technic, Indications and Interpretations in 700 Cases. Am J Cardiol 1964;14:437–47.
[14] Wilkins GT, Weyman AE, Abascal VM, Block PC, Palacios IF. Percutaneous balloon dilatation of the mitral valve: an analysis of echocardiographic variables related to outcome and the mechanism of dilatation. Br Heart J 1988;60:299–308.
[15] Hatle L, Angelsen B, Tromsdal A. Noninvasive assessment of atrioventricular pressure half-time by Doppler ultrasound. Circulation 1979;60: 1096–104.
[16] Galie N, Hoeper MM, Humbert M, Torbicki A, Vachiery JL, Barbera JA, et al. Guidelines for the diagnosis and treatment of pulmonary hypertension: the Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS), endorsed by the International Society of Heart and Lung Transplantation (ISHLT). Eur Heart J 2009;30: 2493–537.
[17] McLaughlin VV, Archer SL, Badesch DB, Barst RJ, Farber HW, Lindner JR, et al. ACCF/AHA 2009 expert consensus document on pulmonary hypertension a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association developed in collaboration with the American College of Chest Physicians; American Thoracic Society, Inc.; and the Pulmonary Hypertension Association. J Am Coll Cardiol 2009;53:1573–619.
[18] Inoue K, Owaki T, Nakamura T, Kitamura F, Miyamoto N. Clinical application of transvenous mitral commissurotomy by a new balloon catheter. J Thorac Cardiovasc Surg 1984;87:394–402.
[19] Palacios IF, Sanchez PL, Harrell LC, Weyman AE, Block PC. Which patients benefit from percutaneous mitral balloon valvuloplasty?. Prevalvuloplasty and postvalvuloplasty variables that predict long-term outcome. Circulation 2002;105:1465–71.
[20] Herrmann HC, Ramaswamy K, Isner JM, Feldman TE, Carroll JD, Pichard AD, et al. Factors influencing immediate results, complications, and short-term follow-up status after Inoue balloon mitral valvotomy: a North American multicenter study. Am Heart J 1992;124:160–6.
[21] Fawzy ME, Choi WB, Mimish L, Sivanandam V, Lingamanaicker J, Khan A, et al. Immediate and long-term effect of mitral balloon valvotomy on left ventricular volume and systolic function in severe mitral stenosis. Am Heart J 1996;132:356–60.
[22] Fawzy ME, Hassan W, Stefadouros M, Moursi M, El Shaer F, Chaudhary MA. Prevalence and fate of severe pulmonary hypertension in 559 consecutive patients with severe rheumatic mitral stenosis undergoing mitral balloon valvotomy. J Heart Valve Dis 2004;13:942–7. discussion 7-8.
[23] Hart SA, Krasuski RA, Wang A, Kisslo K, Harrison JK, Bashore TM. Pulmonary hypertension and elevated transpulmonary gradient in patients with mitral stenosis. J Heart Valve Dis 2010;19:708–15.
[24] Levine MJ, Weinstein JS, Diver DJ, Berman AD, Wyman RM, Cunningham MJ, et al. Progressive improvement in pulmonary vascular resistance after percutaneous mitral valvuloplasty. Circulation 1989;79: 1061–7.
[25] Iung B, Garbarz E, Michaud P, Helou S, Farah B, Berdah P, et al. Late results of percutaneous mitral commissurotomy in a series of 1024 patients. Analysis of late clinical deterioration: frequency, anatomic findings, and predictive factors. Circulation 1999;99:3272–8.
[26] The National Heart, Lung, and Blood Institute Balloon Valvuloplasty Registry Participants. Multicenter experience with balloon mitral, commissurotomy., NHLBI., Balloon Valvuloplasty Registry Report on immediate and 30-day follow-up, results. Circulation 1992;85:448–61.
[27] Otto CM, Davis KB, Reid CL, Slater JN, Kronzon I, Kisslo KB, et al. Relation between pulmonary artery pressure and mitral stenosis severity in patients undergoing balloon mitral commissurotomy. Am J Cardiol 1993;71:874–8.
[28] Zener JC, Hancock EW, Shumway NE, Harrison DC. Regression of extreme pulmonary hypertension after mitral valve surgery. Am J Cardiol 1972;30:820–6.
[29] Park SJ, Kim JJ, Park SW, Song JK, Doo YC, Lee SJ. Immediate and one-year results of percutaneous mitral balloon valvuloplasty using Inoue and double-balloon techniques. Am J Cardiol 1993;71:938–43.
[30] Ben-Farhat M, Betbout F, Gamra H, Maatouk F, Ben-Hamda K, Abdellaoui M, et al. Predictors of long-term event-free survival and of freedom from restenosis after percutaneous balloon mitral commissurotomy. Am Heart J 2001;142:1072–9.
[31] Hernandez R, Banuelos C, Alfonso F, Goicolea J, Fernandez-Ortiz A, Escaned J, et al. Long-term clinical and echocardiographic follow-up after percutaneous mitral valvuloplasty with the Inoue balloon. Circulation 1999;99:1580–6.