Effectiveness of Rescue Percutaneous Balloon Aortic Valvuloplasty in Patients With Severe Aortic Stenosis and Acute Heart Failure

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The prognosis of patients with cardiogenic shock (CS) or refractory pulmonary edema because of severe aortic stenosis remains poor. The purpose of this study was to assess the outcomes of rescue percutaneous balloon aortic valvuloplasty (PBAV) in the transcatheter aortic valve implantation (TAVI) era. Patients were consecutively included between 2008 and 2016. CS was defined as ≥1 sign of systemic hypoperfusion and need of catecholamines. Refractory pulmonary edema was defined as not controlled by optimal medical treatment. A total of 40 patients, 22 men (55%), aged 79 ± 9 years, were included: 17 with CS (42.5%), 23 with refractory pulmonary edema (57.5%). After PBAV, mean transaortic gradient decreased from 47 ± 16 mm Hg to 32 ± 10 mm Hg ($p < 0.001$), aortic valve area increased from 0.60 ± 0.18 cm^2 to 0.88 ± 0.22 cm^2 ($p < 0.0001$), left ventricular ejection fraction increased from 35 ± 15 to 37 ± 14% ($p = 0.02$), and systolic pulmonary artery pressure decreased from 61 ± 15 to 48 ± 12 mm Hg ($p = 0.002$). There was no procedural death. Early death occurred in 12 patients (30%). After PBAV, 16 of the 28 survivors (57%) were bridged to surgical aortic valve replacement (SAVR; n = 7) or TAVI (n = 9), and 12 (43%) were denied definitive therapy. The 2-year estimated survival rate was 71 ± 17% after SAVR, 36 ± 19% after TAVI, and 8 ± 8% after PBAV alone. In conclusion, rescue PBAV is safe in patients with CS and high-risk aortic stenosis or refractory pulmonary edema and may improve their dismal prognosis when followed by TAVI or SAVR. **(Am J Cardiol 2018;121:746–750)**

Percutaneous balloon aortic valvuloplasty (PBAV) to treat patients with severe aortic stenosis was first described by Alain Cribier in 1986.[1] Unfavorable mid-term outcomes related to early restenosis have rapidly limited its indications.[2,3] However, in patients with aortic stenosis and cardiogenic shock (CS), PBAV was shown to be lifesaving in some circumstances.[4,5] Since the development of transcatheter aortic valve implantation (TAVI), PBAV is considered with a renewed interest as part of the procedure, and may be indicated as a bridge to further interventions in very high-risk patients.[6–8] The aim of this study was to assess the early and late outcomes of rescue PBAV in patients with CS or refractory pulmonary edema because of severe aortic stenosis.

Methods

From January 2008 to November 2016, 40 patients with CS or refractory pulmonary edema because of severe aortic stenosis were consecutively admitted in our institution and were treated in emergency with PBAV. CS was defined as (1) need of catecholamines (dobutamine and/or norepinephrine and/or epinephrine at optimal doses) and ≥1 sign of systemic hypoperfusion including sustained arterial hypotension with systolic blood pressure ≤90 mm Hg, altered mental status, cold clammy skin and extremities, oliguria, and serum lactate >2.0 mmol/L; and (2) absence of any clinical or biological evidence for a noncardiac cause of shock, in particular sepsis or hypovolemia. Refractory pulmonary edema was defined as heart failure not controlled by optimal medical treatment including diuretics, noninvasive or mechanical ventilation, and correction of any aggravating condition (arrhythmia, anemia…). According to the severity of the clinical presentation and to the response to treatment, PBAV was performed within 6 to 24 hours from admission.

PBAV was performed using the transfemoral retrograde approach. After placement of a 9- to 12-Fr sheath in the femoral artery, the native aortic valve was crossed and a 0.035″ extra-stiff 2.6-m long J-curved wire was placed at the apex of the left ventricle. Then, a valvuloplasty balloon (Cristal; Balt, Montmorency, France or Nucleus; Numed, Hopkinton, NY) was brought to the aortic valve. The size of the balloon diameter was chosen according to the measurement of the annulus diameter by echocardiography (ratio 1:1). Usually, 2 to 3 successive balloon inflations were performed. In most cases, a rapid right ventricular pacing (140/180 bpm) was used to stabilize the balloon during inflations. Arterial puncture sites were closed with closure devices (6-F Perclose [Abbott Laboratories, Abbott Park, IL] or 8-F Angio-Seal [St. Jude Medical,

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St. Paul, MN]). At the end of the procedure, patients were transferred to the intensive care unit.

Fatal outcomes related to end-stage aortic valve disease, multiorgan failure, or serious adverse events within the 7 days after PBAV were qualified as early deaths. Survivors were bridged to TAVI or surgical aortic valve replacement (SAVR), or treated medically according to the heart team judgment. The decision was made considering the expected quality of life and life expectancy related to co-morbidities, age, general status, and operative risk.

Baseline clinical data and hospital outcomes were collected by chart review by an independent researcher. Major complications related to PBAV were classified using the Valve Academic Research Consortium 2 criteria.[9] Follow-up was obtained from chart review as well as telephone interviews with the referring cardiologist, primary care physician, patient, and family.

Continuous variables are presented as mean ± standard deviation except for the interval between PBAV and TAVI or SAVR, and for the length of follow-up, which are presented as median with 25th to 75th percentiles. They were compared using the *t* test when normally distributed or Mann-Whitney *U* test when non-normally distributed. Categorical variables are reported as n (%) and were compared between groups with the χ^2 or Fisher exact test. Changes in echocardiographic data were analyzed only in patients with known values at baseline and after PBAV with the Wilcoxon signed rank test. Survival rates after PBAV were estimated with the Kaplan-Meier method and cumulative survival rates were compared with the log-rank test. A p-value <0.05 was considered to indicate a statistically significant difference. All statistical calculations were performed with the JMP version 9.0 software (SAS Institute Inc., Cary, NC).

Results

Baseline patients' characteristics are displayed in Table 1. A total of 40 patients, 22 men (55%), aged 79 ± 9 years, were included: 23 patients with refractory pulmonary edema (57.5%) and 17 patients with CS (42.5%). Patients with CS had lower systolic aortic pressure and a lower left ventricular ejection fraction than those with refractory pulmonary edema. The catecholamines used were dobutamine alone for 10 patients, dobutamine and norepinephrine for 5 patients, and epinephrine alone for 2 patients.

Overall, 14 patients were already under mechanical ventilation before rescue PBAV. The mean diameter of the balloon was 22 ± 2 mm. A second larger balloon was used to improve

Table 1
Baseline characteristics

Variables	Overall (n = 40)	Refractory pulmonary edema (n = 23)	Cardiogenic shock (n = 17)	p value
Age (years)	79 ± 9	79 ± 9	79 ± 9	0.9
Men	22 (55%)	10 (43%)	12 (70%)	0.1
Baseline comorbidities				
Prior stroke	2 (5%)	1 (4%)	1 (6%)	1
Peripheral artery disease	12 (30%)	5 (22%)	7 (40%)	0.3
Diabetes mellitus	12 (30%)	9 (40%)	3 (17%)	0.2
Hypertension	28 (70%)	18 (78%)	10 (59%)	0.3
Severe chronic obstructive pulmonary disease	11 (27.5%)	8 (35%)	3 (18%)	0.3
Atrial fibrillation	19 (48%)	9 (39%)	10 (59%)	0.2
Glomerular filtration rate ≤ 30 ml/min	13 (32%)	8 (35%)	5 (29%)	1
Coronary artery disease	16 (40%)	7 (23%)	9 (53%)	0.2
Prior coronary bypass surgery	5 (13%)	2 (9%)	3 (18%)	0.6
Prior percutaneous coronary intervention	4 (10%)	2 (1%)	2 (1%)	1
Prior cancer	6 (2%)	2 (1%)	4 (2%)	0.4
At admission				
Body mass index (kg/m^2)	25 ± 6	26 ± 6	25 ± 6	0.3
Systolic blood pressure (mmHg)	111 ± 24	119 ± 26	99 ± 17	0.01
Heart rate (beats/min)	94 ± 20	91 ± 23	98 ± 16	0.2
Emergency dialysis	10 (25%)	6 (27%)	4 (25%)	1
Inotropic support	17 (43%)	0 (0%)	17 (100%)	<0.0001
Non-invasive ventilation	12 (30%)	10 (44%)	2 (12%)	0.04
Mechanical ventilation	14 (35%)	7 (30%)	7 (41%)	0.7
Logistic euroscore (%)	62 ± 21	56 ± 24	70 ± 13	0.08
Euroscore 2 (%)	28 ± 18	18 ± 12	41 ± 15	<0.0001
Society of Thoracic Surgeons score (%)	26 ± 15	17 ± 15	38 ± 13	<0.0001
Echocardiography				
Left ventricular ejection fraction (%)	35 ± 15	41 ± 15	27 ± 11	0.01
Aortic valve area (cm^2)	0.62 ± 0.16	0.65 ± 0.16	0.57 ± 0.15	0.2
Mean transaortic gradient (mmHg)	47 ± 15	51 ± 12	41 ± 17	0.1
Pulmonary artery systolic pressure (mmHg)	58 ± 14	58 ± 14	59 ± 15	1

Continuous data are presented as mean ± SD and categorical data as n (%).

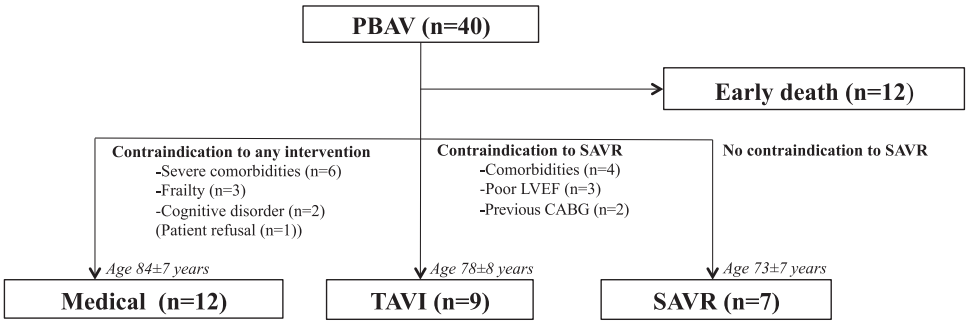

Figure 1. Management of 40 patients with aortic stenosis and cardiogenic shock or refractory pulmonary edema. CABG = coronary artery bypass grafting; LVEF = left ventricular ejection fraction; PBAV = percutaneous balloon aortic valvuloplasty; SAVR = surgical aortic valve replacement; TAVI = transcatheter aortic valve implantation.

dilatation in 16 patients (40%). PBAV was associated with a reduction in mean transaortic gradient (from 47 ± 16 to 32 ± 10 mm Hg, $p < 0.0001$), and systolic pulmonary artery pressure (from 61 ± 15 to 48 ± 12 mm Hg, $p = 0.002$) and an increase in aortic valve area (from 0.60 ± 0.18 to 0.88 ± 0.22 cm^2, $p < 0.0001$) and left ventricular ejection fraction (from 35 ± 15 to 37 ± 14%, $p = 0.02$). Several major complications were observed during PBAV: 3 resuscitated cardiac arrests (2 asystoles and 1 ventricular fibrillation), 1 severe aortic regurgitation, and 3 complete atrioventricular blocks. However, there was no procedural death.

Early deaths occurred in 12 patients (30%): 8 patients had CS (48%) and 4 patients had refractory pulmonary edema (24%). Median time to death was 5 days (3 to 9). The causes of deaths were related to pre-existing co-morbidities in 5 cases: an 85-year-old patient with severe 3-vessel coronary artery disease denied for surgery; an 83-year-old patient with previous coronary artery bypass grafting, severe obesity, and end-stage renal failure; an 84-year-old patient with metastatic cancer and previous coronary artery bypass grafting; a frail 79-year-old patient with altered general condition and cachexia; and a 93-year-old patient with immediate after PBAV severe subaortic obstruction precluding TAVI. The causes of deaths were related to baseline multiorgan failure in 4 cases and to serious adverse events in 3 cases: septic shock in a 77-year-old patient with chronic obstructive pulmonary disease and home-oxygen; massive hemoptysis in an 87-year-old patient, requiring orotracheal intubation and bronchial artery embolization; and aspiration pneumonia 6 days after PBAV, requiring orotracheal intubation in a 76-year-old patient on chronic dialysis. In addition, there was 1 postprocedural major ischemic stroke, which was confirmed by scanner 10 days after PBAV in a patient with new-onset atrial fibrillation. In the 28 survivors after PBAV, 25 patients (89%) experienced a substantial clinical improvement and were weaned from hemodynamic support and ventilation, 3 patients did not improve because of persistent severe aortic stenosis in 2 cases and severe aortic regurgitation in 1 case.

The management of the survivors after rescue PBAV is displayed in Figure 1. In the 28 survivors, 7 patients (25%) underwent SAVR using bioprostheses, 9 patients (32%) underwent TAVI (transfemoral, n = 6; transapical, n = 2; subclavian, n = 1) and 12 patients (43%) were treated medically. The median interval between PBAV and TAVI or SAVR was 15 (range = 1 to 47) days. Most patients bridged to SAVR had no severe extracardiac co-morbidities. Their mean age was 73 ± 7 years. However, 2 patients who did not improve after PBAV were operated on: a 68-year-old woman and an 82-year-old woman with severe aortic regurgitation after PBAV. A 76-year-old man with poor left ventricular ejection fraction was also operated on because of a technical contraindication to TAVI (bicuspid valve with a too large annulus diameter). At the opposite, all patients bridged to TAVI were deemed at high surgical risk. Their mean age was 78 ± 8 years. The high surgical risk was because of lack of improvement after PBAV and concomitant pulmonary embolism in a 1 case; poor left ventricular ejection fraction in 3 cases, previous coronary artery bypass grafting in 2 cases, severe chronic kidney disease in 2 cases, severe respiratory failure with home-oxygen in 1 case. Finally, 12 patients were denied any intervention. Their mean age was 84 ± 7 years. Main reasons for considering medical treatment alone were severe associated co-morbidities in 6 cases: severe chronic obstructive pulmonary disease and dialysis in 2 cases; previous stroke and dialysis in 2 cases; severe chronic obstructive pulmonary disease in a previous lung transplant without vascular access in 1 case; severe 3-vessel coronary artery disease, poor left ventricular ejection fraction, and no vascular access in 1 case; frailty in 3 cases (altered general status, mean age 90 years); cognitive disorder in 2 cases; patient's refusal in 1 case.

No patient was lost to follow-up. The median follow-up was 11 (range 5 to 39) months. Overall, 19 deaths (68% of the 28 survivors) occurred. Of those, 12 were cardiovascular. Overall estimated survival rates were 46 ± 10% and 33 ± 9% at 1 and 2 years, respectively. Estimated survival rates tended to be higher in patients with refractory pulmonary edema than in those with CS (52 ± 12 versus 33 ± 16% and 39 ± 12 versus 22 ± 14% at 1 and 2 years, respectively); however, there was no statistical difference. Survival curves according to the treatment received after PBAV are shown in Figure 2. Survival rates after 1 and 2 years were, respectively, 71 ± 17% and 71 ± 17% after SAVR, 53 ± 17% and 36 ± 19% after TAVI, and 25 ± 12% and 8 ± 8% after PBAV alone ($p < 0.01$). In the SAVR group, 2 deaths occurred during the early postoperative course (at day 4): 1 from multiorgan failure, 1 from sudden unexplained cardiac arrest. There was no death after discharge in this group. In the TAVI group, 5 deaths occurred after a minimal follow-up of 4.5 months. Most of them were noncardiac: they were related to cancer (n = 2), pulmonary embolism (n = 1), acute respiratory failure (n = 1),

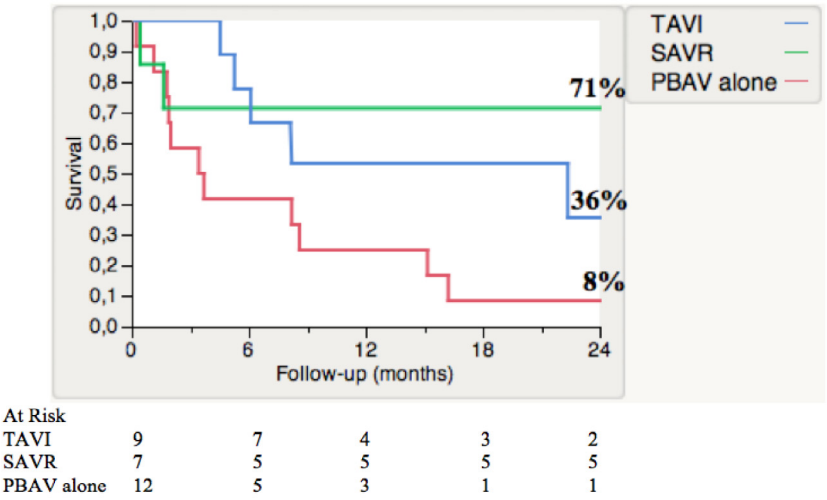

Figure 2. Kaplan-Meier survival curves after PBAV according to treatment. PBAV = percutaneous balloon aortic valvuloplasty; SAVR = surgical aortic valve replacement; TAVI = transcatheter aortic valve implantation.

and of unknown cause (n = 1). In the medical group, all patients had died at last follow-up. Median time to death was 3.6 months (1.8 to 13.5). The causes of death were heart failure (n = 5), respiratory failure (n = 3), sudden cardiac arrest (n = 2), septic shock (n = 1), and peritonitis (n = 1).

Discussion

As of today, very few data have been reported about rescue PBAV and even less in the TAVI era. Moreover, its mid-term outcomes have not been assessed. Our results suggest that rescue PBAV is feasible and safe in critically ill patients with severe aortic stenosis who cannot undergo immediate SAVR or TAVI, because of the severity of their hemodynamic condition. Most early deaths after rescue PBAV are related to their dismal baseline conditions. The patients who survive and are bridged to SAVR or TAVI have acceptable mid-term outcomes.

Nearly 30 years ago, several small reports have shown that PBAV could stabilize patients with CS.[10] Since then, small studies tended to confirm this efficacy. Cribier et al first reported the results of rescue PBAV in 10 patients with CS.[4] All patients clinically improved after the procedure. The 30-day mortality was 20%. Successful SAVR was secondarily performed in 80% of the survivors, but further outcomes of these patients were not specified. Moreno et al published the largest study involving 21 patients with CS.[5] Rescue PBAV was associated with periprocedural deaths (n = 2, 9.5%). The most frequent complication was vascular limb ischemia (24%). Although 76% of the patients were initially weaned from catecholamines, in-hospital mortality rate was 43%, mostly because of associated co-morbidities. SAVR was secondarily performed in only 4 patients of the 12 survivors (33%). The estimated survival rate at 27 months was 50% in the SAVR group, and 38% in the whole population. Lastly, Buchwald et al reported on 14 patients with CS treated with rescue PBAV.[11] Again, the most frequent complication was vascular injury requiring surgery (21%), but there was no per-procedural death. In contrast with the first 2 studies, only 29% of the patients had favorable outcomes and the in-hospital mortality rate was 71%. This high mortality may have been because of persisting multiorgan failure. Indeed, half of the patients required both mechanical ventilation and inotropic support, and all deaths occurred in patients whose shock symptoms occurred at least 48 hours before admission. SAVR was performed in 2 of the 4 survivors. All 4 survivors were alive at 1 year (29% survival rate). Overall, the available data are scarce and fairly contradictory as only small groups of heterogeneous patients have been followed over a limited period of time.

The current availability of TAVI after rescue PBAV appears to be an attractive solution in this population. Indeed, in the Moreno and Buchwald studies most survivors after PBAV were denied SAVR because of co-morbidities and might have benefited from TAVI. Surprisingly, there are nearly no data on this strategy. Only 1 small series of 13 patients with CS described the feasibility of TAVI in 4 of 8 survivors after PBAV.[12] However, the safety of the procedure and outcomes after TAVI were not reported. In our series, rescue PBAV was performed in all patients. There was no death related to the procedure, and we did not observe any severe vascular complication, whereas it was the most frequent serious adverse event reported after rescue PBAV before the TAVI era.[13–15] This improvement is because of the use of vascular closure devices and smaller catheters. The hemodynamic stabilization was achieved with similar significant improvements in gradients and aortic valve area as compared with those recently reported in elective PBAV.[7,16] This hemodynamic improvement allowed us to assess further management: according to clinical condition, we avoided futile procedures and discussed TAVI or SAVR. Indeed, within the SAVR group, patients had lower co-morbidities and their estimated survival rate remained high after 2 years. On the other hand, within the TAVI group, patients had delayed deaths mostly related to their co-morbidities, which represented contraindications to SAVR.

The recently proposed alternative approach of a rescue TAVI has been tested in 27 patients with CS by Frerker et al.[17] Rescue TAVI appeared feasible, with an 88.9% device success. However, despite the theoretical advantage of the immediate larger increase of the aortic valve area, the 30-day mortality remained high (33%). Moreover, the use of much larger catheters requires appropriate vascular access site and implies patient selection upstream. Lastly, in this very high-risk population with consistently high early mortality, the rationale of a systematic rescue TAVI is a matter of concern in terms of cost-effectiveness. Although rescue TAVI can be feasible for highly selected patients**,** rescue PBAV allows to stabilize the hemodynamic and clinical condition of the patients and to select those in whom further interventions may improve the prognostic and quality of life.

Our study has several limitations. This is an observational study from a single center with a retrospective design and patients were included over 8 years. However, they were consecutively and homogeneously treated by the same heart team and data were homogeneously collected. In addition, this is the largest series of patients with CS treated with PBAV with a mid-term follow-up so far reported in the literature.

In conclusion, when followed by TAVI or SAVR, rescue PBAV may improve the dismal prognosis of patients with high-risk aortic stenosis with CS or refractory pulmonary edema. Early and mid-term outcome are mostly related to comorbidities in these elderly patients.

Disclosures

DH is a proctor for Edwards Lifesciences and Medtronic; AV is on the advisory board for Medtronic and receives speaker's fees from Edwards Lifesciences. The other authors have no disclosures.

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