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Left Ventricular End-Diastolic Pressure as an Independent Predictor of Outcome During Balloon Aortic Valvuloplasty

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Abstract

Objectives—In this study, we examined the predictive value of the left ventricular enddiastolic pressure (LVEDP) in patients undergoing balloon aortic valvuloplasty (BAV).

Background—The LVEDP is a useful indicator of hemodynamic status in patients with severe aortic stenosis. In BAV, decompensated heart failure is associated with worse outcomes.

Methods—We identified all consecutive patients with severe symptomatic aortic stenosis who underwent retrograde BAV at the Massachusetts General Hospital from 2004 to 2008. Patients were stratified and compared according to their baseline LVEDP into 15 mm Hg, 16–20 mm Hg, 21–25 mm Hg, and 26 mm Hg. Procedural and in-hospital outcomes and adverse events were compared. Multivariate logistic regression was used for the adjusted analysis.

Results—A total of 111 patients with a mean age of 83±11 years underwent BAV. Of these, the LVEDP was 15 mm Hg in 29 (26%), 16–20 mm Hg in 41 (37%), 21–25 mm Hg in 16 (14%), and 26 mm Hg in 25 (23%) patients. Baseline characteristics were similar among the four groups. Noticeably, patients with high LVEDP levels had significantly higher rates of the combined endpoint of in-hospital death, myocardial infarction (MI), cardiopulmonary arrest, and tamponade was $P = 0.02$. Periprocedural MI was more common among those with higher LVEDP (16% vs. 2.3%; $P = 0.04$). Multivariate analysis revealed LVEDP (OR 1.08, for each mm Hg increase in pressure, 95 % CI 1.02–1.14), small LV chamber size, and New York Heart Association class as independent predictors of adverse outcomes.

Conclusions—The LVEDP is an important independent predictor of poor in-hospital outcome during BAV. In these patients, the immediate hemodynamic status may be more important than the baseline left ventricular systolic function. Hemodynamic optimization before or during BAV should be considered and may be beneficial.

Keywords

balloon aortic valvuloplasty; predictors; outcome LVEDP; TAVI

INTRODUCTION

Percutaneous balloon aortic valvuloplasty (BAV) is increasingly being used as a bridge to surgical valve replacement, as destination therapy in nonsurgical candidates, and as an integral part of transcatheter valve implantation in patients with severe aortic stenosis [1–3].

Several predictors of BAV outcome have been previously reported including advanced age, New York Heart Association (NYHA) Class and impaired left ventricular (LV) systolic function, among others [4–8]. Most of these factors, however, only serve to identify patients at overall greater risk, but do not necessarily help clinicians risk stratify patients requiring BAV.

The left ventricular end-diastolic pressure (LVEDP) has been traditionally used as an important surrogate marker of acute hemodynamic assessment in either systolic or diastolic LV dysfunction. Prior studies have demonstrated that an elevated LVEDP is associated with worse outcome after acute myocardial infarction (MI) [9], cardiac surgery [10,11], and left heart catheterization [12]. Furthermore, recent literature has proposed including estimates of diastolic dysfunction in future risk-stratification models in cardiac surgery [13].

The validity of the LVEDP in patients undergoing BAV has not been systematically evaluated. It remains unclear for example whether high LVEDP levels in the setting of preserved LV systolic function is worse than normal LVEDP in a patient with LV systolic dysfunction undergoing BAV. In this study, we examined the impact of the LVEDP in patients undergoing BAV and its interrelationship with other important clinical variables.

METHODS

Study Population

We identified all consecutive patients with severe, symptomatic calcific aortic stenosis undergoing retrograde percutaneous BAV at Massachusetts General Hospital between December 2004 and December 2008. Patients were stratified into quartiles according to their baseline LVEDP at the time of BAV of 15 mm Hg, 16–20 mm Hg, 21–25 mm Hg and 26 mm Hg. Patients in cardiogenic shock and those requiring mechanical ventilatory support before the procedure were excluded. Patients with bicuspid aortic valves, prior history of BAV, and severe peripheral vascular disease requiring antegrade BAV were also excluded.

Data Collection and Procedure

The data were collected through the individual review of hospital records, echocardiography, and catheterization laboratory databases. Procedural outcomes, complications, and in-hospital adverse events were analyzed and compared.

Standard right and left-sided heart catheterization data were available in all patients. Cardiac output was measured using the thermodilution technique. In the presence of left to right shunting or significant tricuspid regurgitation, cardiac output was measured according to the Fick method using assumed O_2 consumption. Simultaneous transvalvular gradients were measured routinely in all patients using a 6-French double lumen pigtail catheter before and after BAV. The aortic valve area (AVA) was calculated according to the Gorlin formula. Retrograde BAV was routinely performed from the common femoral artery through a 12-French catheter by using standard technique [14]. The balloon size was determined on an individual basis according to the midpoint aortic annulus diameter measured by echocardiography. All patients received intravenous heparin to achieve an activated clotting time ≥ 250 sec during their BAV. Rapid burst ventricular pacing was not routinely used and left to the discretion of the operator. Post-BAV AVA, transvalvular gradient, and cardiac output were measured and compared to those obtained at baseline.

Study Endpoints

The composite endpoint of intraprocedural and in-hospital adverse events were examined. Intraprocedural adverse events included all patients with BAV-related shock requiring intravenous vasopressors, cardiopulmonary resuscitation, endotracheal intubation, and death. Inhospital adverse events included all patients who following their BAV experienced a MI, cardiopulmonary arrest, pericardial tamponade, or death. Individual components were compared separately as a secondary endpoint.

Definitions

Severe aortic stenosis was defined as AVA <1.0 cm² and elevated mean valve gradients >40 mm Hg determined by echocardiography. A periprocedural MI was considered when an increase of creatinine kinase ≥ 3 times the upper limit of normal was measured within 24-hr postprocedure, or any new pathological q-wave on electrocardiogram was obtained. Acute kidney injury was defined as a 0.5 mg/dl or greater increase in creatinine over baseline at 48 hr [15]. Glomerular filtration rate was estimated using the modification of diet in renal disease equation. Small left ventricle chamber size was defined as a left ventricular end-diastolic diameter <4.0 cm. Indexed values were calculated by dividing a variable by body surface area (BSA).

Statistical Analysis

Categorical variables among the four groups were compared by the χ^2 -test of homogeneity or Fisher's exact test for nonparametric data. Continuous variables were compared by an analysis of variance. Confounding and effect modification were evaluated using the Mantel-Haenszel method. Multivariate logistic regression was performed for the composite of in-hospital and procedural outcomes. The clinical and hemodynamic factors were evaluated for their relative risk of association with the composite outcome. Factors that were associated with the composite outcome in the unadjusted comparisons to a significance level of $P < 0.1$ were included in the adjusted model, in addition to prespecified factors: left ventricular ejection fraction (LVEF), Euroscore, and cardiac output. LVEDP and LVEF were included

as continuous variables. Model fit was evaluated using likelihood ratio testing. Analyses were performed using Intercooled STATA version 9.2 (Statacorp, College Station, TX).

RESULTS

Study Population

A total of 126 patients underwent retrograde BAV during the specified study period. Of these, 15 patients were excluded leaving a total study population of 111 patients. The mean age was 83 ± 11 years, and 56% of the patients were male. The mean LVEDP of the study group was 20.2 ± 9 mm Hg, and 15 mm Hg in 29 patients (26%), 16–20 mm Hg in 41 (37%), 21–25 mm Hg in 16 (14%), and 26 mm Hg in 25 (23%) patients. Demographic and hemodynamic characteristics were similar among the four study groups (Table I). Hemodynamic measures of success, including AVA, mean transvalvular gradient, cardiac index, and systemic blood pressure were similar following BAV regardless of the baseline LVEDP.

Study Outcomes

The results of the intraprocedural and in-hospital adverse events obtained in our study are summarized in Table II. There were a total of 20 intraprocedural and 23 in-hospital adverse events. Although not statistically significant, adverse intraprocedural events were more commonly observed among patients with highest LVEDP (>26 mm Hg; $P = 0.30$). Nonetheless, patients with LVEDP 21–25 mm Hg and 26 mm Hg had significantly higher rates of in-hospital adverse events than those with LVEDP 16–20 mm Hg and 15 mm Hg (LVEDP: $>26 = 36\%$; $21-25 = 37.5\%$; $16-20 = 9.8\%$; $15 = 13.8\%$; $P = 0.02$) (Table II). Patients with LVEDP 26 mm Hg had significantly higher rates of periprocedural MI, when compared with patients in all other categories ($P = 0.04$) (Table II). When compared with patients with LVEDP <21 mm Hg, those with LVEDP 21 mm Hg had on average significantly higher rates of in-hospital adverse events ($P < 0.01$; Fig. 1), and a higher trend to intraprocedural adverse events ($P = 0.06$).

The interaction between LVEF and LVEDP did not affect the association between LVEDP and clinical outcomes. Figure 2 shows the outcome of patients when further stratified according to their LVEF. Note that patients with preserved LV systolic function (i.e., LVEF $> 50\%$) and high LVEDP (≥ 21 mm Hg) did significantly worse than those with a depressed LVEF and normal LVEDP ($P = 0.01$).

After adjusting for age, gender, BSA, LVEF, Euro-score, and cardiac index, the LVEDP remained an independent predictor of in-hospital adverse events (OR 1.08, for each mm Hg increase in pressure; 95% CI 1.02–1.14), in addition to the NYHA class (OR 3.00; 95% CI 1.16–7.78), and small left ventricle chamber size (OR 3.78; 95% CI 1.01–14.09) (Table III). Of note, the LVEDP remained a significant independent predictor after adjusting for pre-BAV AVA and transvalvular gradient.

DISCUSSION

Our study demonstrates that an elevated LVEDP level of ≥ 21 mm Hg at the time of BAV is associated with significantly greater risk of in-hospital adverse events.

The LVEDP is an important hemodynamic measure of ventricular compensation in patients with both diastolic and systolic dysfunction. In our study, higher LVEDP and NYHA class, and not LVEF, were independently associated with greater rates of in-hospital death, MI, cardiopulmonary arrest, and tamponade. High LVEDP levels correlated with significantly worse outcome, regardless of the underlying LV systolic function. These findings suggest that the actual hemo-dynamic status of the left ventricle at the time of BAV, based on LVEDP and NYHA class, is more important in identifying patients at increased risk during the procedure than other baseline comorbidities, including LVEF. Our results indicate that the hemodynamic measures of procedural success including post-BAV AVA, transvalvular gradient, cardiac index, and mean aortic pressure were similar, regardless of the baseline LVEDP, suggesting the absence of a relationship between procedural success and in-hospital adverse events as defined in our study. Furthermore, the LVEDP remained a significant independent predictor of outcome even when adjusting for pre-BAV AVA and transvalvular gradient.

It is noteworthy that significantly higher rates of periprocedural MI were observed in patients with LVEDP ≥ 26 mm Hg. This finding is important and may be explained in part by the inverse physiologic relationship that exists between coronary perfusion and high LVEDP [16], as well as the increased myocardial oxygen demand that occurs with higher LV wall tension [17–19].

Although several predictors of BAV outcome have been previously identified and reported (Appendix Table), to our knowledge, the impact of the LVEDP has only been recognized in a single study by O'Neill et al. [23], whereby a greater survival post-BAV was observed among patients with a low LVEDP. This registry, however, dates back to the mid-to-late 1980s, which may not be reflective of contemporary outcomes and patient selection. Furthermore, the study fails to identify an LVEDP threshold above or below which a survival difference was clearly observed. Moreover, the model used to identify LVEDP as an independent clinical predictor of BAV outcome in this study failed to incorporate and adjust for LVEF. In the surgical literature, similar findings have been described among patients with compromised LV relaxation and diastolic dysfunction after surgery [10–12]. The independent predictive value of NYHA class found in our study is consistent with those previously reported by Lewin et al. [21], Dorros et al. [22], and by the NHLBI Balloon Valvuloplasty Registry Group [24].

We believe our study findings are clinically important and provide clinicians with a valuable tool when risk stratifying patients for BAV. It is possible that in patients with high LVEDP, ventricular unloading before BAV with either diuretics or inotropic support may be beneficial and associated with improved outcome. Other options to consider may include the concomitant use of a LV assist device during BAV. In a case reported by Londoño et al., the

use of an Impella 2.5 pump during BAV was safe and resulted in favorable hemodynamic support [27].

Limitations

As the study design is retrospective, unmeasured differences in baseline characteristics between the groups cannot be completely accounted for. It is possible that patients with elevated LVEDP are more likely to be acutely ill and that BAV may have been more commonly performed as a “salvage” procedure in these patients. The number of subjects included in our study is also relatively small and thus underpowered to detect real differences in mortality. We believe, however, that our study findings are clinically relevant and provide further insight to the outcome and prognosis of patients undergoing BAV. One may speculate that the LVEDP level is equally important on outcome of patients undergoing transcatheter valve implantation. Ultimately, however, further research will be necessary to confirm these thoughts.

CONCLUSIONS

The LVEDP is a significant independent predictor of worse in-hospital outcome, regardless of LVEF, cardiac output. In patients undergoing BAV, the periprocedure hemodynamic status may be more important than the baseline risk factors and systolic function. Ventricular unloading before or during BAV in patients with elevated LVEDP may be beneficial and result in lower risk of in-hospital adverse events.

APPENDIX

APPENDIX

PREVIOUS STUDIES OF PREDICTORS OF OUTCOME IN BAV

Reference	N	Outcome measures evaluated	Independent predictors of outcome
Sherman et al. [20]	36	Adverse events and mortality at 2, 8 and 26 weeks	LVEF, sPAP, PVR, RVEDP
Lewin et al. [21]	125	In-hospital death, MI, neurologic deficit. 12-month mortality and symptoms	Severe CHF, Pre-procedure LVEF, CO
Davidson et al. [4]	81	Clinical status Symptom recurrence	LVEF
Dorros et al. [22]	149	In-hospital mortality	NHYA class IV, LVEF, CO, Previous CAD
O'Neill et al. [23]	492	1-Year Survival & event-free survival	Higher LVESP, higher CO, lower LVEDP, greater final AVA, age, fewer balloon inflations
Davidson et al. [5]	170	1-Year cardiac death, AVR, repeat BAV	Baseline LVEF
NHLBI BV Registry Group [24]	674	30-Day mortality	SBP < 100 mm Hg, NYHA class IV, use of antiarrhythmics, CO < 3 l/min
Kuntz et al. [6]	205	Event-free survival at 40 months	LVEF, LV and aortic systolic pressure < 110 mmHg, PCWP > 25 mmHg, < 40% decrease in peak AVG
Otto et al. [25]	674	3-Year survival	Functional class, renal function, cachexia, female gender, severity of MR, LVEF, CO, mean AVG
Lieberman et al. [26]	165	1-Year event-free survival	Young age, low LVEF

Reference	N	Outcome measures evaluated	Independent predictors of outcome
Don et al. [7]	111	In-hospital death, MI, stroke, cardiac arrest, tamponade, emergent intubation	NYHA class
Elmariah et al. [8]	281	30-day mortality	Critical status, renal dysfunction, RAP, CO

Abbreviations: AVG, aortic valve gradient; CAD, coronary artery disease; CHF, congestive heart failure; CO, cardiac output; LV, left ventricle; LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction; LVESP, left ventricular end-systolic pressure; MR, mitral regurgitation; NYHA, New York Heart Association; PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RVEDP, right ventricular end diastolic pressure; sPAP, systolic pulmonary artery pressure.

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Fig. 1.

In-hospital adverse events* according to LVEDP. *The composite of in-hospital death, myocardial infarction, cardio-pulmonary arrest requiring resuscitation, and pericardial tamponade. †*P* value comparing all four categories is 0.02. ‡ χ^2 comparison between patients with LVEDP <21 mm Hg and 21 mm Hg; *P* < 0.01.

Fig. 2.

In-hospital adverse events* according to both LVEDP and LVEF. *The composite of in-hospital death, myocardial infarction, cardiopulmonary arrest requiring resuscitation, and pericardial tamponade. $^{\dagger}\chi^2$ comparison between those with LVEDP <21 mm Hg and ≥ 21 mm Hg in patients with preserved LV systolic function; $P = 0.08$. $^{\ddagger}\chi^2$ comparison between those with LVEDP <21 mm Hg and ≥ 21 mm Hg in patients with depressed LV systolic function; $P = 0.01$.

TABLE I

Demographic, Clinical, and Procedural Characteristics

	LVEDP (15 mm Hg)	LVEDP (16–20 mm Hg)	LVEDP (21–25 mm Hg)	LVEDP (26 mm Hg)	P
Age	83 ± 8	82 ± 8	87 ± 6	79 ± 9	0.47
Gender, male	15 (51.7)	26 (63.4)	8 (50.0)	13 (52.0)	0.68
Race, Caucasian	26 (92.9)	33 (89.2)	15 (93.8)	22/22 (100)	0.31
Body mass index	24.7 ± 7.2	24.2 ± 5.0	24.7 ± 5.2	26.9 ± 5.2	0.13
Tobacco history					0.35
Never	18 (62.1)	15 (37.5)	7 (43.6)	10 (40.0)	
Current	1 (3.5)	3 (7.5)	1 (6.3)	0 (0.0)	
Former	10 (34.5)	22 (55.0)	8 (50.0)	15 (60.0)	
Hypertension	22 (75.9)	35 (85.4)	12 (75.0)	18 (72.0)	0.57
Diabetes mellitus	8 (27.6)	12 (29.3)	5 (31.3)	11 (44.0)	0.57
Hyperlipidemia	22 (75.9)	35 (85.4)	13 (81.3)	20 (83.3)	0.78
Previous CAD	16 (55.2)	23 (56.1)	10 (62.5)	14 (56.0)	0.97
Triple vessel CAD	5 (17.2)	7 (17.1)	1 (6.3)	3 (12.0)	0.70
Family history of CAD	2 (6.9)	2 (4.9)	1 (6.3)	0 (0.0)	0.64
Previous myocardial infarction	7 (24.1)	12 (29.3)	5 (31.3)	6 (24.0)	0.92
Previous PCI	5 (17.2)	7 (17.1)	2 (12.5)	5 (20.0)	0.49
Previous CABG	9 (31.0)	7 (17.1)	4 (25.0)	4 (16.0)	0.46
History of CHF	21 (72.4)	32 (78.1)	12 (75.0)	16 (64.0)	0.66
NHYA class IV	12 (41.4)	15 (36.6)	8 (50.0)	14 (56.0)	0.44
Stroke	11 (37.9)	17 (41.5)	7 (43.6)	10 (40.0)	0.98
COPD	10 (34.5)	15 (36.6)	5 (31.3)	8 (32.0)	0.97
GFR <60 ml/min	20 (69.0)	26 (65.0)	11 (68.8)	15 (60.0)	0.90
Peripheral vascular disease	5 (17.2)	13 (31.7)	4 (25.0)	7 (28.0)	0.59
Left ventricular ejection fraction (%)	53.9 ± 19.1	55.8 ± 20.2	50.7 ± 20.1	43.7 ± 18.3	0.24
Pre-BAV hemodynamics					
Aortic valve area (mm)	0.7 ± 0.2	0.6 ± 0.2	0.6 ± 0.2	0.6 ± 0.2	0.82
Mean transvalvular gradient (mm Hg)	46.0 ± 12.8	47.7 ± 15.5	46.5 ± 16.5	47.2 ± 19.7	0.33
Cardiac index	2.4 ± 0.7	2.4 ± 0.7	2.2 ± 0.6	2.3 ± 0.6	0.63
Mean aortic pressure (mm Hg)	78.7 ± 12.5	82.1 ± 16.3	81.4 ± 14.8	82.4 ± 15.9	0.32
Post-BAV hemodynamics					
Aortic valve area (mm)	0.9 ± 0.3	0.9 ± 0.3	0.9 ± 0.3	0.9 ± 0.3	0.88
Mean transvalvular gradient (mm Hg)	28.8 ± 9.0	29.2 ± 12.6	27.0 ± 10.7	28.4 ± 13.7	0.85
Cardiac index	2.5 ± 0.7	2.4 ± 0.7	2.2 ± 0.6	2.3 ± 0.6	0.93
Mean aortic pressure (mm Hg)	88.1 ± 18.8	89.1 ± 18.9	97.7 ± 25.0	87.9 ± 14.1	0.94

Categorical variables are presented as *n* (%); continuous variables are presented as mean ± SD.

Abbreviations: BAV, balloon aortic valvuloplasty; CABG, coronary artery bypass graft; CAD, coronary artery disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; GFR, glomerular filtration rate.

TABLE II

Study Endpoint Results

	LVEDP (< 15 mm Hg)	LVEDP (16–20 mm Hg)	LVEDP (21–25 mm Hg)	LVEDP (> 26 mm Hg)	<i>P</i>
Intraprocedural adverse events	3 (10.3)	6 (14.6)	4 (25.0)	7 (28.0)	0.30
Vasopressor required	3 (10.3)	6 (14.6)	3 (18.8)	6 (25.0)	0.53
CPR required	1 (3.5)	2 (4.9)	4 (25.0)	3 (12.0)	0.07
Intubation required	0 (0.0)	2 (4.9)	3 (18.8)	2 (8.0)	0.09
Intraprocedural death	0 (0.0)	1 (2.4)	0 (0.0)	0 (0.0)	0.63
In-hospital adverse events	4 (13.8)	4 (9.8)	6 (37.5)	9 (36.0)	0.02
Any hospital death	2 (6.9)	1 (2.4)	3 (18.8)	3 (12.0)	0.19
Periprocedure MI	0 (0.0)	2 (4.9)	0 (0.0)	4 (16.0)	0.04
Vascular complications	4 (13.8)	8 (19.5)	5 (31.3)	5 (20.0)	0.58
Postprocedure AKI	2 (6.9)	2 (4.9)	2 (12.5)	2 (8.0)	0.79

Categorical variables are presented as *n* (%); continuous variables are presented as mean ± standard deviation.

Abbreviations: AKI, acute kidney injury; CPR, cardiopulmonary resuscitation; MI, myocardial infarction.

TABLE III Logistic Regression Analysis for Predictors of In-Hospital Adverse Events^a

	Unadjusted		Adjusted	
	Odds ratio	95% Confidence Interval	Odds ratio	95% Confidence Interval
Baseline LVEDP	1.0761	1.02–1.13	1.08	1.02–1.14
NYHA class	2.8936	1.23–6.80	3.0030	1.16–7.78
Baseline cardiac index	0.5804	0.27–1.23	0.77	0.33–1.90
Euroscore	2.8678	0.34–23.85	2.6636	0.19 –37.67
Left ventricular ejection fraction	0.9971	0.97–1.02	1.0022	0.97–1.04
Small left ventricle ^b	2.6190	0.99–6.92	3.7783	1.01–14.09

Of note, the LVEDP remained a significant independent predictor after adjusting for pre-BAV AVA and transvalvular gradient.

Abbreviations: BAV, balloon aortic valvuloplasty; LVEDP, left ventricular end-diastolic pressure; NYHA, New York Heart Association class.

^a Defined as composite endpoint of in-hospital death by any cause, myocardial infarction, cardiopulmonary arrest and tamponade.

^b Small left ventricle defined as left ventricular end-diastolic diameter <4 cm.