The Heart Surgery Forum #2006-1142
10 (1), 2007 [Epub December 2006]
doi: 10.1532/HSF98.20061142

Online address: http://cardenjennings.metapress.com

Evaluation of Left Ventricular Functions after Aortic Valve Replacement in a Specific Young Male Patient Population with Pure Aortic Insufficiency or Aortic Stenosis: 5-Years Follow-up

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ABSTRACT

Background. The aim of this study was to evaluate the left ventricular functions and the regression of left ventricular hypertrophy after aortic valve replacement (AVR) in young male patients with pure aortic stenosis or aortic insufficiency with no additional disease.

Methods. Young male patients who underwent AVR because of pure aortic stenosis (AS = 68) and insufficiency (AI = 70) were enrolled in the study. The mean age was 23.2 ± 1.3 and 22.6 ± 1.6 years, respectively. The follow-up time was 5 years. The parameters checked by transthoracic echocardiography were interventricular septum diastolic thickness, left ventricular posterior wall diastolic thickness, left ventricular end-diastolic diameter, left ventricle mass, left ventricle mass index, ejection fraction, and peak aortic gradient. Relative ventricle wall thickness was also calculated. Both groups values from the preoperative, postoperative sixth month, second year, and fifth year time intervals were compared.

Results. In the AS group, the preoperative left ventricular ejection fraction (%) value of 53.68 ± 5.04 increased to 63.24 ± 4.11 at the end of the fifth year. In the AI group, the preoperative left ventricular ejection fraction (%) value of 48.40 ± 3.56 increased to 59.77 ± 2.75 at the end of the fifth year. The other left ventricular geometric parameters were also compared within each group. At the end of the fifth year, there were significant and positive changes in each group.

Conclusion. The regression of the left ventricular parameters is a process that occurs over many years following the correction of the primary hemodynamic abnormality. Although the results were similar in the AI and AS group, in the AS group the remodeling process had earlier results than in the AI group.

Received September 19, 2006; accepted October 27, 2006.

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INTRODUCTION

Pure aortic valve stenosis and insufficiency are frequent reasons for prosthetic valve replacement in adults [Flachskampf 2004]. Patients with aortic valve stenosis (AS) and/or aortic insufficiency (AI) are subjected to increased pressure and volume load of the left ventricle (LV), leading to either concentric or eccentric left ventricular hypertrophy (LVH) [Monrad 1988; Orsinelli 1993]. The presence of LVH is associated with a 2- to 3-fold increase in cardiovascular-related mortality [Levy 1990; Devereux 1994; Benjamin 1999]. Regression of LVH was not complete in more than 50% of studied patients 1 to 2 years after operation [Lund 1998]. The normalization of LV mass (LVM) is a complex phenomenon that is determined by several patient and prosthesis-related factors [Lund 1998]. Prognostic studies have given rise to the hypothesis that regression of LVH is the underlying determinant of longevity after aortic valve replacement (AVR) [Lund 1987, 1990, 1996]. It is possible to say that incomplete regression of hypertrophy is a consequence of suboptimal hemodynamic function of the prosthetic aortic valve [Krayenbuehl 1989]. In the literature, there are not many articles dealing with the regression of LVH in a young patient population who underwent AVR for pure AS or AI and the long-term follow-up of these patients. The aim of this study is to evaluate the progression of left ventricular functions after AVR in young male patients with pure AS and AI without additional diseases over a long-term period.

MATERIALS AND METHODS

Preoperative Patient Selection

Young male patients (n = 156) without any additional disease that may affect the regression procedure and who underwent mechanical AVR due to pure AS (AS = 78) or pure AI (AI = 78) between December 1994 and January 2000 were enrolled in the study. The study was designed as a prospective study. Since our hospital is the only and largest referral military hospital in Turkey, many young soldiers with AS and AI were admitted to our department for treatment. Young male patients (the youngest was 18 and the oldest was 25 years) with pure AI or pure AS with an indication for AVR were included in the study. The mean age of the patients was 23.2 ± 1.3 (AS group) and 22.6 ± 1.6 (AI group). The patients with additional congenital heart

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Table 1. Preoperative Demographic Data of the Patients*

	AS (n = 68)	AI (n = 70)
Age, y	23.2 ± 1.3	22.6 ± 1.6
HR, beats/min	72.2 ± 15.0	71.1 ± 9.8
SBP, mmHg	115.4 ± 12.3	128.2 ± 25.2
DBP, mmHg	68.2 ± 7.8	61.1 ± 6.2
PAG, mmHg	88.9 ± 19.7	12.1 ± 4.3
AVA, cm^2	0.66 ± 0.9	2.93 ± 0.7
BSA, m^2	1.76 ± 0.17	1.77 ± 0.15
RG for AI group only	—	3.79 ± 0.42
Functional capacity, NYHA		
Class I	—	—
Class II	47 (69.1%)	46 (65.7%)
Class III	21 (30.9%)	24 (34.3%)
Class IV	—	—
Preoperative LVEF, %		
40-50	16 (23.5%)	31 (44.3%)
50-60	42 (61.8%)	34 (48.6%)
>60	10 (14.7%)	5 (7.1%)
Etiologic reasons		
Rheumatismal disease	23 (33.8%)	29 (41.4%)
Infective endocarditis	20 (29.4%)	21 (30.0%)
Mycotic infections	8 (11.8%)	15 (21.4%)
Bicuspid aorta	17 (25.0%)	—
Marfan syndrome	—	5 (7.2%)

*AS indicates aortic stenosis; AI, aortic insufficiency; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; PAG, peak aortic gradient; AVA, aortic valve area; BSA, body surface area; RG, regurgitation grade; NYHA, New York Heart Association; LVEF, left ventricle ejection fraction.

disease (atrial septal defect, ventricular septal defect, patent ductus arteriosus, aortic coarctation, pulmonary stenosis, aneurysm of sinus valsalva) were excluded from the study. Suitable candidates were counseled before offering informed consent. The study was approved and monitored by the Local Human Research Ethics Committee of our institute. The follow-up period was 5 years, and all of the patients were monitored during this period. Eighteen patients were excluded from the study in the early postoperative and during the follow-up period. In the AS group, excluded patients were 4 in whom aortic root enlargement was performed, 3 in whom number 19–sized valves were implanted, 1 with prosthetic valve endocarditis, and 2 were excluded because of loss of contact. In the AI group, excluded patients were 3 patients with paravalvular leakage, 2 with prosthetic valve endocarditis, and 3 were excluded because of loss of contact. Finally, the study consisted of 138 patients, 68 in the pure AS group and 70 in the pure AI group. The demographic parameters of the patients are detailed in Table 1.

Operative Procedure

All of the patients underwent standard AVR procedure under cardiopulmonary bypass with general hypothermia, topical cooling, and cardiac arrest using crystalloid cardioplegia. We used the same type of prosthetic valves (Bileaflet Heart Valve Regent [for size 21] and Masters Series, St. Jude, St. Paul, MN, USA) for all patients. In the AS group, we used size 21 prosthetic valves in 42 patients and size 23 valves in 26 patients. In the AI group, we used size 23 prosthetic valves in 31 patients and size 25 valves in 39 patients. We tried to provide the optimal orientation of the prosthetic device to avoid turbulent flow and aortic energy loss, which may negatively affect the regression process.

Follow-up Period

There was no operative or hospital mortality. All of the patients except the 5 with whom we lost contact as mentioned above (2 in the AS group and 3 in the AI group) were followed for 5 years, with control period follow-ups in the sixth month, second year, and fifth year. At the follow-up, the patients were assessed by 12-lead electrocardiography, telegraphy, transthoracic echocardiography, and blood tests.

Preoperative and Postoperative Echocardiographic Evaluation

The preoperative and postoperative (sixth month, second year, and fifth year) assessments of the patients were made with the same device (Hewlett Packard Console 2500, Palo Alto, CA, USA). Images were made with patients in the left lateral decubitus position using a commercially available system. The echocardiographic evaluations of the patients were made by the same cardiologist, and the cardiologist was blinded for the patients at the postoperative follow-up controls. Echocardiographic images were obtained using a 3.5-Mhz transducer in standard view. Left ventricular dimensions and wall thickness were determined from parasternal long axis using M-mode at the level of chordae. Left ventricular fractional shortening (LVFS) was calculated from M-mode data according to the standard formula:

$$\text{LVFS} = [(\text{EDD} - \text{ESD})/\text{EDD}] \times 100$$

where EDD indicates left ventricular end-diastolic diameter (LVEDD) and ESD indicates left ventricular end-systolic diameter (LVESD). The biplane modified Simpson's method was used for the determination of left ventricular volumes and ejection fraction (using apical 4- and 2-chamber views). The pulsed and continuous Doppler interrogation of the aortic valve and the Bernouilli equation was used for calculation of peak and mean gradients in patients with AS. Continuity equation was also used for the aortic valve area calculation. In patients with aortic regurgitation, regurgitation severity was determined according to jet width/left ventricular outflow height ratio as grade 1 (<25%), grade 2 (25%-46%), grade 3 (47%-64%), and grade 4 (>65%). LVM was calculated as described by Devereux's formula (Penn convention):

$$\text{LVM (g)} = 0.8 \times \{1.04 \times [(\text{LVEDD} + \text{LVPWDT} + \text{IVSDT})^3 - \text{LVEDD}^3]\} + 0.6$$

where LVPWDT indicates left ventricular posterior wall diastolic thickness; IVSDT, interventricular septum diastolic thickness. LVM was also normalized for body surface area and for height [Gaudino 2005]. The preoperative and postoperative echocardiographic measurements of the patients are detailed in Table 2.

Table 2. Assessment and Comparison of Left Ventricular Geometric Parameters of the Patients in the Aortic Insufficiency (AI) Group during the Follow-up Period*

Parameter	Follow-up Period	Values	*P*
LVM	Preoperative	285.06 ± 81.32	$P_1 = .062$
	Postoperative sixth month	265.94 ± 60.34	$P_2 = .001$
	Postoperative second year	234.38 ± 67.51	$P_3 < .001$
	Postoperative fifth year	211.48 ± 51.45	$P_4 = .011$
			$P_5 = .001$
			$P_6 = .053$
LVMI, g/m^2	Preoperative	196.94 ± 38.35	$P_1 = .059$
	Postoperative sixth month	178.39 ± 25.19	$P_2 < .001$
	Postoperative second year	129.51 ± 31.91	$P_3 < .001$
	Postoperative fifth year	123.19 ± 25.84	$P_4 = .002$
			$P_5 < .001$
			$P_6 = .061$
LVEDD, mm	Preoperative	63.37 ± 4.74	$P_1 = .076$
	Postoperative sixth month	61.92 ± 3.15	$P_2 = .043$
	Postoperative second year	57.37 ± 3.87	$P_3 = .021$
	Postoperative fifth year	54.92 ± 3.81	$P_4 = .038$
			$P_5 = .002$
			$P_6 = .072$
IVSDT (mm)	Preoperative	14.37 ± 1.20	$P_1 = .081$
	Postoperative sixth month	14.15 ± 1.93	$P_2 = .048$
	Postoperative second year	12.72 ± 1.26	$P_3 = .032$
	Postoperative fifth year	11.78 ± 1.13	$P_4 = .076$
			$P_5 = .045$
			$P_6 = .079$
LVPWDT	Preoperative	14.65 ± 1.66	$P_1 = .083$
	Postoperative sixth month	14.40 ± 1.03	$P_2 = .050$
	Postoperative second year	12.25 ± 1.93	$P_3 = .027$
	Postoperative fifth year	11.57 ± 1.05	$P_4 = .055$
			$P_5 = .047$
			$P_6 = .078$
LVEF, %	Preoperative	48.40 ± 3.56	$P_1 = .074$
	Postoperative sixth month	50.15 ± 3.49	$P_2 = .041$
	Postoperative second year	56.72 ± 2.85	$P_3 = .019$
	Postoperative fifth year	59.77 ± 2.75	$P_4 = .048$
			$P_5 = .033$
			$P_6 = .072$
PAG, mmHg	Preoperative	12.1 ± 4.3	SNC
	Postoperative sixth month		
	Postoperative second year		
	Postoperative fifth year		
RWT	Preoperative	>0.45	
	Postoperative sixth month	>0.45	SNC
	Postoperative second year	<0.45	
	Postoperative fifth year	<0.45	

*LVM indicates left ventricle mass; P_1, comparison between preoperative period and postoperative sixth month; P_2, comparison between preoperative period and postoperative second year; P_3, comparison between preoperative period and postoperative fifth year; P_4, comparison between postoperative sixth month and postoperative second year; P_5, comparison between postoperative sixth month and postoperative fifth year; P_6, comparison between postoperative second year and postoperative fifth year; LVMI, left ventricle mass index; LVEDD, left ventricle end-diastolic diameter; IVSDT, interventricular septum diastolic thickness; LVPWDT, left ventricle posterior wall diastolic thickness; LVEF, left ventricle ejection fraction; PAG, peak aortic gradient; SNC, statistically not compared; RWT, relative wall thickness.

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Table 3. New York Heart Association Evaluation of the Patients

	Aortic Stenosis Group				Aortic Insufficiency Group			
	Preoperative	Sixth Month	Second Year	Fifth Year	Preoperative	Sixth Month	Second Year	Fifth Year
Class I	—	6 (8.8%)	23 (33.8%)	51 (75.0%)	—	4 (5.7%)	19 (27.1%)	43 (61.4%)
Class II	21 (69.1%)	27 (39.7%)	42 (61.8%)	17 (25.0%)	24 (34.3%)	36 (51.4%)	38 (54.3%)	27 (38.6%)
Class III	47 (30.9%)	35 (51.5%)	3 (4.4%)	—	46 (65.7%)	30 (42.9%)	13 (18.6%)	—
Class IV	—	—	—	—	—	—	—	—

Evaluation of the Left Ventricular Geometric Parameters

All of the patients in each group were individually evaluated. LVM, LVM index (LVMI), LVEDD, IVSDT, LVPWDT, LVEF, peak aortic gradient (PAG), and relative wall thickness (RWT) parameters were compared preoperatively and in the postoperative sixth month, second year, and fifth year in each group separately in follow-up periods. The results of each parameter were compared in 6 time intervals (preoperative-postoperative sixth month, preoperative-postoperative second year, preoperative–postoperative fifth year, postoperative sixth month–postoperative second year, postoperative sixth month–postoperative fifth year, postoperative second year–postoperative fifth year).

Statistical Analysis

Statistical analysis was performed with SPSS software version 11.0 (Chicago, IL, USA). The nominal variables of the groups were presented as percentages, mean ± standard deviation, and were compared using 1-way analysis of variance with Bonferroni correction. Preoperative demographic data of both groups were compared using nonparametric independent samples t test. Functional capacity, LVEFs, and etiologic factors were compared with nonparametric chi-square test. A P value < .05 was considered statistically significant.

RESULTS

The mean ages were 23.2 ± 1.3 in the AS group and 22.6 ± 1.6 years in the AI group. The mean valve area in the AS group was 0.66 ± 0.9, and the mean regurgitation grade in AI group was 2.93 ± 0.7. For all of the patients in the AS group, the peak gradient was over 60 mmHg (mean gradient, 88.9 ± 19.7). The preoperative data of the patients are given in Table 1. There was no statistically significant difference between the demographic data of the patients. All of the preoperative symptoms vanished in both groups after surgery. At the fifth year, the functional capacity of all of the patients was class 1 or 2 according to New York Heart Association (NYHA) classification. The NYHA classifications of the patients at the preoperative and postoperative follow-up periods are detailed in Table 3. There was no operative or hospital mortality. During the follow-up period, 2 patients had gastrointestinal bleeding due to warfarin.

Comparison of Left Ventricular Geometric Parameters

The left ventricular parameters in both groups were measured and compared (Tables 2 and 4). At the postoperative sixth month in both groups there were not significant changes in the left ventricular geometric parameters. The prominent changes in AS began to return to normal after the postoperative eighteenth month. In the AI group, the significant changes began to return to normal after one year.

DISCUSSION

After AVR, relief of symptoms and improvement in the quality of life and survival are expected. Significant improvement of left ventricular diastolic function and functional capacity of the patients can be observed with regression of myocardial hypertrophy and interstitial fibrosis. The most important factor that affects left ventricular mass regression is the gradient on the prosthetic valve. However, the most important reason for insufficient mass regression is the increase in left ventricular pressure, caused by an extreme gradient that can develop on the prosthesis. Factors such as age, sex, diabetes mellitus, coronary artery disease, the class of left ventricular function, type of replacement device, and use of cardioplegia are other factors that affect the regression in LVH [Jin 1995, 1996; Gaudino 2005]. In our study, we tried to exclude these factors by studying a young male patient group with pure AS and AI without any other additional disease. Also we used the same prosthetic valve with different sizes and the same cold cardioplegia solution (St Thomas II) with different volumes. In addition, hypertension and atrial fibrillation have a negative impact on the regression of LVH [Gaudino 2005], but none of our patients had either hypertension or atrial fibrillation.

Gaasch et al [1983] found that in most patients with aortic regurgitation, left ventricular end-diastolic dimensions and volumes became near normal within 2 weeks after AVR, whereas a significant regression of LVH took at least 6 months. In our study, there was a slight change in LV remodeling parameters in the sixth month for both groups. Nevertheless, the changes in parameters for both groups were much more prominent in the second year in our study. In the AS group, remodeling and an increase in the LVEF was found to be a continuing process that caused a significant improvement at the fifth year; the increase in LVEF began within 6 months after surgery as a result of decreases in left ventricular end-diastolic and end-systolic indices [Hwang 1989]. In contrast, in the AI group

Table 4. Assessment and Comparison of Left Ventricular Geometric Parameters of the Patients in the Aortic Stenosis (AS) Group during the Follow-up Period*

Parameter	Follow-up Period	Values	*P*
LVM	Preoperative	261.93 ± 21.05	$P_1 = .058$
	Postoperative sixth month	245.39 ± 16.95	$P_2 < .001$
	Postoperative second year	163.78 ± 16.23	$P_3 < .0001$
	Postoperative fifth year	141.43 ± 17.33	$P_4 < .001$
			$P_5 < .001$
			$P_6 = .064$
LVMI, g/m^2	Preoperative	149.98 ± 16.59	$P_1 > .05$
	Postoperative sixth month	134.82 ± 13.88	$P_2 < .001$
	Postoperative second year	93.87 ± 12.57	$P_3 < .0001$
	Postoperative fifth year	79.68 ± 12.99	$P_4 = .002$
			$P_5 < .001$
			$P_6 = .063$
LVEDD, mm	Preoperative	59.77 ± 3.84	$P_1 = .083$
	Postoperative sixth month	57.68 ± 2.75	$P_2 = .038$
	Postoperative second year	53.02 ± 1.84	$P_3 = .019$
	Postoperative fifth year	52.25 ± 1.28	$P_4 = .032$
			$P_5 = .021$
			$P_6 = .074$
IVSDT, mm	Preoperative	13.90 ± 1.74	$P_1 = .069$
	Postoperative sixth month	12.07 ± 1.72	$P_2 = .048$
	Postoperative second year	11.12 ± 1.93	$P_3 = .022$
	Postoperative fifth year	9.65 ± 1.63	$P_4 = .071$
			$P_5 = .043$
			$P_6 = .056$
LVPWDT	Preoperative	13.32 ± 1.86	$P_1 = .068$
	Postoperative sixth month	12.37 ± 1.93	$P_2 = .051$
	Postoperative second year	11.65 ± 1.72	$P_3 = .027$
	Postoperative fifth year	9.02 ± 1.63	$P_4 = .066$
			$P_5 = .053$
			$P_6 = .058$
LVEF, %	Preoperative	53.68 ± 5.04	$P_1 = .075$
	Postoperative sixth month	54.90 ± 4.45	$P_2 = .031$
	Postoperative second year	61.70 ± 3.84	$P_3 = .015$
	Postoperative fifth year	63.24 ± 4.11	$P_4 = .047$
			$P_5 = .035$
			$P_6 = .062$
PAG, mmHg	Preoperative	88.9 ± 19.7	SNC
	Postoperative sixth month	23.2 ± 8.4	
	Postoperative second year	21.9 ± 7.3	
	Postoperative fifth year	20.3 ± 5.8	
RWT	Preoperative	>0.45	SNC
	Postoperative sixth month	>0.45	
	Postoperative second year	<0.45	
	Postoperative fifth year	<0.45	

*LVM indicates left ventricle mass; P_1, comparison between preoperative period and postoperative sixth month; P_2, comparison between preoperative period and postoperative second year; P_3, comparison between preoperative period and postoperative fifth year; P_4, comparison between postoperative sixth month and postoperative second year; P_5, comparison between postoperative sixth month and postoperative fifth year; P_6, comparison between postoperative second year and postoperative fifth year; LVMI, left ventricle mass index; LVEDD, left ventricle end-diastolic diameter; IVSDT, interventricular septum diastolic thickness; LVPWDT, left ventricle posterior wall diastolic thickness; LVEF, left ventricle ejection fraction; PAG, peak aortic gradient; SNC, statistically not compared; RWT, relative wall thickness.

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remodeling and an increase in LVEF was significant at the second postoperative year, but during the second and fifth years there was not a significant change. Some studies have shown that several patients with aortic regurgitation who had a severely depressed LVEF and a strongly dilated LV before surgery failed to achieve regression of LVH after AVR [Roman 1989; Bech-Hanssen 1999]. In patients with aortic valve stenosis, the presence of a supernormal ejection fraction and "disproportionately high" RWT before AVR is associated with an excessive perioperative risk of morbidity and mortality [Monrad 1988; Aurigemma 1994]. Alternatively, AS patients with a low RWT and eccentric hypertrophy showed decreased systolic function as well as symptoms of heart failure [Bonow 1988]. Lamb et al [2002] compared the LVEF and left ventricular geometric parameters in 19 patients before and after AVR with 10 control patients. In 12 patients with aortic valve stenosis, LVMI decreased from 126.3 ± 33.1 g/m^2 to 87.5 ± 23.6 g/m^2 postoperatively ($P < .0001$). In 7 patients with aortic regurgitation, LVMI decreased from 146.5 ± 38.2 g/m^2 to 119.1 ± 29.0 g/m^2 postoperatively ($P <. 05$). In both groups of patients, LVMI after surgery was still significantly higher than LVMI in control subjects (68.6 ± 7.9 g/m^2; $P < .05$). After surgery, LVMI was significantly higher in patients with aortic regurgitation than in patients with aortic valve stenosis ($P < .05$). Lund et al [2003] reported the following results of patients who underwent AVR for AS after a 10-year follow-up. LVMI was 202 ±46 g/m^2 preoperatively ($n = 91$), 157 ± 48 g/m^2 at 1.5 years (n = 42; P < .0001), and 159 ± 70 g/m^2 at 10 years (n = 47; P < .0001). In our study the LVMI decrease was similar in the postoperative early period, but at the fifth year the decrease in the AS group was more than in the AI group. Tables 2 and 4 show the significant improvement in left ventricular geometric parameters in the AS and AI groups. In the AS group, improvement in LVMI, LVEDD, LVPWDT, and IVSDT was significant after the operation until the fifth postoperative year; this improvement meant a gradual remodeling. In contrast, the improvement in these parameters in AI patients seemed to be fast during the first 2 years after operation, but then slowed down. In the AI group, the increase in LVEF and remodeling was significant in the fifth year if compared to preoperative values but not significant when compared to the postoperative sixth month. Also there was not a prominent difference between the second and fifth years. In the AS group, improvement in LVEF and remodeling of the LV was continuing during the second and fifth postoperative years.

CONCLUSION

In patients with AVR, the regression of left ventricular parameters is a process that occurs over many years following the correction of the primary hemodynamic abnormality. Many factors affect this process. In our study, we tried to exclude most of these factors by studying a unique group and observing the changes during the 5-year follow-up. Although the results were similar in the AI and AS group, in the AS group the remodeling process had earlier results than in the AI group. As this process of myocardial remodeling occurs, continued improvement in cardiac function may occur.

Study Limitations

In our study, we compared the regression of left ventricular parameters with only mechanical prosthetic valves and did not include other types of prosthetic valves. Also we did not compare the results with an older age group. We have organized a new study in which we will compare the regression parameters in young patients to those in patients over the age of 60 who underwent mechanical prosthetic valve replacement.

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