Evaluation of Post-Stenotic Aortic Dilatation in Patients with Structurally Normal (Tricuspid) Aortic Valve Stenosis

Yapısal Olarak Normal (Triküspit) Aort Kapak Darlığı Olan Hastalarda Stenoz Sonrası Aort Dilatasyonunun Değerlendirilmesi

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ABSTRACT

Objective: The purpose of this study is to assess the impact of aortic valve stenosis on aortic aneurysm development in patients with normal aortic valve structure.
Methods: The echocardiographic images were obtained from 30 patients prospectively and 118 patient records were assessed retrospectively. The mean age of the population was 49.2±17.3y. The aortic dimensions were examined by 2D and 3D echocardiography from prospectively enrolled patients. Echocardiography records were used for retrospective analysis.
Results: A total of 148 patients were enrolled. No difference was observed among groups for traditional cardiovascular risk factors and medication use characteristics. Patients with severe aortic stenosis had more hypertrophied myocardial walls (ANOVA, p<0.001). The left atrial dimension was also higher for patients for severe aortic stenosis. No differences were observed among groups for aortic root dimensions in both retrospective and prospective cohorts. The Framingham risk score (FRS) (P=0.006) was the strongest and only significant determinant of having aortic root dilatation.
Conclusion: Aortic dilatation in patients with aortic stenosis is associated with higher FRS and the degree of aortic stenosis seems to be irrelevant with the prevalence of aortic dilatation. 3D echocardiography is useful for the evaluation of aortic root measurements and provides higher diameters than 2D echocardiography.

Keywords: Aorta, bicuspid, dilatation, echocardiography, stenosis, tricuspid.

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ÖZET

Amaç: Bu çalışmanın amacı, aort kapak yapısı normal olan hastalarda aort kapak stenozunun aort anevrizması gelişimi üzerindeki etkisini değerlendirmektir.
Yöntemler: Ekokardiyografik görüntüler 30 hastadan prospektif olarak alındı ve 118 hasta kaydı retrospektif olarak değerlendirildi. Nüfusun ortalama yaşı 49.2 ± 17.3 yıldı. Prospektif olarak kaydedilen hastalardan alınan 2D ve 3D ekokardiyografik görüntüler ile aort boyutları ile incelendi. Retrospektif analiz için ekokardiyografi kayıtları kullanıldı.
Sonuçlar: Toplam 148 hasta dahil edildi. Geleneksel kardiyovasküler risk faktörleri ve ilaç kullanım özellikleri açısından gruplar arasında fark gözlenmedi. Ciddi aort darlığı olan hastalarda daha hipertrofik miyokard mevcuttu (ANOVA, p <0.001). Sol atriyum boyutu da ciddi aort darlığı olan hastalarda daha yüksekti. Hem retrospektif hem de prospektif kohortlarda aort kökü boyutları için gruplar arasında hiçbir farklılık gözlenmedi. Framingham risk skoru (FRS) (P = 0.006) aort dilatasyonu varlığının en güçlü ve tek anlamlı belirleyicisiydi.
Sonuç: Aort darlığı olan hastalarda aort dilatasyonu daha yüksek FRS ile ilişkilidir ve aort darlığının derecesi aort dilatasyonu prevalansı ile ilişkisiz görünmektedir. 3D ekokardiyografi, aort kökü ölçümlerinin değerlendirilmesi için yararlıdır ve 2D ekokardiyografiden daha yüksek ölçümler sağlar.

Anahtar Sözcükler: Aort, biküspit, dilatasyon, ekokardiyografi, darlık, triküspit

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INTRODUCTION

Aortic stenosis is the most common valvular heart disease in developed countries (1,2). The prevalence of aortic stenosis increases with age (1,2). Underlying aetiologies can be congenital (bicuspid valves), rheumatic, or senile. Senile aortic stenosis is a chronic progressive disease that begins with sclerosis of the valve (1,2). The primary curative therapy of severe symptomatic aortic stenosis is surgery - aortic valve replacement. However, with the new developments in percutaneous treatment options, transcatheter aortic valve implantation has been introduced as an alternative treatment (3).

Post-stenotic aortic dilatation is defined as the dilatation of the aortic wall which locates at the distal area of partial stenosis (4). It refers to a degree of dilatation of the ascending aorta, exceeding 4.0 cm. Post-stenotic aortic dilatation has been reported to be mostly observed in the abdominal aorta. However, post stenotic dilation is especially defined in small vessels as a long term complication of a stenosis (4–7). Although the presence of a relation between aortic stenosis and post-stenotic aortic dilatation had been highly accepted, no studies proving the accepted concept (4–11). Various studies have shown that the aortic wall can withstand pressure load and jet flow turbulence more than other vessels due to its histological structure so post-stenotic aortic dilatation would not occur due to overpressure and turbulence caused by aortic stenosis. It has also been suggested post-stenotic aortic dilatation occurs only in patients with structural aortic abnormalities like bicuspid aortic valves (4,5,7,9–13).

Therefore, we attempted to evaluate the association between the aortic aneurysm and aortic stenosis in patients with the normal (tricuspid) aortic valve by using 2D and 3D echocardiography.

METHODS

Study Population

This study was planned both as a prospective cohort of 30 patients and a retrospective analysis of 118 patients between 2018 and 2020 followed in the Cardiology Department, Gazi University Medical School, Ankara. Patients with aortic stenosis between 18 and 85 years of age were included in the study. Patients with the bicuspid aortic valve and metallic prosthetic valve before aortic surgery were excluded. A control group (n= 50) was established with matching atherosclerotic risk factors among patients who had aortic sclerosis. Patients with severe aortic regurgitation were excluded from the study. Patients who were registered in the prospective cohort were informed of the study protocol and written informed consent was obtained. This study was approved by the local ethical committee on 23/12/2019 with decision number 12.

Study Protocol

Evaluation of Risk Factors

Clinical information related to participants´ personal and general information such as age, gender, height, weight, body mass index, medical history, and the treatments received; results of blood tests that were performed in outpatient clinics were recorded. History of hypertension, hypercholesterolemia, diabetes mellitus, smoking habit, and family history was carefully noted. Three classes recorded the smoking status; active smokers, never smokers, and former smokers. Criteria for a positive family history of coronary artery disease were that males before the age of 55 and females before the age of 65 in a first-degree relative had a history of fatal/nonfatal myocardial infarction, or coronary artery bypass surgery or coronary angioplasty. The Framingham risk score was calculated for each participant. The Framingham risk score was calculated by using a multivariable algorithm which includes age, total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, smoking status and diabetes.(14) Echocardiographic findings were recorded.

Echocardiography

An experienced sonographer (SU) performed all exams with a GE Vivid E95 Dimension ultrasonography machine equipped with 2D M5Sc-D and 4V-D Probe (GE Vingmed Ultrasound, Horten, Norway) for prospective analysis. In a wide room, an exam bed and ultrasound machine were designed to improve the comfort and mobility of the participants. Electrocardiogram and respiration of the patients were monitored during the examination. Echocardiographic images with at least three cardiac cycles were recorded.

For 3D acquisitions, the wide-angle full-volume mode was used during breath-hold in a single expiration to ensure that the entire left ventricle was included in the pyramidal scan volume. For 6 beats, multi-beat images were captured consecutively to obtain data sets of appropriate image quality. The average frame rate was 22 ± 4 frames /sec for 3D images. The acquired ECG-linked images were transmitted for review to the offline EchoPac v201 (GE Vingmed Ultrasound, Horten, Norway) station. Linear dimensions of the left ventricle and left atrium were measured in the parasternal long-axis. Left ventricular volumes and ejection fraction were calculated by the biplane method of disks summation in the apical four- and apical two-chamber views.

The 2D echocardiographic examination of the aorta was performed in the parasternal long-axis to obtain the maximum aortic root diameter during systole. The corresponding 3D echocardiographic acquisitions were independently analyzed, using GE software (EchoPac v201 (GE Vingmed Ultrasound, Horten, Norway). The 3D echocardiographic images were analyzed to find the aortic root image in the short axis having the inner borders being most visible. The signal-to-noise ratio was optimized by adjusting brightness and contrast. The 3D images were manipulated in various planes to obtain the maximum diameter of the aortic root.

Statistical Analysis

Continuous variables given as mean ± standard deviation were investigated by using the Kolmogorov-Smirnov test to determine whether they have a normal distribution. Paired t-test and Wilcoxon test were employed to compare parametric and nonparametric continuous variables, respectively. Categorical variables presented as percentages or frequencies were analyzed by using the Chi-square ($\chi 2$) test. The multivariate linear regression model was constructed to include conventional risk factors for atherosclerosis, and variables with a univariate relationship ($P<0.1$). The model was performed by a block entry option. Multivariate logistic regression models were utilized to evaluate the relationship among conventional cardiovascular risk factors, aortic stenosis severity, and post stenotic aortic dilatation. All statistical analyses were performed by SPSS version 23.0. In the two-tailed tests, a p-value of ≤0.05 was used as a significance level.

RESULTS

A total of 148 patients were enrolled in the final analysis. Baseline demographics, clinical characteristics, and medication use characteristics of patients with aortic stenosis and control group were described in Table 1, and findings of blood analysis were presented in Table 2. There was no difference among groups for traditional cardiovascular risk factors and medication use characteristics. In general, the study population mainly consisted of men (63.8%).

Table 1. Baseline characteristics of the participants.

Parameters	Aortic sclerosis (n=50)	Aortic stenosis (n=98)			
		Mild (n=36)	Moderate (n=42)	Severe (n=20)	P
Age (year±SD)	67.2 ± 14.4	68.7 ± 12.1	68.7 ± 11	69.2.5 ± 10	0.852
Sex (male, %)	33 (66%)	22 (61.7%)	24 (57.1%)	13 (65%)	0.838
BMI (kg/m2)	26.5 ± 3.2	26.2 ± 3.8	26.6 ± 3.2	27.3 ± 5.3	0.870
NYHA (class I)	46 (92%)	33 (91.7%)	41 (97.6%)	17 (85%)	0.349
SBP (mmHg)	130.6 ± 20.3	127 ± 18.7	137 ± 26.8	125.7 ± 26.6	0.301
DBP (mmHg)	81.4 ± 10.8	77.5 ± 9	81.1 ± 13.7	75.5 ± 13	0.289
Diabetes mellitus (%)	10 (20.4%)	15 (42.9%)	9 (21.4%)	6 (30%)	0.101
Hypertension (%)	37 (74%)	26 (72.2%)	30 (71.4%)	14 (70%)	0.986
Hyperlipidemia (%)	15 (30%)	9 (25%)	15 (35.7%)	10 (50%)	0.262
Smoking (%)	9 (18%)	5 (13.9%)	9 (22%)	4 (20%)	0.835
Coronary artery disease (%)	18 (36%)	12 (33.3%)	16 (39%)	8 (40%)	0.945
Chronic heart failure (%)	11 (22%)	14 (38.9%)	10 (24.4%)	8 (40%)	0.215
Stroke, TIA (%)	4 (8%)	4 (11.1%)	1 (2.4%)	1 (5%)	0.247
Aspirin (%)	17 (34%)	13 (36.1%)	15 (36.6%)	12 (60%)	0.215
Clopidogrel (%)	5 (10%)	3 (8.3%)	4 (9.8%)	2 (10%)	0.832
ACEI or ARB (%)	29 (58%)	17 (47.2%)	19 (46.3%)	14 (70%)	0.260
Beta Blockers (%)	33 (66%)	22 (61.1%)	32 (78%)	12 (60%)	0.347
Ca channel blockers (%)	16 (32%)	11 (30.6%)	12 (29.3%)	2 (10%)	0.268
Statins (%)	14 (28.6%)	9 (25%)	15 (36.6%)	8 (40%)	0.560

ACEI; angiotensin converting enzyme inhibitors, ARB; angiotensin II receptor blockers, BMI; body mass index, DBP; diastolic blood pressure, NYHA; New York Heart Academy, SBP; systolic blood pressure, TIA; transient-ischemic attack

Table 2. Basal laboratory test findings of the participants.

Parameters	Aortic sclerosis (n=50)	Aortic stenosis (n=98)			P
		Mild (n=36)	Moderate (n=42)	Severe (n=20)	
Blood urea nitrogen (mg/dl)	23.5 ± 15.8	21 ± 9.9	25 ± 20.4	32.9 ± 22.5	0.097
Creatinin (mg/dl)	1.3 ± 1.4	1 ± 0.4	1.2 ± 0.8	1.1 ± 0.6	0.488
Na (mmol/l)	139.1 ± 3.7	139.3 ± 4	139.5 ± 3.1	140.4 ± 3.7	0.751
K (mmol/l)	4.23 ± 0.5	4.4 ± 0.5	4.3 ± 0.5	4.5 ± 0.3	0.61
Ca (mg/dl)	9.3 ± 0.7	9.5 ± 0.6	9.1 ± 0.7	9.3 ± 0.6	0.147
P (mg/dl)	3.4 ± 0.9	3.7 ± 0.6	3.7 ± 0.6	3.8 ± 0.7	0.098
Albumine (g/dl)	3.8 ± 0.7	3.9 ± 0.8	3.9 ± 1.2	3.6 ± 0.5	0.702
Alanine transaminase (U/L)	22 ± 20.7	17.1 ± 7.3	15.8 ± 6	23.8 ± 21.2	0.104
Aspartate aminotransferase (U/L)	25.4 ± 17.3	20.2 ± 6.3	20.3 ± 7.3	31.6 ± 32.8	0.131
Alkaline Phosphatase (U/L)	96.6 ± 54.4	97.8 ± 41.7	81.1 ± 32.2	95.9 ± 22.7	0.276
Total Bilirubin (mg/dl)	0.9 ± 0.8	0.6 ± 0.4	0.7 ± 0.4	0.9 ± 0.5	0.120
Direct Bilirubin (mg/dl)	0.3 ± 0.5	0.2 ± 0.1	0.2 ± 0.3	0.3 ± 0.3	0.185
Hemoglobin (g/dl)	12.7 ± 3.8	12.2 ± 2.4	12 ± 2.5	12.5 ± 2.1	0.717
White blood cells ($10^3/mm^3$)	8 ± 7.4	7 ± 2.5	7.8 ± 3.4	9.1 ± 2.5	0.515
Platelets ($10^3/mm^3$)	239.3 ± 92.1	255.1 ± 104.4	241.1 ± 75.4	259.1 ± 110.1	0.777
Total Cholesterol (mg/dl)	183.2 ± 51.9	195.3 ± 48.7	208.3 ± 40.2	181.8 ± 27.8	0.056
LDL (mg/dl)	109.6 ± 43.2	120.1 ± 39	125.6 ± 28	106 ± 36.4	0.145
HDL (mg/dl)	48.3 ± 16.9	47.5 ± 12	47 ± 10	40.8 ± 13.9	0.287
Triglyceride (mg/dl)	127 ± 47.7	139.7 ± 55.4	146.4 ± 43.9	134.1 ± 47.8	0.355

HDL; High-density lipoprotein cholesterol, LDL; Low-density lipoprotein cholesterol

Conventional Echocardiography

Conventional two-dimensional echocardiographic parameters of the patients were shown in Table 3. Patients with severe aortic stenosis had more hypertrophied myocardial walls (ANOVA, p<0.001). The left atrial dimension was also higher for patients for severe aortic stenosis. No differences were observed among groups for aortic dimensions in both retrospective and prospective cohorts. Left ventricular ejection fraction showed no significant difference among groups (ANOVA, p = 0.105). 3D analysis of aortic dimensions was presented in Table 4. 3D echocardiography showed slightly higher aortic dimensions compared to 2D.

The multivariate logistic regression analysis revealed that the FRS, (p=0.006) was the strongest and only significant determinant of having aortic dilatation.

Table 3: Comparison of conventional echocardiographic parameters among groups

Parameters	Aortic sclerosis (n=50)	Aortic stenosis (n=98)			P
		Mild (n=36)	Moderate (n=42)	Severe (n=20)	
Aort sinotubular junction	30.1 ± 4.7	30.5 ± 4.3	31.6 ± 4.9	30.6 ± 4.3	0.469
Ascending Aorta	32.7 ± 6.4	32.2 ± 6.4	34.2 ± 7.1	36.6 ± 8.8	0.105
LV end-diastolic diameter (cm)	4.9 ± 0.4	4.8 ± 0.4	4.9 ± 0.4	5.0 ± 0.7	0.110
LV end-systolic diameter (cm)	3.4 ± 0.4	3.3 ± 0.5	3.2 ± 0.4	3.4 ± 0.6	0.383
Posterior wall thickness (mm)	1.1 ± 0.1*	1.2 ± 0.1*	1.2 ± 0.2*	1.4 ± 0.1	<0.001
Septal thickness (mm)	1.3 ± 0.2*	1.4 ± 0.2*	1.3 ± 0.2*	1.6 ± 0.3	<0.001
LV EF Simpson (%)	58.8 ± 4.5	58.3 ± 7.8	57.8 ± 7.6	54.6 ± 9.4	0.105
LA diameter (mm)	4.1 ± 0.5*	4.1 ± 0.4*	4.3 ± 0.5*	4.7 ± 0.6*	<<0.001
Peak transaortic gradient (mmHg)	19.1 ± 4.9*#π	31.1 ± 3.8*#	49.3 ± 9.2*	76.6 ± 8.6	<0.001
Mean transaortic gradient (mmHg)	9.7 ± 3.2*#π	19.7 ± 3.2*#	28.5 ± 6.3*	47.2 ± 5.8	<0.001
Transaortic flow velocity (m/s)	1.9 ± 0.4*#π	2.8 ± 0.2*#	3.4 ± 0.3*	4.4 ± 0.3	<0.001
Estimated sPAP (mmHg)	35.1 ± 11.8	35.5 ± 13.8	33.8 ± 13.9	29.7 ± 12.1	0.400

EF; ejection fraction, LA; left atrium, LV; left ventricle, sPAP; systolic pulmonary artery pressure.

Table 4. Comparison of 2D and 3D echocardiographic measurements of aortic root and aorta in prospective cohort.

	2D echocardiography				3D echocardiography			
	Mild (n=8)	Moderate (n=12)	Severe (n=10)	P	Mild (n=8)	Moderate (n=12)	Severe (n=10)	P
Aortic annulus (mm)	28.0 ± 2.4	29.0 ± 2.8	29.2 ± 1.8	0.457	28.1 ± 2.6	29.3 ± 2.9	29.5 ± 1.9	0.413
Aorta sinüs valsalva (mm)	29.3 ± 4.2	30.8 ± 5.2	30.4 ± 4.1	0.837	29.6 ± 4.4	31 ± 5.6	30.6 ± 4.4	0.801
Aort sinotubular junction (mm)	29.8 ± 4.0	31.4 ± 5.4	31.2 ± 4.3	0.779	30.1 ± 4.3	31.7 ± 5.7	31.6 ± 4.4	0.712
Ascending aorta (mm)	28.9 ± 4.1	31.9 ± 7.4	33.6 ± 8.8	0.318	29.6 ± 4.4	32.1 ± 7.6	33.6 ± 9	0.419
Aortic annulus index (mm/m^2)	15.9 ± 1.2	16.1 ± 1.7	15. ± 1.6	0.518	16 ± 1.8	16.3 ± 1.9	15.9 ± 1.7	0.410
Aorta sinüs valsalva index (mm/m^2)	16.5 ± 2.0	16.8 ± 2.0	16.7 ± 2.3	0.476	16.7 ± 2.2	17.1 ± 2.5	16.9 ± 2.6	0.313
Aort sinotubular junction index (mm/m^2)	16.1 ± 2.1	17.2 ± 2.4	16.4 ± 2.5	0.312	16.7 ± 2.2	17.3 ± 2.5	16.6 ± 2.6	0.298
Ascending aorta index (mm/m^2)	17.1 ± 3.0	18.5 ± 3.5	18.3 ± 4.4	0.570	17.6 ± 3.1	18.7 ± 3.6	18.5 ± 4.5	0.340

DISCUSSION

In this study, we investigated the impact of aortic stenosis severity on post-stenotic aortic dilatation. The main findings can be summarized as follows: (i) Severity of aortic stenosis has no relevant relationships with the occurrence of post-stenotic aortic dilatation (ii) Framingham risk score is the only significant determinant of aortic dilatation.

The pressure towards the aortic wall and the aortic pathology that would affect the organization of the aortic wall histology are the basic causes of dilatation. High blood pressure and any pathology of the aortic wall would be expected to contribute to the development of aortic dilatation. It may be mentioned that the velocity of the blood increases greatly at the valve level when the aortic valve has stenosis and produces high kinetic energy which may cause the ascending aorta to dilate (5,9,15,16). Nonetheless, this theoretical principle is not true for circulation mechanics, as circulation in an elastic system (arterial) is a pulsating flow with friction. The type of flow is considered as another contributing factor to aortic dilatation. Turbulent flow is characterized by the velocity fluctuations. In patients with aortic stenosis, the turbulent flow in the ascending aorta is created by the stenosis of the aortic valve by reaching its maximum level in the post-stenotic region. Cavitation has been identified as a factor that causes severe aortic wall injury which was claimed to result in aortic dilatation (4).

Patients with bicuspid aortic valve have been shown to have a larger aortic root and ascending aorta, regardless of transaortic gradient (5). In a previous study that aimed to determine the prevalence of a dilated aortic root in patients with aortic stenosis, 118 patients were studied and aortic root dilation was found to be common in aortic stenosis but is not related to the severity of stenosis. However, the mentioned study did not compare bicuspid aortic valves and tricuspid aortic valves (5). Morgan-Hughes et al. also concluded that patients with bicuspid aortic valves and pure severe aortic stenosis had moderately dilated thoracic aorta compared to patients with tricuspid aortic valves (17). In a retrospective study that compared the size of the aorta in patients with bicuspid aortic valves accompanied by corresponding valve lesions (stenosis, regurgitation, or mixed), it was found that the degree of dilation is not related with the severity of aortic stenosis (18). This indicates that post-stenotic aortic dilation is a result of bicuspid aortic valve stenosis but not normal- tricuspid aortic valves. The findings of the mentioned study intensify the hypothesis that aortic wall abnormalities are the main trigger for the development of aortic dilatation. Moreover, distorted valve geometry can cause dilatation of the aorta due to various hemodynamic changes of the turbulent flow. The left ventricular outflow tract, the aortic valve, ascending aorta, and the aortic arch are all embryologically linked, as they originate from the neural crest. Neural crest disorders could also result in arterial dissections which can be stated as a common finding in patients with bicuspid aortic valve (11).

Aortic stenosis is the most common valve heart disease and the third most common heart disease in developed countries (1,2). The prevalence of aortic stenosis in the elderly population is around 4%. Aortic sclerosis is common by being seen around 33% of patients over 65 years of age (1,2). Calcification of the aortic valve is the leading underlying cause of aortic stenosis in most cases. Calcified aortic stenosis is a dynamic chronic disease (19). It starts with thickening and calcification of the leaflets that do not affect hemodynamics and ends with a heavily calcified mass at the leaflet by time. Eventually, it affects leaflet motion and causes severe valve stenosis. Recently, some studies have demonstrated that aortic valve calcification is not only a degenerative process. It is also an active process consisting of inflammation and lipid deposition. In this context, atherosclerosis has similar pathophysiology. Concurrently, epidemiological studies have proved that aortic stenosis and atherosclerosis involve several common risk factors. Atherosclerosis is also considered to be one of the major factors in the development of aortic enlargement (1,2).

Aortic root dimensions have been measured by 2D echocardiography conventionally. However, the transection line of the ultrasound beam can cause misalignment and result in underestimated measurements. Recent developments in 3D echocardiography have allowed better spatial and temporal resolution. Technological advances have expanded the routine usage areas of 3D echocardiography. Multi-beat acquisition can provide more proper spatial and temporal resolution which would improve the functional analysis of the ventricles and valves. A full-volume scan area obtained by 3D echocardiography can demonstrate the aortic valve cusps and aorta better than conventional 2D echocardiography (20). In our study, the largest aortic root diameters measured with 3D echocardiography were significantly larger than 2D echocardiography. Also, it has been shown that 3D echocardiography has better inter/intra-observer variability. Three-dimensional echocardiography may be a useful method for periodic monitoring of patients with dilated aortic root.

CONCLUSION

Aortic dilatation in patients with aortic stenosis is associated with higher FRS and the degree of AS seems to be irrelevant with the prevalence of aortic dilatation. 3D echocardiography is useful for the evaluation of aortic root measurements and provides higher diameters than 2D echocardiography. Well designed- prospective trials with larger cohorts are needed to clarify the actual influence of aortic stenosis aortic dilatation.

Conflict of interest

No conflict of interest was declared by the authors.

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