

Left Ventricular Function Assessment by Strain Analysis in Patients with Severe Aortic Stenosis with Preserved Ejection Fraction Undergoing Aortic Valve Replacement – A Prospective Longitudinal Study at a Tertiary Health Care Centre of South India

Syed Waleem Pasha¹, Narasimha D. Pai², Padmanabha Kamath³, Ramanatha L. Kamath⁴, Francis N.P. Monteiro⁵

^{1,4} Department of Cardiology, Kasturba Medical College, (Affiliated to MAHE), Mangalore, Karnataka, India. ^{2,3} Department of Cardiology and Interventional Cardiology, Kasturba Medical College (Affiliated to MAHE), Mangalore, Karnataka, India. ⁵ Department of Forensic Medicine and Toxicology, A. J. Institute of Medical Sciences, (Affiliated to Rajiv Gandhi University of Health Sciences), Mangalore, Karnataka, India.

ABSTRACT

BACKGROUND

Aortic stenosis (AS) is the most common, single, native valvular heart disease in adult population. The purpose of this study was to detect abnormalities in global longitudinal strain (GLS) and strain rate using 2D - STI in patients with severe AS and preserved left ventricular ejection fraction (LVEF). The effect of aortic valve replacement (AVR) on changes in strain parameters 30 days after surgery was also analysed.

METHODS

A total number of 60 patients aged more than 18 years with aortic valve disease scheduled for surgical aortic valve replacement admitted in Department of Cardiology, KMC hospital Mangalore, were included over a period of 18 months from January 2017 to June 2018.

RESULTS

A total of 60 patients with severe AS, defined by an aortic valve area of $< 1 \text{ cm}^2$, mean transaortic pressure gradient (ΔP) of $> 40 \text{ mmHg}$ and maximum aortic velocity (V_{max}) of $> 4 \text{ m/sec}$ were studied. Mean age of the study population was 63.5 years. 60 % of the population were males and 40 % being females. Most common risk factor present in the study population was diabetes mellitus (DM). 83% of the patients in the study population had at least one symptom. Most common symptom with which the patients presented was exertional dyspnoea. All patients had normal left ventricle (LV) cavity dimensions and LVEF prior to surgery with diastolic dysfunction being present in all patients. The LV ejection fraction is not significantly altered. The aortic valve area calculated by continuity equation has significantly increased post AVR with a significant reduction in transaortic peak and means pressure gradients. Mean global longitudinal strain (GLS) improved from -15.1% to -16.9% ($P < 0.001$) and longitudinal strain rate improved from -0.8 to $-0.9/s$ ($P < 0.001$).

CONCLUSIONS

Global longitudinal strain and strain rate can be adequately measured by 2D speckle-tracking imaging and can be used to detect subtle changes of myocardial function in patients with severe AS with preserved LVEF.

KEYWORDS

Aortic Stenosis, Exertional Dyspnoea, Global Longitudinal Strain, Transaortic Pressure Gradient, Ventricular Hypertrophy

Corresponding Author:

*Dr. Narasimha D. Pai,
Associate Professor,
Department of Cardiology and
Interventional Cardiologist
Kasturba Medical College,
(Affiliated to MAHE), Mangalore,
Karnataka, India.
E-mail: narasimhapai@yahoo.com*

DOI: 10.18410/jebmh/2021/446

How to Cite This Article:

Pasha SW, Pai ND, Kamath P, et al. Left ventricular function assessment by strain analysis in patients with severe aortic stenosis with preserved ejection fraction undergoing aortic valve replacement – a prospective longitudinal study at tertiary health care centre of South India. J Evid Based Med Healthc 2021;8(27):2405-2411. DOI: 10.18410/jebmh/2021/446

Submission 05-02-2021,

Peer Review 14-02-2021,

Acceptance 19-05-2021,

Published 05-07-2021.

Copyright © 2021 Syed Waleem Pasha et al. This is an open access article distributed under Creative Commons Attribution License [Attribution 4.0 International (CC BY 4.0)]

BACKGROUND

Aortic stenosis is the most common, single, native valvular heart disease in adult population.¹ By narrowing the left ventricular outflow orifice, AS causes reduction in the effective orifice area for LV ejection and thereby increase ventricular afterload.² The systolic afterload on the LV evokes certain compensatory responses to permit normal LV emptying and maintain cardiac output. The physiologic compensations increase the pressure generated by the LV resulting in increase in gradient between the LV and aorta.^{2,3,4} No measurable gradient develops until the valve area is reduced by 50%.³ The anatomic compensation is an increase in myocardial mass i.e. left ventricular hypertrophy (LVH). These compensations maintain normal LV emptying and cardiac output. However, the compensatory mechanisms have few consequences including LV diastolic dysfunction.^{2,3,4} In the compensated phase, patients may be entirely asymptomatic or may suffer from exertional symptoms.^{4,5} Progressive increase in AS or impairment in LV contractility causes dilation of LV and later pump failure.^{5,6,7,8,9} Patients with AS present with angina, syncope or dyspnoea on exertion.^{4,5,6,7,8,9} They may also present due to infective endocarditis or embolic phenomenon.⁴ Symptomatic patients with AS require surgical treatment.^{4, 8} Without aortic valve replacement (AVR), patients of AS with LV failure have mean life expectancy of less than two years.^{3,4,5,8} For patients who present with syncope or angina, mean life expectancies are three and 5 years respectively.^{3,4,8,9} Also, the risk of sudden death increases to 15 - 20 % in symptomatic patients from 3 - 5 % in asymptomatic patients.⁴ Aortic valve replacement (AVR) is the only treatment option that can interrupt the natural course of the disease.^{4,10,11,12} AVR is recommended when AS patients develop symptoms or myocardial impairment.¹⁰ However, patients can adopt a sedentary lifestyle, thus avoiding symptoms and complicating clinical assessment.¹¹ A symptomatic state reflects failure of myocardial compensatory mechanisms, emphasizing that evaluation of the myocardial condition is of prime importance in decision making regarding AVR.^{11,12,13,14,15} The chronic LV pressure overload results in changes in left ventricular geometry and performance. To compensate for the elevated mid-wall stress, the LV wall thickness increases thereby maintaining LV function. However, when LV pressure exceeds the LV hypertrophy, the increased mid-wall stress results in impairment of LV performance and LV function. Detection of subtle changes in LV systolic function may help in earlier referral for AVR.^{13,14,15,16}

Strain imaging has demonstrated to be the most appropriate method to evaluate LV myocardial contractility and accordingly may enable better characterization of subtle changes in LV performance in AS patients.^{17,18} Two dimensional speckle tracking strain imaging (2D - STI) allows evaluation of myocardial strain and strain rate providing comprehensive information of LV contractility.^{19,20,21} Global longitudinal strain (GLS) has been shown to predict outcomes in patients with AS. Mechanical dispersion by strain echocardiography is independent of LVEF.^{21,22} Two-dimensional speckle-tracking strain imaging

enables angle-independent myocardial deformation analysis by tracking frame-to-frame natural acoustic markers, or speckles, that appear equally distributed within the myocardial wall.^{19,20,21} This novel imaging method provides reliable and accurate information on myocardial strain in the three spatial directions: radial, circumferential, and longitudinal. Applying the strain Lagrangian formula ($(L - L_0) / L_0$), the percentage change in myocardial length (L) relative to the initial length (L_0) derives myocardial strain (expressed in percentage).^{18,19,21,22} The temporal derivation of myocardial strain results in strain rate and is a measure of the rate of deformation (expressed in s^{-1}).²² The thickening (positive strain) and thinning (negative strain) of the myocardial wall indicates the radial deformation. The shortening (negative strain) and lengthening (positive strain) of the myocardial wall along the curvature of the LV in the short-axis view indicates circumferential deformation. Finally, the longitudinal deformation relates to motion from mitral annulus to the LV apex in the apical views and results in shortening (negative strain) and lengthening (positive strain).^{18,21,22}

Objectives

1. To record global longitudinal strain (GLS) of left ventricle using 2D speckle tracking echocardiography to assess left ventricular function in patients with severe aortic stenosis undergoing aortic valve replacement (AVR) pre-operatively and 1 month after the surgery.
2. To evaluate the value of strain imaging in assessment of left ventricular function in aortic stenosis patients and post valve replacement.
3. To assess the role of strain imaging to follow up patients after aortic valve replacement.

METHODS

This is a prospective, longitudinal study that was carried out in the Department of Cardiology, KMC Hospital, Mangalore, India for a period of 18 months from January 2017 to June 2018. A total number of 60 patients aged more than 18 years with aortic valve disease scheduled for surgical aortic valve replacement admitted in Department of cardiology, KMC hospital Mangalore, were included by convenient sampling method.

Ethics Approval and Consent to Participate

Ethical clearance for this study was obtained from the institutional ethics committee of Kasturba Medical College, Mangalore. Informed consent from all participating in this study were sought and obtained after explaining the all the information regarding the study.

Sample Size

With 95 % confidence level and 95% power with respect to Delgado et al study, the sample size calculated is 60.¹⁶

$$n = \frac{Z^2_{\alpha} \sigma^2}{d^2}$$

Z_α = 1.96 at 95% confidence level

σ = combined standard deviation

d = mean difference (1.8) ¹⁶

Selection of Subjects

All the patients of aged more than 18 years, diagnosed to have severe aortic stenosis and scheduled for aortic valve replacement as per American College of Cardiology (ACC)/American Heart Association (AHA) guidelines and preserved left ventricular ejection function i.e., LVEF > 50 % were included in the study.

- Aged above 18 years.

Study Tools

Data was recorded on a proforma.

Study Protocol

Demographic data and cardiovascular risk factors were recorded. Clinical evaluation prior to AVR including assessment of symptoms (angina, syncope, and dyspnoea) and physical examination was done and recorded. Electrocardiogram (ECG), blood counts, blood chemistry including renal and thyroid functions were done in the participant population. Coronary angiogram was done in all participant population prior to aortic valve replacement. Echocardiographic data was acquired with a vivid 9E ultrasound scanner (GE Medical Systems) and a 1.5 - 4.0 MHz phased array transducer (M5S). The included patients were assessed with conventional echocardiography, 2D echocardiography before the surgical aortic valve replacement, as well as at 1 week and 1 month after the surgery.

Imaging of the patient was done in the left lateral decubitus position and information was acquired with 3.5 MHz transducer at a depth of 16 cm in the parasternal (long and short axis views) and apical views (two chamber, four chamber and apical long axis views). LV dimensions were acquired from the standard M-mode images at the parasternal long-axis views and included LV diameters and end-diastolic thickness of the interventricular septum and posterior wall. LV mass was inferred using the formula proposed by Devereux et al. and corrected by the body surface area to derive LV mass index. When LV mass index is > 110 g/m² for women and > 134 g/m² in men defines LV hypertrophy. LV end-diastolic and end-systolic volumes were calculated from apical two and four chamber views and Simpson's rule were used to calculate LVEF.

Left ventricular diastolic function was analysed using early (E - wave) and late (A - wave) transmitral velocities. The E/A ratio, and the E-deceleration time will be calculated from the spectral pulsed-wave doppler recordings. Tissue doppler echocardiography was performed, adjusting gain

and frame rate to obtain a suitable tissue characterization. The peak early diastolic velocity (E') was measured at the basal myocardial segments on the apical four-chamber view and E/E' ratio was calculated.

In patients with aortic stenosis (AS), valve anatomy (bicuspid or tricuspid, calcification, and leaflet motion), valve haemodynamics (transaortic peak velocity; V_{max} and transaortic pressure gradient; ΔP), Aortic valve area (AVA) and Aortic valve area index; AVAI) were analysed and recorded.

The aortic valve area was inferred by the continuity equation and the maximum pressure gradient across the valve will be determined by the modified Bernoulli equation. LV outflow tract (LVOT) diameter was calculated on parasternal long axis views. Pulsed-wave and continuous-wave doppler was utilized to record velocities across LVOT and aortic valve respectively. LV stroke volume index (LVSVI) was calculated as LVOT_{VTI} X LVOT_{area}/body surface area. A cut-off of more than 35 ml/m² was the indication of preserved LVSVI. Severe AS was defined as AVA of less than 1 cm².

Mean pressure gradient was calculated averaging the instantaneous gradients over the ejection period on the continuous-wave doppler recordings. Mean pressure gradient of more than or equal to 40 mmHg was considered as severe AS. Colour doppler echocardiography was performed after optimizing gain and Nyquist limit in order to detect regurgitant valve disease. If present, the severity of valvular regurgitation was determined on a qualitative scale (mild, moderate, and severe) according to the American Society of Echocardiography guidelines. Patients with moderate to severe mitral or aortic regurgitation were excluded from the study.

Right ventricular systolic pressure was determined from the maximal continuous-wave doppler velocity of the tricuspid regurgitant jet using systolic trans tricuspid pressure gradient obtained by modified Bernoulli equation and right atrial pressure.

Comprehensive assessment of LV myocardial S-and-SR was undertaken using 2D - STI. For this purpose, standard 2D grey-scale images of the LV were obtained at parasternal mid-ventricular short-axis view and at conventional apical two- and four-chamber and apical long-axis views, with a mean frame rate of 71 ± 7 frames/s.

Strain quantification was performed by using commercially available software (EchoPAC version 7.0.0, General Electric-Vingmed). The endocardial contour was manually traced at an end-systolic frame. The software then mechanically traced a concentric region of concern including the entire myocardial wall. The myocardial tracking was verified, and the region-of-interest width was adjusted to optimize the tracking, if required. Later, segmental strain evaluation was performed by dividing each LV image into six segments. Peak systolic longitudinal strain was calculated averaging the peak systolic values of the 18 segments, derived from the 6 segments of the 3 apical views (two- and four-chamber and apical long-axis views). All measures were averaged over 3 cardiac cycles. Normal GLS is -16 % to - 20 %.

Statistical Analysis

All data was analysed with statistical package for social sciences (SPSS) software version 17. Continuous data was presented as mean and categorical data was presented as frequencies and percentages. Paired t test was used to find out the differences between the groups.

RESULTS

A total of 60 patients with severe AS, defined by an aortic valve area of < 1 cm², mean transaortic pressure gradient (ΔP) of > 40 mmHg and maximum aortic velocity (Vmax) of > 4 m/sec were studied. Mean age of the study population was 63.5 years. 60 % of the population were males and 40 % being females. Table 1 shows gender distribution of the GLS and strain rate and are comparable between the two groups with no significant difference.

Sl. No.	Gender	No. (%)	GLS (Mean)		Strain Rate (Mean)	
			Baseline	Follow Up	Baseline	Follow up
1	Female	24 (40)	- 15.1	- 16.9	- 0.8	- 0.9
2	Male	36 (60)	- 15.11	- 16.97	- 0.81	- 0.9

Table 1. Comparing GLS and Longitudinal Strain Rate

The presence of cardiovascular risk factors was noted and tabulated in table 2. Study demonstrates that most common risk factor present in the study population was diabetes mellitus. 33.3 % of the study group were smokers and one third of the study population suffered from dyslipidaemia. Hypertension was noted in nineteen out of sixty patients.

	Presence of Cardiovascular Risk Factors				
	HTN N (%)	DM N (%)	Dyslipidaemia N (%)	Smoking N (%)	Family History N (%)
Present	19 (31.6)	24 (40)	20 (33.3)	20 (33.3)	28 (46.7)
Absent	41 (68.4)	36 (60)	40 (66.7)	40 (66.7)	32 (53.3)

Table 2. Risk Factors among the Study Population

83 % of the patients in the study population had at least one symptom. Most common symptom with which the patients presented was exertional dyspnoea. Approximately 16 % of the participants were asymptomatic. Table 3 demonstrates the symptoms of the participant population.

	Symptoms			
	Asymptomatic	Angina	Syncope	Dyspnoea
N	10	28	10	50
%	16.6	46.6	16.6	83.3

Table 3. Symptoms of the Patients

The echocardiographic data of the study population is summarized in tables 4, 5 and 6. All patients had normal LV cavity dimensions and LVEF prior to surgery with diastolic dysfunction being present in all patients. Most had grade 1 diastolic dysfunction which persisted post AVR. Post AVR, the LV dimensions were not significantly altered. The interventricular septum (IVS) thickness (P < 0.001) and the posterior wall (PW) thickness (P < 0.001) with LV mass index were significantly reduced post AVR. The LV mass index which represents an index of LV hypertrophy reduced significantly from 165 g/m² to 125 g/m² (P < 0.001).

	Echo - Dimensions (Mean)				
	LVEDD (mm)	LVESD (mm)	IVS (mm)	PW (mm)	LVMI (g/m ²)
Baseline	50	31	15	13	160
Follow up	49	30	12	12	125
P value	0.3	0.8	< 0.001	< 0.001	< 0.001

Table 4. Linear Dimensions on M-Mode Echocardiography

Table 5 demonstrates the variation of LV volumes and LV ejection fraction as measured by the Simpson's method. There is a significant reduction in end-diastolic volume of the LV. However, the LV ejection fraction is not significantly altered.

	LVEDV (ml)	LVESV (ml)	LVEF (%)
Baseline	108	44	61
Follow up	99	41	60
P value	0.007	0.2	0.7

Table 5. Demonstrates the Variation of LV Volumes and LVEF

	Aortic Valve		
	Peak Pressure Gradient (mmHg)	Mean Pressure Gradient (mmHg)	AVA (cm ²)
Baseline	71	45	0.8
Follow up	23	12	1.6
P value	< 0.001	< 0.001	< 0.001

Table 6. Change in AVA and Transaortic Pressure Gradients Post AVR

Table 6 shows the change in pressure gradients and aortic valve area 30 days after AVR. The aortic valve area calculated by continuity equation has significantly increased post AVR with a significant reduction in transaortic peak and means pressure gradients. This point to a successful surgical outcome.

Mean global longitudinal strain (GLS) improved from - 15.1 % to -16.9 % (P < 0.001) and longitudinal strain rate improved from - 0.8 to - 0.9/s (P < 0.001). Table 7 shows the change in GLS and strain rate. Figure 6 demonstrates significant change in GLS and Figure 7, the change in strain rate post AVR. Also, the mean GLS is lower in patients with severe aortic stenosis despite normal LVEF. The GLS is denoted by a negative sign as the longitudinal deformation results in shortening of the distance between mitral annulus to LV apex. Lengthening was denoted by positive sign.

	GLS	GLSR
Baseline	- 15.1	Baseline - 0.8
Follow up	-16.9	Follow up -0.9
P value	< 0.001	P value < 0.001

Table 7. GLS and Strain Rate Post AVR

DISCUSSION

The study undertaken confirmed that the patients with severe AS and preserved LVEF already demonstrated lower LV strain and strain rate parameters. This demonstrates subclinical LV dysfunction despite a normal LVEF. Also, at follow up after AVR, a considerable enhancement in these parameters was observed when compared to LVEF which remained unaffected. These conclusions highlight the practical application of strain imaging to identify subtle changes of LV systolic dysfunction early in patient with severe AS. The improvement in strain parameters noted after AVR regardless of unchanged LVEF further underscores

the value of strain imaging in demonstrating the mitigation in myocardial function post AVR.

Aortic stenosis results in chronic LV pressure overload leading to LV wall thickness in an attempt to maintain adequate wall stress.¹³ Progressive AS leads to increasing severity of LVH to maintain wall stress and maintain systolic LV function. However, at a certain point during the natural history, LVH cannot compensate the increased LV pressure resulting in afterload mismatch.¹² This afterload mismatch denotes the initial step in LV dysfunction in severe AS. Ultimately LV volumes will increase and LVEF will decrease. Once LVEF is reduced, outcome of AVR is not good. Hence it is favoured to sense subclinical LV dysfunction prior to LVEF decreases. Several studies have reported reduced values of myocardial strain parameters in severe AS patients. 2D - STI allows myocardial strain imaging and measurements and overcomes the limitation of the angle insonation dependency of the tissue Doppler imaging.

Becker et al. evaluated LV myocardial strain in 22 symptomatic severe AS patients with preserved LVEF by 2D - STI.²³ They found a decrease in GLS and radial strain in these patients. The outcome of the present study extends these findings and notes a decrease in global longitudinal strain and strain rate in patients with severe AS and preserved LVEF. This finding also agrees with the findings of Miyazaki et al. who demonstrated that GLS decreased as AS severity increases.

When the effective orifice area is less than or equal to 1 cm², the coronary flow reserve decreases leading to repetitive subendocardial ischemic injury leading to fibrotic areas. Schwarzkopff et al. described a higher amount of peri myocyte fibrosis in patients with severe AS.²⁴ This may have a negative impact on LV strain and strain rate. Becker et al. also demonstrated a significant increase in radial and circumferential strain and strain rate after six months of AVR.

Kleboe et al. included 162 patients with severe aortic stenosis and preserved LVEF (60 + / - 11 %) and followed them up with GLS measurement over 37 months.¹¹ They found that the nonsurvivors had worse GLS compared to survivors. This proves that lower GLS may be a risk marker providing novel prognostic information in patients with aortic stenosis. Decreased GLS equated to increased mechanical dispersion which reflects inhomogeneous myocardial contractions and has been linked to myocardial fibrosis secondary to subendocardial ischemia seen in severe AS patients. At present the best forecaster of sudden cardiac mortality in AS patients is the progression of symptoms. Symptoms may be disregarded in the initial phase due to gradual change in lifestyle adapting to the severity of disease. Early detection of myocardial fibrosis can perhaps lead to early identification of patients at risk for arrhythmic death. It is shown that mechanical dispersion and GLS assessed by strain echocardiography could possibly detect subtle LV abnormalities indicating poor outcomes. Patients with asymptomatic AS with preserved LVEF and marked mechanical abnormalities as detected by GLS might be taken for early AVR.

The findings of the present study also are in agreement with study conducted by Delgado et al. who evaluated

myocardial multidirectional strain and strain rate in severe AS patients with preserved LVEF using 2D - STI.¹⁶ The study compared LV strain and strain rate between patients with AS and normal people and found that severe AS patients had significantly decreased values of strain and strain rate when compared to normal controls. Changes in LV radial, circumferential and longitudinal strain and strain rate were evaluated in 73 severe AS patients with preserved LVEF before and 17 months after AVR. At 17 months after AVR, LV strain and strain rate were significantly improved in all three directions, whereas LVEF remained unchanged. The findings of the present study are similar with reduced GLS and strain rate in severe AS patients with preserved LVEF and significant improvement in strain parameters even 30 days after AVR.

Left ventricular loading situation alter acutely after AVR with a significant decrease in LV pressure overload. The LV adapts to this new condition with regression of LV hypertrophy and a resulting improvement in LV performance. The change in LV geometry and function after AVR may be the reason for improvement in LV function. Delgado et al. also demonstrated a significant decrease in LVMI which is a marker of LVH.¹⁶ The present study also shows similar results with a decrease in LVMI and attendant improvement in LV strain and strain rate.

Dahl et al. evaluated the effect of pre-operative reduced GLS in severe AS on long term outcome after AVR. A total of 125 patients with severe AS and LVEF > 40 % scheduled for AVR were evaluated pre-operatively.²⁵ The patients were divided into four groups as per GLS values. The patients were followed up for 4 years after AVR. They found that GLS was significantly reduced in severe AS patients and showed a liner relation with the outcome of patients [major adverse cardiac events (MACE) and mortality] with surgical AVR beyond standard risk factors. The present study confirms similar finding of decreased GLS in severe AS patients. Hence the present study agrees with Dahl et al. study's conclusion that GLS may provide important prognostic information beyond standard risk factors. Postoperative reverse remodelling is a slow and not fully reversible process, hence, pre-operative structural changes in the myocardium may be of greater importance as they may affect postoperative prognosis. These subtle changes in the myocardium affecting the LV function may be detected earlier by strain analysis than the conventional markers of LV function i.e. LVEF.

Kusunose et al. assessed the utility of LV GLS in predicting mortality in moderate to severe AS patients with preserved LVEF.²⁶ The study included 395 patients with AS patients. It was found that GLS provides incremental prognostic value in addition to standard clinical and echocardiographic parameters. The present study compares similar parameters. LV GLS is reduced in severe AS patients despite preserved LVEF and may represent subtle LV dysfunction. Assessment of LV-GLS offers a non-invasive tool that can detect subtle changes in LV function when other standard measures are still normal. It also improves the risk stratification of AS patients above the standard clinical parameters. Offering AVR to these patients may significantly improve survival compared to medical therapy.

Kafa et al. recorded LV GLS in 208 patients with severe aortic stenosis with preserved EF who underwent AVR and found abnormal GLS despite normal EF in 20 % patients at the end of one year of surgery which contributed to adverse outcomes.²⁷ The present study also demonstrates improvement in GLS even 30 days after AVR.

Poulsen et al. evaluated the changes in doppler derived longitudinal strain after AVR in 40 severe AS patients and preserved LVEF. The authors observed a sustained improvement of longitudinal strain over one-year post AVR without significant changes in LVEF.²⁸ In addition, LVMI decreased significantly from 184 g/m² to 127 g/m². The present study evaluates longitudinal strain and strain rate by speckle tracking imaging which is angle independent. The findings are similar in terms of improvement in GLS and LVMI after AVR.

CONCLUSIONS

Global longitudinal strain and strain rates

- can be adequately measured by 2D speckle-tracking imaging.
- are significantly impaired in severe AS patients.
- can be used to detect subtle changes of myocardial function in patients with severe AS with preserved LVEF.
- at 30 days after AVR, both improved significantly.

Data sharing statement provided by the authors is available with the full text of this article at jebmh.com.

Financial or other competing interests: None.

Disclosure forms provided by the authors are available with the full text of this article at jebmh.com.

REFERENCES

- [1] Lung B, Baron G, Butchart EG, et al. A prospective survey of patients with valvular heart disease in Europe: The Euro heart survey on valvular heart disease. *Eur Heart J* 2003;24(13):1231-1243.
- [2] Levinson GE. Valvular heart disease. In: Gordon BL, edr. *Clinical cardiopulmonary Physiology*. 3rd edn. New York: Grune and Stratton 1969: p. 245-248.
- [3] Ross J Jr, Braunwald E. Aortic Stenosis. *Circulation* 1968;(Suppl 38):61-67.
- [4] Levinson GE, Alpert JS. Aortic Stenosis. In: Alpert JS, Dalen JE, Rahimtoola SH, eds. *Valvular heart disease*. 3rd edn. Philadelphia: Lippincott Williams and Wilkins 2000: p. 183-245.
- [5] Genereux P, Stone GW, O’Gara PT, et al. Natural history, diagnostic approaches and therapeutic strategies for patients with asymptomatic severe aortic stenosis. *J Am Coll Cardiol* 2016;67(19):2263-2288.
- [6] Izumo M, Takeuchi M, Seo Y, et al. Prognostic implications in patients with symptomatic aortic stenosis and preserved ejection fraction: Japanese Multicenter Aortic Stenosis, Retrospective (JUST-R) Registry. *J Cardiol* 2017;69(1):110-118.
- [7] Pellikka PA, Sarano ME, Nishimura RA, et al. Outcome of 622 adults with asymptomatic, hemodynamically significant aortic stenosis during prolonged follow-up. *Circulation* 2005;111(24):3290-3295.
- [8] Baumgartner H. Aortic stenosis: medical and surgical management. *Heart* 2005;91(11):1483-1488.
- [9] Lester SJ, Heilbron B, Gin K, et al. The natural history and rate of progression of aortic stenosis. *Chest* 1998;113(4):1109-1114.
- [10] Nishimura RA, Otto CM, Bonow RO, et al. 2014 AHA/ACC Guideline for the management of patients with Valvular Heart Disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation* 2014;129:e521-e643.
- [11] Klaeboe LG, Haland TF, Leren IS, et al. Prognostic value of left ventricular deformation parameters in patients with severe aortic stenosis: a pilot study of the usefulness of strain echocardiography. *J Am Soc Echocardiogr* 2017;30(8):727-738.
- [12] Ross J Jr. Afterload mismatch and preload reserve: a conceptual framework for the analysis of ventricular function. *Prog Cardiovasc Dis* 1976;18(4):255-264.
- [13] Elmariah S. Patterns of left ventricular remodeling in aortic stenosis: therapeutic implications. *Curr Treat Options Cardiovasc Med* 2015;17(7):391.
- [14] Mihaljevic T, Nowicki ER, Rajeswaran J, et al. Survival after valve replacement for aortic stenosis: implications for decision making. *J Thorac Cardiovasc Surg* 2008;135(6):1270-1278.
- [15] Pai RG, Varadarajan P, Razzouk A. Survival benefit of aortic valve replacement in patients with severe aortic stenosis with low ejection fraction and low gradient with normal ejection fraction. *Ann Thorac Surg* 2008;86(6):1781-1789.
- [16] Delgado V, Tops LF, van Bommel RJ, et al. Strain analysis in patients with severe aortic stenosis and preserved left ventricular ejection fraction undergoing surgical valve replacement. *Eur Heart J* 2009;30(24):3037-3047.
- [17] Heimdal A, Stoylen A, Torp H, et al. Real-time strain rate imaging of the left ventricle by ultrasound. *J Am Soc Echocardiogr* 1998;11(11):1013-1019.
- [18] D’Hooge J, Heimdal A, Jamal F, et al. Regional strain and strain rate measurements by cardiac ultrasound: principles, implementation and limitations. *Eur J Echocardiogr* 2000;1(3):154-170.
- [19] Kaluzynski K, Chen X, Emelianov SY, et al. Strain rate imaging using two-dimensional speckle tracking. *IEEE Trans Ultrason Ferroelectr Freq Control* 2001;48(4):1111-1123.
- [20] Notomi Y, Lysyansky P, Setser RM, et al. Measurement of ventricular torsion by two-dimensional ultrasound speckle tracking imaging. *J Am Coll Cardiol* 2005;45(12):2034-2041.
- [21] Dandel M, Hetzer R. Echocardiographic strain and strain rate imaging—clinical applications. *Int J Cardiol* 2009;132(1):11-24.

- [22] Gorcsan J 3rd, Tanaka H. Echocardiographic assessment of myocardial strain. *J Am Coll Cardiol* 2011;58(14):1401-1413.
- [23] Becker M, Kramann R, Dohmen G, et al. Impact of left ventricular loading conditions on myocardial deformation parameters: analysis of early and late changes of myocardial deformation parameters after aortic valve replacement. *J Am Soc Echocardiogr* 2007;20(6):681-689.
- [24] Schwartzkopff B, Frenzel H, Dieckerhoff J, et al. Morphometric investigation of human myocardium in arterial hypertension and valvular aortic stenosis. *Eur Heart J* 1992;(13 Suppl D):17-23.
- [25] Dahl JS, Videbaek L, Poulsen MK, et al. Global strain in severe aortic valve stenosis: relation to clinical outcome after aortic valve replacement. *Circ Cardiovasc Imaging* 2012;5(5):613-620.
- [26] Kusunose K, Goodman A, Parikh R, et al. Incremental prognostic value of left ventricular global longitudinal strain in patients with aortic stenosis and preserved ejection fraction. *Circ Cardiovasc Imaging* 2014;7(6):938-945.
- [27] Kafa R, Kusunose K, Goodman AL, et al. Association of abnormal postoperative left ventricular global longitudinal strain with outcomes in severe aortic stenosis following aortic valve replacement. *JAMA Cardiol* 2016;1(4):494-496.
- [28] Poulsen SH, Sogaard P, Nielsen-Kudsk JE, et al. Recovery of left ventricular systolic longitudinal strain after valve replacement in aortic stenosis and relation to natriuretic peptides. *J Am Soc Echocardiogr* 2007;20(7):877-884.