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ROLE OF GLOBAL LONGITUDINAL STRAIN IN AN OCTOGENARIAN WITH ASYMPTOMATIC SEVERE CALCIFIC AORTIC STENOSIS: CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Owing to population aging, the prevalence of aortic stenosis (AS) continues to increase. Despite significant advances in noninvasive imaging techniques and treatment options over the last 2 decades, the diagnostic echocardiographic hemodynamic criteria have remained largely unchanged. Complex decisions about treatment timing are presently informed by the calculated aortic valve area (AVA), transvalvular pressure gradients, valve morphology, and symptoms. However, there are several pitfalls of the hemodynamic severity grading of AS. Global longitudinal strain (GLS) is a sensitive marker of subtle hypertrophy-related impairment in left ventricular function and has shown promise as a relatively strong prognostic marker, when added to severity classification systems. The utility of GLS depends on its ability to predict clinically meaningful events, such as death or heart failure hospitalization, in patients with severe AS. In this manner it might function as a viable discriminator for appropriate eligibility for intervention. Published literature support the prognostic superiority of GLS over LVEF. The presence of impaired GLS may be a valuable bio-marker in the early diagnosis for left ventricle (LV) impairment, which would help scrutinize asymptomatic AS patients with high risk of adverse outcomes, such as major adverse cardiovascular events (MACE). Here, we are presenting an octogenarian lady afflicted with asymptomatic severe calcific aortic valve stenosis albeit with normal global strain on speckle tracking echocardiography. Whether to proceed for transcatheter aortic valve replacement (TAVR) or surgical aortic valve replacement (SAVR) or to continue with optimal medical therapy is a big dilemma in this elderly woman.

KEYWORDS: Aortic stenosis, severe asymptomatic aortic stenosis, TAVR, speckle tracking echocardiography, global longitudinal strain.

INTRODUCTION

With increased life expectation and lifespan of the population, AS has become one of the most common valvular heart diseases.[1] Of note, ~40-50% of severe patients with AS are asymptomatic.[2] Although patients with AS can be asymptomatic due to the provisionally sufficient LV function, the myocardial fibrosis resulting from the rising hemodynamic burden could lead to ventricular remodeling and enlargement, which further predisposes patients to sudden cardiac death.[3] The progressive exacerbation of LV dysfunction remains to be a matter of concern in asymptomatic patients. Currently, LVEF is used as the main criterion to select asymptomatic patients for aortic valve replacement (AVR).[4] However, there has been growing awareness that LVEF-based hierarchies may have significant deficiencies for early identification of asymptomatic patients with AS who required interventions since the LVEF assessment is highly affected by hemodynamic load. Thus, the LV deterioration might be cloaked in the setting of reduced after-load, where a more sensitive biomarker may facilitate the recognition of LV systolic impairment (Figure 1).

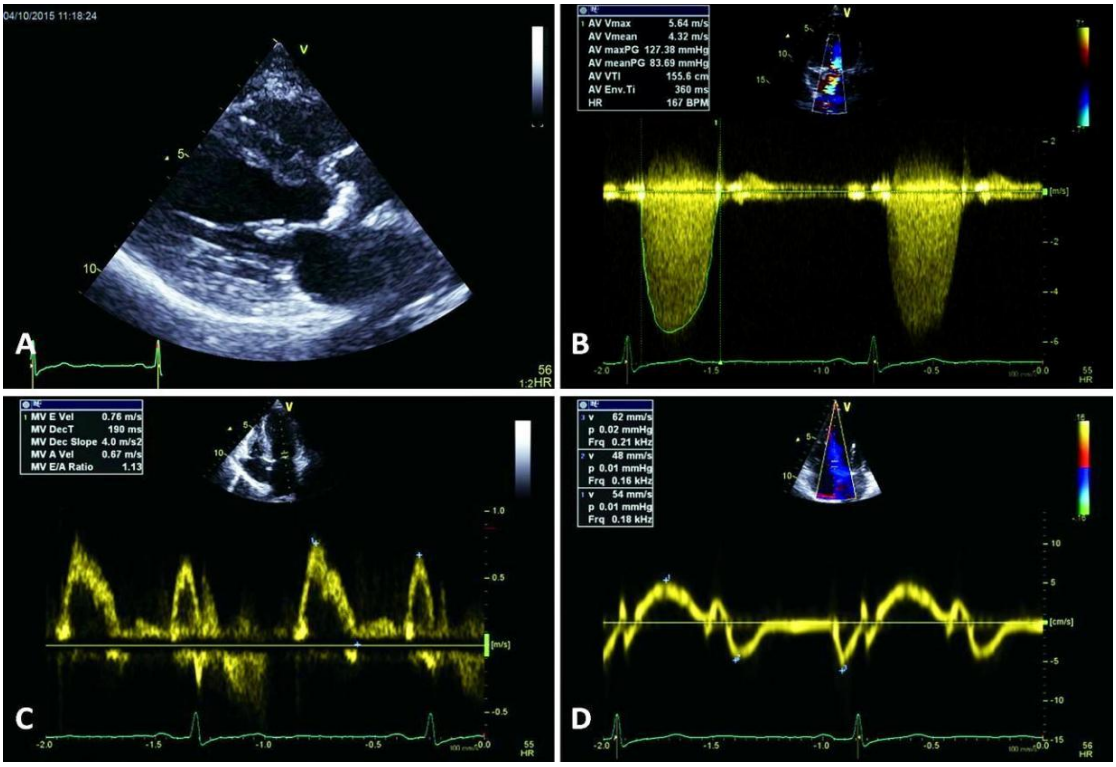


Figure 1: Consequences of very severe calcific aortic stenosis (A) in a patient in their 60s without a history of systemic hypertension. Peak transvalvular gradient was 127 mm Hg (mean gradient of 84 mm Hg) (B) at a blood pressure of 120/70 mm Hg, with an estimated LV systolic pressure of 247 mm Hg and a calculated aortic valve area of 0.6 cm². As a result of severe pressure overload, there was significant concentric LV hypertrophy (A) with an indexed LV mass of 130 g/m² and a relative wall thickness of 0.55. Global systolic LV function was preserved (ejection fraction of 65%) while systolic myocardial velocities measured by tissue Doppler imaging were significantly reduced (septal s' 5.2 cm/s)(D), indicating LV longitudinal dysfunction. There was significant LV diastolic dysfunction: impaired relaxation with a septal e' of 4.8 cm/s (D) and increased filling pressure-pseudonormal mitral inflow (C) with elevated E/e' ratio of 16 and a moderately dilated left atrium. LV, left ventricular.

Left ventricular global longitudinal strain (LVGLS), evaluating the contractile function of the ventricular muscle fiber, appears to be a robust parameter for detecting early LV dysfunction, even before the evident deterioration of LVEF (Figure 2).[5] Simultaneously, an increasing number of literature published with regard to LVGLS in asymptomatic patients with AS has demonstrated its association with adverse outcomes and moreover, impaired GLS has a strong prognostic utility in asymptomatic population.[6] In the current report, we have evaluated GLS by the innovative 4DXStrain technique.

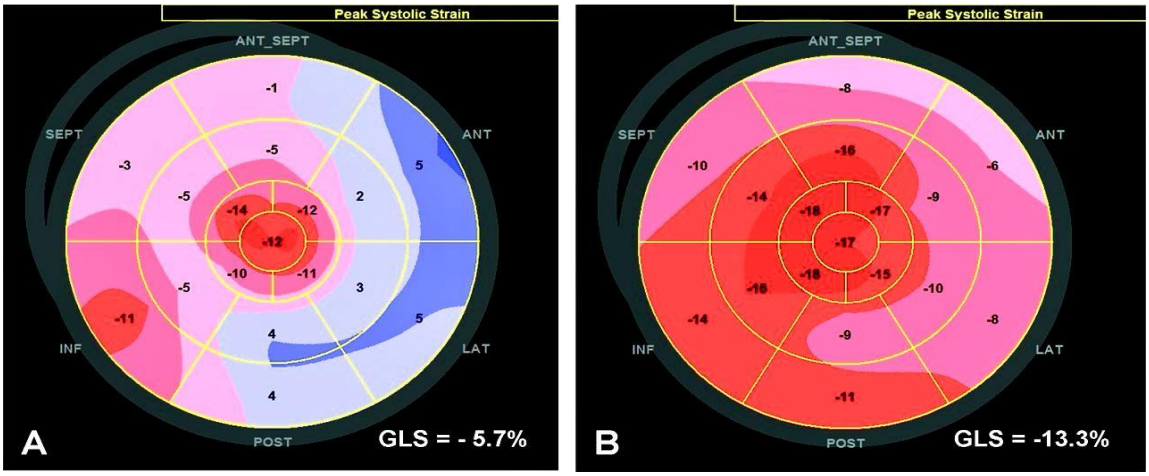


Figure 2: LV global longitudinal strain measured by speckle tracking echocardiography in a patient with severe aortic stenosis before (A) and 1 month after TAVI (B). Before TAVI, the LVEF was 30% and the mean

transvalvular gradient was 70 mm Hg. The GLS was severely reduced: -5.7%. Systolic lengthening is present in the basal and mid-segments of the lateral wall. Angiography revealed no significant coronary artery disease. One month after TAVI, LVEF increased to 55% while GLS significantly improved: -13.3%. GLS, global longitudinal strain; LV, left ventricle; LVEF, LV ejection fraction; TAVI, transcatheter aortic valve implantation.

Global Longitudinal Strain assessment by 4Dimensional XStrain speckle tracking echocardiography (4DXStrain)

4DXStrain™ merges advanced technology with Tomtec GMBH's 3D/4D rendering and Beutel™ computation capabilities. Utilizing LV border tracking obtained with XStrain™ 2D on standard 4CH, 2CH and apical LX views, XStrain™ 4D delivers a more complete picture of cardiac function.[7]

CASE REPORT

An eighty year old asymptomatic woman afflicted with severe calcific AS alongwith hypothyroidism, type 2 diabetes mellitus and hypertension was referred to our cardiology OPD for color echocardiography and opinion regarding management of severe AS. Otherwise, the patient was well controlled on anti diabetic, anti hypertensives and hypothyroid medications. There was no history of syncope, angina or dyspnea.

On clinical examination, the patient was healthy looking and normally built (Figure 3). The patient's weight was 52 kg, height was 150 cm, pulse rate was 70/min, blood pressure was 134/80 mmHg, respiratory rate was 16/min and SPO2 was 97 % at room air. All the peripheral pulses were normally palpable without any radio-femoral delay. Cardiovascular examination revealed Grade 3/6 ejection systolic murmur at the right, left sternal edge and LV apex. Systemic examination was normal.

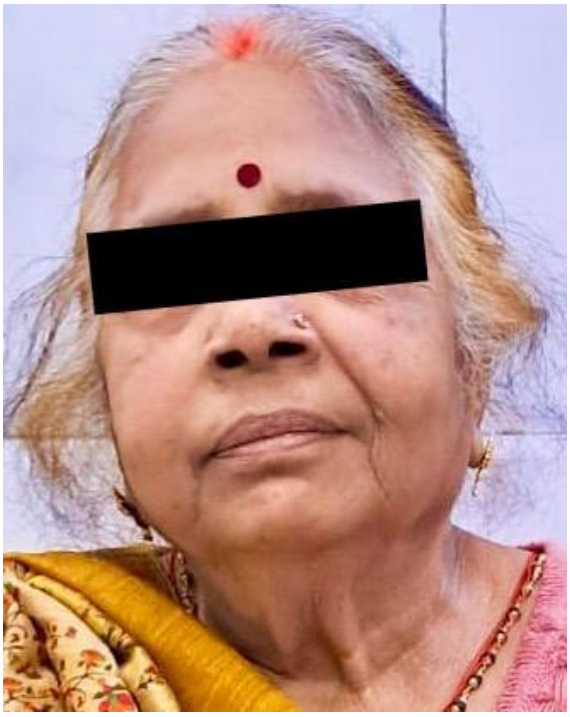


Figure 3: Facial appearance of our index patient.

Xray chest (PA) view (Figure 4) was normal and the cardiac size was within normal limits. There was no evidence of pulmonary venous hypertension.

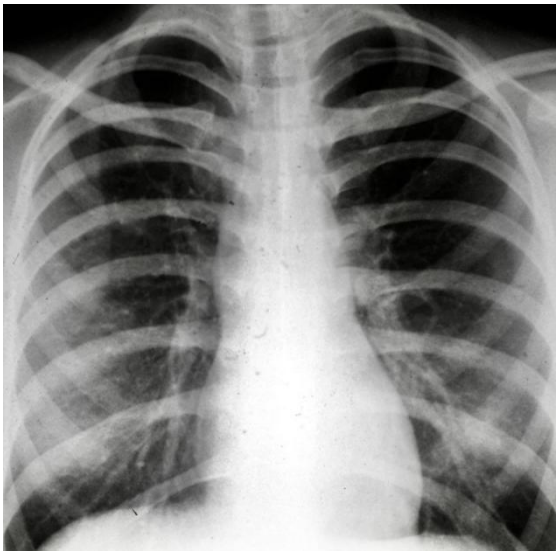


Figure 4: X-ray chest PA view: Cardiac size is normal. There is no evidence of pulmonary venous hypertension.

The resting ECG (Figure 5) was also normal.

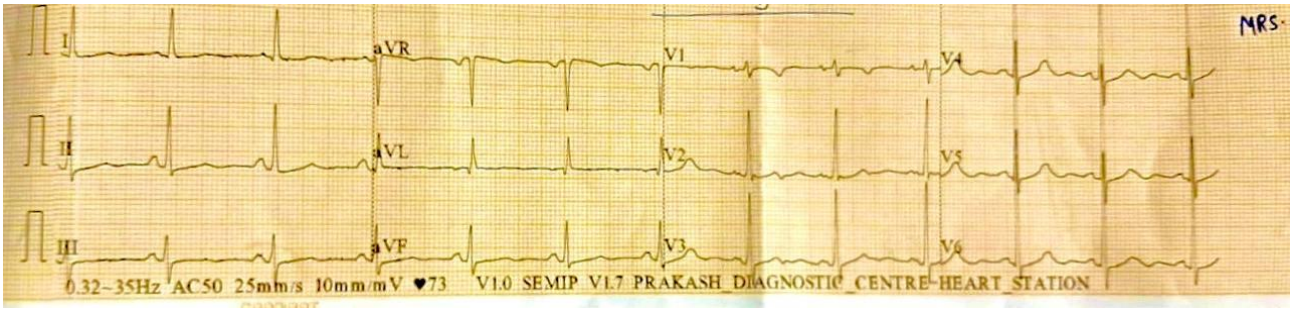


Figure 5: Resting ECG: There is normal sinus rhythm with a ventricular rate of 74/ min. No atrial or ventricular hypertrophy was detected. The QRS axis was normal.

Transthoracic Echocardiography

All echocardiography evaluations were performed by the author, using My Lab X7 4D XStrain echocardiography machine, Esaote, Italy. The images were acquired using an adult probe equipped with harmonic variable frequency electronic single crystal array transducer while the subject was lying in supine and left lateral decubitus positions.

Conventional M-mode, two-dimensional, pulse wave doppler (PWD) and continuous wave doppler (CWD) echocardiography was performed in the classical subcostal, parasternal long axis (LX), parasternal short axis (SX), 4-Chamber (4CH), 5-Chamber (5CH) and suprasternal views (Figures 6-10).

M-mode Echocardiography

M-mode echocardiography of left ventricle was performed and the estimated measurements are outlined (Table 1, Figure 6).

Table 1: Calculations of M-mode echocardiography.

Measurements	LV
IVS d	13.4 mm
LVID d	39.4 mm
LVPW d	11.1 mm
IVS s	20.7 mm
LVID s	21.0 mm
LVPW s	18.4 mm
EF	79 %
% LVFS	47 %
LVEDV	67.5 ml
LVESV	14.5 ml
SV	53.1 ml
LV Mass	167 g

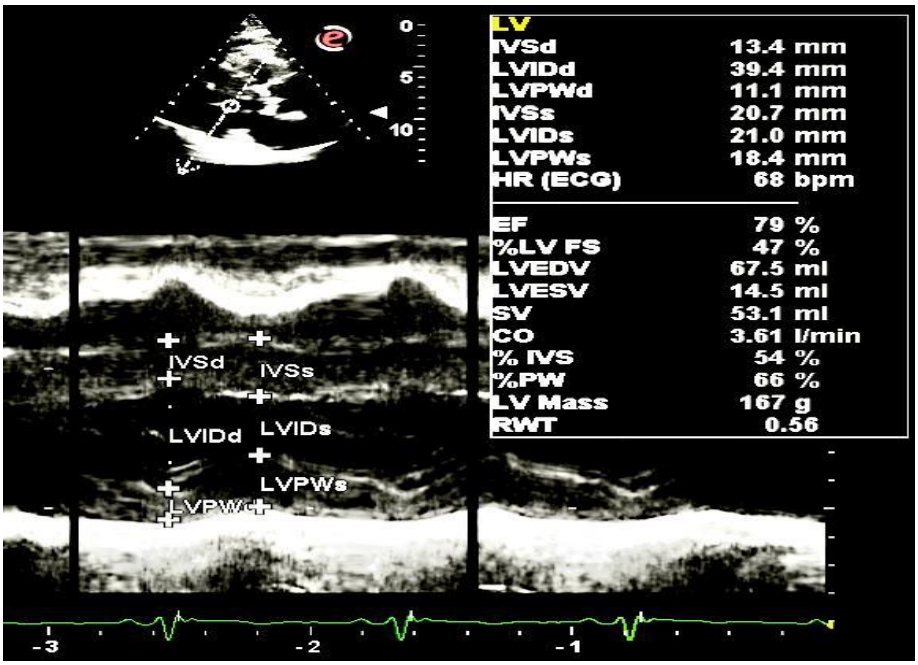


Figure 6: M-mode measurements of LV. There is concentric hypertrophy of LV. LV cavity size was small and the systolic function was normal.

Summary of M-mode echocardiography

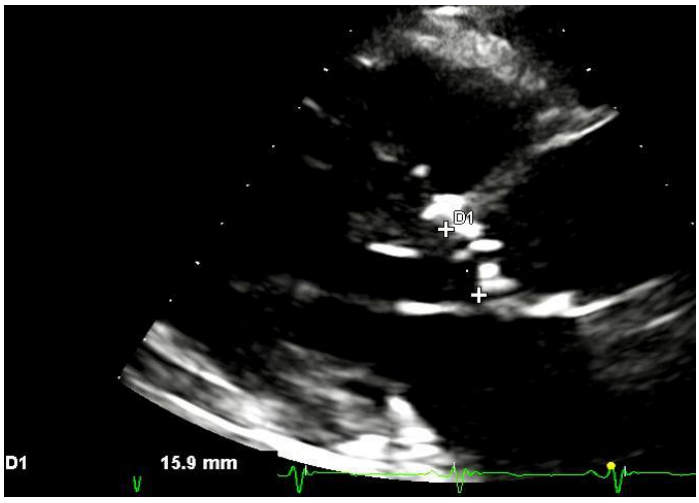
M-mode echocardiography depicted concentric hypertrophy of LV. The LV cavity was small with normal LV systolic function - LVEF 79%.

2 Dimensional transthoracic echocardiography

2-Dimensional transthoracic echocardiography (2D TTE) was performed in explicit detail, particularly to look for any regional wall motion abnormalities or any valvular regurgitation.

2DTTE Demonstrated

1. Aortic valve stenosis (severe) calcified (Figure 7).
AV tricuspid, domed, calcified.
AV velocity = 4.63 m/sec
Peak/mean gradient across AV 85/49 mmHg.
AV area by continuity equation 0.69 sqcm.
Trace AR was detected



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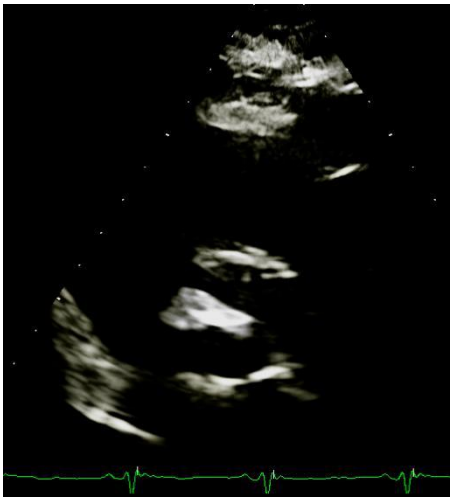

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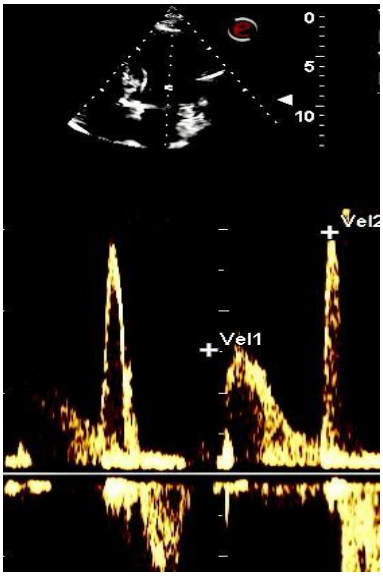

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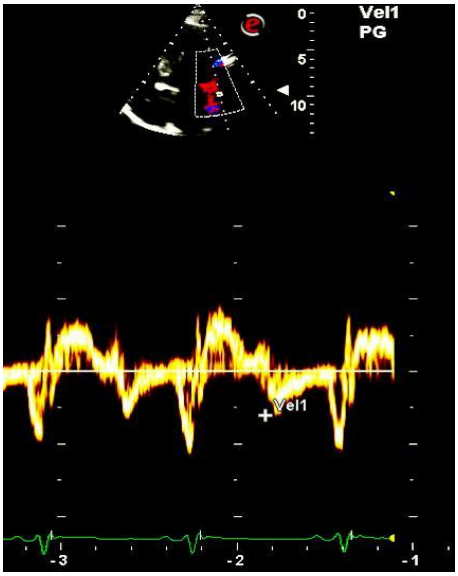

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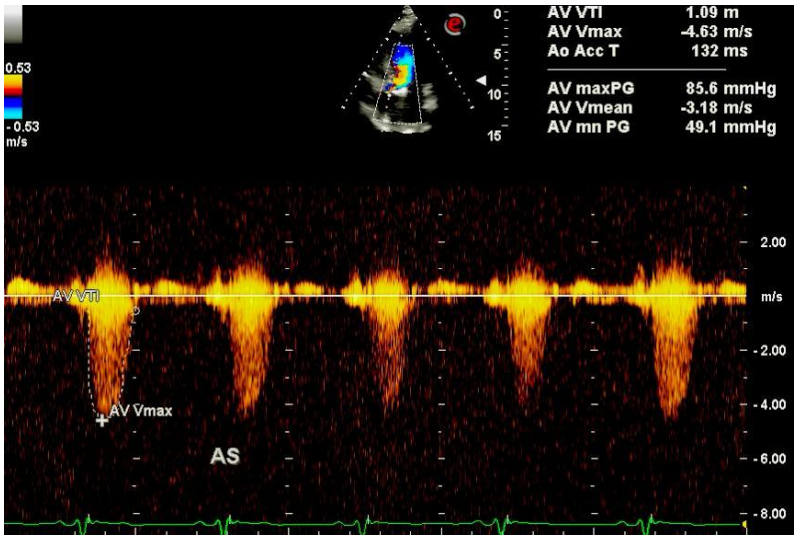


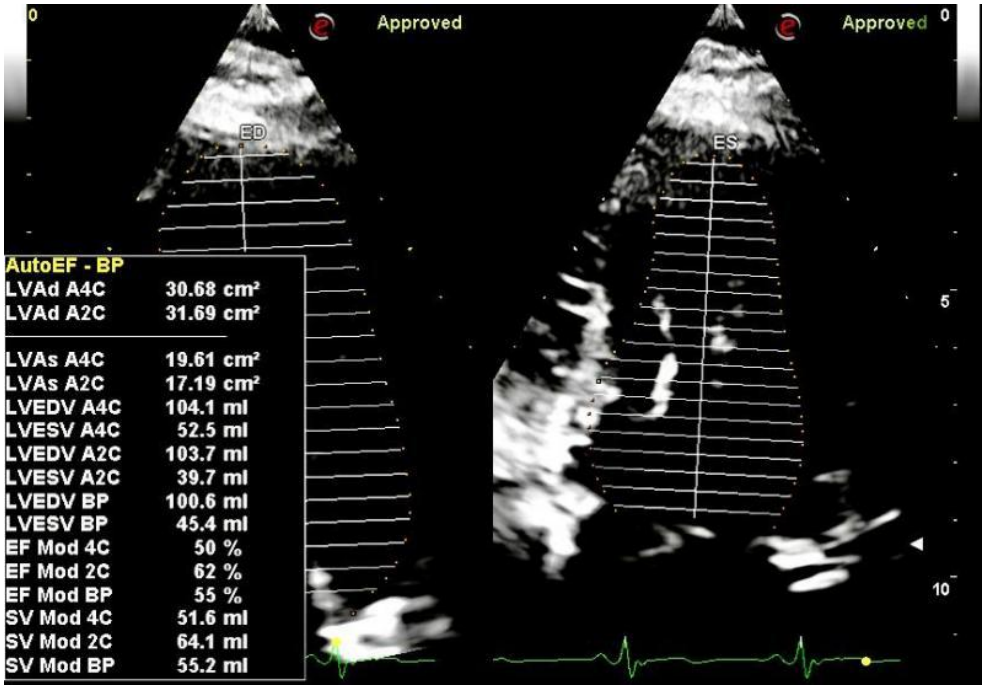
(e)

Figure 7: 2-Dimensional Transthoracic Echocardiography- Severe calcific aortic stenosis, diagnosis and consequences. (a) Parasternal LX view showing small calcific aortic valve annulus (D=15.9 mm); (b) Parasternal SX view identifies tricuspid calcified aortic valve; (c) Pulse wave Doppler analysis across mitral valve highlights LV diastolic relaxation dysfunction (Diastolic dysfunction grade l); (d) Tissue Doppler Imaging of basal lateral LV wall reveals diastolic relaxation dysfunction grade l; (e) Continuous wave Doppler analysis across the aortic valve displays a peak and mean gradient of 85.6/49.1 mmHg.

2. LV volumes and systolic function (Table 2, Figure 8)

Table 2: LV volume and systolic function.

Variable	Simpson's method	4 Dimensional method
EDV	100.6 ml	44.79 ml
ESV	45.4 ml	12.20 ml
Sph i d	-	0.27
Sph i s	-	0.13
EF	55 %	72.77 %



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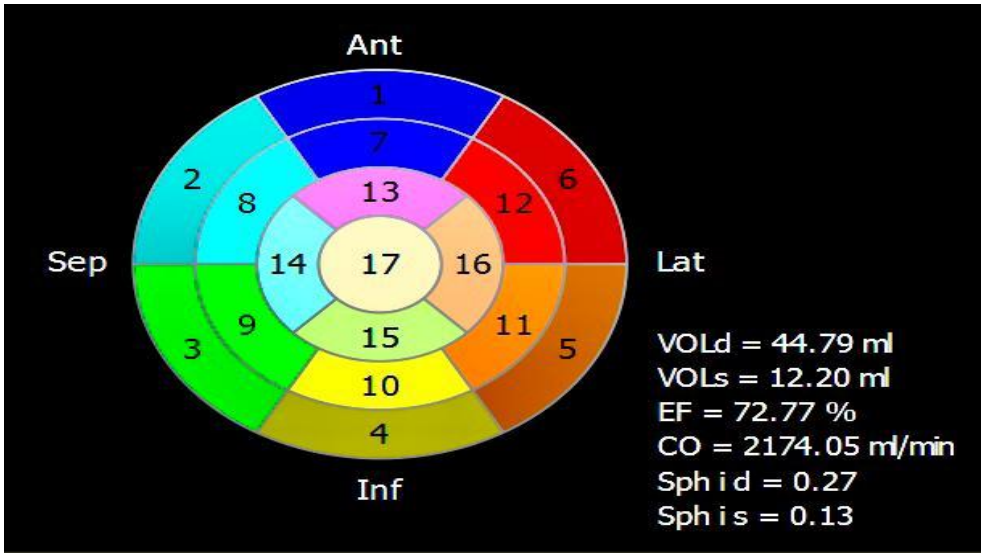


(B)

Figure 8: LV volumes and systolic function. (A) Simpson's biplane method; (B) 4 Dimensional volumes, sphericity index and systolic function. Sph i d , sphericity index; d, diastole; s, systole; EF, ejection fraction.

Table 3: Comprehensive LV volumes and EF calculations by Simpson's biplane method.

Auto EF - Biplane					
LVAd A4C	30.68	cm²	LVAd index A4C	21.0	cm²/m²
LVAs A4C	19.61	cm²	LVAd A2C	31.69	cm²
LVAd index A2C	11.8	cm²/m²	LVAs A2C	17.19	cm²
LVEDV (MOD A4C)	104.1	ml	LVESV (MOD A4C)	52.5	ml
LVEDV (MOD A2C)	103.7	ml	LVESV (MOD A2C)	39.7	ml
LVEDV (MOD BP)	100.6	ml	LVESV (MOD BP)	45.4	ml
LVEDV index (MOD A4C)	71.3	ml/m^2	LVEDV index (MOD BP)	68.9	ml/m²
EF (MOD A4C)	50	%	EF (MOD A2C)	62	%
EF (MOD BP)	55	%	SV (MOD A4C)	51.6	ml
SV (MOD A2C)	64.1	ml	SV (MOD BP)	55.2	ml
SV index (MOD A4C)	35.4	ml/m²	SV index (MOD A2C)	43.9	ml/m²
SI (MOD BP)	37.8	ml/m²			

3. There was distinctive concentric hypertrophy of LV with small cavity size.

- No regional wall motion abnormality was present.
- LV diastolic relaxation dysfunction (Diastolic dysfunction Grade –I) was discerned on pulse wave doppler across mitral valve and tissue doppler imaging of .the basal lateral wall of LV.

4. Mild PAH (RVSP/PAP = 42 mmhg) was observed

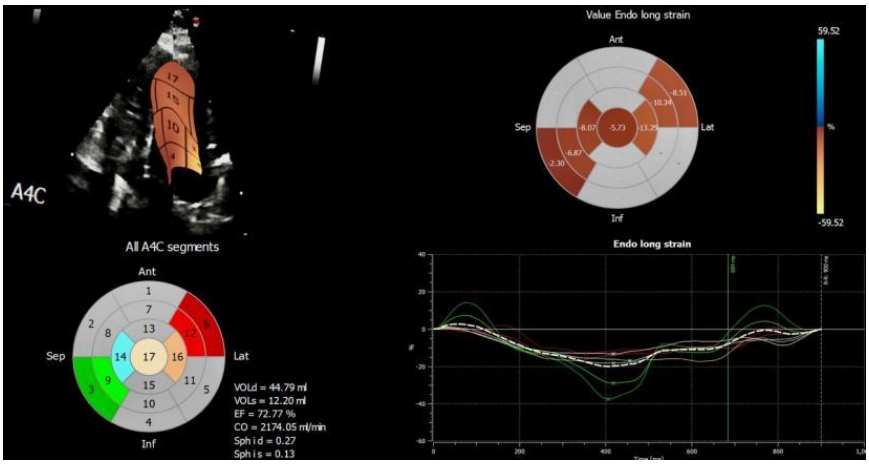
Global longitudinal strain assessment

GLS was estimated by 4DXStrain speckle tracking echocardiograyphy and the peak global longitudinal strain values obtained in our patient are displayed in Table 4 and Figure 9.

Table 4: Peak global longitudinal strain obtained from different echocardiographic views.

Global Strain (A2C)	-16.60 %
Global Strain (ALAX)	-18.35 %
Global Strain (A4C)	-19.42 %
Global Strain	-18.12 %
A, apical; 2C, chamber; LAX, long axis; 4C, 4 chamber	

(A)



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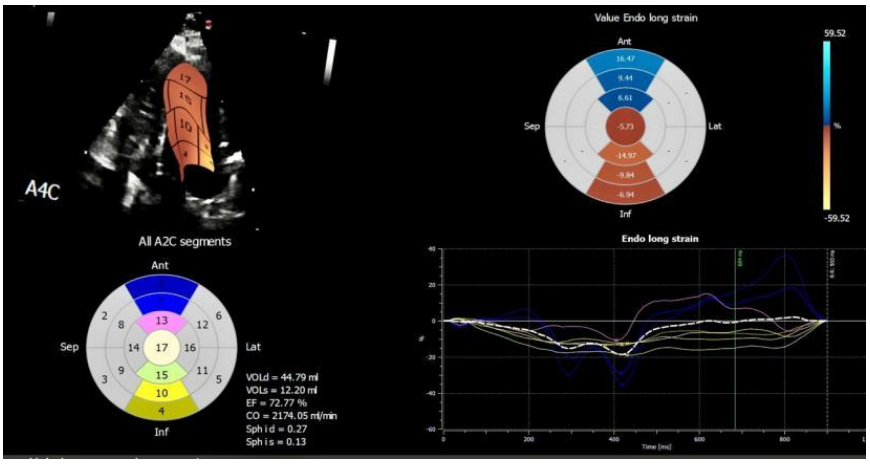

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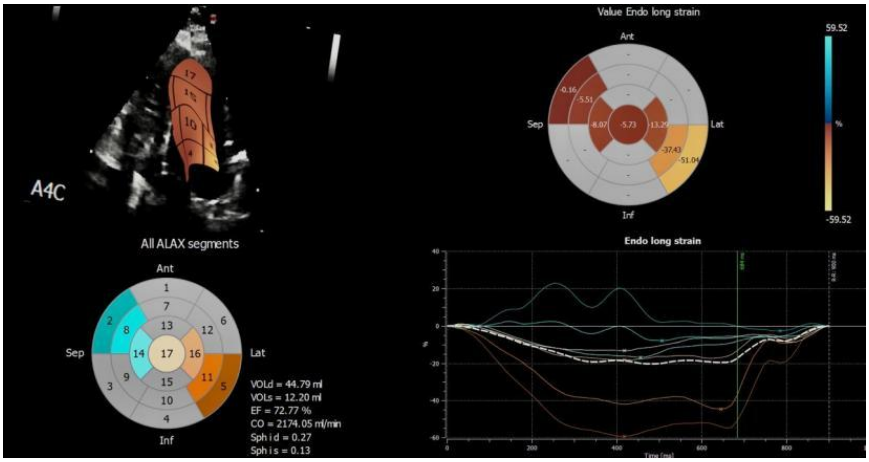

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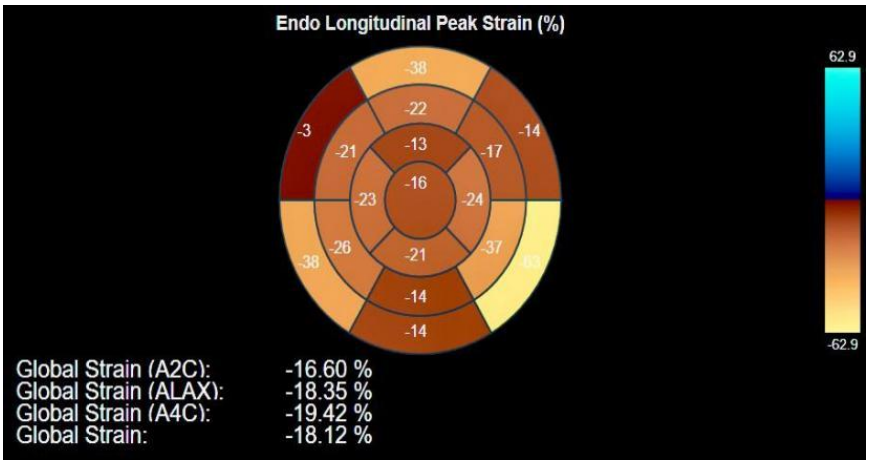


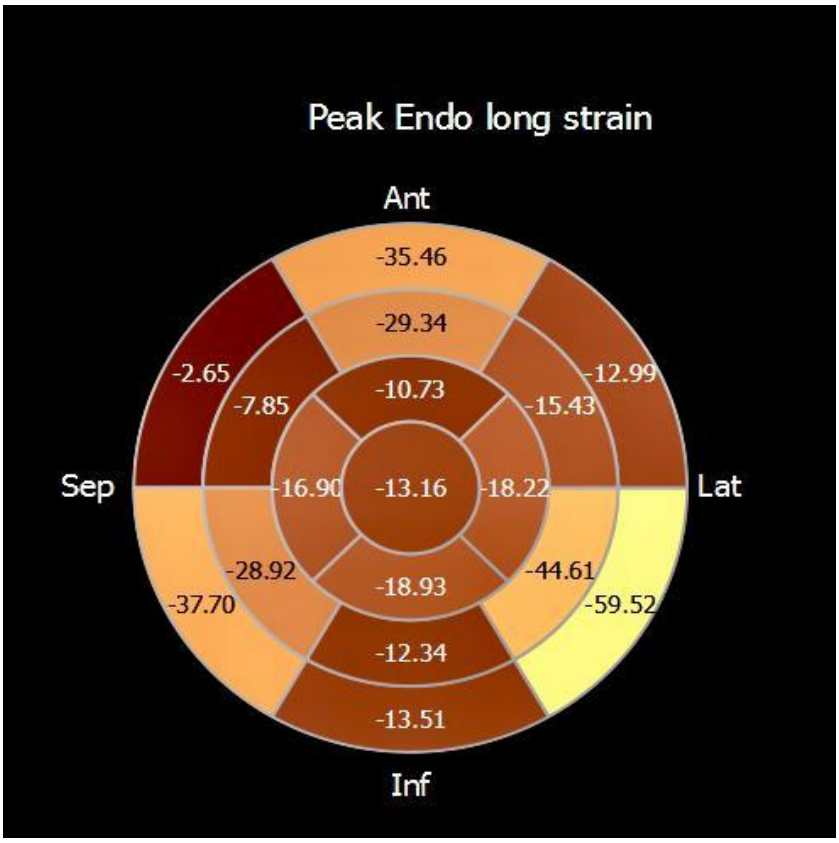
Figure 9: Peak global longitudinal strain evaluation. (A) A4C view; (B) A2C view; (C) ALAX view; (D) Global strain.

17 segment model of 4DXStrain segmental GLS was obtained, based on American Heart Association (AHA) model (Table 5, Figure 10).

Table 5: Bull's eye analysis of LV segmental strain values derived from 4D XStrain speckle tracking echocardiography.

17 segment model	Peak longitudinal strain		Time to peak longitudinal strain	
Bas Ant	-38.00	%	439	ms
BasAntSep	-3.38	%	774	ms
Bas Sep	-38.18	%	386	ms
Bas Inf	-14.37	%	398	ms
Bas Post	-62.92	%	419	ms
Bas lat	-14.25	%	345	ms
Mid Ant	-21.61	%	425	ms
MidAntSep	-20.91	%	419	ms
Mid Sep	-26.07	%	400	ms
Mid Inf	-14.20	%	617	ms
Mid Post	-37.13	%	651	ms
Mid Lat	-16.57	%	331	ms
Apic Ant	-13.26	%	721	ms
Apic Sep	-22.64	%	624	ms
Apic Inf	-20.75	%	377	ms
Apic lat	-23.89	%	656	ms
Apex	-16.26	%	488	ms
Global Strain (A2C)	-16.60	%		

Global Strain (A4C)	-19.42	%		
Global Strain (ALAX)	-18.35	%		
Global Strain	-18.12	%		



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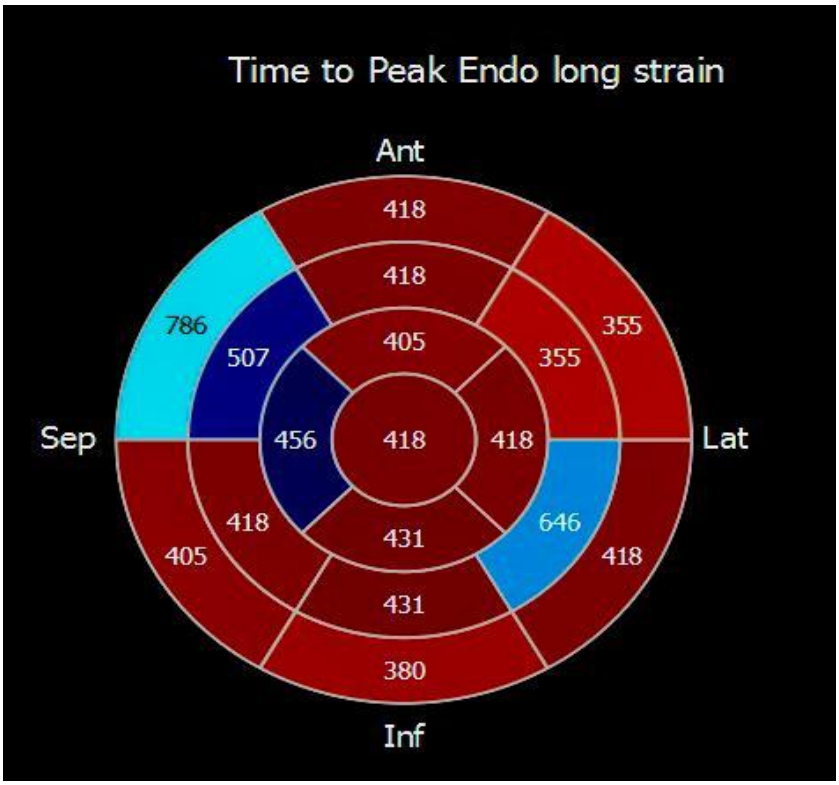

(B)

Figure 10: (A) Peak endocardial strain; (B) Time to peak endocardial longitudinal strain.

DISCUSSION

The best timing of AVR for asymptomatic patients with AS has been the epicenter of considerable interest during the last decade.[8] According to the latest American and European guidelines, the EF of 50% was used as the threshold for surgery among asymptomatic severe patients with AS.[9,10] However, data from recent published studies demonstrated that singular LVEF might be insufficient to risk-stratify those patients for surgery.[11,12] LVEF values could be falsely enhanced by the geometric effect, which might limit its ability to identify mild or moderate LV dysfunction. Of note, Ng et al[13] stated that up to 23 (13.1%) deaths occurred in patients without severely impaired EF, because they fell outside the recommendations.

It is well known that there is reduction in deformation values prior to LVEF impairment in AS. Thus, GLS has emerged as an accurate and reproducible tool to identify the subclinical LV impairment of AS.[5] According to a series of recent studies, identification of reduced longitudinal strain rate caused by the injured subendocardial myocardial fibers had correlations with mortality among asymptomatic patients with AS.[14-16] However, the prognostic value of GLS in asymptomatic patients with AS has been demonstrated merely in small single-center studies without further confirmation in larger patient populations.[14-22]

Only one meta-analysis evaluated the effect of impaired GLS on prognosis in asymptomatic AS patients.[23] This meta-analysis, which used individual participant data from 10 studies, demonstrated that patients with impaired GLS, which defined by the cut-off value of 14.7%, had 2-fold greater risk than those with normal GLS over the course of the follow-up. According to Wang et al[6] GLS could predict or screen patients with asymptomatic AS who were likely to progress into symptomatic stage and required surgery.

Impaired GLS was present in a considerable proportion of patients (52.2%), and it had significant associations with MACE, all-cause mortality, or AVR.[6] It should be noticed that stronger correlations between impaired GLS and MACE was observed when involving patients with EF > 50% only.[6] GLS might help identify asymptomatic patients with AS at high risk of poor outcomes. Hence, one is bound to speculate if early intervention or frequent monitoring might benefit patients with impaired GLS, independent of LVEF.[6]

In terms of accuracy, sensitivity, and reproducibility, GLS by echo seems to perform better than other methods of estimation. But for patients with suboptimal imaging quality on echo, CMR or MDCT could represent a valid alternative.[6]

The presence of impaired GLS offers opportunities to identify asymptomatic patients with AS who are at high risk of adverse prognosis and therefore act accordingly. This could help reduce costs associated with repeat admissions of normal patients with impaired GLS. The meta analysis of Wang et al[6] may help address the unsolved issue of whether signs of LV impairment could be used to optimize the timing of valve intervention SAVR or TAVI. An ongoing trial (Danish National Randomized Study on Early Aortic Valve Replacement in Patients with Asymptomatic Severe Aortic Stenosis (DANAVR); NCT03972644), which randomly assigns asymptomatic patients with AS to undergo AVR or watchful waiting, would shed more light on this hot issue.[6]

Despite the perpetual increase in the global health care burden of AS, there are no preventive or disease-modifying medical treatments. The only curative intervention is aortic valve replacement (AVR), which bears multiple risks. Consequentially, the optimal time and modality of intervention in patients with asymptomatic severe AS (ASAS) are controversial.[24]

Premature intervention may predispose individuals to unnecessary risks of AVR, while irreversible cardiac damage, with resultant heart failure (HF) or even death, may precede delayed intervention. Thus, for the optimal management of the populations with ASAS, a vigilant approach to this increasing dilemma and careful consideration must govern decisions for treatment, keeping in mind patient preferences and targeted goals of possible prognostic predictors.[25]

Current guidelines indications for valve replacement

The indications for AVR in ASAS (Table 6, 7) can be used to guide the management of ASAS by early intervention (TAVR or SAVR) or watchful waiting with meticulous patient education and reassessment at regulated intervals.

Table 6: Indications for aortic valve replacement in asymptomatic severe aortic stenosis.[26]

LVEF <50% (Class Ib recommendation)
Low surgical risk, with decreased exercise tolerance or fall in SBP ≥20 mmHg from baseline to peak exercise evident on exercise testing (Class Ic)
Very severe AS (mean gradient ≥60 mmHg, aortic velocity of ≥5 m/s) and low surgical risk (Class IIa)
High-gradient severe AS with low surgical risk and serial testing reveals an incremental increase in aortic velocity ≥0.3 m/s per year (Class IIa)
Severe AS and low surgical risk with BNP level>3 times the normal range (Class IIa)
LVEF: Left ventricular ejection fraction, SBP: Systolic blood pressure, AS: Aortic stenosis, BNP: Brain natriuretic peptide.

Table 7: Predictors of symptom development and adverse outcomes in asymptomatic severe aortic stenosis.[26]

Clinical characteristics (older age, atherosclerotic risk factors)
Pro-BNP >3 folds of normal value in serial follow up measurements
Peak velocity >5 m/s
Rapid progression of AS severity (peak jet velocity increase>0.3 m/s/year)
Increase in mean gradient >20 mmHg with exercise
Severe LV hypertrophy
Decreased indexed stroke volume
Valvuloarterial impedance >5 mmHg/ml/m2
Reduced LV global longitudinal strain >14.7%
Increased left atrial size (a′: Peak late diastolic velocity by tissue <9 cm/s)
Reduced mitral annular systolic (s') and late diastolic velocities (a')
Systolic pulmonary artery pressure >60 mmHg
Mid-wall LGE on cardiac magnetic resonance imaging
AS: Aortic stenosis, LV: Left ventricular, LGE: Late gadolinium enhancement, Pro-BNP: Pro-B-type natriuretic peptide

As per the latest European Society of Cardiology (ESC)/European Association for Cardio-Thoracic Surgery (EACTS) valvular guidelines 2021[26], asymptomatic patients with severe AS who do not have an indication for intervention, watchful waiting is a safer and more appropriate strategy unless they have one or more of the predictors of rapid hemodynamic progression which can switch the patient management strategy to early surgical intervention instead of watchful waiting as they face a higher risk of adverse outcomes.[25]

CONCLUSIONS

The presence of impaired GLS substantially worsens the outcomes for adverse cardiovascular events in asymptomatic patients with AS regardless of LVEF or AS severity or mean aortic valve pressure gradient, which highlights the importance of incorporating impaired GLS into risk algorithms in asymptomatic AS.

Our index patient was reluctant to undergo AVR by TAVR or by SAVR. Due to her asymptomatic status and normal peak GLS value of -18.2%, we also did not insist on any invasive intervention and advised her to be watchful for appearance of any symptoms (Syncope, Angina or Dyspnea). Moreover, she was suggested regular checkups at 6 monthly interval and thorough aortic valve evaluation by yearly echocardiography.

Compliance with ethical standards

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional committee of Prakash Heart Station, Nirala nagar, Lucknow.

Informed consent

Informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of interests

The authors declare that they have no conflict of interest.

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