Research Brief

Tissue tracking of segmental strain as a predictor of provoked dynamic left ventricular outflow tract obstruction

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ABSTRACT

Background: Left ventricular outflow tract obstruction (LVOTO) is commonly observed in patients with hypertrophic cardiomyopathy (HCM) or left ventricular hypertrophy (LVH). Some patients develop LVOTO provoked by physical exertion, and hence termed dynamic LVOTO (DLVOTO). However, its precise prevalence and mechanism are still unclear.

Aim: Two-dimensional speckle tracking echocardiography (2D STE) seems to be helpful for the detection of early LV structural abnormalities. This study aimed to examine the possible role of segmental as well as global longitudinal strain in identifying DLVOTO non-HCM patients as detected by dobutamine stress echocardiography (DSE).

Methods and results: Two hundred and fifty patients without structural heart disease had undergone conventional transthoracic echocardiography, 2D STE, and DSE. All patients with non-ischemic evidence were divided into two groups according to the DSE results; DLVOTO (+) and DLVOTO (−). Among 250 patients, 50 patients (36%) had shown DLVOTO after DSE (15 males, 35 females; mean age 55±7years). They were compared with 90 non-LVOTO obstruction patients (43 males, 47 females; mean age 57±6years). Based on multivariate logistic regression analysis, the independent predictors of provoked DLVOTO during DSE were resting basal septal longitudinal strain BS-LS $_{average}$ (p < 0.001), resting LA reservoir strain (p < 0.001), and systolic LVOT diameter (p = 0.03). Resting BS-LS $_{average}$ with cut-off -17.5% was recognized as a critical indicator of DLVOTO, with sensitivity 78%, and specificity 95% (better than systolic LVOT diameter of sensitivity 76%, and specificity 15% and resting LA reservoir strain which showed poor AUC at ROC curve 0.007).

Conclusion: We demonstrate that provoked LVOTO during DSE in non HCM symptomatic patients is directly correlated to resting regional LS, where the increased BS-LS of $\geq -17.5\%$ was a key determinant of LVOT gradient provocation. Assessment of baseline BS-LS $_{average}$ might be a bedside simple tool for detection of patients with DLVOTO not able to do DSE.

1. Introduction

It is currently assumed that provoked DLVOTO which is defined as a trans-LVOT peak pressure gradient (PG) higher than 30 mmHg provoked by Valsalva maneuver, and/or LVOT peak (PG) higher than 50 mmHg during stress echocardiography without fixed stenosis; accounts for a crucial underestimated cause of unexplained exertional dyspnea and adverse consequences in patients without structural heart disease.[1,2] Its occurrence requires the coexistence of predisposing anatomic factors and a physiological condition. Exercise or DSE are reliable screening modalities for DLVOTO diagnosis.[3] DLVOTO can develop due to various geometric changes.[4] However, the increased contractility of the basal-septal segment is probably an essential factor in the provoked obstruction and therefore may be used to identify subgroups of patients at increased risk of provoked DLVOTO.

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Tissue tracking technique where tissue regions are identified by the presence of speckles, referred to as speckle-tracking echocardiography STE, has been evolved to express the diagnostic information numerically. It is more sturdy and reproducible for global as well as regional function assessment.[5] In this study, we investigated the resting global and regional left ventricular systolic function using STE in patients who developed the DLVOTO phenomenon while undergoing DSE to evaluate the cause of unexplained exertional dyspnea, chest pain, and/or syncope.

2. Materials and methods

2.1. Study population

Among 250 patients without structural heart disease, who were referred for DSE between march 2021 and January 2022 to evaluate exertional chest pain, dyspnea, and/or pre-syncope, 140 patients who were found not to have stress-induced wall motion abnormalities were included. They were recruited into two groups based on the measurement of LVOT gradients by DSE, 50 patients exhibited DLVOTO at peak stress during DSE (DLVOTO group) and 90 patients with normal LVOT gradient at peak stress (non-obstruction group). Patients with left ventricular hypertrophy are defined as an increased left ventricular mass index (LVMI) of greater than 95 g/m in women and greater than 115 g/m in men according to the American Society of Echocardiography and/European Association of Cardiovascular Imaging.[6] LVOT velocity ≥2.5 m/s at rest, systolic dysfunction (EF <50%), significant valvular heart disease, previous cardiac surgery, bundle branch block, atrial fibrillation, and poor echocardiographic window were all excluded.

This study was approved by the institutional review board (IRB) and conducted at the cardiology department, Zagazig university hospital. All procedures were performed in accordance with the ethical standards of the national research committee. Written informed consents were obtained from all participants. Each patient had undergone conventional transthoracic echocardiography, STE, and DSE.

2.2. Echocardiography

The echocardiographic examination was performed using a VIVID E95 ultrasound machine and images were acquired with the patient in lateral decubitus, 3 consecutive cardiac cycles of each view were recorded during quiet breathing at frame rates: 50–80 frames/sec. All echocardiographic measurements were taken in accordance with the recommendations of the American Society of Echocardiography (ASE).[6] Left ventricular outflow tract diameter was measured in the longitudinal plane during systole as the shorter distance between the anterior mitral valve and the interventricular septum. Left ventricular mass was calculated according to the modified Devereux formula for the ASE standards. BSH was defined as either a diastolic basal septal thickness 2 standard deviations above the normal mean (greater than 1.4 cm), or 50% greater than the thickness of the septum at its mid-point.[7]

Left ventricular ejection fraction (LV EF) and Left atrial (LA) volumes were recorded using biplane methods. Doppler measurements included LVOT maximum gradient, mitral inflow velocities (the peak early (E) and late (A) transmitral flow velocities), deceleration time of E velocity and the ratio of early-to-late peak velocities (E/A). Tissue Doppler imaging was performed to measure the early diastolic (E′) myocardial peak velocity at the lateral and septal corners of the mitral valve annulus and averaged from both positions. Thereafter, the E/E′ ratio was calculated.[8]

2.3. 2D speckle tracking

The contour of the endocardium at apical four, two, and three-chamber views was traced automatically at frame rates between 50 and 80 frames per second, with the inclusion of six segments of each view. The automatically generated ROI was visually assessed with manual correction of inadequate contouring. Peak systolic longitudinal strain for each segment, and average strain for the whole LV were derived during automated aortic valve closure detection[9] (Figs. 1 and 2). Normal GLS assessed by STE is in the range of −18 to −20%, with a smaller magnitude of the longitudinal
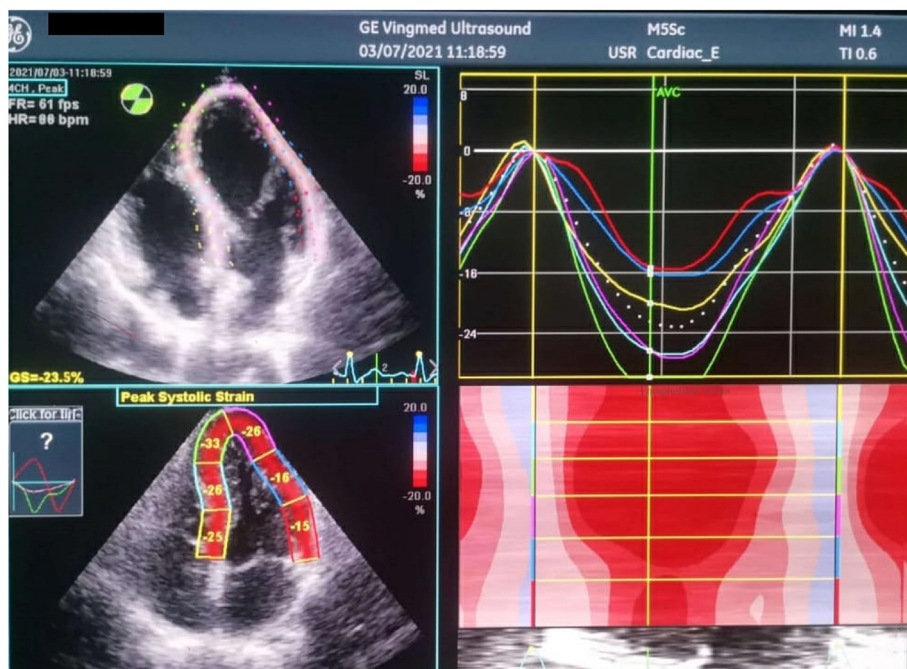

Fig. 1. Segmental Longitudinal strain at apical 4- chamber view showing high value of segmental strain at basal infero-septum.
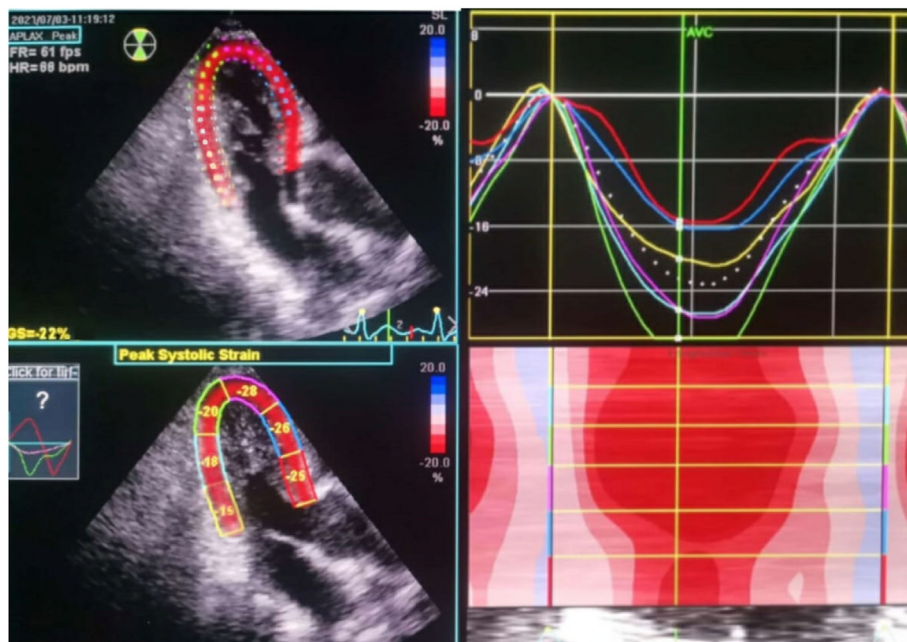
Fig. 2. Segmental Longitudinal strain at apical 3-chamber view showing high segmental strain at basal anteroseptal.

strain of the basal segments as compared with the apical segments[10] (Fig. 3).

Left atrial reservoir strain (LASr) during ventricular systole analysis was obtained from an optimized apical four-chamber view using an automated approach, LAS analysis was calculated with the reference point set at the onset of the QRS complex of the superimposed ECG, the first peak positive deflection corresponds to the value of LAS reservoir function.[11] Normal reference range for LASr is 38%–41%.[12]

2.4. Dobutamine stress echocardiography

DSE procedure was performed according to the ASE guidelines.[8] The electrocardiogram was monitored continuously and blood pressure was measured at rest, at the end of each stage, and during recovery. LVOT peak velocity was acquired at the end of each stage and at peak stress with continuous-wave Doppler. Patients with

LVOT peak gradient >50 mmHg with a late-peaked curve at CW Doppler during DSE were defined as DLVOTO (Fig. 4).

2.5. Statistical analysis

Statistical analysis was done using computerized software statistical packages (SPSS for Windows® version16). Data were expressed as mean ± SD for continuous variables or as numbers & percentages for categorical variables. Variables of the study were compared utilizing the unpaired *t*-test, chi-square, or Fisher's correct test, as fitting. Pearson's relationship coefficient was calculated to precise the connection between different variables. All tests were two-sided. Variables that were statistically significant in univariate analysis, were utilized in multivariate linear regression analysis to detect predictors of DLVOTO. Receiver operating characteristics (ROC) curve analysis was performed to detect the optimal cut-off for predictors to distinguish cases with and without DLVOTO at
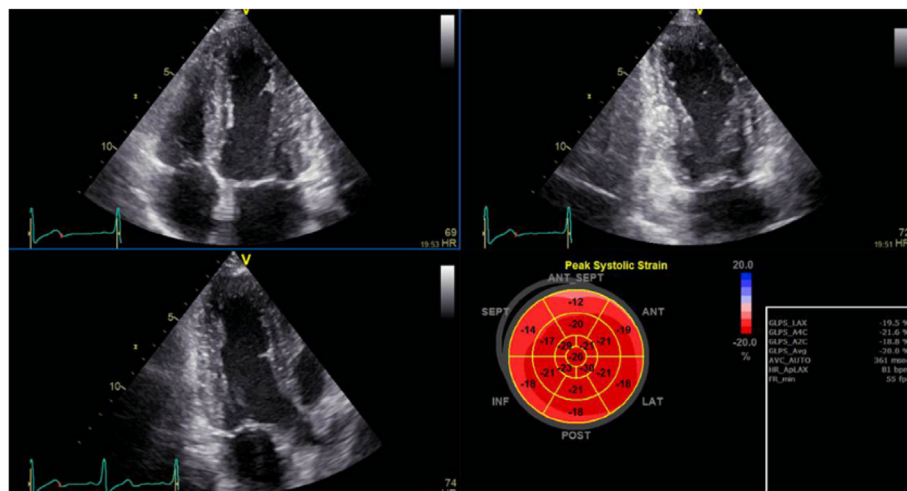

Fig. 3. Global and segmental left ventricular longitudinal strain assessment from the apical 4, 2, and 3- views.
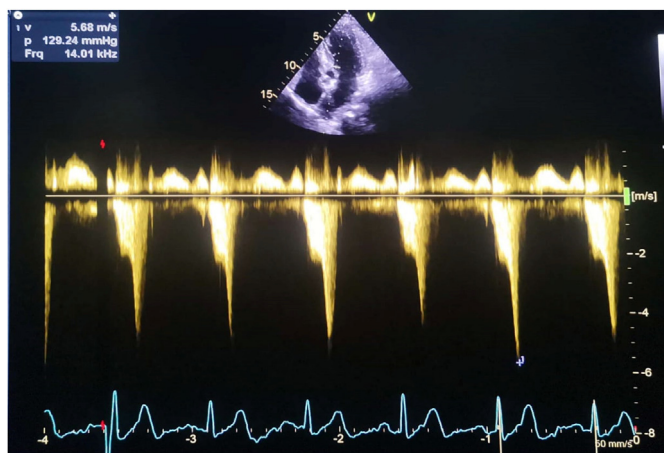
Fig. 4. Dynamic LVOTO detected by CW Doppler during DSE.

resting state. All analysis was considered statistically significant at P value of <0.05.

3. Results

DSE increased LVOT gradient to ≥50 mmHg in 50 Patients (36%) who are assigned as provoked DLVOTO group (15 males, 35 females; mean age 55±7years)., while 90 patients had inconsequential LVOT gradient and were assigned as a non-obstructive group (43 males, 47 females; mean age 57±6years).

No differences in age, the prevalence of hypertension, diabetes, smoking, history of chest pain or syncope, resting heart rate, resting systolic blood pressure, and peak heart rate between DLVOTO and non-obstructive groups (p > 0.05). There was a significant increase in body mass index, and female preponderance with a significant reduction of systolic blood pressure in the DLVOTO group at peak DSE compared to the non-obstructive group (p < 0.01) (Table 1).

No differences in Basal Septal thickness, mid septal thickness, Basal/mid Septal thickness ratio, left ventricular wall thickness, resting EF, resting E/E′, and LV- GLS between both groups (p > 0.05). Patients with DLVOTO had mild mitral regurgitation during DSE (p < 0.02) and a significant increase in longitudinal strain in BS-LS average (p < 0.001), with a significant decrease of LVOTDS, and LASr p < 0.001) (Table 2).

The peak LVOT gradient for the whole cohort using DSE showed a significant positive correlation to BMI, resting EF, and BS-LS average (p < 0.001, p = 0.002 & p < 0.001 respectively) with a significant negative correlation with peak blood pressure, LVOT diameter, and LASr (P < 0.001, P = 0.008 & P < 0.001 respectively) (Table 3).

Based on multivariate logistic regression analysis, resting BS-LS (p = 0.000), resting LASr (p = 0.000), and systolic LVOT diameter (p 0.03) were found to be independent predictors of latent LVOT obstruction during DSE (Table 4).

Only, BS-LS showed the highest AUC at ROC analysis. BS-LS Cut-off value of −17.5% versus LVOTDs and LASr, provided an appropriate diagnostic performance to detect DLVOTO (AUC 0.805; 95% CI 0.92−0.98; p 0.000; sensitivity 70%, specificity 92%) (Table 5, Fig. 5).

4. Discussion

Previous reports had shown that DLVOTO can be provoked in patients without significant LVH and this phenomenon can cause cardiac events,[1] which necessitates its detection. We excluded HCM patients because their natural history and prognostic considerations are very different from those with DLVOTO.

In accordance with previous studies,[2,14] the current study revealed that DLVOTO is prevalent in 36% of non-HCM and non-ischemic patients, especially in women. The prevalence of DLVOTO was found to develop in almost half of non-LVH patients with malignancy who underwent (DSE) to evaluate preoperative cardiac risk.[15] This higher prevalence seems to be in part due to the clinical background of the patient population where the systemic inflammation elicited in malignancy could affect cardiac performance.[16,17]

Table 1
Demographic & clinical characteristics of studied groups.

Parameter	LDLVOTO (n = 50)	Non-obstruction (n = 90)	P value
Age, years (x ± SD)	55 ± 7	57 ± 6	0.1
Female, n (%)	35 (70%)	47 (52%)	0.03
Hypertension, n (%)	25(50%)	22 (55%)	0.6
DM, n (%)	13(26%)	10(25%)	0.9
Smoking, n (%)	18 (36%)	7 (17.5%)	0.05
BMI (x ± SD)	34 ± 7	29 ± 6	0.000
Complaint, (%)			
Dyspnea	39(78%)	19(21%)	0.003
Chest pain	16(32%)	20(22%)	0.08
Pre- syncope	5(10%)	2(2.2%)	0.3
SBPr, mmHg	130 ± 11	133 ± 13	0.3
HRr, bpm (x ± SD)	75 ± 9	77 ± 14	0.4
SBPp, mmHg	120 ± 14	133 ± 10	0.001
HRp, bpm	140 ± 9	140 ± 7.8	0.9

LDLVOTO, Latent dynamic left ventricular outflow tract obstruction; x, mean; ±, standard deviation; DM, diabetes mellitus; BMI, body mass index; SBP, systolic blood pressure; HR, heart rate; r, resting; P, peak; bpm, beat per minute.

Table 2
Echocardiographic parameters of studied groups.

Parameter	LDLVOTO (n = 50) (x±SD)	Non-obstruction (n = 90) (x±SD)	P value
Basal Septal thickness, mm	12 ± 2.3	12 ± 2.4	0.5
Mid Septal thickness, mm	10 ± 1.4	10 ± 1.1	0.4
Basal septal thickness/Mid Septal thickness	0.84 ± 0.15	0.86 ± 0.22	0.6
LV mass index, g/m²	82 ± 13	90 ± 16	0.1
LVOTDs, mm	16.2 ± 1.8	21 ± 1.4	0.000
EF %	63.6 ± 3.8	62.6 ± 3.9	0.2
E/E′	6.5 ± 1.6	6.6 ± 1.1	0.7
Mild MR (%)	14(28%)	10(11.1%)	0.02
No MR (%)	36(72%)	80(88.9%)	
SAM %	10(20) %	0%	0.000
BS- LS %	−21 ± 4.9	−15 ± 2.9	0.000
LV -GLS, %	−24 ± 2.2	- 23.6 ± 1.6	0.6
LA systolic strain, %	19 ± 3.7	34 ± 6	0.000

LV, left ventricle; LVOTDS, systolic left ventricular outflow tract diameter; PG, pressure gradient; MR, mitral regurgitation; **SAM**, systolic anterior motion of mitral valveEF, ejection fraction; E/E′, peak early transmitral/early diastolic myocardial velocities; BS- LS, basal septum longitudinal strain; GLS, global longitudinal strain.

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Table 3
Correlation of LDVOTO with other clinical & echocardiographic parameters.

Parameter	R	p value
AGE	−0.102	0.2
BMI	0.422	0.000
HR r	0.096	0.4
HR p	−0.096	0.3
BP r	−0.159	0.1
BP p	−0.560	0.000
LVOTDs	−0.225	0.008
Basal Septal thickness	−0.114	0.4
Mid Septal thickness	−0.022	0.8
Basal/Mid Septal thickness	−0.096	0.6
EF	0.202	0.02
E/E	0.144	0.4
E/E′	−0.013	0.9
LA systolic strain	−0.786	0.000
LV -G LS	0.142	0.3
BS-LS	0.831	0.000

Tables 4
Multivariate Linear Regression Analyses of independent predictors of LDLVOTO.

Variable	B Coefficient	95% CI Lower Upper	P value
Left atrial strain	−1.5	−2−−1	0.000
BS-LS	3.95	3−4.9	0.000
EF rest	0.657	- 0.2−1.5	0.2
EF peak	0.481	- 0.2−1.16	0.2
LVOTD$_S$	−1.9	- 3.67 to −0.16	0.03
BMI	−0.008	- 0.5−0.5	0.9
BP rest	0.053	- 0.2−0.318	0.7
BP peak	−0.196	- 0.47−0.083	0.2

Table 5
Cut-off values and performance accuracy of two-dimensional conventional & STE as predictors of LDLVOTO.

Variable	Cut off value	AUC	Sensitivity	Specificity	P value
Basal septal strain	17.5%	0.96	78%	95%	0.000
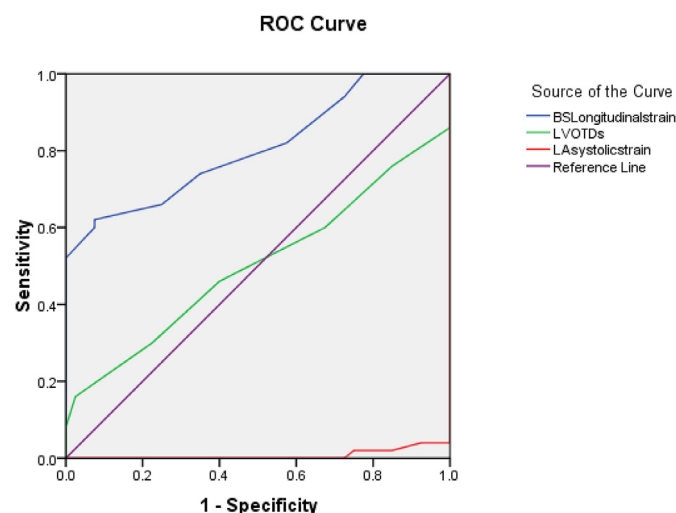
Fig. 5. ROC curve of predictors of DLVOTO.

The clinical symptoms associated with DLVOTO have been noted previously.[18,19] In this study, more patients within the DLVOTO group had experienced exertion-induced dyspnea, pre-syncope, and developed hypotension during peak DSE. It is believed that DLVOTO should be suspected especially in women presenting with hypotension and systolic murmur in critical care settings, this hypotension despite being transient, could be severe.[20]

Ranasinghe et al,[21] stated that DLVOTO occurs only when concomitant mitral valve coaptation and LV hypercontractility facilitate the development of a gradient, rather than the degree of basal septal myocardial hypertrophy. So, it wasn't surprising that the present study found no significant correlation between basal septal thickness and DLVOTO.

The baseline echocardiographic parameters showed that LVOTDS was a significant predictor of DLVOTO. Also, Semba, et al,[15] found that the lower LVOT ratio (systolic LVOT diameter/diastolic LVOT diameter) was a significant predictor of DLVOTO. The hyperdynamic LVOT could be related to the pathogenesis of LVOTO.[22]

LA reservoir strain was also found to be reduced in provoked DLVOTO patients irrespective of E/E'. LA strain could be a promising biomarker to survey early diastolic dysfunction.[23] Similarly, previous studies had revealed that LA strain could be initiated before clinically apparent LVH in hypertensives.[24,25] This may propose an early distinctive abnormal hemodynamics of diastolic function, and hence, we can speculate that DLVOTO may ultimately induce LV diastolic heart failure which could explain the exertional dyspnea in such patients.

There is a lack of reference values for the segmental LV deformation, but previous studies have illustrated an apex-to-base (highest-to-lowest) gradient for the segmental LV LS in normal population. The apex-to-base gradient occurs because the torsional mechanisms of LV deformation are more prominent toward the apex.[26]

In HCM, myocyte disarray/hypertrophy, fibrosis, and coronary arteriole dysplasia are dominant, and predispose to worsening regional LV mechanics, preceding overt LV systolic dysfunction, and can be associated with reduced LV longitudinal myocardial function, especially in the region of the interventricular septum.[27−29] While, in the current study, the main finding was the significant increase in BS-LS in provoked DLVOTO patients without structural heart disease. This unveils the subtle that the provoked DLVOTO is a mild degree of HCM.

BS- LS, rather than LV-GLS, had the finest discriminatory accuracy for DLVOTO presence within the studied patients. Modification of the "physiological" apex-to-base gradient might have the potential to perceive myocardial function change in DLVOTO. Recent findings suggest that in the state of unaltered afterload, STE derived strain reflects LV contractility.[30] This basal septum hypercontractility noticed in the current study can't be attributed to aging-related change as there was no age difference between the studied groups. The earlier mechanical activation of the basal septal segment generates a proximal septal bulge which narrows LVOT resulting in the increased flow velocity,[31] the largest radii longitudinal fibers of the basal septum in human heart and the additional load created by pressure from the right ventricle on the basal septum,[32] could be the proposed mechanisms of basal septal hypercontractility which consequent narrows LVOT in patients with inducible obstruction.

Based on the results of the current study, increased BS-LS may be useful to identify "at risk" DLVOTO patients. The current findings need to be validated, in a multicenter trial.

Additionally, recognizing patients with provoked DLVOTO utilizing the increased BS-LS, might be accommodated in assessing the clinical results of DLVOTO patients. Further studies are required to detect the natural history of provoked DLVOTO. We suggest that negative inotropic medication could relieve the obstruction in such patients.

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4.1. Limitations

- The current study was an observational study from a single center, which could have potential selection bias.
- Although exercise echocardiography is the gold standard for assessing DLVOTO, we used DSE which could be a valuable diagnostic modality for DLVOTO.[3]
- Provoked DLVOTO based on diverse LV geometric changes as anterior mitral leaflet length and thickness, and aorto-septal angel, have been identified in earlier research,[4] therefore it appeared fair to study another parameter as basal septum contractility in the current study.
- We only used LS rather than radial and circumferential strain. It is known that The LV side of interventricular septum has sub-endocardial fibers with a dominant longitudinal motion.[32]

5. Conclusions

We demonstrate that provoked LVOT obstruction during DSE in patients without structural heart disease is directly correlated to resting regional LS, where the increased BS-LS of $\geq -17.5\%$ was a key determinant of LVOT gradient provocation. Assessment of baseline BS-LS $_{average}$ might be a bedside simple tool, equivalent to DSE for the detection of LDLVOTO, and it may be an evaluation option for symptomatic patients not able to do DSE.

Declaration of competing interest

There is no conflict of interest.

Acknowledgment

References

1. Shoji M, Uejima T, Matsuno S, et al. Dynamic narrowing of left ventricular outflow tract - possible mechanism of latent left ventricular outflow tract obstruction. *J Cardiol Cases*. 2010;2(2):74—77.
2. Luria D, Klutstein MW, Rosenmann D, Shaheen J, Sergey S, Tz- ivoni D. Prevalence and significance of left ventricular outflow gradient during dobutamine echocardiography. *Eur Heart J*. 1999;20:386—392.
3. Saeed S, Vegsundvåg J. Usefulness of stress echocardiography in assessment of dynamic left ventricular obstructions: case series and review of the literature. *Cardiology*. 2021;146:441—450.
4. Cha JJ, Chung H, Yoon YW, et al. Diverse geometric changes related to dynamic left ventricular outflow tract obstruction without overt hypertrophic cardiomyopathy. *Cardiovasc Ultrasound*. 2014;3:12—23.
5. Blessberger H, Binder T. Two dimensional speckle tracking echocardiography: clinical applications. *Heart*. 2010;96(24):2032—2040.
6. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2015;28(1):1—39.
7. Kelshiker MA, Mayet J, Unsworth B, Okonko DO. Basal septal hypertrophy. *Curr Cardiol Rev*. 2013;9(4):316—324.
8. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF 3rd, Dokainish H, Edvardsen T, Flachskampf FA, Gillebert TC, Klein AL, Lancellotti P, Marino P, Oh JK, Popescu BA, Waggoner AD Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American society of echocardiography and the European association of cardiovascular imaging. J Am Soc Echocardiogr (4):277-314..
9. Bagger T, Sloth E, Jakobsen CJ. Left ventricular longitudinal function assessed by speckle tracking ultrasound from a single apical imaging plane. *Crit Care Res Pract*. 2012, 361824, 2012.
10. Abou R, Leung M, Khidir MJH, et al. Influence of aging on level and layer-specific left ventricular longitudinal strain in subjects without structural heart disease. *Am J Cardiol*. 2017;120(11):2065—2072.
11. Badano LP, Kolias TJ, Muraru D, et al. Industry representatives; Reviewers: this document was reviewed by members of the 2016—2018 EACVI Scientific Documents Committee. Standardization of left atrial, right ventricular, and right atrial deformation imaging using two-dimensional speckle tracking echocardiography: a consensus document of the EACVI/ASE/Industry Task Force to standardize deformation imaging. *Eur Heart J Cardiovasc Imaging*. 2018;19(6):591—600.
12. Pathan F, D'Elia N, Nolan MT, Marwick TH, Negishi K. Normal ranges of left atrial strain by speckle-tracking echocardiography: a systematic review and meta-analysis. *J Am Soc Echocardiogr*. 2017;30(1):59—70.
14. Meimoun P, Benali T, Sayah S, Luycx-Bore A, Maitre B, Tribouilloy C. Significance of systolic anterior motion of the mitral valve during dobutamine stress echocardiography. *J Am Soc Echocardiogr*. 2005;(1):49—56.
15. Semba H, Sawada H, Uejima T, et al. Basic echocardiographic features of patients with latent left ventricular outflow tract obstruction without left ventricular hypertrophy. *Int Heart J*. 2012;53(4):230—233.
16. Couzin-Frankel J. Inflammation bares a dark side. *Science (New York, NY)*. 2010;330(6011):1621.
17. Manabe I. Chronic inflammation links cardiovascular, metabolic and renal diseases. *Circ J*. 2011;75:2739—2748.
18. Christiaens L, Duplantier C, Allal J, et al. Normal coronary angiogram and dobutamine-induced left ventricular obstruction during stress echocardiography: a higher hemodynamic responsiveness to dobutamine. *Echocardiography*. 2001;18(4):285—290.
19. Siddiqui TS, Dawn B, Stoddard MF. Hypotension during dobutamine stress transesophageal echocardiography: relationship with provoked left ventricular obstruction. *J Am Soc Echocardiogr*. 2006;19:1144—1149.
20. Sobczyk D. Dynamic left ventricular outflow tract obstruction: underestimated cause of hypotension and hemodynamic instability. *J Ultrason*. 2014;14(59):421—427.
21. Ranasinghe I, Ayoub C, Cheruvu C, Freedman SB, Yiannikas J. Isolated hypertrophy of the basal ventricular septum: characteristics of patients with and without outflow tract obstruction. *Int J Cardiol*. 2014;173(3):487—493.
22. Câmara EJ, Oliveira MM, Ribeiro FM. Baseline echocardiographic predictors of dynamic intraventricular obstruction of the left ventricle during dobutamine stress echocardiogram. *Echocardiography*. 2009;26(10):1195—1203.
23. Zhou D, Yang W, Yang Y, et al. Left atrial dysfunction may precede left atrial enlargement and abnormal left ventricular longitudinal function: a cardiac MR feature tracking study. *BMC Cardiovasc Disord*. 2022;22(1):99.
24. Yang L, Qiu Q, Fang SH. Evaluation of left atrial function in hypertensive patients with and without left ventricular hypertrophy using velocity vector imaging. *Int J Cardiovasc Imag*. 2014;30:1465—1471.
25. Li L, Chen X, Yin G, et al. Early detection of left atrial dysfunction assessed by CMR feature tracking in hypertensive patients. *Eur Radiol*. 2020;30:702—711.
26. Urbano-Moral JA, Rowin EJ, Maron MS, Crean A, Pandian NG. Investigation of global and regional myocardial mechanics with 3-dimensional speckle tracking echocardiography and relations to hypertrophy and fibrosis in hypertrophic cardiomyopathy. *Circ Cardiovasc Imaging*. 2014;7(1):11—19.
27. Haland TF, Almaas VM, Hasselberg NE, et al. Strain echocardiography is related to fibrosis and ventricular arrhythmias in hypertrophic cardiomyopathy. *Eur Heart J Cardiovasc Imaging*. 2016;17:613—621.
28. Tanaka H. Efficacy of echocardiography for differential diagnosis of left ventricular hypertrophy: special focus on speckle-tracking longitudinal strain. *J Echocardiogr*. 2021;19:71—79.
29. Yu JJ, Choi HS, Kim YB, et al. Analyses of left ventricular myocardial deformation by speckle-tracking imaging during the acute phase of Kawasaki disease. *Pediatr Cardiol*. 2010;31:807—812.
30. Ruppert M, Lakatos BK, Braun S, et al. Longitudinal strain reflects ventriculoarterial coupling rather than mere contractility in rat models of hemodynamic overload-induced heart failure. *J Am Soc Echocardiogr*. 2020;33(10):1264—1275.
31. Badran HM, Ibrahim WA, Faheem N, Yassin R, Alashkar T, Yacoub M. Provocation of left ventricular outflow tract obstruction using nitrate inhalation in hypertrophic cardiomyopathy: relation to electromechanical delay. *Glob Cardiol Sci Pract*. 2015;2015(15).
32. Salvo GD, Pergola V, Fadel B, Bulbul ZA, Caso P. Strain echocardiography and myocardial mechanics: from basics to clinical applications. *J Cardiovasc Echogr*. 2015;25(1):1—8.