Research Brief

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

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A B S T R A C T

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±7 years). They were compared with 90 non-LVOTO obstruction patients (43 males, 47 females; mean age 57±6 years). Based on multivariate logistic regression analysis, the independent predictors of provoked DLVOTO during DSE were resting basal septal longitudinal strain BS-LSaverage (p < 0.001), resting LA reservoir strain (p < 0.001), and systolic LVOT diameter (p = 0.03). Resting BS-LSaverage 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 −17.5% was a key determinant of LVOT gradient provocation. Assessment of baseline BS-LSaverage might be a bedside simple tool for detection of patients with DLVOTO not able to do DSE.

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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. 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/m² in women and greater than 115 g/m² in men according to the American Society of Echocardiography and European Association of Cardiovascular Imaging. 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). 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.

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.

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 (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.
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

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**Fig. 2.** Segmental Longitudinal strain at apical 3-chamber view showing high segmental strain at basal anteroseptal.

**Fig. 3.** Global and segmental left ventricular longitudinal strain assessment from the apical 4, 2, and 3-views.
resting state. All analysis was considered statistically significant at P value of <0.05.

3. Results

DSE increased LVOT gradient to $\geq 50$ mmHg in 50 Patients (36%) who are assigned as provoked DLVOTO group (15 males, 35 females; mean age 55±7years), while 90 patients had consequential 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 multivariable 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 Cutoff 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,244 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

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**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 |
| Complains, (%) | | | |
| Dyspnea | 39 (78%) | 21 (19%) | 0.003 |
| Chest pain | 16 (32%) | 20 (22%) | 0.3 |
| Pre- syncope | 5 (10%) | 5 (5%) | 0.08 |
| SBP, mmHg (x± SD) | 130 ± 11 | 133 ± 12 | 0.3 |
| HR, 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 |

**Table 2**

| Echocardiographic parameters of studied groups. | LDLVOTO (n = 50) | Non-obstruction (n = 90) | 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 |
| LV mass index, g/m² | 82 ± 13 | 90 ± 16 | 0.1 |
| LVOTD₃, 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 | 6.5 ± 1.1 | 0.7 |
| Mild MR (%) | 14 (28%) | 10 (11%) | 0.02 |
| No MR (%) | 36 (72%) | 80 (88.9%) | 0.000 |
| 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; LVOTD₃, systolic left ventricular outflow tract diameter; PG, pressure gradient; MR, mitral regurgitation; SAM, systolic anterior motion of mitral valve; EF, ejection fraction; E/E', peak early transmitral/early diastolic myocardial velocities; BS-LS, basal septum longitudinal strain; GLS, global longitudinal strain.
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, 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, Sembia, 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 load created by pressure from the right ventricle on the basal septum,32 could be the proposed mechanisms of basal septal hypertrophy.33

In HCM, myocyte disarray/hypertrophy, fibrosis, and coronary arteriolar 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 subtlety 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.

### Tables

#### 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   |

#### Table 4
**Multivariate Linear Regression Analyses of independent predictors of LDLVOTO.**

| Variable               | B Coefficient | 95% CI Lower | 95% CI 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          |
| LVOTDS                 | −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.
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 ≥ −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.

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