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The Role of False Tendons in Left Ventricular Remodeling and Secondary Mitral Regurgitation After Acute Myocardial Infarction

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ABSTRACT

BACKGROUND: Left ventricular false tendons (LVFT) are common structures visualized on transthoracic echocardiography (TTE). The present study tested the hypothesis that LVFT, via a possible 'constraint' mechanism, attenuate left ventricular (LV) remodeling and secondary mitral regurgitation after acute myocardial infarction.

METHODS: Seventy-one patients admitted to the Coronary Care Unit following an ST-elevation (n = 63) or non-ST-elevation (n = 8) myocardial infarction were analyzed; 29 (41%) had LVFT, and 42 (59%) did not (no-LVFT). All had a TTE and at least 1 follow-up study after revascularization. The χ² analysis, Student's t-test, and Mann Whitney U test were used for the statistical analyses.

RESULTS: The mean age (64 vs. 66 years), left ventricular ejection fraction (LVEF) (41% vs. 39%), left ventricular end-diastolic diameter (LVEDd) index (23 mm/m² for both), and prevalence of ≥ moderate secondary/functional mitral regurgitation (MR) (17% vs. 14%) were similar between the LVFT and no-LVFT groups. At 1-year follow-up, there was no significant difference in chamber remodeling amongst the LVFT versus no-LVFT group when assessed by: 1) ≥ 10% decrease in the relative LVEF (24% vs. 26%; p = 0.83); 2) ≥ 10% increase in the LVEDd index (41% vs. 38%, p = 0.98); and, 3) ≥ 10% increase in the LV mass index (48% vs. 41%, p = 0.68). There was no difference in the prevalence of ≥ moderate secondary/ functional MR (17% vs. 12%, p = 0.77). Outcomes remained similar when stratifying by LVFT morphology or ischemic territory.

CONCLUSIONS: In patients with mild to moderate LV dysfunction and normal chamber size, LVFT do not affect the development of LV remodeling or secondary/functional MR post-myocardial infarction.

Keywords: Left ventricular false tendons; Left ventricular remodeling; Myocardial infarction; Ischemic mitral regurgitation; Echocardiography

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INTRODUCTION

Left ventricular false tendons (LVFT) were first described in the 19th century by Sir William Turner.[1] LVFT are fibrous, fibromuscular, or muscular bands arising from and attaching to the trabeculated myocardium from the interventricular septum to the papillary muscle(s), left ventricular (LV) free wall, and/or apex.[2] Sparing of the mitral valve leaflets, as opposed to the true chordae tendinae, is an identifying characteristic. The prevalence of LVFT may be as high as 61%, however, their functional and clinical implications are not clearly defined (**Supplementary Table 1**).[2-9] Based on pathologic and echocardiographic studies, it is theorized that LVFT may attenuate LV remodeling in patients with ischemic or dilated cardiomyopathy via a restraining effect on opposing LV wall segments, and decrease the development of secondary/functional mitral regurgitation (MR) through stabilization of papillary muscle orientation.[10][11] The aim of the present study was to assess whether LVFT attenuate LV remodeling and the development of secondary/functional MR in the acute and follow-up period after myocardial infarction.

METHODS

Patient selection

The Mount Sinai Medical Center Institutional Review Board in Miami Beach, Florida, in accordance with institutional regulations and the ethical guidelines of the 1975 declaration of Helsinki, approved the study protocol. We retrospectively analyzed the Coronary Care Unit database to identify patients admitted with the diagnosis of ST-segment elevation myocardial infarction (STEMI) or non-ST-segment elevation myocardial infarction (NSTEMI) from January 2015 to June 2018. Our institutional guidelines on the clinical diagnosis of STEMI are based on the American College of Cardiology/American Heart Association recommendations on the management of STEMI and includes: 1) symptoms of myocardial ischemia; 2) electrocardiographic ST-segment elevations; and, 3) systemic elevation of cardiac biomarkers signaling myocardial necrosis.[12] In the absence of electrocardiographic ST-segment elevations, the patients were diagnosed as NSTEMI.[13]

Individuals with both a baseline transthoracic echocardiogram (TTE) and at least one post-hospital discharge follow-up examination after revascularization were included in the study. The documented history and physical examination, coronary catheterization report, and consultation and progress notes were thoroughly reviewed to document demographic data, medical history, and clinical outcomes. Of note, the medications reported were the discharge medications after the patients were treated for acute coronary syndrome.

Echocardiographic analysis

Two-dimensional (2D) TTE was performed using a General Electric ultrasound system (GE Healthcare, Chicago, IL, USA). The echocardiographic variables of interest were independently measured by 2 blinded echocardiographers (S.L.P., C.G.M.) utilizing EchoPAC software in accordance with the American Society of Echocardiography guidelines on chamber quantification, and evaluation of native valvular regurgitation.[14][15] The LV shape and mass were determined by utilizing internal chamber dimensions and wall thickness as measured with 2D and M-Mode echocardiography. Secondary/functional MR was defined as incomplete systolic mitral valve closure secondary to LV remodeling and posterolateral and/or apical papillary muscle displacement, with normal leaflet anatomy and pliability.[16][17] The
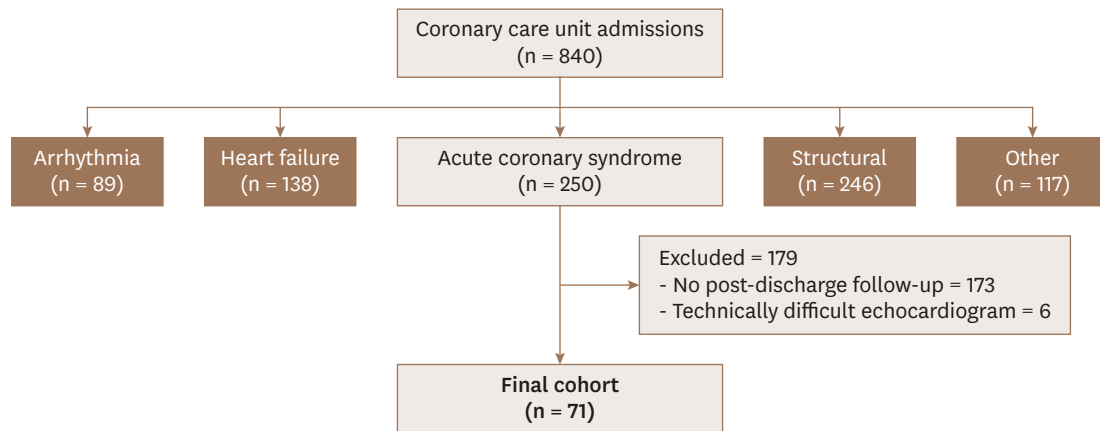
Figure 1. Flow chart depicting study patient selection.

MR severity was graded in an integrative fashion using qualitative, semi-quantitative, and quantitative methods as trace/none (0), mild (1+), moderate (2+), moderate-to-severe (3+), and severe (4+).[18]

The presence of LVFT was assessed in four commonly-acquired echocardiographic views: the 2D-TTE LV apical four and three-chamber views, and parasternal long and short axis views. An LVFT was diagnosed by visualization of a distinct linear echo traversing the LV cavity, without attaching to the mitral valve leaflets, and present in at least two echocardiographic imaging views.[19] The presence of LVFT was definitively diagnosed if the false tendon(s) was visualized in at least 2 cross-sectional imaging planes.

The study cohort was divided into 2 groups—those with LVFT and those without (no-LVFT). The patients with LVFT were further subdivided into 5 groups based on their anatomic phenotype following the classification of Luetmer and colleagues.[6] Type I attaches the posteromedial papillary muscle to the basal anteroseptum; type II the anterolateral and posteromedial papillary muscles; type III the anterolateral papillary muscle to the mid interventricular septum; type IV the inferoapex to the mid interventricular septum; and, type V the anteroapex to the inferoapical LV wall segments (**Figures 1** and **2**). After analysis of the

Parasternal long-axis view of the left ventricle

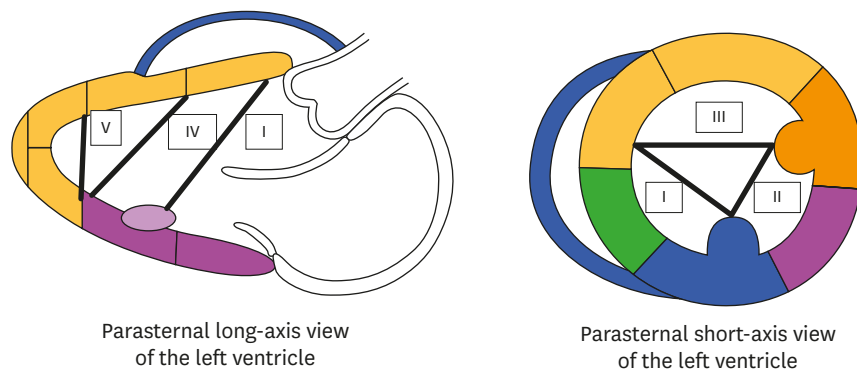Parasternal short-axis view of the left ventricle

Figure 2. Illustrative representation of left ventricular false tendon morphology in the parasternal long-axis (left) and short-axis (right) views.
Type I posteromedial papillary muscle and basal anteroseptum 7 (21%); Type II anterolateral and posteromedial papillary muscles 1 (3%); Type III anterolateral papillary muscle and basal inferoseptum 1 (3%); Type IV inferoapex and mid anteroseptum 11 (33%); Type V anteroapex and inferoapex 13 (40%).

full cohort, a sub-group analysis was performed on patients with type I, III, and IV LVFT. These LVFT all attach a papillary muscle or opposing wall segment to the interventricular septum. which provides the hypothetical restraint effect of LVFT.

Statistical methods

Categorical variables were compared utilizing a χ^2 analysis and the results were reported as frequencies and percentages. Continuous variables with a parametric distribution were analyzed with the Student's t-test, and those with a non-parametric distribution with the Mann Whitney U test. The results were reported as mean ± 1 standard deviation, or median and interquartile range (IQR, 25–75), as appropriate. A 2-tailed p-value < 0.05 was considered statistically significant. All statistical analyses were performed using Statistical Program for Social Sciences software (version 21.0; SPSS Inc., Chicago, IL, USA).

RESULTS

There was a total of 840 Coronary Care Unit admissions during the study time period, of which 250 were for acute coronary syndrome. There were 173 patients excluded due to no available post-hospital discharge follow-up echocardiogram and 6 excluded as a result of technically difficult index echocardiograms. Seventy-one patients were included in the final analysis, of which 63 (89%) had STEMI and 8 (11%) patients had NSTEMI (**Figure 1**). The infarcted myocardial territory was inferior/inferiolateral in 44 (62%), and anterior in 27 (38%). Of the 71 patients studied, 29 (41%) had LVFT. The mean age was (64 vs. 66 years; p = 0.67), and 7 (24%) and 11 (26%) were women in the 2 groups, respectively. Significantly more patients in the LVFT group were using mineralocorticoid receptor antagonists (45% vs. 14%; p = 0.01). Otherwise, the 2 groups were well matched without significant difference in terms of comorbidities and anthropometric variables (**Table 1**).

Baseline echocardiographic parameters including LV wall thickness, Doppler analysis of the mitral inflow, chamber quantification, LV mass quantification, and prevalence of significant

Table 1. Baseline demographics and co-morbidities

Variable	False tendons (n = 29)	No tendons (n = 42)	p-value
Demographics			
Female sex	7 (24.1)	11 (26.2)	1.00
Age (years)	64 ± 13	66 ± 13	0.67
Body mass index (kg/m^2)	27 ± 3	29 ± 5	0.12
STEMI	27 (93.1)	36 (85.7)	0.56
Co-morbidities			
Atrial fibrillation	6 (20.7)	4 (9.5)	0.33
Hypertension	23 (79.3)	35 (83.3)	0.90
Tobacco abuse	21 (72.4)	27 (64.3)	0.64
Diabetes mellitus	14 (48.3)	12 (28.6)	0.15
Prior diagnosis of coronary artery disease	13 (44.8)	16 (38.1)	0.75
Peripheral artery disease	4 (13.8)	4 (9.5)	0.86
Medical therapy			
Antiplatelet agent	29 (100.0)	41 (97.6)	1.00
Beta-blockers	28 (96.6)	34 (81.0)	0.11
ACEi/ARB	24 (82.8)	33 (78.6)	0.90
Mineralocorticoid receptor antagonist	13 (44.8)	6 (14.3)	0.01

Variables presented as total number (percentage) or mean ± standard deviation.
ACEi: angiotensin-converting enzyme, ARB: angiotensin receptor blocker, STEMI: ST-segment elevation myocardial infarction

Table 2. Baseline echocardiographic assessment of LV geometry and function after acute myocardial infarction

Variable	False tendons (n = 29)	No tendons (n = 42)	p-value
LVEF (%)	41 ± 14	39 ± 15	0.45
Interventricular septal thickness (mm)	14 (11–17)	13 (11–15.3)	0.45
Posterior wall thickness (mm)	12 (11–14)	13 (12–14)	0.14
Left atrial anteroposterior diameter (mm)	35 (32–38)	35 (31–40)	0.88
Left atrial anteroposterior diameter index (cm/m²)	18 (16–20)	18 (16–20)	1.00
LVEDd (mm)	42 (39–46)	46 (42–48)	0.05
LVEDd index (mm/m²)	23 (20–24)	23 (20–25)	0.94
LVESd (mm)	31 (28–37)	34 (28–39)	0.49
LVESd index (mm/m²)	17 (14–19)	17 (14–19)	0.93
Relative wall thickness	0.57 (0.5–0.71)	0.58 (0.51–0.68)	0.80
LV mass (g)	189 (170–242)	218 (173–278)	0.25
LV mass index (g/m²)	105 (88–128)	109 (99–138)	0.37
Moderate or greater MR (≥ 2+) (%)	5 (17.2)	6 (14.3)	1.00
Mitral inflow E-wave peak velocity (m/s)	0.8 (0.67–0.93)	0.77 (0.61–1)	0.94
Mitral inflow A-wave peak velocity (m/s)*	0.83 (0.62–1)	0.8 (0.52–0.94)	0.43
Tissue Doppler lateral mitral annulus e′ peak velocity (m/s)†	0.06 (0.06–0.09)	0.09 (0.06–0.1)	0.24
Tissue Doppler septal mitral annulus e′ peak velocity (m/s)‡	0.06 (0.05–0.08)	0.05 (0.04–0.08)	0.83

Variables presented as total number (percentage), mean ± standard deviation, or median (interquartile range).
LV: left ventricular, LVEDd: left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction, LVESd: left ventricular end-systolic diameter, MR: mitral regurgitation.
Analysis performed in: *27 vs. 41; †28 vs. 39 patients; ‡27 vs. 39.

Table 3. Incidence of false tendon based on morphology

Morphology of left ventricular false tendons	No. (%)
1	7 (21.2)
2	1 (3.0)
3	1 (3.0)
4	11 (33.3)
5	13 (39.4)
Total*	33 (100.0)

*Four patients had more than 1 tendon.

MR are summarized in **Table 2**. Left ventricular ejection fraction (LVEF) (41% vs. 39%; p = 0.45) was similar between the LVFT and no-LVFT groups. At baseline, the left ventricular end-diastolic diameter (LVEDd) was significantly larger in patients with no-LVFT; however, there was no difference when the values were indexed to body surface area (23 mm/m² for both groups). The most common morphologic types of LVFT were type IV and V, accounting for 33% and 40%, respectively (**Table 3; Figure 3**).

The time interval between echocardiophic studies was 1.1 ± 0.9 years in the LVFT group and 0.9 ± 0.7 years in the no-LVFT group (p = 0.3) (**Table 4**). At follow-up, there was an absolute decrease in the indexed LVEDd and left ventricular end-systolic diameter (LVESd) in both groups, with no statistically demonstrable difference between them (LVEDd index, –1.5 vs. –1 mm/m²; p = 0.65) (**Table 5**). There was no significant difference in adverse chamber remodeling when assessed by: ≥ 10% decrease in the relative LVEF (17% for both; p = 1.0); ≥ 10% increase in the LV mass index (45% vs. 36%; p = 0.36); and, type of LV geometry (concentric remodeling most common: 38% vs. 43%; p = 0.87) between the LVFT and no-LVFT groups. There was no significant difference in the prevalence of moderate or greater secondary/functional MR at follow-up (17% vs. 12%; p = 0.77). Finally, there were no differences in echocardiographic parameters when patients were stratified according to LV false tendon morphology or ischemic territory affected, except for individuals with anterior wall myocardial infarction and LVFT who had a significant higher relative wall thickness (0.58 vs. 0.5, p = 0.04) and thicker posterior walls (14 vs. 11 mm) (**Supplementary Tables 2-7**).
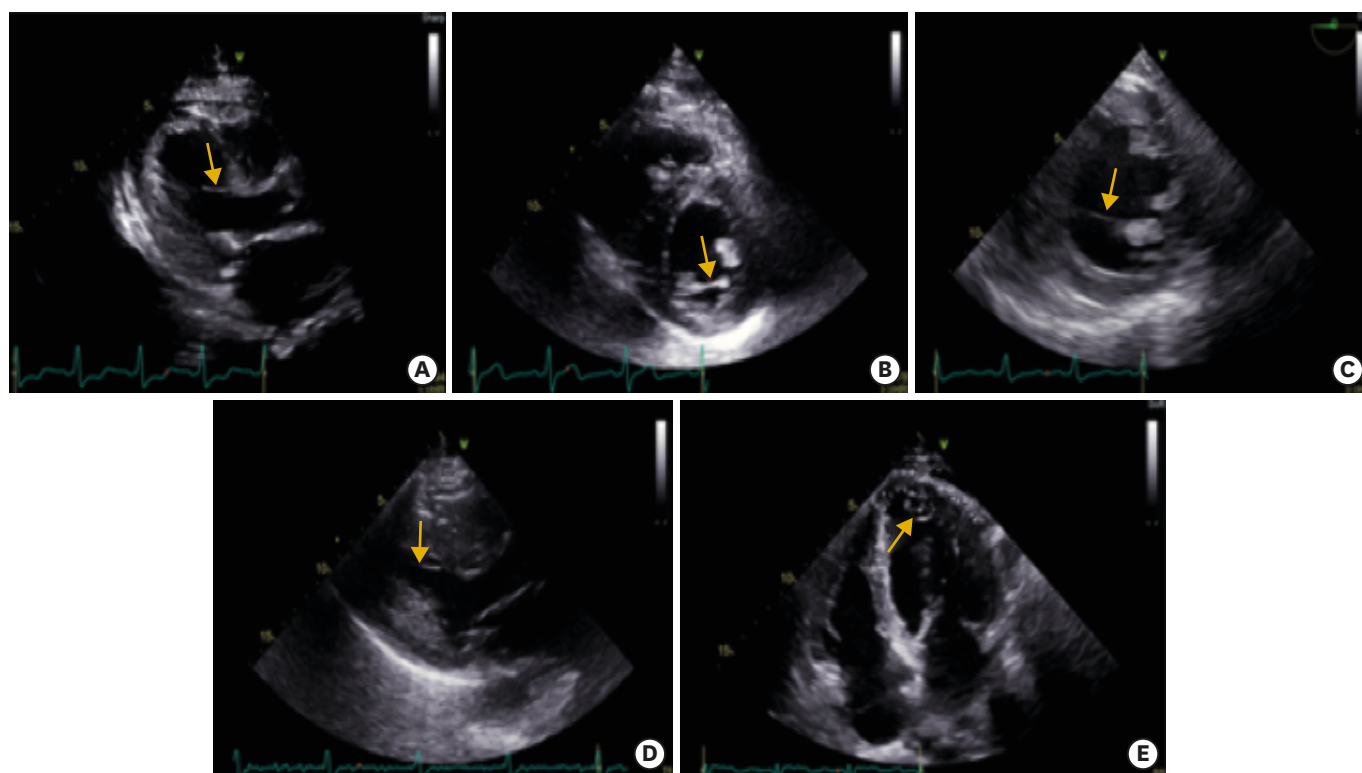
Figure 3. Two-dimensional echocardiographic examples of left ventricular false tendons by specific morphology. (A) Type I posteromedial papillary muscle and basal anteroseptum 7 (21%); (B) Type II anterolateral and posteromedial papillary muscles 1 (3%); (C) Type III anterolateral papillary muscle and basal inferoseptum 1 (3%); (D) Type IV inferoapex and mid anteroseptum 11 (33%); and (E) Type V anteroapex and inferoapex 13 (40%).
Note: 'C' corresponds to a transesophageal transgastric short-axis view of the left ventricle at the level of the papillary muscle, with the anterolateral papillary muscle located at the 5 o'clock position.

Table 4. Follow-up echocardiographic assessment of LV geometry and function

Variable	False tendons (n = 29)	No tendons (n = 42)	p-value
Time to follow-up transthoracic echocardiogram (years)	1.1 ± 0.9	0.9 ± 0.7	0.30
LVEF (%)	43 ± 14	44 ± 15	0.73
Interventricular septal thickness (mm)	12 (11–14)	13 (11–15)	0.32
Posterior wall thickness (mm)	13 (11–15)	13 (11–13)	0.44
Left atrial anteroposterior diameter (mm)	36 (32–41)	38 (34–41)	0.33
Left atrial anteroposterior diameter index (mm/m²)	19 (17–21)	20 (17–22)	0.53
LVEDd (mm)	46 (41–51)	46 (42–50)	0.81
LVEDd index (mm/m²)	24 (21–27)	24 (22–28)	0.91
LVESd (mm)	35 (30–40)	36 (33–40)	0.35
LVESd index (mm/m²)	19 (16–21)	19 (17–22)	0.95
Relative wall thickness	0.56 (0.45–0.73)	0.53 (0.48–0.61)	0.61
LV mass (g)	207 (162–269)	210 (185–279)	0.77
LV mass index (g/m²)	115 (89–131)	108 (87–148)	0.94
Moderate or greater MR (≥ 2+) (%)	5 (17.2)	5 (11.9)	0.77
Mitral inflow E-wave peak velocity (m/s)*	0.71 (0.55–1)	0.7 (0.56–1.1)	0.99
Mitral inflow A-wave peak velocity (m/s)†	0.8 (0.63–0.94)	0.67 (0.52–0.9)	0.18
Tissue Doppler lateral mitral annulus e′ peak velocity (m/s)‡	0.07 (0.06–0.1)	0.08 (0.05–0.1)	0.92
Tissue Doppler septal mitral annulus e′ peak velocity (m/s)§	0.05 (0.04–0.07)	0.05 (0.04–0.07)	0.52

Variables presented as total number (percentage), mean ± standard deviation, or median (interquartile range).
LV: left ventricular, LVEDd: left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction, LVESd: left ventricular end-systolic diameter, MR: mitral regurgitation.
Analysis performed in: *28 vs. 41; †27 vs. 37; ‡27 vs. 41; §26 vs. 39 patients, respectively.

Table 5. Follow-up echocardiographic assessment of LV remodeling

Variable	False tendons (n = 29)	No false tendons (n = 42)	p-value
> 10% decrease in absolute LVEF (%)	5 (17.2)	7 (16.7)	1.00
> 10% increase in LVEDd (%)	10 (34.5)	15 (35.7)	1.00
> 10% increase in LVEDd index (%)	12 (41.4)	16 (38.1)	1.00
> 10% increase in LVESd (%)	14 (48.3)	19 (45.2)	0.90
> 10% increase in LVESd index (%)	14 (48.3)	22 (52.4)	0.92
> 10% increase in LV mass	13 (44.8)	15 (35.7)	0.60
LV geometry			
Normal geometry (%)	4 (13.8)	2 (4.8)	0.36
Concentric remodeling (%)	11 (37.9)	18 (42.9)	0.87
Concentric hypertrophy (%)	13 (44.8)	20 (47.6)	1.00
Eccentric hypertrophy (%)	1 (3.4)	2 (4.8)	1.00

Variables presented as total number (percentage) or median (interquartile range).
LV: left ventricular, LVEDd: left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction, LVESd: left ventricular end-systolic diameter.

DISCUSSION

The main findings of the present study of LVFT in patients with mild to moderately impaired LV systolic function and normal LV size at 1-year post-myocardial infarction are: 1) LVFT appear to be anatomically benign structures that lack their theorized benefit in terms of LV remodeling or the development of secondary/functional mitral regurgitation; 2) type IV (interventricular septum-inferoapex) and type V (anteroapex-inferoapex) are the most common phenotypes of LVFT; 3) the most prevalent LV geometry pattern was concentric remodeling in a largely hypertensive and diabetic cohort; and, 4) there was no difference in outcomes when assessing LVFT effects as stratified by the presence of 'restraining' mid-cavity LVFT or the infarcted myocardial territory.

Due to lack of a standardized classification system for LVFT, head to head comparisons of the results previously published and the most common anatomic location of these structures remains a topic of debate. Malouf and colleagues[7] studied 488 patients referred for echocardiography and found that prevalence of LVFT was 25%, and 96% of patients with LVFT were described to have tendons attaching the interventricular septum to the free wall. These phenotypes are similar to the morphologic description of type IV and V LVFT established by Luetmer et al.,[6] and that represented 73% of the LVFT in the present cohort (**Supplementary Table 1**, **Table 3**). Conversely, other echocardiographic and autopsy studies cite type I LVFT, which attach the posteromedial papillary muscle to the basal anteroseptum, as the most prevalent.[3,4,6,8]

Our results contrast to those published by Bhatt et al.,[10] who studied the impact of LVFT in a cohort of 203 patients with mixed cardiomyopathies (ischemic and non-ischemic) and severe LV dysfunction (LVEF = 20%; LVEDd = 61 mm). The authors reported a decreased incidence of severe secondary/functional MR in patients with LVFT when compared with no-LVFT (4.9% vs. 27%; p < 0.001). Interestingly, individuals with LVFT had less deformed mitral valves with a significantly smaller mitral annular diameter (3.6 vs. 4.0 cm; p < 0.001), shorter mitral valve coaptation depth (0.9 vs. 1.2 cm; p < 0.001) and reduced coaptation areas (1.6 vs. 2.5 cm^2; p < 0.001), with a similar LVEDd (6.0 vs. 6.2 cm; p = 0.22). The attenuation of the progression to at least moderate MR was limited to patients with mid-cavity transverse false tendons (p < 0.001) and not seen in those with apical LVFT (p = 0.27).

We did not observe this protective mechanism, and the prevalence of moderate or greater secondary MR was similar between both groups (17% vs. 12%; p = 0.77). It is important to note that our cohort consisted exclusively of subjects with ischemic cardiomyopathy and mild to moderate LV systolic dysfunction, in whom the LV chamber was not dilated. Perhaps, the role of LVFT becomes relevant in dilated ventricles when the chamber is subjected to higher wall stress, as in the patients studied by Bhatt and colleagues.[10] This phenomenon was also reported by Malouf et al.,[7] who demonstrated a higher prevalence of LVFT when only those patients with dilated ventricles were analyzed (57% vs. 25% in the entire cohort) and proposed that in dilated chambers, LVFT are stretched out, becoming better delineated from the myocardium and more accessible to the ultrasound beam.[6] Another important point to contrast is that in our cohort, the most predominant subtypes of LVFT were types IV and V (apical attachment). However, subgroup analysis according to the anatomical location showed no difference when "mid-cavitary" tendons (those with attachments to the interventricular septum and corresponding to type I, III, and IV) were compared with the rest of the population. The ischemic territory involved was also analyzed and no differences were observed (**Supplementary Tables 2**-**7**).

Another theorized mechanism for the attenuation of secondary/functional MR has been attributed to potential intraventricular resynchronization via the electrical conductive properties of LVFT.[9] Cardiac resynchronization, in iatrogenic cases performed with biventricular pacemaker therapy, is an important treatment for secondary/functional MR in patients with left bundle branch block.[20,21] In our study only 5 patients had either right or left bundle branch block and all corresponded to the control group, which prohibited any meaningful analysis. Furthermore, the potential impact of LVFT on clinical endpoints is unknown, as this was not the aim of our study; further research is needed on this subject. This gap in knowledge comes from studies of animal models that suggest LVFT are a source of atrial natriuretic peptide, which may prevent delayed LV remodeling by decreasing the wall stress tension through their negative feedback on endothelin-1, angiotensin II, and aldosterone.[9,22-24]

There are caveats and limitations to present study that must be considered. Firstly, it is retrospective in nature and the sample size is small, which confers an inherent selection bias and limited statistical power. Analyses to assess for inter- and intra-observer variability in diagnosis were not possible, which may impact the true prevalence of LVFT. Additionally, there was a large number of patients excluded who followed-up outside of our institution and had no available post-discharge echocardiogram for review; this further limits the strength of the study and represents a separate selection bias. Secondly, we did not analyze echocardiographic variables of mitral valve apparatus remodeling such as coaptation area and depth, which are prognostic in regards to development of secondary/functional MR.[25] Nonetheless, progression of at least moderate MR was similar between the LVFT and no-LVFT groups. Thirdly, it is important to note that MR was graded in an integrative manner, carefully interpreting all 2D and Doppler data that could be accurately assessed. However, blood pressure and heart rate measurements were not available; these hemodynamic variables are strong mediators of MR severity and may result in over- or underestimation. Fourthly, patients with LVFT had a smaller LVEDd and greater use of mineral corticoid receptor antagonists at baseline. We acknowledge that this baseline heterogeneity is a confounder. However, when indexed to body surface area there was no difference in LV size between group, and a lack of benefit seems to reinforce the benign nature of LVFT despite a hypothetical advantage with greater neurohormonal blockade. Fifthly, nearly 75% of patients

were male and 89% presented with STEMI; thus, the data may not be applicable to female or NSTEMI populations. Sixthly, data regarding medical therapy during the follow-up period, including regimen and dosage, was not available. The current findings are best interpreted as hypothesis-generating and complementary to the literature regarding LVFT in post-myocardial infarction patients with mild to moderate LV systolic dysfunction and normal chamber size.

In conclusion, LVFT are prevalent in patients presenting with acute myocardial infarction. In a largely male population presenting with STEMI, mild to moderate LV systolic dysfunction, and normal LV chamber size, the presence of LVFT did not demonstrate a benefit as assessed by echocardiographic parameters of LV remodeling or MR progression at 1-year follow-up.

SUPPLEMENTARY MATERIALS

Supplementary Table 1
Prevalence of left ventricle false tendons

Click here to view

Supplementary Table 2
Follow-up echocardiographic assessment of LV geometry and function after anterior wall myocardial infarction

Click here to view

Supplementary Table 3
Follow-up echocardiographic assessment of LV remodeling after anterior wall myocardial infarction

Click here to view

Supplementary Table 4
Follow-up echocardiographic assessment of LV geometry and function after inferior and/or inferolateral wall myocardial infarction

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Supplementary Table 5
Follow-up echocardiographic assessment of LV remodeling after inferior and/or inferolateral wall myocardial infarction

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Supplementary Table 6
Follow-up echocardiographic assessment of LV geometry and function after myocardial infarction in patient with mid-cavitary LVFT

Click here to view

Supplementary Table 7

Follow-up echocardiographic assessment of LV remodeling after myocardial infarction in patient with mid-cavitary LVFT

Click here to view

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