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Original Article

Assessment of left atrial function in dilated cardiomyopathy patients using speckle-tracking echocardiography

Taher Said Abdelkarim[1], Mohamed Abd El-Hafez Fouly[2], Ashraf Zahrah[3]

[1] Islamic Cardiology Center, Al-Azhar University, Cairo, Egypt
[2] Department of Cardiothoracic Surgery, El-Kasr Al-Ainy Hospital, Cairo University, Cairo, Egypt
[3] Department of Cardiothoracic Surgery, Shebin El-Kom Teaching Hospital, Menoufia, Egypt

Abstract

Background: The available methods to assess left atrial function (LAF) have some limitations as angle dependence and opposite distortion. The objective of the current study was to evaluate LAF in dilated cardiomyopathy (DCM) of ischemic (IDCM) and non-ischemic etiologies (NIDCM) using speckle tracking echocardiography (STE).

Methods: 52 patients with systolic heart failure were included in our study; 27 with IDCM and 25 with NIDCM along with 15 healthy controls. All patients underwent conventional echocardiography, tissue doppler imaging, and speckle tracking echocardiography. The later modality was used to compare left atrial function in IDCM and NIDCM groups.

Results: We found the left atrial maximum volume and the left atrial total emptying volume to be higher in patients with dilated cardiomyopathy compared to healthy patients (52.19 ± 6.01 vs. 21.87 ± 1.69 cm3/m2; p <0.001 and 28.67 ± 4.34 vs. 15.67 ± 2.02 cm3/m2, respectively). Conversely, left atrial emptying index and left atrial active ejection fraction were lower in patients with DCM compared to healthy controls (9.60 ± 2.29 vs. 8.27 ± 3.01 cm3/m2; p< 0.001 and 23 ± 2.56 vs. 37.47 ± 3.54 %; p<0.001, respectively). When comparing the IDCM group with NIDCM patients, we found no significant difference in left atrial maximum volume and left atrial active emptying volume. However, the NIDCM patients had significantly lower left atrial total emptying volume, and left atrial active ejection fraction (8.93 ± 1.86 vs. 9.60 ± 2.29 cm3/m2 and 23 ± 2.56 vs. 31.19 ± 1.66 %; p<0.001). on comparing strain function, DCM patients had lower systolic (28.22 ± 3.84 vs. 60.87 ± 3.07 %, p<0.001), and left atrial systolic strain rate (-2.66 ± 0.45 vs. -3.81 ± 0.35; p = 0.003) compared to healthy controls. All strains and strain rates were significantly lower in NIDCM patients compared to IDCM patients.

Conclusion: STE is a promising method for evaluating LAF in DCM patients. Patients with DCM had significantly lower left atrial systolic and late diastolic strains and strain rates compared to healthy patients. Moreover, NIDCM could be differentiated from IDCM by having more impairment in the LA dynamic reservoir and booster pump function.

KEYWORDS
Atrial function; Dilated cardiomyopathy; Doppler tissue imaging; Speckle-tracking imaging

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Corresponding author: Ashraf Zahrah zharha2001@hotmail.com

Introduction

Dilated Cardiomyopathy (DCM) is a progressive cardiac muscle disease characterized by enlargement of the ventricular chambers with contractile and filling dysfunction [1]. The major cause of DCM is ischemic heart disease; more than 60% of DCM cases are due to ischemia, which is responsible for ventricular dilatation [2]. On the other hand, non-ischemic DCM (NIDCM) usually develops with different diseases, such as Takotsubo cardiomyopathy, familial NIDCM, and myocarditis [3-5]. Left ventricular (LV) diastolic dysfunction (as in DCM) interrupts the left atrium (LA) reservoir function as it increases the LV filling pressure (LVFP), which increases LA pressure, causing chamber dilatation [6-8]. Subsequently, LA remodeling occurs, which interferes with the microcirculation of the atrium and subsequent chronic ischemia, fibrosis, and left atrial dysfunction [9].

The LA volumetric parameters and myocardial deformation by tissue Doppler imaging (TDI) have been used to describe the effect of LVFP on the LA. Previous authors have shown by standard echocardiography that the LA bump booster function is more impaired in NIDCM compared to IDCM [10, 11]. However, TDI faced some critique because its measurements are angle-dependent due to Doppler effects and opposite distortion in the long and short axes [12, 13]. Recently, 2D speckle-tracking echocardiography (2D-STE) has shown promise as a new echocardiographic approach that analyzes motion based on tracking speckle patterns on B-mode images of the US beams in the myocardium [14].

In contrast to TDI, 2D-STE has the ability to measure myocardial strain independent of the angle of insonation during each cardiac cycle and also quantify LV function [15, 16]. Although the 2D-STE technique was introduced for the exclusive analysis of LV, it has recently been developed to qualify longitudinal myocardial LA deformation dynamics [17]. In this study, we aimed to assess LA function in DCM patients of ischemic and non-ischemic etiologies using STE.

Patients and Methods:

Study Design:

The study included 67 randomly selected individuals who presented first to the Cardiac Surgery Clinic and were deemed not demanding surgery, then referred to Al-Azhar Islamic Center of Cardiology, Egypt, between January 2017 and October 2017. We included 52 patients with systolic HF, EF less than 50%, and the New York Heart Association (NHYA) ranging from class II to IV (patient group) and 15 healthy individuals (control group). Our patients included 27 patients with IDCM (IDCM group) and 25 patients with NIDCM. We obtained informed consent from all patients, and the study had the approval of Al-Azhar University Institutional Research Board.

Inclusion and Exclusion criteria:

We included patients with systolic HF (NYHA class II to IV) because of IDCM or INIDCM. We excluded patients with any rhythm abnormality, significant valvular lesions, congenital heart disease, recent acute coronary syndrome, poor echogenic window, and renal or liver cell failure. IDCM patients had an old myocardial infarction incident. NIDCM patients met 1996 WHO diagnostic criteria.

Echocardiography Study:

Transthoracic echocardiography was performed using a GE Vivid E9 ultrasonographic unit and an M5S probe (frequency 1.7–3.3 MHz). ECG was simultaneously recorded. Then, we employed the following modes to capture echocardiographic data:

M-Mode: Left atrial diameter (LAD), left ventricular end-diastolic diameter (LVEDd), interventricular septal wall thickness (IVSd), and LV posterior wall (LVPW), and left fractional ventricular shortening (FS) and ejection fraction (EF).

Doppler study: Pulsed wave doppler echocardiography was performed on the mitral valve. We measured the trans-mitral doppler flow velocity and recorded peak early filling velocity (E), peak atrial velocity (A), and the deceleration time (DT) of the E wave. We then calculated the E/A

Table 1: Clinical and basic echocardiographic data in the studied groups. A value of p < 0.05 was considered statistically significant.

Parameters	Control	NIDCM	IDCM	P
Age years (Mean ± SD)	52.87 ± 2.99	51.36 ± 9.45	56.81 ± 3.77	0.064
Sex Male	10 (66.6)	15 (60)	19 (70.4)	0.740
HR b/m (Mean ± SD)	68.87 ± 4.27	72.16 ± 10.32	76.52 ± 5.48	0.007
Current smoking	5 (33.3)	10 (40)	10 (37.03)	0.918
DM	5 (33.3)	12 (48)	16 (59.3)	0.279
Systolic BP mmHg	128 ± 18.3	120.2 ± 17.47	120.37 ± 20.6	0.388
Diastolic BP mmHg	75 ± 11.8	72.2 ± 11.82	72.78 ± 9.93	0.732
Dyslipidemia	7 (46.7)	11 (44)	19 (70.4)	0.125
E (cm/s)	69.73 ± 6.71	63.96 ± 8.75	64.81 ± 9.44	0.111
A (cm/s)	45.53 ± 8.63	35.00 ± 6.25	32.63 ± 3.86	<0.001
E/A	1.55 ± 0.16	1.844 ± 0.37	1.98 ± 0.24	<0.001
DT (ms)	188.33 ± 6.94	160.96 ± 17.04	155.59 ± 15.32	<0.001
Septal e´ (cm/s)	7.84 ± 0.54	4.41 ± 0.67	4.34 ± 0.73	<0.001
E/e´	8.73 ± 0.80	14.68 ± 3.39	15.11 ± 3.21	<0.001
LAD (cm)	3.43 ± 0.24	4.67 ± 0.50	4.77 ± 0.46	<0.001
LVEDd (cm)	4.55 ± 0.18	6.276 ± 0.55	6.28 ± 0.62	<0.001
FS (%)	30.47 ± 1.81	16.44 ± 2.16	14.48 ± 1.86	<0.001
LVEF (%)	60.87 ± 3.07	32.60 ± 4.48	28.22 ± 3.84	<0.001
LAVmax (cm3/m2)	21.87 ± 1.69	48.84 ± 8.24	52.19 ± 6.01	<0.001
LATV (cm3/m2)	15.67 ± 2.02	17.32 ± 4.785	28.67 ± 4.34	<0.001
LAEV (cm3/m2)	8.27 ± 3.01	9.60 ± 2.29	8.93 ± 1.86	0.210
AEI (%)	94.80 ± 10.93	45.28 ± 9.11	54.93 ± 7.97	<0.001
LAAEF (%)	37.47 ± 3.54	23 ± 2.56	31.19 ± 1.66	<0.001

NIDCM: idiopathic dilated cardiomyopathy; IDCM: ischemic dilated cardiomyopathy; HR: heart rate; DM: diabetes mellitus; BP: blood pressure; E: blood flow velocity of mitral valve at early diastolic; A: blood flow velocity of mitral valve at late diastole; DT,: deceleration time of E peak; e': velocity of the initial wave of the septal mitral annulus; LAD: left atrial anteroposterior diameter; LVEDD: left ventricular end-diastolic diameter; FS: fractional shortening; LVEF: left ventricular ejection fraction; LAVmax: left atrial maximum volume; LATV: left atrial total emptying volume; LAEV: left atrial active emptying volume; AEI: left atrial expansion index; LAAEF: left atrial active ejection fraction

ratio. We used two-dimensional echocardiography to measure LA volumes at the end of systole (Max AV), diastole (Min AV), and preceding atrial contraction (VPre-A).

Tissue Doppler imaging: We measured the velocity of the septal mitral annulus at early diastole (e'), and the E/e' ratio to estimate the filling pressures of the LV.

2D speckle tracking echocardiography: We measured the LA strain (Ss), systolic strain rate (SRs), and late diastolic strain (Sa). For the left ventricle, we calculated the average of the

longitudinal systolic strain of all segments to obtain a 2D global longitudinal strain (GLS) value.

Statistical Analysis

We performed statistical analysis using the SPSS software (version 22 for Windows, IBM Inc, Armonk, NY). We employed the ANOVA test to analyze the difference between the three included groups. To compare two groups (for example, DCM vs. control or IDCM vs. NIDCM), we used the t-test. We used the Spearman correlation analysis to assess the correlation between atrial function and strain parameters. A p-value of < 0.05 was considered statistically significant.

Results

Baseline Data: We recorded no significant differences between the DCM and control groups in terms of age ($p = 0.06$), gender ($p = 0.47$), frequency of smoking ($p = 0.9$), diabetes mellitus ($p = 0.27$), dyslipidemia ($p = 0.12$) and systolic/diastolic blood pressure levels ($p = 0.38$ and 0.73) (Table 1).

Basic echocardiographic parameters: The A peak, DT, septal e', FS% and LVEF% were lower in the NIDCM and IDCM groups than in the control group, while the E/A, E/e' and LAD, and LVEDd were higher in the NIDCM and IDCM groups than in the control group ($p < 0.001$). However, there were no significant differences between the NIDCM and IDCM groups in the A peak, DT, septal e', E/A, E/e' and LAD, LVEDd. However, the FS% and LVEF % were lower in the IDCM than the NIDCM group ($p < 0.001$) (Table 1).

Left atrial volume and function indices: The left atrial maximum volume (LAV max) and the left atrial total emptying volume (LATV) was higher in patients with DCM compared to healthy controls ($p < 0.001$). Conversely, left atrial emptying index (AEI) and left atrial active ejection fraction (LAAEF) were lower in patients with DCM compared to healthy controls ($p < 0.001$). No significant difference in LAVmax ($p = 0.07$) and left atrial active emptying volume (LAEV; $p = 0.09$) was found between the IDCM and NIDCM patients. However, the NIDCM patients had significantly lower left atrial emptying index (AEI), left atrial total emptying volume (LATV), and left atrial active ejection fraction (LAAEF) ($p<0.01$) (Table 1).

Left atrial strain and strain rate: DCM patients had lower systolic (LASs), late diastolic (Sa) ($p <0.001$), and left atrial systolic strain rate (SRs) ($p = 0.003$) compared to healthy patients. All strains and strain rates were significantly lower in NIDCM patients compared to IDCM patients ($p<0.05$) (Table 2).

Correlation between LA strain and function: We recorded significant and strong correlations ($p < 0.001$) between all tested LAF parameters (LAVmax, LATV, AEI, and LAAEF) and all tested atrial strain rates (average LA Ss, average LA SRs, Average LA Sa, Average LA SRa). Except for average LA Sra that had negative correlations with all atrial function parameters, other strain rates had all positive correlations with LA function parameters (Table 3).

Discussion

In this study, we found that: all strain parameters were lower in DCM patients compared to healthy controls, all strain parameters were significantly lower in NIDCM patients compared to IDCM patients, and all strain parameters were positively correlated with left atrial emptying function and left atrial active ejection fraction.

Comparison of LA strains and strain rates in NIDCM patients and controls: Strain and strain rate imaging have been introduced recently as an important method for LA function evaluation. The strain is a term used to describe the degree of shortening, thickening, and lengthening of contracting myocardial wall. Strain rate is defined as the strain per unit time which reflects real-time myocardial movement [18]. In the present study, strain and strain rate were significantly lower in DCM patients compared to healthy controls.

In a previous study, Inaba and coworkers showed that SRs, SRe, and SRa could reflect the LA reservoir, conduit, and booster pump function, respectively [19]. Our analysis showed lower Ss, Sa, SRs, and Sra in NIDCM and IDCM patients than the healthy controls indicating that LA dynamic

Table 2: Left Atrial (Strain and Strain Rates) and Left ventricular (longitudinal strain and strain rates) in the three studied groups. A value of p < 0.05 was considered statistically significant.

Parameters	Control	IDCM	NIDCM	P
LA septal wall Ss (%)	59.07 ± 5.84	38.19 ± 3.08	27.96 ± 5.17	<0.001
LA Lateral wall Ss (%)	64.67 ± 5.78	40 ± 3.16	28.76 ± 6.89	<0.001
LA Anterior wall Ss (%)	51.20 ± 5.71	33.19 ± 3.18	22.96 ± 5.94	<0.001
LA Inferior wall Ss (%)	48.73 ± 5.92	32.96 ± 3.16	21.48 ± 5.19	<0.001
Average LA Ss (%)	56.03 ± 9.43	35.59 ± 7.02	27.83 ± 5.95	<0.001
LA Septal wall SRs (s^{-l})	5.26 ± 0.35	3.33 ± 0.30	3.26 ± 0.43	0.534
LA Lateral wall SRs (s^{-l})	6.13 ± 0.36	4.18 ± 0.30	4.06 ± 0.51	0.322
LA Anterior wall SRs (s^{-l})	3.59 ±0.36	3.11 ± 0.30	1.72 ± 0.33	<0.001
LA Inferior wall SRs (s^{-l})	3.03 ± 0.41	2.60 ± 0.30	1.70 ± 0.21	<0.001
Average LA SRs (s^{-l})	4.48 ± 0.97	3.31 ± 0.73	2.77 ± 0.48	0.003
LA Septal wall Sa (%)	25.60 ± 2.746	16.37 ± 2.43	12.84 ± 4.11	<0.001
LA Lateral wall Sa (%)	28.40 ± 2.74	20.11 ± 2.44	14.96 ± 4.31	<0.001
LA Anterior wall Sa (%)	22.80 ± 2.65	15.11 ± 2.62	10.04 ± 4.42	<0.001
LA Inferior wall Sa (%)	21.20 ± 2.65	11.48 ± 2.36	8.48 ± 3.32	<0.001
Average LA Sa (%)	24.57 ± 3.24	15.95 ± 4.12	11.67 ± 2.75	<0.001
LA Septal wall SRa (s^{-l})	-4.62 ± 0.27	-3.28 ± 0.16	-2.77 ± 4.45	<0.001
LA Lateral wall SRa (s^{-l})	-5.21 ± 0.27	-4.23 ± 0.16	-3.36 ±.46	<0.001
LA Anterior wall SRa (s^{-l})	-3.57 ± 0.27	-1.49 ± 0.69	-1.77 ± 0.45	0.095
LA Inferior wall SRa (s^{-l})	-1.85 ± 0.28	-1.41 ± 0.64	-1.61 ± 0.46	0.2
Average LA SRa (s^{-l})	-3.81 ± 0.35	-2.66 ± 0.45	-2.30 ± 0.57	0.016
Basal ANT wall LS of LV (%)	-21.73 ± 1.486	-13.96 ± 1.85	-9.44 ± 1.58	<0.001
Basal Anteroseptal wall LS of LV (%)	-18.73 ± 1.03	-13.67 ± 2.93	-8.56 ± 1.58	<0.001
Basal Septal wall LS of LV (%)	-19.00 ± 0.84	-13.96 ± 1.85	-9 ± 1.44	<0.001
Basal INF wall LS of LV (%)	-22.80 ± 0.94	-14.81 ± 8.46	-12.52 ± 1.63	0.189
Basal POST wall LS of LV (%)	-21.20 ± 0.94	-14.74 ± 4.18	-10.40 ± 1.53	<0.001
Basal LAT wall LS of LV (%)	-22.07 ± 1.39	-12.37 ± 1.94	-10 ± 1.44	<0.001
MID ANT wall LS of LV (%)	-22.60 ± 0.98	-14.70 ± 4.62	-11.12 ± 1.48	<0.001
MID Anteroseptal wall LS of LV (%)	-21.20 ± 0.94	-13.67 ± 2.93	-11.20 ± 1.50	<0.001
MID Septal wall LS of LV (%)	-21.40 ± 1.24	-14.74 ± 2.74	-10.60 ± 1.53	<0.001
MID INF wall LS of LV (%)	-20.20 ± 11.96	-13.74 ± 6.12	-11.52 ± 1.53	0.084
MID POST wall LS of LV (%)	-20.60 ± 0.98	-12.22 ± 3.70	-8.20 ± 1.50	<0.001
MID LAT wall LS of LV (%)	-21.20 ± 0.94	-11.96 ± 2.75	-10.24 ± 1.45	0.007
Apical ANT wall LS of LV (%)	-23.07 ± 0.59	-10.59 ± 7.30	-11.52 ± 1.53	0.537
Apical Anteroseptal wall LS of LV (%)	-24 ± 0.84	-10.48 ± 6.81	-11.68 ± 1.52	0.394
Apical INF wall LS of LV (%)	-24 ± 0.001	-9.78 ± 2.06	-10.80 ± 1.50	0.048
Apical LAT wall LS of LV (%)	-21.4 ± 0.98	-10.81 ± 5.07	-8.72 ± 1.51	0.053
GLS 2-chamber view (%)	-22.40 ± 0.92	-11.65 ± 4.45	-10.50 ± 1.47	0.222
GLS 4-chamber view (%)	-21.31 ± 0.87	-14.02 ± 1.84	-9.12 ± 4.24	<0.001
GLS 2-Long-axis view (%)	-20.60 ± 0.89	-10.86 ± 0.75	-9.60 ± 4.34	0.142
Global LS of LV (%)	-21.51 ± 0.43	-12.40 ± 1.35	-10.29 ± 1.46	<0.001

NIDCM: idiopathic dilated cardiomyopathy; IDCM: ischemic dilated cardiomyopathy; LA: left atrium; LV: left ventricle; Ss: left atrial systolic strain; SRs: left atrial systolic strain rate; Sa: left atrial late diastolic strain; SRa: left atrial late diastolic strain rate; ANT: anterior; INF: inferior; POST: posterior; LAT: lateral; LS : longitudinal strain; GLS: global longitudinal strain

Table 3: Correlations between LA deformation (LA Strains and Strain Rates) and Left Atrial Volume and Function Indexes in NIDCM Patients. (p < 0.05) Correlation is significant.

		Average LA Ss (%)	Average LA SRs (s^{-1})	Average LA Sa (%)	Average LA SRa (s^{-1})
LAVmax (cm3/m2)	**r**	0.932	0.900	0.929	-0.937
	p	<0.001	<0.001	<0.001	<0.001
LATV (cm3/m2)	**r**	0.946	0.927	0.950	-0.952
	p	<0.001	<0.001	<0.001	<0.001
AEI (%)	**r**	0.974	0.947	0.970	-0.972
	p	<0.001	<0.001	<0.001	<0.001
LAAEF (%)	**r**	0.982	0.962	0.980	-0.970
	p	<0.001	<0.001	<0.001	<0.001

LA: left atrium; NIDCM: non-ischemic dilated cardiomyopathy; LAVmax: left atrial maximum volume; LATV: left atrial total emptying volume; AEI: left atrial expansion index; LAAEF: left atrial active ejection fraction; Ss: left atrial systolic strain; SRs: left atrial systolic strain rate; Sa: left atrial late diastolic strain; SRa: left atrial late diastolic strain rate

reservoir and booster pump functions were compromised. Moreover, we found higher E/e' and lower LVEF in DCM patients than healthy controls. In agreement, Gulr and colleagues showed LA function impairment in patients with echocardiographically detected diastolic dysfunction [20].

Comparison of LA strains and strain rates in NIDCM and IDCM patients: We recorded no significant differences between NIDCM and IDCM patients in terms of basic echocardiographic variables, including the A peak, deceleration time (DT), septal e', E/A, E/e', LAD and LVEDD. However, left atrial parameters were lower in NIDCM than in IDCM patients. This was in agreement with Moyssakis and associates (using stress echocardiography) [11] and D'Andrea and coworkers (using 2DSTI) [21]. Left Atrial dynamic reservoir and booster pump functions were decreased in NIDCM patients and partially preserved in IDCM patients despite similar loading conditions and LA sizes.

It is reasonable to assume that the depressed LA function in NIDCM patients is the result of LA involvement in the cardiomyopathy process [22]. This assumption is supported by previous findings of higher degrees of LA changes in NIDCM than in IDCM patients. Moreover, Sen and associates identified lower amplitude of cell motion in NIDCM than IDCM patients, as well as significant differences in the sarcoplasmic reticulum, Ca release dysfunction between NIDCM and IDCM. The latter plays a crucial role in abnormal Ca21 homeostasis in NIDCM patients leading to impaired LV systolic and diastolic function over time. In such circumstances, LA pressure and size would increase, and the AEI and LAAEF would decrease [23].

Molina-Navarro and associates examined the global transcriptome profiles using RNA-Sequencing and GeneMANIA of 31 LV myocardial tissue samples (10 IDCM; 13 NIDCM; 8 control donors). The study showed different expression in nine and 12 nucleocytoplasmic transport-related genes in the IDCM and NIDCM groups. Six altered genes showed identical expression trends in both diseases: XPO1, ARL4, NFKB2, FHL3, RANBP2, and RHOU. However, INIDCM patients showed expression of three unique genes (DDX3X, KPNA2, and PTK2B), whereas, other sex genes were expressed only in NIDCM patients (SMURF2, NUP153, IPO5, RANBP3, NOXA1, and RHOJ) [24].

Correlation between LA strain rates and LA volume index: In our study, the LA strain rate was positively correlated with volume indices such as AEI and LAAEF. This suggests that it could reflect LA dysfunction at different points of the cardiac cycle. We found that LAVmax was higher in NIDCM

and IDCM patients than the control group. However, AEI and LAAEF were lower, indicating changes in the dynamic reservoir and booster pump function. This might be explained by the following: impairment of Left ventricular contraction and active relaxation, which hinders the motion of the mitral valve ring, compensation of left ventricular diastolic dysfunction by atrial booster pump function. Higher LVFP increased left atrial pressure and reduced LAAEF [25]. In addition, we found no significant difference in LAVmax and LAEV between the NIDCM and IDCM groups, but AEI and LAAEF were lower in NIDCM patients, albeit having similar Left ventricular systolic and diastolic function and Left atrial size.

Study limitations

Limitations of Our study includes the small sample size and the ability of STI to estimate deformation in 3D independently of angle, which gives additional information such as the longitudinal, circumferential, radial, and area strain.

Conclusion

STE is a promising method for evaluating LAF in DCM patients. Patients with DCM had significantly lower left atrial systolic and late diastolic strains and strain rates compared to healthy patients. Moreover, NIDCM could be differentiated from IDCM by having more impairment in the LA dynamic reservoir and booster pump function.

Conflict of interest: Authors declare no conflict of interest.

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