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ORIGINAL ARTICLE

The relationship between ascending aortic diameter with left atrial functions and left ventricular mass index in a population with normal left ventricular systolic function

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Abstract

Aim: Ascending aortic dilatation is a common clinical issue. In the present study, we aimed to evaluate the relationship between ascending aortic diameter with left ventricular (LV) and left atrial (LA) functions, and LV mass index (LVMI) in a population with normal LV systolic function.

Methods: A total of 127 healthy participants with normal LV systolic function took part in the study. Echocardiographic measurements were obtained from each subject.

Results: The mean age of the participants was 43 ± 14.1 years and 76 (59.8%) were female. The mean aortic diameter of the participants was 32.2 ± 4.7 mm. A negative correlation was found between aortic diameter and LV systolic function (LVEF $r = -.516$, $p < .001$; Gls $r = -.370$). In addition, there was a strong positive correlation between aortic diameter with LV wall thicknesses, LVMI ($r = .745$, $p < .001$), and systolic and diastolic diameters. The relationship between aortic diameter and diastolic parameters was evaluated, a negative correlation with Mitral E, Em, E/A ratio, and a positive correlation with MPI, Mitral A, Am, E/Em ratio were found.

Conclusion: A strong correlation between ascending aortic diameter with LV and LA functions, and LVMI in individuals with normal LV systolic function.

KEYWORDS
ascending aorta diameter, left atrial functions, left ventricle mass index

1 | INTRODUCTION

Aortic dilatation is the most common pathology of the ascending aorta. In researches based on different aortic segments, significant overlap between factors affecting aortic diameter and cardiovascular risk factors exists.[1–3] Age, gender, race, body surface area (BSA), smoking, alcohol, hyperlipidemia (HL), hypertension (HT), and diabetes mellitus (DM) are traditional cardiovascular risk factors affecting aortic diameter.[4–7]

Remodeling of the aortic wall resulting in increased arterial stiffness (due to decreased elastin, increased collagen and calcium) may increase left ventricular (LV) afterload. During systole, LV and ascending aorta are functionally linked. Therefore, the systolic hemodynamic coupling between the proximal aorta and the LV is a crucial determinant of cir-

culatory performance.[8,9] To maintain the effective arterial-ventricular coupling throughout the life, LV adapts to progressive arterial stiffness and afterload by increased systolic elastance, concentric remodeling, and hypertrophy. However, this adaptation may lead to increased oxygen demand and decreased cardiovascular reserve, resulting in heart failure (HF).[10]

The presence of common risk factors associated with remodeling in the aortic wall and the decrease in elasticity due to remodeling may cause aortic dilatation and deterioration in LV structure and function. Although LV dysfunction has a decisive impact on adverse cardiovascular outcomes, left atrial (LA) dilatation and dysfunction are also substantial. Both circumstances often coexist in a cause-effect relationship.[11] This condition can be demonstrated by increased LV wall thickness, impaired LV systolic and diastolic functions, increased LA volume, and decreased LA strain.[12,13]

Although similar clinical studies exist in the literature, we could find no research showing the mentioned relationship using LV and LA strains in population with normal LV systolic function. Therefore, we aimed to evaluate the relationship between ascending aortic diameter with LV and LA functions, and left ventricle mass-index (LVMI) in this population.

2 | METHODS

The present research is a prospective, single-center, observational cross-sectional cohort study, and included 127 consecutive participants with normal LV systolic functions. All patients had given informed consent and the local ethics committee approved the study. DM and HT were defined according to the current guidelines.[14,15] HL was defined as having a previous diagnosis of hypercholesterolaemia, previous or ongoing oral low-density lipoprotein cholesterol (LDL-C) lowering treatment. Smoking at least one cigarette per day for at least five years was defined as regular smoking. Body mass index was calculated according to the weight (kg)/height $(cm)^2$ formula.

Secondary HT, history of cerebrovascular disease, acute or chronic renal failure, end-stage liver disease, active inflammatory disease, moderate-to-severe heart valve disease, history of cardiac surgery, malignancy, endocrine diseases, electrolyte disorders, anemia, pulmonary embolism, myocarditis, cardiomyopathy (ejection fraction [EF] < 50%) and genetic conditions that affect the body's connective tissue, such as Marfan syndrome, Ehlers-Danlos syndrome, and Loeys-Dietz syndrome were determined as exclusion criteria.

2.1 | Echocardiographic evaluation

Detailed two-dimensional transthoracic echocardiography was performed on all participants using Philips Epiq 7 systems (Philips Medical Systems, Andover, MA) with a 1 to 5 MHz X5-1 transducer. LA diameter, LV end-diastolic and end-systolic diameters, pulsed and continuous Doppler measurements, and E and A wave velocities were quantified with standard two-dimensional M-mode echocardiographic images under current guideline recommendations. LV EF was calculated using the modified Simpson method. Tissue Doppler analysis was performed in apical 4-chamber (A4C) view, by placing the cursor on the septal and lateral mitral ring, to get early mitral annular diastolic velocity (e).

LV global longitudinal strain (GLS) was determined using two-dimensional speckle tracking echocardiography. Three standard apical views, apical two chamber (A2C), apical three-chamber (A3C), and A4C views, were obtained. The mean GLS was measured by averaging the peak GLS values of these chamber images. Measurements of LVGLS were performed offline using a commercially available software (QLAb) program. LV mass was calculated with the linear M-mode Deveraux formula and indexed to the BSA (LV mass index [LVMI]).[16]

The ascending aortic diameter was measured perpendicular to the long axis of the aorta with the use of the leading-edge method in parasternal long-axis view showing the largest aortic diameter following the American Society of Echocardiography guidelines.[17] The largest ascending aorta was also measured in cases of sinus valsalva dilatation.

LA volume was measured using the area-length method through the A4C and A2C images. The LA appendage and pulmonary veins were not included in the LA volume measurement. The LA maximal volume (at ventricular end-systole, just before mitral valve opening), LA minimum volume (at the end of ventricular diastole, after mitral valve closure), and LA pre-A volume (just before atrial systole, before the electrocardiographic P wave) were measured. The LA volume index (LAVI) (mL/m^2) was calculated by dividing the LA volume by BSA. Other parameters were measured using the formulas given below:

LA emptying vol = (LA max vol − LA min vol)
LA emptying fraction% = (LA max vol − LA min vol) /LA max vol
LA passive EF% = [(LA max vol − LA pre Avol) /LA max vol]

2.2 | Statistical analysis

SPSS software package (Version 23.0, SPSS, Inc., Chicago, IL) was used for data analysis. The normality assumption of data was assessed by the visual (histograms, probability plots) and analytical methods (Kolmogorov-Smirnov/Shapiro-Wilk's test). Levene's test was used to check the homogeneity of variances. A mean ± standard deviation chart was used to represent the continuous variables and percentages were used to represent the categorical variables. All linear variables were found to have a normal distribution. Correlation analysis was performed between continuous variables. Continuous variables which had significant correlation with aortic diameter were evaluated by multivariate backward linear analysis. A $p < .05$ was considered significant.

3 | RESULTS

A total of 127 participants (51 male and 76 female) with a mean age of 43 ± 14.1 years were included in the analysis. The mean aortic diameter was 32.2 ± 4.7 mm (Table 1). Aortic diameter was positively correlated

TABLE 1 Demographic, clinical, and laboratory characteristics of participants.

Variable	*n* = 127
Gender (Male) n(%)	51 (40.2)
Age (year)	43 ± 14.1
Hypertension *n* (%)	31 (24.4)
Diabetes Mellitus *n* (%)	12 (9.4)
CAD *n* (%)	11 (8.7)
Current Smoking *n* (%)	27 (11.3)
Hyperlipidemia *n* (%)	16 (12.6)
Acetyl Salicylic Acid *n* (%)	11 (8.6)
ACEI *n* (%)	9 (7.1)
ARB *n* (%)	19 (15)
P2Y12 inhibitors *n* (%)	16 (12.6)
Beta Blocker *n* (%)	17 (13.4)
Statin *n* (%)	15 (11.8)
CCB *n* (%)	13 (10.2)
LV EF %	64.2 ± 2.5
Aortic Diameter (mm)	32.2 ± 4.7
eGFR	100.6 ± 19.2
Glucose	106 ± 26.2
BMI	28.5 ± 5.2
LVEF (%)	63.5 ± 3.2
GLS	18.4 ± 2.7
LAVI	24.7 ± 9.05
LVMI	153.6 ± 47.5
EFT	4.1 ± .17

Abbreviations: ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blocker; BMI, body mass index; CAD, coronary artery disease; CCB, calcium channel blocker; EF, ejection fraction; EFT, epicardial fat thickness; eGFR, estimated glomerular filtration rate; GLS, global Longitudinal Strain; LAVI, left atrial volüme index; LV, left ventricle; LVMI, left ventricular mass index.

with age and fasting glucose level and negatively correlated with eGFR. There was a negative correlation between aortic diameter and LV systolic functions (LVEF $r = -.516$, $p < .001$; GLS $r = -.370$). In addition, there was a strong positive correlation between aortic diameter with LV wall thickness, LVMI ($r = .745$, $p < .001$), and systolic and diastolic diameters (Table 2 and Figure 1).

Remodeling of the LV wall leads to impaired diastolic functions. We found a negative correlation between aortic diameter and Mitral E, Em, E/A ratio, and a positive correlation with MPI, Mitral A, Am, E/Em ratio (Table 2 and Figure 2). These findings showed a correlation between an increase in aortic diameter and an impairment in diastolic parameters. In addition, aortic diameter tended to increase with epicardial adipose tissue volume. This result was consistent with the relationship between epicardial adipose tissue content and LV and aortic diameters obtained in previous studies.

Deterioration in LV diastolic and systolic functions affects the LA structure and function. In the present study, aortic diameter increase was positively correlated with LAVI and LA Total Empty Volume, while negatively correlated with LA Total Empty fraction and LA Passive Empty Fraction (Table 2 and Figure 3). In linear regression analysis, age, PW, LVSD, LVMI, LAPaEmVol, and LAPaEmFr were independently associated with the aortic diameter (Table 3).

4 | DISCUSSION

In the present study, we observed a strong correlation between ascending aortic diameter with LV and LA functions, and LVMI in individuals with normal LV systolic function.

Aortic size increases throughout life, accompanied by loss of compliance and wall stiffness, and this process ultimately leads to arterial dilatation. An increase in ascending aortic diameter is an indicator of cardiovascular and all-cause mortality.[18] Proximal aortic dilatation can be considered an indicator of the effect of cardiovascular risk factors with prognostic importance.

The pathogenesis of ascending aortic dilatation includes various factors with local or systemic effects. Mechanisms such as hemodynamic strength, transmural inflammation, remodeling of the extracellular matrix, and familial predisposition are among these factors.[19–23]

There is no particular evidence for the predictive role of aortic dilatation on cardiovascular mortality. Most studies have examined a combination of non-fatal and fatal stroke, coronary events, and HF requiring hospitalization as an endpoint.[24–30] The Framingham Heart Study showed that participants with larger aortic root diameters experienced more HF-related events over an 8-year follow-up period.[24] Consistent with the previous researches, a negative correlation was observed between aortic diameter and LV systolic functions in the present study.

The LA wall is relatively thin, similar to the aortic wall.[31] Studies have shown that endothelial dysfunction can have a direct impact on LA dilation and function.[32] Factors associated with endothelial dysfunction, such as HT and DM, may cause both subclinical LA dysfunction and aortic dilatation before LV diastolic dysfunction develops.

In the present study, a relationship between aortic diameter and diastolic functions was observed. Masugata et al. reported the relationship of aortic root dilatation (ARD) with LV diastolic function parameters E, E/A ratio, and E'.[33] In the study, increased ARD levels were found to be associated with EDT, mitral E/A, and IVRT, and LA functions were associated with ARD independently of LV diastolic dysfunction. This finding suggests that the systemic effects of HT and DM affect the LA and LV, and changes in the aorta are components of this process. It should be kept in mind in the follow-up of patients that LA mechanical and LV diastolic functions may be impaired and atrial arrhythmias, in terms of stroke risk, may occur in hypertensive diabetic patients diagnosed with ARD.

The elastic properties of the aorta partially determine the LV function. The LV's diastolic functions facilitate an atrial-ventricular pressure gradient that allows blood to fill the LV. Ventricular

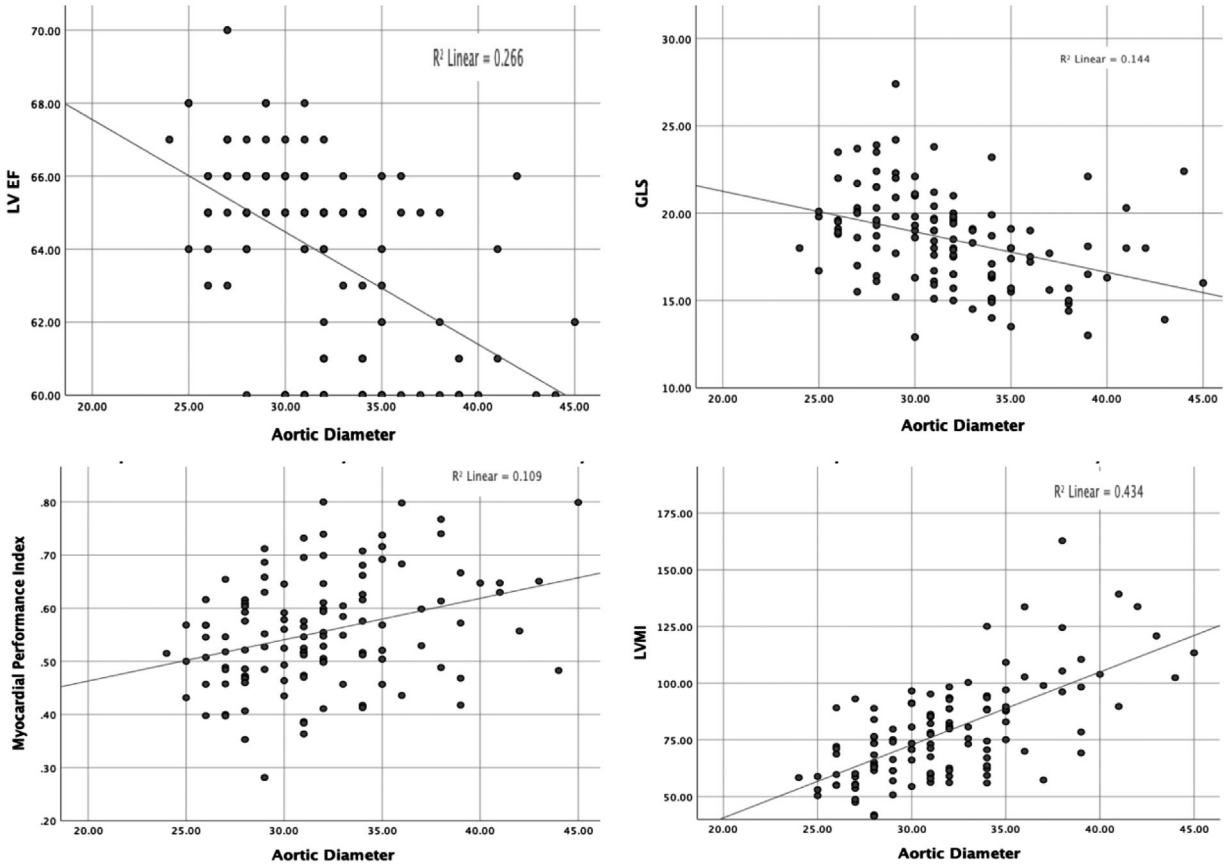

FIGURE 1 Correlation of LV EF, GLS, miyocardial performance index and LVMI with aortic diameter.

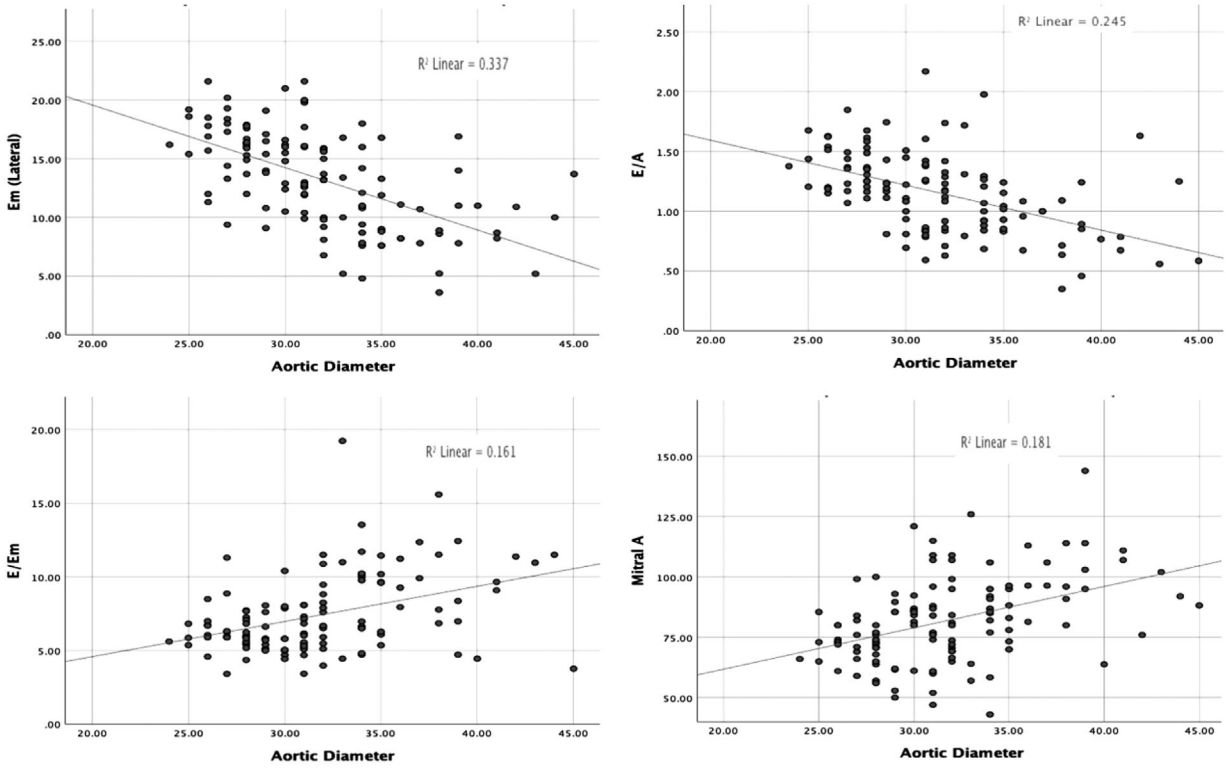

FIGURE 2 Correlation of diastolic parameters with aortic diameter.

TABLE 2 Univariate correlation analysis between aortic diameter and clinical/echocardiographic parameters of the study population.

Variable	Aortic diameter		Variable	Aortic dimeter	
LV EF (%)	*r*	−.516	Mitral E	*r*	−.299
	p	<.001		*p*	.001
GLS (%)	*r*	−.370	Mitral A	*r*	.425
	p	<.001		*p*	<.001
IVS Dia.	*r*	.655	Em (Lat)	*r*	−.580
	p	<.001		*p*	<.001
PW Dia.	*r*	.649	Am (Lat)	*r*	.321
	p	<.001		*p*	<.001
LVD Dia.	*r*	.592	MPI (Mitral)	*r*	.288
	p	<.001		*p*	.002
LVS Dia.	*r*	.520	EA Ratio	*r*	−.511
	p	<.001		*p*	<.001
LAVI	*r*	.370	EEm Ratio	*r*	.401
	p	<.001		*p*	<.001
LA Total Emptying Volume	*r*	.293	EFT	*r*	.496
	p	.001		*p*	<.001
LA Total Emptying Fraction	*r*	−.329	LVMI	*r*	.745
	p	<.001		*p*	<.001
LA Passive Emptying Fraction	*r*	−.358	LVM	*r*	.737
	p	*p* < .001		*p*	<.001
eGFR	*r*	−.545	Glucose	*r*	.237
	p	<.001		*p*	.008
Age	*r*	.739	BMI	*r*	.433
	p	<.001		*p*	<.001

Abbreviations: Am, late diastolic peak; BMI, body mass index; Dia, diameter; EF, ejection fraction; EFT, epicardial fat thickness; eGFR, estimated glomerular filtration rate; Em, early diastolic peak; GLS, global longitudinal strain; IVS, interventricular septum; LA, left atrial; LAVI, left atrial volume index; LV, left ventricle; LVD, left ventricular diastolic; LVM, left ventricular mass; LVMI, left ventricular mass index; LVS, left ventricular systolic; MPI, myocardial performance index; PW, posterior wall.

relaxation and compliance are substantial factors affecting LV diastolic functions.[34] To ensure normal cardiac performance, the LV must be able to provide an adequate stroke volume and be filled without requiring high LA pressure. These (systolic and diastolic) functions should meet the body's needs both at rest and during exertion.[35]

Recently, an independent relationship between the index of diastolic function which is defined as deceleration time/peak E velocity ratio, and aortic root size, has been demonstrated in a population-based sample[36] and in a HT cohort study.[37] All these findings suggest that there is a common pathway in the pathogenesis of ARD and LV diastolic dysfunction. This connection was further supported by the independent relationship of aortic root size observed in the entire study population, reflecting LV relaxation/filling, which is a valid representation of LA diameter and volume.

In the present study, LV wall thicknesses and LVMI were associated with aortic diameter. Echocardiographic LV hypertrophy (LVH) is an important biomarker of hypertensive heart disease and a strong predictor of cardiovascular morbidity and mortality.[38–42] In particular, the relationship between aortic dilatation and LVH highlights the role of combined arterial-ventricular remodeling. These finding shows that remodeling in the aortic and LV wall is interrelated. This is may be due to the factors that cause remodeling in the aortic wall also play a role in the remodeling of the LV wall. Another possible reason is that remodeling in the aortic wall triggers remodeling in the LV wall by creating an additional afterload or disrupting the physiological relationship between the aorta and the LV wall. Findings from the general population and hypertensive cohorts have shown that the incidence of cardiovascular events increases when LV remodeling and aortic wall remodeling proceed in parallel.[26,29]

In nine studies that found a positive relationship between aortic diameter and cardiovascular events, the inclusion of LV mass in the statistical findings eliminates the prognostic importance of aortic diameter in predicting HF, non-fatal and fatal cardiovascular events, and all-cause mortality in hypertensive patients using anti-hypertensives.[26,28,43] On the contrary, two Italian studies have demonstrated that aortic root and ascending aortic diameter are independent predictors of cardiovascular events, independent of LVH and other factors.[29,30]

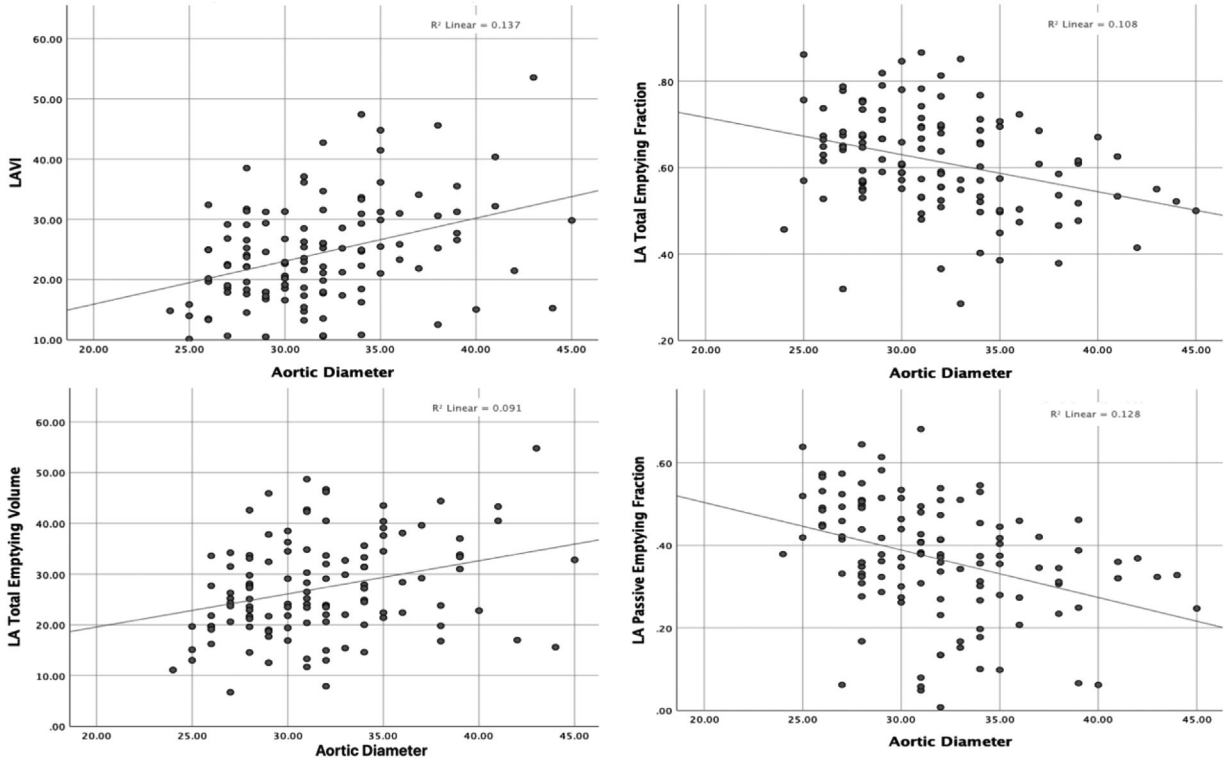

FIGURE 3 Correlation of left atrial function parameters with aortic diameter.

TABLE 3 Linear regression analysis.

Variable	Uns. B	Std. C. B	t	Sig.	%95 CI Lower B.	%95 CI Upper B.
Age	.171	.523	6.949	<.001	.122	.220
PW	−10.2	−.327	−2.392	.018	−18.62	−1.75
LVSD	2.73	.169	2.509	.014	.569	4.837
LAPaEmVol	.098	.177	2.600	.011	.023	.172
LAPaEmFr	−5.689	−.173	−2.222	.028	−10.76	−.616
LVMI	.052	.519	3.634	<.001	.024	.081
Constant	19.3		5.337	<.001	12.1	26.49

Abbreviations: LAPaEmFr, left atrial passive emptying fraction; LAPaEmVol, left atrial passive emptying volume; LVSD, left ventricle systolic diameter; LVMI, left ventricle mass index; PW, posterior wall.

An association between sinus Valsalva dilatation and LV mass has been reported in previous studies.[44–46] It has been shown that an increase in LV mass can be detected in the dilatation of the ascending aorta, supporting our findings.[47]

5 | CONCLUSION

In the current study, we observed a strong correlation between ascending aortic diameter with LV and LA functions, and LVMI in individuals with normal LV systolic function. It would be beneficial to evaluate ascending aorta diameter in clinical practice due to its prognostic importance.

5.1 | Limitations

The study was single-center and was conducted with a limited number of patients. Aortic diameter imaging was performed only with 2D echocardiography. Evaluation with invasive or advanced imaging methods will reduce the margin of error. Large population studies are needed to confirm the findings and clarify the underlying mechanisms.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy orethical restrictions.

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REFERENCES

1. Mensel B, Hesselbarth L, Wenzel M, et al. Tho- racic and abdominal aortic diameters in a general population: mRI-based reference values and association with age and cardiovascular risk factors. *Eur Radiol*. 2016;26(4):969-978.
2. Tarnoki AD, Tarnoki DL, Littvay L, Garami Z, Karlinger K, Berczi V. Genetic and environmental effects on the abdominal aortic diameter development. *Arq Bras Cardiol*. 2016;106(1):13-17.
3. Allison MA, Kwan K, DiTomasso D, Wright CM, Criqui MH. The epidemiology of abdominal aortic diameter. *J Vasc Surg*. 2008;48(1):121-127.
4. Stackelberg O, Wolk A, Eliasson K, et al. Lifestyle and risk of screening-detected abdominal aortic aneurysm in men. *J Am Heart Assoc*. 2017;6(5):e004725.
5. Cetin M, Kocaman SA, Durakoglugil ME, et al. Independent determinants of ascending aortic dilatation in hypertensive patients: smoking, endothelial dysfunction, and increased epicardial adipose tissue. *Blood Press Monit*. 2012;17(6):223-230.
6. Wang JA, Chen XF, Yu WF, et al. Relationship of heavy drinking. lipoprotein (a) and lipid profile to infrarenal aortic diameter. *Vasc Med (Lond, Engl)*. 2009;14(4):323-329.
7. Tanaka A, Ishii H, Oshima H, et al. Inverse association between diabetes and aortic dilatation in patients with advanced coronary artery disease. *Atherosclerosis*. 2015;242(1):123-127.
8. Chirinos JA. Ventricular-arterial coupling: invasive and non-invasive assessment. *Artery Res*. 2013;7:2-14. doi:10.1016/j.artres.2012.12.002
9. Kass DA. Ventricular arterial stiffening: integrating the pathophysiology. *Hypertension*. 2005;46:185-193. doi:10.1161/01.HYP.0000168053.34306.d4
10. Bluemke DA, Kronmal RA, Lima JA, et al. The relationship of left ventricular mass and geometry to incident cardiovascular events: the MESA (Multi-Ethnic Study of Athero- sclerosis) study. *J Am Coll Cardiol*. 2008;52:2148-2155. doi:10.1016/j.jacc.2008.09.014
11. Abhayaratna WP, Fatema K, Barnes ME, et al. Left atrial reservoir function as a potent marker for first atrial fibrillation or flutter in persons !65 years of age. *Am J Cardiol*. 2008;101:1626-1629.
12. Saeed S, Waje-Andreassen U, Nilsson PM. The association of the metabolic syndrome with target organ damage: focus on the heart brain and central arteries *Expert Rev of Cardiovasc Ther*. 2020;18:601-614.
13. Bell V, McCabe EL, Larson MG. Relations between aortic stiffness and left ventricular mechanical function in the community. *J Am Heart Assoc*. 2017;6(1-9):e004903.
14. Cosentino F, Grant PJ, Aboyans V, et al. 2019 ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD. *Eur Heart J*. 2020;41(2):255-323. doi:10.1093/eurheartj/ehz486. Erratum in: Eur Heart J. 2020 Dec 1;41(45):4317. PMID: 31497854.
15. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. *Eur Heart J*. 2018;39(33):3021-3104. doi:10.1093/eurheartj/ehy339. Erratum in: Eur Heart J. 2019 Feb 1;40(5):475. PMID: 30165516.
16. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quan- tification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Journal of the American Society of Echocardiography: official publication of the American Society of Echocardiography. *J Am Soc Echocardiography*. 2015;28(1):1-39.e14. doi:10.1016/j.echo.2014.10.003
17. Mitchell C, Rahko PS, Blauwet LA, et al. Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2019;32:1-64.
18. Gardin JM, Arnold AM, Polak J, Jackson S, Smith V, Gottdiener J. Usefulness of aortic root dimension in persons >or = 65 years of age in predicting heart failure, stroke, cardiovascular mortality, all-cause mortality and acute myocardial infarction (from the Cardiovascular Health Study). *Am J Cardiol*. 2006;97:270-275. doi:10.1016/j.amjcard.2005.08.039
19. Ramanath VS, Oh JK, Sundt TM 3rd, Eagle KA. Acute aortic syndromes and thoracic aortic aneurysm. *Mayo Clin Proc*. 2009;84:465-481. doi:10.1016/S0025-6196(11)60566-1
20. Dobrin PB, Baker WH, Gley WC. Elastolytic and collagenolytic studies of arteries. Implications for the mechanical properties of aneurysms. *Arch Surg*. 1984;119:405-409.
21. Brophy CM, Reilly JM, Smith GJ, Tilson MD. The role of inflammation in nonspecific abdominal aortic aneurysm disease. *Ann Vasc Surg*. 1991;5:229-233. doi:10.1007/BF02329378
22. Patel MI, Hardman DT, Fisher CM, Appleberg M. Current views on the pathogenesis of abdominal aortic aneurysms. *J Am Coll Surg*. 1995;181:371-382.
23. Elefteriades JA. Natural history of thoracic aortic aneurysms: indications for surgery, and surgical versus nonsurgical risks. *Ann Thorac Surg*. 2002;74:S1877-S1880.
24. Lai CL, Chien KL, Hsu HC, Su TC, Chen MF, Lee YT. Aortic root dimension as an independent predictor for all-cause death in adults <65 years of age (from the Chin-Shan community cardiovascular cohort study). *Echocardiography*. 2010;27:487-495. doi:10.1111/j.1540-8175.2009.01072.x
25. Gondrie MJ, van der Graaf Y, Jacobs PC, Buckens SC, Mali WP. Providi Study Group. The prognostic value of vascular diameter measurements on routine chest computed tomography in patients not referred for cardiovascular indications. *J Comput Assist Tomogr*. 2011;35:734-741. doi:10.1097/RCT.0b013e318231824a
26. Cuspidi C, Facchetti R, Bombelli M, et al. Aortic root diameter and risk of cardiovascular events in a general population: data from the PAMELA study. *J Hypertens*. 2014;32:1879-1887. doi:10.1097/HJH.0000000000000264
27. Kamimura D, Suzuki T, Musani SK, et al. Increased proximal aortic diameter is associated with risk of cardiovascular events and all-cause mortality in blacks the Jackson heart study. *Am Heart Assoc*. 2017;6:e005005. doi:10.1161/JAHA.116.005005
28. Qazi S, Massaro JM, Chuang ML, D'Agostino RB Sr, Hoffmann U, O'Donnell CJ. Increased aortic diameters on multidetector computed tomographic scan are independent predictors of incident adverse cardiovascular events: the Framingham heart study. *Circ Cardiovasc Imaging*. 2017;10:e006776. doi:10.1161/CIRCIMAGING.117.006776
29. Canciello G, Mancusi C, Losi MA, et al. Aortic root dilatation is associated with incident cardiovascular events in a population of treated hypertensive patients: the Campania salute network. *Am J Hypertens*. 2018;31:1317-1323. doi:10.1093/ajh/hpy113

30. Leone D, Airale L, Bernardi S, et al. Prognostic role of the ascending aorta dilatation in patients with arterial hypertension. *J Hypertens*. 2021;39:1163-1169. doi:10.1097/HJH.0000000000002752
31. Whitaker J, Rajani R, Chubb H. The role of myocardial wall thickness in atrial arrhythmogenesis. *Europace*. 2016;18(12):1758-1772.
32. Khan AA, Thomas GN, Lip GYH, Shantsila A. Endothelial function in patients with atrialfibrillation. *Ann Med*. 2020;52(1–2):1-11.
33. Masugata H, Senda S, Murao K, et al. Aortic root dilatation as a marker of subclinical left ventricular diastolic dysfunction in patients with cardiovascular risk factors. *J Int Med Res*. 2011;39(1):64-70.
34. Wu EB, Yu CM. Management of diastolic heart failure - a practical review of pathophysiology and treatment trial data. *Int J Clin Pract*. 2005;59:1239-1246.
35. Little WC, Oh JK. Echocardiographic evaluation of diastolic function can be used to guide clinical care. *Circulation*. 2009;120:802-809.
36. Mishra RK, Galloway JM, Lee ET, et al. The ratio of mitral deceleration time to E-wave velocity and mitral deceleration slope outperform deceleration time alone in predicting cardiovascular outcomes: the Strong Heart Study. *J Am Soc Echocardiogr*. 2007;20:1300-1306.
37. Chinali M, Aurigemma GP, de Simone G, et al. Mitral E wave deceleration time to peak E velocity ratio and cardiovascular outcome in hypertensive patients during antihypertensive treatment (from the LIFE Echo-Substudy). *Am J Cardiol*. 2009;104:1098-1104.
38. Kizer JR, Bella JN, Palmieri V, et al. Left atrial diameter as an independent predictor of first cardiovascular events in middle-aged and elderly adults: the Strong Heart Study (SHS). *Am Heart J*. 2006;151:412-418.
39. Pareek M, Vaduganathan M, Bhatt DL, Leósdóttir M, Olsen MH. Prognostic implications of fasting plasma glucose in subjects with echocardiographic abnormalities. *Int J Cardiol*. 2017;241:423-429. doi:10.1016/j.ijcard.2017.01.133
40. Lundorff I, Modin D, Mogelvang R, et al. Echocardiographic predictors of cardiovascular morbidity and mortality in women from the general population. *Eur Heart J Cardiovasc Imaging*. 2021;22:1026-1034. doi:10.1093/ehjci/jeaa167
41. Guzik BM, McCallum L, Zmudka K, Guzik TJ, Dominiczak AF, Padmanabhan S. Echocardiography predictors of survival in hypertensive patientswith left ventricular hypertrophy. *Am J Hypertens*. 2021;34:636-644. doi:10.1093/ajh/hpaa194
42. East MA, Jollis JG, Nelson CL, Marks D, Peterson ED. The influence of left ventricular hypertrophy on survival in patients with coronary artery disease: do race and gender matter? *J Am Coll Cardiol*. 2003;41:949-954. doi:10.1016/s0735-1097(02)03006-1
43. Lam CSP, Gona P, Larson MG, et al. Aortic root remodeling and risk of heart failure in the Framingham heart study. *J Am Coll Cardiol Heart Fail*. 2013;1:79-83. doi:10.1016/j.jchf.2012.10.003
44. Cuspidi C, Negri F, Salvetti M, et al. *Aortic root dilatation in hypertensive patients: a multicenter survey in echocardiographic practice*. Blood Press; 2011.
45. Cuspidi C, Meani S, Fusi V, Valerio C, Sala C, Zanchetti A. Prevalence and correlates of aortic root dilatation in patients with essential hypertension: relationship with cardiac and extracardiac target organ damage. *J Hypertens*. 2006;24(3):573-580.
46. Bella JN, Wachtell K, Boman K, et al. Relation of left ventricular geometry and function to aortic root dilatation in patients with systemic hypertension and left ventricular hypertrophy (the LIFE study). *Am J Cardiol*. 2002;89(3):337-341.
47. Milan A, Tosello F, Naso D, et al. Ascending aortic dilatation, arterial stiffness and cardiac organ damage in essential hypertension. *J Hypertens*. 2013;31(1):109-116. PMID: 23221933. doi:10.1097/HJH.0b013e32835aa588

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