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Research Article

Ascending Aortic Stiffness Analysis Using Tissue Doppler Imaging for the Diagnosis of Coronary Artery Stenosis in Suspected Stable Angina: A Retrospective Study

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Abstract

Background: Coronary artery disease (CAD) and arterial stiffening may coexist. Stable angina pectoris (SAP) is one of the common types of CAD. However, the association between SAP and aortic stiffness metrics remains poorly understood. Tissue Doppler imaging (TDI) measurement of ascending aorta motion velocity may be used to employed to assess the elastic properties of the great arteries. We hypothesized that non-CAD individuals may exhibit higher. TDI velocities in the ascending aorta. Aim: To explore the correlation between ascending aortic stiffness and SAP using parameters derived from two-dimensional and TDI echocardiography of the ascending aorta. Methods: This study comprised 118 patients with clinically suspected SAP. Two-dimensional echocardiography, TDI, electrocardiogram (ECG), and coronary angiography (CAG) were performed on all patients. Patients with coronary lumen area stenosis ≥70% were categorized as having significant CAD (CAD Group n=57) and were compared with patients without significant CAD (non-CAD Group n=61). Using TDI, aortic systolic velocity (SAo), early diastolic velocity (EAo), and late diastolic velocity (AAo) were measured from the anterior wall of the ascending aorta 3 cm above the aortic cusps in the parasternal long-axis view. Aortic stiffness index (β), aortic distensibility (D), and pressure-strain elastic modulus (Ep) were calculated from aortic diameters measured by two-dimensional M-mode echocardiography and blood pressure obtained by sphygmomanometry. Results: SAo was significantly higher in the non-CAD group (11.70 ±1.53 cm/s vs.12.80 ±2.21 cm/s, $p < 0.05$). EAo and AAo velocities of ascending aorta were similar in control and CAD groups. Based on the receiver operating characteristic curve (ROC curve) for diagnosing non-CAD, the optimal cut-off value of SAo was ≥13.35 cm/s (sensitivity, 85.96%; specificity, 40.98%; area under curve (AUC)=0.64; $P<0.05$). There was a significant correlation between SAo velocity and β ($r=-0.34$, $P<0.05$), D ($r=0.32$, $P<0.05$) and Ep ($r=-0.29$, $P<0.05$). Conclusions: Arterial stiffness is lower in patients without significant CAD. Measuring SAo of the anterior ascending aorta using TDI echocardiography has good sensitivity but poor specificity in patients without significant coronary artery stenosis in SAP.

Keywords

Aortic Stiffness, Coronary Artery Disease, Tissue Doppler Imaging, Stable Angina Pectoris, Echocardiography

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1. Introduction

Coronary artery disease (CAD) is a common cardiovascular disease, and its mortality has shown an obvious upward trend in recent years, with stable angina pectorisis (SAP) being most common [1]. Arteriosclerosis is the main pathogenic factor of cardiovascular disease [2]. The aorta plays an important role in regulating left ventricular function, myocardial perfusion, and hemodynamics [3]. Increased aortic stiffness is an effective predictor of cardiovascular morbidity and mortality [4]. Aortic stiffness is strongly correlated with coronary stenosis [5]. At the same time, although some patients with stable angina pectoris have no obvious symptoms, the rate of coronary artery stenosis can reach more than 70%. If stable angina pectoris is not accurately diagnosed, the risk of cardiovascular endpoint events will double [6]. Although Exercise treadmill testing (ETT) is a noninvasive diagnostic test for detection of CAD recommended by the American Heart Association (AHA). However, there are contraindications for its use. Although computed tomography angiography (CTA) is a non-invasive procedure, it usually overestimates the degree of coronary stenosis. The gold standard for the diagnosis of stable angina pectoris is still coronary angiography (CAG). However, CAG is an invasive operation, and there is a certain risk of complications, so it cannot be used as a routine inspection method [7]. Therefore, how to quickly, accurately and non-invasively diagnose stable angina pectoris and prevent the occurrence of missed and misdiagnosed is a thorny problem currently facing.

Echocardiography is the dominant cardiac imaging technique in patients with suspected cardiac disease. However, conventional echocardiography provides little information regarding risk stratification of patients with suspected stable angina. Tissue Doppler imaging (TDI) can easily be implemented in the conventional echocardiographic protocol; hence it would be very appealing if TDI could provide additional information regarding risk stratification of these patients. Hoffman et al report that tissue Doppler is an independent predictor of stable angina pectoris [8]. Finally, TDI has few contraindications and can be used for rapid bedside examination. Therefore, we performed TDI of the ascending aorta in patients with suspected stable angina pectoris, analyzed the Doppler motion velocity spectrum of the ascending aorta tissue, and explored whether it predicted the presence of significant CAD, and whether it was associated with coronary arterial stenosis.

2. Methods

2.1. Study Population

We retrospectively studied 118 patients (male 71, female 47) who were hospitalized with the diagnosis of suspected SAP between January 2020 and May 2022. Baseline characteristics of the study population, including age, sex, weight, height, blood pressure, past medical history, smoking status, family history, and medications, were obtained from inpatient medical records. Fasting blood samples and 12-lead electrocardiography were also obtained. Exclusion criteria included valvular heart disease, heart dysfunction defined by LV ejection fraction (LVEF) <50%, arrhythmias, intraventricular conduction disturbances, pathologic Q waves, chronic kidney disease, aortic aneurysms, and systemic diseases affecting the aorta, such as Marfan syndrome and Takayasu's arteritis. Conventional echocardiography, M-mode echocardiography, and TDI of the ascending aorta were performed in all patients prior to coronary angiography. Coronary angiography was performed irrespective of the echocardiography and TDI findings of the ascending aorta.

2.2. Conventional Echocardiography

All patients underwent conventional echocardiography in the left lateral decubitus position. Studies were performed using a commercially available system (Philips iE33, S5-1 probe, Philips, Bothell, WA, USA). Images were obtained using a 3.5-MHz transducer, at a depth of 16 cm in the parasternal (long- and short-axis) and apical (2- chamber and 4-chamber) views. Standard 2-D and color Doppler data, triggered to the QRS complex, were stored in cine-loop format. A minimum of 3 consecutive beats were recorded from each view. All of the images were obtained at the end of expiration as recommended. All echocardiographic findings were analyzed by one of the authors, who were blinded to the subjects' past histories. Measurements of cardiac chambers were made by transthoracic echocardiography. Conventional echocardiographic values, including LV dimensions, LV ejection fraction (LVEF), LV fractional shortening (LVFS), LV mass index, peak velocity of early diastolic filling (E) and peak velocity of atrial systole (A).

2.3. M-Mode Echocardiography

Aortic diameters were measured at a level 3 cm above the aortic cusps in long axis from the parasternal view (Figure 1). Systolic aortic diameter (As) was measured at the point of maximal anterior motion of the ascending aorta (systole), and diastolic aortic diameter (Ad) was measured at the q wave on electrocardiogram (end diastole). The means of three diameter measurements in sequential cardiac cycles were used for data analysis. Systemic arterial blood pressure (BP) was measured at the right brachial artery by manual sphygmomanometer with the patient supine using an adequately sized cuff. BP was measured three times on each occasion at 2 min intervals and averaged. Pulse pressure (PP) was obtained by subtracting the diastolic BP from the systolic BP.

The elastic properties of the ascending aorta were indexed by calculation of aortic distensibility (D), stiffness index (β)

and pressure strain elastic modulus (Ep) and were as follows: D = 2 (As−Ad)/ [Ad (Ps−Pd)], β = ln(Ps/Pd)/ [(As−Ad)/Ad] and Ep = (Ps−Pd)/ [(As−Ad)/Ad], respectively, where As is the aortic diameter at end-systole, Ad is the aortic diameter at end-diastole, Ps is the systolic BP, Pd is the diastolic BP, and ln is the natural logarithm.

2.4. Tissue Doppler Imaging

Ascending aortic upper-wall velocities were measured by TDI at the same point as in the M-mode measurement (Figure 2). Gain and filter settings were adjusted to optimize the image. The TDI of expansion peak velocity during systole (SAo) and early (EAo) and late (AAo) contraction peak velocities during diastole were obtained with a 1-mm sample volume size. The resulting velocities were recorded for 3 cardiac cycles and stored for later analysis. The averages of velocities measured from the lateral mitral annulus on the transthoracic four-chamber views were reported as Sa (Peak longitudinal systolic velocities), Ea (early diastolic velocities), Aa (late diastolic velocities) (Figure 3).

2.5. Coronary Angiography

Study participants were all subjected to coronary angiography, which was performed by the percutaneous radial or femoral approach. Significant coronary stenosis disease (CAD) was defined as ≥70% reduction in the coronary lumen area.

2.6. Statistical Analysis

All continuous variables were expressed as mean ± standard deviation, and the Student t test was used to compare the differences between groups. Categorical variables are expressed as frequencies and percentages and were compared using the chi-squared test or Fisher's exact test. Multiple stepwise logistic regression was performed to assess the influence of baseline characteristics on TDI of ascending aortic upper-wall velocities, and stiffness parameters. For the optimal predictors, receiver operating characteristic (ROC) curves were constructed, and the area under the curve (AUC) was calculated. Subsequently, the optimal cut-off value of the predictors with the highest sensitivity and specificity was selected using ROC curves. The relationships between parameters were evaluated by Spearman's rank correlation analysis or linear regression analysis. Inter- and intra-observer reproducibility for measurement of the aortic velocities by TDI method was examined in 20 randomly selected cases with the Lin's concordance correlation coefficient. Statistical charts were prepared by GraphPad Prism9.0. A two-tailed P<0.05 was considered statistically significant.

2.7. Ethics

The study was approved by the institutional review board.

3. Results

The characteristics of the study population are shown in (Table 1). Of 118 patients enrolled in the study, 57 were categorized as having significant CAD with coronary area stenosis ≥70% (CAD Group), whereas 61 had non-significant CAD (non-CAD Group). There were no significant differences between the groups regarding age, gender, BMI, and coronary risk factors including diabetes mellitus, hypertension, hypercholesterolemia, and smoking status. The incidence of ST-segment elevation (STEMI) in the CAD group was similar to that in the non-CAD group (P=0.118). In CAD Group, 47 (82.4%), 9 (15.8%), and 1 (1.8%) had 1, 2, and 3 significantly stenotic coronary vessels, respectively, as confirmed by coronary angiography. The most common occurrence of stenosis was in the left anterior descending artery, up to 56 cases.

Echocardiographic measurements in study participants are depicted in (Table 2). Notably, no significant differences regarding LVEF or left ventricular diastolic diameter (LVDD) observed between the CAD Group and non-CAD Group. In terms of M-mode–derived indices of aortic stiffness, β and Ep were lower, and D was higher, in CAD Group (Table 2). Measurement of ascending aorta pulse wave TDI parameters was performed successfully in all study participants. Systolic velocity of ascending aorta (SAo) was significantly higher in the non-CAD group (11.70 ±1.53 cm/s vs.12.80 ±2.21 cm/s, p < 0.05) (Figure 4). Early diastolic (EAo) and late diastolic (AAo) velocities of ascending aorta were similar in the non-CAD and CAD groups (P=0.140 & P=0.179). Area under the ROC curve of SAo for diagnosing significant CAD was 0.64 (95% confidence interval [CI; 0.54–0.74]; P<0.001), which determined the diagnostic performance of SAo (Figure 5). According to the analysis of the ROC curve, the optimal cut-off value of SAo was ≥13.35cm/s with a sensitivity of 85.96% and a specificity of 40.98%.

There was a significant correlation between SAo velocity and β (r=−0.34, p<0.05), D (r=0.32, p<0.05), and Ep (r =−0.29, p<0.05). There was no correlation between EAo, AAo, and any of the aforementioned parameters of aortic stiffness.

A reasonable reproducibility for measurement of ascending aorta TDI parameters with a small bias was shown by the Lin's concordance correlation coefficient. Concordance correlation coefficients were 0.91 (95% CI 0.823–0.945) for SAo, 0.92 (95% CI, 0.883–0.956) for EAo, and 0.89 (95% CI 0.847–0.942) for AAo within the same observer. Concordance correlation coefficients were 0.94 (95% CI 0.927–0.951) for SAo, 0.93 (95% CI, 0.914–0.946) for EAo, and 0.90 (95% CI 0.848–0.939) for AAo between the 2 observers.

4. Discussion

Our study has three main clinical implications: (1) ascending aorta pulse wave TDI parameters had a high feasibility and satisfactory reproducibility; (2) S Systolic velocity of ascend-

ing aorta (SAo) predicted non-significant CAD with high sensitivity (85.96%) in patients with suspected SAP; (3) Because it was a retrospective study and the sample size of the CAD group is small, its poor specificity needs further verification.

The aorta is the most commonly used vessel for measuring local arterial stiffness because it contributes the most to the arterial buffering function [9]. Many studies have attempted to present that cardiovascular mortality is associated with aortic stiffness [10]. Several mechanisms may explain the correlation between aortic stiffness and coronary heart disease. As aortic stiffness raises, central systolic blood pressure increases and diastolic blood pressure decreases [11]. Elevated systolic blood pressure increases myocardial oxygen demand and ventricular load, which induces LV hypertrophy [12]. Decreased diastolic blood pressure reduces coronary perfusion, leading to subendocardial ischemia [13]. Subsequently, raised pulse pressure may lead to arterial wall remodeling, increased intimal wall thickness, and plaque formation [14].

Angiographic measurement of aorta stiffness through catheter conceived to simultaneously record pulse waves on 2 separate arterial sites is used as a direct but invasive gold standard method [15]. Several noninvasive techniques have been validated for studying arterial stiffness, most of which have been shown to predict cardiovascular events. Of these techniques, aortic stiffness assessed using carotid femoral pulse wave velocity (PWV) appears to have the greatest evidence for predicting cardiovascular mortality [16]. PWV can noninvasively assess aortic stiffness by measuring carotid-femoral pulse wave velocity. The most common limitation of PWV is that femoral artery pressure waveforms may be inaccurate in patients with obesity, metabolic syndrome, diabetes, and peripheral arterial disease. Secondly, PWV is estimated indirectly through various parameters, rather than directly measuring the aorta [17]. Elastic modulus, distensibility, and stiffness index β have been used to assess local aortic stiffness. However, since these methods rely on the blood pressure of the subjects, their reproducibility is limited [18]. Magnetic resonance imaging (MRI) is also used to measure aortic stiffness. Compared with other non-invasive methods, the most important advantage of MRI is the accuracy of anatomical location of arterial stiffness measurement. However, MRI has the disadvantages of complex operation, time-consuming and high cost [19].

Recently, it has been suggested to use TDI of the ascending aorta to evaluate aortic elasticity. Compared with other methods, TDI can directly measure the velocity of the ascending aorta quickly, noninvasively and simply, and can reduce medical expenses. Meanwhile, TDI can easily be implemented in the conventional echocardiography [8]. TDI of the ascending aorta is used to assess its elastic properties. It can measure aortic wall motion velocities, during systole and diastole, and correlates these measures with aortic distensibility and compliance [20]. These velocity amplitudes demonstrate the pressure-independent stiffness index that represents the elastic properties of the aorta [21].

Our study provides data that enable researchers to evaluate the elastic properties of the arterial wall directly, and show a high specificity and good reproducibility. Based on the ROC curve of SAo for diagnosing significant CAD was 0.64 (95% confidence interval [CI; 0.54–0.74]; $P<0.05$) and the optimal cut-off value of SAo≥13.35cm/s with a sensitivity of 85.96% and a specificity of 40.98%. Further analysis showed that SAo had a significant association with Elastic modulus, distensibility, and stiffness index β. Our study demonstrated that the availability of TDI is closely related to non-CAD and the high sensitivity of SAO can accurately identify patients without significant CAD, thus avoiding unnecessary CAG examination. Especially for grassroots hospitals, it can reduce unnecessary hospitalizations, improve transfer efficiency, and save medical resources.

5. Conclusions

Arterial stiffness is lower in patients without significant CAD. Measuring SAo of the anterior ascending aorta using TDI echocardiography has good sensitivity but poor specificity in patients without significant coronary artery stenosis in SAP.

Table 1. *Baseline Characteristics of the Study Population.*

	CAD	non-CAD	P value
n	57	61	
age	65.72 ±6.99	63.52 ±6.30	0.075
male	39	32	0.077
Hypertension, n	28	34	0.789
Smoker (current), n	26	20	0.239
Hypercholesterolemia, n	22	18	0.365
Diabetes mellitus, n	24	19	0.299
STEMI, n	29	20	0.118

	CAD	non-CAD	P value
Heart rate (Bpm)	75.47±6.25	75.21±4.98	0.802
SBP, mmHg	127.12±14.50	123.95±13.69	0224
DBP, mmHg	80.23±8.94	79.41±8.90	0.619
PP, mmHg	46.89±15.53	44.54±16.48	0.428
BMI, kg/m^2	25.54±1.32	25.21±0.90	0.113
LDL, mmol/L	2.85±0.38	2.79±0.37	0.387
HDL, mmol/L	1.19±0.10	1.21±0.09	0.255
ACEI/ARB, n	17	14	0.453
β blocker, n	9	10	0.763
CCB, n	11	7	0.424

Continuous data are presented as mean SD and categorical data as number. ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; CAD, coronary artery disease; HDL, high-density lipoprotein; STEMI, ST segment elevation myocardial infarction; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.

Table 2. *Conventional echocardiographic parameters, Ascending aortic stiffness parameters and Aortic TDI parameters of the study population.*

	CAD	non-CAD	P value
Conventional echocardiographic parameters			
LVDD (mm)	49.41±2.32	48.85±2.03	0.165
LVEF (%)	63.11±3.74	64.41±3.88	0.067
As (cm)	3.23±0.31	3.13±0.25	0.056
Ad (cm)	3.00±0.11	2.98±0.08	0.259
E (m/s)	0.70±0.14	0.73±0.17	0.299
A (m/s)	0.76±0.10	0.75±0.13	0.642
Ascending aortic stiffness parameters			
D ($cm^2dyne^{-1}10^{-6}$)	4.48±0.49	4.69±0.61	0.042
β	5.84±0.82	6.23±0.80	<0.01
Ep (kpa)	64.72±3.02	63.39±2.78	<0.01
Aortic TDI parameters			
Ea (cm/s)	11.25±2.77	12.28±3.08	0.059
Aa (cm/s)	10.44±0.75	10.32±0.78	0.37
Sao (cm/s)	11.70±1.53	12.80±2.21	<0.01
EAo (cm/s)	9.69±2.03	10.31±2.46	0.140
AAo (cm/s)	12.88±2.36	12.19±3.10	0.179

A, mitral inflow late diastolic velocity; Aa, annular late diastolic velocity; AAo, aortic wall late diastolic velocity; E, mitral inflow early diastolic velocity; Ea, mitral annular early diastolic velocity; EAo, aortic wall early diastolic velocity; LVEF, left ventricular ejection fraction; LVDD, left ventricular diastolic diameter; Sa, systolic myocardial velocity; SAo, aortic wall systolic velocity; Ad, diastolic ascending aortic diameter; As, systolic ascending aortic diameter; D, aortic distensibility; Ep, pressure-strain elastic modulus; β, stiffness index; TDI, tissue Doppler imaging.

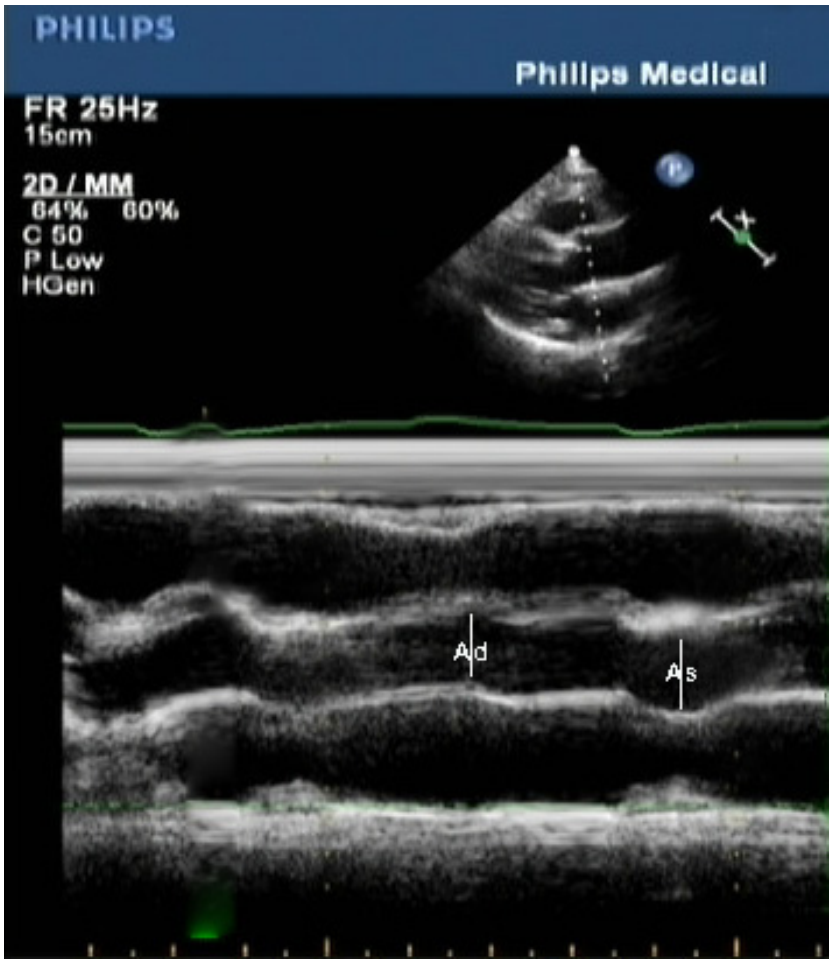

Figure 1. *M-mode echocardiography recordings of the ascending aorta in parasternal long-axis view showing the measurement of systolic aortic diameter (As), and diastolic aortic diameter (Ad). The measurements were made at the same point at a level 3 cm above the aortic cusps.*

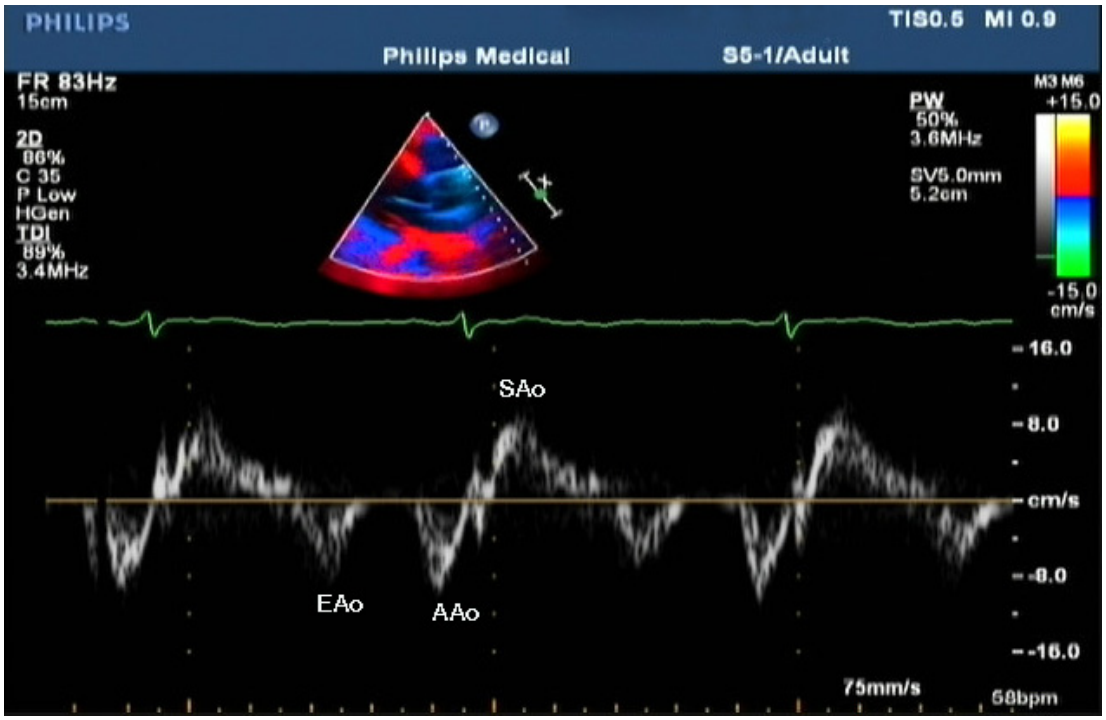

Figure 2. *Ascending aortic upper-wall velocities were measured by TDI at a level 3 cm above the aortic cusps in long axis from the parasternal view. SAo: peak velocity during systole. EAo: early diastolic peak velocities. AAo: late contraction peak velocities.*

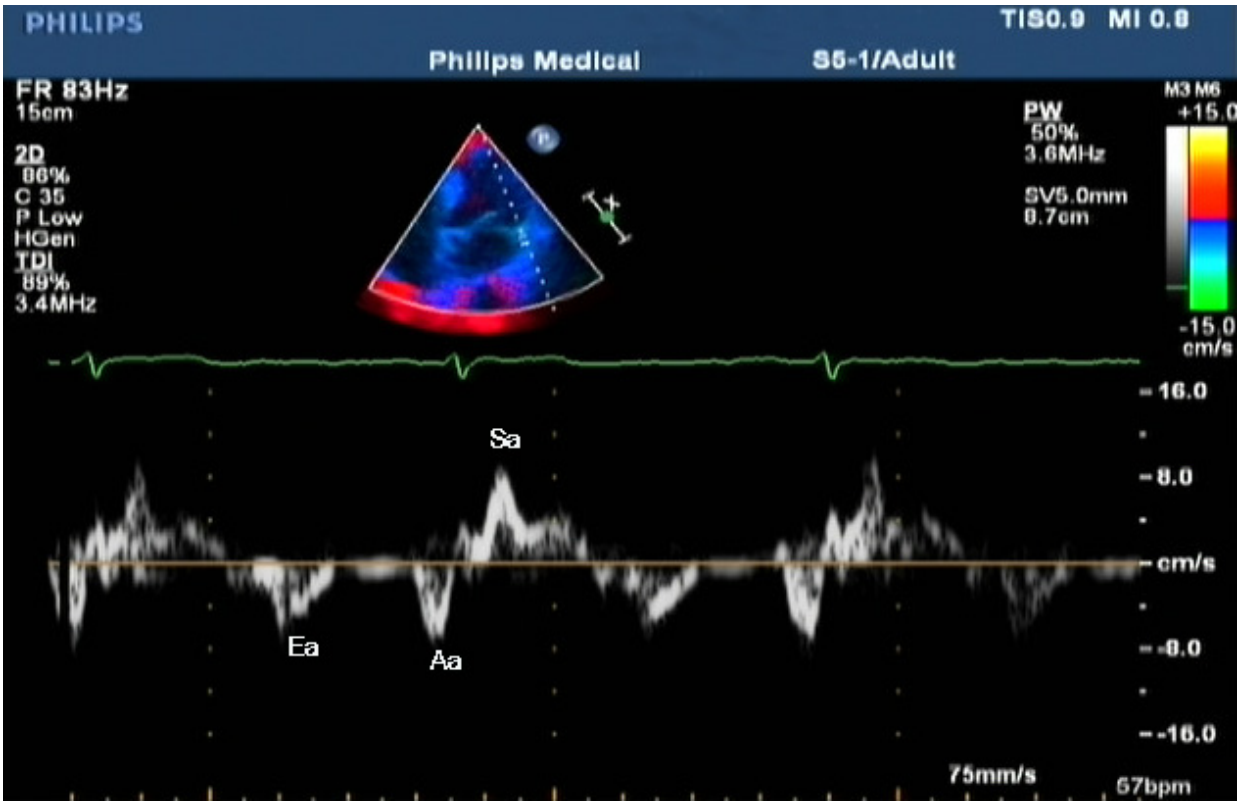

Figure 3. *The averages of velocities measured from the lateral mitral annulus on the transthoracic four-chamber views were reported as Sa (Peak longitudinal systolic velocities), Ea (early diastolic velocities), Aa (late diastolic velocities).*

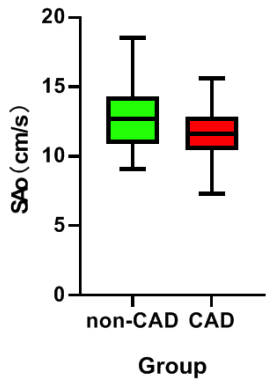

Figure 4. *SAo: Systolic velocity of ascending aorta.*

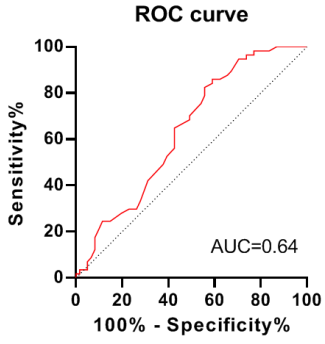

Figure 5. *ROC curve: Receiver operating characteristic curve. AUC.*

6. Limitations

First, because of the sampling location of ascending aorta, image quality may have been affected by breathing and adjacent structures. Aortic TDI parameters were successfully measured in all study participants. However, image quality may affect the accuracy of these measurements. Second, the accuracy of TDI measurement is angle dependent, and it is a semi-automated technology, which depends on the experience of the operator. Finally, the sample size of patients enrollment in the study was small, and selection bias could not be completely precluded, the above reasons may have led to the poor specificity of this study. Further validation and prognostic studies on the sensitivity of SAO are warranted.

Abbreviations

CAD: Coronary Artery Disease
SAP: Stable Angina Pectoris
TDI: Tissue Doppler Imaging
ECG: Electrocardiogram
CAG: Coronary Angiography
SAo: Aortic Systolic Velocity
EAo: Early Diastolic Velocity
AAo: Late Diastolic Velocity
β: Aortic Stiffness Index
D: Aortic Distensibility

Ep: Pressure-strain Elastic Modulus
ROC: Receiver Operating Characteristic Curve
AUC: Area Under Curve
ACEI: Angiotensin-Converting Enzyme Inhibitor
ARB: Angiotensin Receptor Blocker
BMI: Body Mass Index
HDL: High-density Lipoprotein
STEMI: ST Segment Elevation Myocardial Infarction
SBP: Systolic Blood Pressure
DBP: Diastolic Blood Pressure
PP: Pulse Pressure
E: Mitral Inflow Early Diastolic Velocity
Ea: Mitral Annular Early Diastolic Velocity
A: Mitral Inflow Late Diastolic Velocity
Aa: Annular Late Diastolic Velocity
LVEF: Left Ventricular Ejection Fraction
LVDD: Left Ventricular Diastolic Diameter
Sa: Systolic Myocardial Velocity
ACEI: Angiotensin-Converting Enzyme Inhibitor
ARB: Angiotensin Receptor Blocker

Ethical Statements

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1964 and later versions. Informed consent was obtained from the patient for publication of this case report.

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Author Contributions

Ling Wang: Data curation, Formal Analysis, Funding acquisition, Writing - original draft, Writing - review & editing

XiangYu Chen: Conceptualization, Data curation, Formal Analysis, Writing - original draft, Writing - review & editing

Feng Yang: Conceptualization, Data curation, Software, Writing - review & editing

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Conflict of Interest

The authors declare no conflicts of interest.

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