

Aortic Propagation Velocity in Patients with Coronary Artery Disease

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Abstract

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Background: The aortic stiffness is the strongest cardiovascular predictor of morbidity & mortality; it is widely established that cardiovascular hazard factors cause alterations in the histology and function of the aorta. **Aim:** This research set out to assess the frequency with which Aortic propagation velocity (APV) was seen in coronary artery disease patients (CAD). **Methods:** This comparative cross-sectional research was conducted at Mansoura specialized hospital from January 2022 to June 2022. One hundred participants participated in this study. The cases were recommended for optional coronary angiography and separated into a CAD group (n= 57) and a non-CAD group (n= 43). **Results:** Aortic strain (%) 6.10 ± 1.430 versus 11.77 ± 2.809 , $P < 0.0001$), Aortic distensibility ($\text{cm}^2/\text{dyn} \cdot 110^{-3}$) 2.85 ± 0.684 versus 5.35 ± 1.312 , $P < 0.0001$), APV 43.74 ± 10.719 versus 80.18 ± 21.095 , $P < 0.0001$) respectively. APV is a major indicator of the occurrence of CAD ($P < 0.001$). There is statistically significant negative correlation among APV and SYNTAX score ($r = -0.657$; $P < 0.001$). APV is a highly significant predictor of CAD with high sensitivity (82.5%), specificity (97.7%), PPV (97.9%), NPV (80.8%), accuracy (89.0%), and Youden's J index (0.802). The AUC of 0.958 with a 95% CI of 0.925-0.990 ($P < 0.001$). **Conclusion:** Coronary artery disease is substantially predicted by the velocity of aortic propagation.

Keywords: Arterial stiffness, Aortic propagation velocity, Echocardiography, Coronary artery disease.

Introduction

Improvements in percutaneous coronary intervention (PCI) and coronary artery bypass graft surgery (CABG) have made great strides in the treatment of cardiovascular illness, and with this large

increase, the avoidance of its consequences is now a primary priority of healthcare.^{1,2} Changes in the aorta's structure and function due to CVD risk factors are well established.^{3,4} One of the primary focuses of cardiology

is preventing atherosclerosis and its side effects. Increased arterial resistance is a direct result of atherosclerosis, which causes the venous wall to become broader and stiffer. Lower flow propagation velocities inside the arterial lumen are the direct outcome of increased arterial resistance.⁵

Prioritizing care for those with coronary artery disease based on their risk levels is essential, therefore accurate risk assessment is crucial. Clinical, electrocardiographic, 2-D echocardiographic, and biochemistry markers all contribute to risk prediction. Echocardiography is a widely available, accurate, straightforward, and cost-effective method for evaluating arterial stiffness in a non-invasive manner.⁶

Echocardiographic indices such Aortic strain (AS), aortic distensibility (AD), pulse pressure, augmentation index, and pulse wave propagation velocity are all variables to consider. have been proposed for use in determining aortic stiffness.⁷

Aortic velocity propagation (AVP) in the descending thoracic aorta is inversely related to coronary artery disease^(5,7).

The primary purpose of this research is to evaluate APV in adults with CAD.

Patients and Methods

A comparative cross-sectional study was conducted at Mansoura specialized hospital from January 2022 to June 2022. This research was done on A random sample of 100 coronary artery disease group in which there is narrowing of coronary arteries more than 50 percent who have been referred for

elective coronary angiography. Participant were split into CAD group (n= 57) and a non-CAD group (n= 43).

Patients with the following criteria are excluded: Substantial valvular heart disease, known systemic disease affecting aorta (Marfan syndrome, etc.) aortic dilation more than forty mm, chronic renal failure (CRF: eGFR< sixty ml/min/1.73m²)⁸, history of myocardial infarction, LVEF< fifty-five percent, arrhythmia (Each type of ventricular and atrial arrhythmia was eliminated from the research, and none of the patients had APCs or VPCs.), bundle branch block (Left and right bundle branch block), Insufficient suprasternal images and reluctance to participate in the study.

Methods

All studied patients were subjected to: **History taking** (Name, Age, Sex and Risk factors). **Investigations: ECG, ECHO** (Left ventricular ejection fraction, End-systolic and line service diameters, septal & posterior LV diameter, aortic propagation frequency, aortic strain & contractility using ascending aortic systolic and diastolic diameters). **Coronary angiography:** Patients were placed in one of many categories based on the outcome of coronary angiography: 2 groups, first group are patients with normal coronary angiography & second group are patients with CAD, Gensini and Syntax score (SS)⁹⁻¹² indicated the amount and severity of CAD in the CAD group.

The Procedure of Coronary Angiography with the Artis zed cardiac angiography equipment, conventional Judkins or Tiger

catheters have been inserted into the femoral or radial artery to perform a coronary angiogram in a modified Seldinger procedure (Siemens, Munich, Germany). Coronary angiograms were investigated in two orthogonal views & were labeled substantial if a stenosis of more than 50% of such artery's diameter was present and non-significant otherwise.¹³

Speed of aortic wave propagation, Multichannel color mode M Doppler tracings were made using the cursor through a suprasternal window aligned perpendicular to the direction of blood flow in the lumen of the descending aorta. Adjustments were made such that the aliasing velocity is now about thirty and fifty cm/s. When flipping to M-mode tracing, a flaming M-mode color picture was shown. The speed of sound in the aorta was determined by following the initial aliasing contour and measuring its velocity slope.

Ethical Approval: Each participant in the research provided written informed permission, which was obtained after the project was given the green light by the university's ethics committee code no.R.22.01.1605. While conducting this human study, the World Medical Association's Declaration of Helsinki, its code of ethics, was adhered to.

Statistical analysis

SPSS (Statistical Program for the Social Sciences for Windows, rendition 16.0, Chicago, Iowa, USA) was recently used to enter and analyse all data. We presented qualitative parameters as numbers and counts, and quantitative ones as well as

mean & SD. A significant P-Value was defined as one that was less than 0.05, and thus the appropriate analytical test was applied.

Results

Mean, standard deviation, percentage, and frequency belong to the common ways that data is expressed. Confidence interval of the mean difference between the two groups, expressed as a percentage. If P is less than.05.

Table (1) reveals no discernible distinctions in Age, Gender, etc., between the two groups, BMI (kg/m²), Medical history, Positive familial history and Vital signs. There is statistically significant increase in SYNTAX score in CAD group as opposed to Non-CAD group. Number of involved vessels were 1 in 22 CAD patient (38.6%), 2 in 20 CAD patient (35.1%) and 3 in 15 CAD patient (26.3%) (Table 1).

Statistics are often exposed as means, standard deviations, percentages, and frequencies. Confidence interval for the difference in means between the two groups, expressed as a percentage (95% CI). If P value is less than 0.05, then the result is statistically significant.

Table (2) demonstrates that there is no statistically significant mixture of two groups. regarding EF (%), LVEDD (cm), LVESD (cm), PWT (cm), Septal thickness (cm), Diastolic diameter (cm), Systolic diameter (cm). There is statistically significant decrease in Aortic strain (%), Aortic distensibility (cm²dyn⁻¹10⁻³) and Aortic propagation velocity in CAD group versus non-CAD group (Table 1).

Table (3) shows statistically significant negative correlation between the velocity of aortic propagation and SYNTAX score (Table 3).

Table (4) shows that aortic propagation velocity is a significant predictor of CAD occurrence (Table 4).

Table (5) and figure (1) show that the APV is a highly significant predictor of CAD with high sensitivity (82.5%), specificity (97.7%), PPV (97.9%), NPV (80.8%), accuracy (89.0%), and Youden's J index (0.802). The AUC of 0.958 with a 95% CI of 0.925-0.990 and P-value less than 0.001 (Table 5).

Table 2: Patient clinical and demographic characteristics with and without CAD were compared.

| | | CAD group (n=57) | Non-CAD group (n=43) | 95% CI | P |
|-----------------------------------|----------------------------------|-----------------------------|---------------------------------|---------------|----------|
| | Age (years) | 56.28 ± 8.351 | 55.98 ± 9.208 | -3.19, 3.80 | 0.863 |
| Gender | Male | 33 (57.9%) | 31 (72.1%) | - | 0.143 |
| | Female | 24 (42.1%) | 12 (27.9%) | - | |
| | BMI (kg/m²) | 26.59 ± 3.048 | 27.34 ± 2.890 | -1.94, 0.45 | 0.220 |
| Medical history | DM | 24 (42.1%) | 14 (32.6%) | - | 0.330 |
| | HTN | 19 (33.3%) | 17 (39.5%) | - | 0.552 |
| | Smoking | 15 (26.3%) | 10 (23.3%) | - | 0.726 |
| | Hyperlipidemia | 17 (29.8%) | 12 (27.9%) | - | 0.834 |
| | Positive familial history | 22 (38.6%) | 9 (20.9%) | - | 0.059 |
| Vital signs | Heart rate | 79.12 ± 13.565 | 78.81 ± 13.816 | -5.17, 5.79 | 0.911 |
| | SBP (mmHg) | 128.30 ± 12.560 | 131.91 ± 13.872 | -8.87, 1.66 | 0.177 |
| | DBP (mmHg) | 86.98 ± 13.966 | 91.07 ± 15.911 | -10.03, 1.86 | 0.176 |
| | PP (mmHg) | 41.32 ± 6.179 | 40.84 ± 5.516 | -1.89, 2.85 | 0.689 |
| | SYNTAX score | 14.14 ± 6.531 | 0.98 ± 0.771 | 11.17, 15.15 | 0.000 |
| Number of involved vessels | 1 | 22 (38.6%) | | | |
| | 2 | 20 (35.1%) | - | - | - |
| | 3 | 15 (26.3%) | | | |

Table 2: Findings on echocardiography in CAD and non-CAD groups

| | CAD group (n=57) | Non-CAD group (n=43) | 95% CI | P |
|--|-----------------------------|---------------------------------|---------------|-------------------|
| EF (%) | 58.40 ± 5.882 | 57.19 ± 6.631 | -1.27, 3.71 | 0.334 |
| LVEDD (cm) | 4.92 ± 0.282 | 4.84 ± 0.335 | -0.04, 0.20 | 0.196 |
| LVEDD (cm) | 3.05 ± 0.410 | 3.14 ± 0.476 | -0.27, 0.09 | 0.314 |
| PWT (cm) | 0.80 ± 0.087 | 0.80 ± 0.077 | -0.03, 0.03 | 0.936 |
| Septal thickness (cm) | 0.90 ± 0.120 | 0.89 ± 0.114 | -0.04, 0.06 | 0.704 |
| Diastolic diameter (cm) | 3.15 ± 0.271 | 3.25 ± 0.255 | -0.20, 0.01 | 0.084 |
| Systolic diameter (cm) | 3.35 ± 0.283 | 3.44 ± 0.260 | -0.20, 0.01 | 0.099 |
| Aortic strain (%) | 6.10 ± 1.430 | 11.77 ± 2.809 | -6.52, -4.81 | < 0.001 |
| Aortic distensibility (cm 2dyn-110-3) | 2.85 ± 0.684 | 5.35 ± 1.312 | -2.90, -2.09 | < 0.001 |
| Aortic propagation velocity | 43.74 ± 10.719 | 80.18 ± 21.095 | -42, -30 | < 0.001 |

Table 3 Relationship between Aortic propagation velocity and SYNTAX score in the current study

| | Correlation coefficient | P |
|---------------------|--------------------------------|-------------------|
| SYNTAX score | -0.657 | < 0.001 |

P is significant when < 0.05.

This table shows statistically significant negative correlation between the velocity of aortic propagation and SYNTAX score (Table 3).

Table 4: Logistic regression analysis for Aortic propagation velocity in prediction of occurrence of CAD

| | R2 | B | SE | Wald | Exp(B) | 95% CI | Constant | P |
|------------------------------------|-----------|----------|-----------|-------------|---------------|---------------|-----------------|-------------------|
| Aortic propagation velocity | 77.5% | -0.203 | 0.05 | 19.81 | 0.816 | 0.747, 0.893 | 11.92 | < 0.001 |

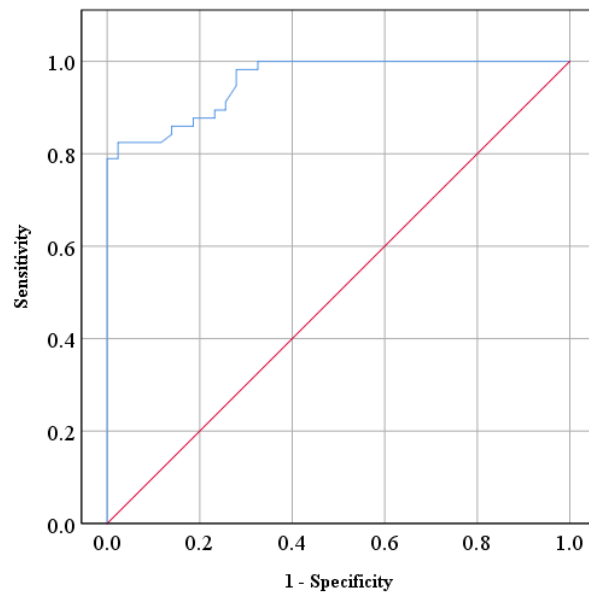
P is significant when < 0.05.

This table shows that aortic propagation velocity is a significant predictor of CAD occurrence (Table 4).

Table 5: Diagnostic profile of APV in predicting CAD

| | Aortic propagation velocity |
|-------------------------|------------------------------------|
| AUC | 0.958 |
| 95% CI | 0.925, 0.990 |
| P | < 0.001 |
| Cut off point | 53.6 |
| Youden's J index | 0.802 |
| Sensitivity | 82.5% |
| Specificity | 97.7% |
| PPV | 97.9% |
| NPV | 80.8% |
| Accuracy | 89.0% |

P is significant when < 0.05 .

**Figure 1:** ROC curve of APV in predicting CAD.

Discussion

Aortic stiffness is connected with cardiovascular hazard factors such as smoking, obesity, hypertension, glucose tolerance, diabetes, and older age¹⁴⁻¹⁶. The breadth and severity of atherosclerosis affects the progression of AD and AS. Atherosclerosis thickens the artery wall and stiffens the aorta¹⁷. Tunica media thickens and stiffens as atherosclerosis advances. As a result, adopting a noninvasive approach to detect atherosclerotic disease before it reveals itself is particularly beneficial. Endothelial dysfunction is the 1st stage of atherosclerosis. The arterial resistance increases when the arterial wall stiffens & thickens. Increasing vascular resistance decreases flow and APV¹⁸.

Based on our research, we found the 2 groups had no discernible difference statistically in terms of Age, Gender, BMI (kg/m²), Medical history, Positive familial history and Vital signs.

A study conducted by¹⁸ found no substantial variance in the mean age of presentation among the 2 groups (Group A with significant CAD was 54 10.8 years & Group B with normal epicardial coronaries was 51.1 7.37 years, [t = 1.56, P = 0.122]). However, they reported that Group A had a significantly higher body mass index (BMI) than Group B (P 0.05). BMI is linked to moderate adiposity, which may contribute to the development of atherosclerosis. Therefore, it is not surprising that the BMI is significantly higher in CAD patients. Similarly, A study conducted before¹⁹ also presented that there were no statistically

substantial variances in the ages of the study groups.

In the present study, there is statistically significant increase in SYNTAX score in CAD group compared to Non-CAD group. Similar findings were reported by⁵.

The SYNTAX score measures the complexity, grade, and severity of atherosclerosis feature values of atherosclerotic lesions and coronary artery structure. Mortality and significant unfavorable acute and long-term cardiovascular events can be predicted independently by SS, according to the books²⁰⁻²². Predicting a high SS in SAP patients prior to CAG is crucial for selecting the most effective therapy and stopping damaging cardiovascular consequences.^{11,23}

In the current study, Number of involved vessels were 1 in 22 CAD patient (38.6%), 2 in 20 CAD patient (35.1%) and 3 in 15 CAD patient (26.3%).

A study conducted previously¹⁸ reported that multivessel disease involving significant lesions (double-vessel and triple-vessel diseases) were more prevalent in cases with CAD group.

In our research, we found statistically significant decrease in Aortic strain (%), Aortic distensibility (cm²dyn⁻¹10⁻³) and Aortic propagation velocity in CAD group matched to Non-CAD group.

In deal with our findings, a study conducted lately⁶ results showed that the APV of those with CAD is much lower than those without

CAD (48.63 ± 10.31 cm/s vs. 77.75 ± 9.97 cm/s). This agrees with similar research.

Patients with significant risk factors over all previous decade had a lower APV level (37.13 cm/s), as shown by research by ²⁴.

Reduced APV (29.9 ± 8.1 cm/s) was seen in CAD patients compared to the proper coronary group (47.6 ± 16.8 cm/s) ²⁵. This is consistent with the current findings, and suggests that declining APV is associated with potential risk factors and chronicity.

A study conducted earlier ¹⁹ similarly found that APV was lower in CAD patients (39.2 - 13.9 cm/s) compared to matched non-CAD controls (81.4- 21.4 cm/s).

Consistent with the present findings, a significantly lower APV in the CAD (41.65 ± 4.94 cm/s) group than in the non-CAD (49.72 ± 6.38 cm/s) group was found ⁵

These results are consistent with the hypothesis that APV contributes to atherosclerotic events and arterial stiffness ^{26,27}. Noting that the 'flow propagation of velocity' is not an accurate estimation of fluid propagation among the base and apex, a correlation exists between maximally detected velocity points. These consequences might be indicative of various fluid components that can be simultaneously stimulated by local pressure gradients at various depths in front of the inflow channel. APV will decrease as arterial stiffness increases; this can serve as an early indicator of arterial stiffness without symptoms. ^{6,28}. Here, we report the discovery of a statistically strong inverse association between aortic propagation velocity and SYNTAX score.

Our results were consistent with other study ⁵ which reported that SYNTAX score is inversely related to APV. This association may be quite useful in representing the severity of CAD before medical therapy is administered.

Also, Our findings coincide with those of others ²⁹ who reported that high SYNTAX scores (SS) Subjects compared to low SS had lower APV. [39.0 (32.0-51.7) vs. 55.0 (45.0-62.0) cm/s, roughly.

The project focuses, Aortic propagation velocity was discovered to be a compelling predictor of the incidence of CAD in a logistic regression research (P 0.001). Similarly, the results of a logistic regression analysis revealed that APV was the only substantial indicator of the occurrence of CAD ⁶. (P < 0.001).

This investigation revealed that the APV is a highly significant predictor of CAD with high sensitivity (82.5%), specificity (97.7%), PPV (97.9%), NPV (80.8%), accuracy (89.0%), and Youden's J index (0.802). The AUC of 0.958 with a 95% CI of 0.925-0.990 and P-value less than 0.001.

Similarly, based on results from study ⁶ from the findings, an APV value below 56 cm/sec may be utilized to predict CAD with a 96% particularity & sensitivity and 79% (P 0.001, AUC = 0.972).

A threshold this same worth of 46.5 cm/s was reported before ²⁴ with sensitive as well as specific of 84% & 86%, respectively, for APV.

The sensitivity and specificity of APV for predicting CAD were 76% & 72%, respectively, as shown previously ⁵.

Also, a study demonstrated that a cut off velocity of 60.5 cm/s for APV was associated with a sensitivity and specificity for prediction CAD of 90.5 & 92.2%, respectively¹⁹.

Conclusion

Our study demonstrated that patients who are suffering from coronary artery disease exhibit a statistically decline steadily aortic propagation velocity compared to those without coronary artery disease. Moreover, our findings suggest that aortic propagation velocity represents a significant predictor of the occurrence cardiovascular disease. These findings highlight the potential clinical relevance of aortic function in the pathogenesis and progression of CAD & suggest that monitoring aortic propagation velocity may be a valuable tool for identifying cases at high risk for CAD. Further investigation is warranted to gain a better seeing the connection between aortic function and coronary artery disease, & also to explore the possible clinical implications of our findings.

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