

The Importance of Arterial Stiffness Parameters for Predicting Syntax Score in Acute Coronary Syndrome

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Abstract. Background: Numerous studies have demonstrated a connection between heightened arterial stiffness (AS) and cardiovascular disease. Over time, several techniques have been devised to gauge arterial stiffness. **Objective:** This study aims to investigate the correlation between AS, measured using the PWV method on the arm, and the SYNTAX (Synergy between PCI with Taxus and Cardiac Surgery) score derived from coronary angiography (CAG). **Materials and Methods:** The cohort comprised 53 patients (51.0%) diagnosed with non-ST elevation myocardial infarction (NSTEMI) and 51 patients (49.0%) diagnosed with ST elevation myocardial infarction (STEMI), all aged between 18 and 80 years. Using the 'Mobil-O-Graph® ARCSolver algorithm' device, we measured parameters such as PWV, augmentation index (AIx), and arterial blood pressure for all participants. The SYNTAX score was employed to gauge the severity and extent of coronary artery disease. **Results:** The patients' average age stood at 61.36 years, with 61 (60.6%) being male and 43 (39.4%) female. The mean BMI was recorded at 28.43 kg/m². Upon comparing the patient groups based on PWV measurements, intriguing insights emerged. Specifically, within the NSTEMI group, a noteworthy positive correlation emerged between PWV and key factors such as age, RDW (Red Cell Distribution Width), blood urea nitrogen (BUN) levels, and SYNTAX score ($p < 0.05$). **Conclusion:** The elevation of PWV levels proved to be notably valuable in anticipating the severity of coronary artery disease in NSTEMI patients. Conversely, in STEMI patients, heightened PWV emerged as a predictor of an unfavorable prognosis, aligned with higher clinical risk scores.

Keywords: coronary artery disease, myocardial infarction, arterial stiffness, SYNTAX, GRACE.

INTRODUCTION

Cardiovascular diseases (CVD) stand as the foremost contributor to global morbidity and mortality [1]. Even with the swift advancements in treatment approaches and enhanced early revascularization techniques, rates of readmission, heart failure, and mortality remain elevated among individuals dealing with acute coronary syndrome (ACS) [2]. The execution of therapeutic strategies aimed at averting and ameliorating coronary artery disease during the initial phase plays a pivotal role in shaping the prognosis of this ailment.

Numerous studies have demonstrated the link between heightened arterial stiffness and the occurrence of significant cardiovascular events in individuals with coronary artery disease (CAD) [3]. This increased stiffness in the arteries is recognized as a risk factor that amplifies the overall cardiovascular risk, alongside factors like age, hypertension (HT), diabetes mellitus (DM), and chronic renal failure

(CRF). The escalation of arterial stiffness, coupled with diminished vascular elasticity, contributes to elevated systolic blood pressure levels. Moreover, this heightened pressure places extra strain on the left ventricle, inducing left ventricular hypertrophy, diminishing diastolic blood pressure, and impairing coronary blood flow [4].

There are several non-invasive techniques available for assessing arterial stiffness, including ultrasound, MR elastography, tonometry, and oscillometric devices. These methods gauge the arterial wall's response to pressure, indirectly providing the Pulse Wave Analysis (PWA) value. Among these measurements, the measurement of pulse wave velocity (PWV) stands out, as it can be easily obtained using pressure-sensitive transducers, Doppler ultrasound, and applanation tonometry. PWV is frequently employed in clinical research. Normalized Augmentation Deflection (NAD), on the other hand, is recognized as an independent cardiovascular risk marker. It showcases the severity

of arteriosclerosis and is correlated with the degree of aortic stiffness [5]. However, findings regarding the correlation between peripheral arterial PWV and the severity of coronary artery lesions have yielded diverse outcomes.

The objective of this study was to investigate the correlation between arterial stiffness parameters and the severity of coronary artery lesions in patients with ACS.

MATERIALS AND METHODS

PATIENT SELECTION

This prospective study involved 104 patients with Acute Myocardial Infarction (both STEMI and NSTEMI), admitted to the Coronary Intensive Care Unit at Tepecik Training and Research Hospital. The study encompassed patients aged between 18 and 80 years. Thorough explanations of the study were provided to all patients, and their participation was voluntary. Consent signatures were obtained from those who agreed to take part in the study. The study was initiated after obtaining approval from the Ethics Committee of İzmir Tepecik Training and Research Hospital (Date: 01.02.2016, Decision no: 22/1).

Sample size and power

We performed an a priori power analysis for the primary correlation of interest (PWV vs. SYNTAX) using Fisher's z transformation (two-tailed, $\alpha=0.05$). Detecting a moderate correlation of $r=0.35$ with 80% power would require **n=62** participants per group. With the available samples (NSTEMI $n=53$, STEMI $n=51$), the achieved powers to detect $r=0.35$ were approximately **0.75** and **0.72**, respectively; in the total cohort ($n=104$), the power was 0.88 to detect a correlation of $r=0.30$ (two-tailed, $\alpha=0.05$). These results indicate that the overall study is adequately powered for a moderate correlation. Although subgroup analyses remain underpowered to detect smaller effects, the analytic approach remains methodologically robust.

Clinical evaluation

After conducting a thorough medical history review and comprehensive physical examinations, we documented baseline characteristics including age, gender, HT, DM, current smoking status, family history of CAD, hyperlipidemia, history of

stroke, body weight, height, waist circumference, and ongoing treatments. Every patient underwent routine laboratory tests. All patients involved in the study received a comprehensive physical examination. Blood pressure measurements were taken using a properly calibrated mercury sphygmomanometer. The diagnosis of ACS was confirmed through symptoms, 12-lead ECG findings, and cardiac marker assessments. Electrocardiograms were used to differentiate between STEMI and NSTEMI. Patients with CRF (glomerular filtration rate < 30 ml/min), congenital heart disease, cardiogenic shock, previously diagnosed coronary artery disease, moderate-to-severe valvular disease, cancer, atrial fibrillation, active infections, and those who couldn't provide consent for participation were excluded from the study.

LABORATORY TESTS

We conducted a comprehensive analysis of several medical parameters, including complete blood count, fasting blood glucose, blood urea nitrogen (BUN), creatinine, serum sodium, potassium levels, HDL and LDL cholesterol, total cholesterol, and triglyceride (TG) levels. Additionally, we assessed levels of creatine kinase isoenzyme MB (CK-MB) and cardiac troponin I (cTnI). To estimate the glomerular filtration rate (e-GFR), we employed age, gender, BUN, and creatinine values from all patients. Furthermore, we evaluated the 12-lead electrocardiograms (ECGs) of the patients.

ECHOCARDIOGRAPHY

All patients underwent transthoracic echocardiography (ECHO) utilizing the GE Vivid 7® system and a 3.5 MHz probe. Echocardiographic assessments were conducted using various methods, including M-mode, two-dimensional imaging, color Doppler, pulse wave Doppler, and color Doppler, all while the patient was in a supine position or left lateral recumbency. By employing these techniques, we measured essential cardiac parameters, such as left ventricular end-systolic and end-diastolic dimensions, ejection fraction, end-diastolic septum and posterior wall thickness, as well as left atrial and aortic dimensions.

ACS TREATMENT

Patients who were admitted to the hospital with a diagnosis of acute coronary syndrome and displayed elevated cardiac markers (CK-MB, TnI)

beyond the upper reference limit during follow-up were identified as having experienced acute MI. The treatment for acute coronary syndrome adhered to the recommendations set forth by the European Society of Cardiology (ESC) [2]. Following the acquisition of consent, we conducted standard coronary angiography using a Philips Integris V5000 Netherland® coronary angiography system. The procedure involved a 6F Judkins diagnostic catheter, inserted through right femoral access after intraducer placement. Subsequently, a right-left selective coronary angiography was performed using the same 6F Judkins diagnostic catheter, opting against the use of nitroglycerin. Instead, a nonionic contrast agent was intracoronarily injected. Within the percutaneous coronary angiography, we scrutinized coronary artery lesions that caused luminal narrowing of over 50% in vessels exceeding 1.5 mm. Comprehensive records were maintained for all percutaneous coronary ballooning and stenting procedures.

CALCULATION OF THE SYNTAX SCORE

The coronary angiography (CAG) images of the patients were meticulously assessed by two seasoned interventional cardiologists. These experts, purposefully kept unaware of the patients' clinical details and the study's context, meticulously evaluated the images. Each lesion's score was individually computed based on the CAG images, culminating in the determination of the overall SYNTAX score using the SYNTAX score calculator v2.02, available at www.syntaxscore.com. Subsequently, the patients were categorized into three distinct groups: low risk (0–11), intermediate risk (11–18), and high risk (>18).

ARTERIAL STIFFNESS MEASUREMENT

Within the initial 24 hours of admission to the coronary intensive care unit, and in a controlled environment to mitigate external influences, two successive measurements were conducted. Mobil-O-Graph® employs a cuff-based approach to estimate carotid-femoral pulse wave velocity (cf-PWV) from a single-point pressure wave recording. Following the acquisition of systolic and diastolic blood pressure readings, the brachial cuff is inflated to the diastolic level and maintained for 10 seconds to capture pulse wave data. Utilizing a transfer function, central pressure curves are generated, and the ARCSolver algorithm (developed by the Austrian Institute of Technology, Vienna, Austria) is employed

for further processing [6,7]. Diverse parameters, ranging from pulse wave analysis to wave splitting analysis, contribute to a mathematical model that integrates age, central pressure, and aortic characteristic impedance [8,9]. These assessments encompassed PWV, augmentation index (AIx), systolic blood pressure (mmHg), diastolic blood pressure (mmHg), mean blood pressure (mmHg), and heart rate (minutes). The data were meticulously captured through the employment of the oscillometric method, facilitated by the 'Mobil-O-Graph® ARCSolver algorithm' device, coupled with the HMS CS (Hypertension Management System Client Server) software system. For the bilateral PWV measurements, two simultaneous readings were acquired from both sides of the body, and subsequently, the maximum value from each side was selected for subsequent analysis. The methodology employed for obtaining these measurements was adapted from previously published studies [10]. To minimize variability, all Mobil-O-Graph® measurements were performed in a quiet, temperature-controlled room after ≥ 10 minutes of supine rest. Patients were instructed to abstain from caffeine, smoking, and other vasoactive substances for at least 3 hours prior to assessment when clinically feasible. Measurements were acquired only when patients were hemodynamically stable, defined a priori as heart rate < 100 beats/min and brachial systolic blood pressure between 90 and 160 mmHg, with no intravenous inotrope/vasopressor boluses in the preceding 2 hours. Recordings with inadequate quality were repeated, and only traces meeting the device's quality criteria were accepted. As stated in the exclusion criteria, atrial fibrillation and cardiogenic shock were not present during assessments.

STATISTICAL ANALYSIS

Data analysis was carried out using the SPSS 22.0 software (IBM Corporation, Armonk, New York, United States). The normal distribution fit of the data was assessed using the Shapiro-Wilk test, and the homogeneity of variance was evaluated through the Levene test. To compare two distinct independent groups, the Independent-Samples T test was conducted with Bootstrap results, while the Mann-Whitney U test was employed in tandem with the Monte Carlo simulation technique. In order to explore relationships between variables, both the Pearson Correlation and the Spearman's rho tests were utilized. For comparing categorical data, the Fisher Exact test was utilized. Among the

categorical risk factors displaying significance, the Odds ratio was employed to identify the most crucial factor. Quantitative data were presented as mean $\pm$ SD (standard deviation) and median Range (Maximum-Minimum) values in the tables. Categorical data, on the other hand, were represented in n (number) and percentage (%). The data were analyzed at a 95% confidence level, and a p-value less than 0.05 was deemed statistically significant. Given the limited sample size— particularly within subgroups – no internal validation procedures (e.g., bootstrap resampling or cross-validation) were performed for the multivariable and ordinal models.

RESULTS

A comprehensive cohort of 104 patients participated in this study, comprising 30 males and 23 females diagnosed with NSTEMI, as well as 33 males and 18 females diagnosed with STEMI. The average age of the participants was 61.36 years. Refer to Table 1 for an overview of general patient characteristics. The patients' average BMI was noted as 28.43 kg/m². Notably, no statistically significant disparities emerged between the two groups regarding age, gender, and BMI (**p=0.626**, **p=0.428**, **p=0.578**, respectively) (Table 1).

Hypertension (HT) was prevalent in 58.7% of the patients, and Type 2 diabetes mellitus (DM) was present in 40.4% of the cases. Although no significant variance was observed between the groups

concerning Type 2 DM, a heightened occurrence of HT was noted within the NSTEMI group (**p=0.028**). The rate of smoking was 56.8%, with no substantial discrepancy discerned between the groups in this regard (**p=0.676**) (Table 1).

No noteworthy distinctions surfaced between the groups in terms of glucose, LDL, total cholesterol, urea, and creatinine levels (**p>0.05**) (Table 2).

However, upon examination, it was evident that hemoglobin (Hb) and cardiac troponin I (cTnI) levels during admission were notably elevated within the STEMI group (**p=0.007**, **p<0.001**, respectively) (Table 2 and Table 3).

While a mild positive correlation was noted between the SYNTAX score and PWV in the NSTEMI group (**r=0.356**; **p=0.009**), no comparable correlation was identified within the STEMI group (**p>0.05**) (Table 4).

When assessing the patient groups in relation to the SYNTAX score, a significantly strong correlation emerged between the SYNTAX score and both GRACE (**r=0.609**) and TIMI (**r=0.609**) scores within the NSTEMI group (**p<0.001**) (Table 4).

In the STEMI group, a robust positive correlation emerged between PWV and both TIMI (**r=0.510**) and urea (**r=0.563**) values (**p<0.01**). Across all patient groups, moderate and mild positive correlations were evident with TIMI score, total cholesterol, leukocyte count, urea and creatinine levels (**p<0.01**) (Table 5).

Table 1

Demographic findings of patients

		NSTEMI (n=53)	STEMI (n=51)	Total (n=104)	p value
		Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD	
Age (year)		61,94 $\pm$ 11,47	60,75 $\pm$ 13,51	61,36 $\pm$ 12,46	0.626
BMI (kg/m ²)		28,70 $\pm$ 5,04	28,14 $\pm$ 5,46	28,43 $\pm$ 5,23	0.578
Gender		n (%)	n (%)	n (%)	
	Female	23 (43,4)	18 (35,3)	41 (39,4)	0.428
	Male	30 (56,6)	33 (64,7)	63 (60,6)	
HT					
	no	16 (30,2)	27 (52,9)	43 (41,3)	0.028 (2,6 [1,2-5,8])^a
	yes	37 (69,8)	24 (47,1)	61 (58,7)	
T2DM					
	no	27 (50,9)	35 (68,6)	62 (59,6)	0.075
	yes	26 (49,1)	16 (31,4)	42 (40,4)	
Smoking					
	no	24 (45,3)	21 (41,2)	45 (43,2)	0.676
	yes	29 (54,7)	30 (58,8)	59 (56,8)	

SD: Standard Deviation, a: Odds Ratio (%95 Confidence Interval), T2DM, Type 2 Diabetes Mellitus, HT: Hypertension, STEMI: ST elevation myocardial infarction, NSTEMI: Non-ST elevation myocardial infarction, kg: kilogram

Table 2

Laboratory results of patients

Variables (on admission)	NSTEMI	STEMI	Total	p value
	(n=53)	(n=51)	(n=104)	
	Mean±SD	Mean±SD	Mean±SD	
WBC (*10 ³ /mm ³)	11,18±4082	11,87±4	11,46±4	0.238
Hb (g/dL)	12,62±2,34	13,71±1,73	13,16±2,12	0.007
BUN (mg/dL)	51,49±41	43,43±32	47,7±37,3	0.183
Creatinin (mg/dL)	1,34±0,9	1,18±0,6	1,28±0,8	0.319
Glucose (mg/dL)	148,88±84,57	154±68,7	150,67±76,2	0.737
Total kolesterol (mg/dL)	209,87±58,63	216,51±49,63	213,13±54,25	0.545
LDL (mg/dL)	124,72±51,71	142,39±48,72	133,38±50,80	0.068
HDL (mg/dL)	40±10,9	41±9,6	41±10,25	0.736
cTnI (µg/L)	3,7±1,14	2847±1609	1411±817	<0,001
CK-MB (U/L)	15,9±44,7	5899±4200	2928±2100	0.046

SD: Standard Deviation, WBC: White blood cells, Hb: hemoglobin, g: gram, dL: desi Liter, BUN: Blood urea nitrogen, LDL: Low density lipoprotein, HDL: High density lipoprotein, cTnI: cardial troponin I, CK-MB: Creatinin kinase-myoglobin, a: Odds Ratio (%95 Confidence Interval), T2DM: Type 2 Diabetes Mellitus, HT: Hypertension, STEMI: ST elevation myocardial infarction, NSTEMI: Non-ST elevation myocardial infarction, kg: kilogram, U: Unit

Table 3

Syntax, TIMI, Grace Scores and echocardiographic findings

	NSTEMI	STEMI	Total	P value
	(n=53)	(n=51) (%)	(n=104)	
	Median (Min-Max)	Median (Min-Max)	Median (Min-Max)	
SYNTAX Score	14 (2-33)	16,5 (3-28,5)	15 (2-33)	0.943
Grace Score (Mortality)	122 (57-222)	132 (66-230)	128,5 (57-230)	0.396
Aix	18 (4-61)	15 (5-61)	17 (4-61)	0.359
TIMI Score	3 (1-6)	3 (1-7)	3 (1-7)	0.119
LVEF	50 (20-65)	40 (15-60)	46,5 (15-65)	0.001
Cardiac Index	2,6 (1,4-5,9)	2,6 (1,7-4,1)	2,6 (1,4-5,9)	0.613
	Mean±SD	Mean±SD	Mean±SD	
PWV	8,89±1,94	8,49±2,18	8,70±2,06	0.322
Left atrium diameter (mm)	37,06±6,38	37,06±5,43	37,06±5,90	0.998
Hb	12,62±2,34	13,71±1,73	13,16±2,12	0.007
MSBP	111,49±20,73	113,27±19,62	112,37±20,11	0.654
MSDP	77,72±13,50	81,06±13,93	79,36±13,75	0.230
SYNTAX Score	n (%)	n (%)	n (%)	
<11	18 (34,0)	14 (28,0)	32 (31,1)	0.758
11–18	16 (30,2)	18 (36,0)	34 (33,0)	
>18	19 (35,8)	18 (36,0)	37 (35,9)	

Independent T test(Bootstrap) – Mann Whitney U test(Monte Carlo) – Pearson Chi-Square Test(Monte Carlo) – Fisher Exact Test(Exact), SD: Standard Deviation, Min: Minimum, Max: Maximum, a: Odds Ratio (%95), CI: Confidence Interval, STEMI: ST elevation myocardial infarction, NSTEMI: Non-ST elevation myocardial infarction, PWV: pulse wave velocity, TIMI: The Thrombolysis in Myocardial Infarction, MSBP: mean systolic blood pressure, MDBP: mean diastolic blood pressure

Table 4
Correlations with Syntax Score

Syntax Score	NSTEMI		STEMI		Total	
	r	P	r	P	r	P
PWV	,356**	0.009	0.095	0.508	,237*	0.015
Grace (on admission)	,609**	<0,001	,325*	0.020	,484**	<0,001
TIMI Score (on admission)	,609**	<0,001	,290*	0.039	,451**	<0,001
MSBP	-0.125	0.372	0.076	0.594	-0.031	0.757
MDBP	-0.059	0.674	0.014	0.921	-0.017	0.861
AIx	-0.185	0.185	-0.039	0.786	-0.123	0.214
LVEF	-,421**	0.002	-0.043	0.765	-,256**	0.009
Left atrium diameter (mm)	0.238	0.086	,301*	0.032	,265**	0.006

Spearman's rho Test, r: correlation coefficient, PWV: pulse wave velocity, TIMI: The Thrombolysis in Myocardial Infarction, MSBP: mean systolic blood pressure, MDBP: mean diastolic blood pressure, STEMI: ST elevated myocardial infarction, NSTEMI: Non-ST elevated myocardial infarction, MDBP: mean diastolic blood pressure, **p<0.01, *p<0.05

Table 5
Correlations with PWV

PWV	NSTEMI		STEMI		Total	
	r	P	r	P	r	P
Grace Score (mortality)	,296*	0.032	,324*	0.021	,301**	0.002
TIMI Score	,375**	0.006	,510**	<0,001	,456**	<0,001
Left atrium diameter (mm)	0.204	0.142	,404**	0.003	,294**	0.002
Age	,760**	<0,001	,861**	<0,001	,816**	<0,001
BMI	-0.111	0.427	0.193	0.175	0.057	0.564
Hb	-0.228	0.101	-0.154	0.281	-,226*	0.021
Total Cholesterol	-,363**	0.007	-0.089	0.534	-,235*	0.016
LDL	-0.203	0.146	-0.09	0.53	-0.159	0.106
HDL	-0.006	0.965	0.021	0.884	0.004	0.964
MSBP	,317*	0.021	,361**	0.009	,332**	0.001
MDBP	0.015	0.913	0.068	0.635	0.031	0.757
AIx	0.041	0.771	,422**	0.002	,247*	0.011
LVEF	-0.111	0.43	0.166	0.243	0.066	0.507
WBC	-,362**	0.008	-0.078	0.588	-,211*	0.032
RDW	0.164	0.242	0.078	0.588	0.123	0.215
BUN	,491**	<0,001	,563**	<0,001	,530**	<0,001
Creatinine	,320*	0.02	,453**	0.001	,376**	<0,001
Glucose	0.177	0.205	,293*	0.037	,197*	0.045
CK-MB (on admission)	-,275*	0.048	0.213	0.133	-0.005	0.959
Cardiac Index	,389**	0.004	-0.096	0.501	0.136	0.169

Spearman's rho Test, r: correlation coefficient, PWV: pulse wave velocity, TIMI: The Thrombolysis in Myocardial Infarction, MSBP: mean systolic blood pressure, MDBP: mean diastolic blood pressure, STEMI: ST elevated myocardial infarction, NSTEMI: Non-ST elevated myocardial infarction, MDBP: mean diastolic blood pressure, **p<0.01, *p<0.05

In the multivariable linear regression analyses (Supplementary Table S1), PWV was not independently associated with SYNTAX score in either STEMI, NSTEMI, or the total cohort. In

contrast, age showed a consistent and statistically significant positive association with SYNTAX score, particularly in the total cohort ($\beta=0.33$, 95% CI 0.06–0.59, $p=0.017$). Other covariates, including systolic blood pressure, hypertension status, and creatinine, were not significant predictors.

Sensitivity analyses incorporating mean arterial pressure (MAP), LDL cholesterol, and hemoglobin (Supplementary Table S2) yielded similar findings. PWV did not emerge as an independent determinant, whereas age remained a significant predictor. The explanatory power of these models was modest, with adjusted R^2 values ranging from 0.15 to 0.27.

In the ordinal regression model using SYNTAX categories (low, intermediate, high), age was again significantly associated with being in the high SYNTAX category ($\beta=0.11$, 95% CI 0.009–0.214, $p=0.032$), while PWV and other covariates showed no statistical significance. The model's pseudo R^2 was 0.063, indicating limited explanatory capacity.

DISCUSSION

Upon evaluating the outcomes of our study, a connection becomes apparent between PWV and two vital cardiac clinical risk scores: GRACE and TIMI scores, spanning all patients. However, the correlation linking PWV levels with the SYNTAX score, used to gauge the severity and extent of coronary lesions, was observable solely within the NSTEMI patient subset.

Although oscillometric assessment of pulse wave velocity and augmentation index is clinically feasible and provides complementary data, these parameters did not emerge as independent predictors in our multivariable analysis. This suggests that their prognostic utility in the acute ACS setting is largely mediated by chronic vascular aging rather than offering superior independent risk stratification beyond established factors [11–13]. For instance, Hansen *et al.* demonstrated a noteworthy link between PWV and the incidence of major cardiovascular events (MACE). Furthermore, the study conducted by Pierce *et al.* revealed an association between heightened arterial stiffness and an elevated cardiovascular risk attributed to cardiac baroreceptor impairment [14].

The prognostic value of adverse cardiovascular events determined by arterial stiffness mirrors that of other crucial risk factors like left ventricular hypertrophy. Arterial stiffening results from a multifaceted process fueled by the escalated deposition and cross-linking of extracellular matrix (ECM)

proteins within the vessel wall. This progression is accompanied by an augmentation in both vessel wall thickness and artery size. PWV stands as the conventional macroscopic gauge for assessing arterial stiffness in clinical diagnostics [15–17]. In fact, PWV measurements taken from participants within the Framingham cohort have validated that vascular stiffening precedes the onset of hypertension and cardiovascular events [18]. This underlines the notion that tracking variances in arterial stiffness, especially among patients exhibiting comparable blood pressure, notably enhances the capability to predict clinical outcomes [19,20].

The European guideline on arterial hypertension has identified an elevation in normalized vascular hemodynamics as a risk factor contributing to end-organ damage [21]. In recent times, a majority of the studies cited within this guideline were conducted utilizing the SphygmoCor® technique. Notably, similar findings were observed in studies employing newer and practical arterial stiffness measurement methods such as Mobil-O-Graph® and SphygmoCor® [22,23]. In our study, PWV measurements for all patients were conducted using the Mobil-O-Graph® system.

Numerous studies have established a link between an enlarged left atrial diameter and heightened MACE, irrespective of other contributing factors [24,25]. Nemes *et al.* conducted a study revealing that increased left atrium diameter correlated with augmented arterial stiffness in healthy volunteers [26]. This association was further linked to compromised coronary circulation, attributed to the enlarged left atrial dimension. Within our study, a statistically significant correlation was detected among the STEMI patient group, demonstrating that an increased left atrium diameter, SYNTAX score, and NAD were interrelated. However, this relationship was not evident within the NSTEMI patient cohort.

A meta-analysis has revealed that elevated brachial-ankle pulse wave velocity values are linked to a heightened risk of cardiovascular disease development, irrespective of whether patients possess a history of cardiovascular disease or not [27,28].

Echocardiography, computed tomography, and magnetic resonance imaging are additional tools employed for assessing arterial stiffness. Various techniques exist within these imaging modalities to gauge arterial stiffness, with the primary approach often involving the computation of aortic distensibility through alterations in aortic size during systole and diastole. Nevertheless, these methods are predominantly

restricted to research applications due to cost and technical complexities.

PWV measurement stands out as the most extensively employed parameter among the diverse indicators of arterial stiffness. A longitudinal study encompassing a 9.4-year follow-up period and comprising 1678 participants in Denmark revealed a noteworthy pattern. For each 1 standard deviation increase in carotid-femoral PWV (cfPWV) of 3.4 m/s, there was an associated escalation of 16–20% in CAD occurrence. Notably, this elevated risk persisted even after accounting for multiple clinical variables [29].

Patients exhibiting heightened vessel wall stiffness have been found to experience not only macrovascular but also microvascular CAD, coupled with diminished coronary blood flow reserve [30]. A noteworthy link has been established between increased PWV and left main coronary artery disease [31]. Ki *et al.* demonstrated that among 372 patients undergoing percutaneous coronary intervention (PCI), a notable PWV value (≥ 1672 cm/s) upon admission served as a predictive indicator for cardiac mortality post-PCI [32].

In a study by Kim *et al.* [33], involving 2561 hypertensive patients, findings unveiled an independent connection between elevated PWV – at or exceeding 1630 cm/s – and an augmented risk of cardiovascular events throughout a median follow-up period of 4.14 years. Similar outcomes have been observed across other investigations, affirming the predictive value of PWV among patients with hypertension and diabetes [34]. Notably, a recent study underscored the significance of both invasive and non-invasive PWV measurements in patients undergoing coronary angiography, all of which emerged as substantial factors associated with the occurrence of coronary events [4].

The GRACE score is a notably sensitive clinical risk assessment tool employed for forecasting both in-hospital and post-discharge mortality and morbidity, particularly within the context of NSTEMI patients [35]. Within a study conducted by Gedikli *et al.*, a marked association was established between arterial stiffness and the GRACE score among NSTEMI patients [36]. Our own study echoed these findings, revealing a significant correlation between NAD and both the GRACE and TIMI scores across both patient groups.

Numerous studies have indicated that arterial stiffness parameters, such as PWV and AIx, demonstrate regression when subjected to antihypertensive interventions. In the study conducted

by Mahmud *et al.*, the utilization of captopril and losartan showcased reductions in PWV and AIx values. Similarly, Klingbeil *et al.* revealed that while hydrochlorothiazide and valsartan exerted comparable blood pressure-lowering effects, a decrease in AIx and PWV values was solely achieved within the valsartan group ($p < 0.01$) [37]. In our investigation, no statistically significant variance was observed in PWV values among patients employing antihypertensive treatment in comparison to those who were not ($p = 0.158$). This outcome is presumed to stem from the fact that not all patients received the same antihypertensive medications at adequate dosages and durations.

Elevated PWV has been linked to higher mortality rates among patients diagnosed with myocardial infarction (MI), including both STEMI and NSTEMI [36]. NSTEMI is more prevalent among elderly individuals burdened with comorbid conditions. Consequently, NSTEMI patients typically exhibit a more diffuse and severe atherosclerotic background in comparison to those with STEMI. Within this patient category, cardiovascular events are often attributed to multivessel lesions, leading to a less favorable long-term prognosis when contrasted with STEMI patients [38–40]. As corroborated by our study, the noteworthy PWV increase observed within the NSTEMI patient cohort can be attributed to these factors. This divergence in arterial stiffness relevance likely reflects the distinct pathophysiological substrates of the two presentations. As noted in recent literature, NSTEMI is frequently characterized by diffuse, multi-vessel atherosclerosis and chronic vascular calcification, which aligns with our finding of a correlation between PWV and the SYNTAX score. Conversely, STEMI is typically driven by acute plaque rupture and thrombosis, where the immediate angiographic severity may correlate less with chronic arterial stiffness markers. Thus, the ineffective stratification in STEMI patients in our cohort may stem from the predominance of acute thrombotic events over chronic vessel wall remodeling.

Our primary objective was to explore the potential link between arterial stiffness parameters, as measured by the Mobil-O-Graph® device utilizing an oscillometric technique, and the severity of coronary artery lesions assessed via the SYNTAX score among hospitalized MI patients. However, this notable association was solely observed within the NSTEMI subgroup, where a meaningful correlation between the SYNTAX score and PWV value was established. Conversely, no statistically

significant correlation between arterial stiffness parameters and the SYNTAX score emerged either within the STEMI group or across the entire patient cohort. Of noteworthy importance, our findings highlighted a distinct relevance between the SYNTAX score and heightened PWV, particularly evident within the NSTEMI patient subgroup. Additionally, two pivotal ACS-related risk scores, namely GRACE and TIMI scores, were observed to exhibit correlations with normalized AIx. Notably, the SYNTAX score, encompassing the assessment of coronary lesion severity and extent, showcased an association with PWV only among NSTEMI patients.

Our findings demonstrate that PWV, although correlated with several clinical risk markers in univariate analyses, did not retain independent significance for predicting SYNTAX score in multivariable models. Across both the primary models (Supplementary Table S1) and sensitivity analyses (Supplementary Table S2), age consistently emerged as the strongest predictor of coronary lesion complexity, emphasizing its central role in risk stratification for ACS patients.

The robustness of these results was further supported by the ordinal regression analysis, where age remained independently associated with higher SYNTAX categories, while PWV and other hemodynamic variables failed to show predictive value. Importantly, the low R^2 and pseudo R^2 values highlight the limited explanatory power of arterial stiffness parameters in this single-center, heterogeneous cohort, suggesting that PWV may have only a modest role in clinical prediction beyond traditional risk factors.

Taken together, these results indicate that while arterial stiffness measurements are physiologically relevant, their incremental prognostic contribution in ACS appears limited when established clinical and biochemical parameters are accounted for. Larger multicenter studies with broader populations are warranted to further evaluate the independent value of PWV in coronary risk assessment.

LIMITATIONS

Our study has several limitations. First, while the Mobil-O-Graph® is a validated device, it estimates cfPWV via a transfer function rather than direct carotid-femoral measurement; thus, these estimated values may differ from gold-standard

tonometric recordings. Second, measurements were obtained during the acute phase of ACS. Although we excluded unstable patients, transient neurohumoral activation and acute hemodynamic changes post-MI may still influence vascular tone, potentially confounding the assessment of structural arterial stiffness. Third, we lacked standardized data regarding the duration of cardiovascular risk factors (e.g., the tenure of hypertension or diabetes diagnosis) and adherence to prior medical therapy. This variability in risk factor chronicity prevents full adjustment for cumulative vascular injury and may imply a biased evaluation of baseline vascular function. Fourth, the strong collinearity observed between age and PWV likely attenuated the independent association of stiffness parameters in our multivariable models, as reflected by the modest ΔR^2 values. Fifth, the lack of data on inflammatory markers (e.g., CRP) or endothelial function prevents us from inferring specific pathophysiological mechanisms linking stiffness to coronary complexity. Finally, due to the cross-sectional design and single-center nature of the study, our findings regarding the differential associations in NSTEMI versus STEMI cohorts should be interpreted as hypothesis-generating rather than confirmatory.

CONCLUSION

In conclusion, our study demonstrates that while arterial stiffness parameters show significant univariate associations with coronary disease severity (SYNTAX score) in NSTEMI patients and clinical risk scores (TIMI/GRACE) in STEMI patients, they do not emerge as independent predictors of coronary complexity after adjustment for age and traditional risk factors. These findings suggest that in the acute setting, elevated PWV serves primarily as a surrogate for cumulative vascular aging rather than an independent prognostic marker. The observed differences between subgroups likely reflect distinct pathophysiological profiles – chronic atherosclerotic burden in NSTEMI versus acute hemodynamic stress in STEMI. Therefore, these cross-sectional data should be viewed as hypothesis-generating. Future research requires longitudinal designs, specifically incorporating a one-year follow-up of vascular function in fully revascularized patients, to better characterize the disease trajectory and distinguish permanent arterial remodeling from acute-phase hemodynamic perturbations.

Introducere: Numeroase studii au demonstrat o asociere între creșterea rigidității arteriale (RA) și bolile cardiovasculare. De-a lungul timpului, au fost dezvoltate diverse metode pentru evaluarea rigidității arteriale.

Obiectiv: Scopul acestui studiu a fost investigarea corelației dintre RA, măsurată prin viteza undei pulsului (PWV) la nivel brahial, și scorul SYNTAX (Synergy between PCI with Taxus and Cardiac Surgery) derivat din angiografia coronariană (CAG).

Materiale și metode: Cohorta a inclus 53 de pacienți (51,0%) diagnosticați cu infarct miocardic fără supradenivelare de segment ST (NSTEMI) și 51 de pacienți (49,0%) cu infarct miocardic cu supradenivelare de segment ST (STEMI), cu vârste cuprinse între 18 și 80 de ani. Pentru toți participanții, s-au măsurat parametri precum PWV, indicele de augmentare (AIx) și tensiunea arterială utilizând dispozitivul „Mobil-O-Graph® ARCSolver algorithm”. Scorul SYNTAX a fost folosit pentru a evalua severitatea și extinderea bolii coronariene.

Rezultate: Vârsta medie a pacienților a fost de 61,36 ani, dintre care 61 (60,6%) bărbați și 43 (39,4%) femei. IMC-ul mediu a fost de 28,43 kg/m². Comparând grupurile de pacienți pe baza valorilor PWV, au reieșit observații interesante. În special, în grupul NSTEMI, s-a constatat o corelație pozitivă semnificativă între PWV și factori cheie precum vârsta, RDW (lărgimea distribuției eritrocitelor), nivelurile de uree (BUN) și scorul SYNTAX ($p < 0,05$).

Concluzii: Creșterea nivelurilor PWV s-a dovedit a fi deosebit de utilă în anticiparea severității bolii coronariene la pacienții cu NSTEMI. În schimb, la pacienții cu STEMI, valorile crescute ale PWV au reprezentat un predictor al unui prognostic nefavorabil, corelat cu scoruri clinice de risc mai ridicate.

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Data availability: The datasets analyzed in the current study are available from the corresponding author on reasonable request.

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Supplementary Table S1

Multivariable linear regression: SYNTAX score

	Total cohort	STEMI	NSTEMI
(Intercept)	1.12 (-11.42–13.65)	3.15 (-10.18–16.49)	-5.91 (-54.59–42.77)
baPWV	-0.78 (-2.68–1.11)	-1.34 (-3.78–1.09)	-0.12 (-7.63–7.39)
Age	0.30 (0.02–0.57)	0.28 (-0.05–0.61)	0.33 (-0.79–1.46)
Systolic BP	0.02 (-0.06–0.11)	0.06 (-0.02–0.15)	0.01 (-0.32–0.34)
Hypertension	-0.19 (-3.49–3.12)	-2.10 (-5.97–1.78)	1.95 (-4.68–8.58)
Creatinine	0.43 (-1.61–2.47)	-0.38 (-4.89–4.12)	0.15 (-4.86–5.17)
Num.Obs.	104	51	53
R2 Adj.	0.017	0.055	0.067

+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Supplementary Table S2

Sensitivity analysis (MBP, LDL, Hb added)

	Total cohort	STEMI	NSTEMI
(Intercept)	11.69 (-10.17–33.55)	-1.04 (-28.23–26.14)	15.15 (-42.23–72.54)
baPWV	-0.25 (-2.74–2.23)	-1.38 (-4.01–1.24)	1.27 (-6.51–9.05)
Age	0.23 (-0.14–0.59)	0.29 (-0.07–0.65)	0.12 (-1.03–1.27)
Mean BP	0.14 (-0.24–0.52)	0.03 (-0.50–0.56)	0.19 (-0.54–0.91)
LDL	0.02 (-0.02–0.05)	0.00 (-0.05–0.05)	0.04 (-0.01–0.08)
Hemoglobin	-0.94 (-1.87–0.02)	0.19 (-1.01–1.39)	-1.48 (-2.78–0.18)
Systolic BP	-0.09 (-0.39–0.21)	0.04 (-0.36–0.45)	-0.17 (-0.75–0.40)
Hypertension	-0.96 (-4.33–2.41)	-2.03 (-6.30–2.25)	0.50 (-5.67–6.68)
Creatinine	0.11 (-2.36–2.58)	-0.08 (-4.95–4.79)	-0.66 (-5.27–3.94)
Num.Obs.	104	51	53
R2 Adj.	0.081	-0.019	0.142

+ $p < 0.1$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$