

The Diagnostic Accuracy of Ankle Brachial Index in Relation to Coronary Computed Tomography in Coronary Artery Disease

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Abstract

Background: Arterial stiffness and loss of recoil indicate arterial aging and increase cardiovascular risk, particularly for ischemic heart disease. Advances in coronary computed tomography angiography (CCTA) enhance the detection of coronary artery disease (CAD) through high-quality imaging and plaque analysis. This study's objective is evaluating the ankle-brachial index (ABI) accuracy in diagnosing CAD compared to CCTA.

Methods: A prospective, observational study was executed with 200 participants who underwent ABI measurement and CCTA, with CAD severity assessed via the Gensini score. In accordance with Gensini scores, the patients were split into two distinct groups, Group 1: low Gensini score and Group 2: high Gensini score.

Results: The mean ABI was $96.26 \pm 9.3\%$, with 29% of patients showing abnormal readings. Patients with abnormal ABI were significantly older and exhibited a higher prevalence of hypertension (60.5%), diabetes (43%), and dyslipidemia (52%). The ABI effectively distinguished between normal and abnormal readings, achieving an AUC of 0.866 at a cutoff of $\leq 98\%$, with 100% sensitivity and 55.63% specificity. Coronary artery calcium (CAC) scores showed superior diagnostic performance with an AUC of 0.920 at a cutoff of >169 , yielding 91.38% sensitivity and 84.29% specificity. The Gensini score demonstrated an AUC of 0.723 at a cutoff of >94 , providing 70.69% sensitivity and 76.60% specificity.

Conclusion: CAC and Gensini scores were significantly elevated in the abnormal ABI group highlighting a clear correlation between lower ABI values and greater CAD severity. CAC scores showed excellent diagnostic accuracy, while ABI had high sensitivity but lower specificity for CAD assessment. The Gensini score is used as invasive method to assess severity of CAD correlated to Calcium scoring and ABI. Overall, the findings support using both ABI and CAC scoring in evaluating CAD, especially in at-risk groups.

Keywords: Ankle-brachial index, Coronary artery disease, Coronary computed tomography angiography, Coronary artery calcium, Gensini score, Ischemic heart disease, Arterial stiffness

Introduction

Arterial stiffness and loss of recoil indicate arterial aging and arteriosclerosis, which heighten the risk of cardiovascular issues, particularly ischemic heart disease. Advances in technology, particularly coronary computed tomography angiography (CCTA), allow for accurate detection of coronary luminal stenosis with high image quality [1,2]. Due to its noninvasive nature and high specificity and negative predictive value, CCTA is widely utilized for assessing coronary artery disease (CAD) [1,3]. It also facilitates the analysis of

plaque characteristics and calcium levels in the coronary arteries, providing further insights into coronary risk [1].

The severity of CAD can be evaluated using various scoring systems, such as the Gensini score, which assigns severity coefficients (0, 1, 2, 4, 8, 16, or 23) based on stenosis degree, with segment importance rated from 5 for the left main trunk to 0.5 for distal segments [4,5].

Coronary artery calcium (CAC) is a marker of overall coronary atherosclerotic burden. It is detected by noncontrast computed

tomography (CT) and quantified by the Agatston score, which is the sum of the products of attenuation (Hounsfield units) and area (mm²) of all lesions in the coronary arteries at each slice [6]. Because CAC is a marker of atherosclerotic disease, it may provide superior risk estimation over traditional risk factors. CAC score is a strong predictor of coronary heart disease (CHD) and Atherosclerotic cardiovascular disease (ASCVD). The presence of CAC indicates a 2.6- to 4.3-fold increased risk of CHD and a 2.1- to 2.6-fold increased risk of ASCVD, compared with a CAC score of 0.4–7 CAC score is also a predictor of all-cause mortality [7].

The ankle-brachial index (ABI), also referenced as the ankle-arm index, represents the comparative measurement between systolic blood pressure readings taken at the ankle and the brachial artery [3]. Among its various nomenclatures, ABI has emerged as the preferred terminology, receiving formal endorsement from the American Heart Association due to its widespread adoption in scientific publications [2]. While this diagnostic tool was originally conceived to identify peripheral artery disease (PAD) affecting the lower extremities, researchers have since discovered its broader clinical significance. The ABI has proven valuable not only in detecting atherosclerosis throughout the vascular system but also in predicting cardiovascular outcomes and functional decline, even in individuals who show no apparent PAD symptoms [3].

The ABI is a simple, cost-effective, and reproducible test for detecting subclinical atherosclerosis. An ABI of less than 0.9 indicates significantly increased risks of cardiovascular events and overall mortality [1,3], while an ABI over 1.4 is linked to higher cardiovascular risks as well. There is debate regarding ABI's effectiveness in reclassifying patients into different risk categories [8]. Recent European Guidelines (2024) recommend considering ABI for cardiovascular risk assessment [9].

In comparison to coronary computed tomography, this study's objective is to evaluate the ankle brachial index accuracy in the diagnosis of CAD.

Materials and Methods

Study population

This prospective, randomized, observational study recruited 200 consecutive participants to demonstrate the relationship between peripheral vascular disease assessed by pulse wave velocity (PWV) and CAD assessed by calcium scoring in CCTA and Gensini score in coronary angiography at Cardiology Department of Benha University Hospital and Cardiology Department of Al-Agoza Hospital.

Following ethical clearance from the Institutional Ethics Committees of both the Faculty of Medicine at Benha University and Al-Agoza University, the research team proceeded with participant recruitment. Each study participant provided

written informed consent after being thoroughly briefed about the study's objectives. To maintain confidentiality throughout the research process, participants were assigned unique identification codes that protected their personal information.

Inclusion criteria were patients of both sexes with stable CAD with justified CCTA due to one or more of the following (uncontrolled symptoms, high risk stress testing, low left ventricular ejection fraction, equivocal diagnosis to ascertain the cause).

The exclusion criteria were patients with one or more of the following (congenital heart disease, valvular heart disease, atrial fibrillation, chronic renal failure on dialysis and severe limb ischemia with ulcerative leg lesions).

Grouping

Patients were selected and divided according to ABI into two groups: Abnormal ABI group (n=58) and Normal ABI group (n=142).

Evaluation

All studied cases were subjected to the following: detailed history taking, full clinical examination, general examination (cardiovascular examination, peripheral vascular examination, and abdominal examination), routine laboratory investigations, and radiological investigations.

64-slice CT technology: This technology provides a noninvasive method to visualize the coronary arteries using a small dose of contrast. The new 64-slice multidetector CT scanner allows providers to analyze coronary artery lesions as well as blockages that were previously impossible to visualize. CT gathers multiple images, which acquire great amount of diagnostic information in a shorter amount of time. Unlike traditional x-ray imaging that produces a 2-D projection, CT takes thin x-ray scans from multiple directions. These multiple scans can be combined to create a 3-D image or volume. CT is used for 3-D volume imaging of the heart. For coronary CTA, a recent blood test for kidney functions and an ordering form is required for this test. To obtain high quality images with CTA many individuals require administration of a beta-blocker prior to the test to keep their heart rates under 70 beats per minute.

Techniques

A total of 232 patients underwent both coronary artery calcium scoring CT and ECG-synchronized CCTA examinations using 64-slice CT technology. In cases where patients exhibited heart rates exceeding 65 beats/min, bisoprolol was administered as premedication, with the study population showing an average heart rate of 64.4 ± 9.5 beats/min. The scanning protocol included preliminary sublingual nitroglycerin administration and image acquisition from the

tracheal carina extending to the cardiac base. The contrast protocol consisted of an initial 70 ml iomeprol bolus injection, subsequently complemented by a combined iodine-saline solution.

The scan parameters included a 64 x 0.625 mm detector array, with rotation times of 420 ms for the 64-slice scanner. CT raw data were reconstructed using different algorithms based on the scanner type, and the coronary artery calcium score (CACS) was calculated using the agatston score with a threshold of 130 Hounsfield units, yielding an effective dose of 13.4 ± 6.4 mSv.

The Gensini score is a quantitative system used to assess the degree of stenosis in coronary arteries, assigning values based on the percentage of narrowing: 1 point for 1-25% narrowing, up to 32 points for total occlusion. This score is then multiplied by a weighting factor that reflects the significance of the artery affected, such as 5 for the left main coronary artery and varying factors for other branches. The overall Gensini score is the sum of individual segment scores, reflecting both the severity of the lesions and their anatomical importance. The minimum score is 0, while the theoretical maximum is 656, assuming complete obstruction of all segments [10].

Additionally, the ABI involves measuring patient weights and heights to calculate BMI and measuring waist and hip circumferences. Blood pressure is taken after a 20-minute rest using a sphygmomanometer, and blood samples are drawn after fasting to analyze serum glucose, cholesterol, and triglycerides.

ABI was measured using a handheld Doppler device and fingertip pulse oximeter. Patients were examined while lying supine after a ten-minute rest in a controlled environment at 22°C. For ABI D, brachial blood pressure was assessed with a 14-cm pressure cuff and Doppler probe, identifying systolic pressure by inflating the cuff until the signal disappeared and then slowly deflating it. The highest recorded brachial pressure

determined the ABI. Ankle pressures were similarly measured over the posterior tibial and dorsalis pedis arteries, with higher value used. If there was a discrepancy greater than 10 mmHg, a second measurement was taken to ensure accuracy.

The operator of the ABI was blinded to the results of the CCTA.

For ABI P, pulse oximeter probes were positioned on the toes for ankle pressure and fingers for brachial pressure. The cuff was inflated until the signal ceased, with systolic digital pressure determined by gradual deflation. Measurements were performed on both legs and arms, with averages calculated and discrepancies addressed similarly to ABI D.

Statistical analysis

Statistical analysis was carried out utilizing SPSS v26, presenting quantitative variables as means and standard deviations, and comparing groups with unpaired Student’s t-test. Qualitative variables were expressed as frequencies and percentages, analyzed via Chi-square or Fisher’s exact tests as appropriate. A two-tailed P value of <0.05 was deemed statistically significant. Diagnostic performance was evaluated through sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV). Receiver Operating Characteristic (ROC) curve analysis was utilized for assessing overall test performance, with the area under the curve (AUC) indicating acceptability (AUC >50%) to excellence (AUC equal to 100%).

Results

Demographic data and risk factors were represented in **Table 1**.

Lipid Profile, cardiac biomarkers, echocardiography, severity of CAD, diseased vessels, clinical scores, and ABI of the studied patients were shown in **Table 2**.

Table 1. Demographic data, risk factors, vital signs, laboratory investigations among studied groups.		
		Total (n=200)
Age (years)	Mean ± SD	67.63 ± 10
	Range	50-85
Sex	Male	123 (61.5%)
	Female	77 (38.5%)
Weight (Kg)	Mean ± SD	85.08 ± 14.87
	Range	59-110
Height (m)	Mean ± SD	1.66 ± 0.04
	Range	1.59-1.73
BMI (Kg/m²)	Mean ± SD	30.77 ± 5.48
	Range	20.05-42.7

Residence	Urban	138 (69%)
	Rural	62 (31%)
HR (beats/min)	Mean $\pm$ SD	85.04 $\pm$ 5.99
	Range	75-95
SBP (mmHg)	Mean $\pm$ SD	132.05 $\pm$ 12.29
	Range	110-150
DBP (mmHg)	Mean $\pm$ SD	84 $\pm$ 9.97
	Range	70-100
Hb (g/dl)	Mean $\pm$ SD	11.49 $\pm$ 1.22
	Range	9.5-13.5
PLT (*10⁹/L)	Mean $\pm$ SD	281.08 $\pm$ 43.35
	Range	200-349
WBCs (*10⁹/L)	Mean $\pm$ SD	7.94 $\pm$ 2.09
	Range	4.5-11.5
Serum creatinine (mg/dL)	Mean $\pm$ SD	1.05 $\pm$ 0.17
	Range	0.8-1.3
Urea (mg/dL)	Mean $\pm$ SD	38.65 $\pm$ 11.85
	Range	20-60
FBG (mg/dl)	Mean $\pm$ SD	123 $\pm$ 50.16
	Range	70-220
PPBS (mg/dl)	Mean $\pm$ SD	187.55 $\pm$ 64.02
	Range	110-298
ALT (U/L)	Mean $\pm$ SD	28.23 $\pm$ 6.32
	Range	18-40
AST (U/L)	Mean $\pm$ SD	32.11 $\pm$ 7.88
	Range	18-46
Smoking		90 (45%)
HTN		121 (60.5%)
DM		86 (43%)
Dyslipidemia		104 (52%)
IHD		44 (22%)
Obesity		106 (53%)
Family history of CAD		65 (32.5%)
Chronic heart failure		34 (17%)
Peripheral vascular disease		26 (13%)
BMI: Body Mass Index; HTN: Hypertension; DM: Diabetes Mellitus; IHD: Ischemic Heart Disease; CAD: Coronary Artery Disease; HR: Heart Rate; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; Hb: Hemoglobin; PLT: Platelets; WBCs: White Blood Cells; FBG: Fasting Blood Glucose; PPBS: Post-Prandial Blood Sugar; ALT: Alanine Transaminase and AST: Aspartate Aminotransferase.		

Table 2. Lipid Profile, cardiac biomarkers, echocardiography, severity of CAD and diseased vessels, clinical scores and ABI of the studied patients.		
		Total (n=200)
Total cholesterol (mg/dl)	Mean ± SD	171.63 ± 63.09
	Range	80-260
Triglycerides (mg/dl)	Mean ± SD	177.49 ± 60.39
	Range	100-300
HDL (mg/dl)	Mean ± SD	44.54 ± 10.82
	Range	30-60
LDL (mg/dL)	Mean ± SD	140.74 ± 33.23
	Range	90-200
CK-MB (IU/L)	Mean ± SD	23.2 ± 9.11
	Range	10-47
Brain natriuretic peptide (pg/mL)	Mean ± SD	71.8 ± 10.59
	Range	55-98
Troponin I (ng/mL)	Mean ± SD	0.07 ± 0.02
	Range	0.041-0.1
EF (%)	Mean ± SD	47.4 ± 4.54
	Range	39-56
LVIDd (cm)	Mean ± SD	52.99 ± 3.12
	Range	47-59
LVIDs (cm)	Mean ± SD	37.06 ± 5.49
	Range	28-49
Severity of CAD		82 (41%)
Diseased vessels	Single	89 (44.5%)
	Double	66 (33%)
	Multivessel disease	45 (22.5%)
CAC score	Mean ± SD	155.18 ± 145.28
	Range	4-800
Gensini score	Mean ± SD	79.52 ± 58.72
	Range	20-280
ABI (%)	Mean ± SD	96.26 ± 9.3
	Range	80-114
Abnormal ABI (%)		58 (29%)
ABI: Ankle-Brachial Index; CAC: Coronary Artery Calcium; CAD: Coronary Artery Disease; EF: Ejection Fraction; LVIDd: Left Ventricular Internal End Diastole Diameter; LVIDs: Left Ventricular Internal End Systolic Diameter; CK-MB: Creatine Kinase Myocardial Band; HDL: High-Density Lipoproteins; LDL: Low-Density Lipoproteins.		

The analysis revealed that the abnormal ABI group had significantly higher age compared to the normal ABI group ($P=0.011$) and showed a significant difference in residence ($P<0.001$). Other baseline characteristics such as gender, weight, height, and BMI were not significantly different between the groups. Among risk factors, hypertension, chronic heart failure, and peripheral vascular disease were significantly

more prevalent in the abnormal ABI group ($P<0.05$), while smoking, diabetes mellitus, family history of CAD, and obesity did not differ significantly. Additionally, heart rate, systolic blood pressure, and diastolic blood pressure were significantly higher in the abnormal ABI group ($P<0.05$), while laboratory investigations showed no significant differences between the two groups (**Table 3**).

Table 3. Demographic data, risk factors, vital signs, laboratory investigations of the patients studied regarding ABI.

		Abnormal ABI (n=58)	Normal ABI (n=142)	P value
Age (years)	Mean $\pm$ SD	70.6 $\pm$ 10.32	66.7 $\pm$ 9.72	0.011*
	Range	51-85	51-85	
Gender	Male	37 (63.79%)	86 (60.56%)	0.631
	Female	21 (36.21%)	56 (39.44%)	
Weight (Kg)	Mean $\pm$ SD	85.2 $\pm$ 15.94	85 $\pm$ 14.47	0.930
	Range	62-108	59-110	
Height (m)	Mean $\pm$ SD	1.7 $\pm$ 0.04	1.7 $\pm$ 0.04	0.617
	Range	1.59-1.73	1.59-1.73	
BMI (Kg/m ²)	Mean $\pm$ SD	30.8 $\pm$ 6.02	30.8 $\pm$ 5.26	0.986
	Range	22.38-42.72	41.66-20.05	
Residence	Urban	50 (86.21%)	88 (61.97%)	>0.001*
	Rural	8 (13.79%)	54 (38.03%)	
HR (bpm)	Mean $\pm$ SD	86.6 $\pm$ 7.42	84.5 $\pm$ 6.17	0.040*
	Range	75-100	70-95	
SBP (mmHg)	Mean $\pm$ SD	136.4 $\pm$ 11.19	130.3 $\pm$ 12.32	>0.001*
	Range	110-150	110-150	
DBP (mmHg)	Mean $\pm$ SD	88.3 $\pm$ 9.39	82.3 $\pm$ 9.7	>0.001*
	Range	70-100	70-100	
Hb (g/dl)	Mean $\pm$ SD	11.5 $\pm$ 1.31	11.5 $\pm$ 1.19	0.801
	Range	9.5-13.4	9.5-13.5	
PLT (*10 ⁹ /L)	Mean $\pm$ SD	277.9 $\pm$ 46.34	282.4 $\pm$ 42.17	0.508
	Range	200-349	202-349	
WBCs (*10 ⁹ /L)	Mean $\pm$ SD	7.9 $\pm$ 2.03	7.9 $\pm$ 2.12	0.982
	Range	4.5-11.5	4.5-11.5	
Creatinine (mg/dL)	Mean $\pm$ SD	1.1 $\pm$ 0.18	1 $\pm$ 0.16	0.755
	Range	0.8-1.3	0.8-1.3	
Urea (mg/dL)	Mean $\pm$ SD	37.3 $\pm$ 11.65	39.2 $\pm$ 11.92	0.314
	Range	20-60	20-60	
FBG (mg/dl)	Mean $\pm$ SD	119.7 $\pm$ 51.43	124.3 $\pm$ 49.75	0.558
	Range	70-217	70-220	
PPBS (mg/dl)	Mean $\pm$ SD	184.2 $\pm$ 67.31	188.9 $\pm$ 62.81	0.638
	Range	112-298	110-297	

ALT (U/L)	Mean± SD	27.5 ± 6.59	28.5 ± 6.21	0.310
	Range	18-40	18-40	
AST (U/L)	Mean ± SD	30.4 ± 7.62	32.8 ± 7.91	0.104
	Range	18-43	18-46	
Smoking		21 (36.21%)	69 (48.59%)	0.110
HTN		51 (87.93%)	70 (49.3%)	>0.001*
DM		31 (53.45%)	55 (38.73%)	0.056
Family history of CAD		21 (36.21%)	44 (30.99%)	0.474
Obesity		29 (50%)	77 (54.23%)	0.587
Chronic heart failure		15 (25.86%)	11 (7.74%)	0.005*
Peripheral vascular disease		28 (48.26%)	6 (4.22%)	<0.001*
BMI: Body Mass Index; HTN: Hypertension; DM: Diabetes Mellitus; IHD: Ischemic Heart Disease; CAD: Coronary Artery Disease; HR: Heart Rate; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; Hb: Hemoglobin; PLT: Platelets; WBCs: White Blood Cells; FBG: Fasting Blood Glucose; PPBS: Post-Prandial Blood Sugar; ALT: Alanine Transaminase and AST: Aspartate Aminotransferase.				

The lipid profile showed that cholesterol, triglycerides, and LDL levels were significantly greater in the abnormal ABI group in comparison with the normal ABI group ($P<0.05$), while HDL was significantly lower ($P=0.041$). Cardiac biomarkers, including CK-MB, brain natriuretic peptide, and troponin I, were also significantly elevated in the abnormal ABI group ($P<0.05$). Additionally, ejection fraction (EF) was significantly

lower ($P=0.033$), though no significant differences were observed in LVIDd and LVIDs between the groups. The coronary artery disease severity (CAD), in addition to CAC and Gensini scores, were significantly greater in the abnormal ABI group ($P<0.001$), which corresponded to a significantly lower ABI in this group ($P<0.001$) (**Table 4**).

Table 4. Lipid profile, cardiac biomarkers, echocardiography, severity of CAD and diseased vessels, clinical scores and ankle-brachial index of the patients studied regarding ABI.

		Abnormal ABI (n=58)	Normal ABI (n=142)	P value
Cholesterol (mg/dL)	Mean ± SD	189.8 ± 63.56	166.9 ± 62.57	0.020*
	Range	81-265	80-260	
Triglycerides (mg/dL)	Mean ± SD	195.8 ± 62.96	173.8 ± 60.72	0.023*
	Range	103-295	100-300	
HDL (mg/dL)	Mean ± SD	41.8 ± 9.02	45.2 ± 10.94	0.041*
	Range	30-58	30-60	
LDL (mg/dL)	Mean ± SD	150.2 ± 35.53	139.6 ± 32.3	0.042*
	Range	90-200	90-200	
CK-MB (IU/L)	Mean ± SD	32.4 ± 9.17	19.5 ± 5.85	<0.001*
	Range	15-47	10-30	
Brain natriuretic peptide (pg/mL)	Mean ± SD	83.6 ± 7.99	67 ± 7.22	<0.001*
	Range	70-98	55-80	
Troponin I (ng/mL)	Mean ± SD	0.1 ± 0.01	0.1 ± 0.01	<0.001*
	Range	0.06-0.1	0.041-0.09	
EF (%)	Mean ± SD	43.6 ± 3.68	49.8 ± 4.42	<0.001*
	Range	39-50	43-56	
LVIDd (cm)	Mean ± SD	52 ± 3.13	52.7 ± 2.98	0.162
	Range	47-59	47-57	

LVIDs (cm)	Mean ± SD	36.7 ± 4.96	37.2 ± 5.7	0.512
	Range	28-48	28-49	
Severity of CAD		36 (62.07%)	46 (32.39%)	>0.001*
CAC	Mean ± SD	307 ± 137.18	77.7 ± 82.08	<0.001*
	Range	29-800	4-414	
Gensini score	Mean ± SD	121.8 ± 60.9	62.2 ± 47.58	<0.001*
	Range	22-280	20-184	
ABI	Mean ± SD	90.7 ± 4.9	101.8 ± 7.9	<0.001*
	Range	80-98	90-114	

ABI: Ankle-Brachial Index; CAC: Coronary Artery Calcium; CAD: Coronary Artery Disease; EF: Ejection Fraction; LVIDd: Left Ventricular Internal End Diastole Diameter; LVIDS: Left Ventricular Internal End Systolic Diameter; CK-MB: Creatine Kinase Myocardial Band; HDL: High-Density Lipoproteins; LDL: Low-Density Lipoproteins.

The study demonstrated that the ABI can effectively differentiate between normal and abnormal ABI readings, with an AUC of 0.866 at a cutoff value of ≤ 98%, achieving 100% sensitivity but only 55.63% specificity. Additionally, at a cutoff value of >210, the AUC improved to 0.920, resulting in 91.38% sensitivity and 84.29% specificity. Lastly, with a cutoff of >94, the AUC was 0.723, yielding 70.69% sensitivity and 76.60% specificity (Figures 1a-1c).

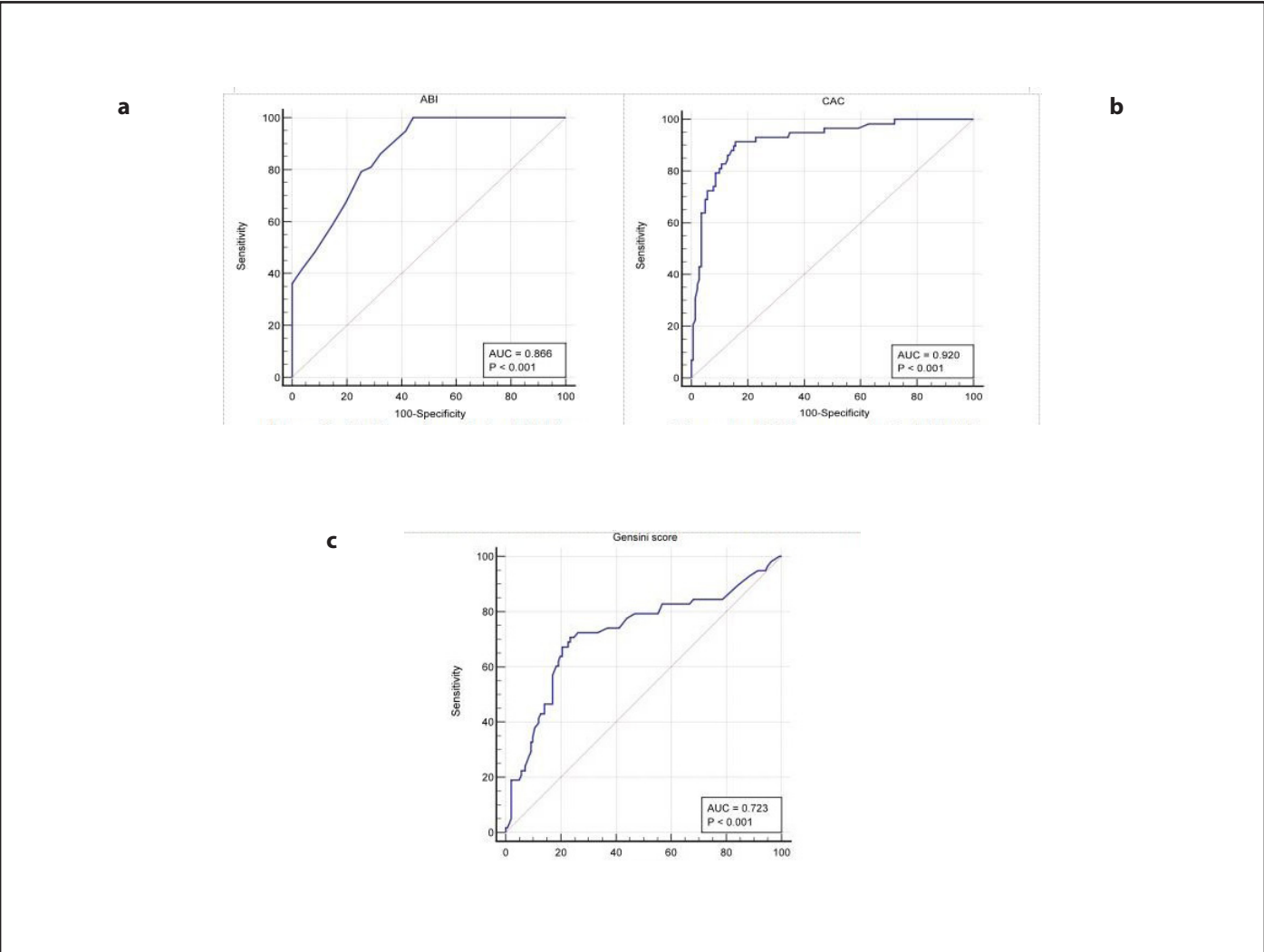


Figure 1. a. ROC curve analysis of ABI for discrimination between normal and abnormal ABI. b. ROC curve analysis of CAC for discrimination between normal and abnormal ABI. c. ROC curve analysis of Gensini score for discrimination between normal and abnormal ABI.

Discussion

Our study analyzed patients aged 50–85 years, participants' weights ranged from 59 to 110 kg, heights from 1.59 to 1.73 m, and BMIs from 20.05 to 42.7 kg/m². Most patients were from urban areas. These findings align with a study by Doğan *et al.* which reported a mean age of 59.1 ± 15.9 years and 82.5% males in a cohort of Turkish subjects [11].

In our study, 45% of patients were smokers, 60.5% were hypertensive, 43% were diabetics, 52% had dyslipidemia, 22% had IHD, 53% were obese, and 32.5% had a positive family history of CAD. These results align with a recently published study [11] which found hypertension in 47.4% of patients, diabetes in 40.4%, obesity in 10.5%, dyslipidemia in 57.9%, and CAD in 42.1%. Smoking rates were similar at 45%, increasing to 82.5% when including ex-smokers. Additionally, some authors reported 75% hypertension, 47% dyslipidemia and 28% diabetes with 15% of their sample being smokers [12].

Our study conducted that the ABI ranged from 80 to 114% with a mean of 96.26 ± 9.3%, and 29% of patients had abnormal ABI. The abnormal ABI group was significantly older than the normal ABI group, and residence patterns differed, with more patients from urban areas. Other baseline characteristics such as gender, weight, height, and BMI showed no significant differences. Supporting these findings Doğan *et al.* found higher age in the abnormal ABI group, although they noted a significant association with female patients, which differed from the current study. Similarly, mean ABI of 0.6 ± 0.2 was found with no gender difference [11].

In our study, risk factors such as smoking, DM, family history of CAD, and obesity showed no significant differences between groups, while HTN was significantly more prevalent in the abnormal ABI group. Supporting this, Kim *et al.* also found higher HTN rates in the abnormal ABI group, with no significant differences in smoking or obesity. However, they reported significant increases in DM and myocardial infarction history in the abnormal ABI group [13], which differed from our findings. Similarly, it was confirmed that HTN was significantly higher in patients with abnormal ABI in a study involving patients undergoing PCI [14].

Our results indicated that EF was significantly lower in the abnormal ABI group compared to the normal ABI group, with no notable differences in LVIDd or LVIDs. Frere *et al.* corroborated our findings, noting a significantly lower EF in the abnormal ABI group and no significant difference in LVIDd, but they reported that LVIDs was higher in the abnormal ABI group [13], which diverged from our results. Similarly, it was found that reduced left ventricular function was more prevalent in the abnormal ABI group [14].

In this study, we found that the CAD severity was significantly greater in patients with abnormal ABI compared to those with normal ABI. This is consistent with Frere *et al.* study who

reported a greater SYNTAX score in the abnormal ABI group [13], and with other authors who also noted significantly greater CAD severity in patients with abnormal ABI [15]. However, it was found that there is no significant correlation between ABI and the extent or coronary disease severity as measured by the Friesinger index, contrasting with our results [12].

Our findings indicated that both the CAC and Gensini scores were significantly greater in the abnormal ABI group compared to the normal ABI group. Conversely, ABI values were significantly lower in the abnormal group. This aligns with Frere *et al.* study who also reported higher Gensini scores and lower ABI in the abnormal group [13].

Furthermore, a published study noted that lower ABI values are associated with generalized vascular atherosclerosis, increasing the risk of cardiovascular events, supporting the notion that abnormal ABI serves as a marker for more severe CAD [16].

In the present study, the ABI demonstrated an AUC of 0.866, effectively distinguishing between normal and abnormal ABI at a cutoff of ≤ 98%, with 100% sensitivity and 55.63% specificity. CAC score showed an AUC of 0.920, significantly differentiating between groups at a cutoff of >169, with 91.38% sensitivity and 84.29% specificity. The Gensini score had an AUC of 0.723, discriminating at a cutoff of >94, yielding 70.69% sensitivity and 76.60% specificity.

Frere *et al.* corroborated our findings with a similar Gensini score cutoff [13], while others noted that higher CAC scores consistently indicated an increased risk for cardiovascular events, with AUC values often exceeding 0.90, highlighting their strong diagnostic ability [17].

Yet the study has several limitations, comprising a small sample size that reduced statistical power and issues related to observational study design. Measuring the ABI was often inaccurate in patients with non-compressible arteries, particularly among older adults and diabetics, and some individuals lacked palpable arteries, complicating reliable measurements. While ABI is sensitive for detecting peripheral artery disease, its lower specificity for coronary artery disease can lead to false positives.

Conclusion

CAC and Gensini scores were significantly elevated in the abnormal ABI group and highlighting a clear link between lower ABI values and greater CAD severity. CAC scores showed excellent diagnostic accuracy, while ABI had high sensitivity but lower specificity for CAD assessment. The Gensini score is used as an invasive method to assess severity of CAD correlated to Calcium scoring and ABI. Overall, the findings support using both ABI and CAC scoring in evaluating CAD, especially in at-risk groups.

Conflicts of Interest

There are no conflicts of Interest.

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Author Contribution

The contributions to this study were made equally by all authors.

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