Original Article

Relationship between Ankle-Brachial Index with Coronary Angiography Outcomes in Patients with Risk of Coronary Artery Disease

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Abstract

Background : The Ankle-Brachial Index (ABI) is a non-invasive diagnostic method that compares the Blood Pressure of the lower limbs with that of the arms. This may indicate Peripheral Artery Disease (PAD).

Aims and Objectives : This study examined the relationship between the ABI and coronary angiographic outcomes.

Materials and Methods : This hospital-based observational study included 210 patients with CAD from the Department of Cardiology at King George's Medical University in Lucknow. Patients with Coronary Angiography were divided into three groups based on their ABI values: ABI>0.9<1.2 (intermediate risk of developing vessel disease, n = 49), ABI<0.9 (high risk of developing CAD, n = 45) and ABI>1.2 (normal range, n = 116). We recorded the lipid profile and covariates including age, gender, smoking status, Body Mass Index (BMI), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Vessel occlusion and Calcification.

Results : The ABI<0.9 category was primarily populated by older individuals, resulting in significant age disparities across the ABI groups (p<0.0001). The ABI <0.9 group was significantly more prevalent among males and smokers (p=0.010 and p<0.0001, respectively). Hypertension and Diabetes Mellitus were most prevalent in the group with an ABI of <0.9 (p<0.0001). A significant correlation was observed between lower ABI values and family history of cardiovascular disease (p<0.0001). Vessel occlusion was primarily observed in the ABI <0.9 group, with significant differences in calcification rates (p<0.0001). A positive correlation was found between ABI values and vessel disease severity ie, extent of vessel involvement. Angiography revealed a significant association between lower ABI and the presence of CAD.

Conclusion : ABI measurements significantly correlated with CAD severity in patients without prior PAD, suggesting their potential use as a non-invasive screening tool in clinical settings.

Key words : Ankle-Brachial Index (ABI), CAD, PAD, Vessel Occlusion, Lipid Profile.

Cardiovascular Diseases (CVDs) cause over 17 million (32%) deaths worldwide, with approximately 80% in middle- and low-income nations[1]. Coronary Heart Disease (CHD) is a major cause of mortality and functional disability among the elderly. The main risk factors included hyper-cholesterolemia, hypertension, diabetes mellitus and smoking. Peripheral Arterial Disease (PAD) and coronary involvement are positively correlated with atherosclerosis, a generalized process affecting the coronary, cerebral and peripheral arteries[2].

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Editor's Comment :
- Ankle-Brachial Index (ABI) is a simple non invasive test which can help in making diagnosis of atherosclerosis of vessels.
- This test has not been utilized in day to day medicine.

Detecting subclinical atherosclerosis is crucial for the early intervention and prevention of cardiovascular disease. However, the widespread use of carotid Ultrasound and coronary Computed Tomography/ Magnetic Resonance Imaging can be costly, potentially leading to delays in the diagnosis and rationing of healthcare resources, progression of the disease and increased morbidity. Therefore, it is essential to consider both economic and clinical aspects. The ABI has emerged as a non-invasive and cost-effective CVD diagnostic method. Lower ABI values mean a two- to three-fold higher risk of cardiovascular and cerebrovascular morbidity and mortality. ABI utility extends beyond the detection of PAD and indicates systemic atherosclerosis and cardiovascular risks. The ABI is calculated by

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comparing the Blood Pressure readings at the ankle with those at the arm, and lower values are indicative of increased risk of cardiovascular events.

Studies have demonstrated that an ABI<0.90 is strongly associated with a higher incidence of CHD and cerebrovascular events. Furthermore, primary and secondary cardiovascular prevention strategies recognize ABI for its predictive value in assessing the CAD severity, making it a valuable method[3,4]. By incorporating ABI into routine evaluations, healthcare providers can classify high-risk individuals and implement appropriate interventions to mitigate the risk of adverse cardiovascular outcomes. This approach is particularly beneficial in resource-limited settings where more expensive diagnostic modalities are not feasible. ABI values are related to angiographic findings and support their use as a substitute for CAD markers. This makes it cost-effective to determine whether the disease is aggressive and make treatment plans specific to each patient. Based on the above background, the present study investigated the relationship between ABI and angiographic findings in patients suspected of or at risk for CAD.

Materials and Methods

Study Design and Setting :

This hospital-based observational study, subjects (n=210) were recruited from the Department of Cardiology, both Outpatient (OPD) and Inpatient (IPD) services, at King George's Medical University (KGMU), Lucknow from October, 2019 to September, 2020. These subjects were identified as having a risk of developing CAD with diabetes, hypertension, history of smoking, and dyslipidaemia. Exclusion criteria included patients with lower limb gangrene, limb deformities, non-consenting individuals, valvular or congenital heart or vascular diseases, proven malignancies or severe pulmonary, renal or hepatic comorbidities. The Institutional Ethics Committee (ref.no 886/ Ethics/2020 of KGMU, Lucknow) approved the study protocol.

The ABI measurement was calculated using a standardized protocol described by D¹browski, *et al*[5]. Measurements were taken using an automated oscillometric device, with patients in a supine position to ensure that the arms and legs were at the level of the heart. Blood Pressure cuffs, adequately sized to exceed the limb diameter by 20%, were used to completely encircle the upper and lower extremities. The cuffs were positioned above the malleoli at the calf muscle for recording ankle blood pressure and the 2.5 cm above antecubital fossa at the arm, ensuring obliteration of the brachial artery. Systolic Blood Pressure (SPB) was measured in both arms, and an average of three readings was recorded. The higher value between the two arms was recorded as the Brachial Systolic Blood Pressure (BSBP) for ABI calculation.

Ankle Brachial Pressure Index (ABPI) Calculation:

The ABPI was calculated by dividing the ankle and BSBP (Fig 1). Both right and left-sided ABPIs were calculated and in instances where the values differed, a lower value was documented. ABI was measured and recorded at two decimal places. The patients were divided into three groups: ABI group I (ABI ≤0.9) and ABI group II (ABI>0.9) and ABI group III (ABI; 0.9-1.2). For coronary involvement, all patients had underwent diagnostic coronary angiography via either the femoral or radial route using a 5F or 6F catheter. The lesions identified during the coronary angiography were then classified. These angiographic findings divided the outcomes into mild and severe CAD group. The vessels studied included the Left Coronary Artery (LCA), Right Coronary Artery (RCA), Left Anterior Descending artery (LAD), first and second diagonal branches (D1, D2), Obtuse Marginal artery (OM), and Left Circumflex artery (LCX). Vessel occlusion was categorized as no occlusion, <50% occlusion or >50% occlusion. Vessel calcification was noted as either present or absent.

Covariant analysis: The association between ABI categories and the extent and severity of CAD, including age, sex, smoking status, SBP, DBP, total serum cholesterol (TC), Low-density Lipoprotein (LDL) cholesterol, High-density Lipoprotein (HDL) cholesterol, triglycerides and angiographic findings, was analyzed. Angiographic findings were subsequently compared with the ABI values. CAD was defined as a >50% stenosis in the major coronary vessels. CAD was assessed and classified as mild to moderate and severe occlusion, corresponding to ≤50% occlusions in diameter and >50% stenosis in major vessels, respectively. The relationship between ABI and angiographic findings was analyzed to determine the potential of ABI as a screening tool in individuals suspected of having cardiovascular dieases with no previous history of PAD and to predict the severity of CAD in the future.

Statistical analysis : All statistical analyses were performed using SPSS software (IBM version 22). The Kolmogorov-Smirnov test was used to determine normality. Fisher's exact or the chi-square test was used for categorical data. ANOVA was used to evaluate differences among the groups. Pearson's correlation coefficient analysis was used to establish relationships between variables. Multiple regression analysis was applied to the ABI and vessel occlusion groups. Statistical significance was set at p<0.05.

Results

Association of Age, Smoking Hypertension, Diabetes, Cardiology Vascular History, Vessel Occlusion and Calcification with ABI :

Participants with ABI <0.9 (indicative of PAD) had a mean age of 60.73 (SD=5.64) years, which was significantly higher than those in the ABI 0.9-1.2 (normal range) and ABI>1.2 (potentially non-compressible arteries) groups, with mean ages of 55.08 (SD=8.80) and 52.60 (SD=7.73) years (p<0.0001), respectively. Male participants predominated in the ABI<0.9 group (95.9%), compared to 79.3% in the ABI 0.9-1.2 group and 91.1% in the ABI>1.2 group (p=0.01). Smoking prevalence was markedly higher in the ABI <0.9 group at 95.9%, compared to 51.7% in the ABI 0.9-1.2 group and 51.1% in the ABI >1.2 group (p<0.0001). A similar trend was noted for tobacco use, with 95.9% in the ABI <0.9 group, 35.4% in the ABI 0.9-1.2 group and 31.1% in the ABI>1.2 group reporting usage (p<0.0001). Hypertension was universally present in the ABI <0.9 group (100%), while it was reported in 47.4% of the ABI 0.9-1.2 group and 33.3% of the ABI>1.2 group (p<0.0001). Diabetes Mellitus was also significantly associated with lower ABI, with 77.5% in the ABI <0.9, 44.0% in the ABI 0.9-1.2 group and 51.1% in the ABI>1.2 group (p<0.0001). A family history of cardiovascular issues was more common in the ABI<0.9 group (71.4%) compared to the ABI 0.9-1.2 (26.7%) and ABI>1.2 (22.2%) groups (p<0.0001). Complete vessel occlusion was observed exclusively in the ABI <0.9 group. Calcification was present in 46.9% of the ABI <0.9 group, which was significantly higher than the 13.8% in the ABI 0.9-1.2 group but comparable to the 42.2% in the ABI >1.2 group (p<0.0001) (Table 1).

Table 1 — *Demographical characteristics of the study population*

Variables	ABI (<0.9) (n=49)N(%)	ABI (0.9-1.2) (n=116)N(%)	ABI (>1.2) (n=45)N(%)	p-value
Age (years)				
mean±SD	60.73±5.64	55.08±8.80	52.60±7.73	<0.0001*
Gender				
Male	47(95.9)	92(79.3)	41(91.1)	
Female	02(4.08)	24(20.7)	04(8.9)	0.010*
Smoking				
Yes	47(95.9)	60(51.7)	23(51.1)	
No	02(4.08)	56(48.3)	22(48.9)	<0.0001*
Tobacco				
Yes	47(95.9)	41(35.4)	14(31.1)	
No	02(4.08)	75(64.6)	31(68.9)	<0.0001*
Hypertension				
Yes	49(100.0)	55(47.4)	15(33.3)	
No	0	61(52.6)	30(66.7)	<0.0001*
DM				
Yes	38(77.5)	51(44.0)	23(51.1)	
No	11(22.5)	65(56.0)	22(48.9)	<0.0001*
Family history				
Yes	35(71.4)	31(26.7)	10(22.2)	
No	14(28.6)	85(73.3)	35(77.8)	<0.0001*
Vessel ocullation				
No	0	13(11.3)	0	
<50%	0	27(23.4)	0	
>50%	49(100.0)	76(65.5)	45(100.0)	<0.0001*
Calcification				
No	26(53.1)	100(86.2)	26(57.8)	
Yes	23(46.9)	16(13.8)	19(42.2)	<0.0001*

The chi-square test and ANOVA test were used to compare the groups. *p<0.05 was considered as statistically significant.

Variations in lipid profiles and heart rate among the ABI Group :

The assessment of lipid profiles across different ABI categories, TG levels were significantly higher in the ABI<0.9 group (200.69±31.23) compared to the ABI 0.9-1.2 (150.27±67.08) and ABI>1.2 groups (172.29±68.04 mg/dL), (p<0.0001). LDL levels decreased with increasing ABI and were higher in the ABI<0.9 group (72.71±31.54) and lower in the ABI >1.2 group (51.06±20.15 mg/dL, p=0.002). Similarly, VLDL levels were elevated in the ABI<0.9 group (48.65±12.54) compared to both ABI 0.9-1.2 (37.59±19.89) and ABI>1.2 (37.68±17.52 mg/dL, p = 0.001). The TC/HDL and HDL/LDL ratios were significant (p=0.003 and p=0.001, respectively), indicating potential cardiovascular risk among the ABI groups. Furthermore, Heart Rate (HR) was significantly elevated in the ABI <0.9 group 90.42±10.0 beats/min (p=0.001), suggesting a correlation with more severe arterial disease (Table 2).

Correlation between ABI and Lipid Profiles :

ABI was negatively correlated with TG levels (r=-0.267,

Table 2 — *Comparison of CAD markers based on ABI categories*

Variables	ABI (<0.9) (n=49)	ABI (0.9-1.2) (n=116)	ABI (>1.2) (n=45)	p-value
TC (mg/dL)	153.58±36.53	149.09±42.18	152.49±13.90	0.731
TG (mg/dL)	200.69±31.23	150.27±67.08	172.29±68.04	<0.0001*
HDL (mg/dL)	48.63±8.04	46.38±9.54	46.34±10.89	0.349
LDL (mg/dL)	72.71±31.54	68.25±36.08	51.06±20.15	0.002*
VLDL (mg/dL)	48.65±12.54	37.59±19.89	37.68±17.52	0.001*
TC/HDL	2.66±0.92	3.14±1.00	3.23±0.65	0.003*
HDL/LDL	1.09±0.50	1.53±0.80	1.58±0.64	0.001*
RBS (mg/dL)	209.33±86.91	162.73±62.32	171.76±60.01	<0.0001*
HR (per min)	90.42±10.0	88.11±12.19	81.79±11.10	0.001*

Abbreviations : TC: Total Cholesterol, TG: Triglyceride, HDL: High-density Lipoprotein, LDL: Low-density Lipoprotein, VLDL: Very Low-density Lipoprotein, TC/HDL: Ratio of Total Cholesterol and High-density Lipoprotein, HDL/LDL: Ratio of High-density Lipoprotein and Low-density Lipoprotein, RBS: Random Blood Sugar, HR: Heart Rate. The ANOVA test was used to compare the groups. *p<0.05 was considered as statistically significant.

p= 0.015). The TC/HDL and LDL/HDL ratios positively correlated with ABI (r=0.311, p=0.002, and r=0.339, p=0.0001, respectively), while the ABI was significantly negatively correlated with VLDL levels (r =-0.307, p= 0.003).

LDL was positively correlated (r=0.354, p<0.0001) with ABI. ABI was significantly negatively correlated with VLDL levels (r=-0.307, p=0.003). TC and LDL levels showed a positive correlation (r=0.731, p<0.0001); similarly, TG and VLDL showed a positive correlation (r=0.735, p<0.0001). In contrast, VLDL levels were negatively correlated with HDL levels (r=-0.390, p<0.0001). The TC/HDL ratio was positively associated with the LDL/HDL ratio (r=0.565, p<0.0001)(Table 3).

Relationship between Affected Vessels and CAD Severity :

With low ABI patients, the prevalence of vessel disease was as follows: Single Vessel Disease (SVD) in 24.48%, Double Vessel Disease (DVD) in 51.02%, and Triple Vessel Disease (TVD) in 24.48%. Regression analysis revealed a positive correlation between the number of affected vessels and the severity of CAD, with increasing beta coefficients indicating greater severity: SVD (β=0.84, p<0.0001), DVD (β=0.849, p<0.0001), and TVD (β=0.86, p<0.0001) (Table 4).

Discussion

In clinical practice and epidemiological studies, ABI serves as a crucial indicator of PAD[5-8]. Our study investigated the correlation between ABI values, the

Table 4 — *Vessel Disease Involvement of subjects with low ABI (<0.9)*

Variables	β-Coefficient	SE	t-value	p-value
Vessel disease (1)	0.84	0.003	250.97	<0.0001
Vessel disease (2)	0.85	0.002	366.21	<0.0001
Vessel disease (3)	0.86	0.003	256.94	<0.0001
ABI (vessel disease =2)	0.23	0.28	0.80	0.43
ABI (vessel disease =3)	0.76	0.28	2.67	0.02

Table 3 — *Correlation of CAD markers with ABI index*

Variables	TC(mg/dL)	TG(mg/dL)	HDL(mg/dL)	LDL(mg/dL)	VLDL(mg/dL)	TC/HDL	LDL/HDL	HR/Minutes	ABI
TC (mg/dL)	1	r=0.322 p<0.0001*	r=0.262 p<0.0001*	r=0.731 p<0.0001*	r=0.184 p=0.007*	r=0.446 p<0.0001*	r=0.558 p<0.0001*	r=-0.171 p=0.013*	r=0.056 p=0.422
TG (mg/dL)		1	r=0.027 p=0.701	r=-0.151 p=0.029*	r=0.735 p<0.0001*	r=0.286 p<0.0001*	r=-0.161 p=0.019*	r=0.119 p=0.087	r=-0.267 p=0.015*
HDL (mg/dL)			1	r=0.024 p=0.726	r=0.012 p=0.866	r=-0.390 p<0.0001*	r=-0.343 p<0.0001*	r=-0.025 p=0.717	r=-0.043 p=0.539
LDL (mg/dL)				1	r=-0.210 p=0.002*	r=0.406 p<0.0001*	r=0.904 p<0.0001*	r=-0.125 p=0.071	r=0.354 p<0.0001*
VLDL(mg/dL)					1	r=0.093 p=0.181	r=-0.223 p=0.001*	r=0.083 p=0.232	r=-0.307 p=0.003*
TC/HDL						1	r=0.565 p<0.0001*	r=0.157 p=0.023*	r=0.311 p=0.002*
LDL/HDL							1	r=-0.088 p=0.203	r=0.339 p<0.0001*
HR (/minutes)								1	r=0.292 p=0.005*

Abbreviations: TC: Total Cholesterol, TG: Triglyceride, HDL: High-density Lipoprotein, LDL: Low-density Lipoprotein, VLDL: Very Low-density Lipoprotein, TC/HDL: Ratio of Total Cholesterol and High-density Lipoprotein, HDL/LDL: Ratio of High-density Lipoprotein and Low-density Lipoprotein, RBS: Random Blood Sugar, HR: Heart Rate. The Pearson correlation coefficient was used to see the association between the two variables. *p<0.05 was considered as statistically significant.

extent and severity of CAD and the confounding factors influencing cardiovascular risk.

Our findings underscore a significant association between ABI and the severity of CAD, reflecting a complex interplay of cardiovascular risk factors. Previous studies have consistently demonstrated that lower ABI values are indicative of more extensive and severe CAD. Criqui, *et al* (2012)[9] and McDermott, *et al* (2005)[10] reported a higher incidence of multi-vessel CAD in individuals with lower ABI values, highlighting the role of ABI in predicting CAD severity.

The observed gender disparity, with a higher percentage of males in the lower ABI groups, aligns with previous research indicating a higher risk of PAD in males. The presence of calcification in both the lowest and highest ABI groups suggests different underlying pathophysiological mechanisms affecting arterial stiffness, which warrants further investigation[9,10].

This study highlights the multifactorial nature of PAD, with ABI significantly associated with Age, Smoking, Hypertension, Diabetes and Cardiovascular history. These factors contribute to the progression of PAD and underscore the importance of comprehensive risk factor management in patients with low ABI[11-13]. Similar to previous studies, our findings show a higher prevalence of smoking and hypertension in the ABI<0.9 group, which correlates with severe arterial occlusion and underscores the utility of ABI in assessing arterial blockages[14].

Our study identified a significant positive correlation between ABI and TC and LDL levels, which are established contributors to atherosclerosis and cardiovascular risk[15,16]. The relationship between TG and VLDL underscores their joint influence on lipid metabolism and cardiovascular risk[17]. Additionally, the negative correlation between ABI and random blood sugar levels highlights the detrimental impact of impaired glycemic control on vascular health, contributing to endothelial dysfunction and atherosclerosis[18].

In our cohort of patients with a low ABI, we observed a significant prevalence of multi-vessel CAD: SVD in 24.48%, DVD in 51.02% and TVD in 24.48%. These findings emphasize the significant burden of CAD in individuals with impaired peripheral arterial circulation, underscoring the need for comprehensive cardiovascular assessment and therapy in this population[19,20].

Regression analysis further supported our findings, revealing a significant correlation between the number of affected vessels and the severity of CAD, as indicated by increasing beta coefficients: SVD (β=0.84, p<0.0001), DVD (β=0.849, p<0.0001) and TVD (β=0.86, p<0.0001). These results suggest that lower ABI values, reflective of more severe PAD, are associated with a higher prevalence of multi-vessel CAD and increased CAD severity. This is consistent with prior research linking PAD severity to heightened cardiovascular risk and poorer clinical outcomes[21]. Studies by McDermott, *et al* (2005)[10] and a meta-analysis by Fowkes, *et al* (2008)[22] have similarly identified ABI as a robust predictor of CAD severity and mortality, emphasizing the clinical relevance of ABI assessment in cardiovascular risk stratification[10,22].

However, the relatively small sample size and specific demographic characteristics of the study population may restrict the generalizability of findings to broader populations. Future research should explore longitudinal outcomes and mechanistic insights further to understand the predictive value of ABI in CAD progression and guide personalized treatment approaches, including larger, more diverse cohorts to validate current findings.

Conclusion

The study reveals a strong correlation between ABI values and the severity of CAD. ABI values ≤0.9 are linked to increasing severity of CAD, with a higher prevalence of TVD, followed by DVD and SVD. This suggests that ABI ≤0.9 is a robust predictor of CAD severity, indicating extensive arterial involvement and heightened cardiovascular risk in these individuals. Individuals with ABI values >0.9 and <1.2 also show a correlation with TVD, indicating an intermediate risk profile. The study emphasizes the importance of ABI assessment in clinical practice for identifying individuals at heightened cardiovascular risk.

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