Ankle Brachial Pressure Index (ABI), A Cardiovascular Risk Prediction Tool

Anwar H Siddiqui*, Nazia Tauheed, Rajiv Gulati*, Akif Ahsan****, Anjum Pervez*** , Najmul Islam******

*Deptt of Physiology, **Deptt of Anesthesiology & Critical care, ***Deptt of Medicine, and ****Deptt of Biochemistry, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh

Abstract: Background: In recent years significant attention has been paid in identifying markers of increased cardiovascular risk, in particular the coronary artery disease. The ankle-brachial pressure index (ABI),an easily accessible, inexpensive bedside test can be a significant tool to assess the vascular risk in symptomatic and asymptomatic cardiovascular patients Objective: To determine the association between an abnormal ankle brachial index (ABI) and coronary artery disease (CAD). Method:The study population included 150 subjects divided in two groups, 80 patients with AMI and 75 age and sex matched healthy subjects as controls. Ankle Brachial Pressure Index (ABI) was measured in all the subjects along with their LDH level.Result: A significantly lowABI (<0.9) was observed among the AMI patients as compared to healthy controls. Moreover the ABI showed negative correlation with the level of LDH in the AMI patient. Conclusion: ABI calculation would be able to identify more patients at high riskand as such it should be considered routine investigation for cardiovascular risk prediction. A follow up study with large cohort will help in stratification of individual risk of developing coronary artery disease (CAD)

Key words:ABI (Ankle brachial index), AMI (Acute Myocardial Infarction)), Lactate Dehydrogenase (LDH)

Author for correspondence: Anwar H Siddiqui, Dept. of Physiology, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, E-mail:anwar@gmail.com

Introduction: Ischemic heart disease (IHD) is the major cause of morbidity and mortality all over the world[1]. It is usually attributable to atherosclerotic obstruction of coronary vessels and clinically presents as a spectrum of signs and symptoms ranging from angina pectoris to acute myocardial infarction (AMI), more aptly termed as acute coronary syndrome[2].A number of risk factors are known to predispose patients to IHD. Some of these cannot be modified, for example age, gender and family history. Modifiable risk factors include dyslipidemia, hypertension, smoking, diabetes mellitus, obesity, physical inactivity, alcohol consumption and psychological factors[3].These conventional risk factors do not account for all cases of atherosclerotic coronary artery disease (CAD) and myocardial infarction (MI) still occurs in individuals having no obvious traditional risk factor. These observations under-score the need to identify an additional marker for coronary atherosclerosis.Although several tools have been proposed[4-6]frequently the clinical utility of measuring such markers remains uncertain for several reasons, including costs, low reproducibility, conflicting studies or lack of confirmatory studies, and lack of measurement standardization[5].The presence of peripheral arterial disease measured non-invasively by ankle brachial index (ABI) is a risk marker for coronary artery disease (CAD). The ankle brachial index (ABI), a ratio of ankle systolic blood pressure to brachial systolic pressure, is used in clinical practice to assess the patency of the lower extremity arterial system and to screen for the presence of occlusive peripheral arterial disease. Epidemiological and clinical studies have found that low ABI levels are associated with cardiovascular risk factors, coronary and carotid artery disease and predict cardiovascular and overall mortality[7].

The objective of this study was to determine the association between an abnormal ankle brachial index (ABI) and the presence of AMI and to correlate it with the level of tissue damage in AMI, as evident by the LDH level.

Material and Method: The study consisted of 80 patients (52 men and 28 women) with a mean age of 49.50 ± 6.28 years admitted in the Coronary Care Unit of Jawaharlal Nehru Medical College Hospital, Aligarh Muslim University, Aligarh, India with the diagnosis of AMI. The diagnosis of AMI was based on a history of prolonged ischemic chest pain, which lasted for up to 3 hours, ECG changes (ST elevation of 2 mm or more in at least two leads) and elevated

creatine kinase isoenzyme MB (CK-MB) and troponin T within 12 h after the onset of pain. The control group consisted of 70 healthy, age matched subjects, 48 men and 22 women, recruited from the institution. The study was duly approved by the Board of Studies/Institutional Ethical Committee and a valid informed consent was obtained from all the subjects (including both the cases and controls).

Inclusion criteria: Patients with diagnosis of AMI and admitted within 24 hours of onset of symptom.

Exclusion criteria: Patients/Control with any history of diabetes mellitus, asthma, smoking, oral antioxidant or vitamin intake.

Measurement of Ankle Brachial Pressure Index: The Ankle Brachial Pressure Index (ABI) is the ratio of the blood pressure in the lower legs to the blood pressure in the arms.American Heart Association recommendations were taken into consideration for ABPI calculation[8]. After resting the subjects for 5 minutes in asupine position, brachial artery systolic and diastolicblood pressure was recorded in both arms using amercury sphygmomanometer. Appropriate sized bloodpressure cuffs were applied over each brachial artery.

The cuff was rapidly inflated to 20 mmHg above the systolic pressure recorded by palpatory method in each arm and then deflatedat a rate of 2 mm per heart beat to the lowest evenreading. Highest systolic reading was measured in botharms as the pressure at which the first sustained soundwas audible. Diastolic pressure was recorded at thedisappearance [phase five] of Korotkoff sounds. Thehigher of the two arms' pressure was taken as indexarm. Two more readings were taken on the same armand the average was taken as the index systolic bloodpressure in the arm.In all cases, ankle pressure in both ankles wasmeasured by Doppler with 8 MHz probe which is theGold standard. The cuff was positioned on the ankleproximal to the malleoli. The pulse was located with aDoppler probe and the cuff inflated until the pulse wasobliterated; the cuff was deflated and the pressure wasrecorded at the point when the pulse

reappeared. Theleg with lower systolic pressures was taken as index leg.Within the index leg dorsalispedis artery pressure wastaken as index ankle pressure if it was higher than theposterior tibial and vice versa. Two more readings weretaken on the same artery and the average wasrecorded.
ABI (ankle brachial index) was calculated by dividing theaverage systolic blood pressure of the index ankle arteryby the average systolic blood pressure of the index arm.A resting ABI value ≤0.90 defines the presence of peripheral arterial disease and it has a sensibility of about 95% in identifying the presence of a hemodynamically significant arterial stenosis at angiography between heart and foot and near 100% specificity in excluding a normal subject[9]

LDH measurement: Peripheral blood samples were obtained from all thepatients and controls. Sera were separated by centrifuging the blood sample at 3000 rpm for 15 minutes. The analysisSerum LDH was determined with standardtechniques using Cobas 8000 Analyzer (Roche DiagnosticsGmbH, Germany).

Statistical Analysis: All data were expressed as mean ± SD. The statistical significance was evaluated by Student's t- test using Statistical Package for the Social Sciences (SPSS) ver 17.0.Pearson's correlation coefficients were determined between the measured parameters at 5% level of significance. A p-value of <0.05 was considered statistically significant

Result: Table 1.shows the demographic and clinical characteristics of normal healthy controls and AMI patients. The control group consisted of 48 males and 22 females with a mean age of 46.75 ±7.69 years whereas among the 80 AMI patients there were 52 males and 28 females with mean age 49.50 ± 6.28 years.

AMI patients had a mean weight of 65.80 ± 7.59 kg which was significantly higher than that of

controls where the mean weight was 60.17 ± 7.73 kg. BMI was significantly high in AMI group as compared with control.A significant rise in both systolic as well as diastolic blood pressure was seen in AMI patients as compared to control.

Table 1: Demographic data and Clinical characteristics of the healthy group and AMI patients

Variables	Control (N=70)	Cases Of Ami (N=80)
Age (years)	46.75 ±7.69	49.50 ± 6.28
Male	68.57%	65%
Female	31.43%	35%
Weight (kg)	60.17 ± 7.73	65.80 ± 7.59*
Height (cm)	163.06 ± 6.38	163.82 ± 5.93
BMI (Kg/m2)	22.44 ± 2.09	25.02 ± 3.01*
Waist-to-hip ratio	0.84 ± 0.05	0.96 ± 0.11*
Systolic BP(mm Hg)	119.75 ± 7.50	132.20 ± 8.51*
Diastolic BP(mmHg)	79.85 ± 6.63	82.95 ± 6.21*
Hypertension	-	52%
Continuous variables are presented as mean ± SEM and the other variables are shown as percentage of patients.* represents 'p' value <0.05		

Table 2.shows level of cardiac markers (CK, CK-MB, troponin T, LDH) were significantly higher AMI groups when compare to control subjects.

The mean ABI was 1.06±0.22 in the control group and 0.98 ± 0.24 in the AMI patients.It was not significantly different in two groups (p=0.428, Figure 1). However the frequency of patients with lower ABI (≤0.9) was significantly higher compared to the frequency of control patients with lower ABPI (33.75% and 5.72%, respectively; p=0.02, Table 3).

The LDH level of AMI patients showed a significant negative correlation with the ABI (r=-0.46, p=0.004)

Discussion: Atherosclerosis and its complications are the leading causes of mortality and morbidity worldwide. Many risk factors have been defined for atherosclerosis and CAD.Interest in the use of formulas and tables to predict an individual's risk of a subsequent cardiovascular event is increasing. To date, these have been based on conventional risk factors, such as cigarette smoking, hypertension, and hypercholesterolemia, and have used data from large observational studies, including the Framingham study[10].

Table 2: Cardiac biomarker levels in the healthy group and AMI patients

Markers	Control (N=70)	Cases Of Ami (N=80)
CK (IU /L)	73 ± 15.6	126 ± 26.5*
CK-MB (IU/ L)	12.5 ± 2.8	97 ± 7.8*
Troponin T(ng/ml)	0.021 ± 0.005	1.27 ± 0.14*
LDH (IU/L)	256.49± 9.30	181.63 ± 5.77*

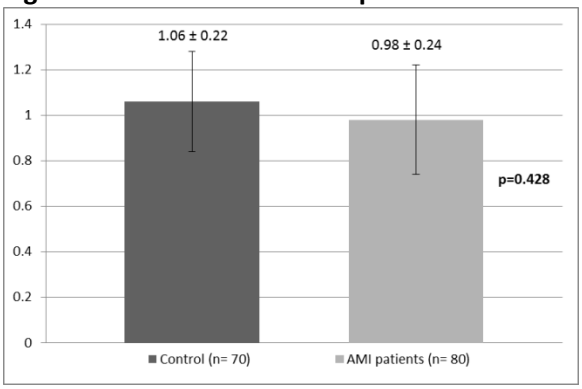
Table 3: Distribution of ABI groups within patient and control groups

		Patient		Control		Total	
		N	%	n	%	n	%
ABI	≤0.9	27	33.75	4	5.72	31	20.67
	>0.9	53	66.25	66	94.28	119	79.33
	Total	80	100	70	100	150	100
Chi-Square=5.421 ; p=0.020*							

Such predictions are increasingly being used in clinical practice to determine whether the benefits of preventive treatment (for example, aspirin administration) outweigh the potential side effects of such interventions. The results of the present analyses suggest that the ABI may add to the sensitivity of the present risk factor assessment tests. ABI value is implemented as an easy and non-invasive method for early determination of atherosclerotic lesions.Many studies have shown that atherosclerosis incidence increases in cases with ABI ≤0.9. Low ABI values in patients with CAD were related

the existence of atherosclerosis and the number of affected coronary arteries[11,12]. In the current study, the mean ABI was 0.98±0.22 in the patient group and 1.06±0.18 in the control group. No statistically significant differences between the patient and control groups were observed in terms of ABI (Figure 1.).Relatively small number of cases may cause these different results. Moreover, there were markedproportions who lacked important risk factors such assmoking, hypertension and diabetes.However the frequency of low ABPI (≤0.9) was significantly higher compared to the control patients (Table 3).

Fig 1: ABI of control and AMI patients.



In addition it has been seenin the study that the ABI of the AMI patients shows a significant negative correlation with the LDH level. LDH, a marker of tissue degeneration can be used as a surrogate marker for the severity of the AMI.Experimental study in ratshas shown that elevation in the LDH enzyme activity inthe serum correlated with a decrease in the activity of cardiac muscle LDH[13].

Data from ARIC (Atherosclerosis Risk In Communities)and other studies suggest that the average risk of futurecoronary heart disease (CHD) events increases withdecreasing ABI as a continuous but not linear function.Similar results have been reported for exertional legpain, for carotid intima media thickness and coronaryartery calcium[14].The choice of relevant ABI cut off atwhich risk factor modification therapy should beinstituted to reduce further CAD risk should be based onabsolute rather than relative risk of future CAD events.

An ABI ≤0.9 has been consistently associated with a 2 to 5 fold increase in all-cause death and a 3 to 8 fold increase in cardiovascular death when compared with an ABI >0.9[15-18].However, there are still some issues to be addressed about the use of the ABI as a diagnostic tool. First, there is limited research on how the risk of vascular events varies across the whole range of ABI in the general population.Second, there is no ABI cut point that is universally accepted as being the best predictor of cardiovascular events, although for screening purposes, it may be hypothesized that an ABI ≤0.9 is likely to be more sensitive than a lower cut point. Finally, although change in ABI has been related to worsening peripheral arterial disease[19] or outcome after vascular operation[20],its predictive value for subsequent vascular events has not been investigated in any detail. Further research with large sample size and longer duration of study is required to reach a substantial conclusion.

Conclusion: ABI calculation would be able to identify more patients at high riskand as such it should be considered routine investigation for cardiovascular risk prediction. A follow up study with large cohort will help in stratification of individual risk of developing coronary artery disease (CAD)

Limitations of the study:The study has been conducted on a relatively small sample size and as such the conclusion arrived may not be sufficient to be implemented on a general population. A larger sample size might provide more conclusive evidence. The severity of AMI in the study would have been best described in terms of the involvement of the coronary artery viz. single vessel disease, double vessel disease and triple vessel disease but unfortunately due to unavailability of a catheterization lab in our setup we had to use LDH as a surrogate marker.

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