



## CORRELATION OF ANKLE BRACHIAL INDEX(ABI) WITH NUMEROUS CLINICAL PARAMETERS IN PATIENTS WITH PERIPHERAL ARTERY DISEASE (PAD)

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Article Received on  
18 December 2018,

Revised on 08 Jan. 2019,  
Accepted on 29 Jan. 2019

DOI: 10.20959/wjpps20192-13212

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### INTRODUCTION

Peripheral arterial disease (PAD) is the occlusive disease of arteries distal to the aortic bifurcation. The prevalence of PAD in the lower limbs in a general population >55 years of age is between 10% and 25% and it increases with age. Majority of affected population have asymptomatic disease.<sup>[1]</sup> Peripheral arterial disease, whether symptomatic or asymptomatic, is a risk factor for non-fatal and fatal coronary disease and cerebrovascular events. Patients with PAD alone have the same relative risk of death from cardiovascular cause as those with coronary or cerebrovascular disease. Risk of death in patients of PAD within 10 years is 4 times more than those without the disease. Several studies have shown that the ankle brachial index (ABI), an index for occlusive vascular disease, is now considered an independent predictor of coronary and cerebrovascular morbidity and mortality.<sup>[2,3]</sup>

Our study in an Indian population was carried out to correlate and substantiate the relation of PAD with coronary artery disease (CAD) using the ABI.

## METHODS

This study was conducted on patients of type 2 diabetes mellitus at Department of medicine and Radiology in Vardhman Mahavir Medical college and Safdarjung Hospital, New Delhi a tertiary care hospital in northern India. Each patient gave written, informed consent to participate in the study. 70 consecutive patients, age between 40-70 years, who satisfied the inclusion and exclusion criteria, were enrolled. Subjects were recruited from medicine OPD and wards. They were subjected to detailed history and physical examination with investigation.

### Inclusion criteria

1. Type 2 Diabetic diagnosed by American Diabetic Association (ADA) Criteria 2011.
2. Duration- any duration.

### Exclusion criteria

1. Patient with aortoiliac and iliofemoral obstruction.
2. Patient with aortoarteritis.
3. Buerger's disease.
4. Smokers.
5. Collagen vascular disease.

A detailed clinical history of the patients was taken followed by a detailed clinical examination, which was recorded in a proforma sheet. The diagnosis of intermittent claudication (IC) was made based on the response to the questions in the proforma sheet.

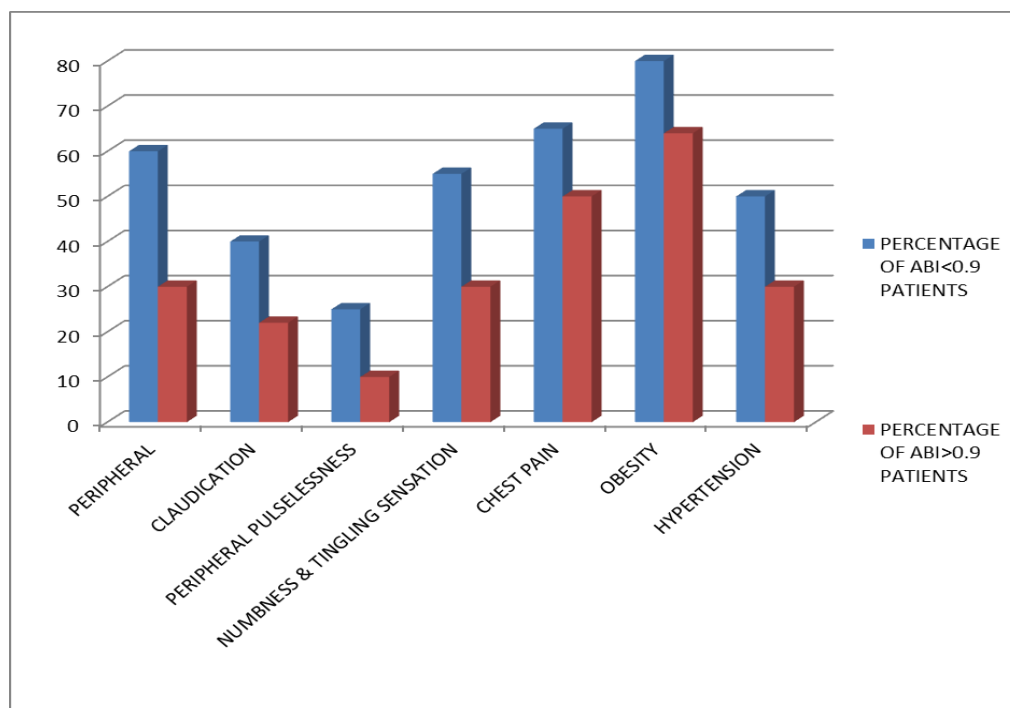
The measurement of ABI was done with the help of VP-1000 from Colins, Japan which works on the oscillometric principle. Blood pressure cuffs are tied to all four limbs and systolic pressure of all the limbs is measured at the same time and the ABI is calculated for each side.

## RESULTS

The present study consisted of a study population of 72 patients. Based on ABI <0.9, PAD was diagnosed in 22 patients and 50 patients had ABI >0.9 on both sides and hence were considered normal. (Table 1 & Figure 1).

**STUDY OF CLINICAL PROFILE IN RELATION TO ABI PATIENTS****Table 1: Percentage Of Clinical Profile Shown In Abi<0.9/Abi>0.9 Patients.**

Group	PERIPHERAL PAIN	CLAUDICATION	PERIPHERAL PULSELESSNESS	NUMBNESS & TINGLING SENSATION	CHEST PAIN	OBESITY	HYPERTENSION
PERCENT AGE OF ABI<0.9 PATIENTS	60	40	25	55	65	80	50
PERCENT AGE OF ABI>0.9 PATIENTS	30	22	10	30	50	64	30
P-VALUE	0.05	0.244	0.432	0.321	0.234	0.054	0.049

**Figure1: Relation Of Abi With Clinical Parameter.**

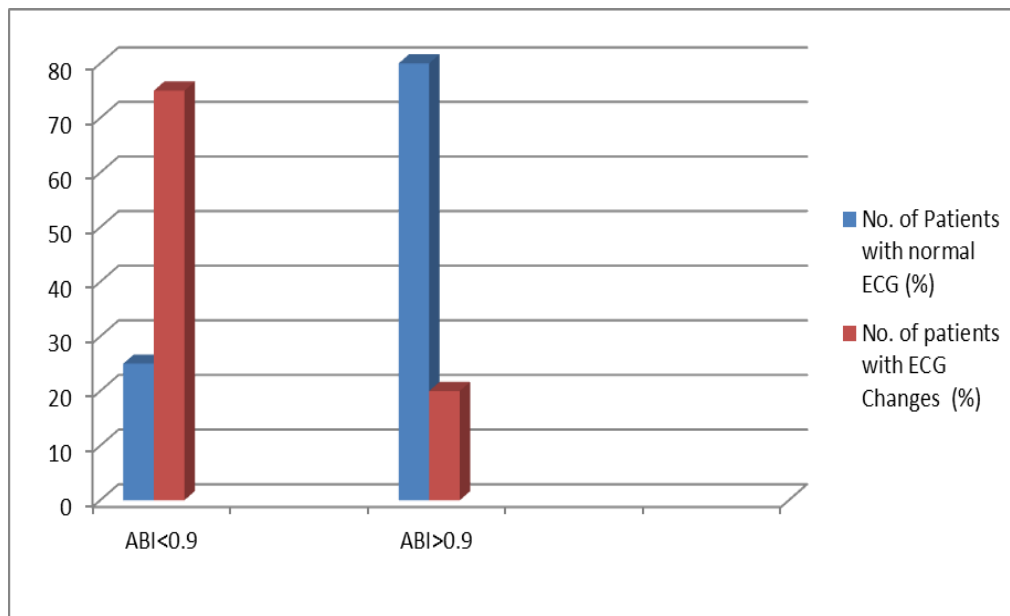
Graph showing clinical profile in ABI<0.9 patients more than ABI>0.9 patients

**RELATIONSHIP OF ABI<0.9 WITH ECG CHANGES FOR CAD**

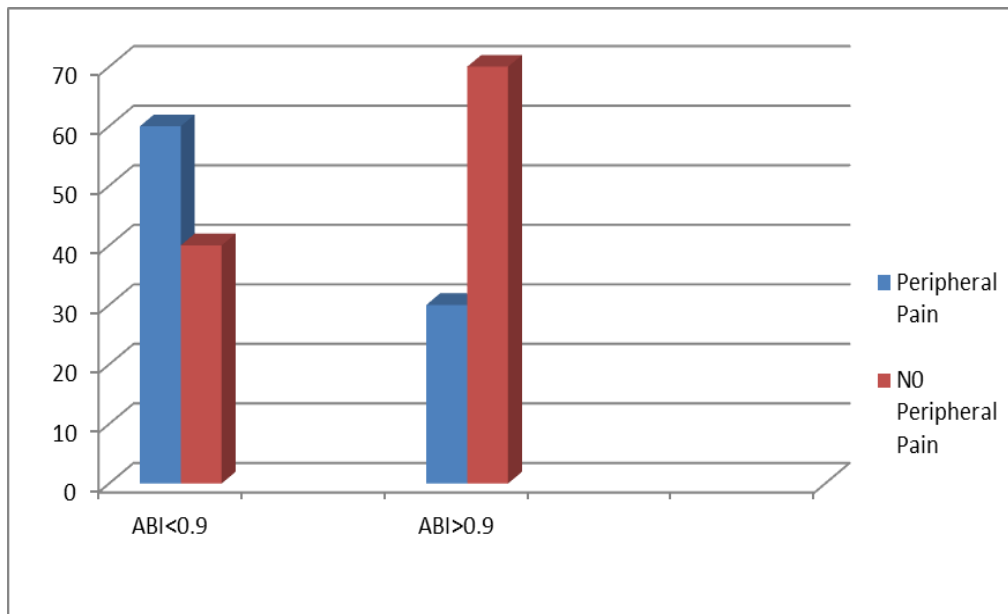
Among ABI<0.9 Patients 75% have ECG changes suggestive of CAD while only 20% have ECG changes. Suggestive of CAD in ABI>0.9 group of patients and P-Value is 0.016 which is significant.

**Table 2: Correlation Of Ecg Changes Of Cad In Abi Patients.**

Group	No. of Patients with normal ECG (%)	No. of patients with ECG Changes (%)	Total	P-Value
ABI<0.9	5(25 %)	15(75%)	20(100%)	0.016
ABI>0.9	40(80)%	10(20%)	50(100%)	0.172

**Figure 2: Correlation Of Abi With Ecg Changes For Cad.****PERIPHERAL PAIN****Table 3: Correlation Of Abi<0.9 With Peripheral Pain.**

Group	Peripheral Pain	N0 Peripheral Pain	Total	P-Value
ABI<0.9	12(60)	8(40)	20(100%)	0.05
ABI>0.9	15(30)	35(70)	50(100%)	0.218



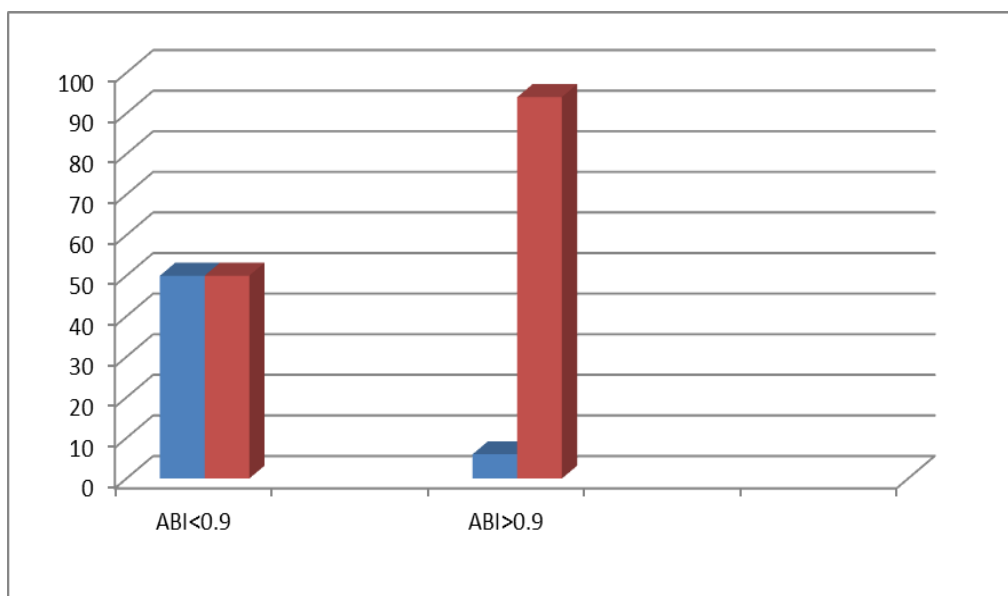
**Figure 3: Correlation Of Abi With Peripheral Pain.**

Among ABI < 0.9 patients 60% have peripheral pain while only 30% have peripheral pain in ABI > 0.9 group of patients and P-Value is 0.05 which is not significant.

#### RELATIONSHIP BETWEEN PAD AND DIABETIC RETINOPATHY(DR)

**Table 4: Correlation Of Abi < 0.9 With Diabetic Retinopathy(Dr)Changes.**

Group	DIABETIC RETINOPATHY Changes	NO DIABETIC RETINOPATHY Changes	Total	P-Value
ABI < 0.9	10(50)	10(50)	20(100%)	0.058
ABI > 0.9	3(6)	47(94)	50(100%)	0.228



**Figure 4: Correlation Of Abi With Diabetic Retinopathy.**

Out of 20 patients 10(50%) have diabetic retinopathy in PAD group but only 3(6%) out of 50 have diabetic retinopathy. P-Value is 0.058 and this is Non-Significant.

## DISCUSSION

Diabetes mellitus affects nearly every vascular bed in the body. Patients with diabetes mellitus suffer from premature and severe atherosclerosis<sup>[13]</sup> leading to morbidity and mortality. The Framingham study pointed out the cause of the much higher incidence of cardiovascular complications in diabetic patients. These individuals have a much higher serum concentration of lipids and a higher occurrence of obesity leading to advanced atherosclerosis. Peripheral arterial disease (PAD) often detected late in patients is a manifestation of atherosclerosis characterized by atherosclerotic occlusive disease of the lower extremities. Reasons for a higher incidence of PAD in diabetics include inflammation, which has been established as both a risk marker and perhaps a risk factor for atherothrombotic disease states.<sup>[3,4]</sup>

Most patients with diabetes demonstrate abnormalities of endothelial function and vascular regulation leading to derangement of Nitric Oxide (NO) bioavailability. Diabetes is associated with significant abnormalities in vascular smooth muscle cell (VSMC) function which trigger proatherogenic activity. In diabetics, platelet uptake of glucose is unchecked in the setting of hyperglycaemia and results in increased oxidative stress. Consequently, platelet aggregation is enhanced.<sup>[4]</sup>

The present study is a cross-sectional study performed on 70 type 2 diabetic patients 42.9% men and 57.1% women, attending medicine O.P.D & WARD. Any other disease which concludes peripheral arterial disease were excluded. The subjects were screened for peripheral arterial disease (PAD) by subjecting them to colour Doppler ultrasound for the presence of PAD on the basis of which the study group was divided into two viz. patients having PAD and patients not having PAD. The presence of various macrovascular (cardiovascular and nephropathy) and microvascular (retinopathy and neuropathy) risk factors in the study subjects was analyzed. These groups were further analyzed for correlation.

Methods for the diagnosis of PAD are

1. BMI
2. Duplex Sonography
3. Magnetic Resonance Angiogram
4. Contrast Angiography

Above 2,3,4 are time consuming and costly. ABI is a simple economical test.

1. ABI may be normal in patients with aorto –iliac disease and in well collateralized patients at rest.
2. It may be artifactually normal or “Supernormal” in patients with arterial calcification.

ABI is a non-invasive test has sensitivity of about 90% and specificity of about 98% for detecting PAD before any clinical presentation of diabetic patients. The ABI is measured by placing the patient in a supine position for 5 minutes. Sphygmomanometer cuff is positioned above the elbow and above the ankle for respective measurement. The cuff is inflated around 20 mm of Hg above the value of BP is noted, when flow just begins to be picked up with colour Doppler pulse wave. Systolic pressure is measured in both arms, and the higher value is used as the denominator of ABI. Systolic blood pressure of the dorsalispedis or the posterior tibial artery is then measured by placing the cuff just above the ankle. The higher value is the numerator of the ABI in each limb.<sup>[6-9]</sup>

#### **Anthropometric parameters**

The correlation of obesity with diabetes is well known. In India, the prevalence of obesity among type 2 diabetics is reportedly lower than in Western studies. Normal BMI, for Indian population is considered to be less than 23 kg/m<sup>2</sup>. In the present study, mean BMI were 26.333 ± 1.89 kg/m<sup>2</sup>. for patients with PAD and in without PAD. were 25.048 ± 2.66 kg/m<sup>2</sup>. (P=0.08) is non significant. The prevalence of obesity (BMI > 25 kg/m<sup>2</sup>) in PAD patients was 80% as compared to 64% in non-PAD patients.

Sixteen of the total 20 patients with (BMI >25 kg/m<sup>2</sup>) 80% had PAD and 32 of 50 patients with BMI > 25 kg/m<sup>2</sup> (64%) in non-PAD. This suggests that factors other than obesity also play a significant role in the occurrence of PAD in type 2 diabetic patients. (P=0.08). In the Fremantle diabetes study<sup>158</sup>, mean BMI in the non-PAD group was 28.2 ± 4.5 kg/m<sup>2</sup> whereas in the PAD group it was 29.7 ± 5.2 kg/m<sup>2</sup> (p<.0001). Obesity was found to correlate significantly in the study. Walters et al. in 1992<sup>153</sup> also found obesity to be a significant predictor of PAD. Indian studies (CUPS17, Agrawal et al<sup>191</sup>) did not find any correlation of PAD with obesity.

In our study there was no correlation found between obesity and PAD. Our results along with those of other Indian studies like CUPS17 and the study by Agrawal et al<sup>191</sup>, suggest that

unlike in Western populations, obesity does not appear to be a significant risk factor for PAD in Indian diabetics.

#### ***Blood glucose control parameters***

The majority of study patients had uncontrolled diabetes. The mean HbA1c was  $8.308 \pm 1.70074\%$ . On comparing the two groups, mean HbA1c was  $7.01 \pm 0.86$  mg% in the non-PAD group as compared to  $7.92 \pm 0.86$  in the PAD group. Using a cut off level of  $>6.5$  mg% for poor control, 76% had poor glycaemic control in the non-PAD group compared to 95% in the PAD group. The mean fasting and post-prandial blood glucose levels in our study were  $173.19 \pm 80.901$  mg% and  $245.19 \pm 100.511$  mg% Blood glucose levels were comparable in the PAD and the non-PAD groups. HbA1C level was found to significantly high in patients with PAD ( $P < 0.05$ ). as compared to those without PAD.

Walters et al. and Janka et al.<sup>156</sup> also found such an association as blood sugar values were found to be significant predictors of PAD. These studies suggest a relationship between inferior glycaemic control and PAD. Our findings, statistically significant similar to previous study. This suggest that HbA1c and poorer glycemic control plays a significant role in occurrence of PAD in type 2 DM.<sup>[10]</sup>

#### ***Peripheral neuropathy***

In our study 12(60%) patient out of 20 peripheral neuropathy in  $ABI < 0.9$  group, but only 15 (30%) out of 50 had neuropathy in  $ABI > 0.9$  group. ( $P = 0.05$ ) significant.

A study done in Arab medical university by Escobedo J Rana JS et.al. Of 624 type 2 diabetes patient showed peripheral neuropathy in 47.1% of patients.

#### **Association of Clinical profile in PAD patient**

Peripheral pain was present in 60% of PAD group compared to 30% in non PAD group. Claudication seen in 40% PAD group and 22% in non PAD group. Peripheral pulselessness seen in 25% PAD group and 25% non PAD group. Numbness and Tingling sensation seen in 55% in pad group and 35% in non PAD group. Chest pain in 65% PAD group and 50% non PAD group. Hypertension in 50% PAD AND 30% NON PAD. Our study show that increased clinical complication in PAD compared to non PAD.<sup>[12]</sup>



### CAD and PAD association

The prevalence of CAD (ECG changes based on Minnesota codes) like MI was 75% in PAD patients and 20% in non-PAD patients. Our study shows higher prevalence of CAD statistically significant ( $p=0.016$ ) may be due to higher age of selection and other comorbid association with PAD in Type 2 diabetes mellitus and less number of subject in study. However this association needs to be confirmed in larger studies.

The Fremantle diabetes study<sup>158</sup> was a prospective study to assess the association between PAD and CAD. This study found that a low ABI was associated with a 67% increase in the risk of cardiac death.

Similarly, the Cardiovascular Health study<sup>164</sup>, a prospective study to evaluate the association of PAD and CAD, enrolled 5,888 participants above 65 years of age. ABI was measured at the baseline. All participants had a detailed assessment of prevalent CVD and were contacted every 6 months to assess total mortality and CVD events (including CVD mortality, fatal and nonfatal myocardial infarction, congestive heart failure and angina.)

The crude mortality rate at 6 years was highest (32.3%) in those with prevalent CVD and a low ABI, and lowest in those with neither of these findings (8.7%). The risk for incident congestive heart failure (relative risk [RR]=1.61) and for total mortality (RR=1.62) in those without CVD at baseline but with a low ABI remained significantly elevated even after adjustment for cardiovascular risk factors.

For each 0.1 decrement in the ABI below 1.0, event rates increased. Even within this group, a low ABI was associated with increased age- and gender-adjusted risk of total and CVD mortality and remained independently associated with CVD mortality.

In the CUPS study<sup>17</sup>, the prevalence of CAD was not found to be significantly higher in the PAD group as compared to the non-PAD group. However, a similar study from South India by Krishaswamy *et al*<sup>192</sup> showed that PAD was common in elderly people with coronary artery disease. All patients in study were admitted to geriatric ward and a cross sectional evaluation was done. They recruited 80 people aged 60 years or above with coronary artery disease. PAD was found to be present in 19 patients (23.7 %).

In another study done by Leng *et al*<sup>193</sup> 1,592 subjects aged 55-74 years were selected randomly and the presence of peripheral arterial disease was determined and classified

into claudicants, major and minor asymptomatic patients. This cohort was followed prospectively over 5 years for subsequent cardiovascular events and death.

Claudicants had a significantly increased risk of developing angina compared with normals (RR : 2.31, 95% CI : 1.04-5.10), and asymptomatic subjects had a slightly increased risk of myocardial infarction and stroke. Deaths from cardiovascular disease were more likely in both claudicants (RR : 2.67, 95% CI : 1.34-5.29) and subjects with major (RR : 2.08, 95% CI : 1.13-3.83) or minor asymptomatic disease (RR : 1.74, 95% CI : 1.09-2.76).

At baseline 288 subjects (18.2%) had an ABI index  $\leq 0.9$ . After five years, subjects with an index  $\leq 0.9$  at baseline had an increased risk of non-fatal myocardial infarction (RR 1.38, 95% CI 0.88 to 2.16), stroke (RR 1.98, 95% CI 1.05 to 3.77), cardiovascular death (RR 1.85, 95% CI 1.15 to 2.97), and all cause mortality (RR 1.58, 95% CI 1.14 to 2.18) after adjustment for age, sex, coronary disease, and diabetes at baseline.<sup>[13,15]</sup>

A higher incidence of CAD has been shown in patients with PAD by several studies. However CUPS study<sup>17</sup>, a cross-sectional study from South India, failed to find a correlation between CAD and PAD.

## CONCLUSIONS

There is a definite and strong correlation between PAD and CAD. In view of the increasingly aging population and associated increase in atherosclerotic vascular disease, confrontation with patients of PAD will increase, which however, continues to be under diagnosed and under treated. The awareness and implementation of ABI in general clinical practice is poor. A simple, inexpensive test like ABI can improve the diagnosis of PAD in clinical practice and thus help in preventing CAD and consequent death by a range of medical therapies. Correct diagnosis and supervision of patients with these disorders is important for the prevention of local progression of the disease and effective secondary prevention of any future coronary and cerebrovascular events.

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