

Ankle Brachial Pressure Index Correlation with Diastolic Blood Pressure, Dyslipidemia and Anthropometric Measurement in Patients of Essential Hypertension

Varun Shetty¹, H R Jain², G S Singh², S Parekh², S Shetty³

¹Associate Professor, Department of Medicine, Padmashree Dr. D.Y. Patil Medical College, Navi Mumbai, Maharashtra, India, ²Postgraduate, Department of Medicine, Padmashree Dr. D.Y. Patil Medical College, Navi Mumbai, Maharashtra, India, ³Professor, Department of Medicine, Padmashree Dr. D.Y. Patil Medical College, Navi Mumbai, Maharashtra, India

Abstract

Background: Hypertension is one of the most common worldwide diseases affecting humans. Due to the associated morbidity and mortality and the cost to the society, it is an important public health challenge. Therefore, health care professionals must not only identify and treat patients with hypertension but must also promote healthy lifestyle and preventive strategies to decrease the prevalence of hypertension in general population.

Methods: Patients after satisfying inclusion criteria given below were divided into three groups as Stage I hypertensive, Stage II hypertensive (according to Joint National Committee [JNC] VII guidelines) and a control or the normotensive group. These are: (1) Essential hypertensive (according to JNC-VII) above 18 years of age, (2) Patients willing to give a written valid informed consent, (3) Control group included normotensive subject of either age above 18 years of age.

Result: Out of the 93 subjects, 16 from Stage I had a coronary artery disease (CAD) and 20 from Stage II had a CAD. At the same time 15 from Stage I did not have a coronary vascular disease, and 11 from Stage II also did not have a CAD. This study shows that an inverse correlation with stage of hypertension, i.e., with an increase in blood pressure there is a decrease in ankle brachial pressure index (ABPI) and increased risk of atherosclerosis.

Conclusion: ABPI is simpler and safe alternative, but the confirmative IS coronary angiography. It cannot replace coronary angiography in the assessment of risk or prognosis of CAD, but it can help clinician to decide that the hypertensive patient is at higher risk for CAD and may help from coronary angiography.

Key words: Ankle brachial pressure index, Atherosclerosis, Coronary artery disease, Dyslipidemia, Hypertension

INTRODUCTION

Cardiovascular diseases caused 2.3 million deaths in India in the year 1990; this is projected to double by the year 2020. Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths in India.¹⁻⁸

Global burden of hypertension is rising and projected to affect 1.5 billion persons – one-third of the World's population - by the year 2025. Currently, high blood pressure (BP) causes about 54% of stroke and 47% of ischemic heart disease (IHD) worldwide [1]. Half of this disease burden is in people with hypertension; the other half is in people with lesser degrees of high BP (prehypertension). Thus, high BP remains the leading cause of death worldwide and one of the world's great public health problems.

Although there is a dramatic age-related increase in prevalence of hypertension several important cardiovascular risk factors, particularly obesity, nutrient intake, physical inactivity, and diabetes mellitus also relate to the likelihood of developing hypertension.⁹⁻¹⁵

Access this article online



www.ijss-sn.com

Month of Submission : 03-2017
Month of Peer Review : 04-2017
Month of Acceptance : 05-2017
Month of Publishing : 05-2017

Corresponding Author: Dr. Varun Shetty, Plot No 247, 2nd Floor, Gokuldham, Sector 21, Nerul, Navi Mumbai, Maharashtra, India.
Phone: 91-9833991811. E-mail: shettyvarun81@gmail.com

The Framingham Heart study has estimated that individual's normotensive at age 55 years have a 90% lifetime risk of developing hypertension.² Hypertension represents a potent risk factor for cardiovascular, peripheral vascular and renal disease. The higher the BP, the greater is the likelihood of myocardial infarction, heart failure, stroke, and kidney disease.

The Framingham risk score 18-91 is often considered the reference standard but has limited accuracy, tending to overestimate risk in low-risk populations and underestimate risk in high-risk populations.¹⁰

The incorporation of other risk markers, such as the metabolic syndrome and plasma C-reactive protein^{12,13} has had partial success in improving prediction, and attention also is being given to indicators of asymptomatic atherosclerosis, such as coronary artery calcium, carotid intima-media thickness, and the ankle brachial pressure index (ABPI), or simply ankle brachial index (ABI).¹⁶⁻²⁴

The ABI, which is the ratio of systolic pressure at the ankle to that in the arm, is quick and easy to measure and has been used for many years in vascular practice to confirm the diagnosis and assess the severity of peripheral artery disease (PAD) in the legs.

Most commonly the ABI is calculated by measuring the systolic BP (SBP) in the posterior tibial and/or the dorsalis pedis arteries either in both legs or 1 leg chosen at random (using a Doppler probe or alternative pulse sensor), with the lowest ankle pressure then divided by the brachial SBP. In addition to PAD, the ABI also is an indicator of generalized atherosclerosis because lower levels have been associated with higher rates of concomitant coronary and cerebrovascular disease and with the presence of cardiovascular risk factors.¹⁴

In population cohort studies in the United States¹⁵⁻¹⁸ and Europe 119-231, a low ABI has been related to an increased incidence of mortality (total and cardiovascular), myocardial infarction, and stroke. These increased relative risks have been shown to be independent of baseline cardiovascular disease and risk factors, suggesting that the ABI might have an independent role in predicting cardiovascular events.²⁵⁻³⁴

In recent times, the measurement of vessel abnormality by the use of handheld Doppler device provides us information regarding atherosclerosis in patients with essential hypertension.

Atherosclerosis causes increased thickening in the anterior tibial/dorsalis pedis and the brachial artery detected by the

Doppler examination which indicates increased the risk of coronary events; which is a direct marker of PAD.

It is a non-invasive preferred screening procedure. Investigation which is valuable in identifying an hypertensive individual at risk for coronary artery disease (CAD). As a hypertensive individual is more prone for an atherosclerotic event.

Several studies have shown that a decreased ABPI is associated with increased cardiovascular risk,²⁴ Newman *et al.*, 1999,¹⁶ the partners program 2001,²⁵ Antonopoulos *et al.*, 2005.²⁶ A first large scale study including 5646 Chinese patients suggested that ABI might be a marker of atherosclerosis.²⁷

Furthermore, non-invasive measurements of ABI can provide an accurate indication of CV abnormality and might be used to screen for atherosclerotic diseases in Chinese patients with risk factors.

Hence, ABPI has shown a strong predictor of cardiovascular morbidity and mortality in near future and very few studies have been conducted for ABPI in patients with essential hypertension.³⁵⁻³⁹

METHODS

We studied in all 93 subjects who had presented to a tertiary care center. The study design and methods were approved by the Ethics Committee, all participants provided informed consent.

- 31 patients of Stage I hypertension (As per Joint National Committee [JNC] VII criteria).
- 31 patients of Stage II hypertension.
- 31 patients of normotensive subject (these will be patients admitted to medical wards with causes other than hypertension).

Subjects attended a clinic and completed a questionnaire including validated questions on occupation, smoking, alcohol, history of diabetes, history of hypertension, and history of ischemic heart disease, or a cerebrovascular accident. A comprehensive clinical examination included brief physical examination including general examination. BP recording SBP and diastolic (Phase V) BPs (DBP) in right arm after 10 min of rest with no consumption of tobacco in any form before attending the clinic and after voiding the bladder; using a random zero sphygmomanometer. Apart from the systemic examination which included the cardiovascular, respiratory, gastrointestinal, and central nervous system; anthropometric examination is also conducted.

Ankle systolic pressure was measured in a posterior tibial artery of the right then the left leg and brachial artery of the right then the left hand using a hand held Doppler ultrasound probe.

A 12-lead electrocardiogram (ECG) was also performed.

A fasting blood sample for glucose and lipid profile and a postprandial blood glucose test along with a complete blood count was performed.

Patients after satisfying inclusion and exclusion criteria given below were divide into three groups as Stage I hypertensive, Stage II hypertensive (according to JNC VII guidelines), and a control or the normotensive group.

Inclusion Criteria

1. Essential hypertensive (according to JNC-VII) above 18 years of age.
2. Patients are willing to give a written valid informed consent.
3. Control group included normotensive subject of either age above 18 years of age.

For the control group, patients were selected such that they had no H/O claudication, angina or stroke.

- No H/O previous arterial/cardiac surgery.
- Had normal pedal pulses.
- No evidence of venous ulcers, gangrene or limb amputation.

Exclusion Criteria

Subjects excluded from the control group if they were:

1. Chronic smokers
2. Diabetics
3. Dyslipidemia
4. Patients with diagnosed peripheral vascular disease.

Methods

ABPI procedure model using Doppler method

Explain the procedure and reassure the patient and ensure that he/she is lying flat and is comfortable, rested and relaxed with no pressure on the proximal vessels.

1. Measure the brachial BP
 - Place an appropriately sized cuff around the upper arm.
 - Locate the brachial pulse and apply ultrasound contact gel.
 - Angle the Doppler probe at 45° and move the probe to obtain the best signal.
 - Inflate the cuff until the signal is abolished the deflate the cuff slowly and record the pressure at

which the signal returns being careful not to move the probe from the line of the artery.

- Repeat the procedure for the other arm.
- Use the higher of the two values to calculate the ABPI.

2. Measure the ankle systolic pressure
 - Place an appropriately sized cuff around the ankle immediately above the malleoli.
 - Examine the foot locating the posterior tibial pulse and apply contact gel.
 - Continue as for the brachial pressure, recording the pressure in the same way.
 - Repeat the procedure for the other leg.
 - Use the highest recording obtained to calculate ABPI for that leg.

Patients in the study group were divided into three groups as Stages I and II hypertensive and control group as given below:
 Stage I - SBP 140-159 or DBP 90-99.
 Stage II - SBP 160-179 or DBP 100-109.

Control group or the normotensive group.

RESULTS

In this study, the number of patients are 93 ($n = 93$) out of which male patients were 16 in number in each of the group, *viz.*, Stage I, Stage II, and the normal (controls), while the female subjects were 15 in number in each of the above said groups (Table 1).

Table 2 shows that of the number of patients (31) in control group; 5 were in the age group of 40-50 years and 26 were in the age group of 51-60 years.

In Stage I of the 31 subjects; 8 belonged to the age group of 40-50 years and 23 belonged to the age group of 51-60 years.

Table 1: Sex wise distribution of patients

Gender distribution	Count (%)	
	Male	Female
Normal	16 (51.6)	15 (48.4)
Stage I	16 (51.6)	15 (48.4)
Stage II	16 (51.6)	15 (48.4)

Table 2: Age-wise distribution of patients

Age distribution	Count (%)	
	40-50 years	51-60 years
Normal	5 (16)	26 (84)
Stage I	8 (26)	23 (74)
Stage II	2 (6)	29 (94)

In Stage II of the 31 subjects; 2 were in the age group of 40-50 years whereas 29 were in the age group of 51-60 years.

Of the selected sample size 10 individuals from control group (32%), 16 from Stage I hypertension (52%) and 20 from Stage II hypertension (65%) had a history or ECG evidence suggestive of IHD (Table 3).

Of the selected sample size 3 individuals from control group (6%), 5 from Stage I hypertension (13%) and 6 from Stage II hypertension (16%) had a history of stroke or a cerebrovascular accident (Table 4).

We found out that of the normal individuals 8 (26%), Stage I; 11 individuals (35%), Stage II; 12 individuals (39%) were on treatment for their IHD or cerebrovascular accident. Moreover, of the normal individuals 23 (74%), Stage I; 20 individuals (65%), Stage II; 19 individuals (61%) were not on treatment of any kind for their IHD or cerebrovascular accident (Table 5).

Patients with normal BP did not show any evidence of retinopathy.

Table 3: Correlation of IHD with stage of hypertension

IHD	Count (%)	
	No	Yes
Normal	21 (68)	10 (32)
Stage I	15 (48)	16 (52)
Stage II	11 (35)	20 (65)
Normal-Stage I	0.123 ^(NS) Chi-square test	
Normal-Stage II	0.11 ^(NS) Chi-square test	

IHD: Ischemic heart disease

Table 4: Correlation of CVA with hypertension

CVA	Count (%)	
	No	Yes
Normal	28 (90)	3 (6)
Stage I	26 (84)	5 (13)
Stage II	25 (81)	6 (16)
Normal-Stage I	0.389 ^(NS) Chi-square test	
Normal-Stage II	0.228 ^(NS) Chi-square test	

Table 5: Correlation of subjects taking treatment for IHD/CVA

Treatment	Count (%)	
	No	Yes
Normal	23 (74)	8 (26)
Stage I	20 (65)	11 (35)
Stage II	19 (61)	12 (39)
Normal-Stage I	0.409 ^(NS) Chi-square test	
Normal-Stage II	0.277 ^(NS) Chi-square test	

IHD: Ischemic heart disease

In patients with Stage I hypertension. 7 of them (23%) had Grade 1 hypertensive retinopathy and 2 or, 1:6% has Grade 2 hypertensive retinopathy.

In patients with Stage II hypertension 3 of them (10%) had Grade 1 hypertensive retinopathy, 4 of them (14%) had Grade 2 hypertensive retinopathy, 5 of them (16%) had Grade 3 hypertensive retinopathy and 2 or (6%) had Grade 4 hypertensive retinopathy (Table 6).

In the control group the mean height was 1.7 ± 0.1 m², in Stage I hypertension it was 1.6 ± 0.1 m² and in Stage II it was 1.5 ± 0.1 m².

In the control group the mean height was 72.5 ± 12.5 kg, in Stage I hypertension it was 80.5 ± 8.3 kg and in Stage II it was 77.7 ± 9.3 kg.

On comparison of the body mass index (BMI), it was found that in control group the mean was 26.2 ± 4.4 kg/m² which was between normal and overweight.

For Stage I hypertension the mean BMI was 32.4 ± 4.0 kg/m² which is between overweight and moderate obesity (Class 1).

And for Stage II hypertension the mean BMI was 34.4 ± 4.8 kg/m² which is between moderate (Class 2) and severe (Class 3) obesity (Table 7).

In the control group, the mean waist circumference was 76.3 ± 6.4 cm, it was 82.5 ± 10.7 cm in Stage I hypertension and was 94.3 ± 11.0 cm for individuals in Stage II hypertensive patients.

Table 6: Correlation of funduscopy findings with grades of hypertension

Fundus	Count (%)				
	Grade 1	Grade 2	Grade 3	Grade 4	N
Normal	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Stage I	7 (23)	2 (6)	0 (0)	0 (0)	22 (71)
Stage II	3 (10)	4 (14)	5 (16)	2 (6)	17 (55)

Table 7: Correlation of height weight and BMI

Parameter	Height	Weight	BMI
Normal			
Mean±SD	1.7 ± 0.1	72.5 ± 12.5	26 ± 4.4
Stage I			
Mean±SD	1.6 ± 0.1	80.5 ± 8.3	32 ± 4
Stage II			
Mean±SD	1.5 ± 0.1	77.7 ± 9.3	34.4 ± 4.8
Normal-Stage I	0.23 ^(NS)		
Normal-Stage II	0.56 ^(NS)		

BMI: Body mass index, SD: Standard deviation

According to the waist circumference and waist-hip ratio (W/H) WHO expert consultation Geneva December 2008 the normal waist circumference was <100 cm for men and <87.5 cm for women. An increase in the same increases the risk of vascular events.

Hip circumference for normal individuals was 98.0 ± 6.1 cm, for Stage I hypertension it was 91.6 ± 7.7 cm and for Stage II hypertension it was 89.3 ± 8.8 cm.

The ratio (W/H) in controls was 0.8 ± 0.1 W/H for Stage I is 0.9 ± 0.1 and for Stage II is 1.1 ± 0.2 , the normal range of W/H is <0.95 for men and <0.8 for women. An increase above this range indicates and increased for cardiovascular and cerebrovascular events (Table 8).

The SBP for controls was in the range of 105.8 ± 9.2 mmHg, for Stage I it was in the range of 151.3 ± 5.5 mmHg whereas for Stage II it was 179.3 ± 13.0 mmHg.

Whereas the DBP for controls was 65.9 ± 8.6 mmHg, for Stage I it was in the range of 94.5 ± 3.0 mmHg, and for Stage II it was 124.1 ± 11.9 mmHg (Table 9).

Table 10 shows that for controls the serum creatinine is 1.0 ± 0.4 mg%, for Stage I it is 1.4 ± 0.5 mg% and 1.6 ± 0.8 mg% for Stage II.

Table 8: Correlation of waist circumference and hip circumference

Parameter	Waist circumference	HIP circumference	W/H ratio
Normal			
Mean±SD	76.3±6.4	98.0±6.1	0.8±0.1
Stage I			
Mean±SD	82.5±10.7	91.6±7.7	0.9±0.1
Stage II			
Mean±SD	94.3±11.6	89.3±8.8	1.1±0.2
Normal-Stage I	0.007 (S)	0.007 (S)	0.001 (S)
Normal-Stage II	0.008 (S)	0.003 (S)	0.004 (S)

SD: Standard deviation

Table 9: Correlation of blood pressure in the stages of hypertension

Parameter	SBP	DBP
Normal		
Mean±SD	105.8±9.2	65.9±8.6
Stage I		
Mean±SD	151.3±5.5	94.5±3.0
Stage II		
Mean±SD	179.3±13.0	124.1±11.9
Normal-Stage I	0.01 (S)	0.023 (S)
Normal-Stage II	0.002 (S)	0.033 (S)

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, SD: Standard deviation

The correlation between serum cholesterol and hypertension in normal is $168.1 \pm 3J$ mg%, Stage I is 220.0 ± 21.7 mg% and Stage II is 246.1 ± 22.2 mg%.

Now, it is clear that there is an obvious direct correlation between the level of total cholesterol and grade of hypertension (Table 11).

The low-density lipoprotein (LDL) cholesterol for normal was 120 ± 23.2 mg% while for Stage I 192 ± 19.9 mg% and for Stage II was 220.0 ± 19.8 mg%.

Here we understand that there is a direct correlation between the level of LDL cholesterol and grade of hypertension.

The controls had high-density lipoprotein (HDL) cholesterol 48.0 ± 6.2 mg% for Stage I-HDL cholesterol 28.0 ± 6.7 mg% and for Stage II HDL cholesterol is 26.1 ± 8.4 mg%.

From the above observation, it is clear that there is an inverse correlation between HDL cholesterol and grade of hypertension.

The correlation between serum triglycerides (TG) and hypertension in normal group is 154.0 ± 22.2 mg%, Stage I is 192.1 ± 24.3 mg% and Stage II is 224 ± 21.3 mg%.

Table 10: Correlation of serum creatinine with stage of hypertension

Parameter	Serum creatinine	BUN
Normal		
Mean±SD	1.0±0.4	13.4±3.7
Stage I		
Mean±SD	1.4±0.5	11.5±3.3
Stage II		
Mean±SD	1.6±0.8	13.5±1.6
Normal-Stage I	0.0003 (S)	0.031 (S)
Normal-Stage II	0.0002 (S)	0.89 (S)

SD: Standard deviation

Table 11: Correlation of lipid profile with hypertension

Parameter	Total cholesterol	LDL	HDL	TG
Normal				
Mean±SD	168.1±23.3	120±23.2	48±6.2	154±22.2
Stage I				
Mean±SD	220±21.7	192±19.9	28±6.7	192.1±24.2
Stage II				
Mean±SD	246.1±22.2	220±19.8	26.1±8.4	224±21.3
Normal-Stage I	0.001(S)	0.0037 (S)	0.0033 (S)	0.0015 (S)
Normal-Stage II	0.005 (S)	0.0048 (S)	0.0001 (S)	0.0028 (S)

LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TG: Triglycerides, SD: Standard deviation

Now, it is clear that there is an obvious direct correlation between the level of TG and grade of hypertension (Table 11).

The normal right brachial pressure is 108.9 ± 7.3 mmHg, for individuals from Stage I it is 158.1 ± 7.1 mmHg and for those in Stage II it is 229.5 ± 16.7 mmHg.

The left brachial pressure in control was 105.6 ± 7.9 mmHg, for Stage I 156.1 ± 7.4 mmHg it was and for Stage II it was 229.9 ± 16.2 mmHg (Table 12).

The normal right ankle pressure in the study was 121.0 ± 6.9 mmHg, in Stage I it was 164.1 ± 7.5 mmHg and for Stage II the same as 169.6 ± 16.7 mmHg.

The left ankle pressure was 105.2 ± 7.8 mmHg, for Stage I it was 160.1 ± 5.6 mmHg, and for Stage II the same was 165.7 ± 16.7 mmHg (Table 13).

Table 14 shows that the right ABPI value in the normal population is 1.1 ± 0.1 , for Stage I it is 0.9 whereas for the Stage II population it is 0.7 ± 0.1 .

It is evident that the left ABPI value in the normal population is 1.0, for Stage I it is 0.8 whereas for the Stage II population it is 0.7 ± 0.1 .

DISCUSSION

Assessment of ABPI in a hypertensive patient is a non-invasive measure of generalized atherosclerosis as ABPI is significantly lower in patients having hypertension.

Table 12: Values of brachial pressure

Parameter	RT-brachial press	LT-brachial press
Normal		
Mean±SD	108.9±7.3	105.6±7.9
Stage I		
Mean±SD	185.1±7.1	156.1±7.4
Stage II		
Mean±SD	229.5±16.7	229.9±16.2
Normal-Stage I	0.0033 (S)	0.0043 (S)
Normal-Stage II	0.0027 (S)	0.0063 (S)

SD: Standard deviation

Table 13: Values of ankle pressure

Parameter	RTS-ankle press	LT-ankle press
Normal		
Mean±SD	121.0±6.9	105.2±7.8
Stage I		
Mean±SD	164.1±7.5	160.1±5.6
Stage II		
Mean±SD	169.6±16.7	165.7±16.7
Normal-Stage I	0.005 (S)	0.003 (S)
Normal-Stage II	0.006 (S)	0.004 (S)

SD: Standard deviation

The assessment shows a lower mean value of ABPI among hypertensive individuals as the grade of hypertension increases progressively. The values of ABPI are comparatively lower in hypertensive individuals having associated obesity, sedentary lifestyle, and dyslipidemia.

Agrawal *et al.* in their study including 121 hypertensive patients had replied a mean ABPI of 0.88 ± 0.10 for Stage I and 0.68 ± 0.16 for Stage II.

Shah *et al.* studied 163 patients with hypertension and found the mean ABPI for Stage I as 0.9 ± 0.10 and for Stage II as 0.72 ± 0.10 .

Pillai while working with the South Indian population with hypertension found that the mean ABPI for Stage I was 0.82 ± 0.15 and for Stage II was 0.75 ± 0.08 .

In our study, the value for mean ABPI in stage hypertension was 0.85 ± 0.0 and for Stage II it was 0.70 ± 0.10 .

Lee *et al.* (2001) investigated ABPI had a similar study on patients ($n = 84$) with essential hypertension found that the ABPI was much lower in patients with Stage II as compared with Stage I and the control group.

Abott (2004) also noticed a significantly lower ABPI in patients with Stage II hypertension as compared with Stage I (Table 15).

Correlation of ABPI with Coronary Risk Factors

In our study, we have correlated the mean ABPI with significant coronary risk factors such as age, DBP, obesity

Table 14: Table with right and left ABPI

Parameter	RT-ABPI	LT-ABPI
Normal		
Mean±SD	1.1±0.1	1.0±0.0
Stage I		
Mean±SD	0.9±0.0	0.8±0.0
Stage II		
Mean±SD	0.7±0.1	0.7±0.1
Normal-Stage I	0.0044 (S)	0.0031 (S)
Normal-Stage II	0.0056 (S)	0.0034 (S)

ABPI: Ankle brachial pressure index, SD: Standard deviation

Table 15: Mean ABPI in hypertension

Variable	Present study	Agrawal <i>et al.</i>	Shah <i>et al.</i>	Pillai <i>et al.</i>
Number of patients	93	121	163	81
Mean ABPI for Stage I	0.85 ± 0.0	0.88 ± 0.10	0.9 ± 0.10	0.82 ± 0.15
Mean ABPI for Stage II	0.70 ± 0.10	0.68 ± 0.16	0.72 ± 0.10	0.75 ± 0.08

ABPI: Ankle brachial pressure index

BMI, hypercholesterolemia, serum LDL level, HDL level, and TG level.

1. Association of mean ABPI and age
 - In our study, the age group varied from 40 to 60 years. The mean ABPI for Stage I is 0.85 ± 0.0 and for Stage II is 0.70 ± 0.10 .
 - The mean age for Stage I was 54.12 ± 7.17 and for Stage II was 56.72 ± 8.81 .
 - Mainly the ABPI decreases with age as atherosclerosis progresses with age. The other factors that influence is the duration of hypertension, associated CAD, other coronary risk factors, and modality of treatment (Tables 16 and 17).
 - In this study, the mean age was 54.12 years, and Agrawal *et al.* reported the mean age as 58.78 years, and Shah reported it as 52.12 years in subjects with Stage I hypertension.
 - In this study, the mean age was 56.72 years and Agrawal *et al.* reported the mean age as 66.22 years and Shah reported it as 58.87 years in subjects with Stage II hypertension (Tables 18 and 19).
2. Association of ABPI with obesity and lifestyle
 - In this study, we compared the BMI in Stages I and II it was found that the BMI for Stage I was

32.4 ± 4.0 as compared to the control group and this finding was statistically significant ($P < 0.01$) (Table 20).

- In this study, we compared the BMI in Stages I and II it was found that the BMI for Stage II was 34.4 ± 4.8 as compared to the control group and this finding was statistically significant ($P < 0.01$) (Table 21).
3. Association of mean ABPI with serum cholesterol
 - In this study, the mean serum cholesterol was 220.0 ± 21.7 mg% for Stage I and 246.1 ± 22.2 mg% for Stage II hypertension.
 - According to the ARIC study as well concludes that hypercholesterolemia is one of the deterring factors for thickening of the vessel wall and consequently for ABPI.
 4. Association of mean ABPI with serum LDL
 - In this study, the level of serum cholesterol $<P$ was 192.0 ± 19.9 mg% for Stage I and was 220.0 ± 19.8 mg% for Stage II.
 - According to the Edinburgh artery study 2004, it too concludes that increased serum LDL has a direct correlation with hardening of the vessel wall which consequently reduces the ABPI with increasing grade of hypertension.
 5. Association of mean ABPI with serum HDL
 - In this study, the level of serum HDL was compared in Stage I and Stage II and was found to be 28.0 ± 6.7 mg%, 26.1 ± 8.4 mg%, respectively, ref Table 11.

Table 16: Correlation of variables in Stage I with other studies

Variable	Present study	Agrawal <i>et al.</i>	Shah <i>et al.</i>
Age (years)	54.12±7.17	58.78±6.12	52.12±4.46
SBP (mmHg)	151.3±5.5	148.8±3.2	152.3±2.6
DBP (mmHg)	94.5±3.0	96.1±8.8	92.7±4.5
BMI (kg/m ²)	32.4±4.0	31.4±3.6	33.1±4.4
Total cholesterol (mg %)	220.0±21.7	226.9±22.2	215.8±32.7
LDL (mg %)	192.0±19.9	184.6±15.6	190.0±18.4
HDL (mg %)	28.0±6.7	27.0±6.6	32.0±5.7
TG (mg %)	192.1±24.2	192.1±34.7	186.1±28.8
ABPI	0.85±0.0	0.88±0.10	0.90±0.10

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, ABPI: Ankle brachial pressure index

Table 17: Correlation of variables in Stage II with other studies

Variable	Present study	Agrawal <i>et al.</i>	Shah <i>et al.</i>
Age (years)	56.72±8.81	66.22±4.84	58.87±7.65
SBP (mmHg)	179.3±13.0	176.3±11.5	180.1±15.3
DBP (mmHg)	124.1±11.9	122.1±14.5	114.1±13.9
BMI (kg/m ²)	34.4±4.8	33.6±4.4	34.8±2.8
Total cholesterol (mg %)	246.1±22.2	244.1±26.2	252.0±11.4
LDL (mgrYo)	220.0±19.8	180.0±11.3	207.3±18.1
HDL (mg %)	26.1±8.4	24.6±6.8	26.9±6.5
TG (mg %)	224.0±21.3	20r. 8±24.2	218.9±18.7
ABPI	0.10±0.10	0.68±0.16	0.72±0.10

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, ABPI: Ankle brachial pressure index

Table 18: Stage I

Variable	Present study	Agrawal <i>et al.</i>	Shah <i>et al.</i>
Number of patients	93	121	163
Age (years)	54.12	58.78	52.12

Table 19: Stage II

Variable	Present study	Agrawal <i>et al.</i>	Shah <i>et al.</i>
Number of patients	93	121	163
Age (years)	56.72	66.22	58.87

Table 20: Stage I

Variable	Present study	Agrawal <i>et al.</i>	Shah <i>et al.</i>
BMI (kg/m ²)	32.4±4.0	31.4±3.6	33.1±4.4
P value	<0.01	<0.05	<0.01

BMI: Body mass index

Table 21: Stage II

Variable	Present study	Agrawal <i>et al.</i>	Shah <i>et al.</i>
BMI (kg/mL)	34.4±4.8	33.6±4.4	34.8±2.8
P value	<0.01	<0.05	<0.01

BMI: Body mass index

- Table 11 shows that $P < 0.01$ for our study was statistically significant, so also for Agrawal *et al.* $P < 0.01$. This implies that a higher HDL cholesterol has a protective effect in the advancement of atherosclerosis.
 - While in Shah $P > 0.02$ which was not statistically significant.
6. Association of mean ABPI with serum TG level
- In the study performed for comparison of serum TG levels in the study population, it was observed that the mean serum TG level in Stage I group was 192.1 ± 24.2 mg% while in Stage II was 224.0 ± 21.3 mg%.
 - Table 11 shows that there is a direct correlation of serum TG level in this study with mean ABPI as a major contributing factor; which is also evident from its statistical significance ($P < 0.01$) ref Table 11.
 - The same is supported by the Pillai study ($P < 0.01$).
 - On the contrary in the Agrawal *et al.* study ($P > 0.01$) makes it statistically insignificant.
 - The Honolulu Heart Program also states that a higher serum TG level in uncontrolled essential hypertension is a direct confounding factor for a 11-cause cardiovascular and cerebrovascular morbidity and mortality.

Summary

- This study comprised 93 patients out of which 62 were recently or previously diagnosed with essential hypertension, and 31 were non-hypertensive.
- ABPI was performed in all the 93 patients.
- Out of 93 subjects, 48 were males (51.6%), and 45 (48.4%) were females.
- Among the 93 patients, the common age group was 51-60 years. The number of patients in 40-50 years age group was 15 and in 51-60 years group were 78. The mean age for Stage I was 54.12 years and for Stage II was 56.72 years.
- Out of the 93 subjects, 16 from Stage I had a CAD and 20 from Stage II had a CAD. At the same time 15 from Stage I did not have a coronary vascular disease and 11 from Stage II also did not have a CAD.
- It was surprisingly found that only 11 individuals from Stage I were on treatment either for their hypertension or IHD (i.e., 35%) and 12 subjects from Stage II were on treatment for their condition (i.e., 39%).
- In this study, the mean ABPI of 31 patients in Stage I was 0.85 ± 0.0 and in the 31 patients in Stage II was 0.70 ± 0.10 .
- This study shows that an inverse correlation with stage of hypertension, i.e., with an increase in BP there is a decrease in ABPI and increased risk of atherosclerosis.
- So also the present study makes, it clear that patients with increasing age have an increased degree of atherosclerosis as deciphered from the fall in ABPI values.
- The mean SBP in Stage I was 151.3 ± 5.5 mmHg and Stage II was 179.3 ± 13.0 mmHg in this study which was comparable with the other studies.
- Similarly, in this study, the DBP for Stage I was 94.5 ± 3.0 mmHg and for Stage II was 124.1 ± 11.9 mmHg, which is statistically significant.
- The correlation of ABPI with other coronary risk factors has been performed. This study and its comparison with other studies give us a conclusion that if the patient is having a coronary risk factor or more number of risk factors are having a lower value of mean ABPI.
- The obesity creating a major risk factor for atherosclerosis was compared in both Stages I and II subjects. It was observed that patients in Stage II (34.4 ± 4.8) were more obese as compared to those in Stage I (32.4 ± 4.0). The mean ABPI suggested a higher BMI in Stage II than Stage I which is statistically significant.
- The mean ABPI as compared with total cholesterol in the two groups, *viz.*, Stage I and Stage II was found to be 220.0 ± 21.7 mg% and 246.1 ± 22.2 mg %, respectively. The higher level of cholesterol in Stage II as compared to Stage I with a low ABPI value in the prior group infers that the correlation is statistically significant ($P < 0.01$).
- In our study, serum LDL was compared with ABPI in Stage I was 192.0 ± 19.9 mg% and in Stage II was 220.0 ± 19.8 mg%. This correlation was statistically significant ($P < 0.01$).
- Serum HDL compares to mean ABPI in Stages I and II was 28.0 ± 6.7 mg% and 26.1 ± 8.4 mg, respectively. This implies a protective effect of HDL in atherosclerosis in Stage I patients as compared to Stage I patients as compared to Stage II who had a lower value of ABPI.
- On comparing the mean ABPI to draw a correlation with hypertriglyceridemia, it was observed that the level of mean TG levels was 192.1 ± 24.2 mg% in Stage I and 224.0 ± 21.3 mg% in Stage II. The increase mean TG level in Stage II with a low mean ABPI signifies a statistically significant correlation.
- The ABPI by ultrasound Doppler is a simple, non-invasive, non-expensive safe technique for measurement of atherosclerosis.
- The ABPI is a valuable screening investigation for identifying a hypertensive patient at risk for CAD. Also

for assessment of the risk of CAD and progression of disease.

- ABPI is simpler and safe alternative, but the confirmative is coronary angiography. It cannot replace coronary angiography in the assessment of risk or prognosis of CAD, but it can help clinician to decide that the hypertensive patient is at higher risk for CAD and may help from coronary angiography.
- In present we are able to show the statistical correlation among patients with hypertension and risk factors including hypercholesterolemia, hypertriglyceridemia, and DBP as well as with age of the subjects.

REFERENCES

1. Lawes CM, Vander Hoorn S, Rodgers A; International Society of Hypertension. Global burden of blood-pressure-related disease, 2001. *Lancet* 2008;371:1513-8.
2. Vasan RS, Beiser A, Seshadri S, Larson MG, Kannel WB, D'Agostino RB, *et al.* Residual lifetime risk for developing hypertension in middle-aged women and men: The Framingham Heart Study. *JAMA* 2002;287:1003-10.
3. Keys A. Seven Countries: A Multivariate Analysis of Death and Coronary Heart Disease. Cambridge, MA: Harvard University Press; 1980.
4. Stamler J, Stamler R, Neaton JD. Blood pressure, systolic and diastolic, and cardiovascular risks. US population data. *Arch Intern Med* 1993;153:598-615.
5. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, *et al.* Blood pressure, stroke, and coronary heart disease. Part 1, Prolonged differences in blood pressure: Prospective observational studies corrected for the regression dilution bias. *Lancet* 1990;335:765-74.
6. Kannel WB, Dawber TR, Kagan A, Revotskie N, Stokes J 3rd. Factors of risk in the development of coronary heart disease - Six year follow-up experience. The Framingham Study. *Ann Intern Med* 1961;55:33-50.
7. Kannel WB, Gordon T, Schwartz MJ. Systolic versus diastolic blood pressure and risk of coronary heart disease. The Framingham study. *Am J Cardiol* 1971;27:335-46.
8. Anderson KM, Odell PM, Wilson PW, Kannel WB. Cardiovascular disease risk profiles. *Am Heart J* 1991;121:293-8.
9. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation* 1998;97:1837-47.
10. Brindle P, Beswick A, Fahey T, Ebrahim S. Accuracy and impact of risk assessment in the primary prevention of cardiovascular disease: A systematic review. *Heart* 2006;92:1752-9.
11. Wannamethee SG, Shaper AG, Lennon L, Morris RW. Metabolic syndrome vs Framingham Risk Score for prediction of coronary heart disease, stroke, and Type 2 diabetes mellitus. *Arch Intern Med* 2005;165:2644-50.
12. Cushman M, Arnold AM, Psaty BM, Manolio TA, Kuller LH, Burke GL, *et al.* C-reactive protein and the 10-year incidence of coronary heart disease in older men and women: The cardiovascular health study. *Circulation* 2005;112:25-31.
13. Tsimikas S, Willerson JT, Ridker PM. C-reactive protein and other emerging blood biomarkers to optimize risk stratification of vulnerable patients. *J Am Coll Cardiol* 2006;47 8 Suppl: C19-31.
14. Newman AB, Siscovick DS, Manolio TA, Polak J, Fried LP, Borhani NO, *et al.* Ankle-arm index as a marker of atherosclerosis in the Cardiovascular Health Study. Cardiovascular Health Study (CHS) Collaborative Research Group. *Circulation* 1993;88:837-45.
15. Weatherley BD, Nelson JJ, Heiss G, Chambless LE, Sharrett AR, Nieto FJ, *et al.* The association of the ankle-brachial index with incident coronary heart disease: The Atherosclerosis Risk in Communities (ARIC) study, 1987-2001. *BMC Cardiovasc Disord* 2007;7:3.
16. Newman AB, Shemanski L, Manolio TA, Cushman M, Mittelmark M, Polak JF, *et al.* Ankle-arm index as a predictor of cardiovascular disease and mortality in the Cardiovascular Health Study. The Cardiovascular Health Study Group. *Arterioscler Thromb Vasc Biol* 1999;19:538-45.
17. Abbott RD, Petrovitch H, Rodriguez BL, Yano K, Schatz IJ, Popper JS, *et al.* Ankle/brachial blood pressure in men >70 years of age and the risk of coronary heart disease. *Am J Cardiol* 2000;86:280-4.
18. Resnick HE, Lindsay RS, McDermott MM, Devereux RB, Jones KL, Fabsitz RR, *et al.* Relationship of high and low ankle brachial index to all-cause and cardiovascular disease mortality: The Strong Heart Study. *Circulation* 2004;109:733-9.
19. Leng GC, Fowkes FG, Lee AJ, Dunbar J, Housley E, Ruckley CV. Use of ankle brachial pressure index to predict cardiovascular events and death: A cohort study. *BMJ* 1996;313:1440-4.
20. Hooi JD, Kester AD, Stoffers HE, Rinkens PE, Knottnerus JA, van Ree JW. Asymptomatic peripheral arterial occlusive disease predicted cardiovascular morbidity and mortality in a 7-year follow-up study. *J Clin Epidemiol* 2004;57:294-300.
21. Ogren M, Hedblad B, Isacson SO, Janzon L, Jungquist G, Lindell SE. Non-invasively detected carotid stenosis and ischaemic heart disease in men with leg arteriosclerosis. *Lancet* 1993;342:1138-41.
22. van der Meer IM, Bots ML, Hofman A, del Sol AI, van der Kuip DA, Witteman JC. Predictive value of noninvasive measures of atherosclerosis for incident myocardial infarction: The Rotterdam Study. *Circulation* 2004;109:1089-94.
23. Kornitzer M, Dramaix M, Sobolski J, Degre S, De Backer G. Ankle/arm pressure index in asymptomatic middle-aged males: An independent predictor of ten-year coronary heart disease mortality. *Angiology* 1995;46:211-9.
24. Vogt MT, Cauley JA, Newman AB, Kuller LH, Hulley SB. Decreased ankle/arm blood pressure index and mortality in elderly women. *JAMA* 1993;270:465-9.
25. Hirsch AT, Criqui MH, Treat-Jacobson D, Regensteiner JG, Creager MA, Olin JW, *et al.* Peripheral arterial disease detection, awareness, and treatment in primary care. *JAMA* 2001;286:1317-24.
26. Antonopoulos S, Kokkoris S, Stasini F, Mylonopoulou M, Lependiotis G, Mikros S, *et al.* High prevalence of subclinical peripheral artery disease in Greek hospitalized patients. *Eur J Intern Med* 2005;16:187-91.
27. Jue LI, Tomohiro AK, Jiming YU, Jingang YA, Xiankai LI, Dayi HU, *et al.* Ankle brachial index as a marker of atherosclerosis in Chinese patients with high cardiovascular risk. *Hypertens Res* 2006;29:23-8.
28. Sharma S, Kmies C, *et al.* A liicle on hypertension: E medicine, June; 2007.
29. Hales S. Statistical Essays Containing Haemostatics; or an Account of Some Hydraulic and Hydrostatical Experiments Made on the Blood and Blood Pressure of Animals. London: Innys and Manby; 1933.
30. Yusuf S, Reddy S, Ounpuu S, Anand S. Global burden of cardiovascular diseases: Part I: General considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation* 2001;104:2746-53.
31. Yusuf S, Reddy S, Ounpuu S. Global burden of cardiovascular diseases: Part 2: Variations in cardiovascular diseases by specific ethnic groups and geographic regions and preventive strategies. *Circulation* 2001;104:2855-64.
32. Indian Hypertension Guidelines-ii; 2007.
33. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: Analysis of worldwide data. *Lancet* 2005;365:217-23.
34. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, *et al.* The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC 7 report. *JAMA* 2003;289:2560-72.
35. Oparil S, Wright JT Jr, Roccella EJ. National Heart Lung and Blood Institute Joint National Committee, Detection, Evaluation, and Treatment of High Blood Pressure; National High Blood Pressure Education Program Coordinating Committee; 2003.
36. Bonow RO, Mann D, Zipes D, Libby P. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 9th ed. Philadelphia, PA: Saunders; 2011.

Shetty, *et al.*: Ankle Brachial Pressure Index Correlation with Diastolic Blood Pressure, Dyslipidemia and Anthropometric Measurement

37. McEniery CM, Yasmin, Wallace S, Maki-Petaja K, McDonnell B, Sharman JE, *et al.* Increased stroke volume and aortic stiffness contribute to isolated systolic hypertension in young adults. *Hypertension* 2005;46:221-6.
38. Franklin SS, Pio JR, Wong ND, Larson MG, Leip EP, Vasan RS, *et al.* Predictors of new-onset diastolic and systolic hypertension: The Framingham Heart Study. *Circulation* 2005;111:1121-7.
39. Franklin SS. Hypertension in older people: Part 1. *J Clin Hypertens (Greenwich)* 2006;8:444-9.

How to cite this article: Shetty V, Jain HR, Singh GS, Parekh S, Shetty S. Ankle Brachial Pressure Index Correlation with Diastolic Blood Pressure, Dyslipidemia and Anthropometric Measurement in Patients of Essential Hypertension. *Int J Sci Stud* 2017;5(2):168-177.

Source of Support: Nil, **Conflict of Interest:** None declared.