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Comparison of Ankle Brachial Index among Diabetic and Non-Diabetic Age Matched Individuals

Rutuja Wayal[1], Jaimala Shetye[2], Kavita Joshi[3], Vibhuti Tiwari[4]

[1]Postgraduate student, Department of Physiotherapy, Seth GSMC & KEMH Mumbai, India.
[2] Associate Professor, Department of Physiotherapy, Seth GSMC & KEMH Mumbai, India.
[3]Professor, Department of Medicine, Seth GSMC & KEMH Mumbai, India.
[4] Postgraduate student, Department of Physiotherapy, Seth GSMC & KEMH Mumbai, India.

Corresponding Author: Rutuja Wayal

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ABSTRACT

Background: Peripheral arterial disease (PAD) is characterized by reduced blood flow through the arteries in the extremities. It is a strong predictor of morbidity and mortality in people with type 2 diabetes. Early detection of PAD based on Ankle brachial index allows for intervention and management of foot problems in an attempt to delay amputation. Most diabetic cases show impaired sympathetic skin response and hence impaired skin temperature which can be used as an outcome measure for impaired blood flow in the feet of diabetic patients.
Aim: To compare Ankle Brachial Index among type 2 diabetic and age-matched non-diabetic individuals.
To compare Surface skin temperature among the two groups
Methodology: This was a comparative study between 23 diabetic individuals between 40-60 years of age with Type-2 DM duration of more than 5 years and on medication (Group A) and 23 age-matched non-diabetics (Group B). Ankle brachial index (ABI) and Surface skin temperature were compared between the two groups using hand-held vascular Doppler and infrared thermometer respectively. The data of ABI and surface skin temperature passed the normality test and, hence were compared using an unpaired t-test.
Result: There was no statistically significant difference between the mean ABI values of the two groups (p=0.841). There was a statistically significant difference between mean surface skin temperature for the two groups (p=0.012). However, the difference was too less to be considered clinically significant.
Conclusion: There is no significant difference in ABI and Skin temperature among subjects with and without diabetes.

Keywords: Type 2 DM, Peripheral arterial diseases, Ankle brachial index, Skin temperature.

INTRODUCTION

Diabetes is a complex metabolic condition affecting over 170 million people in the world. Type 2 diabetes (non-insulin-dependent or adult-onset) results from the body's ineffective use of insulin. More than 95% of people with diabetes have type 2 diabetes. This type of diabetes is largely the result of redundant body weight and physical inactivity.[1]

Peripheral arterial disease (PAD) is a circulatory problem causing a reduced blood inflow through the arteries. This generally reduces blood inflow to the extremities manifesting pain during walking or exertion. The central pathophysiological theme of

PAD in DM is the process of atherosclerosis.[2] It begins with atherogenesis, and progresses to the eventual inhibition and reduction of blood inflow. Fig 1 represent several pathogenetic mechanisms that have been linked to progressive atherosclerosis, including endothelial dysfunction, inflammation, platelet aggregation, and vascular smooth muscle cell (VSMC) dysfunction.[3]

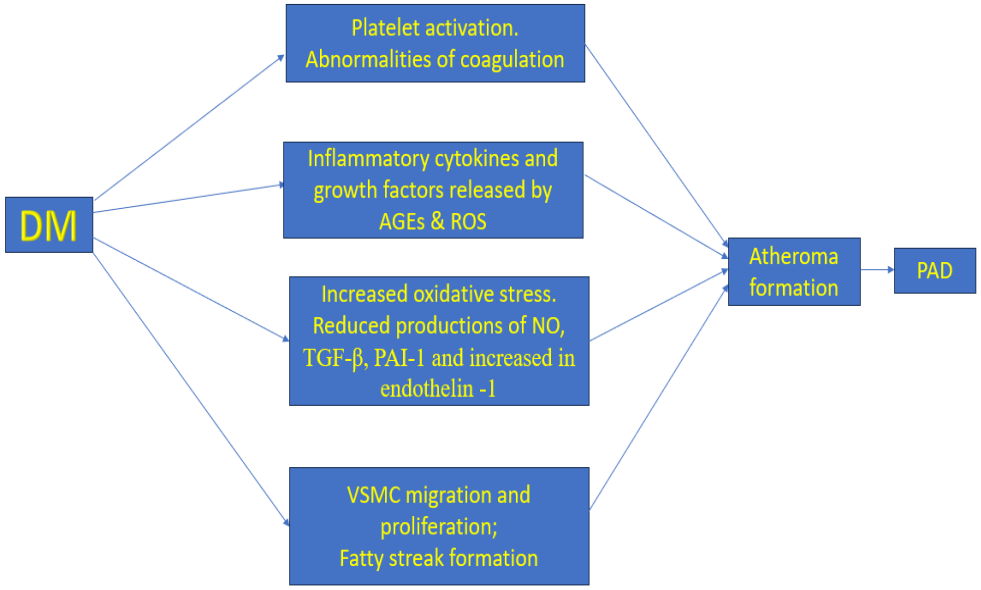

Fig 1. Schematic representation of the pathophysiology of peripheral arterial disease in diabetes mellitus. AGEs: Advanced glycation end products; VSMC: Vascular smooth muscle cell. DM: Diabetes mellitus; NO: Nitric oxide; PAD: Peripheral arterial disease; TGF-β: Transforming growth factor-beta PAI-1: Plasminogen activator inhibitor-1; ROS: Reactive oxygen species.[3]

Peripheral arterial disease (PAD) is estimated to affect up to 12% of the general population, and up to 14% of people with diabetes. Characterized by progressive stenosis of the lower branches, the presence of PAD is explosively prophetic of morbidity and mortality in people with diabetes.[4] Due to the high threat of concurrent cardiovascular complaints, distal artery complications associated with PAD, and the rapid progression of atherosclerosis in people with diabetes, accurate and dependable individual testing is necessary for early opinion and effective ongoing monitoring. Early diagnosis of PAD allows for planning of preventive and rehabilitative management of coexisting cardiovascular disease, including lifestyle advice and medication, and prevention and management of lower limb complications such as foot ulceration and delay or prevent amputation. [4,5] PAD is one of the numerous factors that contribute to the progressive and critical course of distal ischemia in the Type 2 Diabetes Mellitus (DM) cases.[6]

In diabetes, PAD becomes more common with advancing age, diabetic neuropathy, and a longer duration of diabetes mellitus. Diabetes generally affects distal arteries (e.g. Femoral, Popliteal, and Tibial artery) rather than proximal iliac and aorto-iliac vessels.[7] Diabetes, particularly non-insulin dependent diabetes mellitus (NIDDM), becomes more common with advancing age. Diabetes predisposes to accelerated atherosclerosis, so diabetic cases have more severe atherosclerosis than age- matched people without diabetes.[8] Numerous diabetic cases remain asymptomatic until advanced stages of distal ischemia when ulcers and gangrene evolve. This is primarily attributed to the supplemental neuropathy associated with pain that masks the claudication pain of distal artery ischemia.[7] Calcification of the arterial

tunica media or Monckeberg arteriosclerosis is more among diabetics and can cause compression of the distal arteries, leading to a falsely elevated ABI result. Since calcification is more common at the distal arteries of the foot, measuring toe pressure will allow better identification of the lesions. Blood pressure in the digital arteries requires the use of applicable device, which isn't always available in health services, whereas the sphygmomanometer needed to calculate ABI is available in nearly all vascular services.[9]. The ankle brachial index (ABI) is typically calculated as the ratio of the highest of the Dorsalis pedis and posterior tibial artery systolic pressure to the highest of the left and right brachial systolic pressures. [4,6,10] A normal value for ABI assessment within the general population is considered to be 1.00 to 1.40, with values 0.91 to 0.99 codified as 'borderline', while those below 0.91 represent likely PAD.

The ABI threshold most generally used is ≤ 0.90 based on studies reporting > 90% sensitivity and specificity to diagnose PAD. The ABI has also been called the ankle-arm indicator, the ankle-brachial blood pressure indicator, the ankle-arm rate, or the Winsor Index.[10] Current guidelines recommend that ABI should be used routinely to detect PAD in aged people, those with a smoking history, and in people with diabetes [2].

A low ABI (<0.90) has good sensitivity and excellent specificity for detecting PAD compared to the angiographic gold standard. Compared to individual with a normal ABI (0.90 –1.30), person with PAD have poorer physical functioning, more rapid mobility loss, fast functional decline, and have an increased threat of incident cardiovascular events.[12] The natural history of PAD includes a drop in the ABI over time by a mean of 0.06 over 4.6 years.[10]

An increased risk of amputation has been reported when the ABI is <0.50 in non-revascularized patients with leg ulcers. An ABI ≤0.90 is strongly associated with a 7-year risk of amputation in people with diabetes mellitus. However, it is important to point out that characteristics contributing to functional impairment and decline in people with PAD are multifactoral and include muscle size and composition, inflammation, lower-extremity strength, mitochondrial function, and behavioral factors most of which are manageable or reversible. Thus, ABI is just one of many characteristics associated with functional impairment and decline in patients with PAD.[10] If detected early these reversible components of functional decline can be treated.

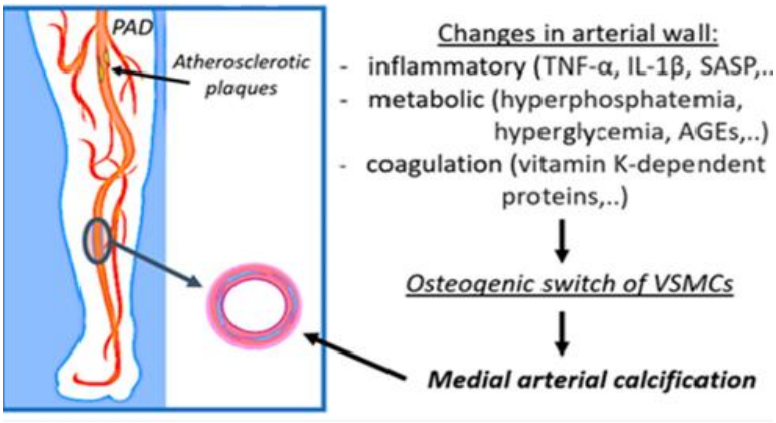

Fig. 2: Schematic presentation of the involvement of inflammatory, metabolic, and coagulation processes in the development of medial arterial calcifications in peripheral arterial disease.
(PAD: peripheral arterial disease, VSMCs: vascular smooth muscle cells; AGEs: advanced glycation end products TNF-α: tumor necrosis factor-alpha, IL-1β: interleukin-1beta, SASP: senescence-associated secretory phenotype).[11]

PAD is also a common form of atherosclerosis, in which adipose tissue deposits atheromatous plaque in the inner walls of the arteries. These blockages prevent

blood flow in the arteries, leading to blockages in the renal system, cardiovascular system and limbs (Fig 2).[11] Since ABI is said to be a marker of the atherosclerotic burden, the low ABI in Type 2 DM frequently indicates significant atherosclerosis in the vascular beds. ABI is 95% sensitive, and also 100% specific in detecting impaired blood flow when validated against angiography.[13]

The clinical signs of inflammation and soft tissue injury are frequently too subtle to be detected by the individual or even by a trained health care professional. The five cardinal signs of inflammation include pain, erythema, edema, loss of function, and hyperthermia. It is difficult to assess these subtle parameters early, with the exception of skin temperature, which can be fluently quantified. The concept of measuring skin temperature as a marker for inflammation and injury in the foot was first addressed by Goller et. al. in 1971, followed by Sandrow et al. in 1972. Goller reported a relationship between an increase in localised temperature and localised pressure whilst Sandrow used thermometry as a tool to diagnose neuropathic fractures.[14]

The autonomic neuropathy in distal arteries of diabetic cases leads to vasomotor disturbance, reduced sweating, and abnormal skin conditions including anhydrosis and dry skin leading to cracks and blisters. Peripheral autonomic dysfunction is associated with the development of foot lesions in diabetic subjects. It was reported that most diabetic cases with generalized autonomic symptoms revealed absent sympathetic skin response.[15]

Foot complications are one of the most frequent problems of diabetes mellitus and crucial contributors to medical costs, as 50% of all inpatient admissions due to diabetes are due to foot complications. The two main causes of diabetic complications are reduced blood flow and loss of sensation in the foot.[15] Skin temperature has long been used as a natural index of vascular conditions in the extremities. The impairment of blood inflow in stenosed (subcutaneous) arteries would impact cutaneous temperature. As plaque builds up in the arteries carrying blood from the heart to the limbs, it leads to the narrowing of the lumen, therefore confining blood inflow to the extremities, and reducing the capability of the body to regulate temperature.[16] Foot skin temperature is a routine method for assessing blood flow in the feet of diabetic cases and can be used as a clinical outcome to treat or improve blood inflow in type 2 DM cases.[17] Previous studies of ABI are inconclusive in terms of the result with reasoning not properly explained. The relationship between the duration of diabetes and the risk of progressive atherosclerosis has been relatively undetermined. Thus, the present topic was to compare the ABI, a marker of the atherosclerotic burden among Type 2 DM cases and normal subjects. Little literature on surface skin temperature is available, a clinical sign of inflammation and ulceration threat in type 2 DM. It is a simple, affordable, and fast method of measuring the foot-skin blood flow.

Operational Definition - Type 2 DM: Individual with physician diagnosed type 2 diabetes in accordance with Indian Council of Medical Research (ICMR), on oral hypoglycemic drugs (OHA) or Insulin.

MATERIALS & METHODS

This Comparative study was approved by Institutional Ethics Committee. Participants were selected on the basis of inclusion and exclusion criteria. Sample size of 46 was calculated. Data was statistically analyzed on 23 Diabetic individuals and 23 Non diabetic age matched individuals. The study was conducted in a tertiary care hospital. The type 2 DM and control group subjects were referred from OPD of internal medicine after screening by a co-investigator who is a professor of medicine. Participants were explained the purpose of the study & written informed consent was taken from the patients. Demographic data was recorded. Every participant was asked to avoid alcohol, exercise, and caffeine for 1 hour prior to

participating. The participant was maintained in a supine horizontal position for 10 minutes prior to and during testing and encouraged to rest and not engage in conversation or other activity during the testing process. The cuff position was ensured for it to be at the heart level by placing the limb on a pillow when needed. BP was measured at the brachial, posterior Tibial, and dorsalis pedis arteries in both left and right limbs. Systolic blood pressure at all sites was to be measured once, with a minimum of 3 minutes between pressure measurements for calculating ABI. (Fig 3).

Fig 3. ABI for right leg.

For measuring brachial pressures the ultrasound probe was placed in the antecubital fossa over the patient's brachial pulse and for measuring ankle pressures the cuff was placed immediately proximal to the malleoli. The ultrasound probe was placed on the skin overlying the dorsalis pedis (DP) and posterior tibial (PT) arteries in the foot one after the other. The systolic pressure at the DP artery and PT artery, was measured. The measurement was done on the opposite leg in the same manner. Lower ABI limb for each participant was selected for inclusion in statistical analysis. The ABI was calculated as the ratio of the higher of the dorsalis pedis and posterior Tibial systolic BP to the higher of the left or right branchial systolic BP. The sensitivity and specificity of ABI using the Doppler method are reported to be 80% and 100%, respectively.[18]

$$\text{Right leg ABI} = \frac{Highest\ pressure\ among\ PT\ and\ DP\ of\ right\ foot}{Highest\ pressure\ among\ both\ arms}$$

Table 1. Interpretation of ABI

ABI Value	Interpretation	Recommendation
Greater than 1.4	Calcification/Vessel Hardening	Refer to vascular specialist
1.0-1.4	Normal	None
0.9-1.0	Acceptable	None
0.8-0.9	Some Arterial Disease	Treat risk factors
0.5-0.8	Moderate Arterial Disease	Refer to a vascular specialist
Less than 0.5	Severe Arterial Disease	Refer to a vascular specialist

[Standford Medicine 25]

Surface skin temperature was measured using an infrared thermometer at 6 different spots i.e. hallux, first, third, and fifth metatarsal heads, metatarso cuneiform joint, and cuboid.

Mean temperature was calculated using temperature at all 6 sites for individual foot. (Fig 4)

Good to excellent intra-rater and inter-rater relative reliability is documented for measuring skin temperature IRT (Infrared thermometer) (ICC, 0.87 to 0.99; ICC, 0.83 to 0.98), respectively.[19]

Fig 4: Surface skin temperature (Head of first metatarsal)

STATISTICAL ANALYSIS

Statistical analysis was done using SPSS software, version 25. Observations are presented in percentage and proportion. Data was assessed for normality using the Shapiro Wilk test. The data of ABI and surface skin temperature passed the normality test, and hence was compared using unpaired t test.

Table 2. Test for normality

	Statistic	Df	Sig.
ABI	.956	46	.077
SST	.975	46	.427
SBP	.963	46	.151

RESULT

Sample size of 23 in each group was Calculated using power calculation. Subjects were included based on inclusion criteria.

23 diabetes and 23 non-diabetic individuals including 13 males and 10 females were recruited. The mean age of participants was 52.04±5.73. Mean BMI for those with and without diabetes was 26.20±3.50 and 24.60±3.77 respectively. Participant characteristics are described in Table 3 and in the pie charts (Fig 5 to Fig 7). There was no significant difference between the diabetic and non-diabetic groups with respect to smoking history, systolic BP, Diastolic BP, and Resting HR. Hence both groups were comparable.

Table 3. Demographic characteristics.

	Diabetes(n=23)	Non diabetes(n=23)	Between group differences (p value)
Males (n)	13	13	
Females (n)	10	10	
Mean age (years)	52.04 ± 5.73	52.04 ± 5.73	
Mean body mass index (kg/m^2)	26.20 ± 3.49	24.60 ± 3.77	0.379
Smoking history (%)	17.4	8.7	0.50
Mean diabetes duration (years)	9.20 ± 4.04		
Addiction (%)	30.4	34.8	0.87

Systolic BP (mmHg)	125.56±11.45	118.78±13.72	0.076
Diastolic BP (mmHg)	82.60±7.94	81.43±5.03	0.552
Resting HR (bpm)	84.69±8.19	80.91±5.21	0.069

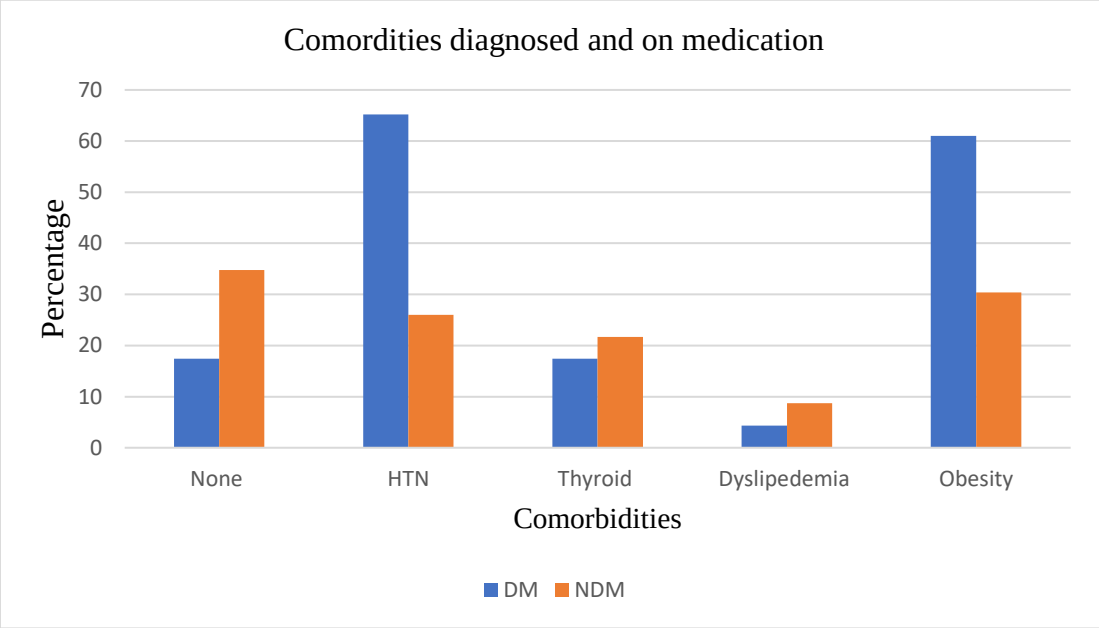

Fig 5: Percentage of subjects with comorbidities diagnosed and on medication.

Subjects in both groups had some or the other diagnosed comorbidity for which they were on medications. The proportion of individuals free of comorbidities was higher in non-diabetic group (34.8%) as compared to diabetic group (17.4%). (Fig 5)

Table 4. Mean ABI and Surface skin temperature in Diabetics and Non diabetics

	Diabetes(n=23)	**Non diabetes(n=23)**	**Between group difference (p value)**
Mean ABI	1.01±0.079	1.01±0.066	0.841
Mean Surface skin temperature (Degree celsius)	35.51±0.83	34.96±0.585	0.012

There was no statistically significant difference between mean ABI values for the two groups (p =0.841). There was a significant difference between mean surface skin temperature for the two groups (p =0.012). (Table 4)

DISCUSSION

In this study, the ABI, a marker of the atherosclerotic burden and surface skin temperature - a predictive of ulceration was studied in the cases with and without Type 2 DM. The two groups were analogous in terms of introductory characteristics like age, gender, and BMI. The study parameters like ABI and surface skin temperature showed sightly high values in the type 2 diabetics as compared to those in the normal.

The clinically nonsignificant difference in skin temperature can be possibly due to the duration of specific (Oral hypoglycemic) medicines which help to reduce vascular remodelling by decreasing vascular smooth muscle proliferation, migration, and inflammation. Also inhibits IL- 6 (Interleukin 6) and IL- 8 (Interleukin 8) release by decreasing NF- kB (Nuclear factor kappa B) nuclear translocation and activation of pro-inflammatory phosphokinases.[20]

The mean ABI in both groups was 1.01 which is within the normal limit, without any significant difference in both the groups (p= 0.841). These findings were in agreement with those of Vanessa S et al. who concluded in their longitudinal study, that there were no differences in mean ABI between diabetic cases and non-diabetic cases. The sensitivity of ABI seems to be limited in cases of

longstanding diabetes due to medial artery calcification (MAC) found in them.[10]

Medial artery calcification (MAC) is a condition characterized by the presence of diffuse calcium deposits along the medial layer of the arterial wall. It's a distinct, largely regulated process that's frequently linked in small and mid-sized arteries of the lower extremities. It is associated with advanced age, diabetes, and chronic renal complaints.[21] According to a study conducted in France by L. Potier the arterial stiffness secondary to MAC results in inadequately compressible vessels (increasing resistance to the cuff pressure) and an elevated ABI.[22] As mentioned in a study by Victor A., there can be several pathways to explain arterial stiffening in DM cases. Insulin triggers smooth muscle cell proliferation. Hyperglycemia is responsible for the nonenzymatic glycosylation of several proteins, including collagen and elastin.[12] Also, elevated ABI could underrate the frequency of PAD in diabetes because ABI values between 0.9 and 1.3 would be falsely considered normal, and advanced values couldn't be interpreted. It is thought that the association between high ABI values and PAD is due to the fact that arterial stiffness is associated with a decrease in blood flow in the lower limbs of diabetic patients. Likewise, elevated ABI values in diabetes could be reflective of PAD. Aboyans V and Suominen V also reported 58% to 84% of diabetics with PAD with elevated ABI values.[23] The individual effectiveness of ABI as a test may be limited in diabetic cases because of its weak sensitivity and the high rate of biased normal values, probably due to the high prevalence of MAC. One way to improve the diagnostic effectiveness of ABI might be to use an advanced threshold, around 1-1.1 or to use the smallest value of ankle systolic pressure for calculating it.[22] In addition, the profunda femoral artery is substantially affected in DM cases, but abnormalities cannot be detected by ABI because it does not contribute to ankle perfusion, except when it supplies a pathologic superficial femoral artery.[23]

In the present study, surface skin temperature was also assessed by using an infrared thermometer and it showed a significant difference among the two groups. ($p = 0.012$). In the current study, the diabetic individuals showed temperatures about 0.5-1 °C higher than the non-diabetic individuals. A difference of more than 2.2 °C is considered to be clinically significant. In the current study indeed though the result is statistically significant, but not clinically significant. A habitual increase in skin temperature in the foot of diabetic cases may be due to an increase in arterio-venous shunt inflow, while an acute increase in foot skin temperature is a prepping sign of pre-ulcer inflammation [19]. A study done by Subramnaiam B indicates that thermoregulatory disturbance and sweating abnormality is an early index of sympathetic damage in the diabetic foot. The capability to increase blood inflow depends on the actuality of normal neurogenic vascular response. Due to disabled neuro-vascular response in diabetic neuropathy subjects, a significant reduction of blood inflow under conditions of injury or infection is observed.[15] A methodical study by Arora S shows that the nerve-axon-related vasodilatory response to iontophoresis of acetylcholine was significantly reduced in diabetes cases when compared with healthy subjects or diabetes cases without complications.

There were limitations to the current study. Confounding factors, similar to the situations of diabetes foot problems and physical activity levels weren't taken into consideration. Brooks et al. showed that toe-pressure was more sensitive than ABI. Toe pressure sensitivity was 100% but was only 53% for ABI. We couldn't study the toe pressure due to the lack of availability of equipment.

CONCLUSION

We conclude that there is no significant difference in ABI and Skin temperature among subjects with and without diabetes.

CLINICAL IMPLICATION

Sensitivity appears to be lower in complicated T2DM, a higher sensitivity index such as toe brachial pressure should be performed. ABI values obtained should be interpreted with caution, in subjects with Diabetes Mellitus because of MAC.

Declaration by Authors

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