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Association of Abdominal Obesity and Metabolic Risk Factors with Subclinical Peripheral Arterial Disease among Young Adults: A Cross-Sectional Study

Sai Vineela Peruri[1], Asma[2], Shorya[3], Disha Tewtia[4], Bikash Kumar Choubey[5]

[1]MBBS, International higher school of medicine
[2]MBBS, Punjab Medical College Faisalabad, Affiliated with University of Health Sciences Lahore
[3]M.B.B.S., M.D., Adesh University, Bathinda, Punjab (M.B.B.S.)
[4]MBBS, Intern, Chhattisgarh Institute of Medical Sciences, Bilaspur
[5]MBBS, Taishan Medical University

Abstract

Background: Peripheral arterial disease (PAD) is traditionally regarded as a disease of older age, yet subclinical atherosclerotic change can begin decades before symptoms appear. Abdominal obesity and its accompanying metabolic derangements are becoming increasingly prevalent among young adults in urban India, but their relationship with subclinical PAD, as measured by the ankle-brachial index (ABI), remains poorly characterised in this age group.

Objectives: To determine the prevalence of subclinical PAD among apparently healthy young adults and to examine its association with abdominal obesity and individual metabolic risk factors.

Methods: This cross-sectional study was conducted among 640 apparently healthy adults aged 18–40 years recruited from an urban community and hospital outpatient population between January and December 2025. Anthropometric measures (waist circumference, waist-hip ratio, body mass index), blood pressure, fasting plasma glucose, and a fasting lipid profile were recorded. Abdominal obesity was defined using International Diabetes Federation criteria for Asian populations (waist circumference ≥90 cm in men, ≥80 cm in women). The ABI was measured bilaterally using a hand-held Doppler device; subclinical PAD was defined as an ABI ≤0.90 in either limb. Multivariable logistic regression was used to identify independent predictors of subclinical PAD.

Results: The mean age of participants was 29.4 ± 6.1 years, and 52.3% were men. Abdominal obesity was present in 34.2% of participants. The overall prevalence of subclinical PAD was 6.4% (41/640), with a further 11.9% showing borderline ABI values (0.91–0.99). Subclinical PAD was significantly more prevalent among participants with abdominal obesity than those without (11.4% vs. 3.8%, $p<0.001$). Mean ABI correlated negatively with waist circumference ($r = -0.29$, $p<0.001$), waist-hip ratio ($r = -0.24$, $p<0.001$), systolic blood pressure ($r = -0.21$, $p<0.001$), fasting triglycerides ($r = -0.19$, $p<0.001$), and

fasting plasma glucose (r = −0.17, p<0.001), and positively with HDL-cholesterol (r = 0.22, p<0.001). On multivariable logistic regression, abdominal obesity (adjusted odds ratio [aOR] 2.71, 95% CI 1.42–5.17), elevated systolic blood pressure (aOR 2.18, 95% CI 1.19–3.99), hypertriglyceridemia (aOR 1.96, 95% CI 1.05–3.66), and current smoking (aOR 2.34, 95% CI 1.18–4.64) were independently associated with subclinical PAD, whereas body mass index alone was not (aOR 1.31, 95% CI 0.71–2.41).

Conclusion: Subclinical PAD is present in a clinically meaningful proportion of young, apparently healthy adults and is more strongly associated with abdominal obesity and individual metabolic risk factors than with general adiposity. Ankle-brachial index screening in metabolically at-risk young adults may allow earlier identification of atherosclerotic risk and support timely preventive intervention.

Keywords: peripheral arterial disease; ankle-brachial index; abdominal obesity; metabolic syndrome; young adults; cross-sectional study; cardiovascular risk factors

1. Introduction

Peripheral arterial disease (PAD) is a manifestation of systemic atherosclerosis characterised by narrowing or occlusion of the arteries supplying the lower limbs, and it shares a common pathophysiological basis with coronary and cerebrovascular disease.[1] Globally, more than 230 million adults are estimated to be living with PAD, with rising numbers reported from low- and middle-income countries over the past two decades.[2] Although PAD has conventionally been considered a disease of the sixth and seventh decades of life, population-based data indicate that the atherosclerotic process begins far earlier, and subclinical vascular change detectable by non-invasive testing can be present well before the onset of symptoms such as intermittent claudication.[1,3]

The ankle-brachial index (ABI), calculated as the ratio of systolic blood pressure at the ankle to that at the brachial artery, is a simple, inexpensive, and well-validated non-invasive tool for detecting PAD, including in asymptomatic individuals.[3,4] An ABI of 0.90 or below is widely accepted as diagnostic of PAD and correlates closely with angiographically confirmed arterial stenosis, while values between 0.91 and 0.99 are generally regarded as borderline or indicative of early subclinical disease.[4,5] Because ABI abnormalities frequently precede overt symptoms, ABI-based screening has been proposed as a means of identifying individuals at elevated cardiovascular risk who might otherwise remain undetected until a coronary or cerebrovascular event occurs.[5,6]

Obesity, and abdominal obesity in particular, has emerged as a key modifiable determinant of cardiometabolic risk. Unlike general adiposity as captured by body mass index (BMI), abdominal or visceral obesity — reflected by waist circumference or waist-hip ratio — is more closely linked to insulin resistance, dyslipidaemia, and endothelial dysfunction, all of which contribute mechanistically to atherogenesis.[7,8] South Asian populations, including Indians, are recognised to develop these metabolic derangements at lower BMI thresholds than Western populations, a phenomenon sometimes termed the "Asian Indian phenotype," which is characterised by higher body fat percentage, greater central adiposity, and higher rates of insulin resistance for a given BMI.[9] This has led professional bodies to recommend lower, ethnicity-specific cut-offs for abdominal obesity in Asian Indian adults.[9]

The relationship between obesity, metabolic risk factors, and PAD has been studied extensively in older and clinical populations, but the findings have not been entirely consistent. Several hospital-based

studies among patients with established PAD have reported a high prevalence of metabolic syndrome, particularly among women, and an association between more severe circulatory insufficiency and metabolic syndrome components.[10] Data from the National Health and Nutrition Examination Survey have similarly shown that metabolic syndrome is independently associated with an abnormally low ABI even after adjustment for conventional cardiovascular risk factors.[11] In contrast, prospective cohort data from the D.E.S.I.R. study found that while individual metabolic factors — fasting glucose, insulin, HDL-cholesterol, and blood pressure — predicted incident PAD over nine years, neither BMI nor a categorical diagnosis of metabolic syndrome did so, suggesting that the mechanistic link may operate through specific metabolic pathways rather than adiposity or clustering per se.[12] A study among asymptomatic premenopausal women likewise reported an inverse association between central obesity measures and ABI, but concluded that this association was not fully mediated through the metabolic syndrome construct as conventionally defined.[13] These discrepancies suggest that the relative contributions of general adiposity, abdominal obesity, and individual metabolic risk factors to early vascular change remain incompletely resolved, and that this relationship may differ by age group, sex, and population.

Data specific to young adults are particularly sparse. Most epidemiological studies of PAD have enrolled participants aged 40 years or older, reflecting the assumption that clinically relevant arterial disease is uncommon before middle age.[2,14] However, with the rising prevalence of abdominal obesity, sedentary behaviour, and early-onset dysglycaemia and dyslipidaemia among young adults in urban India, there is a plausible case for atherosclerotic change to be detectable, at a subclinical level, considerably earlier than previously assumed.[9,15] Identifying such change while it remains reversible could have substantial implications for primary prevention. We therefore conducted this cross-sectional study to determine the prevalence of subclinical PAD among apparently healthy young adults aged 18–40 years, and to examine its association with abdominal obesity and individual metabolic risk factors, independent of general adiposity.

Materials and Methods

Study design and setting

This was an observational, analytical cross-sectional study conducted in the Department of Community Medicine in collaboration with the Departments of General Medicine and Cardiology. Data were collected over a 12-month period, from January 2025 to December 2025, from participants recruited both from the urban field practice area attached to the department and from apparently healthy individuals accompanying patients to the general outpatient department, to obtain a community-representative sample rather than a purely hospital-based one.

Study population

Adults aged 18–40 years of either sex who were permanent residents of the study area and provided written informed consent were eligible for inclusion. Individuals with a prior diagnosis of diabetes mellitus, hypertension, established cardiovascular or cerebrovascular disease, chronic kidney disease, known peripheral vascular disease, connective tissue disorders affecting peripheral vasculature, pregnancy, or any acute illness at the time of examination were excluded, as were individuals already

receiving lipid-lowering or antihypertensive therapy. These exclusions were applied so that any vascular or metabolic abnormality detected could be attributed to underlying, as yet undiagnosed, risk rather than to treated disease.

Sample size and sampling technique

The sample size was calculated using the expected prevalence of abnormal ABI among young adults with abdominal obesity, taken as 12% from comparable regional literature, with an absolute precision of 3% and a 95% confidence level, using the standard formula $n = Z^2p(1-p)/d^2$. This yielded a minimum requirement of 451 participants; after inflating for a design effect of 1.2 and an anticipated non-response of 15%, the final target sample size was set at 620, and 640 participants were ultimately enrolled. Participants were selected using a systematic random sampling technique from the household listing of the field practice area, supplemented by consecutive sampling of eligible outpatient department attendees until the target sample size was achieved.

Data collection and clinical measurements

Data were collected using a pre-tested, semi-structured questionnaire administered by trained investigators, covering sociodemographic characteristics, dietary and physical activity patterns, tobacco and alcohol use, and family history of cardiovascular disease. Physical activity was assessed using the Global Physical Activity Questionnaire (GPAQ).

Height was measured to the nearest 0.1 cm using a stadiometer, and weight to the nearest 0.1 kg using a calibrated digital scale, with participants in light clothing and without footwear. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in metres and classified according to the WHO Asia-Pacific criteria (underweight <18.5 kg/m², normal 18.5–22.9 kg/m², overweight 23.0–24.9 kg/m², obese ≥25.0 kg/m²). Waist circumference was measured at the midpoint between the lowest rib margin and the iliac crest at the end of normal expiration, and hip circumference at the level of greatest gluteal protrusion, both using a non-stretchable measuring tape; waist-hip ratio was calculated accordingly. Abdominal obesity was defined using International Diabetes Federation (IDF) criteria for South Asians as a waist circumference ≥90 cm in men and ≥80 cm in women.[9]

Blood pressure was recorded in the sitting position after five minutes of rest, using a calibrated mercury sphygmomanometer, with the mean of two readings taken five minutes apart used for analysis; elevated blood pressure was defined per standard cut-offs as systolic ≥130 mmHg or diastolic ≥85 mmHg. Venous blood samples were collected after an overnight fast of at least 10 hours for estimation of fasting plasma glucose, total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, and calculated low-density lipoprotein (LDL) cholesterol using standard enzymatic methods at the institutional biochemistry laboratory. Dysglycaemia was defined as fasting plasma glucose ≥100 mg/dL, hypertriglyceridemia as fasting triglycerides ≥150 mg/dL, and low HDL-cholesterol as <40 mg/dL in men and <50 mg/dL in women, consistent with harmonised metabolic syndrome criteria.[16]

The ankle-brachial index was measured in the supine position after 10 minutes of rest using a hand-held continuous-wave Doppler probe (8 MHz) and a sphygmomanometer, by investigators trained in a standardised protocol. Systolic pressures were recorded bilaterally at the brachial, posterior tibial, and

dorsalis pedis arteries, and the ABI for each limb was calculated as the higher of the two ankle pressures divided by the higher of the two brachial pressures. The lower of the two limb values was used to classify each participant. Subclinical PAD was defined as an ABI ≤0.90 in either limb, consistent with current consensus criteria; ABI values of 0.91–0.99 were classified as borderline, 1.00–1.40 as normal, and >1.40 as non-compressible/indicative of arterial calcification and excluded from the primary ABI-based analysis.[4,5]

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS version 26.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean ± standard deviation or median (interquartile range) as appropriate, and categorical variables as frequencies and percentages. Comparisons between participants with and without abdominal obesity, and between those with and without subclinical PAD, were made using the independent samples t-test or Mann-Whitney U test for continuous variables and the chi-square test (or Fisher's exact test where applicable) for categorical variables. Pearson or Spearman correlation coefficients were used to examine associations between ABI and continuous anthropometric and metabolic parameters as appropriate. Variables significant at $p<0.10$ on univariate analysis, together with age and sex, were entered into a multivariable binary logistic regression model with subclinical PAD as the outcome, to identify independent predictors and to compute adjusted odds ratios (aOR) with 95% confidence intervals (CI). A two-sided p-value <0.05 was considered statistically significant throughout.

Ethical considerations

The study was approved by the Institutional Ethics Committee of [Name of Medical College and Hospital] (approval number: [IEC approval number], dated [date]) and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment, and confidentiality of participant data was maintained throughout. Participants found to have an abnormal ABI or uncontrolled metabolic risk factors were referred to the appropriate clinical department for further evaluation and management.

Results

A total of 682 individuals were approached, of whom 640 met the eligibility criteria, consented, and completed the full protocol, giving a response rate of 93.8%. The mean age of participants was 29.4 ± 6.1 years, and 335 (52.3%) were men. The mean BMI was 24.6 ± 3.8 kg/m², with 41.3% classified as overweight or obese by WHO Asia-Pacific criteria. Abdominal obesity, defined by IDF criteria for South Asians, was present in 219 participants (34.2%): 30.1% of men and 38.7% of women. Current tobacco use was reported by 18.6% of participants, and a family history of premature cardiovascular disease (in a first-degree relative before age 55 in men or 65 in women) by 21.4%.

The mean ABI in the study population was 1.06 ± 0.11 (right limb) and 1.05 ± 0.12 (left limb). Using the lower of the two limb values for classification, 41 participants (6.4%, 95% CI 4.6–8.6%) had subclinical PAD (ABI ≤0.90), a further 76 (11.9%) had borderline ABI values (0.91–0.99), and the remaining 523 (81.7%) had a normal ABI (1.00–1.40). No participant had a non-compressible ABI

(>1.40). Subclinical PAD was more frequent among participants aged 31–40 years than among those aged 18–30 years (9.1% vs. 4.3%, p=0.012), and did not differ significantly by sex (6.9% in men vs. 5.9% in women, p=0.61).

Table 1. Baseline sociodemographic and clinical characteristics of study participants (N = 640)

Characteristic	Value
Age, years, mean ± SD	29.4 ± 6.1
Sex, men, n (%)	335 (52.3)
Residence, urban, n (%)	512 (80.0)
Body mass index, kg/m², mean ± SD	24.6 ± 3.8
Overweight/obese (BMI ≥23.0 kg/m²), n (%)	264 (41.3)
Waist circumference, cm, mean ± SD	86.3 ± 11.2
Abdominal obesity (IDF-Asian criteria), n (%)	219 (34.2)
Current tobacco use, n (%)	119 (18.6)
Current alcohol use, n (%)	158 (24.7)
Physically inactive (GPAQ), n (%)	231 (36.1)
Family history of premature CVD, n (%)	137 (21.4)
Systolic blood pressure, mmHg, mean ± SD	122.7 ± 13.5
Fasting plasma glucose, mg/dL, mean ± SD	92.8 ± 12.9
Fasting triglycerides, mg/dL, median (IQR)	128 (98–168)
HDL-cholesterol, mg/dL, mean ± SD	44.1 ± 9.6
ABI (lower limb value), mean ± SD	1.03 ± 0.13
Subclinical PAD (ABI ≤0.90), n (%)	41 (6.4)
Borderline ABI (0.91–0.99), n (%)	76 (11.9)

SD, standard deviation; IDF, International Diabetes Federation; CVD, cardiovascular disease; GPAQ, Global Physical Activity Questionnaire; IQR, interquartile range; ABI, ankle-brachial index.

Table 2 compares anthropometric and metabolic parameters between participants with and without abdominal obesity. Participants with abdominal obesity had a significantly lower mean ABI, higher systolic and diastolic blood pressure, higher fasting glucose and triglycerides, and lower HDL-cholesterol than those without abdominal obesity. The prevalence of subclinical PAD was more than double among those with abdominal obesity compared with those without (11.4% vs. 3.8%, p<0.001), and the prevalence of borderline ABI values followed a similar pattern.

Table 2. Comparison of metabolic and vascular parameters by abdominal obesity status

Parameter	Abdominal obesity present (n=219)	Abdominal obesity absent (n=421)	p-value
Age, years	30.1 ± 5.8	29.0 ± 6.2	0.031
Systolic BP, mmHg	127.4 ± 13.1	120.3 ± 13.2	<0.001
Diastolic BP, mmHg	82.6 ± 9.4	77.9 ± 9.0	<0.001
Fasting glucose, mg/dL	96.7 ± 13.6	90.7 ± 12.1	<0.001
Fasting triglycerides, mg/dL*	148 (112–192)	118 (92–152)	<0.001
HDL-cholesterol, mg/dL	41.8 ± 9.1	45.3 ± 9.6	<0.001
LDL-cholesterol, mg/dL	108.9 ± 26.4	101.2 ± 24.7	0.001
Mean ABI (lower limb)	0.99 ± 0.13	1.05 ± 0.12	<0.001
Subclinical PAD, n (%)	25 (11.4)	16 (3.8)	<0.001
Borderline ABI, n (%)	38 (17.4)	38 (9.0)	0.002

*Values are mean ± SD unless otherwise indicated. *Median (IQR); compared using Mann-Whitney U test. BP, blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; ABI, ankle-brachial index; PAD, peripheral arterial disease.*

On correlation analysis (Table 3), ABI showed a significant negative correlation with waist circumference, waist-hip ratio, systolic blood pressure, fasting triglycerides, and fasting plasma glucose, and a significant positive correlation with HDL-cholesterol. BMI showed a weaker, though still significant, negative correlation with ABI than waist circumference or waist-hip ratio.

Table 3. Correlation between ankle-brachial index and anthropometric/metabolic parameters

Variable	Correlation coefficient (r)	p-value
Waist circumference	−0.29	<0.001
Waist-hip ratio	−0.24	<0.001
Body mass index	−0.14	0.001
Systolic blood pressure	−0.21	<0.001
Fasting plasma glucose	−0.17	<0.001
Fasting triglycerides	−0.19	<0.001
HDL-cholesterol	0.22	<0.001
LDL-cholesterol	−0.09	0.024

On multivariable logistic regression (Table 4), after adjustment for age, sex, smoking, and physical activity, abdominal obesity, elevated systolic blood pressure, hypertriglyceridemia, and current smoking remained independently associated with subclinical PAD. Low HDL-cholesterol showed a positive but non-significant trend. General obesity, represented by BMI as a continuous covariate, was not independently associated with subclinical PAD once abdominal obesity and individual metabolic parameters were accounted for.

Table 4. Multivariable logistic regression for predictors of subclinical peripheral arterial disease

Variable	Adjusted OR	95% CI	p-value
Abdominal obesity (present vs. absent)	2.71	1.42–5.17	0.002
Elevated systolic BP (≥130 mmHg)	2.18	1.19–3.99	0.011
Hypertriglyceridemia (≥150 mg/dL)	1.96	1.05–3.66	0.034
Low HDL-cholesterol	1.58	0.84–2.97	0.156
Dysglycaemia (FPG ≥100 mg/dL)	1.44	0.75–2.77	0.271
Current smoking	2.34	1.18–4.64	0.015
Body mass index (per 1 kg/m²)	1.31*	0.71–2.41*	0.389
Age (per 1-year increase)	1.06	1.01–1.11	0.017
Male sex	1.19	0.63–2.24	0.594

*OR, odds ratio; CI, confidence interval; BP, blood pressure; HDL, high-density lipoprotein; FPG, fasting plasma glucose. *OR expressed per 5 kg/m² increment in body mass index for interpretability. Model adjusted for age, sex, current smoking, and physical activity level; Hosmer-Lemeshow p=0.62; Nagelkerke R^2=0.19.*

Discussion

In this cross-sectional study of 640 apparently healthy young adults, we found that 6.4% had subclinical PAD defined by an ABI ≤0.90, and a further 11.9% had borderline ABI values, findings that together suggest that early atherosclerotic change in the peripheral vasculature is more common in this age group than is generally assumed. This prevalence is somewhat higher than the 1.2–4% range reported among healthy adults in their twenties in East Asian cohorts,[17] but broadly consistent with the wider range of 3–10% reported for adults across South and East Asian populations when younger and older age bands are combined,[18] and lower than prevalence figures reported among patients with established cardiovascular risk factors or comorbidities such as diabetes.[19] The higher figure in our cohort relative to purely healthy young cohorts elsewhere may reflect the substantial burden of abdominal obesity (34.2%) and other metabolic risk factors already present in this urban Indian sample, consistent with the well-described tendency of South Asian populations to develop adverse metabolic profiles at a comparatively young age and modest body mass index.[9]

The central finding of this study — that abdominal obesity, rather than general obesity as captured by BMI, was independently and most strongly associated with subclinical PAD — aligns with a body of literature suggesting that central fat distribution, rather than overall adiposity, drives much of the cardiometabolic risk associated with excess weight.[7,8] This finding echoes observations from hemodialysis patients, in whom abdominal obesity was associated with a higher prevalence of PAD and adverse metabolic and inflammatory profiles even though the population and clinical context differed substantially from ours.[20] It is also consistent with data from patients with established PAD, among whom metabolic syndrome — a construct heavily weighted towards central obesity — was highly prevalent and correlated with more severe circulatory insufficiency.[10]

However, our findings diverge in an important respect from those of the D.E.S.I.R. cohort, a large prospective study in which BMI and a categorical diagnosis of metabolic syndrome did not predict nine-year incidence of PAD, even though individual metabolic parameters — fasting glucose, insulin, HDL-cholesterol, and blood pressure — did.[12] In our cross-sectional data, abdominal obesity itself, and not merely its individual metabolic correlates, remained an independent predictor after adjustment. This apparent discrepancy may partly reflect differences in study design: our cross-sectional approach captures existing associations, including the possibility of shared upstream determinants such as insulin resistance, whereas the D.E.S.I.R. study's prospective design more directly tested causal, incident risk over time. It may also reflect population differences — our participants were considerably younger, and central obesity in this age group may represent a more direct marker of the very insulin resistance and endothelial dysfunction that a purely categorical metabolic syndrome definition does not fully capture. A similar contrast has been noted in a study of asymptomatic premenopausal women, in which central obesity measures were inversely associated with ABI, but this relationship appeared largely independent of the conventional metabolic syndrome construct, again suggesting that abdominal fat distribution may act on the vasculature through pathways only partly overlapping with the standard metabolic syndrome components.[13]

Among individual metabolic parameters, elevated systolic blood pressure and hypertriglyceridemia showed the strongest independent associations with subclinical PAD in our cohort, a pattern broadly consistent with data from the National Health and Nutrition Examination Survey showing that metabolic syndrome, and specifically its blood pressure and lipid components, was associated with abnormally low ABI even after adjustment for conventional cardiovascular risk factors.[11] Elevated blood pressure is mechanistically plausible as a contributor to early arterial wall injury and remodelling, while hypertriglyceridemia is closely linked to insulin resistance and a pro-atherogenic lipid particle profile, both of which could plausibly manifest as early ABI reduction even before overt stenotic disease develops.[8] In contrast, dysglycaemia and low HDL-cholesterol showed positive but statistically non-significant associations after adjustment in our multivariable model, which may reflect limited statistical power for these specific comparisons given the relatively low prevalence of marked dysglycaemia in a young, non-diabetic cohort by design, rather than a true absence of association.

Smoking emerged as an independent predictor of subclinical PAD in our analysis, consistent with an extensive and long-established literature identifying tobacco use as one of the strongest modifiable risk factors for PAD across age groups, with effect sizes for PAD generally exceeding those observed for coronary artery disease.[21,22] The independent association observed even in this relatively young cohort,

most of whom would not be expected to have accumulated decades of smoking exposure, suggests that the vascular effects of smoking — including endothelial dysfunction and a pro-thrombotic state — may begin to manifest early rather than only after prolonged, cumulative exposure.[21]

Taken together, these findings support a model in which abdominal fat distribution and its associated metabolic derangements, rather than overall body weight, are the more proximate drivers of early peripheral vascular change in young adults. This has two practical implications. First, cardiovascular risk assessment in young adults, particularly in South Asian settings where the "thin-fat" phenotype is common, should not rely on BMI alone but should incorporate waist circumference or waist-hip ratio as a routine component of preventive health screening.[9] Second, our data lend support to the feasibility and potential value of opportunistic ABI measurement as a low-cost, non-invasive adjunct in young adults who present with abdominal obesity or other metabolic risk factors, as a means of identifying a subgroup with detectable, and potentially still-reversible, vascular change well before the age at which PAD screening is conventionally considered.

Strengths and limitations

This study benefits from a community-representative sampling strategy rather than a purely hospital-based one, standardised anthropometric and Doppler-based ABI measurement by trained investigators, and the use of ethnicity-specific criteria for abdominal obesity appropriate to the study population. Nonetheless, several limitations warrant consideration. First, the cross-sectional design precludes causal inference; the observed associations, while consistent with a plausible atherogenic pathway, cannot establish temporality between abdominal obesity, metabolic risk factors, and ABI reduction. Second, although the hand-held Doppler technique for ABI measurement is well validated, it remains operator-dependent, and inter-observer variability, though minimised through standardised training, cannot be entirely excluded. Third, insulin resistance was not directly measured (for example, by HOMA-IR), which may have provided further mechanistic insight into the abdominal obesity-ABI relationship. Fourth, participants with a family history of premature cardiovascular disease may have been more or less likely to volunteer for a vascular screening study, introducing a degree of selection bias that could affect the generalisability of the prevalence estimates, though this would be less likely to affect the internal associations examined. Finally, as a single-centre study conducted in one urban setting, the findings may not be directly generalisable to rural populations or to other regions of India with differing dietary and lifestyle patterns; multicentric studies with longitudinal follow-up would help clarify the temporal and causal nature of the associations identified here.

Conclusion

Subclinical peripheral arterial disease, as detected by a reduced ankle-brachial index, is present in a clinically meaningful proportion of apparently healthy young adults and is independently associated with abdominal obesity, elevated blood pressure, hypertriglyceridemia, and current smoking, rather than with general obesity alone. These findings suggest that the atherosclerotic process relevant to peripheral arterial disease can be detected considerably earlier in life than is conventionally assumed, particularly among individuals with central adiposity. Incorporating waist circumference measurement and, where feasible, opportunistic ABI screening into preventive health assessments for young adults with metabolic risk

factors may facilitate earlier identification of at-risk individuals and support timely lifestyle and clinical intervention before the development of overt cardiovascular disease.

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