Review Article

Ankle-Brachial Index as a Predictor of Cardiovascular and Cerebrovascular Events in Hypertension: A Systematic Review

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Highlights

- Abnormal ankle–brachial index (ABI) values are strong predictors of cardiovascular and cerebrovascular risk in individuals with hypertension. Both low (≤0.9) and high (>1.4) ABI readings are associated with adverse vascular outcomes.

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ABSTRACT

Objectives: Hypertension is a major cause of cardiovascular and cerebrovascular morbidity and mortality worldwide. The ankle-brachial index (ABI) is a simple, noninvasive test usually used for peripheral artery disease detection, but its prognostic value in hypertensive patients is still poorly defined. In this study, we sought to systematically review the relationship between abnormal ABI values and cardiovascular and cerebrovascular events in adults with hypertension.

Methods: A systematic literature search was conducted in PubMed, Scopus, and Web of Science up to March 2025 for observational studies evaluating ABI in hypertensive populations. Studies were included if they stratified outcomes by ABI categories and reported at least one cardiovascular or cerebrovascular outcome. Data on study design, population characteristics, measurement and cutoff values of ABI, outcomes, duration of follow-up, and adjustment for confounding variables were extracted. Study quality was assessed according to the Newcastle-Ottawa Scale.

Results: Twenty-two studies from varied geographic and clinical populations were eligible. Both low ABI (≤0.9) and high ABI (>1.4) were consistently associated with elevated risks of myocardial infarction, heart failure, stroke, and all-cause or cardiovascular mortality, independent of conventional risk factors. Low ABI was also associated with subclinical target organ damage, such as left ventricular hypertrophy and silent brain infarct. Some studies have indicated that modified cutoffs (<1.05 or ≤1.10) help to detect risk in some populations, such as older and Asian cohorts. Measurement techniques and ABI cutoffs differed, reducing comparability.

Conclusion: Abnormal ABI is independently associated with increased risks of adverse cardiovascular and cerebrovascular outcomes in patients with hypertension. Standardized protocols for ABI measurement and cutoff points are required to maximize its utility in risk stratification. Prospective studies are needed to determine whether ABI-guided interventions can improve outcomes.

Keywords: Hypertension; Ankle–Brachial Index (ABI); Cardiovascular Risk; Cerebrovascular Events; Peripheral Artery Disease (PAD); Stroke; Myocardial Infarction

RHYTHM
Research in Heart Yield and Translational Medicine

Introduction

Hypertension is one of the most prevalent chronic conditions worldwide and a leading risk factor for cardiovascular disease, stroke, kidney disease, and premature death.[1,2]

More than a quarter of the world's adult population (approximately 1 billion) had hypertension in 2000, with this proportion projected to increase to 29% (1.56 billion) by 2025.[3]

Despite guideline-directed lifestyle interventions and pharmacologic therapy, a substantial proportion of patients with hypertension remain at elevated cardiovascular risk. Studies have shown that clinically significant residual risk persists even among individuals who achieve recommended blood pressure targets.[4-6] This continued risk of vascular events, despite the availability of effective treatments, underscores the need for additional, simple, noninvasive prognostic tools to better identify high-risk individuals beyond office blood pressure levels and traditional risk scores. The ankle-brachial index (ABI) is one such candidate tool.[6]

ABI is a simple, noninvasive, and inexpensive bedside test, calculated as the ratio of systolic blood pressure at the ankle to that at the brachial artery.[7-9] It is widely employed to screen for peripheral artery disease (PAD), typically defined by an ABI of 0.9 or lower,[10,11] and abnormal ABI values have been demonstrated to reflect the systemic burden of atherosclerosis and arterial stiffness rather than limb ischemia alone.[10,12] Both abnormally low (≤0.9) and high (>1.4) ABI values have been associated with increased risks of myocardial infarction, stroke, heart failure, and all-cause mortality.[13,14]

Nonetheless, the prognostic role of ABI, specifically in patients with hypertension, is less clearly established.[15,16] Individuals with hypertension often have hypertension-mediated organ damage, including vascular remodeling, endothelial dysfunction, and microvascular disease, which is not fully captured by routine clinical evaluation.[15,17] ABI may be a convenient marker of such vascular damage and help refine risk stratification in this group. However, existing studies in hypertensive populations have reported heterogeneous findings, used different ABI cutoffs, and evaluated diverse cardiovascular and cerebrovascular outcomes.[18]

To address this knowledge gap, we conducted a systematic review of observational studies evaluating associations between abnormal ABI and cardiovascular, cerebrovascular, and peripheral outcomes among adults with hypertension. Our objectives were to (1) summarize the strength and consistency of associations for low and high ABI categories with major clinical endpoints, (2) describe how ABI was measured and categorized across studies, and (3) highlight unresolved inconsistencies and areas requiring further research.

Methods

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guideline and was registered in PROSPERO (CRD420251236723). We searched PubMed, Scopus, and Web of Science using combinations of the following keywords and Medical Subject Headings (MeSH) terms: "ankle-brachial index," "ABI," "hypertension," "high blood pressure," "cardiovascular outcomes," "cerebrovascular outcomes," "stroke," "myocardial infarction," "mortality," and "peripheral artery disease." Searches of Embase and Google Scholar yielded only duplicates or studies that did not meet the eligibility criteria and are therefore not represented in the PRISMA flow diagram. The detailed search query is available in the Appendix.

All titles and abstracts identified through the database searches were independently screened by two authors (K.O. and P.B.), and conflicts were resolved by a third reviewer (H.B.). Articles deemed potentially eligible based on title and abstract screening underwent full-text review. Disagreements regarding study inclusion were resolved through discussion among all authors. Reference management and screening were facilitated using Rayyan software.

The present study included observational studies of any design (prospective or retrospective cohort studies, case-control studies, and cross-sectional analyses) that evaluated the association between ABI categories and cardiovascular or

cerebrovascular outcomes in adults (≥18 years) with hypertension. Eligible studies were required to report ABI measurements and stratify outcomes according to ABI ranges (eg, <0.9, normal, and >1.4). Studies were excluded if they involved mixed populations of individuals with and without hypertension but did not report results separately for the hypertensive subgroup; did not compare outcomes across ABI categories; or focused solely on ABI measurement techniques or diagnostic accuracy without reporting clinical outcomes. Conference abstracts, review articles, editorials, case reports, and studies without accessible full texts were also excluded. No minimum follow-up duration was required. For cohort studies, the length of follow-up was extracted and reported. Only English-language human studies were considered. The study selection process is summarized in a PRISMA flow diagram (Figure 1).

Data were extracted independently by three reviewers using a standardized template and cross-checked for confirmation. Extracted variables included the following: study characteristics (authors, year, country, design, sample size, and population demographics); ABI measurement methods and cutoff definitions; cardiovascular and cerebrovascular events studied; follow-up duration (for prospective studies); reporting of mortality and PAD outcomes; statistical adjustments for confounders; principal results and risk estimates; and risk-of-bias or quality scores (if available). Continuous variables were reported as mean (SD) and categorical variables as No. (%). Conflicts were resolved through discussion or third-party arbitration. The principal domains assessed were selection bias, exposure and outcome assessment, and statistical adjustment for confounding variables. Because of substantial clinical and methodologic heterogeneity across studies—including differences in study design, baseline risk, ABI measurement devices and protocols, ABI categories, and outcome definitions—a formal meta-analysis was not performed. Evidence was synthesized narratively.

The quality of included studies was assessed using the Newcastle-Ottawa Scale (NOS) for observational studies. Risk of bias was evaluated using the ROBINS-I tool for nonrandomized studies. The NOS assesses selection, comparability, and outcome domains, whereas ROBINS-I evaluates bias due to confounding, participant selection, exposure classification, deviations from intended interventions, missing data, outcome measurement, and reporting. Each study was scored independently by two reviewers. Discrepancies were resolved by consultation with a third reviewer. Further information is provided in the supplementary tables.

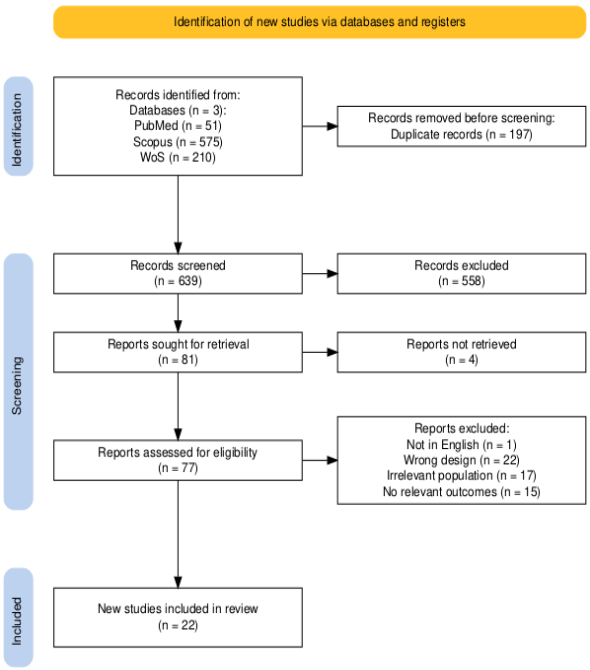

Figure 1. Study flow diagram

Clinical outcomes were categorized as follows: cardiovascular events (myocardial infarction, angina, coronary artery disease, and heart failure); composite cardiovascular events; cerebrovascular events (ischemic stroke, hemorrhagic stroke, transient ischemic attack, and silent brain infarct); mortality (all-cause or cardiovascular); and peripheral events (PAD progression or development, limb events, or lower extremity amputation). Where available, adjusted relative risks, hazard ratios, or odds ratios (ORs) were reported.

Results

Literature Search

The initial search identified 836 records. After removal of duplicates, 81 records were screened. Full-text review of these 81 articles resulted in the exclusion of 55 articles that did not meet the inclusion criteria. A total of 22 articles were included for data extraction.

Study Characteristics

The review included studies from several countries, including Spain, Hungary, Poland, Brazil, and Turkey (Table 1). Study designs included prospective cohorts, cross-sectional studies, and case-control studies. Sample sizes varied from 40 to approximately 21,000 individuals. Study populations consisted of patients with hypertension, most of whom had additional cardiovascular risk factors such as chronic kidney disease, diabetes, or a history of cardiovascular events; however, three studies focusing on such risk factor cohorts were excluded.[19-21]

Mean age ranged from 57 to 69 years, with most populations being middle-aged to elderly. Most participants were male, though sex distribution varied across studies. ABI measurement methods varied, including Doppler ultrasound, oscillometric devices, and mercury manometers. Characteristics of the included studies are presented in (Tables 1 and 2).

ABI was presented as the ratio of systolic pressure at the ankle to that in the brachial artery. The following cut points were used in most studies (eg, Armas-Padrón et al,[22] Farkas et al,[16] and Jiménez-Balado et al[23]): abnormally low ABI (<0.9), indicative of PAD; normal ABI (0.91–1.4); and abnormally high ABI (>1.4), suggesting arterial stiffening or incompressible arteries. Some studies, such as those by Shi et al24 and Zhou et al,[25] employed different cut points, such as less than 1.05 or 1.10 or less for low ABI, particularly in population-specific analyses or in combination with other vascular markers such as the cardio-ankle vascular index (CAVI).

Overall study quality was moderate to good. Based on the NOS, 14 studies scored between 7 and 9 points and were rated as good quality, whereas the remaining studies scored 5 to 6 points, indicating moderate quality. The ROBINS-I assessment showed that most studies had low-to-moderate risk of bias in the domains of participant selection, exposure classification, and deviations from intended interventions. Nonetheless, several studies demonstrated moderate-to-serious risk of bias due to residual confounding (eg, incomplete adjustment for comorbidities) and outcome measurement variability (eg, nonuniform ABI protocols). No study was judged to have critical risk of bias (see Tables S4 and S5).

In addition to demographic and methodologic variability, included studies differed in sample size, follow-up duration, and outcomes assessed. Sample sizes ranged from small clinical cohorts of 40 to 149 participants[18,24,26] to large-scale population-based studies including more than 20,000 hypertensive adults.[22,27,28] Follow-up duration among prospective cohorts varied from 13 months to 14 years,[21,22,29-32] whereas cross-sectional studies reported outcomes at a single time point. Cardiovascular outcomes most commonly included myocardial infarction, angina, heart failure, acute coronary syndromes, and cardiovascular mortality.[20-23,29-32] Cerebrovascular outcomes comprised ischemic stroke, hemorrhagic stroke, transient ischemic attack, and silent brain infarct.[21,27,29,30] Several studies also assessed hypertension-mediated organ damage, including left ventricular hypertrophy, proteinuria, carotid atherosclerosis, and PAD progression.[18,19,24,33-36]

Outcome Comparisons: Low ABI, Normal ABI, and High ABI

Association between ABI and cardiovascular outcomes

The association between abnormal ABI and cardiovascular outcomes was the most commonly reported endpoint, particularly for low ABI (≤0.9). By way of example, a Spanish cohort[22] showed that participants with an ABI of 0.9 or less had significantly increased cardiovascular mortality and hospitalization rates (Table 2). Similarly, a Hungarian study[33] involving more than 21,000 individuals demonstrated that low ABI was associated with a hazard ratio of 1.87 (95% CI, 1.63 to 2.16) for mortality after adjustment for confounding variables.

Table 1. Characteristics of the studies included in the present study

Author	Country	Study Design	Sample Size	Population	Mean Age / Age Range	% Male	Comorbidity	ABI Measurement Method
Armas-Padrón et al [22]	Spain	Prospective cohort	243	Adult Caucasian with hypertension from a primary care urban population	68 (13) / 70.3 (12.1) (abnormal)	46%	CKD, diabetes, prior CVD other than PAD	ABI Measurement Method
Farkas et al [33]	Hungary	Prospective cohort	21,876	Hypertensive patients aged 50 to 75 y	60.9 (8.8)	41.9%	Diabetes (41%), MI (11%), stroke (4.8%), hyperlipidemia (33.6%), CKD (GFR <60: 16.2%), hyperuricemia (20.2%), obesity (38.3%), smoking (19)	Automated oscillometric device (MESI ABPI MD)
Myslinski et al [27]	Poland	Case-control observational	81	51 treated hypertensive patients with first ACS vs 30 hypertensive controls	~64.8 (range, 47–80)	62.7% (32/51 cases), 53% (16/30 controls)	Diabetes, obesity, smoking (similar across groups)	Continuous wave Doppler (ELITE 200, 5 MHz)
de Albuquerque et al [29]	Brazil		40			100%		Color Doppler and PW Doppler; lowest ABI of either leg or brachial artery
Tosun et al [19]	Turkey	Prospective and cross-sectional	149	Male patients with arterial hypertension without cardiovascular complications	57.92 (7.61)		–	
Tao et al [37]	China	Retrospective cross-sectional observational	600	Hypertensive patients aged 18 to 75 y with suspected coronary atherosclerotic heart disease	Median: 62 (IQR, 56–68; CHD) / 59 (IQR, 53–67; non-CHD)	CHD: 49.03%; non-CHD: 53.53%	Diabetes (44.3% CHD vs 34.9% non-CHD), smoking (43.2% CHD vs 27.4% non-CHD), heart failure (1.1% CHD), atrial fibrillation (2.5% CHD), dyslipidemia, elevated homocysteine, elevated HbA_{1c}, elevated LVMI	Doppler ultrasound (highest mean systolic pressure)
Gómez-Marcos et al [30]	Spain	Cross-sectional	391	Patients with hypertension aged 30 to 80 y	Low-moderate risk: 52.64 (11.50); High risk: 59.04 (10.76)	NA	Diabetes, dyslipidemia, metabolic syndrome	OMRON arteriosclerosis detector; BP from brachial and posterior tibial arteries; lowest ABI used
Shi et al [24]	China	Cross-sectional	10750	Adults with hypertension aged 27 to 96 y, excluding those with atrial fibrillation	63.81 (9.23)	47.18% (5072/10750)	Diabetes mellitus: 18.03; CHD: 4.95, stroke: 6.42, PAD: 3.19, smoking, dyslipidemia, alcohol consumption	Portable Doppler system (Minidop Es-100Vx)
Korhonen et al [20]	Finland	Cross-sectional analysis within a population cohort	495	Subjects with hypertension aged 45 to 70 y without established CVD, renal disease, or diabetes	60 (7)	45%	Newly detected: low ABI (≤1.00), LVH, eGFR ≤ 60 mL/min/1.73 m², newly diagnosed diabetes	Omron Colin BP-203RPE III device
Jiménez-Balado et al [23]	Spain	Longitudinal observational cohort	964	Patients with hypertension aged 50 to 70 y, stroke-free at inclusion	Median: 64 (IQR, 59–67)	49.4% male	Hypertension, DM, dyslipidemia, IHD, prior MI, smoking, overweight/obesity, sedentary lifestyle	Doppler-based ABI measurement (UltraTec PD1v)
Kario et al [28]	Japan	Multicenter prospective observational	4716	Adults aged ≥30 y with ≥1 cardiovascular risk factor (mostly hypertension)	68.5 (1.4)	51.9%	Hypertension (91.5), DM (32.4), dyslipidemia (57.3), CVD (22.4), CKD (19.7), atrial fibrillation (9.2), metabolic syndrome (10.5), chronic obstructive	Photoplethysmography with Vicorder device

Vasan et al [34]	USA	Cross-sectional and prospective cohort	7898	Adults (mean age ~51.6), community sample across FHS generations, with or without hypertension	51.6 (15.9)	46%	pulmonary disease (1.7), sleep apnea syndrome (5.0), smoking	Hypertension (treated and untreated), DM (8.3), dyslipidemia, smoking (12), obesity, family history of CVD, elevated cholesterol ratio, CKD	Cuff oscillometric method (Vasera-1500/3000)
Zhou et al [25]	China	Cross-sectional analysis based on the H-type Hypertension Registry Study	10906	Chinese adults with hypertension aged ≥□18 y	63.86 (9.25)	47.08%	DM (11.35), stroke (6.49), current smoking (26.33), current drinking (22.67), hypercholesterolemia, hypertriglyceridemia, CKD, hyperuricemia, CHD (5.07)		Standardized protocol using an oscillometric device
Sun et al [35]	China	Community-based cross-sectional	3953	Han Chinese adults with hypertension aged 40 to 75 y from rural China	58.08	34.50%	Stroke (10.6), CAD (9.0), DM (5.0), elevated TG, high LDL-C, low HDL-C, elevated BUN		Omron Colin BP-203RPE III device; lowest ABI from either leg used
Peng et al [36]	China	Cross-sectional	360	Elderly patients with essential hypertension; control group of healthy individuals	EH group: 62.8 (12.6); Control: 62.5 (11.8)	EH: 54.3% Control: 53.8%	Carotid atherosclerosis (59.6% of the EH group), hyperlipidemia, elevated TG, elevated serum creatinine		Portable Doppler device (ES-101EX, HADECO, 8 MHz); higher of both legs' ABI was used
Ramírez-Torres et al [38]	Spain	Prospective cohort	140	Hospital-recruited patients with hypertension (aged ~68), no prior PAD diagnosis	68.2 (10.6)	54%	DM, hyperlipidemia, smoking		Omron Colin BP-203RPE III; lower ABI value used for analysis
Jager et al [39]	Netherlands	Multicenter prospective observational	631	Patients with hypertension aged ≥40 y hospitalized with ACS	67.4 (11.4)	48% survivors, 55% nonsurvivors	DM, prior CHD or stroke, LVH, CKD		Doppler ABI (Nicolet Vascular Elite 100R); highest ankle / highest brachial
Mulia et al [31]	Indonesia	Prospective cohort	104	Adult Caucasian with hypertension from a primary care urban population	68 (13) / 70.3 (12.1) (abnormal)	82.7%	CKD, diabetes, prior CVD other than PAD		Doppler ABI (BIDOP ES-100V3), lowest ankle / highest brachial pressure
Korhonen et al [21]	Finland	Prospective cohort	972	Hypertensive patients aged 50 to 75 y	60.9 (8.8)	47%	Diabetes (41%), MI (11%), stroke (4.8%), hyperlipidemia (33.6%), CKD (GFR <□60: 16.2%), hyperuricemia (20.2%), obesity (38.3%), smoking (19)		ABI Measurement Method
Luo et al [26]	China	Case-control observational	3047	51 treated hypertensive patients with first ACS vs 30 hypertensive controls	~64.8 (range, 47–80)	52.5%	Diabetes, obesity, smoking (similar across groups)		Automated oscillometric device (MESI ABPI MD)
Monteiro et al [32]	Brazil		65			24%			Continuous wave Doppler (ELITE 200, 5 MHz)
Morillas et al [40]	Spain	Prospective and cross-sectional	1101	Male patients with arterial hypertension without cardiovascular complications	57.92 (7.61)	67.7%	–		Color Doppler and PW Doppler; lowest ABI of either leg or brachial artery

ABI: ankle-brachial index; ABPI: ankle-brachial pressure index; ACS: acute coronary syndrome; AHA: American Heart Association; BP: blood pressure; BUN: blood urea nitrogen; CAD: coronary artery disease; CAVI: cardio-ankle vascular index; CHD: coronary heart disease; CKD: chronic kidney disease; CVD: cardiovascular disease; DM: diabetes mellitus; eGFR: estimated glomerular filtration rate; EH: essential hypertension; HDL-C: high-density lipoprotein cholesterol; HTN: hypertension; IGT: impaired glucose tolerance; IHD: ischemic heart disease; IQR: interquartile range; LDL-C: low-density lipoprotein cholesterol; LVH: left ventricular hypertrophy; LVMI: left ventricular mass index; MI: myocardial infarction; NA: not available; NIDDM: non–insulin-dependent diabetes mellitus; PW: pulse wave; SBP: systolic blood pressure; TG: triglycerides; TIA: transient ischemic attack; CAS: carotid atherosclerosis; SAH: subarachnoid hemorrhage

Table 2. Characteristics of the studies included in the present study

Author	ABI Categories Compared	% Abnormal ABI	% With ABI < 0.9	% With ABI > 1.4	Cardiovascular Outcomes Studied	Cerebrovascular Outcomes Studied	Mortality Reported?	Follow-Up Duration	Adjusted For Confounders?
Armas-Padrón et al [22]	Normal ABI vs ABI ≤0.9 vs ABI >1.4	23.5% (57/243) (6.6% ABI ≤ 0.9; 16.9% ABI > 1.4)	28% (16/57)	72% (41/57)	CVD other than PAD/Death, hospitalization or CVD	Stroke	Yes (9 deaths)	2 years	Yes (age, sex, diabetes, CKD)
Farkas et al[33]	ABI ≤0.9 vs >0.9; high ABI >1.3	23.80%	14.4%	9.4%	Total mortality	Not reported	Yes (5.44% deaths)	5 years	Yes (age, BMI, BP, SCORE etc.)
Myslinski et al [27]	Normal vs abnormal (≤0.9 or >1.3)	Not clearly reported as %	Implied high (mean ABI <0.9 in ACS group)	ABI > 1.3 excluded	First ACS episode (STEMI/NSTEMI); significant CAD	Not reported	Not reported	N/A	Yes
de Albuquerque et al [29]	ABI in relation to CHD vs non-CHD	17.5% (7/40)	Not reported	Not reported	Left-ventricular mass index / LVH prevalence	Not reported	Not reported	N/A	No
Tosun et al [19]	Low-moderate vs high-very high risk	Not reported	Not reported	Not reported	LVH, proteinuria	Not reported	ABI OR 0.01 (0.001–0.010) for LVH/proteinuria	N/A	ABI lower in proteinuria (+) and LVH (+) groups
Tao et al [37]	ABI <1.05 vs ≥1.05	Not classified traditionally	Not reported	Not reported	CAD severity (Gensini score, CHD diagnosis)	Not reported	N Not reported	N/A	Yes multivariate logistic regression adjusted for FMD, baPWV, age, sex, diabetes, smoking, SBP, diastolic blood pressure (DBP), HDL-C, LDL-C, triglycerides, homocysteine, HbA1c, and LVMI.
Gómez-Marcos et al [30]	ABI ≤1.00 vs >1.00	Low-moderate risk: 9.8% (17/173); High risk: 13.8% (30/218)	Not reported	Not reported	Renal and PAD, LVH, Carotid intima-media thickness (IMT), Arterial stiffness	Not reported	ABI <0.9 associated with higher cardiovascular risk	N/A	Not provided
Shi et al [24]	ABI <0.9 vs ≥0.9	24.6% (ABI <1.05)	Not reported	0.06%	Not applicable (focused on stroke)	Stroke (ischemic or hemorrhagic, self-reported)	Not reported	N/A	Yes (multivariate logistic regression: age, sex, SBP, DBP, pulse, BMI, lipids, eGFR, etc.)
Korhonen et al [20]	CAVI ≥8 and ABI ≤1.10 vs other combinations	24.8%	24.8%	Not reported	Target organ damage	Not reported	Not reported	N/A	Yes (age, sex, blood pressure, BMI, glucose, lipids, etc.)
Jiménez-Balado et al [23]	ABI <0.9 vs ≥0.9	9.9% (79/964)	9.9% (79/964)	Not reported	Acute MI, angina, heart failure, PAD	Stroke, TIA, hemorrhagic stroke	Yes	5 years (median follow-up: 4.7-5 years)	Yes (age, sex, diabetes, smoking, cholesterol, HDL, waist circumference, DBP)
Kario et al [28]	ABI ≤1.07 vs >1.07	37.20%	2.90%	Not reported	MI, angina, sudden death, heart failure, aortic dissection	Stroke, cerebral hemorrhage, SAH	Yes	Median 5.0 years	Yes (age, sex, BMI, smoking, alcohol, diabetes, CKD, antihypertensive use, office SBP)
Vasan et al [34]	Quartiles: Q1–Q4 (0.90–1.40)	2.7%	2.7%	Not reported	CVD composite (MI, angina, heart failure, PAD, stroke, TIA, CVD-related death)	Stroke, TIA	Yes - 384 CVD events including fatal events (14.1-year median follow-up)	14.1 years (median)	Yes (age, sex, BMI, diabetes, cholesterol ratio, smoking, BP meds, cohort type)
Zhou et al [25]	ABPI <0.90 vs ≥0.90	Low ABI (<1.05): not directly reported,	Not reported	Not reported	CHD	Stroke prevalence reported but not as outcome	Not reported - CHD prevalence	N/A	Yes multivariate logistic regression adjusted for age, sex, BMI, SBP, DBP, heart

		PAD prevalence: 3.22%					used as primary outcome		rate, smoking, drinking, serum homocysteine, glucose, total cholesterol, triglycerides, LDL-C, eGFR, diabetes, and multiple antihypertensive medications.
Sun et al [35]	ABI ≤0.90 vs 0.91–1.40	ABI <=0.9 (excluded): 386 patients; ABI >=1.4 (excluded): 121 patients	9.5% excluded	3.1% excluded	LVH	History of stroke reported, not a study outcome	Not reported	N/A	Yes adjusted for age, sex, BMI, SBP, DBP, serum glucose, triglycerides, total cholesterol, HDL-C, LDL-C, BUN, history of stroke, coronary artery disease, and diabetes.
Peng et al [36]	ABI <0.9 vs ≥0.9	Not categorized formally	Not specified	Not reported	CAS	Not reported	Not reported	N/A	Yes logistic regression adjusted for GGT, homocysteine, and ABI in relation to plaque stability.
Ramírez-Torres et al [38]	Normal ABI vs ABI ≤0.9 vs ABI >1.4	8.60%	8.6%	Not reported	Presence of subclinical atherosclerotic burden including carotid plaques, elevated IMT, and low ABI	Not reported	Not reported	N/A	Yes (smoking, creatinine, hypertension duration; age for plaque presence)
Jager et al [39]	ABI ≤0.9 vs >0.9; high ABI >1.3	11% total (PAD prevalence = 69/631)	11%	Not reported	Cardiovascular mortality, sudden death	Stroke/TIA based on WHO questionnaire (not a primary outcome)	Yes: all-cause and cardiovascular mortality	5 years	Yes (age, sex, IGT, NIDDM, hypertension, lipids, smoking, obesity, IHD)
Mulia et al [31]	Normal vs abnormal (≤0.9 or >1.3)	16.3%	16.3%	0% (excluded if ABI >1.3)	None specifically listed	Stroke data mentioned, but not outcome-focused	Not reported	Not reported	Yes
Korhonen et al [21]	ABI in relation to CHD vs non-CHD	31% (PAD + borderline PAD in hypertensives)	7.3% (39/532 hypertensives)	Not reported	None specifically listed	Not reported	Not reported	N/A	Yes
Luo et al [26]	Low-moderate vs high-very high risk	27.5%	27.5%	0% (ABI >1.4 considered normal)	All-cause mortality, CVD mortality	Yes	Yes	13 months	Yes
Monteiro et al [32]	ABI <1.05 vs ≥1.05	18% (AHA), 32% (modified definition)	18% (AHA), 32% (modified definition)	ABI > 1.4 excluded	Not directly assessed; Framingham Risk Score used	Not reported	Not reported	N/A	Yes
Morillas et al [40]	ABI ≤1.00 vs >1.00	42.6%	42.6%	ABI > 1.5 excluded	Hospital mortality, heart failure, angina, composite endpoint	Stroke and TIA reported but not primary outcomes	Yes	During hospitalization (acute phase)	Yes

ABI: ankle-brachial index; ABPI: ankle-brachial pressure index; ACS: acute coronary syndrome; AHA: American Heart Association; BMI: body mass index; BP: blood pressure; BUN: blood urea nitrogen; baPWW: brachial-ankle pulse wave velocity; CAD: coronary artery disease; CAS: coronary artery stenosis; CAVI: cardio-ankle vascular index; CHD: coronary heart disease; CKD: chronic kidney disease; CV: cardiovascular; CVD: cardiovascular disease; DBP: diastolic blood pressure; eGFR: estimated glomerular filtration rate; FMD: flow-mediated dilation; GGT: γ-glutamyl transferase; HbA1c: hemoglobin A1c; HDL-C: high-density lipoprotein cholesterol; HTN: hypertension; IGT: impaired glucose tolerance; IHD: ischemic heart disease; IMT: intima-media thickness; LDL-C: low-density lipoprotein cholesterol; LVH: left ventricular hypertrophy; LVMI: left ventricular mass index; MI: myocardial infarction; NIDDM: non–insulin-dependent diabetes mellitus; PAD: peripheral artery disease; SAH: subarachnoid hemorrhage; SBP: systolic blood pressure; SCORE: Systematic COronary Risk Evaluation; TG: triglycerides; TIA: transient ischemic attack; WHO: World Health Organization

Additional studies have associated low ABI with higher rates of left ventricular hypertrophy, subclinical atherosclerosis, proteinuria, and acute coronary syndromes.[19,20,26-30,35,38,40] For example, Luo et al[26] found that low ABI was associated with higher all-cause and cardiovascular mortality, with adjusted hazard ratios of 1.62 and 2.45, respectively, for low vs normal ABI. Morillas et al[40] reported that low ABI was associated with in-hospital mortality (OR, 13.0) and with higher rates of heart failure, angina, and composite in-hospital complications. In some studies, ABI was used as a continuous variable, suggesting a linear trend for poorer cardiovascular outcomes with decreasing ABI.[22,33,34,40]

In one study, the prevalence of coronary heart disease was inversely associated with ABI for lower values, highlighting the potential of ABI as a screening tool.[25] The relationship between the protective role of ABI and the roles of GGT and homocysteine (Hcy) in carotid atherosclerosis has also been studied.[36]

A Brazilian investigation conducted only in male hypertensive patients showed a significant difference in the prevalence of left ventricular hypertrophy in those with normal (4%) vs abnormal ABI (35.3%).[29] Similarly, a Polish case-control study of patients with first-episode acute coronary syndromes identified low ABI as the most predictive parameter for the development of acute coronary syndromes, even in the absence of diagnosed PAD.[27]

Other studies provided broader definitions of PAD, and results were often stratified by comorbidities such as chronic kidney disease and diabetes.[32,38,39] Analysis by sex received less emphasis, although adjustment for sex was important in multivariable analyses. Overall, the evidence strongly supports the prognostic importance of low ABI in patients with hypertension, regardless of geographic or clinical setting.[21,31,34,39]

Association of ABI With Cerebrovascular and Brain Outcomes

Some studies examined the association between abnormal ABI and cerebrovascular events. A study in Spain showed that an ABI of 0.9 or less was strongly associated with higher rates of stroke, cardiovascular mortality, and risk of hospitalization during a 2-year follow-up.22 In a Chinese hypertensive cohort, an ABI of less than 1.05 was associated with higher stroke prevalence compared with an ABI of 1.05 or greater (OR, 1.26; 95% CI, 1.05 to 1.50), particularly among participants aged 65 years or older.24 Some studies showed that low ABI and silent brain infarct predicted a higher risk of future cardiovascular events, and screening for these in hypertensive patients could improve early intervention.[23,28,34,40]

PAD and Hypertension-Mediated Organ Damage

Several studies have examined the association between ABI and markers of hypertension-mediated organ damage. Albuquerque et al29 reported that the prevalence of left ventricular hypertrophy was 35.3% among patients with abnormal ABI compared with 4% among those with normal ABI. Tosun et al19 found that lower ABI values were observed in patients with proteinuria and/or left ventricular hypertrophy (OR, 0.01; 95% CI, 0.001 to 0.010 for ABI in relation to the combined presence of these complications). In a rural Chinese cohort, Sun et al35 showed that higher-normal ABI values were associated with left ventricular hypertrophy: compared with the lowest ABI quartile, the ORs for left ventricular hypertrophy were 1.61 (95% CI, 1.32 to 1.96) and 1.83 (95% CI, 1.50 to 2.24) in the third and fourth ABI quartiles, respectively. Peng et al41 reported an inverse association between ABI and carotid atherosclerosis severity, whereas γ-glutamyltransferase and homocysteine were positively correlated with plaque presence.

Impact of Methodologic Differences

A further source of heterogeneity across studies relates to methodologic differences in ABI measurement. The included studies used a range of devices, including Doppler ultrasound,[19,26,27,29,32,35,38,40] oscillometric systems,[22,25,28,33,34,37] and photoplethysmographic methods,23 as well as differing protocols regarding whether the highest or lowest ankle systolic pressure was recorded. These variations can influence ABI values and may contribute to differences in prognostic associations reported

among studies. In addition, the definition of abnormal ABI was not uniform. While several investigations applied the conventional thresholds of less than 0.9 and greater than 1.4,[22,23,26,33,34,40] others drew upon alternative cutoffs such as less than 1.05, 1.07 or less, 1.10 or less, or quartile-based thresholds.[24,25,28,35] This combination of methodologic and threshold variability likely affects comparability and should be considered when interpreting the predictive performance of ABI in hypertensive populations.

High ABI and Combined Abnormal ABI Categories

Data on high ABI were more limited but suggested adverse prognostic implications. In a primary care cohort of 600 hypertensive patients aged 18 to 75 years with suspected coronary atherosclerotic heart disease, a high ABI cutoff of greater than 1.09 identified patients at significantly higher risk of CAS severity. Combining ABI, flow-mediated dilation, and brachial-ankle pulse wave velocity improved predictive accuracy (area under the curve [AUC], 0.800).[37]

Discussion

Summary of Main Findings

This systematic review shows that the ABI is an important predictor of adverse outcomes in patients with hypertension. Both abnormally low (≤0.9) and high (>1.4) ABI values were associated with higher risks of cardiovascular and cerebrovascular disease. These associations were often independent of traditional risk factors, suggesting that ABI may indicate aspects of vascular damage not fully captured by routine clinical markers. Several studies have investigated modified ABI thresholds, particularly for older and Asian populations, which could aid in earlier risk identification.

Interpretation and Pathophysiologic Implications

Low ABI values are a marker of peripheral arterial disease and, more generally, systemic atherosclerosis. There were consistent associations between low ABI and serious cardiovascular events such as myocardial infarction, heart failure, and cardiovascular mortality.[23,24,26-28,33,38,40] These associations persisted after adjustment for other factors such as blood pressure, lipid levels, and diabetes, indicating that ABI may reflect the total burden of vascular stress.[22,23,26,33] For instance, Farkas et al33 found that patients with low ABI were 1.87 times more likely to die during 5 years of follow-up.

High ABI values are often considered a marker of arterial stiffness, likely due to medial calcification. Several studies identified a J-shaped or U-shaped risk curve, and higher ABI values were associated with adverse outcomes.[25,28,33,35] As reported by Sun et al[35] and Zhou et al,25 high ABI was associated with left ventricular hypertrophy and coronary artery calcification in some cases. Still, heterogeneity in definitions and methods limits generalizability.

Cardiovascular Event-Specific Differences

Low ABI was consistently associated with cardiovascular morbidity, though the specific outcomes varied. Some studies focused on acute coronary syndromes or myocardial infarction. For instance, Myslinski et al[27] showed that low ABI was the strongest predictor of first acute coronary syndromes episodes, even in the absence of previous PAD. Other researchers, such as de Albuquerque et al[29] and Sun et al,[35] investigated structural cardiac changes and found a strong association between low ABI and left ventricular hypertrophy.

Large-scale studies examined broader composite outcomes, such as all-cause mortality or hospitalization due to cardiovascular causes. The population-level cohort of Farkas et al[33] and the acute coronary syndrome registry of Morillas et al[40] both demonstrated that ABI was a strong, independent predictor of adverse cardiovascular outcomes in patients with hypertension. Follow-up periods ranged from days (acute phase) to 5 years.

Cerebrovascular Risks and ABI

The association between ABI and cerebrovascular events was consistently reported. Shi et al[23] and Jiménez-Balado et al[24] found that

low ABI was strongly associated with a higher risk of ischemic stroke and silent brain infarct, even in patients with hypertension and no history of stroke. These associations persisted in multivariable models and were particularly strong in older patients. Kario et al28 also found that ABI values of 1.10 or less, especially when combined with other vascular biomarkers such as CAVI, were associated with a higher risk of hemorrhagic stroke and other cerebrovascular events over 5 years. This suggests that ABI may be a useful marker for cerebral small vessel disease in patients with hypertension.

Comparison ABI With Other Vascular Risk Markers

Another important factor is the performance of ABI compared with other well-established markers of vascular risk in hypertension. Several studies have examined ABI in conjunction with markers such as left ventricular hypertrophy, carotid atherosclerosis, proteinuria, and arterial stiffness indices. For instance, de Albuquerque et al[29] and Sun et al35 reported that ABI provided additional predictive information beyond left ventricular hypertrophy, whereas Peng et al36 found that ABI performed well in conjunction with carotid plaque burden as an indicator of atherosclerosis risk. Tosun et al[19] discovered that ABI was associated with proteinuria and left ventricular hypertrophy, confirming its role as a measure of hypertension-mediated organ damage. Further, Kario et al28 found that combining ABI with vascular stiffness markers such as CAVI improved the prediction of cardiovascular outcomes compared with using either marker alone. These findings indicate that ABI has additive predictive value and may improve risk stratification in hypertensive patients when used with established cardiovascular risk markers.

Strengths and Limitations

Despite consistent findings across geographic and clinical populations, heterogeneity among studies complicates interpretation. Methods for measuring ABI varied, from Doppler ultrasound to oscillometric devices, and patient positioning (supine vs sitting) was not uniform. Furthermore, definitions of normal and abnormal ABI thresholds differed across studies. For example, some studies used cutoffs of less than 1.05 or 1.10 or less as more sensitive thresholds, particularly in Asian cohorts.[20,25,28]

Many studies did not fully adjust for important confounders such as duration of hypertension, medication use, or socioeconomic status. Reporting of age- and sex-specific analyses was also insufficient, which limited understanding of which subgroups might benefit most from ABI screening. Nevertheless, several large, well-conducted cohort studies, such as those by Farkas et al,[33] Shi et al,[23] and Kario et al,[28] support the main conclusions. Because of substantial clinical and methodologic heterogeneity across studies, a quantitative meta-analysis was deemed inappropriate. We relied on a narrative synthesis, which may limit the precision of overall effect size estimates.

Clinical Implications

ABI has potential for routine use in the management of hypertension due to its simplicity, low cost, and noninvasive nature. It may help identify patients with residual risk despite controlled blood pressure, which could prompt earlier lifestyle interventions, imaging, or pharmacologic strategies. Furthermore, combining ABI with other cardiovascular biomarkers (eg, brachial-ankle pulse wave velocity, flow-mediated dilation, and left ventricular hypertrophy indices) may further improve risk stratification.

When to Screen ABI in Hypertension

Recent 2024 guidelines advocate targeted ABI screening, not blanket testing. The ACC/AHA multisociety guideline states that a resting ABI is reasonable for screening in patients at increased PAD risk (Class IIa, LOE B-NR), such as those 65 years or older or aged 50 to 64 years with risk factors such as diabetes or hypertension, but not in low-risk, asymptomatic individuals.[42]

Similarly, the 2024 ESVS guideline suggests that ABI screening may be considered for asymptomatic adults 65 years or older, or aged 50 to 64 years with atherosclerotic risk factors such as hypertension or smoking, and for younger individuals with diabetes plus another risk factor.[43]

Directions for Future Research

Future research should focus on standardizing ABI measurement methods and cutoff definitions across populations. Large, prospective studies are needed to determine whether ABI-guided interventions reduce event rates in patients with hypertension. In addition, integrating ABI into established clinical risk models such as the SCORE2 or Framingham Risk Score and testing its incremental predictive value may help clarify its role in routine cardiovascular screening. Finally, more research is needed on ABI in women, younger patients with hypertension, and underrepresented ethnic groups.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work, the authors used ChatGPT-4o (OpenAI) to improve the readability and clarity of the manuscript. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Declarations:

Ethical Approval

Ethical approval was not required for this study because it is a systematic review based on previously published data.

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Conflict of Interest

The authors declare that they have no conflicts of interest relevant to this manuscript.

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