Review Article

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

Hoda Borooghani [1], Kiana Orangi [1], Parmida Bagher Zadi [1], Hoda Asefi [2], Mohsen Arabi [3]*

[1] School of Medicine, Iran University of Medical Sciences, Tehran, Iran.
[2] Department of Radiology, Sina Hospital, Tehran University of Medical Sciences, Tehran, Iran.
[3] Department of Community and Family Medicine, Preventive Medicine and Public Health Research Center, Psychosocial Health Research Institute, School of Medicine, Iran University of Medical Sciences, Tehran, Iran.

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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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*** Corresponding Author:**
Mohsen Arabi
Department of Community and Family Medicine, Preventive Medicine and Public Health Research Center, Psychosocial Health Research Institute, School of Medicine, Iran University of Medical Sciences, Shahid Hemmat Highway, Tehran, Iran. Postal code: 1449614535.
E-mail: drmohsenarabi@gmail.com

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 related to 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 infarcts. Some studies indicated 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 risk 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, and prospective studies should 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

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RHYTHM
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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 (about nearly one billion) had hypertension in 2000, and that this proportion will increase to 29% (1·56 billion) by 2025.[3]

It also suggests that men and women have simi. Despite guideline-directed lifestyle interventions and pharmacologic therapy, a substantial proportion of hypertensive patients remain at elevated cardiovascular risk. studies have shown that clinically significant residual risk persists even among individuals who achieve recommended blood pressure targets. Furthermore, medication contributes to continued vascular events despite availability of effective treatments.[4-6] need for additional, simple, and non-invasive prognostic tools, such as the ankle-brachial index (ABI), to better identify high risk individuals beyond office blood pressure levels and traditional risk scores.[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 used to screen for Peripheral artery disease (PAD), typically defined by an ABI ≤ 0.9,[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 abnormal 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]

However, the prognostic role of ABI specifically in patients with hypertension is less clearly established.[15,16] Hypertensive individuals often have hypertension-mediated organ damage, including vascular remodeling, endothelial dysfunction and microvascular disease, that are not fully captured by routine clinical evaluation.[15,17] ABI might be a convenient marker of such vascular damage and help refine risk stratification in this group, but existing studies in hypertensive populations reported heterogeneous findings, used different cutoffs for ABI, and evaluated diverse outcomes of cardiovascular and cerebrovascular events.[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 (i) summarize the strength and consistency of associations for low and high ABI categories with major clinical endpoints, (ii) describe how ABI was measured and categorized across studies, and (iii) highlight unresolved inconsistencies and areas in which further research is needed.

Methods

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guideline and it was registered by "PROSPERO 2025 CRD420251236723" to find studies that evaluate the association between ABI values and cardiovascular or cerebrovascular outcomes in hypertensive patients. The databases searched were PubMed, Embase, Google Scholar, Scopus, and Web of Science, with the following keywords and Medical Subject Headings (MeSH) in various combinations: "ankle-brachial index," "ABI," "hypertension," "high blood pressure," "cardiovascular outcomes," "cerebrovascular outcomes," "stroke," "myocardial infarction," "mortality," and "peripheral artery disease." The full search strategy included PubMed, Scopus, Web of Science, Embase and Google Scholar. Although all five databases were searched, only PubMed, Scopus and Web of Science yielded records that met the eligibility criteria and therefore appear in the PRISMA flow diagram. Embase and Google Scholar searches returned either duplicates or studies that did not satisfy inclusion criteria. This clarification has been added to ensure consistency between the Methods and PRISMA reporting. The detailed search query is available in the appendix.

Two authors (KO and PB) independently screened all titles and abstracts identified through the database searches and conflicts has been checked by a third person (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.

We included observational studies of any design (prospective or retrospective cohort studies, case-control studies, and cross-sectional analyses) that evaluated the association between ankle–brachial index (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 (e.g., <0.9, normal, >1.4). The 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 text were also excluded. No minimum follow-up duration was required; however, when cohort studies were included, we extracted and reported the length of follow-up. 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 with the aid of a standardized template, then cross-checked for confirmation. The variables extracted were: study characteristics (authors, year, country, design, sample size, population demographics); ABI measurement method and cutoffs definitions; cardiovascular and cerebrovascular events studied; follow-up duration (for prospective studies); whether mortality and PAD outcomes were reported; statistical adjustments for confounders; principal results and risk estimates; and risk of bias or quality scores (if available). Continuous variables were reported as mean ± standard deviation (SD) and categorical variables as number and percentage (n [%]). If there was any conflict or disagreement, they were resolved through discussion or third-party arbitration. The principal domains were selection bias, exposure and outcome assessment, and statistical adjustment for confounding. Because of substantial clinical and methodological heterogeneity across studies including differences in study design, baseline risk, ABI measurement devices and protocols, ABI categories and outcome definitions, we did not perform a formal meta-analysis and instead synthesized the evidence narratively. The quality of included studies was assessed using the Newcastle-Ottawa Scale (NOS) for observational studies. Also, Risk of bias was evaluated using ROBINS-I tool for non-randomized observational studies. The NOS assessed selection, comparability and outcome domains. While ROBINS-I evaluated bias due to confounding, participant selection, exposure classification, deviations from intended interventions, missing data, outcome measurement and reporting. Two reviewers independently scored each study across the domains of selection, comparability, and outcome/exposure. Discrepancies were resolved by consultation with a third reviewer. For more information, see the supplementary tables.

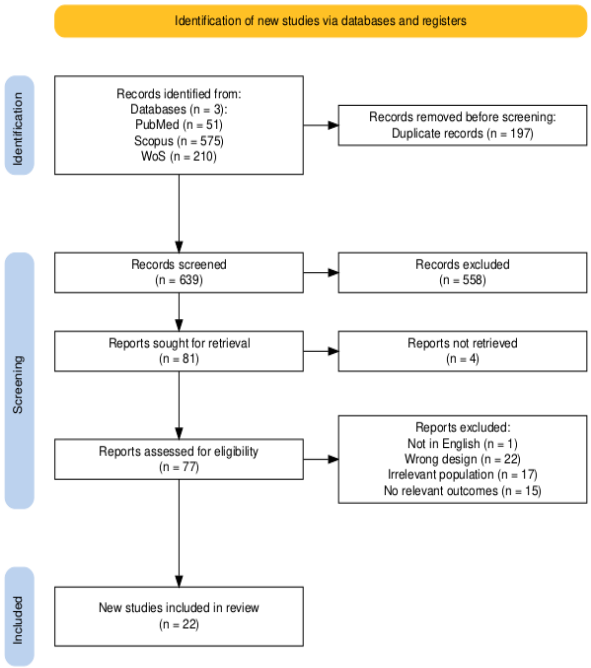


Figure 1. Study flow diagram

Clinical results were delineated as follows: cardiovascular events consist of myocardial infarction (MI), angina, coronary artery disease (CAD), heart failure, and composite cardiovascular events. Cerebrovascular complications include ischemic and hemorrhagic strokes, TIA, and silent brain infarcts. Death included all-cause or cardiovascular-related death, and peripheral events included PAD progression or development, limb event, or lower extremity amputation. Wherever feasible, adjusted relative risks, hazard ratios, or odds ratios were reported to determine the strength of association between ABI categories and outcomes.

Results

Literature search

Using the search strategy, at first 836 articles were found to be useful. After the first screening and deleting the duplicate articles, 81 articles were chosen. Then the full text screen was done and 55 articles were excluded because they didn't meet the criteria. A total of 22 articles were chosen and the needed data were extracted.

Study Characteristics

This review presents evidence from several studies that were conducted in a number of countries, e.g., Spain, Hungary, Poland, Brazil, and Turkey, and others (Table 1). Study designs varied and included prospective cohorts, cross-sectional studies, and case-control studies. The sample sizes all had extensive variations, from 40 subjects to larger cohorts about of 21,000 individuals. The populations were patients with hypertension (HTN), all the subjects in studies suffered from additional cardiovascular risk factors such as chronic kidney disease (CKD), diabetes, or previous cardiovascular events but there were three studies which these risk factors were excluded.[19-21]

Mean age was about 57 to 69 years, with most populations being middle-aged to elderly. Patients were mostly male to a great extent and gender was different in different studies. The way ABI was measured was different; from Doppler ultrasound, oscillometric devices, to mercury manometers. Characteristics of the studies included in the present study is presented in the (Table1 and 2).

ABI was represented as the ratio of systolic pressure at the ankle to the brachial artery. The following cut points were used in most studies in the review (e.g., Armas-Padrón et al.[22] Farkas et al.[16] Jiménez-Balado et al[23]): abnormal low ABI <0.9, indicative of peripheral artery disease; normal ABI 0.91 to 1.4; and abnormally high ABI >1.4, suggesting arterial stiffening or incompressible arteries. Some studies such as Shi et al.[24] and Zhou et al.[25] used different cut points such as <1.05 or ≤1.10 for low ABI, particularly in the presence of population-specific quartiles or with the combination of ABI and other vascular markers such as the cardio-ankle vascular index (CAVI).

Overall study quality was moderate to good. Based on the Newcastle-Ottawa Scale, 14 studies scored between 7 and 9 points and were rated as good quality, while the remaining studies scored 5 to 6 points, indicating moderate quality. 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. However, several studies demonstrated moderate to serious risk of bias due to residual confounding (e.g., incomplete adjustment for comorbidities) and outcome measurement variability (e.g., non-uniform ABI protocols). No study was judged as critical risk of bias. (See table S4 and S5)

In addition to demographic and methodological variability, the 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 population-based studies including over 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] Cerebro-vascular outcomes comprised ischemic stroke, hemorrhagic stroke, transient ischemic attack, and silent brain infarcts.[21,27,29,30] Several studies also assessed hypertension mediated organ damage, including left ventricular hypertrophy, proteinuria, carotid atherosclerosis, and peripheral artery disease progression.[18,19,24,33-36]

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

Association between ABI and cardiovascular outcomes

In the studies that were included, the usual outcome was the link between abnormal ABI and cardiovascular outcomes, and it was much more common with low ABI (≤ 0.9). For example, a Spanish cohort showed that participants with ABI ≤ 0.9 had significantly increased mortality and hospitalizations due to cardiovascular diseases[22] as illustrated in (Table 2). Likewise, a Hungarian study involving over 21,000 individuals demonstrated that low ABI was linked with hazard ratio (HR) of 1.87 (95% CI 1.63 to 2.16) for mortality even with adjusting for some confounding variables.[33]

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	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%	Hypertension (treated and untreated), DM (8.3), dyslipidemia, smoking (12), obesity, family history of CVD, elevated cholesterol ratio, CKD	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%	DM (11.35), stroke (6.49), current smoking (26.33), current drinking (22.67), hypercholesterolemia, hypertriglyceridemia, CKD, hyperuricemia, CHD (5.07)	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%	Stroke (10.6), CAD (9.0), DM (5.0), elevated TG, high LDL-C, low HDL-C, elevated BUN	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%	Carotid atherosclerosis (59.6% of the EH group), hyperlipidemia, elevated TG, elevated serum creatinine	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 (LVH), subclinical atherosclerosis, proteinuria, and ACS.[19,20,26-30,35,38,40] For example Luo et al. 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 versus normal ABI.[26] Morillas et al. reported that low ABI was associated with in-hospital mortality with an odds ratio of 13.0 and with higher rates of heart failure, angina and composite in-hospital complications.[40] Interestingly, in some studies ABI is used as a continuous variable, suggesting a linear trend for poorer cardiovascular outcomes with decreasing ABI.[22,33,34,40]

In a study CHD prevalence was inversely associated with ABI for lower values and it highlights ABI's potential as a screening tool.[25] Also, the relation between protective role of ABI and harmful roles of GGT and Hcy in carotid atherosclerosis has been studied.[36]

The Brazilian investigation was conducted only on male hypertensive patients and showed an outstanding difference in the prevalence of LVH in those who had normal (4%) versus abnormal ABI (35.3%).[29] Similarly, the Polish case-control study of first-episode ACS patients identified low ABI as the most predictive parameter for the development of ACS even in the absence of diagnosed PAD.[27]

Others provided broader definitions of PAD, and results were often grouped by comorbidities like CKD and diabetes.[32,38,39] Gender analysis had less emphasis (but gender adjustment in multivariable analyses was important). Overall, evidence is strongly in favor of the prognostic importance of low ABI in hypertensives regardless of geographic or clinical setting.[21,31,34,39]

Association of ABI With Cerebrovascular and Brain Outcomes

Some studies discussed the effect of abnormal ABI on cerebrovascular accidents. The study in Spain showed that ABI ≤ 0.9 is strongly connected with higher rates of stroke, cardiovascular mortality, and risk of hospitalization in a 2 year follow up.[22] Also, in a Chinese hypertensive cohort, ABI <1.05 was associated with higher stroke prevalence compared with ABI ≥1.05 (odds ratio 1.26; 95% confidence interval [CI] 1.05–1.50), particularly among participants aged ≥65 years.[24] Some studies showed low ABI and silent brain infarcts predicted higher risk of future cardiovascular events and screening these in hypertensive patients could improve early intervention.[23,28,34,40]

PAD and Hypertension-Mediated Organ Damage

Several studies examined the association between ABI and markers of hypertension-mediated organ damage: Albuquerque reported that the prevalence of left ventricular hypertrophy was 35.3% among patients with abnormal ABI compared with 4% among those with normal ABI.[29] Veysel Tosun found that lower ABI values were observed in patients with proteinuria and/or left ventricular hypertrophy, with an odds ratio of 0.01 (95% CI 0.001–0.010) for ABI in relation to the combined presence of these complications.[19] In a rural Chinese cohort, J. Sun showed that higher-normal ABI values were associated with left ventricular hypertrophy: compared with the lowest ABI quartile, the odds ratios for left ventricular hypertrophy were 1.61 (95% CI 1.32–1.96) and 1.83 (95% CI 1.50–2.24) in the third and fourth ABI quartiles, respectively. M. Peng reported an inverse association between ABI and carotid atherosclerosis severity, whereas γ-glutamyltransferase and homocysteine were positively correlated with plaque presence.[36]

Impact of Methodologic Differences

A further source of heterogeneity across studies relates to methodological 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 <0.9 and >1.4[22,23, 26, 33, 34, 40] others used alternative cut-offs such as <1.05,

≤1.07, ≤1.10, or quartile-based thresholds.[24,25,28,35] This combination of methodological 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 in 2024 on 600 hypertensive patients aged 18-75 with suspected coronary atherosclerotic heart disease, the cutoff of the high abnormal ABI was those above 1.09 and those patients were significantly at higher risk of CAS severity. Combining ABI, FMD and baPWV improved predictive accuracy (AUC=0.800).[37]

Discussion

Summary of Main Findings

This systematic review shows that the ankle-brachial index (ABI) is a very important predictor of health in people with high blood pressure. Both abnormally low (≤ 0.9) and high (>1.4) ABI values were linked to higher risks of cardiovascular and cerebrovascular disease in all of the studies that were included. These links were often not related to traditional risk factors, which is important because it suggests that ABI may show aspects of vascular damage that routine clinical markers don't fully show. Also, a number of studies looked into changing ABI thresholds, especially for older and Asian people, which could help find risks earlier.

Interpretation and Pathophysiologic Implications

Low ABI values are a sign of peripheral arterial disease and, more generally, systemic atherosclerosis. There were consistent links between low ABI and serious heart problems like heart attack, heart failure, and death from heart disease in general.[23,24,26-28,33,38,40] These links stayed strong even after taking into account other factors like blood pressure, lipid levels, and diabetes. This shows that ABI can show the total amount of stress on the blood vessels.[22,23,26,33] For example, Farkas et al. found that patients with low ABI were 1.87 times more likely to die after five years of follow-up.[33]

On the other hand, high ABI values were usually seen as a sign that the arteries were getting stiffer, probably because of calcification in the middle. Several studies found a J- or U-shaped risk curve, but these results were less common. They also found that the higher end of the ABI distribution had bad outcomes[25,28,33,35] As seen in Sun et al. and Zhou et al.[25,35] high ABI was linked to left ventricular hypertrophy and coronary artery calcification in some cases. However, the fact that definitions and methods are not always the same makes it hard to generalize.

Cardiovascular Event-Specific Differences

Low ABI was generally linked to cardiovascular morbidity, but the types of these outcomes were different. Some studies looked only at ACS or heart attacks. For instance, Myslinski et al. showed that low ABI was the best sign of first ACS episodes, even when there was no previous peripheral artery disease.[27] Some researchers, like de Albuquerque et al. and Sun et al., looked at changes in the structure of the heart and found a strong link between low ABI and left ventricular hypertrophy.[29,35]

On the other hand, large-scale studies also looked at broader composite outcomes, like all-cause mortality or hospitalization due to cardiovascular causes. Farkas et al.'s population-level cohort and Morillas et al.'s ACS registry both showed that ABI is a strong, independent predictor of bad cardiovascular outcomes in people with high blood pressure. The follow-up periods ranged from days (acute phase) to five years.[33,40]

Cerebrovascular Risks and ABI

The link between ABI and cerebrovascular events came up again and again. Shi et al. and Jiménez-Balado et al. found that low ABI was strongly linked to a higher risk of ischemic stroke and silent brain infarcts, even in people with high blood pressure who had never had a stroke.[23,24] The strength of these links stayed the same in multivariable models, and they were especially strong in older patients. Kario et al. also found that ABI values ≤1.10, especially when combined with other

vascular biomarkers like CAVI, were linked to a higher risk of hemorrhagic stroke and other brain problems over a five-year period.[28] This means that ABI might be a good way to tell if someone has cerebral small vessel disease if they have high blood pressure.

Comparison ABI With Other Vascular Risk Markers

Another important factor to evaluate is ABI's performance in comparison to other well-established markers of vascular risk in hypertension. Several research examined ABI in conjunction with indications such as left ventricular hypertrophy, carotid atherosclerosis, proteinuria, and arterial stiffness indices. For example, de Albuquerque et al. and Sun et al. revealed that ABI provided additional predictive information beyond LVH[29, 35], whereas Peng et al. found that ABI performed in conjunction with carotid plaque burden in indicate atherosclerosis risk.[36] Tosun et al. discovered that ABI was linked to proteinuria and LVH[19], confirming its significance as a comprehensive measure of hypertension-induced organ damage. Furthermore, Kario et al. found that combining ABI with vascular stiffness markers such CAVI enhanced prediction of cardiovascular outcomes compared to using either marker alone.[28] Taken together, our findings indicate that ABI has additive predictive value and may improve risk stratification in hypertensive patients when employed in conjunction with recognized cardiovascular risk markers.

Strengths and Limitations

Even though the results are the same in different places and types of patients, the fact that the studies are all different makes them hard to understand. There were a lot of different ways to measure ABI, from Doppler ultrasound to oscillometric devices, and the positions (lying down vs. sitting) weren't always the same. Also, different studies used different definitions of "normal" and "abnormal" ABI thresholds. For example, some studies used <1.05 or ≤1.10 as more sensitive cutoffs, especially in Asian groups.[20, 25, 28]

Also, a lot of studies didn't fully adjust for important factors like how long someone had high blood pressure, whether they were taking medication, or their socioeconomic status. There were also not enough reports on age- and sex-specific analyses, which made it harder to understand which groups of people might benefit the most from ABI screening. Still, a number of large, well-run groups, like those by Farkas, Shi, and Kario, support the main conclusions. Because of substantial clinical and methodological heterogeneity across studies, a quantitative meta-analysis was deemed inappropriate, and we relied on a narrative synthesis, which may limit the precision with which overall effect sizes can be estimated.

Clinical Implications

ABI has a lot of potential to be used regularly in the care of people with high blood pressure because it is simple, cheap, and doesn't hurt. It might help find patients who are still at risk even though their blood pressure is under control, which could lead to earlier lifestyle changes, imaging, or medication strategies. Also, using ABI with other heart or vascular biomarkers (like baPWV, FMD, and LVH indices) may help even more with risk stratification.

When to Screen ABI in Hypertension

Recent 2024 guidelines advocate targeted ABI screening, not blanket testing. The ACC/AHA Multi society 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 aged 50 to 64 with risk factors like diabetes or hypertension-but not in low-risk, asymptomatic individuals.[41]

Similarly, the 2024 ESVS Guideline suggests ABI screening may be considered for asymptomatic adults who are ≥ 65, or aged 50 to 64 with atherosclerotic risk factors like hypertension or smoking, and for younger individuals with diabetes plus another risk factor.[42]

Directions for Future Research

Future research should focus on making ABI measurement methods and cutoff definitions the same for all groups of people. We need large, long-term studies to find out if ABI-guided treatments lower the number of events in people with high blood pressure. Also, adding ABI to clinical risk models like the SCORE2 or Framingham Risk Score and testing its extra

predictive power may help to make its role in routine cardiovascular screening clearer. Finally, it is still important to do research on ABI in women, younger people with high blood pressure, and ethnic groups that are not well represented.

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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