ORIGINAL ARTICLE

Handgrip Strength Predicts Cardiovascular Disease Prevalence and Mortality Among Hypertension Patients: Results from National Health and Nutrition Examination Survey (NHANES) 2011–2014

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ABSTRACT

Background: Handgrip strength (HGS) has emerged as a potential biomarker for cardiovascular risk, yet its predictive value in hypertensive populations remains unclear. This study aimed to investigate the association between HGS and cardiovascular disease (CVD) prevalence and mortality among hypertensive adults. **Methodology:** This cross-sectional study included 3,431 hypertensive participants from the National Health and Nutrition Examination Survey (NHANES) 2011–2014 cycles, a nationally representative survey in the U.S., linked to mortality data through 2018. HGS was assessed using standardized grip strength protocols and categorized into quintiles. Logistic regression and Cox proportional hazards models were used to examine associations between HGS and CVD prevalence, all-cause mortality, and CVD-specific mortality, adjusting for demographic and clinical confounders. A two-sided p-value < 0.05 was considered statistically significant. **Results:** Individuals in the lowest HGS quintile (Q1: HGS <26.1 kg) had a 38% higher CVD prevalence (OR: 1.38, 95% CI: 1.28–1.52, p < 0.001), a 77% increased risk of all-cause mortality (HR: 1.77, 95% CI: 1.26–2.33, p < 0.001), and a 57% higher risk of CVD mortality (HR: 1.57, 95% CI: 1.10–2.21, p = 0.02) compared to those in the highest quintile (Q5: HGS >47.1 kg), even after adjusting for demographic and clinical factors. **Conclusion:** These findings suggest that HGS could be incorporated into cardiovascular risk stratification models to improve early detection strategies for hypertensive individuals. Unlike prior studies that focused on general or elderly populations, our study uniquely evaluates HGS as a predictor of CVD outcomes in hypertensive individuals, an understudied high-risk group.
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INTRODUCTION

Hypertension is a global health concern that affects a significant proportion of adults worldwide, with its prevalence continuing to rise, particularly in low- and middle-income countries. The World Health Organization (WHO) estimates that as of 2023, approximately 1.4 billion individuals are living with hypertension, yet 46% remain undiagnosed (1). This silent yet progressive condition significantly elevates the risk of cardiovascular diseases (CVDs), leading to increased morbidity and mortality rates. Given its substantial impact, early detection and effective management strategies are essential to mitigate the associated health burden. Over three-quarters of hypertensive individuals live in low- and middle-income countries, and nearly 50% of individuals remain unaware of their condition (2). Hypertension is recognized as a modifiable risk factor for cardiovascular diseases, including stroke, myocardial infarction, and heart failure. To address this, the WHO Global Hearts Initiative emphasizes early intervention through lifestyle modifications, regular screenings, and adherence to pharmacological treatments (1). Despite these preventive efforts, CVD remains the leading cause of death globally, reinforcing the need for additional,

non-invasive risk assessment tools that can complement existing diagnostic methods.

Cardiovascular events and premature mortality can be mitigated by enhanced cardiorespiratory fitness and muscle strength (3). Muscle strength decline, often linked to aging, greatly impacts physical function and elevates the risk of disability and mortality (3). Recent research suggests that handgrip strength (HGS), a simple yet effective indicator of muscle function, may serve as a valuable predictor of adverse health outcomes. HGS has emerged as a potential biomarker of muscular integrity and cardiovascular health, reflecting neuromuscular function, metabolic efficiency, and systemic inflammation, all of which contribute to cardiovascular risk (3). Studies have demonstrated that reduced HGS correlates with higher levels of inflammatory markers (e.g., interleukin-6, C-reactive protein) and endothelial dysfunction, mechanisms that accelerate atherosclerosis and vascular complications (4). Large-scale cohort studies, such as the National Health and Nutrition Examination Survey (NHANES) cohort study, have reported an inverse association between HGS and cardiovascular mortality, suggesting its potential as a cost-effective screening tool for early cardiovascular risk detection (5). Similarly, a recent meta-analysis confirmed that lower HGS is significantly associated with increased mortality in coronary artery disease patients (6).

The biological mechanisms linking HGS decline to CVD mortality are rooted in chronic inflammation and vascular dysfunction. For example, Dhamoon et al. (7) pointed out that muscle attenuation and slowed gait are linked to increased inflammatory markers, such as interleukin-6 and lipoprotein phospholipase A2, as observed in Framingham's offspring studies, contributing to a pro-inflammatory environment. Similarly, Calila et al. (8) indicated that muscle attenuation correlates with a heightened risk of endothelial dysfunction. The Prospective Urban-Rural Epidemiological (PURE) study also found an inverse relationship between HGS and the risk of all-cause and CVD mortality (9). The results from the Survey of Health, Ageing and Retirement in Europe (SHARE) study similarly indicated that low grip strength predicts all-cause and CVD mortality risk (10). In other population-based cohorts, particularly among older adults, a reduction in HGS is linked to early mortality and a higher incidence of cardiovascular events (11, 12). While the association between HGS and CVD has been established in general populations, its predictive value specifically in hypertensive individuals, i.e., those who are at inherently higher cardiovascular risk, remains underexamined. Most research has focused on the general population or elderly individuals, leaving a gap in understanding the predictive value of HGS among hypertensive patients. To address this gap, this study aims to determine whether HGS serves as an independent predictor of CVD prevalence and all-cause mortality in hypertensive individuals. Specifically, we investigated whether reduced HGS is associated with a higher risk of adverse cardiovascular outcomes in this population, independent of other traditional risk factors. Unlike prior investigations that primarily utilized cross-sectional analyses, our study employs longitudinal survival analysis (Cox regression models) to assess the long-term prognostic significance of HGS beyond traditional risk factors. By establishing HGS as a reliable non-invasive marker for cardiovascular risk stratification, this study has the potential to enhance clinical hypertension management and support early intervention strategies.

METHODS

Data sourced from NHANES dataset, which is publicly accessible through the CDC official website (https://wwwn.cdc.gov/nchs/nhanes/Default.aspx), were utilized in this study. This dataset, representing the U.S. population, aimed at evaluating the health and nutritional profiles of American adults and children. As this research relied exclusively on this publicly available data, no additional ethical approval is required (see the Ethics Review Board (ERB) of the NCHS statement at https://www.cdc.gov/nchs/nhanes/irba98.htm).

Materials

Muscle strength was assessed using the NHANES Muscle Strength-Grip Test, following standardized protocols (14). HGS was measured using a Takei Digital Grip Strength Dynamometer (TKK 5401), with three trials performed on each hand, and the maximum value recorded for analysis (15). Data were extracted from the 2011–2012 and 2013–2014 NHANES cycles, as these were the only cycles with available HGS measurements. The study population was initially drawn from 19,931 participants, with inclusion criteria set as adults aged 18–80 years diagnosed with hypertension, defined as systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, based on the International Society of Hypertension guidelines (16). Participants were excluded if they had missing HGS data from both hands, prior hand surgery, hand-related conditions (e.g., stiffness or discomfort), pregnancy, or metabolic and endocrine disorders affecting muscle strength, including thyroid disease, vitamin D deficiency, chronic kidney disease (CKD), and chronic obstructive pulmonary disease (COPD). However, individuals with a prior history of major cardiovascular events (e.g., myocardial infarction, stroke) were not excluded, as the study aimed to assess the predictive value of HGS in hypertensive individuals, including those with pre-existing CVD. While the inclusion of a normotensive control group could offer additional insights, prior studies have already established the association between HGS and overall mortality in the general population. Therefore, as this study focused specifically on a clinically relevant subgroup, i.e., hypertensive individuals, normotensive

individuals were excluded regardless of whether they had pre-existing CVD. With these inclusion and exclusion criteria, 3,431 hypertensive participants were eventually included in the final analysis (see Figure 1 for the study selection flowchart).

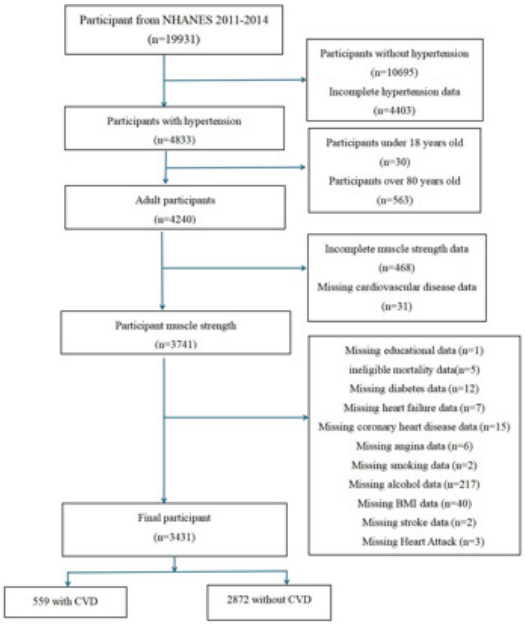

Figure 1: The flowchart of study sample selection form NHANES 2011-2012 and 2013-2014

Outcome Measures

This study examined three key outcomes, i.e., (1) CVD prevalence, (2) all-cause mortality, and (3) CVD-specific mortality. CVD prevalence was determined based on participants' self-reported physician diagnoses of CVDs such as coronary heart disease (CHD), heart failure, stroke, and cerebrovascular accidents (CVAs).

For CVDs and mortality, data were sourced from the 2019 NHANES Public-Use Linked Mortality Files (available at https://www.cdc.gov/nchs/data-linkage/mortality-public.htm). In this dataset, mortality outcomes were classified following the 10th Revision of the International Classification of Diseases (ICD-10). Similarly, the specific ICD-10 codes were used to identify CVD types, including congestive heart failure, stroke, and CVA. Participants were followed from their initial examination date to the last recorded date in 2018, indicating whether they were alive or deceased.

Covariates and Potential Confounders

To account for potential confounding effects, multiple covariates were adjusted in the statistical models. Demographic variables included age, sex, ethnicity, and education level, while clinical factors comprised BMI, diabetes status, and hypertension severity. Lifestyle factors, such as smoking status and alcohol use, were included due to their known influence on muscle strength and cardiovascular health. Additionally, metabolic variables like impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) were also analyzed, as they are associated with muscle function and cardiometabolic risk. Similarly, where available, inflammatory markers (e.g., C-reactive protein (CRP) and interleukin-6) were examined as well, given their role in systemic inflammation and cardiovascular disease progression. These covariates were chosen based on previous literature linking them to HGS and cardiovascular outcomes.

DATA ANALYSIS

Statistical analyses were conducted using R software (v4.3.0), following NHANES complex survey weighting procedures to generate nationally representative estimates, with all statistical tests being two-sided and significance set at $P < 0.05$. To examine the association between HGS and CVD prevalence, weighted multivariate logistic regression models were applied to compute odds ratios (ORs) and 95% confidence intervals (CIs) while adjusting for confounders. Three sequential models were constructed: Model 1 (unadjusted), Model 2 (adjusted for age, sex, race/ethnicity, and education level), and Model 3 (fully adjusted for BMI, smoking status, alcohol use, diabetes, IFG, and impaired glucose tolerance (IGT)). Additionally, HGS quintiles were analyzed to assess a dose-response relationship, comparing individuals in Q1 (lowest HGS) to Q5 (highest HGS). To evaluate HGS's impact on all-cause and CVD-specific mortality, Cox proportional hazards models were employed to estimate hazard ratios (HRs) and 95% CIs, accounting for time-to-event data and censoring, with participants followed from their NHANES examination date until 2018, and mortality data sourced from the National Death Index (NDI). The Cox model was chosen as it accounts for censored data and varying follow-up durations, offering a more accurate estimation of time-dependent risk factors. Sensitivity analyses included stratifications by age groups, sex, and hypertension severity, along with propensity score matching (PSM) and inverse probability weighting (IPW) to mitigate confounding bias. Additionally, likelihood ratio tests and Schoenfeld residuals were used to assess the proportional hazards assumption, while restricted cubic spline analysis was conducted to explore potential nonlinear associations between HGS and mortality risk. These statistical approaches ensured a comprehensive and reproducible analysis, adjusting for key demographic, clinical, and metabolic confounders to strengthen the validity of our findings.

RESULTS

Characteristics of the Study Population

Among the 3,431 hypertensive participants, 559 (16.3%) had one or more CVD, while 2,872 (83.7%) did not have a history of CVD. Participants with CVD were

significantly older (62.65 vs. 53.54 years, p < 0.0001) and exhibited lower HGS (34.68 kg vs. 38.03 kg, p < 0.001) compared to those without CVD. Although BMI was slightly higher in the CVD group (31.89 vs. 31.21, p = 0.1), the difference was not statistically significant. Gender distribution did not differ significantly (p = 0.05), while notable differences were observed in education level, diabetes prevalence, IFG, smoking status, and alcohol consumption (p < 0.0001 for all), with higher proportions of these risk factors in the CVD group. Moreover, the prevalence of stroke, coronary artery disease (CAD), angina, heart failure, and myocardial infarction was significantly higher among those with CVD. These findings highlight distinct demographic and health-related disparities between hypertensive individuals with and without CVD, emphasizing the need for comprehensive risk assessment strategies (Table I).

Table I: General characteristics of the population in NHANESE 2011–2014

Variable	CVD (mean)	Non-CVD (mean)	p-value
Age (years old)	62.65 (0.63)	53.54 (0.41)	< 0.0001
BMI	31.89 (0.45)	31.21 (0.21)	0.1
HGS	34.68 (0.67)	38.03 (0.25)	< 0.001
Gender			0.05
Female	256 (44.31)	1442 (49.66)	
Male	303 (55.69)	1430 (50.34)	
Ethnicity			0.64
Black	179 (14.98)	891 (14.13)	
Mexican	45 (5.03)	288 (6.02)	
Other	96 (11.56)	580 (10.56)	
White	239 (68.42)	1113 (69.29)	
Education			0.001
9-11th grade (Includes 12th grade with no diploma)	109 (14.66)	430 (11.29)	
College graduate or above	81 (18.93)	627 (27.96)	
High school graduate/GED or equivalent	141 (25.96)	675 (21.94)	
Less than 9th grade	63 (6.97)	228 (4.22)	
Some college or AA degree	165 (33.48)	912 (34.59)	
Diabetes			< 0.0001
Yes	272 (43.69)	770 (21.96)	
IFG	26 (6.74)	140 (5.54)	
IGT	23 (3.77)	149 (5.02)	
no	238 (45.81)	1813 (67.47)	
Smoking status			< 0.0001
Ex-smoker	207 (39.82)	779 (29.45)	
Non-smoker	212 (34.31)	1505 (51.04)	
Active smoker	140 (25.87)	588 (19.52)	
Alcohol drinking			< 0.0001
Ex-drinker	179(30.69)	544 (15.15)	
Heavy drinker	67(12.17)	514 (19.97)	
Mild drinker	179(34.82)	962 (36.53)	
Moderate drinker	57(11.68)	436 (17.66)	
Non-drinker	77(10.64)	416 (10.68)	
Stroke			< 0.0001
no	355(66.50)	2872 (100.00)	
yes	204(33.50)	0 (0.00)	

CONTINUE

Table I: General characteristics of the population in NHANESE 2011–2014. (CONT.)

Variable	CVD (mean)	Non-CVD (mean)	p-value
Coronary heart disease			< 0.0001
No	359(61.38)	2872 (100.00)	
Yes	200(38.62)	0 (0.00)	
Angina			< 0.0001
No	429 (73.59)	2872 (100.00)	
Yes	130 (26.41)	0 (0.00)	
Congestive Heart Failure			< 0.0001
No	382 (68.47)	2872 (100.00)	
Yes	177 (31.53)	0 (0.00)	
Heart attack			< 0.0001
No	334 (58.88)	2872 (100.00)	
Yes	225 (41.12)	0 (0.00)	

Link between HGS and CVD prevalence among hypertensive individuals

The association between HGS and CVD prevalence was assessed using three logistic regression models, all of which revealed a significant inverse relationship between HGS and CVD risk. In Model 1 (unadjusted), each 1 kg increase in HGS was associated with a 3% lower risk of CVD (OR: 0.97, 95% CI: 0.96–0.99, p < 0.001). This protective effect remained in Model 2 (adjusted for age, sex, race, and education), with an OR of 0.96 (95% CI: 0.94–0.98, p < 0.001). The association persisted even after full adjustment for BMI, smoking, alcohol consumption, and diabetes in Model 3 (OR: 0.96, 95% CI: 0.95–0.98, p = 0.002). When categorized into quintiles, participants in the lowest HGS quintile (Q1: HGS <26.1 kg) had the highest CVD prevalence, whereas those in the highest quintile (Q5: HGS >47.1 kg) had the lowest prevalence. This trend remained significant across all models, with the most adjusted model (Model 3) demonstrating a significant negative beta coefficient for HGS in relation to CVD. Trend analysis (p for trend < 0.001) confirmed a progressive decline in CVD prevalence with increasing HGS quintiles, reinforcing HGS as a potential independent predictor of CVD risk (Table II & Table III).

Table II: The association between HGS and the prevalence of cardiovascular disease (CVD) in hypertensive patients

	OR	95% CI	p-value
Model 1	0.97	0.96 - 0.99	<0.001
Model 2	0.96	0.94 - 0.98	<0.001
Model 3	0.96	0.95 - 0.98	0.002

Table III: Association between HGS (quintile) and CVD prevalence

HGS	Model 1		Model 2		Model 3	
	B(95%CI)	p-value	B(95%CI)	p-value	B(95%CI)	p-value
Q1	ref		ref		ref	
Q2	-0.87(0.26,0.69)	0.001	-0.74(0.30,0.76)	0.004	-0.81(0.27,0.73)	0.005
Q3	-0.67(0.35,0.75)	0.001	-0.9(0.27,0.62)	0.001	-0.97(0.24,0.61)	0.001
Q4	-0.71(0.33,0.74)	0.001	-1.34(0.16,0.43)	<0.0001	-1.41(0.14,0.43)	<0.001
Q5	-0.98(0.24,0.59)	<0.001	-1.34(0.16,0.43)	<0.0001	-1.31(0.15,0.48)	<0.001
p for trend	<0.001		<0.001		<0.001	

Establish Cox proportional hazard model

The relationship between HGS and both all-cause and CVD-specific mortality was examined using Cox proportional hazards models, revealing a significant dose-response association, where higher HGS quintiles were linked to lower mortality risk (Table IV). In Model 1 (unadjusted), individuals in the highest HGS quintile (Q5: >47.1 kg) had a 77% lower risk of all-cause mortality (HR: 0.23, 95% CI: 0.16–0.33, p < 0.0001) compared to those in the lowest quintile (Q1: <26.1 kg), and this association persisted in Model 3 (fully adjusted for demographic, lifestyle, and clinical factors), where the mortality risk was reduced by 86% (HR: 0.14, 95% CI: 0.07–0.28, p < 0.0001). A similar pattern was observed for CVD-specific mortality, where the highest quintile (Q5) exhibited a significantly lower risk than the lowest quintile (Q1), with the fully adjusted model (Model 3) showing an 86% reduction in CVD mortality risk (HR: 0.14, 95% CI: 0.05–0.29, p < 0.0001). The trend analysis (p for trend < 0.001) further confirmed a significant inverse relationship between HGS and mortality risk across all quintiles, reinforcing HGS as an independent predictor of both all-cause and CVD-specific mortality among hypertensive individuals.

Table IV: Mortality Outcomes by Handgrip Strength (Quintiles) Among Participants During the Follow-up Period

	The quintile of HGS					
	Q1(6.1-26.1)	Q2(26.1-31.8)	Q3(31.8-39)	Q4(39-47.1)	Q5(47.1,81.5)	P for trend
All-Cause Mortality						
Number of deaths	123	79	82	53	30	
Model 1 h(95% CI)P Value	1	0.56(0.40,0.79) <0.001	0.69(0.46,1.03) 0.07	0.50(0.32,0.78) 0.002	0.23(0.16,0.33) <0.0001	<0.0001
Model 2 h(95% CI)P Value	1	0.60(0.41,0.87) 0.01	0.43(0.23,0.79) 0.01	0.21(0.12,0.38) <0.0001	0.12(0.06,0.24) <0.0001	<0.0001
Model 3 h(95% CI)P Value	1	0.60(0.41,0.87) 0.01	0.45(0.25,0.83) 0.01	0.21(0.12,0.35) <0.0001	0.14(0.07,0.28) <0.0001	<0.0001
CVD Mortality						
Number of deaths	44	27	17	12	13	
Model 1 h(95% CI)P Value	1	0.51(0.28,0.91) 0.02	0.47(0.28,0.79) 0.004	0.35(0.15,0.84) 0.02	0.26(0.12,0.60) 0.002	0.004
Model 2 h(95% CI)P Value	1	0.55(0.34,0.91) 0.02	0.25(0.12,0.50) <0.0001	0.12(0.05,0.28) <0.0001	0.12(0.04,0.33) <0.0001	<0.0001
Model 3 h(95% CI)P Value	1	0.57(0.33,0.98) 0.04	0.26(0.13,0.51) <0.0001	0.12(0.05,0.29) <0.0001	0.14(0.05,0.40) <0.001	<0.0001

Note: Model 1 was unadjusted;
Model 2 was adjusted for age, sex, race, and education level;
Model 3 was further adjusted for BMI, smoking and drinking status, and diabetes.

DISCUSSION

This study analyzed NHANES 2011–2014 data and confirmed that HGS, in conjunction with traditional cardiovascular risk factors, contributes to CVD risk stratification and mortality prediction in hypertensive individuals. Lower HGS was consistently linked to higher CVD prevalence and an increased risk of all-cause and CVD-specific mortality, even after adjusting for demographic and clinical confounders. Individuals with the lowest HGS had substantially higher prevalence of CVD and increased risks of both all-cause and cardiovascular-specific mortality compared to those with the highest strength. The presence of a significant dose-response trend further reinforces the potential of HGS as a clinical predictor of cardiovascular risk.

While this study establishes HGS as an independent predictor of CVD prevalence and mortality among hypertensive patients, its clinical utility is likely enhanced when combined with traditional cardiovascular risk factors such as blood pressure, BMI, and diabetes. Existing CVD risk models primarily rely on these markers for risk assessment, yet they may not fully capture functional capacity and muscular health, which are increasingly recognized as indicators of cardiovascular resilience. Since muscle strength decline is linked to metabolic dysfunction, systemic inflammation, and endothelial impairment, HGS could serve as an indirect indicator of overall cardiovascular and metabolic health (16). Notably, previous studies have demonstrated that low HGS is associated with increased insulin resistance, elevated inflammatory markers (e.g., CRP, interleukin-6), and higher arterial stiffness, which in turn contribute to CVD risk (17). Therefore, incorporating HGS alongside blood pressure, BMI, and diabetes status may provide a more holistic assessment of cardiovascular vulnerability in hypertensive patients.

These findings align with prior studies demonstrating that HGS adds predictive value beyond established cardiovascular risk factors such as blood pressure, BMI, and diabetes in hypertensive populations. For example, Li et al. (18) suggested that gut microbiota diversity is linked to muscle strength, highlighting potential metabolic and inflammatory pathways underlying this association. Zhang et al. (19) similarly found that lower HGS correlated with increased cardiovascular mortality, supporting its role as an early risk indicator. Our study extends this by confirming that HGS serves as a

potential risk modifier when combined with traditional cardiovascular risk markers, potentially refining current risk stratification models.

However, some conflicting evidence exists. Park et al. (20) reported that HGS's predictive power for CVD was reduced when adjusting for body composition and inflammatory markers, suggesting that its effects may be mediated by broader metabolic factors. Additionally, certain studies indicate that HGS is a stronger predictor of all-cause mortality rather than CVD-specific mortality, particularly in populations where muscle loss is primarily driven by sarcopenia or malnutrition rather than cardiovascular pathology. For instance, Liu et al. (21) found that while HGS was associated with both outcomes, its predictive value was stronger for all-cause mortality. Accordingly, these discrepancies may stem from population differences, methodological inconsistencies, and variations in confounder adjustments.

Given its probable strong association between HGS and cardiovascular outcomes, its integration into existing risk assessment frameworks such as the Framingham Risk Score or the SCORE model could refine predictive accuracy. Future studies should explore whether adding HGS to these models enhances their discrimination power for high-risk hypertensive individuals. Moreover, further validation in prospective cohort studies is needed to determine whether HGS-guided risk stratification can improve clinical decision-making and cardiovascular event prevention.

The clinical implication of this study is that the strong inverse association between HGS and mortality risk suggests that it could serve as a complementary risk stratification tool for hypertensive individuals (22). However, it should not yet be considered a primary screening method, as it is currently absent from most clinical guidelines. For example, the current hypertension management guidelines, including those from the American Heart Association (AHA 2020) and the European Society of Cardiology (ESC 2023), do not yet incorporate HGS as a standard cardiovascular risk assessment tool. However, accumulating evidence (23,24) suggests that reduced HGS is strongly associated with increased CVD risk and mortality. Our findings further reinforce this association, highlighting the potential role of HGS as an additional screening measure for hypertensive patients. Given its ease of measurement, HGS could be incorporated into routine hypertension assessments as a supplementary biomarker, particularly for patients without access to advanced cardiovascular testing. Integrating HGS into routine cardiovascular assessments could also enhance risk stratification, particularly for individuals who do not exhibit overt clinical symptoms but remain at high risk for adverse outcomes. Future research should focus on validating HGS-based risk models in large-scale prospective cohorts to determine its clinical applicability in hypertension management.

Despite its strengths, this study has certain limitations. First, the cross-sectional nature of the analysis prevents causal inferences regarding the relationship between HGS and cardiovascular outcomes. Second, while adjustments were made for multiple confounders, residual confounding from unmeasured variables (e.g., dietary intake, other unmeasured inflammatory markers) cannot be ruled out. Third, selection bias may exist as participants with missing data on key variables (e.g., HGS, covariates) were excluded from analysis. This could impact the generalizability of findings, particularly if individuals with missing data had systematically different health profiles. Future studies should explore imputation techniques or alternative analytical methods to minimize selection bias.

CONCLUSIONS

This study highlights the value of measuring HGS in the clinical assessment of hypertensive patients, underscoring its potential role in CVD risk stratification and mortality prediction. Our findings indicate that individuals with the lowest HGS had significantly higher CVD prevalence and increased risk of all-cause mortality compared to those with the highest strength, even after accounting for demographic and clinical characteristics.. These results reinforce the importance of integrating muscle strength assessments into routine cardiovascular risk evaluations, particularly in hypertensive individuals.

Given the strong inverse association between HGS and CVD mortality, incorporating muscle strength assessments alongside traditional cardiovascular markers may enhance early detection of high-risk individuals. Moreover, the observed associations support the clinical utility of strength-based interventions, which may provide an additional strategy for improving cardiovascular health outcomes in hypertensive populations. By recognizing the significance of muscle strength as a component of cardiovascular health, clinicians can implement more comprehensive prevention strategies, incorporating HGS assessments into routine risk screening and intervention planning to optimize long-term cardiovascular outcomes in hypertensive patients.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Data supporting this study's findings are available from the National Health and Nutrition Examination Survey (NHANES), accessible via the Centers for Disease Control and Prevention (CDC) at https://www.cdc.gov/nchs/nhanes/. According to the statements given, NHANES study protocols were approved by the National Center for Health Statistics (NCHS) Research Ethics Review Board (Protocol #2011-17). All participants had provided written informed consent and they were thoroughly informed about the study's objectives and procedures,

were assured that their participation was voluntary, and that they could withdraw at any time without any consequences. As this is a publicly available data, no additional ethical approval is required (see the Ethics Review Board (ERB) of the NCHS statement at https://www.cdc.gov/nchs/nhanes/irba98.htm).

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REFERENCES

1. World Health Organization. Hypertension fact sheet [Internet]. Geneva: World Health Organization; 2023 [cited 2025 May 19]. Available from: https://www.who.int/news-room/fact-sheets/detail/hypertension.
2. Schutte AE, Srinivasapura Venkateshmurthy N, Mohan S, Prabhakaran D. Hypertension in low-and middle-income countries. Circulation research. 2021;128(7):808-26. https://doi.org/10.1161/CIRCRESAHA.120.318729
3. World Health Organization. First WHO report details devastating impact of hypertension and ways to stop it [Internet]. Geneva: World Health Organization; 2023 [cited 2025 May 19]. Available from: https://www.who.int/thailand/news/detail/19-09-2023-first-who-report-details-devastating-impact-of-hypertension-and-ways-to-stop-it.
4. Qin Z, Guo Y, Yang W, Kuang Z, Li G. Association between handgrip strength and mortality risk among cardiovascular disease patients: A cohort study from the NHANES 2011–2014. Journal of the Formosan Medical Association. 2024. https://doi.org/10.1016/j.jfma.2024.12.018.
5. Zhang F, Luo B, Bai Y, Zhang Y, Huang L, Lu W. Association of handgrip strength and risk of cardiovascular disease: a population-based cohort study. Aging Clinical and Experimental Research. 2024;36(1):1-12. https://doi.org/10.1007/s40520-024-02856-x.
6. Xiao M, Lu Y, Li H, Zhao Z. Association Between Handgrip Strength and Mortality of Patients With Coronary Artery Disease: A Meta-Analysis. Clinical Cardiology. 2024;47(7):e24322. https://doi.org/10.1002/clc.24322.
7. Pai M, Vennu V. Association between Cardiovascular Disease and Handgrip Strength Risk among Community-Dwelling Older Adults in India. Journal of Applied Gerontology. 2024:07334648241302457. https://doi.org/10.1177/07334648241302457
8. Miura S. Evidence for exercise therapies including isometric handgrip training for hypertensive patients. Hypertension Research. 2025;48(2):846-8. https://doi.org/10.1038/s41440-024-02033-7
9. Leong DP, Teo KK, Rangarajan S, Lopez-Jaramillo P, Avezum A, Orlandini A, et al. Prognostic value of grip strength: findings from the Prospective Urban Rural Epidemiology (PURE) study. The lancet. 2015;386(9990):266-73. https://doi.org/10.1016/S0140-6736(14)62000-6
10. Cai Y, Liu L, Wang J, Gao Y, Guo Z, Ping Z. Linear association between grip strength and all-cause mortality among the elderly: results from the SHARE study. Aging clinical and experimental research. 2021;33:933-41. https://doi.org/10.1007/s40520-020-01614-z
11. Celis-Morales CA, Welsh P, Lyall DM, Steell L, Petermann F, Anderson J, et al. Associations of grip strength with cardiovascular, respiratory, and cancer outcomes and all cause mortality: prospective cohort study of half a million UK Biobank participants.BMJ. 2018;361. https://doi.org/10.1136/bmj.k1651
12. Yamal J-M, Martinez J, Osani MC, Du XL, Simpson LM, Davis BR. Mortality and morbidity among individuals with hypertension receiving a diuretic, ACE inhibitor, or calcium channel blocker: A secondary analysis of a randomized clinical trial. JAMA network open. 2023;6(12):e2344998-e. https://doi.org/10.1001/jamanetworkopen.2023.44998
13. Wang Q, Chen P-p, Guo J-y, Wang S-j, Bao Y-y, Zhang Y, et al. Dietary vitamin K intake in relation to skeletal muscle mass and strength among adults: a cross-sectional study based on NHANES. Frontiers in Nutrition. 2024;11:1378853. https://doi.org/10.3389/fnut.2024.1378853.
14. Unger T, Borghi C, Charchar F, Khan NA, Poulter NR, Prabhakaran D, et al. 2020 International Society of Hypertension global hypertension practice guidelines. Hypertension. 2020;75(6):1334-57. https://doi.org/10.1161/HYPERTENSIONAHA.120.15026
15. Li R, Xia J, Zhang X, Gathirua-Mwangi WG, Guo J, Li Y, et al. Associations of muscle mass and strength with all-cause mortality among US older adults. Medicine and science in sports and exercise. 2018;50(3):458. https://doi.org/10.1249/MSS.0000000000001448
16. Setoyama Y, Honda T, Tajimi T, Sakata S, Oishi E, Furuta Y, et al. Association between dynapenic obesity and risk of cardiovascular disease: The Hisayama study. Journal of Cachexia, Sarcopenia and Muscle. 2024;15(6):2338-48. https://doi.org/10.1002/jcsm.13564..
17. Cominacini M, Federti E, Menotti S, Donini M, Ceolan J, Villaboni S, et al. Exploring Muscular Senescence in Transfusion-Dependent Thalassemia without Cardiac Iron Overload: Roles of FGF21,

ICAM1 and Post-Exercise Pulmonary Congestion. Blood. 2024;144:3858. https://doi.org/10.1182/blood-2024-204793.
18. Li C, Li Y, Wang N, Ge Z, Shi Z, Wang J, et al., editors. Intestinal permeability associated with the loss of skeletal muscle strength in middle-aged and older adults in rural area of Beijing, China. Healthcare; 2022: MDPI. https://doi.org/10.3390/healthcare10061100.
19. Zhang Y, Zhu Y, Guo Q, Wang W, Zhang L. High-throughput sequencing analysis of the characteristics of the gut microbiota in aged patients with sarcopenia. Experimental gerontology. 2023;182:112287. https://doi.org/10.1016/j.exger.2023.112287.
20. Park S, hyun Park S, Lee J, Choi Y-J, Kang DO, Park EJ, et al. Association of Handgrip Strength With Readmission, Mortality, and Exercise Capacity in Patients With Heart Failure. Journal of Aging and Physical Activity. 2025;1(aop):1-11. https://doi.org/10.1123/japa.2023-0298.
21. Liu W, Lai X, Shi D, Wang H, Fan L, Shi W, et al. Associations of grip strength asymmetry with multiple health outcomes. American Journal of Preventive Medicine. 2025. https://doi.org/10.1016/j.amepre.2025.02.003.
22. Patil M, Patil V. P128 ESTABLISHING A RELATIONSHIP BETWEEN BLOOD PRESSURE AND HANDGRIP STRENGTH AMONG OBESE PATIENTS. Journal of Hypertension. 2024;42(Suppl 3):e104-e5. https://doi.org/10.1097/01.hjh.0001063384.54153.82.
23. Mantilla AL, Gomez LP, Camacho-Lypez P, Otero-Wandurraga J, Novella B, Gonz6lez-Medina A, et al. Community-based model for management and follow-up by non-physician healthcare workers to improve awareness, treatment, and control of hypertension: The COTRACO study protocol. Heliyon. 2025;11(2). https://doi.org/10.1016/j.heliyon.2025.e41726.
24. Kagiyama N, Kamiya K, Toki M, Saito H, Iwata K, Matsue Y, et al. Prognostic impact of diaphragm thickness in geriatric patients with heart failure: the SONIC-HF multicenter registry. Cardiovascular Imaging. 2025;18(3):389-91. https://doi.org/10.1016/j.jcmg.2024.09.012..