

Original Article

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Pulse wave velocity as a non-invasive discriminative marker of bone health in postmenopausal women

Abstract

Background: Increased arterial stiffness has been linked to osteoporosis. This study evaluates Pulse Wave Velocity (PWV) as a discriminator of osteopenia and osteoporosis at the lumbar spine and femoral neck in postmenopausal women.

Methods: In this cross-sectional study, arterial stiffness and bone mineral density (BMD) at the spine and femoral neck were assessed using carotid-to-femoral Pulse PWV and dual-energy x-ray absorptiometry (DXA). Eighteen premenopausal women (mean age 41.30±4.30 years) and forty-eight postmenopausal women (mean age 59.40±9.10 years) participated in the study. Osteoporosis (T-score ≤-2.5) and osteopenia (-2.5< T-score ≤-1) were defined by T-score criteria.

Results: Pearson's correlation results showed: PWV and femoral neck BMD ($r = -0.439$, $P = 0.002$), PWV and spine BMD ($r = -0.358$, $P = 0.012$), and PWV and menopausal duration ($r = 0.381$, $P = 0.008$). PWV demonstrated significant discriminative ability for femoral neck osteopenia [$b = 0.6472$, Wald = 5.717, Odds ratio (OR) = 1.910, $P = 0.017$, Area Under Curve (AUC) = 0.704], femoral neck osteoporosis ($b = 0.7206$, Wald = 5.838, OR = 2.056, $P = 0.016$, AUC = 0.758), spine osteopenia ($b = 0.8489$, Wald = 5.639, OR = 2.337, $P = 0.018$, AUC = 0.677), and spine osteoporosis ($b = 1.0829$, Wald = 8.269, OR = 2.953, $P = 0.004$, AUC = 0.804).

Conclusion: PWV was inversely correlated with femoral neck and spine BMD, and positively correlated with menopausal duration. It serves as a significant discriminator of bone health, indicating increased odds of osteopenia and osteoporosis.

Keywords: Pulse wave velocity, Arterial stiffness, Bone mineral density, Osteoporosis, Dual-energy x-ray absorptiometry, Menopause.

Citation:

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Osteoporosis, a metabolic disorder characterized by reduced bone mass and disrupted bone microarchitecture, is commonly assessed by dual-energy x-ray absorptiometry (DXA) of the lumbar spine and femoral neck (1, 2). Osteopenia and osteoporosis have been associated with increased cardiovascular risk and arterial calcification, conditions that share common pathophysiological pathways and traditional cardiovascular risk factors, including hypertension, diabetes, and smoking (3,4). Arterial calcification and vascular remodeling may contribute to increased arterial stiffness, which is commonly assessed by pulse wave velocity (PWV), with carotid-to-femoral PWV considered the gold standard (5, 6). Several studies have reported an inverse association between bone mineral density (BMD) and PWV (7, 8), both of which are associated with increased cardiovascular risk, particularly in women (9). In line with this, Park et al. (2022) found that lower bone mineral density is associated with a higher risk of atherosclerotic cardiovascular disease in women (10). Bone loss accelerates during the menopausal transition (11), contributing to reduced bone mineral density and increased cardiovascular risk.



Previous studies have highlighted the coexistence of osteoporosis, arterial stiffness, and atherosclerotic changes in postmenopausal women (12, 13). Despite limited research in this area, our study investigates the relationship between BMD and arterial stiffness in postmenopausal women, considering that elevated PWV may reflect low BMD, with implications for identifying individuals at risk of reduced bone mass. This study aimed to assess the relationship between arterial stiffness, measured by PWV, and bone health, indicated by BMD at the femoral neck and spine using DXA, in postmenopausal women. Additionally, we aimed to evaluate how PWV relates to the risk of osteopenia and osteoporosis at the femoral neck and spine, and its potential for identifying bone health status in this population.

Methods

Study population: This cross-sectional study was conducted at Clinical Hospital–Bitola between August and October 2023. Sixty-six consecutive women (mean age 56.5 ± 10.7 years) who underwent DXA screening for osteoporosis were prospectively enrolled, and Doppler sonography was used to measure aortic PWV. Patients with malignant tumors, prior osteoporosis treatment, myocardial infarction, coronary revascularization, degenerative disease, chronic kidney disease, or incomplete BMD data at the spine or femoral neck were excluded. Written informed

consent was obtained, and the study was approved by the institutional ethics committee (approval no. 07_2023/45, dated 05.07.2023).

The final study population consisted of 18 premenopausal women (mean age 41.30 ± 4.30 years) and 48 postmenopausal women (mean age 59.40 ± 9.10 years). Rates of diabetes, hypertension, and smoking were 22.2%, 33.3%, and 44.4% in premenopausal women versus 25%, 47.9%, and 22.9% in postmenopausal women. Menopause was defined as absence of menstruation for at least one year.

Pulse wave velocity estimation: PWV measurements were performed when participants had normal blood pressure, defined as systolic 110–125 mmHg and diastolic 70–82 mmHg, to minimize the influence of blood pressure on arterial stiffness measurements. A narrow range was chosen because PWV is highly pressure-dependent, improving measurement accuracy (14). Signals from the left common carotid artery (CCA) and left common femoral artery (CFA) were recorded using a GE Logiq Pro 5 Doppler system (5–10 MHz probe) synchronized with electrocardiography (ECG). As shown in figure 1, D1/T1 and D2/T2 represent the distances and travel times from the heart to the CCA and CFA, respectively. The delay $\Delta T = T2 - T1$ indicates the difference in wave arrival times, and PWV was calculated as $\Delta D / \Delta T$, where $\Delta D = D2 - D1$. Distances were measured on the body surface with a tape. This Doppler method for carotid-to-femoral PWV has been validated in previous studies (8, 15–18), including our own research.

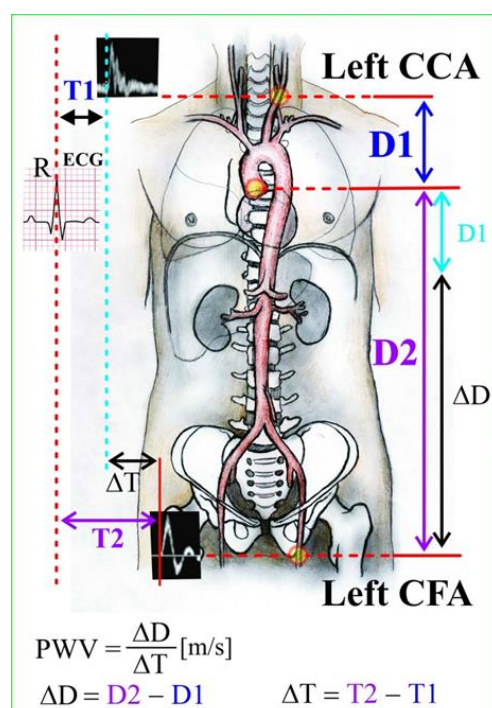


Figure 1. Estimating Pulse Wave Velocity (PWV) through synchronized Doppler and electrocardiography (ECG) signals: Time diversity analysis of carotid and femoral artery signals.

Figure 2 presents representative Doppler recordings from the left CCA (left panel) and the left CFA (right panel), both synchronized with ECG. The measured intervals from the R-wave to the foot of the systolic waveform are indicated as T1 (86 ms) for the CCA and T2 (157 ms) for the CFA. The difference between these values ($\Delta T = 71$ ms) represents the pulse transit time between the two arterial sites. Using the measured carotid–femoral distance of 58 cm, the calculated PWV was 8.17 m/s.

Bone mineral density estimation: DEXA scans were performed using a Hologic Delphi QDR4500A/SL system, measuring BMD at the femoral neck and lumbar spine. For spine assessment, legs were supported to flatten the pelvis; for hip assessment, the foot was positioned to rotate the hip inward. BMD was calculated from x-ray absorption after correcting for soft tissue.

World Health Organization (WHO) recognizes DXA as the gold standard for assessing BMD in postmenopausal women, defining osteoporosis as a T-score ≤ -2.5 and osteopenia as $-2.5 < \text{T-score} \leq -1$. The T-score compares

patient BMD to a young-adult reference population. The DXA procedure and interpretation have been detailed in prior studies (18–21).

Statistical analysis: Statistical analyses were performed using MedCalc for Windows, Version 22.002 (MedCalc Software bv, Ostend, Belgium). Demographic and clinical variables were compared using unpaired Student's t-test. Linear relationships between continuous variables were assessed with Pearson's and Spearman's correlations. Associations between PWV and spine or femoral neck BMD were evaluated using linear and 3D regression analyses. Backward stepwise logistic regression was performed to examine the association of PWV with osteopenia/osteoporosis and to calculate odds ratios. An age-adjusted model was constructed using residualization to ensure age-independent associations. Receiver operating characteristic (ROC) curve analysis was used to evaluate the discriminatory ability of PWV for bone health, with an area under the curve (AUC) > 0.5 indicating meaningful classification.

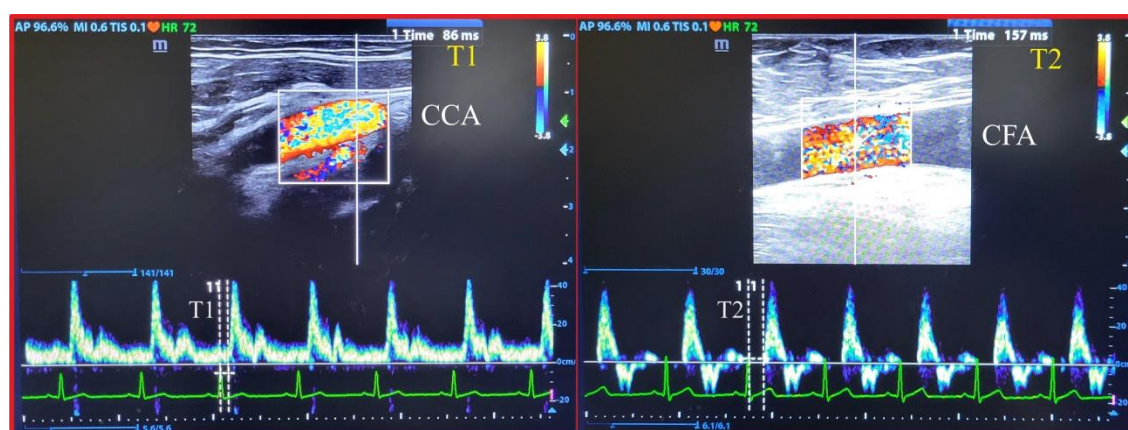


Figure 2. Representative Carotid–Femoral Pulse Wave Velocity Assessment (CCA, common carotid artery; CFA, common femoral artery).

Results

Characteristics of the patients and unpaired t-test: DXA and carotid-to-femoral PWV were measured in 18 premenopausal and 48 postmenopausal women. Significant differences were found between groups for age (41.30 ± 4.30 vs 59.40 ± 9.10 , $P = 0.031$), spine BMD (0.896 ± 0.179 vs 0.852 ± 0.137 , $p < 0.001$), femoral neck BMD (0.904 ± 0.160 vs 0.817 ± 0.141 , $P = 0.035$), and PWV (7.99 ± 1.63 vs 9.69 ± 1.40 , $p < 0.001$). Within the premenopausal group, the difference in bone mineral density between the spine (0.896 ± 0.179 g/cm²) and femoral neck (0.904 ± 0.160 g/cm²) was not statistically significant ($P = 0.248$). In contrast, within the postmenopausal group, a statistically significant difference was observed between these anatomical sites

(spine: 0.852 ± 0.137 g/cm²; femoral neck: 0.817 ± 0.141 g/cm²; $P = 0.047$). These results are presented in table 1. No significant differences were observed for BMI, diabetes, hypertension, smoking, or T- and Z-scores at the spine and femoral neck.

Bivariate correlation analysis: In the postmenopausal group, PWV showed a strong positive correlation with age ($r = 0.796$, $p < 0.001$) and significant inverse correlations with spine BMD ($r = -0.358$, $P = 0.012$), femoral neck BMD ($r = -0.439$, $P = 0.002$), spine T-score ($r = -0.317$, $P = 0.028$), and femoral neck T-score ($r = -0.319$, $P = 0.027$). No significant correlations were observed between PWV and BMI, diabetes, hypertension, smoking, or Z-scores. Clinical data, and correlations with PWV are summarized in table 2.

Table 1. Demographic and clinical characteristics of premenopausal and postmenopausal women

Variables	Premenopausal N = 18 Mean±SD n (%)	Postmenopausal N = 48 Mean±SD n (%)	Test statistics-t	P-value
Age, years	41.30±4.30	59.40±9.10	5.850	0.031
BMI, kg/m ²	25.09±2.26	28.17±3.91	2.282	0.105
Diabetes	4 (22.2)	12 (25)	-0.657	0.455
Hypertension	6 (33.3)	23 (47.9)	-0.573	0.312
Smoking	8 (44.4)	11 (22.9)	6.237	0.048
Menopausal duration, years	/	7.67±7.04	/	/
Spine BMD, g/cm ²	0.896±0.179	0.852±0.137	-0.722	< 0.001
Femoral neck BMD, g/cm ²	0.904±0.160	0.817±0.141	2.152	0.035
Spine T-score	-1.175±0.907	-1.452±1.566	0.705	0.483
Spine Z-score	-0.857± 1.003	-0.344±1.066	1.829	0.082
Femoral neck T-score	-0.496±1.105	-0.873±0.896	1.427	0.158
Femoral neck Z-score	-0.171±1.136	0.360±0.860	1.988	0.051
PWV, m/s	7.99±1.63	9.65±1.40	4.101	<0.001

BMI, Body Mass Index; BMD, Bone Mineral Density; PWV, Pulse Wave Velocity; The results are presented as: Mean and Standard Deviation (SD), number (n) and percent (%).

Table 2. Correlation of demographic and clinical variables with pulse wave velocity (PWV) in postmenopausal women

Variables	PWV, m/s (postmenopausal)	
	Correlation coefficient r (ρ)	P
Age, years	0.796	<0.001
BMI, kg/m ²	0.043	0.771
Diabetes	(-0.219)	0.136
Hypertension	(0.062)	0.694
Smoking	(0.031)	0.818
Menopausal duration, years	0.381	0.008
Spine BMD, g/cm ²	-0.358	0.012
Femoral neck BMD, g/cm ²	-0.439	0.002
Spine T-score	-0.317	0.028
Spine Z-score	0.054	0.713
Femoral neck T-score	-0.319	0.027
Femoral neck Z-score	0.135	0.361

PWV, Pulse Wave Velocity; BMI, Body Mass Index; BMD, Bone Mineral Density. r (ρ), Pearson "r" and Spearman "(ρ)" correlation coefficients.

Linear regression analysis: Linear regression assessed the relationship between PWV (Y) and BMD of the spine or femoral neck (X), with equations ($Y = b_0 - b_1 \cdot X$) and coefficients of determination (R^2). For spine BMD, $R^2 =$

0.1281, indicating that 12.8% of PWV variability is explained by spine BMD. Each 1 g/cm² increase in spine BMD corresponded to a 3.689 m/s decrease in PWV ($P = 0.0125$). For femoral neck BMD, $R^2 = 0.1928$, with each 1

g/cm² increase associated with a 4.403 m/s decrease in PWV ($P = 0.0018$). Figure 3 shows heat scatter plots with regression lines and intervals. Regression parameters b_0 (12.80–13.26) represent theoretical PWV at zero BMD, and b_1 values (-3.689 for spine, -4.403 for femoral neck) quantify PWV reduction per unit BMD increase. (

3D (three-dimensional) linear regression plot: Figure 4 shows a 3D scatter plot illustrating the linear regression

between PWV, femoral neck BMD, and spine BMD. The regression equations are: $y = 13.2613 - 4.4029 \cdot X$ (PWV vs femoral neck BMD, $P = 0.002$), $y = 12.8022 - 3.6892 \cdot X$ (PWV vs spine BMD, $P = 0.013$), and $y = 0.2499 + 0.6675 \cdot X$ (femoral neck BMD vs spine BMD, $p < 0.001$), where y is the predicted value and X the independent variable. These equations visually depict the relationships and interdependencies among the variables.

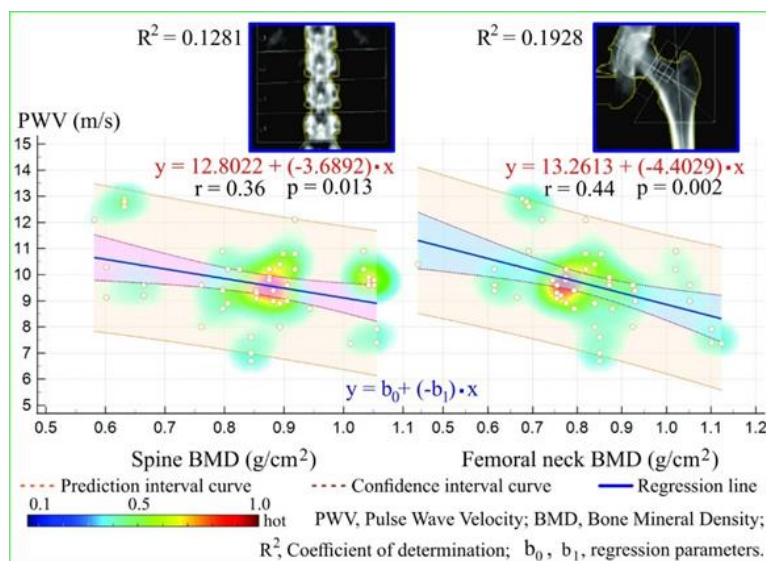


Figure 3. Heat scatter plot with regression line, prediction and interval curve, representing the linear regression between pulse wave velocity and bone mineral density of spine and femoral neck.

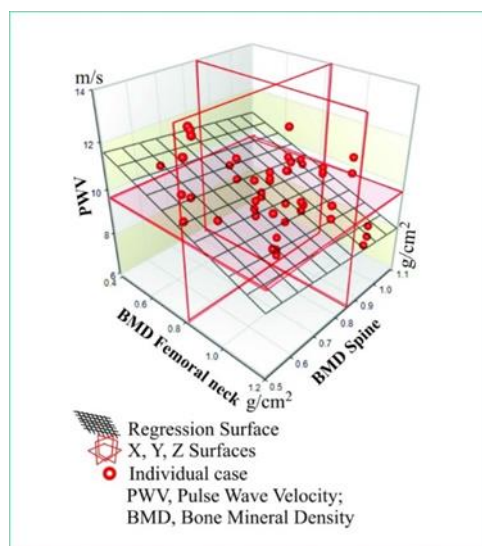


Figure 4. Three - Dimensional scatter plot with regression surface representing linear correlation between the Pulse Wave Velocity (y) with the femoral neck (x) and spine Bone Mineral Density (z).

Diagnostic test evaluation (Receiver Operating Characteristic curve): We used ROC curve analysis to evaluate PWV's ability to discriminate between women with and without osteopenia/osteoporosis. Figure 5 shows

ROC curves for PWV versus DXA at the spine and femoral neck, including AUC, sensitivity, specificity, Youden index (J), and optimal cut-off points. The PWV cut-off for osteopenia is 9.7 m/s (sensitivity 65%, specificity 71.4%),

indicating its moderate potential for identifying individual at risk. The optimal PWV cut-offs were 9.7 m/s for osteopenia and 9.4 m/s for osteoporosis at the femoral neck, and 9.4 m/s for osteopenia and 10.2 m/s for osteoporosis at the spine. **Given the modest AUC values and single-center design, these PWV cut-offs should be considered exploratory and require external validation before clinical application.** Discriminative performance was highest for spine osteoporosis, with AUC 0.804, J = 0.5684, sensitivity 70%, specificity 86.4%, and a PWV threshold >10.2 m/s.

Logistic Regression (Odds Ratios and Risk Assessment for Osteopenia and Osteoporosis): Logistic backward regression assessed PWV as a significant discriminator of osteopenia and osteoporosis at the spine and femoral neck (as shown in table 3 below). Among menopausal women, femoral neck osteopenia and osteoporosis were 41.7% and 22.9%, respectively, while spine osteopenia and osteoporosis were 39.6% and 20.8%.

Table 3. Logistic regression analysis assessing the association and discriminative performance of pulse wave velocity for osteopenia and osteoporosis at the femoral neck

and spine, using an age-matched model with residualization. Mean PWV increased with worsening bone status: femoral neck – normal 8.84±1.13, osteopenia 9.83±1.16, osteoporosis 10.51±1.14 m/s (ANOVA F = 7.68, P = 0.001); spine – normal 9.21±1.06, osteopenia 9.45±1.39, osteoporosis 10.87±1.64 m/s (F = 5.63, P = 0.007). PWV showed the strongest association with spine osteoporosis (Wald = 8.269; OR = 2.96; P = 0.004), with significant associations observed for femoral neck osteopenia/osteoporosis and spine osteopenia. Other variables, including, BMI, diabetes, hypertension, smoking, and menopausal duration, were initially included in the logistic regression model, but were subsequently removed by the backward selection procedure due to lack of statistical significance (p > 0.5). Notably, hypertension was not significantly correlated with PWV in our cohort. Consequently, PWV remained the primary significant discriminator of osteopenia and osteoporosis. Because age showed strong collinearity with PWV, age-adjusted (residual) PWV values were used in the logistic regression model to minimize multicollinearity and ensure stable parameter estimates.

Table 3. Logistic regression analysis assessing the association and discriminative performance of pulse wave velocity for osteopenia and osteoporosis at the femoral neck and spine, using an age-matched model with residualization

Dependent Y					Femoral neck (Osteopenia)					Dependent Y					Spine (Osteopenia)				
Positive cases					20 (41.67%)					Positive cases					19 (39.58%)				
Negative cases					28 (58.33%)					Negative cases					29 (60.42%)				
Coefficients and Standard Error (Std. Err.)																			
Variable		Coefficient		Std. Err.		Wald		P		Variable		Coefficient		Std. Err.		Wald		P	
PWV		0.6472		0.2706		5.717		0.017		PWV		0.8489		0.3575		5.639		0.018	
Age (resid)		0.2457		0.1328		3.420		0.064		Age (resid)		0.2731		0.1653		2.730		0.071	
Constant		-6.6272		2.6593		6.210		0.013		Constant		-10.6132		3.8353		7.657		0.006	
Odds Ratios and 95% Confidence Intervals (CI)																			
Variable		Odds ratio		95% CI		Variable		Odds ratio		95% CI									
PWV		1.910		1.123 to 3.247		PWV		2.337		1.160 to 4.710									
Age (resid)		1.278		0.986 to 1.658		Age (resid)		1.314		0.949 to 1.818									
Dependent Y					Femoral neck (Osteoporosis)					Dependent Y					Spine (Osteoporosis)				
Positive cases					11 (22.92%)					Positive cases					10 (20.83%)				
Negative cases					37 (77.08%)					Negative cases					38 (79.17%)				
Coefficients and Standard Error (Std. Err.)																			
Variable		Coefficient		Std. Err.		Wald		P		Variable		Coefficient		Std. Err.		Wald		P	
PWV		0.7206		0.2982		5.838		0.016		PWV		1.0829		0.3766		8.269		0.004	
Age (resid)		0.2913		0.1487		3.840		0.089		Age (resid)		0.2317		0.1487		2.430		0.119	
Constant		-8.3946		3.0591		7.530		0.006		Constant		-12.2657		3.9049		9.867		0.002	
Odds Ratios and 95% Confidence Intervals (CI)																			
Variable		Odds ratio		95% CI		Variable		Odds ratio		95% CI									
PWV		2.056		1.146 to 3.688		PWV		2.953		1.412 to 6.178									
Age (resid)		1.338		1.000 to 1.791		Age (resid)		1.261		0.941 to 1.689									

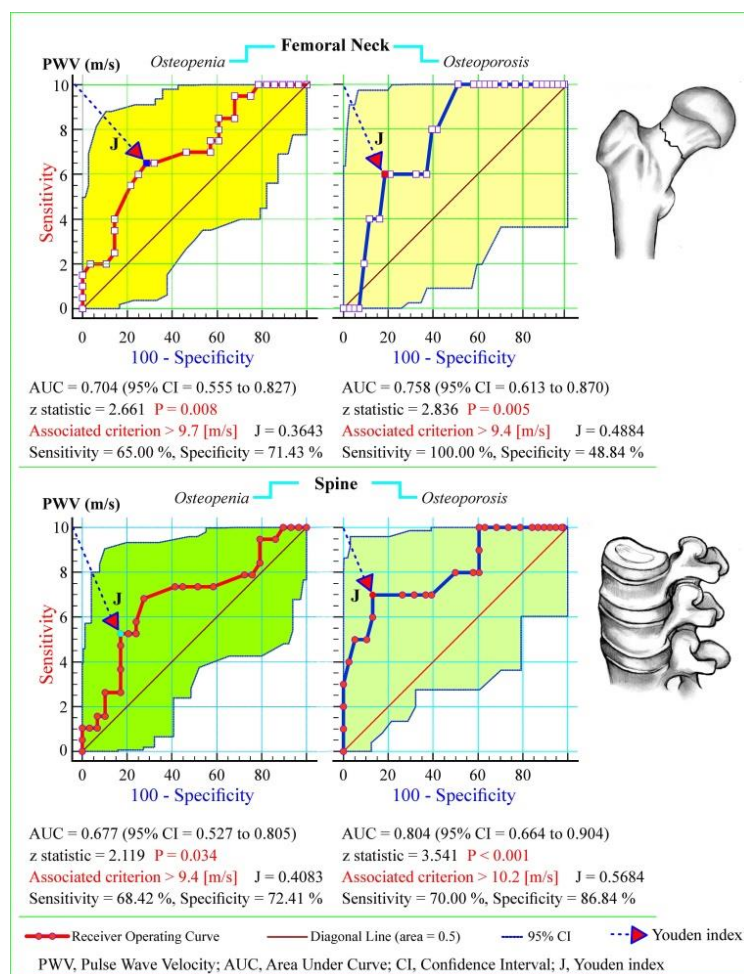


Figure 5. Receiver operating characteristic curves for Pulse Wave Velocity as a marker for detecting the femoral neck and lumbar spine bone health (osteopenia and osteoporosis).

Discussion

To our knowledge, this study represents a pioneering effort to explore PWV as a potential discriminative marker for osteopenia and osteoporosis in menopausal women. While prior studies have examined the inverse correlation between PWV and BMD, as well as links with atherosclerosis and cardiovascular risk (1, 7, 8, 13, 22, 23), none have specifically assessed PWV's discriminatory power for osteopenia and osteoporosis in this cohort using carotid-to-femoral Doppler PWV and DXA. Premenopausal and menopausal groups were well-matched for key demographics, supporting that observed differences reflect menopausal status rather than confounders. The higher spine BMD compared to the femoral neck in menopausal women ($P = 0.047$), unlike in premenopausal women ($P = 0.248$), may reflect DXA measurement artefacts. Denser aortic calcification, lumbar spondylosis, or osteoarthritis can falsely elevate spine BMD, especially in chronic kidney disease (19, 24-27). Zaydun et al. (2006) showed that longer menopausal duration independently contributes to increased

aortic PWV in early postmenopause, indicating progressive arterial stiffening (28). Previous studies have similarly reported an inverse relationship between BMD and PWV (Sumino et al., 2006), consistent with our finding of a negative correlation between femoral neck BMD and PWV ($r = -0.358$) (13). In our study, bivariate correlation confirmed a strong positive association between PWV and age, reflecting normal vascular aging. After adjusting for age, PWV remained associated with menopausal duration ($P = 0.008$), suggesting arterial stiffening during menopause occurs beyond normal aging. Declining estrogen further accelerates stiffness, affecting vascular health and elevating PWV (13, 29, 30). The gradual decline in arterial compliance from the ascending aorta to the iliac arteries occurs with aging, independently of resistance arteries (29). Menopausal duration is an important factor in arterial stiffness. Jaalkhorol et al. (2019) reported that low BMD correlates with higher PWV, identifying individuals with low BMD as a potential target for arterial stiffness assessment (31). Our findings support this, showing

significant correlations between PWV and spine BMD ($p < 0.012$) and femoral neck BMD ($P = 0.002$) (table 2).

Mikumo et al. (2008) reported a significant correlation between lumbar spine BMD and arterial stiffness, measured by brachial-ankle PWV, in 143 postmenopausal women. Despite methodological differences, their findings align with ours, supporting the link between lower bone mass and increased arterial stiffness and emphasizing the need to monitor atherosclerosis in patients with low BMD (32). Samargandy et al. (2020) observed an increase in PWV from 7.74 m/s four years before menopause to 8.85 m/s in the first four years after menopause, independent of age, cardiovascular disease factors, estradiol, and follicle-stimulating hormone levels. These findings, *indicating significantly greater arterial stiffness in menopausal women*, align with our results (7.99 m/s vs. 9.65 m/s, $p < 0.001$). In menopausal women, estrogen deficiency increases the risk of cardiovascular disease and osteoporosis (33), as estrogen acts on bone and arterial tissues via receptors in smooth muscle cells, endothelial cells, osteoblasts, and osteoclasts (34). Hormone replacement therapy (HRT) increases BMD and reduces PWV, highlighting estrogen's role in bone turnover and arterial stiffness (3, 35, 36). Based on linear regression analysis, spine and femoral neck BMD are associated with PWV. For each 1 g/cm² increase, spine BMD decreases PWV by 3.6892 m/s, and femoral neck BMD decreases it by 4.4029 m/s (figure 3), highlighting the stronger influence of femoral neck BMD on arterial stiffness. Individuals with higher BMD at these sites tend to have more elastic arteries, possibly due to beneficial effects of bone health or shared mechanisms affecting both BMD and arterial stiffness. PWV variation is 12.81% dependent on spine BMD and 19.28% on femoral neck BMD, reflecting structural and functional differences. Linear regression allows estimation of PWV from DXA-measured BMD, and vice versa.

The discriminatory power of PWV for spine and femoral neck osteopenia/osteoporosis was assessed via ROC analysis. Femoral neck osteoporosis risk increased at PWV ≥ 9.4 m/s, and spine osteoporosis at PWV > 10.2 m/s. When PWV exceeds 9.4 m/s, DXA evaluation is recommended. AUC values ranged from 0.677 to 0.804, with substantial sensitivity and specificity, supporting these cut-offs (37). ROC-derived J values increased sequentially: 0.3643 (femoral neck osteopenia), 0.4083 (spine osteopenia), 0.4884 (femoral neck osteoporosis), and 0.5684 (spine osteoporosis) (figure 5). PWV showed the highest performance for spine osteoporosis (AUC 0.804, J 0.5684, sensitivity/specificity 70%/86.4%). A meta-analysis reported similar cut-offs (~ 9.5 m/s) for detecting significant

BMD reduction, aligning with our findings (38). In our previous study (8), each unit increase in PWV above 9.4 m/s raised the hazard ratio for cardiovascular mortality by 1.2977, highlighting the importance of maintaining PWV below this threshold. Elevated PWV may increase cardiovascular risk through premature return of reflected waves, raising central pulse pressure and left ventricular load, reducing ejection fraction, and increasing myocardial oxygen demand. Higher PWV can also contribute to left ventricular hypertrophy, impaired coronary perfusion, and progression of atherosclerosis (39, 40).

The prevalence of osteopenia and osteoporosis at the femoral neck and spine among menopausal women in our study is consistent with the findings of Balaguer et al. (2005), who also reported similar rates in a menopausal cohort (41). Specifically, they found that 50.4% had osteopenia compared to our findings of 41.67%, as well as 29.6% had osteoporosis compared to our finding of 22.92% at the femoral neck (table 3). Although prevalence rates vary, differences likely reflect age and menopause duration, yet the overall trend of notable osteopenia and osteoporosis in menopausal women remains evident.

The measured PWV in women with different bone conditions (normal BMD, osteopenia, and osteoporosis) showed a statistically significant increase as BMD decreased from normal to osteoporotic. Specifically, PWV values associated with the femoral neck measurements were 8.84, 9.832, and 10.513 m/s. Also, the PWV values associated with the spine measurements were 9.211, 9.454, and 10.865 m/s, respectively. These results indicate a progressive increase in PWV as bone strength decreases from normal to osteoporotic conditions at the spine and femoral neck. Lebrun et al. (2002) demonstrated in their cross-sectional study of 385 postmenopausal women a progressive increase in PWV, providing evidence that the most established cardiovascular risk factors are determinants of aortic PWV. An increased PWV is associated with a higher risk of stroke, coronary heart disease, and mortality within 10-12 years (8, 27). They found that an increase in PWV of 1 m/s was associated with a 2.2% increase in the absolute risk of death and a 1.3% increase in the risk of coronary heart disease (27). The odds ratios indicate that higher PWV significantly increases the risk of femoral neck osteopenia (OR = 1.91), femoral neck osteoporosis (OR = 2.06), spine osteopenia (OR = 2.34), and spine osteoporosis (OR = 2.95), corresponding to 91%, 106%, 134%, and 195% increased odds, respectively. The Wald statistic indicates the strongest discriminative contribution of PWV for spine osteoporosis (Wald = 8.269), while PWV also showed significant potential for identifying

individuals at risk of femoral neck osteopenia (Wald = 5.717), femoral neck osteoporosis (Wald = 5.838), and spine osteopenia (Wald = 5.639). These findings support PWV as a significant discriminator for bone health, complementing its established role in cardiovascular risk assessment. Although few studies have examined PWV as a prognostic marker of bone health, Tang et al. (2021) (42) reported an 82.69% higher osteoporosis risk ($b = -0.190$) in women under 65 with elevated baPWV, using DXA and Osteoporosis Self-Assessment Tool for Asians (OSTA) score, highlighting baPWV's independent predictive value for osteoporosis.

This aligns closely with the results and conclusions of our study. Despite differences in measurement techniques (our study focuses on carotid-to-femoral PWV and theirs on baPWV) their findings align with our results, underscoring the predictive value of arterial stiffness in bone health assessments. However, it is important to note that the cohorts are only partially comparable. Tang et al. (2021) did not specifically investigate menopausal women, which might explain why they reported a smaller risk for osteoporosis based on elevated PWV compared to our findings. Taken together, our findings indicate that PWV may help identify osteopenia and osteoporosis in postmenopausal women, with the strongest association observed for spine osteoporosis.

These results underscore the potential utility of PWV as a non-invasive marker to aid in identifying women who may benefit from further bone health assessment. In line with previous observations, symptomatic musculoskeletal conditions such as knee osteoarthritis may not be directly associated with reduced bone mineral density, highlighting the need for alternative approaches to detect individuals at risk (Babaei et al., 2025). While our study provides promising evidence, prospective, multicenter investigations are required to confirm these findings and establish the clinical applicability of PWV in routine bone health evaluation (43).

Limitations and strengths: This study has some limitations. The relatively small sample size may limit generalizability, and laboratory biomarkers linked to osteoporosis and atherosclerosis were not assessed. Although PWV measurements were performed under controlled normal blood pressure conditions (systolic 110–125 mmHg, diastolic 70–82 mmHg), we did not explicitly control or record mean arterial pressure (MAP), which may have a residual influence on the results. Larger longitudinal studies with biomarker analysis are needed to confirm these findings. Despite these limitations, PWV was significantly associated with bone health parameters at both the spine and

femoral neck, suggesting its potential utility as a marker of bone status. Importantly, this study represents a novel application of carotid-to-femoral PWV in this context, highlighting its possible clinical relevance.

Conclusion: PWV correlates strongly with age and menopausal duration, showing menopause's specific influence on vascular health independent of the aging process. Higher BMD at the spine and femoral neck is associated with greater arterial elasticity, whereas increasing PWV parallels declining bone strength and demonstrates discriminative ability for osteopenia and osteoporosis. PWV may have potential as a complementary, non-invasive screening marker of bone health, independent of age.

It could help indicate women who might be at risk, support consideration of DXA referral, and shows the greatest observed association with spine osteoporosis, with the risk tripling once PWV exceeds 10.865 m/s. Carotid-to-femoral PWV therefore holds clinical value for early osteoporosis detection and monitoring, with additional relevance for cardiovascular risk assessment. Within the limitations of a cross-sectional design, carotid-to-femoral PWV demonstrates potential clinical utility as an associative and discriminative marker of bone mineral density status, with additional relevance for cardiovascular risk stratification. However, prospective, multicenter studies are needed to confirm these findings and establish clinical utility.

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