



Article

The Correlations of the Neutrophil-to-Lymphocyte Ratio and Platelet-to-Lymphocyte Ratio with Bone Mineral Density in Postmenopausal Women: A Cross-Sectional Study

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Abstract: Background/Objectives: Osteoporosis is a skeletal disorder characterized by reduced bone mineral density (BMD) and increased fracture risk. Chronic inflammation is implicated in osteoporosis pathogenesis, with inflammatory mediators promoting bone resorption. The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) are markers of systemic inflammation and have emerged as potential indicators of bone health. This study's aim was to highlight the potential role of the NLR and PLR as markers of bone health in postmenopausal women affected by osteoporosis or osteopenia and to evaluate the possible influence of autoimmune disease in this context. **Methods:** This cross-sectional study included 124 postmenopausal women diagnosed with osteopenia or osteoporosis at the Orthopedic Unit of the Policlinico G. Rodolico in Catania, Italy. Demographic, clinical, laboratory, and diagnostic imaging data were collected. The NLR and PLR were calculated from complete blood counts, and BMD was measured using dual-energy X-ray absorptiometry (DEXA). Statistical analyses included correlations, group comparisons, and multiple and logistic regressions. **Results:** The NLR and PLR did not directly correlate with BMD or fracture incidence. However, the PLR weakly correlated with vitamin D levels. Notably, women without Hashimoto's thyroiditis exhibited higher NLR values than those with the condition. Hypertensive women had a lower PLR than non-hypertensive women, while euthyroid women had a higher PLR than hyperthyroid or hypothyroid women. Multiple regression analysis revealed that age, BMI, CKD stage, vitamin D levels, NLR, PLR, diabetes, and autoimmune diseases significantly predicted BMD at the femur neck, with the PLR contributing significantly. Logistic regression confirmed these predictors for osteoporosis or osteopenia, with an increased PLR being associated with a higher likelihood of osteoporosis. **Conclusions:** While the NLR and PLR may not independently predict bone health, their inclusion in a multifactorial assessment considering age, BMI, vitamin D, and comorbidities could enhance osteoporosis management.

Keywords: osteoporosis; inflammation; neutrophil-to-lymphocyte ratio; platelet-to-lymphocyte ratio; bone health



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1. Introduction

Osteoporosis is a high-impact, chronic, and progressive bone condition that affects over 200 million people worldwide. It is characterized by a loss of bone mass and the

deterioration of bone microarchitecture, leading to increased fragility and a higher risk of fractures. The prevalence of osteoporosis continues to rise, primarily due to aging populations and changes in lifestyle [1]. Diagnosis is typically performed using dual-energy X-ray absorptiometry (DEXA), which measures bone mineral density (BMD) at key sites such as the lumbar spine, hip, and forearm. According to the World Health Organization (WHO), osteoporosis is defined by a T-score of ≤ -2.5 standard deviations (SD) below that of a healthy, young adult, indicating significantly reduced bone density [2].

The development of osteoporosis results from an imbalance in bone remodeling, where the bone resorption carried out by osteoclasts exceeds the bone formation by osteoblasts. Various factors contribute to this imbalance, including hormonal changes, oxidative stress, and chronic low-grade inflammation. Pro-inflammatory cytokines like IL-6 and TNF- α play a critical role by promoting osteoclast activity while inhibiting osteoblast function, ultimately accelerating bone loss [2,3].

Osteoblasts originating from mesenchymal stem cells are responsible for producing and mineralizing the bone matrix while also regulating osteoclast function through factors such as RANKL and OPG. On the other hand, osteoclasts derived from monocyte/macrophage lineages break down bone tissue. In osteoporosis, this delicate balance is disrupted, often due to increased osteoclast activity or impaired osteoblast function. For example, postmenopausal osteoporosis is closely associated with estrogen deficiency, which leads to decreased OPG levels and increased RANKL expression, which ultimately drives excessive bone resorption [4–6].

Cytokines and immune dysregulation amplify this imbalance through chronic inflammation. Age-related low-grade inflammation (termed “inflammaging”) promotes osteoclast activation and osteoblast dysfunction via the release of pro-inflammatory cytokines by senescent bone cells. It has long been known that chronic inflammatory diseases such as rheumatoid arthritis increase systemic inflammation, which directly enhances bone resorption. Glucocorticoid therapy can control inflammation but also suppresses osteoblast activity, thereby worsening bone loss. Emerging treatments targeting interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and the receptor activator of nuclear factor-kappa B ligand (RANKL) have shown promising results in reducing inflammation-related bone loss. Additionally, lifestyle modifications, such as a healthy diet, regular exercise, and stress management, can complement these therapies by helping to lower systemic inflammation [4–6].

Various systemic inflammation markers have been investigated in their role as possible osteoporotic risk factors, particularly the neutrophil-to-lymphocyte ratio (NLR) and the platelet-to-lymphocyte ratio (PLR). High NLR and PLR values have been linked with increased states of systemic inflammation, and most recent studies show that they may also be associated with lower bone density and an increased risk of fracture [7]. Information regarding the NLR and PLR as clinical biomarkers has shown potential relevance in the pathogenesis and progression of osteoporosis. Both the NLR and PLR are derived from botanical parameters and represent different aspects of the inflammatory milieu. The NLR reflects the balance between innate and adaptive immunity by comparing absolute neutrophil and absolute lymphocyte counts, while the PLR emphasizes the role of platelets as mediators of inflammation and bone metabolism.

As an innate immune response, neutrophils secrete inflammatory mediators including interleukin-1 (IL-1), IL-6, and TNF- α . These cytokines stimulate RANKL, which is important for osteoclast differentiation and activation, hence increasing bone resorption [2]. Concurrent lymphopenia marked by a high NLR contributes to a reduced adaptive immune response, which may subsequently compromise bone repair and remodeling. The association between such parameters and therapeutic approaches is also relevant due to the use of

NLR and PLR as biomarkers. The cytokine inhibitors of IL-6 or TNF- α , which are known to be biomarkers of a high NLR and PLR, might represent potential anti-inflammatory treatments that could ameliorate their negative effect on bone health [2–7].

The use of the NLR and PLR in research is growing. However, the reference values vary in different studies and populations, and broad limits have been set to interpret these ratios clinically. The normal value of the NLR is between 1.0 and 3.0. Levels over 3.0 are often indicative of potential systemic inflammation, stress, or worse, immune dysregulation [8]. Low ratios can suggest an immune dysfunction rather than a systemic inflammatory state. Low NLR values (≤ 1) may indicate either lymphocytosis (a high percentage of lymphocytes in the blood) or neutropenia (a lower percentage of neutrophils in the blood). These may arise in the setting of viral infections, selected autoimmune diseases, bone marrow suppression, or some hematologic disorders.

A low NLR may also reflect an enhanced adaptive immune response that might be protective in specific settings but could also be a signal of disturbed immune regulation. In a similar vein, a PLR below the reference range (generally <100) might be due to thrombocytopenia as either a primary process related to bone marrow dysfunction or a secondary process due to drugs or systemic diseases interfering with platelet production or survival [8,9]. In osteoporosis, low NLR or PLR values are rarely studied as attention has been focused on elevated levels as inflammation markers. However, abnormally low ratios can imply an impaired inflammatory response that may restrict their potential to repair microdamage of bone tissue. Nevertheless, one must always take into account the clinical background as the values might differ according to age, comorbidities, and different methodologies among studies.

2. Materials and Methods

2.1. Declarations, Study Design, Setting, and Participants

Informed consent was obtained from all patients for the publication of all their data and images. We conducted a cross-sectional study from November 2023 to November 2024 and recruited 124 consecutive patients from the osteoporosis service of the Orthopedic Unit of the Policlinico G. Rodolico in Catania, Italy. The inclusion criteria included postmenopausal women with a diagnosis of osteopenia or osteoporosis based on the BMD T-score at the femur neck or lumbar spine (a T-score of at least ≤ -1.5). This study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by the ethics committee of Policlinico G. Rodolico.

2.2. Data Collection and Measures

We collected demographic, clinical, laboratory, and diagnostic imaging data from the patients. EGFR was calculated using the 2021 CKD-EPI creatinine formula. The NLR was calculated by dividing the number of neutrophils by the number of lymphocytes, and the PLR was calculated by dividing the platelet count by the number of lymphocytes. The lumbar spine and femur neck BMD were measured using a DEXA Hologic Discovery A device with the Hologic Apex v4.0 software.

The BMI was calculated as weight (kg) divided by the square of height (m^2). The categories of BMI have been defined as underweight ($<18.5 \text{ kg}/\text{m}^2$), normal weight ($18.5\text{--}24.9 \text{ kg}/\text{m}^2$), overweight ($25\text{--}29.9 \text{ kg}/\text{m}^2$), and obesity ($30 \text{ kg}/\text{m}^2$).

The Charlson Comorbidity Index (CCI) was used to quantify comorbidities, deriving a prognostic score from specific clinical conditions.

2.3. Statistical Analysis

Data were expressed as the mean $\pm$ standard deviation (SD) for parametric variables, the median and interquartile range (IQR) for nonparametric variables, and percentages for categorical variables. All the variables were tested for normal distributions by visual representation and via the Shapiro–Wilk test. To test relationships between two or more variables, we applied Pearson’s product moment correlation for parametric variables and Spearman’s rank order correlation for nonparametric variables. The independent samples *t*-test and the Mann–Whitney U test were, respectively, conducted to compare differences in parametric and nonparametric variables between two independent groups.

One-way ANOVA and the Kruskal–Wallis H test were, respectively, used to compare differences in parametric and nonparametric variables across three or more independent groups. To predict the value and the categorization of a variable, we, respectively, used multiple and binomial logistic regression. Statistical significance was considered for $p \leq 0.05$. Statistical analysis was performed using SPSS software version 29.0 (IBM Corp. Released 2023. IBM SPSS Statistics for Macintosh, Version 29.0.2.0 Armonk, NY, USA: IBM Corp).

3. Results

We included 124 postmenopausal women, of which 70.2% were osteoporotic and 29.8% were osteopenic. The mean age of the patients was 64.5 ± 10.4 years, and the mean BMI was 24.8 ± 4.2 kg/m². BMD T-scores in our series were -2.3 ± 0.78 at the femur neck and -2.5 ± 0.84 at the lumbar spine. Among the participants, 23.4% had experienced fractures and 71.8% were using medication. The prevalence of autoimmune diseases was 34.7%, with coeliac disease being the most common at 22.6%, followed by Hashimoto’s thyroiditis at 6.5%. The CCI in the study population was 2.24 ± 1.28 , indicating a moderate level of comorbidities (Table 1).

Table 1. Main characteristics of participants.

Category	Details	Value(s)
Sample Size	Total participants	124
Age	Mean $\pm$ SD	64.6 ± 10.4 years
BMI	Mean $\pm$ SD	24.8 ± 4.2 kg/m ²
	Underweight	1.7%
	Healthy weight	58.6%
	Overweight	26.7%
	Obesity	12.9%
NLR	Median (IQR)	1.7 (1.26–2.09)
PLR	Median (IQR)	125.1 (96.9–152.7)
BMD T-Score	Femur neck (Mean $\pm$ SD)	-2.3 ± 0.78 SD
	Lumbar spine (Mean $\pm$ SD)	-2.5 ± 0.84 SD
	Osteoporosis	70.2%
	Osteopenia	29.8%
	Fractures	23.4%
Comorbidities	Hypertension	31.1%
	Diabetes	12.2%
	Dyslipidemia	18.9%
	Cancer	5.6%

Table 1. *Cont.*

Category	Details	Value(s)
Pharmacological Therapies	Any therapy	71.8%
	Vitamin D	64.5%
	Calcium	13.6%
	Bisphosphonates	28.2%
	Denosumab	3.6%
	Teriparatide	1.8%
	Raloxifene	0.8%
Autoimmune Diseases	Any autoimmune disease	34.7%
	Coeliac disease	22.6%
	Hashimoto's thyroiditis	6.5%
	Others	5.6%
Number of Autoimmune Diseases	1	81
	2	36
	3	6
	4	1
Chronic Kidney Disease (CKD)	Stage 1	23.6%
	Stage 2	52.8%
	Stage 3a	17.0%
	Stage 3b	4.7%
	Stage 4	0.9%
	Stage 5	0.9%
Thyroid Function	Euthyroidism	89.5%
	Hyperthyroidism	7.9%
	Hypothyroidism	2.6%
Vitamin D Status	Serum levels (Mean $\pm$ SD)	33.3 $\pm$ 13.6
	Normal	55.7%
	Insufficiency	31.1%
	Deficiency	13.1%
Charlson Comorbidity Index (CCI)	Mean $\pm$ SD	2.24 $\pm$ 1.28

No statistically significant direct correlation was observed between the NLR and PLR with any index of bone health, demographic factors, or laboratory results. However, the PLR showed a slight association with vitamin D levels ($\rho = 0.17$, $p < 0.05$) (Table 2). The median values of the NLR and PLR did not differ statistically between individuals with osteoporosis or osteopenia, nor between those with or without fractures ($p > 0.05$).

Table 2. Correlations between NLR/PLR and factors.

Parameter	Correlation with Bone Health	Correlation with Vitamin D	Correlation with Fractures
NLR	Not significant	Not significant	Not significant
PLR	Not significant	$\rho = 0.17$, $p < 0.05$	Not significant

No correlations were found between the NLR, PLR, and bone health, even when stratifying the sample by age quartiles ($p > 0.05$), and there were no differences in PLR and NLR values among fractured subjects based on the type of fracture ($p > 0.05$). Median NLR values were notably higher in women without Hashimoto's thyroiditis compared to those with the condition (1.76, 1.29–2.24 vs. 1.18, 1.09–1.55; $p = 0.028$). Women with diabetes also had higher median NLR values compared to non-diabetic participants (2.14, 1.65–2.5 vs. 1.69, 1.15–2.07; $p = 0.028$). PLR values were elevated in women without hypertension com-

pared to hypertensive individuals (135.76, 99.84–166.15 vs. 101.43, 82.16–131.99; $p = 0.003$). Similarly, euthyroid women had significantly higher PLR values compared to hyperthyroid and hypothyroid participants (124.28, 100.43–148.87 vs. 112.95, 101.08–124.81 vs. 81.40, 66.67–96.30; $p = 0.004$) (Table 3).

Table 3. Comparison of NLR and PLR values in specific groups.

Group Comparison	Values	<i>p</i> -Value
NLR: Women without Hashimoto's vs. with Hashimoto's	1.76 (1.29–2.24) vs. 1.18 (1.09–1.55)	0.028
NLR: Diabetic vs. Non-diabetic women	2.14 (1.65–2.5) vs. 1.69 (1.15–2.07)	0.028
PLR: Women without Hypertension vs. with Hypertension	135.76 (99.84–166.15) vs. 101.43 (82.16–131.99)	0.003
PLR: Euthyroid vs. Hyperthyroid vs. Hypothyroid	124.28 (100.43–148.87) vs. 112.95 (101.08–124.81) vs. 81.40 (66.67–96.30)	0.004

No significant differences were observed between groups based on the presence or absence of other autoimmune diseases, comorbidities, bone-targeting medications, number of autoimmune diseases, vitamin D status, or CKD stage ($p > 0.05$). Similarly, the mean BMD T-score values did not differ significantly regarding the autoimmune status of postmenopausal women ($p > 0.05$). There was also no correlation between the number of comorbidities and NLR or PLR values, and no significant differences in median NLR and PLR values were found when patients were grouped by their total number of comorbidities ($p > 0.05$).

Multiple regression analysis indicated that BMD T-score values at the femur neck could be predicted by a model incorporating factors such as age, BMI, CKD stage, vitamin D levels, NLR and PLR values, diabetes, and autoimmune disease ($R^2 = 0.41$, $p < 0.001$). Notably, the PLR contributed significantly to this prediction ($B = -0.006$, $p = 0.022$) (Table 4). Logistic regression analysis was performed to ascertain the effects of age, BMI, CKD stage, vitamin D levels, the NLR, the PLR, the presence of diabetes, and autoimmune disease on the likelihood that patients have osteoporosis or osteopenia ($R^2 = 0.54$, $p = 0.002$). The model correctly classified 100% of osteoporosis cases, and an increased PLR was associated with an increased likelihood of having osteoporosis ($B = 0.035$, $p = 0.025$) (Table 4).

Table 4. Regression analysis summary.

Model	Predictors	Significant Factor	R^2
Multiple Regression (BMD T-score)	Age, BMI, CKD stage, vitamin D, NLR, PLR, diabetes, autoimmune disease	PLR ($B = -0.006$, $p = 0.022$)	0.41
Logistic Regression (Osteoporosis Likelihood)	Age, BMI, CKD stage, vitamin D, NLR, PLR, diabetes, autoimmune disease	PLR ($B = 0.035$, $p = 0.025$)	0.54

4. Discussion

The results of the study provide further insights into the relationship between systemic inflammation represented by the NLR and PLR and bone health indices. While the connection between chronic inflammation and bone metabolism is well established, this study highlights that neither the NLR nor PLR shows a straightforward correlation with bone health indices, demographic data, or most laboratory findings.

The lack of a significant relationship between the NLR and PLR in our study may be due to the chronic nature of osteoporosis, which affects the bone environment in a similar way to other chronic diseases. Such a chronic inflammatory process may lead to homeostatic adaptations which obscure transient inflammatory markers such as the NLR and PLR. However, relationships between such markers and chronic inflammatory

disease have been reported in previous studies, suggesting that other factors are involved in our study.

An exception is the weak but statistically significant correlation observed between the PLR and vitamin D levels. This indicates a modest relationship between platelet-mediated inflammation and vitamin D status, which is a key factor in bone health.

This finding aligns with the work of Akbas et al., who reported that the PLR and NLR were significantly associated with 25(OH)D levels. They also noted an inverse relationship between vitamin D levels and inflammation. However, this weak correlation alone does not point to a direct effect of the PLR on bone density or fracture risk [10].

4.1. NLR, PLR, and Bone Health Outcomes

There was no significant difference in the median values of the NLR and PLR between individuals with osteoporosis and those with osteopenia, nor between those with and without fractures. Furthermore, stratifying by fracture type revealed no variations in NLR and PLR values, suggesting that the inflammatory response may not significantly differ based on the type of fracture. Multivariate regression analysis identified age, BMI, CKD stage, vitamin D levels, NLR, PLR, diabetes, and autoimmune diseases as significant predictors of BMD at the femur neck. In logistic regression models predicting osteoporosis or osteopenia, systemic factors demonstrated strong predictive power, with an increased PLR linked to a higher likelihood of osteoporosis.

While the PLR contributed significantly to these models, its overall impact was modest, indicating a nuanced role of platelet activity in bone metabolism. This suggests that the PLR might serve as a biomarker for assessing osteoporosis risk, although further research is needed for validation. Lee et al. [11] investigated the relationship between BMD and the NLR and PLR in Korean postmenopausal women and found that the NLR was related to BMD, but the PLR was not. In this study, the sample was bigger and included only postmenopausal women with or without osteoporosis. Moreover, the study was conducted on a more geographically distinct population compared to ours.

A meta-analysis performed by Liu et al. [12] demonstrated that both the NLR and PLR are higher in individuals with osteoporosis in comparison to those with a normal BMD. This suggests associations between these two inflammatory biomarkers and osteoporosis. The difference in the findings of these two studies from ours could be explained by the fact that we did not include women with normal BMDs.

4.2. Autoimmune Diseases and Comorbidities

Autoimmune diseases are known to influence systemic inflammation, which has an impact on hematological inflammatory markers such as the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR). Chronic immune activation in autoimmune diseases leads to persistent cytokine release, e.g., interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and interleukin-1 beta (IL-1 β), that may cause alterations in neutrophil, lymphocyte, and platelet counts [3].

In osteoporosis, systemic inflammation is a key factor in bone resorption. Pro-inflammatory cytokines increase osteoclast differentiation and activity, causing bone loss. As the NLR and PLR reflect systemic immune responses, these markers have been proposed as possible markers of inflammatory activity in autoimmune diseases and disorders of bone metabolism. However, the chronicity of osteoporosis might lead to a steady-state inflammatory profile in which alterations in the NLR and PLR are less pronounced compared to acute inflammatory responses [3–5].

In addition, it is evident that platelets play an active role in the regulation of immune responses by interacting with leukocytes and endothelial cells and influencing inflam-

matory signaling pathways. Such interactions may have an impact on bone homeostasis, particularly in autoimmune-mediated osteoporosis. While some studies reported significant correlations between the PLR and bone diseases, our findings did not confirm such relations, possibly due to the long-term adaptation of immune responses to chronic conditions.

Interestingly, our data highlighted certain subgroup differences. Women without Hashimoto's thyroiditis showed significantly higher NLR values than those with the condition, suggesting that Hashimoto's may suppress systemic neutrophilic inflammation. Hypertension also influenced PLR values, with hypertensive women having a lower PLR compared to non-hypertensive women. Thyroid function had a distinct effect as euthyroid women had significantly higher PLR values than hyperthyroid and hypothyroid women. This hints at a potential connection between thyroid homeostasis and platelet-mediated inflammatory activity. This is supported by the study conducted by Erinc et al., where PLR values were found to be higher in patients with Hashimoto's thyroiditis and non-immunogenic hypothyroidism compared to healthy controls [13].

Future studies should address whether some autoimmune subtypes or levels of disease severity might differently affect the NLR and PLR in patients with osteoporosis. Additionally, considering other inflammatory markers besides the NLR and PLR might provide a clearer picture of immune dysregulation in osteoporotic patients with autoimmune diseases.

4.3. Implications and Future Research

In light of the evidence, it can be concluded that the examined markers are not specific but are influenced by a wide range of conditions and comorbidities. This highlights their sensitivity to various systemic factors rather than serving as exclusive indicators of a single pathological state. This observation is further supported by the heterogeneity of results reported in the current literature on the subject.

These findings suggest that while routine inflammatory markers such as the NLR and PLR may not directly reflect bone health, their association with conditions such as diabetes, hypertension, and thyroid dysfunction could have an indirect effect on bone metabolism. The identification of PLR as a potential predictor of osteoporosis risk introduces a new dimension to the study of systemic inflammation in bone diseases. However, there is currently no evidence to support its use in clinical practice. Future research should focus on longitudinal studies to further elucidate the causal pathways between systemic inflammation and bone health and on the assessment of patient outcomes and prognosis. Additionally, integrating other inflammatory biomarkers and genetic predispositions could enhance the predictive accuracy of models involving the NLR and PLR.

The moderate Charlson Comorbidity Index (CCI) scores we found in our group (2.24 ± 1.28) suggest that our participants are dealing with a significant number of chronic conditions. Conditions like diabetes, cancer, and autoimmune diseases can affect bone health due to systemic inflammation and changes in metabolism, which likely played a role in the prevalence of osteoporosis and the risk of fractures in our population. These results underscore the need to take comorbidity burden into account when assessing and managing osteoporosis. Looking ahead, future research should delve deeper into how the CCI can help predict bone health outcomes and responses to treatment.

4.4. Limitations

This study does come with a few limitations that should be acknowledged. Firstly, the small sample size might have hampered the statistical strength needed to pick up on subtle links between the NLR, PLR, and osteoporosis. Plus, because the study is cross-sectional, we cannot identify any causal relationships or conduct follow-up assessments to see how

the NLR and PLR could act as prognostic indicators or respond to treatment options. What we really need are longitudinal studies to tackle these concerns.

The focus here was mainly on women with osteoporosis and osteopenia, leaving out a control group of healthy women. This makes it tough to draw comparisons between the levels of inflammatory markers across various bone health statuses. On top of that, some participants were not receiving treatment for osteoporosis or osteopenia, which could lead to an inconsistency in the inflammatory profiles observed.

There is also the possibility of selection bias since the study group came from a specific clinical environment, which might limit how broadly we can apply these findings. We also need to consider residual confounding factors like diet, exercise, undiagnosed inflammatory issues, and medication use that were not fully controlled for, meaning they could have affected NLR and PLR levels.

It is important to note that the NLR and PLR are quite non-specific inflammatory markers. They can be influenced by a variety of physiological and pathological factors, such as infections, metabolic disorders, and blood-related issues. The absence of data regarding simultaneous infections, recent surgeries, or medication history could have added yet another layer of variability. A more thorough exploration of inflammatory status, incorporating biomarkers like C-reactive protein (CRP) and interleukin-6 (IL-6), would deepen our understanding of systemic inflammation in osteoporosis.

Finally, we did not perform a receiver operating characteristic (ROC) analysis to determine the optimal cutoff values for the NLR and PLR in relation to bone mineral density (BMD). It is still essential to explore whether certain levels of these biomarkers can be used to predict osteoporosis risk in future research. Even with these limitations, this study provides important insights into the potential role of inflammatory markers in osteoporosis and highlights the need for further investigation in larger, well-controlled prospective studies.

5. Conclusions

In conclusion, we found no evidence from this study that the NLR and PLR independently predict bone health outcomes. However, their integration into a broader clinical context alongside factors such as age, BMI, vitamin D levels, and comorbidities could enhance the understanding and management of bone-related diseases. Future research should focus on validating these markers through longitudinal studies on larger cohorts and exploring their role as components of multifactorial predictive models for bone health.

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