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Original research

Correlation study of multiple inflammatory indices and vertebral compression fracture: A cross-sectional study

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ABSTRACT

Background: Vertebral compression fractures (VCFs) are prevalent in patients with osteoporosis and pose significant health risks. Although chronic low-grade inflammation plays a crucial role in the pathogenesis of osteoporosis, the relationship between various inflammatory indices and the occurrence of fractures remains unclear.

Objective: This study aims to evaluate the correlation between multiple inflammatory indices, neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammatory index (SII), and systemic inflammatory response index (SIRI), and VCFs, to explore the significance of these indices in clinical application.

Methods: Clinical data of 310 patients diagnosed with osteoporosis from November 2020 to June 2023 in the hospital were collected. The general conditions between fracture and non-fracture groups were described. Spearman analysis and binary logistic regression analysis were used to assess the relationship between inflammatory indices and VCFs. Receiver operating characteristic curve was used to evaluate the diagnostic efficacy of these inflammatory indices for VCFs.

Results: VCFs were diagnosed in 43.55 % of patients with osteoporosis. NLR(ρ = 0.169, P=0.003), MLR(ρ = 0.293, P<0.001), SII(ρ = 0.126, P=0.027), and SIRI(ρ = 0.273, P<0.001) were positively correlated with the occurrence of VCFs. NLR(OR=1.480, 95 %CI 1.114 ~ 1.966, P=0.007), MLR(multiplied by 100, OR=1.048, 95 %CI 1.011 ~ 1.087, P=0.011), and SIRI(OR=3.327, 95 %CI 1.510 ~ 7.330, P=0.003) were independent risk factors for VCFs, hip bone mineral density (BMD) (OR=0.011, 95 %CI 0.001 ~ 0.151, P=0.001) was an independent protective factor for VCFs. MLR(AUC 0.671, 95 % CI=0.610 ~ 0.732, P <0.001) had relatively high clinical diagnostic efficacy.

Conclusion: The neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and systemic inflammatory response index (SIRI) are independent risk factors for vertebral compression fractures.

Osteoporosis (OP) is a systemic bone disease characterized by low bone mass and deterioration of bone tissue microarchitecture, leading to increased bone fragility and a heightened risk of fractures. Osteoporotic fractures (OFs), particularly vertebral compression fractures (VCFs), are common complications of osteoporosis. These fractures can occur with minimal or no apparent external force and often develop insidiously. VCFs not only significantly impair patients' quality of life but can also result in physical disability and reduced life expectancy [1]. When a VCF occurs, it may present with symptoms such as low back pain, mobility issues, muscle atrophy, and difficulty performing daily activities. In severe cases, it can lead to spinal deformities, increase the risk of lung infections, and elevate the likelihood of re-fractures in the vertebral body or other areas [2]. Currently, VCFs are diagnosed through lateral thoracolumbar spine X-rays or magnetic resonance imaging (MRI), with diagnostic criteria including a loss of vertebral body height of $\geq$ 20 % or $\geq$ 4 mm from the baseline, along with wedge or "biconcave" deformities [3]. The prevalence of VCFs is high and clinically significant, yet there is limited research on the biomarkers used to diagnose these fractures in patients with osteoporosis.

Low-grade chronic inflammation is a key pathogenic mechanism in the development of osteoporosis [4]. The immune system plays a regulatory role in the skeletal system, as both exist within a shared

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microenvironment [5]. Bone marrow houses hematopoietic stem cells (HSCs), which differentiate into monocytes and macrophages, serving as precursors to osteoclasts, as well as mesenchymal stem cells (MSCs), which give rise to osteoblasts [6]. Persistent inflammation leads to an increased number of peripheral blood monocytes, which can spontaneously differentiate into osteoclasts. This process disrupts the balance between bone formation and resorption, resulting in decreased bone mineral density. Elevated levels of pro-inflammatory factors, such as IL-1, IL-6, and TNF-α, inhibit osteoblast differentiation, promote osteoclast differentiation and activity, and accelerate osteocyte apoptosis [4].

In recent years, several inflammation-related diseases, including chronic obstructive pulmonary disease [7], systemic lupus erythematosus [8], and osteoarthritis [9,10], have been implicated in abnormal bone metabolism, osteoporosis, and increased risk of fragility fractures. Consequently, the inflammatory status has emerged as a predictor of VCFs in patients with osteoporosis. Novel inflammatory indices, such as the neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammatory index (SII), and systemic inflammatory response index (SIRI), are gaining recognition as potential markers for assessing systemic inflammation [11].

In this study, we will examine the relationship between these novel inflammatory indices, inflammatory cell counts, and VCFs in individuals with osteoporosis. We aim to explore the potential application of these markers in the early identification of VCFs, thereby providing a clinical basis for future related research.

Objects and methods

Study subjects

A total of 310 subjects who attended the hospital from November 2020 to June 2023 were selected for this study. All participants signed an informed consent form.

Inclusion criteria: 1) patients with osteoporosis (in this study, we mainly selected men $\geq$ 50 years of age or postmenopausal women).

Exclusion criteria: 1) active infection within 1 month before admission; 2) hematologic diseases; 3) current malignant tumors, undergoing chemotherapy or radiotherapy; 4) severe liver and renal insufficiency.

Methods

Clinical indicators

1) General conditions of the subjects: gender, age, height, weight, history of underlying diseases. 2) Diagnostic results of thoracolumbar spine X-ray lateral radiographs or MRI. 3) Bone mineral density and T-value of lumbar spine and hip (measured by dual-energy X-ray absorptiometry). 4) Blood count: white blood cells, red blood cells, platelets, hemoglobin, neutrophils, lymphocytes, monocytes. Calculate the values of NLR, MLR, PLR, SII, and SIRI. NLR=neutrophil/lymphocyte ratio; MLR=monocyte/lymphocyte ratio; PLR=platelet/lymphocyte ratio; SII=platelet count $\times$ NLR; SIRI=monocyte count $\times$ NLR. 5) Bone metabolism-related indices: parathyroid hormone (PTH), osteocalcin (OC), blood calcium, blood phosphorus, vitamin D, type I procollagen amino-terminal peptide (PINP), type I collagen carboxy-terminal peptide (CTX).

***Diagnostic criteria for osteoporosis** [12]*

According to the Guidelines for the Diagnosis and Treatment of Primary Osteoporosis, osteoporosis is diagnosed in men aged 50 years or older and postmenopausal women when the T-score of either the lumbar spine or hip is $\leq$ -2.5, or when there has been a previous fragility fracture.

***Diagnostic criteria for vertebral compression fracture** [13]*

According to the Diagnosis and Management of Vertebral Compression Fractures, a vertebral compression fracture is defined as a reduction of 20 % or more in the original vertebral height, as determined by lateral X-ray images of the thoracic and lumbar vertebrae. For microfractures that are not clearly visible on X-rays, vertebral MRI may be considered for further evaluation.

Statistical treatment

All data were statistically analyzed using SPSS Statistics 25.0 software. Data from two consecutive groups that conformed to a normal distribution were compared using an independent samples *t*-test and were expressed as mean $\pm$ standard deviation, and those that did not conform to a normal distribution were compared using the rank-sum test and expressed as median (interquartile range). Categorical data were compared using the chi-square test and expressed as n (%). Spearman correlation analysis assessed the relationships among inflammation indices, BMD of the lumbar spine and hip, and VCFs. Binary logistic regression was used to calculate the odds ratio (OR) for VCFs. The efficacy of inflammatory indices in diagnosing VCFs was evaluated by comparing the area under the curve (AUC) using the receiver operating characteristic (ROC) curve. A bilateral P-value of < 0.05 was considered statistically significant.

Results

General condition analysis of fracture group and non-fracture group

A total of 310 osteoporosis patients were included in this study, among which 135 cases had VCFs. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation ($\overline{x} \pm$ sd), and non-normally distributed continuous variables were expressed as median (interquartile range) [M(IQR)]. There were statistical differences between the two groups in terms of age, body mass index, BMD at the 4th lumbar spine and the total hip, blood calcium, neutrophil and monocyte counts, NLR, MLR, SII, and SIRI levels ($P < 0.05$). In the fracture group, the mean levels of age, body mass index, neutrophil and monocyte counts, NLR, MLR, SII, and SIRI were higher compared to the non-fracture group, while the mean levels of BMD at the 4th lumbar spine and hip, and calcium were lower than those in the non-fracture group. However, the gender, the markers of bone metabolism (parathyroid hormone, osteocalcin, type I procollagen amino-terminal peptide, type I collagen carboxy-terminal peptide and vitamin D), and blood phosphorus, PLR did not show significant statistical differences ($P > 0.05$). (Table 1 and Table 2).

Spearman correlation analysis among inflammatory indices, BMD, and VCFs

Spearman correlation analysis of NLR, MLR, PLR, SII, and SIRI with the BMD of the overall lumbar spine and hip, as well as the occurrence of VCFs, revealed that only MLR(ρ = -0.182, P=0.003) had a negative correlation with BMD in the hip. NLR(ρ = 0.169, P=0.003), MLR(ρ = 0.293, P<0.001), SII(ρ = 0.126, P=0.027), and SIRI(ρ = 0.273, P<0.001) were all positively correlated with the occurrence of VCFs. (Table 3).

Binary logistic regression analysis of VCFs

As the occurrence of VCFs was the dependent variable, and age, body mass index, vitamin D, blood calcium, lumbar and hip BMD, NLR, MLR, PLR, SII, and SIRI were the independent variables, to perform binary logistic regression analysis. It revealed that NLR, MLR, SII, and SIRI were risk factors for VCFs with or without correction for age, body mass index, vitamin D, and blood calcium (P<0.05). After correcting for age, body mass index, vitamin D, blood calcium, and BMD of the lumbar

Table 1
Comparison of bone metabolism between fracture and non-fracture groups [$\bar{x} \pm$ sd, M(IQR), n(%)].

	Total (n = 310)	Fracture Group (n = 135)	Non-Fracture Group (n = 175)	t/z/χ2	P
Age(years)	67.38 ± 9.62	70.44 ± 8.81	65.02 ± 9.58	5.108	< 0.001
Female(%)	273 (88.10 %)	114(84.44 %)	159(90.86 %)	2.982	0.084
Body mass index(kg/m^2)	21.91 ± 3.12	22.33 ± 3.09	21.59 ± 3.10	2.074	0.039
L1 BMD	0.672 (0.116)	0.673 (0.137)	0.671 (0.103)	−0.497	0.619
L2 BMD	0.701 (0.130)	0.696 (0.142)	0.703 (0.114)	−0.589	0.556
L3 BMD	0.719 (0.138)	0.715 (0.161)	0.734 (0.129)	−1.873	0.061
L4 BMD	0.735 (0.175)	0.717 (0.184)	0.744 (0.166)	−2.579	0.010
L BMD	0.711 (0.132)	0.694 (0.145)	0.719 (0.121)	−1.508	0.132
Femoral Neck BMD	0.569 ± 0.096	0.538 ± 0.101	0.591 ± 0.087	−4.553	< 0.001
Hip BMD	0.701 ± 0.118	0.668 ± 0.121	0.724 ± 0.111	−3.987	< 0.001
PTH(pg/ml)	43.20 (24.05)	44.40 (28.10)	42.60 (22.50)	−1.130	0.259
OC(ng/ml)	12.09 (8.85)	12.10(9.52)	12.09(8.41)	−0.231	0.817
PINP(ng/ml)	33.50 (30.26)	35.99 (35.42)	31.51 (28.08)	−1.412	0.158
CTX(pg/ml)	263.45 (350.92)	254.00 (322.08)	264.90 (379.30)	−0.862	0.388
VitD(ng/ml)	23.90 (11.26)	22.42 (12.51)	24.82 (10.33)	−1.789	0.074
Calcium (mmol/L)	2.32 ± 0.11	2.31 ± 0.11	2.34 ± 0.11	−2.242	0.026
Phosphorus (mmol/L)	1.14 (0.22)	1.13(0.22)	1.14(0.19)	−0.632	0.527

Table 2
Comparison of inflammatory indices between fracture and non-fracture groups [$\bar{x} \pm$ sd, M(IQR)].

	Total (n = 310)	Fracture Group (n = 135)	Non-Fracture Group (n = 175)	t/z	P
White Blood cell(×10^9/L)	4.94 (1.50)	5.10(1.74)	4.80(1.49)	−1.705	0.088
Red Blood cell (×10^12/L)	4.21 (0.58)	4.19(0.53)	4.23(0.60)	−0.684	0.494
Hemoglobin (g/L)	127.90 ± 13.13	127.90 ± 13.13	129.00 ± 12.77	−0.738	0.461
Platelet (×10^9/L)	199.00 (77.75)	197.00 (71.00)	204.00 (80.5)	−0.208	0.835
Neutrophil (×10^9/L)	3.00 (1.12)	3.15(1.39)	2.89(1.07)	−2.426	0.015
Lymphocyte (×10^9/L)	1.45 (0.68)	1.42(0.73)	1.49(0.64)	−1.385	0.166
Monocyte (×10^9/L)	0.29 (0.14)	0.32(0.16)	0.27(0.11)	−3.700	< 0.001
NLR	2.09 (1.21)	2.32(1.48)	1.97(1.02)	−2.957	0.003
MLR	0.20 (0.10)	0.23(0.12)	0.18(0.08)	−5.129	< 0.001
PLR	138.28 (73.28)	141.13 (79.88)	136.08 (67.86)	−1.081	0.280
SII	402.19 (254.69)	432.73 (295.65)	377.21 (208.97)	−2.212	0.027
SIRI	0.61 (0.45)	0.69(0.44)	0.50(0.43)	−4.771	< 0.001

Table 3
Spearman correlation analysis of various inflammatory indices with BMD of the lumbar spine and hip, and VCFs.

	Lumbar BMD		Hip BMD		VCF	
	ρ	P	ρ	P	ρ	P
NLR	−0.077	0.211	−0.098	0.108	0.169	0.003
MLR	−0.110	0.073	−0.182	0.003	0.293	< 0.001
PLR	−0.110	0.074	−0.111	0.070	0.062	0.280
SII	−0.083	0.179	−0.071	0.244	0.126	0.027
SIRI	−0.052	0.400	−0.099	0.107	0.273	< 0.001

spine and hip, NLR(OR=1.480, 95 %CI 1.114 ~ 1.966, P=0.007), MLR (multiplied by 100, OR=1.048, 95 %CI 1.011 ~ 1.087, P=0.011), and SIRI(OR=3.327, 95 %CI 1.510 ~ 7.330, P=0.003) were independent risk factors for VCFs. Hip BMD(OR=0.011, 95 %CI 0.001 ~ 0.151, P=0.001) was an independent protective factor for VCFs after correcting for age, body mass index, vitamin D, and blood calcium. (Table 4).

Efficacy of inflammatory indices in diagnosing VCFs

ROC curves were plotted based on each inflammation index (NLR, MLR, PLR, SII, SIRI), and the optimal cut-off value, sensitivity, specificity, and youden index were calculated for each index. It revealed that NLR, MLR, SII, and SIRI had clinical values in the diagnosis of VCFs, with MLR having the highest sensitivity (66.4 %) and SII having the highest specificity (79.8 %). The clinical diagnostic efficacy of MLR was relatively high (AUC 0.671, 95 % CI=0.610 ~ 0.732, P < 0.001). (Fig. 1 and Table 5).

Discussion

Vertebral compression fractures(VCFs) are prevalent among patients with osteoporosis, with 43.55 % of individuals in this study diagnosed with such fractures. However, lateral thoracolumbar spine X-rays and MRIs are not routinely performed, and many VCFs present with mild

Table 4
Binary logistic regression analysis of VCFs.

	Univariate		Model 1		Model 2	
	OR (95 % CI)	P	OR (95 % CI)	P	OR (95 % CI)	P
NLR	1.311 (1.075 ~ 1.599)	0.008	1.298 (1.039 ~ 1.621)	0.022	1.480 (1.114 ~ 1.966)	0.007
MLR*	1.067 (1.036 ~ 1.100)	< 0.001	1.048 (1.013 ~ 1.084)	0.006	1.048 (1.011 ~ 1.087)	0.011
PLR	1.002 (0.998 ~ 1.005)	0.305	1.002 (0.998 ~ 1.006)	0.336	1.001 (0.997 ~ 1.006)	0.562
SII	1.001 (1.000 ~ 1.002)	0.020	1.001 (1.000 ~ 1.002)	0.048	1.001 (1.000 ~ 1.002)	0.053
SIRI	3.912 (2.065 ~ 7.411)	< 0.001	2.841 (1.404 ~ 5.752)	0.004	3.327 (1.510 ~ 7.330)	0.003
Lumbar BMD	0.183 (0.017 ~ 1.945)	0.159	0.161 (0.011 ~ 2.333)	0.180	–	
Hip BMD	0.013 (0.001 ~ 0.124)	< 0.001	0.011 (0.001 ~ 0.151)	0.001	–	

Note: Model 1: corrected for age, body mass index, vitamin D, and blood calcium; Model 2: corrected for bone mineral density of the lumbar spine and hip based on Model 1; MLR* = MLR×100. This adjustment was made because the MLR values ranged from 0 to 1, resulting in excessively large odds ratios (ORs) and 95 % confidence intervals (CIs), which led to model instability. Multiplying the MLR values by 100 resolved this issue without affecting the model's construction.

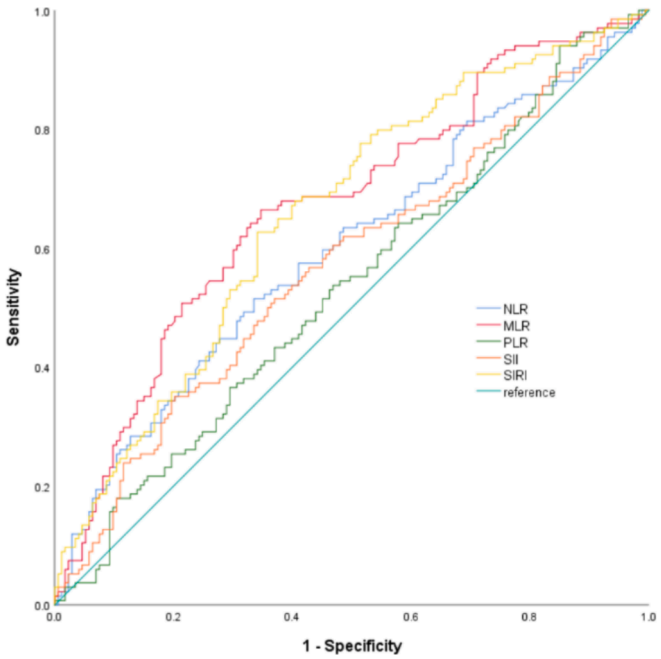

Fig. 1. ROC curves of NLR, MLR, PLR, SII, and SIRI.

symptoms, such as low back pain. This often leads to insufficient attention and a lack of regular anti-osteoporosis treatment. Evidence shows that VCFs can significantly increase the risk of bone loss, further fractures, spinal deformity, cardiopulmonary issues, depression, and even mortality [1]. Therefore, early detection of these fractures is crucial. Routine blood tests, which are simple and cost-effective, can provide insights into systemic inflammation through indicators such as neutrophils, lymphocytes, and monocytes. Excluding acute infections, the neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammatory index (SII), and systemic inflammatory response index (SIRI) derived from these cell counts can reflect the body's overall inflammatory status[14]. Our study found that values of NLR, MLR, SII, and SIRI were closely associated with VCFs, though the exact causal relationships remain unclear.

Chronic low-grade inflammation is a central factor in the development of osteoporosis, as it impairs osteoblast function and accelerates osteoclast formation. This leads to reduced bone formation, increased bone resorption, and an elevated risk of osteoporosis [15]. Markers of inflammation, such as the neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammatory index (SII), and systemic inflammatory response index (SIRI), have shown clinical value in various medical contexts. These indices have been used to assess the severity of autoimmune encephalopathy [16], predict relapse risk in multiple sclerosis [17], and evaluate disease activity in rheumatic polymyalgia [18]. In the context of osteoporosis, inflammatory indices are also significant. Patients with osteoporosis generally exhibit higher levels of NLR, MLR, and PLR compared to individuals with healthy bone [19,20]. Additionally, SII has been found to negatively correlate with BMD in postmenopausal women over 65 years old or those with a normal body mass index of less than 25 kg/m^2 [21].

Age-related osteoporosis is characterized by low bone turnover and increased bone marrow fat accumulation. Studies in aging rats and mice have shown that neutrophils and monocyte-macrophages progressively accumulate in the bone marrow. These cells secrete high levels of granular calreticulin, which binds to the plexin-b2 receptor, partially inactivating the downstream signaling pathway. This process inhibits osteogenesis and promotes adipogenesis in bone marrow mesenchymal stem cells [22]. With aging, the immune system undergoes a decline known as "immune senescence." Neutrophils, the most abundant leukocytes in the innate immune system, become dysfunctional in older individuals. Despite this, their relative abundance increases due to diminished apoptotic pathways. Senescent neutrophils exhibit impaired chemotaxis and exocytosis, leading to reduced mobilization to inflammation sites. As a result, these cells increasingly contribute to bone resorption. This occurs through the stimulation of reactive oxygen species production and the release of large quantities of pro-inflammatory factors, such as IL-8 and IL-17. These factors induce osteoclast generation and activation while causing osteoblast retraction and bone exposure, ultimately resulting in bone resorption and osteoporosis [23]. Monocytes, which are precursors to osteoclasts, also play a role in bone loss. Elevated levels of peripheral blood monocytes can spontaneously differentiate into osteoclasts and secrete pro-inflammatory factors like IL-1, IL-6, and TNF-α, further accelerating bone loss [4]. Lymphocytes, which include T and B cells, are crucial for bone protection [23,24]. T lymphocytes, activated by macrophages through antigen presentation, inhibit osteoclast formation, maturation, and activation mainly through the expression of osteoprotegerin (OPG) and the secretion of cytokines such as IFN-g, IL-4, IL-12, and IL-18. This helps to prevent age-related osteoporosis [24]. Mature B lymphocytes also express OPG to maintain bone homeostasis. However, an inflammatory environment can alter their role, leading B lymphocytes to promote bone resorption by stimulating osteoclast precursor cell proliferation and activation through the secretion of granulocyte colony-stimulating factors (G-CSFs) and receptor activator of nuclear factor κB ligand (RANKL). Regulatory B lymphocytes (Bregs) can promote osteogenic differentiation by secreting anti-inflammatory cytokines like IL-10 and TGF-β1 [23]. Our study aligns with these findings, showing that increased levels of NLR, MLR, SII, and SIRI correlate with elevated neutrophil and monocyte counts and decreased lymphocyte counts in the blood. These changes contribute to greater bone destruction and an increased risk of VCFs. After adjusting for factors such as age, body mass index, vitamin D and calcium levels, and lumbar spine and hip BMD, NLR, MLR, and SIRI were identified as independent risk factors for VCFs. Among these, MLR demonstrated the highest diagnostic capacity with an area under the curve (AUC) of 0.671.

One study has identified the NLR as a risk factor for osteoporotic femoral neck fractures, with higher NLR values correlating with an increased risk of severe fractures [25]. Similarly, another study found that the SIRI was negatively correlated with BMD at the lumbar spine and femoral neck, and positively correlated with the risk of osteoporotic fractures and hip fractures in elderly hypertensive patients over 60 years of age [26]. In patients with osteoporotic fractures who required hospitalization or surgical treatment, baseline SIRI was negatively correlated with bone metabolism markers such as PINP and CTX. Each 1-unit

Table 5
ROC curves of NLR, MLR, PLR, SII, and SIRI.

	AUC	95 %CI	P	Optimal cut-off value	Sensitivity (%)	Specificity (%)	Youden Index
NLR	0.598	0.534 ~ 0.663	0.003	2.307	51.5	66.5	0.180
MLR	0.671	0.610 ~ 0.732	< 0.001	0.201	66.4	65.3	0.317
PLR	0.536	0.471 ~ 0.601	0.279	82.361	94.0	15.0	0.090
SII	0.574	0.509 ~ 0.639	0.027	541.894	35.1	79.8	0.149
SIRI	0.659	0.598 ~ 0.720	< 0.001	0.632	62.7	65.9	0.286

increase in SIRI corresponded to a decrease of 3.77 in PINP values and 0.04 in CTX values, suggesting a link between systemic inflammation and diminished bone metabolism [27]. Byung-Wook Song et al. discovered that NLR, PLR, and MLR could serve as potential markers of systemic bone loss in rheumatoid arthritis patients. Elevated baseline NLR was significantly associated with osteoporosis at various sites. During follow-up, 12.8 % of patients with rheumatoid arthritis experienced non-traumatic vertebral fractures. Multivariate Cox regression analysis indicated that high baseline NLR, PLR, and MLR were independently linked to an increased risk of vertebral fractures [28]. Although there is limited research on the relationship between inflammatory indices, particularly MLR, and VCFs, our study found that MLR demonstrated considerable clinical value in diagnosing VCFs. This suggests that MLR could be a reliable biomarker for VCFs.

The present study also found that hip BMD was an independent protective factor against VCFs. While the BMD of the lumbar spine was lower in the fracture group compared to the non-fracture group, it was not identified as a protective factor for VCFs. This discrepancy might be attributed to the fact that the osteoporotic patients included in the study primarily exhibited bone loss in the lumbar spine, leading to greater interindividual variation in hip BMD.

This study is a single-center case-control study focused primarily on postmenopausal women, with no inclusion of a healthy control group, which presents some limitations. In the future, we plan to expand the sample size to include more younger individuals with osteoporosis, as well as men and healthy controls. This will allow us to analyze whether there are significant differences in NLR, MLR, PLR, SII, and SIRI between those with VCFs and those without in the general population. This broader analysis will help to further elucidate the role of inflammation in bone health and provide a stronger clinical foundation for subsequent related studies.

Conclusion

Neutrophil/lymphocyte ratio (NLR), monocyte/lymphocyte ratio (MLR), and systemic inflammatory response index (SIRI) are independent risk factors for vertebral compression fractures, and the diagnostic capacity of MLR was relatively high.

Author contribution

Qi Fu: experimental designation, implementation of this study, data collection and analyzation, writing the original draft. **Cuiping Zhang**: data collection and analyzation, critical review of the intellectual content of this draft. **Yujiao Yang**: data analyzation, critical review of the intellectual content of this draft, instruction in the use of statistical software. **Ruoling Teng, Fenfen Liu, Ping Liu**: data collection, critical review of the intellectual content of this draft. **Long Wang, Jiao Wang, Yanan Chen**: critical review of the intellectual content of this draft. **Yi Ding**: the corresponding author, methodological guidance, supervision, critical review, funding acquisition.

Trial registration

The study protocol was approved by the Ethics Committee of The First People's Hospital of Changzhou (Clinical Trial Registration No. (2022) Ke No. 078). Written, informed consent was obtained from all individual participants included in the study.

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CRediT authorship contribution statement

Qi Fu: Conceptualization, Data curation, Formal analysis, Methodology, Writing – original draft, Writing – review & editing. **Cuiping Zhang:** Writing – review & editing, Formal analysis, Data curation. **Yujiao Yang:** Writing – review & editing, Software, Investigation. **Ruoling Teng:** Writing – review & editing, Investigation, Data curation. **Fenfen Liu:** Writing – review & editing, Investigation, Data curation. **Ping Liu:** Writing – review & editing, Investigation, Data curation. **Long Wang:** Writing – review & editing, Investigation. **Jiao Wang:** Writing – review & editing, Investigation. **Yanan Chen:** Writing – review & editing, Investigation. **Yi Ding:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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