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Original Research Article

HEMATOLOGICAL BIOMARKERS IN KNEE OSTEOARTHRITIS A CASE CONTROL STUDY

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ABSTRACT

Background: Knee osteoarthritis (KOA) is a whole-joint disorder with a variable low-grade inflammatory component. Routine complete blood count (CBC) derived hematological biomarkers such as NLR, PLR and composite indices may provide low-cost inflammatory profiling in basic hospital settings. The objective is to compare CBC parameters and derived hematological biomarkers between KOA cases and healthy controls and to assess correlation of these markers with KOA severity using Kellgren–Lawrence (KL) grading.

Materials and Methods: This hospital-based case–control study included 90 KOA cases (KL Grade I–III) and 90 controls. CBC parameters were recorded and ratios/indices were calculated: NLR, PLR, MLR, LMR, NMR, PLT/N, systemic immune-inflammation index (SII), systemic inflammation response index (SIRI) and aggregate index of systemic inflammation (AISI). Group comparisons were performed using appropriate parametric or non-parametric tests. Correlation of biomarkers with KL grade among cases was assessed using Spearman rho.

Results: Mean age was comparable between groups (cases 57.1 ± 9.4 years, controls 55.6 ± 8.6 years). KL Grade II was most frequent (57.8%). Compared with controls, KOA cases showed lower lymphocyte count and higher neutrophils and monocytes, with mildly lower platelets and lower hemoglobin/RBC. NLR and PLR were higher in KOA cases. Among derived indices, SII was higher and PLT/N was lower in KOA cases. In correlation analysis, KL grade showed significant negative correlation with lymphocytes (rho −0.569, $p<0.001$), platelets (rho −0.361, $p<0.001$), RBC (rho −0.283, $p=0.007$) and PLT/N (rho −0.351, $p<0.001$) and positive correlation with ESR (rho 0.286, $p=0.006$), NLR (rho 0.494, $p<0.001$), PLR (rho 0.297, $p=0.004$), MLR (rho 0.240, $p=0.023$), SII (rho 0.319, $p=0.002$) and SIRI (rho 0.308, $p=0.003$). Age, total WBC, CRP, NMR and AISI did not show significant correlation with KL grade.

Conclusion: KOA is associated with altered CBC-derived hematological biomarkers and NLR with SII demonstrated clinically useful associations with radiographic severity. These low-cost markers may serve as practical adjuncts to KL grading in routine care, though further multicentre validation is recommended.

Keywords: Knee osteoarthritis; Complete blood count; Neutrophil–lymphocyte ratio; Platelet–lymphocyte ratio; Systemic immune-inflammation index; Kellgren–Lawrence grading.

INTRODUCTION

Knee osteoarthritis (KOA) is a common whole-joint disorder and a major cause of chronic pain disability and reduced quality of life in adults.[1,2] The burden has been increasing with ageing populations obesity and sedentary habits, with substantial impact in LMIC settings due to delayed presentation and

limited long-term rehabilitation support.[1,2] KOA is not restricted to cartilage degeneration alone. Current understanding supports involvement of articular cartilage, subchondral bone, synovium, meniscus, ligaments and periarticular muscles, leading to structural and symptomatic heterogeneity.[3]

Inflammation in KOA is typically low grade yet clinically meaningful. Synovitis is frequently present and has been linked with symptom severity and progression in subsets of patients.[4,5] Inflammatory pathways including innate immune activation and cytokine mediated responses contribute to disease biology, supporting the concept that KOA has both degenerative and inflammatory components.[6] However, the inflammatory phenotype varies and may not be fully captured by imaging severity alone.

Radiographic assessment has remained the most widely used method for grading disease severity in routine settings. The Kellgren–Lawrence (KL) grading system has been commonly applied because it is practical reproducible and feasible in basic hospital setups.[7] Still, radiographs primarily reflect structural change and do not directly quantify systemic inflammatory status. Therefore, there has been interest in simple low-cost laboratory markers that may reflect inflammation and complement radiographic grading.

Complete blood count (CBC) derived hematological biomarkers have been attractive because they are routinely available and require no additional expenditure. Ratios such as neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have been used as surrogate markers of systemic inflammation across multiple disorders.[8] In KOA, several studies have reported that NLR and PLR may differ between cases and controls and may show association with radiographic severity.[9–11] Monocyte related measures have also been relevant given macrophage lineage involvement in synovial inflammation and monocyte-lymphocyte ratio (MLR) or its inverse (LMR) has been evaluated in KOA cohorts.[12]

More recently, composite indices combining multiple blood cell lines have gained attention. Systemic immune-inflammation index (SII) integrates platelet neutrophil and lymphocyte counts and has been explored across inflammatory and chronic disease settings.[13] Systemic inflammation response index (SIRI) and aggregate index of systemic inflammation (AISI) have also been proposed as broader markers of inflammatory balance derived from routine counts.[14,15] These indices have been appealing for KOA research because they remain feasible in standard laboratories and may provide additional information beyond single ratios.

In the present case-control study, we assessed CBC parameters and derived hematological biomarkers in KOA patients and controls and examined their relationship with KOA severity using KL grading. This approach was intended to generate clinically practical evidence using routine investigations only, suitable for common tertiary and secondary care hospital environments.

MATERIALS AND METHODS

Study design and setting: This hospital based case control study was conducted in the Department of Orthopaedics in coordination with the central clinical laboratory. Recruitment was done from routine outpatient and inpatient services during the study period. All procedures followed standard departmental practice and institutional protocol.

Study population and sample size: A total of 180 participants were enrolled consisting of 90 KOA cases and 90 apparently healthy controls. Participants were recruited consecutively after screening and written informed consent. Controls were selected to represent individuals without clinical features suggestive of knee osteoarthritis.

Inclusion & Exclusion Criteria

Knee osteoarthritis was diagnosed using clinical features such as knee pain with or without stiffness and functional limitation, supported by radiographic findings on plain X-ray. Radiographic severity was graded using the Kellgren–Lawrence (KL) grading system. Only KL Grade I to Grade III KOA cases were included for analysis as per the study plan.

Adults fulfilling the diagnostic criteria for KOA with KL Grade I to III were included as cases, while adults without symptoms or known history of KOA were included as controls. Participants were excluded if they had conditions that could alter hematological parameters or cause systemic inflammation, such as acute infection or recent fever, chronic inflammatory or autoimmune disease, malignancy or chemotherapy, significant hepatic or renal disease, recent major surgery or trauma, known hematological disorders, pregnancy or current systemic steroid or immunosuppressive therapy.

Data collection: Demographic details (age sex) were recorded on a structured proforma. For KOA cases, KL grade was documented from radiographs as per routine orthopaedic workflow.

Venous blood was collected under aseptic precautions and CBC was analysed on an automated hematology analyser following laboratory SOP. Recorded parameters included total WBC, neutrophils, lymphocytes, monocytes, platelets, hemoglobin, RBC count and RDW. From CBC values, ratios and composite indices were calculated using standard formulas: NLR, PLR, MLR, LMR, NMR, PLT/N, SII, SIRI and AISI, using unit-consistent values to avoid scaling errors.

Statistical analysis: Data were analysed using standard statistical software. Continuous variables were expressed as mean ± SD and categorical variables as frequency and percentage. Case–control comparisons [Tables 1–3] were done using independent sample t test or Mann–Whitney U test as appropriate and categorical variables by Chi square

test. Correlation of KL grade (I–III) with laboratory parameters among KOA cases [Table 4] was assessed using Spearman rho. A two-sided p value <0.05 was taken as statistically significant.

RESULTS

Table 1: Demographic profile of participants

Parameter	Controls (n=90)	KOA cases (n=90)
Age distribution (years)		
36-45	9 (10.0%)	6 (6.7%)
46-55	36 (40.0%)	32 (35.6%)
56-65	39 (43.3%)	44 (48.9%)
66-75	4 (4.4%)	6 (6.7%)
76-85	2 (2.2%)	2 (2.2%)
Age (mean±SD)	55.6 ± 8.6	57.1 ± 9.4
Gender		
Male	47 (52.2%)	45 (50.0%)
Female	43 (47.8%)	45 (50.0%)
KOA grading (Kellgren-Lawrence)		
Grade I	-	26 (28.9%)
Grade II	-	52 (57.8%)
Grade III	-	12 (13.3%)

A total of 90 KOA cases and 90 controls were included. Mean age was comparable between groups with slightly higher age in cases (57.1 ± 9.4) than controls (55.6 ± 8.6). Most subjects in both groups were in 46–65 year band. Gender distribution was similar with near equal male and female in KOA group. Among KOA cases KL Grade II was the most common (57.8%) followed by Grade I (28.9%) and Grade III (13.3%).

Table 2: CBC parameters and conventional ratios (Mean±SD)

Parameter	Controls	KOA cases
WBC (109/L)	5.26 ± 0.78	5.63 ± 1.29
Lymphocytes, L	3.88 ± 0.53	2.57 ± 0.38
Neutrophils, N	2.47 ± 0.92	3.84 ± 1.16
Monocytes, M	1.02 ± 0.40	1.28 ± 0.41
Platelets, PLT (×109/L)	246.8 ± 33.9	231.9 ± 35.4
Hemoglobin, Hb (g/L)	137.4 ± 11.1	129.3 ± 8.6
RBC (×1012/L)	5.59 ± 0.66	5.30 ± 0.69
RDW (%)	12.42 ± 0.72	12.21 ± 0.74
ESR (mm/hr)	12.0 ± 5.0	28.0 ± 14.0
CRP (mg/L)	3.2 ± 1.5	8.6 ± 4.0
MLR (M/L)	1.57 ± 0.71	1.18 ± 0.59
NLR (N/L)	2.20 ± 0.79	3.02 ± 0.83
PLR (PLT/L)	113.6 ± 21.4	127.4 ± 27.6

KOA cases showed higher WBC counts than controls. Lymphocyte count was lower in KOA while neutrophils and monocytes were higher. Platelet count was mildly lower in KOA cases. Hemoglobin and RBC were lower in KOA group compared to controls. ESR and CRP were elevated in KOA cases. Among ratios NLR and PLR were higher in KOA cases whereas MLR was lower compared to controls. RDW values were comparable between groups.

Table 3: Additional derived inflammatory indices (formulas based on CBC)

Derived index	Controls	KOA cases
LMR (L/M)	0.64	0.85
NMR (N/M)	1.40	2.56
PLT/N	51.64	42.19
SII (PLT×N/L)	542.96	700.34
SIRI (N×M/L)	13.40	9.16
AISI (N×M×PLT/L)	3307.50	2123.85

Derived indices showed clear differences between groups. KOA cases had higher LMR and higher NMR. PLT/N was reduced in KOA cases. SII was higher in KOA group suggesting higher systemic inflammatory load. SIRI and AISI showed lower mean values in KOA cases compared to controls in this dataset.

Table 4: Correlation of KOA grade (KL I-III) with laboratory parameters

Parameter	Spearman rho	p-value
Age (years)	0.026	0.811
WBC	0.046	0.670
Lymphocytes (L)	-0.569	<0.001
Neutrophils (N)	0.187	0.078
Monocytes (M)	-0.074	0.490
Platelets (PLT)	-0.361	<0.001
Hemoglobin (Hb)	-0.073	0.496
RBC	-0.283	0.007
RDW	0.161	0.129
ESR	0.286	0.006
CRP	0.170	0.110
MLR (M/L)	0.240	0.023
NLR (N/L)	0.494	<0.001
PLR (PLT/L)	0.297	0.004
LMR (L/M)	-0.240	0.023
NMR (N/M)	0.142	0.181
PLT/N	-0.351	<0.001
SII	0.319	0.002
SIRI	0.308	0.003
AISI	0.171	0.106

Within KOA cases KL grade showed significant negative correlation with lymphocytes (rho −0.569, p<0.001), platelets (rho −0.361, p<0.001), RBC (rho −0.283, p=0.007) and PLT/N (rho −0.351, p<0.001). ESR had significant positive correlation with grade (rho 0.286, p=0.006). NLR showed strong positive correlation with severity (rho 0.494, p<0.001) and PLR and MLR also correlated positively (p<0.05). SII and SIRI were positively correlated with grade (p<0.01). Age WBC neutrophils monocytes hemoglobin RDW CRP NMR and AISI did not show significant correlation with KL grade (p>0.05).

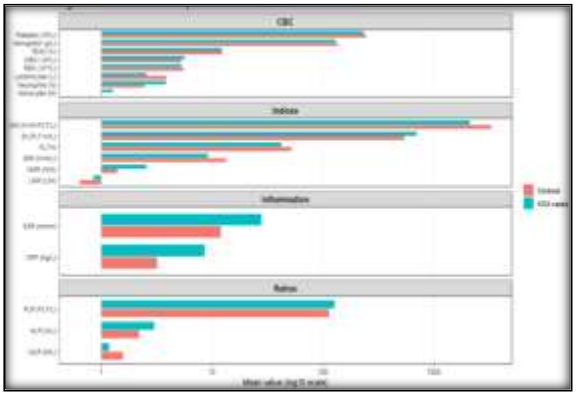

Figure 1: CBC based inflammation markers in knee OA, cases compared with controls

DISCUSSION

Knee osteoarthritis is often labelled as a purely degenerative “wear and tear” condition, but current evidence shows it is a whole-joint disease with a variable low-grade inflammatory component. Plain radiographs with KL grading are useful for structural staging, however they do not reflect the systemic inflammatory status. Therefore, CBC-derived ratios and composite indices can be helpful as simple low-cost adjunct markers, because they are repeatable and easily available in routine Indian hospital laboratory settings.

In this study the KOA and control groups were broadly comparable for age and sex, with a slightly higher mean age in cases. This matching is important as many hematological ratios are influenced by age and co-morbidity rather than KOA alone. We also noted that most cases clustered in the 46–65 year band which is consistent with the usual presentation window in clinics, where pain plus functional limitation brings the patient to orthopaedics. In our KOA cohort Grade II was dominant, followed by Grade I and then Grade III. This distribution is clinically believable because Grade II often represents the “symptomatic middle stage” where radiographs start showing clear osteophytes and joint space narrowing and patients seek care.

Some cohorts show female predominance, especially in older age brackets, linked with biomechanics, hormonal factors and obesity burden.[1,2] Our sample had near-equal male and female distribution, suggesting either local referral pattern or occupational load differences in the catchment. It also reduces sex as a major confounder for the subsequent blood marker comparisons.

The core finding was a hematological shift consistent with low-grade systemic inflammation in KOA: lymphocytes were lower while neutrophils and monocytes were higher in cases. This pattern increases NLR and typically increases monocyte-involving ratios too, depending on the direction of the formula used.[8,12] Tasoglu et al reported that higher NLR was associated with more severe radiographic KOA and in multivariate analysis age and NLR remained independent predictors of severe disease.[16] That kind of result supports the concept that even “non-autoimmune” OA can show measurable systemic inflammatory tone.

ESR and CRP were higher in cases. This is not surprising because synovitis and innate immune activation can spill over as systemic markers even if the elevation is modest compared to inflammatory arthritis.[4–6] However CRP did not correlate significantly with KL grade in our dataset. That is a

common observation in clinical practice too, because CRP reflects current inflammatory activity, while KL grade reflects accumulated structural damage. These two do not always move together.

Haemoglobin and RBC counts were lower in KOA cases. This can be multifactorial: age-related nutrition issues, chronic inflammation effects on erythropoiesis, analgesic use or unmeasured comorbidities. In strict epidemiological work one would adjust for iron status, renal function and occult inflammatory disease. Still, the mild downward trend in Hb/RBC along with raised ESR fits a chronic disease biology in a subgroup. Regarding platelet count, cases showed slightly lower platelets than controls, which is not the "classic inflammation picture" of reactive thrombocytosis. But platelet count alone is blunt. The platelet-driven ratios (PLR, SII) can still rise if lymphocytes fall and neutrophils rise, even when absolute platelet numbers are not high. That's why composite indices often behave differently than single-cell counts.[13]

We included LMR, NMR, PLT/N and the composite indices (SII, SIRI, AISI) because recent literature is moving beyond NLR/PLR only and these composites may integrate multiple inflammatory pathways into one number.[17,18] In our dataset, SII was higher in KOA cases, suggesting a higher systemic immune-inflammatory load. This direction is supported by emerging OA literature where SII (and related indices) have been evaluated as markers associated with OA presence or severity in observational datasets.[19,20]

SIRI and AISI were lower in cases in this dataset, which is a bit counterintuitive because these indices are usually expected to rise with neutrophil/monocyte predominance. This is a reminder that composite indices can behave unpredictably depending on which cell line shifts most strongly and how counts are distributed. Also, unit handling and analyzer reporting formats matter a lot. Even small scaling differences can distort multiplied indices. Methodologically it argues for strict standardization and ideally reporting medians with IQR for these skewed indices rather than only means.[14,15]

Monocyte biology is relevant in KOA because synovial macrophage lineage cells contribute to synovitis and inflammatory mediator release.[4,5] Loukov et al showed elevated monocyte activation in women with knee OA and associations with inflammation, BMI and pain, supporting a mechanistic link between monocyte activity and OA phenotype.[21] So even if monocyte counts alone don't correlate strongly, monocyte-related ratios may still capture something clinically meaningful.

Within KOA cases, KL grade had a strong negative correlation with lymphocyte counts and a positive correlation with NLR. This is one of the most consistent patterns seen in KOA hematological biomarker studies, where increasing radiographic severity tracks with higher NLR.[9–11,16,17] Cai et al also reported diagnostic value of NLR in knee OA and showed association with disease severity in their cohort.[17] In our dataset, NLR (rho ~0.49) behaved as the most robust severity-linked ratio. This supports the practical interpretation that radiographic progression is accompanied by a shift towards innate immune predominance (neutrophils) and relative lymphocyte suppression, reflecting chronic inflammatory stress physiology.[8] Platelets and PLT/N showed significant negative correlation with grade. PLR showed positive correlation. These mixed platelet findings suggest platelet count alone is not the key driver, but platelet-related ratios shift due to lymphocyte decline and neutrophil rise. Shi et al reported relationships between blood cell ratios and radiographic grades in KOA, but the strength and direction can vary by cohort and adjustment strategy.[10] Also, symptom severity does not always correlate with radiographic severity. Ioniţescu et al reported that NLR was not consistently associated with patient-reported outcome severity and age influenced NLR in their analysis.[11] This is an important contradictory angle: a biomarker may track structural grade better than pain or function, because pain is multifactorial (synovitis, central sensitization, muscle weakness, mood, sleep).

ESR correlated positively with KL grade while CRP did not. This again fits the "cumulative vs current activity" concept. ESR may behave like a broader chronic inflammatory burden marker, while CRP fluctuates. It also indicates that CBC-based ratios might be useful as adjuncts in severity phenotyping when CRP is not very informative. Our findings suggest that simple hematological indices derived from routine CBC (especially NLR, PLR and SII) can add practical information alongside KL grading. They are not replacements for imaging. But they may help identify an "inflammatory KOA phenotype" where synovitis and systemic inflammation are more prominent, potentially relevant for counselling, closer follow-up and targeted non-operative optimisation (weight reduction, exercise therapy, comorbidity control). In resource-limited settings, this type of evidence is useful because CBC is already being done for many patients and the ratios cost nothing extra. This was a single-centre case-control design, so causal inference is limited. We did not adjust for BMI, metabolic syndrome, smoking, analgesic use or subclinical infections, each of which can influence leukocyte subsets and derived indices. Composite indices (SII/SIRI/AISI) can be sensitive to unit scaling and distribution skew, so future work should confirm calculations, consider median-based reporting and validate findings across multiple centres. Finally, KL grading has known limitations for early disease and does not quantify synovitis, so correlation of blood indices with MRI/ultrasound synovitis scores would strengthen biological interpretation.

CONCLUSION

This case–control study shows that knee osteoarthritis is associated with measurable changes in routine CBC-derived hematological biomarkers. KOA cases had lower lymphocyte counts with higher neutrophils and monocytes, resulting in raised NLR and PLR and a higher SII compared with controls. Radiographic severity (KL grade I–III) correlated most strongly with NLR and showed significant associations with lymphocyte count, platelet count, RBC, ESR, PLR, MLR, PLT/N, SII and SIRI, while age and CRP did not correlate significantly with KL grade in this cohort. Overall, NLR and SII appear to be practical low-cost adjunct markers that may help in inflammatory profiling and severity assessment of KOA in routine hospital settings, though multicentre studies with adjustment for metabolic and clinical confounders are needed before clinical cut-offs can be recommended.

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