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NEUTROPHIL TO LYMPHOCYTE RATIO (NLR), PLATELET TO LYMPHOCYTE RATIO (PLR), MONOCYTE TO LYMPHOCYTE RATIO (MLR) IN BIPOLAR DISORDER.

Pathology

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ABSTRACT

Bipolar affective disorder (BD) is a chronic, recurrent psychiatric illness associated with significant morbidity and premature mortality. Emerging evidence implicates systemic inflammation in its pathophysiology. **Aim:** To study and compare NLR,PLR,MLR in patients of bipolar disorder and healthy subjects. **Methods:** This hospital-based prospective study was conducted at the Department of Pathology and Psychiatry, Government Medical College, Amritsar, from June 2024 to December 2025. Fifty patients (18–62 years) diagnosed with bipolar affective disorder per ICD-10 criteria were enrolled alongside 50 age- and gender-matched healthy controls. **Results:** All three inflammatory ratios were significantly elevated in cases: NLR (4.43 ± 3.12 vs. 2.41 ± 1.27; p = 0.0001), PLR (158.88 ± 101.20 vs. 104.71 ± 53.51; p = 0.0012), and MLR (0.26 ± 0.20 vs. 0.18 ± 0.10; p = 0.01). Severity-wise analysis revealed a progressive and statistically significant escalation across mild, moderate, and severe groups for NLR (2.28 → 3.17 → 7.52; p < 0.0001), PLR (109.17 → 135.26 → 224.12; p = 0.001), and MLR (0.17 → 0.22 → 0.39; p = 0.004. **Conclusion:** NLR, PLR, and MLR are significantly elevated in bipolar affective disorder and demonstrate a progressive increase with disease severity.

KEYWORDS

Bipolar Disorder, NLR, PLR, MLR, CBC.

INTRODUCTION

Bipolar disorder (BD) is a serious and enduring mental illness marked by emotional disturbances manifesting as mood phases of (hypo)mania or depression, alongside alterations in physical activity.[1] Globally, 2–3% of the general population is impacted by BD, with the average onset occurring at 20 years of age.[2] As a prominent contributor to global disability, individuals with BD exhibit elevated levels of premature mortality attributed to heightened suicide rates, together with increased comorbidity rates associated with cardiovascular disease, diabetes mellitus, and chronic obstructive pulmonary disease (COPD).[3,4]

According to the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), bipolar disorder (BD) is classified under "Bipolar and Related Disorders" and comprises three main subtypes: BD type I, BD type II, and cyclothymic disorder. Individuals with BD type I are defined by the occurrence of at least one manic episode, which involves a persistently elevated or irritable mood associated with marked functional impairment or the presence of psychotic features, lasting for a minimum of seven days, and may occur with or without episodes of major depression. In contrast, BD type II is characterized by at least one hypomanic episode—featuring elevated or irritable mood with associated symptoms and mild to moderate functional changes lasting at least four days—along with at least one major depressive episode.[5]

Diagnosis relies heavily on comprehensive clinical assessment, as no specific laboratory findings or biomarkers exist.[1] Biomarkers play a pivotal role in unraveling disease mechanisms, identifying novel treatment targets, and gauging therapeutic response. The identification of pathophysiological biomarkers in bipolar disorder holds potential for advancing of innovative molecular interventions.[6] Inflammation emerges as one of several etiopathological mechanisms implicated in bipolar disorder.

Among proposed inflammatory markers, the neutrophil-lymphocyte ratio (NLR), monocyte-lymphocyte ratio (MLR), and platelet-lymphocyte ratio (PLR) have garnered attention.[7] These biomarkers, including NLR, MLR, and PLR, offer advantages in terms of accessibility, affordability, and widespread utility.

The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) stand out as straightforward, dependable, as well as economical markers indicative of systemic inflammatory response, conveniently assessed through a hemogram. Higher NLR levels have been linked to poor prognoses in various conditions including malignancies, pancreatitis, and coronary heart disease.[8] Both NLR and PLR have been explored in the context of different psychiatric disorders including bipolar disorder (BD) as well as schizophrenia, encompassing manic and euthymic phases.

Another emerging marker, the monocyte-to-lymphocyte ratio (MLR), presents as a relatively recent addition to this line of inquiry, also ascertainable via CBC. MLR has been posited as a surrogate indicator of inflammation across specific patient populations, exhibiting prognostic and predictive value in diverse disorders including hematological conditions, coronary disease, and malignancies.[9] Notably, elevated monocyte counts or diminished lymphocyte counts have been correlated with adverse prognoses in these disorders. However, investigations into the usefulness of MLR within the field of neuropsychiatric disorders are notably lacking.

Despite these findings, there remains a scarcity of research in this area, particularly in regions such as Punjab. Therefore, further studies are needed to better understand the association between bipolar disorder and neuroinflammatory processes in this population.

This study aims to analyse the Platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and neutrophil-to-lymphocyte ratio (NLR) in individuals diagnosed with bipolar disorder.

METHODOLOGY

This hospital-based prospective study was conducted following institutional ethical committee approval at the Department of Pathology and Psychiatry, Government Medical College, Amritsar, from June 2024 to December 2025.

Study Population

Fifty patients (18–62 years) diagnosed with bipolar affective disorder per ICD-10 criteria, attending the Psychiatry OPD/IPD, were enrolled alongside 50 age- and gender-matched healthy controls. Written informed consent was obtained from all participants.

Patients were excluded if they were on antipsychotics, anti-inflammatory, or immunosuppressive drugs in the preceding six months; had heavy smoking, alcohol use, or substance abuse; had

comorbid psychiatric or systemic conditions (endocrinological, autoimmune, hepatic, renal, cardiac, or active infections); had abnormal leukocyte counts; or had a BMI ≥30 kg/m².

Controls completed the Mood Disorder Questionnaire and Major Depression Inventory and had no personal psychiatric history.

Data Collection and Laboratory Analysis

Sociodemographic and clinical data were retrieved from hospital records. Blood samples were collected prior to any medication via EDTA-anticoagulated tubes and analyzed within two hours. Controls were sampled between 7:30–9:00 a.m. to minimize circadian variation. A 5-part differential hematology analyzer using volumetric impedance and optical light scattering was employed. NLR, PLR, and MLR were calculated as:

NLR = Neutrophil / Lymphocyte count
PLR = Platelet / Lymphocyte count
MLR = Monocyte / Lymphocyte count

Manic and depressive episodes were assessed using the Young Mania Rating Scale and Major Depression Inventory, respectively.

Ethical Approval

Ethical clearance was obtained from the Institutional Ethics Committee, and written informed consent was obtained from all participants before enrolment. Patient confidentiality was maintained throughout the study.

Data Analysis

Data were analyzed using SPSS v17. Quantitative variables are presented as mean ± SD; qualitative variables as frequencies and percentages. Student's t-test or Welch's test was applied based on Levene's test for homogeneity of variance (p >0.05). ROC curve analysis determined optimal cut-off values, with AUC representing discriminatory capacity. A p-value <0.05 was considered statistically significant.

OBSERVATIONS AND RESULTS

Demographic Profile

Table 1. Age and Gender Distribution – Case vs. Control Groups

Age Group (years)	Cases (n)	Cases (%)	Controls (n)	Controls (%)	p-value
18–20	4	8%	4	8%	1.0
21–35	17	34%	17	34%	1.0
36–50	21	42%	21	42%	1.0
51–60	8	16%	8	16%	1.0
Total	50	100%	50	100%	—
Mean ± SD (years)	38.52 ± 11.19		38.52 ± 11.19		—
Female : Male	24 : 26 (48% : 52%)		24 : 26 (48% : 52%)		1.0

The case and control groups were well matched for both age and gender. The mean age was identical in both groups (38.52 ± 11.19 years), with the majority of participants in the 36–50-year age bracket (42%). Gender distribution was also identical, with males comprising 52% (n=26) and females 48% (n=24) in each group (p = 1.0 for both).

Table 2. Clinical Phase and Severity Distribution in Case Group

Parameter	Cases Mean	Cases SD	Controls Mean	Controls SD	p-value
Neutrophil Count (×10³/µL)	6.62	2.30	5.03	2.21	0.0006*
Lymphocyte Count (×10³/µL)	1.91	0.81	2.56	1.71	0.01*
Platelet Count (×10³/µL)	244.08	86.95	226.96	8.59	0.16
Monocyte Count (×10³/µL)	0.42	0.20	0.42	0.23	0.9

In the case group, depressive episodes were slightly more prevalent (52%, n=26) than manic episodes (48%, n=24). Among manic episodes, males were more commonly affected (58.33%), while females predominated in the depressive phase (53.85%), though this difference was not statistically significant (p = 0.38).

Table 3. Hematological Cell Counts – Case vs. Control Groups

Parameter	Mild	Moderate	Severe
No. of Patients	12	21	17
Percentage	24%	42%	34%
Manic Episode (n=24)	3 (12.5%)	11 (45.8%)	10 (41.7%)
Depressive Episode (n=26)	9 (34.6%)	10 (38.5%)	7 (26.9%)

The mean neutrophil count was significantly elevated in the case group (6.62 ± 2.3 ×10³/µL) compared to controls (5.03 ± 2.21 ×10³/µL; p = 0.0006). Conversely, lymphocyte counts were significantly reduced in cases (1.91 ± 0.81 ×10³/µL) versus controls (2.56 ± 1.71 ×10³/µL; p = 0.01). Platelet counts (p = 0.16) and monocyte counts (p = 0.9) did not differ significantly between groups.

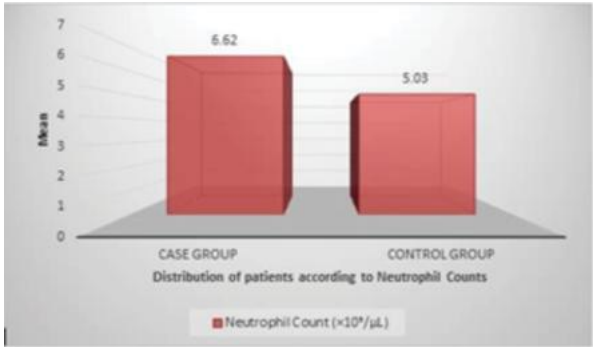

Figure 1. Neutrophil Count: Cases vs. Controls (×10³/µL) — p = 0.0006

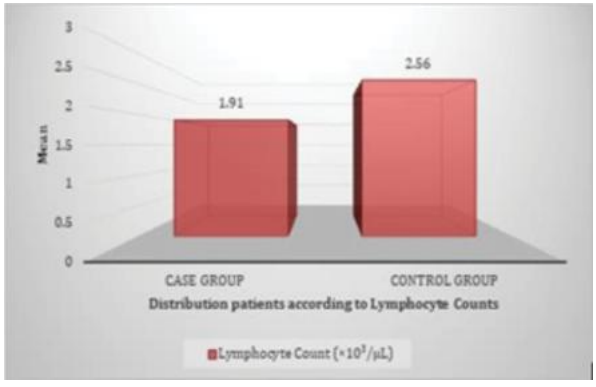

Figure 2. Lymphocyte Count: Cases vs. Controls (×10³/µL) — p = 0.01

Table 4. Derived Inflammatory Ratios – Case vs. Control Groups

Inflammatory Ratio	Cases Mean	Cases SD	Controls Mean	Controls SD	p-value
NLR (Neutrophil-to-Lymphocyte Ratio)	4.43	3.12	2.41	1.27	0.0001*
PLR (Platelet-to-Lymphocyte Ratio)	158.88	101.20	104.71	53.51	0.0012*
MLR (Monocyte-to-Lymphocyte Ratio)	0.26	0.20	0.18	0.10	0.01*

All three inflammatory ratios were significantly elevated in the case group compared to healthy controls. The NLR was markedly higher in cases (4.43 ± 3.12) versus controls (2.41 ± 1.27; p = 0.0001). Similarly, PLR was significantly elevated in cases (158.88 ± 101.2) compared to controls (104.71 ± 53.51; p = 0.0012). MLR was also raised in cases (0.26 ± 0.20) versus controls (0.18 ± 0.10; p = 0.01), indicating a heightened systemic inflammatory state in bipolar disorder.

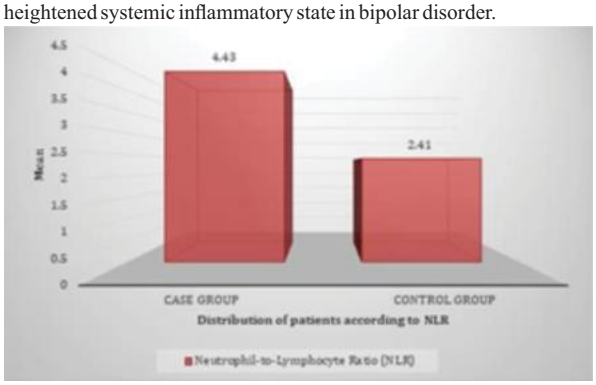

Figure 3. NLR: Cases vs. Controls — p = 0.0001

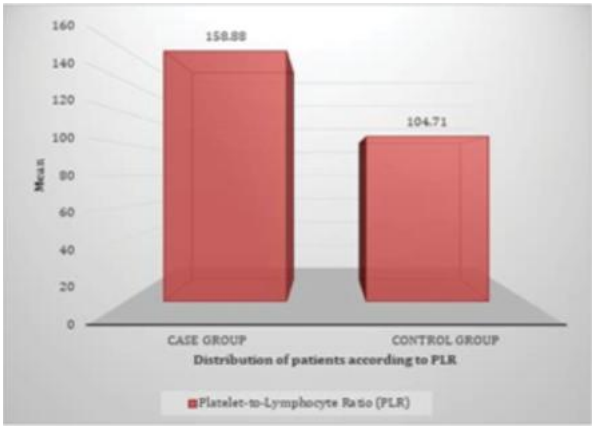

Figure 4. PLR: Cases vs. Controls — p = 0.0012

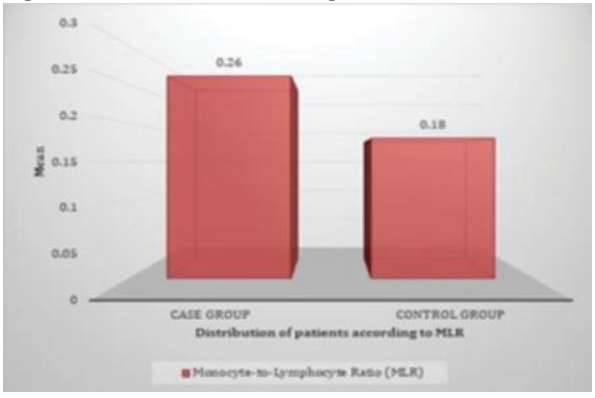

Figure 5. MLR: Cases vs. Controls — p = 0.01

Table 5. Inflammatory Ratios by Severity in Case Group

Ratio	Mild Mean	Mild SD	Moderate Mean	Moderate SD	Severe Mean	Severe SD	p-value
NLR	2.28	0.63	3.17	1.23	7.52	3.47	<0.0001*
PLR	109.17	36.32	135.26	74.97	224.12	121.63	0.001*
MLR	0.17	0.07	0.22	0.15	0.39	0.26	0.004*

Within the case group, all three inflammatory ratios demonstrated a significant progressive increase with increasing disease severity. The NLR rose from 2.28 ± 0.63 (mild) to 3.17 ± 1.23 (moderate) and 7.52 ± 3.47 (severe; p < 0.0001). The PLR similarly escalated from 109.17 ± 36.32 (mild) to 135.26 ± 74.97 (moderate) and 224.12 ± 121.63 (severe; p = 0.001). The MLR showed a parallel trend: 0.17 ± 0.07 (mild), 0.22 ± 0.15 (moderate), and 0.39 ± 0.26 (severe; p = 0.004). These findings indicate that inflammatory ratios mirror disease severity and may serve as useful markers of illness burden.

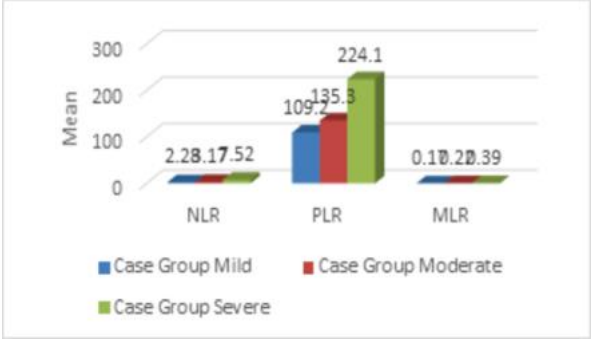

Figure 6. Distribution of patients according to Hematological Ratios

Table 6. Hematological Cell Counts by Severity in Case Group

Cell Count	Mild Mean	Mild SD	Moderate Mean	Moderate SD	Severe Mean	Severe SD	p-value
Neutrophil (×10³/μL)	4.95	0.96	6.01	1.52	8.55	2.49	0.0001*
Lymphocyte (×10³/μL)	2.27	0.57	2.11	0.79	1.40	0.74	0.003*
Platelet (×10³/μL)	239.00	84.62	243.76	103.15	248.05	69.97	0.96
Monocyte (×10³/μL)	0.39	0.15	0.41	0.24	0.46	0.20	0.63

Neutrophil counts increased progressively with severity (Mild: 4.95 ± 0.96; Moderate: 6.01 ± 1.52; Severe: 8.55 ± 2.49 ×10³/μL; p = 0.0001), while lymphocyte counts showed a significant inverse trend (Mild: 2.27 ± 0.57; Moderate: 2.11 ± 0.79; Severe: 1.40 ± 0.74 ×10³/μL; p = 0.003). Platelet (p = 0.96) and monocyte (p = 0.63) counts did not differ significantly across severity groups.

DISCUSSION

Bipolar disorder is increasingly recognized as a condition involving not only neurotransmitter dysregulation but also systemic immune activation. The present study evaluated hematological inflammatory markers — NLR, PLR, and MLR — in patients with bipolar affective disorder and compared them with healthy matched controls, while also examining their variation across disease severity.

Demographic Profile

The case and control groups were well matched for age and gender, with a mean age of 38.52 ± 11.19 years in both groups and an identical gender distribution (males 52%, females 48%; p = 1.0). The predominant age group was 36–50 years (42%), followed by 21–35 years (34%). These findings are consistent with Catak Z et al[10], who reported comparable mean ages across controls, schizophrenia, bipolar disorder, and depression groups (p = 0.30), and with Raj R et al[11], who similarly demonstrated no significant age-related variation between cases and controls ($\chi^2 = 3.40$, p = 0.49). Regarding gender, our results align with Catak Z et al[10], who observed a nearly equal gender distribution across diagnostic groups, while contrasting with Dionisie V et al[12], who reported a statistically significant gender difference across groups ($\chi^2 = 10.06$, p = 0.007).

Clinical Phase and Gender Distribution

Among the 50 cases, depressive episodes were slightly more prevalent (52%, n = 26) than manic episodes (48%, n = 24). Males predominated in the manic phase (58.33%), while females were more common in the depressive phase (53.85%), though this difference was not statistically significant (p = 0.38). This pattern is consistent with Inanli I et al[13], who reported comparable gender distribution in both depression (females 54%) and mania (females 51%; p = 0.922), and with Raj R et al[11], who similarly noted male predominance in bipolar mania and female predominance in bipolar depression. The slight female predominance in the depressive phase observed in our study may reflect known gender-related variations in mood disorder expression, though our sample size limits definitive conclusions.

Neutrophil and Lymphocyte Counts

Neutrophil counts were significantly elevated in cases (6.62 ± 2.30 × 10³/μL) compared to controls (5.03 ± 2.21 × 10³/μL; p = 0.0006), while lymphocyte counts were significantly reduced (1.91 ± 0.81 vs. 2.56 ± 1.71 × 10³/μL; p = 0.01). These reciprocal changes are reflective of a pro-inflammatory shift, as neutrophils are core effectors of innate immunity and tend to increase during inflammatory activation, while lymphocyte suppression may indicate dysregulation of adaptive immune responses. These findings are corroborated by Catak Z et al[10], who reported significantly higher neutrophil counts across psychiatric groups compared to controls (p = 0.025), and lower lymphocyte counts in bipolar disorder relative to controls (p = 0.007).

In contrast, platelet counts (244.08 ± 86.95 vs. 226.96 × 10³/μL; p = 0.16) and monocyte counts (0.42 ± 0.20 vs. 0.42 ± 0.23 × 10³/μL; p = 0.9) did not differ significantly between groups. The non-significant platelet finding aligns with Inanli I et al[13] (p = 0.695) and Kulacaoglu F et al[14] (p = 0.348), both of whom reported comparable platelet levels across bipolar phases and controls. Similarly, non-significant monocyte differences are consistent with Inanli I et al. (p = 0.1).

Neutrophil-to-Lymphocyte Ratio (NLR)

The NLR was markedly and significantly elevated in cases (4.43 ± 3.12) compared to controls (2.41 ± 1.27; p = 0.0001), underscoring the role of immune imbalance in bipolar disorder beyond classical neurotransmitter hypotheses. These findings are in agreement with Giri N et al[15] who reported that 74% of bipolar cases had NLR > 2 compared to only 5% of controls ($\chi^2 = 99.989$, p < 0.0001), and with Inanli I et al[13] who demonstrated significantly higher neutrophil counts in mania (p = 0.002), suggestive of an elevated NLR during active disease phases.

Platelet-to-Lymphocyte Ratio (PLR)

PLR was significantly higher in cases (158.88 ± 101.20) compared to controls (104.71 ± 53.51; p = 0.0012), positioning it as a potential indicator of systemic inflammation in bipolar disorder. Giri N et al[15] similarly reported that 74% of cases had PLR > 210, while all controls fell within the 90–210 range (p < 0.0001). Kalelioglu T et al[16] also found significantly elevated platelet and lymphocyte counts in manic and euthymic groups compared to controls (p < 0.001), though direct PLR comparison was limited in their study.

Monocyte-to-Lymphocyte Ratio (MLR)

MLR was significantly elevated in cases (0.26 ± 0.20) compared to controls (0.18 ± 0.10; p = 0.01), suggesting persistent immune activation. Usta M B et al[17] reported the highest MLR in mania (0.31 ± 0.10) compared to controls (0.22 ± 0.06), with significant differences particularly between manic patients and controls, which is consistent with our findings. Poli L F et al[18]. similarly demonstrated significantly higher MLR in (hypo)mania versus controls (0.28 ± 0.18 vs. 0.23 ± 0.12; p = 0.008), further reinforcing the association between elevated MLR and active disease states in bipolar disorder.

Severity-Wise Variation in Hematological Parameters

A key finding of this study was the progressive and statistically significant escalation of all three inflammatory ratios with increasing disease severity. NLR rose from 2.28 ± 0.63 (mild) to 3.17 ± 1.23 (moderate) and 7.52 ± 3.47 (severe; p < 0.0001). PLR increased from 109.17 ± 36.32 to 135.26 ± 74.97 and 224.12 ± 121.63 (p = 0.001), and MLR from 0.17 ± 0.07 to 0.22 ± 0.15 and 0.39 ± 0.26 (p = 0.004). Correspondingly, neutrophil counts rose significantly across severity groups (4.95 → 6.01 → 8.55 × 10^3/μL; p = 0.0001), while lymphocyte counts declined (2.27 → 2.11 → 1.40 × 10^3/μL; p = 0.003). Platelet and monocyte counts remained non-significant across severity groups (p = 0.96 and p = 0.63, respectively).

These severity-dependent trends indicate that neutrophils, as central mediators of innate immunity, amplify inflammatory signaling during acute disease exacerbation, while declining lymphocyte counts may reflect suppression of adaptive immune regulation. The resulting elevation in NLR, PLR, and MLR thus mirrors the overall shift toward a pro-inflammatory state commensurate with clinical severity.

These findings are supported by Imre O et al[19] who demonstrated significantly higher NLR, MLR, and PLR in manic and euthymic patients versus controls (p < 0.001), and by Usta M B et al[17] who found elevated NLR in bipolar mania compared to controls (p = 0.004). However, Usta M B et al[17] reported non-significant PLR (p = 0.118) and MLR (p = 0.108), which contrasts with our observation of significant elevations across all three ratios with severity. This discrepancy may reflect differences in sample composition, phase distribution, or severity stratification between studies. Poli L F et al[18]. further support our findings, demonstrating significantly higher NLR and PLR in (hypo)mania compared to depression (p < 0.001), along with elevated MLR (p = 0.008).

CONCLUSION

This study demonstrates that NLR, PLR, and MLR are significantly elevated in patients with bipolar affective disorder compared to healthy controls, with all three ratios showing a significant progressive increase with disease severity. These readily available, cost-effective hematological indices may serve as useful surrogate markers of systemic neuroinflammation in bipolar disorder, complementing clinical assessment and potentially aiding in monitoring disease progression and therapeutic response. Further large-scale, longitudinal studies are warranted to establish standardized cut-off values and to evaluate their utility across different phases and treatment outcomes in bipolar disorder.

Conflict of Interest: NIL
Fundings: NIL

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