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Original

Evaluation of hematological inflammatory parameters in patients with chronic migraine: a pilot study

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Abstract

Introduction
Migraine is a prevalent and disabling neurological disorder characterized by recurrent attacks of severe, pulsating, unilateral headache accompanied by phonophobia, photophobia, nausea and/or vomiting. Its pathophysiology involves sterile inflammation within the cortex–meninges interface and activation of the trigeminal system. Despite its burden, there are still no laboratory biomarkers established in routine clinical practice for diagnosis, monitoring, or prognostic assessment.

Objective
To investigate the feasibility of complete blood count (CBC)–derived parameters and composite indices as potential biomarkers in migraine.

Methods
This retrospective case–control study included individuals aged 18–69 years with chronic migraine and age- and sex-matched controls without migraine. CBC parameters and combined indices were analyzed, including the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), neutrophil-to-monocyte ratio (NMR), lymphocyte-to-monocyte ratio (LMR), and platelet-to-monocyte ratio (PMR).

Results
Twenty-five patients with migraine and 29 controls were included. Patients with migraine had a significantly higher NMR compared with controls [11.42 (8.53–12.94) vs 7.76 (6.13–10.17); p=0.029]. No differences were observed in individual CBC parameters or in the other ratios evaluated.

Conclusion
These findings suggest a predominance of neutrophil-related activity in migraine-associated inflammation. Larger, prospective studies are needed to validate the diagnostic and prognostic value of NMR.

Keywords:
Biomarkers
Blood cell count
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Introduction

Migraine is a common neurological disorder affecting approximately 15% of the global population and is the second leading cause of disability worldwide (1). It is a complex, multifactorial condition characterized by recurrent headache attacks lasting 4 to 72 hours. Typical features of a migraine attack include unilateral location, pulsating quality, moderate to severe intensity, aggravation by routine physical activity, and association with nausea and/or photophobia and phonophobia (2).

Despite substantial advances in understanding migraine pathophysiology, the precise mechanisms involved in attack initiation remain incompletely elucidated. Among the most extensively investigated processes in migraine pathogenesis are vascular dysfunction, cortical spreading depression (CSD), activation of the trigeminovascular system, as well as neuroinflammation and neurogenic inflammation (3).

Migraine is currently understood as a process of sterile inflammation within the cortex–meninges complex, characterized by activation of trigeminal nociceptors through the release of multiple pro-inflammatory and excitatory mediators, potentially triggered by cortical spreading depression (4). Another key mediator is calcitonin gene-related peptide (CGRP), a neuropeptide released by trigeminal afferent fibers that perpetuates neurogenic inflammation (5). Experimental studies indicate that peripheral nociceptive stimuli can activate neurons in the trigeminal ganglion, inducing aberrant communication between neurons and satellite glial cells (6). In addition, CGRP released from neuronal somata within the ganglion acts in a paracrine manner on glial cells, increasing the secretion of pro-inflammatory cytokines and contributing to peripheral sensitization and amplification of nociceptive signaling (7), thereby creating a pro-inflammatory intraganglionic microenvironment.

Besides, the trigeminal ganglion is located outside the blood–brain barrier and interacts closely with vascular and immune compartments (8). Accordingly, inflammatory signaling arising from the trigeminovascular system may extend beyond the meninges. In addition, soluble mediators may access peripheral immune pathways through meningeal lymphatic drainage and related neuroimmune communication (9), providing a biologically plausible link between local migraine-related inflammation and low-grade systemic hematologic changes.

Although there is still no definitive consensus regarding the role of inflammation in migraine pathophysiology, multiple lines of evidence support the involvement of neuroimmune mechanisms, including increased pro-inflammatory cytokines, activation of glial cells and the trigeminovascular system, and, in some subgroups, a systemic pro-inflammatory state detectable in peripheral blood (7,10,11). A systematic review investigating the inflammatory profile in migraine reported higher levels of pro-inflammatory cytokines in migraine compared with controls, as well as in chronic migraine compared with episodic migraine and during the ictal period compared with the interictal period, suggesting a role of the immune system in migraine susceptibility and disease activity (12).

From a clinical standpoint, systemic corticosteroids are used in the management of severe and refractory attacks, such as status migrainosus, and as bridging therapy in patients with medication-overuse headache. Although the effects are generally of modest magnitude, they represent clinically relevant benefit and suggest that pharmacologically modifiable inflammatory pathways contribute to the condition (13). In addition, epidemiological studies have shown a higher prevalence of autoimmune diseases among individuals with migraine and possible causal relationships between immune dysregulation and primary headaches, further supporting the link between immunity and migraine (10). Acute viral infections, such as Covid-19 and influenza, are frequently associated with severe headache and, in patients with pre-existing migraine, with worsening in the frequency, duration, or pattern of pain, including chronic transformation after the infectious episode, which further supports the hypothesis that systemic inflammatory processes may exacerbate already vulnerable nociceptive circuits (14,15). In this context, it becomes particularly relevant to investigate simple, widely available, low-cost peripheral inflammatory markers—such as complete blood count parameters and derived indices—as potential adjunct biomarkers for migraine characterization.

More recently, widely accessible, rapidly obtained, low-cost biomarkers have been increasingly used to evaluate autoimmune and infectious diseases, aiming to support diagnosis and to quantify disease activity or severity. In particular, complete blood count parameters—including platelet count—and indices derived from their combinations have been associated with both the presence and activity of different autoimmune and inflammatory conditions and have also proven useful as prognostic markers in infectious diseases, such as sepsis (16–21).

Given that, in clinical practice, there are still no established laboratory biomarkers for migraine and that, even in research settings, available biomarkers are often costly, require imported reagents, and rely on equipment with limited accessibility, it is relevant to investigate feasible alternatives. In this context, the present study seeks to address these gaps by exploring potential complete blood count alterations as an easily accessible, low-cost inflammatory biomarker in migraine, thereby expanding knowledge of the underlying pathophysiological mechanisms and informing future diagnostic and prognostic applications.

Methods

This retrospective case–control pilot study included adults aged 18–69 years followed at an academic outpatient clinic in a tertiary hospital. The case group comprised individuals with chronic migraine, whereas the control group consisted of age- and sex-matched individuals with epilepsy and no history of migraine. Participants were eligible if they had available clinical data in the medical records and at least one complete blood count (CBC) performed at the study institution between January 2023 and May 2025. Individuals were included regardless of the use of migraine preventive therapy and/or medication overuse. Patients with a prior diagnosis of chronic infection, autoimmune disease, active neoplasm, pregnancy, or incomplete medical records were excluded. Because of the small sample size and the pilot nature of the study, potential confounders other than age and sex were not controlled.

The selection of patients with epilepsy as a control group was based on several clinical and methodological factors. First, both groups were recruited from the same tertiary outpatient neurology clinic, ensuring comparable clinical contexts and healthcare access. Second, since epilepsy is a chronic neurological condition that requires periodic laboratory monitoring, blood count data were readily available, facilitating a retrospective comparison. Furthermore, using a control group with a distinct neurological condition, but one that shares the use of several medications commonly prescribed in clinical practice, such as antiseizure drugs and antidepressants.

Migraine diagnosis was established by a neurologist according to the International Classification of Headache Disorders, 3rd edition (ICHD-3) criteria (2). Epilepsy diagnosis and classification were performed by a clinical neurophysiologist in a specialized outpatient clinic, based on the 2017 International League Against Epilepsy (ILAE) Classification of the Epilepsies and subsequent updates, when applicable (22).

Demographic, clinical, and therapeutic data were obtained from electronic medical records. CBC parameters, including platelet count, were compared between groups, as were combined indices: neutrophil/lymphocyte ratio (NLR), platelet/lymphocyte ratio (PLR), neutrophil/monocyte ratio (NMR), lymphocyte/monocyte ratio (LMR), and platelet/monocyte ratio (PMR). There was no standardization of blood collection timing in relation to acute symptomatic episodes in the migraine group (e.g., ictal vs interictal phase).

The study was approved by the Research Ethics Committee of the State University of Londrina (UEL) under CAAE 55804521.9.0000.5231. As this was a medical-record review study, informed consent was waived upon the researcher's signature of a confidentiality and data protection agreement, in accordance with current national regulations for research involving human participants.

Categorical variables were described as absolute number (N) and percentage (%) and compared using Pearson's chi-square test or Fisher's exact test when indicated by the distribution of expected frequencies. Continuous variables were presented as median and interquartile range (25th–75th) and analyzed using the Mann–Whitney U test. Statistical significance was set at $p < 0.05$. Statistical analyses were performed using IBM SPSS Statistics, version 25.0 (IBM Corp., Armonk, NY, USA).

Results

A total of 25 individuals with migraine and 29 controls without migraine (diagnosed with epilepsy) were included, with no significant differences in sex or age between groups ($p > 0.05$), as shown in Table 1.

Table 1. Sample characteristics

		Control	Migraine	P Value
		N=29	N=25	
Age (years)		50 (28-57)	45 (32-53)	0.401
Sex	Male	6 (20.7)	1 (4.0)	0.108
	Female	23 (79.3)	24 (96.0)	
Systemic arterial hypertension	No	26 (89.7)	19 (76.0)	0.275
	Yes	3 (10.3)	6 (24.0)	
Diabetes mellitus	No	26 (89.7)	23 (92.0)	>0.999
	Yes	3 (10.3)	2 (8.0)	

Continuous variables were presented as median and interquartile range (25th–75th) and categorical variables were presented as absolute number (N) and percentage (%). For this analysis, the Mann–Whitney U test and Fisher's exact test were used.

Among patients with migraine, all had chronic migraine; 21 (84.0%) had migraine without aura and 4 (16.0%) had migraine with aura. Nineteen (76.0%) were receiving preventive treatment. Among patients with epilepsy, 21 (72.5%) had focal epilepsy, 2 (6.9%) had generalized epilepsy, and 3 (10.3%) had no classification recorded. In this group, 27 (93.1%) were using antiseizure medications. Considering the continuous use of medications for the underlying condition, there was no difference between the migraine group regarding the use of preventive treatment and the epilepsy control group regarding the use of antiseizure medications (p = 0.125)

There were no statistically significant differences in complete blood count parameters related to red blood cells, white blood cells, or platelets (Table 2). However, when these parameters were combined into ratios, patients with migraine had a higher NMR, with 11.42 (8.53–12.94) in the migraine group compared with 7.76 (6.13–10.17) in controls, and this difference was statistically significant (p = 0.029), as shown in Figure 1. No statistically significant differences were observed for the other indices (Table 3).

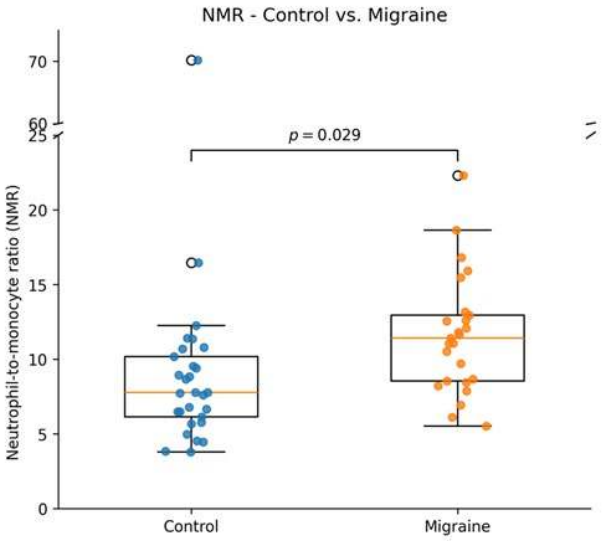

Figure 1. Comparative plot of the Neutrophil-to-Monocyte Ratio (NMR) between groups. Individual patient values are shown as dots. Boxes represent the interquartile range, the central line indicates the median, and whiskers represent data dispersion. The y-axis is broken to improve visualization of the main data distribution while preserving the extreme value in the control group. Groups were compared using the Mann–Whitney U test.

Table 2. Complete blood count parameters compared between groups

	Control	Migraine	P Value
	N=29	N=25	
Red blood cells (10^6/μL)	4.6 (4.2-4.78)	4.57 (4.48-4.86)	>0.999
Hemoglobin (g/dL)	13.4 (12.9-14.6)	13.5 (13.1-14.1)	0.585
Hematocrit (%)	40.5 (37.5-42.2)	40.6 (39.2-41.3)	0.800
Mean corpuscular volume (fL)	88 (87.0-92.8)	88.0 (85.0-89.9)	0.800
Mean corpuscular hemoglobin (pg)	30.1 (29.4-31.1)	29.5 (28.7-30.0)	0.102
Mean corpuscular hemoglobin concentration (g/dL)	33.8 (33.2-34.5)	33.6 (33.1-34.3)	0.376
Red cell distribution width (%)	12.8 (12.6-13.6)	13.1 (12.6-13.5)	0.275
White blood cells ($\times10^3$/μL)	5910 (4970-7820)	7130 (6275-8740)	0.341
Neutrophils ($\times10^3$/μL)	3176 (2265-4388)	4133 (3256-4983)	0.102
Eosinophils ($\times10^3$/μL)	77 (44-157)	149 (92-197)	0.102
Basophils ($\times10^3$/μL)	23 (16-32)	22 (16-37)	0.800
Lymphocytes ($\times10^3$/μL)	1915 (1491-2645)	2227 (1822-2608)	0.275
Monocytes ($\times10^3$/μL)	389 (314-500)	380 (339-456)	>0.999
Platelets ($\times10^3$/μL)	266000 (209000-325000)	282000 (256000-307000)	>0.999

The data were presented as median and interquartile range (25th–75th) and were analyzed using the Mann–Whitney U test.

Table 3. Inflammatory indices derived from the complete blood count in patients with chronic migraine and controls

	Control	Migraine	P Value
	N=29	N=25	
Neutrophil/Lymphocyte Ratio (NLR)	1.53 (1.13-2.21)	1.84 (1.48-2.45)	0.585
Platelet/Lymphocyte Ratio (PLR)	138.33 (94.14-169.31)	118.2 (99.83-138.06)	0.275
Neutrophil/Monocyte Ratio (NMR)	7.76 (6.13-10.17)	11.42 (8.53-12.94)	0.029
Lymphocyte/Monocyte Ratio (LMR)	5.05 (3.29-6.88)	5.18 (4.82-7.01)	>0.999
Platelet/Monocyte Ratio (PMR)	677.57 (419.68-843.19)	687.32 (576.69-851.85)	>0.999

The data were presented as median and interquartile range (25th–75th) and were analyzed using the Mann–Whitney U test.

Discussion

The present study compared routinely available complete blood count parameters in patients with migraine, using patients with epilepsy as controls. The main finding was a higher NMR in individuals with migraine ($p = 0.029$) compared with controls, which may indicate a predominance of the rapid, transient innate neutrophil response over the slower, more sustained innate monocytic activity (23).

There is limited investigation in the literature regarding complete blood count findings in individuals with migraine. Current evidence suggests that people with migraine do not exhibit clinically meaningful deviations from reference ranges in CBC parameters. However, the relative distribution among hematologic components, namely the proportions between cellular subtypes and derived indices, may differ in individuals with migraine. Grazzi et al.(24) reported a higher total circulating leukocyte count in patients with chronic migraine compared with both controls without migraine and individuals with episodic migraine. Similarly, Yazar et al.(25) described increased neutrophil counts in individuals with migraine compared with controls, with even higher values during attacks than in the interictal period.

These findings support the hypothesis of a relative predominance of the innate neutrophil response, although this pattern is not specific, since Eryigit et al.(26) reported a more pronounced neutrophil increase in subarachnoid hemorrhage than in migraine or other headache disorders. In contrast, the monocytic lineage has been reported to be reduced in migraine compared with controls in the studies by Wu et al.(27) and Saricam (28), which is consistent with the higher NMR observed in the present study.

The NMR integrates two central compartments of innate immunity. In general, neutrophils participate in the initial, more immediate inflammatory response, whereas monocytes tend to act in subsequent phases, contributing to amplification and modulation of inflammation (23). To date, only two studies have investigated NMR in migraine. Saricam (28) observed significantly higher NMR values in both patients with migraine with aura and those without aura compared with healthy controls, in agreement with the findings of the present study. In contrast, Wu et al.(27) reported a positive correlation between NMR and pain intensity but, paradoxically, a negative correlation with headache frequency, suggesting that NMR may reflect distinct dimensions of inflammatory activity across the clinical spectrum of migraine.

In this context, NMR has already been applied in other conditions. In rheumatoid arthritis, studies have demonstrated its utility for assessing disease activity (23). In neonatal sepsis, it has been reported as a prognostic marker and predictor of mortality (29). In IgA nephropathy, it has been shown to predict disease progression (30). In parallel with these conditions, NMR may contribute to a better understanding of the inflammatory process associated with migraine and may also serve as a potential biomarker for disease monitoring. Therefore, further studies are needed to correlate this laboratory finding with patients' clinical features and to evaluate its potential utility in the differential diagnosis of other headache disorders and neurological diseases.

This pilot study has methodological limitations that should be considered when interpreting the findings. The sample was small and based on convenience recruitment, conducted at a single center, which increases the risk of selection bias and limits the generalizability of the results. In addition, because

this was a retrospective case–control study, causality cannot be inferred and only associations can be identified. Another relevant limitation is the lack of standardization regarding the timing of CBC collection (e.g., ictal vs interictal phase) and the absence of systematic control for concomitant infectious or inflammatory conditions at the time of blood sampling, factors that may alter leukocyte counts and influence derived indices, particularly those based on neutrophil counts. Finally, although no significant difference was observed in the frequency of continuous use of disease-specific medications between groups, the potential influence of different preventive and antiseizure drugs on hematological parameters cannot be fully excluded. Despite these limitations, the study contributes by generating hypotheses about potential changes in CBC cellular distribution in individuals with migraine, within a field still characterized by heterogeneous evidence and frequently constrained by small samples and retrospective designs.

As a pilot study with a small sample size, our findings should be interpreted as exploratory and hypothesis-generating. In addition, although CBC-derived indices have been previously investigated in migraine, chronic migraine remains underrepresented in this literature, which limits direct comparison with prior studies. Therefore, these results underscore the need for larger, prospective studies with standardized sampling that evaluate simple, low-cost, widely available immunological parameters, such as the CBC and its derived indices, to better characterize their clinical and pathophysiological utility in migraine.

Conclusion

NMR is a simple, low-cost, and readily accessible inflammatory marker that was higher in individuals with migraine compared with controls. This finding is consistent with prior literature suggesting a relative predominance of neutrophil-related activity in inflammatory indices associated with migraine. However, further studies are needed to clarify the role of this marker in migraine diagnosis, prognosis, and treatment.

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