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Hematological Findings in Epilepsy: Associations with Seizure Type

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ABSTRACT

Background: Relating systemic inflammation and blood disorders to inflammatory pathogenesis, epilepsy occurs as a chronic neurological disorder. **Objective:** This study aimed to evaluate the comparative relationship between complete blood count (CBC) and epilepsy in epileptic patients and healthy controls. **Study Design:** Comparative cross-sectional study. **Settings:** Department of Neurology, Lady Reading Hospital, Peshawar, Pakistan. **Duration:** September 2020 to August 2021. **Methods:** 284 patients with epilepsy and 100 control subjects. A one-year duration study was conducted in which epileptic patients recruited. Data analyzed and applied Independent t-tests and multivariate logistic regression models were performed at a *p* < 0.05 significance level. **Results:** Patients with epilepsy showed markedly elevated WBC counts (12.34 ± 5.45 vs. 8.26 ± 2.05 $\times 10^3/\mu L$, *p* < 0.001) and had lower RBC counts (4.37 ± 0.77 vs. 4.75 ± 0.62 $\times 10^6/\mu L$, *p* < 0.001) alongside HGB levels of 11.19 ± 2.13 g/dL (*p* < 0.001) which is significantly lower than the control group's 13.25 ± 1.94 g/dL. Cytometry revealed a statistically significant elevation in PLT count (350.99 ± 175.96 vs 289.73 ± 77.93 $\times 10^3/\mu L$, *p* = 0.001). The most common seizure type observed was generalized tonic-clonic seizures (76.76%), with considerable hematologic findings suggestive of inflammation and anemia. **Conclusion:** The research underscores specific CBC anomalies in epilepsy, which confirm inflammatory and hematological pathways relevant to the disease's mechanisms. These results are consistent with previous studies and also incorporate new findings, indicating that further research is needed to investigate CBC-based biomarkers for epilepsy treatment.

Keywords: *Epilepsy, Hematological, Blood Cell Count, Seizures, Pakistan.*

INTRODUCTION

Epilepsy is a persistent condition of the nervous system that manifests as recurrent spontaneous seizures. It impacts around 50 million individuals globally.[1] Even with progress made in therapy and diagnostics, the mechanisms that underlie epilepsy are still not fully comprehended, especially in instances where the structural abnormalities of the brain are absent.[2,3] Recent studies suggest that the following features may be factors that contribute to the development of seizure phenotypes: perturbation of immune regulation, systemic inflammation, and metabolic imbalances.[4,5] Considering the potential influence of systemic factors in epilepsy, blood parameters obtained from a Complete Blood Count (CBC) test may be readily applicable as biomarkers for the risk, development, or response to treatment of the disease.[6,7] The CBC is a routine laboratory test that assesses the basic constituents of blood, including white blood cells (WBCs), red blood cells (RBCs), hemoglobin (HGB), platelets (PLT) and provides a differential count for the leukocyte populations.[8] These factors indicate systemic inflammation, oxidative stress, and immune activation, which have all been associated with the development of epilepsy.[9] Increased white blood cell count, as well as the ratio of neutrophils to lymphocytes, have been correlated with higher frequency and severity of seizures, indicating a potential inflammatory factor in

epilepsy.[10] Similarly, changes in neurovascular integrity and seizure thresholds may be influenced by alterations in cell adhesion-related signaling pathways.[11] Even considering the above-mentioned information, the more in-depth research comparing CBC profiles in patients with epilepsy and healthy controls is quite limited, especially regarding diverse demographic populations.[12,13]

As noted in the literature, prior research has investigated the individual components of CBC in epilepsy; however, most studies either lacked comparison control cohorts or were limited to specific subgroups.[14] Furthermore, certain sub-areas of South Asia, such as Pakistan, are particularly under-researched when it comes to studying hematological profiles stemming from factors like genetic predisposition, infections, nutritional deficiencies, or even the region's unique epidemiological and socio-cultural constructs.[14] Moreover, the association between seizure types and abnormalities in complete blood count has not been thoroughly studied. Investigating these aspects may advance understanding of the myriad systemic impacts of epilepsy and unveil prospective biomarkers for epilepsy acne and its early detection or subsequent treatment monitoring. This study evaluated the CBC values of patients with epilepsy and compared them with those of healthy controls. It tested the hypothesis that certain types of seizures are associated with specific hematological features and analyzed the impact of certain demographic variables on CBC changes in epilepsy.

METHODS

This comparative cross-sectional study was conducted in duration of one year from September 2020 to August 2021 complete blood count values in epileptic patients. The study was conducted at Lady Reading Hospital, Peshawar, Pakistan, following approval from the ethics committee (Ref. No. 102/LRH/MTI, 03-09-2020). All methods were carried out in compliance with associated policies and procedures. Written informed consent was waived due to the retrospective nature of the study, but patient data were anonymized to ensure confidentiality. The study protocol adhered to the principles of the Declaration of Helsinki and was approved by the local ethics committee.

Two hundred eighty-four patients with epilepsy and one hundred healthy subjects comprised our study sample.[15] Cases were gathered from the outpatient department for neurology, while controls were drawn from patients attending general medical checkups who had no history of neurological diseases. A neurologist confirmed the diagnosis of epilepsy based on history and electroencephalogram (EEG) assessments as per International League Against Epilepsy (ILAE) guidelines. Controls were matched with cases for age and sex to reduce potential confounding variables. Both groups were screened for recent infections (within the last four weeks), chronic inflammatory diseases, hematological conditions, and the use of anti-inflammatory or immunosuppressive drugs, which were excluded as criteria for participation.

Demographic and clinical data were extracted from medical records, including age, gender, seizure type (generalized tonic-clonic, focal, or other), age of onset, family history of epilepsy, and current antiepileptic drug regimen. For all participants, CBC parameters (WBC count, RBC count, hemoglobin, platelet count, neutrophil percentage, and lymphocyte percentage) were obtained from venous blood samples collected in EDTA tubes and analyzed using an automated hematology analyzer (Sysmex XN-1000).

Data were analyzed using SPSS version 26.0. Continuous variables were presented as mean ± standard deviation (SD) or median (interquartile range) based on the normality of the distribution, as assessed by the Shapiro-Wilk test. Categorical variables were expressed as frequencies and percentages. Group comparisons (epilepsy vs. control) were performed using independent t-tests for normally distributed data and Mann-Whitney U tests for non-parametric data. Chi-square or Fisher's exact tests were used for categorical variables. Multivariate logistic regression was applied to identify independent associations between CBC parameters and epilepsy after adjusting for age, sex, and other potential confounders. A p-value <0.05 was considered statistically significant.

RESULTS

The demographic and clinical characteristics of the study participants (N = 284) are presented in Table 1. The cohort comprised 172 males (60.56%) and 112 females (39.44%), indicating a male predominance in the study population. A majority of participants (90.14%, n = 256) reported no family history of epilepsy. At the same time, a small proportion had a positive family history, including siblings (2.11%, n = 6), cousins (1.41%, n = 4), maternal relatives (1.06%, n = 3), sisters (1.06%, n = 3), grandparental and maternal combined (0.7%, n = 2), and paternal history (0.35%, n = 1). Moreover, the control group comprises a total of 100 participants, including 54 females (54%) and 46 males (46%), as shown in Table 1.

Regarding seizure types, generalized tonic-clonic (GTC) seizures (76.76%, n = 218) were the most prevalent, followed by focal seizures (11.27%, n = 32). Subtypes of focal seizures included right-sided (4.23%, n = 12), left-sided (2.82%, n = 8), febrile seizures (2.82%, n = 8), active seizures (1.06%, n = 3), subtle seizures (0.7%, n = 2), and

myoclonic seizures (0.35%, n = 1). These findings highlight the heterogeneity of seizure presentations in the study cohort, with GTC seizures being the most common.

Table 1: Demographic and clinical characteristics of the study population (N = 284)

Variable	Gender	Frequency
Gender	Male	172 (60.56%)
	Female	112 (39.44%)
Gender Control	Female	54 (54%)
	Male	46 (46%)
Family History	No	256 (90.14%)
	Yes	9 (3.17%)
	Yes/Siblings	6 (2.11%)
	Yes/Cousin	4 (1.41%)
	Yes/Maternal	3 (1.06%)
	Yes/Sister	3 (1.06%)
	Yes/Grand F+M	2 (0.7%)
	Yes /Father	1 (0.35%)
Seizure Type	GTC Fits	218 (76.76%)
	Focal Fits	32 (11.27%)
	Focal Fits-Rt Side	12 (4.23%)
	Focal Fits-Lt side	8 (2.82%)
	Febrile Fits	8 (2.82%)
	Active Fits	3 (1.06%)
	Subtle Fits	2 (0.7%)
	Myoclonic Fits	1 (0.35%)

Table 2 presents the comparative analysis of hematological parameters and age distribution between epilepsy patients (cases) and healthy controls. The mean age of epilepsy patients was 11.43 ± 16.88 years (range: 0.08–80 years), with the age of seizure onset averaging 7.32 ± 13.36 years (range: 0.08–69 years). In contrast, the control group had a higher mean age (30.28 ± 15.75 years, range: 1–72 years), reflecting the inclusion of adults in the non-epileptic cohort.

Key hematological findings revealed that epilepsy patients exhibited elevated white blood cell (WBC) counts (12.34 ± 5.45 × 10^3/μL, range: 2.46–41.1) compared to controls (8.26 ± 2.05 × 10^3/μL, range: 3.2–14.33), suggesting potential inflammatory activity. Red blood cell (RBC) counts were lower in patients (4.37 ± 0.77 ×10^6/μL, range: 2.71–6.21) versus controls (4.75 ± 0.62 ×10^6/μL, range: 4.3–6.4), while hemoglobin (HGB) levels were 11.19 ± 2.13 g/dL (range: 6.32–18.1) in patients and 13.25 ± 1.94 g/dL (range: 12.7–16.9) in controls, indicating a trend toward anemia in the epilepsy group. Platelet (PLT) counts were higher in patients (350.99 ± 175.96 × 10^3/μL, range: 12–955) than in controls (289.73 ± 77.93 × 10^3/μL, range: 132–597), which may reflect altered coagulation or inflammatory states.

Differential leukocyte analysis showed that epilepsy patients had lower neutrophil (NEU) percentages (57.47 ± 21.51%, range: 3.7–95.7) compared to controls (61.05 ± 8.33%, range: 31.5–79.5), while lymphocyte (LYM) percentages were comparable between groups (patients: 32.87 ± 20.36%, controls: 31.97 ± 7.52%). These results underscore distinct hematological profiles in epilepsy, potentially linked to underlying pathophysiology or treatment effects.

Table 2: Hematological and age-related parameters of epilepsy patients and control group

Variable	Mean ± Std.	Minimum	Maximum
Age	11.43 ± 16.88	0.08	80
Age Of Onset	7.32 ± 13.36	0.08	69
White Blood Cell (WBC)	12.34 ± 5.45	2.46	41.1
Red Blood Cell (RBC)	4.37 ± 0.77	2.71	6.21
Hemoglobin (HGB)	11.19 ± 2.13	6.32	18.1
Platelets (PLT)	350.99 ± 175.96	12	95.5
Neutrophil (NEU)	57.47 ± 21.51	3.7	95.7
Lymphocyte (LYM)	32.87 ± 20.36	4.3	90.4
Age Control	30.28 ± 15.75	1	72
White Blood Cell (WBC) Control	8.26 ± 2.05	3.2	14.33
Red Blood Cell (RBC) Control	4.75 ± 0.62	4.3	6.4
Hemoglobin (HGB) Control	13.25 ± 1.94	12.7	16.9
Platelets (PLT) Control	289.73 ± 77.93	132	597
Neutrophil (NEU) Control	61.05 ± 8.33	31.5	79.5
Lymphocyte (LYM) Control	31.97 ± 7.52	14.5	51

Table 3 presents the results of independent samples t-tests comparing key hematological parameters between epilepsy patients and healthy controls, including effect size measures (Cohen's d). For white blood cell (WBC) counts, epilepsy patients showed significantly higher values than controls (mean difference = 4.39 ×10^3/μL, 95% CI [3.08, 5.7], t(99) = 6.63, p < .001), with a medium effect size (Cohen's d = 0.66). This suggests systemic inflammation may be associated with epilepsy. Red blood cell (RBC) parameters revealed significantly lower values in patients (mean difference = -0.45 ×10^6/μL, 95% CI [-0.61, -0.28], t(99) = -5.23, p < .001, d = 0.52), as did hemoglobin levels (mean difference = -2.52 g/dL, 95% CI [-3.01, -2.04], t(99) = -10.3, p < .001, d = 1.03), indicating

potential anemia in the epilepsy group. Platelet (PLT) counts were significantly elevated in patients (mean difference = 72.87 ×10³/µL, 95% CI [31.58, 114.16], t(99) = 3.5, p = .001, d = 0.35), possibly reflecting inflammatory or coagulation abnormalities. No significant differences were found for neutrophil percentages (mean difference = -3.77%, p = .081) or lymphocyte percentages (mean difference = 0.61%, p = .749), with both showing negligible effect sizes (d = 0.18 and 0.03 respectively).

Table 3: Comparative analysis of hematological parameters between epilepsy patients and controls using independent samples t-test

	Mean	Std. Deviation	Std. Error Mean	Lower limit	Upper limit	t	df	p	Cohen's d
WBC - WBC control	4.39	6.62	0.66	3.08	5.7	6.63	99	<.001	0.66
RBC - RBC control	-0.45	0.85	0.09	-0.61	-0.28	-5.23	99	<.001	0.52
HGB - HGB control	-2.52	2.45	0.24	-3.01	-2.04	-10.3	99	<.001	1.03
PLT - PLT control	72.87	208.09	20.81	31.58	114.16	3.5	99	.001	0.35
NEUT - NEUT control	-3.77	21.36	2.14	-8.01	0.47	-1.76	99	.081	0.18
LYMP - LYMP control	0.61	18.93	1.89	-3.15	4.36	0.32	99	.749	0.03

DISCUSSION

This study aimed to fill the knowledge gap between complete blood count (CBC) parameters and blood profiles of epileptic patients by comparing their hematological profiles to those of non-epileptic, age- and gender-matched controls. Our findings indicate that patients with epilepsy differed from the controls in multiple hematological parameters. In comparison to the control group, patients with epilepsy were found to have greater WBC counts and PLT levels as well as lower RBC and HGB levels. These observations corroborate the emerging hypothesis proposed by Ahmad *et al*[16] and Alparitti *et al*,[17] suggesting that certain types of epilepsy are at least partially linked to inflammation and altered immune homeostasis. We provide a detailed commentary on the relevant literature, analyzing the clinical findings in light of the prevailing theories.

In patients with epilepsy, our study showed significantly higher WBC counts (12.34 ± 5.45 × 10³/µL) compared to controls (8.26 ± 2.05 × 10³/µL), with a medium effect size of Cohen's d = 0.66. This supports the hypothesis that, at least in part, systemic inflammation may contribute to the pathogenesis of epilepsy.[18] similar findings in patients with generalized tonic-clonic seizures, where elevated WBC counts indicated a state of inflammation.[19] Contrary to these findings, a smaller cohort study found no WBC count differences between epilepsy patients and controls. This discrepancy may be due to differing populations, seizure types, or other methods, for example, our study's exclusion of patients with recent infections.[20]

As an inflammatory biomarker, the NLR (Neutrophil-Lymphocyte Ratio) showed no significant difference in our cohort, which is contrary to the findings reported by Wang *et al*. and Hosseini *et al*., who characterized the NLR as a predictive biomarker for seizure severity and recurrence.[21] The differences in outcomes could be attributed to the more uniform hypothesis employed by these studies compared to ours. The explainable reason why our study did not identify significant differences could be the heterogeneous makeup of our cohort, which comprised different seizure types as well as ages spanning decades. Further research focused on epilepsy is necessary, particularly studies with more controlled parameters and refined sample sizes that help elucidate the role NLR plays in heterogeneous epilepsy.[22]

Our findings showed that epilepsy patients exhibited lower red blood cell (RBC) counts (4.37 ± 0.77 × 10^6/µL vs. 4.75 ± 0.62 × 10^6/µL) and hemoglobin concentrations (11.19 ± 2.13 g/dL vs. 13.25 ± 1.94 g/dL), which is consistent with a trend towards anemia. These results support the findings of Ahmad *et al.*, who diagnosed anemia in 30% of epileptic patients, which may be attributed to Chronic inflammation or inadequate diet. RBC parameters remained unchanged, which supports the idea that some regions may have specific determinants due to their dietary patterns or genetic makeup.[23] The high proportion of patients with generalized tonic-clonic seizures (76.76%) in our cohort could further explain these findings, since frequent seizures are known to cause metabolic stress and disrupt normal blood and bone marrow physiology.[24,25]

Patients with epilepsy had significantly increased platelet counts (350.99 ± 175.96 × 10³/µL) compared to controls (289.73 ± 77.93 × 10³/µL), supporting the findings of Rinawati *et al*. (2024), who associated thrombocytosis with neuroinflammation.[26,27] Increased platelet counts

may be a result of endothelial injury, indicating some form of dysfunction, or could serve as a compensatory mechanism following recurrent seizures (Wang *et al*., 2025). On the other hand, Banote *et al*. (2022) report on other studies that do not find any significant differences in counts, which highlights the need for a deeper investigation into the mechanisms of platelet activation in epilepsy.[18,19]

The mean age of epilepsy patients in our study (11.43 ± 16.88 years) was lower than that of controls (30.28 ± 15.75 years), reflecting the early onset of epilepsy in many cases. This age disparity may confound hematological comparisons, as pediatric populations inherently exhibit different CBC profiles. Nonetheless, our multivariate analysis adjusted for age and sex, strengthening the validity of our findings.[20,22,28]

CONCLUSION

Our study underscores the association between CBC abnormalities and epilepsy, particularly elevated WBC counts, anemia, and thrombocytosis, supporting the involvement of systemic inflammation and metabolic disturbances in epilepsy. While our findings align with some published studies, discrepancies highlight the need for standardized methodologies and larger, diverse cohorts. Future research should investigate longitudinal changes in CBC and their predictive value for seizure outcomes, potentially paving the way for the development of novel biomarkers in epilepsy management.

LIMITATIONS

Several limitations must be acknowledged. First, the retrospective design limits the ability to make causal inferences. Second, the heterogeneity of seizure types and the inclusion of patients on antiepileptic drugs (AEDs) may have influenced CBC parameters. For instance, AEDs like valproate are known to affect platelet function.[29,30] Future prospective studies should stratify patients by seizure type, AED regimen, and disease duration to minimize confounding. Additionally, regional factors, such as endemic infections or nutritional deficiencies in Pakistan, may uniquely influence hematological profiles, warranting cross-cultural comparisons.

SUGGESTIONS / RECOMMENDATIONS

Future research should explore the underlying mechanisms linking systemic inflammation and hematological alterations—particularly elevated white blood cell and platelet counts, and reduced hemoglobin levels—with epilepsy pathophysiology. Longitudinal studies involving larger, age-matched cohorts are recommended to determine whether these hematological profiles are predictive markers, consequences of epilepsy, or effects of treatment. Additionally, investigating sex-based differences, seizure subtypes, and family history in relation to these blood parameters may help uncover distinct biological patterns and guide the development of personalized diagnostic or therapeutic strategies.

CONFLICT OF INTEREST / DISCLOSURE

The authors declare no conflict of interest in relation to this study.

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REFERENCES

1. Chen Z, Brodie MJ, Ding D, Kwan P. Editorial: Epidemiology of epilepsy and seizures. Front Epidemiol. 2023;3:1273163.
2. Stafstrom CE, Carmant L. Seizures and epilepsy: an overview for neuroscientists. Cold Spring Harb Perspect Med. 2015;5(6).
3. Guerrini R, Marini C, Mantegazza M. Genetic Epilepsy Syndromes Without Structural Brain Abnormalities: Clinical Features and Experimental Models. Neurotherapeutics. 2014;11(2):269-85.
4. Mukhtar I. Inflammatory and immune mechanisms underlying epileptogenesis and epilepsy: From pathogenesis to treatment target. Seizure. 2020;82:65-79.
5. Shariff S, Nouh HA, Inshutiyimana S, Kachouh C, Abdelwahab MM, Nazir A, et al. Advances in understanding the pathogenesis of epilepsy: Unraveling the molecular mechanisms: A cross-sectional study. Health Sci Rep. 2024;7(2):e1896.
6. Banote RK, Akel S, Zelano J. Blood biomarkers in epilepsy. Acta Neurol Scand. 2022;146(4):362-8.
7. Huang J, Yang J, Miao J, Wen H. Changes in routine blood parameters of patients with generalized tonic clonic seizure: a retrospective study. Neurosciences (Riyadh). 2023;28(2):123-9.
8. Olivo S, Buoite Stella A, Pavan S, Cegalin M, Furlanis G, Cheli M, et al. Admission neutrophil-to-lymphocyte ratio predicts length of hospitalization and need for ICU admission in adults with Status Epilepticus. Seizure: European Journal of Epilepsy. 2023;106:80-4.
9. Rinawati W, Machin A, Aryati A. The Role of Complete Blood Count-Derived Inflammatory Biomarkers as Predictors of Infection After Acute Ischemic Stroke: A Single-Center Retrospective Study. Medicina [Internet]. 2024; 60(12).
10. Hosseini S, Mofrad AME, Mokarian P, Nourigheimasi S, Azarhomayoun A, Khanzadeh S, et al. Neutrophil to Lymphocyte Ratio in Epilepsy: A Systematic Review. Mediators Inflamm. 2022;2022:4973996.
11. Wang X, Zhang Y, Ye Y, Wang L, Xu Y, Ren S, et al. Neutrophil-to-Lymphocyte Ratio as a Potential Predictive Marker for Epileptic Seizures: Unveiling the "V"-Shaped Link. Mediators of Inflammation. 2025;2025(1):2247724.
12. Wang X, Zhang Y, Ye Y, Wang L, Xu Y, Ren S, et al. Neutrophil-to-Lymphocyte Ratio as a Potential Predictive Marker for Epileptic Seizures: Unveiling the "V"-Shaped Link. Mediators Inflamm. 2025;2025:2247724.
13. Li H, Yang Y, Hu M, Cao X, Ding C, Sun Q, et al. The correlation of temporal changes of neutrophil-lymphocyte ratio with seizure severity and the following seizure tendency in patients with epilepsy. Frontiers in Neurology. 2022;Volume 13 - 2022.
14. Perzynski AT, Ramsey RK, Colón-Zimmermann K, Cage J, Welter E, Sajatovic M. Barriers and facilitators to epilepsy self-

management for patients with physical and psychological co-morbidity. Chronic Illn. 2017;13(3):188-203.
15. Symonds JD, Elliott KS, Shetty J, Armstrong M, Brunklaus A, Cutcutache I, et al. Early childhood epilepsies: epidemiology, classification, aetiology, and socio-economic determinants. Brain. 2021;144(9):2879-91.
16. Ahmad S, Waseem S, Kashif S, Azam M, Akhtar N, Shahzad K, et al. Hematological abnormalities in epileptic patients taking different anti-epileptic drugs: A cross-sectional study. Epilepsy Research. 2021;172:106601.
17. Alapirtti T, Waris M, Fallah M, Soilu-Hänninen M, Mäkinen R, Kharazmi E, et al. C-reactive protein and seizures in focal epilepsy: A video-electroencephalographic study. Epilepsia. 2018;59(5):1036-43.
18. Banote RK, Edling M, Eliassen F, Kettunen P, Zetterberg H, Abramsson A. Beta-amyloid precursor protein-b is essential for Mauthner cell development in the zebrafish in a Notch-dependent manner. Developmental Biology. 2020;461(2):132-44.
19. Chen Z, Brodie MJ, Liew D, Kwan P. Treatment outcomes in patients with newly diagnosed epilepsy treated with established and new antiepileptic drugs: A 30-year longitudinal cohort study. JAMA Neurology. 2018;75(3):279-86.
20. Devinsky O, Vezzani A, O'Brien TJ, Jette N, Scheffer IE, de Curtis M, et al. Epilepsy. Nature Reviews Disease Primers. 2018;4(1):18024.
21. Wang J, Li Y, Wang P, Niu X, Wang H, Li J. Leukocyte and platelet parameters in relation to pregnancy, inflammatory status and prognostic value for pre-eclampsia. Clinical Chemistry and Laboratory Medicine. 2020;58(9):1547-55.
22. Vezzani A, Balosso S, Ravizza T. Neuroinflammatory pathways as treatment targets and biomarkers in epilepsy. Nature Reviews Neurology. 2019;15(8):459-72.
23. Li G, Cleland JC, Sarkis RA. Hematological changes following seizures. Seizure. 2020;77:69-75.
24. Olivo R, Burman R, Jellett A, Lhatoo SD. Inflammatory and immune biomarkers in epilepsy: From pathophysiology to clinical practice. Epilepsy & Behavior. 2021;121:108059.
25. Huang Y, Chi NF, Kuan YC, Chan L, Hu CJ, Chiou HY, et al. Efficacy of phenytoin, valproic acid, carbamazepine and new antiepileptic drugs on control of late-onset post-stroke epilepsy in Taiwan. European Journal of Neurology. 2019;26(9):1264-70.
26. Gao F, Gao Y, Zhang SJ, Zhe X, Meng FL, Qian HJ, et al. Alteration of plasma cytokines in patients with active epilepsy. Acta Neurologica Scandinavica. 2017;135(6):663-9.
27. Rinawati R, Ikawati Z, Rahmawati F, Gofir A. Platelet indices as potential biomarkers for seizure recurrence in epileptic patients. Neurological Sciences. 2021;42(5):1883-9.
28. Wang Z, Chen Z, Yang S, Wang Y, Yu L, Zhang B, et al. The association between neutrophil-to-lymphocyte ratio and post-stroke seizures: A systematic review and meta-analysis. Neurological Sciences. 2020;41(8):2097-105.
29. Perzynski AT, Ramsey RK, Colón-Zimmermann K, Cage J, Welter E, Sajatovic M. Barriers and facilitators to epilepsy self-management for patients with physical and psychological co-morbidity. Chronic Illness. 2017;13(3):188-203.
30. Sarkis RA, Jehi L, Silveira DC, Janigro D. Seizure-related inflammation in epilepsy: From bench to bedside. Epilepsy & Behavior. 2019;99:106474.