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EVALUATION OF THE PREDICTIVE ABILITY OF LABORATORY PARAMETERS IN FIBROMYALGIA SYNDROME COMPARED TO INDIVIDUALS WITH LOCAL MUSCULOSKELETAL PAIN

LOKAL KAS-İSKELET AĞRILI BIREYLERLE KARŞILAŞTIRILDIĞINDA FIBROMİYALJİ SENDROMUNDA LABORATUVAR PARAMETRELERININ ÖNGÖRÜ YETENEĞİNİN DEĞERLENDİRİLMESİ

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ABSTRACT

Introduction: Fibromyalgia syndrome (FMS) is a chronic pain syndrome characterized by widespread pain and various somatic symptoms. This study aims to investigate the differences between FMS patients and individuals with localized musculoskeletal disorders by evaluating inflammatory and metabolic markers that may play a role in FMS pathogenesis.

Methods: This retrospective study included 43 patients diagnosed with FMS and 43 patients with localized musculoskeletal pain, aged between 30 and 65 years. White blood cell count (WBC), red cell distribution width (RDW), mean corpuscular haemoglobin concentration (MCHC), mean platelet volume (MPV), platelet distribution width (PDW), albumin, creatinine, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) values of the participants were recorded. Additionally, CRP/albumin ratio (CAR), neutrophil/lymphocyte ratio (NLR), platelet/lymphocyte ratio (PLR), monocyte/lymphocyte ratio (MLR), and systemic inflammatory indices (SII) of the participants were calculated.

Results: The mean ages were similar between the two groups in our study (Group 1 mean age: 46.76±8.94 years and Group 2 mean age: 46.3±8.67 years). MPV levels were found to be statistically significantly higher in the FMS group compared to the group with localized pain (p=0.02). No statistically significant difference was detected between the two groups in terms of the other evaluated laboratory parameters.

Conclusions: According to our research, we found a statistically significant difference only in MPV levels between FMS patients and controls. This suggests that it may help facilitate the diagnosis of FMS patients.

Keywords: Fibromyalgia, inflammation, laboratory parameters, mean platelet volume, pain

ÖZET

Giriş: Fibromiyalji sendromu (FMS) yaygın ağrı ve çeşitli somatik semptomlarla karakterize bir kronik ağrı sendromudur. Bu çalışmanın amacı, FMS patogenezinde rol oynayabilecek inflamatuar ve metabolik belirteçleri değerlendirerek FMS hastaları ile lokalize kas-iskelet sistemi rahatsızlığı olan bireyler arasındaki farklılıkları araştırmaktır.

Yöntemler: Bu retrospektif çalışmaya 30-65 yaş arası 43 FMS tanılı hasta ve 43 lokalize kas-iskelet bozukluğu olan hasta dahil edildi. Katılımcıların beyaz kan hücresi sayımı (WBC), kırmızı hücre dağılım genişliği (RDW), ortalama eritrosit hemoglobin konsantrasyonu (MCHC), ortalama trombosit hacmi (MPV), trombosit dağılım genişliği (PDW), albümin, kreatinin, eritrosit sedimantasyon hızı (ESR) ve C-reaktif protein (CRP) değerleri not edildi. Ayrıca katılımcıların CRP/albümin oranı (CAR), nötrofil/lenfosit oranı (NLR), trombosit/lenfosit oranı (PLR), monosit/lenfosit oranı (MLR) ve sistemik inflamatuar indeksleri (SII) hesaplandı.

Bulgular: Çalışmamızda yaş ortalamaları her iki grupta da benzerdi (1. grup yaş: 46,76±8,94 yıl ve 2. grup yaş: 46,3±8,67 yıl). FMS grubunda MPV düzeyleri lokalize ağrısı olan gruba göre istatistiksel olarak anlamlı farkla daha yüksek bulundu (p=0,02). Değerlendirdiğimiz diğer laboratuvar parametreleri açısından iki grup arasında istatistiksel anlamlı fark tespit edilmedi.

Sonuç: Araştırmamıza göre FMS hastaları ile kontroller arasında sadece MPV düzeylerinde istatistiksel olarak anlamlı fark bulduk. FMS hastalarının tanısını kolaylaştırmada faydalı olabileceğini düşündürmektedir.

Anahtar Kelimeler: Fibromiyalji, inflamasyon, laboratuvar parametreleri, ortalama trombosit hacmi, ağrı

INTRODUCTION

Fibromyalgia (FMS) is a common cause of chronic pain that affects the muscles and soft tissues. FMS is characterized by a variety of symptoms, including fatigue, cognitive dysfunction, psychiatric issues, and various physical complaints (1, 2). The mechanisms that contribute to the development of FMS are not yet fully understood. However, according to the current mechanism, it is thought to be related to central sensitization, where sensitivity to pain increases due to dysfunction of neural circuits involved in the perception and processing of pain (3). The diagnosis is still made clinically and there are no objective markers that can be used.

Although FMS is considered a non-inflammatory disease, some studies have reported that patients have

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increased levels of C-reactive protein (CRP). This observation suggests the potential presence of low-grade chronic inflammation (4-6). CRP and ESR (erythrocyte sedimentation rate) are laboratory parameters frequently used to assess the presence and severity of inflammation. Although these markers are fast and widely applicable, they have some limitations such as cost, accessibility problems and fluctuations in their results. More stable and reliable indicators are needed to overcome these problems. In this context, the relevance of negative acute-phase reactants like albumin is becoming increasingly significant. CRP-albumin ratio (CAR) is considered a more sensitive and specific marker than CRP levels alone and stands out as a low-cost and accessible biomarker in clinical practice (7).

On the other hand, parameters such as red blood cell distribution width (RDW), platelet distribution width (PDW), and mean platelet volume (MPV), which are routinely reported in the complete blood count (CBC) report, are also associated with inflammation (8-10). The association of RDW with inflammation has been linked to the effects of oxidative stress on erythropoiesis and the release of immature and differently sized erythrocytes into the circulation (11). Studies in different patient groups have shown that inflammatory responses trigger platelet activation via proinflammatory cytokines, which may lead to changes in platelet indices. The role of platelets in inflammation has been associated with the release of cytokines and chemokines, attracting leukocytes and facilitating adhesion to the endothelium at the site of injury (12).

CBC is a widely used and inexpensive test, and subclinical markers of inflammation such as platelet-lymphocyte ratio (PLR), neutrophil-lymphocyte ratio (NLR), monocyte-lymphocyte ratio (MLR), and systemic inflammation index (SII) can be easily obtained (13). These rates have been emphasized as predictors for disease activity in rheumatoid arthritis and ulcerative colitis, survival times of cancer patients, and major cardiac events in diabetic patients (14-18).

In addition to inflammation markers, the relationship between mean corpuscular haemoglobin concentration (MCHC) levels, which reflect tissue oxygenation, and chronic fatigue is also being investigated (19). In this respect, MCHC may be an important indicator for understanding fatigue and other symptoms in FMS patients. Creatinine levels, a product of muscle metabolism, may be affected by environmental and psychological stress as a result of sympathetic nervous system activity (20, 21).

FMS is a chronic musculoskeletal disorder characterized by widespread body pain, in which central sensitization and systemic inflammation are thought to play a role (22, 23). In local musculoskeletal disorders, there is no central sensitisation, but it is caused by injury, inflammation or strain in a specific area of the joint nerve connective tissue (24). We planned to investigate whether there would be a significant difference in FMS evaluation parameters in favour of systemic inflammation by selecting disorders with short-term localised inflammation, which is different from FMS, as a control group.

Despite significant progress in understanding FMS, the lack of reliable biomarkers continues to hinder accurate diagnosis and management strategies. Assessing both inflammatory markers and metabolic changes may enhance the understanding and management of FMS. This study aimed to assess the predictive capacity of laboratory parameters to distinguish FMS patients from individuals with localized musculoskeletal pain.

METHODS

Subjects

A total of 86 female patients aged between 30 and 65 years (FMS group N=43, localized musculoskletal disorders (LMD) N=43) were included in our study. Participants were recruited from the Physical Medicine and Rehabilitation outpatient clinic of Osmangazi University Faculty of Medicine between January 2022 and September 2023. Patients were selected through a cross-sectional file search.

The diagnosis of FMS was established based on the 2016 criteria of the American College of Rheumatology (25). Patients with widespread pain scale scores of 7 and above and symptom severity scale scores of 5 and above were included in the study. The LMD group consisted of patients with a symptom duration of less than one month and a single diagnosis from the following: mild carpal tunnel syndrome, rotator cuff syndrome, knee sprain or strain, or Kellgren & Lawrence grade 1 gonarthrosis. Exclusion criteria included: age below 30 or above 65 years, chronic inflammatory diseases (e.g., Hashimoto's thyroiditis, Crohn's disease, ulcerative colitis, multiple sclerosis), inflammatory rheumatic diseases (e.g., rheumatoid arthritis, Sjögren's disease, ankylosing spondylitis), acute or subacute infections, diabetes mellitus, malignancies, thrombocytopenic conditions associated with bleeding disorders, anticoagulant use, pregnancy, smoking, or incomplete data.

Clinical assessment and laboratory data

The patients' ages and laboratory values were recorded. The serum parameters were obtained from blood test results taken during the outpatient clinic visit, immediately before the diagnosis was made. From the laboratory parameters obtained from the CBC, the values of white blood count (WBC), neutrophils, lymphocytes, platelets, MCHC, MPV, PDW, and RDW, as well as NLR, PLR, and MLR, were calculated respectively by dividing the absolute neutrophil count by the absolute lymphocyte count, the absolute platelet count by the absolute lymphocyte count, and the absolute neutrophil count by the absolute monocyte count.

Additionally, the systemic immune-inflammation index (SII) was calculated using the formula: neutrophils × platelets / lymphocytes.

Among the biochemical parameters, CRP, ESH, creatinine and albumin values were recorded. The CRP/albumin ratio was calculated and noted.

The study adhered to the principles outlined in the Declaration of Helsinki and received ethical approval from the Local Ethics Committee on 31.10.2023 (approval number: 50).

Statistical analysis

Normality of the distribution for each continuous variable was assessed using the Shapiro-Wilk test. Variables with non-normal distributions were analyzed using the Mann-Whitney U-test and reported as medians with interquartile ranges (25th-75th percentiles). Variables that followed a normal distribution were analyzed using the independent samples t-test, with results expressed as mean ± standard deviation. A p-value of less than 0.05 was regarded as statistically significant. All statistical analyses were performed using SPSS software, version 22.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

Out of a total of 110 patient records reviewed, 24 were excluded based on the study's exclusion criteria. The final study cohort comprised 43 patients diagnosed with FMS and 43 patients with LMD. All participants in the study were women. The mean age of participants was 46.7±8.9 years in the FMS group and 46.3±8.6 years in the local pain group. No notable age difference was observed between the groups (p = 0.80). MPV (p=0.02) was significantly higher in the fibromyalgia group compared to the LMD group. No statistically significant differences were identified between the two groups for other laboratory parameters (Table 1).

Table 1. Comparative analysis of laboratory parameters and calculated ratios between groups

	Fibromyalgia group (N=43)	Localized musculoskletal disorders group (N=43)	*P-value*
Age (years)[a]	46.7±8.9	46.3±8.6	0.80*
ESH (mm/h)[a]	17.02±8.5	15.1±6.4	0.25*
CRP (mg/l)[b]	1.8 (1.0-3.5)	1.4 (1.0-2.4)	0.43**
Albumin[a]	4.5±0.2	4.6±0.28	0.50*
Creatinin[b]	0.68 (0.63-0.76)	0.67 (0.61-0.73)	0.34**
PDW[a]	12.3±2.04	12.3±2.05	0.98*
MPV[a]	10.5±0.9	10.01±1.06	**0.02***
WBC (10^3)[b]	6.9 (5.8-8.1)	6.8 (5.8-7.6)	0.73**
PLT (mm^3)[b]	290 (262-334)	301 (255-351)	0.77**
RDW[b]	13.4 (12.8-14.1)	13 (12.5-13.5)	0.051**
MCHC (%)[b]	33.1 (32.8-33.7)	33.3 (32.4-34)	0.63**
NLR[b]	1.7 (1.4-1.9)	1.9 (1.4-2.1)	0.52**
PLR[b]	129.9(113.2-147.2)	138.04(109.2-161)	0.42**
MLR[b]	0.22 (0.19-0.25)	0.21 (0.17-0.29)	0.51**
SII[b]	496.1 (407.4-616.4)	518.7 (375.8-642.8)	0.74**
CAR[b]	0.38 (0.19-0.79)	0.32 (0.22-0.52)	0.45**

[a]: Mean ±SD, [b]: Median (25-75%), *Analyzed by independent samples t-test; **Analyzed by Mann-Whitney U test.

Abbreviations: ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, PDW: Platelet distribution width, MPV: Mean platelet volume, WBC: White blood count, PLT: Platelets, RDW: Red blood cell distribution, NLR: Neutrophil/Lymphocyte Ratio, PLR: Platelet/Lymphocyte Ratio, MLR: Monocyte/ Lymphocyte Ratio, SII: Systemic immune-inflammation index, CAR: CRP/albumin

DISCUSSION

This study aims to analyze the differences between FMS patients and individuals with LMD by evaluating inflammatory and metabolic markers that may contribute to FMS pathogenesis. Central sensitization is considered to be the primary cause of changes in pain perception in FMS. In particular, neuroplastic changes in nerve cells in the spinal cord and brain lead to exaggerated processing of pain signals. Inflammation is thought to play a role in this processing (23). Despite this, no marker has been shown to predict disease severity or activity in FMS in routine use. Metya et al. defined a FMS subgroup with an increased inflammatory response (4). Pro-inflammatory cytokines, including IL-6, IL-8, and TNF-α which mediate inflammatory processes, have been implicated in the pathogenesis of FMS in various studies. Increased levels of cytokines may predispose to hyperalgesia through the increase of substance P (6, 26). Using these cytokines as biomarkers in FMS patients is not possible in clinical practice. Therefore, we investigated whether the systemic inflammatory response markers, blood parameters and ratios (CAR, NLR, PLR, MLR and SII) which are cheaper, and easily accessible, could be used in the diagnosis of fibromyalgia syndrome. Among the laboratory parameters we evaluated in our study, we found only the MPV value to be higher in FMF patients compared to the LMD group.

CRP is a proinflammatory marker whose levels are elevated in chronic inflammatory and rheumatic diseases. Hıra et al. reported higher CRP levels in the FMS group compared to the control group, suggesting that inflammation may contribute to the pathogenesis of FMS (27). However, in some studies, similar to our study, no statistical difference was found in CRP and ESR values when fibromyalgia patients were compared with the control group (28-30). Another suggested marker of inflammation is the CRP-

albumin ratio. The fact that high CRP and low albumin are associated with inflammatory processes suggests that this index may be a better indicator than CRP alone (7). In our study, both groups had similar CAR values. This was also observed by Pamukcu et al. in their study, where no significant difference was found between the control and FMS groups in terms of the CAR parameter (31).

Our findings are consistent with previous studies reporting increased MPV levels in FMS patients (5, 32). MPV is a sensitive measurement of platelet size obtained from CBC. It is considered an indicator of platelet activity, and increased MPV is associated with greater granule content and higher reactivity (33). MPV is an indicator of vascular risk factors and has been found to be increased in some diseases such as diabetes mellitus, acute ischemic stroke, and myocardial infarction (33, 34). In recent years, MPV has been suggested to be used as a marker of rheumatoid arthritis disease activity (10, 35). Proinflammatory cytokines, especially IL-6, may contribute to the increase in TPO and the induction of megakaryopoiesis (36, 37). Haliloglu et al. detected higher MPV in FMS compared to controls, independently of smoking, obesity and inflammation, and emphasized that there may be an increased cardiovascular risk in these patients (28). However, platelet count and MPV levels can be influenced by physiological and pathological conditions (Table 2) (31, 38, 39). Further research and detailed standardization are needed to understand their roles in diseases.

Table 2. Factors Influencing Mean Platelet Volume (MPV)

1. Age
2. Gender
3. Race/ethnicity
4. Genetic variants
5. Body mass index
6. Diet, smoking and alcohol consumption
7. Physical activity
8. Hormonal profile
9. Antiplatelet drugs
10. Preanalytical and analytical procedures

NLR, PLR, MLR and SII are novel inflammatory biomarkers used in various diseases as prognostic factors (11-14). Although it is found to be very useful in determining both disease activity and prognosis in many rheumatic and proliferative diseases, such a relationship was not found in our study. Contrary to our study, Al-Nimer et al. found that NLR and PLR levels of patients with FMS can predict the severity and prognosis of the disease (40). Researchers reported that these indexes were independent predictors of fibromyalgia diagnosis through regression analysis. Similar to our study, Karatas et al. did not find a relationship between NLR, PLR, MLR and FMS (32). A study by Uysal et al. found that the systemic immune-inflammation index (SII) was significantly higher in patients with lateral epicondylitis than in healthy controls (41). Although this finding indicates that the Systemic Immune-Inflammation Index (SII) may be a valuable inflammatory marker for disorders of the musculoskeletal system, our study revealed no significant difference in SII values between the two groups.

One of the primary limitations of our study is its retrospective design. For this reason, the relationship between the patients' pain levels, disease activity and laboratory parameters could not be explained. Another limitation is that our study group was small. Furthermore, the sensitivity, specificity and cut-off values for MPV were not subjected to analysis.

CONCLUSION

MPV levels showed a significant difference between individuals with fibromyalgia and the control group. While this finding suggests a potential association, MPV alone cannot be considered a definitive diagnostic marker for FMS. Further multicenter studies with larger sample sizes are necessary to validate the hematological and biochemical indices, determine their cut-off values, and gain deeper insights into the mechanisms underlying FMS.

Ethics Committee Approval: The study adhered to the principles outlined in the Declaration of Helsinki and received ethical approval from the Eskisehir Osmangazi University Ethics Committee on 31.10.2023 (approval number: 50).

Informed Consent: Not applicable

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REFERENCES

1. Clauw DJ. Fibromyalgia: a clinical review. Jama 2014;311(15):1547-55.

2. Galvez-Sánchez CM, Reyes del Paso GA. Diagnostic criteria for fibromyalgia: critical review and future perspectives. J Clin Med 2020;9(4):1219.
3. Siracusa R, Paola RD, Cuzzocrea S, Impellizzeri D. Fibromyalgia: Pathogenesis, Mechanisms, Diagnosis and Treatment Options Update. Int J Mol Sci. 2021;22(8):3891.
4. Metyas SK, Solyman JS, Arkfeld DG. Inflammatory fibromyalgia: is it real? Curr Rheumatol Rev 2015;11(1):15-7.
5. Korkmaz MD, Ceylan CM. Evaluation of Inflammatory Markers in Fibromyalgia Syndrome. Medical Journal of Istanbul Kanuni Sultan Suleyman 2022;14(3):251-4.
6. Xiao Y, Haynes WL, Michalek JE, Russell IJ. Elevated serum high-sensitivity C-reactive protein levels in fibromyalgia syndrome patients correlate with body mass index, interleukin-6, interleukin-8, erythrocyte sedimentation rate. Rheumatol Int 2013;33(5):1259-64.
7. Yang WM, Zhang WH, Ying HQ, Xu YM, Zhang J, Min QH, et al. Two new inflammatory markers associated with disease activity score-28 in patients with rheumatoid arthritis: albumin to fibrinogen ratio and C-reactive protein to albumin ratio. Int Immunopharmacol 2018;62:293-8.
8. Alghamdi M. Red Blood Cell Distribution Width: A Potential Inexpensive Marker for Disease Activity in Patients with Rheumatic Diseases; Scoping Review. Open Access Rheumatol 2023;15:173-80.
9. Peirovy A, Mahdavi AM, Khabbazi A, Hajialilo M, Sakhinia E, Rashtchizadeh N. Clinical usefulness of hematologic indices as predictive parameters for systemic lupus erythematosus. Lab Med 2020;51(5):519-28.
10. Milovanovic M, Nilsson E, Järemo P. Relationships between platelets and inflammatory markers in rheumatoid arthritis. Clin Chim Acta 2004;343(1-2):237-40.
11. Semba RD, Patel KV, Ferrucci L, Sun K, Roy CN, Guralnik JM, et al. Serum antioxidants and inflammation predict red cell distribution width in older women: the Women's Health and Aging Study I. Clin Nutr 2010;29(5):600-4.
12. Gerrits AJ, Frelinger III AL, Michelson AD. Whole blood analysis of leukocyte-platelet aggregates. Curr Protoc Cytom 2016;78(1):6-15.
13. Liu B, Wang J, Li YY, Li KP, Zhang Q. The association between systemic immune-inflammation index and rheumatoid arthritis: evidence from NHANES 1999–2018. Arthritis Res Ther 2023;25(1):1-10.
14. Fu H, Qin B, Hu Z, Ma N, Yang M, Wei T, et al. Neutrophil-and platelet-to-lymphocyte ratios are correlated with disease activity in rheumatoid arthritis. Clin Lab 2015;61(3-4):269-73.
15. Celikbilek M, Dogan S, Ozbakır O, Zararsız G, Kücük H, Gürsoy S, et al. Neutrophil–lymphocyte ratio as a predictor of disease severity in ulcerative colitis. J Clin Lab Anal 2013;27(1):72-6.
16. Aliustaoglu M, Bilici A, Seker M, Dane F, Gocun M, Konya V, et al. The association of pre-treatment peripheral blood markers with survival in patients with pancreatic cancer. Hepato-gastroenterology 2010;57(99-100):640-5.
17. Kishi Y, Kopetz S, Chun YS, Palavecino M, Abdalla EK, Vauthey JN. Blood neutrophil-to-lymphocyte ratio predicts survival in patients with colorectal liver metastases treated with systemic chemotherapy. Ann Surg Oncol 2009;16:614-22.
18. Azab B, Chainani V, Shah N, McGinn JT. Neutrophil–lymphocyte ratio as a predictor of major adverse cardiac events among diabetic population: a 4-year follow-up study. Angiology 2013;64(6):456-65.
19. Mccully KK, Natelson BH. Impaired oxygen delivery to muscle in chronic fatigue syndrome. Clin Sci 1999;97(5):603-8.
20. Bruce MA, Beech BM, Sims M, Brown TN, Wyatt SB, Taylor HA, et al. Social environmental stressors, psychological factors, and kidney disease. J Investig Med 2009;57(4):583-9.
21. Jung YH, Lee WJ, Lee D, Lee JY, Moon JY, Kim YC, et al. Commonalities and differences in abnormal peripheral metabolites between patients with fibromyalgia and complex regional pain syndrome. Int J Neurosci 2020;130(7):653-61.
22. Häuser W, Fitzcharles MA. Facts and myths pertaining to fibromyalgia. Dialogues Clin Neurosci 2018;20(1):53-62.
23. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain 2011;152(3):2-15.
24. Brukner P, Khan K. Chapter 40: Pain in the Achilles region. In:Brukner & Khan's Clinical Sports Medicine. 5th edition. Sydney: McGraw-Hill, 2017: 865-92.
25. Wolfe F, Clauw DJ, Fitzcharles MA, Goldenberg DL, Häuser W, Katz RL, et al. 2016 Revisions to the 2010/2011 fibromyalgia diagnostic criteria. Semin Arthritis Rheum 2016;46(3):319-29.
26. Rodriguez-Pintó I, Agmon-Levin N, Howard A, Shoenfeld Y Fibromyalgia and cytokines. Immunol Let 2014;61(2):200-3.
27. Hira S, Karatas HG, Haskul I, Gunduz R, Ustun B, Akyuz M. Evaluatıon of relationship between pain score and serum vitamın B12 and folate levels in fibromyalgia patients. Balıkesir Medical Journal, 2(1), 51-9.
28. Aktürk S, Büyükavcı R. Evaluation of blood neutrophil-lymphocyte ratio and platelet distribution width as inflammatory markers in patients with fibromyalgia. Clin Rheumatol 2017;36:1885-9.
29. Ataoglu S, Ankarali H, Samanci R, Ozsahin M, Admis O. The relationship between serum leptin level and disease activity and inflammatory markers in fibromyalgia patients. North Clin Istanb 2018;5(2):102-8.
30. Haliloğlu S, Carlioglu A, Sahiner E, Karaaslan Y, Kosar. A Mean platelet volume in patients with fibromyalgia. Z Rheumatol 2014;73(8):742-5.
31. Pamukcu M, Aydogan RB, Izci TD. Disease activation and laboratory parameters in Fibromyalgia Syndrome: Relationship with C-reactive protein/albumin ratio, neutrophil/lymphocyte ratio, mean platelet volume. Medicine Science. 2021;10 (4):1464-8.
32. Karatas G, Gunduz R. The Significance of inflammation markers in complete blood count in patients with fibromyalgia. MSD 2020;7(1):364-7.
33. Korniluk A, Koper-Lenkiewicz OM, Kamińska J, Kemona H, Dymicka-Piekarska V. Mean platelet volume (MPV): new perspectives for an old marker in the course and prognosis of inflammatory conditions. Mediators Inflamm 2019;17:9213074.
34. Bath PMW, Butterworth RJ. Platelet size: measurement, physiology and vascular disease. Blood Coagul Fibrinolysis 1996;7(2):157-61.
35. Yazici S, Yazici M, Erer B, Erer B, Calik Y, Ozhan H, Ataoglu S. The platelet indices in patients with rheumatoid arthritis: mean platelet volume reflects disease activity. Platelets 2010;21(2):122-5.
36. Kaser A, Brandacher G, Steurer W, Kaser S, Offner FA, Zoller H, et al. Interleukin-6 stimulates thrombopoiesis through thrombopoietin: role in inflammatory thrombocytosis. Blood 2001;98(9):2720-5.
37. Ertenli I, Kiraz S, Öztürk MA, Haznedaroğlu I, Çelik İ, Çalgüneri M. Pathologic thrombopoiesis of rheumatoid arthritis. Rheumatol Int 2003;23(2):49-60.
38. Shameer K, Denny JC, Ding K, Jouni H, Crosslin DR, de Andrade M, Chute CG, Peissig P, Pacheco JA, Li R, Bastarache L, Kho AN, Ritchie MD, Masys DR, Chisholm RL, Larson EB, McCarty CA, Roden DM, Jarvik GP, Kullo IJ. A genome- and phenome-wide association study to identify genetic variants influencing platelet count and volume and their pleiotropic effects. Hum Genet 2014;133(1):95-109.
39. Yazici M, Kaya A, Kaya Y, Albayrak S, Cinemre H, Ozhan H. Lifestyle modification decreases the mean platelet volume in prehypertensive patients. Platelets. 2009 Feb;20(1):58-63.
40. Al-Nimer MSM, Mohammad TAM. Correlation of hematological indices and ratios derived from them with FIQR scores in fibromyalgia. Pak J Med Sci 2018;34(5):1219-24.
41. Uysal A. Investigating the presence of inflammation in lateral epicondylitis with platelet/lymphocyte ratio, neutrophil/lymphocyte ratio, and systemic immune-inflammation index: investigating the presence of inflammation in lateral epicondylitis. Chron Precis Med Res 2023;4(1):29-33.