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Investigation of Peripheral Inflammatory Biomarkers in Trichotillomania

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ABSTRACT

Objective: Immunological factors may play a role in the etiology of trichotillomania (TTM). Peripheral inflammatory markers such as Neutrophil-Lymphocyte Ratio (NLR), Platelet-Lymphocyte Ratio (PLR), Monocyte-Lymphocyte Ratio (MLR), monocyte to high-density lipoprotein (HDL) ratio (MHR), Systemic Immune-Inflammation Index (SII) have not yet been investigated in TTM. This study aimed to compare TTM with healthy control (HC) in terms of various inflammatory markers such as NLR, PLR, MLR, MHR, SII.

Method: The electronic records were examined of patients who were diagnosed with TTM who attended to the outpatient psychiatry clinic between January 2022 and December 2023. 46 TTM and 44 HC were included in the study. Blood samples for 90 participants were examined from electronic records, and NLR, PLR, MLR, and SII values were calculated. Participants with TTM were scored according to disease severity using the Clinical Global Impression–Severity (CGI-S) scale.

Results: NLR, PLR, MLR, MHR, and SII were significantly higher in TTM. No significant correlation was found between CGI-S scores and NLR, PLR, MLR, MHR, and SII values in TTM. According to binary logistic regression analysis, a significant relationship was found between NLR and TTM.

Conclusion: TTM group have elevated peripheral inflammatory markers compared with HC group. Findings of the study support the potential inflammatory process in TTM.

Keywords: Trichotillomania, Inflammation, Neutrophil-Lymphocyte Ratio (NLR), Platelet-Lymphocyte Ratio (PLR), Monocyte -Lymphocyte Ratio (MLR), Monocyte to HDL ratio (MHR), Systemic Immune-Inflammation Index (SII)

INTRODUCTION

Trichotillomania (TTM) is a significant psychiatric disorder characterized by recurrent hair-pulling behavior resulting in noticeable hair loss and functional impairment (American Psychiatric Association 2013). The estimated prevalence of TTM in adults ranges from 0.6% to 3.6%, with women being affected approximately four times more frequently than men (Grant and Chamberlain 2016). The typical age of onset is during late childhood or early adolescence, and comorbid conditions such as anxiety disorders (AD) and mood disorders (MD) are frequently reported in the literature (Grant et al. 2017).

Although the etiology and pathophysiology of TTM have not been fully elucidated, it is classified under obsessive-compulsive and related disorders in the DSM-5 (American Psychiatric Association 2013). This classification is primarily based on the overlapping evidence, albeit limited, regarding the etiological mechanisms of TTM and obsessive-compulsive disorder (OCD). Etiological factors include familial traits and various genetic components such as HoxB8, SAPAP3, and SliTrk5 (Schlosser et al. 1994, Welch et al. 2007, Bienvenu et al. 2009), similar structural and functional abnormalities (Woods and Houghton 2014), neurochemical dysregulations in monoaminergic and glutamatergic systems (Woods and Houghton, 2014) and immunological irregularities (Kutuk et al. 2020). Additionally, factors that disrupt emotional regulation—such as stress—and hormonal fluctuations associated with the menstrual cycle are also considered to play

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a role in the etiology (Christenson et al. 1993, Grant and Chamberlain 2018a).

With recent paradigm shifts in the etiological investigations of psychiatric disorders, neuroinflammation has been increasingly implicated in the pathogenesis of several psychiatric conditions (Najjar et al. 2013). Although research on TTM in this context remains limited, existing studies suggest that inflammation may also be involved in this disorder. One study reported lower salivary levels of IL-1β, IL-6, IL-8, and TNF-α in TTM patients (Grant and Chamberlain 2018b), whereas another study conducted in children reported elevated peripheral blood levels of TNF-α, IL-6, and IL-17, along with decreased IL-4 levels (Kutuk et al. 2020). These findings suggest that the inflammatory response in TTM may indicate a systemic rather than localized inflammatory process. The evidence supporting the use of N-acetylcysteine—known for its regulatory effects on the immune system—in the treatment of TTM also lends support to the hypothesis that immune dysregulation may contribute to TTM's etiology (Grant et al. 2009).

Inflammatory markers such as neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), monocyte-to-HDL ratio (MHR), and the Systemic Immune-Inflammation Index (SII), which are derived from peripheral blood, have not yet been investigated in TTM. Considering the relationship between systemic inflammatory response and both neutrophilia and lymphopenia, NLR has been proposed as a marker of chronic low-grade inflammation (Zahorec 2021). Platelets and monocytes are also thought to be involved in both innate and adaptive immune responses. Platelets interact with other components of the immune system and participate in the release of various cytokines and proinflammatory molecules, while monocytes contribute to phagocytosis, cytokine production, and antigen presentation (Cheng et al. 2022). Accordingly, PLR and MLR are also considered indicators of chronic low-grade inflammation, similar to NLR (Hrubaru et al. 2022).

Studies have demonstrated that psychiatric disorders such as schizophrenia (SZ), mood disorders, attention-deficit/hyperactivity disorder (ADHD), Alzheimer's disease, and OCD are associated with elevated NLR and PLR levels (Forget et al. 2017, Mazza et al. 2018, Herdi et al. 2020, Zulfic et al. 2020, Bhikram and Sandor 2022, Ceyhun and Gürbüzer 2022). Although studies on MLR are more limited, elevated MLR levels have also been reported in SZ and mood disorders (Cheng et al. 2022).

SII, a newer biomarker compared to others, is calculated using platelet count and NLR (SII = Platelet × Neutrophil / Lymphocyte) (Yang et al. 2020). Because it incorporates neutrophil, lymphocyte, and platelet counts, SII is considered a more comprehensive and sensitive indicator of inflammatory response than NLR or PLR alone. Given the roles of neutrophils in inflammatory activity, lymphocytes in immune response, and platelets in inflammation and immune modulation, SII provides a more integrated perspective, reflecting both proinflammatory activity and immunosuppression (Köse 2024). Studies have reported elevated SII levels in various psychiatric disorders, including SZ, ADHD, bipolar disorder (BD), and major depressive disorder (MDD) (Ceyhun and Gürbüzer 2022, Wei et al. 2022, İnaltekin and Yağcı, 2023, Cui et al. 2023).

Another biomarker increasingly studied in recent years as an indicator of inflammation and oxidative stress is the MHR. HDL is believed to exert anti-inflammatory effects by suppressing the proinflammatory and pro-oxidative functions of monocytes through mechanisms such as inhibiting macrophage migration, preventing LDL oxidation, and blocking monocyte activation (Ganjali et al. 2018). Given that low HDL and high monocyte levels are associated with inflammation, MHR may be considered a supporting biomarker for the presence of inflammatory processes in psychiatric disorders (Qiu et al. 2024). Higher MHR levels have been reported in psychiatric disorders such as SZ and mood disorders compared to control groups (Sahpolat et al. 2021, Wei et al. 2022, Korkmaz and Kızgın 2023, Kulacaoğlu et al. 2023), and high MHR scores have also been linked to aggression in SZ (Cheng et al. 2023).

Studies investigating the relationship between inflammatory response and symptom severity in psychiatric disorders have shown that systemic inflammation may influence symptom severity. For instance, elevated IL-6 and CRP levels have been associated with more severe depressive symptoms in MDD (Valkanova et al. 2013); increased IL-6 and TNF-α levels have been correlated with negative and cognitive symptoms in SZ (Goldsmith et al. 2018); and elevated inflammatory markers have been observed during manic episodes in BD (Dickerson et al. 2013). Additionally, higher PLR values have been correlated with depressive symptom severity (Kayhan et al. 2017). These findings support the relationship between inflammation and symptom severity and suggest that inflammatory biomarkers may be useful not only in etiological assessment but also in tracking the clinical course of illness.

The primary aim of this study is to compare the levels of inflammatory markers—NLR, PLR, MLR, MHR, and SII—between individuals diagnosed with TTM and healthy controls, as these biomarkers have not yet been investigated in TTM. The secondary aim is to evaluate the relationship between the severity of illness and levels of inflammation. The hypotheses of this study are as follows: i) Inflammatory markers reflecting systemic immune response (NLR, PLR, MLR, MHR, and SII) will be significantly higher in the TTM group than in the control group; ii)

NLR, PLR, and MLR will serve as predictive indicators for TTM diagnosis in regression analyses; iii) In TTM patients, symptom severity scores will be significantly associated with levels of NLR, PLR, MLR, MHR, and SII.

METHODS

This retrospective, single-center study examined the electronic medical records of patients aged 18 to 65 who presented to the outpatient psychiatry clinic between January 2022 and December 2023 and were diagnosed with trichotillomania (TTM) according to DSM-5 criteria. The study protocol was approved by the Ankara Training and Research Hospital Human Research Ethics Committee on December 6, 2023, with decision number E-23-1884.

Exclusion criteria were as follows: individuals with any psychiatric disorder other than major depressive disorder (MDD) or anxiety disorders (AD); those without available blood samples within the year prior to their admission; those who had used psychiatric medications within the three months preceding blood sampling; those using medications affecting the immune system; and those with hematological diseases, autoimmune disorders, or malignancies. Furthermore, participants were excluded if signs of active infection—such as elevated CRP, erythrocyte sedimentation rate, or leukocytosis—were detected at the time of blood sampling.

For the healthy control (HC) group, 44 individuals were selected using a systematic random sampling method from among those who visited the hospital between January and December 2023 for pre-employment or university registration health screenings. Inclusion criteria for the HC group included: normal scores on the subscales and global symptom index of the SCL-90-R; no psychopathology detected during an unstructured clinical interview; and absence of any comorbid conditions such as autoimmune, infectious, cardiovascular, malignant, or rheumatologic diseases.

Of the 55 patients initially diagnosed with TTM, 3 were excluded due to comorbid psychiatric diagnoses, 3 for active medication use, 2 for systemic illnesses potentially affecting the immune system, and 1 for signs of active infection. However, patients with comorbid MDD (n=6) or AD (n=4) were not excluded. Ultimately, 46 patients with TTM and 44 healthy controls who met the inclusion criteria were enrolled in the study.

Data Collection Tools

Sociodemographic Data

Participants' ages and sexes were obtained from electronic hospital records.

Blood Sampling

Blood samples were collected from the antecubital vein between 08:00 and 10:00 AM following a 10–12 hour fasting period. The samples were drawn into EDTA-containing hematology tubes and analyzed within one hour.

Complete blood count data from all 90 participants were reviewed electronically, and the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII) were calculated. Due to missing HDL data for 12 TTM patients, the monocyte-to-HDL ratio (MHR) was calculated for only 78 participants (TTM: 34; HC: 44).

Clinical Global Impression Scale (CGI)

The Clinical Global Impression Scale (CGI), developed by Guy and colleagues, is a three-item scale used to assess the severity and progression of psychiatric disorders (Guy 1976). In this study, the Severity of Illness subscale (CGI-S) was employed. CGI-S scores range from 1 (not at all ill) to 7 (among the most extremely ill). Based on the frequency of hair-pulling behavior, the extent of the affected area, and the impact on daily functioning, each patient's severity score was determined during the clinical interview and recorded retrospectively from medical records.

Symptom Checklist-90-Revised (SCL-90-R)

The SCL-90-R is a 90-item self-report scale used to screen for psychological symptoms across ten dimensions. The Turkish validity and reliability study of the scale was conducted by Dağ (Dağ 1991).

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 23.0 (IBM Corp., Armonk, NY, USA, 2015). A p-value < 0.05 was considered statistically significant. Categorical variables (e.g., gender, CGI-S categories) were expressed as frequencies and percentages, while continuous variables (e.g., age, CGI-S score, NLR, PLR, MLR, MHR, SII) were reported as mean, standard deviation, minimum, maximum, and median values. Group comparisons were conducted using the Mann–Whitney U test and independent samples t-test. As the continuous variables were not normally distributed, relationships among them were examined using Spearman's correlation coefficient. To evaluate the predictive power of NLR, PLR, and MLR for TTM diagnosis, both unadjusted and age- and sex-adjusted binary logistic regression analyses were conducted. The minimum required sample size was determined using G*Power version 3.1.9.2. with a power of 0.80 and an alpha error of 0.05, the minimum sample size required for adequate statistical power was calculated as 52 participants.

RESULTS

Demographic and Clinical Characteristics

There were no statistically significant differences between the TTM and healthy control (HC) groups in terms of age or sex. Sociodemographic data and Clinical Global Impression–Severity (CGI-S) scores of the participants are presented in Table 1.

Table 1. Sociodemographic data and clinical global impressions scale severity scores

	TTM	HC	Statistic*
Age (mean ± SD)	26.57±6.73	27.20±7.13	p=0.674
Gender			p=0.535
Male (n, %)	9 (19.5%)	11 (25%)	
Female (n, %)	37 (80.5%)	33 (75%)	
CGI-Severe (n, %)			
3	20 (43.5%)		
4	15 (32.6%)		
5	7 (15.2%)		
6	4 (8.7%)		
CGI-Severe (mean ± SD)	3.89±0.97		

CGI: clinical global impressions scale; TTM: trichotillomania; HC: healthy control; SD: standart deviation. *p <0.0 5 was considered statistically significant

Inflammatory Markers

Levels of NLR, PLR, MLR, MHR, and SII were significantly higher in the TTM group compared to the HC group (Z = -3.608, p = 0.001, r = -0.23; Z = -2.252, p = 0.024, r = -0.14; Z = -2.325, p = 0.020, r = -0.15; Z = -2.119, p = 0.034, r = -0.12; Z = -3.263, p = 0.001, r = -0.22, respectively). Detailed results are provided in Table 2.

Correlation Analysis

No statistically significant correlations were found between CGI-S scores and levels of NLR, PLR, MLR, MHR, or SII in the TTM group (r = -0.212, p = 0.157; r = -0.145, p = 0.338; r = 0.081, p = 0.591; r = 0.089, p = 0.556; r = -0.189, p = 0.219, respectively).

Predictive Analyses

Binary logistic regression analysis adjusted for age and sex was conducted to evaluate whether NLR, PLR, and MLR could predict a TTM diagnosis. The analysis revealed a significant association between NLR and TTM (p = 0.004, OR = 3.303, 95% CI = 1.478–7.383). Full results are provided in Table 3.

Table 2. Inflammatory markers comparison

Marker	Group	Mean ± SD (Min-Max)	Statistics
Leukocytes	TTM	7.02±1.30 (4.10–9.50)	t=-1.008, p: 0.31[b]
	HC	6.73±1.40 (4.17–9.20)	
Neutrophils	TTM	4.32±0.95 (2.45–6.61)	Z=-2.531, r=-0.17, p=0.011*[a]
	HC	3.84±1.14 (1.89–7.46)	
Lymphocytes	TTM	2.10±0.52 (1.15–3.60)	t=-1.802, p=0.075[b]
	HC	2.31±0.58 (1.10–3.49)	
Platelets	TTM	283.3±63.1 (175–441)	t=-1.725 p=0.088[b]
	HC	261.6±55.2 (149–399)	
Monocytes	TTM	0.45±0.11 (0.17–0.70)	t=-1.308, p=0.19[b]
	HC	0.42±0.10 (0.25–0.70)	
HDL	TTM	47.35±9.32 (26–74)	Z=-2.095, r=-0.11, p=0.047*[a]
	HC	50.11±8.69 (28–72)	
NLR	TTM	2.15±0.58 (1.39–3.53)	Z=-3.608, r=-0.23, p<0.001*[a]
	HC	1.74±0.63 (0.92–4.01)	
PLR	TTM	143.16±48.64 (69.42–253.28)	Z=-2.252, r=-0.14, p=0.024*[a]
	HC	119.09±34.97 (54.98–224.32)	
MLR	TTM	0.23±0.09 (0.10–0.49)	Z=-2.325, r=-0.15, p=0.020*[a]
	HC	0.19±0.06 (0.10–0.33)	
MHR	TTM	10.04±3.69 (3.95–19.23)	Z=-2.119, r=-0.12, p=0.034*[a]
	HC	8.61±2.61 (4.55–18.57)	
SII	TTM	619.52±210.20 (271.30–979.42)	Z=-3.263, r=-0.22, p=0.001*[a]
	HC	454.56±168.07 (202.76–825.80)	

TTM: trichotillomania; HC: healthy control; NLR: neutrophil-lymphocyte ratio; PLR: platelet-lymphocyte ratio; MLR: monocyte-lymphocyte ratio; MHR: monocyte-HDL ratio; SII: systemic immune-inflammation index; a: Mann-Whitney U test; b: independent samples t-tests; SD: standart deviation; Min: minumum; Max: maximum. *p <0.0 5 was considered statistically significant

Table 3. Logistic regression analysis for predicting TTM diagnosis

Marker	Coefficient (B)	SE	P-value	Odds Ratio (OR)	95% Confidence Interval (95%CI)
NLR	1.195	0.410	0.004*	3.303	1.478–7.383
NLR-ADJ	1.203	0.408	0.003*	3.329	1.470–7.475
MLR	0.027	0.242	0.087	1.131	1.007–1.309
MLR-ADJ	0.029	0.244	0.084	1.138	1.012–1.327
PLR	0.013	0.008	0.101	1.013	0.997–1.030
PLR-ADJ	0.015	0.007	0.099	1.017	1.002–1.044

TTM: trichotillomania; HC: healthy control; NLR: neutrophil-lymphocyte ratio; PLR: platelet-lymphocyte ratio; MLR: monocyte-lymphocyte ratio; ADJ: adjusted for gender and age.
*p <0.0 5 was considered statistically significant

DISCUSSION

In this study, levels of NLR, PLR, MLR, MHR, and SII were significantly higher in individuals with TTM compared to healthy controls. Logistic regression analysis demonstrated that NLR was significantly associated with TTM diagnosis. However, no significant correlation was found between disease severity and inflammatory markers. Previous studies exploring the inflammatory etiology of TTM are scarce and primarily focus on cytokine levels, often yielding inconsistent results (Grant and Chamberlain 2018a; Kutuk et al. 2020). To the best of our knowledge, this is the first study to examine peripheral inflammatory markers such as NLR, PLR, MLR, MHR, and SII in TTM.

The most noteworthy finding of our study is the significant association between NLR and TTM diagnosis. Existing literature supports NLR as a reliable marker of low-grade chronic inflammation and reports elevated levels in various psychiatric disorders such as MDD, schizophrenia (SZ), and OCD (Mazza et al. 2018, Cheng et al. 2022). Given that neutrophils play a central role in acute inflammation and lymphocytes in immune regulation, an elevated NLR in TTM may reflect a pro-inflammatory state and impaired immune regulation (Kolaczkowska and Kubes 2013). Nonetheless, the small effect sizes observed suggest that NLR alone may not be a strong clinical biomarker. While our findings contribute to the literature, further research is required to confirm their generalizability.

Although PLR and MLR were also significantly elevated in the TTM group, their predictive value for TTM diagnosis was not supported by logistic regression analysis. This aligns with previous findings suggesting that NLR is a more robust indicator of systemic inflammation compared to PLR and MLR (Cheng et al. 2022). Unlike PLR and MLR, NLR simultaneously reflects both innate (via neutrophils) and adaptive (via lymphocytes) immune responses, thereby offering a more comprehensive assessment of inflammatory activity (Mazza et al. 2018). The lack of predictive power for PLR and MLR, despite their group differences, may be due to the limited sample size or their inherently lower specificity as inflammatory markers.

SII and MHR are newer biomarkers with limited investigation in psychiatric populations. SII has been used to evaluate inflammatory response and predict prognosis in conditions such as malignancy, cardiovascular disease, dermatologic, and cerebrovascular disorders (Jomrich et al. 2020, Yang et al. 2020, Huang et al. 2022, Turan et al. 2022). Similarly, MHR has been associated with complications and poor prognosis in cardiovascular disease, and has been observed to be elevated in malignancies, dermatologic, and cerebrovascular diseases (Cetin et al. 2016, Ganjali et al. 2018, Sirin et al. 2020, Zhang et al. 2023, Gkantzios et al. 2023).

Psychiatric research has reported elevated SII and MHR levels in patients with psychotic disorders, generalized anxiety disorder, BD, MDD, and ADHD compared to controls (Sahpolat et al. 2021, Wei et al. 2022, İnaltekin and Yağcı 2023, Cui et al. 2023, Korkmaz and Kızgın 2023, Kulacaoğlu et al. 2023, Xiaoyu et al. 2023, Kılıç et al. 2023, Kaşak 2025). However, SII and MHR have not, to our knowledge, been studied in OCD or related disorders. The significantly elevated SII and MHR levels in the TTM group observed in our study support the hypothesis of an inflammatory component in TTM's etiology. That said, the inability to calculate MHR for 12 patients due to missing HDL values limits the generalizability of this finding.

No significant associations were found between disease severity and inflammatory markers, which may be due in part to the retrospective design of the study and the use of CGI-S scores instead of disorder-specific severity scales. Previous research on this topic has yielded conflicting results. For instance, Šagud et al. (2023) reported that associations between inflammatory parameters and negative symptoms in SZ varied by symptom subdomain and that correlation effect sizes were generally low. While some studies found a relationship between PLR and depressive symptom severity (Kucukkarapinar 2024), others reported no significant association between NLR, PLR, MLR, SII, and symptom severity in mood disorders (Paniagua ve ark. 2023). Thus,

our findings are consistent with literature suggesting a weak or inconsistent relationship between inflammatory markers and symptom severity in psychiatric disorders. Moreover, reliance on CGI-S instead of a TTM-specific scale such as the Massachusetts General Hospital Hairpulling Scale may have masked potential associations.

Our findings are consistent with growing evidence indicating the involvement of immune dysregulation and inflammation in various psychiatric disorders including SZ, MDD, OCD, autism spectrum disorder (ASD), and ADHD (Bulut et al. 2021). The observation of similar inflammatory markers across these heterogeneous disorders suggests that TTM may fall within this transdiagnostic spectrum. Furthermore, inflammatory biomarkers may assist in subtype classification or treatment response prediction. In this context, the elevated SII and MHR levels found in TTM patients may contribute not only to research on TTM-specific biomarkers but also to the broader literature on transdiagnostic inflammation in psychiatry.

The identification of shared etiological pathways among distinct neuropsychiatric disorders may promote a shift from phenotype-based diagnoses toward etiology-based treatment approaches. Such paradigms could be particularly beneficial for treatment-resistant patients or those exposed to polypharmacy. Characterizing specific pathophysiological mechanisms may improve understanding of TTM's neurobiological underpinnings and contribute to the development of targeted therapeutic strategies.

This study has several limitations. First, its retrospective design and relatively small sample size may limit statistical power and generalizability. Second, psychiatric diagnoses were not confirmed using structured clinical interviews, and the potential effects of MDD and AD comorbidities—common in TTM—on inflammatory markers could not be fully controlled. Although efforts were made to control for confounding variables such as age, sex, comorbidities, and medication use, other potential influences on inflammation, including smoking, physical activity, and body mass index (BMI), were not assessed. Additionally, the limited ability of peripheral markers to reflect central nervous system inflammation due to the blood-brain barrier may weaken their relevance to central neuroinflammation. A key strength of this study is that it is the first to evaluate peripheral inflammatory markers such as NLR, PLR, MLR, MHR, and SII in patients with TTM. The use of logistic regression analysis allowed for an assessment not only of between-group differences but also of the predictive value of these biomarkers for TTM diagnosis. Importantly, these markers are derived from routine complete blood count tests, making them accessible and potentially applicable in clinical settings.

CONCLUSION

In conclusion, our findings support the presence of a potential inflammatory process in trichotillomania. Given the limited research on this topic, further prospective studies with larger samples and disorder-specific measurement tools are warranted.

Ethics Committe Approval: The study protocol was approved by the Ankara Training and Research Hospital Human Research Ethics Committee on December 6, 2023, with decision number E-23-1884.

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