DOI: 10.4274/gulhane.galenos.2025.35582
Gulhane Med J 2025;67(4):252-258

Assessment of routine hematological and biochemical parameters as markers of systemic inflammation in patients with pterygium

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Cite this article as: Yılmaz AC, Yılmaz H, Çakır İnce BA, Ayyıldız Ö. Assessment of routine hematological and biochemical parameters as markers of systemic inflammation in patients with pterygium. *Gulhane Med J.* 2025;67(4):252-258.

Date submitted:
07.04.2025
Date accepted:
19.09.2025
Publication Date:
11.12.2025

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Keywords: Complete blood count, serum lipid levels, pterygium, inflammation, oxidative stress

ABSTRACT

Aims: This study aimed to evaluate whether routine hematological and biochemical indices reflect systemic inflammation in patients with pterygium and to compare these indices with healthy controls.

Methods: This retrospective study included patients with clinically diagnosed pterygium and age- and sex-matched healthy controls. Main inclusion criteria were documented ophthalmological examination and available complete blood count (CBC) and routine biochemistry results; patients with systemic diseases or conditions affecting laboratory parameters were excluded. The participants' CBC values, glucose, creatinine, and blood lipid profiles were reviewed. The primary endpoint was the comparison of systemic inflammatory markers—neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and monocyte-to-high-density-lipoprotein ratio (MHR)—between the two groups.

Results: A total of 107 patients with pterygium (mean age 56.57±14.47 years; 63.6% male) and 104 controls (mean age 56.05±13.28 years; 69.3% male) were included. Only hematocrit and white blood cell (WBC) count were significantly higher in patients with pterygium (p=0.044 and p=0.009, respectively). There were no significant differences in NLR (p=0.108), PLR (p=0.462), MLR (p=0.190), or MHR (p=0.134) between the groups. The patient group exhibited significantly higher total cholesterol and triglyceride (TG) levels (p=0.004 and p=0.007, respectively).

Conclusions: This study indicated that routine systemic inflammatory indices (NLR, PLR, MLR, MHR) did not differ significantly between patients with pterygium and healthy controls, whereas hematocrit, WBC, total cholesterol, and TG levels were higher in the pterygium group.

Introduction

Pterygium is a fibrovascular growth originating from the subconjunctival tissue and extending onto the corneal surface, typically occurring in the interpalpebral region and most commonly located nasally (1). The etiology of pterygium involves multiple risk factors, including immune system dysregulation, genetic predisposition, chronic environmental irritation caused by exposure to ultraviolet (UV) radiation, hot and dry air, and wind, and the duration of exposure to such conditions (2). One of the proposed pathogenic mechanisms is that UV radiation causes damage to limbal stem cells, leading to the activation of tissue growth factors, which subsequently induce angiogenesis and fibroblast proliferation (3). Human papillomavirus has also been implicated in the pathogenesis of pterygium (4). Although

several risk factors for pterygium are well documented, the precise mechanisms by which these factors promote cell proliferation remain incompletely understood. However, the prevailing hypothesis suggests that chronic exposure to UV radiation increases free radical production (5). Consequently, free radicals, oxidative stress, and inflammation are increasingly recognized as key contributors to the pathophysiology of pterygium (6).

Cytokines that play roles in the immune response and inflammation have been found to be overexpressed in pterygium tissue (7). Likewise, increased expression of the inflammatory cytokine tumor necrosis factor-alpha (TNF-α) has been observed in pterygium samples (8). Recently, there has been increasing interest in the role of systemic inflammatory mechanisms in the development of pterygium. Increased levels of both local and systemic endothelial progenitor cells (EPCs) have been documented (9). Because EPCs play a key role in postnatal neovascularization, it has been proposed that their increased levels—triggered by proinflammatory mediators—may contribute to angiogenic processes underlying pterygium pathogenesis (9). Additionally, patients with pterygium were found to have increased serum immunoglobulin E (IgE) levels and mast cell counts (10). IgE may play a role in promoting inflammation and angiogenesis during pterygium development by causing mast cell degranulation (10). Elevated concentrations of interleukin-6 (IL-6), IL-17A, and nitric oxide have likewise been detected in the tear fluid and serum of patients with pterygium (11). Additionally, accumulating evidence indicates that pterygium behaves as a tumor-like proliferative disorder in which abnormal cellular growth and deoxyribonucleic acid (DNA) replication are closely linked to disturbances in cholesterol metabolism and lipid peroxidation (12). Increased levels of both high-density lipoprotein (HDL) and low-density lipoprotein (LDL) have been reported to be potential risk factors for pterygium, particularly among individuals younger than 50 years or with a normal body mass index (13).

These findings, together with systemic inflammatory and immunological mechanisms, support the notion that some systemic metabolic pathways may contribute substantially to the underlying pathophysiological mechanisms of pterygium. Recent evidence has reinforced the involvement of inflammatory processes in the development of pterygium, irrespective of the initial trigger. While these complex molecular mediators have been shown to be involved in the development of pterygium, there is a need for accessible biomarkers that reliably indicate the presence of systemic inflammation.

This study aimed to assess systemic inflammatory biomarkers by examining complete blood count (CBC) and lipid profile measurements. For this purpose, the neutrophil-to-lymphocyte (NLR), platelet-to-lymphocyte (PLR), monocyte-to-lymphocyte (MLR), and monocyte-to-HDL (MHR) ratios were calculated. These readily available laboratory parameters have been recognized as markers of systemic inflammation in various conditions, particularly in cardiovascular conditions and eye diseases, including glaucoma, dry eye, age-related macular degeneration, pseudoexfoliation syndrome, and keratoconus (14-18). In their study evaluating systemic inflammatory biomarkers across multiple eye diseases, including pterygium, Shirvani et al. (19) found that patients with pterygium exhibited a significantly elevated NLR. The main objective of this study was to assess systemic inflammation in patients with pterygium through analysis of standard hematological and biochemical parameters derived from CBCs and lipid profiles. The study sought to determine whether these commonly available laboratory parameters could serve as reliable biomarkers of systemic inflammatory status and metabolic alterations associated with pterygium.

Methods

This retrospective analysis examined data from 107 eyes of patients diagnosed with pterygium at a tertiary teaching hospital over 11 months (January-November 2024). The study adhered to the ethical principles stated in the Declaration of Helsinki and received approval from the Gülhane Scientific Research Ethics Committee of the University of Health Sciences (approval no.: 2025-71, date: 11.02.2025).

All consecutive patients with pterygium who presented to our clinic during the defined study period were included, regardless of age or sex. Inclusion criteria included having undergone a comprehensive ophthalmological examination with fully documented findings and having CBC and routine biochemistry test results available. Exclusion criteria included refractive error with an absolute value greater than 5.00 diopters, intraocular pressure above 22 mmHg, incomplete medical records or laboratory results, history of ocular inflammatory disease, previous intraocular surgery, and history of a systemic condition. The control group included individuals matched for age and sex who had received a standard ophthalmological examination, a CBC, and biochemical testing. These individuals had no ophthalmological disease other than refractive error within the limits specified in the inclusion criteria and no history of systemic disease.

Laboratory parameters assessed in this study represent well-known indicators of systemic inflammation and may be influenced by age, sex, and various acute or chronic conditions. To minimize bias, comparisons were made with an age- and sex-matched control group. In the patient group, many factors that could affect these parameters were considered, such as acute infections, systemic diseases (e.g., diabetes mellitus, hypertension, dyslipidemia), and medication use (e.g., anti-inflammatory agents, lipid-lowering drugs). Individuals meeting these criteria were excluded from the study.

Blood analyses were performed at the Biochemistry Department of Gülhane Training and Research Hospital. An automated hematology analyzer (Sysmex XN-1000, Sysmex Corporation, Kobe, Japan) was used for CBC testing. Red blood cells, white blood cells (WBC), neutrophils, monocytes, lymphocytes, and platelet (PLT) counts, as well as hemoglobin, mean platelet volume (MPV), platelet distribution width (PDW), red cell distribution width (RDW), and plateletcrit (PCT) values were obtained. Biochemical measurements, including glucose, creatinine, serum lipids [HDL, LDL, triglyceride (TG), and total cholesterol], were conducted using a biochemistry analyzer (Cobas e602, Roche Diagnostics GmbH, Mannheim, Germany). Subsequently, MLR, NLR, PLR, and MHR values were calculated. All CBC and biochemical measurements were collected from venous blood samples drawn after at least 8 hours of fasting. All procedures were performed after patients had been provided with detailed information and their written informed consent had been obtained.

Statistical Analysis

Statistical analyses were conducted using Jamovi software (v.1.6). Continuous variables are summarized as mean ± standard deviation, while categorical variables are presented as frequency (n) and percentage (%). The normality of the data distribution was assessed using the Shapiro-Wilk test. Based on the results of the normality test, comparisons of continuous variables (age, CBC, and biochemical parameters) between the two groups were conducted using the Student's t-test for normally distributed data or the Mann-Whitney U test for non-normally distributed data. Pearson's chi-square test was used to assess differences in the distribution of categorical variables between the groups. A p-value of <0.05 was considered statistically significant.

Due to the exploratory design of the study involving multiple comparisons, effect sizes (such as Cohen's d) and 95% confidence intervals were also provided to aid in interpreting the magnitude of the observed effects. Additionally, the adequacy of the sample size was assessed using G*Power software (version 3.1.9.7). A post-hoc power analysis was performed for an Independent samples t-test including 107 patients and 104 control subjects. Under the assumption of a conventional medium effect size (Cohen's d=0.5) and a Type I error rate (α) of 0.05, the analysis revealed that statistical power (1-β) was 95%. This high statistical power indicates that the sample size was sufficient to detect significant between-group differences, should such differences exist.

Results

The medical records of 134 patients diagnosed with pterygium were retrospectively reviewed. A total of 27 patients were excluded due to incomplete ophthalmological records (n=5), missing CBC and biochemistry results (n=7), systemic conditions such as diabetes, hypertension, or collagen vascular disease (n=7), use of topical cyclosporine for dry eye (n=5), or a diagnosis of pseudoexfoliation syndrome (n=3). Accordingly, the analysis incorporated data from 107 patients with pterygium and 104 healthy individuals as the control group.

The mean age of the patients with pterygium was 56.57±14.47 years, and that of healthy controls was 56.05±13.28 years (p=0.708). Demographic characteristics and CBC test results of the patients are shown in Table 1. No significant differences were found between the two groups in terms of age, sex, and MLR, NLR, PLR, and MHR values. However, hematocrit and WBC count were significantly higher among patients with pterygium (p=0.044 and 0.009, respectively). Biochemical analysis showed that total cholesterol and TG levels were significantly higher in patients with pterygium (p=0.004 and p=0.007, respectively). A detailed comparison of biochemical parameters is provided in Table 2.

Discussion

In this study assessing systemic inflammatory markers in patients with pterygium, the patient group had significantly higher levels of WBC, hematocrit, total cholesterol, and TG compared with controls. However, all other parameters were comparable between the groups.

UV radiation and environmental factors are recognized as significant contributors to the development of pterygium (2). UV exposure, in particular, has been shown to damage limbal stem cells, resulting in the activation of growth factors, angiogenesis, and fibroblast proliferation (3,4). Additionally, prolonged exposure to UV radiation has been linked to increased production of free radicals (5). Lipid peroxidation produces aldehydes, which are markedly elevated in pterygium specimens (19). Mitochondrial DNA is highly susceptible to oxidative damage due to limited repair mechanisms, making it a vulnerable target in ocular cells. Its instability contributes to mitochondrial dysfunction, cellular damage, and the development of age-related and chronic eye diseases. Increased mitochondrial reactive oxygen species (ROS) levels have been strongly associated with ophthalmological disorders affecting both the anterior and posterior segments (6). Pyroptosis—a form of programmed inflammatory cell death—is triggered by ROS via *NLRP3*/caspase-1 activation, thereby amplifying inflammation, fibrosis, and epithelial-mesenchymal transition in pterygial tissues (19).

Several studies have demonstrated that an imbalance between oxidative stress and antioxidant defenses, together with local inflammation, is a central factor in the development of pterygium (6,20). Patients with pterygium exhibit decreased antioxidant capacity, and UV radiation, a primary risk factor, has been demonstrated to induce oxidative DNA damage, leading to apoptosis and unregulated cell proliferation (19). It has also been

Table 1. Demographic characteristics and complete blood count data of patients with pterygium and control group

	Patients (n=107) Mean ± SD	Controls (n=104) Mean ± SD	95% CI of difference (Lower)	95% CI of difference (Upper)	p
Age (years)	56.57±14.47	56.05±13.28	-3.251	4.295	0.708
Sex, n (%) Female Male	 39 (36.4) 68 (63.6)	 32 (30.7) 72 (69.3)			0.467
Hemoglobin (g/dL)	14.07±1.77	13.72±1.63	-0.11933	0.80935	0.090
Hematocrit (%)	42.37±4.56	41.28±4.40	-0.12684	2.31099	**0.044***
MPV (fL)	10.14±0.90	10.31±1.07	-0.44661	0.09237	0.436
PDW (%)	11.75±2.00	11.65±1.90	-0.42350	0.63741	0.632
RDW (%)	13.49±1.73	13.60±1.53	-0.55210	0.33869	0.324
Plateletcrit (%)	0.24±0.06	0.24±0.05	-0.01759	0.01442	0.782
White blood cell (x10³/µL)	7.70±2.21	7.01±2.07	0.10255	1.26822	**0.009***
Neutrophil count (x10³/µL)	4.79±2.02	4.30±1.80	-0.03939	1.00525	0.050
Monocyte count (x10³/µL)	0.61±0.27	0.57±0.20	-0.02296	0.10719	0.272
Lymphocyte count (x10³/µL)	2.13±0.66	2.17±0.71	-0.23026	0.14577	0.916
Platelet count (x10³/µL)	240.03±54.37	235.14±61.20	-10.80860	20.59490	0.431
MLR	0.33±0.23	0.28±0.13	-0.00500	0.10004	0.190
NLR	2.52±1.66	2.12±1.07	0.02415	0.78636	0.108
PLR	124.71±53.59	117.79±44.88	-6.52206	20.35015	0.462
MHR	0.012±0.006	0.011±0.006	-0.00075	0.00280	0.134

*p<0.05
SD: Standard deviation, CI: Confidence interval, MPV: Mean platelet volume, PDW: Platelet distribution width, RDW: Red cell distribution width, MLR: Monocyte to lymphocyte ratio, NLR: Neutrophil to lymphocyte ratio, PLR: Platelet to lymphocyte ratio MHR: Monocyte to high-density ratio

Table 2. Biochemical data of patients with pterygium and control group

	Patients (n=107) Mean ± SD	Controls (n=104) Mean ± SD	95% CI of difference (Lower)	95% CI of difference (Upper)	p
Glucose (mg/dL)	114.39±47.50	87.53±9.10	17.49126	36.21687	0.528
Creatinine (mg/dL)	0.74±0.21	0.73±0.18	-0.04544	0.06475	0.889
Total cholesterol (mg/dL)	192.85±45.72	174.29±28.69	8.15722	28.94757	**0.004***
LDL (mg/dL)	116.21±38.70	94.87±26.81	12.27945	30.40046	0.463
HDL (mg/dL)	50.64±11.68	52.87±14.78	-5.84619	1.37657	0.549
Triglyceride (mg/dL)	144.41±94.40	111.3±55.77	11.98302	54.22402	**0.007***

*p<0.05
SD: Standard deviation, CI: Confidence interval, LDL: Low-density lipoprotein, HDL: High-density lipoprotein

shown that nitric oxide levels are elevated and that superoxide dismutase (SOD) and catalase levels are decreased in primary pterygium (8). Chronic UV exposure leads to depletion of enzymatic antioxidants (SOD, catalase, glutathione peroxidase) and increased oxidative stress in pterygial tissue. Although the cell may upregulate defense mechanisms (e.g., glutathione S-transferase, ALDH3A1), this compensation is often insufficient under sustained UV stress (19). ROS activate mitogen-activated protein kinase pathways including extracellular signal-regulated kinase, c-Jun N-terminal Kinase, and p38, which in turn induce expression of matrix metalloproteinase-1 (MMP-1) and pro-inflammatory cytokines IL6 and IL8. ROS and signaling pathways induce MMPs that degrade Bowman's layer, thereby facilitating fibrovascular tissue invasion onto the cornea. UV also activates the nuclear factor kappa B pathway in conjunctival and keratocyte cells, promoting transcription of genes related to inflammation, cell survival, and angiogenesis (e.g., TNFα, IL1, IL6, cyclooxygenase-2) (21).

Histopathological research has emphasized the important contribution of local inflammation to pterygium development (22). Exposure to UVB radiation elevates IL-6 and IL-8 levels in the epithelial layers of pterygium tissue (23). These pro-inflammatory cytokines contribute to pterygium development by sustaining local chronic inflammatory processes. Overall, this evidence strongly supports the contribution of local inflammation to the development of pterygium.

Research on systemic inflammation in pterygium remains limited. Various hematological parameters serve as indicators of systemic inflammatory status, and in this study, we assessed these markers using patients' peripheral blood. Ratios such as NLR, PLR, and MHR have been used to indicate inflammation in systemic disorders, including cardiovascular conditions (24). These markers have also been studied in eye disorders, including glaucoma, dry eye syndrome, age-related macular degeneration, pseudoexfoliation syndrome, and keratoconus (14-18, 25,26). A meta-analysis by Shirvani et al. (19) found that NLR values were notably elevated in individuals with pterygium compared with the control group. Gokmen and Gokmen (27) also observed elevated NLR in patients with pterygium and attributed this finding to chronic inflammation and neovascularization. However, Gokmen and Gokmen (27) found no significant changes in MPV, PCT, or PDW in these patients, and suggested that this observation can be explained by the predominance of the fibrous component and fibroblast activity over neovascularization process in pterygium pathology. Two studies from Türkiye evaluated NLR and PLR in individuals with pterygium and found that these values did not differ significantly from those in healthy controls (27-29). Our results align with these observations, as we found no significant differences in MPV, PLT, PCT, PDW, PLR, NLR, or MLR between the patient group and controls.

Heterogeneous findings have been reported for these inflammatory markers in patients with pterygium, particularly for NLR, which is frequently emphasized in the literature. These discrepancies may be related to variability in inclusion criteria; however, the exclusion criteria across studies were generally quite strict and similar, considering these laboratory parameters can be influenced by many conditions. In their meta-analysis, Shirvani et al. (19) noted that most of the included studies were from Türkiye and China, which they cited as a limitation. To determine whether variations in ethnicity or other demographic characteristics influence the results, it is essential to conduct comparative studies involving patient groups representing diverse ethnicities under standardized conditions. Additionally, the severity and size of pterygium may impact systemic inflammatory parameters. However, existing studies lack consistent classification, grading, or standardization of these factors. Moreover, methodological differences—such as variations in laboratory techniques and in the timing of blood sample collection—may contribute to the discrepancies observed in the literature.

Monocytes are key players in inflammation, with elevated counts observed in several inflammatory disorders. HDL cholesterol exerts antioxidant and anti-inflammatory effects; hence, MHR is commonly used to reflect inflammatory status in both systemic and ocular conditions. Belviranli et al. (30) observed that MHR levels did not differ notably between the pterygium cohort and the control group, whereas NLR was higher in the pterygium cohort, potentially reflecting systemic inflammation. Similarly, our study detected no significant difference in MHR between the groups. Kilic Toprak et al. (31) provided evidence of systemic oxidative stress in patients with pterygium. HDL participates in antioxidant and detoxification processes, whereas elevated TG levels may counteract these protective effects (31,32). Kilic and Guven (28) suggested that an imbalance between oxidative and antioxidative processes in patients with pterygium may be attributable to decreased HDL levels and elevated TG levels. In our study, TG levels were significantly higher in patients with pterygium, supporting this hypothesis. However, no significant between-group difference in HDL levels was observed. Moreover, findings from prior studies examining the association between pterygium and serum lipids have been inconsistent (33). For instance, the Singapore Malay Eye Study found a positive link between pterygium and serum total cholesterol, whereas a multiethnic Asian cohort showed no such association (34). Additionally, a cross-sectional investigation of Han and Manchu populations in Hebei, China (35) suggested HDL as a risk factor for pterygium in men, while a separate retrospective case-control study found no connection between HDL and pterygium (36). In another case-control study, LDL levels were notably elevated in individuals with advanced pterygium (36). Although the mechanisms linking low HDL cholesterol to pterygium formation have not been fully elucidated, it has been suggested that intracellular alterations in cholesterol homeostasis may be involved in pterygium formation (37,38). While the elevated TG and total cholesterol levels observed in our study lend support to this theory, additional research is needed to clarify whether these results stem from lifestyle factors (such as diet) or are directly linked to underlying biochemical mechanisms. Kilic and Guven (28) proposed that the observed reduction in HDL levels, accompanied by elevated TG levels, in individuals with pterygium may warrant further exploration of HDL's potential role as a novel antioxidant therapy. Interestingly, the same study found no notable differences in NLR, PLR, or MHR between the pterygium group and controls, consistent with our findings. RDW, which reflects variations in the size of red blood cells, serves as an indicator of inflammation and oxidative stress. Elevated RDW levels have been reported in various ocular diseases, including retinopathy and allergic conjunctivitis (39,40). Kurtul et al. (29) observed increased RDW values in patients with pterygium, although the precise mechanism linking RDW to pterygium remains unclear. They proposed that elevated expression of inflammatory cytokines in pterygium tissue may impair erythrocyte maturation, resulting in the release of immature erythrocytes into the circulation, thereby increasing erythrocyte size heterogeneity and RDW values. In contrast, our study did not find significant changes in RDW. As noted for other inflammatory parameters, factors such as ethnicity, demographic characteristics, and pterygium

size may influence RDW results. Achieving methodological standardization across studies is essential for generating more reliable and comparable results.

The study's retrospective nature and the exclusion of patients with incomplete medical records or laboratory data may introduce selection bias. Furthermore, primary and recurrent pterygium may exhibit distinct inflammatory profiles, which are not differentiated in the current analysis. Prospective studies incorporating detailed assessments of UV exposure duration, occupational risks, and dietary habits, which may affect systemic inflammatory status and oxidative balance, are necessary to better understand the fundamental biochemical pathways involved. Additionally, the evaluation of a broader panel of inflammatory and oxidative stress biomarkers, including proinflammatory cytokines (e.g., IL-6, TNF-α), would provide more comprehensive insight into the inflammatory process associated with pterygium. However, these biomarkers are not specific to pterygium and may be influenced by many inflammatory conditions. Therefore, it is important to consider that systemic markers may not directly indicate the presence of pterygium-related inflammation. A main statistical constraint of this research is the heightened possibility of committing a Type I error, since separate analyses were conducted on multiple biomarkers without applying corrections for multiple testing. While this approach increases the chance of spurious findings, it was considered appropriate given the exploratory and hypothesis-generating nature of this research. Consequently, our results should be interpreted as preliminary, requiring confirmation through future hypothesis-driven studies with adequate statistical power specifically designed to validate these specific associations.

Conclusion

The results of our study revealed that systemic inflammatory markers did not differ notably between individuals with pterygium and healthy subjects. Currently, there is no clear consensus regarding the correlation between systemic inflammatory markers and pterygium. These results suggest that systemic inflammation, unlike the local inflammatory response, might make a minor contribution to the mechanisms underlying pterygium. However, focusing on more sensitive and specific oxidative stress markers, such as nitric oxide, SOD, and catalase, which play roles in the oxidant-antioxidant balance, would help elucidate biochemical mechanisms. Additionally, validation of elevated TG levels as an indicator of systemic oxidative stress in a larger patient cohort could inform future research on antioxidant therapeutic strategies for the management of pterygium.

Ethics

Ethics Committee Approval: The study adhered to the ethical principles outlined in the Declaration of Helsinki, and ethics approval for our study was obtained from the ethics committee at the Gülhane Scientific Research Ethics Committee of the University of Health Sciences (approval no.: 2025-71, date: 11.02.2025).

Informed Consent: All patients provided written informed consent to participate in the study after receiving a detailed explanation of the study procedures.

Acknowledgments

We thank the hospital's medical records department for providing access to patient files.

Footnotes

Author contributions

Surgical and Medical Practices: A.C.Y., Ö.A., Concept: A.C.Y., Ö.A., Design: A.C.Y., H.Y., Ö.A., Data Collection or Processing: A.C.Y., H.Y., B.A.Ç.İ., Analysis or Interpretation: A.C.Y., H.Y., B.A.Ç.İ., Literature Search: A.C.Y., B.A.Ç.İ., Writing: A.C.Y.

Conflict of Interest: The authors declare that they have no conflict of interest.

Financial Disclosure: The authors received no financial support for the research, authorship, and/or publication of this article.

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