RESEARCH PAPER

Systemic and Tissue Inflammatory Biomarkers in Benign and Malignant Periocular Tumors: A Comprehensive Review on Diagnostic and Prognostic Perspectives

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ABSTRACT

Background: Chronic inflammation plays a fundamental role in carcinogenesis by promoting genomic instability, angiogenesis, and immune dysregulation. In periocular tumors, both systemic and local inflammatory processes have been implicated in tumor development and progression, yet their clinical significance remains incompletely characterized.

Objective: To evaluate the diagnostic and prognostic relevance of inflammatory biomarkers in benign and malignant periocular tumors.

Methods: A structured literature review was conducted to analyze studies assessing systemic inflammatory indices derived from complete blood count namely neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII) as well as tissue-level cytokine expression, including interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), and interleukin-10 (IL-10)., in periocular tumors.

Results: Malignant periocular tumors demonstrated significantly elevated systemic inflammatory indices, particularly NLR and SII, compared with benign lesions. Increased inflammatory ratios were associated with more aggressive tumor subtypes and higher metastatic risk. At the tissue level, elevated expression of IL-1β, TNF-α, and IL-10 was observed in malignant eyelid tumors and was linked to tumor presence and potential recurrence. In contrast, benign lesions such as xanthelasma and chalazion predominantly exhibited localized inflammatory infiltration without consistent systemic hematologic elevation.

Conclusion: Systemic inflammatory markers, especially NLR and SII, together with cytokine profiling, may serve as accessible adjunctive tools for differentiating benign from malignant periocular tumors and for supporting prognostic stratification. Integration of hematologic and tissue-based inflammatory markers may enhance clinical decision-making in periocular oncology.

Keywords: *Eyelid Neoplasms, Inflammation, Biomarkers, Neutrophils, Cytokines*

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INTRODUCTION

Cancer remains one of the leading causes of morbidity and mortality worldwide, with a steadily increasing global incidence over recent decades (1,2). Although ocular malignancies account for a relatively small proportion of overall cancer cases, they remain clinically significant due to their potential for local invasion, metastasis, and functional morbidity (3,4). Periocular tumors constitute a heterogeneous spectrum of lesions arising from the eyelid and surrounding adnexal tissues, encompassing both benign and malignant entities with diverse histopathological origins and biological behavior (5–7). The majority of eyelid tumors are benign; however, malignant lesions such as basal cell carcinoma, squamous cell carcinoma, melanoma, and sebaceous cell carcinoma carry substantial risks of recurrence and metastatic dissemination (6,7).

Chronic inflammation has been increasingly recognized as a central mechanism in carcinogenesis, contributing to tumor initiation, progression, and metastatic spread (8,9). Inflammation-related carcinogenesis may result from

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persistent exposure to infectious agents, environmental irritants, and autoimmune processes that induce genetic instability and epigenetic alterations (8–10). Systemic inflammatory activation has also been associated with increased cancer risk and disease progression, further supporting the biological link between inflammation and malignancy (11). Activated inflammatory cells release reactive oxygen species, reactive nitrogen species, and reactive sulfur species, which promote DNA damage and sustain a protumorigenic microenvironment (8). This inflammatory niche enhances angiogenesis, immune modulation, and stromal remodeling, thereby facilitating malignant transformation (8,9).

Systemic inflammatory responses in malignancy can be quantified using complete blood count–derived indices, including the neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, monocyte-to-lymphocyte ratio, and the systemic immune-inflammation index, which reflect the balance between protumor inflammation and host immune surveillance (12). Elevated neutrophil-to-lymphocyte ratio and systemic immune-inflammation index have been shown to significantly differentiate malignant from benign periocular lesions and may influence preoperative assessment (12). In periocular basal cell carcinoma, inflammatory markers such as neutrophil-to-lymphocyte ratio and systemic immune-inflammation index have been reported to be significantly higher compared with healthy controls (13). Furthermore, inflammatory indices including neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, and systemic immune-inflammation index have demonstrated discriminatory value among different cutaneous malignancies, with higher levels observed in more aggressive histological subtypes and in cases associated with increased metastatic risk (14). In addition, granulomatous inflammatory lesions may clinically mimic periocular tumor recurrence, highlighting the complexity of inflammation–tumor interactions in this anatomical region (15).

At the tissue level, proinflammatory cytokines including interleukin-1β, tumor necrosis factor-α, and interleukin-10 have been implicated in eyelid tumorigenesis and local inflammatory activation (16,17). Increased expression of interleukin-1β and tumor necrosis factor-α in malignant eyelid tumors has been associated with tumor presence and may serve as potential indicators of local recurrence (16,17). In benign periocular conditions such as xanthelasma, histopathologic and immunohistochemical analyses have demonstrated chronic inflammatory infiltrates, including CD3+ T cells and CD163+ macrophages, indicating localized immune activation (18,19). Similarly, inflammatory alterations have been observed in chalazion, including significant changes in neutrophil-to-platelet ratios, suggesting localized inflammatory dysregulation despite non-malignant pathology (20). Case-based evidence has also reported inflammatory plasma cell infiltration, including IgG4-positive cells, in periocular cystic lesions, further supporting the role of immune-mediated mechanisms in benign tumor-like conditions (21). In contrast, certain infectious periocular lesions such as molluscum contagiosum may demonstrate localized inflammatory infiltrates without significant systemic hematologic abnormalities (22).

Despite accumulating evidence supporting the role of systemic and tissue inflammatory biomarkers in periocular lesions, available studies remain heterogeneous and fragmented across benign and malignant entities (12–22). A structured synthesis integrating complete blood count–derived inflammatory indices, cytokine profiles, and immunohistochemical markers is therefore necessary to clarify their diagnostic and prognostic utility in periocular tumors (12–22).

This review aims to comprehensively evaluate systemic and tissue inflammatory biomarkers in benign and malignant periocular tumors, with particular emphasis on their potential role in differentiating tumor behavior and guiding clinical decision-making.

METHODS

Study Design and Reporting Standard

This review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to ensure methodological transparency and reproducibility. The study selection process followed a structured PRISMA flow framework, including identification, screening, eligibility assessment, and final inclusion stages as illustrated in the flow diagram presented in the manuscript (Figure 1).

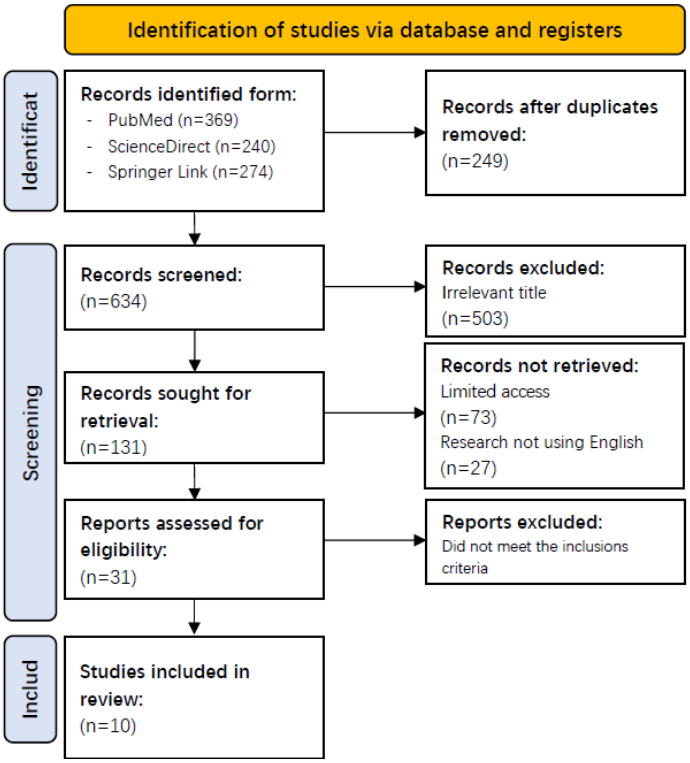

Figure 1. PRISMA flowchart.

Search Strategy

A comprehensive literature search was performed across three electronic databases: PubMed, ScienceDirect, and Google Scholar. The search strategy used combinations of the keywords "tumor," "periocular," "biomarker," and "inflammation," including relevant synonyms where applicable. Boolean operators were applied using the structure ("tumor") AND ("periocular") AND ("biomarker") AND ("inflammation") The final search was conducted in January 2025. In addition to database searching, reference lists of eligible studies were manually screened to identify additional relevant publications. Only full-text articles published in English were considered eligible.

Eligibility Criteria

Studies were included if they investigated inflammatory biomarkers in periocular tumors, were published within the last ten years, focused on benign and/or malignant periocular tumors, and reported differences in inflammatory markers between benign and malignant lesions or described relevant inflammatory profiles. Studies were excluded if they were not written in English, were unavailable in full text, or did not specifically address periocular tumors or inflammatory biomarkers. Conference abstracts without accessible full manuscripts were also excluded.

Study Selection Process

The initial database search yielded 883 records. Duplicate records were removed prior to screening, resulting in 249 unique articles. Titles and abstracts were independently screened to assess relevance. A total of 503 records were excluded due to irrelevance to the study objective. One hundred thirty-one articles were sought for full-text retrieval; 73 articles were not retrieved due to limited access and 27 were excluded because they were not written in English. Thirty-one full-text articles were assessed for eligibility. Of these, 21 were excluded for not meeting the predefined inclusion criteria. Ultimately, ten studies were included in the final qualitative synthesis. Any discrepancies in study selection were resolved through discussion to achieve consensus.

Data Extraction

Data extraction was performed systematically using a standardized data collection framework. The extracted variables included author and year of publication, study design, level of evidence, sample size, tumor type, type of inflammatory biomarker evaluated, and principal outcomes including statistical significance and reported cut-off values where available.

Quality Assessment and Risk of Bias

All included studies were appraised using the Oxford Centre for Evidence-Based Medicine 2011 Levels of Evidence framework to categorize methodological strength. Given that most included studies were retrospective analyses, case-control studies, or case reports, potential risks of bias such as selection bias, small sample size, and heterogeneity of tumor types were acknowledged. Formal quantitative risk-of-bias scoring tools were not applied due to variability in study design and reporting structure (Figure 2)..

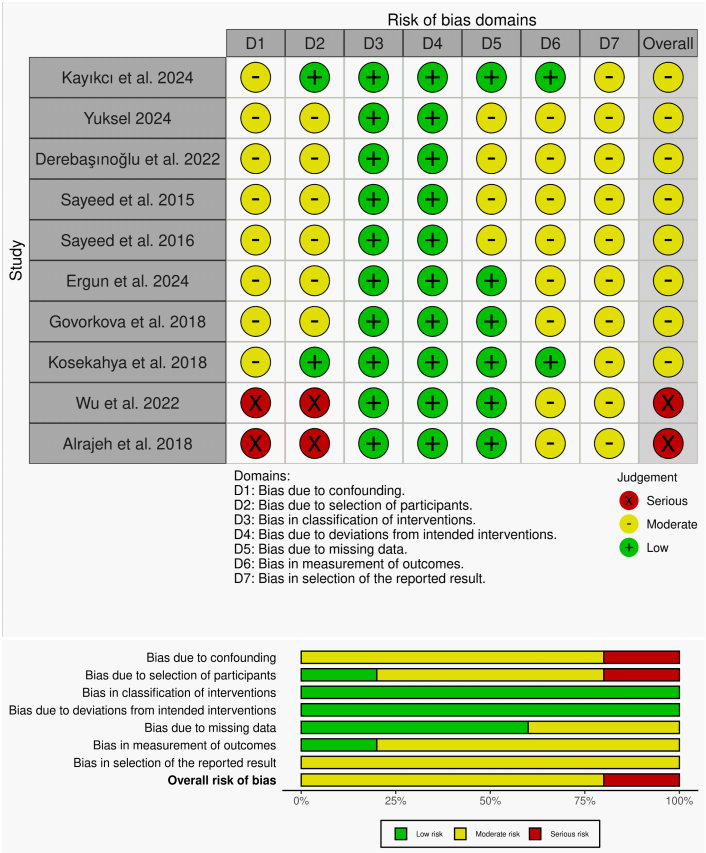

Figure 2. ROBINS-I bias assessment results.

Data Synthesis

Due to substantial heterogeneity in study design, tumor subtypes, biomarker parameters, and outcome measurements, quantitative meta-analysis was not feasible. Therefore, a qualitative synthesis approach was undertaken. Findings were categorized into three principal domains: systemic inflammatory biomarkers derived from complete blood count indices such as neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, monocyte-to-lymphocyte ratio, and systemic immune-inflammation index; cytokine-based tissue biomarkers including interleukin-1β, tumor necrosis factor-α, and interleukin-10; and histopathological and immunohistochemical inflammatory markers including CD3+, CD163+, cyclooxygenase-1, nitric oxide synthase, and IgG4 plasma cell ratios. The synthesized evidence was analyzed to evaluate the diagnostic and prognostic implications of inflammatory biomarkers in differentiating benign and malignant periocular tumors (Table 1)..

Table 1. Summary of Inflammatory Biomarkers in Benign and Malignant Periocular Tumors

Author (Year)	Study Design / Level	Tumor Type	Sample Size	Biomarkers Evaluated	Main Findings	Clinical Implication
Kayıkcı et al. (2024).	Prospective non-interventional / Level II	Benign vs Malignant periocular tumors	88 benign, 46 malignant	NLR, PLR, SII	NLR and SII significantly higher in malignant tumors ($p<0.001$; $p=0.002$).	NLR and SII useful for differentiating benign from malignant lesions
Yuksel (2024).	Retrospective / Level II	Periocular BCC	15 BCC, 20 controls	NLR, MLR, PLR, RDW, SII, SIRI, HALP	NLR and SII significantly elevated in BCC	Suggests systemic inflammatory activation in periocular BCC
Derebaşınoğlu et al. (2022).	Retrospective / Level II	BCC, SCC, MM	144 BCC, 84 SCC, 29	NLR, LMR, PLR, SII, Plt×NLR	Higher inflammatory indices in	Predictive value for metastatic risk and tumor

			MM		SCC; PLR ≥180.7 associated with 10.3-fold increased lymph node metastasis risk	aggressiveness
Sayeed et al. (2015).	Case-control / Level II	Malignant eyelid tumors	25 cases, 26 controls	IL-1β, TNF-α, IL-10	All cytokines significantly higher in tumor tissues	Highlights role of inflammatory microenvironment in tumorigenesis
Sayeed et al. (2016).	Case-control / Level II	Malignant eyelid tumors	38 cases, 26 controls	IL-1β, TNF-α, IL-10	IL-1β and TNF-α significantly elevated; lower levels in safe margins may predict recurrence	Potential biomarker for local recurrence
Ergun et al. (2024).	Retrospective / Level II	Xanthelasma	27 cases, 27 controls	NLR, MLR, SII, AISI	Elevated markers in patients but not statistically significant	Mild systemic inflammatory activity
Govorkova et al. (2018).	Retrospective / Level II	Xanthelasma	9 cases, 8 controls	CD3+, CD163+, COX-1, NOS	Significant lymphocytic and macrophage infiltration; increased COX-1 and NOS expression	Evidence of chronic localized inflammation
Kosekahya et al. (2018).	Retrospective / Level III	Chalazion	200 cases, 200 controls	NLR, PLR, NPR	NPR significantly lower in chalazion (p=0.008).; NLR and PLR not significant	NPR may reflect local inflammatory response
Wu et al. (2022).	Case report / Level IV	Apocrine hidrocystoma	1 case	IgG4/IgG plasma cell ratio	High IgG4-positive plasma cell infiltration; normal serum IgG4	Suggests possible IgG4-related inflammatory mechanism
Alrajeh et al. (2018).	Case report / Level IV	Molluscum contagiosum	1 case	Histopathology, CBC	Local inflammatory infiltrate; normal systemic hematologic profile	Local inflammation without systemic involvement

The synthesized data indicate that malignant periocular tumors consistently demonstrate elevated systemic inflammatory markers, particularly neutrophil-to-lymphocyte ratio and systemic immune-inflammation index, along with increased proinflammatory cytokine expression such as interleukin-1β and tumor necrosis factor-α at the tissue level, supporting their diagnostic and potential prognostic relevance. In contrast, benign periocular lesions predominantly exhibit localized inflammatory infiltration without consistent systemic hematologic elevation. These findings suggest that malignant tumors are characterized by both systemic and local inflammatory activation, whereas benign lesions primarily reflect localized immune responses, highlighting the potential value of integrating blood-based and tissue-based inflammatory biomarkers in clinical assessment.

DISCUSSION

This literature review emphasizes the biological relevance of inflammation in the development, progression, and clinical behavior of periocular tumors. Chronic inflammation has been recognized as a critical enabling characteristic of carcinogenesis, contributing to tumor initiation and progression through sustained cellular and molecular dysregulation (23,24). Large clinicopathological series have demonstrated that eyelid tumors arise within a biologically active microenvironment where epithelial, stromal, and immune components interact dynamically (23,25–27). The periocular region, because of its exposure to ultraviolet radiation and environmental irritants, represents a site susceptible to repeated inflammatory stimulation that may predispose to neoplastic transformation (24,28).

At the mechanistic level, inflammation-related carcinogenesis is characterized by persistent oxidative stress, genomic instability, and the production of proinflammatory mediators that promote angiogenesis and tissue remodeling (32–34). Reactive oxygen and nitrogen species generated during chronic inflammation induce DNA damage and epigenetic alterations that facilitate malignant transformation (32,39). In parallel, inflammatory cytokines and growth factors create a tumor-supportive microenvironment that enhances proliferation, invasion, and metastatic potential (33,34). Thus, inflammation does not merely accompany tumor growth but actively contributes to tumor biology.

Inflammatory cells within the tumor microenvironment including neutrophils and macrophages can adopt tumor-promoting phenotypes. These cells release matrix metalloproteinases, vascular endothelial growth factor (VEGF), and other mediators that support extracellular matrix degradation and angiogenesis (33,34). Although immune surveillance mechanisms attempt to eliminate malignant cells, persistent inflammatory signaling may impair adaptive immune responses and facilitate tumor immune escape (33,34). This dual role of inflammation underscores its importance in understanding periocular tumor behavior.

1. INFLAMMATORY MARKERS IN MALIGNANT PERIOCULAR TUMORS

Cancer-associated systemic inflammation has been increasingly recognized as a determinant of prognosis and survival across multiple malignancies (34,35). Systemic inflammatory activation reflects tumor–host interaction and can be assessed using hematologic parameters derived from complete blood count (CBC) analysis (35,36). These markers provide a simple, reproducible, and cost-effective method for evaluating tumor-related inflammation in clinical settings.

A. Neutrophil-to-Lymphocyte Ratio (NLR), Monocyte-to-Lymphocyte Ratio (MLR), Platelet-to-Lymphocyte Ratio (PLR), and Systemic Immune-Inflammation Index (SII)

Peripheral blood–derived inflammatory indices including NLR, MLR, PLR, and SII reflect the balance between protumor inflammatory activity and host antitumor immune function (35–37). Elevated neutrophil counts are associated with tumor-promoting activities such as reactive oxygen species production and extracellular matrix degradation, whereas lymphocytes, particularly cytotoxic T cells, are essential for tumor immune surveillance (33,34). Consequently, an increased NLR indicates dominance of protumor inflammation over protective immunity. Monocytes, which can differentiate into tumor-associated macrophages, further contribute to angiogenesis and immune suppression within the tumor microenvironment (33,34).

Platelets facilitate tumor progression by protecting circulating tumor cells from immune destruction and by promoting angiogenesis through growth factor release (34,36). The systemic immune-inflammation index (SII), integrating neutrophil, platelet, and lymphocyte counts, therefore represents a composite indicator of systemic inflammatory burden (35–37).

In periocular malignancies, Kayıkcı et al. demonstrated that NLR and SII were significantly elevated in malignant eyelid tumors compared with benign lesions, suggesting their value in differential diagnosis (12). Similarly, Yuksel reported higher NLR and SII levels in patients with periocular basal cell carcinoma compared with healthy controls (13). These findings indicate that even tumors with predominantly local growth patterns can induce measurable systemic inflammatory responses. Furthermore, Derebaşınoğlu et al. observed that NLR, PLR, and SII were significantly higher in squamous cell carcinoma than in basal cell carcinoma and were associated with increased risk of lymph node metastasis (15). Elevated inflammatory indices were correlated with more aggressive tumor behavior, supporting their prognostic relevance. These findings are consistent with broader oncologic evidence demonstrating that systemic inflammatory markers are associated with tumor progression and survival outcomes (35–37).

B. Cytokine-Mediated Tumor Microenvironment

Beyond systemic inflammatory indices, local cytokine expression within tumor tissue provides critical insight into the biological behavior of malignant periocular tumors. Cytokines act as regulatory proteins that modulate immune responses, cell proliferation, apoptosis, and angiogenesis (38,39). In the context of malignancy, dysregulated cytokine signaling contributes to the establishment of a tumor-supportive microenvironment that facilitates progression and immune evasion (33,34). Interleukin-1β (IL-1β) is a potent proinflammatory cytokine involved in innate immune activation and amplification of inflammatory cascades. Its overexpression has been associated with tumor progression and enhanced angiogenic activity in various malignancies (38,39). Tumor necrosis factor-alpha (TNF-α), another multifunctional cytokine, exerts dual roles depending on the microenvironment; however, persistent TNF-α signaling has been linked to increased tumor invasiveness and metastatic behavior (38,39).

In malignant eyelid tumors, Sayeed et al. demonstrated significantly elevated tissue levels of IL-1β, TNF-α, and IL-10 compared with non-neoplastic controls (16). Their subsequent study further suggested that lower levels of IL-1β and TNF-α at histologically clear surgical margins were associated with reduced risk of local recurrence, highlighting the potential prognostic relevance of cytokine profiling (17). Interleukin-10 (IL-10)., although classically described as an anti-inflammatory cytokine, has been implicated in tumor immune escape mechanisms. Elevated circulating IL-10 levels have been correlated with adverse prognosis in metastatic melanoma and other malignancies. These findings suggest that IL-10 may contribute to suppression of effective antitumor immune responses within the tumor microenvironment (41).

2. INFLAMMATORY MARKERS IN BENIGN PERIOCULAR TUMORS

Although benign periocular tumors lack metastatic potential, inflammatory processes remain integral to their pathogenesis. Many benign eyelid lesions arise in the setting of chronic local irritation, glandular obstruction, or lipid metabolism disturbances, where immune cell infiltration and cytokine activity contribute to lesion development (29,31).

A. Hematologic Inflammatory Markers in Benign Lesions

Peripheral inflammatory indices derived from complete blood count analysis have also been evaluated in benign periocular tumors. Kayıkcı et al. reported that NLR, PLR, and SII values were significantly lower in benign lesions compared with malignant tumors, reinforcing their discriminative utility (12). In patients with xanthelasma palpebrarum, Ergun et al. observed elevated inflammatory markers, including NLR and MLR; however, these differences did not reach statistical significance, suggesting predominantly localized rather than systemic inflammation. This aligns with clinical observations that xanthelasma represents a lipid-laden inflammatory process confined to the dermis(18). Similarly, Kosekahya et al. investigated inflammatory ratios in chalazion and found that although NLR and PLR were not significantly different from controls, the neutrophil-to-platelet ratio (NPR) was significantly altered, indicating distinct inflammatory characteristics in this benign granulomatous condition (20). In contrast, routine hematologic parameters were generally normal in patients with molluscum contagiosum, further supporting the concept that benign infectious lesions do not typically induce systemic inflammatory activation (22). Moreover, systemic inflammatory indices in benign periocular tumors tend to demonstrate mild or inconsistent alterations, reflecting limited systemic involvement compared with malignant counterparts.

B. Histopathological and Immunohistochemical Inflammatory Markers

Histopathologic and immunohistochemical analyses provide deeper understanding of localized inflammatory responses in benign periocular lesions. In xanthelasma, Govorkova et al. reported statistically significant chronic lymphocytic infiltration on hematoxylin–eosin staining, resembling early atherosclerotic plaque formation (19). Immunohistochemical analysis revealed increased expression of CD3+ T lymphocytes and CD163+ macrophages, indicating active adaptive immune involvement and macrophage-mediated lipid processing (19). Additionally, increased expression of inflammatory mediators such as cyclooxygenase and nitric oxide synthase has been associated with oxidative stress and local tissue remodeling in benign lesions (33,34). These findings underscore the role of localized inflammatory signaling in lesion formation, even in the absence of malignant transformation.

C. IgG4-to-IgG Positive Plasma Cell Ratio

A more specific immunologic marker in certain benign periocular tumors is the IgG4-to-IgG positive plasma cell ratio. Wu et al. described a case of apocrine hidrocystoma with prominent IgG4-positive plasma cell infiltration, suggesting a localized IgG4-related inflammatory response (21). Similar observations have been reported in IgG4-related disease involving the eyelid, which may clinically mimic recurrent chalazion (44). IgG4 is considered an immunomodulatory immunoglobulin subclass that may reflect chronic antigenic stimulation and attempts to regulate excessive inflammation. An elevated IgG4-to-IgG ratio may therefore indicate a distinct inflammatory pathway contributing to lesion pathogenesis. Although data remain limited, this marker may assist in differentiating benign inflammatory conditions from neoplastic processes in select cases.

Limitations of the Current Evidence

The available literature is predominantly composed of retrospective analyses, case-control studies, and isolated case reports, which inherently carry moderate to serious risks of bias. Sample sizes are frequently limited, and heterogeneity in tumor types, biomarker measurement techniques, and statistical reporting reduces comparability

across studies. Additionally, standardized cut-off values for inflammatory indices are lacking, and prospective longitudinal studies evaluating survival outcomes remain scarce. These limitations highlight the need for well-designed prospective investigations to validate the diagnostic and prognostic performance of inflammatory biomarkers in periocular tumors.

CONCLUSION

In summary, it is well established that chronic inflammation plays a central role in tumorigenesis, and previous studies have shown that systemic inflammatory markers such as neutrophil-to-lymphocyte ratio and systemic immune-inflammation index, along with local cytokines including interleukin-1β and tumor necrosis factor-α, are associated with tumor presence and progression in periocular lesions. This review adds a comprehensive and integrative synthesis of both systemic hematologic indices and tissue-based inflammatory biomarkers, highlighting their complementary diagnostic and prognostic value specifically in periocular tumors. Our findings demonstrate that malignant periocular tumors exhibit both systemic and localized inflammatory activation, whereas benign lesions predominantly show localized inflammation without consistent systemic involvement. Importantly, integrating accessible blood-based markers with tissue-level profiling may serve as a practical and cost-effective approach for differentiating benign from malignant periocular tumors and improving risk stratification, although heterogeneity and the lack of standardized cut-off values remain important limitations that warrant further prospective investigation.

Ethics Approval and Consent to Participate

Not applicable. This study is a literature-based review and does not involve human participants, patient data, or experimental interventions.

Consent for Publication

Not applicable.

Data Availability Statement

All data are available from published sources cited in this article. Further information is available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

A.M.Y.K.Y. conceptualized the study, conducted the literature search, performed data extraction, and drafted the manuscript.

H.P. supervised the study design and critically revised the manuscript.

S.P.A. contributed to methodological refinement and manuscript revision.

A.P. contributed to data interpretation and critical review of the final manuscript.

All authors read and approved the final manuscript.

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ABBREVIATIONS

BCC – Basal Cell Carcinoma

SCC – Squamous Cell Carcinoma

MM – Malignant Melanoma

NLR – Neutrophil-to-Lymphocyte Ratio

PLR – Platelet-to-Lymphocyte Ratio

MLR – Monocyte-to-Lymphocyte Ratio

SII – Systemic Immune-Inflammation Index

ROS – Reactive Oxygen Species

RNS – Reactive Nitrogen Species

RSS – Reactive Sulfur Species

IL – Interleukin

TNF-α – Tumor Necrosis Factor Alpha

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