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Mechanisms and treatment strategies for postoperative complications of pterygium surgery

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翼状胬肉手术后并发症的机制和治疗策略

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摘要

翼状胬肉是一种常见的眼表疾病,以结膜组织异常增生至角膜为特征,其主要治疗方式常需手术切除。尽管手术有效,但翼状胬肉术后常伴随一系列并发症,显著影响患者预后及生活质量。本综述系统分析了这些并发症的分类、潜在病理生理机制及相关风险因素,重点关注了较少被深入探讨的病变,如术后肉芽肿(PPG)、角膜凹陷及巩膜坏死,同时亦探讨了更常见的复发问题。按并发症出现的时间(早期、中期、晚期)分类,并深入分析其普遍性与特异性诱因,包括手术创伤、患者个体特征、手术技术及围手术期管理。此外,本综述综合了预防策略及治疗干预的最新进展,涵盖改良手术技术,如飞秒激光辅助翼状胬肉手术(FLAPS)、翼状胬肉广泛切除联合扩展结膜移植(P.E.R.F.E.C.T.)技术、组织折叠技术,辅助疗法的合理应用,如丝裂霉素 C(MMC)、5-氟尿嘧啶(5-FU)、皮质类固醇、抗血管内皮生长因子(VEGF)药物,以及优化的术后护理方案。通过整合现有证据并明确未来研究方向,文章旨在为眼科医生提供有价值的理论基础,以指导个体化手术规划、动态术后管理,最终减少并发症并提升患者满意度。

关键词:翼状胬肉;术后并发症;复发;肉芽肿;巩膜坏死;辅助治疗;飞秒激光辅助翼状胬肉手术

Abstract

• Pterygium, a common ocular surface disorder characterized by the abnormal growth of conjunctival tissue onto the cornea, often necessitates surgical excision as its primary treatment. While effective, pterygium surgery is frequently associated with a spectrum of postoperative complications that significantly impact patient prognosis and quality of life. This comprehensive review systematically analyzes the classification, underlying pathophysiological mechanisms, and associated risk factors of these complications, with a particular focus on less commonly explored entities such as postoperative granuloma (PPG), corneal dellen, and scleral necrosis, alongside the more prevalent issue of recurrence. We delineate these complications based on their temporal presentation (early, intermediate, and late), and provide an in-depth analysis of general and specific contributing factors, including surgical trauma, individual patient characteristics, surgical technique, and perioperative management. Furthermore, this review synthesizes advancements in preventive strategies and therapeutic interventions, encompassing refined surgical techniques [*e.g.*, femtosecond laser-assisted pterygium surgery (FLAPS), pterygium extended removal followed by extended conjunctival transplant (P.E.R.F.E.C.T.) technique, Tissue Tuck technique], judicious application of adjuvant therapies [*e.g.*, mitomycin C (MMC), 5-fluorouracil (5-FU), corticosteroids, anti-vascular endothelial growth factor (VEGF) agents], and optimized postoperative care protocols. By consolidating current evidence and identifying future research priorities, this review aims to provide ophthalmologists with a valuable theoretical foundation to guide individualized surgical planning, dynamic postoperative management, and ultimately minimize complications and improve patient satisfaction.

• KEYWORDS: pterygium; postoperative complication; recurrence; granuloma; scleral necrosis; adjuvant therapy; femtosecond laser-assisted pterygium surgery

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INTRODUCTION

Pterygium is a prevalent ocular surface condition defined by the triangular, wing-shaped fibrovascular proliferation of bulbar conjunctival tissue that invades the superficial cornea, typically originating from the nasal limbus[1]. Its global occurrence varies significantly, with higher prevalence in regions characterized by elevated ultraviolet (UV) radiation exposure, such as tropical climates, and in outdoor occupations[2]. Clinically, pterygium can manifest with symptoms from mild irritation and foreign body sensation to severe visual disturbances like induced astigmatism and direct visual axis obstruction[3]. Effective management is crucial to alleviate discomfort, preserve visual acuity, and prevent long-term ocular surface compromise.

Surgical excision remains the cornerstone of pterygium treatment, aiming to remove abnormal tissue and restore normal ocular surface anatomy[4]. Common surgical approaches include bare sclera excision, conjunctival autograft, and amniotic membrane transplantation (AMT)[5]. While generally effective, postoperative complications significantly impact prognosis and treatment success. Despite advancements like femtosecond laser-assisted pterygium surgery (FLAPS) and the pterygium extended removal followed by extended conjunctival transplant (P.E.R.F.E.C.T.) technique, coupled with adjuvant antimetabolites [*e.g.*, mitomycin C (MMC)] and corticosteroids, complications persist as critical limitations[6-7].

Existing literature primarily focuses on pterygium recurrence, which often necessitates repeat surgeries, increasing healthcare costs and patient burden[8]. However, comprehensive understanding of less common complications-including postoperative granuloma (PPG), corneal dellen, and subconjunctival hemorrhage (SCH)-remains limited[9-10]. Although these conditions demonstrate lower incidence rates compared to recurrence, they can significantly contribute to postoperative discomfort and reduced patient satisfaction. Particularly concerning is delayed-onset corneoscleral melting-an insidious complication often underdiagnosed yet capable of causing devastating visual impairment[11]. However, these complications are rarely discussed in detail in the literature, and evidence-based guidelines for their prevention and management remain lacking[9-10]. This review aims to address this gap by systematically evaluating both common and underrepresented complications, their mechanisms, and current and emerging treatment approaches.

Systematic Classification and Clinical Manifestations of Postoperative Complications in Pterygium Postoperative complications following pterygium surgery are systematically classified by their typical timing, which greatly aids in their diagnosis and management.

Perioperative/early complications (typically <1 week) These complications manifest during or shortly after surgery. Intraoperative complications include corneal perforation, which is an accidental penetration leading to infection or altered corneal curvature culminating in visual impairment, and rectus muscle injury, which is damage to extraocular muscles potentially causing diplopia (double vision)[12]. Excessive hemorrhage, characterized by significant bleeding, can obscure the surgical field and lead to postoperative SCH[13]. Suture-related issues stem from problems with graft fixation sutures, such as suture loosening or detachment, which can lead to graft instability, and irritation symptoms from exposed suture ends[14]. Initial signs of infection indicate microbial colonization requiring close monitoring.

Intermediate complications (typically 1 week-1 month) These complications develop after the initial healing phase, indicating ongoing inflammation or delayed healing. Inflammatory reactions are characterized by marked conjunctival hyperemia, which may be accompanied by the formation of Tenon's capsule cysts and granuloma formation[15]. Graft-related problems at this stage are more severe, encompassing partial or complete necrosis and complete displacement or loss[16]. Antimetabolite- and cornea-related issues are linked to persistent epithelial defects, filamentary keratitis, and early signs of scleral necrosis[17]. Ocular infections may involve various anatomical layers from superficial to deep structures, including conjunctivitis, keratitis, scleritis, and endophthalmitis[18].

Late complications (typically > 1 month) These complications appear weeks, months, or even years after surgery, representing chronic issues. Recurrence is the most significant long-term complication, involving the regrowth of fibrovascular tissue onto the cornea[19]. Clinical manifestations include revascularization and fibrovascular tissue crossing the limbus, accompanied by hyperemia and irritation. Most recurrences occur within 6-12 mo postoperatively, but can appear later[19]. Scarring and cosmetic concerns involve long-term changes in ocular tissue such as conjunctival scarring (fibrosis and contracture), symblepharon (adhesion between the conjunctiva, shortening the fornix), and corneal scarring, which are opacities affecting vision[17]. Other reported complications include dry eye, scleral necrosis, and glaucoma[20-21].

Mechanisms, Risk Factors, and Evidence-based Management of Pterygium Surgery Complications Understanding the mechanisms and risk factors is crucial for effective prevention and management of complications following pterygium surgery. These factors are broadly categorized into general influences and specific triggers.

General Mechanisms and Risk Factors General factors influence ocular surface healing and the eye's response to surgical trauma . Surgery inherently involves controlled tissue

Table 1 Classification and characteristics of postoperative complications

Complication	Key clinical features	Risk factors
Perioperative/Early (<1 wk)		
Rectus muscle injury	Diplopia, ocular motility restriction	Large/recurrent pterygium, prior strabismus/retinal surgery
Corneal perforation	Acute vision loss, seidel test-positive aqueous leakage, anterior chamber shallowing	Corneal thinning preoperatively
SCH	Asymptomatic red patch, No visual acuity change	Surgical trauma, anticoagulant use, hypertension, Valsalva maneuvers, eye rubbing
Intermediate (1 wk-1 mo)		
PPG	Red, fleshy nodule at surgical site	Scleral exposure, suture irritation, excessive inflammation
Corneal dellen and perforation	Dry spot, pain, epithelial defect	Pre-existing dry eye, limbal stem cell deficiency
SISN	White, thinned sclera, severe pain	Ischemia (over-cautery), MMC 0.02% application, autoimmune disease (*e.g.*, rheumatoid arthritis)
Graft failure	Conjunctival graft misalignment, redness	Poor suture fixation, excessive eye rubbing
Late (>1 mo)		
Recurrence	Fibrovascular growth crossing limbus	Incomplete excision, high UV exposure, young age

PPG: Postoperative granuloma; SCH: Subconjunctival hemorrhage; SISN: Surgically induced scleral necrosis; MMC: Mitomycin C; UV: Ultraviolet.

injury. Insufficient surgical experience may lead to residual tissue (a primary cause of recurrence) or excessive excision (excessive scleral exposure)[22]. Overzealous cauterization can induce tissue necrosis, delay wound healing, exacerbate inflammatory scarring, and even damage the deep sclera[22-23]. Excessive traction may cause tissue tearing and microvascular injury, aggravating postoperative edema and inflammation[24].

Individual Factors Patient-specific biological characteristics significantly influence the healing process. Age plays a role, with younger patients typically exhibiting more vigorous tissue metabolism and stronger inflammatory responses, predisposing them to excessive scarring and neovascularization, thereby increasing the risk of recurrence. Conversely, elderly patients may experience diminished healing capabilities, potentially leading to delayed graft integration[25-26]. Systemic conditions, such as diabetes, impair microcirculation and immune function, increasing the risk of infection and delaying healing. Immunosuppressive states further compromise the body's ability to combat infection and repair tissue[27]. Autoimmune inflammatory conditions, like rheumatoid arthritis, can exacerbate postoperative inflammation and predispose patients to scleral necrosis; medications used for these conditions can also further compromise healing[28]. Local ocular factors are also important. For example, blepharitis introduces inflammatory mediators that can stimulate recurrence, while dry eye syndrome, with its tear film instability, impedes epithelial repair and weakens the ocular surface barrier, increasing susceptibility to persistent epithelial defects and corneal dellen[29-30].

Perioperative Management Effective postoperative care and patient adherence are crucial for successful outcomes. Inadequate patient education, including a lack of clear instructions regarding eye drop application, avoiding eye rubbing, and the importance of follow-up appointments, can lead to infection, SCH, poor healing, and steroid-induced glaucoma[31]. Each complication has distinct mechanisms and risk factors that contribute to its development.

Recurrence Pterygium recurrence, defined as the re-invasion of fibrovascular tissue onto the cornea, is the most challenging long-term complication following surgery. Bare sclera excision has the highest recurrence rate (up to 89.0%), followed by AMT (42.3%), and conjunctival autograft (16.7%)[32].

The recurrence of pterygium primarily stems from three interconnected pathological processes. First, surgical trauma induces an inflammatory cascade characterized by the release of cytokines including tumor necrosis factor-α, interleukin-1β and interleukin-6, which not only promote local inflammation and limbal stem cell dysfunction but also activate recurrence-favoring signaling pathways[33]. Moreover, these inflammatory mediators stimulate angiogenic factors such as vascular endothelial growth factor (VEGF) and basic fibroblast growth factor, leading to pathological neovascularization that sustains lesion growth by providing nutrients and facilitating inflammatory cell recruitment[34]. Additionally, the surgical intervention triggers abnormal proliferation of residual fibroblasts, which excessively deposit extracellular matrix components, ultimately contributing to scar formation and tissue regrowth[35].

Risk factors for recurrence are multifaceted. Preoperative factors include extensive corneal involvement and a history of multiple previous surgeries. Patient-specific factors encompass younger age, recurrent pterygium, aggressive

pterygium (Thoft grade III), and chronic UV radiation exposure[36]. Dry eye can also contribute[37]. Intraoperative risk factors primarily involve incomplete excision or excessive scleral exposure; and postoperative management issues particularly inadequate inflammation control[37]. These processes—surgical trauma, activation of inflammatory cytokines, and proliferation of abnormal fibroblasts—are not isolated, but form a continuous pathological loop that predisposes to recurrent fibrovascular ingrowth.

Diagnosis of recurrence relies on postoperative follow-up. Small, quiescent recurrences may be observed, but significant recurrence typically requires surgical intervention[38]. Treatment for recurrent pterygium is similar to that for primary pterygium, involving autologous conjunctival graft and adjuvant drugs. The PERMISLET technique has shown promising results with zero recurrence and minimal complications, making it suitable for patients with limited healthy conjunctiva or those who may require future glaucoma surgery[39].

Postoperative Granuloma PPG presents as an elevated, red, fleshy mass with neovascularization, often located at the nasal incision site[40]. Histologically, it reveals inflammatory cells, fibrous structures, and sometimes epithelial dysplasia[41]. Patients typically experience red eyes, a foreign body sensation, and increased secretions[9].The pathogenesis of PPG primarily involves scleral exposure and foreign body reactions induced by materials such as sutures, fibrous tissue, or talc particles[21,42]. The chronic inflammation triggered by exposed sclera/Tenon's capsule tissue, combined with mechanical friction between Tenon's capsule and the upper eyelid stimulating cellular proliferation and angiogenesis, constitutes the predominant underlying mechanism[21]. There remains some controversy regarding the inflammatory potential of absorbable versus non-absorbable sutures, with current evidence suggesting that absorbable sutures may paradoxically induce stronger inflammatory responses in ophthalmic procedures[43].The primary risk factors for PPG involve graft fixation methods (notably fibrin glue application that may promote tissue microdisplacement and exposure)[44], incomplete conjunctival flap closure with consequent sclera/Tenon's capsule exposure, mechanical friction between residual Tenon's capsule tissue and the upper eyelid, as well as nonstandardized corticosteroid use[21]. PPG is managed with observation, local corticosteroid injections, or surgical excision. Femtosecond laser excision has also been explored as a treatment option[45]. Surgical resection followed by postoperative corticosteroids is considered an effective treatment approach[46].

Corneal Dellen and Perforation Corneal dellen is a localized concave depression indicating thinning of the cornea[17]. Patients typically experience pain, foreign body sensation, and tearing[47]. If left untreated, the progression can lead to perforation, vision loss, and even endophthalmitis[48].The mechanisms underlying corneal dellen involve tear film instability caused by both mechanical factors (including graft edema, ocular surface irregularities[47], and incomplete conjunctival coverage) and neurotrophic effects (such as trigeminal nerve damage or limbal stem cell deficiency resulting from excessive cauterization)[49]. The condition is further aggravated by antimetabolite toxicity (MMC/5-FU), which not only delays epithelial healing but also directly induces dellen formation[50]. Major risk factors encompass pre-existing ocular surface diseases (like dry eye syndrome and corneal scarring[51]), surgical-related factors (such as limbal stem cell deficiency from multiple procedures, deep corneal excisions, and graft-related complications[51]), and pharmacological risks (particularly improper use of corticosteroids/NSAIDs or prolonged/high-dose antimetabolite application[50-51]). Regular follow-up is critical for asymptomatic dellen, as NSAIDs can mask discomfort[52]. Early dellen is managed with aggressive lubrication, autologous serum, bandage contact lenses, amniotic membrane (AM) coverage, and treating underlying dry eye[53]. Perforation requires prompt surgical intervention, with the technique depending on the defect size and adjacent corneal tissue[54]. For perforations less than 3 mm, tissue adhesives (cyanoacrylate or fibrin glue) are often used, with cyanoacrylate being more prevalent[45]. Moderate perforations (4-6) mm may be repaired using a Tenon patch graft (TPG) or multilayer AM for repair[55]. For perforations greater than 6 mm, full-thickness corneal transplantation is typically required. Often, these strategies are combined for optimal outcome[45].

Scleral Necrosis Surgically induced scleral necrosis (SISN) is a rare but severe postoperative complication in ophthalmology, characterized by scleral thinning and a distinctive "porcelain-white" necrotic appearance[10]. Its onset can be immediate or even decades later, and initial signs are often subtle, complicating early diagnosis[56]. Progression can lead to severe pain, vision loss, and even globe perforation[20]. The pathogenesis of SISN involves a complex interplay of multiple factors, including infection, ischemia, surgical trauma, and immune-mediated mechanisms. Infection is a primary trigger, often originating from a subconjunctival abscess[18]. Bacterial infections, such as Pseudomonas aeruginosa and Staphylococcus aureus, are more common than fungal infections, and the presence of hypopyon can predict infectious SISN[57]. Ischemia is another significant mechanism[58]; antimetabolites like MMC and 5-FU can induce occlusive endarteritis or inhibit endothelial cell proliferation, leading to ischemic necrosis[59]. Surgical trauma, specifically excessive electrocautery, causes thermal damage, delaying healing and directly contributing to necrosis[60]. Pre-existing autoimmune diseases, such as rheumatoid arthritis, are known to worsen SISN[61]. Key risk factors include high-dose or prolonged use of

antimetabolites[59], inadequate scleral coverage[20], systemic autoimmune diseases[61], excessive electrocautery, and postoperative infections[18]. Early detection and intervention are critical to preventing irreversible damage.

The core principles of managing scleral necrosis or necrosis are to protect exposed tissue, promote healing, and prevent perforation. Conservative management for mild cases involves discontinuing suspected drugs, intensifying lubrication, and observing the condition[62]. Pharmacotherapy for moderate to severe cases includes ceasing antimetabolites or steroids[56]. Systemic or local antibiotics are used for infection[63]; corticosteroids or immunosuppressants are employed for inflammatory or autoimmune necrosis. NSAIDs, autologous serum, and erythropoietin can serve as adjuvant agents[64-65]. If pharmacotherapy is insufficient, surgical intervention is essential and timely. This involves debridement of necrotic tissues, AMT, and patch graft coverage using conjunctival, fascial, allograft sclera, or dura mater. Severe infections like endophthalmitis require emergency management[66-67].

Graft Retraction or Displacement Graft retraction refers to the partial withdrawal of the transplanted tissue, while displacement is the complete dislocation, potentially leading to graft loss. This complication is more prevalent in non-sutured techniques, such as those using fibrin glue or autologous serum, with the highest incidence reported with autologous serum[69]. It can manifest as early as the first postoperative day[43].

The mechanisms behind graft issues are varied. In sutured cases, Tenon's capsule contraction causes poor adhesion while improper suture tension (either loose or overtightened) leads to displacement or suture failure[70-71]. For sutureless methods, insufficient adhesive strength of fibrin glue/autologous serum predominates[69]. External mechanical forces such as eye rubbing or trauma may further contribute to displacement[70].

Key risk factors include sutureless fixation, oversized grafts[69], Tenon's capsule remnants[71], excessive graft tension[70], and postoperative trauma from eye rubbing or mechanical forces[72]. Management of graft issues depends on the specific problem. Early cases of displacement or retraction can sometimes be adjusted or repositioned. For necrosis, debridement is followed by re-coverage with a healthy graft[42].

Subconjunctival Hemorrhage SCH is the most prevalent complication following pterygium surgery, occurring in approximately 38.7% of cases[73]. It presents as a painless red patch and is typically self-limiting, resolving within 3 to 4 wk, but can cause minor sensation or cosmetic concern[74].

SCH develops through several pathways: surgical trauma during tissue dissection may cause microvascular rupture, especially when involving the medial rectus sheath[75-76]; sudden venous congestion from coughing, vomiting, or straining can elevate intraocular pressure leading to vascular rupture[75-76]; and postoperative eye rubbing may directly traumatize fragile vessels[76].

Risk factors for SCH include coagulation disorders such as thrombocytopenia or anticoagulant therapy[77], systemic hypertension (particularly when uncontrolled)[77-78], inherent vascular fragility, and postoperative behaviors including straining, vigorous coughing, or eye rubbing[77-78]. These factors collectively increase susceptibility to SCH development.

Most cases of asymptomatic SCH require only observation. In patients with extensive hemorrhage, topical vasoconstrictors (*e. g.*, brimonidine tartrate 0.15%) may promote blood resorption and improve comfort[79]. Artificial tears can help relieve associated ocular irritation[80]. Patients should be advised to avoid eye rubbing, strenuous activities, and Valsalva maneuvers to minimize the risk of recurrent bleeding.

Innovations in Prevention and Treatment of Postoperative Pterygium Complication Effective management of postoperative complications in pterygium surgery integrates meticulous surgical planning with advanced techniques and comprehensive perioperative care. Prevention is the cornerstone of successful pterygium surgery, aiming to minimize the incidence of complications.

Preoperative Assessment and Perioperative Management Successful pterygium management begins with thorough preoperative evaluation encompassing 3 key domains: pterygium morphology (size, vascularity and corneal involvement), ocular surface status (particularly dry eye assessment through tear film evaluation), and systemic health screening[81]. Any preexisting ocular conditions such as blepharitis or dry eye should be active management prior to surgery[82], while systemic comorbidities including diabetes mellitus and rheumatoid arthritis need appropriate medical optimization[83-84]. Patient education should focus on three critical aspects: proper adherence to prescribed eye drop regimens, strict avoidance of eye rubbing, and compliance with all scheduled follow-up appointments[76].

Comprehensive perioperative care is pivotal for surgical success. Topical antibiotic prophylaxis (*e. g.*, fourth-generation fluoroquinolones) forms the first-line defense against postoperative infections[18]. Ocular surface maintenance through regular application of artificial tears helps maintain a healthy microenvironment, crucial for preventing corneal dellen[55]. Bandage contact lenses serve dual purposes of mechanical protection and promotion of epithelial healing, particularly in cases with large conjunctival defects[47]. Critical to long-term success is patient compliance with protective measures including consistent UV-blocking eyewear use and complete avoidance of eye rubbing for at least 4 wk[85]. Postoperative inflammation control follows a standardized steroid tapering regimen over 6 to 8 wk, while activity modifications include temporary avoidance of strenuous activities.

Surgical Strategy Selection and Technique Optimization Adherence to fundamental surgical principles combined with technological innovation is pivotal in preventing complications. The core tenets encompass complete excision of Tenon's capsule, minimization of scleral exposure, tension-free graft fit, aggressive postoperative anti-inflammatory management, and preferential utilization of autologous conjunctival stem cell transplantation[21,86].

Technological innovations have significantly advanced surgical approaches. FLAPS utilizes a laser to prepare precise, ultra-thin conjunctival autografts, which can reduce complications and improve cosmetic outcomes, though equipment cost is a consideration[6]. The choice between fibrin glue *vs.* sutures for graft fixation is important; fibrin glue can shorten surgery time, reduce inflammation, and enhance patient comfort. It may lower recurrence by minimizing inflammation and providing hemostasis[87]. However, some studies indicate an increased granuloma risk due to potential graft displacement[44]. The P. E. R. F. E. C. T. technique involves extensive pterygium removal with a large conjunctival autograft, achieving very low recurrence rates (0.1%)[7];

The Tissue Tuck technique uses cautery to seal Tenon's capsule, followed by a large frozen AM as a physical barrier. When combined with MMC, it significantly lowers recurrence rates (2.3%)[88]. Current evidence does not strongly support the use of MiniSLET for primary pterygium due to higher recurrence rates[89].

Adjuvant Therapies Adjuvant therapies are crucial for controlling inflammation and inhibiting fibroblast proliferation to reduce recurrence. Preoperatively, cyclosporine A eye drops are administered as an immunosuppressive agent to reduce surgical inflammation and block VEGF-induced angiogenesis[90]. Intraoperatively, MMC (0.02% for 5 min or 0.04% for 3 min) is preferentially applied to inhibit DNA replication and fibroblast proliferation, though its corneal/scleral toxicity requires cautious use[91]. Intraoperative topical 5-FU (sponge/soaking) reduces primary pterygium recurrence with good safety[92]. Postoperative regimens include: corticosteroids (drops or subconjunctival injection) to control inflammation and prevent recurrence, requiring tapered dosing and side effect monitoring[93]; 5-FU injections that inhibit inflammatory cell proliferation (81.3%-96.0% success rate) but risk corneal toxicity and scleral lysis[94-95]; anti-VEGF agents to suppress angiogenesis-driven recurrence[96]; interferon-alpha 2b as an immunomodulatory adjuvant[97]; with continued cyclosporine A drops maintaining preoperative immunosuppressive effects[90].

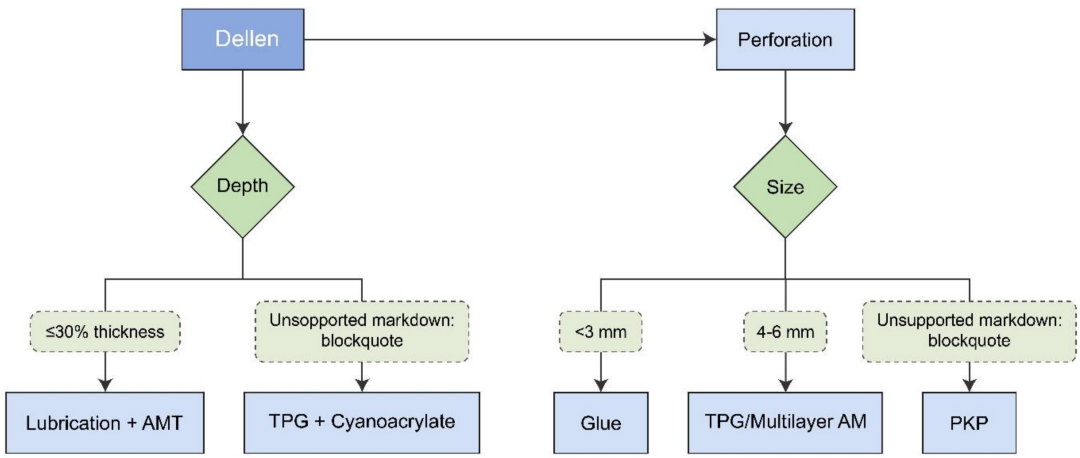

Figure 1 Therapeutic algorithm for corneal dellen and perforation management. AMT: Amniotic membrane transplantation; TPG: Tenon patch graft; AM: Amniotic membrane; PKP: Penetrating keratoplasty.

Table 2 Perioperative adjuvant therapies for inflammation and fibrosis control

Drug	Key Action	Administration
Pre-operative		
Cyclosporine A	T-cell/VEGF inhibition	0.05% drops
Intra-operative		
MMC	DNA/fibroblast inhibition	0.02% (5 min)/0.04% (3 min) surgical-site soaking
5-FU	Antimetabolite	Surgical-site soaking
Post-operative		
Corticosteroids	Anti-inflammatory	Drops/injection
5-FU	Antimetabolite	Injection
Anti-VEGF	Angiogenesis blockade	Injection
Interferon-α2b	Immunomodulation	Drops
Cyclosporine A	T-cell/VEGF inhibition	0.05% drops

MMC: Mitomycin C; 5-FU: 5-Floxuridine; VEGF: Vascular endothelial growth factor.

Future Research Priorities and Clinical Advancements
Future research in pterygium management focuses on improving patient outcomes and minimizing complications.

Advanced Diagnostic and Predictive Tools Future advancements aim for more precise and personalized care. Optical coherence tomography angiography with molecular phenotyping holds promise for developing precise recurrence prediction models by combining detailed vascular imaging with molecular profiling of the tissue[11]. Artificial intelligence algorithms are being explored for their potential in optimizing surgical planning and providing early complication warnings *via* automated image analysis[98]. Furthermore, incorporating patient-reported outcomes, such as pain scores and cosmetic satisfaction, into efficacy evaluation will offer a more holistic view of the treatment's impact on a patient's quality of life[99].

Enhanced Understanding of Genetic and Environmental Factors Further research into genetic predispositions and specific environmental triggers beyond UV exposure could lead to the development of personalized prevention strategies and more targeted therapies for pterygium and its complications[100].

CONCLUSION

Preventing and controlling postoperative complications of pterygium surgery is a multifaceted endeavor crucial for optimal patient outcomes. This review detailed the diverse spectrum of complications, from SCH to severe corneoscleral perforations and scleral necrosis. We elucidated the complex interplay of general factors (surgical trauma, patient characteristics, technique, and perioperative management) and specific pathophysiological mechanisms.

Significant advancements in surgical techniques (P.E.R.F.E.C.T. and FLAPS) and adjuvant pharmacotherapies (MMC, 5-FU and corticosteroids) have improved success and minimized postoperative complications. Nevertheless, the core principles of pterygium surgery remain: complete excision of Tenon's capsule, minimization of scleral exposure, tension-free graft fit, aggressive postoperative anti-inflammatory management, and preferential utilization of autologous conjunctival stem cell transplantation.

Ophthalmologists must continually refine microsurgical techniques, standardize the application protocols for antimetabolites and fibrin glue, and strengthen perioperative inflammatory management to minimize complication rates and prevent recurrence.

Conflicts of Interests: Pan HL, None; Wu SQ, None.
Authors' contributions: Pan HL investigated the literatures, collected the data, visualized results and wrote the manuscript; Wu SQ designed the research, provided guidance, reviewed and edited the manuscript. All authors approved the submitted version.

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