

**ФУНДАМЕНТАЛ ВА
КЛИНИК ТИББИЁТ
АХБОРОТНОМАСИ**

**BULLETIN OF FUNDAMENTAL
AND CLINIC MEDICINE**

2026, №2 (22)

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ
РЕСПУБЛИКИ УЗБЕКИСТАН

**BULLETIN OF FUNDAMENTAL
AND CLINIC MEDICINE**
**ФУНДАМЕНТАЛ ВА КЛИНИК
ТИББИЁТ АХБОРОТНОМАСИ**
**ВЕСТНИК ФУНДАМЕНТАЛЬНОЙ И
КЛИНИЧЕСКОЙ МЕДИЦИНЫ**

Научный журнал по фундаментальным и клиническим
проблемам медицины
основан в 2022 году

Бухарским государственным медицинским институтом
имени Абу Али ибн Сино
выходит один раз в 2 месяца

Главный редактор – Ш.Ж. ТЕШАЕВ

Редакционная коллегия:

*С.С. Давлатов (зам. главного редактора),
Р.Р. Баймурадов (ответственный секретарь),
М.М. Амонов, Г.Ж. Жарилкасинова,
А.Ш. Иноятов, Д.А. Хасанова, Е.А. Харибова,
Ш.Т. Уроков, Б.З. Хамдамов, Ф.К. Халлоқов*

*Учредитель Бухарский государственный
медицинский институт имени Абу Али ибн Сино*

2026, № 2 (22)

Адрес редакции:

Республика Узбекистан, 200100, г.
Бухара, ул. Гиждуванская, 23.

Телефон (99865) 223-00-50

Факс (99866) 223-00-50

Сайт <https://bsmi.uz/journals/fundamental-ya- klinik-tibbiyot-ahborotnomasi/>

e-mail baymuradovravshan@gmail.com

О журнале

*Журнал зарегистрирован
в Управлении печати и информации
Бухарской области
№ 1640 от 28 мая 2022 года.*

*Журнал внесен в список
утвержденный приказом № 370/б
от 8 мая 2025 года реестром ВАК
в раздел медицинских наук.*

Отпечатано в типографии ООО
“Шарк-Бухоро”. г. Бухара,
ул. Ўзбекистон Мустақиллиги, 70/2.

Редакционный совет:

Абдурахманов Д.Ш.	(Самарканд)
Абдурахманов М.М.	(Бухара)
Ахмедов Р.М.	(Бухара)
Баландина И.А.	(Россия)
Бахронов Ж.Ж.	(Бухара)
Бернс С.А.	(Россия)
Газиев К.У.	(Бухара)
Деев Р.В.	(Россия)
Дустова Н.К.	(Бухара)
Зокирова Н.Б.	(Ташкент)
Казакова Н.Н.	(Бухара)
Калашникова С.А.	(Россия)
Каримова Н.Н.	(Бухара)
Курбонов С.С.	(Таджикистан)
Маматов С.М.	(Кыргызстан)
Мамедов У.С.	(Бухара)
Мирзоева М.Р.	(Бухара)
Миршарапов У.М.	(Ташкент)
Набиева У.П.	(Ташкент)
Нуралиев Н.А.	(Хорезм)
Наврұзов Р.Р.	(Бухара)
Нарзиева Д.Ф.	(Бухара)
Орипов Ф.С.	(Самарканд)
Орипова Ф.Ш.	(Бухара)
Одилова Г.Р.	(Бухара)
Очилов К.Р.	(Бухара)
Раупов Ф.С.	(Бухара)
Рахмонов К.Э.	(Самарканд)
Рахметов Н.Р.	(Казахстан)
Рахматова С.Н.	(Бухара)
Султонова Л.Дж.	(Бухара)
Сайдуллаев З.Я.	(Самарканд)
Удочкина Л.А.	(Россия)
Файзиев Х.Б.	(Бухара)
Хамдамова М.Т.	(Бухара)
Хамдамов И.Б.	(Бухара)
Ходжаева Д.Т.	(Бухара)
Худойбердиев Д.К.	(Бухара)
Шодиева М.С.	(Бухара)
Эшонов О.Ш.	(Бухара)

PROPHYLAXIS OF DRY EYE SYNDROME AND REHABILITATION INTERVENTIONS FOLLOWING DACRYOCYSTORHINOSTOMY**Hamdamov H.O.**

Fergana Medical Institute of Public Health, Fergana, Uzbekistan

Resume. This study investigated the causes, prevention, and management of dry eye syndrome (DES) following dacryocystorhinostomy (DCR) in 180 patients. Tear film stability, ocular surface condition, and patient-reported symptoms were assessed using OSDI, TBUT, and Schirmer tests. DES occurred in 30% of patients within three months post-surgery; however, early application of lubricating agents and physiotherapy reduced this rate to 8–10%. The findings confirm that combined use of hyaluronate-based artificial tears, lipid formulations, thermal therapy, and meibomian gland care is effective in minimizing DES after DCR.

Keywords: dacryocystorhinostomy, dry eye syndrome, prophylaxis, rehabilitation, meibomian glands, artificial tears, physiotherapy, quality of life.

ДАКРИОЦИСТОРИНОСТОМИЯДАН КЕЙИН ҚУРУҚ КЎЗ СИНДРОМИНИНГ ПРОФИЛАКТИКАСИ ВА РЕАБИЛИТАЦИЯ ЧОРАЛАРИ**Ҳамдамов Ҳ.О.**

Фарғона жамоат саломатлиги тиббиёт институти, Фарғона ш., Ўзбекистон

Резюме. Ушбу тадқиқотда дакриоцисториностомия (ДЦР) операциясидан кейин қуруқ кўз синдроми (ҚКС) ривожланиш сабаблари, унинг олдини олиш ва даволаш усуллари 180 нафар бемор мисолида ўрганилди. Кўз ёши плёнкасининг барқарорлиги, кўз юзасининг ҳолати ва беморларнинг субъектив шикоятлари OSDI индекси, кўз ёши плёнкасининг узилиши вақти (TBUT) ҳамда Ширмер тести ёрдамида баҳоланди. Операциядан кейинги уч ой давомида беморларнинг 30 фоизда қуруқ кўз синдроми аниқланган бўлса, намловчи воситалар ва физиотерапевтик муолажаларни эрта қўллаш натижасида ушбу кўрсаткич 8–10 фоизгача камайган. Олинган натижалар гиалуронат асосидаги сунъий кўз ёшлари, липидли препаратлар, иссиқлик терапияси ва мейбомий безларни парвариши қилишни ўз ичига олган комплекс ёндашув дакриоцисториностомиядан кейин қуруқ кўз синдроми ривожланиш хавфини самарали камайтиришини тасдиқлайди.

Калит сўзлар: дакриоцисториностомия, қуруқ кўз синдроми, профилактика, реабилитация, мейбомий безлар, сунъий кўз ёшлари, физиотерапия, ҳаёт сифати.

ПРОФИЛАКТИКА СИНДРОМА СУХОГО ГЛАЗА И РЕАБИЛИТАЦИОННЫЕ МЕРОПРИЯТИЯ ПОСЛЕ ДАКРИОЦИСТОРИНОСТОМИИ**Ҳамдамов Ҳ.О.**

Ферганский медицинский институт общественного здравоохранения, г. Фергана, Узбекистан

ORCID ID: 0009-0006-9148-497X

Резюме. В данном исследовании изучены причины развития, профилактика и методы ведения синдрома сухого глаза (ССГ) после дакриоцисториностомии (ДЦР) у 180 пациентов. Стабильность слёзной плёнки, состояние глазной поверхности и субъективные жалобы пациентов оценивались с использованием индекса OSDI, теста разрыва слёзной плёнки (TBUT) и пробы Ширмера. В течение трёх месяцев после операции ССГ был выявлен у 30% пациентов, однако раннее применение увлажняющих препаратов и физиотерапевтических процедур позволило снизить этот показатель до 8–10%. Полученные результаты подтверждают, что комплексное применение искусственных слёз на основе гиалуроната, липидных препаратов, тепловой терапии и ухода за мейбомиевыми железами эффективно снижает риск развития синдрома сухого глаза после ДЦР.

Ключевые слова: дакриоцисториностомия, синдром сухого глаза, профилактика, реабилитация, мейбомиевые железы, искусственные слёзы, физиотерапия, качество жизни.

e-mail: hasanboyxamdammov7@gmail.com

Introduction. Dry eye syndrome (DES) constitutes one of the most prevalent secondary complications arising in the post-dacryocystorhinostomy period, representing a multifactorial pathophysiological condition that significantly impacts patient visual comfort and overall quality of life despite its typically transi-

ent nature. This condition characteristically emerges during the physiological adaptation period of the lacrimal drainage system, wherein the ocular surface undergoes complex biomechanical and biochemical adjustments to accommodate the surgically modified tear film dynamics and drainage patterns established through the DCR procedure. The pathomechanistic development of DES in this clinical context involves intricate dysregulation between tear production and evaporation rates, progressive or acute diminishment of meibomian gland secretory functionality, and fundamental disruption of the delicate ocular surface homeostatic mechanisms that maintain corneal and conjunctival epithelial integrity under physiological conditions. The temporal profile of post-DCR DES typically manifests within the first three months following surgical intervention, during which period the lacrimal system attempts to re-establish equilibrium between tear secretion, distribution, and drainage through the newly created anastomotic pathway between the lacrimal sac and nasal cavity.

The clinical significance of implementing timely prophylactic measures during the post-DCR period cannot be overstated, as evidence-based interventions have demonstrated capacity to accelerate ocular surface regeneration processes, attenuate pro-inflammatory cytokine cascades that contribute to epithelial damage and goblet cell depletion, and substantially enhance multiple dimensions of patient quality of life metrics including visual function, ocular comfort, and psychosocial well-being. Contemporary understanding of post-operative DES pathophysiology suggests that a proactive, multi-modal approach to prevention and early intervention offers superior outcomes compared to reactive treatment strategies implemented only after symptomatic manifestation, thereby establishing the rationale for comprehensive prophylactic protocols initiated in the pre-operative period and systematically continued throughout the rehabilitation phase. This paradigm shift toward anticipatory rather than responsive management reflects broader trends in ophthalmic surgery toward optimization of surgical outcomes through meticulous attention to perioperative ocular surface health, recognition of the tear film as a dynamic and vulnerable system requiring active support during post-operative healing, and acknowledgment that patient-reported outcomes and subjective comfort constitute equally important success metrics alongside traditional objective clinical parameters.

1. Pathophysiological Foundations

The mechanistic emergence of dry eye syndrome following dacryocystorhinostomy represents a complex interplay of biomechanical, neurophysiological, immunological, and morphological factors that collectively disrupt the intricate homeostatic systems governing ocular surface health and tear film stability. This pathophysiological cascade is intrinsically linked to perturbations in the precisely regulated balance between tear production, distribution, and elimination, as well as fundamental alterations in the stratified architecture of the precorneal tear film that normally functions to maintain optical clarity, provide nutritional support to the avascular corneal epithelium, facilitate removal of cellular debris and foreign particulates, and establish the first line of defense against microbial colonization through its complement of antimicrobial proteins and immunoglobulins. The surgical modification of lacrimal drainage pathways inherent to the DCR procedure, while therapeutically necessary for resolution of nasolacrimal duct obstruction, inevitably introduces temporary disruption to the finely tuned hydrodynamics of tear circulation, creating conditions conducive to tear film instability during the adaptation period as the ocular surface system recalibrates to accommodate altered drainage patterns and re-establishes sustainable equilibrium parameters appropriate to the new anatomical configuration.

Understanding the physiological architecture and function of the lacrimal system provides essential context for comprehending post-DCR complications, as the healthy precorneal tear film comprises three biochemically and functionally distinct layers that operate in coordinated fashion to maintain ocular surface integrity. The outermost lipid layer, synthesized and secreted by the meibomian glands embedded within the tarsal plates of both upper and lower eyelids, functions primarily to minimize evaporative water loss from the underlying aqueous layer by creating a hydrophobic barrier at the air-tear interface, with this lipid component consisting of a complex mixture of nonpolar lipids including wax esters, cholesterol esters, and various hydrocarbons that collectively reduce surface tension and retard evaporation rates under normal physiological conditions. The intermediate aqueous layer, representing the volumetrically dominant component of the tear film and secreted primarily by the main and accessory lacrimal glands, provides the bulk of tear film thickness and serves multiple critical functions including maintenance of optical quality through creation of a smooth refractive surface, delivery of oxygen and nutrients to the avascular corneal epithelium, removal of metabolic waste products and cellular debris through its flow characteristics, and provision of antimicrobial defense through its complement of lysozyme, lactoferrin, beta-lysin, and secretory immunoglobulin A. The innermost mucin layer, produced by conjunctival goblet cells and consisting of high-molecular-weight glycoproteins, functions to convert the hydrophobic corneal epithelial cell surface into a hydrophilic substrate capable of retaining the overlying aqueous layer, thereby ensuring uniform distribution and adherence of

tears across the entire ocular surface. DCR surgical intervention, while successfully restoring patency to obstructed lacrimal drainage pathways, temporarily modifies the hydrodynamic pressure gradients and flow characteristics within the tear film circulation system, resulting in transient destabilization of the tear film that persists until adaptive mechanisms re-establish a new steady-state equilibrium appropriate to the surgically modified anatomy, during which transitional period accelerated tear film breakdown and increased evaporation rates contribute to the symptomatic manifestation of dry eye syndrome.

The neurosensory and inflammatory dimensions of post-DCR dry eye syndrome involve complex interactions between corneal sensory innervation, reflex tear secretion pathways, and local inflammatory mediator cascades that collectively compromise tear production and quality during the post-operative period. Surgical manipulation during DCR procedures, as well as inflammatory processes in the immediate post-operative phase, can result in transient impairment of corneal and conjunctival sensory nerve function mediated through trigeminal nerve pathways, with this sensory hypoesthesia directly impacting reflex tear secretion mechanisms that normally respond to ocular surface desiccation or irritation by stimulating increased lacrimal gland output. Simultaneously, subclinical inflammatory processes developing in the perioperative period around the nasolacrimal canal and adjacent tissues trigger upregulation of pro-inflammatory cytokines including interleukin-1 beta, tumor necrosis factor-alpha, and interleukin-6, which exert multiple deleterious effects on ocular surface homeostasis including deceleration of epithelial cell regeneration and wound healing processes, reduction in conjunctival goblet cell density through inflammatory-mediated apoptosis or functional suppression, and alterations in the biochemical composition and viscoelastic properties of the tear film that compromise its stability and protective functions. These inflammatory and neurosensory alterations typically undergo gradual resolution over a period of two to three months as surgical trauma resolves and tissue healing progresses, explaining the characteristic temporal profile of post-DCR dry eye syndrome which most commonly manifests during the early post-operative phase of one to three months and typically presents as a mild chronic evaporative-type condition rather than acute aqueous-deficient disease, with this distinction having important therapeutic implications for selection of appropriate management strategies.

Meibomian gland dysfunction represents a particularly significant contributor to post-DCR dry eye syndrome, as these specialized sebaceous glands play a crucial role in maintaining tear film stability through their secretion of lipid components that form the superficial layer of the precorneal tear film. Following DCR procedures, meibomian secretory activity characteristically experiences temporary suppression, with advanced diagnostic techniques including infrared meibography and meibomian gland expression assessments revealing that lipid secretion decreases by 27-32% in the first month post-operatively, begins to show recovery by three months, and achieves morphological restoration approaching baseline parameters in approximately 90% of patients by six months following surgery. This transient meibomian dysfunction directly compromises the integrity and thickness of the tear film lipid layer, resulting in increased evaporation rates that may exceed normal values by 20-30%, thereby precipitating the development of evaporative-type dry eye disease that manifests clinically as symptoms of ocular irritation, foreign body sensation, and visual fluctuation that correlate with moderate scores on standardized symptom assessment instruments such as the Ocular Surface Disease Index. The pathophysiological mechanisms underlying post-DCR meibomian dysfunction likely involve multiple factors including perioperative inflammation affecting glandular tissues, altered lid dynamics during the healing phase, changes in neural regulation of meibomian secretion, and potential effects of topical medications administered during the post-operative period, with recognition of this multifactorial etiology informing therapeutic strategies that address both inflammatory components and mechanical/functional aspects of meibomian gland performance.

The microbiological environment of the ocular surface undergoes significant alterations during the post-DCR period, with quantitative analysis demonstrating that concentrations of key antimicrobial tear film components including lysozyme, lactoferrin, and secretory immunoglobulin A decrease by 15-20% during the initial month following surgery, creating conditions of relative immunological vulnerability that increase susceptibility to shifts in the commensal ocular surface microflora and elevated risk for opportunistic colonization or infection. This temporary immunocompromise reflects the combined effects of surgical stress, inflammatory processes that may consume or downregulate protective protein production, potential dilutional effects from altered tear dynamics, and direct effects of perioperative topical medications on the resident microbial ecosystem. The resultant alterations in ocular surface microflora composition may contribute to development of mild blepharitis or meibomian gland inflammation, further exacerbating meibomian dysfunction and perpetuating the dry eye condition through establishment of a self-reinforcing pathological cycle. Recognition of this microbiological dimension of post-DCR ocular surface disease provides rationale for prophylactic administration of antiseptic ophthalmic formulations and immunomodulatory agents including dexpanthenol, taurine, and lactobionate compounds during the perioperative period, with the goal of main-

taining a healthy ocular surface microbiome and preventing opportunistic infections that could complicate recovery or contribute to chronic ocular surface inflammation.

2. Prophylactic Phases

The prophylactic management of dry eye syndrome following dacryocystorhinostomy necessitates a systematically structured, phase-specific approach that targets accelerated restoration of ocular surface homeostatic mechanisms, attenuation of inflammatory cascade activation, and normalization of meibomian gland secretory functionality through coordinated application of pharmacological, mechanical, and behavioral interventions tailored to the evolving physiological requirements of each distinct phase of post-operative recovery. This temporal organization of prophylactic strategies reflects recognition that the ocular surface system undergoes dynamic changes throughout the perioperative period, with different pathophysiological processes predominating at different time points and therefore requiring phase-appropriate interventions that address the specific challenges characteristic of each recovery stage. The comprehensive prophylactic framework encompasses three principal phases: the preoperative preparatory phase focused on optimizing baseline ocular surface health prior to surgical intervention, the early postoperative phase emphasizing inflammatory control and epithelial protection during the acute healing period, and the extended rehabilitation phase targeting sustainable restoration of normal ocular surface function and prevention of chronic dry eye sequelae.

The preoperative preparatory phase, typically initiated five to seven days prior to scheduled DCR surgery, serves the critical function of optimizing baseline ocular surface conditions to minimize post-operative complications and establish a foundation for successful healing. During this phase, patients undergo systematic ocular hygiene regimens incorporating specialized lid cleansers such as blepharogel and antiseptic solutions including miramistin to reduce microbial burden on the ocular surface and periocular tissues, thereby decreasing infection risk and minimizing inflammatory triggers that could compromise post-operative recovery. Concurrent administration of hyaluronate-based artificial tear formulations at concentrations of 0.15-0.2%, instilled multiple times daily, serves to maintain optimal epithelial hydration status and reinforce the protective tear film barrier, with the high molecular weight and viscoelastic properties of hyaluronic acid providing superior residence time on the ocular surface compared to conventional lubricants. Nutritional optimization through oral supplementation with omega-3 fatty acids at dosages of 500-1000 mg daily represents an evidence-based intervention that enhances meibomian gland lipid secretion quality and reduces ocular surface inflammation through modulation of eicosanoid metabolism, with clinical trials demonstrating that preoperative omega-3 supplementation can reduce post-operative dry eye severity and duration. Prophylactic antibacterial coverage with topical fluoroquinolone antibiotics such as levofloxacin 0.5% administered twice daily during the preoperative period reduces conjunctival bacterial load and minimizes risk of surgical site infection, while anti-inflammatory preparation using combination formulations containing dexpanthenol and taurine provides epithelial trophic support and antioxidant protection that may accelerate post-operative healing. Clinical outcome data indicates that comprehensive implementation of this preoperative optimization protocol results in 20-25% reduction in post-operative dry eye syndrome incidence compared to patients not receiving systematic preoperative preparation, validating the clinical value of proactive ocular surface management prior to surgical intervention.

The early postoperative period, spanning from the day of surgery through approximately fourteen days post-operatively, represents a critical window during which primary objectives focus on minimizing inflammatory tissue responses, preventing epithelial damage propagation, and controlling microbial proliferation while supporting the natural healing processes inherent to tissue repair. During this phase, frequent instillation of preservative-free hyaluronate artificial tears at 0.3% concentration every two to three hours provides continuous ocular surface lubrication and epithelial protection, with the higher hyaluronate concentration offering enhanced viscosity and prolonged contact time compared to preoperative formulations, while the preservative-free formulation eliminates potential toxic effects of benzalkonium chloride or other preservatives on healing epithelial tissues. Combined antibacterial and mild corticosteroid ophthalmic preparations, typically formulated as tobramycin-dexamethasone combinations, are administered for seven to ten days to simultaneously address infection prophylaxis and inflammatory modulation, with the dual-action approach providing more comprehensive management than either agent alone while the time-limited duration of corticosteroid use minimizes risks of elevated intraocular pressure or delayed wound healing associated with prolonged steroid exposure. Thermal interventions play important roles during this phase, with cold compresses applied intermittently during the first several days providing vasoconstriction-mediated reduction in periorbital edema and analgesic effects through reduced tissue metabolism and nerve conduction velocity, while sterile warm compresses maintained at approximately 37 degrees Celsius and applied after the initial inflammatory phase subsides serve to enhance local microcirculation and facilitate removal of inflammatory

mediators through improved tissue perfusion. Topical anti-inflammatory gels containing panthenol at 5% concentration applied to the periocular region accelerate epithelial regeneration through provision of pantothenic acid, an essential cofactor for cellular metabolism and proliferation, while simultaneously providing occlusive hydration that reduces transepidermal water loss. Gentle thermal massage techniques applied for two to three minutes duration during warm compress application stimulate meibomian gland secretory activity and facilitate expression of inspissated lipid secretions that may have accumulated during the perioperative period, thereby helping to restore normal meibomian function and stabilize the tear film lipid layer essential for controlling evaporative water loss.

The extended rehabilitation phase, encompassing months one through six of the post-operative period, shifts therapeutic emphasis toward achieving sustainable restoration of normal ocular surface homeostasis, prevention of chronic dry eye syndrome development, and optimization of long-term visual comfort and quality of life outcomes through systematic application of mechanical therapies, advanced pharmacological interventions, nutritional optimization, and psychosocial support strategies. Meibomian gland expression performed systematically once daily for approximately five minutes per session with the eyelids maintained at temperatures of 38-40 degrees Celsius through application of specifically designed warming devices represents a cornerstone intervention during this phase, as regular mechanical expression prevents accumulation of inspissated lipid secretions within glandular ducts, promotes normalization of meibum quality and quantity, and stimulates resumption of normal autonomous secretory patterns. Comprehensive physiotherapeutic modalities incorporating low-level light therapy with light-emitting diode or infrared radiation sources applied two to three times weekly provide anti-inflammatory effects through modulation of cellular metabolism and cytokine expression, while microcurrent stimulation techniques facilitate restoration of normal neural impulse transmission patterns that may have been disrupted during surgery, and controlled ultrasonic massage at 20 kilohertz frequency for five-minute treatment sessions enhances tissue perfusion and metabolic activity supporting healing and functional restoration. Advanced pharmacotherapeutic strategies during the rehabilitation phase emphasize long-term maintenance with preservative-free hyaluronate formulations providing baseline ocular surface protection, supplemented with specialized lipid-complex artificial tears such as Systane Balance or Optive formulations that specifically address tear film lipid layer deficiencies common in post-DCR patients experiencing meibomian dysfunction, with these lipid-enhanced preparations containing phospholipid or triglyceride components that integrate into the natural tear film lipid layer and provide sustained stabilization of the air-tear interface to reduce evaporative loss.

Nutritional optimization strategies during the rehabilitation phase extend beyond omega-3 fatty acid supplementation to incorporate comprehensive dietary modifications emphasizing foods rich in vitamins A and E, which serve as essential cofactors for epithelial cell differentiation and provide antioxidant protection against oxidative stress that can compromise cellular function and promote inflammatory cascades. Specific dietary recommendations include regular consumption of egg yolks providing preformed vitamin A and lutein supporting macular health, fatty fish such as salmon or sardines offering high-quality omega-3 fatty acids in bioavailable forms, and nuts including almonds providing vitamin E along with additional beneficial lipids supporting cellular membrane integrity. Psychological rehabilitation interventions address the significant psychosocial dimensions of post-operative recovery through structured stress-reduction techniques including progressive muscle relaxation, mindfulness meditation, or guided imagery practices that reduce autonomic nervous system hyperactivity and may improve tear production through parasympathetic nervous system activation. Sleep hygiene optimization ensures adequate nocturnal recovery essential for tissue healing and hormonal regulation, while environmental modifications including reduced ambient lighting levels, increased humidity through use of room humidifiers, and strategic positioning of work or reading materials to minimize exposure of the ocular surface can substantially reduce evaporative stress and improve comfort. Integration of these multifaceted interventions throughout the six-month rehabilitation period results in progressive symptomatic improvement, with longitudinal outcome data demonstrating reduction in dry eye syndrome prevalence from approximately 30% at one month post-operatively to 8-10% at six months, accompanied by achievement of full visual comfort restoration in 92% of patients, thereby validating the clinical efficacy of comprehensive, phase-specific prophylactic protocols.

Evidence-based outcome analysis derived from systematic clinical trials and observational cohort studies provides quantitative validation of the therapeutic benefits associated with early initiation of comprehensive prophylactic and rehabilitative interventions following DCR surgery, with patients receiving structured early-phase protocols demonstrating 52% reduction in Ocular Surface Disease Index scores reflecting substantial improvement in subjective symptom burden, 25% increase in Tear Break-Up Time measurements indicating enhanced tear film stability and reduced evaporative tendency, 18% improvement in Schirmer test values reflecting augmented aqueous tear production capacity, and remarkable 1.8-fold elevation in overall

quality of life indices encompassing visual function, ocular comfort, psychosocial well-being, and functional capacity domains. These impressive outcome metrics underscore the substantial clinical value of proactive, multimodal management approaches compared to reactive treatment strategies implemented only after symptomatic dry eye syndrome has become established, supporting recommendations for universal implementation of comprehensive prophylactic protocols for all patients undergoing dacryocystorhinostomy procedures regardless of pre-existing dry eye risk factors or baseline ocular surface status.

3. Results and Clinical Analysis

Comprehensive prospective analysis of 180 patients undergoing dacryocystorhinostomy procedures, with systematic assessment of ocular surface status parameters, tear film stability metrics, and subjective symptomatology quantification performed at baseline prior to surgery and at designated intervals throughout a six-month postoperative observation period, provides robust empirical evidence regarding the natural history of post-DCR dry eye syndrome and the therapeutic efficacy of structured prophylactic and rehabilitative interventions. Baseline assessment conducted immediately prior to surgical intervention revealed that 30.2% of patients exhibited measurable dry eye syndrome symptomatology as quantified through validated assessment instruments, with mean Ocular Surface Disease Index scores of 26.8 ± 5.4 points indicating moderate symptom severity, Tear Break-Up Time measurements averaging 9.2 ± 2.3 seconds reflecting marginal tear film stability, and Schirmer test values of 8.9 ± 2.5 millimeters suggesting borderline aqueous tear production capacity. Following implementation of comprehensive prophylactic protocols initiated in the preoperative period and systematically continued throughout post-operative recovery, sequential reassessment at three months post-DCR demonstrated substantial improvement across all measured parameters, with the proportion of symptomatic patients declining to 14.5%, Ocular Surface Disease Index scores decreasing to 18.2 ± 4.1 points representing 32% reduction from baseline values, Tear Break-Up Time extending to 10.1 ± 2.1 seconds indicating improved tear film stability, and Schirmer test results increasing to 9.8 ± 2.2 millimeters reflecting enhanced tear production. By six months post-operatively, continued improvement was evident across all domains, with symptomatic patients reduced to merely 8.7% representing 71.2% decrease from baseline prevalence, Ocular Surface Disease Index scores declining further to 11.6 ± 3.2 points constituting 56.7% total reduction from pre-operative values, Tear Break-Up Time achieving 11.4 ± 1.9 seconds representing 23.9% improvement over baseline measurements, and Schirmer test values reaching 10.6 ± 2.4 millimeters indicating 19.1% enhancement in aqueous tear production capacity compared to preoperative status.

Detailed subgroup analysis revealed that patients receiving the complete integrated therapeutic protocol incorporating artificial tear supplementation, systematic meibomian gland expression, thermal therapies, and physiotherapeutic modalities demonstrated significantly more rapid symptom resolution and objective parameter normalization compared to patients receiving only conventional post-operative care consisting primarily of antibiotic prophylaxis and basic lubricating drop supplementation without structured rehabilitation components. Specifically, patients in the comprehensive intervention group achieved symptomatic remission an average of 6.4 weeks earlier than control subjects, exhibited 34% greater improvement in Tear Break-Up Time measurements at three-month assessment, and reported substantially higher satisfaction scores on quality of life questionnaires assessing visual function, ocular comfort, and treatment-related burden. Correlation analysis examining relationships between specific intervention components and outcome parameters identified meibomian gland expression combined with thermal therapy as particularly impactful for patients exhibiting predominant evaporative-type dry eye physiology, while those with primary aqueous-deficient features demonstrated optimal response to intensive artificial tear supplementation protocols combined with anti-inflammatory pharmacotherapy. These findings support the value of phenotype-specific treatment individualization within the framework of comprehensive prophylactic protocols, suggesting that optimal outcomes may be achieved through tailoring of intervention intensity and modality selection based on patient-specific dry eye pathophysiology rather than applying uniform protocols to all patients regardless of their individual clinical characteristics and risk profiles.

Discussion. The clinical outcomes and mechanistic insights derived from this comprehensive investigation of post-dacryocystorhinostomy dry eye syndrome prophylaxis and rehabilitation underscore the critical importance of adopting integrated, multidimensional therapeutic approaches that simultaneously address the diverse pathophysiological mechanisms contributing to ocular surface dysfunction rather than relying on single-modality interventions targeting only one aspect of the complex disease process. Traditional management paradigms emphasizing exclusive reliance on pharmaceutical interventions, typically consisting of artificial tear supplementation with or without anti-inflammatory agents, while providing symptomatic relief and supporting basic ocular surface hydration, prove insufficient for achieving complete microstructural restoration of the precorneal tear film architecture, normalization of meibomian gland morphology and secretory function, resolution of subclinical inflammatory processes affecting epithelial regeneration and goblet cell

populations, and optimization of neurosensory feedback mechanisms regulating reflex tear secretion. The superior outcomes observed in patients receiving comprehensive multimodal protocols incorporating pharmacotherapy, physiotherapy, nutritional optimization, and psychosocial support validate the conceptual framework of dry eye syndrome as a multifactorial condition requiring correspondingly multifaceted therapeutic responses, with synergistic interactions among intervention components producing aggregate benefits exceeding what could be achieved through simple additive effects of individual therapies applied in isolation.

Thermal therapies specifically designed to stimulate meibomian gland secretory function through controlled elevation of lid margin temperatures to optimal ranges of 38-40 degrees Celsius, thereby reducing meibum viscosity and facilitating expression of accumulated lipid secretions while simultaneously promoting increased autonomous glandular activity, represent particularly valuable interventions for patients exhibiting predominant evaporative-type dry eye pathophysiology characterized by tear film lipid layer deficiency. The mechanistic rationale supporting thermal interventions derives from recognition that meibomian lipid secretions become progressively more viscous at lower temperatures, potentially leading to obstruction of glandular orifices and reduced lipid delivery to the tear film, while controlled warming reduces lipid viscosity, facilitates flow through ductal systems, and may enhance glandular cellular metabolic activity supporting increased lipid synthesis. Clinical evidence demonstrating substantial improvements in meibomian gland expressibility, meibum quality scores, and tear film lipid layer thickness following systematic thermal therapy protocols validates this mechanistic model and supports routine incorporation of warming interventions into comprehensive dry eye management strategies, particularly during the rehabilitation phase when restoration of normal meibomian function represents a key therapeutic objective.

Nutritional interventions emphasizing increased dietary intake or supplementation with omega-3 polyunsaturated fatty acids, along with optimization of vitamin A and E status through consumption of nutrient-dense foods including egg yolks, fatty fish, nuts, and green leafy vegetables, provide systemic support for ocular surface health through multiple complementary mechanisms including modulation of eicosanoid metabolism favoring production of anti-inflammatory mediators over pro-inflammatory species, enhancement of meibomian lipid secretion quality through alterations in fatty acid composition, provision of essential cofactors for epithelial cell differentiation and mucin production, and antioxidant protection against oxidative stress-mediated cellular damage. The systemic nature of nutritional interventions offers advantages over purely topical therapies by addressing underlying metabolic factors contributing to dry eye pathogenesis rather than merely treating superficial manifestations, while the excellent safety profile of dietary modifications and nutritional supplementation combined with potential benefits extending beyond ocular health to include cardiovascular, cognitive, and anti-inflammatory effects makes these interventions particularly attractive components of comprehensive management protocols. Epidemiological evidence demonstrating inverse relationships between omega-3 fatty acid consumption and dry eye syndrome prevalence, along with controlled trial data showing symptomatic improvement and objective parameter enhancement following omega-3 supplementation, provides robust support for routine nutritional counseling and supplementation recommendations as standard components of post-DCR prophylactic care.

Conclusion. This comprehensive investigation of dry eye syndrome prophylaxis and rehabilitation following dacryocystorhinostomy procedures establishes that approximately 30% of patients experience clinically significant dry eye symptomatology during the post-operative period when managed with conventional care protocols, but that systematic implementation of evidence-based prophylactic interventions initiated preoperatively and continued systematically throughout the rehabilitation phase can reduce this symptomatic incidence to 8-10%, representing a clinically meaningful 71.2% relative risk reduction that translates into substantial improvements in patient comfort, visual function, and quality of life outcomes. The most efficacious prophylactic strategies identified through controlled analysis comprise integrated multimodal approaches incorporating preservative-free artificial tear supplementation with hyaluronate-based and lipid-enhanced formulations providing continuous ocular surface protection and tear film stabilization, systematic meibomian gland expression techniques combined with controlled thermal therapy to restore normal lipid secretion patterns and tear film lipid layer integrity, nutritional optimization through omega-3 fatty acid supplementation and vitamin-rich dietary modifications supporting anti-inflammatory pathways and cellular metabolic requirements, and physiotherapeutic modalities including low-level light therapy and microcurrent stimulation facilitating tissue healing and functional restoration.

References:

1. Craig J.P. et al. TFOS DEWS II Management and Therapy Report. *Ocul Surf*, 2017.
2. Kim S. et al. Rehabilitation Strategies after DCR Surgery. *Korean J Ophthalmol*, 2023.

3. Lee H. et al. Preventive Measures for Postoperative Dry Eye. *Br J Ophthalmol*, 2021.
4. Choi Y. et al. Thermal Therapy for Meibomian Gland Dysfunction. *Int J Ophthalmol*, 2022.
5. Bukhari A. Role of Omega-3 Fatty Acids in Dry Eye Prevention. *Saudi J Ophthalmol*, 2019.
6. Bron A.J. et al. TFOS DEWS II Pathophysiology Report. *Ocul Surf*, 2017.
7. Nichols K.K. et al. The International Workshop on Meibomian Gland Dysfunction: Executive Summary. *Invest Ophthalmol Vis Sci*, 2011.
8. Stapleton F. et al. TFOS DEWS II Epidemiology Report. *Ocul Surf*, 2017.
9. Pflugfelder S.C., de Paiva C.S. The Pathophysiology of Dry Eye Disease. *Ophthalmology*, 2017.
10. Schaumberg D.A. et al. Prevalence of Dry Eye Disease among Adults. *Am J Ophthalmol*, 2003.
11. Lemp M.A. et al. Tear Osmolarity in the Diagnosis and Management of Dry Eye Disease. *Am J Ophthalmol*, 2011.
12. Geerling G. et al. Artificial Tears: A Systematic Review. *Br J Ophthalmol*, 2011.
13. Finis D. et al. Evaluation of an Automated Thermodynamic Treatment for Meibomian Gland Dysfunction. *Cornea*, 2014.
14. Sullivan D.A. et al. Meibomian Gland Dysfunction: Advances in Diagnosis and Treatment. *Ocul Surf*, 2014.

For citation: Shayeva R.G. Operative approaches for congenital palate defects: addressing tissue deficiency in the treatment // *Bulletin of Fundamental and Clinic Medicine*. – 2026. – № 2(22). – P. 383–390. doi: <https://doi.org/10.5281/zenodo.18654731>