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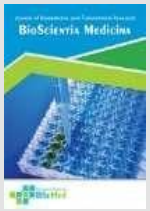

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Pediatric Steroid High-Responder: Irreversible Visual Loss and Secondary Glaucoma Following Chronic Cutaneous-Ocular Dexamethasone Misuse

I Ketut Anom Widyantara Eka Dana Weka Mona[1*], Ni Made Ayu Surasmiati[2], I Gusti Ayu Ratna Suryaningrum[3]

[1]Residency Study Program, Department of Ophthalmology, Faculty of Medicine, Universitas Udayana, Denpasar, Indonesia

[2]Pediatric Ophthalmology and Strabismus Division, Department of Ophthalmology, Faculty of Medicine, Universitas Udayana, Denpasar, Indonesia

[3]Glaucoma Division, Department of Ophthalmology, Faculty of Medicine, Universitas Udayana, Denpasar, Indonesia

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***Corresponding author:**
I Ketut Anom Widyantara Eka Dana Weka Mona

E-mail address:
anomw006@gmail.com

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A B S T R A C T

Background: Chronic misuse of potent topical corticosteroids can lead to irreversible visual loss, particularly in the pediatric population which exhibits a more aggressive trabecular meshwork remodeling response than adults. This case aims to delineate the silent progression of steroid-induced ocular hypertension in the absence of red-eye symptoms and emphasizes the critical pharmacokinetic risks of cutaneous-ocular absorption in children. **Case presentation:** We report a catastrophic case of a 7-year-old male presenting with irreversible vision loss following six years of unsupervised, intermittent use of a combined Neomycin-Polymyxin B-Dexamethasone ointment for recurrent hordeolum. The cumulative exposure exceeded 125 mg of Dexamethasone. Examination revealed bilateral dense posterior subcapsular cataracts (PSC) and advanced glaucomatous optic neuropathy in left eye. Following sequential phacoaspiration, the left eye showed persistently elevated IOP (IOP elevated up to 59 mmHg) attributed to decompensated outflow facility. Management required Trabeculectomy with intraoperative 5-Fluorouracil (5 mg/0.1 mL). The high- responder phenotype in children involves rapid formation of Cross-Linked Actin Networks (CLANs) and MYOC gene upregulation. We discuss the double-hit mechanism where cataract extraction washes out hyposecretory factors, unmasking total trabecular blockage. The choice of 5-Fluorouracil over Mitomycin C is defended based on the safety profile regarding hypotony maculopathy in pediatric myopic eyes**. Conclusion:** Dexamethasone carries a high risk of transcutaneous-ocular absorption in pediatric eye and eyelids. Regulatory reform reclassifying antibiotic-steroid combinations as non-repeatable prescriptions is imperative to prevent such preventable blindness.

1. Introduction

Since their introduction into the pharmacological armamentarium in the mid-20th century, corticosteroids have represented a quintessential double-edged sword in ophthalmology.[1] While their potent anti-inflammatory and immunosuppressive properties are indispensable for preserving vision in conditions ranging from uveitis to post-operative inflammation, their chronic administration carries a well-documented, yet frequently underestimated, risk of ocular morbidity. Among the most insidious of these complications is steroid-induced ocular hypertension (OHT) and its progression to secondary open-angle glaucoma. This phenomenon, historically termed steroid responsiveness, is not merely a side effect but a complex genetic and physiological phenotype

characterized by a significant, dose-dependent elevation in intraocular pressure (IOP) following the administration of glucocorticoids.[2]

The susceptibility to steroid-induced IOP elevation is not uniform across the population. Seminal work by Armaly and Becker in the 1960s established a trimodal distribution of responsiveness in the general population: non-responders, moderate responders, and high responders. While high responders—those exhibiting an IOP elevation of greater than 15 mmHg—constitute approximately 5% to 6% of the adult population, the pediatric demographic presents a distinct, more volatile, and clinically aggressive susceptibility profile. The pediatric eye is not simply a miniaturized adult eye; it is a dynamic, developing physiological system with a trabecular meshwork (TM) that is functionally immature.[3] In the adult eye, the TM possesses a certain degree of resilience and homeostatic regulation. In contrast, the pediatric TM is prone to rapid, often irreversible, extracellular matrix (ECM) remodeling when exposed to corticosteroids. This high-responder phenotype in children is driven by an accelerated alteration in the cytoskeleton of the trabecular cells, specifically the formation of Cross-Linked Actin Networks (CLANs) and the inhibition of protease activity, leading to a deposition of glycosaminoglycans and a catastrophic reduction in outflow facility.[4]

The route of administration plays a pivotal role in the epidemiology of this condition, yet it remains the source of a profound clinical blind spot. While ophthalmologists are acutely aware of the risks associated with topical steroid eye drops, the systemic and ocular absorption of corticosteroids applied to the periocular skin—specifically ointments and creams used for eyelid—is frequently underestimated by non-ophthalmic practitioners, pediatricians, and general practitioners.[5] This underestimation stems from a fundamental misunderstanding of pediatric dermatological pharmacokinetics. The pediatric eyelid is characterized by the thinnest stratum corneum in the human body, combined with a high surface-area-to-body-weight ratio and robust vascularity.[6] These anatomical factors facilitate rapid transdermal absorption that far exceeds that of adult skin. Furthermore, the mechanics of the blink reflex introduce a secondary route of entry: the orbicularis oculi muscle pump mechanism. This physiological action actively transports dermatological preparations from the lid margin and the periocular skin over the mucocutaneous junction and into the tear film. This creates a reservoir effect, where a dermatological ointment effectively functions as a sustained-release ocular depot, mimicking the pharmacodynamics of direct instillation but without the dosing precision.[7]

The clinical presentation of steroid-induced glaucoma in this context is particularly treacherous due to its asymptomatic nature.[8] Unlike acute angle-closure glaucoma, which presents with pain, nausea, and a red eye that immediately alerts parents and providers to a crisis, steroid-induced open-angle glaucoma is a white and quiet disease. The eye remains cosmetically normal, with no conjunctival hyperemia or photophobia, even as the IOP climbs to dangerous levels. In the pediatric population, this silence is compounded by the inability of young children to articulate visual field loss or subtle changes in visual acuity. Consequently, the disease is often permitted to progress unchecked for months or even years. By the time clinical signs become overt—manifesting as leukocoria (white pupil) due to posterior subcapsular cataract formation, sensory exotropia, or behavioral changes due to vision loss—the optic nerve damage is frequently advanced and irreversible.[9]

This silent progression is exacerbated when the inciting agent is Dexamethasone. As a potent synthetic glucocorticoid, Dexamethasone possesses a high intrinsic ability to penetrate ocular tissues and a strong affinity for glucocorticoid receptors in the trabecular meshwork. The combination of a potent agent, a vulnerable pediatric physiology, and an unregulated route of administration creates a perfect storm for ocular disaster. The resulting pathology is often a dual entity: the formation of dense cataracts that obscure the fundus and the concurrent

excavation of the optic nerve. The management of these cases presents a unique surgical paradox. The removal of the cataract, while necessary to restore the visual axis, can precipitate uncontrollable IOP—a phenomenon of decompensated outflow facility—which poses an immediate threat to the remaining islands of vision.[10]

Therefore, this case report documents a catastrophic cascade of irreversible vision loss and secondary glaucoma in a 7-year-old child following chronic, unsupervised cutaneous-ocular use of a Dexamethasone-containing ointment. This study aims to transcend the boundaries of a traditional case report by integrating clinical findings with methodological analysis of the disease mechanism. Specifically, the aim and novelty of this study are twofold. First, we aim to highlight the diagnostic gap where severe glaucomatous optic neuropathy progresses in the absence of external inflammatory signs, emphasizing the critical need for regulatory re-evaluation of antibiotic-steroid dermatological combinations. Second, we provide a novel analysis of the post-phacoemulsification persistently elevated IOP in steroid-hardened eyes. By bridging clinical observation with the molecular pathophysiology of trabecular meshwork exhaustion, we aim to offer a new framework for understanding why standard sequential surgery fails in these high-responder phenotypes, thereby advocating for altered surgical protocols in pediatric steroid-induced glaucoma.

2. Case Presentation

Written informed consent was obtained from the patient's legal guardians for the publication of this case report and accompanying images.

A 7-year-old Asian male was referred to a tertiary ophthalmology center with a chief complaint of progressive, painless vision loss of the left eye over the preceding 12 months. The parents reported a whitish appearance of the pupils (leukocoria) noted three months prior to presentation. Detailed anamnesis revealed a history of recurrent hordeolum starting at age 12 months, especially more frequent in the left eye. The recurrence of hordeolum appears nearly every month in the LE, but only appears every 3-6 months in the RE. The patient had been managed with a combination ointment containing Neomycin Sulfate (3.5 mg), Polymyxin B Sulfate (6.000 IU), and Dexamethasone (1.0 mg) per gram. The parents admitted to applying the ointment to the eyelids and lid margins intermittently for six years. Frequency was reported at 2–3 times weekly during recurrency, with periods of daily use for up to two weeks. Based on the report of purchasing a new tube roughly every 2 months, the cumulative exposure is estimated at >35 tubes (3.5g each), equating to a total Dexamethasone load exceeding 125 mg delivered directly to the periocular surface, with significant transconjunctival seepage. The patient had no history of asthma, nephrotic syndrome, or autoimmune conditions. No systemic (oral or inhaled) steroids were ever prescribed, ruling out systemic confounders (Table 1).

Upon admission, the patient's visual behavior was characterized by a profound degree of severe functional deficits of the left eye revealed during testing. Best corrected visual acuity (BCVA) assessments found the right eye (RE) 6/9 (LogMAR 0.2) and the left eye (LE) reduced to hand motion (LogMAR 2.3). Intraocular pressure (IOP) quantification via Goldmann applanation tonometry (GAT) yielded a normal reading of 14 mmHg of the RE and elevated readings of 29 mmHg in the LE. However, these raw values belied the true severity of the ocular hypertension. Pachymetric evaluation revealed notably thin central corneal thickness measurements of the LE (535 μm RE; 508 μm LE). Methodologically, this introduces a significant negative artifact in applanation tonometry; when corrected for corneal thinning, the adjusted IOP in the left eye was estimated to exceed 32 mmHg, placing the patient in a state of hypertensive emergency despite the absence of acute symptoms.

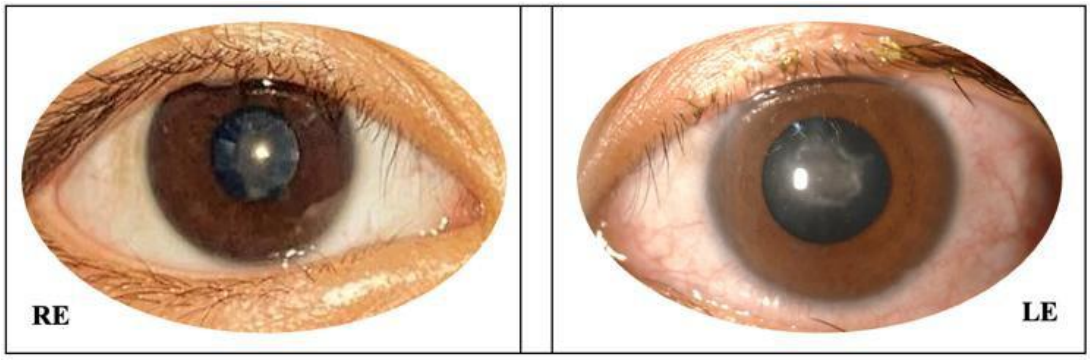

Figure 1. Slit-lamp examination shows opacity in the central posterior part of the lens, the size of the opacity in the right eye is greater than left eye and covers the visual axis.

Slit-lamp biomicroscopy unveiled the deceptive quiet nature of steroid-induced pathology (Figure 1). The conjunctiva remained white and non-hyperemic, devoid of the ciliary flush typically associated with acute glaucoma. The optical axis, however, was obstructed by dense, posterior subcapsular cataracts (PSC, Grade II) in both eyes—a hallmark of glucocorticoid toxicity—which precluded direct fundoscopic visualization of the optic nerve (Figure 2). Gonioscopic examination ruled out angle-closure mechanisms. The angles were open (Shaffer Grade IV) for 360 degrees, yet exhibited a pathological, homogeneous hyperpigmentation of the Trabecular Meshwork. This specific pigmentation pattern is pathognomonic for chronic steroid-induced particulate obstruction, distinct from the patchy distribution observed in pigment dispersion syndrome. Furthermore, ocular biometry provided structural evidence of chronicity. An asymmetry in axial length was noted, with the RE measuring normal 22.08 mm and the LE extending to 24.26 mm. This axial elongation of the left eye indicates a myopic shift driven by scleral stretching, confirming that the uncontrolled ocular hypertension had been exerting mechanical stress on the globe for a prolonged period.

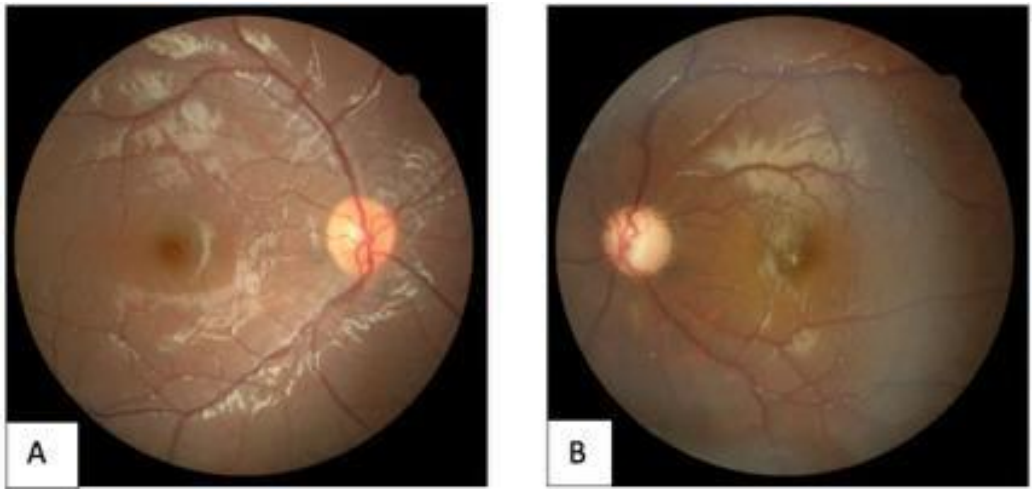

Figure 2. Funduscopy examination on the right eye (RE) and the left eye (LE). A) RE shown normal optic nerve head with CDR 0.3; B) LE shown glaucomatous optic neuropathy of the optic nerve head with CDR 0.9, accompanied by cupping and nasalization.

Table 1. Summary of clinical findings on admission.

Parameter	Right Eye (RE)	Left Eye (LE)	Clinical Interpretation
I. VISUAL FUNCTION ASSESSMENT			
BCVA (LogMAR)	6/9 (0.2)	**Hand Motion (HM)** (Approx. 2.3)	*Severe unilateral impairment of the LE; suggests advanced optic nerve compromise.*
II. TONOMETRY & BIOMETRY PROFILE			
Intraocular Pressure (GAT)	14 mmHg	**29 mmHg**	*Significant Ocular Hypertension (Normal range: 10-21 mmHg).*
Central Corneal Thickness (CCT)	535 μm	**508 μm**	*Thin cornea of the LE (< 520 μm) causes significant underestimation of true IOP.*
Adjusted IOP Estimate	15 mmHg	**32 mmHg**	*High IOP of the LE confirms hypertensive emergency.*
Axial Length (AL)	22.08 mm	**24.26 mm**	*LE asymmetry indicates scleral stretching (Buphthalmic shift).*
III. SLIT LAMP BIOMICROSCOPY			
Conjunctiva & Cornea	White/Clear	White/Clear	*White eye presentation masking underlying pathology.*
Lens Status	Posterior Subcapsular Cataract (Grade III)	Posterior Subcapsular Cataract (Grade III)	*Dense steroid-induced opacity obscuring visual axis and fundus view.*
IV. GONIOSCOPY & ANGLE ANATOMY			
Angle Grade (Shaffer)	Grade IV (Open 360)	Grade IV (Open 360)	*Rules out angle-closure glaucoma.*
Trabecular Meshwork	Mild Homogenous Hyperpigmentation	**Moderate Homogenous Hyperpigmentation**	*Pathognomonic for chronic steroid particulate deposition and remodeling.*

Table 2 synthesizes the clinical trajectory from definitive diagnosis through the sequential surgical management to the final physiological outcomes. The primary diagnosis was established as pediatric steroid-induced glaucoma with a high-responder phenotype, a condition precipitated by the chronic, unsupervised absorption of Dexamethasone. This etiology was confirmed by the pathognomonic clinical triad: the history of substantial cumulative steroid exposure (>125 mg estimated), the presence of dense posterior subcapsular cataracts, and the characteristic hyperpigmentation of the trabecular meshwork. A secondary diagnosis was uncontrolled elevated intraocular pressure (IOP) despite maximal medical therapy, complicated by the double-hit pathology of concurrent functional obstruction of the visual axis and the drainage angle.

The surgical management strategy was dictated by the need to address both the visual obstruction and the persistently elevated IOP, though the sequential approach. Phase 1 involved phacoaspiration with posterior chamber intraocular lens (PCIOL) implantation. A +20.5 D lens was selected to target slight hyperopia (+1.0 D), a strategic choice to accommodate the anticipated myopic shift as the child grows. However, with this intervention, the IOP elevated up to 59 mmHg. This event confirmed the hypothesis that the removal of the cataractous lens washed out endogenous hyposecretory factors, effectively unmasking the total blockade of the trabecular meshwork.

Phase 2 necessitated a trabeculectomy with antimetabolite. Intraoperative 5-Fluorouracil (5-FU) (5 mg/0.1 mL for 2 minutes) was utilized instead of the more potent Mitomycin C (MMC). This decision was driven by

a safety-first philosophy; given the patient's thin sclera and myopic axial elongation (24.26 mm), MMC posed an unacceptable risk of hypotony maculopathy—a complication where low pressure causes choroidal effusion and macular folding. 5-FU allowed for a more modulated healing response, prioritizing the formation of a safe, diffuse filtering bleb over the aggressive, thin-walled cystic blebs often associated with MMC.

The six-month post-operative outcome highlighted the divergence between anatomical success and functional limitation. Anatomically, the surgery was a triumph; the IOP stabilized at a physiological 12 mmHg without the need for topical hypotensive medications, and a functional filtering bleb was established. However, the functional prognosis remained guarded. The final best corrected visual acuity (BCVA) was limited to 1/300 (Hand motion) (LogMAR 2.3). This poor visual potential was not a surgical failure but a reflection of the irreversible structural damage sustained prior to intervention. Fundoscopic and OCT analysis revealed a Cup-to-Disc Ratio of 0.9 and retinal nerve fiber layer (RNFL) thickness near the floor effect (50 µm), confirming that the optic nerve had been destroyed by the silent progression of glaucoma long before the cataracts were removed.

3. Discussion

The case presented herein serves as a grim sentinel event, illuminating the devastating intersection of pediatric physiology, pharmacological potency, and regulatory gaps. The transition of this patient from a manageable, mild ocular condition, like a hordeolum, to a state of irreversible blindness represents a failure of the healthcare safety net at multiple levels. While the clinical narrative describes the what and the when, this discussion aims to dissect the how and why, moving beyond the descriptive to provide a rigorous analysis of the molecular pathology and the complex surgical decision-making required in the management of the pediatric steroid high-responder phenotype. The hallmark of this case—the steroid high-responder phenotype—is not merely a statistical outlier but a distinct biological entity driven by a specific genetic and cellular susceptibility.[11]

TABLE 2. DIAGNOSIS, TREATMENT PROTOCOL, FOLLOW-UP AND FINAL OUTCOME

Clinical Phase	Findings / Intervention	Methodological Rationale / Notes
I. DEFINITIVE DIAGNOSIS		
Primary Diagnosis	**Pediatric Steroid-Induced Glaucoma** (High-Responder Phenotype)	***Etiology:*** *Chronic cutaneous-ocular Dexamethasone absorption.* ***Load: >125 mg est.***
Secondary Diagnosis	1. Bilateral Posterior Subcapsular Cataract (Grade III) 2. Refractory Intraocular Hypertension	***Double-Hit Pathology*** *Functional obstruction of both visual axis and trabecular meshwork.*
II. SEQUENTIAL SURGICAL MANAGEMENT (LEFT EYE)		
Phase 1: Cataract	**Phacoaspiration + PCIOL Implantation** (Lens Power: +20.5 D)	*Target: Hyperopia (+1.0 D) to accommodate pediatric myopic shift.*
CRITICAL EVENT **Complication**	IOP Elevated up to **59 mmHg**	***Pathophysiology:*** *Removal of lens washed out hyposecretory factors, unmasking total TM blockage.*
Phase 2: Glaucoma	**Trabeculectomy** + Intraoperative **5-Fluorouracil (5-FU)** (5 mg/0.1 ml for 2 mins)	***Rationale for 5-FU vs MMC:*** *5-FU chosen to modulate healing safely. MMC poses high risk of hypotony maculopathy in thin, myopic pediatric sclera.*
III. SIX-MONTH POST OPERATIVE OUTCOME		
IOP Control	**Success** Final IOP: **12 mmHg**	*(Without Topical Medications)* *Anatomical success: Diffuse, avascular, functional filtering bleb established.*
Structural Damage	**Severe / Irreversible** Cup-to-Disc Ratio: **0.9** RNFL Thickness: 50 µm (Floor Effect)	*Optic nerve head pallor and vessel bayoneting confirm end-stage neuropathy.*
Visual Function	**Poor Prognosis** Final BCVA: **1/300 (Hand Motion)** (LogMAR 2.3)	*Vision limited by profound optic atrophy despite clear visual axis and normalized pressure.*

The intraocular pressure (IOP) elevation observed in this child is the functional output of a profound structural remodeling of the conventional outflow pathway, a process we characterize as a double hit on the trabecular meshwork (TM).

The primary driver of this pathology is likely the upregulation of the Myocilin (MYOC) gene (formerly known as TIGR).[12] In the general population, the trabecular meshwork functions as a self-regulating sieve, maintaining IOP homeostasis through the active turnover of the extracellular matrix (ECM). However, in genetically susceptible individuals, the binding of Dexamethasone to the glucocorticoid receptor-alpha (GR-α) in the nucleus of TM cells triggers a maladaptive transcriptional program. This initiates the first hit: cytoskeletal reorganization (Figure 3). Under normal physiological conditions, TM cells possess a contractile tone that helps regulate the porosity of the meshwork.[13] Steroid exposure induces the rapid and aberrant formation of Cross-Linked Actin Networks (CLANs). These are not typical actin stress fibers; rather, they are rigid, geodesic, dome-like structures that stiffen the cell and impair its contractility. In the pediatric eye, where the TM is functionally immature and highly plastic, this cytoskeletal rigidification occurs more rapidly and with greater severity than in the adult eye. The TM cells effectively become frozen, losing their ability to mechanically facilitate the pulsatile clearance of aqueous humor.[14]

The second hit is the dysregulation of the extracellular matrix. Concurrently with cytoskeletal changes, corticosteroids exert a potent inhibitory effect on the proteolytic enzymes of the eye. Specifically, matrix metalloproteinases (MMPs) and stromelysins. These enzymes are the janitors of the outflow pathway, responsible for degrading old proteins and keeping the juxtacanalicular tissue (JCT) clear.[15] Dexamethasone suppresses MMP activity while simultaneously upregulating the production of ECM components like fibronectin and glycosaminoglycans (GAGs). This creates an imbalance where debris is produced but not cleared. In the juxtacanalicular region—the site of highest resistance to outflow—this leads to the accumulation of a dense, amorphous plaque of material. The result is a physical, mechanical blockage of the drainage angle. Crucially, unlike the reversible edema seen in some inflammatory conditions, this fibrotic remodeling can become self-perpetuating. In children, whose ECM turnover rates are naturally higher to accommodate ocular growth, this steroid-induced fibrosis hardens faster. This explains the refractory hypertension observed in our patient; even after the drug was withdrawn, the physical barrier to outflow remained, necessitating surgical bypass.

The severity of the clinical presentation is also a direct function of the pharmacological agent employed.[16] The choice of dexamethasone phosphate in the offending ointment was critical. In the hierarchy of ocular steroids, dexamethasone is classified as a high-potency agent with significant intraocular penetration, far exceeding that of soft steroids like Fluorometholone or Hydrocortisone. Its phosphate structure allows for high solubility and rapid transit across biological membranes.

A pivotal learning point from this case is the underestimation of the transcutaneous and trans-conjunctival routes of absorption in pediatrics. There is a prevalent misconception among non-ophthalmic practitioners that ointments applied to the eyelid skin are external and therefore safe from internal ocular side effects. This case definitively refutes that assumption. The pediatric eyelid is an anatomical structure uniquely predisposed to systemic and local drug delivery. It possesses the thinnest stratum corneum in the human body, offering minimal resistance to diffusion.[17] Furthermore, the high surface-area-to-body-weight ratio and the rich vascularity of the pediatric lid margin facilitate rapid uptake. Beyond passive diffusion, the blink reflex creates a mechanical pump. The orbicularis oculi muscle actively transports the ointment from the lid margin over the mucocutaneous junction and into the tear film. Once in the tear film, the ointment base acts as a reservoir, increasing the contact time of the drug with the cornea and conjunctiva significantly longer than a drop would. Consequently, the chronic application of Dexamethasone ointment to a child's eyelids is pharmacokinetically equivalent to, or perhaps more potent than, the chronic instillation of steroid drops. This reservoir effect ensures that the trabecular meshwork is bathed in a continuous, high- concentration solution of glucocorticoids, accelerating the remodeling processes described above.

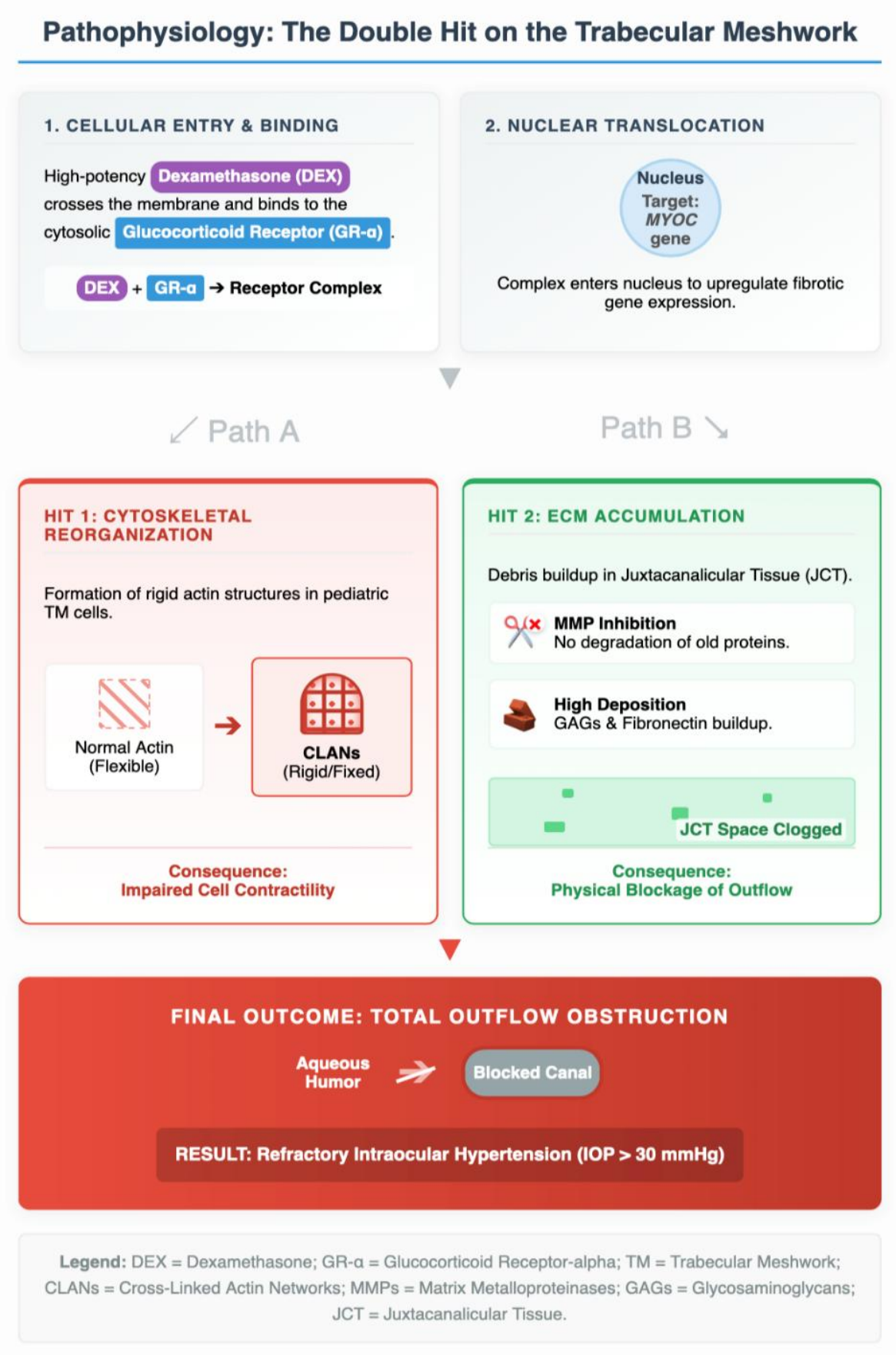

Figure 3. Pathophysiology: The double hit on the trabecular meshwork.

One of the most clinically instructive aspects of this case was the elevated IOP up to 59 mmHg following uncomplicated cataract surgery. This phenomenon, which we term decompensated outflow facility, offers a window into the fluid dynamics of the steroid-damaged eye. Prior to surgery, the eye was maintaining an IOP of 32 mmHg—high, but not explosive. This relative stability was likely maintained by a state of ciliary body hyposecretion. Chronic ocular inflammation, and potentially the mechanical presence of the cataract itself, can suppress the ciliary epithelium, reducing the production of aqueous humor. Additionally, endogenous prostaglandins released during chronic stress may have enhanced the uveoscleral outflow (the secondary drainage pathway), acting as a natural safety valve.[18]

The act of phacoemulsification disrupted this fragile equilibrium. The removal of the lens and the thorough irrigation-aspiration of the anterior chamber achieved two things. First, it removed the suppression of the ciliary body, allowing aqueous production to return to normal, robust levels. Second, the high-volume fluid exchange likely washed out the endogenous hyposecretory factors and prostaglandins that were keeping the uveoscleral pathway open. The eye suddenly had a tap that was fully open (normal aqueous production) but a drain that was completely cemented shut by steroid-induced CLANs and ECM deposition. With the uveoscleral safety valve also washed out, there was nowhere for the fluid to go. The IOP elevated up to 59 mmHg, a pressure level capable of causing central retinal artery occlusion and rapid optic nerve infarction. This confirms that in steroid-induced glaucoma, the absence of an acute spike pre-operatively does not imply a functional trabecular meshwork; it often masks a system that is on the verge of total hydraulic failure.[19]

The management of this persistently elevated IOP proceed with Trabeculectomy using 5- Fluorouracil (5-FU) rather than Mitomycin C (MMC) warrants a specific methodological defense, as it deviates from the common preference for MMC in pediatric glaucoma. MMC is an alkylating agent that cross-links DNA, effectively causing cell death in fibroblasts. It is potent and results in very thin, avascular blebs. While effective at lowering pressure, MMC is associated with a higher long-term risk of bleb-related endophthalmitis and, crucially, hypotony maculopathy. Hypotony maculopathy occurs when the IOP drops too low, causing the scleral wall to collapse and the choroid to swell, leading to permanent wrinkling of the macula and vision loss. In this specific patient, the risk profile for hypotony was prohibitively high. The biometric data revealed an axial length of 24.26 mm, indicating a myopic, stretched eye with thin, elastic sclera. Pediatric sclera is already more compliant than adult sclera; when combined with myopic thinning, it creates a globe that is highly susceptible to collapse if the pressure drops too rapidly or remains too low.

Therefore, we opted for 5-FU (50 mg/mL). Unlike the nuclear option of MMC, 5-FU is an antimetabolite that interferes with RNA synthesis, exerting a more bacteriostatic-like effect on fibroblast proliferation. It allows for a more modulated healing response. By using 5-FU, we aimed to create a broader, thicker, and more diffuse filter, rather than a thin, cystic one. This choice prioritizes safety over absolute potency. It acknowledges that while the re-operation rate might theoretically be higher with 5-FU, the risk of blinding hypotony complications is significantly lower [11]. Furthermore, 5-FU allows for post-operative titration; if the bleb begins to fail, additional 5-FU can be injected subconjunctivally to revive it (bleb needling), offering a dynamic management strategy that MMC does not permit once the initial dose is given.[20]

It is incumbent upon us to acknowledge the limitations of this report to place our findings in the proper scientific context. The primary limitation is the retrospective estimation of the cumulative steroid dosage. Relying on parental recall for the frequency of ointment application (2-3 times a week, every 2 months) introduces a margin of error. However, the clinical consistency of the findings—dense cataracts and advanced glaucoma—validates the assertion that the dosage was substantial and toxic. Additionally,

this study was conducted in a resource-limited setting where genetic testing for MYOC variants was unavailable. Confirmatory genotyping would have definitively categorized the patient as a homozygous or heterozygous responder, adding a layer of molecular precision to the clinical diagnosis. Nevertheless, the phenotypic evidence is overwhelming and sufficient to draw the clinical conclusions presented.

4. Conclusion

This case of a catastrophic cascade is a sobering indictment of the trivialization of topical corticosteroids. It emphasizes that pediatric steroid sensitivity is not merely a localized ocular side effect but a systemic toxicological emergency with life-altering consequences. The white and quiet presentation of this glaucoma serves as a perfect camouflage, misleading parents and non-ophthalmic providers into a false sense of security while the optic nerve is methodically destroyed. The devastation documented here was entirely preventable, underscoring that the current regulatory and educational frameworks surrounding the prescription of dermatological-ophthalmic combinations are inadequate. To prevent the recurrence of such preventable blindness, we propose the following three- pillared clinical and regulatory strategy. The first line of defense is the point of sale. We advocate for a stricter policy regarding antibiotic-steroid combinations. These medications should be reclassified to a higher restrictive status. Pharmacists must be empowered—and legally mandated—to act as gatekeepers. A zero-tolerance policy on unmonitored refills should be instituted, where pharmacists are authorized to refuse refills of steroid-containing ointments without a new, current prescription dated within the last 30 days. Automated pharmacy systems should flag any pediatric patient receiving repeat dermatologic steroids as high risk for ocular toxicity. The prescription of periocular steroids must be inextricably linked to ocular monitoring. We propose a clinical guideline stating that any child prescribed topical corticosteroids (drops or periocular ointments) for a duration exceeding two weeks must undergo baseline and follow-up tonometry. This should be the standard of care for dermatologists and pediatricians. If these specialists cannot perform tonometry, a referral to an ophthalmologist must be a mandatory component of the treatment plan. The concept of treating the skin without checking the eye must be considered a breach of the standard of care. For the ophthalmic surgeon, this case reinforces the need for anticipatory planning. Surgeons must recognize the post-phaco decompensation phenomenon as a predictable event in steroid responders, not a rare complication. In cases of steroid-induced cataracts with elevated or borderline IOP, a sequential approach (cataract first, glaucoma later) carries the high risk of an persistent elevated IOP, as seen in this patient. We strongly advocate for the consideration of combined phaco-trabeculectomy as the primary intervention. This single-stage approach addresses the visual axis obstruction and the outflow blockade simultaneously, sparing the child the risk of an IOP spike and the trauma of a second general anesthesia. In summary, the preservation of vision in the pediatric steroid responder demands a paradigm shift: from reactive management of complications to proactive prevention through strict dispensing regulation and aggressive early monitoring.

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