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Case Report

Acute Intraocular Pressure Elevation as the Initial Presentation of Ophthalmia Nodosa: A Case Report

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Abstract

Purpose: To report an unprecedented presentation of ophthalmia nodosa in which acute secondary intraocular pressure elevation served as the initial clinical manifestation, temporarily obscuring the underlying diagnosis.

Case Presentation: We report the case of a 27-year-old male who presented to the emergency department with acute foreign-body sensation in the eye after exposure to a sudden air burst and vigorous eye rubbing. He subsequently developed escalating ocular pain accompanied by nausea, vomiting, and headache. Initial examination revealed diffuse conjunctival hyperemia, corneal edema with Descemet's membrane folds, epithelial defects, a mid-dilated poorly reactive pupil, hazy anterior chamber precluding visualization of intraocular structures and intraocular pressure of 45 mmHg left eye. Initial management with topical and systemic intraocular pressure-lowering therapy was effective. Persistent corneal edema and mydriasis prompted re-evaluation; after intraocular pressure reduction with early therapeutic response and improved cooperation, detailed slit-lamp biomicroscopy with high magnification revealed multiple filamentous foreign bodies embedded at various depths in the cornea, with several penetrating into the anterior chamber and on the anterior iris surface, while Seidel testing remained negative. Diagnosis of Ophthalmia Nodosawas established based on history, environmental context and characteristic morphology of the setae. Treatment was enhanced with topical and systemic antibiotics, antifungals and corticosteroids. Orbital Computed Tomography and facial sinus radiography were performed. During hospitalization, clinical improvement was observed with stable intraocular pressure control. The patient was discharged with a strict topical regimen and close follow-up. At two-month follow-up, intraocular pressure remained controlled on topical therapy, visual acuity improved though persistent irregular pupil with ectropion uveae, corneal foreign bodies and sectoral iris atrophy remained. Long-term follow-up demonstrated chronic low-grade inflammation, steroid-induced cataract formation and eventual need for phacoemulsification 1.5 years later.

Conclusion: This case represents the first reported presentation of ophthalmia nodosa with acute intraocular pressure elevation as the initial clinical manifestation. The resultant corneal edema temporarily masked the underlying diagnosis until therapeutic intervention allowed visualization of the causative lepidopteran setae. This unique presentation highlights the necessity for high clinical suspicion of ophthalmia nodosa in acute ocular trauma cases, particularly in endemic regions during processionary caterpillar season, and emphasizes the importance of sequential examination after therapeutic intervention. Early recognition and appropriate management are crucial for preventing vision-threatening complications.

Keywords: Ophthalmia nodosa, Caterpillar setae, Acute intraocular pressure elevation, Thaumetopoea pityocampa, Ocular trauma, Lepidopteran setae, Penetrating ocular injury

Abbreviations: IOP: Intraocular Pressure; LE: Left Eye; RE: Right Eye; VA: Visual Acuity; AS-OCT: Anterior Segment Optical Coherence Tomography; CT: Computed Tomography; mmHg: Millimeters of Mercury; BCVA: Best Corrected Visual Acuity

Introduction

Ophthalmia nodosa induced by lepidopteran setae represents a complex ophthalmological condition with potentially severe visual consequences [1,2]. The condition, also known as caterpillar hair-induced ophthalmitis, results from direct ocular contact with urticating hairs from caterpillars, particularly those of the Lepidoptera order [3,4]. The pathophysiology involves both direct mechanical trauma from setae penetration and toxic inflammatory mechanisms affecting multiple ocular structures.

The Pine Processionary caterpillar (Thaumetopoea pityocampa) is endemic to Mediterranean regions and represents a characteristic example of a lepidopteran species with significant ophthalmotoxicity. Its setae contain the protein thaumetopoein, which induces immediate toxic reactions through direct cellular damage and IgE-mediated allergic responses. Climate change has significantly affected the biological cycle of these insects, leading to earlier larval emergence, expanded geographic distribution and consequently increased frequency of human ocular exposure incidents [5,6].

The clinical presentation of ophthalmia nodosa varies widely depending on the anatomical location and depth of setae penetration. Cadera et al. developed a widely-used classification system for ophthalmia nodosa. The classification system is based on the location and type of the inflammatory reaction, with increasing type numbers indicating deeper penetration and greater severity:

- Type 1: Acute Anaphylactoid Reaction This is an immediate, toxic reaction to the hairs beginning within a few days of exposure and causing general inflammation and chemosis (swelling of the conjunctiva).
- Type 2: Chronic Mechanical Keratoconjunctivitis Caused by hairs lodged in the bulbar or palpebral conjunctiva, leading to a foreign body sensation and linear corneal abrasions. This condition is often chronic due to missed or retained hairs.
- Type 3: Granulomatous Nodule Formation Characterized by the development of grayish-yellow nodules in the conjunctiva or cornea where the hair is embedded. Patients may initially be asymptomatic.
- Type 4: Iritis (Anterior Uveitis) Occurs when the hair penetrates the anterior segment of the eye, causing severe inflammation of the iris and potential formation of iris nodules or hypopyon
- Type 5: (Vitreoretinal Involvement) The most severe form, where hairs penetrate the posterior segment via the anterior chamber or transsclerally, leading to vitritis, retinal inflammation, macular edema, or endophthalmitis, which can result in irreversible vision loss.

Intraocular penetration of setae represents a critical prognostic factor in disease severity [7,8]. Although exact mechanisms of progressive intraocular migration have not been fully elucidated, mechanical forces from ocular movements, blinking, and iris motility are hypothesized to facilitate migration of setae deeper into ocular tissues.

Secondary IOP elevation can occur in various forms of ocular trauma through multiple mechanisms, including trabecular meshwork inflammation (trabeculitis), angle recession, inflammatory debris obstruction, pupillary block mechanisms and direct toxic effects [7,8]. To our knowledge, acute secondary IOP elevation as the presenting clinical feature of ophthalmia nodosa has not been previously documented in medical literature. Clinical manifestations of ophthalmia nodosa demonstrate considerable heterogeneity, ranging from mild conjunctival hyperemia to severe intraocular inflammation with vision-threatening sequelae [9,10]. The setae, equipped with microscopic barbs and hooks, possess the capacity to penetrate corneal and scleral barriers, facilitating intraocular migration. While anterior segment involvement with conjunctivitis and keratitis represents the most frequently documented presentation, intraocular penetration occurs in a subset of cases (24%), potentially affecting the iris, anterior chamber, lens, vitreous cavity, and posterior segment structures [11,12].

The epidemiological distribution of ophthalmia nodosa demonstrates geographic and seasonal variation, correlating with the life cycles and habitat distribution of processionary caterpillars [13,5]. Exposure typically occurs through direct contact, airborne dissemination of setae during caterpillar defensive behaviors or inadvertent introduction during outdoor activities [14,15]. Recognition of the condition requires clinical suspicion based on exposure history, characteristic inflammatory findings and identification of setae on slit-lamp biomicroscopy [16,17].

We report a unique case of caterpillar hair-induced ophthalmitis in which acute secondary intraocular pressure elevation served as the initial clinical manifestation, creating a diagnostic challenge by temporarily obscuring the underlying pathology until therapeutic intervention allowed visualization of the causative lepidopteran setae. This case underscores the importance of maintaining a broad differential diagnosis in patients presenting with ocular trauma.

Case Presentation

Patient History and Initial Presentation

A 27-year-old male with no significant past medical or ocular history presented to our ophthalmology emergency department with severe acute pain in LE, accompanied by nausea, vomiting, panic attack and ipsilateral headache. The patient reported that approximately five hours prior to presentation, while outdoors in a windy environment, he experienced the sudden entry of a foreign body into his LE. He immediately felt intense pain and attempted irrigation with copious amounts of water at home. Despite thorough irrigation, he experienced persistent severe irritation and a foreign body sensation, which prompted prolonged and vigorous eye rubbing in an attempt to relieve the discomfort. A self-taken photograph captured by the patient shortly after the incident demonstrated semi-mydriasis, marked conjunctival hyperemia and eyelid edema. His symptoms progressively worsened over the subsequent hours, culminating in a severe pain crisis with systemic manifestations that prompted his emergency department visit.

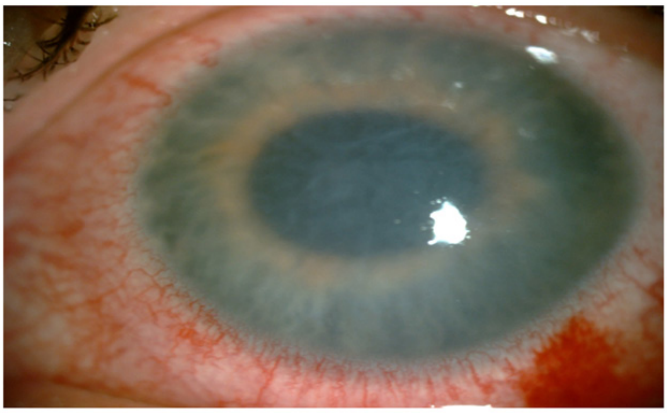

Figure 1: Initial examination. Severe conjunctival hyperemia, diffuse corneal edema with prominent Descemet's membrane folds and mid-dilated pupil.

Figure 2: Enviromental documentation showing multiple caterpillars at the site associated with the reported ocular trauma.

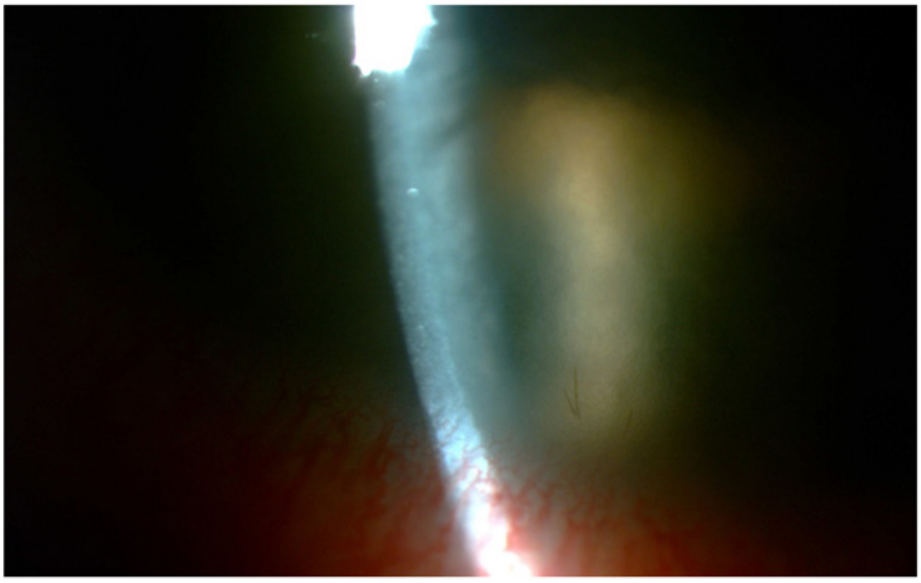

Figure 3: Multiple fine filamentous, hair-like foreign bodies were identified embedded at various depths throughout the corneal stroma.

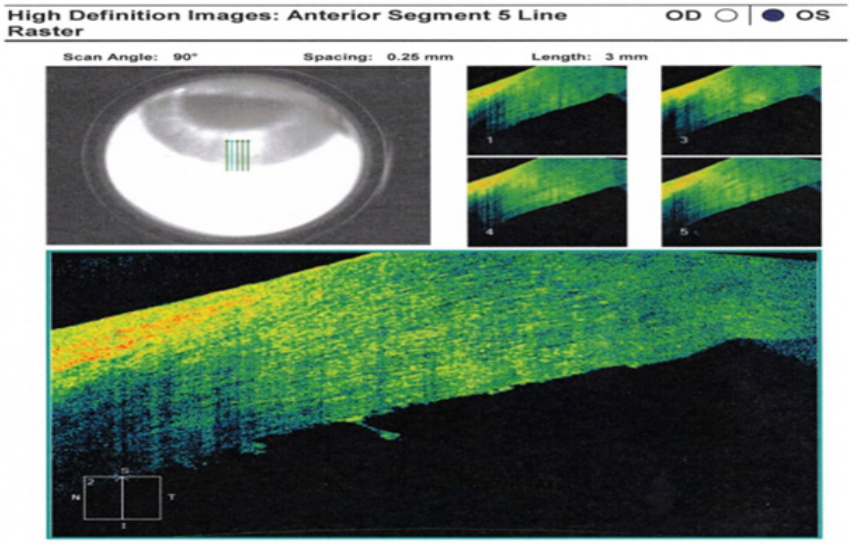

Figure 4: AS-OCT demonstrating the presence of setae in the corneal stroma at multiple depths and documented that some setae protruding into the anterior chamber.

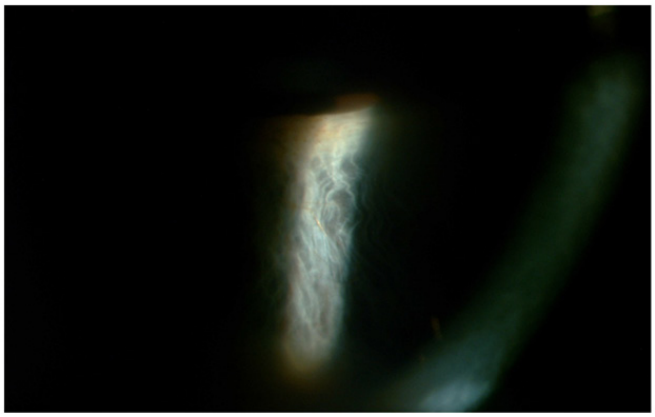

Figure 5: The iris examination demonstrated multiple filamentous foreign bodies visibly embedded in the iris tissue. There was sectoral iris atrophy at the 6 o'clock position corresponding to the location of the main corneal penetration site, with an iris defect noted.

Figure 6: Long-term follow-up. Resolution of the corneal edema with significantly improved clarity. The anterior chamber was quiet with no cells or flare and there had been gradual absorption of the fibrin elements. However, the pupil demonstrated persistent irregular shape measuring 5.4mm by 7mm with ectropion uveae.

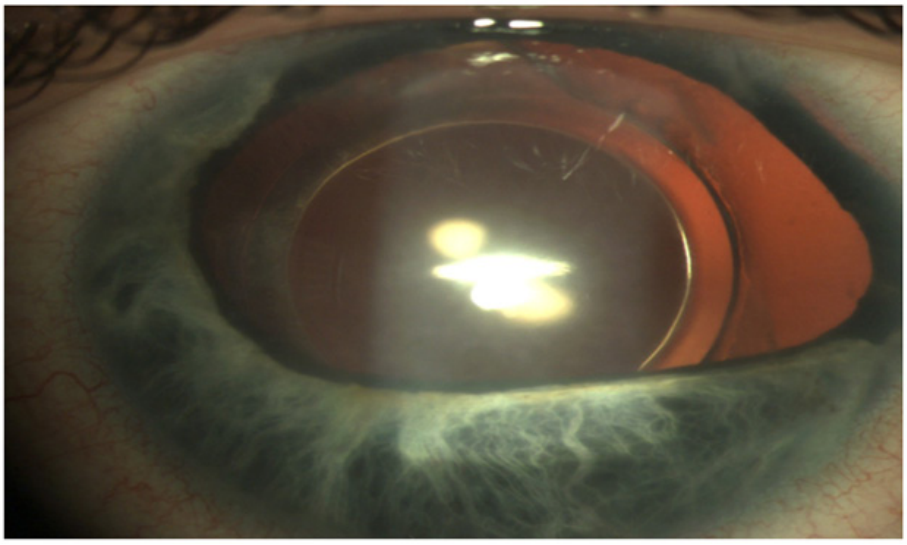

Figure 7: Postoperative findings included with persistent mydriasis due to iridoplegia with ectropion uveae and posterior chamber intraocular lens.

Initial Clinical Examination

On initial examination, VA in the LE was limited to counting fingers at one meter, while the RE maintained 10/10 (equivalent to 20/20) uncorrected vision. External examination revealed marked eyelid edema and severe conjunctival hyperemia affecting the left eye. Slit-lamp biomicroscopy demonstrated diffuse corneal edema with prominent Descemet's membrane folds, which significantly limited visualization of the anterior chamber structures and deeper ocular anatomy. The pupil was in semi-mydriasis with a sluggish and incomplete response to light. Structural details of the iris were obscured by the severe corneal edema. IOP measurement revealed approximately 45 mmHg in the LE and the globe was firm and tender to palpation. The RE demonstrated a normal intraocular pressure. Gonioscopy could only be performed on the RE due to the corneal edema in the affected eye, revealing a Shaffer grade 2 angle throughout 360 degrees. This finding suggested a mild anatomical predisposition to angle closure but provided no evidence of acute angle closure in the fellow eye.

Initial Diagnosis and Emergency Management

Based on the clinical presentation of acute severe pain with systemic manifestations, markedly elevated IOP, significant corneal edema preventing detailed anterior segment examination and the recent history of ocular trauma, the patient was diagnosed with acute secondary intraocular pressure elevation of unclear etiology. At this initial stage, the differential diagnosis included post-traumatic causes (hyphema, angle recession, direct trauma), pharmacologic causes (corticosteroid-induced, etc.), inflammatory-toxic causes (uveitis, toxic reactions, intraocular foreign bodies) and neovascular/obstructive/angle-closure mechanisms.

The patient was immediately admitted to the ophthalmology ward and commenced on intensive medical therapy for urgent intraocular pressure reduction. Topical therapy included timolol 0.5% eye drops (a beta-blocker), brimonidine 0.2% eye drops (an alpha-2 agonist), dorzolamide 2% eye drops (a topical carbonic anhydrase inhibitor) and dexamethasone eye drops administered every two hours to address the presumed inflammatory component. Systemic therapy consisted of acetazolamide 500mg (a systemic carbonic anhydrase inhibitor) and mannitol 20% given as an intravenous infusion (a hyperosmotic agent). Additional sedatives and painkillers were administered.

Laboratory investigations were promptly obtained, including a complete blood count, comprehensive metabolic panel, facial sinus radiography and orbital CT scan. These studies were performed to rule out the presence of a retained metallic intraocular foreign body and to assess for posterior segment involvement.

Subsequent Examination After Intraocular Pressure Reduction

Following intensive IOP-lowering therapy, the patient's symptoms significantly improved with complete resolution of nausea and marked reduction in ocular pain. The panic attack symptoms resolved as the ocular pain subsided. IOP measurements demonstrated a reduction in the LE.

Critically, as the IOP decreased and the corneal edema began to resolve, repeat detailed slit-lamp biomicroscopy performed with high magnification revealed previously obscured pathological findings that fundamentally changed the diagnosis. The corneal examination now demonstrated a diffuse linear corneal epithelial erosion. A Seidel test was performed and proved negative for

active aqueous leak. Most significantly, multiple fine filamentous, hair-like foreign bodies were identified embedded at various depths throughout the corneal stroma. These foreign bodies were specifically located at the 1, 3, 4, 5, 8, 9 and 11 o'clock positions. There was a linear epithelial defect with localized epithelial detachment at the site of the main penetrating wound at the 6:30 position and focal stromal infiltrates surrounded some of the embedded foreign bodies. Examination of the anterior chamber revealed a moderate inflammatory reaction with 2+ cells and flare. Some setae were visible protruding from the posterior corneal surface into the anterior chamber space at the 6:30 position. Fibrin strands were observed in the anterior chamber, and multiple round whitish keratic precipitates were present on the corneal endothelium. The iris examination demonstrated multiple filamentous foreign bodies visibly embedded in the iris tissue. There was sectoral iris atrophy at the 6 o'clock position corresponding to the location of the main corneal penetration site, with an iris defect noted. The iris vessels appeared prominently dilated with vascular engorgement and the pupil shape was irregular with early ectropion uveae formation. The lens examination showed a fine fibrinous membrane visible on the anterior lens capsule surface, but there was no cortical or nuclear lens opacity and no evidence of lens capsule rupture.

Revised Diagnosis

Based on the integrated clinical findings, diagnosis was established. The history of outdoor exposure to wind-borne foreign material in an endemic region for processionary caterpillars (Greece, Mediterranean region), combined with the characteristic appearance of multiple fine filamentous setae at various corneal depths with documented anterior chamber and iris penetration, the presence of a full-thickness corneal wound and the intense acute inflammatory response, all pointed to a diagnosis of ophthalmia nodosa with acute intraocular pressure elevation. According to the Cadera classification system, this case represented Type IV ophthalmia nodosa, given the involvement of multiple anterior segment structures including the cornea, anterior chamber, and iris.

Pathophysiologic Mechanism

The underlying mechanism for IOP elevation was determined to be inflammation and toxicity from the presence of setae at different depths and locations within the cornea and the anterior chamber. The inflammatory-toxic process affects aqueous humor dynamics through obstruction and functional impairment of the trabecular meshwork, immunologic response due to toxicity from thaumetopoein release and potential anatomic changes with development of synechiae or pupillary block in the long term [7,10,17].

Enhanced Therapeutic Management

Upon confirmation of the diagnosis of ophthalmia nodosa with penetrating injury, the treatment regimen was substantially modified and enhanced to address both the intense inflammatory response to the toxic setae and the significant risk of secondary microbial infection associated with penetrating organic foreign bodies.

Enhanced topical therapy included:

- Fortified vancomycin eye drops administered every hour for gram-positive bacterial coverage
- Fortified ceftazidime eye drops administered every hour for gram-negative and Pseudomonas

Coverage

- Levofloxacin eye drops administered four times daily as a broad-spectrum fluoroquinolone
- Voriconazole eye drops administered four times daily for broad-spectrum antifungal coverage
- Dexamethasone eye drops continued every two hours for intensive anti-inflammatory therapy

Topical IOP-loweing medications continued with plans for gradual tapering.

Subconjunctival injection: Amikacin combined with dexamethasone to achieve high local tissue concentrations of both antimicrobial and anti-inflammatory agents.

Systemic therapy included:

- Vancomycin 1g administered intravenously twice daily
- Ceftazidime 2g administered intravenously three times daily
- Voriconazole with a 400mg intravenous loading dose on day one followed by 200mg administered intravenously twice daily
- Methylprednisolone 1mg/kg administered intravenously daily

Attempted surgical removal of the superficial corneal setae was carefully considered but ultimately deemed inappropriate due to several significant factors. Caterpillar setae are extremely fragile structures that fragment easily during manipulation, and such fragmentation could lead to deeper migration of the fragmented pieces, release of additional toxic material from ruptured setae, and exacerbation of the inflammatory response [16,18]. Furthermore, given the multiplicity of locations and the varying depths of embedment throughout the cornea and over the iris surface, surgical removal of all setae was not technically feasible. Therefore, conservative management with intensive medical therapy and close clinical monitoring was elected as the safest approach, with the understanding that some setae might spontaneously migrate or become encapsulated over time [15,18].

Hospital Course

Over days, inflammation subsided, IOP normalized with topical therapy alone, and no posterior migration of setae was documented. Some changes in setae orientation were noted on the iris surface. The patient was discharged with a strict topical regimen and close follow-up.

Long-term Follow-up and Outcome

At the two-month follow-up examination, BCVA in the LE had improved to 3/10 (approximately 6/20). The RE maintained

10/10 BCVA. Intraocular pressure in the LE was 10 mmHg on topical anti-glaucoma monotherapy with timolol eye drops, while the RE measured 9 mmHg. Slit-lamp biomicroscopy demonstrated partial resolution of the corneal edema with significantly improved clarity. Multiple setae were still visible at various stromal depths, but there was no evidence of progressive anterior or posterior migration when compared to earlier examinations. The anterior chamber was quiet with no cells or flare and there had been gradual absorption of the fibrin elements. However, the pupil demonstrated persistent irregular shape measuring 5.4mm by 7mm in mydriasis with ectropion uveae and poor reactivity to light stimulation. The filamentous foreign bodies remained embedded in the iris tissue and the sectoral iris atrophy persisted at the 6 o'clock position. The lens showed near-complete absorption of the perilenticular fibrin membrane with no cataract formation evident at that time. Fundoscopic examination revealed a normal optic disc appearance, a healthy macula without evidence of edema and normal peripheral retina in both eyes. B-scan ultrasonography demonstrated no vitreous opacities and no retained foreign bodies were detected in the posterior segment. AS-OCT confirmed the presence of setae in the corneal stroma at multiple depths and documented that some setae showed minimal displacement compared to earlier imaging.

At 1.5 years, the patient developed cataract requiring phacoemulsification. Postoperative findings included:

- BCVA = 6/10
- Persistent photophobia accompanied by dysphotopic visual phenomena, requiring constant use of sunglasses
- Persistent mydriasis with iridoplegia and ectropion uveae
- Ongoing low-grade inflammation requiring chronic corticosteroids
- Stable IOP at 10 mmHg both eyeys on dorzolamide–timolol eye drops

The patient continues long-term monitoring due to the risk of delayed inflammatory complications, including development of chronic uveitis, progression of secondary glaucoma that might require additional medications and the possibility of posterior segment involvement from setae migration. Long-term indefinite surveillance has been recommended given the unpredictable nature of retained organic foreign bodies and the documented cases in the literature of delayed complications occurring months to years after the initial injury.

Discussion

This case report documents an uncommon presentation of ophthalmia nodosa characterized by acute intraocular pressure elevation as the initial clinical manifestation, a presentation pattern that has received limited attention in the existing literature [7,8]. While the association between intraocular caterpillar setae and secondary glaucoma has been established, the occurrence of acute IOP elevation prior to the development of anterior segment inflammation represents an atypical clinical trajectory that warrants detailed consideration.

Literature Review of Reported Cases

A comprehensive review of published case reports reveals that ophthalmia nodosa typically presents with anterior segment inflammation as the predominant initial manifestation, with secondary complications developing subsequently [4,9]. Conrath et al. reported acute anterior uveitis as the presenting feature, with intraocular pressure elevation developing as a secondary complication [4]. Similarly, Joshi described a case where multiple intraocular setae were identified in the context of established anterior uveitis, with glaucomatous changes occurring chronologically after the inflammatory presentation [19]. Sahay et al. reported ongoing ocular inflammation as the primary concern, with missed caterpillar cilia identified retrospectively as the causative agent [15].

Recent case series have further characterized the typical clinical course. Agarwal et al. documented multiple caterpillar hairs with conjunctivitis and anterior chamber reaction as initial findings [20]. Ashkenazy et al. utilized multimodal imaging to diagnose ophthalmia nodosa presenting with conjunctival nodules and corneal infiltrates [20]. Levy et al. reported a pediatric case with conjunctival injection and photophobia [5]. Liu and Jiang described typical presentation with conjunctival hyperemia and foreign body sensation as chief complaints [6]. Geographic case series provide additional context. Das et al. analyzing big data from a multitier eye care network, identified conjunctivitis (78.3%), keratitis (43.2%), and anterior uveitis (31.7%) as the most common presenting features, with secondary glaucoma occurring in only 8.4% of cases and invariably following established inflammation [9]. Notably, among all reviewed case reports spanning over a century of literature, none specifically documented acute intraocular pressure elevation as the initial presenting feature. This literature review thereby establishes the unique nature of the present case, representing a previously undocumented presentation pattern of ophthalmia nodosa.

Novel Presentation Pattern

The novelty of this presentation lies not simply in the occurrence of elevated intraocular pressure in ophthalmia nodosa, which has been reported as a complication in some cases, but rather in the temporal sequence and diagnostic challenge it created. The acute pressure elevation and resultant severe corneal edema prevented visualization of the pathognomonic findings at initial examination, creating a clinical paradox in which the consequence of the disease process itself obscured the diagnostic clues necessary to identify its cause. This presentation pattern has important implications for clinical practice and highlights the necessity of maintaining a broad differential diagnosis and performing sequential examinations in cases of acute traumatic ocular hypertension.

Pathophysiological Mechanisms of IOP Elevation [8,17]

The pathogenesis of elevated intraocular pressure in ophthalmia nodosa is multifaceted, involving both mechanical and inflammatory components. The acute intraocular pressure elevation observed in this case likely resulted from

multiple concurrent pathophysiologic mechanisms operating simultaneously. The first and likely most significant mechanism was trabeculitis with trabecular meshwork dysfunction. The toxic protein thaumetopoein contained in processionary caterpillar setae induces acute inflammatory responses in ocular tissues upon contact. When setae penetrated into the anterior chamber, thaumetopoein was released directly into the aqueous humor and came into contact with the trabecular meshwork. Direct inflammatory involvement of the trabecular meshwork (trabeculitis) can acutely impair aqueous outflow facility and lead to rapid intraocular pressure elevation. A second contributing mechanism was inflammatory debris obstruction of the trabecular meshwork. The intense anterior chamber inflammatory reaction, characterized by numerous cells, significant flare, fibrin elements and inflammatory proteins, created substantial particulate matter that could physically obstruct the trabecular meshwork pores and further compromise aqueous drainage. The inflammatory response in ophthalmia nodosa exhibits characteristics of both acute anterior uveitis and chronic granulomatous inflammation. Prostaglandin release, cytokine upregulation and breakdown of the blood-aqueous barrier contribute to trabeculitis and reduced trabecular meshwork permeability. In severe cases, formation of posterior synechiae may precipitate pupillary block mechanisms, further elevating intraocular pressure through angle closure pathophysiology. The aqueous humor dynamics may be further compromised by disruption of the ciliary body function secondary to inflammatory mediators and direct setae penetration.

Diagnostic Considerations and Challenges

The diagnosis of ophthalmia nodosa in cases presenting with acute IOP elevation requires high clinical suspicion and systematic anterior segment examination. The primary diagnostic challenge in this case stemmed from the severe corneal edema that developed secondary to the acute intraocular pressure elevation. Corneal edema prevented adequate visualization of the underlying pathology during the initial examination and created a significant diagnostic dilemma. This situation represents a clinical paradox: the very consequence of the disease process (elevated pressure causing corneal edema) served to obscure the diagnostic findings of embedded setae that were necessary to identify the cause of that elevated pressure. Only after successful therapeutic reduction of the intraocular pressure, with resultant corneal clearing, could detailed high-magnification slit-lamp examination reveal the penetrating setae. This case highlights several important diagnostic principles relevant to acute ocular trauma management. First is the importance of sequential examination after therapeutic intervention. In cases of acute ocular trauma presenting with elevated intraocular pressure and significant corneal edema that substantially limits the initial examination, repeated detailed examination after clinical stabilization may reveal critical diagnostic findings. Second is the importance of obtaining a detailed and specific history regarding environmental factors, proximity to vegetation, presence of windy conditions and time of year. Third is maintaining high clinical suspicion in endemic regions during appropriate seasons.

Slit-lamp biomicroscopy with high magnification remains the cornerstone of diagnosis, enabling visualization of fine setae embedded within corneal stroma, penetrating the iris surface or floating within the anterior chamber. Multimodal imaging techniques including anterior segment optical coherence tomography may augment diagnostic capabilities by enhancing visualization of intraocular setae and characterizing associated structural alterations.

Management Strategies and Therapeutic Considerations [7]

The management of ophthalmia nodosa with secondary IOP elevation requires a comprehensive approach addressing both the inflammatory response and the mechanical presence of intraocular foreign material. Initial medical management typically involves topical corticosteroids to suppress inflammation, complemented by aqueous suppressants including beta-blockers, alpha-agonists or carbonic anhydrase inhibitors to control intraocular pressure. The intensity and duration of anti-inflammatory therapy must be individualized based on the severity of inflammatory response. Given the organic nature of caterpillar setae and their invariable contamination with environmental microorganisms, it would be prudent to provide broad-spectrum prophylactic antimicrobial coverage. The regimen in this case provided comprehensive coverage for gram-positive organisms (vancomycin), gram-negative organisms including Pseudomonas(ceftazidime), and fungi (voriconazole).

Attempted surgical removal of deeply embedded caterpillar setae carries substantial risks that in most cases outweigh the potential benefits. Caterpillar setae are extremely fragile structures that fragment readily during any surgical manipulation. Fragmentation leads to release of additional toxic material from ruptured setae, potentially exacerbating the inflammatory response and creates multiple smaller fragments that may migrate more easily through tissues. In this case, given the multiple locations, varying depths of embedment and fragile nature of the setae, conservative management with intensive medical therapy and close monitoring was deemed the safest approach [21].

The concept of setae migration time has important therapeutic implications. Studies documenting the temporal progression of setae movement through ocular tissues suggest that early intervention may prevent deeper penetration and reduce long-term complications. However, the optimal timing for surgical intervention remains debated.

Classification and Prognostic Implications

According to the Cadera classification system, this case represents Type IV ophthalmia nodosa, characterized by involvement of multiple anterior segment structures with documented penetration into the cornea, anterior chamber, and iris. This classification carries significant prognostic implications. Major concerns include risk of progressive posterior migration of retained setae potentially leading to lens involvement

with cataract formation, vitreous extension, or chorioretinal involvement. Retained setae serve as a persistent nidus for chronic granulomatous inflammation that can continue for months or years, leading to chronic anterior uveitis, cystoid macular edema, posterior synechiae formation, peripheral anterior synechiae with angle closure and glaucomatous optic neuropathy. Structural complications represent permanent damage to ocular tissues. The irregular pupil with ectropion uveae observed in this patient at two-month follow-up reflects permanent structural damage to the iris sphincter muscle and underlying iris stromal architecture unlikely to improve over time. Additional potential structural complications include progressive iris atrophy, cataract formation (particularly posterior subcapsular cataracts due to chronic inflammation and corticosteroid therapy), corneal decompensation at sites of deep stromal setae embedment and in severe cases, phthisis bulbi.

Clinical Implications and Climate Change Considerations

This case emphasizes several clinically relevant considerations. First, acute IOP elevation should prompt comprehensive evaluation including detailed exposure history and meticulous biomicroscopic examination, particularly in endemic regions. Second, the diagnosis should be entertained even without recalled exposure, as indirect transmission mechanisms may occur. Third, long-term follow-up is essential given the potential for delayed complications. The increasing frequency of ophthalmia nodosa cases reported in recent years has been directly linked to climate change effects on lepidopteran populations. Global warming has produced several significant alterations in caterpillar ecology directly impacting human exposure risk. Rising average temperatures have progressively shifted the processionary caterpillar life cycle, with larvae now emerging in late winter (January-February) rather than spring (March-April), coinciding with periods when the public is less aware of caterpillar risks. Warming temperatures have enabled caterpillars to successfully colonize higher altitudes and more northern latitudes previously too cold to support breeding populations. Extended warm periods have lengthened the exposure window from a few weeks to several months in some regions. Warmer winters with reduced mortality have led to higher caterpillar population densities in endemic areas. These epidemiological shifts necessitate increased awareness among ophthalmologists, emergency medicine physicians and primary care providers. Healthcare professionals must maintain heightened vigilance throughout an extended season from late winter through late spring. Traditional thinking limiting concern to a brief spring season is no longer adequate.

Public Health and Prevention Strategies

Prevention of ophthalmia nodosa requires community education regarding caterpillar exposure risks, particularly during peak activity periods. Protective measures include avoidance of areas with known processionary caterpillar infestations, use of protective eyewear during outdoor activities and prompt irrigation following suspected exposure. Target populations for education include parents and caregivers of young children, educational institutions near pine forests, outdoor recreation enthusiasts and agricultural and forestry workers facing occupational exposure.

Study Limitations

This case report has inherent limitations. The exact caterpillar species was not definitively confirmed through formal entomological examination, as no setae were successfully removed. The diagnosis of Thaumetopoea pityocampa was based on clinical presentation, geographic location (endemic Greece), seasonal timing and characteristic setae morphology. As a single case report, this cannot establish generalizability or definitive management guidelines. Future research incorporating larger case series and prospective studies may elucidate the relative frequency of this presentation pattern and identify predictive factors. Investigation into specific pathophysiological mechanisms underlying early IOP elevation, including histopathological analysis and aqueous humor cytokine profiling, may inform targeted therapeutic strategies.

Conclusion

Caterpillar hair-induced ophthalmitis represents a complex clinical entity requiring a high index of suspicion, immediate intervention, and individualized therapeutic approach. Understanding the toxicity mechanisms, clinical manifestations and therapeutic options is essential for the effective management of these cases. Such cases should be recognized from the outset as extremely serious conditions, with emphasis on long-term prognosis, especially when setae remain in ocular tissues.Public awareness is also essential, with particular emphasis on children's environments and young people, including parents and educational institutions.

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None.

Conflict of interest

None.

Conferences

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