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Vernal keratoconjunctivitis and corticosteroid therapy: The silent risk of steroid-induced glaucoma

Salma Bennis *, A. Jeribi, Y. Akannour, F. El Ibrahimi, L. Serghini and E. Abdallah

Department of Ophthalmology B, Specialties Hospital, CHU Ibn Sina, Mohamed V University, Rabat, Morocco.

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Abstract

Background: Vernal keratoconjunctivitis (VKC) is a chronic inflammatory disease commonly treated with topical corticosteroids. However, prolonged or unsupervised use exposes patients to steroid-induced glaucoma (SIG), a potentially irreversible condition.

Objective: To highlight the risk of steroid-induced glaucoma in VKC patients and identify contributing factors based on a case series.

Methods: Retrospective study of 21 VKC patients who developed steroid-induced glaucoma following prolonged corticosteroid use.

Results: All patients had a history of prolonged corticosteroid use without adequate monitoring. Mean duration was 8 months. Mean IOP was 32 mmHg, with 75% exceeding 30 mmHg. Advanced optic nerve damage was observed in more than half of cases. Strong correlation was found between duration of exposure and IOP elevation.

Conclusion: Steroid-induced glaucoma in VKC is a silent but preventable complication. Systematic monitoring and rational use of corticosteroids are essential to preserve visual function.

Keywords: Vernal keratoconjunctivitis; Corticosteroids; Steroid-induced glaucoma; Pediatric glaucoma; Ocular hypertension

1. Introduction

Vernal keratoconjunctivitis (VKC) is a chronic, severe allergic ocular disease affecting mainly children and young adults, particularly in warm and sunny climates. It is characterized by recurrent inflammatory episodes that significantly impact quality of life and may lead to corneal complications.

Topical corticosteroids remain the cornerstone of treatment for acute exacerbations due to their rapid anti-inflammatory effect. However, their prolonged or inappropriate use exposes patients to serious complications, including steroid-induced glaucoma (SIG).

SIG is characterized by progressive elevation of intraocular pressure due to increased trabecular resistance. Its insidious onset and absence of early symptoms often lead to delayed diagnosis, especially in pediatric populations.

* Corresponding author: Salma Bennis

This study aims to highlight the silent risk of steroid-induced glaucoma in VKC patients and to emphasize the need for early detection and preventive strategies.

2. Materials and methods

2.1. Study Design

Retrospective descriptive study conducted between 2021 and 2024.

2.2. Population

21 patients with VKC complicated by steroid-induced glaucoma.

2.3. Inclusion Criteria

- Clinically diagnosed VKC
- Corticosteroid use ≥ 2 months
- IOP > 21 mmHg
- Evidence of glaucomatous damage

2.4. Data Collected

- Demographic data
- VKC clinical form
- Type and duration of corticosteroid use
- Mode of use (supervised vs self-medication)
- IOP and optic nerve findings
- Therapeutic management

3. Results

Table 1 VKC Clinical Forms

Form	Percentage
Palpebral	46%
Limbal	15%
Mixed	38%

Table 2 Corticosteroid Exposure

Parameter	Value
Self-medication	100%
Mean duration	8 months (3–24)
Most used steroid	Dexamethasone

Table 3 Relationship Between Duration and IOP

Duration	IOP Trend
< 6 months	Moderate increase
> 6 months	Marked increase
Correlation	r = 0.75

Table 4 IOP According to Steroid Type

Steroid	Mean IOP (mmHg)
Dexamethasone	34.2
Prednisolone	30.5
Fluorometholone	26.7
Loteprednol	23

Table 5 Glaucoma Severity

Parameter	Value
Mean IOP	32 mmHg
IOP > 30 mmHg	75%
C/D > 0.7	53.8%
Visual field defects	60%

4. Discussion

This study highlights steroid-induced glaucoma as a major yet preventable complication of VKC, particularly in young patients exposed to prolonged corticosteroid therapy.

4.1. VKC: A High-Risk Context

VKC predominantly affects children living in hot climates, with frequent recurrences requiring repeated corticosteroid use.

The chronic nature of the disease promotes:

- prolonged exposure
- repeated self-medication
- poor therapeutic adherence
- making VKC patients particularly vulnerable to SIG.

Which makes VKC patients particularly vulnerable to steroid-induced glaucoma.

4.2. Corticosteroids: A Double-Edged Sword

While corticosteroids are highly effective, their prolonged use leads to increased trabecular resistance.

Our findings confirm:

- higher IOP with potent steroids (dexamethasone)
- dose- and duration-dependent effect

These results are consistent with previously published studies demonstrating the relationship between corticosteroid potency and intraocular pressure elevation (Kersey and Broadway, 2006; Jones and Rhee, 2006).

4.3. Silent and Delayed Diagnosis

The absence of symptoms explains why:

- 58% of cases were diagnosed incidentally
- many patients already had advanced damage

Additionally:

- VKC symptoms mask glaucoma progression
- corneal changes alter IOP measurement

4.4. Pediatric Specificity

Children show:

- stronger steroid response
- increased susceptibility to optic nerve damage

This is explained by:

- immature trabecular meshwork
- increased biological sensitivity

4.5. Therapeutic Challenges

Management must balance:

- control of inflammation
- control of IOP

Strategies include:

- steroid withdrawal or substitution
- use of immunomodulators (cyclosporine, tacrolimus)
- hypotensive treatment

4.6. Prevention: The Key Message

This study emphasizes that SIG is largely preventable.

Key measures:

- systematic IOP monitoring
- patient and family education
- limiting corticosteroid use
- promoting steroid-sparing therapies

5. Conclusion

- Steroid-induced glaucoma in VKC is a silent but severe complication affecting young patients.
- It results mainly from prolonged, unsupervised corticosteroid use and often leads to late diagnosis with significant visual damage.
- Preventive strategies and systematic monitoring are essential to reduce this avoidable cause of blindness.

Compliance with ethical standards

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Disclosure of Conflict of Interest

The authors declare no conflict of interest.

Statement of Ethical Approval

This study was conducted in accordance with the principles of the Declaration of Helsinki.

Statement of Informed Consent

Informed consent was obtained from all individual participants included in the study.

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