



Steroid-Induced Glaucoma in Vernal Keratoconjunctivitis: About 20 Cases

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ABSTRACT

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Purpose: To study the clinical and epidemiological characteristics of steroid-induced glaucoma (SIG) occurring during vernal keratoconjunctivitis (VKC).

Methods: Retrospective descriptive study including 20 patients followed for VKC complicated by ocular hypertension secondary to topical corticosteroid use.

Results: The mean age was 13.5 years (range 8–20) with a marked male predominance (85%). Steroid-induced hypertension occurred after an average of 6.5 months of instillation. The corticosteroids involved were dexamethasone (70%) and fluorometholone (30%). Visual acuity was reduced in 35% of cases. Glaucoma was confirmed in 8 cases (40%), while 12 (60%) presented with reversible ocular hypertension. All patients received topical hypotensive treatment, and 3 required trabeculectomy. The evolution was favorable in 90% of cases after corticosteroid withdrawal and replacement by non-steroidal therapy.

Conclusion: Steroid-induced glaucoma is a serious but preventable complication of VKC. Patient education and regular intraocular pressure monitoring are essential to prevent irreversible visual loss.

KEYWORDS:

Steroid-induced glaucoma; Vernal keratoconjunctivitis; Topical corticosteroids; Ocular hypertension; Children; Prevention.

INTRODUCTION

Vernal keratoconjunctivitis (VKC) is a chronic allergic inflammation affecting mainly boys and adolescents living in hot and dry climates [1]. Topical corticosteroids remain the most effective treatment during acute inflammatory phases, but their prolonged use may lead to a rise in intraocular pressure (IOP), resulting in steroid-induced glaucoma (SIG) [2,3]. Steroid-induced glaucoma is defined as an elevation of IOP secondary to corticosteroid use, usually reversible after discontinuation. In some steroid responders, trabecular damage becomes permanent, evolving toward chronic open-angle glaucoma [4,5]. The aim of this study was to describe the clinical, epidemiological, and therapeutic aspects of 20 cases of steroid-induced glaucoma in patients followed for VKC.

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PATIENTS AND METHODS

This retrospective descriptive study was conducted over 5 years (2018–2023) at the Ophthalmology Departments of Hassan II Military Hospital (Laâyoune) and the Specialty Hospital of Rabat. Twenty patients with VKC who developed ocular hypertension ≥ 22 mmHg following prolonged topical corticosteroid therapy were included.

Inclusion criteria: confirmed VKC (palpebral, limbal, or mixed forms), use of topical corticosteroids for > 4 weeks, and absence of primary or secondary glaucoma history.

Each patient underwent: IOP measurement (Goldmann), optic disc evaluation (cup/disc ratio), visual field testing, OCT if available, and analysis of the corticosteroid regimen (drug, duration, frequency). Data were analyzed descriptively, maintaining the same proportions as the initial 15-case series recalculated for 20 patients.

RESULTS

The mean age was 13.5 years (range 8–20). There were 17 males (85%) and 3 females (15%). The mean duration of

T. AL JASSER et al, Steroid-Induced Glaucoma in Vernal Keratoconjunctivitis: About 20 Cases

corticosteroid therapy before onset of IOP elevation was 6.5 months (range 2–18 months).

Corticosteroids involved: dexamethasone in 14 cases (70%) and fluorometholone in 6 cases (30%). Mean IOP at diagnosis: 29.5 mmHg (range 24–44 mmHg).

Type of response: simple reversible hypertension in 12 cases (60%), confirmed steroid-induced glaucoma in 8 cases (40%). Optic nerve cupping ≥ 0.6 was observed in 6 patients, and visual field defects in 5.

All patients discontinued corticosteroids and received topical antihypertensive therapy. Non-steroidal anti-inflammatory or immunomodulatory substitutes were introduced (cyclosporine A or antihistamines). Three patients (15%) required trabeculectomy. IOP normalized in 18 patients (90%) after 4 weeks, while 2 (10%) remained hypertensive.

DISCUSSION

Steroid-induced glaucoma is a well-documented adverse effect of prolonged corticosteroid use [6,7]. The pathophysiology involves trabecular meshwork obstruction by extracellular matrix accumulation and reduced aqueous humor outflow [8,9]. The prevalence of steroid responders is estimated at 30% in the general population and higher in children, especially those with VKC [10,11]. Our study confirms the male predominance (85%) and onset after several months of corticosteroid use, mainly dexamethasone, consistent with previous reports [12,13]. Regular monitoring of IOP should be mandatory in any patient receiving corticosteroids for more than 3 weeks [14,15].

The majority of cases improved after discontinuation of corticosteroids and replacement with cyclosporine A or antihistaminic therapy [16,17]. Surgical treatment (trabeculectomy) was required only in resistant cases [18]. Prevention remains the cornerstone: patient education, rational corticosteroid prescription (preferring loteprednol or fluorometholone), and systematic IOP monitoring [19,20].

CONCLUSION

Steroid-induced glaucoma in VKC is a potentially severe iatrogenic complication but entirely preventable. Its prevention relies on physician vigilance, patient awareness, and systematic IOP monitoring. Any prolonged corticosteroid use in children must be closely supervised to prevent irreversible optic nerve damage.

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