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A 10-YEAR FOLLOW-UP OF CHILDHOOD-ONSET RECURRENT UNILATERAL IDIOPATHIC ANTERIOR UVEITIS: A CASE REPORT

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ABSTRACT

Idiopathic anterior uveitis accounts for a significant portion of uveitis and is responsible for localized intraocular inflammation. The case presents a long-term follow-up of a 38-year-old patient with a unilateral, recurrent and childhood-onset IAU. The patient encounters flare-ups approximately every six months, which are treated with topical corticosteroids and mydriatics, leading to quick resolution without lasting complications or visual acuity decline. Comprehensive diagnostic work-up ruled out infectious and autoimmune causes, highlighting the disease's purely ocular involvement. Examination of local immune reactions in the anterior chamber of eyes affected by IAU is crucial, as together with immune memory may persist even after clinical recovery. These mechanisms account for the persistent nature of inflammation and the drawbacks of existing topical treatments, which manage acute flare-ups but fail to address the fundamental immune dysregulation. New targeted strategies, such as locally delivered biologics or cytokine-modulating agents, could reduce recurrence while limiting systemic exposure.

This case highlights the significance of localized ocular immune activation and underscores the necessity for treatments aimed at the root immunopathogenesis of IAU.

KEYWORDS

Cytokines, Idiopathic Anterior Uveitis, Local Immune Mechanisms, Recurrent Uveitis, Targeted Therapy

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Introduction

Uveitis is defined as an inflammatory condition that arises within the uveal tract. Such inflammation may either be limited to the eye or arise as part of broader systemic disease [1].

Anterior uveitis is the most prevalent type of uveitis within the anatomical classification, constituting nearly two-thirds of all cases [2]. It is marked by inflammatory engagement of the iris and ciliary body [3]. Recurrent uveitis features numerous intraocular inflammatory flare-ups, interspersed with periods of inactive disease that do not necessitate treatment [4].

Idiopathic forms remain the largest etiological group of uveitis, responsible for about 25% to 77% of documented cases [5]. Other most commonly seen etiologies include HLA-B27–associated anterior uveitis, toxoplasmic retinochoroiditis, as well as cytomegalovirus retinopathy [6]. The primary clinical manifestations reported by patients are abrupt unilateral pain accompanied by photophobia. Additionally, ocular redness and miosis can be evident. While isolated occurrences can often be managed without additional diagnostic assessment, the recurrent episodes necessitate an extensive evaluation [1].

Topical corticosteroids (TCS) being employed as primary treatment for idiopathic anterior uveitis (IAU) rarely achieve sustained remission, showing that more focused immunomodulatory approaches are needed [7].

We present a long term follow-up of a patient with recurrent IAU, providing insight into its pathophysiology and emphasizing the need for more targeted therapeutic options.

Case Presentation

Patient history

A 38-year-old patient has been monitored for a decade in an ophthalmology clinic because of repeated acute-onset, solely unilateral anterior uveitis affecting the right eye since childhood. The inflammatory events happen roughly every six months and are associated with eye pain, redness, and photophobia, consistent with clinical presentation of anterior uveitis. On initial presentation the patient stated that the first episode of uveitis occurred during childhood. Over the last decade, the illness has exhibited a consistent pattern of relapsing, without affecting the left eye. The patient provided past medical records, which included a comprehensive systemic assessment due to the persistent nature of inflammation. This evaluation excluded infectious and systemic causes. Diagnostics included, among others, negative Quantiferon TB Gold, toxoplasmosis serology, HLA-B27, ANA, RF, ESR and CRP. The medical history is otherwise insignificant, and no chronic systemic diseases have been identified.

Physical Examination and Imaging

On physical examination, during the 10-year observation of flare-ups the best corrected visual acuity has reliably stayed at 20/20 in each eye. Intraocular pressure has consistently measured at average 15 mmHg in both eyes. Such values were also observed during remission phases. Slit-lamp examination of the right eye in flare-ups showed conjunctival hyperemia and mild to moderate anterior chamber inflammation, with visible 1-15 AC cells, graded as 0.5+-1+, according to the Standardization of Uveitis Nomenclature (SUN) classification, with no to faint flare as described in SUN. Keratic precipitates or hypopyon were not seen. What is important, no posterior synechiae occurred throughout the entire follow-up period. Fundus examination in each eye revealed a pale pink optic disc with minor cup to disc ratio, normal retinal blood vessels, attached retina and no signs of macular edema. Optical coherence tomography of the optic nerve head was performed with the following results of retinal nerve fiber layer (RNFL)- thickness of 108 µm in the right eye and 100 µm in the left eye [Figure 1].

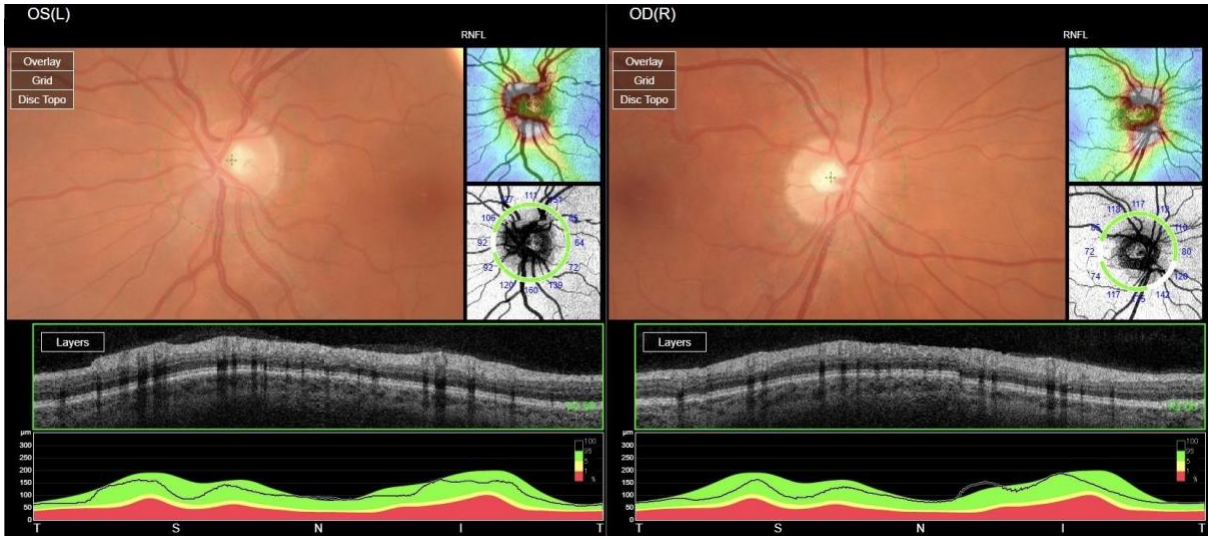

Fig. 1. *The figure shows OCT imaging of the optic nerve with RNFL analysis of the left (left image) and right eye (right image) at the patient's last follow-up appointment.*

Treatment

Every flare-up quickly improved with the application of topical dexamethasone 0.1% given three times a day, as well as tropicamide prescribed twice a day. In more severe cases, brief treatments with systemic methylprednisolone were initiated. With stepwise tapering regimen implantation until complete resolution of uveitis. Significantly, all flare-ups fully resolved after 1-2 weeks, without the need for hospitalization and did not result in any lasting structural damage. The length of episodes stayed similar over the 10-year long observation, showing no signs of disease progression [Table 1].

Table 1. The table summarizes the clinical characteristics of our patient during the 10-year follow-up period, including flare frequency, severity (SUN classification), treatment, and visual outcome.

Parameter	Value
Mean number of flare-ups per year	2
Mean duration of flare-ups	10 days
Mean anterior chamber cells (SUN)	1+
Mean flare (SUN)	Minimal
Systemic disease	None detected
Treatment required	Topical corticosteroids and mydriatics
Systemic immunosuppression	Single short course
Complications	None observed
Final BCVA	20/20

Follow-up

During a decade of ongoing monitoring, uveitis in our patient has shown an isolated unilateral course with maintained visual acuity at 20/20 and no long-term complications were observed [Figure 2].

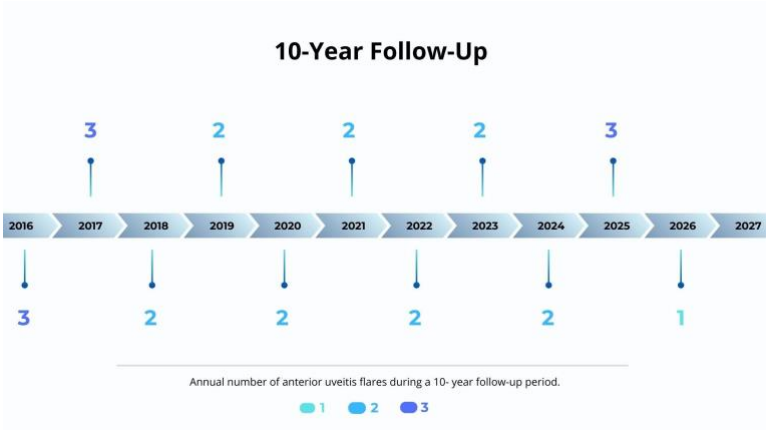

Fig. 2. *The graphic presents the annual number of anterior uveitis flare-ups in our patient throughout the 10-year follow-up period, illustrating a recurrent nature of the disease.*

Discussion

Despite the fact that broad clinical and scientific knowledge regarding uveitis is known, the precise pathophysiology of IAU is still being analyzed by scientists [8]. Grasping these mechanisms is vital for directing suitable treatment approaches, especially in patients who lack systemic disease.

The recurrent and unilateral character of episodes in this patient suggests an immune reaction localized to the impacted eye. Even though systemic forms could also present with unilateral form, it has been observed to be more likely to present with bilateral involvement. [9]

In cases of recurrent uveitis, special attention should be paid to excluding systemic causes. Anterior uveitis may be associated with a wide variety of diseases, including spondyloarthropathies, sarcoidosis, as well as infectious ones. [10] Although our patient did not present any extraocular complaints, in order for comprehensive management all necessary diagnostics were done [11]. HLAb27 testing, together with Quantiferon TB Gold, toxoplasmosis serology and many others were investigated, and no underlying systemic disease has been identified. In each flare the patient has been treated with TCS and tropicamide, to prevent

formation of posterior synechiae [12]. The patient responds well to this treatment. The long history of disease in the patient could suggest the presence of complications related to both recurrent inflammation and steroid treatment. To date, the patient did not develop any complications of anterior uveitis, among which there are posterior synechiae, cataract formation, secondary glaucoma and cystoid macular edema [13].

TCS used in this patient act locally by suppressing intraocular inflammation through inhibition of proinflammatory cytokine production, while mydriatics act on iris sphincter and ciliary muscle, as previously stated preventing formation of posterior synechiae [14]. They primarily exert a transient anti-inflammatory effect and do not eliminate the underlying immune dysregulation responsible for disease recurrence. [15, 16, 17].

In IAU, autoreactive T cells and local immune memory mechanisms may persist despite clinical resolution, predisposing the patient to subsequent flares. [18]. Therefore, topical therapy controls the manifestations of inflammation but does not modify the fundamental immunopathogenic processes driving recurrent disease. Although TCS are effective in our patient, they do not address the underlying immune dysregulation responsible for disease recurrence. What is more, their repeated use is associated with side effects like cataract and increased intraocular pressure [15]. In contrast, systemic immunosuppression has an effect on the systemic immunity, which is reserved for bilateral, severe and most importantly systemic cases [17]. In cases of isolated IAU, systemic treatment may be an overtreatment and carries a higher risk of side effects [19]. This emphasizes a limitation of recent therapeutic strategies and the need for more targeted immunosuppressive treatment and a better understanding of the underlying pathophysiology.

Local inflammatory responses enhance blood vessels permeability, leading to infiltration of the anterior chamber with leukocytes and plasma proteins, which during slit-lamp examination can be seen as cells floating in the aqueous humor [20]. Exploring inflammatory mechanisms in intraocular space is possible thanks to examination of the aqueous humor [21]. Thorough assessment of local cytokines and chemokines helps in the identification and better comprehension of immune responses at affected site, which might not be reflected by systemic markers [22]. Studies show that there is a Th17 driven immune reaction dominance in IAU, featuring raised IL-6, IL-17, and TNF-α concentrations. Changes in regulatory T-cell function and local immune regulation have been noted, suggesting pro-inflammatory and regulatory function disturbances [23].

IL-10-producing T cells count has been proven to be elevated in AH in contrast to their count in PB in individuals with isolated uveitis, whereas this is not observed in those with systemic inflammatory diseases, further emphasizing that there's a localized immunoregulatory response in the eye of isolated uveitis [24]. If autoreactive effector T cells and local immune memory persist, leading to repeated flare-ups and further reinforcing the necessity for focused research on targeted immunomodulation intended to rebalance equilibrium between effector and regulatory immune responses [25]. Instead of systemic inflammation reduction, future treatments might locally target specific cytokines associated with recurrence or adjust IL-23/Th17 pathways, thus targeting the main processes that cause relapse of idiopathic uveitis [26].

There are no approved targeted therapies for IAU, and future approaches may involve, among others, topical agents that modulate cytokines or locally administered biologics aimed at inhibiting specific inflammatory pathways [27].

A better insight into local intraocular immune mechanisms might enable the development of therapies that not only manage acute inflammation but also prevent recurrence of disease, while reducing systemic exposure.

Conclusions

The presented case provides a 10-year long observation of predictable, recurrent IAU. The idea of localized immune dysregulation is underlined, concurrently highlighting the limitations of existing treatment. This case additionally puts emphasis on the importance of differentiating localized ocular immune activation from systemic inflammation. More precise studies on intraocular immune pathways could lead to more targeted local treatment and personalized management, providing a tailored method for addressing IAU.

Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of interests: The authors have declared that no competing interests exist.

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