Glaucoma Secondary to Uveitis Due to Posner Schlossman Syndrome

MD Paulo Henrique Nesi de Campos

ABSTRACT

Considering the clinical relevance of secondary glaucoma due to uveitis in posner-schlossman syndrome (PSS), this study aims to evaluate the natural history of this condition in a patient. To that end, we present the case of a 41-year-old man who experienced hyperemia, mild eye discomfort and decrease in visual acuity in the left eye. The initial ophthalmological examination revealed an intraocular pressure (IOP) of 30 mmhg in the left eye and 19 mmhg in the right, with gonioscopy showing an open angle and trabecular pigmentation. The patient was treated with topical corticosteroids, resulting in normalization of IOP. During follow-up, the patient experienced recurrent episodes of anterior uveitis with IOP ranging from 22 mmhg to 28 mmhg until april 2024. OCT examinations showed thinning of the nerve fiber layer, while visual fields remained normal. Thus, it is observed that early diagnosis and treatment are crucial for the effective management of secondary glaucoma due to uveitis, contributing to the preservation of visual function. It is concluded that rapid intervention with topical corticosteroids is essential to prevent glaucomatous neuropathy secondary to uveitis and to maintain the patient's ocular health.

Keywords: secondary glaucoma, uveitis, posner-schlossman syndrome, intraocular pressure, topical corticosteroids.

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I. INTRODUCTION

Posner-schlossman syndrome (PSS), also known as glaucomatocyclitic syndrome, was first described in 1948 and is characterized by recurrent episodes of mild anterior uveitis, associated with transient elevations in intraocular pressure (IOP). The episodes are typically unilateral and self-limiting, lasting from a few hours to weeks. Although its exact etiology remains unclear, recent studies suggest a possible association with infectious agents such as cytomegalovirus (CMV), herpes simplex virus (HSV), and possibly helicobacter pylori. (1) Additionally, there are investigations indicating a genetic relationship with the presence of HLA-Bw54 in Japanese patients with PSS. This association with HLA suggests a genetic basis for PSS. However, the contribution of HLA to the entire PSS spectrum is unknown. (2)

PSS predominantly affects young adults, with a higher incidence in men, but it can also occur in children and the elderly. The main clinical challenge lies in the proper control of IOP during episodes, as persistent IOP elevation can lead to secondary glaucoma and permanent damage to the optic nerve. Studies reviewing the clinical experience with the syndrome over 10 years highlight that factors such as elevated IOP and prolonged episode duration increase the risk of glaucomatous damage, emphasizing the importance of early diagnosis and appropriate IOP management to prevent long-term complications. (1)

1.1 Objectives

This study aims to report a case of posner-schlossman syndrome that progressed to secondary glaucoma, highlighting the importance of early diagnosis and proper IOP management during episodes. Specifically and measurably, it seeks to evaluate the therapeutic interventions used and their impact on IOP control, as well as discuss the possible complications associated with disease progression.

II. THEORETICAL FRAMEWORK

Posner-Schlossman syndrome was first described in 1948 as a condition characterized by recurrent, acute attacks of mild, unilateral, nongranulo-

matous, anterior uveitis accompanied by markedly elevated intraocular pressure. (1) However, there are some rare descriptions of bilateral simultaneous presentation of PSS (4).

Historically, PSS was seen as a self-limiting condition with a positive prognosis, which led to inadequate long-term follow-up and treatment, particularly regarding intraocular pressure. With advancements in research on PSS and long-term follow-up studies, it became clear that secondary glaucoma can develop in some patients. (3) Since its initial description, the cause of posner-schlossman syndrome has been widely discussed. Recently, some cases have been linked to viral infections, such as cytomegalovirus (CMV), herpes simplex (HSV), and varicella-zoster virus. Polymerase chain reaction (PCR) analyses of aqueous humor from patients with PSS revealed that 52% tested positive for CMV(1) Furthermore, recent studies have indicated that in some patients with PSS, the corneal endothelial cell density (CEC) is lower in the affected eye compared to the healthy eye, and that some of these situations are associated with CMV infections. (1) The overwhelming evidence suggests that the pathogenesis of PSS is infection of the anterior chamber, most commonly by cytomegalovirus, and clinicians should have a high index of suspicion and a low threshold for performing aqueous biopsy to detect the virus by PCR. Why glaucomatocyclitis develops in some patients and endotheliitis in others remains to be resolved and will be a focus of research in the future. (1)

On ophthalmologic examination, patients have open angles on gonioscopy and recurrent unilateral episodes of mild iritis with elevated intraocular pressure. During attacks, blurred vision and mild inflammation in the anterior chamber occur, along with small keratic precipitates. These episodes usually resolve within a few days, and IOP normalizes between attacks. PSS is more common in young individuals, making the prevention of vision loss due to elevated IOP crucial. (1) Although there is considerable evidence that PSS can result in secondary glaucoma, there is limited data on the clinical differences between patients with PSS who develop secondary glaucoma and those who experience just intermittent IOP elevations. (2)

The treatment of posner-schlossman syndrome aims to control both inflammation and elevated intraocular pressure, to prevent optic nerve damage caused by the condition (1).

Frequent attacks of high IOP represent a significant risk, as they can compromise vision, leading to progressive visual field defects. Therefore, controlling IOP is the main treatment goal, with an initial approach combining anti-inflammatory and antiglaucomatous eye drops. However, some patients do not respond to this treatment and may require glaucoma surgery to prevent visual field loss. Trabeculectomy has proven effective in some of these cases. Although surgery is traditionally not indicated for PSS, in cases of frequent recurrence, secondary glaucoma may develop, requiring surgical intervention for control (3). New surgical techniques have been performed with superior efficacy and safety, the microinvasive glaucoma surgeries (MIGs), being a safer alternative to traditional trabeculectomy. (4) However, in general, only a subset of patients with PSS who develop progressive visual field defects require glaucoma surgery. (1)

II. METHODOLOGY

This study is a clinical case report that describes the evolution of posner-schlossman syndrome in a 41-year-old male patient. The initial diagnosis was made through a complete ophthalmological exam, including intraocular pressure measurement, fundoscopy, gonioscopy, and imaging exams, such as optical coherence tomography (OCT) and perimetry. The research was conducted between 2019 and 2024, with periodic follow-up to assess the evolution of the condition and response to treatment. The patient received treatment with topical corticosteroids to control the uveitis, and when necessary, anti-glaucomatous medications were introduced to reduce IOP.

Included in this study were the follow-up records of the patient, laboratory exams, the evolution of IOP and changes in the retinal nerve fiber layer over time. Perimetry exams were also conducted

to monitor potential damage to visual field of the patient. The clinical evolution was monitored based on literature reviews and guidelines on the management of PSS and secondary glaucoma, considering evidence-based therapeutic interventions.

IV. CASE REPORT

The patient was admitted to the medical service in 2019 reporting symptoms of hyperemia, mild eye discomfort and decrease in visual acuity in the left eye for 4 days. He had a family history of a father with glaucoma who had already undergone surgery. On ophthalmological examination he presented an IOP of 19 mmhg in the right eye, IOP of 30mmhg in the left eye, mild iritis and discreet reagent mydriasis in the left eye characterizing a picture of hypertensive anterior uveitis. Fundoscopy revealed a cup/disc ratio of 0.2 in both eyes.

Considering PSS as a plausible hypothesis, treatment with steroids, antiglaucomatous eye drops was started and an etiological investigation was started. The patient did not consent to paracentesis for aqueous humor sample for virus analysis. He underwent OCT and visual field examination at the time, in both eyes, showing no significant changes. Blood serology revealed that the patient was positive for anti-CMV IgG and anti-HSV IgG antibody (table 1)

Subsequently the patient presented 7 more episodes of hypertensive uveitis in the same left eye during the 48-month follow-up, with IOP ranging from 22mmHg to 28mmHg, always treated with corticosteroid and antiglaucomatous eye drops. However, OCT exams demonstrated a progressive thinner of the nerve fiber layer of the retinal in both eyes (Figure 1, 2 and 3), more pronounced in the left eye, although the visual fields remained normal (Figure 4, 5, 6 and 7). The hypothesis of onset of primary open-angle glaucoma in the contralateral eye was risked due to the progressive thinner of the nerve fiber layer and chronic treatment was started in both eyes with frequent monitoring.

PSS appears to be initially self-limited, however, over time, it can result in chronic open-angle glaucoma and consequent permanent loss of visual field. In the case in question, the patient, in addition to developing thinning of the nerve fiber in the eye with the crises, also had thinning in the contralateral eye, indicating the onset of primary open-angle glaucoma, requiring treatment and monitoring in both eyes to prevent visual loss.

Recent studies on PSS corroborate that elevation of IOP during uveitis attacks is one of the main risk factors for the development of secondary glaucoma. Early interventions with topical corticosteroids and antiglaucoma medications have shown to be effective in controlling, preventing long-term visual complications. However, the thinner of the nerve fiber layer observed in the patient reflects the continuous risk of glaucomatous neuropathy, suggesting that close monitoring is essential to adjust interventions according to clinical evolution. (3)

Classically, eyes with presumed PSS have been treated with steroids and antiglaucoma medications. However, the disadvantages of such a practice have been emphasized recently. The repeated use of steroids may be permissive to viral replication, leading to increasingly frequent attacks and attendant glaucomatous damage. Although the optimal management of these eyes is still uncertain, topical NSAIDs theoretically may be a safer option when PCR analysis of the aqueous humor is positive for CMV. (6)

We conclude that a correct diagnosis is important in establishing effective treatment, since the fact that the patient had a hypertensive crisis in one eye did not rule out the possibility of the onset of chronic glaucoma in the other eye. Although it is difficult to establish a therapeutic standard due to the rarity of the disease, the regimen with topical hypotensive and topical NSAIDs or corticosteroids appears to be the most appropriate form of treatment. The surgical procedure is reserved for select and refractory cases. (6)

V. CONCLUSION

The clinical case discussed highlights the importance of early diagnosis and aggressive intervention in the management of

posner-schlossman syndrome, particularly to avoid progression to secondary glaucoma. Treatment with topical corticosteroids and anti-glaucoma medications was effective in normalizing IOP and preserving the patient's visual function, but the thinning of the nerve fiber layer indicates the need for rigorous follow-up and consideration of additional therapies if necessary. Early and continuous IOP management is essential to avoid permanent optic nerve damage and maintain the patient's ocular health.

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Table 1: Laboratory findings

Serum HSV IgG/IgM	+ / -
Serum CMV IgG/IgM	+ / -
Serum HTLV	-
Serum FTABs IgG/IgM	- / -
HIV screen	-
HLA-B27	-
RF Test	-
ANA Test	-

CMV Cytomegalovirus, *HSV Herpes Simplex Virus*, *HIV* human immunodeficiency virus, RF Rheumatoid Factor, ANA Antinuclear antibody

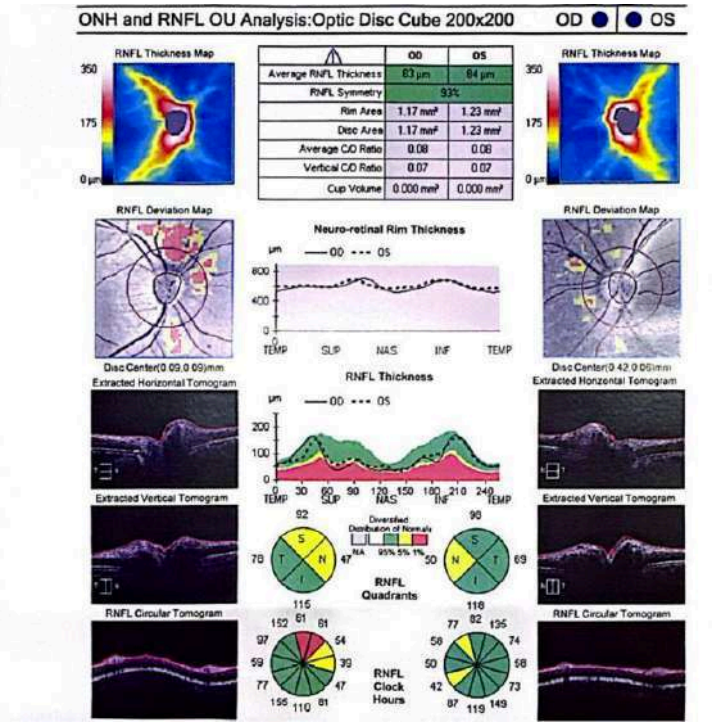

Figure 1

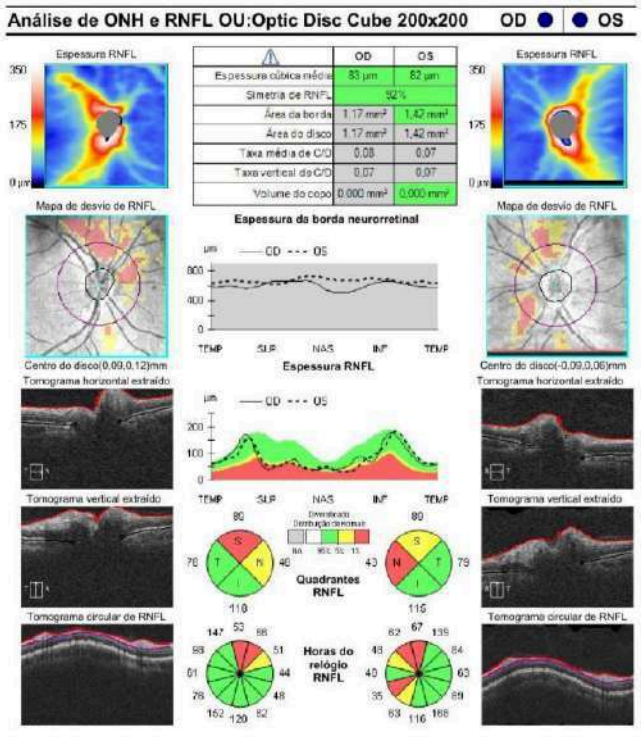

Figure 2

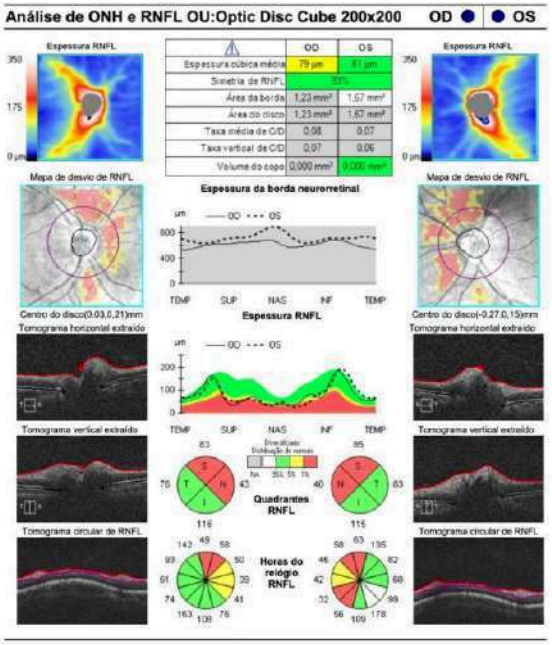

Figure 3

Evolution of nerve fiber layer thinning, Figure 1 (2019), Figure 2 (2023), Figure 3 (2024)

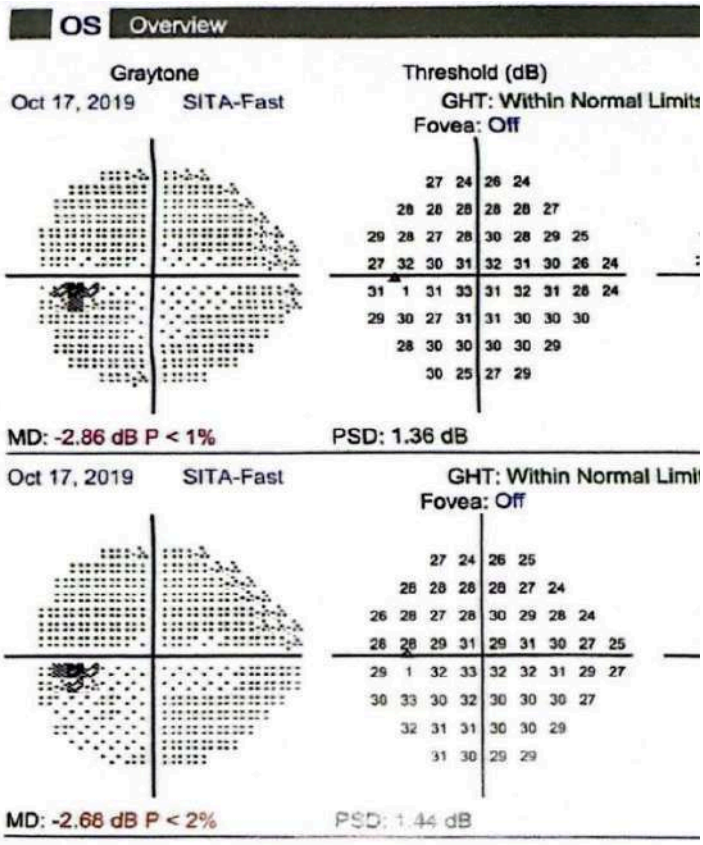

Figure 4

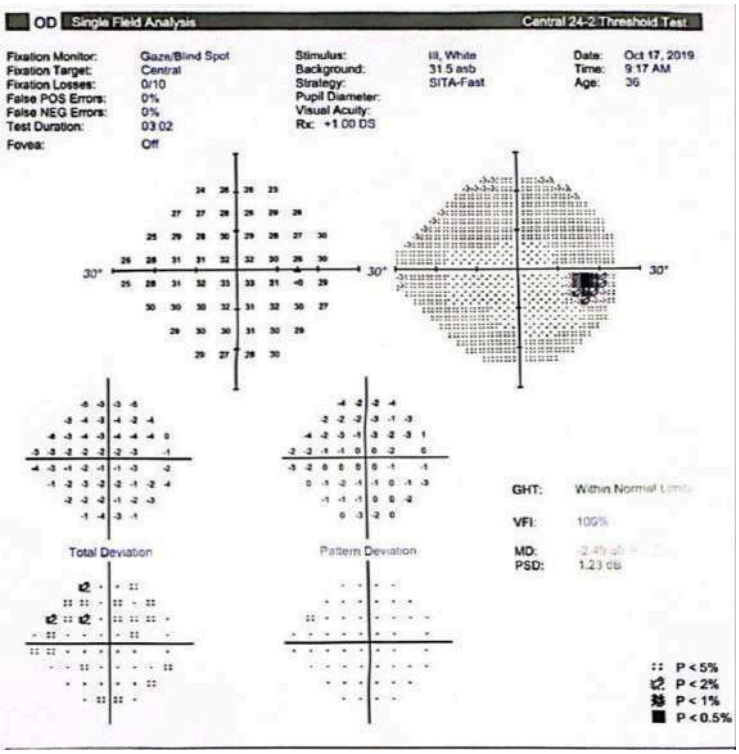

Figure 5

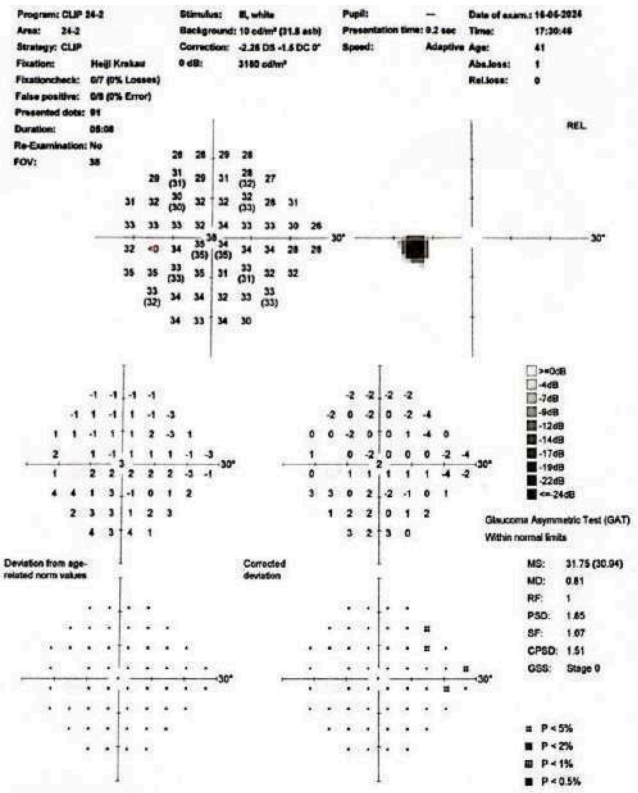

Figure 6

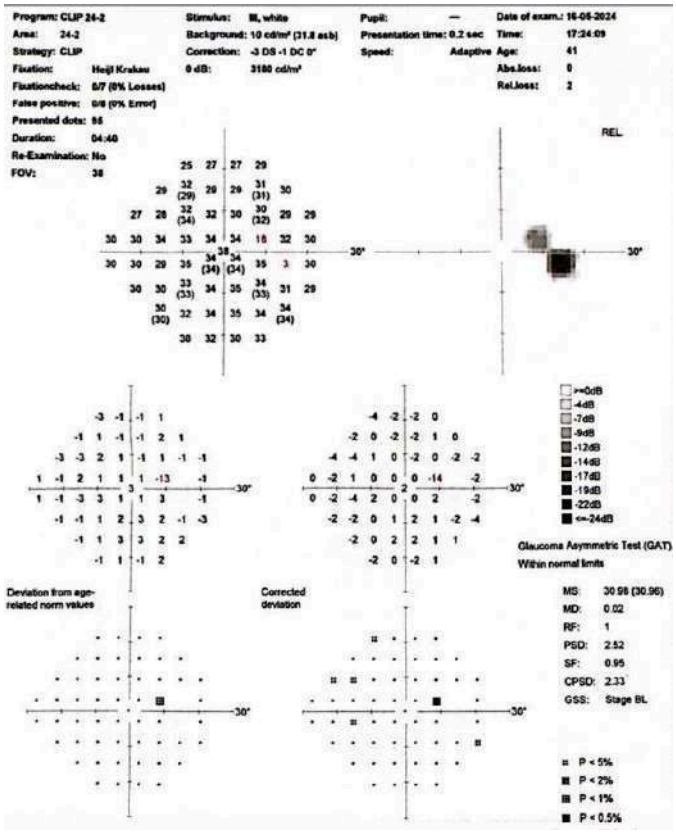

Figure 7

Evolution of the perimetry, Figure 4 and 5 (2019), Figure 6 and 7 (2024)