World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(CASE REPORT)

Check for updates

Posner and Shlossman syndrome: Case report and literature review

Ghizlane Daghouj *, Sara Ettouri, Kawtar Elhadi, Oumama Es-samery, Zyad Laftimi, Loubna El Maaloum, Bouchra Allali and Asma El Kettani

Department of Pediatric ophthalmology, 20 August hospital, CHU Ibn Rochd, Hassan 2 university, Casablanca, Morocco.

World Journal of Advanced Research and Reviews, 2025, 27(01), 1298-1301

Publication history: Received on 06 June 2025; revised on 12 July 2025; accepted on 14 July 2025

Article DOI: https://doi.org/10.30574/wjarr.2025.27.1.2636

Abstract

Posner-Schlossman syndrome (PSS), also known as glaucomatocyclitic crisis, is a rare ocular condition characterized by recurrent, unilateral episodes of elevated intraocular pressure (IOP) accompanied by mild, idiopathic anterior uveitis. First described in 1948, its etiology remains uncertain, with factors such as viral infection, autoimmune response, and genetic predisposition being proposed. PSS typically affects adults between ages 20 and 50, presenting with blurred vision, minimal anterior chamber inflammation, and significant IOP elevation, often out of proportion to the degree of inflammation. Diagnosis relies on clinical findings, and management focuses on controlling both the inflammatory response and IOP, primarily through topical anti-inflammatory and anti-glaucoma medications. In refractory or recurrent cases, surgical intervention may be necessary. While the condition is self-limiting in most cases, recurrent attacks can lead to glaucomatous optic nerve damage over time, underscoring the importance of long-term monitoring and individualized patient care.

We report a case of 48-year-old patient presented with a sudden onset of painful, red right eye and decreased visual acuity. Examination revealed significantly elevated intraocular pressure (48 mmHg), corneal edema, fine white retrodescemetic precipitates, semi-mydriasis, and minimal anterior chamber inflammation, with a normal optic disc and open angles; the left eye was unremarkable. Initial management with intravenous mannitol, topical beta-blocker and carbonic anhydrase inhibitor, and oral acetazolamide provided limited pressure control, requiring the addition of topical alpha-adrenergic therapy. Once intraocular pressure normalized, persistent anterior uveitis was managed with local corticosteroids, resulting in resolution of inflammatory signs. This case highlights the importance of individualized, stepwise management in Posner-Schlossman syndrome to address both intraocular pressure elevation and anterior uveitis.

Keywords: Posner-Schlossman Syndrome; Anterior Uveitis; Glaucomatocyclitic Crisis; Intraocular Pressure

1. Introduction

Posner Adolph and Schlossman Abraham first reported glaucomatocyclitic crisis, by drifting in their paper until the title of 'Syndrome of unilateral recurrent attacks of glaucoma with cyclitic symptoms' in 1948 (1) , an uncommon form of glaucoma characterized by recurrent unilateral episodes of markedly elevated intraocular pressure (IOP) with mild idiopathic anterior chamber inflammation (2).

This disease later became known as the Posner Schlossman syndrome (PSS) (3). In this syndrome patients present with blurred vision, show minimal anterior chamber activity, and raised intraocular pressure (IOP) (4).

* Corresponding author: Ghizlane Daghouj

Several factors such as viral infection, autoimmune, autonomic dysregulation, vascular endothelial dysfunction and allergic conditions have been proposed as possible contributors to the development of this disease , however the exact etiology and pathophysiology are still not fully understood (5).

Successful disease management is often achieved by topical treatment, although systemic therapy and even surgical intervention may be require (6). Treatment of PSS aims at controlling the inflammation and IOP elevation (2) , anti-inflammatory and anti-glaucoma drug are usually the basis of treatment.

2. Case report

We report a case of patient of 48 years, wearer of optical corrective glasses since childhood. The reason for consultation is a painful red right eye with sudden decrease in visual acuity since 04 days. The ophthalmology examination at the right eye found: a corrected visual acuity at hand movement with an automatic refraction (+ 1 (-0,75 à 90°)), an ocular tonus at 48 mmHg. At the anterior segment: corneal edema, fine central white retrodescmetic precipitates, areactive semi mydriasis, calm anterior chamber with normal depth and normal iris (No synechiae or nodules). A normal papilla with an 360°open angle and a normal visual field. The examination at the left eye was totally normal.

The patient received intravenous mannitol in the emergency department. She also received dual therapy in fixed combination: beta-blocker (timolol) and topical carbonic anhydrase inhibitor (dorzolamide) reinforced with oral Acetazolamide. But we noted a persistence of ocular hypertonia so we add an alpha adrenergic (Brimonidine) then the ocular tone was controlled (18mmHg). There was a persistence of signs of anterior uveitis, local corticosteroid therapy was instituted, there was subsequently a disappearance of corneal retrodescmetic precipitates.

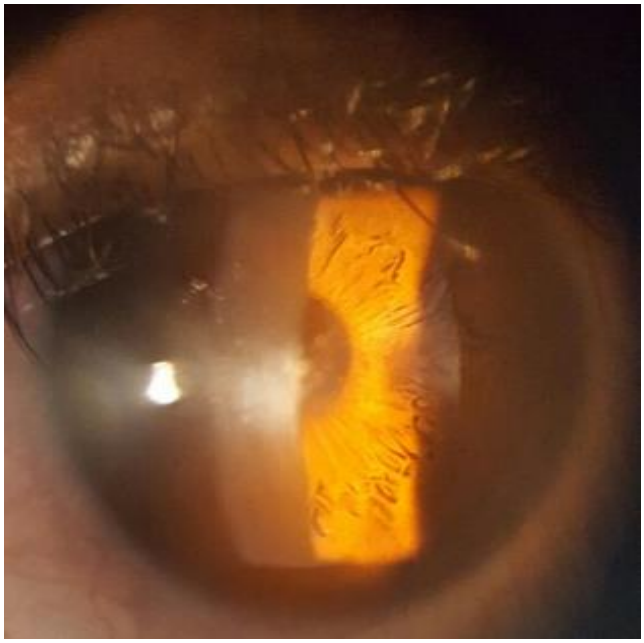

Figure 1 White fine retrodescmetic precipitates persistent after the crisis

3. Discussion

Most PSS studies have documented patient cohorts in the far east, with rates of disease in Japan and Singapore ranging from 1.7% to 4.3% of all uveitis diagnoses. Rates in northern Europe appear similar but the development of glaucoma is higher, with rates varying from 6.5% to 11.4% of all uveitic glaucoma (7)**.** PSS almost exclusively affects individuals aged 20–50 years. This was our patient's age range. However, there are reports of rare episodes in individuals older than 60 years as well as in adolescence (2). Males are more often affected. Recurrence is common and can be in either eye but intervals between attacks tend to increase during the course of the disease (8). PSS is a rare condition with an uncertain etiology and no recognized associated systemic conditions. A Japanese study of 22 patients with PSS showed that HLA Bw54 was positive in nine (41%) implicating a possible CD8 T-cell immunogenetic role (9). Herpes simplex virus has been detected in the anterior chamber of a small sample of three patients during ocular hypertensive attacks (8). PSS diagnosis is a clinical one with characteristic recurrent attacks of mild, unilateral, non- granulomatous, anterior

uveitis accompanied by markedly elevated intraocular pressure. These were the clinical signs that our patient presented with. Diffuse iris atrophy may be noted. Anterior chamber drainage angles remain open, with visual fields and optic discs initially appearing normal. Vision may be mildly affected (6). The eye can show little or no hyperemia, presenting symptoms being blurred vision or halos around lights (8).

The diagnosis is difficult and it may mimic a variety of ocular disorders (2). PSS and uveitic glaucoma have some common features. Both conditions show signs of anterior uveitis, elevated IOP, and respond to topical steroids. The key features to differentiate PSS from uveitic glaucoma include the mild iridocyclitis, lacking the development of posterior synechiae or peripheral anterior synechiae, and that the rise in IOP is typically out of proportion to the inflammatory process (6). Treatment of PSS aims at controlling the inflammation and IOP elevation. This should be customized to meet the patient's individual needs. The favored initial treatment is a combined regimen of an anti-inflammatory and anti-glaucoma drug. Topical b-blockers and/or carbonic anhydrase inhibitors are the drugs of choice and prostaglandin analogs' efficacity in PSS is not well established (2). These two molecules of choice (b-blockers and carbonic anhydrase) were the basic molecules of our patient's antihypertensive treatment. A topical corticosteroid such as prednisolone acetate 1%, followed by rapid taper, is usually successful in controlling inflammation. Potential IOP elevations caused by steroids in steroid-responsive patients may complicate the picture of PSS. Alternatively, a topical non-steroidal anti-inflammatory drug (NSAID) such as Diclofenac 0.1% or the equivalent can be used to control inflammation (2).

Usually, elevated IOP normalizes with the control of inflammation. Well-informed and educated patients often can sense an impending attack based on ocular symptomatology, and they can institute appropriate self therapy using an aqueous suppressant and a topical NSAID to blunt IOP elevations associated with treatment delays (2). If PSS attacks show increased frequency and IOP fluctuations are significant with attacks, surgical intervention may be indicated (2). Subramanian Dinakaran and al presented a patient who had bilateral Posner-Schlossman syndrome and underwent filtering surgery to control raised intraocular pressure in both eyes. During the follow up of more than 4 years, the control of IOP was good and he had no further attacks (4). A significant number of patients with PSS have glaucoma develop over time, and they need to have their optic disc appearance and visual fields carefully monitored. In a retrospective study published by *A. Jap and Al*, here were 28 men and 22 women, and their mean age at onset was 35 years. Fourteen eyes (26.4%) were diagnosed to have developed glaucoma as a result of repeated attacks of PSS. Patients with 10 years or more of PSS have a 2.8 times higher risk of developing glaucoma compared with patients with less than 10 years duration of the disease. Nine eyes (17%) underwent glaucoma filtering surgery with antimetabolites. Their postoperative follow-up ranged from 15 to 50 months (mean, 37 months). Four eyes continued to have episodes of iritis after surgery, and one of these eyes had elevated intraocular pressure during the event (10). It could also cause optic disc changes such as glaucomatous change and ischemic change (11). In their retrospective cohort study, *Tingting Gao* and al , they reported that CEC loss and RNFL thinning were present in the affected eyes (12).

4. Conclusion

Posner-Schlossman syndrome (PSS) or glaucomatocyclitic crisis is a rare, typically unilateral recurrent inflammatory ocular hypertensive disease in which diagnosis can be challenging (8). Whatever the case, the symptoms are self-limiting, with spontaneous resolution within days or weeks, even without treatment. The attacks tend to be recurrent. While PSS is not a common ED presentation, its acute presentation is usually due to elevated IOP, which is an ophthalmic emergency (13). The etiology of this disease remains unknown, however, the role of cytomegalovirus in the pathogenesis of glaucomatocyclitic crisis has frequently been postulated (14). Management during an episode is aimed at controlling the IOP using antiglaucoma drugs and minimizing intraocular inflammation (3) by anti-inflammatory drugs. Sometimes the use of filtering surgery is essential.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of informed consent

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

References

[1] Posner A, Schlossman A. Syndrome of unilateral recurrent attacks of glaucoma with cyclitic symptoms. Archives of Ophthalmology. 1 avr 1948;39(4):517-35.

[2] Shazly TA, Aljajeh M, Latina MA. Posner-Schlossman glaucomatocyclitic crisis. Semin Ophthalmol. 2011;26(4-5):282-4.

[3] Okonkwo ON, Tripathy K. Posner-Schlossman Syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cité 14 août 2023]. Disponible sur: http://www.ncbi.nlm.nih.gov/books/NBK576412/

[4] Dinakaran S, Kayarkar V. Trabeculectomy in the management of Posner-Schlossman syndrome. Ophthalmic Surg Lasers. 2002;33(4):321-2.

[5] Jiang JH, Zhang SD, Dai ML, Yang JY, Xie YQ, Hu C, et al. Posner-Schlossman syndrome in Wenzhou, China: a retrospective review study. Br J Ophthalmol. déc 2017;101(12):1638-42.

[6] Megaw R, Agarwal PK. Posner-Schlossman syndrome. Surv Ophthalmol. 2017;62(3):277-85.

[7] Megaw R, Agarwal PK. Posner-Schlossman syndrome. Survey of Ophthalmology. mai 2017;62(3):277-85.

[8] Green RJ. Posner-Schlossman syndrome (glaucomatocyclitic crisis). Clin Exp Optom. janv 2007;90(1):53-6.

[9] Hirose S, Ohno S, Matsuda H. HLA-Bw54 and Glaucomatocyclitic Crisis. Archives of Ophthalmology. 1 déc 1985;103(12):1837-9.

[10] Jap A, Sivakumar M, Chee SP. Is Posner Schlossman syndrome benign? Ophthalmology. mai 2001;108(5):913-8.

[11] Kim TH, Kim JL, Kee C. Optic disc atrophy in patient with Posner-Schlossman syndrome. Korean J Ophthalmol. déc 2012;26(6):473-7.

[12] Gao T, Song S, Ke X, Li S, Zhang D, Chen X, et al. Clinical Characteristics of Posner-Schlossman Syndrome Patients in China. Biomed Res Int. 2023;2023:4110344.

[13] Lippert J, Falgiani M, Ganti L. Posner-Schlossman Syndrome. Cureus. 12(1):e6584.

[14] Wu KY. Glaucomatocyclitic crisis and glaucomatous optic neuropathy. Taiwan J Ophthalmol. 2015;5(2):49.