

## Posner-Schlossman Syndrome: A Cause of Unilateral Ocular Hypertension: Case Report

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Received date: November 30, 2018; Accepted date: January 03, 2019; Published date: January 10, 2019

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### Abstract

A 40 y old immunocompetent male patient admitted, with the complaints of blurry vision, photophobia, red eye, and severe pain in the left eye for the last 2 days. Examination revealed mild anterior chamber reaction, corneal edema, appearance of keratic precipitates and a very high intraocular pressure (52 mmHg) with an unresponsive, semi-dilated pupil in the same eye. Gonioscopy revealed open angles in both eyes. He was treated with topical steroids and topical pressure-lowering agents with oral acetazolamide. The patient was also evaluated for systemic causes of red eye. Investigations carried out included routine blood tests and ESR, the results of which were all within normal limits. In the following 24 month period, the patient had two further episodes of unilateral IOP spikes associated with cyclitis.

This is an interesting and a rare uveitic condition. Although the list of differential diagnoses is long, the condition is relatively quickly identifiable by the presence of remarkable signs and symptoms. Medical and surgical treatments are indicated to reduce inflammation and to prevent long-term glaucomatous optic nerve damage related to the high intraocular pressure. Posner-Schlossman Syndrome does not always follow a completely uncomplicated course. Repeated episodes of elevated intraocular pressure can cause long-term sequelae such as glaucoma.

**Keywords:** Glaucomatocyclitic crisis; Posner-Schlossman syndrome; Ocular hypertension; Glaucoma

### Introduction

Glaucomatocyclitic crisis, also known as Posner-Schlossman syndrome (PSS), is a condition characterised by unilateral recurrent episodes of acute elevated intraocular pressure accompanied by nongranulomatous mild anterior chamber inflammation or fine white keratic precipitates [1]. The intraocular pressure may increase to 30-60 mmHg at the stage of attack [2]. In the acute stage of attack, the anterior chamber angles are open with the minimal glaucomatous optic neuropathy [3]. Attention has previously been paid to PSS, because some patients can usually be self-healing or misdiagnosed [4]. It is important to distinguish PSS with acute attack of primary angle closure from iridocyclitis [1,4]. This relatively rare disease's etiology and pathophysiology are still not fully understood, but several factors such as viral infection, autoimmune dysregulation, vascular endothelial dysfunction and allergic conditions have been proposed as possible contributors to the cause of glaucomatocyclitic crisis [3,4]. The attacks resolve spontaneously or with medical treatment in a few days or a week, and the intraocular pressure is normal in the remission periods [5]. PSS typically occurs in younger individuals, making the prevention of vision loss caused by high intraocular pressure an important goal in disease management [1,2]. Treatment of glaucomatocyclitic crisis aims at controlling inflammation and elevated intraocular pressure [3,4]. Frequent attacks of high pressure are particularly hazardous because of easily affecting vision by causing progressive visual field defects [3-5]. Therefore intraocular pressure control is the most important goal with anti-glaucoma and anti-

inflammatory eye drops [4,5]. However, some patients are not responsive to medical treatment and must undergo glaucoma surgery to prevent optic nerve damage [4-6].

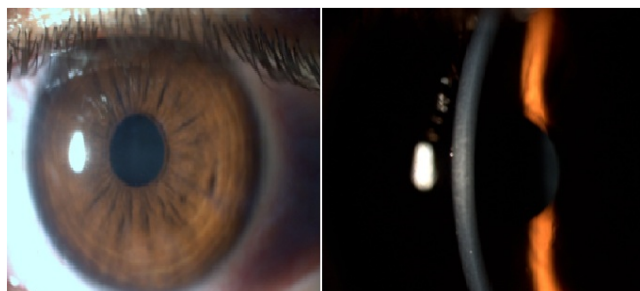
Here we aimed to report a rare initial, unilateral, simultaneous of glaucomatocyclitic crisis case.

### Case Presentation

A 40 y old man presented with decreased vision, photophobia, red eye and pain in his left eye for 2 days. He had no previous medical history or trauma history. He was using no systemic medication. His best corrected visual acuity was 10/10 in the right eye and 8/10 in the left eye. The slit lamp examination of the right eye was normal. On the slit lamp examination of the left eye, there was mild epithelial edema of the cornea with a few keratic precipitates and few aqueous cells (Figure 1). The intraocular pressure was 16 mmHg in the right eye and 40 mmHg in the left eye. Gonioscopy showed an open angle and the absence of peripheral anterior synechiae in both eyes (Schaffer grade 3 to 4). On the fundus examination, the retina and the optic discs were normal in the both eyes. Visual fields and color vision was normal. Pupil responses were normal. All laboratory data were normal, including routine blood tests and ESR. Therefore the patient was diagnosed with Posner-Schlossman syndrome.

Anterior segment inflammation and elevated intraocular pressure were controlled after 2 days of treatment with antiglaucoma medication and topical corticosteroids. Following treatment with fixed combination of timolol 0.5% and dorzolamide 2% twice daily plus latanoprost 0.005% once daily and topical prednisolone acetate 1% thrice daily, the intraocular pressure was maintained at 14-15 mmHg.

Patient's cup/disc ratio was noted 0.2 in the right eye and 0.3 in the left eye. There were no abnormal findings in orbital and brain MRI. Follow up was performed every month over a period 24 month. The patient experienced two repeat attacks during the 24 month follow-up period. All episodes of attacks were well controlled with antiglaucoma and anti-inflammatory medications. A written informed consent was obtained from the patient.



**Figure 1:** Mild epithelial edema of the cornea with a few keratic precipitates and few aqueous cells were seen on slit lamp examination.

## Discussion

Possner-Schlossman syndrome occupies an important and unique position among the many types of glaucoma associated with uveitis [7]. The fact that this condition exists, characterized by recurrent attacks of high tension associated cyclitis which has an essentially benign course, requires intensive medical treatment or sometimes surgery [6,8]. Although abnormal vascular process, autonomic defect, allergic conditions, variation of developmental glaucoma, cytomegalovirus and herpes simplex virus have been suggested, the etiology of glaucomatocyclitic crisis is unknown [2-4]. Possner-Schlossman syndrome is known as a self-limiting eye disease with benign prognosis, but the glaucomatous impairment of visual function in some patients has been increasingly reported [3,5,7]. Our patient also admitted to clinic with complaint of low vision. Glaucomatocyclitic crisis tends to affect patients between 20 and 50 years of age [2,8]. Our patient's age was 40 which fits into the typical age group.

Possner-Schlossman syndrome has some clinical features of glaucoma and uveitis, such as the elevated intraocular pressure and keratic precipitates [1,2,4]. Inflammatory signs (keratic precipitates and anterior chamber cells) and high intraocular pressure were noted in the our patient's initial presentation. Because of the risk of progression to secondary chronic open angle glaucoma, patients with glaucomatocyclitic crisis must be monitored closely [1,2,6]. Our patient had 2 recurrent attacks that required treatment to control intraocular pressure and suppress inflammation during 24 month follow-up period.

Early diagnosis and proper treatment could decrease the risk of advanced glaucoma [3-5]. Trabeculectomy should be considered early in the management of glaucomatocyclitic crisis with persistently high intraocular pressure in order to control intraocular pressure and

prevent glaucomatous changes [1,4,6]. All attacks of our case were well controlled with the fixed combination of timolol 0.5% and dorzolamide 2% twice daily plus latanoprost 0.005% once daily and topical prednisolone acetate 1% thrice daily. Our case was no need to surgical intervention for attacks. Thoughtful attention and aggressive intraocular pressure control with suppressing inflammation are required especially in these patients [4-7].

The rise of intraocular pressure is often out of proportion of the severity of the inflammation [1-3,8]. Glaucomatocyclitic crisis can present challenging diagnosis at first presentation with intraocular pressure that is disproportionate to the mild symptoms and minimal anterior segment inflammatory signs [3-5,6]. Therefore, glaucomatocyclitic crisis may be misdiagnosed as acute or chronic angle closure glaucoma or even as Fuchs heterochromic iridocyclitis [1,6,8]. The definition of acute or chronic angle closure glaucoma includes the close angles which was not a feature of our case. Fuchs heterochromic iridocyclitis and glaucomatocyclitic crisis have some aspects in common [1,6]. Keratic precipitates occurs over the entire surface of the cornea in Fuchs heterochromic iridocyclitis while the precipitates were limited to the central end inferior cornea in the Possner-Schlossman syndrome [1,6,8]. In Fuchs heterochromic iridocyclitis, iris heterochromia and cataract can be seen [6,8]. In addition, Fuchs heterochromic iridocyclitis tends to be unresponsive to steroid therapy [1,8]. Our case had no features of Fuchs heterochromic iridocyclitis.

## Conclusion

Glaucomatocyclitic crisis (Possner-Schlossman syndrome) is an elusive disease and care should be taken in making the diagnosis. Our case which highlights a differential diagnosis and management approach should future similar cases be encountered adds further presenting features to the small body of existing literature.

## References

1. Shazly TA, Aljajeh M, Latina MA (2011) Posner-Schlossman glaucomatocyclitic crisis. *Semin Ophthalmol* 26: 282-284.
2. Hong Y, Wang Mi, Wu L (2017) In vivo confocal microscopy of Posner-Schlossman syndrome: comparison with herpes simplex keratitis, HLA-B27 anterior uveitis and acute attack of primary angle closure. *Sci Rep* 7: 9832.
3. Jiang JH, Zhang SD, Dai ML, Yang JY, Xie YQ, et al. (2017) Posner-Schlossman syndrome in Wenzhou, China: a retrospective review study. *Br J Ophthalmol* 101:1-5.
4. Maruyama K, Maruyama Y, Sugita S, Mori K, Yokoyama Y, et al. (2017) Characteristics of cases needing advanced treatment for intractable Posner-Schlossman syndrome. *BMC Ophthalmol* 17: 45.
5. Jap A, Sivakumar M, Chee SP (2001) Is Posner-Schlossman syndrome benign? *Ophthalmology* 108: 913-918.
6. Campana F, Caramella G, Dallorto L, Rolle T (2015) Long term efficacy of deep sclerectomy in Possner-Schlossman syndrome. *BMJ Case Rep* 2015: bcr2014205783.
7. Tsai JC (2015) Detection of progression of retinal nerve fiber layer loss by optical coherent tomography in a patient with glaucomatocyclitic crisis. *Taiwan J Ophthalmol* 5: 90-93.
8. Green RJ (2007) Possner-Schlossman syndrome (glaucomatocyclitic crisis). *Clin Exp Optom* 90: 53-56.