Case Report

Presentation of Posner Schlossman Syndrome and Viral Uveitis

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Purpose: To discuss an interesting case in which treatment of Posner Schlossman syndrome lead to unmasking signs of viral Uveitis.

Clinical management: A patients was brought to Nishtar hospital Multan for management of unilateral raised intra ocular pressure (IOP) being caused by anterior Uveitis. First examination revealed Posner Schlossman syndrome as a diagnosis of exclusion. After 48 hours of treatment features of viral uveitis appeared that lead to change the diagnosis.

Posner Schlossman syndrome is an inflammatory condition that leads to raised intra ocular pressure (IOP)due to trabeculitis[1]. It affects patients between the ages of 20- 50 years[2]. It has no clear etiology. It is also known as glaucoma to cyclitic crsis. It usually presents as unilateral, episodic, recurrent attacks of elevated intra-ocular-pressure along with mild anterior uveitis, usually with no posterior synechae. Multiple mechanisms may be responsible for most cases of uveitic glaucoma. Herpes uveitis may directly cause trabeculitis and thus increase IOP. In addition inflammatory cells can cause trabecular obstruction.

CASE REPORT

This is an observational case report carried out at Nishtar Hospital Multan. A patient was examined in Out Patient Department and was admitted in ward for the management.

A male 38 years old presented with moderate pain in right eye for two days, it was associated with vomiting and severe right sided headache. Examination revealed ciliary congestion, cornea clear, anterior chamber reactivity +3, no KP's, no flare, anterior chamber angle was widely open (grade IV in 360 degree). IOP was 44mmhg in right side and 18mmhg on left side. Pupil was reactive, round. Lens was clear, posterior segment showed no reactivity and retina was flat. Cup disc ratio was 0.5-0.6 on right side and 0.4 on left side. Other eye was quite normal. No history of IHD, TB, DM, joint pain and allergy. No family history of such ailment was found.

Provisional diagnosis was made of Possner Schlosman Syndrome. Topical acetazolamide + timolol +apraclonidine along with oral acetazolamide were started to lower the IOP. Intensive topical steroids were started to minimize anterior chamber activity.

On the second post treatment day the patient had corneal haze with few punctate epithelial types of erosion. Detailed Slit Lamp examination shows presence of Weiss Ring in deep stroma.

Diagnosis was reconsidered and changed to viral keratouveitis[4]. Therapy was modified to oral valacyclovir 1 g 3 times / day along with topical anti-viral and anti-glaucoma therapy[4]. Mild steroids were also added in regimen under the cover of anti-viral and topical antibiotics. Patient was observed for 3 days and it was found that reactivity responded well, corneal haze was also responding. Patient was discharged on medicine after making sure for close follow-up. On first follow-up after 7 days his corneal haze was markedly reduced and there was no reactivity, IOP was controlled.

DISCUSSION

In 1948, Posner and Schlossman first recognized glaucomatocyclitic crisis and described the features of this syndrome[6]. Posner and Schlossman identified the following features as recurrent episodes of mild

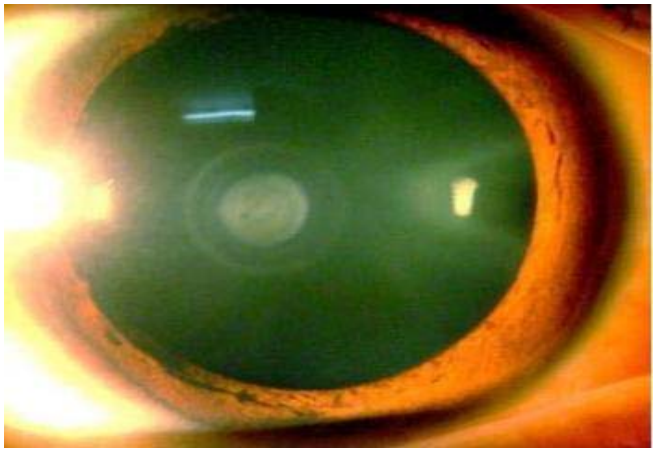

Fig. Central Keratitis after steroid therapy

cyclitis, uniocular involvement, duration of attack varying from a few hours to several weeks, signs of a slight decrease in vision, elevated IOP in the range of 40 – 60 mmHg with open angles, corneal oedema with a few keratic precipitates, heterochromia with anisocoria, and a large pupil in the affected eye. Normal visual fields, normal optic disc, normal IOP, outflow facility and all provocative tests between episodes[5]. Between attacks there are generally no signs or symptoms of inflammation or glaucoma and contra lateral eye is usually normal. Episodic changes in the trabecular meshwork lead to impairment of outflow facility and result in an elevation of IOP. These changes are accompanied by mild intraocular inflammation. In the acute phase of PSS, optic nerve head parameters and retinal flow rates were altered; however, all returned to normal without any permanent damage after control of the elevated IOP. Electroretinogram studies in the acute phase demonstrate a selective reduction in the S-cone b-wave[7]. It is usually of unknown etiology but cytomegalovirus[8] and herpetic[9] infections have been reported.

A significant number of patients with PSS develop glaucoma over time and they need to have their optic disc appearance and visual field carefully monitored.

CONCLUSION

It has been found in this study that viral Uveitis and Posner Schlossman syndrome, both, may present with same signs as reactivity, raised intra ocular pressure etc until confirmation of diagnosis.

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