SYMPATHETIC OPHTHALMIA : A BLINDING COMPLICATION OF OCULAR INJURY

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ABSTRACT

Sympathetic Ophthalmia is a rare and blinding ocular complication due to ocular injury. This condition in a male patient aged 25 years, is reported. The role of early recognition and management of this condition to preserve good vision is discussed.

Key Words: Sympathetic ophthalmia, granulomatous panuveitis, ocular injury.

INTRODUCTION

Sympathetic ophthalmia (SO) is a rare bilateral diffuse granulomatous panuveitis following uveal injury to the eye. The exact cause is not known but it is believed to be related to delayed type of hypersensitivity reaction to the uveal pigments.[1] The traumatized eye is called exciting eye and the fellow eye is referred as sympathizing eye. The onset of inflammation in the sympathizing eye has been reported as early as 5 days and as late as 66 years.[2] In general the clinical features of SO are detected within first 3 months after ocular trauma and it is characterized by mild ocular pain, photophobia, redness, blurring of distant and near vision (due to loss of accommodation). The clinical signs include granulomatous panuveitis with multifocal choroiditis and exudative retinal detachment (ERD). Timely recognition and prompt management with high dose of oral corticosteroid and cytotoxic drugs alone or both in combination are crucial to preserve vision in one or both the eyes. Subtenon injection of soluble steroids, topical steroid, cyclosporine A and cycloplegics are also combined with oral medications. This case is reported for it's rarity.

CASE REPORT

A male patient aged 25 years, farmer by profession, from Gulmi distric of Nepal, presented to retina clinic of Rana Ambika Shah Eye Hospital with the chief complaints of blurring of vision, photophobia and watering from right eye (RE) of 5 days duration. Past history revealed that he had sustained severe trauma (possibly globe rupture) in his left eye (LE) 9 weeks back by an ox foot. His visual acuity (VA) in RE was 6/6 and in LE no perception of light (NPL). His document showed that he was treated conservatively with oral antibiotic and non-steroidal anti-inflammatory drugs, topical antibiotics and steroid.

On systemic examination he had normal findings. On ocular examination RE had best corrected VA of 1/60 and LE phthisis. Slit lamp examination of RE was performed. Conjunctiva was red and circumferentially congested. Cornea showed diffuse white and round fresh keratic precipitates and cells +4 and flares +4 reaction in anterior chamber. Pupil was small, irregular and poorly reacting to light due to extensive posterior synechiae (nearly 360-degree). Lens was clear except few iris pigments

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on its anterior surface. There was poor visibility of posterior segment due to hazy media. Indirect ophthalmoscopy of posterior segment revealed vitritis, hyperemic and swollen optic disc (optic neuritis). B- scan ultrasonography was performed and it revealed suggestive features of exudative retinal detachment (depended retinal detachment and mobile subretinal fluid). Investigation of blood-TLC, DLC, ESR and Hb were within normal findings. Mantoux reaction was 5x5 mm in 72 hrs, VDRL test was nonreactive and chest x-ray was normal. Platelate count, serum creatinin and liver function test were performed before introducing cytotoxic drugs and were also found to be normal. A diagnosis of sympathetic ophthalmia was made and treatment started with high dosage of oral corticosteroid combined with intensive topical steroid (prednisolone acetate one hourly), atropine eye drop (three times daily), subconjuntival injection of mydricain 0.5ml (start dose only) and posterior subtenons injection of triamcinolone acetenoid 20 mg. Initially tablet prednisone 120 mg (2 mg/Kg body weight) once daily was given and once improvement was noticed the dose was tapered gradually. Right eye VA improved from 1/60 to 6/9 (with pinhole) and with minimal vitritis and broken posterior synechia. After 3 months, only oral steroid was substituted by cytotoxic (azathioprine), as oral steroid was complicated with GI bleeding and deterioration of VA to 6/18 (with pinhole) due to recurrence of sympathetic ophthalmia with increased vitritis, choroiditis and exudatative RD. Tablet Azoran was given in 50 mg twice daily initially and tapered gradually and stopped after 2 months. At that time RE visual acuity was 6/6 (unaided) with quiet anterior segment and no posterior synechia. Fundus examination with +90.00 D and indirect ophthalmoscope revealed resolved choroiditis, vitritis and exudative RD. There were diffuse retinal pigment epithelial (RPE) degeneration and atrophic patches at macula including posterior pole and pale disc. (fig.1)

Patient has been kept on regular follow up without any medication since last 11 months and there was no recurrence of disease and maintained VA at 6/6 till the last visit (2004/11/8).

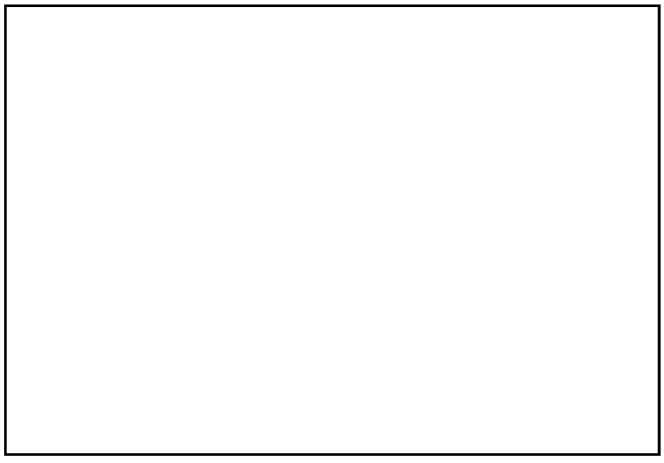

Fig.1 : RE fundus of resolved SO

DISCCUSION

Although the exact incidence figure of SO is difficult to determine, previously it was reported 0.1% to 0.3% following accidental ocular trauma and 0.02 % following ocular surgery.[3] Now this figure of SO has changed and it is found more in cases after intraocular surgeries, particularly vitreoretinal surgeries[4] where the risk is double (0.06%) as compared to preveous reports. It is because of improvement in management of ocular injuries and increased number of intraocular surgeries.

In severely injured eye or retained IOFB, the chances of SO are high. It may occur in 90% of patients within one year, in 80% within 3 months and in 65% within 2 months of ocular injuries [3]. The history of ocular injury plays a key role in the diagnosis of SO. Histopathology is specific investigative tool to differentiate SO from other granulomatous uveitis particularly Vogt-Koyanagi-Harada syndrome, in which choriocapillary is affected. But only 20% of clinically diagnosed cases are confirmed histologically.

Hence, a clinical diagnosis of SO is usually made on the basis of history of ocular injuries associated with early clinical and other features of granulomatous panuveitis.

In our case, SO developed after 9 weeks of injury with typical clinical features.[1,3,5]

Early detection of SO is important for prompt, proper and adequate systemic immunosuppressive treatment and ultimate better visual prognosis as shown in National Eye Institute series[1,5] and in our case.

Untreated sympathetic ophthalmia is a blinding ocular complication of ocular injury. This condition can be prevented by proper surgical repair combined with oral corticosteroid and evisceration or enucleation when indicated. Enucleation or evisceration is indicated mainly in severely traumatized or sightless eye but there is controversy regarding procedure of choice between these methods. In a recent retrospective[6] evaluation in 217 patients, it was concluded that the procedure of choice may be evisceration if primary repair can't be performed or for patients who have panophthalmitis. There is also controversy regarding it's timing. However the chances of SO is almost absent if it is performed within 10 days following trauma. Once SO is diagnosed, either above surgical treatment (evisceration and enucleation) is not effective and systemic corticosteroid or cytotoxic is required.

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