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A Case Report of Sympathetic Ophthalmitis

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Introduction

William Mackenzie provided the first full clinical description of the disease coining the term Sympathetic Ophthalmitis(1)

Sympathetic Ophthalmitis is a rare bilateral non necrotising granulomatous uveitis.(2)

This occurs after perforating eye injury or ocular surgical procedure to one eye. The injured eye is exciting eye and the fellow eye developing inflammation days to years later is sympathising eye.

Although rare, Sympathetic Ophthalmitis remains an important public health problem because it can cause bilateral blindness.

Time from injury to onset varies greatly ranging from 1 week to 66 years,(3) within 1 month in 17% cases, within 3months in 50% cases, within 6 months in 65% cases, within first in after injury in 90% cases.

Incidence ranges from 0.2% to 0.5% following injury and 0.01% following intraocular surgery.(4) incidence has reduced in recent years due to meticulous repair of injured eye utilising microsurgical techniques and use of potent steroids.

The reason for naming this disease Sympathetic Ophthalmitis remains obscure. One possible explanation is that the sympathetic pathways (optic nerve and optic chiasma) may be the pathway from the inciting eye to the sympathising eye.(5) Other is the role of immune dysregulation as an etiology. There appears to be cell mediated immune response directed against ocular self antigens found on photoreceptors, RPE or choroidal melanocytes.

No racial or age predisposition. Incidence is equal in male and female after surgery. Disease occurs most frequently in males after trauma. This likely reflects difference in frequency of ocular injury between genders and more often after non surgical trauma(6).

Case Report

A 45 year old female patient came to Ophthalmology OPD with chief complaints of dragging type of pain in left eye since 20days. Diminution of vision for near since 20 days.

No history of trauma to left eye

History of trauma to right eye with stick 10 years ago while working in the fields, since then she developed gradual progressive painless diminution of vision in right eye. No other ocular complaints. No history of usage of glasses. No known systemic illness.

Detailed ocular examination revealed

Visual acuity in right eye counting fingers 1 metre and left eye 6/24.
BCVA – not improving
Extra ocular movements free and full range in all directions.
Right eye – 15 degrees exotropia taking alternate fixation.
Central corneal nebular opacity
Iridodonesis
Aphakia
Left eye – fine keratic precipitates in inferior cornea
Grade 1 anterior chamber cells.
Intraocular pressure with applanation tonometer: right eye 18mm hg and left eye 24mm hg.
Dilated ophthalmic examination revealed :
Right eye – 0.3 CDR, disc margins well defined, pale disc
Arteriolar attenuations
Hyperpigmented lesions in periphery and midperiphery in all the quadrants.
Dull foveal reflex present
Brown colour solid mass and white fluffy material in inferior part of retina suggestive of nucleus and cortical matter of lens respectively.
Left eye – 0.3 CDR, disc margins well defined.
Dilated tortuous vessels noted
Vitreous haze suggestive of vitritis.
Dull foveal reflex present.

Result

Basing on history and ocular findings the diagnosis of left eye Sympathetic Ophthalmitis was made. Patient was prescribed oral steroids 40mg for 1 week initiated and tapered gradually over a period of 5 weeks along with topical antibiotic steroid eye drops started hourly and tapered over a period of 6 weeks and cycloplegic eye drops bedtime for 3 weeks. Thereafter gradual improvement in visual acuity in left eye noted and decrease in number of cells and vitreous haze was documented.

Discussion

Sympathetic Ophthalmitis also known as sympathetic uveitis is thought to be a delayed hypersensitivity immune response to uveal antigens.

It may manifest with a spectrum of clinical findings ranging from mild uveitis to severe bilateral granulomatous panuveitis.[2]

Etiology and pathophysiology remains unclear, largely thought to be autoimmune in nature and that the injured eye and contralateral eye demonstrate similar pathology suggesting involvement of autoimmune response.[7]

Inflammation is comprised of T lymphocytes that switch from a predominantly composition of CD4+ helper T cells in the early stage of disease to a later predominance of CD8+ cytotoxic T cells.[8]

B cells are identified in less than 5-15% of choroidal infiltrate[9]

Retina and the choriocapillaries are spared in the inflammatory process.[4]

Dalen-fuchs nodules are a feature of Sympathetic Ophthalmitis seen in 35% cases.[10] they are primarily located in choroid beneath RPE or under neuro retina.

They undergo development in which they are primarily composed of macrophages early on but later may be composed of depigmented or degenerated RPE and a small number of lymphocytes.[11]

Genetic predisposition to the development of Sympathetic Ophthalmitis has been proposed with focus on HLA. HLA A11 antigen expression has been linked to patients with Sympathetic Ophthalmitis as well as HLA DRB1*04, HLADQA1*03, HLADQB1*04.[12]

Untill recently accidental penetrating ocular trauma was the classic most common precipitating event for Sympathetic Ophthalmitis. These days ocular surgery particularly vitreoretinal surgeries has emerged as major risk for developing Sympathetic Ophthalmitis. Non perforating ocular procedures like irradiation, laser therapy have been associated.[9]

No organism has ever been consistently isolated from eyes with Sympathetic Ophthalmitis and has never been incited in animal models following injection of an infective agent.[13]

Clinical features reported are insidious onset blurring of vision, pain, epiphora, photophobia in sympathising non injured eye accompanied by anterior segment signs such as conjunctival injection, granulomatous anterior chamber reaction, mutton fat keratic precipitates on corneal endothelium. Iris may thicken from lymphocytic infiltration, formation of posterior synechiae. Raise in IOP secondary to trabecular meshwork blockage from inflammatory cells.

Posterior segment features include vitritis, retinal vasculitis, choroiditis, papillitis, serous retinal detachment, severe cases can range up to optic nerve swelling.

Sequelae of inflammation are quite variable depending on severity of ocular inflammation and the therapy initiated. Can range from secondary glaucoma, cataract, retinal atrophy or optic atrophy, subretinal fibrosis, CNVM, to phthisis bulbi in delayed cases.

Diagnosis of Sympathetic Ophthalmitis is primarily based on patient history and clinical findings. 20% cases diagnosis is confirmed by histology.[14]

Other granulomatous uveitis such as VKH, sarcoidosis, tuberculosis, posterior syphilis should be considered and ruled out with appropriate investigations.

Conclusion

Sympathetic Ophthalmitis is a rare, potentially visually devastating bilateral panuveitis following surgical or non surgical penetrating injury to one eye.

It can be prevented before its occurrence or can be treated if diagnosed early.

Treatment is primarily medical and the mainstay is systemic immunomodulatory therapy with adjunctive cycloplegics.

Clinical criteria for response to therapy incorporates the inflammatory response, if there is significant decrease in inflammation then slow tapering of corticosteroids initiated.

Steroid sparing immunomodulators are advantageous in steroid resistant or in patients having intolerable side effects.

Enucleation the only undisputable way of preventing Sympathetic Ophthalmitis is generally recommended within 2 weeks post injury.[13,15]

However the preference for evisceration over enucleation is increasing with advancements in techniques and greater perceived benefits i.e superior functional and cosmetic outcomes.

Both enucleation and evisceration however seems unnecessary these days due to relative better availability of immunomodulators. These patients needs strict followup and periodic systemic evaluation for early identification and treatment of complications to immunomodulatory therapy.

Early excision of injured eye is best prophylaxis when there is no chance of saving usefull vision.

High index of suspicion is vital to ensure early diagnosis and initiation of treatment thereby allowing good final visual acuity in most patients.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. The patient has given their consent for all the clinical information to be reported in the journal. However their name and initial will not be published.

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