Editorial

Inflammatory Glaucoma

Banu S

Introduction

This is a brief overview on inflammatory glaucoma / uveitic glaucoma that causes a persistent or recurrent elevation of intraocular pressure (IOP) as a result in characteristics in anatomical and physiological changes of primary open angle glaucoma. The paper highlights its nature of anatomical & physiological changes, etiology, disease process, diagnosis and treatment in particular. In addition, this article is a modest attempt to explain the need of careful history taking, appropriate treatment, and regular follow-up of Uveitic glaucoma cases to slow down its progress in deed.

Glaucoma represents a group of diseases defined by a characteristic optic neuropathy that is consistent with excavation and undermining of the neural and connective tissue elements of the optic disc and by the eventual development of distinctive patterns of visual dysfunction.[1]The global prevalence of glaucoma for population aged 40-80 years, is 3.54%. In 2013, the number of people (aged 40-80 years) with glaucoma worldwide was estimated to be 64.3 million, increasing to 76.0 million in 2020 and 111.8 million in 2040.[2]

Inflammatory Glaucoma

Inflammatory glaucoma is known as uveitic glaucoma, in which ocular inflammation causes a persistent or recurrent intraocular pressure (IOP) elevation resulting in anatomical and physiological changes characteristic of primary open angle glaucoma. The anatomical changes include progressive optic nerve cupping with corresponding retinal nerve fiber layer loss. When anatomical changes progress beyond the physiological reserve of the optic nerve, the visual field defects become detectable. Beer and Die (1813) first reported the association of uveitis and glaucoma, describing it as arthritic iritis.[3] Glaucoma is seen in about 20% of the patients with uveitis. Unilateral glaucoma should raise the suspicion of an inflammatory glaucoma.[4]The overall prevalence of glaucoma in eyes with uveitis varies from 10 to 20%, but it is much more common in chronic uveitis and can be as high as 46%.[5]

Etiology

The etiology of uveitis varies among different ethnicities and even among regions of the same country.[6] Higher rates are reported in those with rheumatoid arthritis-associated iridocyclitis, fuchs heterochromic iridocyclitis (27%), sarcoidosis (34%), herpes simplex keratouveitis (54%), zoster uveitis (38%) , lyme-associated uveitis, cancer-associated uveitis , juvenile idiopathic arthritis (JIA) (12–35%), behcet's disease, pars planitis, sympathetic ophthalmia and syphilis.[7]

Correspondence to:
Dr. Sharmin Banu
Assistant Professor, Department of Ophthalmology
US-Bangla Medical College, Rupgonj, Narayangonj.
E-mail: sharminoph@gmail.com

Disease process

The mechanisms that determine an IOP increase in uveitic glaucoma are diverse and complex; many are often present simultaneously in the same patient. Open-angle glaucoma occurs as a result of mechanical obstruction of the trabecular meshwork by inflammatory cells, proteins, debris, fibrin, or inflammatory precipitates. Additionally direct inflammation of the trabecular meshwork and/or the effect of corticosteroids on the trabecular meshwork may contribute to the open-angle mechanism of uveitic glaucoma.[7,8]Secondary angle closure can result from synechial closure, neovascularization of the anterior chamber angle, or seclusion pupillae with subsequent appositional angle closure.[9]

Diagnosis

Symptoms with acute iridocyclitis may include blurred vision, ocular pain, brow ache and other ocular disturbances like photophobia and colored haloes. The cornea may reveal band keratopathy, epithelial dendrites or stormal scarring from herpetic infections. The iris should be examined for evidence of stromal atrophy, nodules, posterior synechiae, peripheral anterior synechiae and neovascularization of iris. The lens may have a pigmentation of the anterior capsule and the posterior subcapsular opacification may be due to uveitis or to chronic corticosteroid therapy. Gonioscopy must be performed to detect the presence of peripheral anterior synechiae and to assess the degree of angle closure. The posterior segment must be examined, paying particular attention to the optic nerve, to document the morphological changes consistent with glaucoma. Other possible posterior segment findings include cystoid macular edema, retinitis, perivascular sheathing, choroidal infiltrates or retinal detachment[4].

Treatment

Treatment of uveitic glaucoma is aimed at controlling intraocular inflammation and elevated IOP, as well as treating any underlying systemic disease.[8]If medical management fails to control IOP,surgery is the next step. About 30% of eyes with uveitic glaucoma may require surgery.[10] Preoperatively, although good control of intraocular inflammation for a number of months is ideal, filtration surgery rarely is an elective procedure, and a regimen of preoperative topical or systemic corticosteroid treatment (e.g., 0.5 to 1 mg/kg/day of oral prednisolone) is

useful to reduce intraocular inflammation and the inflammatory cells in the conjunctiva.[7]

Conclusion

Uveitic glaucoma varies extensively in presentation and can be clinically challenging to treat. Successful management of uvetic glaucoma depends on recognition of the uveitis syndrome and clarification of the mechanisms contributing to it. Careful history taking, appropriate treatment, regular follow-up can be helpful to slow down or preventvision loss and other complications.

References

1. American Academy of Ophthalmology, Glaucoma 2014 - 15, Basic and Clinical Science Course Section 10, San Francisco.\
2. Tham, Y., Li, X., Wang, T.Y., Quigley, H.A., Aung, T. and Cheng, C.Y., 'Global prevalence of glaucoma and projections of glaucoma burden through 2040', Ophthalmology. 2014, 121. 2081- 2090.
3. Beer, G.J. and Die, V.D., 'Teaching of eye diseases', Vienna, 1813. 1. 633.
4. Bodh, S.A., Kumar,V., Raina, U.K., Ghosh, B. and Thakar , M., 'Inflammatory glaucoma', Oman journal of ophthalmology. 2011. 4 (1). 3-9.
5. Netland, P.A. and Denton, N.C., 'Uveitic glaucoma', Contempophthalmol, 2006, 5. 1- 6.
6. Shimizu, A., Maruyama, K., Yokoyama, Y., Tsuda, S., Ryu, M. and Nakazawa, T., 'Characteristics of uveitic glaucoma and evaluation of its surgical treatment', Clinical Ophthalmology, 2014, 8. 2383- 2389.
7. Sung, V.C. and Barton, K., 'Management of inflammatory glaucoma', Current opinion in ophthalmology, 2004, 15 (2). 136-140.
8. Siddique, S.S., Suelves, A.M., Baheti, U. and Foster, C.S., 'Glaucoma and uveitis', Survey of Ophthalmology, 2013. 58 (1). 1-10.
9. Francisco, J., Negrete, M., Montanes,J.M., Martinez, P.H. and Robolleda, G., 'Current approach in the diagnosis and management of uveitis glaucoma' , Biomed research international, 2015. 1- 13.
10. Kalin-Hajdu, E., Hammamji, K., Gagné, S. and Harasymowycz, P., Outcome of viscodilation and tensioning of Schlemm's canal for uveitic glaucoma. Canadian Journal of Ophthalmology,2014, 49(5).414-419.