Editorial

More about Retinal disorders

Dear Friends

Optic neuritis is the inflammation of the optic nerve, which leads to complete or partial loss of vision. The incidence has been reported to be ranging from 1.4 to 6.4 per 100,000 populations.[1,2] Female preponderance has been reported. Majority of the cases are idiopathic and among the known causes, demyelination associated with multiple sclerosis has been found to be the most common.[3] Optic neuritis is characterized by a triad of symptoms such as loss of vision, eye pain, and dyschromatopsia.[4] Optic coherence tomography and visual evoked potential detects the degree of axonal loss and myelin repair. The most reliable study to date regarding the long-term management of optic neuritis is the optic neuritis treatment trial.[5] One significant conclusion from the trial was better response, faster recovery, and less recurrence after administration of intravenous steroids. Hence, intravenous corticosteroid followed by oral therapy forms the mainstay of treatment currently. Although clinical features of optic neuritis are characteristic in other population group, not much exist from the Indian population. Saxena *et al*., in this issue, has published a study related to optic neuritis in Indian population. This is a prospective study involving follow-up of patients for a period of 3 years. The major findings of this study include unilateral presentation with nearly half of the study population exhibiting papillitis; a lesser proportion with idiopathic and optic neuritis related to multiple sclerosis had good visual outcome. Although the study is limited by the fact that automated perimetry was not performed on study participants, it has shed light on the fact that, Indians are clinically diverse from other population in terms of clinical profile of optic neuritis. Although an earlier study[6] had indicated similar findings, this study reiterated the fact.

Of the various retinal vascular disorders, diabetic retinopathy is the most common and retinal vein occlusion (RVO) ranks the second. Although the exact etiology of RVO is unknown, it mainly follows a thrombotic event. Studies have shown that hyperhomocystinemia is a risk factor for thrombosis.[7] A recent meta-analysis of data[8] from 600 patients compared with 700 controls has concluded that RVO is associated with elevated plasma homocysteine levels, although studies with controversial results do exist.[9,10] A case-control study by Wadani *et al*., published in this issue, evaluates the above proposed hypothesis and found that hyperhomocystinemia is a risk factor for RVO. Of the various treatment options available for RVO, a paradigm shift from native procedures to the use of intravitreal injections of bevacizumab, antiangiogenesis agent, and triamcinolone, a potent corticosteroid, has been envisaged. Although, several studies from the West have established the efficacy of these agents in the management of RVO,[11-14] the obtained data is limited due to the fact that patients were followed only for a short time posttreatment. Even, a very recent systematic review of eight studies by Ford *et al*.,[15] regarding the management of macular edema in patients with RVO, concluded that although these agents are very effective, most of these studies followed up patients only for a short-term (maximum 12 months). Demir *et al.* compared intravitreal triamcinolone and bevacizumab and found significant improvement in the visual acuity and central macular thickness with both. Although the study is limited by a small sample size and being retrospective in nature, some of these patients were followed up for a longer time interval ranging between 30 and 40 months, and hence, establishing the long-term safety of these agents. The authors suggest that the intravitreal bevacizumab may be considered superior than triamcinolone due to a better safety profile of the former. I am sure this issue will provide newer insights on a few retinal disorders and their management. This issue would definitely rule out all your visual perception pathology and keep your entire retina fully occupied.

Happy reading!!!

Sundaram Natarajan

Editor, Indian Journal of Ophthalmology,
Chairman, Managing Director, Aditya Jyot Eye Hospital Pvt Ltd,
Wadala (W), Mumbai, Maharashtra, India.
E-mail: editor@ijo.in

References

1. Toosy AT, Mason DF, Miller DH. Optic neuritis. Lancet Neurol 2014;13:83-99.
2. Roshini S, Madat D, Khushbu C, Subodh A. An overview of optic neuritis. Int Res J Pharm 2011;2:49-53.
3. Fisher JB, Jacobs DA, Markowitz CE, Galetta SL, Volpe NJ, Nano-Schiavi ML, *et al*. Relation of visual function to retinal nerve fiber layer thickness in multiple sclerosis. Ophthalmology 2006;113:324-32.
4. Menon V, Saxena R, Misra R, Phuljhele S. Management of optic neuritis. Indian J Ophthalmol 2011;59:117-22.
5. The clinical profile of optic neuritis. Experience of the optic neuritis treatment trial. Optic Neuritis Study Group. Arch Ophthalmol 1991;109:1673-8.

6. Jain IS, Munjal VP, Dhir SP, Gangwar DN. Profile of optic neuritis in Chandigarh and surrounding areas. Indian J Ophthalmol 1980;28:195-200.
7. Karia N. Retinal vein occlusion: Pathophysiology and treatment options. Clin Ophthalmol 2010;4:809-16.
8. Cahill MT, Stinnett SS, Fekrat S. Meta-analysis of plasma homocysteine, serum folate, serum vitamin B(12), and thermolabile MTHFR genotype as risk factors for retinal vascular occlusive disease. Am J Ophthalmol 2003;136:1136-50.
9. Di Crecchio L, Parodi MB, Sanguinetti G, Iacono P, Ravalico G. Hyperhomocysteinemia and the methylenetetrahydrofolate reductase 677C-T mutation in patients under 50 years of age affected by central retinal vein occlusion. Ophthalmology 2004;111:940-5.
10. McGimpsey SJ, Woodside JV, Bamford L, Gilchrist SE, Graydon R, McKeeman GC, *et al.* Retinal vein occlusion, homocysteine, and methylene tetrahydrofolate reductase genotype. Invest Ophthalmol Vis Sci 2005;46:4712-6.
11. Wu WC, Cheng KC, Wu HJ. Intravitreal triamcinolone acetonide vs bevacizumab for treatment of macular oedema duo to central retinal vein occlusion. Eye (Lond) 2009;23:2215-22.
12. Tao Y, Hou J, Jiang YR, Li XX, Jonas JB. Intravitreval bevacizumab vs triamcinolone acetonideformacular oedema for due to central retinal vein occlusion. Eye (Lond) 2010;24:810-5.
13. Ding X, Li J, Hu X, Yu S, Pan J, Tang S. Prospective study of intravitreal triamcinolone acetonide versus bevacizumab for macular edema secondary to central retinal vein occlusion. Retina 2011;31:838-45.
14. Guthoff R, Meigen T, Hennemann K, Schrader W. Comparison of bevacizumab and triamcinolone for treatment of macular edema secondary to central retinal vein occlusion--a matched-pairs analysis. Ophthalmologica 2010;224:126-32.
15. Ford JA, Clar C, Lois N, Barton S, Thomas S, Court R *et al*. Treatments for macular oedema following central retinal vein occlusion: systematic review. BMJ Open 2014;4:e004120.

Access this article online

Quick Response Code:

Website: www.ijo.in

DOI: 10.4103/0301-4738.130427