

AN OVERVIEW ON OPTIC NEURITIS

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ABSTRACT

Optic neuritis is a serious condition which typically involves the young adult population. Years ago it was thought that one third of optic neuritis patients would go on to have neurological symptoms from multiple sclerosis (MS). More recent studies show its association with vaccination & ethambutol treatment in tuberculosis. The present article is alert to the symptoms of optic neuritis, its diagnostic criteria, association with various serious diseases and some herbal treatment to counteract associated symptoms.

KEYWORDS: Optic neuritis, Multiple sclerosis, Tuberculosis.

INTRODUCTION

Optic neuritis is the inflammation of the optic nerve that may cause a complete or partial loss of vision. The optic nerve comprises axons that emerge from the retina of the eye and carry visual information to the occipital cortex of the brain to be processed into vision. Inflammation of the optic nerve causes loss of vision usually due to the swelling and destruction of the myelin sheath covering the optic nerve. Direct axonal damage may also play a role in nerve destruction in many cases. Most of the cases are idiopathic in nature; however, it could be associated with demyelinating lesions, of which multiple sclerosis (MS) is the most common cause. Other less common etiologies include infectious and para-infectious causes, inflammatory and para vaccination immunological responses¹. (figure 1)

EPIDEMIOLOGY

Optic neuritis typically affects young adults ranging from 18–45 years of age, with a mean age of 30–35 years². The annual incidence of optic neuritis ranges from 1.4 to 6.4 new cases per 100,000 populations^{3,4}. There is a strong female predominance. The annual incidence is approximately 5/100,000, with a prevalence estimated to be 115/100,000.⁵

SIGNS & SYMPTOMS

Optic neuritis typically presents with a triad of symptoms like loss of vision, dyschromatopsia and eye pain. The initial attack is unilateral in 70% of adult patients and bilateral in 30%. The mean age of onset of optic neuritis is in the third decade of life, but can occur from the first

to the seventh decades. The visual loss may be subtle or profound. Complete loss of vision may be caused by a single plaque. In some cases the vision may be 20/20 with the only symptoms being blurred vision on exertion or other isolated symptoms⁶. The rate of visual decline varies. Visual loss may occur over hours (rarely) to days (most commonly). The nadir is usually about 1 week after the onset⁷.

DIAGNOSIS

It is particularly based on the visual blurring symptoms & ophthalmoscopy & finally on MRI²⁵. (Table 1)

OPTIC NEURITIS ASSOCIATED WITH VARIOUS DISEASES

(A)Optic Neuritis With Multiple Sclerosis

However a small group of patients will have a poor visual outcome after a single attack and progressive visual loss is seen in MS. The pathogenesis of demyelinating ON is thought to involve an inflammatory process that leads to activation of peripheral T-lymphocytes which cross the blood–brain barrier and cause a delayed type hypersensitivity reaction culminating in axonal loss. Clinical recovery reflects the combined effects of demyelination with conduction block and axonal injury on the one hand, remyelination with compensatory neuronal recruitment on the other. However, irreversible axonal damage occurs early in the disease process. A study using ocular coherence tomography (OCT) demonstrated that axonal injury is common in ON⁸ and observed retinal nerve fibre layer (RNFL) thinning in 74% of individuals within 3 months

of acute ON. In this and another cross-sectional study of MS patients with ON⁹. RNFL was significantly reduced in the affected eye when compared with fellow eyes or disease-free controls. These and other studies have correlated RNFL thinning with impaired visual function¹⁰. OCT can be employed to monitor such progressive axonal loss in both primary and secondary progressive MS¹¹.

(B) Optic Neuritis With Ethambutol

Ethambutol has been used to treat TB since the 1960s. The potential for visual impairment was recognized soon after its introduction¹². The original formulation was a racemic mixture. However, when it was discovered that the L-form was predominantly responsible for its toxicity, and the D-form for its therapeutic effects, the L-form was withdrawn. Despite this, cases of irreversible visual loss have been reported in the literature and some authors have even gone on to suggest that ethambutol should not be used routinely to treat TB^{13,14}

Ethambutol causes loss of visual acuity, colour vision and visual field. The occurrence of ocular toxicity is dose related, loss of vision most likely to occur in patients receiving 25 mg/kg/day or more.

However, vision loss has been documented in approximately 1% of patients receiving the recommended therapeutic dose of 15 to 25mg/kg/day^{15,16,17}. This rarely occurs before the patients have been on treatment for 2 months, with 7 months being the average. Two Patients with impaired renal function from renal tuberculosis may be more prone to ethambutol-associated optic neuropathy; perhaps because ethambutol depends on the kidneys for excretion^{18,19}. It is also important for the clinician to be aware that there are reports of rapid onset, severe, bilateral visual loss despite treatment with therapeutic doses of ethambutol^{20,21}. Patients taking ethambutol should be instructed to discontinue the drug immediately at the onset of any visual symptoms and seek medical consult.

(C) Optic Neuritis With Vaccination

Two types of vaccine for meningitis are distributed in Spain: A) Bivalent vaccines which include non-conjugated purified capsular bacterial polysaccharides of serogroups A and C of *Neisseria meningitidis*, and B) Conjugated vaccines vis-à-vis *Neisseria meningitidis*, C serotype, by conjugation of the oligosaccharide of the *Neisseria meningitidis* capsule through covalent link with a carrier protein (diphtheria or tetanus toxoid) for increasing the immunogenic capacity of the vaccine. Optic neuritis (ON) can leave important visual sequels because many of these processes can be bilateral. Within etiological diagnosis there is a group of ON called post-

vaccine²². Which occur in the presence of diverse viral and bacterial agents^{23,24}.

TREATMENT

In most cases, visual functions return to near normal within eight to ten weeks, but they may also advance to a complete and permanent state of visual loss. Therefore, systemic intravenous treatment with corticosteroids, which may quicken the healing of the optic nerve, is often recommended, but it does not have a significant effect on the visual acuity at one year, when compared against placebo. Intravenous corticosteroids have also been found to reduce the risk of developing MS in the following two years in those patients who have MRI lesions; but this effect disappears by the third year of follow up²⁶.

Paradoxically it has been demonstrated that oral administration of corticosteroids in this situation may lead to more recurrent attacks than in non-treated patients (though oral steroids are generally prescribed after the intravenous course, to wean the patient off the medication). This effect of corticosteroids seems to be limited to optic neuritis and has not been observed in other diseases treated with corticosteroids²⁷.

Very occasionally, if there is concomitant increased intracranial pressure the sheath around the optic nerve may be cut to decrease the pressure. When optic neuritis is associated with MRI lesions suggestive of multiple sclerosis (MS) then general immunosuppressive therapy for MS is most often prescribed (IV methylprednisolone may shorten attacks; initial only oral prednisone may increase relapse rate). The above overall discussion regarding the treatment following the MRI & management protocol is followed¹. (Figure 2)

PROGNOSIS

The pain will go away, usually in a few days. For visual recovery is usually good. The majority of patients (65-80%) recover visual acuity of 20/30 or better^{25, 29}. The vision problems will improve in the majority (92%) of patients. There are rare patients who have continued progressive loss of vision. Even in the 92% that improve, often they do not return completely to normal. Patients may be left with blurred, dark, dim, or distorted vision. Frequently colors look different or "washed out." Visual recovery usually takes place over a period of weeks to months, although both earlier and later improvement is possible.

Late variations in vision are common, often associated with exercise or taking a hot shower or bath. This is known as Uhthoff's phenomena and is probably related to damage to the myelin coating. Patients who notice this problem are not more likely to get worse.

Optic neuritis can recur involving the same eye, the other eye or other parts of the central nervous system (brain and spinal cord). This may result in recurrent episodes of decreased or loss of vision or problems with weakness, numbness or other signs of brain involvement. An MRI scan can give us to give a rough guess as to the likelihood of recurrence.

It will not completely exclude the possibility of future episodes or guarantee that they will happen. Other testing techniques are sometimes used to confirm the suspicion of optic neuritis. These may include visual evoked potentials (a test where you are shown a checkerboard of light and signals are recorded from electrodes on your scalp) that can show a delay in conduction due to the damage to the myelin (Neuro Ophthalmology North American Society).

HERBAL TREATMENT

The Ayurvedic treatment of optic neuritis is aimed at controlling the pain, treating the inflammation of the optic nerve and treating immune dysfunction of the body. Medicines like Triphala-Guggulu, Yograj-Guggulu, Punarnavadi-Guggulu, Maha-Rasnadi-Guggulu, Rasnadi-Qadha, Vat-Gajankush-Ras, Maha-Vat-Vidhwans-Ras, Vish-Tinduk-Vati, Dashmoolarishta and Nirgundi (Vitex negundo) can be used to treat the pain in the eyes. Medicines such as Kaishor-Guggulu, Panch-Tikta-Ghrut-Guggulu, Triphala-Ghrut, Panch-Tikta-Ghrut, Tapyadi-Loh, Saptamrut-Loh and Ekang-Veer-Ras are used to treat the inflammation in the optic nerve²⁸.

Herbal medicines which can be used in optic neuritis are Amalaki (*Emblica officinalis*), Haritaki (*Terminalia chebula*), Behada (*Terminalia bellerica*), Tulsi (*Ocimum sanctum*), Shatavari (*Asparagus racemosus*), Punarnava (*Boerhaavia diffusa*), Rasna (*Pluchea lanceolata*), Guduchi (*Tinospora cordifolia*), Deodar (*Cedrus deodara*), Erandmool (*Ricinus communis*), Gokshur (*Tribulus terrestris*), Apamarga (*Achyranthus aspera*), Guggulu (*Commiphora mukul*), Shallaki (*Boswellia serrata*), Kuchla (*Strychnos nuxvomica*) and Nirgundi²⁸.

The overall prognosis following optic neuritis is generally good, with most people regaining normal vision within 2 to 6 months. Ayurvedic medicines can help to improve the therapeutic response, reduce the duration of treatment and prevent recurrent episodes of this condition.

CONCLUSION

In light of above review there is a comprehensive study is require for counteracting the optic neuritis association with various neurodegenerative diseases & showed some herbal options for the treatment.

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TABLE 1 DIAGNOSTIC TEST

Name Of The Test	Outcome
Ophthalmoscopy	The optic disk becomes swollen in about one-third of people with optic neuritis.
Pupillary light reaction test	Pupils affected by optic neuritis don't constrict as much as healthy eyes do when stimulated by light.
Visually evoked potentials test.	This type of test is able to detect the slowing of electrical conduction resulting from damaged areas on nerves.
Magnetic resonance imaging (MRI) scan.	An MRI is also important to determine whether there are areas in your brain where the myelin has been damaged (lesions), which indicate a high risk of developing multiple sclerosis. An MRI also can help rule out tumors or other conditions that can mimic optic neuritis.
Blood tests	This test may help determine whether optic neuritis is caused by inflamed cranial arteries (cranial arteritis).

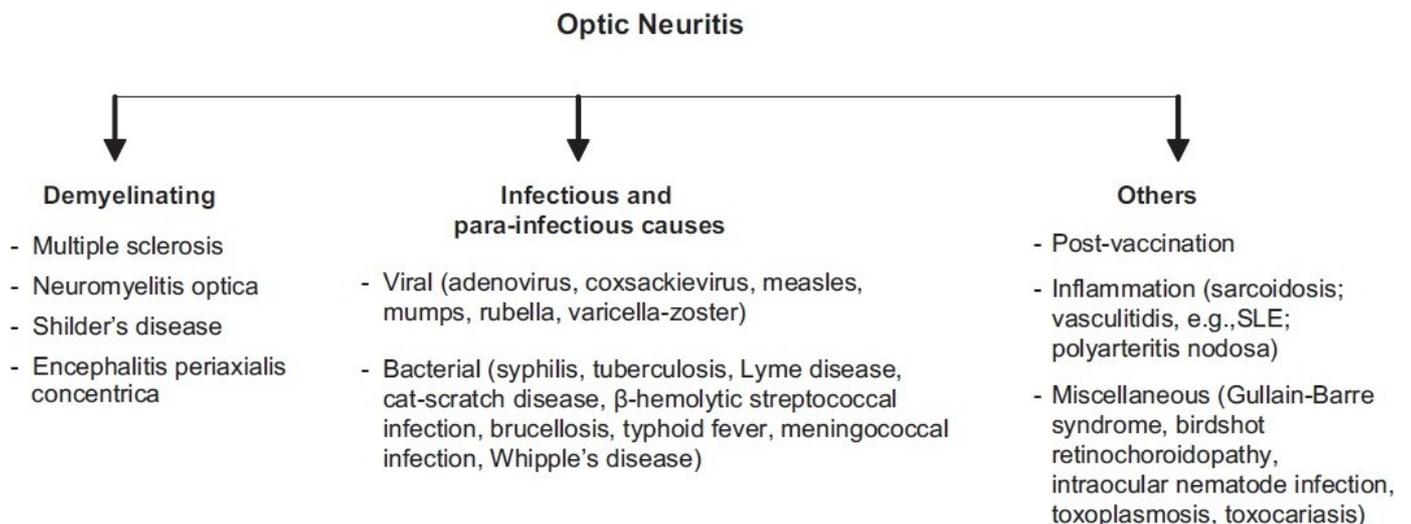


FIGURE 1 CAUSES OF OPTIC NEURITIS

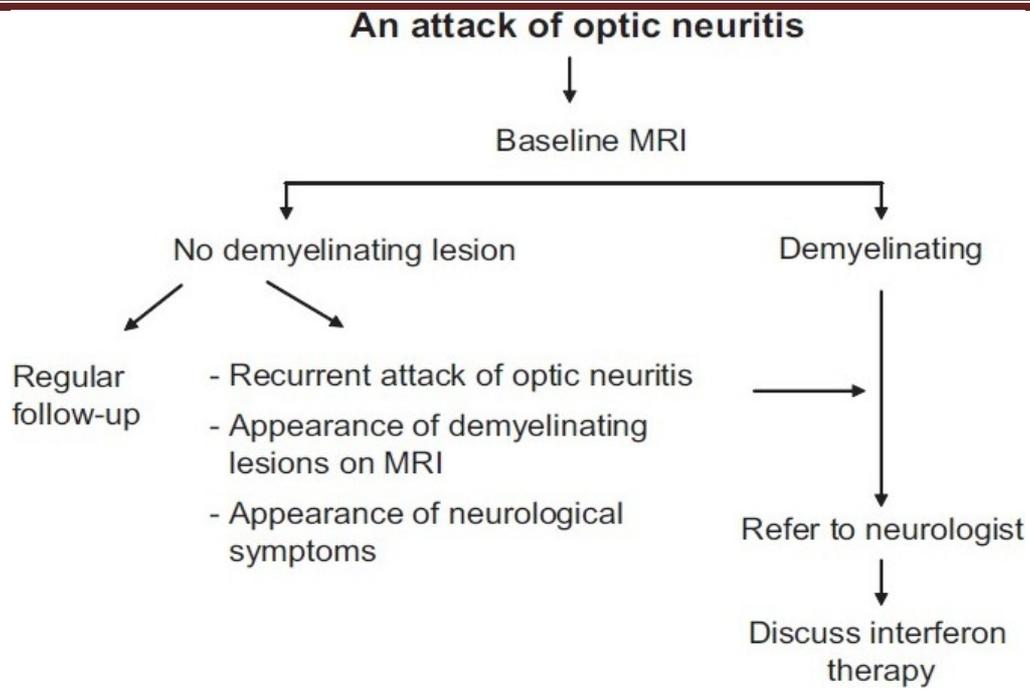


FIGURE 2 MANAGEMENT PROTOCOL FOR OPTIC NEURITIS PATIENT