

Clinical Profile and Short Term Visual Outcome of Optic Neuritis - 7 Years

Aadithya K. R.¹, Dr. Rehna Rasheed²

²Fellow in Advanced Clinical Optometry, Department of Ophthalmology, Amrita Institute of Medical sciences, Kochi, India

Abstract: ***Background:** Optic neuritis is a common clinical manifestation of central nervous system inflammation. Depending on the etiology, visual prognosis and the risk for recurrent injury may vary. Rapid and accurate diagnosis of optic neuritis may be critical for limiting vision loss, future neurologic disability, and organ damage. **Methods:** 45 patients were taken for the study. Patients with optic neuritis who consulted in Ophthalmology Department were obtained from electronic medical records. Data taken including age, sex, visual acuity and cause were assessed. **Results:** In our study of 45 patients, 33 (72.3%) were female and 12 (2.7%) were male. The mean age of those with optic neuritis was 36.91 ± 17.201 . The mean BCVA of the affected eye was 0.30 ± 0.6 , and the mean BCVA of the other eye was 0.0861 ± 0.41286 . 20 (30%) patients were affected binocularly. 43 were idiopathic, 8 were diagnosed with MOG, 4 patients were MS, 2 were Neuromyelitis Optica (NMO) and 1 patient were diagnosed with chronic relapsing inflammatory optic neuropathy (CRION) and infective. Most of the persons, 17 (26 %) were in the age group 31-40 years. 17 patients had done the test in which most of the persons, 4 (23.52%) had central scotoma. Retrobulbar (60% eyes) was more common than Papillitis (40 % of eyes). 26 of the 65 eyes (40 %) recovered visual acuity 20/20 or better. **Conclusion:** Most common cause of optic neuritis in our study was idiopathic. Optic neuritis was found to mainly affect in 31 – 40 age group, females were more affected. Central scotoma was the most common visual defect in optic neuritis. Diabetes mellitus was the associated co morbidity in optic neuritis. Visual prognosis of optic neuritis at 1 month is good in optic neuritis. There was significant improvement in visual acuity at 1 month. In our study Retrobulbar neuritis was more than Papillitis.*

Abbreviations: ON-Optic neuritis, FFA-Fundus Fluorescein Angiography, OCT-Optical Coherence Tomography, HFA-Humphrey Field Analyzer, VA-Visual Acuity, MS-Multiple Sclerosis, CNS-Central Nervous System, ONTT-Optic Neuritis Treatment Trial, MRI-Magnetic Resonance Imaging, VEP-Visually Evoked potential, NMO-Neuromyelitis Optica, RAPD-Relative Afferent Pupillary Defect, RNFL-Retinal Nerve Fiber Layer, MOG – Myelin oligodendrocyte glycoprotein

1. Introduction

Optic Nerve

The optic nerve, also known as the second cranial nerve or CNII, is a bundle of over 1 million nerve fibers that transmits sensory information for vision from the eye to the brain. Damage to the optic nerve can cause vision loss, depending on the location. It is an extension of the central nervous system and is unique in that it is the only cranial nerve that can be visualized clinically and leaves the cranial cavity.

Course

The optic nerve originates at the optic disk, a 1.5 mm diameter structure at the back of the eye, where fibers emerge from the eyeball. The nerve passes through the posterior orbit, the lamina cribrosa, and the bony optic canal to emerge intracranially on the front of the brain. At the optic chiasm, the optic nerve from each eye forms an X-shaped structure, with half of the fibers continuing on the same side of the brain and the remaining fibers crossing over to join those from the opposite eye. This arrangement is essential for producing binocular vision. The nerve fibers travel in optic tracts to various parts of the brain, with some leaving the optic tract without entering the lateral geniculate nuclei and entering the brain stem to determine pupil size.

Pathology

The optic nerve can be categorized into primary and secondary pathologies based on the cause. Primary pathologies result from direct or indirect trauma, such as concussion, avulsion, tear, contusion, or hemorrhage. Secondary pathologies may arise from edema, ischemia,

microvascular thrombosis, or nerve infarction. Damage to the optic nerve or its pathways to the brain causes vision loss, which doctors can identify by understanding the pattern of vision loss.

There are many different types of optic nerve disorders, including:

- Glaucoma is a group of diseases that are the leading cause of blindness in the United States. Glaucoma usually happens when the fluid pressure inside the eyes slowly rises and damages the optic nerve.
- Optic neuritis is an inflammation of the optic nerve. Causes include infections and immune-related illnesses such as multiple sclerosis. Sometimes the cause is unknown.
- Optic nerve atrophy is damage to the optic nerve. Causes include poor blood flow to the eye, disease, trauma, or exposure to toxic substances. Optic nerve head drusen are pockets of protein and calcium salts that build up in the optic nerve over time

The anatomy of the optic nerve makes it a sensitive marker for problems inside the brain. This nerve connects the back of each eyeball and its retina to the brain. In its short span between the brain and the eye, the optic nerve's whole surface is bathed in cerebral spinal fluid. However, even slight increases in the pressure of this fluid, from swelling of the brain, can compress the optic nerve around its whole circumference in a "choking" manner. Some important causes of increased pressure from cerebral spinal fluid and papilledema are brain tumors and brain infections, such as a brain abscess, meningitis or encephalitis. A significant proportion of people who are diagnosed with brain tumors have some evidence of papilledema. A pressure increase

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resulting from bleeding or from very high blood pressure also can cause papilledema.

Optic Neuritis

Optic neuritis, or inflammation of the optic nerves, is a frequent cause of acute optic nerve injury in children and adults. While optic neuritis is frequently associated with multiple sclerosis (MS), As a result, the prognosis and treatment of optic neuritis will vary depending on the etiology, the duration and severity of vision loss, prior injury, and the success of prior treatment. Optimal care of patients with optic neuritis therefore depends on rapid recognition, appropriately diagnosed studies, and the early institution of effective therapies. Multiple causes of optic nerve inflammation exist: autoimmunity, infection, granulomatous disease, paraneoplastic disorders, and demyelination. Rapid determination of the etiology of optic neuritis is important for implementing timely and appropriate treatment. In addition, understanding the cause of optic neuritis informs the visual prognosis, illuminates future health risks, and directs additional evaluation and treatments. Differentiating between various causes of optic neuritis, however, often requires a multifaceted evaluation that extends beyond a clinical history and neuro-ophthalmologic examination. Visual field perimetry and optical coherence tomography (OCT) MRI, serologic testing, and CSF analysis may help to focus the differential diagnosis or identify an alternative diagnosis. Therefore, an initial overview of the clinical presentation, examination findings, evaluation, and treatment of the patient with optic neuritis is warranted.

Etiology

- 1) Idiopathic In a large proportion of cases, the underlying causes are Unidentifiable
- 2) Hereditary optic neuritis (Lebers's disease)
- 3) Demyelination disorders are by far the most common causes of optic neuritis. These include multiple sclerosis, neuromyelitis optica (Devic's disease), and diffuse periaxial encephalitis of the schilder. About 70% of cases of established multiple sclerosis may develop optic neuritis.
- 4) Para-infectious optic neuritis is associated with various viral infections such as measles, mumps, chickenpox, whooping cough, and glandular fever.
- 5) Infectious optic neuritis may be sinus-related (with acute ethmoiditis) or associated with cat scratch fever, syphilis (during the primary or secondary stages), tuberculosis, Lyme disease, and cryptococcal meningitis in patients with AIDS.
- 6) Autoimmune disorders associated with optic neuritis include sarcoidosis, systemic lupus erythematosus, polyarteritis nodosa, Guillain-Barre syndrome, and Wegener's granulomatosis.
- 7) Toxic optic neuritis

Causes

The exact cause of optic neuritis is unknown. It's believed to develop when the immune system mistakenly targets the substance covering your optic nerve, resulting in inflammation and damage to the myelin.

The following autoimmune conditions are often associated with optic neuritis:

- Multiple sclerosis. Multiple sclerosis is a disease in which your autoimmune system attacks the myelin sheath covering nerve fibers in your brain. In people with optic neuritis, the risk of developing multiple sclerosis after one episode of optic neuritis is about 50% over a lifetime.
- Neuromyelitis optica: In this condition, inflammation affects the optic nerve and spinal cord. Neuromyelitis optica has similarities to multiple sclerosis, but neuromyelitis optica doesn't cause damage to the nerves in the brain as often as multiple sclerosis does. Still, neuromyelitis optica is more severe than MS, often resulting in a diminished recovery after an attack compared with MS.
- Myelin oligodendrocyte glycoprotein (MOG) antibody disorder: This condition can cause inflammation of the optic nerve, spinal cord, or brain. Similar to MS and neuromyelitis optica, recurrent attacks of inflammation can occur. Recovery from myelin oligodendrocyte glycoprotein (MOG) attacks is usually better than recovery from neuromyelitis optica.
- Infections: Bacterial infections, including Lyme disease, cat scratch fever, and syphilis, or viruses, such as measles, mumps, and herpes, can cause ON.
- Other diseases: Diseases such as sarcoidosis, Behcet's disease, and lupus can cause recurrent ON.
- Drugs and toxins: Some drugs and toxins have been associated with the development of optic neuritis. Ethambutol, used to treat tuberculosis, and methanol, a common ingredient in antifreeze, paints, and solvents, are associated with ON

Risk Factors

- 1) Age: Optic neuritis most often affects adults ages 20 to 40.
- 2) Sex: Women are much more likely to develop optic neuritis than men are.
- 3) Race: Optic neuritis occurs more often in white people.
- 4) Genetic mutation: Certain genetic mutations might increase your risk of developing optic neuritis or multiple sclerosis.

Anatomical Types

Optic neuritis can be classified into three anatomical types:

1) Papillitis

Papillitis, also known as optic neuritis, is characterized by inflammation and deterioration of the portion of the nerve known as the optic disc. Also referred to as the "blind spot", the optic disc is that portion of the optic nerve that enters the eye and joins with the nerve-rich membrane lining the eye (retina). The optic nerves are the pair of nerves (second cranial nerves) that transmit impulses from the retina to the brain. Individuals with papillitis experience loss of vision in one eye that may occur within several hours of onset. The severity of visual impairment may vary from case to case, ranging from a slight visual deficiency to a complete loss of light perception. In some cases, spontaneous recovery may occur. However, in other cases, permanent visual impairment may result if the underlying cause is not detected or treated. Papillitis may occur for unknown

reasons, after a viral illness, or due to or in association with a number of different underlying disorders or other factors.

2) Neuroretinitis

Neuroretinitis (NR) is defined as inflammation of the anterior optic nerve and peripapillary retina. It presents as a triad of vision loss, optic disc swelling, and macula exudates in the formation of stars. It is a term given to appearance but does not indicate a specific etiology. It is broadly categorized as idiopathic, idiopathic-recurrent, and cat scratch-disease neuroretinitis (CSD-NR). Neuroretinitis may also be categorized based upon the etiology: infectious vs. non-infectious. Idiopathic and idiopathic-recurrent neuritis are usually non-infectious. It is usually unilateral, but bilateral cases have been described, though they should lead the physician to consider other causes.

3) Retrobulbar Neuritis

Retrobulbar neuritis is a form of optic neuritis in which the optic nerve, which is at the back of the eye, becomes inflamed. The inflamed area is between the back of the eye and the brain. The optic nerve contains fibers that carry visual information from the nerve cells in the retina to the nerve cells in the brain. When these fibers become inflamed, visual signaling to the brain becomes disrupted, and vision is impaired.

Optic Neuritis Symptoms

Optic neuritis usually affects one eye. Symptoms might include:

- Pain: Most people who develop optic neuritis have eye pain that's worsened by eye movement. Sometimes the pain feels like a dull ache behind the eye.
- Vision loss in one eye: Most people have at least some temporary reduction in vision, but the extent of the loss varies. Noticeable vision usually develops over hours or days and improves over several weeks or months. Vision loss is permanent for some people.
- Visual field loss and side vision loss can occur in any pattern, such as central vision loss or peripheral vision loss.
- Loss of color vision. Optic neuritis often affects color perception. You might notice that colors appear less vivid than normal.
- Flashing lights. Some people with optic neuritis report seeing flashing or flickering lights with eye movements.

2. Diagnosis

The evaluation of the patient with optic neuritis begins with a careful medical history and examination. The ophthalmologist will likely perform the following eye tests:

- A routine eye exam. The eye doctor will check vision and the ability to perceive colors and measure side (peripheral) vision.
- Ophthalmoscopy: During this examination, the doctor shines a bright light into the eye and examines the structure at the back of the eye. This eye test evaluates the optic disc, where the optic nerve enters the retina of the eye. The optic disc becomes swollen in about one-third of people with optic neuritis.

- Pupillary light reaction test: The doctor may move a flashlight in front of the eyes to see how the pupils won't respond when they're exposed to bright light. If we have optic neuritis, the pupils won't constrict as much as pupils in healthy eyes would when exposed to light.
- Humphrey field analyzer (HFA): This test measures the peripheral vision of each eye to determine if there is any vision loss. Optic neuritis can cause any pattern of visual field loss. Visual field defects due to optic neuritis vary considerably. Therefore, the pattern of visual field loss is not specific to any subtype of optic neuritis. Diffuse or central visual field loss is the most frequent pattern observed in acute idiopathic optic neuritis and MS optic neuritis; altitudinal field loss may be more frequent in NMO optic neuritis than MS optic neuritis.
- Optical coherence tomography (OCT): This is a noninvasive imaging technology capable of identifying subtle optic nerve and retinal pathology. OCT frequently identifies peripapillary retinal nerve fiber layer thickening in acute optic neuritis that evolves into focal retinal nerve fiber layer and macular thinning. It reveals decreased vessel density in the peripapillary retina and macula following optic neuritis. In NMO, reduced peripapillary and parafoveal vessel density is observed independent of a history of optic neuritis and appears to correlate with visual function. In complex cases, OCT may be helpful to document associated retinal abnormalities or identify alternative diagnoses such as chorioretinitis, central serous chorioretinopathy, and acute macular neuroretinopathy. Summarizes the results of paraclinical testing that may impact the diagnosis of optic neuritis in the acute setting.
- Visual evoked potentials (VEP): VEP is a non-invasive technique to detect pathological changes in the visual system during optic neuritis and is used as a routine clinical test. Commonly used visual stimuli are flashing lights or patterns. The main interest in electrophysiological findings for optic neuritis focused on the so-called P100 latency and VEP amplitude. During the acute phase of the disease, a decrease in the VEP amplitude, caused by a conduction block of inflamed optic nerve fibers, is a common pathological feature. The depression of the VEP amplitude correlates with visual acuity. And is associated with the degree of atrophy. The prolongation of p100 latency is the main characteristic in the chronic phase and persists over years in up to 70% of patients who suffer from optic neuritis.
- Fundus fluorescein angiography (FFA): Fluorescein angiography is not routinely performed in the evaluation of optic neuritis and is often normal. Up to 25 percent demonstrate either dye leakage or paravenous sheathing. The findings may identify patients at somewhat higher risk of developing MS.
- Magnetic resonance imaging (MRI): An MRI scan uses a magnetic field and pulses of radio wave energy to make pictures of your body. During an MRI to check for optic neuritis, we might receive an injection of a contrast solution to make the optic nerve and other parts of our brain more visible on the images. An MRI is important to determine whether there are damaged areas

(lesions) in our brain. Such lesions indicate a high risk of developing multiple sclerosis. An MRI can also rule out other causes of visual loss, such as tumors. Lumbar puncture:

- Lumbar puncture is not an essential diagnostic test in optic neuritis but should be considered in atypical cases (e.g., those with bilateral presentation, < 15 years of age, or symptoms suggesting infection).
- Blood tests: A blood test is available to check for infections or specific antibodies. Neuromyelitis optica is linked to an antibody that causes severe optic neuritis. People with severe optic neuritis may undergo this test to determine whether they're likely to develop neuromyelitis optica. For atypical cases of optic neuritis, blood may also be tested for MOG antibodies.

Treatment

The administration of high doses of corticosteroids is the standard treatment for optic neuritis. In the Optic Neuritis Treatment Trial, IV methylprednisolone (1000 mg/d for 3 days), followed by oral prednisone (1 mg/kg/d for 11 days), accelerated visual recovery but failed to improve functional outcomes. Subsequent studies in patients with relapsing MS or optic neuritis have demonstrated that doses of corticosteroid equivalent to 1000 mg IV methylprednisolone administered IV or orally provide an equivalent therapeutic effect of accelerated recovery. IM, or subcutaneous adrenocorticotropic hormone (ACTH), is also approved for the treatment of acute optic neuritis and provides an alternative option for enhancing corticosteroid signaling. Lower doses of oral prednisone (1 mg/kg/d or less) should be avoided in cases of idiopathic optic neuritis, as an increased risk of relapse exists. Chronic treatment with low-dose oral prednisone, however, is important for the treatment of sarcoid optic neuritis and recurrent optic neuritis due to chronic relapsing inflammatory optic neuropathy. IV immunoglobulin (IVIg) and plasma exchange have been evaluated in patients with optic neuritis that is refractory to high-dose corticosteroid treatment. IVIg (2 g/kg) failed to improve contrast sensitivity or visual function in patients with acute optic neuritis or MS with refractory vision loss. Treatment response may have been limited because of the delayed administration of IVIg in both studies. In contrast, plasma exchange has resulted in improved visual outcomes in patients with corticosteroid-refractory optic neuritis and NMOSD optic neuritis. While the frequency of responders varied, the majority of patients with optic neuritis treated with plasma exchange had improvements in their visual function. Increased response to plasma exchange has been associated with male sex, lower baseline disability, rapid initiation of treatment, and shorter relapse duration.

Complications

Taking corticosteroids on a long-term basis can lead to side effects such as high blood sugar, weight gain, and bone problems that affect your whole body. Overall, corticosteroids won't likely lead to a better outcome than letting the condition run its course. However, in people with certain brain changes seen on MRI, intravenous steroids may help prevent future episodes of optic neuritis.

3. Material and Methods

Study Design

A hospital-based retrospective study was done in the department of ophthalmology at the Amrita Institute of Medical Science and Research Center over a period of 7 years (2017–2023).

4. Results

Out of 45 patients 73.3 % (33 patients) were female and 26.7 % (12 patients) were male. Most of the patients from the age group of 31 – 40 with mean age 36.91 ± 17.201 years. 11(24.4%) had diabetes mellitus, 8 (17.7 %) had hypertension and 3(6.6%) dyslipidemia and chronic kidney disease, 1 (2.2 %) had thyroid, 4(8.89 %) had chronic liver disease and asthma, 1(2.2%) had migraine. In out of 65 eyes 58 had blurring of vision, 1 had pain and 6 were both of them. 43 were idiopathic, 8 were diagnosed with MOG, 4 patients were MS, 2 were Neuromyelitis Optica(NMO) and 1 patient were diagnosed with chronic relapsing inflammatory optic neuropathy(CRION) and infective. In out of 45 patients 20 were bilaterally affected, 16 were left eye affected and 9 were right eye affected. In out of 20 patients 15 were idiopathic, 2 were diagnosed with MOG, 1 were CIS,NMO and autoimmune- ANCA. 65 eyes mean BCVA of affected eye is 0.6992 ± 0.6000 and the mean BCVA of fellow eye is 0.0861 ± 0.41286 . The mean of 1 month visual acuity is $.6992 \pm .62167$. There is a significant improvement in visual acuity at 1 month ($p < 0.001$). 17 patients had field test done in which 4 (23.52%) had central scotoma, 3 (17.64%) had scattered scotoma and normal, 2 (11.78%) had superior acute scotoma, 1 (5.89%) had inferior peripheral scotomas, 1 (5.89%) had nasal inferotemporal scotoma, 1 (5.89%) had gross depression, and 1 (5.89%) had hemianopic field. Out of 65 patients, 39 (60%) had retrobulbar neuritis, and 26(40 %) were Papillitis.

Distribution of Gender

Gender	Frequency	Percentage
Female	33	72.3
Male	12	26.6

Correlation between baseline VA of affected eye and 1 month visual acuity of affected eye

	Mean	Std. Deviation
VA . Baseline	1.1321	0.72302
VA.1month	0.6992	0.62167

5. Discussion

Optic neuritis occurs when swelling (inflammation) damages the optic nerve — a bundle of nerve fibers that transmits visual information from your eye to your brain. Common symptoms of optic neuritis include pain with eye movement and temporary vision loss in one eye. The objective of my study is to review cases presenting with optic neuritis with a view to identify the visual outcome.

- In the study done by Beijing Jingmei Group General Hospital, Beijing, China, The mean age of scheduled patients was 34.3 ± 12.4 years, with the majority being female (70.2%). The mean visual acuity score was

58.62 ± 17.62. Twenty-nine (50.9%) patients were affected binocularly

In our study out of 45 patients 73.3 % (33 patients) were female and 26.7 % (12 patients) were male..The mean age of those with optic neuritis was 36.91 ± 17.201. The mean BCVA of the affected eye was 0.30 ± 0.6, and the mean BCVA of the other eye was 0.0861 ± 0.41286. 20 (30%) patients were affected binocularly.

- In another study done by Chiang Mai University, Thailand, out of 150 patients with optic neuritis, 58 were diagnosed with Neuromyelitis Optica spectrum disease (NMOSD), 23 patients were diagnosed with multiple sclerosis (MS), and 69 patients were idiopathic. In our study out of 65 patients, 43 were idiopathic, 8 were diagnosed with MOG, 4 patients were MS, 2 were Neuromyelitis Optica(NMO) and 1 patient were diagnosed with chronic relapsing inflammatory optic neuropathy(CRION) and infective.
- In an article entitled 'Optic Neuritis' by Tahir Mahmood,a population based study was conducted. In this study most of the persons who were affected with optic neuritis were in the age group 30-35 years. In our study out of 65 patients who had optic neuritis, most of the persons, 17 (26 %) were in the age group 31-40 years.
- In an article entitled 'Visual field defects in optic neuritis : distinctive features', by Jürgen Gerling, Jörg Heinrich Meyer, and GuntramKommerell included 99 patients with optic neuritis. It concluded that a scotoma centered on the fixation point with a sloping border is highly characteristic of optic neuritis. In our study out of 65 patients, 17 patients had done the test in which most of the persons,4(23.52%) had central scotoma.
- In the study done by Rohit Saxena et al. Indian J Ophthalmol, Papillitis (53.5 % of eye) was more common than Retrobulbar neuritis(46.5%). In our study Retrobulbar (60% eyes) was more common than Papillitis (40 % of eyes).
- In the study done by Selvakumar Ambika et Sankara Nethralaya, Medical Research Foundation, Chennai, 60 of the 84 eyes (72.3%) recovered visual acuity of 20/20 or better. Visual acuity improvement was stastically significant between initial and final visual acuity in patients treated with the ONTT protocol(P < 0.001). In our study 26 of the 65 eyes (40 %) recovered visual acuity 20/20 or better. Visual acuity improvement was stastically significant between initial and final visual acuity in patients treated with the ONTT protocol (P < 0.001).

6. Conclusion

The most common cause of optic neuritis in our study was idiopathic. Optic neuritis was found to mainly affect the 31–40 age group; females were more affected. Central scotoma was the most common visual defect in optic neuritis. Diabetes mellitus was the associated co-morbidity in optic neuritis. The visual prognosis of optic neuritis at 1 month is good for optic neuritis. There was a significant improvement in visual acuity at 1 month. (P< 0.001). In our study, retrobulbar neuritis was more common than papillitis.

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References

- [1] Very well health Optic Nerve Available from: <https://www.verywellhealth.com/optic-nerve-anatomy-4686150>(accessed 4.2.2021)
- [2] Mcmenamin P, Forrester JV, Dick AD, Lee WR. The Eye –Basic Sciences in Practice
- [3] Department of Neurology, 12700 E 19th Ave, Box B-182, Aurora, CO 80045 Address correspondence to Dr Jeffrey L. Bennett, Department of Neurology, 12700 E 19th Ave, Box B-182, Aurora, CO 80045, Jeffrey.Bennett@ucdenver.edu.
- [4] Britannica Optic Nerve Available from:<https://www.britannica.com/science/optic-nerve> (accessed 4.2.2021)
- [5] Overview of the optic nerve image - © Kenhub <https://www.kenhub.com/en/library/anatomy/the-optic-nerve>
- [6] Lorenzo Crumbie. Cranial neves examination: Optic nerve. Kenhub, 2021. Available at: <https://www.kenhub.com/en/library/anatomy/clinical-examination-of-the-optic-nerve> Accessed on October, 4, 2021
- [7] Tandon V, Mahapatra AK. Current Management of Optic Nerve Injury. Indian Journal of Neurosurgery. 2017 Aug;6(02):083-5.
- [8] ↑ Steinsapir KD, Goldberg RA. Traumatic optic neuropathy: an evolving understanding. American journal of ophthalmology. 2011 Jun 1;151(6):928-33.
- [9] ↑ Thaker A, Tandon DA, Mahapatra AK. Surgery for optic nerve injury: should nerve sheath

- incision supplement osseous decompression?. Skull Base. 2009 Jul;19(04):263-71.
- [10] MSD manuals eye Disorders Available from: <https://www.msmanuals.com/en-au/home/eye-disorders/optic-nerve-disorders/overview-of-optic-nerve-disorders> (accessed 4.2.2021)
- [11] Drugs.com Optic nerve Available from: <https://www.drugs.com/health-guide/optic-nerve-swelling-papilledema.html> (accessed 4.2.2021)
- [12] Beijing Jingmei Group General Hospital, Beijing, China. 2Senior Department of Ophthalmology, The Third Medical Center of People's Liberation Army (PLA) General Hospital, Beijing, China.
- [13] Department of Ophthalmology, Faculty of Medicine, Chiang Mai University, Chiang Mai, 50200, Thailand.
- [14] Alam SM, Kyriakides T, Lawden M, Newman PK. Methylprednisolone in multiple sclerosis: a comparison of oral with intravenous therapy at equivalent high dose. *J Neurol Neurosurg Psychiatry* 1993;56(11):1219–1220. doi: 10.1136/jnnp.56.11.1219. [PMC free article] [PubMed] [CrossRef] [Google Scholar] [Ref list]
- [15] Morrow SA, Fraser JA, Day C, et al. Effect of treating acute optic neuritis with bioequivalent oral vs intravenous corticosteroids: a randomized clinical trial. *JAMA Neurol* 2018;75(6):690–696. doi: 10.1001/jamaneurol.2018.0024. [PMC free article] [PubMed] [CrossRef] [Google Scholar] [Ref list]
- [16] Kidd DP, Burton BJ, Graham EM, Plant GT. Optic neuropathy associated with systemic sarcoidosis. *Neurol Neuroimmunol Neuroinflamm* 2016;3(5): e270. doi: 10.1212/NXI.0000000000000270. [PMC free article] [PubMed] [CrossRef] [Google Scholar] [Ref list]
- [17] Petzold A, Plant GT. Chronic relapsing inflammatory optic neuropathy: a systematic review of 122 cases reported. *J Neurol* 2014;261(1):17–26. doi: 10.1007/s00415-013-6957-4. [PubMed] [CrossRef] [Google Scholar] [Ref list]
- [18] Beck RW, Cleary PA, Trobe JD, et al. The effect of corticosteroids for acute optic neuritis on the subsequent development of multiple sclerosis. The Optic Neuritis Study Group. *N Engl J Med* 1993;329(24):1764–1769. doi: 10.1056/NEJM199312093292403. [PubMed] [CrossRef] [Google Scholar]
- [19] Goodin DS. Perils and pitfalls in the interpretation of clinical trials: a reflection on the recent experience in multiple sclerosis. *Neuroepidemiology* 1999;18(2):53–63. doi: 10.1159/000069408. [PubMed] [CrossRef] [Google Scholar]
- [20] Sharrack B, Hughes RA, Morris RW, et al. The effect of oral and intravenous methylprednisolone treatment on subsequent relapse rate in multiple sclerosis. *J Neurol Sci* 2000;173(1):73–77. doi: 10.1016/S0022-510X(99)00304-4. [PubMed] [CrossRef] [Google Scholar]
- [21] Zivadinov R, Rudick RA, De Masi R, et al. Effects of IV methylprednisolone on brain atrophy in relapsing-remitting MS. *Neurology* 2001;57(7):1239–1247. doi: 10.1212/WNL.57.7.1239. [PubMed] [CrossRef] [Google Scholar]
- [22] Noseworthy JH, O'Brien PC, Petterson TM, et al. A randomized trial of intravenous immunoglobulin in inflammatory demyelinating optic neuritis. *Neurology* 2001;56(11):1514–1522. doi: 10.1212/WNL.56.11.1514. [PubMed] [CrossRef] [Google Scholar]
- [23] Roed HG, Langkilde A, Sellebjerg F, et al. A double-blind, randomized trial of IV immunoglobulin treatment in acute optic neuritis. *Neurology* 2005;64(5):804–810. doi: 10.1212/01.WNL.0000152873.82631.B3. [PubMed] [CrossRef] [Google Scholar]
- [24] Deschamps R, Gueguen A, Parquet N, et al. Plasma exchange response in 34 patients with severe optic neuritis. *J Neurol* 2016;263(5): 883–887. doi: 10.1007/s00415-016-8073-8. [PubMed] [CrossRef] [Google Scholar]
- [25] Aungsumart S, Apiwattanakul M. Clinical outcomes and predictive factors related to good outcomes in plasma exchange in severe attack of NMOSD and long extensive transverse myelitis: case series and review of the literature. *Mult Scler Relat Disord* 2017;13:93–97. doi: 10.1016/j.msard.2017.02.015. [PubMed] [CrossRef] [Google Scholar]
- [26] Bonnan M, Valentino R, Debeugny S, et al. Short delay to initiate plasma exchange is the strongest predictor of outcome in severe attacks of NMO spectrum disorders. *J Neurol Neurosurg Psychiatry* 2018;89(4):346–351. doi: 10.1136/jnnp-2017-316286. [PubMed] [CrossRef] [Google Scholar]
- [27] Koziolok MJ, Tampe D, Bähr M, et al. Immunoabsorption therapy in patients with multiple sclerosis with steroid-refractory optical neuritis. *J Neuroinflammation* 2012;9:80. doi: 10.1186/1742-2094-9-28. [PMC free article] [PubMed] [CrossRef] [Google Scholar]