SYSTEMATIC REVIEW

Features of the Clinical Manifestations of Autoimmune Optic Neuropathy in Multiple Sclerosis on Corticosteroid Therapy

Dalia Sharliz Raihana[1], Lukiasari Agustini[2*], Deasy Fetarayani[3], Agus Ariyanto[4]
[1]Faculty of Medicine, University Airlangga, Surabaya, Indonesia.
[2]Department Opthalmology, Faculty of Medicine, University Airlangga, Surabaya, Indonesia.
[3]Department of Internal Medicine, Faculty of Medicine, University Airlangga, Surabaya, Indonesia; Dr. Soetomo General Academic Hospital, Surabaya, Indonesia.
[4]Department of Opthalmologist, Imanuddin General Hospital, West Kotawaringin, Indonesia.

Article Info
Article history:
Received 02-01-2024
Revised 05-06-2024
Accepted 21-06-2024
Published 01-07-2024

Keywords:
Optic neuropathy
Quality
Multiple sclerosis (MS)
Optic neuritis treatment trial (ONTT)

***Corresponding author**:
Lukiasari Agustini
lukiasari.agustini@fk.unair.ac.id

ABSTRACT

Background: The clinical picture of autoimmune optic neuropathy, known as neuritis, is strongly influenced by the unique structure of the human anterior visual pathway. The central nervous system autoimmune is related to multiple sclerosis (MS). Optic neuropathy is an injury that frequently results in acute inflammatory damage. **Objective**: This study aimed to determine the clinical manifestations of autoimmune optic neuropathy in multiple sclerosis and identify diseases with appropriate corticosteroid therapy using systematic review methods. **Material and Method**: This study used a systematic review method to analyze topic-related kinds of literature on Scopus, PubMed, and Google Scholar databases. The literature screening process was carried out based on the PRISMA 2020 guidelines. **Result**: Regarding the post-treatment recurrence rate of optic neuritis, the use of intravenous corticosteroids alone and intravenous corticosteroid followed by oral administration may clinically reduce the incidence of recurrence in the patients compared those receiving placebo and oral administration. This suggests that intravenous corticosteroid followed by oral corticosteroid treatment is effective in helping to reduce the incidence of recurring optic neuritis. **Conclusion**: Intravenous corticosteroid treatment followed by oral administration may clinically reduce the incidence of post-treatment recurrence of optic neuritis in multiple sclerosis (MS) patients.

How to cite:
Raihana, D.S., Agustini, L., Fetarayani, D. 2024. Features of the Clinical Manifestations of Autoimmune Optic Neuropathy in Multiple Sclerosis on Corticosteroid Therapy. Majalah Biomorfologi-Biomorphology Journal, 34(2): 123-133.

Majalah Biomorfologi (Biomorphology Journal) p.ISSN:0215-8833, e.ISSN: 2716-0920
doi: 10.20473/mbiom.v34i1.2024.123-133

Highlights

1. Intravenous corticosteroid treatment followed by oral clinical treatment can reduce the incidence of post-treatment optic neuritis recurrence.
2. Visual acquisition increases at one-month post-treatment.

BACKGROUND

The clinical picture of autoimmune optic neuropathy, also known as neuritis, is strongly influenced by the unique structure of the human anterior visual pathway. This central nervous system autoimmune disease is related to multiple sclerosis (MS) (Kawachi, 2017). Optic neuropathy is an injury that often causes acute inflammatory damage with the typical diagnoses of vision loss, colour vision impairment, decreased brightness perception, afferent pupillary deficiency, nerve fibre layer disruption, optic nerve pallor, or optic nerve swelling (Ghadiali & Odel, 2019). Optic neuritis has a strong connection with MS, which is an autoimmune disease that attacks certain individuals. Optic neuropathy associated with MS can manifest in two forms: opticospinal multiple sclerosis (OS-MS), which is accompanied by cerebral lesions, and neuromyelitis optica (NMO), which is not accompanied by cerebral lesions. These two forms are still not specific entities. Some associated eye symptoms include pain, especially pain exacerbated by eye movement, diplopia, and positive visual phenomena (Pineles & Balcer, 2019). According to Bennett's (2019) study results, optic nerve inflammation may result from various causes, such as autoimmunity.

Optic neuritis is a type of inflammatory optic neuropathy that is often associated with autoimmune neurological diseases, such as MS, myelin-oligodendrocyte glycoprotein antibody-related diseases, and optic neuromyelitis spectrum disorders (Bennett, et al., 2023). When suspicion of clinically isolated syndrome (CIS) or MS arises, a brain and spinal cord scan should be performed. In addition to providing information regarding the distribution of the lesion in the space, an MRI of the spinal cord is also functional for differential diagnosis when there is uncertainty regarding the nature of the brain lesion. Spinal cord MRI can reveal disease activity without symptoms and predict disease evolution (Tomassini, et al., 2020). The exact cause of MS is not yet known. Still, several journals show evidence that part of the immunopathogenesis of MS is derived from demyelinating antibody responses resulting in inflammation of the central nervous (CNS) with varying clinical presentations leading to the contribution of autoimmune manifestations. MS remains elusive and is thought to involve genetic and environmental factors with no definitive treatment. The prevalence of this disease has a peak between the ages of 20-40 years, but it has been proven also to affect children and elderly people over the age of 60 years. MS affects women about twice as often as men (Bennett, 2019). According to the Optic Neuritis Treatment Trial (ONTT), initial therapy for patients with demyelination high-dose corticosteroids intravenously or orally, which is standard practice for patients with optic neuropathy to reduce the severity of attacks and prevent permanent damage (Morrow, et al., 2018). Corticosteroids are a drug that is often used and is relatively cheap to reduce inflammation (Bennett, et al., 2023).

This study aimed to provide a deeper understanding and overall facts regarding appropriate corticosteroid therapy by differentiating the route of administration for MS patients. The findings will be summarized in detail to cover a comprehensive level of knowledge and understanding. Further case studies of the impact of corticosteroid treatment therapy on patients will be presented by providing relevant facts.

OBJECTIVE

This systematic review aimed to determine the clinical manifestations of autoimmune optic neuropathy in multiple sclerosis and identify diseases with appropriate corticosteroid therapy.

MATERIAL AND METHOD

This study used a systematic review method to analyze topic-related literature in the Scopus, PubMed, and Google Scholar databases. This systematic review registration number in PROSPERO is CRD42023491771. Retrieving literature to determine data quality used the main version of the 2020 PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses). Before entering the screening stage, researchers eliminated studies that were not relevant because the title did not match the topic. Duplicate studies were from the same journal, only manually taken from one database. Manual searches were carried out to reduce the bias of inappropriate keywords, making it easier to continue

screening the articles further. Data screening was done on the condition that it met the inclusion criteria, and the data were excluded if it did not match the research topic.

The PICO (Population, Intervention, Comparison, Outcomes) approach was used in this study. The population was patients suffering from multiple sclerosis (MS) with optic neuropathy. The intervention was intravenous and oral corticosteroids, while the comparison was not applied. The outcome included the clinical features (VEP or Visual Evoked Potential and VA or Visual Acuity)

The included research articles must be accessible in full-text literature that explains the manifestations of autoimmune optic neuropathy in multiple sclerosis (MS) and research that uses case-control or cross-sectional study methods. The articles that could not be included were those involving patients who had MS without symptoms of optic neuropathy, studies that used systematic review methods, and those that did not discuss MS.

RESULT

The PRISMA guidelines were used for the procedure and evaluation of the systematic review, as shown in Figure 1. On December 11, 2023, literature was carried out on the Scopus, PubMed, and Google Scholar databases. The following is the PRISMA flowchart made in this study.

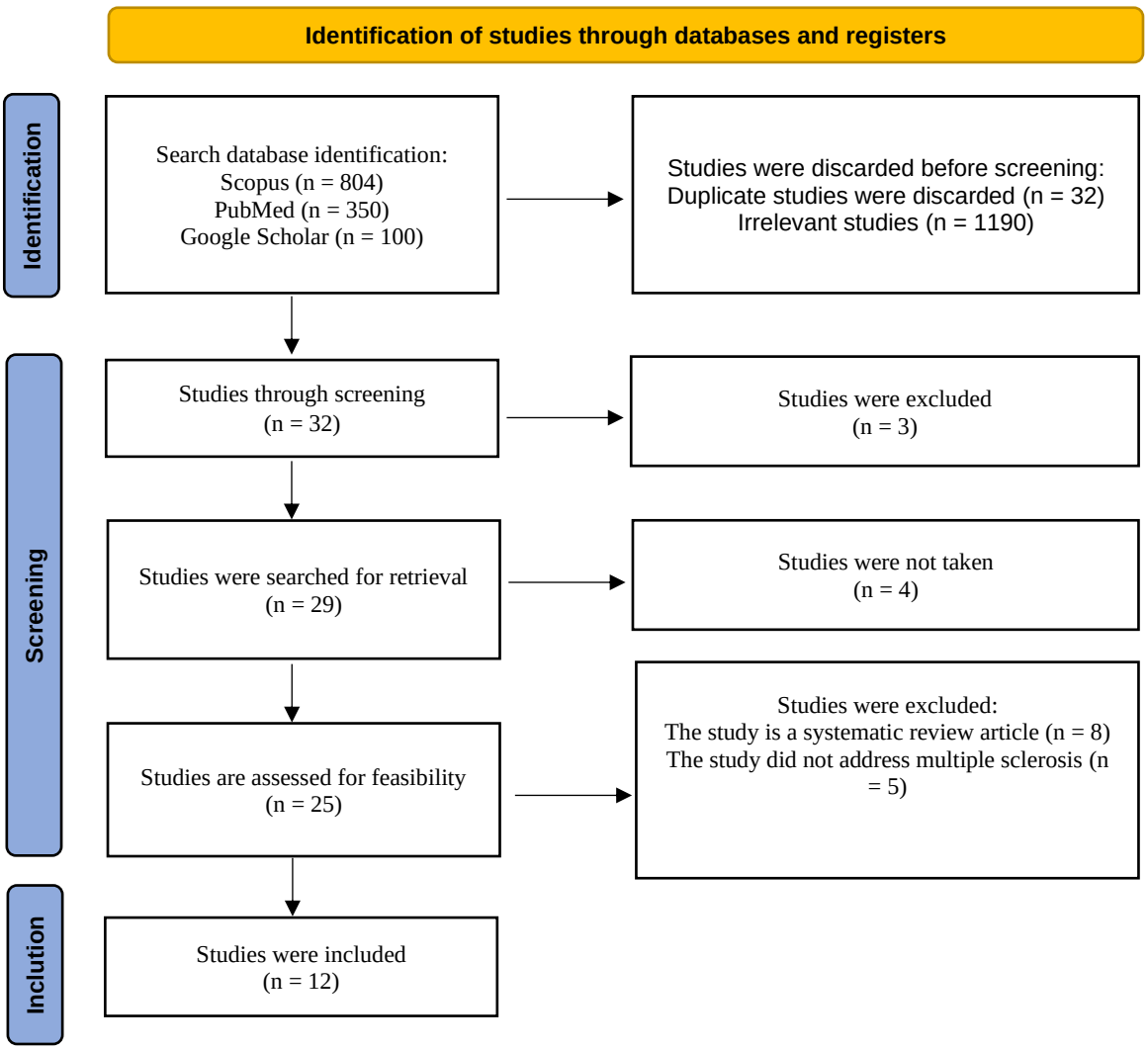

Figure 1. PRISMA systematic review diagram 2020 category.
"new systematic reviews which included searches of databases and registers only"
(Page, et al., 2021).

All of the included articles underwent quality assessment using critical appraisal tools, as shown in Tables 1, 2, 3, and 4.

Table 1. Data extraction results from reviewed journals.

No.	Reference	Amount of samples	Intervention		Results			Information
			Orally	Intravenous	Variable	Before	After	
1.	Smith, et al. (1986)	Eight patients	-	1000 mg Methylprednisolone 1x1 for three days.	P100 latency of visual-evoked potential & visual acuity for one month.	132 ± 3.3 MS & 6/18	127 ± 5.8 MS & 6/6	Shortening mean P100 latency of visual-evoked potential and repaired visual acuity. The shortening latency value shows the process of remyelination from neurons.
2.	Kapoor et al., (1998)	25 patients	-	1000 mg Methylprednisolone 1x1 for three days.	P100 latency of visual-evoked potential & visual acuity for six months.	130.5 ± 19.0 MS & 0.63	122.7 ± 15.0 MS & 0.68	Shortening mean P100 latency of visual-evoked potential and repaired visual acuity. The shortening latency value shows the process of remyelination from neurons.
3.	Morrow, et al., (2018)	23 patients	Prednisone 1250 mg	1000 mg Methylprednisolone 1x1 for three days.	P100 latency of visual-evoked potential & visual acuity for six months.	181.9 ± 53.6 MS & 20/100	119.0 ± 16.5 MS & 20/160	The decline means P100 latency of visual-evoked potential & a decrease in visual acuity.
4.	Menon, et al., (2007)	11 patients	-	200 mg Dexamethasone 1x 1 for 3 days.	LOGMAR visual acuity.	1.10 ± 0.52	0.28 ± 0.33	After three months, the visual acuity was repaired.

5.	Naumovska, et al., (2018)	28 Patients	Methylprednisolone 500 mg	500 mg Methylprednisolone.	LOGMAR visual acuity.	Orally: 0.30 IV: 0.35	Orally: 0.05 IV: 0.05	There is fast repair of visual acuity.
6.	de Lott, et al., (2020)	335 patients	-	500 mg Methylprednisolone.	LOGMAR Visual Acuity	0.305	0.084	It happens to repair visual acuity faster.
7.	Beck, et al., (1992)	15 6 patients orally, 151 patients intravenous	Prednisonene 1 mg/kg ww/day for 14 days	1000 mg Methylprednisolone intravenous 1x1 for three days followed with prednisolone orally 1 mg/kg ww/day for 1 1 day.	Percentage of visual acuity after six months.	58.7%	Intravenous:60.9% PO: 54.5%	There is enhanced sensitivity contrast, color vision, visual acuity, and visual fields, which are faster in the intravenous group.
8.	Goodwin, (1996)	438 patients	Prednisonene 1 mg/kg ww/day for 14 days.	1000 mg Methylprednisolone intravenous 1x1 for three days followed by prednisolone orally 1 mg/kg ww/day for 11 days.	Percentage of patients who Still have visual function six months post-maintenance.	5.3%	IV: 6.0% Orally: 7.1%	Enhancement of visual functionality: One week first, IV is faster than orally.
9.	Al-Eajailat & Al-MadaniSenior, (2014)	50 patients placebo, 50IV patients, 50 patients P.O	Prednisonene 1 mg/kg ww/day for 14 days.	1000 mg Methylprednisolone intravenous 1x1 for three days followed by prednisolone orally 1 mg/kg ww/day for 11 days.	The amount patients who experience enhanced visual acuity is a minimum of 50% after four weeks	IV:84 % Orally: 86%	IV: 94% Orally: 94%	Shortening means P100 latency of visual-evoked potential and improve-ments in visual acquisition.
10.	Halilovic, et al., (2014)	Ten patients	-	1000 mg Prednisolone intravenous 1x3 days followed by prednisolone 1 mg/kg/d for eight days.	Visual acuity	0.2 ± 0.1	0.8 ± 0.2	The visual acquisition increased at one-month post-treatment.
11.	Dahanayake, et al., (2021)	29 patients	-	1000 mg Methylprednisolone intravenous 1x3 days followed with prednisone ne 1 mg/kg ww/day for 11 days.	Decline P100 latency of visual-evoked potential	131.85 ± 10.85 MS	110.7 ± 12.5 MS	Decline P100 latency from visual-evoked potential to average value one month after treatment.

12.	Sellebjerg, et al., (1999)	60 patients	500 mg oral Methylprednisolone 1x1 for five days. Later dose tapering for ten days.	-	Percentage of patients who reach visual acuity normal after 8 Sunday	Placebo :43%	Orally:50%	There isn't any significant difference in visual acuity between the oral and placebo groups.

Table 2. The effect of corticosteroid administration on VEP.

No.	Reference	Intervention		Variable	Results	Information
		Orally	Intravenous			
1.	Smith, et al. (1986)	-	1000 mg Methylprednisolone 1x3 in 3 days.	P100 latency of visual-evoked potential & visual acuity for one month.	Before: 132 ±3.3 MS & 6/18. After: 127 ± 5.8 MS & 6/6.	Shortening mean P100 latency of visual-evoked potential and repaired visual equity. The shortening latency value shows that remyelination from neurons has occurred.
2.	Kapoor et al., (1998)	-	1000 mg Methylprednisolone 3x1 day.	The observed variables are P100 latency of visual-evoked potential & visual acuity for one month.	Before: 130.5 ± 19.0 MS & 0.63 After: 122.7 ± 15.0 MS & 0.68	Shortening means P100 latency of visual-evoked potential and repaired visual acuity. The shortening latency value shows that remyelination process from the nerve cell has occurred.
3.	Dahanayake, et al., (2021)	-	1000 mg intravenous Methylprednisolone 1x1 for three days followed with prednisonene 1 mg/kg ww/day for 11 days.	Declined P100 latency of visual-evoked potential	Before: 131.85 ± 10.85 MS After: 110.7 ± 12.5 MS	Declined P100 latency of visual-evoked potential to normal value one month after treatment, indicating remyelination.
4.	Morrow, et al., (2018)	1250 mg Prednisone	-	P100 latency of visual-evoked potential & visual acuity for six months.	Before : 181.9 ± 53.6 MS & 20/100 After : 9.0 ±16.5 MS & 20/160	The declined means P100 latency of visual-evoked potential, which indicates the remyelination process.

Table 3. The effect of corticosteroid administration on visual acuity.

No.	Reference	Intervention		Variable	Results	Information
		Orally	Intravenous			
1.	Beck, et al., (1992)	1 mg/kg oral Prednisolone ww/day for 11 days.	-	Percentage of patients who have visual acuity after six months.	Intravenous= 60.9 % Orally=54.5%	There is enhanced sensitivity contrast, color vision, visual acuity, and visual fields, which are faster than the oral group.
2.	Goodwin, (1996)	1 mg/kg Prednisone ww/day for 14 days.	-	Percentage of patients who still own visual functions, the bad ones six months post-care.	Intravenous 6% per oral 7.1%.	There isn't any significant difference between IV and placebo at six months; however, enhanced visual function in 1 first week is faster than oral.
3.	Al-Eajailat & Al-MadaniSenior, (2014)	1 mg/kg/day oral prednisolone for 14 days	-	The amount of patients who experience enhanced visual acuity minimum 50% after four weeks compared to placebo.	Intravenous= 47 person Orally= 47 person	There were no differences between the treatment group and the control group.
4.	Halilovic, et al., (2014)	-	-	Visual acuity	Before: 0.2 ± 0.1 After: 0.8 ± 0.2.	The visual acquisition increased one month post-treatment
5.	Sellebjerg, et al., (1999)	-	-	The percentage of patients who reach visual acuity is expected after 8 weeks.	Oral: 50% P placebo 43%.	There isn't any significant difference in visual acuity between oral and placebo groups.

Table 4. The effect of corticosteroid on LOGMAR visual acuity

No.	References	Intervention		Variables	Results	Information
		Orally	Intravenous			
1.	Menon, et al., (2007)	200 mg Dexamet hasone 1x3 days.	-	LOGMAR visual acuity	Before: 1.10 ± 0.52 After: 0.28 ± 0.33	It happens to repair visual acuity three months after treatment.
2.	Naumovska, et al., (2018)	500 mg Methylpre prednisolone	-	LOGMAR visual acuity	Before: Oral: 0.30 IV:0.35 After: Orally: 0.05 IV: 0.05	There is visual acuity improvement, faster compared to placebo.
3.	de Lott, et al., (2020)	-	500 mg Methylpr prednisolone	LOGMAR visual acuity	Before : 1.10 ± 0.52 After : 0.28 ± 0.33	After three months of repair, visual acuity indicating remyelination has occurred.

DISCUSSION

Multiple sclerosis is an autoimmune disease with a prevalence of 35.9 individuals per 100,000 population. Based on existing data, this number has continued to increase every year since 2013 (Walton, et al., 2020). Multiple sclerosis is a disease that can significantly reduce the patients' quality of life. One of the clinical signs of multiple sclerosis is a decrease in the quality of vision, so many studies are looking for the most appropriate way to improve the quality of vision among the multiple sclerosis patients. One treatment widely studied to overcome this problem is corticosteroid therapy (Ozdogar, et al., 2022).

Research conducted by Smith, et al., (1986), Kapoor, et al., (1998), and Morrow, et al., (2018) observed the effects of methylprednisolone corticosteroid treatment at a dose of 1000 mg given to patients once a day for three days through an intravenous route based on improved visual evoked potential (VEP) latency. These three studies found improvements in mean VEP latency after one month and six months of treatment, but these changes were insignificant. There is a possibility that the improvement in mean VEP may not be due to intravenous corticosteroid treatment because the group that did not receive treatment also experienced an improvement in mean VEP (Smith, et al., 1986).

Recent research by Menon, et al., (2007); Naumovska, et al., (2018); de Lott, et al., (2020) showed that 500 mg intravenous methylprednisolone or 200 mg dexamethasone improve visual acuity in symptoms of optic neuritis experienced by multiple sclerosis patients. These three studies, using methylprednisolone and dexamethasone, found a faster visual acuity increase compared to the placebo group. Still, the two groups had no significant difference in the final visual acuity value. Studies by Beck, et al., (1992), Cleary, et al., (1993), Goodwin, (1996), Menon, et al., (2007), Al-Eajailat & Al-MadaniSenior, (2014), Halilovic, et al., (2014), and Dahanayake, et al., (2021) observed the effect of treatment using 1000 mg intravenous methylprednisolone once a day for three days followed by 1 mg/kg oral prednisone once a day for 14 days on visual acuity in multiple sclerosis patients who experienced optic neuritis. These eight studies showed uniform results. The patients who received intravenous corticosteroid treatment followed by oral therapy experienced a faster increase in visual acuity compared to the group that received placebo treatment. However, no significant differences were found between the two groups in evaluating the final visual acuity scores six months or one year after treatment.

Then, the researchers Beck, et al., (1992), Cleary, et al., (1993), Goodwin, (1996), Gal, et al., (2012), and Al-Eajailat & Al-MadaniSenior, (2014) examined the effects of 1 mg/kg oral prednisolone treatment once a day for 14 days on visual acuity, visual function, visual fields, contrast sensitivity, and VEP latency. These three studies showed uniform results in that all parameters improved after treatment, but this did not increase significantly compared to patients who received treatment with a placebo. In addition, these three studies showed that patients who received oral corticosteroid treatment experienced a higher rate of optic neuritis recurrence than patients who received placebo treatment.

Researchers observed the effects resulting from 500 mg oral methylprednisolone treatment once a day for five days continued with tapering doses for ten days (Sellebjerg, et al., 1999; Naumovska, et al., 2018). Both studies showed uniform results, in which there were improvement in visual acuity, colour vision, and contrast sensitivity, between the group that received oral corticosteroid treatment and the group that only received placebo treatment. Still, there were no significant differences between the two groups. A Study using 1250 mg prednisone showed improved VEP six months after treatment (Morrow, et al., 2018).

In general, treatment of optic neuritis in multiple sclerosis patients using intravenous corticosteroids alone, intravenous followed by oral, or oral alone shows an increase in VEP. However, the differences experienced are not significant, and there is an increase in visual acuity. The increase in the average of both parameters is due to corticosteroid treatment, which reduces inflammation and edema that forms on the optic disc. Reducing inflammation and edema that forms on the optic disc can reduce demyelination and improve conduction in the demyelinated nerve area, which can help speed up the course of stimulus conduction (de Lott, et al., 2022). The non-significant difference in VEP latency but significant in visual acuity is probably because the VEP latency test is a test that has higher sensitivity and specificity than visual acuity so that a more important clinical change is needed to show a more statistically significant change in VEP latency values (Ismaiel, et al., 2020).

This suggests that in terms of improving visual function, corticosteroid treatment is effective in accelerating changes but does not improve the outcome of improving visual function (de Lott, et al., 2022). Regarding post-treatment optic neuritis recurrence rates, studies using intravenous corticosteroids alone and intravenous followed oral corticosteroid groups showed reduced incidence of recurrence in patients compared to the placebo and oral groups. The group using oral corticosteroids alone did not show a reduced incidence of optic neuritis recurrence in patients compared to the placebo group (Al-Eajailat & Al-MadaniSenior, 2014).

In ONTT, findings were shown in optic neuritis patients with oral and intravenous prednisolone. Based on the description above, it can be observed that optic neuritis treatment using corticosteroids with various routes can be considered based on multiple considerations. The first one is whether the patient needs immediate improvement in visual function. Patients needing to improve visual function quickly can consider treatment using corticosteroids (Gal, et al., 2012). The second one that can be considered is that in reducing the recurrence rate of optic neuritis, intravenous corticosteroid only or followed by the oral administration has advantages over oral treatment alone (Al-Eajailat & Al-MadaniSenior, 2014). This raises further considerations, that the higher toxicity of intravenous corticosteroid treatment and its adverse effects may occur as a result of the treatment. Some of the adverse effects reported to be experienced by multiple sclerosis patients with optic neuritis and receiving corticosteroid treatment include transient depression, acute pancreatitis, sleep disturbances, mood swings, stomach discomfort, facial flushing and weight gain (Gal, et al., 2012).

Strength and limitations

The strength of this review is that it has conducted a comprehensive analysis of relevant articles to demonstrate that administering intravenous corticosteroids followed by oral clinical therapy can effectively decrease the occurrence of recurrent optic neuritis after treatment. Additionally, it has been observed that visual acquisition improves one month after therapy. However, the limitation of this study is that it should have gathered more recent studies regarding the case.

CONCLUSION

Clinically, intravenous corticosteroid treatment, followed by oral administration, can accelerate visual function improvement in visual acuity and visual evoked potential (VEP) latency. Intravenous corticosteroid only and intravenous corticosteroid followed by oral treatment can also reduce the incidence of post-treatment optic neuritis recurrence.

Acknowledgment

The authors would like to thank the Medical Staff of the Department of Opthalmology, Faculty of Medicine, University of Airlangga, and the Department of Internal Medicine, Faculty of Medicine, University of Airlangga.

Conflict of Interest

All authors have no conflict of interest.

Funding Disclosure

None.

Author Contribution

DSR contributes to the conception and design, analysis and interpretation of the data, drafting of the article, critical revision of the article for important intellectual content, provision of study materials or patients, administrative, technical, or logistic support, and collection and assembly of the data. LA and DF contribute to the final approval of the article and provision of study materials or patients.

REFERENCES

Al-Eajailat, S. M., Al-MadaniSenior, M. V. 2014. The role of magnetic resonance imaging and visual evoked potential in management of optic neuritis. Pan African Medical Journal, 17. doi:

10.11604/pamj.2014.17.54.2462.
Beck, R. W., Cleary, P. A., Anderson, M. M., et al. 1992. A randomized, controlled trial of corticosteroids in the treatment of acute optic neuritis. New England Journal of Medicine, 326(9): 581–588. doi: 10.1056/NEJM199202273260901.
Bennett, J. L. 2019. Optic neuritis. Continuum: Lifelong Learning in Neurology, 25(5): 1236–1264. doi: 10.1212/CON.0000000000000768.
Bennett, J. L., Costello, F., Chen, J. J., et al. 2023. Optic neuritis and autoimmune optic neuropathies: advances in diagnosis and treatment. The Lancet Neurology, 22(1): 89–100. doi: 10.1016/S1474-4422(22)00187-9.
Cleary, P. A., Beck, R. W., Anderson, M. M., et al. 1993. Design, methods, and conduct of the optic neuritis treatment trial. Controlled Clinical Trials, 14(2): 123–142. doi: 10.1016/0197-2456(93)90015-6.
Dahanayake, P., Beck, R. W., Anderson, M. M., et al. 2021. The effect of optic neuritis treatment trial (ONTT) combined corticosteroid regimen on pattern reversal visual evoked potentials: A prospective follow-up study. BMC Ophthalmology, 21(1): 324. doi: 10.1186/s12886-021-02086-z.
Gal, R. L., Vedula, S. S., Beck, R. 2012. Corticosteroids for treating optic neuritis. in Gal, R. L. (ed.) Cochrane Database of Systematic Reviews. Chichester, UK: John Wiley & Sons, Ltd. doi: 10.1002/14651858.CD001430.pub3.
Ghadiali, L. K., Odel, J. G. 2019. Optic neuropathy. in The Columbia Guide to Basic Elements of Eye Care. Cham: Springer International Publishing: 399–427. doi: 10.1007/978-3-030-10886-1_36.
Goodwin, J. 1996. High dosage corticosteroids in the treatment of optic neuritis and prophylaxis of multiple sclerosis. Clinical Immunotherapeutics, 6(5): 405–412. doi: 10.1007/BF03259359.
Halilovic, E. A., Alimanovic, I., Suljic, E., et al. 2014. Optic neuritis as first clinical manifestations the multiple sclerosis. Materia Sociomedica, 26(4): 246–248. doi: 10.5455/msm.2014.246-2481.
Ismaiel, A., Mohsen, A., Anwar, A. 2020. Comparative study between visual evoked potential and visual acuity, a field of vision, and fundus examination as a screening tool for early diagnosis of hydroxychloroquine and chloroquine retinal toxicity in rheumatic patients. Journal of Medicine in Scientific Research, 3(3): 161. doi: 10.4103/JMISR.JMISR_29_20.
Kapoor, R., Miller, D. H., Jones, S. J., et al. 1998. Effects of intravenous methylprednisolone on outcome in MRI-based prognostic subgroups in acute optic neuritis. Neurology, 50(1): 230–237. doi: 10.1212/WNL.50.1.230.
Kawachi, I. 2017. Clinical characteristics of autoimmune optic neuritis. Clinical and Experimental Neuroimmunology, 8(S1): 8–16. doi: 10.1111/cen3.12354.
de Lott, L. B., Burke, J. F., Andrews, C. A., et al. 2020. Association of individual-level factors with visual outcomes in optic neuritis. JAMA Network Open, 3(5): e204339. doi: 10.1001/jamanetworkopen.2020.4339.
de Lott, L. B., Bennett, J. L., Costello, F. 2022. The changing landscape of optic neuritis: A narrative review. Journal of Neurology, 269(1): 111–124. doi: 10.1007/s00415-020-10352-1.
Menon, V., Mehrotra, A., Saxena, R., et al. 2007. Comparative evaluation of megadose methylprednisolone with dexamethasone for treatment of primary typical optic neuritis. Indian Journal of Ophthalmology, 55(5): 355. doi: 10.4103/0301-4738.33821.
Morrow, S. A., Fraser, J. A., Day, C., et al. 2018. Effect of treating acute optic neuritis with bioequivalent oral vs intravenous corticosteroids. JAMA Neurology, 75(6): 690. doi: 10.1001/jamaneurol.2018.0024.
Naumovska, M., Sheikh, R., Bengtsson, B., et al. 2018. Visual outcome is similar in optic neuritis patients treated with oral and i.v. high-dose methylprednisolone: A retrospective study on 56 patients. BMC Neurology, 18(1): 160. doi: 10.1186/s12883-018-1165-6.
Ozdogar, A. T., Baba, C., Kahraman, T., et al. 2022. Effects and safety of exergaming in persons with multiple sclerosis during corticosteroid treatment: A pilot study. Multiple Sclerosis and Related Disorders, 63: 103823. doi: 10.1016/j.msard.2022.103823.
Page, M. J., McKenzie, J. E., Bossuyt, P. M., et al. 2021. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. BMJ: 71. doi: 10.1136/bmj.n71.
Pineles, S. L., Balcer, L. J. 2019. Visual loss. in Liu, Volpe, and Galetta's Neuro-Ophthalmology. Elsevier: 101–196. doi: 10.1016/B978-0-323-34044-1.00005-5.
Sellebjerg, F., Nielsen, H. S., Frederiksen, J. L., et al. 1999. A randomized, controlled trial of oral high-

dose methylprednisolone in acute optic neuritis. Neurology, 52(7): 1479–1479. doi: 10.1212/WNL.52.7.1479.

Smith, T., Zeeberg, I., Sjö, O. 1986. Evoked potentials in multiple sclerosis before and after high-dose methylprednisolone infusion. European Neurology, 25(1): 67–73. doi: 10.1159/000115989.

Tomassini, V., Sinclair, A., Sawlani, V., et al. 2020. Diagnosis and management of multiple sclerosis: MRI in clinical practice. Journal of Neurology, 267(10): 2917–2925. doi: 10.1007/s00415-020-09930-0.

Walton, C., King, R., Rechtman, L., et al. 2020. Rising prevalence of multiple sclerosis worldwide: Insights from the Atlas of MS, third edition. Multiple Sclerosis Journal, 26(14): 1816–1821. doi: 10.1177/1352458520970841.