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CLINICAL FEATURES OF VISUAL ACUITY,CONTRAST SENSITIVITY, AND COLOR VISIONOF OPTIC NEURITIS PATIENTS TREATED WITH STEROIDAT PROF. DR. I. G. N. G. NGOERAH GENERAL HOSPITAL IN 2021-2022

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ABSTRACT

Introduction: Optic neuritis (ON) is an inflammation of the optic nerve that can occur alone or in association with neurological inflammatory or infectious disorders, or local or systemic inflammatory diseases. Vision function will return to near normal in weeks or months regardless of treatment, but it will be hastened with corticosteroid treatment.
Objective: To determine the clinical features of ON patients and difference of visual acuity, contrast sensitivity and color vision before and after steroid therapy.
Methods: Analytical descriptive research with retrospective data collection based on medical records of patients visiting Prof. dr. I. G. N. G. Ngoerah General Hospital in 2021-2022. Statistical analysis regarding comparation of variables before and after treatment used Wilcoxon test.
Results: There were 14 patients with equal percentage of males and females, 15-40 years old (64.3%), bilateral cases (57.1%), and atypical neuritis (92.9%). The chief complaint was blurred vision (57.1%) and onset was less than one week (57.1%). Baseline visual acuity log MAR 2,00±0,51, contrast sensitivity and color vision were mostly not evaluable, respectively 57.1% and 85.7%. After steroid therapy, visual acuity reached log MAR 0,93±1,05, contrast sensitivity and color vision reached normal, both 35.7%.
Conclusion: The NO patients had an equal percentage of males and females, aged 15-40 years, with bilateral atypical neuritis type. There were differences in visual acuity, contrast sensitivity, and color vision before and after steroid therapy. Whereas clinical features were better after steroid therapy.
Keyword: optic neuritis., visual acuity., steroid

INTRODUCTION

Optic neuritis (NO) refers to inflammation of the optic nerve resulting from a variety of causes that may occur isolated or in association with neurologic inflammatory or infectious disorders, or local or systemic inflammatory diseases[1].
NO is broadly divided into typical and atypical optic neuritis. Typical optic neuritis is due to the primary demyelination process of the optic nerve associated with multiple sclerosis (MS) and is reported as the initial clinical manifestation of MS in 20% of cases in Western populations. Atypical optic neuritis is NO that does not have a typical clinical picture, generally caused by autoimmune, inflammatory processes (neuromyelitis optica spectrum disorder/NMOSD, myelin oligodenrocyte glycoprotein/MOG, autoimmune optic neuropathy, chronic relapsing optic neuropathy/CRION), related systemic disorders, or infection of the optic nerve[2].
The annual incidence of new-onset NO is reported to be between 0.56 and 5.1 cases per 100,000 people. Risk factors for NO include age between 20 and 40 years, female gender is found to be twice as much as male, and the most common race is Caucasian. Children rarely develop bilateral NO, but a history of childhood NO is not believed to be a harbinger of MS development[3,4].
Roed et al. believed that demyelination in optic neuritis is immune-mediated, but the specific mechanisms and target antigens involved remain unknown. Systemic T cell activation is identified at the onset of symptoms and precedes cerebrospinal fluid (CSF) changes. Systemic changes return to normal more quickly (within two to four weeks) than central changes. T cell activation leads to the release of cytokines and other inflammatory agents. B cell activation of myelin basic protein is not seen in peripheral blood but can be demonstrated in the CSF of patients with optic neuritis.[5]
Optic neuritis usually presents with an acute onset of monocular eye pain and vision loss in young adults. Pain is usually associated with eye movements and often precedes

the loss of vision. Patients often report having experienced similar events in the same or other eye. The degree of visual impairment can range from near-normal acuity to no light perception.[6]

Almost any visual field impairment can be seen on an automated visual field test, but the most common is central scotoma. Color perception is significantly impaired (red is especially desaturated) and contrast sensitivity is reduced. Patients may experience recurrent photopsia. Vision loss may worsen after exercise or a rise in body temperature (Uhthoff phenomenon). Optic disc edema is seen on fundoscopy in one third of patients during the active phase. In the absence of observable papillitis, the signs and symptoms of NO are usually sufficient to establish the diagnosis of retrobulbar neuritis. Bilateral presentation, severe visual loss, absence of ocular pain preceding visual loss, and atypical neurological findings should prompt a search for alternative etiologies.[7]

Clinical evaluation of suspected optic neuritis includes corrected visual acuity, automated visual field test, color vision test, pupil examination with relative afferent pupil defect (RAPD) measurement, noting that RAPD may not be found in bilateral and symmetrical optic nerve involvement, optic nerve examination, and OCT of the retinal nerve fiber layer or RNFL[8].

If the clinical findings are consistent with ON, additional testing required is MRI of the brain and orbits with and without contrast to look for demyelinating disease. In the active phase, the affected optic nerve will be enhanced. Demyelinating lesions within the brain confirm the diagnosis of MS. Optic neuritis with two or more characteristic lesions (one of which is contrast enhancement) is sufficient to diagnose MS (McDonald criteria). Clinical findings suggestive of neuromyelitis optic spectrum disorder (NMOSD) should prompt serum NMO-IgG testing[8].

Vision function will return to near normal within a few weeks to months, regardless of treatment. In most cases, optic neuritis shows spontaneous improvement within 1 month. However, vision recovery will be faster when treated with corticosteroid therapy. Based on the long-term results of the Optic Neuritis Treatmant Trial (ONTT), the NO treatment protocol has been widely accepted throughout the medical community. Intravenous methylprednisolone (500-1000 mg once daily) for three days followed by oral prednisone (1mg/kg once daily) for eleven days. Note that oral prednisone alone has been found to increase the rate of recurrent NO attacks. If lesions that are typical of MS are evident with (MRI), then immune modulation therapy should be considered to delay subsequent attacks.[1,8]

RESEARCH METHODS

This research is an analytical observational study. Data were collected retrospectively by documenting the characteristics and examination result of patients with optic neuritis according to medical records. The research data included gender, age, domicile, symptoms, onset, etiology, neuritis type, laterality, visual acuity, color vision, and contrast sensitivity. The research was conducted from January 2021 to December 2022 at Prof. dr. I. G. N. G. Ngoerah General Hospital. The number of samples uses *total sampling*. Inclusion criteria includes all of patients with ON who visited the hospital during study period, exclusion criteria includes patients with incomplete medical record data. Patients are categorized into typical optic meuritis if they have the following clinical features; female predilection, unilateral, pain with eye movement, impaired color vision, decreased visual acuity, improvement within a few weeks to 1 month, non-steroid dependent. Funduscopy can be normal (retrobulbar neuritis) or mild optic nerve edema. Neuroimaging shows anterior optic nerve enhancement, demyelinating lesions in the white matter of the brain suggestive of MS. passion is categorized into atypical optic neuritis with the following clinical features; no gender predilection, age <18 or 50 years, unilateral / bilateral, without accompanying eyeball movement pain, decreased visual acuity to no light perception, decreased visual acuity for more than 2 weeks and visual improvement that depends on the etiology and baseline condition of the patient. Some types of NO are steroid dependent, relapsing when steroids are stopped or unresponsive to steroids. Funduscopy can reveal vitritis, uveitis, severe papillary edema with hemorrhage or exudates. Neuroimaging showed extensive nerve enhancement, perineuritis, (predominantly optic nerve sheath block), optic chiasm or optic tract block. The cause of atypical NO due to NMOSD can be established by positive or seronegative AQP4-IgG examination, or by core clinical characteristics. The cause of atypical NO due to SLE is confirmed based on diagnosis by fellow internists. Atypical NO due to toxoplasma or syphilis infection could be confirmed by a positive serologic test for IgM/IgG Anti Toxoplasma or VDRL/TPHA serologic test for Treponema pallidum. Patients with negative neuroimaging, autoimmune, or infectious markers were preliminary diagnosed with idiopathic atypical NO. Data were obtained from medical records and presented as categorical variables. Visual acuity before steroid therapy is the baseline visual acuity when first examined before steroid therapy, measured using a snellen chart or E-chart at a distance of 6 meters or with finger counting, hand movements or flashlight assistance. Data were taken from medical records and converted to log MAR table and considered as numerical variable. Visual acuity after steroid therapy is the visual acuity of patients after 1 month of steroid therapy, measured by snellen chart or E-chart at 6 meters distance or by counting fingers, hand movements or flashlight. Data were obtained from medical records and converted to log MAR table and considered as numerical variable.

Contrast sensitivity after steroid therapy is the ability to distinguish gradations of blackness against the whiteness of a specific object or target. Contrast sensitivity is categorized into normal, visual impairment, and visual disability. An

assessment score of 2.0 indicates 100% normal contrast sensitivity, a score <1.5 is consistent with visual impairment, and a score <1.0 represents visual disability. The scores were assessed based on the Pelli-Robson contrast sensitivity test, 1 month after receiving steroid therapy that obtained from medical records and were considered as categorical variables. Contrast sensitivity vision before steroid therapy is the ability to distinguish gradations of blackness to whiteness of a specific object or target. Contrast sensitivity is categorized into normal, visual impairment, and visual disability. An examination score of 2.0 indicates 100% normal contrast sensitivity, a score <1.5 is consistent with visual impairment, and a score <1.0 represents visual disability. The score was based on the Pelli-Robson contrast sensitivity test, when the patient was first examined before receiving steroid therapy. Data were extracted from medical records and were considered as categorical variables. Color vision before steroid therapy is the ability to distinguish colors assessed by Ishihara examination. Categorized into normal, partial color blindness, total color blindness and unevaluable. The examination was performed when the patient was first examined before receiving steroid therapy. Data were obtained from medical records and considered as
categorical variable. Color vision after steroid therapy is the ability to distinguish colors assessed by Ishihara examination. Categorized into normal, partial color blindness, total color blindness and unevaluable. The examination was conducted 1 month after receiving steroid therapy. Data were obtained from medical records and considered as categorical variable. Color vision after steroid therapy is the ability to distinguish colors assessed by Ishihara examination. Categorized into normal, partial color blindness, total color blindness and unevaluable. The examination was conducted 1 month after receiving steroid therapy. Data were obtained from medical records and considered as categorical variable. All data obtained were tabulated and analyzed with the SPSS version 27.0 program. Data relating to subject profiles were analyzed descriptively. Categorical scaled data were described in frequency and percentage. Normality of numerical data was tested using Saphiro Wilk or Kolmogorov Smirnov. Analysis of differences in visual acuity, contrast sensitivity vision, and color vision before and after therapy was determined by Wilcoxon test.

RESULTS

The results of this study found a total of 19 patients with optic neuritis. Five of them were not treated with steroids for the following reasons, the patient was diagnosed with HIV on HAART with a high viral load, one diagnosed with neurosyphilis, one presented with optic nerve atrophy, one was lost to follow-up, and one was pregnant. Thus, 14 patients (22 eyes) were studied as research subjects.

Table 1. Demograpics characteristics

Characteristics	**n** (%)
Gender	
Man	7 (50)
Woman	7 (50)
Age (years)	
15-40	9 (64.3)
≥40	5 (35.7)
Domicile	
Denpasar	6 (42.9)
Badung	5 (35.7)
Gianyar	1 (7.1)
Karangasem	2 (14.3)
Laterality	
Unilateral	6 (42.9)
Bilateral	8 (57.1)

Table 2. Clinical features of neuritis optic

Karakteristik	**n (%)**
Neuritis Type	
Typical	1 (7.1)
Atypical	13 (92.9)
Chief complaint	
Blurred vision	8 (57.1)
Blurred vision and ocular pain	6 (42.9)
Onset	
<=1 week	8 (57.1)
>1 week	6 (42.9)
Etiology	
Multiple sclerosis	1 (7.1)
NMOSD	3 (21.4)
SLE	1 (7.1)
Infection	8 (57.1)
Idiopathic	1 (7.1)

Table 3. Comparison of visual acuity before and after steroid therapy

	Before steroid therapy	**After steroid therapy**	**p* value**
VA log MAR (Mean±SD)	2,00±0,51	0,93±1,05	< 0,05

Table 4. Comparison of contrast sensitivity before and after steroid therapy

Contrast sensitivity	**Before steroid therapy n** (%)	**After steroid therapy n** (%)	p* **value**
Normal	1 (7.1%)	5 (35.7%)	< 0.05
Visual impairment	3 (21.4%)	3 (21.4%)	
Visual disability	2 (14.3%)	1 (7.1%)	
Unevaluable	8 (57.1%)	5 (35.7%)	

Table 4. Comparison of contrast sensitivity before and after steroid therapy

Color vision	**Before steroid therapy n** (%)	**After steroid therapy n** (%)	p* **value**
Normal	0	5 (35.7%)	< 0.05
Partial color blindness	2 (14.3%)	3 (21.4%)	
Total color blindness	0	1 (7.1%)	
Unevaluable	12 (85.7%)	5 (35.7%)	

***** Wilcoxon test

DISCUSSION

Optic neuritis is a demyelinating condition of the optic nerve that decreases visual function. In typical optic neuritis, activated t cells attack the central nervous system, resulting in inflammatory demyelination by an autoimmune reaction. Inflammatory cytokines are then released in response to this autoimmune reaction. T cells and b cells are also activated to release anti-myelin antibodies that bind to complement and destroy the myelin sheath. No can result from infection, trauma, vascular insufficiency, metastasis, toxins or nutritional deficiencies. Vision function will return to near normal within weeks or months, regardless of treatment. However, vision recovery will be hastened using corticosteroids, based on the long-term results of the optic neuritis treatmant trial (ontt) which included intravenous

methylprednisolone (500-1000 mg per day) for three days followed by oral prednisone (1mg/kg per day) for eleven days.[8,9]

This study shows that optic neuritis patients are dominated by the age group 14-40 years with the average age of 36.7 years. This is similar to the research conducted at Prof. dr. I. G. N. G. Ngoerah General Hospital in 2019-2020, where the majority of subjects were in the age group of 20-30 years and 31-40 years (setiawan, 2021). A research conducted in the uk also showed that the average age of optic neuritis patients was 34.95 □15,6 years (braithwaite et. Al., 2020). A multicenter study in japan also found that the average age of patients was 31.8 years and increased in the 40s, although the mechanism was not explained in detail.[9]

In this study, it was found that the percentage of gender on optic neuritis patients were equal between males and females. This is different from the study conducted at Prof. dr. I. G. N. G. Ngoerah General Hospital in 2010-2011, where 65.2% of the subjects were female.[10] This is in contrast to the study of dogahe et al. Who found that the ratio of women to men was about 3:1 in patients with optic neuromyelitis[11]. The study by ducloyer also found that 77% of patients with multiple sclerosis-related optic neuritis were female.[12] The argument is that many genes associated with immune regulation are located on the x chromosome; although divisions lead to a more diverse gene pool to respond to infection, this may increase susceptibility to autoimmunity.[13]

In this study, the chief complaint was blurred vision in 57.1% of patients and the rest were blurred vision along with eye pain. Bilateral cases were 57.1%, and the baseline visual acuity was logmar 2,00±0,51. Compared to the study conducted at the Prof. dr. I. G. N. G. Ngoerah General Hospital in 2010-2011, the most common complaint was blurred vision, a greater number of unilateral cases, and the best baseline visual acuity was logmar 1.59±0,47.[10] Meanwhile, a study in 2023 found a clinical presentation of sub acute monocular vision loss with or without eye pain, ranging from mild blurred vision to no light perception.[11] Another multicenter international study also found most baseline visual acuity was 1/300.[14]

In this study, among 14 cases who received steroid therapy, the visual acuity after steroid therapy improved in 11 cases and the rest remained unchanged. The baseline visual acuity was logmar 2.00±0.51, and after steroid therapy it was logmar 0.93±1.05. The baseline contrast sensitivity of the majority of patients could not be evaluated. After receiving steroid therapy, 5 of them could reach normal, 3 cases of visual impairment, 1 case of visual disability, and 5 cases remained unevaluable. The baseline color vision of the majority of cases were unevaluable. After receiving steroid therapy, 5 cases could achieve normal, 3 cases were partially color blind, 1 case was totally color blind, and 5 cases remained unevaluable.

A study at Prof. dr. I. G. N. G. Ngoerah General Hospital in 2019-2020, among 24 cases who received ONTT therapy, 14 cases experienced sharp visual improvement after one month, and the rest remained. Color deficiency and contrast sensitivity also improved within the first month after ONTT therapy.[15]

Theoretically, the degree of visual impairment can vary significantly depending on the etiology. In idiopathic optic neuritis and MS-related optic neuritis, the loss of visual acuity in high contrast is moderate, with most patients having visual acuity better than 20/200. In contrast, optic neuritis associated with NMOSD or MOG-IgG often shows worse vision loss below 20/400. The degree of visual impairment associated with infectious, granulomatous and paraneoplastic optic neuropathies varies depending on the extent and duration of the disease.[16]

High-dose corticosteroid administration is standard therapy for acute optic neuritis. In the ONTT study, IV methylprednisolone (1000 mg/day for 3 days), followed by oral prednisone (1 mg/kg/day for 11 days) hastened visual recovery but did not improve functional outcomes. IV immunoglobulin (IVIg) and plasma exchange have been evaluated in patients with optic neuritis refractory to high-dose corticosteroid treatment. Although the frequency of responders varies, most optic neuritis patients treated with plasma exchange experience improvement in visual function. If infection is prominent in the differential diagnosis of patients with optic neuritis, it is wise to start appropriate antibiotic therapy as soon as possible. Symptomatic therapy with corticosteroids can be started simultaneously, unless there are contraindications. The side effects of corticosteroids should be weighed against their modest benefits in the treatment of optic neuritis. Steroid treatment is an appropriate, but not mandatory, therapeutic option.[16,17]

According to a study in Finland in 2008-2012, in addition to initial visual acuity, optic disc edema and optic nerve lesions seen on MRI were associated with worse prognosis. This was also found in NMO cases with positive AQP4 antibody results, poor prognosis was associated with young age and Afro-Caribbean ethnicity. In this study, there were 3 patients with initial visual acuity <3/60 and contrast sensitivity vision with visual disability category who did not experience improvement after ONTT therapy. And there was also 1 female NMO patient aged 26 years with positive results for AQP4 antibodies.[18]

Currently, there are no controlled trials that answer the question of what to do next if visual acuity does not improve. Treatment options in such cases are documented in no more than individual case studies and small case series. In most cases of persistently poor visual acuity, the treatment given initially is given a second time, sometimes in multiple doses and/or for a longer duration. The final option for acute treatment is plasmapheresis, which is sometimes very effective. This should be done within six weeks of the onset of the disease. The decision to or not to perform plasmapheresis is a difficult one, as spontaneous improvement can occur as late as two months after the onset of disease. If there is evidence of optic neuromyelitis rather than ordinary optic neuritis, methylprednisolone is generally given in higher doses and for longer periods; if no improvement occurs, then early plasmapheresis is performed. However, this treatment approach is currently not supported by randomized, blinded trials.[17-20]

CONCLUSION

This study showed differences in visual acuity, contrast sensitivity, and color vision in patients with optic neuritis before and after steroid therapy, whereas after steroid therapy the clinical features improved. It is expected that the results of this study can be a reference for the community and health workers to conduct future research and provide early. management.

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