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ELECTROPHYSIOLOGICAL AND IMAGING ANALYSIS IN OPTIC NEURITIS

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ABSTRACT

Background: To study the electrophysiological and MRI changes in patients with Optic Neuritis. **Design**: Prospective study. **Material And Method**: A prospective single institutional study was conducted on 52 eyes of 49 patients having Optic neuritis who underwent electrophysiological and imaging (contrast-enhanced MRI) analysis. **Results**: The study revealed that 26.92% (14) of patients had contrast enhancement in Contrast (Gd) enhanced MRI whereas 73.08% (38) had no contrast enhancement on MRI. Also, it was revealed that the Visual Evoked Potential was abnormal in 86.54% (45) patients with increased p100 latency, deformed waveform in 7.69%(4) patients, and normal study in 5.77% (03) patients. **Conclusion:** From our study, it was concluded that most of the Optic Neuritis patients have abnormal VEP and their contrast enhancement in Gd- enhanced MRI in fewer patients. The contrast enhancement in fewer patients may be because of the smaller sample size. To determine the systemic and functional activity of the optic nerves and to expose the type of presentation, a detailed diagnostic workup with clinical characteristics, ophthalmological observations, electrophysiological testing, and imaging techniques (MRI) is useful.

KEYWORDS: Optic Neuritis, VEP, MRI.

INTRODUCTION

Optic neuritis (ON) is described as the inflammation of the optic nerve, which is mostly idiopathic. It is also suggested to be associated with demyelinating lesions, autoimmune disorders, infectious and inflammatory conditions. Diagnosis of ON is based on the clinical examination. MRI (magnetic resonance imaging) is suggested to evaluate the risk of multiple sclerosis as clinically silent white matter lesions may be evident. Visual evoked potentials show delayed latencies in the involved eye (long after an acute attack, acute stage shows absent waveforms). The classic presentation with visual loss, periocular pain, dyschromatopsia, and unilaterality define a typical form of optic neuritis. The natural course of most unilateral acute optic neuritis is described as sudden onset of visual loss associated with pain on eye movements, which reaches its maximum deficit over 1-7 days.[1] Adult-onset optic neuritis is typically unilateral and is commonly linked to multiple sclerosis. In adults, simultaneous or sequential bilateral acute optic neuritis has been considered rare, particularly in individuals without known systemic inflammatory or autoimmune disorders.

Impairment of the optic nerve may cause an abnormal increase of the latency of visually induced AEEG-responses, specifically the latency of the P100 peak of the pattern reversal Visual Evoked Potential (VEP). VEP abnormalities are thought to be caused by dysfunctioning of the afferent visual pathway. The afferent pathway is thought to be identical in mediating both evoked responses at least up to the posterior third of the optic tract.[2] In general, VEP abnormalities in Optic Neuritis (ON) have been described extensively. The optic nerves are visualized on CT and MRI. For most neuro-ophthalmic disorders, MRI is superior to CT.[3] MRI is the recommended study in inflammatory, infiltrative and compressive optic neuropathies, with fat suppression technique useful in excluding intraorbital optic nerve enhancement. This study aims to evaluate the changes in VEP and MRI in patients with Optic neuritis.

MATERIAL AND METHOD

This is a Prospective Observational study that was conducted in Government Medical College, Srinagar, Department of Ophthalmology. In this study, 52 eyes of 49 patients were included. After obtaining the ethical clearance from the ethical committee of the institution, the study was conducted for a period of two and half years from Aug. 2017- March. 2020. All patients with sudden unilateral or bilateral visual loss of less than a month, having RAPD or dyschromatopsia with the swollen or normal optic disc were included in the study. The patients with age less than 15 years and having ON due to any definite cause were excluded from the study.

The patients were diagnosed based on characteristic history and clinical examination. Ophthalmic

examinations including slit-lamp examination and pupillary reactions (RAPD) were noted. Aided visual acuity was measured for distance vision by Snellen chart at 6m. Those unable to read any letters at one meter were further examined by counting fingers, identifying hand movements, or perceiving light. Color vision, where visual acuity and central visual function allows, were recorded using Ishihara pseudoisochromatic color vision plates. Visual field determination, where aided visual acuity permitted, was recorded for both eyes by Humphrey automated perimetry. The visual evoked potential was also done. VER was recorded by the checkerboard pattern reversal technique. The patients were seated in a dark quiet room, one meter in front of a TV screen which subtended an angle of 15^{o} sub at the patient's eye and displayed a checkerboard pattern of varying check sizes. A flash stimulus with the apparent disappearance of a checkerboard pattern of 60 minutes of arc was also used as a stimulus. Three silver electrodes were used with the recording electrode at the forehead and the grounding electrode at the mastoid region and hundred responses were analyzed for 250 milliseconds each.

The implicit time of the waveform was recorded from the onset of the stimulus to the peak of the major positive wave in milliseconds. The amplitude was recorded from the trough preceding the major positive wave to its apex in microvolts.

Neurological examinations including orbital and brain MRI was performed with gadolinium (Gd) preferably within two weeks after the onset of symptoms. Contrast enhancement of the optic nerve is a sensitive finding in acute Optic Neuritis but does not correlate with the degree of visual recovery.

RESULTS

The study revealed the following results:

Table 1: Optic Nerve Enhancement.

Optic Nerve Enhancement	Number of eyes	Percentage
No enhancement	38	73.08
Enhancement present	14	26.92
Total	52	100

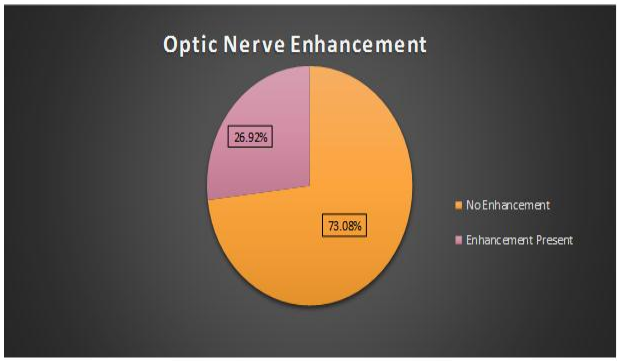

Figure 1: Optic Nerve Enhancement.

From Table 1 and Figure 1, it was revealed that only 26.92% (14) of patients had contrast enhancement in Contrast (Gd) enhanced MRI whereas 73.08% (38) had no contrast enhancement on MRI.

Table 2: Visual Evoked Potential.

Parameter	Number of eyes	Percentage
Increase p100 latency	45	86.54
Deformed waveform	04	7.69
Normal study	03	5.77
Total	52	100

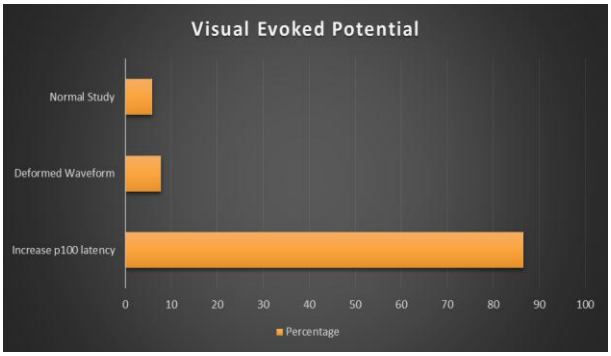

Figure 2: Visual Evoked Potential.

Table 2 And Graph 2 revealed that the Visual Evoked Potential was abnormal in 86.54% (45) patients with increased p100 latency, deformed waveform in 7.69%(4) patients, and normal study in 5.77% (03) patients.

DISCUSSION

The visual evoked potential (VEP) is primarily a relatively large, positive polarity wave generated in the occipital cortex in response to visual stimulation. It measures the conduction time of neuronal activity from the retina to the occipital cortex and is used clinically as a measure of the integrity and function of that pathway. The optic nerve is the primary structure examined. The VEP is of large enough voltage that it can be seen occasionally on a routine EEG as an occipital waveform within the first 150 ms after a single photic stimulus. The standard VEP averages many such waveforms, time-locked to the stimulus. Of primary interest is the latency of the positive wave at a midline occipital EEG electrode, usually at approx 100 ms after stimulation, called the P100. This P100 peak is usually easy to recognize and measure. In our study, most of the patients 86.54% (45) had an increase in p100 latency, and the deformed waveform was seen in 7.69% (04). The normal study was seen in 5.77%(03). In cases of optic neuritis with visual recovery to 20/40 or better, **Griffin JF et al**[4] reported an abnormal VEP in 93% of 30 eyes. Another study conducted by **Ardeb GB et al**[5] reported abnormal VEP in 63% (24 eyes) of patients. **Shahrokhi F et all**[6] reported an abnormal VEP in 95% (58 eyes) of patients with resolved Optic Neuritis and **Byne et al**[7] reported abnormal VEP in 71% (42 eyes) of patients. All these studies are as per the findings of our study. **Neto et al**[8],

in 2013, conducted a study where 19 patients, with Neuromyelitis optica (NOM) diagnosis, with 74% being Afro- Brazilians, underwent a VEP study. Of the 38 eyes examined, 18 (47.37%) showed no visual evocable response. Of the 20 eyes (52.63%) where VEP responses were detected, 18 (90%) had P100 wave latency within a normal range, while only 2 (10%) had an increase in the latency of this wave. Regarding P100 wave amplitude, 11 of the 20 eyes (65%) that generated visual responses had values below that considered normal in the study. Seven (35%) had amplitudes ≥ 5.8 μV, being considered normal. In 65% of the 20 eyes where the visual response was evocable, a reduction of the P100 wave amplitude was found, with normal latency. VER may be an added parameter to complement the findings gained from the subjective tests like colour vision, field charting, and pupillary reaction, each of which is important in its own right. It may be an indicator of previous attacks as seen by the persistent increased VER implicit time.

Magnetic Resonance Imaging (MRI) is routinely used in patients with optic neuritis in which the indication of previous demyelinating episodes in the brain is the main target. The occurrence of three or more T2-weighted white matter lesions or fluid attenuation inversion recovery picture is consistent with the early onset of multiple sclerosis and is most commonly used as the starting criteria for high-dose steroids and b-interferon as a treatment for Optic neuritis. In certain cases, MRI is not needed to diagnose and discern optic neuritis from other typical optic neuropathies if stringent clinical standards are used (**Optic Neuritis Research Group,1991).**[9] Patients with conditions such as non-arteritic anterior ischaemic optic neuropathy or acute compressive neuropathy due to pituitary tumor or cerebral aneurysm or posterior scleritis, however, may sometimes not have the usual characteristics that distinguish optic neuritis from these disorders. In these cases, misdiagnosis can be avoided by MRI. Several small series have shown that there is an irregular enhancement of the optic nerve afflicted by optic neuritis after intravenous gadopentetate dimeglumine (gadolinium) administration**(Guy J et al1992).**[10]

We conducted gadolinium-enhanced optic nerve MRI in all 49 patients in our sample (52 eyes). Optic nerve enhancement was seen in 14 eyes (26.92%) out of 52 eyes, and no enhancement was seen in 38 eyes (73.08 percent). It could be because of our small sample size. The most prominent site of enhancement was the orbital optic nerve segment. Out of 455 patients, optic nerve enhancement on MRI was shown in 77 percent of the eyes in the Optic Neuritis Treatment Trial (**ONTT**).[9] Another study by **Kupersmith MJ et al.**[11] observed optic nerve improvement in 94% of their series of 101 patients. They also observed that the most frequent site of enhancement (43 percent) was the orbital section, which was close to our finding. Even though the number of eyes with optic nerve enhancement was lower in our research. Our results confirm the usefulness of the orbit suppressed by gadolinium-enhanced fat-suppressed MRI of the orbit reported in a small series(**Guy J et al**).[10] The Gadolinium-enhancement of the Optic Nerve is not diagnostic of Optic Neuritis because the enhancement can be noticed in other conditions like a neoplastic invasion, cytomegalovirus, radiation vasculopathy, systemic lupus erythematosus, and rheumatoid arthritis-associated optic neuropathy as well. The Gadolinium-enhanced MRI is especially useful in cases where there have been no previous episodes of demyelination or White matter lesions.

CONCLUSION

Typical or atypical optic neuritis requires a cautious and careful medical approach, since the disorder may have hazardous vision effects. To determine the systemic and functional activity of the optic nerves and to identify the type of presentation, a detailed diagnostic workup with clinical characteristics, ophthalmological observations, electrophysiological testing, and imaging techniques (MRI) is useful. Risk of the occurrence of extreme neurological disorder in normal type whereas the atypical appearance of related infectious etiologies needs adequate diagnostic and prognostic perspectives.

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