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ORIGINAL RESEARCH

Etiological Spectrum, Clinical Profile and Prognosis in Optic Neuritis: An Experience of 40 Patients from Tertiary Care Institute

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ABSTRACT

Introduction: Present study was conducted to study clinical features, short term prognosis and etiological spectrum of optic neuritis (ON) in India.

Materials and Methods: We enrolled fourty consecutive cases of ON. Clinical manifestation, blood and Cerebrospinal fluid studies, brain and spinal cord Magnetic Resonance Imaging(MRI) and Visual Evoked Potential(VEP) were done in all cases on inclusion in study. Intravenous methylprednisolone was given to all cases. They were all followed up upto six months. Outcome was assessed by Snellen's chart.

Results: Female gender was more affected than male (2.3:1). Mean age was 25.6±7.6 (16-55). Papillitis (80%) was more common than retrobulbar neuritis (20%). Bilateral presentation of ON was seen in 32% cases. Multiple sclerosis (MS) was diagnosed in 6(15%) cases, Neuromyelitis optica spectrum disorders (NMOSD) was diagnosed in 14 (35%) cases, Myelin oligodendrocyteglycoprotein antibody associated disorder was seen in 4(10%) cases, idiopathic optic neuritis was seen in 16(40%) cases.

Conclusion: A variety of demyelinating and inflammatory disease cancauseON. NMOSD compared to MS is more common in India.

Key words: Etiological Spectrum, Optic Neuritis, Indian Population, NMOSD.

INTRODUCTION

Optic neuritis (ON) is an inflammatory and demyelinating illness that usually affects one or both eyes. Patients do present with mono or binocular vision loss and periorbital pain. Majority of cases are idiopathic in nature, but variety of inflammatory and demyelinating illness can cause ON. In western literature, Multiple Sclerosis (MS)isthe leading cause of ON but its incidence is low in Indian and Asian population.[1] Indian data are very sparse regarding aetiologyofON.[2,3] There are few studies done in Southeast Asia which showed different clinical profile and prognosis in ON patients.[4]So, Aim of our study was to study and evaluate clinical profile, visual outcome and etiological spectrum in ON patients in Indian population.

MATERIALS AND METHODS

This was prospective observational study conducted in Neurology department of tertiary care centre and for this ethical committee approval was taken.ONpatients were recruited and consent was taken.ON was diagnosed based on history and clinical examination. ONwas defined as an acute onset unilateral or bilateral vision loss less than 4 weeks duration, presence of relative pupillary afferent defect, dyschromatopsia and normal or swollen optic

disc on fundus examination. Other optic neuropathies like ischemic, traumatic, infective, hereditary, toxic and compressive were excluded from study. Patients more than 12 years in agewere included in the study.

Detailed history was taken, which documented onset and progression of vision loss, duration, association with pain or not, previous similar attack and other neurological symptoms. Clinical examination included snellen's visual acuity, papillary evaluation, fundus examination, slit lamp examination, and colour vision examination. Detailed neurological examination was done. Magnetic resonance imaging (MRI)brain and spinal cord with contrast, cerebrospinal fluid(CSF) examination, serum and CSF neuromyelitis optica (NMO)and myelin oligodendrocyte glycoprotein(MOG)antibodylevel were done in all cases. Complete hemogram, erythrocyte sedimentation rate(ESR), chest x ray, serology for syphilis and HIV, antinuclear antibody (ANA) was done in all cases.

All patients were given 3 days course of intravenous methylprednisolone for consecutive days. Follow up was taken at 1 week, 3 months and 6 months. At follow up, Snellen's visual acuity (VA), colour vision, pupillary examination, fundus findings were recorded. Detailed analysis of descriptive data of demographic profile, clinical profile and visual outcome were done. Etiological spectrum of ON was also analysed.

RESULTS

40 out of 52 cases were included in this study. Mean age of presentation was 25.6±7.6 years (16-55 years). Females were more affected than male with the ratio of 2.3:1(28 versus 12). Bilateral presentation was seen in 13 cases (32%). 3 of whom presented with retrobulbar neuritis.

All patients had acute vision loss, whereas pain was seen in 30 cases (75%). 8 patients had retrobulbar neuritis (20%).

32 patients had papillitis while 8 patients had retrobulbar neuritis. All patients had defective color vision and contrast sensitivity at the time of presentation.

MRI brain with orbit with spinal cord with contrast were done in all cases. Out of 40 cases, 25 cases showed contrast enhancement of optic nerve in affected eye. Demyelinating lesions were seen in 6 patients, who subsequently turned out to be MS patients. 6 cases subsequently had attack of myelitis and turned out to be NMOSD. Out of 13 who had bilateral presentation, 4 turned out to be MOGAD cases based on positive MOG ab level in serum.

25 cases on follow up showed good recovery in the vision with achieving 6/6 VA level. 13 cases showed poor recovery having vision <6/60. 2 cases showed partial improvement. Out of 13 cases, 3 cases were of MOGAD, 9 cases were of NMOSD and 1 case was of idiopathic ON.

Recurrence cases of ON were seen in cases of 4 out of 14 cases NMOSD, 2 out of 3 cases of MOGAD and in 1 MS patient during limited follow up of 6 months.

Table 1: Basic clinical profile of all ON patients

Total number of patients	40
Age (mean years)	25.6±7.6 years(16-55)
Female and Male	28:12
Pain on presentation	30 cases
Papillitia and Retrobulbar neuritis	32:8
Bilateral ON cases	13 cases
Range of hospitalization in days	3-7 days
Idiopathic optic neuritis cases	16 cases
Good recovery	25 cases

ON= Optic neuritis

Table 2: MRI and CSF findings in all ON patients

MRI optic nerve abnormality	25 cases
MRI spinal cord abnormality	6 cases
MRI brain demyelinating lesions	6 cases
CSF protein (mean)	55±5.2 mg/dl
CSF cells(mean)	20±5 cells/cumm(2-55)
CSFNMO antibody positive	8 cases
CSF MOG antibody positive	4 cases

MRI=Magnetic Resonance Imaging, CSF= Cerebrospinal fluid, ON=Optic neuritis, NMO=Neuromyelitis optica, MOG= Myelin Oligodendrocyte glycoprotein

Table 3: Recovery in all patients upto 6 month follow up

Recovery	Number of cases out of 40
Good recovery	25 cases
Poor recovery	13 cases
Partial recovery	2 cases

Table 4: Etiology of ON patients

Etiology	Number of patients
Idiopathic ON	16
NMOSD	14
MS	6
MOGAD	4

ON=Optic neuritis, NMOSD=Neuromyelitis optica spectrum disorder, MS=Multiple sclerosis, MOGAD=Myelin oligodendrocyte glycoprotein associated disorder

DISCUSSION

The Optic Neuritis Treatment Trial (ONTT) was taken place to understand role of corticosteroids inONpatients. It was pioneer study in on group of patients which showed light inONpathophysiology and management ofONpatients. In western literature, most of ON cases are because of MS.[5] But in Asia, picture is not same, there are very few studies depicting various etiologies ofON in Asian population.[3,6-8]There are very few studies in India which showed ON patients' clinical profile and prognosis. So, our effort is to find out pattern ofclinical profile of ON patients and its prognosis from western part of India.

Age of presentation and female preponderance are similar findings noted in previous studies.[1,3,4,6,7] Althoughbilateral presentation is higher in Asian group of patients and in current study too, as compared to Europe.[1,3,4,6,7]That could be because of increase NMOSD and MOGAD patients found in India as compared to western population. Papillitisand absence of pain is more common in Asian population.[1,3,4,6,7]

Good prognosis was documented in idiopathic ONandMS patients. ON due to NMOSD and MOGADhad poor visual outcome because of severity of illnesson optic nerves and pathophysiology focused on myelin sheath of optic nerveas well as recurrence nature of disease.So in Asian studies prognosis is not good as compared to western studies.[8,9]

Bilateral involvement ofoptic nerves were more common in NMOSD and MOGADas compared to idiopathic ON andMS. These bilateral involvement patients were eventually turned out to be either NMOSD or MOGAD. These patients do have abnormal MRI of optic nerve showing marked contrast enhancement and also having abnormal brain and spinal cord lesions. Recovery is also poor inthesegroup of patients.

Recurrence was not seen in idiopathic ON as compared to MS, NMOSD and MOGAD. Recurrent ON is suggestive of active inside pathophysiology which may be MS or NMOSD as found in previous studies.[10]

MRIbrain, optic nerves and spinal cord are very essential in evaluation in cases ofON group of patients. In our study we found demyelinating lesions of MSin 6 of our patients. In NMOSDgroup of our patients, therewereoptic nerve enhancement as well as demyelinating brainlesions in area postrema, brainstem, hypothalamus.

Table 5: Comparison of clinical profile ofONpatients in Asian regions with ONTT

	ONTT study	Present study	New Delhi (Saxena et al.)	Singapore (Lim et al.)	Taiwan (Lin et al.)	Japan (Wakakura et al.)
Age range in years	18-46	16-55	15-58	12-70	7-80	14-55
Female: Male ratio	3:1	2.3:1	2.2:1	3:1	1:1	3:1
Papillitis (n%)	35%	80%	53.5%	60%	53%	50%
Pain (n%)	92%	75%	73%	71%	59%	56%
Bilateral (n%)	Nil	32.5%	19%	16.4%	34.9%	Nil
MS (n%)	30.1%	15%	5%	25.5%	14.7%	5.6%
Recurrence (n%)	28%	17.5%	16%	29%	33.95%	Not reported

ON= Optic neuritis, ONTT=Optic neuritis treatment trial, MS=Multiple sclerosis

Saxena et al has done similar study in north India and did find that etiology and clinical profile was having difference in Indian population compared towestern world.[3]Even some more studies done in Southeast Asia also revealed the same point as we want to emphasize in our study that clinical profile and etiology are different in Asia as compared to Europe in ON patients.[4,6,7]Saxena et al compared difference in clinical profile of patients of ON in Southeast Asia as compared to Europe. In our study, we also comparedclinical profile of ON patients in Southeast Asia and Europe. Because of advances in science and more accurate laboratory investigation, we observedthatNMOSD is more prevalent than MS in Asian people.

The limitation of our study was short follow up period. Despite that we observed thatONin Asian population is having different etiological and clinical profileas compared towestern population. Certain environmental factors, ethnic background and genetic predisposition which could play a role in the difference in clinical and etiological profile of Asian population, remains to be studied and evaluated.

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