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A PROSPECTIVE STUDY OF ISOLATED VISUAL LOSS AND NEUROLOGICAL CAUSES

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Abstract

Introduction: Vision impairment can be a reason of severe disability for an individual. Evaluating patients with neuro-ophthalmic symptoms may sometimes seem to be a daunting task as visual deficits in such patients involve complex neuro-anatomic pathways. The vision can be affected from a myriad of local ophthalmologic conditions and those affecting the neural structures conveying visual information from retina to brain. Such neuro-ophthalmologic diseases can be due to damage at any location from optic disc, optic nerve, optic chiasm, optic tract to optic radiation and occipital cortex.

Materials and Methods: This was a prospective study conducted from August 2022 to July 2023. 96 patients met the criteria and were included in the study. All the patients were informed about the study and their consent was taken. Detailed history was taken regarding the mode of onset, progression of visual impairment and associated symptoms like pain. Clinical examination including fundus was carried out in all patients. Visual acuity was assessed by Rosenbaum's pocket screener and visual fields by confrontation method at the bedside.

Results: Majority of the patients were in the category of 10-20 years' age group (38 patients; 39.58%). There was only one patient above 50 yrs. of age indicating that visual impairment of neurological origin is predominantly a disease of younger age group. Most studies showed a female-to-male ratio of 3:2, while the present study showed almost equal distribution. Majority of the patients had acute visual loss (41.66%), while 35.41% had a chronic variety of visual impairment. The remaining patients had sudden visual loss (8.33%) and sub-acute visual loss (14.58%). Sudden visual loss was seen in 8 patients due to stroke and consisted of visual field defects, either hemianopia or altitudinal defect. All patients belonging to this group were above 40 years of age and did not have any recovery during followup.

Conclusion: The commonest cause of acute unilateral and bilateral visual loss was demyelinating optic neuropathy. Chronic visual loss can also be due to demyelinating optic neuropathy as seen in two patients. The patients with acute and sub-acute visual loss without any identifiable cause can be empirically treated with steroids if not contraindicated. Intravenous

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methylprednisolone for 3 days followed by 11 days of oral prednisolone is the best mode of treatment for demyelinating optic neuropathy. Recurrence of optic neuropathy was not seen during the follow-up period of the study. Stroke is the commonest cause of sudden visual impairment.

Key Words: Vision impairment, neuro-anatomic pathways, optic radiation and occipital cortex.

INTRODUCTION

Vision impairment can be a reason of severe disability for an individual. Evaluating patients with neuro-ophthalmic symptoms may sometimes seem to be a daunting task as visual deficits in such patients involve complex neuro-anatomic pathways.[1] The vision can be affected from a myriad of local ophthalmologic conditions and those affecting the neural structures conveying visual information from retina to brain. Such neuro-ophthalmologic diseases can be due to damage at any location from optic disc, optic nerve, optic chiasm, optic tract to optic radiation and occipital cortex.[2]

Various causes of neurogenic vision loss include: Optic neuropathy (demyelinating, ischemic, toxic, infectious, secondary to chronically raised intracranial pressure (ICP)), compression of the visual pathway because of space occupying lesions and tubercular arachnoiditis in the optochiasmatic region[3], ischemic involvement of the post-chiasmatic visual pathway as in case of stroke, cerebral venous thrombosis (CVT), cortical vision loss due to various etiologies such as stroke, posterior reversible encephalopathy syndrome (PRES), subacute sclerosing panencephalitis (SSPE) etc.4

Majority of the cases have visual impairment due to anterior visual pathway disorders. History and clinical examination remain the crucial steps in identifying these diseases. The loss can be unilateral, bilateral or a field defect. Anterior visual pathways include retina, optic nerves and optic chiasm and the involvement results in moderate to profound visual loss along with impaired colour vision and loss of pupillary reflexes.[5] Posterior visual pathways include optic tracts, lateral geniculate body, optic radiation and occipital cortex. Their involvement results in field defects with relatively preserved visual acuity.

MATERIALS AND METHODS

To evaluate the neurological causes of visual loss in patients attending the tertiary care hospital in South India.

Inclusion Criteria

Patients presenting with unilateral or bilateral visual impairment of sudden, acute, sub-acute or chronic onset were included in the study.

Exclusion Criteria

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1. Patients in whom the visual impairment is due to ocular cause were excluded.

2. Patients in whom the visual impairment is associated with other neurological symptoms were also excluded.

Presence of Headache was not an Exclusion Criterion

This was a prospective study conducted from August 2022 to July 2023. 96 patients met the criteria and were included in the study. All the patients were informed about the study and their consent was taken. Detailed history was taken regarding the mode of onset, progression of visual impairment and associated symptoms like pain. Clinical examination including fundus was carried out in all patients. Visual acuity was assessed by Rosenbaum's pocket screener and visual fields by confrontation method at the bedside.

The mode of onset of visual impairment was defined as sudden (Maximum severity within a few minutes), acute (Maximum severity within a few hours to 4 weeks), sub-acute (Progression from 4-8 weeks) and chronic (Progression beyond 8 weeks). Routine blood parameters were done in all the patients including HIV serology. Cerebrospinal Fluid (CSF) examination including biochemical analysis and cytology was done in 10 patients with chronic visual loss. CSF lactic acid levels were estimated in 4 patients. Cranial Computed Tomographic (CT) scan was done in 36 patients and Magnetic Resonance Imaging (MRI) was done in 12 patients. Pattern-shift visual evoked potentials were recorded in all patients. In the subjects whose visual acuity was so low that they could not concentrate on the monitor, the VEP test was done using goggles.

The patients who had acute demyelinating optic neuritis were treated with intravenous methylprednisolone 30 mg/kg/day for 3 days followed by 11 days of oral prednisolone 1 mg/kg/day for 11 days as per the Optic Neuritis Treatment Protocol. All such patients were followed up for 6 months at regular intervals. The data was analysed and the results tabulated (Table).

RESULTS

Majority of the patients were in the category of 10-20 years' age group (38 patients; 39.58%). There was only one patient above 50 yrs. of age indicating that visual impairment of neurological origin is predominantly a disease of younger age group.

Most studies showed a female-to-male ratio of 3:2, while the present study showed almost equal distribution.

Majority of the patients had acute visual loss (41.66%), while 35.41% had a chronic variety of visual impairment. The remaining patients had sudden visual loss (8.33%) and sub-acute visual loss (14.58%). Sudden visual loss was seen in 8 patients due to stroke and consisted of visual field defects, either hemianopia or altitudinal defect. All patients belonging to this group were above 40 years of age and did not have any recovery during followup. Among the 4 patients with

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sudden visual loss, the fundi were normal in three and optic atrophy was present in one patient who had anterior ischaemic neuropathy in addition to an occipital infarct.

Age	No of patients
Below 40 years	80
Above 40 years	16

Table 1: Age distribution

Gender	No of patients
Male	46
Female	56

Table 2: Gender Distribution

Mode of onset	No of patients
Sudden	8
Acute	40
Sub-Acute	14
Chronic	34

Table 3: Mode of onset

Visual Loss	No of patients
Unilateral	38
Bilateral	52
Field Defect	6

Table 4: Visual Loss

Visual Acuity	No of patients
Normal (Field Defects)	6
Reduced	4
Finger counting only	46
Perception of light only	36
No perception of light	4

Table 5: Visual Acuity

DISCUSSION

The diagnosis of acute optic neuritis is to be made as early as possible and treatment must be instituted on an emergency basis. Different types of steroid protocols were followed by ophthalmologists and neurologists. Beck and Cleary et al had concluded that intravenous high dose methylprednisolone for 3 days followed by 11 days of oral prednisolone offers the best outcome at 6 months after the initial insult, and also 2 years after the treatment.[6]

In our series, the patients treated with intravenous methylprednisolone made a good recovery compared to dexamethasone or oral steroid therapy Magnetic Resonance Imaging (MRI),

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serologic studies (Such as the antinuclear antibody test and the fluorescent treponemal antibody absorption test), chest roentgenography and lumbar puncture were of limited utility in defining the cause for visual loss other than Optic Neuritis (ON) associated with demyelinating disease.[7] CSF analysis may not be necessary in the routine evaluation of patients presenting with a typical clinical profile of acute ON, and that most CSF tests add little additional information to MRI results for predicting the 2-year development of Clinically Definite Multiple Sclerosis (CDMS).[8]

Most patients retained good-to-excellent vision more than 10 years after an attack of optic neuritis in many studies.[9] Recurrences were more frequent in patients with MS and the recovery from an episode was also less optimal in patients with MS when compared to patients who did not progress to MS. Arnold had opined that in those patients where risk factors for development of MS can be identified, corticosteroids along with immunomodulating drugs may be given to prevent the progression to CDMS.[10]

CONCLUSION

The commonest cause of acute unilateral and bilateral visual loss was demyelinating optic neuropathy. Chronic visual loss can also be due to demyelinating optic neuropathy as seen in two patients. The patients with acute and sub-acute visual loss without any identifiable cause can be empirically treated with steroids if not contraindicated. Intravenous methylprednisolone for 3 days followed by 11 days of oral prednisolone is the best mode of treatment for demyelinating optic neuropathy. Recurrence of optic neuropathy was not seen during the follow-up period of the study. Stroke is the commonest cause of sudden visual impairment. The visual loss due to stroke has poor prognosis and the treatment of hemianopia due to stroke is still in the experimental stage only. Stroke can also result in only colour blindness, prosopagnosia or micropsia without there being a total blindness. Slowly progressive visual loss needs investigatory workup to exclude intracranial tumour.

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