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Original Article

Short term Visual Outcome of Optic Neuritis in Children: A Single Centre Observational Study

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Abstract:

Optic neuritis in children is rare and different from adult, often resulting in better visual outcomes despite initial severe vision loss. This observational study aimed to assess short term visual outcome of optic neuritis in children at National Institute of Neurosciences and Hospital in Bangladesh. A total of 24 children aged 5- 15 years with acute or subacute loss of vision were evaluated from January 2020 to June 2021. Comprehensive evaluation included history, examination, Cerebrospinal fluid (CSF) study, neuroimaging and anti- NMO Antibody testing. All children received intravenous methylprednisolone followed by oral steroid. Follow up of patients was done at discharge, 1 and 6 months. Mean age was 9.57±2.70 years with female (66.67%) predominance. Bilateral ocular involvement in 79.17% and ocular pain 75% of cases were main presenting features and isolated optic neuritis (79%) was the most common cause. Orbital MRI findings showed optic nerve hyperintensity were more in isolated optic neuritis (33.33%), where ADEM (8.3%) had abnormal brain MRI and ON-NMO (12.5%) had abnormal MRI of spine. Following treatment visual acuity showed improvement in all the eyes at discharge and it was statistically significant. Full recovery (20/20) was seen in 46.50% and 69.8% of cases at 1 and 6 months follow up respectively. Overall, most children responded well to steroid therapy despite poor vision at presentation and significant visual recovery was observed in majority of the cases at 6 months follow-up.

Keywords: Optic neuritis, Childhood, Short term, Visual outcome.

Introduction:

Optic neuritis (ON), a rare inflammatory condition that affects one or both optic nerves in children, impairs vision.[1] Visual impairment is usually occurred due to a swollen and damaged myelin sheath of optic nerves that carry visual information from the retina to the brain.[2] In one population-based study, the incidence of ON was estimated to be 5.1/100000 in adults and 0.5/100000 in children.[3] The hallmark of ON is acute and subacute onset vision loss. Decreased visual acuity, abnormal color perception (especially Red Color Desaturation), central scotoma or reduced Visual Fields are most

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common characteristics of ON. The onset of visual impairment typically begins within a few hours to a few days, followed by gradual improvement over a few days to a maximum of two weeks.[4] Compared to adults, children with ON are more likely to have severe visual impairment at presentation, with the majority of children having 20 / 200 VA or worse.[5,6] Children are also more likely to report bilateral vision loss.

Optic neuritis is a clinical diagnosis based upon the history and examination findings. Children with ON need MRI of brain and orbits with fat suppression with contrast to confirm the presence of optic nerve enhancement and to evaluate for other white matter lesion such as ADEM, multiple sclerosis and NMO.[3] MRI of orbit may be normal. Some patient requires neuroimaging of spinal cord if it is suspected NMO.[3] Lumbar puncture is not essential diagnostic test in ON but should be considered excluding other causes of visual loss.[7] Even though vision loss is very severe in the early stages, the visual prognosis in childhood ON is generally good to excellent. Early diagnosis and appropriate treatment can help prevent long-term complications.

There are limited prospective data on ON in children. Our understanding of the clinical characteristics and natural history of childhood ON primarily based on case reports and retrospective case series. Specific studies on optic neuritis in children are scarce in Bangladesh. This prospective study will help better understanding the clinical profile of optic neuritis in children and may make an opening to find out a better management plan.

Materials and Methods:

This observational study was conducted at the Department of Pediatric Neurology, National Institute of Neurosciences and Hospital, Dhaka, Bangladesh from January 2020 to December 2020. A total 24 children of 5-15 years with first episode of optic neuritis were included using purposive sampling technique. Informed written consent was taken from parents. The clinical diagnosis of optic neuritis was made based on acute or subacute visual loss and one or more of the following: pain with eye movement, a relative afferent pupillary defect, impaired color vision or optic disc swelling. Patients with previous central nervous system (CNS) inflammatory demyelinating episodes, vision loss due to other causes like compressive, vascular, toxic, traumatic, metabolic, hereditary and connective tissue disorders were excluded from the study. The thorough evaluation was done by taking history, physical, neurological and ophthalmological examinations. All visual assessment primarily done at department of pediatric neurology and confirmed by Neuro-ophthalmologist. Best corrected visual acuity in all patients was evaluated using the Snellen's chart, color vision by Ishihara pseudoisochromatic plates and relative afferent pupillary defect (RAPD) by swinging flash light test. Visual acuity of 20/20 was considered as normal vision, 20/30 to 20/40 as mild, 20/50 to 20/160 as moderate, 20/200 as severe, <20/200 (Hand movement, counting finger) as profound visual impairment and no perception of light as total vision loss in this study. Cerebrospinal fluid (CSF) study and immunological study (anti- NMO Ab) were done subsequently. MRI of brain, spine with contrast and MRI of orbit in both axial and coronal view (fat suppression with contrast) was done in all cases. MRI images were reviewed by Neuroradiologist.

Isolated optic neuritis (ON) was considered who had single and isolated episode of optic neuritis with normal brain and spinal cord imaging. ON- Acute disseminated encephalomyelitis (ON-ADEM) was defined as who had ON and features of ADEM clinically and radiologically. ON-Neuromyelitis Optica (ON-NMO) was considered who had ON with 2 or more of the following: i) acute myelitis ii) Spinal MRI lesion extends over three or more segment iii) Brain MRI does not meet criteria for MS iv) Anti-NMO Ab positivity. ON-Clinically isolated syndrome (ON-CIS) was considered who had first attack of ON with monofocal or multifocal CNS symptom without encephalopathy and MRI showed area of white matter demyelination in brain or spine. After evaluation every case were treated with IV MP 30mg/kg/day for 5 days followed by oral steroid 1mg/kg/day for 9 days and tapering over 2 weeks as per protocol of this institute. Follow up was given during discharge (after completion of IV MP), at 1 and at 6 months. At all follow up visit VA, colour vision, RAPD, funduscopic and other neurologic examination findings and further episode of ON if any were recorded. A follow up MRI was done at 6 months. At 6 month follow up, primary end point of the study was to find out the visual outcome among children with ON.

Data was collected by predesigned questionnaire and analyzed by SPSS (version 22.0) and double checked before analysis. Means and proportions of the demographic parameters were calculated. Categorical data was compared with chi-square test. This study was approved by ethical committee of National Institute of Neurosciences and Hospital.

Results:

Mean age was 9.57±2.70 years and the age at disease onset was ≥10 years in 54.2% cases. Majority of them were female (66.67%). Mean duration of symptom at presentation was 8.91± 2.39 (Table I).

Table I: Distribution of study cases according to demographic profile (N=24)

Variable	Frequency (%)
Age (Years)	
Mean Age	9.57 ± 2.70
Age of onset	
<10 years	11 (45.8%)
≥ 10 yeas	13 (54.2%)
Gender	
Female	16 (66.67%)
Male	8 (33.33%)
F:M	2:1
Duration of symptoms at presentation (days)	8.91 ± 2.39

All study cases had visual impairment (100%), ocular pain (75%) and bilateral ocular involvement (79.17%) at presentation. 66.67% had history of viral prodrome. At presentation all patients had reduced vision with majority of the eyes (30) had severe to worse vision loss (20/200 or less). Most of cases had impaired color vision (89.2%). About 14% of affected eyes, color vision was not able to record due to poor vision. Positive RAPD was found in 70.83% of patient. Optic disc edema was seen in most of the eyes (86.05%). Headache, limb weakness, irritability and bowel bladder involvement were other non-ocular presentations (Table II).

Table II: Distribution of study cases according to clinical profile (N=24)

Clinical profile	Frequency (%)
Visual impairment	24 (100%)
Laterality	
Unilateral	5 (20.83%)
Bilateral	19 (79.17%)
Ocular Pain	18 (75%)
Visual acuity	
20/20 (Normal vision)	0%
20/30-20/40 (Mild impairment)	8 (18.50%)
20/50-20/160 (Moderate impairment)	5 (11.60%)
20/200(severe impairment)	14 (32.30%)
<20/200 (Profound impairment)	10 (23.30%)
*NPL (Total visual loss)	6 (14%)
****Color vision**	
Normal	4/37 (10.80%)
Impaired	33/37 (89.20%)
Not Possible	6/43 (13.95%)
Positive RAPD	
Unilateral	8/8 (100%)
Bilateral	9/16 (56.25%)
***Fundus**	
Normal	6/43 (13.95%)
Optic disc oedema	37/43 (86.05%)
Headache	13 (54.2%)
Limb weakness	3 (12.5%)
Irritability	1 (4.2%)
Bladder involvement	1 (4.2%)

*NPL-No perception of light **No of eyes =43

Among the study cases 33.33% had hyper-intensity in the optic nerve on MRI of orbit, 8.3% had abnormal brain MRI and 12.5% had abnormal spine MRI. Bilateral, multifocal and subcortical lesions were common findings on brain MRI, Corpus callosal hyper-intensity were found in 50% cases of abnormal brain MRI. Most of the study cases had normal CSF study, 12.5% had CSF pleocytosis and serum anti-NMO Ab was found in 1 case (Table III).

Table III: Distribution of study cases according to investigation profile (N=24)

Variable	Frequency (%)
Neuroimaging	
MRI of Orbit	
Normal	16 (66.67%)
Hyperintensity in optic nerve	8 (33.33%)
MRI of Bain	
Normal	22 (91.7%)
Abnormal: pattern of lesion (demyelination)	2 (8.3%)

Bilateral and multifocal (Subcortical and cortical)	1 (4.15%)
Corpus callosal involvement	1 (4.155)
MRI of Spine	
Normal	21 (87.5%)
Abnormal (LETM)	3 (12.5%)
CSF study	
Normal	21 (87.5%)
Abnormal	3 (12.5%)
Serum anti NMO Ab (12)	
Seropositive	1/12 (8.3%)
Seronegative	11/12 (91.7%)

Among the study cases isolated ON (79%) were the most common demyelinating disease followed by ON-NMO (13%), ON-CIS (4%) and ON-ADEM (4%) (Figure 1).

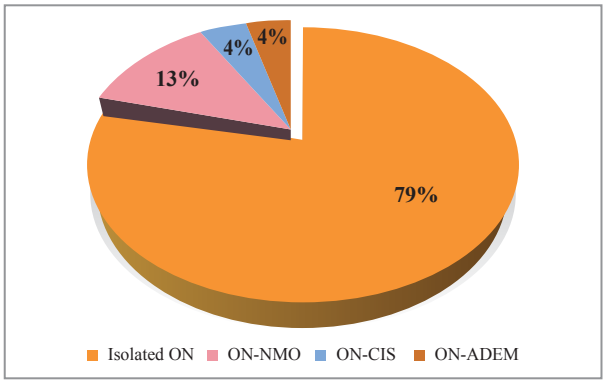
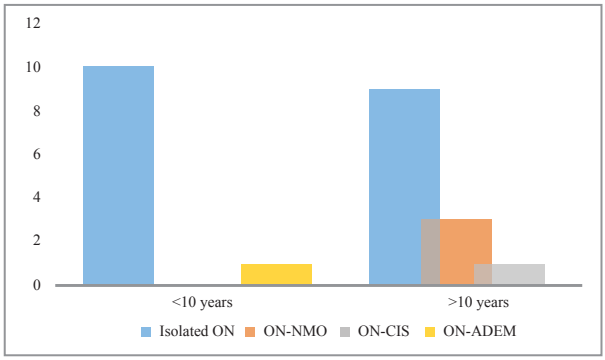


Figure 1: Distribution of study cases according to pattern of optic neuritis (N=24)

All cases of ON-CIS and ON-NMO presented after 10 years. Majority of the cases of isolated ON and ON-ADEM were presented below 10 years. There was not statistically significant (Figure 2).



*Chi-square test p= 0.078

Figure 2: Pattern of ON according to age groups among the study cases (N=24)

On neuroimaging, optic nerve hyperintensity commonly found in isolated ON. ON-ADEM group had bilateral subcortical demyelinating lesions whereas corpus callosal involvement were found in ON-CIS (P=0.000). LETM were prominently found in ON-NMO group (p=0.001). CSF pleocytosis were commonly found in ON-NMO, ON-CIS and ON-ADEM groups (p=0.000). Anti NMO Ab was prominently found in ON-NMO groups (p=0.002) (Table IV).

Table IV: Comparison of neuroimaging and other lab findings of ON groups (N=24)

Variable	Isolated ON (19)	ON-NM0 (3)	ON- CIS(1)	ON-ADEM(1)	p value*
MRI Orbit: Optic nerves					
Normal (16)	14 (87.5%)	2 (12.5%)	0 (0%)	0 (0%)	0.219
Hyperintensity (8)	5 (62.5%)	1 (12.5%)	1 (12.5%)	1 (12.5%)	
MRI of Brain lesions					
Bilateral & Multifocal (2)	0 (0%)	1 (50%)a	1 (50%)b	0 (0%)	0.000
MRI of Spine					
Normal (21)	19 (90.47%)	0 (0%)	1 (4.76%)	1 (4.76%)	0.001
LETM (3)	0 (0%)	3 (100%)	0 (0%)	0 (0%)	
CSF study					
Normal (21)	19 (90.47%)	2 (67%)	0 (0%)	0 (0%)	
Pleocytosis (3)	0 (0%)	1 (33%)	1 (33%)	1 (33%)	0.000
Anti-NMO Ab (1)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	0.002

*Chi-square test a- corpus callosum, b-subcortical, cortical

Most of the cases at presentation had severe to worse vision loss (69.8%). After treatment all the eyes showed improvement in VA during discharge. Among the study cases the frequency of normal vision (20/20) was increased progressively from baseline(00) to discharge in 5 eyes(11.60%), 1 month in 20(46.50%) and 6 months in 30 eyes (69.80%) (Figure 3).

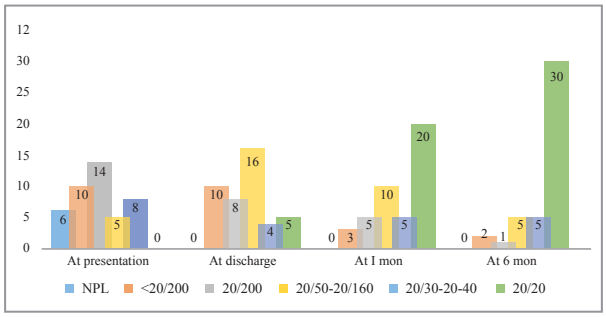
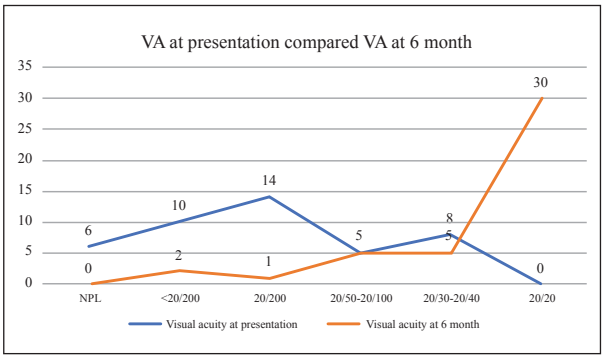


Figure 3: Visual outcomes (VA) among the study cases (n=43 eyes)

Significant improvement of vision at 6 months compared to that at presentation (Figure 4).



Chi-square test (0.04)

Figure 4: Visual outcomes (visual acuity) at presentation and at 6 months among the study cases (n=43 eyes)

Discussion:

In this study, short term visual outcomes of optic neuritis in children in a tertiary care of hospital of Bangladesh was described. Mean age of study cases were 9.57± 2.70 years and female were predominant which is similar to other studies where age ranged from 9.2 to 9.8 years.[8,9] But this contrast with an Indian study (mean age were 12.08 years)[10] which may be due to the variation in the inclusion criteria.

Childhood optic neuritis is considered to be bilateral in nature.[2] In this study, most of the patient presented with bilateral involvement (66.67%), which is consistent with that of other studies where bilateral involvement was reported in the range of 42%-87% of cases.[11-13] In this study 74.41% of cases presented with ocular pain. This finding is differed from the adult study[14], where they found ocular pain in 92% of patients. On the other hand, Lana-Peixoto et al[15] showed pain associated with visual loss in only 10 (37%) which was also not consistent with the present study. Visual acuity is quite variable in children with optic neuritis. An affected eye may have a visual acuity ranging from 20/20 to no perception of light. In this study, visual acuity at presentation was poor in most of the children with over 69.80% (30 of 43 eyes) having visual acuity of 20/200 or worse which is similar to Kumar et al[10] (70.37%). Another study from India[2] reported over 90% of cases having visual acuity of 20/200 or lesser at presentation which is not similar to present study. This disparity may be due to early detection of optic neuritis or small sample size.

Optic disc edema (ODE) is found more commonly in children with optic neuritis.[10] In the current study, Optic disc oedema was present in 86.4% of affected eyes whereas Kriss et al[16] reported 74% of children with ON. This showed that the present study is comparable with other study. In contrast, optic disc oedema was found only in 35% cases of adult onset optic neuritis. Relative afferent pupillary defect was found positive in 70.4% of children with ON cases in this study which is similar to findings of Chang et al.[17] Impaired color vision was found in 74.4% of affected eyes at presentation whereas Kriss et al[16] found color vision defect in 98% of all cases. This dissimilarity may be due to small sample size and poor vision at presentation.

In our study, MRI of brain with orbit and spinal cord were done in all cases. Optic nerve hyperintensity was found in 8 (33.33%) cases at presentation in the current study whereas it was in 16.66% cases an Indian study[5] and Zhou et al[18] found it in 35 (83.33%) which were not consistent with the present study. Isolated ON (79%) were the most common demyelinating disease followed by ON- NMO (13%), ON-CIS (4%) and ON-ADAM (4%) in the current study. Zhou et al[18] found isolated optic neuritis in 76% of cases which is consistent with present study. Sun et al[19] reported ADEM was the most common systemic cause of childhood optic neuritis which differs from the present study. Earlier age of disease onset was found among isolated optic neuritis and ADEM than CIS and NMO. In this study optic nerve hyperintensity was mostly found in isolated ON. At presentation abnormality of brain MRI was found in 12.5% of cases that revealed ADEM (1), CIS (1) in this study. All cases of isolated optic neuritis (19) had normal MRI of brain and spine. MRI of spine revealed abnormality in 3 cases (12.5%) and all of them were longitudinally extended transverse myelitis (LETM) and these all were found in ON-NMO groups.

CSF examination in childhood optic neuritis may show slight pleocytosis and elevated protein content, but more frequently is normal.[11] In present study CSF pleocytosis were found only 3 cases and all in ON-NMO, ON-CIS and ON-CIS groups but none in the isolated ON. Jacobs et al[2] reported nonspecific CSF abnormalities (lymphoc ytosis and elevated protein) in 60% to 80% of their study cases. In our study anti NMO- Ab was found in one patient (ON-NMO) whereas most previous studies of ON in children did not search for serum anit-NMO Ab status due to unavailability of this test.

This study also showed the beneficial effects of corticosteroids in treatment of childhood ON. All the patients in the study received IV Methylprednisolone and showed improvement in visual acuity. This is similar to findings reported by Kumar et al[10] and Brady et al.[20]

In the present study following treatment, visual acuity showed improvement in all the eyes. In this study, full recovery (20/20) was seen in 20 eyes (46.50%) and 30 eyes ((69.8%) at 1 and 6 months follow up respectively. Two eyes (4.70%) had left with VA <20/200 at 6 months follow up. Absoud et al[5] reported severe visual loss (<20/200) in 77% of eyes at presentation and full recovery was seen in 70% of cases after treatment. Singh et al[2] reported 61.4% of eyes having VA better than 20/40 at 6.38 months follow up in their study. The preliminary data of the Paediatric Optic Neuritis Prospective Outcomes Study revealed 76% of children regained 20/20 vision at 6 months follow up following treatment.[21] When compared to other studies, the present study also demonstrated a very good visual recovery in children. During follow-up period, none of the cases developed recurrence of ON in the present study. This may be due to shorter follow up period in the present study.

Conclusion:

In conclusion, this study found that most childhood optic neuritis cases were isolated and often bilateral, with frequent optic disc oedema. Limitations include a small sample size and short follow-up at a single center, which may affect the generalizability of the findings. Larger multicenter studies are needed for broader applicability.

Conflict of interest:

There is no conflict of interest.

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References:

1. Khadse R, Ravindran M, Pawar N, Maharajan P, Rengappa R. Clinical profile and neuroimaging in pediatric optic neuritis in Indian population: A case series. *Indian J Ophthalmol*. 2017; 65: 242-5.

2. Singh D, Phuljhele S, Sharma R, Saxena R, Sharma P, Menon V. Clinical spectrum of pediatric optic neuritis in Indian children. *Ophthalmol Open J*. 2017; 2(2): 20-26.

3. Peragallo JH. How to manage paediatric optic neuritis. *Review of Ophthalmology*. 2018 Apr 4; 15:70-73.

4. Yeh EA, Graves JS, Benson LA, Wassmer E, Waldman A. Pediatric optic neuritis. *Neurol* 2016;87(9 Suppl 2): S53–8.

5. Absoud M, Cummins C, Desai N, Gika A, McSweeney N, Munot P et al. Childhood optic neuritis clinical features and outcome. *Arch Dis Child*. 2011 Sep 1; 96(9):860-2.

6. El-Dairi MA, Ghasia F, Bhatti MT. Pediatric optic neuritis. *Int Ophthalmol Cllin*. 2012; 52(3):29-49.

7. Osborne B, Balcer LJ. Optic neuritis: Pathophysiology, clinical features, and diagnosis. *Upto date*. 2018.

8. Hwang JM, Lee YJ, Kim MK. Optic neuritis in Asian children. *J Pediatr Ophthalmol Strabismus*. 2002;39(1): 26-32.

9. Sri-udomkajorn S, Pongwatcharaporn K. Clinical features and outcome of childhood optic neuritis at Queen Sirikit National Institute of Child Health. *J Med Assoc Thai*. 2011; 94(Suppl 3): S189-S194.

10. Kumar P, Chiranjeevi P. Optic neuritis in Indian children: a one year follow up study. *Opth clin ther*. 2017;1(1):3-5.

11. Morales DS, Siatkowski RM, Howard CW, Warman R. Optic neuritis in children. *J Pediatr Ophthalmol Strabismus*. 2000 Sept 1;37(5):254-59

12. Jarius S, Frederikson J, Waters P, Paul F, Akman-Demir G, Marignier R, Franciotta D, Ruprecht K, Kuenz B, Rommer P, Kristoferitsch W. Frequency and prognostic impact of antibodies to aquaporin-4 in patients with optic neuritis. *Journal of the neurological sciences*. 2010 Nov 15;298(1-2):158-62.

13. Collinge JE, Sprunger DT. Update in pediatric optic neuritis. *Current opinion in ophthalmology*. 2013 Sep 1;24(5):448.

14. Optic Neuritis Study Group. The clinical profile of optic neuritis: optic neuritis treatment trial. *Arch Ophthalmol*. 1991;109:1673-78.

15. Lana-Peixoto MA, Andrade GC. The clinical profile of childhood optic neuritis. *Arq Neuropsiquiatr*. 2001;59 (2-B):311-7.

16. Kriss A, Francis DA, Cuendet F, Halliday AM, Taylor DS, Wilson J, et al. Recovery after optic neuritis in childhood. *J Neurol Neurosurg Psychiatry*. 1988 Oct 1;51(10): 1253-8.

17. Chang YM, Pineless SL. Paediatric optic neuritis. *Semin pediatr neurol*. 2017May;24(2):122-8.

18. Zhou H, Wang W, Xu Q, Tan S, Zhao S, Yang M, et al. Clinical features and visual outcomes of optic neuritis in Chinese children. *J ophthalmol*. 2016;2016:9167361. doi:10.1155/2016/9167361.

19. Sun MH, Wang HS, Chen KJ. Clinical characteristics of optic neuritis in Taiwanese children. *Eye (Lond)*. 2011;25 (11):1457-64.

20. Brady KM, Brar AS, Lee AG, Coats DK, Paysse EA, Steinkuller PG. Optic neuritis in children: clinical features and visual outcome. *JAAOS*.1999 Apr 1;3(2):98-103.

21. Optic neuritis. American Association for Pediatric Ophthalmology and Strabismus. 2019.