Original Article

Etiology and clinical profile of childhood optic nerve atrophy at a tertiary eye care center in South India

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Background: Optic nerve atrophy is an important ophthalmological sign that may be associated with serious systemic conditions having a significant bearing on the overall morbidity of the child. Studies specific to etiology of childhood optic atrophy are scarce, this being the first such study from India to the best of our knowledge. **Aim:** The aim was to analyze the clinical features and etiology of diagnosed cases of optic nerve atrophy in children <16 years of age. **Materials and Methods:** Retrospective review of records of children diagnosed with optic nerve atrophy between the ages of 0 and 16 years from 2006 to 2011. **Results:** A total of 324 children (583 eyes) were identified. Among these 160 (49%) presented with defective vision, 71 (22%) with strabismus, 18 (6%) with only nystagmus. Rest had a combination of two or three of the above symptoms. Sixty-five patients (20%) had a unilateral affection. Hypoxic ischemic encephalopathy seen in 133 patients (41%) was the most frequent cause of childhood optic atrophy, followed by idiopathic in 98 (30%), hydrocephalus in 24 (7%), compressive etiology in 18 (5%), infective in 19 (6%), congenital in 6 (2%), inflammatory in 5 (2%) patients, respectively. **Conclusion:** Hypoxic ischemic encephalopathy appears to be the most common cause of optic atrophy in children in this series. The most common presenting complaint was defective vision.

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Any insult occurring primarily to the anterior visual pathway results in optic atrophy through retinal ganglion cell loss. Posterior visual pathway involvement may also cause atrophy due to transynaptic degeneration.[1] Various causes of optic atrophy in children have been described, but studies are limited.[2-4] There are few population-based studies in India on the causes of childhood blindness.[5-7] A few published studies, mostly conducted at blind schools, establish optic atrophy as the cause of visual impairment in 7–9% of these children.[5-10] The etiology of childhood optic atrophy, however, has not been described. Although not a major cause of blindness in children in India, the importance lies in its association with potentially life-threatening causes, as well as its long-term implication on visual prognosis and overall development of a child. Knowledge of the presentation and causes of optic atrophy in children is, therefore, necessary for appropriate and timely work-up, investigation and intervention.

In 1968, Costenbader and O'Rourk[2] examined a series of children with optic atrophy up to 12 years of age. They were able to establish an etiology for only 50% of the cases, hereditary being the most common. Repka and Miller[3] were able to establish a diagnosis for 89% of cases of optic atrophy in their series of children seen between 1978 and 1987. In their study, an intracranial space occupying lesion (ICSOL) was the major cause of optic atrophy. In 2000 Mudgil and Repka identified the etiology for 96% of children with optic atrophy between 1987 and 1997. Perinatal insults were found to be the leading cause.[4] This illustrates an evolving picture where advances in investigative modalities, as well as changes in childhood morbidity and mortality as a consequence of changing treatment outcomes, has affected the clinical profile of optic atrophy. There has been no similar study in India to the best of our knowledge.

The present study is aimed at describing the etiology and clinical features of cases of optic atrophy in children <16 years of age presenting to our institute.

Materials and Methods

This retrospective case series comprises of children <16 years of age diagnosed with unilateral or bilateral optic atrophy, presenting in the period from November 2006 to October 2011. The diagnosis of optic atrophy was made on the basis of fundus examination and presence of optic disc pallor by trained neuro-ophthalmologists and pediatric ophthalmologists.

A detailed clinical history was obtained, and complete ophthalmological examination was done for every patient. Clinical data of patient age, gender, presenting complaint and associated systemic conditions were recorded. Relevant history such as the birth and perinatal history, family history of ocular or systemic disease, and any prior investigations and treatment were noted. The best-corrected visual acuity (VA) by age appropriate charts (converted to Snellen's equivalent), anterior segment evaluation including ocular motility, alignment and presence and type of nystagmus if any, and posterior segment examination, including optic disc findings were recorded. Additional investigations such as neuroimaging (computed tomography [CT] and magnetic resonance imaging [MRI]), Humphrey visual fields, visual

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evoked potentials, electroretinogram, and blood investigations were ordered when clinically indicated to establish the diagnosis.

The causes of optic atrophy were categorized as follows: Hypoxic ischemic encephalopathy (HIE), idiopathic, hydrocephalus, compressive, infective, traumatic, congenital (associated with ocular, brain or craniofacial anomalies), inflammatory, consecutive to vascular occlusion, and miscellaneous. Those patients found to have hydrocephalus on neuroimaging, irrespective of the underlying cause, were classified under the category of hydrocephalus as being the primary cause of optic atrophy.

Results

We identified 324 children (583 eyes) in whom the diagnosis of unilateral or bilateral optic atrophy had been made. Majority of patients (n = 259; 80%) had bilateral involvement. There were 205 (63%) boys and 119 (37%) girls with a mean age of 7 years (range: 0-16 years). Among these, 160 (49%) presented with defective vision at a mean age of 7 years, 71 (22%) with strabismus at a mean age of 4.7 years, and 18 (6%) with only nystagmus at a mean age of 2.4 years. 60 (18.5%) children presented with a combination of two or three of the above symptoms [Table 1]. Four (1%) children were incidentally found to have optic atrophy on routine eye checkup. Six (1.8%) children were referred from a neurologist. They were treating these children for brain tumors.

Of the 583 eyes, 119 (20%) had VA in the range of 6/6-6/18 [Table 2]. It was not possible to record the vision in 12 (2%) eyes. Two eyes had an eccentric fixation.

Etiology: It was possible to determine the etiology for 226 (70%) children [Table 3].

Hypoxic ischemic encephalopathy seen in 133 patients (41%) was the most frequent cause of childhood optic atrophy. Of these 133 patients, 20 (15%) had preterm delivery, 94 (71%) had history of delayed developmental milestones with one or more episodes of seizures, 7 (5%) had microcephaly, 2 (2%) had a history of intracranial hemorrhage, 1 (1%) underwent laser therapy for retinopathy of prematurity and 1 (1%) underwent bilateral cataract surgery. All 133 patients had radiologically proven (either CT or MRI) diagnosis of HIE.

We could not establish a cause in 98 children (30%) and they were classified as having idiopathic optic atrophy. Among these, 23 children had a history of either delayed developmental milestones or seizures. All (98 children) had normal studies of CT or MRI of the brain.

The third most frequently observed etiology was hydrocephalus (n = 24, 7.4%). 16 (67%) patients had congenital primary hydrocephalus unrelated to any other condition, 5 (21%) patients had optic atrophy secondary to tumors (1 case each of choroid plexus papilloma, pinealoblastoma, subdural hygroma, pilocytic astrocytoma and thalamic abscess respectively), and 3 (13%) secondary to tuberculous meningitis. Eleven underwent ventriculo-peritoneal shunting procedures.

Compressive causes accounted for 18 cases, (5.6%). Among the compressive causes [Table 4], craniopharyngioma (n = 8, 44%) was the most common brain tumor. Three children presented to us after undergoing brain surgery. Five children were diagnosed after undergoing neuroimaging for optic atrophy. Two children among them subsequently underwent surgery. Tumors like arachnoid cyst, porencephalic cyst

Table 1: Presenting features of childhood optic atrophy

Presenting feature	Number of patients (%)	Mean age at presentation (years)
Only defective vision	160 (49.39)	7.09
Only strabismus	71 (21.91)	4.71
Only nystagmus	18 (5.56)	2.38
Defective vision+strabismus	40 (12.34)	7.30
Defective vision+nystagmus	15 (4.63)	7.43
Strabismus+nystagmus	1 (0.31)	1
Defective vision+ strabismus+nystagmus	4 (1.23)	8
Routine check up	4 (1.23)	7
Referral from neurologist	6 (1.85)	8.16
Leucocoria	2 (0.62)	0.5
Headache	3 (0.92)	11

Total number of patients: 324

Table 2: Visual acuity at presentation

VA at presentation	Total number of eyes (%)
6/6-6/18	119 (20.41)
6/18-6/60	82 (14.06)
6/60-FC 1m	73 (12.52)
FC 1m–PL^+	70 (12.0)
PL^-	32 (5.48)
Fixing and following light	102 (17.50)
No fixation to light	91 (15.61)
Not cooperative	12 (2.06)
Eccentric fixation	2 (0.34)

Total number of eyes: 583. VA: Visual acuity

Table 3: Causes of childhood optic atrophy

Diagnosis	Number of patients (%)	Average age at presentation (years)
Hypoxic ischemic encephalopathy	133 (41.05)	4.51
Idiopathic	98 (30.21)	7.38
Hydrocephalus	24 (7.40)	5.73
Compressive	18 (5.56)	6.87
Infective	19 (5.86)	6.14
Trauma	16 (4.93)	9.54
Congenital (ocular, craniofacial, brain anomalies)	7 (2.16)	4.54
Inflammatory	5 (1.54)	11.4
Vascular occlusion	2 (0.62)	12.5
Miscellaneous	2 (0.62)	10
Acute lymphoblastic leukemia	1 (0.31)	11
Thalesemia major	1 (0.31)	9

Total number of patients n=324

and ethmoidal meningocele were diagnosed following neuroimaging to investigate the cause of optic atrophy. Children with occipital encephalocele, choroid plexus papilloma, optic nerve glioma, posterior fossa ependymoma and fibrous dysplasia presented to us after undergoing primary neuro-surgery.

Amongst the infective causes (19 patients, 6%) [Table 5] tuberculous meningitis (n = 8, 44%) was the most common cause of optic atrophy. All eight children completed the course of antituberculous therapy. The next common infective cause observed was viral meningoencephalitis (n = 7, 39%). Two children had TORCH infection most likely Rubella infection.

Head trauma was the cause of optic atrophy in 16 (5%) patients. Most involved a fall from the staircase with loss of consciousness, while four patients had motor vehicle accidents.

Seven (2%) patients had optic atrophy associated with ocular, craniofacial or brain (congenital) malformations [Table 6].

Inflammatory disease was the cause of optic atrophy in 5 (2%) children, four of whom had optic neuritis secondary to demyelinating lesion, one had optic atrophy secondary to typhoid fever (with central nervous system complications).

Of the vascular causes, two children had optic atrophy secondary to ophthalmic artery occlusion. Of the miscellaneous causes, one child was diagnosed to have Thalassemia major and another was diagnosed with acute lymphoblastic leukemia. Neuroimaging done of the child with thalassemia major had revealed diffuse cerebral atrophy.

Table 4: Compressive causes of childhood optic atrophy

Compressive causes	Number of patients (%)
Craniopharyngioma	8 (44.44)
Arachnoid cyst	3 (16.67)
Optic nerve glioma	1 (5.56)
Ethmoidal meningocele	1 (5.56)
Porencephalic cyst	1 (5.56)
Occipital encephalocele	1 (5.56)
Choroid plexus papilloma	1 (5.56)
Posterior fossa ependymoma	1 (5.56)
Fibrous dysplasia	1 (5.56)

Total number of patients n=18

Table 5: Infective causes of childhood optic atrophy

Infective causes	Number of patients (%)
Tuberculous meningitis	8 (44.44)
Viral meningoencephalitis	7 (38.89)
TORCH infection	2 (11.11)
Meningitis secondary to chickenpox	1 (5.56)

Total number of patients: 18

Table 6: Congenital causes of childhood optic atrophy

Malformation	Number of patients
De Morsier syndrome	2
Wolframs syndrome	1
Morning glory anomaly	1
Disc coloboma	1
Crouzon anomaly	1
Aniridia with cataractous lens	1

Total number of patients: 7

Discussion

Optic nerve atrophy is not a disease, but rather a sign alerting the ophthalmologist of a potentially more serious condition. A thorough investigation of a child presenting with optic atrophy is, therefore, mandatory, and knowledge of the prevailing causes aids the clinician in an appropriate work-up and subsequent intervention. This is especially important in a child where timely diagnosis and intervention of a progressive or life-threatening cause may limit the visual disability and systemic morbidity in the ensuing years, or else guide the family towards appropriate visual, educational and social rehabilitation.

To the best of our knowledge, this is the first report in the literature mentioning the causes of optic nerve atrophy in Indian children. The presenting features and common age of presentation of such children have also been described here. 160 children (49%) presented with defective vision, 71 (22%) presented with strabismus, while 18 (6%) presented with nystagmus. Children with strabismus, (mean age of presentation: 4.7 years) and nystagmus (mean age of presentation: 2.4 years) presented at a younger age when compared with those with defective vision alone, (mean age of presentation: 7 years). This highlights the fact that children with obvious ocular misalignment or motor disorders are more likely to seek medical care than those with defective vision alone. Furthermore, all children with strabismus and nystagmus should undergo a detailed ophthalmic evaluation, with a careful posterior segment assessment.

A recent study by Mudgil and Repka found complications related to premature birth to be the most common cause for optic atrophy observed between 1987 and 1997 below 10 years of age.[4] HIE was the most common cause leading to optic nerve atrophy in children in our study. In contrast, an earlier study by Repka and Miller described perinatal insults in only 9% of patients from 1978 to 1987.[3] The increased incidence has been attributed to increased survival rate of very high risk infants in the recent past who subsequently present with cortical visual impairment and optic atrophy. This is well in accordance with the other studies.[11-13] It has also been noted that prevailing social customs in the region of delayed feeding of newborn children may result in HIE changes as a result of hypoglycemia and delayed medical attention (unpublished observation). It is difficult, however, to clinically determine the exact mechanism of HIE in children presenting later in childhood due to lack of old records with most patients, lower educational qualification, or lack of medical attention being sought until presentation for poor vision or delayed developmental milestones. Majority of these children present with multisystem disabilities due to cortical injury, as seen in this study, where 71% presented with delayed developmental milestones and seizures. These children are also seen to

present at a younger age (mean age of presentation: 4.5 years) presumably due to the concomitant systemic diseases. In a case series of 100 patients by Chaddah *et al*., which included 16 patients ≤10 years and 38 patients under the age of 20, tubercular meningitis was the most common cause mentioned in children, although no specific data regarding etiology specific to childhood optic atrophy was given.[14]

In our study, an etiological association could not be established in 98 cases (30%) and this was the second most common category. In these cases, despite evaluation with neuroimaging, systemic investigations and consultation with neurophysicians, no cause for optic atrophy could be found [Table 2]. This is higher as compared to other published studies.[2-4] Repka *et al*. found no diagnosis in 11% of their cases, while Mudgil *et al*. found unknown cause in 4%. The patient profile in our clinical practice has a significant proportion of patients from lower socioeconomic background or patients from distant areas. This precludes the possibility of extensive investigation due to financial constraints as well as problems in follow-up of patients advised for investigations from different areas of residency. There was no group of hereditary optic neuropathy in our study. Genetic work-up is not usually possible or economically feasible for most patients, and the diagnosis is usually assumed in case of positive family history of optic nerve disease or screening of close relatives. However, for patients presenting to us from distant areas, screening is not usually practically possible, making such a diagnosis difficult to make. Hence, there may be a possibility that some cases included in this category may have hereditary optic atrophy.

A compressive etiology was found in 18 cases, (6%). We found craniopharyngioma (8 patients; 44% of children with tumors) to be the most common brain tumor leading to optic atrophy in children. This is in contrast to published literature that has described astrocytomas as being the commonest intracranial tumor in children.[14-16] Jain *et al*. reported a higher frequency of craniopharyngiomas (10%) in a multi-institutional study from India when compared to literature from the West of around 5%.[16] This may be attributable to the differences in the ethnical distribution of the study populations. Three of these children had already undergone surgery at an appropriate age and subsequently developed optic atrophy.

A study by Lee *et al*.[17] in 2005 described the diagnostic yield of neuroimaging and other laboratory investigations in the investigation of optic atrophy. Their sample included only patients with optic atrophy in the absence of any other associated history, signs or symptoms, previous investigations or systemic conditions that could explain the cause. They found compressive causes in 20% of the 91 patients included in their study, while 80% remained unexplained following neuroimaging. The authors felt that this warranted neuroimaging to rule out potentially serious associations in all patients of isolated optic atrophy. In our group of patients, taking into consideration only those patients for whom neuroimaging had been done ($n = 169$), idiopathic optic atrophy ($n = 98$) accounted for 58% of these cases. The "yield" from neuroimaging in our group was much higher when compared to the study by Lee *et al*. This data points toward a need for neuroimaging and other targeted investigations as almost half of the patients had positive findings for which intervention could alter the course of systemic or ocular disease. However, a direct comparison cannot be made as the methodology and study sample are not comparable, where this study aimed at primarily describing the clinical profile and etiology of optic nerve atrophy in children. Patients were investigated based on their overall clinical presentation. Furthermore, the institute being a referral center, the cases presenting here may not be representative of those in the general population to derive a conclusion regarding investigations in optic atrophy.

The authors acknowledge that there may be some overlap in the patients grouped into different etiological categories of optic atrophy. Some cases of tumors and infective etiology, (tubercular meningitis), have been grouped under hydrocephalus. These patients were so grouped according to the findings suggestive of hydrocephalus (or raised intracranial pressure) on neuroimaging that were absent in the case of those patients grouped under infective or compressive causes. There may, however, be similarities in the mechanism of optic atrophy between these groups.

Of the miscellaneous causes, the patient with thalassemia major presented was found to have diffuse cerebral atrophy and optic atrophy. Thalassemia is known to be a hypercoaguable state with cases of asymptomatic brain damage attributable to thrombotic events.[18,19] This may explain optic atrophy in this patient. There have also been studies establishing high dose desfuroxamine therapy as neurotoxic with both auditory and visual dysfunction, advocating regular ophthalmic checkup.[20] In addition, case reports have described optic atrophy occurring secondary to optic nerve compression occurring secondary to hematopoietic tissue and diploic expansion of craniofacial bones, as well as suprasellar extrameduallary hematopoiesis that acts as a space occupying lesion.[21,22] The patient with acute lymphoblastic leukemia had presented with a history of decreased vision for the past 6 years following chemotherapy. Other than direct leukemic infiltration of the optic nerve, toxicity from either chemotherapeutic agents or radiotherapy is a known mechanism in this condition.[23]

Other than the limitations already described, the prevalence of each cause of optic atrophy as determined in this study is subject to at least two biases. First, as previously mentioned, the patients in this study were seen in a tertiary referral setting and may not represent the true incidence in the community. Second, because we limited the review to patients <16 years of age, the incidence of optic atrophy associated with neurodegenerative and hereditary syndromes may be underestimated. In our experience, optic atrophy may develop late in the course of the patient's deterioration from such conditions.[24,25] Ophthalmologic consultation is not always obtained, thus reducing the observed proportion.

Careful follow-up is crucial for all patients with optic atrophy. Progressive visual loss is an indication for re-examination and repeat neuroimaging. As seen in this study, a number of patients primarily presented to the ophthalmologist and were found to have other serious systemic disease such as ICSOL. For patients with a prior diagnosis of tumors, the ophthalmologist also has an important role in detecting recurrence. HIE being the most

common cause in almost half of the patients in this series, a careful history taking with regards to the perinatal period and neuroimaging wherever suspected may be the only clues toward a possible diagnosis of cortical ischemic insult. The need for multidisciplinary care in rehabilitation of these children should also be kept in mind.

Conclusion

Varied etiologies for optic atrophy have been found in this study, the commonest being HIE. A relatively high proportion of patients for which no cause could be found may be explained by the socioeconomic constraints in our patient profile, precluding extensive investigation. Although optic atrophy is not a major cause of childhood blindness in the country, its potential association with life-threatening diseases makes it an important clinical entity for the ophthalmologist. Knowledge of the prevalent causes can aid in appropriate investigation and timely intervention for these children. It is also important to note that the diagnosis of optic atrophy carries with it a guarded prognosis for visual function of the child and may be associated with multiple disabilities. The treating ophthalmologist may be the first point of contact for these patients and must, therefore, be sensitized to the need for guiding the family towards appropriate rehabilitation to improve the functional potential of these children.

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