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Clinical profile and management of sixth nerve palsy in pediatric patients (0–15 years) in Southern India – A hospital-based study

Iva Rani Kalita, Veena K, Fredrick Mouttappa, Priya Sundaralakshmi[1], Harsh Vardhan Singh[2]

Purpose: This study was done to evaluate the clinical profile in pediatric patients (0–16 years) presenting with acute onset esotropia due to sixth nerve palsy and its management options in a tertiary care set up of Southern India. **Methods:** A total of 12 patients presenting to our OPD with acute onset esotropia due to sixth nerve palsies were included in this retrospective study. All patients were observed for 6 months and managed with prism and/or patching while waiting for spontaneous resolution and later managed surgically. Neuroimaging was done in all cases. **Results:** The mean deviation of esotropia at presentation was 30.17 ± 5.7 Prism Diopter (range 12–50 Prism Diopter 95% CI, SD 10.11). Mean age of the patients during presentation was 8.6 ± 2.4 years (range: 1–15 years, SD 4.27). Among the common causes of sixth nerve palsy in our study population were trauma and idiopathic intracranial hypertension followed by tumor and miscellaneous causes. Only three patients underwent surgical correction of residual deviation after a waiting period of 6 months for self-resolution. Spontaneous resolution was observed in 41.6% patients, and surgical correction (unilateral resection–recession) was done in 25% of the patients with good surgical outcome. **Conclusion:** At 1-year follow up, the motor outcome was satisfactory except for one patient who had diffuse pontine glioma and had worsening neurological symptoms on follow-up.

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Sixth nerve has the longest course of travel from dorsal pons to lateral rectus muscle and may present as "False localizing sign" due to its injury or compression anywhere along its pathway. The commonest cause has been found to be tumor in children unlike vascular etiology in adults.[1-3] CN6 palsy in patients with tumor can possibly be a part of the tumor presentation, resection, or progression as mentioned by Dotan *et al*.[2] The second common cause is trauma.[4-7] Other causes being elevated intracranial pressure, congenital, inflammation, idiopathic, and postviral.[8] Except for neoplasm, other causes of acquired abducens palsy mostly presents with esotropia only. Slowing of saccadic velocities of ipsilateral lateral rectus is one important feature that differentiates esotropia due to sixth nerve palsy from other types.[9] Patients are labelled to have Benign Abducens nerve palsies when no definite etiology of paresis is found and mostly follow a viral episode or postvaccination.[10,11] Congenital sixth nerve palsy is one important diagnosis that is underreported as it resolves within first 4–6 months of birth and usually misdiagnosed as infantile esotropia. It can be associated with raised intracranial tension due to trauma during birth and might resolve spontaneously.[12] Congenital esotropia and esotropic duanes retraction syndrome are two close differential diagnosis of CN6 palsy in a child. While globe retraction associated with DRS is difficult to elicit in infants, one can find a much larger angle esotropia in CN6 palsy than in DRS.[12] There is always a dilemma regarding neuroimaging in children with acute onset esotropia. Many suggest that neuroimaging to be done only in patients only with other neurologic signs, whereas some authors prefer in all cases.[3,7] Unlike studies for etiology of CN6 palsy in literature, very few studies have been found to mention long-term follow-up and management in these children. Most patients with benign palsy recovers completely, but if it fails, it must alarm clinician of more serious intracranial pathology. Resolution in traumatic cases occurs in 50% patients and 90% in inflammatory etiologies within weeks to months of onset.[13] In this present study, the authors study the incidence, etiology, and management outcomes of sixth nerve palsy among pediatric population in Southern India.

Methods

All patients (age 0–15 years) with acute onset acquired sixth nerve palsy diagnosed and managed between January 2019 till December 2019 in the Pediatric Ophthalmology Department of (…redacted for review purpose) were retrospectively reviewed. The authors adhered to the tenets of Declaration of Helsinki and the study was approved by both Institutional Ethics Committee and Review boards.

All patients underwent visual acuity testing, squint measurements, test for binocular single vision, and stereopsis for both near and distance, respectively. Snellen chart was used

Departments of Paediatric and Strabismus, [1]Neurophthalmology and Low Vision Services and [2]Vitreo-retina Services, Aravind Eye Hospital, Pondicherry, India

Correspondence to: Dr. Iva Rani Kalita, Department of Paediatric and Strabismus, Aravind Eye Hospital, Puducherry - 605 007, India. E-mail: kalitaiva3@gmail.com

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for visual aquity testing. Binocular response was evaluated with Worth-4 dot test at near (1/3 m) and distance (6 m). TNO test (near) and Randot Dot Stereoacuity test (distance) were used for obtaining stereopsis.

The angles of deviations were assessed by alternate cover test in nine cardinal gaze positions and were noted in Prism Dioptere (PD). Ocular motility patterns and nystagmus were evaluated clinically. Abduction deficit was rated from 0 (no limitation) to -3 (midline not reached). Neuroimaging was done for all cases to rule out underlying intracranial pathology.

The patients were observed for 6 months prior to surgical correction. Unilateral lateral rectus resection and medial rectus recession was done in nonresolving cases as per standard surgical dosing. Following surgery, a minimum follow-up of 6 months was considered to define it as success or failure of surgery. Success surgical outcome was defined as orthotropia or residual of <10 PD with no head turn or diplopia.

Neurosurgeon referral was given for the required cases. Incomitance was defined as limited abduction and larger deviation in lateral gaze toward the paralyzed muscle.

All children known to have infantile esotropia, hyperopia >2D (refractive accommodative ET), previous history of squint surgery, and nonparalytic and restrictive causes were excluded. Hospital-based incidence was calculated based on the total pediatric population attending the tertiary eye care in a year and number of new cases of pediatric CN6 palsy in the given period.

Results

A total of 32,891 (new cases 15,655) pediatric patients (0–15 years) were examined in Department of Paediatric Ophthalmology and Strabismus (….redacted for review purpose), from Jan 2019 to Dec 2019. Out of this, esotropia was diagnosed in 280 patients giving a prevalence of 0.85%. Acute onset esotropia due to CN6 palsy alone was found in 12 patients out of 280. So acute esotropia due to abducens palsy had an incidence of 0.77/1000 children in our hospital. The clinical characteristic of CN6 palsy patients is shown in Table 1. Mean duration of onset of symptoms was 19.75 days (range: 3 days–2 months, SD 16.78) prior to presentation. The age ranged from 1 to 15 years (mean: 8.6 ± 2.42, SD: 4.27). Nine patients were male and three were female. Mean primary deviation was 30.16 ± 5.72 PD, 95%CI (range: 12–50 PD, SD: 10.11). Mean lateral incomitancy of 11.37 ± 1.2 PD (SD 1.76) was found in all unilateral cases of CN6 palsy (8 out of 12 cases).

Patient no. 1, 11: Both patientshad pontine glioma. Patient 1 was known to have diffuse pontine glioma of size 3.6×.8 cm diagnosed 3 months prior to presentation and was on chemoradiation [Fig. 1]. Surgical excision of tumor was not possible for the consulting neurosurgeon because of the diffuse nature of the tumor. He also had difficulty in degglutition and speech and ataxia. Prism glass was advised, but he could not achieve restoration of motor fusion. On follow-up, the tumor size was worsening. The second patient presented with diplopia and showed bilateral papilledema. Prism glasses were advised and had no diplopia on follow-up visit.

Patient no. 2 presented with diplopia 1 week. She also complained of diminishing vision in BEs. She was diagnosed

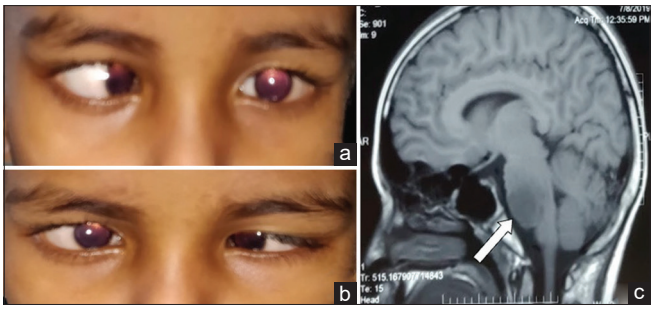

Figure 1: (a and b) Clinical image of patient no. 1 showing the right eye LR palsy with gross abduction deficit and (c) T1W Coronal MRI imaging showing diffuse pontine glioma (white arrow)

to have scrub typhus (scrub typhus rapid card test + ve for IgM antibodies, Inbios, USA). Magnetic resonance imaging (MRI) features were suggestive of central vein thrombosis with mild stenosis at junction of B/L transverse sinus and sigmoid sinus and prominent optic nerve sheath. Papilledema in BEs was noted. She was managed conservatively by neurologist with oral doxycycline and anticoagulants. On 1-month FUP, residual ET of 10 PD was found with minimal abduction restriction in BEs. Stereoacquity of 400 s of arc was regained.

Patient nos 3, 4, and 5 had trauma to head by fall prior to presentation. Patient 3 had normal neuroimaging, whereas neuroimaging in case nos 4 and 5 showed left temporal bone fracture and extradural haemorrhage, respectively. Prism glasses were prescribed for both cases 3 and 4, whereas patching of normal eye in case 5. Only patient 4 underwent surgery for residual deviation after 6 months, but intermittent 8 PD esodeviation was present postsurgery. Stereopsis was regained in two cases and was absent in case 4.

Patients 8, 9, and 10 had episodes of headache prior to presentation. Papilledema was present in both eyes of all three. Neuroimaging showed empty sella suggestive of pseudotumour cerebri and optic nerve sheath edema [Fig. 2]. Only one case (no. 10) underwent surgical correction. All three had good binocular vision and motor alignment on follow-up.

Patients 7 and 12: Both of these patients had viral illness prior to presentation and presented wih squinting of LE following fever. The symptoms occurred almost within 1 week of fever. Patching of good eye was advised for both cases. At 3-month follow-up both had good binocular vision and no deviation of eyes.

Patient 6: presented with squinting of right eye for 1 month with no significant history. On neuroimaging, atrophy of the right lateral rectus muscle was found. The patient was advised patching initially but was later corrected surgically with residual esotropia of 8 PD on final follow-up.

Discussion

The prevalence of childhood esotropia in USA has been found to be approximately 2.0% of all children younger than 6 years out of which paralytic cause has been found in 6.5%.[14] Whereas in adults, annual incidence of sixth nerve palsy was 11.3/100 000. In Asian population, the overall incidence has been reported to be 4.66 per 100,000 person-years.[15] Etiology of sixth nerve palsy can be congenital or acquired. Diplopia mainly on looking toward the gaze of paretic muscle is mainly

Table 1: Clinical characteristics, management, and long-term outcome of patients of sixth nerve palsy in pediatric age group (0-15 years) in the present study

Patient number	Sex	Age yrs	Duration of symptoms	Chief complains	Eye	Vision		Neurological examination	Deviation (PD)	Neuroimaging	Treatment	Outcome (at 1 year)
						RE	LE					
1	M	12	1 month	Diplopia, difficulty in speech, deglutition	RE	6/6	6/6	AD (- 4) RE	D - 35, N - 30 LI: 15 PD	Pontine glioma 3.6×2.8 cm	Prism glasses	Progressive tumor, angle worsened, neurosurgeon referral
2	F	15	1 week	Diplopia	BE	6/36	6/36	AD RE (-2) LE (-3), B/L papilledema	Alternate esotropia 40	Central vein thrombosis d/t scrub typhus	Neurosurgeon follow-up	No diplopia/residual 10 PD squint
3	M	10	1 month	Diplopia, h/o trauma	RE	6/6	6/6	AD RE (-1) face turn to right	Primary 20 Secondary 30 LI 12PD	WNL	Prism glasses followed by Surgery	No manifest deviation with prism
4	M	8	2 months	Diplopia, h/o RTA	LE	6/6	6/6	AD LE (-2) face turn to left	Primary 30 Secondary 40 LI: 10 PD	Left temporal bone#	Prism glasses followed by Surgery	Residual 8 PD esotropia
5	M	9	1 month	Squinting	RE	6/6	6/6	AD (-1)	50 PD LI 10 PD	Rt subgaleal hematoma with soft tissue selling in supraorbital and frontal	LE patching	resolved
6	M	8	1 month	Squinting	RE	6/6	6/6	AD (-1)	Primary deviation 35 Secondary 45 LI: 12 PD	Atrophy of LR muscle	Patching followed by surgery	Residual 8 PD
7	F	5	8 days	Squinting Face turn Following fever	LE	6/6	6/6	AD (-1/2)	P: 20 Secondary 25 LI: 10 PD	WNL	patching	Resolved fully
8	F	12	14 days	Squinting BEs	BEs	6/6	6/9	AD (-2) LE AD (-1) RE	Alternate esotropia 30 PD	Empty-sella papilledema	Alternate patching	Resolved
9	M	9	4 days	Diplopia,	RE	6/6	6/6	AD (-1)	25 PD LI: 10 PD	Empty sella papilledema	Prism	Resolved
10	M	8	2 weeks	Diplopia h eadache, vomitting	BES	6/9	6/6	AD (-1) Alternate face turn	35 PD	Empty sella papilledema	Alternate patching	Ortho
11	M	13	1 week	Diplopia	BEs	6/60-6/ 6-3.5DS	6/60-6/ 6-3.5DS	AD (- 2) AD (-1)	12 PD	B/L papilledema	Pontine glioma	Prism Same comfortable with prism
12	M	1	3 days	Squint Fever 1 week before	LE	6/6	6/6	AD (-2)	30 PD LI 12 PD	WNL	patch	Resolved in 2 months

M: Male, F: Female, RE: Right eye, LE: Left eye, BE: Both eyes, AD: abduction deficit, D: distant deviation, and N: Near deviation. LI: Lateral incomitancy, B/L: bilateral , PD: Prism diopter, WNL: Within Normal Limit, Sx: Surgery, and #Fracture

present in older children, whereas young patient suppresses faster. The various etiologies of sixth nerve palsy in pediatric population mentioned in literature has been shown in Table 2.

Tumor

These children present with other symptoms like ataxia, dysphagia, gait abnormality, and nystagmus.[2] We had two patients who had pontine glioma. One patient had nystagmus, ataxia, and dysphagia, and the tumor size was larger than the other patient who only had esotropia with no other symptoms. The common neoplasms associated with CN6 palsy mentioned in literature are medulloblastoma, glioma, and idomyosarcoma.[2-6,13] Surgical correction of these patients might not always give motor fusion.[18]

Trauma: Trauma prior to onset of paresis was present in 25% of our patients similar to the other studies.[4-7] Two-third of patients resolved completely by 6 month, while one patient had to undergo surgical correction. So it is good to wait for at least 6 months before attempting surgery as spontaneous resolution occurs in most.

Idiopathic intracranial hypertension (IIH)

The nerve is mainly injured within the Dorello canal. Raised intracranial tension can be associated with pseudotumor cerebri, hydrocephalus, shunt failure, central venous thrombosis, lyme disease, tumor, and meningitis. It can present as comitant esotropia in early phases but later show incomitancy and also involve multiple nerves.[19] It is important to know that shunt can cause CN6 palsy possibly due to change in pressure to volume ratio resulting in injury to abducens nerve.[5] Three cases of pseudotumour cerebri were reported in the present study.

South India especially Tamil Nadu is one of the endemic regions for Scrub-typhus with large number of cases per year.[20] We had one diagnosed case of scrub typhus. It can have myriad presentations including sixth nerve palsy in children.[21,22] Isolated involvements of CN6 is very rare in Rickettsia fever when compared to involvement of other cranial nerves.[23-25] It can cause microinfarction, aseptic meningitis, and vascular thrombosis. Immunoassay is the preferred diagnostic tool.

Viral

We had two patients who had fever and upper respiratory tract symptoms prior to onset of squint. Though there is a close association of sixth nerve palsy and viral illness, the exact pathophysiology unknown and it has been attributed to autoimmune-mediated demyelination or direct damage of the nerve or associated arteritis.[26] Over a period of 17-year follow-up of 12 children by Sturm *et al*.,[27] benign CN6 palsy showed spontaneous recovery within 3–6 months.

Idiopathic

Idiopathic cause has been found to range from 9 to 33%. In total, 8.3% of cases in our study had an idiopathic cause where neuroimaging showed hypoplastic transverse sinus on the left side. The patient did not have any other neurological deficit. Commonly, the left transverse is smaller than the right one and is hypoplastic or absent in 20–39% of the population.[28] So it was believed to be an incidental finding in our patient. Out of 12 cases of undetermined etiology in study by Robertson *et al*.,[13] five cases were suspected to have multiple sclerosis because of associated neurological deficit. Recovery was not good in these cases. Dotan *et al*.[2] have rightly stated that without proper neuroimaging the chances of missing-out small tumors and demyelinating lesions are often high.

It is widely accepted that the recommended surgical procedure for treating CN6 palsy is recession–resection of horizontal muscles in cases of paresis and muscle transposition in palsy.[16] However deleterious complications like anterior segment ischemia must be taken into account while planning muscle transposition.[29] In the present study, spontaneous resolution was observed in 41.6% patients and surgical correction (unilateral resection–recession) was done in 25% of the patients with good surgical outcome [Fig. 3]. Although recovery with Botulinum injection was comparable to spontaneous recovery in acute traumatic palsy, still it is important to decide on early intervention to restore fusion and hence prevent amblyopia.[16,17] No Botulinum injection was administered in the present study [Table 3].

Table 2: Various causes of CN6 palsy in pediatric population in different studies and comparison with present study

Etiologies	Merino *et al*.,[1] 2010 (1995-2008) <14 years	Holmes 2001[16] (1978-92) <18 years	Lee *et al*. 1999[3] (1993-97) <18 years	Repka *et al*. 1995[17] (1985-93) <7 years	Harley *et al*. 1999[4] (1968-97) <16 years	Afifi *et al*. 1992[5] (1966-88) <18 years	Kodsi and Younge 1992[6] 1966-88 <17 years	Robertson *et al*. 1970[13] (1952-1964)	Our study (Jan-Dec 2019) <16 years
Tumor	4	2	34	21	17	25	18	52	2
Trauma	2	3	9	12	21	37	37	26	3
Congenital	2	1	8	-	5	17	-	-	-
Idiopathic	3	4	4	3	4	14	13	12	1
Viral	2	2	5	4	8	13	5	23	1
Pseudotumor	-	-	11	15	3	6	2	15	3
Shunt malformation	1	-				8			
Encephalopathy	-	-				5			
Miscellaneous (hydrocephalus, SAH, CVT, postvaccine)	-	-	4	9	4	7	13	5	2
Total sample	15	12	75	64		132	88	133	12

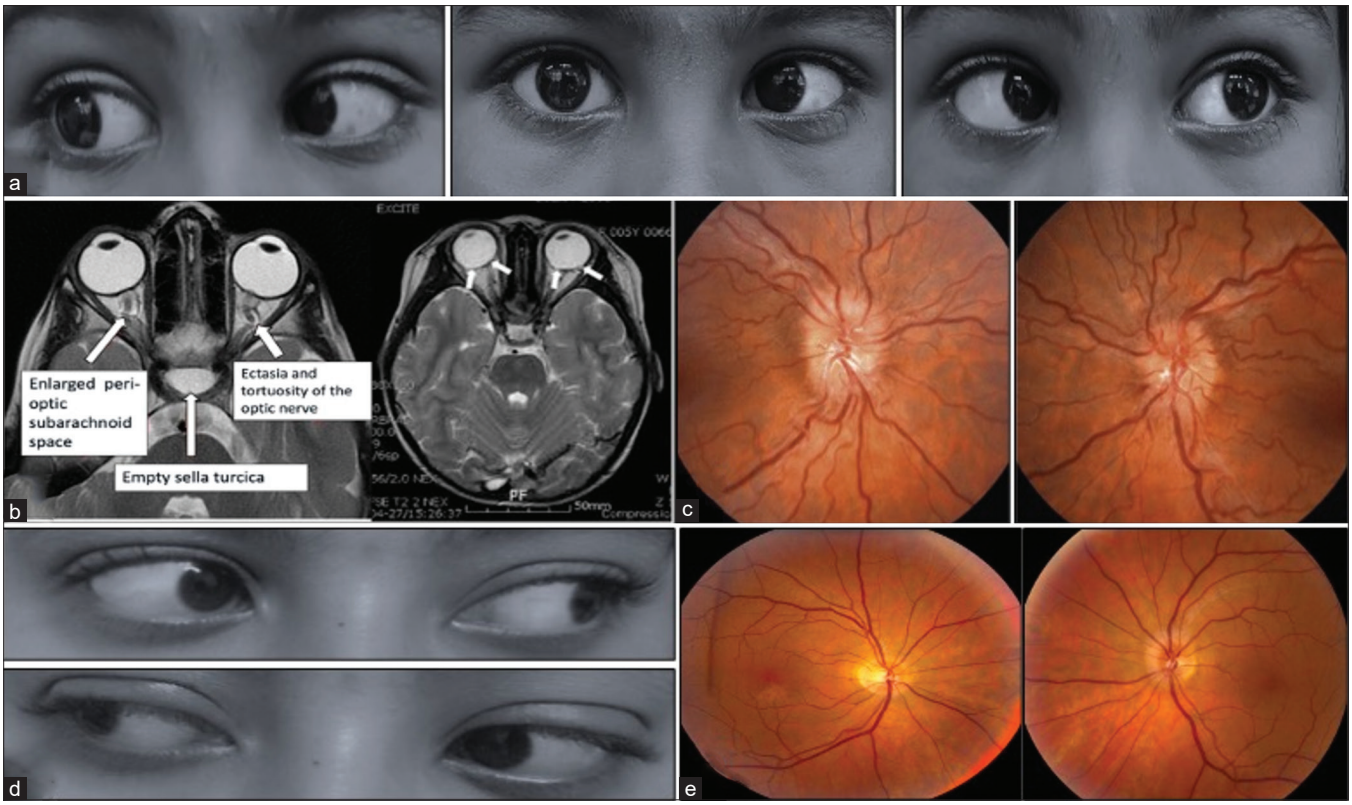

Figure 2: (a) Clinical image of patient no. 8 showing alternating esotropia and bilateral LR palsy, (b) T2W axial MRI imaging showing bilateral enlarged optic nerve sheath with empty sella turcica, (c) fundus image at presentation showing papilledema, and (d) clinical images (e) fundus picture on follow-up showing complete recovery

Figure 3: (a-c) Images showing postoperative outcome of patients who underwent a unilateral resection–recession procedure

Conclusion

To conclude, this study was done to determine the profile and management outcome in pediatric patients presenting with acute onset esotropia due to sixth nerve palsy. It is one of the few study done in Indian subcontinent. This study showed that trauma and IIH are the common causes contributing half of the total cases. Almost half of the patients resolved spontaneously within 3 months of onset. Neuroimaging should be done in all patients with alarming signs. Else children thought to have benign cause should be kept under close observation. Early surgical intervention gives good motor and sensory outcome, and unilateral recession–resection can be a preferable choice of surgery.

Table 3: Management modalities for sixth nerve palsy in various studies

Management	Merino *et al.* 2010[1]	Holmes *et al.* 2000[16]	Present study
Spontaneous	5	20	9
Surgical	3	19	3
Botulinum	7	10	nil
Total	15	56	12

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Conflicts of interest

There are no conflicts of interest.

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