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

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**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.

**Key words:** Abducens palsy, binocular vision, diplopia, neuroimaging, sixth nerve palsy, strabismus

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.⁷⁻⁹ CN6 palsy in patients with tumor can possibly be a part of the tumor presentation, resection, or progression as mentioned by Dotan et al.¹⁰ The second common cause is trauma.¹¹⁻¹² Other causes being elevated intracranial pressure, congenital, inflammation, idiopathic, and postviral.¹³ 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.¹⁴ 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.¹⁵⁻¹⁶ Congenital sixth nerve palsy is an 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.¹⁷ 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.¹⁸⁻¹⁹ 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.²⁰⁻²¹ 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.²² 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 for visual acuity testing.

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for visual acuity 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 patients had 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 deglutition 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 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. Stereoacuity 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 hemmorhage, 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 with 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
| Patient number | Sex | Age yrs | Duration of symptoms | Chief complains                      | Eye | Vision | Neurological examination | Deviation (PD) | Neuroimaging          | Treatment                             | Outcome (at 1 year)                     |
|----------------|-----|---------|----------------------|--------------------------------------|-----|--------|--------------------------|----------------|----------------------|----------------------------------------|----------------------------------------|
| 1              | M   | 12      | 1 month              | Diplopia, difficulty in speech, deglutition | RE  | 6/6    | AD (-4) RE               | D - 35, N - 30 | Pontine glioma 3.6x2.8 cm | Prism glasses                           | Progressive tumor, angle worsened, neurosurgeon referral |
| 2              | F   | 15      | 1 week               | Diplopia                             | BE  | 6/36   | AD RE (-2) LE (-3), B/L papilledema | Alternate esotropia | 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    | AD RE (-1) face turn to right | Primary 20 Secondary 30 LI 12PD | WNL                  | Prism glasses followed by surgery | Residual 8 PD esotropia                   |
| 4              | M   | 8       | 2 months             | Diplopia, h/o RTA                   | LE  | 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    | 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    | 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                   | LE  | 6/6    | AD (-1/2)                | P: 20 Secondary 25 Li: 10 PD | WNL                  | Patching followed by surgery | Resolved fully                          |
| 8              | F   | 12      | 14 days              | Squinting BEs                        | BEs | 6/6    | 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    | AD (-1)                  | 25 PD Li: 10 PD | Empty sella papilledema | Prism | Resolved                  |
| 10             | M   | 8       | 2 weeks              | Diplopia h eadache, vomiting         | BES  | 6/9    | AD (-1) Alternate face turn | 35 PD | Empty sella papilledema | Alternate patching | Ortho                  |
| 11             | M   | 13      | 1 week               | Diplopia                             | BEs | 6/6-6/ 6-3.5DS | AD (-2) AD (-1) | Empty sella papilledema | Pontine glioma patch | Prism Same | Resolved in 2 months |
| 12             | M   | 1       | 3 days               | Squinting Fever 1 week before        | LE  | 6/6    | AD (-2)                  | 30 PD Li 12 PD | WNL                  | Same | Resolved                  |

**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.[21] 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.[24]

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 months, 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.[28] We had one diagnosed case of scrub typhus. It can have myriad presentations including sixth nerve palsy in children.[3,22] Isolated involvement of CN6 is very rare in Ricketsia fever when compared to involvement of other cranial nerves.[23] 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.[5] 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].

| Etiologies              | Merino et al. (1995-2008) | Holmes (1978-92) | Lee et al. (1999) | Repka et al. (1985-93) | Harley et al. (1986-97) | Affifi et al. (1966-88) | Kodisi and Younge (1992) | Robertson et al. (1979) | Our study (Jan-Dec 2019) |
|------------------------|---------------------------|-------------------|------------------|------------------------|-------------------------|-----------------------|------------------------|-----------------------|------------------------|
| 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                         | -                 | -                | -                      | -                       | -                     | -                      | -                     | -                      |
| Encephalopathy         | -                         | -                 | -                | -                      | -                       | -                     | -                      | -                     | -                      |
| Miscellaneous          | -                         | -                 | 4                | 9                      | 4                       | 7                     | 13                     | 5                     | 2                      |
| (hydrocephalus, SAH, CVT, postvaccine) | 15 | 12 | 75 | 64 | 132 | 88 | 133 | 12 |
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 studies done in the 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            |

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

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Conflicts of interest
There are no conflicts of interest.

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