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Clinical Profile and Management of Sixth Nerve Palsy in Paediatric Patients (0-15 Years) in Southern India- a Hospital-Based Study

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Abstract

Purpose: This study was done to evaluate the clinical profile in Paediatric patients (0-16 years) presenting with acute onset esotropia due to Sixth Nerve palsy. 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 Dioptre(range 12-50 PD, 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 Tumour and miscellaneous causes. Only 3 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 (table 1) and surgical correction (unilateral resection-recession) was done in 25% of the patients with good surgical outcome. Conclusion: At one-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.

Introduction:

Sixth nerve which innervates ipsilateral lateral rectus muscle is common to get paralysed in children. It has the longest course of travel from dorsal pons to lateral rectus muscle and may present as “False localising sign” due to its injury or compression anywhere along its pathway. The commonest cause has been found to be Tumour in Children unlike vascular aetiology in adults [1]. CN6 palsy in patients with tumour can possibly be a part of the tumour presentation, tumour resection or tumour progression as mentioned by Dotan et. al. The second common cause is trauma [2, 3]. Other causes being elevated Intra-cranial pressure (ICP), congenital, inflammation, idiopathic, post-viral. 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 which differentiates esotropia due to sixth nerve palsy from other types. Trauma causes shearing forces on the nerve as it crosses the petrous part of temporal bone. Patients are labelled to have Benign Abducens nerve palsies when no definite aetiology of paresis is found and mostly follow a viral episode or post-vaccination.

Congenital sixth nerve palsy, is one important diagnosis that is underreported as it resolves fast within first 4-6months of birth and it is most of the time misdiagnosed as infantile esotropia. It can be associated with raised intracranial tension due to trauma during birth and might resolve spontaneously. 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.

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.

Unlike studies for aetiology of CN6 palsy in literature, very few studies have been found to mention long term follow-up and management in these children. Majority of patients with benign palsy recovers completely and if it fails to happens so, it must alarm clinician of more serious intracranial pathology. Resolution in traumatic cases occurs in 50% patients and 90% in inflammatory aetiologies within a weeks to months of onset. In this present study the authors have tried to study the incidence, aetiology, management outcomes of Sixth nerve palsy among paediatric population in Southern India.

Methology

All patients (Age 0-15 years) with acute onset acquired sixth nerve palsy diagnosed and managed between January 2019 till December 2019 in the Paediatric Ophthalmology Department of (redacted for review) 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(BSV) and Stereopsis for Both Near and Distance respectively. Snellen chart was used for Visual Aquity testing. Binocular response was evaluated with Worth-4 dot test at near (1/3 m) and distance (6m). 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 9 cardinal gaze positions and were noted in Prism Dioptre (PD). Ocular motility patterns and Nystagmus were evaluated clinically. Abduction deficit (AD) was rated from 0 (no limitation) to -3 (midline not reached). Neuroimaging was done for all cases to rule out underlying Intracranial pathology [4-7].

The patients were observed for 6 months prior to surgical correction. Unilateral Lateral Rectus recession and Medial rectus resection was done in non-resolving cases as per standard surgical dosing. Following surgery, a minimum follow-up of 6months was considered to define it as success or failure of surgery. Success surgical outcome was defined as orthotropia or residual of < 10PD 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 towards the paralysed muscle.

All children known to have Infantile Esotropia, Hyperopia 2D (Refractive Accommodative ET), previous history of squint surgery, non- paralytic and restrictive causes were excluded. Hospital-based incidence was calculated based on the total paediatric population attending the tertiary eye care in a year and number of new cases of paediatric CN6 palsy in the given period [8-11].

Results

A total of 32891 (New cases 15655) paediatric patients (0-15 years) were examined in Department of Paediatric Ophthalmology and Strabismus (at.....Redacted for review) from Jan 2019-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 1-15years (mean 8.6±2.42, SD-4.27). 9 patients were male and 3 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 patient had pontine glioma. Patient 1 was known to have Diffuse Pontine Glioma of size 3.6*2.8 cm diagnosed 3 months prior to presentation and was on Chemoradiation. Surgical excision of tumour was not possible for the consulting Neurosurgeon because of the diffuse nature of the tumour. 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 tumour 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. (Table 1)

Table1: Clinical characteristics, management and long term outcome of patients of Sixth Nerve palsy in paediatric age group (0-15years) in present study.

Patient number	Sex	Ageyrs	Duration of symptoms	Chief complains	Eye	Vision	Neurological examination	Deviation	Neuroimaging	Treatment	Outcome(at 1year)	
						RE LE			(PD)			
	M	12	1mnth	Diplopia, difficulty in speech, deglutition	LE	6-Jun	6-Jun	AD-1	D-35, N-30	Pontine glioma 3.6*2.8cm	Prism glasses	Progressive tumour, angle worsened, Neurosurgeon referral
								LE	LI: 15 PD			
	F	15	1week	Diplopia,	BE	Jun-36	Jun-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 10PD squint
	M	10	1month	Diplopia, h/o trauma	RE	6-Jun	6-Jun	AD RE(-1)	Primary-20	Wnl	Prism glasses	No manifest deviatio

												n with Prism
									Secondary-30			
									LI 12PD			
	M	8	2months	Diplopia, h/o RTA	LE	6-Jun	6-Jun	AD LE (-2)face turn to Left ,	Primary-30 Secondary-40	Left Temporal bone #	Prism glasses followed by Surgery	Residual 8 PD Esotropia
									LI : 10PD			
	M	9	1m	Squinting	RE	6-Jun	6-Jun	AD -1	50PD	Rt subgaleal hematoma with soft tissue selling in supraorbital and frontal	LE patching	resolved
									LI 10PD			
	M	2	1 months	Squinting	LE	6-Jun	6-Jun	AD -1	Primary deviation 35	Atrophy of LR muscle	Patching	Residual 8PD
									Secondary 45		Followed by surgery	
									LI: 12PD			
	F	5	8day	squinting	LE	6-Jun	6-Jun	AD -1/2	P: 20	WNL	patching	Resolved fully
				Face turn					Secondary 25			
				Following fever					LI: 10 PD			
	F	12	14d	Squinting BEs	BEs	6-Jun	9-Jun	AD -2 ,	Alternate Esotropia 30PD	Empty-sella	Alternate patching	Resolved
										papilledema		
	M	9	4d	Diplopia,	RE	6-Jun	6-Jun	AD -1	25 PD	Empty sella	Prism	resolved
									LI: 10PD	papilledema		
	M	8	2w	Diplopia ,headache ,vomitting	BES	9-Jun	6-Jun	AD -1	35PD	Empty sella	Alternate patching	ortho
								Alternate face turn		papilledema	And Surgery	
	M	13	1w	diplopia	BEs	6/60-6/6 -3.5DS	6/60-6/6 -3.5DS	AD -2	12PD	B/L papilledema	Pontine glioma	Prism

								AD -1				Same comfortable with prism
	M	1	3d	Squint	LE			AD-2	30pd	WNL	patch	Resolved in 2m
				Fever 1 week before		6-Jun	6-Jun		LI 12PD			

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). 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 anti-coagulants. On 1 month FUP, residual ET of 10PD was found with minimal Abduction Restriction in BEs. Stereoacquity of 400 sec of arc was regained. (Table 1)

Patient no 3,4,5 had trauma to head by fall prior to presentation. Patient 3 had normal neuroimaging whereas neuroimaging in case no 4 and 5 showed left temporal bone fracture and extradural haemorrhage respectively. Prism glasses were prescribed for both case 3 and 4 whereas patching of normal eye in case.

5. Only patient 4 underwent surgery for residual deviation after 6 months but Intermittent 8PD Esodeviation was present post surgery. Stereopsis was regained in two cases and was absent in case 4. (Table 1)

Patient 8, 9, 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. Ony one case (no. 10) underwent surgical correction. All three had good binocular vision and motor alignment on follow-up. (Table1)

Patient 7, 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. (Table 1)

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

Discussion and Conclusion

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 adult's annual incidence of sixth nerve palsy was 11.3/100 000 (95% CI, 9.3-13.2). In Asian population the overall incidence has been reported to be 4.66 (95% CI, 4.26–5.08) per 100,000 person-years.15 We calculated a hospital-based incidence of sixth nerve palsy in children(0-15 years). Aetiology of sixth nerve palsy can be congenital or acquired. Diplopia mainly on looking towards the gaze of paretic muscle is mainly present in older children whereas young patient suppresses faster. The various etiologies of sixth nerve palsy in paediatric population mentioned in literature has been shown in Table 2.

Table2: Various causes of CN6 palsy in paediatric population in different studies & comparison with present study.

ETIOLOGIES	MERINO ET AL1, 2010	HOLMES 200127	LEE ET.AL	REPKA ET.AL	HARLEY ET AL	AFIFI ET.AL	KODSI AND YOUNGE 19926	ROBERTSON ET AL	OUR STUDY
	(1995-2008)	(1978-92)	19993	199529	19994	19925	1966-88	1970 13	(Jan-dec 2019)
	<14 years	<18 years	(1993-97)	(1985-93)	(1968-97)	(1966-88)	<17 years	(1952-1964)	<16 years
			<18 years	<7years	<16years	<18 years			
Tumour	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

Pseudotumour	-	-	11	15	3	6	2	15	3
Shunt malformation	1	-				8			
encephalopathy	-	-				5			
Miscellaneous	-	-	4	9	4	7	13	5	2
(hydrocephalus,									
SAH, CVT, Post vaccine)									
Total sample	15	12	75	64		132	88	133	12

Tumour: These children present with other symptoms like ataxia, dysphagia, gait abnormality, nystagmus. We had two patients who had pontine glioma. One patient had nystagmus, ataxia & dysphagia, and the tumour 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 has been shown in Table 3.

Table3: Various types of tumour associated with CN6 palsy in pediatric population

TUMOUR TYPE	LEE ET AL 19993	DOTAN ET AL 20122	KODSI ET AL	AFIFI ET AL	ROBERTSON ET AL13	OUR STUDY 2019
			19926	19925		
Glioma	10	3	12	6	2	2
Menigioma			2			
Astrocytoma	1		3	7	8	
Medulloblastoma	13			1	6	
Granuloma		2				
Rhabdomyosarcoma	4			1		
Ependymoma	1		2	3	4	
Metastasis	1		4	1		
Craniopharyngioma			2	1		
Miscellaneous	1		8	5		
TOTAL	34	5	33	25	20	2

Surgical correction o f t hese patients might no t always give motor fusion [12].

Trauma: Trauma prior to onset of paresis was present in 25% of our patients similar to the other studies4-7. One patient had normal neuroimaging whereas the other two patients had ipsilateral Temporal bone fracture & sub-galeal hematoma respectively. Two patients resolved completely at 6-month follow-up while one patient had to undergo surgical correction later with a residual ET of 8PD. BSV and Stereopsis was restored in two cases. It's good to wait for at least sixth months before attempting surgery as spontaneous resolution occurs in most.

Table4: Management modalities for 6th nerve palsy in various studies

MANAGEMENT	MERINO ET AL	HOLMES ET.AL 2000 27	PRESENT STUDY
	2010 1		
Spontaneous	5	20	9

Surgical	3	19	3
Botulinum	7	10	nil
	15	56	12

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, tumour, & meningitis. It can present as comitant esotropia in early phases but later show incomitancy and also involve multiple nerves[16]. It's important to know that shunt can cause CN6 palsy possibly due to change in pressure to volume ratio resulting in injury to Abducens nerve [13,14]. We had 3 cases of pseudotumour cerebri who had Bilateral Papilledema and Empty sella on Neuroimaging. Two cases resolved fully without the need for surgery & one case needed surgical alignment with no residual squint.

South India especially Tamil Nadu is one of the endemic regions for Scrub-typhus with large number of cases per year [18]. It can have myriad presentations including 6th nerve palsy in children [15,17,23]. Isolated involvements of CN6 is very rare in Rickettsia fever when compared to involvement of other cranial nerves [19,21,22,24,25]. It can cause micro infarction, aseptic meningitis, vascular thrombosis. Immunoassay is the preferred diagnostic tool. We had one case of scrub typhus (table 1).

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 6th nerve palsy & viral illness, the exact pathophysiology unknown and it has been attributed to autoimmune mediated demyelination or direct damage of the nerve or associated arterities [5]. Over a period of 17 year follow up of 12 children by Sturm et.al, benign CN6 palsy showed spontaneous recovery within 3.6-6 months.

Idiopathic: CN6 palsy due to Idiopathic cause has been found to range from 9- 33%. 8.3% of cases in our study had an idiopathic cause where neuroimaging showed hypoplastic transverse sinus on 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 [26]. So it was believed to be an incidettal finding in our patient. Out of 12 cases of undetermined aetiology in study by Robertson et. Al 13, 5 cases were suspected to have multiple sclerosis because of associated neurological deficit. Recovery was not good in these cases. Dotan et. al2 has rightly stated that without proper neuroimaging the chances of missing-out small tumours & 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. However deleterious complications like anterior segment ischemia must be taken into account while planning muscle transpostion [27]. In the present study, spontaneous resolution was observed in 41.6% patients (table 1) and surgical correction (unilateral resection-recession) was done in 25% of the patients with good surgical outcome. Although recovery with Botulinum injection was comparable to spontaneous recovery in acute traumatic palsy, still it's important to decide on early intervention to restore fusion and hence prevent amblyopia.

To conclude this study was done to determine the profile and management outcome in paediatric 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 3months 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 give good motor and sensory outcome and unilateral recession-resection can be a preferable choice of surgery.

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