Original Article

Pediatric ocular motor cranial nerve palsy: Demographics and etiological profile

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Purpose: The aim of this study was to describe epidemiological and clinical characteristics of pediatric ocular motor cranial nerve palsy. **Methods:** This was a retrospective record-based study, carried out at a tertiary eye care hospital in India, between January 2011 and January 2015 and included patients up to 16 years of age at the time of presentation, diagnosed with third, fourth, sixth nerve palsy or a combination of these with other cranial nerve palsy. Data analyzed included demographic details, etiologies, presence or absence of amblyopia, relevant investigations, and management. **Results:** A total of 90 cases were included in the study. Eighty patients (88.88%) presented with isolated nerve palsy. Forty-three patients (47.77%) had congenital nerve palsy. The most common nerve involved was third (n = 35, 38.88%) followed by sixth (n = 23, 25.55%) and fourth nerve (n = 22, 24.44%). Most common cause of third and fourth cranial nerve palsy was congenital (n = 18, 51.42% and n = 17, 77.30%, respectively), while it was trauma for the sixth nerve (n = 7, 30.40%). Amblyopia was most frequently associated with third cranial nerve palsy (n = 27, 77.14%). The radio-imaging yield was maximum (n = 7, 70%) for combined cranial nerve palsy. Overall 44 (48.88%) patients were managed conservatively, while 46 (51.11%) patients needed squint with or without ptosis surgery. **Conclusion:** The most common ocular motor cranial nerve involved in the pediatric population was the third cranial nerve, and it was found to be the most amblyogenic in this age group. The neuroimaging yield was maximum for combined cranial nerve palsy. The most common conservatively managed nerve palsy in this study group was the fourth nerve palsy.

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Extra-ocular motor cranial nerve palsies are infrequent in the pediatric population with an incidence rate of approximately 7.6 per 10, 00 000.[1] Paralytic strabismus in children differs from adults in terms of presentation, causes, impact on growing visual system, and repair characteristics. Appropriate diagnosis and management in children are crucial and challenging. Unlike adults, treatment should be initially directed towards the management of amblyopia followed by restoration of fusion and stereopsis. To the best of authors' knowledge, studies describing clinical characteristics of extra-ocular motor cranial nerve palsies in the Indian subcontinent are mostly limited to the adult population.[2] The present study describes the clinical characteristics of extra-ocular motor cranial nerve palsies in the pediatric population at a tertiary eye care center of India.

Methods

This was a retrospective record-based study, and case files of patients seen between January 2011 to January 2015 were reviewed. All patients up to the age of 16 years, diagnosed with isolated or combined paralysis of third, fourth, sixth nerves, or a combination of these with other cranial nerves, were included**.** Institutional review board approval was obtained, and the study adhered to the tenets of Declaration of Helsinki. Patients were diagnosed based on the history and clinical examination. Cranial nerve palsies were either congenital or acquired. As defined in previous studies congenital cranial nerve palsy was defined when either ptosis, strabismus or abnormal head posture (AHP) was noted in early infancy.[1] The causes of acquired cranial nerve palsy were further investigated and analyzed, and it was considered idiopathic when squint, ptosis or AHP was of recent onset, was not evident in earlier photographs, and no cause was found on further investigation**.** Patients with congenital cranial disinnervation disorder, myasthenia gravis, and dysthyroid ophthalmopathy were excluded.

Comprehensive ocular examination was done by pediatric and neuro-ophthalmologists, and all relevant findings were noted. Neuroimaging was advised to all cases except those with clinical diagnosis of congenital fourth nerve palsy. Computed tomography scan (CT scan) brain and orbits with contrast was advised in children with history of trauma and those uncooperative for magnetic resonance imaging (MRI) even after sedation. MRI of the brain and orbits with contrast

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was done for rest of the cases. Amblyopia management was advised in all patients in the amblyogenic age group. An urgent neurointervention referral was sought whenever required. Patients with new-onset palsy (<6 months) were treated depending upon the cause. Squint and ptosis surgery was advised by treating ophthalmologist whenever deemed essential. Neurologist clearance was obtained in cases with intracranial pathology. Squint surgery was performed only after adequate amblyopia therapy and ensuring stable angles for a minimum duration of 6 months. Data analyzed included demographic details, etiologies, presence or absence of amblyopia, relevant investigations, and management. Statistical analysis was done using SPSS version 17.

Results

One hundred two eyes of 90 cases were included in the study. There were 63 (70%) males and 27 (30%) females. The mean age at presentation was 7.02 ± 4.30 years (range 1 month–16 years). Eighty patients (88.88%) presented with features of isolated nerve palsy, whereas 10 (11.11%) patients had features of combined cranial nerve palsy. The most common isolated cranial nerve palsy in the present cohort was third (n = 35, 38.88%) followed by sixth (n = 23, 25.55%) and fourth (n = 22, 24.44%). Most common presenting symptom was deviation of eyeball (n = 54, 60%) followed by AHP (n = 45, 50%), drooping of eyelids (n = 22, 24.44%), and diplopia (n = 8, 8.88%). Forty-three (47.77%) patients were of congenital origin, whereas 47 (52.22%) had acquired cranial nerve palsy. The mean visual acuity of the involved eye at presentation was 0.49 ± 0.49 Log Mar units, while it was 0.20 ± 0.29 Log Mar units for the uninvolved eye (P < 0.05). Overall, 44 (48.88%) patients were managed conservatively, while 46 (51.11%) patients needed surgical correction in the form of squint and/or ptosis surgery [Table 1]. The mean follow up period was 2.29 ± 2.72 years.

Isolated third cranial nerve palsy

The third cranial nerve was the most common isolated nerve to be involved (n = 35, 38.88%). All patients had unilateral palsy. Most common presenting feature was ptosis (n = 20, 57.14%) followed by squint (n = 14, 40%). Anisocoria was present in 7 (20%) eyes. The vision was most severely affected in this group of patients as compared to other groups and the mean visual acuity was 0.64 Log Mar units in the affected eye. Twenty-seven (77.14%) patients had amblyopia. Most common etiology was congenital (n = 18, 51.42%) followed by trauma (11, 28.60%). Overall, neuroimaging yield was 31.57% in this group. Most common findings were soft tissue lesion at the orbital apex (n = 3, 15.78%) followed by intracranial hemorrhage (n = 2, 10.52%)(epidural hemorrhage over left parietal lobe and fronto-parietal lobe) and open lip schizencephaly (n = 1, 5.26%). Orbital apex lesion constituted Tolosa Hunt Syndrome, calcified granuloma, and post traumatic orbital hematoma, respectively [Fig. 1a-c]. Sixteen (45.70%) cases were managed conservatively, while 19 (54.30%) cases required surgical correction in the form of either squint surgery (14, 78.94%) or both squint and ptosis surgery (5, 26.31%) [Table 2].

Isolated fourth cranial nerve palsy

Twenty-two (24.44%) patients had isolated fourth nerve palsy out of which 18 (81.81%) were unilateral and 4 (18.18%) were bilateral. The most common presenting feature was AHP (n = 19, 86.36%). Out of 18 unilateral cases, 16 patients had head tilt towards opposite side and 2 patients had head turn towards opposite side. Among bilateral cases three did not exhibit any head posture and one patient had head tilt towards right side. The most common cause for isolated fourth cranial nerve palsy was congenital (n = 17, 77.30%). Five cases (22.5%) were acquired and all of these were idiopathic without any identifiable etiology. Mean visual acuity was 0.30 ± 0.27 Log Mar units. Amblyopia was noted in 7 (31.81%) patients. Conservative management was advised in 13 (59.10%) patients, while 9 (40.90%) cases underwent squint correction [Table 2].

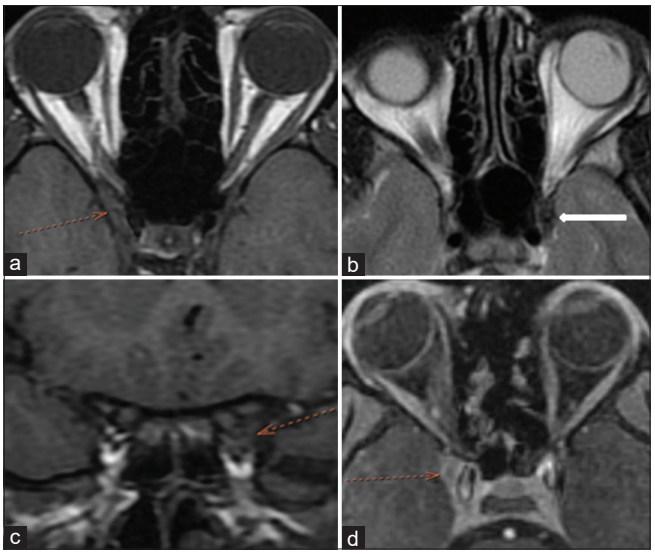

Figure 1: (a) MRI T1 sequence showing an ill-defined soft lesion at the right orbital apex (red arrow) suggestive of Tolosa Hunt Syndrome (b) MRI T2 sequence showing an ill-defined hypointense calcified lesion suggestive of calcified granuloma near the left orbital apex and extending up to cavernous sinus (white arrow) (c) MRI T1 sequence showing collection at left orbital apex following trauma suggestive of orbital apex hematoma (red arrow) (d) MRI T1 sequence showing an ill-defined homogenous contrast enhancing lesion at the right orbital apex and cavernous sinus region suggestive of meningioma (red arrow)

Isolated sixth cranial nerve palsy

Twenty-three (25.55%) patients had isolated sixth cranial nerve palsy out of which 21 were unilateral and 2 were bilateral cases. Most common presenting feature was squint (n = 18, 78.26%) followed by face turn (n = 14, 60.86%) and diplopia (n = 6, 26.08%). The etiology included trauma (n = 7, 30.4%) followed by congenital (n = 5, 21.7%), idiopathic (n = 4, 17.4%), viral infection (n = 3, 13%), presumed raised intracranial tension (n = 3, 13%), and neurosurgical complication (n = 1, 4.5%). Patients with presumed raised ICT had fever with papilledema. All three patients with papilledema refused imaging and were immediately referred to a neurologist. Overall, neuroimaging yield was 33.33%. Most common neuroimaging finding was skull base fracture (n = 2, 16%), temporo-parietal intraparenchymal hemorrhage (n = 1, 8.33%), and thickened sixth nerve (n = 1, 7.69%). Nonspecific incidental hypoxic ischemic encephalopathy changes were also noted (n = 3, 25%). Conservative management was advised to 12 (52.17%) patients and 11 (47.82%) needed squint correction. All patients with suspected or diagnosed intracranial pathology were referred to a neurologist for further opinion and management [Table 2].

Table 1: Demographic details of the study population

Parameters	Number and percentage
Sample size	90,102 eyes
Age in years	7.02±4.30 years (1 month-16 years)
Sex	Males: 63 (70%), Females: 27 (30.00%), Male:Female=2.33:1
Eye involved	Right: 38, 42.22%
	Left: 40, 44.44%
	Bilateral: 12, 13.33%
Mean visual acuity of the involved eye (In Log Mar)	0.49±0.49
Mean visual acuity of uninvolved eye (In Log Mar)	0.20±0.29
Presenting clinical feature (In decreasing order of frequency)	1. Deviation of eye ball (*n*=54, 60.00%)
	2. Abnormal head posture (*n*=45, 50.0%)
	3. Drooping of eye lid (*n*=22, 24.44%)
	4. Diplopia (*n*=8, 8.88%)
Motor nerve palsy (isolated)	Third: 35, 38.88%
	Fourth: 22, 24.44%
	Sixth: 23, 25.55%
Type of motor nerve palsy	Isolated: 80, 88.88%
	Combined: 10, 11.11%
Cause of motor nerve palsy	Congenital: 43, 47.77%
	Acquired: 47, 52.22%
Imaging findings (*n*=51)	Normal: 24 (47.05%)
	Abnormal findings: 16 (31.37%), Coincidental nonsignficant findings: 11 (21.56%)
Management	Conservative: 44 (48.88%)
	Surgical: 46 (51.11%)
Follow-up period	2.29±2.72 years

Table 2: Clinical and management details of different cranial nerve palsies

Parameters	Third cranial nerve palsy (isolated)	Fourth cranial nerve palsy (isolated)	Sixth cranial nerve palsy isolated)	Combined cranial nerve palsy
Eye involved	35 eyes of 35 patients	26 eyes of 22 patients	25 eyes of 23 patients	16 eyes of 10 patients
Mean visual acuity (involved vs uninvolved eye)	0.64±0.62 vs 0.18±0.34	0.30±0.27 vs 0.26±0.29	0.47±0.55 vs 0.15±0.13	0.55±0.42 vs 0.15±0.17
Squint	14, 40%	13,59.09%	18, 78.26%	9, 90%
Abnormal head posture	9, 25.71%	19, 86.36%	14, 60.86%	3, 30%
Ptosis	20, 57.14%	0	0	2, 20%
Anisocoria	7, 20%	0	0	4, 40%
Diplopia	2, 5.71%	0	6, 26.08%	0
Amblyopia	27,77.14%	7,31.81%	11,47.82%	7,70%
Facial asymmetry	0	4,18.18%	0	0
Causes	Congenital: 18 (51.42%) Traumatic: 11 (28.60%) Inflammatory: 2 (5.71%) Postviral: 1 (2.9%) Idiopathic: 3 (8.57%)	Congenital: 17 (77.3%) Idiopathic: 5 (22.7%)	Trauma: 7 (30.40%) Congenital: 5 (21.70%) Idiopathic: 4 (17.40%) Raised ICT: 3 (13.00%) Post viral: 3 (13.00%) Post neurosurgery: 1 (4.30%)	Neoplasm: 4, 40% Congenital: 3, 30% Trauma: 2, 20% Meningitis: 1, 10%
Diagnostic yield on imaging	31.57% (35.71% in acquired cases)	0%	33.33% (44.44% in acquired cases)	70% (All acquired cases)
Management (Conservative Vs Surgical)	16:19 (45.7% vs 54.30%) Squint: 14 Squint + ptosis: 5	13:9 (59.10% vs 40.90%) Squint: 9	12:11 (52.17% vs 47.82%) Squint: 11	3:7 (30% vs 70%) Squint: 5 Ptosis: 2

Combined cranial nerve palsy

Ten patients had combined cranial nerve palsy; six were bilateral and four were unilateral. The etiology included intracranial neoplasm (n = 4, 40%) followed by congenital (n = 3, 30%), trauma (n = 2, 20%), and meningitis with hydrocephalus (n = 1, 10%). The details of various nerves involved in combined cranial nerve palsy have been depicted in Table 3. The neuroimaging yield was 70% (n = 7). Findings seen on neuroimaging included space occupying lesion (SOL) (n = 4, 40%), intracranial hemorrhage (n = 2, 20%) (midbrain and multiple extra and intra-axial hemorrhage, respectively), and hydrocephalus (n = 1, 10%). The SOLs included meningioma [Fig. 1d], medulloblastoma, pilocytic astrocytoma, and intracranial neurofibroma. Seven (70%) patients required surgical correction in the form of surgery for squint, ptosis, or both, while three (30%) were managed conservatively.

The neuroimaging findings for different cranial nerve palsies have been summarized in Table 4.

Discussion

The most common pediatric extraocular motor cranial nerve palsy noted in the present cohort of patients was third (oculomotor) nerve and the mean age at presentation was 7.02 ± 4.3 years. This is in contrast to the study by Holmes and Harley *et al.* who noticed fourth (trochlear) and sixth (abducens) cranial nerve palsy to be the most common pediatric extraocular cranial nerve palsy, respectively.[1,3] The most common cause of third and fourth cranial nerve palsy was congenital, while it was trauma for the sixth nerve in the present cohort. A significant difference in the mean visual acuity was noted between the involved and uninvolved eye ($P < 0.05$). The mean visual acuity of the involved eye was 0.47 ± 0.49 Log Mar units, while it was 0.20 ± 0.29 Log Mar units for the uninvolved eye. We found amblyopia to be present in more than half of the cohort (n = 52, 57.77%), and it was most commonly associated with third cranial nerve palsy (n = 27, 75%) ($P < 0.05$). This finding has rarely been noted in the previous studies. This is important as it re-emphasizes the need for early management in such cases. Neuroimaging yield was the maximum for combined cranial nerve palsy, while imaging was normal in all cases of the fourth cranial nerve palsy. Surgical correction in the form of squint or ptosis correction or both was most commonly needed for the combined cranial nerve palsy group followed by the third cranial nerve palsy.

Table 3: Details of combined cranial nerve palsy

Etiology	Cranial nerves involved
Medulloblastoma (n=1)	Right: VI, VIII Left: V, VI, VII
Cavernous sinus meningioma (n=1)	Right: III, IV, V
Cerebellar pilocytic astrocytoma (n=1)	Bilateral: VI Left: VII
Intracranial neurofibroma (n=1)	Right: III, IV, V, VI Left: VI, VII
Trauma (n=2)	Right III, VI Left III, VI
Meningitis (n=1)	Bilateral: VI Right: IV
Congenital (n=3)	Left: IV, VII Right: VI Left: VII Bilateral : VI, VII

Children with extra-ocular motor cranial nerve palsy may not always complain of diplopia but may present with complaints of ptosis, strabismus, and AHP, depending upon the cranial nerve involved. In our study, only eight (8.88%) patients presented with diplopia. This once again implies the amblyogenic potential of cranial nerve palsy in children.

While only 15% of the cases in the series by Harley were of congenital origin,[3] almost 48% of our cases were congenital. However, our study is in accordance with the study by Holmes *et al*., where 44% of the cases were congenital in origin.[1]

In agreement with other studies, the most common cause of the pediatric third nerve palsy in the present series was congenital.[4-6] Congenital third nerve palsy can result from birth trauma due to prolonged labor, forceps injury which may lead to compression of the nerve at the tentorium.[7] It may also occur from trauma during amniocentesis, congenital absence of the third nerve nucleus, in association with septo-optic dysplasia and PHACE syndrome.[8-11] The most frequent cause of the acquired third nerve palsy in our study was trauma. Though it was in accordance with the study done by Keith, Miller, and Kodsi *et al*.,[4,12,13] Harley and Park *et al.* reported vascular and neoplastic etiologies to be the most common acquired causes, respectively.[3,14] Vascular causes of oculomotor palsy, mainly cerebral aneurysm, are rare in children and are reported to account for 7% of these nerve palsies in them.[4,6] Though 10% of pediatric oculomotor nerve palsies have been reported to be of neoplastic origin, we did not have any case of neoplasm in our study.[4,6] Some rare neuroimaging findings noted in this study were intracranial hemorrhages and open lip schizencephaly. Extradural or subdural hematoma can cause pressure effect over third nerve as it passes over the tentorial edge. Though intracranial hemorrhages in our study were not massive enough to cause direct injury to oculomotor nerve, there are reports of oculomotor nerve palsy after subtle head injury stressing upon shearing injury to oculomotor nerve resulting from differential movement between brainstem and supratentorial structures.[15] Though we cannot deny the fact that it can be an incidental finding reflecting upon the severity of injury. Schizencephaly has rarely been associated with hypoplastic oculomotor nerve and its palsy and strabismus.[16,17] Though we cannot completely rule out the fact that this can be an incidental finding too. The vision was most severely affected in the third nerve palsy group and it was most likely due to the combination of strabismus, ptosis, and anisocoria. Regarding management, 40% of patients required only squint surgery, and 14.28% of patients required both squint and ptosis surgery, which implies that the need for multiple surgeries was more in this group.

Harley *et al.* reported that 67% of their cases of fourth cranial nerve palsy were congenital, while Kodsi *et al.* reported that no cause could be determined in 21% of their cases.[3,13] Our study echoes these findings as far as the fourth cranial nerve palsy is concerned. Seventy-seven percent of our cases too were congenital, while 22.7% of the cases were idiopathic. Trauma has been mentioned as the most common cause of acquired pediatric fourth cranial nerve palsy;[3,18-21] however, none of

Table 4: Neuroimaging findings

Type of cranial nerve palsy	CT	MRI	Both CT and MRI
Third	*n*=4 Multiple orbital fractures +_ intracranial hemorrhage : 2 WNL: 2	*n*=14 Calcified granuloma in the cavernous sinus: 1 Old left parietal lobe hemorrhage: 1 Open lip schizencephaly: 1 WNL: 11	*n*=4 CT: Fracture of multiple orbital walls +_ intracranial hemorrhage: 2 WNL: 2 MRI: Resolving intracranial hemorrhage with gliosis: 1 Tolosa Hunt syndrome: 1 Orbital apex hematoma: 1 WNL :1
Fourth	Nil	*n*=5 All WNL	Nil
Sixth	*n*=4 Multiple orbital wall fracture with skull base fracture: 2 Old intracranial hemorrhage :1 WNL: 1	*n*=8 Nonspecific HIE changes: 4 Thickened sixth nerve: 1 WNL: 3	Nil
Combined	*n*=2 Intracranial hemorrhage +_ fracture of orbital walls: 2	*n*=7 Cerebellar pilocytic astrocytoma: 1 Medulloblastoma: 1 Intracranial NF: 1 Meningioma: 1 Nonspecific changes: 3	*n*=1 CT: Meningitis MRI: Dilated fourth ventricle and aqueduct

CT: Computed tomography, MRI: Magnetic resonance imaging, WNL: Within normal limits, HIE: Hypoxic ischemic encephalopathy, NF: Neurofibroma

our cases had a history of trauma. Visual acuity was noted to be the best in this group, and this can be attributed to AHP seen in 86.36% of the cases. Neuroimaging was nondiagnostic in all of the cases since a majority of the cases were congenital and idiopathic.

Different studies have noted different causes as the most frequently encountered ones for pediatric sixth cranial nerve palsy. While it was trauma by Harley and Kodsi *et al.*,[3,13] Holmes *et al.* reported the most common cause to be idiopathic.[1] Various other authors have found neoplasms to be the most common etiology.[14,22,23] Trauma was the most common cause for pediatric sixth nerve palsy in the present cohort. In contrast to Harley *et al.* who noticed that 14.5% of their pediatric sixth nerve palsies were of congenital origin[3], and Park *et al.* who did not find any congenital case,[14] 21.7% of the cases in the present cohort were of congenital origin. Rare causes of pediatric sixth nerve palsies reported in the literature include ophthalmoplegic migraine,[24] Gradenigo syndrome,[25,26] iatrogenic causes including a lumbar puncture and myelography.[27,28] One patient in our cohort underwent neurosurgery for cerebellar pilocytic astrocytoma and developed shunt failure, consecutive hydrocephalus, and bilateral sixth nerve palsy post-surgery. Although neoplasm was seen, we do not believe that it was the cause of the sixth nerve palsy. The palsy developed post operatively. Though none of the patients in the present series with isolated sixth nerve palsy had neoplastic etiology, some unusual findings noted were skull base fracture and intracranial hemorrhage. Various authors have reported association of sixth nerve injury and basal skull fracture, while few have advocated the indirect mode of injury in setting of trauma.[29-31] The abducens nerve has three angulations in the petroclival region: at the dural entry point, the petrous apex, and at the point where it joins the lateral wall of the internal carotid artery. These angulations are very much vulnerable to injuries. Intracranial hemorrhage reflects the severity of head injury and associated nerve palsy can be a result of indirect injury caused by the downward displacement of the sixth nerve and resultant contusion at these angulations. We feel that intracranial hemorrhages present in our study reflect upon the severity of injury rather than a direct causal relationship**.** Sixth nerve palsy in the pediatric age group causes significant amblyopia.[32] In the present study, amblyopia was found in 11 (47.82%) patients. Diagnostic yield of imaging was found to be 44.44% for acquired cases, which implies that every case of acquired sixth nerve palsy requires imaging. The modality of imaging should be MRI brain and orbit with contrast in cases of suspected intracranial pathologies (neoplasms and inflammation), while it should be CT orbit with brain in post-traumatic cases. Some cases might need both CT and MRI, in situations where one particular modality is not sufficient enough to provide all the relevant details. Sixth nerve palsy due to trauma may resolve spontaneously. Hence, children with post-traumatic sixth nerve palsy should be observed for at least 6 months before any surgical intervention.

Ten patients had features of combined cranial nerve palsy in the present cohort. Out of 10 patients, four had intracranial neoplasm, which included medulloblastoma, cavernous sinus meningioma, cerebellarpilocytic astrocytoma, and intracranial neurofibroma. Posterior fossa tumors (medulloblastoma and pilocytic astrocytoma) can cause multiple cranial nerve palsy because of associated hydrocephalus, while cavernous sinus masses can cause multiple cranial nerve palsies because of their anatomical proximity. Association of mid brain hemorrhage and third and fourth nerve palsy is a reported entity,[33,34] and mechanism of indirect trauma related cranial nerve palsy has already been discussed in the context of third and sixth nerve palsy. The study done by Kodsi *et al.* found trauma to be the most common cause for multiple cranial nerve palsy in the pediatric age group followed by neoplasm.[13] Harley found

Table 5: Review of literature

Parameters	Harley[3]	Kodsi *et al*.[13]	Holms *et al*.[1]	Park *et al*.[14]	Present study
Sample size	121	160	35	66	90
Mean age (Years)	<16	9.4 +_5	<18	9 +_6	7.02 +_4.3
Male:Female	Not mentioned	1.46:1	1.25:1	1.86:1	2.33:1
Presenting features	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Deviation of eye ball (68.88%) Drooping of eye lid (34.06%) Abnormal head posture (20.87%) Diplopia (8.79%)
Type of motor nerve palsy	Congenital + acquired	Only acquired cases	Congenital + acquired	Congenital + acquired	Congenital + acquired
Nerve involved (Percentage)	Third: (26.4) Fourth: (14.8) Sixth: (51.2) Multiple: (7.4)	Third: (21.9) Fourth: (11.9) Sixth: (55) Multiple: (11.3)	Third: (22) Fourth: (36) Sixth: (33) Multiple: (9)	Third: (21.2) Fourth: (19.6) Sixth: (53) Multiple: (6)	Third: (38.8) Fourth: (24.4) Sixth: (25.5) Multiple: (11.1)
Cause of cranial nerve palsies (Percentage)	Congenital (32.14) Trauma (26.78) Neoplastic (17.85) Inflammatory (10.71)	Trauma : (42.5) Neoplasm: (16.9%) Undetermined (14.4%) Post-operative (3.8%) Meningitis (3.1%) Hydrocephalus (2.5%)	Congenital: (44) Trauma: (25) Undetermined: (14) Neoplasm: (8) Viral: (6) Others: (3)	Neoplasm: (23) Idiopathic: (14) Inflammatory: (11) Nonaneurysmal vascular contact: (11) Trauma: (9) Miller Fisher syndrome: (9) Congenital: (9)	Congenital: (48.9) Trauma: (21.1) Idiopathic: (12.2) Post viral: (5.6) Raised intracranial tension: (4.4) Neoplasm: (3.3) Meningitis: (1.1)
Cause of combined cranial nerve palsy (Percentage)	Not Mentioned	Trauma: (55.5) Neoplasm: (16.7) Aneurysm (11.1) Post-operative (5.6) Others (5.6)	Trauma (100)	Cavernous sinus thrombosis: (25) Miller fisher syndrome: (75)	Congenital: (30) Trauma: (20) Neoplasm: (40) Meningitis: (10)
Neuroimaging yield	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Normal :(47.05%) Significant: (31.37%) Coincidental findings:(21.56%)
Management details	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Conservative: 48.88% Surgical: 51.11%

orbital inflammation to be the most common cause followed by trauma and neoplasm.[3] Holmes *et al*. reported all the cases of multiple cranial nerve palsy to be post-traumatic.[1] Miller Fisher syndrome is an important cause for multiple ocular motor cranial nerve palsy and the patients should be screened for anti GQ1B antibody wherever indicated.[35] Compared to the isolated cranial nerve palsy group, the neuroimaging yield was maximum and found to be 70%, and it was 100% for acquired cases.

The overall neuroimaging yield for the present cohort was around 31.37%, and it was 45.71% for acquired ones which implies that one should have a low threshold for imaging, especially for acquired cases. In the present study, neoplastic etiology for isolated cranial nerve palsy was significantly less than the previous studies [Table 5]. Kodsi *et al*. feel that in this era of advanced diagnostic techniques, diagnosis of neoplasm can be efficiently done by the neurologists much before the development of nerve palsy.[16] Ophthalmologist referral for diagnosis of cranial nerve palsy may not be sought due to early diagnosis and presumption of the neoplasm to be a cause of cranial nerve palsy.[16] This might be the reason for less number of intracranial neoplasms noted in the present study too.

Though we have tried our best to present our comprehensive analysis of pediatric extraocular motor cranial nerve palsies; the retrospective design is an inherent limitation of the study. Neuroimaging and surgical treatment were not opted by quite a few parents due to financial constraints, which could have led to nonreporting of few of the causes. Ours being a tertiary care institute, the number of patients needing surgical correction might be higher due to referral bias and the whole data too may not be completely representative of the general population. We have not analyzed the management outcome since it was out of the scope of the present study, and it would have made the article unduly lengthy and confusing.

Conclusion

The third cranial nerve is the most commonly involved amongst pediatric ocular motor cranial nerve palsies and it is the most amblyogenic one. Congenital etiology is almost equally common as acquired cranial nerve palsy in the pediatric age group and among acquired causes; trauma is the major culprit. Though rare, there should be a high index of suspicion for intracranial neoplasm. Most cases of fourth cranial nerve palsy can be managed conservatively, while surgeries are more commonly done for patients with third and combined cranial nerve palsy.

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Conflicts of interest

There are no conflicts of interest.

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Commentary: Pediatric ocular motor cranial nerve palsy: Demographics and etiological profile

The authors have presented an interesting article titled "Pediatric Ocular Motor Cranial Nerve Palsy: Demographics and Etiological Profile" on the etiology and clinical characteristics of pediatric III, IV, and VI cranial nerve palsies based on a retrospective study done in a tertiary care center in India. As mentioned by the authors, palsies of cranial nerves III, IV, and VI are rare in childhood, with an estimated incidence of 7.6 per 100 000 for all three palsies determined on a population-based study.[1] There are limited studies on extraocular motor cranial nerve palsies in children in recent years. Also, most were institution-based studies. Population-based studies derived from databases of epidemiological studies were rare.[1]

The authors have showed that the most common extraocular motor cranial nerve palsy in their cohort was third nerve and the mean age of presentation was 7.02 ± 4.3 years. Homes *et al*. reported that fourth nerve palsy was the most common occurring in children.[1]

Many large pediatric studies reported that the most common etiology of pediatric third (oculomotor) nerve palsy was congenital followed by trauma.[1-3] The present study also reported similar findings. Neoplasm has been a rare etiology.[1] Interestingly in a suprasellar teratoma third nerve palsy was noted after surgical resection. Aberrant regeneration which happens from misrouting of regenerating fibers is quite common