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Original Research Article

Prevalence of Movement Disorders in Pediatric Age Group

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ABSTRACT

The objective of this study outlines the age & sex of distribution, pathophysiology, clinical presentation, various types of movement disorders, their incidence, diagnostic modalities with diagnostic differences and therapy of movement disorders and outcome in children. This prospective study design was done over a period one year. Patients were either admitted in the wards or were attending our special Pediatric neurology clinic at our tertiary referral centre. Total of 64 patients were under 15 years of age. Interventions were done depending on the possible etiology; the patients were subjected to specific investigations. Various modalities of treatment given to our study group were surgical, medical and other therapies. The manuscript does not plan to report the outcome**.** The results indicated that in our study group, 18 cases (28.08%) showed complete recovery at the end of 3 months while 27 patients (42.18%) had partial improvement. 11 cases (17.18%) had persistence of movements while 5 cases didn't follow up (7.80%). 3 patients died in our series. The conclusions characteristics of movement disorders in children are different compared to adults. Movement disorders, if diagnosed early and treated promptly have good prognosis. The patients showing complete recovery were generally those with secondary to transient etiologies such as drug toxicity, acute cerebellar ataxia and TBM. Some special circumstances like inborn errors of metabolism and genetic conditions selectively affect the immature developing brain and outcome of the disease.

Keywords: Movement Disorders, Pediatric.

INTRODUCTION

Movement disorders are the abnormal or excessive involuntary movements that may result in posture, tone, balance, or fine motor control. Most movement disorders are characterized by involuntary movements. [1] Abnormal involuntary movements can be the primary presenting feature of a disease or they can occur as a late manifestation. They are frequently observed in many neurological illnesses, endocrine conditions, metabolic disorders, drug toxicities or psychiatric illnesses and pose a diagnostic challenge to paediatrician. Most movement disorders related to dysfunction of the basal ganglia or regions of brain interconnected with the basal ganglia. The basal ganglia, a network of nuclei in the forebrain, diencephalon and midbrain. [2-4] Dystonia results from lesions in striatum (putamen or caudate), globus pallidus, or thalamus. [5,6] These may subsequently lead to dysfunction in the cerebral cortex and brainstem. [7] The underlying systemic disease requires thorough investigation and management plan to achieve the most favourable outcome. Why Movement disorders in children are different as compared to adults? :

A. Several of the movement disorders described in children are characterised

by transient states that last for days, weeks or months with complete clinical resolution in most cases. Hence the more benign, transient state needs to be differentiated from devastating long standing disorders.

B. Furthermore, children show fluctuations in their clinical manifestations, such as temporary deterioration in children with dystonia or chorea with intercurrent infections. The possibility that PANDAS (pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections) is also a post streptococcal disorder in some has been raised. [8]

C. Children often show a latent period of presentation for example, onset of dystonia in a child with cerebral palsy is much later than its other clinical manifestations. [9]

A movement disorder is a broad term which encompasses the extrapyramidal disorders along with disorders of incoordination and even corticospinal tract dysfunction including spasticity. They are usually associated with abnormalities of basal ganglia.

Movement disorders in children are broadly classified into hypokinetic and hyperkinetic disorders. [10] Hypokinetic movement disorders are characterised by paucity of movement such as akinesia or bradykinesia in which purposeful motor activity is absent or reduced for example, Parkinson's disease. Hyperkinetic movement disorders are those in which an excessive amount of spontaneous motor activity is seen or in which abnormal involuntary movements occur. Hyperkinetic movements are further classified into rhythmical (e.g. tremors) and non rhythmical (rapid such as tics, chorea, ballismus and myoclonus or slow such as athetosis and dystonia). [11] Cerebral palsy is categorized into spastic, extra-pyramidal or dyskinetic, and mixed. [12,13] Perinatal asphyxia in term infants accounts for less than 15% of CP in developed countries with a higher incidence in underdeveloped areas. [14]

Categorisation of movement disorders into various types is done using the amplitude and rhythmicity. Clinical observation is the mainstay of diagnosis. Furthermore, many systemic disorders manifest with several movement disorders (e.g. tremor, dystonia and rigidity in Wilson's disease) and at any point, one manifestation may be prominent and may mask other movements. Additionally, most children attempt to mask the abnormal movement by combining them with normal movement patterns, in order to avoid embarrassment. Thus even most experienced observer will at times mistake one movement for another or will have difficulty conceptualizing the nature of an abnormal movement. Videotaping, at home or in clinics / hospital, proves extremely beneficial.

The spectrum of pathophysiology of movement disorders in children range from structural abnormalities, genetics, neuroimmunologic basis to psychogenic causes. Structural abnormalities or lesions of the central nervous system and of basal ganglial connecting structures may be delineated in some cases of abnormal involuntary movements with the help of neuroimaging. Recent studies have found that movement disorders have genetic basis (Huntington's disease) or a neuroimmunologic basis (e.g. Sydenham's chorea, Tourette syndrome). [15,16] A psychogenic especially in preschool and school going children.

The workup can range from simple blood count, ESR to metabolic work up such as measurement of electrolytes, calcium, glucose etc. Suspected drug toxicities need measurement of drug levels. Neuroimaging especially MRI is very useful. PET and SPECT scans drastically revolutionised the early diagnosis, detection of underlying etiology, follow up and prognostication. If all the above tests are inconclusive, then in such cases, genetic studies are advised.

Various treatment options in such cases include drugs such as neuroleptics, benzodiazepines and β-blockers. They are started with mild drugs and in smaller doses and titrated according to the response. [15,17,18]

We undertook this study to determine the prevalence of movement disorders in different age and sex group, to evaluate their underlying illnesses, to study neuroimaging findings of these patients and treatment outcome.

Aims and Objectives:

1) To estimate the prevalence of movement disorders in different age and sex group.
2) To determine the spectrum of involuntary movements in children.
3) To evaluate underlying etiologies of movement disorders.
4) To study the patterns of clinical presentations of children with movement disorders.
5) To correlate neuroimaging findings in patients with movement disorders.
6) To study efficacy of various treatment options for children with abnormal movements and to determine their outcome.

MATERIALS AND METHODS

This prospective study was done over a period one year and included 64 patients under 15 years of age who were either admitted in the wards or were attending our special Pediatric neurology clinic at our tertiary referral center. Permission of ethical committee was taken. This article adopted STROBE as the standard method for the study design.

Inclusion criteria:

1) Age less than 15 years
2) Patients with any form of involuntary movement.

Exclusion criteria:

1) Non availability of consent

A detailed history was taken with special emphasis on family history, age of onset, pattern of time of onset, presence of infection, exposure to medication, koch's contact etc.

The description of the movement disorder as told by the parents and as observed in wards was the basis for classifying them.

A thorough clinical examination was performed with detailed anthropometric measurements. Abnormal facies, neurocutaneous markers, eye and ear abnormalities were looked for. Vital parameters of patients were recorded. A detailed neurological examination was done with special attention of higher functions, cranial nerve examination, motor and sensory system, cerebellar signs and gait. Abnormal movements were aggravated by asking to perform special tasks relating to the type of involuntary movement.

Depending on the possible etiology, the patients were subjected to specific investigations. These are from ESR, complete blood count, metabolic parameters like RBS, calcium, magnesium, x-ray chest, EEG, MT, neuroimaging (CT/MRI), 24hr urinary copper, serum ceruloplasmin, IQ/DQ, BERA, serum drug levels etc.

Statistical data were compared using chi-square test and the chi-square test of goodness of fit.

$P<0.001$ was statistically highly significant.

$P<0.05$ was considered statistically significant.

$P>0.05$ was considered statistically insignificant.

RESULTS

Table 1- Age and sex distribution of study cases

Age group in yrs	Male (n=38)		Female (n=26)		Total (n=64)	
	No.	%	No.	%	No.	%
0-1	05	7.81%	03	4.82%	08	12.63%
1-3	09	14.04%	05	7.81%	14	21.85%
3-5	09	14.04%	04	6.24%	13	20.28%
5-15	15	23.40%	14	21.84%	29	45.24%
Total	38	59.29%	26	40.71%	64	100.00%

Table 2 - Spectrum of Movement Disorders

Sr .no.	Type of movement disorders	No. of Cases (n=68)	Percentage
1)	Ataxia	09	13.23%
2)	Chorea	02	2.94%
3)	Choreoathetosis	03	4.41%
4)	Dystonia	05	7.35%
5)	Hemiballismus	11	16.20%
6)	Myoclonus	02	2.94%
7)	Tics	07	10.29%
8)	Tremor	26	38.23%
9)	Unclassified	03	4.41%
	Total	68	100%

Table 3 – Classification of various movement disorders in study group

Sr.no.	Movements	No. of cases	%
1)	Hypokinetic	02	2.94
2)	Hyperkinetic	57	83.82

Table 4 – Algorithm Showing Various Movement Disorders in Our Study Group

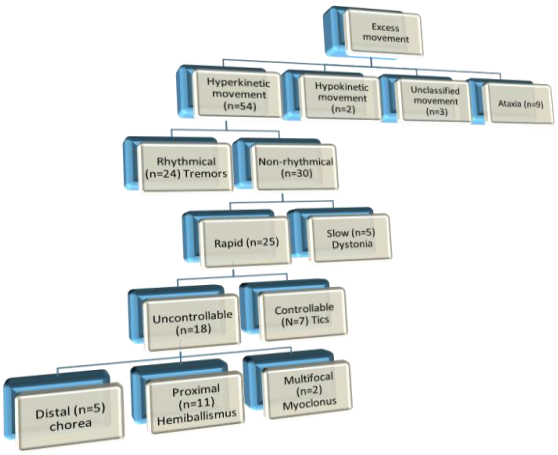

Table 5 – Distribution Of Movement Disorders In Age And Sex Groups

Sr. no.	Type of movement disorder	Male				Female				Total
		0-1	1-3	3-5	5-15	0-1	1-3	3-5	5-15	
1	Ataxia	-	-	02	03	-	-	-	04	09
2	Chorea	-	-	-	01	-	-	-	01	02
3	Choreoathetosis	-	-	01	01	-	01	-	-	03
4	Hemiballismus	-	02	01	-	03	03	02	-	11
5	Myoclonus	-	-	-	01	-	-	-	01	02
6	Dystonia	01	02	-	01	-	01	-	-	05
7	Tics	-	03	-	01	-	-	-	03	07
8	Tremors	02	02	05	08	-	-	02	07	26
9	Unclassified	02	-	-	01	-	-	-	-	03
	Total	05	09	09	16	03	05	04	16	68

Table 6 – Mode Of Presentation Of Various Movement Disorders

A)Primary complaint of involuntary movement only	13	20.28%
B)nvoluntary movement along with other symptoms	16	24.96%
C)Only other symptoms (abnormal movement on clinical examination)	35	54.76%

Table 7 – Etiology Of Movement Disorders

I Neurological causes (n=46)	**No. of cases**	**%**
A) Infective (n=26)		
1)CNS Tuberculosis	17	26.52%
2)Post- varicella	02	3.12%
3)Post- measles	02	3.12%
4)Brain abscess	02	3.12%
5)Viral Meningoencephalitis	02	3.12%
B) Non-infective (n=20)		
1)Cerebral Palsy	08	12.48%
2)Delayed milestones with MR	03	4.68%
3)HSMN type II	02	3.12%
4)Post traumatic	02	3.12%
5)Pontine glioma	01	1.56%
6)Wilson's disease	01	1.56%
7)DRD	01	1.56%
8)Huntingtons disease	01	1.56%
9)Holoprosencephaly	01	1.56%
II. Non neurological causes (n=18)	**No. of cases**	**%**
Drug toxicity	10	15.60%
Psychogenic	04	6.24%
Nutritional recovery syndrome	02	3.12%
Sydenham's chorea	01	1.56%

Table 8 – Congenital vs acquired etiology

Congenital	06	9.36%
Acquired	58	90.64%

Table 9 –

A) Congenital (n=6) (9.36%)

Causes	No.	Involuntary Movement
1.HSMN type II	02	Choreoathetosis and unclassified
2.Holoprosencephaly	01	Ataxia
3.Wilsons disease	01	Tremors
4.Huntingtons disease	01	Chorea
5.DRD	01	Dystonia and tremors

B) Acquired (n=58) (90.64%)

Causes	No. Of cases
1.Neurological	40
a.Infective	26
b.Non infective	14
2.Non neurological	18
a.Drug induced	10
b.Psychogenic	04
c.Nutrition – nutritional recovery syndrome	02
d.CVS – rheumatic chorea	01
e.Metabolic - hyperthyroidism	01

Table – 10 – Demographic and etiological profile of patients with tremors

A) Demographic profile (n=26)

	Male (n=17)	Female (n=9)
0-1 yr	02	-
1-3 yr	02	-
3-5 yr	05	02
5-15 yr	08	07

B) Etiological profile

Neurological (n=15)(57.69%)				Non-neurological (n=11) (42.31%	
Infective		Non-Infective			
CNS TB	05	Post traumatic	02	Drug induced	08
Brain abscess	02	CP	01	Hypertyroidism	01
Pyo. Men	01	Delayed milestones	01	Nutritional recovery syndrome	02
Viral ME	01	Wilsons disease	01		
		DRD	01		

C) Type of tremors

Resting	02
Postural	01
Intentional	23

Table 11 – Profile of Patients With Ataxia

Sr. no.	Age/sex	Diagnosis	Treatment	Outcome
1	13 yr	Phenytoin toxicity in a case of post traumatic lt.Hemiparesis	Phenytoin dose decreased	Complete recovery
2	12 yr	Delayed milestones with severe MR with GTC epilepsy with valparin toxicity	Dose of valproate reduced	Complete recovery
3	7 yr /M	Holoprosencephaly	S	Lost to followup
4	7 yr / F	Pontine glioma	Radiotherapy + S	Lost to followup
5	10 yr	Phenytoin toxicity in a k/c/o TBM with seizure disorder	Reduce dose of phenytoin	Complete recovery
6	9 yr	Phenytoin toxicity in c/o head injury with # Rt. Frontal bone	Reduce dose of phenytoin	Complete recovery
7	8 yr	Phenytoin toxicity in c/o viral meningoencephalitis	Antibiotics	Complete recovery
8	3½ yr/M	Acute cerebellar ataxia	S	Complete recovery
9	4 yr /M	Acute cerebellar ataxia	S	Complete recovery

Table 12 – Spectrum of Abnormal Movements in CNS Tb (N=17)

A)	Hemiballismus	11	64.70%
B)	Tremors	04	23.52%
C)	Unclassified	02	11.78%

Table 13 – Spectrum of Abnormal Movements in Cerebral Palsy (N=8)

A)	Tremors	02	25%
B)	Choreoathetosis	02	25%
C)	Dystonia	04	50%

Table 14 – Neuroimaging Findings In Study Cases (N=46)

Type of imaging	Finding	Case
A) MRI (n=19)	1.Periventricular leukomalacia	04
	2.Cystic encephalomalacia	07
	3.Pontine glioma	01
	4.Hyperintense basal ganglia	01
	5.Volume loss	01
	6.Normal	05
B) CT (n=27)	1.Hydrocephalus	16
	2.Basal exudates	13
	3.Infarcts	09
	4.Tuberculosis	04
	5.VP shunt in situ	04
	6.Brain abscess	02
	7.Holoprosencephaly	01
	8.IV Bleed with PV ooze	01
	9.Extrapontine Myelinolysis	01
	10.Normal	01

Table 15 – Localization of Specific Anatomic Site on Neuroimaging

	Neuroimaging findings	MRI (n=19)		CT (n=27)	
		No.	%	No.	%
A)	Showing specific basal ganglial affection	1	5.27%	07	25.93%
B)	Features of underlying neurological disease only	18	94.73%	20	74.07%

Table 16 – Movement Disorders Giving Direct Clue To Underlying Etiology

Sr.no.	Type of movement disorder	No. of cases	Underlying etiology
1.	Myoclonus	02	SSPE
2.	Choreoathetosis	02	Kernicterus
3.	Hemiballismus	10	TBM

DISCUSSION

A total of 64 cases below 15 years were evaluated in our prospective study.

1) Age and Sex Distribution

There was a male preponderance with ratio of 1.4:1. The age group of the patients ranged from 3 months- 13 years and the mean age of study cases was 5.4 years. Max. numbers of cases were from 5-15 years (45.24%). We had 8 infants of which 5 were males (62.5%). Sex wise

distribution of cases into various age group were statistically not significant (p>0.05).

Table 17 – Various Treatment Modalities in Our Study Cases

Sr.no.	Mode of treatment	No. of cases
A)	**Surgical**	
	VP shunt	17
	Aspiration	02
	Burr hole	08
B)	**Medical**	
	1.Underlying illness	
	a.AKT (cat V IAP)	17
	b.AED	16
	c.D-penicillamine	01
	d.Propylthiouracil	01
	2.Specific	
	a.Levodopa	01
	b.Diphenhydramine	02
	c.Diazepam	02
	d.Baclofen	03
	e.Trihexyphenidyl	01
C)	**Others**	
	1.Radiotherapy	01
	2.Physiotherapy	18
	3.Psychotherapy	04

Table 18 – Outcome in Our Cases at the End of 3 Months

Sr. no.	Outcome	No. of cases	%
1.	Completely improved	18	28.08%
2.	Partially improved	27	42.18%
3.	Persistence of abnormal movement	11	1.26%
4.	Lost to follow up	05	7.80%
5.	Died	03	4.68%
	Total	64	100%

2) Spectrum of Movement Disorders

Tremors were the most common involuntary movement in study comprising of 38.23% of cases followed by Hemiballismus (16.20%).

Ataxia (13.23%), Tics (10.29%), Dystonia (7.35%), Choreoathetosis (4.41%), Myoclonus (2.94%) and chorea (2.94%) were other involuntary movements observed. We encountered 3 cases which did not classify under any term. Tremors were found to be statistically significantly higher in proportion than other movement disorders (p<0.05). In contrast, Most of the popular studies have documented tics as the most common involuntary movement in pediatric age group. [19]

3) Classification of Abnormal Movements into Hypokinetic and Hyperkinetic Movements

In our study we had only 2 cases of hypokinetic movements (3.12%) while 62 cases were with hyperkinetic movements. 2 cases of hypokinetic movements included one case of resting tremors in a patient of Wilson's disease and fine tremors in the case of dopa responsive dystonia who in addition to dystonia had parkinsonian like symptoms. [20,21] The difference in proportion between hypokinetic and hyperkinetic movements was found to be statistically highly significant (p<0.005). [22,23]

4) Classification of Abnormal Movements as Per the Algorithm

In our study, among the hyperkinetic movements (96.88%) observed, 24 were rhythmical tremors while 30 had non rhythmical hyperkinetic. Among non rhythmical movements, 25 were rapid and only 5 were slow (dystonia). Only 7 had controllable rapid movements while 18 had uncontrollable movements among which hemiballismus was most common (61.1%). 3 patients had movements which didn't classically fit under any, while 9 cases had ataxia.

5) Correlation of involuntary movement with age and sex

Males have more tremors (65.38%) as compared to females (34.62%). In both sexes tremors were most commonly seen in the age group of 5- 15 years. Hemiballismic movements were common in females where others were common in males. All cases of hemiballismus were below age 5 years. This is because TBM. Ataxia in 5-15 years. Statistically insignificant.

6) Mode of Presentation of Study Cases

We observed that 13 cases (20.28%) had presented only with complaint of involuntary movement. This was tics (05), drug toxicity (03), post infectious acute cerebellar ataxia (02), Wilson's disease, dopa-responsive dystonia and Sydenham's chorea. Other 16 cases (24.96%) complained of involuntary movement but had other complaints in addition, while 35 cases (54.76%) had not presented with involuntary movement. They were noticed by us on examination. The presentation of patients without any complaint was

statistically significant higher in proportion as compared to those presenting with abnormal movement ($p<0.05$).

7) Underlying Etiology of Movement Disorder

Neurological etiologies (71.87%) were higher than non-neurological causes (28.13%). The neurological causes were more commonly seen secondarily to infections (56.52%) with CNS tuberculosis causing most of involuntary movement in that group (65.38%). Other infectious causes were post varicella (7.69%), post measles (7.69%), viral meningoencephalitis (7.69%), brain abscess (7.69%), and pyogenic meningitis (3.86%). Among neurological non-infectious etiologies (43.48%), cerebral palsy (40%) was the most common cause while other causes observed were delayed milestones with MR (15%), post traumatic (10%) and congenital causes.

In non-neurological etiology group (28.13%), drug toxicity induced abnormal movements were most common factor. (55.56%)

8) Congenital vs acquired

In our study, only 6 cases had their abnormal movements secondary to etiology of congenital group (9.36%) and 58 cases had etiologies of acquired group. (90.64%)

9) Correlation of Different Movement Disorder and Etiology

A. We observed that out of 26 cases of tremors, 16 cases (61.53%) were due to neurological causes while 11 cases (38.47%) were due to non-neurological causes. Amongst neurological, infective causes were common with CNS TB causing tremors. The non infective neurological causes included 2 cases with post traumatic etiology and one case each of cerebral palsy, delayed milestone, Wilson's disease and DRD. The non-neurological causes for tremors included drug toxicity induced movements in 72.72% cases, nutritional recovery syndrome (18.18%) and hyperthyroidism (9.10%).

B. In the group of patients with hemiballismus, all were secondary to CNS tuberculosis. This was in accordance with study of Rashmi Kumar et al. [24] who in their study of 232 cases of TBM found hemiballismus as the most common involuntary movement.

C. Of 9 cases of ataxia, 5 were secondary to toxicities of drugs like phenytoin and sodium valproate (55.56%). Acute cerebellar ataxia was seen in 2 cases (22.22%). Both these cases were secondary to varicella. Other causes of ataxia were holoprosencephaly and pontine glioma.

D. Psychogenic movement disorders were seen in 6.25% of patients. All these had motor tics and were above 5 years of age. This correlates well with study of Lempert T et al [25] who reported incidence of 2%-9% and most commonly adolescent group. Whereas Galvez-Jimenez A et al [26] in their study of 131 adult patients found dystonia in 53%, tremors in 13% and gait disorders in 9% of patients.

E. Myoclonus was seen in 2 cases of subacute sclerosing panencephalitis. Dystonia was seen in 50% of patients with cerebral palsy and in case of DRD. We had one case each of rheumatic chorea and Huntington's chorea.

10) Demographic and Etiological Profile of Patients with Tremors

In our study, tremor was most common involuntary movement (38.23%). Maximum patients (57.69%) belong to age group 5-15 years and there was male predominance (65.38%).

Neurological causes were most common etiology (57.69%) with CNS TB being most common cause. Drug induced tremors were seen in 8 cases of phenytoin and sodium valproate toxicity. Only two patients had resting tremors. Thy were the case of Wilson's disease and DRD. One

patient with hyperthyroidism had postural tremors. Rest all patients had intentional tremors.

11) Clinical Profile of Patients with Ataxia

In our study we had 9 cases of ataxia, most commonly from 5-15 years with male predominance. Phenytoin and sodium valproate toxicity were the most common causes (55.56%). 7 out of 9 cases (77.78%) showed complete recovery on follow up.

12) Spectrum of Abnormal Movement in CNS Tuberculosis

CNS tuberculosis was the most common etiology in our study (26.56%). We found that hemiballismus was the most common involuntary movement disorder (64.70%) among CNS TB, followed by tremors (29.42%) and unclassified movement (5.88%). Similar findings were observed by Udani et al [27] who found hemiballismic movements in 63.3% and tremors in 36.3% of patients with extrapyramidal involvement in TBM.

13) Spectrum of Abnormal Movement in Cerebral Palsy

Cerebral palsy (17.39%) was the second most common neurological cause in our study group, next to CNS TB. In these cases, 4 patients had dystonic posturing (50%) while 2 patients each had tremors and choreoathetosis.

14) Neuroimaging Findings in Study Cases

In our series, 46 patients had undergone neuroimaging (71.87%), of which MRI was done in 19 cases (41.30%) and CT scan done in 27 cases (58.70%).

A. All 17 cases of TBM had undergone CT scan as an emergency procedure. Hydrocephalus was most common finding followed by basal exudates and infarcts. Only 4 patients had tuberculomas (23.52%). This correlates well with all other studies (Rashmi Kumar et al) done on neuroimaging in CNS tuberculosis. [24] Other findings on CT scan were brain abscess, holoprosencephaly, IV bleed and extrapontine myelinosis. One patient had normal scan.
B. MRI was mostly effective. 7 cases had cystic encephalomalacia. 4 had periventricular leukomalacia and 1 had hyperintense basal ganglia while patient with Huntington's disease showed frontal lobe volume loss. Normal MRI scans in 5 cases. We observed that only 7 cases (6 on CT and 1 on MRI) showed specific basal ganglial affection.

CT scan findings of hydrocephalus, infarcts and basal exudates in patients of TBM was found to be statistically significant in proportion ($p<0.001$)

15) Localization of Specific Anatomic Site on Neuroimaging

We observed that only 7 cases (15.21%) showed specific ganglial affection on neuroimaging, while remaining patients (84.79%) had underlying neurological disease on neuroimaging. Out of 7 cases, 6 cases of TBM had abnormality of basal ganglia on CT scan while 1 patient with Wilson's disease showed hyperintense basal ganglia on MRI. The difference in proportion for localising specific anatomic site on CT and MRI scans was statistically not significant ($p>0.05$).

16) Treatment Given to Study Cases

We found 14 cases (21.87%), the movement disorder in the clinical scenario gave us a clue to underlying etiology.

17) Outcome in our cases at the end of 3 months

Various modalities of treatment given to our study group were surgical, medical and other therapies.

A. A total of 17 cases had VP shunt insertion, 2 cases had aspiration and burr hole was required as an emergency procedure in 8 cases.
B. All cases of CNS TB were given AKT. 16 cases required anti-epileptic therapy.
C. Only 9 patients were treated specifically for involuntary movements. The movements were treated were dopa responsive dystonia

(levodopa); side effects were common. [28] Dystonic CP (diphenhydramine and baclofen), choreoathetosis (diazepam) and hemiballismus (trihexyphenidyl).

D. Behavioral therapy has been efficacious in motor stereotypies, [29] but not particularly helpful for patients with disabling tics. [30]
E. Other forms of therapy employed were physiotherapy (18), psychotherapy (04) and radiotherapy (01).

18) Outcome in our Cases at the End of 3 Months

In our study group, 18 cases (28.08%) showed complete recovery at the end of 3 months while 27 patients (42.18%) had partial improvement. 11 cases (17.18%) had persistence of movements while 5 cases didn't follow up (7.80%). The patients showing complete recovery were generally those with secondary to transient etiologies such as drug toxicity, acute cerebellar ataxia and TBM. The traditional treatment of Wilson's disease addresses the acute chelation of copper with δ-penicillamine, but more recent less toxic strategies are trientine and zinc or ammonium tetrathiomolybdate.[21] Three patients died in our series.

CONCLUSION

The study patients were all under 15 years of age, who were either following in our special neurology clinic or admitted in our Pediatric ward.

The maximum incidence of movement disorders was seen in 5-15 years age group with male predominance 1.4:1 ratio.

Tremors was the most common involuntary movement in our study (39%) followed by hemiballismus (17.16%) and ataxia (14.04%). Tremors, choreoathetosis and dystonia were more common in male while hemiballismus was more often seen in females.

The etiology of these various involuntary movements was either neurological (71.87%) or non-neurological (28.13%). Amongst neurological, infective causes predominated with CNS TB being most common. Cerebral palsy was the most common non infectious etiology while drug toxicity induced movement were most common non neurological, the drugs causing are phenytoin and sodium valproate. Of the various causes only 6 cases had movements secondary to congenital etiology.

Patients with CNS TB had movements ranging from tremors (29.42%), hemiballismus (64.70%) and unclassified (5.88%) movements. In children with cerebral palsy, 50% had dystonia while choreoathetosis and tremors were seen each in 25 % of cases.

Neuroimaging was done in 46 out of 64 cases. The most common finding in our study group was hydrocephalus (26.56%), infarcts (14.06%) and basal exudates (20.31%).

All patients were treated for the underlying etiology and only 9 cases required specific treatment for involuntary movement.

Amongst 64 cases, 18 showed complete recovery at the end of 3 months while 27 had partial recovery. In 11 cases there was persistence of movement on follow up while 5 lost to follow up. Three patients died in our series. These were a case of viral meningoencephalitis, pyogenic meningitis and choreoathetoid cerebral palsy.

Summary: The aim of this article is to review movement disorders in pediatric population. They are common but have etiology and different than in adults.

Conflict of interest: none

REFERENCES

1. Nelson textbook of Pediatrics. 19th edition vol.2, pg. no. 2053- 2061.
2. J. W. Mink, "The Basal Ganglia: Focused Selection and Inhibition of Competing Motor Programs," Progress in Neurobiology, Vol. 50, No. 4, 1996, pp. 381-425.
3. J. P. Bolam, J. J. Hanley, P. A. Booth and M. D. Bevan, "Synaptic

Organisation of the Basal Ganglia," Journal of Anatomy, Vol. 196, No. 4, 2000, pp. 527-542. doi:10.1046/j.1469-7580.2000.19640527.x.

4. J. W. Mink, "The Basal Ganglia and Involuntary movements: Impaired Inhibition of Competing Motor Patterns," Archives of Neurology, Vol. 60, No. 10, 2003, pp. 1365-1368. doi:10.1001/archneur.60.10.1365.
5. C. D. Marsden, J. A. Obeso, J. J. Zarranz and A. E. Lang "The Anatomical Basis of Symptomatic Hemidystonia." *Brain*; Vol. 108, No. Pt. 2, 1985, pp. 463-483. doi:10.1093/brain/108.2.463.
6. K. P. Bhatia and C. D. Marsden, "The Behavioural and Motor Consequences of Focal Lesions of the Basal Ganglia in Man," *Brain*, Vol. 117, No. 4, 1994, pp. 859-876. doi:10.1093/brain/117.4.859.
7. M. Hallett, "Overview of Human Tremor Physiology," *Movement Disorders*, Vol. 13, Suppl. 3, 1998, pp. 43-48. doi:10.1002/mds.870131308.
8. S. T. Shulman, "Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococci (PANDAS): Update," *Current Opinion in Pediatrics*, Vol. 21, No. 1, 2009, pp. 127-130. doi:10.1097/MOP.0b013e32831db2c4
9. S. Ashwal, B. S. Russman, P. A Blasco, G. Miller, A. Sandler, M. Shevell and R. Stevenson, Quality Standards Subcommittee of the American Academy of Neurology; Practice Committee of the Child Neurology Society, "Practice Parameter: Diagnostic Assessment of the child with cerebral Palsy: Report of the Quality Standards Sub-committee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society," *Neurology*, Vol. 62, No. 6, 2004, pp. 851-863.
10. Harrison's principle of internal medicine; 18th edition, ch.372, Table no. 372-1.
11. B. L. Schlaggar and J. W. Mink, "Movement Disorders in Children," *Pediatric Review*, Vol. 24, No. 2, 2003, pp. 39-51. doi:10.1542/pir.24-2-39.
12. T. M. O'Shea, "Diagnosis, Treatment, and Prevention of Cerebral Palsy," *Clinical Obstetrics and Gynecology*, Vol. 51, No. 4, 2008, pp. 816-828. doi:10.1097/GRF.0b013e3181870ba7.
13. A.H. Hoon Jr., E. M. Reinhardt, R. I. Kelley, S. N. Breiter, D. H. Morton, S. B. Naidu and M. V. Johnston, "Brain Magnetic Resonance Imaging in Suspected Extrapyramidal Cerebral Palsy: Observations in Distinguishing Genetic- Metabolic from Acquired Cause," *Journal of Pediatrics*, Vol. 131, No. 2, 1997, pp. 240-245. doi:10.1016/S0022-3476(97)70160-4.
14. M. V. Johnston and A. H. Hoon Jr., "Cerebral Palsy," *Neuromolecular Medicine*, Vol. 8, No. 4, 2006, pp. 435-450. doi:10.1385/NMM:8:4:435.
15. J. Menkes, "Heredodegenerative Diseases," In: J. H. Menkes, H. B. Sarnat and B. L. Maria, Eds., *Child Neurolog*, 7th Edition, Lippincott Williams & Wilkins, Phila-delphia, 2005, p. 163.
16. J. H. Menkes and W. R. Wilcox, "Inherited Metabolic Diseases of the Nervous System," In: J. H. Menkes, H. B. Sarnat and B. L. Maria, Eds., *Child Neurology*, 7th Edi., Lippincott Williams & Wilkins, Philadelphia, 2005, p.29.
17. A.S. Golden, S. R. Haut and S. L. Moshé, "Nonepileptic Uses of Antiepileptic Drugs in Children and Adolescents," *Pediatric Neurology*, Vol. 34, No. 6, 2006, pp. 421-432. doi:10.1016/j.pediatrneurol.2005.08.017.
18. F. J. Jiménez-Jiménez and P. J. García-Ruiz, "Pharma-cological Options for the Treatment of Tourette's Disorder," *Drugs*, Vol. 61, No. 15, 2001, pp. 2207-2220.
19. Weiner W.J. Lang A.E.: Movement disorders: a comprehensive survey. Mount Kisco NY: Futura, 1989.
20. Toplaglu H, Gucuyener K, Orkun C et al.: Tremor of tongue and dysarthria as the sole manifestation of Wilson's disease: Clin. Neurol. Neurosurgery 1990; 92: 295.

21. J. Jankovic and K. M. Shannon, "Movement Disorders," In: W. G. Bradley, R. B. Daroff, G. M. Fenichel and J. Jankovic, Eds., Neurology in Clinical Practice, 5th Edition, Butterworth Heinemann Elsevier, Philadelphia, 2008, p. 2081.
22. I.Korn-Lubetzki, A. Brand and I. Steiner, "Recurrence of Sydenham's chorea: Implications for Pathogenesis," *Archives of Neurology*, Vol. 61, No. 8, 2004, pp. 1261-1264. doi:10.1001/archneur.61.8.1261.
23. Centers for Disease Control and Prevention (CDC), "Pre-valence of diagnosed Tourette Syndrome in Persons Aged 6-17 Years United States, 2007," *Morbidity and Mortality Wkeekly Report*, Vol. 58, No. 21, 2008, pp. 581-585.
24. Rashmi Kumar et al: A diagnostic rule for tuberculous meningitis. Arch Dis Child 1999; 81: 221-224.
25. Lempert T, Dieterich M, Huppert D, Brandt T. Psychogenic disorders in neurology: Frequency and clinical spectrum. Acta Neurol Scand 1990; 82: 335-40.
26. Galvez-Jimenez N, Lang A. Psychogenic movement disorders. In Watt RL, Koller WC, eds. Movement disorders: Neurologic principles and practice, 1st ed. New York: McGraw-Hill 1997; 715-32.
27. Udani PM, Parekh UC, Dastur DK. Neurological and related syndromes of CNS tuberculosis, clinical features and pathogenesis. J. Neurol 1971; 14: 341-57.
28. J. Rice and M. C. Waugh, "Pilot Study on Trihexy-phenidyl in the Treatment of Dystonia in Children with Cerebral Palsy," *Journal of Child Neurology*, Vol. 24, No. 2, 2009, pg. 176-182.
29. D. S. Wolf and H. S. Singer, "Pediatric Movement Disorders: An Update," *Current Opinion in Neurology*, Vol. 21, No. 4, 2008, pg. 491-496.
30. D. Shprecher and R. Kurlan, "The Management of Tics," *Movement Disorders*, Vol. 24, No. 1, 2009, pp. 15-24.

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