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Pediatric Movement Disorders: A Comprehensive Review of Pathogenesis, Classification and Evidence Based Treatments

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ABSTRACT:

A movement disorder is characterized by abnormal involuntary movements, dysfunction in posture, performing normal-looking movements at inappropriate or unexpected periods, or malfunction in the aiming and velocity of intentional movements. Dystonia, chorea, athetosis, stereotypies, myoclonus, tics, and tremor are examples of hyperkinetic disorders, which are characterized by aberrant involuntary movements. Hypokinetic disorders, which include uncommon illnesses like parkinsonism, are characterized by a lack of movement. Low disease prevalence and genotypic and phenotypic heterogeneity have hindered our understanding of the disease mechanisms of rare movement disorders, such as hereditary spastic paraplegia, which is characterized by lower limb spasticity primarily due to axonal degeneration in the cortical tract. At least 18 additional putative genes were found to have mutations by whole-exome sequencing analysis, and subsequent network analysis revealed that several of these genes converge in important biological processes such as nucleotide metabolism, cellular transport, and axon and synapse development. Treatment options for movement disorders vary greatly depending on the specific type and classification of the disorder . This review indicates the treatment of movement disorders in paediatrics should be individualized ,taking in to account the underlying severity and patient specific needs. By synthesizing the existing evidence , this review provides a foundation for advancing our need for further research to optimize treatment and improve outcomes in paediatrics clinical practice .

***KEYWORDS** : Movement Disorder ,hypokinetic ,hyperkinetic ,mutation ,outcome*

INTRODUCTION

The occurrence of involuntary motions, decreased voluntary movement, or both are characteristics of movement disorders[1].Generally speaking, a movement disorder is characterized by abnormal involuntary movements, dysfunction in posture, performing normal-looking movements at inappropriate or unexpected periods, or malfunction in the aiming and velocity of intentional movements.[2] Hyperkinetic and hypokinetic disorders are the traditional classifications for movement disorders. Dystonia, chorea, athetosis, stereotypies, myoclonus, tics, and tremor are examples of hyperkinetic disorders, which are characterized by aberrant involuntary movements. Hypokinetic disorders, which include uncommon illnesses like parkinsonism, are characterized by a lack of movement.[3]

Dystonia and chorea are the two most common forms of movement disorders. There are no figures on the prevalence of movement disorders in children or their proportion among pediatric presentations, and it might be difficult to discern between many movement abnormalities that coexist in many instances. Although the precise pathophysiology of movement disorders is not fully known, evidence points to the involvement of either the cerebellar circuits, which encompass portions of the thalamus and cortex, or the basal ganglia in the majority of cases.[4] A heterogeneous clinical picture with both neurological and non-neurological characteristics is frequently present in PMDs.[5]Furthermore, there are an increasing number of potential aetiologies, particularly those related to genetic reasons[6]. Owing to a variable phenotype–genotype correlation, the diagnostic process for PMDs becomes challenging and time consuming, not only for the clinician but also for patients and caregivers.[7]

PATHOPHYSIOLOGY :

The etiology and clinical phenotype are diverse. Research in the field of movement disorders improved our knowledge of pathophysiology, translational aspects, and treatment in 2014.[8]Low disease prevalence and genotypic and phenotypic heterogeneity have hindered our understanding of the disease mechanisms of rare movement disorders, such as hereditary spastic paraplegia, which is characterized by lower limb spasticity primarily due to axonal degeneration in the cortical tract. At least 18 additional putative genes were found to have mutations by whole-exome sequencing analysis, and subsequent network analysis revealed that several of these genes converge in important biological processes such as nucleotide

metabolism, cellular transport, and axon and synapse development.[9]Furthermore, a noteworthy correlation between these genes and several neurodegenerative movement diseases was discovered.[10]

For the spread of pathology, another intriguing pathophysiological link across movement disorders was found. The first neurodegenerative movement condition for which allografted ventral mesencephalic tissue was demonstrated to be affected by a prion-like spread of pathology was Parkinson's disease. Since then, a number of in-vitro and in-vivo studies have demonstrated that neurons release and absorb α-synuclein. According to research conducted in 2014, exogenously produced α-synuclein fibrils or Lewy body extracts may cause α-synuclein to aggregate in neurons.[11]

CLASSIFICATION:

1. DYSTONIA

Muscle contractions that are either continuous or sporadic and result in aberrant, frequently repetitive postures, motions, or both are the hallmark of dystonia. Dystonic motions are usually twisting, patterned (using the same muscles repeatedly), and sometimes tremulous [12]. Exaggerated postures occur during voluntary movements and stressful situations, and they lessen as you sleep. If severe and ongoing, it could be painful. Muscle tone in the afflicted body areas is usually varied, ranging from low to high. Children also exhibit the striatal toe sign, which is the intermittent/persistent elongation of the great toe, and the splaying approach, which is the spreading of fingers as one approaches an item. Dystonic tremors, which are characterized by sporadic muscular spasms, can occasionally cause patients to exhibit oscillatory limb movement [13]

A. Isolated Dystonia

With the exception of tremors , isolated dystonia describes circumstances in which dystonia is the only motor characteristic[13] Within this category, DYT-TOR1 dystonia is the most prevalent entity. It is an autosomal dominant disorder with onset in late youth or preadolescence. Focal limb dystonia is the initial symptom, then generalized dystonia follows. DYT-HCPA dystonia begins with cervical and upper limb dystonia during the first ten years of life. Cranial or cervical dystonia (DYT-THAP and DYT-ANO3) can be the beginningl sign of other types of isolated dystonia.[14]

B. Combined Dystonia

This category comprises disorders when, in the absence of other neurological abnormalities, dystonia is accompanied by parkinsonian or myoclonus-like symptoms. Segawa illness, also known as dopa-responsive dystonia (DYT-GCH1), is a significant ailment in this group. It is an autosomal dominant condition that manifests as limb dystonia in children aged 5 to 10, with lower limb involvement being more severe. Small to moderate dosages of levodopa produce a full response, and dopa-responsive dystonia is a curable disorder. This serves as the foundation for any youngster with dystonia to undergo a levodopa study.Genetically diverse, myoclonus-dystonia can manifest as limb dystonia and upper body myoclonus at any point after infancy. When people with rapid onset dystonia-parkinsonism experience physical or mental stress, their symptoms worsen, and they frequently stammer. [15]

C. Acute-Onset Dystonia

Acute-onset dystonia may be a symptom of encephalitis, stroke, severe medication reactions, or functional disorders. Drug-induced dystonia is most frequently associated with anti-emetics (like metoclopramide) and antipsychotic medications (like haloperidol and risperidone). [16]

D. Paroxysmal Dystonia

Episodic involuntary motions that last anywhere from seconds to hours and typically have clear triggers are a symptom of some hereditary disorders. Typically, the youngster is typical in between bouts. This category comprises disorders such as paroxysmal exertional dystonia (caused by exercise), paroxysmal non-kinesigenic dystonia (caused by stress, alcohol, etc.), and paroxysmal kinesigenic dystonia (caused by abrupt movement). **[17]**

E. Status Dystonicus

Dystonic storm, also known as status dystonicus, is a potentially fatal illness marked by frequent or ongoing severe episodes of widespread dystonic spasms.[18] .Children with cerebral palsy and neurodegenerative diseases are most likely to experience it, while it can happen in any condition that causes dystonia. Triggers include infections (febrile diseases) and other stimuli. Acute renal failure, rhabdomyolysis, respiratory compromise, discomfort, and dehydration can all be consequences of severe spasm.[19]

Management

1 Anticholinergics

Trihexyphenidyl is the most often used anticholinergic drug, which is typically the most effective oral medication for treating dystonia[20].Trihexyphenidyl is an antagonist of the muscarinic acetylcholine receptor. There are significant variations in the therapeutic dose. It has been shown that children and adults respond well to average daily dosages of 41 mg and 24 mg, respectively[21]. Memory loss, disorientation, restlessness, sleeplessness, and nightmares are among the main adverse effects. Children who already have a tic disorder may develop chorea, or an exacerbation of it. Constipation, dry mouth, impaired eyesight, and urine retention are examples of peripheral side effects[22]

2 Baclofen

The primary purpose of the presynaptic GABA receptor agonist Baclofen is to alleviate spasticity. Its precise mode of action in treating dystonia is unknown.[23]Three to four divided daily dosages of baclofen should be taken. 5 mg per day is a common starting dose. Until side effects or advantages are realized, it is usually appropriate to increase by 5 mg/day every 3 to 5 days. Sedation, lightheadedness, dry mouth, and urgency or reluctance in urination are among the adverse effects of baclofen[24]

3 Benzodiazepines:

Despite the lack of large-scale, controlled research to assess their effectiveness, benzodiazepines are frequently used to treat dystonias. Most commonly, clonazepam is utilized. Clonazepam and other benzodiazepines were found to be helpful in 16% of patients with different kinds of dystonia in an open research. [20] A 0.5 mg evening dose of clonazepam is the usual initial dosage. The dosage is gradually raised to an average of 1–4 mg per day, divided t.i.d. Sedation, disorientation, poor coordination, and depression are among the adverse effects. [25]

4 Dopaminergics

Several early studies employed levodopa to treat generalized dystonia, although the outcomes were inconsistent. [26].Carbidopa, a decarboxylase inhibitor, is given in conjunction with levodopa. Constipation, orthostasis, and nausea are among the adverse effects. Carbidopa/levodopa 25/100 mg tablets are usually used once day at first, then increased by one tablet every three to five days. Though some may need doses as high as 1200 mg daily (20 mg/kg for children), the majority of children with DRD will react to doses as little as 25 to 100 mg levodopa.[27]

5 Dopamine receptor antagonists and dopamine depletion

One atypical antipsychotic that merits particular attention is clozapine. It is unknown how clozapine works in dystonia. [28]Until a benefit or adverse effect is experienced, the usual starting dose of clozapine is 12.5 mg daily, with increments of 12.5 to 25 mg weekly.[29] Patients with axial or widespread dystonia may need doses as high as 900 mg per day[30]By blocking the monoamine transporter 2 (VMAT-2), tetrabenazine (TBZ) reduces the amount of dopamine stored in vesicular storage. Numerous hyperkinetic movement disorders, such as chorea, tics, tardive dyskinesia, myoclonus, and dystonia, can be effectively treated with TBZ [31]

To reach a goal of 25–100 mg per day, it can be titrated up by 12.5 mg every 3–5 days from an initial 12.5 mg dose. Among the adverse effects are akathisia, depression, sleeplessness, parkinsonism, sleepiness, and anxiety.[32]

6.Botulinum toxin

Botulinum toxin has completely changed the way dystonia is treated since it was first introduced in the 1980s [33] .Weakness of the injected muscles or weakness of surrounding muscles as a result of local diffusion of the toxin are the main adverse effects. Depending on the injection site, spread can result in additional adverse effects, such as dysphagia following neck injections or ptosis or diplopia following eyelid injections. Systemic symptoms are rare.[34]

2 Tremors

A component of the body moving rhythmically and regularly back and forth or oscillating around a joint axis is referred to as tremor. [35] There are two types of tremors: activity tremors and resting tremors. Although they are uncommon in children, resting tremors can occur in midbrain lesions [36] Wilson disease, Huntington disease, PKAN, and juvenile parkinsonism. Infantile tremor syndrome (ITS) is a significant cause in emerging nations. Typically, tremors begin in the upper limbs and go away while you sleep. Nearly of individuals react to treatment and have vitamin B12 insufficiency[37]

Simple kinetic, intention, isometric, task-specific, and postural are other classifications for action tremors. Simple limb movements are followed by simple kinetic tremor. Usually, it is an aspect of essential tremor. Only the upper limbs are affected by essential tremor, and many individuals have a family history of the condition. Though it can occasionally happen in children, the onset usually happens in maturity or later. Simple kinetic tremors can also be caused by drugs or functional factors. When a bodily component approaches a visual target in intention tremor, the amplitude of the tremor increases. Although it can also be found in midbrain injuries, it is typically observed in cerebellar problems. When a bodily portion is held against gravity, postural tremor occurs. The best way to observe each person's physiological postural tremor is with an outstretched hand. Stress, hunger, illness, intense exercise, thyrotoxicosis, and medications like valproate and salbutamol can all make this worse. When muscles are repeatedly contracted against immobile objects, such as when holding a book, isometric dystonia develops. Essential tremor and exaggerated physiological tremor can be isometric. Writing and playing an instrument are two examples of tasks that are associated with task-specific dystonia**.**

MANAGEMENT:

Patients with physiological tremors who have functional or social impairments, as well as those with severe essential tremors, should take propanolol. Other medications that work well for essential tremors include benzodiazepines and primidone. [38]

3 CHOREA :

Involuntary, erratic, non-repetitive, dance-like bodily movements that seem to flow randomly from one muscle group to another are referred to as chorea[39]Primary chorea is chorea caused by a confirmed or suspected hereditary etiology. Secondary chorea are caused by infections, traumas, infiltrative diseases, or immune-mediated disorders that impact the brain.[40]

MANAGEMENT:

Acute onset chorea or acute aggravation can be effectively treated with atypical antipsychotics such as risperidone or olanzapine. Alternatives include tetrabenazine and anti-epileptic medications such as carbamazepine and valproic acid.[41] Patients who do not respond to anti-psychotic medications may try valproic acid. Intravenous immunoglobulin or corticosteroids may help patients with severe forms of Sydenham chorea or those who are not responding to antipsychotics.[42] Tetrabenazine, olanzapine, risperidone, and more recently, deutetranenazine are the recommended medications for people with Huntington's disease.[43]

4 TIC DISORDER :

Tics are defined as recurring, individually identifiable, sporadic motions, movement fragments, or sounds that are nearly always momentarily suppressible and are typically connected to an awareness of the want to carry out the movement. [35] .Based on their length and type (vocal or mechanical), tics are categorized. Simple tics consists of blinking of the eyes, head jerking, facial grimacing, short vocalization, clearing of the throat, and sniffing. Complex tics include copropraxia, posturing, echolalia, coprolalia, and obscene gestures. The symptoms of Tourette syndrome include at least two different motor tics and one or more waxing and waning vocal tics that have lasted for more than a year [44]

Management :

The first line of treatment for tics is comprehensive behavioural intervention for tics (CBIT), which is effective in reducing tics. To address circumstances that cause or exacerbate tics, the CBIT approach includes relaxation training, habit reversal therapy, and functional interventions. Pharmacotherapy is an option if CBIT is not accessible or is ineffective[45] Numerous medications work well, such as anti-psychotics like haloperidol, pimozide, and risperidone, alpha-agonists like clonidine and guanfacine, and anti-epileptics like topiramate. Despite being less effective than anti-psychotics, alpha-agonists are nonetheless chosen because of their favourable side effect profile. Additionally, behavioural comorbidities are improved by alpha-agonists.[46]

5 MYOCLONUS AND STARTLE SYNDROMES :

An abrupt, fleeting, shock-like involuntary movement of the body is called myoclonus. One body part, one half of the body, or the entire body may be affected. Although myoclonus is primarily spontaneous, there are situations in which an action or sensory inputs like touch, sound, or light can cause it. Sleep starts and hiccups are regarded as physiological manifestations of myoclonus. During sleep initiation, sleep begins, which is characterized by a feeling of falling. [47]

Exaggerated startle in reaction to sound, movement, or touch is a defining feature of startle syndromes. An autosomal dominant condition known as hereditary hyperekplexia causes excessive startles in newborns and young children, along with tonic stiffness of the body and frequent falls. Demonstrating non-habituating head retraction in response to repeated tapping of the nose tip is part of a bedside test. Although the intensity lessens with age, stress or exhaustion may cause it to flare up. The majority of kids react to clonazepam when given doses between 0.01 and 0.1 mg/kg per day.[48]

6 PARKINSONISM

Bradykinesia (slowness or paucity of movements), rigidity (lead pipe type), postural instability, and resting tremors are the hallmarks of Parkinsonism, a hypokinetic movement condition. It can happen as a side effect of tetrabenzine or in diseases like Huntington's and Wilson's. Leg dystonia and parkinsonism are symptoms of juvenile Parkinson disease, a rare genetic condition. [49]

9 FUNCTIONAL MOVEMENT DISORDERS

It describes involuntary motions that are inconsistent with known neurological and medical diseases and arise from abnormal mental states or situations. Common manifestations in children include myoclonus, dystonia, and tremors; other symptoms include tetany, tics, chorea, and abnormalities in gait.[50]. A history of abrupt start, significant symptom fluctuation, and prolonged spontaneous remissions all support the diagnosis. Incongruous motions, symptom variation, distraction during spontaneous speech and behavior, symptom emergence, or worsening during attentiveness and production or suppression of symptoms upon the examiner's suggestion are all common examination findings. Behaviour therapy and relaxation techniques are typically used for management. Counselling and education for parents are crucial.[51]

10. DEVELOPMENTAL AND BENIGN MOVEMENT DISORDERS

These are a collection of disorders that appear throughout particular stages of a child's development without any accompanying neurological symptoms. They have a positive consequence and are seen as a sign of subtle changes in the growing brain. [52]

CONCLUSION:

This systematic review highlights the overview of the current understanding of movement disorders in paediatrics ,encompassing pathophysiology ,clinical presentation, classification and treatment .This study aslo highlights the complex interplay of genetic , environmental and neurobiological factors contributing to the development of movement disorders in children .This review indicates the treatment of movement disorders in paediatrics should be individualized ,taking in to account the underlying severity and patient specific needs. By synthesizing the existing evidence , this review provides a foundation for advancing our need for further research to optimize treatment and improve outcomes in paediatrics clinical practice .

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