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Motor Tics A Case Report

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Abstract

The case involves a 10-year-old male child with a history of involuntary facial movements (tics) for the past 8 months. These tics, characterized by eye blinking and cheek stretching, are sudden and purposeless, aggravated by environmental factors, and relieved spontaneously. The intensity of symptoms varies throughout the day and is influenced by fatigue, stress, and anxiety. The child's medical history includes neonatal hyperbilirubinemia, upper respiratory tract infections, and dental treatments. There is no family history of similar conditions or mental disorders.

Physical examination reveals cervical lymphadenopathy, nasal congestion, tonsillar enlargement, and motor tics. Various referrals are made, including to an ear, nose, and throat specialist for adenoid hyperplasia and allergic rhinitis assessment, a psychiatrist for ADHD evaluation (predominantly hyperactive/impulsive subtype), and a neurologist for further investigation. The child is undergoing treatment for allergic rhinitis, along with occupational therapy, physiotherapy, and cognitive-behavioral therapy. The plan includes considering adenoidectomy if allergic rhinitis symptoms persist.

The case highlights a multidisciplinary approach to address the child's complex symptoms, encompassing medical, psychological, and therapeutic interventions to improve his overall quality of life. Close monitoring and adjustments to the treatment plan are essential for his ongoing well-being.

Keywords: Motor tics, attention deficit hyperactivity disorder (ADHD), Pediatric acute-onset neuropsychiatric syndrome (PANS), Tourette syndrome, Involuntary movements, comprehensive behavioural intervention for tics (CBIT)

Introduction

Tics are sudden, rapid, recurrent, nonrhythmic motor movements or vocalizations. Simple motor tics (e.g., eye blinking, neck jerking) are fast, brief movements involving one or a few muscle groups. Complex motor tics involve sequentially and/or simultaneously produced, relatively coordinated movements that can seem purposeful (e.g., brushing back one's hair bangs, tapping the foot, imitating someone else's movement [echopraxia], or making a sexual or obscene gesture [copropraxia]).

Pediatric acute-onset neuropsychiatric syndrome (PANS) features a heterogeneous constellation of acute obsessive-compulsive disorder (OCD), eating restriction, cognitive, behavioral and/or affective symptoms, often followed by a chronic course with cognitive deterioration. An immune-mediated etiology is advocated in which the CNS is hit by different pathogen-driven (auto)immune responses. Pediatric acute-onset neuropsychiatric syndrome (PANS) is a newly described clinical entity with heterogeneous symptoms presentation, characterized by the acute or subacute onset of obsessive-

compulsive disorder (OCD) and/or a severe food intake restriction, associated with at least two cognitive, behavioral, or affective symptoms such as irritability, anxiety or depression

Case Report

A 10-year-old male child presented to the developmental Clinic, at a tertiary health centre with complaint of involuntary movements of the face since 8 months. Patient was apparently alright 8 months ago when he started to develop involuntary movement of the face that is blinking of the eyes along with stretching of the cheeks. The onset was insidious and progressive in terms of frequency. The involuntary movements were sudden, purposeless and aggravated by dust, cold or anxious situations and relieved on its own. The involuntary movements increased in the evening however were absent during sleep and the intensity and persistence of the symptoms waxed and waned spontaneously but he has never been completely symptom-free since his condition started. Symptoms tended to be less severe when engaged in physical activity but they would exacerbate when he was fatigued, stressed, or frightened. Over the past month, his motor tics showed gradual spontaneous improvement. His mother reported that prior to the development of tics; he showed hyperactivity and aggressive behaviour towards his family members and that he would be embarrassed at the school which had hampered his performance in school activities and academics.

Upon reviewing his perinatal and developmental histories he was the second born to a non-consanguineous marriage with no antenatal complications. He was a post term born via LSCS (in view of oligohydramnios, cried immediately after birth and was Appropriate for gestational age. He was history of neonatal hyperbilirubinemia for which phototherapy was given for 2 days. He was breastfed for 1 month after which he was fed cowmilk and there is history of bottle feeding present. Achieved the normal developmental milestones as per age.

He was fully immunized to schedule.

Past history of root canal treatment 4 years ago 2019 and h/o hospitalisation in the same year for high grade fever for 4 days. h/o repeated upper respiratory tract infection since 3 years every 2-3 months associated with intermittent headaches, blocked nose /nasal discharge and snoring while sleeping. and h/o nebulisation once at 9 months of age. He was an average student at school with no learning disabilities. There was no history of obsessiveness, compulsiveness, second-hand smoke exposure, or substance abuse.

He has one sister who is completely normal and there were no similar conditions in the family and no family history of other mental disorders.

On examination; the patient had fair general condition, temperature was 37ºC, heart rate was 94/min while RR was 26/min. His weight, height, and head circumference were on the 50th percentile for age. On examination was no pallor, cyanosis/jaundice/oedema, skin rashes. He Right cervical anterior lymphadenopathy was present which was single lymph node measuring 0.5 x 1cm, non-tender, not fixed. On examination of the nose, it was pinched nose in appearance, nasal congestion+, left sided DNS +, B/L inferior turbinate hypertrophy +. Throat on examination had granular pharyngitis and B/L grade 1 tonsillar enlargement. Oral cavity on examination had crowding of teeth, caries +, Steel cap + on left 1st premolar. Chest- scar was present from burn. As for neurological examination; the right-handed boy was fully conscious, alert, oriented to person, time, and place with intact thought processes and higher mental functioning. His motor tics at the time of examination were in the form of eye blinking and cheek stretching bilaterally. His local neurological examination showed normal tone, power, and reflexes, his cranial nerves examination revealed no abnormality. There were no wasting or deformities. Physical examination revealed no other abnormalities. Chest and heart examination revealed nothing significant with normal heart sounds, bilateral equal intensity of breath sounds, and no adventitious sounds or murmurs. Abdomen was clinically normal. Laboratory investigations showed a normal ESR, CRP, ASOT, CBC, normal renal functions, serum electrolytes, albumin. His liver functions were normal including ALT, AST, and total and direct bilirubin.

ENT reference was done for allergic rhinitis and grade 1 tonsillar enlargement. for which they advised xray nasopharynx which was suggestive of adenoid enlargement. They advised for psychiatric and

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neurology reference and treatment was started for allergic rhinitis. MRI BRAIN AND PNS was advised

Psychiatric reference was done for which they had advised IQ testing, venderbilt scoring for ADHD, and clozapine and risperidone was advised. It was documented that as per Vanderbilt scoring it he was coming under Predominately Hyperactive/Impulsive Subtype.

Neurology reference was done and RBS monitoring was advised to rule out hypoglycaemia associated history of headache and MRI Para nasal sinuses was advised. Imaging studies were carried out including MRI of the para nasal sinuses was done to rule out probable compression/ irritation of the facial never that could be possible cause for the involuntary movements. However, it was suggestive of T1w hypointense, T2W and STIR hyperintense polypoidal mucosal thickening noted involving bilateral frontal, maxillary, ethmoid and sphenoid sinuses. No restricted diffusion noted on diffusion weighted images. Bilateral osteomeatal units blocked. Right inferior turbinate hypertrophy seen. There is deviated nasal septum towards left with septal spur. A 2.7 x 1.8 cm sized well defined area of altered signal intensity is noted involving the superior wall of nasopharynx. It causing moderate obstruction to nasopharynx. Findings are most likely suggestive of adenoid hyperplasia. The visual extent of brain was normal. The sella and parasellar area appeared normal

The child was discharged with treatment for allergic rhinitis and occupational therapy and physiotherapy and cognitive behavioural therapy.

Plan of action- to perform adenoidectomy if his symptoms do not resolve for allergic rhinitis.

Discussion

Onset of tics is typically between ages 4 and 6 yr. The frequency of tics tends to wax and wane with peak tic severity between ages 10 and 12 yr and marked attenuation of tic severity in most individuals (65%) by age 18-20 yr. A small percentage will have worsening tics into adulthood. New onset of tics in adulthood is very rare and most often is associated with exposure to drugs or insults to the central nervous system. Tics manifest similarly in all age-groups and changes in affected muscle groups and vocalizations occur over time. Some individuals may have tic-free periods of weeks to months.

Prevalence rates for all tics range from 6–18% for boys and 3–11% for girls, with the rate of TD alone estimated as 0.8%. In general, PTD/TD has a male preponderance with a gender ratio varying from 2 : 1 to 4 : 1. Evidence supports higher rates in white youth than black or Hispanic youth.

Tics may be difficult to differentiate from stereotypies. Although stereotypies may resemble tics, stereotypies are typically rhythmic movements and do not demonstrate the change in body location or movement type over time that is typical of tics. Compulsions may be difficult to differentiate from tics when tics have premonitory urges. Tics should be differentiated from a variety of developmental and

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benign movement disorders (e.g., benign paroxysmal torticollis, Sandifer syndrome, benign jitteriness of newborns, shuddering attacks). Tics may present in various neurologic illnesses (e.g., Wilson disease, neuroacanthocytosis, Huntington syndrome, various frontal-subcortical brain lesions), but it is rare for tics to be the only manifestation of these disorders. Individuals presenting with tics in the context of declining motor or cognitive function should be referred for neurologic assessment. Substances/medications that are reported to worsen tics include selective serotonin reuptake inhibitors (SSRIs), lamotrigine, and cocaine. If tics develop in close temporal relationship to the use of a substance or medication and then remit when use of the substance is discontinued, a causal relationship is possible. Although a long-standing clinical concern, controlled studies show no evidence that stimulants commonly increase tics

Provisional Tic Disorder

A. Single or multiple motor and/or vocal tics.

B. The tics have been present for <1 yr since first tic onset.

C. Onset is before age 18 yr.

D. The disturbance is not attributable to the physiologic effects of a substance (e.g., cocaine) or another medical condition (e.g., Huntington disease, post viral encephalitis).

E. Criteria have never been met for Tourette disorder or persistent (chronic) motor or vocal tic disorder

Tics are proposed to be the result of dysfunctional corticostriatal-thalamocortical motor pathways in the basal ganglia, striatum, and frontal lobes associated with abnormalities in the dopamine, serotonin, and norepinephrine neurotransmitter systems. Male predominance in PTD/TD may be attributable to influences of sex hormones on the neurodevelopment of these motor pathways. Candidate-gene association and nonparametric linkage studies have not identified specific susceptibility genes for PTD/TD. Autoimmune-mediated mechanisms have been hypothesized as having a potential etiologic role in some tic disorders. The pediatric autoimmune neuropsychiatric disorder associated with streptococcal infection (PANDAS) designation has been used to describe cases of acute childhood onset of OCD and/or tics following a streptococcal infection. Pediatric acute-onset neuropsychiatric syndrome (PANS) has been used to describe a subtype of acute childhood-onset OCD (tics are not a required feature) in which a link to a prior streptococcal infection is not evident, suggesting that other infectious agents may also be responsible. In addition to a diagnosis of OCD and tics, children with PANS/PANDAS have been reported with symptoms of separation anxiety, nightmares, personality change, oppositional behaviours, and deterioration in math skills and handwriting. Although some studies suggest a prior history of infections may increase the risk for developing tic disorder, this remains controversial.

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