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A Case Report of Cerebellar Atrophy Presenting as Specific Learning Disorder and Developmental Coordination Disorder

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Abstract

Ms. S, an 11-year old female child referred from school for poor academic performance in the form of poor hand writing, spelling errors, and difficulty in mathematical problems. History revealed that she was a preterm delivery with no significant delay in developmental mile stones expect for delay in gross motor and fine motor skills. On psychological examination her IQ was 85, NIMHANS learning disorder index showed significant difficulty in reading and writing, and arithmetic problems leading to a diagnosis of specific learning disorder with developmental coordination disorder. Neurological evaluation showed the presence of cerebellar signs for which she was referred to a neurologist, and found to have bilateral cerebellar atrophy in MRI of brain. This case is reported for the rarity of presentation of cerebellar atrophy as specific learning disorder and developmental coordination disorder.

Keywords

Specific learning disorder, developmental coordination disorder, cerebellum**.**

Introduction

Specific learning disorder means a disorder in one or more of the basic psychological processes involved in understanding or in using language, spoken or written, which disorder may manifest itself in the imperfect ability to listen, think, speak, read, write, spell, or to do mathematical calculations. This disorder includes conditions such as perceptual disabilities, brain injury, minimal brain dysfunction, dyslexia, and developmental aphasia. Life time prevalence of learning disorder is 9.7 percent. Reading disorder is the most common and well-recognized of the subtypes of learning disorders [(8).]

The prevalence of developmental coordination disorder has been estimated at about 5 percent of school-age children. Children exhibiting developmental coordination disorder fail to acquire adequate age-appropriate motor skills, leading to impairment in gross motor skills such as jumping, hopping, and running; fine motor skills, including hand writing; tying their shoelaces; using utensils; and balance.

Motor coordination disorder often occurs in conjunction with attention deficit/hyperactivity disorder, communication disorder, and learning disorders [(8).]

The cerebellum is known to be involved in the co-ordination of movement, in cognitive processes and in automatization of activities such as typing, driving, reading. A weak capacity to automatize would, for example, impair learning and fluency in grapheme – phoneme matching. Cerebellar abnormalities may underlie some of the cognitive deficits found in individuals born very preterm. Children with dyslexia showed specific deficit of implicit learning, but not in the declarative task, suggesting that implicit learning is a cognitive function primarily processed by the cerebellum. Children with developmental coordination disorder/ dyspraxia are at high risk of reading and writing delay [(8)].

Case Report of Cerebellar atrophy presenting as specific learning disorder and developmental coordination disorder

Ms. S, an 11-year-old female child, (Fig 1) studying in the 6th standard was referred from school to the department of Child Psychiatry, Institute of Child health, Madras Medical College, Chennai, with complaints of poor scholastic performance in the form of poor hand writing, spelling errors while writing, and difficulty in doing mathematical problems.

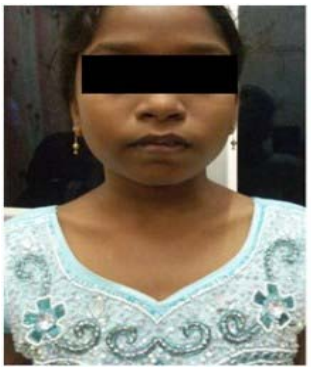

Fig. 1

Past history revealed that Ms. S had difficulty in holding objects, and clumsiness in performing activities of daily living. Ms. S was the second child to her mother and born out of nonconsanguineous marriage. Her mother's antenatal period was uneventful, delivery of the child was by caesarian section and the indication was oligohydramnios. The child's birth weight was 2.5 Kgs. She was kept in NICU after the birth for 3 days for preterm birth. Her developmental mile stones namely, motor, cognitive, language and social development were normal. Her mother informed that Ms. S had poor coordination in her gross motor skills, like running and jumping. She was poor in sports activities because of the clumsiness of hands and difficulties in holding objects and all her activities of daily living like bathing, cleaning and dressing used to be assisted by her mother. There was no family history of any neurodevelopmental disorders, learning disorders or seizures. Child was temperamentally an easy child.

Physical examination was normal. Central nervous system examination showed hypotonia, finger nose incoordination, difficulty in tandem walking and abnormal gait. On mental status examination, the child was found to be left handed and held her pen in an unusual position. Her speech showed dysfluency. Learning and writing test was conducted to assess her ability. She was slow in reading words and had difficulties to read complex words. In writing test, she did some grammatical and spelling errors (Fig 2). She had done some mistakes in graded multiplication and division while doing arithmetic problems (Fig 2). Child did not have any thought or perception disturbances.

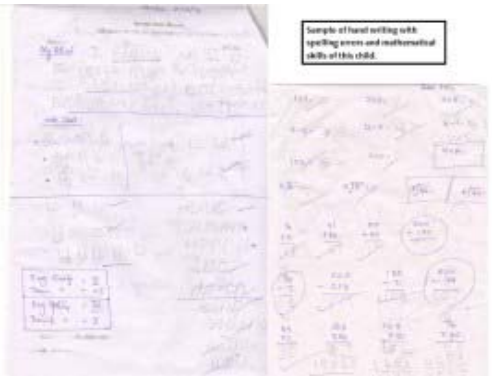

Fig. 2

Psychological assessment test revealed.

Table: 1 The Malins intelligence scale for Indian children

Verbal IQ	84
Performance IQ	87
Full IQ	85

Table: 2 The NIMHANS index for Specific learning disability: (Level II)

Performance Skills			
English		Tamil	
Reading	Class 4		Class 1
Comprehension	Class 3		Less than Class 1
Writing	Class 2		Less than Class 1
Spelling	Class 3		Class 1
Arithmetic		Class 3	
Perceptual visual motor skills		Average	
Visual memory		Average	
Auditory memory		Mild difficulty	
Attention		Class 3	

Based on the history, examination and psychological assessment she was provisionally diagnosed to have specific learning disorder with developmental coordination disorder. Due to the presence of cerebellar signs she was referred to a neurologist, MRI brain (Fig 3) showed diffuse cerebellar atrophy with gliosis in bilateral cerebellar white matter and her EEG was normal.

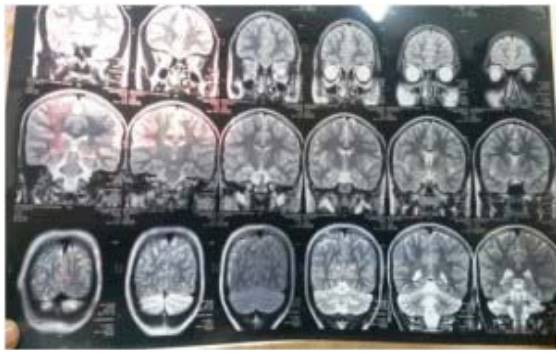

Fig. 3

Laboratory workup showed serum CPK = 145 U/L, serum lactate = 31mg/dl, serum pyruvate = 0.5mg/dl, serum ammonia = 42 µg /dl, all were within normal limits. Urine metabolic screening was negative. Tandem mass spectroscopy, which is a screening tool for inborn error of metabolism, was done which was negative. ENT opinion obtained, bilateral hearing was normal. Speech therapist opinion obtained, diagnosed to have ataxic speech. Ophthalmologist opinion showed no K-F ring and cardiac evaluation revealed normal echocardiography findings. The child was treated by the multidisciplinary team involving a child psychiatrist, neurologist, speech therapist and physiotherapist. The need for the long term care and follow up of the children was explained to her parents.

Discussion

According to the literature, it has been traditionally recognized that the cerebellum plays an important role in fine motor coordination. In recent years, it has been learned that cerebellum is also activated during learning and may participate in the automatization of learned task, including reading [14]. Motor development and cognitive development may be fundamentally interrelated. Contrary to popular notions that motor development begins and ends early, whereas cognitive development begins and ends later, both motor and cognitive development display equally protracted developmental timetables. When cognitive development is perturbed, as in a neurodevelopmental disorder, motor development is often adversely affected. The striatum functions as part of a circuit with dorsolateral prefrontal cortex, the same is true for the cerebellum and that the cerebellum may be important for cognitive as well as motor functions. Like prefrontal cortex, the cerebellum reaches maturity late. Many cognitive tasks that require prefrontal cortex also require the cerebellum [1]. Anatomically (in the macaque monkey), the lateral prefrontal cortex, superior temporal sulcus, and post parietal association cortices project to the cerebellum via pontine nuclei (10, 18) whereas the cerebellum projects to the association cortex via the thalamus [4]. The cerebellum receives input from various magnocellular systems in the brain and therefore can be affected by a general magnocellular deficit [19].

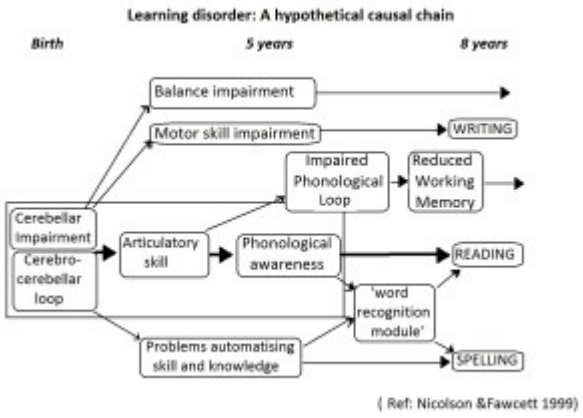

Fig. 4

The cerebellar deficit hypothesis (Fig 4) for the reading disorder attributes the cognitive and motor problems exhibited by dyslexics to abnormal cerebellar development [13, 15]. A cerebellar abnormality can be a cause or a contributing cause of a learning disability in a number of ways.

It can cause a learning disorder through a failure to acquire and automatize reading and writing skills. According to this theory, skill automatization, speed, and fluency of information are areas of independent deficits in dyslexia. It is similar to the suggestion that slow speed of information processing contributes to reading impairment independent of other factors, especially phonological deficits. Another way that cerebellar abnormality can interfere with normal reading and learning is through cerebellar abnormality causing procedural memory deficit or impairing motor control of speech articulation and other motor tasks, including handwriting.

Problems with the rate of reading words, suggesting cerebellar involvement were found in adults and children with dyslexia [6]. Some children with right cerebellar tumor had poor verbal and literacy performance where as children with left hemispherical tumor had spatial deficit. Brain imaging studies have shown anatomical, metabolic, and activation differences in the cerebellum of dyslexics [16, 14, & 9]. Also, the deficit may be in the cerebellum because of a time estimate defect in dyslexia [5, 16, &14].

The critics of this cerebellar theory point to the fact that cerebellar signs are not always seen in learning disorder, and also to the fact that procedural learning is not restricted to the cerebellum. The architecture of the memory system sub serving procedural learning includes cortical, subcortical, and cerebellar connections. Therefore, deficit of procedural learning can result from dysfunction of an extensive neural architecture and are not necessarily caused by a cerebellar abnormality [3]. In addition, phonological representation does not depend on speech development and will not be affected by dysarthria [17].

The neurological symptoms found in children with minimal brain dysfunction or deficits in attention, motor control and perception, seem to suggest a dysfunctioning of certain brain areas, particularly the cerebellum and the basal ganglia [11]. Studies found that children with developmental coordination disorder have cerebellar dysfunction [2]. Jissendi-Tchofo et al (2011) reported 10 cases of children affected with isolated cerebellar cortical dysplasia who presented with different cognitive and socioadaptive developmental disorders on the basis of neuroimaging findings [20].

Two types of dysgraphia may be distinguished: the core ones, which reflect damage to the linguistic orthographic routes and the peripheral ones, produced by alterations in the selection or execution of graphic motor patterns. Maria Concepcion Fournier Castillo (2010) reported a case of an 8-year-old male child, who consulted specialists due to difficulties in writing, with a background of acute cerebellar swelling at the age of 4. His writing pattern showed characteristic errors of a peripheral dysgraphia. The magnetic resonance imaging of the boy taken during the neuropsychological evaluation showed a mild atrophy in the cerebellum cortex [12].

Studies of neural correlates of pediatric dysgraphia by Jessika F Van Hoorn et al., (2013) showed that deficits in handwriting performance are frequently encountered in children with developmental coordination disorder. The two functional imaging studies suggest a contribution of cortical areas and the cerebellum. In patients with reading disorder, cerebellar dysfunction gives rise to a range of motor and cognitive problems associated with reading. For example, the cerebellum is known to play a role in motor control and therefore in speech articulation, which could lead to deficient phonological representations. In addition, the cerebellum plays a critical role in specific timing required for some aspects of speech perception [7].

Brain imaging studies have revealed anatomical, metabolic, and activation differences in the cerebellum of individuals with reading disorder [17]. Impairments in balance and time perceptio–a non-motor cerebellar task and poor performance on a range of cerebellar motor tasks have been reported in studies of individuals with learning disorder [7].

Conclusion

Children presenting with learning disabilities may have lesion in the cerebellum. Learning disabilities and developmental coordination disorder often impact individuals' quality of life, as a result of their continuous difficulties both academically and socially. The provision of suitable and well-time intervention is essential to encourage maximum performance, prevent secondary difficulties and limit associated health-care costs. This case is reported for its presentation as learning disorder and developmental coordination disorder caused by cerebellar atrophy.

Accurate assessment and intervention will enable children, adolescents and adults who are diagnosed with learning disorder and developmental coordination disorder to realize their maximum potential and function as fulfilled members of society in all realms. Future research might throw more light on the involvement of cerebellum in the process of learning.

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