Case Report

Pediatric multiple sclerosis – rare case of multiple sclerosis in an adolescent boy

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ABSTRACT

Multiple sclerosis (MS) is chronic inflammatory, demyelinating neurodegenerative, and autoimmune disorder of the central nervous system of unknown etiology. The peak onset is between age 20 and 40 years and it usually affects more women than men. Although much knowledge has been achieved on the diagnosis and treatment of adult patients with MS, it remains a matter of debate and controversy in childhood. We present a case of MS in 12-year-old-boy, review the current state of the knowledge on pediatric MS and discuss the available tools for the diagnosis and treatment.

Key words: Cerebro spinal fluid, Disease modifying therapy, Magnetic resonance imaging, Multiple sclerosis

Multiple sclerosis (MS) is described as an inflammatory, demyelinating, and neurodegenerative disorder of the central nervous system and is uncommonly seen in pediatric patients [1-4]. Pediatric MS also referred to as pediatric onset MS, is defined as MS with an onset before 16 years of age. Pediatric MS represents about 2.2–4.4% of all MS cases. In late childhood, affects more girls than boys, and is characterized by a relapsing-remitting course in almost all cases [5-8,9].

Pediatric MS has distinctive features and the disease course is different from adults. Children are less likely to develop primary or secondary progressive MS in childhood. Guidelines for pediatric MS recommended that treatment can be started early in the disease course. Disease modifying therapies can also be used in treatment for Pediatric MS. This report describes the case of 12 years and 11-month-old boy with MS and discusses the current knowledge for the diagnosis of MS and therapeutic possibilities.

CASE REPORT

Our patient was 12 years and 11 months old male boy, first born to non-consanguineous marriage, who presented with a history of multiple neurological events. When he was 6 years old, he suddenly developed acute painless diminished vision and was unable to read the question sheet. There was no history of redness of eyes, increased lacrimation, or watering of eyes. There was no history of headache, vomiting, seizures, or altered sensorium. There was no history of preceding fever, cough, cold, vomiting, or loose stools before the onset of illness. On examination, vitals were stable and no focal neurological deficit was found. Ophthalmic and other systemic examination were also normal. There was a history of the headache on and off since 5 years of age which was holocranial, non-throbbing, not associated with vomiting, aura, photophobia, or phonophobia, lasting around 10 min occurring at least once a week, mostly while studying.

There was a history of myopia corrected with refractory lenses. There was no history of hearing disturbances and deviation of angle of mouth, difficulty in chewing or swallowing, nasal twang or nasal regurgitation. In familial history, paternal grandmother had stroke in 2012. Child was admitted and given symptomatic treatment and was on regular follow-up.

The child was asymptomatic till 4 years, and then he gradually developed weakness of the upper and lower limbs with difficulty in walking and in raising the arms above the head. There was difficulty in grasping objects with the left hand. There is a history of slurring of speech and change in handwriting over the next 4 days. There was no history of headache, blurring of vision, vomiting, seizure, or altered sensorium during the event. On examination, vitals were stable, the higher mental function was normal. On cranial nerve examination, there was difficulty in blowing mouth and whistling. On sensory examination, there was a loss of pain sensation in both upper limbs. Power was found to be 4/5 in all limbs.

Cerebrospinal fluid analysis during the hospital stay revealed no cells, protein of 7 mg/dl and oligoclonal bands were detected in cerebro spinal fluid (CSF). CSF anti-aquaporin antibodies were negative. Magnetic resonance imaging (MRI) was done which showed Dawson's fingers (Fig. 1) and hyper-intensities

Access this article online	
Received - 18 January 2021 Initial Review - 14 February 2021 Accepted - 26 March 2021	Quick Response code
DOI: 10.32677/IJCH.2021.v08.i04.008	

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in deep white matter of B/L cerebral parenchyma including corpus callosum in T2 flair image. Some of them also showed post-contrast enhancement (Fig. 2). The child was admitted for the duration of 1 week and was treated with pulse methyl prednisolone for 5 days followed by oral steroids for 8–10 days. There was complete recovery in the limb weakness by 3rd day after initiation of treatment.

The child had been evaluated for the above episodes at referral hospital when he was found to be positive for myelin oligodendrocyte glycoprotein antibody and was treated with a tapering schedule of oral steroids for 3 months and escalating schedule of mycophenolate.

The child again developed gradual onset of weakness of the left-sided upper and lower limbs with occasional falls while walking 1 year after this treatment. There was a history of difficulty in grasping objects with the left hand and slipping of chappals from the left foot and slurring of speech and change in handwriting which worsened over the next 3 days. Mild blurring of vision in the left eye was also reported during this event. There is no history of fever, headache, altered sensorium, or seizures. On examination, vitals were stable, higher mental function was normal. 3rd, 7th, and 10th nerves were found to be affected. There was a loss of pain and temperature sensation in the left upper and lower limbs. Power was found to be 3/5 in the left upper and lower limbs.

Brain MRI revealed confluent areas of T2W hypersensitivity in periventricular white matter and deep white matter of the frontal and parietal lobes (Fig. 3). Left cerebellar white matter and left middle cerebellar peduncle also showed mild expansion and T2W hypersensitivity. Nerve conduction studies and B brainstem auditory evoked response were normal. The visual evoked potential examination revealed left anterior optic pathway dysfunction. CSF analysis revealed nil cells, sugar-60 mg/dl, and protein-27 mg/dl. The polymerase chain reaction tests for multiple viruses were negative. NMOSD screen had shown positivity for MOG antibody during the last visit.

During the ward stay, the child was initiated on pulse methylprednisolone (1 g/day for 5 days) and first dose of Rituximab was given. Physiotherapy was also initiated for the limb weakness and child get relieved from the symptoms. Since then, the child is on regular follow-up and taking subsequent doses of Rituximab monthly. No further complaints were documented till date.

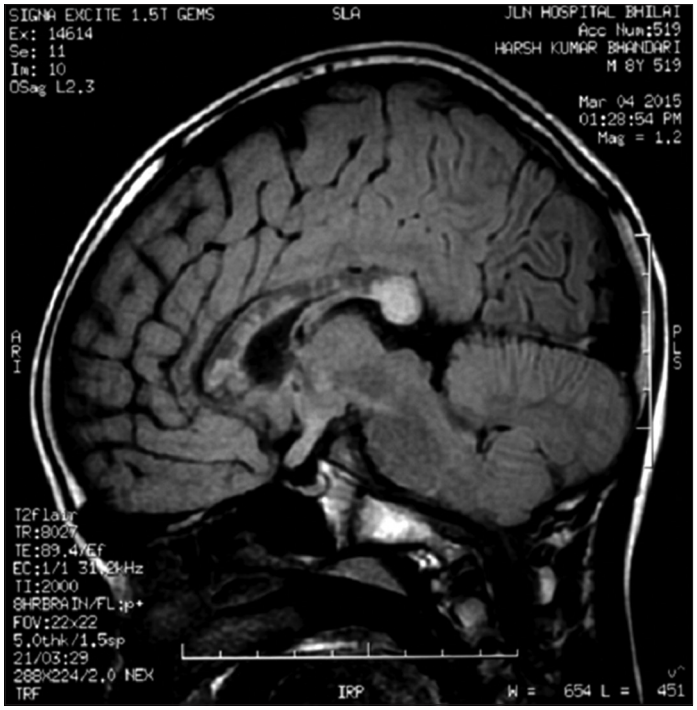

Figure 1: Brain magnetic resonance imaging showing Dawson fingers

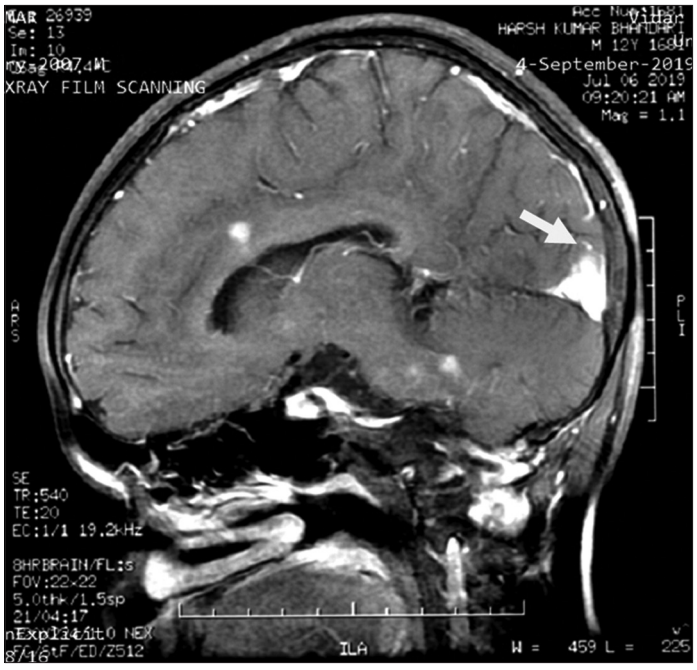

Figure 2: Brain magnetic resonance imaging showing post-contrast enhancement of lesions

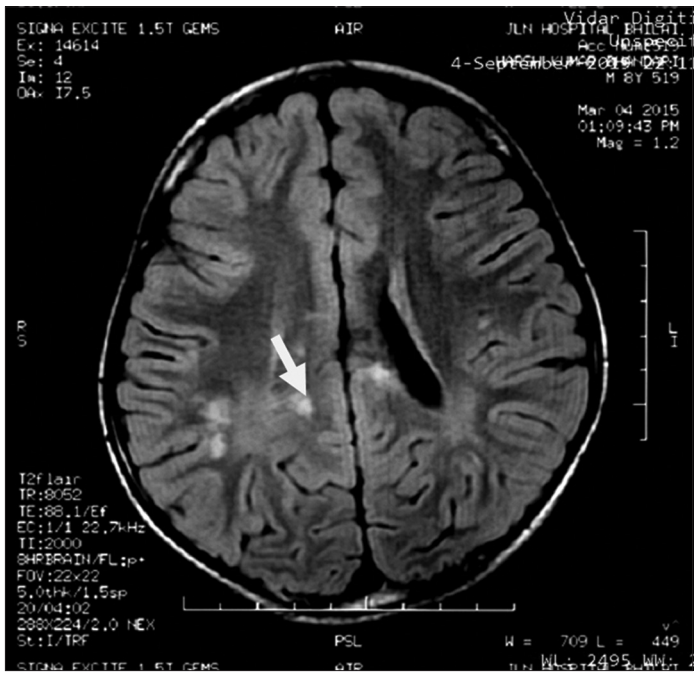

Figure 3: Brain magnetic resonance imaging showing periventricular lesions

DISCUSSION

MS is characterized as an inflammatory autoimmune disorder of the CNS where the fatty myelin sheaths around the axons of the brain and spinal cord are damaged, leading to demyelination and scarring [10-13]. Jean-Martin Charcot, a French researcher, described it for the first time in 1868 [14]. In 2007, Swanton *et al*. [15] and Montalban *et al*. in 2010 [16] have made important contribution on the diagnosis of MS by simpler criteria like MRI evidence for dissemination in space and time to be used in patients who present with clinically isolated syndrome. International panel on the diagnosis of MS affirmed the new criteria, it will also serve well for most pediatric MS patients, especially those with acute demyelination presenting as CIS [17].

Currently available first-line diseases modifying therapies to MS for adults, including interferon-beta and glatiramer acetate, have not been approved by the Food and drug association for the treatment of children with MS [18]. However, it has been proved to be safe and well tolerated in pediatric population [19-21].

In children with relapses, escalation to higher efficacious second-line therapies, such as Natalizumab, rituximab, fingolimod, cyclophosphamide, and daclizumab may be considered based on extrapolated data from adult cohorts. However, the data on safety, efficacy, and tolerability of most of these drug treatments are scarce and has been reported only in small size retrospective case series.

CONCLUSION

Since MS is rarer in pediatric age group that too in an adolescent boy, further study is needed to be done about the etiology and presentation of MS. Pediatric MS has long been an under diagnosed and undertreated condition. It is important to limit the axonal damage secondary to excessive inflammatory changes seen earlier in disease process by initiating early disease modifying therapy (DMT) in these patients. More prospective, randomized, large cohort studies are needed to assess the safety and efficacy of DMT in children with MS.

ACKNOWLEDGMENT

We would like to thank our department for making this manuscript.

AUTHOR CONTRIBUTION

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Funding: None; Conflicts of Interest: None Stated.

How to cite this article: Punj P, Baby EP, Jadon APS, Panda S, Biswal PK. Pediatric multiple sclerosis – rare case of multiple sclerosis in an adolescent boy. Indian J Child Health. 2021; 8(4):172-174.