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Early Recovery in Post *Varicella* Transverse Myelitis

Prem Chand¹, Shahnaz Ibrahim¹, Syed Sohail Zaidi² and Nida Amjad¹

ABSTRACT

A 7 years old boy presenting with acute flaccid paralysis after *Varicella zoster* infection was diagnosed as having acute transverse myelitis on MRI. He recovered fully after treatment with intravenous corticosteroids and acyclovir. The occurrence of this condition during or following *Varicella* infection is uncommon. There are previously very few reported cases of post-*Varicella* acute transverse myelitis in which recovery started after 3 months of treatment. In this case complete recovery occurred in 2 weeks of treatment. This report emphasizes the need for *Varicella zoster* vaccine to prevent not only acute *Varicella*, but also its rare postinfectious neurologic sequelae.

Key Words: Acute flaccid paralysis. *Varicella zoster*. Transverse myelitis. Recovery. Child.

INTRODUCTION

Varicella zoster virus (VZV) infection causes a variety of neurologic complications, including aseptic meningitis, meningoencephalitis, neuralgia, polyradiculoneuritis, transverse myelitis, vasculopathy, leukoencephalopathy, cranial neuropathies and ventriculitis.¹ Neurologic complications occur in approximately 2 per 10,000 cases of *Varicella*.¹ The frequency of transverse myelitis during or after *Varicella* infection is 0.3%.² The characteristic symptoms are typically bilateral sensory deficit at a given level, paraparesis, quadriparesis, motor weakness and abnormal bladder and rectal function.² No diagnostic test is completely accurate, since VZV cannot usually be isolated from blood or cerebrospinal fluid (CSF) in VZV myelitis.²

This report describes early recovery in a child with post-*Varicella* transverse myelitis.

CASE REPORT

A 7-year-old boy presented with sudden onset of weakness of lower limbs, inability to walk and urinary incontinence for 3 days. He had a history of high grade fever 14 days back, followed by appearance of a vesicular rash (chicken pox) on the next day which was treated conservatively (Figure 1). His general physical examination showed scabs of healed chicken pox lesions. Neurological examination showed normal higher mental functions, no signs of meningeal irritation, normal cranial nerves and no evidence of cerebellar involvement. Motor examination revealed normal tone

and power of (5/5) in both the upper limbs. In lower limbs tone was reduced, with power of 2/5 in both lower limbs. Deep tendon reflexes were normal in both upper limbs and absent in both lower limbs. Planters were down going bilaterally. Sensory examination was difficult, as the child was irritable and would not co-operate. His abdominal and cremastic reflexes were absent. Investigations included electrolytes, complete blood count, and urine analysis which were within normal limits. CSF analysis showed slightly raised leukocyte count of 12 cells/mm³, with 90% lymphocytes, proteins were 4 mg/dl and CSF sugar was 116 mg/dl with blood sugar of 125 mg/dl. CSF was positive for anti-VZV antibodies (IgM), however, the PCR was negative for virus detection both in blood and CSF. CSF oligoclonal bands were absent. Stool culture was negative for polio-virus. MRI spine showed hyperintense signal on T2 weighted image that diffusely involved the spinal cord. These were more marked in cervical and lower thoracic region and was associated with swelling of cord. No contrast enhancement was seen (Figure 2).

A diagnosis of acute transverse myelitis (ATM) following chicken pox was made. The patient was treated with intravenous acyclovir for 10 days and intravenous methylprednisolone in a dose of 30 mg/kg/day was given for 5 days. This was followed by oral prednisolone for a period of 4 weeks which was then tapered. The weakness started improving in one week and complete recovery occurred in 2 weeks with above treatment.

DISCUSSION

VZV is a neurotropic human *Herpes* virus.^{3,4} It has common CNS complications including encephalitis, cerebellar ataxia and rare complications are transverse myelitis, aseptic meningitis, Guillain-Barre syndrome, meningoencephalitis, ventriculitis, optic neuritis, post-hepatic neuralgia and stroke.³ Acute transverse myelitis (ATM) is a condition of sudden weakness of lower extremities with sensory involvement caused by inflammation of the spinal cord.³ Approximately 25 - 40%

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Figure 1: Healing chicken pox lesions on the chest.

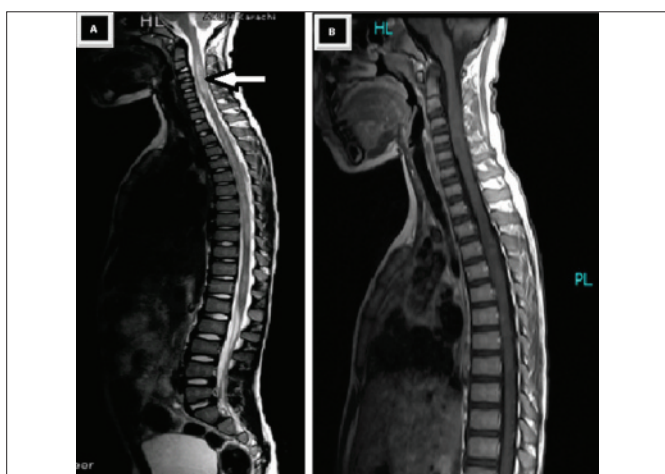


Figure 2 (A,B): MRI Spine (sagittal view). (A) T2 weighted image showing diffuse high intensity signals especially in the cervical and thoracic area. (B) Postcontrast sagittal view showing no enhancement.

cases of ATM are caused by *Herpes* viruses and poliovirus.² The interval between chicken pox and ATM is variable.⁴ La Rovere *et al.* reported that ATM can occur either with the onset of rash or it may be delayed up to 2 weeks.⁶ This patient showed the symptoms within 2 weeks of onset of vesicles.

The pathogenic bases for these complications are varied.⁴ It has been postulated to occur either as a direct viral invasion of the spinal cord or through an immune-mediated allergic mechanism.⁴ In ATM, it is difficult to isolate the virus in CSF, however, in an immunocompromised host, as the virus invades deeper tissues, it can be isolated from brain tissue or ventricles by polymerase chain reaction (PCR).⁴ Viral isolation by PCR was also negative in this patient.

In most cases, transverse myelitis caused by VZV is diagnosed by detection of typical vesicular lesions in dermatomal distribution that are associated with signs and symptoms suggestive of transverse myelitis.² However, transverse myelitis associated with *Varicella*

zoster has been described in the absence of typical skin lesions. Mainstay of treatment is with corticosteroids, and methylprednisolone which is preferable.⁷ The spectrum of clinical outcomes in VZV myelitis ranges from spontaneous recovery to ascending progression and death.⁸

In this case, transverse myelitis related to *Varicella* infection was diagnosed on the basis of appearance of crusted skin lesion, on the development of motor weakness, bladder dysfunction following the appearance of a rash and on the characteristic MRI findings. The patient's CSF contained anti-VZV antibodies. There are no established treatment regimens for transverse myelitis following VZV infection.¹ Most authors recommend high doses of steroid.¹ The use of acyclovir in the literature is not clear, however, in some case reports they have administered 10 mg/kg/dose every 8 hourly for 10 days and revealed it as useful medicine when used in combination with methylprednisolone.^{5,6} This patient was treated with high dose of methylprednisolone, and intravenous acyclovir. We suggest that combination therapy should be used in patients with VZV-associated ATM if the symptoms are within 2 weeks of the onset of the rash.

This patient improved within a week of initiation of therapy, and his symptoms completely resolved in 2 weeks. The American Academy of Pediatrics recommends universal *Varicella* immunization for children aged 12 months and older.¹ The authors emphasize the use of *Varicella* vaccine in children to prevent the CNS morbidity and possible sequelae associated with *Varicella* infections.

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