

Case Report

Varicella-zoster virus transverse myelitis in an immunocompetent patient

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ABSTRACT

Transverse myelitis is one of the rare neurological complications of Varicella-Zoster Virus (VZV) infection in immuno-competent. We report a 26-year-old immuno-competent gentleman who developed virologically confirmed myelopathy caused by VZV which improved with steroids and acyclovir leaving no residual neurological deficits.

Keywords: Varicella-zoster virus, Transverse myelitis, Immunocompetent

INTRODUCTION

Varicella-Zoster Virus (VZV) leads to numerous complications of the central and peripheral nervous systems.¹ These complications are rare in immunocompetent individuals.² Central nervous system involvement in VZV infection has been reported as 0.1-0.75% in several studies.^{3,4} Cerebellar ataxia and encephalitis can be seen very often while other complications like transverse myelitis, aseptic meningitis, Guillian-Barre syndrome, meningoencephalitis, ventriculitis, optic neuritis, delayed contralateral hemiparesis, peripheral neuropathy, cerebral angitis, Reye syndrome and facial paralysis are seldom encountered in clinical practice. Transverse myelitis is a group of disorders characterized by focal inflammation of the spinal cord and results in loss of motor and sensory function below the level of injury. The frequency of transverse myelitis during or following varicella infection is reported as 0.3%.³ We describe a case of virologically confirmed myelopathy caused by VZV to emphasize the possibility of its occurrence in immunocompetent subjects.

CASE REPORT

A 26 year old gentleman was admitted to our hospital with urinary retention, paresthesia and weakness of both lower limbs of 3 days duration. He had varicella infection 10 days prior to the onset of his symptoms. Crusted lesions of varicella were seen all over the body. He was fully conscious, oriented, fundi was normal and cranial nerves intact. Upper limbs were normal. He had normal bulk, spasticity, grade 3/5 power in all muscles, exaggerated tendon reflexes, sustained ankle clonus and extensor plantars in both lower limbs. All modalities of sensations were decreased below T4 level. The rest of the examination was normal. A clinical diagnosis of transverse myelitis was made.

Investigations showed hemoglobin 15 g/dl, total WBC count 8500/ μ l, platelet count 2.5×10^9 /L, ESR 32 mm in 1h. Renal function tests, liver function tests and urinalysis were all normal. MRI spine showed diffuse thickening of spinal cord with central T2W hyper intensity in the lower cervical and thoracic region, without enhancement, indicating a long segment myelopathy (Figure 1, Figure

2). HIV ELISA was negative. Cerebrospinal fluid was positive for VZV-IgG. A diagnosis of post varicella transverse myelitis was made and he was started on intravenous steroids and acyclovir. Steroids were eventually tapered and stopped. By the end of 2nd week, he had regained full power in the lower limbs, but paresthesia, bowel and bladder incontinence persisted. He recovered fully in 2 months.

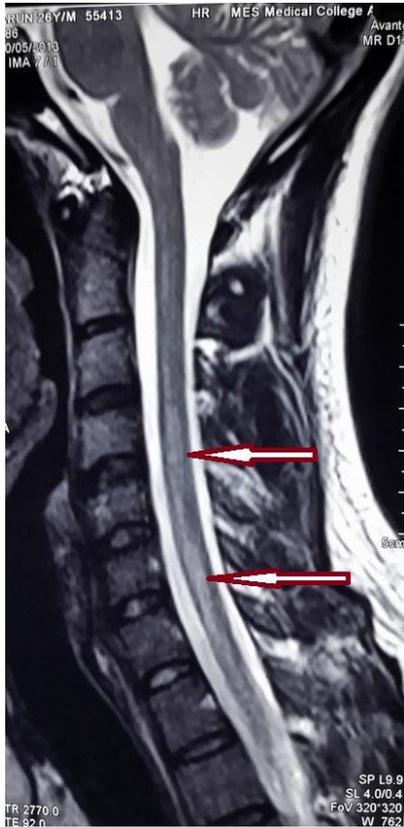


Figure 1: Sagittal T2 weighted image showing central nonenhancing hyperintensity in the lower cervical and thoracic region (red arrow).

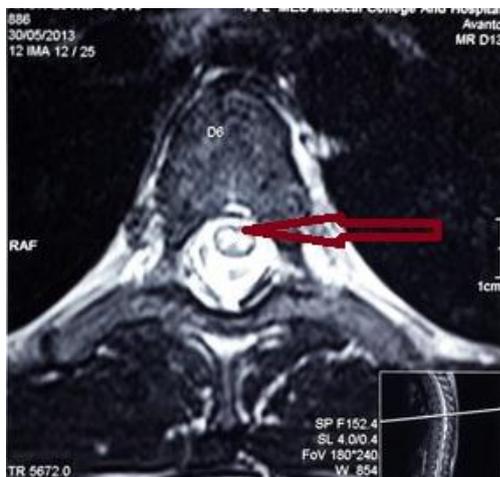


Figure 2: Axial T2 weighted image at D6 level showing central nonenhancing hyperintensity (red arrow).

DISCUSSION

Transverse myelitis is an inflammatory disease involving the entire thickness of the spinal cord. The critical factor seems to be an abnormal immune response to an infection rather than the direct effect of an infectious agent. It is known to occur against a background of viral diseases, vaccinations, systemic lupus erythematosus, vasculitis, multiple sclerosis, heroin abuse and trauma.⁵ Approximately 25%-40% cases of transverse myelitis are caused by viral infections with herpes viruses and poliovirus. Varicella zoster infection is not a common cause of transverse myelitis in immunocompetent patients.⁶

The frequency of transverse myelitis during or after varicella infection is of VZV 0.3%.³ The characteristic symptoms are bilateral sensory deficit at a given level, paraparesis, quadriparesis, and abnormal bladder and rectal function. In most cases of transverse myelitis due to VZV infection the features of transverse myelitis are accompanied by the typical vesicular lesions of VZV. However, transverse myelitis associated with herpes zoster has been described in the absence of typical skin lesions.⁷

The pathogenesis of neurological complications associated with VZV infection is unclear, allergic and vascular mechanisms have been suggested.⁸ In idiopathic acute transverse myelitis, there is an intraparenchymal or perivascular cellular influx into the spinal cord, resulting in the breakdown of the blood-brain barrier and variable demyelination and neuronal injury. However, the pathogenesis of VZV myelitis has been thought to be a direct viral invasion, because VZV particles were found in glial cells, and the virus has been isolated from the spinal cord of patients with zoster myelitis. In a detailed report that included post mortem examination of the spinal cord, Hogan et al. presented evidence of direct invasion of VZV in a patient with transverse myelitis associated with varicella zoster.⁹ Demonstration of the VZV antigen in CSF cells by immunofluorescence or isolation of VZV from the CSF is a confirmative evidence for viral central nervous system infection but it is rarely successful.¹⁰

Early diagnosis of VZV-related myelitis is based on its temporal relationship to the rash and detection of VZV DNA or VZV-specific antibodies or both in the CSF. The CSF analysis usually reveals a mild mononuclear pleocytosis with a normal or elevated protein.¹¹ Magnetic resonance imaging of the spine may produce normal results, or may demonstrate T2 hyperintense lesions in the spinal cord with occasional swelling and enhancement. There are no established treatment regimens for transverse myelitis as a complication of VZV infection. Some researchers recommend high doses of acyclovir and steroids.¹² Although clinical recovery is variable, many immunocompetent patients improve significantly, though fatal cases have been reported.

In our case, transverse myelitis related to VZV infection was diagnosed on the basis of clinical findings, MRI and CSF analysis. We report this case because post varicella transverse myelitis in an immunocompetent patient is very rare and VZV cannot be usually isolated from CSF. But in our patient CSF examination was positive for VZV-IgG. He had improved fully in two months with steroids and oral acyclovir.

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Ethical approval: Not required

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