Case report

Transverse Myelitis following Herpes Zoster in an Immunocompetent Older Patient : A rare case report

Nishchitha K, *Prateeksha Rai G

Department of Medicine, K.S.Hegde Medical Academy, Mangalore.
Corresponding author*

Abstract:
Varicella zoster virus (VZV) is a human neuro tropic alpha herpes virus that causes chickenpox (varicella) in children. VZV reactivation in adults (Herpes Zoster) may lead to neurological complications, including transverse myelitis. However, transverse myelitis caused by VZV reactivation is rare in immunocompetent patients. We report a case of transverse myelitis caused by VZV in an immunocompetent older patient, which was confirmed by polymerase chain reaction. A 43-year-old man visited our hospital with complaints of painful vesicular lesions over left side of the chest and back since 10 days, two days later he developed weakness of both lower limbs and decreased sensation from below the level of the nipple. Five days later he developed retention of urine. MRI Spine showed features suggestive of transverse myelitis extending from C2 to T10 level. His CSF analysis showed lymphocyte predominance and PCR for VZV DNA was positive. The patient was treated with acyclovir and steroids and his neurological functions improved. For older patients, early and aggressive antiviral treatment against VZV may be necessary even though these patients are immunocompetent.
Keywords: Transverse myelitis, Herpes zoster

Introduction

Varicella-zoster virus (VZV) is a human herpes virus that causes chickenpox and herpes zoster [1, 2]. The most common neurological complication of VZV reactivation is herpetic neuralgia, which is usually self-limited. However, VZV reactivation in immunocompromised patients can cause disseminated infections and severe neurologic dysfunctions, including meningitis, neuropathy, myelitis, stroke, and encephalitis [3]. Transverse myelitis is an unusual complication caused by VZV reactivation in immunocompetent patients [4]. To date, few cases of transverse myelitis are reported and most of them were not confirmed microbiologically [5-9]. We report a case of transverse myelitis caused by VZV in an immunocompetent older patient, and this case was confirmed microbiologically by detection of VZV DNA in the cerebrospinal fluid (CSF) by polymerase chain reaction (PCR).

Case report

A 43 year old male patient came to our hospital with history of painful vesicular eruptions over left chest around the nipple and back. He took Ayurvedic medication for the same. However, there was no relief in pain. 2 days after developing the lesions, he developed weakness of both lower limbs, initially he had heaviness which progressed over the next 5 days and patient was unable to walk without support. He also noticed decreased sensation from below the nipple level. 2 days prior to admission he developed retention of urine and was catheterised in a local hospital. On admission, the patient was conscious and oriented, blood pressure was 110/80 mmHg, the heart rate was 76 beats per minute, and body temperature was 36.3°C. Crusted vesicles were observed over T1 dermatome on the left side(Figure 1). Neurologic examination showed decreased power of 3/5

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

in both lower limbs. The deep tendon reflexes of lower limbs were exaggerated and plantar was extensor bilaterally. Also, the sensations of touch, joint position, vibration, pain and temperature was reduced below the level of T4. Magnetic resonance imaging (MRI) Spine indicated diffuse hyperintensity of the spinal cord extending from C2 to T10 level, suggestive of transeverse myelitis(Figure 2). The results of blood tests were as follows: white blood cell count, 9400 cells/cumm; hemoglobin, 15.2 g/dL; platelet count, 2.71,000 cells/cumm; erythrocyte sedimentation rate, 20mm/hour. HIV, HbsAg, HCV spots- non reactive. HIV ELISA- Negative. CSF analysis indicated abnormal values for white blood cell count (220 cells/cumm; 12% of neutrophils, 88% of lymphocytes), and protein (86 mg/dL), sugar (93 mg/dl) and a negative result for bacterial culture. VZV DNA was detected by PCR amplification in CSF. CSF Oligoclonal bands and NMO antiobodies (neuromyelitis optica) were negative. Intravenous acyclovir was initiated at 500 mg every 8 hours and continued for 21 days. Intravenous Methyl prednisolone (1g/day) was given for 5 days and then oral Prednisolone was started at 60 mg/day. The power of both legs improved to 5/5 with a rehabilitation program involving strength training. His bladder dysfunction recovered. However, sensory impairment below level T10 persisted during the time of discharge.

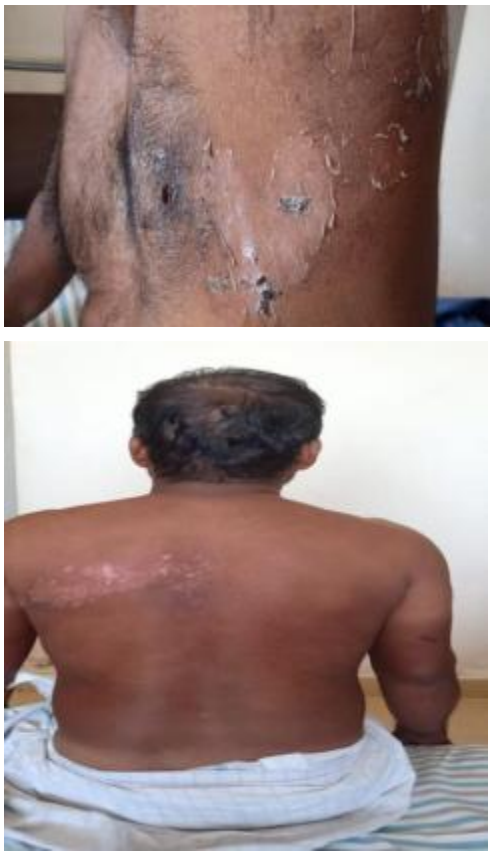

Figure 1- A 43 year old man with Herpes Zoster in the Left T4 dermatome

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

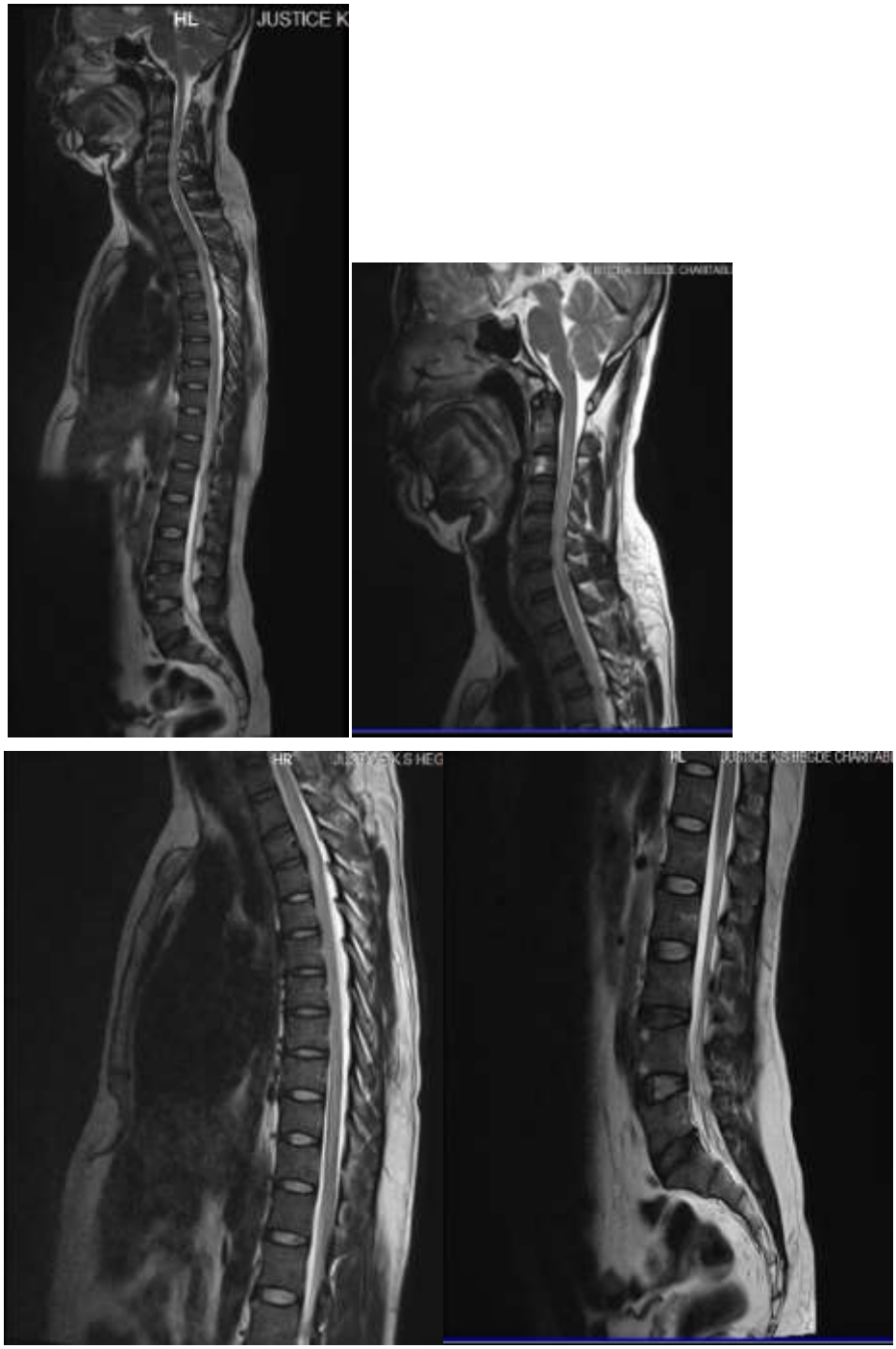

Figure 2- MRI spine showing long segment intramedullary hyperintensity extending from C2 to T10 level suggestive of Transeverse Myelitis

DISCUSSION

VZV is a human neurotropic alphaherpesvirus that causes chickenpox (varicella) in children [1, 2]. After primary infection, the virus becomes latent in cranial nerve and sensory root ganglia [2, 3]. However, VZV reactivation may occur with advanced age or immunosuppression, particularly in cases of cell-mediated immunosuppression [1-3]. VZV reactivation may cause neurological complications such as chronic pain (postherpetic neuralgia), cranial nerve palsy, zoster paresis, meningoencephalitis, cerebellitis, myelopathy,

www.ijbamr.com P ISSN: 2250-284X , E ISSN : 2250-2858

multiple ocular disorders, and stroke [2-4]. The most common manifestation of VZV reactivation is herpes zoster. Unvaccinated individuals aged 85 years or older have a 50% risk of developing herpes zoster [10]. However, transverse myelitis is one of the rarest complications, particularly in immunocompetent patients [1-3].Myelitis and encephalitis due to VZV reactivation are more common in immunocompromised patients [1]. In these patients, VZV myelitis may occur without typical skin lesions and can occur far different level of skin lesion [4]. By contrast, in immunocompetent patients, VZV myelitis has a typical presentation (dermatomal rashes followed by myelitis at the corresponding level) and good outcomes [1, 4]. Older patients may show a variety of neurologic symptoms from local paralysis to severe neurologic dysfunction due to multiple causes; therefore, thinking of several possibilities is critical and various examinations are needed to differentiate the causes. To date, no predictable markers of disease progression are available to patients with VZV myelitis [4]. Therefore, clinical suspicion and aggressive evaluation are crucial for the early diagnosis of VZV myelitis. The detection of VZV antibodies and VZV DNA in CSF are confirmatory diagnostic tests. Imaging studies are useful for the diagnosis of VZV myelitis. MRI of VZV myelitis is likely to show T2-hyperintensity in the spinal cord . Although the standard treatment regimen for VZV myelitis is not yet established, there is anecdotal evidence for treatment of VZV myelitis with acyclovir [4]. Moreover, there is little evidence that early antiviral treatment reduces the risk of VZV myelitis. Therefore, the early diagnosis and antiviral treatment of VZV is essential to recovery from myelitis and minimize its complications, and this treatment is crucial to prevent the development of postherpetic neuralgia.

References:

1.Gilden DH, Kleinschmidt-DeMasters BK, LaGuardia JJ, Mahalingam R, Cohrs RJ. Neurologic complications of the reactivation of varicella-zoster virus. N Engl J Med 2000;342:635-45.

2. Gilden D, Nagel MA, Cohrs RJ, Mahalingam R. The variegate neurological manifestations of varicella zoster virus infection. Curr Neurol Neurosci Rep 2013;13:374.

3. Amlie-Lefond C, Jubelt B. Neurologic manifestations of varicella zoster virus infections. Curr Neurol Neurosci Rep 2009;9:430-4.

4. Hung CH, Chang KH, Kuo HC, Huang CC, Liao MF, Tsai YT, Ro LS. Features of varicella zoster virus myelitis and dependence on immune status. J Neurol Sci 2012;318:1924.

5. Baik JS, Kim WC, Heo JH, Zheng HY. Recurrent herpes zoster myelitis. J Korean Med Sci 1997;12:360-3.

6. Han MK, Lee SS, Han SH. Herper zoster myelitis confirmed by detection of varicell-zoster virus DNA using the polymerase chain reaction. J Korean Neurol Assoc 1996;14:1067-71.

7. Hwang YM, Lee BI, Chung JW, Ahn JH, Kim KW, Kim DI. A case of herpes zoster myelitis. J Korean Neurol Assoc 1988;6:272-7.

8. Yang IS, Lim GD, Shin SS. A case of herpes zoster myelitis: a case report. J Korean Pain Soc 1997;10:266-9.

9. Yi H, Lee Y, Shin HY, Kim SM. Varicella zoster viral myelitis in immunocompetent patient. J Korean Neurol Assoc 2014;32:339-41.

10. Cohen JI. Clinical practice: Herpes zoster. N Engl J Med 2013;369:255-63.