Myelitis: A Rare Neurological Complication of Herpes Zoster

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Sir,

Varicella-zoster virus (VZV) is an uncommon cause of myelitis. Among the zoster patients, the incidence of myelitis is reported to be only 0.3%. Although myelitis following herpes zoster (HZ) has been reported in several neurological journals, there is a dearth of such cases in medical journals. Hence, we are reporting this case.

A 16-year-old boy was referred from the Neurology outpatient Department with a history of skin lesions with weakness and tremors of both the lower limbs for 4–5 days. A detailed history revealed pain in the right thoracic region 10 days earlier, following which fluid-filled lesions developed over the same area within 4–5 days. The patient then developed a sudden onset of bladder incontinence with weakness in both the lower limbs. The patient gave no history of similar complaints in the past. On examination, grouped crusted lesions were seen on an erythematous background at the level of T7–T8 dermatome [Figure 1a and b]. No atypicality in the lesions was seen. Neurological examination revealed 0/5 power in both the lower limbs and complete loss of sensation (pain and temperature) over bilateral lower limbs and lower abdomen up to the involved dermatome. The patient had a urinary catheter *in situ*. There was no motor or sensory loss involving the upper limbs. There was no cranial nerve involvement. Investigations revealed a normal hemogram. Random blood sugar, liver, and renal function tests were within normal limits. Serological test was positive for HIV antibodies and negative for hepatitis B surface antigen. Cerebrospinal fluid (CSF) analysis revealed clear and colorless fluid with a white blood cell count of 27 and red blood cell count of 12, with normal protein and glucose levels. CD4 cell counts were 300 cells/cumm. CSF polymerase chain reaction (PCR) was not done in the case. Magnetic resonance imaging (MRI) of the dorsal spine revealed hyperintensity on T2-weighted 1 [Figure 2] appearing hypointense on T1-weighted 1 involving spinal cord from D1–D5 levels to D10–D12 levels, suggesting the possibility of viral myelitis probably due to HZ.

The sequential appearance of HZ followed by motor weakness of bilateral lower limbs with characteristic MRI findings was a pointer to the diagnosis of HZ myelitis.

The patient was started on injectable acyclovir 500 mg/m^2 intravenous 8 hourly for 7 days with

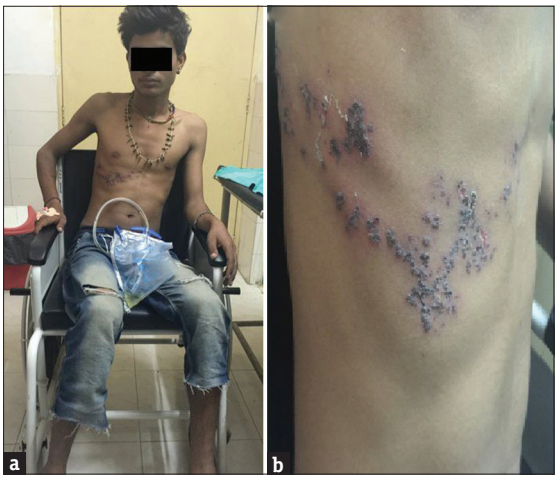

Figure 1: (a) A 16-year-old boy with herpes zoster in T7–T8 dermatome. (b) Grouped crusted lesions on erythematous background

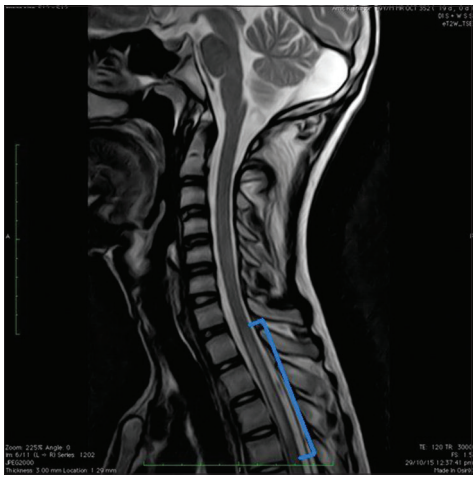

Figure 2: Magnetic resonance imaging dorsal spine showing intramedullary hyperintensity from D1 to D5 levels, suggesting the possibility of transverse myelitis

injection methylprednisolone 1 g intravenous once daily. The patient was subsequently referred to a higher center for commencing antiretroviral therapy and was unfortunately lost to follow-up.

VZV is a member of the herpes virus family.[1] It is a human, neurotropic, alpha-herpes virus.[2] During the course of the primary infection (varicella), the virus passes up the sensory nerves and it is transported centripetally to the sensory ganglion where it establishes a latent infection. With the decline in VZV-specific cell-mediated immunity, VZV reactivates from the ganglion and travels anterograde to the skin causing zoster, however may travel retrogradely to produce neurological complications such as segmental sensory loss or zoster paresis, polyradiculoneuritis, aseptic meningitis, meningoencephalitis, ventriculitis, leukoencephalopathy, vasculopathy, necrotizing angiitis, and transverse myelitis or myelopathy.[3,4]

Transverse myelitis is an unusual inflammatory disease involving the entire thickness of the spinal cord characterized by sensory, motor, or autonomic dysfunction below the level of injury. It excludes extra-axial compressive etiologies.[5] Transverse myelitis is known to occur on a background of viral diseases, vaccinations, systemic lupus erythematosus, vasculitis, multiple sclerosis, heroin abuse, and trauma. Approximately, 25–40% of the cases of transverse myelitis are caused by viral infections with herpes viruses and poliovirus.[3]

Myelitis due to HZ was first reported by Hardy and Zona in 1876.[6] The reported frequency of myelitis during or after varicella or zoster infection is 0.3%.[7] The onset of HZ myelitis is usually acute or subacute with a mean delay of 2 weeks between the initial vesicular rash and the neurological disturbance. Symptoms usually begin unilaterally, ipsilateral to the rash, but subsequently become bilateral. Motor manifestations are followed by spinothalamic and posterior column sensory abnormalities or bladder dysfunction. Fatal myelitis is seen mostly in immunocompromised patients, and a chronic or remitting exacerbating myelopathy may also be encountered.[8] Occasionally, HZ myelitis may be seen in the absence of cutaneous lesions. It has also been reported in association with sickle cell anemia and systemic lupus erythematosus.[9,10]

The pathogenesis of VZV myelitis is unclear. The postulated hypothesis include immunological, wherein delayed type of hypersensitivity after VZV infection is implicated. Direct VZV infection of neuroectodermal cells, particularly oligodendrocytes, has been demonstrated by immunostaining and it is associated with focal demyelination.[11,12] VZV vasculitis, leptomeningitis, and hemorrhagic necrosis of the spinal cord are also described. Pathological involvement is most severe in the spinal cord segment corresponding to the involved dermatome. There is a variable spread both horizontally and vertically in the spinal cord.[11,12]

The diagnosis of HZ myelitis is usually not difficult when the neurological symptoms develop in temporal proximity to the rash, but, however, a high index of suspicion should be kept in atypical cases.[12]

Investigations which help to come to a confirmatory diagnosis include CSF analysis which usually reveals mononuclear pleocytosis with normal or elevated protein. The diagnosis is confirmed by finding VZV-specific DNA or anti-VZV IgG in CSF using PCR.[13,14]

The best imaging tool in viral myelitis is MRI. The findings include diffuse increase in signal intensity through the involved segment. A variable nonfocal enhancement may also be seen.[15]

There are no established treatment guidelines for myelitis due to HZ. High doses of acyclovir and steroid are usually recommended. The longer the duration between myelopathy and treatment, the slower is the observed improvement. Residual neurological deficits may be seen in spite of all the treatments.

Our patient had a rapid onset of lower limb paralysis following HZ, probably due to immunocompromised state. This case is being reported to highlight this unusual neurological complication and to understand the pathogenesis of the same. The association of HZ with paresis, especially when in temporal proximity, is a strong indicator of myelitis. Timely recognition and early institution of treatment may decrease the progression of myelitis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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