CASE REPORT

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# Cervical myelitis due to herpes zoster: case report

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## Abstract

**Purpose:** Varicella-zoster virus (VZV) belongs to the human neurotropic alpha-herpes virus group. VZV reactivation can lead to neurological complications, including transverse myelitis. However, transverse myelitis caused by VZV reactivation is rare in immunocompetent patients. A case of transverse myelitis caused by VZV in an immunocompromised patient is presented in this paper.

**Case description:** A 64-year-old female patient was admitted to our outpatient clinic with complaints of pain, numbness and loss of strength in her right arm, and decreased sensation of warmth, after suffering from zona zoster infection two weeks before. At that time the patient had shingles in the area covering the C4-T1 dermatomes on her right side and was treated with acyclovir.

**Comment:** Consequently, patients presenting with similar symptoms after shingles should undergo appropriate imaging and tests, and treatment should be given for shingles-related transverse myelitis.

**Key words:** varicella-zoster virus, shingles, myelitis, cervical.

## PURPOSE

Varicella-zoster virus (VZV) is a herpes virus that can remain latent in the trigeminal nerve and dorsal root ganglia, causing chickenpox and shingles. The most common neurological complication of VZV reactivation is herpetic neuralgia. However, in immunocompromised patients VZV reactivation can cause disseminated infections, and severe neurological dysfunctions including meningitis, neuropathy, myelitis, stroke, and encephalitis. Neurological complications caused by VZV are reported as 0.1-0.75% [1, 2]. Among these, cerebellar ataxia and encephalitis are frequently seen, while transverse myelitis, Guillain-Barré syndrome, and meningoencephalitis are rarely encountered. Transverse myelitis is one of the rarest complications, especially in immunocompetent patients. The incidence of transverse myelitis during or after varicella infection is 0.3% [1-3].

## CASE DESCRIPTION

A 64-year-old female patient was admitted to our outpatient clinic with complaints of pain, numbness and loss of strength in her right arm, and decreased sensation of warmth, after suffering from herpes zoster infection two weeks before. At that time the patient had shingles in the area covering the C4-T1 dermatomes on her right side and was treated with acyclovir. There was still pain, numbness, and loss of warmth, especially in the area covering the dominant C2-T1 dermatomes on the right. The patient had no history of disease, medication, trauma, or history of febrile illness, and was admitted to our neurology department for further examination and treatment.

On physical examination, her temperature was 36.6°C, her blood pressure was 110/70 mmHg, heart rate 83/minute, respiration rate 18/minute, and oxygen saturation 98%.

In her neurological examination she was conscious, oriented, and cooperative. Her pupils were isochoric, light reflex was present in both eyes, and eye movements were free in all directions. Her speech was normal and she had no facial asymmetry. According to the Medical Research Council (MRC) scale, on motor examination, the right upper extremity was –5/5. On sensory examina-

tion, she had hypoesthesia in her right upper extremity, her cerebellar examination was skillful. Deep tendon reflexes (DTR) were normoactive in both upper and lower extremities, plantar response was flexor. There was no sign of meningeal irritation. The patient had routine blood tests, biochemistry, whole blood, vitamin $B_{12}$ tests, thyroid function tests, $HbA_{1c}$, erythrocyte sedimentation rate, serum electrophoresis, autoantibody screening (antinuclear antibody, anti-SSA, anti-SSB), antithyroid antibodies, syphilis serology (fluorescent treponemal antibody); Schirmer's test was negative.

The patient's serum lymphocyte cell count was within normal limits. Elisa tests (hepatitis A, HIV, hepatitis B, hepatitis C, rubeola, rubella) were negative. Complete urinalysis was within normal limits. When viral meningitis agents were investigated, no agent was found. Also, the *Brucella* tests came back negative.

In the cerebrospinal fluid (CSF) analysis performed by lumbar puncture; lymphocytic pleocytosis (902 cells/µl; 0% of neutrophils, 94% of lymphocytes, and 6% of monocytes), and protein 462.5 mg/dl (150-450 mg/dl) and VZV-DNA positivity were detected in the polymerase chain reaction (PCR) analysis in CSF. Brain computed tomography (CT) and brain magnetic resonance imaging (MRI) were normal. In cervical MRI, hyperintense lesions were detected in the T2 sequence in C1-C4 cervical segments (Figure I).

Contrast-enhanced cervical MRI could not be performed due to the patient's contrast allergy.

Nerve conduction studies were normal in her electromyography (EMG). Somatosensory evoked potentials (SEP) were consistent with cervical spinal cord posterior column dysfunction and especially the right upper extremity deep sensory tracts were significantly affected. Transverse myelitis due to zona zoster infection was considered and intravenous methylprednisolone 1 g/day for 7 days and acyclovir treatment were continued for 7 days.

After the treatment, the patient's symptoms decreased significantly.

## COMMENT

VZV is a human neurotropic double-stranded DNA alpha-herpes virus; primary infection usually occurs in childhood and manifests as chickenpox. After primary infection, the VZV becomes latent in the cranial root ganglia or dorsal root ganglia and may remain so for several decades; however, it may be reactivated as herpes zoster in elderly or immunocompromised persons.

Herpes zoster may develop various complications such as herpes zoster vasculitis and ophthalmic, neurological, or visceral disease [1, 3, 5]. The most common neurological complication is postherpetic neuralgia, a chronic neuropathic pain. Other complications may include cranial or peripheral nerve palsy, myelitis, meningoencephalitis, and stroke [2, 3, 6].

Transverse myelitis occurs as a result of a focal lesion characterized by inflammation, edema and necrosis in one or more spinal segments, with motor, sensory or autonomic involvement. The immune pathogenesis of transverse myelitis is variable and reflects the highly diverse spectrum of this disease, from idiopathic mechanisms to disease-associated myelitis. Perivascular infiltration by monocytes and lymphocytes is present in the lesion [2, 3, 4, 7].

The pathogenesis of neurological complications associated with VZV infection is unclear. It has been stated in the literature that it can involve all parts of the spinal cord and that the pathogenesis may be directly related to viral invasion and consequent axonal degeneration [2, 3, 7].

Allergic and vascular mechanisms have been proposed for some of these neurological complications that occur after primary chickenpox and herpes zoster infection. The pathogenesis of VZV myelitis has been thought

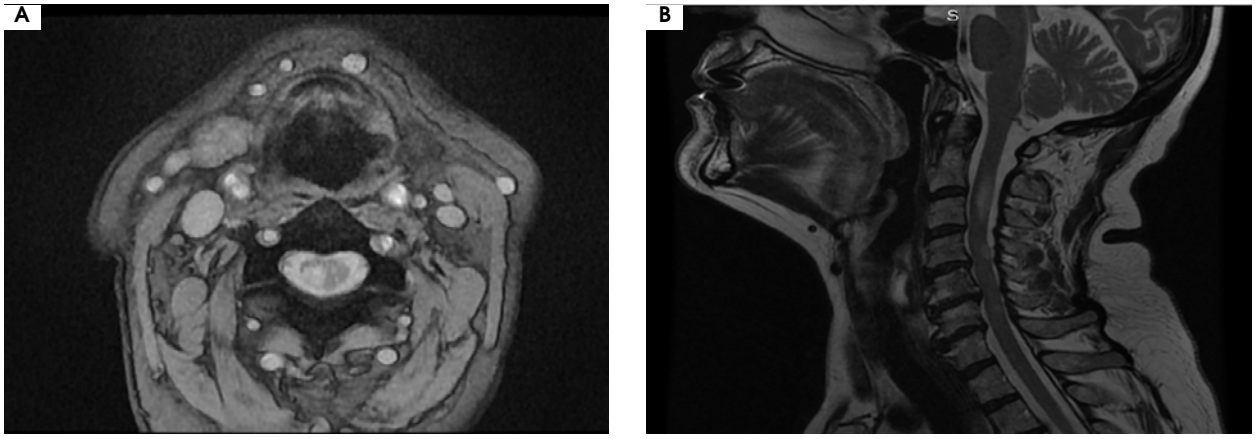


**Figure I.** Hyperintense lesions in C1-C4 cervical segments in cervical magnetic resonance imaging T2 sequence

to be a direct viral invasion, since VZV particles are found in glial cells and the virus is isolated from the spinal cord of patients with zoster myelitis.

It is hypothesized that reactivation of VZV (in the dorsal root ganglia of the spinal cord, Gasser's ganglia of the trigeminal and other cranial nerves) may therefore produce neurological complications, the most common of which are radiculopathies and cranial neuropathies. However, myelitis, encephalitis and vasculitis of the central nervous system can also be triggered. Axonal degeneration has also been reported [1, 3]. Symptoms of transverse myelitis usually develop over hours to days and progress gradually, sometimes over weeks. It often involves both sides of the body below the affected area of the spinal cord but can affect only one side. Common signs and symptoms include pain (back, abdomen, chest, extremities), abnormal sensation, weakness in the arms or legs, and bladder and bowel problems. It is estimated that about 25-40% of myelitis cases are due to viral infections such as herpes and polio [4, 6]. Myelitis is a rare neurological complication of VZV infection, mainly identified in immunocompromised patients, but cases have also been reported in immunocompetent patients. The prevalence of encephalitis due to VZV is particularly high, as reported in numerous studies [2, 8, 9]. In our case, we reported a case of transverse myelitis caused by VZV infection in an adult immunocompetent patient. Cases of VZV myelitis in adult patients have been discussed in the literature. In our case, the patient did not have any additional disease and did not have any pathological findings related to the immune system in his examinations and analyzes. Typical symptoms of VZV-induced transverse myelitis consist of rapidly progressive motor, sensory and autonomic dysfunctions, usually preceded by a dermatomal rash involving the same spinal level. If several diagnostic studies, including MRI, CSF analysis, and complete blood count provide evidence of inflammation, a diagnosis of transverse myelitis can be made after other causes have been ruled out. MRI is one of the most effective diagnostic tools for transverse myelitis secondary to VZV. On MRI, VZV myelitis is shown as hyperintensity in the spinal cord on T2-weighted images. Myelitis findings were detected in our patient approximately two weeks after zona zoster. Hyperintensity was detected in spinal MRI C1-C4 segments of our patient. In the case of VZV myelitis, the diagnosis is based on the detection of VZV DNA in PCR analysis or the production of VZV IgG in the CSF. In our patient, VZV IgG was elevated in CSF.

Since there is not yet a standard treatment regimen for transverse myelitis secondary to VZV, intervention should be tailored to the patient's symptoms. Antiviral drugs, high doses of corticosteroids, and intravenous immunoglobulin have been used, but the evidence for the effectiveness of most of them is not well defined [1, 2, 5]. For our patient, 1 g/day methylprednisolone and acyclovir treatment was given for 7 days. Plasma exchange can be used in cases that do not respond to high-dose treatment with glucocorticoids. In our case, early administration of a combination therapy containing antiviral drugs and corticosteroid significantly reduced the patient's symptoms.

Consequently, patients presenting with similar symptoms after shingles should undergo appropriate imaging and tests, and treatment should be given for shingles-related transverse myelitis.

## Conflict of interest

Absent.

## Financial support

Absent.

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