RESEARCH PAPER

Herpes Zoster-Related Myelitis with Cerebrospinal Fluid Oligoclonal Bands: Immune-Mediated Demyelination in an Uncontrolled Diabetic Patient

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ABSTRACT

Varicella-zoster virus (VZV) reactivation causes herpes zoster, typically presenting with painful vesicular eruptions. Neurological complications, particularly myelitis, are rare yet significant sequelae of VZV infection, even in immunocompetent individuals. In such cases, herpes zoster myelitis can present with varied neurological deficits, frequently involving motor dysfunction, spinothalamic sensory deficits, and, less commonly, posterior column involvement. In this case, the patient presented with gait imbalance, bladder incontinence, and loss of proprioception, indicating posterior column dysfunction. we report this case of herpes zoster myelitis which was further complicated by Cerebero-Spinal fluid (CSF) -specific oligoclonal bands, mimicking autoimmune demyelinating conditions, in a patient with uncontrolled diabetes.

Keywords: Herpes zoster, Myelitis, Oligoclonal bands, Demyelination, Diabetes mellitus, Varicella-zoster virus

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INTRODUCTION

Herpes zoster is caused by the reactivation of the varicella-zoster virus from sensory ganglia . While commonly characterized by dermatomal rashes, Varicella Zoster Virus (VZV) reactivation can lead to a spectrum of neurological complications, including postherpetic neuralgia, meningitis, encephalitis, vasculopathy, and, rarely, myelitis . Herpes zoster myelitis is a rare but severe manifestation of VZV reactivation, characterized by inflammation of the spinal cord . we report this because, distinguishing it from other forms of myelitis, particularly autoimmune demyelinating conditions, can be challenging, especially when CSF analysis reveals oligoclonal bands . The presence of oligoclonal bands restricted to the cerebrospinal fluid indicates intrathecal immunoglobulin G synthesis, a hallmark often associated with neuroinflammatory processes seen in conditions like multiple sclerosis, further complicating the diagnostic landscape in VZV-associated myelitis .

Case Presentation

A 55-year-old woman presented with a 5-day history of burning pain over the left upper chest, followed by the development of vesicular lesions over the same region within three days. The lesions were localized to the T7 dermatome and did not cross the midline. She reported moderate to severe pain and low-grade fever, without respiratory or systemic symptoms. Her past history was significant for type 2 diabetes mellitus, not on regular treatment. On examination, she was afebrile, alert, and hemodynamically stable. A cluster of vesicular eruptions was observed along the T7 dermatome on the left side. Dermatological evaluation confirmed herpes zoster, and

she was prescribed oral valacyclovir 1 g three times daily for seven days.

Laboratory evaluations demonstrated normal hematological and biochemical profiles. Fasting blood glucose was 307 mg/dL, postprandial glucose 340 mg/dL, and HbA1c 13.16%, signifying inadequate glycemic control. Thyroid function tests were unremarkable. On the third day of antiviral therapy, the patient developed dizziness, urinary incontinence, and gait instability.

Neurological examination showed preserved muscle tone and strength in all extremities, with absent patellar and Achilles reflexes. Sensory assessment revealed intact light touch, pain, and temperature sensations, but impaired joint position sense, vibration, and proprioception. Perianal sensation was preserved, although anal sphincter tone was reduced. Romberg's sign was positive, and nystagmus was absent.

Spinal magnetic resonance imaging revealed short-segment T2/STIR hyperintensities spanning the D5 to D9 levels, consistent with demyelinating pathology. Cerebral MRI demonstrated multiple scattered demyelinating foci. Nerve conduction studies indicated sensory and motor axonal demyelinating radiculoneuropathy involving all four limbs, with absent sensory nerve action potentials. (Figure 1)

Cerebrospinal fluid analysis yielded protein of 50 mg/dL, glucose of 93 mg/dL, LDH of 22, and 93 cells. Polymerase chain reaction assays for HSV-1, HSV-2, VZV, and enterovirus were negative, as were aquaporin-4 and myelin oligodendrocyte glycoprotein antibodies. Oligoclonal band testing identified 4–10 discrete bands in the cerebrospinal fluid, absent in serum, indicative of intrathecal IgG synthesis. Herpes zoster–associated immune-mediated demyelinating myelitis was diagnosed on the basis of clinical, imaging, and cerebrospinal fluid findings. Corticosteroid therapy was initiated, yielding gradual neurological recovery.

DISCUSSION

Herpes zoster myelitis constitutes a rare yet clinically significant neurological complication of varicella-zoster virus reactivation, typically occurring in immunocompromised patients but also reported in immunocompetent individualswith an estimated incidence of <0.3% of herpes zoster cases. Its pathogenesis may involve direct viral invasion of spinal cord neuroectodermal cells, particularly oligodendrocytes in the dorsal root entry zone and posterior horn, leading to focal demyelination, or a para-infectious immune-mediated demyelinating process. In the present case, the 3–6 day interval between rash onset and neurological symptom emergence, alongside negative CSF VZV PCR and presence of CSF-restricted oligoclonal bands, strongly supports an immune-mediated mechanism. This immune response can manifest as a post-infectious myelitis, observed days to weeks after acute VZV infection, often responsive to corticosteroids . Such immune-mediated demyelination has been documented to occur even in the absence of active cutaneous lesions, or with a delayed presentation several weeks after rash onset, further highlighting the diagnostic complexity .

Although oligoclonal bands are a hallmark of multiple sclerosis, they have also been documented in viral and post-infectious myelitis, including herpes zoster cases with CSF-restricted bands due to intrathecal IgG synthesisPrevious reports confirm that herpes zoster reactivation can provoke this immune activation, producing discrete CSF-specific OCBs even with negative viral PCR. In our case, the absence of aquaporin-4 and MOG antibodies, combined with short-segment localized spinal cord lesions and negative CSF VZV PCR, strongly supports post-infectious immune-mediated demyelination rather than NMOSD or MS. Furthermore, the presence of diabetes mellitus, particularly uncontrolled, is a recognized risk factor for both VZV reactivation and exacerbated immune responses, potentially contributing to the severity and demyelinating nature of the myelitis observed in this patient. Optimal management involves early antiviral therapy during the acute phase and corticosteroid therapy for inflammatory or demyelinating sequelae. The prognosis is generally favorable if diagnosed early. In our patient, uncontrolled diabetes likely contributed to severe infection and immune dysregulation, predisposing her to demyelination following viral reactivation.

CONCLUSION

This case report highlights the rare occurrence of herpes zoster–induced immune-mediated demyelinating myelitis with CSF-restricted oligoclonal bands, even in the setting of uncontrolled diabetes mellitus. Herpes zoster myelitis affects <0.3% of cases and the clinicians must recognize its potential to mimic demyelinating diseases such as multiple sclerosis due to shared features like oligoclonal bands and spinal lesions. Rash-to-symptom interval, negative CSF VZV PCR, and intrathecal IgG synthesis in this patient strongly support a post-infectious immune-mediated pathogenesis responsive to early antiviral and corticosteroid therapy, scoring the need for prompt intervention to avert long-term neurological disability, especially in diabetic or immunocompromised individuals.

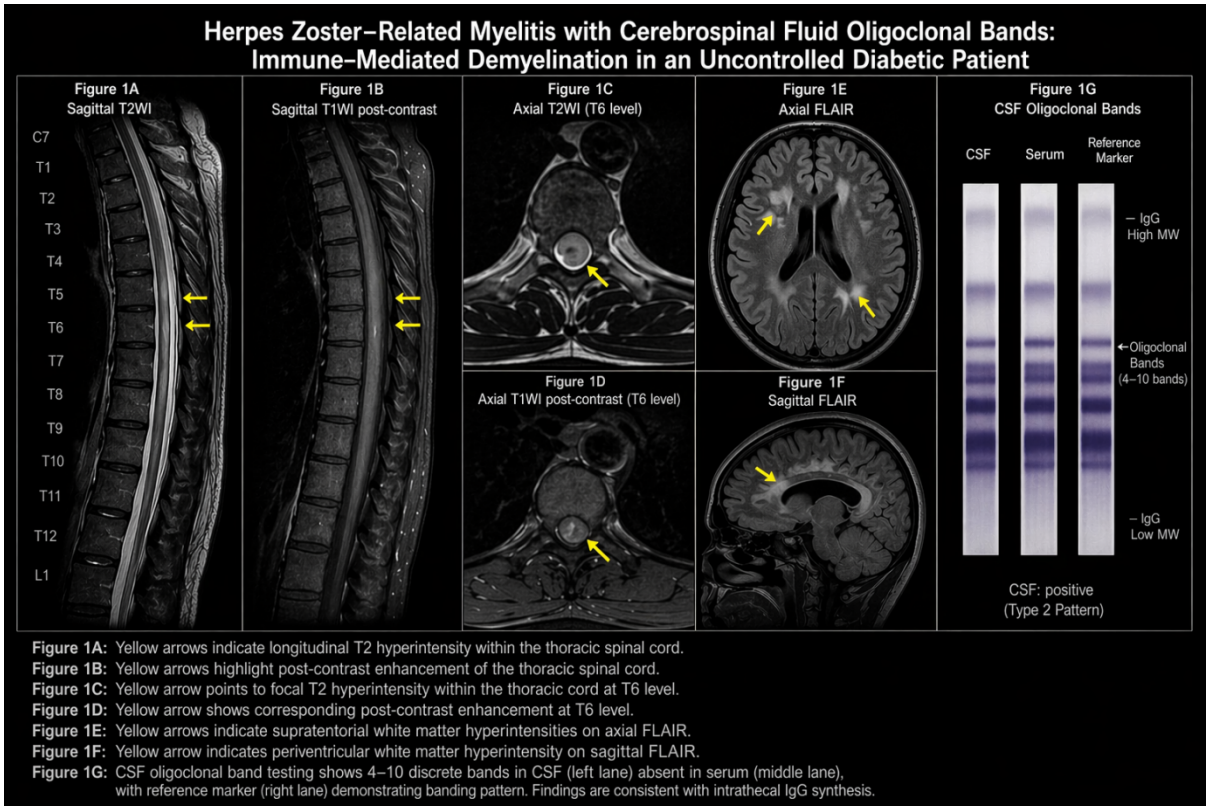

Figure 1A: Yellow arrows indicate longitudinal T2 hyperintensity within the thoracic spinal cord.
Figure 1B: Yellow arrows highlight post-contrast enhancement of the thoracic spinal cord.
Figure 1C: Yellow arrow points to focal T2 hyperintensity within the thoracic cord at T6 level.
Figure 1D: Yellow arrow shows corresponding post-contrast enhancement at T6 level.
Figure 1E: Yellow arrows indicate supratentorial white matter hyperintensities on axial FLAIR.
Figure 1F: Yellow arrow indicates periventricular white matter hyperintensity on sagittal FLAIR.
Figure 1G: CSF oligoclonal band testing shows 4–10 discrete bands in CSF (left lane) absent in serum (middle lane), with reference marker (right lane) demonstrating banding pattern. Findings are consistent with intrathecal IgG synthesis.

Figure Legends:
Figure 1 demonstrates magnetic resonance imaging features consistent with Herpes zoster myelitis. Figure 1A–D show longitudinally extensive signal abnormalities within the thoracic spinal cord with corresponding post-contrast enhancement (yellow arrows). Figure 1E and 1F depict associated supratentorial white matter hyperintensities on FLAIR sequences (yellow arrows). Figure 1G illustrates cerebrospinal fluid oligoclonal band analysis demonstrating 4–10 discrete bands present in CSF and absent in serum, indicative of intrathecal IgG synthesis.

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