



LETTER TO THE EDITOR

Lower lumbar and sacral herpes zoster with central nervous system complications: Two case reports

Mahir Kapmaz¹, Ezgi Yilmaz¹, Elmir Khanmammadov², Tehran Aliyeva², Ali Mert¹

¹Istanbul Medipol University Faculty of Medicine, Department of Infectious Diseases and Clinical Microbiology, Istanbul, Türkiye

²Istanbul Medipol University Faculty of Medicine, Department of Neurology, Istanbul, Türkiye

Dear Editor,

Herpes zoster rarely involves the lower lumbar or sacral dermatomes and only infrequently results in neurological complications such as aseptic meningitis or encephalitis, particularly in immunocompromised individuals (1, 2). We report two cases of neurological complications associated with herpes zoster involving uncommon dermatomes. These cases highlight that varicella-zoster virus (VZV) infection in atypical dermatomes may be complicated by central nervous system (CNS) disease, even in the presence of normal neuroimaging findings. They also underscore the critical role of cerebrospinal fluid (CSF) polymerase chain reaction (PCR) testing in establishing the diagnosis in both immunocompromised and immunocompetent patients presenting with unexplained neurological symptoms.

Case 1: A 68-year-old man with end-stage renal disease who had been receiving hemodialysis for two years presented with a three-day history of speech disturbance. Two days before admission, he experienced dizziness, syncope, and a fall. One week earlier, he had developed a vesicular rash involving the left buttock and thigh (L2–L5 dermatomes), which had been treated with oral acyclovir. On physical examination, the patient was mildly disoriented, had no neck stiffness, and exhibited drying vesicular

lesions (Fig. 1a–c). Laboratory investigations revealed a C-reactive protein (CRP) level of 50 mg/L, a white blood cell (WBC) count of 5,700/μL (lymphocytes: 900/μL), creatinine level of 7.56 mg/dL, a urea level of 135 mg/dL, and a platelet count of 167,000/μL. Testing for antibodies to human immunodeficiency virus (HIV) was negative. Brain imaging, electroencephalography (EEG), and fundoscopic examination were unremarkable. Lumbar puncture demonstrated a CSF protein concentration of 46 mg/dL, a glucose level of 48 mg/dL (serum glucose: 101 mg/dL), and 65 cells/mm³ (98% mononuclear cells). Multiplex PCR testing was positive for varicella-zoster virus, confirming the diagnosis of VZV encephalitis. Cerebrospinal fluid cultures were negative. The patient received intravenous acyclovir (350 mg/day) for seven days, followed by valacyclovir (500 mg/day) for an additional seven days, resulting in complete neurological recovery. Written informed consent for publication of this report and the accompanying images was obtained from both patients.

Case 2: A 47-year-old man with a history of chronic anxiety disorder treated with fluoxetine, venlafaxine, and alprazolam presented with a five-day history of severe bilateral temporal headache accompanied by insomnia, loss of appetite, dizziness aggravated by movement, and photophobia. He had no previous history of headaches. Physical examination revealed

How to cite this article: Kapmaz M, Yilmaz E, Khanmammadov E, Aliyeva T, Mert A. Lower lumbar and sacral herpes zoster with central nervous system complications: Two case reports. *Dusunen Adam J Psychiatr Neurol Sci* 2026;39:264-266.

Correspondence: Mahir Kapmaz, Istanbul Medipol University Faculty of Medicine, Department of Infectious Diseases and Clinical Microbiology, Istanbul, Türkiye

E-mail: mahirkapmaz@yahoo.com

Received: December 14, 2025; **Revised:** May 10, 2026; **Accepted:** May 31, 2026



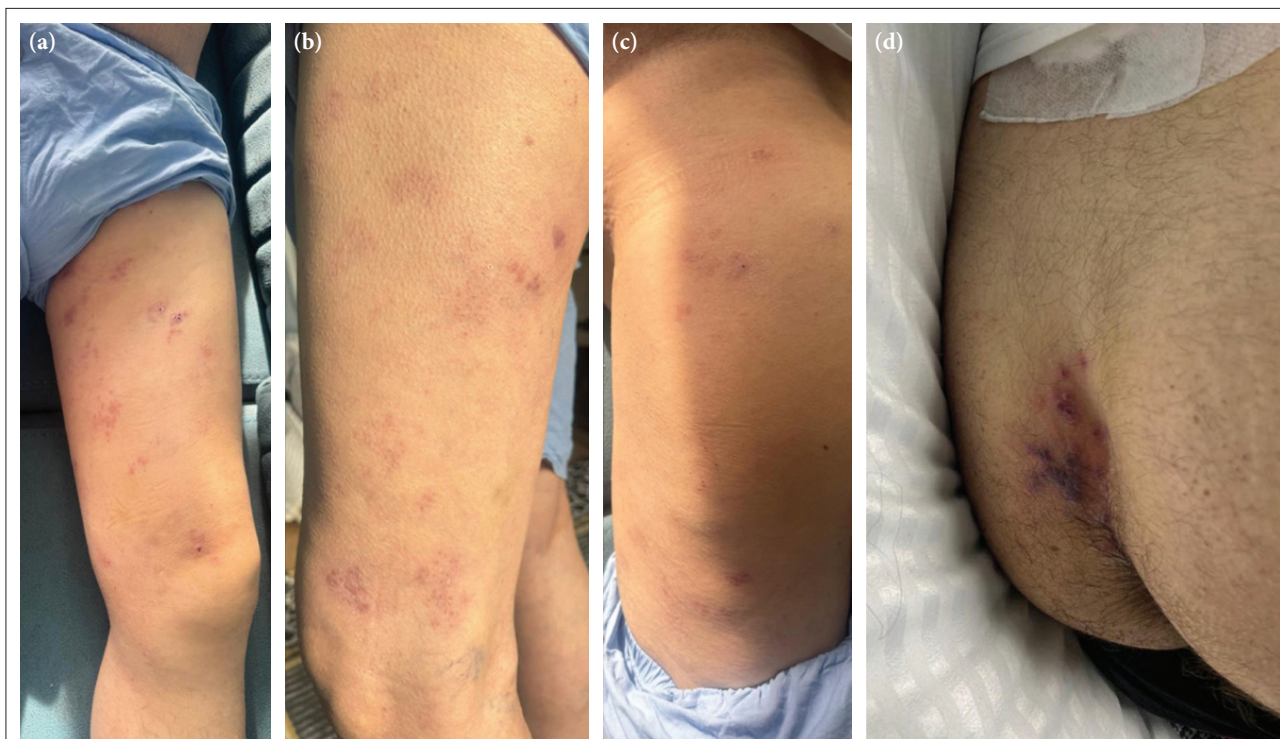


Figure 1. (a–c) Case 1: A 68-year-old man presenting with vesicular lesions involving the medial and lateral aspects of the left thigh and the ipsilateral buttock after approximately one week of oral acyclovir treatment. **(d) Case 2:** A 47-year-old man with sacral vesicular lesions that remained unrecognized until hospital admission following a five-day history of headache. The dressing visible above the lesions corresponds to the lumbar puncture site.

preserved consciousness with neck stiffness, whereas Kernig's and Brudzinski's signs were absent. Vesicular lesions were observed in the sacral S2–S3 dermatomes (Fig. 1d). Laboratory investigations showed a WBC count of 14,400/ μ L and a CRP level of 2.3 mg/L. HIV testing was negative. Brain magnetic resonance (MR) imaging, including diffusion-weighted and contrast-enhanced sequences, MR venography, and fundoscopic examination were unremarkable. CSF analysis revealed an opening pressure of 25 cm H₂O, a protein concentration of 236 mg/dL, a glucose level of 57 mg/dL (serum glucose: 113 mg/dL), and 187 lymphomononuclear cells/mm³. Cerebrospinal fluid cultures were negative. Multiplex PCR testing confirmed varicella-zoster virus infection, establishing the diagnosis of VZV meningitis associated with S2–S3 shingles. The patient was treated with intravenous acyclovir (750 mg three times daily) for 10 days and recovered completely.

The typical clinical presentation of herpes zoster consists of pain accompanied by a vesicular rash, most commonly involving the thoracic and upper lumbar dermatomes (1, 2). Sacral and lower lumbar involvement is relatively uncommon, accounting

for approximately 4–8% of cases (1–3). Herpes zoster involving the cranial and cervical dermatomes (e.g., trigeminal, head, and neck dermatomes) is associated with a higher risk of CNS complications than thoracic involvement (1, 2). Motor and autonomic complications, such as paresis or urinary retention, are uncommon overall but occur more frequently in sacral dermatomes (1–3). In both of our patients, lower lumbar or sacral involvement was present without significant motor or autonomic sequelae.

Multidermatomal herpes zoster is also an uncommon presentation that often suggests an underlying immunosuppressive state, although it may occur in immunocompetent individuals as well (3, 4). In our first patient, involvement of multiple adjacent dermatomes was observed in the setting of significant immune dysfunction.

Varicella-zoster virus meningitis is more commonly reported in younger individuals, whereas VZV encephalitis occurs more frequently in older adults (5, 6). Our cases were consistent with this age-related pattern. Delayed initiation of antiviral therapy has been identified as an important risk factor for CNS involvement in herpes zoster (1). Chronic kidney disease

and hemodialysis are known to impair immune function through mechanisms related to uremia and chronic inflammation, thereby increasing susceptibility to infections (7). Consequently, although our first patient was not receiving conventional immunosuppressive therapy, he was immunocompromised because of end-stage renal disease and long-term hemodialysis. CSF findings in VZV central nervous system infection typically include lymphocytic pleocytosis, elevated protein concentrations, and normal or mildly decreased glucose levels, although atypical presentations and cases without rash have also been reported (8-10). In both patients, the diagnosis was established by CSF PCR testing despite normal neuroimaging and EEG findings (11). Although asymptomatic detection of VZV DNA in the CSF has been reported in both immunocompetent and HIV-positive individuals (11), the clinical presentation and complete response to therapy strongly support true CNS involvement in our cases.

These cases highlight that herpes zoster involving uncommon dermatomes, such as the lower lumbar and sacral regions, may be complicated by serious CNS manifestations, even in the absence of characteristic neuroimaging abnormalities. Clinicians should maintain a high index of suspicion for VZV infection in patients presenting with atypical headaches or unexplained neurological symptoms in the setting of herpes zoster. Prompt CSF PCR testing and early initiation of antiviral therapy remain essential to achieving favorable outcomes, particularly in patients with impaired immune function.

Informed Consent: Written informed consents were obtained from patients who participated in this study.

Conflict of Interest: The authors declare that there is no conflict of interest.

Financial Disclosure: The authors declared that this study has received no financial support.

Use of AI for Writing Assistance: The authors declared that artificial intelligence was not used in the study.

Peer-review: Externally peer-reviewed.

REFERENCES

1. Wang J, Yuan Y, Liu H, Zhang Y, Yan Y. Prospective analysis of central nervous system infection risks in varicella-zoster virus reactivation cases: a single-center prospective study of 1030 cases. *Med Sci Monit* 2024; 30:e945835. [\[CrossRef\]](#)
2. Skripuletz T, Pars K, Schulte A, Schwenkenbecher P, Yildiz Ö, Ganzenmueller T, et al. Varicella zoster virus infections in neurological patients: a clinical study. *BMC Infect Dis* 2018; 18:238. [\[CrossRef\]](#)
3. Alsulaiman OA, Alsaati AA, Alsulaiman F, Aljaafari D, Alabdali M. Herpes zoster of the sacral region without motor dysfunction in a 55-year-old female: a case report and literature review. *Int Med Case Rep J* 2025; 18:677-682. [\[CrossRef\]](#)
4. Alhayyas M, Chaudhry M, Berdouk S. An atypical presentation of multidermatomal herpes zoster: a case report. *Int J Emerg Med* 2020; 13:58. [\[CrossRef\]](#)
5. Tyrberg T, Hagberg L, Nilsson S, Grahn A. Incidence and risk factors for varicella-zoster virus-associated central nervous system infections: a nationwide Swedish retrospective case-control study. *J Med Virol* 2025; 97:e70166. [\[CrossRef\]](#)
6. Lam C, Chen E, Thevathasan A, Yan T, Moso M, Sasadeusz J, Muhi S. Clinical characteristics and treatment of varicella zoster virus central nervous system infection in an Australian tertiary hospital. *Intern Med J* 2025; 55:1152-1160. [\[CrossRef\]](#)
7. Song Z, Tsou S, Martin F, Kayumov M, Xiao Y, Zhou H, et al. Kidney disease as a driver of immunosenescence: mechanisms and potential interventions. *J Am Soc Nephrol* 2026; 37:405-416. [\[CrossRef\]](#)
8. Inamoto A, Taniguchi T, Fujii Y, Miyoshi S. Varicella-zoster virus meningitis with hypoglycorrhachia, presenting with painless occipital herpes zoster mimicking atopic dermatitis. *BMJ Case Rep* 2025; 18:e258230. [\[CrossRef\]](#)
9. Best S, Mustafa G, Daly E. Varicella Zoster meningitis presenting in an Immunocompetent adolescent. *Ir Med J* 2025; 118:25.
10. Uchi T, Konno S, Inoue N, Kihara H, Sugimoto H. Unveiling the masked culprit: central nervous system infection with varicella-zoster virus diagnosed by multiplex PCR in an elderly patient with atypical neurological symptoms. *Cureus* 2024; 16:e71631. [\[CrossRef\]](#)
11. Birlea M, Arendt G, Orhan E, Schmid DS, Bellini WJ, Schmidt C, et al. Subclinical reactivation of varicella zoster virus in all stages of HIV infection. *J Neurol Sci* 2011; 304:22-24. [\[CrossRef\]](#)