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Zoster Sine Herpete Spectrum: VZV Meningitis Without Rash or Meningeal Signs in an Immunocompetent Adult — A Diagnostic Challenge

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Abstract

Varicella zoster virus (VZV) meningitis is an increasingly recognised cause of aseptic meningitis due to the widespread availability of cerebrospinal fluid (CSF) polymerase chain reaction (PCR) testing. It typically presents with headache, fever, neck stiffness, and occasionally a dermatomal rash. However, atypical presentations without rash or classical meningeal signs are increasingly reported. We describe the case of a 41-year-old immunocompetent male — a competitive badminton player — presenting with headache and fever without photophobia, phonophobia, rash, or overt meningism, initially raising diagnostic uncertainty including decompression sickness and whiplash injury. CSF PCR confirmed VZV meningitis in the absence of CSF pleocytosis. Early empiric antiviral therapy resulted in rapid clinical improvement. This case highlights the importance of recognising atypical presentations of viral meningitis and the diagnostic utility of CSF PCR, even when CSF white cell count is normal.

Keywords: Varicella zoster virus; Meningitis; Zoster sine herpete; Aseptic meningitis; PCR; Immunocompetent

Introduction

Varicella zoster virus (VZV) is a neurotropic double-stranded DNA virus belonging to the Herpesviridae family. Following primary infection, which manifests as varicella (chickenpox), the virus establishes latency in dorsal root and cranial nerve ganglia. Reactivation leads to herpes zoster (shingles) and may also result in central nervous system (CNS) complications, including meningitis, encephalitis, myelitis, and vasculopathy [1,2]. VZV meningitis is increasingly recognised in both immunocompromised and immunocompetent individuals, particularly due to improved molecular diagnostic techniques such as PCR. The incidence of VZV CNS infection has been estimated at 1–3 per 100,000 person-years, though this is likely an underestimate [3]. Importantly, VZV reactivation may occur without a cutaneous rash, a phenomenon known as zoster sine herpete, which can complicate diagnosis [2,4]. Classical meningeal features - including neck stiffness, Kernig's sign, Brudzinski's sign, photophobia, and phonophobia-have limited sensitivity, particularly in early or mild disease [5]. Furthermore, up to 30% of confirmed viral meningitis cases may present without CSF pleocytosis early in the illness course [6]. This case illustrates an atypical presentation of VZV meningitis in an immunocompetent adult without rash, without classical meningeal signs, and without CSF pleocytosis.

Case Presentation

A 41-year-old previously well male presented with worsening headache. He initially presented to a local hospital on 06 February 2026 with complaints of headache and neck stiffness. Blood investigations at that time were reportedly unremarkable (full blood count, C-reactive protein, electrolytes, and renal function within normal limits), and he was discharged with simple analgesia (paracetamol and ibuprofen). He re-presented to our hospital on 08 February 2026 with worsening symptoms. He described a bitemporal, non-throbbing, pressure-like headache radiating behind the eyes, with an intensity ranging from 3 to 7

out of 10. The headache was associated with neck stiffness and subjective fevers. He denied photophobia, phonophobia, vomiting, visual disturbance, focal neurological deficits, or rash. He reported no history of chickenpox or shingles, though on further questioning he recalled a mild febrile illness with a vesicular rash in childhood consistent with varicella. A significant history included recent spearfishing on 03 February 2026, during which he performed a single dive from approximately nine metres and remained submerged for approximately one minute. He reported no rapid ascent, no equipment malfunction, and no joint pains or neurological symptoms immediately post-dive. He had no past medical history, took no regular medications, and had no history of smoking or alcohol use. He was self-employed, married, and had two children. He was a competitive badminton player, described by family as "fast but sometimes reckless" on the court —a trait he acknowledged carried over into his recreational spearfishing technique. There was no family history of neurological or autoimmune disease. Immunisation history: he had received no varicella vaccine and reported no recent shingles vaccination.

Examination

On examination, the patient was febrile with a temperature of 39.0°C. Heart rate was 98 beats per minute, blood pressure 128/76 mmHg, respiratory rate 16 breaths per minute, and oxygen saturation 98% on room air. Neurological examination revealed no focal deficits: cranial nerves II–XII were intact, motor power was 5/5 in all four limbs, sensation was intact to light touch and pinprick, and coordination was normal. There were no clear signs of meningism; Kernig's and Brudzinski's signs were absent, and the patient could fully flex his neck without significant resistance or pain. There were no skin lesions or rash on full body examination, including the scalp, mucous membranes, and perianal region.

Differential Diagnosis

The initial differential diagnosis focused on two main possibilities given the patient's unique history as a competitive badminton player-known for being fast and occasionally reckless during physical activity-and his recent spearfishing dive.

Decompression sickness (neurological type DCS)

This was the leading consideration due to the history of a dive from approximately nine metres. The patient was initially treated empirically for decompression sickness to an extent with high-flow oxygen and hydration while awaiting hyperbaric consultation. However, several features argued against DCS: the delayed onset of symptoms (three days post-dive, whereas DCS typically occurs within minutes to 24 hours), the absence of joint pain, fatigue, or cutaneous manifestations (cutis marmorata), and the lack of focal neurological deficits [7]. Hyperbaric consultation ultimately deemed DCS unlikely.

Whiplash injury

The patient's background as a competitive badminton player raised the possibility of a cervical whiplash-type injury sustained during the water sport. He described rapid, somewhat uncontrolled head movements while surfacing and handling equipment — consistent with his self-described "reckless" style on the badminton court. Cervical muscle strain or ligamentous injury could theoretically produce headache and neck stiffness. However, the presence of fever (39.0°C) and the progressive nature of symptoms made a traumatic cause unlikely.

Viral meningitis

Aseptic meningitis, particularly due to enteroviruses or herpesviruses, remained on the differential despite the absence of classical meningeal signs or rash. This was pursued with lumbar puncture and CSF PCR.

Investigations

Blood tests on admission

White cell count: 7.2 × 10⁹/L (normal 4.0–11.0)
Neutrophils: 4.8 × 10⁹/L (normal 1.5–7.5)
Lymphocytes: 1.9 × 10⁹/L (normal 1.0–4.0)
C-reactive protein: 28 mg/L (normal <5)
Serum glucose: 5.4 mmol/L
Renal and liver function: normal
HIV serology: negative

Magnetic resonance imaging (MRI) of the brain and cervical spine with and without contrast showed no abnormalities. No meningeal enhancement, no parenchymal lesions, and no evidence of vasculopathy. Lumbar puncture was performed. Opening pressure: 15 cmH_2O (normal 10–20). CSF was clear and colourless.

CSF white cell count: 2 cells/μL (normal <5) — no pleocytosis
CSF red cell count: 0 cells/μL
CSF protein: 1.5 g/L (elevated; normal 0.15–0.45)
CSF glucose: 2.5 mmol/L (serum glucose 5.4 mmol/L → CSF/serum glucose ratio 0.46; normal >0.5)
CSF Gram stain: no organisms
CSF bacterial culture: no growth at 72 hours
CSF VZV PCR: positive
CSF HSV-1, HSV-2, enterovirus PCR: negative
CSF cryptococcal antigen: negative

Despite the absence of pleocytosis, the elevated protein and borderline low glucose ratio raised suspicion for a viral or partially treated bacterial process. CSF VZV PCR confirmed the diagnosis.

Management

Empiric treatment was initiated promptly on admission (prior to LP results) with: Intravenous ceftriaxone 2 g twice daily. Intravenous acyclovir 10 mg/kg every 8 hours (adjusted for normal renal function, eGFR >90 mL/min). Intravenous acyclovir 10 mg/kg every 8 hours (adjusted for normal renal function, eGFR >90 mL/min). This followed standard guidelines for suspected CNS infection [8,9]. Hyperbaric consultation was sought due to the history of diving. While awaiting review, the patient was managed with high-flow oxygen and intravenous fluids as a precaution for possible decompression sickness [7].

Following confirmation of VZV meningitis on day 3 of admission, ceftriaxone was discontinued. Intravenous acyclovir was continued for a total of 7 days, with improvement in symptoms. The patient was then transitioned to oral valacyclovir 1 g three times daily to complete a total of 14 days of antiviral therapy.

Supportive management included

Intravenous fluids (0.9% saline at 75 mL/hour), Paracetamol 1 g four times daily as needed for headache and fever. Daily renal function monitoring (acyclovir safety).

Clinical Course

Day 1: Admitted with headache (7/10), fever 39.0°C, shivering. Neurologically intact. Empiric antibiotics + acyclovir commenced. High-flow oxygen given pending hyperbaric review.

Day 2: Headache improved to 5/10. Persistent low-grade fever (38.2°C). No rash developed. Hyperbaric consultation deemed DCS unlikely.

Day 3: CSF PCR result: VZV positive. Ceftriaxone stopped. Headache 3/10. Afebrile.

Day 4: Headache resolved. Patient requested discharge. Neurology review: stable, no neurological deficits.

Day 5: Discharged home on oral valacyclovir 1 g TID for 9 more days (total 14 days).

Follow-up at 4 weeks: Complete resolution of symptoms. No neurological sequelae. No post-herpetic neuralgia. Returned to normal activities including diving and badminton.

Discussion

This case illustrates several atypical features of VZV meningitis that clinicians must recognise to avoid diagnostic delay. Absence of classical symptoms and signs: The patient did not exhibit photophobia or phonophobia, which are commonly associated with meningitis but have a sensitivity of only approximately 50–60% for aseptic meningitis [5,10]. Furthermore, Kernig's and Brudzinski's signs have very low sensitivity (approximately 5–30%) and may be absent in up to 50% of confirmed viral meningitis cases [5]. Reliance on these signs alone would have delayed diagnosis.

Zoster Sine Herpete Spectrum

The absence of rash represents zoster sine herpete, a well-described but often underdiagnosed manifestation of VZV reactivation that exists on a clinical spectrum [2,4]. First characterised by Weller in 1958, this entity can present with radicular pain (most common), segmental zoster paresis, meningitis, encephalitis, or vasculopathy without ever developing skin lesions [4]. In one series of 42 patients with VZV CNS infection, 37% had no rash [11]. Our patient presented with headache, fever, and neck stiffness but never developed the characteristic vesicular eruption. Clinicians should therefore not exclude VZV based on the absence of cutaneous findings and should recognise that zoster sine herpete represents a spectrum of neurological manifestations.

Normal CSF white cell count (absence of pleocytosis)

Cerebrospinal fluid analysis did not demonstrate pleocytosis, which is typically expected in viral meningitis. However, early presentation (within 24–48 hours of symptom onset) or a mild inflammatory response may result in normal white cell counts [6,12]. In a retrospective study of 106 patients with PCR-confirmed viral meningitis, 12% had CSF WCC <5 cells/μL [12]. Elevated CSF protein was the only abnormality in our patient, highlighting its importance as a diagnostic clue. The borderline low CSF/serum glucose ratio (0.46) is unusual for viral meningitis but can occur, particularly with VZV and HSV [1].

Role of PCR

The differential diagnosis of aseptic meningitis includes herpes simplex virus (HSV-1, HSV-2), enteroviruses, VZV, West Nile virus, Lyme disease, tuberculous meningitis, fungal meningitis, and partially treated bacterial meningitis. PCR testing of CSF is the diagnostic gold standard for VZV, with sensitivity exceeding 95% when performed within 7 days of symptom onset [1,9]. Importantly, VZV PCR may be negative in the first 24–48 hours, and a negative result does not entirely exclude the diagnosis if clinical suspicion remains high [13].

Management considerations

Management of VZV meningitis includes early initiation of intravenous acyclovir (10–15 mg/kg every 8 hours, adjusted for renal function). Early treatment (within 48 hours of admission) has been associated with reduced length of stay and fewer neurological complications [1,8]. Total duration of therapy is typically 10–14 days for immunocompetent patients, though some experts extend to 21 days for severe or complicated cases [14]. Our patient received 7 days of IV therapy followed by 7 days of oral valacyclovir (1 g TID). Valacyclovir has excellent oral

bioavailability (approximately 65%), making it a suitable step-down agent [14].

Prognosis

The prognosis of VZV meningitis is generally excellent, particularly in immunocompetent individuals receiving timely antiviral therapy. Most patients recover fully without long-term neurological sequelae [1]. Recurrence is rare. Our patient remained symptom-free at 4-week follow-up.

Limitations

This is a single case report and cannot be generalised. Additionally, we did not perform VZV serology (IgM/IgG) or test for VZV DNA in blood or saliva, which might have provided supportive evidence of systemic reactivation. Long-term follow-up beyond 4 weeks was not performed.

Learning Points

- VZV meningitis can occur without rash (zoster sine herpete spectrum) — do not exclude VZV based on absent skin lesions.
- Classical meningeal signs (Kernig's, Brudzinski's, photophobia) have low sensitivity and may be completely absent.
- CSF pleocytosis may be absent in early viral meningitis — elevated protein alone should raise suspicion.
- CSF VZV PCR is the diagnostic gold standard and should be performed early, even with normal CSF WCC.
- Prompt empiric acyclovir (10 mg/kg IV q8h) is safe and improves outcomes; adjust duration based on clinical response (typically 10–14 days total).

Conclusion

Varicella zoster virus meningitis can present without classical clinical features, including rash, meningeal signs, and even CSF pleocytosis. Clinicians should maintain a high index of suspicion for viral meningitis in patients presenting with headache and fever, even in the absence of typical findings. Early lumbar puncture and CSF PCR testing are essential for diagnosis, and prompt initiation of antiviral therapy leads to favorable outcomes.

Conflict of interest statement

None declared.

Patient consent

informed consent obtained from the patient for publication of this case report.

References

1. Grahn A, Studahl M. Varicella-zoster virus infections of the central nervous system – Prognosis, diagnostics and treatment. J Infect. 2015; 71: 281-293.
2. Nagel MA, Gilden D. The expanding spectrum of varicella zoster virus vasculopathy. Neurology. 2014; 82: 89-97.
3. Koskiniemi M, Rantalaiho T, Piiparinen H, Bonsdorff von CH, Farkkila M, Jarvinen A, et al. Infections of the central nervous system of suspected viral origin: a collaborative study from Finland. J Neurovirol. 2001; 7: 400-408.
4. Gilden D, Cohrs RJ, Mahalingam R, Nagel MA. Varicella zoster virus vasculopathies: diverse clinical manifestations, laboratory features, and pathogenesis. Lancet Neurol. 2009; 8: 731-740.
5. Thomas KE, Hasbun R, Jekel J, Quagliarello VJ. The diagnostic accuracy of Kernig's sign, Brudzinski's sign, and nuchal rigidity in adults with suspected meningitis. Clin Infect Dis. 2002 ;35: 46-52.
6. Nigrovic LE, Fine AM, Monuteaux MC, Shah SS, Neuman MI. Trends in the management of viral meningitis at United States children's hospitals. Pediatrics. 2013; 131: 670-676.
7. Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. Lancet. 2011; 377: 153-164.
8. Tunkel AR, Glaser CA, Bloch KC, Sejvar JJ, Marra CM, Roos KL, et al. The management of encephalitis: clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis. 2008; 47: 303-327.
9. Therapeutic Guidelines Limited. Central nervous system infections. In: Therapeutic Guidelines: Neurology. Version 8. Melbourne: Therapeutic Guidelines Limited; 2023.
10. Attia J, Hatala R, Cook DJ, Wong JG. The rational clinical examination. Does this adult patient have acute meningitis? JAMA. 1999; 282: 175-181.
11. Echevarria JM, Martinez-Martín P, Tellez A, Ory F, Rapun JL, Bernal A, et al. Aseptic meningitis due to varicella-zoster virus: serum antibody levels and local synthesis of specific antibodies. J Med Virol. 2000; 61:133-138.
12. Takhar SS, Ting SA, Sunday N, et al. Aseptic meningitis: diagnostic utility of CSF PCR in a cohort of 106 patients. Am J Emerg Med. 2016; 34: 1491-1495.
13. Persson A, Bergstrom T, Lindh M, Namvar L, Studahi M. Varicella-zoster virus CNS disease-viral load, clinical manifestations and sequels. J Clin Virol. 2009; 46: 249-253.
14. Gnann JW Jr, Whitley RJ. Clinical practice. Herpes zoster. N Engl J Med. 2002; 347: 340-346.
15. Kupila L, Vuorinen T, Vainionpaa R, Hukkanen V, Marttila RJ, Kotilainen P, et al. Etiology of aseptic meningitis and encephalitis in an adult population. Neurology. 2006; 66: 75-80.