Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Varicella-Zoster Virus Encephalitis Mimicking Acute Stroke: A Case Report

Felix Owusu Osae

felix.o.osae@gmail.com

Komfo Anokye Teaching Hospital

Emmanuel Ansah Owusu

Komfo Anokye Teaching Hospital

Francis Agyapong

Komfo Anokye Teaching Hospital

Amos Polibu

Komfo Anokye Teaching Hospital

Case Report

Keywords: Varicella-zoster virus, Encephalitis, Right-sided weakness, Seizures

Posted Date: June 28th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9740259/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Version of Record: A version of this preprint was published at Journal of Medical Case Reports on September 9th, 2026. See the published version at https://doi.org/10.1186/s13256-026-06559-x.

Abstract

Background

Varicella zoster virus encephalitis (VZV), although rare, is commonly seen among the paediatric population and immunocompromised adults. It is often misdiagnosed due to its arsenal of myriad presentations mimicking other disease conditions. Late diagnosis with an attending delayed intervention often yields bad outcomes, with death being the most feared complication among affected individuals. In view of that, a high index of suspicion is imperative for early diagnosis and timely intervention.

Case Presentation:

We present a 16-year-old Ghanaian female of Akan ethnicity who presented with a 3-day history of recurrent generalized tonic-clonic seizures and confusion associated with right-sided weakness and persistent drooling, but no prior history of febrile illness, neck stiffness, headache, photophobia, or sick contact with chicken pox or herpes simplex infection. Glasgow Coma Scale (GCS) was 14/15 (Eye – 4, Verbal – 4, Motor – 6), pupils reacted equally to light, neck was supple with a right lateralizing sign; power was 1/5 in both upper and lower limbs and right plantar response was extensor. Other systemic examinations were unremarkable. All laboratory investigations, including brain MRI were normal. However, the CSF Viral panel RT-PCR was positive for Varicella zoster virus.

Management and Outcome:

A diagnosis of VZV encephalitis complicated by right hemiparesis was made. She was then treated with levetiracetam, acyclovir, dexamethasone, and paracetamol, resulting in complete resolution of her symptoms at day 10 on admission.

Conclusion

Viral encephalitis should be considered in patients presenting with vague neurological symptoms, with no focus, especially in low-resourced areas where PCR is unavailable. Rapid recognition and prompt treatment with antiviral agents can lead to complete recovery even in patients with protracted disease, ameliorating the debilitating complications associated with VZV encephalitis, thereby reducing mortality and morbidity.

Introduction

Varicella-zoster virus (VZV) encephalitis is a noticeable complications of VZV reactivation that presents with diverse clinical presentations across the different age groups(1). Among children, it presents as cerebellar ataxia, with adults typically presenting with cranial neuropathies, like Ramsay-hunt syndrome

and optic neuritis (2), seizures and other neurological symptoms(1, 3). This is because varicella-zoster virus (VZV) is a neurotropic alpha-herpes virus that causes varicella (chicken pox) as its primary infection(4) which then establishes latency in the dorsal root ganglia, cranial nerve ganglia and autonomic ganglia and mostly gets reactivated in about 10–30% of affected individuals(5) with adult-onset encephalitis predominantly affecting the aged and immunocompromised individuals (6).

A study conducted in the United States reported that about 1/400 children develop VZV encephalitis, with children who are immunocompromised, such as children with leukaemia and other malignancies, those receiving chemotherapy, and individuals with advanced HIV infection mostly developed disseminated VZV infection, culminating in fatal outcomes such as death(4). Conversely, a systematic study done in Africa recorded a high VZV seroprevalence rate among the adults participants. However, there was a dearth of information about the seroprevalence rate of VZV among the paediatric participants involved in their study(7). Similarly, another study conducted in Columbia, US opined that the incidence of VZV encephalitis increases with increasing age owing to the immunosenescence phenomenon in the elderly.

Various studies conducted worldwide have observed that, immunosuppression conditions such as old age, diabetes mellitus, malignancy, advanced HIV infection and immunosuppressive therapies are the established risk factors for VZV encephalitis. These factors are associated with declined T-cell mediated immunity, increasing susceptibility to viral infections.(3)(8). However, it is also noted that VZV encephalitis can occur in immunocompetent subjects of which the actual mechanism has not been fully elucidated.

Notably among the neurological complications of VZV infection is post VZV stroke owing to the vasculotropic interactions and neurotropism potential of the virus(9, 10) with various epidemiological studies revealing that there is high risk of stroke occurrence after zoster infection(11). In milieu of that, works done in China and Denmark noted that in some cases of VZV encephalitis, there is intracranial haemorrhagic transformation due to the vasculopathy caused by the virus. They further stated that this mostly exacerbates the condition, posing life-threatening complications such as death in about 9–20% of affected individuals(12, 13). Also, a case report involving a 42-year-old male with 3-days history of shingles was noticed to have elevated protein levels and white cell count (predominantly lymphocytes) in CSF analysis. MRI showed hyperdense lesion in the right frontal and temporal lobes which was in keeping with VZV encephalitis(14). Also, a case series by involving 4 patients aged 38–63 years who were on immunosuppressive therapy with virology confirmed VZV infection observed that the interval between the onset of herpetic rash development and the occurrence of stroke was 13–122 days. Also, neuroimaging done showed cerebral infarctions in ACA and PCA circulation territories(15).

Various studies conducted worldwide in relation to VZV encephalitis and stroke have revealed that, the onset of stroke mostly occur after VZV infection with an average interval of about 8 weeks following infection. Also, majority of the affected individuals who had stroke post VZV encephalitis presented with pathognomonic features of VZV infection with radiological confirmation of both haemorrhagic and

ischemic stroke(15–17). However, there is paucity of information regarding acute VZV encephalitis presenting with stroke-like features in the absence of prodromal symptoms and unremarkable neuroimaging findings. This atypical presentation poses a significant diagnostic challenge and may lead to delayed recognition and treatment, particularly when clinicians rely heavily on neuroimaging. Also, it underscores the importance of early CSF viral analysis in suspected cases. We therefore present a case of a 16-year-old female who presented with right-sided weakness with a 3-day prior history of recurrent generalized tonic-clonic seizures and confusion with normal MRI but positive CSF Viral panel for VZV.

Case Presentation

A 16-year-old Ghanaian female of Akan ethnicity, presented with 3-day history of acute behavioural changes which included disinhibition, excessive speech and incoherent verbalization. 12 hours later, she developed generalized tonic-clonic seizure associated with hypersalivation but no urine or faecal incontinence, focal onset features or tongue biting. The first episode lasted for about 30 seconds after which she regained baseline consciousness. However, she had recurrent episodes over the next 48hrs and subsequently developed a right sided weakness on the 3rd day.

There was no prior history of seizures, head trauma, febrile illness, neck stiffness, photophobia, or contact with individuals with varicella or herpes simplex infection. She had no history of chronic medical conditions, including HIV infection, diabetes mellitus, sickle cell disease, malignancy, or prior central nervous system infections. There was no history of recent medication use, including steroids, antimalarials, or herbal preparations or illicit drug use. Immunizations were up to date, with no recent vaccinations. Birth, developmental, and family histories were unremarkable

On examination, she was ill-looking, afebrile (36.4°C), well hydrated, and intermittently confused but alert, with a Glasgow Coma Scale score of 14/15 (E4V4M6). Initial vital signs were; blood pressure 132/83 mmHg, heart rate 63 beats per minute, respiratory rate 22 cycles per minute, and oxygen saturation 97% on room air. She subsequently developed a fever (39.2°C).

Neurological examination revealed normal cranial nerve function and no signs of meningeal inflammation. Motor examination demonstrated reduced tone and power (1/5) in the right upper and lower limbs, with preserved strength (5/5) on the left. Deep tendon reflexes were brisk (3+) on the right, with an extensor plantar response, while the left limbs physical examination was normal.. Sensory examination was intact bilaterally.

Examination of cardiovascular, respiratory, and abdominal systems was unremarkable. No cutaneous stigmata of viral infection or hemorrhage were identified. Investigations done are enlisted in Table 1a and b below.

Table 1
a: Haematological and Biochemical Investigations

Test	Test Value	Reference Range
Full Blood Count		
RBC count ($\times 10^6$/μL)	4.41	3.85–5.20
Haemoglobin (g/dL)	12.4	12.0–15.0
Hematocrit (%)	35.9	34.7–45.0
MCV (fL)	81.4	78.0–95.0
MCH (pg)	28.1	26.0–32.0
RDW-CV (%)	13.6	11.5–14.0
Platelet Count ($\times 10^3$/μL)	365	140–440
WBC count ($\times 10^3$/μL)	5.53	4.00–10.00
ANC ($\times 10^3$/μL)	2.76	1.50–8.00
Neut %	49.8	37.0–75.0
Lymph Count ($\times 10^3$/μL)	2.06	0.80–4.00
Lymph%	37.3	16.00–51.00
Mono Count ($\times 10^3$/μL)	0.44	0.00–1.20
Mono%	8.0	0.00–12.0
Blood film for malaria	No malaria parasites seen	
Sickling Test	Negative	
HB electrophoresis	Genotype – A	
Inflammatory Markers		
ESR (mmfall/hr)	3.0	0.00–20.00
CRP (mg/L)	< 1.0	0.00–10.0
Procalcitonin (ng/mL)	< 1.0	0.000–0.150
Renal Function Test		
Urea (mmol/L)	2.45	2.10–8.30
Creatinine (μmol/L)	59.0	44.0–80.0
BUN/Creatinine	19.4	8.0–35.0

Test	Test Value	Reference Range
Electrolytes		
Sodium (mmol/L)	136	135–145
Potassium (mmol/L)	4.7	3.5–5.5
Chloride (mmol/L)	101	95–110
Total Calcium (mmol/L)	2.25	2.30–2.75
Magnesium (mmol/L)	0.69	0.65–0.90
Phosphate (mmol/L)	1.06	0.36–0.65

Table 1
b: Biochemical (Cont.) Investigations and CSF Analysis

Test	Test Value	Reference Range
Liver Function Test		
AST (U/L)	15.1	0.0–31.0
ALT (U/L)	8.8	0.0–34.0
AST/ALT Ratio	1.7	
ALP (U/L)	122.0	47.0–119.0
GGT (U/L)	16.9	12.0–71.0
Total Protein (g/L)	66.3	63.0–82.0
Albumin (g/L)	39.8	32.0–45.0
Globulin (g/L)	26.5	23.0–35.0
Total Bilirubin (μmol/L)	7.7	1.8–22.0
Direct Bilirubin (μmol/L)	2.1	0.0–5.0
Indirect Bilirubin (μmol/L)	5.6	0.0–17.0
Glycated Hemoglobin (%)	5.52	4.00–6.00
CSF Biochemistry		
Total Protein (g/L)	0.18	0.15–0.45
Chloride	126	120–130
Glucose	6.4	3.33–4.44
CSF Microbiology		
Appearance	Clear	
Colour	Colourless	
Polymorphs	0	0–2
Lymphocytes	0	0–5
Erythrocytes	0	
Culture and Sensitivity	No bacterial growth	
CSF: PCR for Viral Panel		
Herpes Simplex Virus	Negative	
Varicella-zoster Virus	Positive	

Test	Test Value	Reference Range
Enterovirus	Negative	

Brain MRI done showed no evidence of infarction, haemorrhage, mass, demyelination, abnormal enhancement or right hydrocephalous (Fig. 1).

The patient was admitted and managed as a case of acute encephalopathy with recurrent seizures and focal neurological deficit with differential diagnosis of:

1. Viral encephalitis
2. Acute disseminated encephalomyelitis
3. Intracranial space occupying lesion

With the CSF PCR for VZV being positive, a final diagnosis of VZV encephalitis was made.

Treatment

Acute seizures were controlled with intravenous benzodiazepines (Diazepam 10mg prn), followed by maintenance antiepileptic therapy with oral Levetiracetam 500mg twice daily which was continued for 3 months. In view of suspected infectious encephalitis, empiric intravenous Acyclovir 500mg twice daily and broad-spectrum antibiotics (ceftriaxone 2g twice daily) were initiated. Supportive management included intravenous fluids, antipyretics, and close neurological monitoring. The patient showed gradual clinical improvement, with reduction in seizure frequency, and recovery of consciousness. Residual right-sided weakness was addressed with physiotherapy. Seizures completely abated by day 8 of admission, and power in the right upper and lower limbs returned to 5/5 by day 10. She was subsequently discharged after completing a 14-day course of intravenous Acyclovir

Follow-up

At 2 weeks post-discharge, the patient remained clinically stable with no recurrence of seizures or behavioral disturbances. Neurological examination demonstrated complete resolution of the right-sided weakness, with normal power (5/5) in all limbs. She remained compliant with maintenance therapy, including Levetiracetam, and reported no adverse effects.

At 1-month follow-up, she continued to be seizure-free with no focal neurological deficits. Cognitive and behavioral functions had returned to baseline, and she had fully resumed normal school activities. There were no new neurological or systemic complaints.

At 3 months follow-up, the patient remained asymptomatic and neurologically intact, with no recurrence of seizures or encephalopathic features. Medication adherence remained good, and no complications of therapy were reported. Overall, she achieved complete clinical recovery with sustained remission.

Discussion

Varicella-zoster virus (VZV) encephalitis is a rare but potentially life-threatening neurological complication of VZV infection, presenting with a broad spectrum of clinical manifestations that often complicate early diagnosis. Although the disorder is primarily seen in immunocompromised individuals and older adults, there is emerging evidence that indicates that VZV encephalitis can also occur in immunocompetent children and adolescents, frequently with atypical presentations (18).

Primary varicella, a common disease of childhood, typically presents as a pruritic cutaneous vesicular rash associated with low grade fever, popularly known as chickenpox. Following primary infection, VZV goes into a state of dormancy by establishing lifelong latency within the sensory ganglia, including the dorsal root ganglia along the spinal cord, the cranial nerve ganglia particularly the trigeminal ganglia and the autonomic ganglia(18). The virus remains dormant after the initial infection for years to decades and may subsequently reactivate in the setting of immunosuppression, advance aging, illness or even stress. Reactivation presents with a broad clinical spectrum. The most common presentation is herpes zoster (shingles), characterized by a painful vesicular rash in a dermatomal distribution. However, atypical forms such as zoster sine herpete, where reactivation occurs without a cutaneous rash has been clinically described (19).

In addition, VZV reactivation may involve the central nervous system, leading to a wide range of neurological complications including encephalitis and VZV-associated vasculopathy, which may present with stroke-like features(18). This underscores the importance of maintaining a high index of suspicion in patients presenting with unexplained neurological symptoms. Although VZV encephalitis is more commonly reported in immunocompromised individuals, there is evidence that it can present in immunocompetent individuals (20). This case illustrates that it can occur in immunocompetent adolescents without identifiable risk factors. It also demonstrates several atypical features, including presentation as an acute stroke mimic, absence of cutaneous manifestations (zoster sine herpete), normal neuroimaging, and complete neurological recovery following early antiviral therapy.

The acute onset of right sided weakness in this patient initially raised suspicion for a cerebrovascular event. VZV can cause vasculitis by inducing inflammation within the cerebral arteries, increasing the risk of both ischemic and hemorrhagic stroke (21). Ischemic strokes are common in contrast to hemorrhagic strokes which usually complicate herpes simplex virus encephalitis. Following reactivation, there is a high risk of stroke particularly within the first few weeks (22). However, in contrast to classical VZV-associated vasculopathy, which is typically accompanied by radiological abnormalities, this patient had a normal magnetic resonance imaging (MRI) scan. The patient’s focal neurological deficit was initially considered in the differential diagnosis of a post-ictal Todd’s paralysis, a well-recognized stroke mimic following seizures (Libman et al., 1995). However, the prolonged duration of weakness extending beyond the typical 24–48-hour window for Todd’s paralysis makes this diagnosis unlikely. In most cases of Todd’s paralysis, the transient weakness resolves completely within hours to two days, and the persistent deficits beyond this period warrant consideration of alternative etiologies. The absence of

radiological evidence of acute infarction on magnetic resonance imaging, together with cerebrospinal fluid confirmation of varicella-zoster virus infection, supports a diagnosis of focal neurological dysfunction secondary to viral encephalitis rather than primary cerebrovascular disease. VZV encephalitis is known to produce a spectrum of neurological deficits through direct neuronal involvement and inflammatory mechanisms, which may mimic acute stroke clinically despite normal neuroimaging finding (18, 21)

The diagnosis of VZV encephalitis relies on detection of viral DNA in cerebrospinal fluid (CSF) using polymerase chain reaction (PCR), which remains the gold standard (23). However, access to PCR testing is limited in many low- and middle-income countries. A study conducted in Ghana demonstrated the utility of molecular diagnostics in identifying viral causes of central nervous system infections, including VZV, while also highlighting limited accessibility in routine clinical practice (24). In settings where PCR testing is not available, clinicians often depend on clinical judgment and may need to initiate empirical antiviral therapy when suspicion is high. Suspicion for VZV CNS infection should be high when the clinical picture does not fit a single obvious diagnosis (like stroke, malaria, or bacterial meningitis) and especially when there is a mix of neurological features like seizures, focal deficits and encephalopathy, even without rash (25). Additionally, neuroimaging findings in VZV encephalitis are variable. They may be normal, in the early stage of the disease and normal neuroimaging does not rule out the diagnosis. Studies have shown that a subset of patients with confirmed VZV encephalitis may have no detectable abnormalities on MRI, emphasizing that normal imaging does not rule out the diagnosis (26).

Acyclovir therapy remains the cornerstone of the treatment of VZV encephalitis and early initiation improves clinical outcomes and reduce mortality and morbidity (23). In this case, prompt antiviral therapy combined with anticonvulsant treatment resulted in complete neurological recovery within 10 days. There is evidence that delays in the treatment of VZV encephalitis is associated with poorer outcomes, including persistent neurological deficits and increased mortality (26). The favorable outcome observed in this patient highlights the importance of early recognition and intervention.

This case emphasizes several key clinical considerations. First, VZV encephalitis should be considered in patients presenting with acute focal neurological deficits and seizures, even in the absence of rash or typical prodromal symptoms. Second, normal neuroimaging does not exclude encephalitis, particularly in early disease. Third, in resource-limited settings, a high index of suspicion should guide early empirical antiviral therapy when diagnostic tools are unavailable. Finally, differentiation between stroke and stroke mimics such as Todd’s paralysis is essential to ensure appropriate management.

In conclusion, this case highlights an atypical presentation of VZV encephalitis as a stroke mimic in an immunocompetent adolescent with normal neuroimaging and no cutaneous manifestations. Increased clinical awareness of such presentations, particularly in low-resource settings, is essential to facilitate early diagnosis and improve patient outcomes.

Declarations

Ethics approval and consent to participate

Written informed consent for publication of this case report and any accompanying images was obtained from the patient's parent(s) or legal guardian.

Consent for publication

Written informed consent was obtained from the patient's parent(s)/legal guardian for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request. Certain data may be restricted to protect patient confidentiality and privacy.

Competing interests

The authors declare that they have no competing interests.

Funding

The authors received no specific funding for this work.

Authors' contributions

Felix Owusu Osae made the major contribution to conceptualization, investigation, data collection, literature review, methodology, and drafting of the original manuscript. He also participated extensively in manuscript revision and editing.

Emmanuel Ansah Owusu contributed to conceptualization, data curation, investigation, methodology, and manuscript review and editing.

Amos Polibu contributed to the investigation and manuscript review and editing.

Francis Agyapong supervised the study, contributed to the methodology, validation, and critically reviewed and edited the manuscript.

All authors read and approved the final manuscript.

Acknowledgements

The authors would like to acknowledge the staff of Komfo Anokye Teaching Hospital for their support and contribution toward this study.

References

1. Rodrigues F, Santos M, Macedo E, Rocha M, Silva L, Oliveira AS, Alves, I, Apolinário. Varicella-zoster virus: A case of encephalitis. cureus.com. 2023. doi:10.7759/cureus.45378
2. Gershon AA, Breuer J, Cohen JI, Cohrs RJ, Gershon MD, Gilden D, et al. Varicella zoster virus infection. Nature Reviews Disease Primers 2015 1:1. 2015 Jul 2;1(1):15016-. doi:10.1038/nrdp.2015.16 PubMed PMID: 27188665.
3. Gershon AA, Gershon MD, Breuer J, Levin MJ, Oaklander AL, Griffiths PD. Advances in the understanding of the pathogenesis and epidemiology of herpes zoster. Journal of Clinical Virology. 2010 May 1;48(SUPPL 1):S2–7. doi:10.1016/S1386-6532(10)70002-0 PubMed PMID: 20510263.
4. van Oorschot D, Vroling H, Bunge E, Diaz-Decaro J, Curran D, Yawn B. A systematic literature review of herpes zoster incidence worldwide. Taylor & FrancisD van Oorschot, H Vroling, E Bunge, J Diaz-Decaro, D Curran, B YawnHuman vaccines & immunotherapeutics, 2021•Taylor & Francis. 2021;17(6):1714–32. doi:10.1080/21645515.2020.1847582 PubMed PMID: 33651654.
5. Goswami Y, Cureus SG, 2023 undefined. Ramsay Hunt syndrome: an introduction, signs and symptoms, and treatment. cureus.comY Goswami, SS GaurkarCureus, 2023•cureus.com. 2023. doi:10.7759/cureus.33688
6. Horev A, Horev A, Gordon-Irshai A, Gordon M, Andre N, Ifergane G. Herpes zoster and long-term vascular risk: a retrospective cohort study. nature.comA Horev, A Horev, A Gordon-Irshai, M Gordon, N Andre, G IferganeScientific Reports, 2023•nature.com. 123AD;13:2364. doi:10.1038/s41598-023-29667-w
7. Hussey H, Abdullahi L, Collins J, Muloiwa R, Hussey G, Kagina B. Varicella zoster virus-associated morbidity and mortality in Africa – a systematic review. BMC Infectious Diseases 2017 17:1. 2017 Nov 14;17(1):717-. doi:10.1186/S12879-017-2815-9 PubMed PMID: 29137604.
8. Streng A, Pediatrics JLT, 2014 undefined. Fifteen years of routine childhood varicella vaccination in the United States—strong decrease in the burden of varicella disease and no negative effects on the. pmc.ncbi.nlm.nih.govA Streng, JG LieseTranslational Pediatrics, 2014•pmc.ncbi.nlm.nih.gov.
9. Nagel MA, Gilden D. The Relationship Between Herpes Zoster and Stroke. Current Neurology and Neuroscience Reports. 2015 Apr 1;15(4):16-. doi:10.1007/S11910-015-0534-4/METRICS PubMed PMID: 25712420.
10. Carey R, … VCA of I, 2017 undefined. Varicella zoster virus infection of the central nervous system–10 year experience from a tertiary hospital in South India. journals.lww.comRAB Carey, VK Chandiraseharan, A Jasper, T Sebastian, C Gujjarlamudi, S SathyendraAnnals of Indian Academy of Neurology, 2017•journals.lww.com.
11. Amlie-Lefond C, Gilden D. Varicella Zoster Virus: A Common Cause of Stroke in Children and Adults. Journal of Stroke and Cerebrovascular Diseases. 2016 Jul 1;25(7):1561–9. doi:10.1016/J.JSTROKECEREBROVASDIS.2016.03.052 PubMed PMID: 27138380.
12. Wu H, Wang R, Li Y, Sun X, Treatment JL… D and, 2022 undefined. Cerebrovascular complications after adult-onset varicella-zoster virus encephalitis in the central nervous system: a literature review.

pmc.ncbi.nlm.nih.govH Wu, R Wang, Y Li, X Sun, J Li, X BiNeuropsychiatric Disease and Treatment, 2022•pmc.ncbi.nlm.nih.gov.

13. Herlin L, Hansen K, … JBCI, 2021 undefined. Varicella zoster virus encephalitis in Denmark from 2015 to 2019—a nationwide prospective cohort study. academic.oup.comLK Herlin, KS Hansen, J Bodilsen, L Larsen, C Brandt, CØ Andersen, BR HansenClinical Infectious Diseases, 2021•academic.oup.com.
14. Li J, Lin Y, Ni L, Mei Y, Zhou Y, Jiang X, et al. Cerebral Hemorrhage in Varicella-Zoster Virus Encephalitis: A Case Study. The American Journal of Case Reports. 2025;26:e945521. doi:10.12659/AJCR.945521 PubMed PMID: 39745923.
15. Hoshino T, Toi S, Toda K, Uchiyama Y, Yoshizawa H, Iijima M, et al. Ischemic Stroke due to Virologically-Confirmed Varicella Zoster Virus Vasculopathy: A Case Series. Journal of Stroke and Cerebrovascular Diseases. 2019 Feb 1;28(2):338–43. doi:10.1016/J.JSTROKECEREBROVASDIS.2018.10.001 PubMed PMID: 30392831.
16. Lu P, Cui L, Zhang X. Stroke risk after varicella-zoster virus infection: a systematic review and meta-analysis. Springer. 2023 Aug 1;29(4):449–59. doi:10.1007/S13365-023-01144-0 PubMed PMID: 37219811.
17. Zhang Y, Luo G, Huang Y, Yu Q, Wang L, and KLJ of S, et al. Risk of stroke/transient ischemic attack or myocardial infarction with herpes zoster: a systematic review and meta-analysis. Elsevier. 2017 Mar 7;17(1). doi:10.1186/S12879-017-2278-Z PubMed PMID: 28270112.
18. Kennedy PGE, Gershon AA. Clinical features of varicella-zoster virus infection. Viruses. 2018 Nov 2;10(11). doi:10.3390/V10110609 PubMed PMID: 30400213.
19. Yang Y, Mahmood T, Siddiqui AH, Aziz MA. Zoster Sine Herpete: two unusual cases of varicella-zoster reactivation with atypical complaints of acute chest pain and severe headache. BMC Infectious Diseases 2023 23:1. 2023 Apr 18;23(1):239-. doi:10.1186/S12879-023-08093-3 PubMed PMID: 37072696.
20. Chakravorty K, Bhardwaj A, El-Houjairy J. Varicella zoster encephalitis in an immunocompetent patient. BMJ Case Rep. 2023 Dec 23;16(12). doi:10.1136/BCR-2023-257301 PubMed PMID: 38142056.
21. Nagel MA, Gilden D. Neurological complications of varicella zoster virus reactivation. Curr Opin Neurol. 2014;27(3):356–60. doi:10.1097/WCO.0000000000000092 PubMed PMID: 24792344.
22. Marra F, Ruckenstein J, Richardson K. A meta-analysis of stroke risk following herpes zoster infection. BMC Infectious Diseases 2017 17:1. 2017 Mar 7;17(1):198-. doi:10.1186/S12879-017-2278-Z PubMed PMID: 28270112.
23. 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. Clinical Infectious Diseases. 2008 Aug 1;47(3):303–27. doi:10.1086/589747 PubMed PMID: 18582201.
24. El-Duah P, Sylverken AA, Owusu M, Amoako YA, Yeboah R, Gorman R, et al. Genetic characterization of varicella-zoster and HIV-1 viruses from the cerebrospinal fluid of a co-infected encephalitic

patient, Ghana. Virology Journal 2022 19:1. 2022 Jul 26;19(1):122-. doi:10.1186/S12985-022-01854-7 PubMed PMID: 35883083.

25. Varicella Zoster Virus Encephalitis | Encephalitis International [Internet]. [cited 2026 Apr 19]. Available from: https://www.encephalitis.info/types-of-encephalitis/infectious-encephalitis/varicella-zoster-virus-encephalitis/
26. Mailles A, Stahl JP, Group behalf of the SC and the I. Infectious Encephalitis in France in 2007: A National Prospective Study. Clinical Infectious Diseases. 2009 Dec 15;49(12):1838–47. doi:10.1086/648419 PubMed PMID: 19929384.

Figures

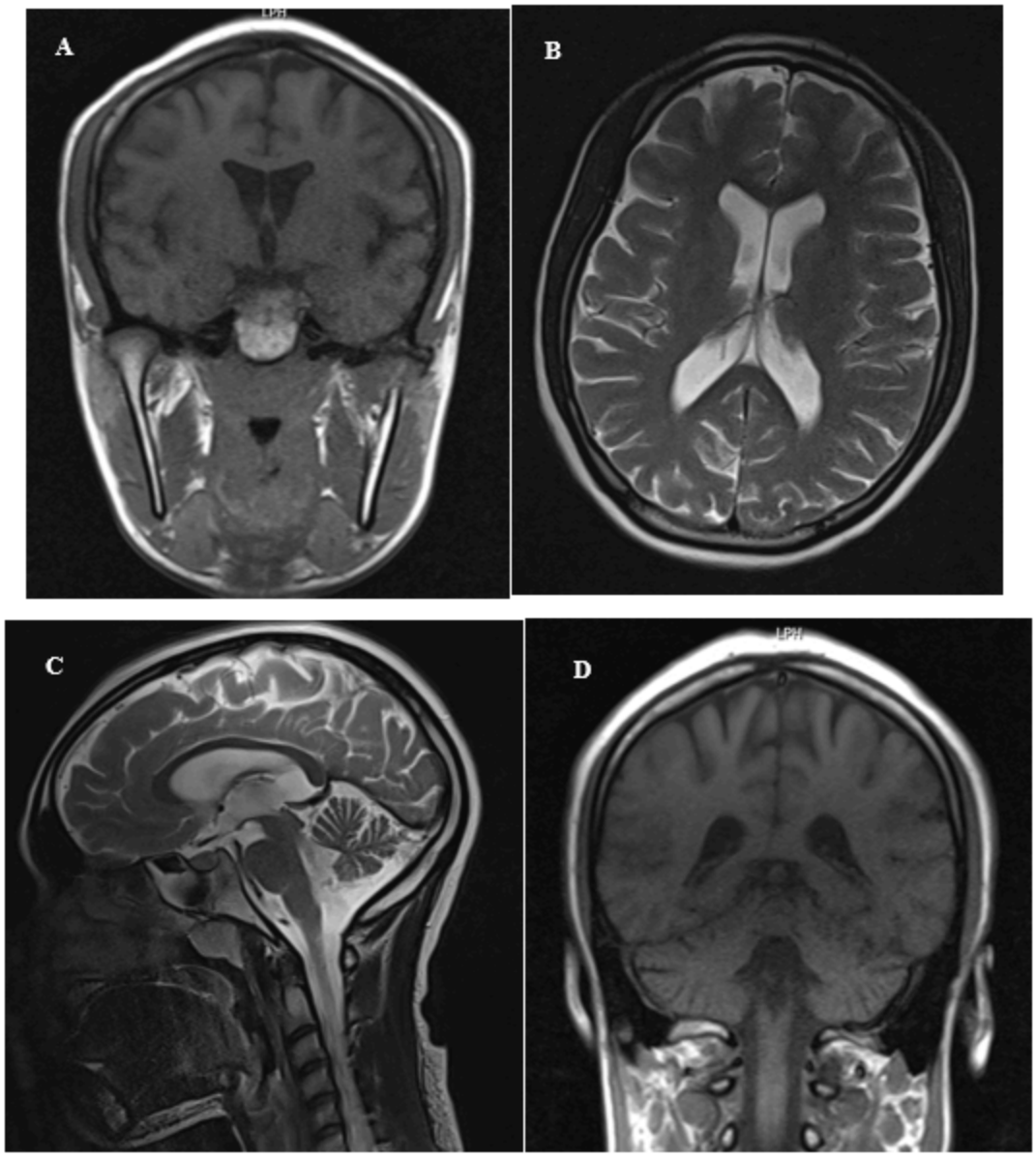


Figure 1

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- CAREChecklist.docx.docx