Cureus
Part of SPRINGER NATURE

Open Access Case Report

Parvovirus B19-Associated Meningoencephalitis in an Adult: A Rare Cause of Central Nervous System Infection

Review began 09/21/2026
Review ended 10/24/2026
Published 10/28/2026

© **Copyright** 2026
Kahwa et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118741

Tevin Kahwa [1, 2], Ayubu E. Mashambo [1, 2], Meghna V. Solanki [1, 2], Philip B. Adebayo [3, 1], Mandela Makakala [2, 1]

1. Internal Medicine, Aga Khan University, Dar es Salaam, TZA 2. Internal Medicine, Aga Khan Hospital, Dar es Salaam, Dar es Salaam, TZA 3. Neurology, Aga Khan Hospital, Dar es Salaam, Dar es Salaam, TZA

Corresponding author: Ayubu E. Mashambo, ayubu.mashambo25@alumni.aku.edu

Abstract

Parvovirus B19 (B19V) is a common childhood pathogen but a rare cause of central nervous system (CNS) disease in adults. Neurological manifestations are diverse, cerebrospinal fluid (CSF) findings are often non-specific, and interpreting a positive molecular result in CSF can be difficult.

A previously healthy 42-year-old woman presented with progressive headache, photophobia, and diplopia, and later developed generalized tonic-clonic seizures and encephalopathy. On hospital day 5, she deteriorated with reduced consciousness, requiring intubation and intensive care. Serial MRI showed progressive gyriform fluid-attenuated inversion recovery (FLAIR) hyperintensity, new bilateral thalamic, basal ganglia, and basal cistern signal change, and diffuse meningeal enhancement. CSF was acellular with elevated protein (117 mg/dL) and glucose of 30 mg/dL (1.7 mmol/L); with a paired serum glucose of 5.5 mmol/L, the CSF-to-serum glucose ratio was 0.30. A multiplex real-time viral PCR panel detected B19V DNA in CSF, with all 10 other viral targets, including herpes simplex virus types 1 and 2 (HSV-1/2), varicella-zoster virus (VZV), and enterovirus, not detected. B19V immunoglobulin M (IgM) and immunoglobulin G (IgG), tested about two weeks later, were negative. She received empirical antimicrobials, high-dose corticosteroids, a five-day course of intravenous immunoglobulin (IVIG), and supportive care, and was extubated on hospital day 16. At follow-up, she had fatigue, photophobia, a postural tremor that later improved, globally reduced reflexes, and a residual left sixth cranial nerve palsy.

This case is consistent with B19V-associated meningoencephalitis, but the discordance between CSF PCR positivity and negative serology limits diagnostic certainty. The residual deficits may reflect post-encephalitic sequelae, raised intracranial pressure, or nerve involvement, and could not be distinguished further. Recovery followed multimodal treatment, so the contribution of any single therapy cannot be determined.

B19V may be considered in adults with unexplained meningoencephalitis when routine investigations are negative. A positive CSF PCR should be interpreted alongside serology, blood PCR where available, and a thorough exclusion of alternative causes. Thus, early recognition facilitated by molecular diagnostics, combined with supportive and immunomodulatory therapy, may improve outcomes and reduce long-term neurological sequelae.

Categories: Neurology, Infectious Disease
Keywords: b19 parvovirus infection, intravenous immunoglobulin (ivig), meningoencephalitis, post encephalitic syndrome, post-encephalitic syndrome

Introduction

Human parvovirus B19 (B19V) is a small, non-enveloped single-stranded DNA virus of the *Parvoviridae* family, classically associated with erythema infectiosum, transient aplastic crisis, arthropathy, and hydrops fetalis [1]. Transmission occurs primarily via respiratory droplets, with additional parenteral and vertical routes described [2]. Seroprevalence of B19V immunoglobulin G (IgG) increases with age and may reach up to 90% in older adults, reflecting widespread cumulative exposure [3]. In Tanzania, a higher prevalence of B19V infection has been documented in high-risk populations, particularly individuals with sickle cell disease [4].

Although typically self-limiting, B19V is increasingly recognized as a multisystem pathogen with hematological and systemic manifestations, including cardiac, hepatic, renal, and neurological involvement in both immunocompetent and immunocompromised hosts [5]. Neurological manifestations include encephalitis, encephalopathy, meningitis, meningoencephalitis, cerebellitis, stroke, peripheral neuropathies, Guillain-Barré syndrome, and myelitis [6].

Despite this expanding clinical spectrum, acute meningitis and meningoencephalitis are more commonly attributed to enteroviruses, herpes simplex virus (HSV), varicella zoster virus, and bacterial pathogens such as *Listeria monocytogenes*, and B19V is not routinely considered in initial diagnostic algorithms for central nervous system (CNS) infections [7].

Over the past two decades, accumulating evidence has increasingly characterized the neurotropic potential of B19V. Systematic reviews indicate that encephalitis and encephalopathy are the most frequently reported CNS manifestations, whereas isolated meningitis remains uncommon [1]. Meta-analytic data further suggest that B19V is an under-recognized cause of CNS infection, often requiring molecular confirmation for diagnosis [8].

Published literature describing B19V-associated meningitis and meningoencephalitis remains limited to isolated case reports and small case series. Reported phenotypes include headache and rash syndromes [6], meningoencephalitis in immunocompetent adults with B19V DNA detected in cerebrospinal fluid (CSF) [9,10], and neuroinvasive disease supported by molecular and serological evidence [11].

The pathophysiological mechanisms underlying CNS involvement remain incompletely understood and are likely multifactorial [12]. Proposed mechanisms include direct viral invasion of endothelial or neural tissue, immune complex-mediated injury, cytokine-driven inflammatory damage, and NS1 protein-associated cytotoxicity [12]. Detection of viral DNA in CSF supports direct neuroinvasion in selected cases, while persistent viremia and delayed neurological manifestations suggest immune-mediated or post-infectious mechanisms [12,13].

Interpretation of CNS infection is further complicated by variability in CSF parameters, which may reduce diagnostic sensitivity in infectious encephalitis and contribute to false-negative or non-specific findings [14]. In severe viral encephalitis, immunomodulatory therapies such as intravenous immunoglobulin (IVIG) have been explored; however, evidence remains limited and largely extrapolated from other viral CNS infections [15].

We report a case of severe meningoencephalitis in an immunocompetent adult in whom B19V DNA was detected in CSF by multiplex PCR, with negative B19V serology. The case illustrates the clinical severity that may accompany B19V-associated CNS disease and the interpretive difficulties that arise when molecular and serological results are discordant.

Case Presentation

A 42-year-old previously healthy woman presented with a five-day history of progressively worsening, throbbing temporal headache radiating to the frontal region, associated with photophobia, nausea, and recurrent non-projectile vomiting. Symptoms were partially responsive to simple analgesics. She reported subjective fever and chills but denied neck stiffness, altered consciousness, seizures, focal neurological deficits, or systemic infectious symptoms. She had no prior comorbidities, did not smoke or consume alcohol, and had undergone laser-assisted in situ keratomileusis (LASIK) surgery in 2010. She was menstruating at presentation (P3L3). A recent travel history to Oman was noted, during which a brain MRI was reportedly performed and found to be normal.

On examination, she was alert, afebrile, and hemodynamically stable, with no focal neurological deficits or systemic abnormalities. Initial differentials included migraine with intercurrent febrile illness and intracranial pathology such as cavernous sinus thrombosis.

Initial investigations are summarized in Table *1*.

Investigation	Result	Reference range
D-dimer	0.77 mg/L	<0.50 mg/L
Prolactin	13.84 ng/mL	5-25 ng/mL
TSH	2.5 mIU/L	0.4-4.0 mIU/L
Hemoglobin	12.8 g/dL	12.5-14.5 g/dL
ESR	23 mm/hr	0-20 mm/hr
Vitamin B12	410 pg/mL	200-900 pg/mL
Ferritin	100 ng/mL	15-150 ng/mL
Electrolytes	Na 140 mmol/L, K 4.0 mmol/L	Na 135-145 mmol/L, K 3.5-5.0 mmol/L
HIV serology	Negative	Non-reactive
Vitamin D (25-OH)	18 ng/mL	30-100 ng/mL
Urine pregnancy test	Negative	Negative

TABLE 1: Baseline hematological, biochemical, and infectious work-up at presentation.

TSH: thyroid-stimulating hormone; ESR: erythrocyte sedimentation rate

Lumbar puncture was performed on the day of admission, before antibiotics and corticosteroids were started. Opening pressure was not measured. CSF was clear and acellular, with protein 117 mg/dL and glucose 30 mg/dL (1.7 mmol/L); the serum glucose taken at the time of lumbar puncture was 5.5 mmol/L (99 mg/dL), giving a CSF-to-serum glucose ratio of 0.30 (Table *2*). CSF was tested with the FTD Neuro9 multiplex real-time viral PCR panel (Fast Track Diagnostics, Siemens Healthineers, Luxembourg), which has been used for CSF testing in meningoencephalitis surveillance [16] and targets HSV-1, HSV-2, varicella-zoster virus (VZV), cytomegalovirus (CMV), Epstein-Barr virus (EBV), human herpesvirus 6 (HHV-6), human herpesvirus 7 (HHV-7), human adenovirus, enterovirus, human parechovirus, and B19V. B19V DNA was detected; all other targets were not detected. CSF and blood cultures, CSF GeneXpert MTB/RIF testing for *Mycobacterium tuberculosis* and rifampicin resistance, cryptococcal antigen, and HIV testing were negative. Autoimmune encephalitis antibodies and flavivirus serology were not tested.

Parameter	Result	Reference range/expected
Timing of lumbar puncture	Day of admission; before antibiotics and corticosteroids	-
Opening pressure	Not measured	10-25 cmH_2O
CSF appearance	Clear	Clear
CSF white cell count	0 cells/µL	0-5 cells/µL
CSF protein	117 mg/dL	15-45 mg/dL
CSF glucose	30 mg/dL (1.7 mmol/L)	50-80 mg/dL
Paired serum glucose	5.5 mmol/L (99 mg/dL)	-
CSF-to-serum glucose ratio	0.30	≥0.6
Multiplex viral PCR (FTD Neuro9)	B19V DNA detected	Not detected
Other FTD Neuro9 targets: HSV-1, HSV-2, VZV, CMV, EBV, HHV-6, HHV-7, HAdV, EV, HPeV	Not detected	Not detected
Serum B19V IgM	Negative (~two weeks after CSF PCR, after IVIG)	Negative
Serum B19V IgG	Negative (~two weeks after CSF PCR, after IVIG)	Variable
Serum or blood B19V PCR	Not performed	-
CSF GeneXpert MTB/RIF	Negative	Negative
CSF culture	No growth	Sterile
Blood cultures	No growth	Sterile
CSF cryptococcal antigen	Negative	Negative

TABLE 2: CSF profile and etiological microbiological testing.

PCR: polymerase chain reaction; FTD Neuro9: multiplex real-time viral PCR; HSV: herpes simplex virus; VZV: varicella zoster virus; CMV: cytomegalovirus; EBV: Epstein-Barr virus; HHV: human herpesvirus; HAdV: human adenovirus; EV: enterovirus; HPeV: human parechovirus; B19V: parvovirus B19; IVIG: intravenous immunoglobulin; CSF: cerebrospinal fluid

She was managed symptomatically with intravenous fluids, paracetamol, lorazepam, and metoclopramide. On hospital day 2, she developed new-onset diplopia and blurred vision while remaining hemodynamically stable. Amitriptyline 25 mg nocte was commenced.

Over the next 48 hours, she had persistent headache with reduced oral intake. Physiotherapy assessment documented evolving neck stiffness and subjective upper limb weakness. Amitriptyline was reduced to 12.5 mg nocte and vitamin supplementation was initiated.

On hospital day 5, she acutely deteriorated with a generalized tonic-clonic seizure lasting approximately two minutes, followed by postictal respiratory distress and a reduced level of consciousness (Glasgow Coma Scale (GCS) score: 8/15). She was hypertensive (167/85 mmHg), tachycardic (112 bpm), and required high-flow oxygen therapy. Arterial blood gas analysis demonstrated metabolic acidosis. Neurological examination revealed new left-sided weakness (power 3/5). She received intravenous midazolam and phenytoin loading and was transferred to the intensive care unit, where she was intubated for airway protection. Empirical therapy was initiated with ceftriaxone, vancomycin, acyclovir, fluconazole, intravenous dexamethasone, enoxaparin, and proton pump inhibitor therapy.

Contrast-enhanced MRI of the brain with MR angiography demonstrated gyriform fluid-attenuated inversion recovery (FLAIR) hyperintensity with gyral thickening following the sulcal contours, without diffusion restriction, susceptibility blooming, mass effect, or leptomeningeal enhancement (Figure *1*). Non-specific subcortical and periventricular white matter changes were noted. The circle of Willis was normal, and normal flow voids were present in the dural venous sinuses. A fat-containing lesion in the right petrous

apex, reported as a possible cholesterol granuloma, was considered incidental. Repeat contrast-enhanced MRI five days later showed progression of the gyriform FLAIR changes, new FLAIR hyperintensity in the bilateral thalami, basal ganglia, and basal cisterns, and diffuse meningeal enhancement, without diffusion restriction or hemorrhage. The petrous apex lesion was unchanged, and fluid was noted in the right maxillary sinus.

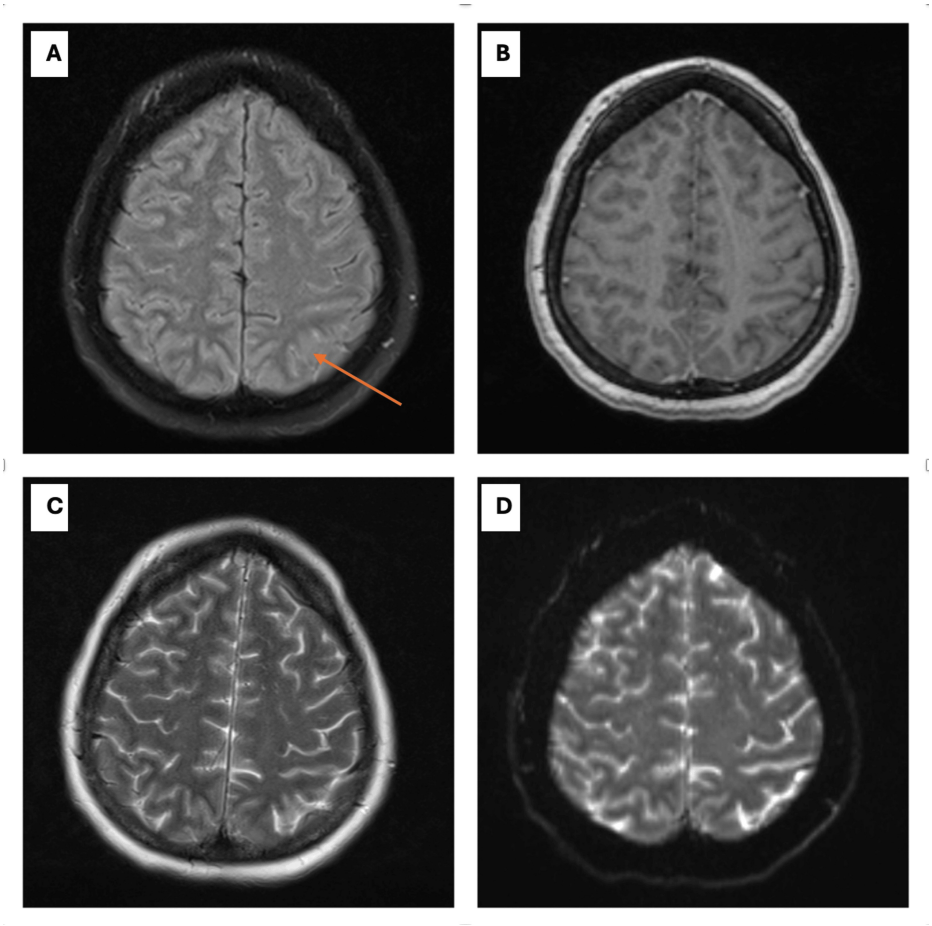


FIGURE 1: Axial MRI brain images.

Axial MRI sequences demonstrating increased signal in the gyri and sulci, with gyral thickening (arrow). (A) Fluid-attenuated inversion recovery (FLAIR) image showing subtle cortical hyperintensity. (B) T1-weighted image showing no corresponding focal structural abnormality. (C) T2-weighted image demonstrating subtle hyperintensity in the corresponding region. (D) Diffusion-weighted imaging showing no convincing diffusion restriction.

The patient required mechanical ventilation, and EEG showed diffuse encephalopathic slowing as shown in Figure *2*. She completed a five-day course of IVIG with gradual neurological improvement. Serum B19V IgM and IgG were tested approximately two weeks after the CSF PCR, after completion of IVIG, and were both negative. Serum or blood B19V PCR was not performed, and serology was not repeated. Deep vein thrombosis prophylaxis, gastrointestinal protection, and strict fluid and electrolyte monitoring were maintained throughout the ICU stay, and there were no episodes of nosocomial sepsis. Ventilatory support was weaned, and she was extubated on hospital day 16.



FIGURE 2: The electroencephalogram shows diffuse slow (delta-theta) activity with intermittent suppression suggestive of diffuse encephalopathy.

Detailed serial examination of tone, sensation, and reflexes was not systematically documented during mechanical ventilation. The documented neurological course was as follows: diplopia from hospital day 2; new left-sided weakness (power 3/5) at the time of the day 5 seizure; progressive improvement in mobility and truncal control with physiotherapy on the ward; at early follow-up, postural and kinetic tremor with globally reduced reflexes and no focal deficits with the left-sided weakness having resolved; and at subsequent follow-up, near resolution of tremor, improved gait, and a newly documented left sixth nerve palsy.

Additional investigations done are summarized in Table *3*.

Investigation	Result	Reference range/expected
Creatinine	77 µmol/L	Creatinine 44-80 µmol/L (female)
Electrolytes	Na 141 mmol/L, K 4.1 mmol/L	Na 135-145 mmol/L, K 3.5-5.0 mmol/L
CA-125	20 U/mL	<35 U/mL
CA19-9	30 U/mL	<37 U/mL
CA15-3	15 U/mL	<30 U/mL
CEA	3.5 ng/mL	<3-5 ng/mL

TABLE 3: Additional biochemical and malignancy screening investigations.

CA-125: cancer antigen 125; CA19-9: carbohydrate antigen 19-9; CA15-3: cancer antigen 15-3; CEA: carcinoembryonic antigen

The patient was transferred to the ward on hospital day 17 in a conscious and haemodynamically stable state. She underwent structured physiotherapy with progressive improvement in mobility, truncal control, and respiratory function. Oral intake improved steadily, and intermittent headaches resolved.

On hospital day 21, ophthalmological examination revealed normal visual acuity, ocular movements, and pupillary responses. Fundoscopy demonstrated bilateral papilledema with disc swelling and loss of physiological cupping. She was discharged on hospital day 21 on acetazolamide, propranolol, short-course rivaroxaban, pantoprazole, baclofen as needed, and iron supplementation, with follow-up arranged in neurology, ophthalmology, and physiotherapy clinics.

At early neurology follow-up, she reported fatigue, poor sleep, and persistent tremors. Examination demonstrated postural and kinetic tremor without bradykinesia, consistent with a post-encephalitic syndrome, along with globally reduced reflexes and no focal deficits. Amitriptyline was reintroduced at a low dose, and propranolol was initiated for tremor control.

At subsequent follow-up, there was marked clinical improvement with near resolution of tremor and improved gait stability. However, she continued to have residual left abducens nerve palsy with end-gaze diplopia and persistent photophobia, without new neurological deficits as described in Figure *3*. Further ophthalmological follow-up was not available, and the subsequent course of the papilledema and abducens palsy is unknown.

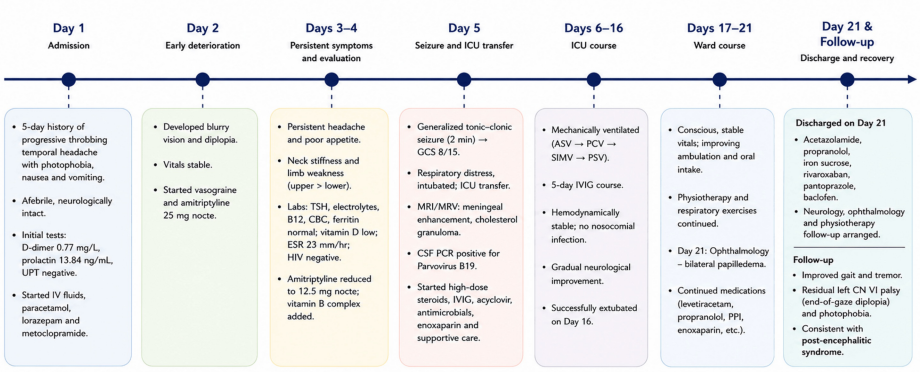


FIGURE 3: Clinical timeline of hospital course, diagnostic evaluation, treatment, and neurological recovery in human parvovirus B19 meningoencephalitis.

CSF: cerebrospinal fluid; CN VI: sixth cranial nerve (abducens nerve); GCS: Glasgow Coma Scale; ICU: intensive care unit; IVIG: intravenous immunoglobulin; MRI: magnetic resonance imaging; MRV: magnetic resonance venography; PCR: polymerase chain reaction; TSH: thyroid-stimulating hormone; CBC: complete blood count; ESR: erythrocyte sedimentation rate; UPT: urine pregnancy test; ASV: adaptive support ventilation; PCV: pressure-controlled ventilation; SIMV: synchronized intermittent mandatory ventilation; PSV: pressure support ventilation; PPI: proton pump inhibitor

The image was created by the authors using Microsoft PowerPoint (Microsoft Corp., Redmond, WA, USA).

Discussion

This case adds to the limited literature describing severe CNS disease associated with B19V infection in adults, in this instance, in an immunocompetent individual. Although B19V is classically associated with erythema infectiosum, arthropathy, and transient aplastic crisis, neurological manifestations are increasingly recognized across both pediatric and adult populations [5,6]. CNS involvement in adults nonetheless remains uncommon and is frequently underdiagnosed because of non-specific clinical features and the variability of CSF and neuroimaging findings.

Neurological involvement in B19V infection encompasses a broad and heterogeneous spectrum, including encephalitis, encephalopathy, meningitis, cerebellitis, cerebrovascular events, and peripheral neuropathies [1,6]. Systematic reviews and case series indicate that encephalitis and encephalopathy are the most frequently reported manifestations, typically presenting with fever, headache, and altered mental status, while seizures are less consistently observed [6,17]. Compared with previously published adult cases, which are generally subacute and self-limiting, the present case followed an unusually severe and rapidly progressive course, with early generalized seizures, profound encephalopathy, and respiratory failure requiring prolonged mechanical ventilation [17]. Such presentations can mimic severe viral or autoimmune encephalitis and may require advanced organ support [14].

Recovery patterns reported in the literature are generally favourable, with neurological improvement occurring over weeks to months and most patients demonstrating substantial recovery within two to six weeks [17]. However, persistent neurological sequelae such as fatigue, cognitive impairment, tremor, and other post-infectious symptoms have been described [14]. In the present case, neurological recovery was gradual but incomplete at discharge, with extubation on day 16 and hospital discharge on day 21. At follow-up, residual deficits included fatigue, photophobia, postural tremor, globally reduced reflexes, and a left abducens nerve palsy.

Several mechanisms could explain the residual left sixth nerve palsy and globally reduced reflexes. Papilledema raises the possibility of a false-localizing sixth nerve palsy from raised intracranial pressure, although opening pressure was not measured and this remains unconfirmed. Diminished reflexes after a 16-day ICU admission with mechanical ventilation and corticosteroids may reflect critical illness polyneuropathy or myopathy. B19V-associated cranial and peripheral nerve involvement has also been described: in a recent case series of B19V-associated multiple mononeuropathy, cranial nerve involvement occurred in a third of patients, including the abducens nerve [18], and peripheral nervous system manifestations account for a substantial share of reported B19V neurological presentations [6]. Nerve conduction studies and electromyography were not performed, so these possibilities could not be distinguished.

CSF findings in B19V-associated neurological disease are variable, and several reported cases, including

severe ones, show no pleocytosis [9,10]. An acellular CSF is therefore compatible with B19V-associated disease but does not specifically support it. The combination of acellular CSF, elevated protein, and a markedly reduced CSF-to-serum glucose ratio of 0.30 also warrants consideration of bacterial meningitis, tuberculous or fungal meningitis, carcinomatous meningitis, and non-infectious inflammatory conditions. Negative cultures, CSF GeneXpert, cryptococcal antigen, and tumour markers reduce but do not eliminate these possibilities, and a single negative GeneXpert does not exclude tuberculous meningitis. Hypoglycorrhachia of this degree is not a typical feature of viral meningoencephalitis, in which the CSF-to-serum glucose ratio is usually preserved, and a ratio below about 0.4 is more often associated with bacterial, tuberculous, or fungal infection or meningeal malignancy. The absence of pleocytosis, and the fact that lumbar puncture preceded antibiotic therapy, argue against bacterial meningitis, but early or paucicellular tuberculous meningitis can present atypically. The low ratio is therefore an important point against B19V being the sole explanation for this illness. Serial MRI findings were consistent with meningoencephalitis but non-specific [14]; their differential is discussed below.

The evolution of the imaging findings broadens the differential diagnosis. Basal cistern involvement with diffuse meningeal enhancement, together with a low CSF-to-serum glucose ratio and elevated protein, is characteristic of tuberculous meningitis; a single negative CSF GeneXpert does not exclude it, although her recovery without antituberculous therapy argues against it. Bilateral thalamic and basal ganglia signal change can also result from deep cerebral venous thrombosis, which was not formally excluded because MR venography was not performed, although normal venous flow voids were seen on both studies. The combination of vomiting, reduced oral intake, diplopia, and thalamic signal change also raises Wernicke encephalopathy. Deep grey matter involvement is described in other viral encephalitides, including flaviviruses such as dengue and West Nile virus, which were not tested. For these reasons, we regard B19V as the most likely identified agent rather than an established cause.

The pathogenesis of B19V-associated CNS disease remains incompletely understood and is likely multifactorial. Proposed mechanisms include direct viral invasion of endothelial or neural tissue, immune complex-mediated vascular inflammation, cytokine-driven disruption of the blood-brain barrier, NS1 protein-mediated cytotoxicity, and post-infectious immune-mediated mechanisms resembling autoimmune encephalitis (Figure *4*) [1]. Which of these mechanisms, if any, operated in the present patient cannot be determined from the available data.

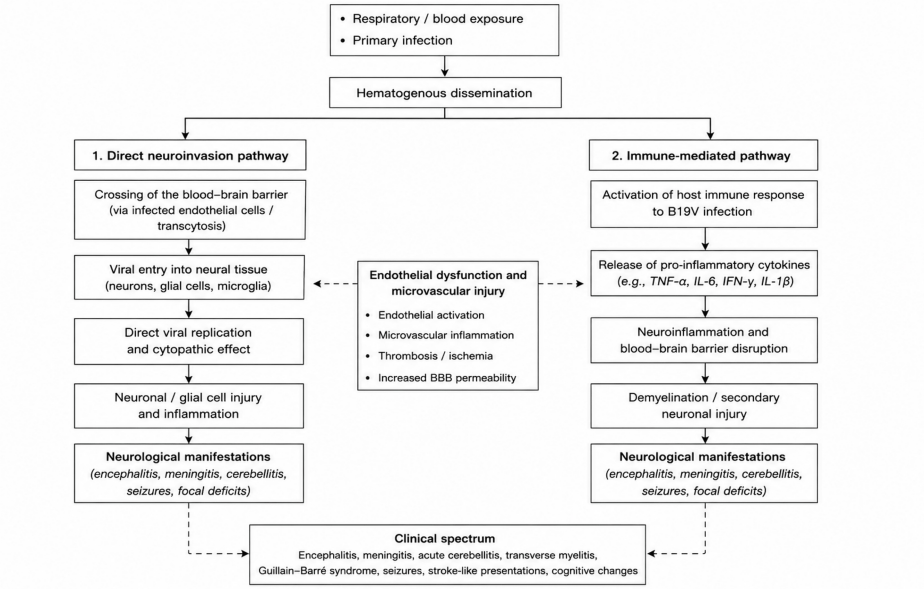


FIGURE 4: Proposed mechanisms of neurological injury in parvovirus B19 (B19V) infection.

The schematic illustrates the hypothesized mechanisms underlying parvovirus B19-associated neurological manifestations, including direct viral neuroinvasion and immune-mediated neurological injury following cytokine activation and neuroinflammation. Endothelial dysfunction and microvascular injury may contribute to both pathways.

The image was conceptualized and created by the authors using Microsoft PowerPoint (Microsoft Corp., Redmond, WA, USA).

There are no established evidence-based guidelines for the management of B19V CNS infection, and

treatment remains largely empirical [7]. High-dose corticosteroids were administered in this case in the context of severe encephalopathy, radiological evidence of meningeal inflammation, papilledema, and rapid neurological decline. The rationale for corticosteroid therapy includes reduction of cerebral edema, stabilization of the blood-brain barrier, suppression of cytokine-mediated neuronal injury, and attenuation of immune-mediated vascular inflammation [7]. Although clinical improvement followed corticosteroid administration, causality cannot be established given concurrent intensive care management and the absence of controlled comparative data [6].

IVIG was administered empirically in view of severe, deteriorating disease without an alternative identified cause. Its proposed actions include passive neutralization of B19V by specific antibodies and broader immunomodulation [19]. IVIG is established for chronic B19V infection with red cell aplasia in immunocompromised hosts, but evidence for its use in B19V-associated neurological disease is limited to individual case reports [10]. A systematic review of IVIG in viral encephalitis found heterogeneous outcomes and no consistent benefit over supportive care [15]. Because corticosteroids, IVIG, antimicrobials, and intensive care were given concurrently, the contribution of any single treatment to this patient's recovery cannot be determined.

The discordance between CSF PCR positivity and negative serum B19V IgM and IgG is the principal limitation to attributing this illness to B19V. Serology was obtained approximately two weeks after the CSF PCR and about three weeks after symptom onset, by which time B19V IgM would usually be detectable after acute infection; this makes very early infection before seroconversion a less likely explanation. A negative IgG is also unexpected after a five-day course of IVIG, as pooled immunoglobulin contains B19V-specific IgG, and raises the possibility of an assay or sampling limitation. Isolated CNS detection has been described: in a recent outbreak series from Madrid, one adult had B19V DNA detected in CSF only, with negative serology and negative plasma PCR, and a previously seronegative child seroconverted three weeks after B19V was found in CSF [17]. Unlike those patients, ours had no plasma PCR or confirmatory assay, and serology remained negative at two weeks. Possible explanations therefore include compartmentalized CNS infection, low-level or persistent B19V DNA unrelated to the acute illness [6,14,20], and a false-positive molecular result, which cannot be excluded without an independent confirmatory assay or blood PCR.

B19V may be considered in the differential diagnosis of unexplained meningoencephalitis in adults when standard investigations are negative, particularly where broad multiplex viral PCR is available. A positive CSF result should be interpreted in the context of serology, blood PCR where available, and a careful exclusion of alternative infectious and inflammatory causes. Severe CNS involvement may occur even in immunocompetent individuals, and normal CSF cellularity does not exclude significant neurological disease. Molecular diagnostic techniques improve diagnostic yield and should be incorporated early in the evaluation of severe or unexplained encephalitic syndromes [12]. Although immunomodulatory therapies such as corticosteroids and IVIG may be considered in severe cases, current evidence remains observational and causality cannot be established [6,15]. Finally, post-encephalitic syndromes may persist beyond the acute illness and warrant structured long-term follow-up and multidisciplinary rehabilitation.

This report has several limitations. First, B19V was detected by a single qualitative multiplex PCR, without confirmation by an independent assay, quantitative viral load, or B19V PCR in blood. Second, B19V serology was negative and was not repeated, so acute infection could not be confirmed serologically. Third, detection of B19V DNA in CSF does not by itself establish causation, since B19V DNA can persist after past infection [6,14]. Fourth, dedicated MR venography was not performed, and opening pressure, autoimmune encephalitis antibodies, flavivirus serology, and nerve conduction studies were not available, limiting interpretation of the CSF profile and residual deficits. Fifth, multiple treatments were given concurrently, so treatment effects cannot be assessed. Finally, serial neurological examination during mechanical ventilation was not systematically documented, formal neurocognitive assessment was not undertaken, and longer-term ophthalmological follow-up was not available.

Conclusions

B19V is an uncommon but potentially important cause of severe meningoencephalitis in immunocompetent adults. We describe a case characterized by rapidly progressive encephalopathy, seizures, respiratory failure, and prolonged intensive care requirements, in which B19V DNA was detected in CSF by multiplex viral PCR, with all other viral targets and B19V serology negative. Although these findings support B19V-associated CNS disease, the serological discordance and absence of confirmatory testing preclude definitive attribution of causality. This case highlights the importance of interpreting multiplex CSF PCR results alongside serology, blood PCR, and investigations for alternative causes. Neurological recovery followed comprehensive supportive care and immunomodulatory treatment with corticosteroids and IVIG; however, the contribution of individual therapies cannot be established from a single case. Long-term follow-up is warranted to identify persistent post-encephalitic sequelae, while further studies are needed to clarify the pathogenesis, optimal management, and long-term outcomes of B19V-associated CNS disease.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ayubu E. Mashambo, Tevin Kahwa, Philip B. Adebayo, Mandela Makakala, Meghna V. Solanki

Acquisition, analysis, or interpretation of data: Ayubu E. Mashambo, Tevin Kahwa

Drafting of the manuscript: Ayubu E. Mashambo, Tevin Kahwa

Critical review of the manuscript for important intellectual content: Ayubu E. Mashambo, Tevin Kahwa, Philip B. Adebayo, Mandela Makakala, Meghna V. Solanki

Supervision: Philip B. Adebayo, Mandela Makakala, Meghna V. Solanki

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Khawaja AA, Mehmood Qadri H, Bashir M, et al.: Charting the neurotropism of human parvovirus B19 and encephalitis: a systematic review of existing literature of 25 years. Cureus. 2025, 17:e89551. 10.7759/cureus.89551
2. Luo Y, Qiu J: Human parvovirus B19: a mechanistic overview of infection and DNA replication . Future Virol. 2015, 10:155-67. 10.2217/fvl.14.103
3. Rogo LD, Mokhtari-Azad T, Kabir MH, Rezaei F: Human parvovirus B19: a review . Acta Virol. 2014, 58:199-213. 10.4149/av_2014_03_199
4. Urio F, George H, Tluway F, Nyambo TB, Mmbando BP, Makani J: Prevalence and factors associated with human parvovirus B19 infection in sickle cell patients hospitalized in Tanzania. Mediterr J Hematol Infect Dis. 2019, 11:e2019054. 10.4084/MJHID.2019.054
5. Bloise S, Cocchi E, Mambelli L, Radice C, Marchetti F: Parvovirus B19 infection in children: a comprehensive review of clinical manifestations and management. Ital J Pediatr. 2024, 50:261. 10.1186/s13052-024-01831-6
6. Barah F, Whiteside S, Batista S, Morris J: Neurological aspects of human parvovirus B19 infection: a systematic review. Rev Med Virol. 2014, 24:154-68. 10.1002/rmv.1782
7. Bookstaver PB, Mohorn PL, Shah A, et al.: Management of viral central nervous system infections: a primer for clinicians. J Cent Nerv Syst Dis. 2017, 9:1179573517703342. 10.1177/1179573517703342
8. Sharma K, Khandia R, Shrivastava R, et al.: Exploring the link between parvovirus B19 and encephalitis: a systematic review and comprehensive meta-analysis of molecular and serological evidence. Virol J. 2025, 22:42. 10.1186/s12985-025-02630-z
9. Lee DS, Lee JH, Huh S, Kim MJ, Kim MK, Yoo BG: Meningoencephalitis associated with parvovirus B19 infection in an immunocompetent patient. Kosin Med J. 2015, 30:181-5. 10.7180/kmj.2015.30.2.181
10. Marouf O, AbuBaha M, Salameh H, et al.: Beyond fifth disease: atypical parvovirus B19 neurological syndromes in adults-first series from the West Bank. Oxf Med Case Reports. 2026, 2026:omag035. 10.1093/omcr/omag035
11. Seye AO, Top FK, Mbanne M, et al.: Clinical features of human parvovirus B19-associated encephalitis identified in the Dakar region, Senegal, and viral genome characterization. Viruses. 2025, 17:111. 10.3390/v17010111
12. Guo J, Wang Y, Zhang M, et al.: Human parvovirus B19 infection in hospitalized patients suspected of infection with pathogenic microorganism. Front Cell Infect Microbiol. 2022, 12:1083839. 10.3389/fcimb.2022.1083839
13. Antezana A, Kister I, Herbert J: A case of encephalopathy in an immunocompetent adult with persistent parvovirus B19 viremia. Open Neurol J. 2015, 9:59-61. 10.2174/1874205X01509010059
14. Douvoyiannis M, Litman N, Goldman DL: Neurologic manifestations associated with parvovirus B19 infection. Clin Infect Dis. 2009, 48:1713-23. 10.1086/599042
15. Wagner JN, Leibetseder A, Troescher A, Panholzer J, von Oertzen TJ: Efficacy and safety of intravenous immunoglobulins for the treatment of viral encephalitis: a systematic literature review. J Neurol. 2022, 269:712-24. 10.1007/s00415-021-10494-w
16. Törün SH, Kaba Ö, Yakut N, et al.: Multicenter prospective surveillance study of viral agents causing meningoencephalitis. Sci Rep. 2021, 11:7216. 10.1038/s41598-021-86687-0
17. Veintimilla C, Catalán P, Estévez A, Alonso R, Valerio M, Muñoz P: Neurologic manifestations associated with parvovirus B19 epidemic, Madrid, Spain, 2024. Emerg Infect Dis. 2025, 31:1621-4. 10.3201/eid3108.250278
18. Theuriet J, Michaud M, Fargeot G, et al.: Multiple mononeuropathy secondary to parvovirus B19 infection: a case series. Eur J Neurol. 2026, 33:e70565. 10.1111/ene.70565

19. Lee R, Garry D, Herrera K, et al.: Intravenous immunoglobulin (IVIG) therapy in pregnancies complicated by acute parvovirus B19 infection in the second trimester: a case series. Case Rep Perinat Med. 2025, 14:20250010. 10.1515/crpm-2025-0010
20. Herr W, Krumkamp R, Hogan B, et al.: A cross-sectional study on risk factors for infection with parvovirus B19 and the association with anaemia in a febrile paediatric population in Ghana. Sci Rep. 2020, 10:15695. 10.1038/s41598-020-72657-5