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Recurrent Ischemic Strokes Caused by HSV-2–Associated Cerebral Vasculitis in Antisynthetase Syndrome

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Statistical Analysis C
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Manuscript Preparation E
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Patient: Female, 76-year-old
Final Diagnosis: Cerebral vasculitis with recurrent ischemic strokes due to HSV-2 encephalitis
Symptoms: Aphasia • concentration difficulty • confusion • epilectic seizure • gait disturbance • headache • syncope
Clinical Procedure: CT brain • digital subtraction angiography • lumbar puncture • MRI brain
Specialty: Infectious Diseases • Neurology
Objective: Rare disease
Background: Antisynthetase syndrome is a rare autoimmune disease characterized by anti-aminoacyl-tRNA synthetase autoantibodies and clinical features including interstitial lung disease, myositis, and arthritis, requiring immunosuppressive treatment. We report a case of a patient with antisynthetase syndrome who developed a severe herpes simplex virus 2 (HSV-2) encephalitis with vasculitis and recurrent strokes.
Case Report: A 76-year-old woman with antisynthetase syndrome on rituximab, mycophenolate mofetil, and prednisone presented with progressive headache, confusion, and gait disturbance. MRI demonstrated bilateral temporal cortical hyperintensities consistent with encephalitis, as well as acute ischemic lesions in the left middle cerebral artery territory. Cerebrospinal fluid analysis showed HSV-2 DNA. Despite intravenous acyclovir, the patient developed aphasia and left-sided neglect. Repeated MRI revealed middle cerebral artery branch occlusion and concentric vessel wall enhancement, consistent with central nervous system vasculitis and multiple acute infarcts. On further follow-up, serial cerebrospinal fluid testing showed delayed viral clearance over 7 weeks. Management included acyclovir and discontinuation of rituximab to allow immune system recovery. Empiric antiplatelet therapy and antiepileptic therapy were added because of suspected intermittent epileptic seizures, with epileptogenic potentials identified on electroencephalogram. After 9 weeks of treatment and rehabilitation, the patient reached partial neurological recovery. Despite residual mild left gaze palsy and fatigue, she regained full functional independence in all activities of daily living.
Conclusions: To the best of our knowledge, this is the first reported case of a patient with antisynthetase syndrome who developed HSV-2 encephalitis complicated by vasculitis and recurrent ischemic strokes. Immunosuppressive treatment may have contributed to delayed HSV-2 clearance, necessitating prolonged acyclovir therapy.
Keywords: Case Reports • Cerebral Vasculitis • Herpes Simplex Virus Encephalitis • Neurology
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952736

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Introduction

Antisynthetase syndrome is a rare autoimmune disease classified as a subtype of idiopathic inflammatory myopathies [1] with a prevalence of 9 per 100 000 people [2]. It is characterized by myositis, interstitial lung disease, arthritis/arthralgia, fever, mechanic's hands, Raynaud phenomenon, and the presence antibodies against aminoacyl-tRNA synthetases, most commonly anti-Jo-1, PL-7, PL-12, EJ, and OJ antibodies [3,4]. Treatment typically involves conventional disease-modifying antirheumatic drugs and immunosuppression therapy with glucocorticoids but can also include B-cell depleting therapy tailored to severity, leading to an increased risk of opportunistic infections [5].

There are no direct data on the risk of stroke in patients with antisynthetase syndrome; however, patients with idiopathic inflammatory myositis have an approximately 2-fold increased risk of ischemic stroke compared with the general population. This risk is lower than that observed in systemic lupus erythematosus or systemic vasculitis [6].

Here, we report a case of herpes simplex virus type 2 (HSV-2) encephalitis in a patient with antisynthetase syndrome on a potent combined immunosuppressive treatment. The clinical course was complicated by central nervous system (CNS) vasculitis with multiple recurrent ischemic strokes.

Although HSV-2 is typically associated with genital infections, it can rarely cause encephalitis, particularly in immunocompromised adults [7]. Among all HSV encephalitis cases, HSV-1 is responsible for approximately 90%, with HSV-2 accounting for the remaining 10% [8]. HSV-2 has a seroprevalence of approximately 12.4% in the general population, with the vast majority of individuals remaining asymptomatic [9].

Case Report

A 76-year-old White woman with a known history of antisynthetase syndrome (anti-PL7 positive) was referred by her general practitioner to a neurologist due to progressively worsening neurological symptoms over a period of 2 weeks, with persistent headache, impaired concentration, confusion, and gait instability.

Her treatment regimen included 2 intravenous cycles of rituximab (each cycle, 1 g), with the recent dose administered 7 weeks prior to presentation. These were initiated to induce remission of a severe disease flare 3 months earlier. She was also receiving daily mycophenolate mofetil 1000 mg twice daily and prednisone 7.5 mg once daily.

Upon arrival at the neurologist's office, she experienced a loss of consciousness, with a sustained upward gaze lasting approximately 1 minute, clinically suggestive of an epileptic seizure, which was not detected on an electroencephalogram (EEG). The patient was immediately referred to the emergency department with suspected metabolic encephalopathy or encephalitis.

Initial assessment in the emergency department showed normal vital signs, and the general physical examination was unremarkable, except for residual erythema of a recurrent gluteal herpes lesion. Neurological examination revealed a left gaze palsy, hemiataxia on the left side, and gait instability with a broad-based gait. Initial laboratory investigations revealed normal inflammatory markers (C-reactive protein and leukocytes), while creatine kinase and lactate dehydrogenase were mildly elevated. Cranial computed tomography (CT) did not show any signs of intracranial hemorrhage or increased intracranial pressure.

Cerebrospinal fluid findings revealed a pleocytosis with 77 mononuclear cells/µL (predominantly lymphocytes; reference range: 0-5 cells/µL), an elevated protein concentration of 1545 mg/L (reference range: 150-500 mg/L), and elevated lactate levels of 3.9 mmol/L (reference range: 1.1-1.9 mmol/L).

Notably, polymerase chain reaction (PCR) testing of cerebrospinal fluid (CSF) was positive for HSV-2 DNA, confirming the diagnosis of HSV-2 encephalitis. The patient was promptly started on intravenous acyclovir at a dose of 10 mg/kg every 8 hours, in addition to supportive care measures, including seizure prophylaxis with levetiracetam.

Brain magnetic resonance imaging (MRI) performed on day 3 after admission revealed bilateral temporal cortical hyperintensities. Additional involvement was noted in the left frontal cortex and right thalamus, with contrast-enhancement consistent with viral encephalitis (**Figure 1**). Two small ischemic lesions were identified in the left middle cerebral artery (MCA) territory (**Figure 2A**). Due to the presence of ischemic lesions, antiplatelet therapy with aspirin and statin therapy was initiated. Cardiac monitoring and echocardiography revealed no indication for a cardioembolic source.

Despite treatment, the following day, the patient presented new neurological deficits, including left-sided hemineglect and expressive aphasia. Repeated MRI demonstrated disease progression with an occlusion of the M2 segment of the MCA (**Figure 3**) and new areas of cerebral infarction (**Figure 2B**). Vessel wall imaging revealed concentric enhancement of multiple affected arterial segments, findings consistent with CNS vasculitis (**Figure 4**).

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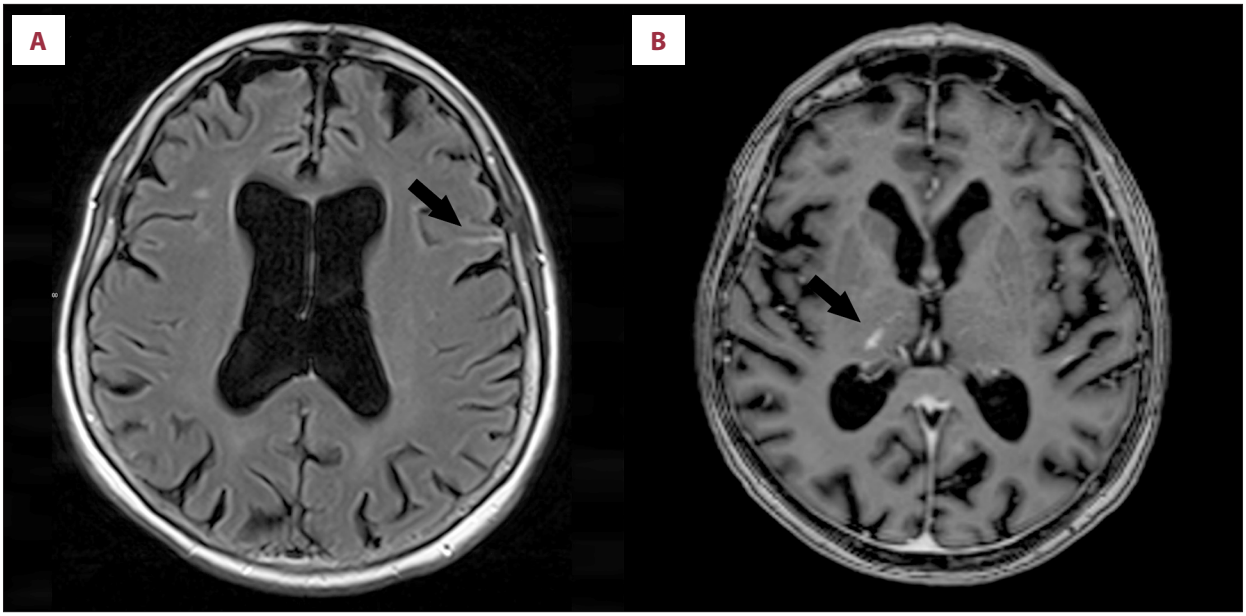

Figure 1. Examples of bilateral temporal cortical hyperintensities (**A**) on turbo inversion recovery magnitude (TIRM) sequences and contrast-enhancement of the thalamus in magnetization prepared rapid acquisition with gradient echoes (MPRAGE) post-contrast sequences (**B**).

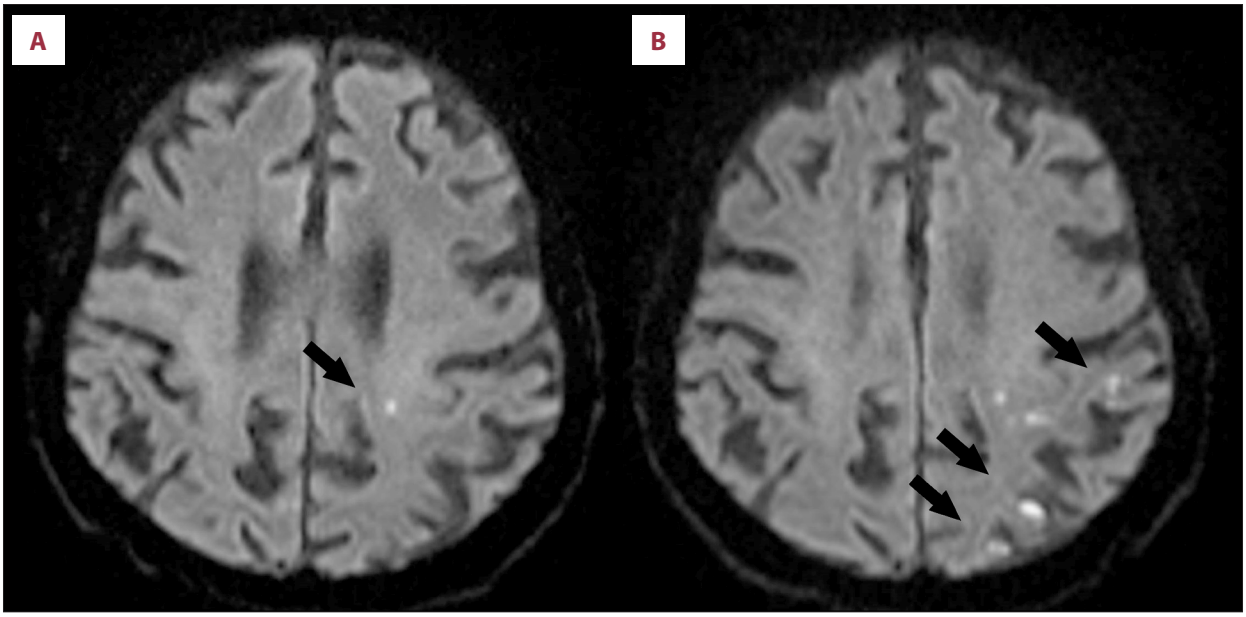

Figure 2. Progression of multifocal ischemia in the left middle cerebral artery territory as seen on day 3 (**A**) vs day 8 (**B**) in diffusion-weighted magnetic resonance imaging.

The EEG demonstrated a multifocal lesion pattern, most pronounced in the right frontotemporal region, with signs of a nonspecific disturbance of cerebral function, including frontal intermittent rhythmic delta activity, which is a nonspecific EEG pattern characterized by transient, rhythmic delta waves over the frontal regions and typically indicates diffuse cerebral dysfunction or encephalopathy. No definite epileptiform potentials were observed.

Discussion

In our patient with antisynthetase syndrome, we presume that the vasculitis and strokes were a consequence of severe and prolonged HSV-2 encephalitis in the context of immunosuppression rather than the underlying autoimmune disease, as there were no signs of previous ischemia. In the following sections, we discuss the various aspects of this case separately.

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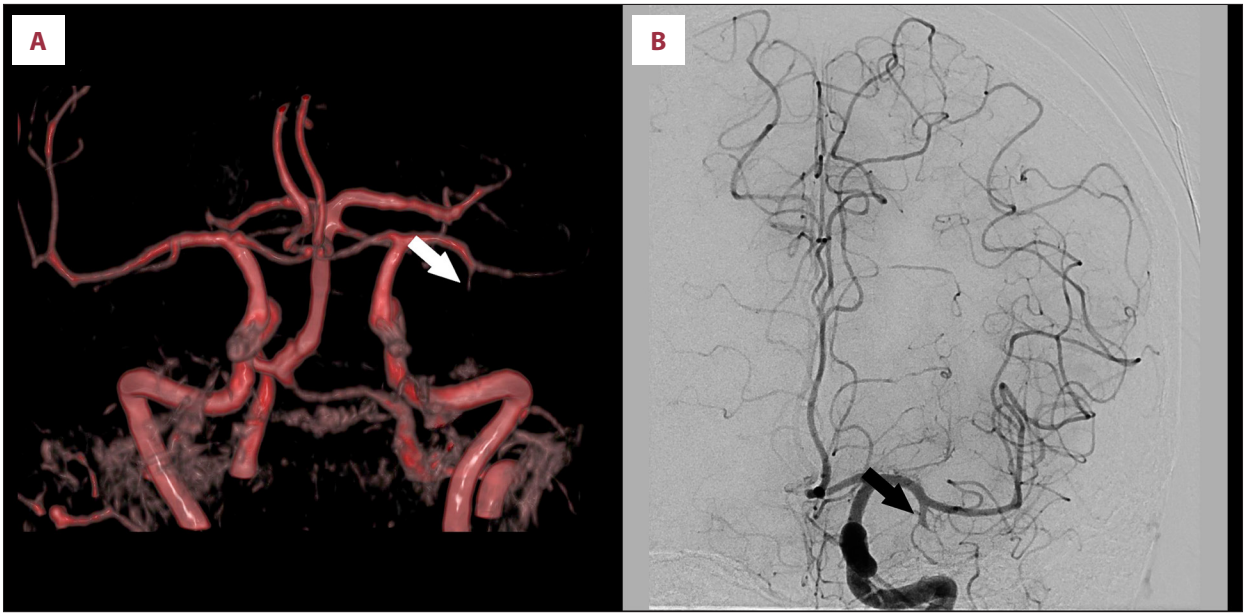

Figure 3. Occlusion of the left middle cerebral artery (M2 segment) in magnetic resonance time-of-flight angiography (**A**) and digital subtraction angiography (**B**).

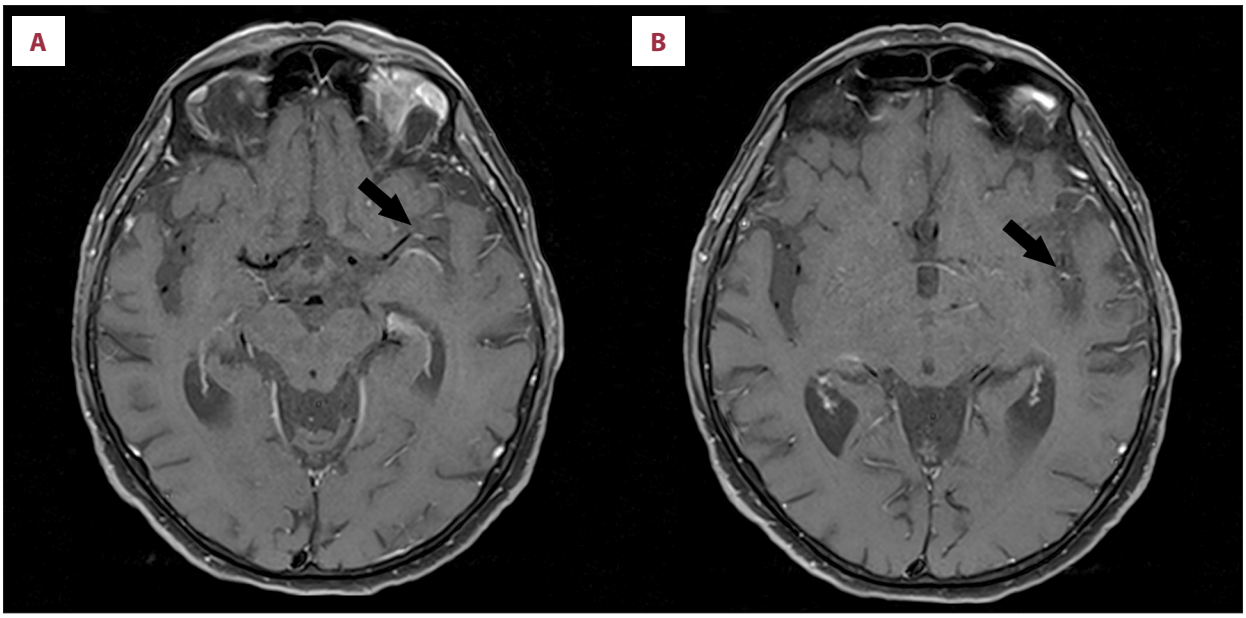

Figure 4. Contrast-enhanced magnetic resonance images showing enhancement of the left middle cerebral artery M2 segment (**A**) and M3 segment (**B**), suggestive of vasculitis.

Infection by HSV-2

HSV-2 reoccurrence is often seen in immunocompromised hosts. In the present case, a preexisting recurrent gluteal herpes infection was the source of infection. The symptoms of headache, confusion, and gait instability are characteristic but nonspecific for encephalitis [10]. The diagnosis was confirmed with symptoms, abnormal imaging, EEG findings, CSF pleocytosis, and a positive HSV-2 PCR (**Table 1**).

Standard treatment consists of intravenous acyclovir, typically administered for 2 to 3 weeks [10]. In immunosuppressed patients, repeated analysis of CSF is recommended with quantitative measuring of HSV DNA to evaluate treatment response, with HSV expected to become undetectable in the same timeframe [11]. In the present case, therapy duration was prolonged due to persistently elevated HSV-2 viral load in the CSF (**Table 1**). A test for rarely occurring acyclovir resistance [12] remained inconclusive.

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Table 1. Overview of the serial cerebrospinal fluid (CSF) analysis showing the delayed HSV-2 clearance.

Analysis	Reference	Day 1	Day 15	Day 22	Day 30	Day 52
Leukocytes $\times 10^6$/L	<5	77	62	78	47	13
Mononuclear cells $\times 10^6$/L		77	61	78	47	13
Polymorphonuclear cells $\times 10^6$/L		0	1	0	0	0
Lactate in CSF mmol/L	1.1-1.9	3.9	3.62	2.58	2.58	1.93
Protein in CSF mg/L	150-500	1545	984	1136	625	437
CSF/serum albumin ratio $\times 10^{-3}$	<9.1	–	19	23.6	12.8	–
BioFire PCR	Negative	HSV 2	HSV 2	HSV 2	HSV 2	–
HSV-2- PCR quantitative GEq/mL		–	12800	2000	1500	200

After 9 weeks of treatment, the patient developed leukopenia with neutropenia of 160/uL (reference range: 2500-7000/uL), most likely secondary to prolonged acyclovir exposure. Although the HSV-2 was still traceable a week prior, the patient was transitioned to secondary prophylaxis with oral valacyclovir, a therapy outside the scope of current guidelines, based on the individual risk profile. The delayed viral clearance was most likely due to the reduced humoral immunity caused by B-cell depletion (no CD-19 cells detected in flow cytometry on day 31) through rituximab, as opposed to the immunosuppression by steroids and mycophenolate mofetil.

Encephalopathy and Secondary Epilepsy

A pathological EEG can be seen in up to 75% of patients with herpes simplex virus encephalitis [13]. Initial management of our patient's case included levetiracetam. Under this treatment, the EEG showed epileptiform temporal activity, supporting the diagnosis of HSV-related encephalopathy with secondary seizures (day 16). Lacosamide was added, but the patient developed dizziness and concentration problems (prevalence of dizziness approximately 15%) [14]. A therapeutic switch to valproate coincided with the development of an exanthema shortly after contrast-enhanced imaging, prompting the transition to eslicarbazepine due to possible drug hypersensitivity.

Cerebrovascular Complications

Cerebrovascular complications are frequent in patients with herpes encephalitis, occurring in approximately 17% to 20% and including hemorrhage, vasculitis, and ischemic stroke [15].

Our patient showed repeated acute ischemic lesions in serial MRI examinations, distributed in the left posterior MCA territory. Additionally, contrast enhancement of the vessel walls up to the M3 segment of the MCA and faintly in other vessels was observed, suggestive of a arterio-arterial embolic origin. MRI angiography showed no relevant findings in the extracranial vessels, and cardiological assessments did not reveal any cardioembolic origin.

We concluded that the HSV-2 associated vasculitis was most likely the cause of the recurrent ischemic strokes. This could be a result of different pathological mechanisms, including the following. (1) Direct viral-induced vasculitis: Invasion of the virus damages cerebral vessel walls, causing inflammation, stenosis, and thrombosis [16]. (2) Endothelial dysfunction: Viral cytotoxicity disrupts endothelial integrity, increasing susceptibility to thrombosis and hemorrhage [17]. (3) Autoimmune vascular damage: The immune system may generate autoantibodies that cross-react with host neuronal or vascular antigens, contributing to delayed vasculopathy [18,19]. (4) Immune complex-mediated vasculitis: Antigen-antibody complexes may deposit in the walls of cerebral vessels, activating complement and triggering secondary immune complex-mediated vasculitis [18]. (5) Coagulopathy from systemic inflammation: Release of pro-inflammatory cytokines and endothelial activation promote a prothrombotic state, increasing risk of stroke [20]. (6) Type I interferon deficiency: Genetic or acquired defects affecting the Toll-like receptor 3 or other parts of the interferon signaling pathway, leading to poor viral control and persistent vascular injury [19].

As the underlying mechanism, we suspect direct virus-induced vasculitis. Systemic inflammation appeared insufficient to account for coagulopathy, and immune complex-mediated vasculitis was most prominent in the first 10 days of hospitalization, making autoimmune vascular damage less likely.

While intravenous acyclovir remains the treatment for HSV encephalitis, the management of CNS vasculitis is less well-defined. This adds complexity to management, as immunosuppression for vasculitis may worsen viral infection, while insufficient treatment risks further strokes and neurological deterioration.

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An association between ischemic strokes and antisynthetase syndrome is generally not recognized in the current literature, except for the association of stroke with idiopathic inflammatory myositis, which includes antisynthetase syndrome [6]. We found 3 published cases of vasculitis in patients with antisynthetase syndrome who had not received prior treatment. The first involved a branch retinal vein occlusion secondary to retinal vasculitis [21]. The second described widespread nonspecific T2 hyperintense lesions in cortical, subcortical, and periventricular white matter, with irregular tapering of the middle and anterior cerebral arteries, interpreted as small- to medium-vessel vasculitis [22]. The third reported hemiparesis due to a acute infarct in the anterior cerebral artery territory, suspected to result from vasculitis [23]. These cases suggest a mechanism distinct from that in our patient, who had previously received immunosuppressive therapy.

Long-Term Outcome

After the acute illness, our patient was transferred to a neurological rehabilitation center. After discharge, oral valacyclovir was continued for at least 6 months to prevent relapses, and long-term antiplatelet therapy with aspirin was started. MRI after 14 weeks showed an unchanged number of small-volume post-inflammatory and post-ischemic gliosis in the frontoparietal regions bilaterally. Previously, some of these lesions were also associated with blood-brain barrier disruption, which have since completely resolved. At 6-month follow-up, the patient demonstrated substantial neurological recovery with resolution of hemineglect and aphasia but continued to experience mild left gaze palsy and fatigue.

Neurological recovery in herpes simplex virus encephalitis depends on the promptness of treatment. Herpes encephalitis has an attributed mortality of 8.9% to 14.0% [24,25], and up to 50% to 60% of patients show persistent neurologic symptoms [26]. Our patient showed improvement but continued to have residual deficits.

Conclusions

This case illustrates that patients with antisynthetase syndrome receiving immunosuppressive therapy are at risk for serious infectious and immunological complications beyond those of their underlying autoimmune disease. Our patient had prolonged HSV-2 encephalitis with delayed viral clearance, complicated by CNS vasculitis and recurrent ischemic strokes. The combination of a rare primary condition necessitating strong immunosuppression and an infrequent opportunistic infection with cerebrovascular complications led to a unique clinical scenario. Our case also emphasizes the central role of a close multidisciplinary collaboration across internal medicine, neurology, infectious diseases, and rheumatology in managing such complex, high-risk patients.

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Department and Institution Where Work Was Done

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Informed Consent

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

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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