Case Report

When viral invasion meets vascular catastrophe: A case report of herpes simplex virus-related stroke

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ABSTRACT

Herpes simplex virus (HSV) encephalitis is the most common viral encephalitis in industrialized nations, typically affecting the temporal lobes. While seizures are frequent complications, cerebrovascular events such as hemorrhagic stroke are rare. We report an 85-year-old man with multiple comorbidities who presented with altered mental status and progressive weakness. Cerebrospinal fluid analysis confirmed HSV-1 DNA, and neuroimaging revealed diffuse microbleeds and a left cerebellar hematoma. The patient was managed conservatively with intravenous acyclovir and antibiotics, showing gradual improvement, and was discharged in stable condition. This case highlights the unusual occurrence of cerebellar hemorrhage in HSV encephalitis, likely due to necrotizing vasculitis and immune-mediated vascular injury. Awareness of such vascular complications is essential for timely diagnosis and management. Early antiviral therapy, supportive care, and close monitoring can improve outcomes, even in elderly patients with significant comorbidities.

Key words: Acyclovir therapy, Cerebellar hemorrhage, Herpes simplex virus encephalitis, Necrotizing vasculitis, Stroke complications

Herpes simplex virus (HSV) encephalitis is the most commonly diagnosed viral encephalitis in industrialized nations, with an annual incidence of 1 in 250,000–500,000 [1]. Both HSV-1 and HSV-2 can cause encephalitis, which is a necrotizing inflammatory process typically affecting the cortex, underlying white matter of the temporal lobe, and the limbic system [2]. The clinical manifestations of HSV encephalitis include fever, mental status changes, seizures, and focal neurological deficits, which are not pathognomonic, as there are various other diseases of the central nervous system (CNS) that can mimic HSV encephalitis [3]. The most common CNS complication of HSV encephalitis is seizures (38.4%), while intracranial hemorrhage is very rare (2.7%) [4]. Close monitoring is recommended for signs of deterioration or the lack of improvement, and imaging studies are essential to evaluate for neurological complications [4]. In adults, HSV encephalitis has a 25% mortality even with antiviral treatment, and on gross examination of the brain specimens, it often presents with characteristic lytic and hemorrhagic lesions in the medial temporal lobes and the inferior frontal lobes [5]. The mechanism behind cerebrovascular pathology leading to ischemic or hemorrhage stroke and its relationship with HSV encephalitis is not well understood, with only a few cases being reported in the literature [6]. Awareness of the potential for stroke in patients with HSV encephalitis is essential for timely intervention and management.

Here, we present a case report of HSV encephalitis-induced hemorrhagic stroke.

CASE REPORT

An 85-year-old gentleman with known comorbidities of hypertension, Type 2 diabetes mellitus, Parkinsonism, dementia, and post-cerebrovascular accident status, presented with a history of altered mental status, progressive weakness of bilateral lower limbs associated with difficulty in walking and doing activities of daily living. He also complained of urinary frequency, urgency, and hesitancy before admission.

On admission, the patient was afebrile with a temperature of 37.6°C, pulse rate of 80/min, blood pressure of 140/80 mmHg, respiratory rate of 18/min, and oxygen saturation of 98% on room air. He was thoroughly examined, and relevant investigations were sent. Physical examination showed rigidity, bradykinesia,

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and power 3/5 in all four limbs. The patient was sleepy but obeying commands, speech showed dysarthria, bilateral pupils equal and reactive to light.

A cerebrospinal fluid (CSF) study was done, which showed 3 cells, increased glucose - 85 mg/dL, protein - 38 mg/dL, absent coagulum, and Lymphocytes - 100%. CSF GeneXpert for Mycobacterium tuberculosis was negative. CSF HSV1 DNA was detected, confirming viral etiology. CSF beta amyloid 42 was found to be decreased at 716 pg/mL (normal range - 725–1777 pg/mL), consistent with underlying dementia.

Ultrasound whole abdomen was done, which showed mildly heterogenous hepatic parenchymal echotexture, a prominent common bile duct at the porta, and an umbilical hernia. Elastography was done, which showed acoustic radiation force impulse of 2.4 kPa, suggestive of mild hepatic stiffness. Magnetic resonance imaging (MRI) brain showed multiple foci of blooming on susceptibility weighted imaging (SWI) sequence in the right parietal lobe, bilateral thalami, pons, and left hemi cerebellum, likely suggestive of microbleeds and slit-like T2/fluid-attenuated inversion recovery hypointensity in the right putamen and the right external capsule, likely suggestive of chronic bleed with volume loss. In view of increasing drowsiness and vomiting, an urgent computed tomography (CT) scan of the brain was done, which showed a hematoma at the left cerebellar hemisphere, significant supratentorial ventricular dilatation with small intraventricular hemorrhage, and diffuse cerebral atrophy with bilateral periventricular deep white matter ischemia (Fig. 1a). The bedside electroencephalogram showed an abnormal awake record suggestive of a non-specific encephalopathic pattern. A repeat CT scan of the brain 20 days post-operative showed minimal residual hematoma with surrounding edema/hypointensity in the left cerebellar hemisphere (Fig. 1b). 24-h Holter monitoring was done, which showed a baseline sinus rhythm with episodes of sinus tachycardia and sinus bradycardia. Urine C/S was sent, which showed the growth of Carbapenem- resistant *Enterobacterales* (CRE) *Klebsiella pneumoniae*.

He was managed conservatively with IV antibiotics (Ceftazidime/Avibactam plus Aztreonam) and IV acyclovir 10 mg/kg every 8 h for 14 days. He gradually improved with the above management and was discharged in a hemodynamically stable condition after 29 days post-admission, with the advice of proper nursing care at home for intermittent nasogastric tube feeding, out-of-bed mobilization, speech and swallow therapy.

DISCUSSION

Studies have shown that intracerebral hemorrhage, which almost exclusively occurs within the temporal lobe, is related to HSV-1 and can cause life-threatening complications [7]. Extra-temporal involvement is well described in herpes simplex encephalitis, occurring in more

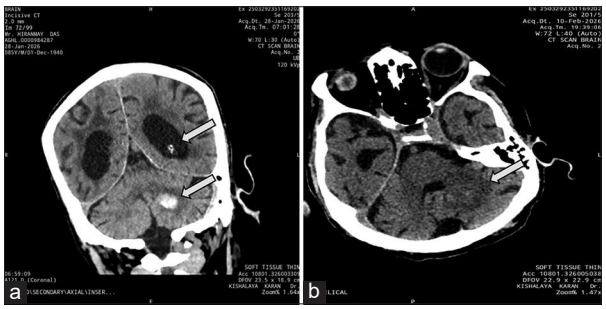

Figure 1: (a) CT scan of brain showing hematoma at left cerebellar hemisphere (lower arrow), significant supratentorial ventricular dilatation with small intraventricular haemorrhage and diffuse cerebral atrophy with bilateral periventricular deep white matter ischemia (upper arrow). (b) CT scan of brain showing minimal residual hematoma with surrounding oedema/hypointensity in the left cerebellar hemisphere

than half of the cases, sometimes even without any temporal abnormalities [7]. In our case, hemorrhage occurred in the cerebellar hemisphere. We have compared our case study to previous reported studies (Table 1). The hemorrhage seen is most likely a complication secondary to the disintegration of vessels in the context of a necrotic encephalitis process [7]. The CNS pathology can be attributed to a combination of cytolytic viral replication and immune-mediated mechanisms leading to axonal and glial damage [7].

MRI is the neuroimaging procedure of choice in patients with suspected HSV encephalitis and is more sensitive and specific than CT [8]. More than 90% of polymerase chain reaction (PCR) proven HSV encephalitis cases have abnormal MRI findings, with increased T2 and decreased T1 signal in the temporal lobes, with associated lesions in the frontal lobe occurring in more than one-third of cases [8]. In our patient, we found multiple foci of blooming on the SWI sequence in the right parietal lobe, bilateral thalami, pons, and the left hemi cerebellum, likely suggestive of microbleeds.

HSV-1 encephalitis has a high morbidity and mortality of up to 70% without treatment [8]. It should be considered among the possible causes of both ischemic and hemorrhagic strokes [3]. A persistently positive PCR HSV in CSF may be a risk factor for worse clinical outcomes [3]. In our case, a repeat CSF to check for the PCR status was not done. The use of the PCR technique is a diagnostic procedure in HSV infection, but negative PCV test results of CSF HSV can occur within the first 72 h of illness, with subsequent test results becoming positive, as in this case. Therefore, diffusion-weighted magnetic resonance may play a major role as a first and early diagnostic step [9].

Although temporal and frontal lobe involvement is most typical in HSV encephalitis, extra-temporal hemorrhage, including cerebellar sites, has been increasingly reported. The pathogenesis is thought to involve a combination of direct viral cytolysis and immune-mediated vasculitis, leading to endothelial damage and vessel wall necrosis [7]. Necrotizing inflammation weakens small vessels, predisposing them to rupture and bleed. SWI often reveals diffuse microbleeds, reflecting widespread microvascular injury

Table 1: Comparison of the current case with previous studies

Study/Report	Patient demographics	Site of hemorrhage	Key findings	Outcome
Current case (85-year-old male)	Elderly, multiple comorbidities (HTN, DM, Parkinsonism, dementia, prior CVA)	Cerebellar hemisphere (rare, extra-temporal)	CSF HSV-1 DNA positive; MRI showed diffuse microbleeds; CT revealed cerebellar hematoma; likely necrotizing vasculitis+ immune-mediated vascular injury	Gradual improvement with IV acyclovir+antibiotics; discharged stable
Zlatanova and Boykinova, (2024) [3]	Adult patient	Ischemic stroke (not hemorrhage)	HSV encephalitis complicated by acute ischemic stroke; PCR confirmed HSV	Partial recovery; highlights stroke risk in HSV
Ehret *et al*., 2022 (Cureus) [4]	Middle-aged adult	Temporal lobe	HSV-1 encephalitis with hemorrhagic conversion; seizures common	Improved with acyclovir; neurological sequelae persisted
Sotomayor Villanueva *et al*., (2022) [6]	Adult	Temporal lobe hemorrhage	HSV-1 encephalitis leading to ischemic and hemorrhagic stroke	Severe neurological deficits; poor prognosis in some cases
Hauer *et al*., (2019) (Systematic Review) [7]	Mixed cohort	Predominantly temporal lobe, occasional frontal involvement	Cerebrovascular complications (ischemic+ hemorrhagic) rare but documented; pathogenesis linked to viral cytolysis+vasculitis	High morbidity; mortality up to 70% without treatment

HTN: Hypertension, DM: Diabetes mellitus, CVA: Cerebrovascular accident, CSF: Cerebrospinal fluid, HSV: Herpes simplex virus, CT: Computed tomography, MRI: Magnetic resonance imaging, PCR: Polymerase chain reaction, IV: Intravenous

that can progress to frank hemorrhage in atypical regions such as the cerebellum. Advanced age and comorbid vascular risk factors further increase vessel fragility, amplifying the risk of hemorrhage outside the classical temporal lobe predilection. Thus, cerebellar hemorrhage in HSV encephalitis likely represents the interplay of viral neurotropism, immune-mediated vascular injury, and host vascular vulnerability. In our patient, the presence of HSV-1 DNA in CSF confirmed viral etiology, while MRI and CT findings of microbleeds and cerebellar hematoma supported the hypothesis of necrotizing vasculitis and immune-mediated vascular injury.

Moderate to severe sequelae like cognitive deficits, aphasia, seizures, and focal weakness have been reported in 32–56% of adult survivors. Several prognostic factors, like age and the level of consciousness of the patient at the time of institution of treatment, influence morbidity and mortality rates. Our patient was an 85-year-old and had a history of altered mental status at the time of admission. Several studies have suggested that the time delay between hospital admission and institution of acyclovir therapy might also influence prognosis. We started our patient on acyclovir on the 2nd day post-admission.

Clinicians should monitor for vascular complications in HSV encephalitis patients presenting with acute neurological deterioration [3]. Early antiviral therapy and supportive care can improve outcomes in patients, but secondary infections and comorbidities may complicate recovery and long-term prognosis. In our patient, urine culture showed growth of CRE Klebsiella for which he was managed with appropriate antibiotics.

CONCLUSION

High index of clinical suspicion, prompt diagnosis, and early treatment are the mainstays of therapeutic success in herpetic encephalitis. Acyclovir treatment should be started as early as possible on clinical suspicion, and this must be done while waiting for further confirmation, such as the lumbar puncture results or serology. Furthermore, the imaging studies can be inconclusive, and not all cases have a typical appearance, both clinically and radiologically.

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