Herpes simplex reactivation following neurosurgery: case report and review of the literature

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Herpes simplex encephalitis is a common viral encephalitis associated with significant morbidity and mortality if not diagnosed and treated early. Neurosurgery may be an impetus for viral reactivation, either from direct nerve manipulation or high-dose steroids often administered during cases. The authors present the 40th known case of herpes simplex virus (HSV) encephalitis following neurosurgical intervention and review the previously reported cases. In their review, the authors observed positive HSV polymerase chain reaction (PCR), which had initially been negative in several cases. In cases in which there is high suspicion of HSV, it may be prudent to continue antiviral therapy and retest CSF for HSV PCR. Antiviral therapy significantly reduces mortality associated with HSV encephalitis.

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KEYWORDS HSV; encephalitis; postoperative; postneurosurgery herpes simplex virus

HERPES simplex encephalitis (HSE) is the most common cause of acute viral encephalitis in the United States.²⁵ Many cases, as many as two-thirds, are due to virus reactivation rather than a primary infection, as 90% of the population is infected with herpes simplex virus (HSV).^{7,25} Neurosurgical intervention can potentially cause reactivation of HSV, although the true incidence rate is unknown since it is rarely considered a postoperative complication. We present the case of a 72-year-old immunocompetent man with HSE following craniotomy for meningioma resection.

Case Report

A 72-year-old man without a known history of HSV infection presented with 2 years of progressive diplopia on leftward gaze and several months of left eye drooping. CT scanning of the head showed a right sphenoid wing/cavernous sinus lesion suspicious for meningioma. The lesion encased the ipsilateral anterior and middle cerebral arteries and extended extracranially to the anterior clinoid process, causing compression of cranial nerves (CNs) II

and III on that side. MRI demonstrated the mass abutting the anterior aspect of Meckel's cave, with the contour of the trigeminal ganglion remaining intact (Fig. 1). CNs III, IV, and VI were engulfed within the tumor, with V1 and V2 of the trigeminal nerve just inferior to the tumor.

The patient underwent partial resection to remove mass effect on CN II and remove tumor from around the middle cerebral artery, anterior cerebral artery, and internal carotid artery, as well as the clinoid due to tumor invasion with associated hyperostosis. To accomplish this, a pterional incision was made with the bone flap centered over the sylvian fissure and the clinoid. The dura was found to adhere to the skull and was opened. The sylvian fissure was opened under microscopic visualization, and the case was completed using microsurgical technique. The tumor was found to be deep and lateral in the fissure over the medial sphenoid wing. The tumor was coagulated and debulked, with time taken to remove tumor from the anterior and middle cerebral arteries and the supraclinoid carotid artery, as well as from CNs II and III. Intraoperatively, CNs IV, V, and VI were not visualized or manipulated in any way. Of note, the patient received 40 mg Decadron intra-

ABBREVIATIONS CN = cranial nerve; HSE = herpes simplex encephalitis; HSV = herpes simplex virus; PCR = polymerase chain reaction; POD = postoperative day.

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FIG. 1. Axial MR image of the right meningioma with cranial nerve involvement.

operatively, 4 mg every 6 hours scheduled postoperatively with taper to off in 2 days. Additional plans were made for follow-up Gamma Knife surgery. The pathology returned as psammomatous meningioma (WHO grade I). The patient's intraoperative course was complicated by a small right frontal ischemic infarction, and postoperatively the patient had worsening of his CN III palsy. He was subsequently discharged home on postoperative day (POD) 4.

On POD 6, the patient presented to the emergency department for a new left-sided headache and increased periincisional edema. He was afebrile but had mild leukocytosis (12.3 K/ μ L), and head CT scanning showed increased postoperative subdural and subgaleal fluid collections. He was admitted and later that day developed disorientation and hallucinations. On POD 8, the patient underwent a lumbar puncture that found xanthochromic CSF with 68,000 RBC/mm³, 1651 WBC/mm³ (43% lymphocytes, 53% monocytes/macrophages, 4% neutrophils), 208 mg/dL protein, and 40 mg/dL glucose. He became febrile to 39.3°C, increasingly confused, and restless. Blood cultures, chest radiography, and urinalysis findings were unremarkable. The encephalopathy continued to worsen, and on POD 12 the patient was admitted to the ICU for atrial fibrillation with rapid ventricular response. His subgaleal fluid was drained later that day but reaccumulated, and a lumbar drain was placed for CSF leakage on POD 14. CSF sent at that time was xanthochromic with 2000 RBC/mm³, 362 WBC/mm³, 343 mg/dL protein, and 42 mg/dL glucose; cultures would ultimately be negative. Acyclovir was started on POD 15 when HSV-1 polymerase chain reaction (PCR) returned positive. Findings from his cognitive examination remained poor; he was inattentive and nonverbal but at times made eye contact, visually tracked, and inconsistently followed simple commands. His examination findings subtly improved over the next 15 days and

he began to speak again on POD 29. On POD 35 he completed his 3-week acyclovir course and he was discharged to a rehabilitation facility on POD 39 alert, oriented, able to converse normally, and with full strength throughout.

Discussion

HSE is a known, but rare, complication following neurosurgical intervention.^{11,29} Encephalitis results from either primary infection with HSV or reactivation of latent HSV residing within the nuclei of sensory neurons, traditionally within trigeminal nerve ganglia.^{17,27} While the specific mechanism for HSV reactivation following neurosurgery is unknown, steroid therapy, radiation, and stress are known factors that increase the likelihood of HSV reactivation. It is hypothesized that these factors contribute to immunocompromise, thereby creating a permissive environment for the herpes virus to escape latency.^{14,37} There is also evidence to suggest that direct manipulation and/or trauma to nerves or surrounding tissue may facilitate reactivation.²⁹ Our patient received dexamethasone both intra- and postoperatively, which could have contributed to postoperative HSE. Additionally, the surgical approach may have resulted in potential trauma to the trigeminal nerve due to tumor resection within the lateral wall of the cavernous sinus, which could reactivate latent virus within the sensory ganglia. Extradural approaches are becoming more common to minimize trauma to the brain from retraction and limit contact with cranial nerves, as well as to prevent CSF leaks, which are a known potential complication of the intradural approach. An additional benefit of utilizing an extradural approach could be to limit intradural spread of HSV. In our case, the patient's dura was essentially shredded due to its adherence to the skull, so regardless of approach it would have been breached.

The latency period for HSV can be greater than 10 days. However, on average, symptom onset occurs 6 days postoperatively.¹ Viral encephalitis may clinically present in a variety of ways. Patients often present with fever, focal seizures, and altered mental status. Some possible psychiatric presentations include agitation, drowsiness, and hallucinations.²⁸ These symptoms can mimic other postoperative complications and can make diagnosis challenging. Thus, there should be a low threshold to evaluate and treat possible HSV encephalitis in postoperative neurosurgical patients presenting with fever and seizures or altered mental status.

If there is a suspicion of HSE, diagnostic workup should include lumbar puncture, CSF analysis, and CSF HSV PCR. Common initial CSF analysis in HSE typically consists of a lymphocytic pleocytosis, erythrocytosis, normal CSF glucose, and elevated protein. However, these findings are relatively nonspecific, thereby requiring concomitant HSV PCR testing. PCR is both sensitive and specific in the diagnosis of HSV encephalitis and has revolutionized practice; however, it is important to note that the results may not become positive for 24–48 hours following initial symptoms and will remain positive for 2–5 days following treatment.^{7,33} If undiagnosed, HSV-1 encephalitis is associated with high mortality and high morbidity in survivors, often leaving patients with dis-

TABLE 1. Review of the literature

Authors & Year	Primary Lesion	Age (yrs)	Sx Onset (POD)	Sxs	Time Until LP	CSF Glucose*	CSF Protein*	CSF WBCs/mm ³	CSF Lymph (%)	CSF PMNs (%)	PCR	Status
Aldea et al., 2003	Rt frontocingular oligodendroglioma	28	7	Fever, POD 8 drowsiness	POD 8	NI	0.8 g/L	38	30	28 neutro, 42 mono	Neg	Alive
					POD 9		1.7 g/L	306			HSV-1	
de Almeida et al., 2015	Refractory epilepsy	11	12	Fever, HA, nausea/vomiting, aLOC	POD 12	55	83	265	99	1	HSV-1	Alive
Alonso-Vanegas et al., 2016	Rt temporal lobectomy	10	4	Fever, HA, aLOC (POD 6)	POD 6						HSV	Alive
Berger et al., 2016	Lt sphenoid wing meningioma	70	12	Fever, HA, nuchal rigidity, CN III palsy	POD 17	24	113	256	100	0	HSV-2	Alive
Bourgeois et al., 1999	Lt AH for intractable complex partial seizure	8	6	Partial status epilepticus, aphasia, fever	POD 6	NI	0.6–1.6 g/L	Pleocytosis	75–95%		HSV-1	Alive
Cunha et al., 2014	WHO grade IV GBM	60	30	Fever, lt facial weakness, rt upper-/lower-extremity strength	POD 30	33	100	0			HSV-1	Dead
Fearnside & Grant, 1972	Case 1: Pituitary adenoma	41	4	Fever, auditory & visual hallucinations	POD 4	60	105			320 PMN, 320 mono	HSV	Dead
	Case 2: pituitary adenoma	11	8	Fever, seizures	POD 9	80	45			4 PMN, 6 mono	HSV	Dead
Filipo et al., 2005	Acoustic neuroma	33	2 & 10	HA; POD 10 fever, hyperreflexia, aLOC	POD 11	53	84.4	39	32	28 neutro, 45 mono	HSV-1	Alive
					POD 1 routine EVD	<2	<6	0			Neg	
Gong et al., 2010	Refractory epilepsy	2	1	Fever, irritability, spells	POD 3 routine EVD	26	638	1050	13	1	HSV-1	Alive
					POD 5 LP		377	4400	4	35	HSV-1	
	Case 1: MVD of lt facial nerve	73	9	Lt facial weakness; fever, malaise, nausea, memory impairment	Unk	47	112	1520			HSV	Alive
Hengstman et al., 2005	Case 2: MVD of lt facial nerve	56	15 20	Nasal CSF leakage Lt-sided numbness, hearing loss	Unk	41	91	2520			HSV	Alive
	Case 3: suboccipital craniectomy w/ excision of a meningioma located at lt petrosal apex	66	90	CN V, VI, VIII, & XII neuropathies	Unk	65	48.3	1			HSV	Alive

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TABLE 1. Review of the literature

Authors & Year	Primary Lesion	Age (yrs)	Sx Onset (POD)	Sxs	Time Until LP	CSF Glucose*	CSF Protein*	CSF WBCs/mm ³	CSF Lymph (%)	CSF PMNs (%)	PCR	Status
Ihekwa & Bat- tersby, 2009	CM-1	35	14	Progressive headaches							HSV-2	Alive
Jalloh et al., 2009	Acoustic neuroma	44	1	Fever, vomiting, confusion							HSV-1	Alive
	Case 1: epidermoid cyst	24	8	Fever, HA	POD 10	2.7 mmol/L	0.94 g/L	1517	95	1 erythro	HSV-1	Alive
	Case 2: WHO grade I	53	18	Fever, drowsiness	POD 18	1.6 mmol/L	1.25 g/L	188	92	26 erythro	HSV-2	Alive
Jaques et al., 2016	Craniopharyngioma											
	Case 3: rt temporal lobectomy & AH	12	11	Fever, headache	POD 14	1.9 mmol/L	2.05 g/L	91	96	837 erythro	HSV-1	Alive
Kim et al., 2013	Epilepsy surgery	11	5	Fever, confusion	POD 20			1			HSV-1	Alive
											Unk	
Kuhnt et al., 2012	Case 1: pituitary adenoma	40	4	Seizure	POD unk (several days later)			80			HSV-1	Dead
	Case 2: petroclival meningioma	46	7	Fever, aLOC	POD 7			720			Unk	Dead
				Fever, aLOC	POD 12			55			HSV-1	
Kwon et al., 2008	Craniopharyngioma (adenomatous type, WHO grade I)	13	15	Fever, decreased LOC	POD 22	39	113.9	2 neutro	3		Pos	Alive
Lellouch-Tubiana et al., 2000	Refractory epilepsy	8	6								HSV-1	Alive
Lund, 2011	Frontal lobe epilepsy	19	10	Fever, HA	POD 10	68	49	10	8		Unk	Dead
					POD 17	68	108		83		HSV	
Mallory et al., 2012	Acoustic neuroma	49	10	Fever, HA	POD 10			Lymphocyte predominant pleocytosis			HSV-1	Alive
Molloy et al., 2000	Medulloblastoma	22	>21	Confusion							HSV	Dead
	Case 6: SAH clipping	62	14	Rash							Unk	Unk
Nabors et al., 1986	Case 7: GBM	60	7	Bilat periorbital swelling w/ chemosis & injection of it eye consistent w/ HSV							Unk	Dead
Navarro et al., 2013	SAH	51	7	HA, somnolence, dysphagia	POD 7	65	74	21			HSV-2	Alive

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TABLE 1. Review of the literature

Authors & Year	Primary Lesion	Age (yrs)	Sx Onset (POD)	Sxs	Time Until LP	CSF Glucose*	CSF Protein*	CSF WBCs/mm ³	CSF Lymph (%)	CSF PMNs (%)	PCR	Status
Perry et al., 1998	Craniopharyngioma	64	8	Lethargy, confusion, vomiting, diarrhea	POD 11 POD 18 POD 27	31 308 64	171 111	0 308 113	98 0	2 100 mono	Unk Unk HSV-2	Alive
Ploner et al., 2005	Meningioma	47	10	Fever, confusion	POD 10			<1 (POD 13, 12)			HSV	Alive
Lo Presti et al., 2015	Refractory epilepsy	17	6	Seizures	POD 7	NI	26.9	2	2	0	Unk	Alive
Samadian et al., 2016	Meningioma	55	2	Fever, aLOC, spastic quadriparesis	POD 4						HSV-1	Dead
Sayal et al., 2018	WHO grade IV GBM	63									HSV on histol	Dead
Sheleg et al., 2001	GBM	28	2								HSV-1	Dead
Spuler et al., 1999	Lt parasagittal meningioma in premotor area	78	10	Focal motor seizures, aphasic, fever, hemiparetic, somnolent	POD ~18 POD 19	NI NI	NI 94	89 38	70 35	26 50 mono	Neg HSV-1	Dead
Tang et al., 2013	Rt MVD for trigeminal neuralgia	29		Fever, HA, confusion							HSV-1	Alive
Uda et al., 2013	MTLE	20	11								HSV	Alive
Vik-Mo et al., 2014	MTLE	25	3	HA, lethargy, confusion/day 11 fever							HSV-2	Alive
Present study	Psammomatous meningioma (WHO grade I)	72	6	HA	POD 8	40	208	1651	62	35	Not tested	
					POD 13	42	343	362	44	55	HSV-1	Alive
					POD 19	53	308	12	94	6	Not re-tested	

AH = amygdalohippocampectomy; aLOC = altered LOC; CM-1 = Chiari malformation type 1; erythro = erythrocyte; EVD = external ventricular drain; GBM = glioblastoma (multiforme); HA = headache; histol = histology;

LOC = level of consciousness; LP = lumbar puncture; mono = mononuclear; MTLE = mesial temporal lobe epilepsy; MVD = microvascular decompression; neg = negative; neutro = neutrophil; NI = normal; PMN = polymorphonuclear leukocyte; pos = positive; SAH = subarachnoid hemorrhage; Sx = symptom; unk = unknown.

* Values are mg/dL unless otherwise noted.

abilities including memory changes, language disorders, and mutism.^{5,28} Early treatment with acyclovir decreases expected mortality from 70% to 30%.¹⁶

This case is unique with regard to the patient's initial subtle presentation, which then progressed to more classic symptomatology for HSV encephalitis. Although not initially in the differential diagnosis, the eventual diagnosis of HSV guided treatment and the patient improved drastically. It is also unclear in this case, as in many others, the exact mechanism of HSV reactivation. The surgical approach might influence HSV reactivation, although this should not necessarily influence surgical planning, but it could more quickly prompt the provider to consider HSV encephalitis testing in patients presenting with altered mental status, fever, or seizures postoperatively.

Review of the Literature

A review of the literature was performed to identify cases of postoperative HSV after neurosurgery. A PubMed search was conducted utilizing the search terms HSV, herpes simplex, and neurosurgery. Study results were screened for cases that described postoperative HSV following neurosurgery. Isolated spine cases were excluded. Other reviews of literature were scanned to ensure inclusion of all reported neurosurgical cases. Thirty-nine cases were extracted from 32 case reports. Pertinent patient data were extracted and compiled into table format (Table 1). In our review of 40 cases of postoperative neurosurgical patients with HSV diagnosis following craniotomy, 12 (30%) patients died and 1 had unreported outcome due to hospital transfer.^{1-4,6-24,26,30-36} Fever, headache, and altered mental status were common presenting symptoms and started anywhere from POD 1 to POD 90, with a majority occurring in a delayed fashion between PODs 7 and 12.^{1-4,6-24,26,30-36} Surgical site varied and does not alone explain potential reactivation of HSV, although it is unknown how many of these patients received perioperative steroids. It is also unclear how many of these patients had a known preexisting history of HSV.

Interestingly, in patients with multiple CSF studies, HSV PCR was at times initially negative and then became positive on subsequent samples.^{1,10,33} In light of this, there may be a role for prophylactic acyclovir in postoperative neurosurgery patients presenting with fevers and seizures or altered mental status. It may also be prudent to continue prophylactic antiviral agents despite initially negative HSV PCR results, particularly within the first 48 hours following symptom onset, and await repeat PCR results before discontinuing. A case could also be made for consideration of perioperative administration of acyclovir in patients with a known HSV history and surgeries with probable cranial nerve manipulation.

Conclusions

HSV encephalitis is rarely reported as a postoperative infection; however, it may be underreported if untested. To our knowledge, this represents the 40th report of HSV detection following neurosurgery. In cases of delayed postoperative fever, headache, seizures, and altered mental status following neurosurgery, there should be a low

threshold to obtain HSV PCR and initiate empirical treatment with acyclovir. Additionally, if the patient has not improved, HSV PCR can be retested in 48 hours prior to discontinuing. Clinical suspicion and prompt treatment of HSV can decrease patient morbidity and mortality in the neurosurgical population.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: McLaughlin. Acquisition of data: McLaughlin, Geertman. Analysis and interpretation of data: McLaughlin, Achey. Drafting the article: all authors. Critically revising the article: McLaughlin, Geertman, Grossman. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: McLaughlin. Study supervision: Geertman.

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