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Unilateral Facial Neurological Symptoms in Patients with Prior Herpes Virus Infections: A Two-Patient Case Series

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ABSTRACT: Herpes simplex virus type 1 (HSV-1) and varicella-zoster virus (VZV) both establish lifelong latency in the sensory ganglia and can reactivate at various times. Both can cause neurological disease involving the craniofacial region, although the nature of the disease and the mechanism of its presentation differ. This case series presents two patients with unilateral facial neurological symptoms, a history of prior herpesvirus infection, and a dental history.
Patient A presented with persistent unilateral facial muscle contractions, which were clinically diagnosed as hemifacial spasm and had a history of herpes labialis (HSV-1). Patient B presented with recurrent unilateral neuropathic facial pain, which was clinically diagnosed as trigeminal neuralgia and had a history of herpes zoster (VZV). Dental procedures are known to be a
possible trigger for reactivation of a latent herpesvirus infection, either due to the timing or the physiological stress placed on the body. However, the records in either case do not support a comparable procedural history or a clear causal link. The clinical features, the cranial nerve involved, and the relevant viral association are considered in both cases. Patient B's management also included a course of varying doses (0.2 cc or 0.5 cc) of a 1 cc solution that included 0.2 cc dexamethasone with 0.8 cc of 1% lidocaine injections at 14 neuropuncture acupoints, with resultant pain relief. The two cases highlight the importance of differentiating facial motor dysfunction from trigeminal neuropathic pain and considering a history of prior herpesvirus infection in the overall clinical picture.

KEYWORDS: Hemifacial spasm, Trigeminal neuralgia, Herpes simplex virus type 1, Varicella zoster virus, Herpesvirus reactivation, Dental procedures, Cranial neuropathy, Facial pain.

1. INTRODUCTION

HSV-1 and VZV are neurotropic human herpesviruses which establish latency in the sensory ganglia. HSV-1 most commonly establishes latency in the trigeminal ganglion, and VZV can remain latent in the cranial nerve and dorsal root sensory ganglia. Reactivation can result in several clinical syndromes, including recurrent orofacial lesions, zoster, and cranial neuropathies.[1,2]
Unilateral facial neurological symptoms require careful localization. Hemifacial spasm (HFS) is defined as uncontrolled contraction of the muscles supplied by the ipsilateral facial nerve (CN VII), usually due to neurovascular contact or compression of the facial nerve. Trigeminal neuralgia (TN), by contrast, is a neuropathic pain condition of the trigeminal nerve (CN V) that can be classical, secondary, or idiopathic.[3,4]
Dental and orofacial procedures have been suggested as possible precipitants of herpesvirus reactivation to cranial neuropathies. The research is conflicting, with some reports of VZV-associated facial palsy following dental/orofacial procedures and prospective studies of HSV-1 reactivation following dental treatment having failed to demonstrate a significant increase.[5-7] This warrants consideration of a potential association, rather than establishing causation.
This case series presents two patients with different unilateral facial neurological presentations, both with a history of prior herpesvirus infection. It examines the clinical features and relevant cranial nerve pathways in each case, considers the potential association with dental work and herpesvirus reactivation, and describes the clinical management.

2. CASE SERIES METHODS

This descriptive case series was compiled from a retrospective review of the available clinical notes and any additional procedural details provided by the treating clinical team. Two de-identified cases have been selected to demonstrate two different unilateral craniofacial neurological presentations in the setting of a history of herpesvirus infection. The clinical features, relevant

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investigations, management and documented outcomes are all presented descriptively. No causal inferences can be made, and no estimates of treatment efficacy can be drawn from this two-patient series. Written publication consent was reported as obtained from both patients.

3. CASE PRESENTATIONS

Patient A: Unilateral facial contractions

A 50-year-old woman was seen in October 2026 for several months of ear fullness or swelling extending into the neck and episodic left-sided facial twitching. At the 14th of October, she had recurrent left periocular twitching and one episode of lower lip twitching, plus one episode of bilateral hand shaking whilst holding her hands up. She also had daily headaches and some morning headaches. On examination, there was a very narrow external auditory canal opening with possible swelling or irritation. There was visible twitching on the left side of the face only, predominantly the lower orbicularis oculi, orbicularis oris and cheek (buccinator, zygomaticus major and minor, levator labii superioris, risorius) muscles.

At the October 28 follow-up, symptoms were still confined to the left side, with ongoing periocular twitching and intermittent pulling/twitching of the left lower lip, cheek, and forehead.

At this appointment, the patient reported that her upper teeth had recently been extracted and dentures were being fitted. She felt the dental work had precipitated or exacerbated her facial symptoms. She had a remote history of fever blisters, with the last one several years earlier. The clinician concluded that the current presentation was consistent with hemifacial spasm and that HSV reactivation was possible. There was no evidence of active herpetic lesions, and no virology was available to confirm this.

Relevant history was of thyroid surgery in 2015 for a multinodular goitre. A surgical consult noted a prior subtotal thyroidectomy with residual tissue in the left thyroid bed, including a solid vascular nodule on the prior ultrasound of approximately 39 x 18 x 14mm. A biopsy was recommended in the radiology report, but no biopsy or subsequent imaging results were available in the records provided. The patient also reported intermittent neck swelling and a previously described vascular nodule in the neck. The October 2026 lab results were TSH 4.27 mIU/L and free T4 0.69 ng/dL, both within the reported laboratory reference range.

Treatment on 28th October was valacyclovir 500mg once daily for 30 days and gabapentin 100mg twice daily for 30 days. The records did not contain any neurology testing to confirm the diagnosis or any data on response to treatment.

Patient B: Trigeminal Neuralgia

A 38-year-old with a known diagnosis of trigeminal neuralgia had a history of recurrent left-sided craniofacial symptoms. On April 9, 2026, she reported ongoing left-sided TMJ and jaw pain, left eye discomfort, facial swelling, tinnitus, head pressure, throat discomfort, episodic headaches, neck stiffness, dizziness and intermittent loss of balance. On examination, there was tenderness over the left TMJ and a blotchy erythematous rash on the left face which extended down the neck to the clavicle. She had planned her dental extractions and denture fitting, but the records don't indicate when this was actually completed.

Patient B has been coming in regularly since then for dexamethasone injections and pain management with gabapentin. She presented again with the same symptoms and agreed to have the dexamethasone injections as part of the neuropuncture therapy. She reported improvement in her symptoms after the injection. The injections were given in the TMJ, periorbital and posterior cervical areas. Her post-injection pain was 3/10 to 5/10 as opposed to 10/10 before the injections. Facial warmth and flushing persisted, while facial burning and shock-like sensations improved.

She also had a separate warm, burning rash on the lateral chest, as well as facial erythema and swelling, shown in Figure 1. The physician attributed the facial flushing to dexamethasone exposure.

She has a history of Chiari and a previous episode of shingles. On her office visit on October 28, 2026, she had an acute flare of left-sided head and facial pain, rating the pain 8/10, which was partially relieved with ibuprofen and her morning gabapentin. She was prescribed valacyclovir 500mg daily for 30 days and continued on gabapentin. At this appointment, she received a course of injections at 14 neuropuncture acupoints (see Table 1 in the discussion section). She reported some pain relief following the injections. She did, however, report that she had to go to the clinic quite frequently to have these injections, as they give her temporary relief for at least 2-3 days until the effect of the medication wears off.

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Figure 1: Clinical picture showing the erythematous burning rash on the lateral chest following dexamethasone exposure.

4. DISCUSSION

This case series examines two separate unilateral facial neurological presentations in patients with a history of herpesvirus infection. Patient A has the classic presentation of HFS, which is essentially a facial nerve abnormality. HFS most commonly arises from neurovascular compression at the facial nerve root exit zone, although secondary structural and other causes should be considered.[4] In the absence of any neurology or imaging to confirm the diagnosis, this would be a purely clinical diagnosis.

Patient B's established TN is a completely different neurological entity. TN is a condition of the trigeminal sensory system. It is classically described as recurrent, unilateral, brief, severe, electric shock-like or stabbing facial pain, often provoked by a very minor stimulus. The notes describe recurrent unilateral shock-like pain, but it does not meet all diagnostic criteria. The current guidelines focus heavily on clinical classification and investigation for a secondary etiology.[3,9]

The herpesvirus histories demonstrate a biologically plausible but unproven link. HSV-1 is well established in the literature as being able to establish latency in the trigeminal ganglion and to reactivate.[1] VZV likewise can establish latency in the sensory ganglia and can result in either an acute case of zoster or persistent or recurrent postherpetic neuralgia. Trigeminal postherpetic neuralgia would be expected to have a temporal relationship between the zoster and the subsequent onset of persistent or recurrent pain in the same trigeminal distribution. Patient B's history meets some but not all criteria, as the pain existed prior to the potential trigger of dental intervention[2,10]

Dental or orofacial procedures are a rather interesting clinical comparison, as the timing isn't as clear in either case. In Patient A, the most recent history was an upper dental extraction and denture insertion in the context of left-sided facial twitching. Patient B's April record is for dental extractions and denture insertion with no temporal link to the subsequent symptoms. There are published reports of VZV-associated facial palsy following dental treatment or orofacial surgery.[6] Reactivation of HSV following trigeminal nerve surgery, a more invasive procedure with a different degree of anatomical exposure than a routine extraction, has also been reported.[11,12] On the other hand, prospective studies of HSV-1 reactivation following dental procedures have not consistently shown a statistically significant increase.[5,7] Dental procedures could therefore be considered potential triggers, or at least temporally associated, but these cases do not demonstrate reactivation or prove causality.

Published reports of post-dental viral facial palsy are almost invariably about facial weakness or paralysis rather than HFS. Patient A's documented phenotype is involuntary contraction rather than weakness. The dental procedure literature therefore confirms a temporal association but does not establish that the dental work or HSV-1 caused the HFS.[6] Patient B's history of prior shingles makes VZV-associated trigeminal neuropathy a plausible differential, though prior zoster in itself does not establish postherpetic neuralgia.

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Patient A's prior thyroid surgery and the residual thyroid bed nodule are separate clinical entities and would require appropriate structural follow-up. There is a reference to a previously identified nodule which would be appropriate to biopsy. There is no suggestion of any relationship between the lesion and the facial twitching. Serum Calcium and PTH were within normal limits, so a metabolic etiology of the neuromuscular irritability was ruled out.

Distinguishing the Treatment Plans

Both patients were given treatment for their individual cases. Although there may be overlap on physical examination, each patient's treatment was determined by their specific presentation and past medical history.

Patient A

On October 14, 2026, patient A received therapeutic acupuncture at ST2, SI18, and GV26 (see Table 1 for descriptions)[8]. This helped reduce the left-sided facial twitching in the inferior portion of the left orbicularis oculi, which was the patient's chief concern at the visit. At the follow-up on October 28, 2026, the patient was prescribed valacyclovir 500 mg daily for 30 days and gabapentin 100 mg twice daily for 30 days.

Patient B

Patient B frequently came to the clinic for dexamethasone injections for her facial pain and associated neuropathy symptoms. At a previous clinic visit, the patient received dexamethasone injections at the temporomandibular joint region, periorbital region and behind the neck. At the follow-up on October 28, 2026, the patient was started on valacyclovir 500 mg daily for 30 days and was told to continue gabapentin. The patient also agreed to take part in neuropuncture treatment at 14 acupoints, as described in Table 1.[8] The patient reported immediate relief after receiving the injections.

Neuropuncture Acupoint Injection Technique and Anatomical Distribution

The procedure was described as injections administered at designated acupuncture points, rather than acupuncture with needles. The point codes identify traditional meridian or extra-point locations. The anatomical descriptions below orient medical readers; they do not imply proven efficacy for trigeminal neuralgia or selective targeting of a trigeminal branch.

Table 1. Anatomical Distribution of Neuropuncture Acupoints Injection Sites in Patient B

Point code / traditional name	Meridian and anatomical location	Dose Administered from Decadron/lidocaine Solution*
ST2 (Sibai)	Stomach meridian; infraorbital region, below the pupil.	0.2 cc
ST6 (Jiache)	Stomach meridian; masseter region over the mandibular angle.	0.2 cc
SI11 (Tianzong)	Small intestine meridian; posterior scapular region.	0.2 cc
SI18 (Quanliao)	Small intestine meridian; cheek/zygomatic region.	0.2 cc
SI19 (Tinggong)	Small intestine meridian; entire side of face (trifacial neuropuncture acupoint; trifacial nerves at temporomandibular joint)	0.5 cc
BL2 (Zanzhu)	Bladder meridian; medial end of the eyebrow.	0.2 cc
GV26 (Shuigou)	Governing Vessel; midline in the philtrum between the nose and upper lip.	0.2 cc
LI4 (Hegu)	Large intestine meridian; dorsum of hand between first and second metacarpal bones.	0.2 cc
GB20 (Fengchi)	Gallbladder meridian; posterior neck/suboccipital region.	0.2 cc
LR3 (Taichong; also written LIV3)	Liver meridian; dorsum of foot between first and second metatarsal bones.	0.2 cc
ST36 (Zusanli)	Stomach meridian; anterolateral lower leg, below the knee.	0.5 cc
ST44 (Neiting) .2	Stomach meridian; dorsum of foot between second and third toes.	0.2 cc
ST45 (Lidui) .2	Stomach meridian; lateral side of second toe near the nail.	0.2 cc
TH5 (Waiguan)	Triple-heater/Sanjiao meridian; dorsal forearm, proximal to the wrist.	0.5 cc

* The Decadron and lidocaine solution consisted of 0.2 cc dexamethasone plus 0.8 cc of 1% lidocaine, making a total mixture of 1 cc. Each neuropuncture acupoint received an injected dose of 0.2 cc or 0.5 cc, depending on the location's anatomical capacity.

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Table 1 documents the distribution of neuropuncture acupoint injection sites, including facial, cervical, scapular, upper-extremity, and lower-extremity sites. Each neuropuncture acupoint
corresponds to a specific meridian and anatomical location, as described in the table.[8]

Comparison of Neurological and Virological Features

The cases share unilateral craniofacial symptoms and histories of herpesvirus infection, with dental care being potentially relevant but not well documented in the clinical histories. Their predominant neurological presentations differ markedly. Patient A has intractable unilateral facial spasms which are clinically consistent with hemifacial spasm and are confined to CN VII. Patient B has established trigeminal neuralgia, which is a neuropathic pain condition of CN V.

Table 2. Comparison of Neurological and Virological Features

Feature	Patient A	Patient B
Dominant syndrome	Unilateral facial motor contractions; clinically assessed as HFS	Unilateral neuropathic facial pain; clinically established as TN
Principal cranial nerve	Facial nerve (CN VII)	Trigeminal nerve (CN V)
Prior herpesvirus history	Remote herpes labialis; HSV-1	Prior shingles; VZV
Dental history	Recent upper tooth extraction and denture placement; patient perceived symptom worsening	Dental extractions/denture placement planned in April records; pain existed before this intervention as well.
Viral hypothesis	Possible HSV-1 reactivation; unconfirmed	Possible VZV-related trigeminal neuropathy; unconfirmed
Key limitations	No viral testing or follow-up results.	No virologic confirmation or longitudinal pain outcome.

Limitations

The main limitations are the retrospective design, the small sample size, the incomplete documentation of Patient B's dental procedure history, no virology, insufficient data to meet the criteria for postherpetic neuralgia, incomplete imaging and specialist assessment and the lack of longitudinal outcomes.

5. CONCLUSION

These cases demonstrate how careful clinical localization can differentiate between unilateral facial motor symptoms involving the facial nerve and neuropathic facial pain involving the trigeminal nerve. The patients' histories of HSV-1 and VZV-associated disease, and the timing of their dental care, provide a relevant clinical picture for considering both viral and non-viral etiologies. The treatment plans were similar, with valacyclovir for possible herpes reactivation and gabapentin for the neuropathic component; the main difference was acupuncture in patient A and neuropuncture in patient B. A causal link cannot be established from these cases. However, they highlight the importance of a detailed history, appropriate neurological assessment, and continued investigation of post-procedural craniofacial symptoms. Further work is therefore required to establish a link between the trigger (e.g. dental procedure) and viral reactivation, and to determine the efficacy of the proposed treatment regimes.

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7. DATA AVAILABILITY

No datasets were generated or analyzed during the preparation of this case report.

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Consent for publication

Verbal and written informed consent were obtained from the patient for the preparation and publication of this case report, including the use of the clinical photographs. Informed consent was obtained by the Department of Internal Medicine, Stanaford Medical Clinic.

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