Acta Scientific Neurology (ASNE)

Volume 9 Issue 9 September 2026

Case Report

Inflammation-Induced Trigeminal Nerve Tethering Following Herpes Zoster Mimicking Classical Trigeminal Neuralgia: A Case Report

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DOI: 10.31080/ASNE.2026.09.0933

Received: August 06, 2026

Published: August 28, 2026

Abstract

Background: Postherpetic trigeminal neuralgia (PHTN) typically presents with persistent neuropathic facial pain and sensory disturbances. Classical trigeminal neuralgia (TN)-like paroxysmal pain following herpes zoster is uncommon, and its underlying pathophysiology remains poorly understood. We report a rare case in which chronic inflammatory changes, trigeminal nerve tethering, and nerve deformity were associated with classical TN-like symptoms.

Case Description: A 65-year-old woman developed paroxysmal electric shock-like pain in the left V1–V2 distribution 18 months after herpes zoster infection. Magnetic resonance imaging demonstrated neurovascular contact with marked trigeminal nerve deformity. Intraoperatively, severe perineural adhesions and inflammatory granulation tissue were identified. Although vascular decompression was achieved, nerve deformity persisted, prompting meticulous perineural dissection and internal neurolysis. Histopathological examination demonstrated fibrosis with lymphocytic infiltration, consistent with chronic inflammation. After internal neurolysis, trigeminal nerve conduction showed unchanged latency and a 183% increase in amplitude. Nerve deformity improved substantially, and the patient experienced marked pain relief without neurological complications.

Conclusion: Chronic inflammatory changes following herpes zoster may promote trigeminal nerve tethering and deformation, potentially rendering otherwise asymptomatic neurovascular contact symptomatic. In selected patients with classical TN-like symptoms after herpes zoster, perineural dissection combined with microvascular decompression and internal neurolysis may address both vascular and nerve-related pathological components.

Keywords: Herpes Zoster; Internal Neurolysis; Microvascular Decompression; Trigeminal Nerve Tethering; Trigeminal Neuralgia

Introduction

Trigeminal herpes zoster commonly causes postherpetic trigeminal neuralgia (PHTN), which is characterized by persistent burning pain, sensory disturbances, and allodynia resulting from inflammatory and degenerative changes of the trigeminal nerve [1]. Most patients are managed conservatively because PHTN is considered a neuropathic pain syndrome resulting from varicella-zoster virus-induced trigeminal nerve injury. In contrast, paroxysmal electric shock-like pain that fulfills the clinical characteristics of classical trigeminal neuralgia (TN) is uncommon following herpes zoster infection, and its underlying pathophysiology remains poorly understood [2].

Classical TN is generally caused by neurovascular compression at the trigeminal nerve root entry zone [3,4]. However, neurovascular contact is also observed in asymptomatic individuals, suggesting that vascular contact alone is insufficient to explain the development of symptomatic TN [4]. Recent studies have suggested that structural abnormalities of the trigeminal nerve, including deformation and chronic inflammatory changes, may contribute to trigeminal nerve hyperexcitability and pain generation [3,4].

Herpes zoster infection can induce chronic inflammatory changes, including demyelination, fibrosis, and adhesion around the trigeminal nerve [1,5]. We speculate that these inflammatory changes may result in trigeminal nerve tethering and deformation, rendering pre-existing neurovascular contact symptomatic and producing clinical manifestations indistinguishable from classical TN.

We report a rare case of PHTN presenting with classical TN-like symptoms associated with severe perineural adhesions, trigeminal nerve deformity, and neurovascular contact. The patient was successfully treated with microvascular decompression (MVD), perineural dissection, and internal neurolysis (IN). This case provides insight into a possible mechanism by which chronic inflammation may convert otherwise asymptomatic neurovascular contact into symptomatic TN.

Case Presentation

The patient was a 65-year-old woman who developed herpes zoster in the left V1 region 18 months previously. Since then, she had experienced paroxysmal electric shock-like pain in the left V1–V2 regions triggered by washing her face. She was diagnosed with TN at a local clinic and was treated with medication, which initially controlled her symptoms; however, her condition became refractory to medical treatment, and she was referred to our department requesting surgical treatment. Her medical history included well-controlled hypertension. No relevant family history was noted. Her Barrow Neurological Institute (BNI) pain intensity score [6] was grade IV. Facial pain severely limited her daily activities. No other neurological deficits were observed including facial numbness.

Magnetic resonance imaging (MRI) (Figure 1) revealed no neoplastic lesions in the left cerebellopontine angle. The superior cerebellar artery (SCA) was in contact with the trigeminal nerve from the cranial and dorsal directions. Furthermore, a vein appeared to be running along the ventral aspect of the nerve, causing trigeminal nerve deformity. Given the possibility of chronic inflammatory changes associated with PHTN and the preoperative trigeminal nerve deformity, we planned to perform additional IN if sufficient improvement of the nerve deformity could not be achieved by vascular transposition alone.

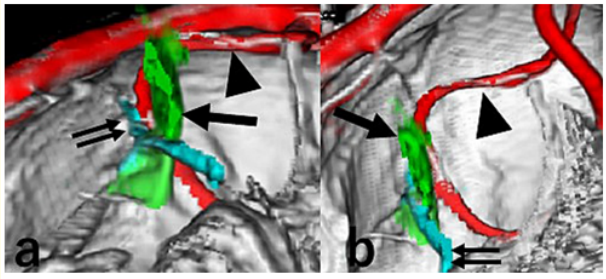

Figure 1: Preoperative magnetic resonance imaging findings.

a: View from the left side. The trigeminal nerve (arrow) was compressed by a vein (double arrows) from the ventral side, while the superior cerebellar artery (arrowhead) coursed dorsally; marked trigeminal nerve deformity was present.

b: Dorsal view of the trigeminal nerve (arrow), demonstrating contact with the superior cerebellar artery (arrowhead).

Microvascular decompression (MVD) was performed in the lower right lateral recumbent position using a left retrosigmoid approach. Auditory brainstem responses and trigeminal nerve conduction were monitored intraoperatively. The horizontal

fissure was opened to expose the trigeminal nerve root entry zone. The SCA contacted the dorsal aspect of the trigeminal nerve without marked compression (Figure 2a), and the trigeminal nerve was severely adherent to thickened arachnoid membrane. Baseline trigeminal nerve conduction was recorded before surgical manipulation. Severe adhesions surrounding the trigeminal nerve were sharply and meticulously dissected, and granulation tissue firmly adherent to the nerve was excised for histopathological examination. The SCA was then transposed cranially and fixed to the tentorium using fibrin glue. A vein running along the ventral aspect of the nerve was carefully dissected and mobilized as much as possible. Despite successful vascular decompression, marked trigeminal nerve deformity persisted (Figure 2b). Therefore, IN was additionally performed by making three longitudinal epineurial incisions along the trigeminal nerve (Figure 2c). After IN, the nerve deformity improved substantially. Trigeminal nerve conduction showed unchanged latency and a 183% increase in amplitude compared with baseline. Histopathological examination (Figure 3) demonstrated fibrosis with lymphocytic infiltration, consistent with chronic inflammatory changes.

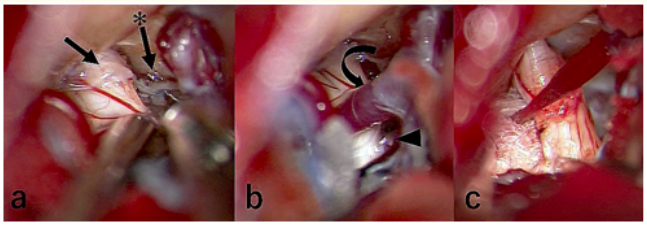

Figure 2: Intraoperative findings.

a: Thickened arachnoid membrane (arrows) was densely adherent to the trigeminal nerve. The adhesions were dissected, and a specimen of the thickened tissue (*) was obtained for histopathological examination.

b: A vein (curved arrow) compressed the ventral aspect of the trigeminal nerve at the cisternal portion, while the superior cerebellar artery (arrowhead) contacted the dorsal aspect; the nerve was markedly deformed.

c: After transposition/mobilization of the surrounding vessels and internal neurolysis, trigeminal nerve deformity improved substantially.

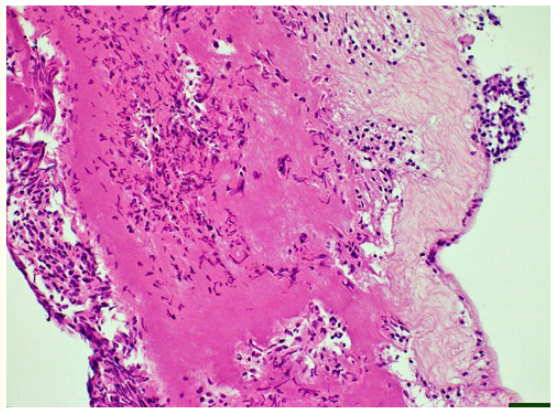

Figure 3: Histopathological findings.

Histopathological examination demonstrated fibrosis with lymphocytic infiltration, consistent with chronic inflammatory changes.

Postoperatively, pain in the first and second divisions of the left trigeminal nerve was immediately relieved without apparent complications. Her BNI pain intensity score [6] improved from grade IV to grade II. Postoperative MRI (Figure 4) indicated successful vascular decompression and improvement of trigeminal nerve deformity. No recurrence was observed during the 6-month follow-up period.

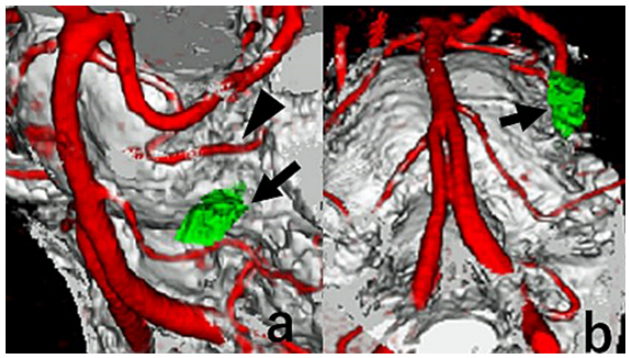

Figure 4: Postoperative magnetic resonance imaging findings.

a: View from the left side. The superior cerebellar artery (arrowhead) had been transposed dorsally, with resolution of contact with the trigeminal nerve (arrow).

b: Inferior-to-superior view demonstrating improvement of trigeminal nerve deformity (arrow).

Discussion

PHTN is typically characterized by persistent burning pain, sensory disturbances, and allodynia resulting from varicella-zoster virus-induced trigeminal nerve injury [1]. In contrast, classical TN is characterized by paroxysmal electric shock-like pain triggered by innocuous stimuli and is most commonly associated with neurovascular compression [2-4]. Li., *et al*. reported favorable outcomes after MVD in selected patients with trigeminal neuralgia following herpes zoster; however, trigger-induced pain was present in only 21.7% of their patients, whereas persistent facial pain and sensory disturbances predominated [5]. The mechanism by which herpes zoster may produce a classical TN-like phenotype therefore remains unclear [1,5].

The present patient had trigger-induced electric shock-like pain typical of TN. MRI demonstrated trigeminal nerve deformity associated with neurovascular contact, whereas surgery revealed thickened arachnoid, dense perineural adhesions, and granulation tissue firmly adherent to the nerve. Histopathological examination showed lymphocytic infiltration and fibrosis. Taken together, these findings support the presence of chronic inflammatory changes affecting the trigeminal nerve and its surrounding tissues [1,5].

Herpes zoster can produce demyelination, axonal injury, inflammatory cell infiltration, and fibrotic changes involving the trigeminal system [1,5]. We hypothesize that persistent fibrosis and adhesion formation may restrict nerve mobility, producing tethering and structural deformation. Neurovascular contact alone does not invariably cause TN because similar vascular relationships can be observed in asymptomatic individuals [3,4]. In the present case, vascular transposition and perineural dissection alone did not restore the normal nerve configuration; morphological improvement was obtained only after additional IN. This observation raises the possibility that chronic inflammatory tethering increased the susceptibility of the trigeminal nerve to otherwise clinically silent neurovascular contact.

The present findings contrast with our previously reported patient who developed PHTN 24 years after herpes zoster infection [7]. That patient had persistent facial pain and numbness with marked trigeminal nerve atrophy but without severe perineural adhesions [7]. The current patient developed classical TN-like symptoms 18 months after herpes zoster and showed severe adhesions, nerve deformity, and histological evidence of chronic inflammation. Although conclusions cannot be drawn from two cases, these observations suggest that postherpetic trigeminal pain may encompass different structural stages or phenotypes, ranging from inflammatory adhesion and tethering to later axonal degeneration and nerve atrophy.

The electrophysiological findings provide additional information regarding functional recovery. In our previous series of TN without apparent neurovascular compression, trigeminal nerve conduction improved after IN in 12 of 14 patients (85.7%), and specific patterns of electrophysiological improvement were associated with favorable pain outcomes [8]. These findings suggested that intraoperative conduction changes may provide a physiological correlate of trigeminal nerve release [8].

In the present patient, latency remained unchanged while trigeminal nerve conduction amplitude increased by 183% after IN. This pattern suggests improved recruitment or conduction efficiency rather than faster neural transmission. Importantly, the electrophysiological improvement occurred in parallel with morphological restoration of the nerve and postoperative pain relief. Although causality cannot be established from a single case, the concordance of these findings supports the concept that releasing a chronically tethered nerve may improve both its structure and function.

IN is an established surgical option for selected patients with TN without neurovascular compression [9]. Our recent studies of IN and superficial epineurial incision have also suggested that procedures directed at the trigeminal nerve itself may provide pain relief when apparent vascular compression is absent [8,10,11]. In the present case, however, IN was not performed as an alternative to MVD; it was added because substantial nerve deformity persisted despite vascular transposition and perineural dissection. Thus, its role was primarily to release residual nerve constriction and restore nerve morphology.

Accordingly, the combination of perineural dissection, MVD, and IN can be viewed as a comprehensive nerve-release strategy addressing both vascular contact and inflammatory tethering. This approach should not be generalized to all patients with PHTN, but it may be considered in carefully selected patients with classical TN-like symptoms, demonstrable neurovascular contact, and persistent intraoperative nerve deformation.

This case supports a multifactorial model in which neurovascular contact, chronic inflammation, trigeminal nerve tethering, and impaired neural conduction may interact to generate pain [1,4]. The principal clinical lesson is that, in patients with classical TN-like pain after herpes zoster, the surgeon should assess not only vascular compression but also perineural adhesion and persistent nerve deformation. Further cases are required to determine whether this inflammatory-tethering phenotype represents a reproducible surgical entity.

Limitations

This report has several limitations. First, it describes a single patient, and therefore the findings cannot establish a causal relationship between postherpetic inflammation, trigeminal nerve tethering, and the patient's pain. Second, the relatively short 6-month follow-up period limits assessment of the long-term durability of pain relief following internal neurolysis. Third, pre-zoster imaging was not available; therefore, we cannot determine whether the observed trigeminal nerve deformity developed after herpes zoster or was pre-existing. Similarly, we cannot determine whether the neurovascular contact observed in this patient had previously been asymptomatic. Finally, the favorable outcome in this individual case does not establish the generalizability or efficacy of internal neurolysis for other patients with postherpetic trigeminal neuropathic pain. Further clinical experience and larger case series with longer follow-up will be necessary to clarify the potential role of internal neurolysis in this setting.

Conclusion

Chronic inflammatory changes following herpes zoster may contribute to trigeminal nerve tethering, deformation, and impaired neural conduction, potentially rendering otherwise asymptomatic neurovascular contact symptomatic. In this patient, perineural dissection, vascular decompression, and internal neurolysis were followed by morphological and electrophysiological improvement and marked pain relief. Recognition of inflammatory tethering may therefore be important when evaluating selected patients with classical TN-like symptoms after herpes zoster infection.

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