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Case Report

A Surgical Case of Postherpetic Trigeminal Neuralgia

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Abstract

Reports on surgical treatment for postherpetic trigeminal neuralgia have not been encountered. Hence, we described a case of postherpetic trigeminal neuralgia treated with microvascular decompression and obtained positive outcomes. We also included a literature review of the surgical findings. We reported a case involving a 70-year-old man who had herpes zoster infection in the region of the third branch territory of the right trigeminal nerve for 24 years prior with persistent burning pain and numbness in the region without a trigger. His initial medical therapy provided by the family physician was no longer effective. Facial pain and numbness substantially reduced his quality of life. Therefore, he presented to our department for further treatment. His Barrow Neurological Institute pain intensity score was IV. Magnetic resonance imaging revealed trigeminal nerve compression by the superior cerebellar artery. Written informed consent was obtained, and microvascular decompression was performed using the right retrosigmoid approach. No substantial trigeminal nerve compression by the superior cerebellar artery was observed. However, the trigeminal nerve was atrophied. Subsequently, the superior cerebellar artery was moved to decompress the trigeminal nerve. Internal neurolysis was performed as an additional procedure because the degree of compression was less severe. Postoperative facial pain and numbness improved. Surgical treatment of postherpetic trigeminal neuralgia may be considered based on careful review, informed consent, and precise surgical techniques.

Keywords: Herpes Zoster; Trigeminal Neuralgia; Microvascular Decompression; Trigeminal Nerve

Abbreviations

TN: Trigeminal neuralgia; QOL: Quality of Life; MVD: Microvascular Decompression; BNI: Barrow Neurological Institute Pain Intensity; MRI: Magnetic Resonance Imaging; SCA: Superior Cerebellar Artery; IN: Internal Neurolysis

Introduction

Postherpetic trigeminal neuralgia (TN) is characterized by unilateral facial pain, which persists for at least 3 months after herpes zoster infection and is generally expected to improve within 6 months to 1 year. However, managing it can be challenging, leading to chronic pain and substantially reducing the quality of life (QOL) [1,2]. Postherpetic TN treatment is mainly conservative [1,2], with a few reports of surgical treatment [3,4]. Furthermore, no reports have detailed these surgical findings. We presented a TN case of a patient who had herpes zoster infection in the right trigeminal nerve 24 years prior and underwent microvascular decompression (MVD) with positive results.

Case Presentation

A 70-year-old man presented to our department with persistent burning pain and numbness in the right trigeminal nerve and

no pain triggers. Twenty-four years prior, he was diagnosed with herpes zoster infection characterized by persistent right facial pain occurring in the third branch of the right trigeminal nerve. He was placed on several medications and followed up by his family doctor. He anticipated undergoing other possible treatments because his medications were no longer effective. His medical history included cerebral infarction, cerebral hemorrhage, and well-controlled hypertension. No family history was observed. Barrow Neurological Institute pain intensity (BNI) score [5] was grade IV. The patient experienced facial pain that severely limited his daily activities. No other obvious symptoms of neurological deficits were observed.

Magnetic resonance imaging (MRI) uncovered no neoplastic lesions in the right cerebellar pontine angle, and the superior cerebellar artery (SCA) was in contact with the right trigeminal nerve (Figure 1A). MVD was considered because a TN due to vascular compression was observed. Written informed consent was obtained for the surgery. MVD was performed in the lower left lateral recumbent position using a right retrosigmoid approach. Auditory brainstem responses were continuously monitored intraoperatively. The horizontal fissure was opened to reach the trigeminal nerve origin. The SCA ran dorsal to the trigeminal nerve in contact but without compression (Figure 2A). Additionally, the trigeminal nerve appeared atrophied (Figure 2A). No substantial adhesions were observed around the trigeminal nerve. The SCA was moved cranially and adhered to the cerebellar tent using fibrin glue to decompress the trigeminal nerve (Figure 2B). Internal neurolysis (IN) was performed because the SCA was in contact with the trigeminal nerve without compression (Figure 2C). IN is an attempt to improve TN by artificially damaging the trigeminal nerve in the long-axis direction [7]. In this case, three long-axis injuries to the trigeminal nerve were induced using an isolator (Fujita Medical Instruments co., LTD. Tokyo, Japan). The surgery was completed with sufficient decompression of the trigeminal nerve (Figure 2D).

Postoperatively, the pain and numbness in the third branch of the patient's right trigeminal nerve were reduced immediately. His BNI score [5] improved to grade II. No apparent complications were observed. Postoperative MRI confirmed no contacting SCA (Figure 1B). No recurrence was observed at one year postoperatively.

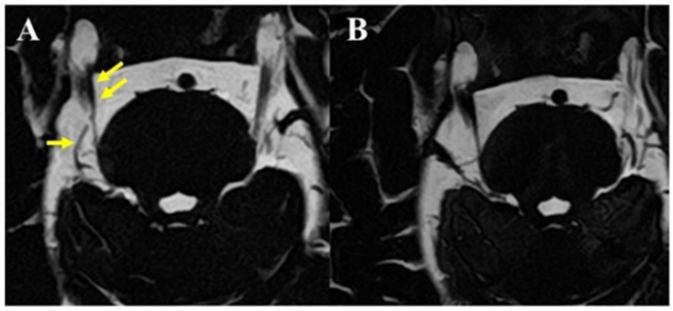

Figure 1: Magnetic Resonance imaging.

- A: Preoperative magnetic resonance imaging (constructive interference in steady state) The superior cerebellar artery (arrow) runs around the trigeminal nerve (double arrow).
- B: Preoperative magnetic resonance imaging (constructive interference in steady state the superior cerebellar artery was demonstrated not to be in contact with the trigeminal nerve.

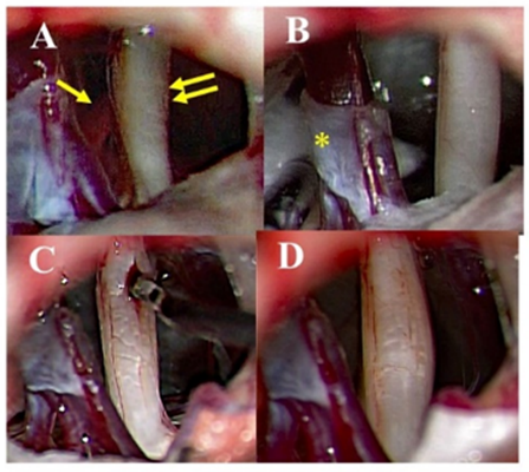

Figure 2: Operative views.

- A: The superior cerebellar artery (arrow) is in contact with the trigeminal nerve (double arrow) without compression.
- B: The responsible vessel is moved to the tentorial side and not in contact with the trigeminal nerve using Teflon felt (*).
- C: Internal neurolysis was carefully performed.
- D: Final image after moving the responsible vessel and performing internal neurolysis.

Discussion

Postherpetic TN is characterized by pain on one side of the face caused by herpes zoster, persisting for ≥3 months or occurring repeatedly. It may be accompanied by various sensory disturbances [1,2] and is associated with the transition from nociceptive pain caused by herpes zoster in the acute phase to neuropathic pain [1,2]. It occurs in 10–20% of herpes zoster infection cases [1,2]. The first choice of treatment is medical therapy [1,2]; when these are ineffective, nerve blocks [7] and gamma knives [8] may be employed. Many cases improve within 6 months to 1 year. However, long-term prolonged pain owing to treatment resistance might occur, significantly reducing QOL [1.2]. MVD is the gold standard treatment for TN globally [9]. However, MVD is not generally accepted for postherpetic TN and has only been reported in a few cases [3,4]. The only detailed report of MVD for postherpetic TN was by Li., *et al*. [3], which included 23 cases and described their clinical characteristics and surgical findings [3]. Patients with typical TN often have pain triggers as a clinical manifestation [9,10]; however, only five patients (21.7%) exhibited triggers. In many cases, triggers were not present [3]. Typical TN presents with paroxysmal electrical pain [10,11]; however, 80% of patients have atypical facial pain-persistent pain [3]. In our case, the pain in the trigeminal nerve third branch area was persistent, accompanied by numbness without triggers, and could be considered an atypical facial pain. Li., *et al.* [3] confirmed the vessels responsible for trigeminal nerve decompression via MRI in all cases before proceeding to MVD. The responsible vessel was the SCA in 15 cases (65.2%), followed by venous involvement in five cases (21.7%) [3]. All patients underwent sufficient trigeminal nerve decompression. The operative findings revealed nerve atrophy; however, no severe vascular nerve compression was observed [3]. Histopathological examination of the trigeminal nerve in postherpetic TN revealed inflammatory cell infiltration in the acute stage and nerve fiber demyelination and fibrosis in the chronic stage [11], consistent with the surgical findings of Li., *et al*. [3] for nerve atrophy. Additionally, neuropathy with slight vascular compression caused by herpes zoster infection-demyelination of the trigeminal nerve-caused TN. In this case, the responsible vessel was in contact with the trigeminal nerve but was not under pressure. Nerve atrophy was also observed. In contrast, Ramiro López-Elizalde., *et al*. [4] reported the surgical findings of a postherpetic TN case, in which no vascular compression was observed; however, arachnoiditis and adhesions around the trigeminal nerve were confirmed. This indicates that adhesion may cause postherpetic TN development. The exact mechanism of postherpetic trigeminal neuralgia is controversial and requires further study. Nineteen patients (82.6%) were successfully treated, and no recurrence was observed at the 1-year follow-up, demonstrating the efficacy of surgery and recommending aggressive surgical treatment based on detailed imaging diagnosis [3]. In this case, the responsible vessel did not cause significant trigeminal nerve compression; therefore, IN was used for manipulation. TN can be caused by adhesion or nerve deformity without vascular compression [10]. Vascular displacement cannot be performed in such cases; however, IN showed positive results [6,7]. IN is a physical procedure that involves damage to the trigeminal nerve at approximately four longitudinal axial locations. However, the exact mechanism is unclear, and future research is needed. No major complications were observed [6]. We consider adding IN in TN cases without vascular compression or in cases of less severe compression. Nonetheless, whether the favorable results, in this case, were because of IN remains unclear.

The incidence of herpes zoster infection has increased in recent years, especially among the geriatric population [1,2]. Furthermore, postherpetic TN cases will likely occur more frequently in outpatient units. Postherpetic TN substantially reduces QOL when conservative treatment is unsuccessful [1,2], and surgical treatment is considered based on a detailed imaging diagnosis. The pathogenesis of postherpetic TN should be elucidated through frequent case reviews in the future and the establishment of treatments based on the essential aspects of the disease.

Conclusion

Effective treatment for postherpetic TN has not yet been established. We reported a surgical approach as a treatment option; however, more case reviews are expected to accumulate, the true mechanism of postherpetic TN will be identified, and subsequently, essential treatment will be provided.

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Conflict of Interest

The authors declare that they have no conflict of interest.

Ethics Approval

The Ethical Committee of the International University of Health and Welfare approved all procedures used in this research.

Submission Statement

This original manuscript has not been submitted elsewhere in part or whole.

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