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Original Article

Microvascular Decompression Combined with Nerve Combing for Atypical Trigeminal Neuralgia

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Abstract

Background and objectives: Atypical trigeminal neuralgia (ATN) is a chronic pain condition characterized by persistent facial pain that does not respond well to conventional medical treatments, often leading to significant impairment in quality of life. This study examined the clinical characteristics and surgical outcomes of microvascular decompression combined with nerve combing in patients with ATN.

Methods: We conducted a retrospective analysis of surgical techniques, clinical data, and treatment outcomes in 40 patients from January 2009 to January 2018. Pain levels and patient prognoses were assessed using the Visual Analog Scale and the Barrow Neurological Institute (BNI) pain score. Dynamic monitoring of arterial blood pressure was performed, and levels of total adrenaline, norepinephrine, and dopamine were measured before and during the nerve combing procedure.

Results: During surgery, veins combined with arachnoid adhesions and arachnoid adhesions alone were observed compressing the trigeminal nerve in seven patients (17.50%) and 33 patients (82.50%), respectively. Immediate postoperative BNI scores indicated excellent outcomes (P = 2) in 30 patients (75.00%) and good outcomes (P = 3) in four patients (10.00%). Long-term postoperative BNI scores showed excellent outcomes (P = 2) in 25 patients (62.50%) and good outcomes (P = 3) in seven patients (17.50%). All patients experienced an increase in arterial blood pressure during nerve combing, and the mean levels of adrenaline and norepinephrine before combing showed significant improvement (P < 0.05).

Conclusion: Microvascular decompression combined with nerve combing achieves favorable results in treating ATN. Long-term trigeminal nerve compression and central sensitization may contribute to the etiology in these patients.

Introduction

Trigeminal neuralgia (hereinafter referred to as TN) refers to severe paroxysmal pain in the distribution area of the trigeminal nerve. It can be divided into classical trigeminal neuralgia (CTN) and atypical trigeminal neuralgia (ATN).[1] Clinical symptoms are mainly facial pain, with significant differences between the two types. CTN is characterized by electric shock-like or needle-like sharp pain in one or more branches of the trigeminal nerve, caused by unknown factors, non-intracranial tumors, or abnormal bone compression. This pain typically lasts only a few seconds, and the patient experiences no significant discomfort during the intervals between pain episodes. Most patients report fixed pain trigger points at the corner of the mouth, nose, or cheek. Oral carbamazepine or other medications are often effective for pain relief or control.[2] In contrast, ATN primarily presents as aching, burning, or indescribable pain in the trigeminal nerve distribution area. Patients typically experience prolonged pain episodes lasting several hours, often without relief or significant resting periods. The trigger point for ATN is usually unclear or generalized, making it difficult to alleviate with medications like carbamazepine. The exact cause of ATN remains unclear, and the diagnosis of CTN and ATN is primarily based on these clinical manifestations.

CTN is commonly treated with microvascular decompression (MVD), which involves relieving neurovascular conflict (NVC) by separating compressing blood vessels from the trigeminal nerve. In

Keywords: Atypical trigeminal neuralgia; Microvascular decompression; Nerve combing; Arterial blood pressure; Outcome; Chronic facial pain; Trigeminal nerve compression; Central sensitization; Neurosurgery.

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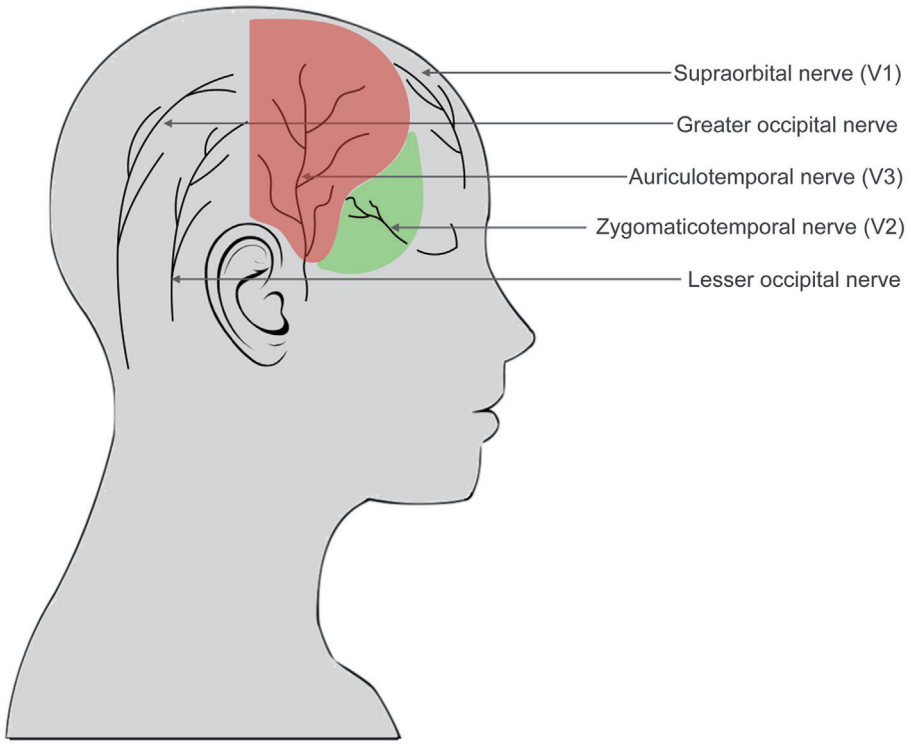

Fig. 1. Illustration of pain sites in patients with atypical trigeminal neuralgia (ATN). Green indicates type A patients with pain in the skin over the anterior temple; red indicates type B patients with pain in the temple at or behind the hairline.

contrast, ATN presents with aching, burning, or indescribable pain, and patients typically experience prolonged pain episodes lasting several hours without relief or significant resting periods.[3,4] The trigger point for ATN is often unclear, and the pain may involve the zygomaticotemporal and auriculotemporal nerves. The etiology of ATN is less well understood compared to CTN and may involve long-term trigeminal nerve compression and central sensitization. Treating ATN is more challenging than CTN due to additional potential factors beyond NVC, such as damage to nerve axons, which can be difficult to recover after compression relief. Additionally, ATN has a higher recurrence rate of pain and often requires alternative surgical approaches, such as nerve combing, when no definite responsible vessels are found or decompression is unattainable due to specific vascular compression.[5]

Temporal ATN might be associated with the zygomaticotemporal and auriculotemporal nerves.[6] When stimulated, the trigeminal nerve produces pain signals transmitted to the trigeminal spinal nucleus's caudal area and then to the cerebral cortex via the brainstem and thalamus, generating the sensation of pain. Trigeminal nerve decompression or MVD is the primary treatment method for CTN in clinical practice, mainly providing pain relief by alleviating NVC.[1] However, in ATN cases, additional factors beyond NVC or significant damage to nerve axons can make recovery challenging, resulting in a worse long-term prognosis for ATN than for CTN and a higher recurrence rate of pain. Trigeminal nerve combing is considered an alternative procedure when no definite responsible vessels are identified, or decompression cannot be achieved due to specific vascular compression.[1,2] Additionally, a recent study by our team indicated that changes in arterial blood pressure during trigeminal nerve combing in MVD were correlated with the prognosis of CTN patients.[7] To our knowledge, few studies have examined the effect of MVD combined with nerve combing in treating ATN.

This study focuses on a group of patients with ATN, characterized by unilateral temporal pain, as defined by the International Classification of Headache Disorders, 3rd edition (ICHD-3).[8] We categorized these cases based on the innervation of the pain location. The objective was to explore the clinical features and surgical effectiveness of MVD and nerve combing, as well as examine the impact of arterial blood pressure changes during the trigeminal nerve combing procedure in these patients.

Materials and methods

Patients

This was a retrospective study in which clinical data were collected from 56 patients with ATN between January 2009 and January 2018 at Peking University People's Hospital. Diagnoses and surgeries were performed by the corresponding author. Six patients were excluded due to secondary conditions, including a history of craniotomy, facial plastic surgery, or orbital fracture. Based on pain distribution, patients were categorized into two groups: (A) pain over the anterior temple skin and (B) pain at or behind the hairline (Fig. 1). To focus on evaluating pure MVD combined with nerve combing for ATN, we excluded 10 patients with involvement of the greater occipital nerve and/or the lesser occipital nerve who required greater occipital nerve or lesser occipital nerve blocks. The diagnosis of ATN followed ICHD-3 criteria: (1) recurrent unilateral facial pain meeting CTN criteria; (2) persistent moderate-intensity facial pain in the affected area; (3) another ICHD-3 diagnosis to explain the symptoms was not applicable. To confirm the diagnosis, 1% lidocaine was used to block the auricu-

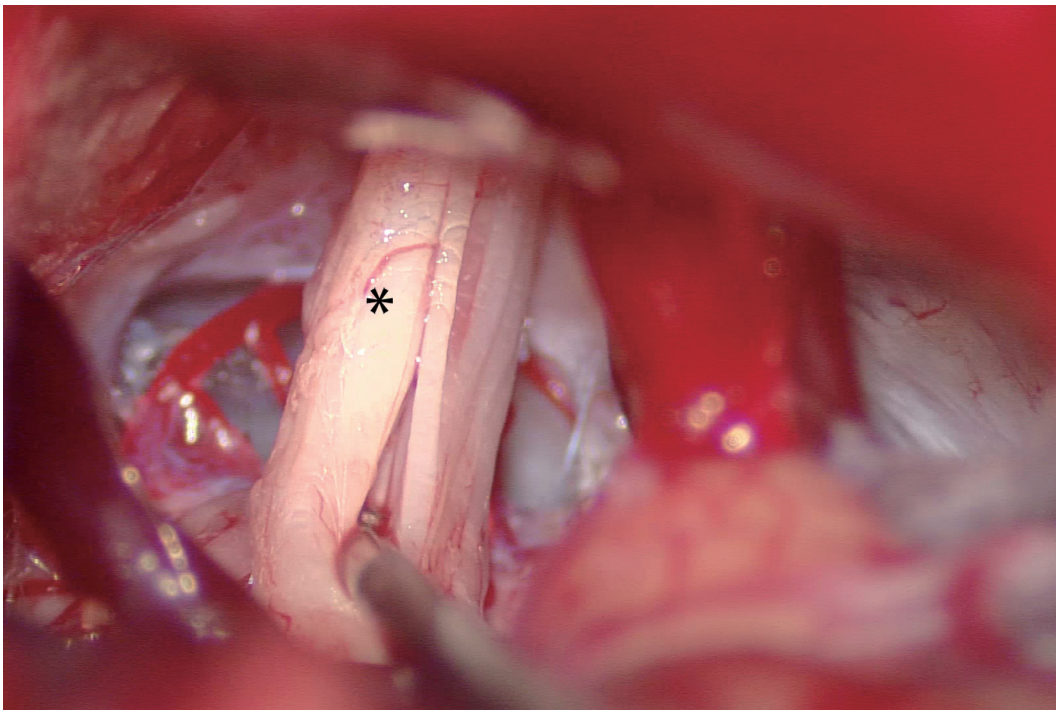

Fig. 2. Nerve combing of the trigeminal nerve. *, trigeminal nerve.

lotemporal or zygomaticotemporal nerves on the affected side. If the treatment was effective, ATN was diagnosed. This study was conducted in accordance with the principles of the Declaration of Helsinki and Good Clinical Practice Guidelines and was approved by the Ethics Review Committee of Peking University People's Hospital (2020PHB033-02). The individual consent for this retrospective analysis was waived.

Magnetic resonance imaging (MRI)

All patients underwent a preoperative MRI examination, which included 3D T1- and T2-weighted high-resolution sequences for detailed visualization of the trigeminal nerve and vascular structures. The 3D time-of-flight magnetic resonance angiography was used to visualize vessels with high flow, primarily arteries. Diffusion tensor imaging was employed to assess for multiple sclerosis. MRI was also used to identify any pathology of the joint or infratemporal fossa; patients with such conditions were excluded.

Operative technique

The operative and anesthesia techniques followed those described in previous studies by our team.[7,9] After inducing general anesthesia, patients were positioned in the lateral park bench position with three-point fixation, and a retrosigmoid craniotomy was performed. If small tributaries of the petrosal veins (PVs) were identified as the responsible vessels, they were cauterized and divided. Arachnoid adhesions (AA) compressing the trigeminal nerve were removed. All patients underwent MVD with nerve combing. During nerve combing, the trigeminal nerve was longitudinally divided along its fibers into four to five bundles from the root entry zone to the petrous bone using a special knife with a 0.90 mm cutting edge (Fig. 2). Levels of total adrenaline (AD), norepinephrine (NE), and dopamine were measured before and during the nerve combing procedure. Brainstem auditory evoked potentials and selective cranial nerve electromyography were used to monitor and protect at-risk neural structures in all patients.

Data collection

Baseline and medical history data were collected from medical records, including age, sex, prior treatments, and history of hypertension. The Visual Analog Scale pain scores were used to evaluate pain changes from preoperative to postoperative periods on an 11-point scale, where zero represents no pain and ten represents the worst possible pain. Postoperative outcomes were assessed using the Barrow Neurological Institute (BNI) pain intensity score, which evaluates pain relief as follows: (1) No pain, no medication; (2) Occasional pain, not requiring medication; (3) Some pain, adequately controlled with medication; (4) Some pain, not adequately controlled with medication; (5) Severe pain/no pain relief. Follow-up data for three or more years were available for all 32 patients (range: 3–11.8 years; mean: 6.8 years; median: 4.9 years).

An arterial cannula was inserted into the radial artery to monitor invasive arterial blood pressure in the upper arm before, during, and after trigeminal nerve combing. To ensure consistency, noninvasive arterial blood pressure was measured with an inflatable cuff twice on both sides at the beginning and end of each measurement.[7]

Statistical analysis

Data analysis was conducted using SPSS statistical software 19.0 (IBM Corp., Armonk, NY, USA). Numerical variables are presented as mean ± standard deviation, and qualitative variables are reported as the absolute number of cases in each group. Statistical significance for quantitative variables was assessed using the χ^2 test, with Yates's or Fisher's correction applied when necessary. The Student's t-test was used to evaluate data following a normal distribution. A *P*-value of less than 0.05 indicated significant differences between groups.

Results

Baseline characteristics

The baseline characteristics of the study participants are detailed in Table 1. The cohort comprised 40 patients, including 10 males and 30 females, with ages ranging from 22 to 56 years, yielding a mean age of 36.20 ± 8.13 years. Notably, seventeen patients exhibited left-sided symptoms, and no instances of bilateral disease were observed. The average duration of illness was 7.38 ± 4.41 years. All patients demonstrated resistance to prolonged, high-dose carbamazepine therapy. Before undergoing MVD, ten patients (25.00%) had received alternative surgical interventions, such as alcohol block and radiofrequency gangliolysis of the trigeminal nerve.

Among the participants, nineteen patients (47.50%) were classified as Type A, characterized by pain in the skin over the anterior temple. Conversely, twenty-one patients (52.50%) belonged to Type B, experiencing pain in the temple at or posterior to the hairline. A history of hypertension was reported by three patients (7.50%). During trigeminal nerve manipulation, all patients exhibited an elevation in arterial blood pressure, with increases ranging from 24 to 80 mmHg, averaging 49.77 ± 16.43 mmHg. Preoperatively, the Visual Analog Scale pain score averaged 9.38 ± 0.79.

Operation outcomes

Within the operative field, AA and PVs were identified as the offending vessels in seven patients (17.50%) (Fig. 3). In contrast, AA alone was found to compress the trigeminal nerve in thirty-three patients (82.50%) (Fig. 4). No arteries were implicated in compressing the trigeminal nerve. The compression observed from AA or veins was characterized by abutment without indenting or distorting the nerve.

Immediate postoperative BNI scores indicated excellent outcomes ($P = 2$) in thirty patients (75.00%), good outcomes ($P = 3$) in four patients (10.00%), and fair outcomes ($P = 4$) in six patients (15.00%), translating to an effective pain relief rate of 85.00%.

Table 1. Clinical characteristics

Characteristic	Patients
Age -yr	36.20 ± 8.13
Female sex – no. (%)	30 (75.00%)
Duration of ATN -yr	7.38 ± 4.41
Left side – no. (%)	17 (42.50%)
History of hypertension	3 (7.50%)
Compressing reason	
AA	33 (82.50%)
AA+PV	7 (17.50%)
Type	
Type A	19 (47.50%)
Type B	21 (52.50%)
The VAS pain score before the MVD	9.38 ± 0.79
Immediate postoperative BNI scores	
Excellent ($P = 2$)	30 (75.00%)
Good ($P = 3$)	4 (10.00%)
Fair ($P = 4$)	6 (15.00%)
Long-term postoperative BNI scores	
Excellent ($P = 2$)	25 (62.50%)
Good ($P = 3$)	7 (17.50%)
Fair ($P = 4$)	6 (15.00%)
Poor ($P = 5$)	2 (5.00%)

AA, arachnoid adhesions; ATN, atypical trigeminal neuralgia; BNI, Barrow Neurological Institute pain intensity score; MVD, microvascular decompression; PV, petrosal vein; VAS, Visual Analog Scale.

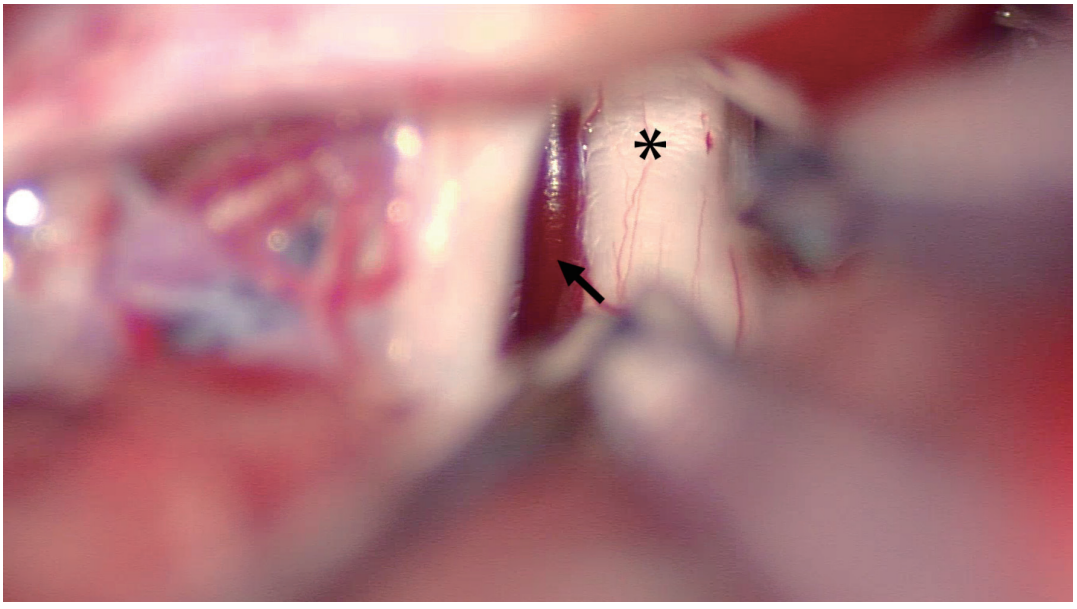

Fig. 3. Veins identified in the operative field during MVD. *, trigeminal nerve; arrow, tributaries of the petrosal vein. MVD, microvascular decompression.

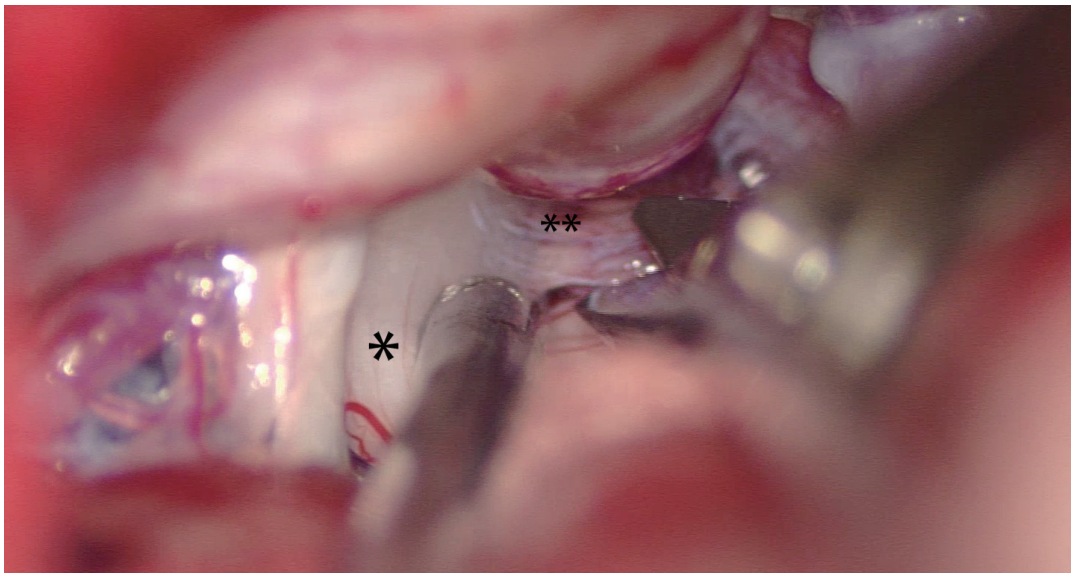

Fig. 4. Arachnoid adhesions (AA) compressing the trigeminal nerve observed during MVD. *, trigeminal nerve; **, arachnoid. MVD, microvascular decompression.

Comprehensive follow-up data, spanning three to 11.8 years (mean: 6.8 years; median: 4.9 years), were available for all patients. Thirty-one patients (77.50%) reported subjective numbness or hypesthesia on the affected facial side immediately postoperatively, which gradually improved during follow-up. No additional complications were noted. Long-term postoperative BNI scores were excellent (P = 2) for twenty-five patients (62.50%), good (P = 3) for seven patients (17.50%), fair (P = 4) for six patients (15.00%), and poor (P = 5) for two patients (5.00%). The long-term effective pain relief rate stood at 80.00% (Table 2).[10–14]

Biochemical outcomes

All patients experienced an increase in arterial blood pressure during trigeminal nerve manipulation. Pre-manipulation levels of total AD and NE averaged 132.26 ± 26.40 pg/mL and 488.40 ± 152.50 pg/mL, respectively. During manipulation, these values significantly surged to 251.12 ± 50.25 pg/mL for AD and 853.01 ± 235.97 pg/mL for NE, as confirmed by paired Student's t-tests ($P < 0.05$). In contrast, mean dopamine levels were 122.77 ± 25.40 pg/mL before manipulation and 141.30 ± 28.88 pg/mL during manipulation, with no statistically significant difference observed ($P > 0.05$) (Fig. 5).

Complications

No serious complications, such as mortality, complete facial paralysis, intracranial hematoma, or postoperative hearing loss, occurred. Five patients experienced scalp tingling, seven had facial numbness, and three had transient vertigo; all of these issues were resolved with symptomatic and supportive treatment. During the follow-up period, no cranial nerve dysfunction was observed.

Discussion

Our study focuses on patients with ATN, characterized by unilateral temporal pain, which may involve the auriculotemporal and zygomaticotemporal nerves–branches of the trigeminal nerve.[15] The zygomaticotemporal nerve, a terminal branch of the maxillary division of the trigeminal nerve (V2), innervates the anterior temple, corresponding to the pain site in type A patients in this study.[16] The auriculotemporal nerve, a terminal branch of the mandibular

Table 2. Summary of microvascular decompression-treated cases of atypical trigeminal neuralgia

Author/year	Patients	Age	Gender	Reason	Outcome
Roski *et al*., 1982[14]	1	6	Male	Petrosal vein	Excellent (100%)
Tyler-Kabara *et al*., 2002[11]	528	46.5 (median age)	NA	NA	Excellent (46.9%); Good (39.7%); No response (13.4%)
Hai *et al*., 2006[13]	26	65 (median age)	Male (42%)	Superior cerebellar artery (73.1%); vein (42.3%); anterior inferior cerebellar artery (26.9%)	Excellent (50%); Good (30.8%); No response (19.2%)
Li *et al*., 2005[12]	17	55.5 (median age)	NA	Artery (58.82%); artery and venous (41.18%)	Complete (35.29%); Incomplete (64.71)
Gressot *et al*., 2012[10]	1	66	male	Basilar artery	Excellent (100%)

NA, not available.

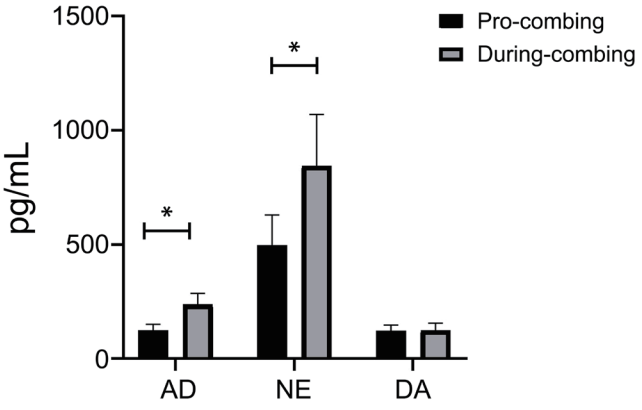

Fig. 5. Total adrenaline (AD), norepinephrine (NE), and dopamine (DA) values, with changes in arterial blood pressure. Values are shown as means ± standard error. *P < 0.05.

division of the trigeminal nerve (V3), innervates the temple skin and area behind the hairline, aligning with the pain site in type B patients (Fig. 1). Additionally, the auriculotemporal nerve innervates the temporomandibular joint (TMJ), parotid gland, external auditory canal, and tympanic membrane. However, none of the patients in this study reported TMJ or ear pain. Since auriculotemporal neuralgia can arise from joint or infratemporal fossa pathology,[17] these secondary causes were excluded through preoperative examination. As branches of the trigeminal nerve, these nerves can manifest as true TN, necessitating similar treatment.[18] After ruling out all secondary pain causes, patients were diagnosed with definite ATN according to the ICHD-3 criteria.[8]

In our study, the mean disease duration was 7.38 ± 4.41 years. This prolonged nerve compression leads to axonal damage and significant myelin sheath degradation, increasing sensory neuron excitability. Notably, no arteries were identified as compressing the trigeminal nerve in our patients; instead, veins and AA were found to be compressing factors during surgery. This suggests that the abnormal conduction hypothesis may not fully explain ATN etiology. Some patients with ATN in our study also experienced symptoms such as eyelid or conjunctival congestion, facial flushing, and nausea and vomiting. We believe these symptoms may be related to activation and sensitization of the trigeminal neurovascular reflex system, where vessel dilation stimulates nerve endings to release neuropeptides, further dilating vessels and causing a rapid inflammatory response. Activated sensory neurons of the trigeminal nerve have functional connections with other critical brainstem nuclei, including nausea and vomiting centers such as the solitary tract nucleus, which can trigger these symptoms.

In this study, all patients had previously received prolonged and high doses of carbamazepine, and some had undergone other surgical treatments, such as alcohol block and radiofrequency ganglliolysis, before MVD. Despite these interventions, the treatments ultimately failed. While MVD is widely accepted for TN treatment, only about half of ATN patients achieve long-term postoperative pain relief. Therefore, it is crucial to find ways to enhance surgical outcomes for ATN patients effectively.

As mentioned, veins and AA were found to compress the trigeminal nerve during surgery. Venous compression is characterized by the close adherence of offending vessels to the nerve. Complete dissection may risk blood vessel rupture, cerebellar or brainstem venous infarction, hemorrhage, or nerve damage. Therefore, while releasing adhesions, it is not always necessary to fully separate the vessels from the nerve; the goal is to relieve compression and restore the natural shape of the trigeminal nerve. In this study, most patients with venous compression had small tributaries of PVs firmly adhered to the trigeminal nerve, making adequate decompression challenging (Fig. 3). These small tributaries were directly cut after electrocoagulation.[19] If AA was found to compress the trigeminal nerve, the adhesions were loosened along the nerve's long axis to achieve full decompression, and any missed responsible arteries were identified and addressed.

Nerve combing is used for patients with intraoperative trigeminal nerve compression without responsible arteries or venous involvement.[7,20] Anatomically, trigeminal nerve fibers from the maxillary division are intermediate, while those from the mandibular division are more dorsal.[21] Therefore, trigeminal nerve decompression can theoretically alleviate pain caused by the auriculotemporal nerve, a branch of the mandibular nerve. Additionally, nerve combing can effectively address pain from the zygomaticotemporal nerve, a branch of the maxillary nerve. Pain relief after combing is thought to result from axonal fracture, where axonal continuity is lost without detaching the surrounding tissue. Unlike neurotomy, combing induces less extensive sensory impairment and allows for gradual recovery.[22,23] In our study, some patients reported subjective numbness or hypesthesia on the affected side of the face immediately after trigeminal nerve decompression; however, these symptoms improved during postoperative follow-up. MVD combined with trigeminal nerve combing has shown good results in treating patients with CTN.[7] The immediate pain relief rate in this ATN study was 85.00%, higher than previously reported in ATN studies. However, the long-term pain relief rate decreased to 80.00%. The high immediate remission rate may be due to short-term neuronal inhibition and halted abnormal electrical conduction from intraoperative stimulation. However, factors beyond vascular compression, such as central sensitization, may also play a role in ATN cases. Additionally, severe axonal damage and difficulty recovering damaged nerve fibers after vascular compression relief may lead to poor long-term outcomes and a higher recurrence rate. There are few reports on using MVD for ATN treatment, with most being case studies.[10] This study is the first to analyze MVD combined with nerve combing for ATN treatment. Compared to previous clinical studies,[11–13] our treatment outcomes are more favorable (Table 2).

In clinical practice, applying this surgical method requires a thorough understanding of the underlying pathophysiology of ATN. As demonstrated in this study, long-term trigeminal nerve compression and central sensitization may be the aetiology of ATN. Therefore, identifying offending vessels or AA and relieving compression on the trigeminal nerve are crucial steps in achieving pain relief. Nerve combing, which involves longitudinally dividing the trigeminal nerve into bundles and gently separating the fibers, may further alleviate pain by disrupting abnormal electrical conduction and promoting axonal healing. However, this combined surgical method is not without risks and challenges. One primary challenge is identifying the offending vessels or AA, which may be difficult to locate in some patients. Additionally, the surgery is technically demanding and requires a high level of surgical skill and experience. Postoperative complications, such as facial numbness, tingling, and transient vertigo, may occur, although these are generally mild and resolve with symptomatic treatment. Despite these risks, the potential benefits of MVD combined with nerve combing in treating ATN outweigh the drawbacks. This combined surgical method may provide lasting pain relief and improve the quality of life for patients with ATN. Thus, clinicians should con-

sider this option for managing patients with ATN, especially those unresponsive to conventional therapies. Future studies are needed to further elucidate the mechanisms of action of this combined surgical method and optimize techniques to minimize complications and improve outcomes.

Our previous study on TN indicated that increased arterial blood pressure observed during nerve combing is related to sympathetic excitation and suggests the procedure's effectiveness.[7] Similarly, this study found comparable results in patients with ATN. Additionally, sympathetic neurotransmitter levels significantly increased during nerve combing compared to pre-combing levels (Fig. 5). This hypertensive response may be due to the activation of a pressor reflex triggered by nerve combing.[7] The disruption of afferent pain regulation and loss of gate control for afferent impulses may result in transmitting signals that rapidly lead to a pain attack. The arterial baroreflex center is located in brainstem cardiovascular areas, such as the nucleus of the solitary tract and the rostral ventrolateral medulla. Therefore, the elevated blood pressure response in ATN patients during nerve combing may partially support the hypothesis of the trigeminal neurovascular reflex and central sensitization.

Due to the low incidence of ATN, the sample size in this study is limited, necessitating further research to understand the mechanisms behind AD, NE, and dopamine responses. Additionally, glycerol, balloon compression, or radiofrequency ablation treatments might be used diagnostically. If patients experience at least transient improvement with these procedures, they may be more likely to respond to nerve combing. However, other teams administered these treatments to patients before they underwent nerve combing, which prevented us from conducting a convincing statistical analysis. We plan to address this issue in future studies. Moreover, we will investigate the relationship between antiepileptic drugs and the response to MVD in future research. While some patients in the study had undergone previous treatments such as alcohol block and radiofrequency gangliolysis, the potential association between these prior treatments and the outcomes of pain relief following MVD and nerve combing was not explored. This is indeed a limitation, as previous interventions could influence the efficacy of the current treatment. Future studies could consider including a larger sample size and specifically examining the effects of prior treatments on the response to MVD and nerve combing. This could involve stratifying patients based on their history of prior treatments and analyzing outcomes accordingly, allowing for a more comprehensive understanding of the factors that influence the success of MVD and nerve combing in treating ATN.

A significant limitation of this study is its retrospective nature. As a retrospective analysis, the study is constrained by the availability and accuracy of previously collected data, which may not always be comprehensive or consistent. This limits the ability to draw definitive conclusions and may introduce biases or confounding factors that are difficult to control. Despite this limitation, the study provides valuable insights into the clinical characteristics and surgical outcomes of MVD combined with nerve combing for ATN.

Conclusions

This study examines a group of patients with ATN who presented with unilateral temporal pain. During surgery, veins and arteries were identified as compressing the trigeminal nerve. Long-term compression of the trigeminal nerve and central sensitization may underlie the etiology in these cases. Combining MVD with trigeminal nerve combing has shown promising results. The observed increase in blood pressure during nerve combing may partially support the hypothesis of a trigeminal neurovascular reflex and central sensitization in ATN patients.

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Conflict of interest

Ruen Liu has served as an Executive Associate Editor of *Neurosurgical Subspecialties* since August 26, 2024. The authors have no other conflicts of interest to declare.

Author contributions

Study concept and design (JL, RL), acquisition of data (JL, RL), analysis and interpretation of data (JL, RL), drafting of the manuscript (JL, RL), critical revision of the manuscript for important intellectual content (GW, BL, JZ, CF, DW, BH, FL, JO, ZL, QH), administrative, technical, or material support (RL), and study supervision (RL). All authors have approved the final version and publication of the manuscript.

Ethical statement

This study was conducted in accordance with the principles of the Declaration of Helsinki and Good Clinical Practice Guidelines and was approved by the Ethics Review Committee of Peking University People's Hospital (2020PHB033-02). The individual consent for this retrospective analysis was waived.

Data sharing statement

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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