## Original Article

# Coexistent neuralgia: stopping NSAIDs abuse and starting microvascular decompression

Rui-zhe Zheng[1], Ting-hua Peng[2], Zeng-xin Qi[1]*

**Abstract**

**Introduction:** The combination of trigeminal neuralgia (TN), glossopharyngeal neuralgia (GPN), and hemifacial spasm (HFS), referred to as combined hyperactive dysfunction syndrome (HDS), is a rare disorder characterized by paroxysmal severe pain and tic convulsions in the innervation region. Interestingly, there is no evidence of the coexistence of TN and GPN with nervus intermedius neuralgia (NIN) in the literature.

**Method:** A 50-year-old man who was surgically treated for two months prior to presentation with paroxysmal pain in the left cheek, tooth root, and tongue, posterior pharyngeal wall, deep ear, and earlobe.

**Case Presentation:** He began experiencing posterior alveolar discomfort a year ago and had his bottom posterior teeth pulled two months ago. It is worth noting that he typically takes ibuprofen orally (0.6~2.4 g per day) to alleviate discomfort associated with paroxysmal condition.

**Results**: Our diagnosis was verified by preoperative magnetic resonance tomographic angiography (MRTA) and intraoperative results. We conducted neurological surgery on these constricted nerves by performing microvascular decompression (MVD). Teflon materials were employed to isolate the artery and nerve enough. TN, GPN, and NIN all ceased to exist immediately after our operation.

**Discussion:** Our case is the first coexisting TN, GPN, and NIN patient who underwent MVD surgery after immediate termination of drug treatment. Ibuprofen therapy on a chronic basis may reduce neuropathic pain but has no neuroprotective impact. Nonsteroidal anti-inflammatory medication usage in the clinic incorrectly may have extra detrimental consequences and disrupt the peripheral neuro-environment of cranial nerves. MVD through the suboccipital retrosigmoid route is an excellent treatment option for such coexisting cranial neuralgia.

**Keywords:** Trigeminal neuralgia; Glossopharyngeal neuralgia; Nervus intermedius neuralgia; Ibuprofen; Microvascular decompression.

## Introduction

Symptoms of cranial nerves overactivity caused by responsibility vascular compression at the root entry/exit zone (REZ) such as trigeminal neuralgia (TN), hemifacial spasm (HFS), and glossopharyngeal neuralgia (GPN) was defined as hyperactive dysfunction syndrome (HDS)(1). As we all know trigeminal neuralgia is sudden, severe facial pain. It's often described as a sharp shooting pain or like having an electric shock in the jaw, teeth, or gums. On the other hand, hemifacial spasm commonly caused by a blood vessel pushing on the facial nerve near where the nerve connects to your brain stem. Combining these cranial neuralgias occurs either synchronously or metachronously in one or both sides is called combined hyperactive dysfunction syndrome (CHDS)(2, 3). To our knowledge, TN and HFS are the most common (5~10 per 100,000), whereas GPN is relatively rare (0.7 per 100,000) presentations of the HDS(4-7). The combination of three diseases is extremely rare in clinics; several case reports and small case series have thus far been reported(2, 7-11). In addition, purely present with neuralgia in the trigeminal nerve, nervus intermedius, and glossopharyngeal nerve innervation areas have not been found in the literature. Furthermore, there is absolutely no evidence suggesting which kind of neuralgia should present as the initial symptom. These cranial neuralgias' time sequence or combination pattern is still essentially unknown (7, 12).

In the present study, we report a case of a 50-year-old man with TN, GPN, and nervus intermedius neuralgia (NIN) presenting with TN as the initial symptom, with total relief from TN, GPN, and NIN after microvascular decompression (MVD). Most notably, he has a long-term medication of ibuprofen administration after he notices the initial pain.

## Case Presentation

### The Patient's Demographics, Symptoms, Signs, and Medical History

A 50-year-old man was referred to our department with complaints of electric shock-like pain in his left cheek, the root of tooth and tongue, posterior pharyngeal wall, and deep ear and earlobe for about two months. This pain occurred without warning, and the pain is triggered especially by swallowing and speaking. When reviewing the course of the disease, it started with the left lower posterior alveolar pain, and he was treated with ibuprofen (0.6 g per day) orally by himself to alleviate pain one year ago. His complaints progressively worsened despite arbitrarily
increasing ibuprofen to 2.4 g per day. He couldn't fall asleep due to the insupportable pain, and he had his left lower posterior tooth extracted at a dentist's ten months after his initial symptom. Besides, he had no complaints of jitters of his face and eyelids and did not have any other systematical illness. He had a history of smoking and alcohol consumption.

[1] Department of Neurosurgery, Huashan Hospital, Shanghai Medical College, Fudan University.
[2] Department of Neurosurgery, Tongren Hospital, Shanghai Jiaotong University School of Medicine, Shanghai, China
Corresponding author email: qizengxin@huashan.org.cn

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***Magnetic Resonance Imaging and Laboratory Findings***

The preoperative brain magnetic resonance tomographic angiography (MRTA) sequences demonstrated vascular compression to the trigeminal and glossopharyngeal nerves. A thin branch of an artery penetrated the facial and vestibulocochlear nerve complex (**Figure 1**). Besides, there were no positive laboratory findings except for elevation of the aspartate aminotransferase and alanine aminotransferase by three times, respectively, at admission

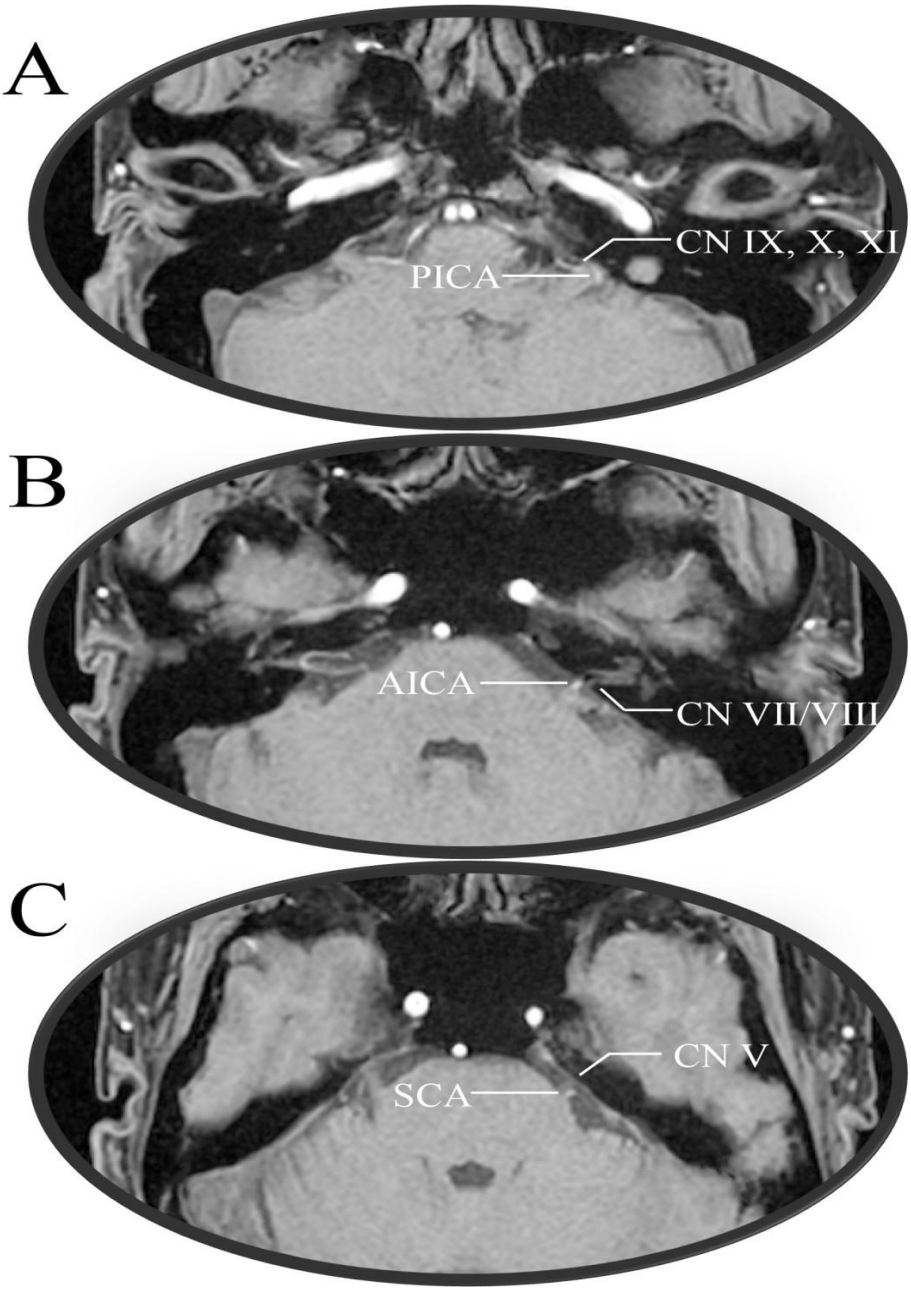


**Figure 1. Axial view of MRTA scans in the patient's left REZ.** (A) The compress the IX by the offending vessel PICA. (B) The branch of AICA penetrated the VII–VIII nerve complex. (C) The compress the V by the offending vessel SCA. (MRTA: magnetic resonance tomographic angiography, REZ: root entry/exit zone, V: trigeminal nerve, VII–VIII: facial and vestibulocochlear cranial nerve complex, IX: glossopharyngeal nerve, SCA: superior cerebellar artery, AICA: anterior inferior cerebellar artery, PICA: posterior inferior cerebellar artery).

***Surgical Findings and Procedure***

The operation was performed with microvascular decompression (MVD) via the traditional suboccipital retrosigmoid approach(13, 14). Our dissection was started from the caudal cranial nerves. The cerebellum was gradually raised after the arachnoid membrane around the nerves was sharp dissection, and then the pontomedullary sulcus was exposed. Then, the relationship between vascular and cranial nerve was carefully studied to identify the offending vessels responsible for the cranial neuralgia. The separation order of the post-inflammatory arachnoid membrane was transferred rostrally from the superior lateral trigeminal nerve to the interior posterior cranial nerve. The cranial nerves were thoroughly visualized when the arachnoid surroundings were opened sharply (**Figure 2**). After these vessels had been carefully moved away from the nerve, small pieces of the Teflon sponge were placed in neurovascular space in the reverse order. Considering the protection of facial nerve during microsurgery, direct visualization of the nervus intermedius was not regarded as necessary when all vascular structures that cause a neurovascular conflict had been separated from the VII/VIII cranial nerve complex(15). Finally, the dura mater was sutured in a

watertight pattern, and the surgical wound was routinely closed. Immediately after surgery, the patient expressed complete relief from his previous symptoms.

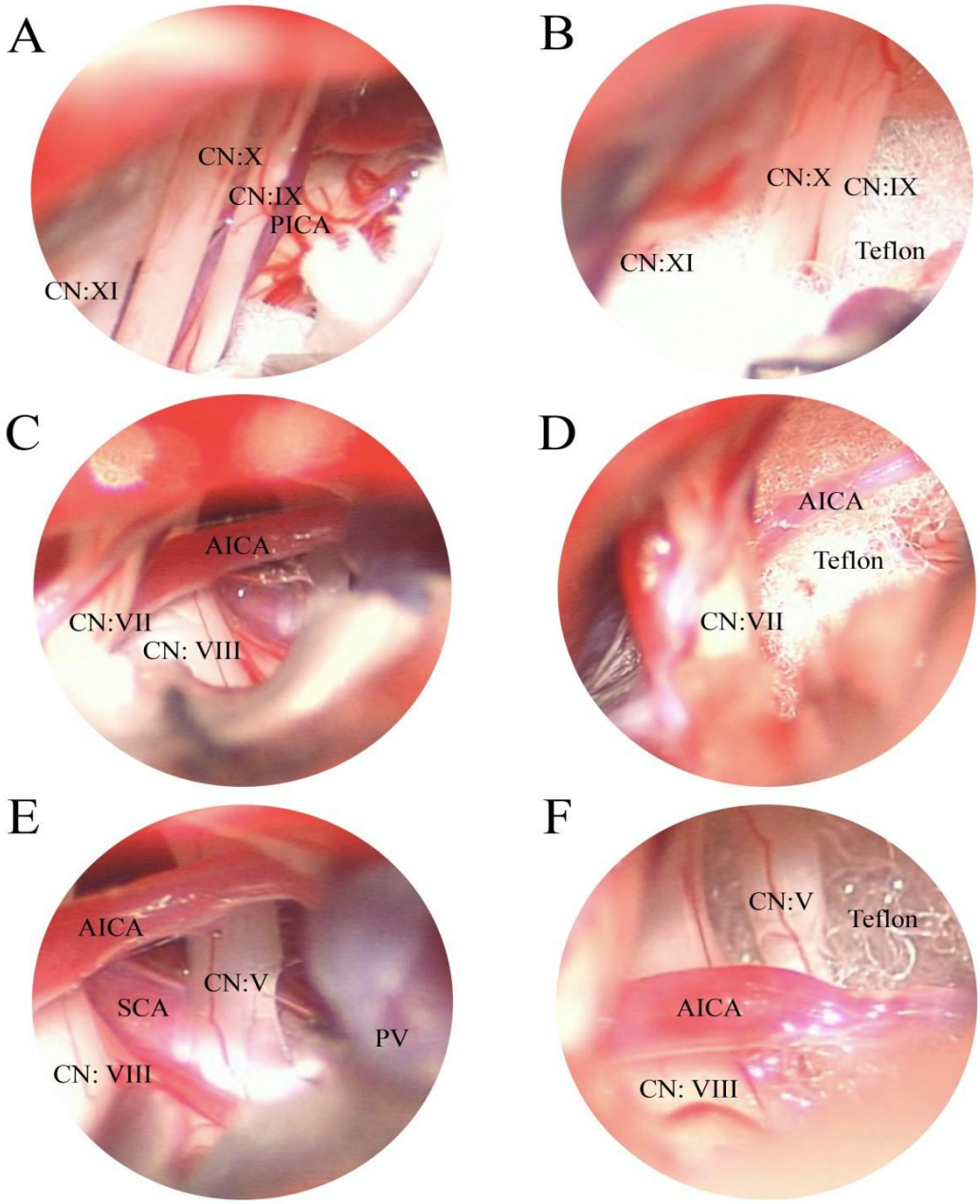


**Figure 2. Intraoperative findings: The Anatomical Structure of left crowded cerebellopontine angle (CPA) visualized under the microscope.** (A) The GIN was cause by the compression of ventromedial region of PICA. (B) Teflon was placed between IX and PICA to treat GIN. (C) A branch of the AICA penetrates to the VII–VIII cranial nerve complex and were shown to obviously compress VIII (D) A Teflon sponge was wrapped over intermediate nerves in VII–VIII complex. (E) SCA was the cause of the vascular compression anterosuperior to the TN. (F) A small piece of Teflon was placed between SCA and V. (TN: trigeminal neuralgia, NIN: nervus intermedius neuralgia, GIN: glossopharyngeal neuralgia, V: trigeminal nerve, VII–VIII: facial and vestibulocochlear cranial nerve complex, IX: glossopharyngeal nerve, SCA: superior cerebellar artery, AICA: anterior inferior cerebellar artery, PICA: posterior inferior cerebellar artery).

**Discussion**

With a prevalence rate of 5~10 per 100,000, HDS was first described in 1998 by Kobata (3). To our knowledge, TN and HFS are the most common vascular compression syndromes of HDS(4). Although there have been a considerable number of reports describing the different combinations of TN, HFS, and GPN of such disease, the combination of all symptoms named CHDS is still an extremely rare phenomenon in previous literature (**Table 1**) (1, 7). The prevalence of CHDS was reported to be less than 3% of all patients with HDS(12). Even rarer is coexistent TN, GPN, and NIN that originated from cranial nerve neuropathies. A tendency of clinical diagnosis was such distributional pains were concomitant symptoms affected by one severe neuralgia, while the coexistence of all neuralgia was always being neglected.

**Table 1. Characteristics of CHDS cases reported in previous literatures.** CHDS: combined hyperactive dysfunction syndrome; TN: trigeminal neuralgia; HFS: hemifacial spasm; GPN: glossopharyngeal neuralgia; V: trigeminal nerve; VII: facial nerve; VIII: vestibulocochlear nerve; IX: glossopharyngeal nerve; X: vagus nerve; XI: accessory nerve; AICA: anterior inferior cerebellar artery; PICA: posterior inferior cerebellar artery; SCA: superior cerebellar artery; VA: vertebral artery; PV: petrosal vein; MCP: middle cerebellar peduncle vein.

| Case reports | Gender/age | Affected symptoms | Time span from initial symptom to CHDS | Offending vessels | Treatment |
|---|---|---|---|---|---|
| Wang Et al[7]. | M/61 | r-TN-GPN-HFS | 10 years | PICA (IX), PV (V), VA & AICA (VII) | MVD |
| | M/56 | l-TN-GPN-HFS | 8 years | SCA (V), PICA (VII & IX) | MVD |
| | F/45 | l-TN-GPN & r-HFS | 2 years | AICA (VII), SCA (V) PICA (IX) | MVD |
| | F/53 | l-GPN-HFS & r-TN | 4 years | VA (VII), SCA (V), PICA (IX) | MVD |
| | F/69 | l-TN-HFS & r-GPN | 15 months | PICA (IX & VII), AICA (V) | MVD |
| | F/77 | l-TN-GPN-HFS | 9 months | PICA (IX), SCA (V), VA & AICA (VII) | MVD |
| Cao et al[2]. | F/60 | TN-HFS-GPN | - | AICA & SCA (V), PICA (VII & IX) | MVD |
| | F/65 | TN-HFS-GPN | - | SCA (V), AICA & PICA (VII), AICA (IX) | MVD |
| Perez-Roman et al[16]. | M/66 | r-TN-HFS-GPN | 3 years | VA (V, VII & IX) | MVD |
| Kuzucu et al[17]. | M/73 | r-GPN-HFS-TN | 1 year | SCA (V), MCP (VII & VIII), AICA (IX, X & XI) | MVD |

Our case is exciting and different from previous cases with several features. Firstly, NIN is a rare disease, and a total of 150 cases have been reported so far, let alone synchronously combined with TN and GIN(16). The fulgurant pains of NIN in the external auditory canal are not easy to identify because the distribution of the nerves is overlapping in the ear(17). Meanwhile, the ear canal is also innervated the tympanic branch of the glossopharyngeal nerve and the auriculotemporal branch of the mandibular division of the trigeminal nerve(18). It leads to missed diagnosis easily due to the radiating pain of TN, and GIN resembles NIN. Therefore, NIN should be taken into consideration when the patient complains about neuralgia symptoms in his external auditory canal. Secondly, not all cranial neuralgia has positive imaging findings. Early diagnosis mainly depends on complex clinical signs, and the aid of diagnostic neuroimaging seems helpful. It has been reported that there are about 60% of cases have been diagnosed with NIN, and up to 20% of cases' nervus intermedius can't be seen in neuroimaging(18, 19). Although we failed to mark the nervus intermedius in pre-operative diagnostic MRTA sequences, we have clarified the orientation of offending vessels and facial-vestibulocochlear nerve complex, thus conducive to locating the sectional anatomy at the surgery. Finally, our case has the shortest period of disease progression than previous cases, from initial symptoms to combined symptoms. So that triggers us to rethink and explore the complicated osteogenesis of such phenomenon profoundly.

We reviewed our patient's medical history carefully, and it is noteworthy that he has a history of nonsteroidal anti-inflammatory drugs (NSAIDs) abuse. The dual cyclooxygenase enzyme (COX) inhibitor, ibuprofen, has become NSAIDs' most widely used anti-inflammatory analgesics. Although ibuprofen is not amongst the first-line drugs for neuralgia, it also exerts some COX-independent effects, such as disturbing neutrophil attraction and activation and G protein activity to inhibit RhoA signaling, reduce Fos expression in the cranial nucleus and modulate the astroglia, microglia and neurons activity centrally(21-23). Ibuprofen can be used as a neuropathic pain ameliorator for all the above-mentioned reasons because it promotes axonal regeneration and increases functional recovery after neural injuries(24-26). However, chronic overuse of ibuprofen may induce a liver function injury because it is leading causative drug to idiosyncratic drug-induced liver injury (DILI) across the world(27, 28). This point was confirmed in the abnormal indicators of liver function in our case. On the other hand, a recent study suggests that no histological

improvement was detected and even worsened cognitive outcomes after chronic ibuprofen administration(22, 29, 30). In addition, it was found that inhibition of the COX pathway may have a negative feedback effect by disturbing the expression of inflammatory cytokines with which increase TNF-α and IL-1β in micro-vessels(31). And the ample evidence that up-regulation of inflammatory factors will contribute to neuralgia was confirmed in the experimental evaluation and the clinical correlative analysis(32-35).

The neuropathic pains resulted from the inflammatory mediators released from immune cells and damaged neurons sensitizing the nociceptors. Specifically, it is the process of neuroinflammation, namely Wallerian degeneration, in which damaged fibers are degraded, replaced and remyelinated(36-38). This is a well-designed and protective physiological process in vivo, and microglia/macrophages, T-lymphocytes, cytokines and chemokines are the key players in such progress(35, 39). If such a regulatory mechanism is disturbed, it may aggravate the improvement of cranial neuralgia. In the past, the effects of ibuprofen on the phagocytic properties and modulation of cytokines related to the inflammatory response of microglia have been extensively studied(40, 41). Moreover, the experimental results show that ibuprofen can reduce the activation and inhibit the reactivity of microglia/macrophages, limiting their capacity for removing cell death and myelin debris(22). These findings motivate our investigation of the inflammatory cytokines disorder of the peripheral microenvironment induced by ibuprofen. Therefore, we innovatively hypothesize that the "disturbance of perineural microenvironment" is a significant cause of coexistent TN, GIN and NIN in patients (**Figure 3**).

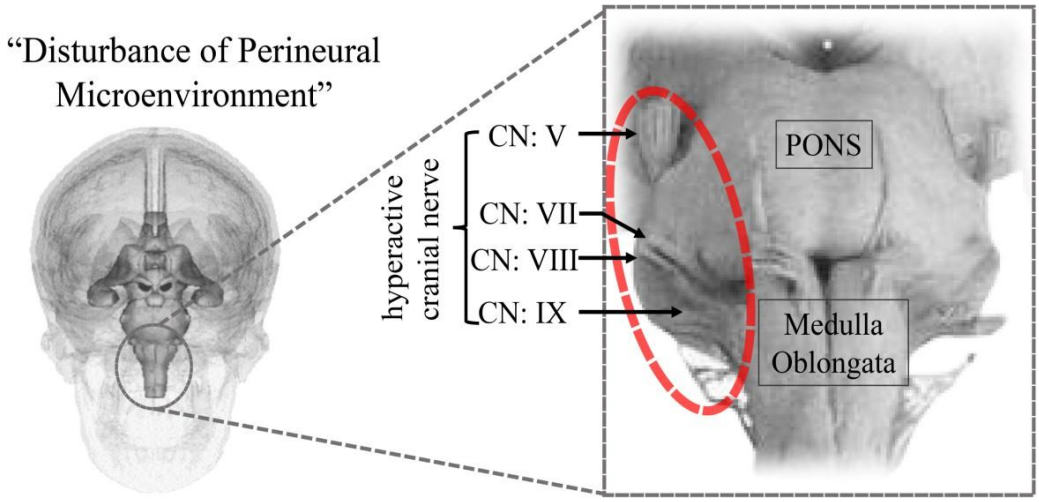


**Figure 3. "Disturbance of Perineural Microenvironment" Hypothesis.** The resolution of neuroinflammation had been disturbed (red-dotted-circles) and generate dysregulated microglia/macrophages and cytokines ln the microenvironment of cranial nerves. (V: trigeminal nerve, VII facial nerve, VIII: vestibulocochlear nerve, IX: glossopharyngeal nerve).

To our knowledge, MVD is the conventional treatment in CHDS(3, 42). Although our case did not show any symptoms of HFS, the outcomes of standard MVD for combined cranial neuralgias are satisfactory. Innovative operative techniques and newfound intracranial implants have been described to date(43-45). No matter which kind of procedure was performed, there are three essentials to share based on our experience: At first, separating the offending vessels and nerves, maximumly opening the arachnoid adhesions and thoroughly releasing the offending vessels. Secondly, dissection starts with the caudal cranial nerves and can achieve a safer operation space. Finally, it is not recommended to blindly separate the nervus intermedius to realize visualization when all conflict vascular had been separated from the VII/VIII cranial nerve complex.

## Conclusion

This is the first case reported of coexistent TN, GIN and NIN after chronic ibuprofen administration and treated successfully with MVD. Chronic ibuprofen administration could alleviate neuropathic pain but not exert a neuroprotection effect. Improper clinical use of the NSAIDs may have additional adverse effects and disturb the peripheral microenvironment of cranial nerves. Detailed clinical inquiry and radiological findings should be taken before the operation. MVD via suboccipital retrosigmoid approach is still a favorable treatment choice for the treatment of such coexistent cranial neuralgia.

## Disclosure Statement

No potential conflict of interest was reported by the authors.

## Data Availability Statement

All datasets presented in this study are included in the article/supplementary material.

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