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A Case of Trigeminal Neuralgia with Hemifacial Spasm – Double Mvd – A Surgical Cure

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KEYWORDS

trigeminal neuralgia, hemifacial spasm, painful tic convulsif, neurovascular compression, microvascular decompression, cranial nerve hyperactive dysfunction syndrome, cerebellopontine angle.

ABSTRACT:

Trigeminal neuralgia (TN) and hemifacial spasm (HFS) are cranial nerve hyperactive dysfunction syndromes most commonly caused by neurovascular compression. The simultaneous occurrence of TN with ipsilateral HFS—historically termed *painful tic convulsif*—is rare and often associated with dual neurovascular conflict. We report the case of a 62-year-old woman with a 15-year history of refractory right-sided TN accompanied by progressive ipsilateral HFS. MRI revealed vascular compression of both the trigeminal nerve and the facial–vestibulocochlear nerve complex. The patient underwent right retromastoid suboccipital craniectomy with double microvascular decompression (MVD). Intraoperatively, the anterior inferior cerebellar artery (AICA) and superior cerebellar artery (SCA) were identified as offending vessels. Successful decompression produced immediate and complete resolution of both facial pain and spasms, with no postoperative deficits. This case highlights the importance of recognizing combined neurovascular compression syndromes and supports early surgical intervention as a definitive, highly effective treatment. Double MVD offers durable symptom relief and can significantly improve patient quality of life.

Introduction

Hemifacial spasm (HFS) and trigeminal neuralgia (TN) are neurological conditions characterized by abnormal overactivity of specific cranial nerves [1, 2]. Cases resulting from blood vessel compression of the facial or trigeminal nerve roots are clinically classified as idiopathic HFS or TN. [3, 4]. However, these conditions can also develop secondary to nerve injury caused by tumors, demyelinating diseases, trauma, infections, and other factors; in such cases, they are referred to as symptomatic HFS or TN [5, 6, 7, 8, 9]. In 1962, Gardner and Sava were the first to report that pathological changes in the cerebellopontine angle (CPA) could give rise to HFS or TN. [10, 11] Subsequently, numerous reports have shown that a wide range of lesions can lead to symptomatic HFS or TN, including posterior circulation aneurysms and vascular malformations, cerebellopontine angle cysts, tumors of the brainstem and fourth ventricle, as well as more distant abnormalities such as remote meningiomas or even contralateral lesions. [12, 13, 14, 15, 16, 17] Even

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so, published studies indicate that the incidence of symptomatic HFS and TN remains low, estimated at about 0.3–2.5% and 1–9.9%, respectively.[18, 13, 19, 15, 20]. This article presents a case of a 62-year-old woman with long-standing TN associated with ipsilateral HFS, successfully treated with double microvascular decompression. The clinical features, radiological findings, surgical management, and postoperative outcomes are discussed, highlighting the importance of early recognition and appropriate surgical intervention in restoring function and improving patient well-being.

Case Presentation.

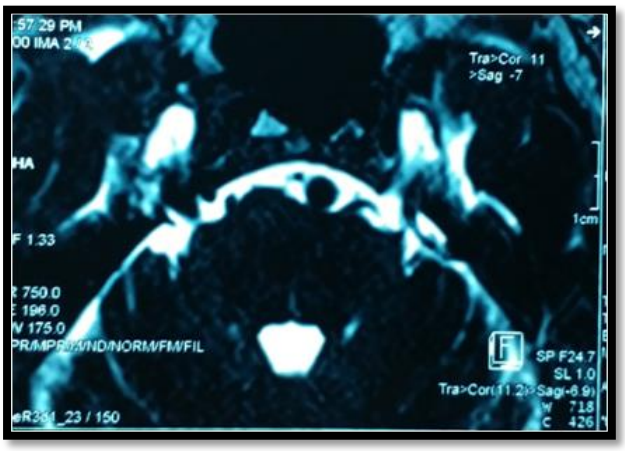
A 62-year-old woman presented with a 15-year history of paroxysmal right-sided facial pain. The pain was described as intermittent, lancinating, and electric shock–like in character, localized predominantly to the cheek and mandibular regions corresponding to the maxillary (V2) and mandibular (V3) divisions of the trigeminal nerve. These episodes were consistently triggered by routine daily activities including chewing, brushing teeth, washing the face, and smiling. Over time, the severity and frequency of symptoms increased to the extent that the patient consciously avoided smiling to prevent precipitation of excruciating pain.

For more than a decade, her symptoms had been partially controlled with pharmacotherapy; however, during the two years preceding presentation, she experienced progressive loss of therapeutic response despite escalating medical management.

Neurological examination revealed no focal deficits. However, involuntary, intermittent contractions of the right lower facial musculature were observed, consistent with hemifacial spasm (HFS). This coexistence of trigeminal neuralgia (TN) with ipsilateral hemifacial spasm suggested a combined cranial nerve hyperactive dysfunction syndrome, an entity historically termed painful tic convulsif.

Radiological Findings

Magnetic resonance imaging (MRI) of the brain demonstrated clear evidence of neurovascular conflict involving both the trigeminal and facial nerve complexes on the right side. A vascular loop was observed abutting the cisternal segment of the right trigeminal nerve (CN V). An additional vascular loop was seen compressing the VII/VIII cranial nerve complex. These findings supported the clinical impression of dual neurovascular compression contributing to the patient's symptoms.

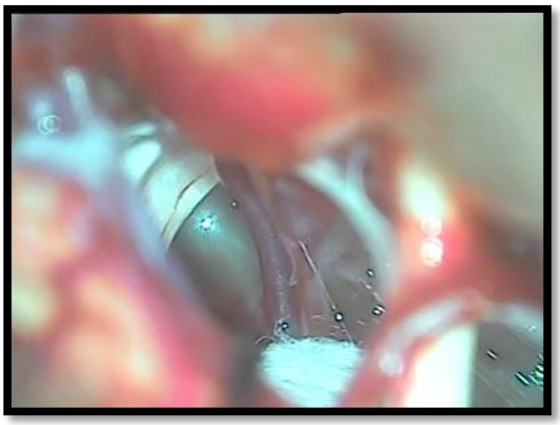


Surgical Intervention

Given the severity, chronicity, and refractoriness of symptoms to medical therapy, the patient underwent a right retromastoid suboccipital craniectomy with microvascular decompression (MVD) of both the trigeminal nerve and the facial–vestibulocochlear nerve complex.

Intraoperatively, the anterior inferior cerebellar artery (AICA) was identified as a major offending vessel, exerting compression upon both the VII/VIII nerve complex and the trigeminal nerve. The superior cerebellar artery (SCA) was also noted to be in conflict with the trigeminal nerve. Following meticulous arachnoid dissection, the conflicting vascular loops were carefully mobilized and separated from their respective cranial nerves. Teflon felt was interposed between the vessels and the nerve root entry zones to maintain durable decompression.

The procedure was completed without complications.



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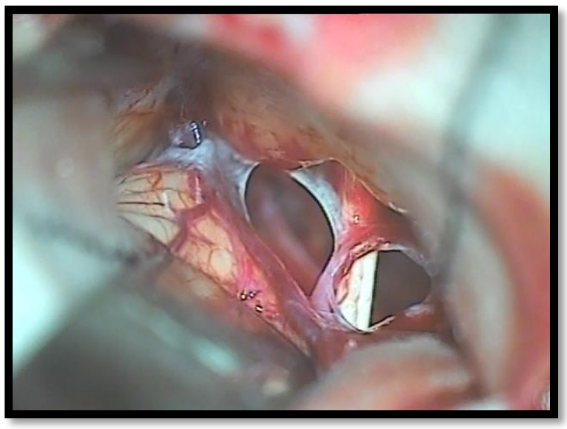

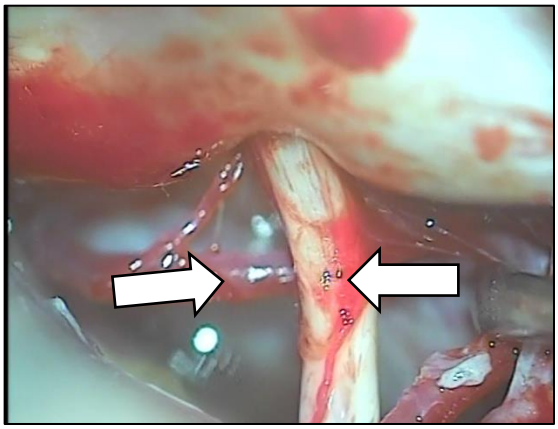

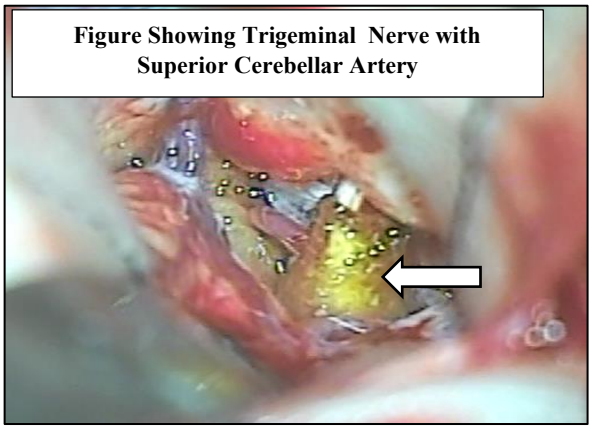

Figure Showing Trigeminal Nerve with Superior Cerebellar Artery

Figure – Teflon between the vessels and the nerve root entry zones to maintain durable decompression.

Postoperative Course

Postoperatively, the patient experienced immediate and complete resolution of her trigeminal pain as well as her hemifacial spasm. No new neurological deficits were observed. All preoperative medications for trigeminal neuralgia were discontinued. At follow-up, the patient remained symptom-free, reporting restoration of normal facial function and improved quality of life—remarking that she was finally able to smile without fear of pain.

Discussion

Mass lesions are an uncommon underlying cause of either HFS or TN. [14, 19, 16]. Shulev and colleagues suggested that mass compression may contribute to the development of trigeminal neuralgia , as the offending vessel was identified in only 5 of their 14 patients with symptomatic Trigeminal Neuralgia . [8]. Ogleznev and colleagues reported four cases of symptomatic HFS caused by epidermoid tumors that completely encased the nerve root, with no offending artery present. [21]. Based on these findings, it has been proposed that chemical irritants within the epidermoid tumor may exert toxic effects on the nerve root, leading to disruption of the myelin sheath. [15, 9]. However, these explanations remain speculative, as no direct evidence has been established. The symptoms also tended to appear during moments of excitement or anxiety. [22,23,24]. With advancing age, varying degrees of brain atrophy and angiosclerosis can develop, potentially bringing blood vessels into closer proximity to the affected nerve. [25,26].

With such close contact, both the epineurium of the nerve and the adventitia of the vessel may gradually wear down due to constant pulsatile friction. Neurotransmitters released from sympathetic nerve endings within the arterial wall can then leak through the damaged adventitia and affect the adjacent, injured nerve fibers. Over time, these demyelinated fibers may develop ectopic excitability, leading to episodes of involuntary facial muscle contractions or painful sensory attacks on the affected side of the face. [27].

Trigeminal neuralgia is managed through a spectrum of medical and interventional modalities tailored to symptom severity, patient comorbidities, and response to therapy. First-line pharmacologic treatment typically includes anticonvulsant agents such as carbamazepine, pregabalin, and gabapentin, which help modulate neuropathic pain by stabilizing neuronal hyperexcitability. For patients with inadequate relief or intolerable side effects, interventional options offer targeted disruption of trigeminal pain pathways. Percutaneous procedures—including glycerol rhizotomy, radiofrequency ablation, and peripheral neurectomy—provide varying degrees of selective nerve injury to reduce paroxysmal pain while preserving function. Noninvasive approaches such as stereotactic radiosurgery deliver focused radiation to the

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trigeminal root entry zone, offering a durable alternative with favorable safety profiles. Additionally, local botulinum toxin injections, particularly useful in cases coexisting with hemifacial spasm, have emerged as an adjunctive therapy with promising outcomes. Collectively, these strategies form a comprehensive, individualized approach to achieving long-term pain control in trigeminal neuralgia.[28,29,30,31]

Conclusion

MVD should be strongly considered for symptomatic Trigeminal Neuralgia/ Hemifacial spasm with radiologically evident neurovascular conflicts for symptomatic cure, to bring back the lost smile

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