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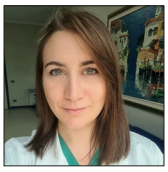
*Case Report*

# Glossopharyngeal neuralgia and hypoglossal nerve palsy: A singular clinical case of two rare concomitant neurovascular conflicts

Luigi Valentino Berra, Francesca Di Cristanziano, Antonio Santoro

Department of Human Neurosciences, Neurosurgery, Sapienza University, Rome, Italy.

E-mail: Luigi Valentino Berra - luigivalentinoberra@gmail.com; *Francesca Di Cristanziano - francescadicri@gmail.com; Antonio Santoro - an.santoro@policlinicoumberto1.it



***Corresponding author:**
Francesca Di Cristanziano, Department of Human Neurosciences and Neurosurgery, Sapienza University, Rome, Italy.

francescadicri@gmail.com

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## ABSTRACT

**Background:** Neurovascular conflict (NVC) is described as a pathological contact between cranial nerves and vessels. Glossopharyngeal neuralgia (GPN) and hypoglossal nerve palsy (HNP) due to NVC represent rare clinical entities. To our knowledge, we present the first reported case of concomitant GPN and HNP caused by vertebral artery (VA)-posterior inferior cerebellar artery (PICA) complex compression.

**Case Description:** We report an extremely rare case of a 52-year-old man with combined unilateral left-sided GPN and HNP because of NVC involving both the VA and the PICA, successfully treated with a retrosigmoid approach for microvascular decompression (MVD). Postoperatively, the patient immediately recovered without new-onset dysfunction of lower cranial nerves, and a complete remission of symptoms was achieved.

**Conclusion:** As far as we know, in this article, we present the first singular case in the literature of concomitant classical GPN and HNP due to NVC involving both the VA and the PICA. Despite the low incidence of GPN and HNP, clinical picture and intraoperative findings represent clear and reliable elements for their diagnosis. MVD is a successful therapeutic strategy that offers a long-term cure for GPN and HNP.

**Keywords:** Glossopharyngeal neuralgia, Hypoglossal nerve palsy, Microvascular decompression, Neurovascular conflict, Posterior inferior cerebellar artery, Vertebral artery

## INTRODUCTION

Neurovascular conflict (NVC) is described as a pathological contact between cranial nerves and vessels. Trigeminal neuralgia (TN) and hemifacial spasm (HFS) are typical conditions associated with NVC. Conversely, glossopharyngeal neuralgia (GPN) and hypoglossal nerve palsy (HNP) secondary to NVC represent rare clinical entities. GPN has an incidence of 0.5/100.000, while HNP is even more uncommon.[6,9] We present the first reported case of combined GPN and HNP due to vertebral artery (VA)-posterior inferior cerebellar artery (PICA) complex compression, successfully treated with a retrosigmoid approach for microvascular decompression (MVD).

## CASE DESCRIPTION

We present a case of concomitant GPN and HNP secondary to VA-PICA complex compression. A 52-year-old man presented to our hospital with a history of 40/day episodes of paroxysmal, stabbing-like pain along the left oropharynx and the left ear and posteriorly to the left mandibular angle that lasted from a couple of seconds to minutes. The patient suffering from the same pain had previously been treated for otitis with antibiotics without benefit. Neurological examination revealed a hypotrophic left side of the tongue, hypoelicitable left gag reflex, and left palatal weakness. Preoperative magnetic resonance imaging (MRI) revealed an NVC between the left glossopharyngeal nerve (GN) and the ipsilateral VA [Figures 1 and 2], and preoperative cerebral computed tomography angiography showed the ectatic left VA and the left tortuous PICA forming a loop [Figure 3]. The patient underwent MVD surgery through a retrosigmoid approach with the aid of intraoperative neurophysiological monitoring (IONM).

### Surgical procedure

The patient was positioned in a park-bench position with the left side up with a 3-pin Mayfield head holder. A standard left retrosigmoid approach was performed. After draining cerebrospinal fluid from the cerebello-medullary cistern, an arachnoidal dissection was carried out from the 7$^{th}$ to 8$^{th}$ nerves rostrally down to the mixed nerves caudally [Figure 4]. Intraoperatively, the curved course of the left ectatic VA behind the GN appeared responsible for NVC, and the hypoglossal nerve (HN) was compressed by the VA and the PICA [Figures 5 and 6]. In particular, the HN appeared tenaciously trapped in the axilla formed at the origin of the anterior medullary segment of the PICA from the VA [Figure 7]. The arteries were carefully moved and separated from the nerves using polytetrafluoroethylene sheets. At the end of the surgical procedure, no variation from the baseline IONM signal was registered.

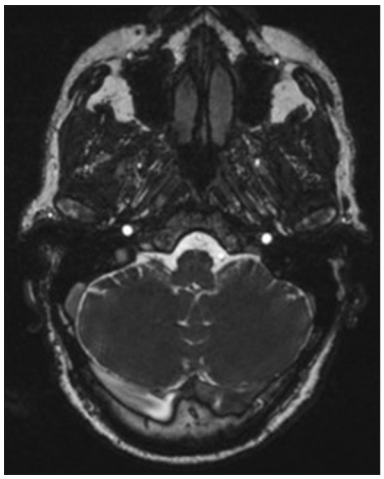

**Figure 1:** Preoperative axial true fast imaging with steady-state-free precession magnetic resonance imaging: Neurovascular conflict between left vertebral artery and glossopharyngeal nerve.

### Postoperative course

Postoperatively, the patient immediately recovered from pain without new-onset dysfunction of lower cranial nerves, and a complete remission of symptoms was achieved.

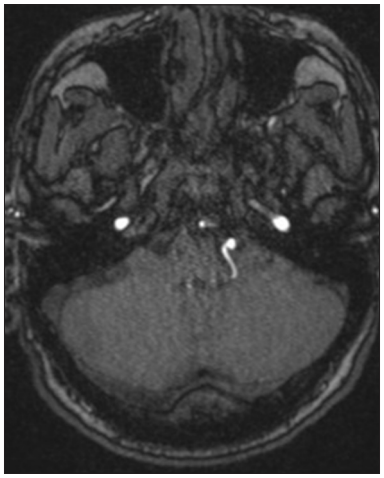

**Figure 2:** Preoperative axial time of flight magnetic resonance imaging: Origin of left tortuous posterior inferior cerebellar artery from vertebral artery.

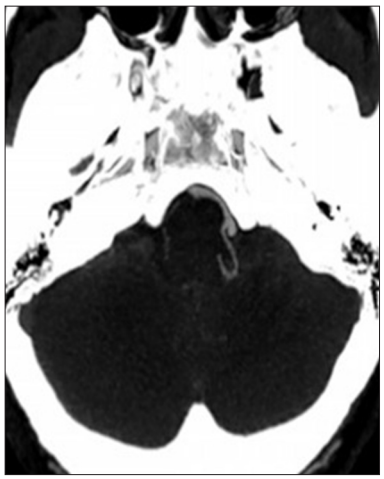

**Figure 3:** Preoperative computed tomography angiography: Ectatic vertebral artery and left tortuous posterior inferior cerebellar artery forming a loop.

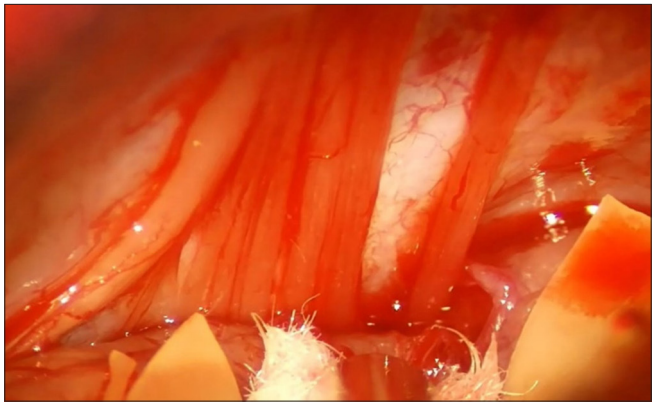

**Figure 4:** Intraoperative image of mixed cranial nerves in the left cerebellopontine angle.

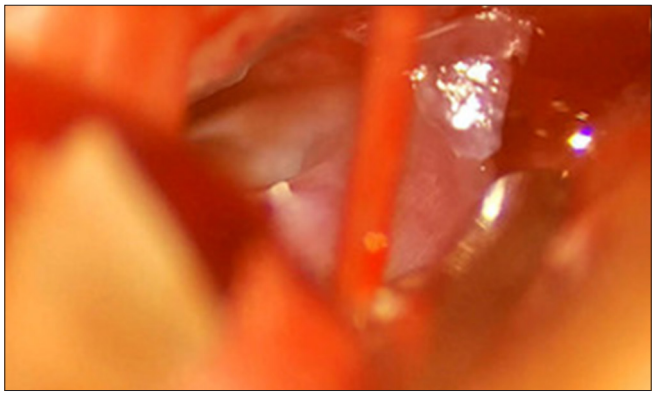

**Figure 5:** Intraoperative image of the curved course of the left ectatic vertebral artery behind the glossopharyngeal nerve responsible for neurovascular conflict.

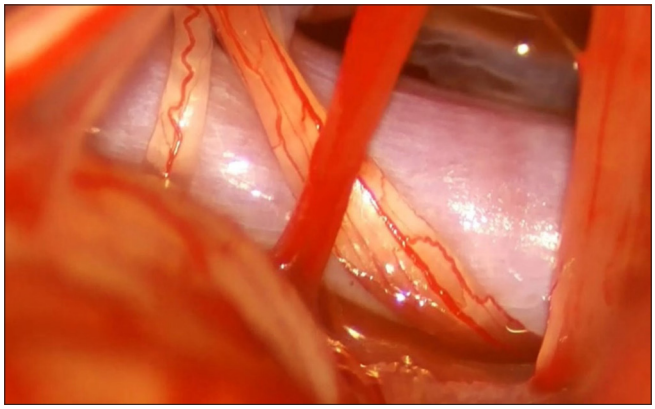

**Figure 6:** Intraoperative image of the evident neurovascular conflict between the left vertebral artery and hypoglossal nerve.

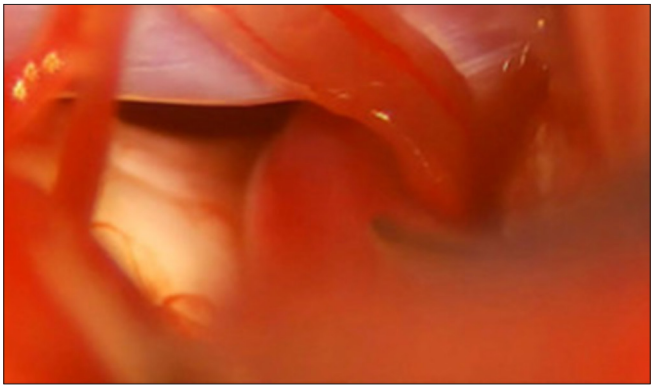

**Figure 7:** Intraoperative image of left hypoglossal nerve tenaciously trapped in the axilla formed at the origin of the anterior medullary segment of the posterior inferior cerebellar artery from the vertebral artery.

## DISCUSSION

GPN and HPN are classified into three categories: Classical, secondary, and idiopathic.[7,14] Classical GPN and HPN are provoked by a compression of the nerves by one or more vessels on the root entry zone, which represents the transitional region where the shift of peripheral Schwann cell myelination to central oligodendroglia myelination takes place. This condition causes nervous fibers' demyelination and subsequent hyperexcitability, generating ectopic impulses responsible for ephaptic transmission.[1,9,14,15,19] Several causes of secondary GPN and HPN are reported in the literature, including carotid artery dissection, internal carotid artery aneurysm, amyloidosis with cranial nerves neuropathy, intracranial space-occupying lesions, trauma, multiple sclerosis, infections such as mononucleosis, Collet–Sicard Syndrome, Guillain–Barré Syndrome, and surgery.[2,3,10-12,18] GPN and HNP are defined as idiopathic when no cause can be identified.[14,17] GPN consists of short and sudden-onset episodes of paroxysmal pain at the base of the tongue or tonsils, radiating back towards the ear. Trigger factors include chewing, swallowing, coughing, and speaking. An impairment of the GN induces the abolition of the velopalatine reflexes anesthesia of the upper part of the pharynx, the tonsils, and the base of the tongue. HNP typically occurs with lingual hemiatrophy and a seemingly crenate tongue. The diagnosis of GPN and HNP is both clinical and radiological through MRI using a heavily T2-weighted sequence, such as fast imaging employing steady-state acquisition or constructive interference steady state.[4,14,16] The offending arteries involved in GPN are the PICA most frequently and the anterior-inferior cerebellar artery less frequently, while HNP is usually caused by VA compression.[8,13]

Simultaneous impairment of V, VII, and IX cranial nerves due to NVC is defined as hyperactive dysfuntion syndrome (HDS), characterized by concomitant TN, HFS, and GPN. Perez-Roman *et al*. described an interesting HDS caused by the dolichoectatic vertebrobasilar system, successfully treated with the clip-sling technique through a retrosigmoid approach. Despite several cases of HDS reported in the literature, the association of classical TN and/or HFS and/or GPN with HNP has never been described.[15,20] Dokdok

*et al*. presented a peculiar isolated unilateral HNP caused by an internal carotid artery loop and concomitant wall irregularities of the loop segment detected in magnetic resonance angiography and digital subtraction angiography; the 42-years-old patient was treated with antithrombotic therapy without the need of any further intervention.[5] As far as we know, in this article, we present the first singular case in the literature of concomitant classical GPN and HNP due to NVC involving both the VA and the PICA. The simultaneous impairment of both GN and HN is described only in secondary GPN and HNP.

## CONCLUSION

To our knowledge, classical GPN and HNP due to NVC represent rare pathological conditions and their simultaneous presence has never been described before in literature. Despite their low incidence, clinical picture and intraoperative findings represent clear and reliable elements for their diagnosis. MVD is a successful therapeutic strategy that offers a long-term cure for GPN and HNP.

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