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Vascular decompression in the treatment of vagoglossopharyngeal neuralgia

© G.Yu. Grigoryan[1], D.Yu. Usachev[2, 3], Yu.A. Grigoryan[1]

[1]Treatment and Rehabilitation Centre, Moscow, Russia;
[2]Burdenko Neurosurgery Center, Moscow, Russia;
[3]Russian Medical Academy of Continuous Professional Education, Moscow, Russia.

ABSTRACT

Background. Vagoglossopharyngeal neuralgia (VGFN) is an uncommon facial pain syndrome caused by neurovascular conflict.
Objective. To evaluate anatomical relationships between cranial nerves IX—X and compressing arteries, as well as effectiveness of vascular decompression (VD) in these patients.
Material and methods. VD via retrosigmoid approach was performed in 9 patients with VGFN. Age of patients was 33—72 years. There were 4 males and 5 females. Two ones had right-sided lesions, 7 — left-sided lesions. Mean follow-up period was 8.5 years (range 3—15).
Results. Neurovascular conflict was caused by posterior inferior cerebellar artery (PICA) in 6 cases, anterior inferior cerebellar artery (AICA) and PICA in 2 cases, PICA and vertebral artery in 1 case. Interposition technique was used in 7 cases, transposition of vessels — in 2 cases. Complete relief of VGFN was achieved in all patients. Mild dysphonia without dysphagia was registered in 1 case and regressed throughout 1.5 months. There were no VGFN recurrences.
Conclusion. VD is a highly effective surgical method for VGFN with low rate of complications and pain recurrences.

Keywords: *vagoglossopharyngeal neuralgia, glossopharyngeal neuralgia, vascular decompression, interposition, transposition.*

INFORMATION ABOUT THE AUTHORS:
Grigoryan G.Yu. — https://orcid.org/0000-0001-5745-8061
Usachev D.Yu. — https://orcid.org/0000-0002-9811-9442
Grigoryan Yu.A. — https://orcid.org/0000-0003-3157-6187
Corresponding author: Grigoryan G.Yu. — e-mail: dr.grigoryangeorgy@yandex.ru

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Abbreviations

VD — vascular decompression
VGPN — vagoglossopharyngeal neuralgia
PICA — posterior inferior cerebellar artery
VA — vertebral artery
AICA — anterior inferior cerebellar artery
ICHD-3 — The International Classification of Headache Disorders

Introduction

VGPN is a rare neurological syndrome characterized by attacks of shooting pain in the root of the tongue, soft palate, tonsil, pharynx and deep in the ear. Short-term paroxysms of pain, lasting from a few seconds to 2—3 minutes, occur during talking, swallowing and chewing. The prevalence of VGPN is 0.2—0.7 cases per 100.000 people [1]. Left-sided localization of pain syndrome is more common by 1.5 times. At baseline, course of disease is characterized by prolonged remissions, but over the years the incidence and intensity of neuralgic attacks increase and sometimes lead to alimentary dystrophy. Pain attacks may be accompanied by bradycardia, arterial hypotension and asystole. In rare cases, these manifestations predominate over pain syndrome [2]. VGPN can accompany various extracranial tumors and inflammatory lesions of the oropharynx and elongation of styloid process, but the main intracranial etiologic factor is compression of cranial nerves IX—X by adjacent vessels, most commonly PICA and AICA, as well as less commonly VA [1, 2].

Ineffective anticonvulsants in VGPN are an indication for surgical treatment. Microsurgical transection and percutaneous radiofrequency thermocoagulation of glossopharyngeal and vagus nerves lead to phonation and swallowing impairment. Moreover, they are characterized by low efficacy and frequent recurrences of neuralgic pain [1—3]. Currently, the main surgical method for VGPN is VD of glossopharyngeal and vagus nerves eliminating neurovascular conflict while preserving ana-

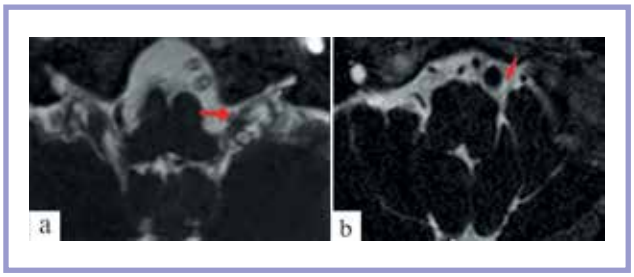


***Fig. 1.* Vascular compression of cranial nerves IX–X, magnetic resonance imaging.**
a — compression by posterior inferior cerebellar artery (red arrow); b — compression by vertebral artery and posterior inferior cerebellar artery (red arrow).

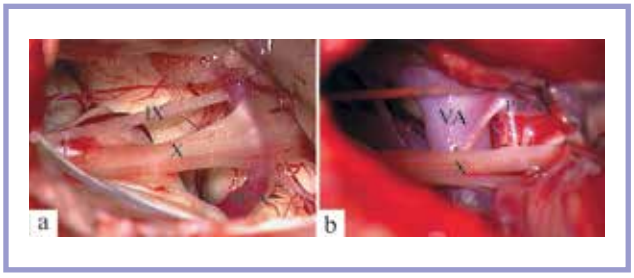


***Fig. 2.* Vascular compression of cranial nerves IX–X, intraoperative images.**
a — compression by posterior inferior cerebellar artery; b — compression by vertebral artery and posterior inferior cerebellar artery.

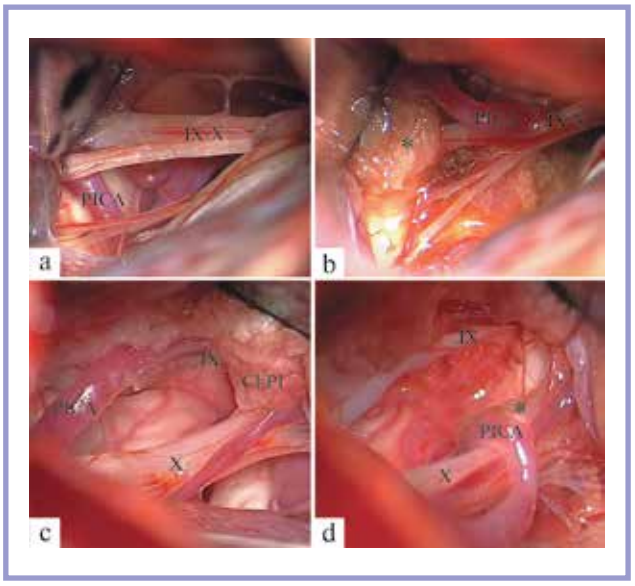


***Fig. 3.* Vascular decompression of cranial nerves IX–X, intraoperative images.**
a — compression by posterior inferior cerebellar artery; b — muscle implants between the artery and the nerve roots (interposition); c — compression by posterior inferior cerebellar artery; d — artery is dislocated and fixed with implants (transposition). ChPl — choroid plexus; * — implant.

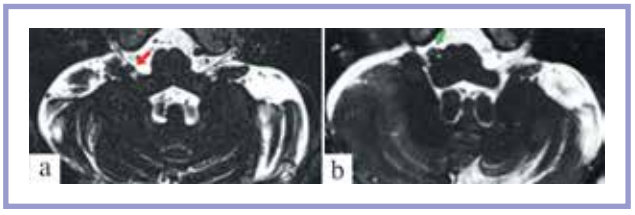


***Fig. 4.* Vascular decompression of cranial nerves IX-X, magnetic resonance imaging.**
a — compression by posterior inferior cerebellar artery (red arrow); b — after vascular decompression (implant — green arrow).

tomical integrity and function of cranial nerves. Low incidence of postoperative cranial nerve dysfunction and recurrence emphasizes the advantages of VD that usually leads to permanent regression of VGPN [1, 4—13].

The purposes of this study were to present an assessment of vascular relationships with cranial nerves IX—X and evaluate the effectiveness of VD for VGPN.

Material and methods

Surgical treatment was performed in 9 patients with VGPN (age 33—72 years, 5 women and 4 men, right-sided lesions in 2 cases, left-sided lesions in 7 cases). Mean duration of disease was 5.5 years (range 2—15). In all cases, clinical presentation of VGPN met ICHD-3 criteria. Intense shooting pain paroxysms and trigger zones were typically localized, and treatment with anticonvulsants failed to relieve neuralgic pain or was accompanied by toxic effects. There was 1 case of bradycardia with moderate arterial hypotension during neuralgic attacks.

All patients underwent neurological examination and MRI, including contrast-enhanced MRI, DWI, TOF and FIESTA sequences, before and after surgery. Retrosigmoid craniotomy up to 2.5 cm at the medial margin of the sigmoid sinus and arachnoid dissection towards pontomedullary fissure and choroidal plexus of the lateral recess of the fourth ventricle provided visualization of cranial nerves IX—X and vessels near the brainstem. Compressing arteries were retracted from nerve fibers and brainstem, and their new position was fixed with synthetic implants or autologous tissue with addition of fibrin glue. In 7 patients, we performed implant interposition with placement of protectors on the brainstem between cranial nerves and vessels. In 2 cases, vascular transposition was accompanied by fixation of their position at a distance to prevent contact of implants with nerve fibers.

Complete regression of VGPN within 2—3 weeks after VD was considered an excellent outcome, while prolonged persistence of pain of any intensity and frequency was a poor outcome. Postoperative follow-up averaged 8.5 years (range 3—15).

Results

MRI revealed compression of cranial nerves IX—X near the brainstem in all patients on the side of pain manifested by transverse arrangement of vessels and nerve fibers **(Fig. 1)**. On contralateral side, there were only vascular contacts at the level of cisternal segments of caudal cranial nerves.

Crossed neurovascular conflict was caused by PICA in 6 cases, PICA and PICA in 2 cases, PICA and VA in 1 case. PICA-induced compression manifested itself as a caudal arcuate curvature of arteries with posterior displacement of cranial nerves IX—X. There was a similar dislocation of nerve fibers following compression by upwardly directed PICA loops. VA-induced compression consisted of transverse effect of VA and PICA on the nerve fibers **(Fig. 2)**. Short cerebellar arteries required implant interposition while vascular transposition was possible in case of sufficient length of cerebellar vessels **(Fig. 3)**.

There were no fatal outcomes or severe neurological complications. One patient had moderate postoperative hoarseness without swallowing impairment. These disorders regressed after 1.5 months. VGPN regressed in all cases, and one patient also experienced regression of bradycardia accompanying pain attacks. Postoperative MRI revealed implants on brainstem surface and no neurovascular conflicts **(Fig. 4)**. There were excellent results throughout the long-term follow-up period without VGPN recurrence.

Discussion

MRI in patients with VGPN allows for assessment of topographic relationships of cranial nerves with cerebellopontine angle vessels and neoplasms. Transverse vascular adhesion to cranial nerves IX—X near the brainstem is a neuroimaging sign of neurovascular conflict. No clear MR signs does not rule out vascular compression of cranial nerves due to difficult assessment of neurovascular relationships.

Xia L. et al. [7] found compression of cranial nerves IX—X by PICA in 72.3% of patients with VGPN, VA-induced compression — in 6.1%, vein-induced compression — in 4.4%. Various types of neurovascular conflict caused by combined effects of PICA, PICA, VA and veins were observed in 17.2% of cases. Similar results are presented in publications summarizing surgical findings in other series of patients with VGPN [1—3, 5—10].

VD implies retraction of vessels from caudal nerves with subsequent fixation of new anatomical relationships. Mobilization of compressing vessels depends on topographic relationships of cranial nerves IX—X. Therefore, we performed arachnoid dissection above and/or below nerve roots, as well as between glossopharyngeal and vagus nerves. Only vessels located transverse to nerve fibers were displaced away from the brainstem. Vessels lying along the roots and not exerting cross-current influence were not surgically manipulated to reduce the risk of traction-induced injury and postoperative cranial nerve dysfunction. VD was complete after eliminating contacts between cranial nerves and vessels, as well as fixation of vessels and implants in new positions. Technical variants of VD depended on topographic location of vascular loops and length of vessels extending to the brainstem. We used vascular transposition in case of sufficient length of arteries allowing their retraction, while implant interposition was preferable for short vessels [4]. If PICA passed between nerve fibers and its retraction was impossible, we displaced the artery laterally from the medulla oblongata for decompression of cranial nerves IX—X near the brainstem.

Compressing vessels in patients with VGPN are usually located on brainstem surface and affect nerve root segments with central myelin. There may be additional or isolated compression of cisternal segments of cranial nerves IX—X, and positive results of VD in these cases indicate significance of distal compression in patients with VGPN. Kim M.K. et al. [8] found variable localization of neurovascular conflicts. Thus, nerve compression only near the brainstem was observed in 63.3% of cases, combined compression of these segments and distal nerves — in 26.7%, isolated compression of distal segments — in 10.0% of cases. The authors emphasize no correlation between location of neurovascular conflicts, clinical manifestations and postoperative outcomes.

Different lengths of glial segments of cranial nerves explain VGPN following compression of cisternal segments of nerve roots. Central myelin in fibers of cranial nerves IX and X comprises 1.51±0.39 and 1.63±1.15 mm, respectively. In case of significant length of glial segments, vascular compression of cisternal segment will lead to VGPN, as in cases of trigeminal neuralgia [14, 15]. However, neurovascular conflicts with simultaneous effect of vessels on proximal and cisternal nerve segments complicates analysis of pathogenetic significance of each site of vascular compression due to impossible intraoperative estimation of length of glial nerve segments. In such cases, VD should eliminate neurovascular contacts along the entire length of cranial nerves [8, 15].

Unilateral combination of trigeminal neuralgia with VGPN is very rare, and differential diagnosis with mandibular neuralgia can be a challenging clinical task. In such cases, surgical treatment cannot be accurately based on MRI due to highly variable neurovascular relationships. Simultaneous exploration of trigeminal root and cranial nerves IX—X appears to be a reasonable surgical approach. Intraoperative confirmation of crossed vascular compression of trigeminal and/or caudal nerve roots allows for regression of neurovascular conflicts and neuralgic syndrome [8, 16, 17].

Although VD is effective for VGPN, some authors additionally transect the glossopharyngeal nerve and the upper fibers of the vagus nerve [2, 3]. However, selective partial rhizotomy may be accompanied by disorders of phonation and swallowing, while destruction of cranial nerves IX—X should be performed under neurophysiological monitoring for intraoperative assessment of functional significance of vagus nerve fibers [2, 3].

The rate of excellent outcomes after VD in small series of patients with VGPN varies significantly (50—94%). In recent years, complete regression of pain syndrome has been achieved in 90—100% of cases [1—13]. Xia L. et al. [7] analyzed early postoperative neurological disorders after VD. These events occurred in 16.2% of cases with facial muscle paresis in 0.88%, dysphagia in 7%, hoarse-

ness in 7.5% and combination of dysphagia and hoarseness in 0.88% of cases. Mean follow-up was 54 months. Cranial nerve dysfunction significantly regressed (dysphagia — 0.44%, hoarseness — 0.44%). These transient dysfunctions, as in our first case, were caused by surgical manipulations and, possibly, interposition of large implants.

Long-term recurrences of VGPN are associated with renewed vascular compression and, more often, adhesions around nerve roots and implants. Vascular transposition is preferable to reduce recurrence because this approach ensures decompression without contact between implants and nerve roots. Redo surgeries combining vascular transection, dissection of dense arachnoid adhesions and selective rhizotomy lead to pain regression, but accompanied by higher incidence of IX—X cranial nerve dysfunction [2, 5, 7, 8, 12, 13].

Conclusion

The main etiologic factor of VGPN is vascular compression of cranial nerves IX—X near the brainstem, most often caused by posterior inferior cerebellar artery and, much less frequently, anterior inferior cerebellar and vertebral arteries. Surgical treatment eliminates neurovascular conflict thorough retraction of vessels from cranial nerves with fixation of their new location by implants. Vascular decompression with implant interposition or vascular transposition is a highly effective surgical treatment for VGPN with low rate of neurological complications and recurrences.

Author contribution:
Concept and design of the study — Grigoryan G.Yu., Usachev D.Yu., Grigoryan Yu.A.
Collection and analysis of data — Grigoryan G.Yu., Usachev D.Yu., Grigoryan Yu.A.
Statistical analysis — Grigoryan G.Yu., Usachev D.Yu., Grigoryan Yu.A.
Writing the text — Grigoryan G.Yu., Usachev D.Yu., Grigoryan Yu.A.
Editing — Grigoryan G.Yu., Usachev D.Yu., Grigoryan Yu.A.

No conflict of interests to declare.

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