Microvascular decompression for glossopharyngeal neuralgia

For glossopharyngeal neuralgia treatment, should pharmacologic management be ineffective, surgical intervention is indicated. The first-choice treatment is typically microvascular decompression (MVD), as it has the highest initial and long-term success rates.

In 1932, Walter Edward Dandy [1] thought that the operative approach of GPN was the same with trigeminal neuralgia or Meniere's disease.

Laha and Jannetta [2] proposed that GPN could be treated by surgically relieving the pressure that offending vascular structures imposed on the glossopharyngeal nerves.

Resnick et al. [3] reporteded excellent postoperative surgical results for 79%.

Patel et al. reported in 217 a immediate success rate of 90% [4].

There are three types of neurovascular compression (NVC): type I – NVC at the root entry zone (REZ) of the IX CN within the retro-olivary sulcus; type II – the vertebral artery causes NVC at the IX CN REZ by the shoulder of the artery, and the type III - a "sandwich-like" compression where the vertebral artery and the PICA perform a combination of NVC [5].

Technique

Once the anesthetic induction and intubation have been performed, the patient should be positioned in lateral decubitus fashion, fixing the head with a Mayfield head clamp, followed by the placement of an axillary roll. The neck should be narrowed with slight flexion and rotated approximately 10 degrees to the affected side. The vertex is tilted 15 degrees toward the floor. The shoulder is pulled out of the way and finally the patient is accommodated in such a way that the table can be rotated laterally or adjusted for a Trendelenburg position or reverse Trendelenburg position. For the incision, the mastoid eminence is initially demarcated, then a line is drawn from the external auditory canal to the inion to mark the transverse sinus. Then, a 3-4 cm arcuate or linear incision is performed, with the concave side toward the ear. Half of the incision should be above the mastoid notch or even more posteriorly in large, muscular or dolichocephalic patients. Subsequently, a retractor is placed and the bone is opened with a perforator, making sure to use bone wax in case of bleeding and filling the mastoid cells.

Ordónez-Rubiano et al. propose to target the opening of the bone depending on the CN affected. Three different approaches could be performed. The superior for the V CN (mini extreme-lateral or microasterional), the middle for VII and VIII CNs (usual for the cerebellopontine angle), and the inferior for the IX to XII CNs (mini far-lateral).

Once the dura is exposed, it is incised and stretched. The form in which the dura is opened includes the L or reverse L shape, 3-5 mm parallel to the sigmoid sinus and to the floor of the posterior fossa, after which they are secured with sutures for a wider exposure. A retractor is placed under the cerebellum and raised from its inferolateral margin, after which the microscope is introduced, and the retractor is advanced anteriorly until the spinal part of the XI CN is observed, the arachnoid is

dissected, which allows to elevate the cerebellum and expose the remaining CNs within the jugular foramen. Once the rootlets of the IX CN are identified, they are separated from the rootlets of the X and XI CNs. The involved vessel is identified and dissected before the decompression and finally, the Teflon is placed between the two structures [6].

If there is no NVC, the glossopharyngeal nerve and the upper bundle of the X CN can be sectioned [7].

Case series

2018

Between 2006 and 2016, 228 idiopathic GPN patients underwent MVD in our department. Those cases were retrospectively reviewed with emphasis on intraoperative findings and long-term postoperative outcomes. The average period of follow-up was 54.3 ± 6.2 months.

Intraoperatively, the culprit was identified as the posterior inferior cerebellar artery (PICA) in 165 cases (72.3%), the vertebral artery (VA) in 14 (6.1%), vein in 10 (4.4%), and a combination of multiple arteries or venous offending vessels in 39 (17.2%). The immediately postoperative outcome was excellent in 204 cases (89.5%), good in 12 (5.3%), fair in 6 (2.6%) and poor in 6 (2.6%). More than 5-year follow-up was obtained in 107 cases (46.9%), which presented as excellent in 93 (86.9%), good in 6 (5.6%), fair in 3 (2.8%) and poor in 5 (4.7%). Thirty-seven (16.2%) of the patients experienced some postoperative neurological deficits immediately, such as dysphagia, hoarseness and facial paralysis, which has been improved at the last follow-up in most cases, except 2.

This investigation demonstrated that MVD is a safe and effective remedy for treatment of GPN [8].

2017

30 patients with intractable primary typical Glossopharyngeal Neuralgia who underwent MVD without rhizotomy and were followed for more than 2 years were included in the analysis. Each MVD was performed using one of four different surgical techniques: interposition of Teflon pieces, transposition of offending vessels using Teflon pieces, transposition of offending vessels using a fibrin-glue-coated Teflon sling, and removal of offending veins.

The posterior inferior cerebellar artery was responsible for neurovascular compression in 27 of 30 (90%) patients, either by itself or in combination with other vessels. The location of compression on the glossopharyngeal nerve varied; the root entry zone (REZ) only (63.3%) was most common, followed by both the REZ and distal portion (26.7%) and the distal portion alone (10.0%). In terms of detailed surgical techniques during MVD, the offending vessels were transposed in 24 (80%) patients, either using additional insulation, offered by Teflon pieces (15 patients), or using a fibrin glue-coated Teflon sling (9 patients). Simple insertion of Teflon pieces and removal of a small vein were also performed in five and one patient, respectively. During the 2 years following MVD, 29 of 30 (96.7%) patients were asymptomatic or experienced only occasional pain that did not require medication. Temporary hemodynamic instability occurred in two patients during MVD, and seven patients experienced transient postoperative complications. Neither persistent morbidity nor mortality was reported.

This study demonstrates that MVD without rhizotomy is a safe and effective treatment option for GPN [9)].

From January 2004 to June 2006, 35 consecutive patients were diagnosed with GPN. All of them underwent MVD. Demographic data, clinical presentation, operative findings, clinical results, operative complications were reviewed.

A total of 33 patients (94.3%) experienced complete pain relief immediately after MVD. Long-term follow-up was available for 30 of these 35 patients, and 28 of these 30 patients continued to be pain-free. There was no long-term operative morbidity in all cases. One patient had a cerebrospinal fluid leak and 1 case presented with delayed facial palsy.

Classic GPN is usually caused by pulsatile neurovascular compression of the glossopharyngeal and vagus rootlets. MVD is a safe, effective, and durable operation for GPN [10)].

2015

A retrospective review of the case notes of patients who had undergone surgery for GPN in the authors' department between 2008 and 2013 was performed to investigate baseline characteristics and immediate outcomes during the hospitalization. For the long-term results, a telephone survey was performed, and information on pain recurrence and permanent complications was collected. Pain relief meant no pain or medication, any pain persisting after surgery was considered to be treatment failure, and any pain returning during the follow-up period was considered to be pain recurrence. For comparative study, the patients were divided into 2 cohorts, that is, patients treated with GPNR alone and those treated with GPNR+VNR.

One hundred three procedures, consisting of GPNR alone in 38 cases and GPNR+VNR in 65 cases, were performed in 103 consecutive patients with GPN. Seventy-nine of the 103 patients could be contacted for the follow-up study, with a mean follow-up duration of 2.73 years (range 1 month-5.75 years). While there were similar results (GPNR vs GPNR+VNR) in immediate pain relief rates (94.7% vs 93.8%), immediate complication rates (7.9% vs 4.6%), and long-term pain relief rates (92.3% vs 94.3%) between the 2 cohorts, a great difference was seen in long-term complications (3.8% vs 35.8%). The long-term complication rate for the combined GPNR+VNR cohort was 9.4 times higher than that in the GPNR cohort. There was no operative or perioperative mortality. Immediate complications occurred in 6 cases, consisting of poor wound healing in 3 cases, and CSF leakage, hoarseness, and dystaxia in 1 case each. Permanent complications occurred in 20 patients (25.3%) and included cough while drinking in 10 patients, pharyngeal discomfort in 8 patients, and hoarseness and dysphagia in 1 case each.

In general, this study indicates that GPNR alone or in combination with VNR is a safe, simple, and effective treatment option for GPN. It may be especially valuable for patients who are not suitable for the microvascular decompression (MVD) procedure and for surgeons who have little experience with MVD. Of note, this study renews the significance of GPNR alone, which, the authors believe, is at least valuable for a subgroup of GPN patients, with significantly fewer long-term complications than those for rhizotomy for both glossopharyngeal nerve and rootlets of the vagus nerve [11)].

2002

Patel et al. present the experience with more than 200 patients and conducted a retrospective review of the database and identified patients who presented for treatment of presumed GPN. When possible, patients were contacted by telephone for collection of follow-up information regarding symptom relief, complications, functional outcomes, and patient satisfaction. Univariate and multivariate analyses were performed to identify predictors of good outcomes after MVD. Subgroup analyses were performed with quartiles of approximately 50 patients each, for assessment of the effects of improvements in techniques and anesthesia during this 20-year period.

They observed GPN to be more common among female (66.8%) than male (33.2%) patients, with an overall mean patient age of 50.2 years (standard deviation, 14.4 yr). The most common presenting symptoms were throat and ear pain and throat pain alone, and the mean duration of symptoms was 5.7 years (standard deviation, 5.8 yr; range, 1-32 yr). Symptoms appeared almost equally on the left side (54.8%) and the right side (45.2%). The overall immediate success rate exceeded 90%, and long-term patient outcomes and satisfaction were best for the typical GPN group (with pain restricted to the throat and palate). Complication rates decreased across quartiles for all categories evaluated.

MVD is a safe, effective form of therapy for GPN. It may be most beneficial for patients with typical GPN, especially when symptoms are restricted to deep throat pain only [12)].

1995

Since 1971, 40 patients have undergone microvascular decompression of the glossopharyngeal and vagus nerves for treatment of typical glossopharyngeal neuralgia. This procedure provided excellent immediate results (complete or > 95% relief of pain) in 79%, with an additional 10% having a substantial (> 50%) reduction in pain. Long-term follow-up (mean, 48 mo; range, 6-170 mo) reveals excellent results (complete or > 95% reduction in pain without any medication) in 76% of the patients and substantial improvement in an additional 16%. There were two deaths at surgery (5%) both occurring early in the series as the result of hemodynamic lability causing intracranial hemorrhage. Three patients (8%) suffered permanent 9th nerve palsy [13)].

1986

20 patients who had undergone microvascular decompression for the treatment of “idiopathic” trigeminal neuralgia (9 cases), hemifacial spasm (7 cases), glossopharyngeal neuralgia (3 cases) and paroxysmal vertigo and tinnitus (1 case) were followed up for 25 months on average. Permanent relief of symptoms was observed in 19 (95%), with sparing of cranial nerve function. Analysis of the clinical data shows that the patients described in the present series did not differ from those considered to suffer from “idiopathic” cranial nerve dysfunction syndromes. The importance of vascular cross compression as etiological factor in such conditions is stressed and the pathophysiology discussed. The term “cryptogenic” applied to trigeminal neuralgia or hemifacial spasm thus needs revising. Lastly, the indications of microvascular decompression in the treatment of “cryptogenic” cranial nerve dysfunction syndromes are defined [14)].

1977

Microsurgical observations werw made of the cranial nerve root entry or exit zones 117 patients operated upon for the treatment of hyperactive-hypoactive dysfunction syndromes (trigeminal neuralgia, hemifacial spasm, acoustic nerve dysfunction, and glossopharyngeal neuralgia). Cross-compression or distortion of the appropriate nerve root at its entry or exit zone was noted in all patients. This compression or distortion was usually caused by normal or arteriosclerotic, elongated arterial loops, it was usually relieved by decompressive microsurgical techniques. A small percentage of patients were found to have compression of the nerve root at the entry-exit zone by a tumor, a vein, or some other structural abnormality; they were relieved by tumor excision or other measures as described. Relief was gradual postoperatively if the treated nerve was not stroked or manipulated at operation but it was immediate if the nerve was manipulated. Preoperative evidence of decreased nerve function improved postoperatively [15].

Case reports

A case of coexistent glossopharyngeal neuralgia and hemifacial spasm was treated by transposition of the vertebral artery. A 60-year-old man was referred to our hospital due to pain in the left posterior part of the tongue that was difficult to control with oral medication at a local hospital. The diagnosis was left glossopharyngeal neuralgia based on the symptoms, imaging findings, and lidocaine test results. Moreover, the patient had left hemifacial spasm. Microvascular decompression was performed, which confirmed that the vertebral artery was compressing the lower cranial nerve and the posterior inferior cerebellar artery was compressing the root exit zone of the facial nerve. The vertebral artery and posterior inferior cerebellar artery were transposed using TachoSil. After the surgery, both glossopharyngeal neuralgia and hemifacial spasm disappeared, and the patient was discharged [16].

1985

A case of combined trigeminal and glossopharyngeal neuralgia is described. The superior cerebellar artery and normal choroid plexus compressed and indented the root entry zones of the trigeminal and glossopharyngeal nerves, respectively. Complete relief was obtained after microvascular decompression and resection of the choroid plexus [17].

A case of glossopharyngeal neuralgia associated with episodic cardiac arrest and syncope is presented. Posterior fossa exploration showed that the left glossopharyngeal and vagus nerves were compressed by the posterior inferior cerebellar artery. Microvascular decompression resulted in complete relief of glossopharyngeal neuralgia, cardiac syncope, and seizure. The mechanism of glossopharyngeal neuralgia associated with cardiac syncope is discussed [18].

Murasawa A, Yamada K, Hayakawa T, Aragaki Y, Yoshimine T. Glossopharyngeal neuralgia treated by microvascular decompression–case report. Neurol Med Chir (Tokyo). 1985 Jul;25(7):551-3. PubMed PMID: 2415848 [19].

1)
Dandy WE (1932) The treatment of trigeminal neuralgia by the cerebellar route. Ann Surg 96:787–795
2)
Laha RK, Jannetta PJ (1977) Glossopharyngeal neuralgia. J Neurosurg 47:316–320
3) , 13)
Resnick DK, Jannetta PJ, Bissonnette D, Jho HD, Lanzino G. Microvascular decompression for glossopharyngeal neuralgia. Neurosurgery. 1995 Jan;36(1):64-8; discussion 68-9. PubMed PMID: 7708170.
4) , 12)
Patel A, Kassam A, Horowitz M, Chang YF. Microvascular decompression in the management of glossopharyngeal neuralgia: analysis of 217 cases. Neurosurgery. 2002 Apr;50(4):705-10; discussion 710-1. PubMed PMID: 11904019.
5)
Tanrikulu L, Hastreiter P, Dörfler A, Buchfelder M, Naraghi R. Classification of neurovascular compression in glossopharyngeal neuralgia: Three-dimensional visualization of the glossopharyngeal nerve. Surg Neurol Int. 2015 Dec 24;6:189. doi: 10.4103/2152-7806.172534. eCollection 2015. PubMed PMID: 26759734; PubMed Central PMCID: PMC4697202.
6)
Ordónez-Rubiano EG, García-Chingaté CC, Rodríguez-Vargas S, Cifuentes-Lobelo HA, Perilla-Cepeda TA. Microvascular Decompression for a Patient with a Glossopharyngeal Neuralgia: A Technical Note. Cureus. 2017 Jul 20;9(7):e1494. doi: 10.7759/cureus.1494. PubMed PMID: 28948114; PubMed Central PMCID: PMC5606712.
7)
Rey-Dios R, Cohen-Gadol AA. Current neurosurgical management of glossopharyngeal neuralgia and technical nuances for microvascular decompression surgery. Neurosurg Focus. 2013 Mar;34(3):E8. doi: 10.3171/2012.12.FOCUS12391. Review. PubMed PMID: 23451790.
8)
Xia L, Li YS, Liu MX, Zhong J, Dou NN, Li B, Li ST. Microvascular decompression for glossopharyngeal neuralgia: a retrospective analysis of 228 cases. Acta Neurochir (Wien). 2018 Jan;160(1):117-123. doi: 10.1007/s00701-017-3347-1. Epub 2017 Nov 4. PubMed PMID: 29103137.
9)
Kim MK, Park JS, Ahn YH. Microvascular Decompression for Glossopharyngeal Neuralgia: Clinical Analyses of 30 Cases. J Korean Neurosurg Soc. 2017 Nov;60(6):738-748. doi: 10.3340/jkns.2017.0506.010. Epub 2017 Oct 25. PubMed PMID: 29142635; PubMed Central PMCID: PMC5678068.
10)
Zhao H, Zhang X, Zhu J, Tang YD, Li ST. Microvascular Decompression for Glossopharyngeal Neuralgia: Long-Term Follow-Up. World Neurosurg. 2017 Jun;102:151-156. doi: 10.1016/j.wneu.2017.02.106. Epub 2017 Mar 2. PubMed PMID: 28263933.
11)
Ma Y, Li YF, Wang QC, Wang B, Huang HT. Neurosurgical treatment of glossopharyngeal neuralgia: analysis of 103 cases. J Neurosurg. 2015 Sep 4:1-5. [Epub ahead of print] PubMed PMID: 26339847.
14)
Michelucci R, Tassinari CA, Samoggia G, Tognetti F, Calbucci F. Intracranial microvascular decompression for “cryptogenic” hemifacial spasm, trigeminal and glossopharyngeal neuralgia, paroxysmal vertigo and tinnitus: II. Clinical study and long-term follow up. Ital J Neurol Sci. 1986 Jun;7(3):367-74. PubMed PMID: 3733417.
15)
Jannetta PJ. Observations on the etiology of trigeminal neuralgia, hemifacial spasm, acoustic nerve dysfunction and glossopharyngeal neuralgia. Definitive microsurgical treatment and results in 117 patients. Neurochirurgia (Stuttg). 1977 Sep;20(5):145-54. PubMed PMID: 198692.
16)

Fujii T, Otani N, Otsuka Y, Matsumoto T, Tanoue S, Ueno H, Tomura S, Tomiyama A, Toyooka T, Wada K, Mori K. [A Case of Coexistent Glossopharyngeal Neuralgia and Hemifacial Spasm Successfully Treated with Transposition of the Vertebral Artery]. No Shinkei Geka. 2017 Jun;45(6):503-508. doi: 10.11477/mf.1436203540. Review. Japanese. PubMed PMID: 28634310.
17)

Yoshioka J, Ueta K, Ohmoto T, Fujiwara T, Tabuchi K. Combined trigeminal and glossopharyngeal neuralgia. Surg Neurol. 1985 Oct;24(4):416-20. PubMed PMID: 4035551.
18)

Tsuboi M, Suzuki K, Nagao S, Nishimoto A. Glossopharyngeal neuralgia with cardiac syncope. A case successfully treated by microvascular decompression. Surg Neurol. 1985 Sep;24(3):279-83. PubMed PMID: 4023909.
19)

Murasawa A, Yamada K, Hayakawa T, Aragaki Y, Yoshimine T. Glossopharyngeal neuralgia treated by microvascular decompression–case report. Neurol Med Chir (Tokyo). 1985 Jul;25(7):551-3. PubMed PMID: 2415848.