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Review Article

Microvascular decompression for glossopharyngeal neuralgia through a microasterional approach: A case series

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Abstract

Background: Glossopharyngeal neuralgia (GPN) is an uncommon craniofacial pain syndrome. It is characterized by a sudden onset lancinating pain usually localized in the sensory distribution of the IX cranial nerve associated with excessive vagal outflow, which leads to bradycardia, hypotension, syncope, or cardiac arrest. This study aims to review our surgical experience performing microvascular decompression (MVD) in patients with GPN.

Methods: Over the last 20 years, 14 consecutive cases were diagnosed with GPN. MVD using a microasterional approach was performed in all patients. Demographic data, clinical presentation, surgical findings, clinical outcome, complications, and long-term follow-up were reviewed.

Results: The median age of onset was 58.7 years. The mean time from onset of symptoms to treatment was 8.8 years. Glossopharyngeal and vagus nerve compression was from the posterior inferior cerebellar artery in eleven cases (78.5%), vertebral artery in two cases (14.2%), and choroid plexus in one case (7.1%). Postoperative mean follow-up was 26 months (3–180 months). Pain analysis demonstrated long-term pain improvement of 114 ± 27.1 months and pain remission in 13 patients (92.9%) ($P = 0.0001$) two complications were documented, one patient had a cerebrospinal fluid leak, and another had bacterial meningitis. There was no surgical mortality.

Conclusions: GPN is a rare entity, and secondary causes should be discarded. MVD through a retractorless microasterional approach is a safe and effective technique. Our series demonstrated an excellent clinical outcome with pain remission in 92.9%.

Key Words: Glossopharyngeal nerve, microvascular decompression, neuralgia, neurovascular compression, vagus nerve

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INTRODUCTION

Glossopharyngeal neuralgia (GPN) is an uncommon craniofacial pain syndrome, representing 0.2–1.3%[3,8] of facial pain syndromes, with an annual incidence of 0.7 cases per 100,000 habitants per year according to a population-based study.[14] It is characterized by a sudden onset of lancinating acute pain, lasting seconds to minutes, usually in the sensory distribution of the auricular and pharyngeal branches of the of the IX and X cranial nerve. The pain is felt in the pharynx, tongue, tonsillar fossa, internal ear, and mandible angle. In some cases, it is associated with excessive vagal outflow; which leads to bradycardia, hypotension, syncope,[16] or cardiac arrest.[37,38]

The first GPN description is attributed to Theodore H. Weisenburg in 1910.[24] Dandy elucidated the pathophysiology of trigeminal neuralgia and proposed vascular compression as the main etiology, causing demyelization, and ephaptic transmission;[4,24] which is the same pathophysiology of GPN. First line medical treatment, including carbamazepine and gabapentin, may sometimes improve pain paroxysms.[28] However, in cases with refractory GPN various surgical approaches have been attempted. In 1920, Sicard and Robineau[31] proposed sectioning the glossopharyngeal nerve through the neck as a definitive treatment; which evolved to intracranial rhizotomy of the glossopharyngeal nerve performed by Dandy.[8] Later on, Sweet,[35] introduced percutaneous compression at the middle fossa and finally Jannetta, popularized microvascular decompression (MVD) as a definitive surgical treatment for this pathology.[12,17,32] MVD series have reported good outcomes in 90–98%, long-term pain improvement have been observed in 64% with a low mortality ranging from 0% to 5.8%.[13]

This study aims to review our surgical experience performing MVD using a microasterional approach in patients with GPN.

METHODS

Patients

This study is a consecutive case series of 14 patients, who underwent MVD for the treatment of idiopathic GPN at the National Institute of Neurology and Neurosurgery "Manuel Velasco Suárez", in Mexico City, between 1994 and 2014. The senior author (Rogelio Revuelta-Gutiérrez) performed all the surgeries. A retrospective analysis of the clinical charts was performed. Patient data including gender, the age of onset, symptoms, previous medical management, operative findings, complications, and clinical outcome were collected. Pain intensity was graded according a three-grade scale: (1) No pain, no need for medication; (2) pain controlled with medical management; (3) pain not controlled with medication. All patients were previously managed with conservative treatment including carbamazepine, gabapentin, and pregabalin. No pain improvement for at least 6 months before surgical procedure was documented. Diagnosis work-up included a 3T magnetic resonance imaging (MRI). T1, T2, gadolinium-enhanced and FIESTA sequences were assessed to discard a secondary cause of the symptoms and identify vascular compression.

A statistical analysis was performed using SPSS Version 20 (IBM SPSS Statistics, New York, USA). Categorical variables were expressed as proportions and continuous variables were expressed using means and standard deviations. Clinical outcome was evaluated according to the surgical management, use of medications, pain recurrence, and postoperative complications. Descriptive statistics was performed for the patient data and the grade of pain preoperatively and postoperatively was analyzed using Wilcoxon signed-rank test. $P < 0.05$ was considered statistically significant.

Surgical technique

Under general anesthesia patients were placed in park bench position with the head fixed in a Mayfield skull clamp. The upper shoulder was retracted, and the head was rotated 60° to the opposite side of the exposure with slight cervical lateral tilting (10°) toward the floor. A 5 cm retrosigmoid incision centered over the asterion was performed and a keyhole (2.5–3 cm) asterional craniectomy exposed the angle of the transverse and sigmoid sinuses [Figures 1 and 2a]. Curvilinear durotomy was performed under microscope magnification and intradural dissection started toward the dural angle between the tentorium and petrous surface [Figure 2b]. Cerebrospinal fluid (CSF) was released through arachnoid dissection without using cerebellar retractors. The dissection was directed caudally, and the lower vascular nervous complex involving the glossopharyngeal

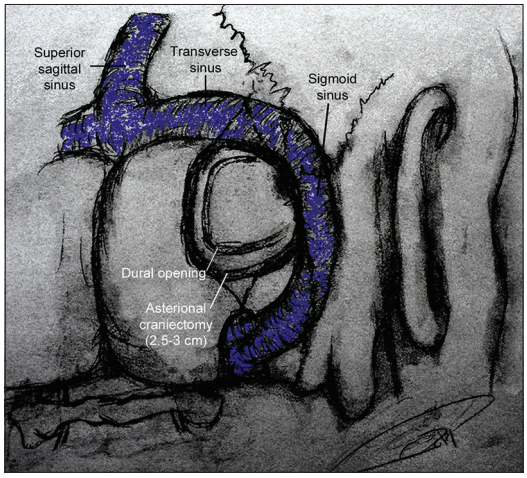

Figure 1: Craniotomy size and reference

nerve was exposed, identifying its exit through the jugular foramen. Once the identification of the vascular element compressing the glossopharyngeal nerve was observed [Figure 2c], blunt dissection was done, and a small piece or multiple pieces of Teflon were placed between the glossopharyngeal nerve and the compressing vessels (arterial or venous) [Figure 2d, Video 1].

RESULTS

Patient demographics

A total of 14 patients were diagnosed with GPN and were surgically treated [Table 1]. The median age of onset was 58.7 ± 11 years, with a male to female ratio of (1:1.8). The mean time duration from symptom onset to surgery was 8.8 years. Pain trigger was described when swallowing in seven cases, talking in four cases and without previous stimuli in three cases. Carbamazepine was the most used medication (78%), followed by gabapentin and pregabalin; 64.2% patients were on more than one drug. All patients from this study had no clinical improvement with full dose carbamazepine, gabapentin, pregabalin, and daily analgesic medication. Three patients were misdiagnosed before they were referred to our institution; stiloidectomy was performed in two patients (14.3%) and previous dental surgery in one patient (7.1%). Mean time from diagnosis to surgery was 106.3 ± 95.7 months (males 86.4 ± 78.4 months and females 117.3 ± 106.9 months; $P = 0.58$).

Table 1: Clinical data and outcome of patients with glossopharyngeal neuralgia

	n (%)
Pain localization	
Pharyngeal	13 (92.9)
Preauricular	1 (7.1)
Pain side	
Left	11 (78.6)
Right	3 (21.4)
Pain trigger	
Abrupt onset	3 (21.4)
Swallowing	7 (50)
Talking	4 (28.6)
Pain irradiation	
Yes	6 (42.9)
No	8 (57.1)
Preoperative pain	
Grade 3	14 (100)
Long-term follow-up postoperative pain	
Grade 1	13 (92.9)
Grade 2	1 (7.1)
Grade 3	0 (0)
Complications	
Cerebrospinal fluid leak	1 (7.1)
Meningitis	1 (7.1)

The pain was more common on the left side (78.6%) compared to the right (21.4%). The primary location of the pain was pharyngeal in 13 cases (92.9%) and preauricular in one case (7.1%). Pain irradiation was referred in 6 cases (42.9%), 5 of them to the preauricular area and one to the pharynx. One patient (7.1%) presented with syncope and another one had an intraoperative vasovagal reflex during decompression.

Neuroradiological and operative findings

MRI showed vascular compression from the posterior inferior cerebellar artery (PICA) [Figure 3] in three patients (21.4%), vertebral-basilar arteries in three patients (21.4%), and an inflammatory process in one patient (7.1%). Seven patients were reported as normal on MRI scan (49.7%). At the time of the surgery, all 14 patients were found to have compression of the vagal and glossopharyngeal nerve roots. Vascular compression was from PICA in 11 cases (78.5%), vertebral artery in two cases (14.2%), and compression from the choroid plexus in one case (7.1%).

Clinical outcome

All 14 patients were contacted for long-term follow-up. Postoperative mean follow-up of was 26 months (3–180 months). All patients referred initial pain relief, and 13 were pain-free with no need of medication in the long-term follow-up. Only one patient referred pain 1 month after surgery and was treated with carbamazepine with complete relief of the pain and no further surgery was required. Pain analysis demonstrated long-term pain improvement of 114 ± 27.1 months and pain remission in 13 patients (92.9%) ($P = 0.0001$) [Table 1].

Complications

Two patients presented complications related to surgical treatment. One patient presented with CSF leak, which resolved with lumbar drainage and acetazolamide 500 mg TID for 5 days without any complications. The second patient presented with meningitis and was treated with intravenous vancomycin 1 g. BID for 5 days recovering completely without clinical sequelae [Table 1]. There was no surgical mortality in this case series.

DISCUSSION

Wilfred Harris applied the term GPN when he described an entity similar to trigeminal neuralgia. At his initial report in 1937, Harris described two types of pathologies: Primary or idiopathic and secondary to carcinoma. Idiopathic GPN is explained due to nerve compression by a vessel, as it exits the medulla oblongata.[24] This theory is supported by the success of MVD in the treatment of this pathology.[32] The main symptom of the GPN is a lancinating pain lasting seconds to minutes. However, some cases have reported the presence of pain associated to syncope.[7] In this regard, Gardner associated the

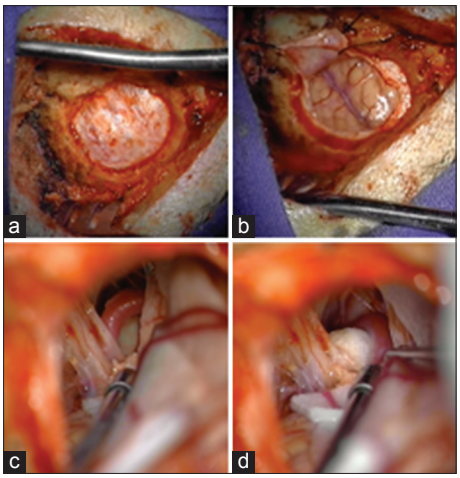

Figure 2: Glossopharyngeal microvascular decompression through a minimal invasive asterional approach. (a) Right microasterional approach (2.5–3 cm). (b) Durotomy exposing right cerebellar hemisphere, the base of the dural opening is reflected at the junction of the sigmoid and transverse sinus. (c) Cerebrospinal fluid drainage after arachnoid dissection allows proper visualization of the vertebral artery compressing the glossopharyngeal nerve. (d) A piece of Teflon is interposed between the affected nerve and the offending vessel

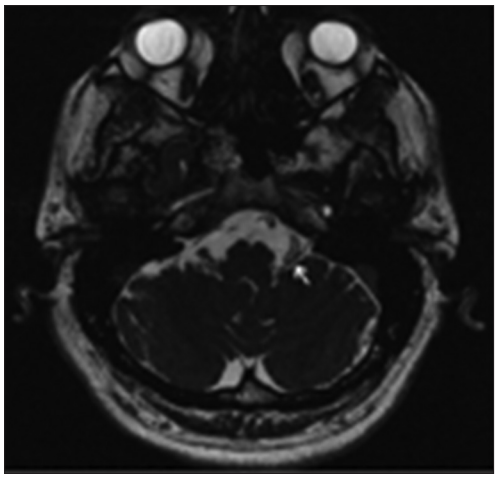

Figure 3: Preoperative axial magnetic resonance FIESTA image demonstrates glossopharyngeal nerve compression from the left vertebral artery

proximity of the glossopharyngeal nucleus to the vagal nucleus. The activation of the nucleus produces activation of the vagal nerve, which results in bradycardia and hypotension secondary to a decrease of the peripheral vascular resistance. Another theory explains the vascular resistance impairment secondary to inhibition of vasomotor centers.[16]

Traditionally, a lateral suboccipital approach provides adequate exposure to the trigeminal, facial, and lower cranial nerves. Kawashima *et al.*[15] proposed a transcondylar fossa approach advocating the wide operative view of the cerebellomedullary cistern, smaller retraction of the cerebellum, less risk of cranial nerve injury, and enough space to perform the sling retraction technique. However, we believed that a minimally invasive technique as an asterional approach described previously by the senior author[25] is enough for adequate exposure of PICA, vertebral artery, and the relationship with the glossopharyngeal nerve and the upper roots of the vagus nerve. There is no need of retractors, and after the CSF is released with adequate and careful arachnoid dissection, the cerebellum is out of the way, and there is enough space for working without the necessity of removing the jugular tubercle.

Jannetta,[12] popularized the MVD using a suboccipital craniotomy. After years of experience, the approach was modified according to the surgical goal. Initially, it is important to focus bone exposure to the junction of the transverse and sigmoid sinuses. A smaller tailored craniectomy according to the cranial nerve approach can be performed. For lower cranial nerve exposure; McLaughlin *et al.*,[20] recommended a triangular craniectomy with the apex at the edge of the jugular bulb. In our experience, our circular microasterional craniectomy [Figure 2a] at the edge of the transverse and sigmoid sinuses gives enough bone exposure to access the trigeminal, facial, and glossopharyngeal nerves.

In the MVD series, the overall surgical mortality is 1.1%. The rate of long-term pain remission is 84.7% with recurrence in 7%. Transient X cranial nerve dysfunction occurred in 13.2% and permanent deficits in 5.5%.[27] In our case series, we did not have any mortality, and no permanent deficits occurred after the surgery. We did not have cerebellar lesions or hearing the loss in this case series; it is explained because we do not use retractors over the cerebellum, the surgical route place minimal traction on the VII–VIII nerve complex and we perform a careful microsurgical vascular dissection with minimal bipolar coagulation. However, we had two complications; a CSF leak and a case of meningitis that was successfully treated.

Rey-Dios and Cohen-Gadol demonstrated in his analyses that the most effective surgical procedure to treat GPN is the MVD.[27] Several studies used rhizotomy[2,9,13,17,29,36] as the preferred procedure, but a 3-fold increase in the risk of permanent postoperative vagus dysfunction[27] is objectionable in comparison to MVD. It is also well demonstrated that the rate of pain control is slightly better with rhizotomy (95%) than with MVD (86%).[27] However, in our series we had 92.9% pain remission with 3–180 months (mean 26 months) of follow-up; only one case had pain recurrence that was treated with carbamazepine. GPN is a rare condition in which the clinical findings are not always typical. The mean duration from symptom onset to surgery is 5–8 years.[13,23,30] In our

case series, we had a mean time for diagnosis of 8.8 years, however, despite the time for diagnosing GPN the clinical outcome of our patients is similar to the reported in the literature.[27]

It is important to rule out secondary causes such as neoplasm,[11] infections,[39] trauma,[39,40] vascular malformations,[10] Chiari malformation,[41] choroid plexus overgrowth,[22] Tornwaldt's cyst,[33] Eagle syndrome,[6] pontine lesions,[19] multiple sclerosis,[21] and previous surgical interventions (vagal nerve stimulator).[5] It is essential to have a careful selection and an accurate diagnosis of idiopathic GPN to avoid negative exploratory operations. Two of our patients were previously diagnosed as Eagle syndrome, in both of them stiloidectomy was performed without pain improvement, and one of them had a tooth extraction before referral to our Institution. During the diagnosis workup, we ruled out secondary causes and confirmed an idiopathic GPN in all patients and MVD was performed.

As Lister *et al.*[18] previously described in a microsurgical anatomic study, PICA has the most variable course of the cerebellar arteries, but most of the time it passes under the glossopharyngeal nerve. In most of the recent clinical series[13,15,30] PICA is the most common vessel compressing the glossopharyngeal nerve. In our series, during dissection we found PICA compression in eleven cases (78.1%), in all of them we did the transposition of the vessel and apply Teflon in between the nerve and the vessel.

In refractory cases to MVD, we believe that sectioning the glossopharyngeal nerve and the upper roots of vagus nerve involved an unaccepted high morbidity. We advocate for compression of the glossopharyngeal and upper roots of the vagus nerve as a last option for pain recurrence as previously demonstrated for trigeminal neuralgia.[26] Other noninvasive treatment options have been described: Percutaneous radiofrequency neurolysis[1] is an alternative in cases who failed medical treatment or in which they cannot undergo intracranial surgery. Gamma Knife radiosurgery is also a potential option to relieve the pain without reported side effects but a high early recurrence risk.[34,42]

CONCLUSION

Glossopharyngeal MVD through a retractorless microasterional approach is a safe technique in which surgical anatomical knowledge is essential to obtain good results with minimal morbidity. Our series demonstrate an excellent clinical outcome (pain remission - 92.9%) following MVD for GPN.

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Conflicts of interest
There are no conflicts of interest.

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