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Glossopharyngeal neuralgia – A diagnostic challenge

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Abstract

Glossopharyngeal neuralgia (GPN), or better named vago-glossopharyngeal neuralgia (VGPN), is a rare disorder amounting to 1 % of the incidence of trigeminal neuralgia (TN). Pain is paroxysmal, of the electrical shooting type, and mainly provoked by stimulation of the pharynx or deep throat, especially during swallowing.

Due to its rarity, VGPN is often misdiagnosed. The front line of medical treatment is based on anticonvulsants. Surgery should be considered when the pain is refractory to medications. In most patients, the cause is neurovascular conflict on root entry zone (REZ) or midcistern portion, of the IXth and/or Xth cranial nerves.

Compressive vessels can be evidenced by means of a high sensibility and a high specificity resolution MR imaging in most centers. Present consensus is that the first option of neurosurgical treatment be microvascular decompression. In patients with precarious general conditions, stereotactic radiosurgery may be considered.

Also, thermo-rhizotomy at the pars nervosa of foramen jugularis or tractotomy-nucleotomy at brainstem may be alternatives, but these methods entail a significant risk of deficits. In this article, we reviewed the molecular and cellular pathogenesis of glossopharyngeal neuralgia and treatments of this disease.

Keywords: Glossopharyngeal neuralgia. Glossopharyngeal nerve. Vagus nerve. Microvascular decompression

Introduction

Glossopharyngeal neuralgia (GN) is a rare and pain syndrome in the sensory distribution of the ninth cranial nerve, also known as the glossopharyngeal nerve.

As per ICHD-3 (International Classification of Headache Disease- 3) classification, glossopharyngeal neuralgia is a disorder characterized by a brief episodic unilateral pain, with sharp and stabbing in character, with abrupt onset and cessation, in the glossopharyngeal nerve distribution (angle of the jaw, ear, tonsillar fossa and the tongue base).

It also involves the pharyngeal and auricular branches of CN X. Pain is commonly triggered by coughing, talking, and swallowing. Pain in glossopharyngeal neuralgia follows a relapsing and remitting pattern. It falls under the International Classification of Diseases (ICD) category as ICD-10-CM-G52.1.[1]

In 1910, T. Weisenberg was the first to describe the symptoms of glossopharyngeal neuralgia in a 35-year-old male with compression of the ninth cranial nerve by a tumor at the cerebellopontine (CP) angle.

W. Harris was the first physician to label these symptoms as "glossopharyngeal neuralgia" in 1921, based on his observation of two of his patients. In 1927, there were two significant developments for the understanding and treatment of glossopharyngeal neuralgia.

These included a published case series of 18 patients by J. Doyle and the first successful intracranial resection for glossopharyngeal neuralgia performed by W. Dandy.[2]

Riley et al. in 1942 noted that occasionally the painful attacks of glossopharyngeal neuralgia were associated with syncopal episodes, transient or persistent bradycardia, asystole, and even seizures.

When these features correlate with glossopharyngeal neuralgia, the term for this condition is vagoglossopharyngeal neuralgia. Glossopharyngeal neuralgia often gets misdiagnosed as trigeminal neuralgia since pain characteristics are very similar in both entities.[3]

History

Severe pain in the distribution of the glossopharyngeal nerve was first described by Weisenberg in 1910, in a 35-year-old male patient with a right cerebellopontine angle tumor [4].

In 1920, Sicard and Robineau presented three patients who had similar pain in the area of distribution of the glossopharyngeal nerve without any known cause, and described the first surgical treatment of GPN—extracranial nerve avulsion [5].

The current nomenclature "glossopharyngeal neuralgia" was introduced by Harris in 1921 to describe a rare condition characterized by paroxysms of agonizing pain located laterally at the back of the tongue, soft palate, throat, and postero-lateral pharynx [6].

Singleton in 1926 [7] and Dandy in 1927 described cases that were cured by intracranial sectioning of the glossopharyngeal nerve [8].

In 1977**,** Laha and Jannetta, after observing glossopharyngeal compression by the vertebral artery in six patients, proposed that GPN could be treated by microvascular decompression [9].

Thereafter, percutaneous surgical techniques used for trigeminal neuralgia treatment were also performed for GPN, including pulsed mode radiofrequency [10] and gamma knife radio surgery [11].

The concurrence of the GPN and TN was first noted in 1931 by Hesse [12], and in 1935 by Peet [13•], who reported on 14 cases, including five that had a combination of GPN and TN.

Etiology and pathogenesis

The majority of GPN are idiopathic, and a comprehensive head and neck clinical examination usually does not reveal any abnormality other than the identification of trigger points, and radiological examinations (including CT and MRI scans) might be within normal limits as well [14].

Idiopathic forms of GPN might possibly be caused by severe demyelination and axon-degeneration of IX and X cranial nerve fibers [15]. In vagoglossopharyngeal neuralgia, arrhythmias and syncope could be associated with neuralgia, due to the nerve supplying the carotid sinus [16].

Although most of the GPN cases are idiopathic, some of them might be secondary to other causes. Secondary glossopharyngeal neuralgia can occur due to the compression of the glossopharyngeal nerve by vascular structures,

lesions, or intracranial tumors such as cerebellopontine angle tumors, carcinoma of the laryngeal and nasopharyngeal tumors, cranial base tumors, oropharynx and tongue tumors, calcified stylohyoid ligament, parapharyngeal abscess, intracranial vascular compression, direct carotid puncture, trauma, dental extractions, multiple sclerosis, Paget's disease, elongated styloid process (Eagle syndrome), occipital cervical malformations, and inflammatory processes, like Sjogren's syndrome [17,18,19,20].

he vast majority of patients with glossopharyngeal neuralgia are thought to have an artery compressing the nerve as it exits from the medulla and travels through the subarachnoid space to the jugular foramen [21].

The glossopharyngeal nerve is a mixed sensory motor nerve that exits the brainstem from the upper medulla. From that point, it leaves the skull through the jugular foramen along with the vagus and accessory nerves.

It continues its path between the internal jugular vein and the internal carotid artery as it descends and then continues beneath the styloid process. It then curves to make an arch on the side of the neck as it passes under the hyoglossus muscle to its final distribution of the base of the tongue, the palatine tonsil, and glands of the mouth [22].

A motor efferent supplies the stylopharyngeus muscle, which is essential in swallowing. Sensory afferents provide information from the inner surface of the tympanic membrane, the upper pharynx as well as the posterior one-third of the tongue.

Another important branch is the one to the carotid body and sinus is known as Hering's nerve. It communicates with the vagus nerve and carries information from chemoreceptors in the carotid body and baroreceptors in the carotid sinus; this is important clinically as activation of the visceral sensory branch of the glossopharyngeal neuralgia can activate the vagus nerve (tractus solitarius and dorsal motor nucleus) and produce a reflex arrhythmia [23].

This vagal activation may explain the cardiac-related syncopal episodes sometimes associated with glossopharyngeal neuralgia. Overall, the glossopharyngeal nerve is a very small nerve that runs deep in the neck, and it is sometimes resected accidentally during open neck dissections.

For this same reason, it is often called 'the neglected cranial nerve.' Any infectious, inflammatory, or compressive etiologies across the glossopharyngeal nerve's path from the end organs to the brainstem may result in hyperexcitability of the nerve and produce pain. [24,25]

Investigation

Laboratory testing includes complete blood count, erythrocyte sedimentation rate, anti-nuclear antibody, and automated serum chemistry that are done to rule out occult systemic diseases like temporal arteritis, infection, inflammation, and malignancy.

Imaging of the brain includes non-contrast MRI,[26] magnetic resonance angiography (MRA), and 3-dimensional computed tomography angiography (3D-CTA), which is useful to rule out nerve compression by a vessel [27] or any tumor or by any other bony structure or signs of demyelination.[28]

High resolution MRI and subsequent image processing with 3D constructive interference in steady state (CISS) provides precise diagnosis of potential neurovascular compression of various cranial nerves (especially vagal, glossopharyngeal, and trigeminal nerves) and hence are the latest promising tools. [29]

MRA allows visualization of the anatomical relationship between the nerves and the vessels in supraolivary fossette. Special attention should be paid to the posterior inferior cerebellar artery (PICA), the anterior inferior cerebellar artery (AICA), and their courses as these vessels often course in the supraolivary fossa, which is the site of origin of glossopharyngeal nerve.

Three radiological findings are also important for diagnosing GPN as vascular compression syndrome. They are,

1) High-origin PICA,

2) The PICA making upward loop,

3) The PICA coursing and compressing the supraolivary fossette.[30]

However, if the offending vessel is AICA, GPN is difficult to diagnose before sugery because of its normal anatomy.[31]

In patients suspected of peripheral origin GPN and responding to therapy may not be subjected to an MRI scan. Imaging of neck is done to rule out tumor of the hypo pharynx, larynx, or piriform sinus.

Panoramic radiograph should be taken to rule out Eagle's syndrome [32]

An electrocardiogram (ECG) should be done (during pain attack) to rule out associated cardiac arrhythmias.[33]

Management

The treatment for Glossopharyngeal Neuralgia can be pharmacological or surgical. The first line of treatment is pharmacological. Surgical options should be considered in situations of drug intolerance, inefficacy, allergies or side effects associated with medical therapy

Pharmacological Treatment

The pharmacological line of treatment for GPN includes the anticonvulsant medications such as carbamazepine, gabapentin, phenytoin, oxcarbazepine, or pregabalin [34-36].

Common analgesics are ineffective, but some antidepressants like amitriptyline can be helpful either alone or in combination with the anticonvulsant medications [37].

In the cardiovascular variant (vagoglossopharyngeal neuralgia) atropine should be used first. Administration of atropine will prevent the associated cardiac phenomena, but not the attacks of pain.

Continued administration of carbamazepine may cure both neuralgia and cardiac symptoms. A polypharmacy approach described by Singh et al. [38] advocates combining extra-oral glossopharyngeal nerve block together with standard oral medical therapy such as antidepressants, opioids, antiepileptics, steroids, and membrane-stabilizing agents.

The nerve blocks can be performed with either non-neurolytic agents (local anesthetic agents) with or without additives (steroid, ketamine, etc.) or neurolytic agents (phenol, alcohol, and glycerol) [39-41].

In the case of Eagle's syndrome the extra-oral glossopharyngeal nerve block targets the styloid process and injections are done just posterior to it.

Carbamazepine: Starting dose 200 mg/day in a single dose (extended-release), or two divided doses (immediate-release tablet) or in four divided doses (oral solution). Increase the dose gradually with increments of 200 mg/day as needed.

If the dose exceeds 200mg per day, it is advisable to administer extended-release capsules in two divided doses. Maintenance dose: 400 to 800 mg daily in two divided doses (immediate-release tablet) forms) or four divided doses (oral solution); maximum dose: no be more than 1,200 mg/day.

The other neuropathic pain medicines recommended by the International Association for the Study of Pain (IASP) are as below:

- Gabapentin (100 to 5000mg/day in 1 to 4 divided doses),
- Duloxetine (20 to 90mg/day),
- Valproic acid (125-2500mg/day in 1 to 2 divided doses),
- Clonazepam (0.5-8mg/day),
- Lamotrigine (50 to 500mg/day in 1 to 2 divided doses),
- Baclofen (10 to 80mg/day in 1 to 4 divided doses),
- Phenytoin (200 to 600mg/day in 1 to 3 divided doses),
- Pregabalin (75 to 500mg/day in 1 to 2 divided doses) and
- Topiramate (50 to 1000mg/day in 1 to 2 divided doses)

As a general rule, these medications should be started at low doses and titrated up as needed based on their effectiveness, tolerability, and side effects. This pain condition often shows a relapsing-remitting course, and so medication can be tapered down to a low maintenance dose. Combining two or more medications with different mechanisms of action can help achieve better pain relief while avoiding side effects. A short course of opioids can be useful for intractable pain.

Adjuvant care: Cold and hot compresses, physical therapy, and psychological counseling are all options in addition to medical therapy. The success rate is variable but can be helpful.[42]

Interventional Pain Management Techniques

Glossopharyngeal nerve blocks merit consideration for both diagnostic and therapeutic purposes. This block can be an option in conjunction with pharmacotherapy. A diagnostic block with a local anesthetic should be tried first to confirm the origin of the pain. If diagnostic blocks are successful, chemical neurolysis or thermal radiofrequency ablation can be performed on the nerve.

Chemical neurolytic agents such as alcohol, glycerol, or phenol are typical choices. Radiofrequency ablation is typically performed at the jugular foramen to target the inferior petrous ganglion of Andersch. Accurate needle placement is critical as life-threatening bradycardia and hypotension can occur if the vagus nerve gets stimulated during the procedure.

There are two common approaches to block the glossopharyngeal nerve: the intraoral and extra-oral approaches. The extra-oral technique is preferred since it is safe and easy to perform. Complications are not uncommon with glossopharyngeal neuralgia blocks. The glossopharyngeal nerve is in the vicinity of the internal jugular vein, and the carotid artery and intravascular injection can easily occur.

The concomitant block of the recurrent laryngeal nerve may cause hoarseness of the voice. Always avoid bilateral glossopharyngeal nerve blocks at the same time to avoid complete vocal cord paralysis. Blockade of the vagus nerve may result in tachycardia and hypertension via blockade of parasympathetic fibers. [43,44,45]

Surgical Management of Glossopharyngeal Neuralgia

Several surgical modalities are used for the treatment of glossopharyngeal neuralgia based on the etiology of the pain. The compression of the glossopharyngeal nerve by a vascular structure is the most common cause of secondary glossopharyngeal neuralgia.

Microvascular decompression (MVD) of the glossopharyngeal nerve is the most widely used surgical modality to correct vascular compression of the nerve. Alternatively, a resection of the glossopharyngeal nerve alone or with branches of the vagus nerve can also be performed.

Extracranial techniques are percutaneous radiofrequency rhizotomy and direct surgical resection. These techniques are ideal in patients with essential glossopharyngeal neuralgia who failed medical management but cannot tolerate an open intracranial resection.

Resection of the ipsilateral styloid process, also known as stylectomy, is a therapeutic option for Eagle syndrome. The physician must rule out other central causes of glossopharyngeal neuralgia before pursuing this surgery.

Intracranial techniques include rhizotomy or an intracranial root resection of the glossopharyngeal nerve and/or vagus nerves from its origin in the brainstem at the cerebellopontine angle.

Persistent dysphagia and hoarseness of voice are the most common complications of these surgeries. Stereotactic radiosurgery with gamma knife surgery provides a less-invasive option, but data on safety and efficacy is limited. [46-48]

Differential diagnosis

- **Trigeminal neuralgia:** Glossopharyngeal neuralgia is most commonly mistaken for trigeminal neuralgia since both have similar pain characteristics. Additionally, both of them are cranial neuralgias, with similar pathophysiology and medical management. In certain rare cases, these conditions can coexist. Since the incidence of glossopharyngeal neuralgia is 1/1000 times less than trigeminal neuralgia, underdiagnoses of glossopharyngeal neuralgia are very common; this is why a thorough history and physical examination are essential since they both differ in the location of pain and provoking factors. Pain from glossopharyngeal neuralgia occurs in the throat and tonsillar region and is exacerbated by swallowing and chewing movement, whereas pain from trigeminal neuralgia occurs on the face in the trigeminal nerve distribution and is exacerbated by a light touch on the face, washing the face and brushing the teeth.
- **Jacobson's neuralgia:** When the only symptom of glossopharyngeal neuralgia is the sensory loss at the ear (the otic form of glossopharyngeal neuralgia), it gets confused with intermedius or Jacobson neuralgia.
- **Temporal arteritis and temporomandibular joint dysfunction**: Pain in these pathologies occurs in the same distribution, but the pain characteristics in both pathologies are entirely different from glossopharyngeal neuralgia.[49]

Prognosis

Glossopharyngeal neuralgia has a variable prognosis based on the patient's symptoms. Most of the patients have a single episode of painful paroxysmal attacks. The annual recurrence rate is as low as 3.6%. Only 25% of patients require surgery, and the rest are manageable medically. Less than one in four patients will have bilateral pain.

Bilateral pain, multiple bouts of severe excruciating pain, and constant pain are poor prognostic indicators.[50]

Complications

Syncope and cardiac dysrhythmias: When the glossopharyngeal nerve gets irritated, it sends feedback via the dorsal motor nucleus of the Xth nerve. These signals also stimulate the nucleus tractus solitarius in the midbrain.

Thus, during the acute glossopharyngeal neuralgia attack, abnormal stimulation produces amplified vagal response, resulting in bradycardia, hypotension, and cardiac dysrhythmias. These autonomic changes cause cerebral hypoperfusion, slow waves on EEG, seizures, and syncope.

Convulsive movements, limb clonus, automatic smacking movements of the lips, and upward turning of the eyes are signs of cerebral hypoxia.

The cardiovascular complications occur during the painful episodes or immediately after the pain symptoms resolve. Management of glossopharyngeal neuralgia pain attacks with drugs and/or surgical treatment can help manage these complications.

Some patients only develop cardiovascular manifestations of glossopharyngeal neuralgia without painful paroxysms, also known as non-neurologic glossopharyngeal neuralgia.

These patients can receive therapy with glossopharyngeal nerve avulsion or microvascular decompression.[51]

Glossopharyngeal neuralgia is a rare but complex pain syndrome and requires a multidimensional management approach. Management of glossopharyngeal neuralgia requires expertise from the fields of neurology, otorhinolaryngology, interventional pain, and neurosurgery.[52]

No clinical trials demonstrate the efficacy of the different groups of medications used in the medical management of glossopharyngeal neuralgia. Since glossopharyngeal neuralgia is a type of neuropathic pain, it would be beneficial to present levels of evidence of medicines used in the management of glossopharyngeal neuralgia. Gabapentin and pregabalin, selective serotonin receptor inhibitors, carbamazepine, and tricyclic antidepressants are first-line treatments.

The primary care provider and nurse practitioner should educate the patient on the potential side effects of these drugs. Second-line treatment includes opioids, certain anti-seizure medicines (like lamotrigine), topical capsaicin, and N-methyl-D-aspartate receptor antagonists.

These classes of drugs have shown the best evidence with clinical relevant effects without any superiority from one over another. One metanalysis study reported that despite an increase of published trials by 66%, there is only a limited improvement in the medical management of neuropathic pain.[53]

Conclusion

Although glossopharyngeal neuralgia is a very rare facial pain syndrome, it is an important neurological disease because of the extreme suffering, and in some instances, life-threatening issues due to cardiac arrhythmia.

Glossopharyngeal neuralgia could be misdiagnosed because of its similarities to much more common trigeminal neuralgia, as well as much less frequent superior laryngeal and nervus intermedius cranial neuralgias. Pain specialists should be trained to differentiate them even if the pharmacological treatment is the same, because of the differences in surgical approaches.

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