Review Article (Systematic review of a subject)

An uncommonly common: Glossopharyngeal neuralgia

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Abstract

Glossopharyngeal neuralgia is a relatively rare condition characterized by severe, paroxysmal episodes of pain localized to the external ear canal, the base of the tongue, the tonsil or the area beneath the angle of the jaw. This pain is many a times confused with Trigeminal Neuralgia and mistreated. There are various diagnostic and management dilemmas which are herein addressed in this review.

Key Words

Glossopharyngeal neuralgia, glossopharyngeal neuralgia, stylalgia

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Introduction

Neuropathic pain is defined as non-nociceptive pain or pain that is not related to activation of pain receptor cells in any part of the body. It is a type of pain caused by a lesion or disease of the somatosensory system.[1]

Glossopharyngeal neuralgia (GPN) is a condition causing throat, ear, and neck pain. The International Association for the Study of Pain (IASP) defines it as sudden, severe, brief, recurrent pain in the anatomical distribution of the glossopharyngeal nerve.[2] Classically, it is described as a severe transient stabbing pain experienced in the ear, base of the tongue, tonsilar fossa, or beneath the angle of the jaw. However, the location of pain can have significantly varied distribution and overlap amongst the nerves supplying the face (trigeminal, vagal, facial).[3] The unusual presentations are cardiac arrhythmias associated with pain episodes, fear to eat (which may be the precipitating cause for pain episode), and syncope.[4]

It must be emphasized that GPN is not as uncommon as reported in the literature due to difficulties in diagnosis, unawareness of the disease and more so with increasing number of patients with styalgia (pain due to elongated styloid process). It is often compared with trigeminal neuralgia in presentation and incidence due to significant overlap of symptoms and thus causing a diagnostic dilemma.[3]

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Historical Aspects

In 1910, Weisenburg first described GPN as a cause of Tic douloureux when a patient presented to him with lancinating pain of the throat and the ear.[5] In 1921, Harris coined the term "glossopharyngeal neuralgia"[6] describing it as a painful syndrome characterized by paroxysms of unilateral and severe lancinating pain in the distribution of the nerve, which may be elicited by stimulation of trigger points in regions of the nerve. The pain may be spontaneous or precipitated by a variety of actions that stimulate the region supplied by the glossopharyngeal nerve namely yawning, coughing, swallowing, and talking. In 1933, Reichert[7] recognized the tympanic branch (Jacobson's Nerve) of glossopharyngeal nerve as a cause of ear pain in GPN. Wortis *et al*. (1942) first described GPN in association with cardiac arrest and syncope that are unusual presentations of GPN.[4,7]

Epidemiology

Because of multiple isolated reports and no population data available, it is difficult to report a specific incidence in various parts of the world. Katusic and colleagues published a 39-year retrospective study from 1945 until 1984[8] on the population of Rochester (USA). It was found that the incidence of GPN in this population was 0.7/100,000 population/year (0.9 and 0.5 in men and in women, respectively). In addition, 25% had bilateral symptoms. They concluded that GPN was generally a mild disease, since mild attacks were not uncommon, with only 3.6% sufferers having a second annual recurrence. Only 25% underwent surgery for symptomatic relief.

Rushton and colleagues published a study (1981) that examined GPN patients at the Mayo Clinic from 1922 to 1977. The authors reviewed 217 cases, of which 57% were over 50-years of age and 43% were between the ages of 18 and 50 years. Hundred sixty one patients had spontaneous remissions, 37 experienced no relief, and 12% had bilateral pain.[9] There are isolated reports of syncope associated with GPN[10,11]; hence its incidence can't be quoted. GPN can be associated with trigeminal neuralgia as seen in a study by Sinay *et al*.[12] Kondo and colleagues (1998) in a study concluded that GPN is rare, seen 100-times less often than trigeminal neuralgia.[13] It occurs more commonly in patients older than 50 years but can occur at any age.

Patel and colleagues published a 20 yrs retrospective study of over 200 patients of Pittsburgh, Pennsylvania who underwent micro vascular decompression (MVD). They found that 66.8% were female and 33.2% were male. Mean age was 50.2 years, with a mean duration of pain of 5.7 years. The most common symptoms were throat and ear pain or throat pain alone.[14] Data about its seasonal recurrence are conflicting.[15] There was no side predilection, 54.8% of patients had left-sided symptoms, and 45.2% had right-sided symptoms.

In summary, GPN is relatively rare when compared to trigeminal neuralgia and is usually unilateral painful condition that tends to be more common in middle-aged females.

Anatomical Aspects

Glossopharyngeal nerve is a mixed cranial nerve with both sensory and motor components. It receives somatic sensory fibers from the oropharynx, posterior third of the tongue, eustachian tube, middle ear, and mastoid. The sensory supply to the middle ear and mastoid passes along the tympanic branch or Jacobson's nerve. The glossopharyngeal nerve also receives special sensory fibers for taste in the posterior third of the tongue as well as chemoreceptor and baroreceptor afferent inputs from the carotid body and carotid sinuses respectively.[16] The motor component supplies the striated muscle stylopharyngeus and secretomotor parasympathetic fibers to the parotid gland. The other important branch is the carotid sinus nerve (Nerve of Hering) that supplies the carotid body and carotid sinus. It conveys chemoreceptor and stretch baroreceptor information centrally for respiratory, circulatory reflex function and may be responsible for arrhythmogenicity of GPN.

Clinical presentation

The characteristics of GPN are similar to trigeminal neuralgia with some differences, which must be identified for the correct diagnosis and treatment.

Clusters of unilateral attacks of sharp, stabbing, and shooting pain localized in the throat radiating to the ear or vice versa are characteristic of GPN. The distribution of pain is diagnostic: The pain shoots from the pharynx, tonsil, and posterior tongue base upwards to the eustachian tube and inner ear or to the mandibular angle [Table 1].[15]

The pain of GPN is subtle in onset with a mean duration of 30 sec. It is excruciating and can recur after a brief period without pain, but an ache may persist in the same region.[16] Many crises can reoccur over days, weeks, or months,[17] and usually the attacks occur during the day. Swallowing is the most common trigger factor, and cold liquids seem especially to induce pain. Chewing, talking, sneezing, cleaning the throat, and touching the gums or oral mucosa, even sudden movements of the head, raising the arm on the side of the pain, and the lateral movement of the jaw may also trigger the paroxysms. Several patients found that touching the external auditory canal, the side of the neck, and the skin anterior to the ear triggered the pain on the same side.[18]

Table 1: International Headache society diagnostic criteria for classical glossopharyngeal neuralgia

Paroxysmal attacks of facial pain lasting from a fraction of a second to 2 min and fulfilling criteria B and C
Pain has all of the following characteristics:
Unilateral location
Distribution within the posterior part of the tongue, tonsillar fossa, pharynx or beneath the angle of the lower jaw and/or in the ear
Sharp, stabbing and severe
Precipitated by swallowing, chewing, talking, coughing, and/or yawning
Attacks are stereotyped in the individual patient
There is no clinically evident neurological deficit
Not attributed to another disorder[1]
The International Headache Society has divided glossopharyngeal neuralgia into the classical and symptomatic type. In the symptomatic form, the description is as in the classical type with the proviso that aching pain may persist between paroxysms, and sensory impairment may be found in the distribution of the glossopharyngeal nerve.

[1]Other causes have been ruled out by history, physical examination, and/or special investigations.

The trigger zone is recognized late as compared to trigeminal neuralgia; therefore, it may not be found during initial clinical examination.[19] Some patients can have the pain triggered by sweet, acid, cold or hot food.[20] Other rare features are tinnitus, vomiting, vertigo, swelling sensation, and involuntary movements.[7] GPN can sometimes be confused with intermedius neuralgia when the only symptom is sensory loss at the ear (Jacobson's neuralgia). Temporal arthritis can have a similar pain. Asystole, convulsions, and syncope are associated with GPN in many patients described in the literature, and this condition is called vagoglossopharyngeal neuralgia (VN).[7,21] These reactions occur due to the complex anatomical relationship between the intermedius, vagus, and glossopharyngeal nerves leading to difficulties during neurosurgical assessment.[22]

Most of the glossopharyngeal neuralgia is idiopathic, but they may be associated with cerebellopontine angle masses, oropharyngeal tumors, arachnoiditis, stylohyoid ligament ossification, multiple sclerosis,[23] and vascular malformation.[24] GPN can be associated with trigeminal neuralgia[25,26] or be a part of combined hyperactive dysfunction syndrome[27] or be associated with Chiari type I malformation.[28]

Life threatening complications of GPN

Harris *et al*. (1921)[6] reported that GPN could be associated

with cardiac dysrhythmia and instability. This relationship is well-accepted and has been documented by many authors. The various reports and case studies have been compiled and summarized by Ferrante *et al*.[29] Intense irritability and hyper-stimulation of glossopharyngeal nerve feedback onto the nucleus of the tractus solitarius of the midbrain and via collaterals reach the dorsal motor nucleus of the vagus nerve. This activation of this abnormal loop during severe neuralgic pain would be responsible for heightened vagal response as cardiac dysrhythmia, bradycardia, and hypotension, with cerebral hypoxia, slowing of EEG activity, syncope, and convulsions. Convulsive movements, limb clonus, automatic smacking movements of the lips, and upward turning of the eyes are signs of cerebral hypoxia induced by the bradycardia. [10,11,30,31] The cardiovascular phenomenon is seen during the pain attack or immediately following it. Both pharmacotherapy and surgical treatment eliminates these. There is a subset of patients with demonstrable cardiac manifestations without typical neuralgic symptoms who have responded very well to glossopharyngeal nerve avulsion or MVD. Such syndromes have been called non-neuralgic GPN,[32] in recognition of the fact that glossopharyngeal nerve irritability may not always give rise to a pain.

Types

There have been multiple attempts to classify GPN on different basis. The various ways the disease has been classified are:

Anatomical Area Involved[7]

Otitic type - Pain in and around the ear

This is commoner form of the two in the anatomical classification. The pain is often described in relation to the ear. The pain can be of any type, ranging from burning, sharp shooting, shock-like, pressure, pinprick etc.

Oropharyngeal – Pain is in and around throat and face region

This form has more varied distribution, and significant overlap may occur with other cranial nerve distribution areas.

The International Headache Society (IHS) Classification of GPN[33]

The basis of classification is that pain occurs as episodic or constant basal pain that persists between the episodes of peaks and troughs of pain.

The types proposed by IHS are

- Classical GPN- episodic pain
- Symptomatic GPN- continuous pain, commoner

Cause-based Classification

Idiopathic type

No demonstrable lesion is found in these cases. Most often, these are attributed to nerve ganglion compression by vessel or by compression of glossopharyngeal nerve as it exits or enters the brainstem. This is supported by the fact that microvascular decompression (MVD) eliminates GPN symptomatology. Most of the cases belong to this type of GPN.

Secondary type (Symptomatic):

In this, a demonstrable lesion can be found, which includes trauma, neoplasm, infection, vascular malformation, or elongated styloid process [Table 2]. Secondary nature of GPN is suspected when there are neurological deficits, like numbness in the distribution of glossopharyngeal nerve, absence of symptom-free interval in between the attacks, and pain distribution different from glossopharyngeal nerve area.[34]

Diagnosis

The diagnosis of GPN is strictly clinical as no imaging findings or other testing can reliably link to the syndrome. The first priority is to ascertain the diagnosis of neuralgia and exclude other causes of pain due to inflammation and neoplasia. A step-wise logistic approach to diagnose GPN is summarized in flowchart of diagnosis [Figure 1]. The description of pain will help. Neuralgic pain is severe, episodic, and lancinating and of short duration, which may be associated with intervening periods of a low-grade dull ache. In contrast, inflammatory or neoplastic pain is more constant, of longer duration, and of deep-seated boring quality. Next, the distribution of the pain has to be mapped out. This is important for two reasons, firstly, there is a need to know if the neuralgic pain is typically glossopharyngeal or it involves the other cranial nerves like the trigeminal nerve or nervus intermedius.

In any pain with typical glossopharyngeal distribution, ascertain its predominant distribution: Tympanic or oropharyngeal. It is important to determine the site of any trigger points; check if the trigger point is in the oropharyngeal area or is in the ear? Is the neuralgic pain precipitated by oral activities e.g. swallowing, talking, yawning, or it is brought about by hearing activities e.g. pain on exposure to loud sounds. Are there any otologic symptoms? By evaluating the patient on above lines, GPN can be differentiated into classical/symptomatic and otitic/non-otitic type.

If the patient does not have pain at that point of time, but anticipates that he would have it later in the day, the trigger point may be injected with lignocaine 2% or bupivacaine 0.5%

Table 2: Cause based classification of glossopharyngeal neuralgia

Idiopathic (Essential) form	Vascular compression
	Central pontine dysfunction
Secondary (Symptomatic)	Trauma-Skull base fractures, penetrating injury, post-radiation
	Neoplasms-Skull base, cerebellopontine, brainstem, pharynx, tongue, tonsils, metastatic head, and neck tumors
	Infections-Tonsillitis, pharyngitis, petrositis, arachnoiditis, Para pharyngeal abscess, tuberculosis
	Surgery-Post-tonsillectomy, post-neck dissection, post-craniotomy
	Vascular malformations-Arteriovenous malformations, fusiform aneurisms, persistent hypoglossal artery, dissection of vertebral artery
	Demyelination-Multiple sclerosis
	Elongated styloid process-Eagle's syndrome
	Miscelleneous: Direct carotid puncture, Chiari I malformation, choroid plexus overgrowth, hyperactive dysfunction syndrome

to see if it can avert another attack of pain. If the symptoms are primarily otologic, inject lignocaine 2% or bupivacaine 0.5% into the external auditory meatus to see if it abolishes the pain that is present at that time or it will avert a subsequent attack. Lastly, determine whether it is idiopathic type or there is a secondary cause. Differential diagnosis of secondary GPN [Table 2] should be checked for. The most important cause of secondary GPN is Eagle's syndrome due to either an elongated styloid process or calcification of the stylohyoid ligament. In history, look for: trauma, radiation, surgery, inflammation, and demyelination. Pathology related to base of the skull, head and neck, nasopharynx,[35] and teeth[36,37] etc. can be the culprit of secondary GPN.

Investigations

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,[38] magnetic resonance angiography (MRA), and 3-dimensional computed tomography angiography (3D-CTA), which is useful to rule out nerve compression by a vessel[39] or any tumor or by any other bony structure or signs of demyelination.[40] 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.[41] 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.[42] However, if the offending vessel is AICA, GPN is difficult to diagnose before sugery because of its normal anatomy.[43] 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[44] [Figure 2]. An electrocardiogram (ECG) should be done (during pain attack) to rule out associated cardiac arrhythmias.[45]

Treatment

Treatment for GPN can be non-surgical or surgical. The treatment protocol has been represented as a flowchart in Figure 3.

Non-Surgical Treatment

Pharmacotherapy for GPN

Pharmacotherapy forms the first line of treatment for GPN. The medications of choice are carbamazepine, gabapentin, and pregabalin although theoretically any membrane stabilizer can be used.[47] In addition, low doses of selective serotonin reuptake inhibitors (SSRI) and vitamin B12 can be used. Baseline complete blood count, blood chemistry, and urinalysis are obtained before initiation of carbamazepine that forms the first line and gabapentin that is reasonable alternative for the treatment of GPN.

The use of NSAIDS is not routinely recommended for treating neuralgic pain.[48] There are isolated reports of neuralgia responsive to NSAIDS and opioids. It is postulated that neuralgia responsive to NSAIDS is more likely to be due to some unknown acute inflammation. Opioids have been used as an adjuvant to the frontline neurogenic agents with limited success.[49] The IASP recommends the following drugs and doses for treating facial neuralgias [Table 3]. However, these medications have to be titrated to effective levels, and gradual tolerance may develop with their prolonged use. Most often, the disease shows relapsing and remitting pattern, with an

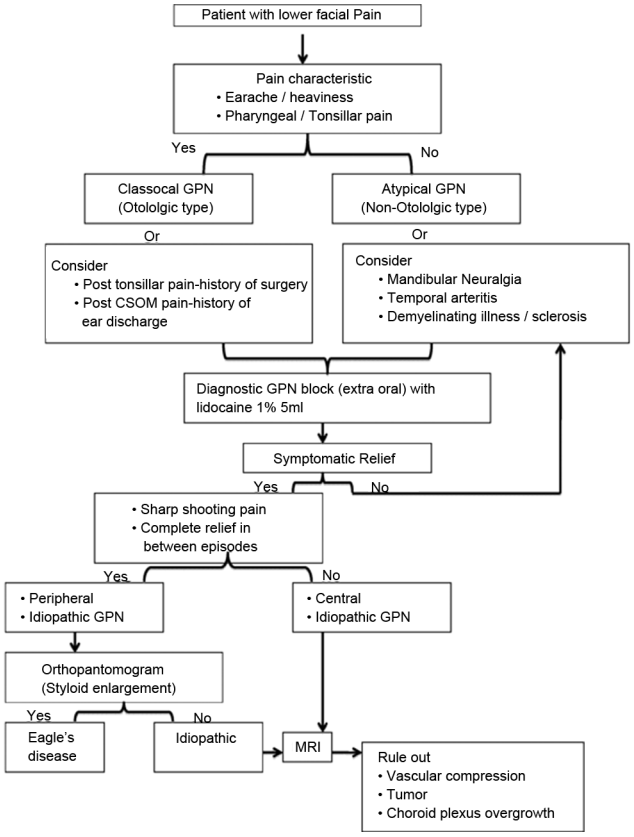

Figure 1: A stepwise logistic approach for diagnosing glossopharyngeal neuralgia

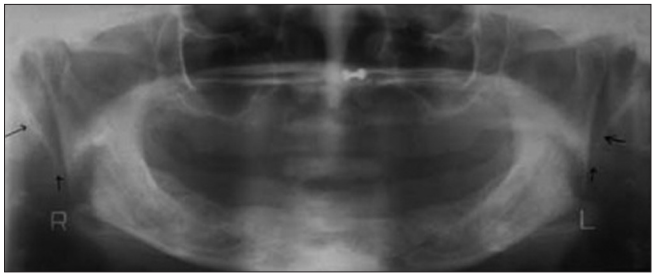

Figure 2: Orthopantomogram showing bilateral elongation of styloid process[46]

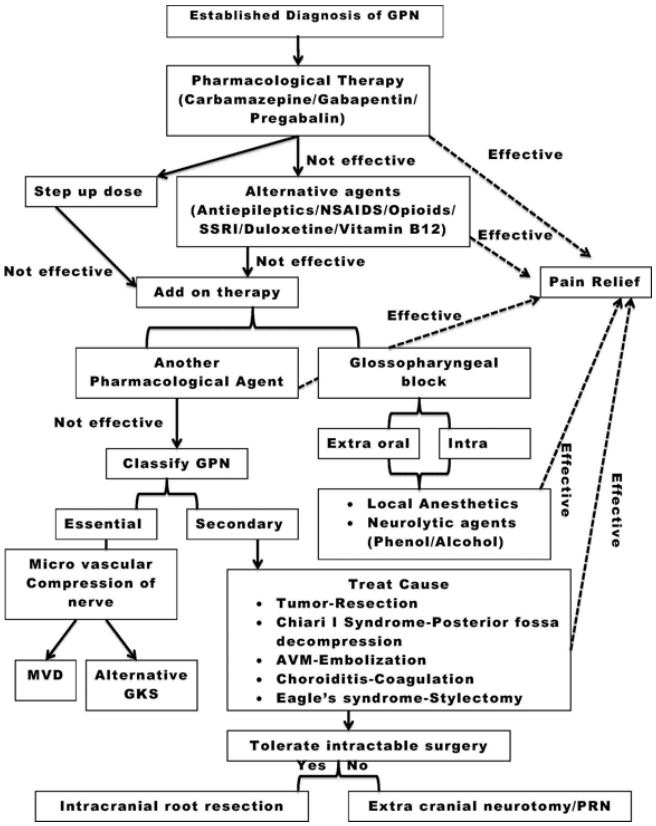

Figure 3: A stepwise logistic approach for the treatment of glossopharyngeal neuralgia

Table 3: The International Association for the Study of Pain (IASP) recommended drugswith their dosages used in glossopharyngeal neuralgia

Drug	Dose (mg/day)
Carbamazepine	100-2000
Gabapentin	100-5000
Duloxetine	20-90
Valproic acid	125-2500
Clonazepam	0.5-8
Lamotrigine	50-500
Baclofen	10-80
Phenytoin	200-600
Pregabalin	75-500
Topiramate	50-1000

acceptable pain relief in around 2 months. These medications can be gradually tapered down to achieve much lower maintenance doses. These remissions can last from months to years. On a relapse, restepping up of dosages can be tried. If, however, adequate pain relief is not achieved, a different agent can be tried. One must have a lower threshold for stylectomy in bilateral styalgia. A large number of patients with neuropathic pain are currently treated with either two or multiple agents in combination. There are limited studies on pharmacologic and non-pharmacologic treatments combinations, hence an additional benefit by add-on physical therapy or psychological treatment is doubtful.[50] The target of medical therapy should be to achieve pain relief, leading to minimal affliction of

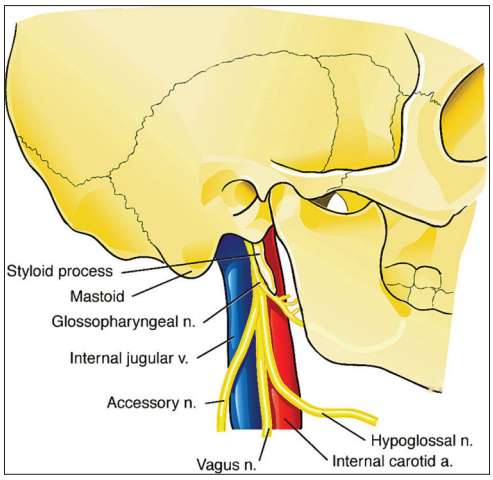

Figure 4: Landmark evaluation for glossopharyngeal nerve block in glossopharyngeal neuralgia

daily activities. The bad prognostic signs are – bilateral GPN, constant pain, or multiple daily bouts of pain.

Glossopharyngeal nerve blocks

Glossopharyngeal nerve block can be used for the evaluation of atypical facial pain, treatment of GPN, and intractable pain caused by pharyngeal cancer.[51] These nerve blocks are excellent adjunct to the pharmacologic treatment of GPN, ensuring rapid palliation of pain. They can be performed with either non-neurolytic agents (local anesthetic agents) with or without additives (steroid, ketamine, etc.) or with neurolytic agents (phenol, alcohol, glycerol, etc.) LA blocks are used for both, diagnostic and therapeutic purpose; thus establishing the diagnosis of GPN. A diagnostic block is given as one of the first interventions to label any lower facial pain simulating GPN as true GPN. Neurolytic agents are safe alternative to more invasive procedures.

The various approaches used to block glossopharyngeal nerve are[52]:

Intra-oral approach: This block is given using a distally bent spinal needle (approximately 25 degrees) up to a depth of 0.5 cm through the mucosa at the lower lateral portion of the posterior tonsillar pillar.[53]

Extra-oral approach: This block is given at the midpoint of on an imaginary line, running from the mastoid process to the angle of the mandible, at a depth of up to 3 cm. The nerve lies immediately below the styloid process at this point [Figure 4]. This technique is simpler to perform and is more comfortable to the patient.[54]

An extra-oral block is preferred for treatment of neuralgia as the intraoral block may spare the tympanic branch of glossopharyngeal nerve. Also, there is an increased likelihood of inadvertent tonsillar artery injection due to decreased dexterity in approaching the site of the block in the posterior part of the mouth in intra-oral approach.

Complications of glossopharyngeal nerve block

Intravascular injection can occur into the carotid artery or into the internal jugular vein. Difficulty in swallowing and

hoarseness can result from the glossopharyngeal and vagus (recurrent laryngeal branch) nerve blocks, respectively. Bilateral GPN block can cause bilateral vocal cord paralysis, hence bilateral block is not recommended. The loss of parasympathetic outflow with vagus nerve blockade could cause tachycardia and hypertensive response.[51]

Surgical Therapy

Once the patient becomes refractory or intolerant to medications, surgery is the next treatment option. However, surgical therapy is associated with high morbidity of the patients and is limited to younger patients. These surgical procedures for the lesions may be classified as follows:

1. Peripheral procedures:
   a. Extra cranial, such as direct surgical neurotomies or percutaneous radiofrequency thermal rhizotomy[55-57]
   b. Intracranial, such as direct section of glossopharyngeal and vagal nerves in the cerebello-pontine angle[58,59]
2. Central procedures, such as percutaneous or open trigeminal tractotomy-nucleotomy or nucleus caudalis DREZ operation

These days, the best-established surgical treatments are MVD of vascular roots[60-62] and rhizotomy of the glossopharyngeal nerve with upper vagal nerve roots.[63] In essential GPN, the primary pathology, being vascular compression of the nerve roots, responds well to MVD. However, in secondary GPN, first address the underlying pathology: Tumor resection, posterior fossa decompression in Chiari malformation, embolization of an arteriovenous malformation, coagulation of choroid plexus overgrowth, stylectomy for Eagle's Syndrome.[34] In secondary GPN, when MVD is not possible, intracranial root section is considered curative and is most widely employed. In the largest case series by Rushton *et al*.[9] and in a smaller series by Taha *et al*.,[59] there were no recurrences after preganglionic section of the ninth and upper tenth nerve roots. However, sectioning of cranial nerve fibers IX–X, open or percutaneous tractotomy-nucleotomy is followed by severe and persistent dysphonia and dysphagia. [55,60,64] This is because all neural destructive or ablative procedures carry the risk of neuritis, deafferentation pain, and neurovascular injury.[65]

With the refinements of microsurgical and anesthesiological techniques (Brainstem evoked potentials), MVD has proven to be an effective and safe available treatment and should be considered the first line treatment in drug-resistant GPN.[58] In a study by Resnic *et al*.,[66] MVD provided complete pain relief in 76% of the cases and substantial improvement in a further 16%. Sampson *et al*.[63] found pain relief of more than 10 years by MVD, hence indicating its efficacy and safety even on long term follow-up. MVD should be considered when a patient experiences typical GPN symptoms and has a PICA loop near the glossopharyngeal nerve[42] and especially in patients with isolated symptom of throat pain.[14]

Extracranial neurotomy and percutaneous radiofrequency rhizotomy are restricted to those patients who have failed medical therapy and cannot tolerate an open intracranial procedure. Stylectomy done for elongated styloid process has been promising, once the central causes of GPN have been ruled out[67,68] and associated styloid enlargement is diagnosed.

Recently, various case reports have been published, which have shown beneficial effects of pulsed radiofrequency neurolysis (PRN) and gamma knife surgery (GKS). PRN is a non-destructive neuromodulatory method to treat both, idiopathic and secondary GPN.[69,70] Short pulses of radiofrequency energy, delivered at a constant temperature, produce central and peripheral neuromodulatory effects.[71,72] In GKS system, an 80 Gy dose is stereotactically directed to the isocenter with MR imaging-based target localization and 4-mm collimation.[73,74] It might serve as a potential alternative to other percutaneous techniques and surgical options for patients with secondary GPN. Stereotactic radiosurgery (SRS) with GKS system offers a less-invasive option for patients with GPN. Till date, Pollock and Boes have reported the largest series of patients (5 patients), with suspected GPN being treated with SRS directed at the glossopharyngeal and vagus nerves, within the jugular foramen with a failure rate of 40%.[74] These new techniques offer a promising direction that might spare patients from pain and potential morbidity of surgery.

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