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UNCOMMON HEADACHE SYNDROMES (J AILANI, SECTION EDITOR)

Glossopharyngeal Neuralgia

Andrew Blumenfeld · Galina Nikolskaya

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Abstract In this review, the clinical characteristics, differentiating features from other forms of neuralgia, etiology and treatment options of glossopharyngeal neuralgia will be discussed.

Keywords Glossopharyngeal neuralgia · Etiology · Treatment options · Facial pain · Vagoglossopharyngeal neuralgia

Introduction

Glossopharyngeal neuralgia (GPN) is a rare facial pain syndrome characterized by paroxysms of excruciating pain in the sensory distribution of the auricular and pharyngeal branches of glossopharyngeal (IX) and vagus (X) cranial nerves [1, 2••, 3••]. Usually, GNP manifests with a deep stabbing pain on one side of the throat near the tonsillar area, sometimes radiating into the ear. In some patients, GNP can be associated with bradycardia, asystole, convulsions and even life-threatening syncopal episodes—if these features are present, the condition is termed vagoglossopharyngeal neuralgia (VN) [4•, 5–7]. Glossopharyngeal neuralgia only represents 0.2–1.3 % of the facial pain syndromes [8]; however, it is often misdiagnosed as the more common Trigeminal neuralgia (TN).

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A. Blumenfeld (✉)
The Headache Center of Southern California, Encinitas, CA 92024, USA
e-mail: Blumenfeld@neurocenter.com

G. Nikolskaya
Neurology Resident UCSD, San Diego, CA 92122, USA
e-mail: gnikolskaya@ucsd.edu

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 [9]. 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 [10]. 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 [11]. Singleton in 1926 [12] and Dandy in 1927 described cases that were cured by intracranial sectioning of the glossopharyngeal nerve [13]. 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 [14]. Thereafter, percutaneous surgical techniques used for trigeminal neuralgia treatment were also performed for GPN, including pulsed mode radiofrequency [15] and gamma knife radio surgery [16]. The concurrence of the GPN and TN was first noted in 1931 by Hesse [17], and in 1935 by Peet [18•], who reported on 14 cases, including five that had a combination of GPN and TN. In 1942, Riley et al. [19] first described the rare association of GNP with life-threatening cardiac syncope identified by brief episodes of bradycardia, asystole, and hypotension—the condition was termed Vagoglossopharyngeal neuralgia [20]. Although analgesic action from an anti-epileptic drugs was first

reported in 1942, pharmacological management only started about 30 years later, when drugs such as phenytoin and carbamazepine [21, 22] were first considered for GPN treatment.

Definition of Glossopharyngeal Neuralgia per International Headache Society Guidelines (IHS)

http://www.ihs-classification.org/en/

GPN is described by the International Headache Society (IHS) as a severe transient stabbing pain experienced in the ear, base of the tongue, tonsillar fossa or beneath the angle of the jaw [23]. The pain is felt in the distributions of the auricular and pharyngeal branches of the vagus nerve, as well as of the glossopharyngeal nerve. The IHS has divided glossopharyngeal neuralgia into the classical and symptomatic types. In the classic type, the pain is only intermittent with no underlying cause or associated neurological deficit (Table 1). The symptomatic type includes the same characteristics of the classic form; however, the aching pain can persist between neuralgic episodes, and sensory impairment can be found in the distribution of the above nerves due to structural lesions (Table 2). This classification does not take associated syncopal events (vagoglossopharyngeal condition) into consideration.

Symptoms

GPN is a syndrome characterized by clusters of attacks of pain. This has been characterized as sharp, stabbing, shooting, lancinating flashes of excruciating to agonizing "electrical shock-like" or "needle-like" pain. This is usually located at the posterior region of the tongue, tonsils, oropharynx, larynx, auditory canal, middle ear, angle of the mandible, and sometimes the retro-molar region [24, 25•, 26, 27]. Pain in the throat can radiate to the ear or vice versa [2••, 28•]. This pain may be elicited by stimulating trigger points in the area of cutaneous distribution of the glossopharyngeal nerve [29]. GPN can have a sudden, abrupt onset, characterized by paroxysms of unilateral pain along the path of the nerve, more frequently on the left than the right [2••]. Pain nearly always remains on one side, but exceptionally may switch to the other side [30], and in rare cases it might be bilateral [20, 31•].

Clinical Appearance

GPN has been divided into two clinical types, based on the distribution of pain: the tympanic (affects the ear) and the oropharyngeal (affects the oropharyngeal area) [26]. Sometimes it is difficult for patients with GPN to identify the triggering zones, as they might be present in deep structures of the mouth, pharynx, and ear. GPN occurs only in adults, with a predilection for females and for patients over the age of 50 [27]. GPN episodes are typically brief, lasting from seconds to minutes, but longer-lasting attacks have also been described [32]. The intervals between the paroxysms range from a few minutes to a few hours. Pain-attacks mainly manifest during the day, but can also disturb sleep, awakening the patient at night. Clusters of attacks could last from weeks to months. The intervals between the clusters range from days to years and are irregular [2••]. Approximately 10 % of patients with GPN experience excessive vagal effects during an attack (vagoglossopharyngeal neuralgia), which can lead to bradycardia, hypotension, syncope, seizures or even cardiac arrest [33–40]. In rare cases GPN can present as syncope with no associated pain syndrome [42], making the diagnosis extremely difficult. During paroxysms resulting from the severe GPN pain, patients may experience pallor, followed by hypotension associated with bradycardia, which can lead to a loss of consciousness and associated tonic-clonic limb jerking movements [2••, 26, 41]. Other rare features are tinnitus, vomiting, vertigo,

Table 1 IHS Diagnostic criteria for classical glossopharyngeal neuralgia [23]

A. Paroxysmal attacks of facial pain lasting from a fraction of a second to 2 min and fulfilling criteria B and C

B. Pain has all of the following characteristics:

1. unilateral location
2. distribution within the posterior part of the tongue, tonsillar fossa, pharynx or beneath the angle of the lower jaw and/or in the ear
3. sharp, stabbing and severe
4. precipitated by swallowing, chewing, talking, coughing and/or yawning

C. Attacks are stereotyped in the individual patient

D. There is no clinically evident neurological deficit

E. Not attributed to another disorder*

*Other causes have been ruled out by history, physical examination and/or special investigations

Table 2 IHS Diagnostic criteria for symptomatic glossopharyngeal neuralgia [23]

A. Paroxysmal attacks of facial pain lasting from a fraction of a second to 2 min and fulfilling criteria B and C

B. Pain has all of the following characteristics:

1. unilateral location
2. distribution within the posterior part of the tongue, tonsillar fossa, pharynx or beneath the angle of the lower jaw and/or in the ear
3. sharp, stabbing and severe
4. precipitated by swallowing, chewing, talking, coughing and/or yawning

C. Attacks are stereotyped in the individual patient

D. A causative lesion has been demonstrated by special investigations and/or surgery

swelling sensation, involuntary movements, sensory loss of the area innervated by the glossopharyngeal nerve and autonomic phenomena such as tearing, sweating, salivation, unilateral mydriasis [5]. Pain attacks may occur spontaneously, but are usually associated with a specific triggering stimulus, such as chewing, swallowing, coughing, yawning, sneezing, clearing the throat, blowing the nose, rubbing the ear, talking, or laughing [2••, 31•]. Some patients can have the pain triggered by sweet, acid, cold or hot food [30], or even turning the head to one side [28•]. Between the attacks, patients are usually completely free from pain or dysfunction [2••]. Patients with GPN may experience pain remission for a period ranging from months to years; the remissions are irregular in nature [2••].

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 [26]. Idiopathic forms of GPN might possibly be caused by severe demyelination and axon-degeneration of IX and X cranial nerve fibers [3••]. In vagoglossopharyngeal neuralgia, arrhythmias and syncope could be associated with neuralgia, due to the nerve supplying the carotid sinus [43]. 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 [5, 9, 20, 24, 25•, 29, 44]. The 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 [45].

Anatomy of Glossopharyngeal Nerve

Detailed knowledge of glossopharyngeal nerve's anatomy and its functions helps to better understand the symptoms of GPN neuralgia as well as its triggers. See Fig. 1 for a summary of the glossopharyngeal nerve sensory and motor pathways.

The glossopharyngeal nerve exits the brainstem as numerous projections from the medulla below CN VII and the pontomedullary junction between the inferior olive and the inferior cerebellar peduncle. The nerve exits the skull through the jugular foramen. It is responsible for numerous functions including general somatic sensation, visceral sensation, brachial motor innervation and parasympathetic innervation.

CN IX provides general somatic sensation of touch, pain and temperature from the posterior one-third of the tongue, pharynx, middle ear, and the area near the external auditory meatus. This sensation is transmitted by primary sensory neurons in both the inferior and superior glossopharyngeal ganglion (both located within or below the jugular foramen).

Arising from the nucleus ambiguous in the medulla CN IX supplies motor innervation to stylopharyngeal muscle responsible for elevation of pharynx during talking and swallowing, and also contributing to the gag reflex.

CN IX preganglionic fibers arise from the inferior salivatory nucleus in the pons, leaving the glossopharyngeal nerve via tympanic nerve and then joining the lesser petrosal nerve to synapse in the otic ganglion, thus providing parasympathetic innervation to the parotid gland.

CN IX plays a role in general visceral sensory function by receiving afferent inputs from chemoreceptors and baroreceptors of the carotid body. Information is transmitted via primary sensory neurons located in the inferior glossopharyngeal ganglion traveling to the caudal nucleus solitarius of the medulla.

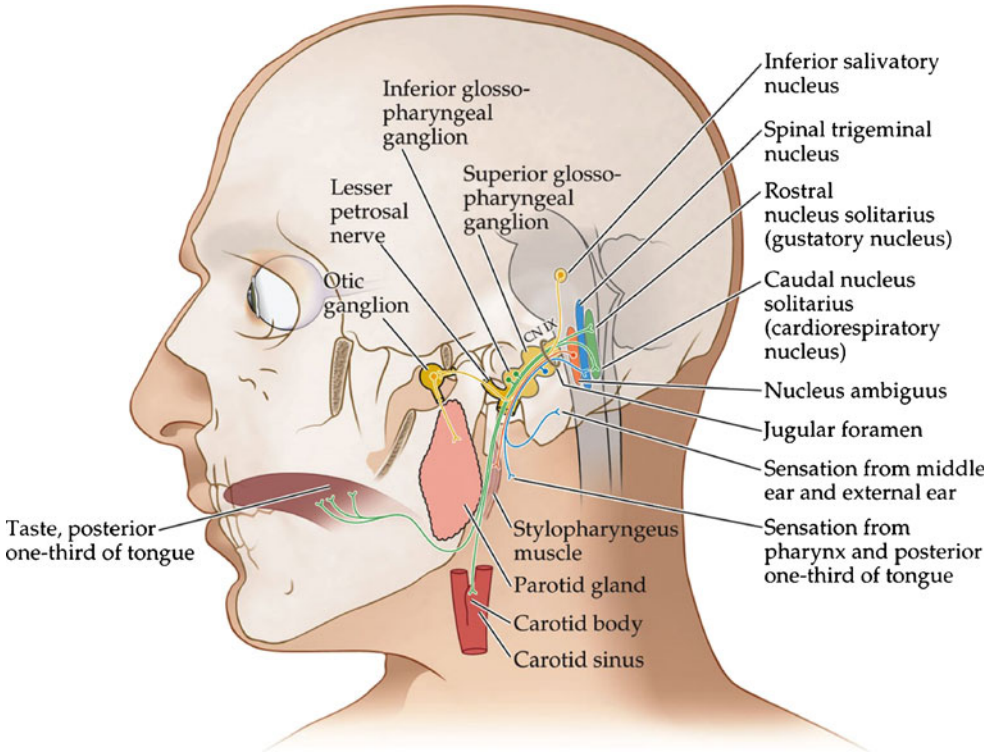

Fig. 1 Summary of the glossopharyngeal nerve sensory and motor pathways (Image from page 531 of Figure 12.20 from Blumenfeld: Neuroanatomy Through Clinical Cases, Second Edition [1]) *Permission to use the image has been granted by the editors

CN IX primary sensory neurons originating in the inferior glossopharyngeal ganglion also send special visceral sensation (taste) from the posterior one-third of tongue to the rostal nucleus solitatius (gustatory nucleus).

Diagnosis

The diagnosis of GPN must be established on comprehensive clinical grounds, and there is no specific test for this condition. Patients with a suspicion of GPN should be seen by an ear, nose and throat physician for exclusion of other diagnoses. GPN could be considered by the pattern of episodic ear and/or throat pain episodes, triggered by touching the palate or tonsil. Topical anesthesia may help to find trigger zones and radiography of the cranial base, vertebral angiography, cerebral tomography and magnetic resonance may indicate a cause for the symptoms. High resolution MRI or CT imaging of the brainstem may reveal the presence of vascular compression, tumors or demyelinating lesions involving the glossopharyngeal nerve. High resolution CT scanning of the neck can indicate the presence of an elongated styloid process, suggestive of Eagle syndrome [5, 23, 25•, 26, 27, 28•, 46, 47]. The diagnosis can be definite by the cessation of pain when this nerve is blocked at the jugular foramen or when topical anesthesia of the pharynx stops the pain [45, 48, 49]. During surgery, stimulation of the nerve may also help to confirm the diagnosis [18•].

Differential Diagnosis

The rarity of GPN and its overlap with other cranial nerve hyperactivity syndromes can lead to misdiagnosis. The majority of the differential diagnostic problems arise with:

- Trigeminal neuralgia (Vth cranial nerve)
- Superior laryngeal neuralgia (superior laryngeal branch of Xth cranial nerve)
- Nevus intermedius neuralgia (somatic sensory branch of VIIth cranial nerve)

Trigeminal Neuralgia

Glossopharyngeal neuralgia is most often misdiagnosed as Trigeminal neuralgia (TN) [25•, 27]. Both trigeminal neuralgia and glossopharyngeal neuralgia are cranial neuralgias with almost the same clinical characteristics, pathophysiology, and adjacent locations of pain. Many trigger factors are also similar. Sometimes GNP and TN even occur in a combined form, as the IXth nerve has ample connections with the mandibular division of the Vth, which may cause confusion [30, 50, 51]. The different anatomic location of the pain (V: alae nasi and upper lip or cheek, versus IX: tonsillar fossa, posterior pharynx), as well as the nature of triggering agents (V: cold breeze, touching or washing face, shaving, talking, chewing, versus IX: swallowing) should permit differentiation between them.

Table 3 Overlapping symptoms of glossopharyngeal neuralgia with trigeminal, superior laryngeal, and nevus intermedius cranial neuralgias

Overlapping symptoms of cranial neuralgias	Cranial neuralgias in which overlapping symptoms are present
Characterization of pain	Unilateral, intense, stabbing, paroxysmal in all four neuralgias
Timing of pain	Seconds to minutes in all four neuralgias
Chewing, yawning, coughing, talking as pain trigger	Present in all four neuralgias
Swallowing as pain trigger	Present in glossopharyngeal and superior laryngeal neuralgias
Throat pain	Present in superior laryngeal and glossopharyngeal neuralgias
Ear pain	Present in glossopharyngeal and nervus intermedius neuralgias

Glossopharyngeal neuralgia is more common on the left side (left: right ratio of 3:2), but TN is more common on the right (right: left ratio 5:3) [54]. Bilateral involvement (usually sequentially and not simultaneously) is more common in TN (4 %) than in GPN (2 %) [55]. The commonly known association of multiple sclerosis and trigeminal neuralgia does not occur with glossopharyngeal neuralgia [5, 8, 27].

When genuine diagnostic difficulty arises, the glossopharyngeal or trigeminal nerves can be blocked with local anesthetic as a diagnostic test [45].

Medical management of trigeminal neuropathic pains is identical to that of GPN [52, 53].

Superior Laryngeal Neuralgia

Glossopharyngeal neuralgia could be confused with superior laryngeal neuralgia [56] because of throat pain with an urge to swallow [57]. Superior laryngeal neuralgia is characterized by paroxysms of shock-like pain on the side of the thyroid cartilage, pyriform sinus, angle of the jaw, and, rarely, in the ear. Occasionally, the pain radiates into the upper thorax or up into the jaw [58]. The trigger zone is usually in the larynx; attacks are precipitated by talking, swallowing, yawning, or coughing. The combination of glossopharyngeal and vagal as well as trigeminal pain has been reported [45]. Laryngeal topical anesthesia or blockade of the superior laryngeal nerve stops the pain and is useful as a diagnostic and prognostic procedure. The pharmacologic treatment of Superior laryngeal neuralgia is identical to that of GPN.

Nervus Intermedius Neuralgia

Glossopharyngeal neuralgia sometimes is misdiagnosed as Nervus intermedius (geniculate) neuralgia [59] when the only symptom is sensory loss in the ear (Jacobson's neuralgia) [28•]. The glossopharyngeal nerve lies in close proximity to the intermedius nerve [60]. Nervus intermedius neuralgia pain is intermittent, stabbing, like electric shock, deep in the ear. The pain can be triggered by non-noxious stimulation of the ear canal or can follow swallowing or talking, but usually it is not triggered but occurs spontaneously. The patient is pain free between attacks. The syndrome is always unilateral. The medical management of Nervus intermedius neuralgia is identical to that of GPN.

With similar pain characteristic, timing of pain and in certain cases overlapping physical territory it can be challenging to distinguish between cranial neuralgias. Table 3 demonstrates the overlapping symptoms of Glossopharyngeal neuralgia with Trigeminal neuralgia, Superior laryngeal neuralgia, and Nevus intermedius cranial neuralgias. Cognizance of the presence of the coinciding features can serve as a helpful guide when categorizing facial neuralgias into different subsets based on clinical history and examination.

Treatment of Glossopharyngeal Neuralgia

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 [23, 61–63]. Common analgesics are ineffective, but some antidepressants like amitriptyline can be helpful either alone or in combination with the anticonvulsant medications [64]. 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 [2••]. A polypharmacy approach described by Singh et al. [31•] 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) [27, 42, 63, 65•, 66–68]. 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.

Surgical Treatment

The surgical procedures with the highest rates of pain relief for GPN are rhizotomy and microvascular decompression (MVD) of cranial nerves IX and X. MVD is the first-choice treatment, as it has the highest initial and long-term success rates [3••, 4•, 18•, 69, 70]; rhizotomy is safe and is a useful back-up procedure if MVD is technically difficult [3••, 4•, 14, 18•, 25•, 71–73]. If exploratory surgery does not identify an offending vessel, sectioning cranial nerve IX and the upper rootlets of cranial nerve X is an option [3••, 74]. However, this maneuver can lead to dysphagia and vocal cord paralysis [20, 75]. Vagus nerve rhizotomy is usually reserved for cases in which vascular effects are not evident [3••, 6]. The pain in Eagle syndrome can be successfully cured by resection of the elongated styloid process via a minimally-invasive approach [46, 76]. The other available techniques are: radiofrequency nerve ablation [15, 77], balloon compression [78], proton beam therapy [79], and gamma knife ablation [16, 81, 82]. Neurostimulation techniques, such as implantable high cervical spinal cord stimulation and motor cortex stimulation, may be used when other treatment methods are ineffective [29, 69, 80].

Conclusions

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.

Compliance with Ethics Guidelines

Conflict of Interest Dr. Andrew Blumenfeld reported receiving consultancies, honoraria, payment for the development of educational presentations including service on speakers' bureaus, and travel accommodations covered or reimbursed from Allergan, Posen, Pfizer, Forrest, and MAP. Dr. Blumenfeld reported receiving a grant from Allergan. Dr. Blumenfeld has a patent (planned, pending or issued) from Allergan.

Dr. Galina Nikolskaya reported no potential conflicts of interest relevant to this article.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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