International Journal of Otorhinolaryngology and Head and Neck Surgery
Swanson D et al. Int J Otorhinolaryngol Head Neck Surg. 2022 Mar;8(3):286-291
http://www.ijorl.com
pISSN 2454-5929 | eISSN 2454-5937

DOI: https://dx.doi.org/10.18203/issn.2454-5929.ijohns20220486

Review Article

An update on the diagnosis, treatment, and management of sphenopalatine neuralgia

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Received: 08 September 2021
Accepted: 12 October 2021

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ABSTRACT

This report intends to summarize the underlying pathophysiology, relevant symptoms, appropriate diagnostic workup, necessary imaging, and medical and surgical treatments of Sphenopalatine neuralgia (SN). This was done through a comprehensive literature review of peer-reviewed literature throughout the most relevant databases. Dr. Greenfield Sluder first observed that a number of his patients had atypical headaches that caused referred pain to the head and neck regions. The current understanding of the pathophysiology of SN states that irritation of the pterygopalatine ganglion secondary to inflammatory processes of the posterior ethmoid and sphenoid sinuses causes symptoms including unilateral persistent headache that begins lateral to the nose or near the eye and radiates across the face. Diagnosis is typically clinical; however, this is challenging due to lack of a definitive diagnostic criteria. Dr. Sluder originally treated his patients with 20-67% cocaine that was injected into the pterygopalatine ganglion to relieve the pain. Today, we use 88% phenol applied to the nasal mucosa. The most definitive way to both diagnoses and treat SN is the injection of cocaine or 88% phenol into the sphenopalatine region. The aim of the study was to update providers on the important clinical signs of SN and the important distinction between the clinically distinct conditions of sphenopalatine neuralgia and cluster headache. This report also outlines the treatment options to address this condition.

Keywords: Sphenopalatine neuralgia, Cluster headache, Sphenopalatine ganglion

INTRODUCTION

Background of sphenopalatine neuralgia

Sphenopalatine neuralgia (SN), formally known as Sluder's neuralgia, was first described by Dr. Greenfield Sluder in 1908. Dr. Sluder became aware that a number of his patients presented with headaches that 'did not follow any of the known rules'.[1] Sluder reported that his patients presented with headaches that caused variable referred pain to the head and neck region. Affected areas included the teeth, retroorbital, neck, soft palate, hard palate, and the temple region. SN is believed to be caused due to irritation of the pterygopalatine ganglion that occurs secondary to inflammatory processes of the posterior ethmoid and sphenoid sinuses. The pain symptoms of SN were described as being autonomic in nature and unilateral.[1,2] Dr. Sluder observed that patients who recovered from inflammatory ailments of the posterior ethmoid and sphenoid sinuses would soon develop headaches accompanied by variable referred pain in the head and neck region.[1] He concluded that the effects of inflammatory processes in the areas of the posterior ethmoid and sphenoid sinuses were the underlying cause of the neuralgic, sensory, motor, and gustatory symptoms that patients experienced.[3] It was noted that the pain due to SN typically began at the root of the nose and remained ipsilateral, spreading to the eye, jaw, and teeth. This

accumulation of symptoms was termed as 'lower half headaches' and the pain could continue to spread to the neck, shoulder, and chest region. Sluder treated pain symptoms with 20-67% cocaine that was injected into the pterygopalatine ganglion which was shown to relieve pain of moderate intensity. However, Sluder noted that pain symptoms could return following recurrent inflammatory events affecting the pterygopalatine ganglion.[4]

Sphenopalatine neuralgia verses cluster headache

Due to much overlap between the two, there has been much discussion of whether cluster headache and SN should be viewed as the same entity or as separate conditions.[2] Both cluster headache and SN fall into the category of trigeminal autonomic cephalalgias. Trigeminal autonomic cephalalgias are headache disorders that are described as producing unilateral pain accompanied with autonomic features that are ipsilateral.[5] Cluster headache is a unilateral headache with pain that radiates to the orbit and temporal regions. Similar to sphenopalatine neuralgia, cluster headache causes unilateral pain that is accompanied by autonomic symptoms which include lacrimation, nasal congestion, ptosis, and hyperhidrosis.[6,7]

However, there are clear differences between cluster headache and SN that suggest that these two conditions are independent of each other. The severity of pain is a clear distinction between these two conditions. The pain experienced in cluster headache has been described as constant excruciating pain while pain in SN is described as a burning or aching pain that is either constant or cyclical. The length of SN attacks has been described as lasting hours to days and attacks can occur daily. Attacks of cluster headache occur in 4-12 weeks periods and episodes can occur multiple times a day lasting up to two hours. Cluster headache includes a remission period that can last 6-12 months in which an individual doesn't experience any headache.[3,8] SN pain is more widespread while pain due to cluster headache is mainly localized to the orbit and temporal regions. SN pain is located in the periorbital, maxillary, or nasal regions and can spread to additional areas of the head and neck.[2] Due to the important clinical differences between the two conditions, SN should be viewed as its own clinical disorder in the category of trigeminal autonomic cephalalgias.[6]

PATHOPHYSIOLOGY

The pterygopalatine ganglion branches off of the maxillary branch of the trigeminal nerve and lies in the pterygopalatine fossa. The sensory portion of the ganglion is made up of the sphenopalatine nerves. The motor and sympathetic portions of the sphenopalatine ganglion are derived from branches of the vidian nerve. The vidian nerve is formed by anastomoses of the greater superficial petrosal nerve and the deep petrosal nerve. The greater superficial petrosal nerve is formed from parasympathetic preganglionic cells bodies in the superior salivatory nucleus in the pons.[9] The greater superficial petrosal nerve supplies motor innervation to the pterygopalatine ganglion while the deep petrosal nerve supplies sympathetic innervation to the ganglion.[10] The greater superficial petrosal nerve transmits taste and presynaptic parasympathetic fibers while the deep petrosal nerve transmits postganglionic sympathetic fibers from the superior cervical ganglion to areas of the nasal cavity, palate, and pharynx.[11] Branches of the pterygopalatine ganglion extend to different regions of the head and neck. Branches to the eye and nasal cavity include the orbital rami, posterior superior lateral nasal rami, and posterior superior medial nasal rami. The orbital rami conduct signals to the eye while the posterior superior lateral and posterior superior medial nasal rami transmit signals to the nasal cavity. Branches of the pterygopalatine ganglion that are transmitted to the palate are the anterior palatine nerve and the middle palatine nerve.[10]

Since the majority of the above structures traverse near the pterygopalatine ganglion, it has been suggested that the pterygopalatine ganglion has a significant role in producing trigeminal autonomic cephalalgias including sphenopalatine neuralgia.[12] Triggers of headache cause activation of brain structures that are related to the superior salivatory nucleus which stimulates the trigemino-autonomic reflex. Stimulation of the superior salivatory nucleus causes activation of parasympathetic fibers in the pterygopalatine ganglion.[9] As a result, there is dilation of cranial blood vessels increasing blood flow and a release of inflammatory mediators from blood vessels. The inflammatory mediators released include acetylcholine, nitric oxide, and vasoactive intestinal peptide. Meningeal nociceptors can become activated by the resultant inflammation leading to production of pain.[8,11,13] The culmination of these physiological responses can cause the symptoms that one experiences in headache disorders such as sphenopalatine neuralgia.

EPIDEMIOLOGY AND RISK FACTORS

SN is rarely diagnosed due to its specific criteria and the increased prevalence of other similarly presenting headache disorders. In a study examining headache symptoms of 895 patients that visited a headache clinic, only 7 patients met the criteria for pterygopalatine neuralgia.[2] Studies that have evaluated the prevalence of SN are rare and there have been no reported studies evaluating the prevalence of SN in the general public. Of 88 published cases of sphenopalatine neuralgia, the ratio of cases between males and females was 1.3:1 and the median age of onset was 41.8 years old.[2] Almost all cases (99%) of SN were described with accompanying unilateral pain and of the patients that reported episodic pain, 72% of them detailed having pain that lasted at least 180 minutes.[2,6,22,14-21] There are many potential risk factors that have been described that could possibly trigger sphenopalatine neuralgia. Emotional stress, cold environments, tooth damage, and nasal surgery were all identified as possible triggers in a study that followed 8 patients with confirmed cases of sphenopalatine neuralgia.

Of the cases, 3/8 were triggered by emotional stress while cold conditions were a causing factor in one case. Nasal surgery and tooth damage each were a trigger of two cases.[2] It has been observed that patients with a history of nasal surgery have an increased incidence of sphenopalatine neuralgia. Out of eleven patients who had a history of nasal surgery, eight patients were diagnosed with SN in a study examining 88 confirmed cases of sphenopalatine neuralgia.[2] Due to the small sample size of confirmed cases of sphenopalatine neuralgia, it is difficult to evaluate the role that risk factors have in causing this disorder. As a result, prevention of SN may prove to be difficult.

METHODS

Our group aimed to bring together all the full-text, peer-reviewed publications that were related to the treatment and management of sphenopalatine neuralgia. Ovid MEDLINE, PubMed, NCBI databases, and Google scholar were searched for all publications related to SN that also involved otolaryngology or head and neck surgery. SN was originally called Sluder's neuralgia and has also been called Pterygopalatine neuralgia. All of these terms were used as MeSH terms to search for literature relating to Sphenopalatine Neuralgia. These papers were filtered according to their relevance to otolaryngology and reviewed. The references of the papers identified were reviewed to gain a more comprehensive understanding of the literature related to sphenopalatine neuralgia.

CLINICAL PRESENTATION AND DIAGNOSIS

SN was first described by Sluder in 1908. This condition was described as a moderately painful condition in which a unilateral persistent headache begins lateral to the nose or near the eye and radiates across the face, sometimes extending as far as the arm. The pain is described as burning, dull, and persistent. Pain can be continuous or occur as intermittent episodes ranging from hour to days. Itchiness in the upper extremities and distortion of taste were noted in rare cases. Recurrence of pain after treatment was observed in some cases, possibly due to inflammation.[3] SN has been characterized similarly since its first description, though specific details continue to evolve regarding details such as pain duration and demographics. Diagnosis of SN remains challenging due to lack of defined diagnostic criteria.

The sphenopalatine ganglion is the largest group of neurons that exist external to the brain. This ganglion has been connected to multiple head and facial pain conditions and therefore is target for treatment in these conditions.[9] Sluder originally described a differential diagnosis defined by the following facts. Firstly, injection of anesthetic to the sphenopalatine region resolves pain. Additionally, the aforementioned treatment does not relieve pain caused by a lesion secondary to inflammation of the sphenopalatine ganglion. Sluder postulated that this condition could be caused by lesions in the nerve trunk that supply the sphenopalatine ganglia and that the origin of the neuralgia is an obstruction at the sphenopalatine foramen. Sluder described the pain as occurring constantly with aggravating episodes or as occurring cyclically, similar to a migraine. In addition, it was noted in two cases that SN was associated with shoulder and arm weakness, which dissipated with resolve of the SN episode. Sluder performed the first anesthetic block of the sphenopalatine ganglia.[3]

The original description of SN is vague and not well defined from a Cluster headache (CH). The distinction between these two conditions has been contested for years. SN is noted as a term that also refers to cluster headaches in the first volume of the International Headache Society classification. The most current International Classification of Headache Disorders characterizes SN as a presentation of CH and not as a separate condition.[23] As previously stated, recent literature suggests that cluster headaches and SN are two distinct conditions and should be regarded as such.

Differences in patient demographics have also been noted between SN and CH. A patient diagnosed with CH is more likely to be male while a SN patient is typically female. Onset of SN is between 30 and 50 years of age, onset of CH is between 18-40 years old. Both CH and SN show autonomic features Patients with SN and CH have responded positively to treatment involving injection of anesthesia to the pterygopalatine ganglion.[24]

A literature review by He et al suggests that SN is an independent condition, separate from CH and recommends SN be further subdivided into idiopathic SN and secondary SN. Idiopathic SN may be a result of the demyelination of the sphenopalatine ganglion. Secondary SN may be due to the presence of structural lesions affecting the sphenopalatine ganglion. Treatments focused on the lesion prove effective, demonstrating the implication of lesions in secondary SN.[2]

Prevalence of SN is described as rare, having no studies exploring the prevalence of SN in any population. This review analyzes 88 cases of SN based on the original criteria described by Sluder and more recent criteria described by Oomen.[24]

The location of pain was most frequently reported to be the orbital and retroorbital regions. Pain in the temporal region and maxillary region were second and third most common, respectively. A minority of patients (8/88) reported distinct factors that elicit symptoms. The most common trigger was emotional stress. Coexisting conditions including nasal mucosal inflammation, dental infections, and septal deviation were noted in some patients. In 87/88 of patients in this study, pain did not shift sides. Pain was described as severe by 75% percent of patients. The remaining 25% of patients described the pain as extreme with a 10 out of 10 on the Visual analog scale (VAS).

Structural lesions around the sphenopalatine ganglion was reported in 59/88 patients. No patients in this study reported a cluster period of pain as characterized by CH. Pain in SN never shifts to a different side of the head, in patients with CH a side shift is noted in in 20% of cases. The ratio of female: male cases in SN is 1:1.3. In CH the female to male ratio is 1:3. This ratio is notable different from that described by Ahmed.[2]

Changes in the hypothalamus have been implicated in the pathophysiology of cluster headaches. SN is considered to be the result of damage to or stimulation of the sphenopalatine ganglion, suggesting that SN and CH have two different pathologies and therefore are distinct conditions.[25] Prevention of cluster headache can be accomplished through inhibition of neurotransmission in the sphenopalatine ganglia. It has also been shown that stimulation of the sphenopalatine ganglia can contribute to alleviation of cluster headaches.[26]

TREATMENT

Medical treatment

Early medical treatment described by Sluder of SN began with the application of cocaine to the area of the Sphenopalatine ganglion (SPG). If this failed to provide relief of the pain, he recommended application of a solution of 2% silver nitrate, 0.4% formaldehyde, 0.5% phenol, and 0.1% iodin. If this too proved to be an ineffective treatment, the next recommendation was injection of phenol-alcohol.[3]

In 1942, Eagle and Durham described treatment of SN with the application of 10% cocaine directly to the mucosal region of the SPG with the potential of being a curative treatment. If there is a recurrence of pain following cocainization and a patient is unable to be treated in person, they recommend treatment of patients with a nasal spray solution of 1-3% butyn sulfate. As a final medical treatment, they recommended injection of 0.5 cc of 5% phenol in 95% alcohol into the SPG if other treatments have failed to provide relief.[29]

Puig et al found a 90% success rate in reduction of pain for an average of 9.5 months following treatment in patients diagnosed with SN with the application of 88% phenol to the nasal mucosa of the SPG.[14] In a case report by Ernest, application of a Marcaine-soaked cotton tip applicator to the nasal mucosa saw a complete elimination of pain within 10 minutes of application.[30]

He et al make an argument for the distinction between SN and CH based on their response to certain treatments. Whereas patients with CH are readily treated by oxygen therapy and indomethacin, patients with SN do not respond to nearly the same degree with these therapies. In their case studies, He et al found effective medical treatment of SN with use of carbamazepine, oxcarbazepine, gabapentin, and pregabalin.[2]

Surgical treatment

Early surgical intervention for SN described by Sluder was an operation aimed at removal of the SPG, though he does not provide detail as to the specifics of the operation he is suggesting.[23] The focus of surgical intervention described by Eagle and Durham depended on the assumed cause of the neuralgia. The first intervention suggested is fracturing of the middle turbinate when it appears to be displaced, including crushing of the bony structure of the turbinate to prevent regrowth back to the original position. In the case of infections of the sinuses, either posterior ethmoid or sphenoid, they recommend opening of the sinuses and removal of all diseased mucosa. They also describe one case study in which a procedure to shorten the left styloid process proved therapeutic for a patient that had continued aural symptoms of SN following submucosal resection of the nasal septum which relieved her other SN symptoms, including facial pains.[29]

A case report involving stereotactic radiosurgery for treatment of SN described by Pollock and Kondziolka resulted in complete elimination of pain. Following the first procedure, the patient had a recurrence of pain at 17 months at 50% of her original pain level. Due to the recurrence of her pain, the patient underwent a repeated radio-surgical procedure, after which she was pain-free at seven months post-procedure.[33]

Although not a treatment explicitly for SN, treatment with SPG stimulation for CH was investigated by Jürgens et al. In patients with CH, an integrated lead was placed near the SPG with a patient-controlled remote that delivered stimulation upon activation. Patients were instructed to treat themselves with this stimulation as quickly as possible upon onset of a CH attack. Out of a total of 5956 CH attacks treated across 33 patient participants in the study, 65% were treated effectively, defined as either pain reduction or pain elimination, and 50% were treated with complete pain elimination. Additionally, a 50% reduction of frequency of CH attacks was seen in 35% of the participants; however, 35% of participants saw at least a 50% increase in attack frequency while 29% of patient did not have a significant change in their attack frequency.[32]

He et al presented a series of case studies for treatment of SN. In the first case, the patient's SN resolved after extraction of the right upper second molar with chronic pulpitis and deep caries followed by anti-infection therapy. The second case had resolution of SN symptoms after extraction of multiple damaged teeth with exposed root crowns and periodontitis predominantly on the right side. Extraction of damaged teeth with gingivitis and tooth pulpitis on the left lower-second and upper-first molars followed by anti-inflammatory therapy resolved symptoms of SN in the third case. In the fourth case, the SN symptoms were resolved after extraction of the C7 tooth with alveolar bone resorption and periodontitis followed by compression hemostasis and anti-inflammatory therapy.

In the fifth case, the patient demonstrated inflammation of the maxillary sinus, and treatment with repeated nasal endoscopic irrigation and rinsing eliminated the SN symptoms. The sixth case study was also treated with multiple nasal irrigation treatments, but his pain was only reduced by 60-70%. Rather than continue nasal irrigation treatments, the patient opted to be treated medically and was prescribed carbamazepine. His symptoms were completely resolved three months after starting the medication. In the final case study, the patient received a dental root canal of the right first and second incisors due to pulpitis and deep caries present in those teeth; however, this proved ineffective in reduction of symptoms. Following this treatment, the patient was treated medically with 5 mg olanzapine qn and 75 mg venlafaxine qd which eventually led to complete resolution of her symptoms. These case studies support the theory that effective treatment of PN is often achieved by addressing the lesions irritating the SPG, whether that be of dental or sinus origin.[2]

There have been multiple studies investigating the effectiveness of Pulsed radiofrequency (PRF) of the SPG in treatment of cluster headaches. Chua et al reported in a 2011 case series that 3 patients received effective reduction of their CH pain from PRF treatment of the SPG.[28] In a study by Fang et al in 2016, Computerized tomography (CT)-guided PRF of the SPG showed an 85% success rate in treatment of Episodic cluster headaches (ECH).[31] Salgado et al investigated the efficacy of both PRF of the SPG and Radiofrequency ablation (RFA) of the SPG in patients with Chronic cluster headaches (CCH).

Complete relief of symptoms was seen in 13.5% in patients and partial and transient relief was seen in 56.8% of patients in this study. Both PRF and RFA were shown to be equally effective at reducing symptoms in this study.[34] In a study by Chen et al., both patients with ECH and CCH who underwent treatment with CT-guided PRF of the SPG were investigated. In patients with ECH, initial remission of symptoms occurred in 95.6% of patients, and in patients with CCH, initial remission of symptoms occurred in 64.3% of patients, both after the first PRF treatment. The number of ECH patients with remission of symptoms dropped to 18.6% at 27 months after the PRF procedure, but no further drops in remission rates occurred out to 43 months. By 30 months after the PRF procedure, the remission rate for CCH patients had dropped to 0%.[27]

CONCLUSION

The current description of SN is a moderately painful condition with a unilateral persistent headache that radiates across the face that is burning, dull and persistent. This pain can be continuous or can have intermittent episodes ranging from hours to days.

Unfortunately, there are no specific diagnostic criteria for SN which is part of the challenge in the identification and diagnosis of SN. The only way to definitively diagnose SN according to Dr. Sluder who initially described the disease is to inject anesthetic into the sphenopalatine region. An important part of the discussion around SN involves the distinction between SN and CH and how important recent literature suggests they are distinct conditions. Especially since recent research shows that damage to or stimulation of the sphenopalatine ganglion shows the symptoms associated with SN and this is required for diagnosis. Treatment of SN begins with anesthetic to the sphenopalatine ganglion and then other topical agents can be applied that have a high success rate in reduction of the pain of SN. Definitive treatment is surgical removal of the sphenopalatine ganglion. The challenge for the modern otolaryngologist and physician in general is that there is a wide variety of symptomatology that make SN a challenge to recognize and diagnose clinically.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Swanson D, Lewis K, Thornhill K, Fabian I, Kaye AD, Viswanath O, Urits I. An update on the diagnosis, treatment, and management of sphenopalatine neuralgia. Int J Otorhinolaryngol Head Neck Surg 2022;8:286-91.