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The role of the otolaryngologist in the evaluation and management of headaches

Shirley Hu[a,*], Samuel Helman[a], Peter Filip[a], Jonathan Cabin[b,c], Patrick Colley[a]

[a] Department of Otolaryngology, New York Eye and Ear Infirmary of Mount Sinai, New York, NY, United States
[b] CENTER for Advanced Facial Plastic Surgery, Beverly Hills, CA, United States
[c] The Migraine Institute, Beverly Hills, CA, United States

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ABSTRACT

Background: Headaches are commonly evaluated in otolaryngology and often represent a diagnostic dilemma. This review addresses rhinogenic headache as well as trigeminal neuralgia and migraine, both of which can masquerade as sinus headache and whose management increasingly involves otolaryngology intervention. Discussion considers diagnostic criteria and novel therapies and derives an algorithm for clinical decision-making.

Data sources: OVID MEDLINE, Cochrane Library, and Google Scholar databases.

Methods: A literature search was performed to identify relevant articles published in the past 10 years addressing the diagnosis and management of rhinogenic headache, trigeminal neuralgia and/or migraine.

Findings: Rhinogenic headache: Identification of the specific cause must be achieved before treatment. No studies have mentioned the effect of certain therapies on the amelioration of headache. New techniques of balloon dilation for sinusitis are controversial, and their use remains contingent on surgeon preference. Removal of mucosal contact points has been shown to benefit quality of life in patients with contact point headache. Trigeminal neuralgia: Microvascular decompression is considered the gold standard for treatment, but percutaneous therapies can be effective for achieving pain control. Migraine: Patients who report amelioration of symptoms after targeted botulinum toxin injection may benefit from definitive decompression or nerve avulsion. Patients with mucosal contact points may have less favorable outcomes with migraine surgery if they are not simultaneously addressed.

Conclusions: A comprehensive understanding of the diagnostic workup and therapeutic options available for common headache etiologies is key to the management of a patient presenting with headache attributed to a rhinogenic cause.

1. Introduction

Headaches have been estimated to affect millions of patients annually [1]. These patients often present to the otolaryngologist with a presumed diagnosis of “sinus headache”. Patients describe sinus headaches as pressure or facial pain over the maxillary, ethmoid, and frontal sinuses, mandibular or maxillary pain, temporal pain, facial spasms or otalgia [2]. However, when patients have associated gastrointestinal or sensory symptoms or certain environmental triggers, they may be suffering from primary headache diagnoses such as migraine and trigeminal neuralgia. This diagnostic challenge is exacerbated by the overlapping symptomatology for all possible headache etiologies. In addition to diagnosis, the otolaryngologist plays a key therapeutic role in the management of rhinogenic headache, trigeminal neuralgia and migraine. Novel therapies for these entities have evolved in recent years and propelled otolaryngology into the forefront of their management.

We discuss the role of the otolaryngologist in the evaluation, triage and treatment of patients who present with headaches. In doing so, we also strive to enumerate the latest evidence-based diagnostic criteria and treatments, both novel and traditional, for three common etiologies of headache – sinus, migraine and trigeminal neuralgia – in the practice of otolaryngology.

2. Methods

We aimed to identify all full-text, peer-reviewed publications pertaining to headaches in the practice of otolaryngology, neurology/neurosurgery, plastic surgery and pain medicine from January 1, 2007

* Corresponding author at: 310 East 14th Street, New York, NY 10003, United States.
E-mail address: shu2@nyee.edu (S. Hu).

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to July 31, 2017. The searches were conducted in the Ovid MEDLINE, Cochrane Library and Google Scholar databases. The following keywords were used: *headache, rhinogenic, sinusitis, sinus, trigeminal neuralgia* and *migraine*. Results were combined with the terms *otolaryngology*, *neurology/neurosurgery*, *plastic surgery* and *pain* to retrieve articles. All articles addressing the management of rhinogenic headache, trigeminal neuralgia or migraine were included in the review. The reference lists of these articles were then inspected and any other pertinent publications added. Three authors (S.H., S.N.H., and P.F.) independently reviewed the titles and abstracts of all citations identified by the literature search. Commentaries, editorials and non-English articles were excluded.

3. Results

3.1. Rhinogenic headache

The otolaryngologist has a crucial role in the appropriate diagnosis of conditions underlying rhinogenic headache. The complexity of this issue and technical limitations in the primary care setting are the cause of frequent misdiagnoses and inappropriate treatment [7]. Correct diagnosis of a rhinogenic headache requires a full history and physical, including nasal endoscopy and imaging, if indicated.

Headache associated with rhinogenic signs and symptoms, or sinus headache, is defined as head or facial pain with a primary pathophysiology originating from the sinonasal cavities. Certain symptom criteria should be met before considering a rhinogenic cause for headache. This includes pain in regions that are consistent with the location of the paranasal sinus, such as the frontalis, midface, retro-orbital, or occipital regions. The pain either worsens with flight, diving, or change in weather; improves with use of a topical nasal decongestant or anesthetic agent; or occurs routinely with congestion or rhinitis [3]. The *International Headache Society* and the *American Academy of Otolaryngology–Head and Neck Surgery* consider acute, subacute and chronic rhinosinusitis as primary etiologies of rhinogenic headache [1,4,5].

In addition to headache, signs and symptoms of rhinosinusitis include facial pain and pressure, particularly of the eyes, cheeks, forehead and teeth, that is increased with straining or bending down, fever and chills, nasal obstruction, hyposmia, purulent nasal discharge, and facial erythema or edema along with other minor criteria [4–6]. Limited data exist concerning the diagnostic study of choice to evaluate rhinogenic headaches. While patient history is useful, classic pain-related symptoms associated with sinus headaches have limited accuracy: Facial pain has been reported to be eight to 30% sensitive for acute sinusitis; headache 33 to 48% sensitive; pain with bending forward 65% sensitive and 59% specific; and maxillary toothache 93% specific but present in only 11% of patients [7].

Acute rhinosinusitis (ARS) is defined as sudden-onset sinonasal inflammation lasting fewer than four weeks. Symptoms include nasal obstruction or congestion, anterior or posterior nasal discharge, facial pain and pressure and hyposmia/anosmia. Computed tomography (CT) and endoscopy are not required for diagnosis [5,6]. However, CT evaluation may frequently yield abnormalities such as outflow tract obstructions, contact points, concha bullosas, Haller cells, mucosal thickening and septal spurs [5,6]. Classically, these findings have been identified as clinical correlates to rhinogenic headaches.

Aggregate Grade A evidence states that ARS management consists of antibiotics, intranasal corticosteroids, analgesics and nasal saline irrigations [5,6]. Otolaryngologists may opt for a trial of watchful waiting for 10 days, since ARS has a high spontaneous resolution rate, or appropriate antibiotics (amoxicillin). Four recent systematic reviews of randomized controlled trials found that antibiotics provide minimal improvement in symptom resolution compared to placebo by days seven to 15 of an episode of acute sinusitis [5,6].

Chronic rhinosinusitis (CRS) is defined as sinonasal inflammation persisting for 12 or more weeks. Symptoms must include at least two of the following: 1) nasal blockage, obstruction, or congestion; 2) nasal discharge (anterior or posterior); 3) facial pain or pressure; and 4) reduction or loss of smell. The diagnosis must be confirmed by evidence of inflammation on direct paranasal sinus examination, CT of the paranasal sinuses, or other evidence of purulence in the sinuses or osteomeatal complex [5,6].

Treatment for CRS revolves around reducing sinonasal mucosa edema and facilitating mucus drainage from the sinuses. Appropriate medical management is the first line of therapy and includes nasal saline irrigations, topical intranasal steroids and appropriate antibiotics. Functional endoscopic sinus surgery (FESS) should be considered for CRS when maximal medical therapy has failed to relieve anatomical obstructions [5,6]. Indeed, a large body of evidence exists supporting the clinical efficacy of FESS in improving both CRS-specific and general quality-of-life measures in patients with CRS [8].

Sinus ostial dilation or balloon ostial dilation is a recent addition to the repertoire of the otolaryngologist, available for select patients with CRS and recurrent acute rhinosinusitis (RARS) who have failed medical therapy. Sinuplasty use has been validated by several multicenter studies and meta-analyses, with safety and effectiveness profiles similar to that of FESS [9–11]. Balloon ostial dilation may be used alone to dilate an obstructed sinus ostium (frontal, maxillary, or sphenoid) or in conjunction with other instruments (e.g., microdebrider, forceps). In CRS patients who are recalcitrant to medical management, the utilization of balloon dilation versus the use of FESS is controversial and remains contingent on surgeon preference. The use of balloon sinuplasty for the treatment of headaches in patients with evidence of minimal to moderate sinonasal inflammation is also controversial. Multiple reports have shown improvement of headaches in small cohorts of patients. However, to date, no high quality studies have been performed in this area.

Outside of the classic entities of ARS and CRS, diagnosing a rhinogenic cause of headache may be challenging for the practicing otolaryngologist. In recent years, contact point headache has gained more interest as a primary cause of sinus headache. This is a headache syndrome secondary to mucosal contact points (i.e. septal deviation, septal spurs, concha bullosa, hypertrophied inferior turbinate, medialized middle turbinate, uncinate bulla, medially or laterally uncinate process, paradoxical middle turbinate and large ethmoidal bulla in the sinonasal cavities) in the absence of inflammatory signs, hyperplastic mucosa, purulent discharge, sinonasal polyps or masses. CT imaging may be helpful, but its use in diagnosis is controversial, since established radiographic abnormalities that definitively identify a rhinogenic cause have not yet been identified. Indeed, the radiological prevalence of mucosal contact points in patients with a suspected rhinogenic cause for headache is not significantly different than that in patients without sinus headache. Further studies are thus necessary to identify clinical scenarios in which anatomic variants may contribute to pain symptoms [12–14]. Several studies have shown that surgical removal of such mucosal contact points is more effective than medical therapy in achieving improved quality of life [15,16]. Other causes of rhinogenic headache that should be considered in workup include RARS and barosinusitis.

3.2. Trigeminal neuralgia

Trigeminal neuralgia (TN) or *tic douloureux* is a complex syndrome whose treatment is generally within the scope of neurology and neurosurgery but should be easily recognized by an otolaryngologist. *Tic douloureux* describes the severe and piercing unilateral facial pain interrupted by brief periods of relief that characterizes this syndrome [17]. It can be spontaneous or elicited by triggers such as tooth brushing or face washing. Given the proximity of the sinus cavities to the source of pain in TN, patients frequently misattribute it to “sinus headache”.

The disorder was ultimately linked to the trigeminal nerve and

coined trigeminal neuralgia [17]. The cause is the compression and/or demyelination of the proximal root of the trigeminal nerve, which has been linked to multiple sclerosis and compressive lesions such as neoplasms, aneurysms and aberrant vessels. Using posterior fossa exploration and magnetic resonance imaging, evidence suggests that classic TN is linked to aberrant vessels. A few case reports have described neoplasms, such as intracranial epidermoid cysts, acoustic neuromas and rhabdomyomatous mesenchymal hamartomas of the face [18–20], masquerading as TN. Notably, patients with zygomaticomaxillary complex fractures have a higher risk of TN [21].

Carbamazepine and oxcarbamazepine are the current first line medications in the treatment of TN and have a more tolerable side effect profile compared to the second-line medication phenytoin [21]. Percutaneous ablative techniques including chemoneurolysis, radiofrequency ablation and balloon microcompression have been used with varying success to treat refractory TN [22,23]. A recent review demonstrated similar short-term (90–97%) and long-term (54–66%) pain control rates among these treatments but with increased complication rates for glycerol rhizotomy [23]. Patients who experience significant pain relief with a trigeminal ganglion block may be appropriate candidates for percutaneous radiofrequency rhizotomy, which involves selective ablation of the trigeminal ganglion or roots using radiofrequency [24]. Open surgery has evolved from nerve sectioning, which with its incumbent morbidity is no longer standard-of-care. The current gold standard surgical treatment is microvascular decompression, pioneered initially by neurosurgeon Walter Dandy, with success rates approaching 90% [17,25,26]. Stereotactic gamma knife surgery is preferable in those with numerous medical comorbidities or who are otherwise unwilling to undergo surgical intervention [17].

3.3. Migraines

Migraines are commonly diagnosed in patients who present with sinus headache complaints. Migraines are a primary headache disorder characterized by recurrent unilateral, pulsating headaches that last from two to 72 h. Associated symptoms may include nausea, vomiting, and sensitivity to light (photophobia), sound (phonophobia) or smell (sonophobia) [27]. The two main subsets of migraines relate to the presence or absence of an aura or a sensory premonition occurring up to 60 min prior to migraine onset. As the name suggests, “common migraine” (migraine *without* aura) is more common than “classic migraine” (migraine *with* aura), with two-thirds of migraine sufferers diagnosed with common migraine versus one-third diagnosed with classic migraine. “Chronic migraine” is diagnosed based on a certain migraine frequency, irrespective of the presence or absence of aura, and is generally required when considering surgical treatment, discussed below [2].

Medical management involves the use of medications, loosely categorized as prophylactic, abortive or rescue. Prophylactic medications, such as beta-blockers and anticonvulsants, are taken regularly and target migraine prevention. Abortive medications, such as triptans and ergotamines, are used to stop or dull a migraine right before or as it starts. Rescue medications are a last resort for symptom alleviation once a migraine is fully present and include pain medications, antiemetics and muscle relaxants. However, population-based surveys suggest that only 6–13% of patients who could benefit from preventive treatment of migraine are actually receiving such therapy, and 35% of patients are noncompliant with their prophylactic medications [28,29]. Recent insights into the pathogenesis of migraines continue to support a central vasogenic source of migraine pain while also describing a peripheral trigger mechanism involving compressed, irritated or otherwise malfunctioning craniofacial sensory nerves that trigger a cascade effect culminating in migraine headache [30]. In this setting, migraine surgery has been pioneered in recent years for patients who are recalcitrant to medical interventions.

Surgical management of migraines typically involves release of the peripheral sensory nerves of the head and neck that are compressed by craniofacial muscles. The concept of trigger sites is well-known. The forehead, temple and occiput are several locations that correlate consistently with specific migraine triggers [31]. In addition, the nasal septal trigger, which has been shown in studies to be the most prevalent trigger site, is often associated with retroocular pain and is readily appreciated through intranasal examination. Less common trigger sites include the auriculotemporalis and lesser occipital trigger sites [30].

Guyuron et al. [32] first proposed the peripheral theory behind migraines after observing that patients who underwent endoscopic brow lift with incidental decompression of the supraorbital (SON) and supratrochlear (STN) nerves experienced amelioration of migraine symptoms. In later years, the post-browlift relief mechanism was expanded to include the likely sacrifice of the zygomaticotemporal (ZTN) and auriculotemporal (ATN) nerves during the dissection. This study coincided with reports demonstrating the efficacy of botulinum toxin injection for the treatment of migraine [33], ostensibly by reducing of muscle tension around the nerve, further substantiating the role of peripheral mechanisms in the pathogenesis of migraine headaches. In recent years, other important triggers have been described, including occipital nerves (greater occipital - GON, lesser occipital - LON and third occipital - TON nerves) and intranasal trigeminal nerve endbranches, which can all be treated surgically [31]. Interventional migraine management involves botulinum toxin injection around suspected migraine triggers and/or surgical decompression or avulsion of one or more of these sensory nerves. Surgery in particular is reserved for migraine patients who: (1) have a chronic migraine diagnosis; (2) have exhausted medication trials (either via inefficacy or intolerance of side effects); and (3) do not have medication overuse headache (MOH) or have been fully withdrawn from medications in suspected MOH and continue to have a diagnosis of chronic migraine. It is important to note that, although effective in ameliorating migraine headaches, neither botulinum toxin nor surgery should be discussed as a “cure” for migraines, as the majority of patients will have significant reduction of frequency, duration and intensity of headaches but not complete elimination.

A number of studies have investigated the efficacy of botulinum toxin injection for the treatment of migraine headache, and efficacy has been shown particularly for patients with chronic migraine. Response rates of up to 85% have been documented in clinical reports [34], and double-blind, randomized, placebo-controlled studies, specifically the Phase III Research Evaluating Migraine Prophylaxis Therapy (PREEMPT) 1 and 2 trials, have demonstrated a significant decrease in migraine headache symptoms and improvement in quality of life measures compared with placebo after injection into various craniofacial muscles [35–39]. Injection protocol follows either one of two paradigms - the fixed-site approach, which uses fixed symmetrical injections within a range of predetermined doses in predetermined sites, or the follow-the-pain approach, in which doses and sites are tailored to patient's symptoms and the location of pain. Muscles that may be injected include the corrugators, procerus, frontalis, temporalis, occipitalis, cervical paraspinal and trapezius [40]. Aside from its efficacy in migraine treatment, botulinum toxin can be used to confirm sites of potential nerve compression. Patients who report significant reduction in migraine symptoms after botulinum toxin injection into a particular site have been shown to have increased success in migraine treatment from surgical release of the associated nerve [41,42].

Although botulinum toxin injection has a promising role in the management of migraines, its effects are temporary, and there are potential side effects, including blepharoptosis (when injected into the glabella) and muscle atrophy when injected into the temporalis. Other more significant obstacles to botulinum toxin treatment are high costs, inconsistent insurance coverage, and the quantity of injections required for the FDA-approved protocol [40].

Migraine surgery is approached through a regional lens, with delineation of the preoperative trigger site and subsequent confirmation to

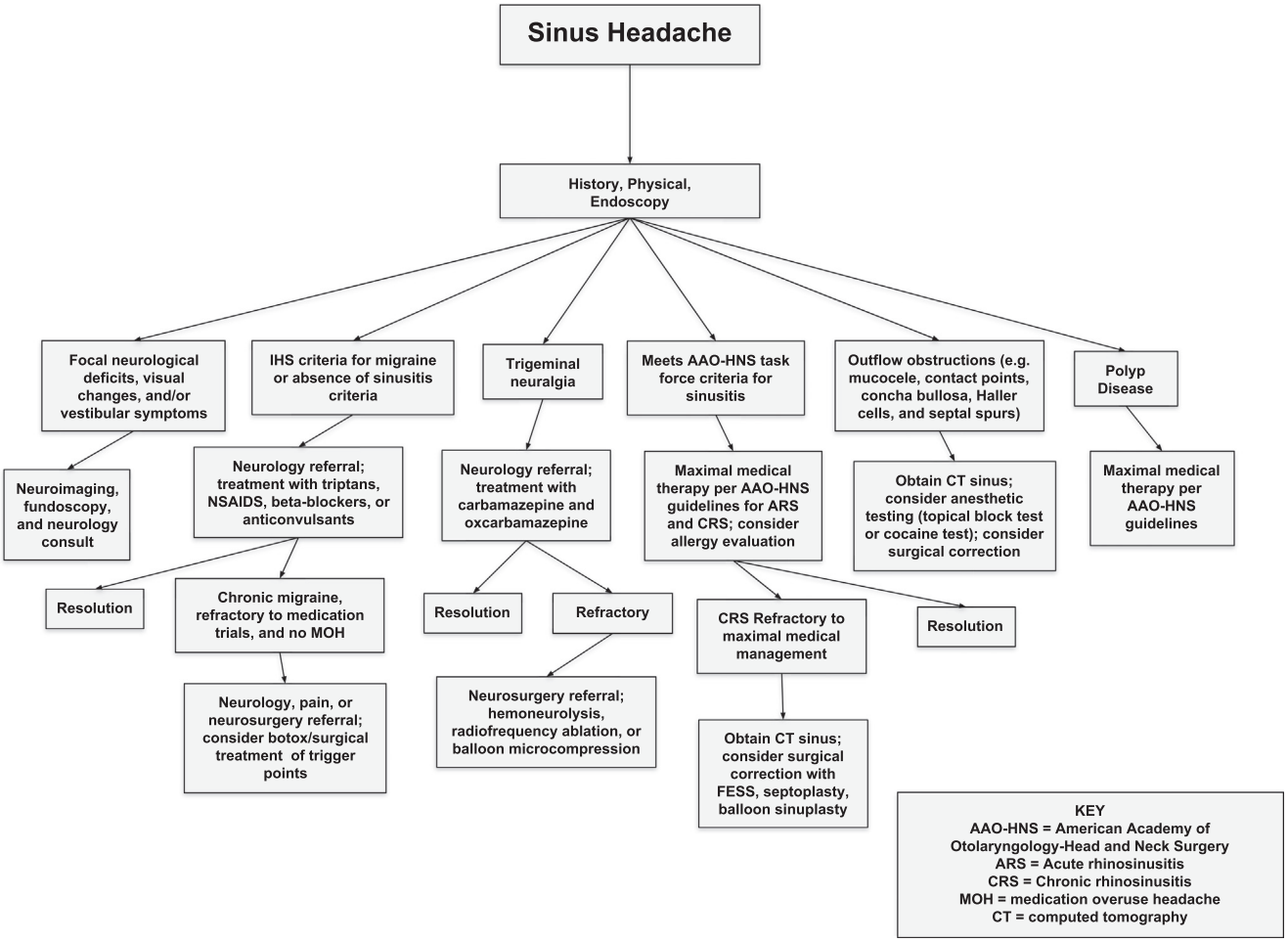

Fig. 1. Proposed algorithm for sinus headache treatment.

determine which nerves to treat. Treatment generally involves decompression via elimination of surrounding muscle, fascia, bone, cartilage and/or vasculature, although the ZTN and ATN can also be avulsed since there is minimal morbidity and surgical success rate may be increased. In a recent prospective trial, efficacy of neurectomy and decompression of the ZTN were compared. Both were found to be appropriate for the treatment of temporal migraine headache. If decompression fails to provide sufficient relief, neurectomy is another valuable option [43].

In patients with a frontal trigger, decompression of the SON and STN can be achieved via endoscopic approach and/or palpebral incisions. Non-invasive neurostimulation, specifically of the SON, is a relatively new field of interest that has been shown to be effective for the treatment of migraine in patients who have failed medical therapy [44]. For temporal triggers, decompression or neurolysis of the ZTN and/or ATN is achieved either endoscopically or via direct incision. Occipital nerve treatment involves decompression of the GON, TON and/or LON by separating the nerve from surrounding muscle, fascia or arterial vasculature, typically requiring debulking of muscle and/or vascular ligation. More recently, ultrasound-guided GON block has been used by pain specialists and noted to have superior efficacy to the standard and previously controversial GON injection [45]. Pulsed radiofrequency may also provide greater pain relief for migraine with occipital involvement than standard steroid injections [46]. Depending on the anatomy, an intranasal trigger can be treated with septoplasty and/or turbinate reduction (including resection of concha bullosa) to disrupt potential nerve irritation from anatomical contact and/or atmospheric nerve pressure [47,48]. In patients with bony septal spurs and turbinate abnormalities causing mucosal contact points not addressed during migraine surgery, less favorable outcomes have been shown [49].

4. Conclusions

Discerning the etiology of headache is a common and frequently frustrating problem for the practicing otolaryngologist. While involving a neurologist is often reflexive, it is imperative to have a working understanding of the more common headache syndromes, since treatment may fall under the purview of the otolaryngologist. This article provides a comprehensive overview of the more salient examples of headache encountered in otolaryngology, specifically rhinogenic headache, trigeminal neuralgia and migraine, with a principal focus on recent therapeutic developments. The authors also propose a suggested algorithm for approaching a patient who presents with a chief complaint of ‘sinus headache’ (Fig. 1).

Importantly, although definitive headache categories are described, there is likely a headache spectrum with overlap among etiologies, especially when considering rhinogenic headaches. Underlying causes are diverse and include ARS and CRS, frontal subperiosteal abscess (Pott's puffy tumor) [50], mucocele [51], and more concerning conditions such as intracranial extension of sinusitis and invasive fungal sinusitis [52]. Rhinogenic contact point headache has also been described and may be associated with a nasal septal trigger, septal deviation, septal spur, middle turbinate concha bullosa, sinus disease or intranasal contact points. In patients with migraine who have such associated intranasal pathology, postoperative success of migraine surgery has been shown to improve after removal of a concha bullosa and attenuate

in patients with higher preoperative contact points [49]. Rhinologic entities such as sinusitis can sometimes coincide with or even precipitate neurogenic syndromes. The central location of the sphenoid sinus explains the dramatic effect that sphenoid sinusitis has on headache [53], visual disturbance and cranial neuropathy including facial numbness. The relationship of structures such as the second, third, fourth, fifth and sixth cranial nerves, dura, internal carotid artery, sphenopalatine ganglion and structures of the pterygoid canal may be all or individually affected by acute or chronic sinusitis [54]. Cranial neuropathy resulting from sinusitis, including abducens palsy, facial numbness and/or pain may be the harbinger of worsening sinusitis and should prompt endoscopic evaluation and craniofacial imaging.

Neurogenic etiologies are also broad and include states with local nerve inflammation, syndromes such as trigeminal neuralgia, and postherpetic states leading to pain along the previously involved dermatome. Trigeminal neuralgia is particularly striking given its extreme and protracted presentation, and its differentiation from sinus headache should be routinely made in otolaryngology practice.

The peripheral mechanisms of certain headaches underlie the role of the otolaryngologist in recognizing, diagnosing and treating these headaches. Specifically, migraine has been demonstrated to result from compression of trigeminal and spinal sensory nerves in the face and neck. In patients with chronic migraine who have definitive triggers, targeting these trigger points with botulinum toxin and surgical release serve to ameliorate the symptoms of certain types of migraine. As discussed previously, migraine can have considerable overlap in symptomatology with rhinologic disease such as sinusitis and allergic rhinitis, both presenting with frontal headache, nasal congestion and lacrimation, and can exist simultaneously in the same patient, necessitating recognition of both [55].

Areas for future research include studies validating the lesser known potential causes of rhinogenic headaches, specifically mucosal contact points. The overlap between migraine and rhinogenic headache should also be explored; randomized controlled trials should be conducted to evaluate the utility of surgical removal of mucosal contact points in patients with a diagnosis of migraine undergoing either medical or surgical therapy.

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