**Abstract**

**Background:** Headache is a common symptom and a challenging one to evaluate, given its wide range of clinical presentations and different etiologies. The concept that headache can be caused by anatomic abnormalities of the nose and paranasal sinuses arose over a century ago.

**Methods:** Structured literature review.

**Results:** The number of cases of migraine or other primary headaches misdiagnosed as “sinus headache” is high in the literature, ranging from 50 to 80%. The potential mechanisms for rhinogenic headaches were classically described as pain secondary to prolonged mucosal contact points, hypoxia in the paranasal sinuses secondary to poor ventilation, or pressure caused by the growth of nasal polyps; however, other mechanisms were also described and are still being studied. Surgery for correction of mucosal contact points in the nasal cavity for the purpose of relieving headache is still controversial but can be done with good outcomes in well-selected patients.

**Conclusion:** Delay in correct diagnosis and treatment can have a direct negative impact on patient quality of life. Most cases of “sinus headache” or “rhinogenic headache” seen in clinical practice are in fact misdiagnosed primary headaches, such as migraine. Otolaryngologists should take particular care in establishing a precise diagnosis when patients present with a chief complaint of headache. A good clinical history, well-performed nasal endoscopy, and, when indicated, CT scan are essential.

**INTRODUCTION**

Headache is as common a symptom as it is challenging to evaluate, due to its wide range of clinical presentations and etiologies [1]. Many studies in the literature have found that a large number of cases of migraine go undiagnosed or are misdiagnosed as chronic rhinosinusitis [2]. Other types of tension, vascular, and tumor-related headache and facial pain can confound and delay correct diagnosis, with negative impacts on patient quality of life.

The concept that headache can be caused by anatomic abnormalities of the nose and paranasal sinuses arose over a century ago [3]. In 1920, Sluder described neuralgia of the sphenopalatine ganglion as a possible cause of chronic headache. In the 1940s, Wolf described the study of referred nasal pain after application of stimuli to internal structures of the nose [4]. However, interest in so-called rhinogenic headache increased with the advent of the endoscope and, consequently, of more precise endonasal surgical techniques, especially in recent decades [3].

On the other hand, the presence of gross nasal deformities in asymptomatic patients [1,5] leads us to believe that there are multiple mechanisms and a multifactorial etiology implicated in such cases of headache, hindering diagnosis and correlation of symptoms with anatomical findings. In this line, the selection of patients for surgical treatment is a crucial challenge. The diagnosis of pathological conditions that manifest as headache is still confounded by the fact that many authors use the term sinus headache to refer to clinical pictures that meet criteria for migraine and tension headache instead [2]. Conversely, a small percentage of patients who meet criteria for migraine may have concomitant sinus pathology [6,7].

Within this context, the objective of the present study was to conduct a review of the literature on rhinogenic headache in order to elucidate the differences between its causes, advance current understanding of this disease entity, and facilitate its diagnosis.

**METHODS**

The authors performed a search on Pubmed database in
December 2020 using the mesh terms “Headache”[Mesh] OR “Pain”[Mesh] AND non-mesh terms, frequently used in literature: “sinonasal OR middle turbinate OR rhinopathic OR rhinogenic OR sinus OR contact point OR Sluder OR nasal”. The search results were then initially refined by including only articles in English, Portuguese and Spanish.

The search produced 6035 articles.

All titles and abstracts were screened by two independent researchers. All case reports were excluded and all articles addressing to rhinogenic or sinonasal headache and related differential diagnosis proceeded in the screening process. There were no duplicates.

The remaining 121 articles after this first screening were then assessed in their full text version. 33 articles ultimately matched the objectives of this review due to their clinical relevance regarding diagnosis and management of rhinogenic headache. An additional of 19 articles were added after checking the reference lists of all of retrieved publications, mostly regarding differential diagnosis, such as migraine.

Therefore, a total of 52 studies ultimately were included in this review (Figure 1).

DEFINITION, HISTORY, AND PATHOPHYSIOLOGY

Many authors define rhinogenic headache as any pain affecting the region corresponding to the nose and paranasal sinuses. Other authors define it as a syndrome of facial pain or headache secondary to mucosal contact points in the sinonasal cavity in the absence of inflammatory signs, hyperplastic mucosa, purulent discharge, nasal polyps, or sinonasal tumors [8]. For purposes of diagnostic elucidation, we prefer to hew to a broader definition of rhinogenic headache as any pain directly related to the nose and paranasal sinuses, excluding secondary causes such as sinonasal tumors [9,10]. Several synonyms have been used to refer to rhinogenic headache, such as sinus headache, rhinopathic headache, middle turbinate headache, contact point headache, and Sluder’s headache [11,12].

The concept of rhinogenic headache secondary to mucosal contact points is not new. John Roe was the first to describe it in 1888 [13]. In the 1920s, Sluder postulated that such headaches could occur even in the absence of inflammation or infection, through rarefaction of the sinus cavity [12,14]. In 1943, McAuliffe et al. called into question the mechanisms underlying sinus pain and theorized that it did not originate in the mucosal lining of the paranasal sinuses but in the structures of the nasal cavity, paranasal sinuses, and sinus ostia [15].

Colley et al. [16] did a review in order to update the differential diagnosis between sinus headache and rhinogenic headache. The authors conclude that the most accurate way to differentiate these two entities is to consider rhinogenic headache when
the pain has no relation to inflammatory diseases of the sinus. Sinus headache, on the other hand, should be understood as a pain arising from inflammatory sinus conditions, such as acute bacterial rhinosinusitis.

Although referred pain was reported as early as 1946 [17], Greenfield, in 1986, better explained its mechanisms in the sinonasal region [18]. Pain resulting from injury or mucosal contact in the sinonasal cavity is not felt locally, but often referred to the dermatomes of the fifth cranial (trigeminal) nerve branches. This is due to the fact that afferent pain fibers and other sinonasal receptors all lead to the same pool of sensory neurons in the trigeminal nerve nucleus, as do sensory fibers originating in cutaneous receptors. These two pathways (nasal cavity and cutaneous receptors) synapse with common cortical neurons, which makes it impossible for cortical neurons to distinguish the origin of the stimulus. Thus, when the mucosa is stimulated, pain impulses are falsely localized when they reach the sensory cortex [1,18-20].

In 1988, Stammberger and Wolf hypothesized that headaches of rhinogenic origin were caused by (1) referred pain due to intense and constant mucous contact, (2) hyposia of the paranasal sinuses due to poor or absent ventilation, and (3) pressure caused by proliferation of nasal polyps [10]. In addition, they described a potential mechanism for mucosal contact point headache. According to their theory, contact between two mucosal surfaces within the nose or paranasal sinuses would generate a stimulus known as an axonal reflex. This reflex triggers release of substance P, a vasodilating neuropeptide found in unmyelinated group C nerve fibers.

Substance P causes vasodilation, plasma leak (neurogenic edema), and histamine release, among other inflammatory events. This vascular phenomenon may be responsible for migraine-like headache secondary to referred pain, as the dura mater is innervated by the trigeminal branches, as are the nasal cavity and the paranasal sinuses. In addition, constant mucosal contact would lead to localized or referred pain following the distribution of the first and second trigeminal branch dermatomes [10,11].

Substance P can be transported and released in both central and peripheral sensory neurons, mediating not only central (orthodromic) reflexes in group C fibers but also peripheral (antidromic) reflexes [21-23]. The peripheral stimulus responsible for triggering the axonal reflex may be infectious, secondary to chemical irritants, or simple mechanical (e.g., pressure).

This model of axonal reflex-mediated Substance P release, with orthodromic impulse causing pain and antidromic impulse causing local reactions, explains why mucosal contact and pressure can cause pain, mucosal edema, hypersecretion, and even increased severity of pulmonary symptoms-especially in patients with asthma-secondary to smooth muscle contraction [24,25].

To be credited as the source of facial pain or headache, the presence of mucosal contact points should be predictive of pain in the entire population. However, mucosal contact has been described in both symptomatic and asymptomatic patients (i.e., patients with and without headache). Furthermore, in symptomatic patients with unilateral pain, mucosal contact points were also observed on the contralateral side in up to 50% of patients [26]. It is also worth noting that contact between mucous membranes it not known to cause pain anywhere else in the body [27].

In a 2001 retrospective study of more than 900 patients, Abu-Bakra and Jones evaluated the correlation between headache and nasal mucosal contact points and identified that the percentage of mucosal contact was the same both in patients who has headache and those who did not [28]. Thus, they suggested that co-occurrence of mucosal contact points and headache might be purely coincidental [1,28].

Mendonça and Filho conducted a review in 2005 which highlighted the main causes of headache of nasal origin, including anterior ethmoidal nerve syndrome, Charlin’s syndrome (nasociliary neuralgia), olfactory fissure syndrome, sepal contact headache, and nasal spur headache [1]. They found that nasal deformities were frequently unaccompanied by headache, agreeing with Abu-Bakra and Jones [28]. Finally, they concluded that surgical treatment, besides correcting the anatomy itself, also influences the sensitivity of the nasal mucosa and exerts a certain placebo effect on the patient, which may be involved in clinical improvement [1].

In 2014, Patel et al. highlighted nasal endoscopy as an essential tool in the diagnosis of headache by otolaryngologists [29]. They also stressed how often cases of trigeminal autonomic cephalalgia and migraine (diagnosed by clinical history, response to treatment and excluding other causes) are erroneously diagnosed as sinus headaches (in approximately 50-80% of cases; Table 1). Finally, they concluded that proper diagnosis requires a detailed medical history and nasal endoscopy so that the physician has sufficient information to distinguish between different causes of headache or, at least, to rule out sinonasal changes that could explain the patient’s complaints [29].

| Table 1: Prospective studies of “sinus headache” suggesting migraine as the correct diagnosis (only studies with N>50). |
|---|---|---|
| N | Level Of Evidence | Conclusion |
| Schreiber Et AL, 2004 | 2991 | 1b | Migraine With Or Without Aura Is Diagnosed In Patients With “Sinus Headache” 80% Of The Time. |
| Eross Et AL, 2004 | 100 | 1b | 52% Of Patients With “Sinus Headache” Have Migraine. The Most Common Causes Of Diagnostic Error Are Triggers, Provocation, And Location. |
| Forgouhipour Et AL, 2011 | 58 | 1b | Most Patients With “Sinus Headache” (68%) Have Migraine. |
| Eloy Et AL, 2019 | - | 1b | 50-70% Of Patients With “Sinus Headache” Have Migraine. Autonomic Symptoms Can Cause Diagnostic Errors. |
Wang et al., followed 185 patients for 2 years and compared the incidence of nasal anatomical abnormalities between those with and without headache. The authors published their findings in 2017 [30]. Mucosal contact was observed in 85.9% of patients with refractory headache, but also in 80.4% of patients without headache. Of all anatomical abnormalities detected, septal deviation was the most frequent (41.1%), followed by pneumatization of the middle turbinate (32.4%); sepal deviation with contact with the lateral wall of the nasal cavity was the only abnormality significantly (P<0.05) more frequent in the headache group (55.1%) than among patients without headache (40.2%). Nevertheless, the authors concluded that mucosal contact points may not be accurately diagnosed prior to surgery and that CT of the paranasal sinuses should be mandatory in patients with refractory headache, even in the absence of sinus signs or symptoms, since, in addition to being the source of pain, mucosal contact may act as a trigger or aggravating factor for primary headaches such as migraine and tension headache [26,30].

Eloy et al. performed a non systematic review regarding the difficulties in correctly differentiate patients with migraine headache associated with autonomic symptoms from patients with sinus disease. The authors concluded that up to 50% of patients diagnosed with sinus infections actually have normal CTs and nasal endoscopy. That number is even higher when considering only pediatric population, averaging 70% in those cases [31].

Kim et al. performed a large cohort study to evaluate if nasal septum deviation is related to headache. The authors followed a group of nearly thirty thousand individuals, one third of them presenting deviated nasal septum and the rest without significant deviation. The authors, after a ten year observational period, conclude that individuals with deviated septum present with significantly greater pain scores in comparison with patients without deviated septum [32].

**DIAGNOSIS**

Most patients who present to the emergency department with headache actually have a primary headache disorder, such as migraine, tension-type headache, or cluster headache; these cases require no further investigation, since simply fulfilling the clinical criteria establishes the diagnosis. Patients who do not meet these diagnostic criteria for primary headaches are candidates for investigation of other etiologies, including rhinogenic headache.

Currently, diagnostic studies that directly compare the different causes of headache (rhinogenic, temporomandibular joint dysfunction, tension-type, medication overuse, trigeminal neuralgia) are scarce in the literature.

The International Classification of Headache Disorders (ICHD-3), published in 2013 and updated in 2018, uses the term “headache attributed to chronic or recurring rhinosinusitis” when the duration of symptoms exceeds 12 weeks. Both headache attributed to acute rhinosinusitis and that attributed to chronic rhinosinusitis have similar four diagnostic criteria (Table 2), all of which must be met (A, B, C, and D) with evidence of causation demonstrated by at least two factors (criterion C). In addition, the ICHD-3 abolished the previously used term “sinus headache” as obsolete, because it was used simultaneously to describe primary and secondary headaches attributable to various pathological conditions or disorders of the nose and paranasal sinuses [7,33-35]. The 2018 update of ICHD-3 states that it is still controversial affirm that chronic rhinosinusitis can produce persistent headache, but it seems that recent evidence seems to support this causation. Besides, it also states that image findings on CT correlating with patient’s pain are not sufficient to secure the diagnosis, if the clinical criteria are not met either [36].

Due to the very limited evidence, the ICHD-3 no longer includes mucosal contact point headache, which was present in the previous Classification (ICHD-2, published in 2004) and based on the criteria in Table 3.

However, according to the EPOS 2020 [27], reference commonly used in otolaryngology practice, acute rhinosinusitis is often accompanied by facial pain, usually severe and unilateral. In addition, patients have a history of upper airway infection immediately prior to onset of pain, in addition to nasal obstruction (which is often unilateral in these cases). The latest version of the EPOS, as well as the ICHD-3, also state that chronic rhinosinusitis rarely causes any type of pain, except when there is obstruction of the sinus ostium, in which case it resembles acute rhinosinusitis [27].

According to EPOS 2020, up to 40% of patients with migraine experience nasal discharge, unilateral nasal congestion, tearing, redness, or ocular swelling during attacks, although the duration of symptoms rarely exceeds 72 hours [27]. This may make it

<table>
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<tr>
<th>Table 2: Diagnostic Criteria for Headache Attributed To Chronic Disorder Of The Nose Or Paranasal Sinuses According To The 2018 International Classification Of Headache Disorders (ICHD-3).</th>
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<tr>
<td>A</td>
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<td>4.</td>
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<td>D</td>
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more difficult to distinguish migraine from acute rhinosinusitis. Thus, it is essential to determine via a detailed clinical history whether the patient had an upper respiratory tract infection prior to the onset of the current symptoms, as well as through physical examination of the middle meatus, either by anterior rhinoscopy when possible or, preferably, by nasal endoscopic examination. On the other hand, it is worth noting that more than 80% of patients with purulent secretion in any region of the nasal cavity at endoscopy do not experience headache or facial pain [27]; hence, the importance of a detailed clinical history and physical and endoscopic examination to ensure a more accurate diagnosis. Table 4 summarizes the diagnostic criteria for rhinosinusitis according to EPOS 2020.

Therefore, according to EPOS, the key features of headache related to the paranasal sinuses are exacerbation of pain during upper respiratory tract infections, with associated nasal Symptoms (obstruction and/or rhinorrhea), as well as improvement of pain when antibiotic therapy is initiated as indicated [27].

Other authors state that once primary headache has been ruled out and rhinogenic headache is suspected, in the absence of a typical history of rhinosinusitis, the workup should continue with some specific sinonasal abnormalities in mind. Negative findings on previous examinations (clinical history and rhinoscopy) do not rule out sinus etiology. Some lesions imperceptible to physical examination with the naked eye can be identified through nasal endoscopy. The best diagnostic outcomes are achieved with a combination of nasal endoscopy and computed tomography of the paranasal sinuses, when indicated. The combination of different diagnostic modalities can provide more of the information necessary to elucidate each case [10,29,35].

Regarding the diagnosis of sinus headache, Maurya et al. [37] evaluated the patterns of pain and location of sinus disease. They conclude that individuals reporting pain near the eyebrows can have frontal sinus disease. On the other hand, patients with facial pain usually present with maxillary sinus disease.

Furthermore, paranasal CT scan should focus on coronal sections, which allow a better interpretation of the most narrow areas of the lateral nasal walls, key regions of which (such as the ethmoidal infundibulum and frontal recess) extend to the frontal plane[10,29,35].

### DIFFERENTIAL DIAGNOSIS

The main differential diagnoses of rhinogenic headache are hemicrania continua, trigeminal neuralgia, tension headache, medication overuse headache, giant-cell arteritis (temporal arteritis), Temporomandibular Joint (TMJ) dysfunction, and migraine.

Paroxysmal hemicrania and hemicrania continua are characterized by strictly unilateral pain with ipsilateral conjunctival injection, tearing, nasal congestion, rhinorrhea, facial (particularly forehead) sweating, miosis, ptosis, and/or eyelid edema. The former is hyperacute, with attacks lasting from 2 to 30 minutes, while the second is persistent (lasting more than 3 months) [29,34]. Trigeminal neuralgia is characteristically unilateral, recurrent, and shock-like, with abrupt onset and resolution of symptoms, and limited to the distribution of one or more divisions of the trigeminal nerve [29,34].

Tension headache is typically bilateral, characterized by a feeling of pressure or tightness, of moderate intensity, lasting minutes to days. The pain does not worsen with routine physical activities, nor is it associated with nausea, although there may be photophobia and phonophobia [29,34].

Medication overuse headache is defined as that occurring on 15 or more days per month, in a patient with pre-existing headache, accompanied by excessive use of one or more acute or symptomatic headache medications for more than 3 months. Pain usually resolves after drug overuse is stopped [29,34].

Giant-cell arteritis, or temporal arteritis, may present with headache as its only symptom, usually with associated scalp hypersensitivity and/or jaw claudication most commonly in women with a mean age of onset of 70 years. There is also an elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) and a wall thickening observed in temporal artery biopsy. It has grave consequences if not diagnosed and treated in a timely manner [29,34].

Headache attributed to TMJ disorders is usually unilateral, when the temporomandibular complex is the cause of pain, or bilateral when there is muscle involvement; there is clinical and/or imaging evidence of a pathological process involving the TMJ. The criteria for the diagnosis include: pain precipitated by jaw movements and/or chewing hard or tough food; reduced range of or irregular jaw opening; noise from one or both TMJ during jaw movements and tenderness of the joint capsule(s) of one or

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**Table 3:** Mucosal contact point headache according to the 2004 International Classification of Headache Disorders (ICHD-2).

| A. | Intermittent pain localised to the periorbital and medial canthal or temporozygomatic regions and fulfilling criteria C and D |
| B. | Clinical, nasal endoscopic and/or CT imaging evidence of mucosal contact points without acute rhinosinusitis |
| C. | Evidence that the pain can be attributed to mucosal contact based on at least one of the following: |
| 1. | Pain corresponds to gravitational variations in mucosal congestion as the patient moves between upright and recumbent postures |
| 2. | Abolition of pain within 5 minutes after diagnostic topical application of local anesthesia to the middle turbinate using placebo or other controls |
| D. | Pain resolves within 7 days, and does not recur, after surgical removal of mucosal contact points |

**Note:** Abolition of pain means complete relief of pain, indicated by a score of zero on a visual analogue scale (VAS).

Table 4: Diagnostic Criteria for Rhinosinusitis According To EPOS 2020.
· Inflammation of the nose and the paranasal sinuses characterized by nasal blockage / obstruction/congestion or nasal discharge (anterior/ posterior nasal drip), plus:
  o Facial pain/pressure, and/or
  o Reduction or loss of smell.
· And either endoscopic signs of:
  o Nasal polyps, and/or
  o Mucopurulent discharge primarily from the middle meatus, and/or
  o Edema/mucosal obstruction primarily in the middle meatus.
· And/or:
  o Mucosal changes in the ostiomeatal complex and/or sinuses on CT.
Note 1: When the duration of symptoms is ≥12 weeks, rhinosinusitis is classified as chronic. When the duration of symptoms is <12 weeks, it is classified as acute.
Note 2: In children, reduction or loss of smell is replaced by cough, as it is the most commonly reported symptom in the pediatric population (50-80%).

Table 5: Differential diagnosis of rhinogenic headache and respective treatments.

<table>
<thead>
<tr>
<th>Differential Diagnoses</th>
<th>Diagnosis</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>Paroxysmal hemicrania and hemicrania continua</td>
<td>Clinical</td>
<td>Oral indomethacin</td>
</tr>
<tr>
<td>Trigeminal neuralgia</td>
<td>Clinical</td>
<td>Anticonvulsants, muscle relaxants, neuroleptic agents and surgery (refractory cases)</td>
</tr>
<tr>
<td>Tension headache</td>
<td>Clinical</td>
<td>Nonsteroidal anti-inflammatory agents and antidepressants</td>
</tr>
<tr>
<td>Medication overuse headache</td>
<td>Clinical</td>
<td>Discontinuation of medication</td>
</tr>
<tr>
<td>Giant-cell arteritis</td>
<td>ESR, CRP, Biopsy</td>
<td>Corticosteroids</td>
</tr>
<tr>
<td>Headache attributed to TMJ disorders</td>
<td>Clinical</td>
<td>Behavioral changes and surgery (refractory cases)</td>
</tr>
<tr>
<td>Migraine</td>
<td>Clinical</td>
<td>Triptans, ergots, nonsteroidal anti-inflammatory agents, among others</td>
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</table>

Both TMJ. Also, headache may be exacerbated by jaw movement and/or provocative maneuvers applied to TMJ structures [29,34].

Finally, migraine, as widely known and prevalent in clinical practice, consists of a typically unilateral headache, lasting 4 to 72 hours, pulsatile in nature, moderate to severe in intensity, typically aggravated by physical activity, and associated with nausea and/or vomiting, in addition to photophobia and/or phonophobia [29,34].

TREATMENT

Once the proper diagnosis has been established among the various possible causes of headache, the most appropriate treatment should be instituted [29].

Paroxysmal hemicrania and hemicrania continua respond well to oral indomethacin, which, in addition to being one of the diagnostic criteria, provides excellent relief for the patient. Indomethacin should be prescribed at a dose of 150 mg or higher, although maintenance doses should be lower [29,34]. Current treatment for trigeminal neuralgia consists anticonvulsants, muscle relaxants, neuroleptic agents, and, in refractory cases, surgery [29,34].

The treatment of tension headache is complex and involves a wide range of medications, such as nonsteroidal anti-inflammatory agents, muscle relaxants, and antidepressants, among others [29,34]. Medication overuse headache should be treated with complete discontinuation of medications, followed by a multidisciplinary approach [29,34].

Despite dosage controversies, corticosteroids remain the treatment of choice for giant-cell arteritis [29,34]. Headache attributed to TMJ disorders should be treated initially with conservative measures, such as behavioral changes and oral appliances; if there is no improvement, invasive procedures can be considered, as occlusion-changes interventions and joint surgery [29,34]. Nevertheless, considering the most prevalent kind of pain in temporomandibular joint disorders is myofascial, muscle relaxants, nonsteroidal anti-inflammatory drugs (NSAIDS) and physiotherapy are important modalities for the treatment of this condition [38].

The abortive treatment of migraine consists of the use of triptans, a class of agonists of the 5-HT1B, 5-HT1D, and, to a lesser extent, 5-HT1F serotonin receptors. Triptan administration leads to reduced meningeal vasodilation, decreased synaptic transmission at trigeminal nerve endings, and reduced release of neuropeptides, including CGRP. Examples include sumatriptan, rizatriptan, zolmitriptan, and naratriptan [29,34,39,40].

Cady et al. (2005) [41], in a prospective cohort study of 47
patients, administered two 50-mg doses of oral sumatriptan to assess reduction in headache severity and concluded that most patients with a previous diagnosis of sinus headache could be treated effectively with targeted therapy for migraine. In 2007, Ishkanian et al. [42] administered a 50-mg dose of oral sumatriptan to 216 patients with a goal of reducing pain on a four-point scale and found that sumatriptan is effective in treating patients with “sinus headache”. In 2008, Kari and DelGaudio reported similar results in 54 patients treated with triptans; the authors found that, in addition to the majority of patients with “sinus headache” deriving effective relief from migraine-specific therapy, a trial of such therapy may even help establish the diagnosis [43].

The combination of these recent studies prompted Patel et al. in 2014 to state that, when faced with a patient with “sinus headache” in whom there is no evidence of soft-tissue content in the paranasal sinuses or obstruction of sinus drainage ostia on CT, and whose signs and symptoms do not clearly point to any of the aforementioned primary headache etiologies, a trial of triptan therapy is the best choice for pain relief until the patient can be referred to a neurologist [29].

As noted above, regarding mucosal contact points as an etiology of headache, there is still a lack of studies providing robust scientific evidence to establish a causallink. Controversies also persist regarding the effectiveness of surgery for headache improvement in these patients. Despite this research gap, some patients may benefit from nasal surgery to eliminate mucosal contacts and relieve headache: those who have failed therapy for primary headache diagnosed by a neurologist while simultaneously experiencing improvement of pain after application of anesthetic to the mucosal contact point. Even in this population, the otorhinolaryngologist should have an in-depth talk with the patient to clarify risks, benefits, and therapeutic alternatives, with emphasis on the fact that surgery may or may not provide relief of facial pain or headache [29].

In 1992, Novak and Makek [44] evaluated subjective improvement in 299 patients with frequent or treatment-refractory migraine and concluded that surgery is a successful approach for patients with headache and mucosal contact points. In 2000, Tosun et al. [45] evaluated subjective improvement of pain in 30 patients with mucosal contact points and no other cause of headache and found that surgery is a favorable approach in patients with headache in the presence of mucosal contact and no other apparent cause. Welge-Luessen et al. [46], in 2003, performed surgery with the aim of achieving subjective pain improvement in 20 patients with refractory migraine or cluster headache and mucosal contact, and concluded that surgery is a successful approach in patients with headache and mucosal contact points.

Bektas et al. [47], in 2009, selected 36 patients with what would they would diagnose as mucosal contact headache (defined as recurrent headache or facial pain in the absence of inflammation, allergy, or tumors in the nasal cavity at CT, along with normal neurological and ophthalmologic examinations and identifiable mucosal contact points on nasal endoscopy and/or CT). The outcome of interest was improvement in visual analogue pain scale scores. The authors concluded that mucosal contact removal surgery is very effective in carefully selected patients. In 2010, Yazici et al. [48], also with an endpoint of pain improvement on a visual analogue scale, assessed 73 patients with migraine or tension headache and mucosal contact points. They concluded that patients with primary headache and mucosal contact points who do not respond to treatment may benefit from nasal surgery. In 2016, Peric et al. [49,50,51] used a visual analogue scale to evaluate patients with headache resistant to pharmacological treatment and nasal mucosal contact points, and concluded that removal of these contacts may be an effective treatment. Outcomes were better for patients with concha bullosa and septal spur than for those with non-contact side-wall septal deviation.

Yilmaz et al. [52] investigated if patients with contact point between nasal septum and inferior turbinates would improve their symptoms with surgery. The authors divided individuals in two groups, one with surgical approach and with the clinical treatment only. Patients who had surgery had significantly lower pain scores one year after the procedure in comparison with the clinical treatment group.

**CONCLUSION**

According to the literature, a very large number of cases of migraine or tension headache are undiagnosed or misdiagnosed as rhinogenic headache. Delay or mistake in diagnosis and treatment can have a significant negative impact on patient quality of life. Otolaryngologists should take particular care in establishing a precise diagnosis. A thorough clinical history, well-performed nasal endoscopy, and, when indicated, CT scan of the paranasal sinuses are essential. Recent evidence suggests that

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**Table 6: Studies of surgical treatment for patients with mucosal contact point headache.**

<table>
<thead>
<tr>
<th>Study Group</th>
<th>Assessment of Improvement</th>
<th>Conclusion</th>
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<tbody>
<tr>
<td>Novak &amp; 299 Makek, 1992</td>
<td>Subjective</td>
<td>Effective for patients with headache and mucosal contact points</td>
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<tr>
<td>Tosun, 2000 et al. [30]</td>
<td>Subjective</td>
<td>Favorable for patients with mucosal contact and no other apparent cause</td>
</tr>
<tr>
<td>Welge-Luessen et al., 2003 [20]</td>
<td>Subjective</td>
<td>Effective for patients with headache and mucosal contact points</td>
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<tr>
<td>Bektas 2009 et al. [36]</td>
<td>Visual analog scale</td>
<td>Effective for selected patients carefully</td>
</tr>
<tr>
<td>Yazici 2010 et al., [48]</td>
<td>Visual analog scale</td>
<td>May benefit some patients with primary headache and mucosal contact points</td>
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triptans may be the best choice for pain relief in patients with suspected rhinogenic headache but no CT evidence of soft-tissue content in the paranasal sinuses or obstruction of sinus drainage ostia and in whom other causes of primary headache for which more specific therapy is available have been ruled out. Surgery for resolution of mucosal contact points can be an excellent therapeutic alternative in carefully selected patients, even in cases of migraine or tension headache.

REFERENCES