



# Debunking Myths: Sinus Headache

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Accepted: 27 May 2021 / Published online: 20 June 2021

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## Abstract

**Purpose of Review** Although sinus headache has been extensively reviewed and described, misdiagnosis remains common. This paper discusses the myths and truths about sinus headaches.

**Recent Findings** Sinus headache is used colloquially to attribute facial pain to allergies or a sinus infection; however, most sinus headaches are migraine. Sinus-region pain from sinusitis and migraine share the same origins in the trigeminovascular system, but their causes are very different. After reviewing sinus anatomy and sinogenic pain, we provide information to assist clinicians in correctly diagnosing patients with the additional goal of avoiding unnecessary investigations and treatments. Migraine medications can be used as both a treatment and a diagnostic tool. Other differential diagnoses of facial pain are discussed.

**Summary** Sinus headache is not a diagnosis. All patients with facial pain or pressure with sinus symptoms should be evaluated for migraine because most sinus headache presentations are migraine and require migraine-directed treatment.

**Keywords** Allergies · Facial pain · Headache · Migraine · Sinus headache · Sinusitis

## Introduction

When patients present to their primary care provider with a chief complaint of recurrent frontal headache, a diagnosis of “sinus headache” is often the focus. The 2016 National Ambulatory Medical Care Survey showed that 4.1 million patients in the USA visited their physician’s office for chronic sinusitis as a primary diagnosis [1]. These visits are an opportune time for clinicians to verify the diagnosis and screen for other conditions, like migraine, before considering treatment. The term “sinus headache” is frequently used both colloquially and medically. However, this inaccurate term has led to frequent misdiagnoses and inappropriate treatments. The neurology, otolaryngology, allergy, and primary care 2005 consensus statement emphasized that it is important to recognize that many patients with sinus headaches actually have migraine, but causes of sinogenic headaches

also exist [2]. In this paper, we argue that the term sinus headache should be abandoned for accurate diagnoses. In particular, migraine is one of the most common etiologies underlying a presentation of sinus headache, and it is important to note that patients can have concurrent migraine and rhinosinusitis.

**The Myth** Head and facial pain secondary to sinus disease are common and should be treated primarily with antihistamines, antibiotics, steroids, and decongestants and labeled as sinus headache.

**The Truth** Misdiagnosis of head and facial pain is common, and sinus headache is not a diagnosis. The most likely diagnosis for these patients is migraine, sometimes with concurrent sinusitis, and these patients require migraine-directed treatment.

## Let’s Talk About the Possible Diagnoses

Headaches related to sinus disorders and migraine have overlapping symptoms, making them difficult to diagnose appropriately. To date, no universal definition of sinus headache is accepted by all related disciplines. The bulk of the medical literature describes pathophysiology, predisposing factors,

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This article is part of the Topical Collection on *Headache*

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and diagnostic imaging tools based on outdated diagnostic criteria from more than 20 years ago.

The American Academy of Otolaryngology-Headache and Neck Surgery provided diagnostic criteria in 1997 [3], which were later updated in 2015 [4]. Rhinosinusitis (RS) is divided into acute (ARS) and chronic (CRS) types. ARS is defined as less than 4 weeks of purulent nasal discharge accompanied by nasal obstruction, facial pain/pressure/fullness, or both. Nasal obstruction can be defined as congestion, blockage, or stuffiness, or it may be seen on examination [4]. If these symptoms occur for less than 10 days without worsening, the presumed cause is viral, whereas ARS symptoms that occur for more than 10 days with worsening are presumed to be caused by bacterial infection. By comparison, CRS is defined as having at least 2 of the following symptoms lasting 12 weeks or longer: mucopurulent discharge; nasal obstruction; facial pain, pressure, or fullness; or anosmia and documented inflammation (purulent mucus, edema, polyps, or radiographic evidence) [4]. The original criteria from 1997 required the presence of 2 or more major factors or 1 major and 2 minor factors [3]. Major factors included facial pain or pressure, nasal obstruction or blockage, purulent nasal discharge, hyposmia or anosmia, or purulence in the nasal cavity on examination [3]. Minor factors included headache, fever, halitosis, dental pain, cough, or ear pain/pressure [3]. Interestingly, many of these symptoms, including facial pain/pressure, headache, dental pain, ear pain/pressure, and congestion, which could be one interpretation of nasal blockage, can be seen in migraine.

The Sino-Nasal Outcome Test (SNOT-22) screens for symptom control in the setting of CRS, with a sensitivity of 71.4% and specificity of 85.5% for identifying poor control [5]. However, a systematic review showed that patients with other conditions, such as asthma and depression, can also have high screening scores, so the normal value is unclear [6]. Furthermore, the items on the SNOT-22 include the need to blow the nose; nasal blockage; sneezing; runny nose; postnasal discharge; thick nasal discharge; ear fullness; dizziness; ear pain; facial pain or pressure; decreased sense of smell or taste; difficulty falling asleep; waking up at night; lack of a good night's sleep; waking up tired; fatigue; reduced productivity; reduced concentration; feeling frustrated, restless, or irritable; feeling sad; and feeling embarrassed [5]. Migraine patients may also have elevated SNOT-22 scores in addition to anosmia and thickened discharge [7]. The pathophysiology of migraine includes autonomic pathways leading to symptoms such as congestion, rhinorrhea, and facial pain. Hence, physicians should consider migraine diagnosis in patients with a high SNOT-22 score, especially if the patients have normal objective test results or unremarkable endoscopy or computed tomography (CT) findings [7].

The International Headache Society updated the 2018 *International Classification of Headache Disorders, Version 3 (ICHD-3)*, for all primary and secondary headache disorders [8]. However, the ICHD-3 includes more than 300 diagnoses, which is a large set to remember even for experienced headache specialists and certainly for other clinicians. Furthermore, the criteria for headache attributed to ARS or CRS are not informative, as the ICHD-3 simply states that there should be objective evidence of sinusitis and that headache onset or resolution should match that of the sinusitis [8]. The ICHD-3 criteria for ARS and CRS are as follows.

Criteria for headache attributed to ARS:

- A. Any headache fulfilling criterion C
- B. Clinical, nasal endoscopic, and/or imaging evidence of acute rhinosinusitis
- C. Evidence of causation demonstrated by at least two of the following:
  1. Headache has developed in temporal relation to the onset of rhinosinusitis.
  2. Either or both of the following:
    - a) Headache has significantly worsened in parallel with worsening of the rhinosinusitis.
    - b) Headache has significantly improved or resolved in parallel with the improvement in or resolution of the rhinosinusitis.
  3. Headache is exacerbated by pressure applied over the paranasal sinuses.
  4. In the case of unilateral rhinosinusitis, headache is localized and ipsilateral to it.
- D. Not better accounted for by another ICHD-3 diagnosis

Criteria for headache attributed to CRS:

- A. Any headache fulfilling criterion C
- B. Clinical, nasal endoscopic, and/or imaging evidence of current or past infection or other inflammatory process within the paranasal sinuses
- C. Evidence of causation demonstrated by at least two of the following:
  1. Headache has developed in temporal relation to the onset of chronic rhinosinusitis.
  2. Headache waxes and wanes in parallel with the degree of sinus congestion and other symptoms of chronic rhinosinusitis.
  3. Headache is exacerbated by pressure applied over the paranasal sinuses.

4. In the case of unilateral rhinosinusitis, headache is localized and ipsilateral to it.

D. Not better accounted for by another ICHD-3 diagnosis [8]

The difficulty with this approach is that a diagnosis of sinus headache (which is likely migraine) often leads to CT or magnetic resonance imaging that then shows some mildly thickened mucosa in the sinus cavities. Essentially, pain leads to diagnosis. But how do we determine whether mucus found on imaging is actually causing the pain? In many cases, there is no correlation. A better screening technique is needed to differentiate ARS or CRS from migraine.

In contrast, the tool called ID Migraine can be helpful for migraine diagnosis [9]. This screen is a 3-item self-administered test often referred to as “PIN the diagnosis.” PIN stands for photophobia, impairment, and nausea [10]. A systematic review from 13 studies with 5866 pooled patients using the ID Migraine found that the sensitivity was 0.84 and specificity was 0.76 [11].

Another potentially helpful tool in differentiating RS from migraine is osmophobia. RS is often associated with anosmia, whereas osmophobia is common in migraine. One study of 235 patients showed that osmophobia had a positive predictive value of 87.6% for migraine [12].

When considering the presentation of sinus headache as facial pain, it is important to mention other differential diagnoses, including trigeminal neuralgia or 1 of the 4 trigeminal autonomic cephalgias (TACs). Trigeminal neuralgia is more common in the V2 and V3 distribution; it is described as electrical, stabbing, or sharp severe paroxysmal pain lasting 1 to 120 s and affects patients' ability to brush their teeth, talk, or even chew [8]. Trigeminal neuralgia is often misdiagnosed as a disorder originating from the teeth or temporomandibular joint, with patients often presenting to dentistry, where likely unnecessary tooth extractions and procedures are performed [13]. Cranial autonomic symptoms can be seen in trigeminal neuralgia but tend to be mild [14]. The first-line treatment is carbamazepine or oxcarbazepine [15]. The 4 TACs are differentiated by their duration, but all share the same location of pain: unilateral V1 or periorbital [8]. Although migraine can also be periorbital, the TACs are excruciatingly painful headache disorders associated with ipsilateral cranial autonomic features, including lacrimation, clear rhinorrhea, conjunctival injection, ptosis, edema, or flushing. Differentiation of the diagnoses of migraine, ARS, CRS, and TACs is provided in Table 1.

Secondary etiologies of TCAs include aneurysms or pituitary tumors, and therefore, recommended initial imaging includes magnetic resonance imaging of the brain with and without contrast, along with magnetic resonance angiography of the head and neck without contrast. Because these disorders

are severe, referral to a headache specialist is also recommended.

Another sinogenic cause to consider is sphenoid sinusitis, for which the most common presenting symptom is a headache that is often in the frontal, temporal, and retro-orbital distribution but that can radiate to the occipital or V1–V3 distribution [16, 17]. Sphenoid sinusitis is rare and estimated to represent only 1.0 to 2.7% of paranasal sinus disease [18]. Many case series review the presentation of sphenoid sinusitis, including one with 12 patients with headache or facial pain in all patients but often without fever or leukocytosis [19]. Another case series with 14 patients reported headaches in 92.2%, retro-orbital pain in 42.9%, nasal symptoms (including rhinorrhea, nasal obstruction, and hyposmia) in 42.9%, diplopia in 35.7%, ptosis in 4%, cranial nerve VI palsy in 14.3%, cranial nerve V2 neuropathy in 7.1%, cranial nerve III palsy in 7.1%, and decreased visual acuity in 7.1% [18]. Because only one third of these patients have abnormal nasal endoscopy, neuroimaging is required for a diagnosis of sphenoid sinusitis [18, 20]. Acute cases of sphenoid sinusitis, in particular, may have nasal symptoms, but classic signs of ARS or CRS are frequently absent [16]. Sphenoid sinus abnormalities can be incidental findings but require immediate attention to avoid severe and even fatal complications such as cranial neuropathy, cavernous sinus thrombosis, orbital cellulitis or abscess, and meningitis [16, 21].

Finally, mucosal contact-point headaches are controversial but may exist. This diagnosis can be ruled out with a trial of topical lidocaine on the mucosal contact point [22–24].

## Anatomy and Pathophysiology of “Sinus Headache”

The paranasal sinuses remain a keen anatomical interest for many specialists, including dentists, maxillofacial surgeons, otolaryngologists, and headache specialists. These structures separate the external world from the brain. The medieval Latin origin of the term sinus refers to a curve, fold, or hollow [25]. The superior maxilla was identified as early as 3700 BCE by the ancient Egyptians [26], who were innovators in their use of instrumentation to remove the brain, potentially through the sinuses. Nathaniel Highmore, in the 1600s, was the first to describe the maxillary sinus, which was later labeled as the antrum of Highmore [26]. Greek physician Claudius Galen laid the anatomical groundwork for subsequent millennia through his dissections and writings in Rome in 130–200 CE [26]. In 1959, Formby [26] summarized the maxillary sinus in the president's address to the Royal Society of Medicine in London, reviewing its historical origin, anatomy, and physiology. The health and integrity of the sinus functionality center on 4 basic principles: air currents, capillary blood flow, cilia, and mucus [26].

**Table 1** Symptom differentiation among facial pain disorders

Symptom	Migraine	Acute sinusitis	Chronic sinusitis	Trigeminal autonomic cephalgias
Facial pain	Unilateral or bilateral	Common	Unclear	Unilateral V1 distribution
Lacrimation	Common	Common	Common	Common
Sinus pressure	Common	Common	Unclear	Rare
Rhinorrhea	Common, but clear	Yes, purulent	Yes, purulent	Unilateral
Congestion	Common	Common	Common	Unilateral
Fever	No	Yes	Sometimes	No
Anosmia	No	Yes	Yes	Rare
Osmophobia	Yes	No	No	Less common
Photophobia	Yes	No	No	Less common
Phonophobia	Yes	No	No	Less common
Nausea/vomiting	Yes	No	No	Less common
Aura	~30% of migraine	No	No	No
Weather trigger	Common	Common	Common	Occasional
Duration	4–72 h; chronic migraine $\geq 15$ days per month	<4 weeks	$\geq 12$ weeks	SUNCT: 1–600 s Paroxysmal hemicrania: 10–30 min Cluster headache: 30 min to 4 h Hemicrania continua: $\geq 3$ months

Abbreviation: *SUNCT*, short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing

The paranasal sinuses can be categorized into 4 pairs: maxillary, frontal, ethmoid, and sphenoid sinuses. The sinuses are lined with a mucosal layer and congruent with the nasal cavity, leading to a fast spread of infection. See Fig. 1 for sinus anatomy including innervation and vasculature. Table 2 describes the anatomy and clinical issues related to these 4 sinuses [27–31].

The paranasal sinus innervation originates in cranial and cervical nerves and encompasses a complex network of parasympathetic and sympathetic nerve pathways. The simple term sinus headache is frequently used to describe facial pain, but the pathophysiology is far from straightforward. To understand the diagnosis, we must first understand the sources of common sinus-related symptoms, including rhinorrhea, sneezing, and facial pain.

The nasal passage defends against external stimuli to maintain homeostasis. Sensory nerves obtain signals from the mucosa when this homeostasis is breached; this may lead to pruritus and sneezing. Once this process continues, the threshold for response is lowered, leading to upregulation and hyperresponsiveness, which triggers higher levels of neuropeptide release and inflammation. Stimulation of the parasympathetic innervation leads to vasodilation, causing excretion of mucosal secretions and cell debris [32]. A cascade of neuropeptides facilitates this process. Acetylcholine is commonly known for its effect on preganglionic parasympathetic nerves, which act on nicotinic receptors [32]. The release of acetylcholine on postganglionic fibers acts on M1 and M3 muscarinic receptors, which leads to the release of other neuropeptides [32].

The sympathetic nervous system controls vasoconstriction and increased patency of the nasal passages [32]. Sympathetic nerve fibers originating from the thoracolumbar region are mediated by adrenoreceptors, which are stimulated by neurotransmitters such as norepinephrine and neuropeptide Y. Histamine released from mast cells can cause angioedema but can also inhibit the release of norepinephrine [32]. Interestingly, mast cell degranulation has also been implicated in the pathophysiology of migraine, especially relating to the sterile inflammation that triggers the release of neuropeptides, such as calcitonin gene-related peptide (CGRP), through the trigeminal pathways [33].

Lastly, the sphenopalatine ganglion is an anatomical crossroad and hub for many symptomatic pathways. Unilateral rhinorrhea and lacrimation are commonly seen in TACs through parasympathetic activation. The sphenopalatine ganglion has been identified as a target for blockage with local anesthetic for both facial pain and TACs [34].

Overall, sinus-related symptoms arise when neuropeptides modulate the junctions within the trigeminovascular system, mast cells release histamine, and sympathetic and parasympathetic autonomic neural pathways are activated.

### Further Complexity Regarding Sinus Symptoms and Headache

Facial pain with cranial autonomic symptoms, such as rhinorrhea or congestion, is often attributed to sinusitis or

**Table 2** Paranasal sinus characteristics

	Maxillary	Frontal	Ethmoid	Sphenoid
Anatomy	Pyramidal shape; the largest sinus (15 mL)	Thin bony lamella separates the 2 frontal triangular-shaped sinuses	Labyrinth lies within the ethmoid bone, housing air cells that vary in size and quantity (3–18 mL) [27]	Paired sinuses behind the nose and eyes
Bordering structures	Lateral wall is formed superiorly to the zygomatic process of the maxilla to the base of the nose, the roof is formed by the floor of the orbit, and the floor is formed by the alveolar process [28]	Extends above the medial end of the supraorbital crest and posterior into the medial orbit [27]	A thin division of bone in the orbital plate (lamina papyracea) separates the sinuses from the orbit [27]	Sphenoid bone embodies the sinus, posterior to other sinuses; above superior concha, each sinus connects to the sphenoidal recess [27]
Mucosal drainage	Into lateral nasal cavity [28]	Travels to the middle meatus of the nose via infundibulum	Drains into middle meatus and sphenoidal recess [27]	Drains through the sphenoid ostium [27]
Blood supply	Facial, infraorbital, and greater palatine arteries [29]	Supraorbital and anterior ethmoid arteries and supraorbital and superior ophthalmic veins [27]	ECA branching into anterior and posterior ethmoidal and sphenopalatine arteries, and ICA branching into ophthalmic artery and an ethmoid sinus artery, with corresponding venous drainage [27]	Posterior ethmoidal artery with corresponding venous drainage [30]
Innervation	Infraorbital nerve and anterior, middle, and posterior superior alveolar nerves [31]	Supraorbital nerve [30]	Sensory input from anterior and posterior ethmoid nerves; orbital branches of the SPG supply parasympathetic secretomotor innervation	Posterior ethmoid nerve with orbital branches also from SPG supplying parasympathetic secretomotor fiber [30]
Pain radiation	Below the orbit and into the upper teeth	Supraorbital [30]	Retro-nasal [27]	Deep occiput and retro-orbital [27]
Clinical fact	Most common sinus infection location	Infection triggers pressure sensation on the face and head [27]	Chronic infections lead to the formation of nasal polyps [27]	Patients rarely blow nose in the setting of sphenoid sinusitis [27]

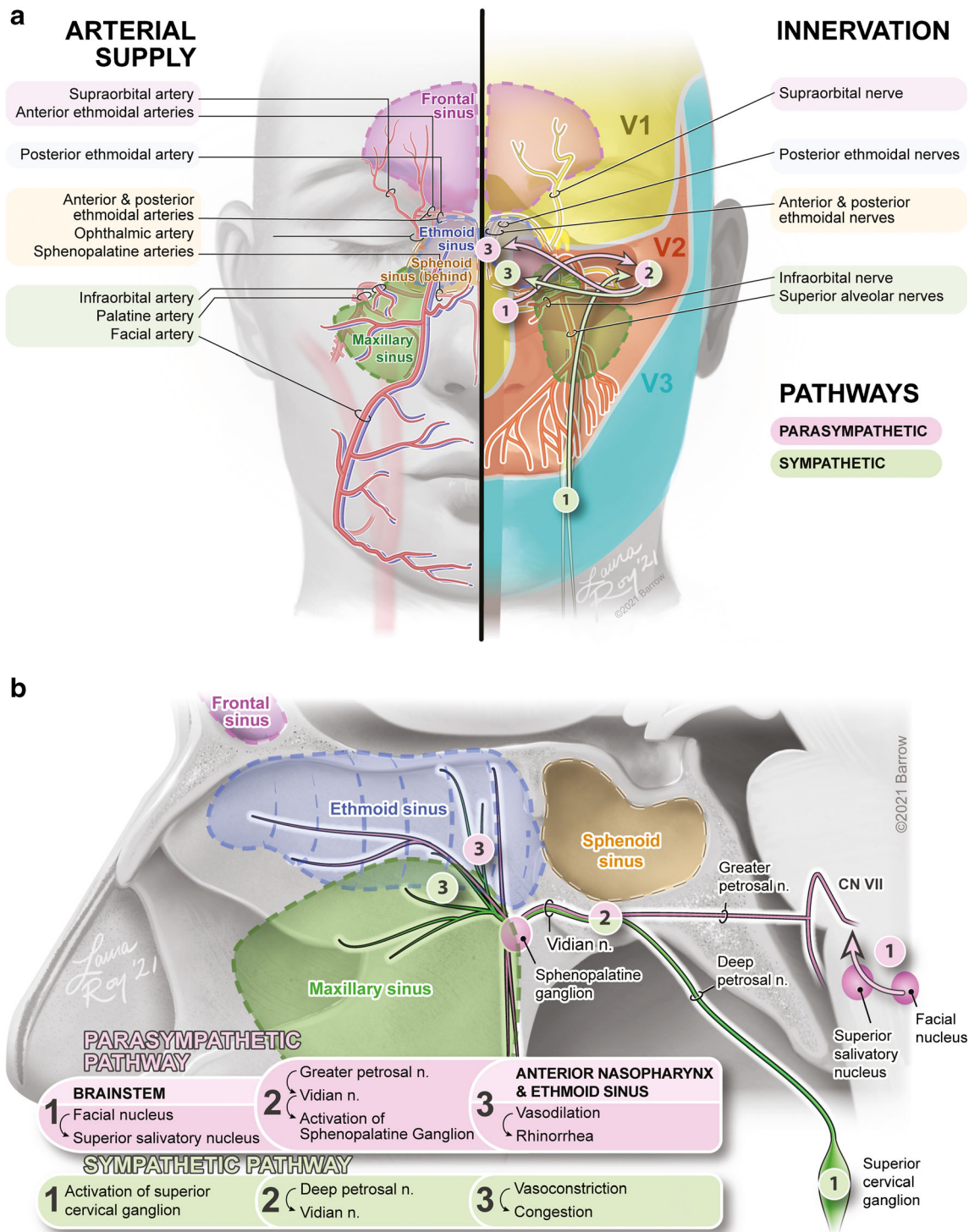
Abbreviations: *ECA*, external carotid artery; *ICA*, internal carotid artery; *SPG*, sphenopalatine ganglion

allergies by patients and healthcare professionals alike and is colloquially termed a sinus headache. We know that pain from RS overlaps pain from migraine due to the trigeminal innervation of the sinuses, as discussed earlier. As part of the trigeminovascular mechanism of migraine, many neuropeptides released from the trigeminal nerve endings, including CGRP, trigger these autonomic symptoms involving the nose and sinuses, likely in part through vasodilation [35]. Studies have confirmed that cranial autonomic symptoms are very common in migraine, ranging from 26 to 71.3% [36–39]. In fact, one study using endoscopy during and between migraine attacks demonstrated changes in total nasal resistance in addition to described nasal congestion [35]. In addition, facial pain is common in migraine, and rarely, migraine patients present with the pain location isolated to the face [40].

The neurology, otolaryngology, allergy, and primary care 2005 consensus statement emphasized that it is important to recognize that many patients with sinus headaches in fact have migraine, but causes of sinogenic headaches also exist [2]. Any patient presenting with a sinus headache, even if a sinogenic cause is found, should be screened for migraine.

Conversely, when prominent sinogenic symptoms are present with migraine, further evaluation is recommended to assess for otolaryngologic conditions [2]. If fever and purulent nasal discharge are present, the diagnosis is likely sinogenic [2]. Multiple studies have reviewed patients presenting with sinus headaches to clarify how often the underlying diagnosis is migraine, RS, or both. The Sinus, Allergy and Migraine Study in 2007 reviewed a population of 100 self-diagnosed sinus headache patients and found that 52% also had migraine, 23% had probable migraine, and 11% had chronic migraine [41]. Another study reviewed 211 patients in an otolaryngology clinic who presented with a sinus headache, and nearly half (48.8%) had a primary headache disorder [42]. On further review, otolaryngology referral was unnecessary in one-third. Interestingly, even among those patients with RS, primary headache management was beneficial in 15.7% [42]. The multisite CHEER network study evaluated 1485 otolaryngology patients (including all patients other than those with a diagnosis of head and neck cancer), and 16.1% screened positive for migraine, leading to the suggestion that all otolaryngology patients should be screened for migraine [43]. Repeatedly,





**Fig. 1 (A)** Arterial supply to sinuses and the trigeminal branches innervation of the sinuses with corresponding sympathetic (in green) and parasympathetic (in pink) pathways from the brainstem to peripherally. V1 = distribution of the ophthalmic branch of the trigeminal nerve; V2 = distribution of the maxillary branch of the trigeminal nerve; V3 = distribution of the mandibular branch of the

trigeminal nerve. **(B)** Arterial supply to sinuses and the trigeminal branches innervation of the sinuses with corresponding sympathetic (in green) and parasympathetic (in pink) pathway from the brainstem to peripherally in the sagittal section. Abbreviations: CN, cranial nerve; n., nerve. *Used with permission from Barrow Neurological Institute, Phoenix, Arizona*

other similar studies of sinus headache have found high rates of migraine as the underlying diagnosis, usually in more than 80% of cases [41, 44, 45]. Despite the high likelihood that sinus headache is in fact migraine, patients are repeatedly misdiagnosed with sinusitis, leading to a delay in migraine diagnosis and inappropriate treatment [44].

The gold standard for sinusitis diagnosis is sinus CT [46]. Although these CT sinus scans are fast and widely available, CT still involves radiation exposure, and findings are often negative. A sinus CT does appear to have a good correlation to findings during functional sinus endoscopic surgery; however, in those patients with CRS symptoms, the positive predictive value of CT was only 39.9% [47, 48]. If endoscopy was used, the positive predictive value improved to 66.0%, so the authors recommended relying on endoscopy over CT, given these results as well as cost and radiation exposure considerations [48]. Imaging studies such as sinus CT also are associated with a delayed diagnosis of migraine. One study of 500 patients who were referred to a headache clinic demonstrated that each diagnostic test or study was associated with a delay of more than 1 year in migraine diagnosis [44]. In addition to delaying a migraine diagnosis, a sinus CT will not rule out migraine. Another study of 26 sinus headache patients found that more than 75% had migraine but that the Lund-Mackay score for sinus abnormalities was not different between those with and without migraine [49]. Nasal endoscopy is likely sufficient to rule out CRS, with CT needed only in prolonged or complex cases [50, 51].

Both limiting radiation exposure and preventing a delay in diagnosis are important. We know from the Chronic Migraine Epidemiology and Outcomes study that less than 5% of patients with chronic migraine will cross all 3 identified barriers of seeing a healthcare provider for headache, getting an accurate diagnosis, and receiving appropriate treatment [52]. However, in these patients, it is not just receiving appropriate treatment that is important but also preventing unnecessary treatment, such as surgical procedures or antibiotic therapy. According to a Cochrane review, the number needed to treat for cure with antibiotics in ARS is 18, and the number needed to harm is 8 [53]. Because sinus headache is often attributed to ARS or CRS, patients are typically treated with antibiotics. Many patients with migraine are inappropriately receiving antibiotic therapy, and it is estimated that 1 in 8 may be harmed from antibiotic use. On the other hand, another study with 54 patients showed that more than 80% of patients with self-diagnosed sinus headaches responded to triptans, again supporting the concept that many have migraine and hence need migraine-directed treatment [54].

## Recommendations

In summary, a sinus headache is not a diagnosis. The consensus statement from otolaryngology, neurology, allergy, and primary care agrees that the majority of cases of sinus headaches are migraine [55]. Migraine is underdiagnosed, and delay in receiving proper migraine treatment is perpetuated by receiving multiple unnecessary tests. We argue that all patients presenting with facial pain or cranial autonomic symptoms such as rhinorrhea and congestion should be screened for migraine using ID Migraine and presence of osmophobia, including by otolaryngologists, primary care clinicians, and allergists. When a screen for migraine is positive, first-line preventive and acute treatment options should be offered to the patient or a referral made to neurology. Cranial autonomic symptoms are common in migraine and should not be ignored. Neurologists should examine patients for cranial autonomic symptoms and RS symptoms to determine whether RS is present. When true ARS or CRS is suspected, a referral can be made to an otolaryngologist for possible endoscopy, which the authors recommend over CT to limit radiation exposure. These patients may also benefit from an allergy assessment and treatment. For all healthcare professionals, it is important to recall that a patient can have concurrent migraine and RS, so they should be screened for both and treated when appropriate.

## Conclusion

Sinus headache is not a diagnosis. The use of the term is vague and leads to a high risk of incorrect management. When a patient presents with a sinus headache, healthcare professionals need to decide whether the patient has ARS, CRS, migraine, another diagnosis, or a combination of these conditions. Only with a clear diagnosis can appropriate treatment be provided. Treatments for these conditions do not overlap; hence, diagnosis matters. When a patient presents with facial pain, even with congestion or rhinorrhea, migraine should be one of the top differential diagnoses.

**Abbreviations** ARS, acute rhinosinusitis; CGRP, calcitonin gene-related peptide; CRS, chronic rhinosinusitis; CT, computed tomography; ICHD, *International Classification of Headache Disorders*; RS, rhinosinusitis; SNOT-22, 22-item Sino-Nasal Outcome Test; TACs, trigeminal autonomic cephalgias

**Acknowledgements** The authors thank the staff of Neuroscience Publications at Barrow Neurological Institute for assistance with manuscript preparation.

**Author Contribution** Robblee and Secora both performed the literature search and wrote the manuscript.

## Declarations

**Ethics Approval** Not applicable.

**Conflict of Interest** Dr. Robblee and Ms. Secora have nothing to disclose.

**Informed Consent** Not applicable.

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