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Review Article

Rhinogenic contact point headache – Frequently missed clinical entity

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ABSTRACT

Background: There are different anatomical situations inside the nasal cavity leading to rhinogenic contact point headache (RCPH), where each contact point has its own characteristics. The precise excision of contact points by endoscopic approach in patients with RCPH is very effective and could be done carefully in selected patients. This review presents an overview of the current aspects in pathophysiology, clinical profile, and management of RCPH.

Method: Relevant literature was searched from PubMed, Science direct, and Scopus databases.

Results: Headache is a common clinical entity and is nearly universal in the course of everyone's life. Pressure of two opposing mucosa in the nasal cavity without evidence of inflammation can be a cause of headache or facial pain. Minor intranasal anatomical variation leading to mucosal contact point may be an etiological factor for causing headache and often misdiagnosed and forgotten by clinician during evaluation of headache patients and sometimes considered as headache of unknown etiology.

1. Introduction

Headache is a common complaint by the patients in day-to-day clinical practice and creates a distressing situation for both patient and the physician. There are myriads of causes for headache varying from simple tension headache, migraine, refractory errors in eye, temperomandibular joint arthralgia, myofacial spasm to severe brain tumors. Headaches may be classified into primary and secondary types, where primary headache does not have specific etiology and include migraine, tension headache and cluster headache. Secondary

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Table 1 – Anatomic variations noted on diagnostic nasal endoscopy and CT scan anatomic variations.

Serial no.	Anatomical variations of nose
1	Deviated nasal septum
2	Septal spur
3	Concha bullosa
4	Hypertrophied superior turbinate
5	Overpneumatised ethmoidal bulla
6	Hypertrophied agger nasi cells
7	Malformed uncinate process
8	Paradoxical middle turbinate
9	Hypertrophied inferior turbinate

headache may arise owing to infections, trauma, tumor, vascular lesions, and metabolic diseases.[1] It needs a multidisciplinary approach to diagnose the causative factors for headache. Often the rhinogenic cause of headache is undiagnosed; even worse, this cause is not suspected on preliminary evaluation. Headache together with facial pain owing to nasal origin in the absence of inflammatory sinonasal pathology is a new clinical entity that has received attention in medicine. This is called as rhinogenic contact point headache (RCPH), which is a new terminology in medicine. Even without the presence of sinusitis, the referred headache often due to pressure on the nasal mucosa because of the anatomical variations in the nose.[2] Contact point headache is a new type of headache in the International Classification of Headache Disorders (ICHD), supported by limited evidence. RCPH is defined as intermittent pain localized in the periorbital and medial canthal or temporozygomatic regions; evidence of mucosal contact points with postural movements; cessation of headache within 5 min following topical use of local anesthesia at contact area and significantly resolution of headache in less than 7 days following removal of contact points.[3] Intranasal contact points denote to a contact between two opposing intranasal mucosal surfaces. Intranasal contact points are present in about 4% of noses.[4] Different intranasal anatomical variations causing RCPH are given in Table 1. Stammberger and Wolf documented the role of substance P (SP) in RCPH. They also described that this kind of headache is not only because of abnormal middle turbinate but also by abnormal mucosal contact causing referred pain.[5] This review article describes the role of anatomical variations in nose leading to headache, which is a prudent evaluation with diagnostic nasal endoscopy and computed tomography (CT) scan before accurate diagnosis of rhinogenic cause of headache. It also describes details of pathophysiology, clinical profile, and management.

2. Pathophysiology

The pathogenesis of RCPH is still the subject of controversy by some authors. The mechanical irritants such as pressure on the nasal mucosa may cause release of neuropeptides through the central orthodromic impulse and peripheral local, antidromic impulse. Neuropeptides like SP and calcitonin gene related peptide (CGRP) cause vasodilatation and edema of mucosal membrane, which again intensifies the pressure of contact area. The release of neuropeptides from central nervous system causes the pain sensation, which is almost similar to migraine without aura. The duration and onset of pain coincide with duration and beginning of the nasal cycle.[6] The middle turbinate is covered with mucosa on the lateral nasal wall. Its anterior wall and nasal septum are supplied by anterior ethmoidal nerve. RCPH is usually a referred pain where two different afferent sensory neurons, one with its receptor in the nasal cavity mucosa and other in the skin of forehead, zygomatic, temple and medial canthal area synapse on the same sensory neuron of sensory nucleus of trigeminal nerve. If the receptors in the nasal mucosa are stimulated, leading to the misinterpretation by the sensory cortex as originated from the skin, causing referred pain to the supraorbital or glabellar region. The cause of RCPH is multifactorial. RCPH may result from nociceptors in the nasal mucosa, which ends up in the sensory nucleus of the trigeminal nerve. Pressure effect on the nasal mucosa is associated with changes in micro vascular supply, followed by release of biologic substances, induces pain or decreasing the pain threshold. The contact between mucosal lining of concha bullosa and nasal septum or the lateral wall of nose results in release of SP, CGRP,[7] and neurokinin A.[8] These chemicals are found in nociceptive fibers in the central nervous system and trigeminovascular system. So the contact point between intranasal mucosa may be a cause of secondary headache or triggering factor to primary headache.[9] This phenomenon is also called as middle turbinate syndrome.[10] SP has a known role in pathophysiology of contact point headache.[5] SP is a neuropeptide that can be identified in the mucosa of the nasal cavity. When SP is released around vascular area, vasodilatation, plasma extravasation and perivascular inflammation, causing headache similar to clinical manifestations of migraine without aura.[9] Normal nasal mucosa has a higher concentration of SP than chronic hyper-plastic mucosa or polypoidal tissue. This explains why contact point headaches are almost always seen in patients without rhinosinusitis.

3. Clinical profile

Headache is a very commonly encountered clinical symptom seen in everyone's life. Facial pain and headache due to sinus and nasal origin in the absence of inflammatory sinonasal pathology is a clinical presentation which has received attention in both otorhinolaryngology and neurology. Different types of intranasal anatomical variations with mucosal contact points can lead to RCPH. The characteristic headache may be different in each type of intranasal anatomical variation. Many clinicians are not well versed with these types of clinical condition with headache. Intranasal mucosal contact headache was added as a secondary headache disorder in the ICHD.[11] Most relevant etiology concerned for otolaryngologists includes anatomical variations of nose causing secondary headache, which includes septal deviation, septal spur, and concha bullosa.[12] Wolf and Tosum et al. documented that nasal septal deviation and spur are causing referred headache in the absence of inflammation.[2] There are different types of septal deviations including cartilaginous deviation, bony deviation, bony spur, and high septal deviation. The significant RCPH is seen in septal spur (Fig. 1). Concha