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Chapter XIV

Contact Points and Migraine Headaches

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Abstract

Background: Migraine is a prevalent and disabling primary headache disorder. Contract point headache is a less known secondary headache disorder caused by pressure between intranasal structures. Herein, we explore intranasal contact points as a treatable factor that contributes to migraine intractability.

Method: We reviewed the literature on the anatomy of the sinonasal cavity and the pathophysiology of migraine and contact point headaches. We summarized our experience in the management of both diseases. We also propose a theory to explain the relationship between migraines and contact points.

Results/Conclusion: Migraine and intranasal contact points often occur independently. In a subset of patients, intranasal contact points are an important exacerbating factor in patients with migraine. During a migraine attack, CGRP and other mediators are released by the trigeminal nerve in the nasal mucosa. The consequent mucosal edema increases the pressure between the two opposing intranasal surfaces causing a self-perpetuating cycle. This may contribute to the development of medical refractoriness and Chronic Daily Headache (CDH). Surgical separation of contact points in carefully selected individuals with refractory migraine may improve or eliminate these headaches.

Introduction

In this chapter, we consider the interactions between migraine headache, a primary headache disorder, and intranasal contact points, a secondary headache disorder. Migraine is a prevalent and disabling primary headache disorder characterized by headache and associated symptoms.

In addition to their role as a potential cause of secondary headache, contact points between opposing surfaces in the nasal cavity are common in migraineurs who are refractory

to treatment. Some of them will benefit from releasing these contact points by modified endoscopic sinus surgery. In this chapter we will discuss the relationship between these conditions. We begin with a brief discussion of migraine pathophysiology. We then describe the contact point and its anatomy, followed by the role of contact points in these individuals. We then present a pathophysiologic theory linking these disorders, our approach to diagnosis and treatment and a few patients that illustrate the principles we discuss.

Pathophysiology of Migraine – Current Understanding

During the migraine attack, neural events are believed to result in dilatation and inflammation of meningeal blood vessels giving rise to the activation of trigeminal afferents which will cause migraine pain. Because neural events are linked to vascular events, migraine is considered a neurovascular headache disorder. The neural events that initiate the attack may be of brainstem or cortical origin. As the attack unfolds, pain results from a combination of altered perception (due to peripheral or central sensitization) of stimuli that are usually not painful, as well as the activation of a feed-forward neurovascular dilator mechanism in the first (ophthalmic) and second (maxillary) division of the trigeminal nervous system [1].

The sensory nuclei of the trigeminal nerve extends from the 2nd cervical segment of the spinal cord and upward towards the mesencephalon [2]. Brainstem sites responsible for craniovascular pain have been mapped using fos-immunohistochemistry. These studies reveal that the cells in the trigeminal nucleus caudalis are activated by meningeal blood and by stimulation of the superior sagittal sinus [3].

Activation of the trigeminal afferents leads to activation of cranial parasympathetics via a reflex connection to neurons in the superior salivatory nucleus. In turn, cranial parasympathetic outflow is mediated through the pterygopalatine, otic and carotid ganglia. This trigeminal autonomic reflex is present in a normal person and is responsible for tearing of the eye and running of the nose with strong stimulation in the distribution of the first division of the trigeminal nerve. The pathway is activated by human experimental pain (capsacin injection in the forehead), in the trigeminal autonomic cephalgias such as cluster headache and in migraine [4].

During the migraine attack, CGRP and other peptides are released at the peripheral ending of the trigeminocervical neurons giving rise to vasodilation, plasma protein extravasation, sterile inflammation and edema [5-6].

The trigeminal nerve also innervates the mucous membranes of the nasal cavity, oral cavity and all the paranasal sinuses [2]. During migraine attacks, the neuropeptides, including substance P (SP), and CGRP are also released at that level. The presence of substance P immunoreactivity in the nasal mucosa has been known for over 20 years [7]. Baumgarten highlighted the important potential functions of substance P stating that, “there is much evidence suggesting that neuropeptides play an important role in the pathogenesis of airway disease (nasal cavity is part of the airway system) and the diverse properties of SP include vasodilatation, vascular permeability, mucous secretion of leukocyte chemotaxis and mast cell degranulation” [8]. Using nasal endoscopy, migraine patients were shown to develop

swelling and edema of the nasal mucosa during migraine attacks, mucosal edema reversed following triptan therapy [9]. This observation is likely attributable to activation of the cranial parasympathetic pathways during migraine attacks and likely accounts for nasal stuffiness and other "sinus" symptoms in migraine.

There is evidence that for convergent input to the neurons in the trigeminal nucleus caudalis. In experimental animals the firing of neurons can be increased or decreased by stimulation of the upper cervical segments. In humans, problems in the upper cervical spine are thought to exacerbate migraine. Occipital nerve and upper cervical blocks are used to treat intractable migraine in the presence of such an exacerbating factor at the back of the head. We postulate that similar mechanisms may exacerbate migraine at the front of the head. In particular, the presence of an intranasal contact point may provide an anatomic basis for migraine intractability.

The Clinical Characterization of Contact Point Headaches

According to the ICHD-2, contact point headaches are defined as a specific headache disorder. The diagnostic criteria are:

a) Intermittent pain localized to the periorbital and medial canthal or temporozygomatic regions as well as fulfilling criteria C and D.
b) Clinical nasal endoscopic and/or CT imaging evidence of mucosal contact point 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 variation in mucosal congestion as patient moves between upright and recumbent postures,
    2. Abolition or reduction of pain within 5 minutes after diagnostic topical application of local anesthesia and decongestant to the contact point area, using placebo or other control

d) Pain Resolves within 7 days, and does not recur, after surgical removal of mucosal contact points [10].

Understanding the role of contact points as an exacerbating factor for migraine requires a brief review of the anatomy of the nasal cavity. The nasal septum divides the nasal cavity into right and left chambers. It consists of a perpendicular plate of the ethmoid, septal cartilage, and vomer. The roof the nasal cavity is formed by the nasal bone, the nasal process of frontal bone and the cribriform plate of the ethmoid bone, which transmits the filament of the olfactory nerve into the frontal fossa. Laterally, the nasal cavity is bounded by the superior turbinates, medial wall of ethmoid sinuses, middle turbinates, and inferior turbinates (figure 1). The innervation of anterosuperior part of the nasal cavity is from the first (ophthalmic)

division of trigeminal nerve while the second (maxillary) division supplies the posteroinferior part. The nasal mucosa and intranasal structures are very sensitive to pressure.

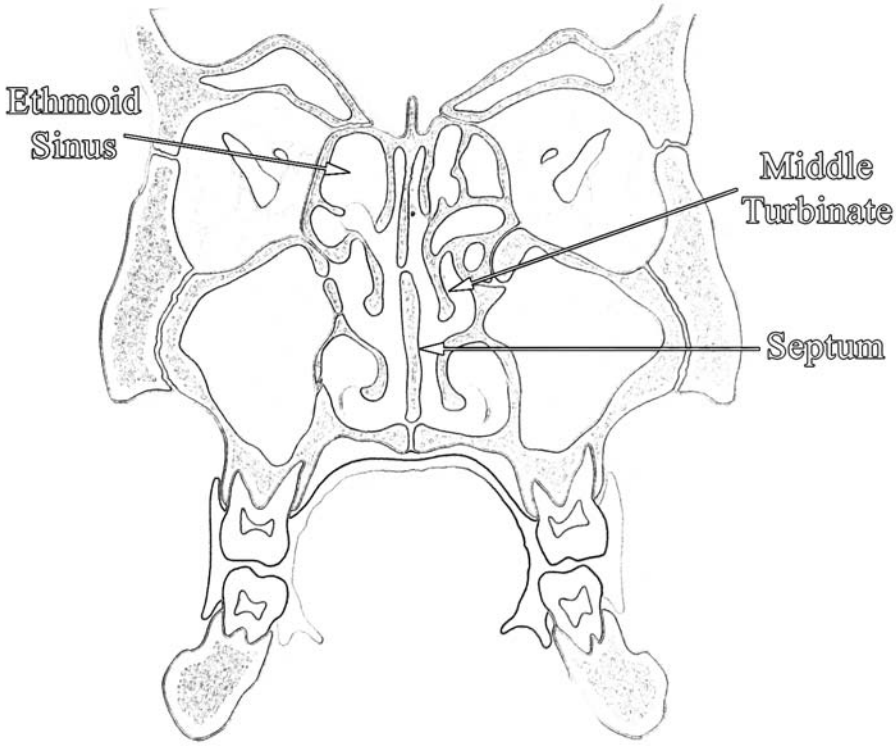

Figure 1. Normal anatomy of nasal cavity.

In figure 1 we can see the anatomy of the nasal cavity which shows the septum, superior turbinates, medial wall of the ethmoid sinuses and middle turbinates. Normally, there should be a space between the septum and the lateral wall of the nasal cavity (superior turbinates, ethmoid sinuses and middle turbinates).

Figure 2 illustrates a contact point between the septum and medial wall of the ethmoid sinus on the left side, septum and middle turbinate on the right side.

Contact point headache was described by Morgenstein and Krieger in 1980, as "The Middle Turbinate Headache Syndrome" [11]. In their original study they noted that, "headache, a most trying differential diagnosis, may be due to contact between the septum and middle turbinate, or bulla ethmoidalis, or the uncinate process." Surgical treatment to prevent the contact between the opposing surfaces, including the medial walls of the ethmoid sinuses and superior turbinates inside the nose, has been advocated as a treatment for the syndrome. Table 1 summarizes the results of published studies using this treatment [11-12-13-14-15-16-17-18-19-20-21]. These studies indicate, that contact point surgery provides striking benefits in some patients refractory to other treatments. The studies have several limitations. Criteria for patient selection varied and were not always well specified. In some studies, it is difficult to determine if patients had contact points as a cause of secondary headache or migraine exacerbated by contact points. They are uncontrolled surgical studies

and not randomized trials comparing actual and shame surgeries. Despite these limitations, the available evidence suggests that contact point surgery is a promising treatment.

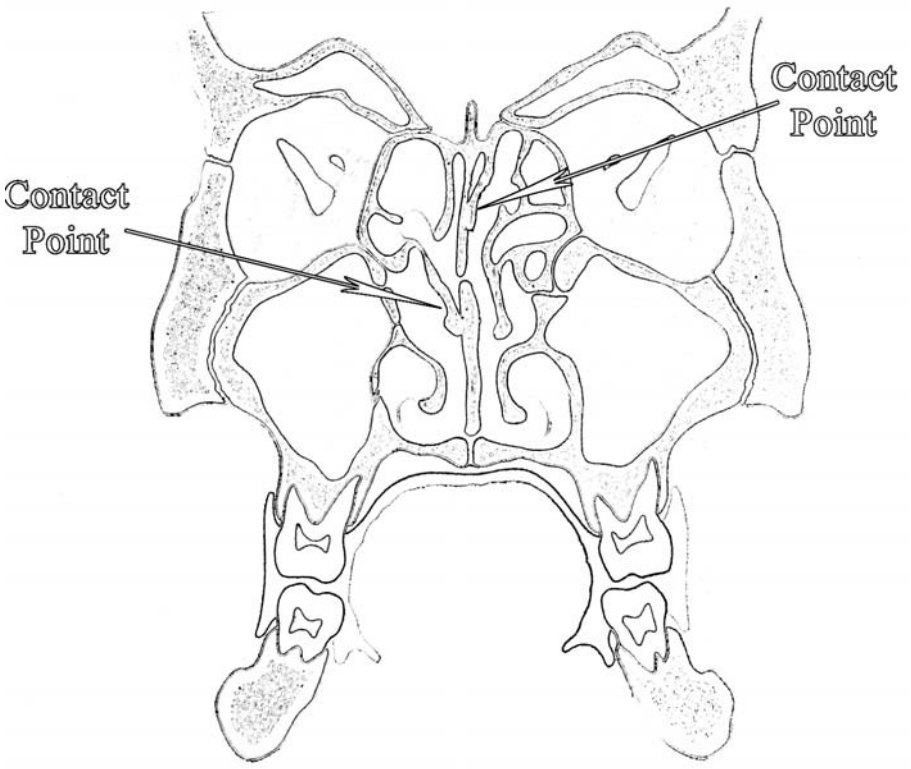

Figure 2. Contact point between the septum and middle turbinate on the right, and medial wall of the ethmoid sinuses on the left side.

Table 1. Result of modified sinus surgery for headaches from literature

Date	Authors	Total patients	Cured N (%)	Improved N (%)	Not improved N (%)
1980	Morgenstein Krieger	19	15 (78)	2 (10)	2 (10)
1993	Goldsmith et al	8	7 (87)	1 (13)	0 (0)
1994	Clerico	9	5 (55)	2 (13)	2 (22)
1994	Chow	13	5 (38)	5 (38)	3 (24)
1997	Anselmo- Lima	5	5 (100)	0 (0)	0 (0)
1998	Parsons	34	12 (35)	19 (55)	3 (8)
1999	Ramadan	15	0 (0)	9 (60)	6 (40)
2000	Tosun	30	13 (43)	14 (47)	3 (10)
2003	Harley	71	18 (25)	18 (25)	35 (50)
2003	Welge Luessen	15	4 (26)	5 (34)	6 (40)
	Average	219	84 (39)	75 (34)	60 (27)
2005	Behin et al	32	13 (41.5)	13 (41.5)	6 (19)

Explaining the Relationship between Migraine and Contact Points

In those patients who have migraine, we postulate that intranasal contact points are sometimes a powerful exacerbating factor. During a migraine attack, there is antidromic stimulation of all branches of the trigeminocervical system including intranasal branches. This leads to secretion of CGRP and other peptides (SP) in turn causing edema and swelling of the nasal mucosa. In the absence of contact points, this may lead to nasal stuffiness or rhinorrhea. In the presence of contact points, it may cause pressure on the nasal mucosa, and as the result more pain. In turn, the pain may lead to further release of neuropeptides causing more swelling and more pain which will create vicious cycle.

The role of allergic rhinitis and sinusitis as migraine triggers remain controversial. It seems likely that any condition that causes swelling of nasal mucosa may trigger migraine, particular in the setting of contact points. Sinus headache, tension type headache and migraine are often confused [22-23]. By separating the opposing surfaces in the nasal cavity in these patients and removing this factor from the cycle, the continuity of the cycle is disrupted. Although these patients will have some headaches, they will be much milder and less in duration and frequency.

Approach and Management of Contact Points

As with all headache disorders, a comprehensive history is the first step in making a firm diagnosis. In patients who otherwise meet criteria for migraine without aura or chronic migraine, if the headaches are intractable to standard medical therapy the diagnosis of contact point headache should be entertained. Diagnosis is based on radiographic evidence of contact points coupled by relief of headache with local anesthetic and decongestant solution applied to the involved area.

To evaluate a patient for contact points we recommend a CT scan of sinuses in coronal view with window: 3000 and level: 300. Figure 3 illustrates a normal CT scan of sinuses. The CT scans must be reviewed by the treating physician regarding the contact points. There are different areas that could host the contact points.

1. Contact between the septum and any part of middle turbinates (figure 4)
2. Contact between the septum and medial wall of ethmoid sinuses, anterior or posterior or both (figure 5).
3. Contact between the septum and superior turbinates (figure 4).
4. Contact between the septum and supreme turbinates.

If the CT scan is positive a treatment trial of local anesthetic and decongestant solution during headache is recommended. Prior to treatment an ENT exam and review of the CT is needed to identify the contact points. During the headache cotton pledgets soaked in a mixture of topical decongestant and anesthesia should be placed at the area of contact points. This process usually is done in multiple stages; first to devascularize the anterior part of the

nose, and second, to progress more accurately to the sensitive areas. If the patient has significant relief from the headache after five minutes of this procedure, then one can assume the surgery (most probably) will be beneficiary for the patient. At this point it is helpful while the nose is anesthetized to look at the contact points with endoscopy (figure 6). A few patients who met criteria did not improve with surgery.

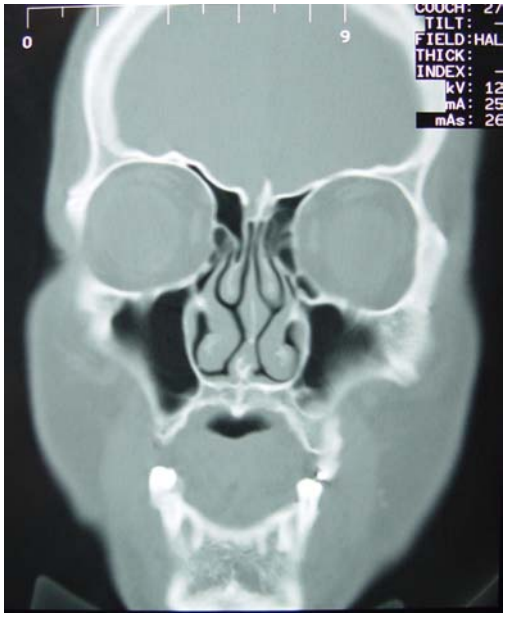

Figure 3. CT scan of a normal nasal cavity and sinuses without contact point.

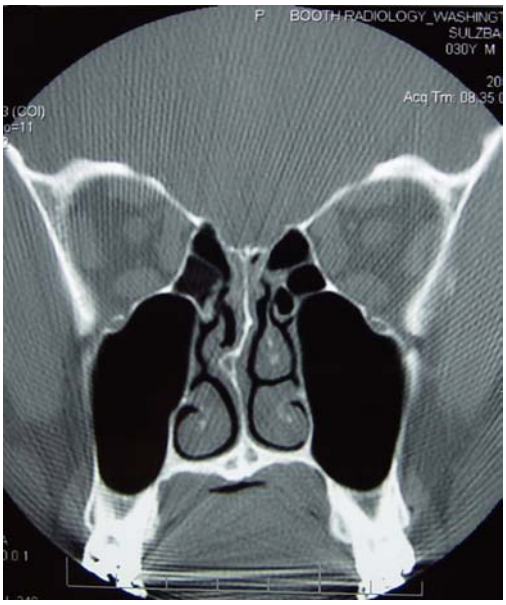

Figure 4. CT scan of contact point between the septum and middle turbinate on the right side and superior turbinate on both sides.

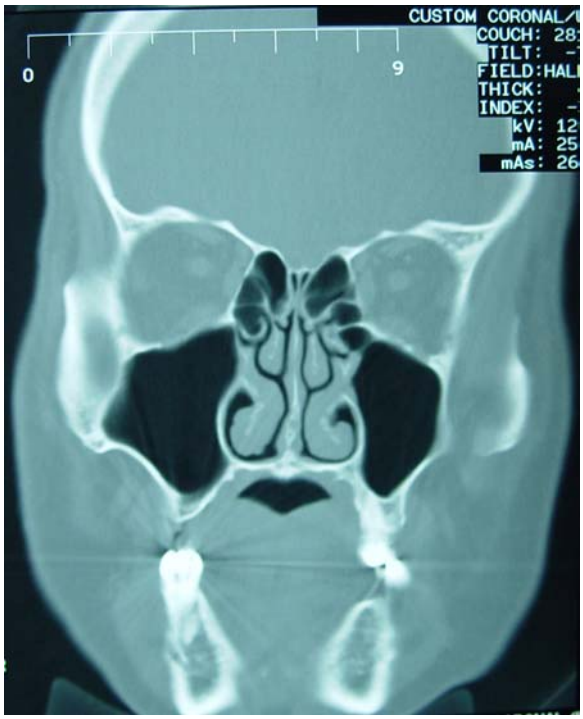

Figure 5. CT scan of intranasal contact point between the septum and medial wall of ethmoid sinuses in both sides.

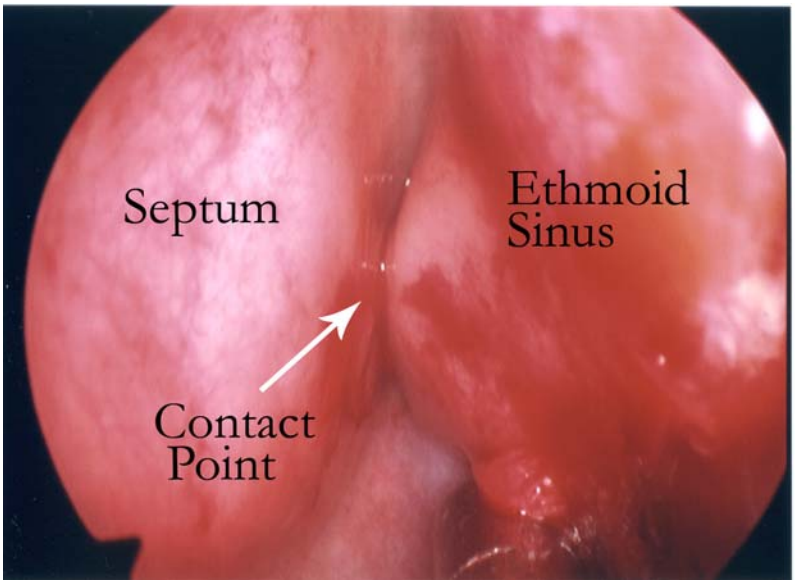

Figure 6. Endoscopic picture of a contact point between the septum and medial wall of the ethmoid sinuses.

Surgical Technique

This operation is usually performed under general anesthesia though in selected cases local anesthesia may be indicated. If patient's nasal mucosa is edematous and fragile, the patient should be treated with a short preoperative course of prednisone and antibiotic.

With the patient under general anesthesia, the nasal mucosa is decongested with the mixture of topical decongestant; at this point if the septal deviation is prohibiting direct access to the contact point areas, or the patient has difficulty in nasal breathing, a septoplasty or SMR is performed. Then the middle turbinate in one side is injected with Xylocaine 1% with adrenaline 1/100,000 followed by partial middle turbinectomy. After bleeding is controlled, ethmodectomy is carried on and the medial wall of ethmoid sinuses is removed. Special attention is made toward the areas of contact points by examining the CT scans of sinuses, as these areas are very crucial to the success of the operation (figures 7- 8).

This procedure is repeated on the other side. Extreme care should be exercised to avoid injury to the septal mucosa to prevent post operation adhesion between the septum and lateral wall of the nasal cavity. In a series of patients who developed adhesions the headache recurred. In certain cases the headache was eliminated by releasing the adhesions.

In some instances insertion of Gelfilm over the superior part of the septum may prevent this adhesion process. If septolasty is performed, intranasal stent is inserted and secured with sutures. Usually no packing is necessary.

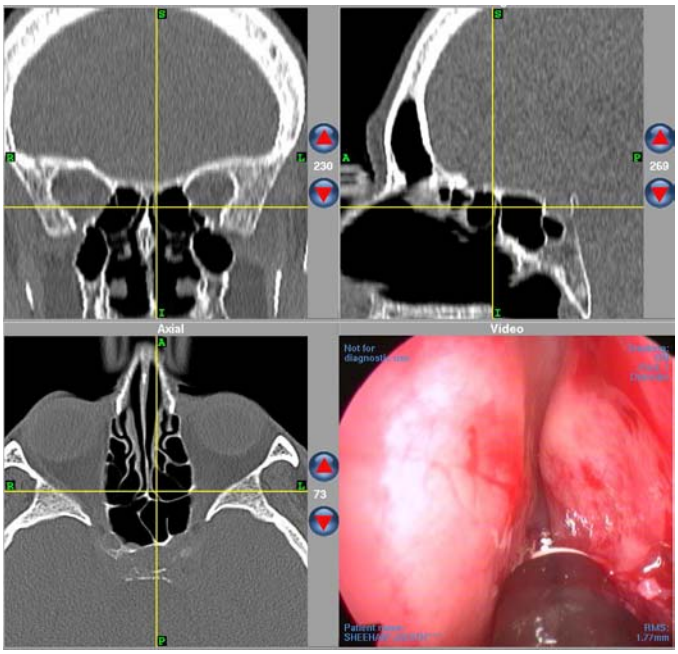

Figure 7. Endoscopic and radiologic view of contact point before the operation.

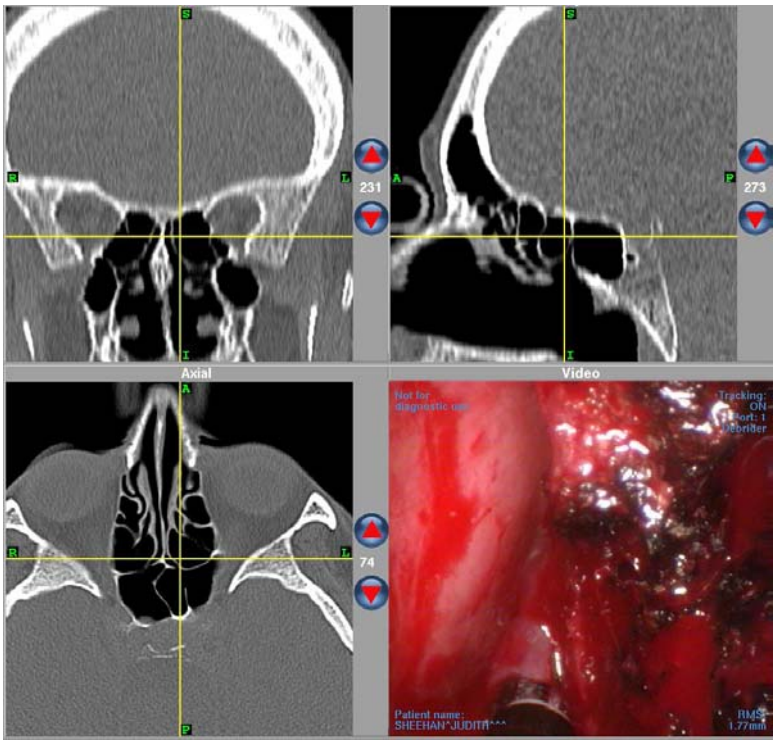

Figure 8. Endoscopic view of the same patient after removing the contact point.

Post Operation Course

After a few hours when patients are awake, stable and not bleeding, they usually are discharged on antibiotic and low dose of prednisone.

The patient usually is seen in the office the next day and the stent is removed; if there is a blood clot that is easily removable without damaging to the nasal mucosa, then it is removed and the patient is instructed to frequently spray the nose with a very diluted topical decongestant (2 cc of Afrin in 60 cc of normal saline solution). After one week the patient is asked to irrigate the nose with a solution of normal saline and sodium bicarbonate.

During post op visits an attempt is made to remove the crusting and debris from the nasal cavity; again one should be careful not to be too aggressive so there will be less damage to the mucosa. Sometimes if the crusting is large and hard it could cause a migraine headache. After removal the headache usually disappears.

Complications

Complications and risks of this operation are the same as for any endoscopic sinus surgery but with different frequencies.

1. CSF leak. The most frequent complication in our practice was CSF leak. Most of these leaks happened at the anterior most end of the middle turbinate attachment; although it also can happen at the posterior attachment or roof of the ethmoid sinuses. This complication is rare in routine sinus surgery due to the fact that the rout of the middle turbinates and medial wall of the ethmoid sinuses are not removed or manipulated in contrast to an operation in which the main part of the operation involves removal of the entire middle turbinates and medial wall of the ethmoid sinuses where the contact points are located. These walls should be removed with cutting forceps to prevent cribriform plate fracture. If CSF leak happens, a mental note should be taken of the area and a small piece of Surgicell is inserted at the area until the end of the procedure. To repair the CSF leak an adequate size of tissue from the middle turbinate preferably with a small piece of bone attached to it is used. The Surgicell is removed and the graft with raw surface and the small piece of bone facing the defect is inserted to cover the defect and kept in place with a few pieces of Gelfoam and Surgicell and covered with flowseal. During the post op period for at least two weeks, the area of the CSF leak is not manipulated and irrigation is avoided for the same time period. The crusting will come out when the graft is taken and the defect is healed; forceful removal should be avoided.
2. Anosmia. As with any other sinus surgery the patient will have anosmia after the operation but the sense of smell will come back slowly during the post op period. As we know the olfactory nerve fibers emerge from the cribriform plate and innervate the upper part of the septum, the rout of the middle turbinates and medial wall of the ethmoid sinuses. By removing the middle turbinates and medial wall of the ethmoid sinuses one has the risk of reducing the sense of smell, but in actuality it is very rare that the patient would notice this reduction. If the cribriform plate is intact and the septal mucosa is not damaged during the operation, either the sense of smell is not reduced or it is so minute that the patient will not notice or would not mind the sensation. Again we should emphasize the key to preventing this complication is to avoid damage to the septal mucosa.
3. Post op bleeding. Post op bleeding usually occurs very rarely it is often from the stump of the middle turbinate, and can happen up to three weeks following the surgery. This complication can be avoided by diligent cauterization of the middle turbinate stump when it is removed.
4. Retro-orbital bleeding. This complication is very rare as the surgical procedure usually does not involve the area of the ethmoid arteries or lamina papyracea. If this happened it should be treated accordingly. If the orbital pressure rises, lateral canthotomy should be performed immediately followed by medical treatment and ophthalmologic consultation.
5. Adhesions. As in endoscopic sinus surgery or septoplasty when there are two raw opposing surfaces close together, there is a possibility of adhesions between them, in these occasions the adhesion may act like contact points which either press against each

other or pull from each other and cause recurrence of the headaches (figure 9). We had some patients who experienced recurrence of their headaches after a short headache free period which responded to a second operation and removal of the adhesion. Sometimes at the end of operation insertion of Gelfilm between two opposing surfaces may prevent adhesion provided that the material not removed prematurely.

6. Chronic sinusitis. In very rare instances patient will continue to have mild infection with crusting and discharge. In these cases one should treat them as chronic sinusitis with culture and sensitivity, appropriate antibiotics and possibly a short period of steroid treatment.
7. Nasal polyposis. There was one occasion of recurrent nasal polyposis in a patient who did not have a history of nasal polyps. After many failed attempts to treat, culture and sensitivity revealed coagulase negative streptococcus, resistant to most medication, but with appropriate antibiotic and two weeks of low dose prednisone patient was cured. Sometimes irrigation or spray with appropriate antibiotic and steroid nasal spray is necessary to prevent nasal polyposis.

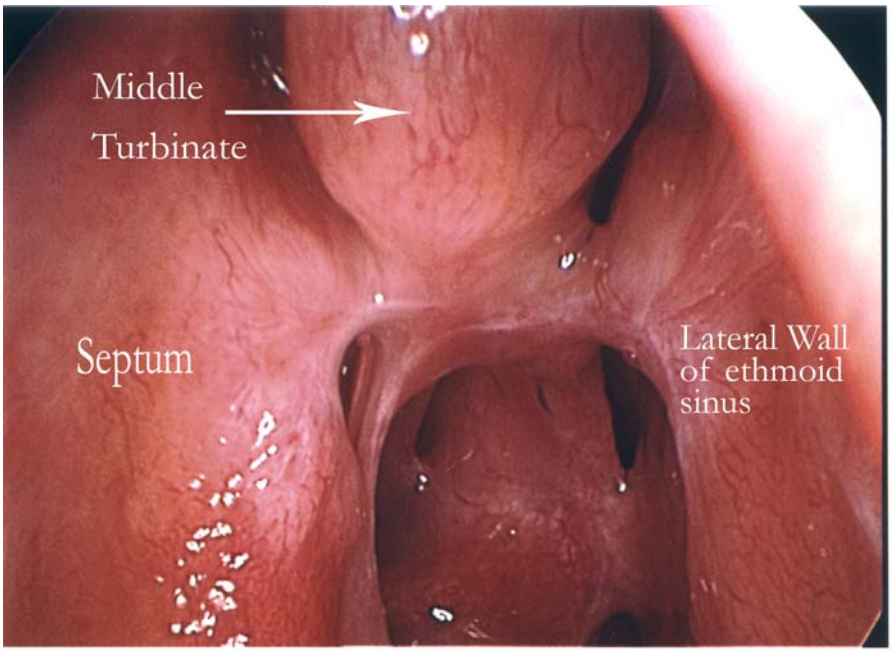

Figure 9. Endoscopic view of post operation adhesions between the septum, middle turbinate and remnant of ethmoid sinuses.

Case Studies

Patient # 1- A 35 year old female presented with a history of migraine headaches worsened following a closed head injury at the age of 17. Her headaches typically started as

frontal and occipital discomfort and would end up to severe headache with photophobia, phonophobia, osmophobia and nausea. She had occasional visual aura.

She was treated by multiple physicians including a neurologist and several otolaryngologists. She had numerous MRIs and CT scans. She tried and failed many acute and preventive migraine medications as well as frequent trigger point injections of head and neck. Her headaches remained quite disabling and prevent her from her activities of daily living. In the past there had been component of analgesic overuse, but she was not abusing analgesic at this time.

She was referred to one of us (FB). CT revealed contact point between the nasal septum and medial wall of the ethmoid sinuses in both side. Treatment of an attack with cotton pledget soaked in a mixture of anesthetic and decongestant resulted in a complete remission of symptoms within five minutes.

She underwent septoplasty, ethmoidectomy and removal of medial wall of the ethmoid sinuses in both sides on 7/24/2003. Since her surgery, she has been free of migraine headaches.

Patient # 2- This 54 year old woman presented with a 38 year history of migraine which occurred 10 to 12 times a month. She was diagnosed as migraine without aura by a prominent neurologist/headache specialist. Exacerbating factors included menses, alcoholic beverages, chocolate and odors. She reported nasal congestion with her otherwise typical headaches. Despite treatment trials with many acute and preventive treatments her headaches were disabling.

CT scans of her sinuses revealed bilateral contact point area. After insertion of cotton soaked in decongestant and anesthetic solution her headache was reduced from # 7 to # 3. On 12/19/05 she underwent septoplasty, bilateral middle turbinectomy and removal of medial wall of the ethmoid sinuses. During the post operative course whenever she developed crusting in her nose her headache came back and was cleared immediately after the crusting was removed. On one occasion about two weeks post operation she complained of constant pain on the left side of her head and behind the left eye; nasal endoscopy revealed adhesion formation on the left side between the septum and remnant of the ethmoid sinuses, which was released under local anesthesia and the headache disappeared immediately.

As recently as July of 2006 patient is not taking any medication. This case illustrates that in migraineurs with intranasal contact points, the pressure that is caused by the crusting inside the nose can and will translate to migraine headache, and when the crusting is removed and the pressure is released the headache is cured.

Patient 3- This 43 year old woman had a history of right sided headache for seven years; the attacks occurred 15 times a month and were characterized by severe pain, photophobia, phonophobia, and nausea. The headaches were worse during her menstrual period. She had been treated by several physicians including a neurologist. She did not do well with acute or preventive migraine medication. She was tried on Oxycontin which brought relief but lead to rebound. After some difficulty, she was detoxified.

Her CT scan of the sinuses revealed she had contact point on the right side of her nasal cavity. She underwent septoplasty, middle turbinectomy, ethmoidectomy and removal of medial wall of the ethmoid sinuses in both sides. Post operation she developed one episode of bleeding which was controlled in the operating room. Since the operation she gets only very

mild generalized headaches during her menstrual period which respond well to medication when required.

Conclusions

Migraine and intranasal contact points often occur independently. In a subset of patients, intranasal contact points appear to be making migraine worse and to lead to contribute to the development of chronic daily headache. The evidence suggests that during migraine attacks, edema of the nasal mucosa develops at least in a subset of patients. Quite likely, the edema is mediated by the release of CGRP and other mediators by the trigeminal nerve as it supplies the nasal mucosa. The consequent mucosal edema increases the pressure between the two opposing intranasal surfaces exacerbating pain and in some cases establishing a self-perpetuating cycle. The possibility of intranasal contact points should be evaluated in migraine patients refractory to medical therapy. Surgery should be limited to patients who have contact points on CT scan and who have a robust pain response to topical application of decongestants and topical anesthesia. Surgical separation of contact points in carefully selected individuals may improve or even eliminate the headaches.

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