Headache & faical pain

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Headache and facial pain are common complaints otolaryngologist evaluate in practice.

85% tension headache, 10% migraine & 5% are due to inflammatory disorders such as sinusitis.
# Headache disorders:


<table>
<thead>
<tr>
<th>Table 1</th>
<th>Primary and secondary headache disorders</th>
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</thead>
<tbody>
<tr>
<td><strong>Primary Headache</strong></td>
<td><strong>Secondary Headache</strong></td>
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<tr>
<td>Tension-type headache</td>
<td>Headache attributed to trauma or injury to the head or neck</td>
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<tr>
<td>Migraine</td>
<td>Headache attributed to cranial or cervical vascular disorder</td>
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<td>Trigeminal autonomic cephalalgias</td>
<td>Headache attributed to a substance or its withdrawal</td>
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<tr>
<td>Primary exercise headaches</td>
<td>Headache attributed to infection</td>
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<tr>
<td>Primary stabbing headaches</td>
<td>Headache attributed to disorder or homeostasis</td>
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<tr>
<td>New daily persistent headache</td>
<td>Headache attributed to psychiatric disorder</td>
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<tr>
<td>Hypnic headache</td>
<td>Painful cranial neuropathies and other facial pains</td>
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</tbody>
</table>
Tension Headaches

- Most common headache
  - Affects 80% of population
  - More common in women
  - Triggered by stress or anxiety
Tension Headaches

Headaches are bilateral, with a tightening/band-like sensation, in the frontotemporal region, radiates to occipital region and trapezius muscles.

- Onset is gradual, pain is non-throbbing and constant.

Pathophysiology: still poorly understood
Tension Headache

Subdivisions

- **Infrequent Episodic**
  
  (12 days/year)

- **Frequent Episodic**
  
  (> 12 and < 180 days/year)
Tension Headaches

Chronic

(>180 days/year)

(<12 days per year) and fulfilling criteria B-D

B. Lasting from **30 minutes to 7 days**

C. At **least two** of the following four characteristics:

1. bilateral location

2. pressing or tightening (non-pulsating) quality

3. mild or moderate intensity

4. not aggravated by routine physical activity such as walking or climbing stairs

D. **Both** of the following:

1. no nausea or vomiting

2. no more than one of photophobia or phonophobia
Treatment

Nonpharmacologic

- Reassurance, muscle relaxation, stress management, biofeedback, physical therapy with thermal modulation or electrical stimulation.

Pharmacological

Abortive

- Acetaminophen, ASA, caffeine, NSAIDs
- Should not be taken >2 days/week

Prophylactic

- Reserved for patients with frequent headaches >2/wk
- Amitriptyline-first line
- Topiramate, valproate, venlafaxine
Migraine Headaches

- Second most common form of headache
- Prevalence 10% of population
  - 18% in women, 6% in men
  - Peak age onset 20’s-30’s

Recurrent episodes of severe, throbbing, unilateral headaches

Associated Symptoms: Nausea, vomiting, phonophobia, phonophobia

Precipitating factors: Stress, lack of sleep, hormonal changes, diet, etc.
Cerebral vasospasm

1. Vessel Constriction
2. Vessel Dilation

Triggers:
- Event eg. Stress

Consequences:
- Reduced Blood Flow
- Aura
- Distended Blood Vessels
- Pain
Cortical Spreading Depression

- Flow of electrical impulses
- Inflamed blood vessel
- Electrical impulses
- Brain
- Pain centers located in brainstem
- Abnormally active brain cell
Table 1. ICHD III 1.1 Migraine without Aura

<p>| | |</p>
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>A.</td>
<td>At least five attacks fulfilling criteria B-D</td>
</tr>
<tr>
<td>B.</td>
<td>Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated)</td>
</tr>
<tr>
<td>C.</td>
<td>Headache has at least <strong>two</strong> of the following four characteristics:</td>
</tr>
<tr>
<td></td>
<td>1. unilateral location</td>
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<tr>
<td></td>
<td>2. pulsating quality</td>
</tr>
<tr>
<td></td>
<td>3. moderate or severe pain intensity</td>
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<tr>
<td></td>
<td>4. aggravation by or causing avoidance of routine physical activity (eg, walking or climbing stairs)</td>
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<tr>
<td>D.</td>
<td>During headache at least one of the following:</td>
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<tr>
<td></td>
<td>1. nausea and/or vomiting</td>
</tr>
<tr>
<td></td>
<td>2. photophobia and phonophobia</td>
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<tr>
<td>E.</td>
<td>Not better accounted for by another ICHD-III diagnosis.</td>
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</table>
Migraine with Typical Aura: 20%

Table 2. ICHD III 1.2.1 Migraine with Typical Aura

A. At least two attacks fulfilling criteria B and C
B. Aura consisting of visual, sensory and/or speech/language symptoms, each fully reversible, but **no** motor, brainstem or retinal symptoms
C. At least two of the following four characteristics:
   1. at least one aura symptom spreads gradually over ≥5 minutes, and/or two or more symptoms occur in succession
   2. each individual aura symptom lasts 5-60 minutes
   3. at least one aura symptom is unilateral
   4. the aura is accompanied, or followed within 60 minutes, by headache
D. Not better accounted for by another ICHD-III diagnosis, and transient ischemic attack has been excluded.
Vestibular Migraine

Table 3. ICHD III Appendix criteria for A1.6.5 Vestibular Migraine

A. At least five episodes fulfilling criteria C and D
B. A current or past history of 1.1 Migraine without aura or 1.2 Migraine with aura
C. Vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours
D. At least 50% of episodes are associated with at least one of the following three migrainous features:
   1. headache with at least two of the following four characteristics:
      1. a) unilateral location
      2. b) pulsating quality
      3. c) moderate or severe intensity
      4. d) aggravation by routine physical activity
   2. photophobia and phonophobia
   3. visual aura
E. Not better accounted for by another ICHD-III diagnosis or by another vestibular disorder.
Cerebral vasospasm

Trigger Event → Migraine vasospasm → AICA → Internal auditory artery → Vestibular branches → Vestibular dysfunction
Differential Diagnosis for VM

Migraine with Brainstem Aura Previously, known as Basilar Migraine

- Requires at least 2 “brainstem symptoms” therefore excludes patients with typical migraine accompanied only by vertigo.

Meniere’s Disease

- Vertigo lasting between 20 min-24 hours, tinnitus, unilateral, low frequency fluctuating hearing loss, not typical for VM
Differential Diagnosis for VM

- **BPPV**
  - Dix-hallpike (+)

VM can have positional vertigo, nystagmus is not fatigable
Migraine with Brainstem Aura

Table 4. ICHD III Migraine with Brainstem Aura

A. At least two attacks fulfilling criteria B-D
B. Aura consisting of visual, sensory and/or speech/language symptoms, each fully reversible, but no motor or retinal symptoms
C. At least **two** of the following brainstem symptoms:
   1. dysarthria
   2. vertigo
   3. tinnitus
   4. hypoacusis
   5. diplopia
   6. ataxia
   7. decreased level of consciousness
D. At least **two** of the following four characteristics:
   1. at least one aura symptom spreads gradually over ≥5 minutes, and/or two or more symptoms occur in succession
   2. each individual aura symptom lasts 5-60 minutes
   3. at least one aura symptom is unilateral
   4. the aura is accompanied, or followed within 60 minutes, by headache
E. Not better accounted for by another ICHD-III diagnosis, and transient ischemic attack has been excluded.
Migraine with Brainstem Aura

Previously “Basilar Type Migraine”.

have symptoms and/or signs suggestive of the posterior cerebral circulation such as bilateral visual symptoms, dysarthria, vertigo, hearing loss, diplopia, or ataxia.
Treatment

- Nonpharmacologic
  - Avoid triggers
  - Symptom Diary
  - Dietary modifications
  - Regularity in exercise, eating, sleeping

- Photophobia/Phonophobia
  - Lay down in a dark/quiet room
Food to AVOID !!

* Ripened cheeses (such as cheddar, Emmentaler, Stilton, Brie, and Camembert)
  - Chocolate
  - Marinated, pickled, or fermented food
  - Foods that contain nitrites or nitrates (bacon, hot dogs) or MSG (soy sauce, meat tenderizers, seasoned salt)
  - Sour cream• Nuts, peanut butter• Sourdough bread Broad beans, lima beans, fava beans, snow peas• Figs, raisins, papayas, avocados, red plums• Citrus fruits
  - Excessive amounts (more than 2 cups total) of caffeinated beverages such as tea, coffee, or cola
  - Alcohol (including red wine and beer)
Pharmacological: Abortive

**Triptans**\  **Ergot derivatives**

5-hydroxytryptamine, dopamine and noradrenaline receptors

Triptans more selective (5-hydroxytryptamine) receptors

If used >2d/wk can cause ergot-induced headache

S/E-nausea, angina

**Fioricet**

Butalbital, acetaminophen, caffeine (CNS DEPRESSOR, ANALGISC, VC)

**Fiorinal**

Butalbital, ASA, caffeine.
PROPHYLAXIS:

- Episodes >5/mo
- Antihypertensives
  - BB-CCB
- Antidepressants
  - TCA-amitriptyline
- Anticonvulsants
  - Gabapentin, valproic acid, topiramate
- NSAIDs
- BOTOX
Treatment (cont’d)

**Nausea and Vomiting/Suppression of Vestibular System**

- Antiemetics

- **Physical Therapy**
  - Effective for patients with VM
Cluster Headaches

Less common than migraine or tension headaches.

- Men>Women (3:1).

- Middle age.

Etiology unknown

- Circadian hormonal fluctuation:

  - Hypothalamic dysfunction

- Excitation of a nerve plexus in the carotid sheath and adventitia may increase trigeminal nerve discharge facial pain
Cluster Headaches

Diagnostic criteria:

A. At least 5 attacks fulfilling criteria B-D
B. Severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15-180 minutes if untreated
C. Headache is accompanied by at least one of the following:
   1. ipsilateral conjunctival injection and/or lacrimation
   2. ipsilateral nasal congestion and/or rhinorrhea
   3. ipsilateral eyelid oedema
   4. ipsilateral forehead and facial sweating
   5. ipsilateral miosis and/or ptosis
   6. a sense of restlessness or agitation
D. Attacks have a frequency from one every other day to 8 per day
E. Not attributed to another disorder
Cluster Headaches

- Remission can last months to years.

**Triggers**

- Alcohol, histamine, nitroglycerin, REM sleep, low oxygen saturation, OSA

- Patients with nocturnal attacks should get a sleep study to r/o OSA
Cluster Headaches: Treatment

- **Abortive**
  - Inhalation of 100% O2 x 10 minutes.
  - Triptans.
  - Dihydroergotamine
  - Intranasal 4% lidocaine may also be effective SPG.
Cluster Headaches

Prophylactic

until headache free for 2 weeks

Transitional prophylaxis: Used during a cluster period to suppress attacks

Prednisone, dexamethasone, ergotamine tartrate

Maintenance prophylaxis: Used before and throughout the duration of the cluster period or in anticipation of a cluster season

CCB-nifedipine, verapamil

Low-dose ergotamine

Lithium carbonate Methysergide

Refractory cases

Trigeminal nerve block and sphenopalatine ganglion block

Most effective is Radiofrequency ablation of trigeminal ganglion
Trigeminal Neuralgia

Most common in adults >50 y/o

- Women > Men (1.5:1)
- Incidence 4/100,000

- Etiology
  - Ignition Hypothesis of Devor

- A trigeminal nerve injury → induces physiological changes that lead to a population of hyper-excitabile and functionally linked trigeminal sensory neurons → quickly spread to activate the entire population, resulting in a sudden jolt of pain.
Trigeminal Neuralgia (ticdoulourex)

- Diagnostic criteria from International Headache Society

<table>
<thead>
<tr>
<th>Trigeminal neuralgia</th>
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<tbody>
<tr>
<td>A. Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, affecting one or more divisions of the trigeminal nerve and fulfilling criteria B and C</td>
</tr>
<tr>
<td>B. Pain has at least one of the following characteristics:</td>
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<tr>
<td>1. intense, sharp, superficial or stabbing</td>
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<tr>
<td>2. precipitated from trigger areas or by trigger factors</td>
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<tr>
<td>C. Attacks are stereotyped in the individual patient</td>
</tr>
<tr>
<td>D. There is no clinically evident neurological deficit</td>
</tr>
<tr>
<td>E. Not attributed to another disorder</td>
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</tbody>
</table>
Trigeminal Neuralgia

Diagnosis

- History & physical examination
- MRI of head → identification of neurovascular compression and can even measure the volume of neurovascular compression at CPA and can predict prognosis of initial

Treatment

LP:

- 2% of patients with TGN have MS.
- Infectious process - neurosyphilis, meningitis
Trigeminal Neuralgia

- **Treatment**
  - Education and Reassurance
  - Pharmacological treatment

- for at least 2 weeks - Carbamazepine (First line treatment)
  - Phenytoin, clonazepam, valproic acid, gabapentin, NSAIDs
  - Amitriptyline

- Opioids are ineffective and should be avoided
Surgical treatment

- Microvascular decompression
- Posterior cranial fossa approach
- Superior cerebellar artery most common
Surgical treatment

- **Trigeminal radiofrequency rhizotomy**
- A radiofrequency needle is inserted through the foramen ovale under the guidance of fluoroscopy.
Surgical treatment

- **Stereotactic radiosurgery/Gamma knife**
  - Currently recommended as 1st line noninvasive surgical technique
  - Median time for pain relief is 3 weeks

- **Percutaneous injection-nerve block**
  - Local anesthetic + steroid.
  - Ultrasound guided.
  - Immediate relief.
Sluder’s Neuralgia

- Symptom complex consisting of neuralgic, motor, sensory and gustatory manifestations attributed to the sphenopalatine ganglion.

- Lower half headache”

- Ipsilateral pain

- Duration: hours to days

- begins at the root of the nose, spreads ispilaterally in and around the eye, frontotemporal area, cheek and teeth, beneath the zygoma to the ear and mastoid.

- Pain is most severe at a point 5cm posterior to the mastoid.

- It can extend to the occiput, neck, shoulder.
Sluder’s Neuralgia

Sensory Signs

- Hyperesthesia along trigeminal nerve

Aura

- Metallic or peculiar acid sense of taste before or during an attack
Motor Signs

- Higher palatine arch on the affected side
- Uvula deviated to unaffected side
Sluder’s Neuralgia

- Parasympathetic Signs
  - Ipsilateral Lacrimation
  - Injected Conjunctiva
  - Nasal obstruction, inflamed nasal mucosa
  - Rhinorrhea
  - Increased salivation
Sluder’s/Cluster headache

**Similarities**

- Pain distribution
- Autonomic manifestations
- Precipitating factors

**Differences**

- Pain in cluster headaches are generally mediated by ophthalmic division of trigeminal nerve, in Sluder’s pain is usually Maxillary
Rhinogenic Headache

- Headache or facial pain secondary to mucosal contact points in the nasal cavity in the absence of inflammatory sinonasal disease.
- hyperplastic mucosa.
- septal deviation contacting nasal wall
- septum to middle turbinate
- septum to inferior turbinate
- concha bullosa
- superior turbinate pneumatization
Rhinogenic Headache

**Diagnostic criteria:**

A. Intermittent pain localized 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:**

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

**Comment:**

Der A11.5.1 Mucosal contact point headache is a new entry to the classification for which evidence is limited. Controlled trials are recommended to validate it using the listed criteria.
Mechanism:

- Contact point pain: stimulation of intranasal receptors that are innervated by afferent C fibers of V1 and V2

- Pressure → Substance P in nasal mucosa → vasodilation, plasma extravasation and perivascular inflammation, resulting in pain.
Patient management:

- A thorough history.
- Medication history.
- Physical examination.
- Radiographic studies may be reasonable if the diagnosis is unclear or if physical findings.
- Medical management is the mainstay of headache treatment.
- Surgical management.
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Headache in Otolaryngology: Rhinogenic and Beyond
Radiological finding & history?


- 973 patients referred for a sinus CT scan, the incidence of radiographic contact points was 4%, and did not differ among those patients with facial pain complaints (42% of the patients) and those who were pain free. Moreover, in patients with unilateral facial pain, the contact point was on the opposite side in 50%.
In office procedure as Treatment rhinogenic headache:


reported successful headache resolution in patients undergoing in-office middle turbinate lateralization. These 55 patients all had headache of endo-scopically proven origin based on anatomic findings and response to topical lidocaine. All had complete responses reported in a follow-up of 6 to 84 months, although 7 of them (13%) required a second procedure.

performed rhinologic evaluations in 99 patients with primary headache, 70 of whom had migraine. Seventy-three of the 99 were found to have nasal abnormalities, consisting of septal deviation, turbinate hypertrophy, contact points, and concha bullosa. Fifty-three patients of 99 were described as not responding to medical therapy, and 38 of these underwent nasal surgery. Headache severity based on visual analogue scales 3 and 6 months after surgery was significantly reduced.
THANK YOU