Headaches

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Primary Headaches (HA)

- Migraine
- Tension-type HA
- Cluster HA

Secondary Headaches

- HA attributed to the other causes eg. head and neck trauma, substance abuse, sinus, disorder of teeth or mouth, psychiatric disorder, infection

Other Headaches

- Cranial Neuralgias Central and Primary Facial Pain, Other Headaches
Migraines

- Migraine is a common, recurrent, severe HA that interferes with normal functioning.
- There are two major types of migraines:
  - Migraines with Aura
  - Migraines without Aura
Migraines: Pathophysiology and Etiology

- Vasodilation of intracranial blood vessels.
- Activation of trigeminal nerves.
- Release vasoactive neuropeptides.
- Central pain transmission (N/V, photophobia, phonophobia)

See more: http://www.youtube.com/watch?v=YZr9Joe85wg&feature=player_embedded

- Calcitonin gene-related peptide (CGRP)
- Neurokinin A
- Substance P
Trigger Factors for Migraine

Environment
- Heat/Cold
- Odors
- Bright light or glare
- Weather change
- Flying /high altitude
- Noise
- Motion
- Physical Strain

Lifestyle Habits
- Chronic high level of stress
- Disturbed sleep pattern
- Skipping meals
- Poor diet
- Smoking

Hormonal
- Puberty
- Menopause
- Menstruation
- Ovulation
- Pregnancy
- Using OC or estrogen therapy

Emotional
- Anxiety
- Depression

Diagnosis and treatment of Headache, ICSI, 2011
Phases of Migraines

1. Premonitory Phase
2. Attack Phase
3. Resolution Phase
Phases of Migraines

• **Premonitory phase**
  
  — Experienced by approximately 20-60% of migraineurs
  
  — Occurs in the hours or days before the onset of HA
  
  — Symptoms vary among people, but consistent within an individual

  • **Neurologic:** phonophobia, photophobia, difficulty concentrating
  
  • **Psychologic:** anxiety, depression, euphoria, irritability, drowsiness, hyperactivity
  
  • **Autonomic:** polyuria, diarrhea, constipation
  
  • **Constitutional:** stiff neck, yawning, thirst, food cravings, anorexia
Phases of Migraines

• **Attack phase**

  — Migraine with or without aura

  **Aura**

  — A complex of positive and negative neurologic symptoms

  — Begins before, at the onset of HA or during the attack.

  — Experienced by 31% of migraineurs on some occasions.

  — Typically evolves 5-20 mins and lasts <60 mins

  — HA usually occurs within 60 mins of the end of the aura.
Aura

• **Visual auras**
  
  — Positive: Scintillations, photopsia, teichopsia, fortification of spectrum
  
  — Negative: Scotoma, hemianopsia

• **Sensory and motor auras**
  
  — Paresthesias, numbness (arm and face), dysphasia, aphasia, weakness, hemiparesis
Symptoms of Migraines

• Recurring episodes of throbbing head pain
• Unilateral pain (frequently)
• Pain is usually gradual in onset and lasts 4-72 hrs
• Sometimes come with associated symptoms
  — N/V
  — Light/Sound sensitive
  — Movement sensitive
Phases of Migraines

- **Resolution phase**
  - Feeling tired, exhausted, irritable, impaired concentration
  - Scalp tenderness, mood changes
  - Refreshed or euphoric
History Gathering

- Age of onset
- Attack frequency, Timing, and Duration
- Precipitating factors/ameliorating factors
- Description of neurologic symptoms
- Characteristics of pain (quality, intensity, location, radiation)
- Associated signs and symptoms
- Treatment history
- Family and social history
- Impact of HA on daily lives
Migraines: Diagnostic Criteria

Migraine: with and without Aura

A. At least two of 1-4, plus one of 5 or 6:
   1. Unilateral location
   2. Pulsating/throbbling quality
   3. Moderate or severe intensity (inhibits or prohibits daily activities)
   4. Aggravation by routine activity
   5. Nausea and/or vomiting
   6. Photophobia and phonophobia

B. Aura criteria
   1. One or more fully reversible aura symptoms
   2. At least one aura symptom develops over more than 4 minutes or two or more symptoms occur in succession
   3. Symptoms do not last more than 60 minutes
   4. Attack follows within 60 minutes

C. Previous similar attacks

D. Organic disorder is ruled out by the initial evaluation or by diagnostic studies. If another disorder is present, the headaches should not have started in close temporal relationship to the disorder.
Treatment Goals of Migraines

- Decrease frequency (<5 times/mo.)
- Decrease severity (respond quickly to an therapy)
- Avoid medication/caffeine overuse HA
- (Prevent another episodes)

Approach to Treatment

- Non-pharmacologic interventions
- Pharmacologic interventions
  - Acute management: Specific and non-specific treatment
  - Prophylactic management

Migraine and Tension Headache, Group Health, 2011
Severity levels of Migraine

- **Mild**
  - Patient is aware of a HA
  - Able to continue daily routine with minimal alteration

- **Moderate**
  - HA inhibits daily activities but is not incapacitating

- **Severe**
  - HA is incapacitating

- **Status**
  - A severe HA that lasted > 72 hrs
Treatment Algorithm for Migraine HAs

Diagnosis of migraines

Pt education

Consider Prophylactic Pharmacotherapy

May pretreat with antiemetic agents

When N/V are severe, consider use of non-oral form (suppository, parenteral, intranasal formulations).

Mild-Moderate symptoms

Severe symptoms

Adapted from Dipiro et al. Pharmacotherapy, 2008: P 1011.
Treatment Algorithm for Migraine HAs

Mild-Moderate symptoms
- Simple analgesic
  - NSAIDs
Inadequate response
- Combination analgesics***

Severe symptoms
- Triptans
  - Inadequate response
- DHE or Ergotamine tartrate
  - Inadequate response
- Opioid combination analgesic

Adapted from Dipiro et al. Pharmacotherapy, 2008: P 1011.
Migraines: Approach for Treatment

Peptide release e.g. CGRP neurokinins triggered by nerve signals

Block peptides released in blood vessel wall

Inhibition of inflammatory factors by NSAIDs

Fluid leakage and sterile inflammation

Activation of 5HT1D receptors reduces the release of peptides

Vasoconstriction of blood vessels by stimulation of 5-HT1 receptors

Inhibition of pain centres in brain and brain stem

Decreased excitation of trigeminal nerve

Inhibition of nausea centres in brain and gut
Acute Migraine Therapies

Analgesics

• Acetaminophen
  — 1000 mg at onset; repeat q 4-6 hrs as needed

• Acetaminophen 250 mg/aspirin 250 mg/caffeine 65mg
  — 2 tablets at onset and q 6 hrs
Acute Migraine Therapies

**NSAIDs**

- **Ibuprofen**
  - 200-800 mg q 6 hrs

- **Naproxen Sodium**
  - 550-825 mg at onset, can repeat 220 mg in 3-4 hrs

- **Diclofenac**
  - 50-100 mg at onset; can repeat 50 mg in 8 hrs
Acute Migraine Therapies

Ergotamine tartrate

• Non-selective 5-HT1 receptor agonist
  • Constricts intracranial blood vessels
  • Inhibits development of neurologic inflammation in the trigeminovascular system.
• Also has α-adrenergic, β-adrenergic, and dopaminergic effects.
• Most effective when administered early in the migraine attack.
• Common ADRs: N/V, abdominal pain, weakness, diarrhea
• Severe ADRs: peripheral ischemia (ergotism), gangrene, MI.
Ergotamine Metabolism is reduced by CYP3A4 inhibitor

**Strong CYP3A4 Inhibitors**

- **protease inhibitors**: ritonavir, indinavir, nelfinavir, saquinavir
- **some macrolide**: clarithromycin, telithromycin
- **chloramphenicol**
- **some azole antifungals**: ketoconazole, itraconazole
- **nefazodone** (antidepressant)
Ergotamine Metabolism is reduced by CyP450 3A4 inhibitor

**Moderate CYP3A4 Inhibitors**

Some calcium channel blockers: verapamil, diltiazem

Some macrolide antibiotics: erythromycin

Someazole antifungals: fluconazole

Bergamottin (constituent of grapefruit juice)

**Weak CYP3A4 Inhibitors**

Cimetidine

Buprenorphine

Cafestol (in unfiltered coffee)
Ergotismo secundario a la asociación ergotamina-ritonavir. 
A propósito de 3 casos

SANTIAGO ACLE, FEDERICO ROCA, 
MARIELA VACAREZZA, ALFREDO ÁLVAREZ ROCHA

Ergotism secondary to ergotamine-ritonavir association. Report of three cases

Ergotism is a complication of the acute intoxication or chronic abuse of ergot alkaloids.
Ergotamine tartrate

- Oral tablet (1mg) with caffeine 100 mg
  - 2 mg at onset; then 1-2 mg every 30 mins as needed
  - Max dose is 6 mg/d or 10 mg/wk
- Sublingual tablet (2 mg)
- Rectal suppository (2 mg) with caffeine 100 mg
  - Insert ½ to 1 suppository at onset; repeat after 1 hr as needed
  - Max dose is 4 mg/d or 10 mg/wk
Contraindications of Ergot

- Pregnancy or nursing
- History of ischemic heart disease
- History of Prinzemtal’s angina
- Severe peripheral vascular disease
- Onset of pain following administration of test dose
- Within 24 hrs of receiving any triptan or ergot derivative
- Elevated or uncontrolled blood pressure
- Patients with hemiplegic or basilar-type migraines
- Cerebrovascular disease
Serotonin agonists (triptans)

- Sumatriptan, Zolmitriptan, Naratriptan, Flovatriptan, Eletriptan
  - Selective agonists of $5\text{-HT}_{1B}$ and $5\text{-HT}_{1D}$ receptors.
- Relief of HA is the result of 3 actions:
  - Direct vasoconstriction of intracranial arteries.
  - Reduction of nerve activation and peptide release
  - Decrease in pain neurotransmission
Acute Migraine Therapies

Serotonin agonists (triptans)

- Class effect ADR:
  - paresthesia, fatigue, dizziness, flushing, somnolence,
  - chest symptoms

- Contraindication:
  - A history of IHD (angina pectoris, Prinzmetal’s angina, or MI)
  - Uncontrolled HT
  - Cerebrovascular disease
Acute Migraine Therapies

Serotonin agonists (triptans)

- Special cautions:
  - Taken with SSRI or SNRI can be life-threatening
    - “serotonin syndrome”
  - Taken within two weeks of therapy with MAO-I
  - Taken within 24 h of ergot treatment
Acute Migraine Therapies

Serotonin agonists (triptans)

• Sumatriptan
  
  - Injection 6 mg sc at onset, can repeat after 1 hr if needed
  
  - Oral tablets 25, 50, 100 mg at onset, can repeat after 2 hrs if needed
  
  - Nasal spray 5, 10, 20 mg at onset, can repeat after 2 hrs if needed

• Zolmitriptan

  • Oral tablets 2.5 or 5 mg at onset as regular or orally disintegrating tablet, can repeat after 2 hrs if needed

  • Nasal Spray 5 mg (one spray) at onset; can repeat after 2 hrs if needed.

• Naratriptan

  • Oral 1 or 2.5 mg at onset, can repeat after 4 hrs if needed.
Acute Migraine Therapies

Opiate Analgesics

- Meperidine, butorphanol, oxycodone, hydromorphone
  - Effective but should be reserved for.....
    - Patients with moderate-severe infrequent HAs.
    - Patients in whom conventional therapies are contraindicated.
  - Used as “rescue medication” for patients who failed to respond to conventional therapies.
- Frequent use of narcotic analgesics can lead to dependency and rebound HA.
Acute Migraine Therapies

**Antiemetics**

- **Metoclopramide and prochlorperazine**
  - A single dose, administered 15-30 min before ingestion of oral abortive migraine medications.
  - Suppository preparations are available when N/V are particularly prominent.
  - Prochlorperazine: 10 mg IV or IM at onset
  - Metoclopramide: 10 mg IV at onset

- **Domperidone**
  - Has a possible role for preemptive treatment of migraine.
  - 10 mg orally 1 tablet at onset and repeat q 6 hrs
Medication-overuse Headache (MOH)

- So called “rebound headache”

- Frequent or excessive use of acute migraine medications can result in a pattern of increasing headache frequency and drug consumption.

- Can caused by
  - Analgesics: use of >15 d/months
  - Opiates, Ergotamine tartrate, Triptans: use of > 10 d/month

- Use of acute migraine therapy should limit to 2-3 days/wk to avoid the development of MOH.
Prophylactic Management

• Prophylactic therapy should be considered in the setting of
  
  — Recurring migraines that produces significant disability despite of acute therapy.
  
  — Frequent attacks requiring symptomatic medication >2 days/wk with the risk of developing MOH.
  
  — Symptomatic therapies that are ineffective, contraindicated or produce side effects.
  
  — Patient preference to limit the number of attacks.
Prophylactic Management

• Prophylactic therapy should be trial for 2-3 months to judge for efficacy.
• Drug should start with low dose and adjust slowly until therapeutic effect is achieved or SE becomes intolerable.
• Prophylactic therapy should continue for at least 3-6 months after frequency and severity of HA have decreased and then is tapered gradually and discontinued.
Prophylactic Management of Migraine HAs

- HA recurs in predictable pattern
- NSAIDs at the time of vulnerability
- Comorbid HT, angina, anxiety
- Comorbid depression or insomnia
- Comorbid seizure or manic depressive illness
- Other agent ineffective

Adapted from Dipiro et al. Pharmacotherapy, 2008: P 1011.
**AHS/AAN Migraine Prevention Guidelines**

**Level A: Established as effective**

— Should be offered to patients requiring migraine prophylaxis

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Divalproex/Sodium valproate</td>
<td>400-1000 mg/d</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>47.5-200 mg/d</td>
</tr>
<tr>
<td>Propranolol</td>
<td>120-240 mg/d</td>
</tr>
<tr>
<td>Timolol</td>
<td>10-15 mg bid</td>
</tr>
<tr>
<td>Topiramate</td>
<td>25-200 mg/d</td>
</tr>
</tbody>
</table>

Loder et al. Headache 2012, 52:933
## AHS/AAN Migraine Prevention Guidelines

**Level B: Probably effective**

- Should be considered for patients requiring migraine prophylaxis

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amitriptyline</td>
<td>25-150 mg/d</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>200 mg bid</td>
</tr>
<tr>
<td>Ketoprofen</td>
<td>50 mg tid</td>
</tr>
<tr>
<td>Naproxen</td>
<td>550 mg bid for Na form</td>
</tr>
<tr>
<td>Riboflavin</td>
<td>400 mg/d</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>150 mg extended release/d</td>
</tr>
<tr>
<td>Atenolol</td>
<td>100 mg/d</td>
</tr>
</tbody>
</table>

Loder et al. Headache 2012, 52:933
Prophylaxis agents

Beta-blockers

• ADRs
  — Drowsiness, depression, impotence, bradycardia, hypotension

• Cautions
  — CHF
  — Peripheral vascular disease
  — Atrioventricular conduction disturbances
  — Asthma
  — Depression
  — Diabetes
Prophylaxis agents

**Anti-depressants**

- Actions may be related to downregulation of central 5-HT2 and adrenergic receptors.
- ADRs
  - Anticholinergic effects
  - Sedation
  - Orthostatic hypotension
  - Slowed aterioventricular conduction
Anticonvulsants or Antiepileptic agents

- Actions may be related to ......
  - Enhancement of GABAergic inhibition
  - Inhibition of Na and Ca channel activity

- ADRs
  - Valproate: N/V, Alopecia, tremor, somnolence, weight gain
  - Topiramate: paresthesia, fatigue, anorexia, diarrhea, weight loss
Lifestyle Modifications/
Non-Pharmacologic Options

• Maintain a healthy lifestyle
  — Proper nutrition
  — Regular physical activity
  — Adequate sleep
  — Stress reduction strategies

• Identify and avoid triggers (smoke, strong odors, or sprays)

• Address workplace ergonomics/self-care of neck tension

Migraine and Tension Headache, Group Health, 2011
Tension-type Headache

- Prevalence: 63% in Men and 86% in women
- Onset: typically early in life (<20 yrs in 40% of patients)
- Frequency: mean 2.9 days/mo.
Tension-type HA: Pathophysiology

• The pain of episodic tension-type HA is thought to originate from myofascial factors and peripheral sensitization of nociceptors.

• Etiology: A local myofascial release of irritants

Triggers:

- Mental/Physical stress
- High caffeine intake
- Irregular sleep/diet
- Menstruation and Hormonal substitution
Pathophysiology of Tension-Type Headache

Tension-type HA: Diagnosis

A. Headache less than 15 days per month.
B. Lasts 30 minutes to 7 days
C. At least two of the following characteristics:
   1. Pressing/tightening (non-pulsating) quality
   2. Mild to moderate intensity (may inhibit, but does not prohibit activities)
   3. Bilateral location
   4. Not aggravated by routine physical activity
D. Both of the following:
   1. No nausea or vomiting (anorexia may occur)
   2. Photophobia and phonophobia are absent, or only one of the two is present
E. Organic disorder is ruled out by the initial evaluation or by diagnostic studies. If another disorder is present, the headaches should not have started in close temporal relationship to the disorder.
Targets for Prevention of TTH

- Tricyclics
  - Central nervous system
    - Coping with stress
    - Depressive mood
    - Central dysnociception
    - Central sensitisation
  - Peripheral nervous system
    - Muscle strain
    - Myofascial factors
    - Peripheral sensitisation
- Stress management
- Relaxation therapy
- Acupuncture?
- New drugs?
- Physical therapy

Tension-type HA: Treatment

Nonpharmacologic therapy

**Psychophysiologic therapy**
- Relaxation training
- Meditation
- Stress management program

**Physical therapy**
- heat/cold packs
- acupuncture
- ultrasound
- exercise
- massage
Tension-type HA: Treatment

### Pharmacologic therapy

<table>
<thead>
<tr>
<th>Simple Analgesics</th>
<th>NSAIDs</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Acetaminophen</td>
<td>• Ibuprofen 200-400 mg</td>
</tr>
<tr>
<td>1000 mg</td>
<td>• Naproxen Na 375-550 mg</td>
</tr>
</tbody>
</table>

- Medication should be taken for episodic tension-type HA no more than 2 days/wk
Tension-type HA: Prevention

Preventive should be considered if......

Frequency of HA: > 2 / wk
Duration: > 3-4 hrs
Severity: results in medication overuse or marked disability

- The principles of preventive therapy for tension-type HA are similar to those for migraine HA.
- TCAs are used most often for prophylaxis.
- Botulinum toxin has been reported to have efficacy in the prophylaxis of chronic tension-headache.
Cluster Headache

- The most severe of the primary HA disorders.
- Men are more likely than women to have cluster HA.
- Onset: generally > 20 years of age
- Characterized by severe attacks of HA which suddenly occur.
- Premonitory symptoms and aura are absent.
Cluster Headache: Pathophysiology

- Not completely understood.
- May cause from a pathogenesis of hypothalamic activation, resulting in alterations in circadian rhythms.
Cluster Headache

A. Severe unilateral orbital, supraorbital and/or temporal pain lasting 15 to 180 minutes untreated
B. Attack is associated with at least one of the following signs on the side of the pain:
   1. Conjunctival injection
   2. Lacrimation
   3. Nasal congestion
   4. Rhinorrhea
   5. Forehead and facial swelling
   6. Miosis
   7. Ptosis
   8. Eyelid edema
   9. Agitation, unable to lie down
C. Frequency from one every other day to eight per day
D. Organic disorder is ruled out by the initial evaluation or by diagnostic studies. If another disorder is present, the headaches should not have started in close temporal relationship to the disorder.
Abortive therapy

• Oxygen
  — 100% oxygen at a rate of 7-10 L/min for 15-20 mins

• Ergotamine Derivatives
  — IV DHE at onset and continue for 3-7 days
  — Rectally and sublingually: similar to those for migraine therapy

• Triptans
  — SC 6 mg is most effective
  — Intranasal is well tolerated.
  — Oral form is not recommended due to slow onset.
Cluster Headache: Treatment

Prophylactic therapy

• Verapamil
  - 360-720 mg/d
  - Effect often appears after 1 week of therapy

• Lithium
  - 600-1200 mg/d
  - Cautions: renal or CVD, dehydration, pregnancy, or concomitant diuretic or NSAIDs use.

• Ergotamine
  - 2 mg at bedtime
# Identification of HA type

<table>
<thead>
<tr>
<th></th>
<th>Migraine</th>
<th>Tension</th>
<th>Cluster</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>Unilateral</td>
<td>Bilateral</td>
<td>Supraorbital/temporal</td>
</tr>
<tr>
<td>Pain intensity (^1)</td>
<td>Moderate to severe</td>
<td>Mild to moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>Duration</td>
<td>4–72 hours</td>
<td>30 minutes to 7 days</td>
<td>15–180 minutes</td>
</tr>
<tr>
<td>Characterization of pain</td>
<td>Pulsing</td>
<td>Pressure/squeezing</td>
<td>Boring/stabbing</td>
</tr>
<tr>
<td>Sensitivity to light/sound</td>
<td>One or both may be present.</td>
<td>Both are absent or only one is present.</td>
<td>No</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>One or both may be present.</td>
<td>No</td>
<td>One or both may be present.</td>
</tr>
<tr>
<td>Aggravated by routine activity</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Aura</td>
<td>May be present</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Associated symptoms</td>
<td>None</td>
<td>None</td>
<td>Miosis, ptosis, rhinorrhea</td>
</tr>
</tbody>
</table>

\(^1\) Pain intensity
- Mild—Patient is aware of a headache, but is able to continue daily routine with minimum alterations.
- Moderate—The headache inhibits daily activities, but is not incapacitating.
- Severe—The headache is incapacitating.
### Warning signs for other possible disorders

<table>
<thead>
<tr>
<th>Signs/symptoms</th>
<th>Alternative diagnoses</th>
<th>Testing/investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subacute and/or progressive headaches that worsen over time (weeks to months)</td>
<td>Intracranial head lesion</td>
<td>• MRI with or without contrast</td>
</tr>
<tr>
<td>• A new or different headache in patients with established headache disorders</td>
<td>• Tumor</td>
<td></td>
</tr>
<tr>
<td>• Statement by the patient that “This is the worst headache of my life.”</td>
<td>• Subdural hematoma</td>
<td></td>
</tr>
<tr>
<td>• Headache of sudden onset (e.g., like a thunder clap)</td>
<td>• Hydrocephalus (acute or obstructive)</td>
<td></td>
</tr>
<tr>
<td>• Vasculopathy</td>
<td>• Subarachnoid hemorrhage</td>
<td>• CT scan without contrast</td>
</tr>
<tr>
<td>• Venous sinus thrombosis</td>
<td>• Carotid dissection</td>
<td>• If there is no evidence of subarachnoid hemorrhage, a lumbar puncture should be performed.</td>
</tr>
<tr>
<td>• Infection</td>
<td>• Bacterial meningitis</td>
<td>• If both tests are normal and suspicion is still high, order an MRI with or without contrast (i.e., gadolinium).</td>
</tr>
<tr>
<td>Structural defect</td>
<td>• Spontaneous cerebral spinal fluid leak</td>
<td>• Discuss next steps with Neurology.</td>
</tr>
<tr>
<td>• Red eye</td>
<td>Angle closure glaucoma</td>
<td>Acute angle closure glaucoma is an ophthalmological emergency.</td>
</tr>
<tr>
<td>• Halos</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Unilateral visual symptoms</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Warning signs for other possible disorders

<table>
<thead>
<tr>
<th>Symptom Category</th>
<th>Condition</th>
<th>Signs/Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age 50 or older</td>
<td>Giant cell arteritis</td>
<td>- Elevated sedimentation rate</td>
</tr>
<tr>
<td>and symptoms</td>
<td></td>
<td>- C-reactive protein</td>
</tr>
<tr>
<td>including:</td>
<td></td>
<td>- Consider brain imaging if associated with focal neurologic findings.¹</td>
</tr>
<tr>
<td>• Polymyalgia</td>
<td></td>
<td>- Discuss next steps with rheumatology</td>
</tr>
<tr>
<td>rheumatica</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Jaw claudication</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Scalp tenderness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Fever</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Firm, nodular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>temporal arteries</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Decreased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>temporal pulses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rhinosinusitis</td>
<td>Acute bacterial rhinosinusitis</td>
<td>- Non-contrast CT is indicated in patients with clinical signs or symptoms of</td>
</tr>
<tr>
<td>symptoms</td>
<td></td>
<td>complicated acute bacterial rhinosinusitis, including:</td>
</tr>
<tr>
<td>lasting</td>
<td></td>
<td>- diminished visual acuity</td>
</tr>
<tr>
<td>7 days or</td>
<td></td>
<td>- diplopia</td>
</tr>
<tr>
<td>longer and</td>
<td></td>
<td>- peri orbital edema</td>
</tr>
<tr>
<td>any of the</td>
<td></td>
<td>- severe headache, or altered mental status</td>
</tr>
<tr>
<td>following:</td>
<td></td>
<td>- Non-contrast CT may also be helpful in recurrent or treatment-resistant</td>
</tr>
<tr>
<td>• Yellow-green</td>
<td></td>
<td>sinusitis to help identify anatomic blockage of the ostiomeatal complex.</td>
</tr>
<tr>
<td>or blood-tinged</td>
<td></td>
<td></td>
</tr>
<tr>
<td>nasal discharge</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Pain, pressure,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>and fullness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>in cheeks,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>brow, or</td>
<td></td>
<td></td>
</tr>
<tr>
<td>forehead,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>especially</td>
<td></td>
<td></td>
</tr>
<tr>
<td>unilateral</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Unilateral</td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td></td>
<td></td>
</tr>
<tr>
<td>sinus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>tenderness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Worsening</td>
<td></td>
<td></td>
</tr>
<tr>
<td>symptoms after</td>
<td></td>
<td></td>
</tr>
<tr>
<td>initial</td>
<td></td>
<td></td>
</tr>
<tr>
<td>improvement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Fever</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Sore throat</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Cough</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Fatigue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Achy feeling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>in upper teeth</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

¹Migraine and Tension Headache, Group Health, 2011
### Warning signs for other possible disorders

<table>
<thead>
<tr>
<th>Temporomandibular joint (TMJ) disorder</th>
<th>History</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaw soreness</td>
<td>Exam</td>
</tr>
<tr>
<td>Pain radiating from the jaw</td>
<td>Consider dental referral.</td>
</tr>
<tr>
<td>Waking with headache</td>
<td>Consider bite splint/night guard.</td>
</tr>
<tr>
<td>Ear pain</td>
<td></td>
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<tr>
<td>Hyperacusis</td>
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<tr>
<td>Tinnitus</td>
<td></td>
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<tr>
<td>Palpable or audible joint click as the jaw is opened and closed</td>
<td></td>
</tr>
</tbody>
</table>

1. In most cases a non-contrast CT is the best initial test. Contrast-enhanced CT or MRI might be appropriate in persons with a rapidly accelerating course, a recent history of head injury, or focal neurologic findings.
Common Locations of Headache and Pain

**Headaches**

- **Sinus:** pain is usually behind the forehead and/or cheekbones
- **Cluster:** pain is in and around one eye
- **Tension:** pain is like a band squeezing the head
- **Migraine:** pain, nausea and visual changes are typical of classic form

[Images of headache locations]