اپروچ به سردرد در خانم ۳۱ ساله باردار

ارایه دهنده : آرین فرامرزی

استاد راهنما: دکتر شفیعی

Patient is a 31 year old pregnant woman. Patient presents to family Medicine clinic, complaining of generalized mid-thoracic and upper cervical pain. Patient reports no recent falls or trauma. However, she is four months pregnant and believes that the additional pregnancy weight is pulling her upper thoracic spine and shoulders down. When questioned about her pain, the patient reports that by the third month of her pregnancy, she started to notice the achy, sore pain when she would be sitting at her desk at work. The patient does not believe any activities make the pain worse, but sitting upright in her desk chair at home provides some relief. In addition, she is fearful of using ibuprofen because of the effects it may have on her unborn child, so she uses a heating pack at night to decrease her symptoms.

The patient was questioned further about the nature of her work and workspace. Currently, the patient is the marketing director of a large business. She works long, stressful hours at her desk. The patient describes how her chair does not accommodate her height well, so often she finds herself leaning forward a lot throughout the day in order to get close enough to the monitor and rest her feet on the ground. Recently, the patient has been increasingly stressed at work because she is behind schedule with multiple upcoming project deadlines. The therapist asks the patient if she has noticed any other changes lately that could be increasing the stress.

The patient reports that in the past few weeks, she has also started to experience severe throbbing headaches on the right side of her forehead and above the ear. Soon after the headache begins, she finds that she becomes very nauseous and occasionally gets dizzy. When asked about the nature of the dizziness, the patient reports that it lasts only as long as the headache, and does not increase with changes in position. The patient believes that her pregnancy has been causing the nausea, but she is concerned with the headaches because they are often so strong and painful that she cannot get any work done. When questioned further, the patient goes on to describe how the sunlight from her large window and staring at her screen makes the headache much worse, and sometimes even causes some ringing in her ears. In addition, the patient believes the glare on her computer may be the cause of her headache because she starts to see bright spots on the screen prior to the onset of the headache. Often, the pain is so extreme that she must leave work early to go home and rest. Normally, after sleeping an hour or two, the pain subsides. The patient is distressed because the prolonged back and neck pain, with the addition of the severe headaches have prevented her from fully participating in work and social activities.

- PMH=negative
- DH=negative
- HH=negative
- PSH=negative

epidemiology

- 90 percent of headaches are primary
- Episodic tension type headache is the most frequent headache in studies
- Migraine is the most frequent diagnosis in patients presenting to primary care physicians
- Cluster headache remains an uncommon diagnosis because of its overall low prevalence (less than 1 percent)

migraine

- It is a disorder of recurrent attacks. Its headache is often but not always unilateral
- Tends to have throbbing or pulsatile quality
- Accompanying features like nausea , vomiting ,phonophobia ,photophobia or osmophobia during attacks

TABLE 422-3 Simplified Diagnostic Criteria for Migraine

REPEATED ATTACKS OF HEADACHE LASTING 4–72 h IN PATIENTS WITH A NORMAL PHYSICAL EXAMINATION, NO OTHER REASONABLE CAUSE FOR THE HEADACHE, AND:

AT LEAST 2 OF THE FOLLOWING FEATURES:	PLUS AT LEAST 1 OF THE FOLLOWING FEATURES:
Unilateral pain	Nausea/vomiting
Throbbing pain	Photophobia and phonophobia
Aggravation by movement	
Moderate or severe intensity	

Source: Adapted from the International Headache Society Classification

Diagnostic criteria for migraine

Migraine without aura
A. At least five attacks fulfilling criteria B through D
B. Headache attacks lasting 4 to 72 hours (untreated or unsuccessfully treated)
C. Headache has at least two of the following characteristics:
Unilateral location
Pulsating quality
Moderate or severe pain intensity
Aggravation by or causing avoidance of routine physical activity (eg, walking or climbing stairs)
D. During headache at least one of the following:
Nausea, vomiting, or both
Photophobia and phonophobia
E. Not better accounted for by another ICHD-3 diagnosis
Migraine with aura
A. At least two attacks fulfilling criteria B and C
B. One or more of the following fully reversible aura symptoms:
Visual
Sensory
Speech and/or language
Motor
Brainstem
Retinal
C. At least three of the following six characteristics:
At least one aura symptom spreads gradually over ≥5 minutes
Two or more symptoms occur in succession
Each individual aura symptom lasts 5 to 60 minutes
At least one aura symptom is unilateral
At least one aura symptom is positive*
The aura is accompanied or followed within 60 minutes by headache
D. Not better accounted for by another ICHD-3 diagnosis
Features of migraine in children and adolescents
Attacks may last 2 to 72 hours ¶
Headache is more often bilateral than in adults; an adult pattern of unilateral pain usually emerges in late adolescence or ear
Photophobia and phonophobia may be inferred by behavior in young children

ICHD-3: International Classification of Headache Disorders, 3rd edition.
* Scintillations and pins and needles are examples of positive symptoms.
¶ The evidence for untreated durations of less than 2 hours in children has not been substantiated.

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headache.org/en/, vbs://journals.sagepub.com/home/cep.

Headache triggers

Diet
Alcohol
Chocolate
Aged cheeses
Monosodium glutamate
Aspartame
Caffeine
Nuts
Nitrites, nitrates
Hormones
Menses
Ovulation
Hormone replacement (progesterone)
Sensory stimuli
Strong light
Flickering lights
Odors
Sounds, noise

Stress
Let-down periods
Times of intense activity
Loss or change (death, separation, divorce, job change)
Moving
Crisis
Changes of environment or habits
Weather
Travel (crossing time zones)
Seasons
Altitude
Schedule changes
Sleeping patterns
Dieting
Skipping meals
Irregular physical activity

Graphic 57424 Version 4.0

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Clinical features

- **Migraine prodrome** — The prodrome occurs in up to 77 percent of migraines and consists of affective or vegetative symptoms that appear 24 to 48 hours prior to the onset of headache .Frequently reported prodromal symptoms include increased yawning, euphoria, depression, irritability, food cravings, constipation, and neck stiffness.
- **Migraine aura** — About 25 percent of people with migraines experience one or more focal neurologic symptoms called aura
- typical migraine auras are characterized by gradual development, duration no longer than one hour, a mix of positive and negative features, and complete reversibility

MIGRAINE SUBTYPES

- Migraine with brainstem aura:

is an uncommon form of migraine with aura wherein the primary signs and symptoms are referable to the brainstem without weakness.

The auras consist of some combination of vertigo, dysarthria, tinnitus, diplopia, ataxia.

Migraine subtypes

- Hemiplegic migraine:

The primary feature that separates hemiplegic migraine from other types of migraine with aura is the presence of motor weakness as a manifestation of aura in at least some attacks.

Migraine subtypes

- Vestibular migraine:

Vestibular migraine is a term used to describe episodic vertigo in patients with a history of migraines or with other clinical features of migraine (photophobia, phonophobia, visual aura, etc)

Migraine subtypes

- Chronic migraine:

Chronic migraine is defined as headache occurring 15 or more days a month for more than three months, which has the features of migraine headache on at least eight days a month

Tension type

- The typical presentation of a TTH attack is that of a mild to moderate intensity, bilateral, nonthrobbing headache without other associated features. Descriptions of TTH pain are characteristically nondescript: "dull," "pressure," "head fullness", "head feels large," or, more descriptively, "like a tight cap", "band-like," or a "heavy weight on my head or shoulders.“
- The pain in TTH may infrequently be unilateral or pulsating
- Stress and mental tension are reported to be the most common precipitants for TTH

Diagnostic criteria

- require at least 10 episodes of headache, each lasting 30 minutes to seven days, which fulfill the following conditions :
- ●At least two of the following:
- •Bilateral location
- •Pressing or tightening (non-pulsating) quality
- •Mild or moderate intensity
- •Not aggravated by routine physical activity such as walking or climbing stairs
- ●Both of the following:
- •No nausea or vomiting
- •No more than one of photophobia or phonophobia

subtypes

- The infrequent episodic TTH subform is diagnosed if the headache episodes occur on <1 day per month on average (<12 days per year)

- The frequent episodic TTH subform is diagnosed if the headache episodes occur on 1 to 14 days per month on average (≥12 and <180 days per year).

- The ICHD-3 criteria for chronic TTH require headache lasting hours to days, or unremitting, occurring on ≥15 days per month on average for more than three months (≥180 days per year)

cluster

- Cluster headache belongs to a group of idiopathic headache entities, the trigeminal autonomic cephalalgias ;all of which involve unilateral, often severe headache attacks and typical accompanying autonomic symptoms. Cluster headache is characterized by attacks of severe unilateral orbital, supraorbital, or temporal pain accompanied by autonomic phenomena. Unilateral autonomic symptoms are ipsilateral to the pain and may include ptosis, miosis, lacrimation, conjunctival injection, rhinorrhea, and nasal congestion. Attacks usually last 15 to 180 minutes.
- Cluster headache may sometimes be confused with a life-threatening headache, since the pain from a cluster headache can reach full intensity within minutes. However, cluster headache is transient, usually lasting less than one to two hours

Clinical features and treatment of the trigeminal autonomic cephalalgias

	Cluster headache	Paroxysmal hemicrania	SUNCT* and SUNA¶	Hemicrania continua
Sex predominance	Male (4:1)	No (1:1)	Female (1.7:1)	Female (2:1)
Pain				
Type	Stabbing	Stabbing or throbbing	Stabbing or burning	Stabbing, throbbing, burning, or aching
Severity	Excruciating	Excruciating	Severe to excruciating	Mild to severe
Site	Orbital or temporal	Orbital or temporal	Orbital or temporal	Orbital, frontal, and/or temporal
Typical attack frequency	1 every other day to 8 daily	5 to 40 daily	1 to 200 daily	Continuous (with exacerbations)
Duration of attack	15 to 180 minutes	2 to 30 minutes	1 second to 10 minutes	Months to years (untreated)
Autonomic features? Δ	Yes	Yes	Yes (conjunctival injection and lacrimation prominent with SUNCT)	Yes
Restlessness and/or agitation?	Yes	Yes	Sometimes	Yes
Associated migrainous features? ◊	Yes	Yes	Rare	Frequent
Triggers	Alcohol	Stress, exercise, alcohol	Tactile stimuli (eg, touching face, shaving, brushing teeth)	Alcohol
Indomethacin responsive?	No	Yes	No	Yes
Abortive treatment	Triptans (intravenous or nasal) Oxygen	None	Lidocaine (intravenous) for frequent and debilitating symptoms	None
Prophylactic treatment	Verapamil Glucocorticoids Galcanezumab Lithium	Indomethacin Verapamil NSAIDs	Lamotrigine Oxcarbazepine Topiramate Gabapentin	Indomethacin

SUNCT: short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing; SUNA: short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms; NSAIDs: nonsteroidal anti-inflammatory drugs.

* Both conjunctival injection and lacrimation are present.

¶ May have either conjunctival injection or lacrimation but not both.

Δ Cranial autonomic symptoms: conjunctival injection, lacrimation, nasal congestion or rhinorrhea, facial sweating, miosis and/or ptosis, palpebral edema; symptoms are ipsilateral to the headache.

◊ Migraine-like features may include associated nausea, vomiting, photophobia, or phonophobia.

Graphic 65541 Version 13.0

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Diagnosing criteria

- Diagnostic criteria for cluster headache, according to the International Classification of Headache Disorders 3rd edition (ICHD-3), require all of the following]:
- ●At least five attacks
- ●Attacks characterized by severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15 to 180 minutes when untreated; during part (but less than half) of the time-course of cluster headache, attacks may be less severe and/or of shorter or longer duration
- ●Either or both of the following:
- •At least one of the following symptoms or signs ipsilateral to the headache:
- -Conjunctival injection and/or lacrimation
- -Nasal congestion and/or rhinorrhea
- -Eyelid edema
- -Forehead and facial sweating
- -Forehead and facial flushing
- -Sensation of fullness in the ear
- -Miosis and/or ptosis
- •A sense of restlessness or agitation
- ●Attacks have a frequency between one every other day and eight per day for more than half of the time when the disorder is active
- ●Not better accounted for by another ICHD-3 diagnosis

Secondary headache

- Physicians who evaluate patients with headache should be alert to signs that suggest a serious underlying disorder

- **Danger signs** — Paying attention to danger signs is important since headaches may be the presenting symptom of a space-occupying mass or vascular lesion, infection, metabolic disturbance, or a systemic problem. The following features in the history can serve as warning signs of possible serious underlying disease

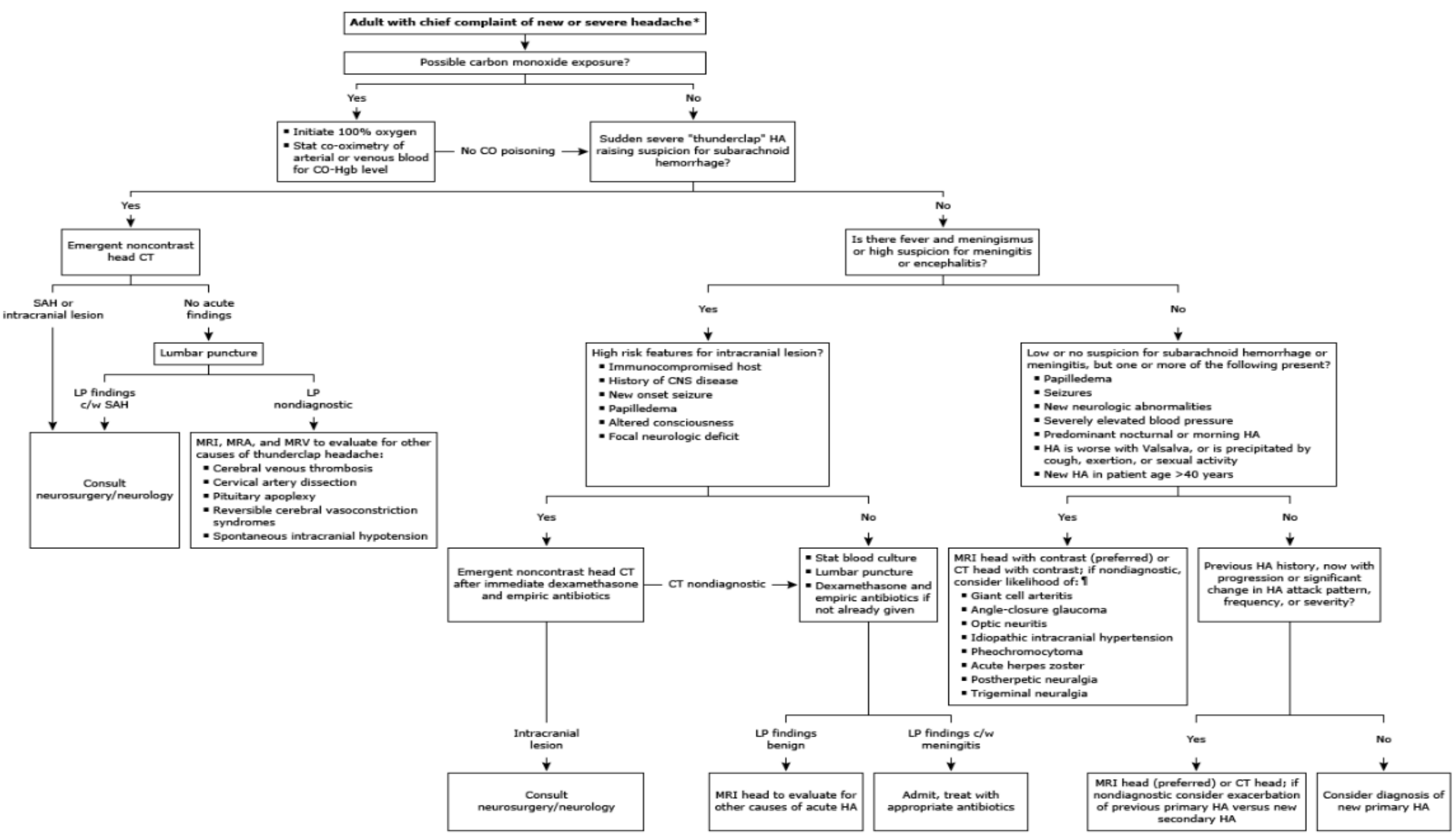
Danger signs

- The mnemon**ic SNNOOP10** is a reminder of the danger signs ("red flags") for the presence of serious underlying disorders that can cause acute or subacute headache:
- •**S**ystemic symptoms including fever
- •**N**eoplasm history
- •**N**eurologic deficit (including decreased consciousness)
- •**O**nset is sudden abrupt onset
- •**O**lder age (onset after age 50 years)
- •**P**attern change or recent onset of new headache
- •**P**ositional headache
- •**P**recipitated by sneezing, coughing, or exercise
- •**P**apilledema
- •**P**rogressive headache and atypical presentations
- •**P**regnancy or puerperium
- •**P**ainful eye with autonomic features
- •**P**ost-traumatic onset of headache
- •**P**athology of the immune system such as HIV
- •**P**ainkiller (analgesic) overuse (eg, medication overuse headache

Indications for imaging studies

- Focal neurologic signs or symptoms
- Onset of headache with exertion, cough, or sexual activity
- Orbital bruit
- Onset of headache after age 40 years
- Recent significant change in the pattern, frequency, or severity of headaches
- Progressive worsening of headache despite appropriate therapy

- MRI is the preferred brain imaging modality for most patients because it is more sensitive than CT scan for detecting edema, vascular lesions, and other types of intracranial pathology, particularly in the posterior fossa. However, CT is more widely available and is therefore more useful in urgent or emergency care situations when there is concern for subarachnoid hemorrhage as the cause of thunderclap headache


Adult with chief complaint of new or severe headache*
Possible carbon monoxide exposure?
Yes
No
Initiate 100% oxygen
Stat co-oximetry of arterial or venous blood for CO-Hgb level
No CO poisoning
Sudden severe "thunderclap" HA raising suspicion for subarachnoid hemorrhage?
Yes
No
Emergent noncontrast head CT
SAH or intracranial lesion
No acute findings
Lumbar puncture
LP findings c/w SAH
LP nondiagnostic
Consult neurosurgery/neurology
MRI, MRA, and MRV to evaluate for other causes of thunderclap headache:
Cerebral venous thrombosis
Cervical artery dissection
Pituitary apoplexy
Reversible cerebral vasoconstriction syndromes
Spontaneous intracranial hypotension
Is there fever and meningismus or high suspicion for meningitis or encephalitis?
Yes
No
High risk features for intracranial lesion?
Immunocompromised host
History of CNS disease
New onset seizure
Papilledema
Altered consciousness
Focal neurologic deficit
Low or no suspicion for subarachnoid hemorrhage or meningitis, but one or more of the following present?
Papilledema
Seizures
New neurologic abnormalities
Severely elevated blood pressure
Predominant nocturnal or morning HA
HA is worse with Valsalva, or is precipitated by cough, exertion, or sexual activity
New HA in patient age >40 years
Yes
No
Emergent noncontrast head CT after immediate dexamethasone and empiric antibiotics
CT nondiagnostic
Stat blood culture
Lumbar puncture
Dexamethasone and empiric antibiotics if not already given
MRI head with contrast (preferred) or CT head with contrast; if nondiagnostic, consider likelihood of: ¶
Giant cell arteritis
Angle-closure glaucoma
Optic neuritis
Idiopathic intracranial hypertension
Pheochromocytoma
Acute herpes zoster
Postherpetic neuralgia
Trigeminal neuralgia
Previous HA history, now with progression or significant change in HA attack pattern, frequency, or severity?
Intracranial lesion
LP findings benign
LP findings c/w meningitis
Yes
No
Consult neurosurgery/neurology
MRI head to evaluate for other causes of acute HA
Admit, treat with appropriate antibiotics
MRI head (preferred) or CT head; if nondiagnostic consider exacerbation of previous primary HA versus new secondary HA
Consider diagnosis of new primary HA

Headache emergencies

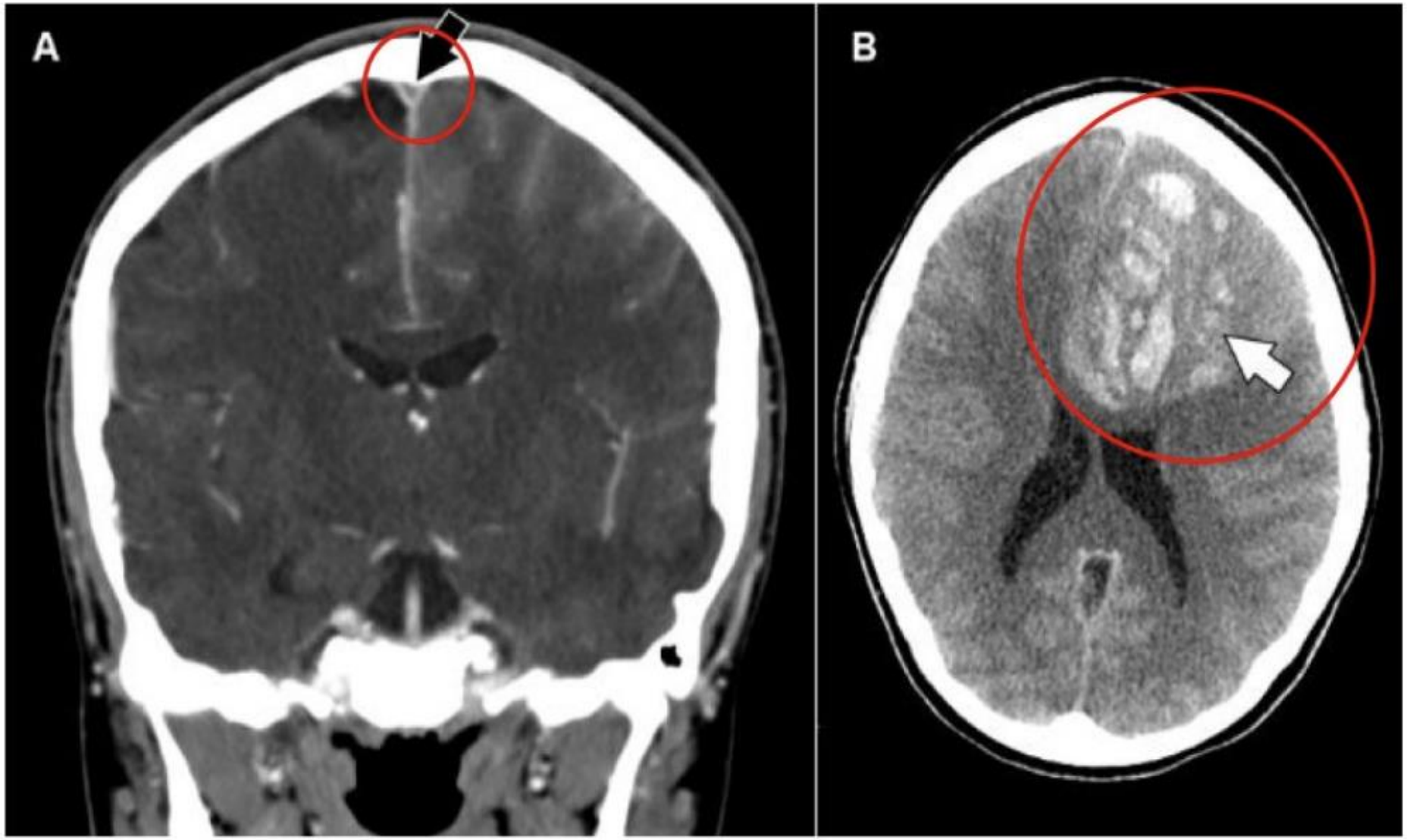
- ●**Sudden onset "thunderclap" headache** – Severe headache of sudden onset (ie, that reaches maximal intensity within a few **seconds** or **less than one minute** after the onset of pain) is known as thunderclap headache because its explosive and unexpected nature is likened to a "clap of thunder." Thunderclap headache requires urgent evaluation as such headaches may be harbingers of subarachnoid hemorrhage and other potentially ominous etiologies
- ●**Acute or subacute neck pain or headache with Horner syndrome and/or neurologic deficit** – Cervical artery dissection is usually associated with local symptoms including neck pain or headache, and often results in ischemic stroke or transient ischemic attack. Horner syndrome is seen in approximately 39 percent of those with carotid and 13 percent of those with vertebral artery dissection
- ●**Headache with suspected meningitis or encephalitis** – Fever, altered mental status, with or without nuchal rigidity may indicate central nervous system infection.

Headache emergencies

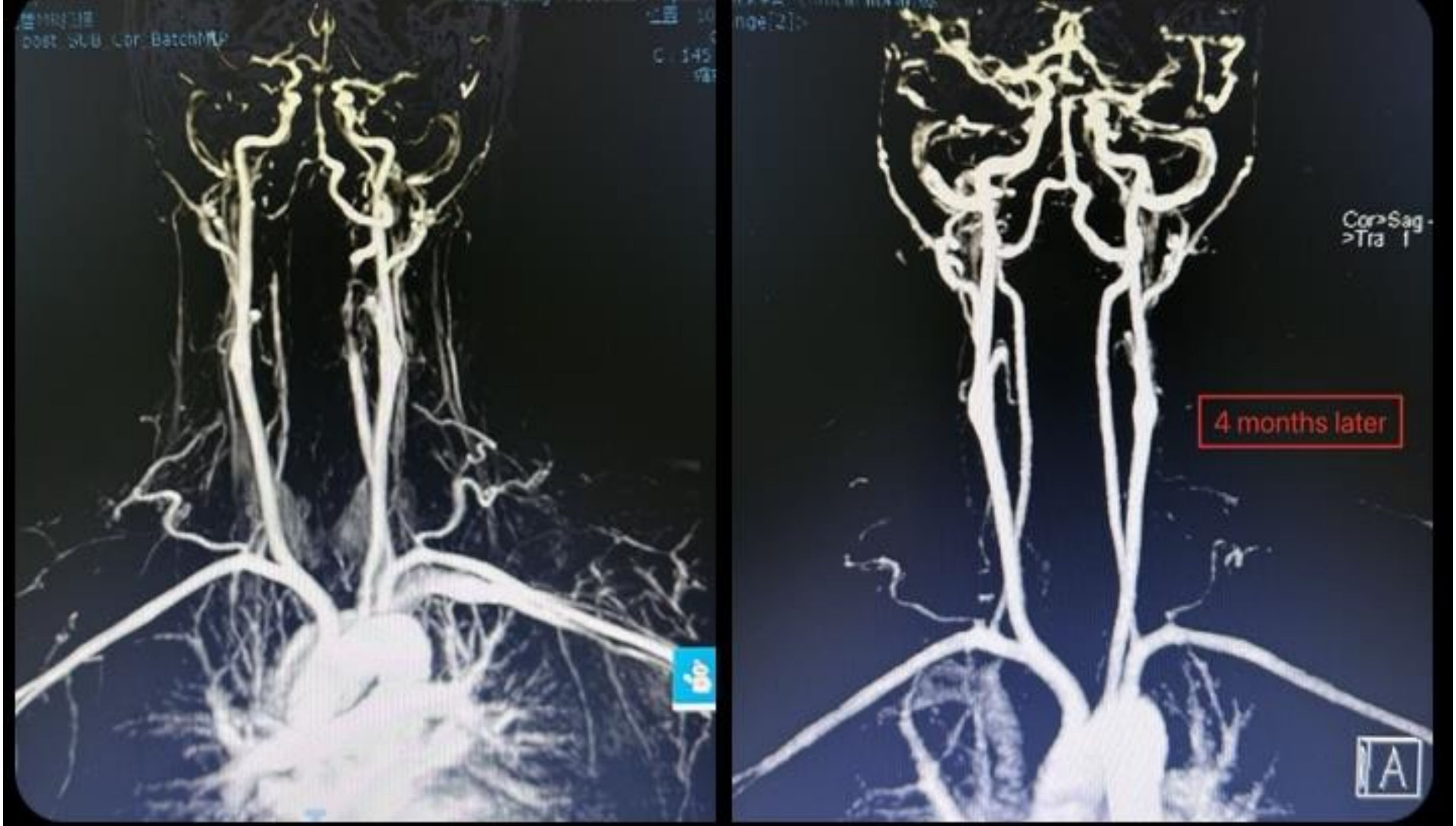
- ●**Headache with global or focal neurologic deficit or papilledema** – Headache is the primary symptom of increased ICP, which should be suspected when accompanied by bilateral papilledema, focal neurologic deficit, or repeated episodes of nausea and vomiting.
- ●**Headache with orbital or periorbital symptoms** – Headache with visual impairment, periorbital pain, or ophthalmoplegia could indicate acute angle closure glaucoma, infection, inflammation, vascular congestion from a cavernous sinus thrombosis or draining arteriovenous malformation, or tumor involving the orbits.
- ●**Headache and possible carbon monoxide exposure –** Headache is a nonspecific symptom of carbon monoxide exposure; the intensity varies with the carbon monoxide level. The headache tends to be bilateral and mild at low levels of carbon monoxide, pulsating at levels of 20 to 30 percent, and severe with nausea, vomiting, and blurred vision at levels of 30 to 40 percent.

Etiologies of thunderclap headache

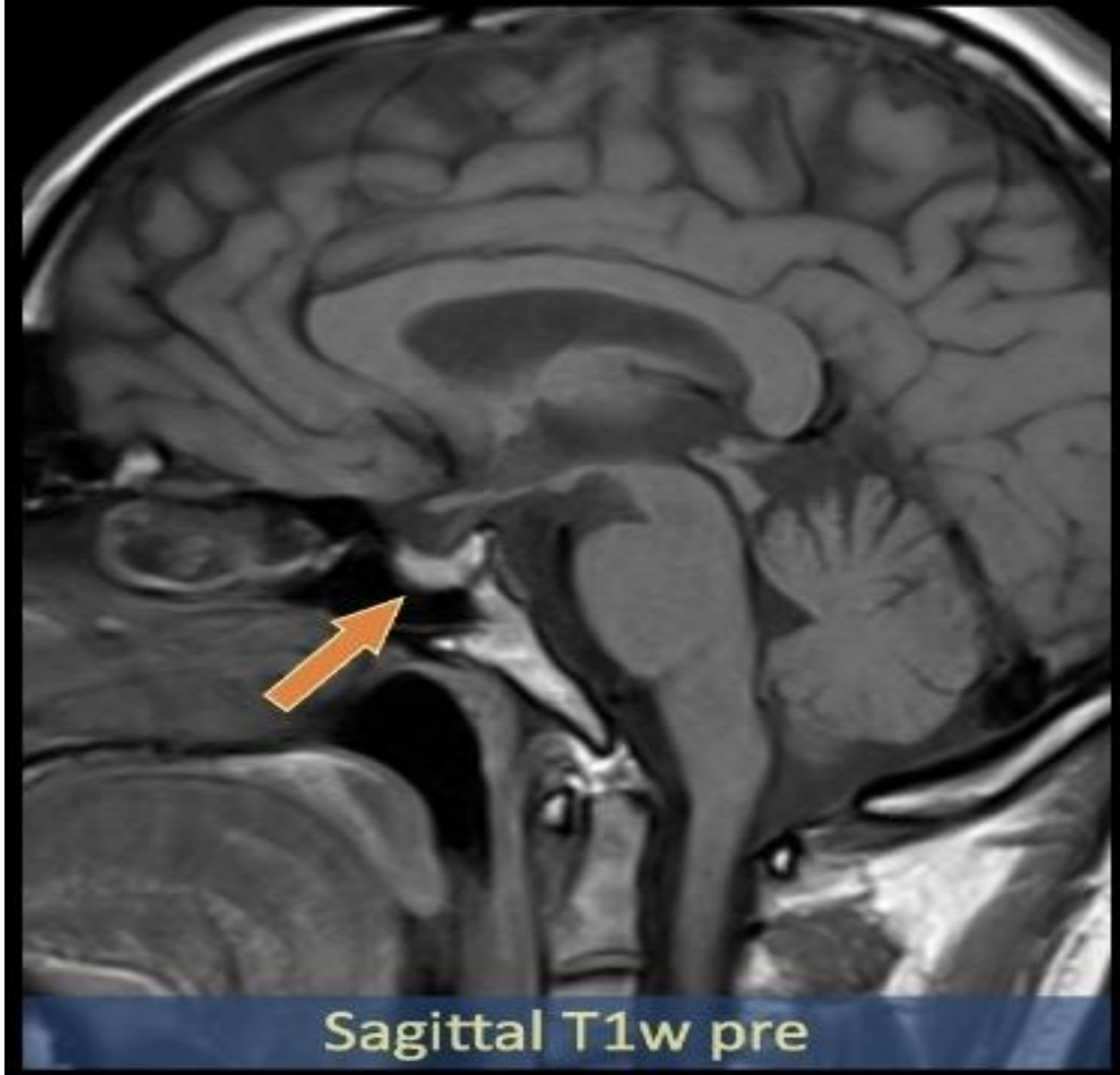
Most common causes of thunderclap headache:
Subarachnoid hemorrhage
Reversible cerebral vasoconstriction syndromes (RCVS)
Conditions that less commonly cause thunderclap headache:
Cerebral infection (eg, meningitis, acute complicated sinusitis)
Cerebral venous thrombosis
Cervical artery dissection
Spontaneous intracranial hypotension
Acute hypertensive crisis
Posterior reversible leukoencephalopathy syndrome (PRES)
Intracerebral hemorrhage
Ischemic stroke
Conditions that uncommonly or rarely cause thunderclap headache:
Pituitary apoplexy
Colloid cyst of the third ventricle
Aortic arch dissection
Aqueductal stenosis
Brain tumor
Giant cell arteritis
Pheochromocytoma
Pneumocephalus
Retroclival hematoma
Spinal epidural hematoma
Varicella zoster virus vasculopathy
Vogt-Koyanagi-Harada syndrome



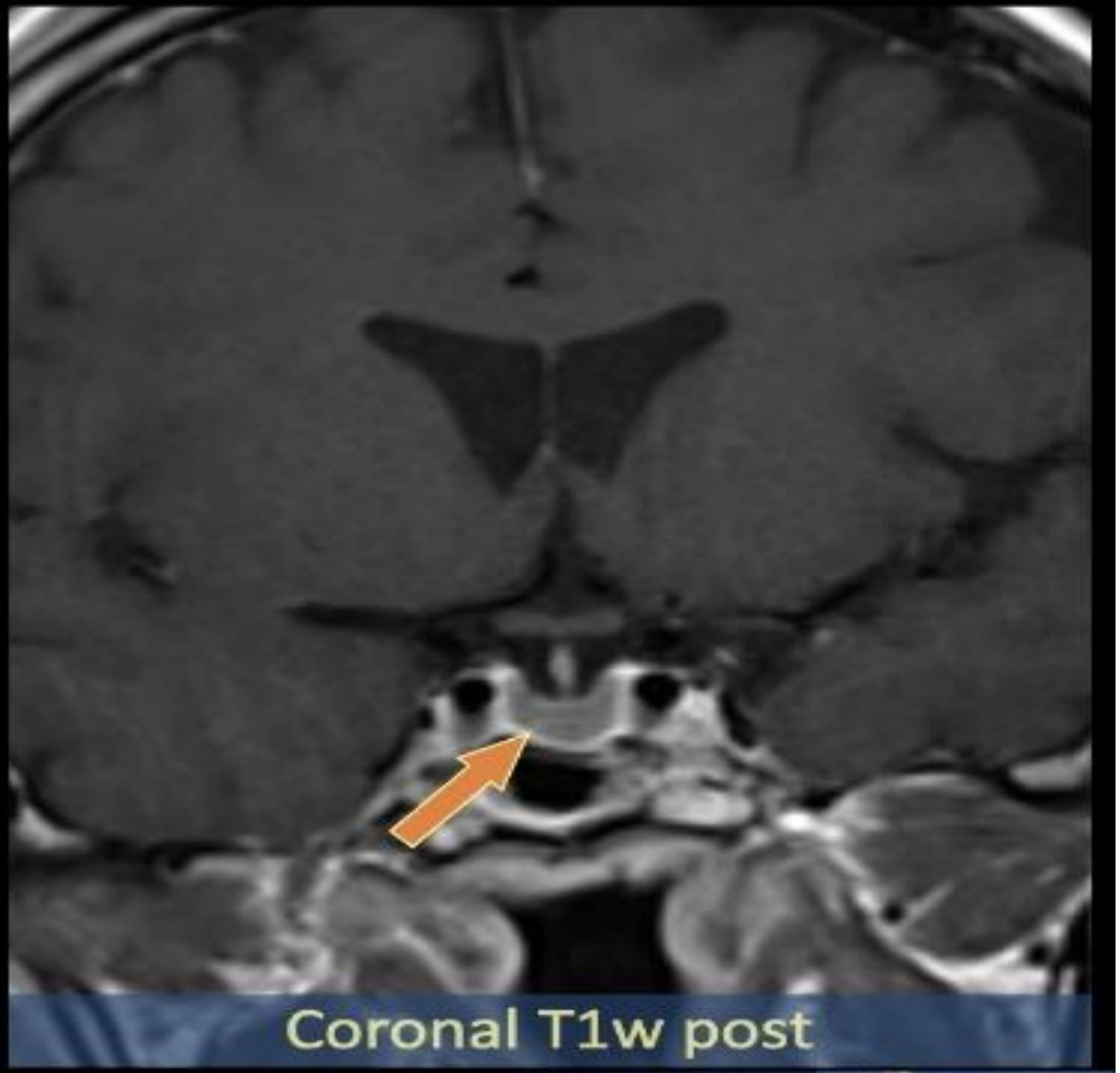
Panel A shows a coronal section from the admission CT venogram, with a filling defect ('empty delta sign') seen in the superior sagittal sinus (black arrow).
An axial section from the unenhanced CT scan performed approximately 6 hours later (Panel B) shows venous haemorrhage in the left frontal lobe (white arrow), with marked mass effect.


4 months later
A

Case 230: Pituitary Apoplexy (normal gland)



Sagittal T1w pre



Coronal T1w post

@RyanBPetersonMD

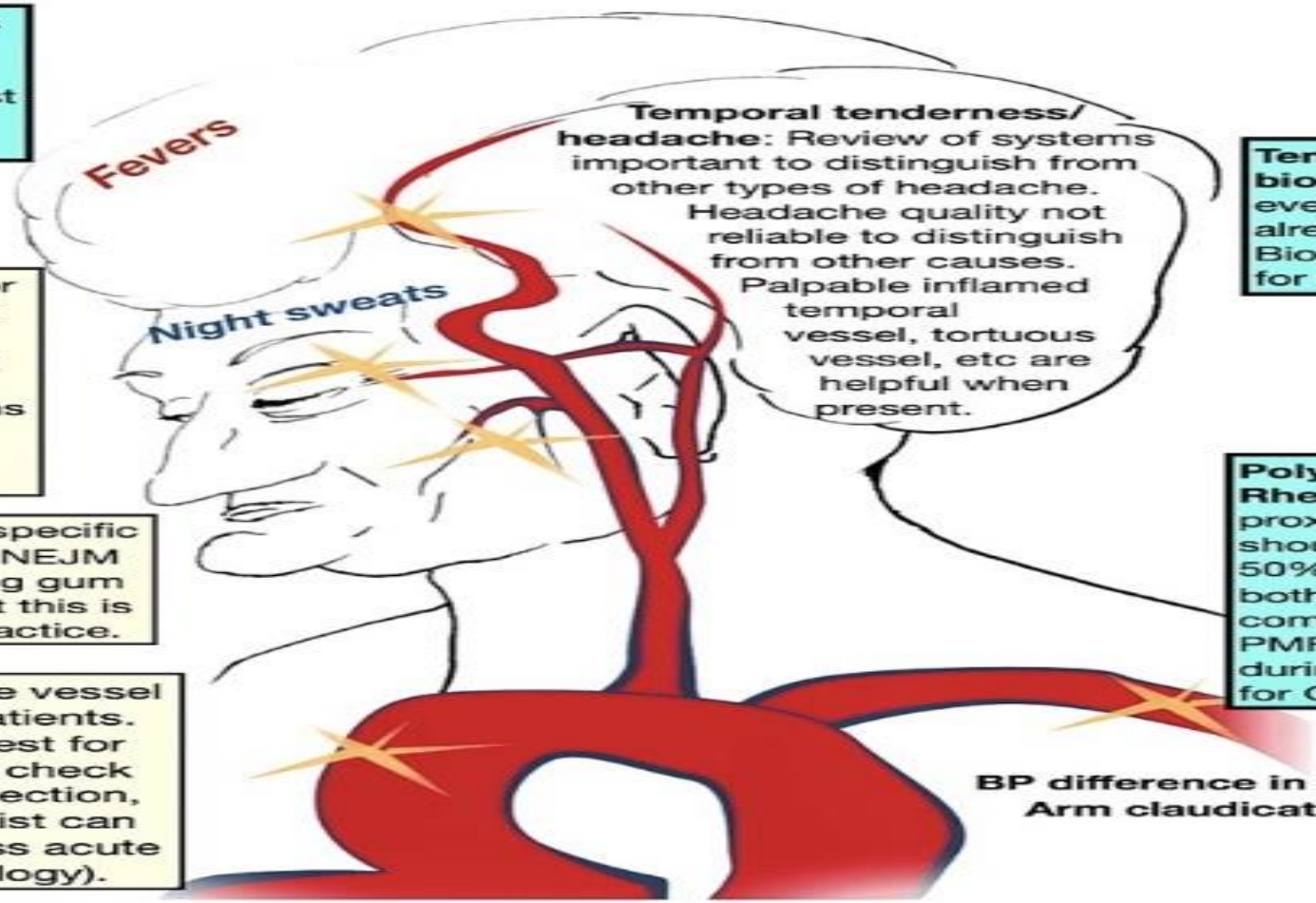
EMORY

Other causes

- ●**Giant cell (temporal) arteritis** (GCA) is a chronic vasculitis of large and medium sized vessels. The disease seldom occurs before age 50 years, and its incidence rises steadily thereafter. A new type of headache occurs in two-thirds of affected individuals. The head pain tends to be located over the temporal areas but can be frontal or occipital in location. The headaches may be mild or severe. Other common symptoms can include fever, fatigue, weight loss, jaw claudication, visual symptoms, particularly transient monocular visual loss and diplopia, and symptoms of polymyalgia rheumatica. Laboratory testing may reveal an elevated erythrocyte sedimentation rate and/or serum C-reactive protein, or thrombocytosis, but these are not specific.
- The diagnosis of GCA is based on histopathology or imaging exams. Histopathologic evidence of GCA is most often acquired by temporal artery biopsy. Color Doppler ultrasound (CDUS) of the head, as performed by experienced operators, is an alternative diagnostic procedure. CDUS can visualize temporal artery abnormalities (eg, mural edema as shown by the "halo sign" and "compression sign") characteristic of GCA. When the diagnosis of GCA is still suspected in a patient who has had a negative temporal artery biopsy and/or CDUS, the possibility of large vessel involvement can be evaluated by imaging the torso with CT/CTA, MRI/MRA, or positron emission tomography (PET)

Giant-Cell Arteritis

Epidemiology: Increases with age, peak in 8th decade of life. Northern European descent highest prevalence, followed by southern European.



Temporal artery biopsy: Try to obtain, even if steroids already initiated. Biopsy stays positive for up to a month.

Vision loss: Temporary or permanent. Mechanism: usually ischemia to vascular supply to optic nerve. Consult ophtho urgently if these symptoms present. Indication for higher steroid doses compared to without.

Jaw claudication: More specific than sensitive if present. NEJM article describes "chewing gum test" to bring this out, but this is not yet part of routine practice.

Aortic Aneurysm: Large vessel involvement 25% of patients. +/- CTA vs MRA of chest for biopsy proven GCA to check aorta for stenosis, dissection, and aneurysm (specialist can decide; not urgent unless acute signs for aortic pathology).

Polymyalgia Rheumatica: Pain / proximal weakness, shoulders and hips. 50% of patients have both. Which one comes first varies. PMR can present during steroid taper for GCA, for example.

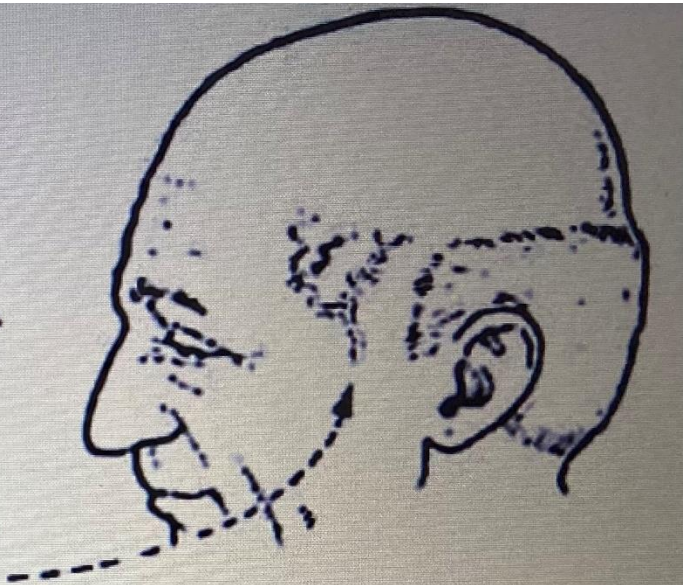
Key Lab Finding: High ESR, often >100
Lab findings sometimes present:
- Anemia
- Thrombocytosis
- Transaminitis

Anorexia/Fatigue:
Feelings of illness will not be subtle. Symptoms will be significant and uncomfortable.

Treatment: Steroids have been the mainstay. 1mg/kg/day or 60mg prednisone. Very long taper. Higher doses in acute setting if eye involvement. PMR alone treatment is lower dose: 15-20mg/day prednisone start.
Steroid sparing: Mixed data on agents such as methotrexate. New IL-6 inhibitor **Tocilizumab** is effective in reducing total steroid requirements.

GIANT CELL (TEMPORAL) ARTERITIS

Giant cell arteritis, an autoimmune disease of unknown cause, presents with throbbing headache in patients over 60 often with general malaise. The involved vessel, usually the superficial temporal artery, may be tender, thickened, and but nonpulsatile.



Neurological symptoms: **strokes, hearing loss, myelopathy and neuropathy.**

Jaw claudication: **pain when chewing or talking due to ischaemia of the masseter muscles is pathognomonic.**

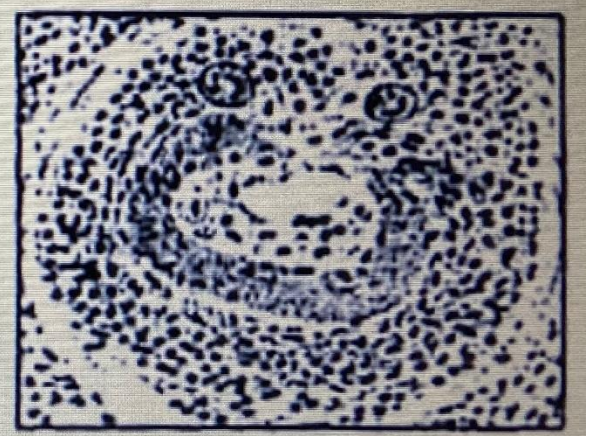
Visual symptoms **are common with blindness (transient or permanent) or diplopia.**

Associated systemic symptoms **– weight loss, lassitude and generalised muscle aches – polymyalgia rheumatica in one-fifth of cases.**

Duration: **the headache is intractable, lasting until treated.**

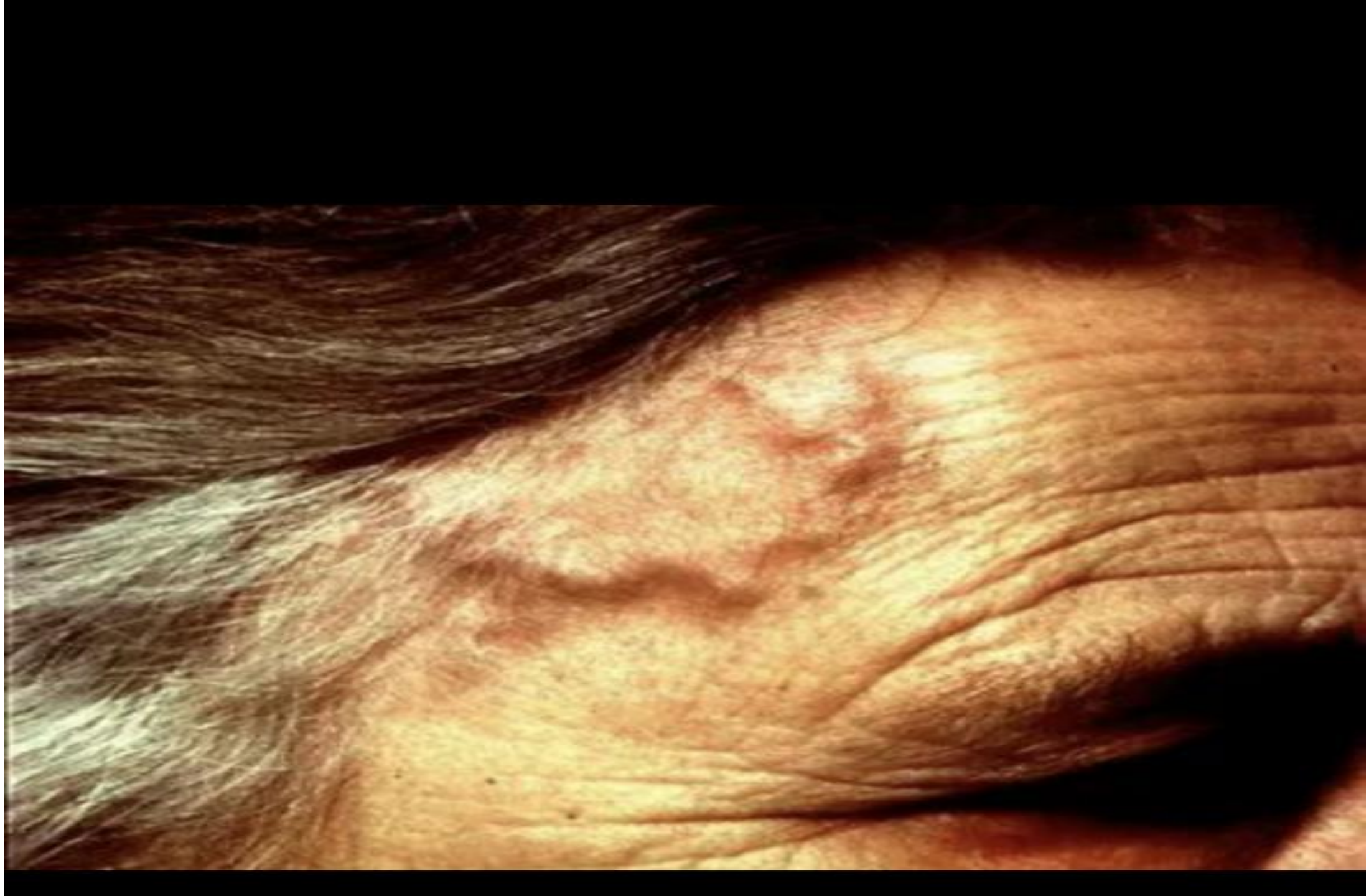
Mechanism:

Large and medium-sized arteries undergo intense 'giant cell' infiltration, with fragmentation of the lamina and narrowing of the lumen, resulting in distal ischaemia as well as stimulating pain sensitive fibres. Occlusion of important end arteries, e.g. the ophthalmic artery, may result in blindness; occlusion of the basilar artery may cause brain stem or bilateral occipital infarction.

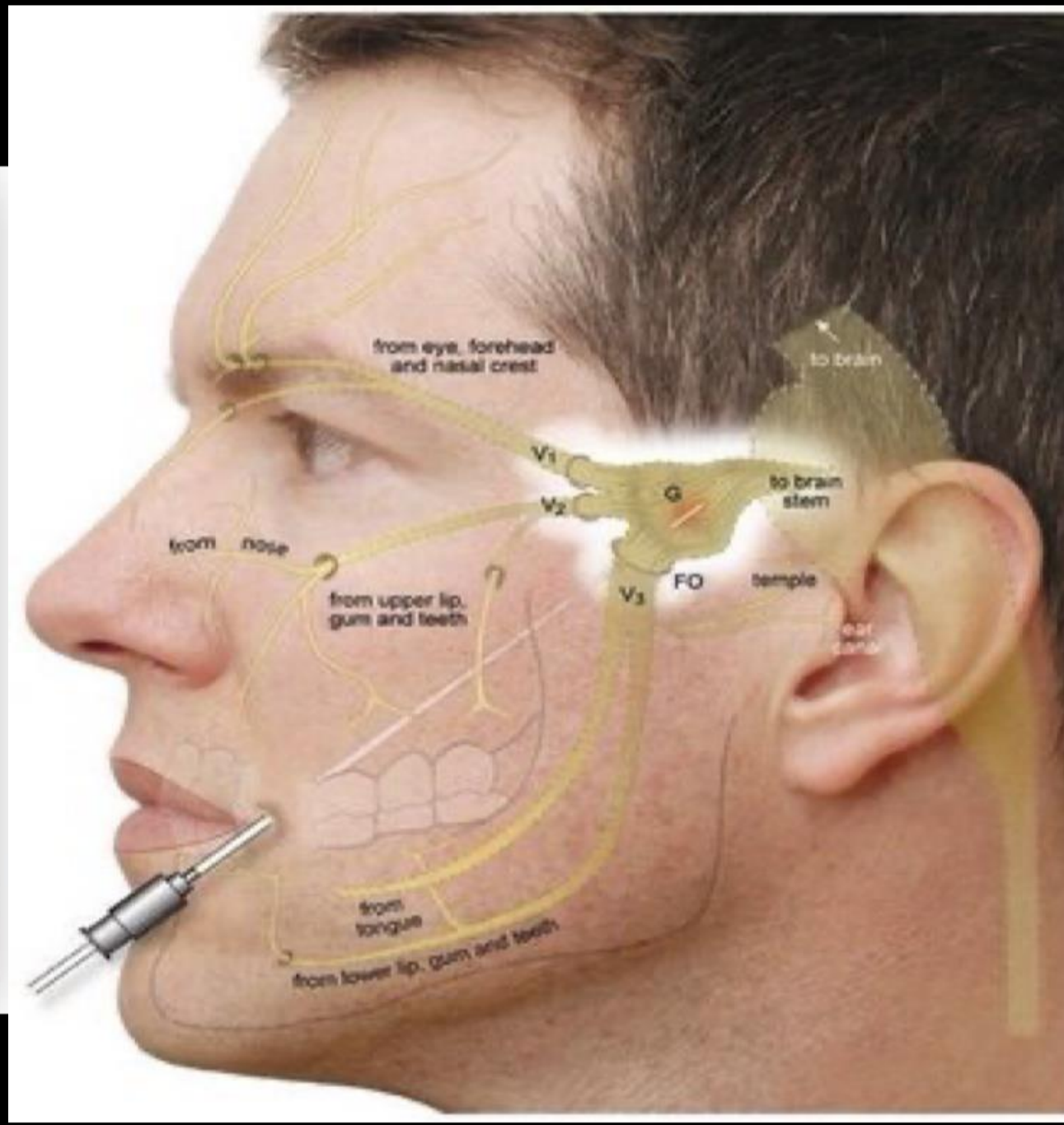


Thickened wall with giant cell infiltrate

Diagnosis: ESR usually high. Blood film shows anaemia or



- **Trigeminal neuralgia** is defined by sudden, usually unilateral, severe, brief, stabbing or lancinating, recurrent episodes of pain in the distribution of one or more branches of the fifth cranial (trigeminal) nerve. The incidence increases gradually with age; most idiopathic cases begin after age 50 years. Once the diagnosis is suspected on clinical grounds, it is important to search for secondary causes. Patients with suspected trigeminal neuralgia or those with recurrent attacks of pain limited to one or more divisions of the trigeminal nerve and no obvious cause (eg, herpes zoster or trigeminal nerve trauma) should undergo imaging to help distinguish classic trigeminal neuralgia from secondary causes. MRI and MRA of the head without and with contrast tailored to evaluate the trigeminal nerve is the preferred imaging exam to evaluate for compression of the nerve by adjacent vessels or other structures


from eye, forehead and nasal crest
to brain
V_1
V_2
G
to brain stem
from nose
from upper lip, gum and teeth
V_3
FO
temple
from tongue
from lower lip, gum and teeth

- ●**Acute herpes zoster** and **postherpetic neuralgia** often involve cervical and trigeminal nerves. Pain is the most common symptom of zoster and approximately 75 percent of patients have prodromal pain in the dermatome where the rash subsequently appears. The major risk factors for postherpetic neuralgia are older age, greater acute pain, and greater rash severity. Acute herpes zoster is usually a clinical diagnosis based upon the characteristic vesicular lesions in a restricted dermatomal pattern. The diagnosis of postherpetic neuralgia is made when pain persists beyond four months in the same distribution as a preceding documented episode of acute herpes zoster

- ●**Brain tumor** should be considered as a possible cause of new-onset headaches in adults over age 50 years, as discussed above

سطوح پیشگیری

Primordial Prevention

Primary Prevention

Secondary Prevention

Tertiary Prevention

Quaternary Prevention

Primordial prevention

- آموزش صحیح به پزشکان و مراقبین سالمت جهت برخورد صحیح با سردرد و علل ایجاد آن
- آموزش های لازم در سطح جامعه در مورد اهمیت سردرد های ناگهانی با علل تهدید کننده حیات
- اطلاع رسانی به تمام افراد جامعه برای تشکیل پرونده الکترونیک
سلامت جهت ثبت اطلاعات و سوابق شخصی و خانوادگی
- کاهش چاقی جمعیت و کنترل دیابت و فشارخون بعنوان عوامل دارای همراهی با میگرن

Primary prevention

- آموزش چهره به چهره به مادران باردار جهت مراجعه به پزشک و اطلاع

وی در صورت بروزسردرد

- اطلاع به افراد با سابقه سردردهای اولیه درمورد عوامل برانگیزنده حملات سردرد و جلوگیری از وقوع انها

Secondary prevention

- درمانهای پیشگیری کننده در مواقع دارای اندیکاسیون در افراد مبتلا به سر درد های اولیه

Tertiary prevention

- درمان و پیگیری مناسب در افراد مبتلا به سردرد
- ارجاع جهت بستری و اقدامات تشخیصی مناسب در افراد دارای اندیکاسیون

Quaternary prevention

- عدم انجام اقدامات تشخیصی و درمانی اضافی
- جلوگیری از بستری بی مورد در سردردهای خوشخیم اولیه
- عدم تجویز نامناسب دارو، پروفیلاکسی و ...

Thanks for your attention