Optometric Role in the Diagnosis and Management of Headaches

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NEUROANATOMY AND NEURO-OPHTHALMIC DISORDERS

Headaches and the Eye

Asthenopia vs ocular or intracranial disease

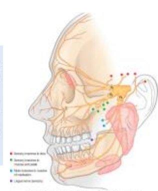
Ocular origin vs. intracranial origin
- Shared innervation

Role in management
- Patients often present to optometrists first
- Patients are frequently managed inadequately

Origins of Pain

Pain sensitive structures of head include muscles, blood vessels, dural and venous sinuses, meninges, scalp

Shared innervation of pain sensitive structures in the eye and adnexa



Headache Types

Asthenopic
- Refractive errors, binocular and accommodative disorders
- Exacerbated by near work

Primary
- No tissue lesion identified
- Pain from activation of trigeminovascular nerve endings

Secondary
- Identifiable tissue lesion (compression, traction, inflammation of dura, nerves and vessels
- Often associated with "red flags"

Migraine

Series of episodic headache disorders with typical features.
- Migraine with aura
- Migraine without aura
- Retinal Migraine
- Ophthalmoplegic Migraine**

Epidemiology of Migraine

- 18% of women, 6% of men have migraine
- 23 million Americans
- Most migraine patients have not been diagnosed by a physician
- Most use only OTC meds and are treated suboptimally

What we now know about migraine

Migraine is a neurovascular disorder
Neurologic event causes a vascular response

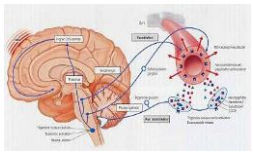
Migraine aura is not caused by ischemia

The predisposition for migraine is inherited
Specific genes have been identified

Neurovascular Mechanism

Defect in ion channels leads to hyperexcitability of neurons leading to activation of the **trigeminal vascular system**

- This system consists of small, sensory neurons that originate from the trigeminal ganglion and upper cervical dorsal nerve roots
- These neurons innervate cerebral blood vessels, meninges and veins
- Dysfunction of brainstem ion channels impairs serotonin release and predisposes patients to migraine, or impairs their self-aborting mechanism.



Activation of the trigeminal ganglion causes:

- Release of neuropeptides associated with neurogenic inflammation
- Vasodilation

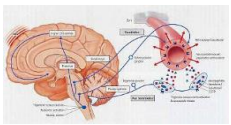
Pathophysiology

Pain signals reach the thalamus which projects to the cortex for perception of pain

Vasodilation also leads to release of more neuropeptides

- Cycle is perpetuated

Migraine medications block the release of neuropeptides, leading to constriction of dilated meningeal vessels. In addition, certain medications also block trigeminal nerve pathways or modulate the neurogenic inflammation.



Calcitonin Gene Related Peptide

Most important neuropeptide released in migraine sufferers

Vasoactive peptide release in the extracerebral circulation of humans during migraine headache

P J Goadsby [1], L Edvinsson, R Ekman

Affiliations + expand

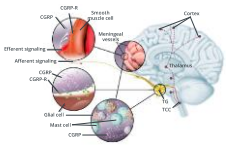
PMID: 1689472 DOI: 10.1002/ana.410280213

CGRP and Migraines

It is theorized that CGRP sensitizes peripheral nerves, which in turn send signals to the central nervous system that trigger the pain and sensitivity to sensory stimuli associated with migraine.

This may occur because when CGRP binds to its receptor, it sensitizes the nerves by increasing their rate of firing.

CGRP might also activate pain receptors by dilating blood vessels, which could push on pain receptors on adjacent nerves.



TG = Trigeminal ganglion
TCC =Trigeminocervical complex

Migraine without Aura

- Moderate to severe headache lasting hours to days
- Nausea, vomiting, photophopia, sonophobia common
- Triggers can occur and include stress, estrogen deprivation, foods, weather changes, sleep deprivation
- Typically relieved by sleep
- Positive family history
- Peak onset in second and third decade

Migraine With Aura

Transient neurologic deficit precedes migraine
- Scintillating scotoma, paresthesias, aphasia, hemiparesis

Aura can occur without headache (acephalgic migraine).

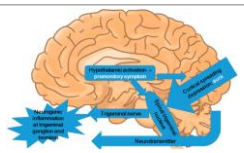
Aura is typically consistent but switches hemifields.

Differential dx includes seizures or TIA

Visual Aura

Migraine aura is NOT due to ischemia!

Neuronal and glial depolarization spreading across the cerebral cortex is thought to cause the aura of the migraine.
- This activates the trigeminal afferents, which cause inflammatory changes in the meninges, leading to pain.



Migraine Pathophysiology Revisited: Proposal of a New Molecular Theory of Migraine Pathophysiology and Headache Diagnostic Criteria

by Yasushi Shibata

Mito Medical Center, Mito Kyodo General Hospital, University of Tsukuba, Tsukuba 310-0015, Ibaraki, Japan

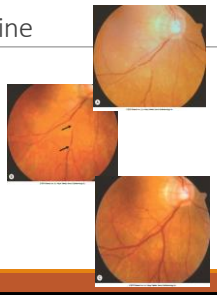
Int. J. Mol. Sci. 2022, 23(21), 13002. https://doi.org/10.3390/ijms232113002

Retinal Migraine

Episodic MONOCULAR vision loss with or without periocular headache.

Narrowed retinal arterioles can be seen at time of attack
- A-narrowed arteries/veins with segmented flow; pale retina
- B-more obvious gaps in flow
- C-resumption of flow

Represents vasospasm; more commonly seen in migraineurs



Ophthalmoplegic Migraine

No longer classified as migraine
- Neuralgia

Episodic, severe hemicranial headache is accompanied by ipsilateral third or sixth nerve palsy that outlasts the palsy.

RARE!

First attack typically occurs in childhood

Differential dx includes cerebral aneurysm, compressive, inflammatory or neoplastic disease.

Migraine precipitants

Stress	Foods
Menstrual cycle	Exercise
Lack of sleep	Sunlight
Excess sleep	Odors
Hunger	Weather conditions

Pinpointing the Triggers

Tension Headache

Mild to moderate, episodic head pain described at tightness

No photophobia or sonophobia, no disruption in activity

Mechanism unknown; might be a migraine variant

Very common

Cluster Headache

At least five attacks of severe, unilateral periocular pain lasting up to 3 hours with associated conjunctival injection, lacrimation, nasal congestion, rhinorrhea or Horner's.

Cluster headaches may involve pain around one eye, along with drooping of the lid, tearing and congestion on the same side as the pain

©ADAM.

Secondary Headaches

Definable structural, toxic or metabolic abnormality causes the headache

Can mimic primary headache disorders

Pain may be from the eye or any pain sensitive structure in the head, including the eye

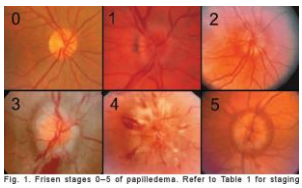


Fig. 1. Frisen stages 0–5 of papilledema. Refer to Table 1 for staging

Ocular Causes of Pain

Corneal disease or desiccation

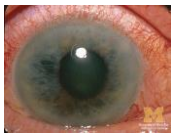
Inflammation

Elevated IOP

Chronic ischemia

Accommodative spasm

Optometric evaluation of headache patients requires ruling out all of the above



Case Report

70 yo male reports pain in the right eye that radiates backward

Fees vision is distorted

Pain is described as a pressure sensation that radiates backward

Denies scalp tenderness or jaw claudication

Problem is intermittent; no provoking features

No significant medical problems

H/O severe MGD with right lower lid ectropion

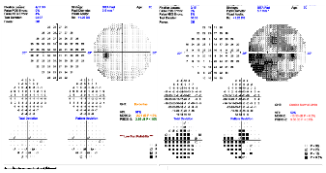
Exam Findings

BVA 20/40 OD; 20/20 OS

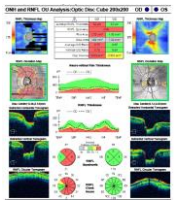
2+ Right APD

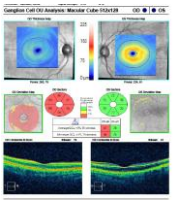
SPK is noted bilaterally; worse OD, with LL ectropion

IOP 23 OD; 18 OS



Additional Findings





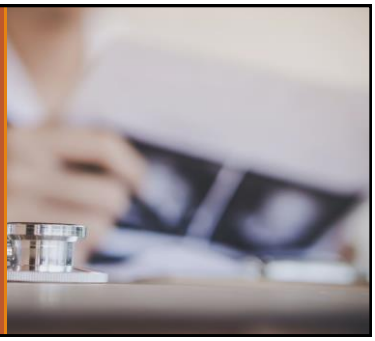
MRI Demonstrates Enhancing Lesion of the Right Optic Nerve

Patient was referred for urgent diagnostic work up

IOP found to be 50mmHg OD; 40mmHg OS

Non-Ocular Causes of Pain

THESE REQUIRE CAREFUL AND THOROUGH CASE HISTORY TO LOOK FOR CHARACTERISTICS THAT AID IN DIAGNOSIS

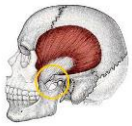


Dental Pain

Exacerbated by chewing or temperature

TMJ

- Exacerbated by jaw movement



Case Report

A 38 year old female is referred for evaluation of headaches

She reports onset several months earlier

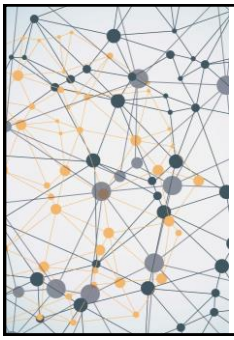
Pain is unprovoked

Pain is nearly constant and is worse when she bends over

She saw her PCP who prescribed Flonase but she discontinued after 2 days bc it gave her a bloody nasal discharge

No FH of headache disorder

Otherwise healthy and takes no other medications; but she is moderately obese



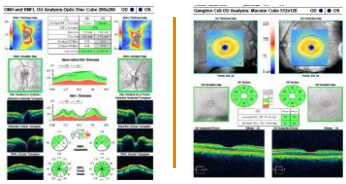
What is the significance of the worsening when bending over?

Additional History

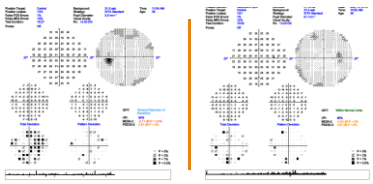
- Headaches are not accompanied by photophobia or sonophobia
- No nausea or vomiting
- Worse on awakening
- Feels a strong pressure sensation
- Frequent pulsatile tinnitus; mostly in the evening; no diplopia or TVO

Exam Findings

- BVA 20/20 OD and OS
- PERRLA; APD neg
- Motility: pursuits, saccades, vergence intact; no nystagmus
- SLE: anterior segment and adnexae intact
- Fundus: clear media, pink, healthy optic nerves with distinct margins; healthy vessels with (+)svp
- Neurologic Exam reveals no disruptions to motor function, reflexes, gait/balance/coordination, sensations or cranial nerves



OCT



Visual Fields

What Kind of Headache?

- ASTHENOPIA?
- PRIMARY
- SECONDARY

Asthenopia?

No exacerbation with near work

Wakes up with them

Primary?

No migraine characteristics

No FH

Wakes up with them

Secondary? Red Flags?

How would you manage this patient?

Impression/Plan

Presentation consistent with chronic sinusitis

Patient advised to restart Flonase

RTC 3 weeks

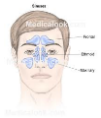
Patient called 3 weeks later to report headaches improved with regular use of Flonase; she canceled her f/u

Paranasal Sinuses

Worse in the am or when bending over

Sphenoid, ethmoid and maxillary sinuses can all cause periocular pain

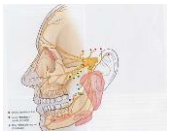
Infections are most common



Neuropathic Pain

Post herpetic neuralgia
- Following HZV dermatitis
- Burning with short, stabbing pains triggered by light touch

Trigeminal neuralgia
- Unilateral jolts of severe, electric shock-like pain
- Triggered by eating, talking, light touch, breezes, brushing teeth



Intracranial Pressure

Increased ICP
- Headache when pressure is very high or acute elevations
- Often accentuated in neck, shoulders, upper back
- Other signs of increased ICP

Decreased ICP
- ICP below 90mmH20

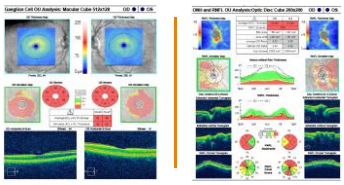
Case Report

48 yo obese female with a history of PTC has a new onset of HA

HA is constant, involve the base of her head, and she "feels out of it"
- Feels different from her ICP related HA

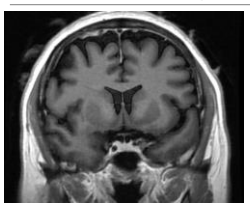
Atrophic papilledema that has been stable for 10+ years

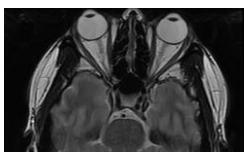
Takes 1g of Diamox daily and has done so since her PTC dx



Has her papilledema returned?

Change in HA pattern/quality warrants additional testing





Patient Referred to PCP for Further Evaluation and Management

PCP feels dehydration may be a factor

Advises increase in sodium/fluids with OTC pain meds

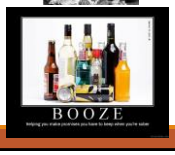
Headaches resolved

Toxic or Metabolic Origin

Toxic offenders include alcohol, caffeine, nitroglycerin, nitrites, niacin, MSG, analgesics, ergotamine

Excessive consumption or withdrawal

Metabolic causes include hypoxia, altitude, air pressure decompression, electrolyte disturbances



Optometric Approach to Headaches

Case History
- It must be thorough and detailed
- Include medications, social and lifestyle habits

Examination
- Check all systems

Additional Testing
- Based on exam findings
- Visual fields

Referral
- To whom?

Case History

Assess frequency, severity, associated findings, triggers, medications, personal habits, family history and medical history

Is the headache pattern consistent with asthenopia or any of the primary headache syndromes?

Are there any "Red Flags" that suggest a secondary headache disorder?

Red Flags

Sudden, severe headache ("worse headache of my life)

New headache pattern

Headache always on same side of head

Ingestion of toxic substances
- New medications

Associated neurologic sx, fever, stiff neck, nausea or vomiting

High risk patients (cancer, pregnancy or post-partum, postoperative, immunocompromised)

Case Report: 58 YO male with headache

Onset 19 days prior

Pain is constant and involves right eye, ear and jaw and is severe

No migraine history or migraine family history

No photophobia/sonophobia

No nausea or vomiting

Additional History

PCP referred him to ER for stat work up
- MRI/MRA (w/o contrast), ESR, CRP
- All negative

No pertinent medical history or medications

He was referred to his dentist to rule out dental origin but no dental issues were found

He presents to the neuro service for neuro-ophthalmic evaluation

Exam Findings

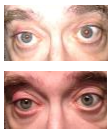
BVA 20/20 OD and OS

2mm ptosis OD

Pupils (see right)
- Dilation lag not observed

Neuro exam normal

Bright (above) vs. dim (below)



Decision-Making

This patient presents with a significant history of pain along with ptosis and miosis that point to the possibility of Horner's syndrome. While there is no dilation lag noted, we did not feel comfortable with his level of pain and felt that the ptosis and miosis was enough to give us a high suspicion of Horner's.

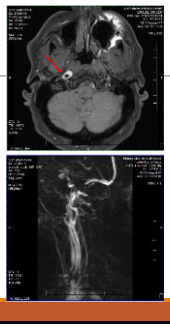
What is the most important rule out in a patient who presents with a painful Horner's?

Plan

Following a phone consult with the radiologist, this patient referred for *contrast-enhanced* MRA of the carotid arteries

Results are shown

The arrow points to an area of post contrast enhancement of the right carotid artery



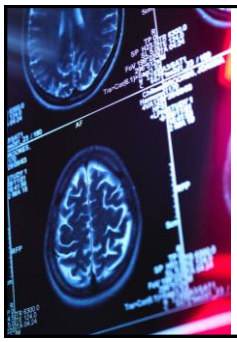
"Optometric" Red Flags

Visual field defects
- Especially homonymous or bitemporal hemianopsia

Papilledema

Pupillary disturbances

Brainstem signs/sx
- Diplopia
  - CN palsies
  - INO
- Nystagmus



Examination/Additional Testing

A thorough examination and work-up should be done to rule out anterior and posterior segment disease, increased IOP, ischemic disease, accommodative spasm, orbital disease, dental origin, sinus origin

Documentation of visual fields is a good idea to rule out visual pathway disease

Which Patients Require Further Evaluation?

Any patient with history or examination features consistent with secondary headache syndromes

Any patient exhibiting any of the "Red Flags"

Manage or Refer

Depending on your clinical findings:

Treat any visual system issues

Investigate with advanced diagnostic testing (MRI)

Communicate with physician

Refer to appropriate specialist:

PCP

Pain management specialist

Management of Migraine

- Establish diagnosis confidently
- Reassurance of a benign condition
- Good doctor-patient relationship
- Education about the condition, setting realistic treatment goals and involving the patient as an active participant in therapy
- Headache diary

Non-Pharmacological Management

- identification and avoidance of triggers
- regular sleep, meals, exercise
- avoid tobacco and caffeine
- relaxation training, biofeedback, etc.

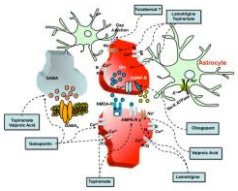
Many patients do not want to take medications for their headaches!

Pharmacologic Management

Prevention
- Decreases frequency

Acute therapy
- Treat the pain when it happens

Approach is often a combination of both



Traditional Prevention Methods

COMMON TRIGGERS

MEDICATIONS

Beta-blockers: effective in reducing blood vessel dilation that occurs during migraines.

Antidepressants target serotonin pathways

Antiseizure medications block electrical impulses in nerves and brain cells.

Sedatives block pain signals

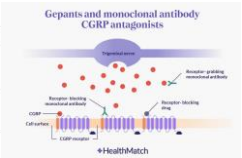
Botox was used to help reduce pain signals being transmitted to areas like the forehead and scalp.

.

New Preventative Therapy: Gepants

CGRP antagonists

Drug Name	Administration	Other information
Qulipta (atogepant)	Oral tablets	• Trials show that most patients have a 50-100% reduction in days with migraine over 12 weeks.[24] • Possible side effects: Reduced appetite, weight loss, elevated liver enzymes, nausea, and constipation.[25]
Nurtec ODT (rimegepant)	Oral disintegrating tablets	• Nurtec ODT is also used for acute migraine treatment (see above table for more details). • It can be taken every other day for long-term management of chronic migraine.



New Preventative Therapy: Monoclonal Antibodies (targeted CGRP antagonists)

Drug Name	Administration	Other information
Vyepti (eptinezumab-jjmr)	Intravenous infusion	• Administered once every 3 months. • One trial showed that patients had a 75% reduction in days with migraine after 3 months of treatment.[2] • Possible side effects: Mild cold symptoms.
Emgality (galcanezumab-gnlm)	Subcutaneous injection	• Administered once a month through self-injection. • Clinical studies show that up to 60% of people with episodic migraine (less than 15 days a month) had at least half their usual number of migraine days after 6 months of treatment.[3] • Possible side effects: Redness, itchiness, pain, and tenderness
Ajovy (fremanezumab-vfrm)	Subcutaneous injection	• Administered once monthly or once every 3 months through self-injection. • In one study, 37.6% of patients managed to have at least a 50% reduction in monthly migraine episodes.[4] • Possible side effects: Itching, rash, and hives.[4]
Aimovig (erenumab-aooe)	Subcutaneous injection	• The first monoclonal antibody CGRP antagonist to be approved. • Administered once a month through self-injection. • 40-60% of patients reported at least a 50% reduction in migraine days after 6 months of treatment.[5] • Possible side effects: Injection site reactions, muscle cramps, and muscle spasms.[5]

Part 1: Basics and effectiveness.

Homeopathic Agents for Migraine Prevention

Magnesium is associated with serotonin release and regulation of vascular tone

300mg BID

Some headache treatment centers offer infusions

Riboflavin reduces inflammation and oxidative stress

Reasons for Failure of Preventative Therapy

Not enough time given
- Should be prescribed with acute medication initially

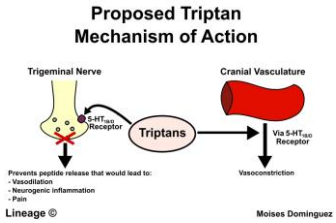
Taken as an acute medication rather than prophylactic

Patient believes it was inappropriately prescribed
- Due to multiple uses

Acute Therapies

Triptans
- Target multiple serotonin receptors (5-HT1B and 5-HT1D) found in the brain and blood vessels
- Blood vessel constriction makes it unsuitable for those with conditions like heart or vascular disease

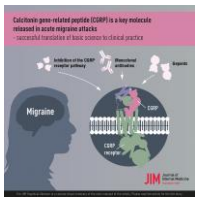
NSAIDS, opioids, barbiturates
- Target pain pathways
- The latter two are habit forming which can limit usage



Newest Migraine Acute Therapies:

Ditans (Serotonin (5-HT)1F receptor agonists):
- Works by binding to brain cell receptors to stimulate serotonin

CGRP inhibitors
- Used for both prophylaxis and abortive approach



Ditans

Reyvow
- Binds to the 5-HT1F receptor mainly within the brain
- Prevents release of CGRP
- Muscle around blood vessels does not have the 5-HT1F receptor
  - Vasoconstriction does not occur
- Can cause drowsiness

REYVOW®
(lasmiditan) Ⓒ
tablets 50mg,100mg

Drug Name	Administration	Other information
Nurtec ODT (rimegepant)	Oral disintegrating tablets	• One of the newest gepants to be approved by the FDA • As it dissolves under your tongue, it acts faster than swallowed pills • Can help to reduce migraine symptoms within 2 hours of taking medication. • One study showed that it helped to relieve most bothersome migraine symptoms in 35.1% of patients. [9] • Possible side effects: Nausea, stomach pain, and indigestion.
Ubrelvy (ubrogepant)	Oral tablets	• First CGRP antagonist to be approved by the FDA in 2019 • It used to be only available as an injection. The pill format now makes it much more convenient and accessible • 64-91% of patients reported regaining normal function within 2 hours of taking Ubrelvy [10] • Possible side effects: Sleepiness, dizziness, nausea, and dry mouth.

Acute CGRP Inhibitors

NOTE THAT NURTEC IS ALSO USED FOR PREVENTION

There have also been Triptan Updates that make them more selective for Serotonin (5-HT)1F receptors

Drug Name	Administration	Other information
Tosymra (sumatriptan)	Nasal spray	• Use as needed, but no more than 30 mg within 24 hours (doses must be at least an hour apart) • A clinical study showed that 57% of patients were relieved from pain within 2 hours of taking Tosymra [42] • Possible side effects: Can range from mild (nausea, nose irritation) to severe (like very high blood pressure or chest pains).[41]
Zembrace SymTouch (sumatriptan)	Subcutaneous injection	• Use as needed, but no more than 12 mg within 24 hours (doses must be at least an hour apart) • One study has shown that it was effective in reducing pain in 70% of patients within 1 hour of taking the medication. [43] • Possible side effects: Can range from mild (drowsiness or tingling) to severe (like chest pains)
Onzetra Xsail (sumatriptan)	Nasal powder	• Use as needed, but no more than 44 mg (2 doses) within 24 hours (doses must be at least 2 hours apart). • Clinical studies have shown that 68% of patients have reduced migraine symptoms within 2 hours after the first dose. [41] • Possible side effects: Ranges from mild (unusual taste in mouth) to severe (like chest pains).

Medication overuse headaches

- Also referred to as rebound or withdrawal headaches
- Insidious increase in headache frequency
- Predictable headache hours to days after last dose; awakening with headache
- Irresistible use of medication at regular intervals

Medication overuse headaches

- Headaches fail to respond to alternative symptomatic medications
- Failure of prophylactic medications
- Physiological and/or psychological dependency

Treating Medication overuse headaches

The best treatment is PREVENTION!

Avoid prescribing combination analgesics, narcotics, or ergotamine in patients with headaches more than 1-2 days per week

Counsel patients regarding caffeine in beverages and in over-the-counter medications

Also occurs with triptans and possibly with simple analgesics and NSAID's

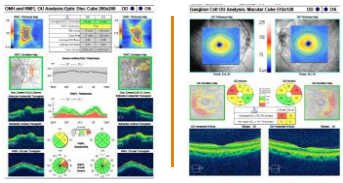
Case Report

32 yo female with a history of obesity related PTC

Chronic papilledema has been responsive to Diamox, 500mg/day

Has persistent headaches despite control of ICP

- Topamax was utilized to address both her headaches and her ICP. It was poorly tolerated
- Tricyclic antidepressant was not effective



OCT Findings

Is there a better way to address her presentation?

Plan

NURTEC WAS ADDED TO HER THERAPEUTIC REGIMENT WITH GOOD RESPONSE

Case Report

- 21 year old female complains of headaches
- Throbbing pain in the back of her head
- Seen in the ER 2-3 months prior and diagnosed with migraines
- Since then, headaches have been increasing in severity and are nearly constant
- Uses OTC Tylenol and Excedrin with minimal effect

Additional History

Headaches are accompanied by photophopia without sonophobia

Also has eye pain that worsen with eye movements
- History of binocular instability 2 years prior with course of VT
- Had episode of gaze evoked visual blur during her VT treatment period

Patient consulted with a neurologist who prescribed Naproxen, an unspecified anxiolytic, and Ibuprofen.
- She did not comply due to gastric discomfort

Patient also admits to severe anxiety that keeps her awake at night
- Does not wish to return to her neurologist because she felt he made her anxiety worse

What do you think of the treatment she was given?

Social History

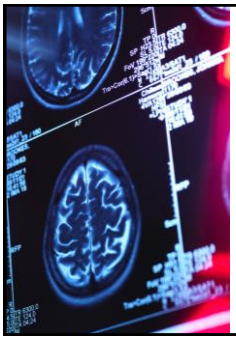
College student

Having great difficulty concentrating in school due to lack of sleep

Moderate marijuana use

Patient is agitated and frequently cries during the examination

She was referred to the Neuro-Op Service for evaluation of her headaches



Exam Findings

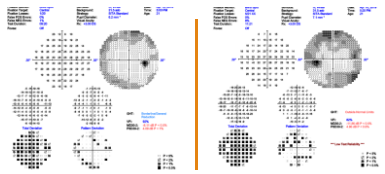
BVA 20/20 OD and OS

PERRLA; APD neg

Motility: Intact pursuits, saccades, vergence; no nystagmus
- Severe pain on eye movements

Ocular Health: healthy anterior segment and adnexae; clear media; pink, healthy optic nerves with distinct margins

Neurologic screening
- Alert/oriented x 3
- Motor/reflexes/gait/balance normal
- Sensory and cranial nerves were not done



Visual Fields

Visual Field Interpretation

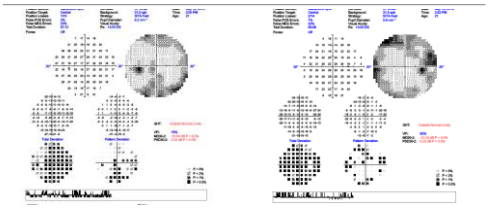
IS IT RELIABLE?

DO THE DEFECTS FOLLOW A PATTERN?

ARE THEY RELEVANT TO THE CASE?

SUGGESTIONS?

Repeat Visual Field



What type of headaches?

ASTHENOPIC?

PRIMARY?

SECONDARY?

Rationale for Asthenopia

H/O binocular instability

Has had eye movement discomfort as part of this presentation

Rationale for Primary Headache Disorder

History of migraines

Are there any triggers or exacerbating features?

Could tension be an issue?

Secondary Headache Disorder?

Are there any red flags?

How should she be managed?

WHAT THERAPEUTIC APPROACH WOULD BE BEST?

Referral to pain management specialist

Pain cycle needs to be broken

- Butalbital was given short term

Anxiety needs to be addressed

- The related lack of sleep may be contributing to her headaches
- She was referred to social work so she could receive appropriate care
- Her pain management strategy revolved around using medications that address both the pain and anxiety.

Venlafaxine (SNRI) was prescribed along with Sumatriptan for episodic pain.

Case Report

45 yo female reports headaches x 15 years

Headaches are unilateral and moderate (6 out of 10)

Photophobia is frequently associated

There has been a recent increase in frequency; now gets them daily and no relief with meds

Continued

Physician is treating her with Acetaminophen/Hydrocodone combo

Uses several times per week without relief

Sometimes uses the medication to prevent headache

No identifiable triggers

Exam Findings

BVA 20/20 OD/OS

Pupils, color, VF, fundus all normal

Neuro exam normal

What Kind of Headache Disorder Does She Appear to Have?

ARE THERE ANY RED FLAGS?

Discussion

Given the longstanding nature of the headache and the accompanying sx, migraine seems likely

On the other hand, she has had a change in her headache pattern, whereby her headaches are now occurring on a daily basis. While a change in headache pattern is considered to be a red flag, it can be explained by her overuse of a combination medication that is a high risk for chronic daily headaches.

- It's always wise to ask about the medication frequency because this is usually what sends patients into a daily headache pattern

How Should She Be Managed?

WHICH PAIN MANAGEMENT STRATEGY IS BEST?

Plan

We were concerned with the habit forming nature of the medication she was using and referred her to a pain management specialist

Follow Up

Patient returns for f/u 6 months later

Her doctor stopped the medication she was using and initiated Midrin, which was very effective in alleviating her pain

Now uses monthly for episodic pain that occurs with menses

What is Midrin (acetaminophen, dichloralphenazone, and isometheptene)?

Acetaminophen is a pain reliever and a fever reducer.

Dichloralphenazone is a sedative that slows the central nervous system and can disrupt the central pain pathways.

Isometheptene causes narrowing of blood vessels (vasoconstriction) and helps break the cycle of neuro-peptide release that occurs with vasodilation.

The prescribed medication had a different mechanism of action than her prior medication and was therefore, very effective at breaking her pain cycle. Because of that, she was able to cut back dramatically on frequency of use because her headaches had returned to their previous frequency. This kept her from needing prophylaxis

Case Report

18 year old female with headaches

Longstanding h/o migraines that occur 4-7 days out of the week

Started in high school and progressively got worse.

Severity is 20 out of 10; sometimes can't leave her bed

Accompanied w/ flashes of lights and starbursts.

Takes Excedrin migraine for the HA w/ some relief.

Additional History

(+) Pulsatile tinnitus - happens episodically and frequently .

(+) Diplopia - hard to catch it as it's coming and gives her HA.

Went to PCP last and had blood work done-awaiting results.

Pt. is currently on wait-list for vision therapy.

Exam Findings

BVA 20/20 OD/OS

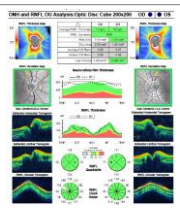
PERRLA; APD neg

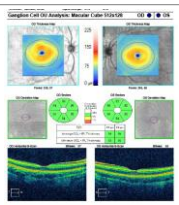
Motility: intact pursuits, saccades, vergence; no nystagmus

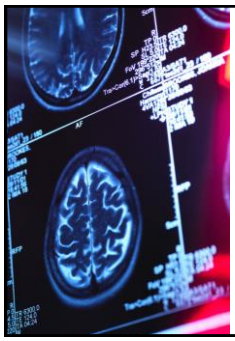
SLE: Healthy Anterior segment and adnexae

Fundus: Clear media; pink, healthy optic nerves with elevated, indistinct margins; (-) SVP

OCT







Headache Type?

Asthenopia?

Diplopia
- Precedes the headache

Intermittent RET

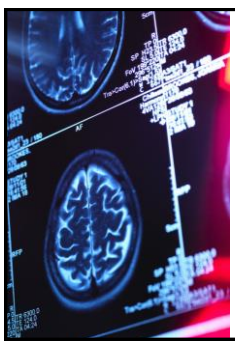
Migraine?

Longstanding

Photopsias

Secondary?

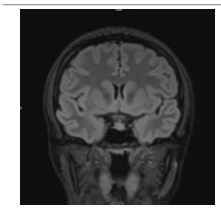
Red Flags?

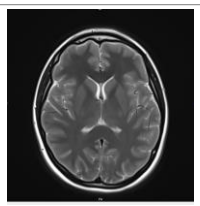


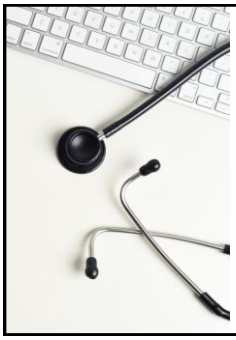
How Should We Manage?

SHOULD SHE HAVE AN MRI?

MRI Results





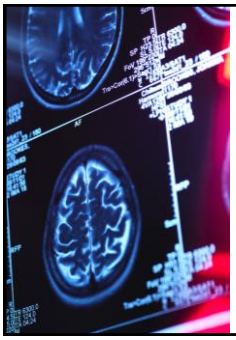


Management

MRI was done; normal

Patient was referred to a pain management specialist
- Magnesium 300mg BID with Riboflavin
- Triptan prn

Will continue her vision training.



Summary

Are the headaches related to an abnormality of the visual system?
- Is there evidence of asthenopia?

Does it sound like a primary headache disorder?
- Are there any "red flags"?

Counsel patients on their medications and non-medical therapies

Refer to headache specialists when necessary
- Most are managed well by PCPs but troublesome cases should see specialists