Disclosures

- No relevant financial relationships to disclose
OUTLINES

- Epidemiology
- Classification
- Evaluation
- Pathogenesis of migraine
- Different types of migraine and other headaches
- Treatment
**EPIDEMIOLOGY**

**HEADACHE PREVALENCE**

- By 3 years of age - 3-8%
- By 5 years of age - 19.5%
- By 7 years of age 37-51.5%
- By 15 years of age 57-82%
- Slightly higher prevalence in boys ages 3-11 years
- Boys -stable prevalence from 7 to 14 years of age and decline in prevalence thereafter
- Girls - increase in prevalence from 7 to 22 years of age
EPIDEMIOLOGY

MIGRAINE

- Up to 7 years of age - prevalence 2% to 3.2%
  - males > females

- Between 7 and 11 years of age - 4% to 11%
  - males = females

- Adolescents above 11 years - 8% to 23%
  - females > males
Two major categories of headaches:

- **Primary** - migraine, tension-type headaches, cluster headaches and other trigeminal autonomic cephalgias, and other primary headaches
- **Secondary** - attributed to the other underlying disorder
SECONDARY HEADACHES

ACUTE GENERALIZED

- Systemic infection
- CNS infection
- Toxins: lead, CO2
- Post-seizure
- Electrolyte imbalance
- Hypertension
- Hypoglycemia
- Temporomandibular

- Post-lumbar puncture
- Trauma
- Embolic
- Vascular thrombosis
- Hemorrhage
- Collagen disease
- Exertional
- Shunt malfunction
SECONDARY HEADACHES

ACUTE LOCALIZED

- Sinusitis
- Otitis
- Ocular abnormality
- Dental disease
- Trauma
- Occipital neuralgia
- Joint dysfunction
COMMON CAUSES of CHRONIC PROGRESSIVE HEADACHES

- Hydrocephalus
- Neoplasm
- Malformations (eg. Chiari/Dandy-Walker)
- Infection: abscess, chronic meningitis
- Subdural hematoma
- Pseudotumor cerebri
- Aneurysm/vascular malformation
- Hypertension
- Medication (eg, birth control, tetracycline)
Headaches in Pediatric ED

Etiology

- Upper respiratory infection 57%
- Migraine without aura 18%
- Undetermined cause 7%
- Serious neurologic problems 17.5%
  - Viral meningitis 9%
  - Brain tumors 2.6%
  - Shunt malfunction 2%
  - Intracranial hemorrhage 1.3%
  - Postictal HA 1.3%
  - Trauma 1.3%
HEADACHES IN PEDIATRIC ED

- 1% of pediatric ED visits
- 40% primary headaches
- 75% of these migraine
EVALUATION

Major goal is to exclude secondary headaches
EVALUATION

HISTORY

- Pain quality, severity, frequency, duration
- Associated symptoms
- Onset triggers or symptoms
- Impact of the disease including an assessment of disability and quality of life
- Response to previous treatments
- Analysis of the child’s and parents’ perception of the etiology
- Thorough family history
Address symptoms of increased intracranial pressure or progressive neurologic disease
- nocturnal occurrence
- ataxia, lethargy
- seizures, visual disturbances
- focal weakness
- personality change, loss of intellectual abilities
EVALUATION

Address potential comorbid or aggravating features

- anxiety, tension, depression and nervousness
- school function
- previous medical problems
- previous medications for both HA and other disorders
- drug and alcohol use
- sleep problems
RED FLAGS

Ominous HA etiologies should be explored if

- HA severity has increased dramatically;
- HA awakens child from sleep;
- change in an established HA pattern;
- “first or worst” HA
EXAM

General Physical Examination:
BP abnormalities, neurocutaneous stigmata, skull and neck, sinuses and jaws exam, signs of trauma or nuchal rigidity, growth charts

Comprehensive Neurologic Exam:
Head circumference, optic fundi, eye movements, strength, reflexes, and coordination
LABORATORY TESTS

- For primary headaches, generally not warranted
- All children with “serious” problems exhibit neurologic symptoms or clear-cut abnormalities on the examination
WHEN TO IMAGE?

- First or worst headache
- Headache pattern changes dramatically
- Progressive headache
- Nocturnal and/or early morning headaches
- Valsalva provokes or aggravates headache
WHEN TO IMAGE?

- Abnormal neurologic signs: focal weakness or numbness, abnormal eye movements, head tilt, diplopia, imbalance, confusion, incoherent speech, seizure
WHEN TO IMAGE?

- Abnormal neurologic exam: papilledema, focal motor or sensory changes (objective)
- Patient with cancer, HIV, immunosuppression
WHICH STUDY?

- MRI is ideal
- CT if suspect bleeding or fracture or MRI is not feasible
- LP if suspect SAH or increased ICP
- EEG- not indicated unless seizures are suspected
Migraine is a primary episodic headache disorder characterized by a cascade of events that involve various combinations of neurologic, gastrointestinal, and autonomic changes.
Two major categories:

- Migraine without aura - previously common
- Migraine with aura - previously classical
MIGRAINE WITHOUT AURA

Most prevalent form in children and adolescents

Unique features in childhood:
- Shorter duration with sleep included as part of the duration
- Increased incidence of bilateral location—especially frontal and bitemporal with exclusive occipital HA being of increased concern
- Ability to include parental observation of photo- and phonophobia
- Pulsating quality of pain is not easily described by children

As in adults, pain is moderate to severe, aggravated by routine physical activity and associated with nausea and/or vomiting.
MIGRAINE WITH AURA

Less frequent in children and adolescents

Aura:
visual
sensory
dysphasic
VISUAL AURA

- Brightly colored lights or moving lights like light bulbs going off everywhere
- Less frequently-distorted images, bizarre visual illusions, or spatial distortions, scotomata, visual field defects, or fortification spectra
- Adult type aura starts during adolescence
SENSORY AURA

Usually unilateral

Parasthesias, sensation of pins and needles
Sometimes described as worms or bugs crawling up the hand to the arm and finally to the face followed by a numb sensation
Dysphasic aura is the least frequent, described more as difficulty speaking than lack of understanding.

This type of aura could explain some of the complex or confusional migraine.
MIGRAINE WITH BRAINSTEM AURA

- Previously used terms:
  Basilar artery migraine; basilar migraine; basilar-type migraine.

- Migraine with aura symptoms clearly originating from the brainstem, but no motor weakness.

- Dysarthria, vertigo, tinnitus, hypacusis, diplopia, ataxia, decreased level of consciousness
MIGRAINE WITH BRAINSTEM AURA

- Visual symptoms simultaneously in both temporal and nasal fields of both eyes
- Simultaneously bilateral parasthesias
- The syndrome affects adolescents more frequently than adults
- Typical aura may be present as well
PATHOPHYSIOLOGY OF MIGRAINE

BASIC CONCEPTS

- Neuronal hyperexcitability during the interictal phase
- Primary dysfunction related to centers in brainstem that regulate vascular tone and pain sensation
PATHOPHYSIOLOGY OF MIGRAINE

- Cortical spreading depression (CSD) as a basis for aura
- Trigeminal nerve activation at a peripheral and central level accounts for headache
- Important role of vasoactive peptides – serotonin (5-HT) and calcitonin gene-related protein (CGRP)
Trigeminocervical complex=TCC; periaqueductal gray=PAG; pontine locus coeruleus=LC; nucleus raphe magnus=NRM. Reproduced from Goadsby PJ. Neurol Clin. 2009;27:335-60
MIGRAINE TRIGGERS

- Inadequate or altered sleep, excessive sleep
- Skipping meals
- Stress or concentration
- Weather changes
- Bright light, and loud noises
- Dehydration
- Adolescent girls may begin to have a menstrual pattern
FOOD TRIGGERS

- Role of specific food triggers has called into question

- However, if a consistent pattern of HA after particular food item is present, this should be discussed with the patient
50-90% of relatives also have migraine

Whether it is inherited as an AD with variable penetrance or is a complex multifactored hereditary disorder has not been determined

Only migraine genes identified are FHM: also seen in families with migraine with aura and migraine without aura
FAMILIAL HEMIPLEGIC MIGRAINE

- Migraine with aura including motor weakness, and at least one first- or second-degree relative has migraine aura including motor weakness.
FAMILIAL HEMIPLEGIC MIGRAINE

Three genes identified

- CACNA 1A (coding for a calcium channel) on chromosome 19p13 - responsible for approximately 50% of all FHM families

- The ATP1A FHM2 gene (coding for a K/Na-ATPase) on chromosome 1q23

- The SCN1A gene (coding for a sodium channel) on chromosome 2q24
COMPLICATIONS OF MIGRAINE

- Status migrainosus
- Persistent aura without infarction
- Migraine infarction
- Migraine aura-triggered seizure
EPISODIC SYNDROMES THAT MAY BE ASSOCIATED WITH MIGRAINE

- Previously used terms: childhood periodic syndromes; periodic syndromes of childhood.
- Migraine precursors
- Diagnoses of exclusion
- Family history of migraine common
- Migraine preventive medications can be effective
ADDITIONAL CONDITIONS

- Motion sickness
- Periodic sleep disorders including sleep walking, sleep talking, night terrors and bruxism
CYCLIC VOMITING SYNDROME

- 2.5% of schoolchildren
- Less common in adolescents
- Recurrent and stereotyped episodes of intense but otherwise unexplained nausea and vomiting which last 1 hour to 10 days in children free of symptoms interictally
- Vomiting occurs at least four times an hour, and no signs of GI disease can be found
- HA is not usually part of this syndrome
- Migraine prophylaxis can be effective
ABDOMINAL MIGRAINE

- 12% of schoolchildren
- Recurrent attacks of abdominal pain lasting 2-72 hours
- Midline location, periumbilical or poorly localized
- Dull or “just sore” (moderate or severe)
- Anorexia, nausea, vomiting, and/or pallor
- No HA or head pain
- PE and investigations exclude other causes
- Many children evolve into migraine, and migraine treatment may be effective
BENIGN PAROXYSMAL VERTIGO

- Not uncommon- in up to 2.6% of children
- Recurrent attacks, each of multiple episodes of severe vertigo resolving spontaneously after minutes to hours
- Nystagmus, ataxia, vomiting, pallor, fearfulness
- Consciousness is not lost
- Neurologic examination and audiometric and vestibular functions are all normal between attacks
- EEG normal
- Evolves into migraine
PAROXYSMAL TORTICOLLIS

- Recurrent episodes of head tilt to one side, with or without slight rotation, which remit spontaneously after minutes or days
- Infants and small children, with onset in the first year
- Often associated symptoms- pallor, malaise, irritability, vomiting, ataxia
- EEG, MRI may be necessary to evaluate for posterior fossa disease
- No studies have shown that specific medications are effective in preventing recurrence
TENSION TYPE HEADACHE

- Bilateral location
- Non-pulsating quality
- Mild to moderate intensity
- Lack of aggravation by routine physical activity
- Not accompanied by nausea, although just one of photo or phonophobia doesn’t exclude the diagnosis
TENSION TYPE HEADACHE

- Duration is an important criterion but hard to obtain this history in children

- The basic mechanisms of different headache types are unclear and overlapping symptoms are frequent

- The distinction between TTH and migraine may, therefore, be difficult, especially in children
CHRONIC DAILY HEADACHE

One of the most misunderstood and hard to treat headache conditions

Collective term, not a diagnosis
PREVALENCE

- Headache specialty clinics - 30%
- General population - 4%
- Pediatric patients - more difficult to define
DIAGNOSIS

Presence of headache for at least 15 days in a month period over a period of three consecutive months and with no underlying organic pathology
CLASSIFICATION

Primary

duration >4 hours

duration <4 hours

Secondary
PRIMARY HEADACHE

- Transformed or chronic migraine
- Chronic tension type headache
- New daily persistent headache
- Hemicrania continua

Silberstein et al.
PRIMARY HEADACHE

Duration < 4 hours

- Cluster headache
- Chronic paroxysmal hemicrania
- Short-lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT)
- Hypnic headache
- Idiopathic stabbing headache
SECONDARY HEADACHE

- HA associated with nonvascular intracranial disorders (intracranial hypertension, infection EBV, HIV), neoplasm

- Other
NEW DAILY PERSISTENT HEADACHE

Abrupt development of HA that does not remit
Develops over <3 days
Persists >15 days/month for >1 month
Frequently caused by viral illness
Absence of past history of HA
Often resistant to treatment
Migraine is currently conceptualized as a chronic disease with episodic manifestations.

Three partially overlapping forms:
- clinical
- physiologic
- anatomic
RISK FACTORS

- High HA frequency
- Female gender
- Obesity (BMI>30)
- Snoring
- Stressful life events
- Migraine
RISK FACTORS

- High caffeine consumption
- Acute medication overuse
- Depression
- Head or neck injury
- Less than high school education
PROGNOSIS

- CDH often remits spontaneously
- One year remission rate 14% in general population
- Poor prognosis associated with older age, long duration of CDH and medication overuse
MEDICATION OVERUSE HA (REBOUND)

- HA are accompanied by asthenia, nausea, and other GI symptoms, restlessness, anxiety, irritability, memory problems, difficulties in intellectual concentration, and depression
- Drug-dependent rhythmicity of HA
  Predictable early morning HA are frequent
MEDICATION OVERUSE HA (REBOUND)

- There is evidence of tolerance to analgesics over time, with patients needing progressively larger doses
- Withdrawal symptoms if patients are taken off pain medications
- Concomitant prophylactic medications are ineffective while patient consumes excess amounts of immediate relief medications
MEDICATION OVERUSE
HEADACHE: HOW MUCH IS TOO MUCH?

According to study by Bigal and Lipton
5-8 days per month of use of opiates and barbiturates associated with migraine progression
MOH: HOW MUCH IS TOO MUCH?

- Triptans induced migraine progression in those with high frequency of migraine at baseline (10-14 days per month) but not overall.
Anti-inflammatory medications were protective in those with <10 days of HA at baseline, and, as triptans, induced migraine progression in those with high frequency of HA.

High frequency of HA seems to be a risk factor for chronic migraine regardless of medication exposure.
GOALS

- Early, rapid treatment
- Use of proper formulation and dose based on the child’s weight
- Limited use of rescue medications to avoid MOH
- Optimization of self-care
- Minimal to no adverse effects
- Cost effectiveness
TREATMENT

- Pharmacological
- Non-pharmacological
PHARMACOLOGICAL TREATMENT

VERY FEW MEDICATIONS THAT ARE USED IN TREATMENT OF PEDIATRIC HEADACHE HAVE FDA APPROVAL FOR THE TREATMENT OF PEDIATRIC HEADACHE

ALMOTRIPTAN (AXERT)
RIZATRIPTAN (MAXALT)
MIGRAINE NONSPECIFIC TREATMENT

Acetaminophen 15 mg/kg/dose Q 4-6h max 75 mg/kg in 24 h in children up to 12 years

Ibuprofen 10 mg/kg/dose Q6-8h max 800mg

Excedrin (ASA, acetaminophen, caffeine) 1-2 tab
**MIGRAINE NONSPECIFIC TREATMENT**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Route</th>
<th>Dose Information</th>
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<tbody>
<tr>
<td>Ketorolac</td>
<td>IV</td>
<td>0.5 mg/kg; max 15 mg</td>
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<tr>
<td></td>
<td>IM</td>
<td>max 60 mg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>with subsequent 5 day total oral every 8 hours course</td>
</tr>
<tr>
<td>Steroids</td>
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</tr>
<tr>
<td>Diclophenac</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Naproxen</td>
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</tbody>
</table>
ANTI-INFLAMMATORY

- Used since the time of Hippocrates
- Evidence based and clinical experience-effective for mild to moderate attacks
- Inhibit prostaglandin synthesis
- Aspirin irreversibly inhibit COX 1 and 2
- NSAIDs reversibly inhibit COX 1 and 2
- Limit use to 2-3 times per week
### Migraine Nonspecific Treatment

<table>
<thead>
<tr>
<th>Medicine</th>
<th>Dosage</th>
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<tbody>
<tr>
<td>Acetaminophen + codeine</td>
<td>0.5-1 mg/kg/dose Q 6-8h</td>
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<tr>
<td>Fioricet</td>
<td>1 tab Q 6 hours</td>
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<tr>
<td>Fiorinal</td>
<td></td>
</tr>
<tr>
<td>Oxycodone</td>
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</table>
TAKE HOME MESSAGE

All efforts must be made to avoid narcotic containing medications
BUTALBITAL

- May have some efficacy in acute migraine, but no placebo controlled studies
- Proven anxiolytic effect, but limited evidence for analgesia
- Risk of clinical and psychological dependency
OPIATES

- No evidence of superiority versus triptans or NSAIDs
- May be pronociceptive and lead to decreased efficacy of other migraine abortives
- May contribute to MOH
<table>
<thead>
<tr>
<th>Antiemetics</th>
<th>Dose/Route</th>
</tr>
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<tbody>
<tr>
<td>Promethazine</td>
<td>0.5 mg/kg Q 4-6h</td>
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<tr>
<td></td>
<td>max 25 mg</td>
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<tr>
<td>Prochlorperazine</td>
<td>0.15 mg/kg IV,</td>
</tr>
<tr>
<td></td>
<td>max 10 mg</td>
</tr>
<tr>
<td></td>
<td>PO- 5-10 mg</td>
</tr>
<tr>
<td>Ondansetron</td>
<td>4-8 mg PO/IV</td>
</tr>
<tr>
<td>Metoclopramide</td>
<td>0.2 mg/kg IV,</td>
</tr>
<tr>
<td></td>
<td>max 10 mg</td>
</tr>
</tbody>
</table>
ANTIEMETICS

- Side effects: postural hypotension, drowsiness, extrapyramidal, acathisia
- Antiemetics could be combined both with other migraine non-specific and migraine specific medications
**MIDRIN/DURADRIN**

- Isometheptene – sympathomimetic
- Dichloralphenazone - muscle relaxant
- Acetaminophen
- Cautious use with cardiac risk factors
MIDRIN/DURADRIN

1-2 caps at onset
1 additional cap/h x 1
>50 kg - max 5 caps
<50 kg - max 3 caps
TRIPTANS

- Revolutionized migraine treatment all over the world, changed lives of millions of migraine patients
- Indeed migraine specific medications
- Two already have FDA approval for children- almotriptan -12 years and older, rizatriptan – 6 years and older
TRIPTANS

Continually being studied in children and adolescents

- Well absorbed
- No significant adverse events were noted
- AE – tingling, dizziness, warm/hot sensations, chest or jaw discomfort, and injection site reactions
TRIPTANS

- Selective serotonin 5-HT1B/1D agonists
- Constrict the dilated meningeal vessels through stimulation of 5HT1B on the blood vessel wall
- Inhibit neurotransmitter release and nociceptive transmission by stimulating 5HT1D receptors on central and peripheral trigeminal sensory nerves
SEROTONIN RECEPTORS

Hypothalamus

Trigger Factors

Cortex

Thalamus

Dura Mater

Dorsal raphe

Nucleus

Locus coerulesus

Medulla

C1

C2

Blood vessel

Trigeminal ganglion

Vasoconstriction

Anti-inflammatory action

Triptans

Mechanism of action of triptan drugs

Adapted from Goadsby PJ, Olesen J; Diagnosis and Management of Migraine. Br Med J 1996; 312:1279-1283
Triptans are similar, but pharmacologically heterogeneous and thus have slightly different efficacy profiles. Failure to respond to one agent does not predict failure to respond to another.
TRIPTANS

- Unable to block ongoing sensitization in the second order trigeminovascular neurons
- Should be used before central sensitization has occurred
- They still have some effect after that time probably due to some action at the level of TNC and therefore should be used, though will be less effective
TRIPTANS: CONTRAINDICATIONS

- risk of coronary artery disease
- peripheral vascular syndromes or other significant underlying cardiovascular disease
- uncontrolled hypertension
- atypical headache or hemiplegic migraine
- pregnancy
TRIPTANS

- Oral triptans can be divided into two groups:
  - fast onset with higher efficacy at 2 hours
  - slower onset with lower response rates at 2 hours
GROUP I – FAST ONSET

- Sumatriptan
- Zolmitriptan
- Rizatriptan
- Almotriptan
- Eletriptan

- Imitrex
- Zomig
- Maxalt
- Axert
- Relpax
GROUP II - SLOWER ONSET

Naratriptan
Amerge
Frovatriptan
Frova
SUMATRIPTAN

- Tmax: 2.5 hr
- T1/2: 2-2.5 hr
- Bioavailability: 14%
<table>
<thead>
<tr>
<th>Formulation</th>
<th>Dosage</th>
<th>Age</th>
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<tbody>
<tr>
<td>Oral 25 mg</td>
<td>&lt; 50 kg</td>
<td>Oral, nasal spray, injections</td>
</tr>
<tr>
<td>Oral 50 mg</td>
<td>&gt; 50 kg</td>
<td>Oral, nasal spray, injections</td>
</tr>
<tr>
<td>Oral 1 mg/kg</td>
<td>1 mg/kg</td>
<td>Oral, nasal spray, injections</td>
</tr>
<tr>
<td>Nasal 5 mg</td>
<td>&lt; 50 kg</td>
<td>Oral, nasal spray, injections</td>
</tr>
<tr>
<td>Nasal 10 mg</td>
<td>&gt; 10-11 yrs</td>
<td>Oral, nasal spray, injections</td>
</tr>
<tr>
<td>Nasal 20 mg</td>
<td>&gt; 12 yrs</td>
<td>Oral, nasal spray, injections</td>
</tr>
</tbody>
</table>
ZOMITRIPTAN

- Tmax: 2 hr
- T1/2: 2.5-3 hr
- Bioavailability: 40-48%
ZOMITRIPTAN

Regular/ZMT
- 2.5 mg <12 yrs
- 5 mg >12 yrs

Nasal Spray
- 5 mg >12 yrs
RIZATRIPTAN

- Tmax 1.2-2.5 hr
- T1/2 2-3 hr
- Bioavailability 45%
- Approved for children age 6 or older
RIZATRIPTAN

Regular/ODT  5mg    <40 kg
             10mg    >40 kg
**NARATRIPTAN**

- **Tmax**: 2-3 hr
- **T1/2**: 5-6 hr
- **Bioavailability**: 63% men
  - 74% women
- **Oral**: 1 mg, 2.5 mg
ELITRIPTAN

- Tmax 1-2 hr
- T1/2 4-7 hr
- Bioavailability 50%
- Oral 20 mg, 40 mg
ALMOTRIPTAN

- Tmax 1.4-3.8 hr
- T1/2 3.2-3.7 hr
- Bioavailability 70-80%
- Oral 6.25mg, 12.5mg, 25mg
- Approved for children 12 y and older
FROVATRIPTAN

- Tmax 2-4 hr
- T1/2 24-30 hr
- Bioavailability 24-30 %
- Oral 2.5 mg
TRIPTANS

- The initial dose may be repeated in two hours
- The triptans should NEVER be combined
- Should not be used for more than 6 headaches per month to avoid medication overuse
ERGOT/ERGOT DERIVATIVES

Still play role (and recently larger role) in the acute treatment when the response to triptans is not adequate.

More side effects (nausea, vomiting, peripheral and coronary vasoconstriction) due to relatively nonselective mechanism of action.
ERGOT/ERGOT DERIVATIVES

- Fast onset of action
- Relatively safe
- Have effect in particularly severe and long lasting attacks
DHE
Nasal Spray

0.1 mg/dose 6-9 yr
0.5 mg/dose 9-12 yrs
0.75 mg 12-16 yrs

Can be used IM, SQ, or IV with a concomitant antiemetic
DHE

- Oral spray is studied
- IV DHE is used for prolonged headaches including CDH
- Well tolerated and longer treatments produce a better outcome
COMBINE ABORTIVES?

- They act on the different paths of migraine pathogenesis and thus could work synergistically
- NSAID’s + antiemetics (DOPA receptor antagonists)
- Triptans + NSAID’s
- Triptans + NSAID’s + antiemetics
PREVENTIVE TREATMENT
PREVENTIVE TREATMENT

- After adequate trial of acute treatment if frequent disabling attacks persist
- Headache interferes with daily function
- Acute medication is insufficient or ineffective
- Specific contraindication to using abortive treatment
- Acute medication is overused
COMMON MISTAKES

- Starting dose too high $\Rightarrow$ side effects, medication discontinued

- Starting dose low, staying low and not titrating up to therapeutic benefit $\Rightarrow$ frustration, medication discontinued
COMMON MISTAKES

- Not scheduling regular visits
- Not offering non-pharmacologic modalities
- Not recognizing associated comorbidities, such as depression and anxiety
- The fair trial is not given
POSSIBLE THERAPEUTIC MECHANISMS

- Stabilization of reactive CNS centers
- Enhancement of central nociceptive pathways
- Inhibition of cortical spreading depression
- Inhibition of peripheral sensitization

BUT THEY DO NOT WORK ACUTELY!!
CYPROHEPTADINE

- 5 HT-2 antagonist
- Usually used in children younger than 9 years of age
- A nightly dose 4-12 mg is used, or 0.25 mg/kg/day 1/3 AM-2/3PM
- Common SE- fatigue, weight gain, appetite stimulation
The utility of certain antidepressants for migraine prophylaxis dates from the early 1970s. Effective, and TCAs are more effective than newer SSRIs.
ANTIDEPRESSANTS

- **Non-sedating** are used in patients who initiate and maintain sleep easily - protriptyline and desipramine.

- **Sedating** - amitriptyline, nortriptyline, imipramine - tolerated better by patients who have difficulties to initiate and maintain sleep.
ANTIDEPRESSANTS

- Anticholinergic side effects – dry mouth, blurred vision, urinary retention, constipation, sedation, weight gain
- Start low-10 mg
- Gradual increase is usually well tolerated and effective
- Obtain a baseline EKG to rule out prolonged QTc
BETA BLOCKERS

- May also have anxiolytic effects
- Propranolol and timolol
- Dose – start 1 mg/kg/day up to 3-4 mg/kg/day divided bid
- Contraindicated in patients with bronchospastic disease, diabetes, and WPW syndrome, CHF
- Side effects - depression, fatigue, and decreased athletic endurance
CALCIUM CHANNEL BLOCKERS

- Verapamil, diltiazem, flunarizine (not available in US), nimodipine, and nicardipine
- May be useful in prolonged or atypical migraine
- Should be avoided in second or third-degree AV block, hypotension, ventricular dysfunction, Duchenne muscular dystrophy
- Dose - 80 mg-240 mg
- The most common side effects are daytime sedation, weight gain, depression, and constipation
Interest in the treatment of migraine goes back over 30 years

Renewal of interest over the last decade
TOPIRAMATE

- Has adult indication
- Has been studied in children
- Initiate at 15 mg/day and titrate over 8 weeks to a dose approximately 2-3 mg/kg/day, or the maximum tolerated dose
- Side effects: weight loss, cognitive, paresthesias, metabolic acidosis, glaucoma
- Pregnancy- category D
VALPROATE

- FDA indication in adults
- Monitor blood work
- Dosing
- ER 250 or 500 mg QHS
- 125 mg sprinkle caps QHS
- Pregnancy- category D
OTHER ANTICONVULSANTS

- Gabapentin up to 2400mg/day
- Zonisamide 200-400 mg /day
- Levetiracetum 500-3000mg/day
- Pregabalin 150-450 mg/day

There are limited studies to support their use.
The antiepileptic doses are usually prescribed.
HOW LONG TO TREAT?

- No definite recommendations
- Generally 3-6-12 months
- Individual approach
SUPPLEMENTS

- Magnesium 300-600 mg/day
- Riboflavin 25-400 mg/day
- Feverfew (Tanacetum parthenium) 10-110 mg/day
SUPPLEMENTS

- Butterbur (Petasites hydrides)
  25-75 mg/day
- Coenzyme Q 10
  25-300 mg/day
- Migrelief = feverfew, magnesium, Riboflavin
NONPHARMACOLOGIC THERAPY

- Food triggers may affect 5-15% of patients
- Acupuncture
- Biofeedback
- Cognitive behavior therapy
- Cognitive-behavior therapy, acupuncture INDEED were proven to be EFFECTIVE
TREATMENT

Healthy life style is IMPORTANT!!!!
ALWAYS ADDRESS

- Healthy diet
- Appropriate hydration
- Sleep schedule
- Daily curriculum
- School performance
- Exercise
- Cessation of bad habits
- Caffeine consumption
CONCLUSION

- Headache is a very common problem in pediatrics and in pediatric neurology.
- It requires the collaboration of patients, parents, pediatricians, neurologists, psychologists, school authorities.
- Avoid using narcotic containing medications as an abortive headache treatment.
CONCLUSION

- Treatment of chronic daily headaches is a challenge
- Know the treatment options
- Address the comorbidities
- From the first visit work with the patient and family as a team
- Set realistic expectations
Treatment of headache is not only science, but also ART
THANK YOU
QUESTIONS