Inservice Exam Review
Neurology

Raymond Fowkes MD
Henry Ford Health System
2/9/2012
Headaches

• Primary
  – Migraine
  – Cluster
  – Tension

• Secondary Headaches
  – Critical 2° causes requiring emergency treatment
  – Critical 2° causes not requiring emergency treatment
  – Generally benign / reversible 2° causes
Secondary causes of Headache

• Critical 2° causes requiring emergency treatment
  – Vascular
    • CS thrombosis, AVM
    • Stroke, ICH, SAH
    • Epidural, subdural
  – CNS Infection
    • Meningitis
    • Abscess
    • Encephalitis
  – Tumor with increased ICP
  – Pseudotumor cerebri
  – Ophthalmic
    • Glaucoma
    • Optic neuritis (pain moving eyes)
  – CO poisoning
  – Pre-eclampsia
  – Hydrocephalus

• Critical 2° causes not requiring emergency treatment
  – Tumor, no increased ICP
  – Inflammatory
    • Temporal arteritis
  – Post traumatic

• Benign/Reversible
  – HTN
  – post LP
  – Non CNS Infection
    • Sinusitis, dental
    • Systemic (flu)
  – Drug related
  – Misc:
    • Trigeminal Neuralgia
Questions #1

- A 25-year-old woman comes to your ED complaining of headaches. She’s been having unilateral headaches for about a year, at a rate of approximately two to three episodes a month. The headaches last for 6 to 8 hours, are pulsating, and are accompanied by nausea, vomiting, and photophobia. She also has been experiencing some rhinorrhea with the headaches. The pain is moderate in intensity and gets worse with routine physical activity. She usually gets relief by taking over-the-counter ibuprofen. Her physical examination, including a neurologic examination, is unremarkable. She has heard on the news that headaches can be a sign of a tumor, and she is concerned about this possibility.
On the basis of this patient’s history and physical examination, what is the most likely diagnosis, and how would you evaluate her?

- A. Tension-type headache; further workup is not indicated at this time
- B. Sinus headache; obtain a sinus CT scan
- C. Migraine without aura; no further workup is indicated at this time
- D. Migraine without aura; CT imaging is indicated to rule out intracranial pathology
**Key Concept/Objective: To understand the clinical characteristics and evaluation of migraines**

* Migraine can occur with/without aura
* Duration of ranges from **4 to 72 hours**
* **At least two** of the following pain characteristics: **unilateral** location, **pulsating** quality, **moderate or severe** intensity, and **aggravation by routine physical activity**
* **At least one of** the following symptoms: **nausea with or without vomiting, photophobia and/or phonophobia**
* Forty-five percent of migraineurs have at least one autonomic symptom (i.e., lacrimation, eye redness, or rhinorrhea) during an attack

* **Tension-type** headaches are usually **non-pulsating**
* **Last from 30 minutes to 7 days**, are **mild or moderate** in severity, and are **bilateral** in location.
* There should be no nausea or vomiting
* Photophobia or phonophobia may be present, but not both.

*Answer: C—Migraine without aura; no further workup is indicated at this time*
Migraine Headache

- Onset usually in teens, 5% males, 15% females
- Classic
  - HA with aura, (lasts < 60 min)
- Common (80%)
  - HA, no aura, + N/V, photophobia
- Complex
  - Ophthalmoplegic - EOM palsies + HA
  - Hemiplegic
- Treatment -
  - NSAIDs, Anti-emetics, Steroids (RRR 30%, NNT 10)
  - Vasoconstrictors: Triptans, Ergotamines DHE
  - Narcotics
A 48-year-old man presents complaining of headaches. The headaches are unilateral, located behind his right eye, severe, and are of rapid onset. The patient notes that the headaches are accompanied by right-sided nasal congestion. The headaches usually last for 30 to 40 minutes. The headaches started 3 or 4 weeks ago, and he has two or three attacks a day. The patient does not have a history of headaches. At this time, the headache is 8/10 in severity. The patient has a medical history of hypertension, for which he takes an angiotensin-converting enzyme (ACE) inhibitor. On physical examination, the patient’s blood pressure is 150/78 mm Hg; right-sided ptosis, miosis, and conjunctival injection are present. The rest of the examination is normal.
What is the most likely diagnosis for this patient, and how would you approach the diagnosis?

- A. Cluster headaches; consider obtaining an imaging study to evaluate for secondary headaches
- B. Hypertension; increase dosage of blood pressure medications
- C. Posterior communicating artery aneurysm; get an urgent cerebral angiography
- D. ACE inhibitor–induced headache; stop use of the ACE inhibitor
Key Concept/Objective: To know the clinical manifestations of cluster headaches

- Uncommon 0.4% of population, 5 x more common in males
- **Recurrent headaches (one to eight a day)**
- **Unilateral and severe**: orbital, retroorbital, temporal, supraorbital, and infraorbital
- May alternate sides
- **Rapid onset**, peak in 5 to 10 minutes, usually of short duration **30 to 45 mins**
- **Autonomic symptoms** present in over 97% *lacrimation and conjunctival injection* 80% *ipsilateral congestion* 74%, *partial Horner* syndrome 65%
- Usually diagnosed on the basis of the clinical criteria alone
- Neuroimaging, preferably MRI, considered with following features: a pattern of not conforming to the clinical criteria; age of onset older than 40 years; a progressive pattern of headaches; chronic cluster headache; and any focal neurologic deficit other than Horner syndrome.

- **A posterior communicating artery aneurysm** can cause a SAH and pupillary changes characteristic of third-nerve palsy (i.e., *mydriasis with ptosis*); it would not cause miosis.
- Mild to moderate hypertension does not usually cause headache.
- ACE inhibitors can cause headaches in some patients; however, in this patient, this possibility seems less likely than cluster headaches.

(Answer: A—Cluster headaches; consider obtaining an imaging study to evaluate for secondary headaches)
Cluster Headache

- Severe unilateral facial pain, lasting < 2 hours
- Comes in bursts / clusters – several times a day for weeks/months – 75 % between 9 pm and 10 am
- Precipitated by alcohol, histamine, NTG, stress
- Indomethacin, NSAIDs, 100 % oxygen, triptans, intranasal DHE, intranasal lidocaine or intranasal capsaicin for acute treatment
Temporal Arteritis

• Vasculitis of the branches of the external carotid artery that typically occurs in elderly women

• May be associated with fever, malaise, weight loss, anorexia, diplopia, polymyalgia, jaw claudication (65%)

• Diagnosed with 3 of 5 criteria:
  • New onset H/A
  • Age over 50 yrs
  • Temporal artery tender or with decreased pulse
  • Sed rate $\geq 50$ (usually over 100)
  • Abnormal temporal artery biopsy

• Vision loss due to ischemic optic neuritis if not treated

• Treat with Prednisone 60 mg/day and follow-up for biopsy
Cavernous sinus thrombosis

- Usually secondary to infection ethmoid, sphenoid, maxillary sinus
- **Presentation**
  - Headache, fever, seizures
  - Cranial palsies, papilledema
  - Chemosis, proptosis, ptosis
- **Diagnosis**
  - CTA
  - MRI/MRV
- **Treatment**
  - Antibiotics
  - Consider anticoagulation
You are asked to evaluate a 25-year-old woman for headaches. Her symptoms started 2 months ago with daily frontal bilateral headaches. The headaches are pulsatile and continuous. She also complains of occasional blurred vision. She has no significant medical history. She takes over-the-counter acetaminophen for her headaches. On physical examination, her blood pressure is 120/76 mm Hg. She weighs 200 lb, and she is 5 ft 2 in tall. Her fundoscopic examination shows papilledema. The rest of her examination is normal. You are concerned about her symptoms and order an MRI, which shows no significant abnormalities.
What should be your next step in the management of this patient?

- A. Perform a lumbar puncture
- B. Start a triptan
- C. Start indomethacin
- D. Start a beta blocker as prophylaxis for migraines
Key Concept/Objective: To recognize the manifestations of pseudotumor cerebri

- Onset usually between the ages of 11 and 58 years
- Ninety percent are young, obese women with irregular menses
- Headache is present in 75% or more, papilledema in 95%, cranial nerve VI palsy in 25%, transient visual changes in 70%, visual loss in 30%, and roaring noises in 70%
- Usually pulsatile, continuous, and daily
- Unilateral or bilateral with a bi-fronto-temporal most common
- Nausea in 60% of cases, vomiting in 40%.
- Diagnosis of exclusion, many other causes of papilledema
- MRI is the test of choice. If the brain scan is negative, a lumbar puncture should be obtained
- Opening pressure usually elevated and CSF normal

(Answer: A—Perform a lumbar puncture)
Pseudotumor Cerebri

• Benign intracranial hypertension
• Severe nonspecific headaches in young obese females with abnormal menstrual cycles, female: male 8:1
• CT - head is normal
• LP: high opening pressure, relieves pain, do opening and closing pressures
• Treat with acetazolamide, steroids, occasional shunts or serial LPs, optic nerve sheath fenestration, cerebral sinus stenting, weight loss or bariatric surgery
A 73-year-old woman comes to the emergency department with a sudden onset of confusion. She had no LOC or focal neurologic findings. She is independent in her activities of daily living. Her medical history is unremarkable except for a remote history of hypertension, and she is currently taking no medications. Her examination reveals a blood pressure of 140/84, a heart rate of 108 beats/min, and a temperature of 98.6° F (37° C). Her neck reveals no bruits, and her heart examination reveals an irregular rhythm without murmurs or extra sounds. Cranial nerve, sensory, motor, cerebellar, and reflex examinations are normal. Mental status examination reveals an intact ability to follow commands and verbal fluency but difficulty in associating meaning with words.
Which of the following is the most likely explanation of this patient’s symptoms?

- A. Acute ischemia to the left temporal lobe
- B. Acute infectious process
- C. Acute ischemia to the brain stem
- D. Central nervous system neoplasia
- E. Acute lacunar infarct of the basal ganglia
Key Concept/Objective: To understand the pathogenesis of acute expressive aphasia and that expressive aphasia in the absence of other neurologic findings may be mistaken for confusion

- Family described an acute confusional episode
- The major findings are probable atrial fibrillation and expressive aphasia in the absence of other neurologic symptoms

- Thromboembolism to the speech area of the left temporal lobe, caused by her atrial fibrillation

(Answer:A—Acute ischemia to the left temporal lobe)
Stroke

- Sudden loss of brain function resulting from an interference with the blood supply to the brain

- 795,000 / yr in USA, every 40 seconds, cost of 69 bln $ direct/indirect
Blood Supply to the Brain

- **Anterior Circulation** - 80% of CBF
  - Carotid Arteries - MCA, ACA, lenticulostriates
  - frontoparietal, anterior temporal, optic N, BG

- **Posterior Circulation** - 20% of CBF
  - Vertebrobasilar - posterior cerebral and cerebellurs, penetrating arteries
  - Upper spinal cord, brainstem, occipital lobe, cerebellum, medial temporal lobes, thalamus

- Circle of Willis connects Anterior – Posterior circulation
Left (Dominant) Cerebral Hemisphere

- Aphasia
- L gaze preference
- R visual field defect
- R hemiparesis
- R hemisensory loss
Right (Nondominant) Cerebral Hemisphere

- Neglect (= L hemi-inattention)
- R gaze preference
- L visual field deficit
- L hemiparesis
- L hemisensory loss
Question #5
What brain injured structure can lead to a pure sensory syndrome after stroke?

1. Posterior internal capsule
2. Cerebellum
3. Thalamus
4. Caudate
Lacunar Syndromes

- **Pure Motor** - most common – 50-75%
  - (post limb internal capsule, pons)
- **Pure Sensory** – 6-7%
  - (thalamus)
- **Clumsy Hand / Dysarthria** – 2-16%
  - (pons or int capsule)
- **Ataxic hemiparesis** – 18%
  - (corona radiata or ant int capsule)
- **Multi-infarct Dementia**
  - Penetrating arteries, lenticulostriates
Cerebellum

• Truncal or gait ataxia
• Ipsilateral limb ataxia
• Patient appears “drunk” but has skew deviation of the eyes, nystagmus
• Dizziness, headache, N/V
• Large cerebellar strokes and cerebellar hemorrhages require Neurosurgical consultation for surgical decompression
Brainstem Stroke

- **The 5 Ds:**
  - Dizziness, Diplopia, Dysarthria, Dysphagia, Dysmetria

- **Hallmarks:** *Crossed findings*
  - Cranial nerve deficits - Ipsilateral
  - Motor / Sensory deficits - Contralateral

- **Clinical Findings:** Depends on the syndrome
  - Range: asymptomatic to comatose

- **Posterior circulation**
Posterior Circulation Strokes

• **Prodrome** very common
  – 60 % of patients with Basilar artery thrombosis
  – Stuttering or progressive onset of symptoms
  – 2 weeks prior to ED presentation

• **Prodromal Symptoms** (in order of frequency)
  Vertigo and Nausea (30%)
  Headache, Neckache (20%)
  Hemiparesis (10%)
  Dysarthria, Diplopia (10%)
  Hemianopsia (6%)
  Ferbert, Stroke 1990
Basilar artery occlusion

• Can produce a **locked-in** syndrome
  – (95% mortality)

• Awake, quadriplegia, bilateral facial and oropharyngeal palsy, **preserved vertical gaze**

• May present comatose if reticular activating system is involved
Treatment of Ischemic Stroke

- Supportive Care, monitoring
- Stroke Vital Signs:
  - Oxygenation, Circulation, Blood Pressure, Temperature, Glucose, Seizures
- ASA, Plavix – NPO until swallow screen
- **t-PA** thrombolysis for patients onset of symptoms to treatment $\leq$ 3 hrs or $\leq$ 4.5 hrs
- No role for Heparin, LMWH
Hypertension in Ischemic Stroke

• Loss of auto regulation
  – Ischemic area pressure dependant
  – Treat BP judiciously if at all
• Treatment Guidelines - not receiving rt-PA
  – AHA: MAP > 130 or Sys BP > 220
    • MAP= [(2x DBP)+SBP]/3
  – NSA: 220/115
  – With t-PA 185/110
• Drugs - (labetalol, nicardipine, enalapril, nitroprusside)
Question #6
Do not administer rt-PA to a stroke patient if the blood pressure remains above?

1. 200/100
2. 170/90
3. 220/110
4. 185/110
5. 180/90
Inclusion Criteria

• Acute Ischemic stroke
• ≤ 3 hours from symptom onset to treatment
• Age > 18
Absolute Exclusion Criteria

- Blood on CT scan
- Possible SAH
- Active internal bleeding
- Bleeding Diathesis
  - Plt < 100,000
  - Coumadin, INR ≥ 1.7
  - Heparin + ↑ PTT
- Major surgery last 14 days
- BP sys >185, diast >110 despite tx
- Stroke or major head trauma - 3 mos
- Hx of ICH
- Hx of AVM, Aneurysm
Relative Exclusion Criteria (Warnings)

- Rapidly improving sx
- Minor symptoms
- GI or GU bleed - 21 days
- Arterial puncture in noncompressible site in last 7 days
- Recent LP
- Glucose < 50 > 400
- Post MI 3 mos
- Seizure at onset
- Pregnant
Question #7

Is following patient a candidate for rt-PA?: A 79 year old woman with a history of diabetes and atrial fibrillation on Coumadin who presents 4 hours after stroke onset with weakness and aphasia and a stroke scale of 10. Her platelets are 105. INR 1.5. BP 160/85. She has no other 3 hour exclusion criteria.

1. Yes
2. No
Additional Exclusion Criteria
rt-PA 3 to 4.5 hours

• Patients older than 80
• Those taking oral anticoagulants even with an INR < 1.7
• NIH-SSS > 25
• History of previous stroke and diabetes
Transient Ischemic Attack (TIA)

- Reversible focal dysfunction, usually lasts minutes
- 75% of TIAs resolve within 1 hour
- Among TIA pts who go to ED:
  - Average of 5.3% have stroke within 2 days
- Stroke risk decreased with proper eval of TIA
A 22-year-old woman presents with seizure. She denies having any previous episodes. She does not remember the episode, which was witnessed by her mother. The mother relates that she noticed that the patient had a blank stare and then, after a few seconds, she started moving her hands repetitively, “like she was washing them.” During this episode, the patient did not respond to any commands; it lasted approximately 1 minute. After this episode, the patient remained confused for about 10 minutes. The patient’s physical examination is unremarkable.
On the basis of clinical presentation, how would you classify this patient’s seizure?

- A. Myoclonic seizure
- B. Absence seizure
- C. Complex partial seizure
- D. Tonic-clonic seizure
Key Concept/Objective: To understand the major classification of seizures

- Based on clinical presentation and EEG criteria

- Major categories:
  
  Partial
  Generalized
  Mixed
  Continuous

(Answer: C—Complex partial seizure)
Partial Seizures

- Simple or complex, depending on whether consciousness remains intact or is impaired

- Simple partial seizures can be motor, sensory, autonomic, or psychic

- Complex partial seizures usually begin with arrest of motion and a blank stare
  - Automatisms, oroalimentary behavior, or verbal utterances may occur
  - Most spells last only a few minutes
Generalized seizures

- Non-convulsive pattern of **simple absence (petit mal)** seizures
  - Brief, usually **10 seconds or less**
  - No aura or postictal effects

- **Myoclonus** consists of **brief jerks** or contractions of a specific muscle or group of muscles

- **Grand mal (tonic-clonic)** – convulsions, muscle rigidity

- **Atonic (drop attack)** involve a **sudden loss of postural tone**
  - Begin in childhood
Mixed seizures

- More than one seizure type in same patient

- Myoclonus that becomes tonic-clonic in setting of fever

- Simple partial arm motor seizures that sometimes becomes grand mal
Continuous - Status Epilepticus

• Definition: > 5 min, 2 or more w/o lucid interval between seizures
• Mortality > 30% when status last > 1 hour
• Treatment
  – ABCs
  – ? Glucose, Thiamine
  – Benzo (Ativan, Valium, Versed), phenytoin load
  – Phenobarbital, Keppra, etc. ………
  – EEG if intubated and paralyzed
    • Propofol, pentobarbital
Seizures

• **History:** from patient and witnesses
  – Aura, localized, generalized, LOC, B&B

• **Physical:**
  – Search for assoc. injuries, systemic illness as precipitant
    • Tongue biting
    • Posterior shoulder dislocations
  – Todd's paralysis (13% szs): post-ictal neuro deficits, resolve on own (mean 173 sec)( range 11 sec - 22 mins)

• **Secondary causes:**
  – ICH, trauma, infection, metabolic disturbances, liver/kidney function, toxins/drugs, BP, eclampsia, hypoxemia, structural lesions (old stroke, AVM, aneurysm, tumor)
Drug Induced Seizures

- **Cocaine**
  - Benzodiazepines

- **INH - refractory**
  - Inhibits pyridoxine kinase
  - Pyridoxine - 5gm IVP over 5-10 mins, may repeat
  - Benzos

- **Theophylline**
  - Benzos, Barbiturates, MDAC, WBI (SR pills), dialysis or charcoal hemoperfusion

- **Cyclic Antidepressants**
  - Seizures, coma, QRS >100 ms … R > 3mm aVR
  - Benzos, bicarb
Seizures

• Neurology Consult (ACEP) clinical policy
  – New onset seizures
  – Focal neuro exam
  – Persistent altered mental status (sub-clinical status)
  – Change in seizure pattern
  – Poorly controlled seizures
  – Pregnancy
Question #9
Assuming this is not a stroke and the patient presents early with moderate symptoms, how would acutely treat this New England Patriot’s season ticket holder and avid hunter?

1. An antiviral
2. An oral steroid
3. Oral steroid and antiviral
4. Oral steroids and antibiotic
5. An antibiotic
Bell’s Palsy

• Most common cause of acute facial paralysis
• Peripheral 7th N palsy, involves the forehead
  • Facial weakness, dysarthria, dry eye, unable to keep eye closed, drool, metallic taste
  • Ramsey-Hunt syndrome: HZV infection, TM with ulceration or vesicles
  • Lyme disease if B/L or at geographical risk

• Treatment:
  • Eye care to prevent corneal abrasion / desiccation
  • Scottish Bell’s Palsy Study, NEJM Oct. 2007
    – Prednisolone within 3 days
    – Acyclovir NOT helpful
  • Anti-virals still recommended in severe disease
  • New England – Lyme disease - Antibiotics
Question #10
Although 60% of cases do not have a known cause, the most common cause of Gullian-Barré syndrome is?

1. Campylobacter jejuni
2. Influenza
3. HIV
4. Mononucleosis
5. Vaccinations
6. EM Residency
Guillain-Barré

• Acute Demyelinating Polyneuropathy
  – Ascending weakness, LOSS of DTRs
  – Onset with pain (50%), mild sensory deficits
  – Develops over days-weeks, relative symmetry

• Causes:
  – Preceding infections, toxins, collagen vasc disease

• Diagnosis:
  – History, HCT, normal LP (increased protein)
  – Nerve Conduction Studies

• Treatment:
  – ABCs, Plasmaphoresis, IVIG
The End