ED Approach to Neurological Emergencies

Core Resident Lectures

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St. Michael's

Inspired Care. Inspiring Science.

Objectives

- Case-based presentations where I will be seeking YOUR input
- Cases are not meant to be diagnostic challenges
- Emphasis will be on the diagnosis and management of the specific conditions

Case 1

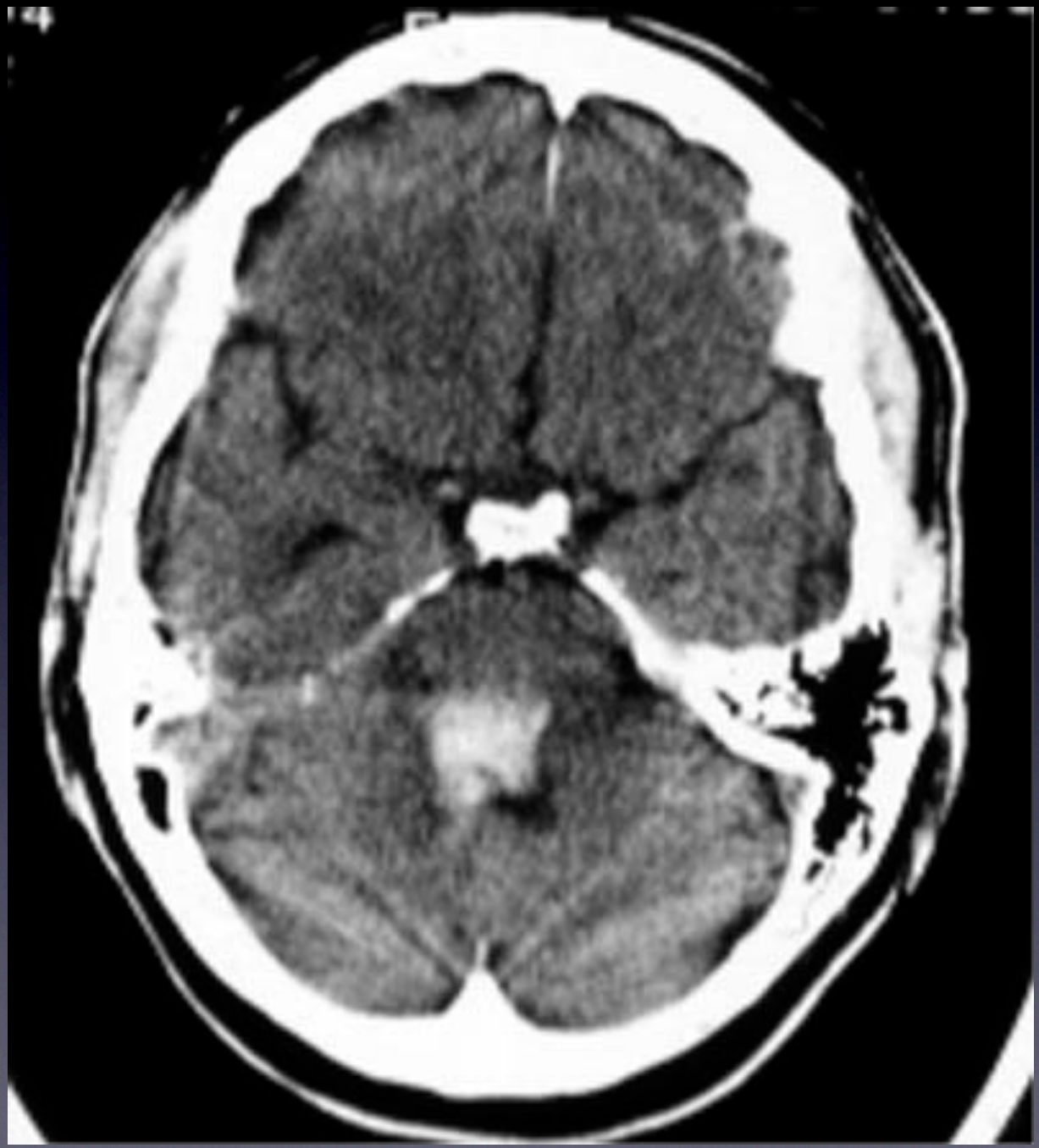
- 56 year old male presents with a decreased level of awareness
- VS: 150/90 - 80 - 18 - 37.2
- Well until today; neighbours found him unresponsive on his front porch
- How do you wish to proceed?

Case 1

- Examination reveals a GCS of 3, pinpoint pupils, no evidence of trauma
- Generally well kempt
- Skin examination normal with good perfusion
- How do you wish to proceed?

Case I

- Bedside glucose - 6.4
- naloxone 2mg given - no response
- serum electrolytes, CBC - normal
- toxicology - negative for EtOH, salicylate, acetaminophen
- How do you wish to proceed?



Coma/Altered LOA

- *consciousness = arousal (ARAS) and cognition (cerebral cortex)*
- metabolic/systemic derangements
- also structural lesions
- wide differential but....
- *consider etiology in context of history, collateral, demographics, and examination of patient*

Metabolic/Systemic Causes

- hypoxia
- hypoperfusion
- infection
- toxic drug effects
- electrolyte and glucose disturbances

Structural Causes

- Head trauma
  - sub/epidural hematomas, parenchymal/subarachnoid hemorrhage, concussion
- Stroke
  - thrombotic, embolic, hemorrhagic
- Tumor
  - mass effect itself or hemorrhage/edema
- Infection
  - meningitis, abscess/empyema, parasitic mass

The Context

- Elderly susceptible to infectious etiologies, medication changes, stroke, occult trauma (ie. chronic SDH)
- Young adults/adolescents - recreational drug use and trauma
- Young children - accidental toxic ingestions
- Infants - infection, trauma/abuse, metabolic

Physical Exam I

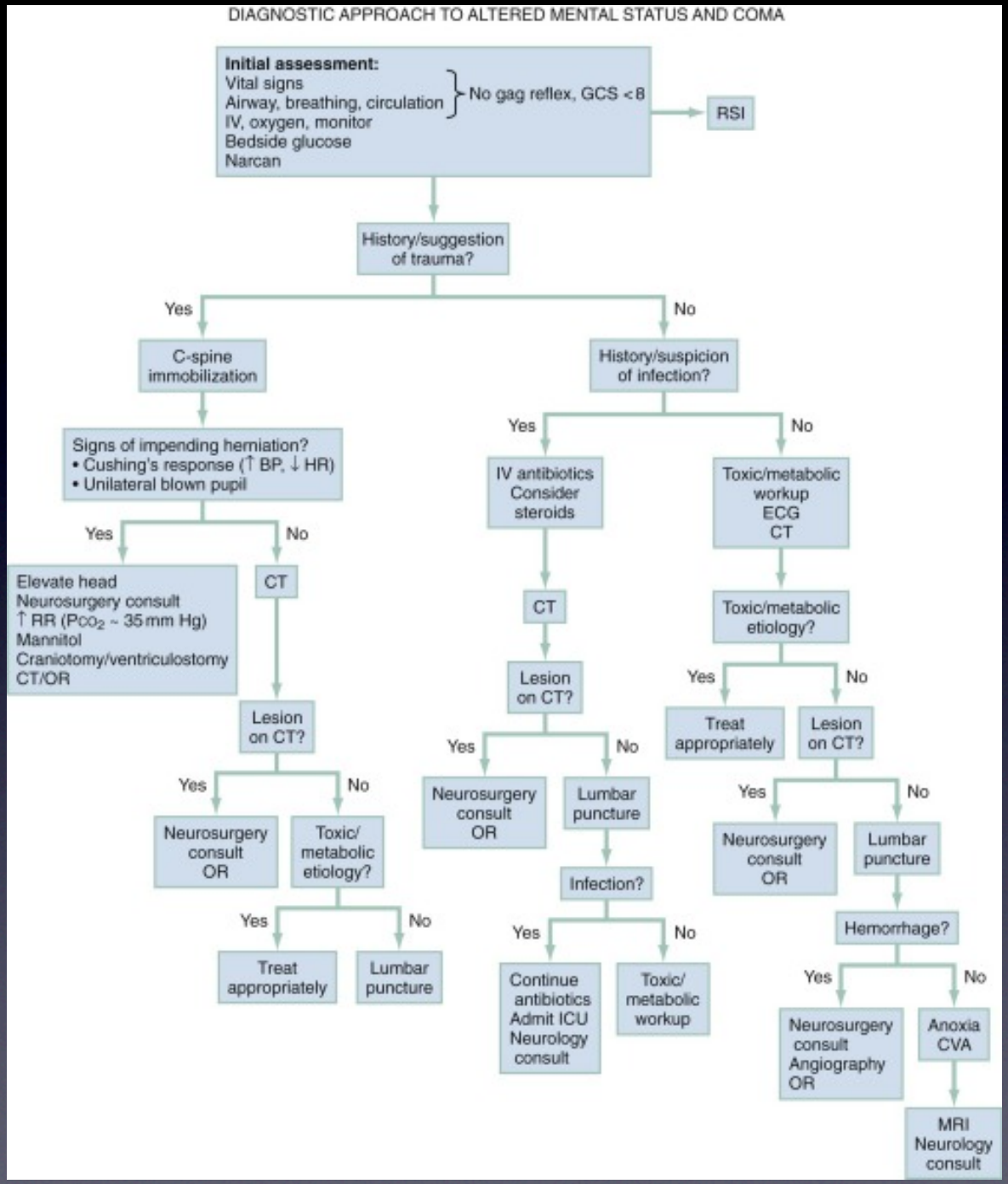
- ABC's first of course...
- level of consciousness, rapid/focused neuro
  - GCS, focal weakness
- vital signs
  - hypotension, temperature abnormalities, Cushing response, respiratory rate

Physical Exam II

- Head to toe exam
  - pupillary reflexes, head trauma, mucous membranes, neck (meningismus, immobilize in trauma)
  - gross blood, pus, retained FB on rectal/GU exam
  - cutaneous - ie. rash/perfusion/needle tracks

Clinical Evaluation

- evaluation/stabilization occur simultaneously with diagnosis



Adapted from Adams J: Emergency Medicine)

Empiric Management

- ABCs
- (and DEFG - “don’t ever forget glucose”)
- IV-oxygen-monitor
- consider “coma cocktail” (next slide)

Coma cocktail I

- Oxygen
- Naloxone
- D50W
- Thiamine

Case 2

- 62 year old male presents with right sided weakness and aphasia
- How do you wish to proceed?

Case 2

- History
  - previously healthy, no meds, co-workers stated this started 60 minutes ago
- Examination
  - 110-140/90-18-37.2
  - ABC-ok, right hemiparesis (arm>leg), aphasic

Case 2

- Investigations:
  - bedside glucose - 7.2
  - ECG - atrial fibrillation, rate of 110
  - Head CT - no hemorrhage, but hyperdense left MCA sign seen
  - CTA confirms diagnosis of left MCA thrombus and tPA is administered
- patient makes complete recovery

Definitions

- **Stroke**: Any vascular injury that reduces cerebral blood flow to a specific region of the brain
- **TIA**: brief episode of neurologic dysfunction caused by a focal disturbance of brain or retinal ischemia, with clinical symptoms typically lasting less than 1 hour, and without evidence of infarction

Stroke - Epidemiology

- Ischemic (80%)
  - large vessel & small vessel (33%)
  - cardioembolic (25%)
  - uncommon mechanisms (ie. vertebral/carotid dissections, hypercoagulability)
  - unknown etiology (>1/3)
- Hemorrhagic (10%)
- Subarachnoid Hemorrhage (10%)

Most Common Sites For Hypertensive Intracranial Hemorrhage

- Putamen (44%)
- Thalamus (13%)
- Cerebellum (9%)
- Pons (9%)
- Other cortical areas (25%)

Anatomy and Physiology

- anterior circulation (perfuses 80% of brain)
    - optic nerve, retina, fronto-parietal and anterior-temporal lobes, and more
- posterior circulation (20%)
    - supplies brainstem, cerebellum, thalamus, visual occipital cortex, and more

Stroke Location I

- ACA (anterior cerebral artery) stroke
  - primarily frontal lobe function
  - paralysis/hypesthesia of lower limb on contralateral side
  - lower limb weakness > upper limb
  - impaired judgement/insight

Stroke Location II

- MCA (middle cerebral artery) stroke
  - marked motor/sensory findings (upper limb > lower limb) on contralateral side
  - ipsilateral hemianopsia
  - agnosia common
  - aphasia common if dominant hemisphere affected

Aphasia

- clear articulation, but language used or understood poorly
- expressive, receptive, or both
- Wernicke's - receptive
- Broca's - expressive

Posterior Circulation Strokes

- ie. vertebrobasilar
- widest variety of symptoms, difficult to diagnose
- cranial nerves, cerebellum, neurosensory tract involvement
- vision and thought processing may be involved (occipital and parietal lobes)
- may have “crossed deficits” - bilateral findings

Stroke - Differential Dx

- epidural/subdural hematoma
- brain tumour/abscess
- air embolism
- metabolic (ie. hypo/hyperglycemia)
- Wernicke's encephalopathy
- migraine
- seizure --> Todd's paralysis
- others

ER Investigations I

- immediate: bedside glucose, *unenhanced* CT, ECG
  - CT usually negative for 6-12 hours, but *may* show:
    - hyperdense artery sign (acute thrombus in vessel), sulcal effacement, loss of gray-white interface, loss of insular ribon, mass effect, acute hypodensity

ER Investigations II

- CTA - useful to identify intravascular thrombosis, dissection, stenosis
- ? MRI - evolving use, more sensitive for posterior circulation stroke and diagnosing early ischemic stroke

Ancillary Investigations

- echocardiogram
- carotid duplex scan
- angiogram
- bloodwork

Code Stroke

- acute stroke <4 hours
- consideration for thrombolysis (tPA)
- organized team approach led by neurology
- history and immediate CT/CTA critical
- blood pressure must be < 185/110, and no other contraindications

Acute Ischemic Stroke - Other Management

- antihypertensives
  - for thrombolysis --> treat to pressure < 185/110
  - non-thromblysis --> treat if >220/120
    - aim for 10-20% reduction
  - agents: NTP, labetalol, nitroprusside
- heparin unproven, but sometimes used
- ? ASA

Intracranial Hemorrhage

- Reversal of any anticoagulation or platelet disorder
- BP control to < ~160/105

Disposition - Ischemic Stroke and TIA

- strong consideration for admission or urgent specialist follow-up
- antiplatelet therapy

Case 3

- 66 year old male
- Abrupt onset of severe occipital/neck pain while shaving
- + nausea, + photophobia
- Recent alcohol binge
- No LOC, no vomiting
- No change in vision/speech
- No focal weakness
- PMHx: none Meds: none

Case 3

- Examination:
- 36.3 - 170/100 - 92 - 16
- Alert and oriented to person, place, time
- No meningismus
- CN II-XII intact
- Sensory, motor, power normal

Case 3

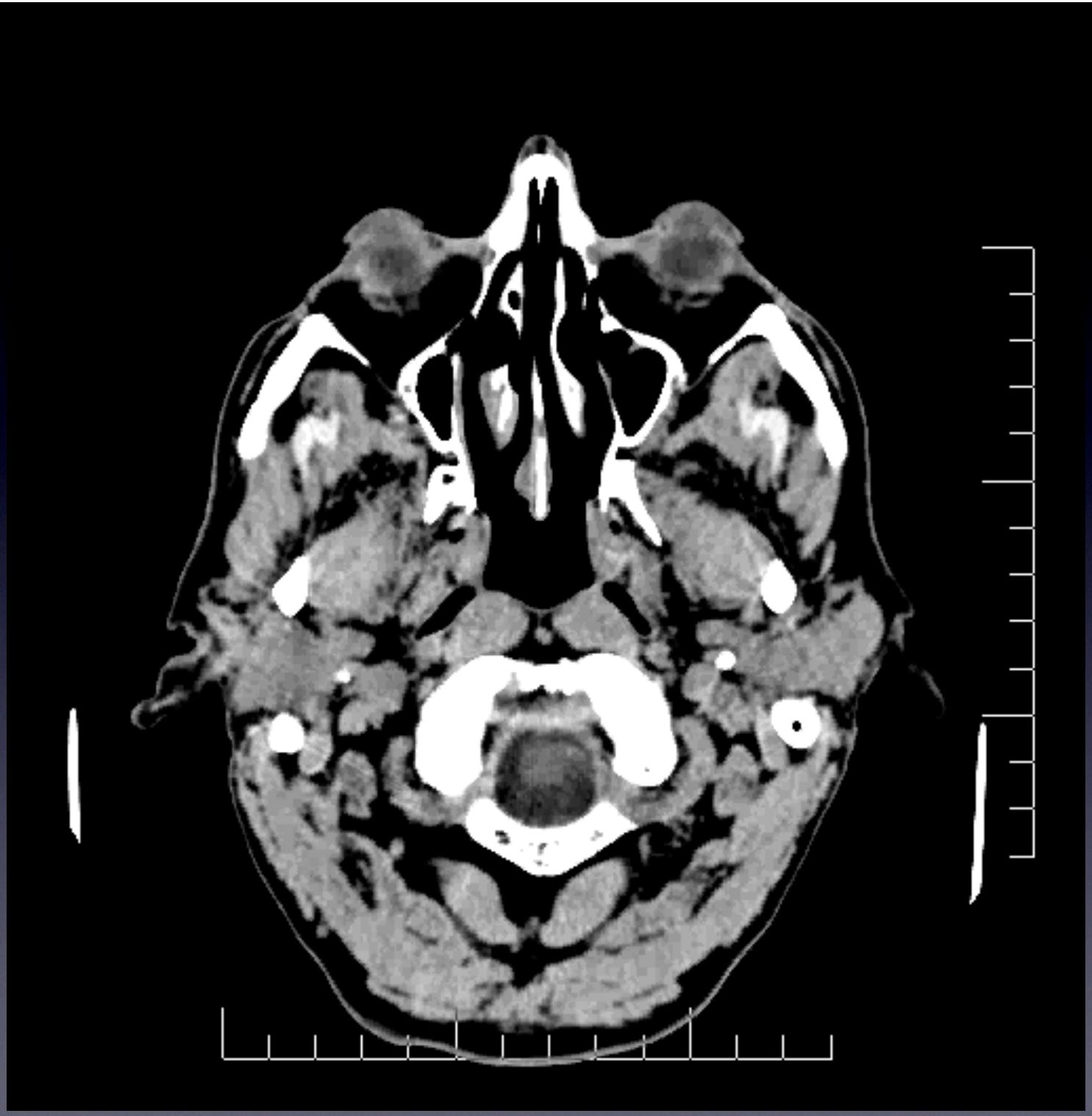
- Is there anything further on history or examination you would like?
- What is your differential diagnosis?

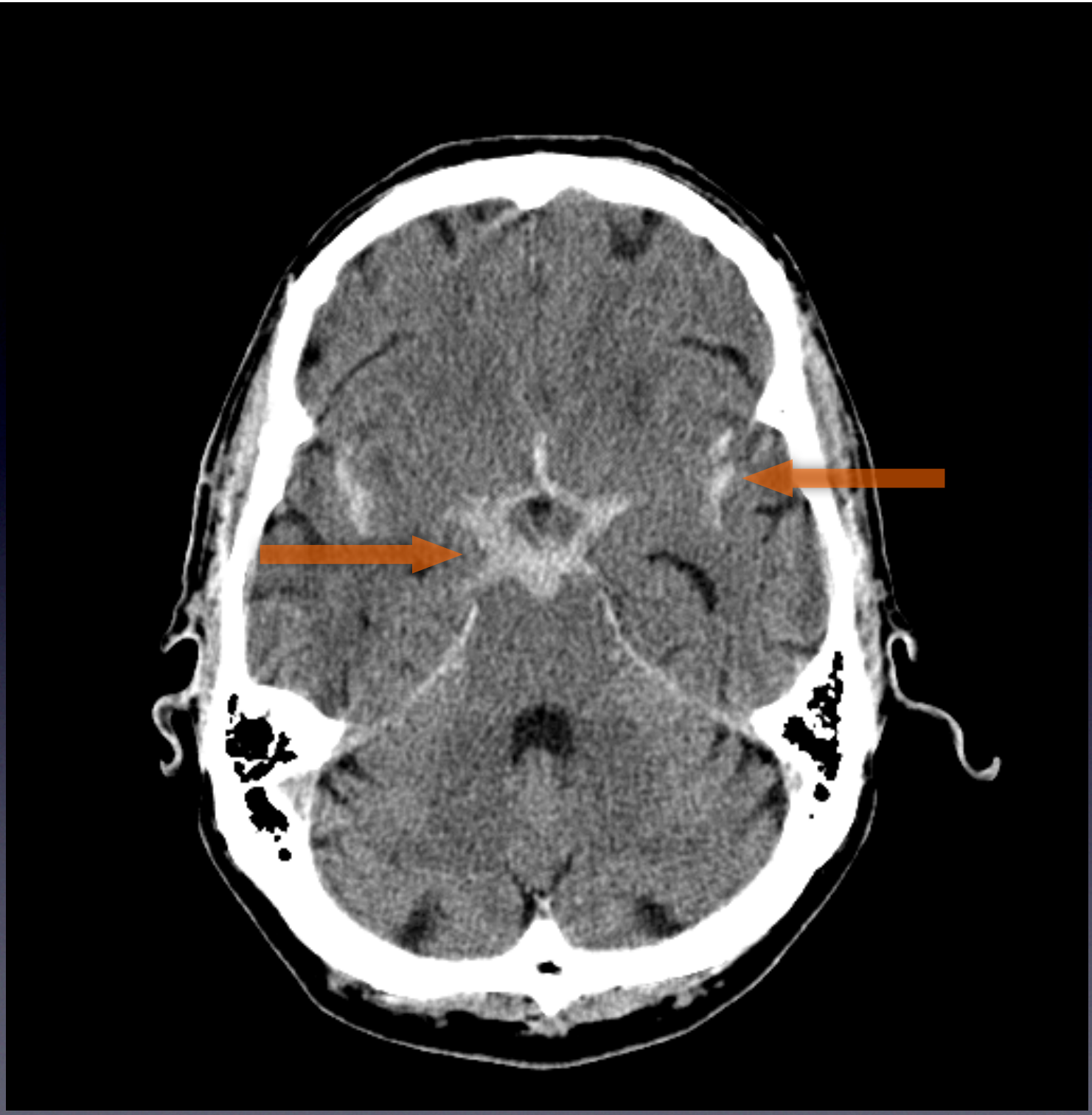
Case 3: Differential Diagnosis

- SAH
- Migraine headache
- Tension headache
- Meningitis
- Space occupying lesion
- Traumatic bleed
- Viral syndrome
- Carotid/vertebral dissection
- Alcohol withdrawal syndrome

Case 3

- What would you like to do?





Diagnosis

- Subarachnoid hemorrhage

Subarachnoid Hemorrhage (SAH)

- Most common cause is trauma
- Non-traumatic cases - 80% aneurismal
- Aneurismal SAH affects 6-10 out of 100,000 persons

SAH - The Basics

- Classic Story:
  - "Worst headache of my life"
  - Sudden onset
- Diagnosis (CT-LP):
  - Unenhanced cranial CT
  - Lumbar puncture if CT normal

SAH: The Three Clinical Presentations

- Decreased LOC or Headache/focal signs
  - CT-LP
- Classic presentation
  - CT-LP
- Atypical presentation
  - Diagnostic challenge
  - "Spectrum bias" exists
    - Patient looks well
    - SAH will be small volume with greater chance of negative head CT
    - MD's less likely to be concerned about SAH!!!

SAH: Four Features of the Headache

- Onset
  - Sudden or abrupt
- Severity
  - “worst” (10/10)
- Quality
  - “unique”
- Associated symptoms
  - Nausea +/- vomiting
  - Syncope
  - Seizure
  - diplopia

History: Pearls

- Quality of pain is *unique* for the patient
  - Very important principle
- Neck pain/symptoms may overshadow headache
- Headache descriptors may vary with caregivers (read EMS/RN notes!)
- Risk factors: HTN, alcohol use, smoking

SAH: Physical Examination

- Examination is often unremarkable
- Hypertension - important clue
- Nuchal rigidity - 70% of cases
- Cranial neuropathy, esp. CN III
  - Cranial nerve III palsy in 10-15% of SAH

SAH:
The Diagnosis

- High index of suspicion
- Step 1: Unenhanced cranial CT
- Step 2: Lumbar puncture if CT is negative, equivocal, or technically inadequate
- These 2 steps are the STANDARD OF CARE
- Don't talk yourself out of the LP

SAH: CT Interpretation

- CT - 90-98% sensitive for SAH
  - Sensitivity studies use neuroradiologists
  - Emergency CT's are usually read by ERP's
- Sensitivity decreases with time
  - Highest in 1st 12 hours
  - 85% by day 3
  - 50% at one week

SAH: LP Interpretation

- RBCs
  - Present in all SAH <12h
  - Presence decreases with time
- Xanthochromia
  - Yellowish discolouration of Hb breakdown
  - Presence increases with time
  - Visual vs spectrophotometry
- Opening pressure
  - Elevated in 2/3's of cases

Traumatic Tap vs SAH

- No exact definition of SAH or for TT
- Xanthochromia best distinguisher
- Interpretation of all data, especially with regards to time of HA onset
  - Xanthochromia
  - Opening pressure
  - RBC count (esp. tube 4 vs 1)

SAH: Pitfalls

- Not doing LP
- Not pursuing diagnosis after headache relieves with analgesics
- Not pursuing diagnosis because it isn't the classic story

Case 4

- 28 year old male
- Acute onset low back pain x 2 days, after lifting heavy boxes at work
- Pain/parasthesias down both legs
- No focal weakness
- Able to walk with assistance, but in severe pain

Case 4

- Any additional history?
  - Bowel or bladder symptoms
  - Saddle anesthesia
  - Erection difficulties
  - PMHx: back injuries, surgeries

Case 4: Examination

- VS: 37.2 - 130/80 - 74 - 18
- A+Ox3; no distress x/c with movement
- Full power in upper extremities
- Decreased power (4/5) in lower extremities (effort limited 2° to pain)
- Sensation decreased to LT in buttocks and lateral thighs

Case 4

- Anything else on exam?
  - Saddle anesthesia
    - Decreased perianal sensation
  - Rectal exam
    - Decreased tone
  - Straight leg raise
    - Positive bilaterally at approximately 40 deg.
  - Post-void residual
    - 900cc

Case 4

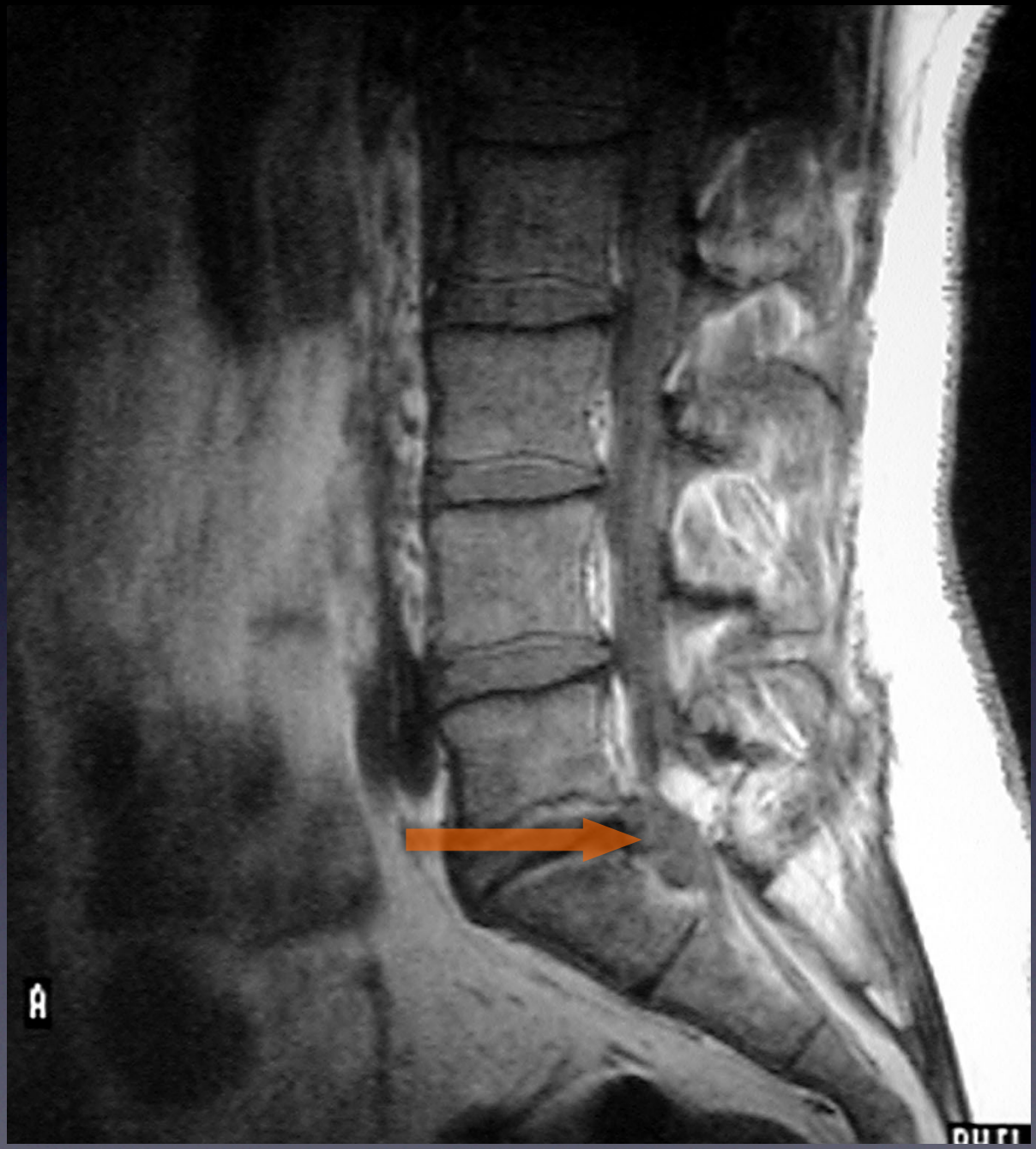
- What is your differential diagnosis?
- What would you like to do?

Differential Diagnosis

- Cauda Equina Syndrome
- Conus Medularis Syndrome
- Sciatica
- Guillain Barre
- Transverse Myelitis
- Epidural hematoma/abscess

Investigation Possibilities

- Plain radiography
- CT
- MRI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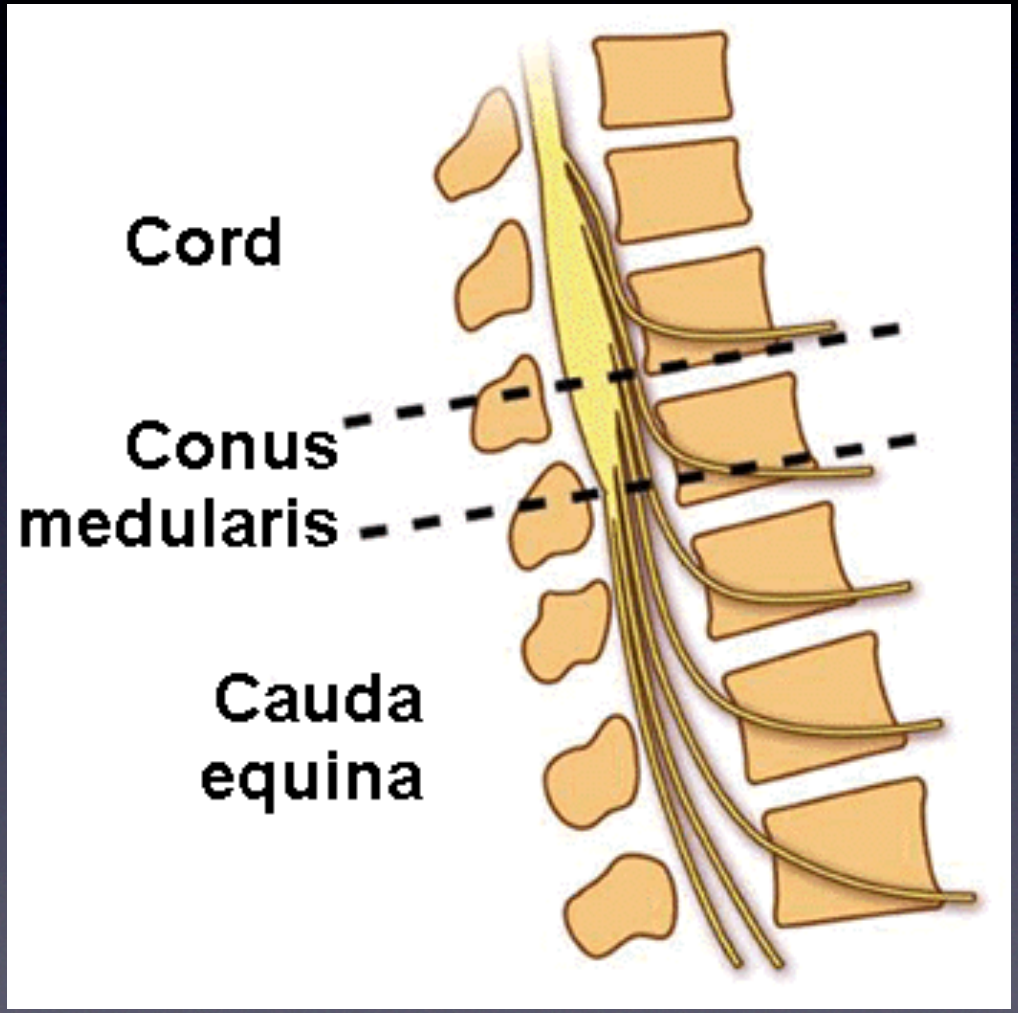
Diagnosis

- Cauda Equina Syndrome

Cauda Equina: Anatomy

- Spinal cord ends ~L1 (conus medularis)
- Cauda equina ("horse's tail")
  - The lumbar and sacral roots continuing from the spinal cord termination

Cauda Equina: Diagram



Cauda Equina Syndrome

- Neurological syndrome caused by compression of the nerve roots of the cauda equina
- Classic Triad:
  - Saddle anesthesia
  - Loss of bowel/bladder function
    - Overflow incontinence
  - Lower extremity weakness
    - Multiple spinal levels

CES: Pathophysiology

- Most common cause:
  - Massive midline disk herniation
- Most common site:
  - L4/5
  - Then L5/S1, L3/4

Quick Stats

- 90% of adults will get low back pain
- 90% spontaneous resolution within 4 weeks, often no specific cause
- ~4 in 10000 will have CES

Clinical Presentation: History

- Low back pain
  - Common (70%)
  - but 30% will have mild/resolved
- Radicular symptoms
- Lower extremity numbness/weakness
- Urinary/fecal retention/incontinence
- Gait disturbance
- Frequent falls

Clinical Presentation: Exam

- Lower extremity weakness and numbness
- Decreased DTR's
- Saddle anesthesia
- Decreased anal sphincter tone
  - Up to 80%
- Positive SLR
- Post void residual > 150cc
  - High sensitivity/specificity

CES: Bedside Diagnostics

- Digital rectal examination
  - Tests for rectal tone and saddle anesthesia
- Post-void residual
  - Patient voids completely
  - Foley catheter to measure residual volume
  - Tests for urinary retention
- Straight leg raise
  - Tests for radicular symptoms

CES: Pearls and Pitfalls

- Include CES in every back pain differential
- Proper history/exam
  - Attention to bowel/bladder function
- Diagnostic dilemna in:
  - the early compression
  - atypical symptoms
  - minimal/no back pain
- Attributing weakness to pain-limited effort
- Explicit discharge instructions critical

CES: Investigations

- Whole spine MRI
  - The "gold standard"
  - In parallel with surgical referral
- CT-myelography - invasive, less sensitive/specific
- Plain films - generally not helpful

CES: Treatment/Consultation

- Emergent surgical consultation
  - A time sensitive diagnosis
- Steroids
  - Ie. Dexamethasone
  - Discuss with your consultant
- Analgesia

Case 5

- 27 year old female presents with continual generalized seizures x 20 minutes
- Presents from home
- Well this morning
- PMHx: none Meds: none
- EMS Rx: diazepam 5mg iv with no response

Case 5

- 37.2 - 110/70 - 114 - 24
- Somnolent
- Moans to simple questions
- Periodic generalized tonic-clonic seizures, every few minutes

Case 5

- Are you happy with the ABC's?
- How would you like to treat the seizures?
- Any idea of the etiology?

Case 5

- Family arrives
- State that patient has recent history of depression
- Empty bottle of family member's white pills found, but left it at home
- Phone call made for drug identification
  - I - S - O - N - I - A - Z - I - D

Diagnosis

- Isoniazid toxicity with status epilepticus

Isoniazid toxicity

- Classic triad:
  - "refractory" seizures
  - Severe metabolic acidosis
  - Coma
- Antidote:
  - Pyridoxine (vitamin B6), 5g IV

Generalized Convulsive Status Epilepticus (GCSE)

- Definitions: Academic vs. operational
- Academic: seizures > 20 min
  - Cerebral injury may begin after this time
- Operational: seizure lasting > 5 min
  - seizure > 5 min rare

GCSE: Etiologies

- Acute vs Chronic
- Acute
  - Ie. Metabolic, infection, trauma, etc.
  - More difficult to control
- Chronic
  - Ie. Pre-existing epilepsy, remote tumor/CVA
  - Respond better to anticonvulsants

GCSE: General Approach

- ABC's
- Stop seizure
- Determine etiology
- Consultation +/- EEG

GCSE: Initial Management

- ABC's, monitor, supportive care
  - Ie. Supplemental oxygen, airway
- Bedside glucose or thiamine/D50W
- Anticonvulsant therapy
- History and Exam
- Investigations
  - Consider b/w, toxicology, EEG, CT/LP

First Line Anticonvulsant?

- Lorazepam vs Diazepam
- Essentially identical onset of action
  - Within 2-3 minutes
- Lorazepam has significantly longer duration of activity (12-24h) vs diazepam (15-30min)
- Lorazepam is the benzodiazepine of choice for seizures

Anticonvulsant Sequence

- Lorazepam
  - 0.1mg/kg IV at 2mg/min
- Phenytoin
  - 20 mg/kg loading dose
- Phenytoin
  - additional 5-10mg/kg
- Phenobarbital
  - 20 mg/kg loading dose
- Phenobarbital
  - additional 5-10 mg/kg

Anticonvulsant Sequence

- If patient still seizing --> now considered refractory
- Actions:
  - Intubate
  - General anesthesia
    - Midazolam or propofol
  - Consultation +/- EEG

No Intravenous Access?

- Midazolam 0.15 - 0.3 mg/kg IM

New-Onset Seizure - What To Do?

- Otherwise healthy adult, return to normal baseline
- Glucose and sodium
  - No evidence for routine Ca, Mg, Phos
- Head CT
  - ER or outpatient
- No antiepileptic medication

ACEP Clinical Policy 2004

GCSE: Pitfalls

- Failure to diagnose underlying etiology
- Failure to identify *ongoing* GCSE in comatose patient with *no* convulsive activity
- Failure to consider pregnancy or post-partum seizures
- Failure to consider toxins

Case 6

- 21 year old male university student
- Presents with fever, headache, vomiting, photophobia x 6 hours
- URTI x 3 days
- PMHx: none Meds: none

Case 6: Exam

- 38.8 - 114 - 110/70 - 20
- Somnolent, but orients x 3
- Brudzinski sign pos. plus neck stiffness
- Kernig sign - negative
- No rash
- CN II-XII intact, normal power/sensory

Case 6

- Do you require any additional history or examination?
- What is your working diagnosis?
- How do you wish to proceed?

Lumbar Puncture

- Fluid appears cloudy
- 12000 WBCs/mm^3
- No RBCs
- Glucose 1.7 mmol/L
- Gram stain: WBC's and gram + cocci

Diagnosis

- Bacterial meningitis

Epidemiology and Etiology

- Community-acquired adult meningitis
  - Most commonly - S. pneumoniae
    - Hib vaccination affect
    - Most common used to be H. influenzae
  - 2nd most common - N. meningitidis
  - Other agents:
    - L. monocytogenes
    - H. influenzae

Clinical Features

- Classic triad:
  - Fever, neck stiffness, altered mental status
  - Sensitivity only 44%
- 95% of cases - 2 out of these 4
  - Headache
  - Fever
  - Neck stiffness
  - Altered mental status
- Large variability in presentation
  - LP is best diagnostic tool

Head CT Prior to LP

- To predict and prevent brain herniation during lumbar puncture
- Indications:
  - —Altered mental status
  - —Focal neurologic signs
  - —Head trauma

Antiobiotic Therapy

- Empiric
  - aimed at most common agents
- Rapid
  - Don't delay for CT or LP
- Ceftriaxone and vancomycin
  - Assumes a DRSP rate of >4%
  - Add ampicillin if L. monocytogenes suspect

Steroid Therapy in Meningitis

- Improved outcomes, especially if pneumococcal meningitis is suspected
- Dexamethasone 10mg IV q6h x 4 days
  - Given before or at same time as ABX
- Some controversy

Meningitis: Post Exposure Prophylaxis

- Close contacts
  - Household
  - Airway operators
- Meningococcal meningitis
  - Rifampin
  - Alternatives: Cipro or ceftriaxone
- Pneumococcal meningitis - none required
- H. influenze meningitis - prophylaxis controversial