ED Approach to Neurological Emergencies

Core Resident Lectures
St. Michael’s Hospital ED
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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 I

- 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
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 ribbon, 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+O x3; 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
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 dilemma 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
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
  - 0.1 mg/kg IV at 2 mg/min
- Phenytoin
  - 20 mg/kg loading dose
- Phenytoin
  - additional 5-10 mg/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. influenzae meningitis - prophylaxis controversial