Stroke, Critical Care, & Infections

An Introduction

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The Intracranial Vessels
Stroke

• Ischemic
  – Thromboembolic sources:
    • Heart
    • Carotid artery
  – Lacunar
  – For academic purposes, distinguish between TIA vs. RIND vs. Stroke

• Hemorrhagic
  – Hypertensive
  – Aneurysm
  – Arteriovenous Malformation
Stroke

- Ischemic Stroke
  - Thromboembolic sources:
    - Heart (70% of cases)
    - Carotid artery (30% of cases)

CT Head showing stroke as a hypodensity
• Treatment Options for ischemic stroke
  - TPA (if w/i 3 hrs) – injected intra-arterially, done with angiogram
  - Heparin short-term, then long-term anticoagulation (e.g. ASA, ticlopidine (Ticlid), clopidogrel (Plavix), and/or warfarin (Coumadin))
  - If carotid disease:
    • Internal carotid artery with >=70% stenosis in symptomatic patients → patient would benefit from carotid endarterectomy
    • Internal carotid artery with >=60% stenosis in ASYMPTOMATIC patients → patient would benefit from carotid endarterectomy
    • Thus, although the study has not been done, >=60% for any patient may be inferred to benefit from CAROTID ENDARTERECTOMY.

• Prognosis is generally better for patients who are surgically treated with >=60% stenosis, according to NASCET & ACAS trials, in comparison to medical anticoagulation therapy
Diagnostic Studies

- Diagnostic studies
  - CT Head - r/o bleed
    - Usually “normal” for 12-24 hrs following ischemic stroke
  - Angiogram - find occlusion, +/- TPA if within 3 hours
  - ECHOcardiogram - To find thrombus
  - MRI/MRA to better define stroke early, with major vessel occlusion
  - Blood panel for more common disorders, e.g. cholesterol/LDL/HDL/Triglycerides
  - Blood panel - for rare disorders
    - Antiphospholipid antibodies (APL), Anticardiolipin antibodies (ACL), Lupus anticoagulant (LA); Blood culture; Cardiac enzymes: Troponin, Creatine kinase (CPK, CK), LDH isoenzymes; Coagulation factors: Antithrombin III, Protein C, Protein S; Factor VIII; activated Protein C resistance (Factor V Leiden); Erythrocyte sedimentation rate (ESR); Hemoglobin electrophoresis; Homocysteine; Syphilis serology (VDRL, FTA, others); Toxicology screen (serum or urine)
MRI shows left parietal infarct

Angiogram shows absence of Internal carotid artery (arrow)

ECHO w/thrombus

Carotid Ultrasound
Carotid Endarterectomy

- A candidate is a patient with the following findings
  - Case study 1: this patient had findings of a right middle cerebral artery stroke, with carotid artery stenosis.

[Image of brain scan and vascular system]
Carotid Endarterectomy

- Case study 2: Intraoperative plaque and the left carotid bifurcation anatomy are pictured.
Stroke

- **Lacunar Stroke**
  - A stroke resulting from small vessel occlusion ("lake") – shown as a small hypodensity on CT scan (arrows)
Hemorrhagic Stroke

- Hypertensive hemorrhage:
  - Typically results in bleed into:
    - Basal ganglia
    - Thalamus
    - Pons
    - Cerebellum

- Treatment includes blood pressure control (but not too low), and if superficial can remove blood clot

- Surgical procedures performed in only 10% of cases.

Stroke - Hemorrhagic

- Aneurysmal Subarachnoid Hemorrhage
  - May cause symptoms by mass effect compression, e.g. III n. palsy in posterior communicating aneurysm, or by bleed (typically subarachnoid hemorrhage)
  - 8% of us have an aneurysm; thankfully, most never rupture
  - After an aneurysm ruptures, 1/3 people Die, 1/3 are significantly disabled, and 1/3 recover reasonably
  - Treatment includes coiling or clipping
  - A delayed risk (occurring from 3-21 days, but most commonly 7-10 days) after bleed is Vasospasm – due to unknown substances from blood, a vessel spasms and can cause serious stroke-like symptoms
  - Vasospasm is treated by hypervolemia, hypertension, and hemodilution (Triple-H), +/- angioplasty
Stroke – Hemorrhagic

- **Arteriovenous Malformation**
  - Abnormal tangle of blood vessels which shunts blood from arteries to veins without capillaries
  - 4%/year risk of bleed, usually in lobar locations
  - Treatment is surgical excision or Gamma Knife

http://www.vh.org/adult/provider/radiology/RCW/1024962/T2MRI.html
Critical Care

• The Intensive Care Unit (ICU) patient is best approached by SYSTEMS:

  - Neurological:
    • Initial assessment of the trauma patient – ABC’s
    • Definition of coma (GCS)
    • External signs of trauma
    • Brain, Spine, Peripheral Nerve Trauma Classification & Protocols
    • Intracranial pressure management (& determining cerebral perfusion pressure)

  - Types of Shock:
    • Hypovolemic vs. Cardiogenic vs. Spinal (neurogenic) vs. Anaphylactic: KNOW & recognize each by their differences.
• Initial assessment of the trauma patient – ABC’s
  – Airway/Breathing (evaluate need for intubation), Circulation (evaluate blood pressure & pulse)

• Definition of coma (GCS)
  – GCS of 15 is normal (highest score) – range from 3 (worst) to 15 (best)

<table>
<thead>
<tr>
<th>Eyes</th>
<th>Verbal</th>
<th>Motor</th>
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<tbody>
<tr>
<td>Not open (1)</td>
<td>No sounds (1)</td>
<td>No movement (1)</td>
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<tr>
<td>Open to pain (2)</td>
<td>Grunts (2)</td>
<td>Decerebrate (2)</td>
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<tr>
<td>Opens to voice (3)</td>
<td>Words, confused (3)</td>
<td>Decorticate (3)</td>
</tr>
<tr>
<td>Open spontaneously (4)</td>
<td>Sentences, confused (4)</td>
<td>Withdraws to pain (4)</td>
</tr>
<tr>
<td>Normal speech (5)</td>
<td>Localizes to pain (5)</td>
<td>Follows commands (6)</td>
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Critical Care

- **External signs of trauma**
  - **Racoon eyes**
    - Usually delayed (6-12 hours), which helps distinguish this from direct trauma (i.e. “black eye”)
    - Sign of anterior skull base fracture
  
  - **Battle sign**
    - Usually delayed
    - Sign of skull base (temporal) fracture

- **Check for CSF rhinorrhea or otorrhea**
  - Can distinguish from phlegm or tears by testing for glucose (2/3 of serum), chloride, or beta-transferrin (most specific)

CT Head shows temporal skull fracture

http://www.emedicine.com/med/topic2894.htm
Critical Care

Traumatic Brain Injury

- Classification
  Severe – GCS 3-8    Moderate – GCS 9-11
  Mild – GCS 12-15

- Protocols for treatment
  Establish ABC’s
  Need for CT scan if h/o loss of consciousness, external signs of trauma, or neurological deficit
  GCS 8 or below (severe brain injury) may need intracranial pressure (ICP) monitor
  DO NOT use D5-containing fluids – Glucose is TOXIC to brain
  Ensure pCO2 is NOT HIGH, as this can increase ICP
  If CT scan shows bleed or comatose patient not improved, repeat CT Head to f/u
Spine Trauma

- Classification
  - Determine if a fracture is “stable or unstable”
    - Any subluxation is considered unstable, and immobilization is key
    - The spine can be developed into 3 columns – if the injury affects 2 of 3 columns the fracture may be unstable and require surgical fixation
    - Flexion-Extension films, in a mild fracture, when okayed by Neurosurgery team, can help detect instability
    - A fracture of the lateral mass of C2-C6 should promote worry about vertebral artery injury, and an MRA should be done at minimum

- Protocols for Treatment
Spine Trauma

- Protocols for Treatment
  - Clearing a Cervical Collar after trauma
    - The C-collar can be removed WITHOUT FILMS, INDEPENDENT OF MECHANISM, if the patient is
      » Awake, alert, and has had no medications to influence sensorium
      » Has no distracting injury (e.g. femur fracture)
      » Is completely non-tender and has full range of motion of the cervical spine
    - If all of these are true but the patient is tender, s/he should undergo AP/Lateral/Odontoid films, plus a CT scan with reconstruction to r/o fracture → if no fracture, then flexion/extension C-spine films → if all films normal, can remove C-collar
  - Any patient with spinal cord injury within ** 8 hours ** must receive a steroid protocol of methylprednisolone for 24 hours
Peripheral Nerve Trauma

• Peripheral Nerve Trauma

  - Classification
    • Neuropraxia: crush injury of nerve, usually recovers without intervention
    • Axontemesis: crush injury of axon, with Wallerian degeneration – axon must grow back @ 1mm/day
    • Neurontmesis: Nerve functionally severed – if no return of function in 6 months, may need surgical apposition

  - EMG/NCV studies indicate nerve continuity/function – can only get these after 2 weeks post-injury (due to artifact)
Peripheral Nerve Trauma

- Protocols for treatment
  - If any function present, do not intervene surgically
  - If no function present, do not intervene surgically, but wait for 6 months → get EMG/NCV studies then, and if STILL no function (conduction) then can intervene to surgically splice nerve
  - Exception to this is stab wound (as discussed)

- Case Study: Stab vs. Gunshot wound resulting in peripheral nerve injury
  - Following a stab wound, nerve re-approximation can be IMMEDIATELY performed
  - Following a gunshot wound, however, nerve reapproximation will not help as a greater portion of the nerve than immediately known may die; also, sometimes the injury can be due to concussive effect of the bullet, so waiting is best
Critical Care

- What is the normal intracranial pressure (ICP)?
  - Normal ICP is 5-20 cm H20, or 5-15 mm Hg

- Intracranial pressure management (& determining cerebral perfusion pressure)
  - Definition of CPP
    - CPP = MAP minus ICP
  - How do you figure out MAP?
    - MAP = 1/3 (systolic BP) plus 2/3 (diastolic BP) → for a “normal blood pressure” of 120/80, the MAP is 93
    - So, for a normal person, normal CPP is 93 minus 10, or 83
• What causes ICP to increase?
  • Anxiety/Stress, elevated pCO2, brain edema, bleed/other mass lesions

• What interventions can be used to decrease ICP for each of these reasons?
  – Mannitol, an osmotic diuretic, can help acutely
  – The Monro-Kelly Doctrine states that there are 3 things in the head – BRAIN, CEREBROSPINAL FLUID, & BLOOD
  – Of these, we can reduce the size of one or more components to decrease intracranial pressure
    • First choice – cerebrospinal fluid → via ventriculostomy
    • Second choice – reduce the amount of blood slightly (but not enough to cause a stroke) – since pCO2 controls cerebral blood flow, a pCO2 of 30-35 is maintained (normal is 40; less than 30 can cause stroke)
    • Final choice – which is controversial, but performed in the posterior fossa occasionally, is removal of damaged brain to reduce ICP (last resort)
Critical Care

• Shock
  - Definition: Critical lack of perfusion to tissues
  - Types of Shock:
    - Hypovolemic vs. Cardiogenic vs. Spinal (neurogenic) vs. Anaphylactic: Recognize features and treatment for each.
      - HYPOVOLEMIC: HR high, BP low, CVP low, Hemoglobin low
      - CARDIOGENIC: HR high, BP low, CVP high
      - SPINAL (NEUROGENIC): HR low, BP low
      - ANAPHYLACTIC: BP low, HR high, Hemoglobin not low (usually + h/o exposure)

  - Treatment for hypovolemic shock and spinal shock is volume first → HEART RATE indicates which type is occurring, and if heart rate still low, needs PRESSORS to correct vascular tone
Infections

- Outline
  - Meningitis
  - Brain abscess
  - Subdural empyema
  - Epidural abscess
  - Osteomyelitis
  - Soft tissue infection overlying central nervous system structure
  - Special consideration: The immunocompromised patient (e.g. HIV+ or s/p transplant)
CNS Infections

- Present in a variety of ways
- May result in death or severe morbidity if not treated promptly
- Usually occur from hematogenous spread
- Can also occur from direct extension from adjacent bone, soft tissue or sinuses
- Pathogens include bacteria, viruses, and fungi
- Most common: Acute bacterial meningitis and cerebral abscess
Infections

- **Meningitis**
  - Serious life threatening infection of meninges
  - Viral meningitis is more common, but is usually self-limiting
  - Common infective organisms are related to the patient’s age and any underlying disease
  - A few organism account for most of the cases but a wide variety may be responsible.
    - Neonate (0-4 weeks) - Group B streptococcus, E. Coli
    - 4-12 weeks - Group B streptococcus, Streptococcus pneumonia, Salmonella, H. Influenza, Listeria Monocytogenes
    - 3 months-5 years - H. Influenza, Streptococcus pneumonia, Neisseria meningitidis
    - Over 5 years and adults - Streptococcus pneumonia, Neisseria Meningitidis
Meningitis

• Major presenting symptoms include:
  - High fever
  - Meningismus including headache, neck stiffness, photophobia and vomiting
  - As illness progresses, mental status changes can ensue
  - Results from septic effect on brain, septic thrombosis and possible infarction

• Infants and neonates: Listlessness and irritability; fever and neck stiffness may be absent; Meningococcal infections frequently have a coexisting petechial rash.

• Complications of bacterial meningitis: Cerebral edema, Seizures, Hydrocephalus, Subdural empyema, Brain abscess

• Original source of infection may be evident, e.g. sinusitis, bacterial endocarditis, otitis media, mastoiditis
Meningitis

- Diagnosis is made by examination of the CSF by lumbar puncture.

- If there are signs of increased intracranial pressure or focal neurological signs, a preceding CT scan of the head is needed to exclude a mass lesion.

- CSF features of meningitis:
  - Raised cell count (5 - 10,000)
  - Elevated protein (>45 mg/dl)
  - Decreased glucose (<30 mg/dl)
  - Positive gram stain

- Treatment:
  - High dose antibiotics should be commenced immediately and then tailored based on culture results.
  - Duration of antibiotics 5-14 days, or longer for some cases.
Brain Abscess

- **Brain abscess**
  - Can occur at any age, single or multiple, usually supratentorial
  - Can result from hematogenous or direct spread from paranasal sinuses – MAY OCCUR in ABSENCE of MENINGITIS
  - May present with fever, malaise, seizures, neurological deficit
  - Appear as ring-enhancing regions on MRI, usually at grey-white junction – starts as cerebritis then becomes encapsulated
  - RUPTURE INTO VENTRICLE results in >90% MORTALITY
Brain Abscess

• Diagnosis
  - Blood cultures usually negative, LP usually negative and risk, so BIOPSY & CX

• Offending Organism:
  - Preantibiotic era: Staph. Aureus and Streptococci
  - Today, Streptococci, specifically Strep. milleri, are isolated from 80% of brain abscesses. Major habitat of Strep. milleri is the alimentary tract, including the mouth and dental plaques
  - Otogenic abscesses usually yield a mixed flora, including bacteroides, various streptococci and enterobacter species
  - Staph aureus is often the pathogen in abscesses resulting from trauma

• Treatment:
  - If > 2 mm., may need to evacuate/aspirate – otherwise continue Abx (Vanco + chloramphenicol or cefotaxime and metronidazole); repeat imaging to eval. resolution
Subdural Empyema

- **Subdural empyema**
  - The true neurosurgical emergency – for surgical evacuation urgently, otherwise venous thrombosis can result from infection
  - Presentation: h/o fever, H/A, focal deficit, seizure; empyema usually from local extension vs. hematogenous
  - Recognize vs. subdural hematoma – ring-enhancing, in absence of history of trauma
  - Main organism: Streptococcus
  - Treatment is surgical evacuation ASAP, + Abx (as for abscess)
  - Prognosis: 10-20% MORTALITY

http://www.medschool.lsumc.edu/Nsurgery/subempy.html#anchor958872
Epidural Abscess

- **Epidural abscess**
  - Sx: Back pain, fever, spine tenderness
  - Risk Factors: Diabetes, IVDA
  - Presentation: dependent upon region → may result in neurological decline
  - Treatment: MUST take for surgical evacuation if patient has worsening neurological function; RARELY can observe small epidural abscess, if no neurological decline. Need Abx.
  - If associated with osteomyelitis → may need surgical stabilization procedure also
  - Usually due to hematogenous spread (S. aureus) → can biopsy (CT-guided) for ID

[http://www.aafp.org/afp/20020401/1341.html](http://www.aafp.org/afp/20020401/1341.html)
Immunosuppressed & Immunocompromised Patients

- In patients on immunosuppressant therapy, fungal infections of the brain can occur
- Differential diagnosis of the brain lesion in an HIV+ patient
  - Toxoplasmosis (or other infectious agent-caused abscess)
  - Lymphoma
  - PML (progressive multifocal leukoencephalopathy)
- BEFORE diagnosis certain, recommended guidelines are to treat for Toxo for 1-2 weeks
- AFTER treatment, reimage ➔ if smaller, then continue tx; otherwise may need biopsy
- If a lesion DOES NOT enhance with contrast, then it is likely PML (and does not require biopsy)
- Since the treatment of lymphoma is steroids, it is not wise to start steroids if an infectious process is ongoing ➔ so biopsy is critical after 2 weeks to rule out infection & confirm lymphoma
• http://www.strokecenter.org/pat/diagnosis/
The End