Neurological Emergencies REVIEW
Goal

• New information on Differential Dx of:

• Hemorrhagic vs. Ischemic Stroke
The areas of the Brains

• Frontal Lobe- behavior, personality, sinus
• Parietal- speech, pupils vision
• Occipital- vision, heart rate, balance
• Temporal- acoustics, facial
• Brainstem- Pupil size, BP, HR, Balance
Cerebral Circulation
Cincinnati stroke scale

- Facial droop: motor facial nerves
- Arm drift: motor cortex
- Speech: speech center

72% accurate to stroke if 1 or > criteria met
Neurological Emergencies

- Acute Ischemic Stroke
- Intracranial Hemorrhage/ Hemorrhagic Stroke
Acute Ischemic Stroke (AIS)

- AIS is caused by the sudden loss of blood circulation to an area of the brain resulting in ischemia and corresponding loss of neurological function.

- **Identical to Myocardial Ischemia**

- The goal of treatment for AIS is to prevent further damage hypoxic tissue.
Acute Ischemic Stroke (AIS) Risk Factors

- Older Age
- High BP
- TIA History
- Diabetes
- High Cholesterol
- Tobacco Use
- Atrial Fibrillation
MI and CVA Similarities
Thoughts????
What I do not have to definitively diagnose CVA

naughty
Cerebral Ventricles
Within seconds to minutes of loss of perfusion, an ischemic cascade is unleashed resulting in a central area of irreversible infarction (similar to a burn) surrounded by an area of potentially reversible ischemic tissue.
Acute Ischemic Stroke

Ischemic Cascade

- Loss of O₂ and Glucose delivery to the brain cell results in cellular depolarization

- The resulting Ca - the un-oxygenated effected brain tissue is in “seizure”, but the motor center is not yet impacted
Acute Ischemic Stroke

Ischemic Cascade

- Seizure or over stimulation of areas causing a brain/motor/sensory tetanic response, followed by flacid/non functioning motor responses.

- Edema ensues causing the release of H+, Lactic Acid and K...effecting non ischemic tissues and cells
Acute Ischemic Stroke

Clinical Presentation

• No 100% clinical feature reliably distinguishes AIS from hemorrhagic stroke

• Signs/Symptoms: headache, N/V, and altered mental status, time of day am:
  – Early daylight vs. mid day / evening

• PMH of HTN, change in GCS of 1 pt at one time make ischemic stroke more likely.
Acute Ischemic Stroke

Clinical Presentation

• Common symptoms of AIS include the abrupt onset of hemiparesis, ataxia, vertigo, aphasia, or change in GCS by one point at a time.

• Establishing the onset of symptoms is essential when considering possible, one sided visual loss thrombolytic therapy.
ACI Statistical
PEARLS

• AM early morning Stroke
• 90% of strokes are ischemic
• AV PU rule = 94% CORRECT
Acute Ischemic Stroke

TIA

Transient Ischemic Attack

- TIA’s are defined as a transient ischemic neurological deficit that resolves within 24 hours
- 80% resolve within 60 minutes
- TIA’s precede 30% of AIS
- Left untreated, $\frac{3}{10}$ TIA’s progress to stroke (20% within the first month and 50% within the first year)
Acute Ischemic Stroke

Physical Examination

- **Goal of PE** is to look for extra cranial causes of AIS and to distinguish AIS from **stroke mimics** ie other differential diagnoses (seizures, tumors, toxic-metabolic disturbances, positional vertigo, etc).
- **HEENT**: Look for trauma signs and nuchal rigidity, evaluate pulse strength.
- **C/V**: Signs of **CHF / LHF (why??)** secondary to: Atrial fibrillation and other arrhythmias.
### Presumptive Differential Diagnosis

<table>
<thead>
<tr>
<th>Adult</th>
<th>Hypoglycemia</th>
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<tbody>
<tr>
<td>Hypoxia</td>
<td>Trauma (head or spine)</td>
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<tr>
<td>Seizure</td>
<td>Shock</td>
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<tr>
<td>Infection (meningitis, encephalitis)</td>
<td>Anemia</td>
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<tr>
<td>Hypertension</td>
<td>Psychiatric</td>
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<tr>
<td>Drug or alcohol ingestion</td>
<td>Environmental (heat/cold exposure, CO poisoning)</td>
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<td>Electrolyte disturbance</td>
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Acute Ischemic Stroke

Neurological Exam

- Goal – establish baseline for monitoring response to therapy and to determine size and location of AIS, watch for changes.
Acute Ischemic Stroke

Neurological Exam

- MS, CN, Motor, Coordination, Sensory and Gait need to be covered, however speed is of the essence!
- **MCA**: Contralateral: Hemiparesis, Hemianopia (Vision change = opposite hemisphere, pupil = same hemisphere (motor)
  - Sensory loss
  - Ipsilateral (same side): Gaze preference/ looks at side of bleed
  - Aphasia (inability to speak)
  - Sensory deficits
Conventional Angiogram
Acute Ischemic Stroke
Further Differentials

Neurological Exam for **Cerebral Accident**

- **ACA (acute):** Disinhibition, primitive reflexes, contralateral hemiparesis (legs/arms), urinary incontinence.

- **PCA (partial):** Contralateral hemianopsia (hemy-an-op-see ya: hemi- half/ opsi- Optic view), cortical blindness, altered mental status, impaired memory.
Hemianopsia

In this example the occlusion would be on the left and would be partial cerebral accident (PCA)
“FAST” FINDINGS

- **F**ace/Feet- droop, sags, smiles
- **A**rms- motor unequal, sensory unequal
- **S**peech- altered, slurred, unrecognizable
- **T**ime- onset, GCS changes by one or multiple
Right vs. Left brain hemisphere

• The right half (hemisphere) of the brain controls the movement of the left side of the body.

• The right half of the brain controls judging distance, size, speed, and position. This may cause a person with a right brain stroke to misjudge distances leading to falls. They have issues with short-term memory.

• The person may not be able to control the hand to pick up an object. Survivors of right-brain strokes often have problems making good decisions and become impulsive.

• Persons with right brain stroke are often unaware of the changes that have happened to them. They believe they can do the same tasks as they did before the stroke.
Right vs. Left brain hemisphere

• **Left-Brain Stroke** effects the right side of the body

  • Trouble speaking or understanding words said or written Slow, careful movements.

  • Not able to see things on the right side of the body.

  • Facial weakness, unclear speech, or problems with swallowing
    – Cincinnati “smile” and “dog tricks”
VENTRICULAR INVOLVEMENT = motor and sensory
Acute Ischemic Stroke

Treatment

• ABCD’s
  – **Airway**: preparation for advanced airway management imminent for GCS <
  – **Breathing**: O₂ if hypoxic. Keep PCO₂ 32-36 mmHg
  – **Circulation**: Maintain adequate CPP (MAP-ICP). Treat HTN per protocols (with MC advise)
  – **D** = Dextrose/core temperature. Maintain normoglycemia (as hyperglycemia worsens neurological outcome)
CO2 and cerebral circulation

• **Increased CO2** form bradypnea or shallow hyperventilation -
  – CAUSES: Cerebral vasodilation

• **Decreased CO2** from over aggressive ventilation or hyperpnea:
  – CAUSES: Cerebral vasoconstriction
Acute Ischemic Stroke

Treatment

• Fever: Hyperthermia worsens ischemic injury

• Cerebral edema: Peaks 72-96 hours. Hyperventilation can decrease cerebral pressure by eliminating CO₂ and causing vasoconstriction.

• Seizure control: As needed, No prophylactic (vs. prophylaxis in the hemorrhagic stroke)
Intracranial Hemorrhage
(non-traumatic)

Differential Diagnosis and Pathophysiology
Intracranial Hemorrhage

Intraventricular Hemorrhage

- Accounts for 3% of all non-traumatic ICH
- Hypertension is the most common etiology
- Often results from an intrahemisphere hemorrhage that extends into the ventricular system - effecting BOTH sides
- S/S: Headache, N/V, Progressive deterioration of consciousness, raised ICP, Nuchal rigidity
Intrahemisphere Hemorrhage
Intracranial Hemorrhage

Intraparenchymal Hemorrhage

• Pontine Hemorrhage
  – Abrupt onset of coma, pinpoint pupils, autonomic instability, horizontal gaze paralysis, and quadriparesis
  – The myopic pupils and depressed LOC may mimic opiate overdose
Cerebellar Hemorrhage:

- Sudden onset of vertigo, severe N/V, and ataxia leading to altered mental status and coma over a few hours
- Edema contributes to brainstem herniation
- Urgent posterior fossa decompression is essential for survival
Hemorrhagic CVA

Causes

• Hypertension is the #1 cause in adults
• Anticoagulation and Anti-Platelet Meds
• Sympathomimetic Drugs
• Aneurysms
• Brain Tumors
  – Metastatic (renal cell CA, malignant melanoma,
Hemorrhagic CVA

Treatment

• ABCD’s
  – Intubation, ETCO₂ on or about 30-33
  – Treat Hypertension per protocol (with MC advise)

• Fluid and Electrolyte Management
  – Use Normal Saline

• Prevent Hyperthermia

• Seizure Prophylaxis +/-

• Hyperventilate only if brain stem herniation is present (seizures, unequal pupils, sudden drop in GCS by 4 or >
Hemorrhagic CVA
(non-traumatic)

• Aneurysmal rupture accounts for 80% of cases
• Risk Factors
  – Advancing age, Smoking, HTN, Cocaine use, Hypertension, Heavy Alcohol use, Connective Tissue Disorders, Sickle Cell Disease, First Degree Relatives with Aneurysms
• Fatality rate is 50% within 2 weeks
• 15% of patients will have > 1 aneurysm
• 30% of survivors require lifelong care
• Outcome largely dependent on clinical presentation and CT findings
• Prehospital GCS is Key to Neurologist response in ED
Hemorrhagic CVA

- Clinical presenting signs
  - Subarachnoid- Sudden-Onset
    “Thunderclap Headache”
  - “Worst Headache of my life” sudden onset
  - CN III palsy – pupil(s) non motor (aneurysm)
  - CN VI palsy- pons controlled, Facial Motor /Sensory (raised ICP)
  - Retinal Hemorrhages
  - Altered Mental Status
  - Nuchal Rigidity
Hemorrhagic CVA

Treatment

• Recognition
• Trending
• BP control as directed
• Airway management
• Seizure control...as this will eventually happen
• GCS “drops” accuracy
Clinical Findings

- Sign and symptoms: AV / PU, %’s
- Allergies:
- Meds: HTN, Cholesterol, A-fib
- PMH: TIA’s
- Last events, med changes
- Events: day vs night/ early am
Status Epilepticus

Definitions

- A single seizure 10 minutes or longer and/or
- back-to-back seizures without return of consciousness
Status Epilepticus

Epidemiology

• 10% of all individuals with epilepsy will have at least one episode of SE in their lifetime

• 10% of patients experiencing a first unprovoked seizure will present in SE
Status Epilepticus

Acute Symptomatic Etiologies

- **Vascular**
  - Stroke (Hemorrhagic > Ischemic)
  - Subarachnoid Hemorrhage
  - Hypoxic Ischemic Swelling Cascade

- **Toxic**
  - Cocaine and other sympathomimetics
  - Alcohol withdrawal
  - Various Medications (Isoniazid, TCA’s, various chemotherapy agents)
Status Epilepticus

**Acute Symptomatic Etiologies**

- **Metabolic**
  - Hyper or Hypo-Natremia
  - Hypoglycemia
  - Hypocalcemia
  - Liver or Renal failure
- **Infectious**
  - Meningoencephalitis
  - Brain Abscess
- **Trauma**
Status Epilepticus

Treatment

• ABCD’s
  – Airway: Risk of aspiration, suction to bedside
  – Breathing: Give supplemental O₂
  – C/V: Initial tachycardia giving way to hypotension (especially when Benzos or Barbiturates are given)
  – Dextrose: Symptomatic hypoglycemia is causing irreversible brain injury until corrected
Status Epilepticus

- **History**
  - Fever, pre-existing epilepsy, trauma, baseline AED’s and their dosing

- **Physical Exam**
  - Signs of trauma, nuchal rigidity, end organ injury
  - Subtle signs of seizures (tachycardia, pupil nystagmus, irregular respirations)
Now you are better ready to go out there and confidently handle patients presenting with these various Neurological Emergencies!
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