Anoxic Brain Injury

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RAD 4001 Diagnostic Radiology
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Clinical History

49yo male with history of quadriplegia, several c-spine injuries 2/2 MVC in 1/2020 (s/p fusion, laminectomy), neurogenic bladder (foley at baseline) admitted with chief complaint of “indwelling catheter is not having output since last night.”

• Found to have UTI, foley exchanged, treated with ceftriaxone

• Began AMS workup on day 5
  • Likely not metabolic, seizure
  • Infection w/u ongoing, including LP
  • Thought to be 2/2 to baclofen withdrawal
Clinical History

Day 9 – unresponsive after returning from LP

• Found to have PEA, code blue called
• ROSC after 7 minutes
Clinical History

- Vitals: hr 85, RR 20, SpO2 98 (intubated) Tmax 93.7 (Tmax 100.9), bp 87/59
- General: sedated, on hypothermia protocol
- Neck: decreased ROM
- Chest: intubated, chest tube in place
- CV: tachycardic, normotensive on pressors
- Abd: soft, nondistended
- Skin: no rashes

- Neuro
  - Mentation: sedated, not following commands
  - CN: 3mm PERRLA, dysconjugate gaze, not tracking, flattening of L nasolabial fold
  - Motor: thin bulk, spasticity throughout, no mvmt of U/Les
  - Sensation: grossly intact to light touch
  - Coordination: could not assess
  - Gait: deferred
Clinical History

- Day 9 CSF (before event, not sedated): Glc 50, Ptn 68, RBC 4, WBC 3, OP 31, negative GS
- EEG with slowing, no epileptiform discharges
- Day 5 CTH: no acute abnormality
- Day 9 MRI (before event): no acute abnormality; chronic R cerebellar hemorrhagic infarct
Clinical History

Transferred to CCU
Pressors as needed
CXR: R tension pneumothorax, chest tube placed
Empiric antibiotics for possible meningitis
Hypothermia protocol initiated
Stat CT brain without contrast
Warning: Not for diagnostic use
Summary of imaging findings

- Loss of gray/white matter interface in both cerebral hemispheres
- Symmetric hypodensity in basal ganglia and thalami bilaterally
- Cerebral edema, effacement of cisterns, sulci
Differential Diagnosis

- Anoxic Brain Injury
- Ischemic Stroke
- Traumatic Brain Injury
Discussion

• Pathophysiology of Anoxic Brain injury
  • Primary injury: neuronal death due to ischemia
  • Secondary injury: neuronal death due to imbalance of cerebral oxygen delivery and use – metabolically active tissue hit hardest (e.g. basal ganglia)

• Common sequela of cardiac arrest

• Management is supportive
Discussion

• Cerebral metabolism is reduced by 5-10% / 1°C decrease
• Hypothermia $\rightarrow$ decreased metabolic demand
  • $\rightarrow$ decreased CO2 production
  • $\rightarrow$ decreased O2 consumption
  • $\rightarrow$ decreased lactate production
  • $\rightarrow$ mitigates inflammation, apoptosis
• Goal temperature: 32-34°C, 24-48hrs
Outcome

• Patient’s neurologic function continued to deteriorate
• Nuclear cerebral perfusion scan performed

Warning: Not for diagnostic use

Cerebral blood flow image of a patient's brain, showing different views (ANT IMMED, LT LAT, RT LAT, 20 MIN ANT).

September 04, 2020

15.0 mCi NEUBRITE
Final Diagnosis

• Anoxic brain injury
• Brain Death
Imaging modalities and cost

- Modalities of choice: CT brain w/o contrast +/- MRI w/o contrast (head trauma from acute injury, with neurologic deterioration)
  - Brain death determination – clinical +/- ancillary studies (such as cerebral perfusion)

- Imaging for case: CT brain without contrast x2, Nuclear cerebral perfusion
- Estimated to be ~ $4200
  - Parameters: without insurance, in AZ
  - CT brain without contrast = $1213
  - Nuclear cerebral perfusion scan ~ 1800 (used heart SPECT scan as proxy)

- Source: https://costestimator.mayoclinic.org/find/medical-services-and-procedures
Take Home Points / Teaching points

• Anoxic brain injury is a common sequela of cardiac arrest
• Common brain CT findings include loss of gray-white matter interface and hypoattenuation of basal ganglia and thalami
• Therapeutic hypothermia can mitigate secondary injury
References


Questions?