Anoxic Brain Injury

Daniel Abramson, MS4

16 September 2020

RAD 4001 Diagnostic Radiology

Maria Patino, MD

UTHealth®
The University of Texas
Health Science Center at Houston
McGovern
Medical School

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

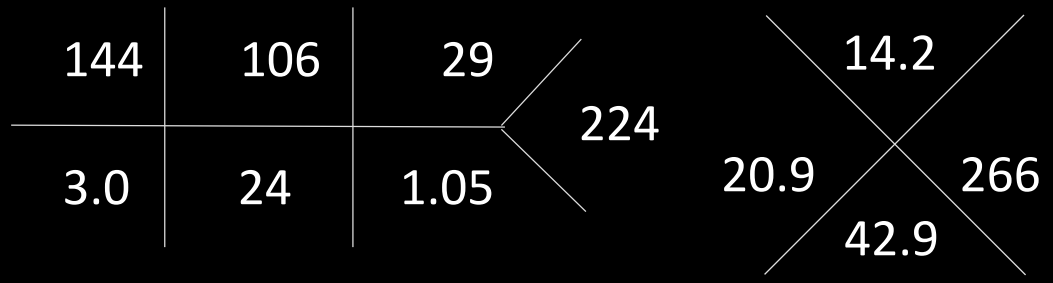
Day 9 – unresponsive after returning from LP

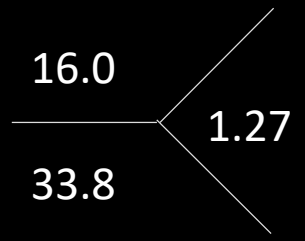
- Found to have PEA, code blue called
- ROSC after 7 minutes

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





Ph 8.2
Ca 10.5
Mg 2.0
NH3 30.0
Lactic Acid 9.8
TSH 4.27

- 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

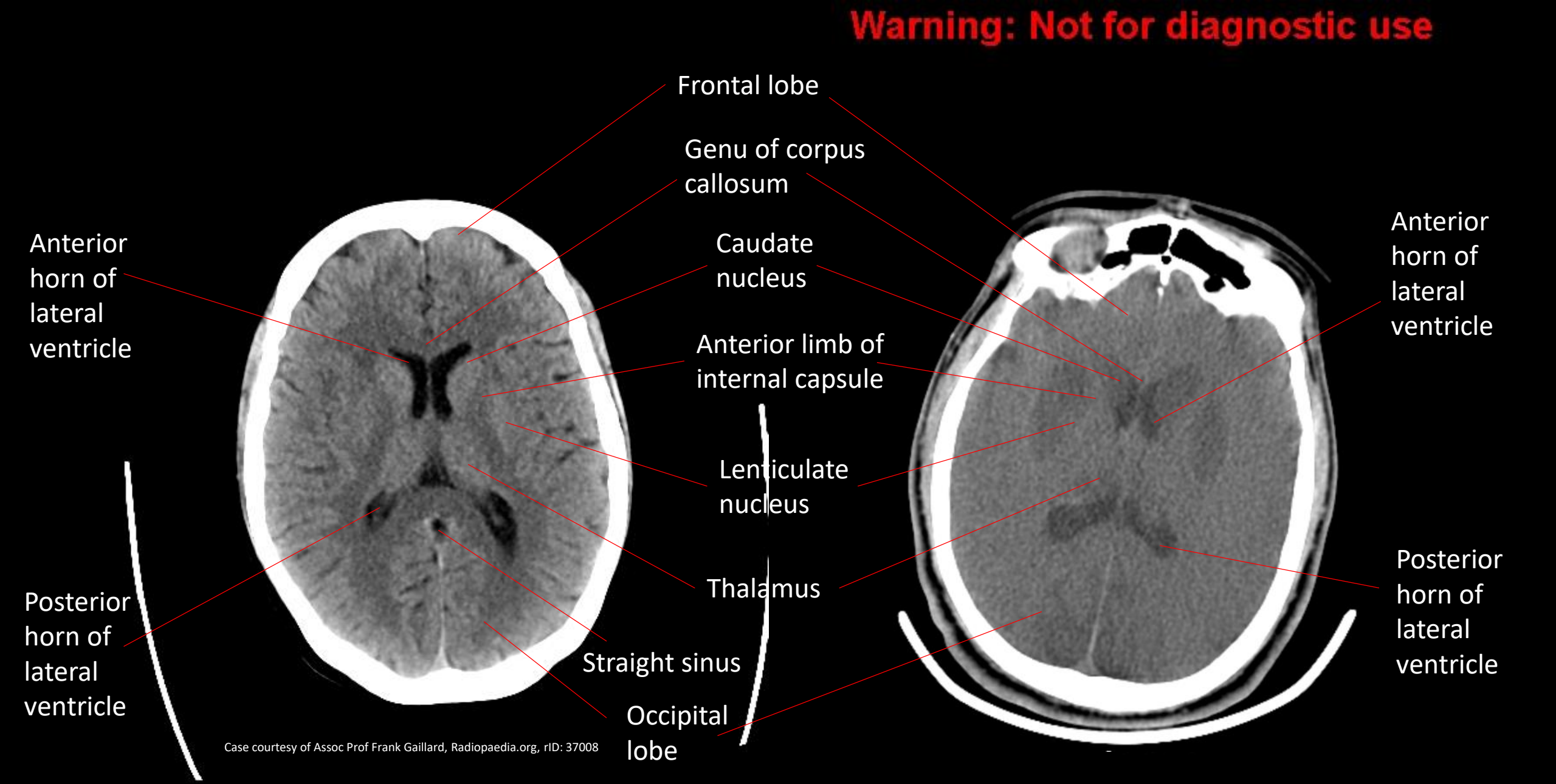
CXR: R tension pneumothorax, chest tube placed

Empiric antibiotics for possible meningitis

Hypothermia protocol initiated

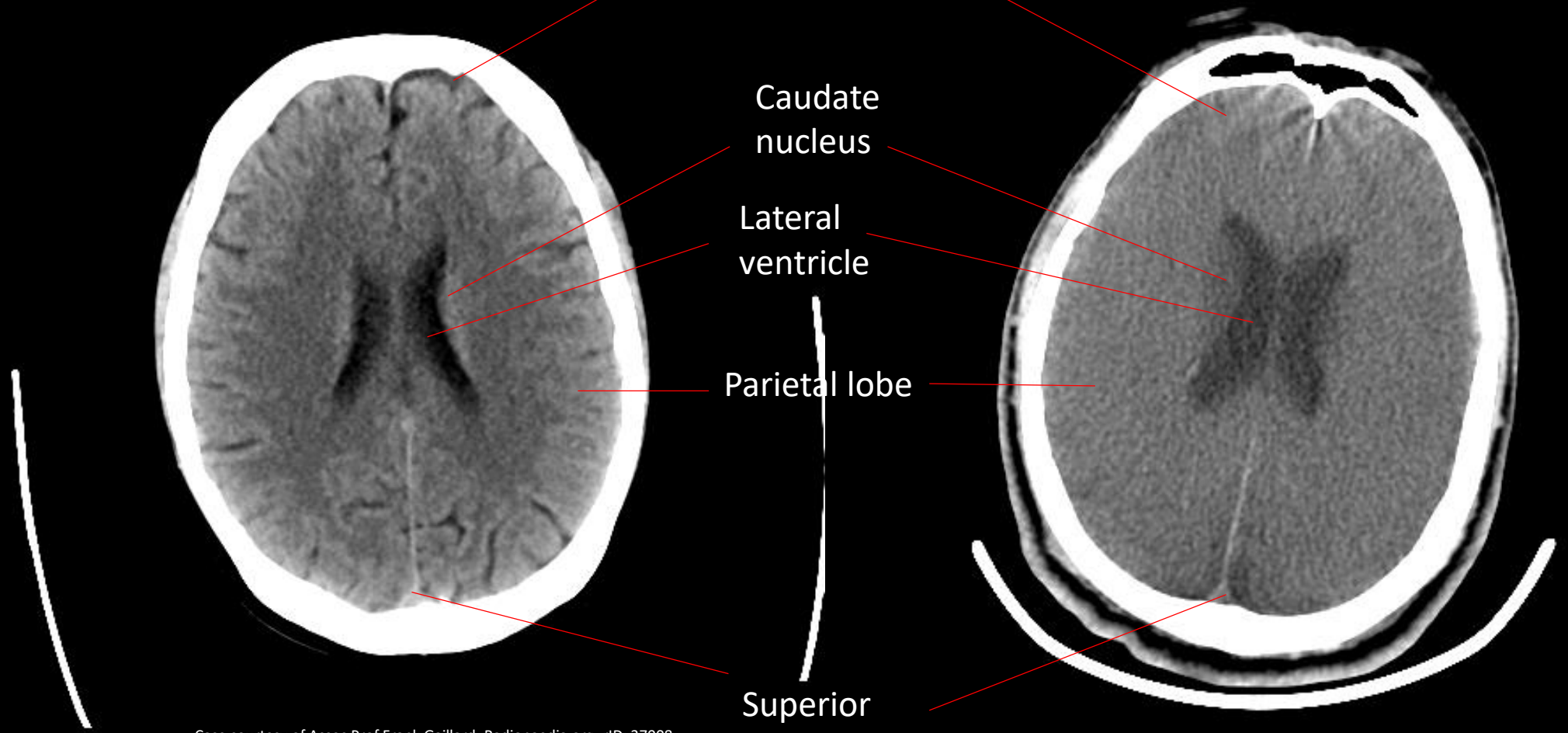
Stat CT brain without contrast

Warning: Not for diagnostic use



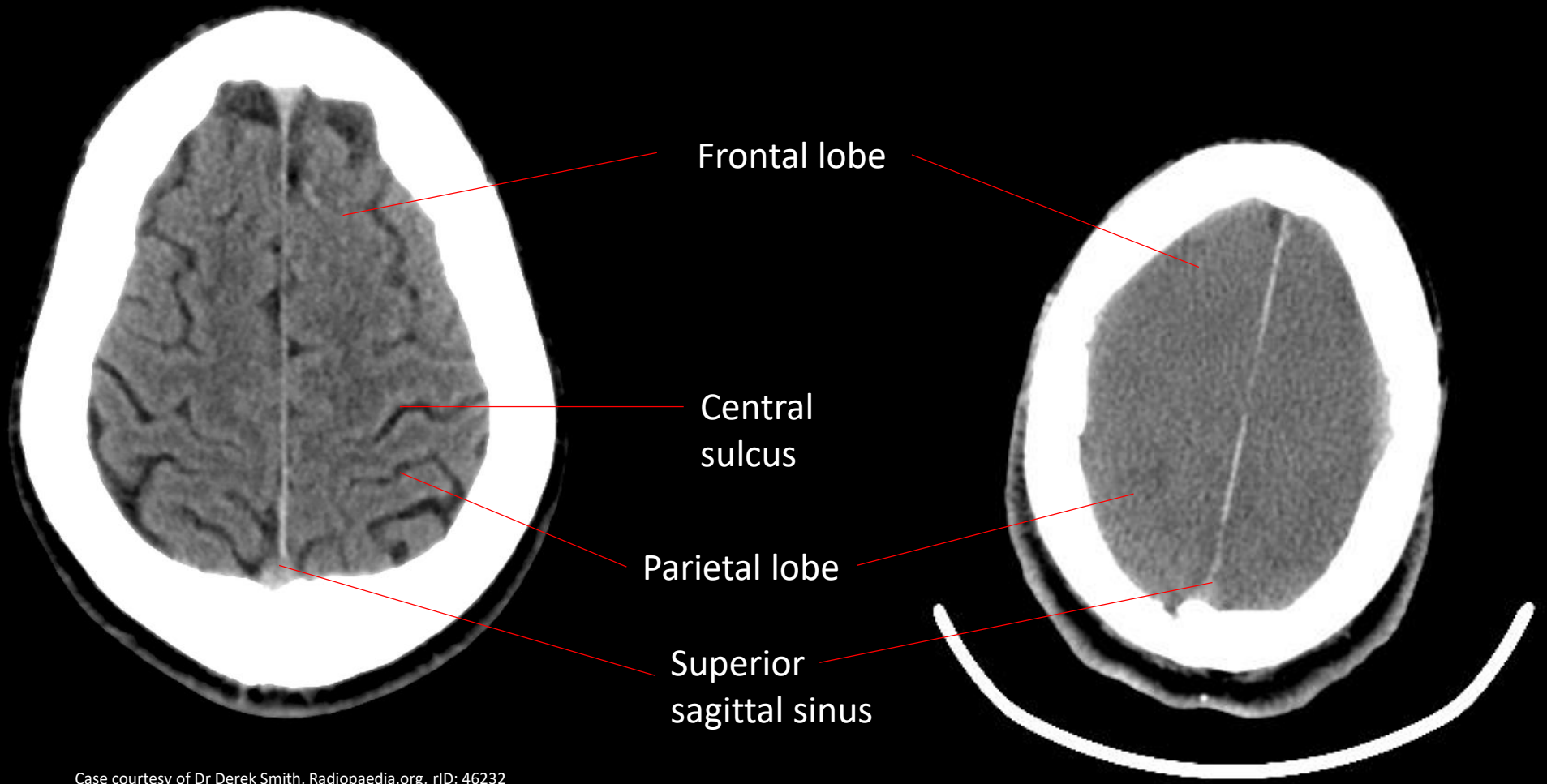
Case courtesy of Assoc Prof Frank Gaillard, Radiopaedia.org, rID: 37008

Warning: Not for diagnostic use



Case courtesy of Assoc Prof Frank Gaillard, Radiopaedia.org, rID: 37008

Warning: Not for diagnostic use



Case courtesy of Dr Derek Smith, Radiopaedia.org, rID: 46232

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

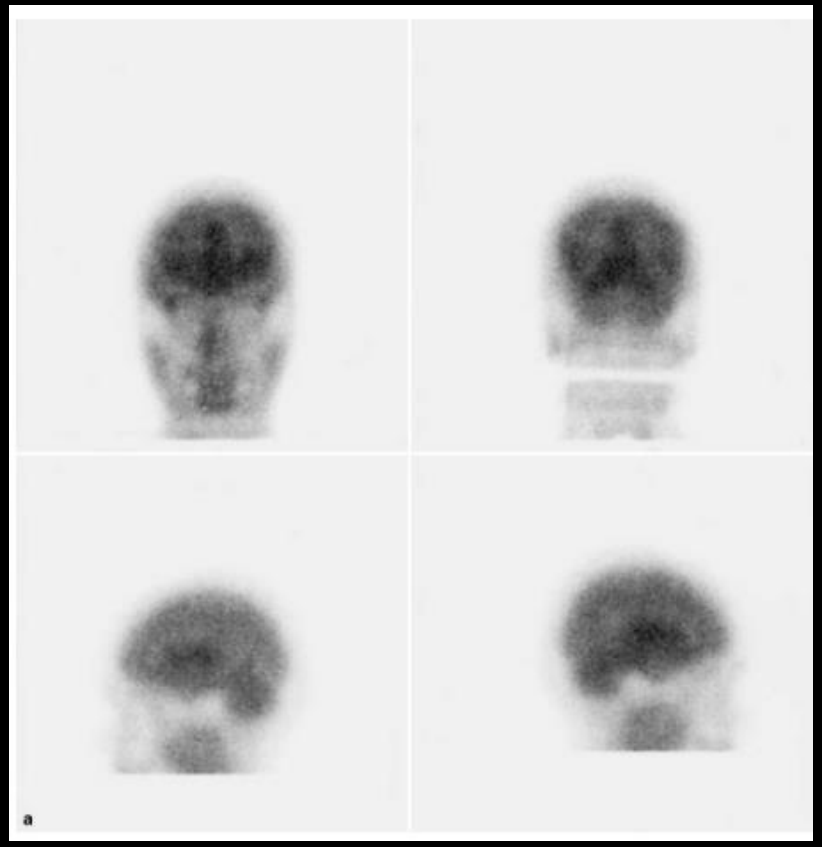
- Pathophysiology of Anoxic Brain injury
  - Primary injury: neuronal death due to ischemia
  - Secondary injury: neuronal death due to imbalance of cerebral oxygen delivery and use – metabolically active tissue hit hardest (e.g. basal ganglia)
- Common sequela of cardiac arrest
- Management is supportive

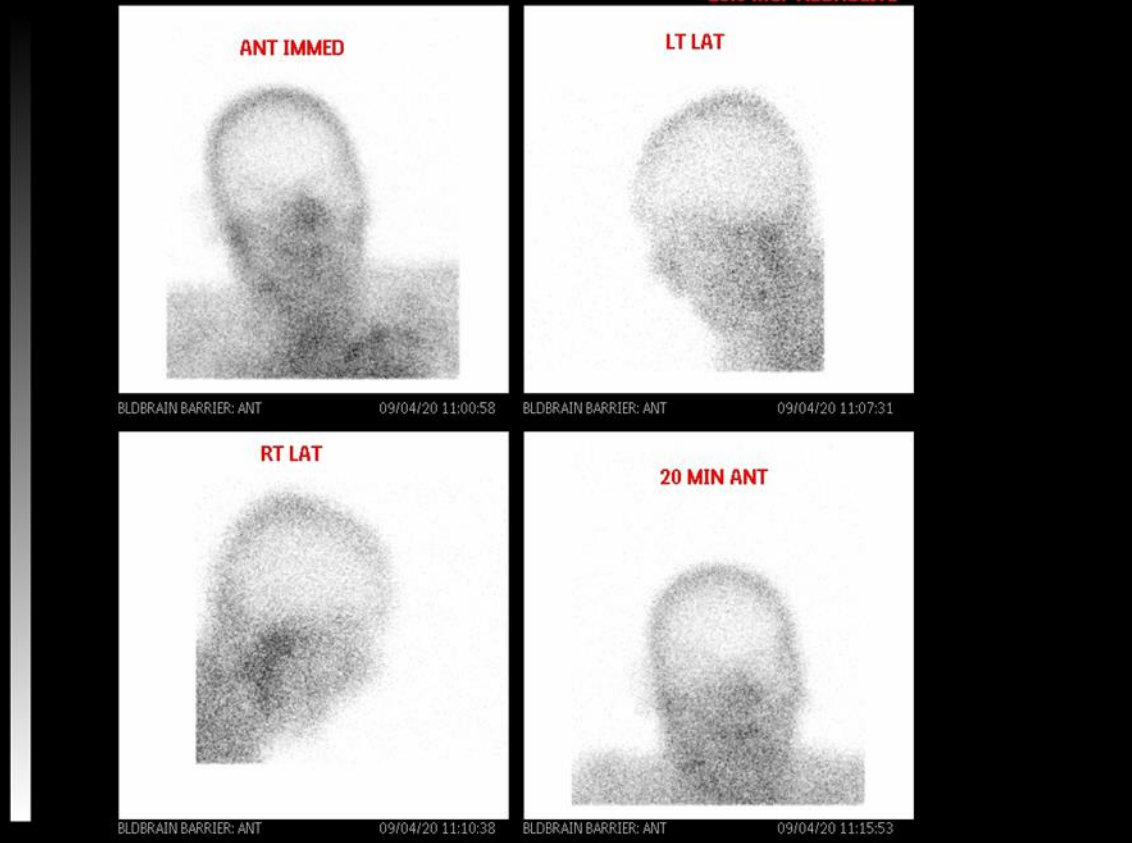
Discussion

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

Outcome

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





Source: Al-Shammri S, Al-Feeli M. Confirmation of Brain Death Using Brain Radionuclide Perfusion Imaging Technique. Med Princ Pract. 2004; 13:267-272.

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

1. Al-Shammri S, Al-Feeli M. Confirmation of Brain Death Using Brain Radionuclide Perfusion Imaging Technique. Med Princ Pract. 2004; 13:267-272.
2. Lacerte M, Hays Shapshak A, Mesfin FB. Hypoxic Brain Injury. [Updated 2020 Aug 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK537310/
3. Sekhon, M.S., Ainslie, P.N. & Griesdale, D.E. Clinical pathophysiology of hypoxic ischemic brain injury after cardiac arrest: a "two-hit" model. *Crit Care* **21,** 90 (2017). https://doi.org/10.1186/s13054-017-1670-9
4. Greer DM, Shemie SD, Lewis A, et al. Determination of Brain Death/Death by Neurologic Criteria: The World Brain Death Project. *JAMA.* 2020;324(11):1078–1097. doi:10.1001/jama.2020.11586


Questions?