

# Neuroradiological findings of hypoxic ischemic encephalopathy post cardiac arrest

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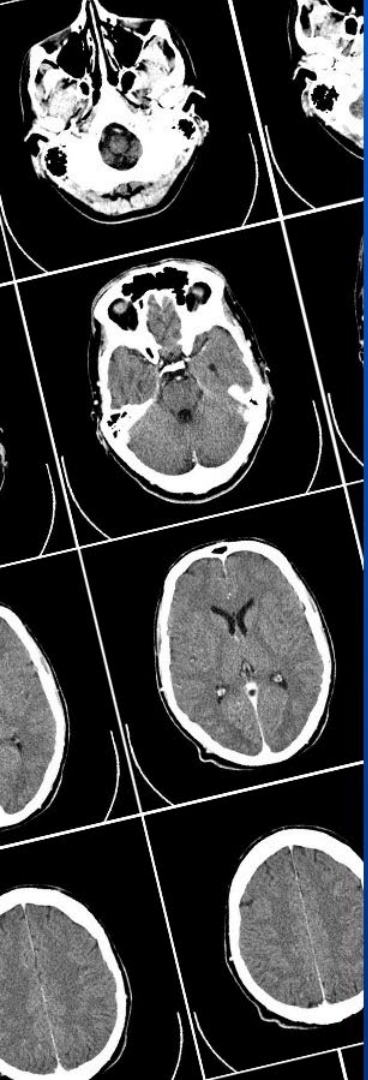


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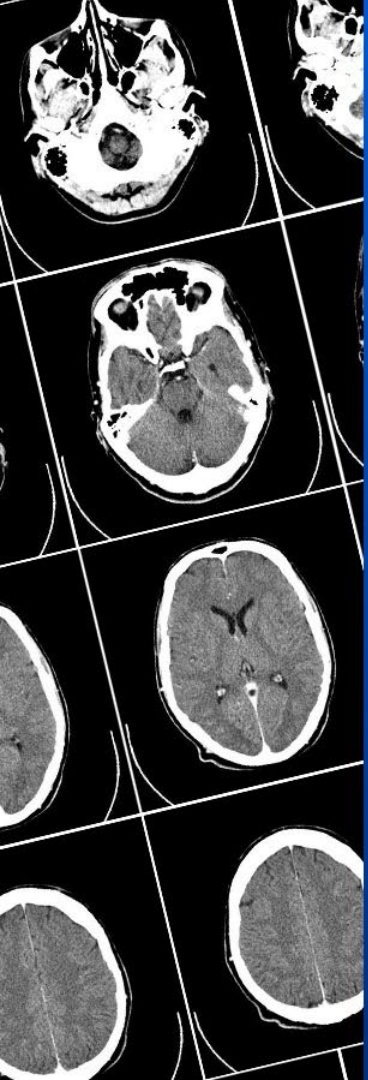
# Disclosure

- Nothing to Disclose



# Teaching points

1. Review the **structures** of the brain most **vulnerable to ischemia** .
2. Review the **imaging protocol post cardiac arrest** to predict neurological outcomes.
3. Review **the imaging appearances of hypoxic ischemic encephalopathy on CT and MRI** and discuss the differential



## Background

Post-cardiac arrest brain injury (PCABI) is the leading cause of death in patients who are resuscitated from cardiac arrest and the leading cause of long-term disability in those who survive the acute phase.



## Areas of brain vulnerable to HIE

- Understanding the selectivity of vulnerable neurons is key in this context. The most vulnerable areas of the brain secondary to anoxic injury are the posterior cerebral areas, including the occipital lobes, which have high resting metabolic rates. Neurons in the hippocampus, cerebral cortex, the striatum, and the Purkinje cells of the cerebellum are also vulnerable. This knowledge about the specificities of PCABI empowers us in our understanding and treatment of this condition.



# Imaging modalities utilized

1. Brain CT and brain MRI are considered in patients post-return of spontaneous circulation (ROSC).
2. CT is preferably performed within 24 hours and commonly shows **cerebral edema with attenuation of grey and white matter**. CT has a high specificity of hospital mortality, and can predict neurological outcomes with a high specificity if read within 24 hours. Current guidelines suggest performing an MRI 2-5 days after ROSC, but it can be performed earlier as well to predict neurological outcomes.
3. The Diffusion Weighted Image (DWI) is best to predict outcomes; **hyperintensity indicates loss of perfusion**, and the absence of DWI abnormalities one week after ROSC predicts good outcomes.
4. But Brain imaging alone is not enough, it is combined with other predictors like **EEG** and neurological examination findings to predict outcomes of the patient.

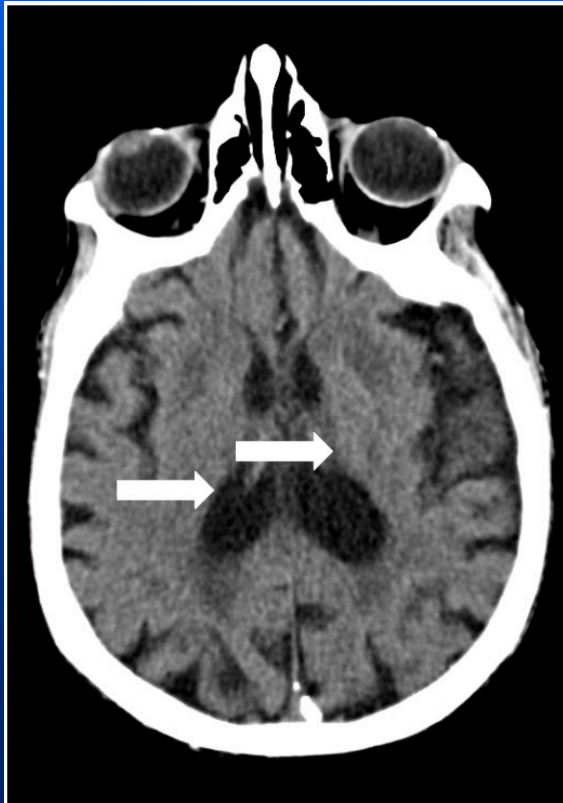


# Findings on imaging

1. On imaging CT displays edema with effacement of the CSF-containing spaces and decreased cortical grey matter and basal ganglia attenuation.
2. Reversal of the standard CT grey and white matter attenuation.
3. White cerebellum signs show high attenuation of the cerebellum and brainstem relative to the cerebral hemispheres, and pseudo subarachnoid signs have hyperdense subarachnoid spaces.
4. When an MRI is read, the DWI and FLAIR are preferred, as DWI displays restricted diffusion in the cerebellar hemispheres, basal ganglia, or cerebral cortex, which are seen as hyperintense regions in the brain. The thalami, brainstem, or hippocampi may also be involved.



**Case 1 An 88 year old Female was brought in after being found unresponsive and in cardiac arrest for approximately 30 to 60 minutes . A CT and MRI was done.**



**Figure 1.**CT head of 88-year-old female without contrast a. shows cerebral volume loss with mild prominence of the lateral ventricles. interval development of **bilateral hypoattenuation of the caudate nuclei and lateral basal ganglia.**



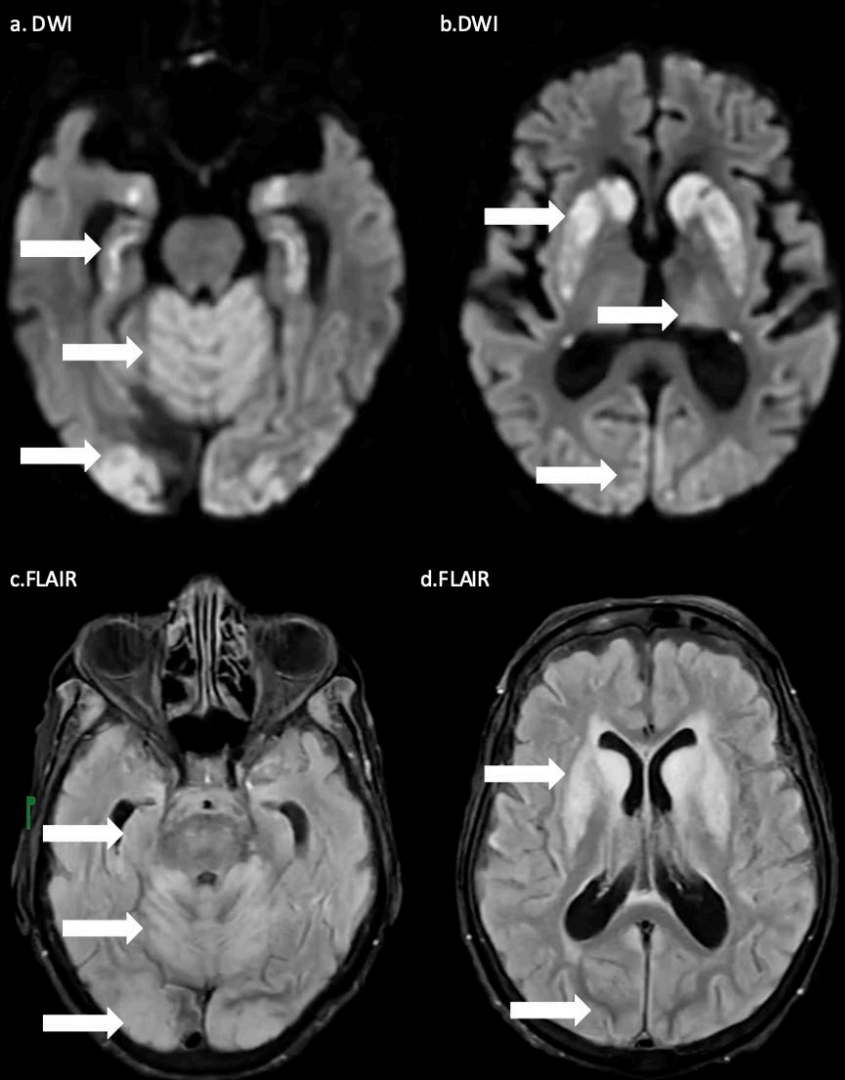


Figure 2. MRI of 88-year-old female a and b . Axial DWI and c and d.FLAIR displays hyperintensity with significant restricted diffusion observed in :

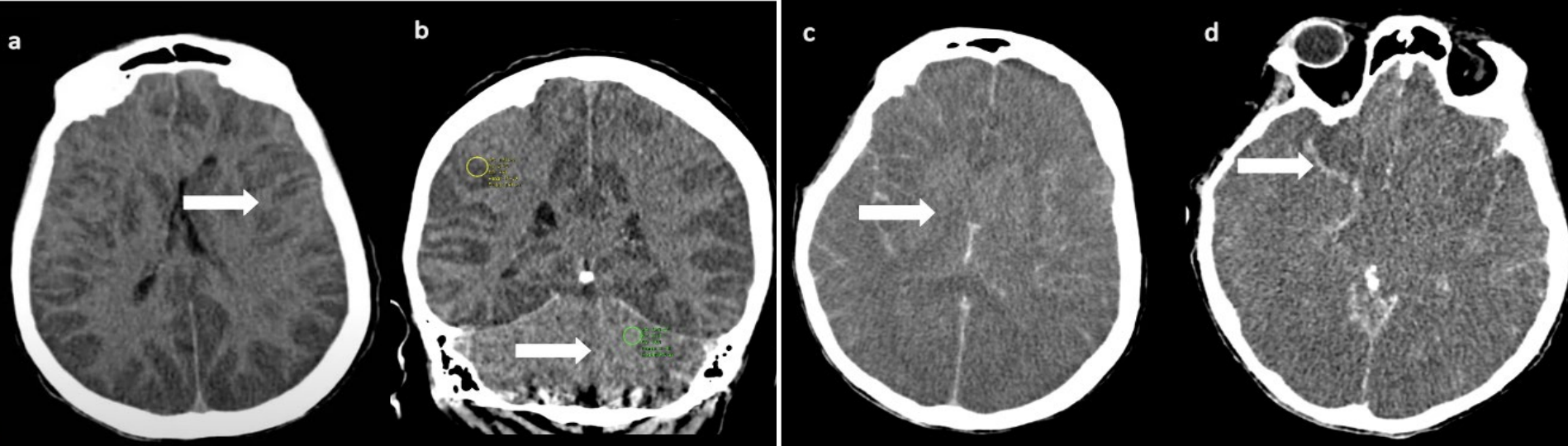
- **a and abnormal hyperintensity bilateral occipital lobes, hippocampus and cerebellum**
- **b and minimally restricted diffusion of bilateral thalami with left greater than right, hyperintensity of bilateral basal ganglia and posterior parietal lobes**





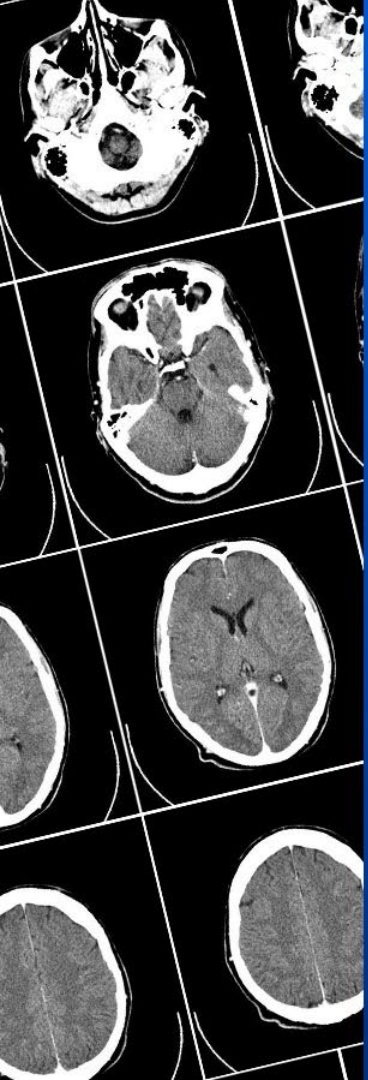
**Case 2** A 75-year-old female was brought in by EMS after being found unresponsive and in a cardiac arrest . Only a CT was done.

- **Grey-white matter reversal** indicates severe, irreversible brain damage. The Grey to White Ratio at the basal ganglia level of brain CT shows the strongest correlation with post-cardiac arrest neurological outcomes.
- **Pseudo-SAH**, caused by basal cistern effacement, elevated intracranial pressure, and enlarged superficial veins, can also be observed.

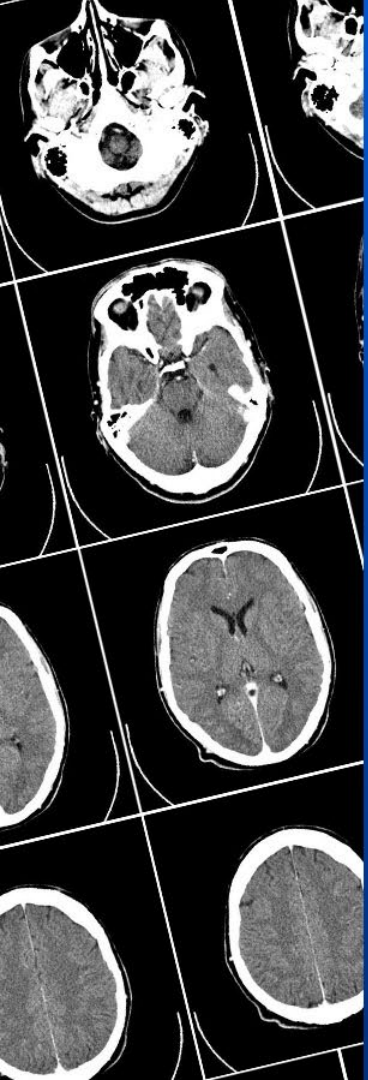


**Figure 3.** CT head of 75-year-old female without contrast on day 1- a. and b. And day 4- c. and d.

- a. Shows sulcal effacement and reversal of grey- white matter on axial soft tissue
- b. CT shows cerebellum sign with higher attenuation of cerebellum than cortex
- c. CT shows progression of edema and loss of contour of the ventricle at the same level
- d. pseudo "subarachnoid sign"



- **Treatment**
  - Derangements in temperature, arterial blood pressure, oxygenation, and ventilation should be managed. No definitive treatment.
- **Prognosis**
  - Outcomes are poor
  - 5% of patients resuscitated with conventional CPR and more than 20% of those resuscitated with extracorporeal CPR were diagnosed with brain death, corresponding to 8% and 28% of all deaths, respectively.
- **Differential diagnosis** : carbon monoxide poisoning , molybdenum cofactor deficiency



# Conclusion

- Neuroimaging is a biomarker of neurological outcomes of the patient , brain CT and brain MRI are important to be considered in patients post-return of spontaneous circulation (ROSC). While the CT gives limited findings , the grey to white matter ratio is not abnormal in this case indicating less severity. Whereas on MRI the DWI is an important marker of poor outcome prediction and long term cognitive deficit in survivors.



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