48-year-old female s/p cardiac arrest now intubated and sedated in the ICU

Bennett Propp, MS3

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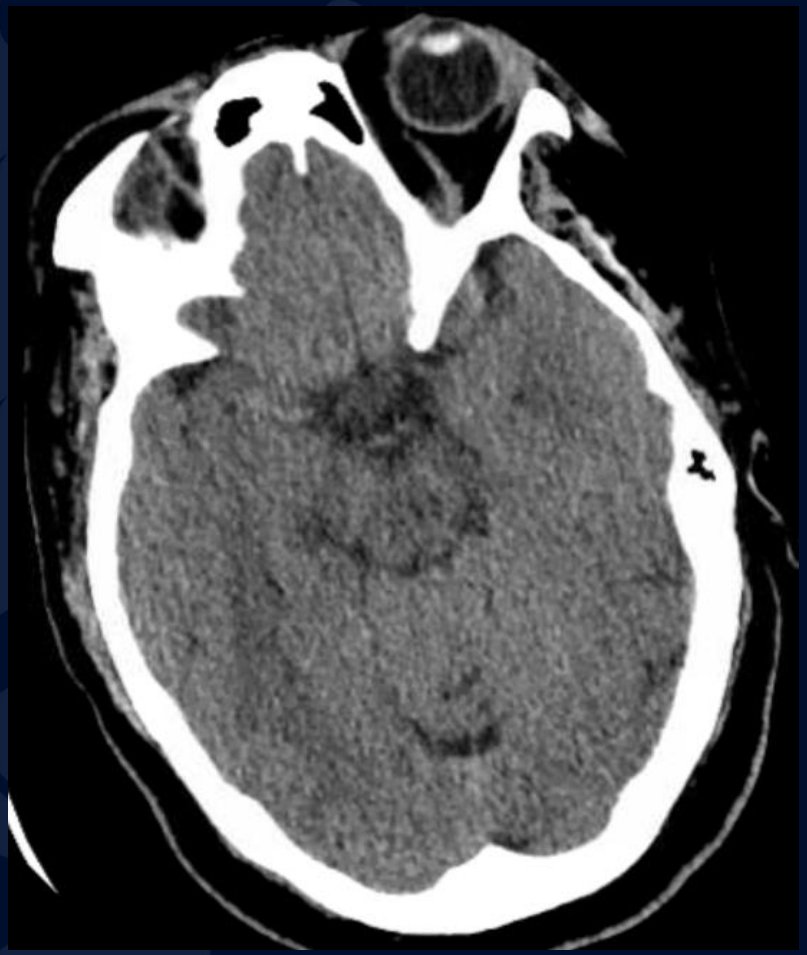
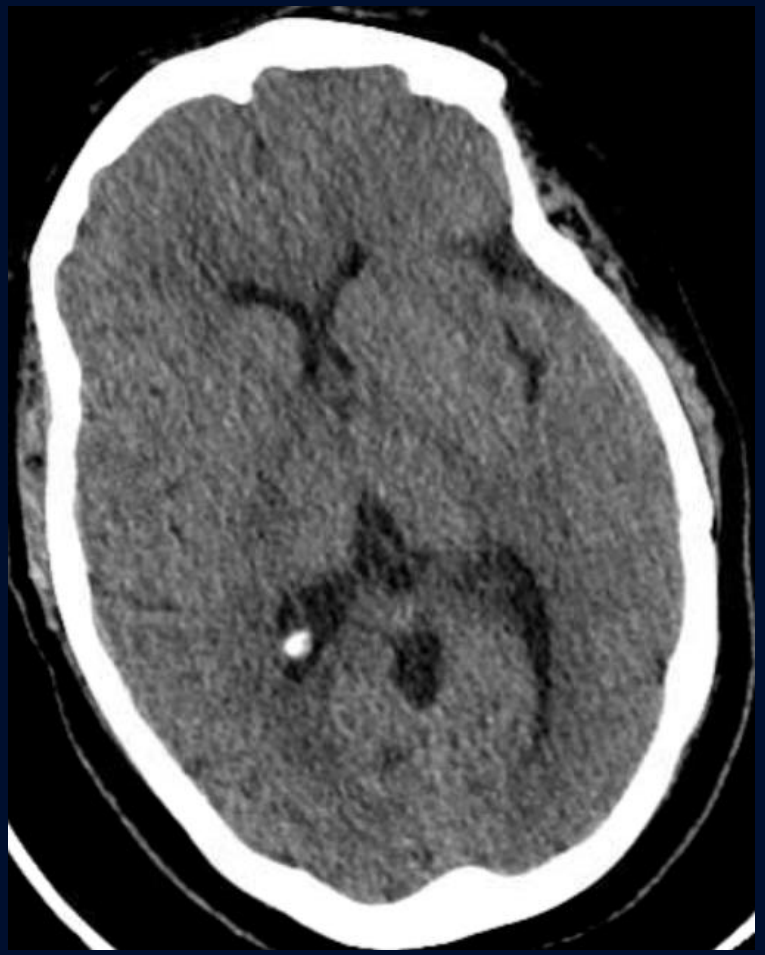
Non-contrast CT





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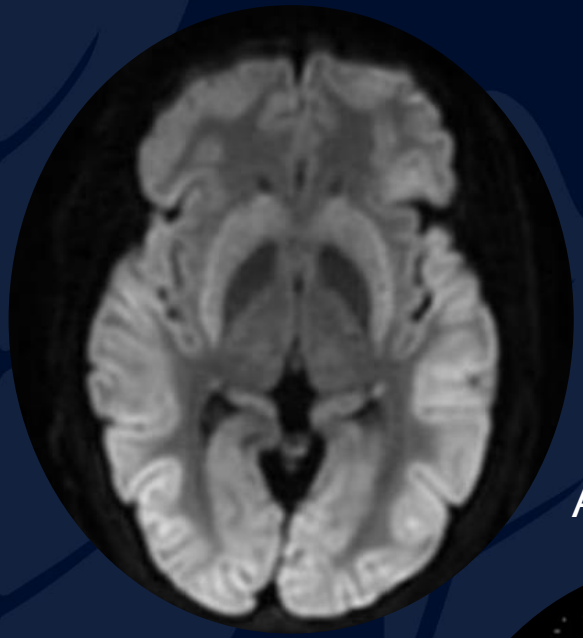
DWI



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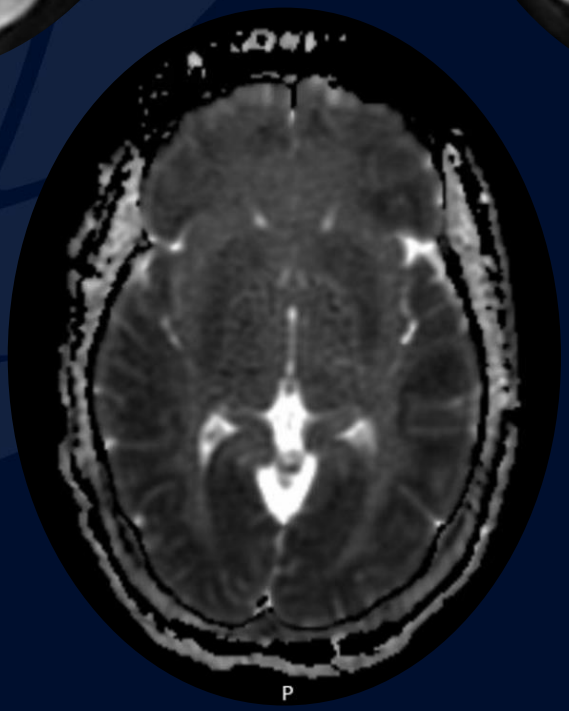

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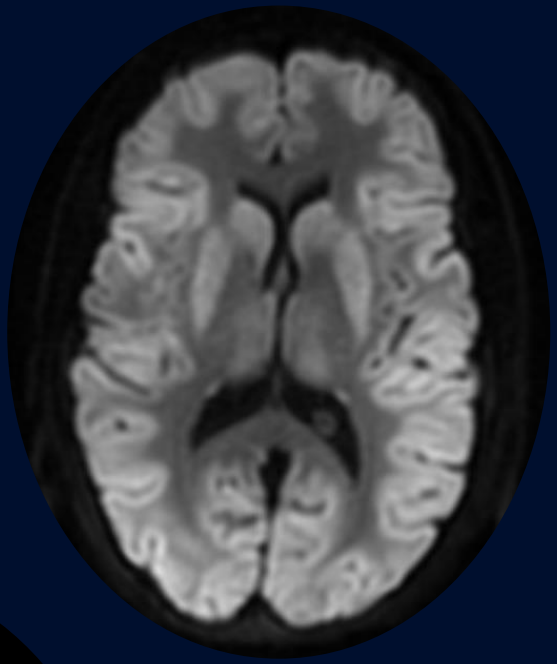

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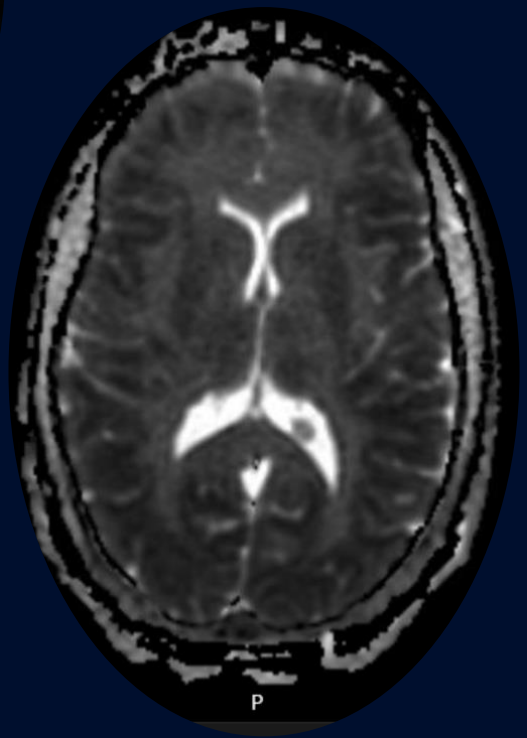

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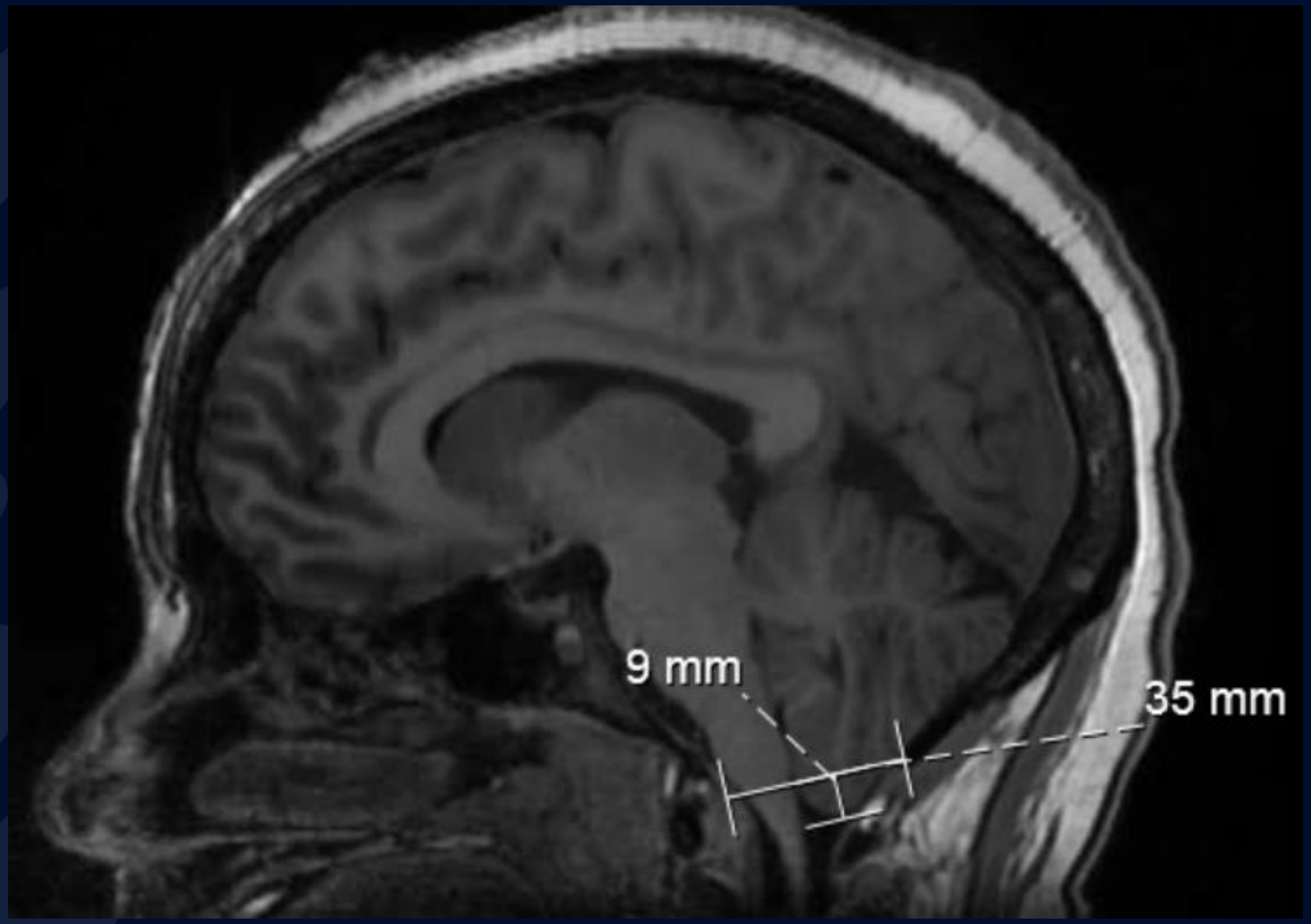
T1 Sagittal



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Anoxic Brain Injury

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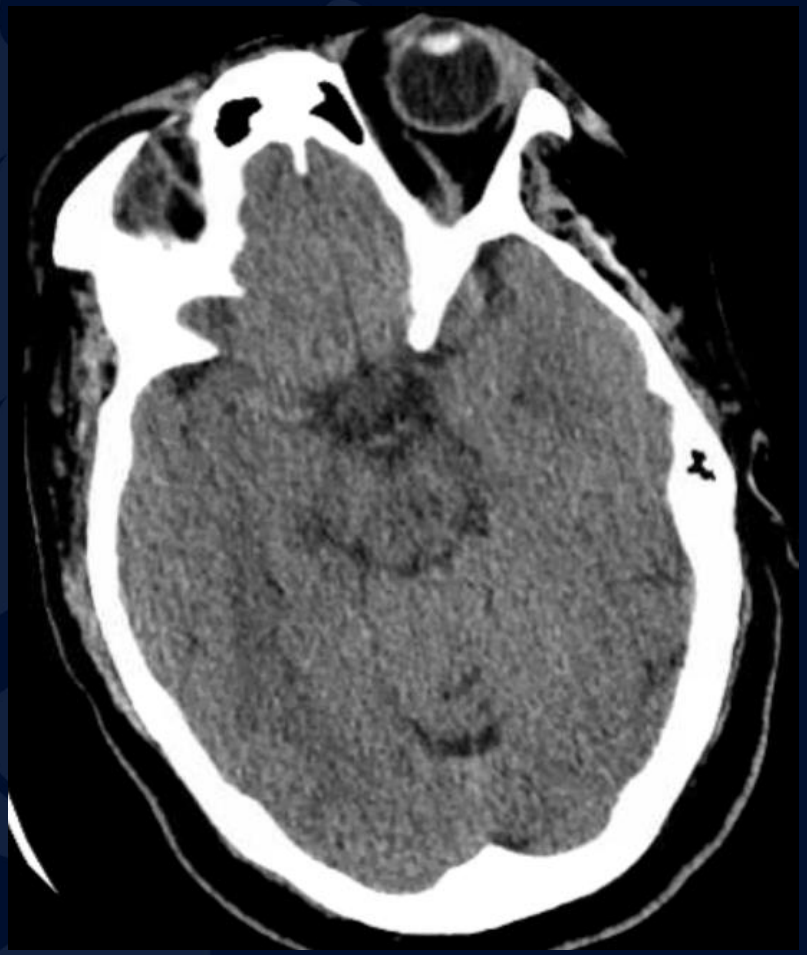
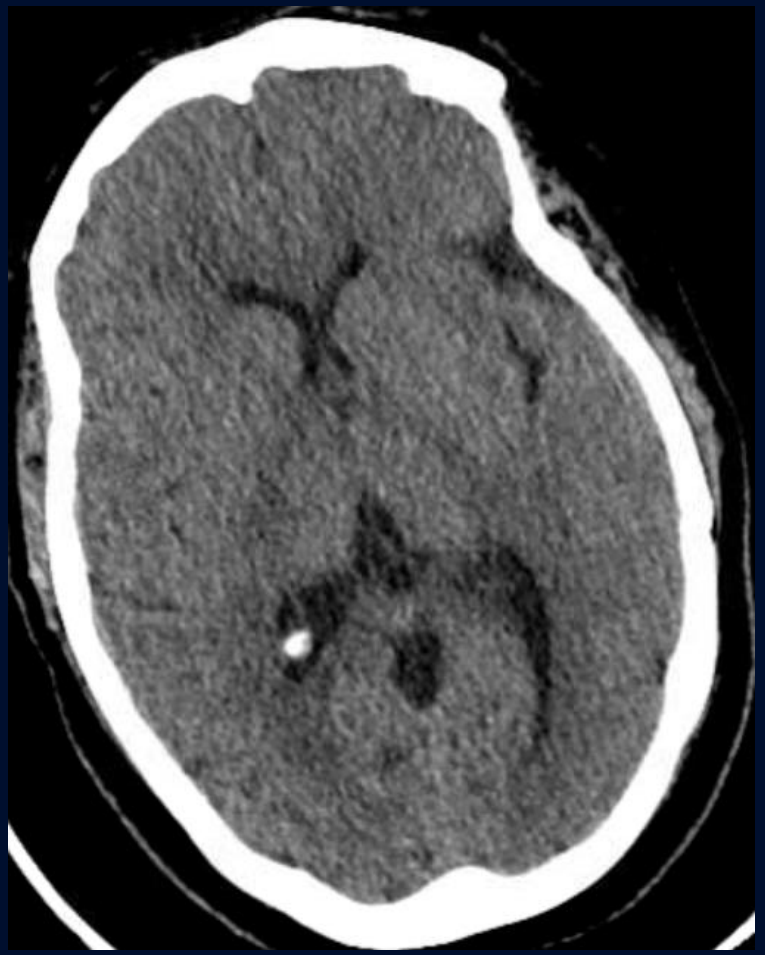
Non-contrast CT





No acute intracranial hemorrhage or territorial infarction

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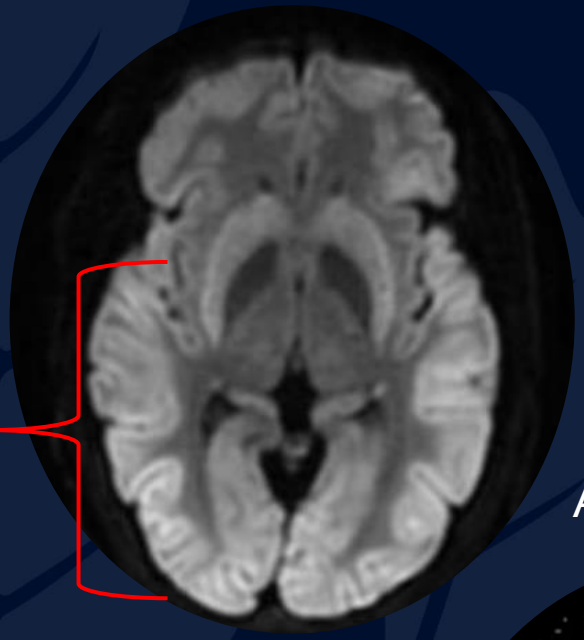
DWI



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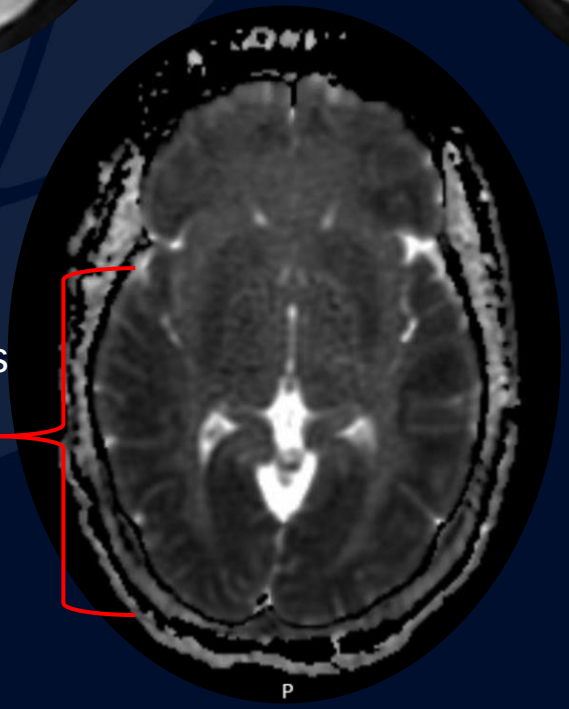
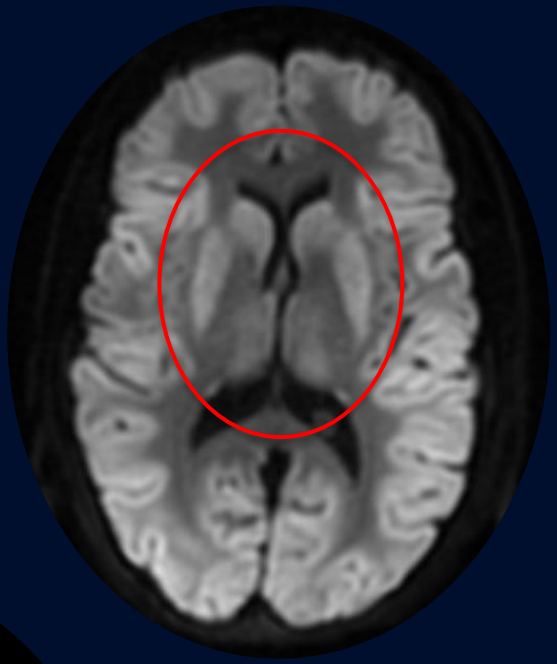


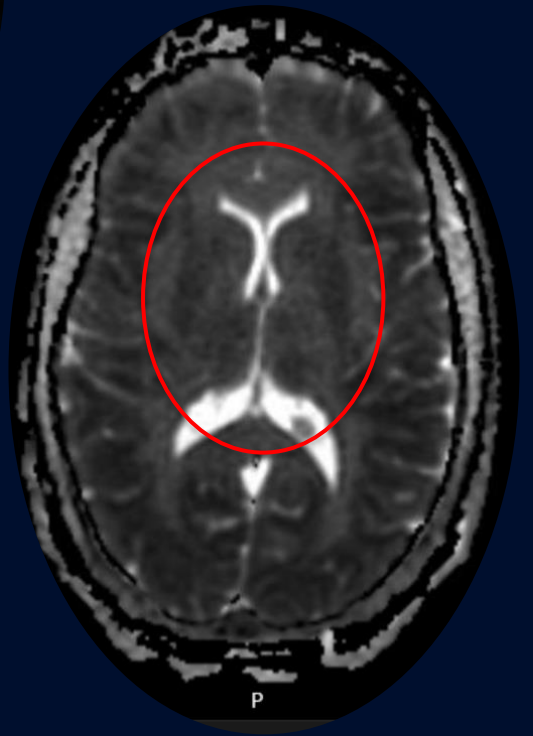
Extensive restricted diffusion throughout bilateral cerebral hemispheric cortices

DWI



Extensive restricted diffusion in bilateral caudate, lentiform nuclei, and thalami

ADC



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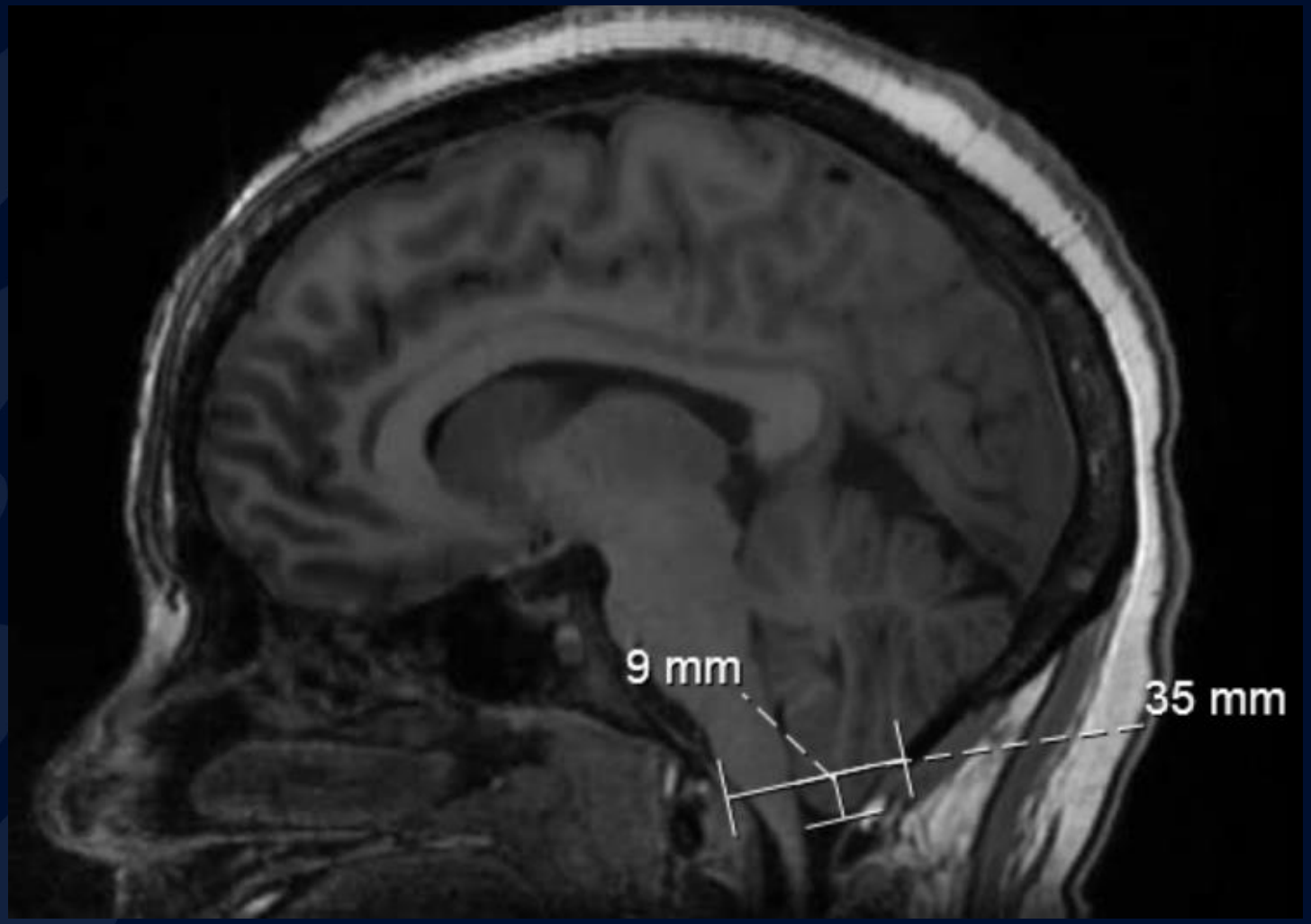
T1 Sagittal



9 mm cerebellar tonsillar herniation

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Anoxic Brain Injury

- Etiology
  - Decreased oxygen delivery to the brain
  - Cardiac arrest
  - Vascular injury/insult
- Pathophysiology
  - Reduced blood supply → reduced O_2 and glucose delivery → anaerobic respiration → reduced efficiency of ATP production → Na/K ATPase fails → influx of sodium into cell → massive depolarization → rapid rise in intracellular calcium → cell death
  - Cell death mechanisms
    - Mitochondrial injury leads to generation of ROS
    - Activation of nitric oxide, caspases, calpains
    - Disruption of protein synthesis
    - Lactic acidosis
- Clinical Findings
  - Must eliminate confounding factors
  - Physical Exam – Myoclonic activity or myoclonic status epilepticus
    - Usually generalized
    - May be observed within 24 hours after hypoxic insult
    - Delayed myoclonus up to 48 hours after insult
- Evaluation
  - Rule out confounding metabolic abnormalities
    - Serum electrolytes
    - Hepatic studies
    - Blood gases
    - H&H
  - Cardiac evaluation – echo, cardiac biomarkers
  - Imaging – CT/MRI

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Diagnostic Findings

Modality	Change	Time for changes to present
Non-contrast head CT	• Cerebral edema • Inversion of gray-white matter density	3 days
EEG	• Isoelectric EEG • EEG with voltage lower than 20 mV • Burst suppression EEG with a subcategory of burst suppression with identical bursts • Epileptiform EEG including status epilepticus and periodic discharges • Continuous activity less than 8 Hz • Continuous activity equal to or greater than 8 Hz	Within 10-40 seconds

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MR Imaging Findings

Chronological magnetic resonance imaging findings in anoxic/hypoxic encephalopathy

	Acute phase (<24 hours)	Early subacute phase (24 hours to day 13)	Late subacute phase (days 14 to 20)	Chronic phase (>21 days)
Characteristics	Brain swelling	Brain swelling	Absence of brain swelling	Diffuse atrophy and dilatation of the ventricles
DWI	Hypersignals in the cortex, in the thalamus and in the basal ganglia	Hypersignals in the cortex, in the thalamus and in the basal ganglia	Progressive disappearance of hypersignals found previously	Normal
T2	Hypersignals in the cortex, in the thalamus and in the basal ganglia	Hypersignals in the cortex, in the thalamus and in the basal ganglia. Possible subcortical hyposignals	Hypersignals of the cortex, the thalamus, the basal ganglia and the pons	Normal or possible hypersignals of the cortex, the thalamus, the basal ganglia and the pons
T1	No abnormalities	No abnormalities	Possible spontaneous subcortical and basal ganglia hypersignals	Can be normal
T1 with gadolinium enhancement	No abnormalities	Possible subcortical enhancement suggestive of cortical laminar necrosis	Possible subcortical enhancement suggestive of cortical laminar necrosis	No abnormalities
Comments	DWI seems more sensitive to mild hypoxic/anoxic injury in the first hours, and the hypersignal in cerebral cortex seems more precocious than in the basal ganglia	Hypersignals on both DWI and T2 become more intense, particularly in the thalamus and the basal ganglia	In some cases, appearance of diffuse white matter, abnormalities of delayed anoxic leukoencephalopathy on both DWI and T2	In some cases, hypersignals of the cortex and hyposignals in the subcortical zone on both T2 and T1, suggestive of cortical laminar necrosis

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