Neuroimaging in hypoglycaemia beyond the neonatal age-group

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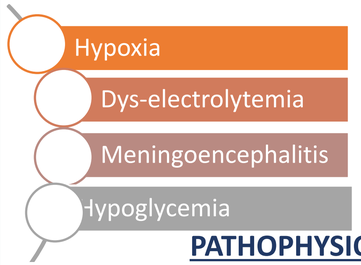
INTRODUCTION

- Hypoglycaemia: decrease in serum glucose level < 54 mg/dL (3.3mmol/L).

- Neurologic symptoms: profound memory loss, transient focal deficits, persistent vegetative state, death in 2- 4% in adults. Symptoms in infants are non-specific.

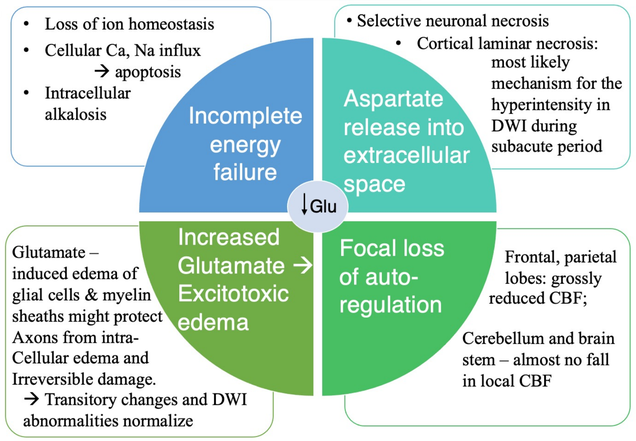
- Cerebral cortex, hippocampus, and basal ganglia are commonly affected. Cerebellum and brain stem are usually spared.

- Scarce literature on infantile hypoglycemia.



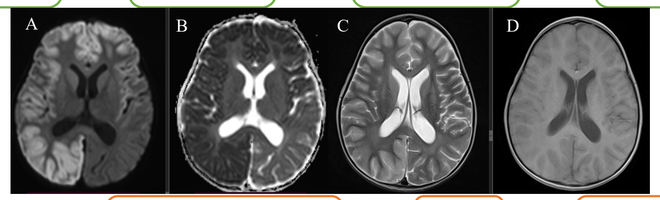
Need to diagnose and treat early for good prognosis.

PATHOPHYSIOLOGY



CASES

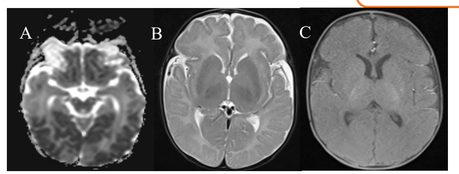
1 yr 8 mon/ F, Born to NCM, Normal Birth, development → Skin hyperpigmentation since 1 yr age → Now presented with hypoglycemia for 2 hr → MRI: diffusion restriction in right hemi-cortex and left frontal cortex → Addison's disease (LP Homozygous mutation in NNT gene) → Succumbed after 1 month



A: DWI- diffusion restriction in right cortex and left fronto-temporal cortex. B: low ADC maps in corresponding areas.

C: Subtle T2 hyperintensities in right hemi-cortex and left fronto-temporal areas. D: T1 normal

32 wk, 1.4 kg, NICU- respiratory distress, No Seizures → 5 mon/ male → H/o poor weight gain C/o fever, vomiting, irritability, lethargy for 2 days → RBS: Low → MRI: diffusion restriction in B/L frontal, occipital cortex → Succumbed after2 days: Refractory shock with ARDS and pneumonia



A: low ADC maps in bilateral parieto-occipital, basi-frontal, temporal region. B, C: T2 and T1 images: normal

DISCUSSION

- 3 imaging patterns: GM involvement- cortex, neostriatum, & hippocampi; WM involvement- periventricular WM, internal capsule, and splenium; Mixed pattern- GM & WM.
- GM can show delayed presentation of DWI abnormalities. So, follow sequential images when initial DWI lesions are limited to WM.
- Asymmetric DWI changes- regional imbalances between energy supply and demand, spreading depression, excitotoxic mechanism, regional brain glucose content, glucose influx, amino acid distribution or selective inhibition of cerebral protein synthesis. Thalamus, brain stem, and cerebellum are generally spared. Symmetrical thalamic lesions in hypoxic encephalopathy differentiates it from hypoglycemic encephalopathy.
- Although the clinical outcome is correlated with the severity and duration of hypoglycemia, the duration is often uncertain. Involvement of ≥ 2 lobes → only partial recovery or death, whereas involvement of 1 lobe → complete recovery. The distribution of DWI lesions may be useful for predicting prognosis. Better outcome: WM involvement > fronto-parietal cortex > diffuse cortical/ BG involvement.

HOW IS IT DIFFERENT FROM NHBI?

Definition of hypoglycaemia (<45 mg/dL)
Presence of maternal and foetal risk factors
Transient neonatal glucose homeostatic changes
Patterns of brain injury: parieto-occipital lobe, frontal lobe, splenium, others: internal capsule (posterior limb), basal ganglia, thalami, focal periventricular white matter
Fast occipital axonal growth and migration, synaptogenesis in neonates require more glucose

CONCLUSION

- MRI (DWI) - best modality for early diagnosis and prognostication of hypoglycaemic encephalopathy.

- Hypoglycemic brain injury is different beyond the neonatal age group. Splenial involvement may not be common beyond neonatal age group.

- Extensive cortical involvement can be a predictor of poor prognosis.

- Sparing of thalami helps in differentiating hypoglycemic from hypoxic encephalopathy.

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