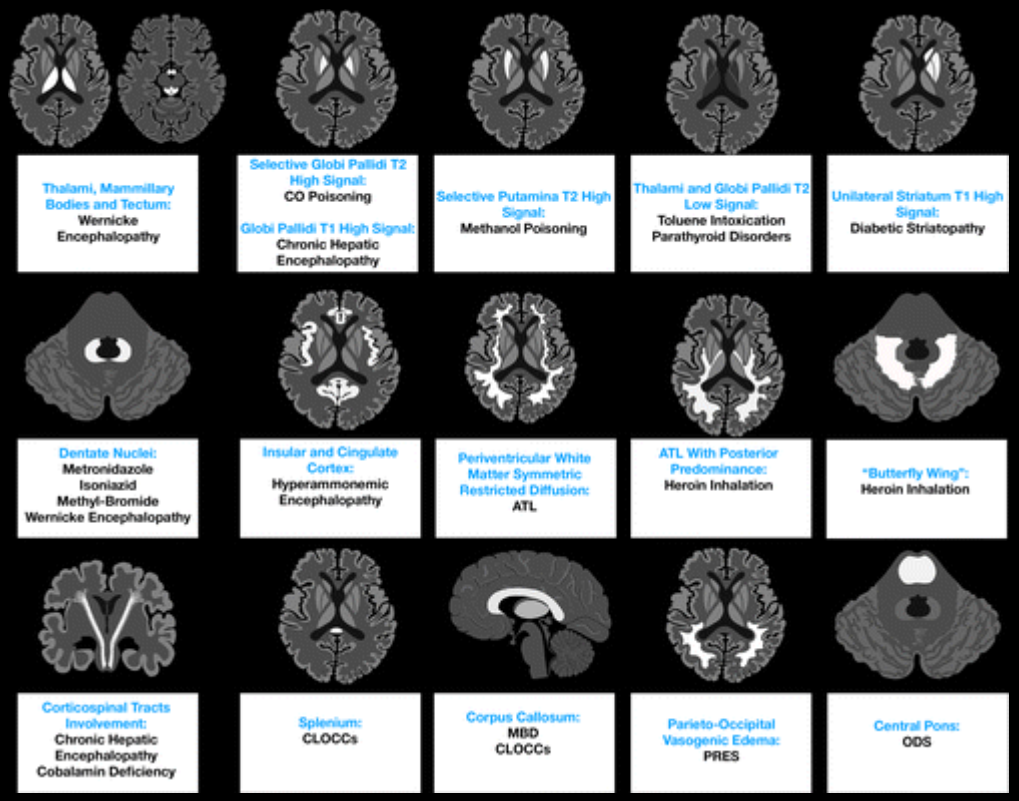

TOXIC/METABOLIC ENCEPHALOPATHIES

METRONIDAZOLE TOXICITY
Dysarthria, ataxia, dysmetria, confusion in patient on prolonged therapy.
MR: bilateral symmetric ↑T2 signal in dentate nuclei, vestibular nuclei, superior olivary nuclei.
May also appear like acute toxic leukoencephalopathy, reversible splenial lesion.
DDx: Isoniazid, Methyl-bromide, WE

WERNICKE ENCEPHALOPATHY/THIAMINE DEFICIENCY
Ataxia, nystagmus, opthalmoplegia memory impairment in alcoholic (inhibits B1 absorption in jejunum).
↓transketolase activity → destruction of MBs, eventual atrophy
MR: ↑T2/FLAIR in medial thalami, PAG, tectal plate, mammillary bodies (MBs enhance*) +/- perirolandic cortical involvement
EtOH ENCEPHALOPATHY
Disproportionate superior vermian atrophy, enlarged lateral ventricles and sulci.

MARCHIAFAVA-BIGNAMI
Confusion, altered gait, seizure, rigidity in red wine drinker.
MR: ↑T2 in corpus callosum (body, genu, then splenum).
"Sandwich sign": selective involvement of middle layers.
Chronic MBD → thinned CC with cysts in body and splenium.
DDx/other splenial:
Reversible Splenial Lesion (related to viral infection, metabolic abnormality, withdrawal. Ovoid ↑T2/FLAIR lesion in the central splenium with restricted diffusion)

METHANOL POISONING
Bilateral hemorrhagic necrosis of putamen
MRS: Large lactate peak
DDx: ↑T2 in GP
Anoxic infarcts
CO2 poisoning (GP >> putamen)
Wilson (also ↑T2 in brainstem)
Leigh (↑T2 in brainstem, BG, WM in infant/kid)
CJD (↑T2 in BGC&P>GP, thalamus, cortex)
Huntington (caudate atrophy, ↑T2 in caudate and putaamen)

CARBON MONOXIDE POISONING
High hgb affinity for CO → impaired O2 transport.
GP highly sensitive to hypoxia → necrosis.
CT: Hypointense GP
MR: ↑T2/FLAIR GP with restricted diffusion.
May also involve hippocampi, caudate, putamina, cerebellum, corpus callosum, cerebral cortex. 1/3rd develop progressive WM demyelination-confluent ↑T2 in periventricular and centrum semiovale WM.

HYPOGLYCEMIA (Adult)
MR: ↑T2/FLAIR + restricted diffusion in parietooccipital-occipital and temporal gyri. BG involvement correlated with worse prognosis. **Spares thalamus** (unlike severe hypoxia, with bilateral thalamic injury), WM, cerebellum.
MRS: ↓lactate, ↓NAA

HYPOGLYCEMIA (Neonate)
MR: Restricted diffusion in bilateral parietooccipital cortex. WM abnormality may predominate in premature infants (highly active oligodendrocytes deprived of glucose), typically involves optic radiations +/- posterior thalami.
DDx:
HIE (symmetric involvement of thalamus and cerebellum)

HERIONE INHALATION LEUKOENCEPHALOPAHY
MR: Confluent T2/FLAIR "butterfly" in centrum semiovale, high signal in PL of IC (spares AL), supratentorial and infratentorial white matter with sparing of dentate nuclei. Rarely involves BG.
Extensive white matter disease → gliosis.

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME
A/w pregnancy, hypertension, TTP, chemotherapy, cyclosporine A, tacrolimus, uremia, interferon-A
MR: Posterior, bilateral vasogenic edema without restricted diffusion (may be DWI bright but no ADC correlate). Parietooccipital preference.
Imaging lags clinical picture for weeks.

CENTRAL PONTINE MYELINOLYSIS
2-6 days after rapid correction of hyponatremia, liver transplant.
MR: restricted diffusion in lower pons, then ↑T2 in central pons.
Spares CST (unlike cyclosporine A toxicity).
Enhancement and mass effect are rare.
MR doesn't correlate with clinical status.

FAHR DISEASE
Dystonia, Parkinsonism, neuropsychiatric manifestations
"BG calcifications in patient < 50 with schizophrenic-like symptoms
CT: bilateral, symmetric calcifications in BG (GP most common), thalami, dentate nuclei, cerebral white matter. Also in IC, cerebral WM. SWI may be more revealing.
DDx BG calcifications:
Normal aging brain
Inherited causes of BG calcifications
(Downs, mitochondrial, Aicardi Syndrome, Cockayne Syndrome, phakomatoses)
Acquired causes of BG calcifications
[HIV (BG calcs and cerebral atrophy), radiation/intrathecal chemotherapy]
Pathologic causes of BG calcifications
(endocrinopathies, lupus, motor neuron disease, post infectious, CO2, lead)

PARATHYROID DISORDERS
CT: Bilateral, symmetric calcifications in GP, P, C
Primary: Salt & pepper skull
Secondary: plaque-like dural calcifications, pipestem calcifications of carotids
Brown Tumor: expansile lytic lesions with nonsclerotic margins
MRI: ↑T1, ↑T2 BG, cortex, dentate nuclei (calcifications).
DDx: (same as Fahr)
↑T1 BG
(hepatic encephalopathy, hyperalimentation, Wilson, nonketotic hyperglycemia)

HEMICHOREA-HEMIBALLISMUS
~70yo with hemiballismus and non-ketotic (~450 mg/dL) hyperglycemia
CT: Unilateral hyperdense BG contralateral to symptoms
MR: corresponding ↑T1 in BG

MANGANESE TOXICITY/CHRONIC HEPATIC ENCEPHALOPATHY
Subtle cognitive/motor impairment in patient on TPN, cirrhosis, PV thrombosis, occupational history of welding
Accumulation of neurotoxins (manganese) in brain tissue
MR:
bilateral, symmetric ↑T1 GP & substantia nigra >> pituitary and hypothalamus
Linear ↑T2/FLAIR in perirolandic cortex (laminar necrosis) and CSTs.
MRS: ↑glutamine:glutamate (ammonia excretion)

HYPERAMMONEMIC/ACUTE HEPATIC ENCEPHALOPATHY
NH4 > 60 mull/L, vomiting, seizure, coma.
MR: ↑T2 & diffusion restriction in bilateral insular and cingulate gyri, spares occipital and perirolandic areas. +/- posterior limb of IC, dorsal brainstem, and periventricular white matter involvement.
Imaging findings persist 3-6 months after therapy.
MRS: ↑myoinositil, ↑glutamine:glutamate (ammonia excretion), ↑Cho

HASHIMOTO ENCEPHALOPATHY
MR: ↑T2 patchy/confluent subcortical and periventricular WM with sparing of occipital lobes, medial temporal lobe edema. Enlarged pituitary.
Tc-99m HMPAO SPECT: ↑CBF 25%, reversible cerebral hypoperfusion/ reversible dementia

TRANSIENT GLOBAL AMNESIA
CT: normal
MRI: T1, T2, FLAIR normal.
Restricted diffusion dots in hippocampus that resolve in 10 days.

SUBACUTE COMBINED DEGENERATION/COBALAMIN DEFICIENCY
Loss of position and vibration sensation, hyperreflexia in a patient with megaloblastic anemia + Crohn, insufficient IF, alcoholism, veganism, H2-blockers, PPIs, metformin, methotrexate.
MR: ↑T2 signal in dorsal and lateral CSTs → inverted V in the spinal cord.
DDx:
Cu deficiency (may be caused by zinc, N2O toxicity),Tertiary Syphilis

DrCynCity

Most important general imaging patterns in toxic and metabolic brain disorders

- A: These include symmetric basal ganglia and/or thalami involvement (axial view)
- *B: S*ymmetric dentate nuclei involvement (axial view)
- *C: P*rominent cortical gray matter involvement (axial view)
- D: Symmetric periventricular white matter involvement (with gray matter sparing)
- *E: C*orticospinal tract involvement
- *F : C*orpus callosum involvement
- *G:* Asymmetric white matter involvement (demyelinating disease pattern)
- *H: P*arieto-occipital subcortical vasogenic edema
- *I:* Central pons involvement