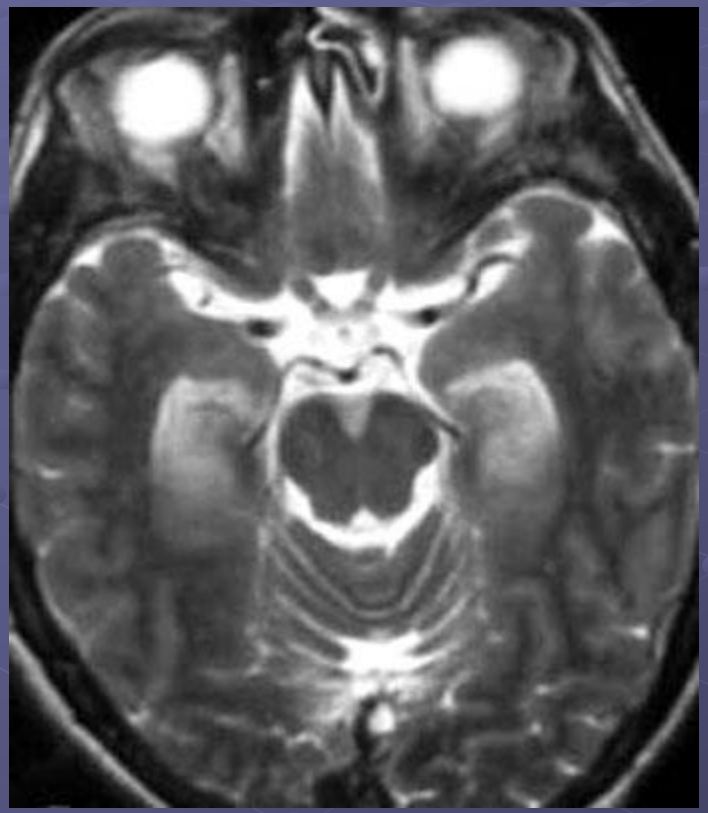
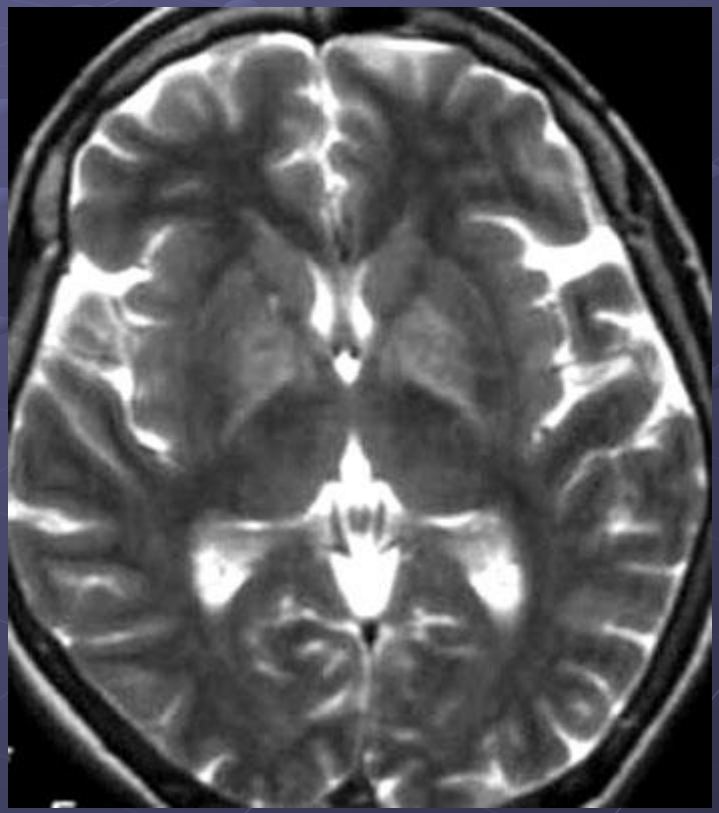
Carbon monoxide poisoning

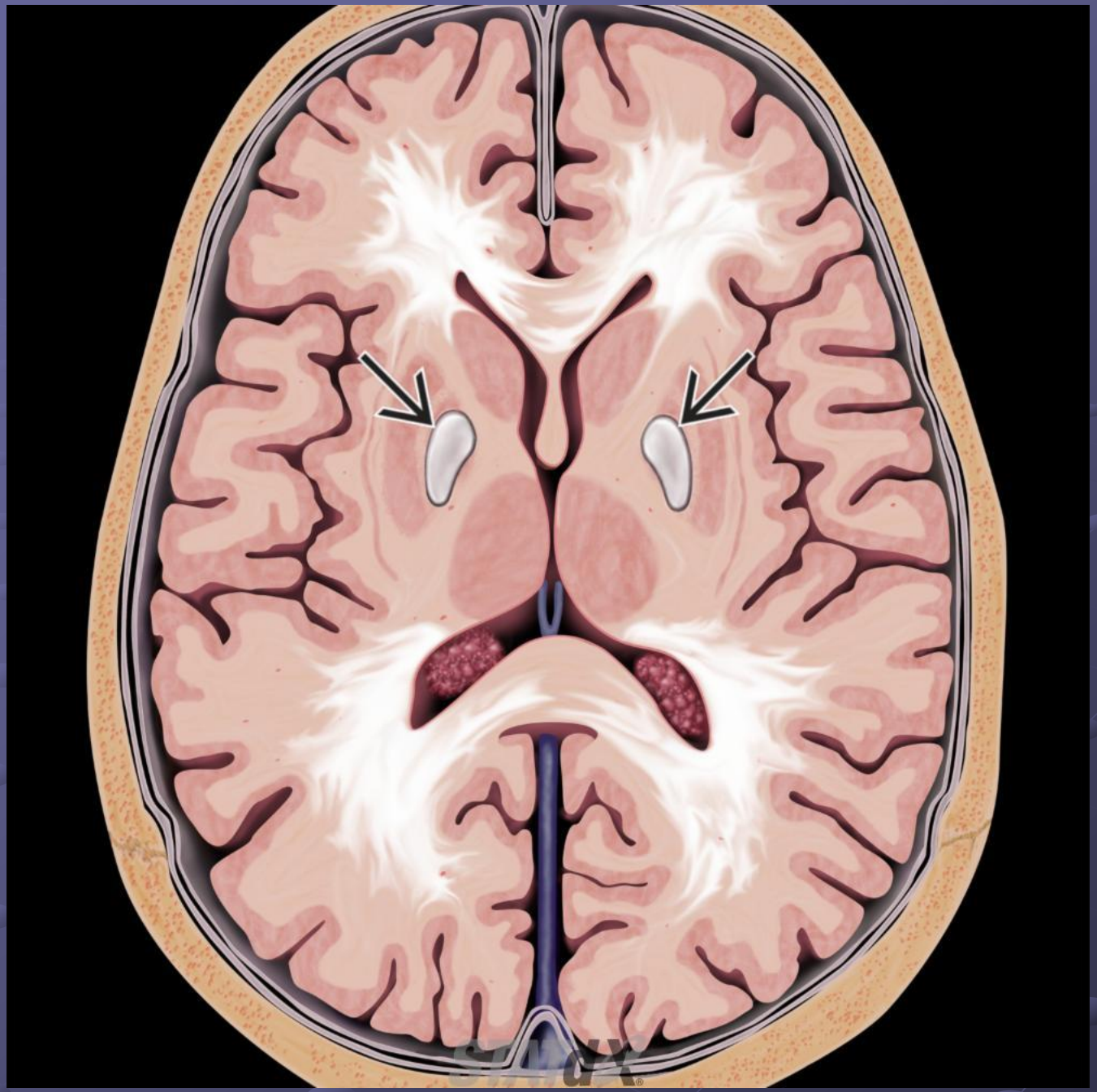




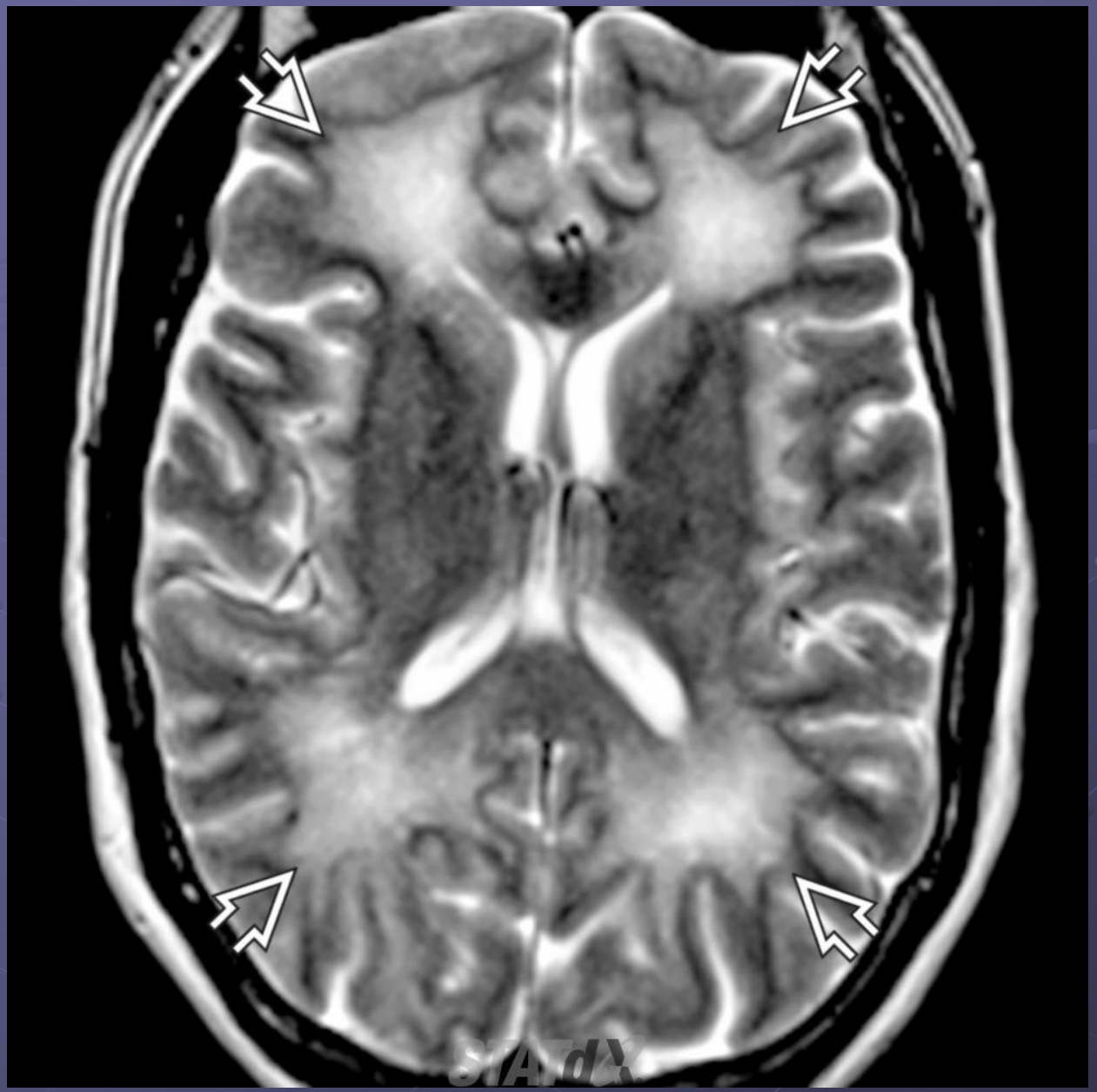
marked hyperintensity in the globus pallidus bilaterally.

DDX:

- Wilson Disease
  - WM/GM lesions, involving BG, dentate nucleus, pons, mesencephalon
  - T1 hypointense (occasionally hyperintense) lesions
  - Variably T2 hyperintense/hypointense
- Japanese Encephalitis (JE)
  - Homogeneous T2 hyperintensities in BG and thalami
  - Most characteristic finding in JE
  - Bilateral thalamic hyperintensities ± hemorrhage
  - JE is meningoencephalitis → meningeal enhancement
- Arteriolosclerosis
  - Focal hyperintensities in corona radiata, centrum semiovale
  - BG lacunae: Typically asymmetric, multifocal
- Creutzfeldt-Jakob Disease (CJD)
  - Progressively symmetric hyperintense changes in BG, thalami, cerebral cortex
  - DWI and FLAIR most sensitive
- Leigh Syndrome
  - Symmetrical spongiform brain lesions with onset in infancy/early childhood
  - Lesions predominantly in brainstem, BG (particularly putamen), and cerebral WM
  - Focal, bilateral, and symmetric T2 hyperintense lesions



Axial graphic shows the typical involvement of the brain by CO poisoning. The globi pallidi (GP) (black solid arrow) are most affected, followed by the cerebral white matter. Pathologically, there is necrosis of the GP with variable areas of necrosis and demyelination in the white matter.



Axial T2WI MR in the same patient shows bilateral diffuse hyperintensity throughout the white matter (white open arrow) with typical sparing of the subcortical U-fibers. The white matter hyperintensity is related primarily to demyelination with variable amounts of necrosis. The hyperintensity typically shows diffusion restriction.