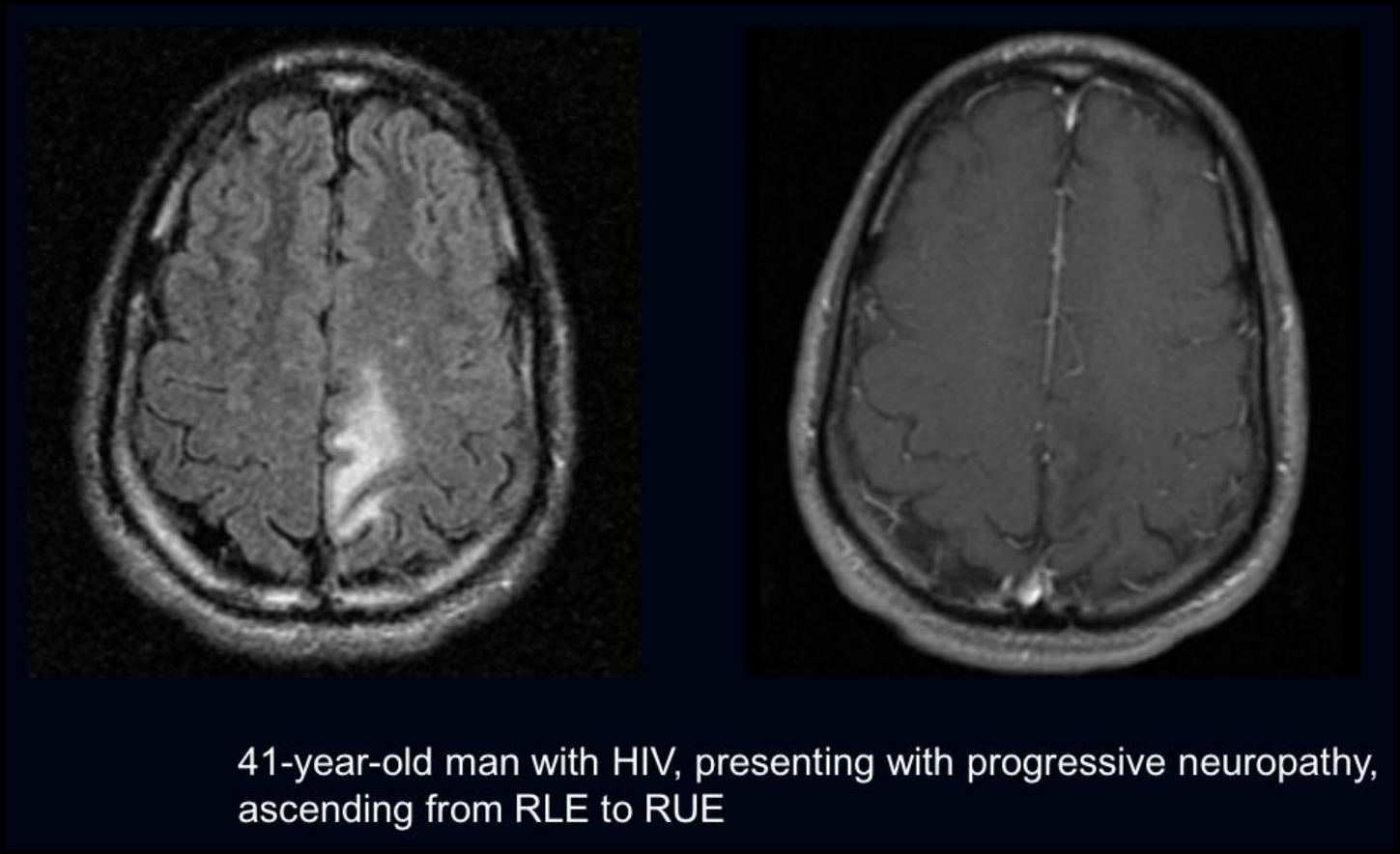
Progressive Multifocal Leukoencephalopathy

- JC polyomavirus infects oligodendrocytes, causes demyelination in immunocompromised patients.
- Associated with immunosuppression, often AIDS
- Organ transplant, cancer, chemotherapy, myeloproliferative disease, and steroid treatment.
- Recently reported in treatment for MULTIPLE sclerosis (MS) and in rheumatic diseases

PML

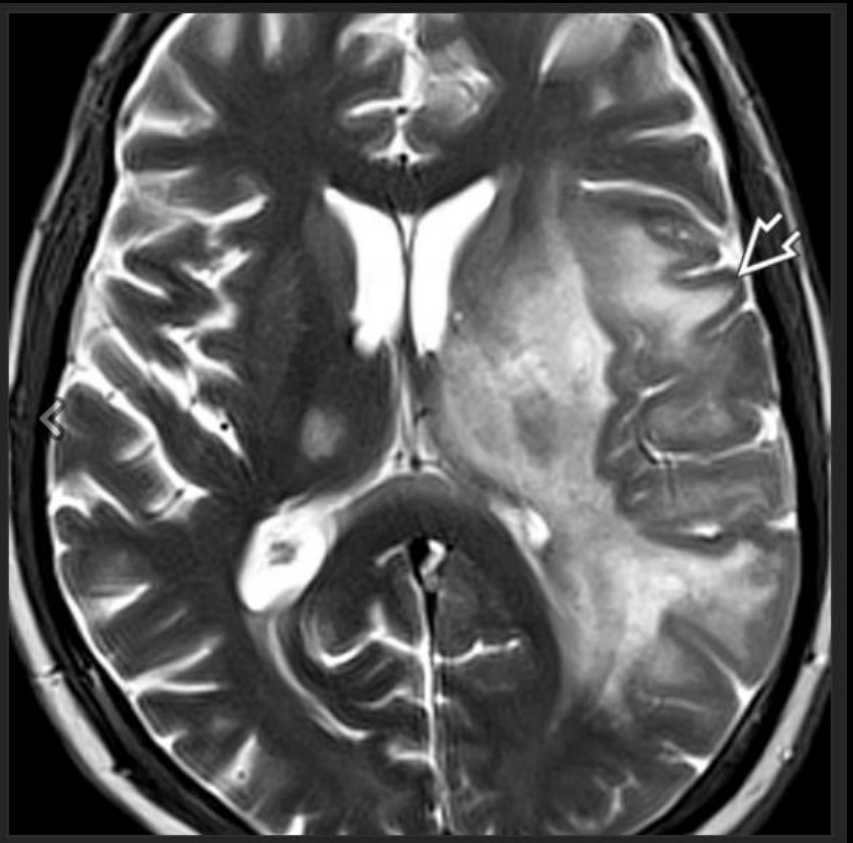
- Multifocal T2 hyperintense demyelinating plaques involve subcortical white matter (WM), extend to deep WM; gray matter often spared until late stage
- Characteristic involvement of subcortical U-fibers.
- May be solitary, multifocal, or widespread confluent.
- Usually no mass effect, no enhancement

PML



41-year-old man with HIV, presenting with progressive neuropathy, ascending from RLE to RUE

PML



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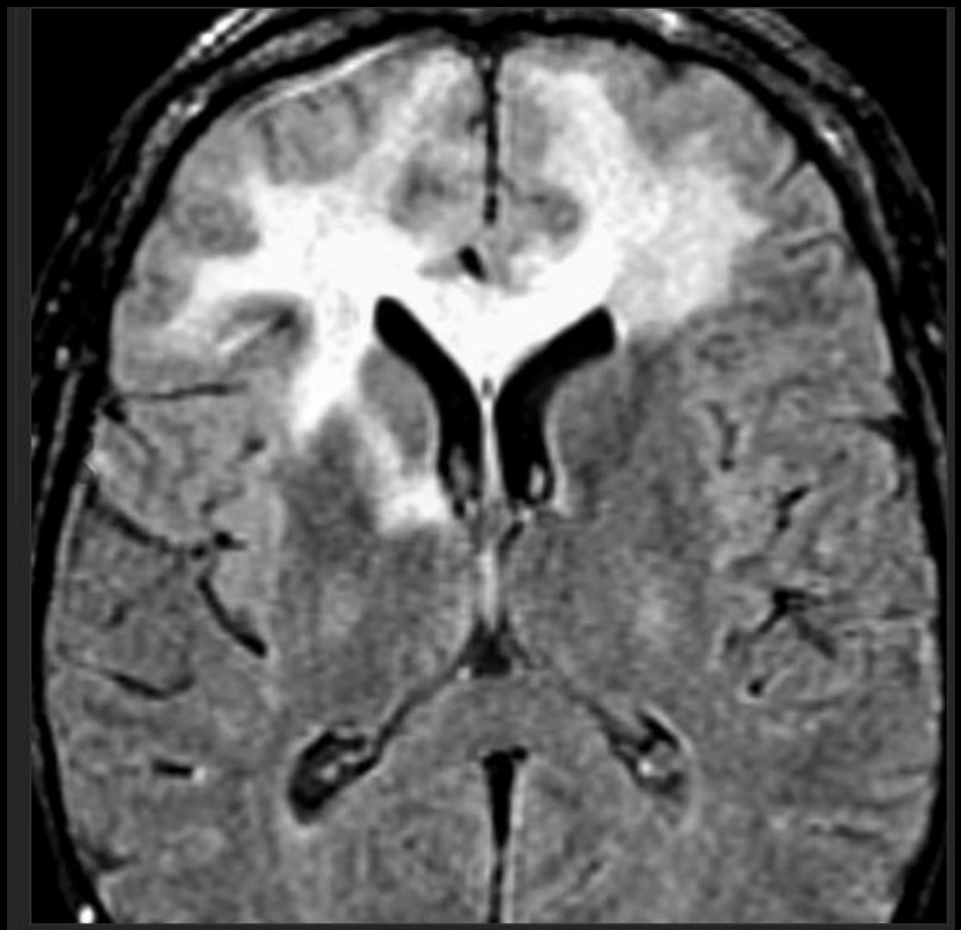
Axial T2WI MR reveals extensive confluent white matter hyperintensity involving the left cerebral hemisphere without significant mass effect. There is involvement of the subcortical U-fibers, left basal ganglia, and bilateral thalami. Note the classic scalloped appearance ⇨ at the gray-white interface. No enhancement is typical.



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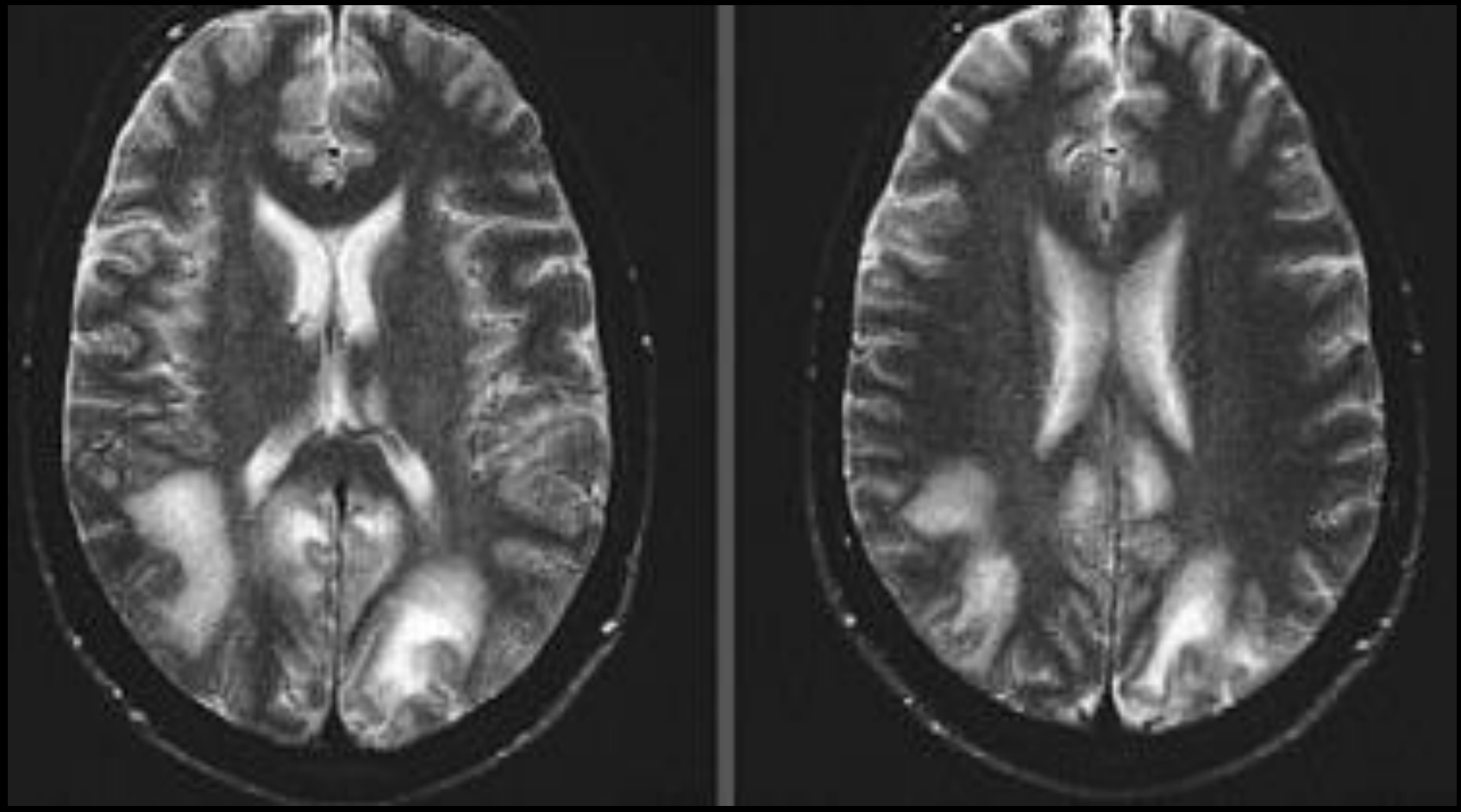
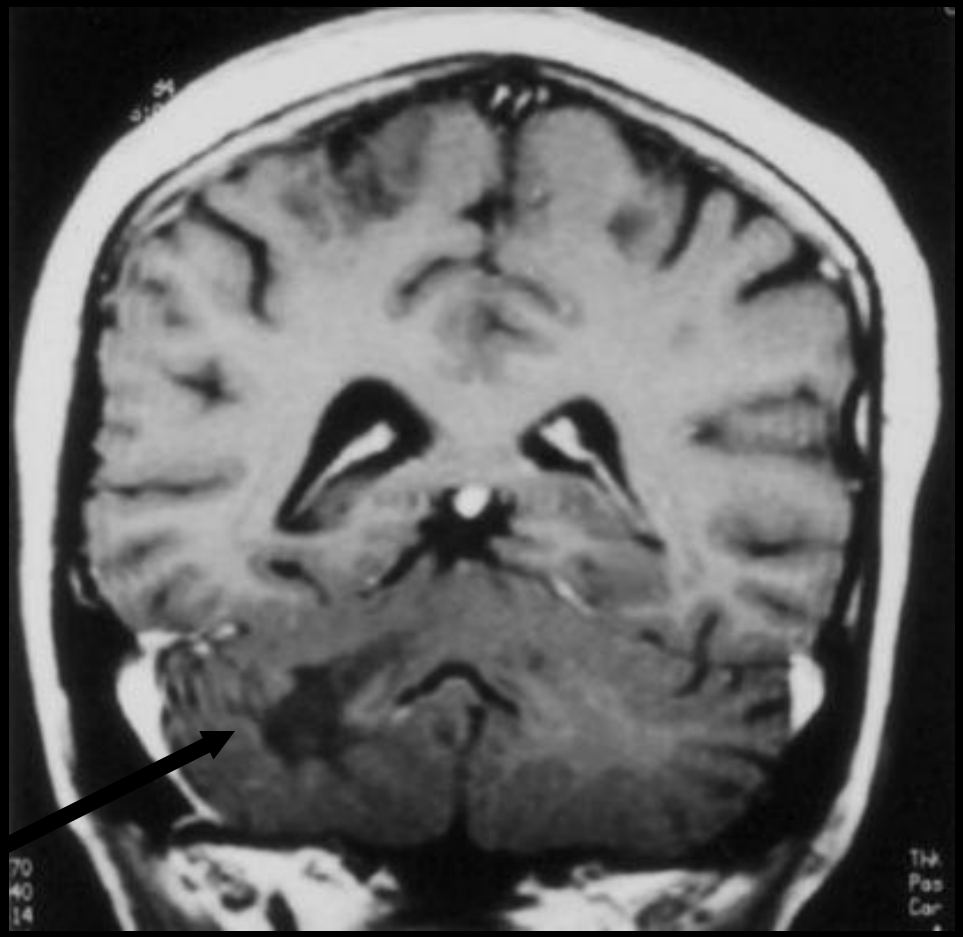
Axial FLAIR MR in an AIDS patient shows bilateral, asymmetric hyperintensity in the frontal lobe white matter with involvement of the subcortical U-fibers, characteristic of PML. Note the lack of mass effect and involvement of the corpus callosum.

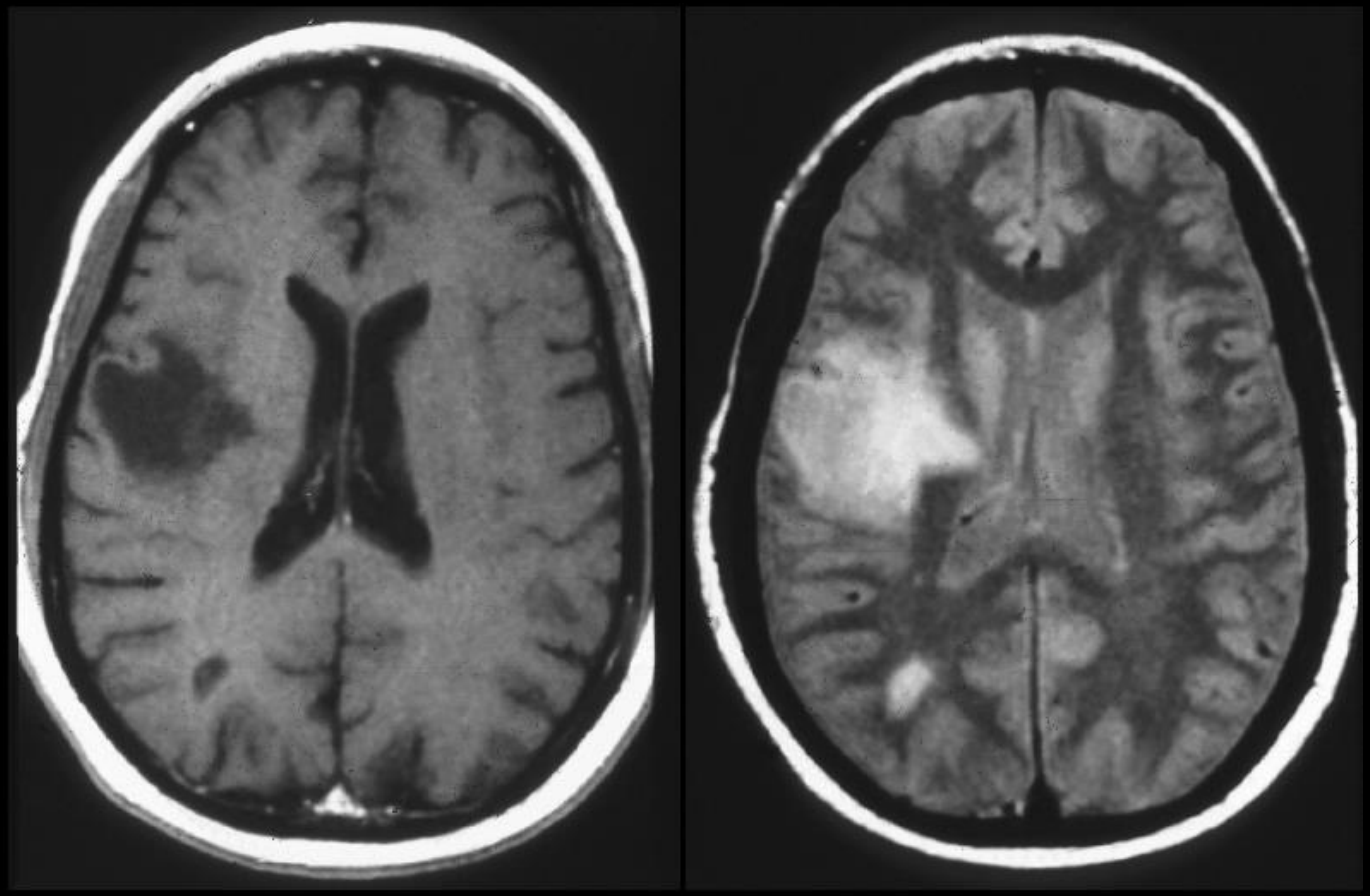
PML



PML



PML



HIV vs PML

- HIV
  - Normal on T1
  - Posterior fossa involvement uncommon
  - Subcortical U fibers uncommon
  - Symmetric
  - Never bleed, no enhancement
  - CMV encephalitis can look identical
- PML
  - Common abnormal on T1
  - Posterior fossa involvement common
  - Subcortical U fibers - common
  - Asymmetric (pariataloccipital)
  - Occasionally bleed and Occasionally enhance