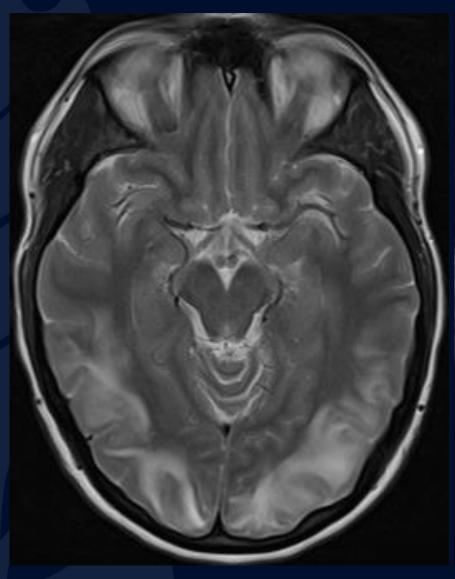
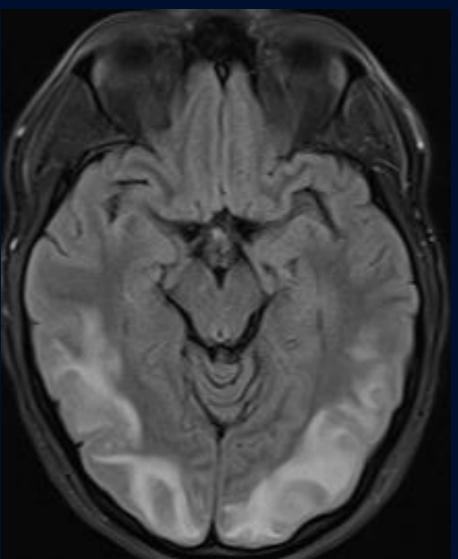
33 F, pregnant w/ seizure

Krithika Srikanthan, MD Leo Wolansky, MD



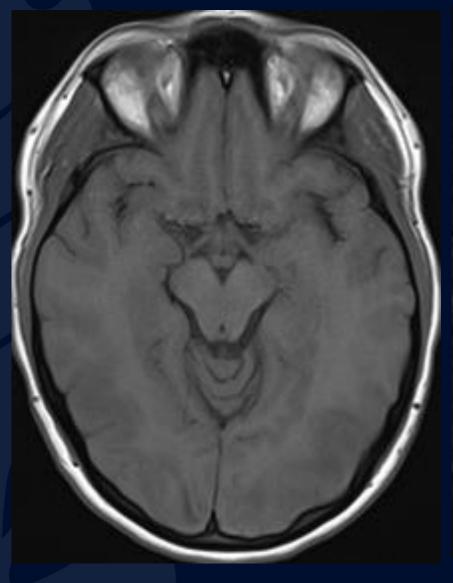


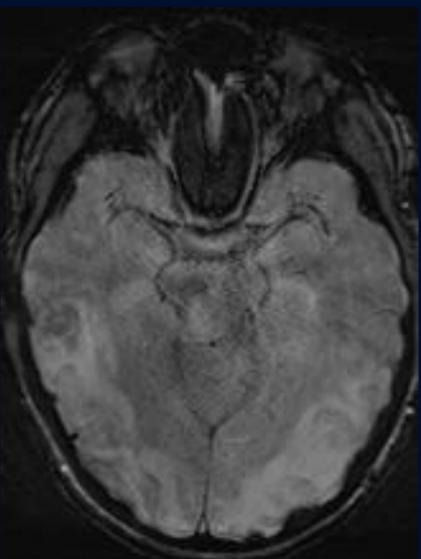


Axial T2

Axial T2 FLAIR



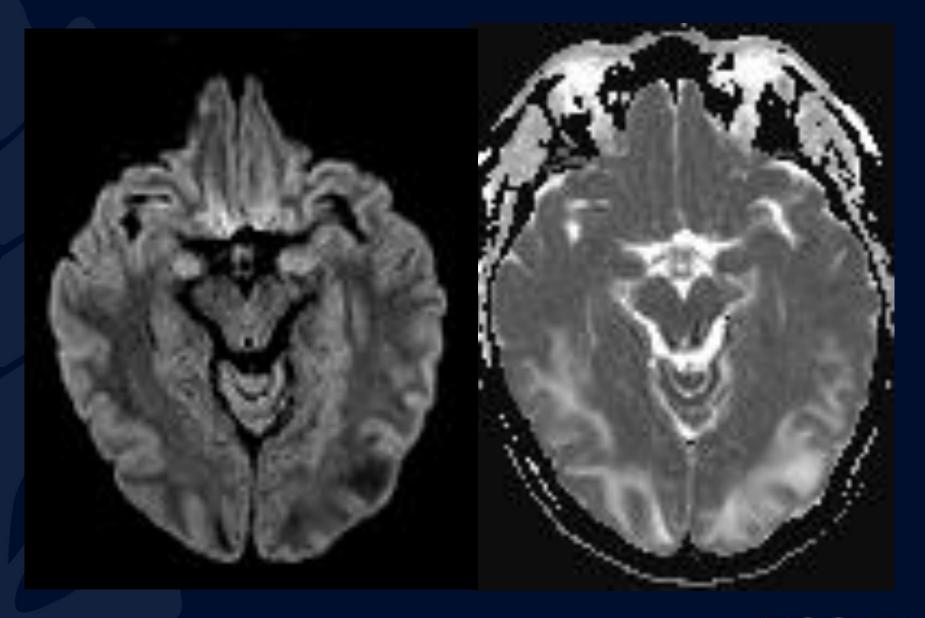




Axial T1

Axial SWI





Axial B1000

Axial ADC



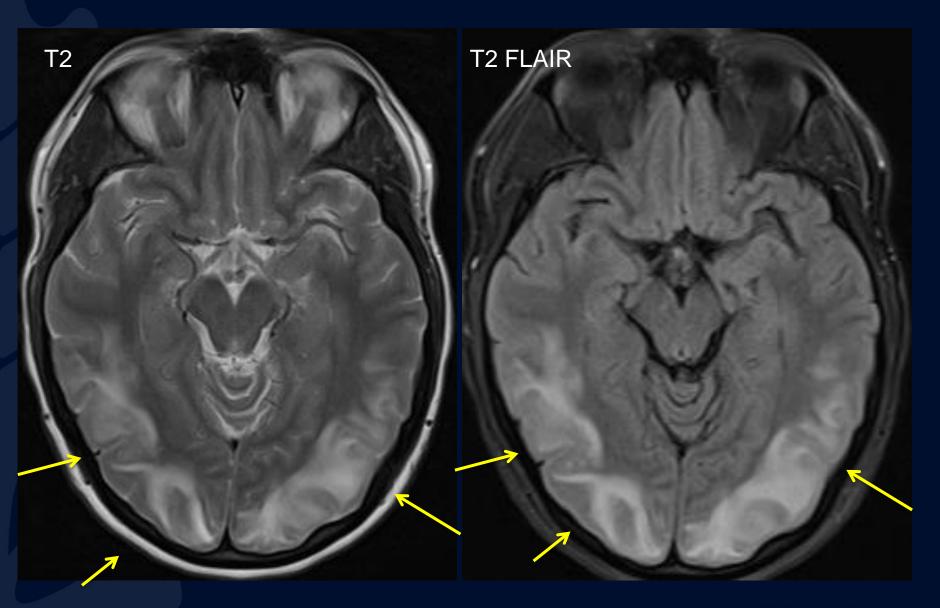




Posterior reversible encephalopathy syndrome (PRES)

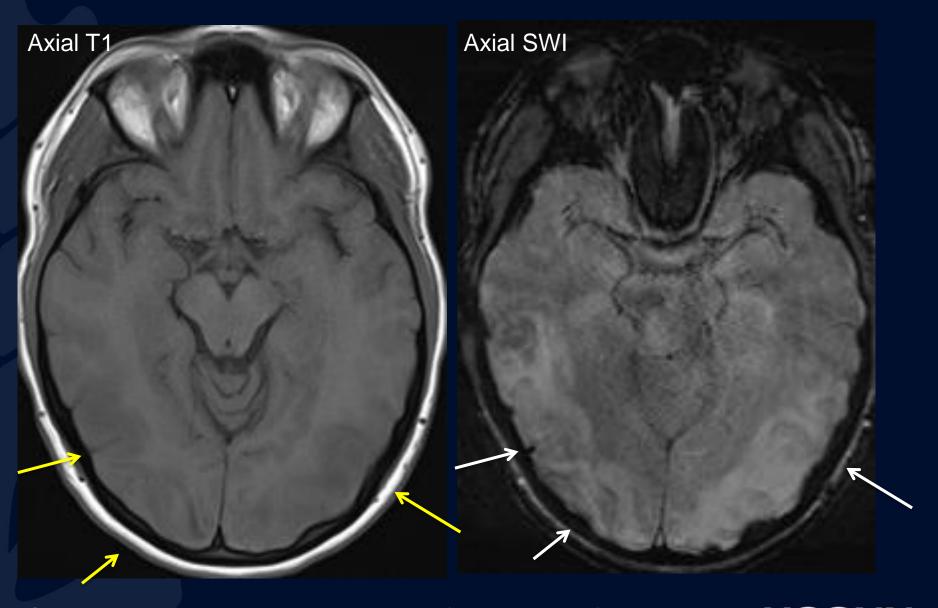
[aka Hypertensive encephalopathy]





Hyperintense signal suggesting vasogenic edema, predominantly in PCA territory (arrows)

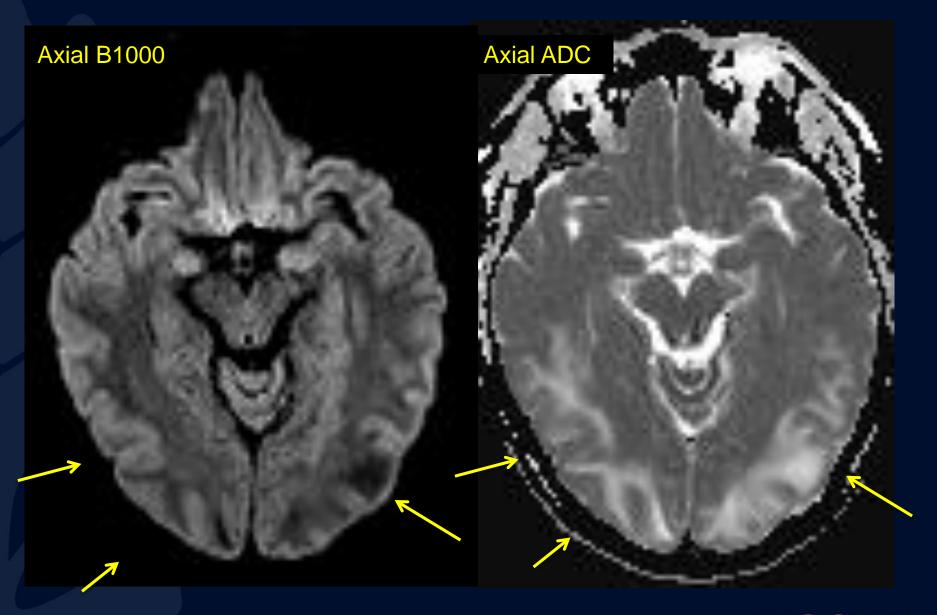




Same regions are difficult to see on T1 (yellow arrows).

SWI suggests edema and no gross hemorrhage (white arrows).





Hypointense signal on B1000 and marked lack of diffusion restriction on ADC is suggestive of vasogenic not cytotoxic edema (arrows)



MR Findings

- T1: Hypointense cortical/subcortical lesions
- T2: Hyperintense cortical/subcortical lesions
 - Typical PRES- parietooccipital
 - Atypical PRES frontal, basal ganglia
- FLAIR: Hyperintense cortical/subcortical lesions (cortical better seen on FLAIR)
 - Typical PRES: Parietooccipital cortical/subcortical lesions in 95%
 - Atypical PRES: Frontal lobe, basal ganglia
- DWI:
 - More common: Normal
 - Less common: Hyperintense on DWI with "pseudonormalized" ADC
- ADC: signal increased in affected regions due to increased diffusion
- T1 C+ (Gd): No enhancement OR patchy variable enhancement (~35% of patients, whether leptomeningeal or cortical pattern)
- GRE: may show hypointense signal in cases of hemorrhage
 - Focal parenchymal hemorrhage
 - Microhemorrhages
 - Convexity SAH
- SWI: may show microhemorrhages in up to 50%



CT Findings

- Bilateral nonconfluent hypodense foci
 - Posterior parietal, occipital lobes
 - Cortical watershed zones
- Most cases of PRES do NOT hemorrhage
 - If they do, multifocal small cortical petechialtype bleeds are typical
- CTA: Distal vessels may show diffuse vasoconstriction, focal irregularity, and beaded appearance, but major vessels usually normal



PRES

- Etiology:
 - Hypertension is the common denominator
 - Preeclampsia
 - Eclampsia
 - Drug toxicity (e.g., immunosuppressants, chemotherapy)
 - Uremic encephalopathy
 - Autoimmune diseases
 - Sepsis
- Physiology: Acute HTN damages vascular endothelium → failed autoregulation causes blood-brain barrier disruption → vasogenic (not cytotoxic) edema.
 - Posterior circulation more sensitive to effects of HTN, ICA sympathetic plexus
- Demographics: Young> Old, F>>M
- Classic presentation profiles:
 - Pregnant female with acute systemic HTN, headache ± seizure
 - Middle-aged, older adult on chemotherapy
 - Child with kidney disease or transplant
- Dx: headache, Sz (most common), encephalopathy (AMS), & visual disturbances
 + radiologic findings of focal reversible vasogenic edema
 - Some may be normotensive or have mildly elevated BP



PRES cont

- Location: Cortex, subcortical white matter; usually bilateral but asymmetric; at vascular watershed zone
 - Parietooccipital lobes (85-95 %)
 - Frontal lobes (75-77%)
 - Temporal lobes (65%),
 - Cerebellum (50-55%)
 - Basal ganglia (34%)
 - Brainstem (27%)
- Rx: Early recognition important for timely institution of therapy
 - gradual blood pressure control + withdrawal of offending agents (e.g. deliver child) + antiepileptics if seizing
 - Recommend repeating MRI after BP normalizes
- Prog: Usually complete neurologic & radiologic recovery in 2 weeks of treatment Usually no residual abnormalities after HTN corrected, BUT...
 - Delay in Dx/Rx can result in chronic neurologic sequelae
 - Secondary complications status epilepticus, intracranial hemorrhage, & massive ischemic infarction (rare), can cause substantial morbidity & mortality
 - 4% of patients develop recurrent PRES



References

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- 3. Statdx
- 4. https://www.ncbi.nlm.nih.gov/pmc/articles/P MC2861971/
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