42 y/o female who presented unresponsive following seizure

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Posterior Reversible Encephalopathy Syndrome (PRES)
Axial T2 & FLAIR images show hyperintensity within the cortical & subcortical white matter within the parietal lobes. Patchy areas of hyperintensity are also present within the frontal lobes (arrows).

Axial T2 & FLAIR images at the level of the ventricles show hyperintensity within the cortical & subcortical white matter of the occipital lobes.
PRES

Axial T2 and FLAIR images further down show hyperintense signal within the cortical and subcortical white matter of the temporal and occipital lobes.
PRES

Imaging Features

- T1 hypointense cortical/subcortical lesions
- T2 hyperintense cortical/subcortical lesions
  - Parietooccipital lobes, cortical watershed zones
  - May see frontal lobe and basal ganglia involvement
- Parietooccipital hyperintense cortical/subcortical lesions on FLAIR
- No restriction on DWI
- May see variable, patchy enhancement
PRES

General Features

• Thought that acute hypertension damages vascular endothelium
• Failed autoregulation damages BBB
• Results in vasogenic edema
  – Cerebral hyperperfusion causes dilatation and damage of arterioles resulting in hydrostatic leakage of interstitial fluid
• Posterior circulation has few sympathetic nerves resulting in less autoregulation & protection for parietal and occipital lobes
PRES

General Features

• Any age, but young more so than old
• F >> M
• Predisposing conditions with HTN as a common component: Preeclampsia > eclampsia, drug toxicity from chemotherapy, uremic encephalopathies (child with renal transplant or kidney disease)
PRES

DDx

• Cerebral Infarction
  – Will restrict on DWI
• Status Epilepticus
  – Transient gyral edema, but most often unilateral where PRES is usually bilateral
• Hypoglycemia
  – Severe parietooccipital edema resembling PRES, clinical history is the key differentiation
References
