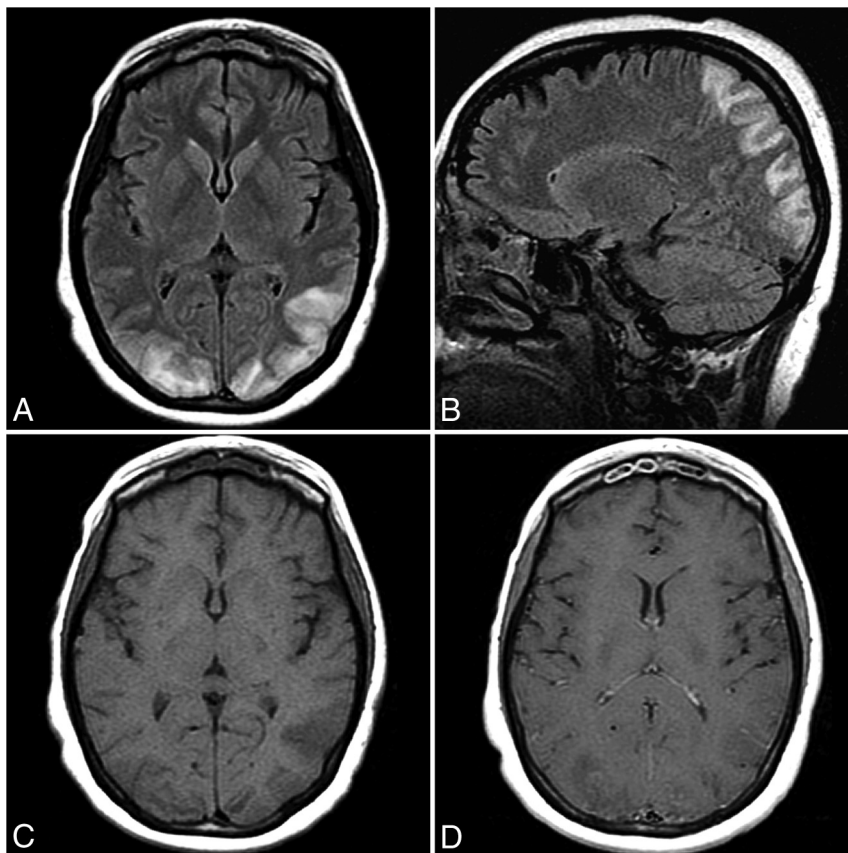


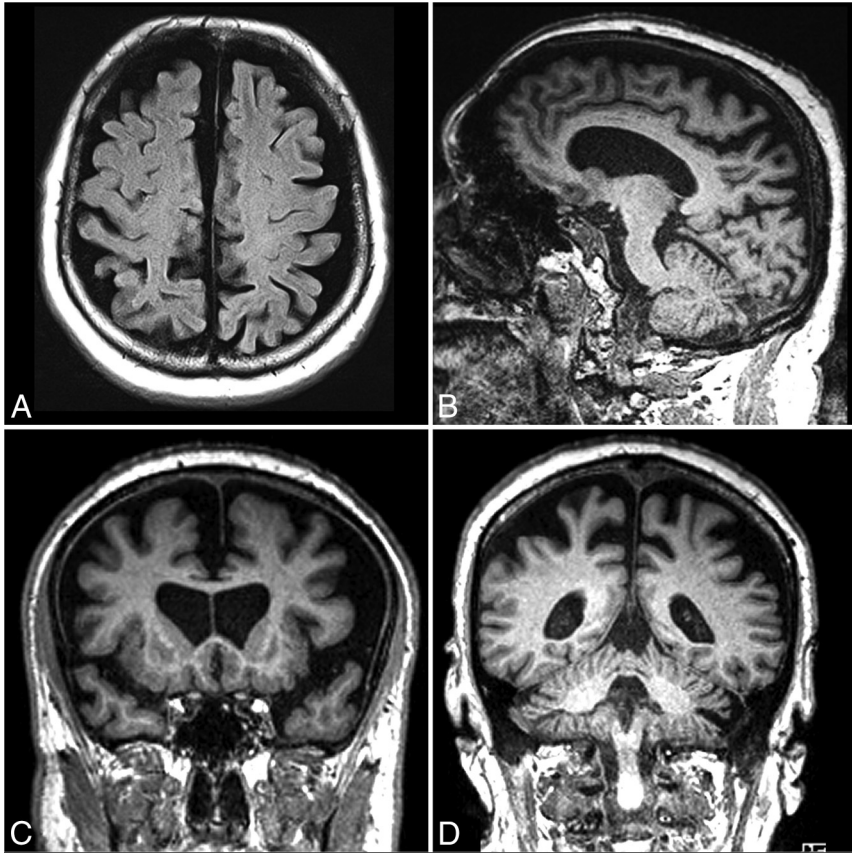
On-line Table: MRI imaging recommendation and summary of key features

Sequence	Pathologies Visible	Key Features
T1 volumetric high-resolution whole-brain reformatted in axial, coronal, and sagittal planes	Lewy body dementia	Less consistent pattern of cerebral volume loss; a pattern of relatively focused atrophy of the midbrain, hypothalamus, and substantia innominata, with a relative sparing of the hippocampus and temporoparietal cortex; relatively little cortical atrophy
	Posterior cortical atrophy Pituitary region	Bilateral parieto-occipital and temporo-occipital atrophy Pituitary macroadenoma: mass lesion intrinsic to pituitary >10 mm; T1 hypointense to gray matter (may be heterogeneous if hemorrhage present), T2 isointense, enhancing solid components; may extend into suprasellar region to distort optic chiasm; laterally may invade cavernous sinus
FLAIR, volumetric whole-brain	Focal cortical dysplasia Seizure (posterior cortical)	T2 hyperintense cortical lesions Blurring of gray-white matter junction Focal white matter abnormal signal Transmantle increased signal and abnormal gyral pattern Mesial temporal sclerosis, possibly others
	Primary brain tumors	Both low- and high-grade gliomas usually have associated FLAIR abnormality, involving cortex and white matter Enhancement, diffusion restriction, elevated cerebral blood volume in higher grade lesions
	Metastases	Location at gray-white matter junction Multiplicity Heterogeneous, depending on primary lesion, hemorrhage Enhancement, variable pattern Edema out of proportion to size of lesion
	PRES	Vasogenic edema with varying cortical and subcortical involvement Classically posterior but can also be in watershed distribution T2/FLAIR hyperintense Non-diffusion restricting Variable contrast enhancement
	RCVS	Initial imaging findings may be normal Convexity subarachnoid blood, lobar hemorrhage Cerebral edema (similar distribution to that in PRES)
	Migraine	T2/FLAIR punctate hyperintensities in deep white matter (often centrum semiovale, coronal radiata)
	SWI including phase and magnitude images	Cavernous venous malformations
DWI	CJD (and variant CJD)	Prominent susceptibility effect (blooming) Abnormality may be bilateral or unilateral, symmetric, or asymmetric Cortical diffusion restriction (most common early feature) T2 hyperintensity in putamen, caudate, and thalamus Hockey stick sign, hyperintense signal in pulvinar and dorsomedial thalamic nuclei vCJD

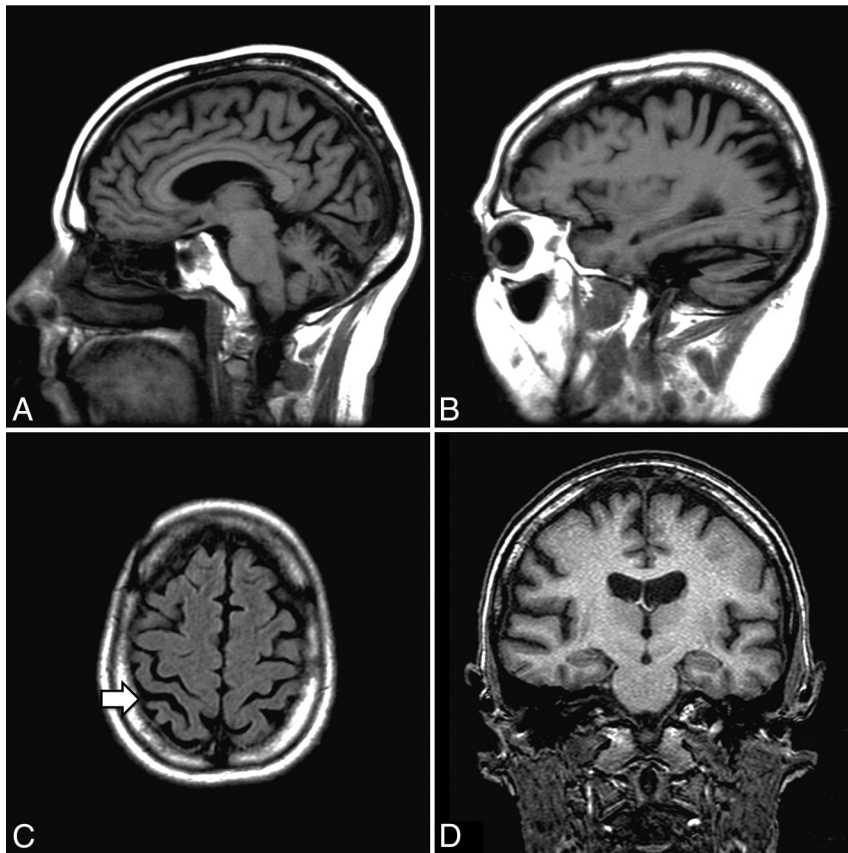
Note:—PRES indicates posterior reversible encephalopathy syndrome; RCVS, reversible cerebral vasoconstriction syndrome; vCJD, variant CJD.



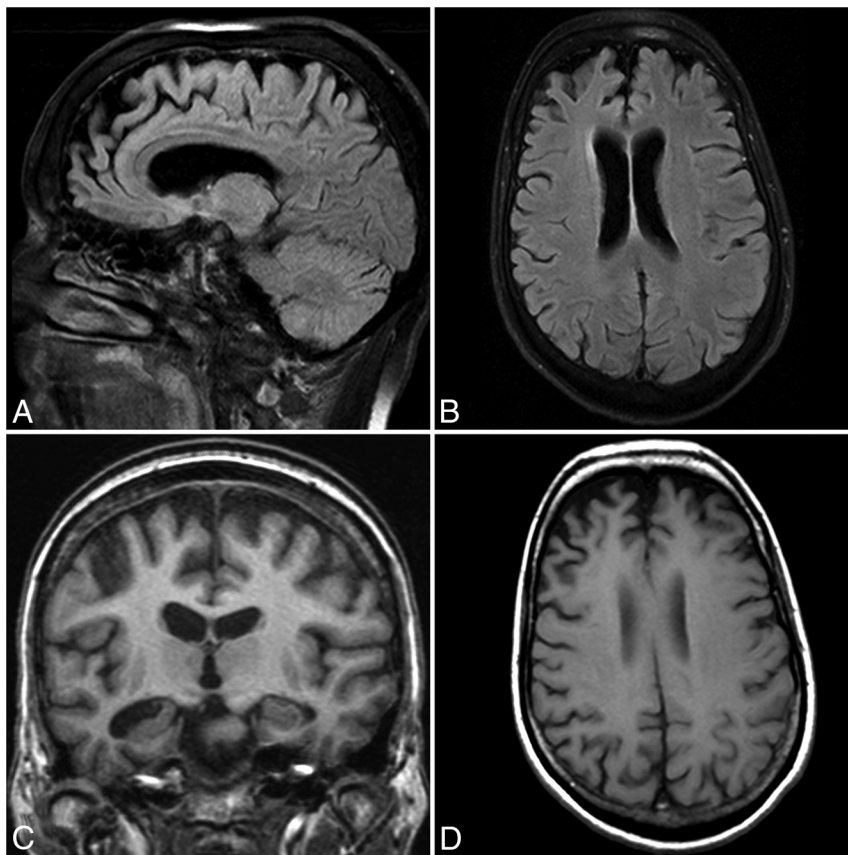
ON-LINE FIG 1. Posterior reversible encephalopathy syndrome: symmetric parieto-occipital vasogenic edema, with FLAIR (A and B), hyperintense T1 (C), and hypointense subcortical white matter abnormalities without contrast enhancement (D). Appearance is characteristic of posterior reversible encephalopathy syndrome.



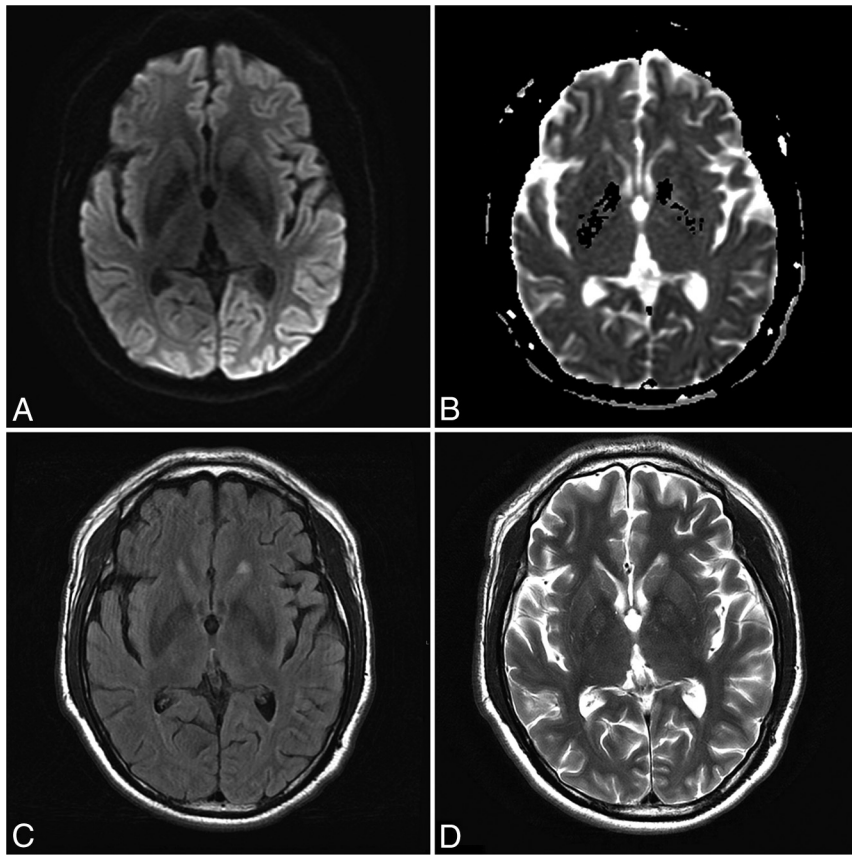
ON-LINE FIG 2. Lewy body dementia: multiplanar T1 imaging demonstrates global cerebral volume loss, most pronounced within the frontal and parietotemporal regions.



ON-LINE FIG 3. Posterior cortical atrophy: sagittal, axial, and coronal T1 MR imaging demonstrate prominent bilateral parieto-occipital (A and B) and temporo-occipital cortical atrophy (D), slightly more prominent within the right hemisphere (C). Features are those typical of posterior cortical atrophy.



ON-LINE FIG 4. Frontotemporal lobar degeneration: sagittal and axial FLAIR (A and B) and T1 MR imaging (C and D) demonstrate bilateral cerebral volume loss with a striking frontotemporal predominance, consistent with the diagnosis of frontotemporal dementia.



ON-LINE FIG 5. CJD: DWI (A) and ADC (B) sequences demonstrate cortical diffusion restriction within the frontal and occipital lobes bilaterally, with corresponding FLAIR (C) and T2 (D) hyperintensity, typical of Creutzfeldt Jakob disease. Cortical diffusion restriction is the most common early manifestation of CJD.