RESIDENT & FELLOW SECTION

Teaching NeuroImage: Cerebral Amyloid Angiopathy–Related Inflammation

An Interesting Evolution of Imaging Findings

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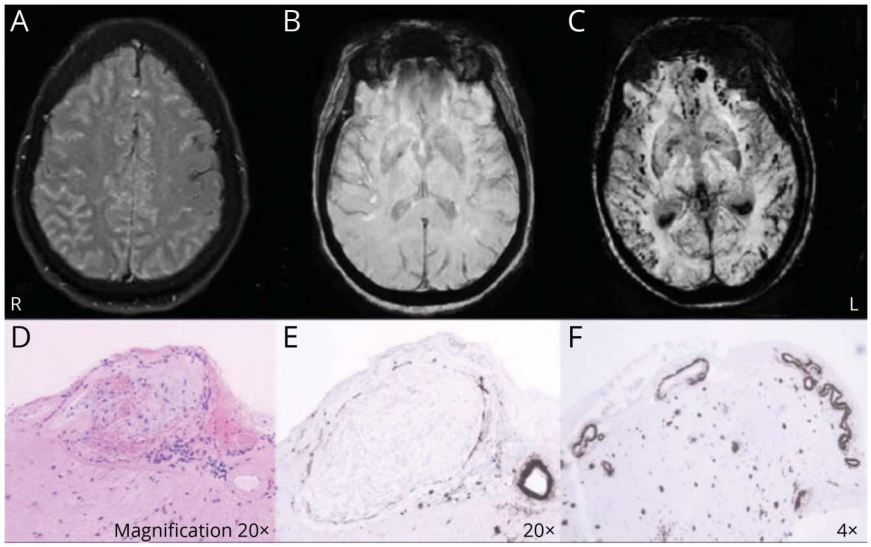
Figure MRI and Histology



(A) Fluid-attenuated inversion recovery sequence shows extensive sulcal hyperintensity. (B) Normal SWI MIP. (C) SWI MIP shows multiple peripheral microhaemorrhages. (D) Aneurysmal meningeal vessel, occluded by recanalising thrombus with extensive perivascular lymphocytic infiltration. (E) Parallel section of cortex with meninges, brown staining indicates accumulated beta-A4. (F) Meningeal and parenchymal vascular beta-A4 immunoreactivity. Plaques also evident. MIP = minimum intensity projection; SWI = susceptibility-weighted imaging.

A 77-year-old previously well woman presented with confusion, visual hallucinations, and signs of parietal lobe dysfunction, preceded by 5 days of headache. MRI of the brain demonstrated diffuse sulcal fluid-attenuated inversion recovery hyperintensities (Figure, A) and subtle leptomeningeal enhancement. Susceptibility-weighted imaging (SWI) was normal (Figure, B). Neurosurgical biopsy-confirmed cerebral amyloid angiopathy with perivascular inflammation (Figure, D–F). Pulsed methylprednisolone was given with excellent clinical response. MRI 7 weeks post symptom onset demonstrated widespread microhaemorrhages (Figure, C).

In the largest prospective study, all patients had abnormal SWI at presentation.[1] Normal SWI at presentation can rarely occur in CAA-ri; therefore, clinical suspicion is important.[2]

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