

Cerebral Amyloid Angiopathy Associated with Granulomatous Angiitis

A Case Report from Mayo Clinic, Rochester, USA

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Utility of Susceptibility-Weighted Imaging (SWI) in the Detection of Subcortical Hemorrhages in the Setting of Granulomatous Angiitis and Amyloid Angiopathy

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Clinical Presentation

A 70-year-old male presented with progressive cognitive decline to include difficulties with short term memory, reading, expressing himself, and doing arithmetic. Neurologic consultation recommended MR of the brain and psychometric testing. MR demonstrated localized vasogenic edema in the left parieto-occipital region without evidence of an underlying mass on the post-contrast T1 sequence (Figures 1 and 2). Diffusion imaging was negative. Susceptibility-weighted imaging demonstrated multiple subcortical deposits of hemosiderin throughout both cerebral hemispheres with localized concentration of these lesions in the area of edema. SWI showed better conspicuity and greater number of lesions than the corresponding gradient echo sequence (Figures 3 and 4). Review

of the spin-echo sequence suggested a diagnosis of an infiltrative primary glial neoplasm. The distribution of the hemosiderin deposits suggested a more diffuse microvascular process such as amyloid angiopathy with localized edema raising the possibility of granulomatous angiitis creating the appearance of a cerebral "mass."

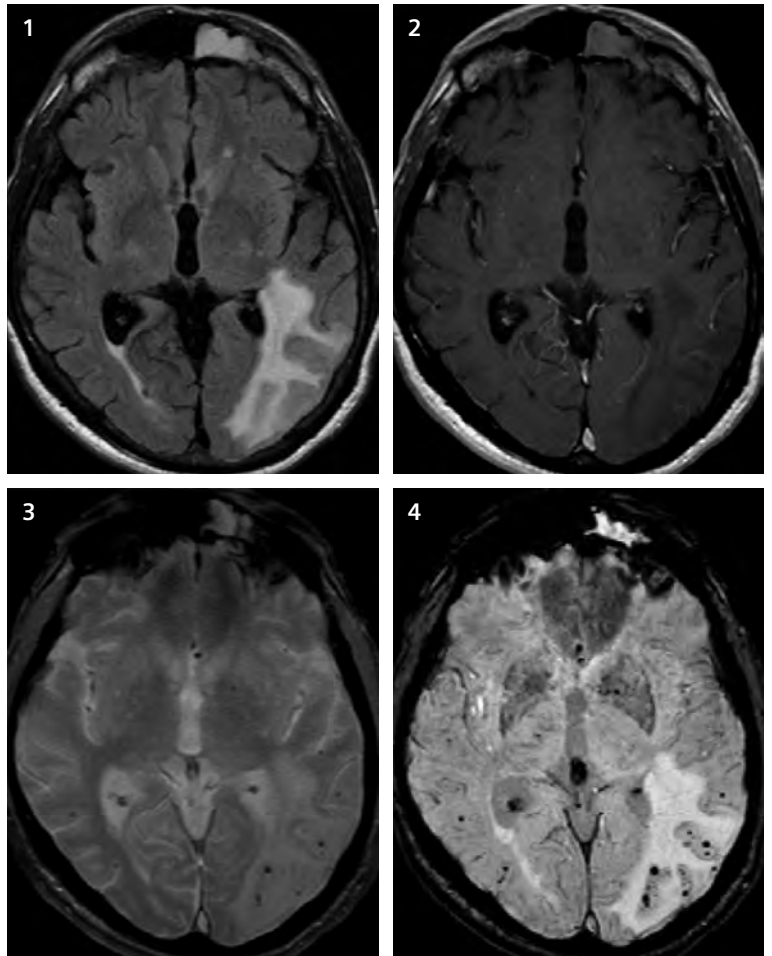
The patient underwent craniotomy and biopsy of the left parietal region to exclude underlying neoplasm. Pathology revealed amyloid-beta-related angiitis (ABRA).

Discussion

Patients with cerebral amyloid angiopathy associated with granulomatous angiitis usually present with progressive intracranial hypertension or dementia.

Imaging studies usually demonstrate focal or multifocal non-enhancing lesions or diffuse leukoencephalopathy.²

The term cerebral amyloid angiopathy (CAA) describes the localized deposition of amyloid in blood vessels within the cerebrum and overlying leptomeninges. CAA encompasses a heterogeneous group of hereditary and sporadic diseases, much the most common of which is sporadic CAA due to the vascular deposition of amyloid b peptide (Ab). Ab vascular deposition affects about 30% of the otherwise normal elderly and over 90% of those with Alzheimer's disease, in whom CAA tends also to be more severe. The most frequent clinical manifestation is lobar cerebral hemorrhage, which may be multifocal and recurrent, but CAA can also cause cerebral infarction and ischaemic leukoencephalopathy.



Imaging findings are characterized by diffuse white matter lesions, focal edema, mass effect, hemorrhage, infarcts and atrophy.

Detection of the hemosiderin deposits in this patient was critical to suggesting the diagnosis of granulomatous angiitis. Susceptibility-weighted imaging was superior to gradient echo imaging in detecting hemosiderin deposition.

SWI was critical to the appropriate interpretation of the MR exam.

References

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Figure 1: Flair sequence demonstrates localized pattern of vasogenic edema in the posterior temporal-occipital region of the left cerebral hemisphere.

Figure 2: T1 image with contrast demonstrates no evidence of an enhancing mass associated with the vasogenic edema.

Figure 3: Gradient echo image demonstrates punctate deposits of hemosiderin in the left parieto-occipital region.

Figure 4: Susceptibility-weighted image demonstrates increased conspicuity of the hemosiderin deposits throughout both cerebral hemispheres consistent with amyloid angiopathy. This finding, in conjunction with the focal edema, led to the suggestion of concomitant granulomatous angiitis.

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