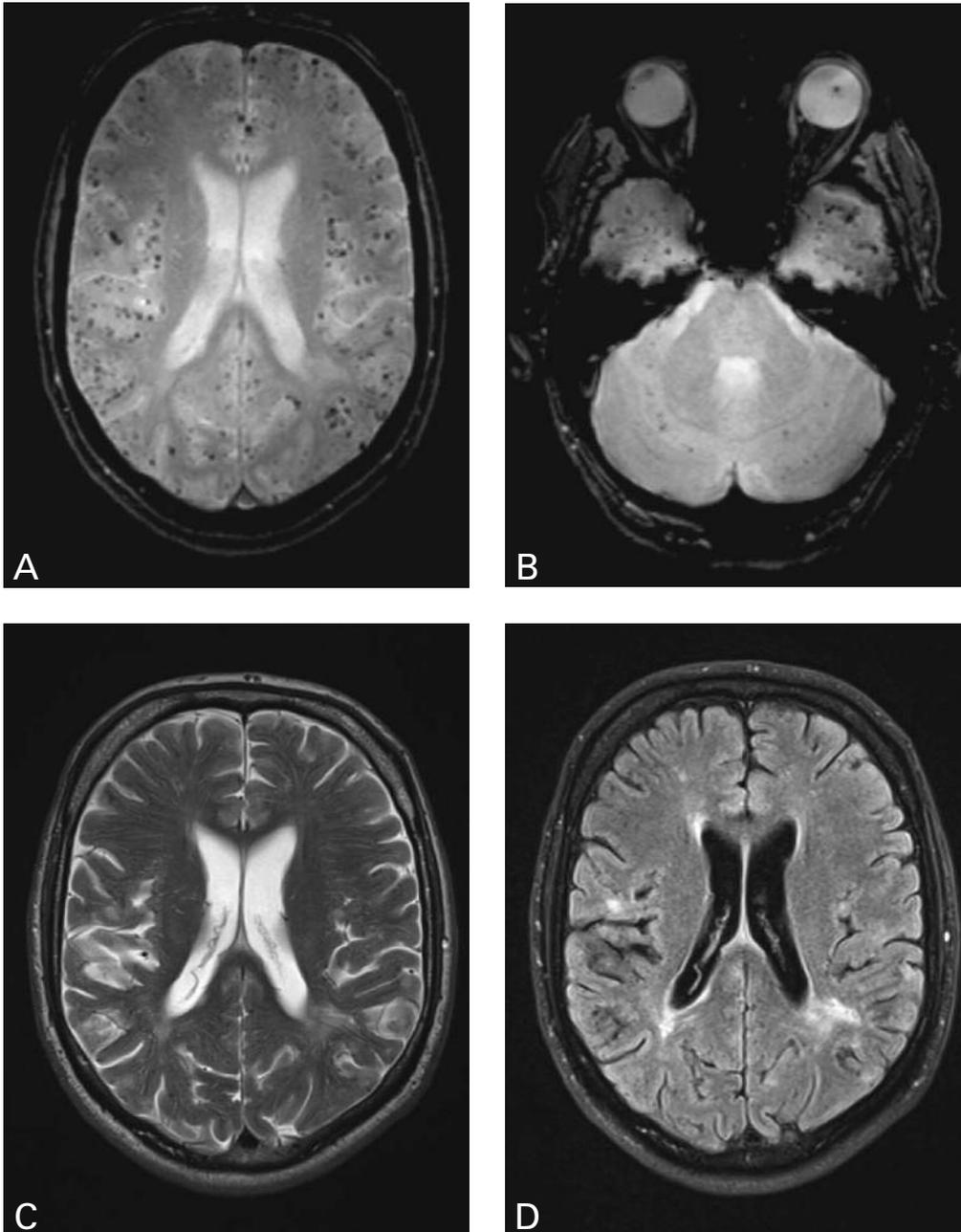


## PROBABLE CEREBRAL AMYLOID ANGIOPATHY

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**Key-word: Amyloidosis**

**Background:** A 55-year-old man presented with short-term memory loss, headache, diplopia and visual hallucinations. Higher mental functions were normal. The patient had a previous medical history of seizures, and was under longstanding anti-epileptic treatment. Recently he had been diagnosed with multiple myeloma.



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## Work-up

MRI of the brain, axial gradient-recalled echo FLASH T2\*-weighted images (Fig. 1) includes a section at the level of the lateral ventricles (A) and a section through the posterior fossa (B). Both images reveal multiple punctate hypointense lesions, both supra- and infratentorially, with sparing of the deep white matter and basal ganglia.

On axial fat-suppressed turbo spin-echo T2-weighted image at the same level as Fig. 1A (C) and axial fat-suppressed turbo FLAIR image at the same level as Fig. 1A (D), the numerous lesions which were easily recognizable on the gradient-recalled echo FLASH T2\*-weighted images, are not visible on the T2-weighted nor on the FLAIR images. Note some patchy white matter abnormalities, consistent with mild subcortical arteriosclerotic leukoencephalopathy.

## Radiological diagnosis

The presence of multiple hypointense foci on T2\*-weighted gradient-recalled echo images, indicates hemosiderin deposition in petechial brain hemorrhages. The age and clinical history of the patient, as well as the typical cortical and subcortical location of the microbleeds, meet the Boston criteria for a *probable diagnosis of cerebral amyloid angiopathy (CAA)*. However, cerebral microbleeds can also occur in other disease conditions.

## Discussion

Cerebral microhemorrhages appear as punctate or ovoid foci of marked signal intensity loss on T2\*-weighted gradient-recalled echo (GRE) MRI. Compared with turbo FLAIR and turbo spin-echo T2-weighted sequences, the T2\*-weighted GRE sequence has a markedly greater sensitivity for local magnetic field inhomogeneities produced by microscopic deposits of hemosiderin. Though microbleeds are in general clinically asymptomatic, they are recognized as a marker of small vessel pathology, most commonly cerebral amyloid angiopathy or hypertensive vasculopathy.

Cerebral amyloid (or 'conophilic') angiopathy is a condition characterized by the deposition of  $\beta$ amyloid in cortical, subcortical and leptomeningeal vessels, with sparing of the deep white matter, basal ganglia, and cerebellum. CAA is more frequently found in elderly patients, and is associated with Alzheimer's disease. CAA manifests radiologically as intracranial hemorrhages (ICH) in a distinctive cortical-subcortical distribu-

tion, leukoencephalopathy, and/or atrophy. In a normotensive elderly patient who presents with ICH without a history of trauma, CAA should always be considered. Although histopathological demonstration of vascular amyloid is still required for a solid diagnosis of CAA, histological analysis is often not practically feasible. Hence it is important to recognize the imaging findings of CAA.

A scoring system (the so-called Boston criteria) can be used to assess the likelihood of CAA on MR examinations of the brain.

Several other conditions must be considered in the differential diagnosis such as subcortical arteriosclerotic leukoencephalopathy, also known as Binswanger's disease or hypertensive cerebral angiopathy, is characterized by the development of intimal hyperplasia and hyalinosis in deep penetrating brain arterioles as the result of chronic systemic hypertension. Hypertensive bleeds, occurring predominantly in men between 60 to 80 years of age, are the underlying cause for 10% of all clinical strokes. Cerebral microhemorrhages associated with chronic hypertension are more commonly found in the thalamus, basal ganglia, cerebellum, and pons.

Another differential diagnosis is multifocal petechial cerebral hemorrhages that can also be found in diffuse axonal injury, also known as shearing injury. These lesions are commonly found in victims of high-speed motor vehicle accidents.

Their underlying mechanism is a combination of acceleration, deceleration, and rotation, affecting portions of the brain where tissue density differs: gray-white matter junctions, basal ganglia, splenium of the corpus callosum, and dorsal mid-brain.

Finally, other unusual causes of cerebral microbleeding include capillary teleangiectasias, vasculitis, micrometastases, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), and Parry-Romberg syndrome.

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