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Case Report

Early stage of cerebral amyloid angiopathy revealed by follow-up of a minimal head injury ☆

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ABSTRACT

Cerebral amyloid angiopathy (CAA) is an age-related cerebral microangiopathy characterized by the accumulation of *amyloid-beta* peptide in the wall of leptomeningeal arteries and cortical vessels. Diagnosis of sporadic amyloid angiopathy is most often made in elderly patient with lobar hematoma. We report a case of a 68-year-old female who had minimal head injury. Cerebral CT showed a right cerebellar hematoma. *Follow-up MRI* after 4 months showed signs of cerebral amyloid angiopathy. Through this observation, we describe the MRI semiology that helps make the diagnosis of cerebral amyloid angiopathy.

Introduction

Cerebral amyloid angiopathy (CAA) is a cerebrovascular disorder caused by the accumulation of cerebral amyloid-beta (Aβ) in the walls of leptomeningeal and cortical vessels. It leads to vascular fragility that causes intracerebral hemorrhage in normotensive patients [1]. CAA is found by imaging in up to 16% of asymptomatic elderly patients and up to 90% in autopsies of patients who have evidence of Alzheimer's disease (AD) [2]. These are 3 forms of cerebral amyloid angiopathy sporadic, familial, and iatrogenic. Diagnosis of sporadic amyloid angiopathy is most often made in the elderly patient with lobar hematoma, in the absence of another cause of cerebral hemorrhage.

Observation

It was a 68-year-old female, without hypertension and any other medical history, who had suddenly dizziness and fell with a head injury followed by loss of consciousness. Cerebral CT showed a right cerebellar hematoma and hemorrhage in the fourth ventricle (Fig. 1). Cerebral CT angiography did not find underlying vascular abnormalities such as aneurysm arteriovenous malformation or cerebral venous thrombosis.

After management in neurology, she presented good outcome. However, she had persistent gait disorders. This motivated an MRI after 4 months to search for an underlying lesion that could explain the symptoms. This MRI found a hematoma

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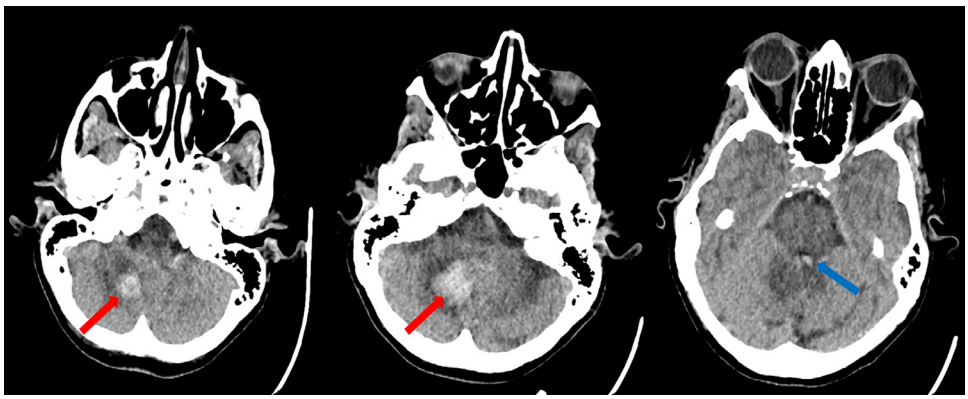

Fig. 1 – Axial CT scan without contrast showing a right cerebellar hematoma and minimal hemorrhage in the fourth ventricle.

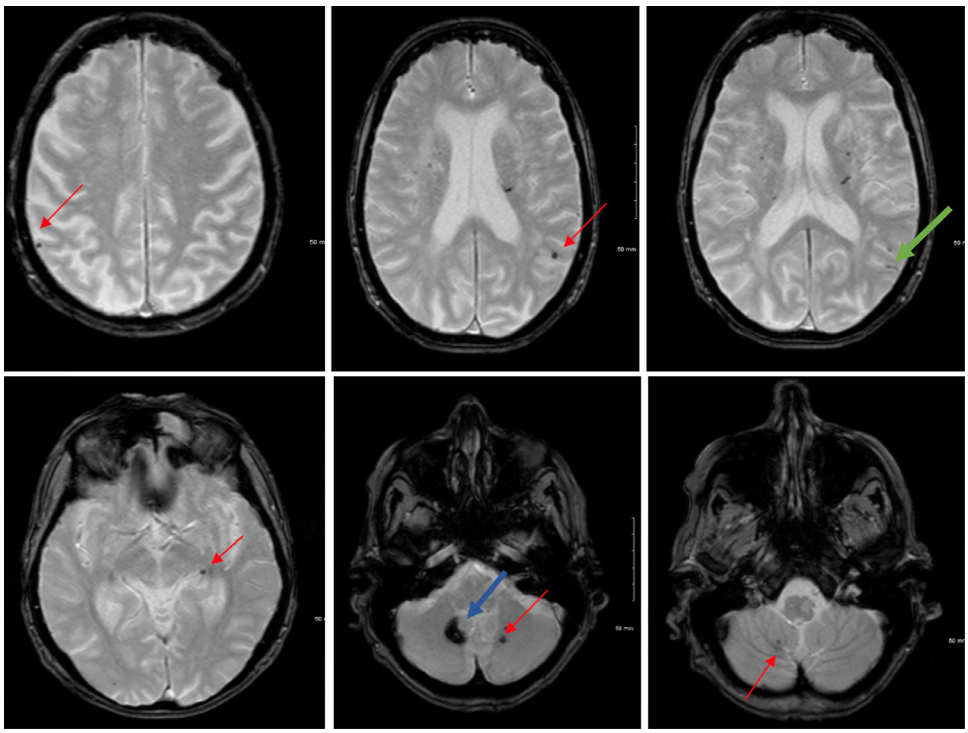

Fig. 2 – Brain MRI on T2* showing a hematoma next to the right cerebellar dentate nucleus (blue arrow) and subcortical microbleeds of both hemispheres and the posterior fossa (red arrows) and a discrete cortical hemosiderosis (green arrow).

next to the right dentate nucleus associated and subcortical microbleeds of both cerebral hemispheres and posterior fossa with minim cortical hemosiderosis (Fig. 2). Probable cerebral amyloid angiopathy in the early stage was retained according to the modified Boston criteria.

Discussion

CAA is a cerebral microangiopathy that can evolve alone or in association with neurodegenerative lesions such as AD. In autopsy series of AD, CAA is present in approximately 90% of cases whereas it is diagnosed in vivo in only approximately 20% of patients with AD. These 2 diseases are characterized by different Aβ peptide aggregates: Aβ1-42 in AD and Aβ1-40 in CAA [1].

The symptomatic population is represented by patients aged over 55, without vascular risk factors in half of the cases. Incidence of CAA also increases with age (10% at age 60 and 60% at age 80) [2]. CAA is a well-recognized factor of cognitive impairment. It is a common cause of dementia in the elderly. The risk of dementia would be 14% after 1 year and would rise to 73% after 5 years [3,4]. Transient focal neurological episodes or symptoms known as TFNE or TFNS (dysesthesia, motor symptoms, language disorders, visual disorders, migraine-like attacks) are reported in different studies. *These symptoms are short in duration, repeated, and often stereotyped* [5–7].

Definitive diagnosis of CAA requires postmortem examination of the brain. However, the modified Boston criteria allow a probability diagnosis based on neuroimaging. In this sense, magnetic resonance imaging (MRI) is the gold standard and the most important techniques used for diagnosis. CAA associates in various degrees, cortical or subcortical leptomeningeal micro-bleeding, multiple recurrent and nonsimultaneous lobar (or cerebellar) hematomas, and hemosiderosis or focal subarachnoid hemorrhage [8].

The presentation is dominated by lobar intracerebral hemorrhage. This hemorrhage affects the cortex and the juxtacortical white matter, with a predilection for the posterior regions of the brain (occipital and temporal lobe) [9]. In this case, it was a cerebellar hematoma that was not explained by the severity of the trauma. Atypical location of a hematoma should always motivate a fellow-up for underlying lesion.

Lobar microbleeds were the first to be identified as markers of CAA. The magnetic susceptibility sequence (SWI or T2*) is particularly useful for identifying microbleeds ("blooming artifact"). Microbleeds are deposits of hemosiderin secondary to bleeding from an arteriole. They are easily detected by the magnetic susceptibility effect [10].

Cortical hemosiderosis is a relatively specific marker of CAA within small cerebral vessel diseases. It corresponds to hemosiderin deposits in the superficial layers of the cerebral cortex. It is the chronic manifestation of repeated episodes of acute subarachnoid hemorrhage of the convexity [11]. Hemosiderosis appears on T2* and SWI MRI sequences as a hyposignal affecting the cortical surface of one or more gyri of the brain convexity [12].

Cortical subarachnoid hemorrhage can be isolated or associated with acute lobar intracerebral hemorrhage. It is a prognostic marker because it is linked to a high risk of recurrence [13,14].

The main differential diagnosis of CAA is hypertensive microangiopathy. It is due to different microvascular lesions causing vascular occlusions (lacunar infarctions) or vascular ruptures that are sources of microbleeding and cerebral hemorrhage. Hypertensive microangiopathy preferentially affects the deep regions (deep microbleeds) of the brain and the pons [9] *whereas CAA causes lobar microbleeding*.

Conclusion

Cerebral amyloid angiopathy is the main cause of lobar intracerebral hemorrhage in the elderly. Follow-up and etiological research for patients presenting lobar hematoma are essential. MRI remains the best imaging tools for diagnostic of probable CAA.

Patient consent

The patient has signed a free and informed consent to the anonymous publication of the material contained in this article.

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