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Letter to the Editor

Cerebral amyloid angiopathy-associated microbleed mimicking transient ischemic attack

A 72-year-old man diagnosed with probable Alzheimer's disease (AD) presented with acute onset motor aphasia. He has been treated for dyslipidemia with statins and he was on aspirin due to a transient ischemic attack (TIA) that presented with motor aphasia a year ago. At that time the patient had a brain MRI scan that showed no acute ischemic lesions but lobar microbleeds on the left hemisphere; the patient also had an extensive work-up with carotid duplex, transcranial Doppler, transthoracic and transesophageal echocardiography and 24-h Holter ECG that were normal. The patient had no clinical signs or laboratory tests suggestive of systemic vasculitis and, as part of his work-up for AD, he had recently had a lumbar puncture with normal cell count and protein. No intracranial hemorrhage was noted on brain CT-scan performed at 150 min from symptom onset but intravenous thrombolysis (IVT) was withheld due to rapidly improving neurological deficit (complete resolution of aphasia). Four hours after the initiation of symptoms the patient had fully recovered. Diffusion weighted MRI showed no acute ischemic lesion but gradient-echo imaging revealed a cortical left parietal microbleed (MB) (Fig. 1A) that was not present on neuroimaging study on the same MRI scanner 9 months ago (Fig. 1B). There was no deep MB present. T2-weighted imaging showed florid perivascular spaces (PVS) in the centrum semiovale (Fig. 1D). FLAIR imaging revealed posterior distribution of white matter lesions (WMLs) (Fig. 1D). Extensive work-up for ischemic stroke has been repeated with no abnormal findings. The patient fulfilled the Boston criteria for probable CAA as he presented multiple brain hemorrhages restricted to the cortex with no apparent cause of brain bleeding and was more than 55 years old. It should be noted that CSF amyloid-beta 42 was low (248 pg/mL, normal >350), tau was normal (326 pg/mL, normal <350) and phosphorylated tau was elevated (76 pg/mL, normal <61). This pattern is seen both in AD and cerebral amyloid angiopathy (CAA). These two conditions are usually overlapping: CAA is found in autopsy in more than 90% of AD patients [1]. Even if there is currently no means of distinguishing acute from chronic MBs and the new MB might have occurred any time during a 9-month period, its topography, the absence of concurrent ischemic lesion and122 the fact that the patient suffered from a probable CAA according to the Boston criteria led us to attribute the episode of transient neurological dysfunction to a CAA-associated MB.

Transient focal neurological episodes have been increasingly described mimicking TIA in CAA patients [1]. CAA-associated MBs may also present with symptoms of acute cerebral ischemia in AD patients and lead to inadvertent administration of IVT with potentially detrimental complications. AD and CAA are usually overlapping: CAA is found in autopsy in more than 90% of AD patients [1]. This is the first report of such an MB presenting as a TIA mimic, as previously reported for superficial cortical siderosis and convexity subarachnoid hemorrhage [1], as well as for hypertension-associated MBs [2,3]. Severe PVS has been long considered a normal variant, rarely associated with neurological deficits, but it has been recently recognized as a new imaging biomarker for CAA [4]. Posterior distribution of WMLs has been recently increasingly recognized in CAA patients [4].

This case report shows that brain CT scan cannot exclude CAA-associated MB in the acute phase; if this first report is further validated, patients with known possible or probable CAA, especially those suffering from AD, should probably not be thrombolysed for a possible ischemic stroke on plain CT scan but only after complementary imaging methods that either show a perfusion deficit (CT perfusion) or acute arterial pathology (transcranial Doppler or CT angiography) or after multimodal MRI proves ischemia as the cause of the neurological deficits. Secondly, as far as secondary prevention is concerned, there are no randomized controlled trials to guide the clinician as to the antithrombotic treatment of such patients. In patients with compelling indications aspirin therapy should probably be pursued. In our case we proposed the discontinuation of aspirin as it has been associated with increased intracranial hemorrhage risk in CAA and there has been a report that cessation of treatment with aspirin put an end to recurrent pseudo-TIAs in a patient with CAA [5].

Authorship contribution statement

Apostolos Safouris: study design, data collection, drafting and revising the manuscript.

Marie-Dominique Gazagnes: critical comments during manuscript revision.

Nikos Triantafyllou: data collection and critical comments during manuscript revision.

Georgios Tsivgoulis: study design, drafting and revising the manuscript.

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Conflict of interest statement

The authors report no disclosures.

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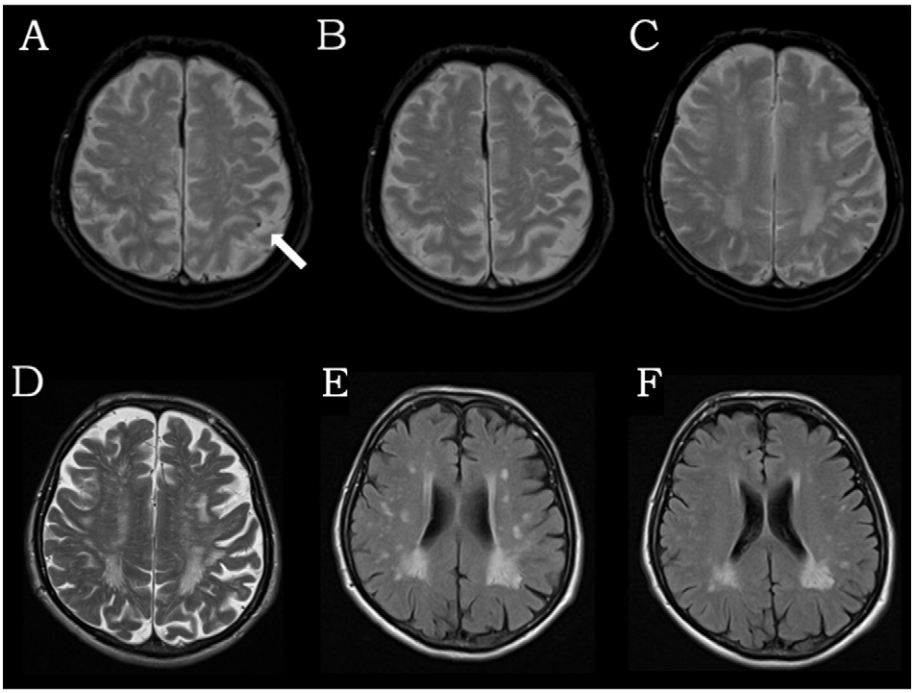

Fig. 1. Gradient-echo axial brain-MRI performed during the first 48 h following symptom onset showing a left parietal microbleed (arrow, panel A) that was not present in previous MRI performed 9 months ago (panel B). Note the presence of older multiple cortical microbleeds with "blurred" appearance on gradient-echo MRI (panel C). Axial T2-weighted imaging shows florid perivascular spaces in the centrum semiovale (panel D). Posterior distribution of white matter lesions (WMLs) on axial FLAIR MRI (panel E). This characteristic distribution of WMLs has been already present 4 years before admission on a brain MRI that has been ordered after an episode of vertigo (panel F).

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