CEREBRAL AMYLOID ANGIOPATHY

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ABSTRACT

Cerebral amyloid angiopathy (CAA) is characterized by the deposition of congophilic material in small to medium-sized blood vessels of the brain and leptomeninges. In its most severe stages, the amyloid deposits cause breakdown of the blood vessel wall with resultant hemorrhage. We present the case of a 70 year old patient who suffered a spontaneous intracranial hemorrage with a probable diagnosis of CAA.

Key words: Cerebral amyloid angiopathy, spontaneous intracranial hemorrhage

BACKGROUND

Cerebral amyloid angiopathy (CAA), although usually asymptomatic, is an important cause of primary lobar intracerebral hemorrhage in the elderly. It can occur as a sporadic disorder, in association with Alzheimer disease (AD), or with certain familial syndromes. CAA is characterized by the deposition of congophilic material in small to medium-sized blood vessels of the brain and leptomeninges. In its most severe stages, the amyloid deposits cause breakdown of the blood vessel wall with resultant hemorrhage. The incidence of cerebral amyloid angiopathy (CAA), is age-dependent and varies according to different autors from 15% for patients under 75 years up to 50% over the age of 75 years. CAA is estimated to account for up to 15% of all ICH in patients older than 60 years and up to one half of nontraumatic lobar ICH in patients older than 70 years.Hypertension seems to exacerbate the deposition of amyloid and increases the severity of CAA. Cognitive impairment is a common feature of CAA. More than 40% of patients with ICH-related hemorrhage have some degree of dementia and CAA.CAA is present in 80-85% of patients with AD, and is severe in one-third to two-thirds of these patients. The Boston Cerebral Amyloid Angiopathy Group has elaborated guidelines for the diagnosis of CAA associated with ICH. Four levels of certainty in the diagnosis of CAA are considered: definite, probable with supporting pathological evidence, probable, and possible. The first 3 require that no other cause of hemorrhage has been identified.

CASE STUDY

We present the case of an 70 years old male who presented to our clinic with acute binocular visual loss and somnolence. His past medical history is notable for cigarette smoking and alchool consumptions in the past, hipertension with moderate values of the systolic blood pressure. He also reported that some years ago he had some neurological problems consisting of seizures and after that he experienced vissual loss in the left visual field witch were never investigated. The familly also noticed some cognitive deterioration and depression.On admision he was slighty somnolent, desoriented, he had decresed visual acuity in both eyes, the pupilary motor reflex was preserved but slowed, the consensual pupilary reflex was preserved, the osteotendinous reflexes were present simetrical and there were no abnormal reflexes. He also had some degree of cataract in both eyes.We tried to test the visual field of the patient using kinetic perimetry(Goldman) but because of the cognitive deterioration the patient couldn't maintain attention and respond appropri-

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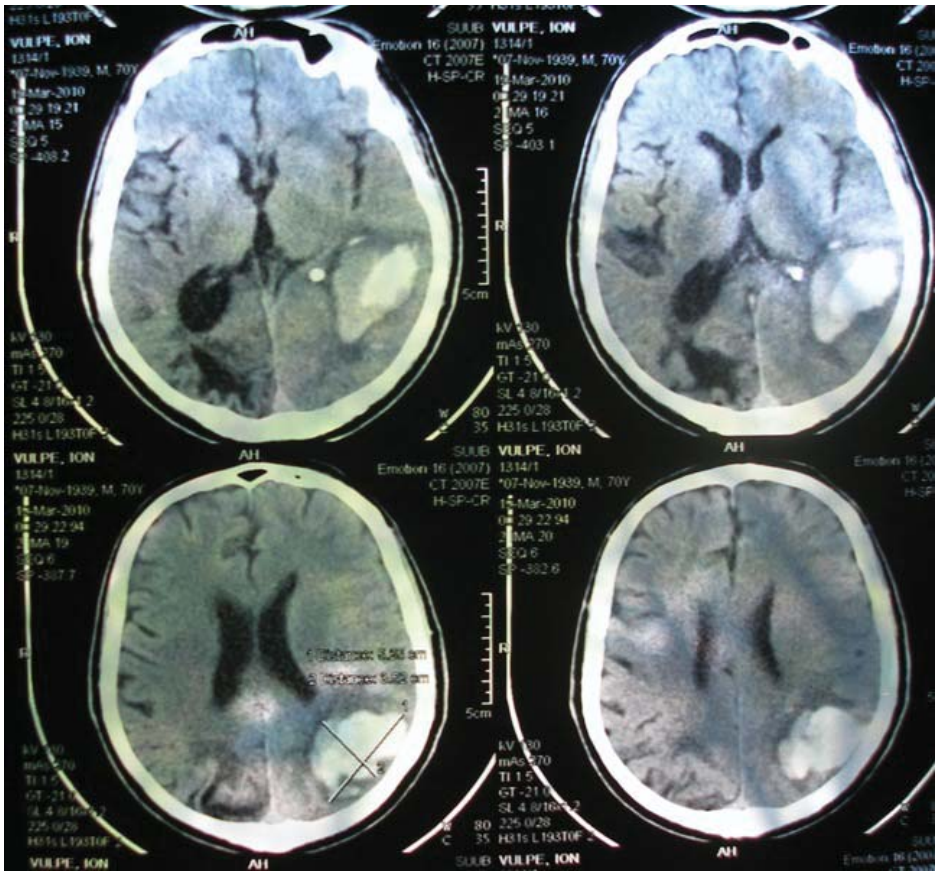
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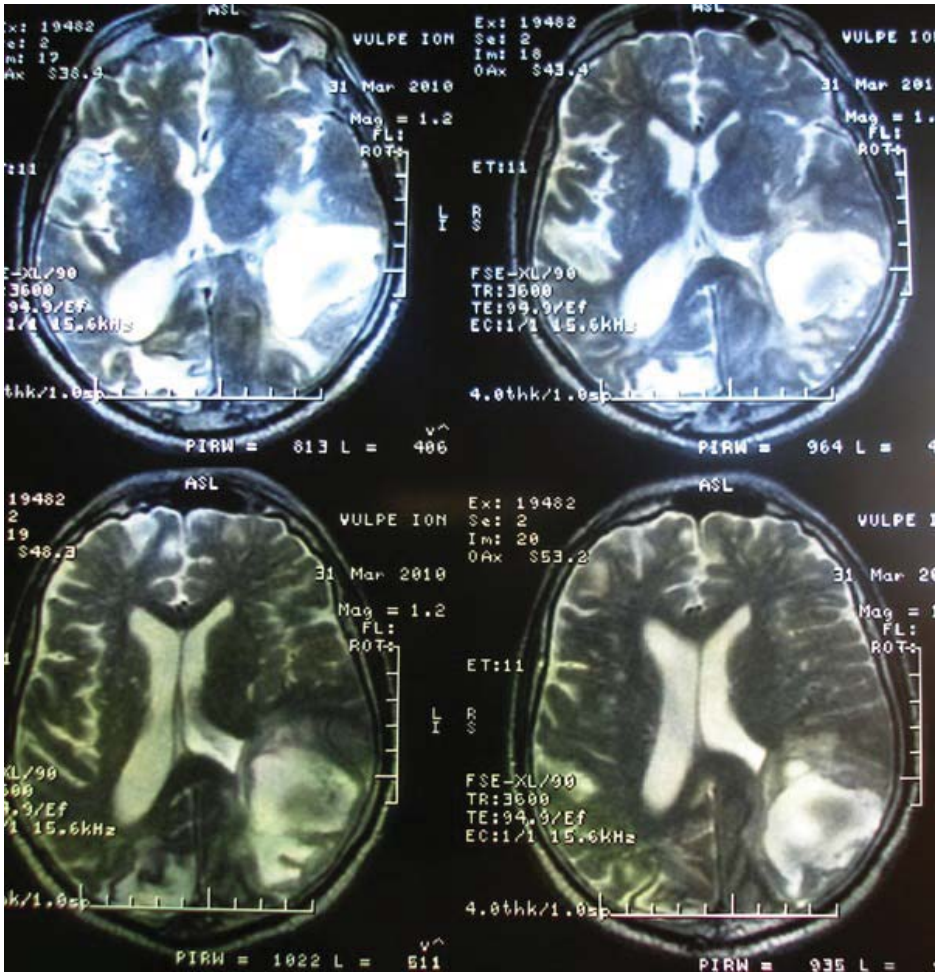
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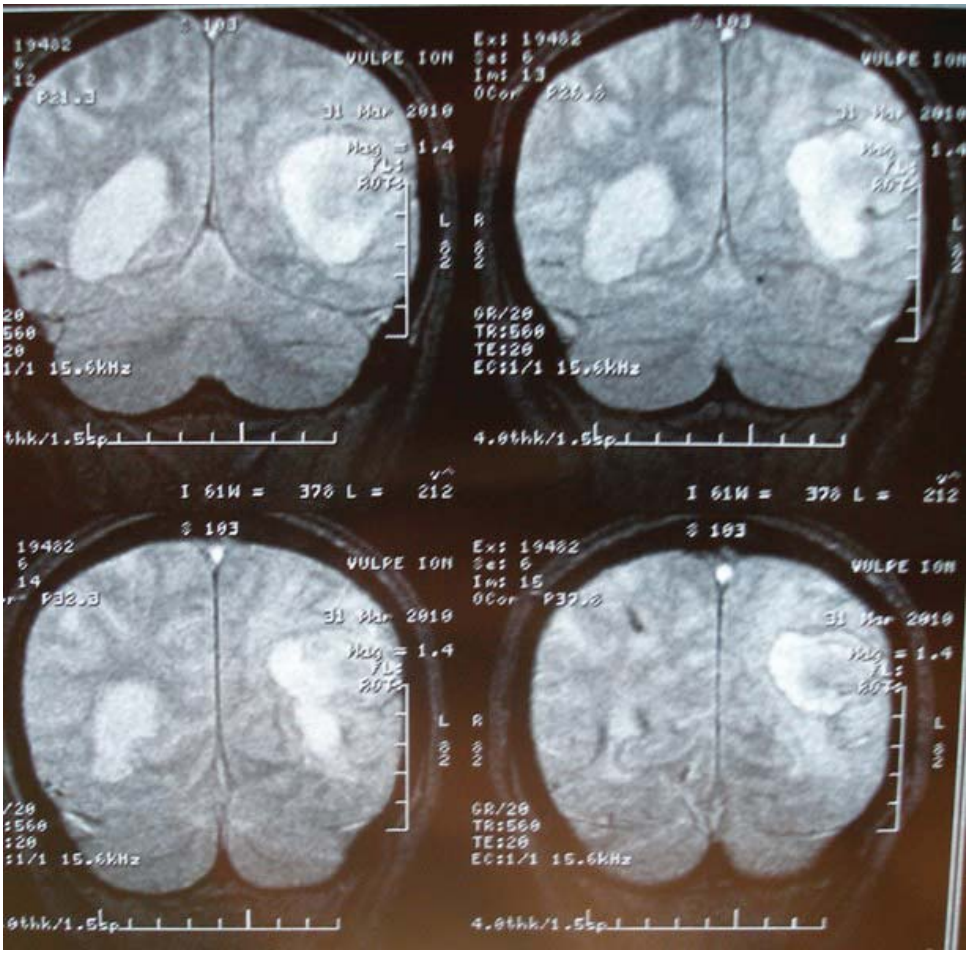

Figure 1. *CT performed at admission, MRI-T2 and T2**

ately. The CT scan performed at the time of the admission revealed an intracerebral hematoma located in the left hemisphere with extension in the parietal and temporal lobes at the surface of the brain near the parietal bone and some degree of edema and slight deviation of the midline. Also, there were two old areas of hypoattenuation, one located in the right occipital lobe and one in the frontal lobe. We further evaluated this patient using MRI of the brain and MRA to detect any venous abnormalities. We asked for specific T2, T1, FLAIR and gradient-echo MRI sequences to asses the abnormalities of the brai of the patient.The T1, T2 sequences revealed an subacute stage of the parieto-temporal hematoma and the gradient-echo-T2* sequence revealed that the right occipital area of hypoattenuation was infact an sequelae of an old hematoma. The evolution of the patient was good, the hematoma resorbed and he was discharged home. This case is particular because it is an cortical cerebral non-spontaneous hemorage in a patient with moderate values of the systolic blood pressure, cognitive deterioration accentuated by the visual loss secondary to the hematoma. We raised the suspicion of an cerebral amyloid angiopathy because of the clinical history, the presence of two cerebral hematoas and cognitive deterrioration in a patient with no remarcable history of hypertension. According to the Boston Cerebral Amyloid Angiopathy Group the patient has a diagnostic of probable CAA.

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