Seizure (2004) **13**, 328—330

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Heraldic seizure

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KEYWORDS
Heraldic seizure;
Hemorrhage;
Stroke;
Epilepsy

Summary *Background*: The term heraldic seizures indicates epileptic seizures caused by cerebrovascular disease, believed to be triggered by silent ischemia and occurring before a stroke. This fact widens the spectrum of possible interrelations between epilepsy and cerebrovascular disease outside the well known context of post-stroke epilepsy. *Methods*: This is a case report of a healthy 67-year-old male who had a new onset epileptic seizure prior to a lobar intracerebral hemorrhage (ICH). This man began to suffer myoclonic jerks in his left arm which progressed to a generalized tonic—clonic seizure. At the emergency area the physical and neurological examination were unremarkable and a CT scan was normal. The next day the patient developed left hemiparesis, hemianopsia and confusion and a new CT scan showed right parietal—occipital ICH. *Conclusions*: This case report exemplifies the concept of heraldic seizures, showing a patient who had a focal seizure preceding an intracerebral hemorrhage. Our etiologic diagnostic work led us to a diagnosis of probable amyloid angiopathy. We suggest that cerebral amyloid angiopathy (CAA) may be the underlying cause, since it may be the origin of both the late event (ICH) and the heralding seizures, resulting from concurrent ischemia.

Introduction

Stroke is the most frequent cause of epilepsy in adults, specially in those over 60 years. The relationship between epilepsy and cerebrovascular disease in the context of post-stroke seizures has been widely reported and its frequency has been estimated at 6—21%.[1, 2] The term heraldic seizures indicates epileptic seizures caused by cerebrovascular disease, believed to be triggered by silent ischemia and occurring before a stroke.[2] It is generally assumed that these seizures may herald ischemic strokes, but few cases of seizures heralding a primary intracerebral hemorrhage have been reported.[3]

Case report

A previously healthy 67-year-old male, without history of neurologic disease or hypertension, suddenly began to suffer myoclonic jerks in his left arm, which rapidly progressed to a generalized tonic—clonic seizure. The physical and neurological examination at the Emergency Department were unremarkable. A computed tomography (CT) scan was normal (Fig. 1) and he was admitted to the Neurology Department. The next day, without any treatment, the patient developed left hemiparesis, hemianopsia and confusion, and a new CT scan showed right parietal—occipital intracerebral hemorrhage (ICH) (Fig. 2). During the following days, the patient improved and serial CT scans showed a gradual resorption of hemorrhage, without evidence of underlying lesion. A magnetic resonance

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1059-1311/$ – see front matter
doi:10.1016/j.seizure.2003.08.008

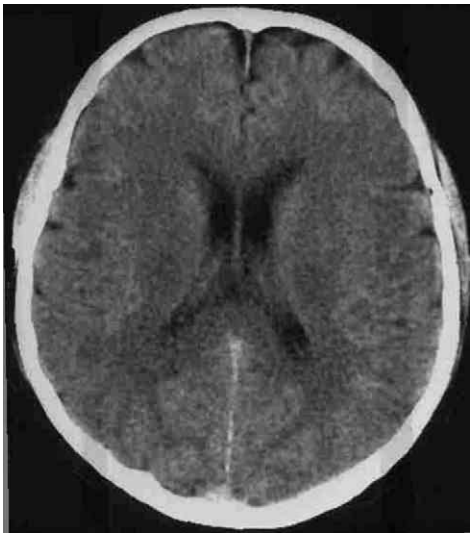

Figure 1 Initial CT scan.

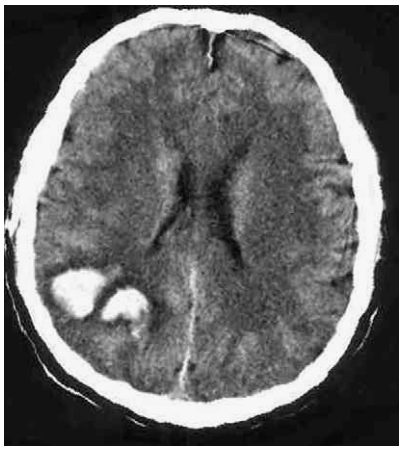

Figure 2 Second CT scan, showing the ICH.

angiography and a transcranial Doppler were normal and a coagulopathy was excluded. The patient was put on antiepileptic treatment and, during a 2-year follow-up, his neurological status has been unchanged.

Comment

This case report exemplifies the concept of heraldic seizures, showing a patient who had an intracerebral hemorrhage preceded by an epileptic seizure. There is not consensus about the definition of heraldic seizure and, in fact, the heraldic seizures are not mentioned in the ILAE classification of seizures. However, two formalities could be established as necessary in order to make a diagnosis of heraldic seizures: a temporal and a topographic relationship between both events.[2, 3] In our patient, the seizure and the ICH were related in time (a few hours), as well as in space (right hemisphere), fulfilling both diagnostic criteria.

It is well known that a silent ischemia could be responsible for a seizure.[4, 5] However, the mechanisms by which a seizure should herald an ICH are hard to explain. Our etiologic diagnostic work excluded a vascular malformation as origin of bleeding, leading us to a diagnosis (taking into account the lobar location and the absence of hypertension) of amyloid angiopathy. The occipital location of hemorrhage in the patient was a further, though indirect, evidence in favour of this hypothesis. According to Cocito et al.[3] we suggest that cerebral amyloid angiopathy (CAA) may be the underlying cause, since it may be the origin of both the late event (ICH) and the heralding seizures, resulting from concurrent ischemia.

An alternative but less probable explanation might be a leakage of blood from a small malformation before the massive event, acting as a triggering factor for seizures.[6] In our case a CT scan performed at the time of seizure was normal so one should postulate that the potential bleeding was below the detection threshold of this technique.

CAA is an entity of unknown origin caused by the deposition of beta-amyloid in the media and adventitia of small- and mid-sized arteries of the cerebral cortex and the leptomeninges, leading to the thickening of the basal membrane, stenosis of the vessel lumen, and fragmentation of the internal elastic lamina.[7] This can result in fibrinoid necrosis and microaneurism formation, predisposing to hemorrhage. Frequently asymptomatic, CAA can present as intracerebral hemorrhage, dementia or transient ischemic attacks.[8] Although CAA has generally been diagnosed by neuropathologic examination, a group of stroke neurologists in the Boston area proposed criteria for the diagnosis of CAA based on clinical and neuroimaging information.[9] In our case report, the patient developed a hemorrhage which fulfilled the Boston Criteria of probable CAA.

In conclusion, we propose the following sequence of facts: a CAA caused a transient ischemia, clinically apparent as a seizure. After an asymptomatic period, the underlying amyloid disease gave raise to bleeding with the same lesional topography.

This case supports the idea that seizures heralding ICH, though uncommon, do occur, and this fact widens the spectrum of possible interrelations between epilepsy and cerebrovascular disease. But not less important is to recognize the significance of CAA as an entity with a much wider clinical polymorphism than generally thought, and growing significance for aging population.

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