A 44 year old man had suffered from ophthalmic migraine since the age of 5. His attacks always started with a brilliant and scintillating visual disturbance in the central part of vision, followed by a left homonymous hemianopsia which lasted about 10 minutes. Then a diffuse headache appeared, worsening over 30 minutes, lasting about 5 hours and accompanied by vomiting. Attacks remained infrequent until he was 40, when their frequency increased from twice a year to twice a month.

At 42, the patient had an attack starting as an usual one, but, a few minutes after the onset of the left visual field defect, he complained of weakness of the left lower limb and of numbness of the left side of the tongue, lip and palate and of the first three fingers of the left hand. He also had a severe headache which lasted several hours. The other neurological symptoms lasted about one week and then disappeared except for a left upper homonymous quadrantanopsia and a persistent numbness of the left side of the palate. Brain CT scan performed two days later showed, without contrast infusion, an area of decreased density compatible with an infarct in the right posterior cerebral artery (PCA) territory (fig. 1); at the antero-internal edge of the hypodensity (fig. 4). Right vertebral selective angiography revealed a saccular aneurysm appended on the division of the right PCA into temporal and occipital arteries (fig. 5). After neurosurgical advice, surgical treatment of the aneurysm was not recommended.

The family history of this patient revealed that his daughter, aged 21, suffered from typical ophthalmic migraine and that her CT scan with contrast infusion was normal.

Discussion

This patient had, since childhood, recurrent attacks typical of ophthalmic migraine with visual symptoms progressing gradually, lasting 10 minutes and followed by a headache of several hours associated with vomiting. When he was 42, he was left after an attack with a permanent field defect due to an occipital infarct with occlusion of the P.C.A.

Lasting neurological deficits, though highly infrequent, do occur in migraineus subjects. In such cases, CT scan studies have shown anomalies suggestive of cerebral infarcts, which have been confirmed by pathological studies. Angiography is usually normal, but some cases show occlusion of one of the major cerebral arteries, most commonly the P.C.A. However, given the high prevalence of migraine, 15 to 30% of all stroke patients would be expected to have a past history of migraine. Therefore, to causally link cerebral infarcts to migraine itself, three criteria are thought to be essential. First, the
neurological deficit should mimic the migrainous symptoms of previous attacks. Secondly, it should occur during an attack. Thirdly, other causes of stroke should be excluded. Our patient fulfilled the first two criteria but not the third one, since CT scan performed two days after the attack showed, in addition to the occipital infarct, a small hyperdensity probably corresponding to a circumscribed area of bleeding. This finding, most unusual in complicated migraine, led us to repeat angiography two years later and to discover an aneurysm of the right PCA. The occlusion of the PCA which precluded the visualization of the aneurysm on the first angiogram was presumably due to vasospasm, induced either by the small hemorrhage or by the migrainous process itself. These findings of an arterial occlusion initially and, later on, of an aneurysm, emphasize the need of thorough and repeated evaluations of patients with so-called “migrainous infarcts” in order to look for other causes of stroke.

The relationships between migraine, stroke and arterial aneurysm in this case are unclear. The concordance between the site of the aneurysm, the site of the infarct, and the symptomatology of the migrainous attacks makes a fortuitous association very unlikely. Two main hypotheses can be put forward. The first is that the patient had two different diseases: migraine and arterial aneurysm. In this hypothesis, the arterial
aneurysm could have been the triggering factor of migrainous attacks in a susceptible individual, its location on the PCA explaining attacks of the ophthalmic type. A second hypothesis is that the patient had only one disease: migraine. The mechanism of vascular occlusion in cases of migrainous infarcts remains widely unknown. Several factors have been incriminated such as arterial vasospasm, edema, or dissection of the arterial wall, and increased platelet aggregation. In this hypothesis, the occlusion of the right PCA in our case could have been secondary to a localized dissection of the arterial wall with subsequent development of an arterial aneurysm.

A remarkable feature of the present case is the sequence of events that took place during the visual aura once the occipital infarct had occurred. The scintillating visual disturbance started as usual in the center of vision, but now disappeared for 5 minutes to reappear as a scintillating crescent at the edge of the left upper quadrantanopsia (fig. 3). Such a pattern suggests that, although scintillations were not present in the blind part of the visual field, the phenomenon leading to scintillations did progress over the cortex despite and across the infarcted area. Such a progression, previously remarkably described by Lashley is difficult to explain on a vascular basis and would suggest a primary neuronal phenomenon, possibly Leao spreading depression as speculated long ago by Milner and more recently by Olesen et al.

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