—Case Reports—

Migraine-Like Headache with Aura Induced by a Small Infarct in the Parieto-Occipital Cortex: A Case Report

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A 56-year-old right-handed man was referred to our hospital for evaluation of sudden-onset transient quadrantanopia, which was followed by throbbing headache consistent with migraine with aura (MA). Magnetic resonance imaging (MRI) of the right parieto-occipital cortex on admission showed a hyperintense region on diffusion-weighted imaging, which disappeared 7 days later. A small cortical infarct in the parieto-occipital cortex can cause MA-like headache, and the present infarct lesion was only detectable on MRI during the acute phase. Performing MRI for patients with suspected acute MA might help identify the cause of MA-like headache and ensure appropriate management of patients.

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Key words: acute ischemic stroke, migraine with aura, pathophysiology, magnetic resonance imaging

Introduction

Ischemic stroke and migraine are common neurological disorders[1]. The relationship between stroke and migraine, especially migraine with aura (MA), has been investigated[2–4], and MA is considered a cause of ischemic stroke, or migrainous infarction[5–7]. This direct relationship may be bidirectional, as some reports suggest that MA can be induced by ischemic stroke[8–10]. However, the evidence is limited, and the prevalence of stroke-induced MA, or the proportion of patients with stroke-induced MA among all those with MA-like headache, is not well known. Here we present a case of typical MA-like headache induced by a small infarct in the parieto-occipital cortex. The infarct lesion was only detectable on magnetic resonance imaging (MRI) during the acute phase and was not detectable on subsequent MRI scans. MRI during the acute phase may help clarify the role of ischemic stroke in MA.

Case Presentation

A 56-year-old right-handed man was referred to our hospital for evaluation of sudden-onset transient quadrantanopia in his left lower visual field. Quadrantanopia occurred suddenly and persisted for approximately 30 min, without change in the shape or extent of the quadrantanopic field. A few minutes after complete recovery from quadrantanopia, a flickering multicolor zigzagged line appeared in his left visual field for about 15 min, followed by 15 min of distorted vision in his left field of vision. After the distorted vision resolved, he experienced a severe throbbing headache on the right side of his head, along with nausea, photophobia, and phonophobia. The headache continued for about 20 h and worsened with mild physical activity. The characteristics of his headache and preceding flickering zigzag and distorted vision were consistent with those of migraine with aura, as described in the International Classification of Headache Disorders, 3rd edition (ICHD-3)[5]. He reported having a pulsating headache after scintillating scotoma, which is characteristic of MA, 5 years earlier, and mild headache without accompanying symptoms once every few months. He also had a history of hypertension, dyslipidemia, high uric acid, and a myocardial infarction 4 years previously. There was no family history of migraine.

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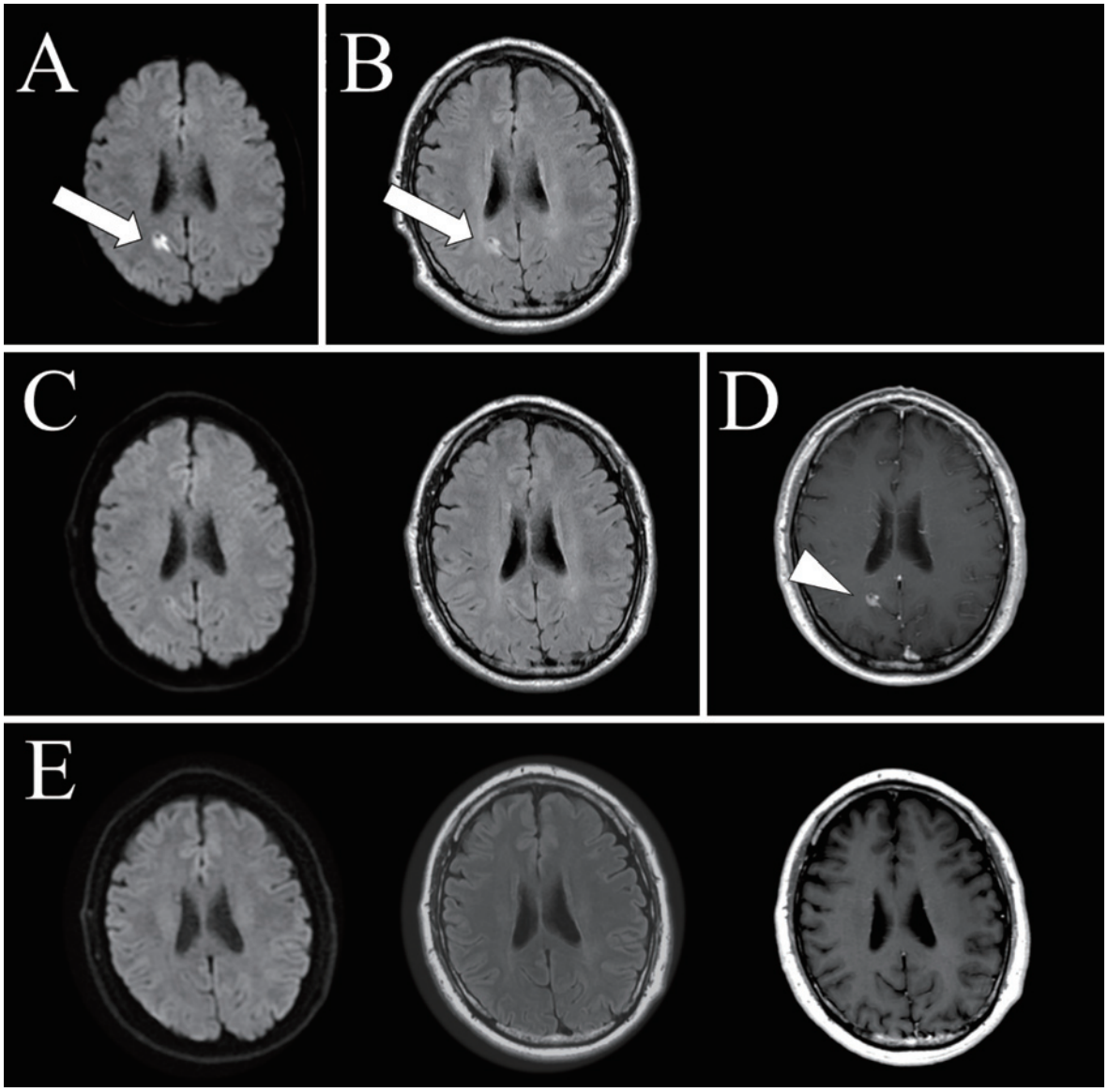

Fig. 1 Serial MRI scans. On admission, diffusion-weighted imaging (DWI, A) and fluid-attenuated inversion recovery (FLAIR, B) show a hyperintense signal near the right parieto-occipital sulcus (arrow). Seven days after onset, the lesion visible on DWI and FLAIR has disappeared (C) but is detectable on gadolinium-enhanced T1-weighted MRI (T1-WI, D, arrowhead). A follow-up MRI 2 months after onset shows no definitive lesion, even on T1-WI with gadolinium enhancement (E).

The results of general physical and neurological examinations, including visual fields, were normal on admission. Diffusion-weighted MRI (DWI; **Fig. 1A**, arrow) and fluid-attenuated inversion recovery (FLAIR, **Fig. 1B**, arrow) sequences on admission showed hyperintensity in the right parieto-occipital cortex around the parieto-occipital sulcus. FLAIR showed only subtle white matter lesions. Magnetic resonance angiography showed no occlusion or stenosis of the main intracranial arteries, including the posterior circulation. Seven days after onset, the lesion disappeared on DWI and FLAIR (**Fig. 1C**) but was visible with gadolinium enhancement on T1-weighted imaging (T1-WI, **Fig. 1D**). The findings of surface electroencephalography (EEG) were unremarkable. Single-photon emission computed tomography (SPECT) using intravenous injection of N-isopropyl-p-[^{123}I] iodoamphetamine (^{123}I-IMP) on day 9 from onset showed decreased ^{123}I-IMP uptake around the lesion. Laboratory testing yielded normal results for thyroid function, autoantibodies (including anti-nuclear, anti-phospholipid, and anti-neutrophil cytoplasmic antibodies), and protein S and C antigens and activities. No JAK2-V617F mutation was found. No right-to-left shunt was seen on transesophageal echocardiography, and 24-h Holter electrocardiography showed no evidence of arrhythmias that could cause ischemic stroke. Transthoracic echocardiography

showed akinetic areas in the antero- and inferoseptal wall, and cardioembolic stroke was diagnosed. His headache was classified as a stroke-induced, MA-like headache because quadrantanopia preceded the aura and DWI showed an ischemic lesion compatible with quadrantanopia. Warfarin was administered for secondary prevention, and he was discharged with no neurological deficit. MRI scans at 2 months after onset showed no evidence of the lesion, even with gadolinium enhancement (**Fig. 1E**).

Discussion

There are few reports of stroke-induced MA[9,10], although stroke-induced MA may be more frequent than previously thought[8]. In the present case, MA-like headache was likely induced by ischemic stroke, rather than migrainous infarction, because quadrantanopia preceded the flickering zigzagged pattern and distorted vision. Migrainous infarction is caused when prolonged (>60 min) cortical spreading depression (CSD) reaches below the critical threshold of infarction; therefore, symptoms of infarction always present after the aura. A small cortical infarct in the parieto-occipital cortex can cause visual symptoms followed by pulsatile headache, the characteristics of which are consistent with those of MA.

In the present case, stroke symptoms resolved completely in 30 min, and the DWI abnormality disappeared 7 days after onset. However, the lesion could be visualized on T1-WI with gadolinium enhancement (**Fig. 1D**). Disappearance of a DWI lesion is strongly associated with recanalization of the occluded artery[11,12], and enhancement with gadolinium results from disruption of the blood-brain barrier (BBB)[13]. Microemboli can cause CSD[14], and the duration and severity of ischemic insult are crucial: ultra-transient occlusion causes CSD, whereas longer or large-artery occlusion causes ischemic stroke[15]. Disruption of the BBB can cause leakage of plasma proteins, including pro-inflammatory cytokines that induce hypersensitivity of meningeal sensory neurons, which leads to headache[16]. The present case may represent a proof-of-concept of the theoretical mechanisms for stroke-induced MA-like headache and suggests that stroke-induced, MA-like headache can occur in embolic stroke patients with mild or transient symptoms and gadolinium-enhanced lesion(s) in the cortex.

In a mild stroke or transient ischemic attack (TIA), the DWI abnormality disappears quickly, and only DWI during the acute phase can show the responsible lesion(s)[17]. Gadolinium-enhanced T1-WI can detect lesions in TIA patients even in the subacute phase[18]. Stroke-induced MA-like headache has been rarely reported, partly because stroke symptoms in such patients are mild, and the ischemic lesion is detectable only during the acute phase, even on MRI. Performing MRI including the DWI sequence for MA patients in the acute phase and T1-WI with gadolinium injection in the subacute phase may help identify the potential cause of an MA-like headache and guide management of patients, an important consideration because some treatments for MA may harm patients with acute ischemic stroke[19].

Conflict of Interest: None.

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