CASE REPORT

Susceptibility-weighted Imaging and Magnetic Resonance Angiography during Migraine Attack: A Case Report

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We performed magnetic resonance angiography (MRA) and susceptibility-weighted imaging (SWI) to observe cerebral changes during a migraine attack in a 28-year-old man. MRA revealed regional arterial spasm, and findings of SWI were consistent with decreased blood flow and/or venous dilatation. Because these noninvasive techniques can be performed repeatedly at short intervals, they can be useful for evaluating hemodynamic changes during and after migraine attacks.

Keywords: *magnetic resonance angiography, migraine attack, MRI, susceptibility-weighted imaging*

Introduction

Migraines affect approximately 8.4% of adults in Japan (male, 3.6%; female, 12.9%), with patients usually asymptomatic except during attacks.

Many studies using single-photon emission computed tomography (SPECT) or positron emission tomography (PET) have described the changes in cerebral blood flow (CBF) during migraine attacks.^{1–3} However, the use of radioactive isotopes with these techniques prohibits their repeated use, so the serial hemodynamic changes during migraine attacks remain unclear. Magnetic resonance (MR) imaging is noninvasive and can be performed repeatedly at short intervals, so MR angiography (MRA) is used to examine the arteries and susceptibility-weighted imaging (SWI), to assess veins.^{4,5}

We report a migraine case in which MRA and SWI scans were repeatedly obtained between the aura and headache phases.

Materials and Methods

A 28-year-old man presented with a 10-year history of migraine with aura (MwA), diagnosed according to the International Headache Society

criteria. The attacks occurred approximately 5 times per year and were distinct from other types of headache.

On one occasion, the patient developed a scintillating scotoma, a typical migraine symptom, in the central visual field. The scotoma moved toward the periphery of the left visual field and eventually disappeared.

We performed MR imaging scans approximately 15 min, 45 min, and 1.5 hours after the aura phase began and a final scan during follow up, at 30 weeks after the attack.

No medication was administered during or after the attack.

All images were obtained using a 1.5-tesla scanner (Siemens Avanto, Erlangen, Germany). We performed MRA with a 3-dimensional time-of-flight sequence (repetition time [TR], 39 ms; echo time [TE], 7.2 ms; flip angle [FA], 25°; field of view, 220 mm; slice thickness, 1 mm; matrix size, 512 × 188) and used maximum intensity projection (MIP) to evaluate images. We performed SWI with a 3-dimensional fast low-angle shot sequence (TR, 49 ms; TE, 40 ms; FA, 20°; field of view, 255 mm; slice thickness, 3 mm; matrix size, 448 × 264) and conducted postprocessing using a previously described method.^{4,5} Briefly, phase mask images were obtained from phase images and were multiplied 4 times followed by multiplication on the magnitude

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images. Then SWI was obtained by minimal intensity projection (minIP) with 12-mm slice thickness.⁵

We retrospectively evaluated venous structures on minIP images of SWI and arterial systems on MIP images of MRA and compared them with follow-up images.

Results

The first MR imaging scan, performed approximately 15 min after the aura phase began, revealed markedly obscure peripheral branches of the right middle cerebral artery (MCA) and posterior cerebral artery (PCA) on MRA (Fig. 1a). SWI revealed normal intracranial veins (Fig. 2a).

MR imaging performed approximately 45 min after the aura phase began, when the patient developed a severe, pulsating headache in the right hemicranium accompanied by nausea, photophobia, and blurred vision, demonstrated normalization of the peripheral branch of the right MCA and visualization of the branch of the right PCA not observed during the aura phase (Fig. 1b); this indicated improvement of the vasospasm. In addition, the surrounding veins were conspicuous (Fig. 2b).

MR imaging performed during intense headache, approximately 1.5 hours after the aura phase began, showed marked dilation of the branch of the right PCA (Fig. 1c), and most intracranial veins were more conspicuous on SWI (Fig. 2c) than on previous scans.

The patient underwent MR imaging again during follow up 30 weeks after the attack, during which interim he experienced no further attacks. MRA revealed slight reduction of the diameter of the right PCA branch (Fig. 1d), which indicated that it was dilated during the headache phase. The other vessels appeared unchanged. SWI revealed that all the previously dilated veins had returned to normal (Fig. 2d). T₂-weighted (T₂WI) and fluid-attenuated inversion-recovery (FLAIR) images showed no abnormalities at the time points considered (Fig. 3).

Discussion

We performed MRA to observe serial changes in the intracranial arteries and SWI, in the veins, during a migraine attack. We believe this is the first SWI-based study that describes MwA-induced changes in cerebral veins.

There are 3 major theories, neuronal, trigeminovascular, and vascular, regarding the pathophysiology of migraine. The neuronal theory involves the depression of cortical spreading¹; the trigeminovascular theory, the release of vasoactive

Fig. 1. Time course of magnetic resonance angiography (MRA) images. The peripheral branches of the right middle (MCA) and posterior (PCA) cerebral arteries are most obscure during scintillating scotoma (a), compared to follow-up image (d). Visualization of these branches improves in the early stage of headache (b). Then, the PCA branch appears most dilated during the most intensive headache (c). Follow-up image (d) shows slight reduction in the diameter of this PCA branch, which indicates that this branch was dilated during the headache phase. The appearance of other branches did not change throughout the attack.

neuropeptides inducing sterile inflammation and consequently causing pain⁶; and the vascular theory, the initiation of cortical hypoperfusion in the

Fig. 2. Time course of susceptibility-weighted images (SWI). Temporal changes in the veins are shown. Intracranial veins appear almost normal on SWI during spreading scotoma (a), compared to follow-up image (d). However, the veins of the right occipital lesion are less conspicuous at the beginning of throbbing headache (b). Then, most of the intracranial veins become more conspicuous on SWI during the most intensive headache (c). On follow-up scan, these veins appear normal (d).

visual cortex that spreads during an attack.^{2,7} The CBF decreases over one hour, independent of the cerebral arterial topography.⁸

Several PET-based studies^{2,3,9} have examined the intracranial hemodynamic changes during migraine attacks and focused on changes in CBF. However, MRA has rarely been used for such investiga-

Fig. 3. There are no abnormal findings on T₂-weighted (T₂WI) (a) and fluid-attenuated inversion-recovery (FLAIR) (b) images throughout the migraine attack and follow up.

tions.¹⁰ MR imaging offers several advantages over PET or SPECT, such as relatively short scan time, high spatial resolution, and the option of emergency and repeated scanning. SWI is a novel technique for noninvasive evaluation of venous systems.^{4,5} Using MRA and SWI, we successfully examined the serial hemodynamic changes during a migraine attack.

MRA performed during the aura phase revealed spasm in the branch of the right MCA and PCA, consistent with previous findings of oligemia in the affected hemisphere during the aura phase.^{2,3,6,11} MRA also revealed slight improvement in the vasospasm after the aura phase that persisted in the early headache phase, as reported by Marshall and associates¹⁰ and Sanin's team.¹² In addition, the apparent dilation of the branch of the right PCA was consistent with previous reports of increasing CBF during the headache stage.^{2,3,9,11} However, Olesen's group reported a persistent reduction in CBF between the aura and headache phases.¹

Deoxygenated hemoglobin (deoxy-Hb) is an intrinsic contrast agent for SWI.^{4,5} Thus, visualization of the venous system depends on the oxygen saturation or intravenous concentration of deoxy-Hb.^{4,5} Arterial spasms or stenoses reduce CBF and increase venous concentration of deoxy-Hb, thereby rendering venous systems considerably conspicuous.^{4,5} Here, SWI scans showed no venous changes during the aura phase, when CBF might have been low because of spasm in the branch of the right PCA. However, during the early headache phase, the veins around the right PCA were prominent. This accords with the above-mentioned mechanism, although there was a slight time lag be-

tween the arterial spasm and venous conspicuity. Almost all cerebral veins were conspicuous on SWI scans obtained when the headache intensified, whereas arterial changes were localized to the right occipital area. This finding is inconsistent with the mechanism. Because no cerebral artery other than the branch of the right PCA showed change, the conspicuousness of the cerebral veins during intense headache is probably not attributable to CBF changes. We speculate that the SWI findings were due to either vein dilation or increased oxygen consumption, which led to an increase in the intravenous deoxy-Hb concentration; however, further studies are needed to ascertain this.

In conclusion, we performed MRA and SWI to observe the arterial and venous changes during a migraine attack. These techniques are useful for evaluating the serial hemodynamic changes during migraine attacks.

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