

The Middle Meningial Artery during a Migraine Attack: 3T Magnetic Resonance Angiography Study

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Abstract

We performed 3T magnetic resonance angiography (MRA) during a spontaneous migraine attack. The patient was a 42-year-old woman migraineur diagnosed by the IHS criteria. The change of the middle meningeal artery (MMA) was measured on the axial brain images using MATLAB for three phases (attack-free period, during an attack, a period after medication). There were no dramatic changes of vasodilation in the MMA during the attack (2.0 mm), attack-free period (diameter 1.9 mm), or period after medication (1.7 mm), resembling extrapolations of observations in experimental animal models. This finding suggests that the dramatic vasomotion might not be associated with migraine pathophysiology.

Key words: migraine, middle meningeal artery, 3T magnetic resonance angiography

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Introduction

Migraine is generally considered to be a neurovascular disorder characterized by attacks of unilateral, throbbing headache associated with photophobia, osmophobia, nausea and vomiting (1). An important event in the pathophysiology of migraine is vasodilation consequent to the stimulation of the cerebral and middle meningeal arteries (MMA) by vasodilative substances such as calcitonin gene-related peptide (CGRP) (2). In the present case, we found no detectable vasodilation of MMA during migraine attack by 3T magnetic resonance angiography (MRA).

Case Report

A 42-year-old woman who had migraine headaches without aura for twenty years was diagnosed with migraine without aura by us in accordance with IHS (ICHD-II) criteria two years previously (3). She usually took the triptan, zolmitriptan (4), at the beginning of the migraine attack. Her migraine attacks occurred once or twice a month and were accompanied by nausea and osmophobia. She had migraine

attacks mainly on the right side of her head. Moreover, she did not take prophylactic treatment. Her headaches usually lasted a whole day, and were unrelated to her menstrual cycle.

One evening she experienced a typical migraine attack on the right side, accompanied by nausea and osmophobia and was immediately examined at our hospital. We performed magnetic resonance angiography (MRA) of both middle meningeal arteries (MMAs) with a 3.0-Tesla whole-body system (Philips Achieva 3.0T Qusar dual, SENSE head coil [8ch]) during the migraine attack (Fig. 1). The timing of the scan was two hours after the beginning of migraine attack. The blood vessel diameter protocol consisted of a thick 2D phase-contrast sagittal localizer survey, followed by a 3D time-of-flight MRA sequence to visualize the MMAs on both sides. The scan had the following imaging parameters: repetition time/echo time: 25 ms/3.45 ms; flip angle 20; field of view: 200×200 mm; Matrix 560×560 (reconstructed voxel size 0.36 mm×0.36 mm×0.50 mm); slice thickness/gap 1 mm/-0.5 mm; number of slices 140; chunks 4; SENSE reduction factor 2 (phase); NSA 1; scan time 6 minutes.

The MR scan was performed three times: the first scan

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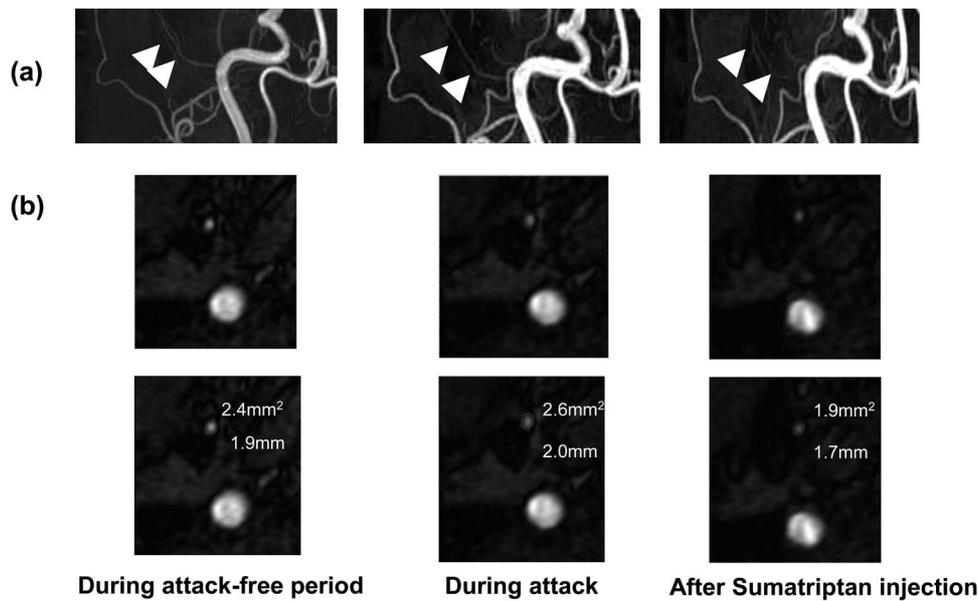


Figure 1. Head-on views of the middle meningeal arteries (MMAs) and coronal sections of the changing right MMA during the migraine attack. (a) The right MMA was slightly dilated during the migraine attack. After the sumatriptan injection, the MMA constricted. (b) The changes in blood vessel area of the right MMA at headache-free period, during the migraine attack, and after the sumatriptan injection are shown. Arrowhead: Middle meningeal artery.

was performed when the patient was free of migraine attack, one week after the previous migraine attack. The second scan was performed while she was experiencing a migraine attack. The last scan was done shortly after she was given an injection treatment. The diameter and the area of MMAs were measured on each of the axial brain images. These measurements were performed using MATLAB (MathWorks, Natick, MA). Since signal intensity of arteries on MRA images obtained using the time-of-flight (TOF) method depends on the flow velocities, intensity variations are usually observed even in the same artery. Therefore, it is often difficult to determine the exact rim of arteries. In our measurement, the pixels of which signal intensities were higher than 30% of the maximum intensity of MMAs were selected. We supposed that they represented actual internal cavity of the right MMA assuming that there was non-negligible flow in these selected pixels. Moreover, simultaneously, we monitored her pulsation by a pulse oximeter. The University Ethics Committee reviewed the study's protocol, and the patient gave their informed consent after receiving an explanation of the procedure.

The patient complained of a severe headache and nausea during the migraine attack on the right side of her head. Dilatation (diameter 2.0 mm; area 2.6 mm²) of the right MMA was observed during the attack when compared with a headache-free period (diameter 1.9 mm; area 2.4 mm²), although it was not marked. However, there was no change in pulsations of the MMA between the migraine attack and headache-free period. Then, we subcutaneously injected a triptan, sumatriptan, and the headache and accompanying symptoms were attenuated 5 minutes after the injection. We

performed MRA on the right MMA again and were able to confirm constriction of the MMA (diameter 1.7 mm; area 1.9 mm²) (Fig. 1).

Discussion

This is the first report of an MRA study performed during a spontaneous migraine attack. Previous reports revealed that the pathophysiology of migraine might be associated with vasoactive substances such as CGRP, nitric oxide, and serotonin. As for CGRP which is a vasodilative substance, the diameter of MMA during the addition of CGRP was extended by more than 50% compared to the baseline in rats (2). On the other hand, the CGRP antagonist constricted the human MMA by 50-60% in vitro (5).

In the present case, there were small changes (5-15% diameter changes compared to the baseline diameter) in the diameter of the MMA during the migraine attack and the diameter of the MMA was smaller after the sumatriptan injection than during the attack-free period. However, we did not observe any dramatic changes in the diameter of the MMA during the migraine attack.

Schoonman et al reported that in migraine patients the baseline MMA diameter showed a standard deviation of approximately 0.2 mm (6). They also observed no dramatic changes in the diameter of the MMA during migraine attacks induced by nitroglycerine (6, 7) and they speculated that migraine attacks might not be related to vasomotion. In the present report, the diameters of the MMA were changed within 0.3 mm.

Previous reports showed that in patients with migraine

with aura, the occurrence of MRI-BOLD signal changes consistent with cortical spreading depression by Leao (8) was observed in the occipital cortex (9).

On the other hand, the middle cerebral artery (MCA) velocity on the headache side was significantly higher than that on the non-headache side, returning to normal values after treatment with sumatriptan (10). However, no change was seen in the cerebral blood flow in the MCA supply territory. Moreover, the vasoconstrictor effect of sumatriptan is not temporally related to headache relief (11-13).

In the present case, we did not observe significant vasodi-

lation during the migraine attack. However, we do not know whether some other part of the MMA was more dilated or whether other arteries in the brain were dilated. Otherwise, the small change of MMA might play an important role in the pathophysiology of migraine. This case is very valuable because it provides important information that dramatic vasomotion might not be associated with migraine.

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