Case Report

Evidence of Vascular Compromise over the Visual Cortex during Migrainous Headache: A Case Report with MRI Study†

Orasa Chawalparit MD*,
Watcharawoot Siriacharwattana MD**

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** Department of Radiology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand*
*** Neuroscience Center, Bangkok General Hospital, Bangkok, Thailand*

The objective of the present study was to demonstrate MRI evidence of vascular compromise as seen in a 17-year-old female presenting with migrainous headache. The patient had been experiencing migrainous headache 2 days ago. She had visual auras lasting for a few hours before the attack. Upon meeting with her physician she indicated symptoms of left sided numbness during the headache. No neurological deficit was detected when the first MRI was performed, and all of her symptoms resolved within 4 days of the attack.

The MRI study showed a focal area of restricted diffusion at the right visual cortex. A short segment of vascular enhancement was noted on the surface of the affected gyrus. MRS showed a normal N-acetyl aspartate, choline, and creatine with no elevation of lactate. A follow-up MRI study 5 months later showed normal finding with no residual lesion. The authors concluded that the abnormality on the first MRI was the ischemic insult of the ictal visual cortex which was transient during the migraine attack. The pathophysiology was more likely from reversible focal venous congestion.

Keywords: *Migraine, Vascular, MRI*

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Migrainous headache is one of the common neurological symptoms presenting in clinics. Investigation is usually not necessary in the typical symptomatic pattern and uncomplicated cases since a negative result is usually obtained from the imaging[1]. Computed tomography is a simple modality to exclude other serious condition mimicking migraine. In complicated cases, magnetic resonance imaging (MRI) is the first investigation of choice since it offers better resolution and sensitivity of the images. Here, the authors reported a case of a young migrainous female with abnormal MRI and discussed about the abnormality.

Material and Method

A 17-year old female had been experiencing migrainous headache 2 days ago. She had experienced a series of visual aura lasting for a few hours before the attack. Upon meeting with a physician she indicated symptoms of left-sided numbness starting right after the attack and during the headache. The numbness did not improved although the symptom of headache had decreased. No neurological deficit was detected when the first cranial MRI was performed. All of her symptoms resolved within 4 days of the attack. The follow-up MRI was performed 5 months later. For a single case report, ethical approval is void in the authors' institute. However, oral informed consent was received from the patient and guardian.

Correspondence to: Chawalparit O, Department of Radiology, Faculty of Medicine Siriraj Hospital Mahidol University, 2 Prannok Road, Bangkok 10700, Thailand. Phone: 0-2419-7086

Results

The first brain MRI study showed a focal area of high signal on T2-weighted image (T2wi)/fluid attenuated inversion recovery image (FLAIR) with restricted diffusion on diffusion weighted image (DWI) along the cortex of right visual gyrus (Fig. 1). A short segment of vascular enhancement was noted on the surface of the affected gyrus. Single voxel MR

spectroscopy showed normal N-acetyl aspartate, choline, and creatine with no elevation of lactate. MR angiography (MRA) demonstrated decreasing size and number of right sided cerebral arteries both for anterior and posterior circulations compared with normal flow signal and pattern of the left (Fig. 1E). No evidence of filling defect in the dural venous structure was detected on gadolinium T1-weighted image (Gd-T1wi). The other cerebral cortex showed no abnormal signal or enhancement.

The follow-up MRI study 5 months later showed a normal finding with no residual lesion (Fig. 2). Susceptibility image (Swi) was performed with Vein-blood-oxygen-dependent (VeinBOLD) technique (Philips, Achieva). When compared with the first MRI, the enhancing vascular structure was seen on Swi as venous structure (Fig. 3). On Gd-T1wi, the enhancement had disappeared. This confirmed that the loop of vascular structure could not be a developmental venous anomaly.

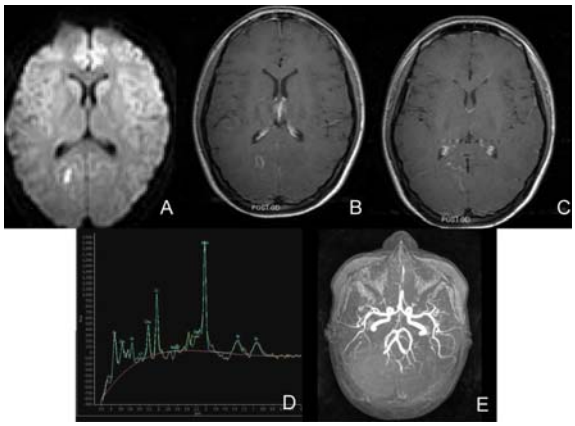

Fig. 1 MRI brain 3 days after the attack:(A) axial DWI shows restricted diffusion of the right visual cortex corresponding with high signal on FLAIR (not shown).(B, C) Gd-T1wi show a short segment of vascular enhancement at the surface of the right visual gyrus.(D) MR spectroscopy shows normal metabolic profile. (E) axial MRA shows decreased size and number of right posterior cerebral artery and branches of right middle cerebral artery compared with the left. No evidence of arterial stenosis or occlusion is demonstrated

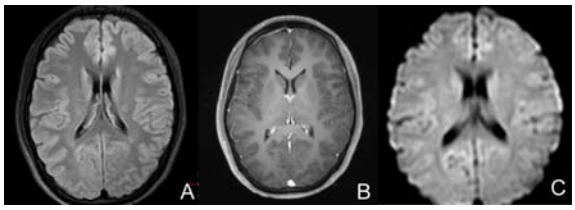

Fig. 2 Follow-up MRI at 5 months after the attack: (A) FLAIR, (B) Gd-T1wi, (C) DWI show no residual abnormal SI or enhancement

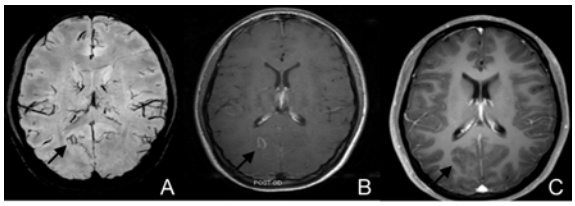

Fig. 3 Comparison of the enhancing vascular structure and the cortical vein: (A) VeinBOLD (Swi) at the follow-up time shows loop of cortical vein at right visual cortex corresponding with the enhancing vascular structure on Gd-T1wi (B) at the ictal time. This implied the evidence of venous stasis or congestion at the affected area. (C) Follow-up Gd-T1wi at the corresponding level shows resolving of the enhancement confirming no developmental venous anomaly

Discussion

Cortical spreading depression (CSD) has been increasingly accepted to describe pathophysiology of migrainous attack[2]. In migraine aura, neurovascular changes at visual cortex were demonstrated by functional MRI (fMRI). How stimuli trigger the process has not yet been well understood. It is believed that inflammation and excitotoxicity play a major role in the symptom process. Evidences of vascular change, either decreased or increased perfusion, were reported by various imaging modalities[3-7]. Posterior and middle cerebral arterial territories were reported as occurring more than other regions[8]. Reports of reversible cerebella ischemic lesions in migraine headache with visual aura were suggestive of constriction of blood vessels in the posterior circulation. Abnormal potassium and glutamate leakage from neuronal depolarization is believed to be the cause of vasoconstriction[9,10]. A case report of hyperperfusion was demonstrated arterial dilatation, however[11]. Reversibility of the high signal on DWI can also be found in transient arterial ischemic process. Though the present study did not show angiographic detail of the dilated small vascular structure or whether it was artery or vein, the MRA demonstrated decreasing size and number of arteries in both anterior and posterior circulations, not dilation. With the imaging characteristic on VeinBOLD (or susceptibility image), the enhancing cortical vessel was venous structure. This led to the possibility of the venous congestion during the attack. The persisting

dilated vein might imply ongoing inflammation of the neuronal process which manifested as hemiparesis of the contralateral side during the attack. The present study did not show evidence of ipsilateral cerebral ischemia or infarct as previously reported in patients with hemiplegic migraine[12]. The severity of the inflammation may thus be less in this present case, with no detectable neurological deficit and a resolving of the lesion.

A case report of migrainous headache with developmental venous anomaly (DVA) and surrounding parenchymal change was suggested evidence of venous congestion[13]. This present case was not DVA because no persistent enhancement of the abnormal vein was found on follow-up scan as is usually seen on DVA cases. Reports of migrainous headache induced by Queckenstedt's test were suggesting venous congestion in the pathophysiologic process[14,15]. The present case was additional evidence of venous congestion in the process of the migrainous headache. The cause of the venous congestion may be from chemical irritation during CSD causing decreased vasomotor activity at the venous site.

Conclusion

This study presented evidence of reversible ischemic process with venous stasis or congestion by MRI. The findings implied a temporary effect of migrainous pathophysiology to the cerebral venous structure.

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ความผิดปกติของหลอดเลือดในบริเวณผิวสมองส่วนการมองเห็น: รายงานภาพ เอ็มอาร์ไอของผู้ป่วยปวดศีรษะไมเกรนหนึ่งราย

อรสา ชวาลภาฤทธิ์, วัชราวุทธิ์ ศิริอาชาวัฒนา

หญิงไทยอายุ 17 ปี มีอาการปวดศีรษะ 2 วัน โดยเห็นแสงผิดปกติก่อนมีอาการ มาพบแพทย์ด้วยอาการซาซีกซ้ายระหว่างปวดศีรษะและไม่หายหลังจากหายปวดศรีษะ ตรวจร่างกายไม่พบความผิดปกติใด ๆ เอ็มอาร์ไอพบรอยโรคที่เปลือกสมองส่วนการมองเห็นข้างขวา มีสัญญาณสูงใน T2wi/FLAIR และ diffusion weighted image ที่ผิวสมองดังกล่าวมีหลอดเลือดซึ่งมีสาร gadolinium ค้างอยู่ หลอดเลือดแดงสมองด้านขวามีจำนวนลดลง และขนาดเล็กลงเมื่อเทียบกับข้างซ้าย การตรวจเอ็มอาร์ไอซ้ำอีก 5 เดือนต่อมา รอยโรคดังกล่าวหายไปเมื่อเปรียบเทียบกับภาพ susceptibility image หลอดเลือดที่ผิดปกติในการตรวจครั้งแรก ตรงกับหลอดเลือดดำที่ผิวสมอง และไม่มีการค้างของ gadolinium ในการตรวจครั้งหลัง ซึ่งแสดงถึงการค้างหรือบวมของหลอดเลือดดำในขณะที่มีอาการ