



Susceptibility-Weighted Imaging Findings in Patients Suffering from Migraine with Aura

Nerses Nersesyan¹ · Sebastian Arnold² · Peter Krieg³

Received: 25 September 2018 / Accepted: 1 March 2019 / Published online: 18 March 2019
© Springer-Verlag GmbH Germany, part of Springer Nature 2019

Introduction

Migraine is a neurological condition characterized by recurrent, unilateral, pulsating headaches that can last from 4 h to 72 h. They can be exacerbated by physical activity and are frequently associated with nausea, photophobia and phonophobia. An aura or the presence of other focal neurologic symptoms during an episode of migraine can be observed in approximately one third of the cases. The neurologic deficits that accompany migraine with aura pose a significant challenge necessitating other conditions, such as stroke, vasculitis or infections to be considered in the differential diagnosis [1]. In pediatric patients, bilateral headache, shorter duration, and nonspecific symptoms are more common than in the adult population. Tension headaches and stroke are both essential and challenging differential diagnoses.

This article presents a case report of two pediatric patients who had migraine with aura. The magnetic resonance imaging (MRI) examinations were performed on a 1.5T scanner (Siemens Quantum, Erlangen, Germany). A circular 8-channel polarized head coil was used and T2-weighted imaging (TR 3730s, TE 112ms, slice thickness 5mm), diffusion-weighted imaging (DWI) and susceptibility-weighted imaging (SWI) were carried out.

The authors declare that the submitted work has not been published before (neither in English nor in any other language) and that the work is not under consideration for publication elsewhere.

✉ Nerses Nersesyan
nerses90@gmail.com

¹ Dept. Radiology, Hospital Clínico Universitario de Valencia, Av. de Blasco Ibáñez, 17, 46010 València, Spain

² Dept. Radiology, Section Neuroradiology, Städtisches Klinikum Karlsruhe, Moltkestraße 90, 76133 Karlsruhe, Germany

³ Dept. Pediatrics, Städtisches Klinikum Karlsruhe, Moltkestraße 90, 76133 Karlsruhe, Germany

Patient 1

A 13-year-old boy was admitted to the neurology department with a left-sided frontal oppressive headache and nausea. The patient had ipsilateral visual loss, aphasia and bilateral leg pain. There was no family history of migraine and the patient was not receiving treatment with any medications. The headache and the accompanying symptoms resolved during the first 24 h. The laboratory, electrocardiography (EKG) and electroencephalography (EEG) findings were unremarkable. To rule out stroke MRI was performed, including SWI, DWI and MR angiography (MRA) sequences (Fig. 1). The SWI images showed well-defined unilateral left hemispheric engorgement of the epicortical venous system with sparing of the deep venous system in the otherwise normal MRI scan. The follow-up imaging performed 5 days later, showed the resolution of these findings.

The patient was diagnosed with migraine with aura and was prescribed ibuprofen for further episodes.

Patient 2

A 14-year-old girl complained of visual loss in the left eye and 30 min later a severe unilateral headache and nausea occurred. The girl had pronounced aphasia, with an otherwise normal neurologic examination. The symptoms resolved in the next 24 h. The patient was examined using MRI including SWI, DWI and MRA sequences (Fig. 2). The presence of prominent cortical venous vasculature was observed without signs of venous thrombosis. The follow-up imaging showed the complete resolution of the findings 2 days later.

Discussion

There are three leading theories attempting to explain the as yet unknown pathophysiology of migraine. The vascular

Fig. 1 **a** Axial susceptibility weighted image with well-defined unilateral left hemispheric engorgement of the epicortical venous system with sparing of the deep venous system.
b Follow-up imaging performed 5 days later showing resolution of these findings

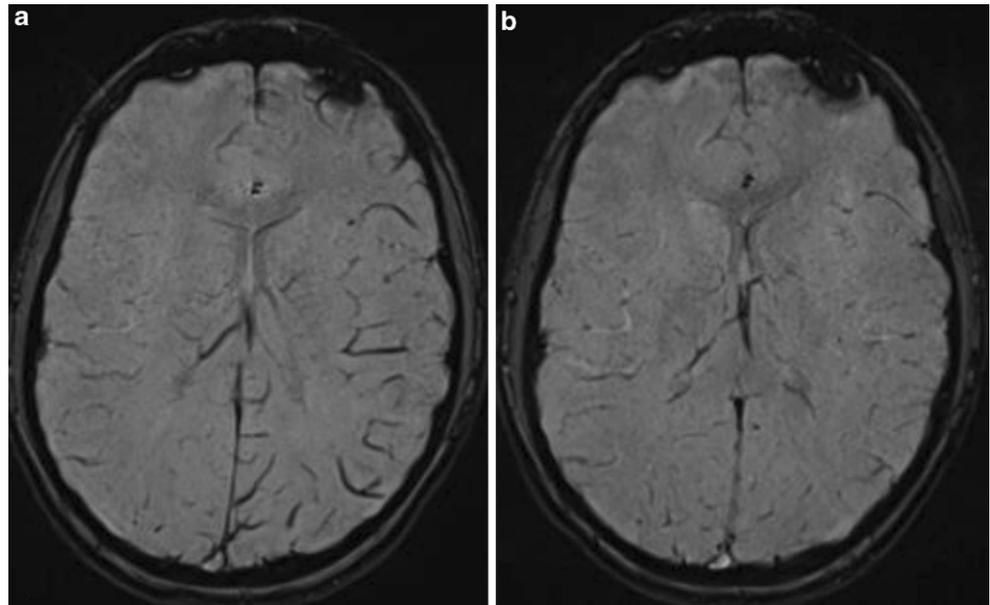
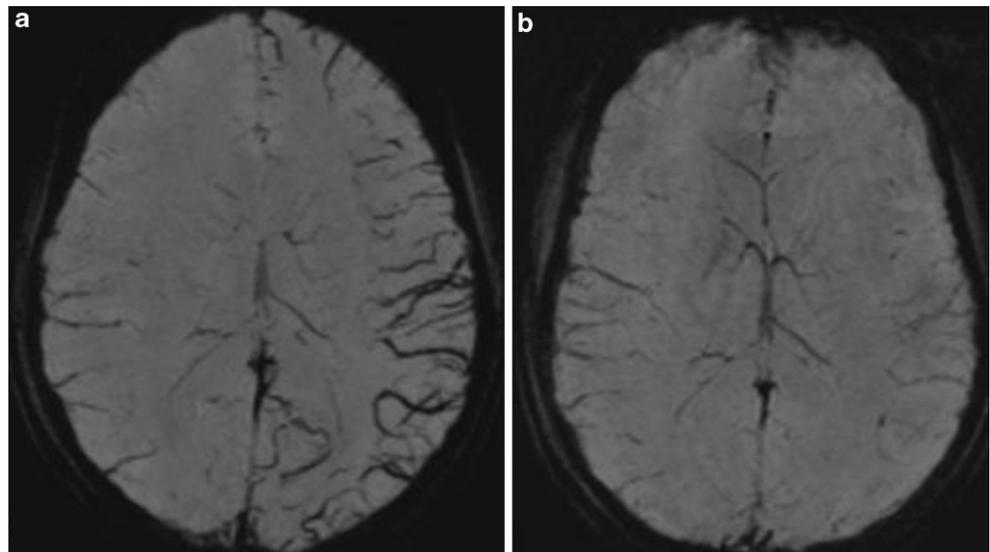


Fig. 2 **a** Axial susceptibility weighted image showing venous epicortical engorgement in the left hemisphere with sparing of the deep venous structures.
b Axial SWI image of the control study of the same patient 2 days later with a complete resolution of the findings



theory involves initial cerebral vasoconstriction, causing the aura and subsequent reactive vasodilatation responsible for the headache. The trigeminovascular theory focuses on the release of vasoactive neuropeptides causing inflammation and pain [2]; however, the most widely accepted theory, referred to as cortical spreading depression, is characterized by neural excitation followed by prolonged inhibition of neural activity in a posterior to anterior fashion [3]. Migraine with aura may present with acute deficits, potentially mimicking an acute ischemic stroke. As recognized stroke mimics, MRI sequences performed in these patients frequently focused on excluding more urgent conditions, such as acute venous or arterial thrombosis [4]. The most prominent findings of patients were the venous vascular engorgement observed on SWI, which was related to the side of

the symptomatic presentation. The signal changes did not have a territorial vascular distribution and all the other sequences (DWI and T2-W images) were completely normal [4, 5]. The technique of SWI is a full velocity compensated high-resolution 3D gradient-echo sequence that uses magnitude and filtered phase information to create new sources of contrast [5]. Although first developed in 1997, it is only in the last 10 years that SWI has become a widely available modality of MRI. Among other uses, SWI is considered to be a very sensitive method to monitor paramagnetic substances such as iron. Furthermore, the signals obtained from iron-containing proteins, such as deoxyhemoglobin, ferritin and hemosiderin, fluctuate depending on the amount of enclosed iron although the iron shielded by oxygen, such as in oxyhemoglobin molecules, is imperceptible on SWI [6];

however, similar imaging appearance has been shown in developmental venous anomalies, cerebral venous thrombosis and Sturge-Weber syndrome; hence, SWI alone is not specific enough to rule out these conditions [7]. An explanation of the findings in the patients described might be a higher oxygen extraction in the neurons during the initial migraine episode, resulting in the presence of more deoxygenated blood discernible on SWI [1, 2]. Other authors have used additional imaging methods such as MR perfusion, observing hypoperfusion of the cerebral regions with decreased unilateral cerebral blood flow (CBF) and increased mean transit time (MTT). In all cases, the findings resolved in follow-up imaging [7, 8]. Moreover, unilateral cortical hypoperfusion in the aura episode and hyperperfusion during the headache episode has been reported using functional MRI. The diminished cerebral vascular autoregulation could probably explain the findings during these episodes [9]. The vascular abnormalities are also mentioned in the brief report by Fedak et al. supporting hypoperfusion as an etiologic factor for the SWI changes [10]. Interestingly, all the reports in the literature center on pediatric patients, which might be due to inherent statistical selection bias or differences in the pathophysiology of migraine between adult and pediatric patients. Subsequent studies featuring this distinction might contribute to the understanding of the pathophysiology of migraine.

Conclusion

The findings show a remarkable venous signal loss in SWI of the affected hemisphere in pediatric patients with migraine, supporting the evidence presented by the recently published case reports. Although the pathophysiologic concept behind these phenomena is not fully understood, it may support the vascular hypothesis of the disease. The inclusion of SWI in addition to DWI is useful in the assessment of these patients to rule out significant differential diagnoses, such as stroke. Also, it might have implications for the understanding of the pathophysiology of migraine with or without aura, although additional studies with broader population groups are needed.

Compliance with ethical guidelines

Conflict of interest N. Nersesyan, S. Arnold and P. Krieg declare that they have no competing interests.

Ethical standards All investigations described in this manuscript were carried out with the approval of the responsible ethics committee and in accordance with national law and the Helsinki Declaration of 1964 (in its current revised form). Informed consent was obtained from the patients in this case if identifiable from images or other information within the manuscript. In the case of the underage patients in this report, informed consent was obtained from the legal representatives.

References

1. Karaarslan E, Ulus S, Kürtüncü M. Susceptibility-weighted imaging in migraine with aura. *AJNR Am J Neuroradiol.* 2011;32:E5–7.
2. Gocmen R, Gunbey C, Arsava EM, Oguz KK, Haliloglu G. Susceptibility-weighted magnetic resonance imaging findings of two pediatric migraine patients with aura. *Neuropediatrics.* 2016;47:46–50.
3. Tottene A, Conti R, Fabbro A, Vecchia D, Shapovalova M, Santello M, van den Maagdenberg AM, Ferrari MD, Pietrobon D. Enhanced excitatory transmission at cortical synapses as the basis for facilitated spreading depression in Ca(v)2.1 knockin migraine mice. *Neuron.* 2009;61:762–73.
4. Adam G, Ferrier M, Patsoura S, Gramada R, Meluchova Z, Cazzola V, Darcourt J, Cognard C, Viguier A, Bonneville F. Magnetic resonance imaging of arterial stroke mimics: A pictorial review. *Insights Imaging.* 2018;9:815–31.
5. Mittal S, Wu Z, Neelavalli J, Haacke EM. Susceptibility-weighted imaging: Technical aspects and clinical applications, part 2. *AJNR Am J Neuroradiol.* 2009;30:232–52.
6. Haacke EM, Mittal S, Wu Z, Neelavalli J, Cheng YC. Susceptibility-weighted imaging: Technical aspects and clinical applications, part 1. *AJNR Am J Neuroradiol.* 2009;30:19–30.
7. Park MG, Yang TI, Oh SJ, Baik SK, Kang YH, Park KP. Multiple hypointense vessels on susceptibility-weighted imaging in acute ischemic stroke: Surrogate marker of oxygen extraction fraction in penumbra? *Cerebrovasc Dis.* 2014;38:254–61.
8. Bosemani T, Burton VJ, Felling RJ, Leigh R, Oakley C, Poretti A, Huisman TA. Pediatric hemiplegic migraine: Role of multiple MRI techniques in evaluation of reversible hypoperfusion. *Cephalalgia.* 2014;34:311–5.
9. Pollock JM, Deibler AR, Burdette JH, Kraft RA, Tan H, Evans AB, Maldjian JA. Migraine associated cerebral hyperperfusion with arterial spin-labeled MR imaging. *AJNR Am J Neuroradiol.* 2008;29:1494–7.
10. Fedak EM, Zumberge NA, Heyer GL. The diagnostic role for susceptibility-weighted MRI during sporadic hemiplegic migraine. *Cephalalgia.* 2013;33:1258–63.