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CASE REPORT

Susceptibility-weighted imaging and computed tomography perfusion abnormalities in diagnosis of classic migraine

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Abstract For the radiologist in the emergency department setting, the accurate diagnosis of acute neurologic change can be challenging—an incorrect or delayed diagnosis can lead to poor outcomes for the patient and potential medicolegal jeopardy. Conditions such as stroke, seizure, and infection are often first considered, as failure to promptly treat these entities can result in high morbidity or mortality. Migraine with aura is associated with neurologic change, most often visual in nature, including scotomas, visual field deficits, and visual hallucinations. However, any neurologic change can occur, including motor, sensory, or verbal deficits, which may mimic signs and symptoms of an acute stroke As neuroimaging is a part of the diagnostic and treatment decision-making process, the radiologist must be aware of the wide range of imaging findings of both common and uncommon etiologies for changes in neurologic status. In this paper, we present a case of an atypical presentation of migraine with aura diagnosed with susceptibility-weighted imaging and computed tomography perfusion.

Keywords Stroke · Migraine with aura · Susceptibility-weighted imaging · Computed tomography perfusion

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Introduction

Migraine headache is a common disorder resulting in severe unilateral headaches, often with associated photophobia, phonophobia, nausea, and vomiting. Approximately one third of migraines are preceded by an aura, an acute neurologic deficit—these are referred to as a "classic" migraine [1]. Neurologic symptoms are most often visual, including scotomas, visual field deficits, and visual hallucinations. However, any neurologic change can occur, including motor, sensory, or verbal deficits, which may mimic signs and symptoms of an acute stroke [2].

For these reasons, the classic migraine patient can represent a diagnostic challenge for not only the emergency physician but also for the radiologist, who must interpret the neuroimaging that almost invariably occurs shortly after these patients present in the emergency department.

In this article, we present a patient with stroke-like symptoms but with advanced imaging findings diagnostic of aura. It is critical that radiologists are aware of such findings so that patients receive accurate diagnosis, therapy, and prognosis.

Case report

A 22 year-old female was transferred to our hospital's emergency department as a "stroke alert" with expressive aphasia and intermittent receptive aphasia. Although medical history was pertinent for migraine headaches, including one episode associated with hemiplegia 2 years previously, there was still clinical concern for acute stroke. Her neurologic examination revealed no motor or sensory deficits. Vital signs were stable. Head computed tomography (CT) was ordered, and the patient was started on antiviral and antibacterial

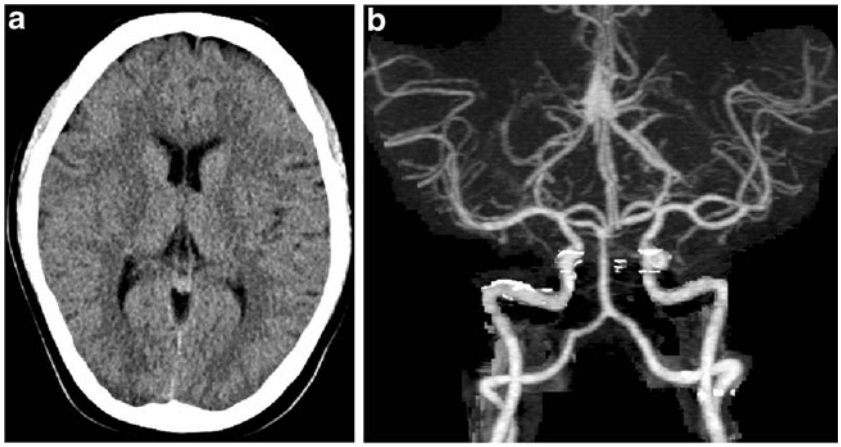

Fig. 1 **a** Normal unenhanced head CT. **b** Maximum intensity projection image of the intracranial CTA revealing no stenosis

agents due to concern for meningitis. Treatment was ultimately discontinued when cerebrospinal fluid cultures returned negative.

An unenhanced head CT and intracranial CT angiogram (CTA) were normal. Figure 1a and b shows normal unenhanced head CT, and maximum intensity projection image of the intracranial CTA reveals no stenosis. CT perfusion revealed diminished cerebral blood flow (CBF), increased mean transit time (MTT), and normal cerebral blood volume (CBV) in the entire left cerebral hemisphere. Figure 2a–c shows CT perfusion during aura, where CBF, CBV, and MTT maps reveal abnormally reduced CBF, normal CBV, and increased MTT throughout the entire left cerebral hemisphere, respectively. Findings were consistent with hypoperfusion. Magnetic resonance imaging (MRI) was performed to more definitively exclude an infarct.

The brain MRI revealed normal signal intensity of the brain parenchyma; specifically, there was no restricted diffusion, thus excluding an infarct. Figure 3a and b shows diffusion-weighted imaging (DWI), and apparent diffusion coefficient demonstrates no restricted diffusion in the region of perfusion abnormality. Susceptibility-weighted imaging (SWI) showed marked prominence of the venous vasculature of the left cerebral hemisphere in the same distribution as the CT perfusion defect. Figure 4a–c shows that SWI from inferior, middle, and superior portions of the brain demonstrate prominence of the venous vasculature within the left cerebral hemisphere.

Twenty-four to 30 h after admission, the patient had improved little, remaining globally aphasic. At this time, the patient was started on valproate and dexamethasone. The patient slowly began to recover, regaining her speech and cognitive abilities. Repeat CT perfusion 3 days after the initial imaging showed findings of hyperperfusion, including minimally increased CBV and decreased MTT in the left cerebral hemisphere. Figure 5a–c shows CT perfusion during headache, where CBF, CBV, and MTT maps reveal abnormally increased CBF, increased CBV, and decreased MTT throughout the entire left cerebral hemisphere, respectively. Findings are consistent with *hyper*perfusion. Based on the imaging findings and the patient's clinical course, a definitive diagnosis of classic migraine was made. She was discharged 3 days after admission with a normal neurologic examination.

Discussion

This case outlines the diagnostic difficulties that are sometimes encountered with a patient who presents with an acute

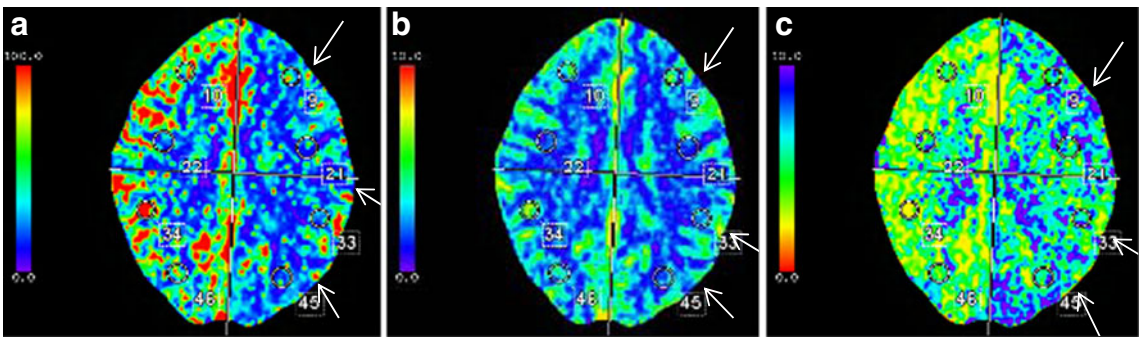

Fig. 2 CT perfusion during aura. **a** CBF, **b** CBV, and **c** MTT maps reveal abnormally reduced CBF, normal CBV, and increased MTT throughout the entire left cerebral hemisphere, respectively. Findings are consistent with hypoperfusion

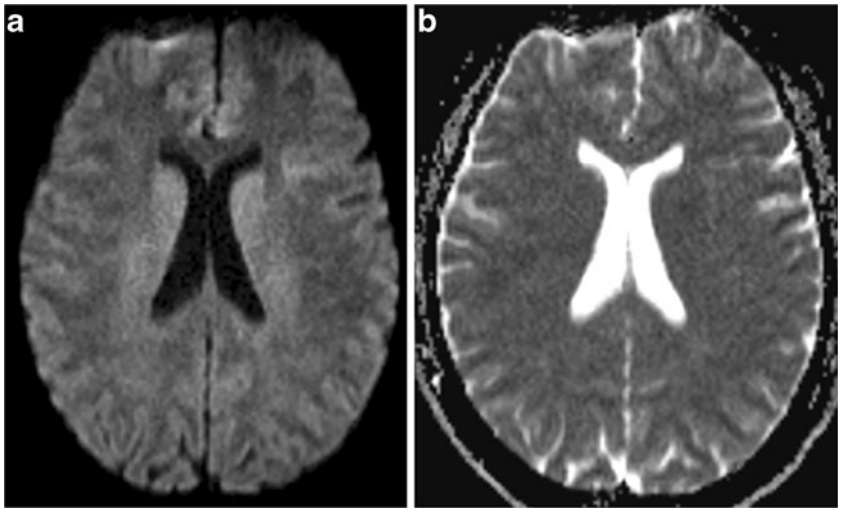

Fig. 3 Diffusion-weighted imaging (**a**) and apparent diffusion coefficient (**b**) demonstrate no restricted diffusion in the region of perfusion abnormality

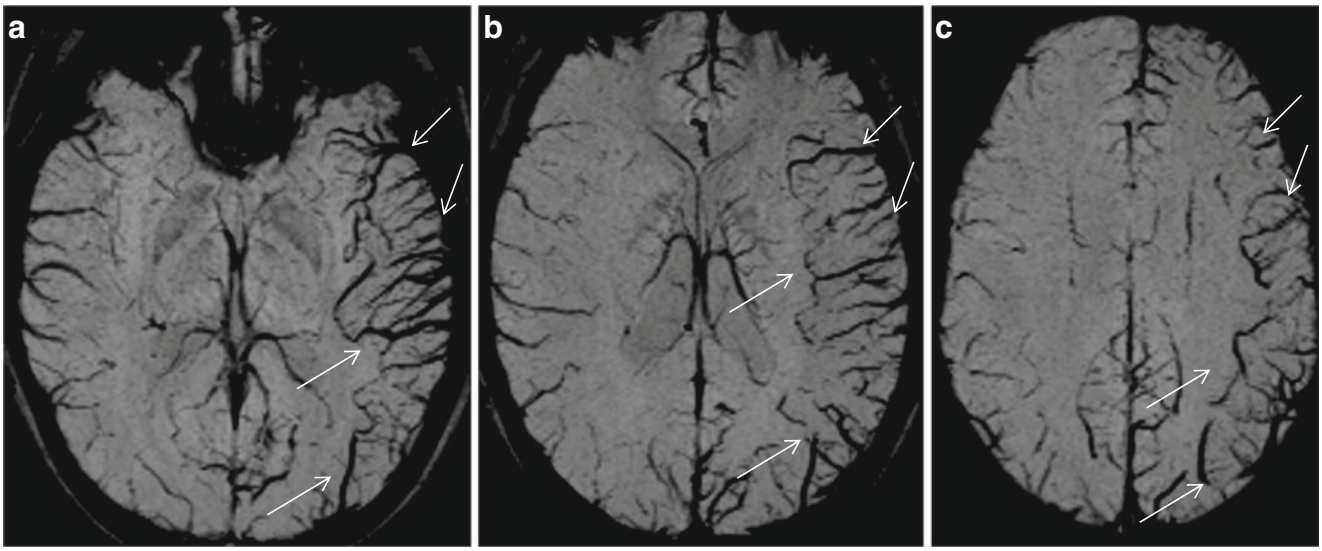

Fig. 4 SWI from **a** inferior, **b** middle, and **c** superior portions of the brain demonstrates prominence of the venous vasculature (*arrows*) within the left cerebral hemisphere

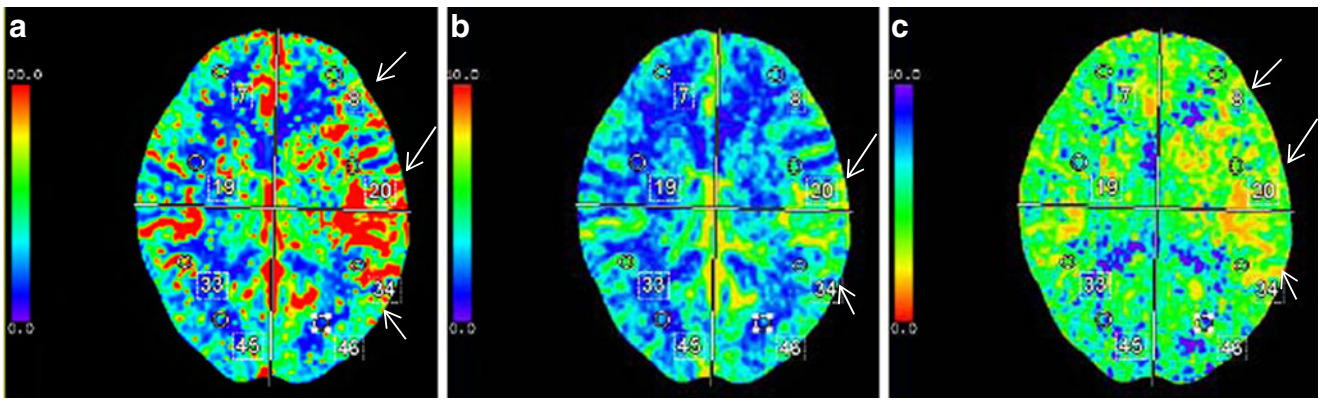

Fig. 5 CT perfusion during headache. **a** CBF, **b** CBV, and **c** MTT maps reveal abnormally increased CBF, increased CBV, and decreased MTT throughout the entire left cerebral hemisphere, respectively; findings are consistent with *hyper*perfusion

neurologic change. In the emergent setting, conditions such as stroke, seizure, and infection are often first considered, as failure to promptly treat these entities can yield poor outcomes. As neuroimaging is a part of the diagnostic and treatment decision-making process, the radiologist must be aware of the wide range of imaging findings of both common and uncommon etiologies for changes in neurologic status.

Classic migraine is defined as a migraine headache preceded by an aura. An aura is a negative or positive neurologic symptom that usually lasts for less than 60 minutes [3]. There are two main theories for the pathogenesis of migraines. The first suggests that migraines are primarily a vascular disorder, with vasoconstriction responsible for the aura and subsequent vasodilation resulting in the headache. It is thought the dilation of cranial arteries in the scalp, dura, and pia triggers the trigeminal pain fibers that innervate these vessels and causes the headache [4, 5]. The second major theory for the pathogenesis of migraines is based upon cortical spreading depression (CSD). It is thought that slowly propagating waves of depolarization in the brain followed by inhibition of the affected areas is what is responsible for the headache as well as the aura [6]. This is thought to be caused by altered cellular excitability which triggers the CSD. Studies have also shown propagated waves of blood flow and brain activity during migraine headaches similar to how CSD is described [7, 8].

Because of this patient's acute onset of aphasia, stroke was considered a diagnostic possibility. Although the normal unenhanced head CT excluded an intracranial hemorrhage, CT is relatively insensitive for early ischemia. The initial CT perfusion demonstrated a marked perfusion deficit in the left cerebral hemisphere, a pattern that has been described in the setting of aura. Assuming this patient was left hemisphere language-dominant, this left-sided hypoperfusion would explain the patient's aphasia. The lack of restricted diffusion on the MRI was definitive in excluding an infarct.

Our experience with the patient reported here is strongly supportive of the vascular theory. During this patient's aura, the CT perfusion showed findings of hypoperfusion, notably the increased MTT and decreased CBF. Three days later, the patient's aura and migraine symptoms had resolved. Repeat CT perfusion showed resolution of the hypoperfusion and development of hyperperfusion, likely secondary to vasodilation, supportive of the vascular theory.

The added neuroimaging tool that confirmed the diagnosis was the use of SWI on the MR imaging. To our knowledge, this is the first report to show reversibility of CT perfusion findings and SWI abnormalities in a classic migraine patient. SWI is a relatively new imaging technique that can be used to visualize deoxygenated blood, hemosiderin, ferritin, and calcium [9]. SWI has been described in serving a complimentary role to conventional MR imaging sequences in evaluating a variety of neurologic disorders, such as traumatic brain injury, hemorrhagic disorders, vascular malformations, cerebral infarction, neoplasms, and neurodegenerative disorders associated with calcium or iron deposition [10].

With its sensitivity for detection of deoxygenated blood, SWI can be used in the evaluation of venous structures. In this case, the prominent veins were seen in the hypoperfused regions of the brain, likely due to increase in deoxyhemoglobin. This increase in deoxyhemoglobin is thought to be secondary to blood demand outstripping supply. One flaw in the vascular theory is that these increased levels of deoxyhemoglobin can be seen in the headache after resolution of the vasoconstriction [11]. Further research will be needed to further characterize the significance of the prominence of the cerebral venous vasculature.

Conclusion

For the radiologist in the emergency department setting, the accurate diagnosis of acute neurologic change can be challenging—an incorrect or delayed diagnosis can lead to poor outcomes for the patient and potential medicolegal jeopardy.

The case presented here demonstrates the essential role that advanced imaging techniques, including CT perfusion, DWI, and SWI can play in excluding acute stroke and correctly diagnosing a classic migraine. To our knowledge, this is the first report to show reversibility of CT perfusion findings and SWI abnormalities in a classic migraine patient. Based on our experience as well as the limited number of reports that have addressed this topic, we feel that SWI should be incorporated into an institution's standard MR stroke protocol, especially if there are atypical features of the patient's clinical presentation. In conjunction with more widely used techniques, such as CT and CT perfusion, SWI will aid the radiologist in arriving at the correct diagnosis.

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