Acta Scientific Neurology (ASNE)

Volume 9 Issue 4 April 2026

Case Report

# Beyond Conventional Magnetic Resonance Imaging: Arterial Spin Labeling in Migraine with Aura

**Hinal Parmar[1]*, Sudhir Kothari[2], Deepak S Phalgune[3], Abhijit Wadekar[4] and Vivek Narayane[4]**

*[1]MBBS, DNB (Medicine), DrNB Neurology, Department of Neurology Poona Hospital and Research Centre, Pune, India*

*[2]MBBS, MD (Medicine), DM (Neurology), Professor and HoD, Department of Neurology, Poona Hospital and Research Centre, Pune, India*

*[3]MBBS, MD, PhD, Research Consultant, Department of Research, Poona Hospital and Research Centre, Pune, India*

*[4]MBBS, DNB (Medicine), Neurology Resident, Department of Neurology, Poona Hospital and Research Centre, Pune, India*

***Corresponding Author:** Hinal Parmar, MBBS, DNB (Medicine), DrNB Neurology, Department of Neurology Poona Hospital and Research Centre, Pune, India.

**DOI:** 10.31080/ASNE.2026.09.0906

**Received:** February 27, 2026

**Published:** March 31, 2026



## Abstract

Migraine with aura is associated with transient, dynamic alterations in cerebral perfusion related to cortical spreading depression, which may not be detected on conventional structural neuroimaging. Arterial spin labeling (ASL) magnetic resonance imaging (MRI) provides a non-invasive method for assessing these perfusion changes and can be particularly valuable in diagnosing and differentiating migraine with aura from other conditions. We report a 12-year-old boy who presented with exertion-triggered episodes of headache and transient focal neurological symptoms, initially raising diagnostic considerations of seizure, acute confusional migraine, and familial hemiplegic migraine.

The electroencephalogram (EEG) showed unilateral slowing. During a subsequent episode, ASL performed at presentation demonstrated unilateral cortical hypoperfusion, followed by hyperperfusion on repeat imaging the next day, with complete normalization of perfusion within one week. Structural MRI remained normal throughout, and genetic testing for familial hemiplegic migraine was negative. Based on the clinical course, the complete reversibility of symptoms, and the characteristic temporal perfusion changes observed on serial ASL imaging, a final diagnosis of migraine with aura was established. This case highlights the utility of serial ASL MRI in capturing dynamic perfusion changes and supporting the diagnosis of migraine with aura when conventional imaging is unrevealing.

**Keywords:** Aura; Arterial Spin Labeling; Electroencephalogram; Migraine; Unilateral Hypoperfusion

## Introduction

Migraine with aura is characterized by fully reversible focal neurological symptoms, most commonly visual, but also sensory, language, or motor disturbances, occurring in association with headache. These symptoms are attributed to cortical spreading depression, which leads to transient and dynamic alterations in cerebral perfusion that may not be detected on conventional structural magnetic resonance imaging (MRI) [1].

The clinical presentation of migraine with aura may overlap with other migraine variants, such as acute confusional migraine and hemiplegic migraine, including familial hemiplegic migraine, leading to diagnostic uncertainty, particularly in pediatric patients. Structural neuroimaging is frequently normal in these conditions, making functional assessment of cerebral blood flow potentially informative.

Arterial spin labeling (ASL) MRI is a noninvasive, contrast-free perfusion technique capable of demonstrating the characteristic temporal perfusion changes observed during migraine aura, including regional hypoperfusion followed by hyperperfusion. These dynamic patterns can assist in differentiating migraine with aura from other episodic neurological disorders when interpreted in conjunction with the clinical evolution [2].

## Case Report

A 12-year-old previously healthy boy, with no previous co-morbidities, presented with the acute onset of headache and vomiting, followed by sudden difficulty in speech. The episode began after exercising in a poorly ventilated gym without air conditioning. He reported feeling uneasy and diaphoretic, after which he returned home and developed a severe headache with two episodes of vomiting. Soon thereafter, he experienced difficulty finding words and was unable to name familiar persons or state his date of birth. He could not verbalize simple responses and relied on gestures to communicate. There was no history of prior headaches, seizures, trauma, fever, neck pain, or similar episodes. There was no prior personal history of recurrent headaches or migraine. However, his mother had a history of migraine without aura. There was no family history of hemiplegic migraine, epilepsy, or other neurological disorders. His birth history, developmental milestones, and academic performance were normal.

He was initially evaluated at another hospital, where magnetic resonance imaging (MRI) of the brain and magnetic resonance angiography were reported as normal (approximately 3 hours from symptom onset), while the electroencephalogram (EEG) showed left-sided slowing. He was referred to Poona Hospital and Research Centre (PHRC), a tertiary care center, approximately five hours after symptom onset. So, first MRI was done in another centre around three hours from symptom onset. ASL was not done at that time.

On arrival, his speech had largely normalized, with residual mild left temporal headache and partial recollection of the event. Neurological examination revealed mild drowsiness and irritability, without focal motor, sensory, or cranial nerve deficits. The electroencephalogram (EEG) performed at our center demonstrated gross left hemispheric slowing (Figure 1), suggestive of focal cerebral dysfunction, which showed partial improvement on repeat recording the following day. The second MRI done in our institute was done at 5 hours after symptom onset and it was also normal (Figure 2). The patient recovered fully and became asymptomatic by the next day. He was discharged on flunarizine with a provisional diagnosis of acute confusional migraine. We advised an ASL MRI, but the patient's family took a discharge and did it on an outpatient basis. This MRI ASL study was done on day five and was normal (Figure 3). All MRI studies were performed on 3-Tesla scanners. Images were acquired with Post Label Delay (PLD) of 2525 ms, and quantitative cerebral blood flow(qCBF) maps were calculated using a standard perfusion model.

On day six, the patient experienced a second episode following football play in the sun and subsequent exposure to an air-conditioned classroom. He developed bilateral tunnel vision, with an inability to see parts of the blackboard, lasting approximately 30 minutes, followed by a headache beginning five minutes after the onset of visual symptoms. After resolution of the visual symptoms, he experienced tingling and numbness in the left upper and lower limbs, along with mild dragging of the left leg while walking. There was no speech disturbance during this episode. He presented to PHRC within three hours of symptom onset. Repeat MRI of the brain was normal, whereas ASL MRI performed approximately 3 hours after symptom onset demonstrated right hemispheric hypoperfusion (Figure 4). The EEG showed right-sided slowing (Figure 5). All symptoms resolved completely within four hours.

This time, EEG slowing was on the right side, and ASL also showed hypoperfusion on the right side, unlike the first time, when the EEG had shown left-sided slowing but ASL was normal. Note that the earlier ASL was done on day five, while the ASL this time was done within three hours of onset.

We repeated the ASL MRI the following day, 24 hours after the onset of symptoms, showed instead of the right-sided hypoperfusion seen the previous day, there was right-sided hyperperfusion (Figure 6). One week after the attack, the ASL perfusion MRI was normal, as was the EEG (Figure 7 and 8, respectively).

Considering the possibility of familial hemiplegic migraine, genetic testing was advised; the results were negative. He was treated as having migraine with aura and managed conservatively with migraine prophylaxis using flunarizine, along with lifestyle modifications, including avoidance of exertional triggers and dehydration. No further neurological deficits were reported during follow-up.

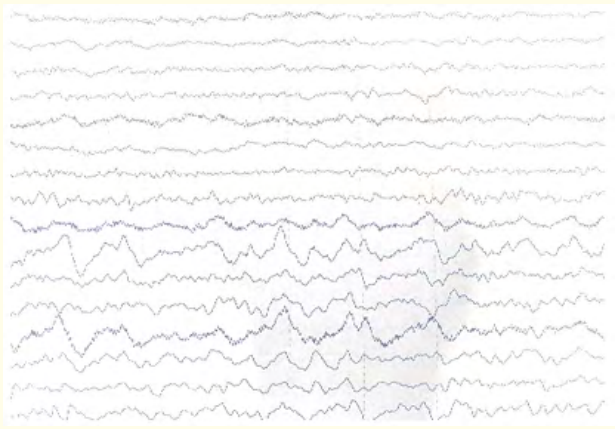
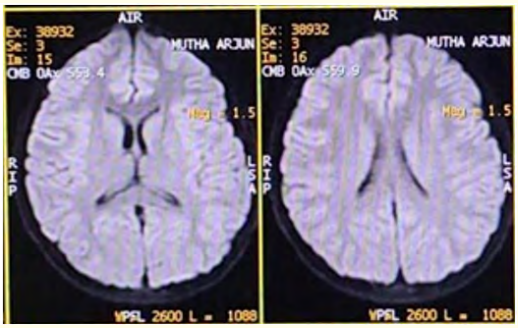


**Figure 1:** Electroencephalogram within 3 hours of first attack shows left hemispheric slowing.



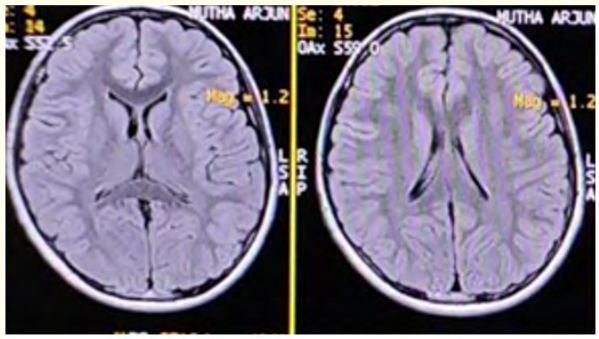




**Figure 2:** Normal MRI brain FLAIR image done at 5 hours after symptom onset in 1st attack.

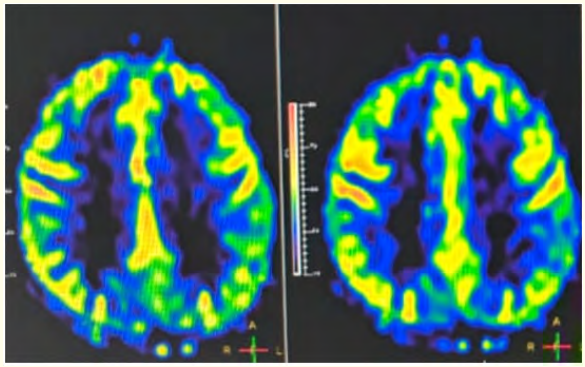


**Figure 3:** ASL MRI image on day 5 which is normal.

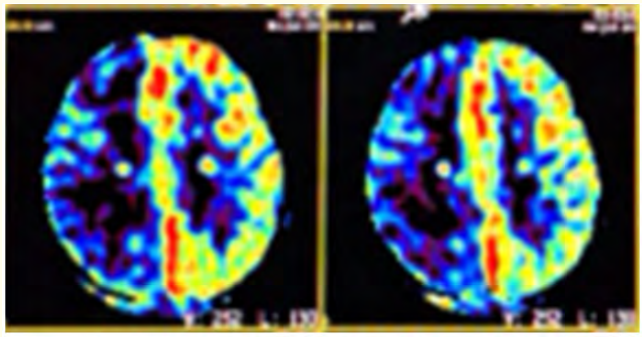


**Figure 4:** ASL MRI image in 2nd attack within 3 hours of symptom onset shows right hemispheric hypoperfusion.

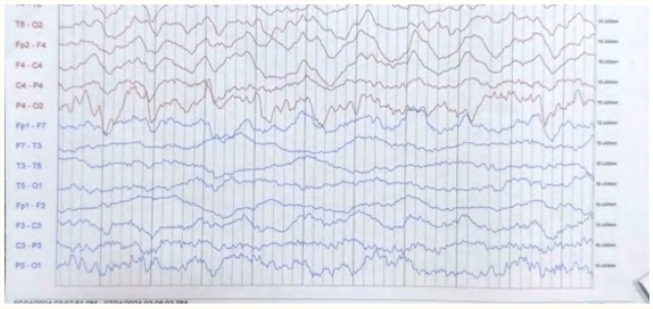


**Figure 5:** Electroencephalogram in 2nd attack around 3 hours of symptom onset shows right hemispheric slowing.

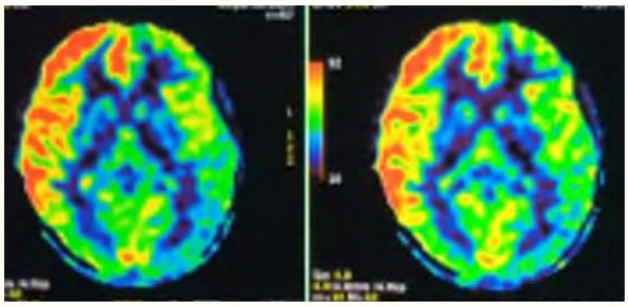


**Figure 6:** ASL MRI image in 2nd attack after 24 hours of symptom onset shows right hemispheric hyperperfusion.

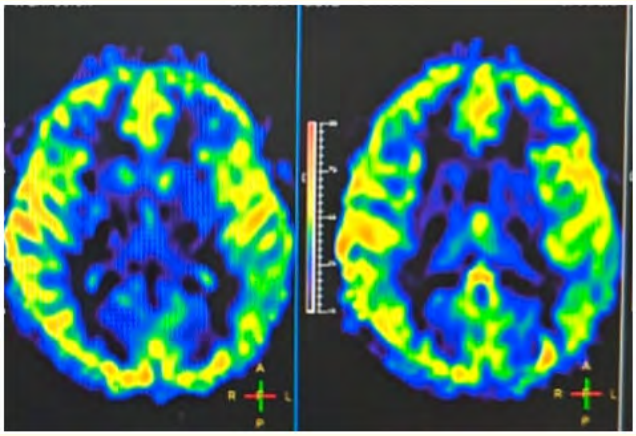


**Figure 7:** ASL MRI image on day 7 of second attack is normal.

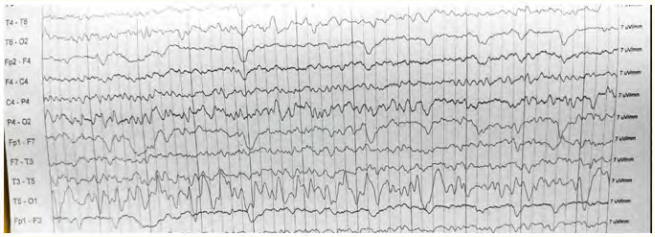


**Figure 8:** Electroencephalogram on day 7 of 2nd attack- normal.

## Discussion

Migraine with aura is a complex neurovascular disorder characterized by transient focal neurological symptoms resulting from cortical spreading depression (CSD), a self-propagating wave of altered neuronal activity and cerebral blood flow across the cortex. CSD has been implicated as the underlying mechanism of aura and is associated with dynamic perfusion changes, including early regional hypoperfusion followed by relative hyperperfusion as the episode evolves [3]. Conventional structural MRI often remains normal during migraine-associated vertigo attacks, thereby limiting its diagnostic utility in capturing these transient hemodynamic alterations.

ASL MRI is a quantitative, non-contrast perfusion technique that measures cerebral blood flow by magnetically labelling arterial water protons. Unlike traditional perfusion methods that require contrast agents, ASL can be safely repeated and is particularly suitable for paediatric populations and for assessing transient neurological phenomena [4]. Recent literature reviews demonstrate that ASL can detect perfusion abnormalities during aura, with studies showing concordant patterns of hypoperfusion corresponding to clinical symptoms in early phases and subsequent hyperperfusion in the later stages of the attack cycle [2]. These dynamic perfusion patterns are not confined to typical visual aura but have been observed across multiple cortical regions involved in multisensory processing and aura propagation [3].

In this case, the patient presented with exertion-triggered episodes of transient language dysfunction, visual symptoms, and unilateral sensorimotor deficits, all of which resolved completely. The evolution of symptoms, beginning with visual disturbance, followed by sensory phenomena and headache, was consistent with the typical sequential pattern of migraine aura. However, the presence of focal neurological deficits and unilateral EEG slowing

raised consideration of other diagnoses, including seizure-related phenomena, transient ischemic attack, acute confusional migraine, and familial hemiplegic migraine.

Several alternative diagnoses were considered. Seizure with post-ictal deficits was unlikely because EEG recordings during both episodes showed only focal slowing without epileptiform discharges, and the clinical events lacked features typical of focal seizures, such as automatisms or impaired awareness preceding the deficits. Transient ischemic attack was also unlikely given the patient's young age, absence of vascular risk factors, complete reversibility of symptoms, and normal vascular imaging. Familial hemiplegic migraine was considered due to the transient motor symptoms; however, there was no family history of similar attacks, and genetic testing was negative. The sequential progression of visual symptoms followed by sensory and headache manifestations, along with the characteristic perfusion evolution on ASL imaging, strongly supported migraine with aura.

The EEG during both episodes demonstrated focal hemispheric slowing without epileptiform discharges, supporting transient cortical dysfunction rather than epileptic activity. The resolution of slowing on repeat EEG further reinforced the reversible and functional nature of the process.

The most distinctive feature of this case was the dynamic perfusion pattern demonstrated on serial ASL MRI. During the acute phase of the second episode, ASL revealed unilateral hemispheric hypoperfusion corresponding to the patient's neurological deficits. Repeat imaging the following day demonstrated hyperperfusion in the same region, with complete normalization after one week. This temporal sequence of hypoperfusion followed by hyperperfusion aligns with the hemodynamic evolution described in migraine aura and reflects the vascular changes associated with cortical spreading depression. Importantly, structural MRI remained normal throughout, underscoring the additional diagnostic value of perfusion imaging.

This case underscores the importance of integrating clinical evolution, EEG findings, and serial perfusion imaging when evaluating patients with acute focal neurological deficits. Recognition of migraine with aura in such presentations is essential to prevent unnecessary investigations, reduce anxiety among patients and families, and avoid inappropriate interventions. Dynamic ASL perfusion patterns, interpreted in conjunction with symptom progression and reversibility, can serve as a valuable adjunct in establishing the diagnosis of migraine with aura when conventional imaging is unrevealing.

## Conclusions

This case highlights migraine with aura as a dynamic neurovascular disorder capable of presenting with transient focal neurological deficits despite normal structural neuroimaging. Serial ASL MRI demonstrated a characteristic evolution from unilateral hypoperfusion to hyperperfusion, followed by subsequent normalization, providing objective physiological evidence of reversible cortical dysfunction. When interpreted alongside the clinical progression and transient EEG slowing, ASL significantly strengthened diagnostic confidence.

In pediatric patients presenting with acute focal deficits, integration of clinical assessment, electrophysiology, and serial perfusion imaging can facilitate accurate diagnosis of migraine with aura and help avoid unnecessary investigations or interventions.

## Bibliography

1. Karsan N., *et al*. "Evaluating migraine with typical aura with neuroimaging". *Frontiers in Human Neuroscience* 17 (2023): 1112790.

2. Schramm S., *et al*. "Perfusion imaging by arterial spin labeling in migraine: A literature review". *Journal of Cerebral Blood Flow and Metabolism* 44.8 (2024): 1253-1270.

3. Russo A., *et al*. "Arterial spin labeling MRI applied to migraine: current insights and future perspectives". *The Journal of Headache and Pain* 24.1 (2023): 71.

4. Haller S., *et al*. "Arterial spin labeling perfusion of the brain: emerging clinical applications". *Radiology* 281.2 (2016): 337-356.