



## Cerebral blood flow in a case of typical aura without headache

Miriam Sansone<sup>1</sup> · Guido Marinelli<sup>2</sup> · Emanuela Piccotti<sup>2</sup> · Mariasavina Severino<sup>3</sup> · Lino Nobili<sup>1,4</sup>

Received: 30 May 2019 / Revised: 24 July 2019 / Accepted: 26 July 2019 / Published online: 2 August 2019  
© Springer-Verlag GmbH Germany, part of Springer Nature 2019

Dear Sirs,

F., a girl of 10 years was admitted to the emergency room (ER) for a sudden onset of confusional state and difficulties in speaking. Prior to ER admission, the patient had paresthesia of the left hemi-face and felt discomfort in her eyes. In the ER, the patient looked extremely confused and upset although she recovered from such a state and the difficulties in speaking in about half an hour. The overall past medical history was negative with no cases of migraine and epilepsy in the family. All performed blood tests (CBC, coagulation, ionogram, inflammatory indices) turned out negative. The neurological examination was negative. The parents of the patient pointed out that their daughter was always very emotional. They also claimed that, during the last month, the patient developed sleep disorders and anxiety due to worries about a schoolmate with serious health problems as well as a cousin who was diagnosed with Hodgkin's lymphoma. Although a functional disorder was anticipated, the neurologist did not rule out a migraine with aura. A decision was made to keep the patient in emergency medicine.

The following day, the patient underwent an EEG during wakefulness which revealed “monomorphic slow waves in the left occipital region” (Fig. 1). The neurological examination detected a campimetric deficit (lateral right homonymous quadrantanopsia) that was previously unnoticed. An urgent MRI ruled out the presence of a brain lesion such as an acute ischemic infarct. Arterial MR angiography was

normal. However, 3D pseudocontinuous arterial spin labeling (3D pCASL) MR perfusion images revealed increased signal, consistent with hyperperfusion, in the posterior temporal and left parietal–occipital areas (Fig. 2). Taken together, the clinical, neuroimaging and EEG data were interpreted as a manifestation of an aura without headache.

After 3 days, a further neurological examination gave negative results with absence of campimetric deficits and a normalized EEG. The patient was then definitively discharged with a diagnosis of typical aura without headache (TAWH) and referred to the Center for Headache. After approximately 1 month, the patient developed a tension-type headache that was not accompanied by other symptoms.

Since the clinical expression of symptoms varies in pediatric patients, clinicians are often left to face issues of differential diagnosis between different conditions such as essential headache, functional disorder, epileptic manifestations or symptoms related to brain lesions. Therefore, neuroimaging is of great importance to rule out the presence of ischemic or haemorrhagic infarcts, space-occupying lesions or inflammatory disorders. Arterial spin labelling (ASL) is a new magnetic resonance imaging technique which assesses brain perfusion without contrast injection. In accordance with previous observations [1–4], recent findings using ASL have suggested that cerebral blood flow is decreased during aura, if performed early after the onset of the symptoms and that regional cortical hyperperfusion, induced by spreading depolarization [5], is observed during the headache phase [6, 7].

Indeed, a study conducted in 17 children with migraine and acute neurological deficit showed hypoperfusion of one or more cerebral lobes in 94% of the cases using ASL [8].

In this case study, the use of ASL revealed hyperperfusion in the posterior temporal and left parietal–occipital areas which are consistent with hyperperfusion of post-aura migraine. The novelty of our case consists in the fact that we identified in a pediatric patient with TAWH the same neuroimaging alterations of patients with migraine with aura. This case report confirms that a non-invasive technique such as

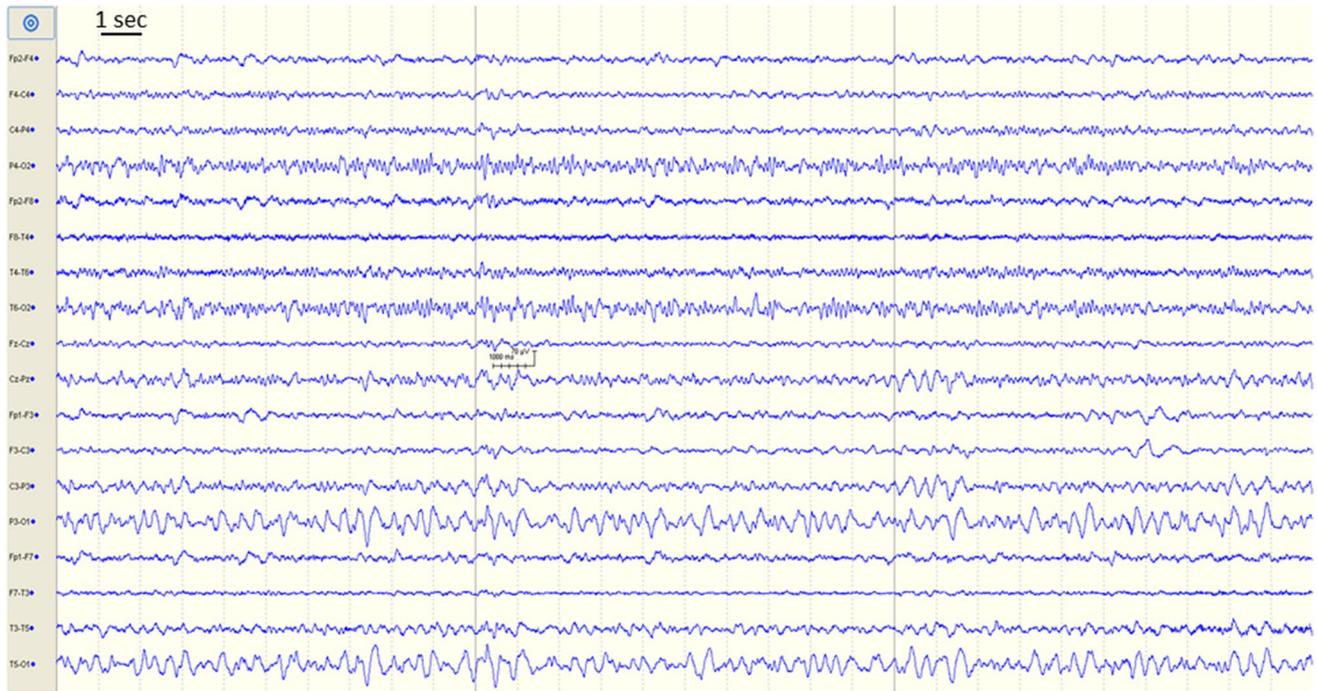
✉ Lino Nobili  
lino.nobili@unige.it

<sup>1</sup> Child Neuropsychiatry Unit, IRCCS G. Gaslini Children's Hospital, Genoa, Italy

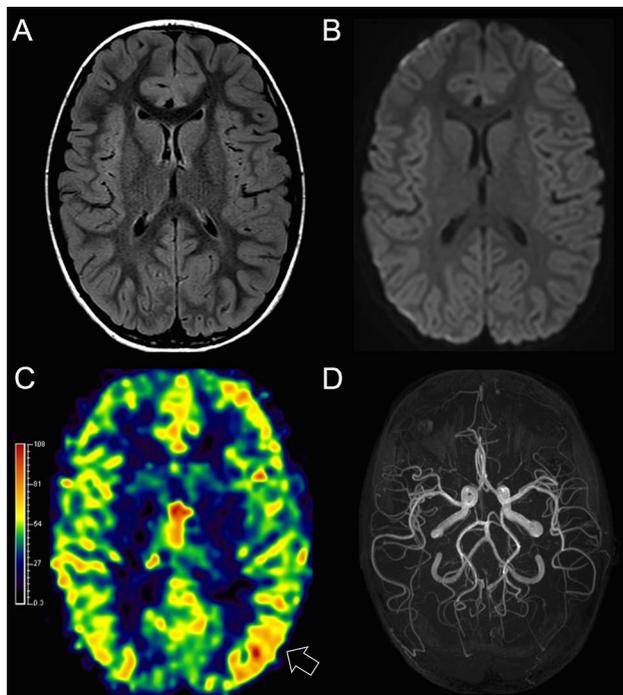
<sup>2</sup> Department of Pediatric Emergency, IRCCS G. Gaslini Children's Hospital, Genoa, Italy

<sup>3</sup> Neuroradiology Unit, IRCCS G. Gaslini Children's Hospital, Genoa, Italy

<sup>4</sup> Department of Neuroscience, Ophthalmology, Genetics and Maternal Infantile Sciences (DINOGLI), University of Genoa, Genoa, Italy



**Fig. 1** EEG trace showing the presence of continuous slow waves in the left occipital region



**Fig. 2** Brain MRI (a–c) and arterial MR angiography (d) performed about 20 h after clinical onset. Axial FLAIR (a) and diffusion-weighted (b) images demonstrate absence of brain lesions, including cytotoxic oedema. Corresponding 3D pCASL perfusion image (c) shows an area of increased signal consistent with hyperperfusion in the left temporo-parietal regions (arrow). Arterial MR angiography with time-of-flight technique (d) reveal normal flow signal at the level of intracranial arteries

ASL can be successfully used to provide important information about challenging cases.

### Compliance with ethical standards

**Conflicts of interest** On behalf of all authors, the corresponding author states that there is no conflict of interest.

**Ethical approval** All procedures performed in the study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

### References

- Olesen J, Larsen B, Lauritzen M (1981) Focal hyperemia followed by spreading oligemia and impaired activation of rCBF in classic migraine. *Ann Neurol* 9:344–352
- Lauritzen M (1994) Pathophysiology of the migraine aura: the spreading depression theory. *Brain* 117:199–210
- Woods RP, Iacoboni M, Mazziotta JC (1994) Brief report: bilateral spreading cerebral hypoperfusion during spontaneous migraine headache. *N Engl J Med* 331:1689–1692
- Hadjikhani N, Del Sanchez RM, Wu O, Schwartz D, Bakker D, Fischl B, Kwong KK, Cutrer FM, Rosen BR, Tootell RB, Sorensen AG, Moskowitz MA (2001) Mechanisms of migraine aura revealed by functional MRI in human visual cortex. *Proc Natl Acad Sci* 98:4687–4692
- Dreier JP, Reiffurth C (2015) The stroke migraine depolarization continuum. *Neuron* 86:902–922

6. Boulouis G, Shotar E, Dangouloff-Ros V, Grévent D, Calmon R, Brunelle F, Naggara O, Kossorotoff M, Boddaert N (2016) Magnetic resonance imaging arterial-spin-labelling perfusion alterations in childhood migraine with atypical aura: a case-control study. *Dev Med Child Neurol* 58:965–969
7. Pollock JM, Deibler AR, Burdette JH, Kraft RA, Tan H, Evans AB, Maldjian JA (2008) Migraine associated cerebral hyperperfusion with arterial spin-labelled MR imaging. *AJNR* 29:1494–1497
8. Cadiot D, Longuet R, Bruneau B, Treguier C, Carsin-Vu A, Corouge I, Gomes C, Proisy M (2018) Magnetic resonance imaging in children presenting migraine with aura: association of hypoperfusion detected by arterial spin labelling and vasospasm on MR angiography findings. *Cephalalgia* 38:949–958