

## Ictal focal hyperperfusion demonstrated by arterial spin-labeling perfusion MRI in partial epilepsy status

Makoto Oishi · Go Ishida · Ken Morii ·  
Kenji Hasegawa · Mitsuya Sato · Yukihiro Fujii

Received: 29 February 2012 / Accepted: 7 March 2012 / Published online: 16 March 2012  
© Springer-Verlag 2012

Dear Sir,

Pulsed arterial spin-labeling (PASL) perfusion magnetic resonance imaging (MRI) is a noninvasive method of measuring regional cerebral blood flow (rCBF) without contrast medium and has been routinely applied in cerebrovascular diseases as a cerebral perfusion measurement [1]. In partial epilepsy, ictal cortical hyperperfusion is believed to be a useful marker for identifying epileptogenic zone, and single-photon emission computed tomography (SPECT) is commonly used for evaluating differences between interictal and ictal cerebral perfusions. Few reports have described hemodynamic changes detected by PASL in interictal [2–4] and peri-ictal states [5, 6]. Recently, we had a rare opportunity to conduct PASL imaging and also diffusion-weighted imaging (DWI) of a patient both in partial epilepsy status (PES) and during the interictal state.

---

M. Oishi · Y. Fujii  
Department of Neurosurgery, Brain Research Institute,  
Niigata University,  
Niigata, Japan

G. Ishida  
Department of Clinical Radiology,  
Shinjinkai Kita-Nihon No-Shinkei Geka Hospital,  
Niigata, Japan

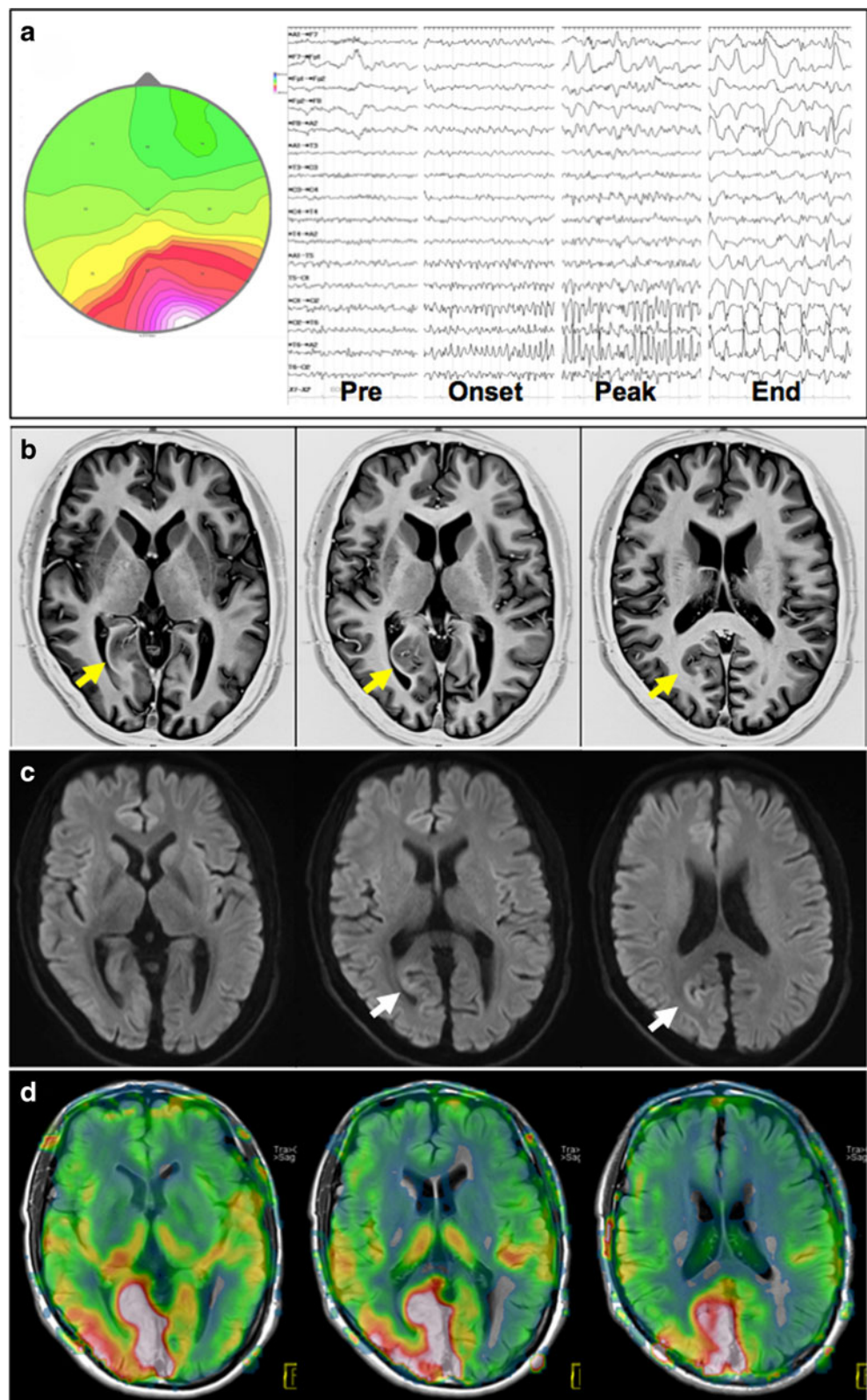
M. Oishi · K. Morii · K. Hasegawa · M. Sato  
Department of Neurosurgery,  
Shinjinkai Kita-Nihon No-Shinkei Geka Hospital,  
Niigata, Japan

M. Oishi (✉)  
Department of Neurosurgery, Brain Research Institute,  
Niigata University,  
1-757 Asahimachidori, Chuo-Ku,  
Niigata 951-8585, Japan  
e-mail: mac.oishi@me.com

Our patient, a 25-year-old man with normal development and no neurological deficits, had sometimes experienced a short-term feeling of visual distortion in the left half of his visual field since age 20 years. He had his first generalized convulsion and was admitted to our hospital. One week later, he complained of frequent occurrences of blurred vision or elementary visual hallucinations like flickering lights in the left half of his visual field, sometimes accompanied by rotating his head to the left. An electroencephalogram (EEG) recorded three ictal events during 30 min. The ictal pattern showed rhythmic bursting epileptic activities arising from the right occipital region, gradually reaching high amplitude and spreading to surrounding areas within approximately 2 min and then finished without generalizing (Fig. 1a). He presented with seizures at a frequency of five to six times per hour in the daytime, so-called PES. After starting carbamazepine administration, the seizures gradually decreased and had disappeared 3 days later. He experienced left visual field loss for a few days even after the seizures had disappeared but ultimately recovered from this deficit.

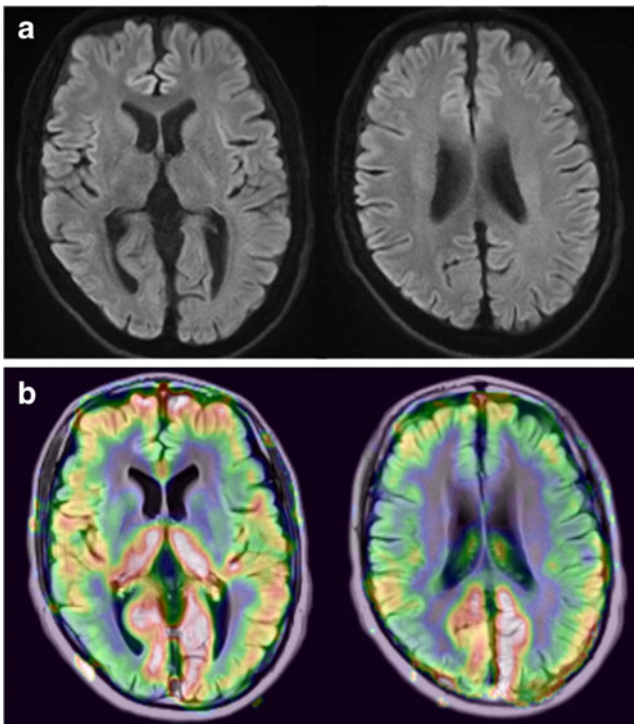
MRI scans were performed on a 3 T MRI system (MAGNETOM Verio; Siemens AG, Munich, Germany) on the first day that frequent seizures occurred (Fig. 1b–d) and 1 week after the seizures had completely disappeared (Fig. 2). Cortical evaluation by reversed Short T1 inversion recovery (STIR) imaging indicated an anomaly consistent with a cortical malformation in the right medial occipital region (Fig. 1b). PASL was performed with a pulsed sequence, using QUIPSII perfusion mode [7] and the following parameters: nine slices; FOV read, 300.0 mm; FOV phase, 206.0; slice thickness, 10.0 mm; TE/TR/T11/T12=20/3,500/800/2,200 ms. PASL results were coregistered with FLAIR images. While focal hyperintense signals on DWI were observed in the limited area of the

**Fig. 1** Examinations during partial epilepsy status. Scalp EEG during one seizure (**a**) showed a focal seizure pattern in the right occipital region. Reversed Short T1 inversion recovery (STIR) imaging (**b**) shows a focal cortical anomaly in the right medial occipital cortex (*yellow arrows*). Diffusion-weighted imaging (DWI) (**c**) shows hyperintense signals in the focal cortical area (*white arrows*) corresponding to the abnormal cortex on STIR. Pulsed arterial spin-labeling imaging (**d**) demonstrates strong cortical hyperperfusion in the medial to lateral occipital cortices, much more widely distributed than the abnormal signal alteration on DWI



cortical lesion during PES, PASL demonstrated strong and extensive hyperperfusion in the right occipital lobe with the center at the cortical lesion (Fig. 1c, d). After the seizures had completely disappeared, hyperintense

signals on DWI also disappeared and PASL demonstrated instead hypoperfusion of the cortical lesion in the right medial occipital area as compared to the same region of the contralateral side (Fig. 2).



**Fig. 2** MRI in the interictal state. Hyperintense signals on diffusion-weighted imaging during partial epilepsy status have completely disappeared (**a**). Pulsed arterial spin-labeling imaging (**b**) also shows a change to relative hypoperfusion in the area presenting a cortical anomaly, as compared with the same region in the contralateral hemisphere

Some authors have investigated the utility of focal interictal perfusion abnormalities shown by PASL as a marker for localizing the epileptic focus in partial epilepsy [2–4]. They observed significant correlations between the interictal hypoperfusion demonstrated by PASL and the results of positron emission tomography or SPECT imaging in the ictal period. We found only two reports presenting PASL studies relating to the peri-ictal state; one was obtained immediately after the seizure [5], while in the other, the seizure incidentally occurred while scanning was ongoing, immediately after the scanning the PASL sequence [6]. Although such peri-ictal findings were valuable, perfusion alterations on PASL imaging between peri-ictal and interictal conditions had relatively little impact in achieving visual distinction unless a statistical analysis was conducted [5]. In our case, PASL imaging during PES showed strong focal hyperperfusion of the region corresponding to the anatomical and physiological epileptic focus and also obtained clear contrast to the relative hypoperfusion in the interictal state. To our knowledge, this is the first clear demonstration of focal hyperperfusion relating to PES by PASL imaging. Furthermore, a discrepancy between cortical extents of perfusion alterations and cytotoxic changes in PES, based on comparing PASL and DWI findings, was also obtained.

In epilepsy status presenting as repeated or continuous seizures, the epileptic cortical areas should be in an electrophysiologically extreme state consisting of seizure-induced vascular alterations and neuroinflammation which play roles in persistent epileptogenesis. The prolonged and extraordinary activation of neurons in the affected area increases glucose and oxygen requirements, thereby causing compensatory regional hyperperfusion. When the hyperperfusion is no longer sufficient to supply the hyperactive cortical area, pathophysiological changes leading to cytotoxic edema will occur and are known to appear as an abnormal signal on DWI [8, 9]. The MRI findings of low apparent diffusion coefficient (ADC) and high signals on DWI in status epilepticus can resemble those of acute ischemic stroke, indicating changes attributable to both cytotoxic and vasogenic edema [8–11]. The spatiotemporal correlation of regional changes on DWI and electroclinical findings indicates that the transient hemodynamic alteration is dependent on electrical activity during prolonged epileptic activity [8–11]. Although such ictal changes on DWI were reversible in most cases on follow-up imaging studies and there were no permanent neurological deficits, they occasionally resulted in regional brain atrophy [9]. Predicting irreversible change from the radiological findings in PES is still controversial. It is obviously necessary to ascertain the critical border of perfusion alterations causing the vasogenic and cytotoxic changes shown on DWI. The large and clear discrepancy between the focal signal change on DWI and strong hyperperfusion on PASL imaging during PES in our patient was striking, and further systematic analyses of similar experiences are anticipated to resolve this critical issue.

**Conflict of interest** We declare that we have no conflict of interest.

## References

1. Wolf RL, Detre JA (2007) Clinical neuroimaging using arterial spin-labeled perfusion magnetic resonance imaging. *Neurotherapeutics* 4(3):346–359
2. Lim YM, Cho YW, Shamim S et al (2008) Usefulness of pulsed arterial spin labeling MR imaging in mesial temporal lobe epilepsy. *Epilepsy Res* 82(2–3):183–189
3. Pendse N, Wissmeyer M, Altrichter S et al (2010) Interictal arterial spin-labeling MRI perfusion in intractable epilepsy. *J Neuroradiol* 37(1):60–63
4. Wolf RL, Alsop DC, Levy-Reis I et al (2001) Detection of mesial temporal lobe hypoperfusion in patients with temporal lobe epilepsy by use of arterial spin labeled perfusion MR imaging. *AJNR Am J Neuroradiol* 22(7):1334–1341
5. Pizzini F, Farace P, Zanoni T, Magon S, Beltramello A, Sbarbati A, Fabene PF (2008) Pulsed-arterial-spin-labeling perfusion 3 T MRI following single seizure: a first case report study. *Epilepsy Res* 81(2–3):225–227

6. Toledo M, Munuera J, Salas-Puig X, Santamarina E, Lacuey N, Rovira A (2011) Localisation value of ictal arterial spin-labelled sequences in partial seizures. *Epileptic Disord* 13(3):336–339
7. Luh WM, Wong EC, Bandettini PA, Hyde JS (1999) QUIPSS II with thin-slice T11 periodic saturation: a method for improving accuracy of quantitative perfusion imaging using pulsed arterial spin labeling. *Magn Reson Med* 41(6):1246–1254
8. Cole AJ (2004) Status epilepticus and periictal imaging. *Epilepsia* 45(Suppl 4):72–77
9. Lansberg MG, O'Brien MW, Norbash AM, Moseley ME, Morrell M, Albers GW (1999) MRI abnormalities associated with partial status epilepticus. *Neurology* 52(5):1021–1027
10. Di Bonaventura C, Bonini F, Fattouch J et al (2009) Diffusion-weighted magnetic resonance imaging in patients with partial status epilepticus. *Epilepsia* 50(Suppl 1):45–52
11. Heiniger P, Koussy M, Schindler K et al (2002) Diffusion and perfusion MRI for the localisation of epileptogenic foci in drug-resistant epilepsy. *Neuroradiology* 44(6):475–480