

Letter to Editor

Transient neocortical MRI abnormalities following initial epileptic seizure



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Dear Editor,

Seizure-induced transient MRI abnormalities (TMA) in the brain were first described by Kramer et al. [1] in 1987. Since then, several other researchers have reported MRI signal alterations following focal or generalised status epilepticus.

There are also rare cases of these alterations being described after an isolated seizure in patients with known epilepsy and even in patients experiencing a first simple or complex partial seizure [2–6].

We present the case of a patient with no history of epilepsy who displayed a transient focal lesion on brain MR images after experiencing 2 complex partial seizures with secondary generalisation.

Our patient was a right-handed 37-year-old man with no relevant medical history except for head trauma a few years previously in his country of origin; he was a former boxer. He reported no history of neonatal disease, meningoencephalitis, or febrile convulsions, and had no family history of epilepsy.

Coinciding with a period of stress and sleep deprivation, our patient experienced a sudden episode of rigidity of all 4 limbs with clonic movements during approximately 2 min while sleeping. According to a witness, the episode was characterised by stertorous breathing, tongue biting, and bladder incontinence, followed by postictal confusion. The patient experienced another nocturnal episode with identical characteristics 7 days later. The patient reported that seizures were preceded by a vaguely described sensation of cephalic deviation.

General and neurological examinations yielded normal results. Results from laboratory blood and urine tests, electrocardiography, chest radiography, and a brain CT scan were normal. Interictal EEG revealed no abnormalities. After the second episode, and while deliberating a diagnosis of partial epilepsy with secondary generalisation, we started treatment with valproic acid dosed at 1500 mg/day. A 1.5-T MRI brain scan performed on an out-patient basis a month after the first episode revealed hyperintensities in the left temporal cortical area on FLAIR sequences, with no contrast uptake (Fig. 1, top). A follow-up 3-Tesla MRI scan performed at 6 months revealed complete resolution of the lesion (Fig. 1, below). An additional 3-Tesla MRI scan performed at 18 months revealed no abnormalities; the patient has remained asymptomatic to date, which confirms favourable progression.

MRI signal changes during status epilepticus and isolated seizures are a consequence of physiological changes occurring during brain seizure activity; these include rupture of the blood-brain barrier and increased blood flow in the epileptogenic focus, ultimately leading to cerebral oedema [6]. These changes in cerebral parenchyma are usually observed in cortical grey matter, although they may present with more heterogeneous patterns and affect a variety of localisations. Several researchers have attempted to categorise TMA based on these patterns as an aid to identification and differential diagnosis. The most recent classification, proposed by Canas et al. in 2010, establishes 2 types of MRI changes: cortical (type 1) and cortico-subcortical (type 2); type 2 TMA are additionally classified according to whether they have a prominent subcortical component (2A), border an old encephaloclastic lesion (2B), or are associated with remote lesions (2C) [7,8].

Although the underlying pathophysiological mechanism of these MRI abnormalities is not well understood, seizureinduced cerebral oedema has been suggested as the most likely cause.

MRI hyperintensities correspond to vasogenic oedema on T2-weighted sequences, cytotoxic oedema on diffusionweighted sequences, and mixed cerebral oedema on FLAIR sequences. According to most researchers, diffusion-weighted imaging is the most sensitive sequence for detecting seizureinduced TMA; it indicates cytotoxic oedema, which in these cases is reversible [8].

Differential diagnosis of seizure-induced TMA should include acute ischaemic stroke, encephalitis, venous



Fig. 1 – Brain MRI scan (axial FLAIR sequences). Top: high signal intensity in the left temporal cortical grey matter. Below: these findings had disappeared completely at 6 months, as shown by a follow-up MRI scan.

infarction due to cerebral venous thrombosis, posterior reversible encephalopathy syndrome, and brain tumour [9].

Our patient had no previous diagnosis of epilepsy and experienced 2 partial seizures with secondary generalisation occurring during night-time sleep; seizures were probably triggered by stress and sleep deprivation. We ruled out symptomatic epilepsy secondary to structural lesions due to our patient's age, clinical symptoms, and the fact that seizures occurred at night. On this basis, and considering the results from the first neuroimaging study, we conducted 2 additional MRI scans (at 6 and 18 months) to screen for other brain abnormalities such as low-grade glioma.

Focal brain abnormalities in our patient seemed to be a consequence of vasogenic and/or cytotoxic oedema as a result of a series of pathophysiological events which were triggered by seizures. The high metabolic demand of the epileptogenic focus, the release of neuroexcitatory amino acids (such as glutamate) to the extracellular domain, and failure of the Na⁺/K⁺-ATPase pump would explain cerebral oedema [7].

Our case is coherent with other studies reporting TMA after a single seizure or isolated seizures. Recognising the characteristics of this phenomenon is essential if we are to avoid using other unnecessary and more invasive diagnostic techniques.

Conflicts of interest

None declared.

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None declared.

Ethics

We declare that this manuscript does not contain clinical studies or patient data.

REFERENCES

- Kramer RE, Lüders H, Lesser RP, Weinstein MR, Dinner DS, Morris HH, et al. Transient focal abnormalities of neuroimaging studies during focal status epilepticus. Epilepsia 1987;28:528–32.
- [2] Gallardo Muñoz I, Portero de la Torre M, Bravo-Rodríguez F, Valenzuela Alvarado S. Transient cerebral changes in

magnetic resonance after isolated epileptic seizures. Neurologia 2011;26:434–6.

- [3] Xiang T, Li G, Liang Y, Zhou J. A wide spectrum of variably periictal MRI abnormalities induced by a single or a cluster of seizures. J Neurol Sci 2014;343:167–72.
- [4] Palmeri B, Talamanca S, Ragonese P, Aridon P, Daniele O, Terruso V, et al. MRI abnormalities following repeated and incoming seizures. Seizure 2011;20:90–2.
- [5] Hicdonmez T, Utku U, Turgut N, Cobanoglu S, Birgili B. Reversible postictal MRI change mimicking structural lesion. Clin Neurol Neurosurg 2003;105:288–90.
- [6] Reversible cortical diffusion restriction, hyperperfusion and T2-hyperintensity caused by two different types of epileptic seizure. Clin Neuroradiol 2012;22:239–43.
- [7] Canas N, Breia P, Soares P, Saraiva P, Calado S, Jordão C, et al. The electroclinical-imagiological spectrum and long-term outcome of transient periictal MRI abnormalities. Epilepsy Res 2010;91:240–52.
- [8] Nuno Canas, Pedro Soares. In: Prof. Peter Bright, editor. MRI abnormalities induced by seizures, neuroimaging – clinical applications. InTech; 2012. DOI: 10.5772/24725. Available from: http://www.intechopen.com/books/neuroimagingclinical-applications/mri-abnormalities-induced-byseizures.
- [9] Ong B, Bergin P, Heffernan T, Stuckey S. Transient seizurerelated MRI abnormalities. J Neuroimaging 2009;19:301–10.

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