Reversible peri-ictal MRI signal changes mimicking structural lesion: A Case Report

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Presence of seizure-induced signal changes on brain magnetic resonance imaging (MRI) have been increasingly recognized in the literature. Reversible MRI changes in epileptic patients may be the result of local brain swelling, a defect of cerebral autoregulation and a blood-brain barrier disruption during sustained epileptogenic activity. This is a Report of a 62-year-old man who was diagnosed with a subacute right parietal syndrome. MRI shows a tumefactive lesion in the right temporo-occipital lobes mimicking a structural lesion. Electroencephalogram (EEG) shows continuous ictal activity in the same region. Antiepileptic drugs were used, thus achieving progressive electro-clinical improvement. Subsequent MRI showed remission of signal changes.

Key Words: *Seizure, Electroencephalography, Magnetic Resonance Imaging, Peri-Ictal Mri Abnormalities.*

INTRODUCTION

In general population, the possibility a person has an epileptic seizure during his/her life is 8% to 10%[1]. Neuroimaging have been quite useful to identify the underlying structural cause in patients who suffer; however, in 60% to 75% of all cases, the etiology cannot be found.[2]

Apart from etiologic structural lesions of epilepsy, showing low or no change in time, imaging disturbances of the encephalon have been recognized to appear short before an epileptic seizure (peri-ictal).[3] These imagery findings have been described in various radiological techniques including computed tomography, SPECT and MRI among others.[4,5,6] Given its resolution and wide use in epilepsy studies, changes in MRI technique have been the most frequently described[3,7-10]. Those signal disturbances known as induced by the epileptic seizure itself, more than its etiology, which appear during and/or after an ictal epileptic seizure[3,11] are defined as temporary peri-ictal abnormalities in magnetic resonance (ATPR).

This is the case of a patient with a background of multiple cavernomatosis malformations who suffered provisional changes of MRI signal, secondary peri-ictal to parieto-temporal focal epilepsy crisis, whose imagery mimicked a structural lesion. Epidemiology, physiopathology, differential diagnosis and ATPR prognosis was reviewed as well.

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CASE REPORT

This is the case of a 62-year-old male patient with a background of multiple cavernomatosis malformations whose diagnosis was made by using imagery in previous years. He works as a heavy machinery operator. He has functional independence. He was taken to hospital, as he had a 10-day progressive condition, characterized by intermittent periods of spatial disorientation and a hard time to estimate proportions of physical spaces and objects he must work with in his work. The last few days prior to his consultation, his daughter found him struggling in his responses, so she decided to take him to our Emergency Room. Neurologic examination showed a bradipsychical patient, with intransitive ideomotor apraxia and sensory extinction in left hemi-body. A brain MRI depicted a cavernoma with a temporo-occipital cortical-subcortical right location, with no bleeding signs and an intra-axial lesion with an adjacent tumescent look (Figure # 1). Differential diagnosis is set as subacute cerebral infarction, syndrome of reversible posterior cerebral encephalopathy, source of localized encephalitis or post convulsive signal changes. The study was complemented with EEG (Figure # 2) which depicted three electrographic crises with long duration right occipito-temporal start. Since hospital admission and during his whole stay, he kept his blood pressure within range. Results of general examinations in serum and increased cerebrospinal fluid was normal. An anticonvulsant therapy was started with Levetiracetam, with progressive/sustained clinical picture in time. Control EEG, 48 hours after the first one did not report any pathological findings. Control MRI, 4 weeks after the crisis reported remission of the prior signal disorder (Figure # 1).

DISCUSSION

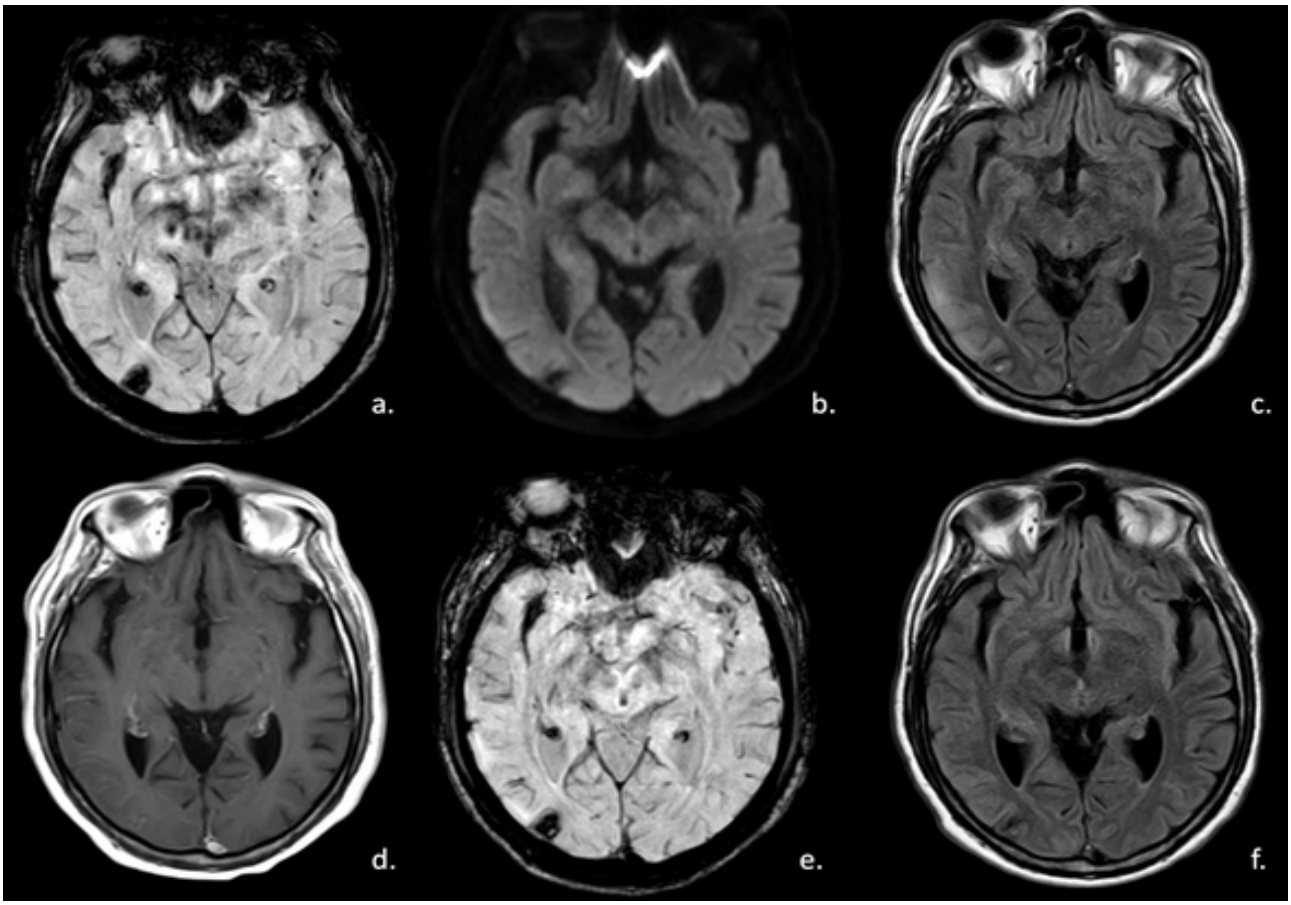
Prevalence of ATPR is unknown. In patients with epileptic status, it is estimated between 4.6% to 29.4%; in patients with isolated crisis or "crises cluster" is 0.007%.[10] Literature reports a wide dispersion in the figures. Such scenario is mainly given by different recruitment strategies, heterogeneous samples study and variable methodologies.[10]

Physiopathology of the signal changes is not fully known. These are explained as a consequence of toxic metabolic disorders, induced by a sustained epileptic discharge.12 An increase of energy demand, secondary to increasing cell activity caused by aberrant neuronal discharge, unleashes an increase of brain bloodstream as a compensation response (hydraulic metabolic coupling). If the remains in time, hemodynamic adaptation becomes insufficient to meet the demand, thus causing ATP depletion, tissue hypoxia, stimulation of the anaerobic glycolysis and failure of sodium/potassium ATPase pump.11-13 This energy failure causes dysfunction of cell membranes, thus affecting water flow between the membranes, between intracellular/extracellular compartments (cytotoxic edema).9,10 On the other hand, compensatory hyper fusion mediated by arteriolar dilatation remains the same, determining an increase of capillary pressure and breakthrough of the blood-brain barrier thus causing vasogenic edema.9,10,14

Among peri-ictal disturbances described in MRI we can find signal increase in the sequence of Diffusion Weighted Image (DWI) and corresponding signal decrease in Apparent Diffusion Coefficient (ADC), which represents a cytotoxic edema. In other cases, the increasing signal in DWI is along with an increasing signal in ADC, associated to hypersignal of T2 sequences and Fluid-Attenuated Inversion Recovery (FLAIR) which represents vasogenic edema.12 Apart from these lesions signal, nonspecific disturbances in T2 were reported and even, this suggests irreversible damage in mesial temporal cortex .7

ATPR may occur close to the epileptogenic zone or in remote structures, probably showing a way to spread the crisis.15 Regarding topography, the most frequent compromised areas are temporal mesial, neocortex, subcortical white substance, splenium corporis callosi, nuclei of the base, thalamus and cerebellum.10 ATPR may have radiological similarities with other pathologic entities including inflammatory causes, vascular, metabolic, neoplasic among others (Table 1). Its proper recognition avoids execution of unnecessary aggressive diagnostic procedures, as cerebral biopsy or angiographic procedures.16,17

Figure # 1. Encephalon MRI: (a) SWI sample lesion with traces of right occipital anterior cortical-subcortical susceptibility compatible with cavernous malformation. (b) DWI does not have restricted diffusion (c) FLAIR highlights right anterior occipital temporal cortical-subcortical edema to cavernous malformation (d) T1 with contrast has a mild cortical meningeal reinforcement. Control images after 4 weeks depicts persistence of cavernous lesions with no (and) and nearly full resolution of the existing edema in the initial study (f).



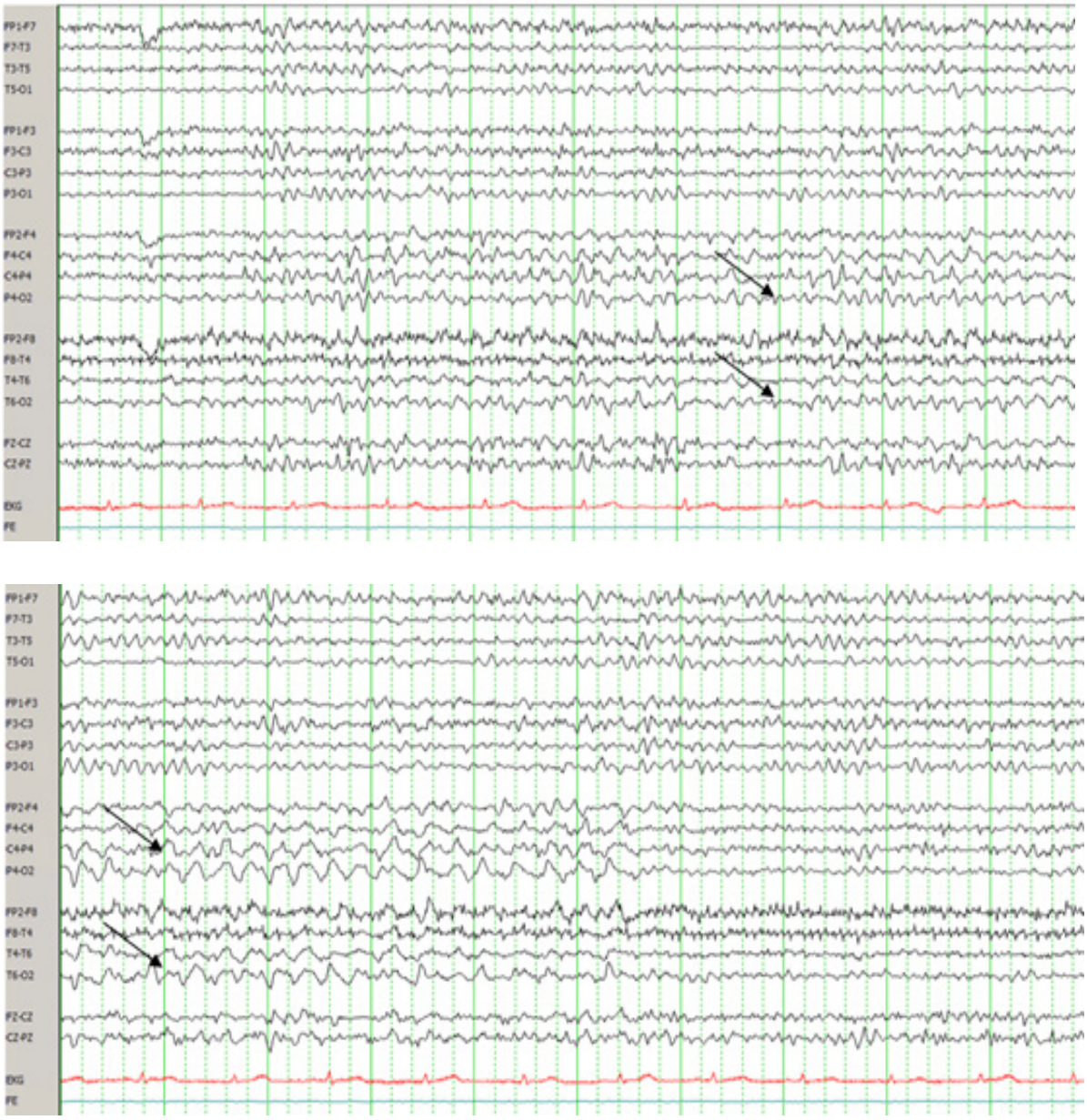
Expectable time to determine ATPR reversibility is unknown. Studies vary in time and follow up frequency (24 hours to 5 years).[10] Most lesions are estimated to revert in the medium term, from weeks to months, thus reflecting the dynamic nature of an epileptic seizure.[3,10,18,19] However, there are some reports regarding evolution to atrophy, gliosis and alteration of permanent signals in T^2 at locations where initially we could see edema and signal alteration in DWI.[7] Factors determining signal changes reversibility are type and duration of the crisis, existence of a previous structural lesion, pharmacological interventions and presence of hypoperfusion or hypoxia seen in MRI.[19]

In the peri-ictal stage there are imaging characteristics of ATPR which help to predict the evolution. One of the most relevant is the type of edema observed; if the ATPR is predominantly made up of cytotoxic edema, the likelihood to induce an irreversible lesion is high, compared with the ATPR represented as an image with vasogenic edema.[19] In general, clinical recovery is reported to precede the resolution of imagery disturbances.[18]

This work describes the case of a patient where a structural lesion (cavernoma) causes a steady focal crisis, thus generating signal changes adjacent to the epileptogenic lesion. Differential diagnoses (Table 1) in this case were categorically discarded, based on the clinical radiological evolution, general examinations and analysis of cerebrospinal fluid.

As a conclusion, peri-ictal signal disturbances, their wide range of imagery manifestations and the high variability of distribution determine a wide variety of differential diagnoses. The importance of their recognition and evolution

Figure # 2. Standard Electroencephalogram (montaje doble banana): record of the electrographic crisis number 3, which starts with rhythmic theta activity in the right posterior occipital and temporal region (pointed out by the arrow, Image # a), which further evolved to rhythmic delta activity with same location (Image # b).



in the time avoids performing examinations or therapies with unnecessary risks. 16

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Table # 1. Differential diagnosis of temporary peri-ictal disturbances in magnetic resonance [8,16,20]

Vascular acute and/or subacute encephalic infarction, hypoxic-ischemic encephalopathy
Inflammatory Viral encephalitis, infectious rhombencephalitis, progressive multifocal leukoencephalopathy, bacterial meningoencephalitis.
Neoplastic Solid primary or secondary encephalon tumor, lymphoma of the central nervous system.
Inflammatory not infectious Autoimmune encephalitis.
Metabolic Hepatic encephalopathy, hyperuricemia, hypoglycemic encephalopathy, mitochondrial diseases, extrapontine osmotic demyelination.
Other Creutzfeld Jackob disease, encephalic contusion, posterior reversible cerebral encephalopathy, mesial hippocampal sclerosis, splenium lesion temporary by another etiology.

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