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The seizing brain. A pictorial review of ictal and postictal MR imaging findings

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Purpose: The purpose of this work is to illustrate the spectrum of seizure-related imaging findings on MRI in the ictal and postictal phase and to try to explain these findings based on the presumed underlying pathophysiology.

Approach: Magnetic resonance imaging (MRI) studies were obtained from our own Picture Archiving and Communication System (PACS) from 2005 until 2014. This way we compiled a database of thirty-nine patients. The clinical and electroencephalography (EEG) data of these patients were obtained from the patient's medical file. The clinical data were used to determine the onset of symptoms and seizure type. The EEG results of the examination closest to the MRI studies were noted to determine ictal or postictal state (mean interval was two days before or after MRI) Only patients were included (1) with clinical and/or EEG data compatible with a ictal or postictal event, (2) with an MRI study prior to or within eight days of the end of symptoms and (3) in whom the MRI findings could not be attributed to other causes. After applying these inclusion criteria 20 patients were withheld for further examination. Recent literature was reviewed using the search terms "perfusion CT", "MRI", "MR", "postictal", "ictal", "todd's paresis" and "status epilepticus".

Results: The clinical data, EEG results and imaging studies of thirty-nine patients were reviewed. After applying the inclusion criteria, a cohort of twenty patients was withheld, of which eight patients received their MRI during status epilepticus (ictal phase) and twelve in the postictal phase. Eight patterns of signal alterations were recognized: cortical FLAIR hyperintensity and diffusion

restriction (N = 14), hippocampal FLAIR hyperintensity and diffusion restriction (N = 10), thalamic FLAIR hyperintensity and diffusion restriction (N = 5), prominent arterial branches (N = 1), sulcal FLAIR hyperintensity (N = 1), leptomeningeal enhancement (N = 1) and crossed cerebellar diaschisis (N = 1). Mesial temporal sclerosis could be seen in 4 patients. We could not discriminate between the ongoing status epilepticus and postictal state based on imaging findings. With the exception of temporal mesial sclerosis and crossed cerebellar diaschisis, these signal alterations disappeared or diminished on follow-up imaging studies where available.

Discussion: The different imaging findings can be explained through the presumed pathophysiological cortical processes during and after seizure. The FLAIR hyperintense signal and diffusion restriction seen in most patients most likely reflects focal cytotoxic edema. Focal cortical hyperexcitability leads to binding of glutamate on postsynaptic non-NMDA receptors, which results in sodium influx and finally cytotoxic edema. The location of these signal alterations can be in the focal area of seizure onset or distant from the focus due to propagation of seizure activity by known pathways (e.g. through the thalamo-cortical or cortico-pontine-cerebellar pathway). The propagation of seizure activity to the thalamus or cerebellum explains why in some patients FLAIR hyperintensity and diffusion restriction can be seen in these structures. The prominent vasculature can be explained through distortion of auto-regulation leading to vasodilatation. The leptomeningeal enhancement and sulcal FLAIR hyperintensity can be attributed to the disruption of the blood-brain barrier and resulting influx of proteins in the subarachnoid space. The susceptibility of the hippocampus to these signal alterations remains unclear.

Conclusion: In conclusion, the most common MR imaging patterns that can

be caused by seizures in the ictal or post-ictal phase are FLAIR hyperintensity and diffusion restriction in typical locations such as the cortex, hippocampus and thalamus. These signal alterations resolve over time in the great majority of cases. Before attributing the imaging findings to an ictal or a postictal event, other possible diagnoses (e.g. arterial ischemia, venous thrombosis, infection, neoplasm, PRES, metabolic encephalopathies), some of which can also cause seizures, should be excluded since the imaging findings are non-specific. The distribution of signal changes and the evolution over time however can help in making the diagnosis.

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