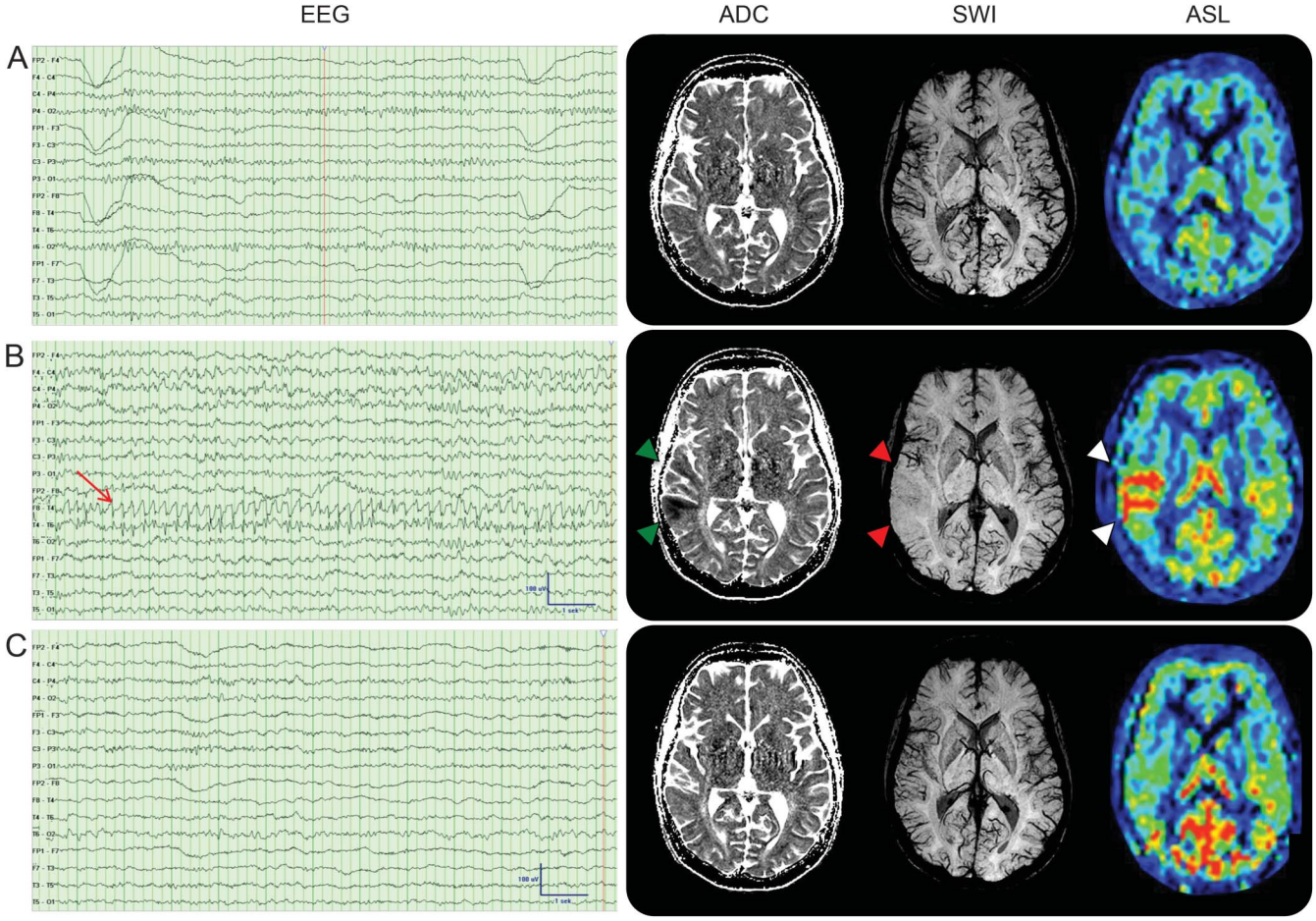
Susceptibility-weighted MRI signs of compensatory mechanism in nonconvulsive status epilepticus

Figure EEG and MRI (apparent diffusion coefficient [ADC], susceptibility-weighted imaging [SWI], arterial spin labeling [ASL]) findings before, during, and after nonconvulsive status epilepticus (NCSE)



Unremarkable EEG and MRI at presentation (A); follow-up EEG (B) demonstrates right frontotemporal rhythmic epileptiform discharges (red arrow). Right temporal ADC reduction (green arrowheads) and matching areas of reduced visibility of cortical veins (red arrowheads) and hyperperfusion on ASL maps (white arrowheads). On follow-up, EEG and MRI findings normalized (C).

A 22-year-old woman was diagnosed with CSF antibody-confirmed NMDA receptor encephalitis after repeated generalized seizures. Initial MRI and EEG were unremarkable (figure). She developed acoustic hallucinations and nonconvulsive status epilepticus (NCSE). MRI at this time showed hyperperfusion on arterial spin labeling maps and reduced apparent diffusion coefficient accompanied by reduced visibility of cortical veins on susceptibility-weighted MRI. The striking correlation between EEG and MRI findings suggests focal metabolic exhaustion (low apparent diffusion coefficient) and increased demand (hyperperfusion) caused by NCSE and reduced paramagnetic effect due to increased levels of oxygenated hemoglobin resulting in reduced venous signal.[1,2] All findings normalized under treatment.

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