

# Magnetic Resonance Imaging Findings in Status Epilepticus: A Case Report

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## Abstract

**Introduction:** Status epilepticus (SE) is a neurological emergency in which prolonged seizures require rapid diagnosis and treatment. It is classified as convulsive, non-convulsive and electrographic patterns. Neuroimaging findings associated with status epilepticus may raise suspicion of ischemia, encephalitis, or neoplastic lesions possibly leading to additional studies and surgical treatment. Further follow-up imaging may prevent unnecessary intervention due to the fact that findings in status epilepticus may be reversible.

**Case Report:** We report a case of convulsive status epilepticus with electroencephalography and cranial magnetic resonance imaging (MRI) findings discussing differential diagnosis.

**Conclusion:** Our case highlights that clinicians should be aware of this potential fatal condition and start the treatment immediately.

**Keywords:** convulsive status epilepticus, seizure, magnetic resonance imaging, transient, T2 hyperintensity.

## Introduction

Status Epilepticus (SE) is defined as a neurological emergency characterized by a single prolonged seizure or a series of seizures occurring close together with incomplete return to baseline. There are acute etiologies including stroke, hypoxia, systemic infection, trauma and metabolic disorders and chronic etiologies such as tumor and low concentration of anti-epileptic drugs. SE can present in convulsive, non-convulsive and electrographic patterns. Convulsive status epilepticus (CSE) is mostly recognized easily by presenting with unresponsiveness and tonic, clonic, or tonic-clonic movements of the extremities. These obvious manifestations of CSE may turn into more subtle extremity or face twitches, or saccadic eye movements<sup>1, 2</sup>. In addition, many neurological diseases manifest as seizure hence differential diagnosis of seizures may be challenging. Herein, we report a case of CSE with clinical and imaging features.

## Case report

A 41-year-old woman admitted to emergency department with unawareness and somnolence, poor oral intake and

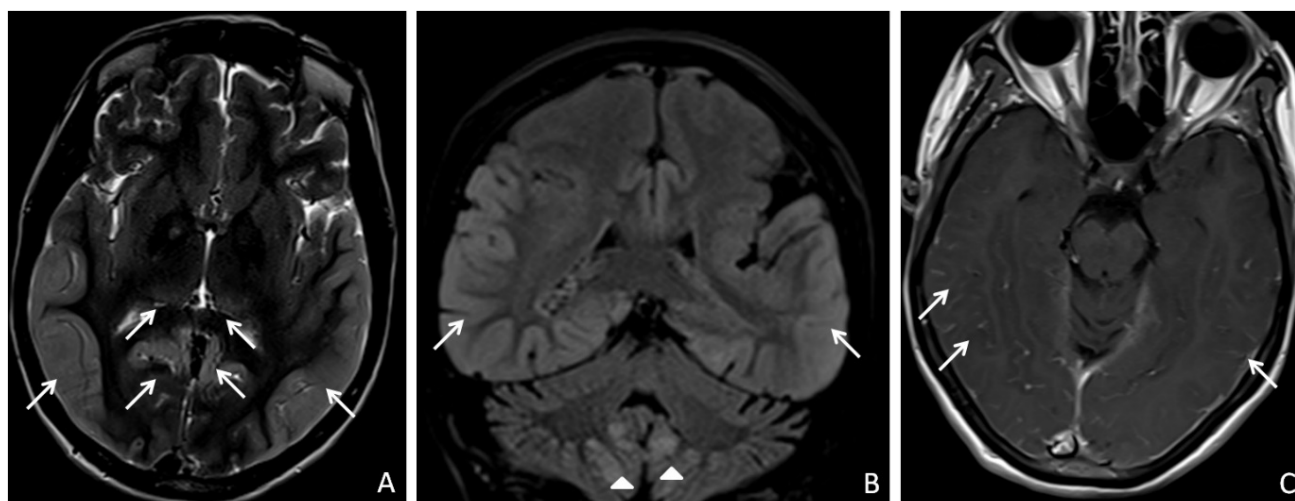
limited cooperation nausea during the preceding week. She was known to have childhood-onset epilepsy and refuse to take her drugs (phenytoin 100mg/day, lamotrigine 50mg/day) for a week. On admission, vital signs of patient were normal. Initial neurological examination revealed drowsiness, disorientation in time and place, she was poorly cooperative. Cranial nerve examination was normal. She was able to localize painful stimulus in all extremities and had no pathological reflexes. No evidence of nuchal rigidity or meningeal irritation was present. Laboratory examination is unremarkable except high serum C-reactive protein level. Patient had evaluated in another hospital a couple of days ago because of recurrent seizures. Clear cerebrospinal fluid with a normal opening pressure, no cells, normal protein, glucose were detected on lumbar puncture performed due to suspicious neck stiffness and fever 38.5°C. She was referred to our institution for further examination and treatment, since her recurrent seizures could not be controlled. Preliminary diagnoses were encephalitis, postictal prolonged confusional state, convulsive status epilepticus, stroke. Patient underwent MRI to investigate underlying cause of clinical symptoms. MRI showed T2 hyperintensity in the grey and subcortical white matter with mild mass effect. Increased diffusion-weighted imaging (DWI) signal and low apparent diffusion coefficient (ADC) values were observed in affect-

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**Received:** 23.12.2020 • **Accepted:** 20.01.2021

**DOI:** 10.33706/jemcr.845999

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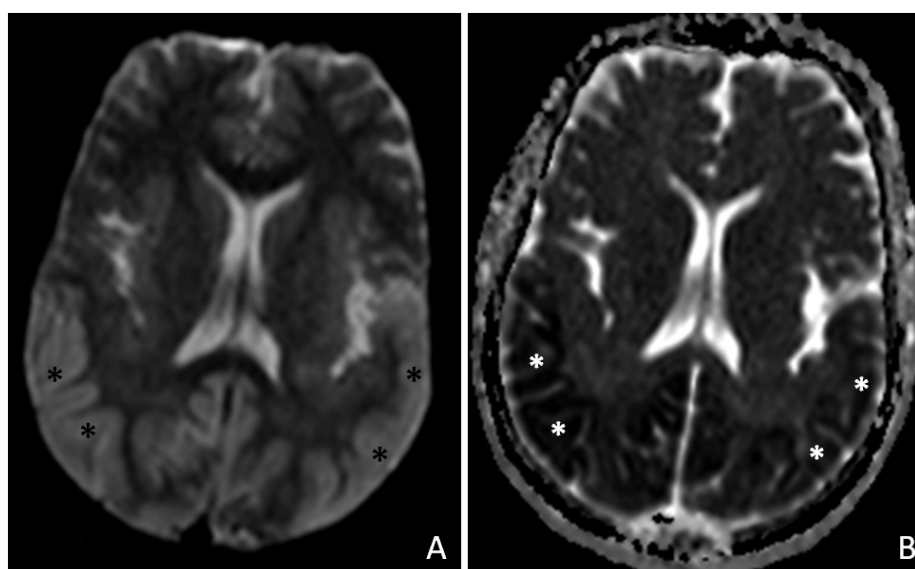
**Figure 1:** Axial T2W (A) and coronal FLAIR (B) MRI sequences reveal cortical and subcortical white matter (arrows), both cerebellum (arrowheads) and bilateral pulvinar nuclei hyperintensities and cortical swelling in bilateral temporo-occipital region (arrows). Axial post-contrast T1W image (C) reveal prominent bilateral occipital vessels (arrows).

ed areas. T2 hyperintensity was also detected in cerebellar hemispheres. Bilateral occipital leptomeningeal enhancement was noted on post-contrast T1 weighted (T1W) image (Figure 1 and 2). Due to unresponsiveness to treatment and ongoing seizures, she was intubated for the third-line treatment and medicated with propofol and followed up in intensive care unit. In addition to propofol infusion, phenytoin therapy (300mg/day) was maintained. Electroencephalography (EEG) showed sharp or sharp-slow wave activities in the right frontal hemisphere. Therefore, lacosamide was added for maintenance therapy. After 48 hours, no seizure activity observed clinically or electrophysiologically. Propofol was reduced and discontinued. Upon the development of seizures of a similar nature in a day, thiopental treatment was started, and valproic acid was added on to maintenance therapy. After six days, there were no seizure activity clinically

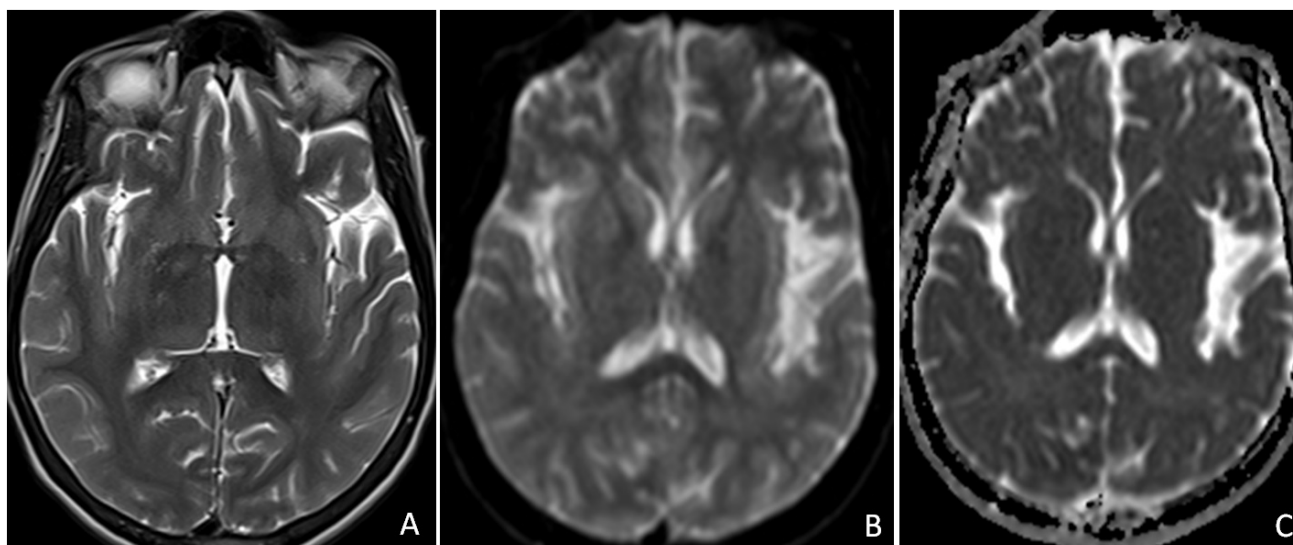
or electrophysiologically. She got better and was oriented in time, place and to her own person. The final diagnosis was CSE. Control MRI performed after a month later was normal, no diffusion restriction or cortical swelling (Figure 3). She was discharged with valproic acid 1500mg/day, phenytoin 300 mg/day, lacosamide 200 mg/day treatment.

## Discussion

One of the main causes of SE in epileptic patients is low blood concentrations of anti-epileptic drugs. Age, seizure duration and treatment response are prognostic factors. SE due to low serum concentrations of anti-epileptic drugs results in a usually good prognosis, with low mortality<sup>3</sup>. Early treat-



**Figure 2:** A diffusion-weighted axial MRI image (A, asterisk) reveals hyperintensities in the corresponding regions. The accompanying lower ADC values is noted as dark signals on ADC map (B, asterisk).



**Figure 3:** Axial T2W and DWI MRI obtained a month later shows resolution of abnormalities. The case report has written in an anonymous characteristic, thus secret and detailed data about the patient has removed. Editor and reviewers can know and see these detailed data. These data are backed up by editor and by reviewers.

ment with intravenous anticonvulsants is a significant step in the treatment of SE. Therefore, clinicians should suspect of SE and plan diagnostic assessment. Benzodiazepines are used as first-line treatment in CSE. Phenytoin and valproate are generally preferred as second-line drug. Levetiracetam and lacosamide are well tolerated popular drugs in second or third line agents. Thiopental was also added to treatment due to refractory seizures in our patient while followed in the intensive care unit. In general, EEG is not essential to start treatment, moreover, none of the ictal EEG patterns is specific to SE. The EEG in CSE shows various seizure patterns related to seizure types<sup>1</sup>. There is unquestionable evidence of MRI to facilitate differential diagnosis. In our case, SE was suspected by MRI findings in the first line. T2 and FLAIR hyperintensity with mild cortical swelling and corresponding restricted diffusion areas are often observed in cortex and hippocampi in SE cases. Basal ganglia, thalami, especially pulvinar nuclei, corpus callosum may also be involved. SE MRI findings may resemble that of posterior circulation ischaemic stroke<sup>4</sup>. It is important to know that T2 signal changes do not respect vascular territories in SE unlike stroke. These changes are suggested to be caused by a combination of vasogenic and cytotoxic edema. Ischemia and metabolic abnormalities trigger cytotoxic edema in gray matter. Increased perfusion and vascular permeability are responsible for vasogenic edema in white matter<sup>5</sup>. Gyral contrast enhancement, may be the reflection of impaired blood brain barrier, is another possible finding in MRI. Encephalitis may mimic SE, too. Herpes Simplex Encephalitis is the most common cause of fatal sporadic necrotizing viral encephalitis. In adults, Type 1 herpes simplex virus is more common than type 2 and involves the cortex and the subcortical white matter of bilateral temporal, frontal lobes, and insula on T2W MRI. SE typically involves in cortex

and post-ictal edema affects entire hemispheric cortex. Hemorrhage is rare in SE cases<sup>6</sup>. Posterior reversible encephalopathy syndrome (PRES) is also a considerable differential diagnosis of SE for presenting with seizures and confusing MRI findings. It is characterized by reversible asymmetric subcortical vasogenic brain oedema, mostly in the bilateral parietooccipital region. Posterior fossa involvement is rare unless patient has an autoimmune disease<sup>7</sup>. Accompanying restricted diffusion and hemorrhagic foci may be seen both in encephalitis and PRES<sup>6,7</sup>. Changes in the cerebellum in SE have been explained as a result of crossed cerebellar diaschisis because of prolonged excitatory synaptic activity via the cortico-cerebellar pathways<sup>8</sup>. Reversibility of changes in follow-up MRI is the main clue to exclude tumoral lesions. We have noticed the resolution of changes at the first month follow-up MRI of our patient. Brain atrophy, mesial temporal sclerosis and cortical laminar necrosis may be seen in long term especially in generalized convulsive SE<sup>9</sup>.

## Conclusion

Status Epilepticus (SE) is a neurological emergency that can mimic and be overlapped with multiple neurological conditions. Although T2W MRI abnormalities are mainly located in posterior regions, changes can also be focal, multifocal, hemispheric, or diffuse in SE. Knowing these findings and performing serial cranial MRI is necessary in order to adequately identify SE and distinguish it from other neurological entities. It has a remarkable mortality rate. Early recognition of these patients and early initiation of aggressive treatment is crucial and can prevent morbidity and mortality.

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