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Periictal magnetic resonance imaging in status epilepticus

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Summary
Purpose: To determine the changes of magnetic resonance imaging (MRI) during the periictal phase in status epilepticus (SE).
Patients and methods: We identified 15 patients diagnosed of status epilepticus with corresponding MRI changes, including 11 patients with generalized convulsive status epilepticus (GCSE), 2 with complex partial status epilepticus (CPSE), and 2 with simple partial status epilepticus (SPSE). All MRI changes, corresponding electroencephalogram, and prognosis were evaluated.
Results: Regional cortical lesions were observed on MRI, including restricted diffusion in diffusion-weighted images (DWIs) (11 out of 15) and hyperintense signal change in fluid-attenuated inversion recovery (FLAIR) images (12 out of 15) with hypervascularity and parenchymal swelling. The remote lesions included crossed cerebellar diaschisis (3 patients), ipsilateral thalamic lesion (4 patients), and basal ganglia lesions (3 patients). Although the periictal MRI changes were usually reversible, irreversible changes were also found, especially in GCSE, such as focal brain atrophy, cortical laminar necrosis, and mesial temporal sclerosis. GCSE patients with periodic epileptic form discharges had higher possibilities of widespread MRI abnormalities and poor prognosis in the future.

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Conclusions: In this study, DWIs and FLAIR images were proved useful in determining the extent and severity of early neuronal damage caused by epileptic discharges in SE patients. Seizure-induced long-term injuries were also observed in the follow-up MRI.

Introduction

Status epilepticus (SE) is a seizure that persists for a sufficient length of time or is repeated frequently enough that recovery between attacks does not occur. It represents the persistent neuronal firing and the release of excess glutamate, which activates postsynaptic *N*-methyl-D-aspartate (NMDA) receptors and triggers receptor-mediated calcium influx. The release of excess glutamate leads to desensitization and internalization of postsynaptic gamma-aminobutyric acid A ($GABA_A$) (Goodkin et al., 2008), increases the expression of proconvulsive neuropeptides (Liu et al., 1999; Mazarati et al., 1999), and continues the vicious cycle of self-sustained seizure. The calcium influx causes a cascade of biochemical changes, mitochondrial dysfunction and oxidative stress (Cock, 2007), gene expression, and initiation of cell death (Fujikawa, 2005). At the same time, large increases in the cerebral metabolic rate of glucose and oxygen continue throughout SE, associated with adenosine triphosphate (ATP) depletion and lactate accumulation, and finally leading to hypermetabolic neuronal necrosis (Wasterlain et al., 1993). Several studies have demonstrated a significant risk of neuronal injury in prolonged seizure (Duncan, 2002; Holmes, 2002).

Recent advances in magnetic resonance imaging (MRI) provide new opportunities for identifying early seizure-related neuronal damage and the affected areas (Cole, 2004). The findings include restricted diffusion in diffusion-weighted images (DWIs), hyperintensity in fluid-attenuated inversion recovery (FLAIR) images, swelling of the focal structure, cerebral hyperperfusion in magnetic resonance (MR) perfusion, and increased vascularity in MR angiography (MRA). MRI abnormalities were found in the focal cortex, thalamus, and splenium in focal SE patients (Bauer et al., 2006; Buracchio et al., 2008; Chu et al., 2001; Doherty et al., 2005; Hormigo et al., 2004; Lansberg et al., 1999; Oster et al., 2003; Szabo et al., 2005; Toledo et al., 2008); in the generalized convulsive SE (GCSE) patients, these MRI abnormalities were usually widespread across the cortical areas and thalamus, accompanied with crossed cerebellar diaschisis sometimes (Men et al., 2000; Nixon et al., 2001; Teixeira et al., 2002). These MRI changes were often reversible but can become irreversible and permanent in severe or prolonged seizures (Chevret et al., 2008; Doherty et al., 2004; Donaire et al., 2006; Men et al., 2000; Nixon et al., 2001; Sirven et al., 2003).

In this study, we selected 15 patients with SE and evaluated each of their MRI findings, corresponding electroencephalogram (EEG), and prognosis. The underlying pathophysiology linking seizure and periictal MRI changes was discussed.

Materials and methods

Between January 2006 and December 2008, we evaluated patients diagnosed with SE and each of their corresponding MRI at Chang-Gung Memorial Hospital. SE was defined as ''at least 5 min of continuous seizure or 2 or more discrete seizures without recovery of consciousness.'' Focal seizure with secondary generalized convulsive seizure over 5 min was classified as GCSE. However, SE patients concomitant with anoxia, central nervous system (CNS) infection, CNS malignancy, shock or severe metabolic disorders were excluded, except one patient with frontal abscess and typical presentation of seizure-related image. All the MRIs were evaluated first by the same neurologist and any abnormalities related to periictal changes were reviewed again by another neuroradiologist. Both reviewers agreed that all the MRI changes in the selected patients were related to seizure rather than other causes.

Fifty-one patients fulfilled the criteria and received MRI exams (23 females and 28 males; 32 GCSE and 19 focal SE; mean age: 57.9 ± 20.0 years; range: 18—89 years). Of these patients, 15 patients were associated with seizure-related changes in MRI (7 females and 8 males; mean age: 59.2 ± 17.4 years; range: 22—79 years). There were 11 GCSE, 2 complex partial status epilepticus (CPSE), and 2 simple partial status epilepticus (SPSE) among them. All of them had been treated at our Neurology Department. A standard case collection form was used to record the age of onset, gender, clinical manifestations, seizure pattern, EEG, cerebrospinal fluid (CSF) analysis results, timing of MRI, acute periictal MRI findings, follow-up MRI findings, and outcomes.

MRI was performed by using a 1.5-T Philips Gyroscan Intera scanner (Philips Medical Systems). The following is the epilepsy protocol: (1) sagittal T2-weighted turbo spin—echo (repetition time [TR]/echo time [TE] = 4510/110 ms) for optimal orientation of the subsequent images; (2) coronal FLAIR (TR/TE = 9000/90 ms); (3) coronal T2-turbo spin—echo (TR/TE = 3700/100 ms); (4) axial images parallel to the long axis of the hippocampus, T1 spin—echo (TR/TE = 405/12 ms); (5) axial T2-sturbo spin—echo (TR/TE = 4780/110 ms); (6) axial single-shot echo-planar imaging (EPI) DWI (TR/TE = 2554/71 ms, diffusion gradient with b value of 1200 s/cm^2 applied along slice axis); (7) axial FLAIR (TR/TE = 9000/90 ms); and (8) three-dimensional time-of-flight (TOF) MRA sequence, 0.7-mm thick (TR/TE = 23/2.3 ms). The contrast agent was injected into the ante-cubital vein at a flow rate of 4 cm^3/s via a large gauge venous cannula.

EEGs were recorded using a 24-channel Nicolet Voyageur digital electroencephalograph, with the time constant adjusted at 0.3 s, lead velocity at 30 mm/s, high frequency filter at 70 Hz, and sensibility between 7 and 10 uV/m. Electrodes were placed according to the International System (10—20).

Results

Clinical profile (Table 1)

All the patients were fulfilled with the diagnosis of SE with duration over 30 min, included GCSE (patients 1—11), CPSE (patients 12 and 13), and SPSE (patients 14 and 15). Among