MR imaging of cerebral and cerebellar hypervascularity associated with status epilepticus: case report

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ABSTRACT

A 20 year old male presented to the emergency department with generalized tonic clonic convulsions and up rolling of eye balls. He had history of seizure disorder for three years and on regular medical treatment and is compliant to medication. A non-contrast CT scan of the head was only done on 14th day of admission which showed hypodense areas in the right and left cerebellar hemisphere. MR imaging was performed four days later revealed high signal intensity in the both cerebellar hemispheres, both external capsules, both occipital and right parietal regions on fluid-attenuated inversion recovery (FLAIR). The post contrast MR imaging revealed diffuse cerebral and cerebellar hypervascularity in the similar region. This change of diffuse hypervascularity of both cerebral and cerebellar associated with seizure activity on post-contrast magnetic resonance imaging (MRI) has not been reported in any literature.

Keywords: Cerebral hypervascularity, spilepsy, magnetic resonance imaging, status epilepticus,

INTRODUCTION

A few reports in the literature have been published demonstrating cerebral hypervascularity in patient with status epilepticus seen with cerebral angiography and MRI.[1,2] It is important to recognize that seizure can produce diffuse cerebral and cerebellar hypervascularity on post contrast MR imaging to avoid unnecessary neurosurgical intervention such as invasive cerebral vascular studies or brain biopsy. The aim of the case report is to share our experience in recognizing that hypervascularity does not only affect the cerebrum but also can affect the cerebellum.

CASE REPORT

A 20 year old male, was seen in emergency department with generalized tonic-clonic convulsions with up rolling of the eye balls. He was given IV diazepam (20 mg in divided doses) but failed to respond and IV phenytoin (750 mg infusion) was later given. His oxygen saturation reduced to 70.0% and he had to be intubated and ventilated. He was extubated after 10 days. A non contrast CT scan of the head obtained on 14th day of admission showed multiple hypodense areas in the right and left cerebellar hemisphere. An MR imaging was performed on 18th day of admission and revealed high signal intensity in both cerebellar hemisphere, bilateral external capsule, bilateral occipital and right high parietal region on fluid attenuated inversion recovery (FLAIR) (Fig. 1, 2). The post contrast T1-weighted MR imaging revealed diffuse cerebral and cerebellar hypervascularity in the similar region (Fig. 3, 4 and 5). The T1 and T2-weighted sequence were unremarkable. No follow-up MR imaging was done. The patient was treated conservatively and discharged after 20 days of admission in a semiconscious state and with focal neurological deficit. He is a known case of seizure disorder for the last three years and on regular medical treatment on tablet sodium valproate (300 mg twice a day) and tablet propanolol (20 mg twice a day). A non-contrast CT scan was done at the time of first presentation and MR imaging of the head done two years later were unremarkable.

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MR imaging at the level of the cerebellum (Fig.A) and the level of the head of caudate nucleus (Fig. B) revealed areas of high signal intensity in both cerebellar hemisphere, bilateral external capsule, bilateral occipital and right high parietal region on fluid-attenuated inversion recovery.

DISCUSSION

Neuroimaging studies are often performed to evaluate patients who are in status epilepticus for ischaemic, haemorrhagic, inflammatory, infectious or neoplastic processes.

It is important to recognise the characteristic neuroradiologic features of these conditions as this will help to differentiate status epilepticus from other disorders because there can be overlap in the findings. We have described a patient with status epilepticus, in whom CT scan and MR imaging techniques were used.

Increased blood flow through cerebral vessels on the affected hemisphere has been reported during status epilepticus.[3,4] Transient computed tomography and magnetic resonance imaging (MRI) abnormalities may occur after a seizure, most likely following status epilepticus.[4-11] Contrast enhancement of various intensities may be observed and may be related to alteration of blood-brain barrier.[9] Lansberg *et al* has demonstrated leptomeningial enhancement on post contrast MR imaging in status epilepticus.[4] They have also demonstrated that leptomeningial enhancement, T2 hyperintensity and asymmetry of the cerebral arteries on MRA were not present on follow-up MR imaging examination; vascular changes and vasogenic edema associated with status epilepticus are reversible (4). To our knowledge, this is the first case report to describe both the cerebral and cerebellar hypervascularity affected diffusely in post contrast MR imaging. In our patient, we were unable to assess the reversibility of these hypervascularity because follow-up MR imaging was not performed.

The post contrast T1-weighted MR images revealed diffuse patchy cerebral and cerebellar hypervascularity in the similar region (Fig. C, D, E).

In our case, the status epilepticus lasted for nearly an hour and then recurred again after some time which required ventilatory support. As the seizure activity could not be controlled early, neurological damage is present. The prolonged status with which the patient suffered from, might have lead to release of unidentified mediators locally, secondary to metabolic demands placed on by the ongoing status. During status epilepticus, excessive release of excitatory amino acids, such as glutamate,[12,13] and increased membrane ion permeability[14] are the mechanisms that could cause cytotoxic edema.

Unfortunately, nuclear medicine studies such as positron emission tomography (PET) was not done in our case. Changes of hyperaemia and glucose hypermetabolism was shown to be present in cases of status epilepticus in PET.[3]

Recognition of cerebral and cerebellar hypervascularity after status epilepticus can encourage follow-up MR imaging to establish their nature and prevent unwarranted intervention.

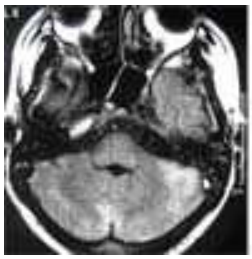

Fig. 1. MR axial FLAIR

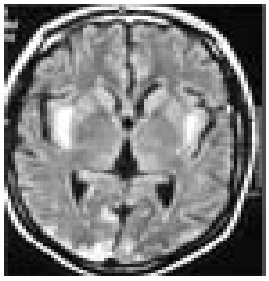

Fig. 2. MR axial FLAIR

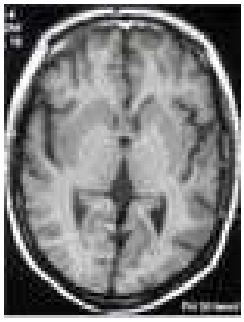

Fig. 3. MR axial T1 pre contrast

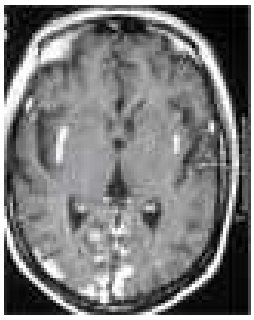

Fig. 4. MR axial T1 post contrast

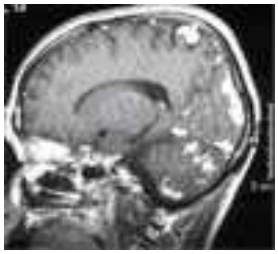

Fig. 5. MR coronal T1 post contrast