Magnetization Transfer (MT) MR Imaging in Patients with Posttraumatic Epilepsy

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Abstract: There is an uncertainty regarding the time period for seizures onset and the possible role of tissue changes for constituting an epileptogenic focus in intractable seizures following head trauma. MT MRI was performed in 44 individuals (13 controls, and 31 with seizures from 1-10 years following trauma) to assess the severity of tissue damage and its relationship with intractability. The group with MT abnormality beyond T2 abnormality had a significantly higher intractability of seizures compared to the one with MT abnormality within T2 abnormality. We conclude that MT imaging may help in predicting the seizure intractability in delayed posttraumatic epilepsy.

Introduction:

Posttraumatic epilepsy is an established consequence of head injury and factors involved in the origin are still unclear. The commonly accepted risk factors for delayed posttraumatic seizures are: early posttraumatic seizures, depressed skull facture, intracranial hematoma, prolonged unconsciousness, low Glasgow coma score, and prolonged posttraumatic amnesia. It was also documented that cortico/subcortical brain lesions represent the main risk factor for delayed posttraumatic epilepsy. It was found that evidence of hemorrhagic and non-hemorrhagic shearing injury on MRI on T2 and T2* weighted images in patients with normal head CT who had suffered even minor head trauma. MRI has demonstrated increased sensitivity for the detection of traumatic white matter abnormalities including diffuse axonal injury when compared with CT. MT MR imaging has been used in better detection and tissue characterization of diffuse axonal injury. There are studies showing the application of MT MRI in neurocysticercosis related seizures; however, there is no study showing its application in patients with posttraumatic epilepsy.

In this study, we have studied the potential of MT MRI in patients with delayed posttraumatic epilepsy.

Methods:

Forty-four patients with severe head injury were subjected to clinical evaluation and MR imaging. There were 38 males and 6 females with age ranging between 11 to 50 years and were imaged between 1 to 10 years following head injury. The patients with head trauma presented with seizures within one week were labeled as early posttraumatic epilepsy while after one week, were labeled as delayed posttraumatic epilepsy. The seizures were considered as intractable when these could not be controlled even after taking 2-3 anti-epileptic drugs (AED).

MR imaging was performed on a 1.5-tesla super conducting system using circularly polarized head coil. Conventional spin echo, proton density, T2 (TR /TE 1,2 /n = 2200 ms /12,80 ms /1) and T1 (TR /TE /n=1000 ms /15 ms /2) weighted imaging was performed in axial plane using 256x256 matrix, 0.5 inter-slice gap and 5 mm slice thickness. MT MRI was also performed with exactly same parameters as for T1 except for the off resonance pulse. Phase corrected gradient echo (GRE) imaging (TR /TE /FA /n = 800 ms /15,35 ms /15° /2) was also performed to look for any susceptibility effect due to the presence of hemosiderin. All cases were evaluated for the presence of hemosiderin, encephalomalacia, as seen on T2 and T2* weighted images with/without phase correction. The MT abnormality beyond T2 abnormality was considered as present when the hyperintensity on MT images extended beyond the normal appearing regions on T2 weighted images. When the abnormality seen on MT images did not extend beyond the hyperintensity seen on T2 weighted images, it was termed as MT abnormality within the abnormality seen on T2 weighted image. The presence of hemosiderin was seen hypointense signal on T2 weighted images that show bloom effect on gradient echo images and negative phase on phase corrected GRE images. The hemosiderin was considered to be focal, when it was seen as hypointense drop/splash on phase corrected GRE images. It was considered diffusing in to the parenchyma when the hemosiderin stained the brain tissue depicted as hypointense signal on T2* weighted and phase imaging.

Results:

There were 13 patients with no history of seizures and 31 patients with history of seizures. All 13 controls showed variable encephalomalacia in the frontal and temporal lobes. Six of these patients had evidence of hemosiderin demonstrated on phase corrected GRE images. None of these patients showed MT abnormality beyond the hyperintensity seen on T2 weighted images. Thirteen of the 31 patients presenting with seizures, showed MT abnormality beyond T2 weighted images and 12 out of these 13 patients had intractable epilepsy. Nine of these 13 patients showed hemosiderin deposit as dot/splash and 2 of these 9 had diffuse staining of the parenchyma with hemosiderin in addition to focal deposits. These two patients showed gliosis around the diffusing hemosiderin signal. There was no significant difference in focal hemosiderin deposition among the different groups with or without seizures ($p>0.05$). In remaining 18 patients with seizures, and no extension of MT abnormality beyond T2 weighted images, only four patients had intractable seizures. The hemosiderin deposit was detected in 3 of these patients. In remaining 14 /18 patients, hemosiderin was seen in 6 patients with no diffusion in to the parenchyma. Patients with MT abnormality beyond T2 abnormality showed significantly higher seizure intractability (12/13, $p<0.05$) compared to the one with T2 abnormality more than MT (4/31) in the remaining 31 patients.

Discussion:

Delayed posttraumatic epilepsy occurs in over 30% of patients with penetrating head injuries, intracerebral and subdural hematoma, depressed skull fracture or early seizures after injury. MT MRI is known to show a larger region of axonopathy in head injury compared to conventional MRI. In the present study, 13 patients we observed abnormal area on MT images that was beyond the abnormality seen on T2 weighted image. It suggests that the brain area affected by the injury is actually is more than T2 weighted images. The patients with MT abnormality beyond T2 weighted images had significantly higher occurrence of intractable epilepsy. It suggests that MT imaging by demonstrating a much larger gliosis may predict intractability in delayed posttraumatic epilepsy. Surgery has been used in patients with posttraumatic epilepsy to minimize the seizure activity. The surgical results depend on the excision of the gliotic region as suggested by T2, T1 weighted images and intraoperative mapping with electrodes. It is believed that incomplete identification of the gliotic region is responsible for the failure of surgery to prevent seizure recurrence. We believe that this combined approach of mapping may be of value in planning of the appropriate therapy and may yield better results in these cases.

In head trauma, blood extravasation causes deposition of ferrous compound into neural tissue and the Haber-Weiss iron catalyzed reaction, with hyperproduction of hydroxyl radicals. These reactive oxygen derived species trigger subsequent formation of peroxidative agents, which in turn cause a self-sustained lipid peroxidation of phospholipid membranes and disruption of the cell wall leading to cell death. We have observed that mere presence of bleed not a significant causative factor in eliciting the posttraumatic gliosis. However, we observed two cases where the iron was seen staining the cortical, subcortical region and extended along the white matter tracts that was demonstrated on T2* with phase imaging. MT imaging showed gliosis around the hemosiderin deposits and both these patients had intractable seizures. These observations indicate that presence of the hemosiderin may not be strongly related to seizure activity but presence of diffusion of the iron along the tract probably elicits large scarring and produce epileptogenesis.

We conclude that MT imaging supplements T2 weighted images in predicting the severity of gliosis in delayed posttraumatic epilepsy and may be of value in predicting the intractability of the seizure. Presence of bleed may not be a sufficient risk factor for delayed posttraumatic epilepsy but diffusion of the iron along the tracts may elicit extensive gliosis and may actually be responsible for the intractable seizures in these patients.