MR Imaging of Traumatic Brain Injury in Animal Model at 21.1 T

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Introduction

Traumatic brain injury (TBI) affects an estimated 1.5 million Americans every year. It has been assessed that as many as 20% of soldiers have experienced a TBI during combat exposure. There is conflicting data about interactions between ethanol and TBI. While there is a pre-clinical report suggesting that ethanol intake may improve spatial learning after TBI, but some clinical studies have shown that ethanol consumption aggravates morbidity and mortality related to TBI. It is known that chronic alcoholism has been linked to deficiencies of zinc and there is evidence that TBI puts patients at significant risk for zinc deficiency. The clinical association between cytotoxicity, edema, and poor long-term outcomes in patients suffering from TBI advocates the need for better understanding the mechanisms associated with recovery after TB injury.

Experimental

Rats were fed a zinc adequate diet (30 ppm of Zn) or a zinc supplemented diet (180 ppm of Zn) for 16-18 days. Subsequently a moderate TBI was implemented using the standard controlled cortical impact method. After injury, rats were given 3 g/kg dose of ethanol daily for 7 days via gavage. Control groups included zinc adequate fed animals that were given the injury without alcohol and sham (uninjured) animals. MR imaging was performed one and 7 days after injury on the 21.1 T MRI scanner (NHMFL, Tallahassee, FL). Diffusion MRI of the rodent head was achieved by spin-echo pulse sequence with flow/motion compensated gradients in all three directions x, y, z. Two b values were used of 100 and 1000 sec/mm2. Echo time was TE=34 ms and 15 slices were acquired using back projection method. All animal experiments were conducted according to the protocols approved by the Florida State University ACUC.

Results and Discussion

The major difficulty of the experiments is the reproducibility of the TBI impact. MR diffusion imaging can efficiently control this stage by detecting the brain metabolically challenged area as a result of TBI (Fig. 1 A, C). The experiments revealed that some of the TBI animals do not have these areas and consequently have a better outcome during recovery. The TBI impact also creates a large edema in nearby brain areas, which can be clearly seen on the diffusion maps immediately after TBI. This edema is not crucial and usually not seen on the images 7 days after TBI. The major consequence for the recovery after TBI is the development of necrosis in the previously detected ischemic areas of the brain (Fig. 1 B, D), especially, if there is no proper treatment.

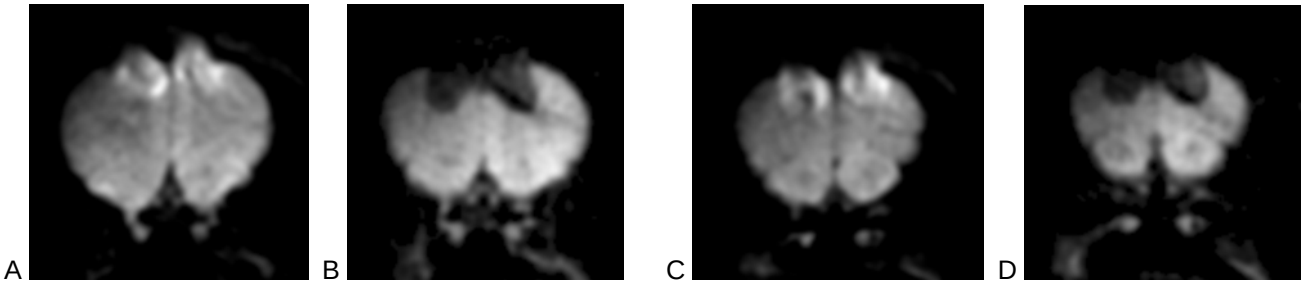

Fig. 1. Soon after TBI the challenged by ischemia areas in rat brain can be detected as bright sports on diffusion weighted MR images (A, C). Later on, in 7 days, these areas, depending on therapy, may develop necrosis which is represented by dark areas (B, D).

Conclusions

Diffusion MRI can efficiently detect the level of TB injury and guide the development of subsequent treatment or testing of the corresponding therapy. The preliminary results of our experiments support the need of Zn supplemental therapy. The experiments provide a background for evaluation the role of sodium MRI during TBI.

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