Regional Variations of Apparent Diffusion Coefficient in the Brain: Correlation with Glasgow Coma Score Among Traumatic Brain Injury Patients

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Introduction

CT scanning is the modality of choice to evaluate patients with acute TBI. However, patients who have a normal CT scan but present with an abnormal Glasgow coma score (GCS) require MR imaging to determine the presence of injuries that are occult to CT. In a small group of these patients, even routine MR scanning fails to explain the reason for the low GCS score. Diffusion weighted imaging (DWI) has been studied in experimental animal models of TBI, but very few studies exist that have looked at its effectiveness on TBI patients.[1,2] We recently reported a strong correlation between the apparent diffusion coefficient (ADC) and the Glasgow Coma Score (GCS) among traumatic brain injury patients.[3] In this study we determined if the changes in ADC's are reflective of a diffuse injury throughout the brain or whether certain regions of the brain are more affected than others.

Methods

Forty-four patients with TBI were sub-divided into (1) Group A: normal MRI imaging findings but low GCS (n=12); (2) Group B: brainstem injury with low GCS (n=13); (3) Group C: patients whose lesions were confined to the cortex with normal GCS (n=7); and (4) Group D: the same as group C but with low GCS (n=12). Normal control group consisted of 12 subjects. ADC maps of the whole brain were obtained following which histograms of the ADC values from the whole brain (with the skull removed) were generated. Histograms from the ROI's drawn in the brainstem, deep nuclei, gray and white matter were also obtained. Mean ADC's and the peak ADC's (mode of the distribution) were determined from each of the histograms. A one-sided t-test between the control group and the above groups was computed for the average and peak ADC. Correlations were performed between the averaged GCS score of each group and their corresponding peak and mean ADC values.

Results

A significant difference in the peak value of whole brain histograms was seen between the control group and Groups A, B, & D ($p<0.0005$, 0.0049, 0.001 respectively) and was consistent with previously published results.[3] A lower level of significance was reached for the peak ADC ($p<0.07$) between Group C and the control group but no difference was found in the average ADC. Both Groups A and D that had the lowest GCS score also showed significant differences in the mean ADC compared to the normal subjects ($p<0.04$, $p<0.05$ respectively). Significant differences were also seen in the peak ($p<0.04$) and average values ($p<0.03$) of the whole brain ADC values between Groups C and D where the difference was mainly in their GCS score but exhibited minimal injury as seen by MRI. A strong correlation between the whole brain peak ADC of each of the groups and the GCS score ($R^2=0.84$) and a somewhat weak but significant correlation were observed between the mean ADC and the GCS scores ($R^2=0.48$) of the different groups. Peak ADC measures for brain stem measurements were significantly different ($p<0.01$-0.08) in all groups when compared to the normals except for Group C, which exhibited normal GCS score. The average ADC's for the brain stem measurements were not significantly different from any of the groups. When comparing the white matter ADC's between the normals and the above groups' normal appearing white matter (away from lesions), both the peak and average ADC's were significantly different in both the hemispheres. Similar results were found for both gray matter and deep nuclei. When comparing the normal controls to those patients who had brainstem lesions, significant regional differences were found in both hemispheres in the gray matter and the deep nuclei, but the differences in white matter did not reach significance.

Conclusions

It is not infrequent that TBI patients exhibit abnormal GCS but have normal morphological findings as evidenced by CT and MR. We have previously shown that in such cases, whole brain ADC's may be an independent indicator of TBI that corresponds to the neurological dysfunction. In this study we examined whether this increase in the ADC among TBI patients is because of diffuse injury to the brain or whether certain regions of the brain are more prone to injury compared to others. Our data here suggests that the neurological dysfunction as assessed by GCS is probably due to a diffuse injury throughout the brain that results from either movement of water protons to the extracellular compartment, or due to cell death. This is supported by the fact that all regions of the brain saw a significant increase in peak ADC values and correlated well with the GCS score. It is interesting to note that in the case of patients that presented with brain stem lesions there was no significant increase in the ADC's in the white matter but a significant increase in ADC in both the gray matter and the deep nuclei. This finding needs to be verified among a larger group of patients. This study, coupled with our previous study, establishes the fact that ADC histograms can be an independent predictor for assessing the TBI patients. Further studies with the use of techniques such as diffusion tensor imaging and fiber tracking may provide further insight into the injury process.

References

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