Correlation of Whole Brain Apparent Diffusion Coefficient with Glasgow Coma Score among Traumatic Brain Injury Patients

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Abstract
Quantitative whole-brain MR diffusion histograms in patients admitted with traumatic brain injury were evaluated and compared to those obtained from normal control subjects. Patients with normal MR imaging findings but low Glasgow coma score (GCS) showed an increased apparent diffusion coefficient (ADC). In addition, patients with lesions confined to the cortex that had minimal abnormal MR imaging findings but low GCS score showed significantly increased whole brain ADC's compared to similar group of patients with normal GCS score. A strong correlation between the ADC values and the GCS scores of the different groups was found.

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 low cognitive function as evidenced by the 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 explored the use of diffusion histograms of the whole brain to determine the differences in histograms between patients with TBI (acute) and normal controls and their relationship to the GCS.

Methods
Data from twenty-one patients (age 37.97±21.6), admitted to our trauma center who received DWI study were examined and compared with normal controls (n=11, age 32.7±19.2). These patients were further sub-divided into (1) Group A: normal imaging finding but low GCS (n=6, age 46±24.6); (2) Group B: brainstem abnormality and with low GCS (n=6, age 30.57±23.2; (3) Group C: patients whose lesions were confined to the cortex with normal GCS (n=6, age 42±22.7); and (4) Group D: the same as group C but with low GCS (n=3, age 44.8±22.3). ADC maps were generated on an off-line workstation using MEDX (Sensor Systems, Sterling, VA) image processing software. For each patient, regions outside the brain were removed using regions of interest. A histogram was then computed for each subject with a bin width of 0.6% of the maximum value of the ADC of 3.0 x 10⁻³ mm²/sec. Histograms were then normalized across subjects in various groups for differences in brain size. The histograms were plotted and the peak locations and the whole-brain average ADC were analyzed from within each group. A t-test between the control group and patients belonging to the four different injured groups was computed for each of the parameters at a p < 0.05 significance level. Correlations were also performed between the GCS score of each group and their corresponding peak and mean ADC values.

Results.
Figure 1a shows the ADC histogram differences between the normal group and patients exhibiting normal MR appearance but with low GCS (Group A). A significant difference (p<0.0075) in the maximum value of the histograms was seen between these groups with a significant skewness on the part of the low GCS patients. Figure 1b shows a similar pattern, albeit with less skewness was seen when the control group was compared with patients exhibiting brainstem abnormality (p<0.0055). The group of patients that had lesions confined to the cortex and had low GCS score also exhibited an increase in the ADC values compared to similar patients with normal GCS as shown in figure 1c but was not statistical significant (p<0.166). The average ADC did not exhibit significant differences from the control except for Group A (p<0.012). A strong correlation between the peak ADC of each of the groups and the GCS score (R²=0.91) and a somewhat weak but significant correlation was observed between the mean ADC and the GCS scores (R²=0.56) of the different groups.

Conclusions
It is not infrequent in TBI patients that exhibit low cognitive function but have normal morphological findings as evidenced by CT and MR. In such cases, ADC may be an independent indicator of TBI that corresponds to the neurologic dysfunction. Our preliminary data suggests a strong relationship between GCS and whole brain ADC. This finding needs to be verified among a larger group of patients.

References: