Traumatic Diffuse Axonal Injury: Radiological Diagnosis and Clinical Implications

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Background and Purpose

The usefulness of T2* GE-imaging for the evaluation and diagnosis of traumatic diffuse axonal injury (DAI) has been shown [1]. Due to the neuropathologically proven frequent hemorrhagic component [2] lesions suspicious of DAI appear as small hypointense signal alterations (traumatic microbleeds/TMBs) (**Figures 1/2**). The possible cognitive impairments of DAI need further clarification. The superiority of MR-imaging at high-field strength (3 T) remains to be proven.

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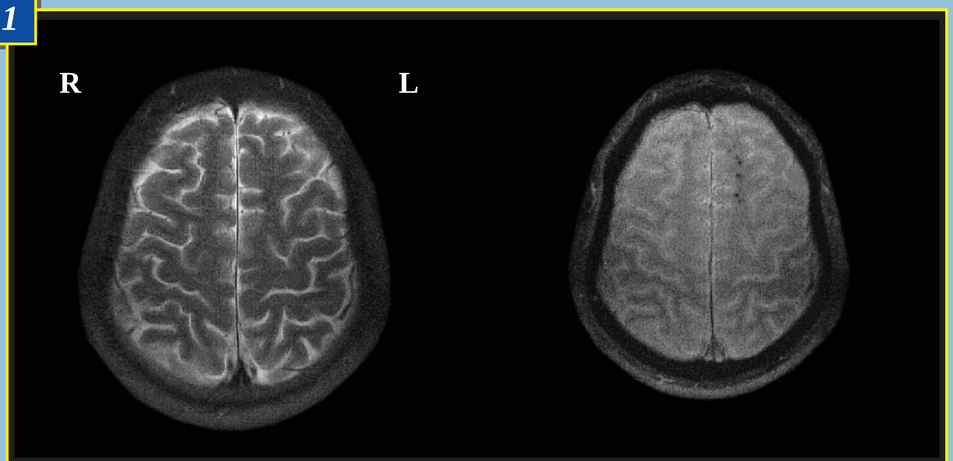

Multiple TMBs in the white matter of the left superior frontal gyrus. Left: T2-weighted image; right: T2-weighted image. Axial slices are in identical locations. Multiple TMBs that are clearly shown by T2* GE are not depicted by T2-weighted MRI. Note that there are no T2-hyperintense foci. 20-year-old man, traffic accident in May 1999 as passenger. The patient did not wear a seat belt. GCS-score 3; GOS-score 5.*

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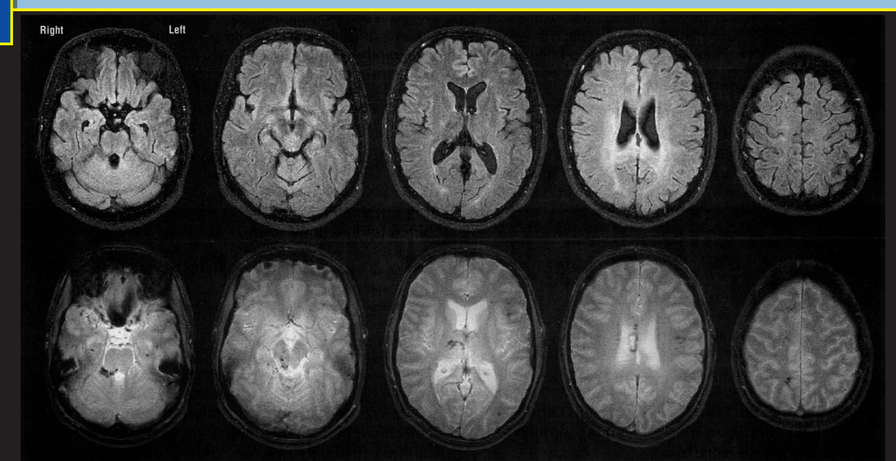

Comparison between FLAIR-images (upper row) and T2-weighted gradient-echo images (lower row). Depicted are axial view sections of exactly corresponding levels. Multiple traumatic microbleeds are shown on the T2*-weighted gradient-echo images. The patient was a 25-year-old driver of a car that collided with a roadworks vehicle in September 2003.*

Methods

Out of a databank with 299 TBI patients, 18 patients (age range 17-50 years, median initial Glasgow Coma Scale score 5) were identified, who showed a MRI lesion pattern compatible with pure DAI.
All patients underwent detailed neuropsychological testing. In addition, in 14 patients prospective MR-imaging at 1.5 T and 3 T was performed.
The images were independently evaluated by two readers for the occurrence of TMBs.

Results

All patients showed impairments of one or more cognitive subfunctions. No cognitive domain was fundamentally spared. (**Tables 1/2**) Memory- and executive dysfunctions were most frequent, the former reaching a moderate to severe degree in half of the patients.
In comparison, deficits of attention, executive functions, and short term memory were mostly mild. Correlations between the amount of TMBs and specific or global cognitive performance were absent. Comparative MR-imaging revealed 0.5-48.5, mean 17, median 7.5 TMBs at 1.5 T, and 2-118, mean 33.5, median 18.5 TMBs at 3 T, respectively (**Figure 3**).

Table 1

Scale	Patients, No.	Raw Score, Mean (SD; Range)	*z* Score, Mean (SD; Range)
Multiple Choice Vocabulary Test			
Correct items, No.	15	26.1 (2.8; 21 to 30)	−0.1 (0.5; −0.9 to 0.8)
TAP alertness subscale			
Mean reaction time, sec	18	299.4 (66.9; 202 to 424)	−1.5 (0.8; −2.4 to 0.1)
Variation or stability, sec	18	57.8 (24.3; 30 to 119)	−1.3 (0.7; −2.4 to 0.3)
Phasic alertness	18	0.07 (0.09; −0.09 to 0.25)	0.2 (0.9; −1.5 to 2.5)
TAP divided attention subscale			
Mean reaction time, sec	17	725.8 (93.8; 512 to 897)	−1.4 (0.8; −2.2 to 0.7)
Variation or stability, sec	17	225.2 (73.6; 144 to 432)	−0.4 (0.7; −2.0 to 0.6)
Omission	17	2.9 (2.7; 0 to 10)	−0.7 (1.1; −3.0 to 1.0)
Errors	17	1.4 (2.3; 0 to 10)	0.0 (1.1; −3.0 to 1.0)
BADS			
Sum	18	18.5 (3.1; 11 to 23)	−0.3 (1.4; −4.0 to 1.6)
Stroop			
Time, sec	13	156.2 (29.4; 98 to 215)	−1.5 (1.0; −2.8 to 0.8)
Spans			
Digit span forward	18	5.9 (1.1; 4.0 to 8.0)	−0.3 (1.2; −2.0 to 2.1)
Digit span backward	18	4.8 (1.0; 3.5 to 7.0)	−0.2 (0.8; −1.2 to 1.2)
Block span forward	18	5.6 (0.7; 4.5 to 7.0)	−0.2 (0.8; −1.9 to 0.8)
Block span backward	18	5.0 (0.8; 4.0 to 6.5)	−0.8 (0.8; −2.0 to 0.7)
CVLT			
Recalled items A1-5	18	48.1 (11.8; 22 to 63)	−1.7 (1.7; −4.5 to 0.6)
Recalled items A6	18	8.4 (3.3; 2 to 14)	−2.4 (1.8; −5.0 to 1.0)
Recalled items A7	18	9.5 (4.1; 2 to 15)	−2.0 (1.9; −5.0 to 1.0)
Recognition hits	18	14.6 (1.9; 8 to 16)	−1.1 (1.6; −5.0 to 1.0)
False positive	18	1.1 (2.0; 0 to 7)	−0.4 (0.9; −3.0 to 0.0)
WMS			
Verbal subscale, raw score	18	69.8 (17.9; 40 to 90)	−0.9 (1.3; −3.1 to 1.2)
Visual subscale, raw score	18	57.3 (6.0; 44 to 67)	−0.2 (1.0; −2.3 to 1.5)
Delay subscale, raw score	18	77.5 (12.7; 56 to 103)	−0.8 (1.3; −2.8 to 1.5)

Abbreviations: BADS, Behavioral Assessment of Dysexecutive Syndromes; CVLT, California Verbal Learning Test; TAP, Test of Attentional Processes; WMS, Wechsler Memory Scale–Revised.

Applied neuropsychological tests and patient's performance as raw scores and as compared to healthy controls (z-scores).

Table 2

Cognitive Subfunction	Unimpaired, No. (%)	Mild Impairment, No. (%)	Moderate Impairment, No. (%)	Severe Impairment, No. (%)	Cognitive Score, Mean (SD; Range)
Attention	4 (22)	9 (50)	2 (11)	3 (17)	0.55 (0.46; 0.00-1.57)
Executive functions	4 (22)	7 (39)	4 (22)	3 (17)	0.67 (0.51; 0.00-1.50)
Spans	8 (44)	8 (44)	1 (6)	1 (6)	0.24 (0.33; 0.00-1.25)
Learning and retaining	4 (22)	5 (28)	5 (28)	4 (22)	0.67 (0.54; 0.00-1.63)
Cognitive score	1 (6)	11 (61)	4 (22)	2 (11)	0.57 (0.35; 0.07-1.45)

*Unimpaired indicates z_i greater than −0.20; mild impairment, z_i greater than −0.60; moderate impairment, z_i between −0.60 and −1.0; severe impairment, z_i less than −1.0.

Frequency and levels of impairment with respect to the tested cognitive subfunctions and to an overall cognitive score (z_r-scores).

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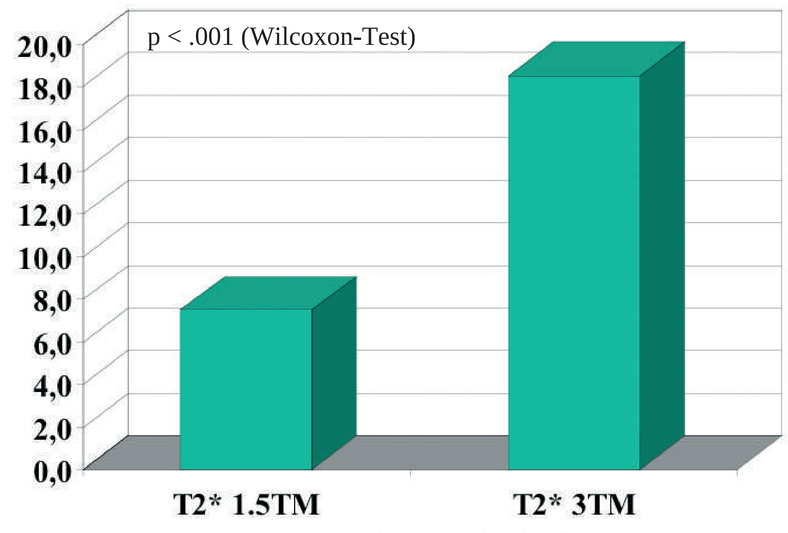

Comparison of MR-imaging at 1.5 T and 3 T for the the depiction of TMBs (median number of TMBs; n = 14).

Conclusions

DAI related brain lesions are mainly haemorrhagic. A MRI lesion pattern compatible with isolated DAI is associated with persistent cognitive impairment. The TMB-load is no sufficient parameter for the assessment of DAI-severity or functional outcome. T2* GE-imaging at high-field strength (3 T) is superior to current routine MRI (1.5 T) for the detection of DAI-related TMBs.

References:

[1] Scheid R, Preul C, Gruber O, et al. Diffuse axonal injury associated with chronic traumatic brain injury: Evidence from T2*-weighted gradient-echo imaging at 3 T. AJNR Am J Neuroradiol 2003; 24: 1049-1056.

[2] Adams JH, Doyle D, Ford I, et al. Diffuse axonal injury in head injury: definition, diagnosis and grading. Histopathology 1989; 15: 49-59.

[3] Scheid R, Walther K, Guthke T, et al. Cognitive sequelae of diffuse axonal injury. Arch Neurol 2006; 63: 418-424.