Correlation between MRI findings and long-term outcome in patients with severe brain trauma

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Abstract Our aim was to relate MRI findings in patients with severe traumatic brain injury (TBI) to clinical severity and long-term outcome. We studied 37 patients with severe TBI, who were submitted to clinical assessment for disability and cognition and to MRI 60–90 days after trauma. Clinical assessment was also performed 3, 6 and 12 months later. The number and volume of lesions in various cerebral structures were calculated semi-automatically from FLAIR and fast field-echo images. Possible correlations between total and regional lesion volume and clinical deficits were then investigated. The frontal and temporal lobes were most frequently involved. Total lesion volume on FLAIR images correlated significantly with clinical outcome, whereas that on FFE images did not.

Regional analysis showed that FLAIR lesion volume in the corpus callosum correlated significantly with scores on disability and cognition scales at the first clinical assessment. FLAIR lesion volume in the frontal lobes correlated significantly with clinical scores 1 year later.

Key words Traumatic brain injury · Magnetic resonance imaging · Pulse sequences

Introduction

Traumatic brain injury (TBI) is defined as severe when it causes unconsciousness for 6 h or more, or post-traumatic amnesia (PTA) lasting 24 h or more. Several studies have addressed prediction of survival and outcome in the very acute phase of severe TBI using clinical variables [1–3] or imaging [4, 5]. Mortality is high, ranging from 14% to 45% in reported series [6, 7]. Survivors have many long-term problems related to physical disability, memory, concentration, cognition and behaviour. Recovery is long: a significant proportion of patients are reported to improve over the first 6–12 months following injury [8, 9]. However, as persistent impairment of executive functions and speed of psychomotor processing causes loss of social autonomy and inability to return to work long after TBI, the social impact of such injuries is great.

While early outcome is affected mainly by hypotension, hypoxia, medical complications, cerebral oedema, intraventricular blood and mass lesions, other factors affect long-term outcome. Residual brain damage, as determined by the number, size and site of lesions, may contribute to outcome.

MRI has become the examination of choice in the study of TBI [10–15]. It shows a wider spectrum of traumatic lesions than CT [10]. The extent of brain damage is seen more clearly because both haemorrhagic...
and nonhaemorrhagic lesions can be detected, using appropriate sequences.

In this study we focussed on patients seen 60–90 days after severe TBI who had overcome problems in the acute phase, but still had severe clinical deficits. Our aim was to look at correlations between MRI findings in the postacute phase and clinical severity and long-term outcome.

Materials and methods

We enrolled 37 patients (22 men and 15 women, mean age 23 ± 8 years, range 12–48 years), consecutive admissions to our neurorehabilitation centre 60–90 days following a severe TBI.

Inclusion criteria were: Glasgow coma scale (GCS) score ≤ 8 in the first 24 h after the trauma; improvement in consciousness (spontaneous eye opening) after more than 1 week after trauma; post-traumatic amnesia (PTA) lasting > 4 weeks. Patients with a history of previous head trauma, alcoholism, drug abuse and neuropsychiatric disorders were excluded, as were those with an extra-axial haematoma, hydrocephalus and/or atrophy on the first CT or MRI. No patient had undergone a neurosurgical operation.

Patients were assessed using the GCS [16], Disability rating scale (DRS) [17] and Levels of cognitive functioning (LCF) [18] on admission to the neurorehabilitation centre (T0) and 3, 6 and 12 months later (T1, T2 and T3, respectively). Glasgow outcome scale (GOS) [19] assessment was performed at T3.

MRI was performed 60–90 days after the trauma, coinciding approximately with T3. It was performed at 1.5 Tesla: contiguous 5 mm slices were acquired in sagittal, coronal and axial planes. Pulse sequences included: proton-density (PD) and T2-weighted spin-echo (TR 2500 TE 30/90 ms), T1-weighted spin-echo (TR 500, TE 15 ms), T2-weighted fast field echo (FFE) (TR 600 TE 15 ms, flip angle 25°) and fluid-attenuated inversion-recovery (FLAIR) (TR 6000 TE 150 TI 2000 ms).

The images were evaluated by two neuroradiologists (AP and LMF).

Axial PD- and T2-weighted spin-echo, FLAIR and FFE images were transferred to a work station for semiautomatic quantitative measurement of the size of foci of abnormal signal. The areas of high signal (and low signal on FFE images) were calculated automatically on each slice by assigning a signal intensity threshold within the lesions. The volume of the lesions was then calculated on the basis of single areas, slice thickness and number of slices using dedicated software. These measurements were performed independently by two neuroradiologists (AP and MB) blinded to symptoms and clinical scores. (Since no significant differences were found between the two measurements, the mean values were used for further analysis.)

The total lesion volume was also subdivided into the following regions: brain stem, cerebellum, basal ganglia, corpus callosum, parietal, occipital, temporal and frontal lobes. When large lesions involved more than a single region, the lesion volume in each was calculated separately.

The development of clinical impairment was assessed by analysis of variance for repeated measures (RM-ANOVA). Differences in total lesion volume between PD- and T2-weighted spin-echo, FLAIR, and FFE and differences in regional lesion volume with a single sequence were analysed by the nonparametric Friedman test, a two-way analysis of variance by ranks for matched samples. Correlations between total and regional lesion volumes and clinical scores were analysed by linear and multiple regression, respectively. Given the exploratory nature of this work, we accepted 95% as the limit for statistical significance, without correction for multiple comparisons.

Results

The GCS, LCF and DRS showed significant progressive improvement in clinical status from T0 to T3 (RM-ANOVA: df = 3, F = 21.31, p < 0.0001; df = 3, F = 44.07, p = 0.0001; df = 3, F = 44.66, p = 0.0001, respectively). Post-hoc analysis (Fisher) showed that scores at T1, T2 and T3 were different from those at T0 (p < 0.05), and scores at T2 and T3 from those at T1 (p < 0.05), while no difference was observed between scores at T2 and T3 (Table 1). GOS at T3 showed scores ranging from 1 to 4 (mean 2 ± 1.1).

MRI showed areas of abnormal signal in 32 of the 37 patients on PD- and T2-weighted spin-echo and FLAIR, and in 31 on FFE images. Only in one patient were no lesions found on any sequence. Brain lesions evident as high signal on FLAIR images were detected on spin-echo PD- and T2-weighted images, but were less clearly defined (Fig. 1). We identified 11 small cortical areas of high signal, seen on FLAIR images, on spin-echo images only after reassessment. In 16 patients, more low-signal areas were seen on FFE than on the other images (Fig. 2).

Total lesion volume was 10.23 cm³ ± 16.3 on PD-weighted spin-echo, 9.85 cm³ ± 15.8 on T2-weighted

<table>
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<tr>
<th>Table 1</th>
<th>Clinical assessment over time</th>
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<tr>
<td>Test</td>
<td>Time of assessment</td>
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<tr>
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<td>T0 (60–90 days after injury)</td>
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<td>11.7 ± 2.7</td>
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* Significantly different from T0 (p > 95%, Fisher test)

* Significantly different from T1 (p > 95%, Fisher test)