Short term neurocognitive and symptomatic outcomes following mild traumatic brain injury: a prospective multicentre observational cohort study

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ABSTRACT

Objective

To determine the short term cognitive and symptomatic outcome following mild traumatic brain injury.

Setting

Emergency Departments of two UK tertiary referral hospitals.

Participants

Adult patients presenting to the Emergency Departments of the Royal London Hospital and Salford Royal Hospital with suspected traumatic brain injury within 24 hours, and Glasgow Coma Score >8. A non-TBI comparison group included adult patients with no head or neck injury.

Design

Prospective multi-centre cohort study.

Main Measures

The Standardised Assessment of Concussion (SAC), the Concussion Symptom Inventory (CSI), and total number of symptoms, measured at baseline and 72 hours.

Results

We enrolled 189 patients with and 51 patients without TBI. Patients with TBI had marked cognitive impairment which persisted at 72 hours (SAC score baseline 25 [23-27] vs 72 hours 25 [22-27]; p=0.1). Patients with TBI had persistent high symptom severity although this had decreased at 72 hours (CSI score baseline 9 [4-22] vs 72 hours 5 [1-19], p=0.002). A similar pattern was observed with the total number of symptoms (baseline 4 [2-8] vs 72 hours 0 [0-4]; p<0.001). Patients with TBI had worse neurocognitive function, higher overall symptom severity, and higher total number of symptoms compared with patients without TBI. Patients without TBI' neurocognitive function and

symptom severity remained constant, but number symptoms reduced between baseline and 72 hours.

Conclusion

There is a cognitive deficit and symptom burden in patients with mild TBI presenting to the Emergency Department which persists at 72 hours.

INTRODUCTION

Around 1.7 million patients attend emergency departments in the US and 1.4 million in the UK annually with a traumatic brain injury (TBI) [1, 2]. A further 500,000 to 800,000 patients a year are estimated to seek clinic and out-patient based care for TBI [3]. Approximately 90% of all TBIs are mild [4]. Mild TBI can result in persistent symptoms and have impact on return to work times following injury [5]. The cardinal features of mild TBI are acute and include alteration in level of consciousness and memory dysfunction, with resolution within 30 minutes. However, decreased cognitive function and symptoms such as headache and dizziness may persist for hours, days or sometimes weeks following the injury. This has been incompletely reported in Emergency Department populations [6]. We have limited understanding of the impact of mild TBI in Emergency Department patients on enduring cognitive deficits.

Mild TBI is an acute condition characterised by transient altered mental status and disorders of memory [7]. There is therefore a cognitive dysfunction associated with mild TBI. The evolution of neurocognitive dysfunction in the early phase of mild TBI is poorly understood. Several small studies report an immediate neurocognitive deficit, however most of these enrolled fewer than fifty patients with TBI and all were single centre studies [8-11]. In a population of more than a million patients in the US alone, many of whom are of working age, the consequences of failure to understand how neurocognitive dysfunction develops are enormous. Mild TBI has been called the silent epidemic because neurocognitive deficits that are not immediately apparent may persist [12]. There remains a need to understand how neurocognitive function deficits develop over the early period following injury. How neurocognitive function is affected in patients that attend the Emergency Department for non-neurological or non-neurotraumatic reasons is completely unknown. There may be a cognitive deficit associated with Emergency Department attendance for any reason.

The objective of this study was to study the cognitive function, symptom severity and number of symptoms in patients with mild to moderate TBI at baseline in the Emergency Department, and to re-evaluate them at 72 hours. The secondary objective of this study was to compare cognitive function, symptom severity and number of symptoms at both time points between patients with mild TBI and without TBI. We hypothesised that there would be an improvement in cognition, symptom severity and number of symptoms between baseline and 72 hours, and a difference between patients with and without mild TBI.

METHODS

Study design & Setting

This was a prospective observational cohort study conducted between September 2011 and March 2012 in the Emergency Departments of the Royal London Hospital and Salford Royal Hospital. Both hospitals are large university hospitals and designated Major Trauma Centres, which is equivalent to level one trauma centres. The annual Emergency Department patient attendance rates are 130,000 and 85,000 respectively. Data were collected as part of a study of a hand-held quantitative electroencephalogram designed for use in mild to moderate TBI. The study was approved by the National Research Ethics Service, North West 6 Research Ethics Committee, Greater Manchester South (reference 11/H1003/6).

Participants

Patients aged 18 to 80 years that attended the Emergency Department and were suspected of an acute traumatically induced structural brain injury and/or clinical manifestations of functional brain injury, as a result of insult to the head from an external force, including acceleration or deceleration movements without direct external trauma to the head, with a Coma Score of >8, within the last 24 hours, were included. Mild TBI was defined as GCS 13-15, and moderate TBI as GCS 9-12. Patients that had chronic neurological, psychiatric or cognitive conditions; temperature ≥37.7°C; critical illness; open head injury; received procedural sedation; were mechanically ventilated; receiving dialysis; in stage four chronic kidney disease; or pregnant were excluded. Patients without TBI were eligible if they were aged 18 to 80 and attended the Emergency Department with any condition excluding: an injury with any trauma above the clavicle; a history of road traffic collision requiring an Emergency Department visit; TBI within the past year; an primary acute neurological complaint or complaint of syncope. Those patients that had a CT scan of the head performed, had one done so in

line with national guidance [2]. Written informed consent was obtained from the patient, and in instances where the patient was unable to provide consent, consultee declaration to participate was obtained from a family member or the primary treating physician.

Screening and enrolment procedures occurred during Monday to Friday, between 0800hrs and 2000hrs due to availability of research staff. Sources of bias were minimised by measures taken to obtain a complete dataset, including data abstraction from medical and pre-hospital emergency services records; and discussion with medical and emergency medical service personnel.

Variables

Neurocognitive function was measured using the Standardised Assessment of Concussion (SAC). The SAC provides an objective reproducible and standardised report of the consequences of concussion [13]. The SAC is a paper-and-pencil assessment consisting of four domains (orientation, immediate memory, concentration, and delayed recall). It has a maximum score of 30, with higher scores indicating better neurocognitive function. It takes between five and ten minutes to complete. The SAC has been extensively validated for use in sport related concussion and has been reported as sensitive to sports concussion if administered within the first 48 hours [14-17].

Symptom severity and quantity was measured using the Concussion Symptom Inventory (CSI). The CSI is a list of 12 symptoms that are graded in severity by the patient on a seven point Likert scale [18]. The symptoms recorded are: headache; nausea; balance problems/dizziness; fatigue; drowsiness; feeling like “in a fog”; difficulty concentrating; difficulty remembering; sensitivity to light; sensitivity to noise; blurred vision; feeling slowed down. It has a maximum severity score of 72 with lower scores indicating lower severity, and a maximum symptom number score of 12. The total number of symptoms reported on the CSI, i.e. any symptom that did not have a score of 0, was calculated. Within group (baseline vs follow up) and between group (TBI vs non-TBI) comparisons were calculated. The comparison of numbers of concussion symptoms between groups was calculated because the sensitivity of the diagnosis of post-concussion syndrome is not limited to

patients that have sustained a concussion, and consequently it is possible that symptoms of acute concussion are not limited to head injured ED patients [19].

Clinical variables collected were assessed by the treating physician or research personnel, and utilised information from the patient, the prehospital medical record, and witness reports. Loss of consciousness and amnesia were dependent on collateral reports. Altered mental state was assessed by the treating physician. Previous TBI was determined as remembered by the patient and defined as 'head injury' with or without loss of consciousness.

The primary outcome measures were overall SAC scores, representing cognitive function, overall CSI scores, representing symptom severity, and total number of symptoms registered on CSI, representing symptom number. These were collected at baseline in the Emergency Department and at 72 hours, either face to face if the participant was an in-patient, or via telephone. Seventy-two hours was chosen as an appropriate follow-up time because it is a suitable time point to determine resolution of signs, duration of symptoms, and repeat CT scans, and also because there is little evidence on short term neurocognitive follow up in ED patients with mild TBI [10, 11, 20]. Demographic and clinical variables, mechanisms of injury and details of the TBI (loss of consciousness, amnesia) were obtained. Symptom presence was assessed using a list of 22 symptoms with binary yes/no answers to indicate presence or absence. A predefined subgroup of participants with acute intracranial haemorrhage on CT head scan was analysed. This was done to assess the cognitive changes in complex mild TBI, in which patients have a GCS of 13 or more but an acute intra-cranial haemorrhage on CT scan [21].

Statistical methods

Continuous data was compared using the paired or unpaired t-test, the related samples Wilcoxon Signed Rank test, or the independent sample Mann-Whitney U test as appropriate. 95% confidence intervals (CI) were calculated for differences between means. Categorical data was compared using the chi-squared test. Normally distributed data is represented as mean (standard deviation, SD). Non-

normally distributed data is represented as median [interquartile range, IQR]. Categorical data is represented as number (percentage). Normality was checked using the Shapiro-Wilk test and by visually assessing the frequency distribution. Analyses were performed using the R Project for Statistical Computing (https://www.r-project.org/). Significance was set at $p<0.05$. There was no imputation of missing data. Loss to follow up was managed with a whole group and longitudinal analysis.

RESULTS

A total of 240 patients were enrolled between September 2011 and April 2012. Of these, 189 patients presented with TBI and 51 patients were included as non-TBIs (figure 1). The mean age was 43 (16) years and 169 (70%) of participants were male. Demographic and clinical details of the TBI and non-TBI groups are given in tables 1, 2 and 3, and in supplementary material table 1. At Royal London 414 patients were screened of which 153 (37%) were recruited, and at Salford Royal 253 patients were screened of which 87 (34%) were recruited. Further detail is given in supplementary material table 2. It was not possible to complete the 72 hours assessment in 110 cases (46%), which comprised 88 (46%) in the TBI group and 22 (43%) in the non-TBI group. Of the 189 patients with TBI, 174 (92%) provided consent themselves on initial recruitment, 15 (8%) were recruited via consultee declaration and 7 (4%) of those were able to provide retrospective consent. No patients withdrew.

Neurocognitive and symptom data are presented in Table 4. Patients with TBI presented with marked neurocognitive impairment which did not improve between baseline and 72 hours. Patients with TBI had poorer neurocognitive function than non-TBIs at baseline (difference in SAC score 1, p=0.02, 95% CI -1.4 to -2.4), and at 72 hours (difference in SAC score 2, p=0.04, 95% CI -3.0 to 0.0) (figure 2). Patients with TBI also reported notably higher symptom scores than non-TBIs. Patients with TBI' symptom scores reduced significantly between baseline and 72 hours but were greater than those reported by non-TBIs at both time points (difference between TBI and non-TBIs in CSI score at baseline 9, p<0.001, 95% CI 8.4 to 13.7; and at 72 hours 5, p<0.001, 95% CI 5.7 to 11.6) (figure 3). Patients with TBI also had high total numbers of symptoms than non-TBIs at both time points (difference in total number of symptoms between TBI and non-TBIs at baseline 4, p<0.001, 95% CI 2.6 to 4.4; and 72 hours 4, p=0.001, 95% CI 1.9 to 4.1) (figure 4). The most frequently occurring symptoms were pain, headache and fatigue, which were experienced by more than 50% of TBI participants (figure 5).

Table 5 contains neurocognitive and symptom data for the subgroups with (CT+) and without (CT-) acute intracranial haemorrhage. Of the 189 TBI participants, there were 25 (13%) CT+ and 154 (87%) CT-. Mean age and gender was similar to the TBI and non-TBI group (43 [15] years, 18 [72%] males). Neurocognitive function was considerably worse in the CT+ compared to CT- subgroup at both time points (difference in SAC score between CT+ and CT- at baseline 3, p=0.009, 95% CI -1.0 to -3.0, and at 72 hours 3, p=0.009, 95% CI -1.0 to -5.0). CT+ patients also had higher symptom scores than CT- patients at baseline (difference in CSI 11, p = 0.01, 95% CI -15.0 to -2.0) and at 72 hours (difference in CSI 10, p = 0.06, 95% CI -13.0 to 0.0). CT+ patients also had greater numbers of symptoms compared with CT- patients at both time points (difference in total number of symptoms 4, p=0.027, 95% CI -4.0 to 0.0; and 3, p=0.038, 95% CI -5.0 to 0.0 at baseline and follow up respectively).

Sensitivity analyses

Sensitivity analyses using mild TBI definitions of GCS 13-15 (n=186) and 14-15 (n=183) were performed. There was no material difference in results when compared with the primary analysis (supplementary material tables 3 and 4). Further sensitivity analyses designed to apply the outcome measure in the lowest acuity patients were performed. In patients with GCS 14-15 that had a negative CT scan or no CT scan performed (n=162), there was no improvement in cognitive function or symptom burden between baseline and follow up (supplementary material table 5). This suggests that patients that qualify for a CT, even if their scan is normal, may have neurocognitive dysfunction and a symptom burden that persists for three days. In patients with GCS 14-15 that had a scan which was negative (n=71), not only cognitive function but also symptom scores and total number of symptoms remained unchanged between baseline and follow up (supplementary material table 6).

An analysis of the patients with TBI that completed the outcome assessments at both baseline and follow up (i.e. excluding the patients that were lost to follow up or were unable to complete an assessment) (n=99) showed no material difference compared to the primary analysis (supplementary material table 7). In a subgroup of patients with TBI that had sustained one or more previous head injuries (n=63) there was no change in cognitive function or symptom burden

between baseline and follow up (supplementary material table 8). This is in contrast to a subgroup of patients with TBI that had never had a previous head injury (n=111), where neurocognitive dysfunction persisted to follow up but symptom burden improved (supplementary material table 8). Three TBI subgroups consisting of CT not done, CT with no intracranial haemorrhage and CT with intracranial haemorrhage were analysed. There is a trend towards improved cognitive function and lighter symptom burden from intracranial haemorrhage to no CT performed (supplementary table 9).

DISCUSSION

The principal finding of this study was that patients with mild TBI have a clinically relevant neurocognitive deficit immediately after the injury that persists to at least 72 hours. A difference in SAC of two or more points is thought to be clinically relevant, although the SAC is not sensitive enough to pick up subtle changes in neurocognitive function, and there is a ceiling effect associated with its application [22, 23]. Patients with mild TBI also have persistently greater severity of symptoms and more concussive symptoms than patients without TBI, both of which also persist to 72 hours. Patients with TBI with acute haemorrhage on their CT scan had poorer neurocognitive function than those without.

To our knowledge, our study is the largest that enrolled patients with mild TBI and followed them over the short term. It is also the only multi-centre study that focuses on the neurocognitive effects of mild TBI in patients presenting to the Emergency Department. Neurocognitive function is usually measured either by psychological test that requires administration by a trained psychologist; by standardised paper and pencil tests such as the SAC; or by computer administered tests such as ImPACT. Our findings of a neurocognitive deficit immediately following mild TBI are similar to previously published studies, however a deficit persisting at 72 hours has not been reported before in this patient group.

When measured using paper and pencil tests, in studies enrolling 100 and 246 patients with TBI, there was a significant difference in neurocognitive function at baseline, but no follow up was performed [8]. In further studies of 29 and 49 patients with TBI, neurocognitive function had significantly improved by one month [10]. In a study of 62 patients presenting to the Emergency Department with concussion, cognitive function measured on the SAC improved between baseline and six hours later (from 21 to 24) [24]. The results reported in this latter study represent poorer baseline neurocognitive function than we report. This may be because the composition of the patients included in that study's population comprised a greater proportion of patients that

reported loss of consciousness and post traumatic amnesia compared with our sample, both of which have been associated with poorer SAC scores [25]. A study of 29 patients with TBI found a significant deficit compared with non-TBIs at around 31 hours post injury [11]. The same authors measured SAC at baseline post injury and a month later and reported significant improvement [10]. We report no improvement by 72 hours, however the authors' studies measured cognitive function at different time points to ours: a single observation at 31 hours; and follow up at one month. The results of our study taken with previous work implies a continuum of recovery, during which there is a neurocognitive deficit present up to and beyond 72 hours but which may resolve at some point before one month. This theory is backed up by the results of neurocognitive function testing by the computerised ImPACT programme, which showed gradual improvement in function measured at 24 hours, one week and three months post injury [26].

Normal values for SAC scores are primarily derived from athletes that completed the SAC prior to a sports season and therefore prior to any injury. A normal SAC varies from 27 to 28 [14-16]. Patients with TBI in our study had baseline and 72 hours SAC scores 2-3 points lower than this, and although the two populations are different, this represents a clinically relevant deficit. Our non-TBI group also had lower than normal SAC scores at baseline. However, they increased by one point, which was not a statistically significant increase, to 27, which seems to be the lower end of normal. We also report that there were significant differences in overall symptom severity as measured on the CSI, and total numbers of symptoms, between baseline and 72 hours, and between patients with and without TBI. These findings are in line with previously published work on symptom pattern post mild TBI, which suggests that both overall symptom severity and total number of symptoms may discriminate between patients with and without mild TBI [27]. However, our findings are important because we have reported the persistence of neurocognitive deficit in the largest group of hospital Emergency Department patients thus far described. In addition we reported several subgroup analyses which suggest that cognitive deficit persists regardless of whether the patient has a GCS of 13, 14, or 15; or whether the patient is in a presumed low acuity group (i.e. did not require or did not have a CT

scan); or whether they had a history of previous head injuries or not. Finally, we report that patients with mild TBI and intracranial haemorrhage have poorer neurocognitive function than those without intracranial haemorrhage.. This adds weight to the concept of complex mild TBI, i.e. mild TBI with positive findings on CT, and emphasises the importance of this group of patients [21].

Our study has strengths and weaknesses. To our knowledge it is one of the largest studies and the only multicentre study examining short term change in neurocognitive function following mild TBI. Although convenience sampling was necessitated based on resources, selection bias was minimised by approaching potential participants that had been admitted to hospital but were still within 24 hours of their TBI as well as by approaching all potential participants in real time. It was not possible to eliminate bias in the form of drop-outs or lost-to-follow-up, and consequently bias was quantified and is reported in figure 1. The lost-to-follow-up rate is high; 46% in the TBI group and 43% in the non-TBI group. This was because, for the purposes of the EEG study, follow up was to be at three days, i.e. 72 to 96 hours. This narrow window presented significant difficulties in contacting participants. The exclusion criteria could be said to be unnecessarily narrow. They are, however, in line with other similar studies [10]. For many participants, 72 hour follow-up was by telephone. Telephone based cognitive assessments are employed in cognitive research, particularly in screening for cognitive defects and dementia, however the SAC is not validated for use over the telephone. The proportion of follow ups completed by telephone was not recorded and so any difference between telephone and face-to-face follow groups is not known. There may be an element of learning that is dependent in part on visual stimulus, which clearly is missing during a telephone follow up. That learning for the SAC memory recall is partly dependent on visual stimulus is enforced by the observation that the domain that represented the greatest decrease in SAC between initial attendance and 72 hours in the non-TBI group was the delayed recall domain. This may explain the results seen that the non-TBI group had a wider SD between baseline and 72 hours. There were many more patients enrolled with than without TBI, which may introduce bias in comparisons between TBI and non-TBIs. This was partly because the primary outcome was the difference

between baseline and follow up within the head injured group, partly because the protocol for the EEG study required a lower number of patients without TBI, and partly because of the nature of convenience sampling. Whilst recognising this as a limitation, we do not believe that this is an insurmountable flaw in the methodology. Finally, because this was an analysis of data from a separate study, there was no specific sample size calculation associated with either TBI or non-TBI based endpoints.

Methods for assessing and managing acute mild TBI in the Emergency Department are varied. This reflects the uncertainty surrounding optimal management strategies. Decision making tools that help determine whether or not a patient should have a computed tomography (CT) scan of the head are based on studies that were designed to assess whether a patient has an intracranial haemorrhage, not whether or not they have concussion [2]. We report that neurocognitive dysfunction is associated with mild TBI but the speed of recovery and the repercussions on patients' work and home lives is still unknown. The clinical follow up for these patients is important. Leaflets explaining the likely clinical course and provision of access to TBI clinics may well contribute to an improvement in clinical variables [28].

CONCLUSION

Emergency Department patients with mild TBI experience cognitive deficit and concussive symptoms that persist to at least 72 hours. This has significant implications on the management of mild TBI, including the potential for early treatment, and explicit explanations to patients on what they can expect following 'normal' scan results. Further work evaluating the pattern of neurocognitive recovery, repercussions on home and work life, and management strategies is warranted.

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TABLES

	TBI	Non-TBI	*p*- value (95% CI)
Demographics			
Number	189 (79)	51 (21)	
Age	43 (16)	40 (15)	0.20 (-1.6 to 7.7)
Male Sex	133 (70)	36 (71	0.98 (-0.1 to 0.1)
Years in education	16 (4)	17 (5)	0.12 (-2.5 to 0.3)
Disposition			
Discharge home from ED	95 (50)	23 (45)	0.97 (-0.1 to 0.1)
Admission to CDU	37 (20)	15 (29)	0.13 (-0.04 to 0.2)
Admission to hospital	57 (30)	13 (26)	0.52 (-0.1 to 0.07)
Neurosurgery performed	1 (0.5)	N/A	
Previous head injury			
Total	62 (33)	10 (20)	0.016 (0.014 to 0.018)
One	39 (21)	6 (12)	0.13 (-0.2 to 0.13)
Greater than one	23 (12)	4 (8)	0.4 (-0.2 to 0.07)

Table 1

Demographics, and characteristics specific to TBI and non-TBI groups. Data are reported as number (%), or mean (standard deviation). TBI, traumatic brain injury; CI, confidence interval; ED, Emergency Department; CDU, clinical decision unit.

Mechanism of TBI	
Motor vehicle collision	20 (11)
Pedestrian struck by vehicle	20 (11)
Bicyclist	15 (8)
Fall	62 (33)
Other	41 (22)
TBI characteristics	
GCS 14-15	183 (97)
GCS 13	3 (1.5)
GCS 9-12	3 (1.5)
LOC	72 (38)
Seizure	3 (1)
PTA	64 (34)
RGA	34 (18)
AMS	90 (48)
Radiological characteristics	
CT performed	102 (54)
Of TBI group, CT+	25 (13)
Diagnosis within CT+ group	
EDH	5 (21)
SDH	6 (25)
SAH	3 (13)
Contusion and IPH	9 (38)
IVH	0 (0)
Mixed	1 (4)

Table 2

Characteristics of TBI group. Data are reported as number (%). TBI, traumatic brain injury; GCS, Glasgow coma score; LOC, loss of consciousness; PTA, post traumatic amnesia; RGA, retrograde amnesia; AMS, altered mental status; CT, computed tomography; CT+, acute haemorrhage seen on CT; EDH, extradural haemorrhage; SDH, subdural haemorrhage; SAH, subarachnoid haemorrhage; IPH, intra-parenchymal haemorrhage; IVH, intra-ventricular haemorrhage.

Presenting complaint (non-TBI group)	
Abdominal pain	12 (24)
Fracture/sprain/dislocation	11 (22)
Back/limb pain	10 (20)
Other	10 (20)
Chest pain	6 (10)
Laceration	3 (6)

Table 3

Presenting complaints of non-TBI patients

	TBI (n=189)				Non-TBI (n=52)			
	Baseline	72 hours	Difference *P* (95% CI)		Baseline	72 hours	Difference *P* (95% CI)	
SAC	25 (23-27)	25 (22-27)	0	0.1 (-0.4 to 1.2)	26 (24-28)	27 (24-29)	1	0.5 (-0.6 to 1.7)
CSI	9 (4-21)	5 (1-18)	4	0.002 (1.2 to 6.3)	0 (0-2)	0 (0-2)	0	0.3 (-0.5 to 3.4)
Total no. symptom	4 (2-8)	4 (1-6)	0	0.051 (-1.5 to 0)	0 (0-2)	0 (0-1)	0	0.15 (-0.1 to 0.9)

Table 4

Neurocognitive outcomes; symptom severity; and total number of concussive symptoms as measured in the concussion symptom inventory, in TBI and non-TBI groups at baseline and 72 hours. TBI, traumatic brain injury; SAC, standardised assessment of concussion; CSI, concussion symptom inventory; IQR, interquartile range. Maximum SAC score possible is 30, indicating best neurocognitive function; maximum CSI score is 72, indicating maximum symptom severity, and maximum number of symptoms possible is 12. Data are reported as median (interquartile range).

	CT+ (n=25)				CT- (n=164)			
	Baseline	72 hours	Difference	*P* (95% CI)	Baseline	72 hours	Difference	*P* (95% CI)
SAC	23 (22-26)	22 (19-24)	1	0.5 (-0.6 to 1.7)	26 (23-28)	25 (22-27)	1	0.2 (-0.6 to 1.2)
CSI	20 (11-30)	15 (6-21)	5	0.3 (-0.5 to 3.4)	9 (4-19)	5 (1-15)	4	0.006 (0.7 to 6.2)
Total no. symptom	8 (4-9)	6 (5-9)	2	0.14 (-0.1 to 0.9)	4 (2-7)	3 (1-6)	1	0.01 (0.2 to 1.7)

Table 5

Neurocognitive outcomes; symptom severity; and total number of concussive symptoms as measured in the concussion symptom inventory, in TBI patients with and without intracranial haemorrhage, at baseline and at 72 hours. CT+, acute intracranial haemorrhage; CT-, no acute intracranial haemorrhage; TBI, traumatic brain injury; SAC, standardised assessment of concussion; CSI, concussion symptom inventory; SD, standard deviation; IQR, interquartile range. Maximum SAC score possible is 30, indicating best neurocognitive function; maximum CSI score is 72, indicating maximum symptom severity, and maximum number of symptoms possible is 12.

FIGURES

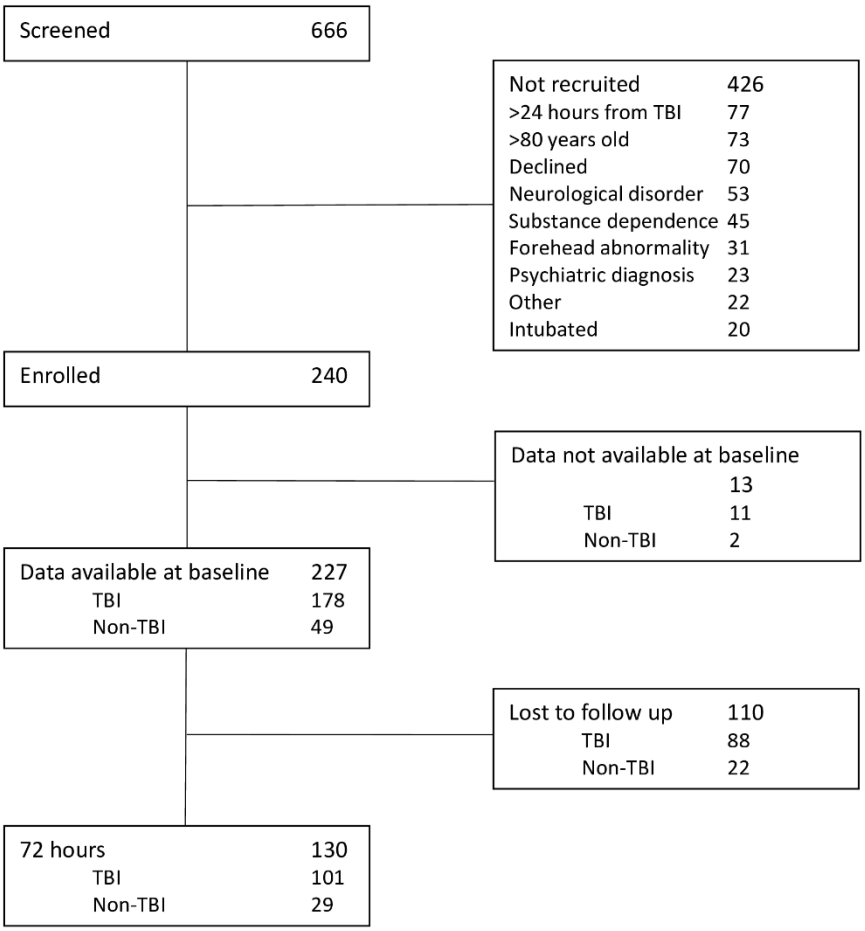

Figure 1.
Patient enrolment flow diagram.

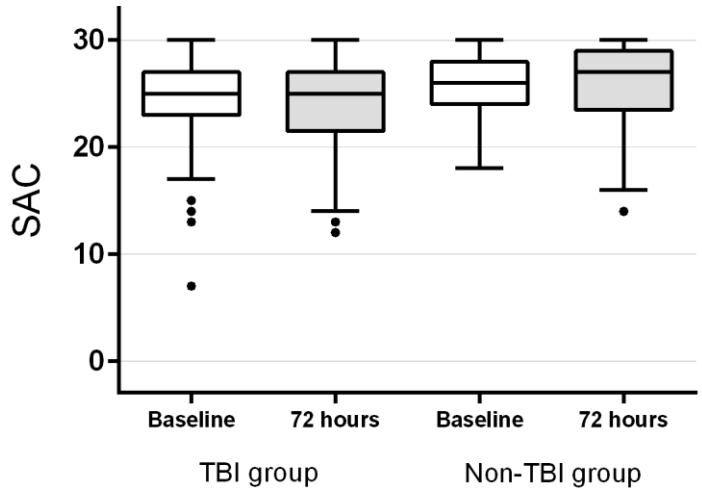

Figure 2.
Neurocognitive function. Box and whisker plot of standardised assessment of concussion (SAC) scores. The SAC is a neurocognitive test comprised of four domains (orientation, immediate memory, concentration, and delayed recall). Maximum SAC score possible is 30, indicating best neurocognitive function. Baseline is initial assessment within 24 hours. TBI, traumatic brain injury.

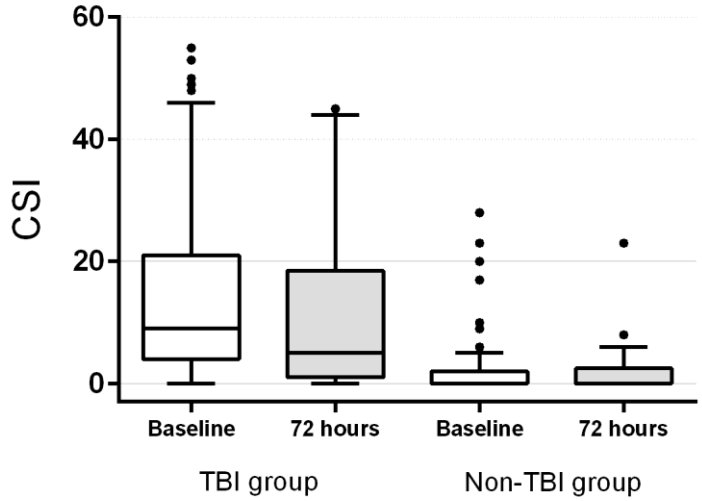

Figure 3.
Symptom scores. Box and whisker plot of Concussion Symptom Inventory (CSI). The CSI is a list of 12 symptoms, the severity of which patients self-report on a seven point Likert scale. Maximum CSI score is 72, indicating maximum overall symptom severity. Baseline is initial assessment within 24 hours. TBI, traumatic brain injury.

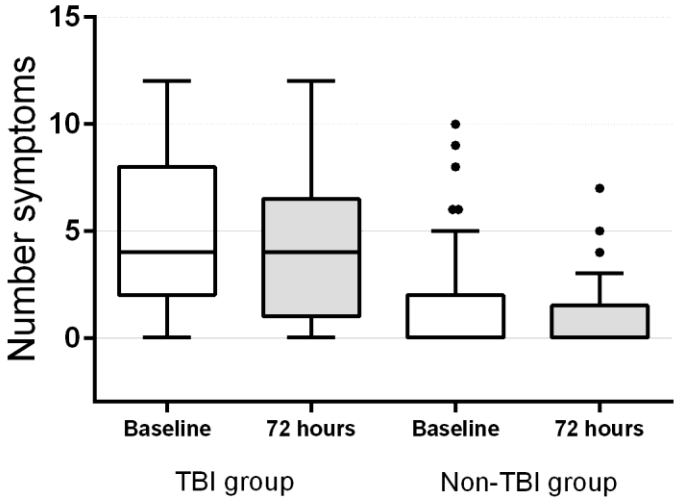

Figure 4.
Total number of symptoms. Box and whisker plot of number of symptoms. The maximum number of symptoms possible is 12. Baseline is initial assessment within 24 hours. TBI, traumatic brain injury.

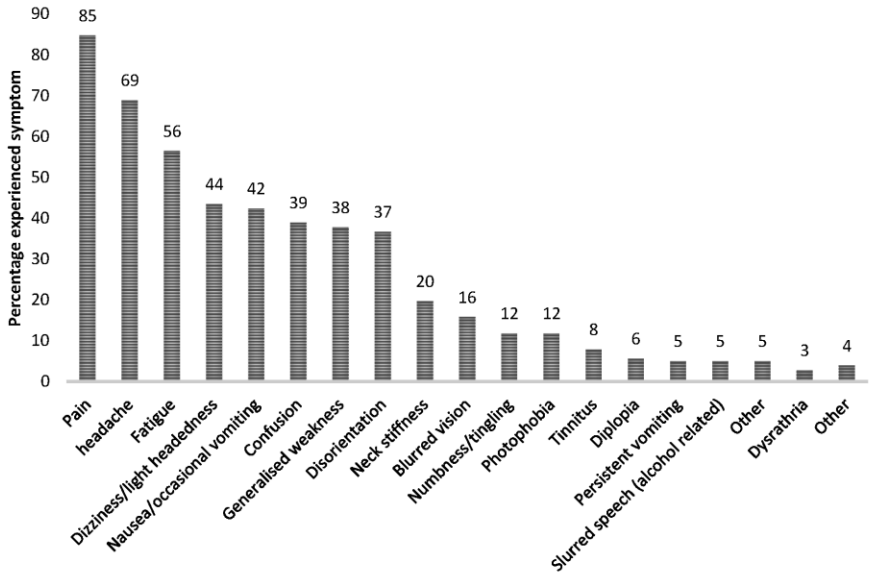

Figure 5.
Symptom frequency. The percentage of patients in the TBI group that experienced each symptom.

CONFLICT OF INTEREST STATEMENT:

No author states any conflict of interest

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Nil