PAPER

A history of loss of consciousness or post-traumatic amnesia in minor head injury: ''conditio sine qua non'' or one of the risk factors?

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Objective: A history of loss of consciousness (LOC) or post-traumatic amnesia (PTA) is commonly considered a prerequisite for minor head injury (MHI), although neurocranial complications also occur when LOC/PTA are absent, particularly in the presence of other risk factors. The purpose of this study was to evaluate whether known risk factors for complications after MHI in the absence of LOC/PTA have the same predictive value as when LOC/PTA are present.
Methods: A prospective multicentre study was performed in four university hospitals between February 2002 and August 2004 of consecutive blunt head injury patients (⩾16 years) presenting with a normal level of consciousness and a risk factor. Outcome measures were any neurocranial traumatic CT finding and neurosurgical intervention. Common odds ratios (OR) were estimated for each of the risk factors and tested for homogeneity.
Results: 2462 patients were included: 1708 with and 754 without LOC/PTA. Neurocranial traumatic findings on CT were present in 7.5% and were more common when LOC/PTA was present (8.7%). Neurosurgical intervention was required in 0.4%, irrespective of the presence of LOC/PTA. ORs were comparable across the two subgroups ($p>0.05$), except for clinical evidence of a skull fracture, with high ORs both when LOC/PTA was present (OR = 37, 95% CI 17 to 80) or absent (OR = 6.9, 95% CI 1.8 to 27). LOC and PTA had significant ORs of 1.9 (95% CI 1.0 to 2.7) and 1.7 (95% CI 1.3 to 2.3), respectively.
Conclusion: Known risk factors have comparable ORs in MHI patients with or without LOC or PTA. MHI patients without LOC or PTA need to be explicitly considered in clinical guidelines.

Head injury is one of the most common injuries seen in emergency departments, minor head injury (MHI) accounting for 90–95% of cases.[1 2] In a minority of patients, MHI is associated with neurocranial complications (6–20%). Neurosurgical intervention is rarely required (0.2–3.1%) and mortality is low (0.04–0.29%).[1 3–11] Definitions of MHI vary considerably, most commonly constituting blunt injury to the head and a normal to minimally altered level of consciousness on presentation (Glasgow Coma Scale (GCS) score = 13–15).[12 13] Loss of consciousness (LOC) and/or post-traumatic amnesia (PTA) are short, with a maximum duration of 15 and 60 min, respectively. Traditionally, a history of LOC or PTA is considered a ''conditio sine qua non'' for MHI.[14 15] The risk of neurocranial complications after head injury in patients presenting with a normal level of consciousness, no history of LOC and no PTA (ie, MHI *without* LOC or PTA) is estimated to be approximately a quarter of the risk in patients with (a history of) an altered level of consciousness or PTA (ie, MHI *with* LOC or PTA).[2 16] Consequently, MHI patients without LOC or PTA are commonly discharged without any imaging, observation or clinical evaluation by a neurologist.[17 18]

While this approach is probably justified for most MHI patients without LOC or PTA, some may have risk factors other than an altered level of consciousness, history of LOC or PTA that may increase their risk of neurocranial complications.[16 19 20] In MHI patients with LOC or PTA, risk factors for neurocranial complications have been well established, and these are commonly used as an indication for performing a head CT.[8 14 21 22] Arguably, these risk factors may also indicate the need for head CT in MHI patients without LOC or PTA, as is indeed recommended in some clinical guidelines for the use of CT in head injury.[1 14 23 24] However, since in MHI patients without LOC or PTA the prior probability of neurocranial complications is lower than in MHI patients with LOC or PTA, this approach may not be optimal. Also, the predictive values of risk factors derived from study populations of MHI patients with LOC or PTA may be biased by the presence of a history of LOC and/or PTA. Using the same risk factors for both MHI patient groups, irrespective of a history of LOC or PTA, as indications for CT may therefore lead to unnecessary CT scanning. On the other hand, CT scanning may be indicated in a selected group of MHI patients without LOC or PTA who are at increased risk of neurocranial complications, to reach a rapid and reliable diagnosis.

The purpose of the present study was to evaluate whether known risk factors for neurocranial complications after MHI with LOC or PTA have the same predictive value after MHI without LOC or PTA.

PATIENTS AND METHODS

Study population

Data were collected in four Dutch university hospitals on 3364 consecutively included patients (fig 1). Patients were included if they presented within 24 h after blunt head injury, were aged 16 years or older, had a GCS score of 13 or 14 on presentation to the emergency department or had a GCS score of 15 with at least one of the following risk factors: history of LOC, short term memory deficit, amnesia for the traumatic event, post-

Abbreviations: CCHR, Canadian CT Head Rule; GCS, Glasgow Coma Scale; LOC, loss of consciousness; MHI, minor head injury; OR, odds ratio; PTA, post-traumatic amnesia

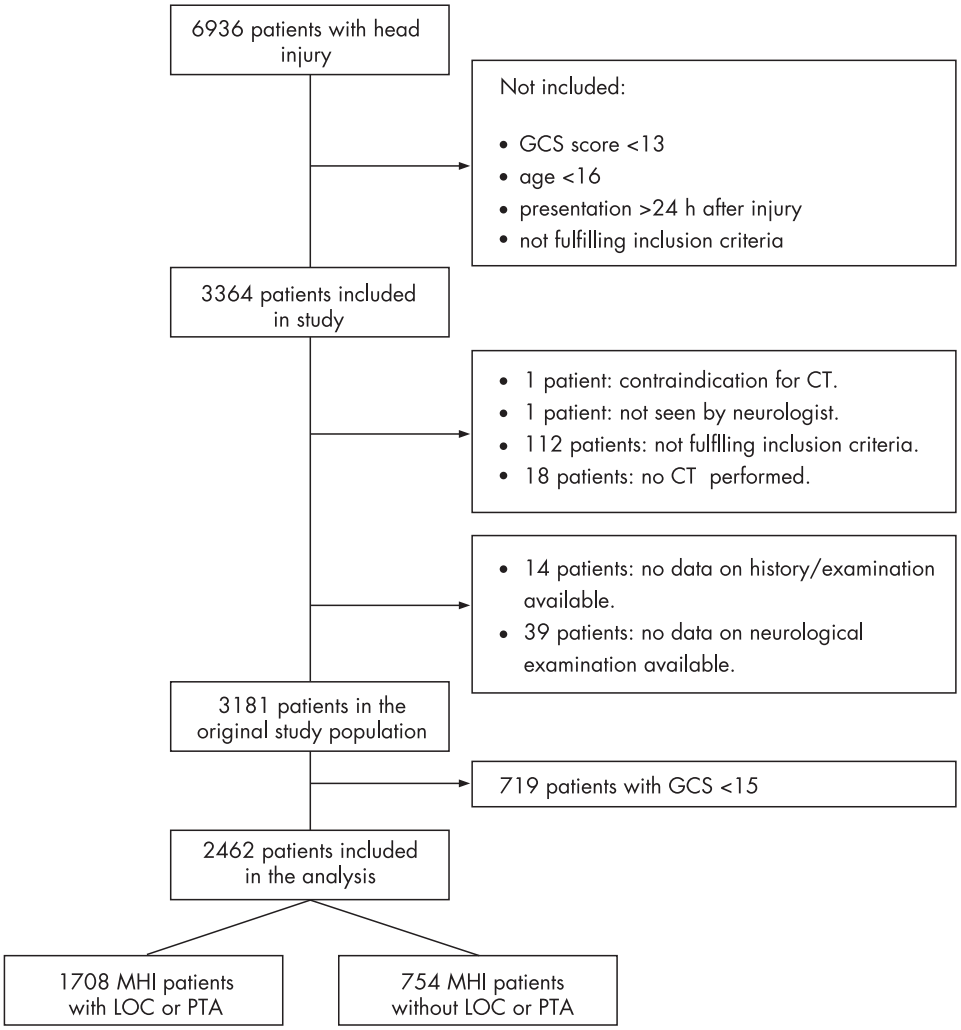

Figure 1 Flowchart of the study population. The number of patients presenting with head injury (6936) is an estimate based on the proportion of patients included out of the total number of trauma patients seen by a neurologist(-in-training) in the emergency department of the participating centre that included the majority of patients.

traumatic seizure, vomiting, headache, clinical evidence of intoxication with alcohol or drugs, anticoagulant treatment or history of coagulopathy, external evidence of injury above the clavicles or neurological deficit. Patients were excluded if there were contraindications for CT scanning or if CT of the head could not be performed because of concurrent injuries.

For the present study, patients with a GCS score of 15 on presentation were selected from the total study population. This study population was further divided into two groups: patients without a history of LOC or PTA (MHI without LOC or PTA) and those with a history of LOC or PTA (MHI with LOC or PTA).

Patient informed consent was waived by the Institutional Review Board and Medical Ethical Committee, after review of the study protocol, as patients meeting our inclusion criteria routinely undergo a head CT according to most local hospital policies and the EFNS guidelines.[1]

Patient assessment

All included patients were examined by a neurologist or by a neurologist-in-training under the supervision of a neurologist, after which all patients underwent a head CT, according to a routine trauma protocol. This consisted of a maximum slice thickness of 5 mm infra- and 8 mm supratentorially, without intravenous contrast administration. All scans were evaluated by a neuroradiologist or a trauma radiologist in bone and brain window settings. Data were collected on patient demographics, history of injury, presence of risk factors, GCS scores on and 1 h after presentation to the emergency department, as well as on CT findings.

Risk factor selection

Selection of risk factors was based on two published prediction rules for the use of CT in MHI, namely the New Orleans Criteria and the Canadian CT Head Rule (CCHR).[8 9] These were age, headache, vomiting, intoxication, persistent anterograde amnesia, PTA, injury above the clavicles (including clinical signs of skull or basal skull fracture), GCS <15 at 2 h post-injury and dangerous trauma mechanism (pedestrian versus motor vehicle, fall from height, ejected from motor vehicle). Additional risk factors commonly used in clinical guidelines for the management of MHI were also assessed.[1 14 17 18 23–25]

Definitions

A history of LOC was considered to be present when reported by a witness or by the patient. Amnesia for the traumatic event and PTA were defined as the inability to recall the traumatic event and subsequent events; its duration (in min) was estimated by the treating physician. Persistent anterograde amnesia was defined as the patient's inability to capture and retain any new information in memory. Post-traumatic seizure was classified as either a witnessed or suspected seizure having occurred after the head injury. Vomiting constituted any episode of emesis after the injury. Headache included both diffuse and localised pain. Presence and severity of intoxication

Table 1 Demographic characteristics and neurocranial traumatic CT findings

	MHI with LOC or PTA (n = 1708) (n (%))	MHI without LOC or PTA (n = 754) (n (%))	p Value
Demographics			
Age (y) (mean (range))	40.2 (16.0–94.2)	42.2 (16.2–102)	0.020
Male gender	1160 (67.9)	531 (70.4)	0.117
Traumatic CT findings	148 (8.7)	37 (4.9)	0.001
Skull fracture	82 (4.80)	28 (3.71)	0.229
Skull base fracture	37 (2.17)	8 (1.06)	0.059
Depressed skull fracture	7 (0.41)	4 (0.53)	0.679
Linear skull fracture	49 (2.87)	15 (1.99)	0.206
Subdural effusion	1 (0.06)	1 (0.13)	0.552
Subdural haematoma	29 (1.70)	8 (1.06)	0.231
Epidural haematoma	15 (0.88)	2 (0.27)	0.090
Traumatic subarachnoid haemorrhage	38 (2.22)	7 (0.93)	0.027
Intraparenchymal contusion	65 (3.81)	8 (1.06)	0.000
Haemorrhagic	51 (2.99)	7 (0.93)	0.002
Non-haemorrhagic	7 (0.41)	1 (0.13)	0.265
Diffuse axonal injury*	7 (0.41)	0 (0.00)	0.078
Intraventricular haemorrhage	3 (0.18)	0 (0.00)	0.250
Neurosurgical intervention	6 (0.35)	4 (0.53)	0.519
Intracranial CT findings only	114 (6.67)	21 (2.79)	0.000

LOC, loss of consciousness; MHI, minor head injury; PTA, post-traumatic amnesia.
Multiple findings may be present in one patient.
p Values were calculated with the independent sample t test for continuous variables and Pearson's χ^2 test for nominal variables.
*Diffuse axonal injury was defined as multiple, small, focal traumatic lesions in the typical locations of shearing injury (lobar white matter at the grey–white matter junction, corpus callosum, brainstem).

with alcohol or drugs were evaluated clinically, evidenced by slurred speech, alcoholic foetor or nystagmus. Anticoagulant treatment included coumarine derivatives only and not platelet aggregation inhibitors (eg, aspirin, clopidrogel); no blood coagulation tests were performed and the presence of coagulopathy was assessed by patient history. External evidence of injury consisted of extensive bruising or clinically significant discontinuity of skin; injury suspect of a fracture was classified as clinical signs of skull or facial fracture, whereas other injuries such as contusions, lacerations or abrasions were classified as skull or facial contusion. Focal neurological deficit was defined as any abnormality on routine clinical neurological examination indicating a focal cerebral lesion. High energy accident was derived from the description of the trauma mechanism and defined as: a fall from height (>1 m or >5 stairs), pedestrian or cyclist versus vehicle, ejected from vehicle, any motorised vehicle accident or high velocity cycling accident. Pre-traumatic seizure was also derived from the description of the trauma mechanism.

Outcome measures

Our primary outcome measure was any traumatic finding of the neurocranium on CT. A traumatic finding on CT that was considered clinically relevant was a secondary outcome measure, as was a traumatic CT finding that subsequently led to neurosurgical intervention. A clinically relevant finding on CT was defined as any intracranial finding due to trauma, including depressed skull fracture (ie, any neurocranial traumatic finding on CT except for an isolated linear skull fracture).[9 26] A neurosurgical intervention was defined as any neurosurgical procedure (craniotomy, intracranial pressure monitoring, elevation of depressed skull fracture, ventricular drainage) within 30 days after the traumatic event.

Data analysis

Missing patient data included in the analysis were assumed to be missing at random and imputed based on the available data means to avoid bias.[27] The proportion of imputed missing data was 3.6%, which included both items documented as unknown and items that were not documented.

We evaluated our patient population for demographic characteristics, mechanism of injury, traumatic findings on CT and for neurosurgical intervention. Differences between the two subgroups (MHI with versus MHI without LOC or PTA) were tested for significance using an independent sample t test for continuous variables and the Pearson's χ^2 test for nominal

Table 2 Indications for neurosurgical intervention

	MHI with LOC or PTA (n = 6) (n (%))	MHI without LOC or PTA (n = 4) (n (%))
Isolated depressed skull fracture	–	1 (25)
Epidural haematoma	4 (67)	1 (25)
Isolated	1 (17)	1 (25)
In combination with subdural haematoma	1 (17)	–
In combination with depressed skull fracture	2 (33)	–
Subdural haematoma	3 (50)	2 (50)
Isolated	1 (17)	2 (50)
In combination with epidural haematoma	1 (17)	–
In combination with depressed skull fracture	1 (17)	–

LOC, loss of consciousness; MHI, minor head injury; PTA, post-traumatic amnesia.

variables. A p value <0.05 was considered statistically significant.

To assess the association of each of the risk factors with the primary outcome measure, a common odds ratio (OR) was estimated using the stratified Mantel–Haenszel statistic for categorical variables and univariable logistic regression analysis for continuous variables. Homogeneity of the OR across the two subgroups was assessed with the Breslow–Day statistic (p<0.05 considered as an indication of heterogeneity) for categorical variables.[28] For continuous variables, the crude OR, as estimated with univariable logistic regression analysis, was compared with the OR adjusted for the presence of LOC or PTA. A difference of >10% between the crude and adjusted ORs was considered an indication of heterogeneity.[29]

Data were analysed using SPSS v12.0 software.

RESULTS

Between 11 February 2002 and 31 August 2004, an estimated 6936 patients presented with head injury to the emergency departments of the participating centres. A total of 3572 patients were not included because they did not meet the inclusion criteria of our study. Of the 3364 patients originally included in the study, 183 were excluded from further analysis for various reasons (fig 1). A further 719 patients presented with a GCS score of 13 or 14, leaving 2462 patients to be included in the analysis (fig 1). These included 1708 MHI patients with LOC or PTA, and 754 MHI patients without LOC or PTA. Patient characteristics are summarised in table 1.

Mean patient age was 40.8 years, MHI patients with LOC or PTA being slightly younger (40.2 years) than MHI patients without LOC or PTA (42.2 years; p = 0.02). Patients presented to the emergency department at an average of 97 min after the injury. The duration between the time of injury and presentation to the emergency department was not different for the two subgroups (99 min for MHI patients with versus 93 min for MHI patients without LOC or PTA, respectively; p = 0.42). The majority of patients were male (68.7%), which was not different between the two subgroups (67.9% of MHI patients with and 70.4% of MHI patients without LOC or PTA; p = 0.22) (table 1).

Neurocranial traumatic findings on CT were present in 185 patients (7.5%) and were more common in MHI patients with than in MHI patients without LOC or PTA (148 patients (8.7%) vs 37 patients (4.9%); p = 0.001). Neurosurgical intervention was required in 10 patients (0.4%) and was just as frequently needed in MHI patients with as in MHI patients without LOC or PTA (six patients (0.4%) and four patients (0.5%), respectively; p = 0.52) (table 1).

Indications for neurosurgery included isolated depressed skull fracture (n = 1), epidural haematoma (n = 4), subdural haematoma (n = 4) and a combination of epidural and subdural haematoma (n = 1) (table 2).

Univariable analysis of the associations for each of the risk factors with a neurocranial traumatic finding on CT is shown in table 3.

Risk factors indicating a significantly increased risk of neurocranial traumatic CT findings were pedestrian/cyclist versus vehicle, fall from (some) height, vomiting, PTA, a history of LOC, clinical signs of a skull or facial fracture, skull contusion, the presence of multiple injuries, focal neurological deficit, GCS score deterioration, anticoagulant treatment and

Table 3 Univariable analysis of common risk factors for neurocranial traumatic findings on CT

Variable	MHI with LOC or PTA (n (%))	MHI without LOC or PTA (n (%))	OR (95% CI)	p Value	p Value for heterogeneity
Trauma mechanism					
Pedestrian/cyclist versus vehicle	196 (11)	67 (8.9)	2.3 (1.6–3.4)	0.000	0.215
Fall from (some) height*	442 (26)	147 (19)	1.7 (1.2–2.3)	0.002	0.877
Ejected from vehicle	34 (2.0)	17 (2.3)	1.7 (0.7–4.0)	0.234	0.717
Symptoms					
Persistent anterograde amnesia†	174 (10)	21 (2.8)	1.1 (0.6–1.8)	0.806	0.257
Vomiting	166 (10)	65 (8.6)	2.5 (1.6–3.6)	0.000	0.844
PTA, average duration (min)	18.3	0.0	1.7 (1.3–2.3)‡	0.000	n/a
Loss of consciousness	1419 (83)	0 (0.0)	1.9 (1.3–2.6)	0.000	n/a
Headache					
Diffuse	719 (42)	286 (38)	1.1 (0.8–1.5)	0.467	0.447
Localised	275 (16)	160 (21)	1.3 (0.9–1.9)	0.199	0.107
Post-traumatic seizure	13 (0.8)	3 (0.8)	2.7 (0.8–9.8)	0.122	0.487
External evidence of injury					
Signs of skull fracture	35 (2.0)	12 (1.6)	25 (13–47)	0.000	0.028
Contusion of the skull	591 (35)	329 (44)	2.1 (1.6–2.9)	0.000	0.063
Signs of facial fracture	120 (7.0)	68 (9.0)	2.0 (1.3–3.2)	0.003	0.253
Contusion of face	874 (51)	400 (53)	1.0 (0.8–1.4)	0.922	0.638
Multiple injuries	373 (22)	173 (23)	1.8 (1.3–2.5)	0.000	0.814
Neurological examination					
Neurological deficit	146 (8.5)	61 (8.1)	1.8 (1.1–2.8)	0.011	0.230
GCS score deterioration at 1 h	40 (2.3)	10 (1.3)	3.9 (2.0–7.6)	0.000	0.723
Miscellaneous					
Age (y)	40.2	42.2	1.2 (1.1–1.3)§	0.000	n/a
Use of anticoagulant therapy	35 (2.0)	33 (4.4)	2.2 (1.0–4.5)	0.038	0.370
Intoxication					
Mild	151 (8.8)	91 (12)	0.8 (0.4–1.3)	0.355	0.712
Moderate	279 (16)	145 (19)	0.6 (0.4–0.9)	0.023	0.372
Severe	177 (10)	52 (6.9)	1.0 (0.6–1.7)	0.977	0.258

GCS, Glasgow Coma Scale; LOC, loss of consciousness; MHI, minor head injury; n/a, not applicable; OR, odds ratio; PTA, post-traumatic amnesia.
Shown are the prevalence of the risk factors, Mantel–Haenszel odds ratios (OR), p values of the ORs and p values for heterogeneity of the ORs across the two subgroups according to the Breslow–Day statistic
*Fall from (some) height included falls from any elevation.
†Persistent anterograde amnesia was defined as the inability to capture and retain any new information in memory.
‡Per 60 min of PTA.
§Per 10 years.

increased age. Clinical evidence of intoxication, however, indicated a reduced risk of neurocranial traumatic findings on CT. ORs were comparable across the two subgroups of MHI patients with or without LOC or PTA, which was demonstrated by homogeneity according to the Breslow–Day statistic: all p values for heterogeneity were larger than 0.05, except for clinical evidence of a skull fracture. The difference between the crude and adjusted ORs for age was 1%, indicating homogeneity of the ORs across the two subgroups. Although the ORs for signs of a skull fracture were different for the two subgroups—namely, 37 (95% CI 17 to 80) for MHI patients with and 6.9 (95% CI 1.8 to 27) for MHI patients without LOC or PTA—they both indicated a substantial and significantly increased risk of neurocranial traumatic findings on CT in both subgroups and the 95% CI overlapped considerably. Clinical evidence of a skull fracture predicted a skull fracture on CT in 71.4% (25/35) of MHI patients with and 25.0% (3/12) of MHI patients without LOC or PTA. In contrast, clinical evidence of a skull fracture was indicative of a depressed skull fracture on CT in only 8.6% (3/35) of MHI patients with and 16.7% (2/12) of MHI patients without LOC or PTA.

DISCUSSION

Our findings indicate that known risk factors for neurocranial complications after MHI have comparable ORs for patients with or without a history of LOC or PTA. The implication of this finding is twofold. Firstly, patients without a history of LOC or PTA are at risk of neurocranial complications after MHI, even occasionally requiring neurosurgical intervention. Neurosurgical intervention was in fact required just as often in patients with as in patients without LOC or PTA after MHI. Secondly, a history of LOC or PTA should be considered as one of the risk factors for neurocranial complications, and not as a ''conditio sine qua non'' for MHI.

A history of LOC or PTA is commonly used as a means of triaging MHI patients for referral to a neurologist/neurosurgeon, or for imaging or observation.[15 30] In many clinical guidelines for the management of MHI patients, it is recommended that patients without LOC or PTA, who have a normal level of consciousness on presentation and have no focal neurological deficit, are discharged without imaging or observation.[16 17] Our findings suggest that this approach is not justified if other known risk factors are present. The incidence of neurocranial traumatic findings on CT, however, was found to be lower in MHI patients without than in those with a history of LOC or PTA. Simply extending existing clinical guidelines to MHI patients without LOC or PTA therefore may lead to an unnecessary increase in CT scanning for MHI. Some clinical guidelines do recommend CT scanning of MHI patients with a risk factor, irrespective of a history of LOC or PTA.[1 14 23] In a previous validation study, we demonstrated that these guidelines have a very high sensitivity for identifying patients with neurocranial traumatic findings on CT, but also that specificity is extremely low, indicating that many patients are probably scanned unnecessarily.[31] In contrast with these guidelines, therefore, decision algorithms will need to be developed that may be implemented in clinical guidelines in which patients without a history of LOC or PTA are explicitly considered.[32]

We have reported ORs for variables that are commonly considered risk factors for complications after MHI, based on neurocranial traumatic findings on CT. In a meta-analysis of 35 papers containing more than 83 000 patients, Dunning *et al* reported relative risks for risk factors for intracranial injury in adults with MHI.[25] As the incidence of the outcome of interest is relatively low, ORs and relative risks are similar and may be compared.[33] For most of the risk factors we assessed, ORs were similar to the reported relative risks. For the variables post-traumatic seizure and intoxication, the ORs we observed were lower than those reported. For post-traumatic seizure, we estimated an OR of 2.7 which was not found to be statistically significant, whereas Dunning *et al* reported a relative risk of 6.4. Very few patients in our study population (n = 13) had a post-traumatic seizure, which may explain why the OR for this variable did not reach statistical significance. The reported relative risk of 6.4 does, however, fall within the 95% CI of our estimate, suggesting that the risk estimates are comparable. Clinical evidence of intoxication, however, was not associated with an increased risk of neurocranial complications in the present study, whereas a relative risk of 1.8 was reported by Dunning *et al*. As a large proportion of our study population was intoxicated, we cannot assign this difference in risk estimates to lack of data. The study populations included in the meta-analysis, however, had patients with GCS scores of 13–15, while all of our patients had a maximal GCS score on presentation. Clinically evident intoxication is often associated with a submaximal GCS score, which automatically places these patients in a high risk category.[14] Reported risks related to intoxication may therefore be associated with GCS scores, rather than with the intoxication itself. In their large study for the development of the CCHR, Stiell *et al* also failed to find an increased risk of intoxication.[9] They found that an unreliable neurological examination due to suspected intoxication was neither reliable nor discriminating and stated that the CCHR would be effective regardless of possible intoxication.

We found a very high predictive value for clinical evidence of a skull fracture, which was much higher for patients with than for patients without a history of LOC or PTA after MHI. This may indicate an interaction between the severity of the injury, as evidenced by the presence of LOC or PTA. In contrast, clinical evidence of a skull fracture was more often indicative of a depressed skull fracture on CT in MHI patients without than in MHI patients with LOC or PTA. This may be a result of selection bias, introduced by the fact that MHI patients without LOC or PTA required the presence of at least one risk factor to be included in our study; MHI patients with LOC or PTA were included irrespective of the presence of any risk factors other than LOC or PTA. One could argue that the need for neurosurgical intervention would be obvious if a depressed skull fracture were already clinically evident, and that the significance of the predictive value of this risk factor may be limited. However, in only a minority of patients with clinical evidence of a skull fracture was a depressed skull fracture actually present on CT. In the majority of patients with clinical signs of skull fracture, CT demonstrated a linear fracture. The association of a linear skull fracture and the development of extra-axial haematomas has been well established.[16 20 34] Clinical evidence of a skull fracture, therefore, may not only be regarded as indicative of a (depressed) skull fracture, but also needs to be considered as a risk factor for other important intracranial complications.

LOC and PTA were associated with ORs of 1.9 and 1.7, respectively. These risk estimates are in line with those reported previously.[25 35] The risk factors we assessed were not affected by the presence or absence of LOC or PTA. We therefore propose to use the variables LOC and PTA as another two risk factors for neurocranial complications, rather than using them as a means of triaging MHI patients. This is best achieved with a prediction rule, in which the presence of one or multiple risk factors may be used to estimate the patient's risk of neurocranial complications.[32] This risk assessment may then be used to decide on further management, such as clinical observation or CT scanning, that may further be based on analysis of the costs and effectiveness of several of these management strategies.[36]

The main limitation of our study was that MHI patients without LOC or PTA and without any further risk factors were not included, which may have biased the predictive values of the risk factors studied. This is inherent in our study design, in which we were bound by the currently implemented clinical guidelines for the use of CT in the Netherlands, that only indicate CT in patients without LOC or PTA if at least one other risk factor is present. The second limitation of our study was that we did not consider actual health outcomes, but limited our outcome measures to neurocranial complications and neurosurgical intervention. The relationship between neurocranial complications on CT and functional outcome is complex. For the purpose of our study, however, we feel that our pragmatic approach of only considering neurocranial traumatic CT findings and neurosurgical interventions as outcomes was sufficient.

CONCLUSION

Neurocranial complications after MHI, including those requiring neurosurgical intervention, occur both in patients with and in those without a history of LOC or PTA. Therefore, MHI patients without a history of LOC or PTA also need to be carefully evaluated and may also need imaging or clinical observation. Clinical guidelines for the management of MHI patients need to explicitly consider these patients without a history of loss LOC or PTA after MHI.

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