Characterizing the type and location of intracranial abnormalities in mild traumatic brain injury

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OBJECTIVE The incidence of intracranial abnormalities after mild traumatic brain injury (TBI) varies widely across studies. This study describes the characteristics of intracranial abnormalities (acute/preexisting) in a large representative sample of head-injured patients who underwent CT imaging in an emergency department.

METHODS CT scans were systematically analyzed/coded in the TBI Common Data Elements framework. Logistic regression modeling was used to quantify risk factors for traumatic intracranial abnormalities in patients with mild TBIs. This cohort included all patients who were treated at the emergency department of the Tampere University Hospital (between 2010 and 2012) and who had undergone head CT imaging after suffering a suspected TBI (n = 3023), including 2766 with mild TBI and a reference group with moderate to severe TBI.

RESULTS The most common traumatic lesions seen on CT scans obtained in patients with mild TBIs and those with moderate to severe TBIs were subdural hematomas, subarachnoid hemorrhages, and contusions. Every sixth patient (16.1%) with mild TBI had an intracranial lesion compared with 5 of 6 patients (85.6%) in the group with moderate to severe TBI. The distribution of different types of acute traumatic lesions was similar among mild and moderate/severe TBI groups. Preexisting brain lesions were a more common CT finding among patients with mild TBIs than those with moderate to severe TBIs. Having a past traumatic lesion was associated with increased risk for an acute traumatic lesion but neurodegenerative and ischemic lesions were not. A lower Glasgow Coma Scale score, male sex, older age, falls, and chronic alcohol abuse were associated with higher risk of acute intracranial lesion in patients with mild TBI.

CONCLUSIONS These findings underscore the heterogeneity of neuropathology associated with the mild TBI classification. Preexisting brain lesions are common in patients with mild TBI, and the incidence of preexisting lesions increases with age. Acute traumatic lesions are fairly common in patients with mild TBI; every sixth patient had a positive CT scan. Older adults (especially men) who fall represent a susceptible group for acute CT-positive TBI.

<https://thejns.org/doi/abs/10.3171/2017.7.JNS17615>

KEY WORDS traumatic brain injury; head injury; computed tomography; neurodegenerative diseases; common data elements; trauma

MILD traumatic brain injuries (MTBIs) are common in sports,⁹ daily life,¹⁵ and military service.¹⁸ The “mild” classification is delineated from more severe forms of TBI by a loss of consciousness (LOC) ≤ 30 minutes, a Glasgow Coma Scale (GCS) score ≥ 13, and posttraumatic amnesia (PTA) not exceeding 24 hours. MTBIs occur on a broad spectrum—from extremely mild injuries in sports from which an athlete seems to recover within hours or days, to a high-velocity injury in a motor

vehicle collision that abuts the moderate TBI severity range. The MTBI classification includes patients with a trauma-related macroscopic intracranial abnormality on a day-of-injury CT scan, such as an epidural hematoma, subdural hematoma, or contusion, as long as the operational criteria for mild injury are met based on LOC, GCS score, and PTA. This subgroup with acute traumatic intracranial abnormalities has been termed a “complicated” MTBI.⁶³ As a group, patients with complicated MTBIs are more likely to

ABBREVIATIONS CDE = Common Data Element; DAI = diffuse axonal injury; ED = emergency department; GCS = Glasgow Coma Scale; LOC = loss of consciousness; MTBI = mild traumatic brain injury; PTA = posttraumatic amnesia; WHO = World Health Organization.

SUBMITTED March 15, 2017. **ACCEPTED** July 6, 2017.

INCLUDE WHEN CITING Published online January 12, 2018; DOI: 10.3171/2017.7.JNS17615.

have early cognitive deficits^{2,23,29,63} and worse medium-^{62,63} and long-term⁵⁸ functional outcomes. However, there have been studies that have not found worse outcomes.^{24,31,37,43}

The incidence of acute CT-positive intracranial lesions in patients with MTBI varies widely between 4.7% and 38.9% across individual studies.^{25,54} The wide range in incidence is partially explained by varying enrollment of patients with lower GCS scores, because GCS scores < 15 are associated with an increased risk for intracranial injury.⁴² For those with GCS scores in the mild range (that is, 13–15), each 1-point drop in GCS score is associated with a substantially increased rate of intracranial abnormalities.⁶¹ Epidural hematomas, focal contusions, subarachnoid hemorrhages, and subdural hematomas are the most common CT-positive lesions seen in patients with moderate to severe TBI.^{36,45}

The frequency is lower but the distribution seems similar in patients with MTBI.^{17,55} In those who have sustained MTBIs, only approximately 1% of lesions require acute neurosurgery.^{21,52,55} Certain patient and injury factors have been associated with increased risk of trauma-related intracranial abnormalities after sustaining an MTBI, including older age,⁴⁷ preexisting medical conditions such as coagulopathy,^{26,40} alcohol intoxication at the time of injury,^{12,17} lower GCS score (that is, 13 or 14 vs 15),⁶¹ and dangerous causes of injury such as motor vehicle accidents and falls from a height.⁵⁵ The effects of other characteristics such as neurodegenerative diseases, brain atrophy, or chronic alcohol abuse on risk for traumatic intracranial lesions are not well known. In addition, the relative strength of association of characteristics such as age and cause of injury with CT findings is not well understood.

To systematically document and control for heterogeneity in TBI clinical trials, neuroimaging Common Data Elements (CDEs) were developed for TBI.^{10,16} The motivation for creating a CDE database was to enable the eventual characterization of the natural history and predictive factors in TBI. The structure of a CDE set for neuroimaging has implications for how patients entering clinical trials will be classified, stratified, and treated. To our knowledge, no study has described in detail the intracranial abnormalities after MTBI according to the CDEs.

The primary aim of this study was to describe the type and location of intracranial abnormalities (both acute and preexisting lesions) in a large generalizable inception cohort (n = 3023) of patients who sustained head trauma and underwent CT scanning in an emergency department (ED), providing incidence rates for the TBI neuroimaging CDEs. Secondary aims were to 1) compare and contrast the CT abnormalities in MTBI versus moderate to severe TBI, 2) investigate the association between preexisting and acute CT lesions, and 3) investigate the impact of common preinjury factors (such as age and alcohol abuse) on the incidence of acute intracranial lesions.

Methods

The patient pool for this study included all consecutive patients who were treated at the ED of the Tampere University Hospital between August 2010 and July 2012 and who underwent head CT after a suspected TBI (n =

3023; age range 0.3–103.8 years). The ED provides health services for a joint municipal authority of 22 municipalities (both urban and rural), with a catchment area of approximately 470,000 residents. The Tampere University Hospital is the second-largest trauma center in Finland and the only neurosurgical referral center in the area. Each year, the ED of Tampere University Hospital treats approximately 3000 patients with head injury. This includes everything from a bump to the head to severe surgically treated TBI. Approximately 6000 patients with head injury were treated during the data collection period. Ethics approval for the study was obtained from the Ethics Committee of Pirkanmaa Hospital District, Tampere, Finland.

Data were extracted retrospectively from hospital records. Participant-related data included age, sex, substance abuse, and diagnosed diseases. The data on preexisting and comorbid conditions were extracted from the hospital records related to the index injury and all available medical history from the records; no additional diagnostic testing was performed. Chronic alcohol abuse was defined according to codes F10.1 and F10.2 of the *International Classification of Diseases, Tenth Revision*. Injury-related variables included time of injury, time of arrival to the ED, cause of injury, and alcohol (breathalyzer and/or blood alcohol level) and/or narcotics intoxication at the time of injury. The cause of injury was categorized. Clinical variables related to TBI included eye-witnessed LOC, seizures, disorientation, PTA, and focal neurological deficits. The on-call ED physician identified amnesia by conducting an open interview regarding pre- and postinjury events. Only the presence or absence of amnesia was coded; the duration of amnesia was usually not noted in the records. No structured forms or standardized measures for amnesia were used.

We used the World Health Organization (WHO) definition of MTBI.³ Based on the chart review, we grouped patients into 3 categories according to their injury severity: 1) patients with head injury who were triaged to undergo head CT scanning but had missing data in their medical records relating to TBI severity indicators (these cases are called “suspected MTBI” hereafter), 2) patients who met WHO criteria for MTBI based on the details documented in their medical records (“confirmed MTBI”), and 3) patients with moderate to severe TBI (more severe TBI than defined by the WHO criteria, that is, GCS score < 13, LOC > 30 minutes, or PTA > 24 hours). When the chart review revealed evidence that the GCS score was probably lowered by non-brain injury factors (n = 92), TBI severity classification was made on the basis of LOC and PTA. This is consistent with WHO criteria, which state that “These manifestations of MTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries.”¹⁹ The GCS score obtained 30 minutes postinjury or upon presentation to the ED (whichever came later) was used in the present analyses.

In the ED, a noncontrast head CT was performed with a 64-row CT scanner (GE, LightSpeed VCT) for all consecutive patients with a head injury. Referral criteria for acute head CT were based on the 2000 Scandinavian guidelines for initial management of minimal, mild, and moderate head injuries.²² Head CT was performed in the ED within 72 hours of injury. All CT scans were analyzed and sys-

tematically coded by 2 neuroradiologists (A.B. and A.K.) using a structured and detailed case report form designed for this study. CT findings were defined as acute or pre-existing. Acute lesions included subdural hemorrhage and effusion, epidural hemorrhage and effusion, diffuse axonal injury (DAI) lesions, edema, compression of the CSF spaces, midline shift, contusions, hydrocephalus, pneumocephalus, skull and facial bone fractures, subarachnoid hemorrhage, nontraumatic intracranial hemorrhage, and acute ischemia. Preexisting findings included ischemic lesions, posttraumatic lesions, microangiopathy/small-vessel ischemic disease, general atrophy, and intracranial tumors. Preexisting small-vessel disease was considered if either periventricular or subcortical patchy low-density areas in cerebral white matter were found and compared with normal brain parenchyma with no mass effect. Study data were collected before the CDEs for TBI imaging were established. However, all CDEs possible with noncontrast structural CT scan were included.¹⁶

MTBI and moderate to severe TBI groups, as well as subgroups with versus without acute intracranial abnormalities, were compared using the chi-square test for proportions and t-tests for group mean differences. Unconditional logistic regression modeling was performed to estimate the odds ratios (ORs) and 95% CIs of acute intracranial abnormalities while controlling for multiple confounders (demographic data, clinical variables, and preexisting imaging features).

Results

Demographic data and clinical characteristics of the injury severity groups are presented in Table 1. The rates of trauma-related intracranial abnormalities were 11.6%, 16.1%, and 85.6% in the suspected and confirmed MTBI group, confirmed MTBI subgroup, and moderate to severe TBI group, respectively. All of the acute lesions in the suspected and confirmed MTBI group were from patients in the confirmed MTBI subgroup. Within the suspected and confirmed MTBI group, the percentages of patients with complicated MTBIs stratified by GCS scores were as follows: 15 (10.1%), 14 (36.1%), 13 (48.1%), and 11.5% in those patients for whom the GCS score was not available. Within the confirmed MTBI subgroup, the percentages of patients with complicated MTBIs stratified by GCS scores were as follows: 15 (14.2%), 14 (40.7%), 13 (52.0%), and 15.2% in those patients for whom the GCS score was not available. In the total sample, the number of patients and percentages of traumatic intracranial abnormalities, stratified by time between injury and CT scanning, were as follows: 0–2 hours (158 [29.3%]), 3–6 hours (129 [23.9%]), 7–11 hours (58 [10.7%]), 12–23 hours (49 [9.1%]), 24–47 hours (37 [6.9%]), and ≥ 48 hours (109 [20.2%]).

Specific CT findings stratified by the injury severity groups are presented in Table 2. In the total MTBI group, the incidence of specific abnormalities was as follows: subdural hematoma (8.4%), subarachnoid hemorrhage (5.0%), and contusion (3.9%). Of the 142 contusions (in 109 patients), 52.8% were frontal and 39.4% were temporal in location. Of the 495 subdural hematomas (in 232 patients), 38.8% were frontal, 21.8% were temporal, 22.0% were pa-

rietal, and only 7.5% were occipital. In comparison, in the moderate to severe TBI group, the incidence of specific abnormalities was as follows: subdural hematoma (63.0%), subarachnoid hemorrhage (55.6%), and contusion (43.6%). Of the 150 contusions (in 112 patients), 50.0% were frontal and 40.0% were temporal in location. Of the 439 subdural hematomas (in 162 patients), 32.1% were frontal, 24.8% were temporal, 21.0% were parietal, and only 9.8% were occipital.

The total MTBI group included 2766 patients with a suspected or confirmed MTBI. Patients in the subgroup of those with complicated MTBIs were older (mean $\pm$ SD 66.7 ± 19.9 years) than those with uncomplicated MTBIs (53.4 ± 24.5 years; $p < 0.001$), and men were significantly more likely to have complicated MTBIs than women (12.8% vs 10.1%, chi-square test 4.99, $p = 0.026$). Within the complicated MTBI group, the mechanisms of injury were as follows: motor vehicle accident (2.2%), traffic accident as pedestrian or bicyclist (7.5%), fall from a height (10.3%), and ground-level fall (66.3%). Older adults (≥ 55 years) who were injured in a ground-level fall had a greater incidence of trauma-related abnormalities than younger adults injured in a fall (17.0% vs 5.8%, chi-square test 85.89, $p < 0.001$). Rates of complicated MTBIs, stratified by age group, are presented in Fig. 1. Rates of complicated MTBIs increased with age.

The study population included 275 patients who were < 20 years old. Stratified by age, 27 patients were 0–5 years, 24 were 6–10 years, 82 were 11–15 years, and 142 were 16–19 years. Of the 275 patients < 20 years old, 19 had a traumatic lesion (6.9%): 6 in the 0–5 years age group (22.2%), none in the 6–10 years age group, 7 in the 11–15 years age group (8.5%), and 6 in the 16–19 years age group (4.2%). The most common lesions were subarachnoid hemorrhages and subdural hematomas. Subarachnoid hemorrhage was found in 8 patients (3 in the youngest group, none in the 6–10 years age group, 1 in the 10–15 years age group, and 4 in the oldest age group). There were 8 subdural hematomas: 4 in the 11–15 years age group and 4 in the 16–19 years age group. Each of the age groups had more males than females: 18 males in the 0–5 years age group, 16 males in the 6–10 years age group, 52 males in the 11–15 years age group, and 88 males in the 16–19 years age group.

Rates of preexisting abnormalities on CT, stratified by age group, are also presented in Fig. 1. Rates of preexisting lesions increased dramatically with age. In the total MTBI sample, 34.0% of patients had a preexisting abnormality, and 18.7% had > 1 preexisting abnormality. Small-vessel ischemic disease was present in 21.0% of patients, ischemic lesions in 7.2%, generalized atrophy in 17.9%, and posttraumatic lesions in 2.8%. Among older adults (≥ 55 years), those with CT evidence of preexisting atrophy were less likely to have a complicated MTBI than those without obvious atrophy (12.6% vs 19.0%, chi-square test 8.52, $p = 0.004$). The rate of complicated MTBIs in older adults (age ≥ 55 years) who had a preexisting neurodegenerative disease was similar to that in those who did not have a neurodegenerative disease (14.1% vs 17.6%, chi-square test 1.64, $p = 0.200$).

In a logistic regression analysis with the total MTBI sample ($n = 2766$), a number of variables were associated

TABLE 1. Characteristics of the study cohort

Variable	Suspected or Confirmed MTBI	Confirmed MTBI	Moderate to Severe TBI
No. of patients	2766	1990	257
Age, yrs	56.4 (34.2–77.1)	58.4 (34.7–77.8)	57.5 (40.0–74.9)
Time interval, hrs			
Injury to ED admission	4.5 (1.4–45.0)	4.5 (1.4–40.4)	2.5 (1.0–8.9)
Injury to head CT	6.5 (2.6–47.7)	6.5 (2.6–44.8)	4.3 (1.8–11.2)
ED admission to head CT	1.0 (0.6–1.8)	1.0 (0.6–1.9)	0.7 (0.4–1.2)
Head CT to discharge	6.0 (2.2–27.6)	6.6 (2.3–28.4)	88.1 (19.9–196.1)
Hospital stay	8.2 (3.8–29.1)	9.0 (3.9–31.2)	89.5 (22.1–197.6)
Sex			
Male	1542 (55.1)	1131 (56.8)	181 (70.4)
Female	1242 (44.9)	859 (43.2)	76 (29.6)
CT w/in 24 hrs	1865 (67.4)	1355 (68.1)	218 (84.8)
CT w/in 48 hrs	2076 (75.1)	1503 (75.5)	235 (91.4)
Cause of injury			
Motor vehicle collision	275 (9.9)	200 (10.1)	23 (8.9)
Traffic accident as pedestrian or bicyclist	145 (5.2)	118 (5.9)	22 (8.6)
Falls from a height	304 (11.0)	216 (10.9)	33 (12.8)
Ground-level falls	1454 (52.6)	1087 (54.6)	130 (50.6)
Other	535 (19.3)	324 (16.3)	29 (11.3)
Follow-up treatment			
Home	1215 (43.9)	813 (41.3)	28 (7.6)
Health center	628 (22.7)	489 (24.6)	28 (10.9)
Other health care facility	87 (3.1)	68 (3.4)	4 (1.6)
Hospital	809 (29.2)	599 (30.1)	172 (66.9)
Death	27 (1.0)	17 (0.9)	41 (16.0)
Alcohol intoxication			
Yes	660 (23.9)	520 (26.1)	81 (31.5)
No	1151 (41.6)	826 (41.5)	92 (35.8)
Unknown	955 (34.5)	644 (32.4)	84 (32.7)
LOC			
Yes	481 (17.4)	462 (23.2)	103 (40.1)
No	764 (27.6)	384 (19.3)	25 (9.7)
Not witnessed/unknown	1521 (55.0)	1144 (57.5)	129 (50.2)
Amnesia			
Yes	646 (23.4)	637 (32.0)	38 (14.8)
No	916 (33.1)	438 (22.0)	16 (6.2)
Unknown	1204 (43.5)	915 (46.0)	203 (79.0)
GCS score, points			
13–15	1682 (60.8)	1223 (61.5)	34 (13.2)
9–12	67 (2.4)	13 (0.7)	75 (29.2)
3–8	25 (0.9)	2 (0.1)	83 (32.3)
NA	992 (35.9)	752 (37.8)	65 (25.3)
CT			
Traumatic intracranial abnormality	320 (11.6)	320 (16.1)	220 (85.6)
Preexisting abnormality	941 (34.0)	717 (36.0)	44 (17.1)

NA = not available.

Values are expressed as median (interquartile range) age or time interval or as number (%) of patients.

with a higher rate of acute intracranial lesions. The results of the logistic regression analysis are presented in Table 3. In the single covariate models (producing unadjusted

ORs), male sex, older age, cause of injury, lower GCS score, chronic alcohol abuse, and preexisting traumatic lesions on CT were significant predictors.

TABLE 2. Intracranial abnormalities following TBI

Variable	Suspected & Confirmed MTBI	Confirmed MTBI	Moderate to Severe TBI
No. of patients	2766	1990	257
Abnormality/location			
Epidural hematoma*	6 (0.2)	6 (0.3)	15 (5.8)
Subdural hematoma*	232 (8.4)	232 (11.7)	162 (63.0)
Frontal	192 (6.9)	192 (9.6)	141 (54.9)
Temporal	108 (3.9)	108 (5.4)	109 (42.4)
Parietal	109 (3.9)	109 (5.5)	92 (35.8)
Occipital	37 (1.3)	37 (1.9)	43 (16.7)
Cerebellar	3 (0.1)	3 (0.2)	3 (1.2)
Tentorial	46 (1.7)	46 (2.3)	51 (19.8)
Subarachnoid hemorrhage*	139 (5.0)	139 (7.0)	143 (55.6)
Intraventricular*	33 (1.2)	33 (1.7)	53 (20.6)
Basal cisterns	24 (0.9)	24 (1.2)	36 (14.0)
Convexity sulci	113 (4.1)	113 (5.7)	124 (48.2)
Contusion*	109 (3.9)	109 (5.5)	112 (43.6)
Frontal	75 (2.7)	75 (3.8)	75 (29.2)
Temporal	56 (2.0)	56 (2.8)	60 (23.3)
Parietal	6 (0.2)	6 (0.3)	3 (1.2)
Occipital	1 (0.04)	1 (0.1)	2 (0.8)
Cerebellar	2 (0.1)	2 (0.1)	8 (3.1)
Brainstem	2 (0.1)	2 (0.1)	2 (0.8)
DAI*	5 (0.2)	5 (0.3)	9 (3.5)
CSF space compression*	35 (1.3)	35 (1.8)	75 (29.2)
Ventricles*	22 (0.8)	22 (1.1)	58 (22.6)
Basal cisterns*	5 (0.2)	5 (0.3)	41 (16.0)
Convexity sulci	22 (0.8)	22 (1.1)	61 (23.7)
Fractures	168 (6.1)	142 (7.1)	112 (43.6)
Skull fractures*	76 (2.7)	75 (3.8)	95 (37.0)
Facial fractures	103 (3.7)	78 (3.9)	44 (17.1)
Pneumocephalus	10 (0.4)	10 (0.5)	20 (7.8)
Hydrocephalus	8 (0.3)	6 (0.3)	6 (2.3)
Nontraumatic hemorrhage	16 (0.6)	13 (0.7)	16 (6.2)
Acute ischemia*	24 (0.9)	17 (0.9)	4 (1.6)
Midline shift*	45 (1.6)	44 (2.2)	89 (34.6)
Diffuse edema*	2 (0.1)	1 (0.1)	15 (5.8)
Preexisting abnormalities			
Preexisting posttraumatic lesions	77 (2.8)	62 (3.1)	5 (1.9)
Preexisting ischemic lesions	198 (7.2)	159 (8.0)	6 (2.3)
Microangiopathy/small-vessel ischemic disease	581 (21.0)	441 (22.2)	26 (10.1)
Generalized atrophy*	495 (17.9)	377 (18.9)	25 (9.7)
Tumors	21 (0.8)	17 (0.9)	1 (0.4)
Summary			
Any traumatic intracranial abnormality	320 (11.6)	320 (16.1)	220 (85.6)
>1 traumatic intracranial abnormality	128 (4.6)	128 (6.4)	144 (56.0)
Any preexisting abnormality	941 (34.0)	717 (36.0)	44 (17.1)
>1 preexisting abnormality	518 (18.7)	400 (20.1)	27 (10.5)
Any traumatic intracranial & preexisting abnormality	146 (5.3)	133 (6.7)	37 (14.4)
Any traumatic intracranial or preexisting abnormality	1212 (43.8)	904 (45.2)	227 (88.3)

Values are expressed as the number (%) of patients.

* Pathoanatomical lesions listed within the CDEs.²⁹

FIG. 1. Graph comparing incidence of intracranial lesions by age in the whole cohort (n = 3023). Gray line indicates acute intracranial lesions; black line indicates preexisting intracranial abnormality.

In the multivariable model, significant independent predictors were male sex (OR 1.68, 95% CI 1.27–2.21), age (OR 1.04, 95% CI 1.03–1.05), having a preexisting traumatic lesion (OR 1.99, 95% CI 1.12–3.56), chronic alcohol use (OR 1.84, 95% CI 1.29–2.63), certain mechanisms of injury (being a pedestrian or a bicyclist in a traffic accident [OR 7.54, 95% CI 3.05–18.65], or falling from a height [OR 4.32, 1.82–10.23], ground-level falls [OR 3.9, 95% CI 1.75–8.68] vs a motor vehicle accident), and a GCS score of 14 (OR 4.35, 95% CI 2.69–7.05) or 13 (OR 7.70, 95% CI 3.26–18.18) versus 15.

Interestingly, cerebral atrophy was associated with significantly lower risk for an acute traumatic intracranial abnormality (OR 0.48, 95% CI 0.34–0.68). Small-vessel ischemic disease was also associated with lower risk for having a complicated MTBI (OR 0.58, 95% CI 0.41–0.81). Of note, the multivariable model indicated that age and sustaining an MTBI due to a ground-level fall were independently associated with having a positive head CT scan, suggesting that the high rate of CT abnormalities associated with ground-level falls was not entirely confounded by older adults having more falls than younger adults.

Discussion

MTBIs vary in terms of mechanisms, pathophysiology, and clinical outcomes.^{32,35} We have provided a detailed description of CT findings in patients with MTBI (with a reference group of patients with moderate to severe TBI) and documented factors associated with CT abnormalities in patients with MTBI. This study has several strengths. It involved a large unselected sample of patients with head

injury who were consecutively treated in an ED. Other than requiring a clinically indicated CT scan of the head, we did not apply eligibility criteria, making the study findings generalizable and enabling us to examine the association between a large number of factors and the incidence of intracranial abnormalities.

Another strength of this study was that traumatic lesions were systematically coded using prespecified CDEs recently developed for TBI research.^{10,16} We also included a comparison group of patients with moderate to severe TBI. A key finding was that a substantial number of patients who sustained an MTBI had traumatic intracranial lesions on head CT, even those without clear documentation of acute clinical signs of MTBI in their ED medical record. No individual historical or physical examination features can completely rule out intracranial injury following minor head trauma.¹³ In our study, 11.6%–16.1% of patients with MTBI had an acute intracranial lesion, which falls within the range of prior studies.^{1,27,33,41,49,53,54,59} We further observed that the type and location of traumatic lesions are similar across the whole severity spectrum of TBI. Subdural hematoma, subarachnoid hemorrhage, and contusions occurring in the frontal and temporal regions are most common in MTBI and more severe TBIs.

It is unclear the extent to which macrostructural lesions help explain the heterogeneity of clinical outcomes from MTBI. Widely variable patterns of pathology may be seen on structural imaging in patients with similar injury severity as assessed by GCS scores.⁴⁸ A few prior studies have included CT positive versus negative as a prognostic factor. Some studies have suggested that CT abnormalities are associated with worse outcome,^{23,63} but some studies have

TABLE 3. Logistic regression analysis of acute intracranial lesions in 2766 patients with suspected and confirmed MTBI

Predictor (no. [%])	Unadjusted			Adjusted		
	p Value	OR	95% CI	p Value	OR	95% CI
Sex						
Female (1242 [44.9])	Ref			Ref		
Male (1524 [55.1])	0.026	1.31	1.03–1.66	<0.001	1.68	1.27–2.21
Age, yrs	<0.001	1.03	1.02–1.03	<0.001	1.04	1.03–1.05
Cause of injury						
Motor vehicle collision (275 [9.9])	Ref			Ref		
Traffic accident as pedestrian or bicyclist (145 [5.2])	<0.001	7.59	3.19–18.11	<0.001	7.54	3.05–18.65
Fall from a height (304 [11.0])	<0.001	4.66	2.03–10.72	0.001	4.32	1.82–10.23
Ground-level fall (1454 [52.6])	<0.001	6.54	3.04–14.04	0.001	3.90	1.75–8.68
Other (535 [19.3])	0.019	2.68	1.18–6.12	0.008	3.15	1.34–7.39
Unknown (53 [1.9])	<0.001	7.83	2.77–22.11	0.032	3.36	1.11–10.21
Alcohol use preceding injury						
No (1151 [41.6])	Ref			Ref		
Yes (660 [23.9])	0.297	1.17	0.87–1.57	0.559	1.12	0.76–1.64
Unknown (955 [34.5])	0.625	1.07	0.82–1.40	0.579	1.09	0.81–1.46
Chronic alcohol abuse (488 [17.6])	<0.001	1.63	1.24–2.15	<0.001	1.84	1.29–2.63
GCS score						
15 (1558 [56.3])	Ref			Ref		
14 (97 [3.5])	<0.001	5.00	3.20–7.81	<0.001	4.35	2.69–7.05
13 (27 [1.0])	<0.001	8.23	3.80–17.82	<0.001	7.70	3.26–18.18
3–12 (92 [3.3])*	—	—	—	—	—	—
Unknown (992 [35.9])	0.282	1.15	0.89–1.49	0.860	1.02	0.78–1.34
Small-vessel ischemic disease (581 [21.0])	0.154	1.22	0.93–1.61	0.001	0.58	0.41–0.81
Neurodegenerative disease (229 [8.3])	0.236	1.27	0.86–1.88	0.750	0.93	0.60–1.45
Preexisting traumatic lesion on CT (77 [2.8])	<0.001	3.00	1.79–5.02	0.02	1.99	1.12–3.56
Cerebral atrophy (495 [17.9])	0.967	0.99	0.73–1.35	<0.001	0.48	0.34–0.68

Unadjusted parameter estimates were derived from single-predictor models. Adjusted parameters were derived from a model containing all predictors and therefore refer to the independent effect of a given predictor, controlling for other predictors.

* There were no intracranial lesions in this subgroup.

presented opposite results.⁵⁶ Few studies have examined the relationship between specific CT findings and outcome.⁶⁵ Further research is needed on neuroimaging as a prognostic factor. Consideration of other biopsychosocial factors (e.g., preinjury mental health, sex, personality factors, resilience, and acute postinjury psychological distress) in a multivariate model will be important in prognostic research.^{4,51}

Preexisting nontraumatic lesions were common in patients with TBI, but they were not associated with increased risk of sustaining a CT-positive MTBI. Specifically, preexisting, degenerative, and ischemic lesions in the brain did not increase the risk for acute traumatic lesions. Instead, the underlying increased risk for intracranial abnormalities associated with aging may be caused by other comorbidities and antithrombotic medication. Poorer health and functioning predict the occurrence of TBI in elderly persons.⁸ A common belief is that cerebral atrophy is a risk factor for acute and chronic subdural hemorrhage. To our knowledge, only 1 study has suggested that cerebral atrophy may predispose to traumatic intracranial hemorrhage.¹¹ In our study, atrophy was not associated with risk for hemorrhage; in fact, the association was the opposite.

Advanced age is a well-known risk factor for an intracranial abnormality in head injury.^{7,17,38–40,46,55,57,61} The present study highlights that older age increases the risk of any CDE intracranial abnormality fairly linearly across adulthood, although such risk may plateau at approximately 60–70 years of age. Falls are the leading cause of TBI among elderly people.^{5,7,28,57,60} The risk of falling could be associated with intrinsic risks that occur with aging (e.g., chronic medical conditions, impaired balance, slower reaction times, decreased muscle strength, impaired cognition, and use of medications that predispose patients to postural hypotension and dizziness).

Similar to previous studies, chronic alcohol abuse increased the risk of having an acute traumatic lesion on head CT. Acute intoxication, however, did not increase the risk of intracranial abnormalities. Alcohol abuse is common before TBI, with a prevalence of 37%–51%.⁴⁴ In our study, 18.4% of patients had a history of alcohol abuse. Both acute and chronic alcohol abuse may impair decision making, motor control, and inhibitory control.⁵⁰ Binge drinking is the most common pattern of drinking among trauma patients, and the injuries of such patients

typically result from assaults, traffic accidents, and falls.⁵⁰ Those who have severe alcoholism may not present at the ED as often as occasional binge drinkers after a head injury. Chronic alcohol abusers are often brought to the ED by emergency medical services, indicating a more severe trauma. This may partially explain why chronic alcohol abuse was associated with increased risk for traumatic lesions but alcohol intoxication was not.

The incidence of acute traumatic lesions is much higher in patients with moderate to severe TBI, but it is possible to suffer a severe TBI without intracranial trauma visible on CT. CT underestimates many intracranial nonhemorrhagic lesions, especially DAI.⁴¹ The decades-old approach of classifying TBI severity based on the GCS could be improved by considering neuroimaging findings. The GCS is often supplemented by the clinical assessment of PTA and LOC, but both are prone to confounding factors. An injury severity classification system that incorporates neuroimaging findings could improve outcome prediction, resource allocation, and patient stratification in clinical trials.³⁴

The present study has important limitations. First, the clinical variables were collected retrospectively from hospital records and hence some relevant information was missing. This may have introduced bias into our estimation of the relationship between patient and injury characteristics and CT findings. Second, the decision to order a head CT scan was left to the on-call ED physician's clinical judgment. This usual care methodology probably resulted in some patients undergoing CT scanning that would not have been needed according to the Scandinavian guidelines.²² Also, we might have missed some patients who should have undergone a CT scan but who were discharged without imaging (although the results suggest that a fairly liberal use of CT scanning was present over the study period).

Third, although the Scandinavian guidelines were used in the ED, these were probably not followed consistently. We do not have data in patients who did not undergo head CT scanning. The percentages of patients with MTBI with specific pathologies are an approximation because they do not include the subgroup of patients who were not triaged for CT. Some people with the mildest form of an MTBI probably did not undergo scanning; thus the rate of abnormalities in the MTBI group is likely higher than in the total population of those presenting to the ED (and, of course, that rate does not apply to those who do not present to the ED). It is probable that the number of CT-positive injuries would be smaller among patients with mild head trauma for whom a head CT scan was not performed.

Fourth, in our sample, 1 in 4 (776 patients [25.7%]) did not have clearly documented clinical signs or symptoms of TBI in their medical records, which might reflect the absence of those signs, incomplete documentation, or both. Fifth, our sample contained a small number of patients who were classified as having sustained an MTBI, even though they had a GCS score < 13. In this group, low GCS score probably resulted from intoxication, medications, or other medical conditions. There is a possibility that the injury of some of these cases was incorrectly classified. Finally, all CT scans were analyzed and systematically coded by 2 neuroradiologists (A.B. and A.K.) using a structured and detailed case report form designed for this study, but we

did not conduct a study related to the intrarater or interrater reliability of this case report form.

The reliability of CT findings may be limited by the subjective nature of the visual interpretations, by the qualitative nature of reporting,²⁰ as well as by the experience and training of the reader (e.g., ED physician, radiologist, or neuroradiologist).^{14,30} In our study, the scans were coded by 2 experienced neuroradiologists. In prior studies, interobserver reproducibility was good even among multiple readers with varying backgrounds when interpreting CT imaging features of TBI.^{6,14} Some studies have reported poorer interobserver agreement, but it seems that major findings are seldom missed.³⁰ Overall, the discrepancy rate is low (0.8%; 95% CI 0.4%–1.6%) in head CT scans.⁶⁴ Future research could examine both intrarater and interrater reliability of this coding system.

Approximately 2 of 3 patients underwent CT scanning within the first 24 hours after injury. On the other hand, 1 of 4 patients underwent scanning > 48 hours after injury. The temporal trends on how well certain traumatic lesions are visible on brain CT scans should be noted. Acute imaging (performed in the first hours after injury) can result in false negative scans; for example, some contusions do not demarcate as early as others. Furthermore, with delayed scanning (done days after injury), some minor subarachnoid hemorrhages can be missed because these lesions have already been reabsorbed. These CT-related limitations should be kept in mind when interpreting our findings.

The anatomical characteristics of a child's brain might make it more susceptible than the adult brain to certain types of injuries following head trauma. The head is larger in proportion to the body surface area, and stability is dependent on the ligamentous rather than bony structure. Subdural and subarachnoid hemorrhages were the most common intracranial lesions in the current study. The assessment of pediatric patients with head trauma is different than that of adults. Even in the pediatric population, the mainstay of imaging is CT, but the significant radiation exposure must be considered. It is possible that some of the pediatric patients with TBI at our hospital had MRI as their first imaging study.

In this study, there was a large amount of missing data in the medical records on PTA, LOC, and GCS scores, which are part of the foundation of TBI diagnosis. These findings highlight the importance of a thorough medical history, focusing on the injury mechanism and confounding factors as well as a careful neurological examination that includes GCS score, mental state, and PTA measurements. The presence of PTA was noted much more often in patients with MTBI than in patients with more severe TBI. Often patients with moderate and severe TBIs have additional injuries that dominate clinical attention or they are unconscious at arrival and only progress to PTA after being transferred from the ED. There also might be a tendency for some physicians to document more specific injury criteria following MTBI, especially in patients with isolated head trauma, and to document fewer criteria in ED situations when the person has an obvious moderate or severe TBI. Finally, the study examined only patients with MTBI who presented to an ED. Rates of CT abnormalities in patients who do not seek acute care at an ED are not known.

Conclusions

Approximately 1 in 6 patients who present to the ED with suspected or confirmed MTBI will have a CDE trauma-related intracranial abnormality on day-of-injury CT scan. The frequency of CT findings is much lower than in patients with moderate to severe TBI, but the type and location of lesions is very similar. Clinicians and researchers should also be aware that within the spectrum of MTBI, patient demographic variables (older age and male sex), preinjury exposures (alcohol abuse, but not preexisting brain lesions), mechanism of injury (falls and being struck by a motor vehicle), and reduced consciousness following injury (GCS score < 15) are all associated with an increased incidence of intracranial abnormality. Further research is needed to translate this knowledge into refined MTBI clinical stratifications and prognostics.

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Disclosures

Professor Iverson is a consultant for NeuroHealth Research and Rehabilitation, Inc. and NeuroHealth, LLC.

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Conception and design: Isokuortti, Iverson, Silverberg, Luoto. Acquisition of data: Isokuortti, Brander, Luoto. Analysis and interpretation of data: Isokuortti, Silverberg, Kataja, Brander, Luoto. Drafting the article: all authors. Critically revising the article: all authors. Reviewed submitted version of manuscript: Isokuortti, Iverson, Silverberg, Kataja, Öhman, Luoto. Approved the final version of the manuscript on behalf of all authors: Isokuortti. Statistical analysis: Isokuortti, Silverberg. Administrative/technical/material support: Isokuortti, Kataja, Brander. Study supervision: Luoto.

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