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Post-traumatic Headache After Pediatric Traumatic Brain Injury: Prevalence, Risk Factors, and Association With Neurocognitive Outcomes

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Abstract

Post-traumatic headache is common after pediatric traumatic brain injury and affects thousands of children every year, but little is known about how headache affects recovery after traumatic brain injury in other symptom domains. We aimed to determine the association between headache and other common symptoms after pediatric traumatic brain injury and explore whether subjective complaints of headache are associated with objective deficits on specialized neurocognitive testing. We conducted a retrospective cohort study of children ages 3–19 years following traumatic brain injury with a completed Sports Concussion Assessment Tool (SCAT) questionnaire. Post-traumatic headache was defined by a score more than 2 on the SCAT question for headache and define headache groups for comparison. In our cohort, we analyzed data from the Delis-Kaplan Executive Function System and the Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-II). Headache was reported in 40 (33%) patients presenting for post-traumatic brain injury care among 121 pediatric traumatic brain injury patients and did not differ by injury severity. Median total SCAT symptom score in the headache group was 5-fold higher compared to patients without headache (median 45.5 vs 9; $P < .001$). Significantly lower-scaled scores in color naming, matrix reasoning, letter sequencing, and letter switching were also found in the headache group (all $P < .03$). Our study shows that headache, as reported by patients on the SCAT, is associated with higher symptom scores in all other symptom domains, including sleep, mood, sensory, and

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Supplemental Material

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Declaration of Conflicting Interests

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cognitive. Headache was also associated with worse objective neurocognitive measures and may identify patients who could benefit from specialized follow-up care and management.

Keywords

cognition; concussion; headache; pediatric; outcome; traumatic brain injury; risk factor

Traumatic brain injury, defined as the alteration of brain function or brain pathology following external force, is a leading cause of long-term morbidity in children. More than 500 000 pediatric patients present to the emergency department for traumatic brain injury and 50 000 require hospitalization in the United States each year.^{1,2} From 2001 to 2012, the rate of emergency department visits for a diagnosis of traumatic brain injury or concussion more than doubled among children aged 19 years or younger.³ Traumatic brain injury leads to a range of long-term morbidities including physical, psychosocial, and neurocognitive deficits.⁴

Post-traumatic headache is one of the most common complaints following pediatric traumatic brain injury across all injury severities and may persist long term.⁵ Chronic headache in otherwise healthy children is an important morbidity to consider when providing care to patients because it interferes with school, social function, and parental productivity.⁶ It may be more relevant for consideration in children with traumatic brain injury who concurrently suffer functional and neurocognitive impairments related to direct brain injury. While some neurocognitive impairment after traumatic brain injury is related to location and severity of brain injury, even patients with mild traumatic brain injury and no identifiable brain injury on imaging suffer impairments in memory, attention, processing speed, language, and executive function that can persist for years and few effective interventions exist.^{7,8} Data are limited in pediatric post-traumatic headache as it relates to other morbidities including neurocognitive dysfunction, and whether headache treatments can modify other important morbidities following pediatric traumatic brain injury is unknown. Limited research has left clinicians without guidance on the identification and management of post-traumatic headache, which may have a key role in improving other outcomes.^{9,10}

Our institution provides systematic care for children with traumatic brain injury including symptom evaluation and management through multidisciplinary concussion and pediatric neurocritical care programs.^{11,12} Both programs provide pediatric traumatic brain injury expertise that includes systematic symptom and comorbidity evaluations with medical and neuropsychological assessments. In this study, we evaluated post-traumatic headache in children presenting for post-traumatic brain injury evaluation and management. We aimed to determine the prevalence of headache, association between headache and other post-traumatic symptoms, and explore the relationship between headache and neurocognitive outcome. We hypothesized that post-traumatic headache after pediatric traumatic brain injury would be a prevalent morbidity that was associated with neurocognitive impairment regardless of traumatic brain injury severity.

Methods

Participants and Procedures

We conducted a retrospective cohort study of pediatric patients following traumatic brain injury to evaluate post-traumatic headache. The Oregon Health and Science University Institutional Review Board approved the study with a waiver of informed consent (IRB 17497). Consecutive children aged 3–19 years evaluated in the Oregon Health and Science University concussion program or in the pediatric neurocritical care program from March 2015 to January 2018 following a traumatic brain injury were included. Data were collected on the first outpatient visit to the clinics a median of 7 months after injury (interquartile range 3, 17 months).

Demographic information, injury characteristics, past medical histories, and radiology data were extracted from chart review. All severities of traumatic brain injury were included and defined by the Glasgow Coma Scale recorded at the time of injury (mild 13–15, mild complicated 13–15 with identified radiologic abnormality, moderate 9–12, and severe 3–8). Patients with mild traumatic brain injury and no radiologic abnormalities are termed concussion patients. Patients not evaluated at the time of initial injury were recorded as Glasgow Coma Scale score = 15, unless documented otherwise in the medical record. Our institution uses the PECARN guidelines¹³ when evaluating patients to determine need for head imaging after trauma. Intracranial injury (eg, hematoma or hemorrhage, contusion, axonal injury, edema) and skull fracture were recorded from radiologic data at the time of injury when available to distinguish mild and mild complicated injuries; all patients with moderate or severe traumatic brain injury had identified radiologic abnormalities. Prior head injury was documented from notes. Subconcussive head injury was defined as involvement in sports with potential for contact and in our cohort was defined as involvement in football or soccer.

Measures

Post-traumatic headache was defined in this study as a self-reported score more than 2 on the Sports Concussion Assessment Tool (SCAT)^{14–16} for headache and was used to define headache groups for comparison. The SCAT is used routinely in both clinics to screen for traumatic brain injury–related symptoms. The SCAT symptom evaluation utilizes a self-report format to measure symptom severity scored from 0 to 6 on each item. Scores of 1 to 2 indicate mild, 3 to 4 moderate, and 5 to 6 severe symptoms. Patients were excluded from analysis if the SCAT form was missing or incomplete (n = 27/54 pediatric neurocritical care program patients and n = 76/170 concussion program patients).

Neuropsychological assessment is part of routine care in both Oregon Health and Science University programs and is conducted by the same neuropsychology team. We collected standardized scores for all neurocognitive assessments performed and compared between headache groups. Neurocognitive testing in the clinics utilizes a flexible battery approach, with age- and function-appropriate measures, so not all patients receive the same tests. The majority of patients are evaluated with the Delis-Kaplan Executive Function System (D-KEFS) and the Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-II); thus,

we focused our evaluation of neurocognitive outcomes on the subset of patients undergoing these tests ($n = 98$).^{17–19} The Delis-Kaplan Executive Function System contains 9 separate tests (with multiple process levels), which measure a variety of different aspects of executive function in children aged 8 years. In our cohort, we analyzed data from the Delis-Kaplan Executive Function System verbal fluency test (ie, letter fluency and category fluency), colorword interference test (ie, color naming, word reading, and inhibition speed), and the trail making test (number sequencing, letter sequencing, and letter-number switching). From the WASI-II, we report the vocabulary (assessing verbal comprehension) and matrix reasoning (assessing nonverbal reasoning abilities) subtests in children 6 years, as well as the 2-subtest derived (hence abbreviated) Full Scale Intelligence Quotient. Testing was most often performed in clinic visits 6 months after injury, particularly for those with moderate or severe head injuries. Most patients without neurocognitive testing reported in the initial clinic visit ($n = 23$) were <6 months from injury (87%). Given constraints of Delis-Kaplan Executive Function System and WASI-II tests, patients undergoing neurocognitive testing were also older (median 14.8 vs 7.6 years), though testing was performed and reported for 1 patient <6 years of age.

Statistical Analysis

Categorical variables were reported as counts with percentages and continuous variables were reported as medians with interquartile range. Chi-square tests with Fisher exact correction as indicated were used for comparison of categorical variables between headache groups. Mann-Whitney U tests were performed for comparison of continuous variables between headache groups. Statistical analyses were performed using SPSS version 24. Significance was defined as $P < .05$, and all tests were 2-tailed.

Results

Traumatic Brain Injury Cohort

Following traumatic brain injury, 121 children were evaluated. Median time to outpatient assessment was 7 months after primary injury. Median age overall was 14.3 years, and 51% were male. Most patients ($n = 106$, 88%) had mild traumatic brain injury as defined by their Glasgow Coma Scale scores. Documented intracranial injury was found in 28 (23%) patients requiring hospitalization at the time of injury. Thirteen (46%) patients with intracranial injury had mild complicated traumatic brain injury. The most common mechanisms of injury included sports-related injury (44%), motor vehicle accidents (18%), and falls (14%). Comorbidities were found in 69 (57%) children in the overall cohort including psychiatric ($n = 48$, 40%), attention-deficit disorders ($n = 21$, 17%), and asthma ($n = 11$, 9%). Prior head injury ($n = 49$, 40%) and subconcussive injury risk ($n = 24$, 20%) were common.

Post-traumatic Headache

Headache was reported in 40 (33%) patients presenting for post-traumatic brain injury care. Table 1 presents the demographic characteristics of the traumatic brain injury cohort by headache group. No differences were found in the frequency of headache in the 2 clinics ($P = .37$). More females and those with Hispanic ethnicity or Medicaid insurance reported headaches, though differences did not reach statistical significance. Prior head injury and

subconcussive head injury risk did not increase risk for post-traumatic headache. The frequency of chronic conditions overall was similar between headache groups ($P = .39$), though pre-existing psychiatric comorbidities were more common in the headache group (50%) versus patients without headache (35%; $P = .1$).

Table 2 presents injury characteristics by headache group. No difference was found in Glasgow Coma Scale scores between groups, and headache was found in 33 patients with mild traumatic brain injury (35%), 2 with mild complicated traumatic brain injury (15%), 2 with moderate traumatic brain injury (29%), and 3 with severe traumatic brain injury patients (38%). Other injuries, such as extremity fractures, were more frequent in the headache group (23% vs 11%, $P = .1$). Headache risk was not increased among patients with documented intracranial injury by imaging, but notably some patient characteristics were different between those with intracranial injury versus concussion. Patients with intracranial injury ($n = 28$ mild complicated, moderate, severe traumatic brain injury) were more likely to present to care in the first 6 months after injury compared to concussion (81% vs 32%, $P < .01$). Patients with intracranial injury were less likely to have a comorbidity (15% vs 69%, $P < .01$) or a sports-related injury (11% vs 53%, $P < .01$).

Headache and Other Post-traumatic Morbidities

Headache was associated with significantly more subjective complaints of other symptoms after traumatic brain injury, including sleep, mood, sensory, and cognitive disturbances. Table 3 presents total SCAT score and SCAT symptom scores by headache group. Median total SCAT score in the headache group was 5-fold higher compared to patients without headache (median 45.5 [interquartile range 28, 68.5] vs 9 [interquartile range 3, 17]). One-quarter of patients in the headache group reported moderate or severe trouble falling asleep and half reported moderate to severe fatigue or low energy levels, whereas these symptoms were found in <10% of patients without headache. More than 25% of children in the headache group and <5% without headache reported at least moderate or severe sensitivity to light and noise. In regard to mood, 25% of the headache group reported moderate or severe disturbances, including feeling sad or anxious in contrast to <10% in those without headache. Further, moderate to severe difficulties with concentration and remembering were reported in more than 50% of patients with headache and in <10% of those without headache.

Objective neurocognitive testing broadly examining the cognitive domains of language, processing speed, intelligence, learned knowledge/memory, and executive function was performed on a subset of patients ($n = 98$; see Supplemental Table 1 for subgroup characteristics by headache group). Patients with headache had lower standardized scores in all neurocognitive tests, and statistically significant differences were found in some subdomains of the Delis-Kaplan Executive Function System and WASI-II batteries (Table 4). Median FSIQ score in the headache group (105; interquartile range 95, 117) was significantly below that of the patients without headache (117; interquartile range 104, 125; $P < .01$), as well as a similar pattern of significant group differences on the verbal (ie, vocabulary) and nonverbal (ie, matrix-reasoning) subtests that comprise the overall intelligence estimate (see Table 4). Significantly lower scaled scores regarding the Delis-

Kaplan Executive Function System conditions of color naming, letter sequencing, and number-letter switching were also found in the headache group (all $P < .04$) reflecting significant differences in processing speed and executive function between headache groups.

Discussion

We found post-traumatic headache to be a prevalent morbidity in children who present for post-traumatic brain injury care, regardless of severity and characteristics of injury. In our study, post-traumatic headaches were found in one-third of patients at a median of 7 months after injury. Youth with post-traumatic headache also reported significantly more dysfunction with sleep, mood, sensory, and cognitive disturbances. Self-reported headache was associated with objectively worse neurocognitive function, particularly for tasks that tax processing speed abilities (see Table 4). Post-traumatic headache represents an important morbidity in pediatric traumatic brain injury patients that may compound other morbidities, offer a therapeutic target for other post-traumatic symptoms, and identify patients in need of formal neurocognitive testing.

Previous studies in adults and pediatrics have shown prevalence of post-traumatic headache to be anywhere from 9% to 44% at 3 months, 20% to 44% at 6 months, and 20% to 23% at 12 months, but have all used different methods for identifying headaches and ranges of time from injury.^{20–23} The largest study evaluating pediatric post-traumatic headache to date used a similar dichotomization of a single question to define headache and found a similar prevalence of headache, 42% at 3 months postinjury, and persistence of headache 12 months after injury.⁵ Our study adds to this literature showing a high prevalence and persistence of post-traumatic headache in the pediatric traumatic brain injury population. One-quarter of our patients with headache complained of moderate to severe sensitivity to light and noise, consistent with migraine headache features. Moderate to severe neck pain and pressure were also present in almost 25% of headache patients, consistent with tension headaches. Headache phenotypes are poorly defined in pediatric traumatic brain injury literature, though prior studies suggest migraine and tension are most common and consistent with our findings.²¹

Prior studies have identified several risk factors for post-traumatic headache, though findings are often inconsistent between studies.²¹ Females were more likely to endorse headache symptoms in prior studies,^{5,20,24,25} and older age has been associated with an elevated risk for post-traumatic headache.^{5,26} Our study did not find any statistically significant differences in risk for headache among the demographic and injury characteristics we evaluated. A recent systematic review of recovery after concussion noted inconsistencies when evaluating risk factors, and these differences may be due to variability in patient populations, definitions of headache, and variable time to evaluation after traumatic brain injury.²⁷

Pre-existing conditions were found in more than half of the patients in our study, though no difference was found between headache groups. The most common comorbidities in our cohort included psychiatric, attention-deficit hyperactivity disorders (ADHD), and asthma. Previous studies in pediatric traumatic brain injury evaluating outcomes like neurocognitive

function, sleep disturbances, and psychological disturbances have shown premorbid status to be a key risk factor determining outcome. A prior study in children evaluating post-traumatic headache found 51% had pre-existing headaches, 31% had migraine/probably migraine preinjury, and 56% had a family history of migraine.²⁸ Preinjury parental factors were also associated with greater rates of post-traumatic headache in prior studies.^{26,29} We were unable to systematically assess pre-existing headache or family history with our study, and this may account for differences from prior studies.²⁷ Further research is needed to determine if pre-existing comorbidities are driving the high prevalence of headaches found after pediatric traumatic brain injury to inform future intervention studies.

We did not find a difference in post-traumatic headache based on severity of primary traumatic brain injury measured by Glasgow Coma Scale, though patients with concussion versus intracranial injury had important differences, such as time to evaluation, frequency of pre-existing conditions, and mechanism of injury that may confound the association between headache and injury severity. Regardless of headache status, patients were more likely to present to care early after traumatic brain injury with the presence of radiographic intracranial injury versus concussion. Patients with concussion may present later to specialized care after referral from primary providers or related to persistence of symptoms, whereas patients with intracranial injury may present earlier because of presence of symptoms or direct referrals after hospitalization. Early intervention regardless of injury severity may prevent the evolution of secondary or tertiary injury factors that contribute to headache status. Although comorbidities were found in more than half of all patients, comorbidities were far less likely in patients with identifiable intracranial injury versus concussion. Mechanisms of injury also varied by the presence of intracranial injury, with sports-related injuries being more common in patients with concussion. Most studies to date have included only concussion patients or have stratified patients only by Glasgow Coma Scale scores.⁷ Although the presence of intracranial injury compared to concussion alone was not associated with increased risk of post-traumatic headache in our study, the differences in demographic characteristics between these 2 groups is an important consideration in future studies evaluating risk factors for post-traumatic headache.

Post-traumatic headache was associated with higher symptom scores in all other domains of the SCAT. It is unclear if headache drives development of other symptoms or is reflective of a common pathophysiology driving the development of all post-traumatic morbidities. Prior studies similarly show that these symptoms are highly inter-related and that headache may be a risk factor for persistence of postconcussive symptoms.²⁷ One study in adult traumatic brain injury showed there was a relationship between headache, sleep problems, psychiatric symptoms, and dizziness on SCAT measures.³⁰ Another study in adult patients with traumatic brain injury similarly showed that patients with post-traumatic headache had higher total symptom severity scores than the nonheadache group.³¹ Sleep, mood, and sensory disturbances after traumatic brain injury are all known morbidities, but it is unclear how headache impacts these symptoms in children. Headache may cause difficulty falling asleep, but sleep disturbances have also been associated with headache.^{32–34} Similarly, headache may cause anxiety or depressed mood by impairing quality of life or social function, but headache is also a known morbidity among patients with psychological disorders.^{35,36} Alternatively, traumatic brain injury, both through direct brain injury or

secondary injury processes like inflammation, may be driving the development and persistence of all of the symptoms evaluated by SCAT, including headache. More research is needed to evaluate the relationship between headache and other post-traumatic symptoms to determine if treating headaches can improve other symptoms and lower the risk for persistence of postconcussive symptoms, or if targeting symptoms like sleep disturbances can improve headaches after traumatic brain injury.

Headache may be an important predictor of neurocognitive deficits after traumatic brain injury. Prior research on neurocognitive outcomes in pediatric traumatic brain injury have shown that young age at injury, lower premorbid developmental and academic achievement, and severity of injury increase risk of cognitive deficits, but have not evaluated headaches as a risk factor.^{37–41} We demonstrated that patients with headache had lowered neurocognitive performance among all of the commonly employed neuropsychological tests in our clinic, particularly in measures that tax processing speed abilities, but also subtests that have the highest factor loadings for overall intelligence estimates (ie, Full Scale Intelligence Quotient). The former finding is consistent with prior work,⁴² yet meta-analytic findings regarding intelligence after traumatic brain injury have demonstrated that Full Scale Intelligence Quotient impairments were absent for mild traumatic brain injury (compared to moderate/severe traumatic brain injury) in the subacute recovery phase, and only small intelligence impairments have been noted in the chronic recovery phase for mild traumatic brain injury. Further, adults with traumatic brain injury in the chronic recovery phase had larger intelligence differences than children who had sustained traumatic brain injury.⁴³ Considering these meta-analytic findings, current significant differences in Full Scale Intelligence Quotient found between the headache and nonheadache group may reflect preinjury patient characteristics, including coping skills, which may mediate recovery from traumatic brain injury and reflect overall better health.^{27,44,45} In our study, the number of patients with a history of special education or individualized education programs was not statistically different between headache groups, but a comprehensive evaluation of premorbid status was not possible. Further, the possibility of preinjury population differences in cognitive performance underscores the importance of early evaluation to avoid the “good old days bias,”⁴⁶ and early intervention to improve the trajectory of recovery. It is also possible that differences in cognitive performance between headache groups represent differences in severity of primary injury not captured by measures like Glasgow Coma Scale, ongoing secondary processes like inflammation, or are due to other post-traumatic symptom–related impairments in test performances.

Pain, including headache, may be particularly important in evaluations of neurocognitive outcomes in children with brain injury. Chronic pain and headaches outside of traumatic brain injury have been shown to reduce school performance and attendance, increase psychosocial comorbidities, and impair cognition.^{47–49} Additionally, avoidance of mental exertion following traumatic brain injury as it relates to headache has been shown to lower cognitive testing scores.⁵⁰ Measures taxing processing speed abilities were particularly impaired with headache in our study and may reflect the effects of active pain during testing or avoidance of mental exertion as a perceived trigger. However, the global differences in cognitive performance between headache groups we found (considering trends toward significance in addition to significant group differences) suggests that headache may identify

a group of patients at risk for post-traumatic cognitive impairment. Access to Pediatric Neuropsychology expertise is limited and often not provided as part of routine post-traumatic brain injury care, making screening tools valuable for clinicians to ensure high-risk patients access these services.⁵¹ This study provides preliminary evidence that the presence of headache, defined by SCAT score, may indicate which patients need specialized neurocognitive testing. Given the impact of neurocognitive deficits on learning and development in children, the relationship between post-traumatic headache and neurocognitive outcomes should be investigated because treatment for headaches could lead to improved cognitive performance after traumatic brain injury.

This study has limitations to consider including the retrospective design, use of SCAT to define headache, and use of a clinical sample from clinics providing post-traumatic brain injury care. The International Classification of Headache Disorders III-beta defines post-traumatic headache as onset of headache within 7 days of injury, but given the retrospective design of our study, timing of onset cannot be ensured.⁵² Additionally, SCAT data were missing or incomplete in the medical record for some patients evaluated after traumatic brain injury, which could contribute to selection bias. Further, although the original version of SCAT was not validated in pediatric patients, the child SCAT3 and SCAT3 have been validated in children aged 5–16 years and in non-sports injury concussions.⁵³ The portion of the SCAT used in this study was limited to the symptom evaluation portion of the SCAT, which is very similar to that of the SCAT3. We chose to use SCAT scores given its widespread use, free availability to clinicians, and ease of administration.⁵⁴ There are few headache-specific questionnaires available that have been validated in children or employed in pediatric traumatic brain injury populations. Our clinical sample may select patients with more post-traumatic symptoms overall as patients with milder or no symptoms may not complete a clinic visit. Although our clinics routinely assess some important comorbidities, preinjury headaches or family history of headache is not systematically assessed, which may be important when assessing risk of headache after traumatic brain injury in future work. Because of the retrospective nature of our study, preinjury cognitive testing scores were not obtained for comparison. Additionally, our study had a wide range of time to assessment, which likely affects our estimates of prevalence and risk factors. Our sample size was small, limiting our ability to detect potential differences between headache groups in some variables or to conduct rigorous multivariable adjustment when assessing risk for post-traumatic headache and the association between headache and neurocognitive outcomes. Nonetheless, our results show post-traumatic headache is an important morbidity that may be a useful predictor of important neurocognitive deficits requiring further research.

Conclusion

Post-traumatic headache is an important and persistent morbidity in pediatric traumatic brain injury patients, regardless of primary injury severity. Our study shows that headache, as reported by patients on the SCAT, is associated with higher symptom scores in all other post-traumatic symptom domains including sleep, mood, sensory, and cognitive. Headache was also associated with worse objective neurocognitive measures and may identify patients who could benefit from comprehensive cognitive evaluations. More studies are needed to define post-traumatic headache phenotype, determine effective headache interventions, and

determine the impact of headache treatments on recovery from the multidimensional sequelae of pediatric traumatic brain injury.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1.Demographic and Baseline Characteristics by Headache Group.^a

	Headache negative (n = 81)	Headache positive (n = 40)	P value
Median age in years (IQR)	14.3 (11.1, 15.9)	14.8 (11.3, 16)	.74
Male	44 (54)	18 (45)	.34
White race	69 (85)	30 (75)	.15
Hispanic ethnicity	9 (11)	9 (23)	.07
Medicaid insurance	23 (28)	17 (43)	.12
Prior head injury	30 (38)	19 (48)	.29
Subconcussive injury risk	14 (17)	10 (25)	.32
Pre-existing comorbidity	44 (54)	25 (63)	.39
Psychiatric	28 (35)	20 (50)	
Attention deficit	17 (21)	4 (10)	
Asthma	6 (7)	5 (13)	
History of special education or IEP	10 (12)	4 (10)	.70

Abbreviations: IEP, Individualized Education Program; IQR, interquartile range.

^aUnless otherwise noted, values are n (%).

Table 2.**Injury Characteristics by Headache Group.^a**

	Headache negative (n = 81)	Headache positive (n=40)	P-value
Median GCS (IQR)	15 (15, 15)	15 (15, 15)	.92
GCS group			.94
Mild	60 (74)	33 (82)	
Mild complicated	11 (14)	2 (5)	
Moderate	5 (6)	2 (5)	
Severe	5 (6)	3 (8)	
Intracranial injury	21 (26)	7 (18)	.37
Skull fracture	12 (15)	5 (13)	.73
Mechanism of injury			.9
Sports injury	36 (44)	17 (43)	
Motor vehicle accident	15 (19)	7 (18)	
Fall	10 (12)	7 (18)	
Other ^b	20 (25)	9 (23)	
Loss of consciousness	18 (22)	9 (23)	.90
Other injuries ^c	9 (11)	9 (23)	.1

Abbreviations: GCS, Glasgow Coma Scale; IQR, interquartile range.

^aUnless otherwise noted, values are n (%).

^bOther includes: Auto-pedestrian/auto-bicycle, all-terrain vehicle accidents, bike/skateboard/scooter.

^cOther injuries included bone fractures, pulmonary contusions, liver and splenic lacerations.

Table 3.

SCAT Scores by Headache Group.

	Headache negative, median (IQR) (n = 81)	Headache positive, median (IQR) (n = 40)	P value
SCAT Total Score	9 (3, 17)	45.5 (28, 68.5)	<.001
Symptom			
Pressure in head	0 (0, 1)	2 (0, 3)	<.001
Neck pain	0 (0, 1)	1 (0, 2.75)	.002
Balance problems or dizzy	0 (0, 0)	1 (0, 2)	<.001
Nausea/vomiting	0 (0, 0)	0 (0, 1)	<.001
Vision problems	0 (0, 0)	1 (0, 3)	<.001
Hearing problems	0 (0, 0)	0 (0, 2.75)	.009
Do not feel right	0 (0, 0)	2.5 (1, 4)	<.001
Feeling dinged	0 (0, 0)	1.5 (0, 3)	<.001
Confusion	0 (0, 0)	1 (0, 3)	<.001
Feeling slowed down	0 (0, 1)	1.5 (0, 3.75)	<.001
Feeling in a fog	0 (0, 0)	2 (0, 3)	<.001
Drowsiness	0 (0, 1)	2 (0, 4)	<.001
Fatigue or low energy	0 (0, 2)	3 (1, 4.75)	<.001
More emotional	0 (0, 1)	2 (0, 4)	<.001
Irritable	0 (0, 2)	3 (0, 4)	<.001
Difficulty concentrating	0 (0, 1)	3 (1, 4)	<.001
Difficult remembering	0 (0, 2)	3 (1, 4.75)	<.001
Sadness	0 (0, 0)	1 (0, 3)	<.001
Nervous or anxious	0 (0, 1)	2 (0, 3.75)	<.001
Trouble falling asleep	0 (0, 1)	2 (0, 4)	.001
Sleeping more than usual	0 (0, 1)	0 (0, 3)	.015
Sensitivity to light	0 (0, 0)	2 (0, 3)	<.001
Sensitivity to noise	0 (0, 0)	2 (0, 4)	<.001

Abbreviations: IQR, interquartile range; SCAT, Sports Concussion Assessment Tool.

Table 4.

Neurocognitive Scaled Scores by Headache Group.

Neurocognitive test	Headache negative		Headache positive		P value
	Median score (IQR)	n (%)	Median score (IQR)	n (%)	
Color Naming ^b (n = 89)	10.0 (8.0, 11.8)	57 (64%)	8.0 (6.0, 10.0)	32 (36%)	.03
Inhibition Speed ^{b, d} (n = 89)	10.0 (7.3, 11.0)	57 (64%)	10.0 (7.0, 11.0)	32 (36%)	.22
Number Sequencing ^b (n = 95)	10.5 (9.0, 13.0)	62 (65%)	10.0 (8.0, 12.0)	33 (35%)	.14
Letter Sequencing ^b (n = 95)	12.0 (10.0, 13.0)	62 (65%)	10.0 (8.0, 12.0)	33 (35%)	<.01
Number-Letter Switching ^{b, d} (n = 93)	11.0 (9.0, 12.0)	61 (66%)	9.0 (7.0, 11.0)	32 (34%)	.04
Word Reading ^e (n = 89)	11.0 (9.0, 12.0)	57 (64%)	9.0 (7.0, 11.0)	32 (36%)	.1
Full Scale IQ ^c (n = 81)	117.0 (104.3, 124.5)	52 (64%)	105.0 (95.0, 116.50)	29 (36%)	<.01
Matrix Reasoning ^{c, d} T-Score (n = 86)	58.0 (53.3, 63.0)	57 (66%)	53.0 (47.50, 58.50)	29 (33%)	.03
Vocabulary ^c T-Score (n = 84)	59.5 (51.5, 67.5)	56 (67%)	54.5 (47.5, 61)	28 (33%)	.02

Abbreviations: IQ, intelligence quotient; IQR, interquartile range.

Testing Domains: (a) = language, b = processing speed, c = intelligence, d = executive function, e = learned knowledge/memory).