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Family History of Migraine is Associated with Posttraumatic Migraine Symptoms following Sport-related Concussion

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Abstract

Objective—To determine if family history of migraine increased the likelihood of posttraumatic migraine symptom (PTM) presentation in adolescents following concussion, and examine the influence of family history of migraine and PTM on post injury outcomes.

Setting—Outpatient concussion clinic

Participants—153 concussion patients (103 males, 50 females) aged 15.72 +/− 1.48 (range 12–18)

Design—Cross-sectional, observational study of patients presenting for initial evaluation 4.72+/−3.05 days (range 1–14) post injury

Main Measures—Computerized neurocognitive testing, symptom report, and vestibular/oculomotor screening

Results—Patients with family history of migraine were 2.6 times (OR=2.60, [CI=1.35–5.02], p=.003) more likely to present with PTM compared to patients without family history. Results of MANCOVAs, controlling for concussion history, revealed significant main effects for PTM on 1) ImPACT/PCSS (F=15.43, p<.001), and 2) VOMS (F=8.52, p<.001). There was no main effect for family history of migraine on ImPACT/PCSS (p=.22) and VOMS (p=.83) or interaction between family history of migraine and PTM on ImPACT/PCSS (p=.84) and VOMS (p=.52).

Conclusion—Family history of migraine is associated with PTM symptoms following sport-related concussion, suggesting a genetic predisposition for migraine may serve as a catalyst or trigger for onset of PTM. However, only presence of PTM, rather than family history of migraine, was related to worse neurocognitive and vestibular/oculomotor outcomes.

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Approximately 1.6 to 3.8 million sport-related concussions (SRC) occur each year in the United States[1]. Symptoms (physical, cognitive, emotional, sleep-related) and impairments (balance, cognitive, vestibular, oculomotor) following SRC are heterogeneous[2]. Recently, researchers have proposed clinical profiles to reflect the heterogeneity of SRC[3,4]. One clinical profile that has previously been documented following SRC is post-traumatic migraine (PTM)[5–7]. According to International Headache Society (IHS) guidelines, a migraine is defined as a headache with nausea and photo and/or phonophobia[8]. Concussions and migraines are believed to share similar pathophysiology[9] resulting in a spreading cortical depression that leads to migraine symptoms[8]. Although headache occurs in up to 93% of athletes following concussion[10], PTM is reported in only 15–33%, but is associated with more pronounced impairment and prolonged recovery[5,11]. Concussed patients with PTM symptoms have exhibited more cognitive deficits than patients with non-migraine headaches or no headache[6,11], and have demonstrated reduced brain network activation during a cognitive task compared to controls and concussed athletes without PTM[12]. Kontos et al.[11] reported that concussed athletes with PTM were 7 times more likely to experience a protracted recovery compared to those not experiencing headache or PTM, and more than two times more likely to experience protracted recoveries than those athletes experiencing headache only. Post-traumatic migraine may also contribute to increased balance deficits following SRC[13]. Vestibular and oculomotor impairments are common following SRC and have also been reported in non-concussed individuals with migraine[14,15]. However, the relationships between vestibular/oculomotor impairments and PTM following SRC have yet to be explored.

Researchers have proposed that certain clinical profiles such as PTM may be linked to specific pre-injury risk factors[4]. The concussion in sport group (CISG) has suggested migraine is a “concussion modifier” as a co and pre-morbid factor, serving as a primary (i.e., risk of sustaining a concussion) and secondary risk factor (i.e., risk of poor outcomes following concussion)[16]. While studies have linked personal and familial history of migraine to persistent posttraumatic headache[17] and development of “post-concussion syndrome”[18], another study found neither personal or family history of migraine to be predictive of symptom duration in a cohort of emergency department (ED) patients[19]. Further, only one study has examined history of migraine and post injury neurocognitive testing among those with and without personal history of migraine, and did not find a relationship[20]. Migraine is a common condition with a lifetime prevalence of 18% for men and 40% for women[21,22], with the peak onset is late adolescence and early adulthood[22]. In contrast, a recent review suggests prevalence in children and adolescents is only 7–11% due to later onset of this condition[23]. Migraine has a strong genetic basis[24] and previous studies suggest that first degree relatives of migraineurs are 2–19 times more likely to be diagnosed with migraine[25,26]. However, it is unclear if SRC acts as a catalyst for onset of PTM in adolescent athletes who may be genetically vulnerable based on family history. Further research examining the relationship between family history of migraine and PTM is warranted due to the emerging evidence that PTM is linked to poor outcomes following SRC.

The primary purpose of the current study was to determine if family history of migraine increases the likelihood of PTM in adolescent athletes following SRC. We expected that family history of migraine would be associated with an increased risk for PTM following

SRC in this population. We also wanted to extend previous research examining PTM and SRC outcomes (e.g., cognitive, vestibular, oculomotor) by examining the interaction between family history of migraine and PTM. We hypothesized that concussed athletes with PTM would demonstrate higher levels of cognitive, vestibular and oculomotor symptoms and impairment compared to concussed athletes without a history of family history of migraine or PTM, based on prior research[5,6,11]. We expected that family history of migraine, independent of current PTM, would be less likely to be associated with adverse outcomes following SRC, based on preliminary evidence that personal history of migraine was not associated with adverse outcomes following SRC[20].

METHODS

Study Design and Participants

We conducted a retrospective study that included two cohorts of 232 consecutive concussion patients who presented to a sports medicine concussion clinic September through December of 2014 (n= 122) and August 2015 through February 2016 (n= 110), secondary to research assistant availability to enroll participants into the clinical research registry. Eligible participants were athletes between the ages of 12–18 years, who had sustained their concussion during an organized sports practice or competition in the previous two weeks. Athletes with a history of prior concussion and ADHD/LD were included, as sample prevalence was consistent with population prevalence estimates[27,28] and no group differences existed. Athletes with no clear mechanism of injury, positive imaging findings, or other neurological history were excluded from the study. Athletes who were diagnosed with a concussion within a three-month period preceding the current injury were excluded. Athletes were also excluded if data were missing for family history of migraine, if family background data (e.g., adoption, health history of biological parent unknown) were unavailable, or the individual had a personal history of migraine.

Definitions and Measures

Concussion—Concussion was defined as a “complex pathophysiological process affecting the brain, induced by biomechanical forces” as specified in the most recent consensus statement on concussion in sports[16], consistent with ICD-10 codes of concussion (S06.0X1A), with or without loss of consciousness <30 minutes. Concussions were diagnosed at the time of injury (e.g., by certified ATC on the field) or at clinical evaluation (e.g., physician, neuropsychologist). For the purpose of this clinical study, the following criteria were implemented for concussion diagnosis: 1) clear mechanism of injury, and 2) presence of signs (e.g., loss of consciousness, amnesia, disorientation/confusion, balance difficulties) and/or at least one symptom (e.g., headache, dizziness, nausea) of concussion with onset immediately following the mechanism of injury.

Family history of migraine—Family history of migraine (as well as personal history) was self-reported to the clinician via the clinical interview. The athlete and parent were present and asked “Have you been diagnosed with migraines by a healthcare provider in the past?” and “Does [patient name] have any first degree relatives diagnosed with migraines?”

If the answer was “yes”, then the question was followed up with “which family member(s)?” to verify the patient or parent was referring to a biological mother, father or sibling.

Post-traumatic migraine—PTM was defined using the IHS guidelines for migraine (i.e., headache, nausea, and photo- and/or phono-sensitivity), and presence of PTM was determined based on responses to the Post Concussion Symptom Scale (PCSS).

Neurocognitive Testing—The Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) is a computer-based neurocognitive test battery comprised of 6 subtests designed to evaluate neurocognitive impairment in individuals with an SRC[29]. The ImPACT test yields four composite scores for verbal and visual memory, processing speed, and reaction time, and takes approximately 20 to 25 minutes to administer. ImPACT has adequate reliability and validity, and sensitivity[30,31]. ImPACT demonstrates moderate test-retest stability over time[32,33].

Post-concussion Symptom Scale (PCSS)—The Post-concussion Symptom Scale (PCSS) is a computerized self-report symptom inventory that includes 22 items representing the most commonly reported concussion symptoms, including somatic (e.g., nausea, headache), cognitive (difficulty concentrating, memory problems), affective (e.g., anxiety, depression), and sleep-related symptoms of concussion. The PCSS takes 5 minutes to administer, and participants rate each symptom on a 7-point Likert scale from 0 (none) to 6 (severe), based on their experience across the past two days. The PCSS has adequate reliability and validity for assessing and monitoring of SRC-related symptoms[34].

Vestibular/Ocular Motor Screening (VOMS): The VOMS is a screening tool developed to assess vestibular and ocular motor impairments via patient-reported symptom provocation on a scale of 0–10 symptom intensity for four symptoms (i.e., headache, dizziness, nausea, and fogginess) after each assessment domain[35]. Total symptom scores are computed for each VOMS component. The VOMS assesses the following 5 domains: (1) smooth pursuits, (2) horizontal and vertical saccades, (3) convergence, (4) horizontal vestibular ocular reflex (VOR), and (5) visual motion sensitivity (VMS). The VOMS also includes a measurement of near point convergence (NPC) distance based on the average of three measures. The internal consistency of the VOMS is excellent (Cronbach $\alpha = .92$)[35], and takes approximately 5 minutes to administer.

Procedures

The study procedures were approved under an expedited protocol by the institution’s human subjects review board. Participants and their parents provided written consent/assent (for minors) to be in the study. Licensed clinical neuropsychologists specializing in concussion care then conducted the following clinical evaluations, which took approximately 45–60 minutes, with each patient individually in private exam rooms (listed in order of administration): 1) Post-concussion Symptom Scale (PCSS), 2) computerized neurocognitive testing, 3) medical history (including migraine, concussion, learning disability, etc.), and 4) vestibular and oculomotor assessment. All measures were conducted in conjunction with each participant’s initial clinical visit for their concussion.

Data Analysis

Chi square analyses and t-tests were employed to determine if groups (family history of migraine and no family history of migraine) differed on any demographic characteristics (i.e., sex, age, and history of concussion, ADHD/LD), and injury characteristics (i.e., posttraumatic amnesia and loss of consciousness). A univariate nonparametric test (i.e., $\chi 2$ with odds ratios [ORs], 95% CI) was used to examine the association between family history of migraine and PTM status. Then two 2 × 2 MANCOVAS with grouping factors of family history of migraine (yes or no) and PTM status (yes or no), with covariate of concussion history, were employed to examine differences on 1) ImPACT composites scores/PCSS and 2) VOMS scores. When omnibus analyses with significant interactions were present, pairwise comparisons were conducted to examine between group differences. Bonferroni correction was applied and statistical significance for all tests was set at $p \leq .05$ for all analyses. Statistical analyses were performed using SPSS version 21.

RESULTS

Demographics

Of the 232 participants who met criteria for the study, complete data were available for 66% (153/232) participants, (103 males, 50 females) with a mean age of 15.72 +/− 1.48 (range 12–18). Overall, 45% (n=69) of the sample reported family history of migraine in one or more immediate family members, consistent with published lifetime incidence rate[22]. Participants were excluded due to missing data regarding family history of migraine (n=27) secondary to lack of clarity regarding diagnosis (e.g., self-reported headache with uncertainty regarding diagnosis from healthcare professional) or knowledge of family history (e.g., no contact with other biological parent), incomplete VOMS (n=41), and personal history of migraine (n=11). While the VOMS is a standardized component of the clinical evaluation patients may discontinue upon request due to discomfort, or it may be deferred due to other injury or pain (e.g., neck pain during VOR). Excluded participants did not differ on age (t=1.08, p=.28), days to initial evaluation (t=1.59, p=.11), sex ($\chi 2(1)$=.08, p=.47), history of LD ($\chi 2(1)$=.14, p=.58), ADHD ($\chi 2(1)$=.00, p=.63), or history of concussion ($\chi 2(1)$=.28, p=.37) compared to participants who had complete data and were included in analyses. Participants were athletes competing in contact/collision sports or limited contact sports, based on developed classification system[36]. The majority of participants sustained an SRC while playing American football (n=57, 37.3%) or boys'/girls' soccer (n=33, 21.6%). Participants also represented ice hockey (n=24, 15.7%), boys'/girls' basketball (n=20, 13.1 %), volleyball (n=8, 5.2%), rugby (n=3, 2.0%), field hockey (n=3, 2.0%), wrestling (n=3, 2.0%), softball (n=1, 0.6%), and martial arts (n=1, 0.6%). Demographic history is summarized in Table 1. Groups (family history of migraine and no history) did not differ on gender ($\chi 2(1)$=.23, p=.69), age (t=−.51, p=.61). History of LD ($\chi 2(1)$=2.05, p=.15) and ADHD ($\chi 2(1)$=.985, p=.32) were equally represented across groups. Groups did not differ on the presence of injury characteristics, including LOC ($\chi 2(1)$=1.45, p=.48) or PTA ($\chi 2(1)$=1.45, p=.23). There was no difference between groups for days from injury to first evaluation (t=−1.80, p=.08). However, participants with family history of migraine were more likely to report a personal history of concussion ($\chi 2(1)$=5.82, p=.02).

Relationship between Family history of migraine and PTM

Based on clinical evaluations that occurred 4.72+/−3.05 days (range 1–14 days) post injury, 44% (n=67) of participants met designated criteria for PTM. The results from the chi-square analyses revealed that 33% (n=28) of athletes with no family history of migraine met criteria for PTM compared to 57% (n=39) of athletes with familial history of migraine. Participants with a family history of migraine were 2.6 times (OR=2.60, [CI=1.35–5.02], p=.003) more likely to present with PTM symptoms in the first two weeks following SRC compared to participants without family history of migraine.

Interaction between Family history of migraine and PTM

Results of the 2 (Family history of migraine- yes or no) × 2 (PTM- yes or no) MANCOVA with covariate concussion history (yes or no) revealed a significant main effect for PTM (Wilk's λ=.65, $F_{5,144}$=15.43, p<.001, η2=.35) on neurocognitive scores (Table 2). Specifically, after controlling for concussion history (yes or no) worse performance among athletes with PTM was supported for all composites, including verbal memory (F=11.47, p=.001, η2=.07), visual memory (F=18.30, p< .001, η2=.11, visual motor speed (F=10.86, p=.001, η2=.07), and reaction time (F=5.42, p=.021, η2=.04). Similarly, after controlling for concussion history there was a main effect for PCSS symptom score (F=75.54, p<.001, η2=.34). There was no main effect for family history of migraine (Wilk's λ=.95, $F_{5,144}$=1.41, p=.223, η2=.05) or interaction for family history of migraine and PTM (Wilk's λ=.95, $F_{5,144}$=.836, p=.523, η2=.03). The second MANCOVA (covariate concussion history) with grouping factors, family history of migraine (yes or no) and PTM post injury (yes or no), comparing group performance on VOMS total scores also revealed a significant main effect for PTM (Wilk's λ=.70, $F_{7,\ 142}$=8.52, p < .001, η2=.30), with athletes meeting criteria for PTM reporting more symptoms on smooth pursuits (F=57.84, p<.001, η2=.28), horizontal saccades (F=52.68, p<.001, η2=.26), vertical saccades (F=39.00, p<.001, η2=.21), horizontal VOR (F=40.67, p<.001, η2=.22), vertical VOR (F=38.05, p<.001, η2=.21), and VMS (F=36.36, p<.001, η2=.20), but not NPC distance. After controlling for concussion history there was no main effect for family history of migraine (Wilk's λ=.98, $F_{7,142}$=.503, p=.831, η2=.02) or interaction of family history of migraine and PTM (Wilk's λ=.96, $F_{7,142}$=.888, p=.518, η2=.04) on VOMS scores.

DISCUSSION

This study examined the relationship between family history of migraine and the presence of PTM symptoms following SRC. The primary finding indicated that family history of migraine was associated with an increased likelihood of PTM following SRC. The current study also compared neurocognitive and vestibular/oculomotor outcomes of concussed athletes with PTM to those without. The results suggested that participants with PTM experienced worse neurocognitive and vestibular/oculomotor impairment following SRC, regardless of family history of migraine. These results extend findings from previous studies[6,11] documenting worse neurocognitive outcomes among athletes with PTM to also include increased vestibular/oculomotor symptoms and impairments. Therefore, despite the association between family history of migraine and onset of PTM following SRC, clinical outcomes following SRC were only influenced by the presence of PTM in the current study.

Few studies have examined the relationship between family history of migraine despite the strong genetic predisposition for migraine[24] and anecdotal reports of onset of migraine following concussion. One study that examined a cohort of children recruited for the ED found personal or family history of migraine was present in 82% of participants who reported persistent posttraumatic headaches three months post injury[17]. In the current study, athletes with family history of migraine were at an increased likelihood for PTM following concussion compared to athletes with no history of migraine, which indicate that SRC may be a catalyst for manifestation of PTM symptoms in adolescents who are genetically vulnerable. These data provide preliminary support for family history of migraine as a risk factor for exhibiting a PTM clinical profile following SRC.

The current study lends support to the previous research suggesting the presence of PTM predicts more severe neurocognitive outcomes following SRC[11,12]. Participants with a family history of migraine in the absence of PTM did not experience worse outcomes on neurocognitive measures. This finding is similar to other studies[20,37] that did not report a relationship between personal migraine history and neurocognitive deficits post injury. As hypothesized, athletes with PTM reported more symptoms on the PCSS[6,11] and were more symptomatic on vestibular screening, regardless of family history of migraine. This is not surprising, provided the complex relationship between vestibular functioning and migraines[38] and studies documenting vestibular dysfunction in migraine patients[14,39].

Another interesting finding was that 43.5% of our SRC sample met criteria for PTM, which is much higher than prior similar studies that included a more homogenous sample of only male football players[11] and a slightly older group[6]. These data highlight the high frequency of PTM symptoms in the subacute stages of injury, and may also be representative of more severe injuries as a product of being referred to a specialty clinic. It is also noteworthy that athletes with family history of migraine were more likely to have a history of prior concussion. Some researchers[40,41] have suggested athletes with a personal or family history of migraine may be at a greater risk for sustaining a concussion. McCrory noted an increased prevalence of migraine in athletes compared to the general population[42]. It is difficult to infer the nature of the relationship between migraine history and concussions from current studies. However, it is important to note that our study controlled for concussion history, and still found an effect for posttraumatic migraine.

Limitations

Although this study expands our understanding of the relationship between migraine and SRC outcomes there are limitations. Patients who could not tolerate the VOMS due to severity of symptoms, neck pain, etc. were excluded from the analysis, potentially biasing the sample. Patients were recruited from a specialty concussion clinic with a large time interval (1–13 days post injury), which introduces the possibility of selection bias (e.g., more severe injury) – this is certainly possible provided the very high rate of PTM compared to other studies. Relying on a symptom inventory may not be the ideal method for defining PTM, as it does not specify the co-occurrence of headache, nausea, and photo/phonophobia, and includes varying symptom intensity – future prospective studies may find benefit in developing more sophisticated approaches to defining PTM. Additionally, while our low

base rate of personal migraine history in adolescents (n=11, 6.7%) was consistent with population prevalence for this young age group[43,44], we were unable to further break down the migraine history group into those with personal history and those with family history of migraine. While our groups' were similar with regard to other preexisting and comorbid conditions (e.g., ADHD)[45,46], larger studies with multiple personal and family risk factors are needed. While age was available for all participants, grade level was missing data on the ImPACT demographics section for several participants. We did not collect data on race or ethnicity, although our sample was predominantly white, and findings may not be generalizable to other populations. Future research with recruitment of a more diverse sample is necessary. Although participants were asked if migraine was previously diagnosed by a healthcare provider, data were all collected via self-report and accuracy cannot be confirmed. Similarly, participants with undiagnosed migraines pre-injury may been have erroneously categorized, and other comorbidities that may also lend to genetic predisposition for worse outcomes (e.g., mental health history) were not included or controlled for in our analyses. Future research should explore outcomes at multiple time points post injury, consider pubertal status, and determine if risk for PTM extends beyond the acute and subacute phases of SRC recovery.

Clinical Implications

Understanding risk factors and modifiers for concussion and recovery outcomes is vital to providing appropriate education and feedback to athletes and their families[47]. This study provides an opportunity to educate athletes and parents on family history of migraine as a potential risk factor for poor outcomes following concussion, possibly in a preseason educational setting. Our findings may help inform management immediately following concussion. Specifically, knowing that a patient has family history of migraine in conjunction with the patient's initial presentation, may help individualize treatment recommendations. For example, providers may implement behavioral recommendations for migraine[48], and consider pharmacological intervention for headache earlier than usual following injury. Further research is necessary to clarify the complex relationships among concussion risk, poor post injury outcomes, and migraine. Nonetheless, our findings on family history of migraine are congruent with the literature suggesting personal history of migraine is associated with concussion risk, and clinicians should consider these findings when providing feedback and recommendations to patients with these comorbidities or genetic predisposition for migraine. Specifically, this subgroup of athletes should be counseled on increased risk for sustaining another concussion, as well as potentially higher risk of posttraumatic migraine, as these are important considerations in deciding to return to play. Likewise, clinicians may consider more conservative management among this subgroup of athletes, provided potential increased risk for reinjury and negative outcomes.

Conclusion

The current study supported an association between family history of migraine and PTM symptoms following SRC, suggesting a genetic predisposition for migraine may serve as a catalyst or trigger for onset of PTM. Results of the study were consistent with prior research suggesting increased symptom report and more severe neurocognitive impairment in athletes with PTM, but also expanded findings to increased symptom report on vestibular screening.

Further, in addition to personal migraine history and PTM, family history of migraine may also be regarded as a secondary risk factor to consider in clinical management.

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References

1. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. The Journal of Head Trauma Rehabilitation. 2006; 21(5):375–378. [PubMed: 16983222]
2. Henry LC, Elbin R, Collins MW, Marchetti G, Kontos AP. Examining recovery trajectories after sport-related concussion with a multimodal clinical assessment approach. Neurosurgery. 2016; 78(2):232–241. [PubMed: 26445375]
3. Ellis MJ, Leddy JJ, Willer B. Physiological, vestibulo-ocular and cervicogenic post-concussion disorders: An evidence-based classification system with directions for treatment. Brain Injury. 2014; (0):1–11.
4. Collins M, Kontos A, Reynolds E, Murawski C, Fu F. A comprehensive, targeted approach to the clinical care of athletes following sport-related concussion. Knee Surgery, Sports Traumatology, Arthroscopy. 2014; 22(2):235–246.
5. Mihalik JP, Register-Mihalik J, Kerr ZY, Marshall SW, McCrea MC, Guskiewicz KM. Recovery of posttraumatic migraine characteristics in patients after mild traumatic brain injury. The American journal of sports medicine. 2013; 41(7):1490–1496. [PubMed: 23696213]
6. Mihalik JP, Stump JE, Collins MW, Lovell MR, Field M, Maroon JC. Posttraumatic migraine characteristics in athletes following sports-related concussion. Journal of Neurosurgery. 2005; 102(5):850–855. [PubMed: 15926709]
7. Heyer GL, Young JA, Rose SC, McNally KA, Fischer AN. Post-traumatic headaches correlate with migraine symptoms in youth with concussion. Cephalalgia. 2015; 36(4):309–316. [PubMed: 26054363]
8. Society HCCotIH. The international classification of headache disorders, (beta version). Cephalalgia. 2013; 33(9):629–808. [PubMed: 23771276]
9. Giza CC, Hovda DA. The neurometabolic cascade of concussion. Journal of Athletic Training. 2001; 36(3):228. [PubMed: 12937489]
10. Meehan WP, d'Hemecourt P, Comstock RD. High School Concussions in the 2008–2009 Academic Year Mechanism, Symptoms, and Management. The American journal of sports medicine. 2010; 38(12):2405–2409. [PubMed: 20716683]
11. Kontos AP, Elbin R, Lau B, et al. Posttraumatic migraine as a predictor of recovery and cognitive impairment after sport-related concussion. The American journal of sports medicine. 2013; 41(7): 1497–1504. [PubMed: 23698389]
12. Kontos AP, Reches A, Elbin R, et al. Preliminary evidence of reduced brain network activation in patients with post-traumatic migraine following concussion. Brain imaging and behavior. 2015:1–10. [PubMed: 25724689]
13. Register-Mihalik JK, Mihalik JP, Guskiewicz KM. Balance deficits after sports-related concussion in individuals reporting posttraumatic headache. Neurosurgery. 2008; 63(1):76–82. [PubMed: 18728571]
14. Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria. Journal of Vestibular Research. 2012; 22(4):167. [PubMed: 23142830]
15. Singman EL, Matta NS, Silbert DI. Convergence Insufficiency Associated with Migraine: A Case Series. American Orthoptic Journal. 2014; 64(1):112–116. [PubMed: 25313120]
16. McCrory P, Meeuwisse WH, Aubry M, et al. Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012. British Journal of Sports Medicine. 2013; 47(5):250–258. [PubMed: 23479479]

17. Kuczynski A, Crawford S, Bodell L, Dewey D, Barlow KM. Characteristics of post-traumatic headaches in children following mild traumatic brain injury and their response to treatment: a prospective cohort. Developmental Medicine & Child Neurology. 2013; 55(7):636–641. [PubMed: 23560811]
18. Morgan CD, Zuckerman SL, Lee YM, et al. Predictors of postconcussion syndrome after sports-related concussion in young athletes: a matched case-control study. Journal of Neurosurgery: Pediatrics. 2015; 15(6):589–598. [PubMed: 25745949]
19. Eisenberg MA, Andrea J, Meehan W, Mannix R. Time interval between concussions and symptom duration. Pediatrics. 2013; 132(1):8–17. [PubMed: 23753087]
20. Covassin T, Crutcher B, Belanger S. Preinjury History of Migraine Headache: Effects on Neurocognitive Performance and Symptoms in Athletes With Concussion. Athletic Training & Sports Health Care. 2014; 6(5):220.
21. Merikangas KR. Contributions of epidemiology to our understanding of migraine. Headache: The Journal of Head and Face Pain. 2013; 53(2):230–246.
22. Stewart W, Wood C, Reed M, Roy J, Lipton R. Cumulative lifetime migraine incidence in women and men. Cephalalgia. 2008; 28(11):1170–1178. [PubMed: 18644028]
23. Wöber-Bingöl Ç. Epidemiology of migraine and headache in children and adolescents. Current pain and headache reports. 2013; 17(6):1–11.
24. Gasparini CF, Sutherland HG, Griffiths LR. Studies on the pathophysiology and genetic basis of migraine. Current genomics. 2013; 14(5):300. [PubMed: 24403849]
25. Russell MB, Olesen J. Increased familial risk and evidence of genetic factor in migraine. Bmj. 1995; 311(7004):541–544. [PubMed: 7663209]
26. Stewart WF, Staffa J, Lipton RB, Ottman R. Familial risk of migraine: A population-based study. Annals of neurology. 1997; 41(2):166–172. [PubMed: 9029065]
27. Abu Arafeh I, Razak S, Sivaraman B, Graham C. Prevalence of headache and migraine in children and adolescents: a systematic review of populationâ€ based studies. Developmental Medicine & Child Neurology. 2010; 52(12):1088–1097. [PubMed: 20875042]
28. Pastor PN, Reuben CA. Diagnosed attention deficit hyperactivity disorder and learning disability: United States, 2004–2006. Vital and health statistics. Series 10, Data from the National Health Survey. 2008; (237):1–14.
29. Immediate Post-Concussion Assessment Testing (ImPACT®) Test. ImPACT Applications – technical manual. 2011
30. Barr WB, McCrea M. Sensitivity and specificity of standardized neurocognitive testing immediately following sports concussion. Journal of the International Neuropsychological Society. 2001; 7(06):693–702. [PubMed: 11575591]
31. Schatz P, Pardini JE, Lovell MR, Collins MW, Podell K. Sensitivity and specificity of the ImPACT Test Battery for concussion in athletes. Archives of clinical neuropsychology. 2006; 21(1):91–99. [PubMed: 16143492]
32. Broglio SP, Ferrara MS, Macciocchi SN, Baumgartner TA, Elliott R. Test-retest reliability of computerized concussion assessment programs. Journal of Athletic Training. 2007; 42(4):509. [PubMed: 18174939]
33. Schatz P. Long-term test-retest reliability of baseline cognitive assessments using ImPACT. The American journal of sports medicine. 2010; 38(1):47–53. [PubMed: 19789333]
34. Lovell MR, Iverson GL, Collins MW, et al. Measurement of symptoms following sports-related concussion: reliability and normative data for the post-concussion scale. Applied neuropsychology. 2006; 13(3):166–174. [PubMed: 17361669]
35. Mucha A, Collins MW, Elbin R, et al. A Brief Vestibular/Ocular Motor Screening (VOMS) Assessment to Evaluate Concussions Preliminary Findings. The American journal of sports medicine. 2014; 42(10):2479–2486. [PubMed: 25106780]
36. Rice SG. Medical conditions affecting sports participation. Pediatrics. 2008; 121(4):841–848. [PubMed: 18381550]
37. Sandel N, Johnson E, Pardini J, Sufrinko A, Lovell M. C-31Gender and History of Migraine Do Not Modify Performance on Computer-Based Neurocognitive Testing at Baseline or Post-Concussion. Archives of Clinical Neuropsychology. 2014; 29(6):584–584.

38. Bisdorff A. Migraine and dizziness. Current opinion in neurology. 2014; 27(1):105–110. [PubMed: 24316729]
39. Dieterich M, Brandt T. Episodic vertigo related to migraine (90 cases): vestibular migraine? Journal of neurology. 1999; 246(10):883–892. [PubMed: 10552234]
40. Gordon K, Dooley J, Wood E. Is migraine a risk factor for the development of concussion? British Journal of Sports Medicine. 2006; 40(2):184–185. [PubMed: 16432009]
41. Kutcher JS, Eckner JT. At-risk populations in sports-related concussion. Current sports medicine reports. 2010; 9(1):16–20. [PubMed: 20071916]
42. McCrory P, Heywood J, Coffey C. Prevalence of headache in Australian footballers. British journal of sports medicine. 2005; 39(2):e10–e10. [PubMed: 15665188]
43. Lu SR, Fuh JL, Juang KD, Wang SJ. Migraine prevalence in adolescents aged 13–15: a student population-based study in Taiwan. Cephalalgia. 2000; 20(5):479–485. [PubMed: 11037745]
44. Lipton RB, Bigal ME. The epidemiology of migraine. The American Journal of Medicine Supplements. 2005; 118:3–10.
45. Genizi J, Gordon S, Kerem NC, Srugo I, Shahar E, Ravid S. Primary headaches, attention deficit disorder and learning disabilities in children and adolescents. The journal of headache and pain. 2013; 14(1):1. [PubMed: 23566305]
46. Genizi J, Matar AK, Schertz M, Zelnik N, Srugo I. Pediatric mixed headache-The relationship between migraine, tension-type headache and learning disabilities-in a clinic-based sample. The journal of headache and pain. 2016; 17(1):1.
47. Elbin R, Covassin T, Gallion C, Kontos AP. Factors influencing risk and recovery from sport-related concussion: reviewing the evidence. SIG 2 Perspectives on Neurophysiology and Neurogenic Speech and Language Disorders. 2015; 25(1):4–16.
48. Singer AB, Buse DC, Seng EK. Behavioral treatments for migraine management: Useful at each step of migraine care. Current neurology and neuroscience reports. 2015; 15(4):1–8.

Table 1

Summary of Demographic and Descriptive Data

	Familial Migraine (n=69)	No History (n=84)	Total (n=153)
Sex	69.57% male (n=48)	65.48% male (n=55)	67.30% male (n=103)
Sport type (collision/contact or limited contact)	98.55% collision/contact (n=68)	97.62% collision/contact (n=82)	98.04% collision/contact (n=150)
Age	15.79+/−1.27	15.67+/−1.64	15.72+/−1.48
Concussion History (%) *	43.47% (n=30)	10.96% (n=21)	33.33% (n=51)
History of ADHD (%)	4.35% (n=3)	8.33% (n=7)	6.54% (n=10)
History of LD (%)	7.25% (n=5)	2.39% (n=2)	4.58% (n=7)
Days until evaluation	5.20+/−3.60	4.30+/−2.49	4.72+/−3.05

Values are expressed as mean +/− standard deviation, except where % is indicated

* group differences, p<.05

Table 2

Neurocognitive and Vestibular/Oculomotor scores by groups

Measure/Score	No History (n=84)		Familial Migraine (n=69)	
	No PTM (n=56)	PTM (n=28)	No PTM (n=30)	PTM (n=39)
Neurocognitive Test Scores (ImPACT)				
Verbal Memory *	80.54+/−15.54	69.25+/−17.74	80.24+/−15.30	72.23+/−13.73
Visual Memory*	68.32+/−13.16	59.46+/−14.00	73.83+/−14.22	62.69+/−13.04
Motor Speed *	34.70+/−8.53	31.41+/−9.51	37.96+/−7.20	31.10+/−6.48
Reaction Time *	0.66+/−0.14	0.70+/−0.12	0.63+/−0.10	0.73+/0.18
Symptoms (PCSS) *	17.32+/−13.31	43.29+/−19.18	17.93+/−15.97	44.44+/−20.54
Vestibular/Oculomotor Screening				
Pursuits *	4.21+/−3.64	9.89+/−5.11	3.57+/−3.05	10.22+/−6.53
H Saccades *	4.52+/−3.62	10.70+/−5.76	4.63+/−4.43	10.79+/−6.26
V Saccades *	5.01+/−5.15	10.55+/−5.59	4.70+/−4.53	11.08+/−6.61
H VOR *	5.31+/−5.51	11.77+/−5.49	5.40+/−4.97	11.71+/−6.89
V VOR *	5.46+/−4.95	11.80+/−5.57	5.27+/−4.96	11.32+/−7.50
VMS *	6.46+/−6.04	13.55+/−6.16	6.33+/−5.69	12.55+/−7.66
NPC	5.19+/−5.90	4.24+/−5.08	5.03+/−5.50	6.13+/−6.55

*Main effects for PTM