ORIGINAL ARTICLE

Does the Face Protect the Brain?

A Case-Control Study of Traumatic Brain Injury and Facial Fractures

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Background: The relationship between facial fractures and traumatic brain injury is controversial. Some studies show an increased risk of brain injury with the presence of facial fractures while others claim that facial fractures protect against brain injury.

Objective: To examine the association between facial fractures and traumatic brain injuries.

Design: Case-control study.

Setting: Subjects were recruited from the emergency departments of 7 hospitals in the Seattle, Wash, area.

Patients: Three thousand eight hundred forty-nine injured bicyclists and 5 scene deaths were identified from March 1, 1992, to August 31, 1994, with complete data available on 3388 bicyclists.

Interventions: None.

Results: The study group was composed of 1602 cases with injuries to the head, face, or brain and 1540 control subjects. There were 203 bicyclists with traumatic brain injuries, of whom 62 had an identifiable intracranial injury and 141 suffered a concussion. A total of 81 patients sustained facial fractures. The odds ratio for the risk of intracranial injury associated with facial fractures after adjustment for significant confounders was 9.9 (95% confidence interval, 5.1-19.3). The effect was less strong but still present when all traumatic brain injuries including concussions were considered (odds ratio, 2; 95% confidence interval, 1.1-3.7). No association was found for concussion only.

Conclusions: This study demonstrates no evidence that facial fractures help prevent traumatic brain injury. Data indicate that facial fractures are markers for increased risk of brain injury.

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IT HAS BEEN proposed that the face protects the brain from injury the way an airbag protects the chest in a motor vehicle crash. Actual data on this are scant and conflicting. Lee et al[1] reported that facial fractures are associated with a decreased risk of a traumatic brain injury, while Davidoff et al[2] found facial fracture to be highly associated with traumatic brain injury. This question has important clinical implications. Multiple origins and potentially significant confounding variables make accurate assessment of the association between traumatic head injury and facial fractures difficult.

In our study, we used a large database of individuals with injuries from bicycle crashes to examine the association between traumatic brain injury and facial fracture using a case-control design.

RESULTS

Seven hospital EDs treated 3849 injured bicyclists, 5 of whom died at the scene during the 2½-year study. Two bicyclists were excluded because of incomplete data, leaving 3388 injured cyclists for analysis. Of these bicyclists, 1602 had head, face, or brain injury or recounted that they hit their helmet, head, or face. They constitute the study group. There were 203 bicyclists (14.4%) with traumatic brain injuries of whom 62 (3.9%) had intracranial injuries.

Cases (intracranial injuries, n = 62) and controls (all other face or head injured bicyclists, n = 1540) did not differ significantly by age or sex; however, more controls (45.8%) than cases (24.2%) were wearing a helmet (**Table 1**). Characteristics of the crash (**Table 2**), including the bicyclists' self-reported speed and the type of surface (paved vs other) were not different between cases and controls; however, bicyclists whose speed was unknown were at a higher risk of intracranial injury (OR, 10.7; 95% CI, 3.3-33.3), most likely reflecting a recall bias for patients suffering intracranial injury. Col-

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MATERIALS AND METHODS

DATA COLLECTION

Our current study data were collected as part of a case-control study examining head injuries caused by bicycle crashes. Methods of data collection are described in detail in a larger study that examined the effectiveness of bicycle safety helmets in preventing head injuries.[3] Our case-control study was conducted at 7 major hospitals in the Seattle, Wash, area: Central and Eastside Hospitals of Group Health Cooperative of Puget Sound, a large health maintenance organization; University of Washington Medical Center; Harborview Medical Center, a regional level I trauma center; Overlake Hospital, a community hospital that cares for a large portion of trauma patients; and Children's Hospital and Medical Center of Seattle and the Mary Bridge Hospital and Medical Center of Tacoma, 2 children's hospitals. The medical examiner's offices of Pierce and King counties (Washington) were visited regularly to identify deaths at the scene that might have eluded the surveillance system.

SUBJECT IDENTIFICATION

Subjects were identified prospectively by regular surveillance (1 to 2 times a week) of the emergency department (ED) logs and records in each of the 7 study hospitals from March 1, 1992, to August 31, 1994. Automated hospital admission records were screened monthly by *International Classification of Diseases, Ninth Revision, Clinical Modification* coding (*ICD-9-CM* E codes 800-802, 805-807, 810-816, 818-823, 825-829) to identify admissions that were not recorded in ED records. Any individual injured on a bicycle was eligible for the study, including child passengers (<6 years) riding in a child carrier, (n = 4), but excluding older bicycle passengers, pedestrians injured by a bicycle, and individuals assaulted while riding a bicycle.

MEDICAL RECORD ABSTRACTION

Trained abstractors reviewed the complete medical record of each patient, recording the following: mental status (nausea, headaches, amnesia, and length of unconsciousness), description of the incident, Glasgow Coma Scale scores, radiological procedures, helmet use, and a detailed description of all injuries. All radiology reports, surgical procedures, and discharge summaries were reviewed. Medical examiners' reports were abstracted for all deaths. A computer software program (TRICODE, TRIANALYTICS, Bel Air, Md) was used to convert text descriptions into *ICD-9-CM* codes and to calculate Abbreviated Injury Scale score and Injury Severity Score.

DEFINITIONS

Patients who sought care for a bicycle-related traumatic brain injury in the ED of a study hospital during the period from March 1, 1992, to August 31, 1994, were enrolled in this study. They were classified by injury into 2 groups: those with intracranial injuries and those with concussions. Intracranial injury included the following: cerebral lacerations, cerebral contusions, and subarachnoid, subdural, and extradural hemorrhages. Patients with loss of consciousness without signs of intracranial hemorrhage were defined as having a concussion. Loss of consciousness was defined by either a transient state of witnessed unresponsiveness or a patient report of temporary loss of awareness. A large group of individuals with traumatic brain injury were defined as having intracranial hemorrhage/contusions and a concussion. Control subjects were defined as bicyclists treated in the same EDs who reported hitting their helmet, head, or face or sustaining an injury to their head or face, without any traumatic brain injury. This control group was chosen to include only those bicyclists at risk of traumatic brain injury and/or facial fracture. Facial injury was defined as any injury to the jaw, lips, cheeks, nose, ears (external), eyes (external), forehead, or mouth (intraoral). Facial fracture was defined as injury to the maxilla, orbits, zygoma, or mandible.

QUESTIONNAIRES

Detailed questionnaires were sent to all injured bicyclists treated in this period regarding the circumstances of the crash, including helmet use, speed, and motor vehicle involvement. Telephone follow-up occurred within 2 weeks of the ED visit. Response rate to the questionnaires was 88%; complete data were available for analysis on 3388 injured bicyclists.

STATISTICAL ANALYSIS

The SAS statistical package was used for all analyses (SAS Institute Inc, Cary, NC). Descriptive information and crude odds ratios (ORs) were generated for all traumatic brain injuries. Unconditional logistic regression modeling was performed for traumatic brain injuries to estimate the OR and 95% confidence intervals (CIs) of traumatic brain injury while controlling for multiple confounders.[4]

To determine whether facial fracture was associated with severity of brain injury, analyses of all cases of traumatic brain injury, as well as the subcategories of intracranial injury and concussions were done.

lision with a motor vehicle occurred in 55% of cases, which significantly increased the odds of intracranial injury (OR, 6.6; 95% CI, 4.2-10.4).

A total of 81 patients had facial fractures distributed between cases (29%) and controls (4.1%), including the following: 31 mandibular fractures or dislocations (28.3%); 6 maxillary fractures (7.4%); 29 nasal fractures (35.8%); and 15 orbital fractures (18.5%). Characteristics of cyclists' injuries are given in **Table 3**. Bicyclists with intracranial injuries were more likely to have concomitant neck injuries (OR, 3.1; 95% CI, 1.3-7.1). Injury Severity Scores ranged from 0 to 75; 24.2 % of the patients and 99.6% of controls had scores lower than 15. Patients with traumatic brain injuries had higher Injury Severity Scores, with 66.1% of the cases having scores of 16 to 40 compared with 0.4% of controls, and 9.7% of the cases having scores of 41 to 75 compared with no controls. The Glasgow Coma Scale scores were higher in the control group, with 98.9% of controls receiving scores from 12 to 15 compared with 56.5% of the cases.

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Table 1. Demographic Characteristics of and Helmet Use by Patients and Controls With Intracranial Injury Due to a Bicycle Crash

Characteristic	No. (%) of Intracranial Injury: Patients	No. (%) of Intracranial Injury: Controls	Unadjusted Odds Ratio (95% CI*)
Sex			
Male	46 (74.2)	1116 (72.5)	1.1 (0.6-2.0)
Female	16 (25.8)	424 (27.5)	1.0†
Age, y			
<13	32 (51.6)	758 (49.2)	1.0 (0.6-1.7)
13-19	6 (9.7)	238 (15.5)	0.6 (0.2-1.4)
≥20	24 (38.7)	544 (35.3)	1.0†
Helmet use	15 (24.2)	720 (45.8)	0.4 (0.2-0.7)
No helmet use	47 (75.8)	820 (54.2)	1.0†

*CI indicates confidence interval.
†Reference group.

Table 2. Characteristics Contributing to Crash Severity for Patients Involved in Bicycle Crashes

Characteristic	No. (%) of Intracranial Injury: Patients	No. (%) of Intracranial Injury: Controls	Unadjusted Odds Ratio (95% CI*)
Self-reported speed			
Slow (<5 mph)	24 (38.7)	517 (33.6)	1.0†
Moderate (5-15 mph)	17 (27.4)	657 (42.6)	0.6 (0.3-1.1)
Fast (>15 mph)	16 (25.8)	356 (23.1)	1.0 (0.5-2.0)
Unknown	5 (8.1)	10 (0.7)	10.7 (3.3-33.3)
Paved surface	54 (87.1)	1259 (81.8)	1.5 (0.7-3.2)
Other surface	8 (12.9)	281 (18.2)	1.0†
Motor vehicle collision	34 (54.8)	240 (15.6)	6.6 (4.2-10.4)
Other collisions	28 (45.2)	1300 (84.4)	1.0†

*CI indicates confidence interval.
†Reference group.

The OR for the effect of facial fracture on the risk of intracranial injury was adjusted for significant confounders including helmet use, motor vehicle involvement, and speed. Further adjustment for other variables including sex, age, and surface type had almost no effect on the OR. The adjusted OR for the risk of intracranial injury (n = 62) associated with facial fracture was 9.9 (95% CI, 5.1-19.3). The effect was weakened, but still present, when all traumatic brain injuries (n = 203), including concussions, were considered (OR, 2; 95% CI, 1.1-3.7); when only concussions (n = 141) were considered, no association was found (OR, 0.6; 95% CI, 0.2-1.6).

COMMENT

This study demonstrates no evidence that facial fractures help prevent traumatic brain injury. In fact, the risk of intracranial injury in those bicyclists with facial injury was increased almost 10-fold, and the risk for all brain injuries including concussion was doubled. This does not imply that facial fractures cause traumatic brain injury, but suggests that blunt impact with enough force to break facial bones could also produce

Table 3. Characteristics of Injuries Sustained From Bicycle Crashes for Patients Seen in an Emergency Department

Characteristic	No. (%) of Intracranial Injury: Patients	No. (%) of Intracranial Injury: Controls	Unadjusted Odds Ratio (95% CI*)
All facial fractures	18 (29.0)	63 (4.1)	9.6 (5.8-15.9)
Orbit	7 (11.3)	8 (0.5)	24.4 (7.6-77.3)
Maxilla	5 (8.1)	1 (0.07)	135 (14.6-6388)
Nose	2 (3.2)	27 (1.8)	1.9 (0.2-7.7)
Mandible	4 (6.4)	27 (1.8)	3.9 (0.95-11.6)
Neck injury	6 (9.7)	52 (3.4)	3.1 (1.3-7.1)
Injury Severity Score			
0-15	15 (24.2)	1534 (99.6)	1.0†
16-40	41 (66.1)	6 (0.4)	698.8 (238-2179)
41-75	6 (9.7)	0 (0)	. . .‡
Glasgow Coma Scale scores			
<7-9	22 (35.5)	6 (0.4)	160 (57-502)
10-12	5 (8.0)	10 (0.7)	21.8 (5.5-73.9)
12-15	35 (56.5)	1524 (98.9)	1.0†

*CI indicates confidence interval.
†Reference group.
‡Odds ratio not calculated due to no injuries in the control group.

brain injury. Patients with intracranial injuries were more likely to have been involved in a crash involving a motor vehicle, to have had a higher Injury Severity Scale score, and to have coexisting neck injuries than bicyclists who had an injury to the head or face that suggested a more severe crash. Importantly, no protective effect of facial fracture was found, even in bicyclists with less severe brain injury such as concussion. In the group who had concussions, no association with facial fracture was observed.

These results disagree with the previously reported decreased risk of traumatic brain injury in patients with facial fracture. Lee et al[1] theorized that the facial bones act as a protective cushion for the brain to explain why injuries that crush the facial bones frequently caused no apparent brain damage. Our results are more in agreement with Davidoff et al[2] who reported a 55% incidence of concomitant facial fracture and brain injury (defined as loss of consciousness or posttraumatic amnesia) and a 6% incidence of intracranial injury in a retrospective case series of 156 patients admitted to an ED with facial fractures from a variety of causes; however, the Davidoff study lacked a control group.

There are some limitations to our study. The controls were limited to patients who fell and hit their head or face and sought care for a bicycle-related injury. Those patients who fell, but were not badly injured, and did not seek medical care were excluded from this study. This could underestimate the association of facial fracture with brain injury.

Our study only examined 1 population, bicyclists who crashed, so we cannot comment on other mechanisms of injury that are reported as causes of facial fractures such as motor vehicle crashes or assaults.[5,6] While our choice of patient population limits generalizability, it is a homogeneous data set from which to draw associations.

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CONCLUSIONS

Our study shows no evidence that facial fractures are protective of traumatic brain injury. In fact, the risk of intracranial hemorrhage in bicyclists with a facial injury was increased by almost 10-fold, and the risk of any traumatic brain injury including a concussion was doubled. This suggests that the presence of enough force to cause facial fractures is likely to produce brain damage. Facial fractures are a marker for an increased risk of injury to the brain rather than protection against traumatic brain injury. Patients with facial fractures seen in the ED need to be evaluated for potentially serious brain injury.

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JAMA

Molecular Staging of Malignant Melanoma: Correlation With Clinical Outcome

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Context: For most solid tumors, the metastatic status of regional lymph nodes is the strongest predictor of relapse and survival. However, routine pathological examination of lymph nodes may underestimate the number of patients with melanoma who have nodal metastases.
Objective: To determine the clinical significance of a highly sensitive molecular assay for occult nodal metastases for the staging of patients with melanoma.
Design: A prospective cohort study of consecutive patients in which lymphatic mapping and sentinel lymph node (SLN) biopsy were performed on 114 melanoma patients with clinical stage I and stage II disease. The SLNs were bivalved, and half of each specimen was submitted for routine pathological examination. The other half was submitted for molecular detection of submicroscopic metastases using a reverse transcriptase–polymerase chain reaction (RT-PCR) assay for tyrosinase messenger RNA as a marker for the presence of melanoma cells. Patient follow-up averaged 28 months.
Setting: A major university-based melanoma referral center at a National Cancer Institute–designated cancer center.
Patients: A total of 114 patients with newly diagnosed cutaneous malignant melanoma who were at risk for regional nodal metastases.
Main Outcome Measure: Melanoma recurrence and overall survival.
Results: Twenty-three patients (20%) had pathologically positive SLNs, and all of these patients were also RT-PCR positive. Of the 91 pathologically negative patients, 44 were RT-PCR negative and 47 were RT-PCR positive. There was a recurrence rate among 14 (61%) of the 23 patients who were both pathologically and RT-PCR positive and a recurrence rate among 1 (2%) of 44 patients who were both pathologically and RT-PCR negative. For patients who were upstaged by the molecular assay (pathologically negative, RT-PCR positive), there was a recurrence rate among 6 (13%) of 47 patients. The differences in recurrence rates and overall survival between the pathologically negative, RT-PCR–negative and pathologically negative, RT-PCR–positive patient groups were statistically significant ($P = .02$ for disease-free survival and for overall survival). In both univariate and multivariate regression analyses, the histological and RT-PCR status of the SLNs were the best predictors of disease-free survival.
Conclusions: The use of an RT-PCR assay for detection of submicroscopic melanoma metastases in SLNs improved the prediction of melanoma recurrence and overall survival over routine pathological examination. (*1998;280:1410-1415*)

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