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Blunt cerebrovascular injury incidence, stroke-rate, and mortality with the expanded Denver criteria ☆☆☆☆☆

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ABSTRACT

Background: Blunt carotid and vertebral artery injury, collectively termed blunt cerebrovascular injury occur in less than 1% of blunt traumas. Conventional indications for screening miss up to 20% of blunt cerebrovascular injuries. Therefore, the expanded Denver criteria were created in 2012. We hypothesized the introduction of the expanded Denver criteria would lead to an increase in the national detection of blunt cerebrovascular injury with a subsequent decrease in stroke rate.

Methods: The National Trauma Data Bank was queried for blunt trauma admissions. Patients were divided into 2 groups: pre-expanded Denver criteria (2007–2011) or post-expanded Denver criteria era (2013–2015). The primary endpoint was the incidence of blunt cerebrovascular injury, which was used as a surrogate for detection.

Results: There were 10,183 blunt cerebrovascular injuries with 5,364 blunt cerebrovascular injuries in the pre-expanded Denver criteria group (0.19%) and 4,819 blunt cerebrovascular injuries in the post-expanded Denver criteria group (0.22%; $P < .001$). The stroke-rate in the post-expanded Denver criteria was significantly higher (9.2% vs 5.5%; OR 2.73, CI 2.29–3.25, $P < .001$). The strongest associated injury with blunt cerebrovascular injury was skull-base fracture (OR 3.61, CI 3.46–3.77, $P < .001$).

Conclusion: The detection of blunt cerebrovascular injury has increased by 16% since the publication of the expanded Denver criteria. Skull-base fracture is the strongest traumatic risk factor for blunt cerebrovascular injury. Although detection may have increased, the stroke-rate nearly doubled in the post-eDC era. This warrants future research.

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Introduction

Blunt carotid artery injury (CAI) and vertebral artery injury (VAI), collectively termed blunt cerebrovascular injury (BCVI), occur in less than 1% of blunt traumas.¹ They typically result from hyperextension or hyperflexion of the neck in the setting of a high-speed deceleration injury.² BCVI was historically underrecognized, with the diagnosis established only after the onset of neurologic symptoms. Delayed diagnosis is associated with morbidity and mortality rates of up to 80% and 40%, respectively.³ In an effort

to increase the early diagnosis of BCVI in the late 1990s, Dr. Biffi and colleagues at Denver Health proposed several screening criteria based on the mechanism of injury and physical signs.⁴ Additionally, formal angiography has been supplanted by a more readily available computed tomographic angiography (CTA) of the neck as the initial screening test of choice.^{5,6}

In 2012, the Denver group expanded their neck CTA screening criteria for BCVI, since older criteria missed 16–20% of these injuries.^{7–9} These additional criteria included presence of mandible fracture, complex skull fracture, traumatic brain injury (TBI), scalp degloving, upper rib fractures, and thoracic vascular injuries.⁹ The expanded Denver criteria (eDC) has been subsequently shown in a single center study to identify patients not previously captured by the pre-eDC criteria.¹⁰ We hypothesized the introduction of eDC would lead to an overall increase in the national detection of BCVI. We additionally attempted to determine whether an increased detection of BCVI would lead to a decrease in stroke rate and mortality for patients with BCVI. Finally, we sought to identify separate

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risk factors for CAI and VAI, as well as the development of stroke in patients with BCVI, using a national database.

Methods

We queried the National Trauma Data Bank (NTDB) from January 2007 through December 2015 to identify all blunt trauma patients presenting with BCVI using the International Classification of Diseases version 9 (ICD-9) diagnosis codes listed in [Appendix A](#), along with a blunt mechanism of injury. Patients were then divided into 2 groups: pre-eDC (2007–2011) or post-eDC era (2013–2015). We separated the 2 time periods by 1 year to allow a transition period while trauma centers started to adopt the eDC. The primary endpoint of interest was the incidence of BCVI, which we used as our surrogate for increased detection as there is no compelling evidence to suggest that the true incidence of BCVI has increased. Secondary outcomes included in-hospital cerebrovascular accident (CVA) and mortality.

Patient demographic information including age, gender, and presence of hypotension (systolic blood pressure < 90 mmHg) on admission were collected. Prehospital comorbidities included congestive heart failure, smoking, end-stage renal disease, prior CVA, diabetes, myocardial infarction, peripheral arterial disease, hypertension, and obesity. The injury profile included the injury severity score (ISS), Glasgow coma scale, abbreviated injury scale for body region, and any associated skull-base, mandible, cervical spine, thoracic spine, or lumbar spine fracture, along with cervical abrasion (neck seat belt sign). Other hospital outcomes evaluated included total hospital length of stay (LOS), intensive care unit (ICU) LOS, and ventilator days. All variables were coded as present or absent. All missing data points were not imputed but treated as missing data.

Descriptive statistics were performed for all variables. A Student's t-test was used to compare continuous variables and chi-square was used to compare categorical variables for bivariate analysis. Categorical data were reported as percentages, and continuous data were reported as medians with interquartile range or means with standard deviation.

The magnitude of the association between predictor variables and BCVI, as well as CVA in patients with BCVI, were measured using a univariable logistic regression model. Predictor variables were chosen based on risk factors available in the NTDB that have been shown to increase risk for BCVI and CVA.^{4,9,11} Covariables with statistical significance ($P \leq .20$) were included in a multivariable logistic regression model. Covariates were controlled for using a hierarchical logistic regression model and risk of outcomes was reported with an odds ratio (OR) and 95% confidence intervals (CI). All P values were 2-sided, with a statistical significance level of < .05. All analyses were performed with IBM SPSS Statistics for Windows (Version 24, IBM Corp., Armonk, NY).

Results

Patient demographics

Out of 4,932,645 blunt trauma admissions in the study period, more than half the patients were in the pre-eDC group (56.2% vs 43.8%). The post-eDC group was older (mean, 47.5 vs 43.5 years, $P < .001$) with a lower mean ISS (9.4 vs 10.8, $P < .001$). They had a higher prevalence of prior CVA (2.4% vs 1.9%, $P < .001$), smoking (14.6% vs 6.1%, $P < .001$) and hypertension (30.5% vs 21.3%, $P < .001$). The post-eDC group had a lower incidence of skull-base (5.1% vs 6.3%, $P < .001$), mandible (2.1% vs 2.3%, $P < .001$), and cervical spine fractures (9.4% vs 10.3%, $P < .001$; [Table I](#)).

Blunt cerebrovascular injury

There were 5,364 BCVIs in the pre-eDC group (0.19%) and 4,819 BCVIs in the post-eDC group (0.22%; $P < .001$) translating to a 16% increase in the incidence of BCVI ([Table I](#)). In a multivariable analysis of all blunt trauma admissions, post-eDC was associated with a 26% increased ability to detect BCVI (OR 1.26, 95% CI 1.21–1.31, $P < .001$). The strongest associated injuries with BCVI were skull-base fracture (OR 3.61, 95% CI 3.46–3.88, $P < .001$) and mandible fracture (OR 2.84, 95% CI 2.67–3.02, $P < .001$). Cervical abrasion was not associated with BCVI ($P = .82$; [Table II](#)).

Cerebrovascular accident in patients with blunt cerebrovascular injury

The rate of stroke in BCVI patients was significantly higher in the post-eDC group (9.2% vs 5.5%, $P < .001$; [Table I](#)). In a multivariable analysis of all BCVIs, patients in the post-eDC group were associated with a nearly 3-fold increased risk for stroke, compared to patients in the pre-eDC group (OR 2.73, CI 2.29–3.25, $P < .001$). The strongest associated injury for in-hospital CVA in patients with BCVI was a mandible fracture (OR 3.32, 95% CI 2.66–4.14, $P < .001$). Cervical abrasion was not associated with stroke in patients with BCVI ($P = .06$; [Table III](#)).

Mortality in patients with blunt cerebrovascular injury

The overall mortality rate in patients with BCVI was 16.1%. The mortality rate was similar in patients in the pre-eDC and post-eDC groups (16.8% vs 15.8%, $P = .10$). In a multivariable analysis for risk of mortality in all patients with BCVI, the strongest independent predictors include ISS ≥ 25 (OR 3.67, 95% CI 3.22–4.18, $P < .001$) and hypotension on arrival (OR 3.14, 95% CI 2.71–3.64, $P < .001$; [Table IV](#)). Despite increased detection of BCVIs in the post-eDC era, this group was not associated with decreased risk for mortality ($P = .18$).

Carotid artery and vertebral artery injury

CAI and VAI were separately analyzed using the same multivariable logistic regression model used for our analysis of all BCVI patients. The strongest associated injuries for CAI in all blunt trauma admissions were skull-base (OR 3.62, 95% CI 3.46–3.78, $P < .001$) and mandible fractures (OR 2.85, 95% CI 2.67–3.03, $P < .001$; [Table V](#)). The strongest associated injury for in-hospital CVA in patients with CAI was a mandible fracture (OR 3.26, 95% CI 2.61–4.08, $p < .001$; [Table VI](#)).

The strongest associated injuries for VAI in all blunt trauma admissions were cervical spine (OR 19.98, 95% CI 15.85–25.19, $P < .001$) and mandible fractures (OR 2.13, 95% CI 1.13–4.00, $P < .05$; [Table VII](#)). The strongest associated injury for CVA in patients with VAI was a cervical spine fracture (OR 21.93, 95% CI 13.76–34.95, $P < .001$; [Table VIII](#)). Overall, the association with stroke was significantly higher in patients with CAI as compared to those with VAI (OR 4.52, CI 3.12–5.57, $P < .001$).

Discussion

Our study focused on the impact that the eDC has had on the national detection of BCVI since its introduction in 2012.⁹ We were able to demonstrate that the detection of BCVI increased in the post-eDC era by 16%. Interestingly, there was a paradoxical increase in the stroke-rate of patients with BCVI in the post-eDC era. We confirmed previously described risk factors for both BCVI and for CVA in patients with BCVI. The injury most strongly associated with BCVI was a skull-base fracture.

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