Blunt cerebrovascular injury

Blunt cerebrovascular injury is a non-penetrating injury to the carotid and/or vertebral artery that may cause stroke in trauma patients.

Epidemiology

Blunt cerebrovascular injury epidemiology.

Classification

Denver grading scale.

Risk Factors

● high energy transfer mechanism associated with:

○ displaced midface fracture: Le Fort fracture type II or III

○ basilar skull fracture involving carotid canal

● TBI consistent with DAI and GCS < 6

● cervical vertebral body or transverse foramen fracture, subluxation, or ligamentous injury at any level

● any fracture involving C1–3

● near hanging with anoxic brain injury

● clothesline-type injury or seat belt abrasion with significant cervical swelling, pain, or mental status changes

Diagnosis

A new screening model that incorporates an easy-to-use nomogram to quantify the risk of BCVI and assist clinicians in identifying patients who warrant additional evaluation was established by Shibahashi et al. [1)]

Treatment

Blunt cerebrovascular injury treatment.

Diagnosis

The diagnosis of blunt cerebrovascular injuries (BCVI) has improved with widespread adaptation of screening protocols and more accurate multi-detector computed tomography (MDCT-A) angiography. The population at risk and for whom screening is indicated is still controversial. To help determine which blunt trauma patients would best benefit from screening we performed a comprehensive analysis of risk factors associated with BCVI. METHODS: All patients with BCVI from June 12, 2000 (the date at which our institution began screening for these injuries) to June 30, 2009 were identified by the primary author (JDB) and recorded in a prospective database. Associated injuries were identified retrospectively by International Classification of Diseases, Ninth Revision (ICD-9) code and compared with similar patients without BCVI. Demographic information was also compared from data obtained from the trauma registry. Univariate analyses exploring associations between individual risk factors and BCVI were performed using Fisher's exact test for dichotomous variables and Student's t test for continuous variables. Additionally, relative risk (RR) was calculated for dichotomous variables to describe the strength of the relationship between the categorical risk factors and BCVI. Multivariate logistic regression models for BCVI, BCAI (blunt internal carotid artery injury), and BVAI (blunt vertebral artery injury) were developed to explore the relative contributions of the various risk factors. RESULTS: One hundred two patients with BCVI were identified out of 9935 blunt trauma patients admitted during this time period (1.03% incidence). Fifty-nine patients (0.59% incidence) had a BVAI and 43 patients (0.43% incidence) had a BCAI. Univariate analysis found cervical spine fracture (CSI) (RR = 10.4), basilar skull fracture (RR = 3.60), and mandible fracture (RR = 2.51) to be most predictive of the presence of BCVI ($P < .005$). Independent predictors of BCVI on multivariate logistic regression were CSI (OR = 7.46), mandible fracture (OR = 2.59), basilar skull fracture (OR = 1.76), injury severity score (ISS) (OR = 1.05), and emergency department Glasgow Coma Scale (ED-GCS) (OR = 0.93): all $P < .05$.

Blunt trauma patients with a high risk mechanism and a low GCS, high injury severity score, mandible fracture, basilar skull fracture, or cervical spine injury are at high risk for BCVI should be screened with MDCT-A [2)].

Computed tomography angiography (CTA) is frequently used to examine patients for blunt cerebrovascular injury (BCVI) after cranial trauma, but the pediatric population at risk for BCVI is poorly defined. Although CTA is effective for BCVI screening in adults, the increased lifetime risk for malignant tumors associated with this screening modality warrants efforts to reduce its use in children.

The objective was to evaluate the incidence of BCVI diagnosed by CTA in a pediatric patient cohort and to create a prediction model to identify children at high risk for BCVI. METHODS Demographic, clinical, and radiographic data were collected retrospectively for pediatric patients who underwent CTA during examination for traumatic cranial injury from 2003 through 2013. The primary outcome was injury to the carotid or vertebral artery diagnosed by CTA. RESULTS The authors identified 234 patients (mean age 8.3 years, range 0.04-17 years, 150 [64%] boys) who underwent CTA screening for BCVI. Of these, 24 (10.3%) had a focal neurological deficit, and 153 (65.4%) had intracranial

hemorrhage on a head CTA. Thirty-seven BCVIs were observed in 36 patients (15.4%), and 16 patients (6.8%) died. Multivariate regression analysis identified fracture through the carotid canal, petrous temporal bone fracture, Glasgow Coma Scale (GCS) score of < 8, focal neurological deficit, and stroke on initial CT scan as independent risk factors for BCVI. A prediction model for identifying children at high risk for BCVI was created. A score of ≤ 2 yielded a 7.9% probability of BCVI and a score of ≥ 3 a risk of 39.3% for BCVI.

For cranial trauma in children, fracture of the petrous temporal bone or through the carotid canal, focal neurological deficit, stroke, and a GCS score of < 8 are independent risk factors for BCVI [3].

Pediatric blunt cerebrovascular injury (BCVI) lacks accepted treatment algorithms, and postinjury outcomes are ill defined.

Among 645 pediatric patients evaluated with computed tomography angiography for BCVI, 57 vascular injuries (82% carotid artery, 18% vertebral artery) were diagnosed in 52 patients. Grade I (58%) and II (23%) injuries accounted for most lesions. Severe intracranial or intra-abdominal hemorrhage precluded antithrombotic therapy in 10 patients. Among the remaining patients, primary therapy was an antiplatelet agent in 14 (33%), anticoagulation in 8 (19%), endovascular intervention in 3 (7%), open surgery in 1 (2%), and no treatment in 16 (38%). Among 27 eligible grade I injuries, 16 (59%) were not treated, and the choice to not treat varied significantly among centers (P < .001). There were no complications from medical management. Glasgow Coma Scale (GCS) score <8 and increasing injury grade were predictors of injury progression (P = .001 and .004, respectively). Poor GCS score (P = .02), increasing injury grade (P = .03), and concomitant intracranial injury (P = .02) correlated with increased risk of mortality. Treatment modality did not correlate with progression of vascular injury or mortality.

Treatment of BCVI with antiplatelet or anticoagulant therapy is safe and may confer modest benefit. Nonmodifiable factors, including presenting GCS score, vascular injury grade, and additional intracranial injury, remain the most important predictors of poor outcome [4].

1)
Shibahashi K, Hoda H, Ishida T, Motoshima T, Sugiyama K, Hamabe Y. Derivation and validation of a quantitative screening model for blunt cerebrovascular injury. J Neurosurg. 2021 Feb 12:1-10. doi: 10.3171/2020.8.JNS202589. Epub ahead of print. PMID: 33578388.
2)
Berne JD, Cook A, Rowe SA, Norwood SH. A multivariate logistic regression analysis of risk factors for blunt cerebrovascular injury. J Vasc Surg. 2010 Jan;51(1):57-64. doi: 10.1016/j.jvs.2009.08.071. PubMed PMID: 19954917.
3)
Ravindra VM, Riva-Cambrin J, Sivakumar W, Metzger RR, Bollo RJ. Risk factors for traumatic blunt cerebrovascular injury diagnosed by computed tomography angiography in the pediatric population: a retrospective cohort study. J Neurosurg Pediatr. 2015 Jun;15(6):599-606. doi: 10.3171/2014.11.PEDS14397. PubMed PMID: 25745952.
4)
Dewan MC, Ravindra VM, Gannon S, Prather CT, Yang GL, Jordan LC, Limbrick D, Jea A, Riva-Cambrin J, Naftel RP. Treatment Practices and Outcomes After Blunt Cerebrovascular Injury in Children. Neurosurgery. 2016 Dec;79(6):872-878. PubMed PMID: 27465848; PubMed Central PMCID: PMC5115974.