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ORIGINAL SCIENTIFIC REPORT

Blunt Cerebrovascular Artery Injury and Stroke in Severely Injured Patients: An International Multicenter Analysis

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Abstract

Introduction Blunt cerebrovascular injury (BCVI) is considered to be a rare entity in patients with high-energy trauma and is a potentially preventable cause of secondary brain damage. If it occurs, it may be fatal or associated with poor outcomes related to devastating complications. We hypothesized that analyses of epidemiology and concomitant injuries may predict the development of BCVI and associated complications.

Methods The TraumaRegister DGU® (TR-DGU), a prospectively maintained database, was used for retrospective data analysis (01/2009–12/2015). Inclusion criteria: adult trauma patients (≥16 years) with severe injuries (ISS ≥ 16 points) with and without BCVI. Subgroups: carotid artery injury (CAI) and vertebral artery injury (VAI). The degree of vascular injury was classified according to the Abbreviated Injury Scale values. Demographic, injury, therapy and outcome characteristic data (length of stay, stroke, multiple organ failure and mortality) were collected and analyzed for each patient with SPSS statistics (Version 23, IBM Inc., Armonk, NY).

Results Out of 76,480 individuals, a total of 786 patients with BCVI (1%) were identified. The 435 CAI patients included 263 dissections, 78 pseudoaneurysms and 94 bilateral injuries. The 383 VAI patients presented with 198 dissections, 43 pseudoaneurysms, 122 thrombotic occlusions and 20 bilateral injuries. The risk for stroke was excessive in BCVI patients versus controls (11.5 vs. 1.1%, $p < 0.001$) and increased with vascular injury severity, up to 24.1% in CAI patients and 30.0% in VAI patients. We confirmed that cervical spine injuries were a major BCVI predictor (OR 6.46, $p < 0.001$, 95% CI 5.34–7.81); furthermore, high-energy mechanisms (OR 1.79), facial fractures (OR 1.56) and general injury severity (OR 1.05) were identified as independent predictors. Basilar skull fractures (BSF) were found with comparable frequency ($p = 0.63$) in both groups, and the predictive value was found to be insignificant (OR 1.1, $p = 0.36$, 95% CI 0.89–1.37). Age ≥ 60 years was associated with a decreased risk for BCVI (OR 0.54, $p < 0.001$, 95% CI 0.45–0.65); however, in BCVI patients over 60 years of age, mortality was excessive (OR 4.33, $p < 0.001$, 95% CI 2.40–7.80). Even after adjusting for head injuries, BCVI-associated stroke remained a significant risk factor for mortality (OR 2.52, $p < 0.001$, 95% CI 1.13–5.62).

Conclusion Our data validated cervical spine injuries as a major predictor, but the predictive value of BSF must be scrutinized. Patient age appears to play a contradictory role in BCVI risk and BCVI-associated mortality. Predicting which patients will develop BCVI remains an ongoing challenge, especially since many patients do not present with concomitant injuries of the head or spine and therefore might not be captured by standard screening criteria.

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Abbreviations

ACCP	American College of Chest Physicians
AIS	Abbreviated Injury Scale
BCVI	Blunt cerebrovascular injury
BSF	Basilar skull fracture
CAI	Carotid artery injury
CI	Confidence interval
DGU	Deutsche Gesellschaft für Unfallchirurgie/ German Trauma Society
ER	Emergency room
GCS	Glasgow Coma Score
GOS	Glasgow Outcome Score
ICU	Intensive care unit
ISS	Injury Severity Score
MCA	Motorcycle accident
MOF	Multiple organ failure
mmHg	Unit millimeters of mercury
MVC	Motor vehicle collision
OR	Odds ratio
PVS	Persistent vegetative state
SBP	Systolic blood pressure
SCCM	Society of Critical Care Medicine
SD	Standard deviation
SOFA	Sequential organ failure assessment score
TR-DGU	TraumaRegister DGU®
VAI	Vertebral artery injury

Introduction

Blunt cerebrovascular injuries (BCVIs) were considered rare entities (0.08–1.55%), even in individuals with multiple injuries [1–4]. However, more recent publications reported higher rates for BCVIs (2.7–4–6%) and suggested that these injuries might have been underappreciated in the past [5, 6], potentially due to inadequate imaging techniques or the absence of validated and established screening guidelines [1–3, 7, 8].

Cervical arteries are prone to injury, especially in high-energy injuries, due to their unique anatomic exposure. The potential for devastating complications related to permanent neurologic deficits is well documented in the literature. Therefore, more liberal screening indications and early therapy are recommended [9–13].

A consensus opinion was established that a high index of suspicion, prompt detection and early initiation of treatment remain crucial elements in the management of BCVI to prevent stroke and associated neurologic sequelae [3, 14, 15].

However, concerns about the definition of standard screening criteria and optimal management remain. Bruns et al. studied the database of the *R Adams Cowley Shock Trauma Center* in Baltimore and identified a relevant number of patients with BCVI after blunt multisystem trauma that would not be screened for BCVI when using standard screening guidelines. The authors reported that 30% of the patient cohort with BCVI had no radiographic or clinical risk factors and concluded that current BCVI screening guidelines might lead to missed BCVI and stroke risk [16].

Burlew et al. suggested that screening criteria be expanded to include mandible fractures, complex skull fractures, traumatic brain injury (TBI) with thoracic injuries, scalp degloving and thoracic vascular injuries.

Franz et al. performed a systematic review of the current BCVI literature. The meta-analysis encompassed 418 BCVI patients and 22,568 non-BCVI patients and identified cervical spine injuries as a major risk factor (OR 5.45, 95% CI 2.24–13.27; $p < 0.0001$). A recent study evaluated a cohort of 564 patients diagnosed with BCVI between 1985 and 2015 and reported an increasing incidence from 0.33 to 2% over time as well as a decreasing risk of BCVI-related stroke (14%) within the 30-year study period [17].

The data involved in the previous study were derived from North America; the purpose of the current study was to gather epidemiologic, injury, therapy and outcome data from TraumaRegister DGU® (TR-DGU) and to answer the following questions:

1. What is the overall incidence of BCVI and associated complications (BCVI-related stroke, mortality, MOF, LOS) in an international trauma database?
2. Do severely injured adult patients exhibit specific indicator injuries (e.g., cervical spine/facial/basilar skull fractures) and/or other risk factors that should be implemented in the current BCVI screening guidelines?
3. What is the impact of patient age? Preexisting vessel degeneration in the older population (e.g., arteriosclerotic plaques/stenosis) might increase the risk for both BCVI- and BCVI-associated mortality.
4. What is the outcome of BCVI and non-BCVI patients as measured by the Glasgow Outcome Scale (GOS)?
5. What are the major risk factors for mortality in the context of multiple injuries? Since many BCVI patients exhibit multiple severe traumas, regression analysis will elucidate the impact of BCVI-associated

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stroke, advanced age (≥60 years) and general injury severity after adjusting for head injuries.

Patients and methods

Inclusion and exclusion criteria

All adult patients (age ≥ 16 years) with severe injuries (ISS ≥ 16) with admission to a participating trauma center in a German-speaking country (Germany, Austria, Switzerland) between January 2009 and December 2015 were included in this study. Patients transferred to another center within 48 h after admission were excluded due to missing outcome data (7.6% of the total population). However, all cases transferred in (12.0%) were included to prevent bias in prevalence rates.

TraumaRegister DGU® and data acquisition

The TraumaRegister DGU® of the German Trauma Society (Deutsche Gesellschaft für Unfallchirurgie, DGU) was founded in 1993 [18]. The aim of this multicenter database is to pseudonymize and standardize the documentation of severely injured patients. Data are collected prospectively in four consecutive time phases from the site of the accident until discharge from hospital: (A) pre-hospital phase, (B) emergency room and initial surgery, (C) intensive care unit and (D) discharge. The documentation includes detailed information on demographics, injury pattern, comorbidities, pre- and in-hospital management, course on the intensive care unit, and relevant laboratory findings, including data on transfusions and outcomes of each individual. The inclusion criterion consists of admission via the emergency room with subsequent ICU/ICM care or admission to the hospital with vital signs and death before admission to the ICU. The infrastructure for documentation, data management, and data analysis is provided by the Academy for Trauma Surgery (AUC—Akademie der Unfallchirurgie GmbH), a company affiliated with the German Trauma Society. Scientific leadership is provided by the Committee on Emergency Medicine, Intensive Care and Trauma Management (Sektion NIS) of the German Trauma Society.

The participating hospitals enter their pseudonymized data into a central database via a web-based application. Scientific data analysis is approved according to a peer review procedure established by Sektion NIS. The participating hospitals are primarily located in Germany (90%), but an increasing number of hospitals in other countries contribute data as well (Austria, Switzerland, Belgium, China, Finland, Luxemburg, Slovenia, The Netherlands and the United Arab Emirates). Currently, approximately 25,000 cases or more than 600 hospitals are entered into the database per year. Participation in TraumaRegister DGU® is voluntary. For hospitals associated with the TraumaNetzwerk DGU®, however, the entry of at least a basis data set is obligatory for reasons of quality assurance. The present study is in line with the publication guidelines of TraumaRegister DGU® and is registered as TR-DGU project ID 2012-052.

Definitions

Injury severity

Since 2009, coding has followed a uniform protocol and data management has been previously described [18]. All injuries were coded according to the *Abbreviated Injury Scale* (AIS Version 2005/Update 2008, Association for the Advancement of Automotive Medicine, Barrington, IL, USA). The severity of injuries was documented as: 1 (minor), 2 (moderate), 3 (severe, not life-threatening), 4 (serious, life-threatening), 5 (critical, survival uncertain), 6 (maximum, currently untreatable). The *Injury Severity Score* (ISS) was subsequently calculated from AIS values. Severe trauma was defined as ISS ≥ 16 points [19, 20].

BCVI

Identification according to AIS codes; carotid artery injury (CAI) codes: *3202xx and 3204xx,* and vertebral artery injury (VAI) codes: *3210xx*. As a noninvasive, cost-effective and widely available modality, computed tomography angiogram (CTA) was applied for primary BCVI screening.

Stroke

Stroke was diagnosed according to the current World Health Organization (WHO) definition. It includes "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 h or leading to death, with no apparent cause other than that of vascular origin" [21]. An acute post-traumatic infarction coded as AIS 140676.3 was considered a stroke in this study. Furthermore, the registry captures strokes in the subsequent hospital course as one of four different thromboembolic events in the acute care phase. This documentation was available in the standard documentation, which is performed in the majority of trauma centers that manage BCVI patients (69.7%). No imputation or missing data treatment was performed. A mismatch analysis excluded cases with duplicate documentation.

Multiple organ failure (MOF)

Organ failure was defined as 3 or 4 points in the SOFA score [22]; MOF was present in case of two or more failing organs [23]. These data were available only in patients with standard documentation.

Glasgow Outcome Scale (GOS)

Neurologic outcome was based on the GOS [24–26] and included the following categories: (1) good recovery (resumption of normal life), (2) moderate disability (can work in a sheltered setting), (3) severe disability (dependent on daily support), (4) persistent vegetative state (minimal responsiveness) and (5) death.

Mortality

Mortality was defined as in-hospital death from any cause.

Statistical analysis

Categorical data were presented as frequencies and percentages. Metric variables were reported as the means and standard deviation (SD). In case of a skewed distribution, the median is also provided. The Chi-square test was used for the comparison of categorical variables, and the Mann–Whitney *U* test was applied for metric variables. Logistic regression analysis was performed to elucidate the possible impact of various risk factors on the development of BCVI- and BCVI-associated mortality. Regression model performance measures are provided as area under the curve (AUC). The results are considered statistically significant if $p < 0.05$. The analysis was performed with SPSS for Windows (Version 23, IBM Inc., NY, USA).

Results

During the 7-year study period, 76,480 patients fulfilled the inclusion criteria. Of these, a total of 786 patients (1.0%) had sustained a BCVI (Fig. 1). Consequently, the control group included 75,694 non-BCVI patients. In the BCVI group, 435 patients were diagnosed with CAI (0.6%), and in 383 individuals a VAI was observed (0.5%). Thirty-two cases had a combined injury of the carotid and vertebral artery.

Of the 786 patients with BCVI, the mean age was 46 years (SD 19) and the mean ISS was 35 points (SD 15). Sixty-nine percent ($n = 543$) were men. The control group was also predominantly male (71.2%, $p = 0.28$).

Patients with BCVI were more often injured by high-energy mechanisms (Table 1), especially in motor vehicle collisions (38.1%, $p \leq 0.001$). High and low falls were underrepresented in the BCVI group ($p \leq 0.001$).

Patients with BCVI often presented with severe facial and spinal injuries ($p \leq 0.001$). We did not find a statistically significant difference in the rate of head injuries and basilar skull fractures (Non-BCVI: 16.7% vs. BCVI: 16.0%, $p = 0.63$).

Additionally, chest injuries were distributed equally (55.4 vs. 55.3%, $p = 0.99$). However, BCVI patients suffered from abdominal, pelvic and extremity injuries less often (Table 2). An associated penetrating injury was found in 6.3% of cases ($n = 47$). Further details of the injury distribution are described in Table 2. In terms of injury scoring, BCVI patients were more likely to suffer more severe injuries, indicated by ISS scores of 35 versus 27 points, $p \leq 0.001$. Furthermore, BCVI patients were more often in shock both at the scene and during ER admission. Despite a comparable rate of head injuries and basilar skull fractures, BCVI patients presented with an inferior neurologic status at ER admission (Table 3). Nearly half of all primary admitted patients with BCVI (46.4%; $n = 296$) presented with a primary loss of consciousness (LOC, defined as a GCS of ≤8 points at the scene). This was observed in only 28.0% of cases without BCVI ($p \leq 0.001$). Consequently, 62.5% of all BCVI patients ($n = 423$) were intubated in the pre-hospital setting ($p \leq 0.001$). Additional detailed patient characteristics are tabulated in Table 3. BCVIs were associated with prolonged ventilation and a longer ICU stay ($p \leq 0.001$); however, the total hospital LOS was not significantly prolonged.

Stroke was observed more often (Fig. 2) in BCVI patients (11.5%; 58 of 503) when compared to non-BCVI cases (1.1%). This difference was found to be statistically significant ($p \leq 0.001$). The prevalence of stroke by vascular injury grade is listed in Fig. 3. Furthermore, more patients with BCVI developed multiple organ failure compared to controls (47.3 vs. 32.0%, $p \leq 0.001$). The majority of strokes developed despite medial prophylaxis: 30 BCVI-associated strokes developed despite medical prophylaxis with heparin, and 10 patients sustained an acute post-traumatic cerebral infarction and were therefore not yet under anticoagulation or antiplatelet therapy.

In terms of neurologic outcome, non-BCVI patients achieved a favorable outcome with good recovery more often (43.9%, $n = 32.109$), when compared to patients with BCVI (24.4%, $n = 185$). In fact, the largest GOS group of BCVI patients died (26.6%, $n = 202$), whereas the largest GOS group of non-BCVI patients experienced good recovery (43.9%, $n = 32{,}109$). Moderate disabilities were found in both groups at a comparable frequency (23.9 vs. 23.5% in BCVI patients and non-BCVI patients, respectively). However, more BCVI patients experienced

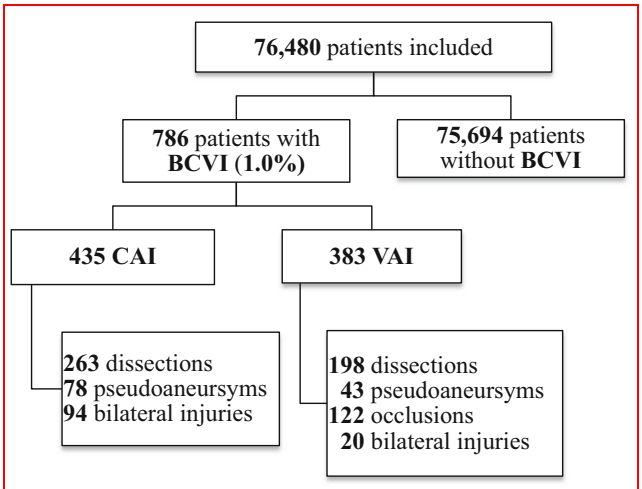

Fig. 1 Flow diagram of study population

poor outcomes with severe disabilities compared to non-BCVI patients (21.0 vs. 10.9%, respectively) or remained in a persistent vegetative state (PVS), both $p \leq 0.001$ (Figs. 4, 5). The median hospital LOS was 23 days for BCVI patients and 21 days for patients without BCVI; this difference was found to be insignificant.

A logistic regression analysis model (Table 4) indicated that the following variables were associated with an increased risk of BCVI in severely injured patients: cervical spine injury (OR 6.62, 95% CI 5.49–7.97, $p \leq 0.001$), road traffic collision (OR 1.79, 95% CI 1.51–2.11, $p \leq 0.001$) and ISS (OR 1.03, 95% CI 1.02–1.03, $p \leq 0.001$). Neither basilar skull fractures nor head injury was significantly associated with BCVI. Advanced age (≥60 years) was found to be an inverse predictor of BCVI (OR 0.54, 95% CI 0.45–0.65; $p < 0.001$). When independent predictors for mortality were analyzed in BCVI patients after adjusting for head injuries, BCVI-associated stroke (OR 2.52, 95% CI 1.13–5.62, $p = 0.024$) and advanced age (≥60 years) were both found to be robust predictors. General injury severity measured by ISS-predicted mortality with an OR of 1.05 per point, which was also significant (Table 5).

Discussion

The definition of accurate screening criteria and an optimal management protocol continues to be an ongoing challenge in the care of patients with multiple injuries and blunt cerebrovascular injury. A primary objective is the identification and treatment of individuals at risk for BCVI prior to the onset of ischemic cellular brain damage and the symptomatic manifestation of devastating complications. A variable onset and wide range of neurologic symptoms, including asymptomatic BCVI, contribute to the clinical dilemma because in multisystem trauma, the diagnostic and therapeutic prioritization of life-threatening injuries remains imperative [1, 2, 7, 10, 12, 27–29].

Blunt injuries to the carotid and vertebral artery (blunt cerebrovascular injury [BCVI]) were thought to be rare; however, the true incidence remains unknown. The current literature reports a variable incidence for BCVI and a stroke rate of 30–50% in untreated patients and a mortality rate up to 80% [2–4, 7, 9, 27, 30–34]. Recognizing the relative infrequence and limited experience in most institutions, we followed a multicentric approach to contribute data to the issue from outside North America.

We report an overall incidence of 1% for blunt cerebrovascular injury for patients managed in trauma centers (TR-DGU®) in Germany, Austria and Switzerland.

To our knowledge, the current study encompasses one of the largest BCVI series ever reported; a recently published systematic review involved 418 cases [5].

In the current study, 435 patients (0.6%) suffered from carotid injury and 383 patients were diagnosed with vertebral artery injury (0.5%). In comparison, Miller et al. [33] suggested an incidence of 0.50% for CAI and 0.40% for VAI. Fabian and colleagues from Memphis reported an overall incidence of 0.69% among victims of motor vehicle

Table 1 Injury mechanisms

	Non-BCVI	BCVI	*p*
Motor vehicle collision, MVC (*n*, %)	16,300 (22.2%)	274 (35.7%)	≤0.001
Motorcycle accident, MCA (*n*, %)	9257 (12.6%)	98 (12.8%)	0.89
Bicycle (*n*, %)	5912 (8.0%)	77 (10.0%)	0.045
Pedestrian (*n*, %)	5144 (7.0%)	47 (6.1%)	0.34
Low fall <3 m (*n*, %)	15,824 (21.5%)	84 (10.9%)	≤0.001
High fall ≥3 m (*n*, %)	13,263 (18.0%)	88 (11.5%)	≤0.001
Penetrating injury (*n*, %)	2276 (3.9%)	47 (6.3%)	≤0.001

Table 2 Anatomic patterns of injury

Head injury (AIS ≥ 3)	50.2% (38,030)	48.3% (380)	0.29
Basilar skull fracture	16.7% (12,627)	16.0% (126)	0.63
Facial injury (AIS ≥ 3)	6.5% (4919)	10.6% (83)	≤0.001
Cervical spine (AIS ≥ 3)	4.4% (3366)	26.3% (207)	≤0.001
Chest (AIS ≥ 3)	55.4% (41,905)	55.3% (435)	0.99
Abdomen (AIS ≥ 3)	15.5% (11,752)	15.3% (120)	0.84
Extremity injury (AIS ≥ 3)	30.9% (23,389)	28.0% (220)	0.079

Table 3 Patient characteristics

	Non-BCVI	BCVI	*p*
Male gender % (*n*)	71.2% (53,664)	69.4% (543)	0.28
Age: mean (SD), years	53 (21)	46 (19)	≤0.001
ISS: mean (SD), points	27 (11)	35 (15)	≤0.001
New ISS: mean (SD), points	33 (14)	43 (16)	≤0.001
Glasgow Coma Scale: mean (SD), points	11.2 (4.6)	9.0 (5.1)	≤0.001
Glasgow Coma Scale: ≤8 points, % (*n*)	28.7% (17,399)	46.4% (296)	≤0.001
Shock at scene (SBP ≤ 90 mmHg), % (*n*)	15.2% (8828)	27.7% (165)	≤0.001
Shock at ER admission, % (*n*)[†]	13.1% (8899)	20.8% (149)	≤0.001
Intubation at scene, % (*n*)[†]	39.9% (25,810)	62.5% (423)	≤0.001
Immediate head/neck CT	90.1% (67,414)	92.5% (718)	0.024
Immediate whole-body CT	77.8% (58,218)	87.1% (676)	≤0.001
Ventilator days, mean (SD) median[β]	5.1 (9.7) 1	8.2 (11.8) 3	≤0.001
ICU stay, mean (SD) median[β]	9.3 (12.7) 4	13.0 (13.8) 8	≤0.001
Length of stay, mean (SD) median	21 (22) 16	23 (25) 18	0.19
Multiple organ failure (MOF), % (*n*)[β]	32.0% (12,707)	47.3% (235)	≤0.001
Stroke, % (*n*)[β]	1.1% (423)	11.5% (58)	≤0.001
In-hospital mortality	19.0% (13,914)	26.6% (202)	≤0.001

ER emergency room, *SBP* systolic blood pressure

[†]Only primary admitted cases

[β]Available for cases with standard documentation

crashes [27]. Our reported incidence is higher when compared to the incidence reported by Berne et al. [2]. They reported an overall incidence of 0.49% for BCVI in 3480 blunt trauma admissions in Texas. A total of 14 of their patients had a CAI (0.40%) and 3 were diagnosed with a VAI (0.09%). Patients suffered from complications, especially if the diagnosis was delayed ≥48 h, and a catastrophic mortality (80%) was consequently observed [2]. In the 1980s, Davis et al. [1] suggested an incidence as low as 0.08% for blunt carotid artery dissection in blunt trauma victims managed in six centers in the San Diego area. In the late 1990s, Biffl et al. [7] reported an overall incidence of 0.24% in patients ($n = 37$) diagnosed with blunt carotid artery injury, and an incidence of 0.53% for blunt vertebral artery injury ($n = 47$) in blunt trauma admissions during a 3.5-year study period [35]. Drain et al. [36] reported an incidence of 0.49% for vertebral artery injury in 144 screened trauma patients. Stein et al. [3] reported a BCVI incidence of 1.2%, and Miller et al. [33] reported an incidence of 1.03%; both very comparable to our incidence rate. In fact, a recent study confirmed the hypothesis that the rate has been increasing over the past three decades [17].

The leading expertise and aggressive screening protocol of the Denver group might have contributed to the higher BCVI incidence in a more recent study [35]. A key feature of their liberal screening protocol was to include asymptomatic individuals that were considered to be at risk. This strategy is likely to identify more BCVIs, however, complications and costs associated with invasive or time-

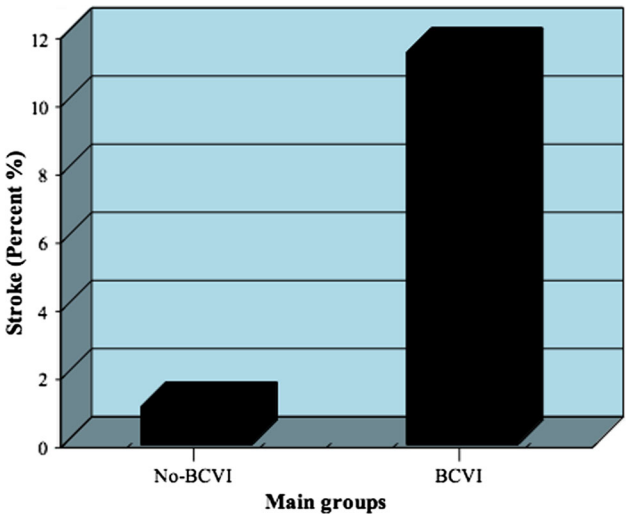

Fig. 2 Prevalence of stroke in patients with and without BCVI ($p \leq 0.001$)

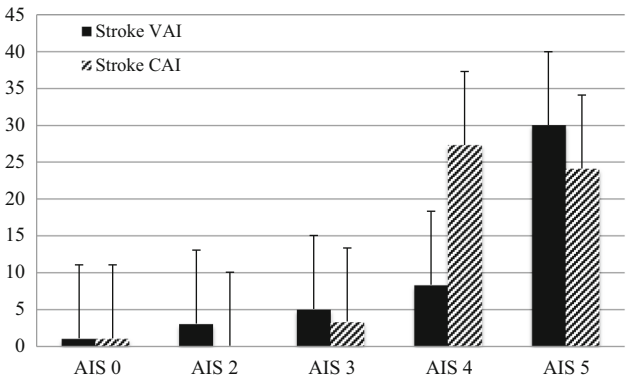

Fig. 3 Prevalence of stroke by severity of CAI and VAI

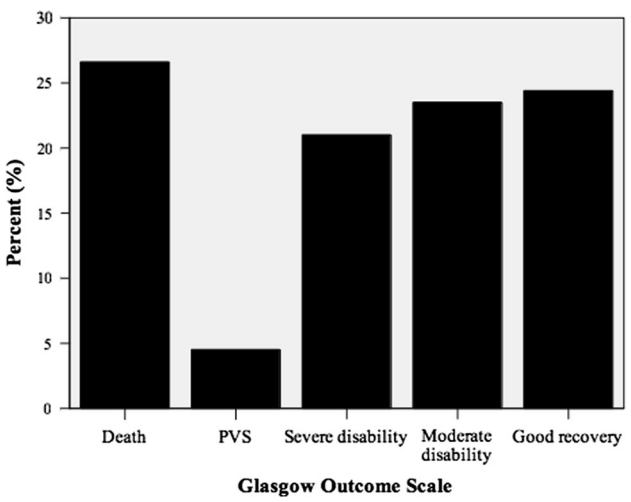

Fig. 4 BCVI patient outcomes at discharge ($n = 786$)

Fig. 5 Non-BCVI patient outcomes at discharge ($n = 75{,}694$)

consuming screening techniques must also be taken into account.

On the other hand, risk factors that were proposed in the early times of BCVI screening in high-risk patients must be validated by current data. Therefore, we intended to review the impact of basilar skull fractures and head trauma. Since beneficial effects were reported for severely injured patients who underwent whole-body CT [37], more liberal contrast-enhanced whole-body CT protocols were integrated in many trauma centers. Therefore, major head trauma (e.g., BSF) is also recognized more frequently in the non-BCVI control group, which challenges the predictive value for BCVI. In our cohort, the liberal use of CT resulted in very high screening rates with 92.5% of patients undergoing a CT of the head and neck region and 87.1% undergoing a whole-body CT scan. In early studies, more than 90% of patients were symptomatic during the diagnostic workup [7, 27]. Recent studies suggest an increasing incidence of BCVI after the implementation of liberal screening policies and improved imaging techniques. Correspondingly, a larger proportion of asymptomatic patients and patients without clinical or radiographic risk factors for BCVI were identified [2, 6, 16, 38–44]. Biffl et al. attempted to define high-risk patients that should undergo angiographic screening to rule out BCVI. These risk factors included neurologic abnormalities (GCS ≤ 6), injuries of the head (e.g., diffuse axonal brain injury, petrous bone fracture), facial injuries (Le Fort II or III fractures) or cervical spine injuries [30]. Burlew et al. [8] described redefined screening criteria in the era of noninvasive diagnosis and recommended the inclusion of any basilar skull fracture.

In the analysis of concomitant injuries, special attention has been paid to basilar skull fractures (BSFs). This type of

Table 4 Logistic regression analysis: independent predictors for BCVI

Risk factor for BCVI	Odds ratio (OR)	95% confidence Interval (CI)	*p* value
Injury Severity Score (*per point*)	1.03	1.02–1.03	≤0.001
Age (≥60 years)	0.54	0.45–0.65	≤0.001
Road traffic collision	1.79	1.51–2.11	≤0.001
Head injury (AIS ≥ 3)	1.097	0.93–1.30	0.284
Basilar skull fracture (BSF)	0.95	0.68–0.95	0.68
Facial injury (AIS ≥ 3)	1.56	1.22–1.99	0.088
Cervical spine injury (AIS ≥ 3)	6.62	5.49–7.97	≤0.001

Total number included in calculation: 70,828; model performance measure for regression analysis: area under the curve (AUC): 0.762 (CI 0.744–0.781)

Table 5 Logistic regression analysis: independent predictors of mortality in BCVI patients

Risk factor for mortality	Odds ratio (OR)	95% confidence interval (CI)	*p* value
Any head injury	1.0	Reference	–
Injury Severity Score (*per point*)	1.05	1.03–1.07	≤0.001
Stroke	2.52	1.13–5.62	0.024
Age (≥60 years)	4.33	2.40–7.80	≤0.001

injury has been considered an indicator injury and a risk factor for BCVI [10, 38, 40]. In the Denver series with 171 BCVIs, a total of 34 patients (20%) suffered from BSF [4]. Cothren et al. [12] published a prospective series with 114 patients with confirmed CAI. Their screening criteria also included basilar skull fracture with carotid canal involvement. Emmett et al. [40] reported that a basilar skull fracture was detected in 16% ($n = 124$) of patients with multiple screening indications and in 16% ($n = 68$) of patients with a single criterion for screening. However, our results indicate that basilar skull fractures are not associated with BCVI (16%) more often when compared to patients without BCVI (16.7%). These findings also fit into the experience of Stein et al. [3], who detected BSFs in 13.6% of VAI patients and 20.4% of CAI patients.

A higher rate of basilar skull fractures (35%, $n = 7$) was described by Eastman et al. [45], but the entire study population included only a total of 20 individuals with CAI. Logistic regression was unable to confirm BSF as a significant risk factor for BCVI.

Carrillo et al. [32] suggested that BCVI cannot be predicted based on clinical parameters or the mechanism of injury. In our data, the largest group of BCVI-affected individuals ($n = 161$, 38.1%) sustained a motor vehicle collision (MVC). This phenomenon was previously described by a number of authors: Biffl et al. described a series of 171 BCVI cases between 1990 and 2001. The Denver group included 157 CAI and 97 VAI patients; 86 patients (50%) were involved in an MVC [30]. The same group also published another series with 249 patients [30], the majority of whom experienced an MVC ($n = 113$, 45%). Theoretically, a hyperextension/hyperflexion mechanism of the neck, potentially combined with forces applied to the cervical region by a seat belt, plays a role in this distribution pattern, since the impact of a high-energy trauma load would also be applied to victims of motorcycle crashes.

Although 20% of BCVI patients do not present with conventional screening criteria, many protocols include "injury mechanism" as a viable screening trigger [8]. In fact, we identified "road traffic collision" to be significantly associated with BCVI.

The current study involves a comparable number of male patients ($n = 543$, 69.4%) in the groups with and without BCVI. Furthermore, other demographic characteristics, including age and injury severity, were comparable to previous reports.

Furthermore, we were able to identify bilateral CAIs in 21.6% of patients ($n = 94/435$) and bilateral VAIs in 5.2% of patients ($n = 20/383$). Biffl and coworkers [4] previously described a higher rate of bilateral injuries in CAI patients ($n = 42$, 38%) compared to VAI patients ($n = 97$, 23%). While the ratio between bilateral CAI and VAI appears to vary, bilateral injuries of the carotid arteries appear to be more common. According to our expectations,

patients with bilateral BCVI also had an increased risk of thromboembolic complications and stroke (Table 3).

Since we observed 10 immediate strokes and 30 strokes that developed under anticoagulation, the therapeutic window and optimal type of medial therapy needs to be evaluated in further studies. Unfortunately, the registry does not provide data on the exact timing and dose of prophylactic or therapeutic heparin application, and no data on endovascular procedures or related outcomes.

However, a recent study by McNutt et al. reported an identical incidence of stroke in patients with isolated BCVI (34.4%) and in those with BCVI complicated by multisystem injuries (65.7%). Furthermore, the authors suggested that accompanying multisystem injuries (TBI, solid organ injury, or spinal cord injury) should not be considered as contraindication for antithrombotic therapy [28]. Unfortunately, we cannot confirm a delayed or less aggressive antithrombotic therapy from the registry data, but a more cautious anticoagulation in patients with TBI has most likely been practiced in many centers, since the management guidelines have been delineated for cases with isolated BCVI. In this light, our data reflect the past treatment reality, associated complications and clinical outcome.

Our data suggests a high mortality rate of 26.6% ($n = 202$) within BCVI patients. Miller et al. [33] reported a mortality rate of 25% in CAI patients ($n = 6$) and 9% in VAI patients ($n = 4$). The mortality rate reported by Stein et al. [3] from Baltimore was only 13%, likely due to a large number of patients with low-grade lesions.

A number of limitations have to be considered when interpreting our data. Unfortunately, the nature of TraumaRegister DGU® imparts several limiting factors. First, TraumaRegister DGU® cannot provide detailed insight into the onset and course of neurologic symptoms, except GCS values. Another limitation is related to the impact of BCVI in the context of multisystem injuries, and the fact that associated head or spinal cord injuries might have confounded neurologic outcomes. Furthermore, diagnostic imaging in patients with polytrauma might be limited in the clinic. Patients in unstable or borderline conditions might be unable to undergo a diagnostic workup or die before a definitive diagnosis of BCVI is confirmed.

After an initial resuscitation and orthopedic fixation, some devices or ventilator equipment might be incompatible with diagnostic modalities. However, we found a very high rate of CT utilization in both cohorts, which reflects a strict adherence to the German S3 guideline for polytrauma and the ATLS® protocol. Another limitation is the use of AIS values to differentiate the severity of BCVI. The Biffl grading system is currently widely appreciated [7, 30, 35, 43, 44, 46]. However, we know that participating centers are familiar with the uniform AIS classification, as it is the single most used system throughout the study period. However, variable intercentre consistency in the grading of BCVI might bias our results. On the other hand, Biffl et al. [4] reported that low-grade lesions (Grade I/II) might change frequently, e.g., Grade II lesions progress to Grade III lesions in 43% of patients, and 61% of patients required a change in the management protocol.

Finally, our hypothesis that advanced age might play a role in BCVI development due to degenerative changes of the cerebrovascular arteries was not confirmed. In fact, this patient subgroup is known to be affected by low-energy mechanisms more often and might therefore sustain BCVI less frequently. On the other hand, the regression analysis corroborated that advanced age is a major independent predictor for mortality after BCVI, reflecting the reduced physiologic reserve in patients of advanced age.

Conclusion

Blunt cerebrovascular injury in severely injured patients is uncommon but often under-recognized and might occur even in the absence of indicator injuries and clinical risk factors. In severely injured adult patients, 1% are affected by BCVI. Our data validated cervical spine injuries as a major predictor, but the predictive value of basilar skull fractures must be scrutinized. Patient age appears to play a contradictory role in BCVI risk and associated mortality. The prediction of BCVI remains an ongoing challenge, especially since many patients feature no concomitant injuries of the head or spine.

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