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Factors Associated with Blunt Cerebrovascular Injury in Patients with Cervical Spine Injury

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Abstract

Blunt cerebrovascular injury (BCVI) is known to be a potentially fatal complication of cervical spine injury (CSI). Methods for screening the appropriate population remain to be elucidated, especially in Japan. This retrospective study was conducted to predict the risk factors relevant to BCVIs. Among 92 patients with CSI transferred to our institution from April 2007 to March 2012, 40 patients (35 men, 5 women) with neurological deficits and/or significant cervical spine fracture including fracture of transversarium, facet, body, lamina, and spinous process, underwent multi-detector computed tomography angiography (MDCTA) and magnetic resonance angiography (MRA), which identified 10 patients with BCVI [2 carotid artery injuries (BCAIs) and 9 vertebral artery injuries (BVAIs); 1 patient suffered both]. Univariate analyses exploring associations between individual risk factors and BCVI and BVAI were performed using Fisher's exact test and Chi-square test for dichotomous variables and the unpaired *t*-test for continuous variables. Multiple logistic regression analyses for BCVI and BVAI were carried out using stepwise methods. On univariate and multivariate analysis, hyperextension injury was significantly associated with BVAI ($p = 0.01$ and $p = 0.02$), and subluxation (dislocation of vertebral body > 5 mm) was a significant predictor of BCVI ($p = 0.04$ and $p = 0.03$) and BVAI ($p = 0.01$ and $p = 0.01$). Prompt evaluation for BCVIs is recommended in CSI patients with hyperextension injury and dislocation of the vertebral body.

Key words: blunt cerebrovascular injury, cervical spine injury, carotid artery, vertebral artery, risk factor

Introduction

Blunt cerebrovascular injury (BCVI) to the cervical vasculature supplying the cerebral circulation (the carotid and vertebral arteries) is known to be a potentially devastating injury in trauma patients. Previously, BCVI was thought to be infrequent, because the majority of BCVI is asymptomatic and invasive conventional angiographic screening has rarely been performed. With the popularization of noninvasive magnetic resonance angiography (MRA)[1–3] and computed tomography angiography (CTA),[4–6] the diagnosis of patients with BCVI has evolved rapidly over the last decade. According to recent reports, BCVI occurs in approximately 1% of all trauma patients[7–9] and is frequently associated with cervical spine injury (CSI).[7,10,11] However, methods for screening the appropriate population remain to be elucidated, especially in Japan, due to the paucity of clinical studies. The purpose of this study was to quantify the risk of BCVI in patients with CSI admitted to a Japanese comprehensive trauma and stroke center with an aggressive screening protocol.

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Materials and Methods

I. Patients

This study was conducted at the International Medical Center at Saitama Medical University, a major regional trauma and stroke center. Between April 2007 and March 2012, 92 patients with CSI including fracture and dislocation were evaluated. On admission, the level of consciousness and neurological status of the patients were determined according to the Glasgow Coma Scale (GCS) and Frankel grade[12] (A, complete motor and sensory deficit; B, complete

motor deficit with some remaining sensory function; C, inefficient motor function; D, useful remaining motor function; and E, no neurological signs or symptoms). Three-dimensional cervical CT scans were obtained in all patients to access the injury pattern of the cervical spine. Additionally, the mechanism (hyperflexion/hyperextension/others) and location (upper/middle/lower cervical) of CSI were classified using the Allen criteria.[13]

Subsequently, 40 patients (35 men, 5 women) with neurological deficits and/or significant cervical spine fracture underwent brain CT, multi-detector computed tomography angiography (MDCTA) and magnetic resonance angiography (MRA) to identify the associated intracranial hemorrhagic/ischemic insults and BCVI. Significant fractures warranting screening for BCVI in this study included subluxation, extension into the foramen transversarium, facet, spinous process, and vertebral body. MDCTA scans were obtained on a Lightspeed VCT (GE Healthcare, Tokyo), and MRA on an Achieva 1.5 T and 3.0 T (PHILIPS, Tokyo). BCVIs were classified using Denver grading[14] (Grade I, irregularity of vessel wall or dissection/intramural hematoma with < 25% luminal stenosis; grade II, intraluminal thrombus, or raised intimal flap is visualized, or dissection/intramural hematoma with > 25% luminal narrowing; grade III, pseudoaneurysms; grade IV, vessel occlusion; grade V, transaction). The remaining 52 patients with minor CSI without neurological deficits were managed conservatively and discharged without neurovascular events.

The ethics review board of Saitama Medical University approved all aspects of this study (No.12-210).

II. Statistics

Data were analyzed using commercially available software (SigmaPlot, SAS JMP 10.0.2, and SAS 9.1.3 SP4 for Windows; SAS Institute, Inc., Cary, North Carolina, USA). Continuous data are expressed as mean ± the standard deviation. Univariate analyses exploring associations between individual risk factors and BCVI and blunt vertebral artery injuries (BVAI) were performed using Fisher's exact test and Chi-square test for dichotomous variables and the unpaired *t*-test for continuous variables. Multiple logistic regression analyses were carried out using stepwise methods, i.e., a combination of forward and backward selection of the independent variables to choose the variables that are significantly associated with BCVI and BVAI. Multiple logistic regression analyses were performed repeatedly with inclusion and elimination of the independent variables one by one. The likelihood-ratio test was used to determine inclusion or elimination of a specific variable. The criteria for inclusion and elimination of the variables were $P < 0.25$ and $P > 0.25$, respectively.

Results

I. Incidence and characteristics of BCVI

Ten of 40 patients (25%) evaluated with MRA and MDCTA in this protocol had injury to the cervical vessels (BCVI patients). All patients were men with a mean age of 51.5 ± 5.8 years (range, 31–77). The causes of injury included falls in 6 patients, traffic accidents in 3 patients, and unknown causes in 1 patient. The mechanisms included hyperextension in 6 patients, hyperflexion in 3 patients, and vertical compression in 1 patient. On neurological examination, 5 patients showed disturbance of consciousness (coma or semicoma, GCS 3-8 in 2; confusion, GCS 14 in 3), and Frankel grades included A in 5, D in 3, and E in 2. The most common level of CSI associated with BCVI was C4/5 (4 patients), followed by C5/6 (3) and C6/7 (3). There was no BCVI at upper CSI (C1-3). Facet fracture was evident in 8 patients and transversarium fracture in 7. Dislocation of the vertebral body (> 5 mm) was evident in 6 patients. Associated intracranial traumatic hemorrhages were identified in 4 (subarachnoid hemorrhage in 3 and epidural hematoma in 1), and cerebral infarction was noted in 2 patients.

There were two carotid artery injuries (BCAI) and nine vertebral artery injuries (BVAI). One patient sustained concurrent BCAI and BVAI. Two carotid injuries were unilateral (Rt 0, Lt 2). Vertebral artery injuries were unilateral in eight patients (Rt 3, Lt 5) and bilateral in one. Clinical characteristics of BCVI patients are summarized in Table 1.

II. Factors associated with BCVI

There were no significant differences between BCVI and non-BCVI patients in age, sex, mechanism of injury, neurological status, associated intracranial traumatic hemorrhage, or cerebral infarction. The level of CSI, presence of facet, and transversarium fracture were also not predictive of BCVI. Conversely, dislocation of the vertebral body is significantly correlated with BCVI on univariate analysis ($p = 0.04$) (Table 2). Also, dislocation of the vertebral body was the only factor significantly associated with BCVI in multivariate analysis [adjusted odds ratio (aOR): 6.00; 95% confidence interval (CI): 1.19–36.05; $P = 0.03$].

III. Factors associated with BVAI

Because there were few patients with BCAI to analyze, risk factors for BVAI were also investigated. There was no significant difference between BVAI and

Table 1 Clinical characteristics of 10 patients with BCVI

Patient no.	Age/ Sex	GCS	Frankel grade	Cause	Mechanism	Level	Dislocation	Transversarium fracture	Facet fracture	Spinous process fracture	Body fracture	ICH	Vessels	Denver grade
1	36 M	E4V5M6	A	TA	Extension	C6	–	–	+	–	–	–	Rt VA	II
2	62 M	E3V5M6	E	Fall	Extension	C7	–	–	–	–	–	EDH	Lt ICA Lt VA	IV IV
3	59 M	E4V5M6	D	Fall	Extension	C5/6	–	+	+	–	–	–	Rt VA	II
4	34 M	E4V4M6	A	TA	Extension	C4/5	+	+	+	–	+	SAH	Rt VA	II
5	58 M	E4V4M6	A	Fall	Flexion	C5/6	+	+	+	–	–	SAH	Lt VA	II
6	31 M	E4V5M6	A	Fall	Flexion	C4/5	+	+	+	–	–	–	Rt VA Lt VA	IV II
7	77 M	E4V5M6	D	Fall	Flexion	C4/5	+	+	+	–	–	–	Lt VA	IV
8	41 M	E1V1M2	A	TA	Extension	C5/6	+	+	+	–	–	–	Lt VA	IV
9	69 M	E4V5M6	D	NA	Extension	C4/5	+	+	+	–	–	–	Lt VA	IV
10	48 M	E1V2M5	E	Fall	Others	C6	–	–	–	–	+	SAH	Lt ICA	I

BCVI: blunt cerebrovascular injury, EDH: epidural hematoma, GCS: Glasgow Coma Scale, ICA: internal carotid artery, ICH: intracranial hemorrhage, M: male, NA: not available, SAH: subarachnoid hemorrhage, TA: traffic accident, VA: vertebral artery.

Table 2 Comparison of BCVI and non BCVI patients

Variable	BCVI patients (N = 10)	non-BCVI patients (N = 30)	P value
Age (mean)	51.5 ± 15.8	58.8 ± 19.2	0.29
Gender (M/F)	10/0	25/5	0.31
Frankel grade (A/other)	5/5	7/23	0.13
Injury classification (E/others)	6/4	7/23	0.10
Dislocation	6	6	**0.04**
Transversarium fracture	7	14	0.28
Facet fracture	8	16	0.26
Upper cervical spine fracture	0	13	0.02
Middle and lower cervical spine fracture	10	21	0.08
Traumatic intracranial hemorrhage	4	7	0.42
Cerebral infarction	2	2	0.26

BCVI: blunt cerebrovascular injury, E: extension, F: female, M: male.

non-BVAI patients in age, sex, neurological status, or associated intracranial traumatic hemorrhage or cerebral infarction. The level of CSI, presence of transversarium fracture was also not predictive of BVAI. However, there was a significant correlation with mechanism of injury (P = 0.01), and presence of dislocation (P = 0.01). Facet fracture was observed in a high proportion of BVAI patients, but the difference was only marginally significant (P = 0.06) (Table 3). On multivariate analysis, mechanism of injury (aOR: 2.81; 95% CI: 1.16–8.19; P = 0.02) and dislocation (aOR: 9.53; 95% CI: 1.64–80.82; P = 0.01) were found to be significant predictors of BVAI.

IV. Treatment and outcome

Immediately, the CSIs were treated with posterior fusion in 21, with anterior fusion in 3, and halo-vest immobilization in 6 patients, whereas 10 patients without instability were managed conservatively. One patient underwent urgent craniotomy and hematoma evacuation for associated acute epidural hematoma.

Cerebral infarction relevant to BCVI was found in 2 patients (20%). One patient showed rapid deterioration of his neurological condition and died 8 days after injury before antithrombotic therapy was considered (Fig. 1, case 8). In the other patient, asymptomatic small infarction was identified after urgent craniotomy for traumatic intracranial hemorrhage. Antiplatelet therapy (oral clopidogrel 75 mg/d) was rapidly introduced and he has remained neurologically stable with a follow-up period of 3 years (Fig. 2, case 2). Another patient with unilateral traumatic vertebral artery occlusion with severe sclerotic stenosis in contralateral vertebral artery has undergone prophylactic antiplatelet therapy (oral aspirin 100 mg/d) without subsequent neurological event. None of the remaining eight patients without acute ischemic insult due to BCVI developed delayed neurological deterioration during the observation period. Those included two patients who were subsequently found to have deep venous thrombosis and anticoagulation therapy was temporary introduced.

Discussion

BCVI is known to result from rapid acceleration-deceleration of the body by subsequent stretching of the involved vessels. Proposed mechanisms include a direct blow to the neck, hyperextension with contralateral rotation of the head, laceration of the artery by adjacent fractures involving the sphenoid, or petrous bones and direct intraoral trauma with a hard object.[5] These mechanisms cause an intimal tear that exposes subendothelial collagen, thereby initiating platelet aggregation to form a healing thrombus, which may produce stenosis or occlusion. More frequently, vascular injuries have been observed in CSI patients.[7,11,15] This study also supports the notion that BCVI is a common sequela of CSI, occurring in 25% of patients who undergo screening using 3D-CTA and/or MRA in patients with significant fractures including subluxation, extension into the foramen transversarium, facet, spinous process, and vertebral body. The incidence of BCAI and BVAI were 5% and 22.5%, respectively, and comparable to the previous reports with screening based on various identifiable risk factors.[2,9,16] Reported risk factors for BCVI include a Glasgow Coma Scale measure of less than or equal to six,[17] petrous bone fracture,[17] diffuse axonal injury,[17] and Lefort II or III fractures.[17] The relation between traumatic intracranial hemorrhage

Table 3 Comparison of BVAI and non BVAI patients

Variable	BVAI patients (N = 9)	non-BVAI patients (N = 31)	P value
Age (mean)	51.9 ± 16.7	58.4 ± 19.0	0.36
Gender (M/F)	9/0	26/5	0.57
Frankel grade (A/other)	5/4	7/24	0.10
Injury classification (E/other)	6/3	7/24	**0.01**
Dislocation	6	6	**0.01**
Transversarium fracture	7	14	0.13
Facet fracture	8	16	0.06
Upper cervical spine fracture	0	13	0.02
Middle and lower cervical spine fracture	9	22	0.40
Traumatic intracranial hemorrhage	3	8	0.60
Cerebral infarction	2	2	0.27

BVAI: blunt vertebral artery injuries , E: extension, F: female, M: male.

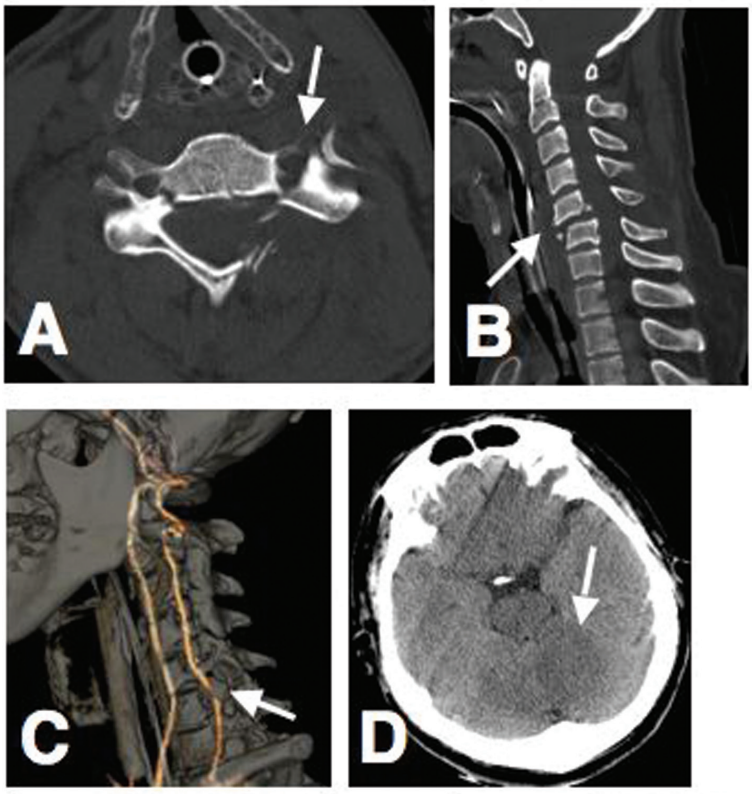

Fig. 1 Several imaging studies in a 41-year-old male (case 8). A: Cervical spinal computed tomography (CT) axial scan showing significant facet fractures extending into the foramen transversarium (*arrow*). B: Cervical spinal CT sagittal scan showing a significant subluxation at the level of C5/6 (*arrow*). C: Three dimensional (3D)-CT angiogram demonstrating an abrupt discontinuance of left vertebral artery (*arrow*) at the site of the fracture. D: Brain CT axial scan demonstrating a cerebral infarction in left cerebellar hemisphere (*arrow*).

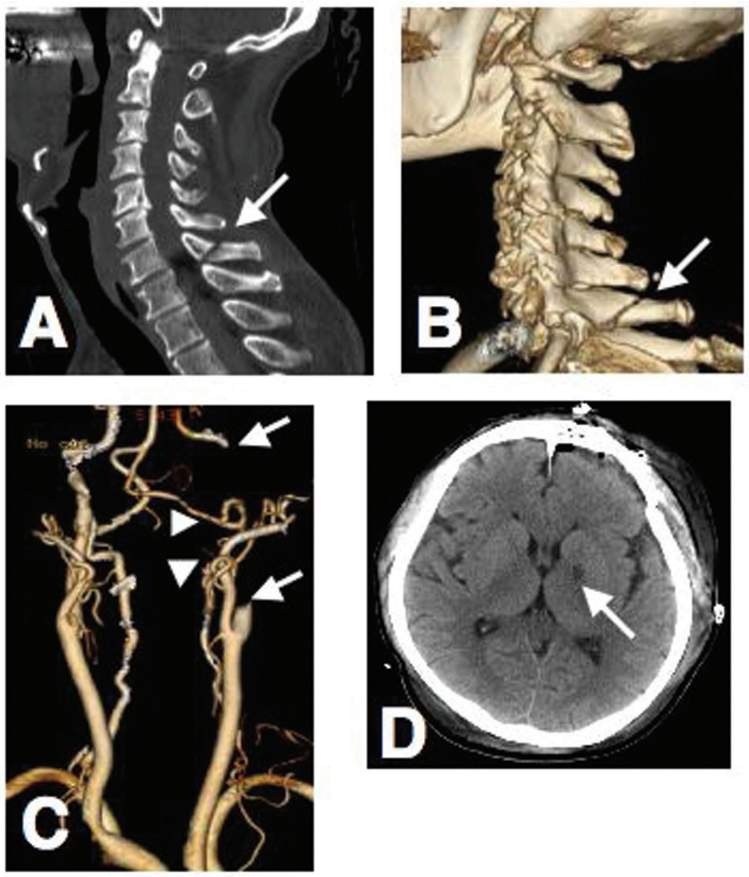

Fig. 2 Several imaging studies in a 62-year-old male (case 2). A,B: Cervical spinal computed tomography (CT) sagittal scan (A) and three dimensional (3D)-CT (B) showing spinous process fracture of 7th cervical spine (*arrow*). C: 3D-CT angiogram demonstrating an abrupt discontinuance of left Internal cortid artery (*arrows*) and vertebral artery (*arrowheads*). D: Brain CT axial scan obtained after urgent craniotomy and epidural hematoma evacuation demonstrating a cerebral infarction in the left basal ganglia (*arrow*).

(tICH) and BCVI were elucidated,[2,7,10] and there was no significant association in the present study.

BCAI is more likely to be associated with head injury. The common mechanism included hyperextension caused by stretching of the carotid artery over the lateral articular processes of C1 to C3.[18,19] Hyperflexion compresses the cervical internal carotid artery (ICA) between the angle of the mandible and the cervical spine, and extreme head rotation impinges the ICA against the styloid process.

Convincingly, BVAI is more frequently associated with CSI. Several authors reported CSI as the only independent predictor of BVAI.[7,11,15] Most commonly, BVAI occurs in the V2 or V3 segments where the vessels travel through the transverse foramina or windows around C1. Other mechanisms include direct injury caused by associated fractures of the vertebrae involving the transverse foramen through which the artery courses and hyperextension-stretch injury caused by tethering of the artery within the lateral masses of the cervical spine.[18,19]

Concerning the specific patterns of CSI related to BVAI, fractures involving the transverse foramen, subluxation, and fractures involving the upper cervical spine have been noted.[16,20,21] Cothren et al.[22,23] reported a 37% frequency of BVAI in patients with these three types of fractures, and screening protocol using these three fracture patterns can detect 93% of BVAI. Shimokawa et al.[24] stated that BVAI was recognized in 62.5% of cases with dislocation of the vertebral body (> 5 mm), and emphasized a significant association between BVAI and subluxation. Conversely, several authors reported that the middle cervical spine (C4/5 or C5/6) as the most affected level of CSI in patients with BVAI.[11,24,25]

Generally, hyperflexion is a much more common mechanism than hyperextension in CSI,[25] but it remains controversial whether hyperflexion is the most common mechanism in patients with BVAI.[2,24] The present study showed hyperextension injury is the independent predictor. Our data support the notion that there is no correlation between the severity of spinal cord injury and BVAI,[2,16] reflecting that the vertebral artery is located on the lateral aspect of the cervical spine and is assumed to be more susceptible to rotational injury and the spinal cord can be severely injured by sagittal plane insults such as compression and hyperflexion.

Numerous single center studies have reported that anticoagulation and antiplatelet therapis reduce the risk of ischemic stroke in these patients,[26–29] but the optimal antithrombotic regimen remains to be elucidated. The Cervical Artery Dissection in Ischemic Stroke Patients (CADISP) Study Group recommends antiplatelet therapy in most patients with BCVI, but advocates anticoagulation in patients with recurrent symptoms while on antiplatelet therapies, with vessel occlusion, or with injuries associated with free-floating intralaminal thrombus.[30] Accordingly, our indication of antiplatelet therapy in the case with existing or impending cerebral infarction seems acceptable.

There has been no consensus on the optimal diagnostic modality for patients with possible BCVI. Conventional catheter angiography is the most accurate method for simultaneous evaluation of collateral flow. However, it is not practical as a screening method due to its invasiveness, especially in critically ill patients with unstable spines. CTA and MRA are evolving as key imaging modalities for the noninvasive assessment of the vascular system. With increasing availability and improved accuracy, CTA has widely replaced DSA as the first choice diagnostic tool for BCVI in many institutions.[6,7,31] Because of the well-developed collateral circulation in the neck, the majority of BCVIs can remain asymptomatic. Unrecognized BCVI can result in a poor functional outcome and potentially fatal ischemic sequelae. The prevalence of BCVI in patients with any CSI (10.9%; 10/92) in this study is comparable to the previous studies.[5,15,22] Conversely, it is relatively high (25%; 10/40) in selected patients with neurological deficits and/or significant fracture including fracture of foramen transversarium, facet, spinous process, and vertebral body. Our data suggest the need for early imaging check-ups for those high-risk patients in institutional practice guidelines.

Conclusion

Patients with significant CSI including subluxation, extension into the foramen transversarium and vertebral body are associated with a high risk of BCVI, not depending on neurological status. Patients with hyperextension injury and dislocation of the vertebral body require urgent evaluation for BCVI.

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Conflicts of Interest Disclosure

The authors have no personal, financial, or institutional

interest in any of the drugs, materials, or devices in the article.

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