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Transcranial Doppler microemboli monitoring for stroke risk stratification in blunt cerebrovascular injury

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Abstract

Objective—To assess whether microemboli burden, assessed noninvasively by bedside transcranial Doppler (TCD) ultrasonography, correlates with risk of subsequent stroke >24 hours after hospital arrival among patients with blunt cerebrovascular injury (BCVI). The >24 hour timeframe provides a window for TCD exams and therapeutic interventions to prevent stroke.

Design—Retrospective cohort study.

Setting—Level I trauma center.

Patients—1,146 BCVI patients over ten years.

Interventions—None.

Measurements and Main Results—We identified 1,146 BCVI patients; 54 (4.7%) experienced stroke detected >24 hours after arrival. Among those with isolated ICA injuries, 5/9 with delayed stroke had positive TCDs (at least one microembolus detected with TCDs) before stroke, compared to 46/248 without (R.R. = 5.05, 95% C.I. 1.41–18.13). Stroke risk increased with the number of microemboli (a.R.R. 1.03 per microembolus/hour, 95% C.I. 1.01–1.05) and with persistently positive TCDs over multiple days (R.R. 16.0, 95% C.I. 2.00–127.93). Among patients

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who sustained an ICA injury with or without additional vessel injuries, positive TCDs predicted stroke after adjusting for ipsilateral and contralateral ICA injury grade (a.R.R. = 2.91, 95% C.I. 1.42–5.97). No patients with isolated VA injuries had positive TCDs before stroke, and positive TCDs were not associated with delayed stroke among patients who sustained a VA injury with or without additional vessel injuries (R.R. 0.90, 95% C.I. 0.21–3.83).

Conclusions—Microemboli burden is associated with higher risk of stroke due to ICA injuries, but monitoring was not useful for VA injuries.

Keywords

Blunt cerebrovascular injury; Transcranial Doppler ultrasound; Stroke; Vascular system injuries; Diagnostic imaging; Trauma

Introduction

Injury to the carotid (ICA) or vertebral arteries (VA) occurs in up to 1% of patients with blunt trauma and confers a 5-10% risk of stroke (1-5). Implementation of screening protocols in patients at risk for blunt cerebrovascular injury (BCVI) has enabled detection of asymptomatic injuries, and has therefore provided an opportunity to prevent stroke (3, 6-8). Still, there is debate regarding optimal medical therapy to mitigate stroke risk following BCVI diagnosis. Both preventative anticoagulation (6, 9-12) and antiplatelet therapy (4, 5, 8, 13-16) have been advocated, but there are no data to support the superiority of either approach.

At our institution, we routinely employ transcranial Doppler (TCD) microemboli monitoring, which is a safe, noninvasive, bedside ultrasonography test that may enable stroke risk stratification. The presence of microemboli on TCD exams is associated with stroke in atrial fibrillation, atherosclerotic carotid stenosis, non-traumatic dissections, and in patients undergoing surgery (17-22). TCDs are routinely performed during stroke workup at some centers. In BCVI, our group has previously shown that individuals with microemboli distal to a traumatic carotid pseudoaneurysm or occlusion were more likely to have suffered a stroke, although the timing of stroke relative to the TCD exam was not considered (14, 15).

It remains unknown how well TCD microemboli monitoring predicts the risk of subsequent stroke in BCVI. Furthermore, earlier studies have not examined TCD findings in lower-grade injuries, which are more common than high-grade injuries (23). If the probability of stroke can be predicted, aggressive interventions may be targeted selectively to those at greatest risk.

Materials and Methods

Patient selection and data collection

This was a retrospective cohort study performed at Harborview Medical Center, the only Level I trauma center in Washington State and a regional trauma referral center for Alaska, Montana, and Idaho. This study was approved by the University of Washington Institutional Review Board, which waived the need for informed consent.

We included all adult and pediatric patients with BCVI treated at our center from 4/1/2005 through 6/30/2015. Patients were identified from our institutional trauma registry and a separately maintained database of TCD exams using ICD-9-CM and AIS codes for BCVI. Patients who died within 48 hours were excluded, as their degree of illness frequently limited our ability to detect BCVI and stroke.

Demographic, injury, clinical, radiology, TCD and medication data were obtained from patients' medical records and our institutional trauma registry. Stroke presence was ascertained using the trauma registry, which utilizes prospective, manual assessment of in-hospital complications by trained reviewers in accordance with American College of Surgeons Trauma Quality Improvement Program criteria (24). All patients with stroke underwent detailed chart review to confirm stroke occurrence, location, timing, progression (defined as discrete enlargement of initial stroke or new stroke in a distinct territory), and etiologies.

TCD ultrasonography for BCVI

TCD monitoring was performed over at least three once-daily exams by a Registered Vascular Technician using a Natus SONARA ultrasound machine with a 2MHz pulsed wave probe. For ICA injuries, monitoring was performed on the ipsilateral middle cerebral artery at 50mm depth. For VA injuries, monitoring was performed on either the basilar artery at 90mm depth or the bilateral posterior cerebral arteries at 60-70mm depth, depending on bone windows and whether the patient required a cervical collar. Microembolic signals (MES) were defined as ≥ 3 dB above background intensity, lasting <0.1 second, randomly occurring, unidirectional, and causing a spike in the intensity trace. For each injured vessel, microemboli were counted during approximately 20 minute exams (as permitted by clinical stability) and extrapolated to hourly rates. The relatively brief assessment time was chosen to minimize clinical disruption for these injured patients. The primary analysis utilized a binary assessment of TCDs as either positive (≥ 1 microembolus) or negative; a secondary analysis considered the hourly rate of microemboli. For patients with delayed BCVI stroke (as defined below), we considered only TCD exams performed before stroke.

BCVI grading

BCVI grade was assigned through manual review of radiology reports according to the Denver Criteria: Grade 1 for $<25\%$ luminal narrowing; Grade 2 for $>25\%$ luminal narrowing, intraluminal thrombus, or intimal flap; Grade 3 for pseudoaneurysm; Grade 4 for vessel occlusion; and Grade 5 for vessel transection and/or active extravasation (23). When radiology records were insufficient to assign injury grade, images were reviewed by a board-certified neuroradiologist blinded to clinical data (M.M.B.).

BCVI-related stroke

BCVI-related strokes were those that occurred in the vascular distribution of the injured vessel and were not definitively determined to be due to pre-existing cerebrovascular disease, global hypoxia/hypoperfusion (e.g. following cardiac event, drowning, hypotensive event, strangulation), paradoxical embolism, vasospastic process, cardiac arrhythmia, venous infarction, neurosurgical intervention, direct trauma, or herniation syndrome (flowchart

available in Supplemental Digital Content). Etiologic determination was based on directed workup and assessment by the inpatient teams at our Level I Stroke Center. The primary outcome was new BCVI-related stroke, or documented progression of a stroke, detected more than 24 hours after arrival. The 24-hour cutoff provides time to perform TCD exams and make therapeutic changes to prevent stroke, and allowed us to focus on potentially-preventable strokes definitively occurring subsequent to TCD exams.

Medication use

Our institutional approach is to initiate aspirin immediately upon BCVI diagnosis. For patients at risk for hemorrhagic complications from concomitant injuries, therapy is initiated upon stabilization of imaging studies and hemodynamic trends. Patients deemed at high risk of stroke based on imaging characteristics or TCD exams are escalated to more aggressive therapy as permitted by other injuries (14, 15). For the present study, medication escalation was defined as a transition from aspirin to more aggressive regimens that included clopidogrel (e.g. dual antiplatelet therapy) or therapeutic anticoagulation. Medication use prior to stroke or in the first 15 days (corresponding to the latest stroke in our cohort) was assessed.

Data analysis

Descriptive evaluations were performed, as were bivariable analyses assessing injury grade, medication administration, number of vessels injured and TCD results, stratified by stroke status and injured vessel. Due to the large number of injury permutations with five grades of injury across four vessels and the possibility that concomitant vascular injuries may influence the risk of stroke, the primary analysis focused on patients with isolated vascular injuries. Multivariable analyses adjusting for injury grade were conducted using Poisson regression to estimate risk ratios. For the primary analyses focusing on isolated injuries, exact Poisson regression was used given the small number of strokes.

We also performed a secondary analysis of patients who sustained an ICA injury with or without concomitant ICA or VA injuries. Due to the complexity of analyzing all possible permutations of injury, this analysis was conducted at the level of the injured vessel with stroke in the ipsilateral hemisphere as the outcome. We used a Poisson regression with robust standard errors and adjusted for ipsilateral ICA injury grade, and contralateral ICA injury grade (when present). This model was clustered at the level of the patient to account for the non-independence of these observations. An analogous analysis was not performed for non-isolated VA injuries because both vessels supply largely the same vascular territory and, thus, posterior circulation strokes could not definitively be attributed to a single VA. Statistical analysis was performed using Stata/IC version 14 (StataCorp LP, College Station, TX).

Results

We identified 1,955 patients using registry queries. BCVI was confirmed upon chart review in 1,215; of these, 69 were excluded due to death within 48 hours. The final cohort included

1,146 patients. The total number of blunt trauma patients captured in our trauma registry over the same timeframe was 47,733, yielding a BCVI rate of 2.4%.

Patient characteristics and outcomes

Of 108 patients with any stroke, 15 were definitively due to causes other than BCVI and another 41 were present on arrival or detected within the first 24 hours, leaving 52 delayed strokes attributed to BCVI. Stroke was confirmed with MRI in 75% of the 108 patients with any stroke, and in 71% of the 52 patients with delayed BCVI stroke; the remainder were diagnosed using longitudinal CT evaluation. There was no significant shift in diagnostic modality over the study duration ($p = 0.914$, linear regression).

Table 1 tabulates demographic, injury, hospitalization, and outcome characteristics for patients with early stroke, delayed stroke or no stroke. Ninety percent of all patients underwent at least one TCD exam, which was similar across injury grades. Antiplatelet or anticoagulant therapy was administered to 63.1% of patients who did not develop BCVI stroke, and to 75.0% of patients who did develop delayed BCVI stroke. Patients with BCVI stroke had high mortality, with stroke directly causing or contributing to death in 77.8% and 76.5% of those whose events occurred before or after 24 hours, respectively.

Among patients without stroke, 74.2% had an isolated vascular injury compared to 48.1% of patients with delayed stroke. As shown in Table 2, the risk of delayed BCVI stroke increased with the number of injured vessels.

Internal carotid artery injuries

Of 257 patients with isolated ICA injuries undergoing TCD exams, nine had delayed BCVI stroke. Five (55.6%) of these nine patients had positive TCDs prior to stroke detection, compared to only 52 (18.5%) of the 248 without delayed BCVI stroke. Thus, patients with positive TCDs had a 5.05-fold increase in the likelihood of delayed stroke compared to those with negative TCDs (95% CI 1.41–18.13). Table 3 shows the associations between TCDs and stroke stratified by ICA injury grade; all strokes following positive TCDs occurred in patients with grade 2-4 injuries.

The number of microemboli detected was also an important determinant of stroke risk. Among patients with isolated ICA injuries, stroke risk was 1.9% in patients without microemboli, 0% in patients with 1-6 MES/hour, 6.7% in patients with 7-40 MES/hour, and 40.0% in patients with >40 MES/hour ($P = .001$, Spearman correlation).

Adjusted analyses are shown in Table 4; after adjusting for injury grade, the observed trend persisted although the association between positive TCDs and stroke was no longer statistically significant. However, there was a significant association between the numbers of microemboli detected and stroke, with the risk of stroke increasing by 3% per MES/hour. We performed a secondary analysis to explore the generalizability of these estimates to patients with non-isolated ICA injuries. After adjustment for ipsilateral and contralateral ICA injury grade, patients with 1 MES were nearly 3 times as likely to stroke (95% CI 1.42–5.97), with the risk increasing by 1% per MES/hour (95% CI 1.01–1.02).

We then examined the relationship between serial TCD results, medication use, and stroke in patients with isolated ICA injuries. As shown in Figure 1, delayed stroke was more common in patients whose TCDs remained positive than in patients whose TCDs became negative ($4/10 = 40\%$ vs $1/40 = 2\%$; R.R. 16.0, 95% C.I. 2.00–127.93). However, escalation of medical therapy did not significantly affect TCD results or the risk of stroke. TCDs were just as likely to remain positive in patients receiving escalated therapy as those without ($4/16$ (25%) with escalated therapy; $7/39$ (17.9%) without; R.R. 1.39, 95% C.I. 0.47–4.11), and all patients who developed stroke after positive TCDs did so despite more aggressive care.

Vertebral artery injuries

Delayed BCVI stroke was observed in eight (1.8%) of 449 patients with isolated VA injuries who underwent TCD exams (Table 3). No patients with stroke had positive TCDs beforehand. Among patients with single or multiple injuries, two suffered delayed posterior circulation BCVI stroke following positive TCDs. However, positive microemboli were not more common in patients with delayed posterior circulation BCVI stroke than in patients without stroke (R.R. 0.90, 95% CI 0.21–3.83).

Medical therapy did not clearly affect TCD results in patients with isolated VA injuries. In those with positive TCDs, continued microemboli were seen in one (12.5%) of eight patients whose medications were escalated, compared to eleven (23%) of 41 patients whose medications were not escalated (R.R. 0.46, 95% C.I. 0.07–3.12).

Discussion

1,146 patients with 1,527 blunt cerebrovascular injuries were studied over the 123 months of this study, representing the largest BCVI cohort to date. Consistent with previous data, the frequency of BCVI-attributed stroke was 8.1% (1-5). While many strokes occurred early, 4.5% of our cohort had strokes that were detected >24 hours after arrival, and were therefore potentially preventable. Overall, 32.7% of patients with delayed BCVI stroke died, with stroke either causing or contributing to death in 77%; while this rate compares favorably with historical series (2, 25, 26), it remains unacceptably high and highlights the importance of stroke prevention.

Carotid artery injuries

Our data demonstrate that TCD exams may help providers identify patients at higher risk of stroke due to blunt ICA injuries. Emboli detection was a significant predictor of stroke in crude analyses and in most adjusted models, despite the small number of delayed strokes in the dataset.

Ideally, detection of emboli should inform effective stroke prophylaxis. However, all patients sustaining stroke after positive TCDs did so despite up-titration of their medical therapy. Likewise, escalated medical management was not associated with normalization of TCD exams. This finding is in contrast to the CARESS study of symptomatic carotid stenosis, which demonstrated that addition of clopidogrel to aspirin monotherapy led to a reduction in microembolic burden (27). This discrepancy may be due to differences between asymptomatic acute BCVI and symptomatic chronic carotid stenosis, as well as the

nonrandom assignment of treatments in our cohort. Still, given the retrospective nature of this study we cannot ascertain what patients' embolic burden or extent of stroke might have been in the absence of medical therapy, since this was targeted toward patients at the highest risk. Thus, the ability of TCDs to drive medical management and procedural interventions to prevent stroke therefore remains unclear; further prospective study is warranted to more clearly elucidate the effect of TCD-guided management on stroke prevention.

Vertebral artery injuries

We did not identify an association between the results of TCD exams and subsequent BCVI-related posterior circulation stroke due to VA injuries, suggesting that TCDs are not useful in stratifying risk in these patients.

Commentary

We acknowledge several limitations to this study. While it is the largest BCVI cohort to date, our primary outcome was rare. Therefore, we could include only a small number of predictor variables in multivariable models, which may not represent all factors influencing stroke risk. Additionally, we faced limitations regarding stroke ascertainment: identification of stroke timing requires recognition of subtle clinical changes, which were not always clearly documented in the medical record, and which may have been obscured by concomitant brain injury, spinal cord injury, and mechanical ventilation with sedation. This limitation is confounded by the limited sensitivity of CT for acute stroke (28); strokes present on admission in patients with unreliable exams may not be detected until follow up imaging is obtained hours or even days later. Furthermore, stroke was identified using our institutional trauma registry. While we expect good capture given the use of trained coders, stroke's significance as a quality metric, and the importance of capturing stroke for billing purposes, ascertainment may have been incomplete.

We also faced analytic limitations related to the nonrandom treatment of patients within our retrospective study. Decisions on medication administration may have been influenced by a host of other factors besides TCD results, including concomitant injuries (e.g. intracranial hemorrhage), comorbidities (e.g. venous thromboembolism), and anatomic features of the BCVI. It is therefore difficult to know with certainty whether medication changes were made in response to TCD exam findings or other factors. Additionally, although all patients who experienced stroke after positive TCDs did so despite escalation in their care, it is impossible to know if these strokes would have been worse if the interventions had not been made.

Finally, while a brief monitoring duration was used to minimize clinical disturbance, the TCD literature suggests that longer monitoring durations can enhance the detection of microemboli in other scenarios (29). Given these optimistic results with short-duration exams, longer duration monitoring may further improve stroke risk discrimination.

Conclusions

In a large series of patients with BCVI, we found that TCD microemboli monitoring identified patients at higher risk of delayed BCVI stroke due to grade 2, 3, or 4 ICA injuries. However, our data do not support the use of TCD microemboli monitoring in patients with

grade 1 ICA injuries or VA injuries of any grade. Further prospective study with randomized trials is warranted to determine if TCD-guided management can effectively prevent stroke due to higher-grade blunt carotid injuries.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
Trends on serial transcranial Doppler exams and delayed stroke in patients with isolated carotid artery injuries.

Table 1

Demographics, injury characteristics, and outcomes.

Variables	No BCVI stroke N=1,053	BCVI stroke	
		BCVI stroke detected N=41	24 hours BCVI stroke detected >24 hours N=52
Demographics			
Median age (IQR)	44 (27, 62)	38 (24, 50)	42.5 (24.5, 63)
Male sex	698 (66.3%)	29 (70.7%)	32 (61.5%)
Hispanic ethnicity	85 (8.1%)	3 (7.3%)	2 (3.8%)
Payer			
Private/Commercial	344 (32.7%)	14 (34.1%)	25 (48.1%)
Medicaid	230 (21.8%)	14 (34.1%)	9 (17.3%)
Medicare	221 (21.0%)	5 (12.2%)	9 (17.3%)
Other	258 (24.5%)	8 (19.5%)	9 (17.3%)
Mechanism			
MVC or other transport	597 (56.7%)	18 (43.9%)	27 (51.9%)
Fall	303 (28.8%)	9 (22.0%)	14 (26.9%)
Pedestrian	59 (5.6%)	6 (14.6%)	3 (5.8%)
Struck by or against	27 (2.6%)	1 (2.4%)	0 (0.0%)
Other or unknown	67 (6.4%)	7 (17.1%)	8 (15.4%)
Injury and hospitalization characteristics			
Transferred from outside hospital	537 (51.0%)	26 (63.4%)	25 (48.1%)
Median ISS (IQR)	26 (14, 38)	34 (26, 45)	39.5 (27.5, 50)
Median maximum head AIS (IQR)	3 (0, 4)	3 (2, 5)	3.5 (2.5, 5)
Median maximum neck AIS (IQR)	2 (2, 3)	3 (2, 3)	3 (2.5, 3)
Hypotension in ED (SBP <90 mmHg)	206 (19.6%)	10 (24.4%)	10 (19.2%)
At least one TCD exam	936 (88.9%)	38 (92.7%)	49 (94.2%)
Median number of TCD exams	3 (2, 3)	3 (2, 3)	4 (3, 6)
Patient outcomes			
Hospital disposition			
Home or independent care	475 (45.1%)	9 (22.0%)	6 (11.5%)
Inpatient rehabilitation	216 (20.5%)	13 (31.7%)	14 (26.9%)
Intermediate care facility	224 (21.3%)	9 (22.0%)	14 (26.9%)
Deceased >48 hours post-arrival ^a	91 (8.6%)	9 (22.0%)	17 (32.7%)
Other	47 (4.5%)	1 (2.4%)	1 (1.9%)
Median hospital LOS (IQR)	9 (4, 18)	16 (6, 26)	15 (9, 27)

^aAny patients deceased <48 hours were excluded from the study, which may differentially effect the estimates of >48h mortality for 24 hour BCVI stroke patients compared to >24h BCVI stroke patients and no BCVI stroke patients.

BCVI: blunt cerebrovascular injury; IQR: interquartile range; MVC: motor vehicle crash; ISS: Injury Severity Score; AIS: Abbreviated Injury Scale; ED: emergency department; SBP: systolic blood pressure; TCD: transcranial Doppler; ICU: intensive care unit; LOS: length of stay.

Table 2

Risk of stroke by number of vessels with blunt cerebrovascular injury and vascular distribution of strokes.

Number of injured vessels ^a	No BCVI stroke N=1,053	BCVI stroke	
		BCVI stroke detected N=41	24 hours BCVI stroke detected N=52
1 vessel (n=826)	782 (94.7%)	19 (2.3%)	25 (3.0%)
2 vessels (n=276)	241 (87.3%)	17 (6.2%)	18 (6.5%)
3 vessels (n=27)	17 (63.0%)	4 (14.8%)	6 (22.2%)
4 vessels (n=17)	13 (76.5%)	1 (5.9%)	3 (17.7%)
Stroke vascular territory			
ICA distribution	—	20 (48.8%)	29 (55.8%)
VA distribution	—	19 (46.3%)	17 (32.7%)
Both ICA and VA distributions	—	2 (4.9%)	6 (11.5%)

^aP < .001 by Fisher's exact test. Percentages shown are row percentages.

BCVI: blunt cerebrovascular injury. ICA: internal carotid artery. VA: vertebral artery

Table 3

Association between injury grade, transcranial Doppler results, and blunt cerebrovascular injury-associated stroke detected after 24 hours in patients with isolated carotid injuries or isolated vertebral injuries.

Vessel & Grade	BCVI stroke detected > 24 hours		No BCVI stroke	
	TCD positive	TCD negative	TCD positive	TCD negative
ICA Grade				
Grade 1 (n = 115)	0	1	19	95
Grade 2 (n = 78)	2	0	14	62
Grade 3 (n = 53)	2	2	9	40
Grade 4 (n = 10)	1	0	4	5
Grade 5 (n = 1)	0	1	0	0
Total ICA (n = 257)	5	4	46	202
VA Grade				
Grade 1 (n = 187)	0	1	20	166
Grade 2 (n = 114)	0	1	9	104
Grade 3 (n = 23)	0	0	3	20
Grade 4 (n = 131)	0	5	20	106
Grade 5 (n = 2)	0	1	0	1
Total VA (n = 457)	0	8	52	397

BCVI: blunt cerebrovascular injury; ICA: internal carotid artery; TCD: transcranial Doppler; VA: vertebral artery.

Table 4

Effect of transcranial Doppler results on grade-adjusted risk of delayed blunt cerebrovascular injury-associated stroke in patients with carotid artery injuries.

Multivariable Models	Adjusted risk ratio of BCVI stroke detected >24 hours	95% confidence interval
Isolated internal carotid artery injuries ^a		
Maximum emboli per hour	1.03	1.01–1.05
Emboli positive	3.78	0.80–19.42
All internal carotid artery injuries ^b		
Maximum emboli per hour	1.01	1.01–1.02
Emboli positive	2.91	1.42–5.97

^aExact Poisson regression, adjusting for grade of the injured vessel.

^bThese analyses included patients with multiple vascular injuries. Poisson regression with robust confidence intervals, adjusting for grade of the ipsilateral and contralateral carotid injuries (if present) and clustering at the level of the patient.

BCVI: blunt cerebrovascular injury.