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Influence of luminal stenosis in aneurysmal and non-aneurysmal blunt cerebrovascular injury

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ABSTRACT

Background: Current blunt cerebrovascular injury (BCVI) grading grossly differentiates injury characteristics such as luminal stenosis (LS) and aneurysmal disease. The effect of increasing degree of LS beyond the current BCVI grading scale on stroke formation is unknown.

Study Design: BCVI over a 3-year period were retrospectively reviewed. To investigate influence of LS beyond the BCVI grading scale within aneurysmal and non-aneurysmal BCVI, grade 2 BCVI were subdivided into BCVI with ≥ 25% and ≤ 50% LS and BCVI with > 50% and ≤ 99% LS. Grade 3 BCVI were subdivided into BCVI with pseudoaneurysm (PSA) without LS and BCVI with PSA and LS. We hypothesized increased LS beyond the current BCVI grade distinctions would be associated with higher rates of stroke formation.

Results: 312 BCVI were included, of which 140 were carotid BCVI and 172 vertebral BCVI. Sixteen carotid BCVI underwent endovascular intervention (EI) and 19 suffered a stroke. In carotid BCVI stroke rates increased sequentially with BCVI grade except in grade 3. There was a stroke rate of 12% in grade 1 carotid BCVI, 18% in grade 2, 6% in grade 3, and 31% in grade 4. In subgroup analysis for grade 2 carotid BCVI, BCVI with > 50% and ≤ 99% LS had higher rates of stroke (22% vs. 15%, p = 0.44) than BCVI with ≥ 25% and ≤ 50% LS. In subgroup analysis of grade 3 carotid BCVI, BCVI with PSA and LS had higher rates of stroke (9% vs. 4%, p = 0.48) than BCVI with PSA without LS. Higher rates of EI in grade 2 carotid BCVI with > 50% and ≤ 99% LS (22% vs. 5%, p = 0.14) and grade 3 carotid BCVI with PSA and LS (35% vs. 4%, p = 0.01) were noted in subgroup analysis.

Conclusion: Higher percentage LS beyond the currently used BCVI grading scale has a non-significantly increased rate of stroke in both aneurysmal and non-aneurysmal BCVI. Grade 3 BCVI with PSA and LS seems to be a high-risk subgroup. Use of EI confounds modern measurement of stroke risk in higher LS BCVI.

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Introduction

In high grade blunt cerebrovascular injury (BCVI), rates of stroke can be substantial with stroke frequently responsible for patient mortality [1,2]. Pharmacologic therapy with antiplatelet agents or anticoagulation and early BCVI diagnosis decrease stroke rates [3–5]. Focus is placed on rapid screening and initiation of pharmacologic therapy to prevent strokes. Pharmacologic and endovascular BCVI treatment are chosen based on BCVI grade, which stratifies BCVI from 1 to 5 based on radiographic characteristics [6].

As BCVI grade increases so does morbidity, with higher BCVI grade associated with higher stroke rates [1,7]. However, there are many lesion characteristics which comprise BCVI, including degree of luminal stenosis (LS), intraluminal thrombus, intimal flap, and arteriovenous fistula. The presence of pseudoaneurysm (PSA) confounds the grading scale as well. It is difficult to encompass pathophysiology this complex into a watertight grading scale.

The influence of LS on stroke formation is mostly unknown. LS is only grossly differentiated between grade 1 BCVI (< 25% LS), grade 2 BCVI (≥ 25% LS and ≤ 99% LS) and grade 4 BCVI (100% LS or complete vessel occlusion) within the currently used BCVI grading scale. There is no differentiation for LS in grade 3 BCVI, with grade 3 BCVI comprising all degrees of LS with PSA. In chronic carotid stenosis higher degree of LS is associated with need for

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intervention to decrease stroke risk, and similar principles may hold true in BCVI [8]. For example in BCVI, a 30% LS is a grade 2 injury just as 90% LS is a grade 2 injury, but these BCVI are unlikely to have the same risk of stroke.

The primary aim of this study was to investigate the association between LS and rates of stroke formation for both aneurysmal and non-aneurysmal disease. We hypothesized that higher degree of LS beyond the currently used BCVI grading scale would be associated with higher rates of stroke.

Materials and methods

Institutional Review Board (IRB) approval was obtained from the University of Maryland School of Medicine. A single-institution retrospective review of BCVI was undertaken from 2012-2014. BCVI were included if the first computerized tomography (CT) scan diagnosed a BCVI, and excluded if BCVI diagnosis was initially seen on a subsequent CT. Multiple BCVI in a single patient were considered separate BCVI.

BCVI were graded with the current BCVI grading scale of 1, 2, 3, 4, or 5 [6] (Fig. 1). The BCVI grade was the first CT grade. Previous work at our institution has investigated concordance between whole-body arterial phase CT and CT angiography (CTA) [9], and we use both whole-body arterial phase CT and CTA for BCVI diagnosis in our practice. Whole-body arterial phase CT is used in a "pan scan" initial work-up and CTA is used when risk factors for BCVI are identified or with insufficient whole-body arterial phase CTs.

LS was measured from the first CT scan and was an estimation as to the percentage luminal narrowing. To investigate LS beyond BCVI grade, grade 2 BCVI and grade 3 BCVI were further subdivided by LS. Grade 2 and grade 3 BCVI were chosen as these grades have the broadest range of LS. Grade 2 BCVI was subdivided into BCVI with $\geq$ 25% and $\leq$ 50% LS, and BCVI with > 50% and $\leq$ 99% LS where our institutional guidelines already divide grade 2 BCVI. Grade 3 BCVI was subdivided into BCVI with PSA without LS and BCVI with PSA and LS. This cut-point was chosen as there is no stratification at all for LS in grade 3 BCVI in the current BCVI grading scale. LS was also measured on a continuous scale to fully investigate the effect of LS on stroke formation. Grade 1, 2, and 4 BCVI were considered non-aneurysmal BCVI, while grade 3 BCVI was considered aneurysmal BCVI.

We follow BCVI with serial CTA imaging after diagnosis. BCVI worsened if BCVI grade increased and improved if the BCVI grade decreased. CT evolution represented the change in BCVI grade from the initial CT to the last CTA obtained. If follow-up imaging was not obtained, this was noted.

Pharmacologic therapy is per institutional guidelines and varies by luminal stenosis and grade of injury. Grade 1 carotid, grade 1 vertebral and grade 2 vertebral ($\geq$ 25% and $\leq$ 50% LS) BCVI get aspirin 325 mg daily. Grade 2 carotid ($\geq$ 25% and $\leq$ 50% LS), grade 2 vertebral (> 50% and $\leq$ 99% LS), grade 3 vertebral and grade 4 vertebral BCVI get aspirin 325 mg and Plavix® 75 mg daily. Grade 2 carotid (> 50% and $\leq$ 99% LS), grade 3 carotid and grade 4 carotid BCVI get anticoagulation with either heparin, Lovenox®, or warfarin. Medical therapy often changes after stenting to dual antiplatelet therapy with aspirin 325 mg and Plavix® 75 mg daily.

Stroke was defined as stroke in the cerebral distribution of the BCVI. Strokes occurring outside the BCVI distribution (i.e.: contralateral strokes) were excluded. Strokes required radiographic evidence of stroke formation for inclusion. Stroke-related mortality was considered to be a death directly as a consequence of the BCVI, which was a qualitative assessment.

The decision for endovascular intervention (EI) is made as a joint decision between the trauma attending and neuro-interventional radiologist. The decision for EI is without a standardized protocol. EI was defined as stenting or embolization and was exclusive of diagnostic catheter angiography alone.

Statistical analysis was performed using SPSS version 24 (IBM, Armonk, New York). Univariate analysis with means and frequencies was first calculated. Histograms were assessed visually for normality. Trends in univariate variables were assessed graphically with calculation of binomial confidence intervals given the small group numbers. Bivariate analysis was performed using chi-square, ANOVA, Mann-Whitney U and linear by linear association (for ordered data) tests. Statistical significance was considered $p < 0.05$. The primary outcome was stroke. Secondary outcome was stroke-related mortality.

Results

Overall 312 BCVI were included, with 140 carotid BCVI and 172 vertebral BCVI. Injury severity score (ISS), diastolic blood pressure, systolic blood pressure, and heart rate were all normally distributed for both carotid and vertebral BCVI, allowing for assessment of means and standard deviations. Overall 24/312 (8%) of BCVI had a stroke and 2/312 (1%) had stroke-related mortality. Stroke was present on admission in 7/312 (2%) BCVI. 16/312 (5%) BCVI underwent EI.

In carotid BCVI, gender ($p = 0.05$), diastolic blood pressure ($p = 0.05$), and CT evolution ($p = 0.58$) varied by injury grade (Table 1). 19/140 (14%) carotid BCVI had a stroke. Seven of these were seen on admission: 3 strokes in grade 1 carotid BCVI, 2 strokes in grade 2 carotid BCVI, and 2 strokes in grade 4 carotid BCVI. Overall in carotid BCVI, rate of stroke did not vary significantly by BCVI grade on the initial CT imaging ($p = 0.65$).

In vertebral BCVI, gender ($p = 0.009$) and CT evolution ($p = 0.01$) varied significantly by grade. No vertebral BCVI had stroke-related mortality (Table 2). Five patients out of the 63 with grade 4 vertebral BCVI had a stroke (8%). No strokes in vertebral BCVI were present on admission. Stroke rate varied significantly by vertebral BCVI grade ($p = 0.009$). Vertebral BCVI were not further subdivided by LS given this stroke distribution. In vertebral BCVI 0/172 (0%) underwent EI.

Percentage LS was non-normally distributed for carotid BCVI. Median LS was 20% (IQR 55) for carotid BCVI. For carotid BCVI with

Accepted BCVI Grading Scale

Grade	Grade Description
1	< 25% luminal stenosis
2	≥ 25% and ≤ 99% luminal stenosis
3	Pseudoaneurysm with all degrees of luminal stenosis
4	100% luminal stenosis
5	Active extravasation with all degrees of luminal stenosis

Fig. 1. Levels of luminal stenosis in the currently used BCVI grading scale.
BCVI blunt cerebrovascular injury, LS luminal stenosis, PSA pseudoaneurysm.

Table 1
Presenting variables, radiographic progression, and outcomes for carotid BCVI stratified by the initial BCVI grade.

	Total (n = 140)	Grade 1 (n = 42)	Grade 2 (n = 38)	Grade 3 (n = 47)	Grade 4 (n = 13)	p-value
Admission Variables						
Age, mean (SD)	41(18)	44 (19)	38 (14)	41 (19)	42 (18)	0.53
Gender (Male), N(%)	79/140 (56%)	19/42 (45%)	19/38 (50%)	34/47 (72%)	7/13 (54%)	0.05
Admission SBP, mean (SD)	137 (36)	141 (51)	125 (30)	139 (39)	149 (21)	0.09
Admission DBP, mean (SD)	82 (22)	85 (25)	76 (19)	81 (22)	93 (11)	0.05
Admission HR, mean (SD)	103 (30)	96 (30)	101 (25)	111 (33)	106 (29)	0.11
ISS, mean (SD)	29 (15)	28 (16)	32 (16)	28 (12)	30 (16)	0.58
TBI, N(%)	65/139 (47%)	22/41 (54%)	17/38 (45%)	19/47 (40%)	7/13 (54%)	0.49
CT Evolution						
Post-endovascular intervention, N(%)	11/140 (8%)	0/42 (0%)	3/38 (8%)	7/47 (15%)	1/13 (8%)	0.58
Resolved, N(%)	34/140 (24%)	22/42 (52%)	8/38 (21%)	2/47 (4%)	2/13 (15%)	
Worsened, N(%)	14/140 (10%)	5/42 (12%)	7/38 (18%)	2/47 (4%)	0/13 (0%)	
Stable, N(%)	56/140 (40%)	11/42 (26%)	11/38 (29%)	30/47 (64%)	4/13 (31%)	
Improved, N(%)	10/140 (7%)	0/42 (0%)	3/38 (8%)	5/47 (11%)	2/13 (15%)	
No follow-up imaging, N(%)	15/140 (10%)	4/42 (10%)	6/38 (16%)	1/47 (2%)	4/13 (31%)	
Outcomes						
Stroke, N(%)	19/140 (14%)	5/42 (12%)	7/38 (18%)	3/47 (6%)	4/13 (31%)	0.65
Stroke-related mortality, N(%)	2/140 (1%)	2/42(5%)	0/38 (0%)	0/47 (0%)	0/13 (0%)	0.08

N number, SD standard deviation, SBP systolic blood pressure, DBP diastolic blood pressure, HR heart rate, ISS Injury Severity Score, TBI traumatic brain injury, CT computerized tomography.

Table 2
Presenting variables, radiographic progression, and outcomes for vertebral BCVI stratified by the initial BCVI grade.

	Total (n = 172)	Grade 1 (n = 47)	Grade 2 (n = 42)	Grade 3 (n = 20)	Grade 4 (n = 63)	p-value
Admission Variables						
Age, mean (SD)	52 (21)	49 (20)	54 (21)	48 (22)	55 (21)	0.32
Gender (Male), N(%)	112/172 (65%)	33/47 (70%)	25/42 (60%)	7/20 (35%)	47/63 (75%)	0.009
Admission SBP, mean (SD)	135 (36)	134 (36)	137 (24)	133 (45)	135 (35)	0.98
Admission DBP, mean (SD)	79 (20)	79 (22)	80 (16)	81 (25)	78 (20)	0.97
Admission HR, mean (SD)	87 (22)	88 (24)	90 (20)	86 (28)	84 (21)	0.52
ISS, mean (SD)	24 (16)	24 (15)	21 (11)	22 (16)	26 (18)	0.37
TBI, N(%)	56/172 (33%)	15/47 (32%)	12/42 (29%)	7/20 (35%)	22/63 (35%)	0.62
CT Evolution						
Post-endovascular intervention, N(%)	0/172 (0%)	0/47 (0%)	0/42 (00%)	0/20 (0%)	0/63 (0%)	0.01
Resolved, N(%)	53/172 (31%)	22/47 (47%)	18/42 (43%)	8/20 (40%)	5/63 (8%)	
Worsened, N(%)	9/172 (5%)	6/47 (13%)	3/42 (7%)	0/20 (0%)	0/63 (0%)	
Stable, N(%)	77/172 (45%)	15/47 (32%)	10/42 (24%)	7/20 (35%)	45/63 (71%)	
Improved, N(%)	11/172 (6%)	0/47 (0%)	5/42 (12%)	2/20 (10%)	4/63 (6%)	
No follow-up imaging, N(%)	22/172 (13%)	4/47 (8%)	6/42 (14%)	3/20 (15%)	9/63 (14%)	
Outcomes						
Stroke, N(%)	5/172 (3%)	0/47 (0%)	0/42 (0%)	0/20 (0%)	5/63 (8%)	0.009
Stroke-related mortality, N(%)	0/172 (0%)	0/47 (0%)	0/42 (0%)	0/20 (0%)	0/63 (0%)	–

N number, SD standard deviation, SBP systolic blood pressure, DBP diastolic blood pressure, HR heart rate, ISS Injury Severity Score, TBI traumatic brain injury, CT computerized tomography.

stroke formation median LS was 25% (IQR 90) and for carotid BCVI without stroke formation median LS was 20% (IQR 48) ($p = 0.56$).

On subdivision of grade 2 carotid BCVI, 20/38 (53%) had $\geq$ 25% and $\leq$ 50% LS and 18/38 (47%) had > 50% and $\leq$ 99% LS. 3/20 (15%) of grade 2 carotid BCVI with $\geq$ 25% and $\leq$ 50% LS had a stroke compared with 4/18 (22%) grade 2 carotid BCVI with > 50% and $\leq$ 99% LS ($p = 0.44$). On subdivision of grade 3 carotid BCVI, 24/47 (51%) had PSA without LS and 23/47 (49%) PSA and LS. Of grade 3 carotid BCVI with PSA without LS, 1/24 (4%) had a stroke compared with 2/23 (9%) grade 3 carotid BCVI with PSA and LS ($p = 0.48$).

Stroke rates in carotid BCVI were examined graphically. Stroke rates in carotid BCVI did not increase sequentially with increasing BCVI grade, with grade 3 carotid BCVI having the lowest stroke rate (Fig. 2, panel A). When aneurysmal and non-aneurysmal carotid BCVI were examined separately the pattern of increasing stroke rate with increasing grade was restored. Non-aneurysmal carotid BCVI had a stepwise increase in stroke rate with increasing LS (Fig. 2, Panel B). This pattern for stroke rate was also true for aneurysmal carotid BCVI, although only 2 data points are shown (Fig. 2, Panel C).

In carotid BCVI, EI was undertaken in 16/140 (14%) (Table 3). 0/42 (0%) of grade 1, 5/38 (13%) of grade 2, 9/47 (19%) of grade 3, and 2/13 (15%) of grade 4 carotid BCVI underwent EI ($p = 0.01$). In grade 2 carotid BCVI with $\geq$ 25% and $\leq$ 50% LS 1/20 (5%) underwent EI compared with 4/18 (22%) grade 2 carotid BCVI with > 50% and $\leq$ 99% LS ($p = 0.14$). Similarly, of grade 3 carotid BCVI with PSA without LS, 1/24 (4%) underwent EI compared with 8/23 (35%) grade 3 carotid BCVI with PSA and LS ($p = 0.01$)

Three carotid BCVI had both a stroke and underwent EI. One grade 2 carotid BCVI with > 50% and $\leq$ 99% LS that progressed to a grade 3 carotid BCVI with PSA and LS on serial CT imaging developed a stroke prior to stenting. Another patient with bilateral

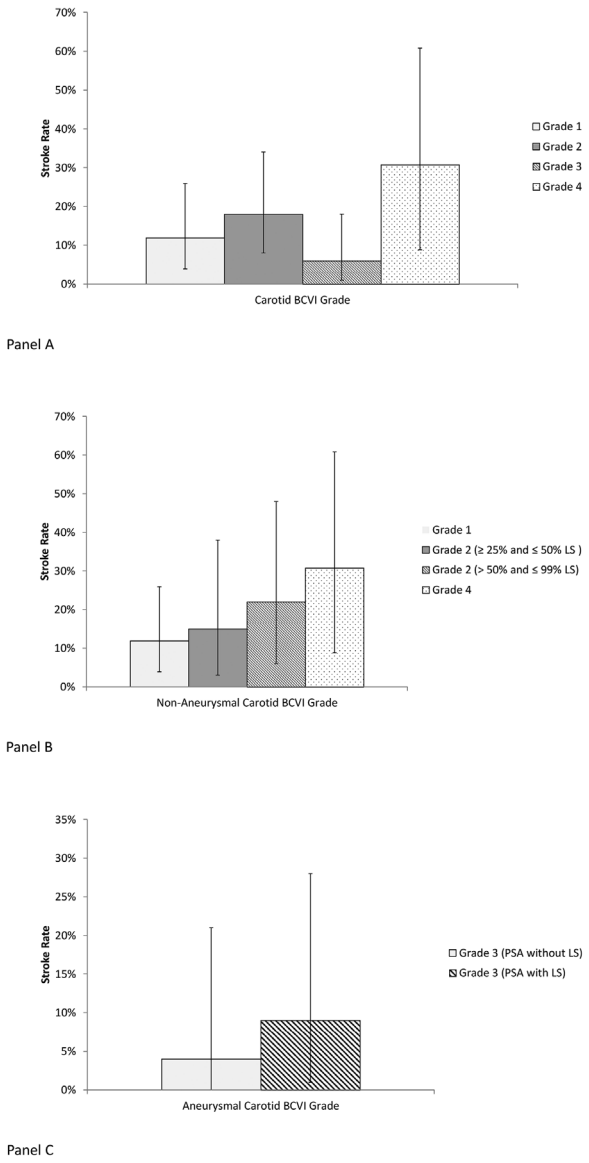

Fig. 2. Rates of stroke formation in carotid BCVI by grade (panel A), for non-aneurysmal injuries (panel B) and for aneurysmal injuries (Panel C).

grade 3 carotid BCVI with PSA and LS underwent EI for decreased cerebral perfusion and developed bilateral strokes 1 day following stenting. Overall 2/15 (13%) of BCVI undergoing stenting had a post-stenting stroke.

Discussion

The BCVI grading scale has previously shown increasing BCVI severity with higher grade [1,7]. However, multiple factors in BCVI grading make true stroke risk with LS difficult to measure. Foremost, EI may lower stroke risk by preventing strokes which otherwise would have occurred. Similarly grade 3 BCVI represents PSA with all degrees of LS and grade 2 BCVI represents ≥25% and ≤99% LS, which may hide influence of higher degrees of LS on stroke formation given these broad ranges. By accounting for such factors, this study suggests that LS influences stroke formation to a degree not reflected in the BCVI grading scale.

When examined graphically, stroke rate did not increase sequentially by BCVI grade as seen in prior studies, with a far lower rate of stroke in grade 3 carotid BCVI compared with other grades of injury. However, the direct relationship between carotid BCVI grade and stroke rate seen in prior studies was restored when examining aneurysmal and non-aneurysmal carotid BCVI separately in this manuscript. When examining aneurysmal and non-aneurysmal carotid BCVI separately, aneurysmal carotid BCVI had a higher stroke rate for carotid BCVI with LS when compared to carotid BCVI without LS. Similarly, non-aneurysmal carotid BCVI had a higher stroke rate in grade 4 injury, with sequentially lower stroke rates with decreasing grade of injury, to the lowest stroke rate in grade 1 carotid BCVI.

If stroke risk is different within subgrades on the current BCVI grading scale (of 1, 2, 3, 4, and 5), management of BCVI may need to vary within the BCVI grade. At our institution we treat grade 2 carotid (≥ 25% and ≤ 50% LS) BCVI with aspirin and Plavix® dual therapy and grade 2 carotid (> 50% and ≤ 99% LS) BCVI with anticoagulation. We treat grade 2 vertebral (≥ 25% and ≤ 50% LS) BCVI with aspirin alone and grade 2 vertebral (> 50% and ≤ 99% LS) BCVI with aspirin and Plavix®. We view grade 3 carotid BCVI with LS as a more serious injury than grade 3 carotid BCVI without LS and more strongly consider endovascular stenting. Understanding these small distinctions in BCVI characteristics may improve stroke prevention.

While the trend seen in this manuscript was for increased stroke rate with higher percentage LS in carotid BCVI, this was not statistically significant. This lack of statistical association between percentage LS and stroke rate may be due to a number of factors. Foremost, there may be no association between percentage LS and stroke formation, although the trends in stroke rates with increasing LS and our knowledge of chronic vascular disease suggests otherwise. This lack of statistical association may also be due to the low number of BCVI within categories created in this study, with decreasing number with each sequential stratification (carotid vs. vertebral BCVI, grade of BCVI, within grade stratification by LS). Thirdly, association between stroke and percentage LS may be masked by our use of EI in BCVI with higher percentage LS.

It is difficult to discuss stroke rates in BCVI without discussing use of EI, given that EI is used mostly to prevent stroke formation in high-risk BCVI. Appropriate use of EI may decrease what would have been a higher stroke rate without use of EI, and make these BCVI appear less severe than they actually are. This would also diminish ability to detect significant association between percentage LS and stroke rate.

The role of EI and indications for EI are unclear. Indications for EI in BCVI are according to expert opinion, and include decreased cerebral perfusion, stroke, worsening radiographic characteristics, and aneurysm rupture [10]. There are not specific indications for EI by injury grade. However, our use of EI is especially compelling in grade 3 carotid BCVI, which in this study had the lowest stroke rate compared to other grades and a lower stroke rate than many reported historical rates for grade 3 carotid BCVI. In general in these previously published series of grade 3 carotid BCVI, those series in which EI were used had lower stroke rates, while those series in which EI were not used had higher stroke rates [1,7,11–18] (Table 4).

However, the need for EI in BCVI is not universally accepted. Work by Burlew et al and Shahan et al have suggested that EI is not commonly necessary in BCVI and EI certainly can be used at lower rates than in earlier studies [19,20]. The 8.9% rate of stenting in grade 2 and 3 BCVI recently reported by the Memphis group is a similar rate to this study, and utilization of EI may be trending towards optimal use 20].

The benefit of EI for stroke prevention is balanced with risk of stroke with EI. The post-EI stroke rate of 13% in this study for BCVI is not negligible. What the stroke rate of BCVI undergoing EI would have been without EI is unknown, as these were severe BCVI. The 2 BCVI which developed post-EI strokes underwent EI for decreased

Table 3
Lesion evolution, development of stroke, and indication for EI in carotid BCVI undergoing EI.

BCVI Number	Endovascular Intervention Performed	Initial Carotid BCVI Grade	Carotid BCVI Grade Prior to Endovascular Intervention	Stroke	Indication for Endovascular Intervention
1	stent	2 (> 50% and ≤ 99% LS)	3 (PSA and LS)	no	Increased LS
2	stent	4	3 (PSA and LS)	no	Increase in PSA and LS
3	stent	3 (PSA and LS)	3 (PSA and LS)	no	Increase in PSA and LS
4	stent	2 (> 50% and ≤ 99% LS)	3 (PSA and LS)	no	Development of PSA
5	embolization	4	4	no	Recanalization prevention
6	stent	3 (PSA and LS)	2 (> 50% and ≤ 99% LS)	no	Increased LS
7	stent	3 (PSA and LS)	3 (PSA and LS)	no	Increased LS
8	stent	2 (> 50% and ≤ 99% LS)	3 (PSA and LS)	yes	Increased LS
9	stent	2 (≥ 25% and ≤ 50% LS)	3 (PSA and LS)	no	Development of PSA
10	stent	3 (PSA and LS)	3 (PSA and LS)	no	Increasing PSA
11	stent	3 (PSA and LS)	3 (PSA and LS)	yes	Bilateral LS and decreased cerebral perfusion
12	stent	3 (PSA and LS)	3 (PSA and LS)	yes	Bilateral LS and decreased cerebral perfusion
13	stent	3 (PSA without LS)	3 (PSA without LS)	no	Increasing PSA
14	stent	3 (PSA and LS)	3 (PSA and LS)	no	Increase in PSA and LS
15	stent	2 (> 50% and ≤ 99% LS)	3 (PSA and LS)	no	Development of PSA
16	stent	3 (PSA and LS)	3 (PSA without LS)	no	Increasing PSA

BCVI blunt cerebrovascular injury, EI endovascular intervention, LS luminal stenosis, PSA pseudoaneurysm.

Table 4
Stroke rates reported in the literature for grade 3 carotid BCVI.

Authors	Year of Publication	Number of Grade 3 Carotid BCVI Reported	Endovascular Intervention Reported	Stroke Rate Reported
Esnault P, et al	2017	2	1 with coils	0%
Morton RP, et al	2016	43	4 with stents, 1 with coils, 3 with endovascular vessel sacrifice	16%
Geddes AE, et al	2016	112	None noted	11.6%
Scott WW, et al	2015	68	7 with stents, 1 with coils	7%
Burlew CC, et al	2012	61	None noted	29.5%
Stein DM, et al	2009	43	15 with stents	9.3%
Malhotra AK, et al	2007	4	2 with coil and stent combinations	0%
Cothren CC, et al	2005	33	None noted	26%
Biffle WL, et al	2002	35 (highest grade)	Unclear number for grade 3 specifically	26%

cerebral perfusion, and EI in these BCVI does not seem avoidable. Previous reported post-EI rate of neurologic deficits was 3.5% [21]. The rate of post-EI stroke is higher in this manuscript, although the severity of BCVI undergoing EI is high as well. Standardizing indications for EI in BCVI will delineate an acceptable rate of post-EI stroke and target EI to only BCVI which will benefit from this therapy.

Grade 3 BCVI with PSA and LS appear to be a high-risk subgroup within BCVI. Given the low rate of stroke seen in grade 3 BCVI in this study combined with the high rates of EI in grade 3 BCVI with PSA and LS, we postulate that use of EI in many of these patients prevents stroke formation. However, causation cannot be assigned in this retrospective review.

This study also underscores the difference in severity between carotid BCVI and vertebral BCVI. Vertebral BCVI had a far lower stroke rate, with strokes only occurring within grade 4 vertebral BCVI. Further delineation of the severity of LS in this patient population did not appear necessary given this result.

Limitations of this study include its retrospective nature. BCVI is rare and all available patients were included, making Type II error a possibility. Additionally, the multiple subdivisions required to look at individual BCVI grades created small categories for analysis (even though all available patients were included) which resulted in percentages with wide confidence intervals. Indications for EI are not standardized at our institution, and it is possible that some BCVI underwent EI when managed by one surgeon that would have been observed by another. Both whole-body arterial phase CT and CTA are used for initial BCVI diagnosis at our institution and we did not compare whole-body arterial phase CT and CTA in this study. Previous research has shown 48% concordance between whole-body arterial phase CT, although whole-body CT detects most BCVI [9]. We also did not include long-term outcomes after discharge, which may have excluded strokes occurring after discharge. Knowledge of post-discharge stroke rates would help determine necessary duration of pharmacologic therapy and radiographic follow-up. LS measurement is per the judgement of the physician reading the CT, and has unknown reproducibility. We did not compare inter-reviewer variation for LS, and it is likely that percentage of LS reported will vary.

Additionally, the choice of 50% as the cutoff for LS in grade 2 BCVI merits discussion. This cutoff was chosen as we already delineate BCVI at this cutoff for grade 2 BCVI in our institutional guidelines. However, if a different cutoff was chosen, a significant difference in stroke formation may have been shown. Further research will need to investigate the effects of varying degrees of LS beyond this initial description.

Conclusion

LS seems to influence stroke formation beyond the current BCVI grades. Appropriate use of EI may cloud ability to detect the full

influence of LS on stroke development, as we often used EI in BCVI with higher percentage LS. This is especially true in grade 3 BCVI with combined LS and PSA, which appear to be a high-risk subgroup. Trauma practitioners should be aware of BCVI characteristics beyond the grading scale to adequately determine risk for each individual BCVI.

Meeting presentations

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