

Management of Vertebral Artery Injuries Following Non-Penetrating Cervical Trauma

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RECOMMENDATIONS

Diagnostic

Level I

- Computed tomographic angiography (CTA) is recommended as a screening tool in selected patients after blunt cervical trauma who meet the modified Denver Screening Criteria for suspected vertebral artery injury (VAI).

Level III

- Conventional catheter angiography is recommended for the diagnosis of VAI in selected patients after blunt cervical trauma, particularly if concurrent endovascular therapy is a potential consideration, and can be undertaken in circumstances in which CTA is not available.
- Magnetic resonance imaging is recommended for the diagnosis of VAI after blunt cervical trauma in patients with a complete spinal cord injury or vertebral subluxation injuries.

Treatment

Level III

- It is recommended that the choice of therapy for patients with VAI—anticoagulation therapy vs antiplatelet therapy vs no treatment—be individualized based on the patient's vertebral artery

injury, the associated injuries, and the risk of bleeding.

- The role of endovascular therapy in VAI has yet to be defined; therefore, no recommendation regarding its use in the treatment of VAI can be offered.

RATIONALE

The association of cerebrovascular insufficiency and cervical fracture was first described by Suechting et al¹ in a patient with Wallenburg's syndrome occurring 4 days after a C5-C6 fracture-dislocation. Although Schneider et al² implicated vertebral artery injury at the site of dislocation as a cause of ischemia, Gurdjian et al³ suggested that unilateral vertebral artery occlusions might be asymptomatic. Subsequent articles^{4,5,6} described larger series of patients with asymptomatic VAI after blunt cervical trauma. However, in 2000, Biffel et al⁷ published a prospective study of 38 patients with VAI diagnosed by angiography. They identified more frequent strokes in patients not initially treated with intravenous heparin anticoagulation despite an initially asymptomatic VAI. Fractures through the foramen transversarium, facet fracture-dislocation, or vertebral subluxation are almost always seen in patients with VAI.⁵⁻¹¹ A cadaveric study¹² demonstrated progressive vertebral occlusion with greater degrees of flexion-distraction injury, confirming this clinical observation.

In 2002, the guidelines author group of the Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons reviewed the medical evidence on this topic, and produced and published a guideline on The Management of Vertebral Artery

ABBREVIATIONS: BCVI, blunt cerebrovascular injuries; DSA, digital subtraction angiography; PPV, positive predictive value; NPV, negative predictive value; VAI, vertebral artery injury

Injuries after Non-penetrating Cervical Trauma.¹³ The current review was undertaken to update the medical evidence on the diagnostic and treatment recommendations for VAI after blunt cervical trauma. Specific questions that were addressed include: the clinical and radiographic criteria used to prompt diagnostic evaluation, appropriate diagnostic tests for identifying VAI, the treatment of VAI (observation compared to anticoagulation with heparin or to aspirin therapy), and the potential role of endovascular techniques for patients with VAI.

SEARCH CRITERIA

A National Library of Medicine (PubMed) computerized literature search of publications from 1966 to 2011 was performed using the following headings: vertebral artery injury, vertebral artery dissection, cervical fracture, and cervical dislocation. The search was limited to the English language and human subjects and identified 2226 citations. The titles and abstracts of these references were reviewed to determine relevance. Isolated case reports, small case series, editorials, letters to the editor, and review articles were eliminated. The bibliographies of the resulting full-text articles were searched for other relevant citations. A total of 37 articles met inclusion criteria and 21 key citations are summarized in Evidentiary Table format.

SCIENTIFIC FOUNDATION

Diagnosis

The diagnosis of VAI can be made with a variety of imaging studies. Angiography has been the traditional “de facto” gold standard imaging technique utilized to diagnose VAI, and was used for most patients in the studies reviewed for the 2002 guidelines publication on the Management of Vertebral Artery Injuries.¹³

Biffi et al⁷ reported the largest prospective study using angiography in 2000, selecting patients from 7205 blunt trauma victims using specific clinical and radiographic criteria, subsequently known as the Denver Screening criteria. “Symptomatic” patients were selected for angiography if they had facial hemorrhage (bleeding from mouth, nose, ears), cervical bruit (in those younger than 50 years of age), expanding cervical hematoma, cerebral infarction by computed tomography (CT), or lateralizing neurological deficit. “Asymptomatic” patients were selected for angiography if they had cervical hyperextension/rotation or hyperflexion injuries, closed head injury with diffuse axonal injury, near hanging, seat belt or other soft tissue injuries to the neck, basilar skull fractures extending into the carotid canal, and cervical vertebral body fractures or distraction injuries. Between 350 and 400 angiograms were performed, identifying 38 patients with VAI. However, neither the exact number of angiograms performed nor the number of patients meeting the various criteria without VAI were reported. As a result, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of the selection criteria could not be

determined. Cervical spine injuries were observed in 27 of 38 patients with VAI, including fractures through the foramen transversarium in 4, facet dislocations in 6 patients, vertebral subluxations in 2, and more than 1 of these injuries in 2 patients. Twenty-nine patients had unilateral VAI (18 left, 11 right); 9 had bilateral VAI. A vascular injury scale was used to stratify patients into 5 categories:

Grade I - arterial dissections with less than 25% luminal narrowing.

Grade II - arterial dissections with more than 25% luminal narrowing.

Grade III - pseudoaneurysm of the vertebral artery.

Grade IV - occlusion of the vertebral artery.

Grade V - vertebral artery transection.

Seven patients died, of whom 5 had bilateral VAI (Grade I), and 2 had unilateral VAI (1 Grade I, 1 Grade IV). Three patients with either no neurological deficit or mild deficit had bilateral VAI. The authors concluded that stroke incidence and neurological outcome appeared independently of the grade of vertebral artery injury.

Another prospective study by Willis et al⁶ in 1994 identified 30 patients with midcervical fractures and/or dislocation injuries considered criteria for angiographic assessment. However, only 26 patients who met the criteria agreed to proceed with angiography. Twelve patients sustained VAI demonstrated by angiography (6 left occlusion, 3 right occlusion, 1 left intimal flap, 1 left pseudoaneurysm, and 1 left dissection). The authors provided sufficient data regarding the presence of foramen transversarium fracture, facet dislocation, and subluxation to determine the utility of these radiographic findings in identifying patients with VAI. The calculated sensitivity, specificity, PPV, and NPV of foramen transversarium fracture, facet dislocation, and vertebral subluxation as criteria for VAI are listed in Table 1. Any combination of foramen transversarium fracture, facet dislocation, and/or vertebral subluxation revealed a calculated sensitivity for identifying VAI of 92% and a specificity of 0%. The positive predictive value of the presence of any of the 3 criteria and VAI was 44%. The negative predictive value was 50%.

Alternatively, magnetic resonance angiography (MRA) has been used as a noninvasive means to diagnose VAI. Weller et al¹⁰ prospectively examined 12 patients with nonpenetrating cervical trauma who sustained fractures through the foramen transversarium. Three patients had unilateral vertebral artery occlusion

TABLE 1. Accuracy of Potential Imaging Indicators of Vertebral Artery Injury

	Sensitivity %	Specificity %	PPV %	NPV %
Foramen transversarium fracture	58	36	44	50
Facet dislocation	42	57	45	53
Vertebral subluxation	67	29	80	50

and 1 had focal narrowing, all at the site of fracture. MRA was not performed on the 26 patients without these fractures. In 1997, Giacobetti et al⁸ prospectively evaluated all patients admitted with cervical spine injuries with MRA. Twelve of 61 patients had vertebral artery occlusion demonstrated by MRA and all injuries were unilateral (6 left, 6 right). Although 7 of 12 patients with VAI had flexion-distraction injuries with facet dislocations, the types of cervical spinal injuries sustained by the 49 patients with normal MRA were not reported. Since none of these 4 articles⁸⁻¹¹ provided sufficient information regarding the types of injury and results of vertebral artery imaging in the entire population of patients studied, sensitivity, specificity, positive predictive value, and negative predictive value of the injury types could not be determined. All 4 provide Class III medical evidence on the value of MRA in the diagnosis of VAI.

In 1995, Friedman et al⁵ prospectively examined 37 patients admitted with “major” blunt cervical spine injuries using MRA and compared these patients with a size-matched control group of patients without a history of cervical trauma (Table 2). Nine patients had VAI (6 unilateral occlusion, 2 narrow, 1 bilateral injury). Both vertebral arteries were visualized in all 37 control subjects. Complete spinal cord injuries were observed in 12 of 37 patients with cervical trauma, 6 of whom had VAI ($P < .02$; chi-square test). More than 3 millimeters of vertebral subluxation was observed in 13 of the 37 patients, 5 of whom had VAI ($P < .14$; chi-square test).

Friedman et al’s report provides Class I medical evidence for the presence of VAI in association with complete spinal cord injury and with cervical vertebral subluxation, and yet provides Class III medical evidence on the ability of MRA to diagnose VAI after blunt trauma.

Other diagnostic modalities have also been used to identify VAI. CT with intravenous contrast demonstrated a unilateral vertebral artery occlusion in 1 patient with a Jefferson fracture, which was subsequently confirmed by angiography.¹⁴ Duplex sonography has also been used to diagnose VAI.¹⁵⁻¹⁷ Angiography has occasionally been used to confirm the results of MRA or ultrasonography, but there has not been a study comparing ultrasonography with angiography in the diagnosis of VAI.

The use of CTA as a screening tool for VAI has expanded exponentially since the original guideline on the management of vertebral artery injuries was published in 2002. In 2002, Miller et al¹⁸ prospectively compared catheter angiography screening in 143 patients with suspected blunt cerebrovascular injuries (BCVI) to CTA and MRA in selected (but not all) patients. They noted that CTA identified 53% of VAI found on catheter angiography. MRA identified 47% of VAI confirmed by catheter angiography.

In 2006, Eastman and colleagues prospectively compared CTA to catheter angiography as a screening tool for vascular injuries in the neck in 146 trauma patients who met the Modified Denver Screening Criteria (Table 3).¹⁹

They determined that the sensitivity, specificity, and positive and negative predictive value of CTA for blunt cervical vascular

injury were 97.7%, 100%, 100%, and 99.3%. They concluded that CTA had an accuracy of 99.3% for a cervical vascular injury following blunt trauma. For patients meeting the Modified Denver Screening Criteria, CTA has almost 100% accuracy. Their report provides Class I medical evidence on the utility of CTA to diagnose vascular injuries. CTA can, therefore, be considered the new gold standard reference test for VAI in patients who have sustained blunt trauma.

In 2006, Biffi et al²⁰ adopted a “liberal screening protocol” for BCVI, and screened 331 trauma patients with CTA. They identified 20 vascular injuries in 18 patients with CTA (5.4%) confirmed by angiography, of which 11 were carotid injuries and 9 were vertebral artery injuries (2.4% incidence of VAI). None of the patients who had normal CTAs went on to develop clinical evidence of vascular injury, providing supportive evidence for CTA as an accurate and reliable screening tool for VAI.

Utter et al²¹ studied 372 trauma patients who underwent screening CTAs and noted a 16% incidence of vertebral artery and a 10% incidence of carotid artery injuries. Digital subtraction angiography (DSA) was performed in 82 patients. Their 2002 retrospective review identified concordance between CTA and DSA in 80 of the 82 patients; 1 had artifact on CTA images that hindered diagnosis, and the second patient had a previously existing vascular anomaly not related to trauma. The authors concluded that CTA is accurate and supplants DSA as a screening tool for VAI.

In 2006, Berne and colleagues reported their experience with screening CTA for BCVI in 435 trauma victims.²² They noted a much lower incidence of vascular injury: 1.2% of all blunt trauma patients and 5.5% among trauma patients who met the modified Denver (Biffi) Screening Criteria and had CTAs performed. They reported that no patient with a negative CTA went on to develop symptoms or signs of a missed vascular injury. That same year, Schneiderei et al²³ evaluated 1313 blunt trauma patients. One hundred thirty-seven CTA studies were performed. The incidence of blunt vascular neck injuries in their series was 1.4%. Only 23 patients underwent angiography to confirm or refute the CTA findings. The calculated sensitivity and specificity of CTA to detect a vascular injury in this study was 65% and 50%, respectively. No attempt was made to define the accuracy of CTA to identify isolated vertebral artery injuries after trauma. Like the report of Berne et al,²⁴ no patient with a negative CTA developed symptoms or signs of a missed vascular injury. The authors concluded that CTA is an effective means to assess blunt vascular injuries after trauma.

In 2007, Malhotra and colleagues studied 92 blunt trauma patients who underwent both CTA and DSA for potential BCVI.²⁵ They calculated that CTA had sensitivity, specificity, and positive and negative predictive values of 74%, 86%, 65%, and 90%, respectively. Of 119 patients who underwent screening CTA, 3 patients refused consent for angiography and 24 patients were excluded from DSA due to the risk of contrast nephropathy. The authors concluded that CTA is less accurate than DSA for the identification of VAI and could not reliably be used to

TABLE 2. Accuracy of Potential Clinical and Imaging Indicators of Vertebral Artery Injury

	Sensitivity %	Specificity %	PPV %	NPV %
Complete spinal cord injury	67	79	50	88
Vertebral subluxation	56	71	38	83

exclude a blunt vascular injury. Of note, 20% of the CTAs in this study were suboptimal and uninterpretable, a finding not reported in other studies. The loss of 27 patients for consent/nephropathy concerns and the loss of 20% of the CTA studies performed due to poor image quality reduced the quality of the medical evidence the authors offered from Class I to Class III. Despite this, the negative predictive value of 90% that the authors calculated in their study supports the value of a negative CTA in indicating the absence of a clinically significant VAI.

Catheter angiography remains the practical “de facto” gold standard for diagnosis of vertebral artery injury after trauma. However, catheter angiography is an invasive and labor-intensive procedure, is not always readily available, and has a low, but finite risk. CTA, on the other hand, is non-invasive, easily performed, readily available, and has lower risk than catheter angiography. CTA has been compared to catheter angiography in patients who have sustained trauma and who meet screening criteria for suspicion of blunt vascular injury. As discussed above, there is Class I and supportive Class III medical evidence documenting the accuracy, and specifically, the negative predictive value, for the use of CTA as a screening tool for the assessment of patients with potential VAI. The low incidence of VAI after blunt cervical trauma and the relatively benign natural history of a documented traumatic vertebral artery injury, however, raise the question of the utility of screening all asymptomatic blunt trauma patients with the potential of VAI. For these reasons, it is recommended that CTA to assess for the potential of traumatic VAI be used on

TABLE 3. Modified Denver Screening Criteria for BCVI^{a,b}

Lateralizing neurologic deficit (not explained by CT head)
• Infarct on CT head scan
• Cervical hematoma (nonexpanding)
• Massive epistaxis
• Anisocoria/Homer’s syndrome
• Glasgow Coma Scale score <8 without significant CT findings
• Cervical spine fracture
• Basilar skull fracture
• Severe facial fracture (LeForte II or III only)
• Seatbelt sign above clavicle
• Cervical bruit or thrill

^aAdapted from: Biffi WL, Moore EE, Offner PJ, et al. Optimizing screening for blunt cerebrovascular injuries. *Am J Surg*, 178:517 to 522, 1999.³⁷

^bCT, computed tomography.

a selective basis. Catheter angiography remains a valuable diagnostic tool for the detection of VAI in selected patients based on Class III medical evidence, particularly if concurrent endovascular therapy is a potential consideration. The evidence used to develop these recommendations is shown in Table 4.

Treatment

Traumatic VAI of any injury grade has the potential to cause distal posterior circulation ischemia or stroke. For this reason, anticoagulation or antiplatelet therapy to reduce the risk of stroke following known VAI must be considered. Traumatic VAI occurs in multiple injured trauma patients and is more likely to occur in association with the most severe cervical spine and spinal cord injuries—all of which represent relative contraindications to anticoagulation and antiplatelet therapies in the treatment of potential posterior distribution stroke. For these reasons, multiple investigators have examined treatment options for trauma patients with VAI including anticoagulation with heparin, oral antiplatelet agents, and observation alone.^{5-7,11,18,20,22,26-28}

The previous iteration of the medical evidence-based Guideline¹³ on this subject, published in 2002, failed to identify Class I or Class II medical evidence in support for the various treatment strategies for traumatic VAI, or for any treatment at all. The previous review did identify a 31% incidence of complications ascribed to intravenous heparin therapy (anticoagulation) in the literature on the treatment of VAI, 6 of which (14%) were significant hemorrhages.

The current review identified 10 contemporary citations in which the treatment of VAI after trauma was investigated. All 10 provide Class III medical evidence on this topic. It appears that many of the strokes attributable to VAI occur at the time of injury and are identified during the initial work up. A common pattern in the literature, identified by Fusco and Harrigan,²⁹ is that these patients are typically included in the “no treatment group” because their ischemic event occurred prior to treatment. The “no treatment group,” including those patients with early stroke, is then compared to the “treatment group,” a population not burdened with early stroke and/or death. This distorted logic and prejudicial assignment strategy limits the ability to identify a consistent or scientifically valid treatment strategy for VAI.²⁹

In 2000, Biffle et al⁷ identified 38 patients with blunt traumatic VAI by catheter angiography. They reported 9 patients who had posterior circulation strokes (24%) during the course of their study. There was no correlation between vertebral artery injury grade and stroke. The authors did not describe the incidence of stroke from the initial injury, identified at the time of the diagnostic workup. They did report 3 patients who had stroke before treatment who were subsequently treated with intravenous heparin. They described 3 asymptomatic patients treated with heparin to prevent stroke who went on to develop a posterior circulation stroke during anticoagulation therapy. They offered no details on the 3 other patients with stroke. They described 21 asymptomatic patients treated with heparin. Three had a stroke (14%). Conversely, 6 of 17 patients (35%) suffered a stroke

before or without heparin therapy, but the relationship of the stroke to the initial injury was not explained. Two patients sustained hemorrhagic strokes while receiving heparin therapy.

Miller et al, in 2001, identified 75 patients with blunt traumatic carotid artery injuries and 50 patients with blunt traumatic VAI with 4 vessel angiography.³⁰ Six patients with VAI presented with posterior ischemia/stroke. Sixty-four percent were identified following BCVI screening protocols. Thirty-nine asymptomatic patients with VAI were treated (31 heparin, 8 aspirin). The incidence of posterior circulation stroke after treatment was 2.6% (1 patient assumed, but not stated, to be on aspirin therapy). The authors described 5 patients with complications related to heparin therapy among 65 treated with heparin (8%), but did not select out those with VAI. They erroneously reported a 54% stroke rate for untreated VAI. The authors concluded that anticoagulation therapy is effective for acute traumatic VAI. This study provides Class III medical evidence for the treatment of VAI with either heparin or aspirin, but offers no comparison to a “no treatment” group.

In 2002, Miller et al¹⁸ described 43 patients with VAI diagnosed by digital subtraction angiography. Patients were treated with heparin anticoagulation (n = 8), aspirin only (n = 24), aspirin and clopidogrel (n = 8), or no treatment (n = 3). No patient experienced a posterior circulation stroke in the 2-year patient accrual period with limited follow up. They compared these results to their previously published cohort³⁰ (see above). Severe hemorrhagic complications were noted in 2 patients receiving anticoagulation. One patient on aspirin-only therapy developed bleeding from a gastric ulcer. The authors concluded that asymptomatic patients with VAI should be treated with systemic anticoagulation, this despite a higher hemorrhagic risk with the use of heparin in both of their series, and the absence of a future stroke in patients who were assigned to “no treatment.” Their report offers Class III medical evidence on this issue.

Beletsky et al³¹ described 116 patients with cervical arterial dissections, 67 vertebral, and 49 carotid artery lesions. In their 2003 report, 68 injuries were due to blunt cervical trauma. In the 105 patients with complete follow up, the stroke rate was 8.3% for those treated with intravenous anticoagulation vs 12.4% among patients treated with aspirin (not statistically significant, $P = .63$). The authors did not select out or separately report treatment strategies, numbers of patients, or stroke incidence in patients with isolated VAI.

In 2006, Schneidereit et al²³ performed 137 CTA studies in evaluation of blunt vascular neck injuries after trauma. They found an incidence of blunt vascular neck injuries of 1.4%. Thirteen patients with VAI were treated with anticoagulation (3), antiplatelet therapy (4), endovascular treatment (3), endovascular and antiplatelet therapy (2), or no treatment (1). No patient had neurological sequelae due to VAI or a complication of treatment. Length of treatment and follow up were not specified. The Beletsky et al and Schneidereit et al studies offer Class III medical evidence on treatment for VAI. No treatment recommendations can be derived from either study.

In 2009, Eastman et al³² published a follow-up study to their 2006 study documenting the merits and accuracy of CTA to

identify BCVI, including VAI following blunt trauma. They identified 19 VAI patients in their contemporary cohort. Nine patients were treated with antiplatelet therapy, 3 patients with anticoagulation, 1 with embolization, and 6 patients received no treatment. One of the 19 patients had a stroke (5.3%), a patient with multiple associated injuries who received no treatment until the stroke occurred and then was treated with antiplatelet therapy thereafter. No other data are provided. In comparison, the stroke rate following VAI in their earlier cohort when catheter angiography was used to diagnose BCVI was 18.2%. The authors concluded that CTA in the contemporary workup of patients with BCVI reduces the time to diagnosis, and subsequently time to treatment, reducing the effective stroke rate following BCVI including VAI. This report provides Class III medical evidence on treatment for VAI. No specific treatment recommendations can be offered from the medical evidence provided in their study.

Berne and Norwood reported on blunt injuries to the vertebral artery (BVI) in 2009.²² Forty-four patients out of 8292 admissions following blunt trauma were found to have BVI diagnosed by CTA. Two patients were treated with anticoagulation, 19 were treated with aspirin only, 2 with dual antiplatelet agents (clopidogrel and aspirin), 10 were treated with endovascular therapy \pm antiplatelet agents, and 11 patients received no treatment. Four patients developed a stroke and 3 of those 4 patients died. The overall mortality in their series was 16% (7 of 44 patients), but BVI mortality was identified in only the 3 patients described above. These patients were the most severely injured patients, 2 with bilateral VA occlusion, and 1 with a VA transection. The authors concluded that despite an aggressive screening and individualized treatment protocol for BVI, they had very few potentially preventable BVI-related strokes and deaths. They were unable to conclude that either screening or treatment of any kind improved outcome from BVI. No recommendations on treatment could be derived from their report.

Cothren et al²⁶ in 2009 described a retrospective review of a prospective database in comparison of anticoagulation and antiplatelet agents in the treatment of BCVIs. Two-hundred eighty-two asymptomatic patients were treated with heparin (192), aspirin (67), or aspirin and/or clopidogrel (23). One hundred seven asymptomatic patients with BCVI were not treated. The reported stroke rate in the treated group was 0.5%. The stroke rate in the “no treatment” group was 21.5%. The authors offer Class II medical evidence in favor of treatment for BCVIs. Regrettably, the authors did not offer specifics on the timing or the significance of “stroke” in the 10 “asymptomatic” VAI patients found to have a stroke in the “no treatment” group. Several of these patients had incidental, asymptomatic imaging findings of stroke on follow-up CT studies. Late comparative imaging to assess for silent stroke/asymptomatic stroke was not routinely accomplished in the vast majority of study patients (>80%). Follow-up of patients beyond discharge was “limited.” For these reasons and others, this report offers Class III medical evidence on the treatment of VAI after injury. The authors did report serious bleeding complications in 8 patients treated with heparin, adding to the body of evidence that heparin therapy after

TABLE 4. Evidentiary Table: Diagnosis of Vertebral Artery Injury^a

Malhotra et al, ²⁶ <i>Ann Surg</i> , 2007	Prospective comparison 92 trauma patients who underwent both CTA and DSA.	III	Sensitivity, specificity, positive and negative predictive values of CTA were 74%, 86%, 65%, and 90%, respectively. No accuracy data for VAI specifically.
Schneiderei et al, ²⁴ <i>J Trauma</i> , 2006	Prospective study of CTA to identify blunt vascular neck injuries after trauma	III	Sensitivity, specificity, positive and negative predictive values of CTA were 65%, 50%, 65% and 94%, respectively. No accuracy data provided for VAI specifically.
Berne et al, ²⁵ <i>J Trauma</i> , 2006	Retrospective review of 435 patients who underwent CTA for suspected BCVI.	III	1.2% incidence of BCVI among all trauma victims. 5.5% incidence among those meeting Biffle criteria. None of the patients with normal CTA went on to have signs/symptoms of missed injury.
Utter et al, ²² <i>J Am Coll Surg</i> , 2006	Retrospective review of 82 patients with normal CTA who underwent confirmatory DSA.	III	92% negative predictive value for CTA. Transverse foramen fractures most predictive of vertebral artery injury.
Biffel et al, ²¹ <i>J Trauma</i> , 2006	Prospective review of 331 patients who underwent CTA for suspected BCVI.	III	5.4% incidence of BCVI, specifically 2.4% incidence of VAI among those who met screening criteria. None of the patients with normal CTA went on to have signs/symptoms of missed injury.
Eastman et al, ²⁰ <i>J Trauma</i> , 2006	Prospective comparison of CTA and catheter angiography in 146 trauma patients.	I	Sensitivity, specificity, positive predictive value, and negative predictive value of CTA were 97.7%, 100%, and 100%, respectively. Accuracy of CTA for VAI was 99.3%.
Miller et al, ¹⁹ <i>Ann Surg</i> , 2002	Prospective nonrandomized comparison of CA to CTA and MRA screening in 216 trauma patients.	III	Sensitivity of CTA and MRA were 53% and 47%, respectively.
Weller et al, ¹¹ <i>J Trauma</i> , 1999	Prospective MRA in 12 patients with foramen transversarium fractures.	III	Three of 12 had VA occlusion; all remained asymptomatic on aspirin. One of 12 with stenosis had delayed syncope on aspirin, resolved with brief intravenous heparin followed by aspirin.
Giacobetti et al, ⁹ <i>Spine</i> , 1997	Prospective study with MRA in 61 patients with cervical injuries found 12 patients with VA occlusion.	III	One of 4 with transverse foramen fractures had occlusion. Six of 15 with facet dislocation had occlusion. Three of 12 with transient blurred vision resolved with 3-month anticoagulation.
Friedman et al, ⁶ <i>AJR Am J Roentgenol</i> , 1995	Prospective study of 37 patients with nonpenetrating cervical trauma found 9 VA injuries by MRA.	III	Fifty percent of patients with complete cord injuries had VA injury vs 12% of patients with incomplete cord injuries ($P < .02$). Five of 13 patients with >3 mm subluxation had VA injuries vs 4 of 24 patients with <3 mm subluxation. One patient with bilateral VA injuries died of large cerebellar infarct (bilateral foramen transversarium fractures). Eight asymptomatic (1 of 8 with anticoagulation also had carotid occlusion).
Woodring et al, ¹² <i>J Trauma</i> , 1993	Retrospective study of 216 patients with cervical fractures showed 52 with TP fractures. Eight had angio.	III	Seventy-eight percent of TP fractures extended into foramen transversarium. Four of 8 patients had occlusion, 3 of 8 had dissection, 1 of each had stroke that improved with anticoagulation. Three asymptomatic patients treated with anticoagulation.

^aBCVI, blunt cerebrovascular injuries; CA, contrast angiography; CTA, computed tomographic angiography; DSA, digital subtraction angiography; MRA, magnetic resonance angiography; TP, transverse process; VAI, vertebral artery injury.

TABLE 5. Evidentiary Table: Treatment of Vertebral Artery Injury^a

Citation	Description of Study	Evidence Class	Conclusions
Franz et al, ²⁸ <i>Vascular & Endovascular Surgery</i> , 2010	Retrospective follow up of 29 BCVI patients, 24 with VAI after discharge.	III	No neurological sequelae in any patient with follow up.
	Treatment for VAI included anticoagulation (6), antiplatelet (6), anticoagulation + antiplatelet (10), and no treatment (10). Twelve patients with VAI with follow-up mean 9.2 weeks. Specifics of their treatment not reported.		No complications of therapy.
Stein et al, ²⁷ <i>J Trauma</i> , 2009	Retrospective study of 147 pts with BCVI treated with endovascular management, antiplatelet agents, anticoagulants, a combination, or no treatment.	III	Significantly higher risk of stroke with no treatment BCVI (25.8% vs 3.5%).
			Stroke rate of VAI = 8.2%, but 2 had stroke after initial injury, 3 others incidental or asymptomatic.
	Sixty-eight patients with VAI.		One-third of patients not candidates for treatment.
			Treatment appears to reduce stroke risk for BCVI, but no therapy recommendations offered for VAI.
Cothren et al, ²⁶ <i>Ann Surg</i> , 2009	Retrospective comparison in 282 asymptomatic BCVI patients treated with heparin, aspirin, and aspirin + plavix, vs no treatment.	II for BCVI, III for VAI	Significantly higher rate of stroke for BCVI with no treatment (21.5% vs 0.5%).
			Equivalence between anticoagulation and antiplatelet regimens.
			No routine follow-up screening and limited follow up.
			No specifics offered for VAI. Increased bleed complications with heparin.
Berne and Norwood, ²² <i>J Trauma</i> , 2009	Forty-four patients with VAI by CTA out of 8292 admits.	III	Aggressive screening and individualized treatment failed to prevent VAI stroke and death.
	Two treated with anticoagulation, 19 aspirin, 2 dual antiplatelet, 10 with endovascular/antiplatelet, and 10 no treatment.		No recommendations on treatment for VAI.
Eastman et al, ¹⁸ <i>J Trauma</i> , 2009	Four strokes from VAI, 3 on admission with most severe injuries.	III	CTA to diagnose BCVI (including VAI) reduces time to diagnosis and treatment.
	Follow-up study on CTA for BCVI including 19 patients with VAI.		No evidence in support of treatment recommendations offered for VAI.
	Stroke rate for BCVI = 15.2% in prior study with DSA to diagnose BCVI.		
	Stroke rate for BCVI = 3.8% with CTA to diagnose BCVI.		
Schneiderei et al, ²³ <i>J Trauma</i> , 2006	Nine VAI patients treated with antiplatelet, 3 with anticoagulation, 1 endovascular, and 6 no treatment.	III	No conclusive treatment recommendations for VAI offered.
	One stroke (5.3%)		
	137 CTA studies to assess for blunt vascular neck injuries.		
	Incidence 1.4%.		

(Continues)

TABLE 5. Continued

Citation	Description of Study	Evidence Class	Conclusions
	Thirteen patients with VAI treated with anticoagulation (3), antiplatelet (4), endovascular (3), endovascular and antiplatelet (2), and no treatment (1). No neurological sequelae from VAI. Length of treatment and follow up not specified.		
Beletsky et al, ³¹ <i>Stroke</i> , 2003	Nonrandomized comparison of aspirin and anticoagulants in 116 patients with traumatic and atraumatic dissection BCVI injuries. Stroke rate in 105 patients with follow up = 8.3%.	III	Rate of stroke with ASA was 12.4% vs 8% with anticoagulation (not statistically significant, <i>P</i> = .63). No conclusive treatment recommendations for VAI offered.
Miller et al, ¹⁸ <i>Ann Surg</i> , 2002	No specific data on patients with traumatic VAI. Prospective screening identified 43 patients with VAI diagnosed by DSA, treated with anticoagulation (8), aspirin (24), aspirin and clopidogrel (8), or no treatment (3). No stroke in variable follow-up period. Two bleeding complications with heparin.	III	Authors favor treatment of VAI with anticoagulation despite increased risk of bleeding complications and absence of stroke with other treatments including “no treatment.”
Miller et al, ³⁰ <i>J Trauma</i> , 2001	Retrospective analysis of prospective database on screening for BCVI. Seventy-five blunt carotid injury patients. Fifty patients with VAI. Six VAI patients presented with stroke.	III	Authors concluded anticoagulation is effective treatment for VAI despite increase in complications. No comparison with “no treatment” group.
Biffi et al, ⁷ 2000, <i>Ann Surg</i>	Thirty-nine asymptomatic VAI patients treated with heparin (31) or aspirin (8). One posterior circulation stroke (2.6%) while on aspirin. Five hemorrhagic complications with heparin. Prospective angiography screening for BCVI identified 38 patients with VAI. Nine patients with postcirculation stroke (24%).	III	Three of 21 asymptomatic patients treated with heparin had stroke (14%) vs 6 of 17 patients without heparin had stroke (35%) (not statistically significant, <i>P</i> = .13). Three strokes from initial injury. Three strokes on heparin therapy, 2 of which were hemorrhagic. Three strokes, no data offered.

^aBCVI, blunt cerebrovascular injuries; CTA, computed tomographic angiography; DSA, digital subtraction angiography; VAI, vertebral artery injury.

BCVI has higher risk than that associated with antiplatelet therapy in the treatment of BCVI.

Stein et al,²⁷ also published in 2009, reported on 147 patients with BCVI after trauma. Sixty-eight of these patients sustained VAI, 5 of whom had posterior circulation strokes (8.2%). Two of these patients had stroke from VAI at the time of imaging/diagnosis (both died). The other 3 patients had “asymptomatic”

incidental strokes identified on follow-up imaging while hospitalized. The 2 early fatal strokes were counted in the “no treatment” group. The true incidence of “asymptomatic stroke” could not be discerned from the study because routine, follow-up surveillance of all study patients was not accomplished. Treatment groups in Stein et al’s study included anticoagulation (n = 8), antiplatelet agents (n = 23), endovascular therapy (n = 12), endovascular and

antiplatelet therapy (n = 4), and no treatment (n = 21). Complications of therapy were not offered. The authors concluded that nearly one-third of the patients with BCVI are not candidates for treatment. In their experience, treatment appeared to reduce the risk of stroke following BCVI, but could not offer a specific recommendation on treatment for BCVI once it is identified.

In 2010, Franz et al²⁸ described retrospective follow up of 29 BCVI patients following discharge from their initial blunt trauma injury hospitalization. Twelve of 24 patients who sustained acute traumatic VAI returned for follow-up assessment, with a mean follow up of 9.2 weeks following discharge. Therapy for the original 24 patients with VAI included anticoagulation (6), antiplatelet therapy (4), anticoagulation and antiplatelet therapy (10), and no treatment (4). No patient seen in follow up had neurological sequelae attributable to VAI. There were no reported complaints or complications of therapy.

In several studies, patients with VAI were reimaged to determine whether disease progression or resolution occurred after treatment for vertebral artery injury. Biffel et al⁷ reported follow-up angiography on 21 patients. Of 16 patients treated with heparin, 2 improved to a lesser grade of vascular injury and 4 worsened to a poorer grade. Of 5 patients not receiving heparin, 1 improved and 3 had worse vascular injury grades. Vaccaro et al³³ found reconstitution in 1 of 6 vertebral artery injuries by MRA 12 days after the original diagnosis. This patient was not treated with anticoagulation.³³ The remaining 5 still had vertebral artery occlusion more than 1 year later, including 2 treated with anticoagulation. Willis et al⁶ described the results of follow-up angiography in 3 patients with VAI. One patient with a pseudoaneurysm received 1 week of intravenous heparin followed by aspirin; the pseudoaneurysm had slightly enlarged 7 days after treatment was begun, but had disappeared on angiography performed 6 weeks later. One patient treated with intravenous heparin for a vertebral artery dissection had an asymptomatic occlusion of the artery demonstrated by angiography 2 days later; the heparin was subsequently discontinued. The third patient was treated with intravenous heparin for a vertebral artery intimal flap. That patient had a normal vertebral arteriogram 10 days later. Thibodeaux et al¹⁷ found a patent vertebral artery 6 months after a VAI dissection was diagnosed; this patient was not anticoagulated. Sim et al¹⁶ reported delayed Duplex sonography in 11 patients with a history of facet dislocation, but unknown vertebral artery status at the time of the original cervical spine injury. Two of these studies demonstrated VAI: 1 with persistent cervical spinal dislocation had vertebral occlusion, and 1 patient with a reduced cervical injury had vertebral artery stenosis. Stein and colleagues found that of the treated VAI patients in their series, 23.5% were radiographically improved and 76.5% were stable at early follow up, as compared to 66.7% improved and 33.3% stable in the untreated group. They concluded that few VAI lesions progress and most improve radiographically, regardless of whether or how they are treated.²⁷

More recently, endovascular intervention has been described for the management of blunt cerebrovascular injury, particularly in

cases of traumatic pseudoaneurysm, dissection, and fistulae.³⁴⁻³⁶ However, the need for dual antiplatelet therapy after endovascular procedures and their potential for bleeding complications is a relative contraindication to the application of endovascular therapy in multiple injury trauma patients with VAI. The evidence used to develop these recommendations is shown in Table 5.

SUMMARY

The incidence of vertebral artery injury may be as high as 11% after nonpenetrating cervical spinal trauma in patients meeting specific clinical and physical exam criteria. The modified Denver Screening Criteria for BCVI are the most commonly used.^{19,37} Many patients with VAI have complete spinal cord injuries, fractures through the foramen transversarium, cervical spinal facet dislocation injuries, and/or vertebral subluxation, but many patients with these spinal and spinal cord injuries have normal vertebral arteries when imaged, thus reducing the specificity of these injury patterns with respect to VAI. Many comparative studies in which sensitivity, specificity, and positive and negative predictive value have been, or can be, calculated examined various tests against each other, but not against the gold standard of intravenous catheter angiography, thereby producing Class III medical evidence. However, recent literature providing Class I medical evidence does support CTA as a highly accurate alternative to catheter angiography for screening for VAI in blunt injury trauma patients, with a very high negative predictive value.^{19,21,24}

It appears that a significant number of the symptomatic strokes resulting in neurological deficits following VAI are attributable to the initial blunt traumatic injury. The majority of patients with VAI are asymptomatic, including a number of patients with incidental cerebellar and posterior circulation strokes found on imaging studies at the time of diagnosis or in follow-up. To date, there has been no definitive longitudinal study defining the stroke risk of VAI, asymptomatic or otherwise, among patients being treated for known VAI and/or among patients receiving “no treatment” for known VAI. There is no Class I or Class II medical evidence on the issue of therapy for VAI. Class III medical evidence suggests that a small number of patients with VAI will develop a posterior circulation stroke in delayed fashion beyond deficits associated with the initial traumatic injury. While no conclusive medical evidence supports treatment for VAI, most clinicians support treatment for patients with symptomatic VAI with either anticoagulation or antiplatelet therapy. Because of an increased relative risk of hemorrhagic complications from anticoagulation therapy for VAI, without clear superior efficacy,^{7,13,18,26,30} anticoagulation therapy is not considered ideal treatment in multiple trauma patients with VAI, symptomatic or asymptomatic. Antiplatelet therapy (aspirin the most studied) appears to be a safe and comparable option for symptomatic patients with VAI after blunt trauma.

No treatment or antiplatelet therapy appears to be a comparable option for the treatment of asymptomatic patients with documented VAI. Because antiplatelet therapy has the potential to

reduce future stroke risk, treatment with aspirin for documented VAI after trauma should be considered in patients if there exist no contraindications to antiplatelet therapy. At present, the choice of therapy, if any, for patients with VAI should be individualized based on the patient's vertebral artery injuries, associated traumatic injuries, and the relative risk of bleeding associated with that form of therapy.

KEY ISSUES FOR FUTURE INVESTIGATION

A multicenter, randomized, prospective study comparing anticoagulation with intravenous heparin to antiplatelet agents to no treatment in asymptomatic patients with VAI is recommended to determine which method of treatment of these injuries, if any, is most efficacious. Studies to assess the role of endovascular intervention in patients with VAI are needed to determine the application and merits of endovascular therapy to this acute traumatic vascular disorder.

Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

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