Vertebral Artery Injuries Associated With Cervical Spine Injuries: A Review of the Literature

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Study Design: Literature review.

Objective: To determine the incidence of vertebral artery injuries (VAIs) in association with cervical spine trauma and investigate the optimum diagnostic and treatment protocols.

Summary of Background Data: VAIs may result from cervical spine trauma and have the potential to cause cerebral, brainstem, and even spinal cord ischemia. Screening and treatment for traumatic VAI are very controversial, with conflicting recommendations within the trauma and spine literature.

Methods: A literature review was performed to identify publications pertaining to VAIs associated with cervical spine trauma. These publications were evaluated to determine the incidence, radiographic evaluation, and treatment options of VAIs.

Results: Approximately 0.5% of all trauma patients will have a VAI, and 70% of all traumatic VAIs will have an associated cervical spine fracture. Cervical spine translation injuries and transverse foramen fractures are most commonly cited as having a significant association with VAIs. The incidence of neurologic deficits secondary to VAI ranges from 0% to 24% in published series that incorporate a screening protocol for asymptomatic patients. Catheter angiography has been the gold standard for the diagnosis of VAIs; however, new 16-slice computed tomography angiography seems to have sensitivity and specificity close to that of catheter angiography. Treatment options include observation, antiplatelet agents, anticoagulation, and endovascular treatments. Although some authors have advocated antithrombotic therapy for most asymptomatic VAIs, there is a lack of class I evidence to support any strong guidelines for treatment.

Conclusions: VAIs can occur in association with cervical spine trauma and have the potential for neurological ischemic events. Screening for and treatment of asymptomatic VAIs may be considered, but it is unclear based on the current literature whether these strategies improve outcomes.

Key Words: vertebral artery, cervical spine, trauma, dissection, fracture

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INTRODUCTION

Vertebral artery injury (VAI) can occur spontaneously, as a result of minor trauma, or in association with major trauma such as penetrating injuries or cervical spine fractures.[1,2] Although blunt VAI is uncommon, occurring in only 0.5% of all nonpenetrating injuries, the incidence is much higher in select subpopulations, such as patients sustaining cervical spine injury.

Anatomy

The vertebral arteries arise from the subclavian arteries in most individuals and can be divided into 4 segments (Fig. 1). The first segment, V1 (*extraosseous segment*), extends from the origin at the subclavian artery to the transverse foramen of the sixth cervical vertebra (C6) typically. In approximately 5% of the population, the vertebral artery will enter a transverse foramen at C7. The *foraminal segment* (V2) consists of the portion of the vertebral artery passing through the transverse foramen of C6 to C1. The third segment, the *extraspinal segment* (V3), starts as the artery exits the foramen transversarium of C1. Upon exiting the foramen of C1, the artery courses posteromedially along the upper surface of the posterior ring of the atlas. The artery then abruptly turns ventral and cephalad to enter the foramen magnum. The V3 segment ends at the point where the artery penetrates the dura at the foramen magnum. V4, the *intradural segment*, extends from dural penetration to the pontomedullary junction where the two vertebral arteries unite in the midline to form the basilar artery.

Types (Classification) of VAI

Different types of arterial injuries, from intimal flaps to complete occlusions, exist, and risk of neurologic deficits and management strategies can vary with type of VAI. *Intimal tears* are the most subtle form of injury, where, as the name implies, the intima of the vessel tears free in a short section flapping out into the vessel lumen. *Dissections* are formed when a defect is created in the vessel intima and blood extravasates into the arterial wall. Thus, a false lumen is formed that can propagate in tissues planes within the arterial wall. Most dissections are subintimal (blood between intima and media), but some can be subadventitial (between media and adventitia).[3] Dissection causes the vessel wall to expand, resulting in compromise of the vessel lumen, and may result in thrombus formation at the site of intimal violation. *Pseudoaneurysms* are created when blood

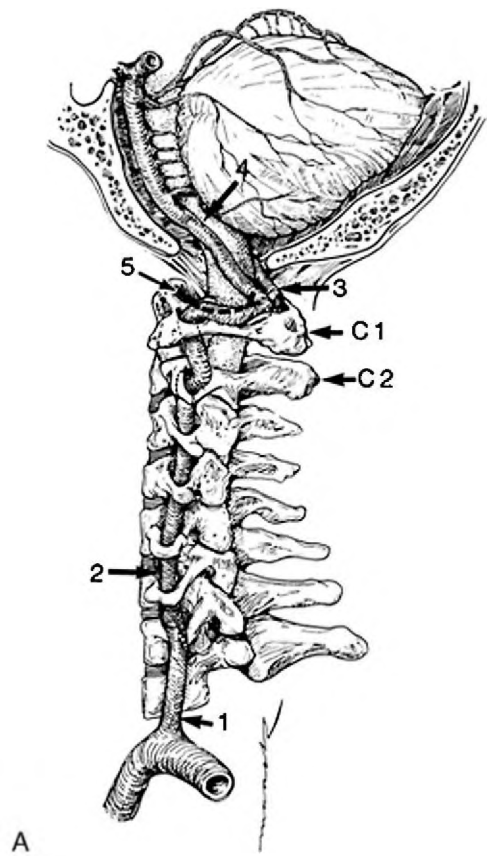

FIGURE 1. The vertebral artery can be divided into 4 segments as depicted on this lateral view illustration. (1) The extraosseous segment, V1, starts at the origin of the subclavian artery and typically extends to the transverse foramen of the sixth cervical vertebrae (C6). (2) The *foraminal segment* (V2) consists of the portion of the vertebral artery passing through the transverse foramen of C6 to C1. (3) The third segment, the *extraspinal segment* (V3), starts as the artery exits the foramen transversarium of C1. Upon exiting the foramen of C1, the artery courses posteromedially along the upper surface of the posterior ring of the atlas. The artery then abruptly turns ventral and cephalad to enter the foramen magnum. The V3 segment ends at the point where the artery penetrates the dura at the foramen magnum. (4) V4, the *intradural segment*, extends from dural penetration to the pontomedullary junction where the 2 vertebral arteries unite in the midline to form the basilar artery. (5) Dotted lines depict the location of the occipital condyles (with permission from *Diagnostic Cerebral Angiography*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 175).

ruptures through the vessel wall forming an extravascular hematoma. As the hematoma evolves, a cavity can form within the hematoma creating a pseudoaneurysm. If vessel injury is severe, the lumen may become completely obstructed, resulting in arterial *occlusion*. The most severe type of VAI is arterial *transection*, which is usually fatal.[4] A cerebrovascular injury classification (Table 1),[5] which was originally created for traumatic carotid artery injury, has been applied to VAI.[6–9]

TABLE 1. Cerebrovascular Injury Grading Scale Based on Angiographic Appearance

Grade	Description
Grade I	Irregularity of vessel wall or a dissection/intramural hematoma with less than 25% luminal stenosis
Grade II	Intraluminal thrombus or raised intimal flap is visualized, or dissection/intramural hematoma with 25% or more luminal narrowing
Grade III	Pseudoaneurysms
Grade IV	Vessel occlusions
Grade V	Vessel transactions or hemodynamically significant arteriovenous fistula

Although VAI may occur in up to 48% of some types of cervical fractures, the number of patients that will become symptomatic from these lesions is hard to predict.[6,10] Therefore, screening and treatment for traumatic VAI are very controversial, with conflicting recommendations within the trauma and spine literature. The purpose of this literature review was to determine the incidence of VAIs in association with cervical spine trauma and investigate the optimum diagnostic and treatment protocols.

MATERIALS AND METHODS

We performed a computerized search of the database of the National Library of Medicine from 1966 to December 2006 (www.pubmed.gov) using combinations of the following keywords: "vertebral artery," "trauma," "injury," and "cervical spine." The search was restricted to the English language and yielded 344 references. Abstracts were reviewed, and 52 articles were identified that provided direct or supporting evidence regarding the identification and treatment of VAIs. These articles were reviewed, and further relevant references from their bibliographies were identified to include all available class III or better medical evidence in this summary.

RESULTS AND DISCUSSION

Association of VAI With Cervical Fractures

In 1961, Carpenter[11] was the first to describe an association between cervical spine fracture and VAI. VAIs associated with cervical spine fractures are most likely to occur in the foraminal (V2) segment where the artery is in close proximity to osseous structures. Although it is typically protective of the artery, the path through the transverse foramen can put the artery at risk after spine trauma. Arterial injury can occur as a result of direct trauma from bone fragments or from excessive stretch in fracture-dislocations.

The results of multiple series using aggressive screening protocols for carotid and VAIs have shown that approximately 70% of VAIs identified in blunt trauma patients have an associated cervical spine fracture.[5,10] Obviously, this may be skewed because of a selection bias, since cervical spine fractures are a commonly cited indication for screening. Torina et al[12] retrospectively reviewed magnetic resonance angiography findings in 632 patients with cervical spine injuries and reported vertebral artery thrombosis in 13% of patients. Miller et al[6] and Biffl et al[5] screened all cervical spine fractures prospectively with 4-vessel cerebral angiography and reported VAI in 33% and 39% of cases, respectively.

Three types of cervical fractures are cited in the literature as putting the patients at significant risk for VAI: (1) fractures involving a transverse foramen[13–19]; (2) subluxations[11,13,20–29]; and (3) fractures involving the upper cervical spine (C1-C3).[13,30] Woodring et al[19] noted that transverse process fractures account for approximately 13% of all cervical fractures and found VAI in 7 of 8 (88%) patients undergoing screening. Vaccaro et al[31] reported VAI in 25% of transverse process fractures and 40% of facet dislocations. Kral et al[15] evaluated 119 consecutive cervical trauma patients and found an 8% incidence of VAI with foramen transversarium fractures and 21% with fracture-dislocations. Miller et al[6] reported VAI in 48% (28/58) of transverse process fractures and 44% (12/27) of traumatic subluxations. Cothren et al[13] reported a 37% incidence of VAI in fractures that have either transverse foramen involvement, subluxations, or location within the upper cervical spine, and a screening protocol using these three fracture patterns would detect 93% of VAIs. Incidence of each respective grade of injury varies substantially in the literature (Table 2).

Sequelae of VAI

Outcomes for patients with VAI can range from asymptomatic to posterior circulation stroke and death. Some authors report that most of these arterial injuries are clinically silent.[5,10] Other authors report significantly higher rates of transient ischemic attacks, vertebral artery thrombosis, and death after an injury.[22,33–36] Those patients who do develop symptoms often do so in a delayed fashion from the time of initial injury.[8,37]

Injury to the vertebral arteries can cause neurologic deficits by multiple mechanisms, including: (1) vertebral artery obstruction with resulting insufficient blood flow to the posterior circulation of the brain (vertebrobasilar insufficiency); (2) thrombus formation at the site of arterial luminal injuries with embolization downstream; (3) obstruction of blood flow into the posterior inferior cerebellar arteries (PICAs) with lateral medullary syndrome infarcts; and (4) anterior spinal artery compromise with resulting spinal cord ischemia.

Vertebrobasilar insufficiency can develop in situations where both vertebral arteries are occluded or when the dominant vertebral artery is injured.[5,10] The incidence of simultaneous bilateral VAIs is quite high, approaching 25% of all VAIs in some series.[9] The left vertebral artery is most often the dominant of the 2 vessels (75% of patients), and significant atresia of 1 vertebral artery is found in approximately 5% of cases.[38,39] If the dominant vessel becomes occluded or stenosed because of injury, a unilateral VAI could result in insufficient blood flow to the posterior circulation of the brain. Symptoms of

TABLE 2. Published Results of Screening for Traumatic VAIs

Publication	Location	Years Screened	Screening Modality	No. Patients Screened	Patients With VAI	Total VAI	Grade % of Injuries: Grade I (%)	Grade II (%)	Grade III (%)	Grade IV (%)	Grade V (%)	% With Stroke
Biffl et al[5]	Denver, CO	1996-1999	Catheter angiography	?	39	46	42	13	21	24	0	24
Cothren et al[29]	Denver, CO	1996-2004	Catheter angiography	727	124	?	41*	12*	18*	29*	0*	9
Miller et al[10]	Memphis, TN	1995-1999	Catheter angiography	?	50	64	—	75	22	3	0	14
Miller et al[6]	Memphis, TN	2000-2002	Catheter angiography	216	39	49	—	55	45	0	0	0
Utter et al[32]	Seattle, WA	2004	16-slice CTA	372	27	29	17	24	55	3	0	?

*Percentages are only for patients who were initially asymptomatic.

vertebrobasilar insufficiency can include dizziness, visual changes, dysequilibrium, ataxia, or depressed level of consciousness.[40–44] If sufficient collateral blood flow, via either the anterior cerebral circulation or external carotid circulation, is not present, more severe brainstem and posterior cerebral ischemia can result.

Embolic strokes are also possible with VAIs. A thrombus at the injured arterial site may break free and shower emboli throughout the posterior circulation, including the brainstem, cerebellum, posterior cerebral hemispheres, and thalamus.[45] Emboli are more common in nonocclusive vessel injuries like dissection or pseudoaneurysm. Once the vessel becomes completely occluded, the likelihood of emboli is smaller, unless a distal "stump" of vertebral artery that can provide a site for thrombus formation and emboli remains. Although "stump syndrome" has only been described for the carotid circulation, the situation of a stump in the posterior circulation may be just as treacherous in terms of potential for emboli.[22,39]

Another situation that can result in posterior fossa ischemia occurs when there is damage to a vertebral artery that terminates in the PICA without joining the other vertebral artery or when the damaged area involves the PICA takeoff from the vertebral artery. PICA obstruction can result in a lateral medullary syndrome (Wallenberg syndrome) infarct with ischemia to the lateral medulla and inferior cerebellum, resulting in a myriad of potential findings including dysphagia, ipsilateral facial numbness with contralateral trunk and extremity numbness, ipsilateral cerebellar findings, vertigo, and an ipsilateral Horner syndrome.[46] The anterior spinal artery also usually arises from the vertebral artery and occlusion may result in anterior spinal cord ischemia. Spinal cord ischemia can also result from interruption of intersegmental arteries arising from the vertebral artery to supply the middle and lower cervical cord.[5,6,10]

The data available in the current literature are quite variable regarding the incidence of neurologic sequelae associated with VAI. Incidence of posterior circulation stroke ranges from 0% to 24% in case series with more than 40 patients (Table 2).[5,6,13,28] In the setting of posterior circulation ischemia, the outcome can be very poor, with mortality rates as high as 33%.[47] In general, grade I, II, and III arterial injuries (intimal flaps, dissections, and pseudoaneurysms) are considered more treacherous lesions because they have a greater risk of embolic stroke than vertebral artery occlusions.[5] However, some series report approximately equal incidence of posterior circulation stroke with complete occlusions (grade IV) in comparison with the other grades of injury.[5,10,21,29]

Evaluation (Screening) of Potential VAI

It is estimated that 0.5% of all trauma patients will have a VAI.[5,6,29,48] Difficulty arises in determining which patients, if any, should be screened for vertebral artery trauma. To screen all trauma patients is not recommended since the screening measures like cerebral angiography have complications associated with them and are resource intensive. Some centers screen aggressively for blunt cerebrovascular injuries with a protocol that determines which patients get screened and typically includes patients with facial fractures, cervical spine injuries, depressed level of consciousness, or any neurologic deficits.[5,6,10]

More specifically, there is great debate about which, if any, cervical spine fractures indicate a need for routine screening for VAI. Although some authors[5,6,10,29] have recommended screening all patients with cervical spine injuries, others have recommended screening only those patients with cervical spine injuries that are at high risk for VAI, such as those with foramen transversarium fractures, dislocations, or high cervical injuries (C1 to C3). The subject of screening for VAI was debated at the 2006 Cervical Spine Research Society Instructional Course, at which a large majority of the audience and panel participants reported that they did not screen for vascular injuries in most patients with cervical spine injuries unless there were neurologic findings potentially of vascular cause.

The choice of appropriate modality for VAI screening is another controversial topic. Conventional catheter cerebral angiography is the gold standard for screening but carries risk for iatrogenic injury, stroke, and death. Complication rates with catheter angiography have been reported up to 4%, with iatrogenic strokes occurring in up to 1% of angiography studies.[3,24,49] Duplex scanning has been tried as a screening tool but does not appear to be a viable option because it offers poor visualization of the vertebral artery because of the surrounding bony anatomy.[3,5,6,50]

Other modalities, like magnetic resonance angiography and computed tomography angiography (CTA), are less invasive but historically have had poor sensitivity compared with conventional angiography.[3,6,21] Recent studies with new 16-slice multidetector CTA, however, have shown encouraging results.[32,51–53] Berne et al[51] screened 435 patients for potential blunt carotid and vertebral vascular injuries with a 16-slice CT scanner and reported a combined incidence of 1.2%, which is consistent with the combined incidence in conventional angiography series. Of the 411 patients with negative CTA findings, none developed neurologic deficits to suggest that vascular injuries had been missed. Biffl et al[52] screened 331 patients with 16-slice CTA and reported that no strokes occurred in the 311 patients with a normal CTA study. Eastman et al[53] screened 146 trauma patients with risk factors for blunt vascular injury with both 16-slice CTA and conventional catheter angiography and reported concordant results in 98% of cases. There was one false-negative CTA result in a patient with a grade I vertebral artery lesion that was asymptomatic, yielding a negative predictive value for CTA in excess of 99%. As the sensitivity of CTA improves in detecting vascular abnormalities, this modality will likely replace catheter angiography. In addition to being noninvasive, CTA can also be performed expeditiously in conjunction with other studies routinely ordered for trauma patients.

Once a VAI is noted, follow-up studies may be clinically useful for certain types of injuries. Some injuries may heal and thus allow for possible discontinuation of anticoagulation. Other injuries may become more severe and prompt new intervention. Biffl et al[21] found that repeat angiography was valuable for grade I, II, and III VAIs and recommended reimaging approximately 7 to 10 days after initial injury. In their series of 97 patients with VAIs, they found that 57% of grade I injuries and 8% of grade II injuries had healed by the time of repeat angiography, allowing cessation of therapy. Surprisingly, they also noted that 8% of grade I and 43% of grade II injuries (dissections) went on to form pseudoaneurysms requiring new treatment strategies. Other series have confirmed that significant percentages of these injuries can change with time and justify repeat imaging at 7 to 10 days after initial injury.[5,6,10,21,28,29,31]

Management of VAI

Management of VAI is very controversial as there are no class I (prospective randomized studies) and limited class II (prospective, randomly assigned, non-blinded studies) data available to assist in decision making. The most controversial issue of management is the treatment of the asymptomatic patient with a VAI. As with all clinical decisions, a risk-to-benefit ratio must be considered. Many trauma patients have multiple organ system injuries, and anticoagulation increases the risk of hemorrhagic complications. Although the authors of many articles support aggressive screening and anti-thrombotic therapy even for asymptomatic patients, most of these recommendations are based on the nonrandomized studies from just 2 centers (Denver, Colorado, and Memphis, Tennessee).[5,6,10,21,28,29,48]

Biffl et al[5,21] have recommended anticoagulation for patients with all grades of VAI except grade V (arterial transections) VAI or when absolute contraindications to systemic anticoagulation, such as significant brain injury with intracranial hemorrhage, are present. In their most recent publications, these authors have recommended low-dose systemic heparin anticoagulation to achieve a partial thromboplastin time (PTT) of 40 to 50 seconds.[21,28] They recommend starting with a continuous infusion of heparin at 15 U/kg/h, without a loading bolus, and gradually titrating to achieve the desired PTT. Previously, their protocol was more aggressive, with full heparin anticoagulation (PTT > 60), including bolus, but this protocol resulted in unacceptably high complication rates.[5] For patients who have contraindications to systemic heparin, the authors recommend the use of antiplatelet agents (aspirin 325 mg/d and clopidogrel 75 mg/d) or observation, depending on the severity of associated injuries. Patients are typically treated with oral anticoagulation or antiplatelet agents for 3 to 6 months or until normalization of angiogram results. The authors' recommendations for the treatment of blunt VAI are based on small retrospective studies (class III data) from their institution.

In 2000, Biffl et al[5] retrospectively reviewed 38 patients with VAI and reported improved neurologic outcomes in patients treated with systemic heparin. They found a 3-fold greater risk for posterior circulation stroke and a 10-fold greater risk of poor neurologic outcome in patients that were not treated with heparin. In addition to potential problems related to significant selection bias as a nonrandomized study, however, a close review of their published data indicate that additional limitations are present because the authors' conclusions were based on a very small subanalysis of only 5 patients that did not receive any heparin and 16 patients that received heparin.

In 2005, Cothren et al[29] reported on 235 patients diagnosed with blunt carotid injuries and VAIs in another nonrandomized study from the Denver Health Medical Center. They reported that "adequate" antithrombotic therapy (systemic heparin, antiplatelet, or low-molecular-weight heparin) reduced the risk of "ischemic neurological event" from 21% to 0.05%. In this study, there also seems to be a significant selection bias because the patients with "inadequate antithrombotic" therapy included patients with "subtherapeutic" antithrombotic therapy, on which they do not elaborate, and patients with contraindications to antithrombotic therapy, which included severe closed head injuries. It can be speculated that patients with severe closed head injuries would be at significant risk for ischemic neurologic events for a multitude of reasons other than vascular injury and, as a result, bias the study results.

Miller et al[10] have also supported aggressive screening and the use of antithrombotic agents based on 2 studies they have performed. In 2001, they reviewed 50 patients diagnosed with VAI and reported that treatment with either heparin anticoagulation or aspirin "appeared to prevent infarct" in VAI. However, in reviewing their retrospective series closely, we determined there were 7 (14%) strokes in 50 patients with VAI including 0 in the heparin group, 1 in the aspirin group, and 6 that were included in the "no treatment" group. However, including patients who never received treatment and those who received no treatment before the onset of ischemia in the "no treatment" group biased the results. Patients included in the treatment groups (heparin and aspirin groups) only included those patients who were asymptomatic at the time of diagnosis. It seems that all of the 6 strokes in the "no treatment" group occurred before the diagnosis of VAI was made. A better analysis would have been to compare the outcomes (stroke rates) of treatment versus nontreatment in patients who were asymptomatic at the time of diagnosis of VAI.

Miller et al[6] prospectively evaluated trauma patients who were aggressively screened for blunt cerebrovascular injuries. Trauma patients having cervical fractures, LeFort II and III facial fractures, Horner syndrome, soft tissue injuries in the neck, skull base fractures involving the foramen lacerum, or neurologic findings unexplained by traumatic brain injuries were screened for vascular injury. The authors diagnosed 24 carotid injuries and 43 VAIs in the 216 patients screened with 4-vessel cerebral angiography. Of the 43 patients with VAI evaluated prospectively, 32 patients were treated with antiplatelet therapies, 8 received heparin anticoagulation, and 3 received no treatment. The stroke rate was 0% in all groups in this study. Despite these inconclusive findings, the authors still suggested that early diagnosis and treatment of VAI reduced stroke rates.

In patients with a symptomatic vascular injury, the decision to treat is less controversial, but the type of treatment can vary. Treatment (anticoagulation, antiplatelet agents, blood pressure supportive measures, thrombolytic therapy, endovascular treatments, or surgery) depends on the type and location of vascular lesion, and the extent and location of the infarction. Consultation with a neurology stroke expert or neurosurgeon is warranted to determine the optimal treatment of these patients.[1] The optimal treatment (anticoagulation vs. antiplatelet agents) of cervical arterial pathologies (dissections and stenosis) is currently being debated in the neurology literature with results, including those from class I prospective randomized studies, showing no benefit of anticoagulation over antiplatelet agents.[23,54,55] The issue of blunt VAI was also addressed by a committee reviewing the literature for acute cervical spine injuries in 2002.[56] This committee concluded that there was insufficient evidence to support any treatment guidelines at that time. At the present time, our spine services are recommending antiplatelet therapy for approximately 3 months in an asymptomatic patient once surgical intervention has been completed.

In addition to anticoagulation and antiplatelet agents, other therapies including thrombolytic therapy, surgery, and endovascular techniques have been used to treat VAIs.[1,38,57–59] Thrombolytic agents have been used predominantly to treat vertebral artery thrombosis that has propagated to cause occlusion of the basilar artery.[1,58] Surgery, in the form of thrombectomy, bypass, or ligation procedures, has been reported, but overall the role of surgery seems to be small and shrinking.[25] With new endovascular techniques and the technical challenges of approaching the vertebral artery, the role for surgery for vertebral artery lesions is diminishing.[27] Endovascular stenting and coiling have been used to treat pseudoaneurysms, dissections, and even arterial transections.[20,21,26,56,57,59–61]

Summary

The risk for VAI seems to be significant with certain fractures patterns. Considerable controversy exists regarding indications for screening and treatment of VAI. Catheter cerebral angiography has been the gold standard for diagnosis of vascular injury but is likely to be replaced by CTA as the sensitivity of this study has improved significantly with the use of new 16-slice CT scanners. Treatment options include close observation in asymptomatic patients, heparin anticoagulation, antiplatelet agents, thrombolytics, surgery, and endovascular treatments. Consultation with stroke neurologists and vascular neurosurgeons is often warranted to determine the optimal treatment plan for each individual.

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