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Diagnosis and Treatment of Arterial Dissections

Ricky Medel · Robert M. Starke · Edison P. Valle-Giler · Sheryl Martin-Schild · Ramy El Khoury · Aaron S. Dumont

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Abstract Dissections of the cervical and intracranial vessels represent an important source of stroke in those less than 50 years of age. This can occur spontaneously or following trauma, minor or major. Rapid diagnosis is essential to limit subsequent sequelae and modern computed tomographic angiography represents an appropriately sensitive modality. Treatment must be individualized to the patient and can consist of an antiplatelet regimen, anticoagulation, or endovascular intervention. No evidence demonstrates superiority of either medical modality and even aspirin alone may be efficacious. Consideration should be given to this in the multi-trauma population in which more aggressive anticoagulation is contraindicated. In addition, thrombolytic administration should not be withheld would it otherwise be indicated. Endovascular intervention is reserved for those with hemodynamically significant narrowing, enlarging pseudoaneurysms, fistulas formation, or subarachnoid hemorrhage.

Keywords Carotid artery · Dissection · Vertebral artery · Stroke · TIA · SAH · Trauma · Anticoagulation · Antiplatelet

Introduction

Although relatively uncommon for the population as a whole, dissections of the cervical and intracranial arteries account for a significant percentage of strokes in those less than 45 years of age [1]. A dissection can occur spontaneously or as a result of trauma, both major and minor. Given the young age of these patients and the severity of the sequelae, early detection is critical. Diagnosis is increasing due to the frequent use of noninvasive angiography and once discovered, appropriate management is essential to minimize the associated morbidity and mortality. Many of these injuries will heal spontaneously and treatment is directed toward the prevention of thromboembolic and hemodynamic injury. This often consists of anticoagulation or antiplatelet agents; however, in some circumstances endovascular or surgical intervention is indicated. It is the purpose of this article to review the available literature regarding diagnosis and management of this important condition. The focus will be on the adult population as a recent review of the diagnosis and treatment in pediatrics already exists [2]. Iatrogenic dissections while being managed in a similar fashion; however, are not explicitly discussed.

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R. Medel · E. P. Valle-Giler · A. S. Dumont (✉)
Department of Neurosurgery, Tulane University School of Medicine, 131 S. Robertson St, 8047, New Orleans, LA 70112, USA
e-mail: adumont2@tulane.edu

R. M. Starke
Department of Neurosurgery, University of Virginia School of Medicine, Charlottesville, VA 22908, USA

S. Martin-Schild · R. El Khoury
Department of Neurology, Tulane University School of Medicine, New Orleans, LA 70112, USA

Epidemiology

Internal carotid artery dissection (ICAD) has been estimated to occur at an incidence of 2.5–3 per 100,000, slightly higher than the 1–1.5 per 100,000 estimated for vertebral artery dissection (VAD) [3–5]. These values likely underestimate the true incidence, as those patients with minimal symptomatology often remain undiagnosed. In an analysis of 1008 consecutive patients aged 15–49 presenting with a first time ischemic stroke, cervical or intracranial dissection was the underlying etiology in 15.4 % [1]. While dissection is often classified as spontaneous or traumatic, there is considerable overlap. Minor events, some of which may be difficult for the patient to recall, have been associated with arterial dissection and include: chiropractic manipulation [6, 7], sports-related, heavy lifting, infection, substantial movement of the neck [8, 9], even coughing, sneezing, retching, and sexual intercourse. In a recent study using data from the Cervical Artery

Dissection and Ischemic Stroke Patients (CADISP) study, 40.5 % of patients with dissection reported prior trauma, 88 % of which was mild, in contrast to only 10.8 % having prior trauma in the nondissection group [8]. Concerning those patients with a history of significant blunt-force trauma, up to 31 % have been found to have evidence of cerebrovascular injury on angiography [10].

Clinical Presentation

The symptoms of cerebral ischemia due to dissection are obviously the same as for those due to other etiologies. As such, the description below will focus on the additional clinical features that are unique to arterial dissections of the cervical and intracranial circulations. While often assessed as a combined entity, and there are many commonalities, ICAD and VAD have been demonstrated to be different in terms of incidence, clinical presentation, associated comorbidities, and outcomes [11–13]. These differences are described below; however, it is essential to note that these patients commonly present with nonspecific symptoms (ie, dizziness, vertigo, headache, and neck pain) [13].

The classic presentation of ICAD includes unilateral pain in the head or neck sometimes radiating to the ipsilateral eye, partial Horner's syndrome (anhidrosis not being present), followed by cerebral or retinal ischemia. However, the complete triad is only present in the minority of patients. Patient's with ICAD have been found to be significantly older (46 vs 41, $P<0.0001$), more often male (60 % vs 51 %, $P=0.006$), and are more likely to have an infection (21.7 % vs 14.6 %, $P=0.009$) preceding the dissection [11]. In an analysis of the CADISP data there was a greater incidence of headache in this population (67.8 % vs 64.5 %, $P=0.04$)[11], but others have found the rates to be equivocal [12]. Horner's syndrome and other cranial nerve palsies vary based on their anatomic associations (ie, Horner's being isolated to ICAD), with pulsatile tinnitus (10.9 % vs 3.4 %, $P<0.001$) being the only other clinical symptom that is more likely with anterior circulation lesions [12].

Concerning vertebral artery dissection, the frequency is similar between men and women. They are more likely to present with neck pain (66 % vs 39 %, $P<0.0001$), have bilateral dissections (15.2 % vs 7.6 %, $P<0.001$), have recent history of trauma [14], and to be current smokers [11, 12]. Aside from smoking, the only other classic risk factor for vascular disease that has been found more frequently in the CAD population is hypertension [15]. VAD are also more likely to present with symptoms of ischemia (84.4 % vs 70.4 %, $P<0.001$), although they tend to have a lower stroke severity as measured by the NIHSS examination than with ICAD (NIHSS 5±.00 vs 10±1001)$P<0.001$) [12, 16, 17]. When comparing those patients with and without ischemia, vascular risk factors such as male sex, smoking, and increased age are more common in those presenting with ischemia [18]. There was also a higher rate of subarachnoid hemorrhage in this population secondary to intracranial involvement (6.0 % vs 0.6 %, $P<0.001$) [12].

Diagnosis

Although the majority of patients present with signs and symptoms of cerebral ischemia, approximately 15 % of those with vertebral artery dissection and 30 % with carotid artery dissections do not [12]. This necessitates a high index of suspicion when evaluating this patient population. For those who do present with concerns for ischemia, the current American Heart Association (AHA) guidelines for the management of acute ischemic stroke recommend that patients undergo parenchymal imaging of the brain, as well as vascular imaging of the head and neck, with this imaging being interpreted within 45 minutes of arrival to an emergency department [19]. This should be performed in such a manner as to not delay the administration of thrombolytics or mechanical intervention if appropriate. Given the urgency of this evaluation, whichever modality is chosen, it must be performed and interpreted expeditiously.

When considering the trauma population, several studies have shown a rate of 0.6 % to 3.5 % of those with blunt force trauma have underlying CAD [20–23]. This increases to 5.5 %–29 % in patients who have positive findings on neurological examination, cervical spine injuries, skull base or facial fractures, and soft tissue injuries to the neck [20–23]. Criteria for screening, initially published by Biffl et al [24] has since become known as the Denver Screening Criteria and consists of facial hemorrhage, cervical bruit (age <50 years old), expanding cervical hematoma, lateralizing neurological deficit, cervical spine injuries, diffuse axonal injury, near hanging, soft tissue injury to the neck, or basilar skull fractures extending into the carotid canal. Again, expeditious evaluation and treatment remains critical if ischemic sequelae are to be prevented; however, this is not universally accepted. Several studies [25–27] have suggested that aggressive screening does not represent an appropriate use of resources for 2 reasons, first, the occurrence of stroke prior to diagnosis and treatment, or second, due to the presence of contraindications to the initiation of therapy. Importantly, some of the same series have demonstrated a significant decrease in the rate of stroke in the treated population, usually with either antiplatelets or anticoagulation (3.9 % vs 25.8 %, $P=0.003$) [27], thereby raising doubts as to the legitimacy of this suggestion. It is the authors' contention that aggressive screening should continue and further evaluation of the adverse effects of anticoagulation or antiplatelets in the group with reported contraindications be performed.

Very little quality literature exists regarding the sensitivity and specificity of noninvasive angiography compared with Digital Subtraction Angiography (DSA) [20–23, 28–31], and DSA remains the gold standard to confirm and potentially intervene upon arterial dissection [21, 30]. Nevertheless, modern computed tomographic angiography (CTA) and magnetic resonance angiography (MRA) have been found to be reasonably sensitive in several studies and are an appropriate choice for initial screening [20, 22, 23, 28, 32, 33]. Zuber et al [32] published a series in 1994 of 15 consecutive patients with 19 angiographically confirmed arterial dissections (9 ICAD, 10 VAD) comparing 0.5 Tesla MRA in all 15 patients and dynamic CT in 12 patients. The sensitivity of MRA and CTA was 78 % and 100 % in ICAD and 60 % and 57 % for VAD respectively. Although these results are encouraging, they are not uniform, likely secondary to differences in technology and technique. Miller et al [22] compared MRA and CTA to DSA in 143 patients and found sensitivities of 50 % and 47 % for CAD and 47 % and 53 % for VAD, respectively. This group used a 0.2 Tesla open MR and for CTA used a helical scanner with 1-mm slices, but only printed every third image for review. This group concluded that using these techniques with the available technology, noninvasive angiography was not a suitable substitute vs DSA to screen for arterial injury. More significantly however, was the reduction in stroke rate in vertebral artery injuries achieved (0 % vs 14 %) when using an aggressive screening paradigm and early institution of treatment for the dissection as compared with historical controls.

As is evident above, detection of VAD poses a greater problem given the proximity to bony structures throughout its extracranial course. Chen et al [34] retrospectively analyzed data where a multi-detector (4-slice) helical CT scanner was used to evaluate 34 patients, 17 controls, and 17 with dissections. Following acquisition, the data were sent to a workstation for processing and both the reconstructed maximum intensity projections and source images reviewed. In this series, CTA was 100 % sensitive for the dissection of arterial injury. Further support of the sensitivity of modern CTA in the evaluation of arterial dissection comes from 2 large series where multi-detector (16-slice) CTA was used to screen a total of 766 patients who had sustained blunt force trauma [20, 23]. Screening criteria varied, but both were similar to that described above. Both yielded a similar rate of CAD, approximately 5.5 % in the screened population and more importantly, no patient with a normal CTA developed symptomatology attributable to an undiagnosed arterial dissection. Given these findings and the availability and efficiency of CTA, it is recommended as an initial screening modality in patients where arterial dissection is suspected.

Management

The primary goal of treatment in extracranial arterial dissection is to prevent ischemic complications, whereas for intracranial dissections it involves both this and the prevention of intracranial hemorrhage (usually subarachnoid hemorrhage) or propagation of fistulous connections. Dissection is a dynamic process with the potential for secondary ischemia, pseudoaneurysm formation, or hemorrhage. Damage to the intima of the artery and exposure of the underlying media stimulates the coagulation cascade and platelet activation. This allows for thromboembolic phenomenon to occur at the time of injury, as well as in the intervening period until the institution of therapy. In addition, the surge of arterial blood within the wall of the artery can result in the formation of a false lumen, a pseudoaneurysm, or an intramural hematoma, with the latter posing the risk of hemodynamic compromise to the vessel. Pseudoaneurysms, with the concomitant turbulence in arterial flow serve as both a site for thrombosis as well as having the potential to compromise the true vessel lumen. In the intracranial circulation, more aggressive interventional treatment of pseudoaneurysms, dissection-related subarachnoid hemorrhage, or traumatic fistulas is indicated; with antithrombotic therapy being initiated postoperatively depending upon the modality used. Again, dissection is not a static process and the need for different therapies may evolve over the patients' course.

Antithrombotic Therapy

There is neither consensus nor convincing evidence as to the superiority of either antiplatelet agents (aspirin, clopidogrel, ticlopidine) or anticoagulation (coumadin, low-molecular weight heparin, unfractionated heparin) in the treatment of cervical artery dissection [35–38, 39••, 40]. Furthermore, throughout the literature there is also no consistency regarding the exact regimen utilized. In 2010 a Cochrane Review was published by Lyrer and Engelter [40] evaluating the efficacy of these 2 approaches in carotid artery dissections. Comparing the results of 36 observational studies comprised of 1285 patients revealed no significant differences in the risk of stroke or death between the 2 modalities. There was a nonsignificant trend toward lower death and disability in the anticoagulation group (OR 1.77, 95 % CI 0.98 to 3.22; $P=0.06$). Hemorrhagic complications were only observed in the anticoagulation group and occurred at a rate of 0.8 % for symptomatic intracranial hemorrhage and 1.6 % for major extracranial hemorrhage.

To date, no randomized controlled trial evaluating the safety and efficacy of antiplatelets or anticoagulation has been published. In June of 2013, the Cervical Artery Dissection in Stroke Study (CADISS) completed recruitment, but the results are not yet available. In 2012, they reported the nonrandomized results in a total of 88 patients demonstrating no significant difference in the risk of stroke or death between the 2 treatments [39••]. Overall, the rate of recurrent stroke and transient ischemic attack (TIA) was 1.7 % and 5.1 % with antiplatelets and 3.6 % and 0 % with anticoagulation, respectively. There were no serious adverse events in either group. Importantly, even aspirin alone has been demonstrated to be effective in an analysis of prospectively collected data [38]. Given this, nearly all patients with a cervical arterial dissection should be initiated on some form of antithrombotic agent as soon as possible after diagnosis, even if it consists of aspirin as monotherapy [41].

Thrombolysis

In the evaluation of patients with cervical artery dissections for thrombolysis, there exists a concern for extension of the mural hematoma, pseudoaneurysm formation, and even arterial rupture in addition to the usual concerns for hemorrhagic transformation. Several articles have been published regarding the use of thrombolytics in this circumstance [42•, 43–45], including a meta-analysis of 180 patients [45] and a recent evaluation of the CADISP database [42•]. While the study by Zinkstok et al [45] did not show an increase in the rates of symptomatic intracranial hemorrhage compared with matched controls; the more recent review of the CADISP database [42•] also did not demonstrate any benefit. Engelter et al [42•] compared the outcomes of 68 patients with CAD who received thrombolysis to 64 matched controls, also with stroke in the setting of CAD. The odd-ratio for a favorable recovery was 1.00, with a 5.9 % rate of hemorrhage in the thrombolysis group, compared with 0.6 % in the controls. Since the emergency evaluation for thrombolytic eligibility calls for a noncontrasted head CT as the sole neuroimaging modality, detection of a dissection may occur long after emergency treatment has been provided, limiting the comparison of intravenous thrombolysis and endovascular treatment in this population. This and the above data suggest that while thrombolytics should remain within the therapeutic arsenal, focus toward a more effective means of treatment should be maintained.

Interventional Therapy

The indication for endovascular intervention depends upon the location and anatomy of the lesion. Intracranially, it is often necessary to treat pseudoaneurysms or dissections presenting with or threatening subarachnoid hemorrhage, to obliterate fistulous connections, or to restore flow to at risk brain tissue in the case of luminal compromise. Extracranially, treatment is more centered on restoring the lumen to correct hemodynamic insufficiency with stenting, or in the case of vertebral artery lesions, deconstructive therapy to prevent thromboembolic complications. Either way, the patients must be suitable for heparinization during the procedure and antiplatelet medications both prior to and following stent placement.

Cervical Arterial Dissections

The literature regarding endovascular therapy for cervical lesions consists primarily of case reports, small case series [46–52, 53•, 54, 55], or literature reviews and meta-analysis [56, 57]. In a recent review of historical data from 1997 to 2008 [57], the technical success rate was 99 %–100 % with 1.3 % periprocedural complications and 1.4 % rate of new symptoms during follow-up. Ninety-eight percent of treated pseudoaneurysms were occluded at follow-up. The criteria for intervention varied as many of the included series were isolated case reports. The use of antiplatelets and anticoagulation was also inconsistent and depended on the practitioner. Concerning the new symptomatology, there were 2 episodes of TIA with subsequent normal angiograms and without further symptoms.

Seth et al [53•] recently published a retrospective review of their data regarding the interventional management of internal carotid artery injuries. Fifty arterial lesions were treated in 47 patients, 44 of which underwent stenting alone, 4 stent-assisted coilings, and 2 coiling alone. To be selected for intervention, patients had to have greater than 70 % stenosis, enlargement of a pseudoaneurysm, or intraluminal thrombus and irregularity with 25 %–70 % stenosis. All patients received clopidogrel and aspirin prior to the procedure and this was continued for at least 12 weeks. Self-expanding stents were used in all instances. Luminal diameter was improved in all patients to a level with no further hemodynamic compromise, and normalization of the lumen was achieved in 50 %. Transient complications were noted in 6.4 % of patients without any permanent morbidity or mortality. One patient (2 %) had complete occlusion of the stent on follow-up with neurological sequelae and 1 developed a pseudoaneurysm following stenting requiring an additional stent and coil embolization. While these results demonstrate relative safety and feasibility of intervention for these lesions, it is important to note that the results are similar to medical management alone, emphasizing the imperative for appropriate patient selection.

We have generally reserved interventional therapy for patients with ischemic symptoms refractory to medical therapy, patients with acute ischemic symptoms and a tandem lesion (cervical and intracranial) unresponsive to intravenous thrombolysis (or out of the window of thrombolysis) and those who develop delayed pseudoaneurysms despite medical therapy.

Intracranial Arterial Dissections

There are numerous small case series and case reports concerning the interventional management of intracranial dissections [55, 58–68]. A lesion of the V4 segment of the vertebral artery with or without involvement of the posterior inferior cerebellar artery (PICA) origin is one of the more common locations. The associated pseudoaneurysms have been estimated to represent approximately 28 % of all posterior circulation aneurysms [69]. A high propensity for rupture exists in this situation, with approximately 73 % presenting with subarachnoid hemorrhage and the remainder with signs and symptoms of ischemia. Depending on the location of the dissection and the relevant anatomy, either reconstructive or deconstruction therapies may be utilized. Additionally, consideration must be given to the presentation, as reconstruction often involves stent placement, which necessitates antiplatelet therapy. This has the potential for hemorrhagic complications in the ruptured population. While open surgical intervention was previously employed, modern endovascular techniques often afford lower rates of morbidity and mortality.

In 2011, Kim et al [64] published their series of 111 patients treated for vertebrobasilar dissecting aneurysms. Fifty-two percent were treated with stenting with or without coiling to reconstruct the vessel lumen, with the remainder being treated with internal trapping using coils to occlude the parent vessel. The rate of periprocedural complications is not available, but all patients with unruptured aneurysms (38) and 77 % of those with rupture achieved a modified Rankin Score of 0–2, indicating independence. Nine patients (12 %) with a ruptured aneurysm died, 5 of which were secondary to rebleeding. Recurrence of the pseudoaneurysm was identified in 13 % with rebleeding occurring in 5 %. All of those in the latter category initially presented with hemorrhage. The rate of recurrence did not differ by technique and the only independent risk factor was involvement of the PICA origin. In a more recent series by Kashiwazaki et al [63], 73 patients were treated with parent artery occlusion. Only 2 patients had a recurrence, but there was a 15 % rate of neurological complications with a 1.2 % rate of mortality. The neurological complications were secondary to ischemic complications involving a branch vessel, including 2 infarctions in the territory of the anterior spinal artery. These 2 series elucidate the aggressive nature of these lesions and the relatively high rates of concomitant morbidity and mortality, especially in the population presenting with rupture.

As endovascular technology continues to evolve, new equipment has become available to treat intracranial dissections. Most recently this is in the form of a tightly woven stent that provides flow diversion as a means of vessel reconstruction. Although not approved for this indication, some preliminary reports exist for use of the Pipeline Embolization Device (EV3, Irvine, CA) [58, 59, 68, 70]. In 1 of the larger series, de Barros Faria et al [58] reported their experience in 23 patients with dissecting intracranial aneurysms, 91 % of which were in the posterior circulation. The majority of patients presented with subarachnoid hemorrhage (52 %), with the remainder presenting with ischemia (4 %), mass effect (22 %), or incidentally. Total occlusion of the aneurysm was achieved in almost 70 % of patients with a 90 % technical success rate per device insertion and 74 % good clinical outcome (Glasgow Outcome Score 4 or 5). Importantly, only 4 of those patients with hemorrhage at presentation were treated acutely and all patients were treated with heparin during the procedure and aspirin and

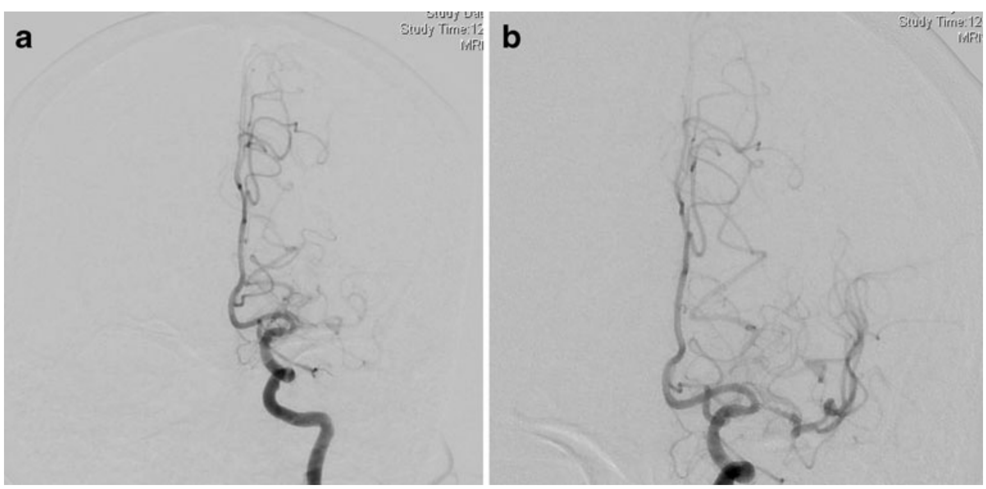

Fig. 1 A 69-year -old male presented with right-sided hemiplegia and aphasia and an acute left middle cerebral artery occlusion in the M1 segment initially diagnosed on CT angiography (not shown). There was no history of trauma. The patient was administered intravenous tPA and taken emergently for endovascular intervention. Injection of the left middle cerebral artery redemonstrated occlusion of the M1 segment (a). The segment was revascularized using a stent-retriever device; however, on control angiography an underlying dissection was visualized. A self-expanding stent was used to maintain patency of the segment and 2 mg of abciximab administered for a small amount of clot within the stent. Post control angiography (b) demonstrated patency of the stent with revacularization of the effected territory. The patient made a near complete recovery with only minor word finding difficulty

clopidogrel prior to and for at least 6 months following the procedure. There were 2 procedure related complications; 1 retroperitoneal hematoma and 1 thromboembolic complication from which the patient made a complete recovery. These preliminary data seem to indicate that this device represents a feasible strategy in those that are not amenable to other treatment modalities; however, further confirmation is necessary.

While less common, this disease affects the anterior circulation as well with a seemingly greater propensity for ischemia as opposed to subarachnoid hemorrhage (Fig. 1). Fields et al [60] recently analyzed the Merci registry of 980 patients and identified 10 in whom there was an underlying arterial dissection. Seventy percent of these lesions affected the middle cerebral artery. Treatment consisted of a combination of intravenous tissue plasminogen activator (tPA) (20 %), intra-arterial tPA (50 %), and mechanical thrombectomy in all. Stenting was performed in 44 % of patients due to the associated dissection. There was only 1 complication, which resulted in an extension of the dissection. This was treated with the stenting procedure. Eighty percent of patients in this population achieved a good functional outcome (mRS ≤ RS t This latter group of patients likely represents an underdiagnosed cohort as the etiology of the stroke is only realized following mechanical thrombectomy. It is important to delineate these patients from others with acute stroke however, due to the frequent need for stenting of the affected segment following recanalization.

Conclusions

Arterial dissections of the cervical and intracranial circulation are a frequent cause of stroke in those under 50 years of age. This diagnosis must be considered in the primary evaluation of patients because, not only is treatment different, but 20 %–30 % of patients will present with nonspecific symptoms instead of ischemia or hemorrhage. Digital subtraction angiography remains the gold standard in diagnosis; however, CT angiography is an appropriately sensitive initial screening modality using modern technology. Medical management is the preferred therapy for those without hemodynamically significant stenosis or a large/enlarging compressive pseudoaneurysm in the cervical region. At this point, either an antiplatelet regimen or full anticoagulation seems to be equally effective. This is of significance in the trauma population, as full anticoagulation is often not an option. Intracranial dissections are more likely to necessitate endovascular intervention to prevent hemorrhagic complications. The morbidity and mortality associated with vertebrobasilar lesions is significant, but endovascular therapy substantially improves upon the natural history. Further evidence is required before routine use of flow diversion devices can be recommended.

Compliance with Ethics Guidelines

Conflict of Interest Ricky Medel declares that he has no conflict of interest. Robert M. Starke declares that he has no conflict of interest. Edison P. Valle-Gil declares that he has no conflict of interest. Sheryl Martin-Schild declares that she has no conflict of interest. Ramy El Khoury declares that he has no conflict of interest. Aaron S. Dumont declares that he has no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1. Putaala J, Metso AJ, Metso TM, et al. Analysis of 1008 consecutive patients aged 15 to 49 with first-ever ischemic stroke: the Helsinki Young Stroke Registry. Stroke. 2009;40:1195–203. doi:10.1161/STROKEAHA.108.529883.
2. Stence NV, Fenton LZ, Goldenberg NA, et al. Craniocervical arterial dissection in children: diagnosis and treatment. Curr Treat Options Neurol. 2011;13:636–48. doi:10.1007/s11940-011-0149-2.
3. Lee VH, Brown Jr RD, Mandrekar JN, Mokri B. Incidence and outcome of cervical artery dissection: a population-based study. Neurology. 2006;67:1809–12. doi:10.1212/01.wnl.0000244486.30455.71.
4. Schievink WI, Mokri B, Whisnant JP. Internal carotid artery dissection in a community. Rochester, Minnesota, 1987-1992. Stroke. 1993;24:1678–80.
5. Giroud M, Fayolle H, Andre N, et al. Incidence of internal carotid artery dissection in the community of Dijon. J Neurol Neurosurg Psychiatry. 1994;57:1443.
6. Smith WS, Johnston SC, Skalabrin EJ, et al. Spinal manipulative therapy is an independent risk factor for vertebral artery dissection. Neurology. 2003;60:1424–8.
7. Albuquerque FC, Hu YC, Dashti SR, et al. Craniocervical arterial dissections as sequelae of chiropractic manipulation: patterns of injury and management. J Neurosurg. 2011;115:1197–205. doi:10.3171/2011.8.JNS111212.
8. Engelter ST, Grond-Ginsbach C, Metso TM, et al. Cervical artery dissection: trauma and other potential mechanical trigger events. Neurology. 2013;80:1950–7. doi:10.1212/WNL.0b013e318293e2eb.
9. Grau AJ, Buggle F, Ziegler C, et al. Association between acute cerebrovascular ischemia and chronic and recurrent infection. Stroke. 1997;28:1724–9.
10. Ringer AJ, Matern E, Parikh S, Levine NB. Screening for blunt cerebrovascular injury: selection criteria for use of angiography. J Neurosurg. 2010;112:1146–9. doi:10.3171/2009.6.jns08416.
11. Debette S, Grond-Ginsbach C, Bodenant M, et al. Differential features of carotid and vertebral artery dissections: the CADISP Study. Neurology. 2011;77:1174–81. doi:10.1212/WNL.0b013e31822f03fc.

12. von Babo M, De Marchis GM, Sarikaya H, et al. Differences and similarities between spontaneous dissections of the internal carotid artery and the vertebral artery. Stroke. 2013;44:1537–42. doi:10.1161/STROKEAHA.113.001057.
13. Gottesman RF, Sharma P, Robinson KA, et al. Clinical characteristics of symptomatic vertebral artery dissection: a systematic review. Neurologist. 2012;18:245–54. doi:10.1097/NRL.0b013e31826754e1.
14. Yaghi S, Maalouf N, Keyrouz SG. Cervical artery dissection: risk factors, treatment, and outcome: a 5-year experience from a tertiary care center. Int J Neurosci. 2012;122:40–4. doi:10.3109/00207454.2011.622453.
15. Debette S, Metso T, Pezzini A, et al. Association of Vascular risk factors with cervical artery dissection and ischemic stroke in young adults. Circulation. 2011;123:1537–44. doi:10.1161/CIRCULATIONAHA.110.000125.
16. Chandra A, Suliman A, Angle N. Spontaneous dissection of the carotid and vertebral arteries: the 10-year USCD experience. Ann Vasc Surg. 2007;21:178–85. doi:10.1016/j.avsg.2006.10.020.
17. Sarraj A, Medrek S, Albright K, et al. Posterior circulation stroke is associated with prolonged door-to-needle time. Int J Stroke. 2013. doi:10.1111/j.1747-4949.2012.00952.x.
18. Arnold M, Kurmann R, Galimanis A, et al. Differences in demographic characteristics and risk factors in patients with spontaneous vertebral artery dissections with and without ischemic events. Stroke. 2010;41:802–4. doi:10.1161/STROKEAHA.109.570655.
19. Jauch EC, Saver JL, Adams Jr HP, et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2013;44:870–947. doi:10.1161/STR.0b013e318284056a.
20. Berne JD, Reuland KS, Villarreal DH, et al. Sixteen-slice multi-detector computed tomographic angiography improves the accuracy of screening for blunt cerebrovascular injury. J Trauma. 2006;60:1204–9. doi:10.1097/01.ta.0000220435.55791.ce. discussion 1209–10.
21. Hoit DA, Schirmer CM, Weller SJ, et al. Angiographic detection of carotid and vertebral arterial injury in the high-energy blunt trauma patient. J Spinal Disord Tech. 2008;21:259–66. doi:10.1097/BSD.0b013e318141fce8.
22. Miller PR, Fabian TC, Croce MA, et al. Prospective screening for blunt cerebrovascular injuries: analysis of diagnostic modalities and outcomes. Ann Surg. 2002;236:386–93. doi:10.1097/01.SLA.0000027174.01008.A0. discussion 393–385.
23. Biffl WL, Egglin T, Benedetto B, et al. Sixteen-slice computed tomographic angiography is a reliable noninvasive screening test for clinically significant blunt cerebrovascular injuries. J Trauma. 2006;60:745–51. doi:10.1097/01.ta.0000204034.94034.c4. discussion 751–42.
24. Biffl WL, Moore EE, Elliott JP, et al. The devastating potential of blunt vertebral arterial injuries. Ann Surg. 2000;231:672–81.
25. Griessenauer CJ, Fleming JB, Richards BF, et al. Timing and mechanism of ischemic stroke due to extracranial blunt traumatic cerebrovascular injury. J Neurosurg. 2013;118:397–404. doi:10.3171/2012.11.JNS121038.
26. Mayberry JC, Brown CV, Mullins RJ, Velmahos GC. Blunt carotid artery injury: the futility of aggressive screening and diagnosis. Arch Surg. 2004;139:609–12. doi:10.1001/archsurg.139.6.609. discussion 612–603.
27. Stein DM, Boswell S, Sliker CW, et al. Blunt cerebrovascular injuries: does treatment always matter? J Trauma. 2009;66:132–43. doi:10.1097/TA.0b013e318142d146. discussion 143–134.
28. Chen CJ, Tseng YC, Lee TH, et al. Multisection CT angiography compared with catheter angiography in diagnosing vertebral artery dissection. Am J Neuroradiol. 2004;25:769–74.
29. Elijovich L, Kazmi K, Gauvrit JY, Law M. The emerging role of multidetector row CT angiography in the diagnosis of cervical arterial dissection: preliminary study. Neuroradiology. 2006;48:606–12. doi:10.1007/s00234-006-0100-5.
30. Houser OW, Mokri B, Sundt Jr TM, et al. Spontaneous cervical cephalic arterial dissection and its residuum: angiographic spectrum. Am J Neuroradiol. 1984;5:27–34.
31. Sliker CW, Shanmuganathan K, Mirvis SE. Diagnosis of blunt cerebrovascular injuries with 16-mdct: accuracy of whole-body MDCT compared with neck MDCT angiography. Am J Roentgenol. 2008;190:790–9. doi:10.2214/AJR.07.2378.
32. Zuber M, Meary E, Meder JF, Mas JL. Magnetic resonance imaging and dynamic CT scan in cervical artery dissections. Stroke. 1994;25:576–81.
33. Eastman AL, Muraliraj V, Sperry JL, Minei JP. CTA-based screening reduces time to diagnosis and stroke rate in blunt cervical vascular injury. J Trauma. 2009;67:551–6. doi:10.1097/TA.0b013e3181b84408. discussion 555–6.
34. Chen WL, Chern CH, Wu YL, Lee CH. Vertebral artery dissection and cerebellar infarction following chiropractic manipulation. Emerg Med J. 2006;23:e1. doi:10.1136/emj.2004.015636.
35. Cothren CC, Biffl WL, Moore EE, et al. Treatment for blunt cerebrovascular injuries: equivalence of anticoagulation and antiplatelet agents. Arch Surg. 2009;144:685–90. doi:10.1001/archsurg.2009.111.
36. Cothren CC, Moore EE, Biffl WL, et al. Anticoagulation is the gold standard therapy for blunt carotid injuries to reduce stroke rate. Arch Surg. 2004;139:540–5. doi:10.1001/archsurg.139.5.540. discussion 545–6.
37. Engelter ST, Brandt T, Debette S, et al. Antiplatelets versus anticoagulation in cervical artery dissection. Stroke. 2007;38:2605–11. doi:10.1161/STROKEAHA.107.489666.
38. Georgiadis D, Arnold M, von Buedingen HC, et al. Aspirin vs anticoagulation in carotid artery dissection: a study of 298 patients. Neurology. 2009;72:1810–5. doi:10.1212/WNL.0b013e3181a2a50a.
39. •• Kennedy F, Lanfranconi S, Hicks C, et al. Antiplatelets vs anticoagulation for dissection: CADISS nonrandomized arm and meta-analysis. Neurology. 2012;79:686–9. doi:10.1212/WNL.0b013e318264e36b. *Demonstrates equal effectiveness of both antiplatelet and anticoagulation in arterial dissections*.
40. Lyrer P, Engelter S. Antithrombotic drugs for carotid artery dissection. Cochrane Database Syst Rev. 2010; CD000255. doi: 10.1002/14651858.CD000255.pub2.
41. Harrigan MR, Hadley MN, Dhall SS, et al. Management of vertebral artery injuries following nonpenetrating cervical trauma. Neurosurgery. 2013;72 Suppl 2:234–43. doi:10.1227/NEU.0b013e31827765f5.
42. • Engelter ST, Dallongeville J, Kloss M, et al. Thrombolysis in cervical artery dissection–data from the cervical artery dissection and ischaemic stroke patients (CADISP) database. Eur J Neurol. 2012;19:1199–206. doi:10.1111/j.1468-1331.2012.03704.x. *Demonstrates safety of thrombolysis in this patient population*.
43. Engelter ST, Rutgers MP, Hatz F, et al. Intravenous thrombolysis in stroke attributable to cervical artery dissection. Stroke. 2009;40:3772–6. doi:10.1161/STROKEAHA.109.555953.
44. Georgiadis D, Lanczik O, Schwab S, et al. IV thrombolysis in patients with acute stroke due to spontaneous carotid dissection. Neurology. 2005;64:1612–4. doi:10.1212/01.WNL.0000159548.45013.C1.
45. Zinkstok SM, Vergouwen MD, Engelter ST, et al. Safety and functional outcome of thrombolysis in dissection-related ischemic stroke: a meta-analysis of individual patient data. Stroke. 2011;42:2515–20. doi:10.1161/STROKEAHA.111.617282.
46. Cohen JE, Ben-Hur T, Rajz G, et al. Endovascular stent-assisted angioplasty in the management of traumatic internal carotid artery dissections. Stroke. 2005;36:e45–7. doi:10.1161/01.STR.0000158910.08024.7f.

47. Cohen JE, Gomori JM, Grigoriadis S, et al. Intra-arterial thrombolysis and stent placement for traumatic carotid dissection with subsequent stroke: a combined, simultaneous endovascular approach. J Neurol Sci. 2008;269:172–5. doi:10.1016/j.jns.2007.12.013.
48. Cohen JE, Gomori JM, Itshayek E, et al. Single-center experience on endovascular reconstruction of traumatic internal carotid artery dissections. J Trauma Acute Care Surg. 2012;72:216–21. doi:10.1097/TA.0b013e31823f630a.
49. Cohen JE, Gomori JM, Umansky F. Endovascular management of symptomatic vertebral artery dissection achieved using stent angioplasty and emboli protection device. Neurol Res. 2003;25:418–22.
50. Edgell RC, Abou-Chebl A, Yadav JS. Endovascular management of spontaneous carotid artery dissection. J Vasc Surg. 2005;42:854–60. doi:10.1016/j.jvs2005.06.029. discussion 860.
51. Fava M, Meneses L, Loyola S, et al. Carotid artery dissection: endovascular treatment. report of 12 patients. Catheter Cardiovasc Interv. 2008;71:694–700.
52. Lavallee PC, Mazighi M, Saint-Maurice JP, et al. Stent-assisted endovascular thrombolysis versus intravenous thrombolysis in internal carotid artery dissection with tandem internal carotid and middle cerebral artery occlusion. Stroke. 2007;38:2270–4. doi:10.1161/STROKEAHA.106.481093.
53. • Seth R, Obuchowski AM, Zoarski GH. Endovascular repair of traumatic cervical internal carotid artery injuries: a safe and effective treatment option. Am J Neuroradiol. 2013;34:1219–26. doi:10.3174/ajnr.A3337. *Demonstrates safety and efficacy of endovascular repair of arterial dissections in a select population*.
54. Ansari SA, Thompson BG, Gemmete JJ, Gandhi D. Endovascular treatment of distal cervical and intracranial dissections with the neuroform stent. Neurosurgery. 2008;62:636–46. doi:10.1227/01.NEU.0000311350.25281.6B. discussion 636–46.
55. Kansagra AP, Cooke DL, English JD, et al. Current trends in endovascular management of traumatic cerebrovascular injury. J Neurointervention Surg. 2013. doi:10.1136/neurintsurg-2012-010605.
56. Hassan AE, Zacharatos H, Rodriguez GJ, et al. Long-term clinical and angiographic outcomes in patients with spontaneous cervicocranial arterial dissections treated with stent placement. J Neuroimaging. 2012;22:384–93. doi:10.1111/j.1552-6569.2012.00724.x.
57. Pham MH, Rahme RJ, Arnaout O, et al. Endovascular stenting of extracranial carotid and vertebral artery dissections: a systematic review of the literature. Neurosurgery. 2011;68:856–66. doi:10.1227/NEU.0b013e318209ce03. discussion 866.
58. de Barros Faria M, Castro RN, Lundquist J, et al. The role of the pipeline embolization device for the treatment of dissecting intracranial aneurysms. Am J Neuroradiol. 2011;32:2192–5. doi:10.3174/ajnr.A2671.
59. Ducruet AF, Crowley RW, Albuquerque FC, McDougall CG. Reconstructive endovascular treatment of a ruptured vertebral artery dissecting aneurysm using the pipeline embolization device. J Neurointervention Surg. 2013;5:e20. doi:10.1136/neurintsurg-2012-010358.
60. Fields JD, Lutsep HL, Rymer MR, et al. Endovascular mechanical thrombectomy for the treatment of acute ischemic stroke due to arterial dissection. Interv Neuroradiol. 2012;18:74–9.
61. Ioannidis I, Nasis N, Andreou A. Endovascular treatment of ruptured dissecting posterior inferior cerebellar artery aneurysms. Interv Neuroradiol. 2012;18:442–8.
62. Ishihara H, Tateshima S, Jahan R, et al. Endovascular treatment of ruptured dissecting aneurysms of the posterior inferior cerebellar artery. J Neurointervention Surg. 2012;5:557–561. doi:10.1136/neurintsurg-2012-010500.
63. Kashiwazaki D, Ushikoshi S, Asano T, et al. Long-term clinical and radiological results of endovascular internal trapping in vertebral artery dissection. Neuroradiology. 2013;55:201–6. doi:10.1007/s00234-012-1114-9.
64. Kim BM, Shin YS, Kim SH, et al. Incidence and risk factors of recurrence after endovascular treatment of intracranial vertebrobasilar dissecting aneurysms. Stroke. 2011;42:2425–30. doi:10.1161/strokeaha.111.617381.
65. Kocak B, Tureci E, Kizilkilic O, et al. Dissecting aneurysms of posterior communicating artery itself: anatomical, diagnostic, clinical, and therapeutical considerations. Neuroradiology. 2013;55:1103–12. doi:10.1007/s00234-013-1212-3.
66. Lama S, Dolati P, Sutherland GR. Controversy in the management of lenticulostriate artery dissecting aneurysm: a case report and review of the literature. World Neurosurg. 2012. doi:10.1016/j.wneu.2012.12.006.
67. Wang H, Du R, Stary J, et al. Dissecting aneurysms of the posterior cerebral artery: current endovascular/surgical evaluation and treatment strategies. Neurosurgery. 2012;70:1581–8. doi:10.1227/NEU.0b013e31824c00f4. discussion 1588.
68. Yeung TW, Lai V, Lau HY, et al. Long-term outcome of endovascular reconstruction with the pipeline embolization device in the management of unruptured dissecting aneurysms of the intracranial vertebral artery. J Neurosurg. 2012;116:882–7. doi:10.3171/2011.12.JNS111514.
69. Ali MS, Amenta PS, Starke RM, et al. Intracranial vertebral artery dissections: evolving perspectives. Interv Neuroradiol. 2012;18:469–83.
70. Narata AP, Yilmaz H, Schaller K, et al. Flow-diverting stent for ruptured intracranial dissecting aneurysm of vertebral artery. Neurosurgery. 2012;70:982–8. doi:10.1227/NEU.0b013e318236715e. discussion 988–9.