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Carotid and Vertebral Dissection Imaging

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Abstract Carotid or vertebral artery dissection is the result of a tear in the vessel lining wherein the intima separates the media. This creates a false or pseudo lumen, often accompanied by hemorrhage into the arterial wall. Dissection of these craniocervical vessels often manifests with pain alone but, if untreated, may result in severe neurologic compromise. The causes of dissection are multifactorial, including spontaneous, iatrogenic, and traumatic insults. Regardless of etiology, treatment consists primarily of anticoagulation, whereas endovascular therapy is reserved for cases with persistent thrombus or flow limitation. Given the high risk of neurological compromise or death and the propensity of these injuries to occur in younger individuals, early diagnosis of carotid and vertebral artery dissections is critical. Although angiography remains the criterion standard for diagnosis, advances in noninvasive imaging have placed magnetic resonance and computed tomography at the forefront of diagnosis. This article examines the current imaging modalities used to diagnose this under-recognized entity.

Keywords Craniocervical dissection · Vertebral artery · Carotid artery · Magnetic resonance imaging · Magnetic resonance angiography · Computed tomographic angiography · Digital subtraction angiography

Abbreviations

CT Computed tomography
CTA Computed tomographic angiography
DSA Digital subtraction angiography
MR Magnetic resonance
MRA Magnetic resonance angiography
MRI Magnetic resonance imaging
TOF Time-of-flight

Introduction

Dissections of the carotid and vertebral arteries can be broadly categorized on the basis of pathogenesis and location. Traumatic dissections may result from blunt trauma or rapid movement of the head in relation to the neck in any axis [1]. Spontaneous (or nontraumatic) carotid and vertebral artery

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dissections have been estimated to have an annual incidence of 1–1.5 per 100,000 persons, with extracranial segments more prone to dissection than intracranial segments [2]. Iatrogenic dissection may result from catheter or surgical manipulation of vessels in the course of procedures such as angiography and endarterectomy. Spontaneous dissections may not have an identifiable precipitating event; however, predisposing factors such as family history, smoking, and connective tissue disorders may have an association. Although dissection affects all age groups with no known sex-based predilection, this diagnosis should be highly considered for young adults presenting with stroke.

Clinical manifestations of dissection range from minor neck pain to significant neurologic disability and death. Carotid dissection presents initially with neck pain, usually preceding a stroke. This presentation distinguishes this entity from classic stroke, in which a headache usually precedes the ischemic event. Patients with vertebral artery dissections typically present with symptoms of vertigo and, most commonly, ipsilateral facial dysesthesia [3]. Rapid identification of dissection within either the carotid or vertebral artery followed by subsequent treatment could prevent neurologic sequelae and even death. The identification of dissection is heavily dependent on the available noninvasive and/or invasive imaging modalities. There have been several studies using various modalities for diagnosis of carotid or vertebral artery dissection (Table 1 [4•, 5, 6, 7•, 8–12, 13•, 14, 15•, 16, 17, 18•, 19, 20•, 21, 22, 23•]).

Although distinctions between cranial location and pathogenesis are important for determining treatment pathways, for the sake of this article, we focus broadly on the term dissection, paying particular attention to the imaging modalities used to identify craniocervical pathology.

Ultrasonography

The least invasive and most risk-free of the noninvasive imaging modalities is duplex ultrasonography. Dissections that occur in more distal segments of the carotid or vertebral arteries cannot be imaged with ultrasonography. Because the sound waves cannot readily penetrate the bone, this test is primarily limited to assessment of the cervical portions of the carotid and vertebral arteries, where it can be quite useful in detailing flow aberrations, intramural hematoma, luminal thrombus, and mobile flaps [24, 25]. Arterial dissection often presents with a "double lumen" sign, consisting of an echogenic flap that divides a single lumen into a "true" lumen (i.e., the original vessel lumen that is circumferentially lined by intima) and a "false" lumen (i.e., the lumen created by blood flowing into the potential space between intima and media). An eccentric echogenic component may be noted if an intramural hematoma is present. In cases of carotid dissection, velocities within the carotid bulb may decrease and are accompanied by high resistance due to stenosis that yields a biphasic pattern. Compensatory increased blood flow may be seen in the unaffected vertebral artery, with either low or absent flow in the dissected vertebral artery [26•]. Although ultrasonography is not usually utilized in the setting of acute dissection, for example in the emergency department where computed tomographic angiography (CTA) is usually performed in rapid fashion, it can be quite useful as either an orthogonal technique to confirm diagnosis or as a nonionizing means of monitoring dissections over time.

Computed Tomographic Angiography

Because of the widespread availability of high-quality CT scanners and the speed of image acquisition, in most hospital settings, CTA is the most efficient modality for diagnosing dissection. Dissections are easily identified on CTA obtained through 3D images reconstructed from axial source images (Fig. 1a). However, bony regions may create artifact that can obscure pathology, for example, as the vertebral arteries course proximally through the C2-6 foramina transversaria. In such cases, it is important to consult the axial source images, which are more reliable for identifying a dissection. An intimal tear within the vessel is often accompanied by formation of a medial or subendothelial hematoma that is readily identifiable. Typically circular, the vessel lumen becomes irregular and asymmetrical. An intramural hematoma usually manifests as a crescentic hyperdensity or a suboccipital rim with thickening of the vascular wall without a change in the caliber of the vessel. CT perfusion imaging can further management decisions by yielding information regarding the extent to which the dissection impacts flow dynamics (Fig. 1b).

CTA is a relatively safe and well-tolerated modality with cognizance of inevitable exposure to radiation and patient allergies to contrast material. Compared to conventional, catheter-directed digital subtraction angiography (DSA), CTA is a less-invasive study that usually uses less contrast material. At our institution, patients with clinical suspicion of dissection primarily undergo a CT stroke study (Fig. 2), which includes CTA with 3D reconstruction of vessels as well as CT perfusion imaging to elucidate any potential ischemic change.

The limitations of CTA are few but nevertheless worthy of mention. For certain specific groups, such as children, adolescents, or pregnant women, CTA may be relatively contraindicated due to radiation exposure. Furthermore, the low-attenuation crescent that is nonspecific for intramural hematoma can also be seen as an atheromatous plaque, which may yield readings.

Table 1 Clinical and radiological characteristics of patients with intracranial arterial dissection (IAD) in reported series including more than 40 patients

	N	Country origin (department)	Imaging method	Mean age (range)	Sex	Anatomical location	Presenting symptoms
All types							
Yamaura et al. (2000) [5]	357	Japan (neurosurgery survey)	DSA	51 (8–86) years (SAH 53 years, non-SAH 49 years)	Ratio of men to women 2:1; with non-SAH 2·6:1	3 % anterior circulation (SAH 2 %, non-SAH 5 %); 97 % posterior circulation (SAH 98 %, non-SAH 95 %), in VA (261 patients), BA (22 patients), ICA (10 patients), or other artery (29 patients)[a]	SAH (206 patients [58 %]), cerebral ischemia (112 patients [31 %]), headache alone (26 patients [7 %]), other (13 patients [4 %])
Mizutani (2011) [6]	190	Japan (neurosurgery, radiology)	MRA, DSA, or CTA	49 (0–74) years (SAH 52 [0–65] years, non-SAH 45 [22–47] years)	69 % men (SAH 62 %, non-SAH 77 %)[b]	14 % anterior circulation (SAH 11 %, non-SAH 14 %); 88 % posterior circulation (SAH 89 %, non-SAH 86 %), VA (155 dissections), PICA (11 dissections), ACA (11 dissections), BA (10 dissections), MCA (8 dissections)[b]	SAH (108 dissections [52 %]), headache, or cerebral ischemia (98 dissections [48 %])[b]
Ono et al. (2013) [7•]	143	Japan (neurosurgery)	DSA and CT in all patients, MRI in some patients	51 (7–82) years (SAH 53 [31–83] years, non-SAH 48 [10–74] years)	59 % men (SAH 58 %, non-SAH 61 %)	22 % anterior circulation (SAH 15 %, non-SAH 32 %); 78 % posterior circulation (SAH 85 %, non-SAH 68 %), VA (99 patients; 16 [11 %] IADs in the VA were bilateral), ACA (11 patients), MCA (11 patients), ICA (8 patients), BA (7 patients), PICA (5 patients), PCoA (1 patient), PCA (1 patient)	SAH (86 patients [60 %]), headache, or cerebral ischemia (57 patients [40 %])
Kwak et al. (2011)[c] [8]	92	South Korea (radiology)	DSA	51 years[d]	58 % men[d]	24 % anterior circulation (SAH 7 %, non-SAH 44 %); 76 % posterior circulation (SAH 93 %, non-SAH 57 %)	SAH (25 patients [27 %]), SAH and ischemia (3 patients [3 %]), infarction (20 patients [22 %]), other cerebrovascular symptoms (44 patients [48 %])
Metso et al. (2007) [9]	45[e]	Finland (neurology, neurosurgery)	SAH: DSA (50 %) or CTA (50 %); non-SAH: MRA (100 %), MRI (96 %), US (39 %), or CTA (9 %)	46 (21–67) years (SAH 51 [32–67] years, non-SAH 42 [21–56] years)	58 % men (SAH 50 %, non-SAH 65 %)	16 % anterior circulation (SAH 14 %, non-SAH 22 %); 84 % posterior circulation (SAH 86 %, non-SAH 78 %), VA (28 patients; one [2 %] IAD in the VA was bilateral), ICA (5 patients), BA (4 patients), PICA (3 patients), ACA (2 patients), SCA (1 patient), PCA (1 patient), pericallosal artery (1 patient)	SAH (22 patients [49 %]), headache, or cerebral ischemia (23 patients [51 %])[e]
Vertebrobasilar IAD							
Ahn et al. (2012)[c] [10]	210	South Korea (neurosurgery, radiology)	DSA in all; CTA, MRI, or MRA in some	Median 47 (21–80) y (SAH 45 y, non-SAH 48 y)	61 % men	Vertebrobasilar IAD included: 20 (10 %) IAD in the VA were bilateral	SAH (48 patients [21 %]), non-SAH (182 patients [79 %]; ischemia frequency unknown)
Kim et al. (2011)[c] [11]	111	South Korea (neurosurgery, radiology)	DSA	45 (24–78) years	63 % men	Vertebrobasilar IAD included: BA involved (10), PICA involved (47), 8 (7 %) IADs were bilateral	SAH (73 patients [66 %]), ischemia, or headache (38 patients [34 %])
Matsukawa et al. (2012)[c] [12]	103	Japan (neurosurgery)	MRI, MRA, CTA, DSA	53 (IQR 45–66) years (SAH 50 [46–59] years, non-SAH 54 [45–69] years)	69 % men (SAH 77 %, non-SAH 67 %)	Vertebral IAD included: 3 (3 %) IADs were bilateral	SAH (22 patients [21 %]), ischemia, or headache (81 patients [79 %])
	73		Not specified	52 (SD 9) years	55 % men		

Table 1 (continued)

	N	Country origin (department)	Imaging method	Mean age (range)	Sex	Anatomical location	Presenting symptoms
Kashiwasaki et al. (2013) [13•]		Japan (neurosurgery)				Vertebral IAD without PICA involvement	SAH (45 patients [62 %]), non-SAH (28 patients [38 %], asymptomatic, or headache)
Takemoto et al. (2005) [14]	62	Japan (neurosurgery)	DSA, MRI	51 (38–62) years (SAH 57 [54–62] years, non-SAH 48 [38–61] years)[f]	86 % men (SAH 80 %, non-SAH 89 %)[f]	Vertebral IAD	SAH (5 patients [8 %]), headache (8 patients [13 %]), cerebral ischemia (49 patients [79 %])
Shin et al. (2014) [15•]	60	South Korea and USA (neurology, neurosurgery)	DSA, MRA, CTA	48 (SD 19) years	86 % men	Vertebral IAD	SAH (6 patients [10 %]), headache (10 patients [17 %]), cerebral ischemia (44 patients [73 %])
Nakazawa et al. (2011) [16]	47	Japan (neurosurgery)	DSA, MRA, CTA	53 (34–70) years (SAH 53 [34–70] years, non-SAH 52 [39–64] years)	66 % men (SAH 58 %, non-SAH 81 %)	Vertebral IAD	SAH (31 patients [66 %]), headache (10 patients [23 %]), asymptomatic (4 patients [9 %]), other (2 patients [4 %)]
Jin et al. (2009)[c] [17]	42	South Korea (neurology, radiology)	DSA in all; CTA or MRA in some	47 (25–73) years (SAH 47 [25–63] years, non-SAH 47 [36–73] years)	62 % men (SAH 66 %, non-SAH 54 %)	Vertebrobasilar IAD: VA (41 patients), BA (1 patient)	SAH (29 patients [69 %]), cerebral ischemia (3 patients [7 %]), headache or neck pain (8 patients [19 %]), asymptomatic (2 patients [5 %])
Zhao et al. (2014)[c] [18•]	97	China (neurosurgery)	DSA	Median 46 (27–80) years	64 % men	Vertebral IAD	SAH (57 patients [59 %]), symptomatic or unruptured artery (40 patients [41 %])
Vertebrobasilar IAD with SAH							
Nakajima et al. (2010) [19]	109	Japan (neurology, neurosurgery)	DSA, MRA, CTA	Not reported	Not reported	Vertebrobasilar IAD included	SAH (109 patients [100 %])
Zhao et al. (2013)[c] [20•]	57	China (neurology, neurosurgery)	Not reported	Median 48 (27–69) years	51 % men	Vertebral IAD included	SAH (57 patients [100 %])
Vertebrobasilar IAD without SAH							
Kim et al. (2011)[c] [21]	191	South Korea (neurosurgery, radiology)	DSA (92 %), MRA (79 %), CTA (44 %)	49 (21–78) years	67 % men	Vertebrobasilar IAD included: BA (15 patients), PICA (51 patients); 15 (8 %) IADs were bilateral	Cerebral ischemia (110 patients [58 %]), headache alone (81 patients [42 %])
Kai et al. (2011) [22]	100	Japan (neurology, neurosurgery)	MRI	61 (33–83) years	72 % men	Vertebral IAD without SAH	Cerebral ischemia (30 patients [30 %]), headache alone (66 patients [66 %]), mass effect (four patients [4 %])

Table 1 (continued)

	N	Country origin (department)	Imaging method	Mean age (range)	Sex	Anatomical location	Presenting symptoms
Matsukawa et al. (2014)[c] [23•]	77	Japan (neurosurgery)	MRI or MRA (99 %), CTA (60 %), or DSA (23 %)	56 (SD 14) years	70 % men	Vertebrobasilar IAD included: BA involved (eight patients), PICA involved (20 patients)	Cerebral ischemia (33 patients [43 %]), headache or neck pain (27 patients [35 %]), asymptomatic (17 patients [22 %])

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N number of patients, *SAH* subarachnoid hemorrhage, *VA* vertebral artery, *BA* basilar artery, *ICA* internal carotid artery, *MRA* MR angiography, *DSA* digital subtraction angiography, *CTA* CT angiography, *PICA* posterior inferior cerebellar artery, *ACA* anterior cerebral artery, *MCA* middle cerebral artery, *IAD* intracranial artery dissection, *PCoA* posterior communicating artery, *PCA* posterior cerebral artery, *SCA* superior cerebellar artery, *US* ultrasound.

[a] Information about IAD site is missing in 29 patients and there are discrepancies between text and tables.

[b] Numbers and percentages of dissected arteries (206) are presented, not numbers and percentages of patients (190 patients).

[c] These series partly overlap.

[d] Numbers and percentages only reported all patients with intracranial and extracranial dissection (133 patients), and not for subgroup of patients with IAD (92 patients).

[e] 103 patients in total were included in the study, but only 45 patients had pure IAD (the remaining 58 patients had cervical artery dissection with intracranial extension).

[f] Numbers and percentages reported only for 14 patients with aneurysm and surgical treatment.

MRI/MRA

MRI with fat saturation has replaced conventional DSA as the "gold" standard for diagnosis of craniocervical arterial dissection [27]. MRI and MRA have several advantages that make them more desirable for initial evaluation of patients with suspected dissection. MR methods are the least invasive means of monitoring dissections over time and involve no radiation exposure. MRA is especially helpful in younger patients or those with compromised renal function because it does not utilize ionizing radiation or iodinated contrast agents.

MR evaluation of arterial dissection consists of three basic components: (1) diffusion-weighted imaging (DWI) and fluid attenuation inversion recovery (FLAIR) imaging to evaluate for evidence of infarction, potentially as a result of dissection, (2) T1- and T2-based imaging to evaluate for intramural hematoma, and (3) MRA to evaluate the vascular lumen. The classic MRI dissection finding is an eccentric periluminal rim that is indicative of an intramural hematoma. This intramural blood and mural expansion can be readily observed with fat saturation techniques [27].

The age of the dissection will determine its appearance on MRI. In the acute phase, the hematoma consists primarily of deoxyhemoglobin and appears relatively isointense to the surrounding muscle tissue. Subacute hematomas contain both intracellular and extracellular methemoglobin and thus appear hyperintense on both T1- and T2-weighted images. This appearance can persist for several months, after which the hyperintensity fades to isointensity. Intimal flaps can also be seen on MRI (Fig. 3, left), primarily with proton density or T2-weighted images. On long TR sequences, the flap appears as a curvilinear, hypointense line that separates the true lumen from the hematoma.

MRA should accompany MRI evaluation for dissection. MRA can be performed using a variety of techniques, including 2D time-of-flight (TOF), 3D TOF (Fig. 3-right), and phase-contrast. 2D TOF has the advantage of imaging longer arterial segments in a relatively short time, but it is subject to complex flow-related signal artifacts. 3D TOF partially overcomes flow-related artifacts and yields better spatial resolution, but the image acquisition time is longer. TOF sequences also tend to have the least amount of background-signal suppression and, as such, may make the hyperintensity of an intramural thrombus more apparent on source images. Often, an intramural hematoma may be seen as a periarterial rim between the high intensity of the flow and the low intensity of the soft tissues surrounding the vessel. Phase-contrast studies require the use of gadolinium-based dyes but yield high quality images, especially in cases wherein there may be nearby metal artifact.

MR detection of intramural hematomas has been studied extensively, with widely ranging sensitivity. The variability has largely been ascribed to age differences of the hematoma

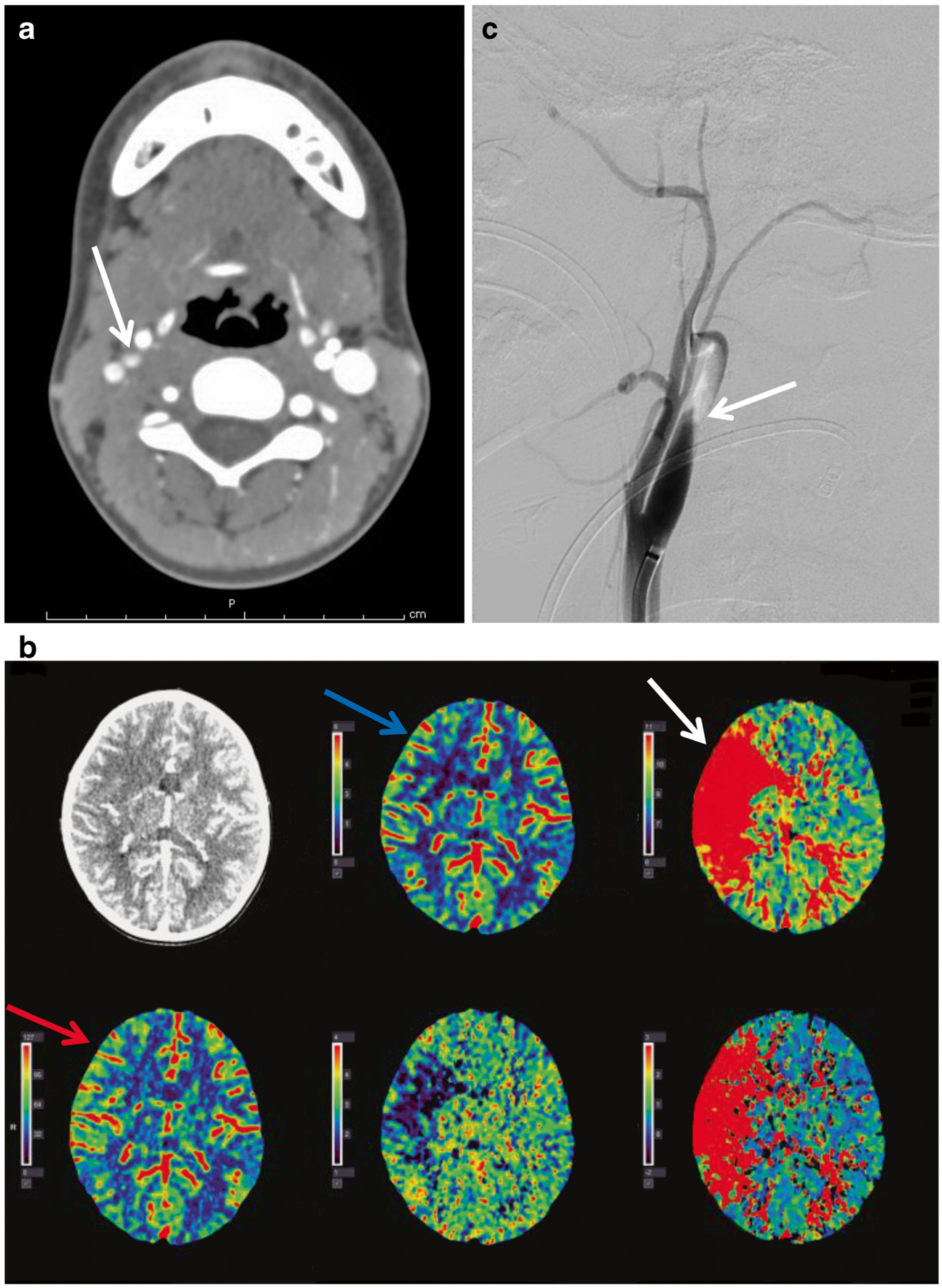

Fig. 1 **a** Computed tomographic (CT) angiographic image, axial view, showing crescentic hyperdensity (*arrow*). **b** CT perfusion mapping, axial view, showing increased time-to-peak (*white arrow*) but reasonable cerebral blood flow (*red arrow*) and volume (*blue arrow*). **c** Digital subtraction angiogram, lateral view, showing tapering of vessel, also known as the "flame sign" (*arrow*), which is indicative of a dissection

as well as the use of fat-suppression imaging. In general, detection of dissections is more difficult in vertebral arteries than in carotid arteries. Furthermore, MRI is less sensitive in detecting those of intracranial origin compared to cervical dissections.

Digital Subtraction Angiography

Although noninvasive imaging modalities have become quite good at detecting dissections, DSA remains the standard because it yields both high spatial and temporal resolution of blood flow in the vessels. Although invasive, angiography removes the guesswork from the questionable findings on noninvasive modalities that may stem from artifact (motion or otherwise) and/or the quality of study. Classic signs of dissection that are appreciated on DSA are the "double-barrel sign" or "double-lumen sign," which primarily result from subadventitial dissections. Angiographically, the sign is appreciated when there is a communication between true and false (pseudo) lumen at the distal end of the false lumen. Although an intimal flap or the double-lumen sign is a specific

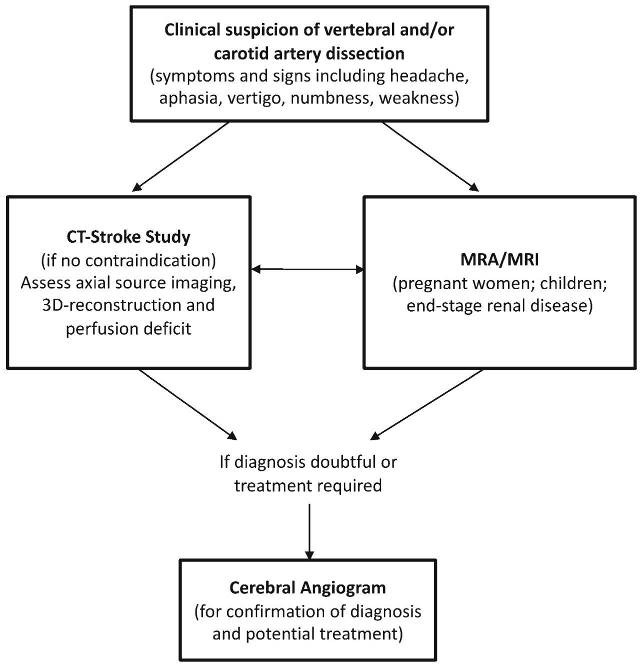

Fig. 2 Algorithm for diagnostic neuroimaging evaluation of craniocervical dissection

finding for arterial dissection, it is seen in less than 10 % of patients [28]. Contrast stasis and thrombus in a dissected artery or emboli in the distal branches are seen in only approximately 10 % of patients [29].

The "pearl and string" sign, not to be confused with the "string of pearls" sign associated with fibromuscular dysplasia, is another angiographic finding in cases of dissection but is rarer than the "double-lumen sign." The pearl and string sign represents associated stenosis and proximal dilatation within the dissected vessel. However, the most common finding on DSA is an irregularly shaped vessel with noticeable tapering. The sign associated with this finding is the "flame sign" (Fig. 1c) or "rat-tail" narrowing, which occurs when there is a tapered occlusion sparing the carotid bulb.

Limitations of DSA lie in its inability to assess arterial wall thickness, as it only truly images the vessel lumen. In the cases wherein the dissection is subadventitial, there may be no significant narrowing of the vessel lumen. DSA also lacks the capacity for simultaneous representation of surrounding vessels in the brain; although with advanced 3D rotational angiography and reconstruction, this drawback can be overcome. Furthermore, DSA is considered an invasive measure that costs more than noninvasive modalities and has associated risks, such as perforation, hematoma, stroke, and renal injury.

Conclusion

Craniocervical dissections are tears in the vessel wall that may alter blood flow or serve as a nidus for thrombus formation. Whereas extracranial dissections are most often nontraumatic or spontaneous and have an underlying vasculopathy associated with them, intracranial dissections generally result from a traumatic insult, with higher incidence of iatrogenic dissection from endovascular procedures seen in recent years. Although uncommon, dissection is a cause of stroke in all age groups with potential for even death. Therefore, rapid identification of dissection is vital to preserve neurological function. Advances in noninvasive imaging and imaging protocols have significantly improved in recent years, allowing for early recognition of dissection and more efficient treatment. The patient's clinical examination findings and history should be taken into consideration before selecting the noninvasive imaging modality. DSA remains the gold standard for identification of dissection, but MRI/MRA and CTA have an increasing role in modern diagnostic algorithms because they yield nearly equivalent information without significant risk of harm or use of resources.

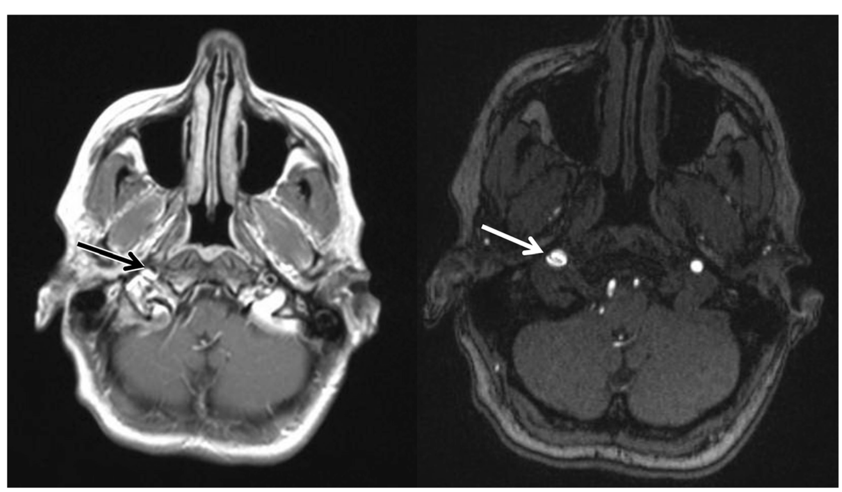

Fig. 3 T1-weighted flow correction magnetic resonance (MR) image, axial view, with gadolinium enhancement, showing a periluminal rim indicative of an intramural hematoma (*arrow*) (*left*). Axial view—3D Time-of-flight MR angiogram, showing vessel contour abnormality suggesting a possible dissection (*arrow*) (*right*)

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Compliance with Ethical Standards

Conflict of Interest Hakeem J. Shakir, Jason M. Davies, and Hussain Shallwani declare that they have no conflict of interest.

Adnan H. Siddiqui declares financial interests in Buffalo Technology Partners Inc., Cardinal, International Medical Distribution Partners; Medina Medical Systems, Neuro technology Investors, StimSox, and Valor Medical. He serves as a consultant to Amnis Therapeutics Ltd., Cerebrotech Medical Systems Inc., CereVasc LLC, Codman, Corindus Inc., Covidien (acquired by Medtronic), GuidePoint Global Consulting, Lazarus (acquired by Medtronic), Medina Medical (acquired by Medtronic), Medtronic, MicroVention, Neuravi, Penumbra, Pulsar Vascular, Rapid Medical, Rebound Medical, Reverse Medical (acquired by Medtronic), Silk Road Medical Inc., Stryker, The Stroke Project Inc., Three Rivers Medical Inc., and W.L. Gore & Associates. He is aprincipal investigator or serves on the National Steering Committee for the following trials: Covidien SWIFT PRIME, LARGE, Medtronic SWIFT DIRECT, MicroVention CONFIDENCE, MicroVention FRED, Penumbra 3D Separator, Penumbra COMPASS, Penumbra INVEST, and POSITIVE Trial. He is a member of the board of the Intersocietal Accreditation Committee. (Dr. Siddiqui receives no consulting salary arrangements. All consulting is per project and/or per hour.)

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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

1. Hufnagel A, Hammers A, Schonle PW, Bohm KD, Leonhardt G. Stroke following chiropractic manipulation of the cervical spine. J Neurol. 1999;246:683–8.
2. Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Engl J Med. 2001;344:898–906.
3. Kim YK, Schulman S. Cervical artery dissection: pathology, epidemiology and management. Thromb Res. 2009;123:810–21.
4.• Debette S, Compter A, Labeyrie MA, Uyttenboogaart M, Metso TM, Majersik JJ, et al. Epidemiology, pathophysiology, diagnosis, and management of intracranial artery dissection. Lancet Neurol. 2015;14:640–54. **This is a comprehensive review of epidemiology, pathophysiology, diagnosis, management and outcomes of spontaneous intracranial artery dissection, along with consensus statements from experts in the field**.
5. Yamaura A, Ono J, Hirai S. Clinical picture of intracranial nontraumatic dissecting aneurysm. Neuropathology. 2000;20:85–90.
6. Mizutani T. Natural course of intracranial arterial dissections. J Neurosurg. 2011;114:1037–44.
7.• Ono H, Nakatomi H, Tsutsumi K, Inoue T, Teraoka A, Yoshimoto Y, et al. Symptomatic recurrence of intracranial arterial dissections: follow-up study of 143 consecutive cases and pathological investigation. Stroke. 2013;44:126–31. **This is a detailed series and analysis of 143 cases with intracranial cerebral arterial dissection. The results suggest that intracranial cerebral arterial dissection may carry a high risk of recurrence; histopathological characteristics may help in treatment decision and follow-up strategies**.
8. Kwak JH, Choi JW, Park HJ, Chae EY, Park ES, Lee DH, et al. Cerebral artery dissection: spectrum of clinical presentations related to angiographic findings. Neurointervention. 2011;6:78–83.
9. Metso TM, Metso AJ, Helenius J, Haapaniemi E, Salonen O, Porras M, et al. Prognosis and safety of anticoagulation in intracranial artery dissections in adults. Stroke. 2007;38:1837–42.
10. Ahn SS, Kim BM, Suh SH, Kim DJ, Kim DI, Shin YS, et al. Spontaneous symptomatic intracranial vertebrobasilar dissection: initial and follow-up imaging findings. Radiology. 2012;264:196–202.
11. Kim BM, Shin YS, Kim SH, Suh SH, Ihn YK, Kim DI, et al. Incidence and risk factors of recurrence after endovascular treatment of intracranial vertebrobasilar dissecting aneurysms. Stroke. 2011;42:2425–30.
12. Matsukawa H, Shinoda M, Fujii M, Takahashi O, Murakata A, Ishikawa R. Differences in vertebrobasilar artery morphology between spontaneous intradural vertebral artery dissections with and without subarachnoid hemorrhage. Cerebrovasc Dis. 2012;34:393–9.
13.• Kashiwazaki D, Ushikoshi S, Asano T, Kuroda S, Houkin K. Long-term clinical and radiological results of endovascular internal trapping in vertebral artery dissection. Neuroradiology. 2013;55:201–6. **This is the largest series reviewing the long-term clinical and imaging outcomes of vertebral artery dissection treated with endovascular internal trapping. The results show that endovascular internal trapping is a stable and durable treatment option for vertebral artery dissection**.
14. Takemoto K, Abe H, Uda K, Inoue T. Surgical treatment of intracranial VA dissecting aneurysm. Acta Neurochir Suppl. 2010;107:51–6.
15.• Shin DH, Hong JM, Lee JS, Nasim R, Sohn SI, Kim SJ, et al. Comparison of potential risks between intracranial and extracranial vertebral artery dissections. Eur Neurol. 2014;71:305–12. **This study retrospectively reviewed data for consecutive patients with intracranial or extracranial vertebral artery dissections to identify potential risks. The results indicate that risks of dissection are different in the intracranial versus the extracranial vertebral artery**.
16. Nakazawa T, Takeichi Y, Yokoi T, Fukami T, Jito J, Nitta N, et al. Treatment of spontaneous intradural vertebral artery dissections. Neuroradiol J. 2011;24:699–711.
17. Jin SC, Kwon DH, Choi CG, Ahn JS, Kwun BD. Endovascular strategies for vertebrobasilar dissecting aneurysms. AJNR Am J Neuroradiol. 2009;30:1518–23.
18.• Zhao KJ, Zhao R, Huang QH, Xu Y, Hong B, Fang YB, et al. The interaction between stent(s) implantation, PICA involvement, and immediate occlusion degree affect symptomatic intracranial spontaneous vertebral artery dissection aneurysm (sis-VADA) recurrence after reconstructive treatment with stent(s)-assisted coiling. Eur Radiol. 2014;24:2088–96. **This study identified the risk factors of recurrence after reconstructive treatment of symptomatic intracranial spontaneous verebral artery dissection aneurysms. The authors identified stent implantation, posterior inferior cerebellar artery involvement and immediate occlusion degree as potential factors associated with recurrence of dissection aneurysms**.

19. Nakajima S, Tsukahara T, Minematsu K. A study of vertebrobasilar artery dissection with subarachnoid hemorrhage. Acta Neurochir Suppl. 2010;107:45–9.
20.• Zhao KJ, Fang YB, Huang QH, Xu Y, Hong B, Li Q, et al. Reconstructive treatment of ruptured intracranial spontaneous vertebral artery dissection aneurysms: long-term results and predictors of unfavorable outcomes. PLoS One. 2013;8:e67169. **This study evaluated the outcomes after endovascular treatment of ruptured intracranial spontaneous vertebral artery dissection aneurysms. The authors concluded that immediate obliteration grade was affected by the involvement of the posterior inferior cerebellar artery, size of lesions, number of stents and coil types, and was the only risk factor for angiographic recurrence**.
21. Kim BM, Kim SH, Kim DI, Shin YS, Suh SH, Kim DJ, et al. Outcomes and prognostic factors of intracranial unruptured vertebrobasilar artery dissection. Neurology. 2011;76:1735–41.
22. Kai Y, Nishi T, Watanabe M, Morioka M, Hirano T, Yano S, et al. Strategy for treating unruptured vertebral artery dissecting aneurysms. Neurosurgery. 2011;69:1085–91. discussion 1091-1082.
23.• Matsukawa H, Shinoda M, Fujii M, Takahashi O, Uemura A, Niimi Y. Basilar extension and posterior inferior cerebellar artery involvement as risk factors for progression of the unruptured spontaneous intradural vertebral artery dissection. J Neurol Neurosurg Psychiatry. 2014;85:1049–54. **This study evaluates the association of vertebrobasilar morphologies and clinical characteristices with the progression of sponatenous intradural vertebral artery dissections that are treated conservatively. The results suggest that basilar extension and posterior inferior cerebeller artery involvement may be associated with progression of dissection**.
24. Gardner DJ, Gosink BB, Kallman CE. Internal carotid artery dissections: duplex ultrasound imaging. J Ultrasound Med. 1991;10:607–14.
25. Nebelsieck J, Sengelhoff C, Nassenstein I, Maintz D, Kuhlenbaumer G, Nabavi DG, et al. Sensitivity of neurovascular ultrasound for the detection of spontaneous cervical artery dissection. J Clin Neurosci. 2009;16:79–82.
26.• Mitsumura H, Miyagawa S, Komatsu T, Hirai T, Kono Y, Iguchi Y. Clinical characteristics of intracranial reversed vertebral artery flow evaluated by transcranial color flow imaging. J Stroke Cerebrovasc Dis. 2015;24:1775–80. **This is an experimental study investigating the prevalence of reversed verebral artery flow using transcranial color flow imaging. The authors identified vertebral artery dissection as a plausible cause of stroke in patients with intracranial reversed vertebral artery flow**.
27. Zohrabian D. Carotid artery dissection workup http://emedicine.medscape.com/article/757906-workup#showall Accessed 5 Aug 2016. Medscape 2015.
28. Thanvi B, Munshi SK, Dawson SL, Robinson TG. Carotid and vertebral artery dissection syndromes. Postgrad Med J. 2005;81:383–8.
29. Kochan JP, Kanamalla US. Imaging in carotid and vertebral artery dissection http://emedicine.medscape.com/article/417341-overview#a7 Accessed 5 Aug 2016. Medscape 2015.