Multimodality evaluation of craniocervical arterial dissection: A pictorial review.

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Learning objectives

1. To illustrate the most frequent imaging findings in internal carotid and vertebral artery dissection studied by ultrasound, computed tomography, magnetic resonance and digital subtraction angiography.
2. To review each modality's strengths and limitations and suggest how to avoid the most common pitfalls.
3. To highlight the importance of an accurate and early diagnosis and to name the most important differential diagnosis.
Background

Spontaneous craniocervical arterial dissection is rare, with an estimated incidence of 5 per 100,000 per year\(^1\). Nevertheless, it is an important cause of stroke, particularly in young patients.

Craniocervical arterial dissection involves the separation of artery layers caused by a primary intimal tear and subsequent blood penetration, or by a primary intramural hemorrhage of the vasa vasorum. The dissection extends in the same direction as blood flow (cranially). Commonly, an intramural hematoma compresses the artery’s lumen increasing its external diameter. If the dissection extends to the adventitial layer it can form a dissecting aneurism (pseudoaneurism) which, in turn, can facilitate thrombus formation and distal thromboembolism.

Dissections can be either traumatic (after a severe trauma) or spontaneous. In spontaneous dissection, history of a trivial trauma, such as vomiting, exercise or cervical manipulation, can be a precipitating factor. Connective tissue disorders, most frequently fibromuscular dysplasia, have been found in these patients. Other factors associated with dissection have been reported (hypertension, alcohol and tobacco use, infections, migraine). Some studies found a male gender is also more frequently implicated (up to two thirds\(^2\)).

Data from a large retrospective study\(^3\) reported spontaneous cervical artery dissection most frequently involving the internal carotid artery (68%) followed by the vertebral artery (27%), and both in a minority of patients (5%). Anatomic distribution favors extracranial segments of both internal carotid artery and vertebral artery, which can be explained by their greater mobility\(^4\). Internal carotid dissection usually spares the carotid bulb and tends not to extend distally from its entrance into the petrous bone. Vertebral artery dissection is more often found in the pars transversaria (V2) or the atlas loop (V3)\(^3\).

Clinical manifestations are diverse and range from local signs and symptoms to carotid or posterior territory ischemia in, respectively, carotid or vertebral artery dissection. Therefore, imaging plays an essential role in its diagnosis or exclusion.

Improvements in neurovascular imaging and increased awareness of its clinical manifestations allow an increased recognition of the role of dissection in stroke.
Findings and procedure details

Generally speaking, in all modalities, the most important imaging findings include mural hematoma, thickening of the arterial wall, intraluminal thrombus, intimal flap, double lumen and eccentric narrowing of the lumen.

1- Digital Subtraction Angiography

Digital subtraction angiography has been considered the gold standard in diagnosis of craniocervical arterial dissection. However, it can only be used to study the arterial lumen and not its wall. Furthermore, digital subtraction angiography is invasive and is currently being replaced by other non-invasive imaging modalities.

Imaging findings:

- Long, tapered and irregular stenosis distal to the carotid bulb ("string" sign) is frequently the only present sign.
- Stenosis with distal dilatation ("string and pearl" sign).
- Tapered occlusion (Fig. 1 on page 9, Fig. 2 on page 9).
- Double lumen or intimal flap confirm the diagnosis, though, are rarely observed.

2- Ultrasound with Color Doppler

Ultrasound is a cheap, non-invasive and readily available exam. Nevertheless, intracranial segments cannot be optimally imaged and it is operator-dependent.

High frequency linear transducers (4-8 Mhz) are used to study the proximal internal carotid artery wall, while low frequency transducers are used to study the distal parts. The use of low frequency transducers lowers the ability to detect wall abnormalities.

When carotid artery dissection causes ischemia, color Doppler US sensitivity is 95%, reducing to 71% in cases without ischemic events. Color Doppler US sensitivity to detect vertebral artery dissection was measured at 75%. V2 and V3 segments are frequently difficult to study and, in those cases, diagnosis relies on indirect hemodynamic changes.

Imaging findings:
• Thickened hypoechoic vessel wall corresponding to hematoma and/or thrombus, (which may be hyperchoic if acute).
• Intimal flap separating two lumina, although highly specific, is infrequent.
• Arterial lumen tapering stenosis or occlusion (Fig. 3 on page 10).
• Enlarged vessel diameter.

Hemodynamic changes:

• Diminished proximal flow velocity, especially diastolic flow, with high resistance waveform (Fig. 4 on page 11), however this occurs in stenosis or occlusion of any etiology\(^6\).
• Diminished or absent distal flow (Fig. 5 on page 12).
• Focal increased flow velocity in stenotic segment (Fig. 6 on page 13).
• True and false lumina with different flow (Fig. 7 on page 14).

Other conditions associated with internal carotid artery increased flow (internal carotid artery redundancies, fibromuscular dysplasia, vasospasm, brain arteriovenous malformations, carotid cavernous fistulas, persistent trigeminal artery, anemia, hyperthyroidism) or diminished flow (occlusion of the carotid siphon, severe stenosis/occlusion of the intracranial internal carotid artery, occlusion of the M1 middle cerebral artery) are important pitfalls and have to be excluded. Ultrasound is useful to assess artery repermeabilization after therapy (Fig. 6 on page 13, Fig. 7 on page 14).

3- Computed Tomography

Computed tomography angiography provides high spatial resolution with the ability to make multiplanar and 3D reconstructions, although it depends on correct imaging acquisition. The delay between injection time and image acquisition should be optimal to minimize venous enhancement (Fig. 11 on page 17) and potential false positive results (especially concerning the vertebral venous plexus). Cumulative radiation dose may also be a concern. Unenhanced CT helps to document associated ischemic and hemorrhagic events.

Its diagnostic accuracy was found to be comparable to digital subtraction angiography for cervical internal carotid dissections\(^7\).

When compared to magnetic resonance (with angiography) it seems to be able to identify more dissection signs\(^8\) (namely pseudoaneurisms, which can change therapeutic options), particularly when vertebral artery dissection is involved (due to its smaller size).
The CT protocol we use for cervical artery dissection includes, besides unenhanced brain CT, a volumetric acquisition after contrast injection (60cc at 4cc/second) with ROI located in the aortic arch. The scan starts 3 to 4 seconds after reaching 120 Hounsfield units threshold. Post processing includes multiplanar and tridimensional reconstructions.

Imaging findings:

- Spontaneous hyperdense crescent shape area corresponding to wall hematoma sometimes appears in unenhanced scans of acute dissections. Standard CT angiography window settings don't allow easy differentiation of wall hematoma from the surrounding soft tissue.
- Eccentric narrow lumen associated with crescent-shaped mural thickening (Fig. 10 on page 16, Fig. 11 on page 17). The "target" sign, which adds peripheral enhancement (probably due to vasa-vasorum enhancement) to the previous features, is highly specific.
- Intimal flap separating two lumina.
- Progressive lumen narrowing and occlusion (Fig. 12 on page 18, Fig. 13 on page 19).
- Dissecting pseudoaneurism (Fig. 12 on page 18, Fig. 13 on page 19).
- Enlarged vessel diameter (Fig. 11 on page 17).

Artifacts caused from bones or metallic material have to be accounted for to correctly interpret CT angiography images. Other conditions (such as absence or carotid hypoplasia, fibromuscular dysplasia, atherosclerosis and vasculitis) have overlapping signs with dissection.

4- Magnetic Resonance

Magnetic resonance imaging can provide cross-sectional images to study the vessel wall and assess the presence of intramural hematoma. To study the arterial lumen it is possible to use magnetic resonance angiography with contrast enhancement and without contrast (time-of-flight).

Both MR imaging and angiography showed better results in accessing internal carotid artery dissection (similar to conventional angiography) than vertebral artery dissection\(^9\).

The MR protocol we use for cervical artery dissection, besides brain MRI, includes T1-weighted fat-suppression images of the neck (TR/TE: 553/20; FOV: 13.2 cm; Matrix: 312/206) and either three dimensional phase contrast MRA of the neck (TR/TE: 15/3.9;
FOV: 30 cm; Matrix: 256/122) or three dimensional time-of-light MRA of the neck (TR/TE: 23/6.9; FOV: 16.5 cm; Matrix: 248/93).

Imaging findings:

- Narrow eccentric flow void (Fig. 14 on page 20, Fig. 15 on page 21, Fig. 16 on page 22, Fig. 17 on page 23), although it may not be present in cases with occlusion or slow flow. Furthermore flow void narrowing can be encountered in other conditions.
- Increase of artery's external diameter (Fig. 15 on page 21, Fig. 16 on page 22)
- Intramural hematoma changes its appearance though time. It is hyperintense on T1 sequences in a subacute stage (between 7 and 60 days after the primary event Fig. 14 on page 20, Fig. 16 on page 22, Fig. 17 on page 23) and more clearly visualized in T1 sequences with fat saturation. In the early (Fig. 15 on page 21) and chronic (6 months) stage it is usually isointense to surrounding tissue.
- Intimal flap can be viewed as a curvilinear hypointense image separating the two lumina.
- MR angiography helps stenosis, occlusion (Fig. 18 on page 24) and pseudoaneurism detection.

If there are gaps between imaging slabs, short dissections may be missed. Flow voids can be absent not only in occluded segments but also when there is a slow flow (Fig. 19 on page 25). A poststenotic laminar flow in the horizontal part of the internal carotid artery ("train track" sign Fig. 20 on page 26) should not be mistaken for internal carotid artery dissection extension.

Vertebral artery dissection MR imaging poses various challenges. V1 is frequently excluded from the studied volume. Inflow enhancement in the venous plexus may mimic subacute hematoma in V2. Contrast enhanced MR angiography sometimes produces blurring/loss of signal intensity ("feathering" artifact), particularly in young patients, which may mimic vertebral artery dissection.

**Differential Diagnosis**

Internal carotid artery hypoplasia or absence may mimic, respectively, long stenosis or occlusion. Absence or hypoplasia of the ipsilateral carotid canal is useful to diagnose this developmental anomaly.
Atherosclerosis courses with vessel lumen narrowing and, sometimes, increased vessel diameter (positive remodelation). However, it involves the carotid bifurcation and carotid bulb has at least a few calcifications and generally occurs in older patients. An ulcerated plaque may mimic an intimal flap.

Fibromuscular dysplasia is an angiopathy that affects medium-sized arteries, predominantly in young women. In this patients renal involvement occurs in 60-75% and cerebrovascular involvement in 25-30%. It should be noted that fibromuscular dysplasia is associated with increased risk of craniocervical artery and coronary artery dissections. Imaging studies may show multifocal focal or long stenosis with adjacent dilatations ("string of beads" sign).

Takayasu arteritis is a chronic large-vessel arteriopathy that mainly affects the aorta and its branches, as well as the pulmonary arteries. Subclavian artery and common carotid artery are often bilaterally affected, showing regular wall thickening. Other luminal abnormalities can be found such as stenosis, occlusion and aneurysm.

Regular increased thickness of carotid artery wall is frequently found after radiation therapy.

Moyamoya disease is a progressive, occlusive disease of the cerebral vasculature with particular involvement of the circle of Willis. It is characterized by intimal thickening in the walls of the terminal portions of the internal carotid vessels bilaterally.
**Fig. 1:** 37 year old woman with headache and altered state of consciousness. Anterior-posterior projection angiogram shows irregular stenosis with tapered occlusion of the left vertebral artery (arrow).

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Fig. 2: 37 year old woman with headache and altered state of consciousness. Lateral projection angiogram shows irregular stenosis with tapered occlusion of the left vertebral artery (arrow).

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Fig. 3: 52 year old male with left hemiparesis. Right internal carotid artery progressive lumen tapering and occlusion (arrow).

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**Fig. 4:** Right common carotid artery showing high resistance waveform. Notice the low diastolic flow (arrows).

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Fig. 5: 52 year old male with left hemiparesis. Right internal carotid artery occlusion showing absence of color Doppler flow and respective pulse waveform in dissected segment (arrow).
**Fig. 6:** Right internal carotid artery stenosis (arrow in B) with focal increased flow velocity (arrow in A).

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**Fig. 7:** Right internal carotid artery dissection with two lumina (white arrows).

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**Fig. 8:** 52 year old male patient with left hemiparesis 2 days after the exam shown in Fig. 5. Right internal carotid artery partial repermeabilization, with a high resistance waveform.

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**Fig. 9:** 52 year old male patient with left hemiparesis 24 days after the exam shown in Fig. 5. Right internal carotid artery complete repermeabilization.

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Fig. 10: 37 year old woman with headache and altered state of consciousness. CT angiography axial slice shows left vertebral artery eccentric narrow lumen associated with crescent-shaped mural thickening (arrow).

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Fig. 11: 40 year old man with Horner Syndrome. CT angiography axial slice shows left internal carotid artery eccentric narrow lumen associated with crescent-shaped mural thickening (black arrow) and enlarged vessel diameter (blue line). Notice the venous contamination in the left internal jugular vein (white arrow).

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Fig. 12: 40 year old man with Horner Syndrome. CT angiography sagittal MIP slab shows left internal carotid artery progressive lumen narrowing and occlusion (white arrow) and pseudoaneurism (blue arrow).

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Fig. 13: 40 year old man with Horner Syndrome. CT angiography 3D render in a coronal oblique view shows left internal carotid artery progressive lumen narrowing and occlusion (white arrow) and pseudoaneurism (blue arrow).

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**Fig. 14:** 41 year old female with occipital headache. T1 with fat suppression (SPIR) axial image shows left vertebral artery with a narrow eccentric flow void surrounded by a hyperintense crescent shaped intramural hematoma (arrow).

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**Fig. 15:** 40 year old man with Horner Syndrome. T1 with fat suppression (SPIR) axial image shows left internal carotid artery with a narrow eccentric flow void surrounded by a slightly hyper almost isointense (acute) crescent shaped intramural hematoma (arrow). The left internal carotid artery has an enlarged external diameter when compared to the contralateral artery.

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**Fig. 16:** 52 year old male with left hemiparesis. T1 with fat suppression (SPIR) axial image shows right internal carotid artery with a narrow eccentric flow void surrounded by a hyperintense crescent shaped intramural hematoma (arrow).

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**Fig. 17:** 49 year old male with stroke. T1 with fat suppression (SPIR) axial image shows left vertebral artery with a narrow eccentric flow void surrounded by a hyperintense crescent shaped intramural hematoma (arrow).

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Fig. 18: 37 year old woman with headache and altered state of consciousness. 3D anterior posterior (coronal) view MR angiography (time of flight) shows occlusion of a dissected left vertebral artery (arrow).

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Fig. 19: 40-year-old man with Horner Syndrome and left internal carotid artery dissection. T1 with fat suppression (SPIR) axial image (A) shows left internal carotid artery bulb without flow void (white arrow), however in axial CT angiography image (B) the left internal carotid artery bulb shows contrast enhancement (blue arrow).

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Fig. 20: 40-year-old man with Horner Syndrome and left internal carotid artery dissection. T1 with fat suppression (SPIR) axial image (A) shows train track sign (white arrow) due to poststenotic laminar flow in the horizontal part of the left internal carotid artery, while
in axial CT angiography image the horizontal part of the left internal carotid artery is normally enhanced (blue arrow).

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Conclusion

Knowing the spectrum of imaging findings in ultrasound, computed tomography, magnetic resonance and digital subtraction angiography and understanding the potential pitfalls of each modality allows one to achieve an accurate diagnosis and therefore to reduce the risk of stroke and long term sequelae by administering proper and early treatment.
References


