Cervical arteries dissections: Imaging findings and pitfalls

Poster No.: C-1810
Congress: ECR 2010
Type: Educational Exhibit
Topic: Head and Neck
Authors: O. S. R. Soares¹, M. S. R. Soares¹, J. Savatovsky²; ¹Presidente Prudente/BR, ²Paris/FR
Keywords: supra aortic vessels dissections, MRI, CT angiography
DOI: 10.1594/ecr2010/C-1810

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method is strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

• To review and illustrate the most frequent findings of cervical arteries dissections in different imaging modalities (US, CTA, MRA and DSA).

• To display the differential diagnoses and pitfalls.

• To describe important technical aspects to improve the diagnosis.

• To show the most common and severe complication.

Background
Carotid and vertebral dissection is defined by the presence of hemorrhage into the vessel wall. It usually arises from an intimal tear.

Cervical arteries dissections (CAD) have a wide spectrum of precipitating factors, symptoms and signs, leading the patients to different specialists (ER physician, neurologist, ophthalmologist, ENT specialist, rheumatologist...).

A high grade of suspicion is needed in order to make the diagnosis, as overdiagnosis may result to a needless and potentially hazardous treatment. Urgent treatment is required in order to avoid severe complication, so an early and accurate diagnosis is mandatory.

CAD account for only about 2% of all ischemic strokes, but they are the most frequent cause in young and middle-aged patients (10 to 25% of such cases).

Dissections can occur after a trauma or could be spontaneous. For the latest, there may be an underlying cause like mechanical factor, unknown arterial disease or both. Recently a large number of publications have been show that inflammatory biological markers are elevated in spontaneous dissections, suggesting that spontaneous dissections may result from arterial inflammation.

Many vascular risk factors and diseases are thought to be associated with CAD like: Hypertension, diabetes mellitus, smoking status, hyperlipidaemia, oral contraceptives, cystic medial necrosis, Marfan syndrome and Ehlers-Danlos syndrome type IV.

The symptoms and signs of CAD have a wide spectrum. Those related with internal carotid are headache or neck pain at the side of dissection without arm irradiation, Horner's syndrome and cranial nerves palsies; and those related with vertebral are occipital headache or neck pain, symptoms of vertebrobasilar TIA or infarct, amnesia, tinnitus, nausea and vomiting.

Imaging findings OR Procedure details

THE MOST COMMON IMAGING FINDINGS ARE:

- Irregular narrowing of the lumen,
- Thickening of the arterial wall,
- Intramural hematoma,
- Flap and double lumen,
• Occlusion.

LOCATION:

• Internal carotid artery (ICA) dissections generally occur a few centimeters above carotid bulb.
• Common carotid artery involvement is rare. If present, associated aortic dissection has to be ruled out.
• In vertebral artery (VA), the most common site is at the level of V1-V2.
• Several vessels may simultaneously or successively be involved.

ULTRASOUND

US is a cheap exam and easily available. However, it is operator-depend and cannot show dissections of skull base (fig 1 on page 13).

• Recent intramural hematoma is usually echogenic (fig 2 on page 14). Subacute and chronic may be hypoechoic (fig 3 on page 15),
• Abrupt smooth tapering of arterial lumen (fig 2),
• US can directly assess artery wall and display the increase vessel diameter (figs 2 and 3),
• The most specific sign is the echogenic intimal flap. However it’s present in a minority of cases.
  Hemodynamic features:
• Local increase in flow velocity,
• Slow flow and high resistance proximal to the dissection,
• Transcranial Doppler can display findings lying distal to the site of dissection.

NON-ENHANCED CT

• Usually no abnormality is seen in arteries in a non-enhanced CT,
• Sometimes a hyperdense mass can be present in acute dissection, that represents intramural hematoma (fig 4 on page 16).
CT ANGIOGRAPHY (CTA)

- CTA can show the irregular narrowing of the luminal diameter (figs 1, 5, 6, 7, 16, 21 and 50),
- Intramural hematoma is generally iso or hypodense and thus could be difficult to see (figs 5, 6 and 7).
- The vessel outer limits can usually be delineated thanks to its natural contrast with surrounding fat.
- A thin rim of contrast-enhancement surrounding intramural hematoma, probably due to enhancement of vasa vasorum can indirectly assess the thickening of the arterial wall (figs 7 on page 19 and 50 on page 61).
- A flap separating true and false lumens is displayed as a linear lucency within enhancing vessels (fig 8 on page 20),
- Occlusion is seen as a progressive narrowing of the vessel until no residual opacification can be seen (fig 9 on page 21),
- A large number of pitfalls and differential diagnosis (discussed later) are found in CTA, so their knowledge is required in order to make an accurate diagnosis.

MRI an MR ANGIOGRAPHY (MRA)

Lumen imaging

- Usually, an eccentric signal void that represents the lumen stenosis is seen (figs 10 on page 23 and 11 on page 23),
- Flow void can be absent in case of occlusion,
- Slow flow can also cause loss of flow void,
- High-resolution MR imaging (HR-MRI) can also display luminal thrombus,
- Contrast-enhanced MRA and MRA ToF (time of flight) both are able to show lumen alterations like stenosis and occlusion (fig 12 on page 24).

Wall imaging

- Intramural hematoma is seen as crescent, curvilinear semilunar-shaped signal intensity (figs 10, 11 and 13) that widens external diameter of artery.
- The appearance of the intramural hematoma depends on the state of the blood products. In the early and chronic stage, the hematoma is usually
isointense/slightly hiperintense to surrounding structures, and in subacute stage is bright in T1 and T2W images,

- Intimal flap can be displayed as thin curvilinear hypointense partition separating true and false lumen,
- FLAIR and diffusion images performed in a routine cerebral studies can also show the intramural hematoma as a high signal round or crescent-shaped structure in the skull base (figs 14, 15, 16 and 54),
- HR-MRI is able to display the periarterial edema that appears as hyperintense T2 and PD, and hypointense T1 soft tissue surrounding the dissected artery. This periarterial edema is likely to be found in recent spontaneous dissection,
- As for CTA, a large number of pitfalls and differential diagnosis are found.

**ANGIOGRAPHY**

- Angiography can only display intraluminal contents as irregular progressive stenosis (fig 17 on page 29), occlusion (fig 18 on page 31) and double lumen,
- Intramural hematoma and the thickening of the arterial wall cannot be assessed.

**NORMAL CONDITIONS THAT MIGHT SIMULATE DISSECTIONS**

- ICA and VA hypoplasia
- Kinking
- Fenestration
- Vertebral venous plexus
- Flow artefacts
- Venous structures

**ICA and VA hypoplasia**

ICA is one of the most stable arteries and carotid dysgenesis are very rare. Differentiation between acquired vs congenital narrowing is important, because the first require proper treatment and the second may be compatible with normal life. In congenital hypoplasia the entire artery not only the lumen that is too small and there is no thickening of the
arterial wall. Hypoplasia is also associated with a well developed collateral circulation and a small carotid canal.

On the other hand, vertebral artery hypoplasia is more common. This variant may be suspected when the whole artery keeps the same small diameter from the ostium to V3. It may not contribute to the flow of basilar artery and terminate in ipsilateral postero-inferior cerebellar artery. It is also associated with a small vertebral canal (figs 19, 20 and 21).

Kinking

kinking (sharp angulation of one or more segments of a vessel) can mimic a flap and double lumen (figs 22 and 23).

Fenestration

Fenestration is defined as a duplication of a segment of a vessel. It is an extremely rare anatomic variation in ICA, but more common in VA. Fenestration can be mistaken with dissection because duplication assumes an appearance of two lumens and the wall between then can be confused with a flap. However in fenestration the limbs are symmetrical and the contour is regular. Oppositely in dissection the lumens are asymmetric and the contours show some irregularities.

Vertebral venous plexus (figs 24, 25, 26 and 30)

Vertebral venous plexus can be confused with dissection in CTA exams because it mimics a flap and double lumen. They become visible when the delay between the injection time and the acquisition is suboptimal. It’s one of the most common source of pitfalls. In MRI vertebral venous plexus can display high signal in exams performed without flow saturations bands.

Flow artifacts

Flow artifact usually happens in MR studies when it's performed without flow saturation bands and can be mistake with intramural hematoma because they are hyperintense in T1 and T2 (figs 27 on page 38 and 28 on page 39).

Venous structures

MRI studies performed without superior flow saturations bands can display high signal in venous structures that can be easily misdiagnose as dissection (fig 29, 30 and 31).

In CTA and contrast enhanced MRA techniques, There might be an inappropriate delay between injection time and acquisition. Therefore, with the images being acquired during
venous filling, some venous structures can be confused with dissections because of the irregularities of contours.

PHATOLOGICAL CONDITIONS THAT MAY SIMULATE DISSECTIONS

- Atherosclerosis
- Tumors
- Inflammatory pseudotumors
- Infections
- Erdheim-Chester Disease
- Arteritis
- Fibromuscular dysplasia
- Moya moya

Atherosclerosis

Atherosclerotic plaque can narrow the vessel lumen, the plaque itself can be confused with intraluminal hematoma, and it may sometimes increase the vessel diameter (positive remodeling). However dissections generally occur in younger patients, it spares carotid bulb and has no calcifications.

Atherosclerosis usually happens in older patients, carotid bulb is one of the most common site and at least a few calcifications are usually seen (figs 32 to 36).

Tumors

Some tumors, as cavernous meningiomas, have the characteristic to involve nearby vessels and cause irregular stenosis (fig 37 on page 49 and 38 on page 48).

Inflammatory pseudotumor

Inflammatory pseudotumor (IP) is a chronic granulomatous disease of unknown origin that presents as an enhancing, infiltrating meningeal mass that may involve the cavernous sinus and narrow ICA. A young adult with chronic headaches and cranial nerves palsies is the most common clinical presentation. As dissection, IP can narrow the vessel lumen, but on the other hand IP always display enhancement (figs 39 to 42) oppositely to dissection.

Infections
Infections display a high signal in T2, FLAIR and diffusion images. When located in sphenoid sinus nearby the carotid channel, it can be a challenge to diagnose or rule out an associated dissection, since infection can mimic intramural hematoma. Sinus infections show partial enhancement after contrast, is either iso or hypointense in T1W images. It does not narrow vessel lumen unless there is associated cavernous sinus involvement.

**Erdeim-Chester disease**

Erdeim-Chester disease is a rare form of non-Langerhans cell histiocytosis of unknown origin with multiple organ involvement. Vascular involvement of cervical and intracranial arteries is uncommon and is characterized by a perivascular infiltrate that can cause stenosis. In Erdheim-Chester disease the perivascular infiltration is usually circumferential instead of crescent shape of dissection, and there is signal enhancement after contrast (fig 43 on page 54).

**Arteritis**

Arteritis is an inflammation of an artery that can infiltrate the wall or the perivascular tissues. Inflammatory infiltration usually affects more than one vessel or a segment of a vessel. Imaging findings show irregularities, stenoses and occlusion with a perivascular infiltration that enhances after contrast (figs 44 on page 55, 45 on page 56 and 46 on page 57).

**Fibromuscular dysplasia**

Fibromuscular dysplasia is an idiopathic multifocal arteriopathy consisting of smooth muscle hyperplasia or thinning, proliferation of fibrous tissue, and elastic fiber destruction. More than 95% of the cases of cephalic involvement occur in ICA, and it is bilateral in 60 to 85%. Imaging finding demonstrates focal or long tubular, multifocal stenoses with adjacent dilatations, the so-called "String of beads".

Fibromuscular dysplasia is a risk factor for dissections. It may be discovered in the setting of an acute dissection.

**Moya Moya**

Moya Moya is an idiopathic progressive arteriopathy of childhood characterized by progressive narrowing and occlusion of the distal ICA and proximal circle of Willis.

**FALSE NEGATIVES : CONDITIONS THAT MAY LEAD TO MISS A DISSECTION**
MRI study performed before 3 days

Intramural hematoma will only be displayed in MRI in the subacute stage, after the 3rd day, because it’s when methemoglobin is formed (fig 47 on page 59, 48 on page 59 and 4 on page 16). Methemoglobin is hyperintense in T1, T2, FLAIR and diffusion images. The best sequence to show intramural hematoma is T1 with fat saturation.

In the 3 first days, T2 with fat saturation, NECT and CTA might be relevant if T1 fails to show the mural hematoma.

Venous structures and flow artifacts

Venous structures and flow artifacts can look like dissections, but the opposite is also true. The high signal intramural hematoma can mimic venous structures and flow artifacts in MRI studies performed without flow saturation bands (fig 29 on page 40).

Pseudofenestration

When dissection appears as short vascular segment with two lumens, it can mimic a fenestration, a condition known as pseudofenestration. The same finding that helps to differentiate between fenestration versus dissection can help between pseudofenestration versus fenestration. In pseudofenestration (dissection) the lumens are asymmetric and the contours show some irregularities (fig 49 on page 60) whereas in fenestration the limbs are symmetrical and the contour is regular.

Styloid process

CTA performed in critically ill patients can be of unsatisfactory quality mainly due to motion artifacts. In those patients a dissection with a high grade of stenosis or occlusion can be misdiagnosed, because hyperdense styloid process may be confused with a normal carotid (fig 50 on page 61). This error is unlikely to occur if proper window level settings are selected.

Flap and double lumen
Dissections presenting as thin flap and double lumen without intramural hematoma can be easily misinterpreted as normal vessel in T1 fat-sat images, since there is no high signal and no flow void narrow (figs 51 on page 62 and 52 on page 63).

IMPORTANT TECHNICAL ASPECTS TO IMPROVE THE DIAGNOSIS

ULTRASOUND

Good operator training greatly improves sensitivity.

The V1 and V3 arterial segments are frequently overlooked: they have to be seen during the examination. The internal carotids have to be followed as far as possible.

TCD (transcranial Doppler) is a critical "add-on", in order to assess hemodynamic consequences of a known dissection or to raise suspicion on a dissection of an inaccessible carotid segment.

CTA

The acquisition height must include proximal aorta and circle of Willis. Any involvement of these vessels has to be detected as it might dramatically change the treatment.

Metallic artifacts may greatly compromise examination quality and vessel analysis: remove earrings and make the patient bend the head upward to make dental artifacts move away from the more critical regions (ie. Sub-petrous internal carotids and distal cervical portions of vertebral arteries)

In faster scanners, such as 64 section multidetector CT, flow artifacts mimicking dissections, may occur.

Don’t rely only on MIP or curved reconstructions, which assess mostly the lumen size. The analysis of axial slices or sections of vessels are very important to evaluate the total size of the artery (ie. Lumen + vessel wall including hematoma). Some dissections display very limited or absent lumen narrowing and correct identification of the wall makes the diagnosis.

CT Perfusion may be added to the protocol, before or even a few minutes after CTA acquisition, in order to asses hemodynamic repercussions of the dissection.
MRI

Lumen imaging:

CEMRA (Contrast-enhanced MR Angiography) assesses only the lumen.

Be sure to use small voxel size (<0.9mm) in all directions. Some vendors display an interpolated out-plane resolution; in these cases, the real resolution you should expect is doubled (eg. If you want a real 0.8mm thickness, you should set your interpolated thickness at 0.4mm).

If the timing of acquisition wasn’t good and you get a lot of veins in your images, try to do thin-slab MIP reconstructions for the main regions of interest, instead of full-volume MIP.

3D-ToF (Time of Flight) imaging is a great alternative to CERMA, if injection is not possible.

It takes a longer time (around 5 minutes for an acquisition covering from carotid bulbs to skull base or 12 minutes from the proximal aorta to the circle of Willis). For that coverage, you will need to use multislab acquisitions in order to keep a good signal.

Lumen assessment is done with Thin-slab MIP reconstruction. Suppression of static tissues doesn't work well for hematomas. Therefore, looking at axial slices, one can usually see the wall hematoma in millimetric resolution. Its signal is usually darker than the circulating part of the vessel and brighter than adjacent fat (fig 53 on page 64).

Wall imaging:

Get used to look at the vessels in standard brain MRI (low slices of FLAIR and diffusion sequences) as wall hematoma may already be visible in many patients (fig 54 on page 65).

The acquisition height must be wide and include intrapetrous carotid segments.

For the Fat Suppression T1 sequence, use superior and inferior rest slabs to suppress arterial and venous signal. Avoid LR phase encoding as it raises artifacts.

In the first days, an additional Fat Suppression T2 may be useful.

For short dissections, millimetric axial images of 3D-ToF are sometimes more sensitive than thick Fat Suppression T1 slices.

It's always a good idea to add MR Diffusion and perfusion imaging even if there is none or few neurologic deficit. If perfusion study is done in the same examination than a CEMRA...
(Contrast-enhanced MR Angiography), a 2nd injection is usually not a problem. In case of severe renal failure, ASL techniques may be used, although its accuracy is not well studied in this indication.

COMMON AND SEVERE COMPLICATION

- Ischemic stroke

Ischemic stroke

Ischemic stroke is the most common and severe complication of a CAD, and can be present in up to 80% of the patients.

According to the current conception of the relationship between the mechanism of infarction and stroke patterns, cortical or subcortical infarcts are more likely to be of embolic origin (figs 55 on page 65, and 56 on page 66), whereas junctional or watershed infarcts (fig 57 on page 67) are more likely to be of hemodynamic origin.

Thromboembolism, not hemodynamic infarction, is the essential stroke mechanism in CAD.

Images for this section:
Fig. 1: CTA of skull base shows irregular stenosis of right ICA (white arrow). Compare with the normal left ICA (black arrow). Skull base dissections are not reached by US.
Fig. 2: US of the right ICA showing: narrowing of the luminal diameter (black arrows), intramural hematoma (white arrows) and thickening of the arterial wall (white bar).
Fig. 3: Vertebral artery (intertransverse segment) dissection. Hypoechoic intramural hematoma (arrows) is clearly seen between the lumen and vessel wall.
**Fig. 4:** NECT performed 2 days after the onset of symptoms show the hyperdense intramural hematoma (white arrows).
**Fig. 5:** CTA of the right ICA showing lumen narrowing (white arrow), and intramural hematoma (black arrow). The whole vessel size (lumen + wall) is raised (bar).
**Fig. 6:** CTA of double vessel dissection (right VA and left ICA). Note intramural hematoma (white arrows) and narrowing of the arterial lumen (black arrows)
Fig. 7: CTA display a thin rim of contrast-enhancement surrounding intramural hematoma (white arrows). Notice the narrowing of the lumen (orange arrow).
Fig. 8: CTA of the neck display a flap (white arrow) in the right VA.
Fig. 9: Curved MPR reconstruction of the left carotid artery shows occlusion of the ICA. Opacification of the top of ICA from the circle of Willis and ophtalmic artery.

Fig. 10: Axial T1W image with fat suppression. Reduction of lumen diameter (white arrow) and hyperintense intramural hematoma (black arrows).
Fig. 11: VA dissection. Axial fat-sat T1W image display the reduction of lumen diameter (black arrow) and hyperintense crescent-shaped intramural hematoma (white arrows).
Fig. 12: CE-MRA (Contrast-Enhanced MR Angiography) ; MIP reconstruction. Occlusion of the left ICA is clearly seen 1 cm above carotid bulb (arrow).
**Fig. 13**: Axial T1 fat-suppression at the level of the carotid canal. Hyperintense intramural hematoma (white arrows) at the carotid channel that narrows the lumen. Compare with the normal opposite side (black arrow).
**Fig. 14:** Double carotid dissection. This is the first FLAIR image in a cerebral MRI study performed in a patient with a suspicion of stroke. FLAIR image clearly depicts the hyperintense intramural hematoma and the eccentric flow void that represent lumen stenosis (white arrows).
Fig. 15: Same case as fig 14. Double carotid dissection. This is the first diffusion image in a cerebral MRI study performed in a patient with a suspicion of stroke. Diffusion image display two high signal intensity (arrows) that represents intramural hematoma.
**Fig. 16:** Same case as fig 14; Double carotid dissections. CT angiography images confirming lumen narrowing.
Fig. 17: Irregular stenosis of ICA. Note that DSA is not able to display intramural hematoma.

Fig. 18: DSA show complete occlusion of ICA (white arrow).
**Fig. 19:** Right VA hypoplasia (white arrow). There is no intramural hematoma and no increase in the vessel diameter. Even though, a differentiation between dissection and hypoplasia may be difficult. A narrow vertebral foramen can help one to make accurate diagnosis - see figures 20 and 21.
Fig. 20: Same case of fig 16. Asymmetric size with a small right vertebral foramen (white arrow) confirms VA hypoplasia.
**Fig. 21:** Right VA dissection. Both foramens have almost the same diameter, lumen stenosis (black arrow) and intramural hematome (white arrow) is present in the right one. Compare with figures 19 and 20.
Fig. 22: CTA of the vertebral vessels show kinking of the left VA. This finding may mimic a flap and double lumen (black arrow). The analysis of adjacent slices helps ruling out dissection (see fig.23).
Fig. 23: Same patient as Fig.22. Kinking of the left VA (black arrow)- one level above previous image.
Fig. 24: CTA show the vertebral venous plexus (white arrows) that can be confused with double lumen and flap.

Fig. 25: Same case as figure 24, one level above, Vertebral venous plexus (white arrows) and vertebral artery.
Fig. 26: CTA display another case of a crescent-shaped vertebral venous plexus that can be misdiagnosed as a dissection. Compare with figure 8. To avoid this pitfall, it is recommended to follow all extentions of the vessel.
Fig. 27: Axial T1 with fat saturation performed without inferior saturation band. Note the high signal intensity in both ICAs (large white arrows) and VAs (small white arrows).
**Fig. 28:** Axial T1 with fat suppression performed without superior saturation band, shows two round high signal venous structures (arrows).
Fig. 29: Axial T1 with fat suppression. Dissection and normal jugular vein. A right ICA dissection (straight white arrow) can be easily misdiagnosed as a venous structures. And the opposite is true as the left jugular (curved white arrow) may mimic a intramural hematoma. Note the left ICA flow void (black arrow).
Fig. 30: Axial T1 with fat suppression performed without superior saturation band shows high signal intensity vertebral venous plexus (white arrow) that can easily be confused with intramural hematoma (compare with figure 11). Also note the high signal intensity in jugular veins adjacent to a round flow void that represents ICA.
Fig. 31: CTA display a left jugular vein (white arrow) adjacent a ICA (white arrow) that simulate a double lumen and flap.
Fig. 32: Axial T1 with fat suppression and after injection of contrast material. Hyperintense atherosclerotic plaque (arrow) that narrows the lumen diameter.Courtesy of Dr Damien Galanaud.
Fig. 33: Same case. T2 with fat suppression shows a hyperintense atherosclerotic plaque (arrow) that narrows the lumen. Courtesy of Dr Damien Galanaud,
**Fig. 34:** Same case. Atherosclerotic plaque with a soft tissue component (arrows). Note that the place is located in carotid bulb and have calcifications. Dissections usually begins above carotid bulb, and do not have any calcifications. Courtesy of Dr Damien Galanaud.
**Fig. 35:** Atherosclerotic plaque. CTA of the right ICA. The soft tissue components of the place (white arrows) can mimic intramural hematoma. Peripheral calcifications (black arrows) can help to make the correct diagnosis.
Fig. 36: CTA : axial slice at the level of the right proximal ICA. Atherosclerotic plaque. CT clearly shows that the soft tissue component (white arrows) has a vast lipid core (-25 UH). Note also some peripheral calcifications (black arrows).
Fig. 37: Axial T1 after fat suppression and gadolinium injection at the level of distal carotid canal. The left sphenoid meningioma extends to Meckel's Cave, cavernous sinus and carotid canal (black arrow) and leads to a lumen stenosis (white arrow).
Fig. 38: Same case, above fig 37. Compression and narrowing of carotid artery in the cavernous sinus from the meningioma.
Fig. 39: Inflammatory pseudotumor. T1 fat-sat after gadolinium shows an enhancing infiltrating meningeal infiltration (black arrow) that involves the cavernous sinus and narrows carotid artery (white arrow).
**Fig. 40:** Inflammatory pseudotumor, same as fig 39, 7 months after steroid treatment. There is a reduction of meningeal infiltration (white arrow) with an increase in lumen diameter (black arrow).
**Fig. 41:** Inflammatory pseudotumor, same case as fig 39. MRA ToF, clear depiction of lumen stenosis of the left ICA (arrow) compared to the opposite side.
Fig. 42: Inflammatory pseudotumor, same case as fig 39, 7 months after treatment. MRA ToF, there is a clearly increase in lumen diameter (arrow) of the ICA.
**Fig. 43:** T1 after gadolinium. Image shows an enhanced perivascular infiltration of left VA (arrow). In this patient, associated intraconal orbital masses, pituitary and bone abnormalities suggested Erdheim-Chester Disease.
Fig. 44: Takayasu arteritis. 23 years old girl, presenting with left cervical pain. T1 after gadolinium shows a enhanced perivascular inflammatory tissue (arrow) that lead to a slight decrease in lumen diameter. She also had a stenosis of the left subclavian artery (next figure).
Fig. 45: Takayasu arteritis. Same patient as fig 44. CEMRA (Contrast enhanced MRA) with MIP reconstruction isolating posterior circulation. Mild stenosis of the right subclavian artery (arrows).
Fig. 46: Sagittal oblique T2 with fat saturation through left ICA. Occlusion of common and internal carotid artery; the thrombus is clearly depicted as an heterogeneous T2 hypersignal. Perivascular T2 hyperintensity suggests arteritis (white arrows).

Fig. 47: Fat suppression T1W performed in the same day of the beginning of symptoms. The intramural hematoma (white arrows) is isointense to adjacent structures. Same case as fig 4 (unenhanced CT).
**Fig. 48:** Fat suppression T1W performed 7 days after the beginning of symptoms, same case as figures 4 and 47. The intramural hematoma is now clearly depicted as a high T1 signal intensity (white arrow).
**Fig. 49:** Pseudofenestration. CTA show a double lumen (arrow) and flap in this dissected VA. Note that the limbs are asymmetrical and the boundaries are irregular.
Fig. 50: CTA performed in a critically ill patient. This patient has a long left styloid process (black arrow) that was misinterpreted as a normal carotid. In fact, there is a dissection in the left ICA with an important narrowing of the lumen (white arrow) and an obvious thickening of the wall.
Fig. 51: A patient with dissection in both carotids. This T1 fat sat image was not able to show dissection, since there was a flap and double lumen and no intramural hematoma. White arrow shows the normal flow void in the left carotid artery.
Fig. 52: Same case as fig 51. CTA curved reconstruction clearly shows a CCA, ICA and ECA dissection presenting as flap and double lumen (arrow). The patient also had a dissection in the opposite carotid artery (not shown). Common carotid dissections as well as multiple dissections should always raise suspicion on aortic dissection.
**Fig. 53:** Axial slice of a Tof MRA (left side) and axial T1 fat-sat after contrast (right side) in a 56 yo patient with spontaneous right ICA dissection. In Tof MRA axial slices, the wall hematoma (orange arrow) displays a signal that is usually darker than the circulating part of the vessel (white arrow) and brighter than adjacent fat. T1 fat sat also displays the wall hematoma and the lumen narrow. This T1 was done after contrast because it was a suspicion of right oculomotor nerve palsy. Notice the enhancing perivascular inflammation (small yellow arrows) around dissected vessel.

**Fig. 54:** FLAIR (left side) and diffusion (right side), performed in a cerebral routine MRI, showed the intramural hematoma (white arrows) and lumen stenosis (orange arrows).
**Fig. 55:** Diffusion image show 2 foci of embolic ischemia.
Fig. 56: Diffusion-weighted image display a recent, massive ischemic stroke due to a ICAD. Involvement of both MCA and PCA territories was due to a fetal origin of right PCA.
Fig. 57: Diffusion-weighted image shows a right junctional infarction in a patient with right ICA dissection.
Conclusion

CAD is one of the leading causes of ischemic stroke in young and middle-age patients. It may lead to severe disability without early diagnosis and treatment.

Conversely, overdiagnosis of dissection may result to a needless and potentially hazardous treatment.

US, CTA and MRA interpretation is sometimes difficult and represents a challenge to the radiologist, mainly during the first 3 days and in vertebral arteries dissections.

Therefore, imaging features and pitfalls must be known and one must not hesitate to use two or more non-invasive imaging tests in association, in order to make or to rule out the diagnosis.

Personal Information

Ocacir de Souza Reis Soares, Radiologist
Imed diagnosticos por imagem. Presidente Prudente/Brazil
ocacirsoares@gmail.com

Marlus de Souza Reis Soares, Radiologist
Imed diagnosticos por imagem. Presidente Prudente/Brazil
marlus.srs@gmail.com

Julien Savatovsky, Neuroradiologist
Praticien Titulaire, Service d'imagerie médicale
Conflicts of interest: none

The authors would like to acknowledge for their help:

Damien Galanaud, MD, PhD, Hôpital Pitié-Salpêtrière, Paris, France
Jean-Claude Sadik, MD, Fondation Rothschild, Paris, France
Marc T Williams, MD, Fondation Rothschild, Paris, France
Frédérique Charbonneau, MD, Fondation Rothschild, Paris, France
Isabelle Klein, MD, PhD, Hôpital Bichat, Paris, France

We would like to thank for their work technicians and doctors of radiology departments where the images come from, especially:

Hôpital Pitié-Salpêtrière - Neuroradiology unit (Pr Jacques Chiras), where two of the authors worked as fellow (JS, 2004-2006) and resident (OSRS, 2004-2005)

Fondation Rothschild - Medical Imaging unit (Dr Françoise Héran)

References

3. Christian Lucas, MD; Thierry Moulin, MD; Dominique Deplanque, MD; Laurent Tatu, MD; Didier Chavot, MD the DONALD Investigator. Stroke Patterns of Internal Carotid Artery Dissection in 40 Patient. *Stroke* 1998;29:2646-2648


