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## Imaging of cervical artery dissection



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### KEYWORDS

Arterial dissection;  
Mural haematoma;  
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**Abstract** Cervical artery dissection (CAD) may affect the internal carotid and/or the vertebral arteries. CAD is the leading cause of ischemic stroke in patients younger than 45 years. Specific treatment (aspirin or anticoagulants) can be implemented once the diagnosis of CAD has been confirmed. This diagnosis is based on detection of a mural haematoma on ultrasound or on MRI. The diagnosis can be suspected on contrast-enhanced MRA (magnetic resonance angiography) or CT angiography, in case of long stenosis, sparing the internal carotid bulb, or suspended, at the junction of V2 and V3 segments of the vertebral artery, in patients with no signs of atheroma of the cervical arteries. MRI is recommended as the first line imaging screening tool, including a fat suppressed T1 weighted sequence, acquired in the axial or oblique plane at 1.5T, or 3D at 3T. Complete resolution of the lumen abnormality occurred in 80% of cases, and CAD recurrence is rare, encountered in less than 5% of cases. Interventional neuroradiology (angioplasty and/or stenting of the dissected vessel) may be envisaged in rare cases of haemodynamic effects with recurring clinical infarctions in the short-term.

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*Abbreviations:* TIA, transient ischemic attack; MRA, magnetic resonance angiography; CVA, cerebrovascular accident; HS, Horner's syndrome; CAD, cervical artery dissection; SAT, supra-aortic trunks.

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## Overview

Cervical artery dissection (CAD) is an increasingly commonly identified cause of cerebrovascular accidents. CAD is involved in nearly 2% of all cerebrovascular accidents in the general population and nearly 20% of CVA in patients over 45 years of age [1,2]. CAD are defined as the presence of a mural haematoma in the wall of an artery leading into the intracranial space. They can affect the internal carotid arteries and/or the vertebral arteries in their various extracranial segments. The internal carotid artery is the most common site of CAD, the haematoma can form in any of its different segments: supra-bulbar, cervical, sub- and intrapetrous, but always sparing the bulb. The vertebral artery, less commonly affected, may be affected in its various segments: ostial (V0), post-ostial (V1), transverse (V2), or atloidian (V3).

The embolic complications of CAD result in ischemic accidents, making rapid treatment and a good knowledge of imaging techniques and their respective performances essential.

## Epidemiology

A constant increase in the diagnosis of CAD has been reported, essentially due to the improved availability of MRI and the continuing improvement in sequences enabling a positive diagnosis [3]. The incidence of CAD is 2.6 to 2.9 for every 100,000 inhabitants per year for spontaneous dissections of the internal carotid artery [1]; it is lower, estimated at around 1 for every 100,000 inhabitants for dissections of the vertebral arteries [4]. These figures are probably underestimated. The peak in frequency is at around 46 years, with no gender predisposition [4–6].

## Etiologies

CAD are usually spontaneous, in a healthy artery, with no identifiable etiological factor [3,7]. A certain number of events have been associated with the onset of CAD (trauma, disease of the collagen, infections, etc.).

### Traumatic dissections

These generally occur after cervical trauma (direct shock, road traffic accident [RTA], strangulation, etc.), responsible for direct injury to the arterial wall. These traumatic events may precede the onset of the first symptoms by several hours to days [8]. A CAD is present in 1–2% of patients that have undergone major trauma and the risk seems to be greater in cases of fracture to the face, cervical spine (with damage to the transverse canal), the base of the skull, or traumatic cerebral lesions [9]. There is a greater risk of carotid dissection after thoracic trauma, and of vertebral dissection after spinal fractures and damage to the spinal cord [10,11].

### Spontaneous dissections

CAD are described as spontaneous in the absence of any clearly identified trauma. In 30 to 40% of patients with a spontaneous CAD, the history may reveal a notion of minor cervical “trauma” that went unnoticed, as these are common in everyday life (hyperextension, cervical

manipulations, etc.) [12,13]. A certain number of factors are associated with the onset of spontaneous CAD.

### *Constitutional anomalies of the wall*

Histological anomalies affecting notably the connective tissue have been identified in 50% of patients with CAD. Certain genetic diseases such as Marfan disease or type IV Ehlers Danlos syndrome, involving collagen III, may include an episode of CAD over the course of the disease [14–19].

### *Infection*

An infection, notably ENT, is commonly reported in cases of CAD. It is accompanied by an inflammatory syndrome on blood tests, also found in patients with CAD [7,20]. A local infection may promote an inflammatory cascade inducing lesions of the vascular wall, resulting in CAD [7,21–25].

### *Transient vasculitis*

The often multiple character of CAD (around 15%) [26] and the low recurrence rate (around 0.8% per year) [6,27–29] are evocative of a transient inflammatory pathology of the arterial network. A histological study, on nine patients with CAD, demonstrated the presence of inflammatory extensions in the wall of the superficial temporal arterial, suggestive of diffuse arterial damage [30].

## Pathophysiology of cervical dissection

### *Mural haematoma*

Mural haematomas are responsible for cleavage of the arterial wall, along a variable distance [31]. The exact process behind the formation of mural haematomas is disputed [32]: the haematoma could be the result of rupture of the vasa vasorum of the media, without communication with the arterial lumen, or could be secondary to a breach in the intima, allowing blood to enter the arterial wall from the lumen. An animal model inducing intimal breaches reproduces a histological aspect of CAD that is comparable to that observed in man [33,34]; the intimal breach is rarely found, notably under angiography, and could simply be secondary to the rupture of the mural haematoma into the arterial lumen.

### *Consequences of mural haematoma*

Irrespective of the mechanism of onset of the haematoma, sub-intimal dissections should be distinguished from sub-adventitious dissections. In sub-intimal dissections, the haematoma compresses the arterial lumen, leading to variable degrees of stenosis, or even occlusion [35]. In sub-adventitious dissections, one observes a fusiform deformation of the artery with an increase in the external diameter of the artery and preservation of the arterial lumen. This dilation may result in a conflict with the adjacent structures (cranial nerves in the cervical path or cervical sympathetic fibres) and the onset of clinical symptoms such as cervical pain or Horner’s syndrome.

### *Topography of mural haematomas*

CAD are usually found in the mobile arterial segments, not fixed by the bony structures. The sub-petrous segment of the internal carotid artery is affected in the majority of cases (Fig. 1), with a haematoma that spares the bulb, but

may extend in height to the sub- and intrapetrous segments (Figs. 2 and 3), but rarely as far as the intracranial segments. Segment V3 is the most common site of CAD in the vertebral arteries (Figs. 3 and 4) [36–38].

## Clinical aspects

CAD are responsible for local signs via compression of the neighbouring nervous structures or neurological deficits via migration of emboli or significant haemodynamic stenosis [38,39].

### Local signs

The local signs of CAD include cervical pain, Horner's syndrome, and cranial nerve damage. They are probably the consequence of local compression by the mural haematoma [40–42]. A carotid CAD is suspected whenever a cranial nerve pair is affected (IX, X, XII) or there is compression of the sympathetic fibres leading to Horner's syndrome, which is present in nearly 50% of cases [42,43]. Vertebral dissection should be evoked whenever there is damage to a cervical nerve root [44]. Frontal or periorbital headaches orientate the diagnosis to a carotid dissection, occipital headaches to a vertebral dissection.

## Consequences and long-term signs

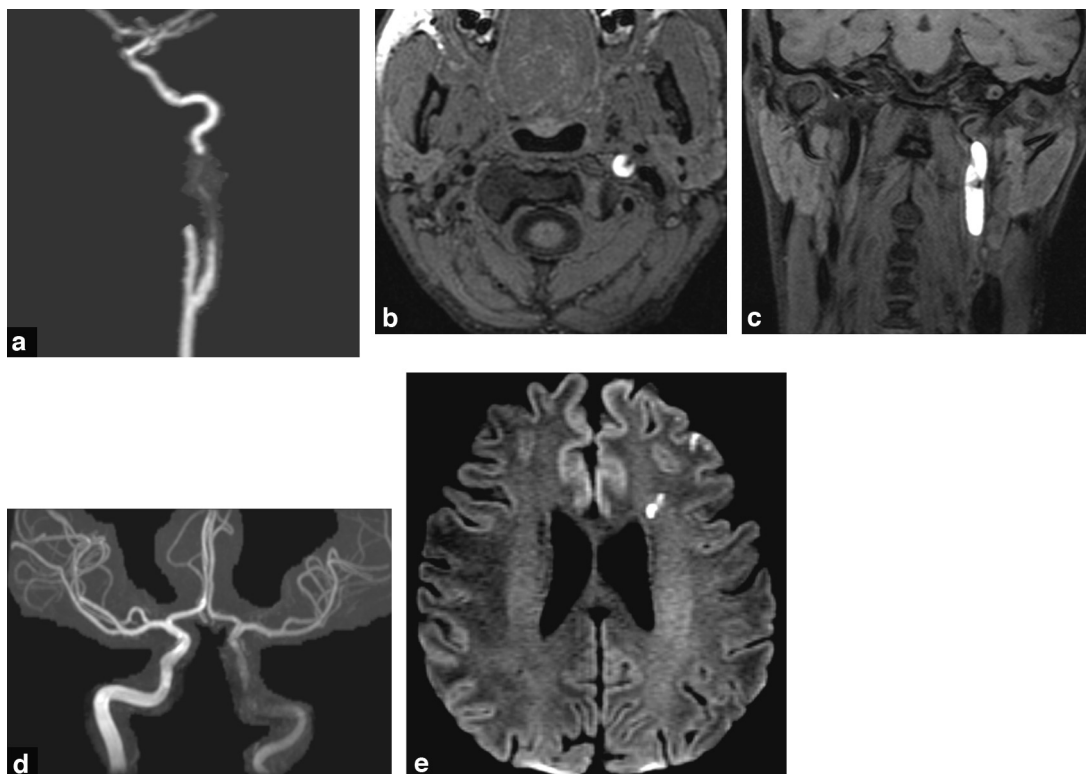
Neurological deficits are inconsistent and come after local signs. They occur within the first two weeks of the formation of the CAD in 80% of cases [40]. The sylvian territory is the most commonly affected [6,45,46].

The data in the literature concerning the mechanism of long-term signs are contradictory, evoking a purely haemodynamic origin by stenosis [35] or an embolic origin after formation of a thrombus developed on an intimal breach [47–49]. In MRI, the anomalies reported are more often evocative of an embolic mechanism (visualisation of the thrombus in T2\* [50], cortical territory of the ischaemia in diffusion) (Fig. 5) than a haemodynamic origin (asymmetry of the intracranial artery signals, circulatory slowing in FLAIR) (Fig. 1) [51–54].

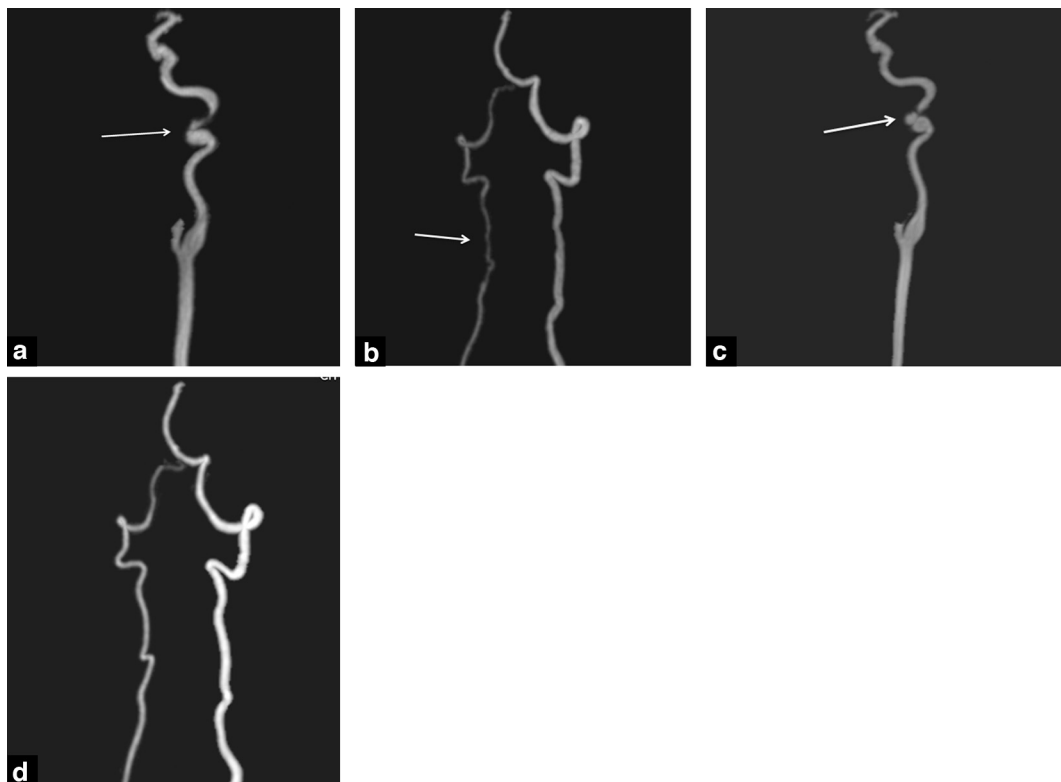
## Progression

### Prognosis

The prognosis of a CAD depends on the severity of the CVA [55], with an overall mortality of between 2 and 5%. The prognosis is generally good, with no sequelae in 80 to 90% of cases [4,6,56]. The factors associated with an unfavourable prognosis are the presence of cerebral ischaemia, an arterial



**Figure 1.** A 52-year-old patient with a stenotic CAD of the left internal carotid artery. Dynamic MRA after injection of gadolinium in maximum intensity projection reconstruction after decoupage, including only the left internal carotid artery seen from the side (a), MRI with T1 volume sequence with suppression of the fat and circulating blood in axial slice reconstructions (b) and coronal slice reconstructions, MRA in time of flight of the vessels of the circle of Willis in maximum intensity projection reconstruction after decoupage including only the internal carotids and the anterior and middle cerebral arterial branches seen from the front (d), axial slice in diffusion weighting  $b = 1000$  (e). CAD of the left sub-petrous internal carotid artery with a mural haematoma, which is hyperintense in T1 in an "eccentric crescent" (b and c), a long and progressive stenosis of the supra-bulbar arterial lumen (a), a reduced flow in the left internal carotid, appearing "grey" in time of flight MRA (d), and a recent ischemic accident of the left anterior junctional territory which is hyperintense in diffusion  $b = 1000$ , by a haemodynamic mechanism (e).



**Figure 2.** Dynamic MRA of the same patient as in Fig. 6. Dynamic MRA after injection of gadolinium in maximum intensity projection reconstruction after decoupage including only the left internal carotid artery seen from the side (a) and the two vertebral arteries seen from the front (b), with check-up at 4 months (c and d). Irregularity of the calibre with alternation between dilation and stenosis of the sub-petrous segment of the left internal carotid artery (a), and of the right vertebral artery (b, arrows). MRA check-up at 4 months shows the appearance of a pseudo-aneurysm of the sub-petrous segment of the left internal carotid artery (c, arrow) and the disappearance of the disparity in calibre of the right vertebral artery.

occlusion, a carotid location, elderly patient, and a severe deficit on admission [39,55–58].

### Relapse

Relapse of CAD may be expressed by the reappearance of clinical symptoms or the onset of a new mural haematoma during imaging check-ups. These two forms of relapse are rare: 1 to 12% [6,59–61] for clinical relapse and 0 to 14% for radiological recurrence. In the vast majority of cases, relapses occur within the first two months of the initial CAD [6,46]. The risk factors for CAD recurrence are younger patients [27], a family history of CAD [29], a disease of the connective tissue (Ehlers Danlos syndrome or fibromuscular dysplasia) [39,45,62], the presence of multiple CAD, arterial hypertension [6,60]. An arterial occlusion is not a significant prognostic factor [63].

### Natural progression

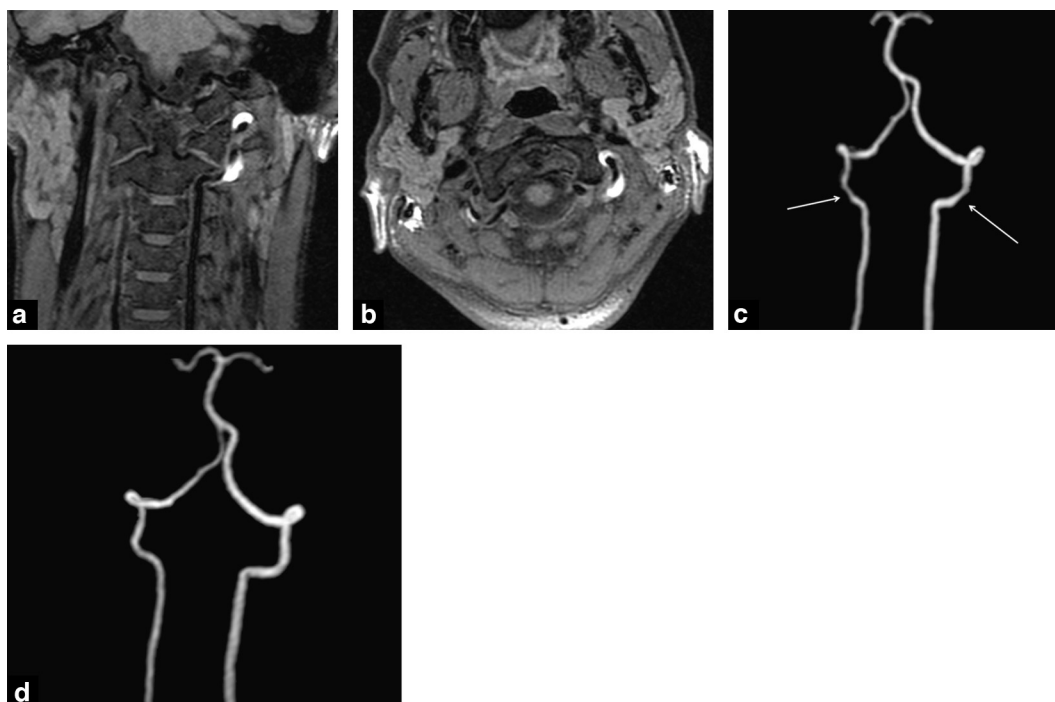
Regression of the dissection and disappearance of the mural haematoma is observed in over 80% of cases at radiological follow-up (ultrasound, slice imaging, or angiography) (Figs. 2, 3 and 5) [56,64–70]. This regression may leave sequelae on the vascular wall and arterial lumen, with varying degrees of residual stenosis. The regression usually occurs very rapidly with an improvement in the condition

of the arterial lumen seen on ultrasound within two weeks of the initial symptoms [71,72]. In the event of a dissecting aneurysm, the arterial lumen rarely returns to normal (Fig. 3) [59,70]. On follow-up imaging examinations, the arterial lumen does not seem to alter after one year [4]. There does not seem to be any difference in progression between carotid and vertebral CAD.

## Radiological diagnosis of CAD

### Conventional angiography

Conventional angiography (Fig. 4) is the reference imaging technique for studying the arterial lumen, but does not provide any information about the vascular wall. This technique does not enable the detection of the mural haematoma, and angiography may be considered normal in the event of CAD, especially if the latter does not cause any alteration in the arterial lumen. In the event of carotid occlusion, the evocative signs of CAD are a “candle flame” appearance sparing the carotid bifurcation, a long and irregular stenosis that spares the bulb, a dissecting aneurysm, an intimal flap, or a false channel. This invasive and irradiating technique is not performed in first intention for the positive diagnosis of CAD.



**Figure 3.** Patient with a bilateral stenosing vertebral CAD. MRI with T1 weighting with suppression of the fat and circulating blood in coronal (a) and axial reconstruction slices, dynamic MRA after injection of gadolinium in maximum intensity projection reconstruction after decoupage including only the two vertebral arteries seen from the front (c) with check-up at 3 months (d). CAD with mural haematoma which is hyperintense in T1 and extends to portion V3 of the left vertebral artery (a and b), but also partial involvement of portion V3 of the right vertebral artery. Slightly stenotic irregular appearance to the lumens of the two V3 portions of the vertebral arteries (c, arrows), which are no longer visible on the check-up at 3 months.

## Ultrasound

Ultrasound is usually performed in first intention, enabling confirmation of the diagnosis of CAD with good results [73]. The ultrasound signs of dissection are a direct visualisation of the mural haematoma, an increase in the external calibre of the artery or an intimal flap. Non-specific signs of dissection are frequently observed (stenosis or occlusion with haemodynamic repercussions) on Doppler examination. The principal limitation of ultrasound resides in the inability to examine the entire vascular axis due to some regions being inaccessible to the ultrasound beam (sub- and intrapetrous internal carotid, vertebral artery V2 within the transverse bony foramens and V3 atloidian), which are responsible for numerous false negatives [73,74].

## Slice imaging MRI and CT

MRI offers direct visualisation of the mural haematoma, with a sensitivity of detection and specificity that are superior to those of CT. It is therefore the examination of choice if CAD is suspected [3]. MRI also enables the detection of any cerebral ischemic consequences of the CAD. CT should be proposed in second intention or in the event of cervical trauma [75].

### Imaging of the mural haematoma

#### *Axial T1 slices with saturation of the fat signal*

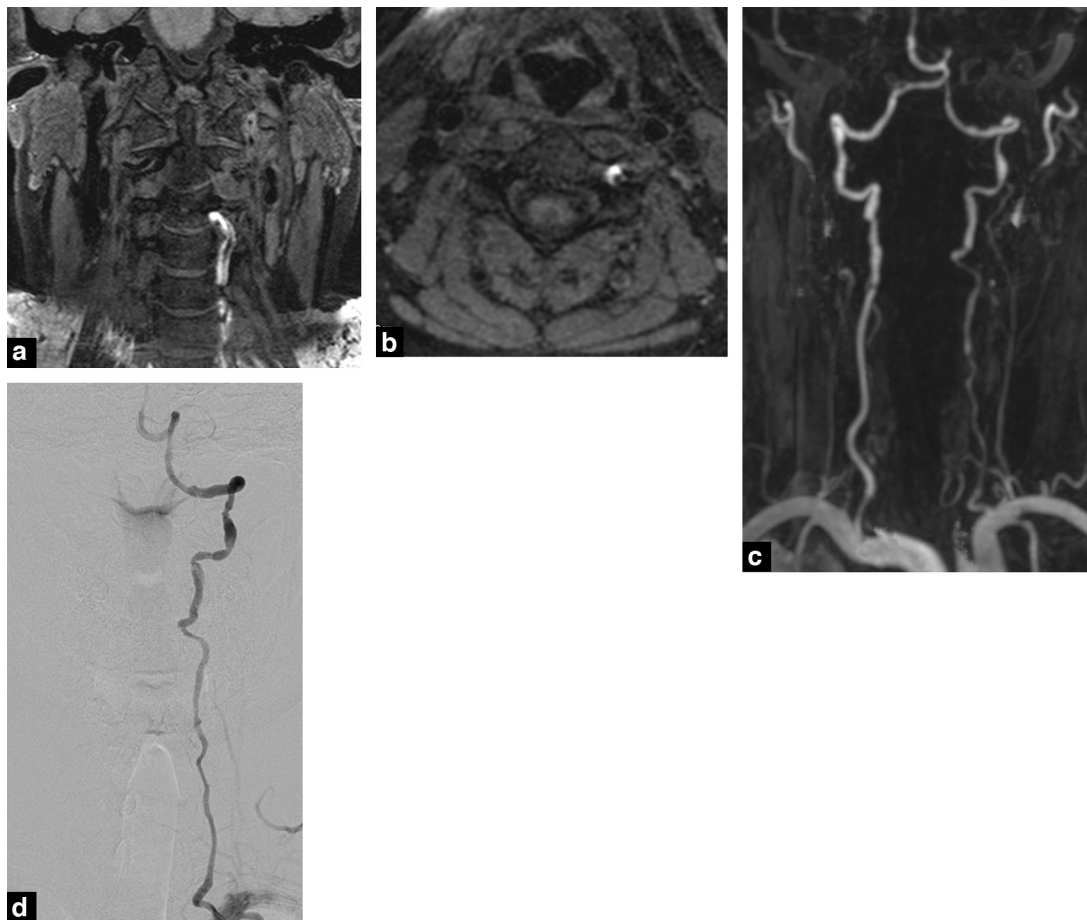
The T1 weighted sequence with fat saturation enables visualisation of the mural haematoma. The latter appears hyperintense with a “crescent” shape that is eccentric in

comparison with the residual arterial lumen (Figs. 1, 5 and 6) [76]. As with the signal of an intracerebral haematoma, T1 signal anomalies of the mural haematoma vary as a function of the time of imaging [77]. The oxyhaemoglobin present in the hyperacute stage (a few hours) and the deoxyhaemoglobin present in the acute stage (before 72 hours) make the haematoma isointense in T1 [78,79]. After 72 hours, the haematoma appears hyperintense in T1 due to the appearance of intra- then extracellular methaemoglobin [80]. The sensitivity of MRI is therefore lower when the MRI is performed early, within the first three days of the formation of the haematoma [76]. The sensitivity of MRI may also be reduced in cases of vertebral dissection (small haematoma or one that is localised in a tortuous segment such as V3) [67]. The proximity of the venous plexus around the vertebral artery may affect the specificity of MRI, since the blood circulating within the venous plexus gives false images of dissection with a hyperintense crescent-shape in T1 [81,82].

#### *High resolution MRI at 1.5 and 3T*

High resolution MRI sequences enable analysis of the arterial wall with improved sensitivity for the detection of a haematoma of the wall [83–85]. At 1.5T, these sequences require the use of flexible surface antenna adapted to the cervical anatomy. At 3T, the reference sequence is a volume acquisition in 3D with suppression of the fat and circulating arterial blood (“black blood” effect) (Figs. 1, 2 and 6) [74,86,87]. High resolution MRI is particularly useful in cases of suspected vertebral CAD, whose diagnosis is hindered by the proximity of bony structures, the presence of peri-arterial venous plexi, the tortuosity of segment V3, the





**Figure 4.** A 36-year-old patient with a CAD of the left vertebral artery. MRI with T1 weighting with suppression of the fat and circulating blood in coronal (a) and axial reconstruction slices, dynamic MRA after injection of gadolinium in maximum intensity projection reconstruction after decoupage including only the vertebral arteries seen from the front (c), digital angiography after catheterisation and selective opacification of the left vertebral artery, seen from the front (d). CAD with mural haematoma, which is hyperintense in T1, of portion V2 of the left vertebral artery (a and b), irregular calibre of the arterial lumen of portions V2 and V3 of the left vertebral artery in dynamic MRI (c) and digital angiography (d).

variability of the diameter of the vertebral artery, and of the small dimensions of the haematoma. High resolution MRI is therefore indicated in patients with suspected vertebral dissection and with a non-conclusive imaging profile, with a protocol oriented towards studying anomalies of the arterial wall detected on Doppler ultrasound, T1 sequence with fat saturation, or MRA of the supra-aortic trunks [83–85].

### CT

Evaluation via CT scan does not reveal any specific signs of CAD or confirm the mural haematoma, which does not appear spontaneously dense. CT can reveal a non-specific, asymmetrical, eccentric, crescent-shaped parietal thickening, reducing the diameter of the circulating lumen.

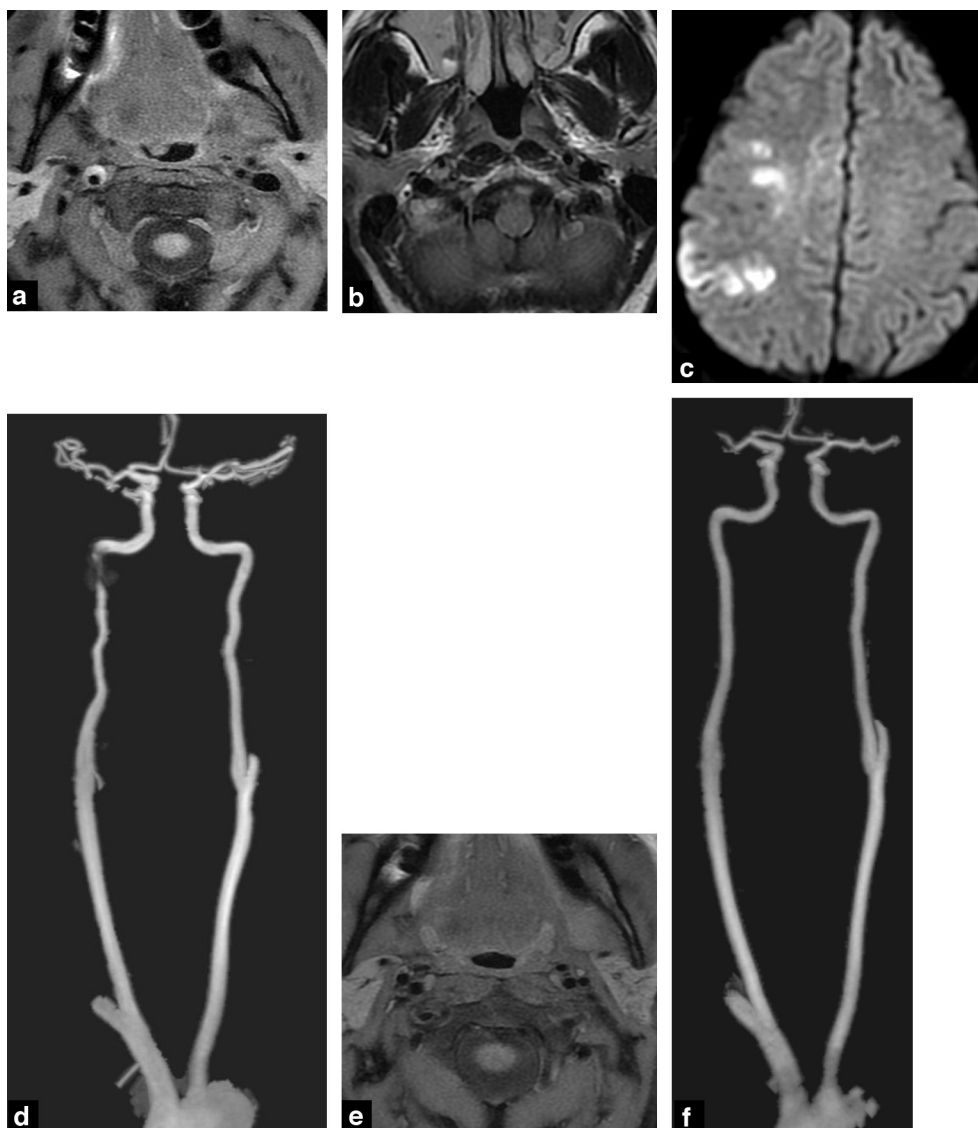
### Cerebral MRI

Although the principal objective of cerebral MRI sequences to explore a neurological deficit (diffusion, FLAIR, T2\*, MRA TOF of the triangle of Willis) is not the examination of the arterial wall, its field of exploration covers the sub-petrous segment of the internal carotid artery and segment V3 of the vertebral arteries. In nearly 75% of carotid CAD, the mural haematoma extends up to this level and to segment V3 in

50% of cases [88–90]. The various sequences of cerebral MRI are evocative of CAD when the mural haematoma extends into the field of view (Fig. 5) [91].

### Examination of the arterial lumen

The consequences of dissection on the arterial lumen are studied in MRA of the supra-aortic trunks or in CT angiography, with a similar semiology. These techniques are used to quantify the degree of stenosis, determine if there is an occlusion, and detect the development of a possible dissecting aneurysm. As with angiography, false negatives are possible with both these techniques in cases of arterial dissection with a normal lumen. In cases of carotid CAD, the most evocative appearance is that of a long and progressive stenosis, sparing the carotid bulb (Fig. 1) and in cases of vertebral CAD, that of a suspended stenosis of the V2–V3 junction (Fig. 6). Although the initial diagnosis is challenging, a retrospective diagnosis can be made at follow-up examinations, by demonstrating complete or partial restoration of the arterial lumen, or in the event of the onset of a dissecting aneurysm (Fig. 3).



**Figure 5.** A 48-year-old patient with a CAD of the right internal carotid artery. MRI with T1 sequences after fat saturation in axial slices (a), FLAIR in the axial plane (b), diffusion  $b = 1000$  in the axial plane (c), dynamic MRA after injection of gadolinium in maximum intensity projection reconstruction after decoupage including only the internal carotid arteries seen from the front (d). MRI check-up at 9 months, with T1 sequences after fat saturation in axial slices (e), and dynamic MRA after injection of gadolinium in maximum intensity projection reconstruction after decoupage including only the internal carotid arteries seen from the front (f). Dissection of the right sub-petrous internal carotid artery with a mural haematoma, which is hyperintense in T1 in the form of an "eccentric crescent" (a), already visible on a low slice of the axial encephalic FLAIR sequence, with tight luminal stenosis of the sub-petrous portion (d), responsible for a recent embolic ischemic accident of the right middle cerebral territory (c). The check-up at 8 months shows resolution of the mural haematoma (e) with disappearance of the luminal stenosis (f).

CT angiography can also be used and shows the same modifications of the lumen as in conventional angiography and in MRA with contrast [86,92,93].

## Therapeutic approach

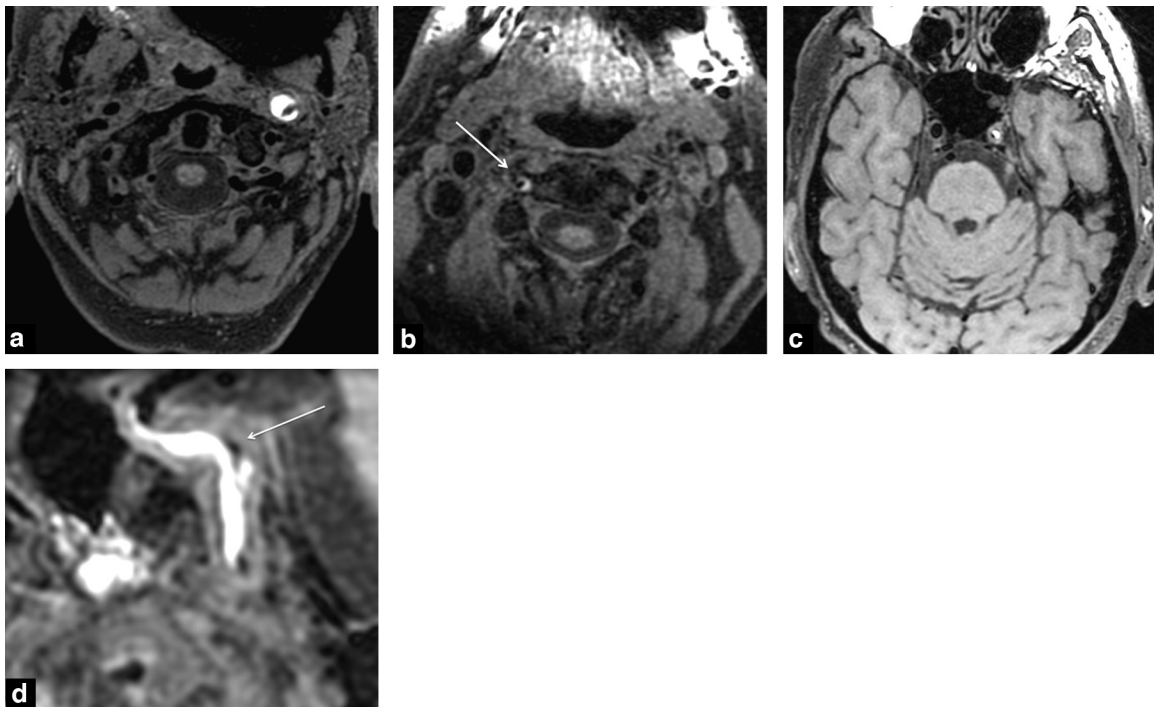
### Non-specific measures

The general recommendations for the treatment of CAD are the same as those prescribed for an acute cerebral infarction with the same vascular risk factors [94]. Intravenous thrombolysis is not contraindicated. There are no validated

scientific data concerning the treatment of dissection without intracranial extension. The debate over whether to initiate treatment with aspirin or anticoagulant has not been resolved. However, it is standard practice to initiate anticoagulant treatment [91]. Treatment with aspirin or anticoagulants does not seem to increase the dimensions of mural haematomas [95,96].

### Interventional vascular neuroradiology

The treatment of CAD via interventional neuroradiology should be discussed on a case by case basis, given the



**Figure 6.** Patient with an internal carotid and vertebral CAD. MRI with T1 weighting with suppression of the fat and circulating blood in axial (a–c) and sagittal slice reconstructions (d). CAD with a haematoma, hyperintense in T1, affecting the sub-petrous and cavernous portions of the left internal carotid artery (a, c and d, arrow) and portion V3 of the right vertebral artery (arrow).

lack of scientific data evaluating this method. In the event of narrow stenosis with haemodynamic repercussions or recurrent infarction under anti-thrombotic treatment, an angioplasty with or without stenting may be proposed [97]. In the event of tandem occlusion, internal carotid - middle cerebral, the rate of sylvian recanalisation after angioplasty-stenting is greater than with intravenous fibrinolysis alone [98].

## Conclusion

Cervical arterial dissections represent the primary cause of ischemic cerebral vascular accidents in patients under 45 years of age and are commonly located in the sub-petrous area, sparing the bulb for the internal carotids and at the level of the V2–V3 junction for the vertebral arteries. They are manifest by the formation of a mural haematoma, which may or may not communicate with the arterial lumen via an intimal breach and can thus cause stenosis, or even occlusion of the arterial lumen. The mean time taken for the arterial lumen to return to normal is around 3 months, with an annual relapse rate of around 1%. In the majority of cases, the ischaemic signs are preceded by local signs (cervical pain, headaches, Horner's syndrome) with a delay that is generally less than 15 days. MRI is the reference examination, enabling identification of the mural haematoma (T1 slices with fat saturation) to assess the effects on the lumen (stenosis, occlusion, pseudo-aneurysm in MRA) and to diagnose any possible cerebral ischaemia (diffusion). Treatment in the acute phase may include anti-platelet aggregation or an anticoagulant. Endovascular treatment combining angioplasty and possibly stent placement at the site of the

dissection, may be proposed, after multidisciplinary consultation, in the event of recurrent neurological deficits with haemodynamic anomalies.

## TAKE-HOME MESSAGES

- Cervical arterial dissection comprises the formation of a mural haematoma, which may or may not communicate with the arterial lumen via an intimal breach. Sub-intimal dissections affect the arterial lumen (stenosis, occlusion), sub-adventitial dissections enlarge the external diameter of the artery (pseudo-aneurysm).
- Carotid dissections often have a sub-petrous location and always spare the bulb. The most common site of vertebral dissections is the V2–V3 junction.
- The ischaemic signs are preceded by local signs (cervical pain, headaches, Horner's syndrome) within 15 days in the majority of cases.
- MRI is the reference examination, enabling identification of the mural haematoma (T1 slices with fat saturation), an assessment of the effects on the lumen (stenosis, occlusion, pseudo-aneurysm in MRA), and the diagnosis of any possible cerebral ischaemia (diffusion).
- The mean time taken for the arterial lumen to return to normal is around 3 months. At one year, around 80% of dissections cease to change. The annual risk of recurrent dissection is around 1%.



## Clinical case study

This 28-year-old patient gave birth at 38 weeks amenorrhoea, 24 hours previously. She presented with unusual headaches, with a left Horner's syndrome on clinical examination.

### Questions

- The MRI protocol should include:
  - cerebral MRI alone
  - cerebral MRI + MRA of the supra-aortic trunks + axial cervical slices
  - brain: FLAIR, TOF, T2\*
  - axial cervical slices: T1 weighting with fat saturation (2D, 3D)
- What are the commonly found anomalies in cases of carotid dissection occurring within the past 24 hours?
  - Stenotic forms: long and regular stenosis
  - Aneurysmal forms: sacciform or fusiform aneurysm, with or without stenosis
  - Occlusive forms: filiform flame-shaped thrombosis
  - Intimal flap and double channel
- What would you expect to find on this examination?
  - Sometimes no anomaly at all
  - Increase in the external diameter of the artery
  - Mural haematoma surrounding the arterial lumen, which is hypointense (black blood)
  - The mural haematoma would probably be hypointense in T1; hyperintensity appears in the subacute stages, from day 3–5 (intracellular MetHb)
  - It would be useful to repeat the examination after 3 to 4 days to confirm the diagnosis

### Answers

- What is your MRI protocol?
 

B and d. Imaging of the supra-aortic trunks and axial slices essential given the symptoms, which are evocative of carotid dissection. Always perform a diffusion sequence ("silent" infarction).
- What are the commonly found anomalies in cases of carotid dissection occurring within the past 24 hours?
 

A and c. Stenotic and occlusive forms are the most common forms. "Aneurysmal" forms are exceptional in the acute phase. In which case, a recurrent dissection should be suspected. Intimal flap and double channel are seen exceptionally.
- What would you expect to find on this examination?
 

A, b, d, and e. The examination may be normal. An increase in the external diameter of the artery is one of the cardinal signs of dissection. The mural haematoma is hypointense in T1 for the first three days. It is therefore a good idea to repeat the examination 3 or 4 days later.

## Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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