Traumatic Dissection of Arterial Cervical Vessels: Report of Two Cases and Literature Review

Dissecção arterial traumática de vasos craniocervicais: Relato de casos e revisão da literatura

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Abstract

Keywords
► internal carotid dissection
► vertebral artery dissection
► cerebrovascular trauma

Even though traumatic dissection of cervical arterial vessels is the major cause of stroke among adults, it is still an underdiagnosed disease in neurosurgical emergencies, since most patients do not have or present subtle clinical signs in the acute phase. The authors report two interesting cases of cervical artery dissection with different traumatic mechanisms and present a broad literature review about this subject.

Resumo

Palavras-chave
► dissecçao artéria carótida
► dissecçao vertebral
► trauma cerebrovascular

Embora a dissecção traumática de vasos cervicais seja um das principais causas de isquemia cerebral em adultos, ainda é um patologia subdiagnosticada nas emergências, uma vez que os pacientes são assintomáticos ou oligossintomáticos na fase aguda. Os autores descrevem dois casos interessantes de dissecção de artérias cervicais por diferentes mecanismos traumáticos, seguidos de ampla revisão da literatura sobre o tema.

Introduction

Arterial dissection corresponds to a detachment between the layers of the artery wall creating a false blood path. This phenomenon either impairs the perfusion of tissues downstream or causes bleeding.

According to its etiology, it can be classified as spontaneous, iatrogenic or traumatic. The mortality of traumatic cases range from 20 to 40%, depending on the association with other traumas. Furthermore, it is usually an underdiagnosed etiology of stroke, whose neurologic sequelae, in this scenario, affects between 40 and 80% of the patients.[1]

Traumatic dissection requires specific considerations that will be addressed in this article associated with the reports of two cases.

Case Report

Case 1
A 52-year-old male patient sought hospital care reporting pain in the left anterior cervical region. The pain started abruptly, after rotational movement of the neck to hit a ball with the head during a soccer match, and persisted with intensity 4/10 on the following days. Two days after the event, he woke up with complete right hemiparesis associated with motor aphasia and mild dysarthria. The patient underwent cranial tomography without contrast, which did not show hemorrhages nor hematomas, but revealed loss of cortico-subcortical differentiation in the vascular territory of the upper trunk of left middle cerebral artery. The patient was not submitted to the thrombolysis criteria due to having an ictus delta longer than 4 ½ hours. During hospitalization and etiological investigation of the ischemic event, signs compatible with dissection of the left carotid artery were identified on angiotomography (►**Fig. 1**). The patient received treatment with antiplatelet therapy associated with strict blood pressure control and motor physiotherapy. During follow-up, a significant paresis improvement in the right lower limb and aphasia was shown, despite monoparesis in the right upper limb, that persisted during 6 months of follow-up after the event, with a strength grade 3.

Case 2
Female patient, 39 years old, victim of car accident, received medical attention at the scene and was transported to the polytrauma reference hospital. On hospital arrival, at the emergency room, the patient was alert, oriented in space and time, Glasgow coma scale was 15/15, there was no motor deficits and cranial nerves were intact. During abdominal examination, she reported abdominal pain, whose investigation showed a small
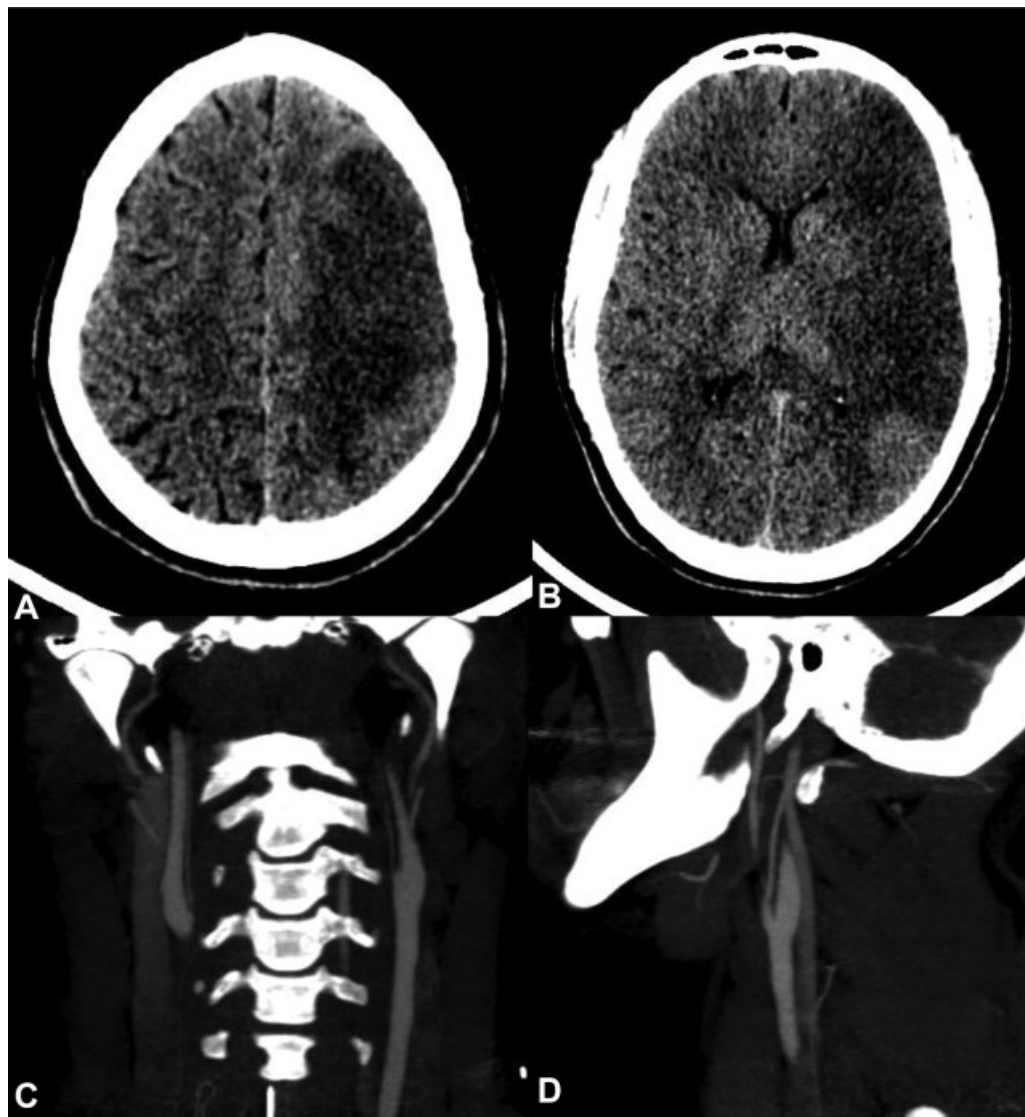

Fig. 1 Traumatic dissection of the left internal carotid with ischemia on the vascular territory of the left middle cerebral artery. Non-contrasted CT scan (**A** e **B**), hypodensity in the territory of the left middle cerebral artery. Angiotomography of cervical arteries showing gradual narrowing and blood flow obstruction in the left internal carotid artery (**D**). Angiotomography showing right internal carotid dissection

amount of free fluid in the pelvis and pneumoperitoneum. Exploratory laparotomy was performed and a laceration in the jejunum was identified at 20 cm from the Treitz angle, qualified for primary correction. The patient had a good evolution in the immediate postoperative period. On the 2nd postoperative day (and after the trauma), the patient developed hypoesthesia and monoparesis in the left upper limb, without hyporeflexia or alterations restricted to a dermatome and myotome (thus excluding injury to the second radicular motor neuron or brachial plexus). In the subsequent hours, the neurological condition worsened, evolving with complete left hemiplegia. The CT investigation evidenced sparse areas of hypodensity in the territory of the right middle and right anterior cerebral arteries. Further investigation with angiotomography pointed the dissection of the right internal carotid artery in its cervical portion (►Fig. 2). The patient received antiplatelet therapy, with slight neurological improvement, and was discharged from the hospital with complete and disproportionate left hemiparesis, with force 1 on the distal left upper limb and grade 2 proximal, and force 2 on the left lower limb.

Discussion

Arterial Anatomy

The arteries that irrigate the brain originate from the brachiocephalic branch (common right carotid artery), from the aortic arch (common left carotid artery) and from the right and left subclavian arteries (right and left vertebral artery, respectively). The common carotid artery divides itself at the

carotid bulb level (topography of the 4th cervical vertebra) in internal carotid artery (ICA) and external carotid artery (ECA). Based on the classification published by Bouthillier, the ICA is divided in 7 segments, from C1 to C7 (►Fig. 3).[2] The cervical segment from the ICA is located medially to the internal jugular vein and anteriorly to the vagal nerve, ascending in the neck toward the petrous portion of the temporal bone.

The vertebral artery (VA) is classified in 4 segments. The first segment (V1) starts in the origin of the VA and goes up to the vertebral foramen of the sixth cervical vertebra. The V2 segment ascends throughout the cervical foramen to the second cervical vertebra. From the exit of this vertebra foramen ahead it is considered the V3 segment, in which there is an external curvature followed by an internal curvature that stays along the superior border of the Atlas and ascends from the foramen magnum to the intracranial space. From this point on, it is considered the V4 segment (►Fig. 4).
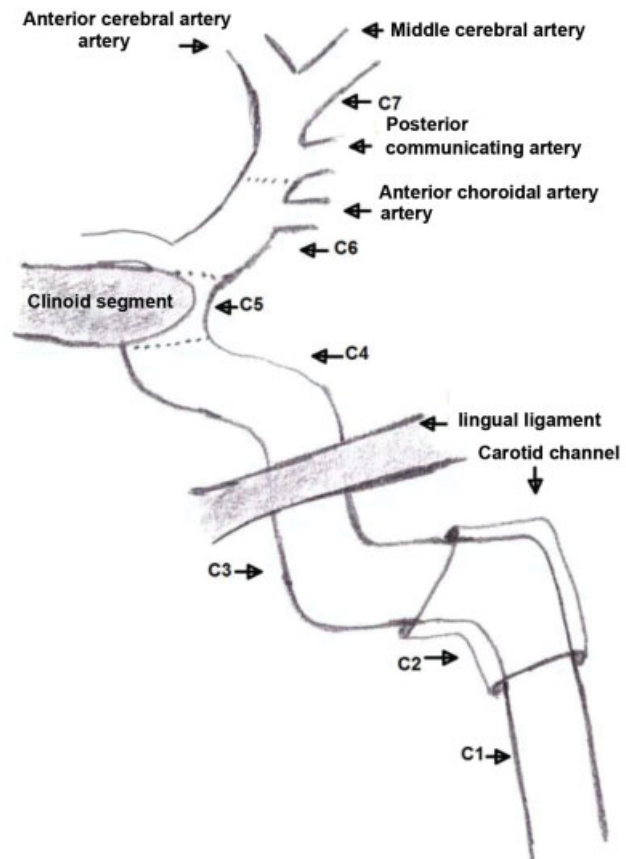
Fig. 3   Internal Carotid Artery. C1–cervical segment: ascends from the carotid bifurcation into the entrance of the temporal bone. C2–petrous segment, divided in two subsegments inside the carotid channel – ascending and horizontal parts. C3–lacerum segment, from the carotid channel to the lingual ligament. C4–cavernous segment from the lingual ligament through the cavernous sinus (posterior ascending, horizontal and anterior ascending parts). C5–clinoid segment, a wedge shaped segment between the proximal and distal dural ring. C6–ophtalmic segment, originates in the distal dural ring extending up to the origin of the posterior communicating artery. C7–communicaitng segment, from the emergence of the posterior communicating artery until carotid bifurcation.
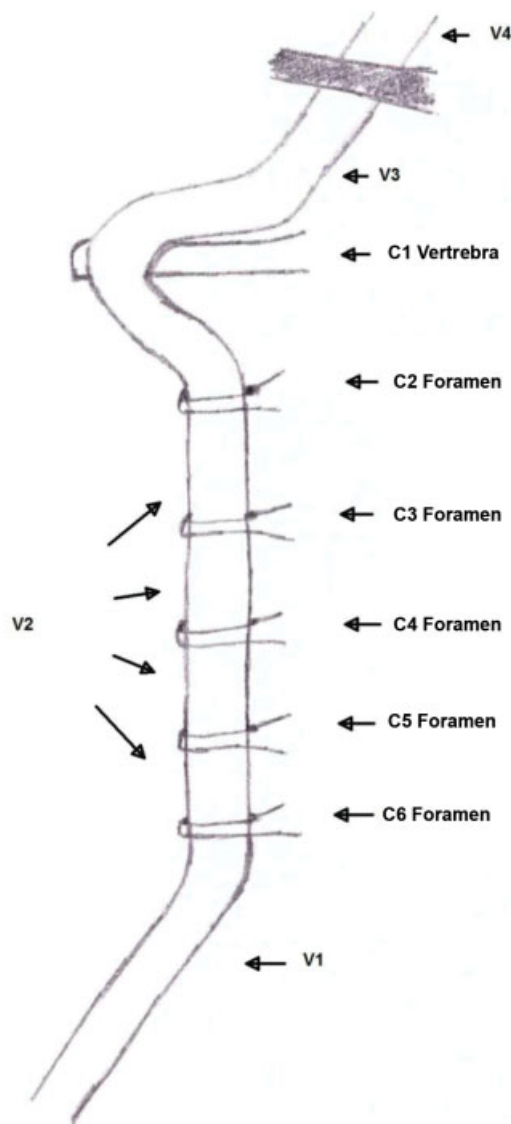
Fig. 2   Angiotomography showing dissection of the right carotid artery without arterial flow above the dissection

Fig. 4 Vertebral Artery

Epidemiology

Arterial dissection of extra- and intracranial vessels is the main cause of stroke in young adults.[3] Yet, they present different patterns of incidence; traumatic carotid dissection has its peak in the 6th decade of life, while vertebral dissection presents a more homogeneous distribution between the 3rd, 4th and 6th decades of life.[4]

Dissection of the extradural portion of the craniocervical arteries is more common than intradural, since it is a mobile segment with no rigid structures of protection. Analyzing extradural branches, the ICA is more affected than the VA, in a proportion of 3:1.[5] When considered intradural lesions, the VA is more affected than the ICA.

Considering patients victims of craniocervical trauma, without stratifying the cinematics, the incidence of carotid artery or VA dissection varies from 1.7 to 4.9%. Also, the incidence of combined traumatic lesion of the carotid artery with the vertebral is 6.5%. The segment C1 of the ICA is the

most common site of the dissection, with an estimated incidence of 2.5 to 3.0/100,000 people. On the other hand, the extradural VA dissection has an estimated incidence of 1.0 to 1.5 cases/100.000 people, independent of the trauma mechanism.[4,6–8]

Analyzing only craniocervical blunt trauma victims, the incidence of carotid lesions varies from 0.1 to 2.6%; and, only among polytrauma patients, the incidence reaches 2.7%. On the other hand, the incidence of vascular lesions in victims of penetrating trauma varies from 3 to 40%.

However, the true incidence of carotid dissection due to trauma may be greater, since the diagnosis is established usually when symptoms set in, which can be immediately after the trauma or during its segment (due to ischemia, progressive dissection, thromboembolic symptoms or bleeding).[9,10]

Etiology

Dissections can occur in penetrating or blunt craniocervical trauma. Carotid dissection of blunt trauma is predominantly associated with severe kinematics and direct trauma to the cervical region – being its main cause automobile collisions. Traumas that result in skullbase fractures (especially in the petrous segment of the carotid canal) also represent a high risk for carotid dissection.[8]

In 1974, Crissey et al described four mechanisms that can lead to C1 injury: 1–direct trauma through anterolateral direction of the neck, 2–cervical hyperextension associated with rotation, 3–blunt intraoral trauma and 4–fractures of the skull base bones involving the carotid canal. In addition, mechanisms of distraction/flexion, distraction/extension or lateral flexion forces on the cervical spine can result in carotid or vertebral dissection.[9]

On the other hand, vertebral dissection in blunt trauma is not necessarily associated with the severity of the trauma. It can occur in trivial traumas, such as spinal manipulation maneuvers or even Valsalva maneuvers. The VA dissection in car crashes of severe kinematics is usually associated with a fracture or dislocation of the cervical spine. The VA is more susceptible to dissection by blunt trauma in V1 and V3, which are segments of greater mobility. The V2 segment is more susceptible to foraminal bone lesions or cervical dislocations.[7]

Penetrating craniocervical injuries can be caused by stab wounds, explosives or projectiles. The kinematics of these cases can cause direct lesions in the tissues they pass through, destroying structures along their path. More severe kinematic injuries, such as high-speed projectiles of civil and military war, cause direct and indirect injuries, which are disruptions of adjacent tissues by shock waves. These injuries can affect the blood vessels, leading to ischemia (intimal dissections with obstruction of the vessels) or bleeding with arteriovenous fistulas and traumatic pseudoaneurysms (when they affect the adventitial layer).

Although the external factors are crucial conditions, some underlying pathologies may predispose to traumatic dissection of cervical vessels. Among them are Marfan syndrome, Ehler-Danlos syndrome type IV, autosomal dominant

polycystic kidney disease, type I imperfecta osteogenesis, α1-antitrypsin deficiency and fibromuscular dysplasia.

Pathogenesis

The arterial wall consists of three layers (endothelium/intima, middle muscle layer and adventitia). Dissection is characterized as rupture and separation of these three layers, hence occurring either between the endothelium and the middle muscle layer (subintima dissection), or between the middle muscle layer and the adventitia (subadventitial dissection). Factors such as size of the endothelium lesion, associated hemodynamic forces and vascular resistance, determine whether the dissection will result in stenosis or aneurysmatic dilatation of the artery. Subintima dissection tend to cause stenosis, while subadventitia dissections tend to cause pseudoaneurysms.

The ICA dissection normally occurs in the first two centimeters after the carotid bulb, ending usually proximal to its entrance in the temporal bone. The VA is more susceptible at the entrance point in the transverse foramen of C6, because the artery is relatively fixed at the bony orifice of the foramen while the segment C5/C6 has elevated mobility. Besides, the greater mobility the vertebral artery has in rotation at the atlantoaxial junction or flexion/extension at the atlanto-occipital junction makes this region particularly susceptible to dissection by minor trauma.

The traumatic extradural dissections of the ICA and VA usually presents with thromboembolic events with subintima lesions, resulting in stenosis, occlusions and thromboembolism. The intradural dissections tend to cause subadventitia lesions, resulting in vessel rupture (with bleeding) or formation of a pseudoaneurysm (with immediate or late bleeding).

Clinical Manifestations

Neurological changes can occur over the first 24 hours after the trauma and only 10% of patients have clinical manifestations when arriving at the hospital.[4,7] Clinical warning signs include cervical hematoma, cerebral infarction identified at computed tomography (CT), type II or III Le Fort fracture, Glasgow coma scale (ECG) < 6 points and skull base fractures. In penetrating skull trauma, lesions close to the pterion, or crossing the midline, lead to intracranial vascular injury in up to 40% of cases.[10]

When present, clinical manifestations of dissection range from headache and cervical pain to severe neurological compromise. Because of different vascular territory supply, neurological manifestations of carotid lesions are distinct from the vertebral lesions. Despite this, extradural segments show common signs and symptoms such as cervical pain, hemorrhage externalized by the upper airway or expanding cervical hematoma.

A classic triad of traumatic carotid dissection is characterized by pain (cervical region, face and head), Horner's syndrome and ischemic cerebral symptoms or ipsilateral retina ischemia (secondary to the ophthalmic artery embolization). The three components are found concurrently in less than a third of patients with C1 dissection.

The usual clinical manifestation of V1 dissection includes cervical and occipital pain, of high intensity, with symptoms of cerebellar ischemia (such as vertigo, dysdiadochokinesia and ipsilateral dysmetria).

Intracranial vessels dissections tend to bleed immediately after rupture of the vessel, or later, due to bleeding from pseudoaneurysms. The characteristic clinical picture of ICA dissection and rupture of its intracranial portion is a carotid-cavernous fistula when the rupture is found in the cavernous segment (C4). This lesion comes along with a cavernous syndrome: paresis of the extrinsic ocular musculature, eyeball protrusion, chemosis, eye pain, but preservation of visual acuity (►Fig. 5).

Diagnostic Imaging

Diagnosis of craniocervical vessels dissection is confirmed with imaging tests that allow visualization of the vessels as well as their blood flow. For this matter, CT, magnetic resonance imaging (MRI) and arteriography are the exams that can provide the diagnosis, with different degrees of sensitivity and specificity. When there is a reduction in the lumen of the vessel > 50%, the use of cerebral ultrasound with Doppler is another method that can be used.[11]

It is a challenge to make the diagnosis in asymptomatic patients and determine which patient should be submitted to a vascular investigation. Some criteria have been described to increase the investigative positive predictive value. Vascular lesions can be found in 44 to 90% of the patients with the following findings: blunt trauma of severe cinematic, diffuse axonal injury, ECG ≤ 8, face fractures Le Fort II or III or fractures of the skull base (especially from the temporal bone). In these cases, an image study must be performed.
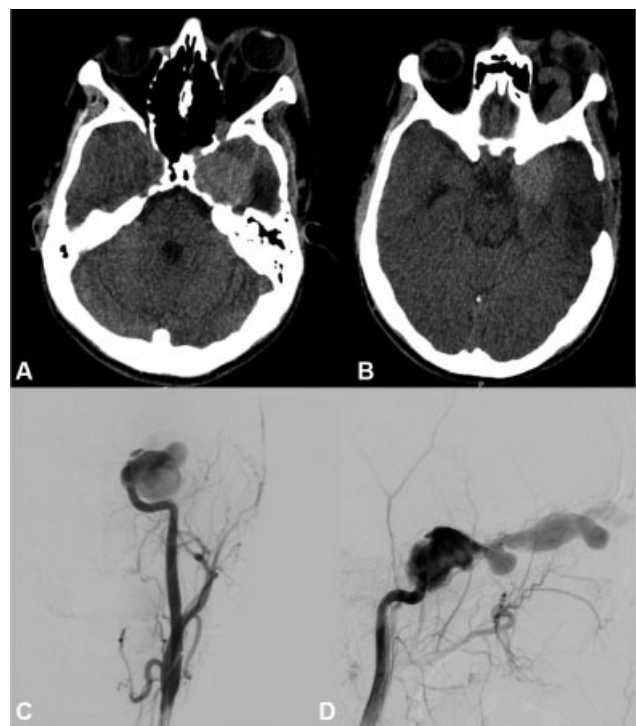

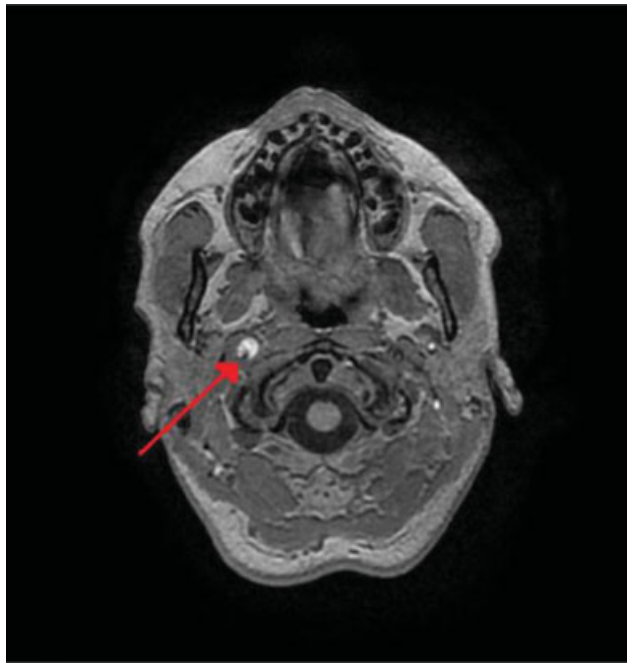**Fig. 5** Patient with traumatic carotidcavernous fistula

Fig. 6 Angioresonance showing right internal carotid dissection.

Computed Tomography

Although the gold-standard exam for diagnosis of dissection may be arteriography, computed angiotomography is, possibly, a more appropriate test for screening severe polytrauma patients. This examination allows the visualization of the cerebral parenchyma in search of signs of infarction, in addition to the visualization of the patency of the cervical vessels and brain vascularization. The limitation of the angiotomography is the exposure to radiation (mainly for children, adolescents and pregnant women). Furthermore, the hypodense image in the region of the cervical vessels, which corresponds to intramural hematomas, can also be seen in atheromatous plaques, making it difficult to distinguish and establish the right diagnosis.[12]

Magnetic Resonance Imaging

Craniocervical MRI and angiotomography combined present a sensibility of 99%. These methods are especially useful in pregnant women, young patients or patients with renal failure, since they do not use ionizing radiation or iodinated contrast agent.[12]

The evaluation of the arterial dissection in the MRI consists of acquisition phases[1]: diffusion images – diffusion weighted imaging (DWI) and fluid attenuation inversion recovery (FLAIR) to assess the occurrence of cerebral infarction secondary to dissection[2]; T1 and T2 images to assess the presence of intramural hematoma[3]; angioresonance (angioRM) to assess the vessel lumen. The classic finding of dissection in MRI is an eccentric periluminal halo, indicative of intramural hematoma. The intramural bleeding and its expansion can be easily identified in sequences with the fat saturation technique (►**Fig. 6**).[13]

Time between dissection onset and images in MRI or angiotomography is a potential limitation for this method, because the sensibility is greater in the first 2 days after the dissection. Hence, the evolutive pattern of presentation must be considered. In the acute phase, the hematoma consists primarily of deoxyhemoglobin and has an isointense presentation when compared with the underlying muscle tissue. Subacute hematomas contain intra and extracellular methaemoglobin and therefore present hypersignal in both T1 and T2. This pattern persists for months, when the hyperdensity gradually becomes isointense. Ruptures of the intima may also be visualized in MRI, especially in T2.

Arteriography

Arteriography is the gold standard method for diagnosis of craniocervical arterial dissection. Besides the sensitivity of 97 to 100%, it allows endovascular therapy when indicated.[13]

The most common finding in arteriography is the irregular size of the vessel with its gradual thinning from the breaking point of the wall. Other arteriographic finding of carotid dissection is the "double-lumen" sign[13] (►**Fig. 7**).
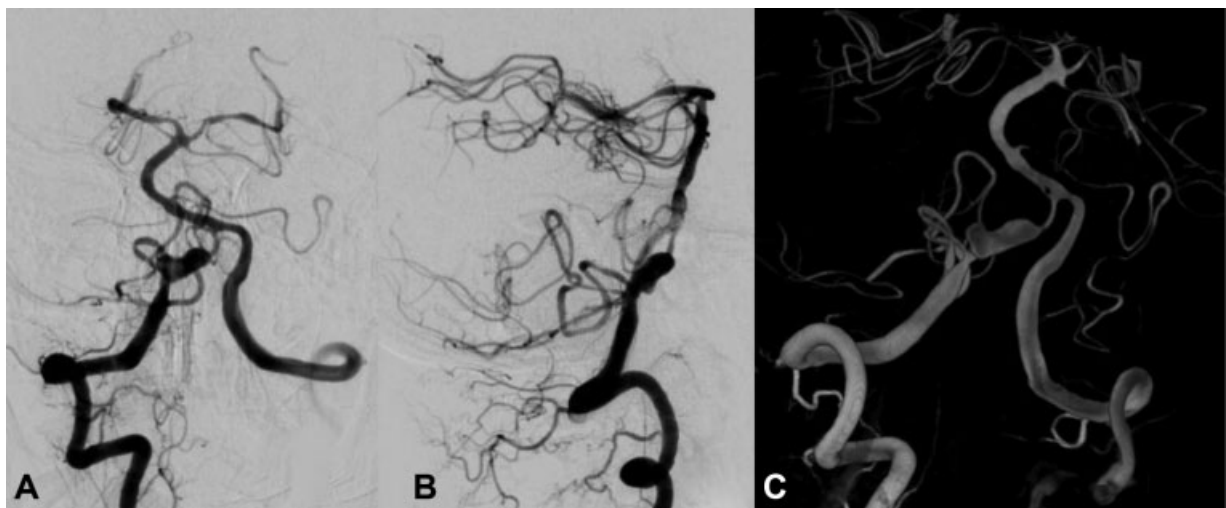

Fig. 7 Dissecting traumatic aneurysm on the right vertebral artery with luminal narrowing distal to the aneurysm

Although it is considered the gold standard, arteriography is unable to assess the thickness of the vessel wall, because it shows only its lumen. In cases of subadventialial dissections, there may be no significant lumen stenosis, thus providing a false-negative result.

It is worth mentioning that arteriography is an invasive exam with risks related to the insertion of the catheter (vascular perfusion, hematomas or pseudoaneurysms in the femoral artery), to the contrast administration and also risk of cerebral ischemia.

Ultrasound
The sensitivity of ultrasound to detect carotid and vertebral dissection varies from 70 to 90%, with the exception that it will only be evident on Doppler when the stenosis reduces the vessel light by at least 50% and there is high resistance to distal flow. The direct signs are the visualization of the intramural hematoma itself, a double lumen in the inner layer of the vessel, a double lumen with a thrombus in the center, the pencil tip sign (abrupt termination of the flow due to the thrombus), the guitar rope sign (when the light narrowing is > 75%), and the rat tail sign (progressive reduction of the vessel lumen).

On the other hand, the indirect signs are the increase in the pulsatility index (the intramural hematoma compromises the flow at the dissection site) and a difference > 50% in the flow speed when compared with the normal side.[11]

Another contribution of ultrasound is with transcranial doppler (TCD) for tracking internal carotid dissection in cases of traumatic brain injury (TBI). Because the clinical manifestation may be difficult to establish the diagnosis, asymmetry > 25% in the speed flow, as well as reduction of pulsatility index (< 0.8), suggests dissection of the ipsilateral internal carotid artery. This finding in the routine TCD of the TBI can serve as an alert for suspected dissection, and a more accurate assessment of the craniocervical vessels is indicated.

Prognosis
Mortality due to traumatic carotid and VA dissection, without treatment before clinical manifestations, is ∼ 25%. About ¼ of vertebral injury survivors evolve with neurological sequelae, reaching 40% in cases of carotid injuries.[14] However, retrospective studies suggest a 6% incidence of cerebral ischemia in traumatic dissections if treated in the asymptomatic phase.[4]

Treatment
Conservative treatment of traumatic lesions in the cervical region is usually adopted, with endovascular or surgical therapy reserved for exceptional cases. Surgical or endovascular treatment is reserved for cases with refractory luminal irregularities, worsening of the neurological condition despite drug treatment or expanding pseudoaneurysm. About 16.3% of patients fail drug therapy and require surgical or endovascular intervention.

Conservative Treatment
The treatment of carotid or vertebral dissection involves antithrombotic therapy with either anticoagulation or anti-platelet therapy, whose determination of which therapy is more efficient is still controversial.[15] In either choice, drug treatment should be started promptly, especially for the prevention of future thromboembolic events, and maintained for a long period. Besides, drug treatment is recommended even in asymptomatic patients or with small dissections.

When anticoagulation is chosen, the initial choice is by parenteral route, with unfractionated heparin. After starting the therapeutic dose, the maintenance dose is adjusted according to the activated partial thromboplastin time (APTT), which must be between 50 and 70 seconds. Then oral anticoagulation is started, which must be continued for 3 to 6 months with the goal of maintaining the international normalized ratio of prothrombin time (RNI) between 2.0 and 3.0.

If antiplatelet therapy is chosen, it can be performed with acetylsalicylic acid 100mg/day and Clopidogrel Bisulfate 75 mg/day.[16]

Both platelet antiaggregation and anticoagulation are equivalent in terms of effectiveness in reducing neurological sequelae, without increasing the risk of bleeding.[17]

It is noteworthy that, although many patients with cervical vessels dissection present signs and symptoms of neurological impairment and brain infarction present on cranial CT, intravenous thrombolysis is not indicated. This last therapy is contraindicated due to the high risk of bleeding at the site of the lesion in the vessel wall.

Endovascular Treatment
Endovascular treatment indications in this scenario include cases of complete vessel transection with blood leakage to the neck, recurrent symptoms despite drug treatment, cerebral hypoperfusion (in situations of multiple vessel involvement or poor collateral circulation), patients with anticoagulation contraindication (previous intracranial hemorrhage) and patients with symptomatic or expanding pseudoaneurysms.

Previously, endovascular treatment consisted of the original vessel occlusion through embolization. Recently, reconstructive stent-assisted coil embolization and placement of a flow-diverted stent have been reported as effective therapeutic modalities. The stent-assisted pseudoaneurysm embolization technique in the original vessel is recommended for dissections with saccular portions, since coils can obliterate the weakest area of the aneurysmal sac and the stent can promote endothelial growth, preventing narrowing secondary to dissection. However, this technique requires the use of combined antiplatelet therapy, so it needs careful assessment.[14]

Surgical Treatment
Surgical procedures are preferentially reserved for complex lesions that are unable to be treated by endovascular technique. Arterial obliteration, in the vertebral artery, by surgical ligation is considered tolerable in the context of therapeutic failure of the other modalities. Extraintracranial vascular reconstruction for the carotid system by bypass may be indicated in specific cases.

Conflict of Interests
The authors have no conflict of interests to declare.

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