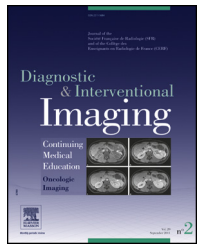
Diagnostic and Interventional Imaging (2014) **95**, 1151—1161



CONTINUING EDUCATION PROGRAM: FOCUS...

Imaging of cervical artery dissection

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KEYWORDS
Arterial dissection;
Mural haematoma;
MRA;
High resolution MRI;
Cerebrovascular accident

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.

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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http://dx.doi.org/10.1016/j.diii.2014.10.003

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 vascularitis

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