Carotid & Vertebral Artery Dissection - Update on Proper Management

Ioannis Stamatatos1*, Stavroula Ksylogiannopoulou2, Fragkiskos Tzagkarakis1, Konstantinos Bouboulis3,4, Basileios Mpoumis1, Maria Lykouri2, Efstatios Metaxas4, Dimitrios Lioumpas4, Dimitrios Ksekalakis3, and Ioannis Markakis2

1Department of Vascular & Endovascular Surgery, General Hospital Nikaia, Greece
2Department of Neurology & Neuroscience, General Hospital Nikaia, Greece
3Second Department of General Surgery, General Hospital Nikaia, Greece
4Department of Cardiothoracic Surgery, General Hospital Nikaia, Greece

Abstract

Background: Dissection of the internal carotid and vertebral arteries is increasingly recognized as a cause of ischemic stroke in young people. Spontaneous cervical-artery dissection typically occurs in young and middle-aged persons, with a slight preponderance among women.

Methods: PubMed, Scopus and Google Scholar databases were systematically searched according to the recommendations of the PRISMA statement for administrative dataset registries reporting outcomes after spontaneous dissection of the internal carotid and vertebral arteries.

Results and Conclusions: Antithrombotic therapy has been the mainstay of medical treatment of cervical artery dissection. Despite the lack of level I evidence, most patients with cervical dissection are treated with systemic heparin followed by vitamin-K antagonists for 3 to 6 months.

INTRODUCTION

Cervical carotid artery dissection occurs most frequently in the third through the fifth decades of life, and the mean age at diagnosis is 45 years. The overall incidence of clinically apparent spontaneous carotid dissection is reported to be between 1.7% and 2.6% [1,2].

The incidence of cerebrovascular events related to vertebral artery dissection is greater than previously reported and might be more frequent than carotid artery dissection [3].

Arterial dissection occurs when disruption of the intima allows blood to extravasate between layers of the vessel wall. The resulting intramural hematoma usually extends distally and can lead to acute stenosis or occlusion.

Atherosclerosis and other known risk factors for vascular disease such as smoking, diabetes, hypercholesterolemia, and oral contraceptive use are usually absent in patients with spontaneous dissection. These patients do have a higher incidence of hypertension, and a migraine disorder has also been shown to have an independent association with spontaneous dissection [4].

Cervical artery dissection is classified based on the artery involved (vertebral vs. carotid) and the location of involvement (intracranial vs. extra cranial). The most common type is extra cranial internal carotid dissection which typically occurs 2-3 cm above the bifurcation and accounts for up to 2.5% of all first strokes [5].

Clinical findings

Carotid artery dissections typically begin with ipsilateral neck pain or headache and a partial Horner’s syndrome followed by retinal or cerebral ischemia. The presence of any two of the three elements in the triad strongly suggests the diagnosis of carotid dissection [6]. The Horner is usually partial (miosis and ptosis without anhidrosis) due to the fact that sudomotor fibers of the face travel along the external carotid artery.

Vertebral artery dissections typically present with occipito-cervical pain, which may be followed by a variety of posterior circulation ischemic symptoms including vertigo, dysarthria, visual field deficit, ataxia, and diplopia. Although strokes from vertebral artery dissection most frequently involve the lateral medulla and cerebellum, spinal cord infarction may occur when extra cranial branches are affected.

Patients with cervical carotid artery dissection also suffer from neck pain, amaurosis fugax, anisocoria, pulsatile tinnitus, and cranial nerve palsy [7]. Cranial nerves IX through XII and in particular the hypoglossal nerves are most commonly involved [8].

Diagnostic evaluation

Although ultrasound is readily available and thus commonly
used as the initial test, four-vessel selective cerebral angiography remains the "gold standard". Angiography allows visualization of the arterial lumen and characterization of the lesion. An intimal flap or double lumen is pathognomonic for dissection. The internal carotid stenosis caused by dissection is usually irregular, originates 2 to 4 cm distal to the bulb, and has a long tapering stenosis that usually ends before the internal carotid artery enters the petrous portion of the temporal bone [9].

Another method of diagnosis is magnetic resonance angiography (MRA) along with a T1 axial cervical MRI with fat saturation technique due its lack of radiation, high sensitivity and specificity, and ability to visualize an intramural hematoma [10]. Computed tomography angiography (CTA) also has high sensitivity and specificity and may also be used but is associated with radiation exposure and potential technical challenges [11].

**TREATMENT**

**Medical Therapy**

Antithrombotic therapy has been the mainstay of medical treatment of cervical artery dissection. Concerns regarding anticoagulation include possible worsening of intramural bleeding at the site of dissection and bleeding from unrelated sources.

The American Heart Association (AHA) guidelines state that for patients with dissection with a stroke or transient ischemic attack, antithrombotic treatment for at least 3 to 6 months is reasonable. These guidelines also state that the relative efficacy of anticoagulation versus antiplatelet therapy is unknown [12].

Anticoagulation has been historically used in stroke prevention without good scientific evidence, which triggered several randomized trials including Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) and Warfarin-Aspirin Recurrent Stroke Study (WARSS) that showed no benefit of Warfarin over Aspirin in reducing stroke recurrence [13-15].

Additionally recently published Cervical Artery Dissection in Stroke Study (CADISS trial) randomized 250 patients within 1 week from stroke onset to antiplatelet or anticoagulation therapy. This study showed that there was no difference in stroke risk between the anticoagulant arm (1%) and the antiplatelet arm (3%) (Odds ratio 0.335, 95% CI 0.006-4.233; p=0.63) [16].

Despite the lack of level I evidence, most patients with spontaneous or traumatic dissection are treated with systemic heparin followed by warfarin for 3 to 6 months. Therapy after this is typically individualized and depends on vascular imaging [17].

**Surgical treatment**

Indications for surgical treatment of acute cervical artery dissection are fluctuating or deteriorating clinical neurologic symptoms despite medical treatment, compromised cerebral blood flow, contraindications to antithrombotic therapy, and a symptomatic or expanding aneurysm [18,19].

Once surgical exposure is obtained, therapeutic options include cervical artery ligation, interposition saphenous vein graft to a cervical or intracranial segment, and patch angioplasty. Carotid ligation is considered safe, if necessary, in patients with systolic stump pressure greater than 70 mm Hg [20].

Endovascular treatment is increasingly being applied to a number of acute cerebrovascular conditions, including stroke secondary to atherosclerosis and dissection [21].

In a recent endovascular study among 20 patients with anterior circulation strokes due to carotid artery dissection-related occlusions only 5 patients (25%) necessitated cervical stent placement and no early ipsilateral stroke recurrence was recorded, despite the absence of stent placement in 15 patients (75%) [22].

Furthermore Marnat G. and colleagues found no recurrence of ipsilateral stroke in 19 patients treated endovascular stent-free for tandem occlusion stroke secondary to internal carotid dissection [23].

Given the morbidity and mortality associated with open procedures, preliminary results describing endovascular treatment of carotid dissection seem promising.

Multidisciplinary management and full cooperation between doctors of different specialties ensures the optimal result in terms of time-brain saving in such of life threatening situations [24].

**DISCUSSION**

Dissection of the internal carotid and vertebral arteries is increasingly recognized as a cause of ischemic stroke in young people.

Although strokes caused by vertebral artery dissection most frequently involve the lateral medulla and cerebellum, spinal cord infarction may occur when extra cranial branches are affected.

Cervical vessels ultrasound is readily available and thus commonly used as the initial test, but four-vessel selective cerebral angiography remains the "gold standard" in diagnosis.

Despite the lack of level I evidence, most patients with spontaneous or traumatic dissection are still treated medically (heparin-warfarin) with good results.

Given the morbidity and mortality associated with open procedures, preliminary results describing endovascular treatment of carotid dissection seem promising.

Future randomized control studies on cervical dissection disease comparing outcomes after antiplatelet or anticoagulation therapy are needed to persuade that recommendations should be revised.

**REFERENCES**


