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Cerebrovascular Disorders (D Jamieson, Section Editor)

Treatment of Extracranial Arterial Dissection: the Roles of Antiplatelet Agents, Anticoagulants, and Stenting

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Abstract

Purpose of review Cervicocephalic arterial dissection (CeAD) is the most commonly identified cause of stroke in young healthy individuals. The management of acute ischemic stroke due to the diagnosed or suspected CeAD is well established and is appropriate for thrombolysis. There is a substantial risk of stroke recurrence in the early post-stroke period. The optimum method of stroke prevention in the subacute period remains debatable. In our review, we focused on the management of recurrent stroke in CeAD, the choice of various antithrombotic agents for stroke risk reduction with regard to specific pathogenetic mechanisms of dissections, and the utility of endovascular therapy.

Recent findings Recent studies suggest that various pathogenetic types of CeAD based on radiologic characteristics may be associated with greater risk of thrombogenicity, especially in the early post-stroke period. The use of anticoagulants has been shown to be effective in the eliminating microembolic signals (MES) detected by transcranial Doppler (TCD). The only randomized trial that compared combinations of antiplatelet agents and vitamin K-agonist anticoagulation did not find significant difference in risk of stroke, major bleeding, or mortality. The benefit of dual antiplatelet therapy cannot be excluded. Limited data on the use of direct oral anticoagulant agents (DOAC) is currently available. Endovascular therapy with stenting, while potentially effective, may

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pose significant risk of complications. Therefore, it needs to be carefully considered on a case-to-case basis.
Summary The recurrence of ischemic stroke in patients with CeAD is overall rare. No significant difference in treatment with various antiplatelet and anticoagulant agents has been shown in randomized trials. Only a few studies were based on radiological characteristics of dissections. An ongoing randomized trial is investigating the role of MES and the efficacy of antiplatelet versus anticoagulation agents. The role of DOAC agents has yet to be determined in clinical trials. Stenting in CeAD is an effective revascularization technique and may be considered in selected patients. However, current data is only based on low evidence level findings from small studies, lacking longitudinal outcomes and prognosis.

Methods

We searched and reviewed available, relevant, randomized trials; systematic reviews; meta-analyses; case series; retrospective reviews and other peer reviewed publications between January 1, 2015 and January 10, 2019. We used Medline, EMBASE, Cochrane Stroke Group Trials, and PubMed databases to search keywords: "carotid dissection," "vertebral dissection," and "cervical arterial dissection," in combination with "antithrombotic agent," "anticoagulation agent," "antiplatelet agent," and "endovascular treatment." We identified evidence-based peer-reviewed articles with the main focus on treatment of cervicocephalic arterial dissection (CeAD).

Introduction

The incidence of carotid or vertebral dissections is estimated at 3.5 to 4.5 per 100,000 [1–3]. CeAD accounts for up to 25% of all ischemic infarcts in individuals under 45 years [4, 5]. The diagnosis of CeAD may be challenging and is often missed due to the delayed onset of cerebral ischemia and the unclear temporal relation with the triggering event. Prior studies have shown that the majority of CeAD-related infarcts are secondary to a thromboembolic mechanism rather than a hemodynamic one [6, 7].

Dissection is an arterial wall intramural hemorrhage due to the rupture of vascular layers and adjacent vasa vasorum with or without an identifiable environmental trigger or underlying arteriopathy. Based on imaging characteristics, three main forms of dissections are recognized: stenotic, occlusive, and aneurysmal (Figs. 1 and 2). Occlusive dissections are associated with worse functional outcome [8]. It is hypothesized that injury to the intimal layer leads to the formation of both a subintimal false lumen and intravascular flap that may completely occlude the involved artery due to hematoma expansion. Involvement of the medial layer may lead to subadventitial dissection and cause formation of dissecting pseudoaneurysms with resultant local mass effect (e.g., Horner syndrome) rather than hemodynamic impairment [9].

Attempts were previously made to propose classification of CeAD types based on the presence of an intimal tear [10]. Dissections with preserved intima may not require antithrombotic treatment; moreover, it may potentially worsen

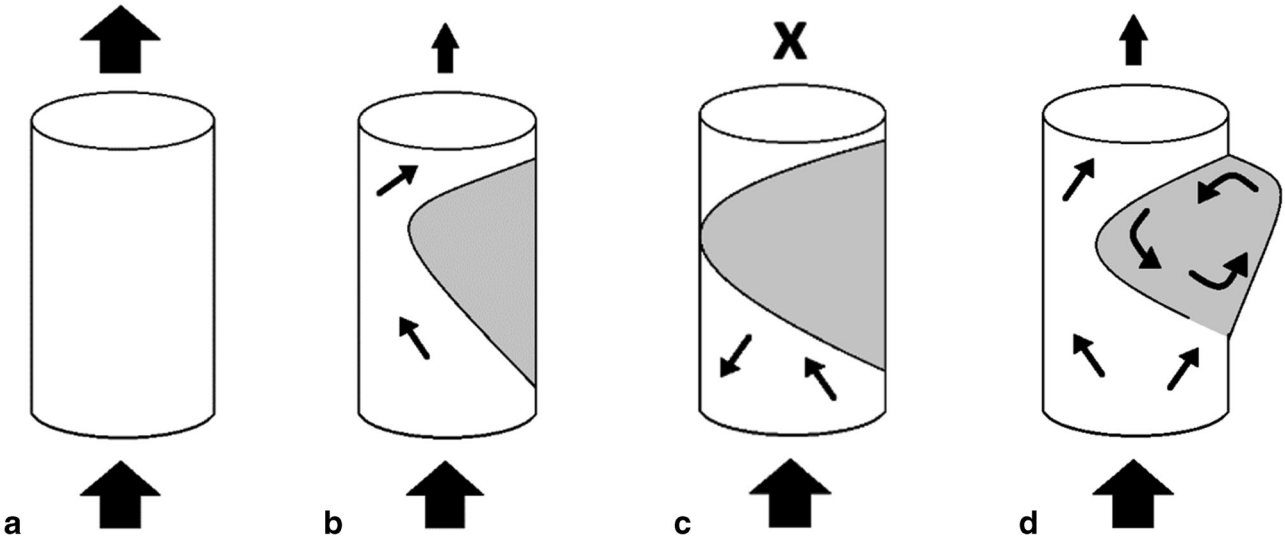

Fig. 1. Schematic of the three most common CeAD patterns. **a** Normal flow through an unaffected artery. **b** Reduced flow through a stenotic vessel after a dissection and formation of a false lumen in the gray area. **c** A completely occluded dissection pattern with no flow past the false lumen. **d** Reduced flow from a pseudoaneurysm formation secondary to dissection. Figure 1 is generated by Scott Le.

hematoma expansion and lead to progression of dissected vessel stenosis. The role of endovascular treatment in cases of hemodynamically significant stenosis may be of value and will be discussed elsewhere in this review. Whereas in vessels with an intimal tear that are considered highly thrombogenic, antithrombotic treatment decreases the risk of strokes. In most cases, in clinical practice with current imaging techniques, it is difficult to distinguish between the different types of dissections. Thus, it is not proven that a certain type of dissection poses a greater risk of stroke or that a specific treatment strategy is more effective in stroke prevention. None of the existent randomized trials were based on targeted pathophysiologic mechanism of dissections.

Acute management

The acute management strategy of patients presenting with CeAD-related strokes is well established, while long-term treatment is still debatable. It has been widely accepted [11, 12] and subsequently reflected in current AHA/ASA guidelines that patients presenting with an acute stroke in the settings of CeAD benefit from and should be treated with intravenous thrombolysis [13•].

Prevention of stroke recurrence

The risk of stroke in patients with CeAD who present without signs of cerebral ischemia is increased in the first 5 weeks after dissection, with the majority of infarcts occurring within the first 2 weeks [14]. Recent analysis of 30-day readmission rate of patients with CeAD from 2014 Nationwide Readmission Database showed 4.14% and 1.60% readmission with ischemic stroke in patients with symptomatic carotid dissection and symptomatic vertebral

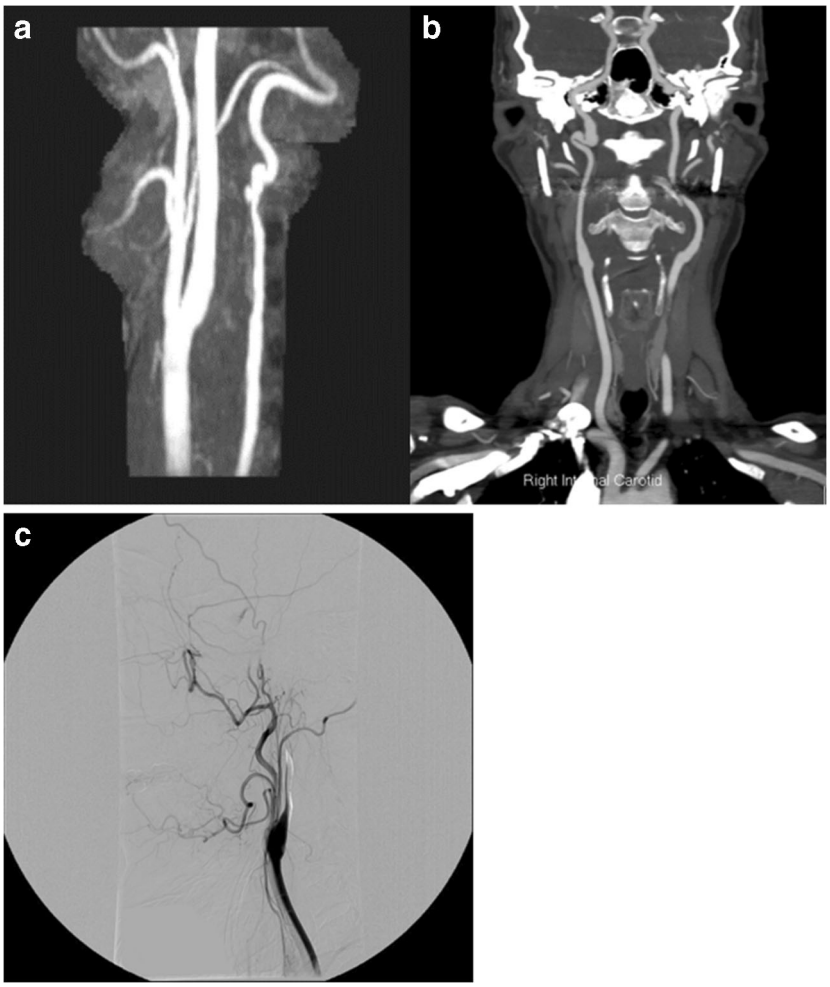

Fig. 2. Examples of common CeAD patterns. **a** Magnetic resonance angiography (MRA) in shows stenosis of the right vertebral with an accompanying pseudoaneurysm. **b** Coronal view of a computed tomography angiography (CTA) depicting pseudoaneurysm formation in the right internal carotid artery (ICA) just before entering the skull through the foramen lacerum. **c** Digital subtraction angiography showing tapering of the left ICA before completely occluding distally. Figure 2 is generated from depersonalized images from patients at University Hospitals Cleveland Medical Center.

dissection, respectively. Fifty percent of patients readmitted with ischemic stroke presented in first week [15]. Prior attempts were made to distinguish patients with CeAD at a greater risk of embolic events with transcranial Doppler (TCDs): a subgroup of patients with of microembolic signals (MES) and abnormal measure of vasoreactivity based on breath holding index (BHI) had increased rate of ischemic events. MES can be detected in up to 60% of the patients presenting with strokes secondary to dissections [2, 16]. The presence of MES was associated with increased frequency of small cortical infarctions in patients presenting with CeAD. Anticoagulation with intravenous heparin eliminated MES in 70% of patients within the first 24 h, demonstrated by consecutive daily TCDs in 3 days after presentation [17]. The role of MES in recurrence of stroke and the question whether the number of MES will be influenced by

antiplatelet versus anticoagulation treatment will be further investigated in the TREAT-CAD randomized study, which is currently recruiting patients [18].

Role of antiplatelet and anticoagulant medications

The arterial wall injury site with an adjacent mural hematoma and/or pseudoaneurysm becomes a potential thromboembolic source. Various strategies have been tried in the past for stroke prevention. In 2011, collaborative US guidelines on extracranial and vertebral artery disease management had no clear recommendations on the treatment of symptomatic CeAD; however, they delineated the traditional conservative approach: anticoagulation is used until symptom resolution, followed by an antiplatelet agent afterwards. The CeAD is considered symptomatic if any related symptom is present [19]. Numerous meta-analyses based on case series and observation studies were published in recent years showing no significant difference in stroke recurrence, symptomatic intracerebral hemorrhages, other major bleeds, and mortality with anticoagulation versus antiplatelet therapy. There were no randomized trials until recently when the Cervical Artery Dissection in Stroke Study (CADISS) phase II feasibility data was published in 2015, and no controlled trials have been ever conducted.

The CADISS trial was designed to answer the key question: antiplatelet or anticoagulant? The feasibility study recruited 250 patients with CeAD and randomized patients into anticoagulation and antiplatelet groups. The antiplatelet group included aspirin alone (22%), dipyridamole alone (1%), clopidogrel alone (33%), or combinations: aspirin and clopidogrel (28%) or aspirin and dipyridamole (16%), with the particular agent per physician's discretion. Patients in the anticoagulation group were treated with intravenous heparin or low molecular heparin, followed by warfarin to maintain an international normalized ratio (INR) of between 2 and 3. At 3-month follow-up, three strokes recurred in the antiplatelet group versus one associated with anticoagulation, with one major subarachnoid hemorrhage (SAH) in the anticoagulation group and no deaths. There was no significant difference in stroke recurrence, major bleeding, or mortality [20•] between the two treatment groups. However, the study was limited and will be discussed below in detail. A very important limitation was the time of randomization; thus, guidance is lacking in the treatment of patients in the hyperacute period. Current 2018 AHA/ASA guidelines, based on CADISS results and prior observational studies, suggest that 3–6-month therapy with either antiplatelet or anticoagulant therapy may be reasonable (Class of Recommendation (COR) IIB, Level of Evidence (LOE) B-R). Further trials on the choice of antithrombotic therapy for prevention of cerebral ischemia in CeAD are not feasible due to the low incidence of recurrent ischemic events.

The CADISS trial results are generally in agreement with multiple preceding meta-analyses and observational studies. However, there are still questions that were left unanswered. Randomization time was 3.9 days in the antiplatelet group and 3.4 in the anticoagulation group. Therefore, it remains unclear which treatment is more effective in the hyperacute period. The incidence of stroke in the study was lower than in previously reported observational studies, and therefore, a phase III trial requiring about 10,000 participants does not seem

feasible. The low incidence of primary outcomes could be explained in part by the possible non-inclusion of non-consentable patients presenting with severe strokes. This is especially concerning because all strokes occurred in patients who initially presented with cerebral ischemia. Moreover, the diagnosis of dissection was not centrally confirmed in about 20% of the patients due to technical difficulties or the wrong diagnosis. No direct oral anticoagulants were used in the study, and their role in CeAD patients has yet to be investigated. The antiplatelet regimens used in the study were very heterogeneous, which could possibly obscure the benefit of dual antiplatelet therapy.

A single-center retrospective study of patients with CeAD (52 dissected arteries in 44 patients) treated with dual antiplatelet therapy (aspirin 81 mg and clopidogrel 75 mg, after initial loading dose of 300 mg) showed 0% stroke recurrence. The researches in this study proposed a new classification (Borgess classification) of CeAD based on radiologic characteristics in an attempt to separate patients by pathophysiologic mechanism. Patients with intact intima were labeled "type I," and those associated with intimal injury were labeled "type II" dissections. Follow-up imaging at 6 months showed that 41% of dissected vessels were healed, with 75% of Borgess type I dissections versus 15% type II dissections. No recurrent strokes, symptomatic intracranial or major extracranial hemorrhages, or deaths were reported [10]. The CADISS trial design does not allow to draw clear answer on the efficacy of various antiplatelet regimens and is probably obscuring the potential beneficial effect of dual antiplatelet therapy [20•].

Clinicians should be aware of certain situations where patients with CeAD may present with a mobile intraluminal clot or may have early recurrence of ischemic events. More aggressive treatment may be warranted if no significant contraindications exist. Based on available data, the safety of anticoagulation may be comparable to antiplatelet drugs, with only 1 of 124 patients developing significant SAH in the CADISS population and 5 of 697 patients developing symptomatic ICH in large meta-analysis [21]. However, a dissecting hematoma is more likely to expand with anticoagulation and may, in theory, worsen arterial stenosis without clear benefit in recanalization rate between antiplatelet and anticoagulant treatment groups [22]. The duration of acute anticoagulation is unclear; however, it can probably be concluded based on MES or healing of the injured artery on follow-up imaging as deemed by the clinician on a case-by-case basis.

Direct oral anticoagulation agents

Very limited information on the treatment of CeAD with direct oral anticoagulant agents (DOAC) is available. A small retrospective study in Helsinki compared patients who presented with CeAD-related stroke: six patients were treated with non-vitamin K anticoagulants (three with dabigatran and three with rivaroxaban) and 34 with vitamin K inhibitors (VKA). Complete recanalization within 6 months on a DOAC occurred in 5 of 6 patients and in 34 of 62 patients on a VKA. One patient had an intracerebral hemorrhage (ICH). One patient died in the VKA group while no one died in the DOAC group. No information is available on the recurrence of cerebral ischemia, but all patients in the DOAC group had a modified Rankin scale (mRS) ≤ 1 at 6 months (77%

in VKA group). Due to the small sample size, statistical significance was not reached [23].

In single academic center retrospective study, patients with CeAD ($n = 149$) were treated with a DOAC (dabigatran, rivaroxaban, or apixaban) (26.2%), a traditional anticoagulant (AC) such as VKA or low-molecular weight heparin (LMWH) (47%), and antiplatelet agents (aspirin, clopidogrel, or aspirin/dipyridamole combination) (26.8%). Patients treated with a DOAC or an AC tended to have more severe stenosis than those on antiplatelet agents (61.8%, 60.0% and 22.5% respectively). Follow-up imaging obtained (median time 5 months) in 83.9% of patients showed two recurrent strokes in the DOAC group and one in the AC and antiplatelet groups each. No major hemorrhagic events occurred in the DOAC group but hemorrhage occurred in 11.4% in AC group and in 2.5% in the antiplatelet group. Three patients in the DOAC group and none in the AC or antiplatelet groups had worsened degree of stenosis [24].

In a small case series, four patients with CeAD (three patients with severe stenosis and one with occlusion) were treated with a DOAC (rivaroxaban) for 6 months without recurrent stroke or major bleeding; however, no change in the degree of stenosis on CTA was reported in three patients with improvement in one patient [25].

Endovascular stenting

Another potential treatment option for patients with CeAD is endovascular treatment, typically with the deployment of a stent. There have yet to be any randomized clinical trials of endovascular treatment for patients with CeAD [26]; thusly, the indications, efficacy, safety, and timing of when to perform endovascular treatment for CeAD is still largely unknown. Current AHA guidelines recommend that "endovascular therapy may be considered for patients with CD who experience definite recurrent cerebral ischemic events while on appropriate antithrombotic therapy" [5].

Our literature search parameters yielded several case reports and case reviews of attempted endovascular stenting in patients with CeAD, most of which reported successful stent deployment and restoration of flow through the diseased artery [27–34]. The similarities tend to end there, as some cases had endovascular stenting before any medical treatment while other cases had endovascular stenting after the deemed failure of medical treatment. Many of these cases introduced novel techniques such as the addition of intravascular ultrasound to help identify calcified plaques, something that other imaging modalities may not be able to consistently identify and characterize [29]. Plaque characterization can be helpful for prognosis and treatment decisions as there is an association with lower stent success rates and high in-stent stenosis rates with stent placement in calcified lesions [29, 35]. There were also novel techniques that were used to overcome difficult anatomy. In the case of a dissected, highly tortuous cervical internal carotid artery (ICA), a microguidewire was used to straighten the ICA before deploying the stent [32].

In some cases, there can be tandem lesions with CeAD and intracranial large vessel occlusions. Simonetti et al. looked at patients who developed iatrogenic arterial dissections in the setting of endovascular treatment of an acute ischemic stroke. They looked at 866 patients that underwent endovascular treatment for

acute ischemic stroke and had 18 patients that suffered an iatrogenic dissection, 15 of which were extracranial—14 involving the internal carotid artery and one involving the vertebral artery. Of these patients, eight were treated with endovascular stenting. Patients with iatrogenic dissections did not have clinical or radiographic evidence of new or additional neurological lesions or deficits within 24 h of their procedure, regardless of whether or not they received stenting [33].

These cases also raise the question of the potential risks associated with endovascular stenting. While the data for stenting complications is limited in the setting of stent placement in CeAD, data from carotid artery and vertebral artery stenting for other indications have shown that complications can include events such as in-stent restenosis and stent fracture [36, 37]. Rates of in-stent restenosis can be decreased with the use of antiplatelet agents, and the question of when to initiate antiplatelet therapy and for how long may further complicate medical treatment for patients with other comorbidities. As with other similar endovascular procedures, risks of ischemic stroke, arterial dissection, hemorrhage, myocardial infarction, and death must also be considered [36].

While the above cases have largely been successful with restoration of blood flow with stenting in CeAD, date regarding long-term prognosis is scarce at best. Until more data is provided, endovascular stenting may best be reserved for cases that have otherwise failed maximal medical therapy, echoing the recommendations set by the AHA/ASA.

Multiple CeAD and early recurrent CeAD

Another type of clinical presentation of CeAD is early recurrence and multiple dissections, both of which possibly share a similar pathophysiologic mechanism. From the large multicenter registry Cervical Artery Dissection and Ischemic Stroke Patients (CADISP) data of 1958 patients, 17.4% presented with multiple dissections, of whom 2% had early recurrent dissections. Of patients presented with single dissections, 1.5% had early dissection recurrence. All patients had similar characteristics and risk factors: recent infections, family history of strokes suggestive of genetic risk factors, presence of prior head and neck surgery, and surgical manipulation. Multiple CeAD and early recurrent dissections were associated with a tripled rate of stroke incidence, increased rate of transient ischemic attack (TIA), and ICH. Recurrent and multiple dissections suggest the presence of an underlying vasculopathy [38, 39]. However, no difference was observed in functional outcome in 3-month follow-up. No data is available on the best treatment strategy for multiple CeAD, and the approach should probably be individualized based on contraindications and radiological characteristics of dissected vessels.

Conclusion

Antithrombotic therapy remains the mainstay of prevention of symptomatic cerebral ischemia in CeAD. There is no clear evidence to support the use of a specific type of antithrombotic medication, antiplatelet agent, or anticoagulant, based on the radiological appearance and thromboembolism risk stratification. While being non-inferior to anticoagulants in treatment of CeAD, antiplatelet

agents are less expensive, do not require monitoring, and are easy to use. They can be used in certain situations with a large size infarct, signs of hemorrhagic transformation, and other situations where anticoagulants pose a high risk of hemorrhagic complications. Acute anticoagulation may be beneficial in patients with high risk of thrombosis, as suggested by imaging: severe stenotic and occlusive dissections, presence of mobile thrombus in arterial lumen, pseudoaneurysm formation, and positive MES on TCD. Significant knowledge gaps still exist in the management of CeAD. Clinical trials addressing the efficacy and safety of DOAC agents, dual antiplatelet regimens, and endovascular management strategies in the hyperacute period and risk stratification are warranted.

Compliance with Ethical Standards

Conflict of Interest
The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent
This article does not contain any studies with human or animal subjects performed by any of the authors.

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