Review Article

The Clinical Features of Dissection of the Cervical Brain-Supplying Arteries

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Summmary

Background: Dissections of the cervical brain-supplying arteries are a leading cause of ischemic stroke in young adults, with an annual incidence of 2.5–3 / 100 000 for carotid artery dissection and 1–1.5 / 100 000 for vertebral artery dissection. It can be assumed that many cases go unreported. We present the clinical features here to help physicians diagnose this disease entity as rapidly as possible.

Methods: This review is based on pertinent publications retrieved by a selective search in PubMed.

Results: Spontaneous dissection of the internal carotid or vertebral artery is characterized by a hematoma in the vessel wall. It often arises in connection with minor injuries; underlying weakness of the arterial wall (possibly only temporary) may be a predisposing factor. Acute unilateral pain is the main presenting symptom. In internal carotid dissection, the site of the pain is temporal in 46% of cases, and frontal in 19%; in vertebral artery dissection, it is nuchal and occipital in 80%. Pain and local findings, such as Horner syndrome, are generally present from the beginning, while stroke may arise only after a latency of hours to days. If the diagnosis is made early with MRI, CT, or ultrasound, and anticoagulation or antiplatelet drugs can help prevent a stroke, yet none of these methods can detect all cases. Recurrent dissection is rare, except in patients with connective tissue diseases such as Ehlers–Danlos syndrome or fibromuscular dysplasia. Spontaneous dissection of the great vessels of the neck must be differentiated from aortic dissection spreading to the supra-aortic vessels and from traumatic dissection due to blunt or penetrating vascular trauma.

Conclusion: Dissection of the cervical brain-supplying vessels is not always revealed by the imaging methods that are used to detect it. Stroke prevention thus depends on the physician's being aware of the symptoms and signs of this disease entity, so that early diagnosis can be followed by appropriate treatment.

D issection of the neck arteries is characterized by a hematoma in the wall of a great vessel, usually the internal carotid artery or the vertebral artery. It is a leading cause of ischemic stroke in young adults (1). The mean age at the time of dissection is 44 years (e1), and men are affected slightly more commonly than women (53–57% of cases are in men) (e2). The annual incidence of dissection is reportedly 2.5–3 per 100 000 persons for the internal carotid artery, and 1–1.5 per 100 000 persons for the vertebral artery (2). These are probably underestimates, however, because dissections may go undetected if the patient does not go on to have a stroke (3, 4). Stroke usually occurs only after a delay, rather than being an early symptom of dissection (5); thus, good knowledge of this clinical entity and its causes and manifestations might increase the likelihood of early detection and treatment.

Methods

In January 2022, we carried out a selective literature search in PubMed for original articles and reviews, using the terms "dissection" in conjunction with "carotid,"vertebral," or "cervical artery."

Pathology

Spontaneous dissection of the internal carotid or vertebral artery (including, by definition, dissection after minor trauma) is characterized by a mural hematoma that initially arises without an intimal tear (6). The likely cause is rupture of the vasa vasorum (7). The
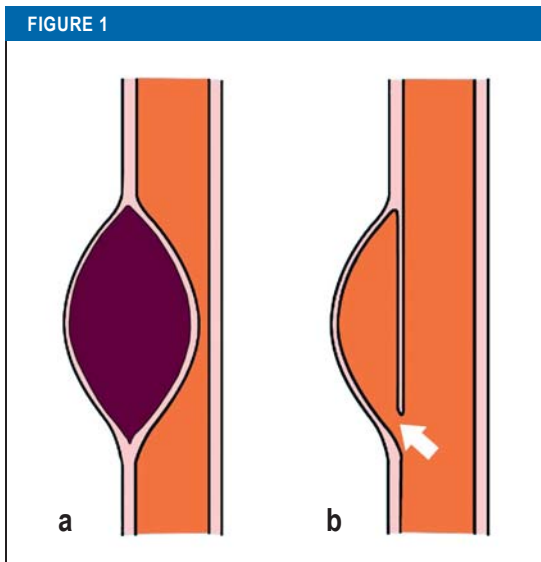
Pathoanatomy of arterial dissection
a) Dissection of the internal carotid artery or vertebral artery with a hematoma in the vessel wall due to rupture of the vasa vasorum. The intramural hematoma can progress toward the lumen with secondary rupture of the intima.
b) Dissection of the aorta with rupture of the intima and formation of two lumina. Modified from (20).

mass effect of the hematoma causes the outer wall of the vessel to bulge, while also constricting the lumen *(Figure 1a)*. The hematoma can spread along the vessel wall in both directions and an also penetrate towards the lumen with secondary tearing of the intima (7). An intimal flap is formed in this way, and there may be short stretches with two lumina (the original lumen and a pseudolumen) (8).

The pathoanatomy of arterial dissection changes over time. The mural hematoma usually regresses, at least in part, in a few weeks or months (9), with associated recanalization of stenoses. In their early stages, however, mural hematomas can expand, especially under treatment with anticoagulant drugs (9).

In this article, there will be no further discussion of two other types of dissection of the cervical arteries which are distinct from spontaneous dissection, with fundamentally different pathoanatomy and clinical manifestations:
- aortic dissection with spread to the supra-aortic vessels. Here we see the findings of aortic dissection extending into the neck arteries, most commonly the common carotid artery *(Figure 1b)*. A long-stretch double lumen is typical, with the pseudolumen either ending rostrally in a blind pouch or re-entering the true lumen (10).
- traumatic dissection after blunt or penetrating vascular injury, which can also be iatrogenic (during attempted jugular venous puncture). This also most commonly involves the common carotid artery. The pathoanatomy is varied; such trauma may cause intimal ruptures and mural hematomas, vascular stenoses and pseudoaneurysms, and AV fistulae (11).

Clinical features

Three varieties of clinical presentation of dissection of the great vessels are illustrated in *Figure 1a:*
- Vessel wall symptoms: the stimulation of nociceptors in the vessel wall causes ipsilateral headache and/or facial pain. In carotid artery dissection, there is temporal headache in 46% of cases and frontal headache in 19% (12); in vertebral artery dissection, there is nuchal headache in 80% of cases (12). Isolated extracranial pain occurs in only ca. 10% of cases (3). The pain is of sudden onset (13), severe from the beginning, and unlike any pain the patient has had before (12).
- Outward bulging of the vessel: the mural hematoma exerts local mass effect, in the internal carotid potentially damaging sympathetic fibers that run along the outer surface of the vessel wall (and thereby causing Horner's syndrome) or compressing the caudal cranial nerves (14). Vertebral artery dissection can damage the cervical nerve roots (15).
- Vessel stenosis: the mural hematoma also pushes inward, compressing the vessel lumen to cause stenosis whose severity depends on the size of the hematoma; stenosis can be very severe, especially in the early stages (16). Intraluminal thrombi can develop at the site of the stenosis and embolize distally, causing stroke (17). This why anticoagulant drugs are used for primary and secondary prevention (18; cf. the "Treatment" *Box*). Less commonly, there can also be ischemia on a hemodynamic basis if there is inadequate collateralization. Another potential symptom of stenosis is unilateral pulse-synchronous tinnitus, which is heard by ca. 25% of patients with internal carotid artery dissection (3). The sound heard by the patient is due to turbulent flow at a site just distal to the stenosis, which is near the inner ear.

The various symptoms of dissection do not arise at the same time. Headache is earliest, arising when the mural hematoma comes about (5, 12); stroke due to arterio-arterial embolism occurs after a latency of hours to days (3), depending on how long it takes for an intraluminal thrombus to form. In patients with internal carotid artery dissection, the mean time to the onset of cerebral symptoms is 8.8 days (median, 96 hours) (5). Thus, there may often be an opportunity to detect and treat dissection early, before cerebral ischemia occurs (3).

A clinically important fact is that dissections occur simultaneously in multiple brain-supplying arteries in approximately 15% of cases. There can be bilateral dissection of the vertebral arteries or of the internal carotid arteries, or simultaneous dissection of a vertebral artery and an internal carotid artery (19).

Typical constellations of findings

Acute temporal headache or other unilateral headache and/or facial pain, together with any of the following manifestations, should arouse the suspicion of a dissection of the internal carotid artery:

- ipsilateral Horner's syndrome. One-third of patients who have (transient or permanent) cerebral ischemia exhibit a characteristic triad of manifestations, namely, headache, Horner's syndrome, and ipsilateral cerebral or ocular transient ischemic attack (TIA) (3). The headache and Horner's syndrome persist after the TIA subsides.
- ipsilateral pulse-synchronous tinnitus (13). Patients often will not report this spontaneously and need to be asked about it.
- ipsilateral caudal cranial nerve dysfunction. There may be a hypoglossal palsy, with deviation of the extended tongue to the paretic side; vagus palsy, with ipsilateral drooping soft palate and dysphagia; or glossopharyngeal palsy, with dysphagia (3).

No frequencies are reported in the literature for most of these findings and constellations of findings.

In vertebral artery dissection, clinical manifestations of outward mass effect are rare. Unilateral nuchal and occipital pain is usually the only symptom, but there may also be dysfunction of the cervical nerve roots (3). The sudden onset (13), immediate severity, and previously unknown quality (12) of the pain distinguish it from cervical spondylogenic pain. Pain arising suddenly in the setting of a rapid, unusual head movement should also arouse suspicion of dissection (3).

Localization of the dissection

The typical sites of dissection are at locations where a vessel passes from a mobile section to a section fixed in bone and is thus vulnerable to sudden mechanical stress. For the internal carotid artery, this is the point where it enters the petrous bone *(Figure 2a)*.

The dissection may extend caudally along the internal carotid artery from the petrous bone to several centimeters above the bifurcation of the common carotid artery (14). In the vertebral artery, the V3 segment above and below the first cervical vertebra (C1) is especially vulnerable *(Figure 2b)*. Dissections also occur before entry into the transverse foramen at C6, between C6 and C2, and at the passage through the dura at the level of the foramen magnum (6). Dissection may propagate intracranially from the V3 segment into the basilar artery (6), and may also extend extracranially for long distances (3, 6). Rarely, other intracranial vessels are involved (e3, 6).

Imaging studies

The diagnosis is made in two steps, of which clinical suspicion is the first step. The second step, definitive diagnosis, requires imaging studies, which are performed to answer specific questions (dissection can easily be missed if studies are performed as a matter of routine

BOX

Treatment

The tendency of dissection to cause arterio-arterial embolism and stroke motivates the use of anticoagulant drugs for primary and secondary prevention (18). The CADISS trial, published in 2015, revealed no significant differences in any of the outcome parameters studied in patients with symptomatic dissection of the neck arteries who were treated with either antiplatelet or anticoagulant drugs (36). The risk of stroke recurrence in the overall trial population was 2% at 3 months, with no difference between the patients in the two arms of the trial. Both types of drugs are apparently effective, as stroke was much less common in both arms of the trial than in previously conducted observational studies, where up to 41% of patients sustained a stroke in the first six months of observation (e11). Nonetheless, aspirin was found to be less effective than anticoagulant drugs in the recently published TREAT-CAD trial (37). Anticoagulation for three months is recommended for patients with dissection in the current North American Stroke Secondary Prevention Guideline (17).

It is suggested in a recent review on stroke prevention in patients with neck artery dissection that the optimal drug therapy might be patient-specific (24): anticoagulant drugs would be indicated for patients with dissection causing high-grade stenosis, while antiplatelet drugs would be indicated for those with low-grade stenosis. In patients with dissection causing high-grade stenosis initially, potential recanalization could be checked by ultrasonography every three months, so that anticoagulation could be switched to antiplatelet therapy if possible. Drug therapy is usually given for 1–2 years, although there are no evidence-based recommendations for its duration. Dissection in a patient with fibromuscular dysplasia or vascular Ehlers-Danlos syndrome is more likely to recur; patients of these types who suffer a dissection with cerebral ischemia should be given permanent secondary prophylactic treatment (e2, 30).

A special case is that of a tight stenosis with poor collateral arterial supply, causing a recurrent neurologic deficit on a hemodynamic basis, with similar clinical manifestations accompanying every drop in blood pressure. Endovascular treatment may be indicated (17).

rather than in targeted fashion). An international panel of experts has stated that any of these findings suffices for the diagnosis of a dissection of a great vessel of the neck: mural hematoma, pseudoaneurysm, elongated tapering stenosis, intimal flap, double lumen, and occlusion more than 2 cm above the carotid bifurcation with evidence of a pseudoaneurysm (19). The studies that can detect these findings include duplex sonography, computed tomography (CT) and CT angiography (CTA), magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA), and digital subtraction angiography (DSA). All of these studies can yield false-negative findings (3).

Ultrasonography

Duplex ultrasonography is particularly well suited for detecting dissections of the vertebral artery because the vessel can be seen all the way from its origin to its junction with the contralateral vertebral artery to form the basilar artery (20), with brief discontinuities because of bony structures *(Figure 3a,b)*. Imaging just above and below the first cervical vertebra is
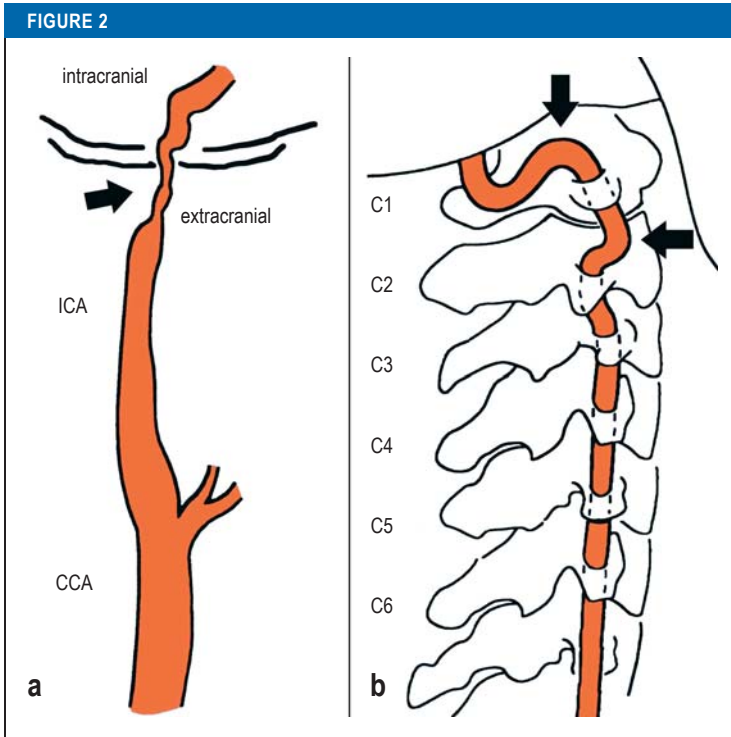
Typical dissection sites at transitions from a mobile vascular segment to a segment fixed in bone.
a) Internal carotid artery (ICA): Dissection at the point of entry into the petrous bone.
b) Vertebral artery: The V3 segment above and below the first cervical vertebra are especially vulnerable (modified from [20]).
CCA, common carotid artery; ICA, internal carotid artery.

particularly important (20). The sensitivity of duplex sonography is reported to be 92% (21), although less experienced examiners are unlikely to match this result.

The internal carotid artery cannot be directly seen with ultrasound just below the skull base, and thus ultrasound may fail to detect a short, low-grade stenosis associated with an internal carotid artery dissection. Its sensitivity for internal carotid artery dissection causing only local symptoms is reported to be 69% (22). High-grade stenoses, however, can be detected by hemodynamic criteria, e.g., a bilateral comparison of current pulse curves in the common carotid artery, or the demonstration of collateral flow (23). On the other hand, duplex ultrasonography is 96% sensitive and 94% specific for dissections of the internal carotid artery that have led to ischemia, with catheter angiography as the reference standard (23).

Magnetic resonance imaging / magnetic resonance angiography

The standard study is native (i.e., non-contrast) MRI with fat-suppressed T1-weighted sequences in the transverse and coronal planes *(Figure 3c)*. 3D sequences with additionally suppression of the proton signal of blood flow ("black-blood" sequences) are particularly recommended. A characteristic finding in dissection is eccentric signal enhancement in the vessel wall in the fat-suppressed T1 sequences, representing the mural hematoma. This signal enhancement, due to methemoglobin in the hematoma, is often absent in the first 72 hours after the onset of dissection (24). As the signal intensity of the mural hematoma changes over time, multisequence MRI can help date the dissection (25). MRA also reveals narrowing of the lumen resulting from the mass effect of the hematoma, ranging to complete vessel occlusion *(Figure 3d)*. Contrast-enhanced MRA, which is always performed after the native sequences, yields high-quality images of the entire extracranial and intracranial arterial vasculature, even in the vicinity of metal artifacts (26).

Computed tomography / computed tomographic angiography

CT with CTA and 3D reconstruction enables the evaluation of dissection with visualization of the vessel wall and lumen. Its advantages include high spatial resolution, rapid performance, and wide availability. A disadvantage is the radiation exposure, which is relevant as patients with dissections of the great vessels of the neck are generally young. Common CT findings in dissection are irregular luminal stenosis and evidence of intramural hematoma: the vessel wall appears thickened, with a crescent-shaped hyperdensity. The finer resolution of CT/CTA makes it more sensitive for the detection of intimal flaps and intraluminal thrombi adjacent to the vessel wall (27). Difficulties arise at the sites of entry of vessels into the skull base. Findings of wall thickening are harder to classify by CT/CTA than by MRI. A mural hematoma may be difficult to tell apart from vasculitis or atherosclerosis by CT alone and can be more reliably distinguished by MRI (28,24).

Digital subtraction angiography

DSA was the standard method of diagnosing dissection for many years, as it enables optimal visualization of the vessel lumen and its pathologic changes. Nonetheless, newer methods of imaging the arterial wall have revealed that DSA yields false negative findings in up to 17% of cases (3). The applicability of DSA is further restricted by its risks as an invasive method and by the wide availability of non-invasive imaging (3). It is clearly indicated as an adjunct to emergency thrombectomy in cases where dissection has led to embolism into the proximal portion of a cerebral artery or arteries.

Thus, all four types of diagnostic study can be used to detect dissections of the great arteries of the neck, but none of them is sensitive enough to rule out a dissection with certainty. The American Heart Association and the American Stroke Association, in their joint statement on the diagnosis of cervical artery dissections, explicitly decline to endorse the prior recommendation of the European Federation of Neurological Societies, which considers MRI/MRA the imaging modality of first choice in dissection (29); rather, they do not consider any of the imaging modalities to be a modality of first choice (3) but state that they should be used in a complementary manner. In
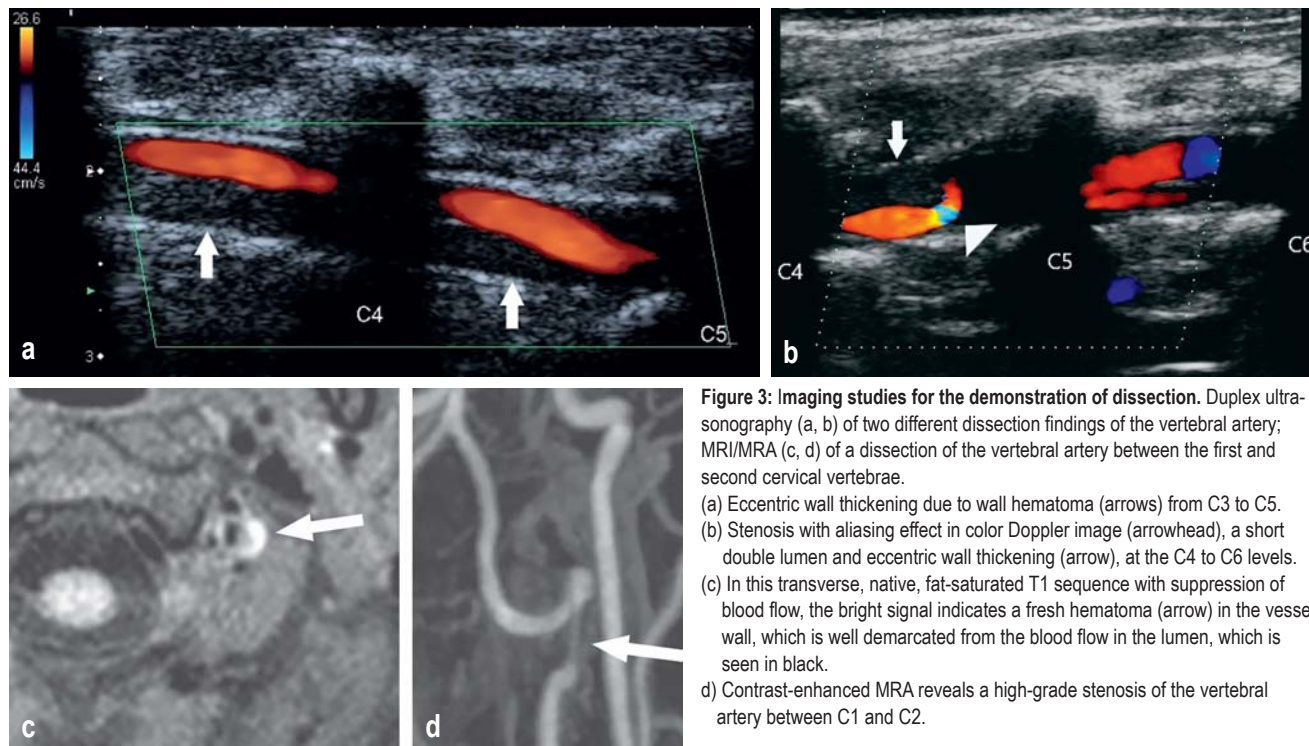
Figure 3: Imaging studies for the demonstration of dissection. Duplex ultrasonography (a, b) of two different dissection findings of the vertebral artery; MRI/MRA (c, d) of a dissection of the vertebral artery between the first and second cervical vertebrae.

(a) Eccentric wall thickening due to wall hematoma (arrows) from C3 to C5.

(b) Stenosis with aliasing effect in color Doppler image (arrowhead), a short double lumen and eccentric wall thickening (arrow), at the C4 to C6 levels.

(c) In this transverse, native, fat-saturated T1 sequence with suppression of blood flow, the bright signal indicates a fresh hematoma (arrow) in the vessel wall, which is well demarcated from the blood flow in the lumen, which is seen in black.

d) Contrast-enhanced MRA reveals a high-grade stenosis of the vertebral artery between C1 and C2.

the German guideline, it is even recommended that a combination of two imaging studies should always be used for diagnosis: ultrasound with either MRI/MRA or CT/CTA (30). Some important differential diagnoses of dissection are listed in the *Table.*

In the vascular diagnostic evaluation, evidence of fibromuscular dysplasia in the internal carotid or vertebral arteries should also be sought (31), as this connective tissue disease increases the risk of recurrence.

Etiology

Dissections often occur in the setting of a rapid, large-amplitude movement of the cervical spine, such as during a tennis serve, golf swing, or sudden neck extension in a volleyball game (6). Many persons with dissection associated with such movements have made similar movements many times before without any adverse consequences; it is thought that, in the symptomatic event, several vessels may be affected by the dissection simultaneously (19). A current pathogenetic hypothesis is of a predisposing arterial wall weakness that may be only temporary (14). For example, a viral infection involving the arteries might transiently make them more sensitive to a mechanical stress (32). Two older studies revealed an association between recent infections and dissection (33, e4).

Connective tissue disease may also be a predisposing factor (6, 34). In dissection registries, fibromuscular dysplasia is found in 5.6% of cases (19). 1.9% of patients with Ehlers-Danlos syndrome, suffer a neck artery dissection (e5).

There have been multiple reports of dissection after supposedly therapeutic manual manipulation of the neck (e6), yet the causal relationship has not been confirmed to date, and only very few patients treated in this way suffer a dissection (3). Perhaps the rare cases of dissection after manual therapy are explicable as the product of a temporary vulnerability to mechanical stress. The hypothesis that, in rare cases, previously unrecognized dissection can be worsened by manual manipulation (e7) was recently confirmed in a well-documented case (4).

Many other risk factors for dissection have been reported, including arterial hypertension, migraine, vascular loops, and kinking (14,3). The significance of these common conditions and anatomical findings in the pathophysiology of dissection has not yet been definitively established. Many uncertainties remain concerning the etiology and pathogenesis of dissection (14).

Course and prognosis

In most cases, the mural hematoma and the resulting stenosis regress over time. Even tightly stenotic or occluded arteries become completely or partially recanalized within six months in 60–67% of cases, and in the following six months in an additional 6.8% (e8).

The risk of recurrence of dissection is very low after the post-acute period (30). It is 3–6% after the fourth month (e9). A long-term recurrence rate of 0–1% per year has been reported (e1, e10).

Patients with dissection should avoid strenuous physical activity for a few months. They may resume

TABLE

Differential diagnosis

Diagnose	Symptoms	Vascular findings	Frequency	Ref.
Giant-cell arteritis	headache, usually bilateral; sudden onset is very unusual	vessel wall thickening similar to dissection, but typically concentric	relatively common; incidence 3.5 per 100 000 in persons over age 50	(20; e12)
Idiopathic carotidynia	acute unilateral neck pain, very rarely radiating to the head; spontaneous resolution in 3–4 weeks	eccentric wall thickening with outward vessel bulging and (usually minor) lumen narrowing; always located near the carotid bifurcation; contrast enhancement on MRI	common pathology, frequent cause of misdiagnosis; initial description of ultrasound finding in 2004	(38)
Fenestration of a cervical or cerebral artery	asymptomatic normal variant	short double lumen; particularly often seen in basilar artery, rarely in extracranial portion of vertebral artery	rare finding; no incidence data available	(39)
Carotid web (intimal form of fibromuscular dysplasia)	asymptomatic; may cause cerebral ischemia by ipsilateral arterio-arterial embolism	double lumen or multiple lumina; always near carotid bifurcation; no wall thickening bulging outward	rare finding; no incidence data available	(40)
Spontaneous vasospasm of the internal carotid artery	acute uni-or bilateral headache and neurologic deficits, as in TIA; recurrent episodes of same deficit	stenosis of the distal extracranial portion of the internal carotid artery (as in dissection), regressing in a few hours or days	rare finding; initial description of ultrasound finding in 1998; no incidence data available	(e13)

MRI, magnetic resonance imaging; TIA, transient ischemic attack

endurance sports in three months at the earliest, and other sports in 6–12 months. Jerky head movements should be avoided. An international expert panel has issued more detailed recommendations in a consensus paper entitled "Cervical Artery Dissection and Sports" (35).

Overview

Dissection can cause stroke even in persons without any known vascular disease. Because stroke is generally not the initial manifestation of dissection, but occurs only after a delay, knowledge of this clinical entity and its manifestations can help prevent stroke.

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► **Supplementary material:**

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Questions on the article in issue 35–36/2022:

The Clinical Features of Dissection of the Cervical Brain-Supplying Arteries

cme plus +

The submission deadline is 4 September 2023. Only one answer is possible per question.

Please select the answer that is most appropriate.

Question 1
What is a typical symptom of vertebral artery dissection?
a) restriction of range of movement of the neck
b) unilateral nuchal and occipital pain
c) ipsilateral Horner's syndrome
d) contralateral sensorineural deafness
e) contralateral impairment of visual acuity

Question 2
What symptom is highly compatible with internal carotid artery dissection?
a) restriction of range of movement of the neck
b) biilateral nuchal and occipital pain
c) ipsilateral Horner's syndrome
d) contralateral sensorineural deafness
e) contralateral impairment of visual acuity

Question 3
What is usually the first symptom of dissection?
a) restriction of range of movement
b) headache
c) cervical nerve root dysfunction
d) ocular transient ischemic attack
e) cerebral transient ischemic attack

Question 4
Which of the following is *not* a typical pathological feature of spontaneous dissection of the internal carotid or vertebral artery?
a) mural hematoma
b) primary intimal tear
c) inward narrowing of the lumen
d) outward bulging of the vessel
e) rupture of the vasa vasorum

Question 5
What imaging study yields the best visualization of the vascular lumen, but nevertheless has false negative findings in more than 10% of cases?
a) magnetic resonance imaging
b) magnetic resonance angiography
c) duplex sonography
d) computed tomography
e) digital subtraction angiography

Question 6
What is the most common site of vertebral artery dissection?
a) directly above and below C1
b) origin of the vertebral artery from the subclavian artery
c) directly above and below C3
d) directly above and below C4
e) junction with the opposite vertebral artery to form the basilar artery

Question 7
A dissection can be complicated by arterio-arterial embolism leading to stroke. When does this complication most commonly occur?
a) a few seconds after the dissection
b) 15–30 minutes after the dissection
c) hours to days after the dissection
d) 3–6 weeks after the dissection
e) 3–6 months after the dissection

Question 8
What measure is recommended in the North American guideline for the secondary prevention of arterio-arterial embolism after a dissection?
a) anticoagulation for three months
b) minimally invasive stent implantation in the vicinity of the dissection
c) 1-2 days of monitoring in the hospital
d) 1-2 weeks of monitoring in the hospital
e) 1-2 days of intravenous high-dose steroid therapy

Question 9
What is the long-term annual recurrence rate of dissection after the post-acute phase?
a) 0–1%
b) 5%
c) 10%
d) 15%
e) 20%

Question 10
What recommendation about exercise should be given to patients with a dissection?
a) do not recommence endurance sports for at least three months
b) avoid cervical spine movement for at least a year
c) avoid vigorous exercise for at least a year
d) avoid sports that can cause dissection (e.g., golf) for life
e) do not climb stairs for at least three months