Chapter 111

Carotid or Vertebral Artery Pain

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HEADACHE IN CERVICAL ARTERIAL DISSECTION

Definitions

International Headache Society (IHS) code and diagnosis: 6.5.1 Carotid or vertebral dissection

World Health Organization (WHO) code and diagnosis: G44.81 Headache associated with other vascular disorders

Short description: Dissection of the cervical cerebral arteries is a relatively uncommon disorder in which blood enters into the wall of the artery, presumably through a tear on the endothelial surface. It is increasingly recognized as an important cause of stroke, especially in young persons (1,6,14,36,37,43), and accounts for up to 20% of ischemic strokes in patients under the age of 40 (43). The internal carotid artery is affected more often than the vertebral artery, and the location of the dissection is more often in the extracranial than in the intracranial segments (1,14,37,36,43).

Other terms: Dissecting aneurysm

EPIDEMIOLOGY

The annual incidence of spontaneous carotid artery dissection ranges from 2.5 to 3 per 100,000 (43); between 0.5 and 2.5 cases of vertebral dissection per year are reported from large referral-based hospitals (1,14,36, 37,43). The overall frequency of head pain is extremely high, ranging from 60 to 95% in carotid artery dissections (1,4,6,14–16,34,35,37,43,47) and around 70% in vertebral artery dissections (15,34–36,43,47). Pain is the most frequent inaugural symptom of both carotid and vertebral dissections (1,3,4,6,7,9,14–17,20,27,30, 34–37,42,43,47,48,52), potentially leading to recognition of the condition before the occurrence of ischemic signs.

GENETICS

Patients with a spontaneous dissection of the carotid or vertebral artery are thought to have an underlying structural defect of the arterial wall, although the exact type of arteriopathy remains elusive in most cases (19,21–23,43,53). Ehlers-Danlos syndrome type IV, Marfan syndrome, autosomal dominant polycystic kidney disease, and osteogenesis imperfecta type I have been identified in 1 to 5% of patients with spontaneous dissections of the carotid or vertebral artery (21,43). In addition, approximately 5% of patients with a spontaneous dissection of the carotid or vertebral artery have at least one family member who has had a spontaneous dissection of the aorta or its main branches, including the carotid and vertebral arteries. In some of these families, there is also a history of multiple cutaneous lentigines or a congenitally bicuspid aortic valve (21,23,43). The arterial media of the aorta and its branches, melanocytes, and the aortic valvular cusps are all derived from neural-crest cells, suggesting that the underlying defect in these families is an abnormality of neural-crest cells. Angiographic changes of fibromuscular dysplasia are found in about 15% of patients with a spontaneous dissection of cervical arteries, and cystic medial necrosis is a common finding at postmortem examination (21,43). In addition, indirect evidence of a generalized arteriopathy is suggested by the association of such spontaneous dissections with intracranial aneurysms, a widened aortic root, arterial redundancies (e.g., coils, kinks, and loops), and increased arterial distensibility (21,22,43,53). Finally, ultrastructural abnormalities of dermal connective tissue components have been detected in up to two thirds of patients with a spontaneous dissection of the carotid or vertebral artery (23).

ANATOMY AND PATHOLOGY

Dissections of the carotid and vertebral arteries usually arise from an intimal tear. The tear allows blood under

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arterial pressure to enter the wall of the artery and form an intramural hematoma, the so-called false lumen. The intramural hematoma is located within the layers of the tunica media, but it may be eccentric, either toward the intima or toward the adventitia. A subintimal dissection tends to result in stenosis of the arterial lumen, whereas a subadventitial dissection may cause aneurysmal dilation of the artery (43).

PATHOPHYSIOLOGY

The cause of cervical cerebral arterial dissection remains largely unknown. It is often classified as spontaneous or traumatic. A history of a minor precipitating event is frequently elicited in patients with a spontaneous dissection of the carotid or vertebral artery. Neck movements, particularly when they are sudden, may injure the artery as a result of mechanical stretching. Chiropractic manipulation of the neck has been associated with carotid and vertebral artery dissection (43). A recent history of a respiratory tract infection is also a risk factor for spontaneous dissections of the carotid or vertebral artery (19). Two case-control studies suggested migraine as a risk factor for dissection (11,51).

The pain of cervical arterial dissections is a referred pain to the face, head, and neck originating from the dissected artery. In carotid artery dissection, the pain is referred to ipsilateral cephalic and facial regions, the location of which follows the patterns outlined during electric stimulation of the carotid bifurcation or during balloon inflation in the distal carotid artery (4,16,47). In vertebral artery dissection, the location of pain at the posterior neck and also to anterior head regions is likely to be explained on the basis of innervation of these arteries by the upper cervical roots and the convergence of those with descending trigeminal impulses (3,21,30). The pain mechanisms remain largely undetermined. Proposed potential mechanisms include mechanical stimulation of the pain-sensitive receptors in the vessel wall as a result of dilation or distention of the artery and ischemia of the perivascular pain-sensitive nerve fibers resulting from direct mechanical injury and occlusion of the vasa nervorum. The variability of the observed head pain patterns, including whether it occurs, the various localizations, the different degrees of severity, and the different pain qualities, are not explained adequately by any of the proposed mechanisms (3,4,7,9, 15–17,20,21,27,30,42,43,47,48,52).

CLINICAL FEATURES

The IHS diagnostic criteria (Revised International Classification of Headache Disorders [ICHD-II]) for headache in cervical arterial dissection are as follows:

A. Any new headache or facial pain of acute onset with or without neurologic symptoms or signs.
B. Dissection demonstrated by appropriate vascular and neuroimaging investigations.
C. Headache occurs in close temporal relation to and on the same side as dissection.
D. Disappearance of pain within 1 month.

Pain Characteristics

Frequency

Headache with or without cervical pain can be the only manifestation of cervical artery dissection (1,3–7,9,14–17,20,27,30,34–37,42,43,47,48,52). It is by far the most frequent symptom (55 to 100% of cases), and it is also the most frequent inaugural symptom (33 to 86% of cases). Isolated orbital or facial pain is reported by 10% of patients (4,15,16,43,47). Similarly, 69% of patients with vertebral artery dissection report headache, and 46% report neck pain, either isolated or in conjunction with headache (15,43,47).

Timing in Relation to Other Neurologic Manifestations

Headache is often (45%) the initial symptom of carotid dissection, with other manifestations appearing with a delay ranging from 1 hour to 90 days (mean, 9 days) (4–6,47). Rarely, headache develops after the appearance of other manifestations, with a delay ranging from 1 to 96 hours. In the remaining cases, headache appears simultaneously with other clinical symptoms and signs, such as those of cerebral ischemia (4,5,47).

In almost two thirds of patients with vertebral dissection (61%), headache occurs simultaneously with the signs of vertebrobasilar ischemia or cranial nerve dysfunction (4,6,15,16,42,43,47), and in one third the head pain may precede those by a variable interval ranging from 1 hour to 14 days (5,47).

Mode of Onset

In carotid dissection, the onset of pain is occasionally sudden, severe, and with thunderclap qualities raising suspicion of subarachnoid hemorrhage (3,4,6,7,9,15–17,20,27,30,42,43,47,48,52). However, the most frequently reported pattern is that of a gradual-onset pain, mentioned by 85% of patients (47). Similarly, many patients with vertebral dissection (72%) report head pain of gradual onset, and only 22% report a sudden-onset headache (47). Occasionally, a gradual onset of pain is followed by sudden escalation (47).

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Location

The headache of carotid dissection is almost always (91%) ipsilateral to the affected side; generalized or bifrontal headache is uncommon, but it can occur even if the dissection is unilateral (3,4,6,7,9,15–17,20,27,30,42,43,47, 48,52). It is more often localized than diffuse and has a predilection for the frontal and temporal areas (3,4,6,7,9, 15–17,20,27,30,42,43,47,48,52), although any part of the head may be affected, either in isolation or in combination. Facial pain, including ear pain, and orbital pain may accompany the dissection in 48% and 61% of cases, respectively (47). Facial or orbital pain may occur in the absence of headache. Neck pain at the lateral cervical region is reported by 26% of patients at the time of dissection (4).

Almost half of patients with vertebral artery dissection (44%) report a unilateral occipital or parietooccipital pain (43,47). Less often, a bilateral occipital, unilateral frontal, or generalized headache is reported. When the pain has an occipital location, it frequently is felt medially rather than laterally, even when dissection is unilateral (43,47). About 46% of patients report neck pain, usually unilateral, involving the upper and midposterior cervical areas.

Quality

The pain of carotid dissection is perceived as a constant, steady aching by two thirds of patients, as throbbing by one quarter of patients, and infrequently as a steady, sharp pain (47). In vertebral dissection, most patients report the head pain as either steady, pressurelike, or throbbing; only a few patients report the pain as being sharp (47). The severity of the pain is highly variable, ranging from a mild sensation of tenderness to an excruciating headache simulating subarachnoid hemorrhage (3,4,7,9,15–17,20,27,30,42,43,47,48,52).

Duration

Often, published series do not mention the duration of the pain. In the vast majority of patients (90%), the head pain of carotid dissection resolves within 1 week (4,47). In vertebral dissection, the headache may last up to 5 weeks (47). In rare cases, however, the headache may last for years after the dissection, particularly in patients who develop pseudoaneurysms (36,37,47).

In summary, a unilateral cervical pain of sudden onset, radiating to the ipsilateral eye or ear, is suggestive of carotid artery dissection. This condition can occur almost as frequently with other varieties of head and neck pain, however, and it can mimic migraine, cluster headache, carotidynia, subarachnoid hemorrhage, or even Raeder syndrome (3,48).

ASSOCIATED CLINICAL MANIFESTATIONS

Carotid Artery Dissection

Signs and symptoms of retinal or cerebral ischemia are the most common associated manifestations of carotid artery dissection. For example, a unilateral head or neck pain in a patient presenting with amaurosis fugax, transient ischemic attacks, or stroke strongly suggests the possibility of internal carotid artery dissection (1,4–6,14–16,34–37, 42,43,47). Also, in almost half of the cases, the presence of ipsilateral local signs associated with the cephalic pain is a key to the diagnosis. A third-order Horner syndrome is the most frequent, and a highly suggestive, local sign of internal carotid artery dissection (6,16). Painful Horner syndrome has been found in up to 58% of patients (1,4,6,14–16,34–37,43,47), and in about 10% it is the only clinical manifestation of carotid dissection (6,16,47). Other manifestations include pulsatile tinnitus, dysgeusia, tongue paresis, and diplopia (1,4,6,14–16,34–37,42,43,47). The absence of both ischemic and local signs is unusual; occasional case reports have described patients presenting only with head or neck pain (3,6,7,9,16,17,20,27,30,48,52).

Vertebral Artery Dissection

The usual associated signs are those of brainstem or cerebellar ischemia and those of ischemia to more remote areas, such as the occipital lobes, as a result of artery-to-artery embolism (36,43,47). Of the recognizable syndromes, Wallenberg syndrome is most commonly reported (17,37,43). Consequently, an occipital or posterior neck pain associated with signs of vertebrobasilar territory ischemia, especially with Wallenberg syndrome, in a young patient strongly points to the diagnostic possibility of vertebral artery dissection (17,27,47). Rarely, dissection may result in spinal cord ischemia (43).

As previously indicated, ischemic signs often are delayed, occasionally up to 1 month after the onset of pain (5). It is thus crucial to maintain a high index of suspicion and recognize the arterial dissection before a stroke occurs.

PROGNOSIS

Headache usually precedes the onset of ischemic signs and therefore requires early diagnosis and treatment (4,15,16,47). The headache usually resolves spontaneously within a few days (4,15,16,47).

The prognosis of stroke is related to the severity of the ischemic insult and the extent of collateral circulation. The potential for the development of collateral circulation may be compromised when more than one vessel is dissected.

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The reported rate of death from dissections of the carotid and vertebral arteries is less than 5%, and about three fourths of patients who have had a stroke make a good functional recovery (43).

It is very uncommon to see recurrence of dissection in a previously dissected and healed cervical artery, but it is not uncommon for recurrent dissection to occur in the other cervical arteries of the same patient. The risk of such recurrence is maximal within the first 2 months (2%) after initial dissection and becomes much less thereafter (approximately 1% per year) (43).

MANAGEMENT

Progression of the neurologic deficit regularly occurs in the early days following dissection, and rapid recanalization and resolution of the radiologic signs of the dissection are known to occur, which could obscure the diagnosis (6,43,44,47). Therefore, prompt diagnostic investigations should be performed within the first few days after the onset of pain, if possible, when cervical artery dissection is suspected. Diagnostic investigations include noninvasive vascular studies such as extracranial duplex ultrasound scanning, magnetic resonance imaging (MRI) and angiography (MRA) or computed tomography angiography (CTA) (Figs. 111-1 and 111-2), and, if necessary, conventional cerebral angiography. Noninvasive vascular studies have become very reliable in suggesting the diagnosis and are useful tools for following the vascular response to treatment (43). It should be kept in mind, however, that each of the noninvasive investigations can give false-negative results, even at an early stage.

Treatment of cervical artery dissections revealed only by the presence of headache is aimed at prevention of ocular and cerebral infarction. Because of the unpredictable risk of hemodynamic stroke in dissections with occlusion or severe stenosis, initial therapy should include immediate strict bed rest and prevention of hypotension until recanalization of the artery or effective collateral circulation can occur. The value of each of these treatment modalities has not been established (2,29,44).

Empiric treatment with anticoagulants usually is prescribed to prevent complete occlusion of the dissected artery or artery-to-artery embolization (Grade C recommendation) (44). Careful monitoring with noninvasive vascular studies, such as extracranial duplex scanning, transcranial Doppler sonography, CTA, and MRA, are extremely helpful to assess the adequacy of the intracranial circulation and should be used to monitor these patients to prevent subsequent ocular or brain infarction (5,43,44).

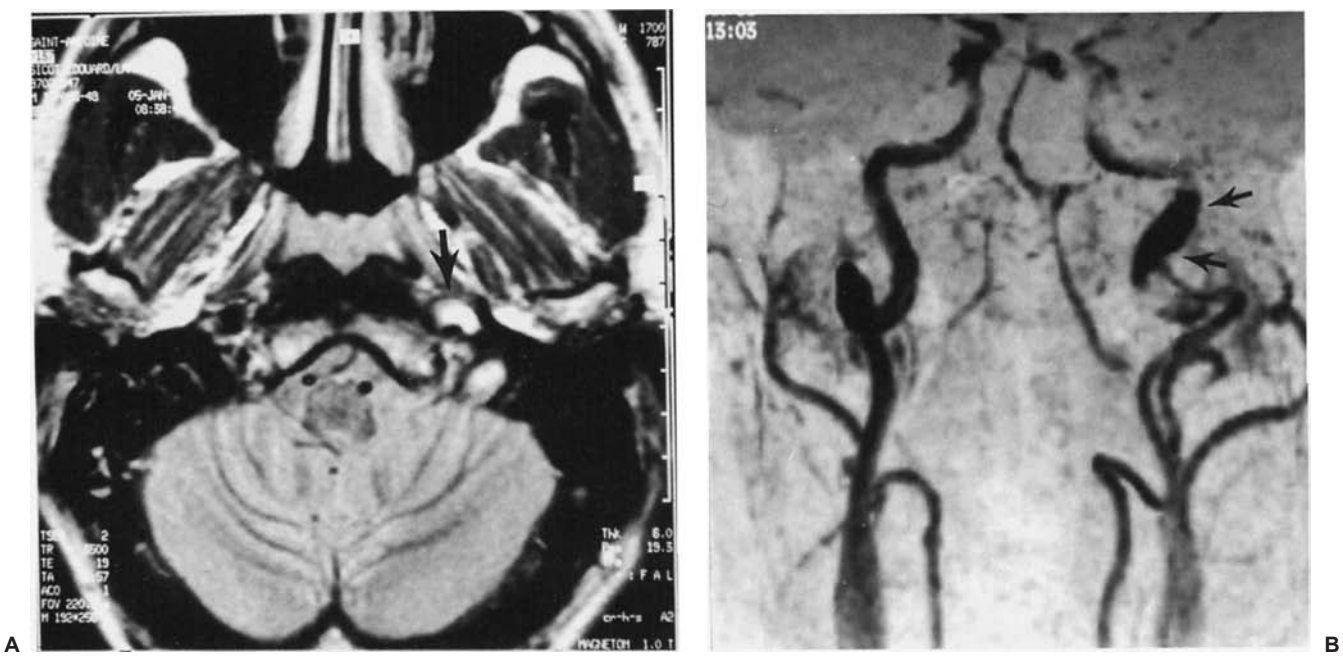

FIGURE 111-1. Dissection localized to the petrous segment of the left extracranial internal carotid artery. **A:** Axial T2-weighted imaging of the brain demonstrating a typical eccentric hyperintense signal of the internal carotid artery corresponding to the mural hematoma (arrow) surrounding the residual lumen. **B:** Magnetic resonance angiography showing a left internal carotid artery stenosis associated with a subpetrous hematoma (arrows) with increased external diameter typical of dissection.

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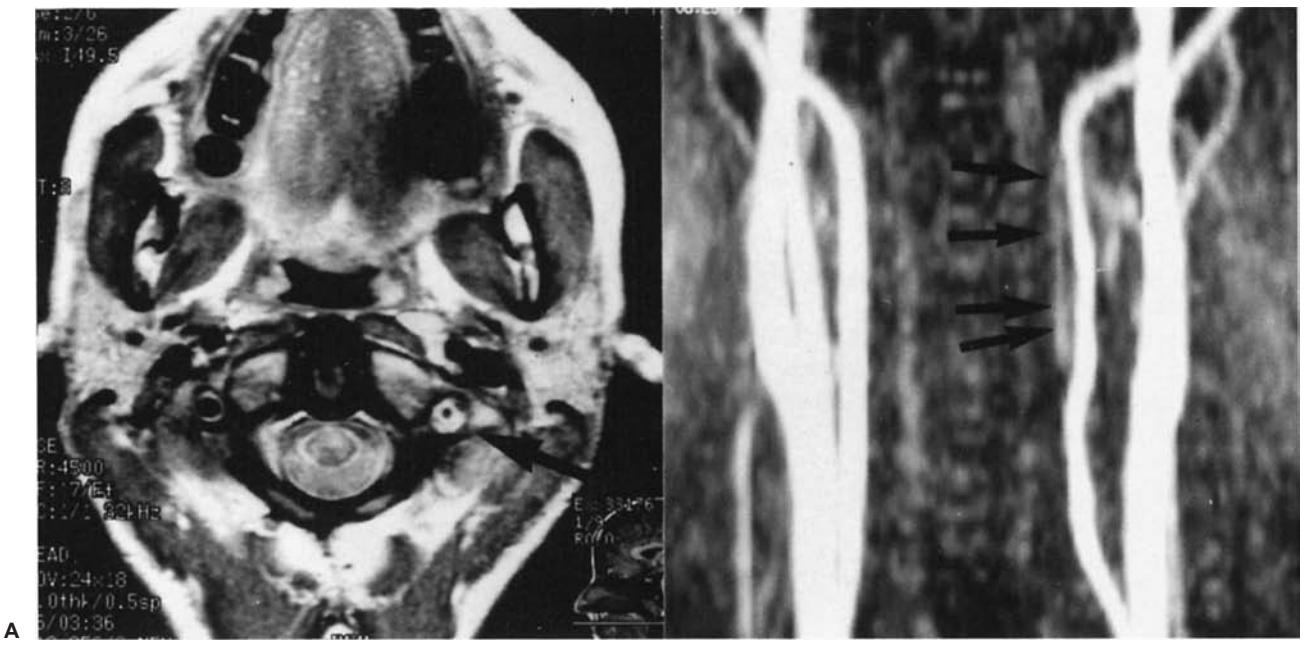

FIGURE 111-2. Left extracranial vertebral artery dissection. **A:** Axial T2-weighted imaging of the brain demonstrating a hyperintense signal of the left vertebral artery corresponding to the mural hematoma (arrow) surrounding the residual lumen. **B:** Magnetic resonance angiography showing a signal corresponding to the mural hematoma (arrows) with increased external diameter consistent with a dissection.

CERVICAL ARTERY DISSECTIONS AND MIGRAINE

The relation between migraine and carotid artery dissection is complex and not clearly defined. It has been suggested that migraine is a predisposing factor for cervical artery dissection (11,51). Also, dissection can present with features of an attack of migraine with or without aura (42,47). Indeed, some cases of so-called migrainous infarcts have radiologic features typical of internal carotid artery dissection. Conversely, vascular spasm during cerebral angiography may be mistaken for dissection in migraineurs.

POSTCAROTID ENDARTERECTOMY HEADACHE

Definition

IHS code and diagnosis: 6.5.2 Postendarterectomy headache

WHO code and diagnosis: G44.81 Headache associated with other vascular disorders

Short description: Postendarterectomy headache is defined as headache ipsilateral to the side of the procedure, beginning within a few days of carotid endarterectomy, and where appropriate diagnostic studies fail to reveal occlusion or dissection of the carotid artery (49).

CLINICAL FEATURES

IHS diagnostic criteria (ICHD-II) for postcarotid endarterectomy headache are as follows:

A. Any new acute headache.
B. Carotid endarterectomy.
C. Headache, in the absence of dissection, begins within 1 week of surgery.
D. Headache lasts <1 month after surgery.

Comment: Most reported studies on this variety of headache (8,10,12,13,25,28,32,40,50) did not use these diagnostic criteria, because carotid angiography or ultrasonography were not systematically performed to assess the internal carotid artery patency, and several studies included patients with bilateral headache or headache occurring more than a few days after the operation. Taking into account patients in whom headache occurs within 1 month after carotid surgery, with either no past history

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of headache or previous headache of clearly different type and with a patent internal carotid artery as assessed by angiography or ultrasonography, the following varieties can be described:

1. Headache accompanying a postoperative ischemic or hemorrhagic stroke occurring in the absence of carotid occlusion or dissection. This type of headache, which is always associated with focal neurologic deficits or alteration of the level of consciousness, should not be included in postcarotid endarterectomy headache (8,10,12,13,25,28,32,49).
2. A mild, diffuse, nonspecific headache that is not associated with focal deficits, seizures, or an increase in systemic blood pressure (32,40,49). This is the most frequently reported postendarterectomy headache (up to 60%) (49). It occurs in the first 5 days following surgery, particularly in the first 2 days (49). It is more often bilateral than unilateral and preferentially affects the frontal regions. It is usually of mild or moderate intensity, is described as a sensation of pressure or heaviness, and requires no treatment (49). The headache could be either continuous or intermittent, and its average duration is approximately 3 days. A clear relationship between this type of headache and the surgery remains elusive in the absence of properly conducted case-control studies.
3. Clusterlike headache, which has been reported in up to 38% and resolves in about 2 weeks after carotid endarterectomy (12,32,49). The headache is ipsilateral to the surgical side, occurs hours to days after the operation, consists of attacks lasting 2 to 3 hours and occurring once or twice daily, and resolves spontaneously in 2 to 25 days in most cases (32,49). There are no prodromal symptoms. The pain is pulsating, moderate or severe, and located mainly in the retro-orbital and temporoparietal regions. Occasionally, the headache is accompanied by ipsilateral conjunctival injection, lacrimation, rhinorrhea, nasal stuffiness, Horner syndrome, and decreased activity of the oculosympathetic system, based on pharmacologic pupillary testing (12). The latter dysfunction suggests that direct surgical damage to the pericarotid sympathetic plexus is an essential element for its production.
4. A severe unilateral headache of the cerebral hyperperfusion syndrome, which is rare and observed mainly after correction of a high-grade stenosis in patients with long-lasting, severe, chronic cerebral ischemia (8,10,12,13,25,28,32,40,49). This type of headache is severe, unilateral, and throbbing. It starts postoperatively after a headache-free latent interval of about 3 days. It is often preceded by an increase in systemic blood pressure and is accompanied by seizures and contralateral focal deficits (8,10,13,25,28,32,49). It may herald the occurrence of cerebral hemorrhage (10) but has been reported mainly in the absence of stroke (9,11). CT scan of the brain is often normal, but it may demonstrate diffuse or patchy cerebral edema consistent with hyperperfusion. This syndrome requires careful management. A high index of suspicion will lead to prompt diagnostic evaluation with invasive or noninvasive vascular studies to exclude the possibility of carotid or other major cerebral vessel occlusion and thus avoid the unnecessary use of potentially dangerous treatment modalities, such as anticoagulants.
5. Other headaches, such as gustatory pain, severe hemicrania, delayed cluster headache, chronic paroxysmal hemicrania, cervical pain, and Eagle syndrome (32,49,50). In all these cases, however, any past history of headache, the time course of the pain, and assessment of carotid patency were poorly documented; consequently, no firm conclusions about their direct connection with the carotid surgery can be reached.

CAROTID ANGIOPLASTY HEADACHE

Definition

IHS code and diagnosis: 6.5.3 Postendarterectomy headache

WHO code and diagnosis: G44.81. Headache associated with other vascular disorders

Short description: Carotid angioplasty headache is defined as headache or cervical pain ipsilateral to the side of the procedure, beginning during or within a few days of carotid angioplasty, and where appropriate diagnostic studies fail to reveal occlusion or dissection of the carotid artery.

CLINICAL FEATURES

IHS diagnostic criteria (ICHD-II) for carotid angioplasty headache are as follows:

A. Any new acute headache.
B. Extra- or intracranial angioplasty.
C. Headache, in the absence of dissection, begins during angioplasty or within 1 week.
D. Headache lasts <1 month.

Comment: Carotid angioplasty and stenting are currently under evaluation (18,26). There are very few data on headaches and most studies do not even mention headaches in the patients' outcome. Cervical pain or ipsilateral headache are commonly reported by patients during the balloon inflation and mostly disappear with seconds of balloon deflation (38). Headache as part of the hyperperfusion syndrome (see postendarterectomy headache) has also been reported after angioplasty and stenting (24,33,45).

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HEADACHE IN INTRACRANIAL ENDOVASCULAR PROCEDURES

Definition

IHS code and diagnosis: 6.5.4 Headache associated with intracranial endovascular procedures
WHO code and diagnosis: G44.81. Headache associated with other vascular disorders
Short description: Localized headaches have been reported after endovascular procedures such as balloon inflation or embolization of intracranial arteriovenous malformations and aneurysms. The pain is usually severe, develops immediately during or after the procedure, and resolves rapidly.

CLINICAL FEATURES

IHS diagnostic criteria (ICHD-II) for headache in intracranial endovascular procedures are as follows:

A. Unilateral severe localized pain of abrupt onset.
B. Intracranial angioplasty or embolization.
C. Headache starts within seconds of the procedure.
D. Headache disappears within a few hours of the end of the procedure.

Comment: As for carotid angioplasty and stenting, there are very few data on headaches and most studies do not mention headaches in the patients' outcome. The pain is always acute, severe, and very localized to the head or face depending on the artery involved (31,39). The pain occurs within a few seconds after the procedure and resolves rapidly.

ANGIOGRAPHY HEADACHE

Definition

IHS code and diagnosis: 6.5.5 Angiography headache
WHO code and diagnosis: G44.81. Headache associated with other vascular disorders
Short description: Angiography headache is defined as headache beginning during or within minutes of carotid or vertebral angiography and where appropriate diagnostic studies fail to reveal occlusion or dissection of the artery.

CLINICAL FEATURES

IHS diagnostic criteria (ICHD-II) for angiography headache are as follows:

A. Any new acute headache.
B. Intraarterial carotid or vertebral angiography.
C. Headache occurs during angiography.
D. Headache lasts <72 hours.

Comment: The intracarotid or intravertebral injection of contrast may induce a severe headache with a burning sensation. It usually disappears immediately after the injection. The injection can also trigger a migraine attack (41,46,54).

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