Cerebellar Stroke

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Abstract

Cerebellar infarction and hemorrhage share symptoms and signs according to the cerebellar structures affected. Although the clinical features of isolated cerebellar infarction are similar across the three main cerebellar vascular territories, dysarthria is most likely to be associated with lesions in the territory of the superior cerebellar artery, whereas vertigo and lateropulsion appear to be more often seen in lesions involving the territory of the posterior inferior cerebellar artery. Audiovestibular symptoms are characteristic in lesions in the anterior inferior cerebellar artery territory. The main etiologies of cerebellar stroke are artery-to-artery embolism and cardioembolism, followed by in situ branch atherosclerosis. The increasing availability of MRI and advent of new imaging techniques allow us to better define the cerebellar stroke syndromes and identify the mechanisms of stroke. Acute cerebellar stroke requires constant vigilance by clinicians owing to often rapid clinical deterioration. This deterioration results from brainstem compression by mass effect, evolving hydrocephalus, or irreversible brainstem infarction. Medical therapy, endovascular procedures, or surgical interventions can improve patients' outcomes. This chapter reviews cerebellar stroke, from clinical features to the mechanism, potential complications, diagnosis, and treatment of patients.

Keywords

Cerebellar · Clinical feature · Hemorrhage · Infarction · Mechanism

Introduction

Cerebellar infarction accounts for 2–10% of cases in clinical series of infarctions in the brain (Bogousslavsky et al. 1988; Amarenco 1991; Tohgi et al. 1993; Amarenco et al. 1994). Although the incidence of cerebellar infarcts has increased along with the improvement of brain imaging techniques, the majority of cerebellar infarcts have a benign clinical course. The hydrocephalus and brainstem compression resulting from post-infarct edema are associated with a high morbidity and mortality but remain relatively rare (Kase et al. 1993; Chaves et al. 1994). The mean age of patients is 60–70 years, and about two thirds of patients are men (Tohgi et al. 1993). As the population of developed countries becomes older, the incidence of cerebellar infarction is expected to rise. Overall, the infarction in posterior inferior cerebellar arteries (PICA) territory is more common than that in the superior cerebellar arteries (SCA), and anterior inferior cerebellar artery (AICA) infarction is the least common (Kase et al. 1993; Tohgi et al. 1993; Kumral et al. 2005a, b, 2006). In the largest series of cerebellar infarctions, about 90% of cases were unilateral (Tohgi et al. 1993), but the unilateral infarctions sometimes involve more than one vascular territory (Canaple and Bogousslavsky 1999). As for patients with stroke in the anterior circulation, the two common etiologies of cerebellar infarction are cardioembolism and large artery atherosclerosis (Amarenco et al. 1990a;

Bogousslavsky et al. 1993; Kase et al. 1993; Tohgi et al. 1993; Chaves et al. 1994; Dziadkowiak et al. 2016). Vascular risk factors for ischemic stroke including hypertension, diabetes, dyslipidemia, cigarette smoking, atrial fibrillation, and history of stroke or TIA also apply to cerebellar infarction (Kase et al. 1993; Tohgi et al. 1993). The relative contribution of the different etiologies and the distribution of large artery atherosclerosis depend on ethnic origin, age, and sex (Caplan et al. 1986). Vertebral artery dissection has been noted as another important cause of cerebellar infarction, particularly in younger patients (Schievink 2001; Arnold et al. 2006). Rare causes that have been associated with cerebellar infarction include vasculitis (McLean et al. 1993), hypercoagulable states (Amarenco et al. 1994), acute drug intoxication (Aggarwal and Byrne 1991), and migraine (Reid et al. 2006). Cerebellar hemorrhage is the least common type of intracranial hemorrhage (ICH) constituting 10% of all ICH cases (Flaherty et al. 2005). The mortality rates of cerebellar hemorrhage range from 20% to 75% (Kirollos et al. 2001; Salvati et al. 2001). Initial mortality is high because of swelling in the posterior fossa (Jauss et al. 1999). Hypertension is the major risk of cerebellar hemorrhage and is reported to account for 60–89% of all cases, while arteriovenous malformation (AVM), aneurysms, amyloid angiopathy, and coagulopathy account for the rest (St Louis et al. 1998; Kirollos et al. 2001). This chapter summarizes current understanding of cerebellar stroke and related clinical syndromes, causes of infarcts and hemorrhages, and therapeutic challenges. It also emphasizes the importance of recognizing subtle indicators of potentially life-threatening complications.

General Clinical Features

Cerebellar stroke syndromes depend on the vascular territory, infarct size, and associated lesions in the brainstem (Amarenco et al. 1991; Kase et al. 1993; Lee 2009). The main clinical features include vertigo/dizziness, nausea and vomiting, gait instability, and headache. The important signs of the neurological examination, which are commonly discounted in primary care, may include dysarthria, ataxia, and oculomotor dysfunction. However, cerebellar signs might be absent, subtle, or difficult to distinguish from more common and benign, self-limited disorders. The most common symptoms in patients with isolated cerebellar infarctions are vertigo (90%) and lateropulsion (85%) (Min et al. 1999a; Ye et al. 2010). However, about 40% of the patients have isolated vertigo and/or lateropulsion without any other cerebellar signs, and cerebellar ataxia is observed only in 40% of patients with isolated cerebellar infarctions (Ye et al. 2010). Thus, special attention has to be paid to patients with vertigo or lateropulsion and stroke risk factors, even if they do not have other cerebellar symptoms or signs (Lee et al. 2006). The symptoms of cerebellar infarction often begin simultaneously with brainstem symptoms or signs (e.g., Horner's syndrome, hearing loss, tinnitus, limb weakness, sensory loss, facial palsy, dysphagia, or altered consciousness) if multiple locations in the posterior fossa are involved (Min et al. 1999a). Pyramidal symptoms can occur, especially when multiple cerebellar artery territories are affected. The presence of pyramidal

symptoms indicates poor prognosis of acute stroke in the cerebellum (Dziadkowiak et al. 2016). In rare cases, the presentation of cerebellar infarction, particularly in association with incipient basilar artery thrombosis, can be severe (e.g., coma and quadriplegia) (Kase et al. 1993; Tohgi et al. 1993).

Headache

Headache is more common in strokes in the posterior circulation than in the anterior circulation and particularly in cerebellar infarction (Tentschert et al. 2005). About 40% of patients with cerebellar infarction suffer from headache (Kumral et al. 2005a, b). Headache is more common in lesions involving the folium-tuber, pyramis, or posterior hemisphere, which are parts of the PICA territory (Kase et al. 1993). The headache can result from the stroke itself or the causative vascular lesion (e.g., vertebral artery dissection). In patients with cerebellar stroke, headache was more common in the cervical and occipital areas than in other areas, and the location was ipsilateral to the infarct when the headache was unilateral (Kase et al. 1993). Although headache is extremely common in a number of neurologic and systemic illness, more detailed interpretation of the history and neurologic examination and laboratory studies are required in several situations. This is exemplified by young patients presenting with severe headache that either precedes the onset of posterior circulation symptoms, especially following vigorous physical activity, or that persists for a longer time than the typical duration of primary headache, raising the concern for vertebral artery dissection (Saeed et al. 2000).

Dizziness

Dizziness and/or vertigo are common symptoms in general practice and occur in nearly three quarters of patients with cerebellar stroke (Tohgi et al. 1993; Kumral et al. 2005a, b). Although dizziness can be classified into one of four categories including vertigo, presyncope, imbalance, and nonspecific dizziness, based on the symptom characteristics described by patients, recent studies suggest that this approach would not be optimal to identify cerebellar stroke (Kerber et al. 2006; Newman-Toker et al. 2007). Rather, the duration and triggering factors of dizziness are more reliable and useful than the characteristics of the dizziness (Newman-Toker et al. 2007). Distinguishing central from peripheral causes can be straightforward when posterior neurologic signs manifest. Clues to central dizziness are most likely to be found in the severity of gait imbalance and detailed assessment of oculomotor function, but these signs are present in fewer than 50% of cerebellar strokes (Newman-Toker et al. 2008). The territory of cerebellar infarction associated with dizziness frequently involves the medial PICA (medPICA) (Norrving and Magnusson 1995), because the medPICA supplies the uvula (vermal lobule IX) and nodulus (vermal lobule X), key components of the vestibulocerebellum with strong connections to the ipsilateral vestibular nuclei (Voogd et al. 1996;

contemporary nomenclature according to Schmahmann et al. 2000). Nausea and vomiting occur in over half of cerebellar strokes in association with dizziness (Kase et al. 1993; Tohgi et al. 1993). However, in some cases, nausea and vomiting might be disproportionate to any associated dizziness. In one study, seven of 12 patients with SCA infarcts had vomiting without vertigo at onset of stroke (Kase et al. 1993). The key signs distinguishing acute vestibular symptoms associated with cerebellar infarction from peripheral vertigo are the normal head thrust test and caloric test results (Lee et al. 2006). HINTS (head impulse, nystagmus, and test of skew) is a key examination in accurately diagnosing stroke in the acute vestibular syndrome (Lee and Kim 2015). Normal head impulse test, direction changing nystagmus and skew deviation is strongly suggestive of cerebellar lesion (Kattah et al. 2009).

Ataxia

Limb ataxia is often thought to be an essential sign of cerebellar stroke as it is present in about 40% of patients with cerebellar stroke (Ye et al. 2010). However, this symptom is not seen in 40% of patients with cerebellar lesions (Tohgi et al. 1993; Kumral et al. 2005a, b) and is sometimes absent even in patients with sizeable infarcts (Lee et al. 2006). Moreover, limb ataxia might also manifest in patients with supratentorial lesions (Gorman et al. 1998). Limb ataxia is a sign of infarction of the lateral branches of SCA and PICA. Limb ataxia in PICA infarction has been associated with damage in the relevant output structure, the interposed or dentate nuclei, or additional damage in proximal branches supplying the inferior cerebellar peduncle, one of the important input structures of the cerebellum (Deluca et al. 2007). Given that the main activation areas for control of the hands and feet are located in the ipsilateral anterior lobe (Grodd et al. 2001), the limb ataxia occurs more frequently in lesions of the culmen (lobules IV and V), anterior paravermis, and anterior hemisphere. About half of the patients with cerebellar stroke describe gait instability (Kase et al. 1993; Tohgi et al. 1993). Although acute peripheral vestibulopathy may cause gait instability and veering tendency, the inability to sit, stand, or walk increases the likelihood that the cause is central (Baloh 2003). Most patients fall toward the side of the lesion when the stroke is unilateral (Kase et al. 1993; Lee et al. 2006). However, in one study, 32.5% of patients with unilateral cerebellar lesions fell to the contralateral side of the lesion or bilaterally (Ye et al. 2010). Truncal ataxia of stance and gait are more severe in medial superior cerebellar artery branch (medSCA) and medPICA compared to lateral SCA (latSCA) and lateral PICA (latPICA) infarction (Amarenco et al. 1991; Sohn et al. 2006).

Dysarthria

Dysarthria is also a common sign (50%) among patients with cerebellar stroke (Tohgi et al. 1993; Kumral et al. 2005a, b). Although dysarthria is not unique to posterior circulation infarcts, cerebellar-type dysarthria is characterized by ataxic

and slurred speech, explosive staccato scanning vocalization, and wavering modulation. Dysarthria that co-occurs with dizziness almost always supports a central rather than a peripheral cause. Responsible lesions of cerebellar dysarthria include the lingual (lobuels 1, II)-centralis (lobule III), anterior paravermis, and anterior hemisphere, supplied by the medial superior cerebellar artery (medSCA) (Kase et al. 1993; Urban et al. 2003; Kumral et al. 2007).

Ocular Motor Dysfunction

Oculomotor signs include nystagmus, impaired smooth pursuit, and ocular tilt reaction (OTR). Nystagmus is present in almost half of patients with cerebellar infarction but is also common with peripheral vestibulopathies (Ye et al. 2010). Among patients with unilateral lesions, ipsilateral nystagmus was more common than contralateral nystagmus (Ye et al. 2010). The type of nystagmus can help distinguish central from peripheral lesions. Vertical or torsional nystagmus occurring spontaneously and horizontal nystagmus that is evoked by lateral gaze generally indicate a central cause (Newman-Toker et al. 2008). Nystagmus is more common in medPICA compared to latPICA and SCA infarction (Amarenco et al. 1990b; Barth et al. 1994). Horizontal sinusoidal smooth pursuit eye movements are impaired in patients with lesions in the uvula and the pyramid of the vermis (Baier et al. 2009). Likewise, the fixation suppression of the vestibulo-ocular response (VOR) and slow phase of optokinetic nystagmus (OKN) can be deficient (Baier et al. 2009). Ocular tilt reaction (OTR: head tilt, conjugated eye cyclotorsion, skew deviation, impaired vertical perception) also occurs in patients with cerebellar infarctions (Min et al. 1999a; Lee et al. 2005). Although no systematic clinical studies are available on the OTR occurrence in patients with cerebellar stroke, the lesion in the dentate nucleus is associated with contralateral signs of OTR, whereas the ipsilateral signs involve the middle cerebellar peduncle, tonsil (lobules IX), biventer (lobule VIII), and inferior semilunar lobules (crus II and lobule VIIB) (Baier et al. 2008).

Cognitive Functions

The cerebellum is an integral component of the distributed neural circuits that contribute to cognitive functions. Anatomical, functional, and radiological data of human cerebellar lesions support this assumption (Schmahmann 1991; Schmahmann and Pandya 1997; Middleton and Strick 2001; Ramnani et al. 2006; Stoodley and Schmahmann 2009). Thus, a localized cerebellar infarction may lead to a specific behavioral deficit resulting from functional disruption anywhere within that circuitry. Several lines of evidence suggest that patients with cerebellar infarction are impaired in performing a fast and efficient visual search (Machner et al. 2005) and in everyday executive functioning abilities (Manes et al. 2009). Cerebellar cognitive affective syndrome has been defined in patients with impairments of executive functions, difficulties with spatial cognition including visual-spatial

organization, and memory and personality changes with blunting of affect or inappropriate behavior (Schmahmann and Sherman 1998). A predominant role of the posterior parts of the cerebellum in cognitive and affective processes has been identified (Exner et al. 2004). It is known that there can be crossed cerebellocerebral diaschisis as shown by single photon emission tomography (SPECT) study in cerebellar infarction. Diaschisis is explained as a functional depression of cerebello-thalamic-cerebral pathway (Boni et al. 1992). Arterial spin labeling imaging can also detect the diaschisis in 75% of patients with hyperacute infarction in the anterior circulation (Kang et al. 2017). The occurrence is influenced by the degree of the anterior perfusion reduction and location rather than infarct volume (Sommer et al. 2016). This phenomenon can support the nature of cerebellar cognitive dysfunction.

Cerebellar Infarction

The cerebellum is supplied by three main arteries, i.e., PICA, AICA, and SCA (Fig. 1). Cerebellar infarctions can be divided into territorial infarcts and non-territorial infarcts that are small cerebellar infarcts, which are either unilateral or

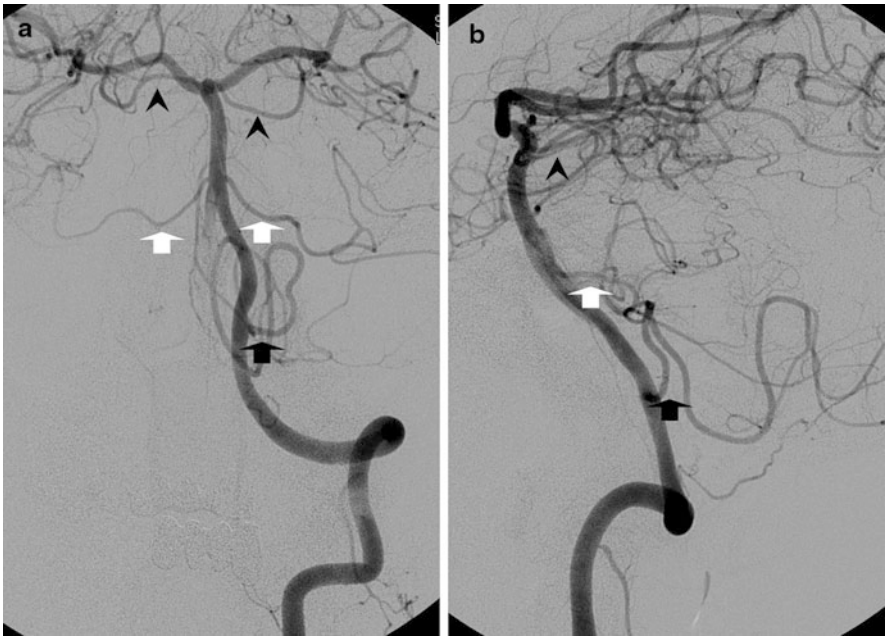

Fig. 1 Cerebellar vascularization. Conventional angiography (**a**, frontal view; **b**, lateral view) shows the general anatomy of vessels in the posterior fossa. *Black arrowheads*, *white arrows*, and *black arrows* indicate superior cerebellar artery (SCA), anterior inferior cerebellar artery (AICA), and posterior inferior cerebellar artery (PICA), respectively

bilateral. Whereas PICA and SCA infarcts are frequently restricted to the cerebellum, AICA territory infarcts almost always include the lateropontine area, and brainstem signs predominate (Amarenco and Hauw 1990a; Amarenco et al. 1993a; Barth et al. 1993). Territorial infarction topography is demonstrated in Fig. 2. The leading cause of cerebellar infarcts has been suggested as emboli arising from cardiac or intra-arterial sources at the orifice of the vertebral artery (Caplan et al. 2004). But cerebellar infarctions, especially in PICA territory, with distal vertebral arterial disease are also common in the authors' personal view. In a large-scale study

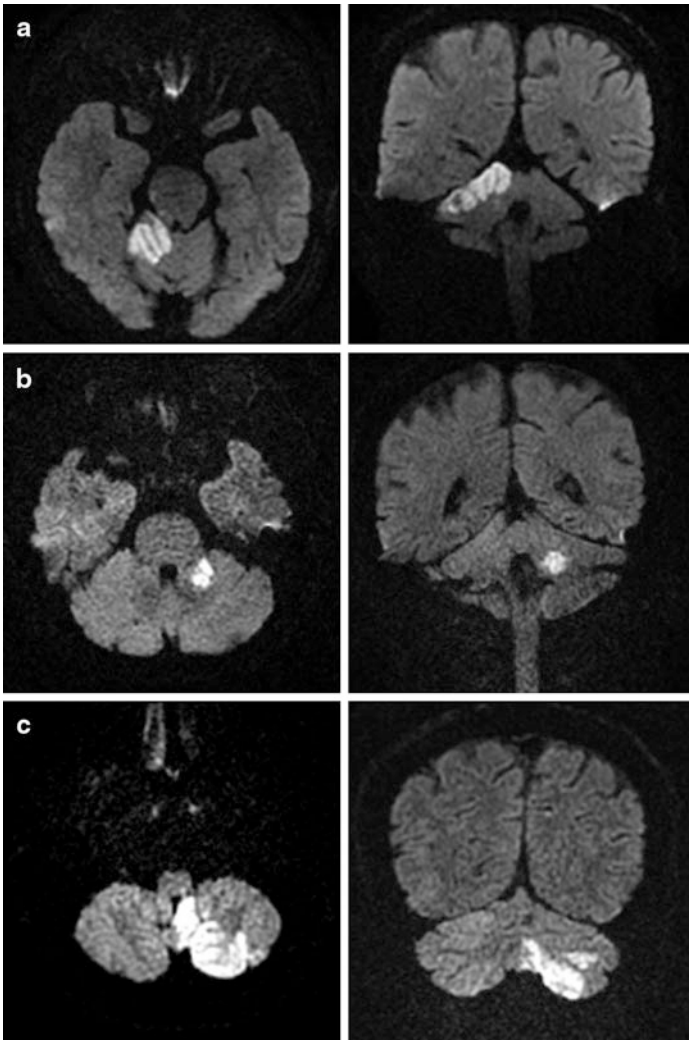

Fig. 2 Cerebellar territorial infarctions. Representative diffusion-weighted magnetic resonance images demonstrate the topography of SCA (**a**), AICA (**b**), and PICA infarction (**c**)

to analyze the MRI-identified lesions with conventional angiographic findings, cerebellar infarctions were associated with proximal large artery disease in 71% of patients, in situ branch artery disease in 19.4%, and no angiographic abnormality in 9.6% (Min et al. 1999b). In this series, V1 disease of the vertebral artery was the most common angiographic feature in large artery disease. The most frequent recipient sites of intracranial artery-to-artery embolism were the PICA and SCA regions (Caplan et al. 1992; Wityk et al. 1998). The mechanism for stroke in this group can be explained by either artery-to-artery embolization or diminished blood flow. In patients with in situ branch artery disease, the extent of cerebellar lesions appears to depend on the status of collateral flow. In those patients, the potential role of embolization to a more distal artery remains unknown. Nonterritorial small infarcts are also found commonly in patients with large artery disease, suggesting that even a small cerebellar infarct can be a clue to the presence of a large artery lesion in the posterior circulation (Min et al. 1999b). The mechanism of small infarcts can be explained by an artery-to-artery embolism or hemodynamic compromise, but small artery disease may be a possible mechanism in patients with no angiographic disease. Clinical features and potential mechanisms for cerebellar stroke type are depicted in Table 1.

Table 1 The spectrum of cerebellar strokes

		Symptom and sign	Mechanism
PICA	Whole	Vertigo, vomiting, gait and trunk ataxia, headache, nystagmus, limb hypotonia	Embolus from the heart or great arteries (60%) Intracranial vertebral artery (VA) disease (40%)
	Medial	Isolated vertigo, ipsilateral axial lateropulsion of the trunk and gaze, dysmetria, incomplete Wallenberg syndrome	
	Lateral	Vertigo, ipsilateral dysmetria, hypotonia of ipsilateral limb	
AICA	Unilateral AICA	Vertigo, vomiting, tinnitus, dysarthria	Branch atheromatous disease (50%) Basilar artery (BA) or bilateral intracranial vertebral artery disease (50%)
		Pure vestibular syndrome	
	AICA plus	Ipsilateral: trigeminal sensory loss, facial palsy, hearing loss, dysmetria, Horner's sign, conjugate lateral gaze palsy	
		Contralateral: hemibody hypesthesia, hemiparesis	
SCA	Dorsomedial	Dysarthria, gait ataxia	Cardioembolism (40–70%) Artery-to-artery embolism from BA, VA, and aortic arch (15–30%)
	Lateral	Limb ataxia, ipsilateral axial lateropulsion, dysarthria, mild truncal ataxia, gait unsteadiness	
Small nonterritorial		True vertebrobasilar insufficiency Lightheadedness, pitching sensation, vertigo, disequilibrium, transient loss of consciousness	Large artery disease (50%) Embolic Hypoperfusion Cardioembolism (20%) Small artery disease (15%)

PICA Infarction

The prominent neurological characteristics of medPICA infarction include dizziness, vertigo, truncal ataxia, axial lateropulsion, and nystagmus without affecting coordination of the four extremities (Amarenco et al. 1990b; Ogawa et al. 2013). The prominent vestibular signs in medPICA infarct may be caused by involvement of the uvulo-nodular complex of the vermis. Isolated nodular infarction is extremely rare (Jeong et al. 2007). Patients with latPICA infarction usually complain of slight neurological signs including dizziness or vertigo, dysmetria, nystagmus, and mild hypotonia. By contrast to patients with medPICA infarct, ipsilateral axial lateropulsion is rare in those with latPICA infarct (Barth et al. 1994). No prominent clinical differences may be found between patients with small lesions in medPICA and latPICA regions, which may present with only dizziness or gait instability. The most striking findings of whole PICA territory infarction are vertigo, vomiting at onset, disturbances of consciousness, and sensorimotor deficits resulting from brainstem compression or hydrocephalus. While ataxia or impairment of fine motor task is noted in infarction in the anterior lobe, language, spatial, and executive dysfunction are more often associated with posterolateral cerebellum damage (Schmahmann and Sherman 1998; Stoodley et al. 2016). The PICA territory infarct usually results from an embolus from the heart or great vessels (Nadeau et al. 1992; Kim et al. 2004). In unilateral PICA infarction, cardioembolism is found in one fifth of the cases (Kumral et al. 2005b). More than one third of patients have steno-occlusive subclavian-vertebral atherosclerotic disease (Fig. 3a), which may cause multiple artery-to-artery embolisms or hemodynamic compromise (Wityk et al. 1998; Min et al. 1999b; Shin et al. 1999). In situ branch disease is present in one fifth of PICA infarctions and is associated with PICA territorial or nonterritorial lesions. However, Kim et al. (1998) have found that cerebellar involvement is rare in patients with isolated PICA disease, suggesting the presence of effective collateral flow within the cerebellum through the AICA or the SCA. The potential role of embolization to a more distal region remains poorly understood. Although small PICA territory cerebellar infarction generally has a benign prognosis, recurrent emboli may need to be treated.

AICA Infarction

AICA ischemic stroke has suggestive acoustic nerve symptoms. There are two causes of hearing problems in AICA infarction: one is infarction of the cochlear and vestibular nuclei in the pons, and the other is inner ear infarction of the internal auditory artery (IAA), which stems from the AICA with few exceptions (Hotson and Baloh 1998; Kim et al. 1999; Raupp et al. 2004). AICA infarction can involve combined peripheral and central vestibular damage, because AICA supplies both peripheral and central vestibular structures (Amarenco and Hauw 1990a; Amarenco et al. 1993a). Inner ear dysfunction usually appears as prodromal signs before other neurologic signs. A classical syndrome of AICA territory infarction has been found

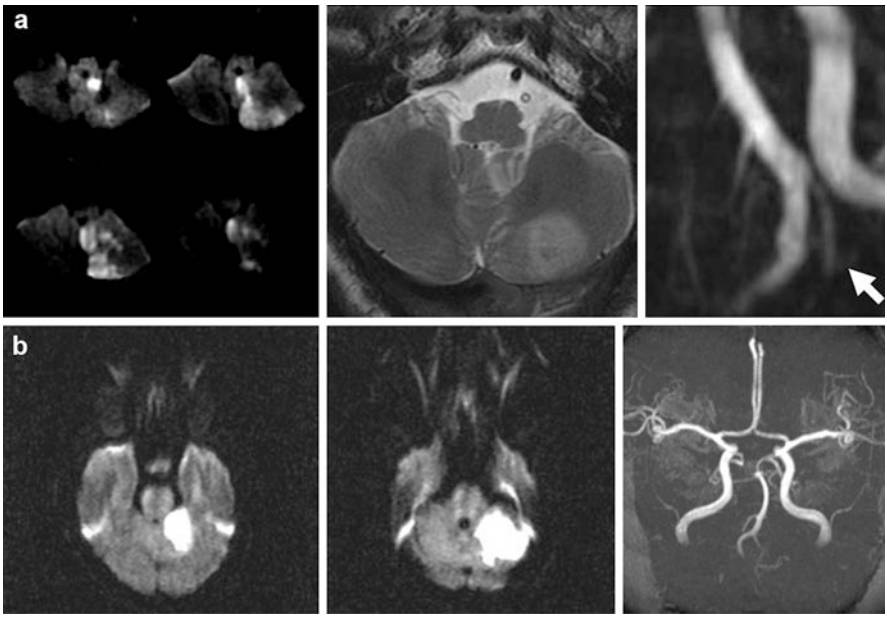

Fig. 3 Mechanism of cerebellar stroke. Brain MRI and MRA (**a**) show left cerebellar infarction in the PICA territory (*left panel*) and diffuse stenosis of the left vertebral artery (*right panel)*. The stroke mechanism is thought to be artery-to-artery embolization from the left vertebral artery proximal to the orifice of the left posterior inferior cerebellar artery. *Lower images* (**b**) show left SCA infarction. Echocardiography revealed moderate mitral stenosis and dilated left atrium with sludge and suspected thrombi in the left atrium apex. The mechanism of stroke is thought to be cardioembolism to the left SCA

in two thirds of patients with restricted lesion to the AICA territory, including vestibular and acoustic dysfunction, facial palsy, trigeminal sensory impairment, deafness, tinnitus, dissociated pain (Ogawa et al. 2017), and temperature sensory loss in the face (Amarenco and Hauw 1990a). Acoustic nerve symptoms without cerebellar ataxia can occur if infarction does not involve the brainstem and cerebellum (Hotson and Baloh 1998; Kim et al. 1999). In more than two thirds of cases, the mechanism of AICA infarction is the thrombotic occlusion of AICA itself or the extension of the vertebrobasilar system atherosclerosis into the AICA (Oas and Baloh 1992; Amarenco et al. 1993a; Tohgi et al. 1993; Min et al. 1999b; Kumral et al. 2006). In one fifth of patients, the causes of cardioembolism are detectable, but more than three fourths of these patients also have a concomitant vertebrobasilar atherosclerosis (Kumral et al. 2006). While the posterior circulation infarcts extending beyond AICA territory are mostly due to basilar artery occlusive disease (Amarenco et al. 1993a), patients with isolated AICA infarction generally have intact great arteries on magnetic resonance angiography (MRA), suggesting that isolated AICA infarcts can be caused by basilar branch occlusive disease (Lee et al. 2009).

SCA Infarction

The infarction in the SCA territory presents with a broad-spectrum of clinical manifestations such as cerebellovestibular signs, the lateral branch of SCA (latSCA) syndrome, the classical SCA syndrome, rostral basilar artery syndrome, and when occurring together with brainstem stroke, deep coma with tetraplegia (Amarenco and Hauw 1990b; Amarenco 1991; Terao et al. 1996; Kumral et al. 2005a). The most common type is latSCA territory infarction, either alone or with other cerebellar artery territory involvement (Kumral et al. 2005a). The latSCA syndrome is characterized by unsteadiness, gait ataxia, dysarthria, dizziness, and dysmetria, whereas medSCA territory infarction represents dysarthria, vertigo, truncal ataxia, and axial lateropulsion. The classical SCA syndrome combined with latSCA and medSCA infarcts presents prominent cerebellar dysfunction with dizziness, headache, dysarthria, dysmetria, and gait ataxia (Amarenco and Hauw 1990b; Amarenco 1991; Terao et al. 1996; Kumral et al. 2005a). SCA territory infarcts can be associated with multiple lesions involving other brainstem structures. Previous data reported that SCA infarcts are often accompanied by other infarcts in the territory of rostral basilar artery (Amarenco and Hauw 1990b). Motor weakness and sensory deficits involving face either alone or with limbs are present in one third of patients with territorial infarcts, mainly in those with involvement of the pons (Kumral et al. 2005a). The most common mechanism of SCA infarction is embolic, resulting from either cardioembolism or artery-to-artery embolism. A cardioembolic source can be detected in one third of the patients with the SCA infarction (Kumral et al. 2005a) (Fig. 3b) and atherosclerotic disease of the vertebrobasilar system in one third of cases (Amarenco and Hauw 1990b; Barth et al. 1993; Amarenco et al. 1994). In general, the clinical outcome of patients with the SCA infarction is better than those with a stroke in the PICA territory (Bultmann et al. 2014).

Nonterritorial Small Infarcts

Nonterritorial small infarcts typically affect the cortex and often manifest as incidental cavities on MRI (De Cocker et al. 2017). Embolism from a cardiac or arterial source is the most common cause of territorial cerebellar infarcts (Barth et al. 1993; Kase et al. 1993; Min et al. 1999b; Caplan et al. 2004). Small cerebellar infarcts are frequently associated with large artery disease involving the basilar or vertebral arteries and cardioembolism (Amarenco et al. 1993b, 1994; Mounier-Vehier et al. 1995), and they are more commonly found than territorial infarcts in patients with large artery disease (Min et al. 1999b). Therefore, small cerebellar infarcts may be a clue to the presence of a large artery disease in the posterior circulation. Small cerebellar infarcts are usually located in cortical areas, not within well-defined arterial regions, and thus are regarded as border zone infarcts (Amarenco et al. 1993b). Border zone infarction can be explained by a combination of hemodynamic mechanism and embolism (Caplan and Hennerici 1998). Hemodynamic failure was reported to be the likely mechanism in 14% of patients with nonterritorial cerebellar

infarcts (Amarenco et al. 1994). As the deep cerebellar territories have a pattern of morphologically tapering arteries with only few anastomoses, they may be susceptible to hypoperfusion-related border zone infarction (Duvernoy et al. 1983). A longer course of SCA branches supports the prevalent involvement of SCA border zones, as compared with PICA and AICA branches (Duvernoy et al. 1983). Alternatively, small cerebellar infarcts may be end zone infarcts from the small artery disease in patients with no embolic source from the heart or large arteries (Canaple and Bogousslavsky 1999). A previous pathologic report showed that the deep cerebellum is one of the predilection sites of lacunar infarction (Fisher 1965). However, small deep cerebellar infarcts have been underestimated because these lesions are mostly asymptomatic, and it is difficult to detect this small lesion during the acute period on brain CT. Additionally, less common diseases associated with small infarcts include coagulopathy, arteritis, and microembolism (Canaple and Bogousslavsky 1999).

Bilateral Cerebellar Infarction

Bilateral cerebellar infarcts have rarely been investigated (Amarenco et al. 1994; Stangel et al. 1999; Kang et al. 2000; Kim et al. 2006). However, bilateral cerebellar infarcts are not rare, constituting 31% of all cerebellar infarcts (Tohgi et al. 1993). Clinical features of bilateral cerebellar infarcts result from bilateral disturbances of cerebellar function, a high prevalence of brainstem compression, and bilateral depression of cerebral function by cerebello-cerebral diaschisis. Compared with unilateral infarcts, patients with bilateral lesions have a more severe or fluctuating clinical course and a poorer outcome and need more intracranial pressure-lowering therapy (Hong et al. 2008). The characteristics of bilateral cerebellar infarction are similar to those of unilateral cerebellar infarction, but the stroke mechanism is more likely to be large artery atherosclerosis, and they have a higher rate of stroke recurrence (Petty et al. 2000; Hong et al. 2008). Bilateral cerebellar infarcts are most common in the PICA territory, and several hypotheses have been suggested to explain their pathogenesis. Both PICAs can originate from occluded vertebral arteries, and a common PICA can arise from one side. The most plausible explanation of bilateral PICA infarction may be that about one third of medPICA is usually supplied by contralateral PICA (Kang et al. 2000). Therefore, bilateral PICA infarction often includes one-sided large PICA infarction with the other-sided small medial PICA territorial infarction. Hemodynamic compromise in the distal branches with bilateral vertebral artery disease, and multiple embolic mechanisms from the heart or proximal great arteries may contribute to bilateral infarctions (Tada et al. 1994; Hong et al. 2008). The predominant clinical presentation of bilateral SCA territory infarction is cerebellar dysarthria and prominent gait ataxia with falls (Nitschke et al. 1996). Compared with unilateral SCA territory infarction, bilateral infarctions frequently occur together with multiple infarcts in other vertebrobasilar territories (Stangel et al. 1999) and are not infrequently associated with the top of the basilar syndrome. Therefore, bilateral SCA territory infarctions should be considered a spectrum of distal basilar artery occlusion and are probably of embolic origin.

Complications

The complications of cerebellar stroke can cause neurologic deterioration and mortality. Edema formation following the initial infarct becomes space occupying within the posterior fossa, leading to brainstem compression, hydrocephalus, cardiorespiratory complications, coma, and death (Simmons et al. 1986; Koh et al. 2000). This complication occurs in 10–25% of patients, peaking on the third day after the infarction, although it can occur any time within the first week (Chen et al. 1992; Hornig et al. 1994; Koh et al. 2000). The main predisposing factor for this process is thought to be the infarct size, but other factors including type of underlying vascular lesion, hemorrhagic transformation, and inadequate collateral blood flow may be involved (Kase et al. 1993). Although the clinical deterioration seems to be independent of the affected vascular area, PICA territory infarcts are more likely to develop a mass effect than other territory infarcts (Amarenco 1991; Kase et al. 1993). Figure 4 shows a representative large PICA infarction with hemorrhage and brainstem compression. A progressive decline in level of consciousness and secondary brain stem

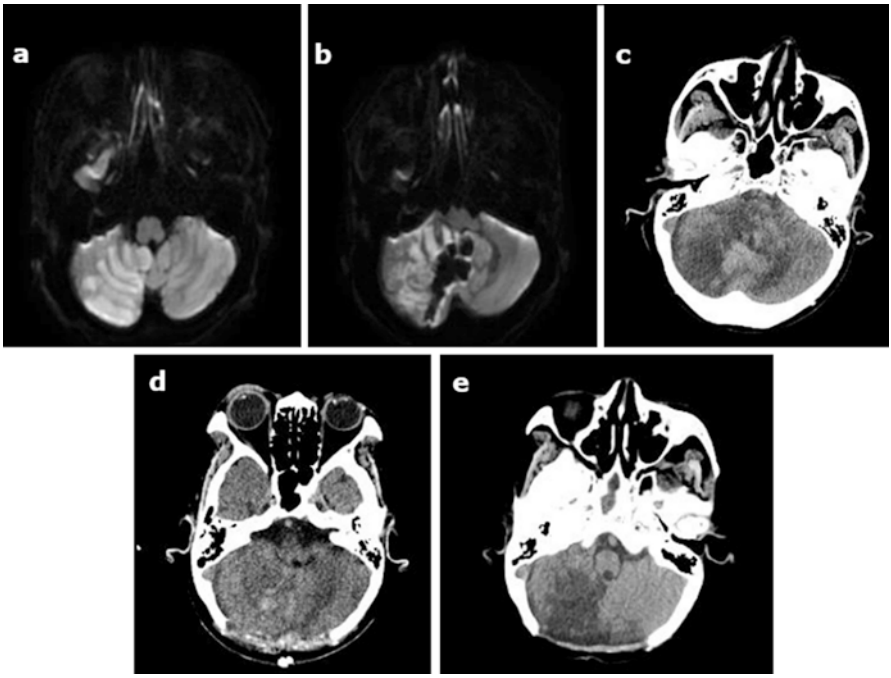

Fig. 4 Space-occupying cerebellar infarction. Brain MRIs show acute infarction in the right PICA territory (**a**) and hemorrhagic transformation in the infarcted area 1 day later (**b**). Brain CT images (**c**–**e**) show serial changes of brainstem compression resulting from cerebellar infarction. This patient underwent decompressive surgery due to brainstem compression (**c: before; d: after**). The alteration in the consciousness and respiratory depression was improved with resolution of brainstem compression (**e**: 1 week after decompression)

signs are common clinical manifestations of mass effect associated with cerebellar infarction (Heros 1992; Hornig et al. 1994; Jauss et al. 1999). The outcome after massive cerebellar infarction depends principally upon the level of consciousness (Hornig et al. 1994; Jauss et al. 1999). Therefore, close clinical monitoring for signs of deterioration is crucial. If deterioration occurs, the progression from the original vascular lesion with or without brainstem ischemia must be distinguished from secondary brainstem compression or hydrocephalus, because the treatments differ. Neurosurgical intervention by external ventricular drainage and/or decompressive craniotomy is necessary for a patient with a space-occupying cerebellar infarct to survive without disabling neurological deficit (Heros 1992). However, the timing and strategy of surgery require further clarification. Hemorrhagic transformation also increases the likelihood of mass effect (Koh et al. 2000), usually occurring within the first week of stroke onset, but sometimes in the second and third week after stroke onset (Chaves et al. 1996). The main mechanism of cerebellar hemorrhagic infarction is embolic from cardiac or proximal artery sources, similar to the mechanism in the anterior circulation (Chaves et al. 1996). Hemorrhagic transformation is more common in patients with larger infarcts, when the full cerebellar arterial territories are involved. This may be explained by the frequent association with extensive edema that can compress small vessels and induce endothelial damage. After edema reduction, reperfusion of the damaged vessels induces the leakage of blood (Moulin et al. 1993). Although hemorrhagic transformation is usually clinically silent because the bleeding occurs within an area of infarcted tissue, clinical deterioration may be noted, and death may occur (Pessin et al. 1992).

Cerebellar Hemorrhage

Cerebellar hemorrhage is another spectrum of cerebellar stroke. Patients with cerebellar hemorrhage are at higher risk of neurologic deterioration and mortality (St Louis et al. 1998). Spontaneous cerebellar hemorrhage occurs most frequently in the area of the dentate nucleus and can spread to involve most of a cerebellar hemisphere and occasionally cross the midline (Fig. 5). Not infrequently, they extend into cerebellar peduncles and rupture into the fourth ventricle (Heros 1982). Patients with cerebellar hemorrhage have similar clinical features to cerebellar infarction on initial examination, which are dependent on the cerebellar structures involved. However, cerebellar hemorrhage is more frequently associated with fatal complications than cerebellar infarction (St Louis et al. 1998). The differential diagnostic possibilities with deteriorating cerebellar hemorrhage are brainstem compression by direct mass effect and obstructive hydrocephalus from compression of the fourth ventricle. Alternatively, the hemorrhages can extend into the fourth ventricle and induce a hydrocephalus, even though the size of hemorrhage is small, if it involves the vermis (Jensen and St Louis 2005). In addition, the cerebral aqueduct may be obliterated as a secondary phenomenon accompanying upward herniation of cerebellar vermis through the tentorial notch. As with cerebellar infarction, the patients with cerebellar hemorrhage have a high risk of atherosclerosis such as hypertension. About two thirds of cerebellar hemorrhage patients have hypertension. However, it

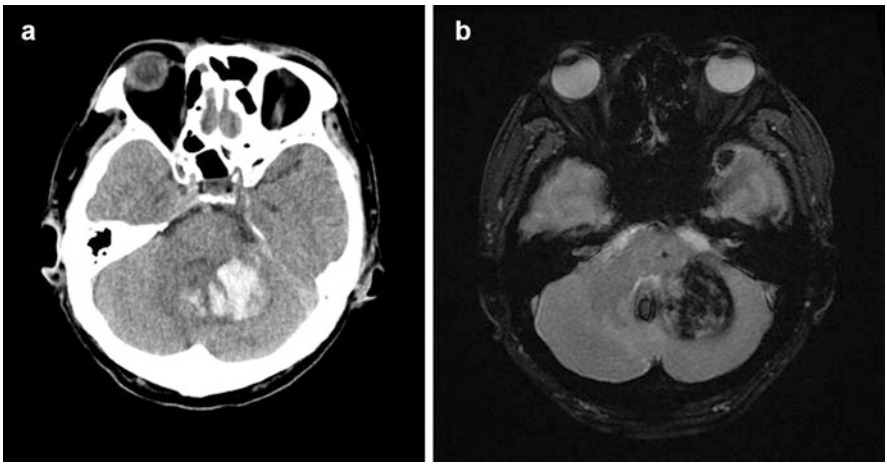

Fig. 5 Cerebellar hemorrhage. Brain computed tomography (**a**) shows hemorrhage in the left cerebellum. This lesion is demonstrated as low signal intensity on T2*-weighted gradient-echo imaging (**b**)

does not seem necessary for cerebellar hemorrhages to have very high levels of blood pressure. In the report by Terayama et al. (1997), the average blood pressure of cerebellar hemorrhage patients at the time of onset was about 125 mmHg in fatal cases and 115 mmHg in nonfatal cases, whereas the major cause of recurrent cerebellar hemorrhages was irregular control of blood pressure (Wu et al. 2010). Amyloid angiopathy is one of the causes of cerebellar hemorrhage, but its incidence is likely to be low, because cerebellar hemorrhage frequently occurs under the age of 60 (Itoh et al. 1993). In spite of uncertain mechanisms of cerebellar hemorrhage, it is important to maintain optimal blood pressure to prevent progression and recurrence. Several clinical and radiological factors have been reported to be associated with the outcome of cerebellar hemorrhage. These predictors include abnormal corneal and oculocephalic reflexes, a Glasgow Coma Scale (GCS) score less than 8, and systolic blood pressure greater than 200 mmHg (Kobayashi et al. 1994; Dahdaleh et al. 2012). Moreover, the CT scan features indicative of a poor prognosis are hematoma size greater than 3 cm in diameter, visible brainstem distortion, acute hydrocephalus, and the presence of intraventricular hemorrhage on hospital admission CT (Jensen and St Louis 2005). A widely accepted surgical intervention is to evacuate a cerebellar hemorrhage by suboccipital craniotomy.

Cerebellar Venous Infarction or Hemorrhage

Venous thrombosis of the posterior fossa is a rare condition that displays a wide-spectrum of clinical manifestations and prognosis (Ferro et al. 2004). The abundant collateral venous drainage of the posterior fossa prevents blood stasis in the posterior

structures and explains the rarity of isolated venous thrombosis in this area (Rothon 2000). The parenchymal finding most frequently associated with venous thrombosis of the posterior fossa is cerebellar venous infarction with or without a hemorrhagic component, but it can also manifest as a pure hemorrhage (Eng et al. 1990; Nayak et al. 1994; Ruiz-Sandoval et al. 2010). Venous infarction of the posterior fossa has been reported by CT or MRI in 3% of cases and parenchymal hemorrhage in 1.5% (Ferro et al. 2004). The most common sinuses affected are the straight and lateral sinuses followed by the superior petrosal vein (Eng et al. 1990; Nayak et al. 1994; Ruiz-Sandoval et al. 2010). However, the thrombosis of straight or lateral sinuses usually accompanies multiple lesions in the supratentorial structures (Damak et al. 2009). Imaging findings sometimes make the diagnostic confusing. A venous infarction resulting from venous thrombosis of the posterior fossa can present vasogenic edema and gadolinium enhancement (Ruiz-Sandoval et al. 2010) (Fig. 6). Thus, the differential diagnosis of cerebellar venous infarction or hemorrhage includes rapidly growing cerebellar tumors, which may also have an acute or subacute presentation with a vasogenic edema, profound mass effect, hemorrhage, and compression of brainstem and the fourth ventricle.

Diagnosis

Even if clinical differences are apparent among different vascular presentations, they are not usually critical from a diagnostic perspective, because the physician's first priority is to confirm that a cerebellar stroke has occurred. Moreover, the nonspecific manifestations frequently occur in inconsistent patterns and complicate the diagnosis. It is crucial to keep in mind that early identification and treatment of the underlying vascular lesions can prevent fatal complications and second stroke. The diagnostic confirmation of cerebellar stroke so far has been based on CT and MRI in cases of clinically suspected cerebellar stroke. The most commonly used brain imaging technique is CT, which provides images very quickly and accurately excludes acute hemorrhage. However, this technique is hampered by a low sensitivity for acute ischemic stroke within the first few hours, especially for infarctions in the posterior fossa (Mullins et al. 2002; Chalela et al. 2007). Large cerebellar infarctions present with displacement of the fourth ventricle, hydrocephalus, and obliteration of the basal cistern on CT (Hornig et al. 1994; Jauss et al. 1999), but the initial CT scan in patients who will develop mass effect is normal in one fourth of cases (Koh et al. 2000). Brain CT perfusion is helpful for the early identification of patients at risk for malignant cerebellar edema development in acute cerebellar stroke (Fabritius et al. 2017). MRI is a more sensitive method for visualization of affected cerebellar structures within the posterior fossa (Schmahmann et al. 2000) and makes it possible to establish the type of stroke, including small and large territorial infarcts, hemorrhages, and venous infarcts, and to correlate them with the clinical findings and outcome (Barth et al. 1993; Canaple and Bogousslavsky 1999). MRI becomes more sensitive when diffusion-weighted imaging (DWI) is added, having 80–95% sensitivity in the first 24 h (Mullins et al. 2002; Chalela et al. 2007).

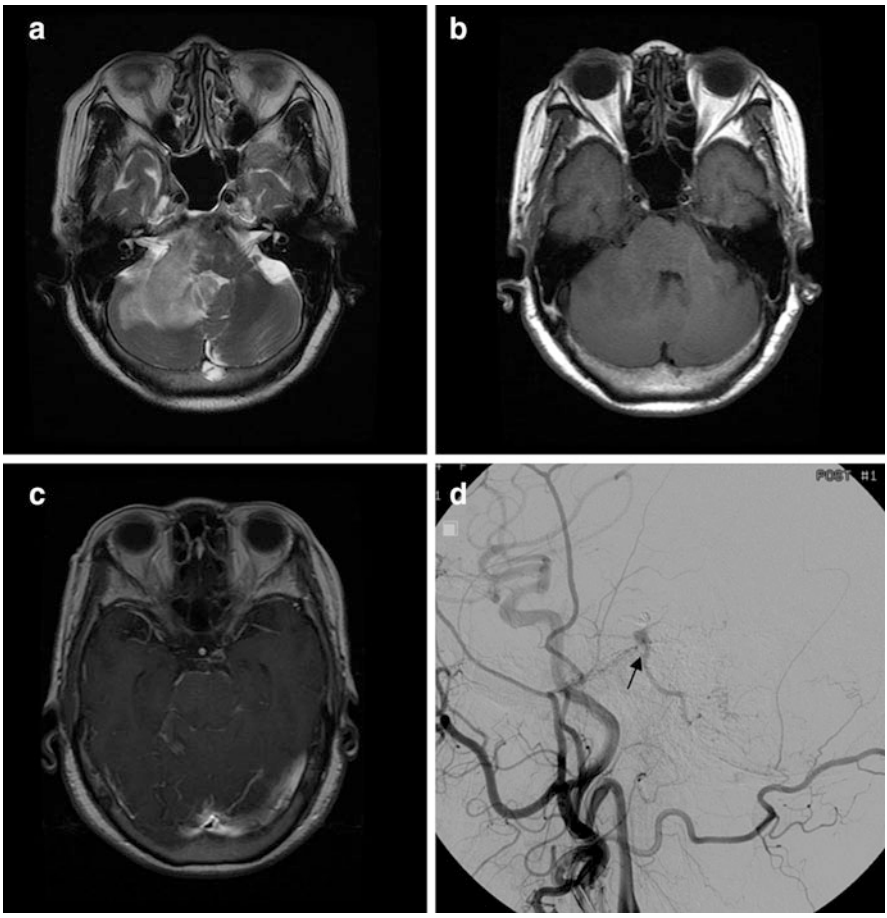

Fig. 6 Cerebellar venous infarction. MR images show high signal intensity on T2-weighted images (**a**) and low signal intensity on T1-weighted images (**b**) at right cerebellar hemisphere, cerebellar peduncle, and pons. Subtle peripheral enhancement on gadolinium-enhanced T1-weighted images (**c**) at lower pons and cerebellar peduncle is observed. Conventional angiography (**d**) confirms dural arteriovenous fistula draining into the right superior petrosal vein (*arrow*)

DWI can detect small ischemic lesions as early as in the first hour after the onset of symptoms, in contrast to the CT and conventional MRI. Moreover, multiple ischemic lesions may be detected in the early phase of stroke on DWI (Warach et al. 1992; Lutsep et al. 1997). A pattern of multiple acute brain infarcts on DWI is associated with arterial and cardiac sources of embolism and indicates a high risk of recurrent embolism or progression (Warach et al. 1992; Lutsep et al. 1997). Multiple lesions in DWI and MR angiography (MRA) can also identify the vascular origin of the emboli (Roh et al. 2000; Kang et al. 2000). Identification of the underlying vascular pathology is the next necessary step. The methods to evaluate cerebrovascular status include Doppler ultrasound, CT angiography (CTA), MRA, and conventional

angiography. Doppler ultrasound has advantages of early availability, monitoring, and low cost, whereas it is limited by a relatively poor accuracy in the posterior circulation as compared to the anterior circulation. CTA is known to be more sensitive than Doppler ultrasound and MRA in patients with vertebral artery pathology (Bash et al. 2005). However, a potentially toxic dye load and radiation limit the use of this technique. MRA overcomes this limitation, but it is less available to physicians and requires more time to acquire images compared with CTA. Conventional angiography can demonstrate the exact pathology of cerebellar vessels, but this technique has procedure-related complications, in addition to all the disadvantages of CTA. As in cases of other strokes, cardiac evaluations such as echocardiography and Holter monitoring are useful to identify a potential cardioembolic source.

Treatment

Since no randomized trials exist regarding patients with cerebellar infarction, treatment guidelines accord with information on acute ischemic stroke in general. Airway, breathing, and circulation should be maintained as first priorities. Arterial hypertension should be managed with different threshold pressures depending on the individual hemodynamic profile. Any patient whose blood pressure is greater than 220/120 mmHg should be treated with antihypertensive drugs, while, in the occasional patient with hemodynamically fluctuating symptoms, therapy to augment cerebral blood flow might be considered (Adams et al. 2007). Patients with cerebellar infarction can be treated with intravenous thrombolysis according to the NINDS criteria (1995; Köhrmann et al. 2009). However, randomized trials specific for cerebellar stroke are scarce, and most trials have generally excluded patients with cerebellar infarction. Furthermore, the majority of patients have been excluded owing to low National Institutes of Health Stroke Scale (NIHSS) scores. Nonetheless, thrombolysis might be equally beneficial for both territories, because embolic etiology is more frequently observed in cerebellar infarction (Tohgi et al. 1993). Intra-arterial thrombolysis has better recanalization rates with comparable outcome as compared to intravenous thrombolysis (Lindsberg and Mattle 2006). However, data for either approach are missing when it comes to isolated cerebellar strokes. Endovascular angioplasty, stenting, and stent-assisted coiling are being increasingly tried. These interventions have been effective in patients with vertebral artery dissections and extracranial and intracranial vertebral artery atherosclerosis, sometimes in combination with intra-arterial thrombolysis (Chow et al. 2005; Ahn et al. 2006; Dabus et al. 2006; Eberhardt et al. 2006), but all of these studies are nonrandomized trials. The recommended therapy in international guidelines is surgical intervention in case of a space-occupying infarction (Morgenstern et al. 2010). Surgical procedures include external ventricular drainage (EVD), suboccipital craniotomy, or some staged or concurrent combination of the two. Larger craniotomy size is associated with decreasing mortality in patients undergoing emergent posterior fossa decompression (Puffer et al. 2016). The clinical status of

a patient determines the treatment protocol. Craniotomy is considered to be essential in obtunded patients with clinical and neuroimaging signs of brainstem compression (Mathew et al. 1995). In patients in whom hydrocephalus coexists with altered consciousness, hydrocephalus should be treated with EVD first (Mathew et al. 1995; Jauss et al. 1999; Raco et al. 2003). However, EVD in patients with posterior fossa mass lesions may enhance the risk of upward transtentorial herniation (Kase and Wolf 1993). If consciousness does not improve, decompression of the posterior fossa should be urgently undertaken (Raco et al. 2003). A conversion from external to permanent internal drainage or the initial implantation of permanent shunt systems are currently being used in patients with cerebellar infarctions and associated hydrocephalus (Bertalanffy and De Vries 1992). Although there is widespread agreement that surgical intervention is required to salvage the majority of patients with brainstem compression, in the absence of randomized trials, patient selection criteria, type of surgery, and timing of the procedures are a matter of ongoing debate. When the patient exhibits brainstem sign such as conscious deterioration or respiratory change and eye signs, not confined to cerebellar dysfunction, surgical intervention is essential. The guidelines suggest that hemorrhages >3 cm in maximum diameter should be considered for early decompressive surgery even in the absence of hydrocephalus, brainstem compression, or clinical deterioration (Morgenstern et al. 2010). Corticosteroids are ineffective, and the effects of hyperventilation or osmotic diuretics are transient (Adams et al. 2007). Elevation of the head can improve venous drainage, but the effect is not dramatic. These measures should not delay a surgical approach. In contrast to patients with cerebellar infarction, the course of progressive clinical deterioration in cerebellar hemorrhage develops more rapidly (Mathew et al. 1995). Although the therapeutic guidelines of cerebellar hemorrhage also remain debatable, patients who have GCS scores of 14 or more, a small hematoma less than 4 cm, and no evidence of hydrocephalus are usually treated conservatively (Kirollos et al. 2001; Morioka et al. 2006). In contrast, the hematoma should be removed surgically as soon as possible in cases where the diameter of the hematoma is more than 4 cm, or its volume is more than 15 mL and in which there is brain stem compression or hydrocephalus. The findings of ongoing clinical trials highlight the importance of hypertension management and prompt treatment of coagulopathy (Elijovich et al. 2008). Adequate control of BP is likely to minimize the chances of hemorrhage rebleeding.

Conclusion and Future Directions

Stroke in the posterior circulation accounts for up to 15% ischemic strokes and is associated with high morbidity and mortality (Jauss et al. 1999). However, most studies for diagnosis and treatment are confined to the anterior circulation. For patients with spontaneous prolonged vertigo without associated neurologic symptoms or signs, a more refined protocol including clinical characteristics, physical examination, and imaging techniques to rule out cerebellar stroke should be applied in clinical practice. With improvements in hardware and software, a number of

noninvasive neuroimaging techniques are currently being tried. As documented before, most cerebellar infarcts are atheroembolic or cardioembolic. However, a significant portion of cases is still classified as stroke of undetermined cause in clinical practice. The vascular supply in the cerebellum may vary from patient to patient. Understanding of pathogenetic mechanisms by systematic topographical analysis in a large population should be achieved. Issues regarding the prediction of neurologic deterioration are also important in cerebellar stroke. A discriminating analysis of the clinical and radiological features of acute cerebellar stroke is needed to predict early outcome. In the absence of data from a randomized controlled trial, a reasonable framework for patient care remains inconclusive. Intravenous thrombolysis based on advanced imaging techniques, effectiveness of anticoagulation, and surgical option should be determined in further studies.

Cross-References

- ▶ Ataxic Hemiparesis
- ▶ Imaging Vascular Anatomy and Pathology of the Posterior Fossa
- ▶ Vascular Supply and Territories of the Cerebellum

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