Cerebellar Infarction

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Introduction/Epidemiology

The cerebellum is a structure that is located at the back of the brain, underlying the occipital and temporal lobes of the cerebral cortex. It accounts for approximately 10% of the brain's volume, but it contains more neurons than the rest of the brain. The cerebellum is involved in the maintenance of balance and posture, coordination of voluntary movements, motor learning, and cognitive functions.

Infarcts in the cerebellum are an uncommon localization, with a frequency of 2% [1] but a higher mortality than that of other vascular territories, which makes it important to diagnose in early stages.

As cerebellar infarction frequently manifests by nonspecific symptoms such as nausea, vomiting, dizziness, unsteadiness, and headache, its true frequency may be higher, as suggested by autopsy series [2] and MRI series [3].

Compared to hemorrhagic stroke, infarcts are three to four times more frequent in autopsy series [4] and in CT series [5]. There is a male preponderance of two to three times [4, 6]. The mean age is 65 ± 13 years, with one-half of the cases occurring between the ages of 60 and 80 years [7].

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Classification

Classically, cerebral infarcts are classified based on arterial territories (Table 6.1) as a function of the three long circumferential arteries arising from the vertebrobasilar system in a rostrocaudal disposition: the posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA), and superior cerebellar artery (SCA) (Fig. 6.1). The PICA and SCA are arterial pairs with medial branches that supply mostly the vermian and paravermian portions of the cerebellum, and lateral branches for the cerebellar hemispheres (Fig. 6.2).

Posterior Inferior Cerebellar Artery Infarcts

Infarcts in the PICA territory were extensively studied and once considered to be the most frequent of cerebellar infarcts, but further autopsy studies showed that SCA infarcts may be as or more frequent [2, 6, 8]. The overestimation was partly due to the erroneous consideration that all lateral medullary infarcts (i.e., Wallenberg's syndrome) were due to PICA occlusion. But as Miller Fisher showed, the lateral region of the medulla is mainly supplied by three or four small direct branches arising from the termination of the vertebral artery between the PICA ostium and origin of the basilar artery, and less frequently by small branches arising from the PICA, and in

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Table 6.1 Cerebellar stroke syndromes (Amarenco 1991, with permission)

Location of cerebellar infarct	Associated infarcts	Clinical syndrome
Rostral (SCA)	Mesencephallum, subthalamic area, thalamus, occipitotemporal lobes Laterotegmental area of the upper pons	Rostral basilar artery syndrome or coma from onset+/−tetraplegia Dysmetria and Horner's syndrome (ipsilateral), temperature and pain sensory loss, and IVth nerve palsy (contralateral) Dysarthria, headache, dizziness, vomiting, ataxia, and delayed coma (pseudotumoral form)
Dorsomedia (mSCA)		Dysarthria ataxia
Ventrolateral (lSCA)		Dysmetria, axial lateropulsion (ipsilateral), ataxia, and dysarthria
Medial (AICA)	Lateral area of the lower pons	VII, V, VIII, Horner's syndrome, dysmetria (ipsilateral), temperature and pain sensory loss (contralateral) Pure vestibular syndrome
Caudal (PICA)		Vertigo, headache, vomiting, ataxia, and delayed coma (pseudotumoural form)
Dorsomedial (mPICA)	Dorsolateromedullary area	Wallenberg's syndrome Isolated vertigo or vertigo with dysmetria and axial lateropulsion (ipsilateral) and ataxia
Ventrolateral (lPICA)		Vertigo, ipsilateral limb dysmetria AICA syndrome+/−delayed coma (pseudotumoral form)
Caudal and medial	Lateral area of the lower pons and/or lateromedullary area	Vertigo, vomiting, headache, ataxia, dysarthria, and delayed coma (pseudotumoral form)
Rostrocaudal	Brainstem, thalamus, occipitotemporal lobes	Coma from onset+/−tetraplegia

only up to 22% of individuals, this region is supplied by PICA [9].

The posterior inferior cerebellar artery is usually the largest branch of the vertebral artery, and it arises extracranially from its intradural segment, approximately 1.5 cm from the origin of the basilar artery. PICA may also be the termination of the vertebral artery, which, in this case, is smaller than the contralateral vertebral artery. After its origin, it reaches the caudal part of the cerebellar hemisphere and vermis.

It courses transversely and downward along the medulla, and then it makes a first caudal loop, ascending in the sulcus separating the dorsal medulla from the tonsil of the cerebellum. It then makes a second loop above the cranial part of the tonsil and descends, following the inferior vermis, where it divides into a medial branch (mPICA) and a lateral branch at a variable level between the two first loops [10, 11], On an axial mid-medullary and cerebellar section, the mPICA supplies a triangular area with a dorsal base and a ventral apex toward the fourth ventricle, first described in 1990 [12]. The medial branch of the PICA supplies the inferior vermis (nodulus, uvula, pyramis, tuber, and sometimes clivus) and the internal parts of the lobulus semilunaris inferior, lobulus gracilis, and tonsil; mPICA exists even when the PICA is hypoplasic. In this case, the lateral branch of the PICA arises from the anterior and inferior cerebellar artery [13] as there is a reciprocal relation between these two arteries. At times, the sole medial branch participates in the blood supply of the medulla [14] in its dorsal region, and sometimes in the lateral retro-olivary area [15]. This latter region is usually supplied by small short circumferential arteries arising from the vertebral artery [9, 16].

Infarcts of the medial branch may be clinically silent [6, 7, 17]. Its anatomo-clinical manifestations have been first described in 1990 [17] and present with three main patterns: (1) Wallenberg's

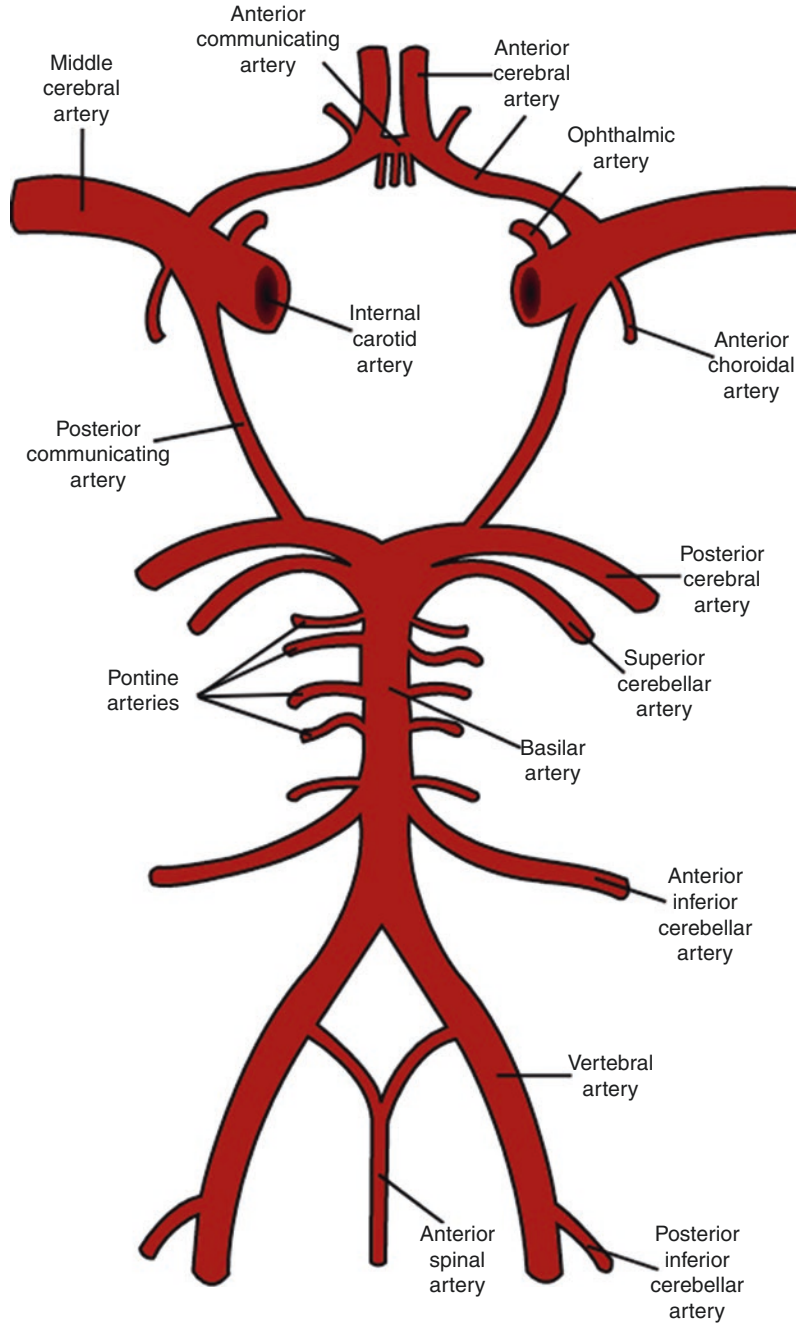

Fig. 6.1 Schematic representation of the basilar and vertebral arteries and their branches Gross anatomy

syndrome when the medulla is also involved, (2) vertigo together with ipsilateral axial lateropulsion of trunk and gaze, and dysmetria or unsteadiness (hence, cerebellar signs can be minimal, and MRI may be required for diagnosis), and (3) isolated vertigo often misdiagnosed as labyrinthitis [6, 7, 17].

1. When PICA includes the dorsal lateral part of the medulla, patients present with Wallenberg's syndrome that can be complete or not, including vertigo, nystagmus, Vth, IXth, and Xth cranial nerve palsies, ipsilateral Horner's syndrome, appendicular ataxia, and contralateral temperature and pain sensory loss (Fig. 6.3).

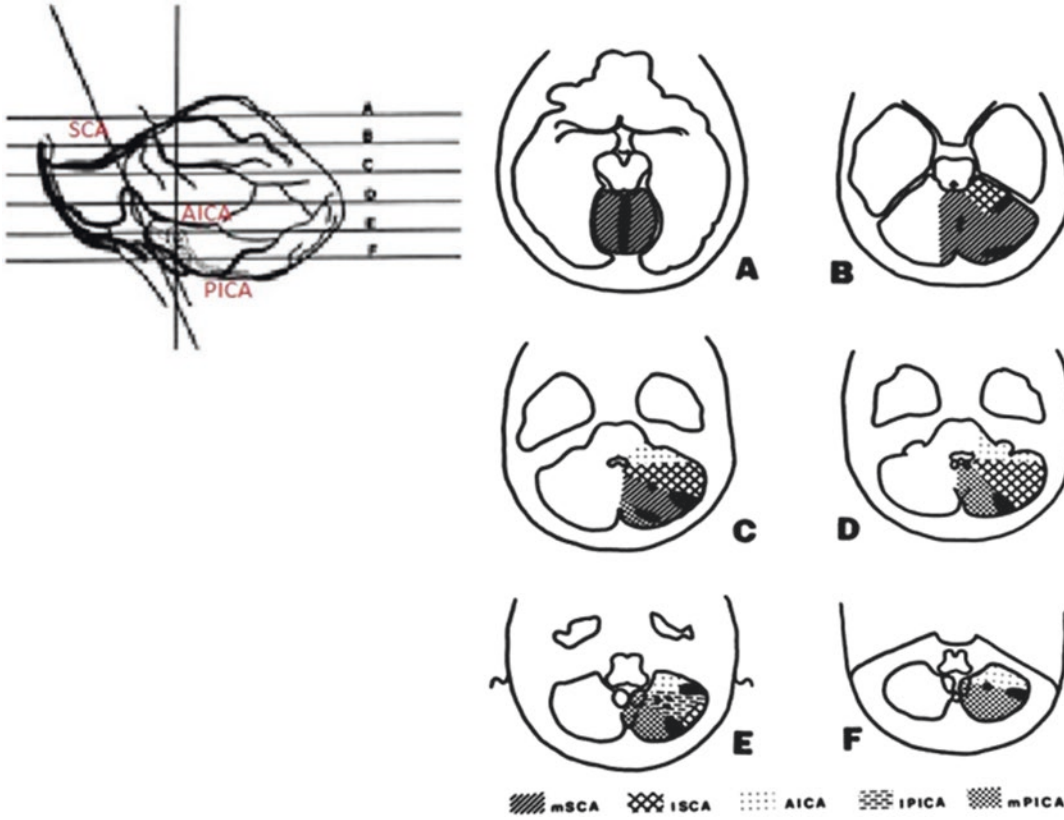

Fig. 6.2 Anatomical drawings of the territory of branches of the cerebellar arteries (modified from Amarenco et al., 1993b, with permission)

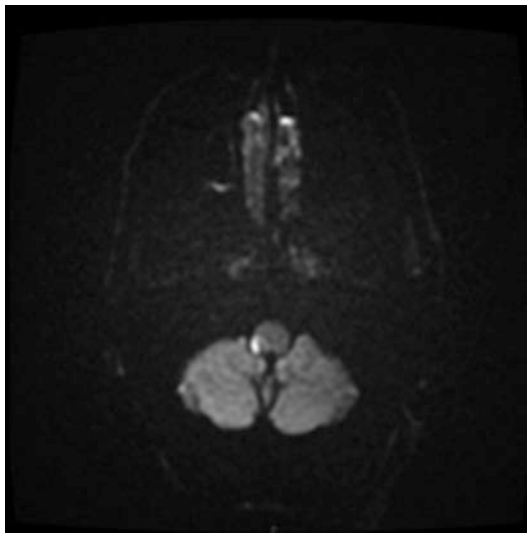

Fig. 6.3 A 70-year-old patient with dorsolateral medullar stroke

2. When PICA territory infarcts spare the medulla (Fig. 6.4), they mainly present with vertigo, headache, gait ataxia, appendicular ataxia, and horizontal nystagmus. Headache is cervical, occipital, or both plus occasional periauricular or hemifacial-ocular radiation. Unilateral headaches are ipsilateral to the cerebellar infarction [18]. Nystagmus is the most frequent sign (75%) either horizontal (ipsilateral in 47% of patients, contralateral in 5%, bilateral in 11%) or vertical (11% of patients) [19]. In addition to vertigo, one of the most striking findings in PICA infarcts is ipsilateral axial lateropulsion [17] as if there was a lateral projection of the central representation of the center of gravity. This sign is totally different from the lateral deviation of the limbs (i.e., past-pointing) and gait veering. Attempts at

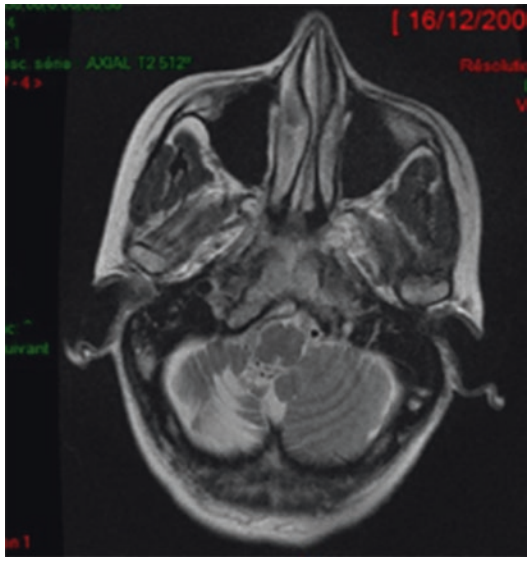

Fig. 6.4 Right medial PICA stroke in a 45-year-old patient

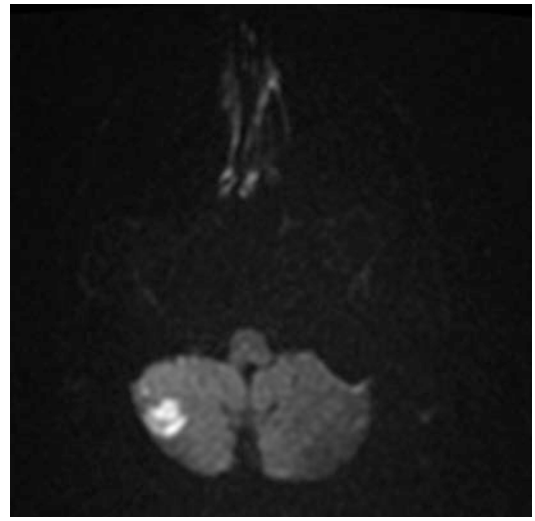

Fig. 6.5 Infarct in the lateral branch of the right PICA

standing or walking led to falling toward the side of the cerebellar infarction [18]. In one-quarter of patients, there may be signs of brainstem compression such as drowsiness and lateral gaze palsy, followed by progressive coma [18].

3. An isolated acute vertigo form, mimicking labyrinthitis [17], may be seen in patients with medial and caudal cerebellar infarct with involvement of the uvulonodular complex of the vermis, which is part of the vestibular portion of the cerebellum. The MRI has shown the high frequency of such infarcts [8], which should be done when there are vascular risk factors or in the circumstances supporting a vascular mechanism. Normal caloric responses and direction-changing nystagmus on gaze to each side, or after changing of head posture, or lying down, are two other signs that can suggest a "pure" vestibular syndrome in a patient with a PICA territory infarct [6, 20].

PICA infarcts may be in association with AICA or SCA infarcts with a more severe clinical presentation.

These multiple cerebellar infarcts, constituting about 20% of all cerebellar infarcts in autopsy series, often present with a pseudotumoral pattern or deep coma with quadraplegia [7].

The lateral branch of the posterior inferior cerebellar artery (lPICA) supplies the anterolateral region of the caudal part of the cerebellar hemisphere. Infarcts of the lateral branch of PICA (Fig. 6.5) are less frequent, initially described as chance autopsy findings with no available clinical information [6]. With time, clinical manifestations were described, mainly rotatory vertigo and isolated ipsilateral dysmetria or nystagmus [8].

The main cause of infarcts in the PICA territory is arterial occlusion, mainly involving the intracranial portion of the vertebral artery facing the PICA ostium and the origin of the PICA. The mechanisms of occlusion are equally divided into cardioembolic and atherosclerotic causes [6, 18], with maybe the predominance of atherosclerosis for infarcts of lPICA [8]. Other mechanisms are vertebral artery dissection [18], ulcerated plaques in the aortic arch [7], PFO (Fig. 6.6), and occlusion of the mPICA by tonsillar herniation due to raised posterior fossa pressure [21].

The evolution of infarcts in PICA territory is usually favorable with good outcome [18].

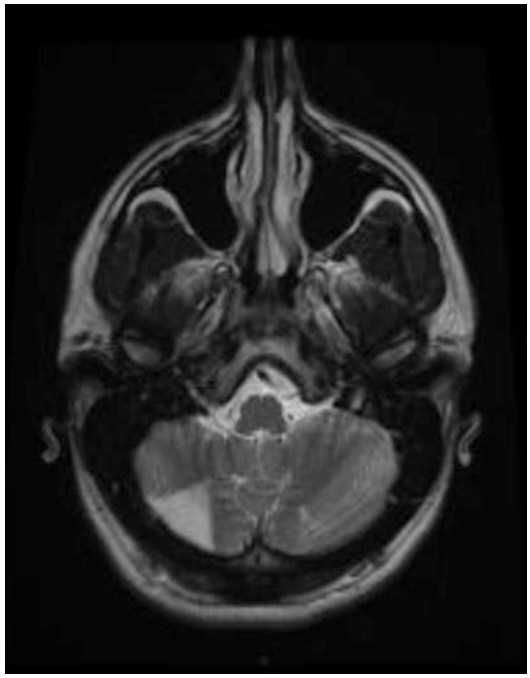

Fig. 6.6 Bilateral PICA infarcts in a 33-year-old patient with PFO

Anterior Inferior Cerebellar Artery Infarcts

The infarction in the territory of the anterior inferior cerebellar artery (AICA) is rare, but it is probably underestimated.

The anterior cerebellar artery is an almost constant artery. It usually arises from the basilar artery, from its lower third in 75% of cases. It can also arise from the vertebral artery or by a common trunk with the posterior inferior cerebellar artery (PICA) from the basilar artery. Infrequently, there are small arteries directly from the basilar artery that replace the AICA [13].

It supplies a small area of the anterior and medial cerebellum, the middle cerebellar peduncle, and the flocullus. Proximal branches of the AICA usually supply the lateral portion of the pons, including the facial, trigeminal, and vestibular nuclei, the root of the VIIth and VIIIth cranial nerves, and the spinothalamic tract.

The clinical presentation typically involves several cranial ipsilateral nerves: trigeminal sensory impairment, facial palsy, deafness, vestibular syndrome, or lateral gaze palsy. Frequently, we can find cerebellar signs, and, at times, there is contralateral pain and temperature sensory loss. This last characteristic and the other signs associated (like sometimes Horner's syndrome and the cranial nerve involvement with ataxia) may be confused with Wallenberg's syndrome due to lateral medullar infarction. Considering some signs that are unusual in Wallenberg's syndrome, such as deafness with or without tinnitus or lateral gaze palsy, may help in clinical differential diagnosis [13, 19].

Dysphagia can be due to an extension of the infarction to the superior part of the lateral medulla, and contralateral limb weakness can be observed when the corticospinal tract in the pons or mesencephalon is involved [22].

There may be an isolated vestibular manifestation but is rare. AICA infarcts can also cause isolated cerebellar signs.

The classic syndrome of AICA occlusion was described by Adams from a single neuropathologic case [23]. It involved vertigo, tinnitus, ipsilateral hearing loss, dysarthria, peripheral facial palsy, Horner's syndrome, multimodal facial hypoesthesia, and ipsilateral limb ataxia accompanied by contralateral thermanalgesia of the limbs and trunk [23]. The AICA syndromes were fully described in 1990 in the only large clinico-neuropathological series available [13].

The main cause is atherosclerotic occlusion. Pure AICA infarcts are usually due to basilar branch occlusion. Plaques in the basilar artery extent into AICA, or small atheroma occludes the AICA origin. We can also find arterial occlusion that involves the lower basilar artery and less frequently the end of the vertebral artery above the PICA ostium at postmortem examination [24]. Patients with "AICA plus" infarcts mainly have proximal basilar artery occlusion.

Nevertheless, atrial fibrillation should not be ruled out. Other less frequent etiologies such as vasculitis or dolichoectasia are also described (Fig. 6.7).

These patients usually present with vascular risk factors like high blood pressure or diabetes.

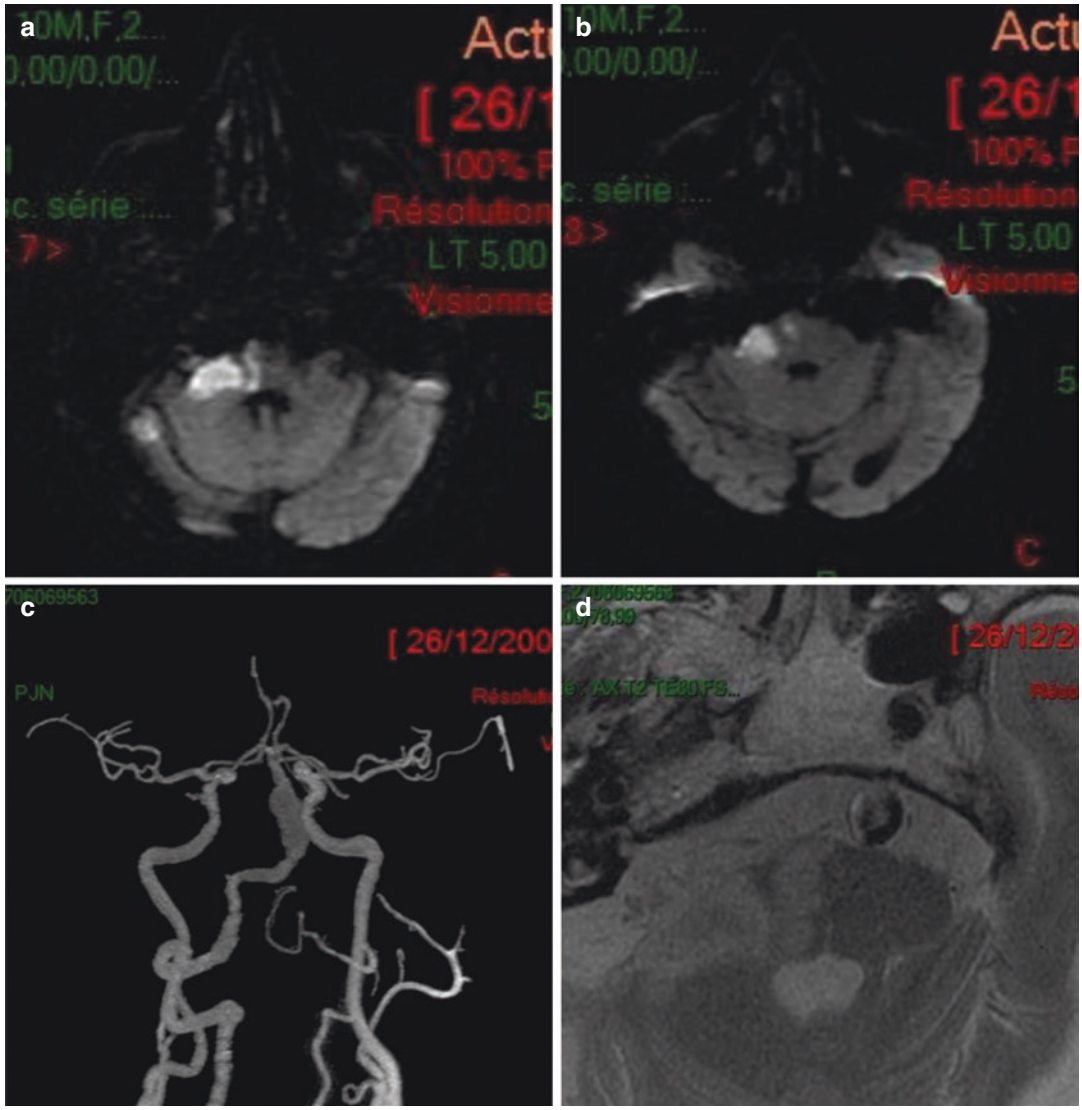

Fig. 6.7 An 85-year-old patient with AICA infarct (A, B) due to a dolichoectatic basilar artery (C). High-resolution MRI (D) showing a thrombus inside the basilar artery

Superior Cerebellar Artery Infarcts

Infarctions in the territory of the superior cerebellar artery (SCA) are among the most common of the cerebellar stroke syndromes.

The SCA supplies the rostral half of the cerebellar hemisphere and vermis as well as the dentate nucleus. This artery also vascularizes a small portion of the brainstem, the laterotegmental portion of the rostral pons, and lower midbrain.

Full clinicopathological description of SCA infarcts has been done in the largest clinical-neuropathological series available [25]. Infarcts in the full territory of the SCA are usually accompanied by other infarcts in the rostral territory of the basilar artery, involving uni- or bilateral occipitotemporal lobes, thalamic and subthalamic areas, and the mesencephalon [25].

The typical clinical features of the infarcts in the SCA territory are dysarthria and ipsilateral limb ataxia. Dysarthria can be useful for differen-

tiating from the PICA stroke [2]. Nystagmus is caused by involvement of the medial longitudinal fasciculus and the cerebellar pathways.

When the dorsal mesencephalic territory is involved, the clinical presentation may include Horner's syndrome, fourth nerve palsy, and contralateral temperature and pain sensory loss. Ipsilateral abnormal limb movements (choreiform or athetotic) can be associated. These features characterize the classic SCA syndrome as described by Guillain, Bertrand, and Péron, but it is rare to find [26].

Isolated occlusion of the lateral branch of the SCA was also described in 1991 [27, 28]. The lateral SCA syndrome includes ipsilateral limb dysmetria, ipsilateral axial lateropulsion, dysarthria, and gait unsteadiness. Similarly, an involvement of the medial branch of SCA can cause a dorsomedial SCA infarction with a clinical manifestation that includes unsteadiness of gait and dysarthria [27].

In the case of infarcts in the occipitotemporal lobes or in the thalamic or subthalamic areas, we can find other clinical signs such as hemianopsia, memory loss or confusion, Balint's syndrome, multimodal sensory loss, transcortical aphasia, and motor weakness.

There is sometimes a deep coma from onset, with or without quadriplegia when there is sudden occlusion of the basilar artery.

SCA infarcts may have a pseudotumoral presentation, especially if the territory of the PICA is also involved, with rapidly progressive cerebellar edema that leads to obstructive hydrocephalus and acute intracranial hypertension [1, 21].

In the SCA territory, more than one-half of infarcts are due to cardioembolism (with atrial fibrillation as the main cause) (Fig. 6.8).

Sometimes, the responsible stroke mechanism is artery-to-artery embolism either atherosclerotic, from vertebral artery occlusion, or ulcerated plaques in the aortic arch. There are also cases described with embolisms from vertebral artery dissection.

Multiple Infarcts

Initially described in autopsy series [2], the occurrence of multiple infarcts in the posterior circulation territory was further documented with the development of imaging techniques, such as CT and MRI. Multiple infarcts can appear in the cerebellum in different arterial territories, PICA and SCA or PICA, AICA, and SCA, or may be associated with ischemic lesions in the brain stem or other regions of the posterior circulation. Basilar occlusion or occlusion of the dominant

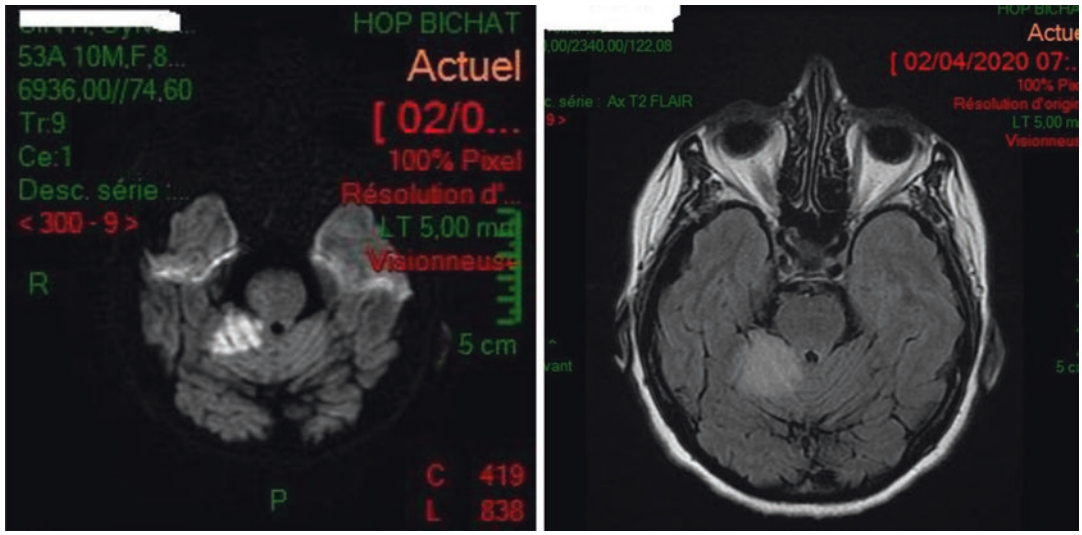

Fig. 6.8 Lateral SCA infarct in a 53-year-old woman with atrial fibrillation

vertebral artery may manifest in the same way with infarcts in multiple cerebellar territories and brainstem that can a have a severe course.

In the New England Medical Center Posterior Circulation Registry [29], the multiple territory infarcts were divided into two groups: proximal and distal intracranial territories (PICA and SCA), and those that included the middle intracranial territory (PICA and AICA, AICA and SCA, and PICA, AICA, and SCA). Embolism was the predominant stroke mechanism in patients with proximal and distal territory cerebellar infarction: emboli from the extracranial vertebral artery or cardiac sources like cardiomyopathy, atrial fibrillation, valvular disease, and PFO. In patients with emboli that arose from the extracranial vertebral artery (ECVA), the emboli presumably first stopped at the intracranial part of the vertebral artery (ICVA) and then it traveled distally, or a part to the SCA-distal basilar artery region.

In contrast to the proximal+distal territory, when the middle territory was involved, the most common cause was large artery intracranial occlusive disease. Embolism was a less common cause occurring in about one-third of patients. Basilar artery lesions are more often due to in situ occlusive disease of the basilar artery itself or to propagation of thrombus from the ICVA. ICVA and basilar artery occlusive disease often coexist.

Small Cerebellar Infarcts

With the MRI gaining its place in diagnosing cerebellar infarcts, very small cerebellar infarcts (<2 cm) are now a frequent finding and were first described in 1993 [30]. These small infarcts, also known as border-zone cerebellar infarcts, have yet unclear mechanism. They are located in boundary zones (or end zones), also called non-territorial infarcts [8], between the SCA and PICA or between left and right SCAs on the cortex [8, 30], and between SCA and PICA in the deep cerebellar white matter.

The location can be divided into three groups: cortical border-zone infarcts, very small deep infarcts in the deep watershed territory, usually limited to a small hole outside the dentate nucleus, cortical and superficial small infarcts along the boundary zone between cortical superficial branches of the SCA and PICA.

1. Cortical border-zone infarcts in a parallel direction with the penetrator branches, which are perpendicular to the cortex, are most frequent and located at the boundary zones between SCA and PICA territories, corresponding to the AICA-PICA, mPICA-lPICA, mPICA-SCA, and mSCA-lSCA border zones. Other border-zone infarcts involved the medial rostral cerebellum between the right and left SC [30].
2. Very small deep infarcts in the deep watershed territory [30] in a small hole outside the dentate nucleus: The infarcts involve usually the caudal cerebellum and are located at the deep boundary zones of the AICA, lPICA, mPICA, lSCA, and mSCA territories. These arteries supply the dentate nucleus area, and they anastomose with superficial branches penetrating the cortex perpendicularly.
3. Cortical dorsal border-zone infarcts between PICA and SCA: They are strictly cortical and superficially located along the boundary zone between cortical superficial branches of the SCA and PICA.

Small cerebellar infarcts were classified according to border zones in between perfusion territories, but a functional topographic classification according to an anatomical location in the cerebellum was also proposed [31]. Thus, they were classified according to their midline or hemispheric location in either the anterior, posterior, or flocculonodular lobe. Thus, they can be localized in the anterior or posterior vermis, in the nodulus, in the anterior or posterior hemisphere, or in the flocculus. A more precise classification in terms of affected lobule(s) was proposed for research purpose [31].

Border-zone infarcts do not differ clinically from territorial infarcts [8, 30]. Some patients may have transient loss of consciousness, postural trunk or head position-related symptoms for

days, weeks, months, or years before or after the infarct, light headedness, pitching sensations, vertigo, and disequilibrium, resulting from a low flow state in the posterior circulation.

The etiology also does not differ from territorial infarct [8, 30]. Small nonterritorial cerebellar infarcts have the same high rate of embolic mechanism (47%) with the same frequency of the cardiac source of embolism (42%) and of large artery occlusive disease (19%). They differ by the presence of more frequent low flow states distal to bilateral vertebral artery occlusion (14% in nonterritorial infarcts vs. 0% in territorial infarcts) and by the presence of more frequent hypercoagulable states resulting in end-artery disease (17% in nonterritorial infarcts vs. 1.25% in territorial infarcts) [8, 32].

Three circumstances can be distinguished.

1. Focal hypoperfusion distal to large artery occlusion is the most frequent mechanism [30]. It often involves the proximal basilar artery+/− AICA and sometimes a distal vertebral artery occlusion ipsilateral to the border-zone cerebellar infarct. The rostral basilar artery can be supplied by retrograde filling from the superior cerebellar arteries or posterior communicating arteries. Other cases are due to bilateral vertebral artery occlusion, either distal, or proximal on one side and distal on the other, and the lack of anastomoses causes the infarct in a border-zone area.
2. Small or end (pial) artery disease associated with primary or secondary hypercoagulable states, which are known to give border-zone infarcts: thrombocythemia, polycythemia, hypereosinophilia, and disseminated intravascular coagulation [8]. Arteritis and cholesterol emboli are occasionally encountered [8]. Other patients have severe intracranial distal atheroma with MRI showing multiple small cortical and deep infarcts of the cerebral hemispheres and angiography demonstrating multiple intracranial arterial stenoses and no extracranial atheroma.
3. Systemic hypotension due to cardiac arrest is seldom the cause of border-zone cerebellar infarcts as the cerebellum seems to be relatively protected from deep systemic hypotension [30].

The more recent SMART-Medea study [3] showed that small cerebellar infarcts predominantly involved the posterior lobes, sparing the subcortical white matter and occurring in characteristic topographic patterns. This could be explained by the distribution of the white matter in shape of a tree with branches of subcortical white matter and stem of deep white matter. These branches with the surrounding cortex form the cerebellar folia. These folia receive arterial supply from arterial branches in two fissures. This dual cortical arterial supply accounts for the subcortical white matter sparing of the observed cortical infarcts [33] in larger infarcts caused by a more proximal occlusion of a cerebellar artery. However, arterial branches may be occluded in both the fissure above and beneath the infarcted folium, leaving no collateral arterial supply [3] (Fig. 6.9).

Four patterns of small cerebellar infarct were described: infarcts occurring in the apex of a large (pattern 1) or a small fissure (pattern 2), infarcts occurring more superficially alongside one (pattern 3) or opposite sides (pattern 4) of a fissure, and infarcts bridging multiple fissures.

Lacunar Infarction

Lipohyalinosis has never been reported in the cerebellum in association with a stroke syndrome. Lacunes of vascular origin have been scarcely described in postmortem studies and rarely seen in radiologic studies [34]. The arterial anatomic disposition with progressively tapered arteries reaching the deep cerebellar white matter does not favor lacunar stroke [34]. Small deep infarcts with CT and MRI appearance of lacunae have been described in the watershed area between the SCA, PICA, and AICA and were associated with large artery occlusive disease, cardiac source of embolism, and end-artery disease, aortic arch atheroma, and intracranial atheroma.

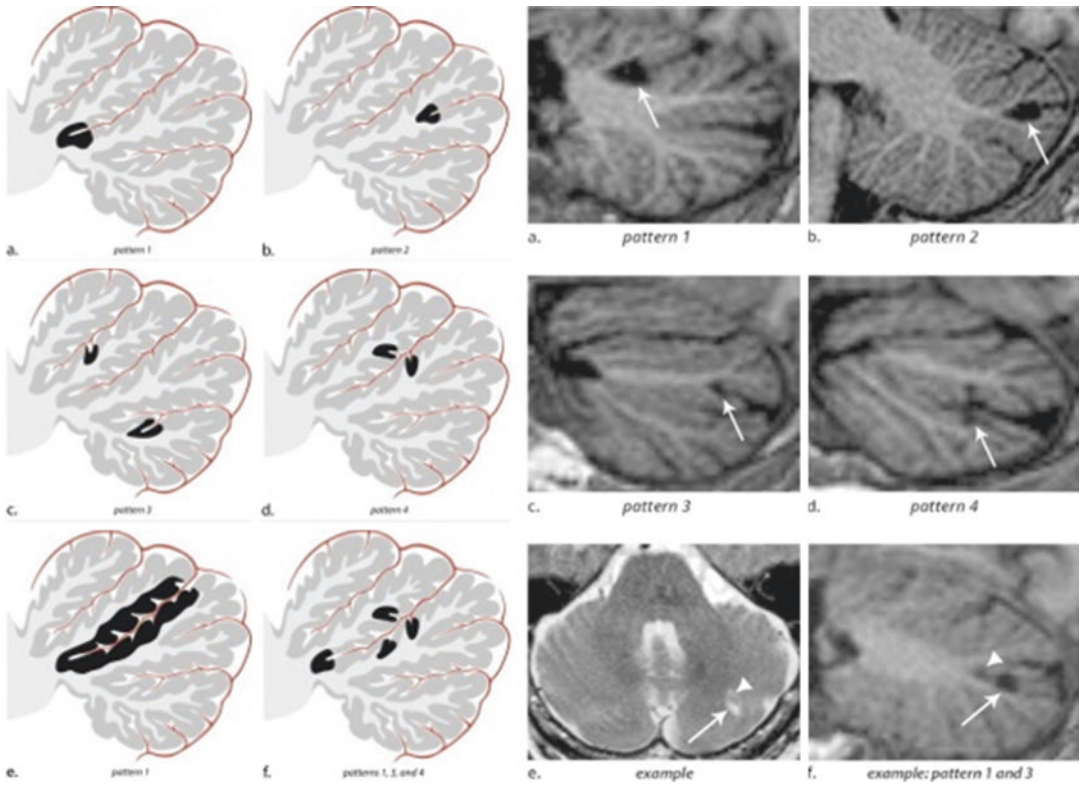

Fig. 6.9 Cerebellar infarct patterns: the SMART-Medea study (with permission)

Etiology of Cerebellar Infarcts

The causes and risk factors of cerebellar infarcts do not differ from other cerebral infarcts [2, 7, 8], and we can find large vessel atherosclerosis, cardioembolism, and arterial dissection as well as less frequent causes like hypercoagulable states or vasculitis [8].

Cardioembolic causes seem to be more frequent, and up to 54% of small nonterritorial infarcts as well as large territorial infarcts [8, 25] may be of cardiac origin. Infarcts in the SCA and PICA territory are more associated with a cardiac source, and up to 80% of SCA infarcts and 50% for PICA [8, 18, 25] come from cardiac causes such as atrial fibrillation, valvular disease, cardiomyopathy, PFO, or angiographic complication. Certain studies have shown that PFO was associated with strokes more often in the vertebrobasilar territory [35].

An atheromatous mechanism is described in 23–32% of infarcts, mainly in the AICA territory [2].

The histologic features do not differ qualitatively from atherosclerosis elsewhere, but ulceration in plaques is less frequent than in the anterior circulation [7, 36]. When ulceration is present, it affects the subclavian artery or the vertebral arteries in their proximal segments [7]. Ulcerated atherosclerotic plaques in the aortic arch can also be a source of arterio-arterial embolism and were first described as a cause of cerebellar infarcts [6, 7, 13, 21] before being described in ischemic stroke overall [37].

Atherosclerotic stenosis is common at the origin of the vertebral arteries and also in the intracranial portion of them (V4). Thrombus formed in V4 frequently extends into the proximal basilar artery [7, 38, 39].

For the basilar artery, stenosis is more frequently found in the proximal 2 cm of the vessel. At its distal end, the origin of the posterior cere-

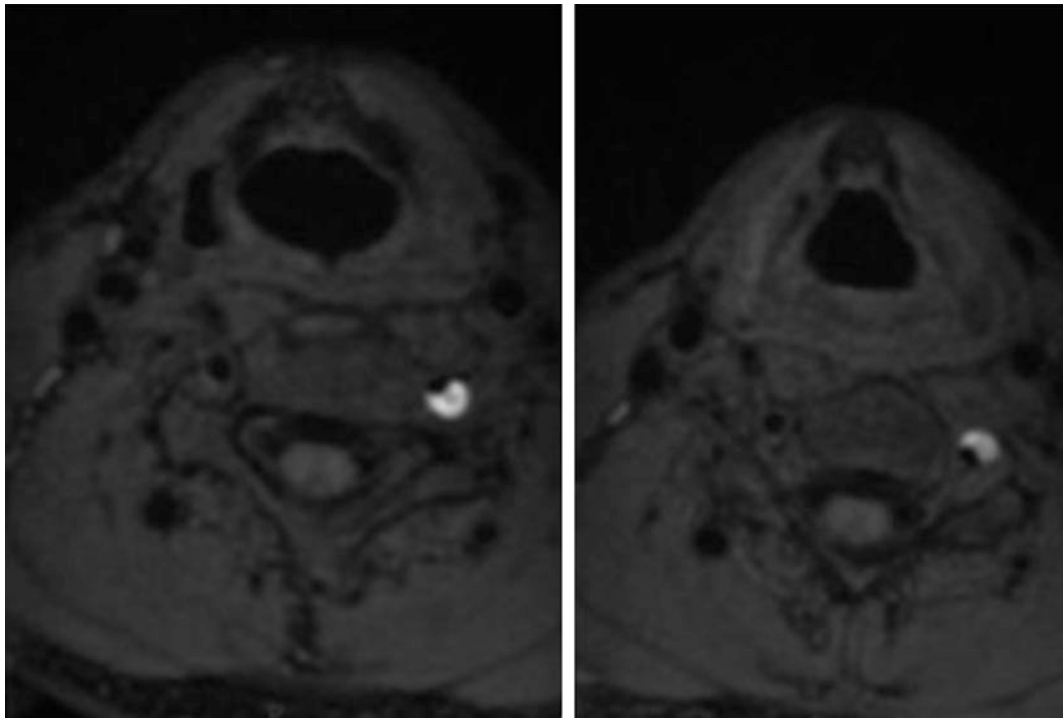

Fig. 6.10 Fat-saturated T1 axial images in MRI: left vertebral artery dissection (intramural hemorrhage seen as hyperintensity)

bral arteries is also a common site of atheromatous lesions [7].

Thrombi within the basilar artery frequently extend to the orifice of the next long circumferential cerebellar artery (the AICA or SCA) [7].

Atherosclerosis may also affect the branches of the vertebral and basilar arteries. AICA occlusions are atherothrombotic and in situ in most cases [6, 7, 13, 21].

Small artery disease can also be the cause of cerebellar infarcts. A process called lipohyalinosis is the responsible mechanism for this type of infarcts, which are generally small, usually less than one/two centimeters of diameter. This process is characterized by fibrinoid vessel wall necrosis and segmental arteriolar disorganization that can obliterate the lumen and leads to ischemia distal to the lesion. This same mechanism can produce hemorrhage due to the weakness of the wall. The small artery disease is usually associated with vascular risk factors such as high blood pressure or diabetes.

In the case of artery dissection, the vessel most commonly affected in the posterior circulation is the extracranial vertebral artery. It is important to evaluate all the arterial axes as we can find bilateral artery dissection or concomitant dissection of the internal carotid. The diagnosis is based on echographic, tomographic, and MRI findings. Fat-saturated T1 axial images in MRI are more sensitive at imaging intramural hemorrhage.

Vertebral artery dissection [8] should be especially considered in young patients with no known predisposing vascular risk factors for atherosclerosis or cerebral embolism, especially if there is neck pain, recent trauma, or neck manipulation, or in patients with Marfan's syndrome, Ehlers–Danlos syndrome (Fig. 6.10), systemic lupus erythematosus, fibromuscular dysplasia, or pseudoxanthoma elasticum.

Dissection of the basilar artery and its major branches is very uncommon but real with possible fatal complications (Fig. 6.11).

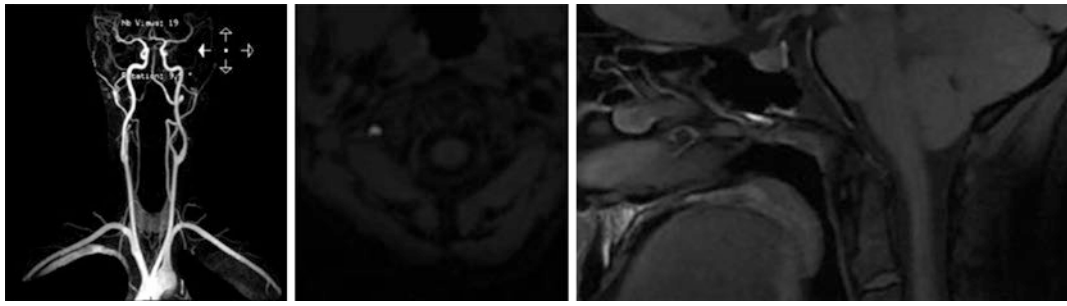

Fig. 6.11 Right vertebral artery dissection extending to the basilar artery in a 38-year-old patient with vertebrobasilar stroke due to Ehlers–Danlos syndrome

Less common causes include hypercoagulable states (like antiphospholipid syndrome) or vasculitis (Wegener more frequently) [30].

In 20–30% of patients, the etiology remains undetermined.

Treatment

The acute management of cerebellar infarction is similar to the management of the rest of strokes.

The fibrinolysis with rTPA iv has proven its efficacy within the 4 or 5 h of symptom onset in selected patients (after evaluating radiological and clinical information, absence of contraindications) [40].

In the case of basilar artery occlusion, mechanical thrombectomy within 6 h of symptom onset in carefully selected patients may be indicated, but its benefits have not been proven yet (there are clinical trials ongoing). Nevertheless, the clinical data are in favor of this procedure [40], also given the high mortality and complications of this condition. There are experts who considered that the therapeutic window could be extended up to 8–12 h after onset (always considering the clinical features and radiological findings).

During the surveillance after a cerebellar infarction, if clinical deterioration occurs (decreasing level of consciousness, new oculomotor signs, etc.), it is indicated to repeat brain imaging to distinguish brainstem ischemia from secondary brainstem compression or hydrocephalus [40].

Suboccipital decompressive craniectomy should be considered in the case of pronounced edema with brainstem compression. The decompressive craniectomy may or may not be combined with resection of the necrotic tissue.

External ventricular drainage should be considered if an obstructive hydrocephalus occurs [40] (Fig. 6.12). Hydrocephalus usually occurs in the first 48–72 h, although it can occur any time within the first week.

The secondary stroke prevention with antiplatelet/anticoagulant agents and control of the risk factors is the same as the treatment for the ischemic stroke in the anterior circulation and depends on the cause and the identified vascular risk factors.

The optimal time to start oral anticoagulation in acute cardioembolic infarction is uncertain. However, it is probably between 4 and 14 days after stroke onset, depending on the balance between the risk of recurrent stroke and the risk of hemorrhagic transformation of the infarcted brain.

Dual antiplatelet therapy might be indicated for 3 months if there is significant aortic arch atheroma (>4 mm) or intracranial stenosis. Dual antiplatelet therapy can also be administrated during the first weeks after the stroke, especially if the cause is atherosclerosis (currently there are ongoing trials). The individual risk of bleeding and the potential benefits before starting should be evaluated.

In the case of artery dissection, anticoagulation has no proven better efficacy than antiplatelet therapy [41], but it can be used within the first

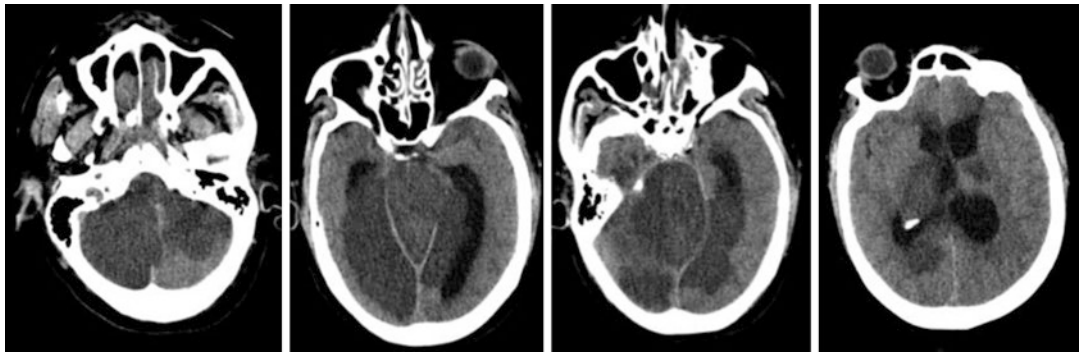

Fig. 6.12 Ventricular drainage was performed in a patient with multiple, bilateral infarcts in the cerebellum and posterior cerebral artery territories associated with ventricular hydrocephalus

6–12 weeks. Some authors recommend anticoagulation if there is an arterial occlusion or severe stenosis. After a few weeks, antiplatelet or anticoagulant therapy can be stopped if *"restitution ad integrum"* is achieved or the occlusion persists, and it is unclear whether it should be continued if in the follow-up imaging there are aneurysms or remaining stenosis. Intravenous thrombolysis is not contraindicated in this case, only in the case of subarachnoid hemorrhage, which also contraindicates the anticoagulation.

In the case of discovery of a patent foramen ovale with no other stroke etiology found, if it is associated with atrial septal aneurysm or large interatrial shunt, in patients up to 60 years, the closure of the foramen ovale can be proposed [42].

Prognosis

Patients with cerebellar stroke usually have a good prognosis and good recovery, more frequent than that of stroke in the anterior circulation. Excellent recovery with a little or no assistance, as evaluated by the Functional Independence Measure, may be obtained in over 80% of survivors [43]. In most series, more than two-thirds of patients have Rankin scores consistent with independence at 3 months.

Nevertheless, in the early phase, cerebellar infarction has greater fatality rate than any other location of brain infarction, and significant morbidity, due to rapidly progressing cerebellar edema with acute hydrocephalus, brainstem compression, and death.

The rapid diagnosis and the surveillance in stroke units with an early recognition (using a combination of clinical and radiological findings) of patients eligible for surgical decompression may improve the outcome. About half of the patients who progress to coma and who are treated with decompressive craniectomy have good outcomes (modified Rankin scale score ≤ 2) [44].

We can distinguish different groups of patients according to their clinical course [1]:

- Patients whose conscious state remains unimpaired.
- Some patients suffer a sudden deterioration of consciousness, within the first few hours, usually due to extension of the ischemic process to the brainstem.
- Patients with delayed alteration of consciousness, from a few hours to 10 days, due to compressive edema. Decompressive surgery is needed when deterioration of consciousness appears. Total recovery is obtained in 63% of published cases after ventricular drainage or opening of the dura mater by suboccipital craniectomy, but prognosis depends on whether there is an associated brainstem infarct or not [21].

As regards functional outcome, the modified Rankin Score should be preferred to the NIHSS, which is a score mainly oriented for anterior cir-

culation strokes, since patients with a low NIHSS may have disability. Worse outcome occurs for lesions >20 cc, and there is also an association with the development of hydrocephalus and brainstem compression. The outcome is also worse if there is more than one arterial territory affected.

Regarding the arterial territory, functional disability occurred most frequently in those with SCA infarcts compared with those with lesions in other single artery regions [21, 45].

Several studies have reported that a reduced level of consciousness at the initial presentation is strongly correlated with poor outcome. On the other hand, the presenting syndrome of vertigo/vomiting/ataxia/headache is correlated with a better functional recovery (probably related with an isolated cerebellar involvement) [2].

An activation of contralateral cerebellar and neocortical areas may explain a good recovery of the territorial infarcts [46].

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