Cerebellar Infarcts: Key Features

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Cerebellar infarctions often go unrecognized and misdiagnosed. Easily confused with peripheral vestibular system dysfunction, physicians often miss the cardinal symptoms of dizziness and an abnormal gait. If not treated appropriately and quickly, cerebellar infarcts can lead to coma and death. This review discusses the key features of cerebellar infarction, including the anatomical origination and clinical symptomology of the infarcts. Evaluation recommendations include neuroimaging analysis, which can help clarify the etiology and aid in making therapeutic decisions. Management of patients with cerebellar infarcts is similar to that of patients with posterior circulation ischemia. Antithrombotic drugs, thrombolytics, surgery, and angioplasty/stenting are options.

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Why then are cerebellar infarcts so important to recognize and treat appropriately?

Large cerebellar infarcts often become edematous. Within the relatively small confines of the posterior cranial fossa, the edematous cerebellum can compress the IVth ventricle and the brainstem, leading to coma and death if not treated appropriately and quickly. Cerebellar infarcts, like cerebral infarcts, are often caused by serious cardiac and cerebrovascular disease that poses a threat for further serious and even fatal brain infarction. Often caused by occlusive vertebrobasilar disease, further brainstem infarction is an important consideration.

Cerebellar infarcts often go unrecognized and misdiagnosed. They are easily confused with peripheral vestibular system dysfunction. Emergency room physicians, family doctors, and internists usually do not test for the cardinal sign—abnormal gait. I personally have never seen a cerebellar infarct diagnosed correctly by a non-neurologist unless a brain image yielded the correct diagnosis. Herein I attempt to review concisely the key features of cerebellar infarction.

Anatomy
The cerebellum is supplied by 3 main paired arteries: the posterior inferior cerebellar arteries (PICAs), the anterior inferior cerebellar arteries (AICAs), and the superior cerebellar arteries (SCAs), each arising from a different portion of the posterior circulation1,2 (Figure 1). The PICAs originate from the intracranial vertebral arteries (ICVAs) and divide into medial (mPICA) and lateral (lPICA) branches.

The medial branch supplies mostly the inferior cerebellar vermis and the nodulus and gives rise to a small branch that supplies a portion of the dorsal medullary tegmentum. The lateral branches supply the posterior inferior portion of the cerebellar hemispheres on each side. The AICAs originate from the lower half of the basilar artery and supply the lateral pontine tegmentum and base, the brachium pontis, the flocculus, and the anterior inferior portion of the cerebellum. The internal auditory arteries that supply the VIIIth nerve and the cochlea and vestibule most often arise as branches of the AICAs. At times either the anterior or posterior inferior cerebellar arteries are absent or hypoplastic, in which case one AICA-PICA supplies the usual territory of both arteries.

The SCAs originate from near the terminal end of the basilar artery, give off branches to the superior pontine tegmentum, and branch into a medial (mSCA) branch to the superior vermis and the medial portion of the superior surface of the cerebellum. The lateral (lSCA) branches supply the lateral portion of the superior surface of the cerebellum.

Posterior circulation brain regions can be conveniently divided into 3 territories in relation to the major arterial supply: the proximal, middle, and distal intracranial posterior circulation territories (Figure 2).2-5 The proximal intracranial posterior circulation territory includes regions supplied by the ICVAs—the medulla oblongata and the PICA-supplied region of the cerebellum. The middle intracranial posterior circulation territory includes the portion of the brain supplied by the basilar artery up to its SCA branches—the pons and the...
AICA-supplied portions of the cerebellum. The distal intracranial posterior circulation territory includes all of the territory supplied by the rostral basilar artery and its SCA, posterior cerebral artery (PCA), and penetrating artery branches—midbrain, thalamus, SCA-supplied cerebellum, and the occipital and temporal lobe regions supplied by the PCAs.

**Clinical Findings, Underlying Vascular Lesions, and Stroke Mechanisms**

A feeling of being off balance or dizzy, and abnormal gait are the 2 cardinal symptoms of cerebellar ischemia. Dysarthria, limb incoordination, and nystagmus are the second most common findings. Symptoms and signs vary depending on the location and size of the cerebellar infarction and the presence of ischemia to other posterior circulation structures.

**PICA Territory Cerebellar Infarction**

The most common symptoms are vertigo and gait and trunk imbalance. Headache and vomiting are also common. Nystagmus, gait ataxia, and limb incoordination are the most frequent neurological signs. In some patients with pseudotumoral infarcts, severe headache, vomiting, and decreased alertness develop 24 to 72 hours after onset.

Infarction of the inferior cerebellum and vermis involves 2 major systems: vestibular input to the nodulus and the medial portion of the cerebellar vermis, and interruption of proprioceptive (medial lemniscus) and spino- and vestibulo-cerebellar afferent pathways to the cerebellum and its nuclei. As a result, the 2 main dysfunctions relate to vestibular function, limb and trunk balance, and tone. Figures 3 and 4 show computed tomography (CT) and magnetic resonance imaging (MRI) examples of PICA territory cerebellar infarcts.

Vestibular symptoms are prominent especially if the mPICA territory is involved. Infarcts limited to the mPICA territory can present as purely vestibular syndromes. The patients describe vertigo as if the environment is spinning and rotating and nystagmus is prominent. Vomiting often ensues. Patients find it very difficult to walk. Some patients with mPICA territory infarcts have ipsilateral lateropulsion and lean and veer toward the side of infarction, making standing and often sitting difficult. Attempts at walking are impeded by sudden veering toward the ipsilateral side. Sometimes gaze lateropulsion is associated in which the eyes are driven to the ipsilateral side.

When the lateral portion of the inferior posterior cerebellum is infarcted (IPICA territory) the abnormalities are those of trunk and gait imbalance and hypotonia of the ipsilateral arm and leg. Patients may have difficulty sitting up and when they do sit they frequently lean to the side or backward. They often require support to remain sitting up-right. Similarly, attempting to stand is difficult. They may lean to one side as if their trunk was tilted. Upon trying to walk, they will often veer or lurch to one side or the other and frequently need help.
The limb abnormalities are best shown by asking patients to rapidly drop their outstretched arms toward their laps, stopping short before they hit. Then ask them to rapidly raise their outstretched arms upward, again stopping quickly. The arm on the side of the cerebellar infarct often lags behind the contralateral arm in the initial movement and overshoots when attempting to check the arm’s descent or ascent. In contrast, finger to nose and toe to object movements may be preserved. Vertigo, nystagmus, and dysarthria are not common in patients with infarcts restricted to lPICA territory. The signs can be missed if the patients are not watched when they stand and walk.

The most frequent cause of PICA territory cerebellar infarction is embolism, and the most frequent signs and symptoms of such infarcts are:

- Vertigo
- Vomiting
- Inability to walk
- Veering or leaning to one side when standing or walking
- Nystagmus.

Large infarcts, and those limited to the branch territories—mPICA or lPICA—are particularly likely to be embolic. The source of the emboli can be the heart, aorta, or the vertebral arteries in the neck. Atherosclerotic occlusive disease at or near the origins of the vertebral arteries in the neck and vertebral artery dissections are frequent sources of embolism in patients with PICA territory cerebellar infarcts. Angiography and autopsy studies show that the most common occlusive lesions in patients with PICA territory cerebellar infarcts involve the ICVAs before their PICA branches. Many of the occlusions are embolic.

Lateral medullary infarcts accompany PICA territory cerebellar infarcts in about one fifth of patients. Although the syndrome of lateral medullary infarction was originally considered by Wallenberg to be caused by PICA occlusion, anatomical studies have shown that the lateral medulla is supplied by penetrating arteries that arise from the vertebral artery and course through the lateral medullary fossa before penetrating into the lateral tegmentum of the medulla. PICA only supplies a very small portion of the dorsal medulla through its mPICA branch.

Lateral medullary infarctions are usually caused by atherosclerotic occlusive disease or dissections of the ICVA, whereas PICA territory cerebellar infarcts are more often caused by embolism to the ICVA. Lateral medullary and PICA cerebellar infarcts coexist when the ICVA occlusive disease is long and encompasses both the orifices of the penetrating branches to the lateral medulla and PICA. ICVA intrinsic occlusive disease may cause small cerebellar infarcts.

**AICA Territory Infarcts**

The AICAs are the most variable of the long circumferential cerebellar arteries and they have the smallest zone of supply within the cerebellum. The AICAs always supply the lateral pontine tegmentum, the brachium pontis, and the flocculus. As a result, in contrast to other cerebellar artery territory lesions, the infarcts always involve the brainstem and are virtually never limited to the cerebellum itself. Figure 5 shows an AICA territory infarct at necropsy and Figure 6 shows AICA territory infarcts on MRI.

When infarction is limited to unilateral AICA territory, the clinical findings are identical to those found in patients with lateral medullary infarction except that VIIth and VIIIth nerve findings are present rather than symptoms and signs related to Xth nerve (nucleus ambiguus) dysfunction. Table 1 outlines the...
major clinical findings of AICA territory infarction. The ischemic region may include the facial, vestibular, and cochlear nuclei or may affect the VIIth nerve fibers within the lateral tegmentum and base, or affect the VIIIth nerve peripheral fibers or the cochlea and vestibule. Weakness of the contralateral limbs and an extensor plantar sign occur when the infarct extends to the pontine base.

The internal auditory artery is most often a branch of the AICA, explaining the involvement of the VIIIth nerve and the cochlea and vestibular structures in the inner ear. In some patients, especially diabetics, ischemia of the inner ear structures supplied by the internal auditory artery can precede and herald a full AICA territory infarct.38 Tinnitus, hearing loss, and vertigo are the most common symptoms related to ischemia involving the inner ear.

Patients with AICA territory infarcts can be divided into 2 groups: those with infarcts limited to the territory of the AICA on one side, and those with infarcts that involve bilateral AICA territory or unilateral AICA territory + other brainstem and cerebellar vascular supply territories AICA + infarcts.37 When infarction is limited to unilateral AICA territory the cause is invariably intrinsic disease of the AICA, a form of intracranial atheromatous branch disease.39,40 The great majority of such patients have diabetes, and vascular imaging shows a normal or irregular basilar artery without stenosis. When infarction extends beyond unilateral AICA territory, occlusive disease within the basilar artery or bilateral ICVAs is the cause.

SCA Territory Cerebellar Infarcts
Isolated SCA territory infarcts are not as common as isolated PICA territory infarcts. Most often SCA territory infarcts are accompanied by other infarcts in regions supplied by other arteries that arise at the rostral end of the basilar artery. The symptoms and signs in patients with partial SCA territory infarcts are less severe and disabling than those in other cerebellar artery territories, and can be easily overlooked clinically. The signs and symptoms of SCA territory infarction include:

- Slight dizziness
- Dysarthria
- Limb incoordination and dysmetria
- Gait ataxia with veering.

Early reports described the so-called classic SCA syndrome that develops when the rostral pontine tegmentum and a portion of the midbrain are infarcted as well as the superior aspect of the cerebellum. The syndrome was said to consist of ipsilateral limb ataxia; ipsilateral Horner’s syndrome; contralateral loss of pain and temperature sensibility of the face, arm, leg, and trunk; and a contralateral IVth nerve palsy.2,41-44 Abnormal ipsilateral spontaneous involuntary movements also occur.43 The classic syndrome is quite rare. Figure 7 shows typical unilateral SCA territory infarcts on MRI.

Some patients with SCA territory cerebellar infarcts have relatively minor symptoms of sudden onset.

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Table 1
Symptoms and Signs of AICA Territory Infarction

<table>
<thead>
<tr>
<th>Ipsilateral</th>
<th>Contralateral</th>
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</thead>
<tbody>
<tr>
<td>Loss of pain and temperature and occasionally touch - Vth nerve</td>
<td>Dissociation (pain and temperature loss only) or complete loss of sensitivity, body and limbs</td>
</tr>
<tr>
<td>Horner’s syndrome</td>
<td>Hemiparesis</td>
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<tr>
<td>Loss of hearing</td>
<td></td>
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<tr>
<td>Vertigo</td>
<td></td>
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<tr>
<td>Peripheral facial nerve palsy</td>
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<tr>
<td>Cerebellar ataxia</td>
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AICA, anterior inferior cerebellar artery.
Cerebellar Infarcts continued

with rather rapid improvement. Slight dizziness, vomiting, ipsilateral limb dysmetria and ataxia, gait ataxia, and dysarthria are the most common findings. Vertigo is usually not prominent in patients with isolated SCA territory infarcts. Nystagmus is also much less common than in patients with PICA territory infarcts. Limb incoordination, limb ataxia, intention tremor, and dysarthria are more common in SCA territory cerebellar infarcts than in either AICA or PICA territory cerebellar infarcts.\(^2\) The lateral cerebellar hemispheres are related predominantly to limb movements under voluntary control whereas the vermis relates more to posture, stance, truncal movements, and gait.

The dentate nucleus is the major outflow tract of the cerebellum for modulating voluntary limb movements. The dentate nucleus and surrounding cerebellar white matter are supplied by SCA branches. SCA territory infarcts may involve the superior vermis and a large portion of the hemispheres in addition to the dentate nuclei. This explains the frequent occurrence of both limb and gait ataxia in patients with SCA territory infarcts.

SCA territory infarcts can involve the entire SCA territory including the rostral pontine and midbrain tegmentum and the SCA supplied cerebellum. They can involve the full cerebellar territory but spare the brainstem, or be limited to territory supplied by the medial (mSCA) or lateral (lSCA) branches. Dysarthria is a prominent symptom, especially when the left mSCA territory is involved. In one study, the paraverbal zone of the left cerebellum was the most frequent site of damage among 31 patients with focal lesions who had ataxia of speech.\(^{45}\) Speech abnormalities are an integral sign in many patients with SCA territory infarcts, no matter whether the lesion involves the full territory of the medial or lateral branches.

The great majority of SCA territory infarcts are embolic. Emboli may arise from the heart, aorta, or extracranial vertebral arteries (ECVAs) or ICVAs. When the bilateral SCA territories are involved (Figure 8), the cause is either embolism to the rostral basilar artery or intrinsic atherosclerotic stenosis of the distal basilar artery.

Combined PICA and SCA Territory Infarcts
Infarction in some patients is limited to or includes PICA territory cerebellum on one side and SCA territory on either side. This infarct pattern is important because virtually all cases are explained by one of 2 mechanisms: intrinsic disease of one ICVA causing local PICA territory infarct with artery-to-artery embolism to the SCA, or an embolus arising from the heart, aorta, or ECVAs that stops at the ICVA, causing local infarction and then moving distally to block an SCA.\(^2,4,5\)

Pseudotumoral Cerebellar Infarcts
The syndrome of pseudotumoral cerebellar infarction is most often found after large full PICA territory infarcts. It is also common when multiple cerebellar artery territory infarcts are present and can develop in full SCA territory cerebellar infarcts. After the first day or so, patients develop increased headache, vomiting, and decreased consciousness.\(^2,4,6-48\) At first they become drowsy and later stuporous. Bilateral Babinski signs are an early indication of cerebellar mass effect.

Most characteristic of large cerebellar space-taking infarcts are the oculomotor abnormalities that...
develop. Sometimes the pupil on the side of the infarction is smaller than the contralateral pupil. Most common are a conjugate gaze paresis to the side of the lesion or a paresis of abduction limited to the ipsilateral eye. Bilateral VIth nerve weakness may occur. Later bilateral horizontal gaze palsy can develop, often accompanied by ocular bobbing. These signs are due to compression of the pontine tegmentum by the swollen cerebellar infarct.

The IVth ventricle can also be compressed, leading to obstructive hydrocephalus further increasing intracranial pressure. The cerebellar tonsils can be pushed through the foramen magnum, compressing the rostral spinal cord. Stupor is followed by deep coma when the oculomotor abnormalities become bilateral. Once coma develops the mortality rate is very high. Brain imaging confirms compression of the posterior fossa cisterns and the IVth ventricle and the development of hydrocephalus.49-51 Figure 9 shows an example of pseudotumoral cerebellar infarcts. Ventricular drainage or removal of necrotic cerebellar tissue can be life saving if performed early enough.2,49-51

Cerebellar Infarction Related to Hypotension and Cardiac Arrest

Little information is available about hypoxic-ischemic cerebellar injury that results from cardiac arrest and hypotension. The lesions usually involve border-zone regions between the cerebellar artery territories and between the medial and lateral branches of PICA and SCA territories.52 Figure 10 shows a CT scan of a patient with bilateral but asymmetric border-zone infarcts in the cerebellum and cerebral cortex after a cardiac arrest. Most often the cerebellar injury is very much overshadowed by the cerebral injuries. Occasionally, patients have prominent ataxia after cardiac arrest or shock. The so-called Lance-Adams syndrome of intention myoclonus is well known after hypoxic-ischemic injury.53 This may represent selective injury to Purkinje and other cerebellar neurons.

Evaluation

The history and examination are very important. The patient should be asked to sit unsupported at the side of the bed or gurney. Standing balance and gait are essential to test. A neurological patient is not completely examined unless gait is tested. The tone in the arms should be tested. Have the patient rapidly descend their outstretched arms towards their laps stopping short before they hit. Then ask them to rapidly raise their outstretched arms upward, again stopping quickly. Finger to nose and toe to object testing is also useful.

The introduction of CT scanning into clinical neurology in the mid-1970s, and MRI a decade later, entirely changed the recognition of cerebellar infarction. These brain imaging modalities now make it possible to localize the cerebellar infarcts to the territory of one or more of the cerebellar supply arteries and to recognize small cerebellar infarcts that were not pseudotumoral.54-57 Figure 11 shows the location of various cerebellar artery territories as shown on CT. Both CT and MRI also make it possible to recognize the development of increased pressure within the posterior fossa and intracranially and to study brainstem and ventricular compression.49 However, CT scans, especially early
Cerebellar Infarcts continued

generation scanners, often do not show acute cerebellar infarcts during the first 6 hours after symptom onset. Neuroimaging is necessary to recognize pseudotumoral infarcts and to identify coexistent brainstem infarction. MRI is superior to CT for this purpose. An important sign of pressure, obliteration of posterior fossa cisterns, may be easier to determine on CT scans. Midline shifts, compression or obliteration of the IVth ventricle, and hydrocephalus are readily detected on both CT and MRI scans. Upward and downward cerebellar herniation is best shown on midsagittal MRI sections.

Especially helpful are T2-weighted sagittal sections because they optimally show the localization of the cerebellar infarcts as well as vertical shifts and herniations. Figure 12 shows various cerebellar-related herniations. Vascular diagnostic studies such as magnetic resonance angiography, transcranial Doppler, CT angiography, and catheter dye contrast angiography are very helpful in clarifying the etiology of the infarcts and in making therapeutic decisions.

**Management**

Because pseudotumoral cerebellar infarcts can be fatal if not treated effectively, patients with sizable cerebellar infarcts should be watched carefully by trained and experienced neurological nurses and clinicians, preferably in intensive care units. Any hint of decreasing level of consciousness, new oculomotor signs, or Babinski responses should trigger repeat brain imaging. The optimal treatment of patients with pseudotumoral infarcts is still unsettled. Corticosteroids and osmotic agents as well as hyperventilation are useful in decreasing cerebellar edema in patients with slight mass effect.

It is generally agreed that ventricular drainage should be used in patients who have hydrocephalus and have deteriorated into the second stage of decreased level of consciousness and/or exhibit new brainstem signs. Decompression of the cerebellum with removal of necrotic tissue has been advocated as the initial definitive approach in patients who become comatose. Occasionally after ventricular drainage, signs of so-called “upward transtentorial herniation” develop in which the swollen cerebellum moves upward through the tentorial notch and compresses the midbrain. Many neurosurgeons perform ventricular

Figure 11. Illustration showing CT scan cuts and localization of the various cerebellar artery zones. CT, computed tomography; mSCA, medial branch of superior cerebellar artery; LSCA, lateral branch of superior cerebellar artery; AICA, anterior inferior cerebellar artery; mPICA, medial branch of posterior inferior cerebellar artery; LPICA, lateral branch of posterior inferior cerebellar artery.

Figure 12. Sketch showing various cerebellar herniations. Drawing by Dr. Juan Sanchez-Ramos and used with permission.
drainage watching carefully for signs of deterioration; if such occur, they then perform a craniotomy to remove dead cerebellar tissue.

Other than monitoring for cerebellar edema and increased posterior fossa and intracranial pressure, the management of patients with cerebellar infarcts is the same as for patients with posterior circulation ischemia. Treatment depends on the causative stroke mechanism and the location, nature, and severity of any cardiac-cranio-cerebral occlusive vascular-hematological lesions.

Thrombolysis has only been well studied in patients with basilar artery occlusion and most studies are of intra-arterial administration.\textsuperscript{59} Thrombolysis should also be effective in patients with acute ICA\textsubscript{V} and top-of-the-basilar embolic occlusions if performed before the development of major infarction.

Antiplatelet drugs should be effective in preventing platelet-fibrin white clot emboli in patients with irregular vascular lesions that are not severely stenotic. Heparins, warfarin, and ximelagatran are used in patients with situations that promote red erythrocyte-fibrin thromboemboli, such as atrial fibrillation. Surgery and angioplasty and stenting have been used in patients with severe stenosis of the EC\textsubscript{V}As, and angioplasty and stenting have been used in patients with severe intracranial stenotic lesions. To date there have been no randomized controlled trials of any treatment in patients with known posterior circulation lesions.

**Main Points**

- Relatively uncommon when compared to cerebral infarcts, cerebellar infarctions are important to recognize because they often become edematous and can lead to coma and death if not treated appropriately and quickly.
- Cerebellar infarcts often go misdiagnosed and are easily confused with peripheral vestibular system dysfunction. The 2 cardinal symptoms of cerebellar ischemia are a feeling of being off balance or dizzy and abnormal gait.
- Common symptoms of posterior inferior cerebellar artery (PICA) infarctions are vertigo and gait and trunk imbalance. Headache and vomiting are also common. Nystagmus, gait ataxia, and limb incoordination are the most frequent neurological signs.
- Patients with superior cerebellar artery (SCA) infarcts have relatively minor symptoms of sudden onset with rather rapid improvement. Slight dizziness, vomiting, ipsilateral limb dysmetria and ataxia, gait ataxia, and dysarthria are the most common findings. Vertigo is usually not prominent in patients with isolated SCA territory infarcts and nystagmus is much less common than in patients with PICA territory infarcts.
- Pseudotumoral cerebellar infarction is most often found after large full PICA territory infarcts. Patients develop increased headache, vomiting, and decreased consciousness. These infarcts are most often characterized by the oculomotor abnormalities that develop.
- Brain imaging modalities now make it possible to localize cerebellar infarcts to the territory of 1 or more of the cerebellar supply arteries. Neuroimaging is necessary for recognition of pseudotumoral infarcts and identification of coexistent brainstem infarction. Vascular diagnostic studies also help in clarifying etiology and in making therapeutic decisions.
- With the exception of monitoring for cerebellar edema and increased posterior fossa and intracranial pressure, the management of patients with cerebellar infarcts is the same as for patients with posterior circulation ischemia. Treatment depends on the causative stroke mechanism and the location, nature, and severity of any cardiac-cranio-cerebral occlusive vascular-hematological lesions.

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Cerebellar Infarcts continued


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