Brief review in cerebellar stroke

Diagnosis and Management of Acute Cerebellar Infarction

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Outline

• Introduction
• General anatomy
• Function
• Clinical manifestation
• Neurological examination
• Management
• Take-home points
Introduction

• This paucity of data might be partly because the clinical presentation of cerebellar infarction is diverse
• The main symptoms were dizziness, nausea and vomiting, gait instability, headache
• Coordination, gait, and eye movements help to identify cerebellar stroke
• Early edema from infarction in the posterior fossa can result in potentially deaths
Epidemiology

- Ischemic > hemorrhagic
- In nine studies of consecutive ischemic strokes, cerebellar infarction accounted for almost 3% (660 of 23,426) of stroke
- The average age of patients is about 65 years
- Two-thirds of patients are men
- However, they may still be underdiagnosed by the CT scan cause of some limitations

General anatomy

- The largest structure in the posterior fossa
- Consists of a midline **vermis** and two large **cerebellar hemispheres**
- **Primary fissure** separates the cerebellum into an **anterior lobe** and a **posterior lobe**
- **Posterolateral fissure** separates the posterior lobe from the **flocculonodular lobe**, a region with important connections to the vestibular nuclei
- Gyri, the small ridges that run from medial to lateral on the surface of the cerebellum are called **folia**
Function

- Three functional regions, from medial to lateral, based on their input and output connections
- The superior parts of the cerebellum are primarily concerned with limb (by lateral hemispheres)
- Trunk (midline vermis) movements and motor control of Speech articulation (by paravermal area)
• Inferior areas are primarily associated with oculomotor control and vestibular adaptation
• Appendicular motor deficits related to unilateral cerebellar lesions tend to be ipsilesional
# Cerebellar specific function

## Table 18.2 - Subdivisions of the Cerebellum by Specific Function

<table>
<thead>
<tr>
<th>Subdivision</th>
<th>Topography</th>
<th>Function</th>
<th>Clinical Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Motor</strong></td>
<td>Anterior lobe</td>
<td>Gait, posture</td>
<td>Ataxia</td>
</tr>
<tr>
<td></td>
<td>Posterior lobe</td>
<td>Multijoint movement</td>
<td>Dysarthria</td>
</tr>
<tr>
<td><strong>Oculomotor</strong></td>
<td>Flocculus, parafocculus, and nodulus</td>
<td>Control of vestibular ocular reflex</td>
<td>Impaired pursuit</td>
</tr>
<tr>
<td></td>
<td>Dorsal vermis and fastigial nucleus</td>
<td>and smooth pursuit</td>
<td>Gaze-evoked nystagmus</td>
</tr>
<tr>
<td><strong>Cognitive</strong></td>
<td>Posterior lobe</td>
<td>Control of saccades</td>
<td>Saccadic dysmetria</td>
</tr>
<tr>
<td></td>
<td>Dentate</td>
<td></td>
<td>Opsoclonus</td>
</tr>
<tr>
<td><strong>Affective (limbic)</strong></td>
<td>Posterior lobe (vermal)</td>
<td>Executive function</td>
<td>Impaired executive function, visuospatial disorientation, verbal fluency, and visual memory</td>
</tr>
<tr>
<td></td>
<td>Fastigial nucleus</td>
<td>Visuospatial</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Language</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Memory</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Emotion, affect</td>
<td>Blunted or disinhibited behavior</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Autonomic</td>
<td></td>
</tr>
</tbody>
</table>
Regulation of muscle tone, coordination of skilled voluntary movement

Planning of voluntary activity

Maintenance of balance, control of eye movements
Blood supply

- Blood supply from three paired arteries
  - The posterior inferior cerebellar artery (PICA)
  - The anterior inferior cerebellar artery (AICA)
  - The superior cerebellar artery (SCA)
• The cerebellum and brainstem locate within the tightly constrained posterior fossa
• It bounded above by a rigid dural reflection brainstem
• Co-incident brainstem signs are common in patients with cerebellar stroke
• Variations of normal vascular anatomy are common
<table>
<thead>
<tr>
<th>Typical origin</th>
<th>Posterior Inferior cerebellar artery</th>
<th>Anterior Inferior cerebellar artery</th>
<th>Superior cerebellar artery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Vertebral artery</td>
<td>Proximal or mid-basilar artery</td>
<td>Distal basilar artery</td>
</tr>
<tr>
<td>Major branches</td>
<td>Medial branch, lateral branch</td>
<td>Cerebellar branches, internal auditory artery</td>
<td>Medial branch, lateral branch</td>
</tr>
<tr>
<td>Key brainstem structures supplied by proximal branches</td>
<td>Posterolateral medulla: cranial nerve nuclei (V, VIII [vestibular], IX, X) and fascicles (IX, X); sympathetic tract; spinothalamic tract; inferior cerebellar peduncle</td>
<td>Posterolateral pons: cranial nerve nuclei (V, VII, VIII [vestibular, cochlear]) and fascicles (VII, VIII); sympathetic tract; spinothalamic tract; middle cerebellar peduncle</td>
<td>Posterolateral midbrain (and upper lateral pons): cranial nerve nuclei (IV*, V) and fascicle (IV*); sympathetic tract; spinothalamic tract; medial lemniscus; superior cerebellar peduncle</td>
</tr>
<tr>
<td>Cerebellar and distal structures supplied by major branches</td>
<td>Postero-inferior cerebellum, including: inferior vermis (including uvula, nodulus); paraflocculus</td>
<td>Antero-inferior cerebellum, including: flocculus. Inner ear: vestibular labyrinth; cochlea</td>
<td>Superior cerebellum, including: superior vermis; dentate nucleus</td>
</tr>
<tr>
<td>Core cerebellar syndrome</td>
<td>Isolated acute vestibular syndrome without auditory symptoms (pseudo-vestibular neuritis)</td>
<td>Isolated acute vestibular syndrome with auditory symptoms (pseudo-labyrinthitis)</td>
<td>Acute gait or trunk instability with associated dysarthria (pseudo-intoxication); nausea or vomiting (pseudo-gastroenteritis)</td>
</tr>
<tr>
<td>Indicative neurological signs</td>
<td>Lateral medullary syndrome: hemifacial analgesia†; unilateral absent gag reflex; palatal palsy; vocal cord palsy; Horner’s syndrome; body hemianalgesia†; limb hemiataxia; dysmetria</td>
<td>Lateral pontine syndrome: hemifacial sensory loss; facial palsy (lower motor neuron type); Horner’s syndrome; body hemianalgesia†; limb hemiataxia; dysmetria</td>
<td>Lateral midbrain syndrome: fourth nerve palsy*; hemifacial sensory loss; Horner’s syndrome; body hemisensory loss; limb hemiataxia; dysmetria</td>
</tr>
<tr>
<td></td>
<td>Vertebral artery syndrome: 12th nerve palsy; body hemisensory loss; hemiplegia or quadriplegia</td>
<td>Mid-basilar syndrome: impaired arousal or coma; sixth nerve palsy or internuclear ophthalmoplegia; horizontal gaze palsy; body hemisensory loss; hemiplegia or quadriplegia</td>
<td>Top of the basilar syndrome: impaired memory or attention; visual field cut; ptosis; third nerve palsy; vertical gaze palsy; hemiplegia or quadriplegia</td>
</tr>
</tbody>
</table>

Data from [2,4,34,55]. * Note that because the fourth nerve fascicle crosses before exiting the brainstem posteriorly, a superior oblique palsy can occur either ipsilateral (fascicular post-decussation) or contralateral (nuclear, fascicular pre-decussation) to the cerebellar infarction. † Analgesia (loss of sharp sensitivity) and thermanaesthesia (loss of temperature sensitivity) typically cluster together.

Table 1: Typical arterial supply of the cerebellum and associated clinical vascular syndromes
Pathogenesis

• Two most common causes of cerebellar infarction are cardioembolism and large vessel atherosclerosis.

• Who were aged less than 40 years and had cerebellar stroke, patent foramen ovale is an important consideration.

• Vertebral artery dissection is another important cause of cerebellar infarction, particularly in younger patients.

• Don’t forget to recognised major or minor head or neck trauma, including chiropractic manipulations

• “Beauty parlour stroke\(^1\)” due to prolonged neck hyperextension

• Less common disorders include hypercoagulable states, vasculitis, CVST, acute marijuana or cocaine

• Overall, PICA strokes are more common than SCA, and AICA strokes are the least common

Clinical manifestations

Clinical presentations of isolated cerebellar infarction are similar the three main cerebellar vascular areas

Neurological signs might be absent, subtle, or difficult to distinguish from benign disorders of the peripheral vestibular system

Patients typically only experience non-specific symptoms ie. dizziness, nausea, vomiting, unsteady gait, and headache

*Lancet Neurol* 2008; 7: 951–64
Clinical manifestations

- Ataxia
- Tremor
- Headache
- Dysmetria
- Dysdiadokokinesia
- Dysarthria/scanning speech
- Nystagmus
- Hypotonia
- Vertigo and unsteadiness
<table>
<thead>
<tr>
<th>Clinical finding</th>
<th>n (%)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prior event</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of posterior circulation TIA</td>
<td>65 of 295 (22)</td>
<td>TIA suggests the need for rapid work-up and treatment, as with anterior circulation TIA</td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dizziness or vertigo</td>
<td>404 of 557 (73)</td>
<td>Whether the patient specifically describes vertigo rather than dizziness does not alter the likelihood of stroke</td>
</tr>
<tr>
<td>Nausea or vomiting</td>
<td>298 of 557 (54)</td>
<td>Nausea or vomiting can occur without dizziness and can sometimes be posturally provoked</td>
</tr>
<tr>
<td>Gait disturbance</td>
<td>186 of 389 (48)</td>
<td>The inability to walk independently suggests a central rather than a peripheral cause</td>
</tr>
<tr>
<td>Headache</td>
<td>207 of 557 (37)</td>
<td>Location and quality of headache are not diagnostic; abrupt onset can mimic haemorrhage; head or neck pain in younger patients should prompt consideration of vertebral dissection</td>
</tr>
<tr>
<td>Slurred speech</td>
<td>122 of 417 (29)</td>
<td>Slurred speech is more commonly the result of anterior circulation strokes than posterior circulation strokes; as a symptom, slurred speech must be distinguished from partial aphasia</td>
</tr>
<tr>
<td><strong>Signs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Limb ataxia</td>
<td>298 of 513 (58)</td>
<td>Limb ataxia (clumsy, wavering, dysynergic movements) and dysmetria (pastpointing or mismeasured reaching) cluster together clinically and are coded together</td>
</tr>
<tr>
<td>Truncal ataxia</td>
<td>263 of 513 (51)</td>
<td>Truncal ataxia is typically assessed with the patient seated at the bedside (or in an armless chair) and arms folded</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>204 of 447 (46)</td>
<td>Anterior circulation strokes might be more likely to produce labial (facial) dysarthria, and posterior circulation strokes more likely to produce lingual and guttural dysarthria; excludes a peripheral cause in a patient with dizziness</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>226 of 513 (44)</td>
<td>Nystagmus that is direction-changing or vertical strongly suggests a central rather than a peripheral cause</td>
</tr>
<tr>
<td>Confusion or somnolence</td>
<td>116 of 447 (26)</td>
<td>Altered mental status is more common in strokes of the superior cerebellar artery, perhaps because of its association with top-of-the-basilar ischaemia that extends to the paramedian thalamus and medial temporal lobes</td>
</tr>
<tr>
<td>Coma</td>
<td>14 of 447 (3)</td>
<td>Frank coma typically suggests either mid-basilar occlusion or the onset of secondary complications (direct brainstem compression or obstructive hydrocephalus with herniation)</td>
</tr>
</tbody>
</table>

Numbers were tabulated from several studies. Patients’ data were pooled independent of infarct areas. When data were provided in sufficient detail that enabled the distinction of patients with pure cerebellar stroke from those with brainstem association, the latter patients were excluded. Therefore, the denominators are not the same for each category. However, some of these numbers might have included some patients who had associated brainstem infarction. TIA—transient ischaemic attack.

**Table 2:** Frequency of common published clinical findings of cerebellar infarction listed in order of descending frequency.
Neurological examination

• Ataxia
  • Limb : cerebellar hemisphere lesion
  • Truncal : cerebellar vermis lesion
  • Gait : wide base gait

• Ocular symptom
  • Nystagmus, broken pursuit, hypometric saccade, ocular dysmetria

P. Amarenco, Cerebellar stroke syndromes P540-554
Tremor
  • Kinetic with exacerbation at the end of the movement
• Rebound phenomenon: patient puts arm out and examiner pushes down onto wrist, observe on swaying
• Titubation: Head and body tremor (midline zone)
• Dysarthria: Scanning speech
• Hypotonia
• Normo,hyporeflexia
Head impulse test

• Test of VOR function that can be done by non-experts at the bedside
• To distinguish with acute vestibular neuritis or labyrinthitis.
• Patients with cerebellar infarction typically have a normal test.
The head impulse test

The head impulse, or head thrust, manoeuvre is a test of vestibular function that can be easily done during bedside examination. This manoeuvre tests the vestibulo-ocular reflex (VOR), and can help to distinguish a peripheral process (vestibular neuritis) from a central one (cerebellar stroke). With the patient sitting on the stretcher, the physician instructs him to maintain his gaze on the nose of the examiner. The physician holds the patient's head steady in the midline axis and then rapidly turns the head to about 20° off the midline. (A) The normal response (intact VOR) is for the eyes to stay locked on the examiner's nose. (B) An abnormal response (impaired VOR) is for the eyes to move with the head, and then to snap back in one corrective saccade to the examiner's nose. The test is usually "positive" (i.e., corrective saccade is visible) with peripheral lesions (vestibular neuritis), and the test is usually normal in cerebellar stroke. This occurs because the primary VOR pathway bypasses the cerebellum.

Lancet Neurol 2008; 7: 951–64
Clinical hints to Ix for cerebellar stroke

• Epidemiological context
  • Age over 50 years
  • Prior history of stroke or TIA
  • Stroke risk factors (smoking, HT, DM, DLP, AF known coronary or peripheral vascular disease)
  • Recent head or neck injury (including chiropractic manipulation or motor vehicle collision) or known collagen–vascular disorder, predisposing to vertebral artery dissection

• History
  • Abrupt onset of symptoms
  • N/V in the absence of other localising symptoms (e.g., diarrhoea, abdominal or chest pain, fever) or disproportionate to amount of dizziness or vertigo
  • Headache (sudden, severe, or sustained), particularly with other neurological symptoms (especially motor complaints such as limb weakness or abnormal speech)
  • Dizziness that persists more than 24 h, particularly with stroke risk factors, or in association with sudden hearing loss at onset (whether transient or persistent)
  • Symptoms of cranial nerve dysfunction (particularly diplopia, dysarthria, dysphagia, dysphonia, or facial dysaesthesia)
Figure 4: False negative brain CT in a patient with inferior cerebellar stroke
(A) Brain CT scan of the posterior fossa of a 77-year-old woman who presented to the emergency department with nausea, vomiting, and severe gait instability. This scan is normal with no indication of infarction. (B) Within about 1 h, the MRI was obtained. This diffusion-weighted image, taken at the same level, shows an infarction of the right posterior inferior cerebellum. Reproduced from Savitz et al., with permission from Wiley-Blackwell.

Figure 5: Infarction restricted to the superior cerebellar artery territory
Patients with infarction in the superior cerebellar artery territory typically present with the sudden onset of gait and truncal instability in association with dysarthria and limb ataxia or dysmetria. Dizziness, although frequently present, is typically a milder (non-vertiginous) sensation than the dizziness experienced by patients with infarcts of the inferior cerebellum. (A) T2 coronal MRI and (B) T2 sagittal MRI demonstrating superior cerebellar infarction. Arrows indicate the location of the infarction.
Clinical hints to Ix for cerebellar stroke

**Physical examination**

- Normal vestibular–ocular reflex by head impulse test (absence of a corrective saccade)
- Spontaneous nystagmus that is direction-changing or dominantly vertical or torsional
- Severe difficulty or inability to stand or walk
- Any other abnormal neurological finding, particularly cranial nerve dysfunction, Horner’s syndrome, or long tract signs (hemimotor, hemisensory, limb ataxia, or dysmetria)
Diagnosing and defining the vascular lesion

**Brain imaging**

- MRI is the preferred test, 80% - 95% sensitivity in the first 24 h with DWI
- For CT scan, widely available, acquires images quickly, and accurately excludes acute haemorrhage
- CT has lower sensitivity in the posterior fossa; the bone of the skull base artifacts (sensitivity 40-50%)
General management

• Similar to infarcts of other areas of the brain
• Close neurological monitoring, clinically significant cerebellar edema typically occurs within 1 to 7 days, with a mean peak of 3 days
• Osmotic diuretics such as mannitol or hypertonic saline may be useful.
• Surgical treatment is the management of choice
• Decompressive Surgery
  • suboccipital craniectomy; preventing and treating herniation and brain stem compression

• External Ventricular Drainage
  • in cerebellar infarction is still widely debated
  • In acute hydrocephalus, where transition to the operating room may be delayed
  • EVD placement will be rapid reduction in intracranial pressure may decrease mortality and prove lifesaving
RESULTS A total of 79 patients with cerebellar stroke.

17.7% died and 31.6% had poor outcomes at six months after discharge.

Patients with cerebellar haemorrhage have poor outcomes as compared to patients with cerebellar infarct (odds ratio [OR] 4.3, 95% CI (1.3–14.1).
Six months after discharge (OR 5.2, 95% CI 1.6–17.2). When compared to small lesions (< 5 cm³), lesions > 20 cm³ were significantly associated with poorer outcomes and the development of hydrocephalus and brainstem compression.
• A cerebellar stroke with a lesion is 3 cm in diameter on CT would give rise to a lesion with an estimated volume of 15 cm$^3$.

• These findings more likely to have a poor outcome
### 5.1. Cerebellar and Cerebral Edema

<table>
<thead>
<tr>
<th>5.1. Cerebellar and Cerebral Edema</th>
<th>COR</th>
<th>LOE</th>
<th>New, Revised, or Unchanged</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Ventrílocistomía is recommended in the treatment of obstructive hydrocephalus after a cerebellar infarct. Concomitant or subsequent decompressive craniectomy may or may not be necessary on the basis of factors such as infarct size, neurological condition, degree of brainstem compression, and effectiveness of medical management.</td>
<td>I</td>
<td>C-LD</td>
<td>Recommendation revised from 2014 Cerebral Edema.</td>
</tr>
</tbody>
</table>

Ventrílocistomía is a well-recognized effective treatment for the management of acute obstructive hydrocephalus and is often effective in isolation in relieving symptoms, even among patients with acute ischemic cerebellar stroke.⁵⁴,⁵⁵ Thus, in patients who develop symptoms of obstructive hydrocephalus from a cerebellar stroke, emergency ventriculostomy is a reasonable first step in the surgical management paradigm. If cerebrospinal fluid (CSF) diversion is planned, however, it is important to document the degree of obstruction before proceeding with surgical intervention. See Table LIX in online Data Supplement 1.

<table>
<thead>
<tr>
<th>5.1. Cerebellar and Cerebral Edema (Continued)</th>
<th>COR</th>
<th>LOE</th>
<th>New, Revised, or Unchanged</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Decompressive suboccipital craniectomy with dural expansion should be performed in patients with cerebellar infarction causing neurological deterioration from brainstem compression despite maximal medical therapy. When deemed safe and indicated, obstructive hydrocephalus should be treated concurrently with ventriculostomy.</td>
<td>I</td>
<td>B-NR</td>
<td>Recommendation revised from 2014 Cerebral Edema.</td>
</tr>
</tbody>
</table>

The data support decompressive cerebellar craniectomy for the management of acute ischemic cerebellar stroke with mass effect.³⁴-³⁶ This surgery is indicated as a therapeutic intervention in cases of neurological deterioration caused by cerebral edema as a result of cerebellar infarction that cannot be otherwise managed with medical therapy or ventriculostomy in the setting of obstructive hydrocephalus. See Table LIX in online Data Supplement 1.

| 3. When considering decompressive suboccipital craniectomy for cerebellar infarction, it may be reasonable to inform family members that the outcome after cerebellar infarct can be good after sub-occipital craniectomy. | Iib | C-LD | Recommendation and Class unchanged from 2014 Cerebral Edema. Wordings revised and LOE amended to conform with ACC/AHA 2015 Recommendation Classification System. |

| 4. Patients with large territorial supratentorial infarctions are at high risk for complicating brain edema and increased intracranial pressure. Discussion of care options and possible outcomes should take place quickly with patients (if possible) and caregivers. Medical professionals and caregivers should ascertain and include patient-centered preferences in shared decision making, especially during prognosis formation and considering interventions or limitations in care. | I | C-EO | New recommendation. |

Cerebellar edema can cause serious and even life-threatening complications in patients with large territorial supratentorial infarctions. Although less severe edema can be managed medically, surgical treatment may be the only effective option for very severe cases; in such instances, timely decompressive surgery has been shown to reduce mortality.⁵⁷ Nevertheless, there is evidence that persistent mortality is common and individual preexisting decisions about end-of-life and degree of treatment performed in the face of severe neurological injury must be considered.
### 5.1 Cerebellar and Cerebral Edema (Continued)

<table>
<thead>
<tr>
<th></th>
<th>COR</th>
<th>LOE</th>
<th>New, Revised, or Unchanged</th>
</tr>
</thead>
<tbody>
<tr>
<td>8. Although the optimal trigger for decompressive cranectomy is unknown, it is reasonable to use a decrease in level of consciousness attributed to brain swelling as selection criteria.</td>
<td>Ila</td>
<td>A</td>
<td>Recommendation, Class, and LOE unchanged from 2014 Cerebral Edema.</td>
</tr>
<tr>
<td>9. Use of osmotic therapy for patients with clinical deterioration from cerebral swelling associated with cerebral infarction is reasonable.</td>
<td>Ila</td>
<td>C-LD</td>
<td>Recommendation reworded for clarity from 2014 Cerebral Edema. Class unchanged. LOE amended to conform with ACC/AHA 2015 Recommendation Classification System. See Table LXXIII in online Data Supplement 1 for original wording.</td>
</tr>
<tr>
<td>10. Use of brief moderate hyperventilation (Pco₂ target 30–34 mmHg) is a reasonable treatment for patients with acute severe neurological decline from brain swelling as a bridge to more definitive therapy.</td>
<td>Ila</td>
<td>C-EO</td>
<td>New recommendation.</td>
</tr>
<tr>
<td>Hyperventilation is a very effective treatment to rapidly improve brain swelling, but it works by inducing cerebral vasoconstriction, which can worsen ischemia if the hypopcapnia is sustained or profound. Thus, hyperventilation should be induced rapidly but should be used as briefly as possible and avoid excessive hypopcapnia (&lt;30 mmHg).</td>
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<tr>
<td>11. Hypothermia or barbiturates in the setting of ischemic cerebral or cerebellar swelling are not recommended.</td>
<td>III: No Benefit</td>
<td>B-R</td>
<td>Recommendation and LOE revised from 2014 Cerebral Edema. COR amended to conform with ACC/AHA 2015 Recommendation Classification System. See Tables LXIX and LX in online Data Supplement 1.</td>
</tr>
<tr>
<td>The data on the use of hypothermia and barbiturates for the management of AIS continue to be limited. Such data include only studies with small numbers of patients and unclear timing of intervention with respect to stroke onset. Hypothermia use has recently been shown to have no impact on stroke outcomes in a meta-analysis of 6 RCTs. Further research is recommended.</td>
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<tr>
<td>12. Because of a lack of evidence of efficacy and the potential to increase the risk of infectious complications, corticosteroids (in conventional or large doses) should not be administered for the treatment of cerebral edema and increased intracranial pressure complicating ischemic stroke.</td>
<td>III: Harm</td>
<td>A</td>
<td>Recommendation wording modified from 2013 AIS Guidelines to match Class III stratifications. LOE unchanged. Class amended to conform with ACC/AHA 2015 Recommendation Classification System.</td>
</tr>
</tbody>
</table>
Take-Home Points

• Patients with cerebellar stroke may present with generalized symptoms.
• A high index of suspicion must be maintained in patients with significant risk factors.
• Head impulse test may help for distinguish pheripheral or central lesion
• CT has low sensitivity for infarctions of the posterior fossa. If suspicion of infarction is high, MRI/diffusion weighted imaging sequences should be obtained.
• Emergent placement of an external ventricular drain or posterior fossa decompression may be lifesaving in patients with hydrocephalous or brain stem compression.
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