Cerebellar infarction

- COVID-19-Related Spontaneous Vertebral Artery Dissection: A Case Report
- Endovascular treatment of a craniocervical junction dural arteriovenous fistula associated with lateral medullary syndrome: A case report
- Cerebral Perfusion Features in Takayasu Arteritis: Insights From pCASL MRI
- Cytomegalovirus meningoencephalitis in an immunocompetent host with evolution of cystic encephalomalacia
- Wallerian degeneration of the middle cerebellar peduncles secondary to pontine infarction, case report, and review of literature
- A case of recurrent hemangioblastoma receiving blood supply from the mastoid and transosseous branches of the occipital artery
- Successful endovascular occlusion of multiple fusiform aneurysms on the persistent primitive lateral basilovertebral anastomosis
- Diagnostic yield of an abbreviated MRI protocol in the evaluation of dizziness in the emergency department, a single institutional experience

Epidemiology

Relatively rare (seen on only 0.6% of all CTs obtained for any reason).

see Cerebellar infarction in childhood.

Classification

cerebellar infarctions may be classified as involving the PICA distribution (cerebellar tonsil and/or inferior vermis), superior cerebellar artery distribution (superior hemisphere or superior vermis), or other indeterminate patterns.

	Vermis (midline)	Hemispheres (right or left)
Lobar classification (clinical purposes)		
Anterior lobe	anterior vermis	anterior hemisphere
Posterior lobe	posterior vermis	posterior hemisphere
Flocculonodular lobe	nodulus	flocculus
Lobular classification (research purposes)		
Anterior lobe	lobule I, II, III, IV and/or V	lobule I, II, III, IV and/or V
Posterior lobe	lobule VI, crus I, crus II, lobule VII, VIII and/or IX	lobule VI, crus I, crus II, lobule VII, VIII and/or IX
Flocculonodular lobe	lobule X (nodulus)	lobule X (flocculus)

[1].

Very small cerebellar infarctions (diameter <2 cm) are a frequent finding on MRI. With an increasing scientific interest in cerebral microinfarcts, very small infarcts in the cerebellum deserve more of our attention as well.

Very small cerebellar infarctions have been referred to as lacunar infarcts, as junctional, border zone or watershed cerebral infarctions, as nonterritorial infarcts, as very small territorial or end zone infarcts, or simply as (very) small cerebellar infarctions. Since the original clinicoradiological study on

these small infarcts, the classification into border zones remains in common use. This classification is based upon the assumption that these infarcts occur secondary to low flow in between arterial perfusion territories, where flow is believed to be the lowest. Later studies, however, have suggested occlusion of small (end-) arteries as a prerequisite for the pathogenesis of even small cerebellar infarctions, with low flow merely as a potential contributor. Therefore, it is likely that infarcts may as well occur in a nonborder zone distribution. Moreover, the classification into border zones may be considered unreliable since the location of border zones is highly variable among individuals and is not known in a particular patient. Recently, a functional topographic organization has been found in the cerebellum with evidence for a motor-nonmotor dichotomy between the anterior and posterior lobe. Since the cerebellar lobes can be easily and reliably distinguished with both CT and MRI, we recommend the classification of very small cerebellar infarctions according to topographic location.

There are several fundamental concerns with the current classification of very small cerebellar infarctions according to border zones, which we would like to overcome by recommending a new classification system based on topography. This will allow for a reliable and reproducible way of classifying very small cerebellar infarctions and is expected to improve clinicoradiological correlation [2)].

Etiology

see Cerebellar infarction after superior petrosal vein injury.

see Atlantoaxial rotatory subluxation.

see Vertebral artery dissection.

Clinical Features

Often presents with common and non-specific symptoms such as dizziness, nausea and vomiting, unsteady gait, and headache.

Early clinical findings

In most cases the onset is sudden, without premonitory symptoms.16 The first 12 hrs after onset were characterized by lack of progression. Early findings are due to the intrinsic cerebellar lesion (ischemic infarction or hemorrhage): 1. symptoms a) dizziness or vertigo

b) nausea/vomiting

c) loss of balance, often with a fall and inability to get up

d) headache(infrequent in one series)

2. signs

a) truncal and appendicular ataxia

b) nystagmus

c) dysarthria

Later clinical findings

Patients with cerebellar infarction may subsequently develop increased pressure within the poste- rior fossa (due to cerebellar edema or mass effect from clot), with brainstem compression (particu- larly posterior pons). Clinical findings generally increase between 12 and 96 hrs following onset. Compression of the Sylvian aqueduct can cause acute hydrocephalus with attendant increased ICP.

Diagnosis

cerebellar infarction Diagnosis

Differential diagnosis

The differential diagnosis is broad and includes many common and benign causes. Furthermore, early-stage posterior fossa ischaemia is rarely seen with brain CT–the most commonly available initial imaging test that is used for stroke. Insufficient examination and imaging can result in misdiagnosis. However, early correct diagnosis is crucial to help prevent treatable but potentially fatal complications, such as brainstem compression and obstructive hydrocephalus.

Treatment

Cerebellar infarction treatment.

Complications

The frequency of cerebellar infarction with cerebellar edema and symptoms is reported to be 17% to 54% [3].

Space-occupying brain edema is a frequent and one of the most dreaded complications in ischemic cerebellar stroke. Because the tight posterior fossa provides little compensating space, any space-occupying lesion can lead to life-threatening complications through brainstem compression or compression of the fourth ventricle and subsequent hydrocephalus, both of which may portend transtentorial herniation/transforaminal herniation [4].

The acute phase in a cerebellar infarction may become complicated with transient obstructive hydrocephalus, subsequent intracranial hypertension, and the need for surgical management. Although many patients respond well to medical treatment, clinical findings and neuroimaging methods must be considered to determine whether the hydrocephalus can be surgically treated in a timely fashion.

In fourteen patients, six required surgery for hydrocephalus management. Three of the cases had an endoscopic third ventriculostomy without complications, the rest were managed conservatively. As an average, patency was re-established in the aqueduct three months post ictus.

Management of obstructive hydrocephalus in the acute phase of a cerebellar stroke must be individualized. In cases with transient obstructive hydrocephalus, endoscopic third ventriculostomy is a good surgical treatment option that avoids the risks of a long-term ventricular shunt [5)].

Outcome

In particular, advanced age and additional brainstem infarction seem to be predictors for poor outcomes [6)].

80% of patients developing signs of brainstem compression will die, usually within hours to days.

The identification and treatment of the underlying vascular lesions at an early stage can also prevent subsequent occurrences of stroke and improve patients' outcomes [7)].

Quantitative volumetric measurement predicts poor outcome of cerebellar stroke patients, even when controlling for age and NIHSS. Quantitative analysis of diffusion MRI may assist in identification of patients with cerebellar stroke at highest risk of neurological deterioration. Prospective validation is warranted [8)].

A study assessed motor deficits in the acute phase after isolated cerebellar stroke focusing on postural impairment and gait ataxia and outlines the role of lesion site on motor outcome, the course of recovery and the effect of treadmill training. 23 patients with acute and isolated cerebellar infarction participated. Deficits were quantified by ataxia scores and dynamic posturography in the acute phase and in a follow up after 2 weeks and 3 months. MRI data were obtained to correlate lesion site with motor performance. Half of the patients that gave informed consent and walked independently underwent a 2-week treadmill training with increasing velocity. In the acute phase patients showed a mild to severe ataxia with a worse performance in patients with infarction of the superior in comparison to the posterior inferior cerebellar artery. However, after 3 months differences between vascular territories were no longer significant. MRI data showed that patients with larger infarct volumes had a significantly more severe ataxia. In patients with ataxia of stance, gait and lower limbs lesions were more common in cerebellar lobules IV to VI. After 3 months a mild ataxia in lower limbs and gait, especially in gait speed persisted. Because postural impairment had fully recovered, remaining gait ataxia was likely related to incoordination of lower limbs. Treadmill training did not show significant effects. Future studies are needed to investigate whether intensive coordinative training is of benefit in patients with cerebellar stroke [9)].

Retrospective multicenter cohort studies

In a retrospective multicenter cohort study, patients with cerebellar infarction treated at 5 tertiary referral hospitals or stroke centers within Germany between 2008 and 2021 were included. Data were analyzed from November 2020 to November 2023.

Exposures: Surgical treatment (ie, posterior fossa decompression plus standard of care) vs conservative management (ie, the medical standard of care).

Main outcomes and measures: The primary outcome examined was functional status evaluated by the modified Rankin Scale (mRS) at discharge and 1-year follow-up. Secondary outcomes included the predicted probabilities for favorable outcomes (mRS score of 0 to 3) stratified by infarct volumes or Glasgow Coma Scale score at admission and treatment modality. Analyses included propensity score matching, with adjustments for age, sex, Glasgow Coma Scale score at admission, brainstem involvement, and infarct volume.

Results: Of 531 included patients with cerebellar infarcts, 301 (57%) were male, and the mean (SD) age was 68 (14.4) years. After propensity score matching, a total of 71 patients received surgical treatment and 71 patients conservative treatment. There was no significant difference in favorable outcomes (ie, mRS score of 0 to 3) at discharge for those treated surgically vs conservatively (47 [66%] vs 45 [65%]; odds ratio, 1.1; 95% CI, 0.5-2.2; P > .99) or at follow-up (35 [73%] vs 33 [61%]; odds ratio, 1.8; 95% CI, 0.7-4.2; P > .99). In patients with cerebellar infarct volumes of 35 mL or greater, surgical treatment was associated with a significant improvement in favorable outcomes at 1-year follow-up (38 [61%] vs 3 [25%]; odds ratio, 4.8; 95% CI, 1.2-19.3; P = .03), while conservative treatment was associated with favorable outcomes at 1-year follow-up in patients with infarct volumes of less than 25 mL (2 [34%] vs 218 [74%]; odds ratio, 0.2; 95% CI, 0-1.0; P = .047).

Conclusions and Relevance: Overall, surgery was not associated with improved outcomes compared with conservative management in patients with cerebellar infarcts. However, when stratifying based on infarct volume, surgical treatment appeared to be beneficial in patients with larger infarct volumes, while conservative management appeared favorable in patients with smaller infarct volumes [10)].

Case series

All patients treated with suboccipital decompressive craniectomy (SDC) due to space-occupying cerebellar infarction between January 2009 and October 2015 in the Rigshospitalet, were included in the study. Data was retrospectively collected from patient records, CT/MRI scans and surgical protocols. Long-term functional outcome was determined by the modified Rankin Scale (mRS) and mRS ≥ 4 was defined as unfavorable outcome.

Twenty-two patients (16 male, 6 female) were included in the study. Median age was 53 years. Nine patients were treated with external ventricular drainage as an initial treatment attempt prior to SDC. Median time from symptom onset (stroke ictus) to initiation of the SDC surgery was 48 h (IQR 28-99 hours) and median GCS before SDC was 8 (IQR 5-10). At follow up, median mRS was 3 (IQR 2-6). Outcome was favorable (mRS 0-3) in 12 patients and unfavorable in 10 (3 with major disability, 7 dead). Brainstem infarction and bilateral cerebellar infarction were associated with unfavorable outcome.

In this small study, functional long-term outcome in patients with space-occupying cerebellar infarction treated by SDC was acceptable and comparable to previously published results (favorable outcome in 54% of patients). Brainstem infarction and bilateral cerebellar infarction were associated with unfavorable outcome [11].

Case reports

A 51-year-old female who developed bilateral cerebellar hemorrhages following opioid and benzodiazepine overdose. Malignant cerebellar edema is a rare but recognized complication following opiate overdose in children or chronic heroin toxicity. However, acute cerebellar involvement is rarely reported in adults. Ahmed et al. feel that clinicians and radiologists should keep in mind the possibility of opioid toxic encephalopathy in their differential for adults with acute bilateral cerebellar infarctions and/or hemorrhages [12]

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