Cerebellar infarction requires quick diagnosis and immediate treatment with intravenous thrombolytics. A case report

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Case report

A 61-year-old woman presented at the Emergency Department with acute vertigo, headache, nausea, vomiting and dysarthria. The patient was known for type 2 diabetes mellitus, hypercholesterolemia, hypothyroidism and hypertension. She used antihypertensive drugs, lipid-lowering drugs, thyroid hormone suppletion and oral antidiabetic drugs.

On examination she had a bidirectional gaze-evoked 1st degree nystagmus. There was diminished left-sided facial sensation, a right-sided facial palsy with peripheral features, a right-sided Horner’s syndrome, limb ataxia on the right side and diminished sensation to pinprick in the left-sided limbs.

Diagnostic evaluation

Unenhanced cerebral computed tomography (CT) showed no signs of ischemia or haemorrhage (Figure 1). As shown in Figure 1B, CT angiography (CT-A) showed an occluded right superior cerebellar artery (SCA). A few calcifications of the aortic arch were noted, but additional CTA showed normal brachiocephalic arteries and no stenoses in both vertebral arteries. CT perfusion (CT-P, Figure 1C) showed a perfusion defect in the upper part of the right cerebellar hemisphere and extending into the right dorsal side of the pons, which corresponded to the vascular territory of the right SCA. An electrocardiogram (ECG) revealed no abnormalities. Two days after the first scan, CT-A showed recanalization of a very small right SCA and the CT perfusion confirmed ischemia of the vascular territory of the SCA (Figure 1E and 1F).

Treatment and outcome

The patient received treatment with intravenous alteplase directly after the unenhanced CT was performed. The door to needle time was 26 minutes. The patient was admitted to the stroke unit and a platelet inhibitor (clopidogrel) was started after several hours. During several days on our ward the patient’s vertigo and nausea improved. She made a close to complete recovery of the right hemi-ataxia.

Risk factors for cardiovascular diseases were evaluated. We intensified secondary stroke prevention by increasing antihypertensive medication. Other risk factors like diabetes and dyslipidaemia were already well controlled with medication. No direct cause of the infarction, either atherothrombotic or cardiogenic, was found.

Discussion

Cerebellar infarcts account for 1.5% to 3% of ischaemic strokes. The clinical presentation is diverse and highly variable; the main symptoms are vertigo, nausea and vomiting, gait instability and headache. Lesions of the cerebellar hemispheres cause varying degrees of ataxia.
Case Report

Most of these symptoms are often caused by common and benign disorders, which may explain the frequent misdiagnosis and delay in treatment.[2]

As shown in Figure 2 blood flow in the cerebellum is supplied mainly by three arteries: the posterior inferior cerebellar artery (PICA), which branches from the vertebral artery, the anterior inferior cerebellar artery (AICA) and superior cerebellar artery (SCA), both of which branch from the basilar artery.[3] The PICA provides the postero-inferior part, the AICA distributes to the antero-inferior part, and the SCA distributes to the superior part.[3]

Proximal branches of the three main cerebellar arteries typically supply the lateral part of the brainstem; therefore coincident brainstem signs can be found in patients with cerebellar stroke. [2] So called “crossed” signs like ipsilesional cranial nerve and contralesional long tract signs suggest involvement of the brainstem.[2] Our patient presented with an ipsilesional right-sided facial palsy together with a diminished sensation of the left-sided face and left-sided limbs, both contralesional, suggesting brainstem involvement at the level of the pons.

Unenhanced CT rarely identifies early-stage cerebellar infarction, but is used to rule out intra-cerebral haemorrhage. Accurate neurological examination is crucial to identify cerebellar stroke.[2] Recognizing a cerebellar infarction is very important for quick treatment with thrombolytic agents and preventing potentially fatal complications, such as brainstem compression and obstructive hydrocephalus.[2] CTA is important to identify the occluded vessel. CT-P is required to visualize the ischemic region and to determine the presence of potentially treatable penumbra.

When the clinical diagnosis of acute cerebral infarction is made, and unenhanced CT rules out intra-cerebral haemorrhage, prompt treatment with intravenous recombinant tissue plasminogen activator (iv rt-PA (alteplase)) within 4.5 hours of the onset of symptoms needs to be established unless there are contraindications.[4,5] The aim of treatment with intravenous rt-PA is to recanalize the occluded artery by lysis of the obstructing clot, which should lead to restored perfusion of brain tissue in the affected area. Intravenous thrombolysis has shown to be effective in improving outcome [4,6] The sooner that rt-PA is given to stroke patients, the greater the benefit, especially if started within 90 minutes after onset.[4,5] In our patient early intravenous thrombolysis has led to complete recovery, although an infarct remained visible on repeat CT scanning.

An antiplatelet agent like clopidogrel is started after a delay of several hours after treatment with alteplase, because immediate treatment with antiplatelet agents has been associated with the occurrence of intracerebral hemorrhage. Identification and treatment of the underlying cardiovascular and cerebrovascular pathology will be done in the next 48 hours to prevent additional ischemia.[2] Most cerebellar infarcts do have a benign outcome.[7] Prognosis is poorer when the brainstem is also affected.[1]

Other or additional options for treatment are intra-arterial thrombolysis or mechanical thrombectomy. These treatments are still considered experimental, and their indication depends on the presence of a relevant arterial occlusion and on contraindications for intravenous thrombolysis like increased bleeding risk or elapsed time since first symptoms.[8,11]

**Conclusion**

This case illustrates that a cerebellar infarction often presents with common and non-specific symptoms. Basic knowledge of the symptoms of brainstem and cerebellar infarction is essential for an early and accurate diagnosis. Neurological examination is important to distinguish between cerebellar stroke and a more benign condition, since brain CT rarely identifies early-stage cerebellar infarction.[2] New imaging techniques like CT-P will contribute to the diagnosis. Rapid recognition of a cerebellar infarction and differentiation from more benign disorders presenting with vertigo, headache, dysarthria or vomiting, is crucial to achieve an optimal outcome with early treatment with intravenous alteplase.

**References**