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Case report

Bilateral cerebellar infarction in the medial branches of posterior inferior cerebellar arterial territory—Using endoscopic third ventriculostomy to relieve acute hydrocephalus

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Abstract

Simultaneous bilateral cerebellar infarctions in posterior inferior cerebellar arterial (PICA) territory, without brain stem involvement are rare. We herein report a 51-year-old man developed sudden dizziness, nausea and vomiting. CT revealed hypodense bilateral lesions over the cerebellum corresponding to the medial PICA (mPICA) branch territory. His mental state deteriorated 2 days after onset; repeated CT showed severe third and lateral ventricular dilation. Endoscopic third ventriculostomy (ETV) was done to relieve the acute obstructive hydrocephalus. The patient was later discharged with only mild residual ataxia. Compared with conventional surgical treatments (external ventricular drainage, craniectomy and cerebellectomy), ETV has several advantages, including less risk and minimal invasiveness. However, further study is needed on its safety and efficacy under such circumstances.

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1. Introduction

Cerebellar infarction usually occurs in posterior inferior cerebellar arterial (PICA) and superior cerebellar arterial territory. Simultaneous bilateral PICA infarctions are rare because a PICA usually originates from the ipsilateral vertebral artery. There are only a few reported cases of cerebellar infarction involving the medial PICA (mPICA) in both hemispheres [1–4]. However, the prognoses of such infarctions are usually good as hydrocephalus is only occasional. This case presented bilateral cerebellar infarction of mPICA territory, leading to obstructed hydrocephalus, which was relieved by endoscopic third ventriculostomy (ETV); this procedure has been recently applied in such cases [5,6].

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2. Case report

The subject was a 51-year-old male with a history of tobacco and alcohol abuse. He developed acute nonvertiginous dizziness, nausea, vomiting and an unsteady gait without specific lateropulsion. His medical history revealed a surgically closed perforated peptic ulcer from 5 years before. Except smoking, there were no risk factors for an arteriosclerotic/atherosclerotic disease, i.e. diabetes mellitus, hypertension or heart disease. He had been transferred from a local hospital to our emergency department. He had an initial blood pressure of 170/108 mmHg and heart rate of 76 bpm. The patient was clearly conscious but had severe dizziness. Neurologically, he did not exhibit findings attributable to brain stem dysfunction, e.g. numbness, diplopia, dysphagia and/or dysarthria, despite cerebellar signs. He did have bilateral dysmetria with intentional tremor, and was unable to stand due to severe truncal dysbalance. Routine blood count, biochemistry and coagulation profiles were all within norms.

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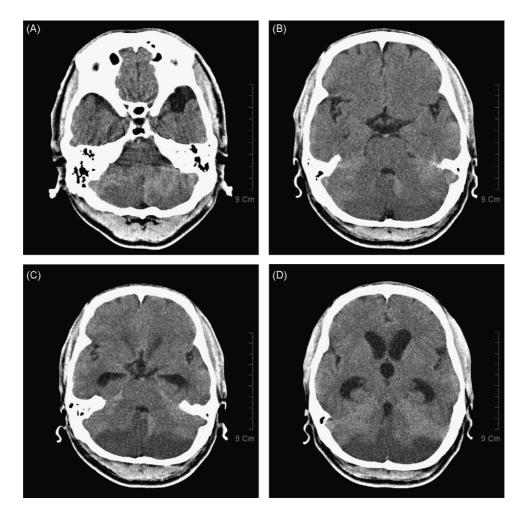


Fig. 1. (A and B) Non-contrasting brain CT, 12 h after onset, showing typical hypodense triangular bilateral lesions of the caudal cerebella caused by acute cerebellar infarction confined to bilateral mPICA territory. (C and D) CT after 53 h with deteriorated consciousness, showing infarcted cerebellar swelling and dilated third and lateral ventricles suggestive for obstructive hydrocephalus.

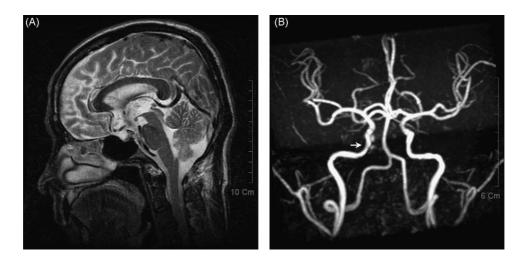


Fig. 2. (A) MRI showing patent functioning and a flow void at the floor of the third ventricle after third ventriculostomy. (B) MRA showing stenosis in the petrous portion of right internal carotid artery (arrow).

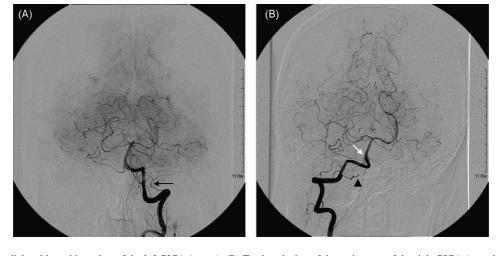


Fig. 3. (A) Patent medial and lateral branches of the left PICA (arrow). (B) Total occlusion of the main stem of the right PICA (arrowhead) and a dominant right AICA (white arrow).

Electrocardiography showed normal sinus rhythms. Computerized tomography (CT), 12 h after onset, showed bilateral cerebellar infarction attributable to both mPICA territory (Fig. 1A and B).

After 2 days, the patient deteriorated, and became agitated, confused and disoriented. His Glasgow Coma Scale dropped from full to E3V4M6. Repeated CT showed severe third and lateral ventricular dilation suggestive for obstructive hydrocephalus (Fig. 1C and D). A neurosurgeon was consulted and emergent endoscopic third ventriculostomy (ETV) was done to relieve the obstructed hydrocephalus and an external ventricular drainage (EVD) was left. After operation, the EVD was kept closed without drainage of cerebrospinal fluid and functioned as an intracranial pressure (ICP) monitor. Since the ICP was within normal limit on the following days, the EVD was removed on post-operative day 3. The patient regained consciousness 6h after surgery; the endotracheal tube was removed 12 h later. Magnetic resonance imaging, 11 days after onset, showed patent third ventriculostomy function, with no hydrocephalus remaining (Fig. 2A) and this was also verified by a cine MRI. No significant abnormalities were noted from magnetic resonance angiography except moderate stenosis of the petrous portion of right internal carotid artery (Fig. 2B). Transthoracic echocardiography revealed no intracardiac thrombus. Vertebral arteriography (Fig. 3A and B) showed normal path and lumen size for the upper cervical bilateral vertebral artery. The left PICA and bilateral anterior inferior cerebellar artery were both normal. The main trunk of the right PICA was completely occluded. The patient was rehabilitated for ataxia and discharged 4 weeks after onset with only mild residual dizziness.

3. Discussion

Tada et al. [1] reported the first clinicoradiological case of bilateral cerebellar mPICA territorial infarction. There were several possible contributing factors, in that case and this one, such as both PICAs arising from the basilar artery, both mPICAs arising from a single PICA on one side, emboli to bilateral PICA, or pressure effects exerted by a large PICA infarction compressing the cerebellar cistern arteries, inducing a smaller opposite-side infarction. Since vertebral angiography showed the medial and lateral branches of the left PICA to be completely patent, and the right PICA totally occluded, we believe that the left mPICA territory must have been mainly supplied by a more dominant right medial PICA, and the right lateral PICA territory is mainly supplied by a right dominant AICA, as supported by the angiogram. This would most likely explain the CT and angiographic findings. According to Kang et al. [3], the etiology of such infarctions includes stenoocclusive PICA and extracranial vertebral arterial disease. An arteriosclerotic etiolgy or embolic disease cannot completely be ruled out, since the patient showed findings attributable to atherosclerotic stenosis of the right internal carotid artery as shown in the MR angio. However, classical risk factors were not present. Regarding an embolic disease, the findings were also not contributive, at least as evidenced by the electrocardiography and the transthoracic echo. A 24 h long-term ECG and a transesophageal echo were not performed.

Obstructive hydrocephalus can be derived from various processes, like the aqueduct stenosis, tumors from pineal area or cerebellum, and cerebellar infarction or hematoma. Permanent ventriculoperitoneal shunt or a 1–2-week period EVD is the treatment of choice for the obstructive hydrocephalus. However, the risks of post-operative ventriculitis and meningitis are high and problems of shunt dependency and dysfunction can pose significant additional morbidity to the patient. One study indicated that the overall infection rate after EVD is 8.6% in a setting of neurosurgical care unit [7].

The obstructive hydrocephalus secondary to a cerebellar infarct ranges from 13 to 30% [8,9] and is transient in nature. As long as the tissue swelling resolves, the CSF passage will

regain patency. The "transient" feature offers the rationale for ETV as an alternative for suboccipital craniectomy and EVDs or VP shunts in selective conditions. From the literature, only 13 cases receiving ETV have been reported in the scenario of cerebellar infarction [5,6]. Except for one patient who receiving suboccipital craniectomy due to progressive brain edema after ETV [5], the remaining 12 cases were well tolerant to ETV and had uneventful courses. However, not all patients with obstructive hydrocephalus secondary to cerebellar infarct are candidates for ETV. According to the series from Baldauf et al., they excluded the comatous patients which implicated the brain stem compression, and the mean Glasgow Coma Scale is 11.2 before surgery [5]. We inserted an EVD for monitoring the ICP with the fear of the unexpected increasing ICP. Besides, this short-term (3 days) EVD does not increase the risk of ventriculitis compared to the longer period (1–2 weeks).

In conclusion, the subject suffered bilateral cerebellar infarctions confined to mPICA vascular territory; ETV successfully relieved the resulting hydrocephalus. These infarctions may have been caused by occlusion of the right PICA, but this anatomical variation cannot be proven. In selective cases, ETV is a rational treatment modality for cerebellar infarction-induced hydrocephalus.

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