Case Report

Infarction in Anterior Inferior Cerebellar Artery Territory Caused by Occlusion of Vertebral Artery

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Although the anterior inferior cerebellar artery (AICA) has the smallest territory of all the cerebellar arteries, its exact location and extent varies depending on the calibre and supply of the posterior inferior cerebellar artery (PICA) [1,2]. However, the AICA invariably supplies the middle cerebellar peduncle, inferolateral pons, inner ear, and the anterior surface of the cerebellar hemisphere, including the flocculus [1,3]. Terminal AICA branches usually feature anastomoses with the PICA or superior cerebellar artery [1,4–7].

In cases of occlusion of the PICA or vertebral artery (VA), the clinical signs and territory of the infarction are characterized as “lateral medullary syndrome”, which are clearly distinct from those of occlusion of the AICA [7,8]. We present here a very unusual case with the clinical features of infarction in the territory of the AICA as a result of occlusion of the ipsilateral VA.

CASE REPORT

A 74 year-old man with a history of hyperlipidaemia and ischaemic heart disease presented with a sudden onset of dizziness, dysarthria, tinnitus, hearing disturbance, nausea and vomiting. He was referred to the emergency room of our hospital. Neurological examination found trunkal ataxia, Brun’s nystagmus, right peripheral facial palsy, weakness of the right corneal reflex and right sensoineural deafness. Blood pressure was within normal limits, and electrocardiography and laboratory data disclosed no abnormalities.

Computed tomography (CT) and magnetic resonance imaging (MRI) showed infarction of the inferolateral pons, middle cerebellar peduncle, and the anterior and inferomedial surface of cerebellar hemisphere including flocculus (Fig. 1a–c). Diffusion imaging of MRI showed the lesion as an extremely hyperintense signal, indicating an acute phase infarction. The patient was admitted to our department and received anti-platelet therapy. T1-weighted MRI demonstrated a hyperintense signal in front of the medulla, suggesting the presence of a fresh thrombus at the distal portion of VA (Fig. 1d). Magnetic resonance angiography also revealed occlusion of the right VA (Fig. 1e).

Digital subtraction angiography performed 1 week after the onset showed occlusion of the right VA and multiple stenosis of the left VA (Fig. 2a–c). The left PICA supplied the medial part of the contralateral PICA territory via an anastomosis between the bilateral inferior vermian branches (Fig. 2b). Right AICA was hypoplastic, but patent. Both carotid angiograms were within normal limits. Measurement of the cerebral blood flow with 99mTc-labelled D,L-hexamethyl-propyleneamine oxime showed a flow defect in the inferolateral pons, and the anterior and inferolateral surface of the right cerebellar hemisphere, which was compatible with the CT and MRI findings.

The patient’s neurological status has improved except for deafness and tinnitus. Because the follow-up angiography, performed 2 months later, showed that the abnormalities of neither VA had diminished, a stent was placed in the left VA to maintain the flow of the basilar artery and prevent further stroke (Fig. 3). After this procedure, the patient was discharged without any additional neurological deficits. He has been treated as an outpatient and given medication consisting of ticlopidine hydrochloride and aspirin.

DISCUSSION

The AICA invariably supplies the inferolateral pons, middle cerebellar peduncle, and the anterior surface of the cerebellar hemisphere including the flocculus, inner ear, but rarely the upper lateral medulla [1,3]. This area is considered to be the proper territory of the AICA, with the middle cerebellar peduncle being regarded as the “core” of this territory [1,9].

The classic neurological signs of the AICA territory described by Adams consist of ipsilateral cerebellar ataxia, Horner’s syndrome, involvement of cranial nerves, such as V, VII and VIII, together with contralateral sensory disturbance of temperature sensation [1,4,10]. A peripheral vestibular syndrome secondary to a lesion affecting the vestibular nucleus has been attributed to occlusion of the internal auditory artery, originating from the AICA in about 80% of all cases, which may also account for cochlear symptoms [1,4,11]. Deafness and facial palsy are thought to be the most accurate clinical signs for distinguishing between AICA and PICA syndromes [1,4,11]. In our case, the symptoms could be attributed to the AICA territory.

Two mechanisms of AICA territory infarction have been described by Amarenco [5]. One is an isolated unilateral AICA
Infarction caused by basilar artery (BA) plaques extending into the AICA or by microatheroma blocking the origin of the AICA [12]. The other is a widespread infarction including the AICA territory caused by BA occlusion [9]. In our case, no occlusions of the AICA or plaques in the BA were found. Moreover, no symptoms of BA occlusion, such as disturbance of consciousness were seen, nor was the infarction limited to the proper AICA territory.

Although the many different varieties of the AICA anatomy have been reported, Atkinson has described the two most

Fig. 1 – (a) Computed tomography scan at admission showed low-density area at the anterior surface of the cerebellar hemisphere. (b) T2-weighted image of magnetic resonance imaging (MRI) revealed hyper signal intensity at the inferolateral pons, middle cerebellar peduncle and anterior surface of cerebellar hemisphere including flocculus. (c) Diffusion image of MRI revealed hyper signal intensity corresponded to the hyper signal intensity area of T2-weighted image. (d) T1-weighted image of MRI revealed hyper signal intensity area in front of medulla, suggesting the fresh thrombus in right vertebral artery (VA). (e) Magnetic resonance angiography showed the occlusion of right VA.

Fig. 2 – (a) Right vertebral angiogram showed the occlusion of VA. Anterior–posterior (b) and lateral (c) view of left vertebral angiogram showed the multiple stenotic lesions in VA (arrow). (b) The left posterior inferior cerebellar artery had collateral supply to the contralateral PICA territory (double arrows).
common [4,5,13]. One is a major anastomosis between the AICA and PICA, which is consistently found in patients with both arteries equally dominant. The other features AICA dominance on one side associated with ipsilateral vertebral hypoplasia and contralateral PICA dominance [4,6]. Oas [4] reported that the root entry zone of the seventh and eighth nerves is sometimes spared because of abundant collateral flow between the AICA and PICA. In our case, the left PICA supplied part of the territory of the right PICA via the inferior vermian branch, therefore medulla and inferior vermis were spared. Because the AICA is hypoplastic, the right PICA could supply the territory of the ipsilateral AICA, in addition to the inferomedial surface of cerebellar hemisphere, which is considered as the proper territory of PICA.

The PICA is the cerebellar artery with the most different configurations and in case of asymmetrical PICAs, branches of one PICA partially feed the territory of the other, while the “extensive” PICA may supply the cerebellum bilaterally [6,7]. As reported previously, there are various dominance patterns between the AICA and PICA [14]. In our patient, because the right PICA was considered to replace the ipsilateral AICA, infarction of the proper territory of AICA, such as inferolateral pons, middle cerebellar peduncle, and the anterior surface of cerebellar hemisphere, and of partial territory of right PICA, the inferomedial surface of cerebellar hemisphere, developed with the occlusion of right VA.

In addition, our patient’s condition also suggested that after occlusion of the right VA, the shortage of blood flow from the left VA due to multiple stenotic lesions was attributable to ischaemia of the border zone of the AICA and PICA. The inferred anatomical relationship between the AICA and PICA, and the ischaemia due to VA stenosis seemed to account for the discrepancy between the findings of MRI and angiography. In spite of the many variations of AICA and PICA distribution, there is no report of infarction due to VA occlusion of the AICA territory proper, so that attention should be paid for the possible occurrence of variations between the area of infarction and corresponding arteries.

REFERENCES