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An Autopsy Case of Cerebellar Hemorrhagic Infarction in the Region of the Bilateral Superior Cerebellar Arteries

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Abstract

The authors describe an autopsy case of cerebellar hemorrhagic infarction in the region of the bilateral superior cerebellar arteries (SCA). A 79-year-old woman suddenly developed dysarthria, bilateral blepharoptosis, external ophthalmoplegia, left hemiparesis, dysphagia and dysuria. CT scan revealed low density in the bilateral cerebellar hemispheres. Angiography revealed narrowing of the distal portion of the basilar artery. The patient made a gradual recovery in the first two weeks, but she died of a hyperglycemic non-ketotic diabetic coma on the 32nd day. Autopsy disclosed recent hemorrhagic infarctions of the bilateral SCA regions and of right Ammon's horn and numerous softenings of the brain stem, together with an old cystic infarction in the region of the middle cerebral artery. There were no occlusions in either the basilar or superior cerebellar arteries. It was suggested that an embolization due to severe atherosclerosis of the distal portion of basilar artery affected its pontine branches and bilateral SCA.

Key words: cerebellar infarction, hemorrhagic infarction, superior cerebellar artery, basilar artery, autopsy

Introduction

Although cerebellar infarctions may involve any portion of the cerebellum,[2,3,5,6,8] the most common site is its postero-inferior half.[8] Infarctions of hemorrhagic type usually represent only one quarter of the total.[8] Therefore, cerebellar infarctions of the hemorrhagic type in the region of the bilateral superior cerebellar arteries (SCA) are extremely rare.[1-6,8] The authors report an unusual case, which presented with dysarthria, bilateral blepharoptosis, external ophthalmoplegia, left hemiparesis, dysphagia and dysuria.

Case Report

History: A 79-year-old woman collapsed on January 6, 1982. She could respond to voices, but could not speak and open her eyes. Vomiting occurred twice. On the next morning, she was admitted to our hospital, complaining of dysuria of 18 hours duration.

Physical and neurological examinations: The patient was supine with both eyes closed. Blood pressure was 110/60 mmHg, and the pulse was 76/min and irregular. The radial artery was sclerotic. The lower abdomen was slightly distended to discharge about 350 ml of urine through a catheter.

The patient was almost alert and oriented, but could not speak. She could only respond to orders by nodding or writing a few words. There were bilateral blepharoptosis and left homonymous hemianopsia. The right eye was fixed in the midposition and the left in the abducted position. Both eyes revealed almost complete gaze palsy. Bilateral nasolabial folds were shallow. There was no movement of the bilateral soft palate, and the pharyngeal reflex was absent. The left extremities disclosed motor weakness, and bilateral Babinski signs were positive. Hypotonia, hypermetria and incoordination were seen in the bilateral extremities;

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more marked in the left. Deep tendon reflexes were absent throughout. There were analgesia and thermanesthesia over the left side of the body including the face.

Laboratory findings: ESR was 25 mm/1 hr and 52 mm/2 hrs. Biochemical data revealed LDH: 1,465 units, ZTT: 18.4 units, Na: 129.9 mEq/l, K: 3.05 mEq/l and Cl: 94.4 mEq/l. The urinalysis revealed positive protein. ECG showed an enlargement of the left ventricle and atrial fibrillation. EEG showed diffuse θ waves and spikes.

Examination of cerebrospinal fluids: Lumbar punctures revealed xanthochromic spinal fluid (red blood cells: 154/3) on the 13th day, and bloody spinal fluid (red blood cells: 480/3) on the 27th day with normal pressure.

CT scans: CT on the 3rd day revealed a large infarction in the region of the right middle cerebral artery, a small infarction in the right occipital lobe and a large low density in the bilateral cerebellar hemispheres (Fig. 1A). The fourth ventricle was not identified, but the lateral and third ventricles were normal in size. CT on the 15th day revealed a marked decrease in the size of cerebellar low density, with the appearance of a normal fourth ventricle (Fig. 1B).

Brachial angiography (Fig. 2): Angiography on the 6th day revealed narrowing of the distal portion of the basilar artery. Bilateral SCA and posterior cerebral arteries were incompletely filled.

Clinical course (Fig. 3): By fibrinolytic and anticoagulant therapy, the patient made a gradual recovery. On the 13th day, she could open her eyes as much as 7/9 mm. The downward movement of the eyes became normal, and bilateral Babinski signs, ataxia and left homonymous hemianopsia were improved. On the 19th day, however, the patient developed pneumonia. She ceased to follow orders for the next 6 days, and died of a hyperglycemic non-ketotic diabetic coma

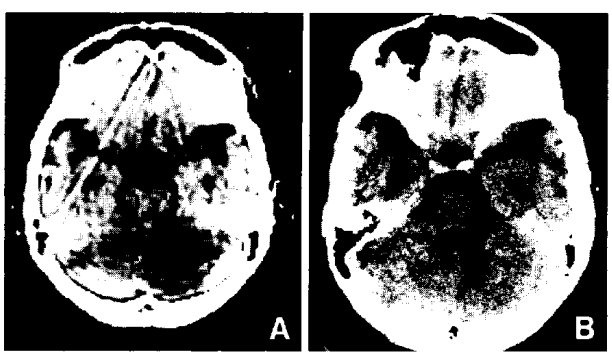

Fig. 1 A: CT scan on the 3rd day shows low density of the bilateral cerebellar hemispheres, especially on the right. B: CT scan on the 15th day shows decreased low density.

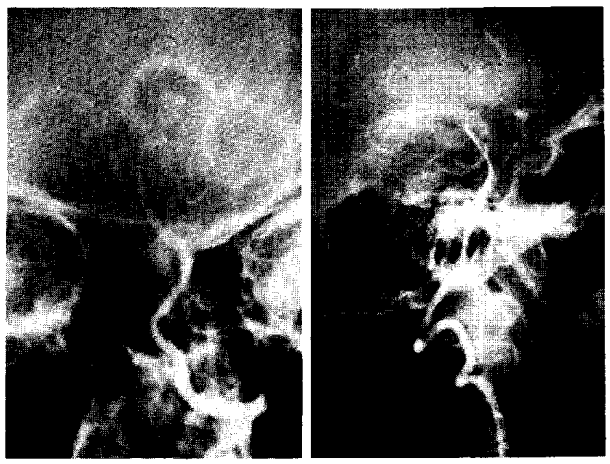

Fig. 2 Brachial angiography on the 6th day shows narrowing of the distal portion of the basilar artery.

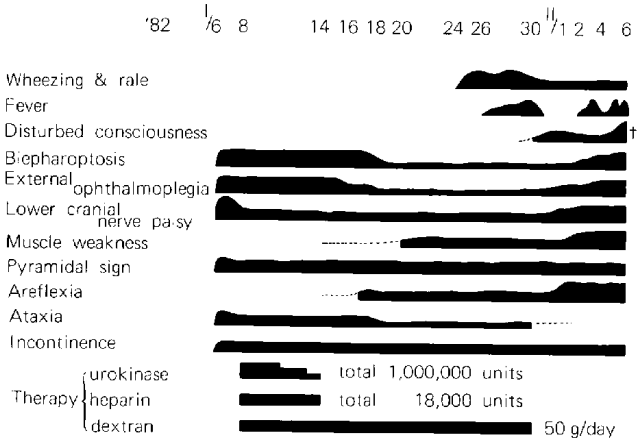

Fig. 3 Clinical course.

on the 32nd day. Blood sugar was over 500 mg/dl and urinary ketone was negative.

Neuropathological findings: There were moderate atherosclerotic involvement of the circle of Willis and aplasia of left anterior inferior cerebellar artery. Although the basilar artery showed severe atherosclerosis in its distal portion, there were no embolic occlusions of either the basilar artery or bilateral SCA.

The brain was slightly atrophic. There was an old cystic necrosis 30 × 25 mm in diameter in the region of the right middle cerebral artery. In addition, there was an old cystic infarction measuring 5 × 10 mm in the right occipital lobe. Sections of the cerebrum revealed a recent hemorrhagic infarction of the right Ammon's horn, which showed loss of neurons with an invasion of red blood cells, hemosiderin pigments and numerous foamy cells microscopically.

An old healed infarction was observed in the in-

feromedial portion of the right hemisphere of the cerebellum. The cerebellum showed a recent hemorrhagic infarction in the medial two thirds of the superior portion of the bilateral hemispheres (Fig. 4A). The left cerebellar hemisphere was reddish and the right yellowish-brown. The infarcted area was clearly circumscribed and extended as far down as the center of white matter of the bilateral hemispheres, superior to the plane of the dentate nuclei. The bilateral globose, fastigial and emboliform nuclei and the superior portion of the left dentate nucleus were destroyed. Microscopically, most of the left cerebellar hemorrhagic infarction retained an almost normal configuration (Fig. 4B). The molecular layer showed a decrease in cells. Purkinje's cells were completely necrotic. The granular layer was replaced by numerous red blood cells and foamy cells. The right cerebellar hemorrhagic infarction showed a loss of normal configuration (Fig. 4C). The molecular layer was largely destroyed, all of Purkinje's cells disappeared, and the granular layer was replaced by red blood cells.

Many sections of the brain stem were examined. Sections through the aqueduct disclosed small softenings of the right superior colliculus and the interpeduncular nucleus. The latter affected intermedullary portions of the bilateral oculomotor nerves. A section through the upper pons showed complete demyelinization of the right superior cerebellar peduncle and extensive cystic necrosis of the right pontine tegmentum which included the spinothalamic tract, lateral lemniscus, central tegmental tract, reticular formation, and bilateral medial longitudinal fasciculi (Fig. 5A). Histology of these lesions showed an ischemic infarction of several weeks duration with complete loss of neurons and myelinated fibers, and invasion of numerous foamy cells or mononuclear cells (Fig. 5B). The right locus caeruleus was also partially necrotic (Fig. 5B). The left pontine basis involving the corticospinal tract and the frontopontine tract showed slight ischemic changes (Fig. 5A). A section through the medulla oblongata disclosed pseudohypertrophy of the left inferior oliva.

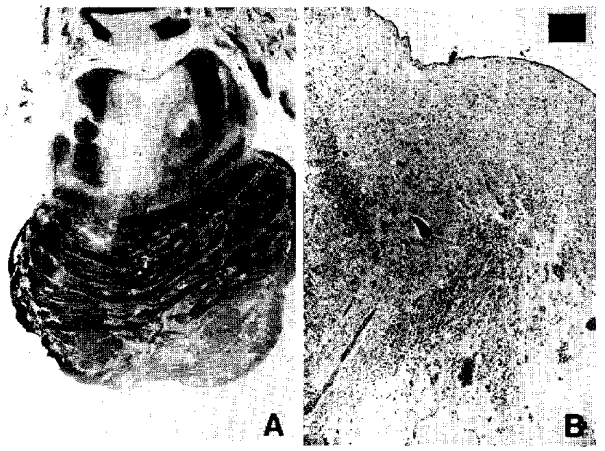

Fig. 5 A: The upper pons shows demyelinization of the right superior cerebellar peduncle, extensive cystic necrosis of the right pontine tegmentum and an ischemic change in the left pontine basis. Klüver-Barrera stain, × 2. B: Higher magnification of the right pontine tegmentum shows involvements of the bilateral medial longitudinal fasciculi and right locus caeruleus. K-B stain, × 17. IV: fourth ventricle.

Discussion

Among 5,494 autopsies of adults, Sypert and Alvord[8] found 58 cases (1.1%) of acute cerebellar infarction (30 complicated and 28 uncomplicated). Cerebellar infarctions were of the hemorrhagic type in seven of the latter. The most common site was the posteroinferior half of the cerebellum, while cerebellar infarctions located superiorly or bilaterally were rare. It was suggested from their data that bilateral and superior

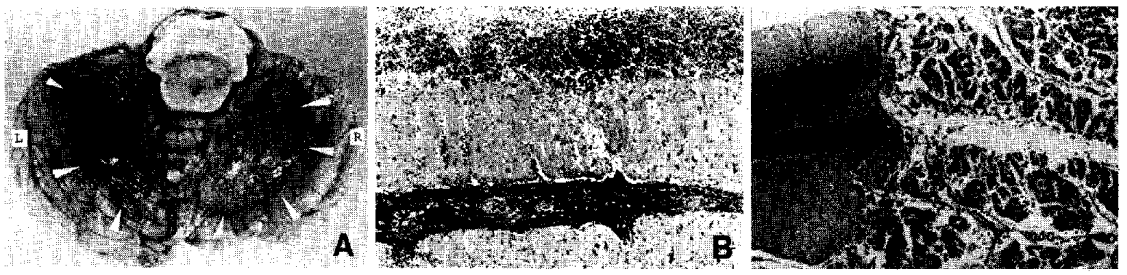

Fig. 4 A: Gross inspection of the cerebellum shows recent hemorrhagic infarction of the bilateral hemispheres. The infarcted area is more hemorrhagic on the left. B: Microscopic picture of the left cerebellum shows necrosis of Purkinje's cells and hemorrhage in the granular layer. HE stain, × 40. C: The right cerebellum shows a clear demarcation between the normal cerebellum and the infarcted area. HE stain, × 15.

cerebellar hemorrhagic infarctions as in the present case are extremely rare.

The pathologic processes in the present case consisted of both old and recent infarctions. Among the recent lesions, the infarction was ischemic in the midbrain and the pons, slightly hemorrhagic in the right Ammon's horn and the right cerebellum, and extremely hemorrhagic in the left cerebellum. In this case, recent pathologic changes disclosed an obvious asymmetry; Ammon's horn was affected only on the right side, the infarcted area in the pontine tegmentum was larger on the right than on the left, and the extent of the hemorrhage was greater in the left cerebellum than in the right. It was thus suggested that the blood flow was affected more on the right than on the left. The infarcted area in the right cerebellum appeared to be slightly hemorrhagic, presumably due to a collateral circulation from the left hemisphere.

The recent infarctions in this case seem to be embolic, because autopsy disclosed severe atherosclerosis of the top of the basilar artery, but no evidence of thrombosis. In the nine cases of occlusion of the SCA reported by Davison *et al.*,[1] pontine reticular formation, pontine nuclei and pontine basis remained intact despite the wide distribution of infarcted areas. The present case showed necrotic changes not only in the region of the SCA, but also in that of the pontine branches of the basilar artery. Moreover, brachial angiography showed narrowing of the distal portion of the basilar artery. It was suggested that the distal portion of the basilar artery became transiently embolized, affecting the bilateral SCA, pontine branches and right posterior cerebral artery.

Lehrich *et al.*[6] stated that acute cerebellar infarctions might present clinically as a posterior fossa mass with compression of the brain stem. There are two reasons why the neurological deficits in the present case recovered initially. First, the brain stem compression made a gradual recovery as the perifocal edema in the cerebellum improved. Second, the blood flow in the pons, especially in the left pontine basis recovered, presumably by recanalization of the distal portion of the basilar artery. We could not find any history of previous strokes consistent with the old infarctions in the region of the middle cerebral artery, the occipital lobe and the right lower cerebellum. However, these lesions, as well as the recent infarctions of the brain stem and the cerebellum, seem to have contributed to the revelation of interesting neurological signs in the present case. The main components in the present complicated syndrome might have resulted from three pathologic processes: *i.e.*, 1) bilateral Mill's syndrome,[7] 2) impairment of the oculomotor nerves, bilateral medial longitudinal fasciculi and the descending sympathetic pathways, and 3) pseudobulbar palsy. They might be exaggerated by compression of the brain stem.

Each neurological sign can be explained as follows. The involvement of bilateral central sympathetic pathways could cause bilateral blepharoptosis. Fisher[4] speculated that bilateral blepharoptosis was the result of bilateral central sympathetic paralysis in a basilar artery infarction or ischemia. External ophthalmoplegia might result from lesions of the right area 8 (frontal motor eye field), the left frontopontine tract and bilateral medial longitudinal fasciculi, together with impairment of the oculomotor nerves. The lesions in the right motor cortex and the left pontine basis could produce the signs of pseudobulbar palsy. The cerebellar signs of ataxia and dysmetria were seen in the bilateral extremities, and were especially prominent on the left side. It was suggested that the involvement of the left cerebellar hemisphere might play a more important role in producing the left cerebellar signs than that of the right superior cerebellar peduncle in producing the right signs. The left-sided loss of pain and temperature sensation was the result of softening of the right spinothalamic tract.

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