

Bilateral Caudate Infarct-A Case Report

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Abstract

Caudate strokes comprise only a small proportion of all subtypes of strokes. Bilateral caudate infarcts are even rarer and only a few cases have been reported in the literature. We report an 86-year-old woman with bilateral caudate infarcts. She had no past medical history of note. She presented with headache for several days and drowsiness on day of admission. Clinical examination revealed abulia, inability to comprehend or verbalize (acute mutism), right-sided neglect and right-sided hemiparesis. Computed tomographic (CT) scan brain revealed decreased attenuation in both heads of both caudate nuclei with extension across the anterior limb of both internal capsules to involve the lentiform nuclei. Echocardiography showed aortic valve sclerosis, mild mitral and aortic regurgitation and normal left ventricular function. Carotid ultrasound revealed mild stenosis of proximal right internal carotid and left distal common carotid and adjacent proximal internal carotid arteries. She showed initial improvement in the first week, but subsequently had a progressive downward course despite rehabilitation and died 44 days after her stroke. A patient with bilateral caudate infarcts is likely to have poor prognosis for rehabilitation and survival.

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Key words: Abulia, Mutism, Prognosis, Rehabilitation, Stroke

Introduction

The head of caudate nucleus forms a prominent bulge in the lateral wall of the anterior horn of the lateral ventricle. Its blood supply is from the deep penetrators from the middle cerebral and anterior cerebral arteries.¹ The caudate nucleus is thought to have an important role in behavioural functions.^{2,3} Caudate strokes comprise only a small proportion of all subtypes of strokes.⁴ Bilateral caudate infarcts are even rarer and only a few cases have been reported in the literature.^{2,3,5-7} We report a case of an elderly woman with bilateral caudate infarcts.

Case Report

SAN is an 86-year-old Chinese lady with no past history of note. Prior to her hospitalisation, she was working as a cleaner. She presented with headache of 3 to 4 days. On the day of admission, she was noted to be drowsy, less responsive and not moving the right side of her body.

On admission, she was afebrile. Her blood pressure was 160/80 mmHg and her pulse rate was 80/min and was regular. Cardiovascular, respiratory and abdominal examinations were unremarkable. She was drowsy but was able to localise pain. Her eyes were deviated to the left. The tone of the limbs was normal and the reflexes were brisk. The plantar responses were flexor. It was difficult to assess her power because of her drowsi-

ness but it was noted that the motor power on the right side was at least 3/5 and at least 4/5 on the left. Further assessment over the next few days (when she became more alert) revealed that she was abulic. There was no spontaneous speech or activity. She would just stare blankly and passively lie in bed without moving the whole day. She had difficulty understanding commands and was not able to verbalise (acute mutism). She could only occasionally nod her head to questions. She also had right-sided neglect. Self-care had to be totally assisted.

Investigations showed haemoglobin 11.4 g/dl; white blood count $3.76 \times 10^9/l$; platelets $159 \times 10^9/l$; urea 6.5 mmol/l; sodium 133 mmol/l; potassium 4.3 mmol/l; chloride 108 mmol/l; glucose 6.0 mmol/l; creatinine 65 $\mu\text{mol/l}$; creatinine kinase 74 U/l; total cholesterol 3.31 mmol/l; triglycerides 1.73 mmol/l; high density lipoprotein (HDL)-cholesterol 1.27 mmol/l; electrocardiography showed sinus rhythm with left axis deviation; chest X-ray was normal; computed tomographic (CT) scan brain (on admission and 1 week later) revealed decreased attenuation seen in both heads of both caudate nuclei with extension across the anterior limb of both internal capsules to involve the lentiform nuclei (Fig. 1); echocardiography showed aortic valve sclerosis, mild mitral and aortic regurgitation and normal left ventricular systolic function; ultrasound carotids showed

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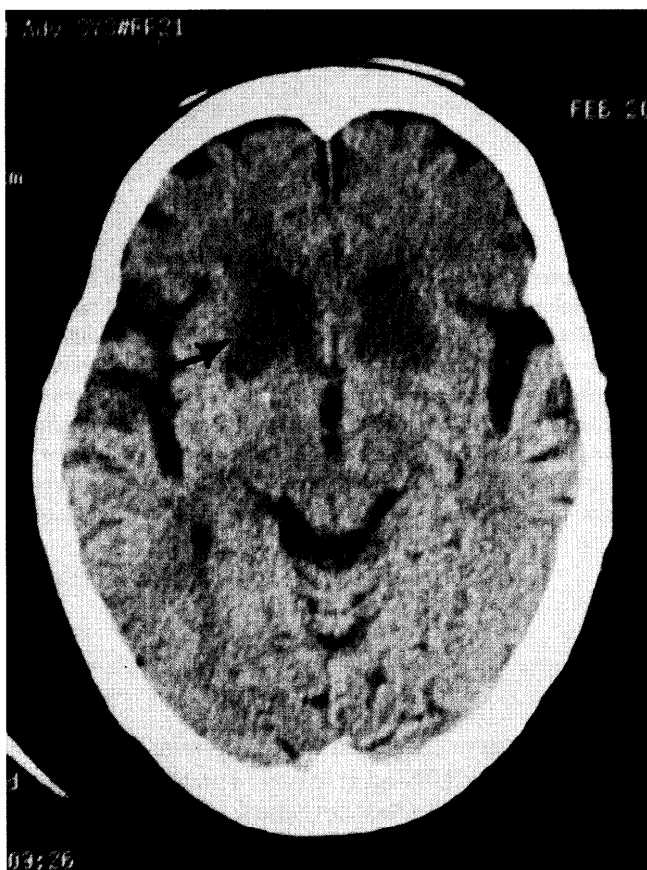


Fig. 1. CT scan brain showing bilateral caudate infarcts.

20% to 30% stenosis of proximal right internal carotid artery and 30% stenosis of left distal common carotid and adjacent proximal internal carotid arteries.

After 1 week in hospital, the patient made some progress. She required minimal assistance to stand up and required moderate assistance from another person to walk. Although she would do as supervised, she did not appear to comprehend what she was doing. At day 10 in hospital, she developed sepsis, probably from pneumonia, which was successfully treated with intravenous antibiotics. Her fever settled within a few days, repeat chest examination was clear and repeat chest X-ray and white blood cell count were normal at the end of 2 weeks of therapy. At day 17 in hospital, she developed a probable but uncomplicated non-Q myocardial infarction based on raised creatine kinase (CK-MB) levels. Despite continued rehabilitation in the ward, she progressively deteriorated in function. On day 40, she was again totally dependent in activities of daily living and unable to sit without support. She was subsequently discharged to a nursing home. She died 44 days after her stroke in the nursing home.

Discussion

Bilateral caudate infarcts are rare. In the literature, Trillet et al⁵ reported (in French) 3 cases while Richfield

et al,³ Rodier et al,² Mendez et al⁶ and Kuriyama et al⁷ (in Japanese) reported one case each. The reason why it is so rare is unknown. The precise stroke mechanism is unclear.

Caplan and associates⁸ divided caudate infarcts into several groups according to the anatomic structures involved. In their case series of 18 patients with unilateral caudate infarcts, the lesions were limited to the caudate nucleus in 4 patients, included the caudate nucleus and the anterior limb of the internal capsule in 9, and affected the caudate nucleus, the anterior limb of the internal capsule, and the anterior putamen in 5.

Most patients with caudate infarcts that have been reported were not completely evaluated for stroke mechanisms, but the majority appear to have risk factors for penetrating artery disease. In a combined series of 49 previous reports and 52 other cases from the University of Illinois (consisting mostly of unilateral caudate infarcts) studied by Caplan and Helgason,¹ 69% had hypertension, 31% had diabetes mellitus, 12% had large-artery lesions and 10% had cardiac embolic source. Our patient had previously undiagnosed hypertension.

Cognitive and behavioural abnormalities, movement disorders, neglect, dysarthria, and motor dysfunctions are often found in patients with caudate infarcts. Left reflex rhinorrhoea induced by gustation after a right caudate infarct⁹ and acute mutism⁷ from bilateral infarcts have also been reported. Some of the features that were present in our patient included abulia, inability to comprehend or verbalise (acute mutism), right-sided neglect and mild right hemiparesis. Cognitive dysfunction was difficult to assess in our patient. Contralateral limb weakness occurs when a lesion extends beyond the caudate into the anterior limb of the internal capsule and putamen, interrupting the striatopontine fibres. Though CT brain revealed bilateral infarcts extending into both of the internal capsules, it is not clear why our patient only had predominant right-sided weakness.

Patients with bilateral caudate infarcts have been reported to have more severe and more persistent behavioural abnormalities. The reason for this observation is unclear. The 3 cases reported by Trillet et al⁵ had apathy, flattened affect and lack of initiative for usual daily activities. One of the patients had prolonged akinetic episodes. The 25-year-old woman reported by Richfield et al³ had persistent behavioural disorder. The 67-year-old woman reported by Rodier et al² developed worsening state of apathy progressively and died 10 weeks after the onset of symptoms. Kuriyama et al⁷ reported another 67-year-old woman who had sudden mutism, abulia, slowness and apathy. She deteriorated despite rehabilitation and died a year after discharge. The 86-year-old lady that we reported deteriorated despite rehabilitation and died 44 days after the stroke. Apathy, uninterest, abulia and lack of motivation make rehabilitation diffi-

cult, if not impossible. They also result in self-neglect, and subject the victims to complications of immobility, e.g. pneumonia, pressure ulcers.

Conclusion

Bilateral caudate infarcts are rare. The patients often have severe and persistent behavioural abnormalities and consequently have poor prognosis for rehabilitation and survival.

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