

Monochorea Caused by a Striatal Lesion

Masayuki Ikeda^a, Hiroshi Tsukagoshi^b

^aDivision of Neurology, Department of Medicine, Asahi General Hospital, Chiba, Japan, and

^bDepartment of Neurology, School of Medicine, Tokyo Medical and Dental University, Tokyo, Japan

Key Words. Chorea · Striata · Somatotopic organization

Abstract. A 76-year-old man developed choreic movement restricted to the arm. Computed tomography suggested an old infarction mainly involving the head of the right caudate nucleus. Monochorea may indicate a striatal lesion and suggests the somatotopic localization.

Introduction

Involuntary movement of sudden onset is usually attributed to a vascular lesion in the contralateral deep hemispheric structures [1]. In the majority of recorded cases, the limbs on one side were both affected (hemiballism or hemichorea). The question of somatotopic organization within the basal ganglia has long been debated. However, monoballism and monochorea, suggesting a correlation between the specific body parts and the basal ganglia, are relatively uncommon [2–5]. The purpose of this case report is to elucidate the somatotopic organization of the basal ganglia and the diagnostic significance of monochorea.

Case Report

A 76-year-old right-handed man was admitted to our hospital to be evaluated for choreic movement of his arm. He suddenly developed left hemiparesis 10 years before. An equivocal low-density lesion in the right caudate head was indicated on computed tomography (CT). There was no hemorrhage. The result of cerebral angiography was negative and aspirin was prescribed. Within 2 months, motor weakness was alleviated, but choreic movement appeared in his left arm and hand. His symptoms remained unchanged throughout the next 10 years. Neurologic examination on admission revealed mild dysarthria and left hyperreflexia without overt weak-

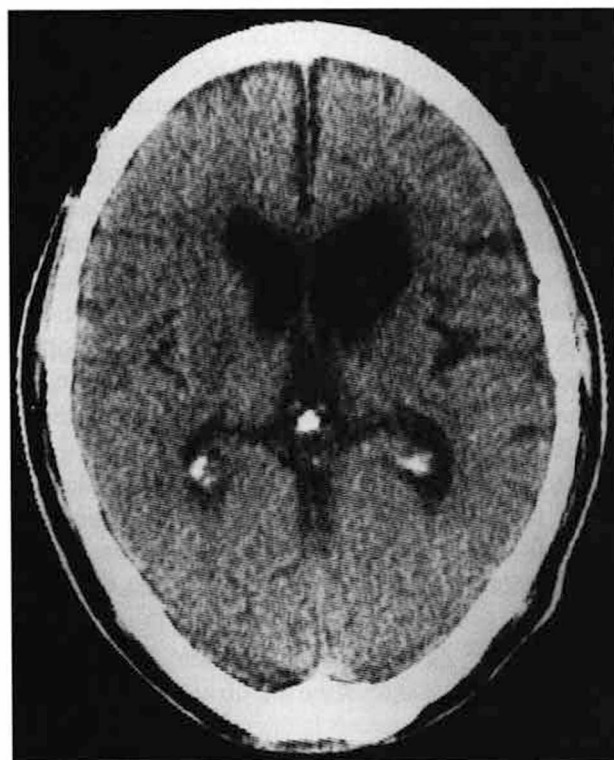


Fig. 1. CT scan reveals dilation of the anterior horn of the right lateral ventricle, indicating old infarction around the head of the caudate nucleus.

ness. Coordination and sensorium were unaffected. There was choreic movement with some dystonic component in the arm. The movement was composed of a shrug of the shoulder, flexion of the elbow and involuntary shaking hand, namely flexion and extension of metacarpophalangeal and interphalangeal joints, at 1–1.5 Hz. Concentration and volitional movement could suppress this movement, but not eliminate it. It ceased during sleep. Other extremities showed no involuntary movement. CT demonstrated an enlarged anterior horn of the lateral ventricle indicating an old infarction around the caudate head (fig. 1). There was no other lesion. EEG and somatosensory evoked potentials of short and long latency were normal. Clonazepam diminished the chorea but haloperidol, carbamazepine, diphenylhydantoin and propranolol did not affect it.

Discussion

Monochorea involves the relationship between the specific body parts and the basal ganglia, but there are few reports on it. Davison and Goodhart (2) reported a monochorea of the arm. Pathological examination identified a lesion in the putamen and caudate nucleus. Some of symptomatic dystonia is limited to one arm or leg. In most cases the principal lesion lay in the striata [3, 4]. Saris [5] described a monochorea in a patient whose hand movement is almost identical with that of the present case, namely flexion and extension of metacarpophalangeal and interphalangeal joints. A CT scan revealed an infarction of the anterior body caudate nucleus and the corona radiata. In the present case, the caudate head is the main lesion with less involvement of the putamen and internal capsule. This clinico-pathological relationship suggests topographic localization in the striata. Recent basic studies also show somatotopic organization [6]. Concerning the subdivisions of the basal ganglionic region, lesions of the gray matter alone do not always cause involuntary movements [7]. Development of chorea or ballism may need disruption of the tracts connecting the nuclei such as striatopallidal or striatonigral fibers.

In the present case, chorea appeared after the hemiparesis had been resolved. Why there should be a delay in the development of chorea following a stroke is not certain. Motor weakness itself may prevent the appear-

ance of the involuntary movement but this cannot always be true [4]. Delayed onset may be the result of slowly evolving aberrant neuronal sprouting [4].

Although it is difficult on the basis of only 1 case to draw a firm conclusion, monochorea in the hand may be symptomatic of a lesion of the striata and somatotopic localization may exist. We need to investigate it further both clinically and basically.

Acknowledgements

We are grateful to Dr. Kaichirou Tanba who referred the patient to us for evaluation.

References

- 1 Martin JP: Choreatic syndromes; in Vinken PJ, Bruyn GW (eds): *Diseases of the Basal Ganglia. Handbook of Clinical Neurology*. Amsterdam, North-Holland, 1968, vol 6, pp 435–439.
- 2 Davison C, Goodhart SP: Monochorea and somatotopic localization. *Arch Neurol* 1940;43:792–803.
- 3 Dooling EC, Adams RD: The pathological anatomy of post-hemiplegic athetosis. *Brain* 1975;98:29–48.
- 4 Marsden CD, Obeso JA, Zarranz JJ, Lang AE: The anatomical basis of symptomatic hemidystonia. *Brain* 1985;108:463–483.
- 5 Saris S: Chorea caused by caudate infarction. *Arch Neurol* 1983; 40:590–591.
- 6 Alexander GE, DeLong MR: Microstimulation of the primate neostriatum. II. Somatotopic organization of striatal microexcitable zones and their relation to neuronal response properties. *J Neurophysiol* 1985;53:1417–1430.
- 7 Richfield EK, Twyman R, Berent S: Neurological syndrome following bilateral damage to the head of the caudate nuclei. *Ann Neurol* 1987;22:768–771.

Received: August 27, 1990

Accepted: November 5, 1990

Masayuki Ikeda, MD

Wellcome Surgical Institute and Hugh Fraser Neurosciences
Laboratories University of Glasgow
Garscube Estate
Bearsden Road
Glasgow G61 1QH (UK)