A case of Parkinson's disease that occurred in camptocormia

Kazuyuki Kawamura, M.D., Yoshiko Shibuta, M.D., Takaharu Arii, M.D., Toshio Inui, M.D., Takao Mitsui, M.D.

Department of Neurology, Tokushima National Hospital, National Hospital Organization, 1354 Shikiji, Kamojima, Yoshinogawa, Tokushima 776-8585 Japan

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Abstract

The patient, a 73-year-old woman, noticed waist curve when she was 72 years old. Shaking of the left hand developed when she was 73, and Parkinson's disease was diagnosed. There was a myogenic change in an electromyogram of the paraspinal muscles of the lumbar vertebrae level. Mild swelling with fatty degeneration was detected in the chest lumbar vertebrae MRI in the paraspinal muscles. Myopathy localized in the paraspinal muscles of the waist curve it was thought that was caused by the fact.

Key Words : Parkinson's disease, myogenic change, paraspinal muscles, fatty degeneration, myopathy

Introduction

Camptocormia is extreme anteflexion posture of the chest lumbar vertebrae, which is exacerbated by rising maintenance and a walk and completely disappears in the dorsal position [1,2]. Camptocormia is rarely complicated for extrapyramidal disease such as Parkinson's disease or multiple system atrophy, stress reaction, muscular disease, a muscular junction disease, and motor neuron diseases.

As for the pathogenesis, it is thought that trunkal dystonia and muscle weakness of the paraspinal muscles are involved. In Parkinson's disease, camptocormia appears in advanced patients, 7-8 years from the onset. The frequency is considered to affect 2-18% [3,4]. An example of Parkinson's disease that occurred in camptocormia was reported recently [5]. We experienced a case in which camptocormia had already appeared one year before a diagnosis of Parkinson's.

Case report

The patient was a 73-year-old woman. A compression fracture of the twelfth thoracic vertebra had occurred when she was 55 years old. A paternal aunt had Parkinson's disease. At the age of 71, camptocormia developed; therefore she began to use a walking frame. In January, 2011, shaking of the left hand developed. In March, Parkinson's disease was diagnosed. The tremor was improved by administration of levodopa, but the camptocormia did not change. On August 19, she was hospitalized in our hospital. Her height was 147cm, the weight was 52.0 kg, body temperature was 36.3 degrees Celsius, the blood pressure was 148/80mmHg, and the pulse rate was 83

Correspondence to: Kazuyuki Kawamura , M.D., Tokushima National Hospital, National Hospital Organization, 1354 Shikiji, Kamojima, Yoshinogawa, Tokushima 776-8585 Japan Phone: +81-883-24-2161 Fax : +81-883-24-8661

beats per minute. There were no abnormalities in the systemic physical findings. In the neurologic findings, consciousness was lucid. The left half of the body exhibited muscle rigidity. The left finger had a slight positional tremor, but passive tremors were absent. There was no muscle atrophy, and there was no muscle weakness of the neck or four extremities. Long tract signs, coordination disturbance, and sensory disturbance were absent. On rising, she adopted a posture that pushed out the abdominal region slightly. When she walked a distance of several meters, camptocormia developed. During a walk, the decrease and the wince of the step were not outstanding. The retropulsion phenomenon was positive. Laboratory findings were normal including the thyroid function. The antinuclear antibody was negative. In the head MRI, the cerebrum was withered in the frontal lobe predominance. A mild white matter lesion was detected in the cerebral ventricle circumference and the cortex bottom. There was no abnormal signal of the putamen outside. Atrophy of the brainstem and cerebellum were absent.

A wedge-shaped transformation of the twelfth thoracic vertebra was shown in a backbone X-p. In a needle electromyogram, there was fiber spontaneous discharge and positive shrp waves at rest in the right paraspinal muscles of the lumbar vertebrae level. In the chest lumbar vertebrae MRI (Figure 1), the paraspinal muscles of the chest lumbar vertebrae level swelled mildly. There was the abnormality signal that is high signal at T1 and a T2-weighted images and low signal with SPIR image, suggesting fatty degeneration. The disease severity of Parkinson's disease was Yahr 3, 47 UPDRS total (part1 1, part 2 9, part 3 36, part 4 1).

Discussion

With this patient, camptocormia preceded the onset of Parkinson's disease. We consider the cause of the camptocormia below. Myositis, thyroid gland myopathy, and mitochondrial myopathy were negligible. It is very likely that the presence of the myopathy of the paraspinal column line was the cause of the waist curve. The pathogenesis of the waist curve in Parkinson's disease is not unitary, and it is important that the cause of the waist curve is examined in individual cases. Even if most cases are given an antiparkinson agent including the L-dopa preparation, the symptoms do not improve and the condition is treatment-resistant. There are some cases in which camptocormic symptoms are improved by deep brain stimulation of the subthalamic nucleus. There have been cases in which myopathy limited to the paraspinal column muscle was improved by an electromyogram, MRI, or muscle biopsy. It is thought that muscle weakness of the trunkal dystonia and paraspinal muscles is associated with the onset of the camptocormia [6-8].

The muscular strength of the trunkal line was enhanced in this patient by hospitalization rehabilitation, and the camptocormia was also improved.

The camptocormia is an important negative factor impairing convalescence in Parkinson's disease, and special measures are needed to treat it.

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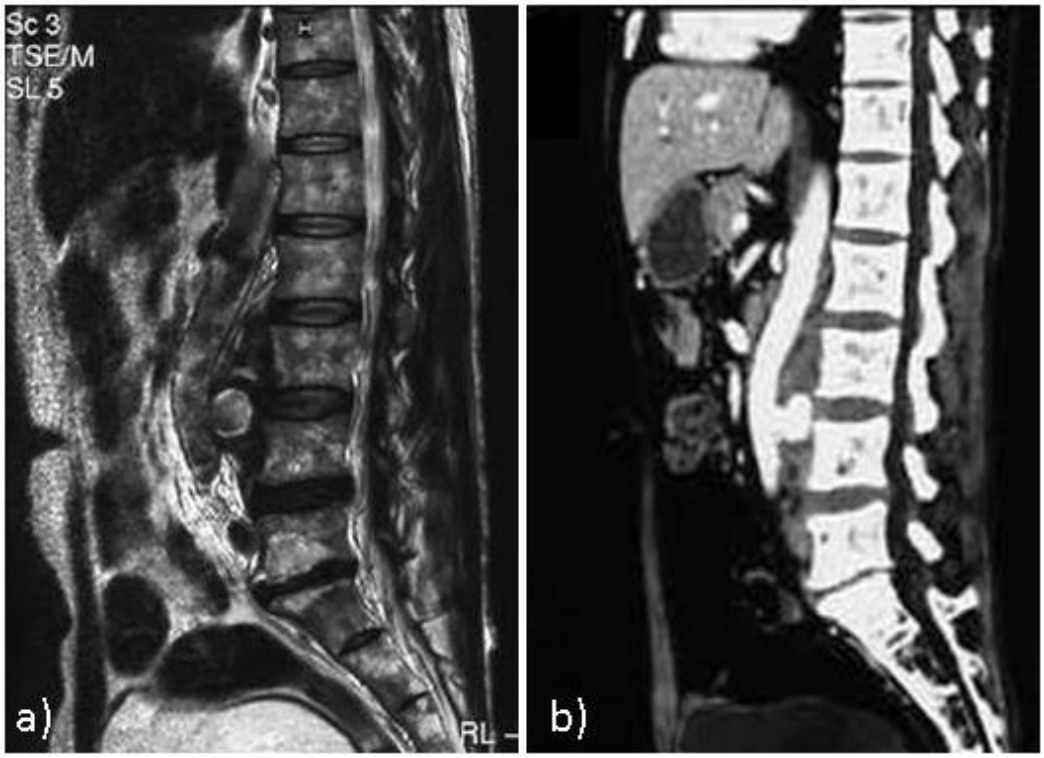

Figure1. MRI of the paraspinal muscles at the chest lumbar vertebrae level. There was the abnormality signal that is high signal at T1 and a T2-weighted images and low signal with SPIR image, suggesting fatty degeneration.