

## A case of Parkinson's disease with levodopa-responsive camptocormia

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### 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 by extrapyramidal disease such as Parkinson's disease or multiple system atrophy, stress reactions, muscular disease, muscular junction disease, or 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 after the disease onset. The frequency is considered to be 2-18% [3, 4].

### Case report

The patient was a 69-year-old man. He received a diagnosis of pemphigus in 2004 at the age of 62. His parents did not have a consanguineous marriage. There was no Parkinson's disease. In the summer of 2006, camptocormia developed. In the summer of 2007, a step shrank, and it was noticed that we became the shuffle. In October, 2007, Parkinson's disease was diagnosed, and internal use of neo-dopa stone was started. The Parkinsonism improved subsequently.

The camptocormia also improved slightly. In about March, 2011, the camptocormia increased. The patient was received by our hospital in July, 2011. There were no abnormalities in physical findings, which were: height, 176cm; weight, 60.8 kg; temperature, 36.5 degrees Celsius; blood pressure, 128/74mmHg; pulse 75 a minute. Neurologic findings showed no mentation abnormality. The limb muscles had "cogwheel" rigidity. There was no laterality. The paraspinal muscles were strong in strain of the right side. There was no resting tremor. Both fingers had a slight postural tremor. The sensory system and coordinated movement were normal. Significant camptocormia is accepted from the lower thoracic vertebra to the lumbar vertebrae. The camptocormia disappears in the dorsal position. Laboratory data were normal, including thyroid function. The antinuclear antibody was negative. A head MRI showed mild cerebral atrophy of the frontal lobe predominance. There was ventricular distention and moderate leukoariosis. There was no external indication of abnormality of the putamen, or atrophy of the brainstem or cerebellum. A backbone X-p showed compression fractures in all lumbar vertebrae. The vertebral body transformation of the 5th lumbar vertebra was extensive from the third in particular.

### Discussion

In Parkinson's disease, camptocormia often develops in the advanced stage, 7-8 years from the disease onset. It is very rare that camptocormia develops one year before a diagnosis of Parkinson's disease, as in the present patient. Regarding this patient, the possibility that camptocormia developed for myopathy and vertebral body transformation of the paraspinal muscles is unlikely. We do not think it likely that other systemic myopathy was involved. Because a tendency for improvement of the symptoms was noted with use of L-dopa, it is inferred that trunkal dystonia was the cause of the camptocormia.

## References

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