A Case of Parkinson's Disease Combined with camptocormia and compression fracture of cervical vertebrae

Ai Tsukamoto #1, Toshiaki Takeuchi #1, Yoshiharu Arii #1, Takao Mitsui #1, Shinjiro Takada #1, Syuji Hashiguchi #1

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

Abstract

A 67-year-old female patient had Parkinson's disease that developed at the age of 58, and camptocormia with cervical pain that appeared at the age of 64. She visited our hospital orthopedic and neurological department. There was no obvious fracture of the cervical spine XP at that time. In orthopedic surgery, she was diagnosed as having osteoporosis and had internal medicine treatment. We administered an anti-Parkinson's disease drug. The cervical pain occasionally increased, but it eased naturally and was tolerable. In May 2017, severe neck pain accompanied by compression fracture of the cervical vertebra appeared, and the patient was admitted. Bone fracture induction was not observed other than osteoporosis. Although rare, when neck fall of Parkinson's disease merges severe neck pain, it is necessary to keep in mind the cervical fracture. In this patient, severe cervical pain appeared on neck down of Parkinson's disease, and a cervical compression fracture was revealed. We investigated the factors that caused the compression fracture of the cervical vertebra in the falling neck of Parkinson's disease.

Case report

A 67-year-old woman started to suffer tremors in the left upper limbs in 2008. Parkinson's disease was diagnosed and internal medicine treatment was started. Left hand cognitive disorders and off symptoms gradually appeared. Neck descent appeared in January 2015, and cervical pain appeared in April of the same year. She visited the Tokushima Hospital Orthopedic Surgery, Neurological Medicine in September the same year. She underwent a cervical spine XP exam, but had no fractures. Bone density was measured, and her condition was diagnosed as osteoporosis. Treatment was started with minodronic acid hydrate. Cervical pain and headache increased in May 2017. She was hospitalized at Tokushima Hospital because her daily life was restricted. Her height was 148 cm, weight was 38 kg, and BMI was 17.35. Neurologically there was right side bending, cervical abnormal posture of front flexion, left dominant limb consolidation, motion slowing, and dyskinesia on on. At the time of Off, there was contraction, slowing of movement, and exacerbation of abnormal posture of the neck (Figure 1). The deep tendon reflexes of the four limbs increased, Babinski signs were bilateral positive. Cervical pain and headache worsened in a sitting posture, standing position and when in motion. There was no abnormality in the biochemical examination. Mild ischemic lesions of bilateral deep white matter were observed in a brain MRI. In a cervical CT, the C5 vertebral body was revealed to be flattened, slightly protruding rearward, and this compressed the spinal canal (Figure 2). A cervical vertebral MRI showed flattened C5 vertebral body similar to CT, but STIR showed no vertebra high signal. The spinal canal was stenosed, but there was no abnormal signal in the spinal cord (Figure 3). There was no obvious compressive fracture in the thoracolumbar spine XP. We used a collar (Polynec soft) for the cervical vertebrae to ease the cervical pain and ordered rest.
From various examination findings, bone metastasis of malignant tumors other than osteoporotic, bone lesion of multiple myeloma, abnormal parathyroid function, and abnormal thyroid function were found to be negative as causes of the fracture. As the neck down worsened at the time of Off, administration of rotigotine and OMT inhibitor were increased for the purpose of alleviating symptoms at off time. As a result, the neck lowering symptoms improved. Initially she was only able to move to a portable toilet, but before discharge she improved her everyday life without problems.

Discussion

The causes of neck fall of Parkinson's disease are roughly divided into two types. Firstly, it may be caused by abnormal muscle tone, i.e. dystonia [1] Secondly, there may be a muscle weakness in the posterior neck muscle group which makes the head clench. Lava and colleagues reported localized myositis in muscle biopsy of the stretcher muscles of the neck of a patient with camptocormia, which could cause neck lowering [2]. As regards the cause of neck down, in this example there was no atrophy of the cervical muscles at the cervical vertebra MRI and there was no abnormal signal and there was no rise of the muscle escape enzyme, so myositis was negative. It was considered as one symptom of dystonia symptom at off time, because neck down worsens at off time. Therefore, the cause of neck descent was considered to be dystonia. Adjustment of anti-Parkinsonian drugs improved the neck falling symptoms. Cervical vertebra compression fractures are relatively rare in compression fractures of vertebral bodies. The existence of other underlying diseases was negative, and it was considered that hyperflexion due to neck lowering and osteoporosis caused the cervical compression fracture. The bone mineral density of Parkinson's disease patients of women aged 60 years and over is markedly decreased, particularly in high-grade and severe cases [3]. There is only one case report of similar cases [4]. It should be noted that a cervical vertebral fracture is caused by neck descent of Parkinson's disease and osteoporosis in combination.

References

2. Lava NS, Factor SA: Focal myopathy as a cause of anterocollis in Parkinsonism. Mov Disord. 16; 754-756, 2001

Figure 1
At wearing-off
At wearing-on

Figure 2. Cervical CT
Figure 3. Cervical MRI