

Aggravation of Pre-Existing Cervical Spondylotic Myelopathy with Limb Paralysis Following Lumbar Puncture: A Case Report

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This case report highlights a rare occurrence of paraplegia following lumbar puncture (LP) in the presence of cervical disc herniation during cerebrospinal fluid (CSF) drainage. The patient was an elderly woman undergoing investigation for suspected neoplastic meningitis who experienced sudden acute paraplegia on the day following the LP procedure. LP is often contraindicated in patients with intracranial lesions. If patients exhibit myelopathy symptoms, such as increased lower extremity reflexes and positive Hoffmann and Trömner's signs without intracranial lesions, a thorough evaluation for occupying lesions, including cervical spondylotic myelopathy, is recommended before LP is carried out.

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Introduction

The risk of neurological deterioration following lumbar puncture (LP) performed below a spinal space-occupying lesion is relatively poorly recognized in comparison with the risk associated with intracranial mass lesions¹. Although acute neurological deterioration following LP is known to occur in some cases of spinal cord tumors², rapid worsening of neurological symptoms following LP in patients with cervical spondylotic myelopathy has not been widely reported¹.

To date, there have been no reports of the exacerbation of pre-existing cervical spondylotic myelopathy following LP in cancer patients during investigation for meningeal carcinomatosis. Abrupt changes in cerebrospinal fluid (CSF) pressure following LP below the level of the lesion can lead to impingement of the spinal mass on the spinal cord, a phenomenon termed “spinal coning”^{3,4}. We hypothesized that spinal coning was associated with the acute neurological deterioration observed in a patient we treated.

In this report, we present a case of exacerbation of pre-

existing cervical spondylotic myelopathy following LP in a cancer-bearing patient with systemic metastasis who required evaluation for meningeal carcinomatosis.

Case Report

The patient was a 56-year-old female patient who had been diagnosed with gastric cancer with bone metastasis five years previously, leading to her receiving chemotherapy. Despite receiving a variety of treatments, including bevacizumab, nivolumab, and denosumab, the patient experienced persistent numbness and leg weakness, which led to a switch to ramucirumab. However, her symptoms persisted, and she was subsequently referred to the neurology department.

During the initial consultation, the patient was conscious, and no cranial nerve symptoms were observed. In terms of motor function, both Barre and Mingazzini tests were negative, but an upper limb dexterity disorder was observed. She also exhibited significant sensory impairment in the distal parts of both her upper and lower extremities, as well as numbness of the hands. Hyperactive

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Table 1 Results of plasma and cerebrospinal fluid examinations

Blood Glucose	93 mg/dL
Cerebrospinal fluid tests	
Color	clear
Cell count	2 /mm ³
Protein	59 mg/dL
Cl	126 mEq/dL
Glucose	52 mg/dL
Cytology	class II

deep tendon reflexes were observed in the lower extremities, without any notable differences between the left and right sides. Bilateral pathological reflex with positive Hoffmann and Trömner reflexes was further observed. The finger-nose-finger test revealed clumsiness on the left side, whereas no coordination issues were observed in the lower extremities. She displayed a positive Romberg's sign, negative Mann's test result, and was unable to perform tandem gait. Her gait was wide, necessitating the use of a cane for walking.

Given the patient's history of cancer, we considered the possibility of spinal infiltration, neoplastic meningitis, and metastasis to the brain parenchyma. To further investigate this hypothesis, LP was performed. A 21-gauge needle was used and the stylet was reinserted before removing the needle. The initial LP pressure was 10 cmH₂O, while the final LP pressure was 7 cmH₂O (Ayala index was 7.0). A 13 mL sample of cerebrospinal fluid was collected, and the LP procedure proceeded smoothly. Cerebrospinal fluid protein levels were slightly elevated, but the cell count was normal (**Table 1**). Cytological examination of the cerebrospinal fluid revealed Class II findings. After examination, the patient was discharged without any complications. However, the night before, she had experienced pain from her lower back to the left leg. Over time, progressive weakness in the left lower extremity worsened, making walking difficult. Consequently, she was urgently readmitted to the hospital on the fourth day after the LP.

Upon readmission, she presented with quadriplegia, predominantly in the legs, as well as sensory deficits in both the upper and lower extremities. She further reported thermal hypoalgesia in her extremities. In terms of motor function, the patient was able to lift her lower right leg, but her left leg exhibited weakness, making kneeling and standing challenging (MMT score was 4/5 on the right side and 3-/5 on the left side). Blood tests revealed evidence of an inflammatory response, indicat-

Table 2 Blood test results on readmission

WBC	8,810 /μL
RBC	382×10 ⁴ /μL
Hb	11.5 g/dL
Plt	23.6×10 ⁴ /μL
Alb	2.7 g/dL
AST	25 IU/L
ALT	21 IU/L
Na	139 mEq/L
K	4.3 mEq/L
Cl	104 mEq/L
Ca	7.6 mg/dL
LDH	227 IU/L
T-Bil	0.43 mg/dL
BUN	8.9 mg/dL
Cr	0.51 mg/dL
CRP	4.40 mg/dL

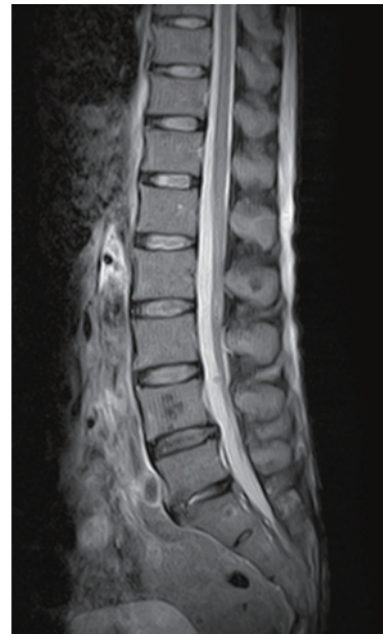


Fig. 1 MRI T2-weighted image of the lumbar spine. The image shows mild disc protrusion at L4/5, but no hematoma.

ing a urinary tract infection due to bladder dysfunction (**Table 2**). At the time of admission, a hematoma in the spinal canal as a result of the LP was suspected. However, MRI of the lumbar spine showed no evidence of a hematoma (**Fig. 1**). Considering the hyperreflexia in the lower extremities, MRI of the cervical spine was performed, which revealed cervical spondylotic myelopathy with severe stenosis at the C4/5 and C6/7 levels (**Fig. 2**). The patient was diagnosed with spinal shock secondary to cervical myelopathy. She was subsequently evaluated

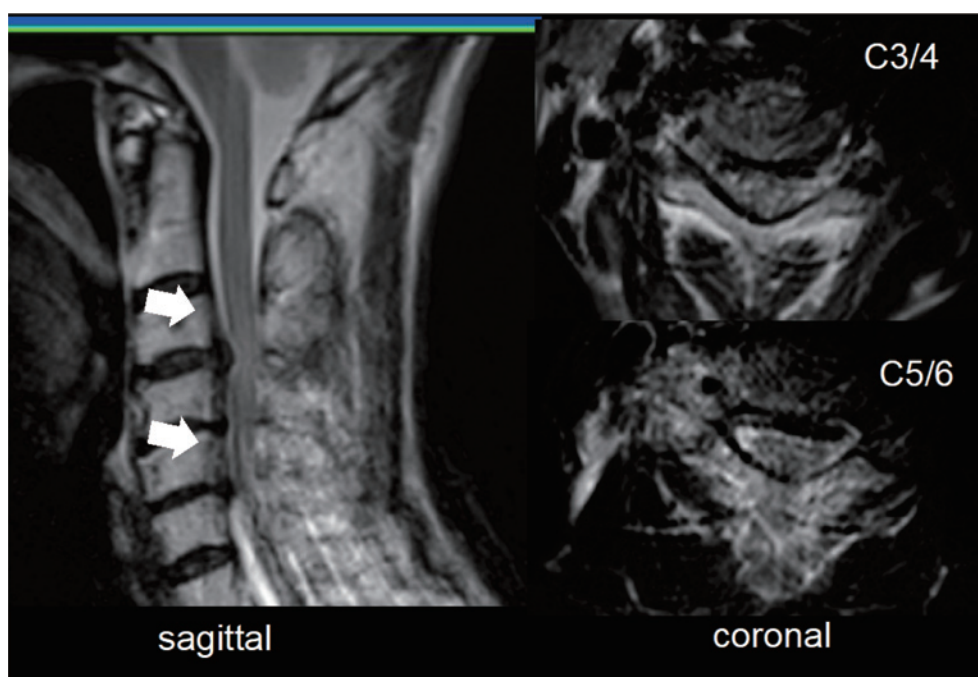


Fig. 2 MRI T2-weighted images of the cervical spine

We observed cervical spondylotic myelopathy with severe stenosis at the C3/4 and C5/6 levels. The disc osteophytes are indenting the ventral aspect of the spinal cord, causing narrowing of the spinal canal (arrows).

by an orthopedic surgeon and transferred for cervical myelopathy surgery. Following surgery, numbness persisted only in the hands. She demonstrated an improved ability to perform activities of daily living that she had been previously unable to perform, resulting in an improvement in her overall functional status.

Discussion

Herein, we present a case of suspected meningeal carcinomatosis in a patient who was undergoing chemotherapy for gastric cancer. The patient underwent LP during chemotherapy, which resulted in acute quadriplegia. Neurological symptoms associated with meningeal carcinomatosis include numbness in the lower extremities due to infiltration of the intracranial and spinal cord areas. LP is generally considered safe, but various complications may still arise even if standard infection control measures and proficient techniques are employed. Headache and backache are the most commonly documented adverse effects of LP, with headache reported as manifesting in as many as 60% of patients undergoing the procedure, although estimates vary owing to discrepancies in the inclusion criteria and definition of headache⁵. Other studies have shown that transient headache is the most prevalent symptomatic complication, occurring in 11-70% of cases, with fewer than 1% of patients experi-

encing additional complications⁶⁻⁸.

In cancer patients, LP may be promptly performed following the appearance of neurological symptoms. The risk of LP in cases of elevated intracranial pressure caused by mass lesions is well recognized. However, the risk of complications in patients with space-occupying spinal cord lesions remains poorly understood^{1,2}. Meningeal carcinomatosis resulting from direct invasion of the spinal cord can lead to quadriplegia.

Several hypotheses have been proposed regarding the mechanisms underlying acute neurological deterioration following CSF drainage with spinal cord compression. One hypothesis is that the spinal cord loses its original cushioning effect after LP and loss of cerebrospinal fluid, leading to spinal shock⁹.

In this case, we speculated that neurological deterioration was caused by spinal cord compression due to positional changes in a preexisting cervical spondylotic herniation following LP. Several previous studies^{1,9,10} have documented neurological deterioration in three patients following lumbar puncture with pre-existing cervical spondylosis (Table 3).

Clinicians must be aware of the risk of LP in the presence of increased intracranial pressure due to mass lesions. However, the risk of neurological deterioration following LP in patients with spinal cord compression is

Table 3 Reports of Neurological Deterioration Following Lumbar Puncture with Pre-existing Cervical Spondylosis

Age	Gender	Diagnosis	Time interval	Treatment	Outcome	Paper
42	male	cervical disc herniation	4 hr	operation	recovered completely	Doh et al. ¹
66	male	cervical myelopathy	5 hr	operation	recovery	Lee et al. ¹⁰
Unknown	male	cervical spondylotic myelopathy	24 hr	operation	recovery	Bao et al. ⁹
56	female	cervical myelopathy	12 hr	operation	recovery	

not well known. In 1940, the first report of neurological worsening after LP involving a spinal epidural neurofibroma was published²⁴. Hollis et al.² reviewed 50 patients with complete spinal subarachnoid block who underwent LP for myelography, reporting an incidence of neurological deterioration of at least 14% after LP below a spinal block. According to this report, all seven patients who exhibited neurological deterioration after spinal block showed some degree of weakness, with severe weakness observed in two and sphincter difficulty in three. Among these seven patients, rapid deterioration within 30 min of lumbar puncture was observed in only one, whereas the others showed deterioration over periods ranging from 1 to 4 days. According to Hollis et al.², this suggests sustained leakage of cerebrospinal fluid from the puncture site over the course of several days. However, the underlying mechanism remains to be fully elucidated, meaning that additional studies are required to fully understand these phenomena.

Although not performed in this case, the Queckenstedt test, a method involving compression of the jugular veins, can be used to detect blockages in the spinal cord passage. If there is a blockage in the CSF flow, the typical fluctuation in pressure is disrupted, resulting in a positive Queckenstedt sign. However, no studies have yet demonstrated the accuracy of this examination. Therefore, it is crucial to conduct a thorough evaluation of the patient's history and to perform a neurological examination with careful consideration of the potential for cervical spondylopathy before proceeding with LP to mitigate the risks associated with the procedure.

In conclusion, acute quadriplegia is a potentially hazardous complication of LP in patients with cervical myelopathy. Clinicians should obtain a comprehensive medical history, perform neurological examinations, and consider cervical spine MRI as a precautionary measure before performing LP. If cervical lesions cannot be ruled out based on neurological findings, cervical MRI should be conducted before proceeding with LP.

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