Urinary Retention Secondary to *Listeria* Meningitis

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Abstract

We report a rare case of urinary retention secondary to *Listeria* meningitis. A 90-year-old woman presented with high fever, nausea, diarrhea, and incontinence of urination and feces. Lumbar puncture was performed. The total cell number of the cerebrospinal fluid (CSF) was elevated indicating that the glucose level was decreased. A CSF culture and a blood culture revealed *Listeria monocytogenes* (*L. monocytogenes*). We treated this bacterial meningitis with antibiotic medicine. One month after onset, stiff neck and laboratory data greatly improved and only urinary retention continued. Lumbar magnetic resonance imaging (MRI) showed no responsible lesions for the urinary retention. She received urological examination for urinary retention and was diagnosed with a neurogenic bladder. Two months later, she was able to walk after rehabilitation. However, the urinary retention continued despite urological therapy. We are not aware of descriptions on urinary retention resulting from bacterial meningitis in the recent literature. This is a rare case of prolonged urinary retention caused by bacterial meningitis.

Key words: *Listeria* meningitis, urinary retention, bacterial meningitis

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Introduction

Listeria monocytogenes (*L. monocytogenes*) is isolated from soil, milk, and raw meat, and often causes outbreaks of food poisoning (1). The incidence rate of listeriosis in the world is 0.2 to 7.4 per one million (2). It is well known that infants and the elderly are easily infected. *Listeria* meningitis accounts for 3%-4% of all cases of adult bacterial meningitis, and has many types of acute complications as compared to other types of meningitis (3-7). However, there has been no description of urinary retention in the recent literature (8). Here, we report a rare case of urinary retention secondary to *Listeria* meningitis.

Case Report

A 90-year-old woman with a history of iron-deficiency and anemia complained of fever, nausea, diarrhea, and incontinence of urination and feces with difficulty in ambulation. She was admitted to our hospital on day 3 after the on-

set. Her general physical examination showed no abnormalities. On neurological examination, her cranial nerves were intact. Motor examination revealed 4/5 strength in both lower extremities. Deep tendon reflexes were normal, and no pathological reflexes, ataxia or sensory disorders were noted. She had a stiff neck, but Kernig's sign was absent. Laboratory data showed a white blood cell (WBC) count of 7,200/ μ L, and C-reactive protein (CRP) count of 11.24 mg/dL. At lumbar puncture, the cerebrospinal fluid (CSF) pressure was 180 mmH₂O. Cell count was 939/ μ L (neutrophil 384, lymphocyte 555). The CSF protein concentration of 277 mg/dL was elevated and glucose level of 52 mg/dL (blood glucose 172 mg/dL) was decreased. Cranial computed tomography (CT) showed no parenchymal abnormality. Gram staining of the CSF showed few gram-positive rods. From the time of admission, she was treated with ceftriaxone 2 g/day as empirical therapy for bacterial meningitis. CSF culture and blood culture revealed *L. monocytogenes*. We changed ceftriaxone for ampicillin 2 g/day and gentamicin 80 mg/day. A urinary catheter was inserted on the third day because of urinary retention after admission.

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Figure 1. Clinical course of the patient. ABPC: ampicillin, CRP: c-reactive protein, CSF: cerebrospinal fluid, CTRX: ceftriaxone, FRPM: faropenem, GM: gentamicin, *L. monocytogenes*: *Listeria monocytogenes*, MINO: minocycline, WBC: white blood cell.

Laboratory data on the eighth day showed pancytopenia. In consideration of a side effect from ampicillin, only gentamicin was used. Laboratory data on the 22nd day showed a WBC count of 2,800/ μ L, and a CRP count of 0.46 mg/dL. We changed her medication from intravenous use of antibiotic medicine to internal use of faropenem 600 mg/day. Stiff neck and laboratory data greatly improved and only urinary retention continued. However, her superficial and deep sensations were decreased in her bilateral distal lower extremities. The distribution of hypesthesia was corresponding to S 2-3 segments. The site of lesion was thought to be in the sacral region. Lumbar magnetic resonance imaging (MRI) was performed to check for organic factors of urinary retention, but there was no lesion responsible for the urinary retention. On the 24th day of illness, she underwent a urological examination for urinary retention, and was diagnosed with a neurogenic bladder. She had slight micturition and overflow incontinence. Postvoid residual volume was 300 mL. Combined use of medical therapy with distigmine 10 mg/day and urapidil 30 mg/day was started. In spite of use of faropenem for 17 days, CRP did not return to negative in

laboratory data and the total cell number of CSF did not decrease on the 38th day of illness. Thus, internal use was replaced with minocycline 200 mg/day. CRP turned negative and the total cell number of CSF finally decreased to 14/ μ L (neutrophil 1, lymphocyte 13) on the 45th day of illness. Although urinary retention remained and forced intermittent self-catheterization was used, she was able to walk after rehabilitation, and was discharged from the hospital on the 62nd day of illness (Fig. 1).

Discussion

This patient almost completely recovered from *Listeria* meningitis after two months; however, urinary retention remained. Urinary retention caused by aseptic meningitis is well known, and includes Elsberg syndrome, which occurs with herpes virus meningitis. The recent literature does not contain descriptions on urinary retention resulting from bacterial meningitis, apart from those with tubercular meningitis (8). Of 46 reports we found after a literature search, 42 cases involved urinary retention due to aseptic meningitis, 3

involved tubercular meningitis, and 1 was a result of spinal cord abscess. Precentral gyrus of the brain cortex, pons, sacral cord, and centrifugal nerves from the sacral cord were believed to exist in the deficit regions, which caused urinary retention. In the present case, the sacral cord and centrifugal nerves were suspected as the deficit regions because the patient had sensory disturbance at the sacral level, a decrease of micturition and remaining urine impression, without a serious brain condition, such as conscious deficit, agnosia, or

aphasia. In cases of urinary retention due to the aseptic meningitis in our literature search, almost all cases improved within one month, except for one case that required six months, and no cases reported sequelae. Cranial nerve deficit from bacteria is thought to be stronger than that from viruses; therefore, the prognosis of urinary retention caused by bacterial meningitis may be worse than that by aseptic meningitis. In conclusion, we report a rare case of prolonged urinary retention caused by bacterial meningitis.

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