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【 CASE REPORT 】

Acute Urinary Retention Induced by Chemical Meningitis Which Occurred Due to a Ruptured Dermoid Cyst

Keiko Yamagami, Naoko Kakuta, Kaori Seki, Ryota Nakamura, Yusuke Hanioka, Tomoyuki Nakamura and Hitoshi Goto

Abstract:
Meningitis retention syndrome (MRS), a rare complication of aseptic meningitis, can present with acute urinary retention. The rupture of a dermoid cyst, which is a benign intracranial tumor, can sometimes induce chemical meningitis. We herein present a case of chemical meningitis and acute urinary retention that was induced by the rupture of a dermoid cyst. The patient experienced urinary retention for approximately 60 days, and then made a complete recovery thereafter. This is the first reported case of acute urinary retention due to the rupture of a dermoid cyst.

Key words: dermoid cyst rupture, meningitis retention syndrome, chemical meningitis

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Introduction

Acute urinary retention due to meningitis retention syndrome (MRS) is a rare complication of aseptic meningitis. Acute urinary retention can occur during the clinical course of meningitis with mild pyramidal involvement, and recovers within several days to weeks (1). A dermoid cyst is a benign intracranial tumor. Spontaneous tumor rupture is a rare phenomenon that can induce chemical meningitis (8.2%) (2). We herein present a case of chemical meningitis and acute urinary retention that was induced by the rupture of a dermoid cyst. Our case is the first report of acute urinary retention induced by the rupture of a dermoid cyst.

Case Report

A 41-year-old man was admitted to our hospital with consciousness disturbance, urinary retention, and gait disorder. He had developed a fever of 38℃, 12 days previously, which did not respond to antimicrobial therapy. Three days before admission, he complained of progressive difficulty in urination and gait disorder. One day before admission, his lower abdomen appeared distended; thus, a urinary catheter was inserted and 1,000 mL of urine was drained. On admission, he was semiconscious, closing his eyes without stimuli, and could not explain his condition. Kernig's sign was positive, and he had neck stiffness, and accelerated tendon reflexes. His body temperature was 37.8℃, his heart rate was 76 beats per minute, his blood pressure was 105/55 mmHg, and his oxygen saturation was 96% on ambient air.

The laboratory findings were as follows: white blood cell count, 8,120/mm^3 with a differential count of 74.7% neutrophils and 14.5% lymphocytes; and C-reactive protein, 0.03 mg/dL. His human immunodeficiency virus (enzyme immunoassay) status was negative. The other laboratory findings, including his liver function and renal function, were almost normal. His cerebrospinal fluid (CSF) pressure was 23 mmHg, with 14 white blood cells/μL (differential count: 95% mononuclear leukocytes and 25% polymorphonuclear cells). His CSF glucose level was 46 mg/dL (39% of the serum glucose level), while the protein level was elevated to 114 mg/dL. Ceftriaxone, vancomycin, and acyclovir were initially administered due to a preliminary diagnosis of bacterial and herpes meningitis. The urinary catheter remained in place because he remained unable to urinate.

T1-weighted cranial magnetic resonance imaging (MRI) revealed multiple fat and lipid droplets, as high intensity signals, and small multilocular fatty masses in the left cavernous sinus (Figure). He was therefore diagnosed with chemi-

Department of Internal Medicine, Osaka City General Hospital, Japan
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Correspondence to Dr. Keiko Yamagami, yamasanz@qb3.so-net.ne.jp

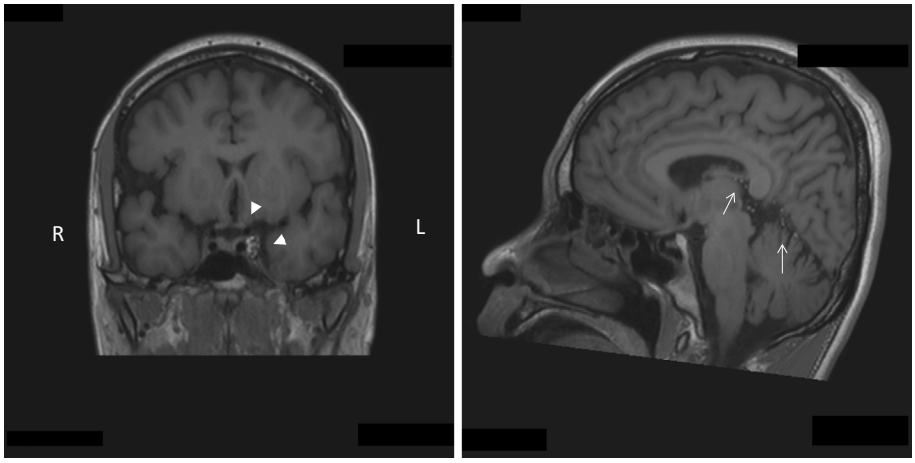

Figure. **T1-weighted cranial MRI revealed multiple fat and lipid droplets, as high intensity signals (arrows), and small multilocular fatty masses in the left cavernous sinus (arrowheads).**

cal meningitis induced by the rupture of a dermoid cyst. On the third day after admission, methylprednisolone (250 mg, intravenous) was administered for three days to treat his chemical meningitis, after which prednisolone (60 mg, oral) was administered and gradually tapered. Although the fever continued, his consciousness level improved on the fifth day after admission. Blood and CSF cultures were negative for bacterial infection. Furthermore, since CSF virus polymerase chain reactions were negative for herpesvirus types 1 and 2, cytomegalovirus and *herpes zoster virus*, ceftriaxone, vancomycin and acyclovir were discontinued on the seventh day.

The urinary balloon catheter was removed on day 7, but it was reinserted as he was unable to urinate after its removal. Lumbar MRI performed at this time revealed no abnormal findings. On the tenth day after admission, a CSF examination indicated that his myelin basic protein (MBP) level was increased to 219 pg/mL (normal <102) and a positive oligoclonal band. We therefore diagnosed his acute urinary retention as MRS induced by chemical meningitis. The administration of prednisolone was discontinued on the twentieth day after admission and the patient was transferred to another hospital on the thirty-third day for rehabilitation. At approximately 60 days after admission, he was observed to have made a complete recovery from urinary retention.

Discussion

The cause of acute urinary retention during MRS is thought to be inflammation of the sacral spinal nerve roots or demyelination of the peripheral or central neurons. However, our case was not typical MRS. First, in typical MRS, only mild pyramidal and meningitis symptoms are present, such as headache, fever, and neck stiffness, without consciousness disturbance or convulsions (1). Moreover, the clinical symptoms of aseptic meningitis are sometimes mild or absent, while urinary retention is occasionally observed (3). In our case, the consciousness disturbance and gait disorder appeared simultaneously early in the clinical course and continued for a long time. Second, in typical MRS, acute urinary retention occurs approximately 1-17 days after the onset of meningitis. The recovery time from acute urinary retention is approximately 6-61 days (4). In our case, recovery from urinary retention took approximately 60 days, which is long in comparison to the previously reported MRS cases (3-5). The initial disturbance of consciousness in our case seemed to indicate severe meningitis, similar to acute disseminated encephalomyelitis (ADEM) (6); thus, it was assumed that the recovery from urinary retention would take more time than previously reported.

In MRS patients, the brain and spinal MRI scans and neurogenic examinations usually reveal normal findings. CSF examinations sometimes reveal elevated levels of MBP in the CSF, with a positive oligoclonal band, as was observed in our case; thus, MRS is considered to be a very mild form of ADEM (1). Assuming that this is indeed the case, prednisolone treatment was a reasonable therapeutic choice for our patient, as it is used in the treatment of ADEM.

Intracranial dermoid cysts, which account for 0.04-0.7% of intracranial tumors, are rare. They contain lipids, fat, hair follicles and sweat glands (7). A rupture is a rare event, and usually occurs either spontaneously or secondary to trauma (8, 9). Although the pathophysiology of ruptures is not clearly understood, it results in the spread of fat and lipid droplets from the cyst into the subarachnoid and intraventricular spaces, resulting in the symptoms of aseptic meningitis. The clinical symptoms of a rupture include headache (32.6%), seizures (26.5%), cerebral ischemia (16.3%), and chemical meningitis (8.2%) (2).

To facilitate a patient's recovery from neurological dysfunction, large residual dermoid cysts should be treated by surgical resection (10). In our case, lipid droplets and small multilocular fatty masses in the left cavernous sinus were the only visible abnormalities on MRI. Thus, prednisolone was administered without surgical resection in order to alle-

viate the symptoms of chemical meningitis. Although dermoid cysts can accompany neurogenic bladder dysfunction as congenital sacral malformation, the course of our patient was acute, and his bladder dysfunction disappeared completely after the administration of steroids. The administration of prednisolone as a treatment for ruptured dermoid cysts has been previously reported (9, 11, 12); however, the therapy is temporary and the appropriate dose and duration of treatment have not been determined.

Ruptured dermoid cysts can be diagnosed by computed tomography (CT) or MRI. T1-weighted MRI of the brain shows the dissemination of cholesterol droplets-which show a high signal intensity-in the subarachnoid space (10). Before their rupture, dermoid cysts sometimes grow asymptomatically in the intracranial compartment and their spontaneous rupture is sometimes the first clue of their presence-as occurred in our case (8).

In summary, we herein presented a case of chemical meningitis induced by the rupture of a dermoid cyst, which led to urinary retention. In the present case, the patient successfully recovered from his clinical symptoms after treatment with prednisolone.

The authors state that they have no Conflict of Interest (COI).

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