Acute Severe Spinal Cord Dysfunction in Bacterial Meningitis in Adults

MRI Findings Suggest Extensive Myelitis

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Background: Bacterial meningitis is rarely complicated by acute spinal cord involvement (eg, myelitis, ischemic infarction, spinal abscess, or epidural hemorrhage). In spinal cord dysfunction, magnetic resonance imaging (MRI) is the imaging modality of choice. Still, MRI findings of myelitis due to bacterial meningitis in adults have not been reported.

Methods: Spinal MRIs were obtained during the acute stage of meningitis and on follow-up in 3 adults with bacterial meningitis that was complicated by paraparesis or tetraparesis and bowel and bladder incontinence. The causative pathogens were Streptococcus pneumoniae and Neisseria meningitidis; in 1 patient, the pathogen was not identified.

Results: In all cases, spinal MRI ruled out a compression of the cord by an extramedullary mass but demonstrated hyperintensities on T2-weighted images that predominantly involved the gray matter and extended from the cervical to the lumbar cord. Leptomeningeal and discrete nodular intramedullary enhancement on T1-weighted images was detected only in 1 patient. Follow-up examinations revealed that hyperintensities resolved completely in 1 patient, while a central cavitation developed in the cervical spinal cord of another, and the MRI findings were progressive during the first 4 weeks in the third patient. In all cases, severe paresis and bowel and bladder incontinence persisted.

Conclusion: We demonstrate for the first time the MRI findings of adults with acute spinal cord involvement during bacterial meningitis. Magnetic resonance imaging showed central intramedullary hyperintensities on T2-weighted images that extended from the cervical to the lumbar cord, indicating myelitis. Clinical follow-up examinations suggest that myelitis during bacterial meningitis has an unfavorable prognosis.

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Bacterial meningitis is frequently accompanied by intracranial complications, such as cerebrovascular involvement, brain edema, hydrocephalus, or hearing impairment, as well as systemic complications, such as septic shock, adult respiratory distress syndrome, or disseminated intravascular coagulation.1-3 Spinal cord involvement is a rare complication of bacterial meningitis.4 Besides cord compression by a spinal abscess or epidural hemorrhage following lumbar puncture, the cord can be affected by ischemia due to vasculitis, shock, herniation, or arachnoiditis, and by myelitis.5 Spinal magnetic resonance imaging (MRI) during the acute stage of spinal cord dysfunction has been reported in only 2 children: no abnormalities were detected in one child,6 and enhancement of the cauda equina and lumbosacral nerve roots was seen in the other.7

We describe 3 patients who developed severe spinal cord dysfunction during the acute stage of bacterial meningitis and their MRI findings during this condition.

REPORT OF CASES

CASE 1

A 36-year-old woman was admitted to a local hospital with a 4-day history of fever, back pain, and weakness of both legs.

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Three years previously, she had undergone splenectomy for idiopathic thrombocytopenic purpura, and 3 months prior to admission, she had received her last pneumococcal vaccination. On admission, the patient was febrile, agitated, and
arms and 2/5 in the legs). Sensibility was intact, but bladder incontinence were present. Seventeen days after onset, the patient was transferred to our department. Despite repeated corticosteroid treatment, the spinal cord dysfunction was progressive until 4 weeks after onset. Seven weeks after onset, the patient still had a flaccid tetraparesis (muscle power grade, 4/5 in the arms and 2/5 in the legs) and a sensory level at T8 were noted. The patient was given intravenous prednisolone (150 mg/d for 3 days, then 75 mg/d) for suspected myelitis. Seventeen days after onset, the patient was transferred to our department.

Because myelitis was suspected, the patient was given intravenous dexamethasone (24 mg/d) for 4 days. The clinical course was further complicated by ischemic infarction in the right fronto- lobe due to cerebral vasculitis. Three months after the onset of disease, the patient still had a flaccid tetraparesis (muscle power grade, 4/5 in the arms and 2/5 in the legs). Sensibility was intact, but bladder and bowel control were still absent.

CASE 2

A 33-year-old man who had been hospitalized because of bronchitis and mild graft-vs-host disease of the liver following allogeneic bone marrow transplantation for chronic myeloid leukemia 6 months previously developed fever and headache despite oral antibiotic therapy (amoxicillin, ciprofloxacin, and fluconazole). Within 24 hours of admission, weakness of both legs and bowel and bladder incontinence appeared. Examination revealed discrete neck stiffness, spastic paraplegia, and a sensory level at C1. The CSF contained 2976 cells/µL (94% granulocytes, no malignant cells), the protein level was 1.07 g/L, and the CSF glucose level was less than 40% of the serum glucose level. There were no findings on Gram stain of CSF, and cultures of blood and CSF were sterile. Transcranial Doppler sonography and cranial MRI were normal. Nosocomial bacterial meningitis was suspected, and the patient was treated with intravenous vancomycin, meropenem, metronidazole, and a 4-day regimen of oral dexamethasone, 24 mg/d. Six weeks after onset of meningitis, follow-up examination showed a motor level at C8, with a power grade of 4/5 in the small muscles of the hand and 2/5 in the legs. No clear sensory level could be determined, but sensibility and vibratory sense were decreased in both legs. Bowel and bladder incontinence were still present.

CASE 3

A previously healthy 17-year-old boy was admitted to a local hospital with a 1-day history of fever, nausea, and headache. Examination revealed no focal neurologic deficit, but a discrete neck stiffness and a petechial rash on the trunk and extremities. Shortly after admission, he had a respiratory arrest, at which time no blood pressure could be detected. The patient required mechanical ventilator support, sedation, and treatment with vasopressors. Intravenous treatment with ceftriaxone was started. His CSF contained 300 cells/µL (71% granulocytes), the protein level was 1.66 g/L, and the CSF glucose level was less than 40% of the serum glucose level. There were no findings on Gram stain and cultures from CSF and blood, but DNA from Neisseria meningitidis was detected in the CSF and blood by polymerase chain reaction. When sedation and ventilator support were discontinued 5 days after admission, flaccid paraplegia and a sensory level at T8 were noted. The patient was given intravenous prednisolone (150 mg/d for 3 days, then 75 mg/d) for suspected myelitis. Seventeen days after onset, the patient was transferred to our department. Despite repeated corticosteroid treatment, the spinal cord dysfunction was progressive until 4 weeks after onset. Seven weeks after onset, the patient still had a flaccid tetraparesis (muscle power grade, 4/5 in the arms and 2/5 in the legs) and a sensory level at T8. Furthermore, bowel and bladder incontinence were present.

METHODS

In all patients, MRI was performed on a 1.5-T MRI scanner (Magnetom Vision; Siemens, Munich, Germany). Detailed information on the sequences is given in the figure legends.

RESULTS

For all 3 patients, T2-weighted images showed intramedullary hyperintensities during the acute stage of spinal cord dysfunction (day 5 of meningitis/day 5 of spinal cord dysfunction in patient 1; day 2 of meningitis/day 2 of spinal cord dysfunction in patient 2; day 6 of meningitis/after day 1 of spinal cord dysfunction in patient 3), which appeared to be most pronounced in the gray matter. The signal abnormalities extended from the cervical to the lumbar cord in patients 1 and 2 and from the cervical to the thoracic cord in patient 3 (Figure 1A and Figure 2). While swelling of the spinal cord was clearly evident in patient 3, it was not observed in the other patients. Intense leptomeningeal and discrete nodular intramedullary gadolinium enhancement was observed in patient 1 but not in patients 2 and 3 (Figure 1A). On native T1-weighted images, the central spinal cord lesion appeared hypointense in patient 1 and isointense in patients 2 and 3. In none of the patients did we detect an intramedullary abscess or compression of the cord by a mass, such as an epidural hemorrhage, extramedullary abscess, or subdural empyema.

On follow-up, the intramedullary signal abnormalities had completely resolved in patient 2. In patient 1, the extensive hyperintensities had almost completely resolved by day 17 after onset of meningitis, but on day 24, a central hyperintense lesion was detected in the cervical spinal cord at the level of C6. This lesion was interpreted to be a newly formed syrinx. In patient 3, the T2 signal abnormalities that initially involved only the thoracic cord extended to C4 by day 17 (Figure 2) and to C3 by day 29. Furthermore, the high intensity of the T2 signal was substantially unchanged in the thoracic and cervical cord compared with day 17 after onset of meningitis.
Figure 1. Patient 1 on day 5 of meningitis due to Streptococcus pneumoniae. A, Sagittal postcontrast T1-weighted image (repetition time [TR], 587 milliseconds; echo time [TE], 12 milliseconds) shows intense leptomeningeal enhancement and discrete intramedullary enhancement. Sagittal (B) (TR=3894 ms, TE=112 ms) and axial (C) T2-weighted images at the level of T8 (TR, 5700 milliseconds; TE, 120 milliseconds) show central intramedullary hyperintensities in the cervical, thoracic, and lumbar cord that predominantly involve the gray matter.
Acute spinal cord dysfunction is a rare complication of bacterial meningitis; since 1971, 29 cases have been reported.4-7 Of these 29 patients, 4 were adults and 25 children. Causative pathogens were *N meningitidis* (n=10), *S pneumoniae* (n=6), *Escherichia coli* (n=5), *Haemophilus influenzae* (n=4), *Streptococcus agalactiae* (n=2), *Klebsiella pneumoniae* (n=1), and *Corynebacterium jeikeium* (n=1). The most frequent initial symptom was quadriplegia or paraplegia. Spinal cord symptoms became evident from the time of diagnosis of meningitis until 4 days after the initiation of therapy. Six patients died, and only 3 of the 23 survivors had a complete neurological recovery. The most common residual deficits were spasticity and weakness, walking difficulties, and bowel and bladder dysfunction.

Possible causes of spinal cord dysfunction in acute bacterial meningitis are mass effect, ie, cord compression, vascular compromise, or myelitis. Compression of the spinal cord was ruled out in all of the above-mentioned cases examined by myelogram (10 patients) or MRI (5 patients). Magnetic resonance imaging of the spinal cord during the acute illness was performed in 2 children. While spinal MRI was reported to be normal in one child,6 it showed enhancement of the cauda equina and lumbosacral nerve roots in the other,7 which was interpreted to be lumbosacral polyradiculopathy. Three patients were examined only later. Five weeks after the acute illness, cystic dilatation of the upper thoracic cord was present in one case,7 which was believed to be due to vasculitis. Seven weeks after onset of meningitis, atrophy of the cervical spinal cord was detected in another patient,8 and 1 year after meningitis, spinal MRI was reported to be normal in one patient.9 We first performed spinal MRI in our patients during the first days of meningitis and spinal cord involvement. The dominant finding—in addition to the exclusion of cord compression—was the high T2 signal in the central spinal cord, which appeared to predominantly involve the gray matter. A low intramedullary signal on T1-weighted images and discrete nodular contrast enhancement of the cord were present only in patient 1. In view of the MRI findings and the exclusion of spinal cord compression, the differential diagnoses for our patients were venous congestion, ischemic infarction, and myelitis, alone or in combination.

Cerebral septic venous thrombosis is a well-recognized complication of purulent meningitis,10 and compromised venous drainage of the spinal cord due to septic venous thrombosis or adhesive arachnoiditis is also conceivable in meningitis. High T2 signals in the central spinal cord are a common finding in venous congestion, eg, because of spinal dural arteriovenous fistulas.11 However, there is no pathological report of spinal venous thrombosis in bacterial meningitis in the absence of myelitis. Nevertheless, it may have contributed to the spinal cord damage in our patients.

Ischemic infarction of the cord during bacterial meningitis can be caused by vasculitis, systemic hypotension due to shock, or arachnoiditis with secondary vasculitis.6 Severe hypotension or shock, which may have contributed to vascular compromise of the cord, preceded manifestation of spinal symptoms in 12 of the above-mentioned cases and in 1 of the patients presented herein (patient 3). However, the respiratory arrest and hypotension in this patient were probably not the only cause of spinal cord dysfunction because MRI signal abnormalities were progressive for 4 weeks after the event. Adhesive arachnoiditis with constriction of the spinal cord and putative interference with the blood supply has been reported to occur from 10 days to several years after acute bacterial meningitis.12,13 However, adhesive arachnoiditis seems unlikely in our patients, since spinal cord symptoms developed during the first days of meningitis. Furthermore, the resolution of the extensive intramedullary signal abnormalities in patients 1 and 2 at follow-up makes ischemia unlikely.

Myelitis during purulent meningitis has been rarely demonstrated on postmortem examination. In parti-
lar, edema, focal hemorrhages, perivascular inflammation in the subarachnoid space, capillary thrombosis, dilatation and thrombosis of the anterior spinal artery, and myelomalacia have been reported. In 3 patients, myelomalacia primarily affected the gray matter and spared the white matter. In our patients, the high T2 signal possibly reflected edema due to inflammation, while discrete nodular contrast enhancement was seen only in patient 1. Enhancement was not observed in the other 2 patients, possibly because of early antibiotic and anti-inflammatory (steroid) therapy.

On follow-up, signal abnormalities resolved in patient 2. In patient 1, a small syrinx had developed in the cervical cord (a known late complication of purulent meningitis). In patient 3, neurologic deficits and MRI alterations were progressive for 4 weeks. All 3 patients had persisting tetraparesis and bowel and bladder incontinence, although MRI signal alterations of the cord had resolved in 2 of them. In at least these 2 cases, the neurologic deficits must be attributed to diffuse damage to the spinal cord, which cannot be discerned on MRI.

In conclusion, we have demonstrated for the first time the MRI findings of adults with spinal cord involvement during bacterial meningitis. The observed extensive central intramedullary hyperintensities and the follow-up MRIs (signal abnormalities resolved completely in one patient, left a small syrinx in the cervical cord of another patient, and even progressed in the third patient) are consistent with myelitis. Findings of clinical follow-up examinations suggest that myelitis during bacterial meningitis has an unfavorable prognosis.

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