CASE REPORT

Acute Meningitis Complicated by Transverse Myelitis

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Abstract:
Myelopathy is an infrequently reported complication of bacterial meningitis. We report the case of a 32-year-old Nepali male who developed acute transverse myelopathy, confirmed by MRI, subsequent to acute bacterial meningitis.

Case Presentation:
A 32-year-old Nepali male, not known to have any chronic medical illness, was admitted through the Emergency department on 26 January complaining of rapidly progressive bilateral lower limb weakness of one day duration associated with urinary retention. The weakness started suddenly, ascending symmetrically, and was so severe that he was unable to move at all, and was associated with altered sensation up to the abdomen. Apart from mild neck pain, the patient denied any history of backache, trauma, upper limbs involvement, headache, nausea, vomiting, blurred vision, altered level of consciousness, abnormal movements, fever, skin rash, or contact with a sick patient.

He was fully conscious, oriented, with: BP 172/102 mmHg (that normalized spontaneously after admission), PR 117 beats/minute, temperature 37.1°C, neurologically cranial nerves were intact, significant neck stiffness, normal upper limbs, and flaccid paralysis of both lower limbs (power grade 0/5) with loss of abdominal reflexes, equivocal planter response and a sensory level up to T4 (D4). Cardiorespiratory systems examination was unremarkable and abdominal examination revealed a palpable urinary bladder. A Foley’s catheter was inserted to drain 1000 ml of urine.

Initial laboratory tests in the Emergency Department showed a WBC count of 18,000/μL with 85% neutrophils, hemoglobin 15.5 gm/dl, platelets 415,000/μL, BUN 21.9 mmol/l, creatinine 194 umol/l, calcium 1.7 mmol/l, s. Potassium 5.6 mmol/l, bicarbonate 17 mmol/l. An ECG showed sinus tachycardia, chest x-ray was normal, head CT scan was normal apart from an incidentally found left sublenticular cyst. Abdominal ultrasound excluded obstructive uropathy.

He was admitted to MICU with a provisional diagnosis of acute meningitis with transverse myelitis. Further investigations showed CPK of 20,469 iu/L, CK-MB 356 ng/ml, Troponin T <0.03, normal coagulation profile. Cerebrospinal fluid was colorless, slightly hazy, WBC 840/μL (neutrophils 95%, lymphocytes 5%), RBC 135/μL, protein 1.21 g/l, glucose 5 mmol/l (60-80% of blood glucose), negative for Gram stain, Indian ink for C. neoformans, latex bacterial antigen detection, acid fast bacteria, and produced no bacterial growth on culture.

Triple antibiotics therapy was started (Ceftriaxone+Vancomycin+Acyclovir) for meningitis and intravenous fluids to treat sepsis-induced rhabdomyolysis. The next day, renal function showed a dramatic improvement with a drop of BUN to 12.3 mmol/l and creatinine to 96 μmol/l, glucose 5 mmol/l (60-80% of blood glucose), negative for Gram stain, Indian ink for C. neoformans, latex bacterial antigen detection, acid fast bacteria, and produced no bacterial growth on culture.

MRI of the brain was normal. MRI of the spine showed a long segment of lower cervical and dorsal spinal cord intramedullary abnormal, T2 bright signal with subtle post-contrast enhancement opposite T4 and T5 suggestive of myelitis (Figure 1). Repeat CSF examination on day 4 of admission showed a drop of WBC to 48/μL and protein to 0.62 g/l.

Clinically, there was no improvement in the power of the lower limbs but the neck stiffness was resolving. The patient was referred to rehabilitation care two weeks after admission.

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Discussion:

A variety of unusual neurologic complications can occur rarely in meningitis including spinal cord involvement. Between 1971 and 2001, only 29 cases had been reported; four adults and 25 children. The most frequent initial symptom was quadriplegia or paraplegia. Spinal cord symptoms became evident from the time of diagnosis of meningitis until four days after the initiation of therapy. Six patients died and only three 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.

In view of the MRI findings (central intramedullary hyperintensities on T2-weighted images that extended from the cervical to the lumbar cord) and the exclusion of spinal cord compression, the differential diagnoses were venous congestion, ischemic infarction, and myelitis, alone or in combination. Ischemic infarction of the cord during bacterial meningitis can be caused by vasculitis, systemic hypotension due to shock, or arachnoiditis with secondary vasculitis.

References:

Conclusion:

A literature review of patients aged two years or more with similar complications showed that young children have cervical cord lesions, whereas the majority of adolescents and adults have thoracic or lumbar lesions. Clinical follow up suggests that myelitis during acute bacterial meningitis, has an unfavorable prognosis and all patients had persistent neurologic deficits, regardless of age.

This case shows that myelopathy may complicate acute bacterial meningitis, possibly due to vasculitis, stroke, autoimmune myelopathy, or direct infection of the spinal cord, and emphasizes that in evaluating the early or late neurological deficits in meningitis, spinal cord lesions, though uncommon, do occur and should be considered. Patients with myelopathy associated with acute meningitis should receive spinal MRI and meningitis should be considered in patients presenting with acute myelopathy.