CASE REPORT

Acute flaccid paraplegia: a rare complication of meningococcal meningitis

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Study design: A case report of spinal cord dysfunction following meningococcal meningitis.

Objectives: To describe a rare complication of meningococcal meningitis.

Setting: Spinal Unit, Armed Forces Institute of Rehabilitation Medicine, Rawalpindi, Pakistan.

Methods: A young healthy male developed meningococcal meningitis followed by acute onset low thoracic flaccid paraplegia with complete motor and sensory loss and sphincter disturbance. He responded well to antibiotics but was not investigated for causes of paraplegia. While at home in a rural area, he developed pressure ulcers, anemia and depression. Magnetic resonance imaging of the whole spine and computed tomography scan of the brain performed after 4 and 10 weeks were normal.

Results: The patient had a comprehensive rehabilitation at our institute. Recovery was complicated by ossification in the right thigh, which responded well to radiotherapy. At 1-year follow-up, the motor deficit and neurogenic bladder and bowel persisted and the patient remained wheelchair dependent for mobility.

Conclusion: Several mechanisms have been proposed to explain spinal cord damage after meningitis. These include spinal cord infarction; autoimmune-mediated inflammatory myelopathy and direct infection of the cord. Most probable cause of spinal cord dysfunction in this case was thoracic myelopathy.

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Keywords: meningococcal meningitis; non-traumatic spinal cord injury; paraplegia; thoracic myelopathy

Introduction

Neisseria meningitidis is a frequent pathogen in the causation of acute bacterial meningitis in the developing world.¹ If not treated aggressively, it leads to a number of complications such as meningococcal sepsis, obstructive hydrocephalus or cranial nerve palsies.² Paraplegia is a very rare complication of meningococcal meningitis that has only been reported once from Pakistan.³ We report a case of flaccid paraplegia developing as a secondary complication of meningococcal meningitis and discuss the possible mechanisms and pit falls in the early identification of the problem.

Case report

A 20-year male subject was admitted with 2 days history of high-grade fever, headaches and obtunded consciousness.

He had neck rigidity and his Glasgow Coma Score was 8/15. Lumbar puncture revealed turbid color cerebrospinal fluid (CSF) with reduced glucose (29 mg dl⁻¹), increased neutrophils (2300 cm⁻³) and increased globulins. Gram staining was positive for Gram-negative intracellular diplococci. Efforts were made to trace out the CSF culture report from the primary hospital but it could not be located. CSF lactate and PCR amplification were not carried out because of non-availability. Rest of the investigations were normal. Mantoux test and blood/CSF syphilis serology were negative. A diagnosis of meningococcal meningitis was made and he was managed with third generation cephalosporin (inj. Ceftriaxone 1 g i.v. × b.i.d.) and benzyl penicillin (30 million units × 4 hourly) to which he responded very well.

He regained consciousness and Glasgow Coma Score improved to 13/15 over the next 4 days. He complained of loss of sensations below umbilicus and inability to move his lower limbs. Neurological examination revealed a flaccid paraplegia with 0/5 power in lower limbs, sensory level at T10, areflexia with equivocal plantars, patulous anal tone and urinary incontinence. No attempt was made to identify the cause and he was discharged after 3 weeks when he was

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afebrile for the last 7 days. His parents kept on consulting different hospitals and he underwent non-contrast magnetic resonance imaging (MRI) of the whole spine (Figure 1), computed tomography scan of the brain and electroencephalogram, which all turned out to be normal. He was put on standard 04 drugs anti-tuberculosis treatment (ATT) on suspicion of paraspinal abscess.

He was admitted to our institute 3 months after the onset of paraplegia with anemia, hypoalbuminemia, reactive depression and grade IV pressure ulcers on sacrum and greater trochanters. The neurological examination was same as before with bilateral extensor plantar response and Ashworth grade II spasticity in lower limbs. The diagnosis was reviewed, ATT stopped and repeat MRI thoracolumbar spine was performed, which was normal. Based on clinical findings, the most likely diagnosis was thoracic myelopathy secondary meningococcal meningitis. He was managed conservatively for the pressure ulcers with wound debridement and dressings. Psychotherapy was carried out and fluoxetine was commenced for depression. Anemia was corrected with hematinics. A bowel routine was established with high-fiber diet and manual evacuations. He was trained in clean intermittent catheterization for bladder management. At 1-year follow-up, there was minimal improvement in the motor function and

sphincter disturbances and he was wheel chair dependent for mobility.

Discussion

Pyogenic meningitis is quite often complicated by cerebrovascular involvement, cerebral edema, hydrocephalus, along with systemic complications like septic shock and disseminated intravascular coagulation.² It is very rare for bacterial meningitis to be complicated by spinal cord dysfunction.⁴ Reported mechanisms for spinal cord dysfunctions include infarction due to vasculitis, direct infection of the cord- and immune-mediated inflammatory myelopathy.⁵⁻⁷ Choudhary *et al.*⁷ recently reviewed the literature and could locate only eight reported cases of meningococcal meningitis causing myelopathy.

In our case, development of flaccid paraplegia 96h after meningococcal meningitis infection suggests the possibility of epidural/subdural abscess or transverse myelitis. The exact cause of spinal cord dysfunction in our case is difficult to determine as there was no effort to identify the cause of this complication by the primary treating physician in the acute phase. Neurological examination did not fit into any spinal artery territory. However, presence

Figure 1 Sagittal T1 and T2 weighted MRI scans of the thoracolumbar spine 4 weeks after the onset of paraplegia and administration of antibiotics showing a normal spinal cord without any evidence of cord edema or myelitis.

of a definite sensory level at T10, flaccid paraplegia with incontinence of bladder and bowel suggests transverse myelitis as the most probable cause in this case. One may question the normal MRI findings. The possible explanations are as follows:

1. Patient received high-dose antibiotics initially and the first MRI scan was performed after 4 weeks of onset of paraplegia. There is a possibility that the abnormal intensity signals of thoracic myelopathy might have resolved by that time.
2. Similar observations were reported by Kastenbauer *et al.*,⁶ when follow-up MRI of spinal cord in two patients on 17th day showed complete resolution of intramedullary hyperintensities present during the acute phase of spinal cord dysfunction.

An important limitation in this case is that the diagnosis of meningococcal infection is not certain and that there are other possibilities. It is important to highlight that false-negative and false-positive Gram stain results occur as the properties and morphology of organisms seen on CSF Gram's stain can be misinterpreted.

This report also highlights the plight of patients in developing countries being managed in peripheral hospitals where neither expertise nor advanced radiological investigations are available. At the same time, it also demonstrates the firm resolve of concerned parents to optimize their son's health and abilities, resulting in consultation with our department and his successful rehabilitation. The residual deficits of motor weakness, spasticity and sphincter

disturbances are similar to other reported cases of thoracic myelopathy after meningococcal meningitis.⁶

Conclusions

Uncommon neurologic complications like spinal cord dysfunction should be considered while treating patient with bacterial meningitis. Complete neurologic examination may save us from ordering injudicious investigation and reducing the financial burden on poor patients. Steroids may play a role in meningococcal meningitis in reducing the severity of complications.

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