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Case report
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Meningococcal meningitis with myelopathy: case report and review of literature

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Abstract: *Neisseria meningitis* is a common etiological agent in bacterial meningitis in humans. Common complications associated with meningococcal meningitis include cranial nerve palsies, hydrocephalus, seizures, stroke, cerebritis or brain abscesses. Spinal cord dysfunction is a very rare complication of meningococcal meningitis and its occurrence is limited to case reports only. We report such a case along with a review of available literature and underlying pathogenetic mechanisms. Case report: Our patient diagnosed as a case of meningococcal meningitis developed multiple cranial nerve palsies followed by flaccid paraplegia with bowel bladder involvement during the recovery phase of the disease. Spine MRI showed mild cord edema and diffuse patchy areas of altered intramedullary signal intensity in dorsal cord suggestive of myelitis. The patient was treated with antibiotics and intravenous methyl-prednisolone along with supportive physiotherapy. His neurological deficits showed marked improvement on subsequent follow up.

Keywords: Meningococcal meningitis, paraplegia, myelopathy, spinal cord dysfunction, India, MRI findings

1 Introduction

Meningococcal meningitis is a disease with worldwide distribution. Neurological sequelae of meningococcal meningitis are rare especially after the advent of antibiotics. Myelopathy is an uncommon complication of this disease. We report such a case along with review of literature.

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2 Case report

A 19 year old male presented with high grade fever, headache, vomiting, skin rash and loss of consciousness of one day duration. Examination revealed an unconscious patient with Glasgow Coma Score of 6. Vitals were: - pulse rate 90/min, BP 100/66, respiratory rate 29/min and temperature 102F. At presentation no focal neurological deficits pertaining to cranial nerves and motor system were detected. Reflexes were normal and plantars flexor bilaterally. Neck rigidity was positive. Petechial skin rash was noticed; maximum over the lower limbs and abdomen. Lumbar puncture showed - WBC count of 13,400; 100% polymorphs, protein 238 mg% and glucose 12 mg/dl (corresponding blood glucose was 108 mg/dl). Gram stain showed Gram negative diplococci. Latex agglutination was positive for *N. meningitidis* Group A. CSF culture was subsequently reported as sterile. A diagnosis of acute meningococcal meningitis was made. Patient was started on IV antibiotics (Ceftriaxone 2g IV q12h) and Inj. Dexamethasone 10 mg IV q6h (steroids continued for the initial four days); showed good response to treatment, became conscious and afebrile by day 2.

On day 4, ptosis of left eyelid was noticed. Examination revealed complete left 3^{rd} and 7^{th} cranial nerve palsy. Rest of neurological examination was normal. Next day, patient complained of deafness. Pure tone audiometry could not be done as the patient could not be educated in the procedure. CECT head on day 5 was normal. Brainstem evoked response audiometry was suggestive of bilateral sensori-neural hearing loss.

Two days later (day 7), patient noticed weakness and sensory loss in both lower limbs with urinary retention. Examination revealed hypotonia with grade 0/5 power in bilateral lower limbs with no sensory perception below D_7 segment. Plantars were mute with absent abdominal, anal, cremasteric and lower limb deep tendon reflexes. Brain MRI was normal. Spine MRI showed patchy altered intramedullary signal intensity (hyper intense signal on T_2 W images) in dorsal cord from D2 –D9 vertebral level. (Figure 1). Dorsal cord was mildly edematous. No significant post gadolinium enhancement of cord or meninges was noticed. MRI was suggestive of myelitis. Lumbar puncture was repeated which showed a TLC of 20 with 60% polymorphs and 40% lymphocytes, glucose 70 mg/dl (corresponding blood glucose 98 mg/dl) and protein 80 mg/dl. Gram and AFB staining were negative. CSF PCR for *M. tuberculosis* was negative twice. ANA was negative. Brucella, Lyme, HSV, CMV, mycoplasma and chlamydia serologies were negative with a non-reactive VDRL.

A final diagnosis of acute meningococcal meningitis with bilateral sensorineural hearing loss with complete Left 3rd, 7th cranial nerve palsy with meningitis related myelopathy was made. The patient was continued on intravenous antibiotics and was given Inj. Methylprednisolone 1g i.v. for three days followed by oral prednisolone 1mg/kg/d for another 10 days. Urinary retention necessitated bladder catheterization in the acute stage of illness. Constipation was managed with oral laxatives and enemas or rectal suppositories as appropriate. No dramatic response to steroids was seen; at the time prednisone was stopped, patient had grade 1/5-2/5 power in the lower limbs; however urinary retention

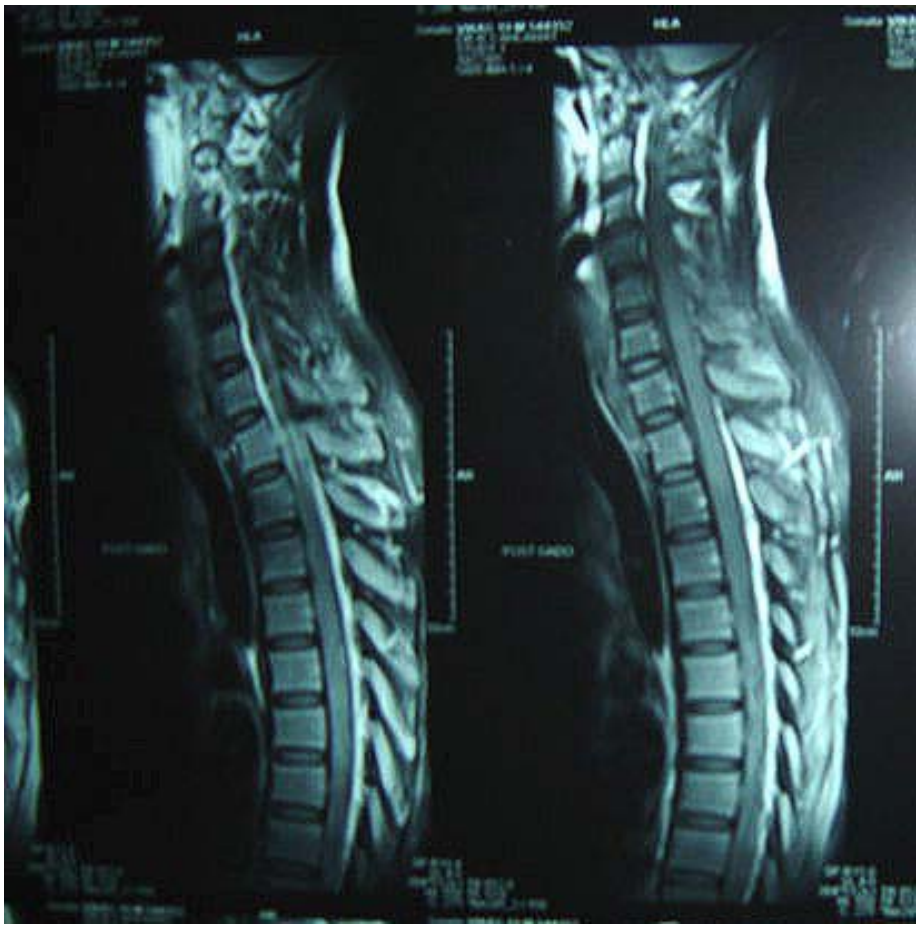

Fig. 1 Spine MRI showing area of cord edema and hyper intense signals (on T2W images) in dorsal cord extending from D2 – D9 vertebral level.

had not improved and patient continued to be on an indwelling urinary catheter. At discharge (after one and half months of hospital stay), neurological examination revealed near complete recovery of oculomotor and facial nerve function and grade 3/5 power in the lower limbs. There was no improvement in hearing loss. The patient had symptoms of urinary urgency and a sensation of incomplete evacuation of urine; ultrasonography showed post residual urine volume of 30 cc and he was managed with oral anticholinergic medications alone. No detailed urodynamic studies were performed. Follow up of the patient at the end of six months showed that muscle power in lower limbs had improved to grade 4/5; patient was able to walk with support. Recovery of sensory function was nearly complete and the patient had almost no bladder symptoms. Bowel functions appeared to have been restored and the patient no more required oral laxatives for normal bowel clearing.

3 Discussion

Neurological sequelae to meningococcal meningitis are usually not seen in the antibiotic era [1]. Common complications are cranial nerve palsies (6^{th}, 7^{th}, 8^{th} nerves), hydro-

cephalus, seizures, stroke, cerebritis and brain abscesses. Spinal cord dysfunction is a rare complication and is confined to case reports only. A search in Pubmed yielded only eight cases of meningococcal meningitis related myelopathy till date [1–8]. Majority of these cases are from the pre MRI era, some of whom underwent myelography which turned out to be normal (Table 1).

Table 1 Details of cases of myelopathy complicating meningococcal meningitis.

No	Authors, Year	Age Sex	Neurological findings	CSF	Method of diagnosis	Imaging	Treatment and Outcome
1.	Gotshall RA [1] 1972	20/M	Conus medullaris syndrome apparent during recovery.	TLC 12700, P 100%, Prot 629, Glucose 10	Gram stain, cultures		Prednisolone X 14 days Recovered except for perianal anesthesia and difficult urine voiding
2.	Swart SS [2] 1980	15/M	Multiple cranial n. palsies, resp arrest, flaccid quadriplegia, loss of all sensations below T4 except vibration sense.	-	Gram Stain		Antibiotics, Full recovery except slight impairment in heel walking.
3.	Graus F [3] 1981	15/F	Left leg became weak within several hrs after fever. Loss of pain and temp sense on right leg below T10. Proprioception normal b/l legs	TLC 2330, P 98%, Prot 96, Glucose 60	Gram stain, cultures	Myelography normal	Antibiotics, mannitol. Gradual neurological improvement with complete recovery in 3 months.
4.	Khan J [4] 1990	29/M	Paraplegia with impaired sensations below T10, absent DTR in knee and ankle, plantars flexor	TLC 16660, P 95% Prot 748, Glucose 8	Culture	CT head-cerebral edema, enhancement of basal cisterns	Antibiotics, mannitol, dexa. Mild residual paraparesis with persistent urinary incontinence
5.	Khare KC [5] 1990	21/M	Paraplegia with impaired sensations below T10, absent DTR in knee and ankle, plantars extensor, Beevor's sign +, pericardial rub.	TLC 5300, P 90%, Prot 960, Glucose nil, LDH 560	Gram stain		Antibiotics, mannitol. Complete recovery by 30^{th} day. Pericardial rub disappeared after 2 days.
6.	O' Farrell R 2000 [6]	25/F	Flaccid areflexic tetraplegia with respiratory arrest, intact cranial N.	Predominant pleomorphic pleocytosis	Gram stain	MRI - increased signal intensity in lower medulla, upper cervical cord, tonsillar ectopia.	Antibiotics, mannitol, dexa. Multi-organ failure leading to death.
7.	Kastenbauer S 2001 [7]	17/M	Respiratory arrest and recovery followed by flaccid paraplegia and a sensory level at T8	TLC 300, P 71%, Prot 166, Glucose less than 40% of blood Glucose	CSF DNA PCR for *N.meningitidis*	MRI intramedullary hyperintensities cervical to dorsal cord with cord swelling.	Flaccid tetraparesis (muscle power 4/5 inarms and 0/5 in legs) and a sensory level at T6. boweland bladder incontinence present
8.	Bhojo AK [8] 2002	17/M	Weakness of lower extremities with absent knee and ankle jerk and absent plantars	TLC 40000, P 90% Prot 297, Glucose 16	Latex particle agglutination for meningococci	MRI brain-hyperintensities on T2 images T4 to T9 region.	Antibiotics, dexa. No improvement in paraplegia with sensory level T4.

Several mechanisms have been proposed to explain spinal cord damage in meningitis [1, 4, 7, 9]. These are cord infarction; auto-immune mediated inflammatory myelopathy and direct infection of the cord. Vasculitis of the spinal arteries due to the underlying arachnoiditis may be responsible for infarction. Vasculitis is most likely to present early on in the illness when the infection is active. Hypoxia and systemic hypotension may contribute to the ischemic damage. Meningitis related myelopathy has a predilection for the mid-thoracic region especially in adults which is the watershed zone of the cord suggesting a vascular pathology [9]. In contrast, an autoimmune etiology usually presents in the 1^{st} to 3^{rd} week when the disease is in the phase of resolution.

Clinical suspicion of cord involvement mandates an MRI study. The MRI findings of altered signal intensity extending over several spinal cord segments with cord swelling are suggestive of autoimmune myelitis or direct infection of the cord. These findings are however non-specific and can also be seen in myelitis related to post infectious, post vaccination, Devic's disease, sarcoidosis, post irradiation and collagen vascular disorders. Lack of post contrast enhancement on MRI with absence of cord swelling is suggestive of vascular compromise [7].

A close differential diagnosis in our case is a first attack of multiple sclerosis. In the literature, there is emphasis on differentiating transverse myelitis from the first attack of multiple sclerosis (MS) by MRI features. Reports of MR imaging findings in patients of parainfectious transverse myelitis have described local enlargement of the spinal cord and increased signal intensity on long repetition time/echo time sequences [10, 11]. Scans done early in the course of the disease may not show any signal intensity on T2 weighted images [11]. Commonly, three to four segments are involved [10]. In MS the spinal cord involvement is usually less than 2/3rd in axial section, vertical extent is also less than 2 segment and there is central homogeneous enhancement [10, 12]. Jeffery et al highlighted the importance of cord swelling in parainfectious myelitis and its absence in MS [13].

It is difficult to pin-point the exact etiology for cord dysfunction in our case. Clinical findings of acute onset motor paraplegia and complete sensory loss in both lower limbs with bladder involvement were consistent with a diagnosis of transverse myelitis. The neurological involvement did not fit into any specific spinal artery territory. MRI findings of diffuse involvement of a long segment of the cord and edema of the cord are compatible with either direct infection of the cord or auto-immune damage. Hence, we keep the possibility of infectious insult to the cord probably compounded by an auto-immune injury as the most likely pathology of cord dysfunction in our patient.

This case report depicts a devastating and ill understood complication of a common disease. The advent of MRI has made the work up relatively straightforward. Steroids are used empirically in view of a possible underlying autoimmune mechanism. Prognosis of this condition is generally favorable.

There are several learning points regarding this case report:

(1) Meningococcal meningitis may lead to structural lesions of the spinal cord due to vasculitis or direct infection.
(2) Treatment options are not clearly defined and role of steroids remains largely empir-

ical. It is difficult to determine the contribution of steroids in neurological recovery.

(3) Usual outcome of meningococcal myelopathy is a fair neurological recovery. However prognosis should be kept guarded.

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