Infarction of the spinal cord as a complication of pneumococcal meningitis

Case report

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Spinal cord infarction in association with pneumococcal meningitis has not been previously recognized. The case is reported of a 5½-year-old boy who had *Streptococcus pneumoniae* meningitis complicated by the sudden onset of flaccid paraplegia and loss of all sensory modalities below the level of T-2. At operation, the spinal cord was pale and enveloped by dense adhesions, suggesting compromise of the arterial vasculature with concomitant infarction.

KEY WORDS · spinal cord · transverse myelitis · infarction · meningitis · *Streptococcus pneumoniae* · myelopathy · paraplegia

Neurological sequelae of bacterial meningitis have included hearing deficits,1,3,4,9,11 seizures,1,4,9,11 hydrocephalus,1,4,9,11 mental retardation,9 blindness,3 cranial nerve palsies,1 hemiparesis4,11 and quadriplegia.10 Necrotizing vasculitis and venous and arterial thromboses may accompany meningitis.1,4,6,7,10,11 Clinical documentation of spinal cord involvement following meningitis has been rare; only one case of quadriplegia following *Haemophilus influenzae* meningitis9 and one case of “conus medullaris syndrome” following meningococcal meningitis6 have been reported. We report a case of T-2 transverse myelopathy with resultant paraplegia and total sensory deficit following *Streptococcus pneumoniae* meningitis.

Case Report

This 5½-year-old boy was taken to a local hospital with fever to 107°F, cough, rhinorrhea, pharyngitis, disorientation, and progressive lethargy of 2 days' duration. Lumbar puncture revealed cloudy cerebrospinal fluid (CSF) (Table 1). Numerous Gram-positive diplococci were seen on CSF smear. Hematocrit was 36%, hemoglobin 12.1 gm/dl, and leukocyte count 10,800/cu mm (62% bands, 34% neutrophils). Treatment was initiated with intravenous penicillin, 1.2 million units every 4 hours, and chloramphenicol, 500 mg every 6 hours, and the patient was transferred to The Johns Hopkins Hospital.

On admission, he was disoriented, somnolent, and hypotonic. There was no evidence of papilledema or middle ear infection. Respiratory and cardiovascular examinations were within physiological levels. The abdomen was moderately distended. The neurological examination was remarkable for roving eye movements, a stiff neck, positive Brudzinski's and negative Kernig's sign, intact sensory examination, generalized hypotonia with 1+ deep tendon reflexes, and bilateral plantar Babinski responses. Repeat spinal tap demonstrated an opening pressure of 240 cm H2O and CSF parameters as shown in Table 1. The antibiotic regimen was changed to intravenous penicillin, 1.2 million units every 6 hours. A repeat spinal tap on the 6th day revealed CSF values as shown in Table 1.

The 1st week was complicated by intermittent fever episodes, transient ileus, and transient hypochloremic alkalosis. On the 7th day, the antibiotic regimen was changed to intravenous ampicillin, 900 mg every 6 hours, to cover for a presumed urinary tract infection. A urine culture taken prior to treat-
TABLE 1
Results of cerebrospinal fluid testing*

<table>
<thead>
<tr>
<th>Day</th>
<th>Blood Cells</th>
<th>Differential Testing</th>
<th>Protein (gm/dl)</th>
<th>Glucose (mg/dl)</th>
<th>CIE Results†</th>
<th>Culture Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>44,100</td>
<td>51,250</td>
<td>80 PMN's</td>
<td>1.040</td>
<td>positive</td>
<td>Streptococcus pneumoniae</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>20 MMN's</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>34</td>
<td>1250</td>
<td>92 PMN's</td>
<td>1.139</td>
<td>positive</td>
<td>no growth</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>8 MMN's</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>4392</td>
<td>927</td>
<td>78 PMN's</td>
<td>0.366</td>
<td>negative</td>
<td>no growth</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>22 MMN's</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* RBC = red blood cells; WBC = white blood cells; CIE = counterimmunoelectrophoresis; PMN's = polymorphonuclear leukocytes; MMN's = monomorphonuclear leukocytes.

† Testing for Streptococcus pneumoniae.

Discussion

Vascular-induced transverse myelopathy is a rare complication of meningitis. Review of the English literature produced reports of only two similar cases, neither of which was associated with pneumococcal meningitis.
Occlusive vasculitis may accompany bacterial meningitis.\textsuperscript{1,4,6,7,10,11} This often involves veins, although necrotizing arteritis\textsuperscript{4,11} and arterial thrombosis\textsuperscript{3} have been observed. Direct bacterial invasion of vessel walls may account for inflammation, necrosis, and thrombosis.\textsuperscript{10} Vascular insults have been implicated in the etiology of focal cerebral neurological deficits seen in association with meningitis.\textsuperscript{1,2,4,7,10,11} The sudden onset of flaccid paraplegia, areflexia, and total sensory loss observed in our patient implicates a total transverse spinal cord lesion, probably secondary to vascular compromise.

Several syndromes of vascular insult to the spinal cord have been described.\textsuperscript{5} The anterior spinal artery syndrome involves occlusion of the arterial blood supply to the anterior two-thirds of the spinal cord. Clinically, this presents as sudden paralysis, loss of sphincter control, and loss of pain and temperature sensation but not vibratory or position sense. Posterior spinal artery thrombosis is rare and produces a syndrome of mild, usually reversible, spastic paraparesis, loss of sphincter control, and loss of sensation below the level of the lesion. Venous infarction may follow thrombosis of the veins of both the spinal cord and the meninges. This results in necrosis of the entire transverse segment, clinically presenting as areflexic flaccid paraplegia and loss of all sensory modalities below the lesion.\textsuperscript{8}

The clinical presentation of our patient, with the acute onset of total sensory and motor deficits below T-2, is consistent with a vascular insult resulting in total transverse destruction of the spinal cord. While the extent of the lesion is suggestive of venous infarction, the fact that the cord was pale and of normal size at the time of surgery is evidence against this pathogenetic mechanism. Arterial compromise, either secondary to vasculitis or adhesive arachnoiditis, probably led to the acute infarction of the cord. The fact that the onset of neurological deficits occurred on the 10th day of illness, 4 days after a lumbar puncture in which the CSF was found to be negative for Streptococcus pneumoniae by both counterimmuno-electrophoresis and culture, suggests that arachnoid adhesions developing in the wake of resolving inflammation probably played a major role. Adhesion may have compromised flow in small arteries supplying both the anterior and posterior aspects of the cord, which resulted in total transverse myelopathy.

Paralysis following bacterial meningitis may be caused by subdural effusion or empyema, as well as epidural or intracerebral abscess.\textsuperscript{12,4,10,11} Vascular injury to the spinal cord has been described with Haemophilus influenzae\textsuperscript{3} and meningococcal meningitis.\textsuperscript{6} The present report suggests that infarction of the spinal cord may follow pneumococcal meningitis as well.

References

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