

raised intracranial pressure,¹³ facial palsies are distinctly uncommon in this setting. A case of BIH with facial diplegia has been reported but was thought to be the result of non specific pressure related phenomenon displacing and stretching the seventh nerves.⁶ One report mentions three children with BIH and an associated seventh nerve palsy, however, the weakness in all cases took over five weeks to resolve and was attributed to an associated Bell's palsy.³ In 18 children with BIH only one had facial weakness⁴ and a larger series of 79 children reported facial palsy in only three cases.¹⁴ Given the rapid resolution of cranial nerve palsies following lumbar puncture, the most plausible explanation is that seventh nerve palsy can occur, albeit rarely, as a false localising sign of raised intracranial pressure. Other focal neurological signs with long tract involvement have been attributed to BIH,¹⁵ but it must be stressed that in this and all cases of atypical BIH close follow up is essential.

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Lumbosacral meningoradiculitis associated with *Chlamydia pneumoniae* infection

Infections due to *Chlamydia pneumoniae* have recently been identified^{1,2} and usually give rise to oropharyngeal and pulmonary complications. Neurological manifestations have not, to our knowledge, been described. We

Table Antibody levels in serum specimens and CSF to *Chlamydia (Chl)* species by indirect microimmunofluorescence test

Dates Day/month/year	Specimens	<i>Chl pneumoniae</i>		<i>Chl trachomatis</i>		<i>Chl psittaci</i>
		Total	IgM	Total	IgM	Total
08/09/90	Serum	128	64	64	16	32
08/09/90	CSF	16*	<2	<2	<2	ND
25/09/90	Serum	128	32	64	<16	32
30/01/91	Serum	64	<16	32	<16	ND

*Ratio serum/CSF = 8
ND = Not Done

report the case of a child presenting with lumbosacral meningoradiculitis.

A nine year old child without a previous medical history presented with a cough and rhinitis beginning on the 21 August 1990 and lasting six days. His temperature had not been recorded. He was treated initially with amoxicillin which was stopped on the 27 August due to an erythematous macular rash on the knees and thighs. On the 31 August the patient complained of weakness of the lower extremities, predominantly the hips and thighs, resulting in a waddling gait and an inability to rise from a crouching position. Neurological evaluation on the 8 September revealed significant back stiffness, bilateral Lassegue's sign, motor weakness of the lower extremities, especially affecting flexion and adduction of the thighs, less so the flexors and extensors of the legs. Knee jerk was absent on the right, diminished on the left. Ankle jerks were intact. Plantar responses were flexor. There were no objective sensory deficits nor sphincter dysfunction. Lumbar puncture (8 September) obtained clear fluid containing 6 lymphocytes/mm³, 97 mg% protein and normal glucose. Saccoradiculography was normal. Electromyography of the lower limbs (10 September) was normal as were nerve conduction velocities. Clinical recovery was gradual and almost complete by six months.

Acute and convalescent serology taken 16 days apart excluded the following infectious agents: *Mycoplasma pneumoniae*, *Legionella pneumophila*, *Rickettsia burnetti*, *conori* and *mooseri*, herpes simplex virus, herpes zoster virus, Epstein-Barr virus, cytomegalovirus, measles, mumps, adenovirus, enterovirus (Echoviruses 7, 25, 30, 33; Coxsackieviruses A9 and B2). Indirect immunofluorescence techniques³ were used to determine serum and CSF antibodies against various *Chlamydia* species (table). These showed a recent infection with *Chlamydia pneumoniae*, indicated by an elevated titre of serum IgM which disappeared by the fifth month. Furthermore, whereas the serum/CSF antibody ratio for measles and herpes zoster viruses was 1:128, it was 1:8 for *C pneumoniae*, suggesting a local synthesis of antibodies.

Our report concerns a case of localised meningoradiculitis, especially in the proximal lower extremities, associated with an infection with *C pneumoniae*. The negativity of all other serological tests and the existence of intrathecally secreted antibodies against *C pneumoniae* are in favour of this being the causative agent of the neurological presentation. The presence of IgM proves the recent nature of the infection.⁴ The other *Chlamydia* species have been implicated in rare cases of CNS complication.^{5,6} The absence of any other reports of neurological complications due to *C pneumoniae* is probably due to the fact that identification methods have only recently been available.

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Postoperative fourth ventricle tension pneumocephalus

We report an unusual case of postoperative tension pneumocephalus (PTP) after posterior fossa surgery carried out with the patient in the sitting position.

A 65 year old man was admitted to our hospital in May 1987 because of dizziness, vertigo, vomiting and diplopia. He had right extra motor ocular muscle paresis and right sided ataxia. CT showed a haematoma in the right hemisphere cerebellar. The clot was larger than 3 cm, but there was no hydrocephalus (figure 1a). General anesthesia was administered via endotracheal intubation and consisted of a mixture of 60% nitrous oxide, oxygen and halothane. He was placed in the sitting position. Hyperventilation and furosemide were used to reduce brain bulk. A right suboccipital craniectomy was per-