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Letter to the editor

Direct invasion of the brain parenchyma by *Mycoplasma pneumoniae*

Mycoplasma pneumoniae causes central nervous system (CNS) complications, fatal encephalitis being the most serious one. CNS complications occur in about 1/1000 patients. However, CNS symptoms may occur in up to 7% in patients hospitalized with *M. pneumoniae* infection (1). Direct CNS invasion of *M. pneumoniae* has never been demonstrated, although *M. pneumoniae* has been isolated from cerebrospinal fluid (CSF). Antibodies are now routinely analysed from CSF. The mechanism of *M. pneumoniae* encephalitis is unclear and direct invasion as well as para- and postinfectious immunological mechanisms have been suggested. We present a case of *M. pneumoniae* encephalitis, with demonstration of *M. pneumoniae* RNA in the brain parenchyma.

The patient was a previously healthy 42-year-old male. Five days before admission to hospital a chest X-ray suggested pneumonia. He was treated with peroral erythromycin. The day before admission he got a worsening headache. At admission he was conscious but painful, nauseous, photophobic, dehydrated, febrile (t_{ax} 38.5) and had neck stiffness. His pupils were miotic, optic discs appeared normal, and he had bilaterally enhanced tendon reflexes with clonus on the right. Plantar reflexes were extensor. CT of the brain was normal. The CSF leukocyte count was increased ($519 \cdot 10^6$/l; 19% mononuclear), as also the protein concentration (1370 mg/l). Numerous serum and CSF tests to diagnose aetiology were analysed (e.g. HIV, tuberculosis, bacterial cultivation, serum- and CSF-antibody screening). Intravenous cefotaximine was started. Later, also antituberculotic medication was given. On the following day, the patient's level of consciousness lowered. He became disoriented, but cooperated when awake. The EEG background consisted of alpha and subalpha activity, no irritation or side-to-side differences existed. The level of consciousness further lowered and on day 4 after admission pain responses were extensor. CT revealed diffuse brain oedema. On day 7 transtentorial herniation on CT was seen and i.v. mannitol was administered. However, in 3 days all brain stem reflexes vanished. The patient died of cardiac arrest, before the diagnosis of brain death was established.

Despite excessive screening, the only microbial aetiology that could be found was *M. pneumoniae*. In a serum sample taken at day 2 a high CF antibody titre suggested recent *M. pneumoniae* infection and the CSF titre was high. A pathological serum/CSF antibody ratio (256/32) suggested intrathecal antibody production.

At autopsy, the immediate cause of death was tentorial herniation. Bilateral bronchopneumonia was also found. Microscopic examination of the brain showed perivascular mononuclear inflammation with congestion and oedema. These changes were most abundant in the white matter, but were seen also in the meninges and the grey matter. At places, there was damage and oedema of the endothelium. *M. pneumoniae* RNA was detected in a temporobasal brain tissue specimen by nucleic acid hybridization (Gen Probe, San Diego, CA, USA) performed to the manufacturer's specifications. Other post-mortem viral and bacterial cultures were negative.

The demonstration of the direct CNS invasion of *M. pneumoniae* has important theoretical and therapeutic consequences. Our patient was initially treated with erythromycin. He might have survived if an antibiotic that penetrates well into the CNS had been used.

Reference

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