Guillain-Barré syndrome and optic neuritis after *Mycoplasma pneumoniae* infection

Infections and vaccinations can be complicated by acute inflammatory demyelination in the peripheral nervous system (PNS), central nervous system (CNS) or in both the PNS and CNS. Here we report a case of Guillain-Barré syndrome and bilateral optic neuritis following an acute *Mycoplasma pneumoniae* infection.

A 22-year-old man presented in February 1997 with a history of a lower respiratory tract illness ten days prior to admission. Two days prior to admission he had noticed weakness in his upper and lower limbs and numbness in his hands and feet. There was no significant past medical history. There was no history of recent vaccinations or gastrointestinal symptoms.

On initial examination he had bilateral facial weakness and a weak cough. There was moderate weakness in his upper and lower limbs. The deep tendon reflexes were absent in the upper limbs and depressed in the lower limbs. The plantar responses were flexor. Chest auscultation and chest X-ray were normal.

He required admission to the intensive care unit and mechanical ventilation within 24 hours because of poor respiratory function. Cerebrospinal fluid (CSF) examination revealed a normal leukocyte count and a normal protein level. He was treated with intravenous immunoglobulin (Ig) therapy 50 mg daily for five days. It was noted that his blood pressure was labile and he was sweating excessively. His muscle strength deteriorated over the initial three days. Magnetic resonance imaging (MRI) of the brain and cervical spine was normal. The *M. pneumoniae* particle agglutination titre was 2560 (normal less than 40) and *M. pneumoniae* IgM antibody was detected by indirect fluorescence.

Four days after admission a partial left sixth nerve palsy was noted, but the strength in his limbs was beginning to improve. Nerve conduction studies showed changes consistent with primary demyelination, namely reduced amplitudes of the compound muscle action potentials with temporal dispersion, and absent F waves in the upper and lower limbs. A repeat CSF examination showed a leukocyte count of 5 (normal <5) and CSF protein of 775 mg/dL (normal <600). Oligoclonal IgG bands were present in the CSF and serum.

A further five day course of intravenous Ig was commenced on day 9. Ten days after admission a tracheostomy was performed. His sixth nerve palsy resolved and the muscle strength in his limbs progressively improved. He was weaned from the ventilator on day 15 and able to walk by day 18.

Nine days after admission he began to experience blurring of vision. Examination on day 14 revealed that he could perceive hand movements only with each eye and that both optic discs were swollen. Ocular movements were normal. On day 16 he was given intravenous methylprednisolone 1 g daily for three days followed by a brief course of oral prednisone. His visual acuity gradually improved and 24 days after admission was 6/9 in his left eye and 6/12 in his right eye. He still had mild upper and lower limb weakness. The eye jerks were depressed and the other deep tendon reflexes were absent. The *M. pneumoniae* total particle agglutination titre was now 1280. Antibodies to *Legionella pneumophila* and *longbeachae* were not detected and there was a low (32) Chlamydia group titre by complement fixation.

He was discharged 25 days after admission.

Acute inflammatory demyelination of the nervous system after infection or vaccination is thought to be mediated by autoimmune mechanisms. The present case of combined Guillain-Barré syndrome and bilateral optic neuritis is similar to a previously reported case of Guillain-Barré syndrome and bilateral optic neuritis after *M. pneumoniae* infection, in which cerebral white matter lesions were also present on MRI. These cases and previously reported ones of combined acute PNS and CNS demyelination suggest that the target of the immune response may be a myelin antigen present in both the PNS and CNS, such as myelin basic protein, myelin-associated glycoprotein or galactocerebroside. Interestingly, Kusunoki et al., found that patients with Guillain-Barré syndrome following *M. pneumoniae* infection had elevated serum anti-galactocerebroside antibodies. T-cell proliferation assays performed on our patient's blood did not show increased autoreactivity against myelin basic protein or myelin proteolipid protein; however, our studies may have missed the peak reactivity as they were performed during the convalescent period.

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LETTERS AND CASE REPORTS

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Systemic lupus erythematosus (SLE) and neonatal morbidity and mortality

I wish to raise some additional points in response to the otherwise excellent article on pregnancy in systemic lupus erythematosus (SLE), by Johns et al., which appeared in the February issue of the Journal. Although the authors found a significantly increased risk of spontaneous abortion and fetal death in utero, and they commented on an increased risk of prematurity and low birth weight, they did not discuss any other aspects of neonatal morbidity. It would have been interesting to know whether any of the live-born infants developed congenital heart block, thrombocytopenia, or a photosensitive rash. These features, along with other such complications as leucopenia, haemolytic anaemia and neonatal hepatitis, have a significant impact on the wellbeing of the neonate.

Maternal antibody to the Ro antigen is associated with irreversible neonatal complete heart block. The incidence of this complication has not been fully established, but occurs with a frequency in the order of 10-50%. Similarly, it is well established that maternal SLE can produce neonatal thrombocytopenia, although the incidence, again, is uncertain. Does the current study have data to answer these questions?

When counselling a family, who has experienced a pregnancy loss, the importance of possible serious neonatal morbidity and mortality should also be raised.

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References