SHORT REPORT

Miller Fisher syndrome associated with Q fever

Antonio Diaz Ortuño, Concepción Maeztu, Jose A Muñoz, Rosa Reigadas, Tomás Rodríguez, Mariano Valdés

Abstract

A 51 year old woman with pneumonia developed Miller Fisher syndrome. Indirect immunofluorescence tests showed antibodies against *Coxiella burnetii*. Miller Fisher syndrome associated with Q fever has not been described previously.

The syndrome of ophthalmoplegia, ataxia and areflexia was described by Miller Fisher in 1956¹ as a variant of acute idiopathic polyneuritis. Most of the patients have an antecedent acute febrile illness of probable viral cause up to two weeks before the onset of neurological symptoms.² Q fever is an illness caused by *Coxiella burnetii*. A febrile flu-like illness is the most common feature, and pneumonia, endocarditis, hepatitis, glomerulonephritis, optic neuritis,³ and meningoencephalitis⁴ have also been described. We report a case of Miller Fisher syndrome associated with Q fever infection. The evidence is based on serological data.

Case report

On 2 April 1988, a 51 year old woman was admitted to hospital after six days of headache, nausea, vomiting, unsteady gait, diplopia, blurred vision and distal paresthesiae in all limbs. Two weeks before her neurological complaints, she experienced fever and a productive cough for five days. On admission her temperature was 36.8°C, pulse 80, and respiration 16. The blood pressure values were 140/90 mmHg, without postural changes.

On examination she was alert. There was total paralysis of both sixth nerves, paralysis of upgaze, and mild bilateral ptosis. The pupils were large and did not react to light. The fundi were normal. There was complete areflexia of the limbs without weakness. Vibration sense was slightly impaired in the legs. There was severe gait and limb ataxia.

The white-cell count was 18 000, with 91% neutrophils. The ESR was 7 mm per hour. A chest radiograph showed right lower lobe infiltrate. CT brain scan showed no abnormality. Lumbar puncture (LP) showed a clear CSF, pressure 240 mmHg, containing 5 white cells, the protein level was 45 mg/dl.

On day 17 another LP was carried out and the protein was 70 mg/dl at that time. No oligoclonal bands were detected.

On the tenth day of admission, nerve conduction studies showed reduced amplitude of sensory and motor action potentials from all the nerves tested (median sensory 1.5 uV; ulnar sensory 2.1 uV; sural sensory 1.2 uV; median motor 5.5 mV; peroneal motor 1.3 mV). The fastest sensory and motor conduction velocities and the shortest distal motor latencies, and F latencies were normal. EMG studies with a concentric needle electrode were normal, except for a slight increment of polyphasia. Blink reflex studies were normal, as was repetitive stimulation. Brain stem auditory evoked responses, somatosensory evoked potential studies and pattern-shift visual evoked potentials were normal.

No serological evidence of infection with *Treponema pallidum*, *Mycoplasma pneumoniae*, *Chlamydia psittaci*, *Legionella pneumophila*, *Toxoplasma gondii*, Epstein-Barr virus or Cytomegalovirus was found. An elevated antibody titre to *Coxiella burnetii* was found on the fifth day of admission, 1/640 on 7 April, (Abnormal values >1/80). The course of the antibody titre was followed for several months, 1/640 (19 April), 1/320 (6 May), 1/80 (5 June) and 1/20 in October.

While waiting for the result of serological examinations and with the diagnosis of atypical pneumonia and Miller Fisher syndrome, the patient was treated with erythromycin 500 mg six hourly iv for 21 days, and methylprednisolone 80 mg daily. This dose was gradually decreased in the following three months. The headache, nausea and vomiting disappeared after four days. Pupillary responses became sluggish and there was only a slight reaction to light after five days with subsequent recovery of reactivity. Areflexia disappeared in two weeks. Vibration sense returned to normal and the ataxia disappeared in three weeks. The eye movements returned to near normal although horizontal bidirectional nystagmus appeared while ophthalmoplegia was improving. Mild diplopia and nystagmus continued to be present at seven months.

Discussion

Neurological signs in the course of Q fever are rare.⁵ The following neurological com-

General Hospital,
Murcia, Spain
Division of Neurology
AD Ortuño

Division of Clinical
Neurophysiology
C Maeztu

Department of
Internal Medicine
J A Muñoz
M Valdés

Department of
Ophthalmology
R Reigadas

University School of
Medicine, Murcia
Department of
Microbiology
T Rodríguez

Correspondence to:
Dr Diaz Ortuño, Gran Vía 8,
1º Esc, 3 Izda 30004 Murcia,
Spain

Received 20 June 1989
and in revised form
21 September 1989.
Accepted 13 November 1989

plications have been reported: neuritis with atrophy of the proximal musculature, polyradiculopathy and isolated cranial nerve palsies,⁶ optic neuritis,³ encephalitis,⁴ encephalomyelitis,⁷ dementia and toxic confusional states,⁸ and extrapyramidal disease.⁸

Headache in patients with Q fever has often been found.⁴ Frequently, headache is severe but CSF fluid is usually normal.⁹ Its mechanism is unclear but in our patient increased intracranial pressure might be considered.

Our patient had the typical neurological symptoms of Miller Fisher syndrome. The relationship between this syndrome and Q fever is established by the course of the antibody titre against *C burnetti*. In the acute phase of the neurological complaints the titre against *C burnetti* was 1/640. The titre continued to increase for at least two weeks and then decreased progressively. There are two possible causes for Miller Fisher syndrome in Q fever: a direct effect of the micro-organism on

the nervous tissue, or an indirect effect through an immunological mechanism.

To our knowledge this patient is the first reported case of Miller Fisher syndrome associated with Q fever. Q fever should be considered in patients who present with the Miller Fisher syndrome.

- 1 Fisher M. An unusual variant of acute idiopathic polyneuritis (syndrome of ophthalmoplegia, ataxia and areflexia). *N Engl J Med* 1956;255:57-65.
- 2 Fross RD, Daube JR. Neuropathy in the Miller Fisher syndrome: Clinical and electrophysiologic findings. *Neurology* 1987;37:1493-8.
- 3 Schil J, Richardus JH, Baarsma GS, Schaap GJP. Q fever as a possible cause of bilateral optic neuritis. *Br J Ophthalmol* 1985;69:580-83.
- 4 Marrie TJ. Pneumonia and meningo-encephalitis due to *Coxiella burnetti*. *J Infections* 1985;11:59-61.
- 5 Christie AB. *Infectious diseases*, 3rd ed. London: Churchill Livingstone, 1980:800-12.
- 6 Masbernard A. Les localisations neurologiques des Rickettsioses. *Bull Soc Pathol Exot* 1963;56:714-51.
- 7 Derrick EH. The course of infection with *Coxiella burnetti*. *Med J Aust* 1973;1:1051-7.
- 8 Turck WPG. Q fever. In: Braude AI, David CE, Fierer J, eds. *Medical microbiology and infectious diseases*. Toronto: WB Saunders, 1981:932-7.
- 9 Laing-Brown G. Q fever. *Br Med J* 1973;219:539-41.