



Case Report

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

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Abstract

We report the case of a 12-year-old girl who developed Guillain-Barré syndrome (GBS) and optic neuritis (ON) following *Mycoplasma pneumoniae* infection.

Her symptoms, including bilateral vision impairment and tingling in her hands and right foot, were resolved after methylprednisolone pulse therapy. Serum anti-galactocerebroside (Gal-C) IgM antibodies were detected in our patient.

This is the first report of a child with GBS and ON associated with *M. pneumoniae* infection.

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Keywords: *Mycoplasma pneumoniae*; Optic neuritis; Guillain-Barré syndrome, anti-Gal-C

1. Introduction

Neurological manifestations have been reported to occur in 1–10% of *Mycoplasma pneumoniae* infections [1]. Some adults have reportedly developed Guillain-Barré syndrome (GBS) and optic neuritis (ON) [2–5]. This is the first report of a child developing GBS and ON following *M. pneumoniae* infection.

2. Case report

A previously healthy 12-year-old girl was admitted to a local hospital with fever and cough. Based on clinical

symptoms, she was diagnosed with *M. pneumoniae* infection and treated successfully with a short course of minocycline. Fourteen days after this diagnosis, she noticed tingling in the palms and fingertips of both hands. She was diagnosed with a straight neck on both cervical computed tomography and magnetic resonance imaging (MRI), and her symptoms improved slightly after taking vitamin supplements. About 30 days after the diagnosis of *M. pneumoniae* infection, she experienced sudden bilateral vision loss and was admitted to our hospital.

On admission, a general clinical examination revealed no abnormality. Neurological symptoms revealed severe loss of visual acuity in both eyes (20/1000, right; 20/50, left) and numbness in both ulnar palms and the first to third right toes. She had no motor weakness, walking disturbance, or paresis after admission. Her level of con-

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sciousness, muscle strength, sensitivity, and deep tendon reflexes were normal.

Laboratory testing showed normal peripheral blood and biochemical studies. Her *M. pneumoniae* particle agglutination titers were elevated (1:1280 on admission). Her cerebrospinal fluid revealed a protein concentration of 90.7 mg/dL, cell count of 6/ μ L, and no oligoclonal band. Plain head MRI showed swelling in both optic nerves (Fig. 1). Plain spinal MRI results were normal. We diagnosed her with bilateral ON associated with *M. pneumoniae* infection based on imaging findings, examination, and clinical course.

She was treated with methylprednisolone pulse therapy (MPT) (1000 mg/day for 3 days) for her ON. At the end of MPT (day 3 of admission), her right eyesight recovered to 20/200. On day 4 after the commencement of MPT, a nerve conduction study showed prolonged duration of proximal and distal compound muscle action potential (CMAP) and delayed F-wave latency in several nerves, and as well as reduced motor nerve conduction velocity, reduced CMAP, and reduced sensory nerve action potential in the ulnar nerve (Table 1),

consistent with demyelinating polyneuropathy. We diagnosed the tingling of the hands and foot as GBS. These findings suggested that the GBS and ON were associated with the *M. pneumoniae* infection. Anti-galactocerebroside (Anti-Gal-C) IgM antibody was detected, but not IgG. Anti-aquaporin-4 antibody results were negative. We observed that the tingling improved after beginning MPT. On day 10, the tingling disappeared completely and her eyesight recovered completely. Plain head MRI showed improvement, except for some abnormal signals in the optic nerves. She was discharged on day 11. She continued taking prednisolone (PSL) and the dose was gradually tapered off. One month after PSL cessation, she was symptom-free, and nerve conduction studies showed that her peripheral nerve was normal.

3. Discussion

Our patient experienced GBS and ON following *M. pneumoniae* infection. Four adults with GBS and ON after *M. pneumoniae* infection have been reported in

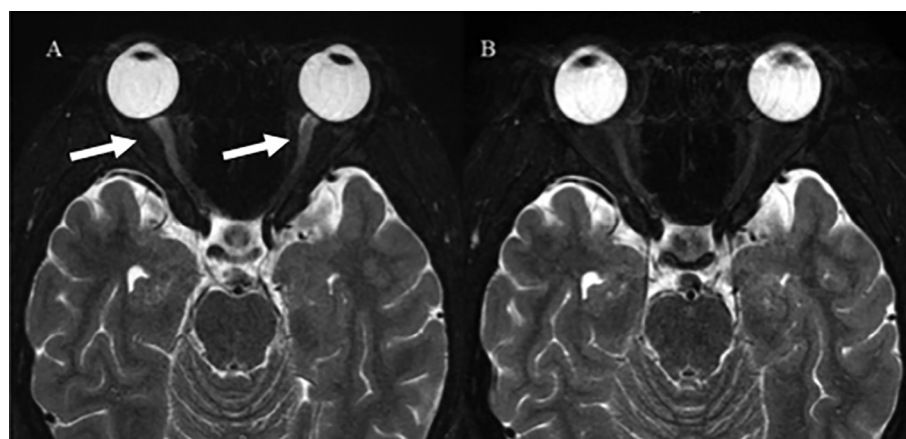


Fig. 1. T2 magnetic resonance image showing swelling in both optic nerves pre-treatment (A) and reduced swelling post-treatment (B).

Table 1
Nerve conduction study.

Motor Nerve	Right Median	Left Median	Right Ulnar	Left Ulnar	Right Peroneal	Right Tibial
DML (ms)	3.5	3.6	3.2	3.0	6.3	3.6
dDur (ms)	<u>13.6</u>	<u>12.1</u>	<u>11.2</u>	<u>8.1</u>	<u>9.6</u>	6.5
dCMAP (mV)	3.4	4.7	<u>3.6</u>	5.6	3.6	14.3
pDur (ms)	<u>13.9</u>	NE	<u>14.9</u>	NE	<u>10.1</u>	7.1
pCMAP (mV)	3.2	NE	<u>2.8</u>	NE	<u>3.7</u>	12
NCV (m/s)	55.6	NE	<u>35.3</u>	NE	48.3	51.1
F-wave latency	<u>29.8</u>	NE	<u>35.8</u>	NE	46.5	43.2
Sensory Nerve	Right Median		Right Ulnar		Right Sural	
SNAP (uV)	9.6		<u>5.8</u>		21.6	
NCV (m/s)	52.0		51.9		46.1	

Skin temperature: upper extremity: 32.5 °C, lower extremity: 32.1 °C, underline; abnormal value DML; distal motor latency, CMAP; compound muscle action potential, dCMAP; distal CMAP, pCMAP; proximal CMAP, NCV; nerve conduction velocity, dDur; Duration of distal CMAP, pDur; Duration of proximal CMAP, SNAP; sensory nerve action potential, NE; not examined.

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