Miller Fisher syndrome linked to Norovirus infection

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SUMMARY

This is a case of Miller Fisher syndrome (MFS) linked to the Norovirus syndrome. To our best knowledge, this is the first case report describing MFS associated with Norovirus infection.

BACKGROUND

Miller Fisher syndrome (MFS) is a clinical variant of Guillain-Barré syndrome (GBS), characterised by ophthalmoplegia, ataxia and areflexia, which occurs in 25% of cases in Japan.¹ There has been a case report on Norovirus-associated GBS.² This is the first case of MFS linked to Norovirus infection.

CASE PRESENTATION

A 28-year-old nurse presented with 7-day history of progressive ascending weakness and altered sensorium of bilateral upper extremities. She had been in usual state up until 14 days before admission, when she had severe vomiting and watery diarrhoea. The symptoms lasted for 3 days and resolved spontaneously. Three days prior to admission, she noticed blurred vision and gait disturbance. At the same time, she started to feel weakness and 'pins and needle'-like sensation in both her hands. Over the following 3 days, the symptoms ascended up to her elbows. She reported that the nursing home she worked for, had a proven outbreak of Norovirus with seven infected residents and four staff. She was one of the patients, diagnosed by PCR testing for Norovirus. She denied any history of similar events, any respiratory, gastrointestinal or neurological symptoms. She was currently not on any medications. She denied any recent travel to a developing country, nor animal contact. She also denied weakness or numbness of the lower extremities, fever, night sweat, weight change, headache, neck pain, back pain, shortness of breath or palpitation.

INVESTIGATIONS

On examination, she looked a lean and exhausted young lady. The movement of extraocular muscle was impaired. The upward gaze and downward gaze were impaired, although light reactivity and convergence were intact. Facial motor and sensory functions were intact. Hearing and speech were intact. Muscle strength was 4 of 5 in lower extremities with hyporeflexia. Pathological signs were negative. There was no muscle atrophy and fasciculation. Deep tendon reflexes were absent in the upper extremities (biceps, triceps) bilaterally. Sensory functions were intact in touch, pinprick, temperature, vibration, position and two-point discrimination (cortical), except for numbness of both arms up to the elbows. Dysautonomic symptoms such as incontinence, hypotension, hypertension

and arrhythmia were not present. Gait was ataxic and impaired. Cerebrospinal fluid analysis showed cyto-albumin dissociation with protein of 1.1 g/dl. Neurophysiological findings at several days after admission showed reduced sensory conduction velocities. Brain and spinal MRI did not show any findings. Antibody against GQ1b was positive. Other anti-ganglioside antibody testing, including GM1, GD1a and GD1b, were negative. Serology for *Campylobacter jejuni*, Epstein-Barr virus, cytomegalovirus, herpes zoster, hepatitis A, B and C and syphilis was all negative. Stool cultures for Norovirus as well as PCR were both positive.

TREATMENT

She was started on a 5-day course of intravenous immunoglobulin at 0.4 g/kg/day in conjunction with close respiratory and cardiac monitoring.

OUTCOME AND FOLLOW-UP

Two days after initiation of the treatment, her symptoms including ophthalmoplegia, ataxia, hyporeflexia, weakness and numbness on her upper extremities started to diminish and completely disappeared over the next 7 days. She was discharged at hospital-day 18 after completion of rehabilitation. She did not have any pain on her trunk or extremities.

DISCUSSION

This is a case of MFS being thought to be linked to Norovirus infection. The case clearly showed typical symptoms of MFS, which are of ophthalmoplegia with ataxia and areflexia.³⁻⁴ Positive antibody against GQ1, present in 90% of MFS, and neurophysiological finding also suggested diagnosis of MFS.⁵⁻⁷ As seen in this case, approximately 25% of patients with MFS develop some extremity weakness.

The timing of onset of the symptoms suggested that Norovirus was the causative microorganism. Our case showed the symptoms occurred 7 days after the diarrhoeal symptom. The first report of Norovirus-associated GBS described by Eltayeb *et al*,² and the symptom in the case occurred after 10 days of the diarrhoeal episode.

The DNA of Norovirus was detected by PCR using her stool. No other microorganisms were detected through investigation. There have been reports of infection with cytomegalovirus or Epstein-Barr virus, both are viruses, which are associated with demyelinating GBS, whereas *C jejuni* infection is associated with axonal GBS and MFS.⁸⁻¹¹ To our knowledge, this case is the first documented case of MFS linked to Norovirus infection. We agree with the comment made in Eltayeb's article, the association between Norovirus

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and GBS has not been found to date because the serology for Norovirus is not available or not often tested in routine clinical practice.² Our patient experienced the symptoms which began in the arms, which was relatively rare.^{1,2}

Learning points

- ▶ The most common postinfection-related peripheral nerve disorder is Guillain-Barré syndrome (GBS).
- ▶ As seen in other viruses, Norovirus can cause GBS/Miller Fisher syndrome.
- ▶ Weakness of GBS may begin in the arms or facial muscles in about 10% of patients, while it usually starts in the legs.

Competing interests None.

Patient consent Obtained.

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