

Miller Fisher syndrome without ataxia with pupil involvement

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The classical triad of Miller Fisher syndrome (MFS) comprises ophthalmoplegia, areflexia, and ataxia. Miller Fisher syndrome is an immune-mediated condition with anti-GQ1b antibody positivity. Cases and case series of MFS with pupil involvement are reported in the literature.^[1,2] Moreover, variants of MFS without ataxia have also been documented.^[2] Diagnostic difficulties exist in cases that do not manifest with the classic triad. Herein, we present an atypical case with pupil involvement that does not fulfill the triad, complicating the diagnosis.

A 17-year-old female patient presented to the emergency department with diplopia and noticed enlargement of the pupils. Patient history revealed an upper respiratory tract infection one month prior and that the patient consumed fish and mustard greens the day before. In the neurological examination, the patient was conscious, oriented, and cooperative. There was a restriction in the lateral gaze of both eyes. Pupils were bilaterally dilated with no light reflex. Despite the absence of light response, the near reflex was preserved. Bilateral miosis was observed following the application of 0.1% pilocarpine (Figure 1). In the neurological examination, the patellar and Achilles reflexes were hypoactive. The patient had no ataxia. The examination of other cranial nerves was normal. The cranial magnetic resonance imaging with contrast performed in the emergency department was normal. Needle electromyography (EMG) displayed small amplitude motor unit potentials in the periocular muscles during voluntary muscle contraction. Nerve conduction studies were unremarkable. No increment or decrement response was observed

in the facial and ulnar repetitive nerve stimulation tests for botulism. However, increased mean and individual jitter were detected in single-fiber EMG. A lumbar puncture could not be performed due to the patient's refusal. The ganglioside panel revealed a positive anti-GQ1b antibody.

Given that the patient's clinical symptoms remained stable and there was partial regression until the tests were concluded, treatment was not started. The patient is still under clinical follow-up. The complaint of diplopia resolved within three months. Pupil dilation and the absence of light reflex were still present at the four-month follow-up.

DISCUSSION

Pupil involvement and ophthalmoplegia were prominent in the patient's clinical symptoms. It was considered whether the mustard greens eaten with fish could be suspicious for botulism. However, the absence of accompanying dysphagia and hypersalivation, along with the patient's clinical stability in the following days, clinically led away from botulism. Normal nerve conduction in the EMG and the presence of increased jitter in single-fiber EMG, despite the lack of an increment response, supported our diagnosis. However, single-fiber EMG studies in MFS have shown that antibodies disrupt the neuromuscular junction and that there is electrophysiological improvement over months.^[3,4]

Adie's tonic pupil was considered in the differential diagnosis; however, the onset of symptoms in our case was clearly stated. In the

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Figure 1. Miosis was observed after application of 0.1% pilocarpine to bilaterally dilated pupils.

medical history, the patient mentioned having an infection a month ago. The detection of positive anti-GQ1b antibodies supported our diagnosis. Diagnostic difficulties may arise, particularly in cases where the triad is not met. Miller Fisher syndrome should also be considered in the differential diagnosis of patients presenting only with ophthalmoplegia.^[1] There are varying opinions regarding the effect of intravenous immunoglobulin on MFS. It was suggested that intravenous immunoglobulin treatment may slightly accelerate the improvement of symptoms but does not affect the prognosis.^[5]

Variants outside the classic findings should be considered in the differential diagnosis in cases of clinical diversity. In recent years, we believe that the spectrum of neurological diseases secondary to classic viral infections included in neurology textbooks has varied.

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