Miller Fisher syndrome with acute angle-closure glaucoma as the first manifestation
A case report

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1. Introduction
Miller Fisher syndrome (MFS) is a variant of Guillain–Barre syndrome, characterized by ophthalmoplegia, loss of tendon reflexes, and ataxia. Pupil abnormality caused by autonomic nerve involvement account for about 33.7% of MFS. It has been reported that acute angle closure glaucoma could occur due to autonomic nerve involvement in the course of MFS, but there were no reports of acute angle-closure glaucoma as the first manifestation of the MFS. In this paper, we present a case of MFS with acute angle-closure glaucoma as the first manifestation.

2. Case report
A 78-year-old female patient presented with pain in her left eye, blurred vision, along with nausea and vomiting for 2 days, and she was admitted to our hospital. Two weeks before admission, the patient underwent upper respiratory infection, but she did not take any medications. The patient reported with no history of other diseases and special drugs. Visual acuity results showed that the patient’s corneal opacity during acute attack stage of glaucoma, and the degree of her cooperation, the fundus examination was not performed. Mannitol and timolol were given to reduce intraocular pressure, and pilocarpine was given for myosis. On second days after admission, the left eye pressure decreased to 20 mm Hg, eye pain, nausea, and vomiting disappeared, the vision in the left eye was restored to 0.8, but the patient remained dizziness. Two days after admission, the patient saw things in pairs and walked unstable. The physical examination results showed limited abduction of the left eye, slight ptosis of both eyelids, the right pupil diameter was 5 mm, and the light reflex disappeared, the diameter of left pupil was 2 mm, and the light reflex also disappeared (pilocarpine effect). On the fifth day after admission, the patient’s limbs muscle strength class was 4+, no obvious limb
weakness performance, and disorders in limb balance and coordination, numbness of limbs were the main manifestations. She was unable to stand up and walk because of limb ataxia and dizziness, then the patient was transferred to the Department of Neurology. The physical examination results showed double eyelid fixation and binocular ptosis, both pupil diameter and light reflex were the same as those of the day before. Lumbar puncture pressure was 126 mm water column. Because of the patient’s standing difficulty and difficulty of cooperation, the evaluation of other autonomic nerve function has not been carried out, such as supine position test and postural change test. But in the course of disease, the patient’s heart rate was always greater than 100/min, suggesting the possibility of cardiac autonomic nervous system involvement, but after a long time of the dynamic ECG monitoring, and no arrhythmia evidence was found. The bilateral finger-nose tests and heel-knee-tibia test were not stable enough. Bilateral tendon reflex was not elicited. Pain and temperature sensation in extremities was decreased. Electromyogram examination showed that the amplitude of sensory nerve in the limbs decreased and the conduction velocity slowed down. Serum anti-GQ1b antibody was positive. Cerebrospinal fluid test showed that the protein was 526 mg/L, and no cells were found, it suggested that proteins and cells were separated. The final diagnosis was MFS, then intravenous immunoglobulins were administered. Ophthalmoplegia, walking instability, and ataxia, gradually improved in the patient. After 3 months discharge, the patient can completely self-care, and there was no neurological deficit.

3. Discussion

Angle closure glaucoma is a common form of glaucoma, because the anterior chamber of eyeball is closed, and then the aqueous drainage of the eye is blocked.

It has been found that oculomotor nerve dysfunction can lead to acute attack of angle closure glaucoma, and oculomotor paralysis complicated with acute closed-angle glaucoma also has been reported. Dilatation of pupils are more common in MFS, then intravenous immunoglobulins were administered. Ophthalmoplegia, walking instability, and ataxia, gradually improved in the patient. After 3 months discharge, the patient can completely self-care, and there was no neurological deficit.

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