Clinical Case Report

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

A case report

Jingzhe Han, MD[a], Ye Ji, MD[a], Duanhua Cao, MD[a], Zhilei Kang, MD[b], Jianguo Zhu, MD[a,*]

Abstract

Rationale: There were no reports of Miller Fisher syndrome (MFS) with acute angle-closure glaucoma as the first manifestation.

Patient concerns: A 78-year-old female patient was admitted to our hospital with pain in her left eye, blurred vision along with nausea, and vomiting for 2 days. It was extremely rare that ophthalmoplegia, loss of tendon reflexes, and ataxia, did not occur in the early stages of MFS, and initial presentation was only dilated pupis and an increase in intraocular pressure.

Diagnoses: The final diagnosis of the patient was MFS.

Interventions: Intravenous immunoglobulins were administered.

Outcomes: Ophthalmoplegia, walking instability, and ataxia gradually improved. At 3 months follow-up, there was no neurological deficit, and the patient could completely self-care.

Lessons: This is the first report of MFS patient with acute angle closure glaucoma as the first manifestation. Consideration should be given to the possibility of incorporating autonomic nervous system dysfunction, or even MFS, in patients with acute angle-closure glaucoma in order to reduce missed diagnosis rate.

Abbreviations: IOP = intraocular pressure, MFS = Miller Fisher syndrome.

Keywords: acute angle-closure glaucoma, MFS, miller fisher syndrome

1. Introduction

Miller Fisher syndrome (MFS) is a variant of Guillain–Barre syndrome, characterized byophthalmoplegia, loss of tendon reflexes, and ataxia.[1] Pupil abnormality caused by autonomic nerve involvement account for about 33.7% of MFS.[2] It has been reported that acute angle closure glaucoma could occur due to autonomic nerve involvement in the course of MFS,[3–5] 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 only light perception in the left eye and 0.6 in the right eye. In addition, it was found that the bulbar conjunctiva was edematous, and the anterior chamber became shallow. The pupillary diameter of the left eye was 5 mm, and the light reflex disappeared, the pupil diameter of the right eye was 3 mm, but the light reflex was sensitive. The eyeball moved freely in all directions. No abnormalities were found in other neurological examinations. The intraocular pressure (IOP) was 48 mm Hg in the left eye and 18 mm Hg in the right eye. Gonioscopic findings included a closed angle in the left eye and a Schaffer grade 2 angle in the right eye. Then the patient was diagnosed with acute angle closure glaucoma in the left eye. The patient presented with acute glaucoma as the first manifestation, indeed mixed with conjunctival congestion, corneal edema, fog turbidity, and other manifestations. Unfortunately, due to 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

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[a] Department of Neurology, Harrison International Peace Hospital, [b] Department of MRI, Harrison International Peace Hospital, Hengshui, Hebei, China.

[*] Correspondence: Jianguo Zhu, Department of Neurology, Harrison International Peace Hospital, No. 2 Renmin Zhong Road, Taocheng District, Hengshui, Hebei, 050000, China (e-mail: jiangz286@sina.com).

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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 eyeball 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.[6] Dilatation of pupils are more common in MFS pupillary abnormalities, and its structural basis is GQ1b ganglioside in the ganglia ciliare, whereas anti-GQ1b antibodies are associated with MFS internal ophthalmoplegia and mydriasis.[2] MFS patients with acute angle closure glaucoma have been reported, and most of them were related to ocular autonomic nerve involvement.[3–5] In this report, it was extremely rare that ophthalmoplegia, ataxia, and loss of tendon reflex did not appear in the early stage of MFS, but mydriasis and elevated intraocular pressure were the main manifestations. The first diagnosis was difficult, and the order of involvement of the oculomotor nerve, trochlear nerve, abducens nerve, and ciliary ganglion enriched with GQ1b ganglioside were considered. In addition, angle closure glaucoma can cause mydriasis and fixation of the pupil. Also pupils will return to normal after the intraocular pressure is normal. In this case, application of miotic pilocarpine caused clinicians' judgment on the pupil was interference; this was also an important reason for delayed diagnosis. Therefore, the etiology of sudden acute angle closure glaucoma should be fully analyzed, the possibility of autonomic nervous system dysfunction needs to be excluded.

This is the first report on acute angle closure glaucoma as the first manifestation of MFS. Therefore, we should consider the possibility of merging autonomic nervous system dysfunction and even MFS in patients with sudden closure angle glaucoma, so as to reduce the missed diagnosis rate.

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This study was approved by the institutional review board of our Hospital. Written informed consent was obtained from the members of the patient's family for publication of this report.

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