THE MILLER FISHER SYNDROME

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Abstract:
Fisher's syndrome is a neurological condition rarely described in the ophthalmic literature. The characteristics of this syndrome, external ophthalmoplegia, ataxia and areflexia are noted and two case histories are discussed.
Key words: *Ophthalmoplegia, ataxia, areflexia, diplopia, polyneuritis, ptosis, pupil response.*

INTRODUCTION

In 1956 Miller Fisher reported three cases of an acute neurological illness, in which there was a triad of "total external ophthalmoplegia, severe ataxia and loss of the tendon reflexes". Since the original description about thirty cases have been reported in the literature and this condition has become known as The Miller Fisher syndrome.

There is however very little written in the ophthalmic literature about these patients. Diplopia is often the presenting symptom and ocular signs are generally marked, including: moderate ptosis, symmetrical, complete external and almost complete internal ophthalmoplegia, sluggish pupil reaction to light.

As it is an acute atypical form of idiopathic polyneuritis, symptoms are not confined to the eyes. The most common other clinical findings are: dizziness, severe ataxia, loss of tendon reflexes, chest pain, difficulty chewing, diminished or absent sense of vibration, numbness of fingers.

The course of this disease is about three months and according to the ophthalmic literature[1,2,3] recovery is spontaneous.

As this syndrome is rare it was of great interest that two cases presented to the orthoptic department within a couple of weeks of each other.

The first patient was a 16 year old male referred from the casualty department for orthoptic assessment. His appearance was deceptive and the first reaction to his ocular and general signs was one of drug abuse. Diagnosis was complicated by a history of trauma five days previously. On presentation no ptosis was evident but he had very restricted ocular rotations with nystagmoid movements, diplopia, and sluggish pupil reaction to light and accommodation. The diplopia was difficult to join with prisms due to the lack of motor fusion and the variability of the angle size. A patch was used for comfort and management involved monitoring progress and reassurance. Recovery was complete within three months.

When the second case, a 34 year old male, was seen some degree of spontaneous recovery had already taken place. The diagnosis of Miller Fisher syndrome was made by the neurologist. At the time of referral to the orthoptist the patient was complaining of diplopia resulting

from an intermittent convergent squint of 10 to 14 prism dioptres. There was underaction of both lateral recti muscles. He was encouraged to remove his glasses briefly (high myope) in order to join the intermittent diplopia. Final recovery was achieved after surgery, approximately eighteen months after onset.

Definitive diagnosis of this syndrome requires full neurological examination. Initially the clinical picture is striking and the orthoptist's main role is help relieve diplopia and monitor progress while recovery takes place.

References

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