When the Eyes Lie: An Atypical Presentation of Miller Fisher Syndrome presenting as a Thyroid Eye Disease (TED)

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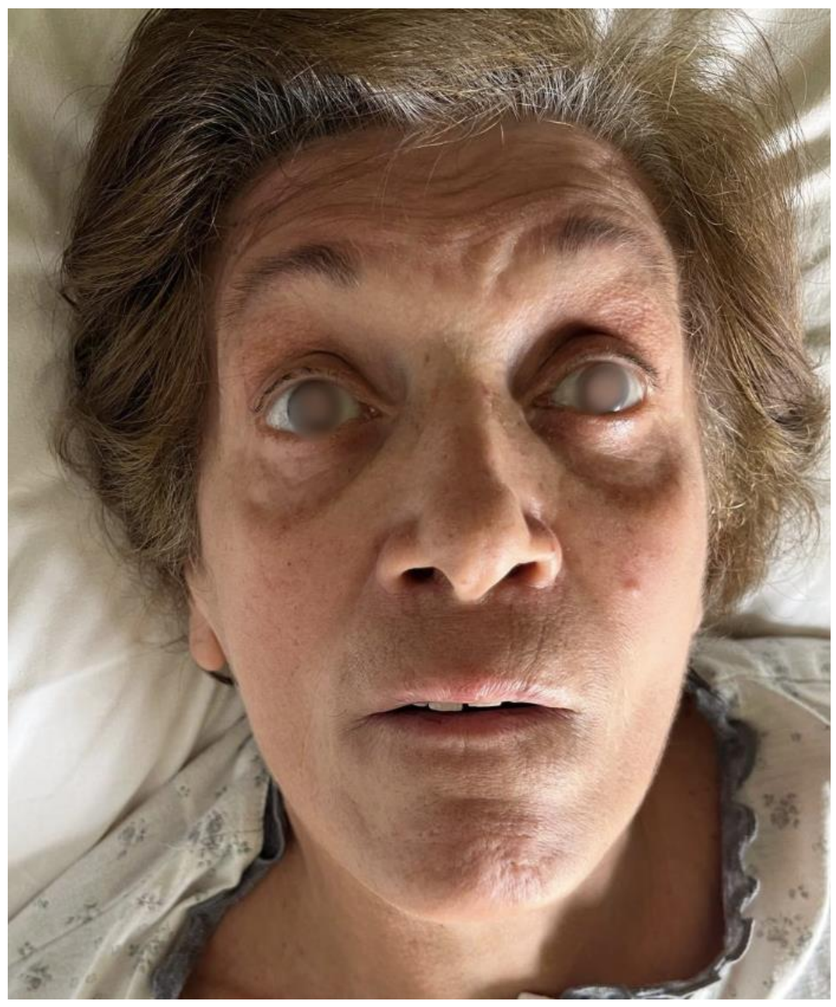
INTRODUCTION

Miller-Fisher syndrome (MFS) is a rare variant of Guillain-Barré syndrome characterized by the classic triad of ophthalmoplegia, ataxia, and areflexia, with an incidence of approximately 1-2 cases per million population annually [1]. Graves' disease and thyroid eye disease (TED) share multiple ocular manifestations with MFS, including ptosis, diplopia, and apparent eyelid retraction, creating significant diagnostic challenges in clinical practice. The overlapping presentation between these conditions has been documented in rare cases where both pathologies can coexist [2], further complicating the diagnostic process. This case reports a patient initially presenting with apparent Stellwag's sign and Moebius's sign, classical indicators typically associated with thyroid ophthalmopathy, who was ultimately diagnosed with Miller Fisher syndrome following comprehensive neurological evaluation



METHODS

A 66-year-old woman presented with acute diplopia and bilateral ptosis following a respiratory infection. Initial examination documented apparent Stellwag's sign (reduced blink frequency) and Moebius's sign (inability to converge the eyes properly), alongside bilateral ptosis and internuclear ophthalmoplegia predominantly affecting the right eye. The patient remained alert, conscious, and oriented. Method: Comprehensive neurological assessment included detailed ocular motility examination and systematic cranial nerve evaluation. The presence of apparent thyroid eye signs initially suggested thyroid eye disease (TED). Laboratory investigations included thyroid function tests, thyroid antibodies (TSH, TRAb, anti-TPO, anti-TG), and anti-ganglioside antibodies (GQ1b, GT1a, GM1). Orbital imaging evaluated extraocular muscle morphology.

RESULTS

Despite apparent Stellwag's and Moebius's signs, detailed examination revealed that these were neurological mimics rather than actual thyroid signs. The "Stellwag's sign" represented reduced blink frequency due to facial nerve weakness rather than eyelid retraction. Anti-ganglioside antibodies were strongly positive for GQ1b and GT1a, confirming a diagnosis of Miller Fisher syndrome. Thyroid function remained normal, and orbital imaging showed no extraocular muscle enlargement.

DISCUSSION

This case illustrates how Miller-Fisher syndrome closely mimics Graves' ophthalmopathy through neurological mechanisms that simulate classic signs of thyroid eye disease. The documented Stellwag's sign was a reduction in blink frequency due to facial nerve involvement, rather than actual eyelid retraction. Moebius's sign represented compensatory mechanisms secondary to oculomotor dysfunction.

CONCLUSION

Clinicians must exercise caution when interpreting apparent thyroid eye signs in cases of acute ophthalmoplegia. A detailed neurological examination, systematic anti-ganglioside testing, and orbital imaging are essential for differentiating true thyroid ophthalmopathy from neurological mimics, thereby ensuring appropriate immunomodulatory treatment.

References

1. Waung, M.W. and M.A. Singer, An unusual case of Miller Fisher syndrome presenting with proptosis and chemosis. Muscle Nerve, 2012.
2. Vetsch, G. et al., A combined presentation of Graves' disease and Miller Fisher syndrome. The Lancet, 2008.