OCULAR NEUROMYOTONIA

NEUROMIOTONÍA OCULAR

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ABSTRACT

Case report: We describe a 58-year-old man, without history of radiation therapy, who presented with transient episodic diplopia following sustained lateral gaze lasting around 30-50 seconds. He developed a large angle exotropia of the left eye that gradually returned to normal after 50 seconds. Between episodes he was asymptomatic. Partial resolution of his symptoms was obtained after treatment with carbamazepine.

Discussion: Neuromyotonia must be considered when evaluating a patient with intermittent diplopia (Arch Soc Esp Oftalmol 2008; 83: 673-674).

Key words: Ocular neuromyotonia, intermittent diplopia, intracranial disease, carbamazepine.

INTRODUCTION

Ocular neuromyotonia (ON) is a rare ocular motility disease characterized by the involuntary contraction of one or more ocular muscles (1). Generally it is secondary to other associated processes, although idiopathic cases have been described (2, 3). In this paper we present a typical ocular neuromyotonia case defined as idiopathic.

CASE REPORT

A 58-year-old man without relevant history went to the emergency service referred by his primary care physician to discard «retinal pathologies» due to intermittent diplopia starting one year ago but more frequent in the last week. The patient’s profession is truck driver. He referred between 10 and 15 events of horizontal diplopia per day, each lasting 30-50 seconds only when
looking through the left rear view mirror. After each event no symptoms appeared until the next episode. In exploration, ocular motility was normal (fig. 1), although after fixing the gaze to the left, a 20-degree exotropy developed (fig. 2) which resolved spontaneously after 50 seconds. The orbitary and cranial nuclear magnetic resonance study was normal. The edrophonium test was negative. Treatment was initiated with carbamazepine, first 200 mg and subsequently 400 mg per day, producing a considerable improvement in symptoms. The patient referred a 50% reduction in the number and intensity of daily episodes, without complete remission.

**DISCUSSION**

Ocular neuromyotonia is a rare clinical process characterized by contraction of one or several ocular muscles. This contraction is a generally spontaneous or occurs in an attempt to fix the gaze and represents the severest phenotype of hyperexcitability of ocular cranial nerves (1, 4). Inappropriate discharges of the ocular motor nerve neurons and/or instability of cellular membranes could account for the contraction. About 40% of patients with acquired neuromyotonia exhibit antibodies against potassium channels (1, 4, 5). Processes such as ocular myasthenia, demyelinizing diseases, uncompensated phorias, Graves disease, convergence spasm and mychemia of the superior oblique must be considered in the differential diagnostic (5). In most described ON cases there is a history of surgery, more specifically brain radiotherapy, due to tumors in the sellar and/or parasellar region or two compressive lesions in the ocular motors (2, 3, 5). Any motor nerve could be affected.

Ophthalmological exploration in these patients can yield normal results and only after prolonged periods of fixation or eccentric gaze the characteristic findings may emerge. In the literature, only six cases were found as idiopathic ocular neuromyotonia. This paper presents a 50-year-old patient with neuromyotonia of the VI left cranial pair without a history of radiotherapy treatment in which no lesion was found, accordingly it was considered as idiopathic ocular neuromyotonia. ON patients benefit from medications such as carbamazepine as membrane stabilizers (1-5), and all that must be studied to discard intracranial diseases.

**REFERENCES**