CASE REPORT

Ischaemic Oculomotor Nerve Palsy Isolated to the Levator: A Case Report

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ABSTRACT

Ischaemic oculomotor nerve (CN III) palsies frequently present with abrupt onset ptosis, ophthalmoplegia, diplopia, ipsilateral pain, and little to no anisocoria. An isolated microvascular ischaemic insult to the superior division of CN III is uncommon, and usually affects both the superior rectus and levator muscles. We present a rare case of an ischaemic CN III palsy isolated to the levator palpebrae superioris muscle only. Although rare, microvascular ischaemic CN III palsies should be on the differential of isolated ptosis. Other causes of isolated ptosis, such as myasthenia gravis or an orbital lesion, should be excluded.

ARTICLE HISTORY

Received 2 June 2018
Revised 7 August 2018
Accepted 27 August 2018

KEYWORDS

Oculomotor nerve palsy; CN III palsy; microvascular; ptosis

Introduction

A microvascular infarct of the oculomotor nerve (CN III) frequently presents with abrupt onset ptosis, ophthalmoplegia, diplopia, and ipsilateral pain.^{1,2} Pupil-sparing has classically been an identifying feature of ischaemic CN III palsies, but small amounts of anisocoria can be present in up to 38% of cases.³ CN III anatomically separates into a superior and inferior division in the anterior cavernous sinus or superior orbital fissure, although the topographical division occurs more proximally.⁴ An isolated microvascular insult to the superior division is uncommon, and usually affects both the superior rectus and levator muscles.^{4–6} Herein, we present a rare case of an ischaemic CN III palsy isolated to the levator palpebrae superioris muscle only, which resolved completely within one month.

Case report

A 76-year-old man presented to our clinic with acute onset right upper eyelid ptosis. He had a past medical history significant for well-controlled Diabetes Mellitus Type II, with a recent HbA1c of 6.5%, coronary artery disease status post-coronary artery bypass graft, peripheral arterial disease status post bilateral femoral endarterectomy and profundoplasty, hypertension, and a branch retinal artery occlusion in the

left eye 20 years prior. Four days prior to presentation, the patient woke up with right upper eyelid ptosis that progressively worsened. He described a mild right-sided headache the night preceding the ptosis, but denied current headache, scalp tenderness, jaw claudication, numbness, weakness, or other neurologic symptoms. He had no recent trauma. He had no eye pain or diplopia, and thought that his vision was at baseline. On exam, visual acuity was 20/25 +2 in the right and 20/20–1 in the left. Ocular motility was full in both eyes and he was orthotropic in all gazes. Ishihara color plates were full in both eyes. Pupils were equal in light and dark and reacted promptly. There was no relative afferent pupillary defect. There was marked right upper eyelid ptosis with palpebral fissures measuring 2 mm on the right and 10 mm on the left (Figure 1). Margin-reflex distance 1 was –4 mm on the right and +4 mm on the left. He had decreased lid excursion, measuring 4 mm on the right and 17 mm on the left. He had no up gaze fatigue, no Cogan's lid twitch, and no lagophthalmos. Orbicularis strength was symmetric between the two eyes and within normal limits. An ice-pack test did not improve his ptosis. Dilated fundus exam was unremarkable.

Computed Tomography (CT) and CT angiography of the brain, complete blood count, erythrocyte sedimentation rate, and anti-acetylcholine receptor antibody were all normal. The C-reactive protein (CRP) was elevated at 25.1 mg/L (normal <8.0 mg/L), but he reported a recent upper

Figure 1. Top: Primary gaze. Note significant right-sided ptosis. Middle: Upgaze. Note absence of levator function. Bottom: Upgaze with lid lifted: Note full supraduction and no anisocoria.

respiratory infection. A temporal artery biopsy was negative for inflammation or transmural scarring. Magnetic resonance imaging (MRI) brain and orbits (resolution 3 Tesla units, 1 mm slice thickness) were also unremarkable; therefore, it was presumed that this was a microvascular insult to the terminal branch of CN III affecting the levator palpebrae superioris muscle in isolation. He returned to clinic 1 month later with complete resolution of the ptosis and normal levator function (Figure 2). Repeat CRP was also performed 1 month later and was within normal limits.

Discussion

Diabetes-associated microvascular CN III palsies are a common cause of acute onset ptosis, ophthalmoplegia, and diplopia, which generally

Figure 2. Top: Primary gaze 1 month after presentation. Note resolution of ptosis and no anisocoria. Bottom: Upgaze. Note full motility and full levator function.

resolve within 3 to 6 months. Ischaemic damage to CN III frequently spares the pupil because the parasympathetic fibers serving pupillary constriction are located in a superficial and dorsomedial position along the nerve and are therefore at a lower risk of ischaemic injury.⁷ A significantly dilated pupil occurs in the majority of patients with aneurysmal compression CN III palsy, although cavernous sinus aneurysms can spare the pupil early on.^{8–10} Some degree of pupil involvement may also occur in diabetes-associated CN III palsies, but typically to a much lesser degree.³

Microvascular ischaemic injury to CN III typically involves parts or all of the nerve without following the anatomic superior and inferior divisions of the nerve. We present a case of microvascular ischaemic superior divisional CN III palsy affecting only the levator palpebrae superioris muscle, with complete sparing of the superior rectus muscle. Although our patient's diabetes was well-controlled, he did have a medical history significant for microvascular disease. After a thorough workup including blood testing for myasthenia gravis, a temporal artery biopsy to rule out giant cell arteritis, and neuro-imaging, the time frame of resolution matched a microvascular ischaemic insult. To our knowledge, microvascular

involvement of only the fibers innervating the levator palpebrae superioris muscle has only been reported in a single prior case.⁵

Although rare, microvascular ischaemic CN III palsies should be on the differential of isolated ptosis. Other causes of isolated ptosis, such as myasthenia gravis should be ruled out. Mass lesions compressing the oculomotor nerve may be the cause of a pupil-sparing CN III palsy,^{10,11} therefore, we recommend neuroimaging on all patients with acute CN III palsies. Lastly, vascular insults to cranial nerves should resolve within several months. We recommend monitoring patients for resolution of ophthalmoplegia after an acute cranial mononeuropathy.

Acknowledgements

This work was supported in part by an unrestricted grant to the Department of Ophthalmology by Research to Prevent Blindness, Inc., New York, NY.

Declaration of interest

No conflicting relationship exists for any author.

Funding

Research to Prevent Blindness, Inc., New York, NY. The sponsor or funding organization had no role in the design or conduct of this research.

Meeting Presentation

None.

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