

Neuro-ophthalmology

E-00029

Transient vision loss associated with sexual intercourse

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ABSTRACT (AS SUBMITTED)

Purpose: To present the first reported case of vasospastic amaurosis fugax associated with retinal nerve fibre layer infarctions.

Methods: Case report

Results: A 66 year old man presented with a history of acute vision loss in his left eye immediately following ejaculation. Onset was within 2 seconds of ejaculation and he described a complete blackout in his left eye (confirmed with cover testing) for an hour at which time he fell asleep. He awoke several hours later with a 'technicolor' positive visual scotoma lasting several hours. He had three similar episodes that occurred three years prior to his presentation. At that time a CT of his head and carotid ultrasound were reportedly normal and he was started on lipitor and aspirin. Three months prior to his last episode he discontinued his lipitor and aspirin and started himself on Wobenzym, a nonprescription "immune system supplement". A systemic work up for underlying inflammatory, hematological or embolic phenomena was negative and he was diagnosed with vasospastic amaurosis fugax.

Conclusions: Amaurosis fugax has many possible causes including embolic, arteritic, hypotensive, migrainous and vasospastic phenomena. The diagnostic dilemma occurs as a result of the presentation of a symptomatic patient who often presents with no physical findings. The initial workup is guided by history and will often include ESR/CRP, carotid and cardiac ultrasound and a hypercoagulable or other work-up as indicated by history. Vasospasm is a rarely reported cause of amaurosis and is a diagnosis of exclusion. Our case appears to be unique in that there were visible clinical findings in the retina consistent with nerve fibre layer infarctions. While the exact nature of these nerve fibre layer infarctions cannot be known, it can be speculated that they are the result of vasospasm sufficient to produce ischemia.

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Effects of pictorial illusions on monocular and binocular prehension tasks

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ABSTRACT (AS SUBMITTED)

Purpose: Researchers generally agree that there are two distinct, yet interacting visual pathways that process information about objects; one pathway mediates the perception of object properties (i.e. ventral stream) and the other guides actions towards these objects (i.e. dorsal stream). Consistent with the division of labour between action and perception, numerous studies have shown that actions like grasping are insensitive to illusions that influence participant's perception of object properties (size and location). With binocularity, there are various visual cues which give information about the absolute distance of an object from its viewer; however, the extent to which these cues are needed to resist visual illusions is poorly understood. The present study assessed the effect of a pictorial illusion on grasping under binocular and monocular conditions when distance is fixed or variable in order to determine if the resistance of grasping to pictorial illusions is dependent on the availability of binocular visual cues.

Methods: Fifteen participants of normal binocularity were asked to reach and grasp objects of different sizes under monocular and binocular conditions, while distance was either kept constant or varied randomly. Target objects were presented beside a flanking object which created the illusionary effect. Grip aperture was measured using the OPTOTRAK motion analysis system via infra-red emitting diodes placed on the thumb and forefinger.

Results: Participants were able to resist the illusion under binocular conditions in which distance was kept constant or varied; however, under monocular conditions, grip aperture was always affected by the perceptual effects of the illusion.

Conclusions: The resistance to visual illusions appears to be dependent on binocular cues when performing skilled actions like prehension. When binocular cues are unavailable, the visuomotor system seems to use remaining monocular cues causing the system to be susceptible to illusions. Contrary to our predications, however, the perceptual bias is so strong under monocular conditions, that participants were susceptible to the illusion even when the distance was held constant or fixed.

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A case of orbital apex syndrome associated with Wegener's granulomatosis and Herpes zoster
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ABSTRACT (AS SUBMITTED)

Purpose: Wegener's granulomatosis and Herpes zoster are rare cause of orbital apex syndrome, sporadically reported in the literature. No case of simultaneous involvement of both disease processes has been reported in the past.

Methods: We report a case of a 50-year-old Asian male with a history Wegener's granulomatosis who presented with Herpes zoster ophthalmicus and severe unilateral vision loss with ophthalmoplegia.

Results: The case patient had a several year history of Wegener's granulomatosis affecting the lung and causing hearing loss. Several months prior to the zoster episode, he presented with decreased vision in the left eye to HM secondary to a left CRAO related to the Wegener's granulomatosis. One month later, after medical treatment of his vasculitis, the left eye vision had returned to near baseline. However, when this patient presented with Herpes zoster ophthalmicus affecting the same left eye, several month later, he had NLP vision, keratitis, proptosis, complete ptosis, a frozen globe, and a V1 rash with dysesthesia. Cranial nerves II, III, IV, VI and V1 were all affected. Neuro-imaging failed to demonstrate any disease process in the orbit, orbital apex and cavernous sinus that could be causing this. Given the time course of the patient's disease progression and that he has been on prednisone as a baseline since his Wegener's related CRAO, it was felt that zoster was the more likely aetiology, although Wegener's granulomatosis as a contributing cause cannot be ruled out.

Conclusions: Wegener's granulomatosis and Herpes zoster are conditions that can profoundly compromise neurovascular structures in the orbital apex and cause severe vision loss and cranial nerve deficits.

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Ethambutol optic neuropathy: cases and suggested screening guidelines

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ABSTRACT (AS SUBMITTED)

Purpose: Ethambutol is a commonly used antimycobacterial agent. A well known toxic effect of this medication is optic neuropathy. Ethambutol toxicity has been found to be reversible in the medical literature, however recovery from the neuropathy has been incomplete in numerous cases. Currently, there are few guidelines regarding ophthalmologic screening and monitoring of patients on ethambutol. Following a review of five cases of ethambutol toxicity, we have developed suggested screening guidelines to follow patients on this medication.

Methods: Five cases of ethambutol optic neuropathy were retrospectively reviewed from the years 1989 - 2007. All five individuals were evaluated at varying intervals between two to nine months. Each patient underwent bilateral visual acuity assessments, visual field testing, colour testing – either HRR or D15, - visual evoked potential testing (VEP) and fundoscopic examinations. When patients developed symptoms of ethambutol optic neuropathy including disruptions in visual acuity, colour vision and visual fields they were advised to promptly discontinue ethambutol and were seen in follow-up to assess their progression.

Results: The five patients reviewed exhibited optic neuropathy between 6-10 months with an mean of 7 months from starting ethambutol. The first symptom experienced by all patients was a rapid decline in visual acuity. Four patients developed bilateral central scotomas on visual field testing, and 1 patient showed bilateral patchy scotomas. On D15 colour vision testing all affected patients exhibited decline. One patient showed specific defects along the blue-yellow axis bilaterally. Four out of five patients made some recovery following the cessation of ethambutol.

Conclusions: Optic neuropathy is a severe complication of ethambutol and leaves lasting deficits. We suggest that all patients who will be exposed to ethambutol have a baseline exam including visual fields within central ten degrees, visual acuity measurements, baseline pictures of their optic nerves as well as HRR colour plate and D15 colour testing. The medical literature has indicated that toxicity has not been found to develop in less than 1.5 months. We suggest routine ophthalmologic examinations of patients every 4-6 months for patients on 15mg/kg or less of ethambutol. The baseline exam should be completed within 6 weeks of starting therapy and follow-ups should be no more than 4 months apart. Those on increased dosages, with low lean body mass, and prolonged therapy may need to be followed more closely.