performed. Permission to perform a post-mortem was not granted.

Comments

Dysthyroid eye disease was considered the most likely diagnosis on the grounds of the patient's age, bilateral disease, MRI scan findings and response to treatment. However, there was no documentary evidence of an association between dysthyroid eye disease and gangrene of limb extremities. Therefore an assumption that the orbital and limb problems were features of the same disease would eliminate dysthroid eye disease from the list of likely causes unless this could be the first reported case. A very high ESR of 120 mm/h would be more in favour of inflammatory disease than dysthyroid eye disease, although acute thyroiditis is associated with moderately elevated ESR.

Generalised Wegener's granulomatosis can cause both orbital proptosis and gangrene and involvement of the kidneys and the respiratory tract is pathognomonic.[3] Our patient did not show signs of kidney and respiratory diseases as evidenced by normal levels of urea and electrolytes and a clear chest x-ray. Unfortunately an ANCA test for Wegener's granulomatosis was not performed on our patient and this disease has not been reported among blacks in Africa.[4]

Granulomatous orbital myositis is known to cause bilateral extraocular muscle enlargement. One reported case had elevated ANCA, normal thoracic imaging and characteristic histology findings on muscle biopsy.[5] Reported association of this condition with gangrene of the limbs could not be established.

Traditional medicines have been reported to cause gangrene of the limb extremities in Zimbabwe because of their powerful vasoconstriction effects.[6,7] Our patient admitted to taking oral traditional medicine prior to development of limb problems. Unfortunately the nature of the medicine was not disclosed to the patient. The onset of orbital symptoms in relation to the time when traditional medicine was ingested and the resolution of proptosis on systemic corticosteroids exonerate herbal medicine as a cause of orbital symptoms.

The rapid deterioration of the patient despite appropriate treatment could be attributed to HIV infection. Medical practitioners should beware of the modifying effect of traditional medicines and HIV infections on symptoms of various diseases. We emphasise that accurate and meticulous clinical history, examination and investigations are of paramount importance in reaching a correct diagnosis.

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A Magava, R Masanganise, KKN Kalangu and K Naik

Department of Surgery
University of Zimbabwe Medical School
Parirenyatwa Hospital, Box A178
Avondale, Harare, Zimbabwe

Correspondence: R Masanganise
Tel: 263 4 791631 ext 2169
Fax: 263 4 791995
E-mail: surgery@healthnet.zw

Sir,

Therapeutic external ophthalmoplegia with bilateral retrobulbar botulinum toxin—an effective treatment for acquired nystagmus with oscillopsia

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Acquired nystagmus is often associated with oscillopsia and visual impairment. Therapeutic options for this incapacitating condition remain limited. We report the case of a young male with nystagmus and oscillopsia, treated successfully with retrobulbar botulinum toxin A.

Case report

A 39-year-old male, confined to a wheelchair due to multiple sclerosis, manifested bilateral visual

impairment associated with distressing subjective perception of rhythmic movement of his surroundings.

Examination revealed fine pendular bilateral horizontal conjugate nystagmus in the primary position, changing to jerk nystagmus on lateral versions, without a definite null point, though vision was subjectively clearest in dextroelevation. Snellen acuity measured 6/36 and 1/60 in right and left eyes respectively. A left relative afferent pupillary defect corresponded with asymmetrical optic disc pallor, more pronounced in the left eye, from previous optic neuritis.

Right retrobulbar injection of botulinum toxin A (Dysport 40 units) was administered, 2 hours subsequent to which subjective visual improvement was first experienced. Later the same day, this evolved into diplopia, which however was clear, and anomalous spatial perception, his bed appearing to be inclined downhill. Vision continued to improve subjectively over the next week. However, by the 4th day the upper eyelid began to droop; the patient overcame this by taping it open, though this resulted in ocular irritation and watering. To alleviate this, he then employed tape to open the lid partially, and used his eyebrow to lift the lid enough to see, so that he could close his eye at will.

At review a month later, acuity had objectively improved to 6/12 in the right eye, which manifested total external ophthalmoplegia, with no nystagmus and profound ptosis with no levator function. Another month later, acuity had dropped to 6/36, with recurrence of fine nystagmus. The patient also reported bothersome oscillating images from his untreated left eye, which he actively ignored. At the patient's request bilateral retrobulbar toxin injections were administered.

Vision has subsequently remained stable, at around 6/12 in the right eye and 6/60 in the left eye, on regular 2-monthly bilateral injections of botulinum toxin (Dysport 20–40 units), 28 in total over the past 5 years. The injections are often followed by nausea and headache for 2 days, which however, resolve as ophthalmoplegia sets in by the third day. The patient prefers to tape both eyes open, despite persistent diplopia, since images from the left eye are no longer distressing. On lower doses of toxin, ptosis is less profound, though the degree and duration of visual benefit remain unchanged.

Comment

Acquired nystagmus is often associated with impaired visual function, due to excessive movement of retinal images. Failure of the visual cortex to adapt to nystagmus results in oscillopsia, which may be incapacitating.[1]

Therapeutic options include correction of refractive errors,[2] optical dampening of oscillopsia with contact lenses and spectacles,[3] gabapentin,[4] memantine[5] and botulinum toxin, none of which are universally effective.

External ophthalmoplegia with retrobulbar botulinum toxin as a treatment for acquired nystagmus was first described in 1988,[6] subsequent to which there have been few subsequent reports.[7–9] Repeated injections are required to maintain therapeutic effect, and entail the risks of retrobulbar haemorrhage, damage to orbital contents and ptosis.[7,9] Inadvertent intravascular injection of toxin however, appears to be of little significance, the dose being insufficient to produce systemic toxicity.[6]

Our patient experienced relief from intractable oscillopsia, with significant improvement of spatial acuity on therapeutic ophthalmoplegia, suggesting that nystagmus was the cause, rather than the effect of reduced vision. Subjective visual improvement 2 hours after treatment suggests an initial placebo effect. Subsequently the only side effect noted was ptosis of insufficient severity or permanence to warrant surgical intervention. Images from the weaker left eye are no longer bothersome after bilateral ophthalmoplegia.

External ophthalmoplegia entails loss of saccadic and pursuit movements, including those involved in the vestibulo-ocular reflexes, which may cause oscillopsia on head movement.[8] This however appears of little significance in the context of visual improvement in our patient with debilitating neurological disease. We submit that retrobulbar injection of botulinum toxin is therefore an effective and relatively safe therapeutic option in the treatment of distressing oscillopsia in non-ambulatory patients.

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GJ Menon and VT Thaller

Royal Eye Infirmary
Apsley Road
Plymouth, PL4 6PL, UK

Correspondence: GJ Menon FRCS FRCOphth
Tel: 01752 315149
E-mail: sharmilajk@aol.com