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A case of constant esotropia with diplopia associated with weight loss in anorexia nervosa

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Abstract

Aim: **To describe the case of a 21-year-old woman who presented with a constant esotropia with diplopia following the onset of anorexia nervosa, in whom the control of her strabismus and diplopia improved as body weight was regained.**
Method: **The orthoptic findings are presented and the outcome of treatment with botulinum toxin injection is reported.**
Results: **Botulinum toxin injection reduced the angle of deviation from 50$^{\Delta}$ base-out (BO) for distance and 45$^{\Delta}$ BO for near, to 30$^{\Delta}$ BO for distance and 25$^{\Delta}$ BO for near. After the injection the patient reported a marked improvement in symptoms and experienced diplopia only when tired. Over time and in conjunction with weight gain the deviation was controlled to a microtropia with an associated latent component with demonstrable anomalous binocular vision.**
Conclusion: **This case shows the possible effects of anorexia on the balance of the oculomotor system, with the onset of a convergent squint at a time of severe weight loss. Botulinum toxin injection under local anaesthetic may be the wisest and least invasive therapeutic choice in these patients, thereby avoiding a surgical procedure under general anaesthesia, which may be unsuitable in cases of anorexia.**

Key words: Anorexia, Decompensation, Esotropia

Introduction

Anorexia nervosa is an eating disorder resulting in profound weight loss with malnutrition. Ophthalmological signs have been described in regard to conjunctival, lens and retinal changes.[1,2] Patients with anorexia have been shown to have a metabolic myopathy of skeletal muscle.[3] Although smooth pursuit abnormalities have been reported in anorexia nervosa,[4] ocular motility defects are not well described. We report a patient who presented with a constant esotropia associated with diplopia following the onset of anorexia nervosa, in whom the control of her strabismus and diplopia improved as she regained her body weight.

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Case report

A 21-year-old woman presented with a left convergent squint with diplopia of 3 years' duration. At the time the diplopia commenced the patient had been diagnosed with anorexia nervosa and she attributed the onset of her symptoms to the beginning of her weight loss, with gradual deterioration of her ocular alignment over this time. Her lowest weight recorded was 28.2 kg, resulting in a body mass index (BMI) of 11.0 (<18.5 is considered underweight).

There was no history in childhood of squint or occlusion therapy. The referring consultant had seen the patient at the age of 10 years due to complaints of blurred vision and reading difficulty coinciding with a period of family stress. At this time there was no refractive error or diplopia. When aged 18 years and at her lowest body weight the patient's optometrist had advised her that she had developed a squint and prescribed low-strength base-out prismatic glasses. No orthoptic assessment was carried out at that time.

At presentation to our clinic at age 21 years corrected acuity was 6/4–2 in each eye and unaided acuity 6/5 + 2 right and 6/5–2 left. The refractive correction was right and left, plano/–0.75 × 90. On cover test there was a left convergent squint, which could be 'controlled' briefly. The angle of deviation was 50$^{\Delta}$ base-out (BO) for distance and 45$^{\Delta}$ BO for near using prism and cover test. Diplopia was appreciated for near and suppression for distance, which was confirmed on Bagolini glasses. Synoptophore assessment indicated intermittent suppression with no demonstrable fusion range. Ocular movements, including saccades, were normal.

Ophthalmological examination was otherwise normal, with normal-sized pupils and no evidence of lens or mucous membrane changes. The patient was treated with an injection of botulinum toxin (2.5 international units of Dysport; Ipsen, Slough, UK) into the left medial rectus muscle. To ensure correct muscle placement this was carried out under electromyographic control, which indicated a normal amplitude and normal density.

Three weeks after the botox injection, cover test at near and distance revealed an esophoria, with recovery aided by blinking, which measured 35$^{\Delta}$ BO. Near binocular visual acuity was 6/6; stereopsis was 30″ of arc using TNO and diplopia was appreciated only when tired. At 3 months after the injection the patient's body weight had begun to increase and she did not wish for further treatment at that time. During the following 12 months the patient continued to gain weight, stabilising

at 42 kg (BMI 16.4), and this coincided with an improvement in her ability to control the residual deviation to the extent that she was aware of diplopia only when very tired. At final orthoptic assessment there was a minimal left convergent squint with an associated large esophoria. The maximal deviation measured was 25^{Δ} BO for near and 30^{Δ} BO for distance. An X response for near and distance was reported using Bagolini glasses and stereopsis of 55″ of arc was achieved using the Frisby stereotest. No further follow-up was arranged.

Discussion

Anorexia nervosa affects 1–3% of middle and upper class women, with a prevalence of 20:1 in women compared with men.[2] Sufferers from anorexia severely limit their intake of carbohydrate and fatty foods but usually have a relatively good protein intake.[2,3] In severe cases the malnutrition becomes worse, producing protein energy malnutrition.[3] Other forms of malnutrition such as kwashiorkor* have severe protein and calorie deficiency. Eye findings in patients with malnutrition have been reported to relate to vitamin A deficiency, with conjunctival, lens and retinal changes.[5] The ophthalmological signs in anorexia nervosa have been presumed to be due to vitamin A deficiency although the evidence for such changes is not strong.[1] Ophthalmic changes reported in anorectics include a higher incidence of episcleral capillary aneurysms, subconjunctival haemorrhages, superficial punctate keratopathy and reduced tear formation compared with normal controls.[2] However, there is no report in the literature regarding the structural and functional changes in ocular muscle in anorexia nervosa and the consequent effect on oculomotor control.

Impaired muscle function, myopathy on electromyography and myopathic change with severe type 2 muscle fibre atrophy have been shown in patients with severe induced weight loss.[3] The myopathy was metabolic with an accumulation of glycogen between the myofibrils and was not due to a neuropathy. There are no reports on the histopathological findings in the extraocular muscles in anorexia or other forms of malnutrition, but changes in skeletal muscle have been described in patients with anorexia.[3] In light of this histological change in skeletal muscle it is possible that there may have been an alteration in the extraocular muscle structure of our patient. However, the electromyogram for this patient obtained at the time of toxin injection was normal.

This patient presented with a left convergent squint with intermittent diplopia, which was associated with weight loss in anorexia nervosa. We presume that the patient had a previously unrecognised left microesotropia that had increased due to her systemic illness. Following toxin treatment the squint returned to a left microesotropia with a larger latent component. The increase in deviation could have been due to loss of central control but may also have a structural basis. In favour of a possible structural causation, muscle power in anorectic patients with myopathy reportedly recovers with improved nutrition and this may have been a factor in our case.

This report shows the possible effects of anorexia on oculomotor balance, with a convergent squint occurring at a time of severe weight loss. In such cases the effect of improved nutrition may be helpful in improving oculomotor balance. Toxin therapy under local anaesthetic may be the wisest and least invasive therapeutic choice in these patients, thereby avoiding a surgical procedure under general anaesthesia for which they may be unsuitable.

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*A disease of protein malnutrition in which there is insufficient protein intake to meet bodily demands for protein synthesis and tissue repair, with progressive erosion of lean body mass.