the three patients with myasthenia with bilateral EOM atrophy reported by Okamoto *et al*,[4] who were positive for only acetylcholine-receptor antibodies, our patient was seronegative for these antibodies but positive for MuSK antibody. In ocular myasthenia, only 50% of patients are found to have anti-acetylcholine receptor antibodies,[6] and of 38 patients with seronegative ocular MG, none show increased titres of the anti-MuSK antibody.[9 10] Between 38% and 47% of patients with seronegative MG have increased anti-MuSK antibodies.[9 10] Similar to the clinical course of patients in the study by Okamoto *et al*,[4] our patient had an approximately 15 year duration of limited ocular motility that did not improve with medication or thymectomy.

Diplopia and ptosis are well-recognised signs of MG, but bilateral EOM atrophy is exceedingly rare. Although ocular involvement is a less common presentation of the anti-MuSK MG syndrome, it may be reasonable to check for anti-MuSK antibody in any patient who presents with progressive external ophthalmoplegia.

Jane W Chan
Department of Neurology, University of Nevada, School of Medicine, Las Vegas, Nevada, USA

William W Orrison
Nevada Imaging Center, Las Vegas, Nevada, USA

Correspondence to: J W Chan, Department of Neurology, University of Nevada School of Medicine, 1707 W Charleston Blvd, Suite 220, Las Vegas, Nevada 89102, USA; worjun@aol.com

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Botulinum toxin injection causing lateral rectus palsy

A 35-year-old woman with brow spasms, treated in the past with Botox, was referred for increasing tightness in the brow. She reported having experienced an episode of difficulty focusing after a previous injection of Botox by another physician. The patient had no response to a subcutaneous injection consisting of 10 units of Botox above the eyebrows, nor to a subsequent injection of 20 units in the same location 2 months later. A third injection of 35 units of Botox resulted in improvement of her brow spasms, but only for 48 h. Accordingly, 2 months after the third injection, she was injected with 75 units of Botox, with 30 units injected in three separate locations above each eyebrow and 7.5 units injected just lateral to each lateral canthus. At 1 week after this injection, the patient had difficulty focusing, and shortly thereafter noted horizontal binocular double vision, worse on left gaze.

On examination 48 h after the onset of her symptoms, the patient had normal visual acuity of 20/20 OU, with normal colour vision and full visual fields. She had anisocoria, with the right pupil being 0.5 mm larger than the left, but both pupils reacted briskly to light stimulation and both dilated equally well after topical administration of a 10% cocaine solution. The right eye moved fully in all directions. The left eye had mild limitation of abduction, but otherwise moved fully. Duction measurements revealed 60° of abduction of the right eye versus 50° of the left eye. In primary position, the patient had an esotropia of 9 prism diopters (PD) at distance and 2 PD at near. The esotropia was incomitant, and increased on left gaze to 15 PD. Corneal and facial sensation were equal and normal bilaterally. Slit-lamp examination and fundoscopy were normal. MRI of the brain and orbit were normal.

The patient was treated with a 6-PD Fresnel prism placed base out on her left spectacle lens for temporary symptom relief. One month later, her diplopia had resolved and she was able to remove the prism.

Comment

This is the first reported case of lateral rectus paresis after an injection of Botox into the lateral canthal region. Inferior oblique paresis is an uncommon adverse effect of Botox injection into the lower lid,[1] with a reported incidence of 1.7%.[2] The mechanism is postulated to be diffusion of the medication to the underlying inferior oblique muscle. Diffusion of botulinum toxin to surrounding muscles has also been reported after treatment of dystonia with large doses of the drug.[3] We believe that, in our case, Botox spread from the lateral canthus to the left lateral rectus muscle, producing a transient paresis of the muscle characterised by an incomitant esotropia and a mild left abduction deficit. It is interesting that patients who experience diplopia after a periorbital injection of Botox tend to have recurrence of diplopia when they are reinjected, suggesting that they have some predisposition to this complication.[2] Our patient had an episode of difficulty focusing after a previous periorbital injection of Botox by another physician, which was similar to the prodrome that she experienced just before she developed diplopia after the injection we gave her. Fortunately, the diplopia was transient, resolving as the effects of the botulinum toxin dissipated. We believe that patients who experience diplopia or have difficulty focusing after an injection of botulinum toxin should be counselled as to the potential recurrence of such an effect with subsequent injections of the drug, regardless of the care taken to avoid these sequelae.

Celia S Chen
Neuro-Ophthalmology Unit, Wilmer Eye Institute, The Johns Hopkins Hospital, Baltimore, Maryland, USA

Neil R Miller
Wilmer Eye Institute, The Johns Hopkins Hospital, Baltimore, Maryland, USA

Correspondence to: Dr N R Miller, Maumenee 127, Wilmer Eye Institute, Johns Hopkins Hospital, 600 North Wolfe Street, Baltimore, MD 21287, USA; nrmiller@jhmi.edu

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Abducens palsy and Sjögren's syndrome induced by pegylated interferon therapy

Interferons (IFNs) and their pegylated forms (PEG-IFNs) are widely used in the treatment of viral hepatitis and some neoplasms. Although ophthalmic symptoms are common among their various side effects,[1 2] abducens palsy is rarely observed. Here, we describe a case where abducens palsy developed during PEG-IFN therapy, and discuss the management.

Case report

A 65-year-old man had undergone removal of stage IV renal cell carcinoma 5 years ago. He had been managed with IFNα therapy (6×10^6 IU three times a week) for recurrence. Although the therapy suppressed tumour growth effectively, it had moderate side effects including influenza-like symptoms, erythema and depression. After 4 years of conventional IFN therapy, PEG-IFNα became available for the patient.

On the first day of PEG-IFN therapy (3×10^6 IU), the patient had the expected fever and malaise. The next day he developed diplopia, which worsened gradually. After 1 week, the patient was referred to ophthalmologists and was diagnosed as having right abducens palsy (fig 1, upper panel). IFN-induced retinopathy was absent. Diabetes mellitus was negative with fasting glucose (73 mg/dl) and haemoglobin A1c (5.5%). He did not have hypertension. MRI showed no evidence of metastasis or intracranial hypertension, but showed enhancement of the right abducens nerve (fig 2).

After 1 month of onset of diplopia, painful swelling of the right parotid gland was noted. In another 1 month, dry eye and dry mouth sensations developed. The result of Schirmer's test (3 mm) and fluorescent corneal staining suggested presumable Sjögren's syndrome (SS). Furthermore, serum examination showed increased SS A and SS B antibody levels (128