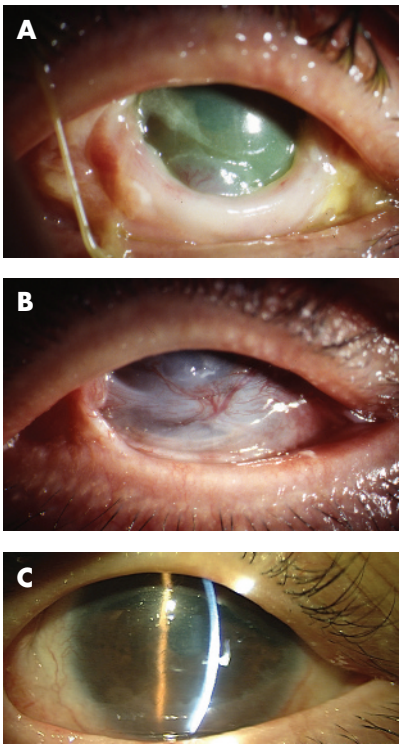

Figure 1 (A) Slit-lamp examination showing severe conjunctival inflammation and conjunctival shortening before intravenous administration of immunoglobulin (IVIg) treatment. (B) Ten months after the IVIg treatment, scarring and conjunctivalisation on the cornea can be observed with progressive cataract. (C) Six months after the cultivated oral mucosal epithelial transplantation, improvement in clarity of the cornea and conjunctival shortening can be observed.

test value was 0 mm and BCVA was decreased to hand motion in both eyes (fig 1B). To obtain visual improvement, surgery was performed in the right eye 10 months after the start of IVIg. Firstly, we removed all fibrotic tissue, then we applied 0.04% mitomycin C for 3 min, followed by irrigation with saline solution. Phacoemulsification and intraocular lens insertion were performed after the cornea had regained clarity. Cultivated autologous oral mucosal epithelial sheets prepared on preserved human amniotic membrane for 2 weeks[4] were transplanted on the exposed ocular surface. Postoperatively, both the cornea and sclera were promptly epithelialised, and marked reduction in fibrosis was noted. One year after surgery, BCVA had improved to 20/300 and the corneal clarity was maintained (fig 1C). Improvements in vision and reduction in photophobia enabled the patient to return to social activities.

For the treatment of an active OCP, both control of autoimmune reaction and ocular surface reconstruction are necessary. We believe that the combination of IVIg and COMET is a powerful treatment modality while minimising the risk of postoperative consequences inevitably associated with immunosuppression.

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The potential role of testosterone in central serous chorioretinopathy

Risk factors for central serous chorioretinopathy (CSCR) are male gender, psychological stress, type-A personality, corticoid–steroid treatment and pregnancy. The reason for the presence of male gender as a risk factor is not yet clear. One possibility is a direct influence of androgens. We report a case of a female patient who developed CSCR under testosterone treatment.

Case report

A 45-year-old non-pregnant woman presented with a 1-day history of metamorphopsia and scotoma in the right eye. She had no eye problems in the past, but her current profession as a manager exposed her to several stress situations. On examination of the right eye, the best-corrected visual acuity (BCVA) was 20/25; paracentral scotoma and metamorphopsia were confirmed using the Amsler grid. Fundus examination disclosed a neurosensory serous detachment, and fluorescein angiography ruled out choroidal neovascular membrane. Optical coherence tomography (OCT) showed a typical CSCR (fig 1A). The left eye had a BCVA of 20/20 and did not show any pathology. At the second visit 3 weeks later and on being questioned again, the patient reported that she was on oral testosterone undecanoat 40 mg/day for 2 months because of a general loss of energy and symptoms of fatigue attributed to a low level of endogenous testosterone. At this stage, the plasma-testosterone level was markedly increased (table 1).

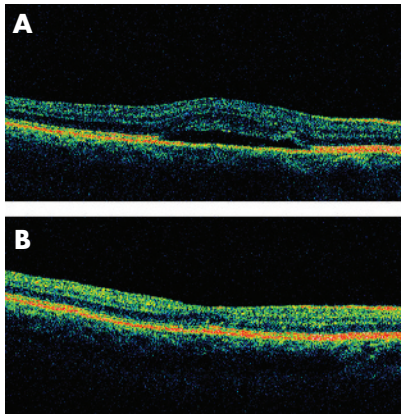

Figure 1 (A) Optical computerised tomography (OCT) confirming serous neurosensory and retinal pigment epithelium detachment in the right eye. (B) OCT of the right eye at 4-month follow-up showing reattachment of the retina.

The testosterone treatment was stopped immediately. Oral oestradiol 1 mg/day was given for climacteric symptoms and for prevention of osteoporosis. This produced an increase in circulating oestradiol levels together with feedback suppression of both gonadotropins, luteinising hormone and follicle-stimulating hormone, as well as an increase in the sex-hormone-binding globulin. Sex-hormone-binding globulin enhances the binding of testosterone on this protein, thereby decreasing free circulating testosterone.

At 1 month follow-up, BCVA was 20/20, and angiography showed no signs of leakage or pigment epithelial detachment. Her blood level of testosterone dropped to values in the lower normal range (table 1). The neurosensory retina was reattached as confirmed by OCT at 4 months (fig 1B).

Table 1 Summary of the hormone levels

	Values on testosterone treatment*	Values after cessation of androgen treatment†	Normal range for females
Total testosterone	5.30	0.69	0.5–3.0 nmol/l
SHBG	37	141	20–118 nmol/l
Oestradiol	73	383	40–200 pmol/l
DHEA-S	3.6	1.2	3–12 μmol/l
LH	60	24	>10 E/l (menopausal)
FSH	56	29	>15 E/l (menopausal)

DHEA-S, dehydroepiandrosterone sulphate; FSH, follicle-stimulating hormones; LH, luteinising hormone; SHBG, sex-hormone-binding globulin.
*Oral testosterone undecanoat (40 mg/day).
†Oral oestradiol (1 mg/day).

Comment

The pathogenesis of CSCR is not yet fully understood. It is known, however, that it affects mainly men from 20 to 45 years of age, with type-A personality, and is often triggered by emotional stress.

Our patient had indeed a type-A personality and was under professional stress. Nevertheless, we were puzzled to see CSCR in a 45-year-old woman who was not hypertensive, pregnant or under steroid treatment. Detailed questioning, however, disclosed testosterone intake. No relapse of CSCR was observed following cessation of testosterone treatment. We assume therefore that the testosterone treatment in this case may have contributed to the disease. We even postulate that a relatively high level of testosterone may be a risk factor for CSCR in general, as androgen receptors have been found in human retinal pigment epithelial cells.[1] This hypothesis would also explain the preponderance of males CSCR as well as the with age range of patients by age dependence of testosterone level.[2] Further, it is known that subjects with type-A personality on average have higher testosterone levels,[3] which may be further increased under emotional stress.[4]

Most diseases result from the interplay of many factors. For CSCR, testosterone seems to be one of these acting factors.

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High-speed, ultra-high-resolution optical coherence tomography of acute macular neuroretinopathy

Acute macular neuroretinopathy (AMN) is a rare, macular disorder of unknown aetiology. Patients with AMN are typically young women who present with paracentral scotomata in one or both eyes corresponding to red wedge-shaped parafoveal lesions. The retinal location of the lesion in patients with AMN is not clear. High-speed, ultra-high-resolution optical coherence tomography (hsUHR-OCT) is an investigational research prototype instrument capable of producing cross-sectional images of the retina; it supports an axial resolution of about 3.5 μm compared with about 10 μm in Stratus OCT (Dublin, California, USA)[1] which enables enhanced imaging of intraretinal morphology including photoreceptor inner segments, outer segments and the external limiting membrane.[2] We report a patient with AMN who underwent imaging with hsUHR-OCT suggesting that the lesion in AMN is located in the outer retina.

Case report

A 51-year-old woman presented with a 10-day history of a sudden onset of a grey, oval paracentral scotoma in her right eye. Her medical history was notable for systemic hypertension; her drugs included trivoral and ramipril. Best-corrected visual acuity was 20/25 in both eyes. She was able to precisely demarcate the paracentral scotoma on an Amsler grid. Funduscopy of the right eye showed a focal, reddish petaloid lesion superior to fixation. Fluorescein angiography and images on the Stratus OCT were unremarkable. hs UHR-OCT images showed focal depression of the external limiting membrane, inner/outer photoreceptor segment (IS/OS) junction, photoreceptors and retinal pigment epithelium. Changes in the photoreceptor outer segments (fig 1A) in the region of the petaloid lesion were also noted. The inner retina appeared normal.

After 3 months, visual acuity was 20/25 in the right eye, and the patient reported a slight reduction of the scotoma. Funduscopy showed resolution of the petaloid lesion. hsUHR-OCT showed realignment of the outer photoreceptor layer and the IS/OS junction (fig 1B).

Comments

The pathogenesis of AMN remains unknown, although an acute inflammatory process or

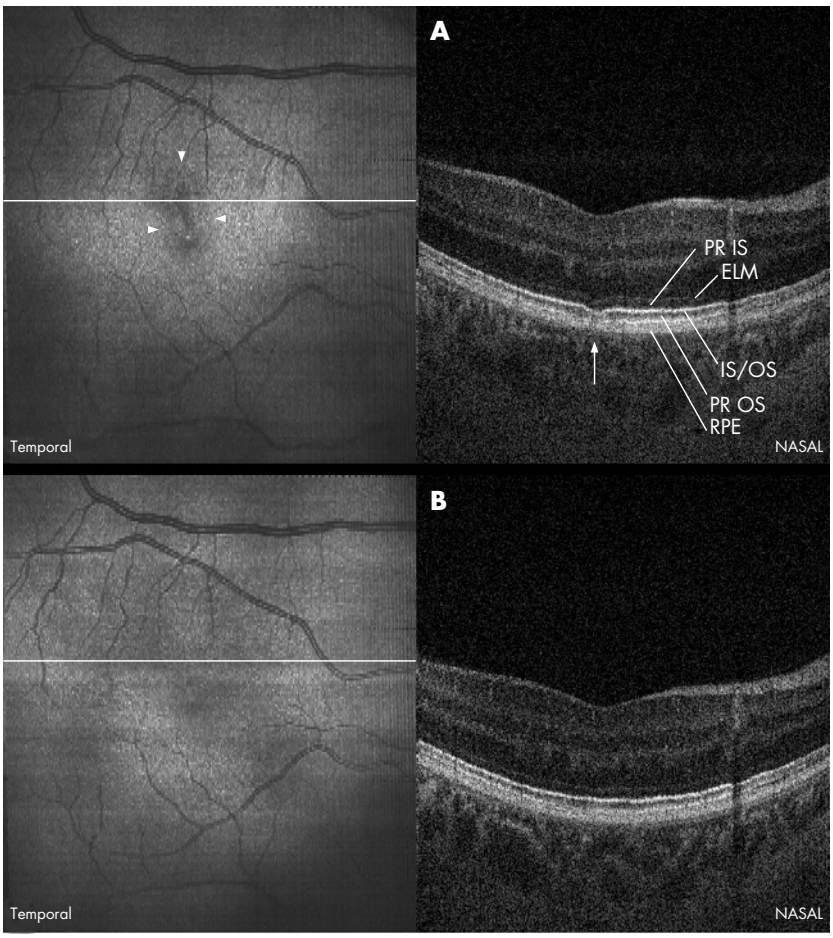

Figure 1 Three-dimensional, high-speed ultra-high-resolution optical coherence tomography scan of the macula. A 180-image raster scan (512 axial scans per image) provides a 6×6-mm pattern. The cross-sectional images are precisely registered to a fundus image created from an anterior view of all 180 images. (A) Perimacular petaloid lesion (arrowheads), corresponding to a focal reduction in the photoreceptor outer segments (arrow). (B) Marked resolution of both the petaloid lesion and outer segment morphology at 3-month follow-up. ELM, external limiting membrane; IS/OS, inner/outer segment photoreceptor junction; PR IS, photoreceptor inner segments; PR OS, photoreceptor outer segments; RPE, retinal pigment epithelium.