Central serous chorioretinopathy (CSCR) is a sporadic self-limited disease, characterised by the idiopathic detachment of the neural retina due to accumulation of transparent fluid in the macular region. It predominantly affects men aged between 25 and 50 years. Schatz described the concept of leakage at the level of the retinal pigment epithelial layer, while more recent studies using indocyanine green angiography have indicated that the possible site of primary pathology is the choroidal vessels and the involvement of the pigment epithelial layer is only secondary (for a review of pathophysiology see Schatz).

Visual prognosis is good, as the fluid resolves spontaneously, that is, the average resolution time without treatment is between three and six months, with most of the eyes retaining the initial visual acuity after the resolution of the acute stage. The cause of CSR is unknown, although the condition is commonly associated with stress or ‘type A’ personalities. Other potential risk factors include systemic steroid use and pregnancy. It is a poorly understood disease, as there is no significant correlation between time and progression of the retinal pigment epitheliopathy following resolution of CSCR.

THE CASE
A 39-year-old hyperopic male was referred for laser refractive treatment. In the course of the pre-operative evaluation he complained of a recent deterioration of vision. The suspicion of unilateral central serous chorioretinopathy (CSCR) was confirmed by contrast sensitivity testing and by ocular fundus examination. Contrast sensitivity (CS) for six spatial frequencies (1, 2, 4, 8, 12 and 16 c/deg) was evaluated using Gabor patches of gratings projected on a high-resolution display by means of a stimulus generator card. Although VA remained unaltered, the pattern of contrast sensitivity function varied at different stages of CSCR: during the acute stage, performance at all spatial frequencies was depressed, while at two-month follow up, intermediate and high spatial frequencies were mainly affected. It is concluded that the level of visual deficit in CSCR cannot be evaluated by measuring visual acuity. History and contrast sensitivity can play a central role in setting the correct diagnosis and characterising its stage.
Contrast sensitivity in central serous chorioretinopathy
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(Decimal 1.20) and LE: 0.24 logMAR (decimal 0.57). The cycloplegic refraction was RE: +6.00/-0.25 × 135, LE: +7.50/-0.75 × 30. The patient reported reduced brightness perception and some distortion of lines on the Amsler grid tested with his RE.

Monocular contrast sensitivity (CS) was evaluated using Gabor patches of gratings displayed on a Sony GDM F-520 CRT monitor (mean luminance: 30 cd/m², refresh rate: 120 Hz) by means of a VSG2/5 stimulus generator card (CRS, Rochester, UK). Gabor patches measured 100 pixels in diameter at half height, with a standard deviation subtending 1.2 deg at a two metre distance. Six spatial frequencies, 1, 2, 4, 8, 12 and 16 c/deg, were tested. The average of three thresholds was taken. Threshold was determined using a binary-search staircase with a contrast resolution of one decibel (0.05 log units).

Although VA was normal and better in the affected RE compared to the fellow asymptomatic (but amblyopic) eye, CS was depressed in all spatial frequencies tested, with the effect being more pronounced for the intermediate and high spatial frequencies (greater than four cycles per degree) (Figure 1). In addition, both eyes had decreased CS values when compared to normal data. Fundus photography, performed using the Kowa PRO 1 fundus camera (Kowa Co Ltd, Japan), revealed a local elevation of the neurosensory retina typical of CSCR (Figure 2).

Two months following the acute stage, the patient reported improved vision. The manifest refraction was RE: +3.50/-1.00 × 120, LE: +5.50/-1.00 × 20. Although no difference was observed in VA [RE: -0.02 logMAR (decimal 1.05), LE: 0.22 logMAR (decimal 0.60)], CS performance of the affected eye was improved, especially for high spatial frequencies but remained below normal for the whole range of spatial frequencies. This correlated well with fundus photography, associated with several macular retinal pigment epithelial (RPE) changes, with the subretinal fluid being absorbed (Figure 2), indicating a partial resolution of CSCR.

Figure 1. Upper graph: Contrast sensitivity (CS) evaluation of the symptomatic RE (left) and asymptomatic but amblyopic LE (right) at presentation (filled circles) and two months later (open squares) for a range of spatial frequencies. The dashed line represents average CS data (recorded with the same procedure). The grey shaded area covers the 95 per cent confidence intervals for a population of 28 normal eyes.

Lower graph: Difference in CS between average values in normal young eyes and the RE (left) and LE (right) of the patient at presentation and two months later. Note the improved CS in RE at two months and the attenuation of the high spatial frequency in CS for the amblyopic LE.

Figure 2. Fundus photography at presentation (left) and two months later (right). Symptomatic RE, at presentation, shows a typical central patch of subretinal fluid, which is resolved after two months, although several macular RPE changes are still evident.
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DISCUSSION

It has been proposed that visual acuity is satisfactory even at the acute stage of CSCR. In this case report, the patient reported a loss in perceived brightness of objects in his dominant RE, with the VA being unaffected. The hyperopic shift in refraction was minor, possibly due to its partial recovery at the time of pre-operative evaluation.

In an amblyopic patient in his late 30s, with some degree of latent hyperopia, visual disturbances could be attributed to pre-presbyopic accommodative decompensation. Given the transient hyperopic shift induced by the disease, a failure to identify CSCR in pre-operative evaluation for hyperopic refractive correction could have resulted in inappropriate over-correction. Contrast sensitivity forms a powerful non-invasive tool for the clinician to determine selective deficits in the visual function. The pattern of the contrast sensitivity function at presentation, that is, attenuation at all spatial frequencies, indicated the presence of a pathology that does not affect primarily the spatial resolution (which is the pattern observed in the presence of low magnitudes of optical defocus), and thus is not well represented in clinical measures such as VA. The limit of resolution for the RE as deduced by the CS function was 16 c/deg (Figure 1), corresponding to a visual acuity of 0.27 logMAR. The higher recognition (letter) acuity compared to resolution (grating) acuity, especially for patients with retinal pathologies, agrees with findings from previous studies.

Two months following the acute stage of the CSCR, the fluid was resolved but the deficiency persisted in the intermediate and high spatial frequencies in the contrast sensitivity function (Figure 1), which is the typical pattern reported elsewhere. Moreover, it has been suggested that in unilateral CSR, the fellow eye may be sub-clinically affected. This could not be investigated in our case as the contrast sensitivity of the fellow eye was decreased due to amblyopia.

Although fundus examination is the best method for diagnosing and monitoring CSCR, a full history and measurement of contrast sensitivity can help in confirming the diagnosis, especially in cases in which the level of visual loss cannot be established by the measurement of visual acuity. Finally, it should be noted that alternative, more sophisticated imaging equipment, such as optical coherence tomography (OCT), has been used to evaluate three-dimensional morphological retinotopic changes and monitor the progress in acute CSCR. OCT scans usually show a posterior layer dipping of the neurosensory retina onto the RPE, associated with subretinal ink-block leakage in fundus fluorescein angiography. However, OCT equipment is expensive and usually not available in refractive surgery clinics and optometric practice.

REFERENCES


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