BJO Online First, published on August 22, 2013 as 10.1136/bjophthalmol-2013-303707

Central serous chorioretinopathy in a 91-year-old woman

CASE REPORT

A 91-year-old Caucasian woman presented at the New England Eye Center at Tufts Medical Center in Boston, Massachusetts in February 2012, with decreased vision in her left eye. She had a history of high blood pressure, thyroid disease and polymyalgia rheumatica. She was pseudophakic and had primary open angle glaucoma. On presentation, she was taking 2 mg of prednisone daily for polymyalgia rheumatica.

On ophthalmic examination, the best-corrected visual acuity (BCVA) was 20/25 in the right eye and 20/60 in the left eye. Dilated fundus examination showed mild retinal pigment epithelium (RPE) changes in the macula in her right eye and subretinal fluid involving the macula in her left eye (figure 1). Fluorescein angiography (FA) showed a well-demarcated area of hyperfluorescence in her left eye with leakage in the late phase (figure 1). Optical coherence tomography (OCT) of the left eye revealed a thickened choroid that could not be accurately measured because the choroidoscleral interface was not visualised. By estimates, it was more than two-times thicker than the age-adjusted normal of 225 µm.[1] OCT also showed an exudative retinal detachment consistent with the diagnosis of central serous chorioretinopathy (CSCR) (figure 1). The right eye also had a thicker than normal choroid measuring 459 µm from the outer border of the hyper-reflective RPE to the inner aspect of the choroidoscleral interface beneath the fovea, but without exudative retinal detachment, RPE detachment or evidence of age-related macular degeneration (AMD) (figure 1).

Over the ensuing 3 months, she was tapered off prednisone but the subretinal fluid in the left eye persisted. Three months following the discontinuation of corticosteroids, her BCVA decreased to counting fingers at 8 feet. An indocyanine green (ICG) angiography was performed, which showed diffuse leakage in the late frames in the left eye (figure 1). She was treated with a session of half-time photodynamic therapy (PDT) which resulted in significant subjective and anatomical improvement (figure 2). On follow-up 4 months after PDT, her BCVA was 20/30, and OCT showed minimal subretinal fluid (figure 2).

QUESTIONS

1. What is CSCR and what are its symptoms?
2. How is CSCR diagnosed?
3. How are patients with CSCR managed?

ANSWERS

1. What is CSCR and what are its symptoms?

CSCR is a chorioretinal disease that involves dilation and hyperpermeability of choroidal vessels that results in a serous/exudative detachment of the neurosensory retina from the RPE with accumulation of serous subretinal fluid involving the macula.[2–3] Patients usually present with metamorphopsia, blurred vision, colour desaturation and a relative scotoma in the affected eye.[2] Typically, CSCR is seen in men aged between 20 and 50 years who present with unilateral metamorphopsia.[2] In women, it occurs less commonly, with the mean age of 51 years on presentation.[3]

2. How is CSCR diagnosed?

CSCR is diagnosed on clinical examination and investigations such as fundus autofluorescence, FA, ICG angiography and OCT imaging. Typically, CSCR is characterised by RPE alterations seen as atrophic dependent tracks on fundus autofluorescence.[4] FA shows leaking dye from the choroid through a defect in the RPE that accumulates in the subretinal space. ICG angiography is specially useful for diagnosis,[2] and shows a distinct area of hyperfluorescence suggesting leakage in the mid to late phase, as observed in the present case (figure 1). In elderly patients, it is difficult to distinguish CSCR from wet AMD. Recent advances in OCT helps distinguish CSCR from AMD. An exudative retinal detachment with an increase in the extent of subretinal fluid accumulation and hypertrophic outer retinal changes are more suggestive of CSCR than AMD.[5] In addition, the choroid is thicker than normal in CSCR as in the present case, while it is significantly thinner than normal in AMD.[6 7]

3. How are patients with CSCR managed?

Patients with CSCR are first observed for spontaneous resolution. Those with known risk factors for the disease such as corticosteroid use as in the present case, are tapered off of the

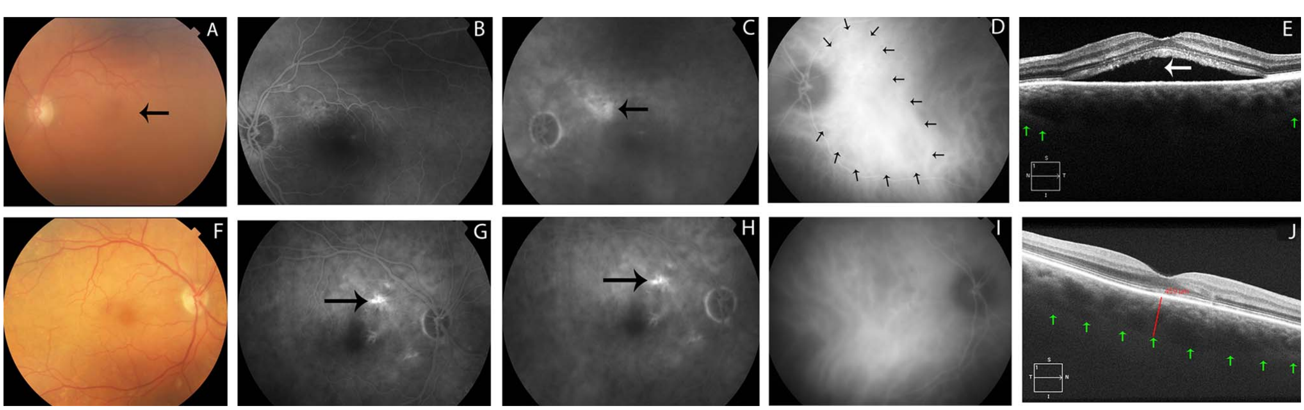

Figure 1 (A) Colour fundus photograph of the left eye showing subretinal fluid in the macula (black arrow). (B and C) FA (early and late frames respectively) of the left eye showing a well-demarcated area of leakage in the late frame (black arrow). (D) ICG angiography (late frame) of the left eye showing diffuse leakage (black arrows). (E) OCT of the left eye showing a thickened choroid and subretinal fluid (white arrow). Choroidoscleral interface is barely visible (green arrows). (F) Colour fundus photograph of the right eye showing mild RPE changes without evidence of subretinal fluid (G and H) FA (early and late frames respectively) of the right eye, showing a window defect due possibly to the mild RPE changes (black arrows) without evidence of leakage. (I) ICG angiography (late frame) of the right eye showing no evidence of leakage. (J) OCT of the right eye showing a clearly visible choroidoscleral interface (green arrows) allowing measurement of choroidal thickness (459 µm), and no subretinal fluid. For colour figures please see the online version of the journal. FA, fluorescein angiography; ICG, indocyanine green; OCT, optical coherence tomography; RPE, retinal pigment epithelium.

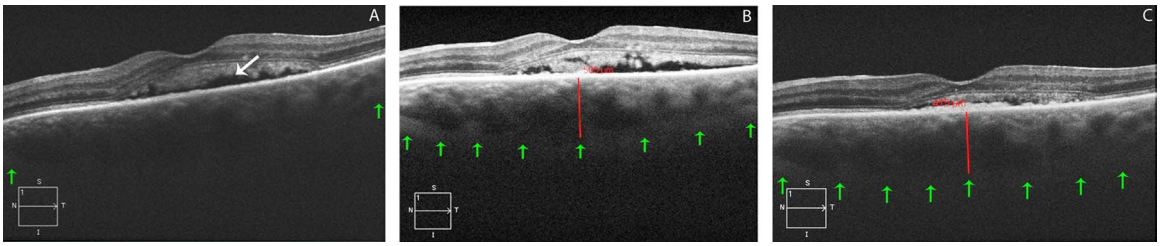

Figure 2 (A) OCT of the left eye after discontinuation of corticosteroids and before PDT showing a reduction in subretinal fluid (white arrow) and a thickened choroid that could not be measured because the choroidoscleral interface is barely visible (green arrows). (B) OCT of the left eye 1 month post-PDT showing a reduction in subretinal fluid and a thickened choroid with a visible choroidoscleral interface (green arrows) allowing choroidal thickness measurement (505 μm) (C) OCT 4 months post-PDT showing minimal subretinal fluid and a thick choroid measuring 475 μm. Green arrows depict the choroidoscleral interface. For colour figures please see the online version of the journal. ICG, indocyanine green angiography; OCT, optical coherence tomography; PDT, photodynamic therapy.

corticosteroids and observed for resolution of the visual symptoms. Patients with chronic CSCR (CSCR continuing for more than 6 months)[2] as in the present case, are treated with either laser photocoagulation or PDT.

DISCUSSION

The present report describes a case of CSCR, occurring in a 91-year-old woman associated with corticosteroid use. To our knowledge, this is the oldest reported case of new-onset CSCR in a woman.

Risk factors for CSCR include type A personality, Cushing's syndrome, pregnancy, systemic corticosteroid use, collagen vascular disease, obstructive sleep apnoea, antibiotic use, alcohol use, allergic respiratory disease, hypertension and psychosocial stressors.[3] Recent OCT studies show that patients with CSCR have an abnormally thickened choroid,[6] consistent with the theory that the basis of the disease is hyperpermeability of the choroidal vessels.

CSCR can present outside of the typical age range.[2] A recent report described a 12-year-old girl with CSCR without any known risk factors that resolved without treatment.[8] CSCR has also been reported to occur in elderly patients such as a 65-year-old woman with a history of recurrent anterior uveitis, Addison's disease and hydrocortisone use, where it resolved without treatment.[9] An additional report described a 70-year-old woman with a history of ocular hypertension where the symptoms were alleviated with oscillatory PDT.[10] The present case is the first report of a new-onset CSCR occurring in a woman in her 90s. Withdrawal of corticosteroids and one session of PDT resulted in marked subjective and anatomical improvement.

In conclusion, the present report suggests that CSCR should be given due consideration in all patients regardless of their age and gender, especially in patients with known risk factors for the disease and a clinically compatible presentation.

FINAL DIAGNOSIS

Central serous chorioretinopathy.

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Funding This work was supported in part by a Research to Prevent Blindness Unrestricted grant to the New England Eye Center/Department of Ophthalmology-Tufts University School of Medicine and the Massachusetts Lions Eye Research Fund.

Competing interests JSD receives research support from Carl Zeiss Meditech and Optovue.

Patient consent Obtained.

Ethics approval Institutional review board of Tufts Medical Center.

Provenance and peer review Not commissioned; externally peer reviewed.

To cite Gobuty M, Adhi M, Duker JS. *Br J Ophthalmol* Published Online First: [*please include* Day Month Year] doi:10.1136/bjophthalmol-2013-303707

Br J Ophthalmol 2013;**0**:1–2. doi:10.1136/bjophthalmol-2013-303707

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doi: 10.1136/bjophthalmol-2013-303707