Bilateral multifocal retinal pigment epithelium detachments associated with systemic corticosteroids

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INTRODUCTION

Central serous chorioretinopathy (CSC) is a relatively common retinal disease characterized by the accumulation of subretinal fluid at the posterior pole of the fundus, creating a circumscribed area of serous retinal detachment. It typically affects young and middle-aged men with no previous medical and family history, and no systemic symptoms or signs. However, it has been noted that CSC is associated with different conditions, characterized by exposure to increased levels of endogenous or exogenous corticosteroids. In fact, CSC has been described in patients with endogenous Cushing syndrome (1). In addition, many cases of CSC have been described during or following treatment with corticosteroids, administrated by any route, for various systemic or ocular conditions. When related to the exposure to exogenous corticosteroids, CSC presents more often with a chronic or atypical form, and is frequently bilateral (2). We report an unusual case of CSC presenting as bilateral and multifocal isolated serous retinal pigment epithelium detachments (RPEDs) following systemic corticosteroid treatment that regressed after discontinuation of the drug.

PURPOSE. To report an unusual case of central serous chorioretinopathy (CSC), presenting as bilateral and multifocal isolated serous retinal pigment epithelium detachments (RPEDs) following corticosteroid treatment.

METHODS. An otherwise healthy 39-year-old man was evaluated for visual loss following blunt trauma of his right eye (RE). The patient underwent complete bilateral ophthalmologic examination, including optical coherence tomography and fluorescein (FA) and indocyanine green angiography (ICGA).

RESULTS. At presentation, best-corrected visual acuity (BCVA) was 20/200 in the RE and 20/200 in the left eye (LE). Treatment included topical and oral corticosteroids. Three days later, the patient complained of metamorphopsia and further decrease in the VA of his RE. Fundus examination showed bilateral serous RPEDs. Optical coherence tomography, FA, and ICGA confirmed the diagnosis. Topical and oral corticosteroids were stopped and a follow-up examination 5 days later demonstrated marked resolution of the RPEDs in the RE. Five weeks later, RPEDs regressed in the RE while they persisted in the asymptomatic LE. Visual acuity in the RE further improved to 120/200. Nine months after the first visit, BCVA in the RE was 200/200. At that time, both eyes demonstrated retinal pigment epitheliopathy.

CONCLUSIONS. Central serous chorioretinopathy is a known complication of corticosteroids. The classic variant of CSC consists of a shallow neuroretinal detachment located at the posterior pole of the fundus. Bilateral and multifocal isolated serous RPEDs represent an atypical form of CSC. (Eur J Ophthalmol 2008; 18: 649-51)

KEY WORDS. Corticosteroids, Bilateral retinal pigment epithelial detachments

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METHODS

An otherwise healthy 39-year-old man was evaluated for visual loss following blunt trauma of his right eye (RE) after he had fallen down the stairs. He underwent complete bilateral ophthalmologic examination, including optical coherence tomography (OCT) and fluorescein (FA) and indocyanine green angiography (ICGA).

RESULTS

At presentation, best-corrected visual acuity (BCVA) was 20/200 in the RE and 200/200 in the left eye (LE). Slit-lamp examination of the RE revealed subconjunctival hemorrhage and traumatic iridocyclitis. Funduscopic examination revealed Berlin’s retinal edema of the posterior pole. Examination of the LE, including funduscopy, was without any abnormal findings. Treatment included topical prednisolone acetate 1% in the RE and 40 mg of oral prednisone, supposed to reverse retinal edema. Three days later the patient complained of metamorphopsia and further decrease in the VA of his RE. Fundus examination revealed three large serous RPEDs in the posterior pole. The asymptomatic LE demonstrated two smaller RPEDs. Optical coherence tomography (Fig. 1A) and FA and ICGA confirmed the diagnosis and showed one more small RPED in the RE (Fig. 2) and several more in the LE (Fig. 3). Topical and oral corticosteroids were stopped and a follow-up examination 5 days later demonstrated marked resolution of the RPED in the RE, confirmed by OCT (Fig. 1B). Five weeks later, angiography revealed regression of the RPEDs in the RE while they persisted in the LE. The OCT examination confirmed resolution of the two RPEDs located in the macula (Fig. 1C). Visual acuity in the RE further improved to 120/200. Nine months after the first visit,
BCVA in the RE was 200/200. At that time FA and ICGA showed regression of the larger RPED of the LE, while both eyes demonstrated retinal pigment epitheliopathy that was more marked in the RE.

**DISCUSSION**

There is increasing evidence implicating the use of exogenous steroids as a risk factor for the development of CSC regardless of their route of administration. As early as in 1966, Jain and Singh reported a case of maculopathy which resembled CSC in a patient receiving corticosteroid therapy for Reiter syndrome (3). Since then, a considerable number of cases of CSC have been reported during or after treatment with corticosteroids administered by various routes: oral, intravenous, intramuscular, epidural injection, inhaled, intranasal, and topical skin application (2). The time period between initiation of treatment and development of CSC ranges from a few days to several years. There have been reports of patients who developed CSC for a variety of systemic or ocular diseases. Proposed theories incriminate the effect of steroids either to the choroidal vasculature and/or to the function of the RPE (2).

Central serous chorioretinopathy is typically characterized by the accumulation of subretinal fluid at the posterior pole of the eye, causing a circumscribed serous neuroretinal detachment with or without a small underlying RPED. When the detachment spreads into the central macular area, the patient develops metamorphopsia, a relative scotoma, and micropsia. Central serous chorioretinopathy is part of a larger group of diseases, called diffuse retinal pigment epitheliopathy. One or more leaking points at the level of the RPED may undergo multiple remissions and exacerbations during lifetime so that new active CSC sites appear while older ones subside and cause RPE alterations. Such recurrences can lead to progressive enlargement of lesions and widespread RPE decompensation and atrophy.

Bilateral and atypical forms seem to be more likely associated with the use of corticosteroids. Diffuse retinal pigment epitheliopathy, acute bullous retinal detachment, and subretinal exudates have been reported to complicate the use of systemic corticosteroid treatment (2). Multiple and bilateral serous RPEDs with no associated neuroretinal detachment have been reported in association with sarcoidosis (4), neurosyphilis (5), or may be idiopathic (6). Beyrer reported two cases of serous RPEDs following contusion injury to the anterior segment of the globe; the RPEDs were unilateral in both cases and appeared in the injured eye (7). In our case, corticosteroids caused bilateral RPEDs that rapidly regressed when the corticosteroids were stopped and thus their origin cannot be directly attributed to the eye trauma.

This case report demonstrates that multiple and bilateral isolated serous RPEDs represent a variant of CSC associated with systemic corticosteroid treatment. Furthermore, dilated funduscopic examination of both eyes should be recommended on a regular basis for these patients, as they may be asymptomatic.

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