

# Central serous chorioretinopathy in uveitis patients under corticosteroid therapy

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**Background:** The etiology of central serous chorioretinopathy (CSCR) remains uncertain despite the numerous studies which have been published since 1948. The recent systematic review and meta-analysis have revealed the following CCRS risk factors: systemic hypertension, steroid usage, sleeping disturbance, autoimmune disease, psychopharmacologic medication use, and type-A behavior (Liu B, et al. *Retina*. 2016). In numerous studies the oral corticosteroids usage was found to be the CCSR risk factor. Nevertheless, reports on CCSR in uveitis patients administered corticosteroid therapy is limited.

The aim of this study is to report cases of CCSR among uveitis patients.

**Patients & Methods:** A retrospective chart review of uveitis patients seen from 1995 to 2015 at the Centre for Ophthalmic Specialised Care, Lausanne, Switzerland. The ophthalmic and systemic features are presented. Patients underwent the complete routine work-up applied to patients with uveitis, comprising, in addition to routine features such as Snellen visual acuity, slit-lamp examination, applanation tonometry, and fundoscopy, laser flare photometry (LFP), computerized visual field (VF) testing, optical coherence tomography (OCT) including EDI-OCT, and dual fluorescein angiography (FA) and indocyanine green angiography (ICGA).

**Results:** Out of 1793 uveitis patients followed at the Centre for Ophthalmic Specialised Care, 6 patients (0.3%) developed CCSR under corticosteroid therapy. The mean age of patients was  $40 \pm 13.4$  years; disease incidence was not associated with gender (3 men; 3 women). 3 patients had idiopathic uveitis, 1 patient - birdshot retinchoroiditis, 1 patient - presumed tuberculosis, and 1 patient - Vogt-Koyanagi-Harada disease. The mean duration of corticosteroid therapy before CCSR occurred was  $4.95 \pm 4.0$  months. At the moment of CCSR, the mean BCVA was  $0.6 \pm 0.26$ ; after the discontinuation of corticosteroids, at the last follow-up the mean BCVA was  $0.73 \pm 0.3$ . The mean LFP was  $44.7 \pm 91.4$  ph/ms showing a mild inflammation when CCSR occurred. The mean delay in diagnosis of CCSR was  $6.8 \pm 1.5$  months. Demographics and clinical data are presented in Table 1.

Patient	Gender	Age	Diagnosis	Duration of corticosteroid treatment	BCVA at the moment of CCSR	BCVA at the last follow-up
1	M	43	BRC	10 weeks	0,4	0,7
2	F	35	VKH presumed	2 months	0,9	0,7*
3	M	47	TB	5 weeks	0,6	1
4	M	57	idiopathic	6 months	0,7	1
5	F	41	idiopathic	6 months	0,2	0,2**
6	F	17	idiopathic	12 months	0,8	0,8

Table 1. Demographics and clinical data

BCVA – best corrected visual acuity. BRC – birdshot retinchoroiditis.

VKH – Vogt-Koyanagi-Harada disease. TB – tuberculosis.

\* - due to choroidal neovascularization successfully treated with intravitreal aflibercept injection. \*\* - due to cataract.

## Case 1 :

A 47-year-old Caucasian male presented with photophobia and vision loss on the right eye. BCVA was 0.2 for the right eye, while BCVA was 0.8 on the left eye. LFP revealed anterior chamber inflammation (RE: 49.4 ph/ms, LE: 21.2 ph/ms). Vitritis, snowballs, retinal scars, and cystoid macular edema were observed on the right eye. Mantoux – test showed a hyper-reaction with skin papilla's size 12\*11 mm. The diagnosis of presumed tuberculous-related uveitis was done. After administration of triple anti-TB therapy and oral prednisone 30 mg, BCVA improved to 1.0 in both eyes in 10 days. Nevertheless, the anterior chamber inflammation did not change (LFP RE - 52.6 ph/ms, LE - 18.2 ph/ms). It was decided to taper prednisone slowly. Afterwards, in 5 weeks, patient presented with vision loss on the right eye: BCVA decreased to 0.6, while anterior chamber inflammation was quite low (LFP RE 18.1 ph/ms, LE 9.3 ph/ms) under prednisone 20 mg. The condition was considered as a recurrence of inflammatory process and dosage of prednisone was raised up to 50 mg. 5 days later, the patient's condition did not change but laser flare photometry decreased slightly (16.0 ph/ms on the right eye). Central serous chorioretinopathy was diagnosed and steroid treatment was discontinued. Representative graph on the change of BCVA, LFP, and dosage of corticosteroids is presented (Figure 1).

6 months later, on the last follow-up, no signs of active inflammation were seen, BCVA was 1.0 on the right eye, and LFP remained stable – 21.7 ph/ms.

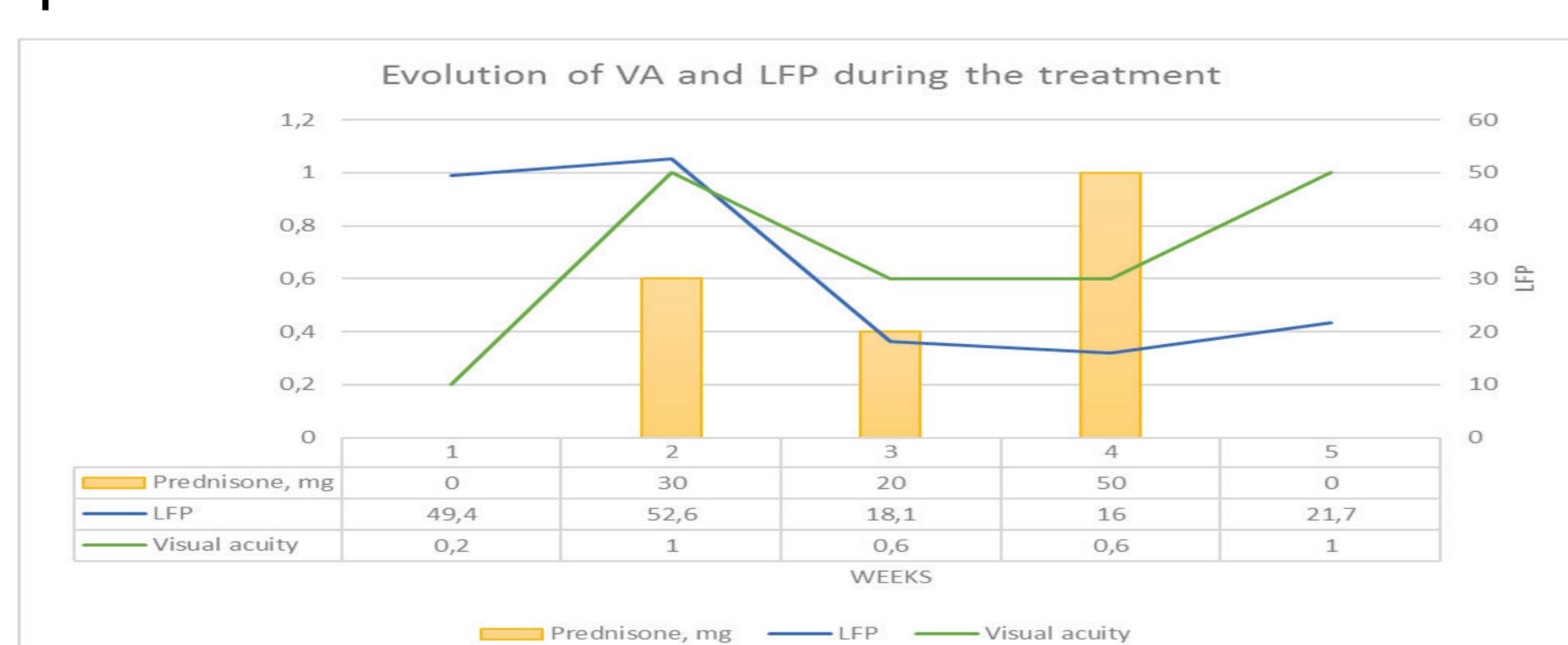


Figure 1. Graph showing the change in BCVA, LFP, and prednisone dosage during the treatment

## Case 2:

A 43-year-old Caucasian male presented with birdshot retinchoroiditis. For 2 years, the patient's condition remained relatively stable without treatment, BCVA was 1.0 for both eyes, laser flare photometry detected a subclinical anterior chamber inflammation in the right eye (5.2 ph/ms, with normal value of 3-4 ph/ms). Both eyes had a slight vitritis and their fundus showed scattered, punched-out creamy lesions. At the moment of corticosteroid and immunosuppressive therapy administration, the BCVA of the right eye was 0.8, the anterior chamber inflammation became more evident (laser flare photometry - 15.1 ph/ms OD and 5.1 ph/ms OS), and visual fields deteriorated in both eyes. After 10 weeks of systemic treatment (oral prednisone (1 mg/kg) and cyclosporine A (5 mg/kg), the BCVA on the right eye was 1.0 and LFP decreased to 5.2 ph/ms. However, the patient presented with vision loss on the left eye (BCVA 0.4) due to central serous chorioretinopathy. FA revealed hyperfluorescent leaking point and ICGA showed a focal brightly hyperfluorescent spot that corresponded to the leakage area on FA (Figure 2). Therefore, prednisone was progressively tapered. Consequently, BCVA on the left eye improved to 0.7.

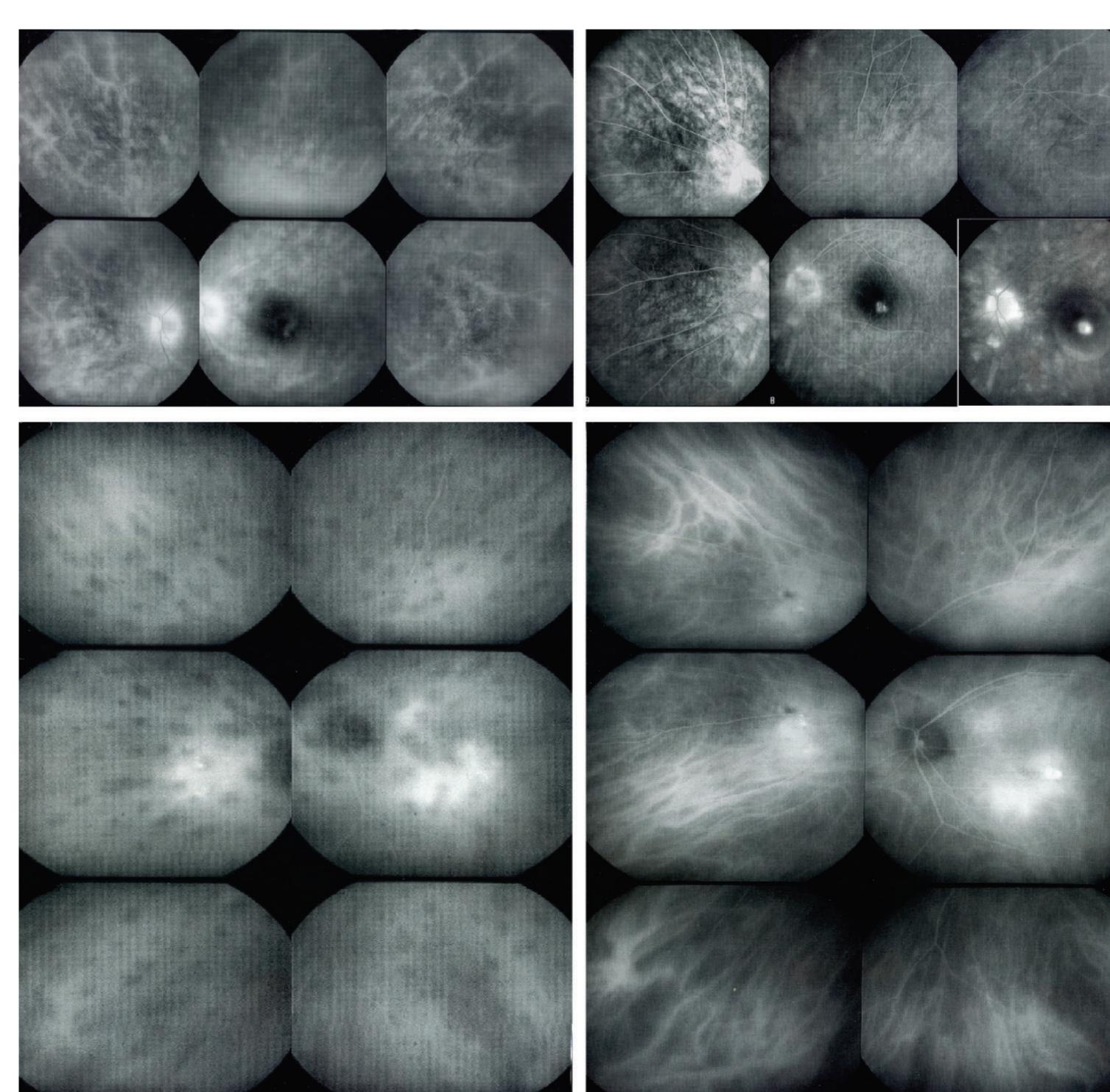


Figure 2. Fluorescein (top) and indocyanine green (bottom) angiograms at the moment of BRC onset (left) and at the moment of CCSR (right)

**Conclusion:** Central serous chorioretinopathy should be suspected when the vision deterioration occurs in uveitis patients administered corticosteroid therapy with no signs of active inflammation. This complication is extremely rare but serious condition which needs a prompt tapering and discontinuing of corticosteroids.