

Case Report

# Significant Visual Impairment after Short-Lasting Central Serous Chorioretinopathy

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## Keywords

Central serous chorioretinopathy · Fundus autofluorescence · Spectral-domain optical coherence tomography · Subretinal fluid

## Abstract

A 37-year-old man experienced two episodes of central serous chorioretinopathy (CSCR) with the onset within a 7-month period, one in each eye. The diagnosis was made based on spectral-domain optical coherence tomography (SD-OCT), fundus autofluorescence (FAF), and fluorescein angiography. The presence of subretinal neovascularization and polypoidal choroidal vasculopathy were excluded. Each CSCR episode lasted for approximately 6 months and resolved completely after laser photocoagulation (left eye) and photodynamic therapy (right eye). In the right eye, subthreshold micropulse laser treatment and oral eplerenone were initially administered because of a verteporfin shortage, but they were not effective. Final best-corrected visual acuity was 0.8 logMAR in the left eye and “counting fingers” in the right. SD-OCT revealed significant retinal thinning in both eyes despite FAF, showing no major loss of retinal pigment epithelial cells. A significant reduction of ganglion cell complex thickness occurred in the right eye. Acute CSCR can result in significant visual impairment, even when short-lasting.

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## Introduction

Central serous chorioretinopathy (CSCR) is common, but its etiopathogenesis and treatment remain subjects of intensive research. The disease usually is self-limiting – approximately 84% of cases resolve spontaneously within a few months without treatment [1]. The remaining cases become chronic and progress to visual impairment of various degrees. This typically occurs over long time periods [2, 3]. Acute CSCR is believed to be benign and not lead to vision deterioration; however, this is not always the case [4, 5]. We present a patient who developed severe bilateral visual impairment after an episode of CSCR.

## Case Presentation

A healthy 37-year-old man was diagnosed with acute CSCR in the right eye (RE) and referred for subthreshold micropulse laser (SML) treatment. A previous episode of CSCR had affected the left eye (LE). The first episode lasted for 6 months and resolved after laser photocoagulation. However, his final best-corrected visual acuity (BCVA) was 0.8 logMAR (0.16 on the Snellen chart); significant retinal thinning was noted on spectral-domain optical coherence tomography (SD-OCT), with a central subfoveal thickness (CST) of 142  $\mu\text{m}$ . At the time, the CSCR resolved in the LE, and SD-OCT of the RE did not show any deficit in retinal morphology: CST and mean ganglion cell complex (GCC) thickness, which is defined as the thickness of the ganglion cell and inner plexiform layers, were normal (282  $\mu\text{m}$  and 82  $\mu\text{m}$ , respectively).

The patient was referred after a 1-month history of decreased RE vision because of the significant decline in BCVA after the first CSCR episode. The referring ophthalmologist recommended prompt SML treatment. Baseline examination at our center included measurement of refraction (automated refractor Huvitz China 2020), evaluation of BCVA, on the Snellen chart, fundus autofluorescence (FAF) performed with the use of VISUCAM 524 (Carl Zeiss Meditec AG 2019), SD-OCT, and angio-OCT (Revo Optopol 2019). SD-OCT showed a large amount of subretinal fluid (SRF) in the RE with elongation of photoreceptors. Optical coherence tomography angiography showed no signs of subretinal neovascularization. A detailed medical history did not reveal any systemic disorder, and the patient was not taking any medications. He denied any excessive stress before the onset of symptoms. Intraocular pressure measurements were normal at each visit.

SML treatment was performed using a 810-nm laser according to the panmacular protocol recommended by the International Retinal Laser Society LIGHT [6], which resulted in a significant reduction of SRF after the first treatment session. Oral eplerenone 50 mg daily was also initiated. Follow-up examination revealed persistent SRF and no gain in vision. Therefore, another SML treatment was performed; however, it did not result in any SRF reduction or improvement in BCVA. Indocyanine green angiography (ICGA) was performed at that time to rule out polypoidal choroidal vasculopathy, but it only showed findings typical for CSCR (hyperpermeability of choriocapillaris) not polyps or subretinal neovascularization. This patient was managed during the verteporfin shortage, so photodynamic therapy was initially not available for use. However, after 5 months of follow-up, during which time his BCVA decreased to “counting fingers,” we were able to procure a vial of verteporfin to perform half-dose photodynamic therapy based on his ICGA findings. After this procedure, the SRF resorbed; however, significant retinal thinning was present on SD-OCT, and final BCVA remained at “counting fingers.”

SD-OCT of the RE revealed the damage was most significant in the GCC (mean thickness of 53  $\mu\text{m}$ ). GCC thickness was measured within the elliptic ring with outer dimensions of 4.8  $\times$  4.2 mm and inner dimensions of 1.2  $\times$  1.0 mm. The RE findings were significantly worse than those in the left. Mean thickness of the outer retinal layers in the RE within the central 1 mm was 89  $\mu\text{m}$

and myoid zone/ellipsoid zone boundary-to-retinal-pigment-epithelium (RPE) distance was 67  $\mu\text{m}$ . Although these two values were lower than normal, they were no different from the left-eye measurements. A summary of RE examination findings and treatment is shown in Table 1. Fluctuations in SRF after SML and photodynamic therapy are shown in Figure 1.

The patient's LE maintained stable visual parameters throughout the follow-up period. BCVA stayed at 0.8 logMAR and has not improved. Fundus ophthalmoscopy revealed minor anomalies of the RPE, including depigmentation of the central macula, which in general evolved toward singular points of hyperpigmentation after a few months of observation. At the first visit, FAF showed anomalous autofluorescence in the posterior pole with foci of hyperautofluorescence and a spot of hypoautofluorescence at the supratemporal vascular arcade. During 7 months of follow-up, some of the hyperautofluorescent foci transformed into spots of pigment clumps presenting as spots of hypoautofluorescence. SD-OCT at follow-up visits showed significant retinal thinning within the central 1 mm<sup>2</sup>, ranging from 147 to 152  $\mu\text{m}$ . Final GCC layer thickness stayed within normal range (75  $\mu\text{m}$ ). Similar to the RE measurements, the outer retinal-layer thickness within the central 1-mm circle was reduced to 88  $\mu\text{m}$  and myoid zone/ellipsoid zone boundary-to-RPE-layer distance was 66  $\mu\text{m}$ . Subfoveal choroidal thickness ranged from 580 to 600  $\mu\text{m}$ .

Images of the fundus (fluorescein angiography – FA, ICGA, and FAF) are presented in Figure 2, and final GCC measurements of both eyes are presented in Figure 3. After 3 months of unsuccessful treatment and deterioration in BCVA, the patient was referred for a neurological consultation and magnetic resonance imaging of the brain. Neither revealed central nervous system pathology.

## Discussion/Conclusion

The case presented here shows that even short-lasting CSCR with rather minor disturbances of the RPE on FAF may result in significant retinal thinning and visual impairment. Our patient experienced significant loss of retinal ganglion cells within 6 months of disease onset, which resulted in a dramatic decrease in visual acuity.

Generally, major visual impairment is reported in patients with CSCR after a long duration of disease; however, a linear correlation has not been shown [3, 7]. Therefore, significant damage to the retina may occur within the first few months [4, 5]. To the best of our knowledge, the profound decrease in visual acuity that our patient experienced after short-lasting CSCR without subretinal neovascularization has not been previously reported.

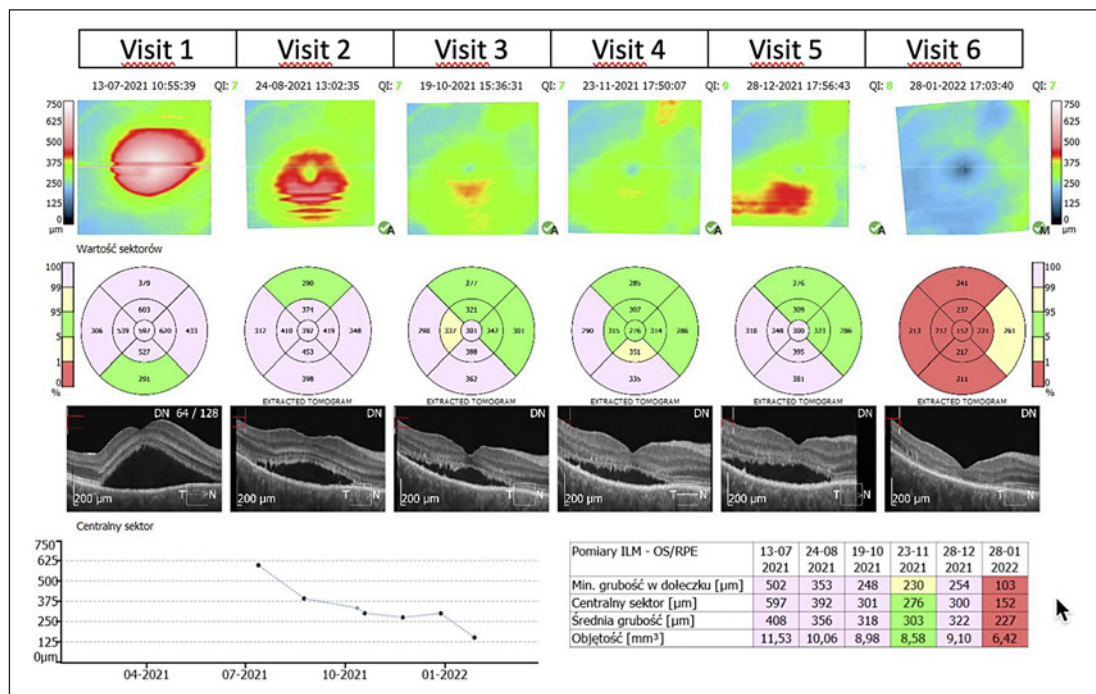
Looking closely at retinal and choroidal morphology in the course of CSCR may provide information on the potential damage caused. Currently, it is accepted that the origin of pathology in CSCR is thickened choroid [8, 9]. Dilation of choroidal large vessels results in pressure on the choriocapillaris. This causes choriocapillaris atrophy, changes in the RPE, and impaired nourishment of the outer retina [3, 10, 11]. Additionally, photoreceptors separated from the RPE by the SRF lack normal metabolism in the outer segments and are prone to atrophy as a result [12].

These mechanisms are reflected in CSCR studies by Hata et al. [5] and Ersoz et al. [13], who report early damage to the outer nuclear layer. In the patient described in our case report, significant thinning of the outer retinal layers was noted; however, the GCC was damaged as well. This phenomenon has rarely been analyzed. In a retrospective study of 30 patients with acute CSCR, Nam and Kim [14] reported only a transient thinning of the GCC that returned to normal after resolution of SRF accumulation. Demirok et al. [15] reported GCC thinning in both acute and chronic CSCR. Therefore, significant visual loss from CSCR can be explained by both cell loss in the GCC and alterations in the outer retinal layers.

**Table 1.** Results of diagnostic and therapeutic procedures at all follow-up visits of the RE.

Exam/procedure	Visit 1 (one month after the onset)	Visit 2 (1.5 months after visit 1)	Visit 3 (3 months after visit 1)	Visit 4 (4.5 months after visit 1)	Visit 5 (5 months after visit 1)	Visit 6 (6 months after visit 1)
BCVA (logMAR)	0.5	1.0	1.0	1.0	2.3	2.3
SD-OCT, $\mu\text{m}$	Elongation of POS CST: 625 SRF 421 701	Elongation of POS and granularities CST: 399 SRF 223 540	More granularities CST 307 SRF 131 520	Like visit 3 CST 282 SRF 110 532	Like visit 3 and 4 CST 316 SRF 145 540	Retinal thinning. CST: 151 SRF level : 0 400
Fundus examination (CF)	Oval in shape SRF with retinal edema. No pigmentary abnormalities.	Moderate retinal edema, fine yellow granulations at the site of edema	Smaller oval edema, more yellow granulations	Increasing number of yellow deposits.	Like visit 4	Like visit 4 and 5
FAF	Regular area of hyperautofluorescence corresponding to the area of SRF and retinal edema	Oval shaped moderate hyperfluorescence corresponding to the SRF presence, several fine foci of intensive hyperfluorescence corresponding to yellow granulations on CF	Numerous fine foci of hyperfluorescence corresponding to yellow deposits on CF	More foci of hyperfluorescence corresponding to yellow deposits	Like visit 4	Like visit 4 and 5
ICGA		Placoid areas of hyperfluorescence in mid-phase of the angiogram, no hot spot				
SLM	810 nm, panmacular 450 exp. 1.7 W, 0.3 s, spot diameter 500 $\mu\text{m}$		810 nm 380 exp 1.7 W, 0.3 s, spot diameter 500 $\mu\text{m}$			
PDT					hdPDT 3 mg/ $\text{m}^2$	
Eplerenone		50 mg	50 mg	50 mg	50 mg	50 mg

BCVA, best-corrected visual acuity; SD-OCT, spectral-domain optical coherence tomography; SRF, subretinal fluid; POS, photoreceptor outer segments; SFCT, subfoveal choroidal thickness; CST, central subfoveal thickness; CF, color photo; FAF, fundus autofluorescence; ICGA, indocyanine green angiography; SML, subthreshold micropulse laser; hdPDT, half-dose photodynamic therapy.



**Fig. 1.** Changes in retinal morphology in the course of CSCR in the RE.

Loss of thickness in the GCC has also been reported in neurodegenerative diseases, such as multiple sclerosis; however, our patient had no evidence of a neurologic disease [16]. Moreover, the SD-OCT measurements in the RE (CST and GCC) before the occurrence of SRF were normal. Therefore, we believe that the thinning of the GCC in our patient can be attributed solely to CSCR.

In conclusion, our patient represents an unusual presentation of acute CSCR with poor visual outcome and illustrates that the disease can cause early and irreversible visual loss even when short-lasting. Further research is warranted to determine the retinal-layer disturbances measured by SD-OCT.

### Statement of Ethics

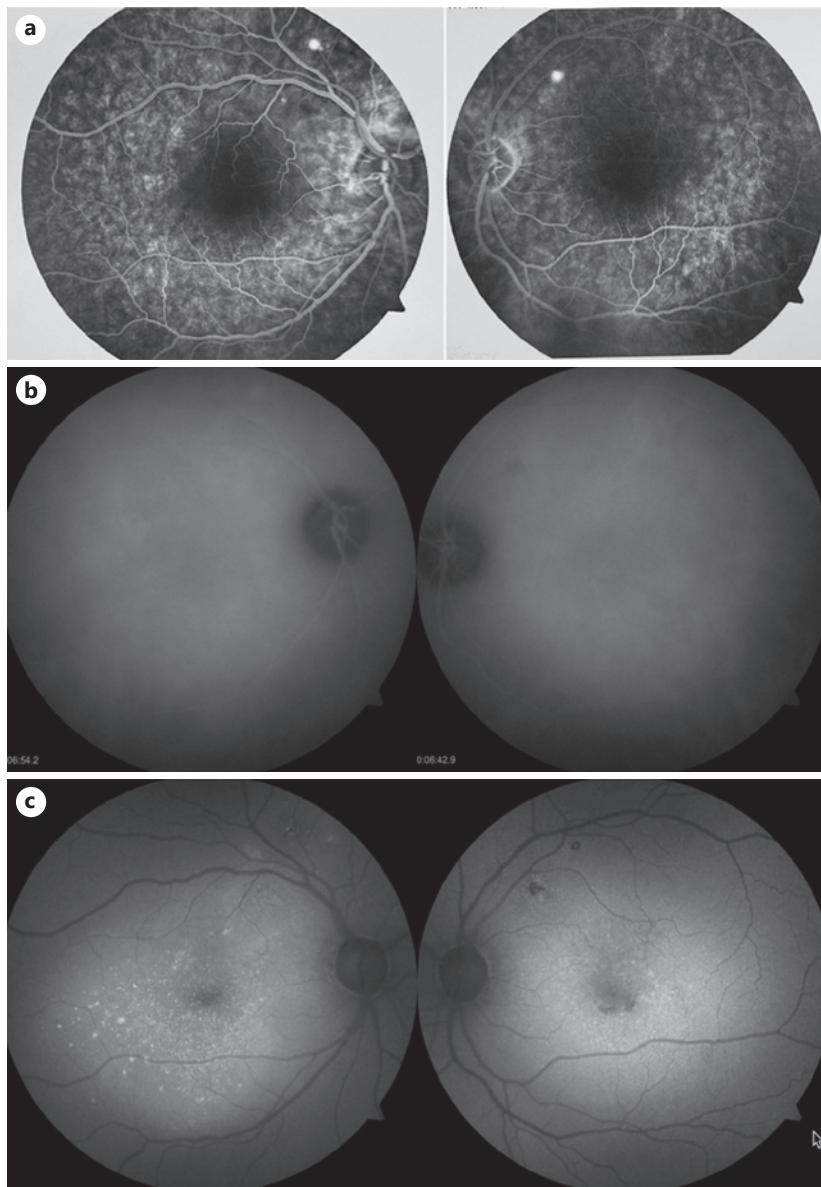
Ethical approval is not required for this study in accordance with local or national guidelines. A written informed consent was obtained from the participant of the study for publication of the details of their medical case and accompanying images.

### Conflict of Interest Statement

The authors have no conflicts of interest to declare.

### Funding Sources

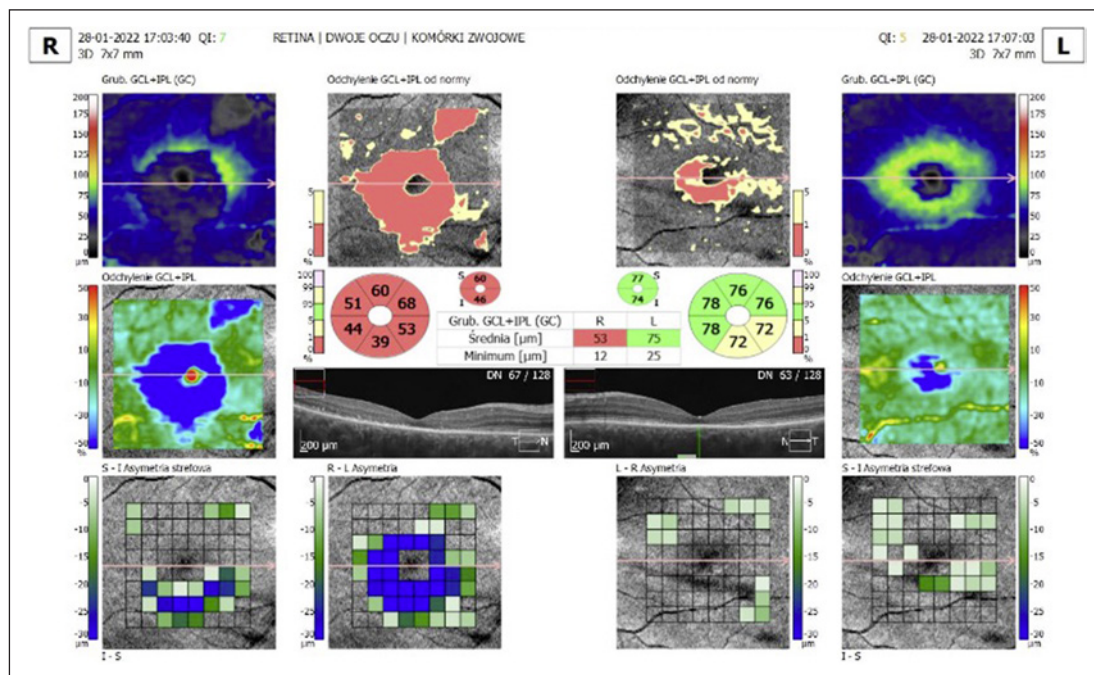
The study has not received any funding.



**Fig. 2.** Fundus imaging at different timepoints of the follow-up. **a** Images of fluorescein angiography of the active phases of the first (LE) and second (RE) episode of CSCR. A single leakage point outside the fovea is observed during both episodes. **b** ICGA images obtained during second episode of CSCR in the RE. Middle phase images show placoid areas of hyperfluorescence in both eyes corresponding to areas of choriocapillaris hyperpermeability. **c** Final FAF of the right (RE) and left eye (LE). Numerous spots of hyperfluorescence are noted in the macular area of RE, while paracentral spots of hypofluorescence can be observed in LE.

### Author Contributions

Maciej Gawęcki – writing, methodology, data collection, research, and references. Andrzej Grzybowski – analysis of the data and reviewing of the manuscript, final approval. Monika Pompein-Batkiewicz – data collection, research, providing references, final approval.



**Fig. 3.** Ganglion cell complex (GCC) thickness of the right (RE) and left eye (LE) at the end of the follow-up. Significant thinning is observed in the RE with normal GCC values in the LE.

### Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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