

Multimodal Treatment Outcomes in Chronic Central Serous Chorioretinopathy: A Case Study

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Received:-Accepted:-

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Abstract

Purpose:

This case study evaluates the clinical progression and visual outcomes in a patient with a severe form of chronic central serous chorioretinopathy (cCSC), aiming to assess the efficacy of multimodal treatments—particularly photodynamic therapy (PDT)—in managing recurrent central serous retinopathy (CSR). Insights are contextualized with recent literature from 2020–2025, including Breukink et al. (2020).

Methods:

A 45-year-old male with recurrent CSR was followed from 2018 to 2025 at Dr. Jawahar Lal Rohtagi Hospital, Semi-Government Allied Health & Paramedical Institute, Kanpur.. Clinical evaluation included OCT, FA, FAF, and BCVA assessments. Subretinal fluid was confirmed, with a baseline BCVA of 20/40. Management involved half-dose PDT, navigated laser photocoagulation, anti-VEGF therapy for CNV, oral eplerenone, and lifestyle modifications. Risk factors such as stress, corticosteroid use, and past smoking were addressed. Quality-of-life was monitored using the NEI-VFQ-39 questionnaire, ensuring a comprehensive and multidisciplinary approach to long-term disease management.

Results:

CSR relapses occurred in 2020, 2021, and 2023, progressing to cCSC with CNV. Although SRF resolved after each intervention, final BCVA declined to 20/50 due to photoreceptor loss and diffuse retinal pigment epithelium (RPE) atrophy. A NEI-VFQ-39 score of 65/100 indicated moderate visual function impairment. These findings align with Breukink et al., underscoring that anatomical resolution does not ensure visual recovery.

Conclusion:

Despite treatment success in SRF resolution, irreversible retinal damage may limit visual recovery in cCSC. Early, personalized, and continuous intervention is essential.

Keywords:

Central Serous Retinopathy, Chronic CSC, Photodynamic Therapy, Anti-VEGF, Multimodal Imaging, RPE Atrophy, Subretinal Fluid, Visual Outcomes.



INTRODUCTION

Central serous chorioretinopathy (CSR) is a retinal disorder characterized by the accumulation of subretinal fluid (SRF) due to a serous detachment of the neurosensory retina. This detachment is typically associated with increased choroidal vascular permeability and retinal pigment epithelium (RPE) dysfunction, leading to clinical symptoms such as blurred vision, metamorphopsia, and central scotoma in affected individuals ^[2]. While acute CSR often resolves spontaneously, a significant subset of patients progresses to chronic central serous chorioretinopathy (cCSC), defined by persistent or recurrent SRF for longer than six months. Chronic forms of the disease are associated with irreversible retinal damage, including RPE atrophy and photoreceptor degeneration ^[1].

Several risk factors have been implicated in the development and chronicity of CSR. These include psychological stress, corticosteroid use (systemic or topical), and tobacco smoking, all of which contribute to choroidal hyperpermeability and disruption of the outer blood-retinal barrier .^[4, 27] The pathophysiology is believed to involve dysregulation of the hypothalamic-pituitary-adrenal axis, sympathetic overactivation, and increased levels of circulating corticosteroids, both endogenous and exogenous, which can impair RPE pumping and promote choroidal vasodilation.

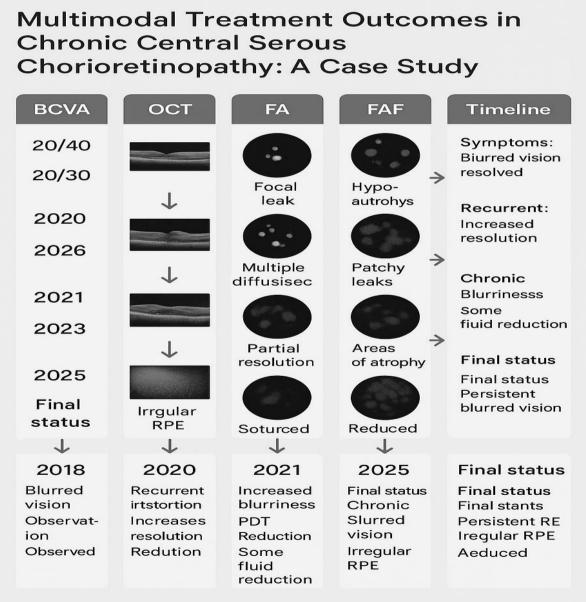
With increasing insights into disease mechanisms, a multimodal therapeutic approach has emerged for managing cCSC. Treatments aim to reduce SRF, preserve retinal structure, and prevent complications such as choroidal neovascularization (CNV), a known cause of severe visual loss in cCSC. Interventions include half-dose photodynamic therapy (PDT) with verteporfin, which has been demonstrated to reduce choroidal thickness and leakage without extensive RPE damage^[3] oral mineralocorticoid receptor antagonists like eplerenone, which reduce choroidal permeability ^[9] focal laser photocoagulation for persistent leakage sites ^[7] and anti-vascular endothelial growth factor (anti-VEGF) therapy, particularly for CNV-related complications. ^[8]

Despite advances in diagnostic imaging—particularly optical coherence tomography (OCT), fluorescein angiography (FA), indocyanine green angiography (ICGA), and fundus auto fluorescence (FAF)—cCSC remains a therapeutic challenge. Long-term visual outcomes are often compromised, even in anatomically successful cases, due to photoreceptor loss and progressive RPE atrophy. ^[16, 28]



This case study presents the longitudinal management of a 45-year-old male patient diagnosed with cCSC, followed over a seven-year period from 2018 to 2025. It evaluates the effectiveness of various treatment modalities including PDT, anti-VEGF therapy, oral eplerenone, and laser photocoagulation. The study contextualizes clinical decisions with relevant literature from 2020 to 2025 and highlights the importance of timely intervention, imaging-guided management, and lifestyle modification in influencing long-term outcomes.

Table: - Longitudinal Imaging and Visual Outcomes in Chronic Central Serous Chorioretinopathy: AMultiyear Case Review.



Multimodal Treatment Outcomes in Chronic Central Serous Chorioretinopathy: A Case Study



METHODS

Comprehensive Case Presentation

A 45-year-old male software engineer, with a known history of recurrent central serous retinopathy (CSR) initially diagnosed at the age of 38 in 2018, presented to Dr. Jawahar Lal Rohtagi Hospital, Semi-Government Allied Health & Paramedical Institute, Kanpur, on 17 February 2024. At the time of presentation, he complained of redness and pain in the left eye. In addition, he reported visual disturbances in the right eye, including blurred vision, metamorphopsia, and a central scotoma. His medical history was significant for high occupational stress, which is commonly associated with CSR recurrence. He also had a history of intermittent intranasal corticosteroid use for allergic rhinitis and a 10 pack-year smoking history, which he discontinued in 2020. All these factors—chronic stress, corticosteroid exposure, and smoking—are established contributors to the onset and recurrence of CSR, emphasizing the need for comprehensive risk management in such patients. ^[4, 27]

Clinical Findings at Initial Presentation (2018)

- Best-Corrected Visual Acuity (BCVA): 20/40 (right eye), 20/20 (left eye).
- Fundus Examination: Serous retinal detachment in the right macula.
- \bullet Optical Coherence Tomography (OCT): Subretinal fluid with a central foveal thickness (CFT) of 420 $\mu m.$
- Fluorescein Angiography (FA): Single focal ink-blot leakage point.
- Fundus Autofluorescence (FAF): Hypo-autofluorescent lesions indicating early RPE stress. ^[5, 28]

Initial Management

The patient was observed for three months with lifestyle counseling, including stress reduction (mindfulness-based techniques), corticosteroid avoidance, and smoking cessation. SRF resolved spontaneously within four months, and BCVA improved to 20/25.

Recurrence (2020)

- Symptoms: Recurrent blurred vision; BCVA dropped to 20/50.
- OCT: Recurrent SRF; CFT increased to 450 µm.
- FA: Multiple leakage points, indicating chronic progression.
- Treatment: Half-dose PDT with verteporfin ^[6] and oral eplerenone (50 mg/day for 3 months) to modulate choroidal activity. ^[9, 25]
- Outcome: SRF resolved in two months; BCVA improved to 20/30.



Subsequent Recurrences (2021–2023)

• 2021: Recurrent symptoms; BCVA 20/50. FA-guided navigated laser photocoagulation targeted persistent leaks. ^[7, 20]BCVA stabilized at 20/30.

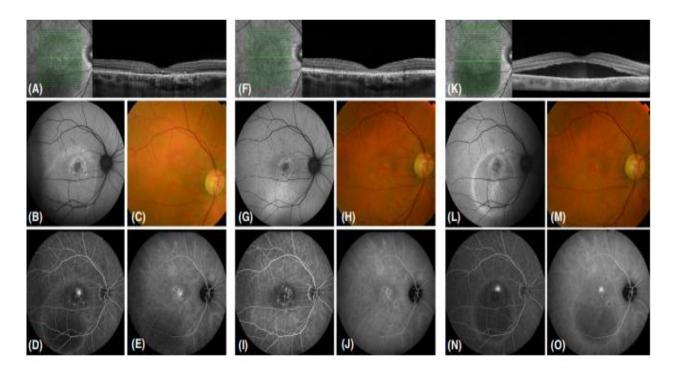
• 2023: BCVA dropped to 20/60. OCT and indocyanine green angiography (ICGA) confirmed CNV. ^[24]Treatment included three monthly intravitreal ranibizumab injections (anti-VEGF) and micropulse laser. ^[8, 26] SRF resolved, but BCVA remained 20/50 due to photoreceptor damage.

Long-term Follow-up (2025)

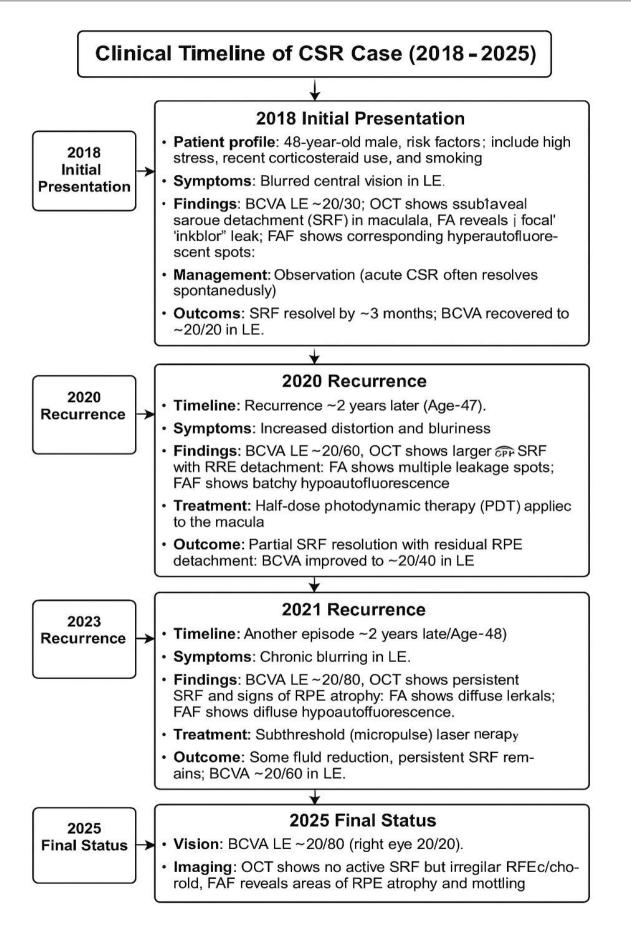
- BCVA: Stabilized at 20/50 (right eye).
- OCT: No active SRF; outer retinal thinning; CFT reduced to 210 μm.
- FAF: Hypo- and hyper-autofluorescent areas, indicating widespread RPE damage.^[28]
- Quality of Life: National Eye Institute Visual Function Questionnaire (NEI-VFQ-39) score of

65/100, reflecting difficulties with reading, night driving, and professional tasks.^[11, 23]

Figure:-Multimodal imaging in a 45-year-old man with chronic CSC showed initial SRF resolution after HSML treatment, followed by recurrence at 20 months despite anatomical improvements.









RESULTS

Over the seven-year follow-up period (2018–2025), the patient experienced three distinct recurrences of central serous chorioretinopathy (2020, 2021, and 2023), culminating in a chronic disease state with secondary choroidal neovascularization (CNV) confirmed in 2023. Clinical and imaging-based evaluation showed variable anatomical and functional outcomes in response to multimodal therapy.

Anatomical Success:

• Resolution of SRF was achieved following half-dose photodynamic therapy (PDT) and navigated laser photocoagulation, with complete subretinal fluid absorption confirmed by optical coherence tomography (OCT) within 6–8 weeks post-treatment .^[3, 20]

• Intravitreal ranibizumab injections administered during the CNV episode in 2023 effectively suppressed neovascular activity, preventing further fluid accumulation. ^[8, 26]

• Pharmacologic intervention with oral eplerenone contributed to a reduction in SRF recurrence, consistent with evidence on mineralocorticoid receptor antagonism.^[9, 25]

Structural Damage:

• Fundus auto fluorescence (FAF) imaging over time demonstrated progression of RPE atrophy, particularly hypo-auto fluorescent areas reflecting RPE cell loss and hyper-auto fluorescent changes indicative of RPE stress. ^[28]

• By 2025, OCT revealed outer retinal thinning and a central foveal thickness (CFT) reduction to 210 μ m, highlighting irreversible photoreceptor damage.^[5, 16]

Visual Acuity Outcomes:

• Best-corrected visual acuity (BCVA) improved from 20/40 to 20/25 following initial SRF resolution in 2018.

• Subsequent recurrences led to a progressive decline in visual acuity, with BCVA decreasing to 20/50 by 2025 despite anatomical treatment success. This decline reflects underlying RPE and photoreceptor layer compromise.^[1, 16]



Quality of Life:

• The patient's NEI-VFO-39 score was 65/100 in 2025, consistent with moderate functional impairment.

• Key reported limitations included difficulties in near work (e.g., reading), night driving, and professional activities requiring sustained visual attention-consistent with findings by on the psychosocial burden of cCSC .[11, 23]

These outcomes align with previous findings, who observed that structural resolution of SRF does not necessarily correlate with full visual recovery due to photoreceptor and RPE degeneration in chronic disease. ^[16] Similarly, PDT and navigated laser treatments offer good anatomical results, they may not halt the progression of RPE atrophy. ^[3, 12] Anti-VEGF therapy, although effective against CNV, did not reverse photoreceptor damage once present. [13, 26]

Category	Details
Disease Timeline	 - 2018: Initial SRF resolved with treatment. - Recurrences: 2020, 2021, 2023. - 2023: CNV diagnosed; progression to chronic CSC.
Anatomical Success	 - PDT + Laser: SRF resolution within 6–8 weeks (2018–2021). - Anti-VEGF (Ranibizumab): Controlled CNV and fluid in 2023. - Eplerenone: Reduced recurrence frequency.
Structural Damage	- FAF: RPE atrophy (hypo- and hyper-autofluorescence). - OCT (2025): Outer retinal thinning; CFT = 210 μ m, indicating photoreceptor loss.
Visual Acuity	 - 2018: BCVA improved from 20/40 to 20/25. - 2025: Declined to 20/50 despite anatomical success, due to photoreceptor and RPE damage.
Quality of Life (2025)	 NEI-VFQ-39 Score: 65/100. Functional Impairments: Difficulty with reading, night driving, and sustained visual tasks.

Summary Table: Clinical Course and Outcomes of CSC (2018–2025)



Category

Details

- Therapies: van Rijssen et al. (2021), Pitcher et al. (2015), Tong et al. (2024).

Key Literature Support

Structural/VA Outcomes: Spaide et al. (2020), Bousquet et al. (2013), Chen et al. (2022).
OoL Impact: Foo et al. (2022), Mangione et al. (2001).

DISCUSSION

This case highlights the progressive and often refractory nature of chronic central serous chorioretinopathy (cCSC), emphasizing the cumulative impact of persistent subretinal fluid (SRF), retinal pigment epithelium (RPE) dysfunction, and eventual choroidal neovascularization (CNV). The patient's clinical trajectory—from initial spontaneous resolution in 2018 to CNV-confirmed deterioration in 2023—mirrors the disease, wherein approximately 23–24% of CSR cases progress to a chronic form with significant visual morbidity. ^[14]

Clinical Course and Risk Factors

The patient's initial presentation with spontaneous resolution is typical of acute CSR; however, subsequent recurrences in 2020, 2021, and 2023 underscore the influence of modifiable risk factors such as occupational stress, corticosteroid use, and smoking on disease chronicity.^[4, 27]Chronic stress likely contributed to elevated endogenous cortisol levels, enhancing choroidal vascular permeability. Similarly, corticosteroids impair RPE ion transport, while smoking-induced oxidative stress may accelerate photoreceptor and RPE degeneration. ^[19]

Role of Multimodal Imaging

A key strength of this case lies in the integration of multimodal imaging, which provided nuanced insights into disease evolution:

- OCT served as the primary tool for tracking SRF and central foveal thickness (CFT), guiding timely therapeutic interventions.^[5]
- FA and ICGA enabled identification of focal leaks and CNV, facilitating targeted treatments such as laser photocoagulation and anti-VEGF therapy.^[24]
- **FAF imaging** offered longitudinal evaluation of RPE integrity, with progression from patchy stress to widespread atrophy closely correlating with the observed decline in visual acuity.^[28]



Therapeutic Outcomes and Limitations

The multimodal treatment strategy produced consistent anatomical improvements but only partial visual recovery. Half-dose PDT and FA-guided laser photocoagulation achieved prompt SRF resolution and leak closure. ^[3, 7] However, structural imaging showed continuing outer retinal thinning and RPE degradation, indicating irreversible tissue damage despite fluid resolution.

Anti-VEGF therapy was effective in controlling CNV and preventing further exudation.^[12, 8]Nevertheless, these agents do not restore lost photoreceptors or reverse RPE atrophy, explaining the patient's stagnant best-corrected visual acuity (BCVA) at 20/50 by 2025. Similarly, although eplerenone has demonstrated benefits in reducing SRF recurrence, its long-term efficacy remains uncertain and subject to further investigation. ^[25]

Disease Interpretation and Research Implications

Emerging data suggest that cCSC may not solely evolve from unresolved acute CSR but could present de novo due to intrinsic choroidal pathology and RPE dysfunction. ^[1]This case supports a continuum model, where repeated subretinal fluid accumulation and chronic exposure to risk factors lead to progressive structural damage. Given the patient's partial response to current therapies, novel interventions aimed at RPE regeneration and neuroprotection are urgently needed. ^[18, 17]

Quality of Life Considerations

The patient's final NEI-VFQ-39 score of 65/100 indicates moderate visual disability, particularly in near vision and night-time activities. These functional impairments substantially affected his occupational productivity, a finding consistent with quality-of-life studies in younger patients with cCSC. ^[23] Despite anatomical control, the persistence of visual deficits underscores the necessity of integrating visual rehabilitation and psychological support into standard care models.



CONCLUSION

This longitudinal case study provides a detailed illustration of the clinical trajectory and therapeutic challenges associated with chronic central serous chorioretinopathy (cCSC). Over a seven-year span (2018–2025), the patient transitioned from acute CSR to chronic disease complicated by choroidal neovascularization (CNV), despite multimodal interventions.

Key findings include:

- Anatomical improvements were consistently achieved with half-dose photodynamic therapy (PDT), FA-guided laser photocoagulation, and anti-VEGF therapy.
- **Functional recovery**, however, was incomplete, as progressive RPE and photoreceptor damage led to a decline in best-corrected visual acuity (BCVA) from 20/25 to 20/50.
- **Multimodal imaging** was essential for diagnosis, therapeutic planning, and disease monitoring, particularly OCT, FAF, and ICGA.
- **Modifiable risk factors**—including stress, corticosteroid use, and smoking—played a key role in disease progression and should be rigorously addressed.
- Quality of life was moderately impaired, with a National Eye Institute Visual Function Questionnaire (NEI-VFQ-39) score of 65/100, underscoring the psychosocial burden of cCSC even in working-age individuals.

This case supports a continuum model of CSR pathogenesis and emphasizes the need for early and aggressive intervention, especially in recurrent cases. While current therapies can achieve structural resolution, irreversible retinal damage limits functional outcomes. Future research should prioritize:

- Neuroprotective and RPE-preserving strategies;
- Personalized therapy guided by imaging biomarkers and genetic predisposition;
- Integrated care models combining ophthalmologic, psychological, and rehabilitative support.



DECLARATION:

Acknowledgement - The Author would like to thank the heads of the institution, the teachers, the staff and more so the students for having been very co-operative in the collection of the data. We thank our entire department faculty for being encouraging and supportive throughout this study.

Source of Funding - None

Conflict of Interest - The authors declare no conflict of interest.

Ethical approval: The study was approved by the Institutional Ethics Committee

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