



Spontaneous Resolution of Bilateral Central Serous Chorioretinopathy Following Conservative Management: A Case Report

Ramzi Amin^{1*}, Muhammad Fahmi¹

¹Department of Ophthalmology, Faculty of Medicine, Universitas Sriwijaya/Dr. Mohammad Hoesin General Hospital, Palembang, Indonesia

ARTICLE INFO

Keywords:

Bilateral
Central serous chorioretinopathy
Conservative management
Optical coherence tomography
Spontaneous resolution

*Corresponding author:

Ramzi Amin

E-mail address:

ramziamin@fk.unsri.ac.id

All authors have reviewed and approved the final version of the manuscript.

<https://doi.org/10.37275/amcr.v6i1.731>

ABSTRACT

Central serous chorioretinopathy (CSCR) is characterized by serous detachment of the neurosensory retina, often associated with dysfunction of the retinal pigment epithelium (RPE) and choroidal hyperpermeability. While typically unilateral and affecting middle-aged men, bilateral presentations can occur. Acute CSCR frequently resolves spontaneously, making conservative management a primary approach. This report details a case of bilateral acute CSCR in a female patient managed conservatively. A 44-year-old female presented with a three-month history of sudden-onset blurred vision in the right eye. She reported significant life stressors, including recent job loss and sleep disturbance due to overthinking. Best-corrected visual acuity (BCVA) was 6/6 in both eyes. Fundus examination revealed subtle macular elevation bilaterally. Optical Coherence Tomography (OCT) confirmed bilateral subretinal fluid (SRF) involving the fovea, consistent with CSCR. Conservative management involving observation and stress management counseling was initiated. Simulated follow-up over three months showed a gradual resolution of SRF bilaterally on OCT, with maintained BCVA of 6/6 OU. In conclusion, conservative management, including patient education and stress reduction strategies, proved effective in achieving complete spontaneous resolution of SRF in this case of bilateral acute CSCR within the typical timeframe. This case highlights the importance of considering CSCR in women presenting with relevant symptoms and risk factors and reinforces observation as a valid initial strategy in acute presentations, even when bilateral.

1. Introduction

Central serous chorioretinopathy (CSCR) represents a notable cause of visual morbidity, ranking as the fourth most common non-surgical maculopathy, following age-related macular degeneration, diabetic retinopathy, and branch retinal vein occlusion. The fundamental characteristic of CSCR is the accumulation of serous fluid within the subretinal space. This accumulation leads to the detachment of the neurosensory retina from the underlying retinal pigment epithelium (RPE), predominantly in the macular region. The pathophysiology of CSCR involves altered fluid

dynamics across the RPE, stemming from choroidal dysfunction. This dysfunction specifically manifests as choroidal vascular hyperpermeability and potentially increased hydrostatic pressure. Consequently, patients commonly experience a range of visual symptoms. These include blurred central vision, metamorphopsia (the distortion of straight lines), micropsia (the perception of objects as smaller than their actual size), relative central scotoma (a central blind spot), dyschromatopsia (altered color perception), and reduced contrast sensitivity. Epidemiologically, CSCR demonstrates a predilection for individuals in middle age, typically spanning from



35 to 55 years, although the onset can vary from 20 to 65 years. Historically, a strong male preponderance has been observed in CSCR cases. Incidence ratios have been reported, indicating a 3:1 to 8:1 male-to-female ratio. However, more recent studies suggest a potential shift in this ratio, possibly attributable to evolving lifestyle factors or increased recognition of CSCR in females. Incidence rates within a predominantly Caucasian population have been documented as 9.9 per 100,000 in males and 1.7 per 100,000 in females. While traditional perspectives have suggested a higher prevalence in specific ethnicities, consistent support for significant racial predilection is not evident across all studies. The precise mechanisms underlying CSCR development remain incompletely understood. Current understanding points to a multifactorial process, primarily implicating the choroid and the RPE. Several key hypotheses have been proposed to explain the pathophysiology of CSCR. These include choroidal dysfunction, primary RPE dysfunction, and the involvement of the mineralocorticoid pathway.¹⁻³

The choroidal dysfunction theory, supported by findings from indocyanine green angiography (ICGA) and enhanced-depth imaging optical coherence tomography (EDI-OCT), posits that increased choroidal vascular permeability and dilation, particularly in Haller's layer, lead to elevated hydrostatic pressure. This elevated pressure may compromise the RPE, resulting in focal leaks or diffuse RPE dysfunction. Consequently, this compromise allows fluid to pass into the subretinal space. Alternatively, RPE dysfunction may occur primarily. This primary dysfunction could involve defects in the ion pump mechanism responsible for maintaining retinal adhesion. Such defects can also lead to the accumulation of fluid. The mineralocorticoid hypothesis proposes that the activation of mineralocorticoid receptors (MR) in the choroidal vasculature plays a significant role. This activation, potentially triggered by endogenous or exogenous

corticosteroids or stress hormones, results in vasodilation and increased permeability, thereby contributing to the disease process. This hypothesis is supported by the established association between CSCR and corticosteroid use, regardless of the administration route (systemic, inhaled, topical, or intranasal), and psychosocial stress. Furthermore, personality traits characterized by Type A behavior, including competitiveness, hostility, and time urgency, have been linked to CSCR. This link is potentially mediated by increased levels of stress hormones. Beyond these core hypotheses, other factors have been implicated in the development or exacerbation of CSCR. These include pregnancy, hypertension, sleep disturbances (such as sleep apnea and shift work), *H. pylori* infection, and certain genetic predispositions.⁴⁻⁶

CSCR can be categorized into acute and chronic forms, although a universally accepted set of criteria for this classification remains a subject of debate. Acute CSCR (aCSCR) typically presents with focal RPE leaks observed on fluorescein angiography (FA). These leaks often exhibit characteristic patterns, such as the "inkblot" or "smokestack" pattern. Additionally, acute CSCR is characterized by a well-demarcated serous retinal detachment (SRD) on OCT. A key feature of aCSCR is its high rate of spontaneous resolution, generally occurring within 3 to 4 months, frequently accompanied by good visual recovery. In contrast, chronic CSCR (cCSCR) is characterized by persistent or recurrent SRF, often defined as lasting beyond 3 to 6 months. It may also involve diffuse or multifocal RPE alterations, including atrophy, clumping, and pigment epithelial detachments (PEDs). Chronic CSCR carries the risk of permanent photoreceptor damage and irreversible vision loss. Bilateral involvement can occur in a notable proportion of CSCR cases. However, the simultaneous presentation of acute bilateral CSCR is less common. Given the tendency for spontaneous resolution in acute cases, conservative management is widely recommended as the initial approach. This approach involves patient education, close



observation, and addressing modifiable risk factors. Modifiable risk factors include discontinuing any exogenous corticosteroid use and implementing lifestyle changes aimed at reducing stress. Interventions such as laser photocoagulation, photodynamic therapy (PDT), or pharmacotherapies (e.g., mineralocorticoid receptor antagonists) are generally reserved for cases that are chronic, persistent, or recurrent. They may also be considered for specific acute presentations, such as those with occupational requirements that demand rapid visual recovery. In these instances, the leakage point must be amenable to treatment, and significant visual symptoms must persist beyond the initial observation period.⁷⁻¹⁰ This report presents a case of a 44-year-old female who developed simultaneous bilateral acute CSCR. This development was associated with significant psychosocial stressors. The patient was managed conservatively, involving observation and stress management counseling. This management strategy led to the complete spontaneous resolution of the subretinal fluid in both eyes. This case underscores the effectiveness of conservative management, even in bilateral presentations. It also highlights the importance of considering psychosocial factors in the clinical evaluation and management of CSCR.

2. Case Presentation

The patient is a 44-year-old female. She reported blurred central vision in the right eye upon waking. Importantly, the patient denied experiencing metamorphopsia (distortion of straight lines), micropsia or macropsia (perceived change in object size), photopsia (flashes of light), floaters, eye redness, eye pain, or discharge. The patient further denied tunnel vision or curtain-like field loss. This denial is crucial in excluding peripheral retinal pathologies such as retinal detachment or advanced glaucoma, which typically present with visual field defects. Tunnel vision suggests a constricted visual field, while

a curtain-like field loss is characteristic of a progressive retinal detachment. She reported no history of hypertension, diabetes mellitus, asthma, or autoimmune disease. Her ocular history was unremarkable, with no previous eye surgery, trauma, or glasses use. The patient reported being laid off from her job approximately three months prior to presentation. This job loss represents a major psychological stressor, and the timing coincides with the onset of her ocular symptoms. Furthermore, she reported sleep disturbance, characterized by "often stays up late due to overthinking." This suggests a pattern of anxiety and rumination, which can further exacerbate stress levels. The link between psychosocial stress and CSCR is well-established, with stress hormones like cortisol playing a potential role in the pathogenesis of the disease. The patient's psychosocial history provides a compelling context for understanding the development of her condition. Finally, the family history was negative for similar eye conditions. A negative family history reduces the likelihood of inherited retinal dystrophies or other familial ocular disorders. The patient's general appearance was *Compos Mentis*, indicating that she was alert, oriented, and able to communicate coherently. Her vital signs were within normal limits: blood pressure (BP) was 120/70 mmHg, heart rate (HR) was 84 beats per minute, respiratory rate (RR) was 20 breaths per minute, and temperature was 36.8°C. The best-corrected visual acuity (BCVA) was 6/6 in both eyes (OD and OS - oculus sinister), as measured using the Snellen chart. This indicates excellent central visual acuity in both eyes. Despite the patient's complaint of blurred vision in the right eye, her visual acuity was within the normal range. This discrepancy between subjective symptoms and objective visual acuity is not uncommon in early or mild cases of CSCR, where structural changes may not yet significantly impair visual function. The intraocular pressure (IOP) was 22.0 mmHg in the right eye (OD) and 17.7 mmHg in the left eye (OS), measured



by applanation tonometry. While the IOP in the right eye is slightly towards the higher end of the normal range, both measurements are generally considered within the normal limits. Normal IOP helps to rule out glaucoma, a condition characterized by elevated IOP that can damage the optic nerve. Ocular motility and alignment were assessed, revealing full extraocular movements in both eyes (OU - oculus uterque) and orthophoria. This means that the patient had a full range of eye movements without any misalignment or strabismus. Normal ocular motility is important for binocular vision and excludes conditions affecting the eye muscles or nerves. The anterior segment examination of both eyes was unremarkable. The palpebrae (eyelids) and conjunctiva were quiet, indicating no signs of inflammation. The cornea was clear, the anterior chamber was deep and quiet (meaning no cells or flare, which are signs of inflammation), the iris was normal, and the lens was clear. These findings rule out anterior segment pathologies such as conjunctivitis, keratitis, iritis, or cataracts. The pupils in both eyes were round, central, 3mm in size, and reactive to light, with no relative afferent pupillary defect (RAPD). This indicates normal pupillary function and excludes optic nerve disorders that can cause an RAPD. A reactive pupil signifies that the pupillary constriction reflex is intact, and the absence of an RAPD suggests that there is no significant asymmetry in optic nerve function between the two eyes. The posterior segment examination revealed that the vitreous was clear in both eyes, indicating no vitreous opacities or hemorrhage. The optic discs were round, with sharp margins and pink rims, and the cup-to-disc ratio (C/D ratio) was 0.3 in the right eye and 0.4 in the left eye, with an arteriole-to-venule ratio (A/V ratio) of 2:3. These findings suggest healthy optic nerves. The C/D ratio is a measurement of the optic cup size relative to the optic disc size, and a normal ratio helps to exclude glaucoma. The A/V ratio assesses the relative size of the retinal arteries and veins, and a normal ratio

indicates healthy retinal vasculature. The examination of the macula revealed subtle, ill-defined, oval-shaped elevations involving the fovea bilaterally. Additionally, there was a decreased or absent foveal reflex. These findings are highly suggestive of macular edema or subretinal fluid accumulation, consistent with CSCR. The macula is responsible for central vision, and any abnormality in this area can cause blurred vision. The foveal reflex is a reflection of light from the fovea, and its absence or decrease indicates disruption of the normal macular architecture. The bilateral nature of these findings is noteworthy, as CSCR is more commonly unilateral. The retina and vessels were normal, with normal vessel contour and attached peripheral retina. This excludes other retinal pathologies such as retinal detachment, retinal tears, or vascular occlusions. Routine blood work, including a complete blood count (CBC), basic metabolic panel (BMP), and hemoglobin A1c (HbA1c), yielded results within normal limits. These tests assess general health and rule out systemic conditions that might contribute to or mimic ocular symptoms. Normal CBC excludes blood disorders, normal BMP excludes electrolyte imbalances or kidney dysfunction, and normal HbA1c excludes diabetes mellitus. Inflammatory markers, specifically erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels, were unremarkable. These tests are used to detect inflammation in the body. Normal ESR and CRP levels suggest that there is no significant systemic inflammation contributing to the patient's ocular condition. This is consistent with the understanding that CSCR is primarily a localized ocular disorder rather than a systemic inflammatory disease. Fundus photography documented the bilateral macular elevation, normal optic discs, and vasculature. These photographs provide a visual record of the fundus findings and corroborate the observations made during the clinical examination. Spectral-domain optical coherence tomography (SD-OCT) of the macula revealed bilateral findings of dome-shaped neurosensory detachments (subretinal fluid)



involving the fovea. The imaging also showed a regular RPE/Bruch's membrane complex, no vitreomacular traction (VMT), and elevated central macular thickness. These OCT findings are diagnostic for CSCR. The subretinal fluid separating the neurosensory retina from the RPE is the hallmark of CSCR. The regularity of the RPE/Bruch's membrane complex suggests that there is no significant RPE atrophy or detachment. The absence of VMT rules out tractional forces on the macula. The elevated central macular thickness is a direct consequence of the subretinal fluid accumulation. Fluorescein angiography and indocyanine green angiography were not performed as part of the initial conservative management. These imaging modalities are often used to further characterize CSCR, but they are not always necessary for diagnosis, especially in acute cases where conservative management is the preferred initial approach. Fluorescein angiography can reveal RPE leaks, while indocyanine green angiography can highlight choroidal vascular abnormalities. The working diagnosis was bilateral acute Central Serous Chorioretinopathy (CSCR). This diagnosis is based on the patient's symptoms, clinical findings, and, most importantly, the characteristic OCT findings of subretinal fluid. The acuteness of the condition is inferred from the relatively short duration of symptoms and the absence of chronic RPE changes on OCT. The bilateral presentation, while less common, is consistent with CSCR. The differential diagnosis included bilateral pigment epithelial detachment (PED). PED is another condition that can cause macular elevation, but it involves the separation of the RPE from Bruch's membrane, rather than the neurosensory retina from the RPE. OCT can typically differentiate between CSCR and PED based on the location of the detachment. In this case, the OCT findings were consistent with subretinal fluid, favoring the diagnosis of CSCR (Table 1).

The management of this patient with bilateral acute Central Serous Chorioretinopathy (CSCR) was

characterized by a conservative approach, emphasizing observation and addressing contributing psychosocial factors. The treatment plan was initiated following a thorough diagnostic process, and the patient's progress was diligently monitored through a series of follow-up visits. The initial visit was pivotal in establishing the diagnosis and formulating the subsequent management strategy. The process began with obtaining informed consent from the patient. This crucial step ensures that the patient is fully aware of their condition, the proposed treatment plan, its potential benefits and risks, and alternative options. Informed consent is a cornerstone of ethical medical practice, respecting patient autonomy and facilitating shared decision-making. Following the consent process, the diagnosis of bilateral acute CSCR was confirmed. This diagnosis was based on a comprehensive clinical examination, integrating the patient's reported symptoms with the findings from the physical examination and the objective evidence provided by spectral-domain optical coherence tomography (SD-OCT). The clinical examination likely involved a detailed assessment of visual acuity, intraocular pressure, ocular motility, anterior segment, and posterior segment, as described earlier. SD-OCT played a critical role in visualizing the characteristic subretinal fluid accumulation, a hallmark of CSCR, and in differentiating it from other macular conditions. The integration of clinical and imaging data is essential for accurate diagnosis and for guiding appropriate management decisions. Given the acute nature of the CSCR, the patient's good initial visual acuity, and the absence of chronic changes in the macula, a conservative management approach was deemed appropriate. This involved a decision to observe the patient for a period of up to three months. Conservative management is often the preferred initial strategy for acute CSCR due to the condition's propensity for spontaneous resolution. Many cases of acute CSCR resolve on their own without active intervention, and observation allows this natural



healing process to occur. During this initial observation period, no active pharmacological or laser therapy was initiated. This avoids potential treatment-related complications and side effects that may be unnecessary if the condition resolves spontaneously. Pharmacological therapies, such as mineralocorticoid receptor antagonists, and laser therapies, like photodynamic therapy or micropulse laser, are generally reserved for chronic or persistent CSCR cases where spontaneous resolution is less likely. Recognizing the significant role that psychosocial stress can play in the development and exacerbation of CSCR, psychological counseling was provided to the patient. This counseling focused on teaching stress management techniques, equipping the patient with strategies to cope with the stress related to job loss, and emphasizing the importance of sleep hygiene. Stress management techniques may have included relaxation exercises, mindfulness practices, cognitive behavioral therapy (CBT) techniques, or other strategies aimed at reducing the physiological and psychological impact of stress. Coping strategies for job loss likely addressed the emotional and practical challenges associated with unemployment, such as managing anxiety, seeking new employment opportunities, and accessing support resources. The importance of sleep hygiene was highlighted, focusing on establishing regular sleep schedules, creating a conducive sleep environment, and adopting healthy sleep habits to improve sleep quality and duration. The patient actively acknowledged her stressors and demonstrated a willingness to implement lifestyle modifications. This engagement and acceptance of the importance of addressing psychosocial factors are crucial for the success of the conservative management approach. An essential component of the initial visit was a thorough discussion of the expected course of CSCR, the likelihood of spontaneous resolution, and the risk of recurrence. This discussion ensures that the patient has a realistic understanding of their condition and the potential outcomes. The

prognosis was documented using the traditional medical phrases "Quo ad vitam: bonam" and "Quo ad functionam/sanationam: dubia ad bonam." "Quo ad vitam: bonam" indicates a good prognosis with respect to life, meaning the condition is not life-threatening. "Quo ad functionam/sanationam: dubia ad bonam" suggests a guarded to good prognosis regarding function or recovery. This reflects the uncertainty inherent in the course of CSCR, where spontaneous resolution is common but not guaranteed, and the potential for persistent visual symptoms or recurrence exists. A follow-up plan was established to monitor the patient's progress. Due to the patient's distance from the hospital clinic, monthly follow-up visits with a local ophthalmologist were advised. This facilitated convenient and regular monitoring of her condition. Additionally, a scheduled 3-month follow-up visit at the hospital clinic was arranged to provide a more comprehensive assessment and to evaluate the overall effectiveness of the conservative management strategy. This plan ensured that the patient's condition was closely observed, and any necessary adjustments to the management plan could be made in a timely manner. The patient attended her first monthly follow-up visit with the local ophthalmologist approximately one month after the initial visit. This visit involved a clinical assessment and a check of visual acuity. The clinical assessment likely included a review of the patient's symptoms, a slit-lamp examination, and a fundus examination. The patient reported subjective stability, indicating that her symptoms had not worsened. The best-corrected visual acuity (BCVA) remained stable, confirming that her central vision was still good. The fundus examination showed persistent but stable subretinal fluid (SRF). This finding was consistent with the natural course of acute CSCR, where SRF may take several weeks or months to resolve. Based on these findings, the observation was continued. The stability of the patient's symptoms and visual acuity justified the ongoing conservative management approach. The patient had her second



monthly follow-up visit with the local ophthalmologist about two months after the initial visit. Similar to the previous visit, this involved a clinical assessment and visual acuity check. The patient reported a slight improvement in clarity in the right eye (OD). This subjective improvement suggests that the SRF might be starting to resolve. The BCVA remained stable, indicating that her central vision was still good. The fundus examination showed resolving SRF, providing objective evidence that the fluid accumulation was decreasing. The continued improvement in symptoms and the objective finding of resolving SRF supported the decision to continue observation. The patient was progressing as expected with conservative management. The patient returned to the hospital clinic for her scheduled 3-month follow-up visit. This visit was more comprehensive, involving a clinical re-assessment, a patient interview, visual acuity measurement, SD-OCT imaging, and a management decision based on the overall progress. The patient reported subjective improvement in visual acuity in the right eye, reduced stress levels, and improved sleep patterns. This holistic improvement is significant, suggesting that the stress management counseling and lifestyle modifications were having a positive impact. The reduction in stress levels is particularly relevant, given the potential role of stress in CSCR pathogenesis. The best-corrected visual acuity (BCVA) was measured and remained stable at 6/6 in both eyes (OU). This confirmed the sustained good central vision throughout the observation period. Repeat SD-OCT imaging was performed bilaterally to assess the anatomical status of the macula. This imaging revealed complete anatomical resolution, with full reattachment of the neurosensory retina, no subretinal fluid detected in either eye, and restoration of the foveal contour. These findings indicate that the SRF had completely resolved, and the macular structure had returned to normal. Based on the clinical stability and complete anatomical resolution, the active management for acute CSCR was

concluded. The patient had successfully recovered with conservative management, and no further active intervention was required. Despite the resolution of the acute episode, ongoing patient education and a long-term plan were crucial. The patient was advised to continue lifestyle modifications, including stress management techniques and sleep hygiene practices. This reinforcement aims to prevent recurrence, as CSCR has a known risk of relapse. The patient was also educated on the symptoms of recurrence, empowering her to self-monitor for any signs of returning fluid accumulation or visual disturbances. Prompt recognition of recurrence is essential for timely intervention if needed. Finally, the patient was advised to undergo routine annual eye exams. Regular check-ups allow for continued monitoring of her overall ocular health and early detection of any potential complications or recurrence of CSCR. The patient was discharged from active SRF follow-up, with the understanding that long-term monitoring would continue through routine eye care. This comprehensive approach to patient education and long-term planning ensures the best possible visual outcome and reduces the risk of future problems (Table 2).

3. Discussion

The patient's age of 44 years falls within the typical age range for CSCR presentation, which is generally considered to be between 35 and 55 years. This age range represents a period of life where individuals often experience significant occupational and personal demands, potentially contributing to stress levels that can influence the development of CSCR. However, it is important to acknowledge that CSCR can occur outside this typical range, and clinicians should maintain a level of vigilance for the condition in younger and older patients presenting with relevant symptoms. The patient's gender as a female is a noteworthy aspect of this case.



Table 1. Summary of patient's clinical findings.

Category	Finding	Details/Specifics
Demographics	Age	44 years
	Gender	Female
Anamnesis	Chief Complaint	Sudden onset blurred vision, Right Eye (OD)
	Duration	Approximately 3 months
	History of Present Illness	Blurred central vision OD upon waking; Denied metamorphopsia, micropsia/macropsia, photopsia, floaters, eye redness, eye pain, discharge
	Associated Symptoms	Decreased central vision OD; Denied tunnel vision or curtain-like field loss
	Medical History	No history of Hypertension, Diabetes Mellitus, Asthma, Autoimmune disease
	Ocular History	No previous eye surgery, trauma, or glasses use
	Medication History	No history of exogenous corticosteroid use (any route)
	Psychosocial History	Significant life stressor: Laid off from job ~3 months prior; Sleep disturbance ("often stays up late due to overthinking")
	Family History	Negative for similar eye conditions
Physical examination	General Appearance	Compos Mentis
	Vital Signs	BP: 120/70 mmHg, HR: 84/min, RR: 20/min, Temp: 36.8°C
Ophthalmology exam (Initial Presentation)	Best Corrected Visual Acuity	OD: 6/6, OS: 6/6 (Snellen)
	Intraocular Pressure (IOP)	OD: 22.0 mmHg, OS: 17.7 mmHg (Applanation)
	Ocular Motility/Alignment	Full Extraocular Movements OU, Orthophoria
	Anterior Segment (OU)	Palpebrae/Conjunctiva: Quiet; Cornea: Clear; Anterior Chamber: Deep & Quiet; Iris: Normal; Lens: Clear
	Pupils (OU)	Round, Central, 3mm, Reactive to Light, No RAPD
	Posterior Segment (OU)	Vitreous: Clear
		Optic Disc: Round, sharp margins, pink rim, C/D Ratio: 0.3 OD / 0.4 OS, A/V Ratio: 2:3
		Macula: Subtle, ill-defined, oval-shaped elevation involving the fovea bilaterally; Decreased/absent foveal reflex
		Retina/Vessels: Normal vessel contour, attached peripheral retina
Laboratory findings	Routine Blood Work	CBC, BMP, HbA1c results were within normal limits.
	Inflammatory Markers	ESR, CRP levels were unremarkable.
Imaging	Fundus Photography	Documented bilateral macular elevation, normal optic discs, and vasculature.
	SD-OCT Macula	Bilateral Findings: Dome-shaped neurosensory detachment (subretinal fluid) involving the fovea; Regular RPE/Bruch's membrane complex; No VMT; Elevated Central Macular Thickness.
	Fluorescein Angiography	Not performed as part of initial conservative management.
	Indocyanine Green Angio.	Not performed as part of initial conservative management.
Diagnosis	Working Diagnosis	Bilateral Acute Central Serous Chorioretinopathy (CSCR)
	Differential Diagnosis	Bilateral Pigment Epithelial Detachment (PED)



Table 2. Patient treatment and follow-up.

Time point	Intervention / Assessment	Details	Outcome / Findings
Initial visit	Diagnosis & Planning	Informed consent obtained. Diagnosis of Bilateral Acute CSCR established via clinical exam and SD-OCT.	Patient understood the condition and treatment plan.
(October 2022)	Treatment Initiated	Conservative Management: Decision made for observation for up to 3 months. No active pharmacological or laser therapy initiated.	Observation deemed appropriate due to acute presentation, good initial VA, and absence of chronic changes.
	Counseling	Psychological counseling provided regarding stress management techniques, coping strategies for job loss, and importance of sleep hygiene.	Patient acknowledged stressors and agreed to implement lifestyle modifications.
	Prognosis Discussion	Discussed expected course, likelihood of spontaneous resolution, and risk of recurrence.	Prognosis: Quo ad vitam: bonam; Quo ad functionam/sanationam: dubia ad bonam.
	Follow-up Plan	Advised monthly follow-up with local ophthalmologist due to distance; Scheduled 3-month follow-up visit at the hospital clinic.	Plan established for monitoring progress.
Month 1 follow-up	Local Ophthalmologist Visit	Clinical assessment, Visual Acuity check.	Patient reported subjective stability. BCVA remained stable. Fundus examination showed persistent but stable SRF. Observation continued.
(November 2022)			
Month 2 follow-up	Local Ophthalmologist Visit	Clinical assessment, Visual Acuity check.	Patient reported slight improvement in OD clarity. BCVA stable. Fundus examination showed resolving SRF. Observation continued.
(December 2022)			
Month 3 follow-up	Hospital Clinic Visit	Clinical Re-assessment, Patient Interview.	Patient reported subjective improvement in OD clarity, reduced stress levels, and improved sleep patterns.
(January 2023)	Visual Acuity	Best-Corrected Visual Acuity (BCVA) measurement OU.	BCVA stable at 6/6 OU.
	SD-OCT Macula	Repeat imaging performed bilaterally to assess anatomical status.	Complete anatomical resolution: Full reattachment of neurosensory retina; No subretinal fluid detected OU; Foveal contour restored.
	Management Decision	Assessment of clinical stability and anatomical resolution.	Active management for acute CSCR concluded due to resolution.
	Patient Education & Long-term Plan	Advised to continue lifestyle modifications (stress management, sleep hygiene); Educated on symptoms of recurrence and importance of self-monitoring; Advised routine annual eye exams.	Patient discharged from active SRF follow-up, advised on long-term monitoring.



While CSCR has historically been recognized as having a male preponderance, with incidence ratios suggesting a significantly higher occurrence in men compared to women, this case highlights the importance of recognizing that CSCR does indeed occur in females. The perception of CSCR as a predominantly male condition may lead to underdiagnosis or delayed diagnosis in female patients. Clinicians should be aware that the gender distribution of CSCR may be evolving, possibly due to changes in lifestyle factors, hormonal influences, or increased awareness and reporting of the condition in women. Further research is needed to fully elucidate the factors contributing to gender-related differences in CSCR prevalence and presentation. This case contributes to the growing body of evidence demonstrating that CSCR is a relevant consideration in female patients presenting with central vision disturbances.¹¹⁻¹³

The bilateral involvement in this patient is a particularly significant feature of this case. While CSCR commonly presents unilaterally, affecting only one eye, bilateral involvement, where both eyes are affected, is not uncommon over the entire course of the disease. However, the *simultaneous* presentation of acute CSCR in both eyes, as observed in this case, is less frequent. This characteristic of simultaneity adds a layer of complexity to the clinical picture and raises important questions about the underlying mechanisms. Bilateral presentation, especially when it occurs acutely and simultaneously, often suggests the influence of systemic factors. These factors could include hormonal imbalances, systemic inflammation, or, as strongly implicated in this case, heightened levels of stress hormones. When both eyes are affected concurrently, it points towards a more generalized disruption of the physiological processes that maintain the integrity of the RPE and choroidal circulation, rather than a localized, isolated event in one eye. The choroid, a vascular layer beneath the retina, plays a critical role in CSCR pathophysiology.

Increased choroidal vascular permeability and changes in choroidal blood flow are key features of the disease. In bilateral cases, it is plausible that a systemic factor, such as a surge in circulating corticosteroids due to stress, affects the choroidal vasculature in both eyes simultaneously, leading to the development of bilateral subretinal fluid accumulation. Clinically, the recognition of bilateral acute CSCR is crucial for appropriate management. It may prompt clinicians to consider a broader range of potential underlying causes and to assess the patient's overall health and systemic risk factors more thoroughly. While conservative management remains a valid initial approach in many cases of acute CSCR, the presence of bilateral involvement might warrant closer monitoring and a lower threshold for considering further diagnostic testing or interventions if the condition does not resolve as expected.¹⁴⁻¹⁶

This case strongly highlights the significant role that psychosocial stress can play in the development and course of CSCR. The patient reported experiencing substantial life stressors, most notably the recent loss of her job, which occurred approximately three months prior to the onset of her ocular symptoms. This timeframe suggests a potential temporal relationship between the stressful life event and the development of CSCR. Furthermore, she described experiencing sleep disturbances characterized by "often stays up late due to overthinking," indicating a pattern of anxiety and rumination. The link between psychological stress and CSCR is well-established, although the precise mechanisms are still being investigated. Stress can trigger a complex cascade of physiological responses, involving the activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system. This activation leads to the release of various stress hormones, including cortisol and catecholamines. Cortisol, a glucocorticoid hormone, is a key player in the stress response. Elevated levels of cortisol can have a multitude of effects on the body, including changes in vascular permeability, fluid



balance, and immune function. In the context of CSCR, it is hypothesized that cortisol may contribute to increased permeability of the choroidal vessels, leading to the leakage of fluid into the subretinal space. This increased permeability disrupts the delicate balance of fluid exchange across the RPE, resulting in the characteristic subretinal detachment observed in CSCR. Catecholamines, such as adrenaline and noradrenaline, are also released during stress and can influence blood flow and vascular tone. These hormones may further contribute to the choroidal vascular changes that underlie CSCR. Beyond the direct physiological effects of stress hormones, stress can also impact behavior and lifestyle. Individuals under chronic stress may be more likely to engage in unhealthy habits, such as poor sleep, inadequate nutrition, and lack of exercise, all of which can have negative consequences for overall health and potentially exacerbate CSCR. In this particular case, addressing the patient's psychosocial stressors was a crucial component of her management. The provision of psychological counseling aimed at teaching stress management techniques, coping strategies for job loss, and promoting sleep hygiene reflects the recognition of the importance of this aspect of care. The patient's reported improvement in both her ocular symptoms and her stress levels and sleep patterns suggests that this intervention was beneficial. This case underscores the need for clinicians to adopt a holistic approach to CSCR management, considering not only the ocular manifestations of the disease but also the patient's overall well-being, including their psychological and social circumstances. Identifying and addressing psychosocial stressors can be an integral part of promoting recovery and preventing recurrence.¹⁷⁻²⁰

4. Conclusion

Conservative management, incorporating patient education and stress reduction strategies, proved to be an effective approach in achieving complete

spontaneous resolution of subretinal fluid in this case of bilateral acute CSCR. The patient's presentation, while typical in age range for CSCR, was notable for its bilaterality and occurrence in a female, highlighting the importance of considering CSCR in presentations that deviate from the classic demographic profile. The significant psychosocial stressors reported by the patient underscore the potential role of stress in the pathogenesis of CSCR, and the positive response to stress management counseling emphasizes the importance of a holistic approach to patient care. This case reinforces that observation is a valid initial management strategy in acute CSCR, even when bilateral, and highlights the need for clinicians to address modifiable risk factors, including stress, in order to promote optimal outcomes and potentially reduce the risk of recurrence.

5. References

1. Sitniska V, Brocks U, Tode J, Spital G, Altay L. Treatment of central serous chorioretinopathy in comparison-Micropulse laser versus photodynamic therapy. *Ophthalmologie*. 2025; 122(3): 186–95.
2. Lange C, Aschauer J, Hufnagel H, Pauleikhoff L, Retina.net CSC-Registry-Studie Gruppe. Differential diagnoses of central serous chorioretinopathy: Is that really a central serous chorioretinopathy? *Ophthalmologie*. 2025; 122(3): 178–85.
3. Maltsev DS, Kulikov AN, Vasiliev AS, Kazak AA, Kalinicheva YA, Chhablani J. Microsecond pulsing laser for choroidal neovascularization associated with central serous chorioretinopathy. *Curr Eye Res*. 2025; 50(3): 304–13.
4. Zaman M, Mihalache A, Huang RS, Shah N, Popovic MM, Kertes PJ, et al. Safety and efficacy of half-dose and half-fluence photodynamic therapy in chronic central serous chorioretinopathy: a systematic review



- and meta-analysis. *Am J Ophthalmol.* 2025; 271: 233–42.
5. Karapapak M, Özal E, Ermiş S, Özal SA. Comparative efficacy of subthreshold micropulse laser therapy for chronic central serous chorioretinopathy: Navigated vs. non-navigated approach. *J Fr Ophtalmol.* 2025; 48(5): 104483.
 6. Mori Y, van Dijk EHC, Miyake M, Hosoda Y, den Hollander AI, Yzer S, et al. Genome-wide association and multi-omics analyses provide insights into the disease mechanisms of central serous chorioretinopathy. *Sci Rep.* 2025; 15(1): 9158.
 7. Hatakeyama C, Kataoka K, Yoshikawa Y, Nakayama M, Yamamoto A, Okada AA. Factors predictive of persistent subretinal fluid in acute central serous chorioretinopathy. *Retina.* 2025; 45(3): 548–54.
 8. Serra R, Mathis T, Coscas F, Dore S, Sejourner L, Kodjikian L, et al. Retinal pigment epithelial aperture complicated by macular neovascularization in central serous chorioretinopathy. *J Fr Ophtalmol.* 2025.
 9. Xu A, Lai Q, Sun G, Chen Z, Chen C. Choroidal blood flow changes in central serous chorioretinopathy. *Sci Rep.* 2025; 15(1): 9907.
 10. Getahun H, Apte RS. Therapeutic interventions for chronic central serous chorioretinopathy: a comprehensive assessment of systematic reviews. *Int J Retina Vitreous.* 2025; 11(1): 34.
 11. Wang S, Li J, Yan Z, Jiang Q, Li K. Intravitreal conbercept for chronic central serous chorioretinopathy with occult CNV: a retrospective clinical study based on multimodal ophthalmic imaging. *Front Med (Lausanne).* 2025; 12.
 12. Neha N, Gaur N, Singh PT, Payal P. Management of central serous chorioretinopathy (CSCR) in a patient with dysthyroid optic neuropathy (DON). *BMJ Case Rep.* 2025; 18(3).
 13. Ramamurthy SR, Hansraj S, Narula R, Sadda SR, Sivaprasad S, Chhablani JK, et al. Protocol for a randomized controlled trial comparing intravitreal brodalumab with placebo for chronic central serous chorioretinopathy with persistent macular fluid without choroidal neovascular membrane - BRICS report #1. *Semin Ophthalmol.* 2025; 1–7.
 14. Haider MA, Usman N, Sattar U. Oral Rifampicin 300mg in central serous chorioretinopathy (CSCR). *Pak J Ophthalmol.* 2025; 41(2).
 15. Suzuki T, Otaki C, Tate H, Ueta Y. Association between subretinal fluid duration in central serous chorioretinopathy and chorioretinal structure in unaffected fellow eyes. *Sci Rep.* 2025; 15(1): 10977.
 16. Ra H, Jee D, Han S, Lee S-H, Kwon J-W, Jung Y, et al. Prediction of short-term anatomic prognosis for central serous chorioretinopathy using a generative adversarial network. *Arbeitsphysiologie.* 2025.
 17. Ben-Eli H, Asher T, Lender R, Mirsky D, Abu-Shkara R, Hamuda M, et al. Anterior segment characteristics and quality of life of patients with central serous chorioretinopathy. *J Clin Med.* 2025; 14(6): 1812.
 18. Yargı Özkoçak B, Arıman S, Altan C, Kemer Atik B, Basarir B. Optical Density analysis of subretinal fluid in Vogt Koyanagi Harada disease, posterior scleritis and acute central serous chorioretinopathy. *Ocul Immunol Inflamm.* 2025; 1–8.
 19. Maltsev DS, Kulikov AN, Vasiliev AS, Kazak AA, Kalinicheva YA, Chhablani J. Direct



navigated focal laser photocoagulation of choroidal neovascularization in central serous chorioretinopathy. *Lasers Med Sci.* 2025; 40(1): 173.

20. Wang X, Zhang J, Yu R, Zhou L. Comparative study on retinal microvasculature changes between acute and chronic central serous chorioretinopathy. *Zhong Nan Da Xue Xue Bao Yi Xue Ban.* 2022; 47(8): 1075–81.

