Central serous retinopathy: update on disease understanding and treatment

Mary-Grace R. Reeves and Katherine E. Talcott

Purpose of review
Investigations over the past 2 years involving novel genomics pathways, imaging techniques, risk factors, and therapeutic interventions have sought to better understand and manage central serous retinopathy (CSR). While most cases of acute CSR are self-resolving, chronic CSR remains a challenging condition to manage given response to therapy may be limited and the risk of permanent, severe vision loss. In this work, we present the latest insights on disease pathophysiology and management for acute and chronic cases of CSR, highlighting data from randomized control trials and meta-analyses to compare efficacy of treatment options.

Recent findings
There is no difference in best corrected visual acuity (BCVA) or resolution of subretinal fluid (SRF) in cases of chronic CSR that are treated with half-dose/half-fluence photodynamic therapy (PDT) versus full-strength/full-fluence therapy, making half-dose/half-fluence a reasonable option to avoid the atrophic retinal changes that may be more likely with full-strength treatment. Laser therapy could be considered as an alternative to PDT for treating chronic CSR in cases of verteporfin shortage; however, the statistically significant reduction in subretinal fluid seen on optical coherence tomography (OCT) does not translate to a significant improvement in BCVA after intervention. While there may be an early improvement in BCVA and SRF in cases of chronic CSR for which treatment with mineralocorticoid receptor antagonist is initiated, this effect does not appear to persist with extended follow up.

Summary
With ongoing extensive research on the disease process of CSR and a more nuanced understanding of the factors that increase risk of disease, observation remains the mainstay of management for acute CSR. For chronic CSR, PDT, laser, or anti-VEGF may be considered, with advances in imaging allowing OCTA to be a less invasive alternative method to dye angiography for detecting neovascularization.

Keywords
central serous retinopathy, pachychoroid disease, photodynamic therapy, subretinal fluid

INTRODUCTION

Central serous retinopathy (CSR) presents as macular or extramacular subretinal detachments with accompanying pigment epithelial detachments (PEDs) and can result in atrophic changes of the proximal retinal pigment epithelium (RPE). The modern description of CSR is often attributed to Gass, who postulated that the serous detachment of the retina was due to pathology of the underlying choroid, neovascularization, and leakage into the sub-RPE or subretinal space [1]. Other earlier descriptions of similar phenomena are attributed to von Graefe and Bennett [2,3]. Nearly 6 in 100 000 people are diagnosed with CSR, with men being affected between 5.7 and 10 times as much as women [4]. Reported symptoms include metamorphopsia, color vision defects, decreased visual acuity, scotoma or visual field loss, and decreased contrast sensitivity [5]. In many cases, acute CSR is managed with close observation and spontaneous resolution of the subretinal fluid occurs with no or minimal effect on vision. For recurrent or chronic CSR, visual defects can be more severe and long-lasting. For nearly 13% of patients with chronic CSR, followed for a period of 10 years after their first visit, BCVA was 20/200 or worse at final visit (patients with other ocular comorbidities were excluded from this study) [6]. Photodynamic therapy

Cole Eye Institute, Cleveland Clinic, Cleveland, Ohio, USA

Correspondence to Katherine E. Talcott, MD, Cole Eye Institute, Cleveland Clinic, Cleveland, Ohio, USA, 9500 Euclid Avenue, i32, Cleveland, OH 44195, USA. Tel: +1 216 444 6296; e-mail: talcotk@ccf.org

Curr Opin Ophthalmol 2026, 37:143–148

DOI:10.1097/ICU.0000000000001206

KEY POINTS

- Observation and reduction of risk factors is still favored for management for acute CSR.
- Ongoing work in genomics is helping to better characterize the pathway involved in disease presentation and progression.
- Advances in imaging have helped facilitate more accurate and earlier diagnoses of choroidal neovascularization in chronic CSR, a source of the more severe and permanent visual limitations of this disease.
- For chronic CSR, half-dose/half-fluence options for PDT seem comparable in effectiveness to full-dose therapy with a lower risk profile and no difference in posttreatment BCVA.

(PDT) or laser may be considered, in addition to anti-VEGF injections, for management of any choroidal neovascularization. Although this condition was first recognized nearly two centuries ago, much about the pathophysiology of the disease process remains unknown. Recent efforts have incorporated advances in genomics and imaging to better elucidate the pathophysiology of CSR, predict patient outcomes, define risk factors, and optimize management.

MECHANISM AND DIAGNOSIS

The past few years have had remarkable progress in imaging and genomics analyses investigating CSR. Understanding the genetic biomarkers associated with CSR risk can help better define the clinical disease pathway and potentially allow development of targeted therapeutics in the future.

Genomic studies and similarities to age-related macular degeneration

While there are abundant clinical descriptors for the presentation of CSR, additional insight into disease pathophysiology has more recently been obtained through genome-wide association studies (GWAS). Initially, a case-control GWAS identified variants in complement factor H (CFH) gene expression as being associated with CSR risk [7]. This work followed suspicion from targeted candidate gene study for an inverse relationship between AMD risk and CSR risk based on variant expression at the CFH gene [8]. The complement pathway has been a similar area of interest in studying AMD. While the complement pathway may be upregulated in AMD, there is an inverse relationship with downregulation noted in CSR [7,9▪]. Further analyses have shown genetic similarities between CSR and wet age-related macular degeneration, with shared genetic loci being identified, including WBP1L, GATA5, CFH, C2/FB, TNFRSF10A, NOTCH4, and ARMS2 [9▪,10,11]. This new understanding of the overlap of these disease processes will contribute to continued advancement of treatment for both conditions.

PTPRB has been recognized as a gene of interest in the vascular hyperpermeability hypothesis for CSR pathophysiology. Loss of PTPRB disrupts tyrosine kinase receptor Tie-2 and VE-cadherin expression, which are important for angiogenesis and endothelial cell integrity, respectfully. Consistent with an underlying vascular etiology of CSR, the PTPRB allelic variant associated with increased CSR risk was also associated with developing varicose veins [12]. Another meta-analysis found CDH7, expression of which was shown to be associated with cortisol levels, induces higher odds of CSR in male patients [13]. This finding provides a molecular basis for the known disease risks of steroid use and male sex. The full extent of the clinical applications of recent GWAS is yet to be completely understood, but there is certainly promise in what can be offered by genomics to better describe and manage the disease process.

Imaging

Diagnosis of CSR is made based on the presence of subretinal and sub-RPE fluid on optical coherence tomography (OCT), as seen in Fig. 1. However, for those eyes with CSR that develop macular neovascularization, optical coherence tomography angiography (OCTA) has excellent sensitivity (92.9%) and specificity (99.4%) and is less invasive than the oft-considered gold standard of retinal dye angiography [14]. Choroidal thickening is present on enhanced depth imaging optical coherence tomography (EDI-OCT) in eyes with acute CSR and has been found to persist in chronic CSR [15]. Another step forward is the recent use of deep learning models to recognize and diagnose CSR. Models have already been developed with near 100% accuracy, sensitivity, and specificity on smaller datasets [16]. However, while these models could provide significant diagnostic aid for interpretation of OCT findings, they are not yet being widely used due to the limited size of training datasets (for example, a deficit of eyes with subretinal fluid relative to without in the model dataset). If these algorithms can be shown to be equally effective with a larger and more diverse population, they will likely become more universally adopted to assist in diagnosis.

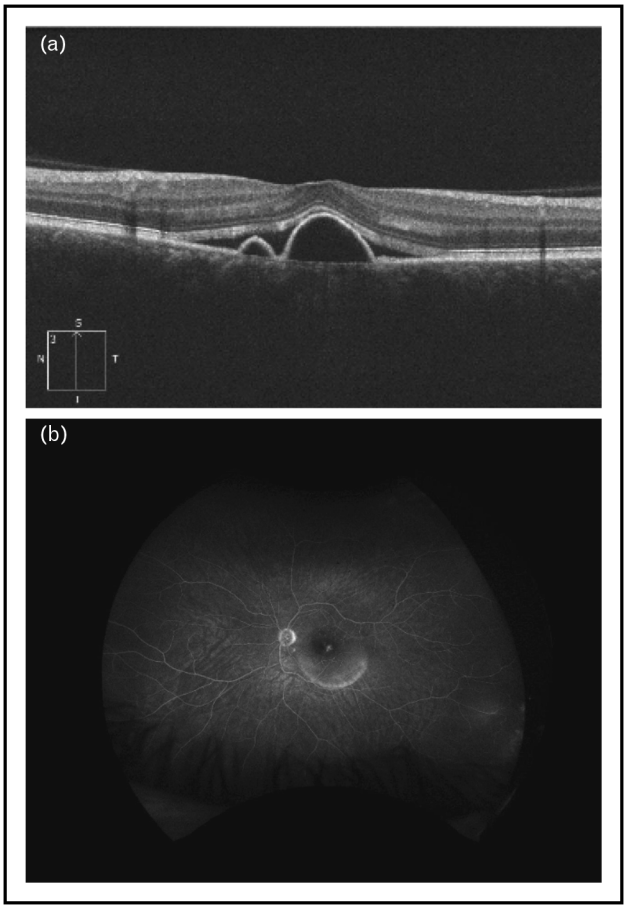

FIGURE 1. A 40-year-old female patient with history of recurrent central serous retinopathy in both eyes. (a) Macular OCT showing subretinal and sub-retinal pigment epithelium fluid, as seen in active disease. (b) Fluorescein angiogram showing late juxtafoveal leakage.

RECENT REVELATIONS IN RISK FACTORS

Obstructive sleep apnea

A few prior studies have supported an association between obstructive sleep apnea and CSR, including a prior meta-analysis of more than 7000 patients (roughly 1500 of which had CSR) which showed 1.56 higher odds of sleep apnea in CSR patients [17]. A more recent, larger meta-analysis including more than 332 000 patients (nearly 11 000 of which had CSR) similarly found that individuals with CSR had 2.28 higher odds to also have a diagnosis of obstructive sleep apnea [18]. Despite this statistically significant association and while obstructive sleep apnea can have severe effects on cardiopulmonary and neurologic health, in addition to increasing risk of other ocular conditions including nonarteritic anterior ischemic optic neuropathy, screening for sleep apnea in patients with CSR has not yet been widely adopted.

Tobacco use

There is also emerging evidence for the role of tobacco consumption as a risk factor for development of CSR. A meta-analysis involving nearly 28 000 patients with CSR and 105 000 controls found that patients who used tobacco products had 2.99 higher odds ($P<0.01$) of developing CSR. While the authors determined that this effect size is smaller than other risk factors typically associated with CSR (male sex, stress, corticosteroids, pregnancy, and sleep apnea), tobacco consumption is a modifiable risk factor which can be intervened upon [19]. This effect is attributed to the reduction in choroidal vascularity index seen on OCT in tobacco smokers, which in some studies appears to be dose dependent [20].

Clinical risk assessment

While the above are risk factors that can be reduced with intervention, intrinsic risk factors also contribute to chronic CSR. In directly comparing the patient populations with resolved versus chronic CSR, the chronic CSR patients had poorer visual acuity at presentation, longer duration of symptoms, and lower central macular thickness as well as reduced neurosensory detachment height [21]. The authors propose that the difference in OCT measurements are representative of defective RPE in the chronic CSR patient population. The Macula Society CSCR Study Group then used this data to develop a logistic regression model to predict likelihood of developing chronic CSR based on patient characteristics at initial presentation. In the regression model, only older age was a statistically significant risk, with a 22.3% increased odds of chronicity per year of age [21]. Investigations with larger patient populations are merited to see if these OCT trends hold both clinical and statistical significance, but for now, macular thickness and neurosensory detachment height may anecdotally guide physicians in triaging likelihood of chronicity and inform decisions for scheduling patient follow up or planning intervention.

MANAGEMENT OPTIONS

Topical NSAIDs

While topical NSAIDs may have a role in managing some ocular inflammatory diseases like cystoid macular edema, no benefit has been found for CSR. A meta-analysis involving 1001 eyes found no statistical significance in visual acuity improvement or subretinal fluid resolution at 3months following diagnosis in patients who did or did not take topical NSAIDs [22].

Mindfulness

CSR is frequently associated with psychological stress and an intense, driven personality, scenarios in which cortisol levels can be elevated. A randomized control trial including 60 patients explored the role of mindfulness as a viable intervention to manage initial presentation of acute CSR. Those patients assigned to the group who practiced mindfulness-based stress reduction had better BCVA and reduced mean central macular thickness than the control group at months 1, 3, and 6 of evaluation [23]. Mindfulness practices as an intervention for CSR offer the advantage of avoiding medication side effects or procedural risks. However, a larger study would be needed to adequately appreciate the potential benefit of this intervention in management of CSR.

Mineralocorticoid receptor antagonists

Innovations in imaging with OCT have illustrated the characteristic hyperpermeability and dilation of the choroid vasculature and atrophic changes within the inner choroid that is seen in CSR [24]. Genetic studies have hypothesized an association between variants in cadherin 5 expression, which controls cellular adhesion in the endothelium, and changes to the choriocapillaris resembling those seen in CSR upon exposure to steroids (a known risk factor for CSR) [25].

Further exploration of the glucocorticoid and mineralocorticoid pathways have suggested a role for mineralocorticoid receptor antagonists in treatment of CSR. A randomized control trial involving 60 patients with chronic CSR showed the spironolactone group had statistically significant improvement in BCVA at 1month after treatment initiation, while the control group did not. Both the spironolactone and eplerenone groups had reduction in subretinal fluid 1 month after treatment while the control group did not [26]. To further investigate this observed effect, a meta-analysis of the role of spironolactone and eplerenone in treatment of chronic CSR was undertaken using a population of 646 eyes. There was no statistically significant difference in BCVA between the mineralocorticoid receptor antagonist groups and the observation group at any of the follow up visits throughout the 1year after treatment initiation. The mineralocorticoid receptor antagonist group did have a reduction in subretinal fluid at 1 month compared to the observation group, but this effect was lost at later follow up checks, such that the observation group in fact had a higher rate of subretinal fluid resolution at 1year compared to the mineralocorticoid receptor antagonist group [27▪]. While there could be a small benefit for subretinal fluid reduction with short-term use of mineralocorticoid receptor antagonists in chronic CSR, this effect does not translate to a difference in BCVA. Further, these medications are not without risks, which include hyperkalemia, gynecomastia, and vaginal bleeding [28].

Photodynamic therapy

Photodynamic therapy has previously been adopted for treatment of chronic CSR. Activation of verteporfin causes choroidal vascular occlusion and achieves a reduction in choroidal hyperpermeability, a change notably found in eyes affected by CSR. However, full-dose and full-fluence PDT has been associated with dangerous side effects including juxtafoveal RPE atrophy [29], which could theoretically cause scarring and permanent decrease in BCVA. To avoid this risk, interest has been shifted to efficacy of half-dose and half-fluence PDT for management of chronic CSR. A meta-analysis of 561 eyes showed that there was no statistically significant difference in half-dose versus half-fluence PDT when comparing BCVA or retinal thickness during follow up [30]. Prior studies have demonstrated that BCVA improvement, SRF resolution, recurrence, and number of sessions needed to treat are comparable for half-dose, half-fluence, and standard PDT in chronic CSR [31]. Therefore, in those cases where PDT is considered for treatment, we suspect there will be a growing role for half-dose and half-fluence treatment, given the similar efficacy and associated reduction in risk.

Anti-vascular endothelial growth factor treatment

A meta-analysis of chronic CSR (defined as at least 3 months of symptoms prior to intervention) showed no statistically significant difference in BCVA following treatment with anti-VEGF. However, treated eyes did experience a significant decrease in central macular thickness [32]. Of note, this study excluded eyes with choroidal neovascularization, for which there would be clear benefit to use of anti-VEGF. The authors suggest that anti-VEGF could be considered an alternative to PDT for management of chronic CSR in settings where PDT is not readily available.

Given recent GWAS findings revealing the role of Tie-2 as protective against CSR, attention has turned to use of faricimab to increase Tie-2 expression by targeting its inhibitor Ang-2. A retrospective study including 16 eyes with chronic CSR showed that 14/16 patients had improvement in central macular thickness after treatment with faricimab [33]. Less (6/16) had visual acuity improvement of two or more lines, likely limited due to ellipsoid zone loss seen on OCT prior to initiation of the faricimab treatment.

Nearly half of these patients had received prior intervention with anti-VEGF or PDT, yet still had persistent sub-retinal fluid before treatment with faricimab. While the sample size of this study is small, it is a valuable first step in clinical applications of the recent genetic studies to further elucidate the CSR pathway.

In one patient with chronic CSR who failed treatment with multiple sessions of PDT, oral eplerenone, topical NSAIDs, intravitreal bevacizumab, intravitreal faricimab, and intravitreal aflibercept, resolution of her subretinal fluid occurred following a single off-label treatment with high-dose intravitreal aflibercept. At multiple points throughout her care, the absence of choroidal neovascularization was confirmed on fluorescein angiography [34]. The encouraging response seen here with use of high-dose aflibercept also merits further investigation in a larger population, especially given the potential for spontaneous resolution of CSR.

Laser treatment

Direct comparison of micropulse laser treatment to half-dose PDT in the PLACE trial has previously favored PDT, given a higher proportion of patients treated with PDT had complete resolution of subretinal fluid at their first and final evaluations [35]. Patients treated with PDT also had a statistically significant improvement in BCVA relative to the laser patients [35]. Laser treatment is still considered an alternative to PDT for managing macular edema, particularly in areas with a verteporfin shortage. A multicenter prospective study in Germany showed a statistically significant decrease in subretinal fluid at 3 months in eyes with chronic CSR that were treated with nanosecond subthreshold laser. 85% of patients had no residual SRF 1 year after treatment. BCVA however did not show any significant difference following treatment [36]. In a randomized control trial of 23 eyes, microsecond pulsing laser used to treat CSR eyes with choroidal neovascularization saw resolution of subretinal fluid in 61% of eyes and improvement in subretinal fluid in another 22% of eyes in the follow-up period of at least 6 months [37].

Focal thermal laser is another option for small, extrafoveal CNV due to CSR. Navigated laser photocoagulation may allow for more precise laser application to reduce risk of large scotoma and tends to only require a single treatment for resolution of CNV [38]. A study of 16 eyes with CSR complicated by CNV showed that SRF completely resolved in all eyes 1.1 months after treatment with navigated focal laser photocoagulation. Nine of the included eyes had been resistant to prior treatment attempts with anti-VEGF, PDT, and/or micropulse laser. In the 1-year follow-up period, there was no recurrence of SRF in any of the 16 eyes [38]. In larger studies of conventional laser photocoagulation, a similar effect of complete fluid resolution (evaluated 4 months after intervention) was upheld in all eyes treated with laser, while fluid recurrence was noted in the control group during this time [39].

While it is difficult to directly compare efficacy of different types of interventions, one network meta-analysis attempted to do so using a primary outcome of best corrected visual acuity (BCVA) 1 year after diagnosis. In comparing each intervention to the control group, none was statistically superior. However, surface under the cumulative ranking curve (SUCRA) ranked BCVA (in declining rank order) as best for those treated with less than 50% (low-dose) PDT, followed by antioxidant supplements, eplerenone, anti-VEGF, observation alone, more than 50% PDT, and finally pulsed laser (which the authors use to refer to subthreshold micropulse laser and selective retina therapy) [40▪].

CONCLUSION

We present a review of the most recent research on CSR disease mechanism and management. While investigation on this subject has generated much discussion, observation continues to be the mainstay of management for acute CSR. There is a paucity of support for statical or clinical superiority of other therapeutic interventions in comparison. Management of chronic CSR is more complex with potential benefit from PDT and insight to be gained on a molecular level from innovations in genomics analysis. Our understanding of the disease process will continue to evolve with further research efforts to study this complex disease.

Acknowledgements

None.

Financial support and sponsorship

M.R.R. is the recipient of the Vitreo-Retinal Surgery Foundation Research Award, supporting her research during 2023-2025.

Conflicts of interest

Disclosures for K.E.T.: 4DMT (consultant), Alimera (consultant), Abbvie (consultant), Apellis (consultant), Astellas (consultant, speaker's bureau), Bausch and Lomb (consultant), Eyepoint (consultant), Genentech (consultant, speaker's bureau), Harrow (consultant), Ocular Therapeutics (consultant), Outlook (consultant), Regeneron (consultant, research), Zeiss (consultant, research, speaker)

M.R.R. has no disclosures.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ■ of special interest
- ■■ of outstanding interest

1. Donald J, Gass M. Pathogenesis of disciform detachment of the neuroepithelium. Am J Ophthalmol 1967; 63:573/1–585/13.
2. Von Graefe A. About central recurrent retinitis. Graefes Arch Clin Exp Ophthalmol 1866; 12:211–215.
3. Bennett G. Central serous retinopathy. Br J Ophthalmol 1955; 39:605–618.
4. Kitzmann AS, Pulido JS, Diehl NN, *et al.* The incidence of central serous chorioretinopathy in Olmsted County, Minnesota, 1980-2002. Ophthalmology 2008; 115:169–173.
5. Baran N, Gürlü V, Esgin H. Long-term macular function in eyes with central serous chorioretinopathy. Clin Exp Ophthalmol 2005; 33:369–372.
6. Mrejen S, Balaratnasingam C, Kaden TR, *et al.* Long-term visual outcomes and causes of vision loss in chronic central serous chorioretinopathy. Ophthalmology 2019; 126:576–588.
7. Schellevis RL, Van Dijk EHC, Breukink MB, *et al.* Role of the complement system in chronic central serous chorioretinopathy: a genome-wide association study. JAMA Ophthalmol 2018; 136:1128–1136.
8. De Jong EK, Breukink MB, Schellevis RL, *et al.* Chronic central serous chorioretinopathy is associated with genetic variants implicated in age-related macular degeneration. Ophthalmology 2015; 122:562–570.
9. ■ Rämö JT, Abner E, Van Dijk EHC, *et al.* Overlap of genetic loci for central serous chorioretinopathy with age-related macular degeneration. JAMA Ophthalmol 2023; 141:449–457.

This meta-analysis compares 1176 CSR patients to controls, confirming shared genetic risk loci with AMD and the inverse relationship of complement pathway expression in CSR relative to AMD.

10. Akiyama M, Miyake M, Momozawa Y, *et al.* Genome-wide association study of age-related macular degeneration reveals 2 new loci implying shared genetic components with central serous chorioretinopathy. Ophthalmology 2023; 130:361–372.
11. Hosoda Y, Miyake M, Schellevis RL, *et al.* Genome-wide association analyses identify two susceptibility loci for pachychoroid disease central serous chorioretinopathy. Commun Biol 2019; 2:468.
12. Rämö JT, Gorman BR, Weng LC, *et al.* Rare genetic variation in PTPRB is associated with central serous chorioretinopathy, varicose veins and glaucoma. Nat Commun 2025; 16:4127.
13. Mori Y, Van Dijk EHC, Miyake M, *et al.* Genome-wide association and multiomics analyses provide insights into the disease mechanisms of central serous chorioretinopathy. Sci Rep 2025; 15:9158.
14. Kiilgaard HC, Nissen AHK, Balaratnasingam C, *et al.* Diagnostic accuracy of OCT angiography for macular neovascularization in central serous chorioretinopathy: a systematic review and meta-analysis. Acta Ophthalmol 2024; 102:749–758.
15. Hanumunthadu D, Van Dijk EC, Dumpala S, *et al.* Evaluation of choroidal layer thickness in central serous chorioretinopathy. J Ophthalmic Vis Res 2019; 14: 164–170.
16. Shojaeinia M, Hosseini A, Naderi M, *et al.* A comprehensive overview: deep learning approaches to central serous chorioretinopathy diagnosis. BMC Ophthalmol 2025; 25:549.
17. Wu CY, Riangwiwat T, Rattanawong P, *et al.* Association of obstructive sleep apnea with central serous chorioretinopathy and choroidal thickness: a systematic review and meta-analysis. Retina 2018; 38:1642–1651.
18. Bulloch G, Seth I, Zhu Z, *et al.* Ocular manifestations of obstructive sleep apnea: a systematic review and meta-analysis. Graefes Arch Clin Exp Ophthalmol 2024; 262:19–32.
19. Fakhril-Din Z, Arnold-Vangsted A, Boberg-Ans LC, *et al.* Is tobacco consumption a risk factor for central serous chorioretinopathy? A systematic review and meta-analysis. Acta Ophthalmol 2025; 103:136–145.
20. Wei X, Kumar S, Ding J, *et al.* Choroidal structural changes in smokers measured using choroidal vascularity index. Invest Ophthalmol Vis Sci 2019; 60:1316–1320.
21. Gandhi P, Hasan N, Gidwani K, *et al.* Predictors of persistent central serous chorioretinopathy: a multicenter retrospective study – MICRoN report number four. Graefes Arch Clin Exp Ophthalmol 2025; 1–8.
22. Larsson JME, Boberg-Ans LC, Vangsted A, *et al.* Topical nonsteroidal anti-inflammatory drugs for central serous chorioretinopathy: a systematic review and meta-analysis. Acta Ophthalmol 2024; 102:274–284.
23. Özcan D, Karapapak M. Effect of mindfulness-based stress reduction on acute central serous chorioretinopathy: a randomized control trial. Int Ophthalmol 2024; 44:183.
24. Yang L, Jonas JB, Wei W. Optical coherence tomography–assisted enhanced depth imaging of central serous chorioretinopathy. Invest Ophthalmol Vis Sci 2013; 54:4659–4665.
25. Schubert C, Pryds A, Zeng S, *et al.* Cadherin 5 is regulated by corticosteroids and associated with central serous chorioretinopathy. Hum Mutat 2014; 35: 859–867.
26. Pichi F, Carrai P, Ciardella A, *et al.* The Central Serous Chorioretinopathy Study Group. Comparison of two mineralcorticosteroids receptor antagonists for the treatment of central serous chorioretinopathy. Int Ophthalmol 2017; 37: 1115–1125.
27. ■ Huang RS, Mihalache A, Benour A, *et al.* Eplerenone and spironolactone for chronic central serous chorioretinopathy: a systematic review and meta-analysis. Am J Ophthalmol 2025; 278:22–37.

This meta-analysis investigates a population of more than 600 eyes, determining that there is no difference in BCVA at follow up time points for eyes treated with mineralocorticoid receptor antagonists versus observation. While there was an initial statistically significant reduction in SRF at 1 month for treated eyes, at 1 year, the observation group had a statistically signification reduction in SRF compared to the treatment group. Thus, while there may be a short-term benefit to use of mineralocorticoid receptor antagonists, this did not translate to an improvement in BCVA and did not persist at extended follow up.

28. Lainscak M, Pelliccia F, Rosano G, *et al.* Safety profile of mineralocorticoid receptor antagonists: spironolactone and eplerenone. Int J Cardiol 2015; 200: 25–29.
29. Postelmans L, Pasteels B, Coquelet P, *et al.* Severe pigment epithelial alterations in the treatment area following photodynamic therapy for classic choroidal neovascularization in young females. Am J Ophthalmol 2004; 138: 803–808.
30. Zaman M, Mihalache A, Huang RS, *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: 233–242.
31. Altinel MG, Kanra AY, Totuk OMG, *et al.* Comparison of half-dose versus half-fluence versus standard photodynamic therapy in chronic central serous chorioretinopathy. Photodiagnosis Photodyn Ther 2021; 33:102081.
32. Palakkamanil M, Munro M, Sethi A, Adatia F. Intravitreal antivascular endothelial growth factor for the treatment of chronic central serous retinopathy: a meta-analysis of the literature. BMJ Open Ophth 2023; 8:e001310.
33. Rämö JT, Kim LA, Stryjewski T, *et al.* Targeting the Tie-2 receptor with faricimab in central serous chorioretinopathy: a case series motivated by a genetic finding. Am J Ophthalmol 2025; 269:246–254.
34. Lin JB, Leung LSB. Resolution of subretinal fluid in intractable central serous chorioretinopathy with high-dose intravitreal aflibercept. Am J Ophthalmol Case Rep 2025; 37:102266.
35. Van Dijk EHC, Fauser S, Breukink MB, *et al.* Half-dose photodynamic therapy versus high-density subthreshold micropulse laser treatment in patients with chronic central serous chorioretinopathy. Ophthalmology 2018; 125: 1547–1555.
36. Fraenkel D, Kaymak H, Hartmann M, *et al.* Subthreshold nanosecond laser therapy for chronic central serous chorioretinopathy: a prospective study. J Fr Ophtalmol 2025; 48:104372.
37. Maltsev DS, Kulikov AN, Vasiliev AS, *et al.* Microsecond pulsing laser for choroidal neovascularization associated with central serous chorioretinopathy. Curr Eye Res 2025; 50:304–313.
38. Maltsev DS, Kulikov AN, Vasiliev AS, *et al.* Direct navigated focal laser photocoagulation of choroidal neovascularization in central serous chorioretinopathy. Lasers Med Sci 2025; 40:173.
39. Burumcek E, Mudun A, Karacorlu S, *et al.* Laser photocoagulation for persistent central serous retinopathy. Ophthalmology 1997; 104:616–622.
40. ■ Lange CA, Qureshi R, Pauleikhoff L. Interventions for central serous chorioretinopathy: a network meta-analysis. Cochrane Central Editorial Service, editor. Cochrane Database Syst Rev 2025; 1–255.

This large network meta-analysis included more than 4000 patients and directly compared several interventions for CSR including PDT, anti-VEGF, and laser, finding no significant difference between these interventions and final BCVA.