

VISION ACADEMY VIEWPOINT

The Vision Academy is a partnership between Bayer and ophthalmic specialists, established with the aim of addressing key clinical challenges in the field of retinal diseases: www.visionacademy.org.

Treatment of Central Serous Chorioretinopathy: New Options for an Old Disease

Background

Central serous chorioretinopathy (CSC) is a common disease that leads to vision loss, primarily affecting people of working age.¹ It is six times more common in men than women and can involve both eyes in up to 40% of cases, although bilateral involvement at diagnosis is around 4%.^{1,2} Advances in genetics and ocular imaging have improved the understanding of the pathophysiology of CSC, and growing evidence from randomized controlled trials and large, retrospective, non-randomized treatment studies can shape treatment recommendations.^{3,4}

A review of the literature and available evidence was conducted to:

- Provide an overview of the risk factors and pathogenesis of CSC
- Propose an evidence-based flowchart containing recommendations for practical disease management and the treatment of phenotypic variations

Endorsed by the
Vision Academy in July 2025

Viewpoint

CSC results from choroidal dysfunction, leading to ischemia and oxidative stress, which disrupts retinal pigment epithelial (RPE) function and causes choroidal thickening and subretinal fluid accumulation.^{4,5} RPE dysfunction may be due to microcirculation abnormalities in choroidal capillaries, which can contribute to disease recurrence and permanent tissue changes.⁶⁻⁸ Corticosteroids may also be involved in CSC pathogenesis due to their role in choroidal vasodilation and vessel hyperpermeability.⁹

CSC is linked to systemic and local corticosteroid use, as well as excessive endogenous corticosteroid production.⁵ Other contributing factors include sympathetic overactivation and decreased parasympathetic activity, obstructive sleep apnea, hyperopia, infections (e.g., *Helicobacter pylori*), and potentially some medications (e.g., sympathomimetic agents and phosphodiesterase-5 inhibitors).¹⁰⁻¹³ Psychological stress, personality traits, and genetic variations in *CFH*, *VIPR2*, and *CDH5* may also influence CSC susceptibility, but further research is needed.^{4,10,14-18}

Knowledge of the updated evidence for the pathogenesis of CSC, as well as risk factors for the disease and treatment outcomes, will provide clinicians with pathophysiology-based treatment guidance. Following a review of the literature, a set of recommendations for the management of CSC was developed.

Step 1: Confirm the diagnosis of CSC and reduce modifiable risk factors

Confirm the diagnosis by identifying key features of CSC through multimodal imaging. Once confirmed, identify and address modifiable risk factors by collaborating with physicians.

Step 2: Determine subfoveal or extrafoveal involvement and duration of symptoms

Assess patients with CSC for subfoveal or extrafoveal involvement and determine whether the disease is acute (<3 months) or chronic (≥3 months), as these factors can be used to guide treatment decisions.

Step 3: Consider disease characteristics

For acute or chronic CSC with extrafoveal involvement only and no focal leakage, observe for up to 3–4 months to monitor for spontaneous resolution or start treatment immediately (see Step 4 below). Acute subfoveal CSC should also be monitored, but treatment earlier than 3–4 months may be needed if no improvement is seen on serial optical coherence tomography, due to risk of potential vision loss from persistent subretinal fluid. Early treatment should also be considered in patients with reduced visual acuity, visual disturbances, recurrent CSC episodes, cystoid macular changes or RPE atrophy, photoreceptor damage, or CSC in their only or better-seeing eye, or in cases of patient preference for early treatment.

Vision Academy Viewpoints are intended to raise awareness of a clinical challenge within ophthalmology and provide an expert opinion to engage in further discussion.

They can be downloaded from <https://www.visionacademy.org/resource-zone/resources/all>

The Vision Academy is a group of over 80 international experts who, through their collective expertise, provide consensus guidance for managing clinically challenging situations, especially in areas of controversy or with insufficient conclusive evidence.

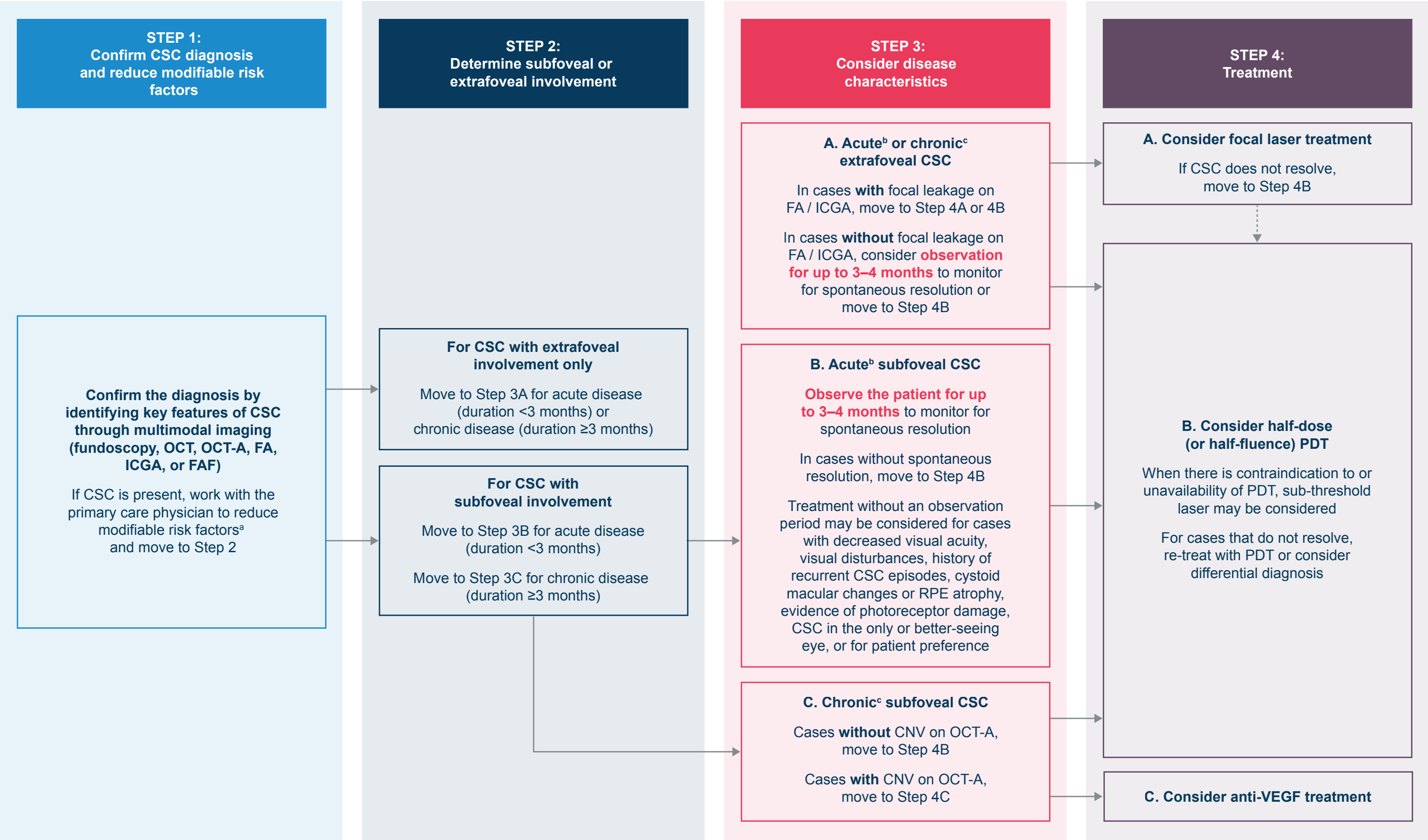
The Vision Academy is funded and facilitated by Bayer. This document is based on the Vision Academy publication: Kim YJ *et al.* *Eye* 2025; Epub ahead of print. doi: 10.1038/s41433-025-03894-z developed by Yoon Jeon Kim, Sobha Sivaprasad, Tariq Aslam, Polona Jaki Mekjavić, Vilma Jūratė Balčiūnienė, Linda Visser, Antonia M. Jousen, Young Hee Yoon, Timothy Y.Y. Lai, and Annabelle A. Okada. Medical writing and editorial support was provided by Caudex, an IPG Health Company, and funded by Bayer.

The opinions expressed, and guidance laid out, by the Vision Academy are developed independently by the members and do not necessarily reflect the opinions of Bayer.

Intended for healthcare professionals only. Always refer to local treatment guidelines and relevant prescribing information.

July 2025 | MA-PFM-OPHT-ALL-1431

Figure. Vision Academy recommendations for the management and treatment of CSC



^aModifiable risk factors may include the use of steroid cream or other drugs, or psychological or physical stress.
^bAcute CSC is defined as CSC present for <3 months.
^cChronic CSC is defined as CSC present for ≥3 months.

CNV, choroidal neovascularization; CSC, central serous chorioretinopathy; FA, fluorescein angiography; FAF, fundus fluorescein angiography; ICGA, indocyanine green angiography; OCT, optical coherence tomography; OCT-A, optical coherence tomography angiography; PDT, photodynamic therapy; RPE, retinal pigment epithelial; VEGF, vascular endothelial growth factor.

Figure reproduced from Figure 5 in Kim, Y.J., Sivaprasad, S., Aslam, T. *et al.* Treatment of central serous chorioretinopathy: new options for an old disease. *Eye* (2025). <https://doi.org/10.1038/s41433-025-03894-z>. Reproduced under Creative Commons license BY 4.0, <https://creativecommons.org/licenses/by/4.0/>.

References

1. Daruich A, Matet A, Dirani A *et al*. Central serous chorioretinopathy: recent findings and new pathophysiology hypothesis. *Prog Retin Eye Res* 2015; 48: 82–118.
2. Ross A, Ross AH, Mohamed Q. Review and update of central serous chorioretinopathy. *Curr Opin Ophthalmol* 2011; 22 (3): 166–173.
3. van Rijnssen TJ, van Dijk EHC, Yzer S *et al*. Central serous chorioretinopathy: towards an evidence-based treatment guideline. *Prog Retin Eye Res* 2019; 73: 100770.
4. Kaye R, Chandra S, Sheth J *et al*. Central serous chorioretinopathy: an update on risk factors, pathophysiology and imaging modalities. *Prog Retin Eye Res* 2020; 79: 100865.
5. Saito M, Saito W, Hirooka K *et al*. Pulse waveform changes in macular choroidal hemodynamics with regression of acute central serous chorioretinopathy. *Invest Ophthalmol Vis Sci* 2015; 56 (11): 6515–6522.
6. Gass JD. Pathogenesis of disciform detachment of the neuroepithelium. *Am J Ophthalmol* 1967; 63 (Suppl): 1–139.
7. Lam DSC, Boon CJF, Radke NV *et al*. Central serous chorioretinopathy. In: Sadda SR, Schachar AP, Wilkinson CP *et al*. (eds). *Ryan's Retina*. 7th edn. Elsevier 2022; 1530–1554.
8. Ten Berge JCEM, van Dijk EHC, Schreurs MWJ *et al*. Antiretinal antibodies in central serous chorioretinopathy: prevalence and clinical implications. *Acta Ophthalmol* 2018; 96 (1): 56–62.
9. Zhao M, Célérier I, Bousquet E *et al*. Mineralocorticoid receptor is involved in rat and human ocular chorioretinopathy. *J Clin Invest* 2012; 122 (7): 2672–2679.
10. Chatziralli I, Kabanarou SA, Parikakis E *et al*. Risk factors for central serous chorioretinopathy: multivariate approach in a case-control study. *Curr Eye Res* 2017; 42 (7): 1069–1073.
11. Dang Y, Mu Y, Zhao M *et al*. The effect of eradicating *Helicobacter pylori* on idiopathic central serous chorioretinopathy patients. *Ther Clin Risk Manag* 2013; 9: 355–360.
12. Semeraro F, Morescalchi F, Russo A *et al*. Central serous chorioretinopathy: pathogenesis and management. *Clin Ophthalmol* 2019; 13: 2341–2352.
13. Smal C, Lepière G, Bonnet S. [Central serous chorioretinopathy following the use of phosphodiesterase 5 inhibitors.] *Rev Med Liege* 2017; 72 (11): 475–477.
14. Fok ACT, Chan PPM, Lam DSC *et al*. Risk factors for recurrence of serous macular detachment in untreated patients with central serous chorioretinopathy. *Ophthalmic Res* 2011; 46 (3): 160–163.
15. Genovese G, Meduri A, Muscatello MRA *et al*. Central serous chorioretinopathy and personality characteristics: a systematic review of scientific evidence over the last 10 years (2010 to 2020). *Medicina (Kaunas)* 2021; 57 (6): 628.
16. van Haalen FM, van Dijk EHC, Andela CD *et al*. Maladaptive personality traits, psychological morbidity and coping strategies in chronic central serous chorioretinopathy. *Acta Ophthalmol* 2019; 97 (4): e572–e579.
17. Moschos MM, Gazouli M, Gatzoufas Z *et al*. Prevalence of the complement factor H and *GSTM1* genes polymorphisms in patients with central serous chorioretinopathy. *Retina* 2016; 36 (2): 402–407.
18. Hosoda Y, Yoshikawa M, Miyake M *et al*. *CFH* and *VIPR2* as susceptibility loci in choroidal thickness and pachychoroid disease central serous chorioretinopathy. *Proc Natl Acad Sci U S A* 2018; 115 (24): 6261–6266.
19. van Rijnssen TJ, van Dijk EHC, Scholz P *et al*. Focal and diffuse chronic central serous chorioretinopathy treated with half-dose photodynamic therapy or subthreshold micropulse laser: PLACE trial report no. 3. *Am J Ophthalmol* 2019; 205: 1–10.

Step 4: Treatment

For acute or chronic CSC with extrafoveal involvement only, focal laser or half-dose or half-fluence photodynamic therapy (PDT) is recommended if focal leakage is detected; otherwise, observation for spontaneous resolution for 3–4 months may be considered. For subfoveal involvement without type 1 choroidal neovascularization, half-dose or half-fluence PDT is recommended, with possible initial observation (3–4 months) in acute cases. Sub-threshold laser treatment may be an option for patients with extensive RPE damage, previous poor PDT response, contraindication to PDT, or unavailability of PDT. In cases of chronic subfoveal CSC with type 1 choroidal neovascularization, anti-vascular endothelial growth factor therapy can be used.

Further considerations

Optimal treatment timing and treatment options for CSC depend on a patient's baseline clinical characteristics. Half-dose or half-fluence PDT is the treatment of choice for chronic CSC based on efficacy and safety data, including findings from the PLACE trial.¹⁹ However, further research is needed to understand subtype-specific pathophysiology and treatment outcomes. Large, multicenter, randomized trials will help to clarify long-term PDT outcomes and compare available treatment options.