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Treatment of central serous chorioretinopathy: new options for an old disease

Based on the Vision Academy publication: Kim YJ *et al*. *Eye* 2025; 39: 2375–2388. This presentation is based on material developed by the Vision Academy members and does not necessarily reflect the views of Bayer. Medical writing and editorial support was provided by Caudex, an IPG Health Company, and funded by Bayer.
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This presentation is based on the Vision Academy publication: Kim YJ *et al. Eye* 2025; 39: 2375–2388 and is intended for healthcare professionals. 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.

Contents

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CSC, central serous chorioretinopathy.

Objectives

To provide an overview of the risk factors and pathogenesis of CSC

To summarize published evidence for the treatment of CSC

To develop an algorithm with recommendations for practical disease management and the treatment of phenotypic variations

QUESTION

How can we optimize current practice for the management and treatment of phenotypic variations of CSC?

CSC, central serous chorioretinopathy.

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Background

Central serous chorioretinopathy (CSC)

CSC:

- Is a common chorioretinal disease that causes vision loss
- Predominantly affects people of working age
- Is six times more common in men than in women[1]

Reported cases of CSC involving both eyes are as high as 40%, and rate of bilateral involvement at time of initial diagnosis is ~4%[2]

Recent developments in genetics and ocular imaging have improved understanding of CSC pathophysiology

The evidence base is growing from randomized controlled trials (e.g., PLACE) and large, retrospective, non-randomized treatment studies[3,4]

?

CHALLENGE REQUIRING VISION ACADEMY GUIDANCE

Guidance is needed for the management and treatment of phenotypic variations of CSC in clinical practice

RECOMMENDATION

CSC, central serous chorioretinopathy; PLACE; Half-Dose Photodynamic Therapy versus High-Density Subthreshold Micropulse Laser Treatment in Patients with Chronic Central Serous Chorioretinopathy trial.

1. Daruich A *et al. Prog Retin Eye Res* 2015; 48: 82–118; 2. Ross A *et al. Curr Opin Ophthalmol* 2011; 22: 166–173; 3. van Rijssen TJ *et al. Prog Retin Eye Res* 2019; 73: 100770; 4. Kaye R *et al. Prog Retin Eye Res* 2020; 79: 100865.

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Clinical challenges

Clinical challenges requiring guidance

Click on a section

Evidence on clinical characteristics and treatment

What evidence is available regarding the pathogenesis, risk factors, clinical characteristics, and treatment of CSC?

Practical recommendations

How can we use this evidence to provide recommendations for the management and treatment of phenotypic variations of CSC in clinical practice?

CSC, central serous chorioretinopathy.

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Pathogenesis of CSC, risk factors, and clinical characteristics

Pathogenesis of CSC

Choroidal dysfunction

- Increased choroidal thickness and choroidal vessel dilation are widely accepted as characteristics of CSC
- CSC is thought to result from abnormal choroidal blood flow regulation, causing ischemia at the choriocapillaris,[1] which results in RPE dysfunction and SRF accumulation[2]
- Pro-inflammatory factor production and hyperpermeability due to oxidative stress cause choroidal thickening and can lead to subretinal material deposition[2]
- Compared with controls, patients with chronic CSC have a higher choroidal vascularity index,[3,4] a reduced stromal area to choroidal area ratio,[5] and enlarged choroidal vessels[6]
- Studies indicate a relationship between CSC and pachychoroid spectrum diseases, in which choroidal thickening is key[7]

RPE dysfunction

- While the role of the RPE is not well understood, it is believed that microcirculation abnormalities in choroidal capillaries lead to increased fluid leakage at the RPE into the subretinal space[8]
- Damaged RPE cells in areas of leakage may overburden the RPE, causing serous fluid persistence[2,9]
- Continuous insufficient microcirculation may result in disease recurrence and permanent tissue damage[10]

The corticosteroid hypothesis

- In rats, choroidal vasodilation, and other features similar to CSC, occurred after intravitreal aldosterone injection[11]
- Analysis of SRF samples from patients with CSC vs controls (with rhegmatogenous retinal detachment) suggested dysregulation of the alternative complement pathway, glucocorticoid, and mineralocorticoid systems[12]

CSC, central serous chorioretinopathy; RPE, retinal pigment epithelium; SRF, subretinal fluid.

1. Saito M *et al. Invest Ophthalmol Vis Sci* 2015; 56 (11): 6515–6522; 2. Kaye R *et al. Prog Retin Eye Res* 2020; 79: 100865; 3. Agrawal R *et al. Retina* 2016; 36 (9): 1646–1651; 4. Kim RY *et al. Retina* 2020; 40: 1395–1402; 5. Lee M *et al. Invest Ophthalmol Vis Sci* 2018; 59, 4896–4908; 6. Aslam TM *et al. Retina* 2022; 42: 88–94; 7. Castro-Navarro V *et al. Graefes Arch Clin Exp Ophthalmol* 2021; 259 (6): 1385–1400; 8. Gass JD. *Am J Ophthalmol* 1967; 63 (3): Suppl 1–139; 9. Lam DSC *et al*. In: Sadda SR, Schachat AP, Wilkinson CP *et al*. (eds). Ryan's Retina. 7th edn. (Elsevier, 2023) 1530–1554; 10. ten Berge JCEM *et al. Acta Ophthalmol* 2018; 96: 56–62; 11. Zhao M *et al. J Clin Invest* 2012; 122 (7): 2672–2679; 12. Kowalczuk L *et al. Transl Vis Sci Technol* 2018; 7: 3.

Risk factors for CSC

Category	Risk factors	Key considerations
Corticosteroid use	Systemic corticosteroid use (oral, intravenous), local corticosteroid administration (inhaled, intranasal, epidural, intra-articular, topical, periocular)[1]	Steroid-induced CSC shows frequent bilaterality, atypical presentation, and less male predilection than non–steroid-induced CSC[1]
Endogenous hormonal changes	Endogenous hypercortisolism (Cushing's syndrome, adrenal / pituitary tumors, ectopic adrenocorticotropic hormone-secreting tumors),[2] pregnancy-related hormonal changes (elevated cortisol, progesterone, testosterone),[3] renin–angiotensin system activation, increased blood volume[2]	CSC is the leading cause of pregnancy-related acquired retinal / choroidal visual impairment[3]
Autonomic dysfunction and sleep disorders	Sympathetic overactivation,[4] decreased parasympathetic activity,[5] obstructive sleep apnea (5 × increased risk)[4]	Association between CSC and autonomic imbalance;[4] sleep apnea screening is recommended in patients with CSC[6]
Infectious and drug-related factors	*Helicobacter pylori* infection, sympathomimetic agents (pseudoephedrine, oxymetazoline), MMDA,[7] phosphodiesterase-5 inhibitors (sildenafil, tadalafil)[8]	Pathophysiological mechanisms of *H. pylori* in CSC remain unclear;[9] drug-induced CSC should be considered in at-risk patients[10,11]
Psychological and personality factors	Psychological stress, Type A personality,[4] antipsychotic medication use, depression, adjustment disorder[12]	Recent studies using validated questionnaires found no clear association between CSC and maladaptive personality traits[13]
Genetic risk factors	Single-nucleotide polymorphisms in *CFH* (rs3753394, rs1329428, rs800292),[14,15] *VIPR2* (rs3793217),[16] *CDH5*,[2] *TNFRSF10A-LOC389641*, *GATA5*[17]	Genetic variations may influence choroidal circulation and steroid response, but functional impacts require further research[15]

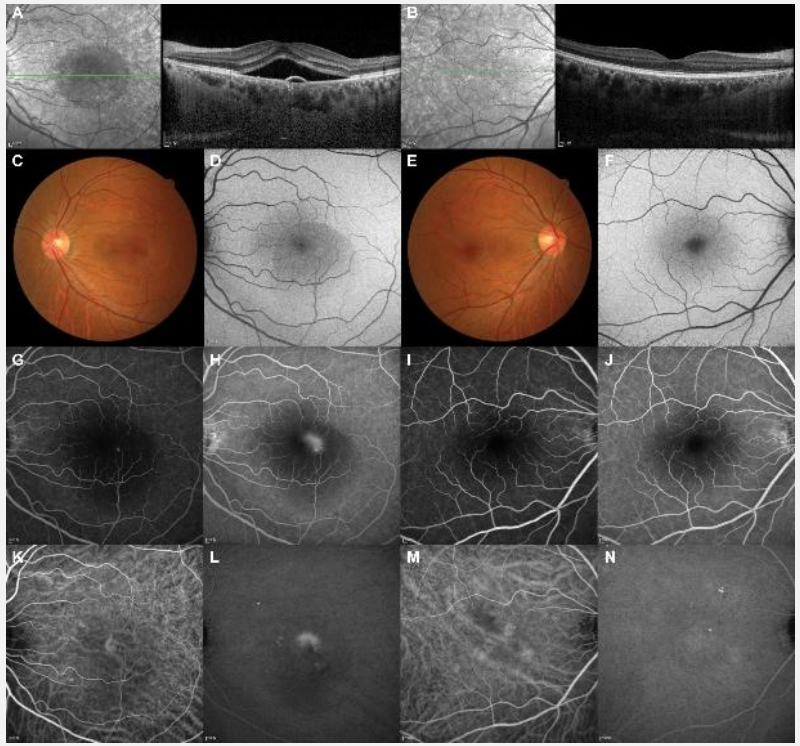
CSC, central serous chorioretinopathy; CFH, complement factor H; MMDA, 3-methoxy-4,5-methylenedioxyamphetamine.

1. Nicholson BP *et al. Surv Ophthalmol* 2018; 63 (1): 1–8; 2. Kaye R *et al. Prog Retin Eye Res* 2020; 79: 100865; 3. Park YJ *et al. Korean J Ophthalmol* 2017; 31 (4): 320–327; 4. Chatziralli I *et al. Curr Eye Res* 2017; 42 (7): 1069–1073; 5. Tewari HK *et al. Invest Ophthalmol Vis Sci* 2006; 47 (8): 3474–3478; 6. Pan CK *et al. Am J Ophthalmol* 2020; 218: 148–155; 7. Semeraro F *et al. Clin Ophthalmol* 2019; 13: 2341–2352; 8. Smal C *et al. Rev Med Liege* 2017; 72 (11): 475–477; 9. Dang Y *et al. Ther Clin Risk Manag* 2013; 9: 355–360; 10. van Rijssen TJ *et al. Prog Retin Eye Res* 2019; 73: 100770; 11. Loo RH *et al. Retina* 2002; 22 (1): 19–24; 12. Genovese G *et al. Medicina (Kaunas)* 2021; 57: 628; 13. van Haalen FM *et al. Acta Ophthalmol* 2019; 97: e572–e579; 14. de Jong EK *et al. Ophthalmology* 2015; 122 (3): 562–570; 15. Moschos MM *et al. Retina* 2016; 36 (2): 402–407; 16. Hosoda Y *et al. Proc Natl Acad Sci U S A* 2018; 115 (24): 6261–6266; 17. Hosoda Y *et al. Commun Biol* 2019; 2: 468.

Clinical characteristics of acute and chronic CSC

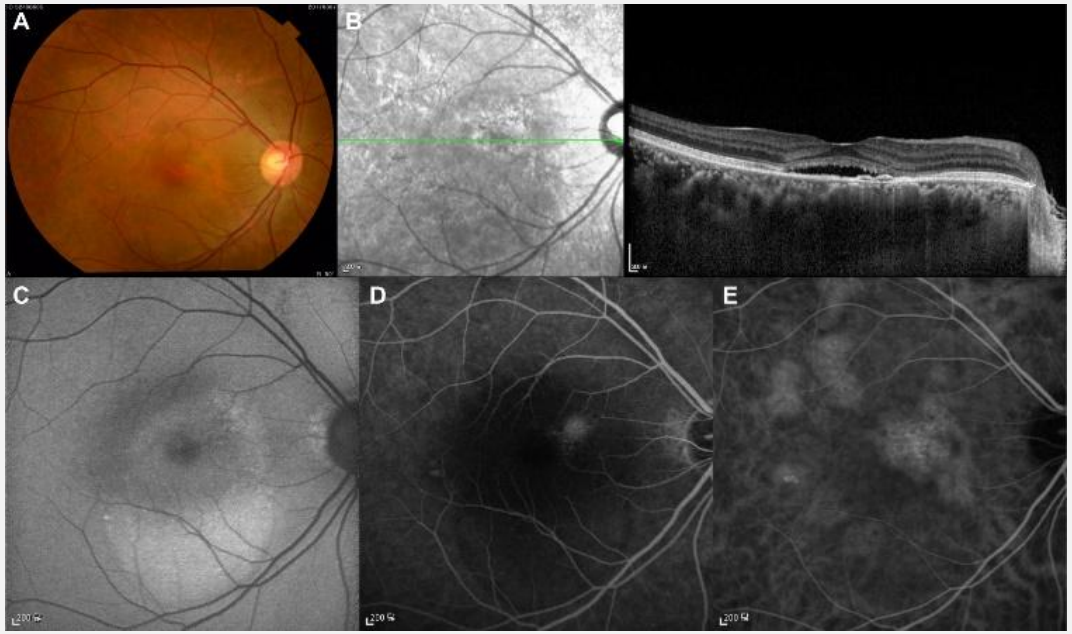
Acute CSC

- Characterized by acute-onset accumulation of SRF (of 3–4 months' duration) and/or PED with a good visual prognosis[1]
- Resolves spontaneously in the majority of cases[2,3]



Chronic CSC

- Characterized by SRF with various levels of PED and/or RPE decompensation secondary to choroidal abnormalities[1-3]
- Patients typically have persistent SRF for >4–6 months[1]
- Widespread RPE decompensation with changes on fundus autofluorescence is characteristic[3]
- Risk factors for prolonged CSC duration include subfoveal choroidal thickness ≥500 µm, PED height ≥50 µm, and age ≥40 years[4]
- Recurrences are common, particularly in cases with no intervention, and range from 15–51%, depending on study design and follow-up periods[5]



CSC, central serous chorioretinopathy; PED, pigment epithelial detachment; RPE, retinal pigment epithelial; SRF, subretinal fluid.

1. Daruich A *et al. Prog Retin Eye Res* 2015; 48: 82–118; 2. van Rijssen TJ *et al. Prog Retin Eye Res* 2019; 73: 100770; 3. Kaye R *et al. Prog Retin Eye Res* 2020; 79: 100865;
4. Daruich A *et al. Retina* 2017; 37 (10): 1905–1915; 5. Loo RH *et al. Retina* 2002; 22 (1): 19–24.

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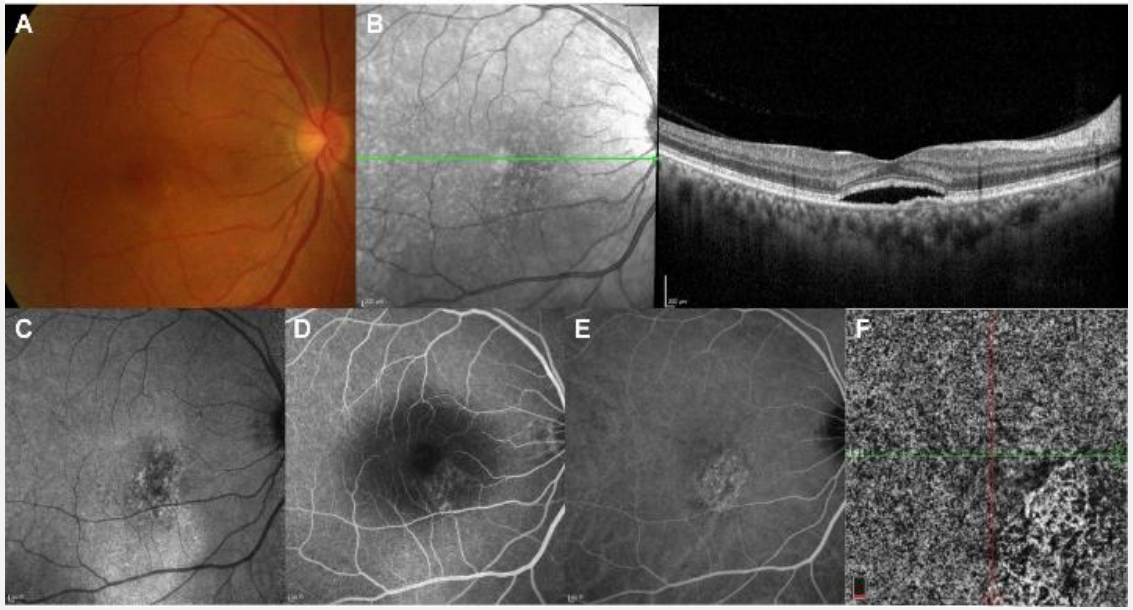
Clinical characteristics of CSC with CNV and bullous CSC

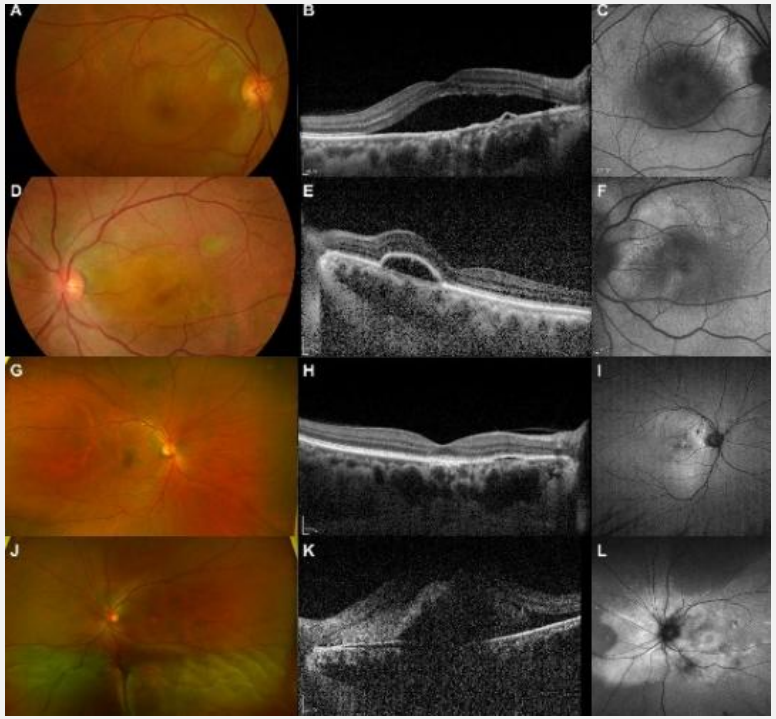
CSC with CNV

- A subset of patients with chronic CSC can develop CNV, primarily type 1 macular neovascularization[1]
- Clinical overlap between CSC with CNV and pachychoroid neovasculopathy exists, with recent studies suggesting that they may ultimately be the same disease[2]
- In a retrospective case series, CNV was present in the clinical course of 2–4% of chronic CSC cases[3]
- CNV is significantly associated with chronic CSC, choroidal vascular hyperpermeability, and choriocapillary hypoperfusion[4]
- Possible risk factors for type 1 CNV secondary to CSC are older age, hypertension, pigmentary changes, and double-layer sign[4,5]



Bullous CSC

- Multifocal posterior pigment epitheliopathy associated with exudative retinal detachment in CSC, or bullous CSC, is a rare disease variant characterized by severe serous retinal detachment[6]
- RPE tears are very common in this form of CSC[6]



CNV, choroidal neovascularization; CSC, central serous chorioretinopathy; RPE, retinal pigment epithelial.

1. Shiragami C *et al. Am J Ophthalmol* 2018; 193: 80–86; 2. Siedlecki J *et al. Ophthalmol Retina* 2022; 6 (9): 807–813; 3. Loo RH *et al. Retina* 2002; 22 (1): 19–24; 4. Yeo JH *et al. J Ophthalmol* 2020: 7217906; 5. Lee GI *et al. Sci Rep* 2019; 9: 3927; 6. Kaye R *et al. Prog Retin Eye Res* 2020; 79: 100865.

Differential diagnosis of CSC

Category	Conditions	Key differentiation
Pachychoroid-associated diseases	Pachychoroid pigment epitheliopathy, peripapillary pachychoroid syndrome, PCV, CNV with increased choroidal thickness, focal choroidal excavation,[1] peripheral exudative hemorrhagic chorioretinopathy[2]	PCV shows a branching neovascular network on ICGA; CSC exhibits focal leakage without neovascularization[2]
Neovascular diseases	AMD with type 1 macular neovascularization[3]	AMD and PCV show persistent leakage on FA[4]
Inflammatory diseases	VKH disease,[5] posterior scleritis[6]	VKH disease is always bilateral, and patients may recall systemic prodromal symptoms. In the acute phase, the choroid is diffusely and massively thickened to an extent not seen with CSC, and anterior chamber inflammatory cells may be observed. VKH disease presenting in the chronic phase may show severe anterior segment inflammation, with posterior iris synechia and vitritis[5] Posterior scleritis can be distinguished by B-mode ultrasonographic findings[6]
Choroidal tumors and systemic malignancies	Choroidal melanoma,[7] choroidal hemangioma,[8] metastases, leukemia infiltration, Waldenström's macroglobulinemia, choroidal lymphoma, bilateral diffuse uveal melanocytic proliferation, paraneoplastic vitelliform maculopathy[3]	Tumors and hematological malignancies can be identified using ultrasound,[8] angiography, computed tomography / magnetic resonance imaging, and systemic evaluations[3]
Genetic and developmental disorders	Best vitelliform macular dystrophy, RP1L1-associated occult macular dystrophy, central areolar choroidal dystrophy, optic pit maculopathy, uveal effusion syndrome, tilted disc syndrome, dome-shaped macula[3]	Genetic disorders show family history, absence of leakage on FA, and distinct patterns on OCT; developmental anomalies present with structural variations on OCT and ultrasound[3]
Drug-induced serous retinopathy	MAPK inhibitor-associated retinopathy,[9] FGFR inhibitor-related CSC[10]	History of systemic therapy with MAPK or FGFR inhibitors and absence of choroidal thickening help differentiate drug-induced cases from CSC[9,10]

AMD, age-related macular degeneration; CNV, choroidal neovascularization; CSC, central serous chorioretinopathy; FA, fluorescein angiography; FGFR, fibroblast growth factor receptor; ICGA, indocyanine green angiography; MAPK, mitogen-activated protein kinase; OCT, optical coherence tomography; PCV, polypoidal choroidal vasculopathy; RP1L1, retinitis pigmentosa 1–like 1; VKH, Vogt-Koyanagi-Harada.

1. Borooah S *et al. Acta Ophthalmol* 2021; 99 (6): e806–e822; 2. Shroff D *et al. Retina* 2021; 41 (7): 1518–1525; 3. van Dijk EHC *et al. Prog Retin Eye Res* 2021; 84: 100955; 4. Lee GI *et al. Sci Rep* 2019; 9: 3927; 5. O'Keefe GAD, Rao NA. *Surv Ophthalmol* 2017; 62 (1): 1–25; 6. Dong ZZ *et al. Int J Ophthalmol* 2019; 12 (7): 1151–1157; 7. Higgins TP *et al. Oman J Ophthalmol* 2016; 9 (3): 174–176; 8. Olguin-Manríquez F *et al. Int J Retina Vitreous* 2018; 4: 30; 9. Francis JH *et al. Ophthalmology* 2017; 124 (12): 1788–1798; 10. Francis JH *et al. JAMA Ophthalmol* 2021; 139 (10): 1126–1130.

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Treatment of CSC and special CSC subtypes

CSC, central serous chorioretinopathy.

Overview of CSC treatment

- Due to the natural course of CSC and its various clinical manifestations, **establishing optimal treatment guidelines is complicated**
- Most acute CSC cases recover within 3–4 months; therefore, no treatment (**observation**) is an appropriate first-line approach
- Treatment should be considered if retinal detachment persists for >3 months[1,2]
- Chosen treatment modality must be **evidence-based** and demonstrate a **favorable safety profile** due to the relatively good visual prognosis of patients with CSC
- Most published studies have analyzed retrospective data; large, prospective, randomized controlled trials over a defined treatment period are of particular interest

Reduction of risk factors:

High levels of endogenous or exogenous corticosteroids are associated with the development of CSC; therefore, **discontinuation of all steroids is recommended**[1,3]

Lifestyle modifications and **psychosocial therapy** for patients prone to psychological stress, and **treatment for sleep apnea** where required, are also helpful[2]

CSC, central serous chorioretinopathy.

1. van Rijssen TJ *et al. Prog Retin Eye Res* 2019; 73: 100770; 2. Lam DSC *et al*. In: Sadda SR, Schachat AP, Wilkinson CP *et al*. (eds). Ryan's Retina. 7th edn. (Elsevier, 2023) 1530–1554; 3. Kaye R *et al. Prog Retin Eye Res* 2020; 79: 100865.

Laser photocoagulation and sub-threshold laser

Laser photocoagulation

- Navigated laser photocoagulation has been suggested for CSC associated with extrafoveal leakage on FA[1]
- Laser treatment may not affect overall CSC prognosis; however, a non-randomized study demonstrated faster fluid resolution, fewer CSC recurrences, and better visual acuity after 5 years with focal laser vs no active treatment[2]

Sub-threshold laser

- To reduce retinal damage from laser photocoagulation, laser exposure duration can be decreased[3]
- Although studies have shown that various sub-threshold laser treatments are effective in reducing SRF and improving functional outcomes, long-term clinical outcomes have yet to be determined[4,5]

Micropulse laser

- Effectiveness is controversial, with the lack of an established protocol a limitation
- In a retrospective case series, complete SRF resolution occurred in 36–100% of patients with chronic CSC[6,7]
- In the PLACE trial (n=79) complete resolution of SRF was reported in 41% and 21% of patients with chronic CSC with focal and diffuse leakage, respectively[8]

End-point management laser

- Treats the entire macular area within a certain range with a standardized pattern[4]

Selective retina therapy

- Selectively destroys the RPE with high peak temperatures around the melanosomes without damaging neurosensory retinal tissue[5]

CSC, central serous chorioretinopathy; FA, fluorescein angiography; PLACE; Half-Dose Photodynamic Therapy versus High-Density Subthreshold Micropulse Laser Treatment in Patients with Chronic Central Serous Chorioretinopathy trial; RPE, retinal pigment epithelium; SRF, subretinal fluid.

1. Müller B *et al. Graefes Arch Clin Exp Ophthalmol* 2018; 256: 1581–1588; 2. Burumcek E *et al. Ophthalmology* 1997; 104: 616–622; 3. Battaglia Parodi M *et al. Pharmaceuticals (Basel)* 2020; 13 (11): 359; 4. Schworm B *et al. Graefes Arch Clin Exp Ophthalmol* 2021; 259: 3271–3281; 5. Büttner M *et al. Graefes Arch Clin Exp Ophthalmol* 2021; 259 (6): 1401–1410; 6. Scholz P *et al. Eye* 2016; 30: 1371–1377; 7. Scholz P *et al. Adv Ther* 2017; 34 (7): 1528–1555; 8. van Rijssen TJ *et al. Am J Ophthalmol* 2019; 205: 1–10.

Photodynamic therapy (PDT)

- **Reduced-setting PDT** regimens were developed to avoid possible complications of full-setting PDT, e.g., profound angiographic closure[1,2]
  - **Half-dose PDT:** reducing the amount of verteporfin injected while maintaining full fluence
  - **Half-fluence PDT:** halving the fluence after administering the standard verteporfin dose
- The PLACE trial of half-dose PDT in CSC found that changes in choroidal thickness usually decrease after 1 month,[3,4] suggesting that **PDT reduces choroidal vascular hyperpermeability**[5]

In acute CSC:

- **Observation**, **early half-dose**, or **half-fluence** PDT may be considered[3,6]
- Both ICGA-guided and FA-guided PDT may be effective[7]
- Early PDT is recommended in cases of decreased visual acuity, severe visual discomfort, recurrent episodes, and only one functioning eye[7]
- **PDT may decrease risk of SRF recurrence:**
  - Ozkaya A *et al*. found that 51% of untreated patients had recurrence vs 25% of those treated with low-fluence PDT[8]
  - Mohabati D *et al*. reported SRF recurrence in 24% of untreated eyes vs 4% of eyes that received early treatment consisting primarily of FA-guided half-dose PDT[9]

In chronic CSC:

- **Half-dose PDT** appears to be the most effective treatment
  - PLACE demonstrated complete resolution of SRF in 51% and 67% of patients after 6–8 weeks and 7–8 months, respectively[4]
  - Other studies have reported long-term resolution rates of 91% and 81% at 19 and 50 months of follow-up, respectively[10,11]
  - Reduced risk of recurrence compared to observation alone (20% vs 53.8% after ≥3 years of follow-up) has also been demonstrated[12]
- Patients with chronic CSC and extensive foveal RPE atrophy should be counselled regarding risk of further vision loss following PDT; further research is needed on this[3]

CSC, central serous chorioretinopathy; FA, fluorescein angiography; ICGA, indocyanine green angiography; PDT, photodynamic therapy; PLACE; Half-Dose Photodynamic Therapy versus High-Density Subthreshold Micropulse Laser Treatment in Patients with Chronic Central Serous Chorioretinopathy trial; RPE, retinal pigment epithelial; SRF, subretinal fluid.

1. Nicoló M *et al. Am J Ophthalmol* 2014; 157: 1033–1037.e1032; 2. Shin JY *et al. Retina* 2011; 31 (1): 119–126; 3. van Rijssen TJ *et al. Prog Retin Eye Res* 2019; 73: 100770; 4. van Dijk EHC *et al. Ophthalmology* 2018; 125 (10): 1547–1555; 5. Izumi T *et al. Br J Ophthalmol* 2017; 101 (4): 433–437; 6. Zhao M *et al. JAMA Ophthalmol* 2015; 133 (3): 333–340; 7. Lu HQ *et al. Eye* 2016; 30: 15–22; 8. Ozkaya A *et al. Eye* 2016; 30: 1005–1010; 9. Mohabati D *et al. Clin Ophthalmol* 2020; 14: 1165–1175; 10. Dhirani NA *et al. Clin Ophthalmol* 2017; 11, 2145–2149; 11. Haga F *et al. PLoS One* 2017; 12 (7): e0181479; 12. Lai TYY *et al. Trans Am Ophthalmol Soc* 2015; 113: 1–27.

Anti-VEGF agents and other treatments

Intravitreal injection of anti-VEGF agents:

- A meta-analysis did not confirm the efficacy of bevacizumab, ranibizumab, or aflibercept for treatment of acute CSC; however, the authors suggested that such treatment may be of benefit in chronic CSC[1]
- Three monthly intravitreal aflibercept injections resulted in greater BCVA improvement than sham treatment in patients with CSC (of >6 weeks' duration), in a prospective, randomized study[2]
- Intravitreal anti-VEGF injections are the standard treatment for CSC complicated by active CNV[3]

Mineralocorticoid and glucocorticoid receptor antagonists:

- Some research suggests that eplerenone is effective in the treatment of CSC, and that oral spironolactone results in faster absorption of SRF vs observation alone[4]
  - However, the randomized, placebo-controlled VICI trial did not demonstrate superiority of eplerenone vs placebo in improving BCVA in chronic CSC[5]
- Oral mifepristone may be an effective treatment in chronic CSC;[6] further evidence is needed to fully evaluate its efficacy in this indication

Finasteride:

- While efficacy was demonstrated in chronic CSC in a small, retrospective study review,[7] finasteride is not currently considered a viable treatment due to frequent side effects and a lack of conclusive evidence

Rifampicin:

- Rifampicin has been proposed as a treatment for CSC due to its impact on P450 3A4 induction and endogenous steroid metabolism
- An observational study found that rifampicin improved BCVA in patients with CSC;[8] however, its side effects should not be ignored[9]

BCVA, best corrected visual acuity; CNV, choroidal neovascularization; CSC, central serous chorioretinopathy; SRF, subretinal fluid; VEGF, vascular endothelial growth factor.

1. Ji S *et al. Int J Clin Pharm* 2017; 39 (3): 514–521; 2. Yoon YH *et al. J Vitreoretin Dis* 2017; 1 (2): 101–108; 3. van Dijk EHC *et al. Prog Retin Eye Res* 2021; 84: 100955; 4. Sun X *et al. Br J Ophthalmol* 2018; 102 (8): 1060–1065; 5. Lotery A *et al. Lancet* 2020; 395: 294–303; 6. Nielsen JS, Jampol LM. *Retina* 2011; 31 (9): 1928–1936; 7. Moisseiev E *et al. Eye* 2016; 30: 850–856; 8. Khan MS *et al. Pak J Med Sci* 2016; 32 (5): 1158–1163; 9. Nelson J *et al. Retin Cases Brief Rep* 2014; 8 (1): 70–72.

Treatment of special subtypes of CSC

Complex chronic CSC with CNV

- The standard treatment for CSC complicated by active type 1 CNV is **intravitreal anti-VEGF injections**; several studies have demonstrated good efficacy in such cases[1]
- The Phase 3 **MINERVA** study demonstrated that anti-VEGF therapy is more effective than sham injections for CNV of uncommon causes, including in eyes with CNV secondary to CSC[2]
- Further research is required to understand the role of **PDT** for this subtype of CSC

Bullous CSC

- Kawamura R *et al*. reported complete resolution of SRF in five of eight patients with atypical CSC (defined as bullous retinal detachment with diffuse or several leakages, severe leakage with fibrin formation under serous retinal detachment, or leakage within a PED) within 1 month of **transpupillary thermotherapy**[3]
- PDT can be considered an alternative option for such patients, since thermotherapy is no longer widely used[4]
- In such treatment-resistant and refractory cases, a **combined** therapeutic approach may be beneficial; further discussion on this topic is warranted

CNV, choroidal neovascularization; CSC, central serous chorioretinopathy; PDT, photodynamic therapy; PED, pigment epithelial detachment; SRF, subretinal fluid; VEGF, vascular endothelial growth factor.

1. van Dijk EHC, Boon CJF. *Prog Retin Eye Res* 2021; 84: 100955; 2. Lai TYY *et al. Retina* 2018; 38 (8): 1464–1477; 3. Kawamura R *et al. Clin Ophthalmol* 2012; 6: 175–179;
4. Ng WW *et al. J Med Case Rep* 2011; 5, 208.

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Vision Academy recommendations

Confirm the diagnosis of CSC and reduce modifiable risk factors

STEP 1

- Diagnosis of CSC should be confirmed by **identification of key features of CSC** through multimodal imaging
  - At this stage, it is important to look for the presence of type 1 CNV using OCT angiography and to rule out other diseases that may mimic CSC
- Once the diagnosis of CSC has been confirmed, physicians should **check for any modifiable risk factors**
  - If present, physicians should work with the prescribing or other physicians to reduce modifiable risk factors, such as use of steroid cream or other drugs, or reduction of psychological or physical stress

CNV, choroidal neovascularization; CSC, central serous chorioretinopathy; OCT, optical coherence tomography.

Determine subfoveal or extrafoveal involvement and duration of symptoms

STEP 2

- Patients with CSC should be assessed to determine whether there is **subfoveal involvement** or **extrafoveal involvement** only
- Duration of disease can influence the recommended management options; therefore, it is also important to establish, wherever possible, whether the disease is:
  - **Acute** (duration <3 months), or
  - **Chronic** (duration ≥3 months)

CSC, central serous chorioretinopathy.

Consider disease characteristics

STEP 3

- Acute or chronic CSC with extrafoveal involvement only and no obvious focal leakage:
  - **Observation for up to 3–4 months** should be considered to monitor for spontaneous resolution, or commence treatment immediately
- Acute subfoveal CSC:
  - An **observation period of 3–4 months** is also recommended, to monitor for spontaneous resolution
- If no improvement is observed initially through serial OCT monitoring, **treating earlier than 3–4 months** should be considered, as persistent SRF could lead to vision loss
- Early treatment should also be considered in patient subsets with certain characteristics, namely:
  - Decreased visual acuity, visual disturbances, history of recurrent CSC episodes, cystoid macular changes or RPE atrophy, evidence of photoreceptor damage, CSC in the only or better-seeing eye, or patient preference for early treatment

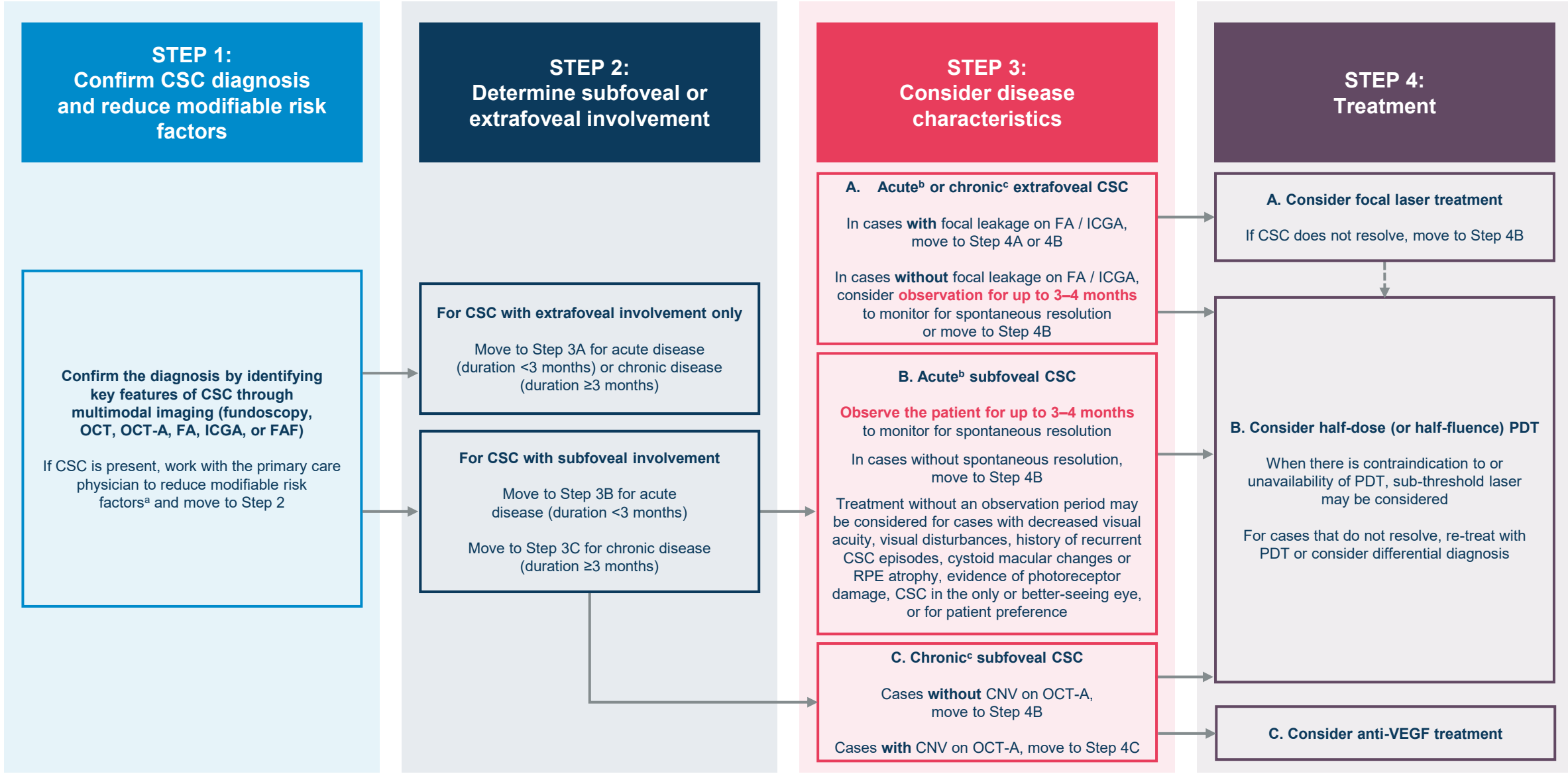
CSC, central serous chorioretinopathy; OCT, optical coherence tomography; RPE, retinal pigment epithelial; SRF, subretinal fluid.

Treatment

STEP 4

- Acute or chronic CSC with extrafoveal involvement only:
  - **Focal laser or half-dose (or half-fluence) PDT** should be considered if focal leakage is present on FA or ICGA
  - In the absence of focal leakage, an observation period of 3–4 months can be considered to monitor for spontaneous resolution
- Acute or chronic CSC with subfoveal involvement and absence of type 1 CNV on OCT:
  - **Half-dose (or half-fluence) PDT is recommended** (possibly after an observation period of 3–4 months in acute CSC)
  - Sub-threshold laser treatment may be considered for cases with extensive RPE damage, previous poor response to PDT, contraindication to PDT, or unavailability of PDT
- Chronic subfoveal CSC with type 1 CNV on OCT angiography:
  - **Anti-VEGF treatment should be considered**

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



[a]Modifiable risk factors may include the use of steroid cream or other drugs, or psychological or physical stress; [b]Acute CSC is defined as CSC present for <3 months; [c]Chronic 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.

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Summary

Vision Academy recommendations

Diagnosis of CSC should be confirmed and modifiable risk factors reduced

Subfoveal or extrafoveal involvement and duration of symptoms should be determined

Disease characteristics should be considered

Focal laser, half-dose (or half-fluence) PDT, or anti-VEGF treatment should be considered

CSC, central serous chorioretinopathy; PDT, photodynamic therapy; VEGF, vascular endothelial growth factor.

Further considerations

The **baseline clinical characteristics** of the patient with CSC are important to determine, to establish both the optimal treatment option and timing of treatment

Based on efficacy and safety data from retrospective and prospective studies, **half-dose (or half-fluence) PDT** should be considered the treatment of choice for chronic CSC; however, pathophysiology according to various subtypes, and treatment outcomes of those subtypes, remain to be investigated

Future large, multicenter, randomized trials will further evaluate the long-term outcomes of PDT and the efficacy of other treatments, providing an **improved comparative overview of currently available treatment options**

CSC, central serous chorioretinopathy; PDT, photodynamic therapy.