Asia-Pacific Journal of Ophthalmology 14 (2025) 100252

Contents lists available at ScienceDirect

Asia-Pacific Journal of Ophthalmology

journal homepage: www.sciencedirect.com/journal/asia-pacific-journal-of-ophthalmology

# International consensuses and guidelines on central serous chorioretinopathy (CSC) by the Asia Pacific Vitreo-retina Society (APVRS), the Academy of Asia-Pacific Professors of Ophthalmology (AAPPO) and the Academia Retina Internationalis (ARI)

Nishant V. Radke [a,b,c,1], Elon H.C. van Dijk [d,at,1], Richard F. Spaide [e], Frank G. Holz [f], Hideki Koizumi [g], K. Bailey Freund [e,h], Yousif Subhi [i,j,k], Clemens Lange [l,m], Sumit Randhir Singh [n], Haoyu Chen [o], Li Jia Chen [p,q,r,s], San-Ni Chen [t,u], Jay Chhablani [v], Francine Behar-Cohen [w,x], Taraprasad Das [y,z], Adrian T. Fung [aa,ab,ac], Fumi Gomi [ad], Min Kim [ae], Chi-Chun Lai [af,ag], Timothy Y.Y. Lai [p], Christopher Seungkyu Lee [ah], Andrew Lotery [ai,aj], Sobha Sivaprasad [ak,al], Mingwei Zhao [am,an,ao,ap,aq], Dennis S.C. Lam [a,b,c,p,ar,*], Camiel J.F. Boon [d,as,**]

[a] The Primasia International Eye Research Institute of The Chinese University of Hong Kong (Shenzhen), Shenzhen, China
[b] Eye Department, C+ Health CKJ (Shenzhen) Hospital, Luohu, Shenzhen, China
[c] The C-MER (Shenzhen) Dennis Lam Eye Hospital, Shenzhen, China
[d] Department of Ophthalmology, Leiden University Medical Center, Leiden, the Netherlands
[e] Vitreous Retina Macula Consultants of New York, New York, NY, USA
[f] Department of Ophthalmology, University of Bonn, Bonn, Germany
[g] Department of Ophthalmology, Graduate School of Medicine, University of the Ryukyus, Ginowan, Okinawa, Japan
[h] Department of Ophthalmology, New York University Grossman School of Medicine, New York, NY, USA
[i] Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark
[j] Department of Ophthalmology, Copenhagen, Rigshospitalet, Denmark
[k] Department of Clinical Research, University of Southern Denmark, Odense, Denmark
[l] Ophthalmology Department, St. Franziskus Hospital, Münster, Germany
[m] Eye Center, University Hospital of Freiberg, Freiburg, Germany
[n] Akhand Jyoti Eye Hospital, CoE Mastichak, Saran, Bihar, India
[o] Joint Shantou International Eye Center, Shantou University and The Chinese University of Hong Kong, Shantou, China
[p] Department of Ophthalmology and Visual Sciences, The Chinese University of Hong Kong, Hong Kong, China
[q] Department of Ophthalmology and Visual Sciences, Prince of Wales Hospital, Hong Kong, China
[r] Hong Kong Eye Hospital, Hong Kong, China
[s] Hong Kong Hub of Paediatric Excellence, The Chinese University of Hong Kong, Hong Kong, China
[t] Department of Ophthalmology, China Medical University Hospital, China Medical University, Taichung, Taiwan
[u] School of Medicine, College of Medicine, China Medical University, Taichung, Taiwan
[v] Department of Ophthalmology, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA
[w] Centre de Recherche des Cordeliers, INSERM UMRS 1138, Sorbonne Université, Physiopathology of Ocular Diseases: Therapeutic Innovations, Paris, France
[x] Assistance Publique, Hôpitaux de Paris, Hôpital Cochin, Ophtalmopole, Paris, France
[y] LV Prasad Eye Institute, Kalam Anji Reddy Campus, Anant Bajaj Retina Institute, Srimati Kanuri Santhmma Centre for Vitreoretinal Diseases, Hyderabad, India
[z] Shantilal Shanghvi Eye Institute, Mumbai, India
[aa] Save Sight Institute, Specialty of Clinical Ophthalmology and Eye Health, The University of Sydney, Sydney, New South Wales, Australia
[ab] Department of Ophthalmology, Westmead Hospital, Sydney, New South Wales, Australia
[ac] Department of Ophthalmology, Faculty of Medicine and Health Sciences, Macquarie University, Sydney, Australia
[ad] Department of Ophthalmology, Hyogo Medical University, Nishinomiya, Hyogo, Japan
[ae] Department of Ophthalmology, Institute of Vision Research & Gangnam Severance Hospital, Yonsei University College of Medicine, Seoul, South Korea
[af] Department of Ophthalmology, New Taipei Municipal TuCheng Hospital, New Taipei City, Taiwan
[ag] College of Medicine, Chang Gung University, Taoyuan, Taiwan
[ah] Department of Ophthalmology, Institute of Vision Research, Severance Hospital, Yonsei University College of Medicine, Seoul, South Korea
[ai] Clinical and Experimental Sciences, Faculty of Medicine, University of Southampton, Tremona Road, Southampton, UK

* Correspondence to: 2001 Longxiang Boulevard, Longgang District, Shenzhen, China.
** Correspondence to: Department of Ophthalmology, Amsterdam University Medical Centre, University of Amsterdam, Amsterdam, the Netherlands.
*E-mail addresses:* dennislam@pieri.cuhksz.org (D.S.C. Lam), camiel.boon@amsterdamumc.nl (C.J.F. Boon).

https://doi.org/10.1016/j.apjo.2025.100252
Received 15 May 2025; Received in revised form 14 October 2025; Accepted 14 October 2025
Available online 15 October 2025

[aj] Eye Unit, University Hospital Southampton NHS Foundation Trust, Southampton, UK
[ak] UCL Institute of Ophthalmology, London, UK
[al] Moorfields NIHR Biomedical Research Centre, Moorfields Eye Hospital, London, UK
[am] Department of Ophthalmology and Clinical Centre of Optometry, Peking University People's Hospital, Beijing, China
[an] Institute of Medical Technology, Peking University Health Science Center, Beijing, China
[ao] College of Optometry, Peking University Health Science Center, Beijing, China
[ap] Eye Disease and Optometry Institute, Peking University People's Hospital, Beijing, China
[aq] Beijing Key Laboratory of the Diagnosis and Therapy of Retinal and Choroid Diseases, Beijing, China
[ar] The C-MER Dennis Lam & Partners Eye Center, C-MER International Eye Care Group, Hong Kong, China
[as] Department of Ophthalmology, Amsterdam University Medical Center, University of Amsterdam, Amsterdam, the Netherlands
[at] Rotterdam Eye Hospital, Rotterdam, the Netherlands

ARTICLE INFO

*Keywords:*
Controversies
Consensuses
Guidelines
Central Serous Chorioretinopathy
APVRS
AAPPO
ARI

ABSTRACT

*Purpose:* To establish consensus-based guidelines on the diagnosis, classification, and management of central serous chorioretinopathy (CSC) through a structured expert panel initiated by the Asia-Pacific Vitreo-retina Society (APVRS), the Academy of Asia-Pacific Professors of Ophthalmology (AAPPO), and the Academia Retina Internationalis (ARI), addressing the existing clinical controversies.
*Methods:* An international panel of 26 experts from 13 countries collaboratively drafted consensus statements spanning five key areas: disease definition, pathophysiology, investigations, current management, and future developments. Consensus was reached through an iterative Delphi process and anonymous voting using a five-point Likert scale. Statements were accepted when >75 % agreement ('agree' & 'strongly agree') was achieved.
*Results:* Consensus was achieved for all 25 statements, reflecting strong alignment among experts. Key agreements included defining CSC as a pachychoroid-driven chorioretinal disorder characterized by neurosensory retinal and/or RPE detachment, with multimodal imaging (optical coherence tomography, fundus autofluorescence, fluorescein angiography, and indocyanine green angiography) recognized as essential for diagnosis. Half-dose photodynamic therapy (PDT) was unanimously endorsed as the first-line treatment for chronic CSC. Oral mineralocorticoid receptor antagonists (MRAs) lacked consensus for therapeutic benefit, aligning with evidence from the VICI and SPECTRA trials. Anti-vascular endothelial growth factor receptor therapy was recommended solely for CSC complicated by a macular neovascularization. Future priorities highlighted standardizing disease classification and exploring targeted therapies through genetic and nanomedicine research.
*Conclusion:* This consensus initiative provides a robust, evidence-based framework for the diagnosis and management of CSC, promoting standardization across clinical practices and guiding future research directions to address persistent gaps in CSC care.

## 1. Introduction

Central serous chorioretinopathy (CSC) is a common cause of non-surgical retinopathy besides age-related macular degeneration, diabetic retinopathy, and retinal vein occlusion,[1] predominantly affecting patients in the productive age groups from 20 to 50 years in a male: female ratio ranging from 2:1–6:1.[2] This demographic challenge is compounded by patient-related visual symptoms such as blurred vision, metamorphopsia, dyschromatopsia, and reduced contrast sensitivity. These symptoms are often linked to established risk factors including stress, type A personality, hypertension, sleep apnea, and steroid use. Together, these factors increase the risk of recurrence, bilateral involvement, and progression to chronic disease. In chronic central serous chorioretinopathy (cCSC), persistent subretinal fluid (SRF) can lead to irreversible damage to the photoreceptors and retinal pigment epithelium (RPE), resulting in reduced visual acuity over time. Additionally, studies have shown that delayed treatment and recurrent disease are associated with poorer visual outcomes despite anatomical improvements.[3] Management is still challenging due to cost concerns as well as a paucity of access to verteporfin-based photodynamic therapy (PDT), the evolving roles of subthreshold micropulse laser, mineralocorticoid receptor antagonists (MRA), and intravitreal anti-vascular endothelial growth factor (anti-VEGF) agents. Due to its relatively high prevalence and common natural history of resolution in the acute phase, CSC often gets managed by general ophthalmologists before the patients get referred to retina specialists. Diagnostic advances in the form of multimodal imaging using spectral-domain optical coherence tomography (SD-OCT), fundus auto-fluorescence (FAF), OCT-angiography (OCT-A), fluorescein angiography (FA), and indocyanine angiography (ICGA), have paved the way in localizing the disease at the level of the choroid and outer retina. Increased scleral thickness was first reported by Imanaga et. al. and is also shown to be associated with CSC.[4–6] This newer anatomical aspect offers CSC the unique distinction of having research value as a model of choroidal vascular and RPE barrier dysfunction and the principles thus identified can be applied to other chorioretinal disorders.

CSC demands an updated consensus regarding diagnosis and classification due to its variable clinical course and significant risk of chronic vision loss. As inconsistent definitions of acute CSC (aCSC) versus cCSC and overlapping features with conditions like polypoidal choroidal vasculopathy (PCV) lead to misdiagnosis, particularly in regions lacking advanced imaging tools like OCT, standardizing diagnosis and classification are critical.

Clear criteria and unified terminology would improve risk stratification, helping clinicians identify high-risk features like diffuse RPE atrophy, posterior cystoid retinal degeneration, and macular neovascularisations (MNVs). The need to optimize treatment pathways is equally pressing, as current practices vary widely.[7]

While aCSC often resolves spontaneously, cCSC management is complex with the underuse of proven therapies like PDT due to several factors including unavailability, cost, and insurance non-coverage.[8] A consensus could establish evidence-based thresholds for intervention and prioritize therapies like half-dose PDT, which reduces SRF more effectively than laser photocoagulation, MRA, and other proposed management strategies, but remains underutilized.

Furthermore, research comparability is hindered by heterogeneous study designs and endpoints. Harmonizing outcomes, such as OCT-based fluid resolution, and refining of studies in which CSC patients are

[1] contributed equally to this work and should be considered as co-first authors.

**Table 1**
Flowchart of review of literature.

| Stage | Records (n) |
|---|---|
| Records identified | 929 |
| **Records excluded before screening:** | |
| └ Duplicates | 11 |
| └ Non-English articles | 33 |
| **Records screened using title/abstract** | 885 |
| └ Records excluded (irrelevant abstracts) | 748 |
| **Full-text articles assessed for eligibility** | 137 |
| └ Full-text exclusions (e.g. retracted, poor quality) | 9 |
| **Final No. of Studies included** | **128** |

included, would accelerate robust development of current and novel therapies like MRAs or intravitreal anti-VEGF agents.

Consensus-driven strategies to standardize diagnosis and management protocols of CSC would reduce preventable vision loss, enhance research quality, and ensure patient-centered management across diverse healthcare landscapes, ultimately transforming CSC from a nebulous clinical challenge into a systematically addressable cause of vision loss.

Given its relatively high prevalence and its propensity for visual morbidity, [2,9,10] the latest advancements in our understanding of CSC, supported by newly available literature and data, the Asia Pacific Vitreo-retina Society (APVRS), the Academy of Asia-Pacific Professors of Ophthalmology (AAPPO) and the Academia Retina Internationalis (ARI) felt the need of such consensus statements and guidelines for CSC, and two of the senior authors (CFB and DSCL) of this manuscript were appointed to coordinate this consensus project. This consensus statement aims to synthesize evidence-based real-world practice recommendations from leading global experts to guide diagnosing and managing CSC.

## 2. Methods

Further to appointing the coordinators, the APVRS, AAPPO, and ARI formed an international expert panel comprising 26 panellists from 13 countries/territories. A core group of 4 members (CFB, DSCL, NVR and EVD) selected from the panellists was then established to perform an extensive literature search and review on CSC and prepare the first draft of the consensus statements with explanation and elaboration. PubMed was used to do the literature search and only English articles were selected for inclusion. Search terminologies included and used in combinations were: central serous chorioretinopathy, CSC, CSR, CSCR, diagnosis, management, multimodal imaging, treatment, PDT, and review. A flowchart of review of literature is shown in Table 1. Cross references were used from some of the studied articles as well wherever found relevant after the primary review. Since the main purpose of the study is to highlight consensus statements and address the controversies using such statements, the scope of review was restricted to identifying these common points rather than conduct a meta-analysis or systematic review. These statements were organized into five categories: the disease entity, pathophysiology, investigation and diagnosis, current management, and future developments.

Each panel member independently and anonymously reviewed each statement and provided comments to the core group. The core group then reviewed and evaluated the feedback and comments, revised it, and sent out the second draft for further opinions. The process was repeated until the statements were finalized. Subsequently, each panel member voted on each statement anonymously in the final draft using a five-point Likert scale—ranging from "strongly agree", "agree", "neutral", "disagree", to "strongly disagree". A consensus was reached when at least 75 % of the experts voted either "agree" or "strongly agree" for a statement as per the methodology described in a previous consensus paper.[11]

## 3. Controversies and consensus statements

### 3.1. Section 1: disease entity: history, terminology, and definition

CSC was first described in 1866 by Albrecht von Graefe under the name of relapsing central luetic retinitis.[12] Since then, multiple descriptive names have been proposed including central angiospastic retinitis,[13] central serous chorioretinitis,[14] annular central retinitis,[15] and vasoneurotic central retinitis.[16] One of the important terms – "serous" - was added to this condition's nomenclature by Horniker while retaining the "central" term as described by Fuchs.[17] In 1965 Klein used the term central serous retinopathy, and chorioretinopathy was upheld by Gass in 1967, whereafter it became more universally accepted (17). Maumenee was the first to describe the leak at the level of RPE using FA and subsequently Gass described the angiographic details in his studies.[12,17] Remarkably, a PubMed search with the terms "Central Serous Retinopathy and CSR" revealed 28 articles in the past 10 years, where the papers still refer to the condition as CSR without including the mention of choroid, highlighting the need to have an established standard nomenclature and abbreviation – like CSC or CSCR; for the purpose of this paper we prefer to use the abbreviation CSC.

CSC is a complex chorioretinal disorder of varied or diverse etiology which is characterized by a serous neuroretinal detachment, and usually affects the macular region.[18] CSC is a clinically heterogeneous disease, with presentations ranging from acute, transient, spontaneously resolving SRF accumulation to chronic, vision-threatening clinical pictures that cause extensive retinal damage. The absence of a universally accepted classification system for CSC has led to inconsistent definitions of disease subtypes (e.g., acute vs. chronic), variable diagnostic criteria, and fragmented management protocols, all of which undermine patient care and research progress. A standardized classification system is essential to address these challenges. First, it would clarify general diagnostic thresholds, such as the duration of SRF (e.g., generally defining "cCSC" as fluid persisting beyond 4–6 months on OCT) and distinguishing CSC from mimicking diseases like PCV or neovascular age-related macular degeneration (nAMD).[19] In addition, multimodal imaging often points towards chronicity,[20,21] even if the patients experience symptoms only for a shorter duration (e.g. extrafoveal locations and gravitational tracts). This precision is critical in guiding treatment decisions—for instance, initiating PDT in cCSC versus observation in aCSC —and ensuring that patients receive timely, appropriate care. Second, standardized classifications are vital for research comparability. Currently, studies use conflicting inclusion criteria, leading to inconclusive or contradictory results, which is even more challenging in retrospective studies on CSC – since it is a disease that can wax and wane. A unified system would harmonize study populations, endpoints (e.g., OCT-based fluid resolution), and outcome measures, enabling robust meta-analyses, and accelerating therapeutic innovation. Finally, standardized criteria would empower emerging technologies like artificial intelligence (AI), which rely on structured data to develop predictive models for recurrence risk or treatment response. Without consensus, AI algorithms trained on heterogeneous datasets risk bias or limited generalizability.

A recent multimodal imaging-based classification system for CSC was proposed by Chhablani et al.[20] This system requires two major criteria for diagnosis: (1) the presence or evidence of a prior serous neuroretinal detachment at the posterior pole on OCT, unrelated to other diseases, and (2) at least one area of RPE alteration identified on FAF, SD-OCT, or infrared imaging. Additionally, at least one minor criterion must be met: (1) mid-phase hyperfluorescent areas of indistinct leakage on ICGA, (2) one or more focal leaks on FA, or (3) subfoveal choroidal thickness (SFCT) $\geq 400$ μm.[20]

CSC is then categorized as simple or complex, with the difference between them defined as an RPE atrophy area $> 2$ disc areas.[20] Cases exhibiting bullous detachment, RPE tears, or an association with other retinal diseases are classified as atypical. Both simple and complex CSC

are subdivided into three groups: (1) primary CSC (first episode of SRF), (2) recurrent CSC (SRF with evidence of previous episodes), and (3) resolved CSC (no SRF on OCT after prior SRF).

The long-term clinical relevance and treatment outcomes based on this system remain to be elucidated. Pertaining to SFCT, Chen et al. found that it is increased not only in eyes clinically affected by CSC but also in their fellow eyes, supporting the concept of CSC as a bilateral disorder with mostly unilateral presentation.[22] They discussed that, a "threshold of awareness" can be estimated as the midpoint between the mean SFCT of control eyes plus one standard deviation and the mean SFCT of affected eyes minus one standard deviation. SFCT above this threshold may indicate heightened risk of subretinal fluid accumulation, making it a practical biomarker for early detection, monitoring progression, and evaluating treatment response, including corticosteroid sensitivity. Based on their calculations they suggested that thresholds closer to ~340–350 μm may be more appropriate in East Asians.[22] The concept of monitoring SFCT also needs to be understood in the light of the changes in thickness that happen with age, refractive status and axial length which affect measurements.[22]

***Consensus Statement 1.1:*** *Central serous chorioretinopathy is a chorioretinal disorder characterized by serous detachment of the neurosensory retina and/or retinal pigment epithelium in the macula, associated with choroidal vascular hyperpermeability and venous congestion. It typically affects middle-aged men and may present as an acute, self-limiting episode or progress to a chronic form with persistent subretinal fluid and RPE damage. (Consensus score: 100 % [strongly agree: 65.4 %; agree: 34.6 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

***Consensus Statement 1.2:*** *Standardized classification of CSC based on multimodal imaging is critical for improving diagnosis, treatment selection, and research comparability. (Consensus score: 100 % [strongly agree: 65.4 %; agree: 34.6 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

### 3.2. Section 2: pathophysiology

Imaging modalities, such as OCT and ICGA, have revealed that a thickened choroid (pachychoroid) and choroidal vascular hyperpermeability are hallmarks of CSC.[23–25] Hence, the disease is a part of the pachychoroid spectrum, in which there is thinning of the inner choroid, containing the medium- and small sized choroidal vessels (possibly including the choriocapillaris, which may cause ischemia and type 1 MNV), possibly due to compression by dilated large choroidal vessels of the outer choroid.[23,26–28] Although there are no conclusive and uniformly accepted definitions of pachychoroid, the term does find its presence in terms of a spectrum of disorders where choroidal venous overload is at play. Multimodal imaging findings suggest that changes in the choroidal blood vessels play a significant role in CSC. In some patients, these changes include enlarged outer choroidal vessels that compress and thin the inner layers, a finding that also seems to correspond with the flow voids seen on OCTA.

Evidence indicates that modifications in scleral dimension, particularly thickness, may contribute to the pathophysiological mechanisms underlying CSC. Research by Terao et al. has noted that alterations in the sclera can lead to fluid accumulation in the suprachoroidal space, a phenomenon that is well-documented in conditions like uveal effusion syndrome. Uveal effusion syndrome is characterized by exudative retinal and choroidal detachments due to scleral thickening, highlighting how increased scleral thickness may promote resistance in venous drainage, and may thus also induce diseases like CSC.[29] Similarly, Spaide et al. reported that eyes diagnosed with CSC possess a significantly thicker posterior sclera compared to normal eyes.[5] They connect these findings to a theory of venous overload choroidopathy, suggesting that venous outflow impairment, possibly related to scleral anatomy, can induce the observed fluid dynamics in CSC.[5] This relationship between scleral anatomy and vascular health underlines the complex interplay between structural changes and clinical manifestations of CSC. A prospective case-controlled study by Keidel et al. explored the relationship between scleral thickness in CSC, suggesting that increased anterior scleral thickness may correlate with venous outflow obstruction, adding evidence that such changes can contribute to a patient's risk of developing CSC.[6] Imanaga et al. also elucidated the relationship between scleral thickness and choroidal structure in patients with CSC confirming that a thicker sclera correlates with structural alterations in the choroid, contributing to the understanding of fluid dynamics.[30] Moreover, the impact of scleral thickness has also been assessed in different manifestations of CSC. Imanaga et al. discussed variations in scleral thickness patterns between simple and complex forms of CSC, providing insights into the nuances of scleral involvement in different presentations of the disease.[31] Elevated ocular perfusion pressure (OPP) appears to play a role in central serous chorioretinopathy (CSC), either alone or together with impaired venous outflow.[32] Yun et al. demonstrated that CSC patients, particularly those with fellow-eye pigment epitheliopathy, exhibit significantly higher OPP and thicker choroids compared to controls, suggesting hemodynamic stress as a driver of disease.[33] Systemic hypertension alone may not disturb choroidal hemodynamics in controlled patients,[34] indicating that CSC represents a distinct failure of protective vascular regulation.[35] This was a case-control study specifically aimed to demonstrate a dysfunction of mechanisms regulating choroidal blood flow in patients with CSC. Using stress/rest ICG angiography the study showed that in these patients the choriocapillaris are particularly vulnerable to variations of the systemic blood pressure.[35] A recent large case-control study found a significant link between vigorous physical activity and CSC, suggesting that such activity, which temporarily raises ophthalmic artery pressure, may trigger choroidal hyper-perfusion and dysregulation.[36] In a recent study, Piccolino et al. reported that low intraocular pressure (IOP), elevated mean ocular perfusion pressure, and high anterior scleral thickness values are linked with CSC.[32] IOP was found to be the strongest predictor amongst these three. Reduced IOP probably contributes by raising OPP, which promotes choroidal overperfusion and leakage, while also impairing fluid clearance through the sclera, together facilitating disease development.[32]

Studies have also reported that eyes with CSC exhibit such distinct choroidal phenotype, which is said to result in a mechanical and vascular compromise of the adjacent RPE.[23,27,37] Historical perspectives, based on earlier descriptions, have gradually shifted toward an integrative model of choroidal vascular dysfunction, [38] not only taking choroidal thickness into account.[39,40] Signs of venous overload choroidopathy, such as intervortex venous anastomoses, enlarged choroidal vessels (pachyvessels), and especially choroidal vascular hyperpermeability on mid-to-late-phase ICGA are seen in 80–90 % of CSC patients, even in clinically unaffected fellow eyes.[24] Specific patterns of choroidal leakage on ICGA can be seen in the context of CSC, and these patterns appear to correlate with age and disease severity,[25] and this hyperfluorescent leakage become less pronounced after treatment with PDT.[41,42]

In summary, CSC, as a member of the pachychoroid disease spectrum, results from thickened sclera and impaired vortex-vein drainage, causing venous congestion, dilated outer choroidal vessels, thinning of the inner choroid, choroidal hyperpermeability, and damage to the RPE, which leads to subretinal fluid leakage. Pachychoroid is a descriptive term but not essential for the diagnosis of CSC.

***Consensus Statement 2.1:*** CSC is linked to choroidal venous congestion and structural abnormalities, including dilated choroidal vessels, choroidal hyperpermeability, and increased scleral thickness, all of which contribute to its pathogenesis. *(Consensus score: 100 % [strongly agree: 57.7 %; agree: 42.3 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

The original association between CSC and type A personality was described by Yannuzzi in 1986.[43] Moreover, psychiatric illnesses have been shown to be a risk factor for recurrence in CSC.[44] A case-control study involving 31 subjects each revealed higher levels of depression, somatization, emotional distress and hostility in patients with CSC.[45] Activation of the sympathetic nervous system and

hypothalamic-pituitary-adrenal (HPA) axis is said to elevate endogenous catecholamine and corticosteroid levels, which might predispose the patient to CSC. However, a recent study could not reproduce these findings[46] A possible connection between CSC and maladaptive personality traits and psychiatric symptoms like anxiety, depression, insomnia and endocrinological disorders has been reported as well in the recent past.[47] Some other studies have found conflicting data with regards to the association of stress and CSC.[48,49]

Obstructive sleep apnea syndrome (OSA) has been reported in about 22 % of patients with CSC, which is much more compared to 2 %–4 % as reported in the general population.[50] A retrospective questionnaire-based study of 48 CSC patients and 48 matched controls found similar rates of OSA syndrome (46 % in CSC vs. 44 % in controls).[51] Shift work may trigger CSC by disrupting the circadian clock and altering cortisol and melatonin secretion.[52] Moreover, circadian disruption from artificial light at night or frequent jetlag is recognized as a significant cause of systemic morbidity.

***Consensus Statement 2.2:*** *Psychological stress and specific personality traits have been associated with an increased risk of CSC. Dysregulation of the hypothalamic-pituitary-adrenal axis has been proposed as a potential contributing mechanism. (Consensus score: 96.2 % [strongly agree: 42.3 %; agree: 53.9 %; neutral: 3.8 %; disagree: 0 %; strongly disagree: 0 %])*

***Consensus Statement 2.3:*** *Obstructive sleep apnea syndrome (OSA), and shift work, in predisposed individuals may be potential triggers for CSC. (Consensus score: 88.5 % [strongly agree: 30.8 %; agree: 57.7 %; neutral: 11.5 %; disagree: 0 %; strongly disagree: 0 %])*

Genetic predisposition has also been shown to further modulate an individual's vulnerability to develop CSC, suggesting that inherent factors and environmental triggers may work in unison to initiate and maintain the disease process.[53] Emerging evidence suggests that genetic variants involved in mineralocorticoid receptor function, vascular endothelium, and the complement system, may predispose patients to develop CSC by altering the choroidal vascular system and RPE integrity.[54–56]

***Consensus Statement 2.4:*** *Genetic factors have been associated with increased susceptibility to CSC.[57,58] The precise mechanisms by which these variants contribute to disease development remain under investigation. (Consensus score: 96.1 % [strongly agree: 26.9 %; agree: 69.2 %; neutral: 3.9 %; disagree: 0 %; strongly disagree: 0 %])*

In CSC, either an exogenous increase in blood corticosteroid levels or a stress-induced (endogenous) elevation in blood cortisol levels may disrupt the regulation of choroidal blood flow and impair the RPE, thereby allowing SRF accumulation.[37,59] Remarkably, for exogenous corticosteroids this is independent of the dosage and route of administration. Activation of mineralocorticoid and glucocorticoid receptors – receptors to which corticosteroids bind - have been shown to play a role in the pathogenesis of CSC.[60] Daruich et al. then discussed the potential role of MRA agents like eplerenone and spironolactone in the management of CSC.[18] Bousquet et al. reported that the effectiveness of eplerenone appears to be influenced by baseline SFCT measurements; specifically, patients with SFCT significantly above 515 microns may experience more substantial treatment responses.[61] Although several smaller studies described the potential benefit of using MRA in the management of CSC, two large randomized controlled trials, VICI and SPECTRA, did not find any evidence supporting the role of using these agents in the management of CSC. [3,62,63] To date, there is no direct evidence that the MR pathway is over-activated in the retina of CSC patients. On the other hand, MR activation causes coronaropathy, hypertension, and psychological stress, which have all been associated with CSC.[64–67]

***Consensus Statement 2.5:*** *Corticosteroid exposure is recognized as one of the major risk factors. Exposure to corticosteroids, whether exogenous or (stress-induced) endogenous, is one of the major risk factors for CSC due to its effects on choroidal circulation and RPE function. (Consensus score: 100 % [strongly agree: 42.3 %; agree: 57.7 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

***Consensus Statement 2.6:*** *Large randomized controlled trials, including the VICI and SPECTRA studies, have not demonstrated a significant benefit of the mineralocorticoid receptor antagonist eplerenone in the treatment of CSC. (Consensus score: 100 % [strongly agree: 38.5 %; agree: 61.5 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

CSC and gastro-oesophageal reflux disease have been shown to share stress and the adaptive response to stress as the risk factors. A retrospective case-control study reported a higher risk of GE reflux in patients with CSC [68] Another retrospective study from Taiwan reported a higher association of peptic ulcer in patients with CSC.[69] H. pylori-dependent immune mechanisms and molecular mimicry between pathogenic antigens and host proteins have been hypothesized, but additional studies are needed to confirm the relationship between CSC and H. pylori, and the benefit of H. pylori treatment on the course of CSC.[70,71] Interestingly, some conditions are linked to this disruption such as hypertension, insomnia, and the occurrence of peptic ulcers, which have been shown to have some association with CSC.[72] As of now the role of H. pylori in CSC is still considered controversial.

***Consensus Statement 2.7:*** *It is currently unclear if H. Pylori infection has a significant role in the pathogenesis of CSC. (Consensus score: 92.3 % [strongly agree: 34.6 %; agree: 57.7 %; neutral: 7.7 %; disagree: 0 %; strongly disagree: 0 %])*

### 3.3. Section 3: investigations and diagnosis

Multimodal imaging techniques provide details of structural and vascular information, and aid in the diagnosis of CSC.[21,57,73,74] This integrative approach of multimodal imaging in CSC, as opposed to dependence on a single modality, facilitates a more comprehensive assessment of the underlying chorioretinal changes which critically distinguish CSC from other conditions with similar presentations, which helps in prognostication and management.[19,21,59,75] FA and ICGA are used in the diagnosis and management of CSC with treatment modalities involving FA-guided laser photocoagulation or ICGA-guided PDT. A description of typical FA and ICGA findings in CSC is provided in the subsequent part.

OCT has emerged as a cornerstone in the diagnostic armamentarium for CSC. High-resolution cross-sectional images obtained with SD-OCT allow for detailed visualization of serous neuroretinal detachments, RPE alterations, and pigment epithelial detachments (PED).[76] Enhanced depth imaging OCT (EDI-OCT) and swept-source OCT (SS-OCT) further delineate the choroidal morphology, including changes in choroidal thickness that correlate with disease chronicity and severity.[23,77] Biomarkers on OCT like intraretinal hyperreflective dots (HRD) suggest chronicity; subretinal HRD point towards an increased risk of recurrence, while persistent HRDs in resolved CSC have been associated with poorer visual acuity.[21] Similarly, posterior cystoid retinal degeneration, ellipsoid zone loss and/or attenuation, external limiting membrane loss and/or attenuation, outer nuclear layer thinning, and the presence of a MNV are associated with poor visual prognosis.[21]

FAF imaging reflects the metabolic disturbance at the level of RPE more so as an indirect effect. The FAF imaging patterns differ between aCSC and cCSC by reflecting disease stage and RPE health. Early blocked hypo-autofluorescence evolves to mottled and then diffuse hyper-autofluorescence, while chronic disease shows mixed patterns and descending tracts from RPE atrophy. These changes help localize pathology, estimate chronicity, and anticipate visual outcomes.[2,78] Gravitational tracts are identified more prominently using FAF as opposed to simple colour fundus images. This multimodal approach of using FAF images along with OCT and fundus photography ensures that even subtle changes, which may be overlooked by a single modality, are accurately detected even during the first presentation in the clinic. Additionally Ayata et al. showed that near-infrared (NIR) and short-wavelength autofluorescence (SW-AF) reveal distinct patterns in acute CSC. NIR-AF effectively highlights leakage sites and serous detachment areas, while SW-AF detects granular changes earlier.

Together, these modalities provide a non-invasive way to monitor disease course and differentiate recent versus past CSC episodes.[79]

Optical coherence tomography angiography (OCT-A) evaluates the retinal and choroidal microvasculature in CSC without the need for intravenous dye injection.[80–82]

OCT-A can detect alterations in choriocapillaris flow and may reveal secondary neovascular complications that can influence treatment decisions. Flat irregular pigment epithelial detachments (FIPEDs) with a mid- to hyperreflective content are often harboring a nascent or a non-exudative MNV which is likely to be missed on conventional angiography but can be identified on OCT-A. [83,84] OCT-A-guided PDT has been found to be effective in management as well as opening up avenues in treating CSC patients refusing or unsuitable for invasive angiography.[85]

***Consensus Statement 3.1:*** *Multimodal imaging (fundus photography, OCT, OCT-A, FAF, FA, and ICGA is ideal and essential for the accurate diagnosis, monitoring, and classification of CSC. (Consensus score: 100 % [strongly agree: 57.7%; agree: 42.3 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

***Consensus Statement 3.2:*** *EDI-OCT and SS-OCT enable choroidal thickness measurements and identification of dilated outer choroidal vessels. These metrics should be monitored in both eyes before and during treatment. (Consensus score: 96.2 % [strongly agree: 38.5 %; agree: 57.7 %; neutral: 3.8 %; disagree: 0 %; strongly disagree: 0 %])*

***Consensus Statement 3.3:*** *Although multimodal imaging is ideal, SD-OCT with or without fundus photography/FAF may be enough to diagnose and monitor an acute first episode of CSC with no clinical signs of chronicity or complications. (Consensus score: 92.3 % [strongly agree: 34.6 %; agree: 57.7 %; neutral: 7.7 %; disagree: 0 %; strongly disagree: 0 %])*

FA and ICGA are pivotal imaging modalities to assess leakage patterns and choroidal hyperpermeability, providing a robust framework for the (differential) diagnosis of CSC when combined with OCT.[19,80] On ICGA, most patients with CSC show signs of choroidal venous overload,[27] as well as specific choroidal leakage patterns that correlate with disease severity and response to treatment.[25,41] Traditionally, ICGA-guided PDT is the preferred method to treat CSC, as it is generally recommended to treat the more widespread underlying choroidal abnormalities in this disease.[3] However, ICGA is not universally available in all centers and is more expensive as well as time consuming as compared to FA. Some studies have shown that FA-guided PDT offers beneficial structural and functional outcomes in CSC.[86–88] In patients allergic to ICG dye, FA still helps in identifying leaks, that need to be taken into account during treatment with PDT or conventional focal laser to extramacular leaks in the absence of PDT availability. Different leakage patterns like smoke stack and ink-blot leaks are identified with conventional FA, contributing to the disease classification.

***Consensus Statement 3.4:*** *FA remains useful in detecting active leakage points and planning focal laser photocoagulation in chronic or non-resolving CSC cases. It may also be useful in guiding PDT treatment when ICGA is unavailable. (Consensus score: 96.2 % [strongly agree: 50 %; agree: 46.2 %; neutral: 3.8 %; disagree: 0 %; strongly disagree: 0 %])*

***Consensus Statement 3.5:*** *ICGA plays a critical role in assessing choroidal hyperpermeability and differentiating CSC from mimicking conditions such as polypoidal choroidal vasculopathy. ICGA should be strongly recommended in all patients with suspected chronic CSC before initiating treatment. (Consensus score: 100 % [strongly agree: 50 %; agree: 50 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

Thus, multimodal imaging plays a pivotal role in the diagnosis and management of CSC, providing detailed insights into both retinal and choroidal structure and function and ultimately facilitating clinical decision-making. As the field continues to evolve, the combination of these imaging techniques will remain vital to both research and clinical practice in CSC.[21,42,59,74,76–78,80,81]

### 3.4. Section 4: current management

Acute, first-episode CSC is often a self-limiting condition, in contrast to cCSC which may lead to persistent visual impairment and reduced quality of life.[89] The most reported treatment options are generally reserved for non-resolving, recurrent, or cCSC and include pharmacotherapy, PDT, laser modalities, and intravitreal anti-VEGF injections. Half-dose PDT and green or micropulse laser treatment options stand out amongst these as modalities with better outcomes as compared to (oral or intravitreal) pharmacotherapy in patients with chronic CSC without MNV. The common goal of management is a complete resolution of subretinal fluid on OCT. Here, we aim to review these modalities, and their relative efficacy based on recent evidence.

In aCSC, observation remains the mainstay of management because of the high rate of spontaneous SRF resolution.[3] When the fluid persists beyond 3–6 months, or when recurrences are frequent, intervention is warranted to prevent progressive photoreceptor and RPE atrophy.[3,90,91] Studies have shown that even a brief period of SRF can cause irreversible photoreceptor damage.[92] Left untreated, nearly half (43–51 %) of patients with aCSC develop at least one recurrence.[93,94] Mohabati et al. reported that in a retrospective cohort study of 295 eyes with aCSC, recurrent CSC was seen in 24 % of the untreated group while only 4 % in the treated cases had a recurrence after early treatment with PDT.[94] A randomized, double-masked trial by Chan et al. evaluated half-dose verteporfin PDT in aCSC. At 12 months, 94.9 % of treated eyes showed complete resolution of subretinal fluid versus 57.9 % in the placebo group. Treated patients also had significantly better visual acuity, reduced central foveal thickness, and no adverse events. The study also confirmed that half-dose PDT is safe and an effective treatment for aCSC.[95] These studies underscore the potential importance of early treatment in aCSC for patients who depend on optimal visual acuity for occupational or professional reasons.

***Consensus Statement 4.1:*** *Observation is appropriate for most aCSC cases, but early intervention may be considered in selected patients. (Consensus score: 100 % [strongly agree: 50 %; agree: 50 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

PDT with verteporfin has emerged as the most effective treatment option for cCSC. Modified protocols, such as half-dose PDT or reduced-fluence PDT, have been employed to minimize adverse effects while achieving complete fluid resolution.[3,96–99] PDT has been shown to be safe and effective even when repeated for patients with persistent SRF or recurrent CSC post PDT in a recent study.[100] Recent prospective randomized treatment trials have shown superiority of half-dose PDT over subthreshold micropulse laser in cCSC, regardless of whether the fluid leakage is focal or diffuse.[101–104] In addition, prospective randomized treatment trials have also shown that half-dose PDT is markedly superior over the MRA eplerenone for the treatment of cCSC.[63,105] Systematic reviews and meta-analyses lend further support for PDT as an effective modality in reducing SRF and enhancing visual outcomes, particularly in chronic cases that are refractory to conservative management.[3,8] The reason why PDT is superior over other treatments in CSC is likely because it more directly and effectively targets the choroid, being the primarily abnormal tissue in CSC. [3,42,106,107]

Based on the available evidence, half-dose PDT seems to be the most preferred means of treatment.[3] The superiority of PDT over other treatments has been shown in randomized controlled trials, which is important because CSC is a disease that can wax and wane – even when no treatment is performed. A long-term retrospective study of pachychoroid spectrum disorders, FCE and PPE showed the most favorable visual outcomes, with ~90 % maintaining vision over seven years. CSC and PNV were linked to worse prognosis, and in CSC, prolonged subretinal fluid—rather than treatment type—was the main factor influencing long-term visual decline.[108] Retrospective studies are challenging to interpret,[3,62,109] however, it is relevant to note that a large retrospective multicenter study has also pointed to the benefit of PDT in cCSC.[110]

***Consensus Statement 4.2:*** *Half-dose PDT with verteporfin is the most evidence-based and effective treatment for cCSC, demonstrating superior anatomical and visual outcomes compared to subthreshold micropulse laser*

*and eplerenone, with a favorable safety profile. (Consensus score: 100 % [strongly agree: 57.7 %; agree: 42.3 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

### 3.4.1. Chronic CSC

Pharmacological approaches have been explored extensively in cCSC. Oral MRA—such as spironolactone and eplerenone—have been evaluated for their potential to normalize choroidal hyperpermeability, which is implicated in CSC pathogenesis.[111–113] Although several studies suggest that these agents can lead to a reduction in SRF and improvement in visual acuity, findings from larger randomized controlled trials, such as the VICI and SPECTRA trial have not demonstrated a significant benefit of MRA over placebo or PDT.[62,63,105,114] Other oral agents, including methotrexate [115] and various other anti-inflammatory or steroid-sparing medications,[116] have also been investigated with variable outcomes; however, the evidence remains inconclusive regarding their widespread clinical adoption.

Laser-based interventions have also been used in an attempt to treat CSC. Subthreshold micropulse laser therapy has been studied in patients with cCSC and appears to improve morphological parameters and visual acuity without the collateral damage associated with conventional thermal lasers.[117] In a recent prospective randomized trial in cCSC, treatment with a 577 nm subthreshold micropulse laser also showed promising results.[104] However, in this study, which compared this micropulse laser modality with half-dose PDT, the patient group treated with PDT showed superiority in speed of SRF resolution, percentage of patients achieving fluid resolution, and earlier improvement of best-corrected visual acuity. Still, the study does highlight the potential role of micropulse laser as an option where PDT as a treatment option is unavailable or not feasible. It is important to note that this study had relatively low patient numbers, a relatively short follow-up time of 12 months, and patients with multiple leakage points and/or more extensive RPE abnormalities on FA were excluded. Selective retina therapy (SRT) is another emerging laser technique that targets the RPE with a minimal impact on the neurosensory retina, and has shown promise in refractory cases.[118] Although these methods are less invasive and are associated with fewer complications, further randomized trials are needed to establish their long-term efficacy – as CSC is a disease that can wax and wane, and retrospective studies are therefore suboptimal in terms of assessing possible treatment outcomes.

***Consensus Statement 4.3:*** *Randomized controlled trials have demonstrated that photodynamic therapy is more effective than subthreshold micropulse laser in treating chronic CSC. However, subthreshold micropulse laser may be considered in cases where PDT is contraindicated or unavailable. (Consensus score: 92.3 % [strongly agree: 42.3 %; agree: 50 %; neutral: 7.7 %; disagree: 0 %; strongly disagree: 0 %])*

***Consensus Statement 4.4:*** *Advancement of subthreshold laser therapies could offer potential treatment options and benefits to patients where PDT is not possible or unavailable. (Consensus score: 88.5 % [strongly agree: 23.1 %; agree: 65.4 %; neutral: 11.5 %; disagree: 0 %; strongly disagree: 0 %])*

Treatment with intravitreal administration of anti-VEGF agents has also been examined in CSC, especially for cases complicated by MNV or persistent subfoveal SRF. [119–121] Although anti-VEGF therapy is well established for other retinal vascular diseases, its role in CSC without the presence of a MNV is less clear and does not appear to be indicated. Some studies have reported anatomical improvements; however, the benefits are often modest and do not consistently translate into significant visual improvement.[8,119,122,123] As such, anti-VEGF treatment may be considered to be an adjunct or treatment for atypical cCSC cases, and those complicated by MNV, rather than a first-line option.[121–123]

***Consensus Statement 4.5:*** *In cases of cCSC without MNV, intravitreal anti-VEGF injections are not indicated, and their use would be off-label. However, when cCSC is complicated by an MNV, intravitreal anti-VEGF injections are the first-line treatment. (Consensus score: 100 % [strongly agree: 38.5 %; agree: 61.5 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

### 3.4.2. Multifocal CSC/ Bilateral CSC/ CSC in pregnancy

A standard treatment protocol of half-dose PDT as the treatment of choice in managing CSC is well established.[3] For both aCSC and cCSC, a flowchart of treatment has been described earlier.[3] While treating multifocal CSC – where leakage is present on several (non-overlapping) locations, treating the macula first is the logical approach. However, for scheduling PDT some controversies remain. Some of them are as follows: is an overlap between treatment spots allowed? Which eye should be chosen first in cases of bilateral CSC? Some surgeons may prefer to treat the better eye first while some may prefer to treat the worse eye since these are eyes with good visual potential usually, and PDT may result in temporarily reduced visual acuity in some patients. It is to be considered that treating bilateral cases on separate days would mean opening two vials of verteporfin, which makes the procedure more expensive, while bilateral simultaneous PDT in cCSC has been shown to be effective and safe,[124] Still, sharing the vial in half-dose strategy may not be allowed in some countries.

Pregnancy has been recognized as a potential risk factor in the development of CSC. CSC has been linked to elevated cortisol levels, which may be associated with pregnancy-related physiological changes, particularly during the third trimester. Elevated cortisol and hormonal changes could lead to dysregulation of fluid dynamics within the RPE and subsequent accumulation of SRF.[125] Additionally, conditions such as pregnancy-induced hypertension and Hemolysis, Elevated Liver enzymes and Low Platelets syndrome have been implicated, where compromised choroidal blood flow may contribute to the development of CSC.[126] These factors create an environment conducive to CSC, resulting in the estimated occurrence of CSC in approximately 0.008 % of pregnancies.[127] Interestingly, Kim et al. found no significant change in choroidal thickness during uncomplicated pregnancy.[128] However, in cases of pre-eclampsia, they noted changes such as choroidal vascular congestion and increased choroidal permeability.[128] Additional data from a small case series in China suggest that pregnancy-associated CSC typically presents in the third trimester, and generally resolves spontaneously after delivery, with most patients achieving good visual acuity outcomes.[129] Given these observations, it is advisable to consider pregnancy status in women of reproductive age who present with CSC, as this may influence management and prognosis. Monitoring such patients closely throughout pregnancy is prudent, especially as symptoms often improve post-partum.

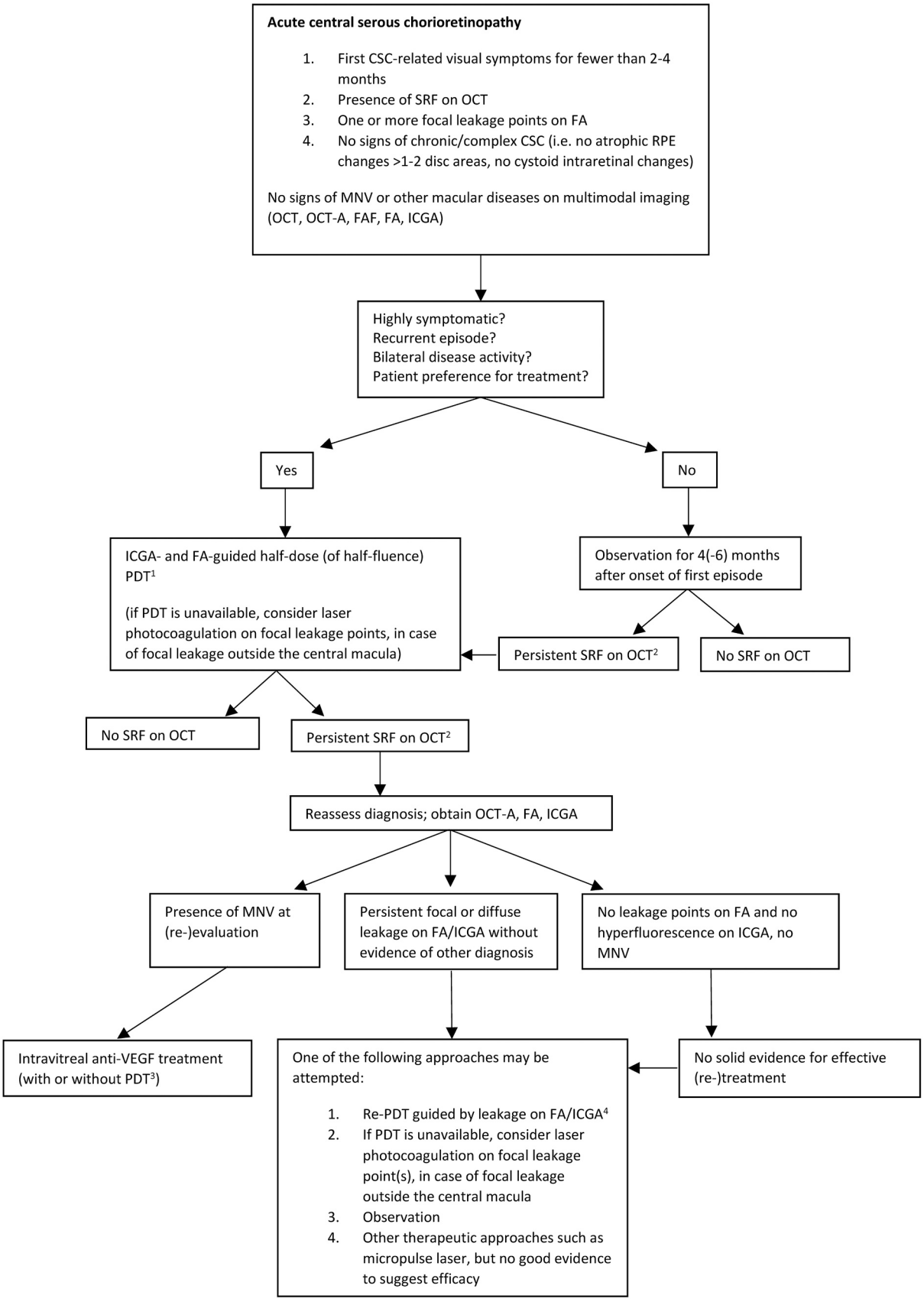
***Consensus Statement 4.6:*** *Pregnancy may get complicated by CSC. CSC during pregnancy can be safely monitored, and usually resolves completely after child-birth, with most patients recovering good vision. (Consensus score: 100 % [strongly agree: 34.6 %; agree: 65.4 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

### 3.4.3. Pharmacotherapy in several types of CSC

This section briefly describes an incomplete election of medications that have been tested in CSC. For an exhaustive list of pharmacotherapeutic means tried in the treatment of CSC, the readers are encouraged to go through other resources, for example, as included in the review paper and evidence-based guideline of Feenstra et al.[3]

**Mineralocorticoid receptor antagonists (MRA):** MRA as a class of pharmaceutical agents (primarily eplerenone and spironolactone) stand out in terms of usage compared to other medications that have been tried in the course of management of CSC. It has been suggested that since glucocorticoid receptors – and to a lesser extent mineralocorticoid receptors - are present in both retina and choroid, and they may play a role in the pathogenesis of CSC. [130–133] Several studies have elaborated the efficacy with mixed results. To date, 2 large prospective trials on cCSC as mentioned earlier have not shown any evidence to support the use of the MRA eplerenone for as a first-line treatment for cCSC.[62,63]

**Rifampicin**: Rifampicin is a cytochrome P4503A4 inducer, which decreases serum levels of endogenous corticosteroids, which may




*(caption on next page)*

**Fig. 1.** Suggested management algorithm for acute central serous chorioretinopathy. Note: if the patient is currently taking corticosteroids, discuss stopping their use prior to treatment.[1] Treat hyperfluorescent areas on indocyanine green angiography (ICGA) that correspond to the area of (focal) leakage on fluorescein angiography (FA) and subretinal fluid on optical coherence tomography (OCT). In case of multiple areas with focal leakage, a large spot including all areas can be used, or multifocal immediately sequential spots may be used, starting with the area including the fovea (if fovea is involved). [2] In case of only a small amount of residual subretinal fluid (SRF), a conservative approach may be followed, with a follow-up visit including OCT imaging after 1–3 months to see if SRF eventually resolves completely. In case of persistent/increased SRF at that stage, the downstream treatment decision pathway may be followed.[3] Half-dose or half-fluence photodynamic therapy (PDT) may be added in order to treat the choroidal dysfunction/pachychoroid factor of the disease, but limited data are available to support this combined treatment. When a neovascular component of polypoidal choroidal vasculopathy (aneurysmal type 1 neovascularization) is present, PDT (either full-dose, half-dose, or half-fluence) can also be added to anti-vascular endothelial growth factor treatment. [4] Another half-dose or half-fluence PDT can be performed, but full-dose with full-fluence PDT may also be considered.

accelerate SRF resolution in CSC. Small studies have suggested reductions in SRF upon treatment with oral rifampicin. [134,135]

**Methotrexate**: Traditionally used as an antimetabolite, methotrexate has also been explored in CSC management. This agent may alleviate SRF through its immunomodulatory effects, although its role remains less defined compared to other treatments. A study indicated that various treatments, including methotrexate, have been proposed for CSC, but definitive efficacy has not been well established, and methotrexate can have significant side effects.[115,136]

**Aspirin:** Based on a higher plasma concentrations of plasminogen activator inhibitor 1 in patients with CSC, the role of aspirin has been explored.[137] A prospective case series that included 109 patients with aCSC or cCSC who were treated with low-dose aspirin, found that the treatment seemed to increase the rate of visual improvement, with fewer recurrences, compared to patients in an historical control group.[138]

**Carbonic anhydrase inhibitor:** A prospective non-randomized comparative trial by Pikkel et al. involving 15 patients with CSC who underwent treatment with oral acetazolamide and 7 untreated control patients found that acetazolamide accelerated both the improvement of subjective complaints and SRF resolution, but did not affect the final vision or the rate of recurrence of CSC.[139]

***Consensus Statement 4.7:*** *In the absence of large randomized controlled trials showing conclusive treatment benefit of oral medications in the management of CSC, the role of such medications is considered speculative. (Consensus score: 100 % [strongly agree: 50 %; agree: 50 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

Figs. 1 and 2 showcase an algorithmic approach reported by Feenstra et al.[3] in cases of aCSC and cCSC respectively.

MNV, macular neovascularization; CSC, central serous chorioretinopathy; FA, fluorescein angiography; FAF, fundus autofluorescence; ICGA, indocyanine green angiography; OCT, optical coherence tomography; OCT-A, optical coherence tomography angiography; PDT, photodynamic therapy; RPE, retinal pigment epithelium; SRF, subretinal fluid; VEGF, vascular endothelial growth factor.

MNV, macular neovascularization; CSC, central serous chorioretinopathy; FA, fluorescein angiography; FAF, fundus autofluorescence; ICGA, indocyanine green angiography; OCT, optical coherence tomography; OCT-A, optical coherence tomography angiography; PDT, photodynamic therapy; RPE, retinal pigment epithelium; SRF, subretinal fluid; VEGF, vascular endothelial growth factor

### 3.5. Section 5: future developments

Recent research has explored innovative evaluation techniques and emerging treatment strategies that hold promise for enhancing management outcomes for CSC. Artificial intelligence incorporation is expected to explode in the field of ophthalmology with artificial intelligence-based analysis of retinal multimodal imaging and treatment planning algorithms or strategies taking the centre-stage. Existing classification systems of CSC have been fraught with discrepancies, lack clarity, as well as uniformity in their descriptions, for which artificial intelligence could be of use. [140–142] Widespread interobserver variability has been noted among doctors, while reporting type of CSC suggesting importance of better disease definitions.

***Consensus Statement 5.1:*** *More clarity is needed in accurately defining what constitutes a particular type of CSC, because there is a wide variability among physicians while classifying CSC. (Consensus score: 100 % [strongly agree: 42.3 %; agree: 57.7 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*

***Consensus Statement 5.2:*** *Genetic factors linked to susceptibility for example mineralocorticoid receptor function hold promise as therapeutic targets, and these areas need to be explored further. (Consensus score: 92.3 % [strongly agree: 15.4 %; agree: 76.9 %; neutral: 7.7 %; disagree: 0 %; strongly disagree: 0 %])*

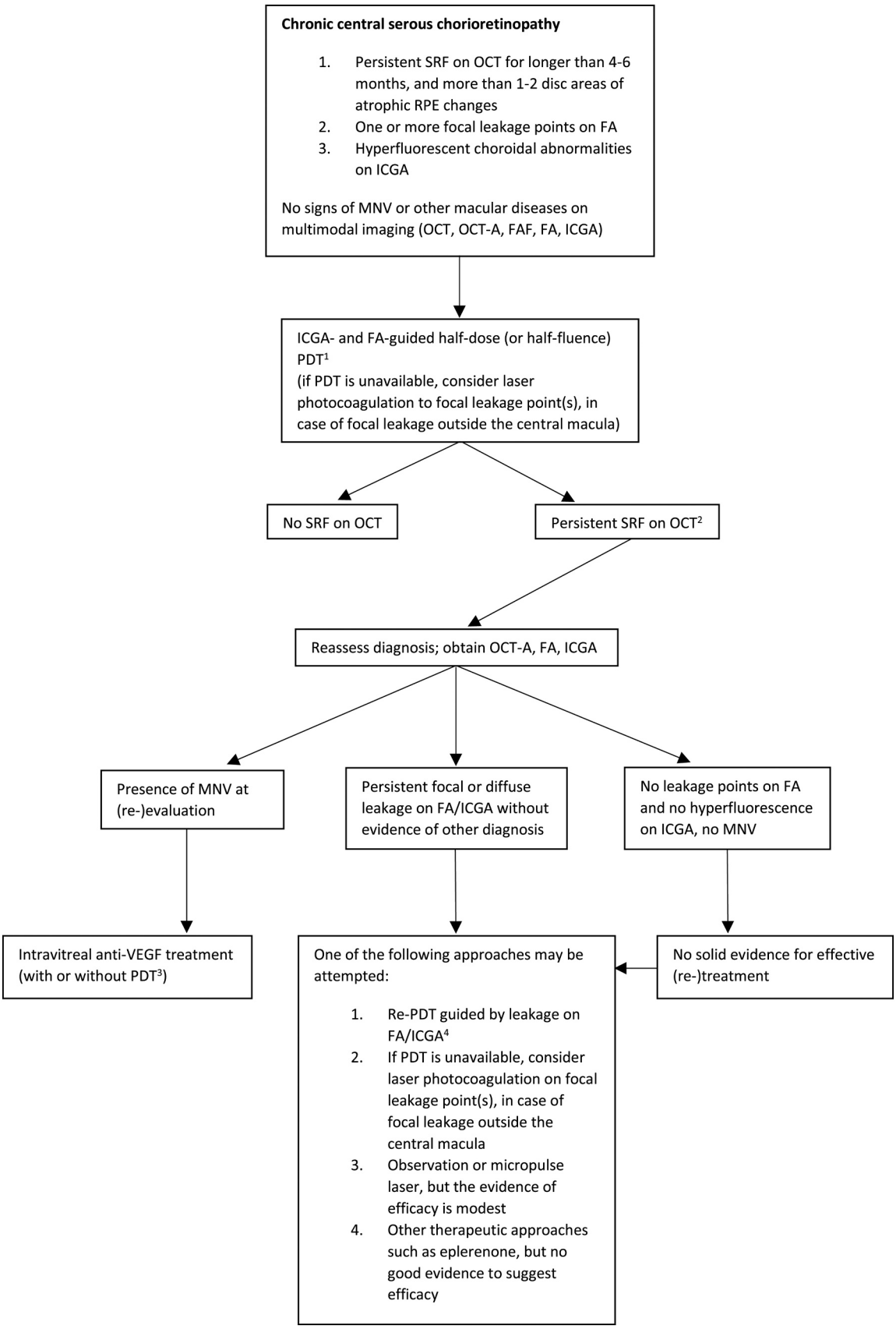
Novel agents, including oral formulations of curcumin and other anti-inflammatory compounds, are being explored to foster recovery in cCSC.[143] Additionally, genomic studies are focusing on elucidating genetic susceptibilities in patients with CSC, aiming to personalize treatment approaches based on individual risk profiles. [144,145] Bousquet et al. reported that eplerenone may be more effective in patients with baseline SFCT over 515 microns.[61] However, while smaller studies suggested benefits of MRAs in CSC, larger trials (VICI and SPECTRA) found no supporting evidence. [62,63] Abdelhakeem et al. reported that the approach of nanoemulsion formulations of medication such as the eplerenone holds the potential to increase drug availability.[146] While large randomized trials have not established a definitive role for eplerenone in CSC, its reformulation as a nanoemulsion for intraocular or suprachoroidal delivery could offer theoretical benefits by enhancing choroidal targeting and warrants further investigation. However, we are still in the early stage of these possibly pivotal steps, and due diligence is necessary as visual acuity of these patients is usually good before treatment is initiated.

At present, these therapeutic strategies remain a distant prospect; however, with ongoing advances in AI and machine learning, AI-driven CSC classification and the integration of real-world registry data appear increasingly achievable

***Consensus Statement 5.3:*** *Nanoemulsion drug delivery systems and formulations targeting a better ocular bioavailability of medicines may be explored further for safety and efficacy. (Consensus score: 84.6 % [strongly agree: 15.4 %; agree: 69.2 %; neutral: 15.4 %; disagree: 0 %; strongly disagree: 0 %])*

The Asia-Pacific region, comprising a large portion of the global population, faces shortage of verteporfin dye, as does Europe, reflecting a real-world situation of its limited availability, although some regions like the USA and Japan have no obvious shortage. Regional committees with government-pharmaceutical tie-ups could be established to address the shortages and arrange timely procurements of verteporfin dye since the shortages have been protracted.[147] With newer molecular targets being identified and incorporated in for example intravitreal agents, the use of these injections should be investigated for their potential efficacy in treating CSC, given the paucity or lack of availability/affordability of verteporfin, as some studies have shown some amount of promise.[123,148,149]

***Consensus Statement 5.4:*** *Regional experts-led, government-pharmaceutical tie-ups could help address the shortage problems of verteporfin dye till such a time that other treatment methods are under active exploration. (Consensus score: 100 % [strongly agree: 46.2 %; agree: 53.8 %; neutral: 0 %; disagree: 0 %; strongly disagree: 0 %])*




*(caption on next page)*

**Fig. 2.** Suggested management algorithm for chronic central serous chorioretinopathy. Note: if the patient is currently taking corticosteroids, discuss stopping their use prior to treatment.[1] Treat hyperfluorescent areas on indocyanine green angiography (ICGA) that correspond to the area of (focal) leakage on fluorescein angiography (FA) and subretinal fluid on optical coherence tomography (OCT). In case of multiple areas with focal leakage, a large spot including all areas can be used, or multifocal immediate sequential spots may be used, starting with the area including the fovea (if fovea is involved). [2] In case of only a small amount of residual subretinal fluid (SRF), a conservative approach may be followed, with a follow-up visit including OCT imaging after 1–3 months to see if SRF eventually resolves completely. In case of persistent/increased SRF at that stage, the downstream treatment decision pathway may be followed.[3] Half-dose or half-fluence photodynamic therapy (PDT) may be added in order to treat the choroidal dysfunction/pachychoroid factor of the disease, but limited data are available to support this combined treatment. When a neovascular component of polypoidal choroidal vasculopathy (aneurysmal type 1 neovascularization) is present, PDT (either full-dose, half-dose, or half-fluence) can also be added to anti-vascular endothelial growth factor treatment.[4] Another half-dose or half-fluence PDT can be performed, but full-dose with full-fluence PDT may also be considered.

## 4. Results of voting and discussion

Table 2 provides a summary of the key consensus statements along with the corresponding voting result.

*Consensus Score (C Score) was defined as the value of the summation of the 'strongly agree', and 'agree' percentages; C Score ≥ 75 % was considered 'consensus achieved' and C Score < 75 % was 'consensus not reached'.*

The consensus voting revealed strong agreement among experts on key aspects of CSC, including its definition, classification, pathophysiology, diagnosis, and treatment while also identifying areas requiring further clarity. Notably, 100 % of respondents agreed with the definition of CSC as a chorioretinal disorder characterized by serous detachment of the neurosensory retina and/or RPE in association with choroidal vascular hyperpermeability and venous congestion. This aligns with the widely accepted pathophysiological model proposed in multiple imaging and histopathological studies.[3,27]

A critical highlight was the unanimous endorsement (100 %) for the use of multimodal imaging—including OCT, FAF, FA, and ICGA—in the diagnosis and monitoring of CSC. Enhanced-depth and swept-source OCT, enabling choroidal profiling, were supported by 94.2 % of experts, confirming the importance of identifying pachychoroid features such as dilated outer choroidal vessels and thinning of the inner choroid, consistent with reports by Spaide et al. and Imamura et al.[5,27,150]

In the domain of pathophysiology, 100 % of respondents supported the role of choroidal venous congestion and structural abnormalities as key mechanisms, reflecting findings from ICGA-based studies.[25,151] Additionally, psychological stress and dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis were recognized as potential contributors (96.2 %), echoing previous work by Yannuzzi et al.[43,67,152]

Risk factors such as corticosteroid exposure were unanimously acknowledged (100 %) as major contributors. Similarly, the potential influence of genetic susceptibility (96.1 %) and systemic factors like obstructive sleep apnea (88.5 %) align with current literature suggesting a multifactorial etiology.[2,3,60]

Regarding management, there was strong consensus on observation being appropriate in most aCSC cases (100 %), while early intervention may be warranted in selected patients. For cCSC, 100 % endorsed half-dose PDT with verteporfin as the most evidence-based treatment, citing its superiority over subthreshold micropulse laser and the MRA eplerenone in randomized trials such as PLACE and SPECTRA.[63,101] Although this exercise re-confirms the established role of half-dose PDT in the management of CSC, the need to explore treatment options with subthreshold micropulse laser was agreed upon by 88.5 % of experts due to lack of universal availability or access to verteporfin dye. Though MRA like eplerenone had once shown promise, 100 % of experts acknowledged that large randomized controlled trials including VICI and SPECTRA did not support their use.[62,63]

Intravitreal anti-VEGF agents were supported (100 %) only in CSC cases complicated by a MNV, corroborating previous similar reports. [121,153,154]

Future directions emphasized the need for improved phenotypic classification (100 %), exploration of genetic underpinnings (92.3 %), and development of innovative therapies, including nanomedicine for ocular drug delivery (84.6 %).

## 5. Conclusions

This consensus initiative reflects a broad agreement among clinicians and researchers on the key aspects of diagnosing and managing CSC. Multimodal imaging was unanimously recognized as essential for accurate evaluation and decision-making. For cCSC, half-dose PDT emerged as the most consistently supported treatment option, offering both efficacy and safety. There was also a shared view that the use of oral agents such as MRAs remains speculative due to limited high-quality evidence. Similarly, anti-VEGF therapy was recommended only in cases where MNV is present.

The panel also acknowledged important areas where further research is needed—particularly the roles of genetic predisposition, systemic factors, and the need for more precise disease classification. Advancements in these areas could help to tailor treatments more effectively and improve long-term outcomes. Overall, the insights gained from this consensus provide a valuable framework for refining clinical practice and guiding the design of future research and therapeutic trials in CSC.

The study utilizes the Delphi process of iterations to arrive at a consensus and is thus subject to conformational bias and data gathering which may skew perception. Nevertheless, agreements among experts from diverse geographic regions do suggest a uniform understanding of the disease among experts and try to reflect upon mitigating regional variations which may confound doctors. At the moment, it seems that the choice of treatment for CSC is highly individualized. Clinicians must consider the duration of the disease, patient comorbidities, patient requirements, and the specific anatomical features observed on multimodal imaging including contrast dye imaging to establish its diagnosis and optimize therapeutic options and its outcomes.[91,155] Emerging therapeutic strategies and novel pharmacologic agents are being subjected to investigation and re-investigation, and their integration with current imaging modalities may further refine patient selection and treatment monitoring.[59,90,113]

## Consent

All authors consent to be co-authors of this manuscript.

## Declaration of competing interests

**Andrew Lotery:** Chief investigator Vici and Paint trials for CSC

**Timothy Y. Y. Lai:** Research grants from Bayer, Chengdu Kanghong Biotech, Novartis, and Roche; consulting fees from Astellas, Bayer, Boehringer Ingelheim, Novartis, Ocular

Therapeutix, Oculis, and Roche; lecture fees from Alcon, Bayer, Chengdu Kanghong

Biotech, Gaush Meditech, Novartis, and Roche

**K. Bailey Freund:** A consultant for EyePoint Pharmaceuticals and Regeneron Pharmaceuticals, Inc **Frank G. Holz:** Research Support/Grants: Acucela, Allergan, Annexon, Apellis, Astellas, Bayer, Bioeq, Centervue, Geuder, Genentech/Roche, Heidelberg Engineering, NightStar, Novartis, Science, Zeiss, 4D Molecular Therapeutics

Consulting/Advisory: Abbvie, Acucela, Alcon, Alexion, Annexon, Apellis, Astellas, Bayer, Biogen, Boehringer-Ingelheim, Cirrus, Galimedix, Genentech/Roche, Grayburg Vision, Heidelberg Engineering, Lin

**Table 2**
Voting results of consensus statements on CSC.

| Section | | C Score | Strongly Agree | Agree | Neutral | Disagree | Strongly Disagree |
|---|---|---|---|---|---|---|---|
| **1. Disease Entity: History, Terminology, and Definition Controversies** | | | | | | | |
| **1.1** | Central serous chorioretinopathy is a chorioretinal disorder characterized by serous detachment of the neurosensory retina and/or retinal pigment epithelium in the macula, associated with choroidal vascular hyperpermeability and venous congestion. It typically affects middle-aged men and may present as an acute, self-limiting episode or progress to a chronic form with persistent subretinal fluid and RPE damage. | **100 %** | 65.4 % | 34.6 % | 0 % | 0 % | 0 % |
| **1.2** | Standardized classification of CSC based on multimodal imaging is critical for improving diagnosis, treatment selection, and research comparability. | **100 %** | 65.4 % | 34.6 % | 0 % | 0 % | 0 % |
| **2. Pathophysiology Consensus and Controversies** | | | | | | | |
| **2.1** | CSC is linked to choroidal venous congestion and structural abnormalities, including dilated choroidal vessels, choroidal hyperpermeability, and increased scleral thickness, all of which contribute to its pathogenesis. | **100 %** | 57.7 % | 42.3 % | 0 % | 0 % | 0 % |
| **2.2** | Psychological stress and specific personality traits have been associated with an increased risk of CSC. Dysregulation of the hypothalamic-pituitary-adrenal axis has been proposed as a potential contributing mechanism. | **96.2 %** | 42.3 % | 53.9 % | 3.8 % | 0 % | 0 % |
| **2.3** | Obstructive sleep apnea syndrome (OSA) and shift work, in predisposed individuals may be potential triggers for CSC. | **88.5 %** | 30.8 % | 57.7 % | 11.5 % | 0 % | 0 % |
| **2.4** | Genetic factors have been associated with increased susceptibility to CSC. The precise mechanisms by which these variants contribute to disease development remain under investigation. | **96.1 %** | 26.9 % | 69.2 % | 3.9 % | 0 % | 0 % |
| **2.5** | Corticosteroid exposure is recognized as one of the major risk factors. Exposure to corticosteroids, whether exogenous or (stress-induced) endogenous, is one of the major risk factors for CSC due to its effects on choroidal circulation and RPE function. | **100 %** | 42.3 % | 57.7 % | 0 % | 0 % | 0 % |
| **2.6** | Large randomized controlled trials, including the VICI and SPECTRA studies, have not demonstrated a significant benefit of mineralocorticoid receptor antagonists such as eplerenone in the treatment of CSC. | **100 %** | 38.5 % | 61.5 % | 0 % | 0 % | 0 % |
| **2.7** | It is currently unclear if H. Pylori infection has a significant role in the pathogenesis of CSC. | **92.3 %** | 34.6 % | 57.7 % | 7.7 % | 0 % | 0 % |
| **3. Investigations and Diagnosis** | | | | | | | |
| **3.1** | Multimodal imaging (fundus photography, OCT, OCT-A, FAF, FA and ICGA), is ideal and essential for the accurate diagnosis, monitoring, and classification of CSC. | **100 %** | 57.7 % | 42.3 % | 0 % | 0 % | 0 % |
| **3.2** | Enhanced depth imaging OCT (EDI-OCT) and swept-source OCT (SS-OCT) enable choroidal thickness measurements and identification of dilated outer choroidal vessels. These metrics should be monitored in both eyes before and during treatment. | **96.2 %** | 38.5 % | 57.7 % | 3.8 % | 0 % | 0 % |
| **3.3** | Although multimodal imaging is ideal, SD-OCT with or without fundus photography/FAF may be enough to diagnose and monitor an acute first episode of CSC with no clinical signs of chronicity or complications. | **92.3 %** | 34.6 % | 57.7 % | 7.7 % | 0 % | 0 % |
| **3.4** | FA remains useful in detecting active leakage points and planning focal laser photocoagulation in chronic or non-resolving CSC cases. It may also be useful in guiding photodynamic therapy (PDT) treatment when ICGA facility is unavailable. | **96.2 %** | 50 % | 46.2 % | 3.8 % | 0 % | 0 % |
| **3.5** | ICGA plays a critical role in assessing choroidal hyperpermeability and differentiating CSC from mimicking conditions such as polypoidal choroidal vasculopathy. ICGA should be strongly recommended in all patients with suspected chronic CSC before initiating treatment. | **100 %** | 50 % | 50 % | 0 % | 0 % | 0 % |
| **4. Current Management** | | | | | | | |
| **4.1** | Observation is appropriate for most acute CSC (aCSC) cases, but early intervention may be considered in selected patients. | **100 %** | 50 % | 50 % | 0 % | 0 % | 0 % |
| **4.2** | Half-dose PDT with verteporfin is the most evidence-based and effective treatment for chronic central serous chorioretinopathy (cCSC), demonstrating superior anatomical and visual outcomes compared to subthreshold micropulse laser and eplerenone, with a favorable safety profile. | **100 %** | 57.7 % | 42.3 % | 0 % | 0 % | 0 % |
| **4.3** | Randomized controlled trials have demonstrated that photodynamic therapy is more effective than subthreshold micropulse laser in treating chronic CSC. However, subthreshold micropulse laser may be considered in cases where PDT is contraindicated or unavailable. | **92.3 %** | 42.3 % | 50 % | 7.7 % | 0 % | 0 % |
| **4.4** | Advancement of subthreshold laser therapies could offer potential treatment options and benefits to patients where PDT is not possible or unavailable. | **88.5 %** | 23.1 % | 65.4 % | 11.5 % | 0 % | 0 % |
| **4.5** | In established cases of cCSC, the benefits of intravitreal anti-VEGF injections as a treatment modality remain questionable, even when PDT is unavailable and its off-label use should be clearly explained. However, when cCSC is complicated by an MNV, intravitreal anti-VEGF treatment is the first line treatment. | **100 %** | 38.5 % | 61.5 % | 0 % | 0 % | 0 % |
| **4.6** | Pregnancy may get complicated by CSC. It can be safely monitored and usually resolves completely after childbirth, with most patients recovering good vision. | **100 %** | 34.6 % | 65.4 % | 0 % | 0 % | 0 % |
| **4.7** | In the absence of large randomized controlled trials showing conclusive treatment benefit of oral medications in the management of CSC, the role of such medications is considered speculative. | **100 %** | 50 % | 50 % | 0 % | 0 % | 0 % |
| **5. Future Developments** | | | | | | | |
| **5.1** | More clarity is needed in accurately defining what constitutes a particular type of CSC because there is a wide variability among physicians while classifying CSC. | **100 %** | 42.3 % | 57.7 % | 0 % | 0 % | 0 % |
| **5.2** | Genetic factors linked to susceptibility to CSC for example mineralocorticoid receptor function hold promise as therapeutic targets, and these areas need to be explored further. | **92.3 %** | 15.4 % | 76.9 % | 7.7 % | 0 % | 0 % |
| **5.3** | Nanoemulsion drug delivery systems and formulations targeting a better ocular bioavailability of medicines like eplerenone may be explored further for safety and efficacy. | **84.6 %** | 15.4 % | 69.2 % | 15.4 % | 0 % | 0 % |
| **5.4** | Regional experts-led, government-pharmaceutical tie-ups could help address the shortage problems of verteporfin dye till such a time that other treatment methods are under active exploration. | **100 %** | 46.2 % | 53.8 % | 0 % | 0 % | 0 % |

Bioscience, Merck, Novartis, OCULIS, Okuvision, Opthea, Oxurion, Sanofi, Science, Stealth Biotherapeutics, Topcon, Zeiss

## Acknowledgement / funding

Ms. May Lam Research and Education Fund of The Primasia International Eye Research Institute (PIERI) of The Chinese University of Hong Kong (Shenzhen); Shun Hing Education and Charity Fund; Bright Future Charitable Foundation Postgraduate Education Fund; Daniel & Co Scholarship.

## References

1. Wang M, Munch IC, Hasler PW, Prünte C, Larsen M. Central serous chorioretinopathy. *Acta Ophthalmol*. 2008 Mar;86(2):126–145.
2. Fung AT, Yang Y, Kam AW. Central serous chorioretinopathy: a review. *Clin Exp Ophthalmol*. 2023 Apr;51(3):243–270.
3. Feenstra HMA, van Dijk EHC, Cheung CMG, et al. Central serous chorioretinopathy: an evidence-based treatment guideline. *Prog Retin Eye Res*. 2024 Jan 31, 101236.
4. Imanaga N, Terao N, Nakamine S, et al. Scleral thickness in central serous chorioretinopathy. *Ophthalmol Retin*. 2021 Mar;5(3):285–291.
5. Spaide RF, Fisher YL, Ngo WK, Barbazetto I. REGIONAL SCLERAL THICKNESS AS a RISK FACTOR FOR CENTRAL SEROUS CHORIORETINOPATHY. *Retina*. 2022 July 1;42(7):1231–1237.
6. Keidel LF, Schworm B, Langer J, et al. Scleral thickness as a risk factor for central serous chorioretinopathy and pachychoroid neovasculopathy. *J Clin Med*. 2023 Apr 24;12(9):3102.
7. PentaVision [Internet]. [cited 2025 June 24]. New Insights on Management of Central Serous Chorioretinopathy. Available from: ⟨https://www.retinalphysician.com/issues/2021/may/new-insights-on-management-of-central-serous-chorioretinopathy/⟩.
8. van Dijk EHC, Feenstra HMA, Bjerager J, Grauslund J, Boon CJF, Subhi Y. Comparative efficacy of treatments for chronic central serous chorioretinopathy: a systematic review with network meta-analyses. *Acta Ophthalmol*. 2023 Mar;101(2):140–159.
9. Zhang X, Lim CZF, Chhablani J, Wong YM. Central serous chorioretinopathy: updates in the pathogenesis, diagnosis and therapeutic strategies. *Eye Vis (Lond)*. 2023 July 11;10(1):33.
10. Zarnegar A, Ong J, Matsyaraja T, Arora S, Chhablani J. Pathomechanisms in central serous chorioretinopathy: a recent update. *Int J Retin Vitr*. 2023 Jan 20;9(1):3.
11. Ruamviboonsuk P, Ng DSC, Chaikitmongkol V, et al. Consensus and guidelines on diagnosis and management of polypoidal choroidal vasculopathy (PCV) from the Asia-Pacific Vitreo-retina society (APVRS). *Asia Pac J Ophthalmol (Philos)*. 2025;14(1), 100144.
12. Elsevier eBooks+: Ryan's Retina [Internet]. [cited 2024 Feb 26]. Available from: ⟨https://bookshelf.health.elsevier.com/reader/books/9780323722155/epubcfi/6/246⟩[%3Bvnd.vst.idref%3DCHP0073_1530-1554_B9780323722131000822_0]!/4/2[c0073]/4[otl0010]/4/2/8[li0055]/6/10[li0080]/4[cref80070].
13. Horniker E. Central angiospastic retinitis. *Klin Mon Augenheilkd*. 1937;98.
14. Kitahara S. Ueber klinische beobachtungen bei der in Japan häufig vorkommenden chorioretinitis centralis serosa. *Klin Mon Augenheilkd*. 1936;97:345–362.
15. Kraupa E. Die retinitis centralis annularis. Z augenheilkd. 1923;50:335–343.
16. Horniker E. U di una forma di retinite centrale di origine vasoneurotica. *Ann Ottalmol*. 1927;55:578–600, 830–40, 865–83.
17. Chhablani J. Central Serous Chorioretinopathy. 1st ed. Elsevier; 2019. XXXIX–XLI.
18. Daruich A, Matet A, Dirani A, et al. Central serous chorioretinopathy: recent findings and new physiopathology hypothesis. *Prog Retin Eye Res*. 2015 Sept;48:82–118.
19. van Dijk EHC, Boon CJF. Serous business: delineating the broad spectrum of diseases with subretinal fluid in the macula. *Prog Retin Eye Res*. 2021 Sept;84, 100955.
20. Chhablani J, Cohen FB, Aymard P, et al. Multimodal Imaging-Based central serous chorioretinopathy classification. *Ophthalmol Retin*. 2020 Nov 1;4(11):1043–1046.
21. Ho M, Li G, Mak A, Ng D, Iu L, Lai F. Applications of multimodal imaging in central serous chorioretinopathy evaluation. *J Ophthalmol*. 2021;2021, 9929864.
22. Chen G, Tzekov R, Li W, Jiang F, Mao S, Tong Y. Subfoveal choroidal thickness in central serous chorioretinopathy: a Meta-Analysis. *PLoS One*. 2017;12(1), e0169152.
23. Cheung CMG, Dansingani KK, Koizumi H, et al. Pachychoroid disease: review and update. *Eye (Lond)*. 2025 Apr;39(5):819–834.
24. Pauleikhoff LJB, Diederen RMH, Chang-Wolf JM, et al. Choroidal vascular changes on ultrawidefield indocyanine Green angiography in central serous chorioretinopathy: CERTAIN study report 1. *Ophthalmol Retin*. 2024 Mar;8(3):254–263.
25. Pauleikhoff LJB, Diederen RMH, Chang-Wolf JM, et al. Choroidal hyperpermeability patterns correlate with disease severity in central serous chorioretinopathy: CERTAIN study report 2. *Acta Ophthalmol*. 2024 Sept;102(6):e946–e955.
26. Semeraro F, Morescalchi F, Russo A, et al. Central serous chorioretinopathy: pathogenesis and management. *Clin Ophthalmol*. 2019;13:2341–2352.
27. Spaide RF, Gemmy Cheung CM, Matsumoto H, et al. Venous overload choroidopathy: a hypothetical framework for central serous chorioretinopathy and allied disorders. *Prog Retin Eye Res*. 2022 Jan;86, 100973.
28. Spaide RF. The midwest, downtown, and Sattler's layer: when everyone knows what we mean, but nobody knows where it is. *Retina*. 2025 May 1;45(5):807–809.
29. Terao N, Imanaga N, Wakugawa S, et al. CILIOCHOROIDAL EFFUSION IN CENTRAL SEROUS CHORIORETINOPATHY. *Retina*. 2022 Apr;42(4):730–737.
30. Imanaga N, Terao N, Sonoda S, et al. Relationship between scleral thickness and choroidal structure in central serous chorioretinopathy. *Invest Ophthalmol Vis Sci*. 2023 Jan 3;64(1):16.
31. Imanaga N, Terao N, Wakugawa S, et al. Scleral thickness in simple versus complex central serous chorioretinopathy. *Am J Ophthalmol*. 2024 May;261:103–111.
32. Piccolino FC, Arrigo A, Forte P, et al. Intraocular pressure and ocular perfusion pressure in central serous chorioretinopathy. *Am J Ophthalmol*. 2025 Aug 22;S0002-9394(25), 00458-1.
33. Yun C, Han JY, Cho S, Hwang SY, Kim SW, Oh J. OCULAR PERFUSION PRESSURE AND CHOROIDAL THICKNESS IN CENTRAL SEROUS CHORIORETINOPATHY AND PIGMENT EPITHELIOPATHY. *Retina*. 2019 Jan;39(1):143–149.
34. Schocket Niknam RM, Metelitsina LS, DuPont T, Grunwald JC. JE. Effect of hypertension on foveolar choroidal haemodynamics. *Br J Ophthalmol*. 2004 Oct;88(10):1263–1265.
35. Cardillo Piccolino F, Lupidi M, Cagini C, et al. Choroidal vascular reactivity in central serous chorioretinopathy. *Invest Ophthalmol Vis Sci*. 2018 Aug 1;59(10):3897–3905.
36. Piccolino FC, Fruttini D, Eandi C, et al. Vigorous physical activity as a risk factor for central serous chorioretinopathy. *Am J Ophthalmol*. 2022 Dec;244:30–37.
37. Brinks J, van Dijk EHC, Meijer OC, Schlingemann RO, Boon CJF. Choroidal arteriovenous anastomoses: a hypothesis for the pathogenesis of central serous chorioretinopathy and other pachychoroid disease spectrum abnormalities. *Acta Ophthalmol*. 2022 Dec;100(8):946–959.
38. Hanumunthadu D, Van Dijk EHC, Gangakhedkar S, et al. Gender variation in central serous chorioretinopathy. *Eye (Lond)*. 2018 Nov;32(11):1703–1709.
39. Cheung CMG, Lee WK, Koizumi H, Dansingani K, Lai TYY, Freund KB. Pachychoroid disease. *Eye (Lond)*. 2019 Jan;33(1):14–33.
40. Dansingani KK, Balaratnasingam C, Naysan J, Freund KB. EN FACE IMAGING OF PACHYCHOROID SPECTRUM DISORDERS WITH SWEPT-SOURCE OPTICAL COHERENCE TOMOGRAPHY. *Retina*. 2016 Mar;36(3):499–516.
41. Pauleikhoff LJB, Chang-Wolf JM, Diederen RMH, et al. ULTRAWIDEFIELD INDOCYANINE Green ANGIOGRAPHIC CHANGES AFTER PHOTODYNAMIC THERAPY IN CENTRAL SEROUS CHORIORETINOPATHY: certain study report 3. *Retina*. 2025 June 1;45(6):1143–1150.
42. Maruko I, Iida T, Sugano Y, Ojima A, Ogasawara M, Spaide RF. Subfoveal choroidal thickness after treatment of central serous chorioretinopathy. *Ophthalmology*. 2010 Sept;117(9):1792–1799.
43. Yannuzzi LA. Type-A behavior and central serous chorioretinopathy. *Retina*. 1987;7(2):111–131.
44. Fok ACT, Chan PPM, Lam DSC, Lai TYY. Risk factors for recurrence of serous macular detachment in untreated patients with central serous chorioretinopathy. *Ophthalmic Res*. 2011;46(3):160–163.
45. Conrad R, Weber NF, Lehnert M, Holz FG, Liedtke R, Eter N. Alexithymia and emotional distress in patients with central serous chorioretinopathy. *Psychosomatics*. 2007;48(6):489–495.
46. Scarinci F, Ghiciuc CM, Patacchioli FR, Palmery M, Parravano M. Investigating the hypothesis of stress system dysregulation as a risk factor for central serous chorioretinopathy: a literature Mini-Review. *Curr Eye Res*. 2019 June;44(6):583–589.
47. 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 (2010–2020). *Med (Kaunas)*. 2021 June 16;57(6):628.
48. 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 June;97(4):e572–e579.
49. Kumar M, van Dijk EHC, Raman R, et al. Stress and vision-related quality of life in acute and chronic central serous chorioretinopathy. *BMC Ophthalmol*. 2020 Mar 6;20(1):90.
50. Eom Y, Oh J, Kim SW, Huh K. Systemic factors associated with central serous chorioretinopathy in Koreans. *Korean J Ophthalmol*. 2012 Aug;26(4):260–264.
51. Brodie FL, Charlson ES, Aleman TS, et al. Obstructive sleep apnea and central serous chorioretinopathy. *Retina*. 2015 Feb;35(2):238–243.
52. Bousquet E, Dhundass M, Lehmann M, et al. Shift work: a risk factor for central serous chorioretinopathy. *Am J Ophthalmol*. 2016 May 1;165:23–28.
53. Giannopoulos K, Gazouli M, Chatzistefanou K, Bakouli A, Moschos MM. The genetic background of central serous chorioretinopathy: a review on central serous chorioretinopathy genes. *J Genom*. 2021;9:10–19.
54. 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 May 3;16(1):4127.
55. Mori Y, van Dijk EHC, Miyake M, et al. Genome-wide association and multi-omics analyses provide insights into the disease mechanisms of central serous chorioretinopathy. *Sci Rep*. 2025 Mar 17;15:9158.
56. 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 May 1;141(5):449–457.
57. Chen ZJ, Ng DS, Ho M, et al. Genetic associations of central serous chorioretinopathy subtypes, neovascular age-related macular degeneration, and

polypoidal choroidal vasculopathy. *Asia Pac J Ophthalmol.* 2024;13(1), 100003. https://doi.org/10.1016/j.apjo.2023.100003.
58. Chen LJ, Chen ZJ, Pang CP. Latest development on genetics of common retinal diseases. *Asia Pac J Ophthalmol.* 2023;12(2):228–251. https://doi.org/10.1097/APO.0000000000000592.
59. R K., S C., J S., Cjf B., S S., A L. Central serous chorioretinopathy: An update on risk factors, pathophysiology and imaging modalities. Progress in retinal and eye research [Internet]. 2020 Nov [cited 2025 Apr 14];79. Available from: ⟨https://pubmed.ncbi.nlm.nih.gov/32407978/⟩.
60. Ge G, Zhang Y, Zhang Y, Xu Z, Zhang M. Corticosteroids usage and central serous chorioretinopathy: a meta-analysis. *Graefes Arch Clin Exp Ophthalmol.* 2020 Jan;258(1):71–77.
61. Bousquet E, Dhundass M, Lejoyeux R, et al. Predictive factors of response to mineralocorticoid receptor antagonists in nonresolving central serous chorioretinopathy. *Am J Ophthalmol.* 2019 Feb;198:80–87.
62. Lotery A, Sivaprasad S, O'Connell A, et al. Eplerenone for chronic central serous chorioretinopathy in patients with active, previously untreated disease for more than 4 months (VICI): a randomised, double-blind, placebo-controlled trial. *Lancet.* 2020 Jan 25;395(10220):294–303.
63. van Rijssen TJ, van Dijk EHC, Tsonaka R, et al. Half-Dose photodynamic therapy versus eplerenone in chronic central serous chorioretinopathy (SPECTRA): a randomized controlled trial. *Am J Ophthalmol.* 2022 Jan;233:101–110.
64. DuPont JJ, Jaffe IZ. 30 YEARS OF THE MINERALOCORTICOID RECEPTOR: the role of the mineralocorticoid receptor in the vasculature. *J Endocrinol.* 2017 July;234(1):T67–T82.
65. Dutzmann J, Bauersachs J, Sedding DG. Evidence for the use of mineralocorticoid receptor antagonists in the treatment of coronary artery disease and post-angioplasty restenosis. *Vasc Pharm.* 2017 Dec 21;S1537-1891(17), 30281-30281.
66. van der Heijden CDCC, Deinum J, Joosten LAB, Netea MG, Riksen NP. The mineralocorticoid receptor as a modulator of innate immunity and atherosclerosis. *Cardiovasc Res.* 2018 June 1;114(7):944–953.
67. Juruena MF, Werne Baes CV, Menezes IC, Graeff FG. Early life stress in depressive patients: role of glucocorticoid and mineralocorticoid receptors and of hypothalamic-pituitary-adrenal axis activity. *Curr Pharm Des.* 2015;21(11):1369–1378.
68. Mansuetta CC, Mason JO, Swanner J, et al. An association between central serous chorioretinopathy and gastroesophageal reflux disease. *Am J Ophthalmol.* 2004 June;137(6):1096–1100.
69. Chen SN, Lian I, Chen YC, Ho JD. Increased incidence of peptic ulcer disease in central serous chorioretinopathy patients: a population-based retrospective cohort study. *Retina.* 2015 Feb;35(2):231–237.
70. Giusti C. Association of *helicobacter pylori* with central serous chorioretinopathy: hypotheses regarding pathogenesis. *Med Hypotheses.* 2004 Jan 1;63(3):524–527.
71. Bagheri M, Rashe Z, Ahoor MH, Somi MH. Prevalence of helicobacter pylori infection in patients with central serous chorioretinopathy: a review. *Med Hypothesis Discov Innov Ophthalmol.* 2017;6(4):118–124.
72. Smolensky M.H., Hermida R.C., Reinberg A., Sackett-Lundeen L., Portaluppi F. Circadian disruption: New clinical perspective of disease pathology and basis for chronotherapeutic intervention. Chronobiology International [Internet]. 2016 Sept 13 [cited 2025 June 1]; Available from: ⟨https://www.tandfonline.com/doi/abs/10.1080/07420528.2016.1184678⟩.
73. Nkrumah G, Maltsev DS, Manuel PEA, et al. Current choroidal imaging findings in central serous chorioretinopathy. *Vis (Basel).* 2020 Oct 16;4(4):44.
74. Szeto SK, Hui VWK, Siu V, et al. Recent advances in clinical applications of imaging in retinal diseases. *Asia Pac J Ophthalmol.* 2023;12(2):252–263. https://doi.org/10.1097/APO.0000000000000584.
75. Chhablani J, Behar-Cohen F. Central serous chorioretinopathy international group. Validation of central serous chorioretinopathy multimodal imaging-based classification system. *Graefes Arch Clin Exp Ophthalmol.* 2022 Apr;260(4):1161–1169.
76. Montero JA. Ruiz-Moreno JM. Optical coherence tomography characterisation of idiopathic central serous chorioretinopathy. *Br J Ophthalmol.* 2005 May;89(5):562–564.
77. Ferrara D, Mohler KJ, Waheed N, et al. En face enhanced-depth swept-source optical coherence tomography features of chronic central serous chorioretinopathy. *Ophthalmology.* 2014 Mar;121(3):719–726.
78. Lee WJ, Lee JH, Lee BR. Fundus autofluorescence imaging patterns in central serous chorioretinopathy according to chronicity. *Eye (Lond).* 2016 Oct;30(10):1336–1342.
79. Ayata A, Tatlipinar S, Kar T, Unal M, Ersanli D, Bilge AH. Near-infrared and short-wavelength autofluorescence imaging in central serous chorioretinopathy. *Br J Ophthalmol.* 2009 Jan;93(1):79–82.
80. Min JY, Lv Y, Yu S, Gong YY. Findings of OCT-angiography compared to fluorescein and indocyanine Green angiography in central serous chorioretinopathy. *Lasers Surg Med.* 2018 Dec;50(10):987–993.
81. Hu J, Qu J, Piao Z, et al. Optical coherence tomography angiography compared with indocyanine Green angiography in central serous chorioretinopathy. *Sci Rep.* 2019 Apr 16;9(1):6149.
82. Teussink MM, Breukink MB, van Grinsven MJJP, et al. OCT angiography compared to fluorescein and indocyanine Green angiography in chronic central serous chorioretinopathy. *Invest Ophthalmol Vis Sci.* 2015 Aug;56(9):5229–5237.
83. Chhablani J, Anantharaman G, Behar-Cohen F, Boon C, Manayath G, Singh R. Management of central serous chorioretinopathy: expert panel discussion. *Indian J Ophthalmol.* 2018 Dec;66(12):1700–1703.
84. 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 Nov;102(7):749–758.
85. Hu J, Qu J, Li M, et al. OPTICAL COHERENCE TOMOGRAPHY ANGIOGRAPHY-GUIDED PHOTODYNAMIC THERAPY FOR ACUTE CENTRAL SEROUS CHORIORETINOPATHY. *Retina.* 2021 Jan 1;41(1):189–198.
86. Koytak A, Bayraktar H, Ozdemir H. Fluorescein angiography as a primary guide for reduced-fluence photodynamic therapy for the treatment of chronic central serous chorioretinopathy. *Int Ophthalmol.* 2020 July;40(7):1807–1813.
87. Ebrahimiadib N, Mirzaei A, Esfandiarifard S, et al. Comparison of the fluorescein angiography-guided and indocyanine Green angiography-guided photodynamic therapy in the treatment of non-resolving central serous chorioretinopathy. *Sci Rep.* 2023 Jan 30;13(1):1682.
88. Ladas ID, Andreanos KD, Ladas DS, Moschos MM, Rotsos T, Kotsolis AI. Three-Year results of fluorescein Angiography-Guided standard photodynamic therapy with multiple spots for central serous chorioretinopathy. *Ophthalmol Retin.* 2018 July;2(7):703–711.
89. Breukink MB, Dingemans AJ, den, et al. Chronic central serous chorioretinopathy: long-term follow-up and vision-related quality of life. *Clin Ophthalmol.* 2017;11:39–46.
90. Salehi M, Wenick AS, Law HA, Evans JR, Gehlbach P. Interventions for central serous chorioretinopathy: a network meta-analysis. *Cochrane Database Syst Rev.* 2015 Dec 22;2015(12), CD011841.
91. Sartini F, Figus M, Nardi M, Casini G, Posarelli C. Non-resolving, recurrent and chronic central serous chorioretinopathy: available treatment options. *Eye (Lond).* 2019 July;33(7):1035–1043.
92. Behnia M, Khabazkhoob M, Aliakbari S, Abadi AE, Hashemi H, Pourvahidi P. Improvement in visual acuity and contrast sensitivity in patients with central serous chorioretinopathy after macular subthreshold laser therapy. *Retina.* 2013 Feb;33(2):324–328.
93. Matet A, Daruich A, Zola M, Behar-Cohen F. RISK FACTORS FOR RECURRENCES OF CENTRAL SEROUS CHORIORETINOPATHY. *Retina.* 2018 July;38(7):1403–1414.
94. Mohabati D, Boon CJF, Yzer S. Risk of recurrence and transition to chronic disease in acute central serous chorioretinopathy. *Clin Ophthalmol.* 2020;14:1165–1175.
95. Chan WM, Lai TYY, Lai RYK, Liu DTL, Lam DSC. Half-dose verteporfin photodynamic therapy for acute central serous chorioretinopathy: one-year results of a randomized controlled trial. *Ophthalmology.* 2008 Oct;115(10):1756–1765.
96. Iacono P, Da Pozzo S, Varano M, Parravano M. Photodynamic therapy with verteporfin for chronic central serous chorioretinopathy: a review of data and efficacy. *Pharm (Basel).* 2020 Oct 29;13(11):349.
97. Ma J, Meng N, Xu X, Zhou F, Qu Y. System review and meta-analysis on photodynamic therapy in central serous chorioretinopathy. *Acta Ophthalmol.* 2014 Dec;92(8):e594–e601.
98. Vasconcelos H, Marques I, Santos AR, et al. Long-term chorioretinal changes after photodynamic therapy for chronic central serous chorioretinopathy. *Graefes Arch Clin Exp Ophthalmol.* 2013 July;251(7):1697–1705.
99. Feenstra HMA, Diederen RMH, Lamme MJCM, et al. INCREASING EVIDENCE FOR THE SAFETY OF FOVEA-INVOLVING HALF-DOSE PHOTODYNAMIC THERAPY FOR CHRONIC CENTRAL SEROUS CHORIORETINOPATHY. *Retina.* 2023 Mar 1;43(3):379–388.
100. Chang-Wolf JM, van Dijk EHC, El Boujdaini K, et al. Outcome and safety of multiple photodynamic therapy sessions in chronic central serous chorioretinopathy. *Acta Ophthalmol.* 2025 Apr 17.
101. 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: the PLACE trial. *Ophthalmology.* 2018 Oct;125(10):1547–1555.
102. van Rijssen TJ, van Dijk EHC, Scholz P, et al. Crossover to photodynamic therapy or micropulse laser after failure of primary treatment of chronic central serous chorioretinopathy: the REPLACE trial. *Am J Ophthalmol.* 2020 Aug;216:80–89.
103. van Rijssen 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 Sept;205:1–10.
104. Brelen ME, Ho M, Li S, et al. Comparing Half-Dose photodynamic therapy with subthreshold micropulse laser for the treatment of central serous chorioretinopathy. *Ophthalmol Retin.* 2024 May;8(5):490–498.
105. Feenstra HMA, van Dijk EHC, van Rijssen TJ, et al. Crossover to Half-Dose photodynamic therapy or eplerenone in chronic central serous chorioretinopathy patients. *Ophthalmol Retin.* 2022 Oct;6(10):930–938.
106. van Rijssen TJ, Hahn LC, van Dijk EHC, et al. RESPONSE OF CHOROIDAL ABNORMALITIES TO PHOTODYNAMIC THERAPY VERSUS MICROPULSE LASER IN CHRONIC CENTRAL SEROUS CHORIORETINOPATHY: place trial report no. 4. *Retina.* 2021 Oct 1;41(10):2122–2131.
107. Feenstra HMA, Hahn LC, van Rijssen TJ, et al. EFFICACY OF HALF-DOSE PHOTODYNAMIC THERAPY VERSUS HIGH-DENSITY SUBTHRESHOLD MICROPULSE LASER FOR TREATING PIGMENT EPITHELIAL DETACHMENTS IN CHRONIC CENTRAL SEROUS CHORIORETINOPATHY. *Retina.* 2022 Apr 1;42(4):721–729.
108. Azuma K, Tanaka N, Aoki S, et al. Long-term visual outcomes in pachychoroid spectrum diseases and its associating factors of eyes with chronic central serous chorioretinopathy. *Sci Rep.* 2023 Dec 11;13, 21876.
109. Larsson JME, Boberg-Ans LC, Vangsted A, et al. Topical non-steroidal anti-inflammatory drugs for central serous chorioretinopathy: a systematic review and meta-analysis. *Acta Ophthalmol.* 2023 Aug 8.

110. Lim JI, Glassman AR, Aiello LP, et al. Collaborative retrospective macula society study of photodynamic therapy for chronic central serous chorioretinopathy. *Ophthalmology*. 2014 May;121(5):1073–1078.
111. Nicolò M, Desideri LF, Vagge A, Traverso CE. Current pharmacological treatment options for central serous chorioretinopathy: a review. *Pharm (Basel)*. 2020 Sept 23;13(10):264.
112. Gergely R, Kovács I, Schneider M, et al. MINERALOCORTICOID RECEPTOR ANTAGONIST TREATMENT IN BILATERAL CHRONIC CENTRAL SEROUS CHORIORETINOPATHY: a COMPARATIVE STUDY OF EXUDATIVE AND NONEXUDATIVE FELLOW EYES. *Retina*. 2017 June;37(6):1084–1091.
113. Chatziralli I, Vlachodimitropoulou A, Daoula C, et al. Eplerenone in the treatment of central serous chorioretinopathy: a review of the literature. *Int J Retin Vitr*. 2018; 4:33.
114. Feenstra HMA, van Dijk EHC, van Rijssen TJ, et al. Long-term follow-up of chronic central serous chorioretinopathy patients after primary treatment of oral eplerenone or half-dose photodynamic therapy and crossover treatment: SPECTRA trial report no. 3. *Graefes Arch Clin Exp Ophthalmol*. 2023 Mar;261(3):659–668.
115. Abrishami M, Mousavi M, Hosseini SM, Norouzpour A. Treatment of chronic central serous chorioretinopathy with oral methotrexate. *J Ocul Pharm Ther*. 2015 Oct;31(8):468–475.
116. Petkovsek D, Singh RP. *Chapter 21 - oral agents in the treatment of central serous chorioretinopathy. In: Chhablani J, editor. Central Serous Chorioretinopathy [Internet]*. Academic Press; 2019 [cited 2025 Apr 14]. p. 293–303. Available from: ⟨https://www.sciencedirect.com/science/article/pii/B9780128168004000206⟩.
117. Scholz P, Ersoy L, Boon CJF, Fauser S. Subthreshold micropulse laser (577 nm) treatment in chronic central serous chorioretinopathy. *Ophthalmologica*. 2015;234 (4):189–194.
118. Büttner M, Luger B, Abou Moulig W, et al. Selective retina therapy (SRT) in patients with therapy refractory persistent acute central serous chorioretinopathy (CSC): 3 months functional and morphological results. *Graefes Arch Clin Exp Ophthalmol*. 2021 June;259(6):1401–1410.
119. Lu HQ, Wang EQ, Zhang T, Chen YX. Photodynamic therapy and anti-vascular endothelial growth factor for acute central serous chorioretinopathy: a systematic review and meta-analysis. *Eye (Lond)*. 2016 Jan;30(1):15–22.
120. Park SU, Lee SJ, Kim M. Intravitreal anti-vascular endothelial growth factor versus observation in acute central serous chorioretinopathy: one-year results. *Korean J Ophthalmol*. 2014 Aug;28(4):306–313.
121. Radke N, Kalamkar C, Mukherjee A, Radke S. Intravitreal Ziv-Aflibercept in treatment of Naïve chronic central serous chorioretinopathy related choroidal neovascular membrane. *Case Rep Ophthalmol Med*. 2017;2017:5036248.
122. Pitcher JD, Witkin AJ, DeCroos FC, Ho AC. A prospective pilot study of intravitreal aflibercept for the treatment of chronic central serous chorioretinopathy: the CONTAIN study. *Br J Ophthalmol*. 2015 June;99(6):848–852.
123. Lim JW, Kim MU. The efficacy of intravitreal bevacizumab for idiopathic central serous chorioretinopathy. *Graefes Arch Clin Exp Ophthalmol*. 2011 July;249(7): 969–974.
124. Pauleikhoff LJB, Diederen RMH, Feenstra HMA, Schlingemann RO, van Dijk EHC, Boon CJF. SINGLE-SESSION BILATERAL REDUCED-SETTINGS PHOTODYNAMIC THERAPY FOR BILATERAL CHRONIC CENTRAL SEROUS CHORIORETINOPATHY. *Retina*. 2023 Aug 1;43(8):1356–1363.
125. Ochinciuc R, Munteanu M, Baltă G, Baltă F. Central serous chorioretinopathy in pregnancy. *Rom J Ophthalmol*. 2022;66(4):382–385.
126. Hirji N, Watt L, Richardson E. Central serous chorioretinopathy secondary to childbirth. *BMJ Case Rep*. 2010 Oct 3;2010. bcr1020103387.
127. Said-Ahmed K, Moustafa G, Fawzy M. Incidence and natural course of symptomatic central serous chorioretinopathy in pregnant women in a maternity hospital in Kuwait. *Middle East Afr J Ophthalmol*. 2012;19(3):273–276.
128. Kim JW, Park MH, Kim YJ, Kim YT. Comparison of subfoveal choroidal thickness in healthy pregnancy and pre-eclampsia. *Eye*. 2016 Mar;30(3):349–354.
129. Yu J, Li L, Jiang C, Chang Q, Xu G. Clinical characteristics of Pregnancy-Associated central serous chorioretinopathy in the Chinese population. *J Ophthalmol*. 2021 Dec 16;2021, 5580075.
130. Bousquet E, Zhao M, Daruich A, Behar-Cohen F. Mineralocorticoid antagonists in the treatment of central serous chorioetinopathy: review of the pre-clinical and clinical evidence. *Exp Eye Res*. 2019 Oct;187, 107754.
131. Leclercq B, Weiner A, Zola M, et al. The choroidal nervous system: a link between mineralocorticoid receptor and pachychoroid. *Acta Neuropathol*. 2023 Nov;146(5): 747–766.
132. Brinks J, van Dijk EHC, Kiełbasa SM, et al. The cortisol response of Male and female choroidal endothelial cells: implications for central serous chorioretinopathy. *J Clin Endocrinol Metab*. 2022 Jan 18;107(2):512–524.
133. Brinks J, van Dijk EHC, Klaassen I, et al. Exploring the choroidal vascular labyrinth and its molecular and structural roles in health and disease. *Prog Retin Eye Res*. 2022 Mar;87, 100994.
134. Venkatesh R., Agarwal M., Kantha M. Efficacy of oral rifampicin in chronic central serous chorioretinopathy. Therapeutic Advances in Ophthalmology [Internet]. 2018 Oct 16 [cited 2025 June 1]; Available from: ⟨https://journals.sagepub.com/doi/full/10.1177/2515841418807130⟩.
135. Shulman S, Goldenberg D, Schwartz R, et al. Oral rifampin treatment for longstanding chronic central serous chorioretinopathy. *Graefes Arch Clin Exp Ophthalmol*. 2016 Jan;254(1):15–22.
136. Kurup SK, Oliver A, Emanuelli A, Hau V, Callanan D. Low-dose methotrexate for the treatment of chronic central serous chorioretinopathy: a retrospective analysis. *Retina*. 2012;32(10):2096–2101.
137. Iijima H, Iida T, Murayama K, Imai M, Gohdo T. Plasminogen activator inhibitor 1 in central serous chorioretinopathy. *Am J Ophthalmol*. 1999 Apr 1;127(4):477–478.
138. Caccavale A, Romanazzi F, Imparato M, Negri A, Morano A, Ferentini F. Low-dose aspirin as treatment for central serous chorioretinopathy. *Clin Ophthalmol*. 2010 Aug 9;4:899–903.
139. Pikkel J, Beiran I, Ophir A, Miller B. Acetazolamide for central serous retinopathy. *Ophthalmology*. 2002 Sept 1;109(9):1723–1725.
140. Lim JI, Rachitskaya AV, Hallak JA, Gholami S, Alam MN. Artificial intelligence for retinal diseases. *Asia Pac J Ophthalmol*. 2024;13(4), 100096. https://doi.org/10.1016/j.apjo.2024.100096.
141. Feng X, Xu K, Luo MJ, et al. Latest developments of generative artificial intelligence and applications in ophthalmology. *Asia Pac J Ophthalmol*. 2024;13(4), 100090. https://doi.org/10.1016/j.apjo.2024.100090.
142. Clark P, Oermann EK, Chen D, Al-Aswad LA. Federated AI, current state, and future potential. *Asia Pac J Ophthalmol*. 2023;12(3):310–314. https://doi.org/10.1097/APO.0000000000000614.
143. Fusi-Rubiano W, Saedon H, Patel V, Yang YC. Oral medications for central serous chorioretinopathy: a literature review. *Eye (Lond)*. 2020 May;34(5):809–824.
144. Hosoda Y, Yamashiro K, Miyake M, et al. Predictive genes for the prognosis of central serous chorioretinopathy. *Ophthalmol Retin*. 2019 Nov;3(11):985–992.
145. Hayashida M, Miki A, Nakai S, et al. Genetic factors associated with treatment response to reduced-fluence photodynamic therapy for chronic central serous chorioretinopathy. *Mol Vis*. 2020;26:505–509.
146. Abdelhakeem E, El-Nabarawi M, Shamma R. Eplerenone repurposing in management of chorioretinopathy: mechanism, nanomedicine-based delivery applications and future trends. *Br J Clin Pharm*. 2022 June;88(6):2665–2672.
147. Sirks MJ, Subhi Y, Rosenberg N, et al. Perspectives and update on the global shortage of verteporfin (Visudyne®). *Ophthalmol Ther*. 2024 July;13(7): 1821–1831.
148. Zhao T, Li J, Wang Y, Guo X, Sun Y. Intravitreal injections with anti-VEGF agent aflibercept versus subthreshold micropulse laser for chronic central serous chorioretinopathy: the alternative treatment regimens for verteporfin-shortage in China. *Ann Med*. 2023 Dec;55(1), 2227424.
149. Sirks MJ, van Dijk EHC, Rosenberg N, et al. Clinical impact of the worldwide shortage of verteporfin (Visudyne®) on ophthalmic care. *Acta Ophthalmol*. 2022 Nov;100(7):e1522–e1532.
150. Imamura Y, Fujiwara T, Margolis R, Spaide RF. Enhanced depth imaging optical coherence tomography of the choroid in central serous chorioretinopathy. *Retina*. 2009;29(10):1469–1473.
151. Prünte C, Flammer J. Choroidal capillary and venous congestion in central serous chorioretinopathy. *Am J Ophthalmol*. 1996 Jan;121(1):26–34.
152. Soares RR, Samuelson A, Chiang A. Association of chronic central serous chorioretinopathy with subclinical Cushing's syndrome. *Am J Ophthalmol Case Rep*. 2022 June;26, 101455.
153. Chhablani J, Kozak I, Pichi F, et al. OUTCOMES OF TREATMENT OF CHOROIDAL NEOVASCULARIZATION ASSOCIATED WITH CENTRAL SEROUS CHORIORETINOPATHY WITH INTRAVITREAL ANTIANGIOGENIC AGENTS. *RETINA*. 2015 Dec;35(12):2489.
154. Chan WM, Lai TYY, Liu DTL, Lam DSC. Intravitreal bevacizumab (Avastin) for choroidal neovascularization secondary to central serous chorioretinopathy, secondary to punctate inner choroidopathy, or of idiopathic origin. *Am J Ophthalmol*. 2007 June 1;143(6):977–983.e1.
155. Hanumunthadu D, Tan ACS, Singh SR, Sahu NK, Chhablani J. Management of chronic central serous chorioretinopathy. *Indian J Ophthalmol*. 2018 Dec;66(12): 1704–1714.