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Central serous chorioretinopathy management: current evidence and future directions in pharmacologic and phototherapeutic interventions – a literature review



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HIGHLIGHTS

Central serous chorioretinopathy is challenging to manage. This review explores current therapies, focusing on photodynamic therapy and subthreshold micropulse laser as the most effective and promising options.

ABSTRACT

Central serous chorioretinopathy is a retinal disorder marked by subretinal fluid leakage causing visual impairment, image distortion, and color perception changes. Despite its prevalence, managing chronic central serous chorioretinopathy is challenging due to its complex pathogenesis and recurrent nature. Treatment options include mineralocorticoid receptor antagonists, carbonic anhydrase inhibitors, photodynamic therapy, and subthreshold micropulse laser. This review evaluates current therapies in terms of safety and efficacy and explores emerging strategies for improved management.

Key words: central serous chorioretinopathy, photodynamic therapy, subthreshold micropulse laser, mineralocorticoid antagonist

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INTRODUCTION

Central serous chorioretinopathy (CSC) is a chorioretinal disease characterized by idiopathic retinal detachment resulting from choroidal leakage and dysfunction of the retinal pigment epithelium (RPE) [1]. It was first described in 1866 by the German ophthalmologist Albrecht von Graefe [2]. CSC is the fourth most common cause of retinopathy associated with pathological fluid leakage, following age-related macular degeneration (AMD), diabetic macular edema, and retinal vein occlusion (RVO) [1].

The peak incidence occurs in the fifth decade of life, and men are affected approximately 7 times more frequently than women. According to population-based studies conducted in Olmstead County, Minnesota, the annual incidence of CSC is 1.7 cases per 100,000 women and 9.9 cases per 100,000 men [3]. Although CSC most commonly affects one eye, it can be bilateral in up to 40% of cases [4, 5].

In the typical form of CSC, there is a serous detachment of the neurosensory retina involving the macula, without the presence of subretinal hemorrhage or lipid exudates [6]. The pathogenesis of CSC is currently believed to involve both the RPE and the choroid. Increased choroidal vascular permeability leads to elevated hydrostatic pressure, which compromises the integrity of the RPE barrier and promotes pathological accumulation of subretinal fluid (SRF) between the retina and the RPE [1, 7]. The elevated hydrostatic pressure may cause RPE decompensation through pigment epithelium detachment (PED), ultimately resulting in neurosensory retinal detachment and subsequent vision loss [8].

Despite the lack of a universally accepted terminology and classification system [9], the literature most commonly distinguishes 2 forms of CSC: acute and chronic [1, 6].

Acute CSC typically follows a benign course and resolves spontaneously in most cases [6], although sudden and irreversible vision loss may also occur [10].

Chronic CSC, on the other hand, may persist for over 4 months and is characterized by frequent recurrence episodes.

In acute CSC, there is usually a single or at most several leakage points and a dome-shaped isolated PED, where SRF often resolves spontaneously without clinical consequences. In contrast, chronic CSC is associated with multiple leakage foci, limited RPE elevation, and more frequently persistent atrophic changes that may lead to irreversible vision loss or, in extreme cases, complete blindness. Prognosis is favorable in 90–95% of cases, with spontaneous resolution of SRF [6]. However, although spontaneous regression of SRF without structural retinal damage is possible in some patients, many – particularly those with chronic CSC – experience clinically significant sequelae due to persistent SRF activity. This includes photoreceptor damage, visual acuity decline, RPE atrophy, and in 2–9% of cases, subretinal neovascularization [11, 12]. While SRF is the primary cause of RPE dysfunction in most cases, in some patients, multifocal choroidal vascular dysfunction can impair RPE independently of SRF presence. Patients with CSC may experience reduced visual acuity, image distortion (metamorphopsia), decreased contrast sensitivity, perceived reduction in object size (micropsia), and color vision abnormalities (dyschromatopsia) [12, 13]. The resulting visual impairment is associated with a reduced quality of life [11, 14]. Over the years, numerous factors contributing to the pathogenesis of CSC have been identified. Among external risk factors, systemic corticosteroid use has proven to be the most significant, with an odds ratio of 37.1 [15]. A population-based study reported an annual incidence of CSC of 54.5 per 100,000 men and 34.2 per 100,000 women using oral corticosteroids [16]. It is now widely accepted that corticosteroid use promotes CSC development regardless of formulation or route of administration (excluding topical eye drops) [17]. Genetic predispositions [18, 19], pregnancy [15], and elevated androgen levels [20] have also been indicated, with the latter being consistent with the typical patient profile of a working-age male [13]. Psychological and behavioral factors – such as type A personality, heightened stress susceptibility, emotional instability, and perfectionism – although difficult to quantify, may increase the likelihood of CSC development [21]. Patients with such personality traits generally have elevated cortisol levels and heightened sensitivity to catecholamines [22, 23], which identifies these substances as potential therapeutic targets in CSC treatment. Accurate diagnosis of CSC requires the use of advanced imaging techniques to assess retinal morphology. Commonly employed methods include fluorescein angiography, indocyanine green angiography, optical coherence tomography and fundus autofluorescence [6, 12, 24]. Fluorescein angiography enables localization of SRF leakage sites, optical coherence tomography allows for quantitative SRF assessment through central retinal thickness measurements, and fundus autofluorescence helps estimate episode duration and guides further therapeutic strategy. Indocyanine green angiography and fluorescein angiography also aid in visualizing choroidal neovascularization features. Clinical data derived from these modalities are essential for confirming a CSC diagnosis [25]. Dye-based imaging methods – considered the gold standard – are invasive and not without systemic impact. This has prompted a search for alternative techniques, such as optical coherence tomography angiography – a novel, contactless, and non-invasive method. Despite limited large-scale studies and lack of standardized interpretation criteria, optical coherence tomography angiography allows precise visualization of choroidal pathology regardless of lesion depth and presents a promising prognostic tool for future implementation of a fused diagnostic model that is both precise and safe [26]. Choosing the optimal treatment for CSC is challenging due to the absence of clear guidelines. The primary goal of therapy should be

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the restoration of the functional and anatomical relationship between photoreceptors and the RPE, improvement in choroidal perfusion, and complete resolution of neurosensory detachment [6]. The ideal method should combine safety and efficacy while aiming to prevent recurrences.
The purpose of this article is to review selected therapeutic approaches for CSC. Achieving this objective requires a thorough analysis of both domestic and international literature on the topic.

PHOTOTHERAPEUTIC METHODS

Conventional laser therapy

Conventional laser photocoagulation has been one of the pioneering invasive treatment modalities for CSC, utilized since the late 20th century. Initial studies employing argon laser photocoagulation demonstrated long-lasting therapeutic effects; however, even at that time, concerns were raised regarding potential complications, particularly the development of choroidal neovascularization at the site of serous fluid leakage subjected to laser treatment [27]. More recent comparative studies, such as the 2020 analysis contrasting subthreshold micropulse laser (SML) with threshold conventional laser (TCL), reported comparable efficacy between the two approaches while indicating a higher risk of scotoma formation with traditional laser therapy. Additionally, SML was shown to have a more favorable safety profile with respect to RPE integrity [28, 29]. Despite its clinical efficacy, the use of conventional laser photocoagulation is associated with increased risks of adverse events due to its destructive effect on the RPE, potentially resulting in central scotoma or visual field defects [30–32]. To improve precision, variations using argon or krypton lasers have been developed depending on the anatomical location of the pathological changes on the retina [33]. While TCL has demonstrated therapeutic benefits in restoring best-corrected visual acuity (BCVA) and reducing recurrence [28, 34], it is currently being phased out in favor of SML and half-dose photodynamic therapy, which provide comparable clinical outcomes while minimizing collateral damage through selective targeting of the RPE and reduced laser energy exposure [29, 30, 34].

Photodynamic therapy

Photodynamic therapy (PDT), initially developed for oncologic applications [35], is now considered one of the primary non-pharmacological treatments for CSC. PDT employs laser light in combination with verteporfin, which upon activation releases reactive oxygen species that damage the vascular endothelium. These effects result in enhanced SRF absorption and anatomical remodeling of the choroid through decreased vascular permeability [36]. A critical factor in PDT efficacy for CSC is the appropriate dosing of verteporfin. A half-dose regimen (3 mg/m^2) has been shown to be as effective as the full dose (6 mg/m^2) [37], with the dose reduction correlating with a lower incidence of adverse effects [1, 38]. Even at a 30% dose, beneficial clinical outcomes have been observed, including improvements in BCVA, reduction in SRF, and choroidal remodeling [39]. Nevertheless, the half-dose protocol appears superior in limiting leakage, promoting fluid reabsorption, and improving visual acuity outcomes [40]. Half-dose PDT plays a significant role in CSC management due to its effectiveness in SRF reduction and its lower recurrence rate of fluid leakage [6, 41, 42]. It is effective in improving retinal anatomy in patients with chronic CSC, both when administered early (3–6 months of disease duration) and later (>6 months), with SRF resolution and BCVA improvement observed in both groups, albeit from a lower baseline in the latter [43]. In the large randomized PLACE trial [44], PDT demonstrated significantly greater SRF reduction compared to SML therapy. PDT also successfully halted leakage via choroidal vascular remodeling in cases unresponsive to observation or other pharmacological and laser treatments [45]. However, potential complications of PDT must be taken under consideration. The most commonly reported adverse effect is transient visual deterioration, which typically resolves within one week post-treatment [46]. Rare complications described in the literature include juxtafoveal choroidal neovascularization [45], whitening of the treated area, RPE proliferation, subretinal edema [47], and choroidal hypoperfusion [29]. Exceptionally rare but serious adverse effects include RPE atrophy [48]. Despite these risks, PDT remains a safe and effective treatment option for CSC [38, 49]. Based on current literature, half-dose verteporfin PDT should be regarded as the treatment of choice for chronic CSC [1, 6, 38, 50]. Unfortunately, a global shortage of verteporfin [51] has limited access to this therapy. Short-term follow-up of CSC patients awaiting verteporfin treatment did not show significant BCVA loss [52]. A potential alternative may lie in no-dose PDT, where preliminary results have shown improvements in visual acuity and choroidal thickness reduction; however, this approach requires further investigation [53].

Subthreshold micropulse laser

The SML operates by delivering short laser pulses in interval sequences that minimize damage to surrounding tissues while precisely targeting areas requiring treatment. In this way, SML selectively affects the RPE without damaging photoreceptors [54–56]. The ability to fine-tune laser parameters – such as duty cycle, power, spot size, and pulse duration – allows for optimal therapeutic outcomes while reducing the risk of complications [57]. Furthermore, depending on the location of the CSC lesion, therapy may be adjusted based on the laser's wavelength. For example, the

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577 nm yellow-wavelength laser remains outside the absorption spectrum of xanthophyll, which is most concentrated in the macula, yielding the most beneficial results in this region [58, 59]. Other wavelengths used in micropulse laser therapy include 810 nm (infrared), 577 nm (yellow), 532 nm (green), and 527 nm (green) [58, 60–62]. Micropulse laser can serve as an effective alternative to traditional treatment methods, offering patients precision, low risk of complications, and therapeutic efficacy [1]. Li et al. conducted a meta-analysis involving eleven studies with a total of 834 eyes: 428 treated with SML and 406 receiving conventional therapy. Their findings indicate that the clinical efficacy of SML was comparable to that of PDT. Although SML did not surpass PDT in therapeutic effect, it proved to be a safe and effective treatment modality with no significant adverse effects. Compared to conventional laser treatments, SML has a favorable safety profile, with no damage to the choroid or retina, and no scarring or fibrosis [63]. Preferences and recommendations for CSC treatment vary among researchers and clinicians, largely due to the lack of standardized guidelines regarding optimal SML parameters [30, 64]. Recent efforts have focused on adjusting pulse energy and the number of micropulses based on fundus imaging. This titration-based approach has shown promising results in both functional and anatomical outcomes during CSC treatment [65].

PHARMACOLOGICAL METHODS

Mineralocorticoid receptor antagonists

Mineralocorticoid receptor antagonists (MRAs), commonly used in the treatment of arterial hypertension [66] and heart failure [67], have also been applied in CSC therapy. Clinical studies in rats revealed that aldosterone contributes to choroidal thickening. Inhibition of the mineralocorticoid pathway in these models reduced choroidal thickening, suggesting a role of mineralocorticoids in the pathogenesis of CSC [68]. Both spironolactone and eplerenone have been shown to effectively facilitate SRF reabsorption, with spironolactone demonstrating greater efficacy in improving BCVA [69]. However, eplerenone is associated with fewer anti-androgenic side effects – such as gynecomastia and impotence – making it the preferred selective MRAs over the non-selective spironolactone [70]. Typical MRA-related side effects include hyperkalemia and acute kidney injury, which can be mitigated through proper treatment monitoring [71]. This understanding has led to the use of MRAs in CSC therapy, with eplerenone being favored for its tolerability and ability to induce anatomical changes [72, 73]. In short-term evaluations, spironolactone showed comparable efficacy to half-dose verteporfin PDT [74]. A key advantage of MRAs is their ease of administration and relatively low cost [75]. Despite some studies showing therapeutic benefits of MRAs in CSC [69, 72–74], the randomized, double-blind, placebo-controlled VICI trial [76] did not demonstrate significant advantages of MRAs over placebo in terms of SRF absorption or choroidal thickness reduction. No improvement in BCVA was observed either, leading to a recommendation against the use of eplerenone for CSC treatment. Earlier studies showing MRAs efficacy may have been influenced by insufficient follow-up durations, as CSC frequently resolves spontaneously in its early stages. The randomized trial found no significant difference in SRF absorption between the MRAs and placebo groups [77].

Carbonic anhydrase inhibitors

Carbonic anhydrase inhibitors (CAIs) are commonly used to treat glaucoma, altitude sickness, intracranial hypertension, and congestive heart failure [78]. Their application in CSC has been explored due to the observation that CAI-induced inhibition of carbonic anhydrase in the RPE can promote SRF resorption and retinal adhesion [79]. One study showed that CAIs use led to symptom reduction and subjective visual improvement, though it did not affect final BCVA or recurrence rate [80]. Subsequent studies found a faster resolution of SRF, though without significant functional or anatomical benefits compared to untreated groups [81–83]. To date, large-scale studies on CAIs use in CSC are lacking. Despite their efficacy in lowering SRF, current evidence does not support their clinical use due to a lack of meaningful improvement [1].

β-adrenergic blockers

β-blockers are widely used in cardiovascular disease treatment [84, 85] or anxiety disorders [86], and may have applications in CSC therapy. Excess adrenaline, acting through β-adrenergic receptors, may influence RPE function [87], though it remains debated whether stress reduction affects recurrence or progression of CSC [6]. β-blockers act by inhibiting β-adrenergic receptors, thereby reducing catecholamine effects [88]. Their ability to suppress the sympathetic nervous system prompted trials of oral or intravitreal β-blockers for CSC [89, 90]. The efficacy of β-blockers in CSC remains unclear. Case reports have documented improved visual acuity and reduced SRF following intravitreal metoprolol in patients unresponsive to previous therapies [89]. Oral propranolol was associated with improved SRF resorption, shorter time to remission, and fewer recurrences [90]. A comparative study of propranolol, rifampicin, and intravitreal anti-VEGF showed propranolol to be beneficial and cost-effective, though anti-VEGF therapy yielded superior results in SRF resolution, contrast sensitivity, and faster BCVA improvement [91]. However, a randomized controlled trial [92] found no difference in CSC duration between patients treated with metipranolol and placebo. In summary, β-blockers may be useful in CSC due to their

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sympathetic inhibition. However, their effectiveness remains uncertain, with no conclusive evidence favoring their use over other treatment modalities [93].

Rifampicin

Rifampicin is primarily used as a component of anti-tuberculosis therapy. It is also known to be a cytochrome P-450 3A4 inducer, which is involved in the metabolism of endogenous steroids [94]. It is hypothesized that even low doses of rifampicin may inhibit hypoxia-induced neovascularization, a mechanism also implicated in the pathophysiology of CSC [95]. It has been suggested that the dose should not be lower than 500 mg/24 h, as cytochrome activation occurs above this threshold [96, 97]. Preliminary pilot studies have reported a sustained reduction in SRF and central retinal thickness both during and after therapy [98]. However, other findings indicate that this effect may only be observed in cases with focal retinal changes, suggesting rifampicin may be effective only in earlier stages of the disease, before widespread RPE damage has occurred [99]. Unfortunately, this scenario overlaps with the natural history of CSC, which often undergoes spontaneous remission, casting doubt on the true efficacy of rifampicin. Rifampicin therapy is not neutral and poses systemic consequences. When combined with other anti-tuberculosis drugs, it may induce hepatotoxicity and result in cholestatic hepatitis [100–102]. Caution is warranted regarding concurrent medications, as rifampicin can alter their metabolism, enhancing or reducing their effects, or compounding hepatotoxicity [103]. Similarly to MRAs, this cytochrome inducer offers an interesting alternative approach for CSC management by targeting the pathological effects of steroid hormones on the retina. Despite being affordable and widely accessible, the evidence for its efficacy is limited to specific clinical contexts, and rifampicin should not currently be recommended as a standard treatment.

Methotrexate

Methotrexate (MTX), a folic acid analog and antagonist, was originally developed as an antineoplastic agent and is now commonly used in autoimmune disease management [104]. In a retrospective analysis, complete SRF resolution was achieved in 83% of patients treated with oral MTX (at doses of 5–10 mg) [105]. The mean treatment duration was 89 days, with improvements in BCVA, central macular thickness (CMT), and total macular volume noted after 8 weeks. Another study evaluating oral MTX in CSC treatment showed that 62% of patients achieved SRF resolution after taking 7.5 mg/week for 12 weeks [106]. Neither study reported methotrexate-related toxicity. MTX may thus represent an effective option for chronic CSC; however, both cited studies were uncontrolled, highlighting the need for randomized controlled trials to further investigate its efficacy [105, 106].

Anti-VEGF

Vascular endothelial growth factor inhibitors (anti-VEGF) may help reduce choroidal vascular permeability in CSC, thereby decreasing SRF [107]. Anti-VEGF therapy has demonstrated greater efficacy in cases with choroidal neovascularization than in those without [108]. In the absence of choroidal neovascularization, the use of anti-VEGF in CSC is generally not recommended [109]. A large meta-analysis did not find anti-VEGF therapy beneficial in acute CSC, although improvements in BCVA and SRF reduction have led some to consider it a potential alternative in chronic CSC [110]. Another literature review reported no significant improvement in BCVA despite favorable anatomical outcomes, such as macular thinning and SRF reduction [111]. Anti-VEGF agents may be especially useful when PDT is not feasible or in the presence of choroidal neovascularization [108]. While they effectively reduce CMT and resolve SRF, their impact on visual acuity appears limited. Some studies suggest reduced effectiveness in hypertensive patients and those with thickened choroid [112]. Given the high prevalence of hypertension and its shared risk factors with CSC, the generalizability and effectiveness of anti-VEGF therapy remains in question. Further research is needed to define the optimal therapeutic conditions and evaluate clinical outcomes.

Sildenafil

Sildenafil, a phosphodiesterase type 5 inhibitor (PDE5i), is widely used in erectile dysfunction and pulmonary arterial hypertension, and has recently attracted interest for its potential in CSC management [113]. Its mechanism involves increasing intracellular cyclic guanosine monophosphate (cGMP), which promotes vasodilation and enhanced choroidal blood flow [114, 115]. Recent studies suggest sildenafil may reduce SRF and improve visual acuity, particularly in patients resistant to conventional treatments such as PDT [114–116]. These findings, however, serve more as a rationale for future large-scale trials than as proof of efficacy. Earlier reports had hypothesized that PDE5i could induce CSC [117–119], though current evidence does not support this correlation [120]. Long-term therapeutic effects, safety across various clinical contexts, potential drug interactions, and optimal dosing still require evaluation [114–116]. Nonetheless, this treatment may be beneficial in patients with CSC who also have comorbidities for which PDE5 inhibitors are indicated, such as certain forms of pulmonary hypertension.

Platelet-rich plasma and electromagnetic stimulation

Growth factors present in plasma – particularly those from platelets – are increasingly used to accelerate wound healing [121]. When combined with electromagnetic stimulation (2000 mG, 42 Hz), which modulates growth factor activity [122, 123], they offer a novel therapeutic avenue for re-

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generating damaged RPE. Factors such as platelet-derived growth factor (PDGF), epidermal growth factor (EGF), and transforming growth factor β (TGF-β) [124], administered via sub-Tenon injection and paired with stimulation, may support RPE healing by enhancing pump function and maintaining a robust blood–retina barrier [123]. This method appears effective as an adjunct therapy in cases refractory to conventional treatments, including PDT and SML, which are otherwise considered effective. Due to limited evidence – mostly from a prospective study conducted in 2018–2019 in treatment-resistant CSC – this combination therapy shows promise as a safe and relatively accessible option, especially in refractory cases [123]. However, the lack of large-scale studies, meta-analyses, and the low incidence of treatment-resistant cases, currently limit its broader implementation.

CONCLUSION

CSC remains a therapeutic challenge due to its complex pathogenesis and the absence of standardized treatment guidelines. A review of available treatments shows that while traditional pharmacotherapies such as MRAs, CAIs, β-blockers, and anti-VEGF agents are widely accessible and offer partial effectiveness, they are often insufficient due to low rates of sustained success and limited evidence of long-term efficacy. Moreover, many of these treatment options offer only modest improvements in visual acuity or SRF reduction and are associated with potential adverse effects and drug interactions. Among modern therapeutic options, PDT with verteporfin and SML are particularly notable. Reduced-dose PDT has demonstrated high efficacy in reducing SRF, improving retinal anatomy, and preventing recurrences, and is currently the preferred treatment for chronic CSC, although verteporfin shortages limit its availability. SML, by contrast, offers a precise, tissue-sparing, and effective alternative, improving retinal function and structure with minimal side effects [1, 55]. Given the restricted access to verteporfin [51], SML is emerging as the default treatment in an increasing number of ophthalmology practices [59].

In conclusion, while numerous therapeutic options exist, PDT and SML remain the most promising, balancing efficacy and safety. Further randomized, controlled trials are essential to refine treatment protocols and evaluate long-term outcomes. Novel treatments – such as platelet-rich plasma therapy [122] or verteporfin-free PDT [53] – also warrant exploration, especially as existing therapies face availability challenges. Future efforts should focus on optimizing current strategies and developing complementary or replacement therapies.

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