ISSN: 2766-9556

Journal of Clinical Case Studies and Reviews

Review Article Open Access

Bilateral Central Serous Chorioretinopathy Following High-Dose Systemic Corticosteroid Therapy: A Review of Published Cases

Georgios Antoniou[1*], Francesca Moretti[2] and Niamh Gallagher[3]

[1]Department of Ophthalmology, Hellenic Institute of Clinical Medicine, Patras, Greece

[2]Department of Ophthalmology, Adriatic University Hospital, Bari, Italy

[3]Department of Ophthalmology, Lee Valley Medical Centre, Cork, Ireland

***Correspondence to:** Georgios Antoniou, Department of Ophthalmology, Hellenic Institute of Clinical Medicine, Patras, Greece

Received: 21 April 2022; **Accepted:** 12 July 2022; **Published:** 30 October 2022

Copyright: © 2022 GA, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Central serous chorioretinopathy is usually a unilateral, self-limiting disease of middle-aged men. The corticosteroid-associated form behaves differently: it is more often bilateral, more often severe, and it appears in patients who do not fit the classical demographic. Since the drug is frequently being given for a condition that also requires treatment, the clinical problem is rarely a simple matter of stopping it.

Objectives:*To review the published evidence linking systemic corticosteroid exposure to central serous chorioretinopathy, the features that t distinguish the corticosteroid-associated form, and the management options where the drug cannot readily be withdrawn.*

Material and Methods:*A narrative review. MEDLINE, Embase and the Cochrane Library were searched from January 1990 to May 2026 for r epidemiological studies, case-control studies, randomised treatment trials, imaging series and reviews addressing central serous chorioretinopathy and corticosteroid exposure. Values were extracted from the primary reports and tabulated without pooling.*

Results:*Population incidence is 9.9 per 100,000 in men and 1.7 per 100,000 in women. Systemic corticosteroid use is the strongest reported d external risk factor, with a multivariate odds ratio of 37.1 in the largest case-control study, and remains significant across independent cohorts. Corticosteroid-associated disease is disproportionately bilateral, multifocal and severe. In the only adequately powered randomised comparison of treatments for chronic disease, half-dose photodynamic therapy resolved subretinal fluid in 67.2% against 28.8% for subthreshold micropulse laser, while a randomised trial of eplerenone found no benefit over placebo.*

Conclusion:*Recognising the association matters mainly because withdrawal or dose reduction of the corticosteroid is the intervention with the e clearest rationale, and because the diagnosis is easily missed in patients who do not resemble the classical phenotype. Where the drug must continue, half-dose photodynamic therapy has the best randomised evidence.*

Abbreviations:*CSCR – Central Serous Chorioretinopathy, SRF – Subretinal Fluid, RPE – Retinal Pigment Epithelium, OCT – Optical l Coherence Tomography, PDT – Photodynamic Therapy, BCVA – Best-Corrected Visual Acuity, OR – Odds Ratio.*

Keywords: *Central Serous Chorioretinopathy; Corticosteroids; Subretinal Fluid; Photodynamic Therapy; Choroid*

Introduction

Central serous chorioretinopathy is a serous detachment of the neurosensory retina at the posterior pole, arising from choroidal hyperpermeability with secondary failure of the retinal pigment epithelium to keep the subretinal space dry. It was described in its modern form as an idiopathic disease of young and middle-aged men, and the classical episode is unilateral, self-limiting and visually benign [1,2].

That description is accurate for the majority and misleading for an important minority. The only population-based incidence study reports 9.9 cases per 100,000 per year in men and 1.7 in women, a sixfold difference that has anchored clinical suspicion around the

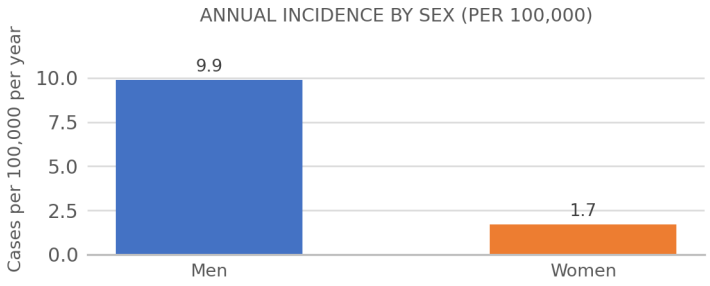

Figure 1: Annual incidence of central serous chorioretinopathy per 100,000 population by sex, from the only population-based study [3].

middle-aged male phenotype ever since (Figure 1) [3]. When the disease appears outside that group —in a woman, in an older patient, bilaterally —it is more likely to be attributed to something else, and the something else is frequently age-related macular degeneration.

Terminology has been a persistent obstacle to reading this literature. The same patient has been described as having acute, chronic, recurrent, diffuse retinal pigment epitheliopathy or persistent disease depending on the decade and the author, and treatment series have therefore enrolled populations that are not straightforwardly comparable. A consensus classification built on multimodal imaging rather than on duration has narrowed that problem, separating simple from complex disease by the extent of pigment epithelial involvement, and it is worth checking which definition a given series used before transferring its results to a patient [31].

The strongest identified external risk factor is corticosteroid exposure. The association was suspected from the 1960s, described in detail in the 1980s and 1990s, and confirmed in a case-control study of 312 patients and 312 controls that found a multivariate odds ratio of 37.1 for systemic steroid use [4,5,6]. It is also observed in endogenous hypercortisolism, which argues that the effect is pharmacological rather than an artefact of who receives steroids [7].

Incidence data outside Olmsted County are sparse but consistent in direction. A population-based Taiwanese analysis found a comparable male preponderance, and reviews synthesising the epidemiological literature place the peak incidence between the fourth and sixth decades with a male-to-female ratio ranging from roughly three to seven to one [32]. The consistency of that skew across populations is what makes the steroid-associated cases conspicuous when they appear outside it.

This matters clinically for a specific reason. In idiopathic disease the treatment decision is whether to observe or intervene. In corticosteroid-associated disease there is a third option that acts on the cause, but exercising it means altering treatment for whatever condition the steroid was prescribed for. This review examines the strength of the association, how the corticosteroid-associated form differs in presentation, and what the randomised evidence supports when the drug cannot be stopped.

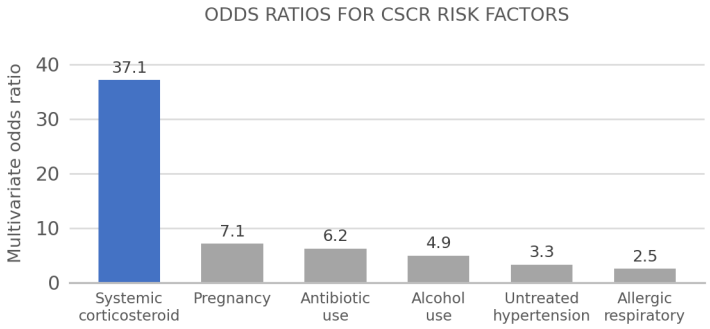

Figure 2: Multivariate odds ratios for risk factors for central serous chorioretinopathy in a case-control study of 312 cases and 312 controls [4].

Risk factor	Odds ratio	95% confidence interval
Systemic corticosteroid use	37.1	6.2 to 221.8
Pregnancy	7.1	1.0 to 50.7
Antibiotic use	6.2	1.0 to 37.9
Alcohol use	4.9	1.5 to 16.3
Untreated hypertension	3.3	1.3 to 8.5
Allergic respiratory disease	2.5	1.2 to 5.1

Table 1: Reported risk factors for central serous chorioretinopathy from the largest case-control study, by multivariate odds ratio.

Material and Methods

Search strategy:

MEDLINE, Embase and the Cochrane Library were searched from January 1990 to May 2026. Terms combined controlled vocabulary and free text for central serous chorioretinopathy, chorioretinopathy, corticosteroid, glucocorticoid, mineralocorticoid receptor, photodynamic therapy and subretinal fluid. Reference lists of reviews and treatment guidelines were hand-searched.

Eligibility and synthesis:

Population-based incidence studies, case-control and cohort studies reporting risk estimates, randomised treatment trials, imaging and classification studies, and narrative or systematic reviews were eligible. Individual case reports were used only where they document a distinct presentation or exposure route. Values were extracted by one reviewer and checked by a second. The included studies differ in case definition, imaging modality and duration of follow-up, so results are presented descriptively and no pooling was attempted. No individual patient data were accessed and ethics approval was not required.

Results

Strength and consistency of the association

The risk estimates are summarised in Figure 2 and Table 1. In the largest case-control study, systemic corticosteroid use carried a multivariate odds ratio of 37.1, far above pregnancy at 7.1, antibiotic use at 6.2, alcohol at 4.9, untreated hypertension at 3.3

and allergic respiratory disease at 2.5 [4]. Reviews of the same dataset quote a lower univariate figure of 10.3, and the discrepancy between the two is a reminder that the confidence intervals around these estimates are wide [1,8].

Independent work is directionally consistent. A separate case-control cohort found corticosteroid use associated with an odds ratio of about 7, alongside alcohol and smoking [9]. Topical ophthalmic corticosteroids, which might have been expected to be innocuous, carried a substantially raised risk during current use in a large database analysis [10]. Endogenous excess produces the same picture: patients with Cushing syndrome develop the disease and improve when cortisol is normalised [7].

Mechanism supports association. Choroidal thickening and hyperpermeability are the consistent structural findings on enhanced-depth imaging and indocyanine green angiography [11,12,13]. The mineralocorticoid receptor is expressed in choroidal vasculature and its inappropriate activation reproduces the phenotype experimentally, which provided the rationale for antagonist therapy and explains why a drug given for its glucocorticoid effect can act through a mineralocorticoid pathway [14,15].

The receptor argument is worth stating precisely, because it explains an otherwise odd clinical fact. Glucocorticoids given at high dose occupy mineralocorticoid receptors as well as glucocorticoid ones, and in the choroid it appears to be the mineralocorticoid pathway that drives vasodilatation and leakage. That is why the effect is not confined to drugs chosen for mineralocorticoid activity, why potency and dose matter more than the particular preparation, and why the retinopathy can follow routes of administration that were assumed to produce negligible systemic exposure [14,15].

How the corticosteroid-associated form differs

Steroid-associated disease is not simply idiopathic disease in a patient who happens to be taking prednisolone. It is disproportionately bilateral, more often multifocal, and more often accompanied by extensive pigment epithelial change than the classical unilateral episode [5,16]. Severe phenotypes, including bullous variants with large exudative detachments, are described predominantly in the context of exogenous or endogenous steroid excess, and carry a materially worse visual prognosis [17,18].

The demographic distortion is equally important. Because the association operates through drug exposure rather than through the constitutional factors that produce the male preponderance, steroid-associated cases appear in women and in older patients, precisely the groups in whom the diagnosis is least expected [3,19]. In a patient over fifty with bilateral subretinal fluid, the differential moves towards neovascular age-related macular degeneration, and the distinction rests on imaging rather than on demography.

Route of administration does not confer safety. Cases follow oral, intravenous, intra-articular, inhaled, intranasal and topical ocular exposure, and patients frequently do not volunteer non-oral steroids when asked about medication [10,20]. The practical consequence is that the drug history has to be taken deliberately and by route.

Imaging carries most of the diagnostic weight in the atypical presentations. Optical coherence tomography demonstrates the serous detachment and any pigment epithelial detachment; enhanced-depth imaging shows the thickened choroid that distinguishes the disease from age-related macular degeneration, in which the choroid is typically thinned; and indocyanine green angiography shows the patchy choroidal hyperpermeability that underlies it [11,12,13]. In a bilateral case in an older patient, that combination is what separates a treatable drug effect from a diagnosis with an entirely different management pathway, and it is the reason the distinction should not be attempted on fundoscopy alone.

What happens when the steroid is stopped

Withdrawal is the intervention with the clearest rationale, and observational series report resolution of subretinal fluid following discontinuation or dose reduction, sometimes over weeks to months [16,21]. The evidence is uncontrolled and cannot easily be otherwise, since the natural history of acute disease is spontaneous resolution in most patients within three to four months regardless of intervention [22].

Withdrawal is also frequently not available. The corticosteroid may be treating a condition in which interruption carries greater risk than the retinopathy —transplant rejection, vasculitis, an acute inflammatory relapse —and the decision then belongs jointly to the ophthalmologist and the prescribing physician rather than to either alone. Where the dose can be reduced rather than stopped, that is worth attempting before committing to ocular treatment.

Timing complicates the assessment further. Because acute disease resolves spontaneously in most patients within a few months, a resolution observed after steroid withdrawal may be coincidental rather than causal, and the literature contains no design capable of separating the two. What can be said is that persistence of subretinal fluid beyond four months while the drug continues, and resolution after it stops in a patient who had failed to resolve before, are both suggestive. Neither is proof, and neither should delay a decision that needs making on other grounds.

Treatment when the drug must continue

The randomised evidence is summarised in Figure 3 and Table 2. The PLACE trial randomised patients with chronic disease to half-dose photodynamic therapy or high-density subthreshold micropulse laser: complete resolution of subretinal fluid was achieved in 67.2% against 28.8%, with a greater gain in visual

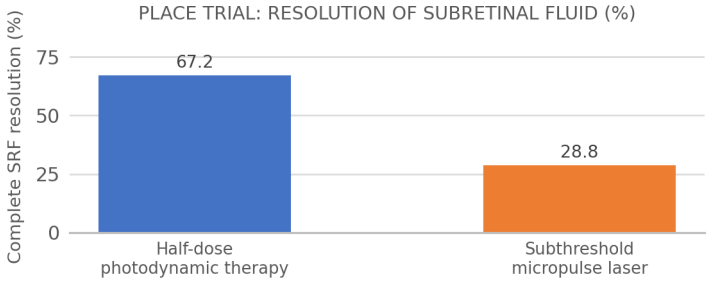

Figure 3: Complete resolution of subretinal fluid in the PLACE trial, by treatment arm [23].

Trial	Comparison	Primary outcome	Result
PLACE [23]	Half-dose PDT vs micropulse laser	Resolution of subretinal fluid	67.2% vs 28.8%
PLACE report 3 [24]	Focal vs diffuse phenotype	Resolution by phenotype	PDT superior in both
VICI [28]	Eplerenone vs placebo	Visual acuity at 12 months	No benefit over placebo
Bae 2011 [27]	Low-fluence PDT vs ranibizumab	Anatomical and visual outcome	PDT favoured

Table 2: Randomised evidence for treatment of chronic central serous chorioretinopathy.

acuity of 4.60 against 1.39 ETDRS letters and better retinal sensitivity on microperimetry (Figure 4) [23]. Subsequent reports from the same trial found the advantage held across focal and diffuse phenotypes [24].

Photodynamic therapy at reduced dose or fluence is supported by earlier randomised work in acute disease and appears to act by remodelling the choroidal vasculature rather than by closing a leak, which is consistent with the pathophysiology [25,26]. Anti-vascular endothelial growth factor injection, tested against low-fluence photodynamic therapy in chronic disease without neovascularisation, did not perform comparably and has no clear role unless secondary neovascular membrane is present [27].

Practical constraints shape what is actually offered. Verteporfin supply has been intermittent in several health systems, photodynamic therapy is not available in every unit treating these patients, and the half-dose protocol is a modification of a licensed regimen rather than a separately approved one. Where it cannot be delivered promptly, the alternatives are observation with the steroid dose minimised, or referral onward; substituting a treatment with weaker evidence because it is locally available is the decision most likely to be regretted, given how much of the eventual visual outcome is determined by how long fluid persists [29,30].

The mineralocorticoid hypothesis produced an attractive oral option that did not survive testing. Pilot data with spironolactone and eplerenone were encouraging, but the VICI trial randomised patients with chronic disease to eplerenone or placebo and found no benefit in visual acuity at twelve months [14,28]. That result is a

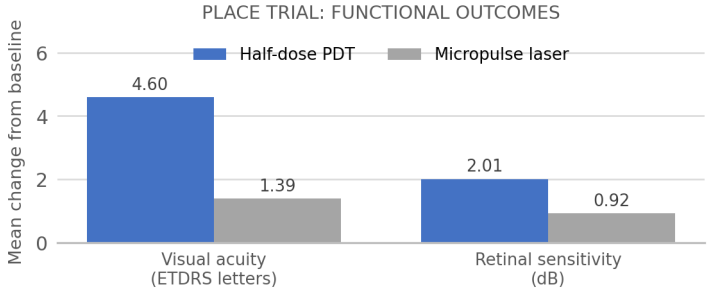

Figure 4: Functional outcomes in the PLACE trial: change in best-corrected visual acuity in ETDRS letters and in retinal sensitivity on microperimetry [23].

Step	Detail	Rationale
Drug history by route	Oral, intravenous, intra-articular, inhaled, nasal, topical ocular	Non-oral routes are under-reported [10,20]
Consider despite demographics	Bilateral disease, women, age over 50	Association is drug-driven, not constitutional [3,19]
Discuss withdrawal with prescriber	Stop or reduce where the primary condition allows	Only intervention acting on the cause [16,21]
Treat if exposure must continue	Half-dose photodynamic therapy	Best randomised evidence [23]
Avoid treating on mechanism alone	Eplerenone not supported	Negative randomised trial [28]

Table 3: Practical steps when corticosteroid-associated disease is suspected.

useful corrective: a coherent mechanism and a positive pilot study are not evidence of efficacy, and the drug should not be offered on mechanistic grounds alone.

Chronicity itself is the main determinant of outcome. Long-standing subretinal fluid produces photoreceptor loss that no subsequent treatment reverses, and long-term follow-up of chronic disease shows persistent deficits in vision-related quality of life even after anatomical success [29,30]. This is the strongest argument for identifying the corticosteroid link early, since it is the one factor that can be modified before structural damage accrues (Table 3).

Recurrence deserves separate mention because it shapes how patients are counselled. A substantial minority of patients experience further episodes, and continuing corticosteroid exposure is among the factors associated with recurrence and with progression to the chronic form. Where the drug cannot be stopped, that argues for scheduled review rather than discharge after a first resolution, and for setting the expectation at the outset that monitoring will continue while exposure does [22,29].

Discussion

The association between corticosteroid exposure and central serous chorioretinopathy is about as well established as an association of this kind can be without a randomised trial: a large effect size in case-control data, consistency across independent

cohorts and routes of administration, a coherent receptor-level mechanism, and the same phenotype in endogenous cortisol excess. What remains uncertain is the absolute risk, which the available designs cannot estimate, and which is presumably low given how many patients take corticosteroids without ocular consequence.

For practice, three things follow. The drug history should be taken by route rather than by recall, since inhaled, intra-articular and topical preparations are systematically under-reported. Bilateral or atypical presentations should raise the possibility rather than lower it, because the steroid-associated form is where bilateral disease concentrates. And the decision about withdrawal belongs to a conversation with the prescribing physician, in which the retinopathy is weighed against whatever the steroid is preventing.

Where the drug must continue, half-dose photodynamic therapy is the treatment with the best randomised support, and eplerenone is not an alternative to it. The evidence base has a notable gap here: PLACE and VICI enrolled chronic disease irrespective of corticosteroid exposure, so the treatment effect specifically in steroid-associated disease, where the causative stimulus persists, is inferred rather than measured. A trial stratified by ongoing exposure would be informative and does not exist.

A final point concerns how these patients reach ophthalmology at all. Central serous chorioretinopathy produces a relative scotoma, micropsia and reduced contrast rather than dense visual loss, and patients receiving high-dose steroids for a serious systemic illness frequently attribute those symptoms to the illness or to the drug in general terms. The referral is therefore often late, by which time fluid has been present long enough to matter. Asking directly about visual symptoms in patients on prolonged high-dose therapy costs nothing and is the only realistic way of shortening that interval.

Study limitations

This is a narrative rather than a systematic review: studies were selected against stated criteria but without duplicate screening of the full yield, and no risk-of-bias instrument was applied. The risk estimates derive from case-control designs vulnerable to recall and selection bias, with wide confidence intervals. Much of the evidence on withdrawal is uncontrolled and confounded by spontaneous resolution. The randomised treatment data were not generated in steroid-exposed populations specifically. The review does not cover paediatric disease, pregnancy-associated cases or the surgical management of secondary neovascularisation.

Conclusion

Corticosteroid exposure is the strongest modifiable risk factor for central serous chorioretinopathy, and the disease it produces is more often bilateral, more often severe and more often missed than the idiopathic form, because it appears in patients who do not match the expected phenotype. The drug history should be taken by route, the diagnosis should be considered in bilateral subretinal fluid at any age, and withdrawal or dose reduction should be discussed with the prescriber before ocular treatment is committed to. Where exposure must continue, half-dose photodynamic therapy has the best randomised evidence; eplerenone, despite a persuasive mechanism, does not.

References

1. Nicholson B, Noble J, Forooghian F, Meyerle C. "Central serous chorioretinopathy: update on pathophysiology and treatment". Surv Ophthalmol 58(2013):103-126.

2. Gass JD. "Pathogenesis of disciform detachment of the neuroepithelium". Am J Ophthalmol 63(1967):1-139.

3. Kitzmann AS, Pulido JS, Diehl NN, Hodge DO, Burke JP. "The incidence of central serous chorioretinopathy in Olmsted County, Minnesota, 1980-2002". Ophthalmology 115(2008):169-173.

4. Haimovici R, Koh S, Gagnon DR, Lehrfeld T, Wellik S. "Risk factors for central serous chorioretinopathy: a case-control study". Ophthalmology 111(2004):244-249.

5. Carvalho-Recchia CA, Yannuzzi LA, Negrao S, Spaide RF, Freund KB, et al. "Corticosteroids and central serous chorioretinopathy". Ophthalmology 109(2002):1834-1837.

6. Wakakura M, Song E, Ishikawa S. "Corticosteroid-induced central serous chorioretinopathy". Jpn J Ophthalmol 41(1997):180-185.

7. Bouzas EA, Scott MH, Mastorakos G, Chrousos GP, Kaiser-Kupfer MI. "Central serous chorioretinopathy in endogenous hypercortisolism". Arch Ophthalmol 111(1993):1229-1233.

8. Bouzas EA, Karadimas P, Pournaras CJ. "Central serous chorioretinopathy and glucocorticoids". Surv Ophthalmol 47(2002):431-448.

9. Karimi S, Payandeh N, Mahmoudi Nejad Azar S, Nouri H, Abtahi SH. "Central serous chorioretinopathy risk factors in an Iranian cohort: a case-control study". J Ophthalmic Vis Res 18(2023):396-405.

10. Chen SN, Chen YC, Lian I. "Temporal association between topical ophthalmic corticosteroid and the risk of central serous chorioretinopathy". J Clin Med 9(2020):4023.

11. Imamura Y, Fujiwara T, Margolis R, Spaide RF. "Enhanced depth imaging optical coherence tomography of the choroid in central serous chorioretinopathy". Retina 29(2009):1469-1473.

12. Prunte C, Flammer J. "Choroidal capillary and venous congestion in central serous chorioretinopathy". Am J Ophthalmol 121(1996):26-34.

13. Chan WM, Lam DS, Lai TY, Tam BS, Liu DT, et al. "Choroidal vascular remodelling in central serous chorioretinopathy after indocyanine green guided photodynamic therapy with verteporfin". Br J Ophthalmol 87(2003):1453-1458.

14. Zhao M, Celerier I, Bousquet E, Jeanny JC, Jonet L, et al. "Mineralocorticoid receptor is involved in rat and human ocular chorioretinopathy". J Clin Invest 122(2012):2672-2679.

15. Bousquet E, Beydoun T, Zhao M, Hassan L, Offret O, et al. "Mineralocorticoid receptor antagonism in the treatment of chronic central serous chorioretinopathy: a pilot study". Retina 33(2013):2096-2102.

16. Sharma T, Shah N, Rao M, Gopal L, Shanmugam MP, et al. "Visual outcome after discontinuation of corticosteroids in atypical severe central serous chorioretinopathy". Ophthalmology 111(2004):1708-1714.

17. Mohabati D, van Rijssen TJ, van Dijk EH, Luyten GP, Missotten TO, et al. "Clinical characteristics and long-term visual outcome of severe phenotypes of chronic central serous chorioretinopathy". Clin Ophthalmol 12(2018):1061-1070.

18. Spaide RF, Campeas L, Haas A, Yannuzzi LA, Fisher YL, et al. "Central serous chorioretinopathy in younger and older adults". Ophthalmology 103(1996):2070-2079.

19. Tittl MK, Spaide RF, Wong D, Pilotto E, Yannuzzi LA, et al. "Systemic findings associated with central serous chorioretinopathy". Am J Ophthalmol 128(1999):63-68.

20. Nicholson BP, Atchison E, Idris AA, Bakri SJ. "Central serous chorioretinopathy and glucocorticoids: an update on evidence for association". Surv Ophthalmol 63(2018):1-8.

21. Hardwig PW, Silva AO, Pulido JS. "Forgotten exogenous corticosteroid as a cause of central serous chorioretinopathy". Clin Ophthalmol 2(2008):199-201.

22. Daruich A, Matet A, Dirani A, Bousquet E, Zhao M, et al. "Central serous chorioretinopathy: recent findings and new physiopathology hypothesis". Prog Retin Eye Res 48(2015):82-118.

23. van Dijk EHC, Fauser S, Breukink MB, Blanco-Garavito R, Groenewoud JMM, et al. "Half-dose photodynamic therapy versus high-density subthreshold micropulse laser treatment in patients with chronic central serous chorioretinopathy: the PLACE trial". Ophthalmology 125(2018):1547-1555.

24. van Rijssen TJ, van Dijk EHC, Scholz P, Breukink MB, Blanco-Garavito R, 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 205(2019):1-10.

25. Chan WM, Lai TY, Lai RY, Liu DT, Lam DS. "Half-dose verteporfin photodynamic therapy for acute central serous chorioretinopathy: one-year results of a randomized controlled trial". Ophthalmology 115(2008):1756-1765.

26. Gemenetzi M, De Salvo G, Lotery AJ. "Central serous chorioretinopathy: an update on pathogenesis and treatment". Eye 24(2010):1743-1756.

27. Bae SH, Heo JW, Kim C, Kim TW, Lee JY, et al. "A randomized pilot study of low-fluence photodynamic therapy versus intravitreal ranibizumab for chronic central serous chorioretinopathy". Am J Ophthalmol 152(2011):784-792.

28. Lotery A, Sivaprasad S, O'Connell A, Harris RA, Culliford L, 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 395(2020):294-303.

29. Breukink MB, Dingemans AJ, den Hollander AI, Keunen JE, MacLaren RE, et al. "Chronic central serous chorioretinopathy: long-term follow-up and vision-related quality of life". Clin Ophthalmol 11(2017):39-46.

30. van Rijssen TJ, van Dijk EHC, Yzer S, Ohno-Matsui K, Keunen JEE, et al. "Central serous chorioretinopathy: towards an evidence-based treatment guideline". Prog Retin Eye Res 73(2019):100770.

31. Chhablani J, Cohen FB. "Multimodal imaging-based central serous chorioretinopathy classification". Ophthalmol Retina 4(2020):1043-1046.

32. Liew G, Quin G, Gillies M, Fraser-Bell S. "Central serous chorioretinopathy: a review of epidemiology and pathophysiology". Clin Exp Ophthalmol 41(2013):201-214.

Citation: Antoniou G, Moretti F, Gallagher N. "Bilateral Central Serous Chorioretinopathy Following High-Dose Systemic Corticosteroid Therapy: A Review of Published Cases." J Clin Case Stud (2022): 001-006 doi: 10.5281/zenodo.22206016