

Topical dermal steroid–induced retinopathy



Taylor Hilton, MS,^a Andrew DeCrescenzo, MD,^b Alan Menter, MD,^b and Sai H. Chavala, MD^{a,c}
Fort Worth and Dallas, Texas

Key words: central serous chorioretinopathy; psoriasis; steroid.

INTRODUCTION

Central serous chorioretinopathy (CSCR) is a condition defined by subretinal fluid accumulation in the area of the macula leading to serous detachment. CSCR is a significant vision-threatening retinopathy that can lead to irreversible vision loss. Although presentation can vary, the classic case comprises unilateral vision loss with the patient reporting a dark spot in the center of their visual field.¹ Corticosteroid use has long been considered to be a factor contributing to CSCR in patients, with oral administration having the most common occurrence.² However, there have been previous cases reported of CSCR presenting following topical dermal steroid use.³ We report a case of a patient with two separate episodes of CSCR induced by use of topical steroids.

CASE REPORT

A 48-year-old man with a history of psoriasis was using intermittent topical clobetasol for a recent flair of his psoriasis. He presented with new-onset blurred vision in the left eye after application of clobetasol to the tip of his nose, eyebrows, and periauricular skin. The patient was evaluated by a retina specialist and was found to have CSCR in the left eye (Fig 1), with no significant visual acuity loss. Additional pertinent medications were azelastine hydrochloride nasal spray and fluticasone propionate nasal spray. The patient was advised to discontinue topical and intranasal steroids. At 2-month follow up, the subretinal fluid associated with CSCR was completely resolved (Fig 2). Six

Abbreviation used:

CSCR: central serous chorioretinopathy

months later, the patient restarted only clobetasol without the intranasal preparations. Within 2 weeks, the patient presented to the retina specialist with a relapse in CSCR (Fig 3). The patient once again discontinued topical steroids and the subretinal fluid resolved (Fig 4).

DISCUSSION

CSCR is a relatively rare diagnosis; however, the potential for poor visual prognosis combined with the common frequency of steroid use shows the importance of recognition of this finding. Although many retinal conditions are more commonly seen in older populations, CSCR typically affects young to middle-aged adults. In idiopathic cases, men have a 6-times higher incidence than women; however, corticosteroid-associated cases do not show a strong predilection.⁴ Time of onset can vary widely, and there appears to be no correlation between dose of steroid used and increased incidence of CSCR.⁵ Visual acuity changes can range from mild to moderate, with most cases of CSCR resolving spontaneously. However, 35% to 40% will recur or become chronic resulting in poor visual prognosis.⁶

A pathophysiologic cause is yet to be identified for CSCR. One mechanism currently being investigated is the stimulation of mineralocorticoid receptors in the retinal pigment epithelium

From Texas College of Osteopathic Medicine, North Texas Eye Research Institute, University of North Texas Health Science Center, Fort Worth^a; the Division of Dermatology, Baylor University Medical Center, Dallas^b; and the Department of Surgery, Texas Christian University and University of North Texas Health Science Center School of Medicine, Fort Worth.^c

Funding sources: The Nancy Lee and Perry R. Bass Endowment provided funding support for Dr. Chavala.

Conflicts of interest: None disclosed.

Correspondence to: Sai H. Chavala, MD, Department of Surgery, Texas Christian University and University of North Texas Health

Science Center School of Medicine, Fort Worth, TX 76129. E-mail: sai.chavala@tcu.edu.

JAAD Case Reports 2020;6:868-70.

2352-5126

© 2020 by the American Academy of Dermatology, Inc. Published by Elsevier, Inc. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

<https://doi.org/10.1016/j.jidcr.2020.07.015>

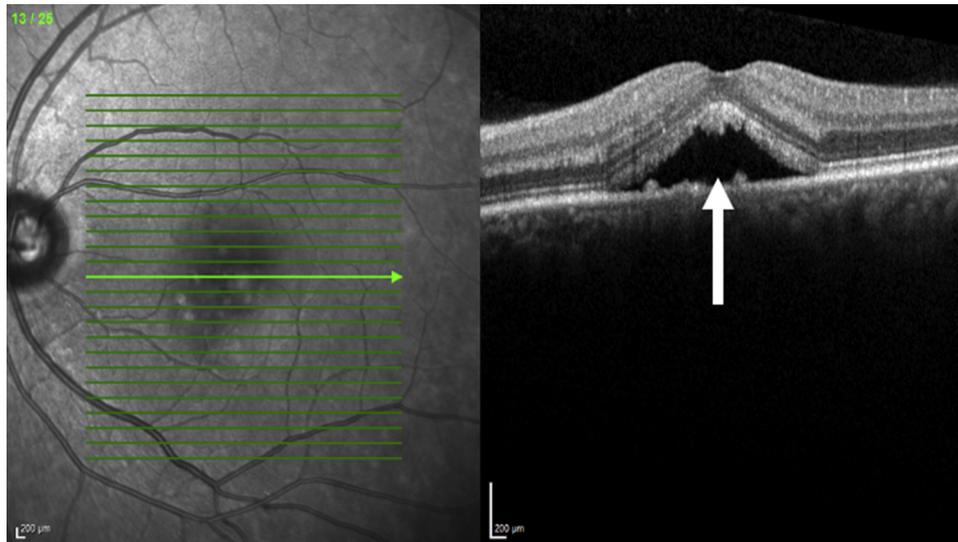


Fig 1. CSCR, episode 1. Optical coherence tomography demonstrates subretinal fluid is seen beneath the fovea indicated by the white arrow.

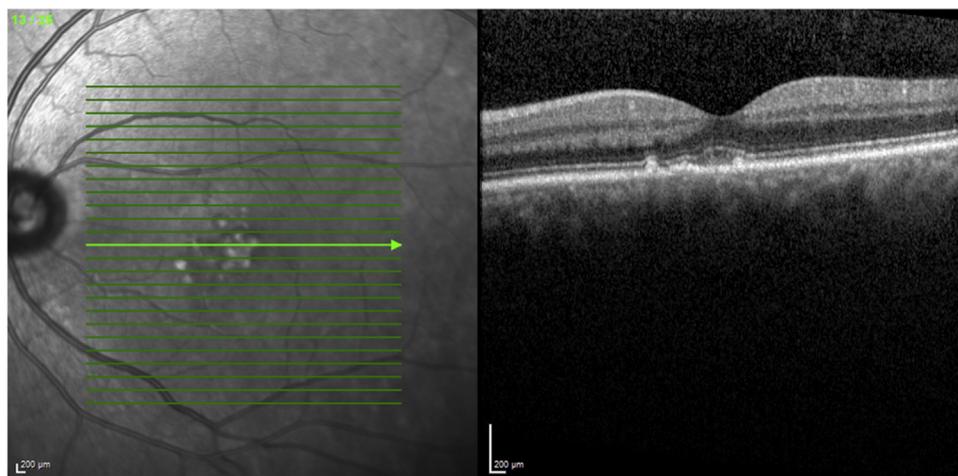


Fig 2. CSCR, resolved. Subretinal fluid resolved with restoration of foveal contour.

and choroid by exogenous and endogenous glucocorticoids resulting in vasodilation and fluid accumulation. It is thought that this sensitivity of mineralocorticoid receptors explains retinal fluid accumulation in addition to the other comorbidities associated with CSCR, which are hypertension, psychological stress, and coronary artery disease.⁷ Because the mechanism of steroid use and complication of CSCR is not fully elucidated, further study is needed to show causality.

Well-studied triggers include antibiotic use, pregnancy, type A personality, psychosocial stress, and corticosteroid use.⁴ Recurrence rates have been difficult to elicit; however, case reviews have

reported recurrence rates between 15% and 50%.⁴ For recurrent cases or cases with persistent subretinal fluid for more than 3 months, thermal laser to the site of retinal injury, photodynamic therapy, and other experimental agents have been proposed with good prognosis. Thus, it is important to refer patients for dilated ophthalmologic examination if patients on oral, topical, or inhaled corticosteroids experience vision changes. If complications of CSCR do arise, alternate topical therapies for psoriasis may be considered, including topical calcineurin inhibitors or lower-potency topical steroids if the patient is not already a candidate for systemic therapy.

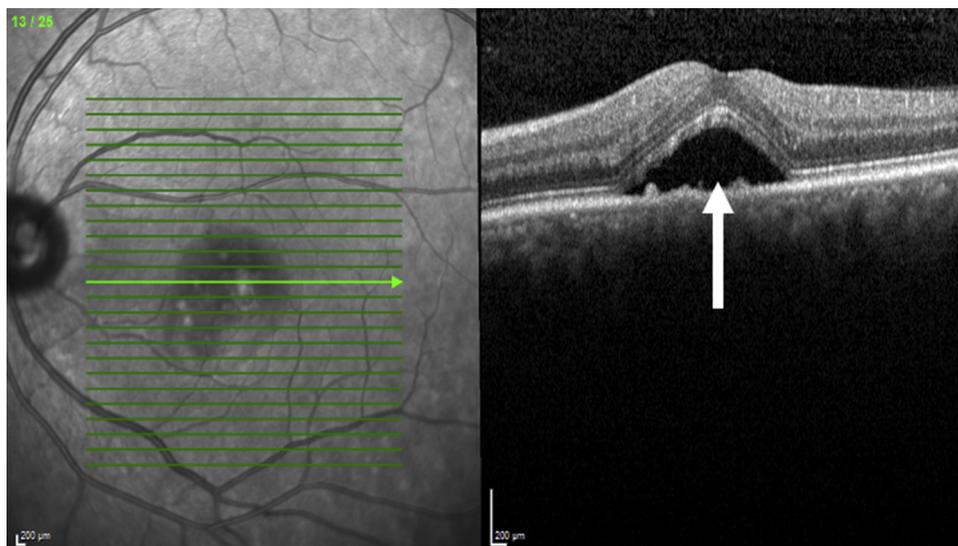


Fig 3. CSCR, episode 2. Subretinal fluid return when clobetasol was restarted indicated by the white arrow.

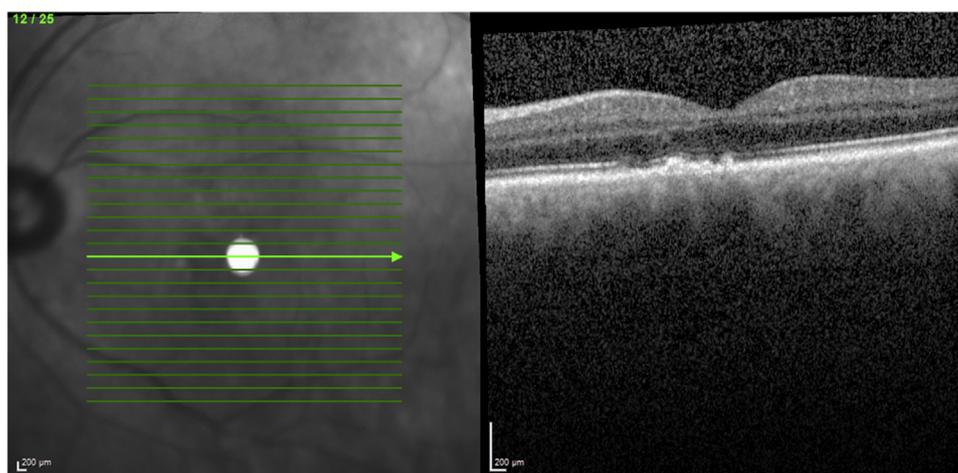


Fig 4. CSCR, resolved. Subretinal fluid again resolved with foveal contour reestablished.

REFERENCES

1. Wang M, Munch IC, Hasler PW, Prunte C, Larsen M. Central serous chorioretinopathy. *Acta Ophthalmol.* 2008;86(2):126-145.
2. 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;258(1):71-77.
3. Karadimas P, Kapetanios A, Bouzas EA. Central serous chorioretinopathy after local application of glucocorticoids for skin disorders. *JAMA Ophthalmol.* 2004;122(5):784-786.
4. Daruich A, Matet A, Dirani A, et al. Central serous chorioretinopathy: recent findings and new physiopathology hypothesis. *Prog Retin Eye Res.* 2015;48:82-118.
5. Han JM, Hwang JM, Kim JS, Park KH, Woo SJ. Changes in choroidal thickness after systemic administration of high-dose corticosteroids: a pilot study. *Invest Ophthalmol Vis Sci.* 2014; 55(1):440-445.
6. Nicholson BP, Atchison E, Idris AA, Bakri SJ. Central serous chorioretinopathy and glucocorticoids: an update on evidence for association. *Surv Ophthalmol.* 2018;63(1): 1-8.
7. Zhao M, Celerier I, Bousquet E, et al. Mineralocorticoid receptor is involved in rat and human ocular chorioretinopathy. *J Clin Invest.* 2012;122(7):2672-2679.