Case Report

Spironolactone for Secondary Central Serous Chorioretinopathy: A Challenge-Rechallenge Case

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Keywords

Central serous chorioretinopathy · Spironolactone · Corticosteroids

Abstract

Central serous chorioretinopathy (CSCR) is potentially sight-threatening and has been associated with corticosteroid use. CSCR secondary to steroid use can sometimes be challenging to treat, especially if continuing steroid use is medically necessary. In this case report we demonstrate the efficacy of spironolactone as an effective agent in countering CSCR secondary to steroid use. This challenge-rechallenge case may be helpful to clinicians in delineating a treatment paradigm for these patients.

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Published by S. Karger AG, Basel

Introduction

Central serous chorioretinopathy (CSCR) is a potentially sight-threatening condition with a complex pathogenesis that up to this time has defied precise elucidation. It is characterized by choroidal vascular dilation, increased choroidal thickness (pachychoroid), and subretinal fluid accumulation, and linked with endogenous and exogenous corticosteroid exposure [1, 2].

Up to half of patients have either a recurrent or chronic course (>3months), increasing the risk for visual morbidity secondary to retinal pigment epithelial atrophy or choroidal

neovascularization [3]. Despite this, definitive treatment has remained particularly elusive. Recently, overaction of mineralocorticoid receptor (MR) pathways in choroidal vessels has been implicated in the pathophysiology of CSCR. Glucocorticoids also have affinity for MRs, further suggestive of a targeted role for the MR pathway in this condition [4]. A study in rat eyes demonstrated similar choroidal vascular dilation and leakage after activating the glucocorticoid and mineralocorticoid pathways with corticosterone and aldosterone, respectively [4].

Isolated small reports have described the use of MR antagonists clinically. One case report described subretinal fluid resolution in a patient with CSCR 6 weeks following treatment with eplerenone 25 mg/day, with recurrence of subretinal fluid 2 months after discontinuation of eplerenone [5].

Another report described subretinal fluid resolution in 2 patients with chronic CSCR 5 weeks following treatment with oral eplerenone 25 mg/day, with no recurrence for 5 months [6]. A subsequent pilot study evaluated 13 patients with chronic CSCR of at least 4-months' duration. They were started on 25 mg/day of eplerenone, which was increased to 50 mg/day, and improvements in vision and OCT were noted [6]. More recently, reports have described the efficacy of spironolactone for CSCR [7–9], with one report in particular being a challenge-rechallenge case in a type A patient with no definitive evidence of a secondary cause [9]. These reports have helped build a database suggestive of the role of different MR antagonists having efficacy in CSCR.

It is important to remember that systemic corticosteroid use is an independent risk factor that has been associated with occurrences, prolongation, exacerbation, and recurrences of CSCR [7]. The mechanism in the paradoxical proedematous and subretinal fluid accumulation effects of glucocorticoids in CSCR pathogenesis stems from the aldosterone/MR pathway [10]. When corticosteroid use is involved, the typical treatment regimen is to avoid exogenous steroids when clinically possible. In this case, the patient required repeated steroid injections in the back to continue with activities of daily living and steroid avoidance was not possible. In cases of continued steroid exposure, CSCR management can prove quite challenging.

Though previous case reports have been published for the support of MR antagonists as a treatment option for chronic CSCR, this case is a unique contribution to the literature because case reports on CSCR thus far have focused on recalcitrant or persistent CSCR patients. In this case report we discuss a novel management regimen for a patient with recurrent, secondary CSCR due to steroid use.

Case Report

A 75-year-old Caucasian female presented to our clinic with 5 months of decreasing central vision with floaters in her left eye status after cataract surgery. Her visual acuity at that visit was OD 20/30 and OS 20/50-2 PHNI. Her past medical history was significant for 3 recent steroid injections in her back.

Her OCT revealed a pachychoroid in both eyes, with the left eye revealing foveal subretinal fluid with shaggy photoreceptors and a small pigment epithelial detachment (Fig. 1). There was no evidence of macular edema or subretinal hemorrhage on examination. Fluorescein angiography revealed an expansile dot of leakage. In concordance with the above findings, a diagnosis of CSCR secondary to her steroid injections was favored. An initial conversation of the condition, prognosis, and need for follow-up care was discussed. A course of

oral spironolactone 50 mg p.o. b.i.d. was initiated for presumed CSCR secondary to her steroid injections given 1 month previously. She was advised to avoid systemic steroid use if possible and to return for follow-up in 1 month. After 1 month, subretinal fluid was regressing only slightly, so the dose was increased to 50 mg p.o. t.i.d. (Fig. 2). One month later, all subretinal fluid had resolved. Visual acuity had improved to 20/20. (Fig. 3)

Due to improvements, the patient was slowly tapered off spironolactone to 50 mg p.o. b.i.d. with 1 month of follow-up. On the next follow-up, she continued to remain stable, so the spironolactone was tapered to 25 mg p.o. b.i.d.. On the subsequent follow-up, she was tapered to 25 mg p.o. daily with intent to stop spironolactone if continuously stable on the next follow-up. At the following follow-up she had a recurrence of subretinal fluid with an associated pigment epithelial detachment, which was also 3 weeks following another steroid injection in her back (Fig. 4). Her spironolactone was increased to 50 mg p.o. b.i.d. again and the subretinal fluid regressed 2 months later. She was again tapered to 25 mg p.o. b.i.d. and maintained on this maintenance dose.

It was apparent from her debilitating back condition that she would need steroid injections in her back approximately every 6 months, so the decision was made not to further taper her dose. She was monitored every 2 months following these injections for over a year and despite getting 2 more steroid injections in this time period, she had no recurrent subretinal fluid. Given her interim stability, the decision was made to conduct a retreat of spironolactone taper to 25 mg p.o. daily. Three weeks later she presented for a nonscheduled appointment for acute metamorphopsia and had recurrent subretinal fluid with a pigment epithelial detachment. Given that this was approximately 6 months after her last steroid injection, it was thought to be related to a taper of her spironolactone maintenance dose rather than an acute response to a steroid injection as she had previously. Her spironolactone dose was increased to 25 mg p.o. b.i.d., and 1 month later her metamorphopsia and subretinal fluid had resolved. Given this recurrence and her overall tolerance of the 25 mg p.o. b.i.d. dosage, she was maintained on this dose with no recurrence for the ensuing 6 months.

Conclusion

Increasing evidence supports the increased risk of exogenous steroids with CSCR. When corticosteroid avoidance is not possible, this can lead to recalcitrant or recurrent CSCR with potential visual loss. This patient required ongoing steroid use to maintain her activities of daily living, and her CSCR recurrences were successfully managed with spironolactone. This challenge-rechallenge case shows definitively that spironolactone was therapeutic in dehydrating the subretinal space and inducing remission in this patient's secondary CSCR. This case may be helpful to clinicians in delineating a treatment paradigm for managing these patients. To our knowledge, this is the first case of treating a patient with secondary CSCR with spironolactone in a challenge-rechallenge case. This case demonstrates an avenue for managing patients who require steroids for maintaining activities of daily living and develop secondary CSCR in response to this. Titrating spironolactone doses judiciously and safely in response to this steroid-induced CSCR can be an important aspect of this therapeutic regimen. Further research is needed in randomized controlled trials to better understand the precise mechanism.

Statement of Ethics

The authors confirm that the subject has given informed consent and that the study protocol has been approved by the institute's committee on human research.

Disclosure Statement

The authors have no disclosures.

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Fig. 1. OCT reveals subfoveal subretinal fluid and shaggy photoreceptors.

Fig. 2. The image depicts a slight reduction in subretinal fluid compared to Figure 1.

Fig. 3. A dry retinal contour is revealed with some retinal pigment epithelial stippling from previous central serous chorioretinopathy activity.

Fig. 4. The image reveals a slight recurrence of subretinal fluid.