Central Serous Retinopathy in the Fellow Eye following Treatment for Probable Vogt-Koyanagi-Harada Disease

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ABSTRACT

Purpose: To report a case of probable Vogt-Koyanagi-Harada (VKH) leading to central serous chorioretinopathy (CSR) in the fellow eye following treatment with systemic steroids.

Materials and methods: Patient presenting with unilateral probable VKH was treated with intravenous methylprednisolone and developed CSR in the fellow eye. This eye further went on to develop VKH eventually.

Results: We describe an unusual case of probable VKH disease which developed CSR in the fellow eye following treatment and eventually developed characteristics of probable VKH in that eye too.

Conclusion: CSR following systemic steroids is a well-known entity. In this case the patient developed CSR in the fellow eye of VKH and eventually developed characteristics of VKH. The clinical course along with the follow-up is discussed to manage this unique situation.

Keywords: Central serous retinopathy, Probable VKH, Steroids.

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INTRODUCTION

Central serous retinopathy (CSR) is a common disorder of the retina characterized by neurosensory detachment of the macula. We report an unusual case of probable Vogt-Koyanagi-Harada’s (VKH) disease which developed CSR in the fellow eye after treatment with steroids. Patient recovered from CSR but later went on to develop characteristics of probable VKH in that eye too.

CSR is a condition described as serous detachment of the retina. It is associated with a number of risk factors including type A personality, psychosocial stress, pregnancy and hypercortisolism. Exogenous administration of steroids and endogenous hypercortisolism have been associated with CSR. There have been reports of CSR following treatment with steroids for ophthalmic disorders, such as uveitis associated with sarcoidosis and juvenile idiopathic arthritis. In this case patient developed CSR in the fellow eye following treatment with steroids for posterior uveitis suggestive of probable VKH. Eventually the CSR regressed and the same eye went on to manifest features of probable VKH as well.

CASE REPORT

A 45-year-old female patient reported to us with decreased vision in right eye for 3 days. Her BCVA was 20/120 in the right eye and 20/20 in the left eye. On examination anterior segment was within normal limits. Fundus examination revealed extensive multifocal serous exudation in the right eye and localized chorioretinal scar in the superotemporal equatorial region of the left eye. Fluorescein angiography revealed multiple mottled areas of early hypofluorescence followed by late hyperfluorescent dots. Since, there were no associated systemic features of VKH a diagnosis of probable VKH was made and the patient was started on intravenous methylprednisolone, 1 gm daily for 3 days followed by a course of oral steroids under physician supervision. At 1 month follow-up the exudation seemed to have regressed and BCVA improved to 20/80.

Patient was maintained on low dose steroids (10 mg/day) with regular follow-up. After 8 months she again complained of decreased vision in the left eye. BCVA was 20/80 in both eyes. Anterior segment examination of the left eye was within normal limits and the right eye had very early posterior subcapsular cataract changes and a few keratic precipitates but no flare or cells. Fundus examination showed a single sensory retinal elevation with well-defined margins (Fig. 1) and multiple chorioretinal...
atrophic patches in the right eye. Fluorescein angiography was done which showed a classic ink blot leak superior to fovea suggestive of CSR (Figs 2A and B). We treated the CSR conservatively but stopped the steroids and switched the patient to alternative immunosuppression using methotrexate (under physician supervision). At 1 month follow-up the CSR had regressed and the BCVA improved to 20/30. The right eye had remained stable through this phase. The patient was followed up monthly and was continued on systemic immunosuppression. After 6 months, the patient had further fall in her vision in the right eye due to increase in the posterior subcapsular cataract. Posterior segment examination showed multiple depigmented atrophic lesions suggestive of sunset glow fundus (Fig. 3). She was advised cataract extraction. A month later she underwent cataract extraction with intraocular lens implantation and an uneventful postoperative recovery. At this stage the left eye was quiet with no evidence of any inflammation.

After 1 month she presented with decreased vision in the left eye for 3 days. BCVA was 20/40 in both the eyes. The right eye fundus examination was same as before and no fresh activity was seen while the left eye revealed multiple serous elevations along with disk hyperemia (Fig. 4). Simultaneous fluorescein and indocyanine green (ICG) angiography was done. Fluorescein angiography showed diffuse mottled areas of early hypofluorescence followed by late hyperfluorescence with areas of multiple pinpoint hyperfluorescence (Fig. 5). ICG showed early choroidal and stromal vessel leakage and hypofluorescent dark dots (Fig. 6) with diffuse late choroidal hyperfluorescence. OCT showed multiple sensory elevations (Fig. 7). It was felt that the left eye had also developed characteristics of probable VKH. At this stage she was started on intravenous methylprednisolone 1 gm daily for 3 days followed by oral steroids. She responded well and recovered to BCVA of 20/30 within 15 days. She has been switched again to immunosuppression therapy since then and has remained stable for 6 months.
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Those patients who are treated later in the course of the disorder have a more guarded prognosis for recovery of visual acuity and probably have a greater risk for chronic inflammation.4

CSR appears as a blister of transparent fluid on fundus evaluation. There have been reports of CSR following treatment with steroids for ophthalmic disorders, such as uveitis associated with sarcoidosis and juvenile idiopathic arthritis.2 Usually CSR typically can come up with high doses of steroids and it is indeed rare for it to occur at a time when the patient has been on low dose for 8 months.

To the best of our knowledge CSR in the fellow eye following treatment with steroids for VKH has not been reported. Moreover, once the CSR resolved the same eye also went on to develop VKH characteristics which were treated with systemic steroids.

Here, it was important to make the diagnosis of CSR in the fellow eye since, a serous elevation in the other eye of a patient having had a VKH episode could be easily misinterpreted clinically. Fluorescein angiography findings revealing the typical ink blot leak was the key diagnostic feature of CSR and ruled out the possibility of VKH in this situation. Without the angiography one could have very easily presumed that the serous elevation could be an onset of VKH and it would only be natural to start such a patient on higher dose steroids to treat it. However, once the diagnosis of CSR was made it became imperative to stop the ongoing steroids and in fact the timely stoppage of oral steroids helped early regression of the condition.

This case also highlights the importance of anticipating the occurrence of CSR in patients on long-term steroids and the need to switch them to alternative immunsuppression once the acute phase is stabilized.

DISCUSSION

VKH disease is a multisystem disorder characterized by granulomatous panuveitis with exudative retinal detachments that is often associated with neurologic and cutaneous manifestations.

The key to successful therapy for VKH disease is early and aggressive treatment with systemic corticosteroids.

Fig. 5: Fluorescein angiography of left eye: Late angiographic phase showing large hyperfluorescent areas due to pooling of dye under the sensory retina

Fig. 6: Hypofluorescent dark dots in ICG angiography

Fig. 7: Multiple serous elevations in OCT

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REFERENCES

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