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CASE REPORT

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Central serous chorioretinopathy due to low dose exogenous corticosteroid administered for a bee sting: a complication or coincidence?

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Abstract

Central serous chorioretinopathy (CSCR) is a posterior segment disorders characterized by localized and limited serous retinal detachment and/or retinal pigment epithelium detachment. The etiopathogenesis has not been fully elucidated. Local or systemic steroid administration through various routes and endogenous steroid increase are known to cause CSCR. It is believed that steroids cause CSCR development by delaying the healing of retinal pigment epithelium or increase choriocapillaris permeability through various mechanisms. The role of an allergic reaction in CSCR development is controversial. We evaluated a case that developed CSCR following a single intramuscular injection of 8 mg dexamethasone (Dekort, Deva) for a bee sting in this study. The systemic steroid dose used in our case was much lower than other doses reported to cause CSCR in the literature. This suggests that some molecules in the bee venom may have contributed to CSCR development in our case.

Keywords: Bee venom, central serous chorioretinopathy, corticosteroid

Introduction

Central serous chorioretinopathy (CSCR) is a posterior segment disease most commonly seen in the 20-50 years age group, characterized by localized and limited serous retinal detachment sometimes accompanied by retinal pigment epithelium detachment [1]. The etiopathogenesis of CSCR has not been fully elucidated but there are some well-defined risk factors that cause the disorder. These include type A personality, pregnancy, systemic corticosteroid (CS) use and Cushing syndrome [2,3]. There is increased CS in the body in all these conditions and many studies have indicated a relationship between CSs and CSCR pathogenesis [2-5]. Although almost all CS administration routes cause CSCR, a long usage duration and/or high doses of CS are generally required for CSCR development [4]. The role of an allergic reaction in CSR development is controversial. We evaluated a case that developed CSCR following a single intramuscular injection of 8 mg dexamethasone (Dekort, Deva) for a bee sting in this study.

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Case Report

A 29-year-old male patient presented to our clinic with a symptom of blurred vision in the left eye that had started 4 days ago. The history revealed that he had been administered 45.5 mg phenyramine (Avil, Sandoz) and 8 mg dexamethasone (Dekort, Deva) by intramuscular injection for a bee sting five days ago and had started to have blurred vision the next day. He had suffered a local allergic reaction in his arm because of the bee sting. The visual acuity was 1.0 in the right eye and 0.2 in the left eye with the Snellen chart. Intraocular pressures were bilaterally normal. The right anterior and posterior segments were normal, as was the left anterior segment. There was loss of foveal reflection on left fundus examination (Figure 1). Optical coherence tomography (OCT) evaluation revealed a normal right eye and left serous foveal detachment (Figure 2). The right eye was normal on fundus fluorescein angiography (FA) but a typical leakage described as a smokestack pattern was seen in the left macula (Figure 3).

The patient had no systemic diseases and history of any medications including CS. A diagnosis of left CSCR was made with these findings and topical nonsteroidal anti-inflammatory drops four times a day were recommended for the treatment.

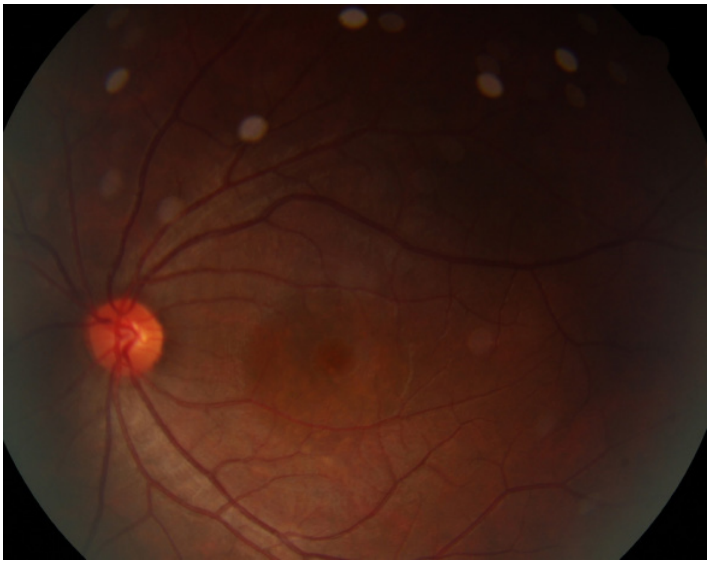

Figure 1. There was loss of foveal reflection on left fundus examination

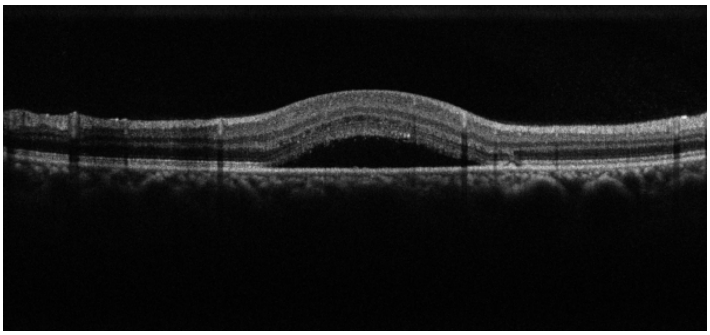

Figure 2. Optical coherence tomography (OCT) evaluation revealed a normal right eye and left serous foveal detachment

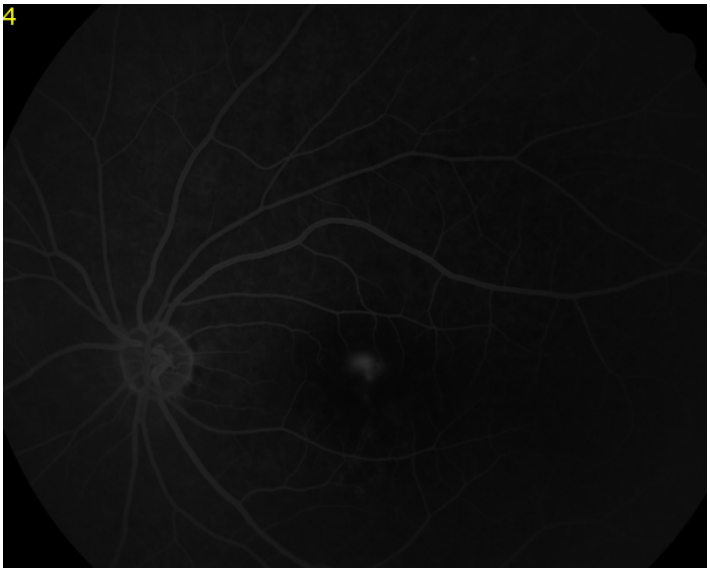

Figure 3. The right eye was normal on fundus fluorescein angiography (FA) but a typical leakage described as a smokestack pattern was seen in the left macula

Discussion

Many studies have demonstrated that CSs are a risk factor for acute CSCR [2-5]. CSs can damage the retina pigment epithelium (RPE) or the tight connection between the retina pigment epithelium (RPE) cells and delay RPE healing. It is also believed that they cause CSCR by increasing choriocapillaris permeability by various mechanisms [1,5]. Mineralocorticoid receptors and CS receptors are quite similar and endogenous or exogenous steroids are thought to also affect CSCR pathogenesis by triggering the mineralocorticoid pathway of steroids [1]. CSCR due to CS use develops more commonly in males. CS use also increases the possibility of CSCR associated with exogenous CS. Bilaterality is more common in CSCR cases due to exogenous CS use and atypical CSCR is also more common although classic CSCR can also be seen [4].

Almost all routes of CS use can cause CSCR. CSCR development has been reported after oral, intravenous, intramuscular, epidural, dermal, intraarticular, intranasal, periocular and intravitreal administration [2,5]. Development of CSCR in cases using systemic steroids usually requires long usage durations and/or high doses of CS [4]. However, a study has reported no relationship between the CS dose and treatment duration and the risk of CSCR development [6]. Koyama et al. have reported CS usage durations of 5 days to 23 years in the 17 cases they monitored with CSCR development due to systemic steroid use. The total CS dose was 650 mg-136.500 mg [7]. Chaine et al. reported a time to CSCR onset of 6 days to 10 years following systemic CS use [8]. Our case developed CSCR after a single intramuscular injection of 8 mg dexamethasone.

Bee venom contains peptides, vasoactive amines such as histamine and serotonin, enzymes such as phospholipase A and B and hyaluronidase, and non-enzyme proteins such as apamin, melitin, kinin and mastoparan [9]. Local or systemic allergic reactions and non-immunologic toxic signs can be seen following a bee sting. Allergic reactions can be early, i.e. within the first four hours, or late reactions. It is not possible to differentiate whether the etiology is allergy or steroid usage when CSCR develops following steroid usage for allergic reactions [1]. Recurrent CSCR has developed in a nonhistaminergic angioedema case and it was stated that it could be helpful to evaluate whether the bradykinin thought to play a role in the disorder's pathogenesis has an effect on the choroidal leakage in CSCR [10].

Conclusion

A local allergic reaction developed after a bee sting in our case and was followed by the CSCR symptoms. The steroid dose used by our patient is much lower than the doses reported to cause CSCR in the literature. This suggests that some molecules in the bee venom may have contributed to CSCR development in our case. Further investigations are involved to confirm that allergic reactions may play a role in the pathogenesis of CSCR.

Competing interests
The authors declare that they have no competing interest.

Financial Disclosure
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References

1. 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.

2. Wang M, Munch IC, Hasler PW, et al. Central serous chorioretinopathy. Acta

Ophthalmol. 2008;86(2):126-45.

3. Zakir SM, Shukla M, Simi Z, et al. Serum cortisol and testesterone levels in idiopathic central serous chorioretinopathy. Indian J Ophthalmol. 2009;57(6):419-22.

4. Bouzas EA, Karadimas P, Pournaras CJ. Central serous chorioretinopaty and glucocorticoids. Surv Ophthalmol. 2002;47(5):431-48.

5. Levy J, Marcus M, Belfair N, et al. Central serous chorioretinopathy in patients receiving systemic corticosteroid therapy. Can J Ophthalmol. 2005;40(2):217-21.

6. Tsai DC, Chen SJ, Huang CC, et al. Risk of central serous chorioretinopathy in adults prescribed oral corticosteroids: a population-based study in Taiwan.. Retina. 2014;34(9):1867-74.

7. Koyama M, Mizota A, Igarashi Y, et al. Seventeen cases of central serous chorioretinopathy associated with systemic corticosteroid therapy. Ophthalmologica. 2004;218(2):107-10.

8. Chaine G1, Haouat M, Menard-Molcard C, et al. Central serous chorioretinopathy and systemic steroid therapy. J Fr Ophthalmol. 2001;24(2):139-46.

9. Clarck RF. Hymenoptera. In: Olson KR, edition. Poisoning and Drug Overdose, 4th edition, NewYork Lange Medical Books/McGrawHill. 2004;225-6.

10. Edalati K, Roesch MT, Buchanan ML, et al. Central serous chorioretinopathy and idiopathic nonhistaminergic angioedema. Can J Ophthalmol. 2009;44(5):606-7.