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A patient with ulcerative colitis and central serous chorioretinopathy

Shergiye Bayramova

Ophthalmology Department, Ankara Etlik City Hospital, Varlık, 5 Halil Sezai Erkut Cd., 06170 Yenimahalle/Ankara, Türkiye

For correspondence: *drshargiyya@gmail.com*
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To report a case of a patient with ulcerative colitis and central serous retinopathy, which is a chronic ophthalmological condition that is frequently aggravated by corticosteroid treatment and may sometimes result in severe visual impairment. This case represents an interesting therapeutic dilemma pertaining to the treatment of ulcerative colitis exacerbation in a patient with this rare condition.

Keywords: *Ulcerative colitis, chorioretinopathy, corticosteroids*

INTRODUCTION

Ulcerative colitis (UC) is an inflammatory bowel disease limited to the mucosal layer of the colon. The disease course is often characterized by exacerbations requiring intensive anti-inflammatory treatment (Ilvio et al., 2011).

Central serous chorioretinopathy is a chorioretinal disease usually characterized by limited serous detachment of the neurosensory retina with a focal detachment of the affected retinal pigment epithelium that mostly affects young people and impairs central vision (Semeraro et al., 2019). This disease has a multifactorial and complex etiopathogenesis. Combining full ophthalmologic examination (anamnesis and clinical) and multimodal imaging methods will help us to reach an accurate and definitive diagnosis. The disease has features in the 25-51 age range, 40% of which show bilaterality and 80% of the patients have spontaneous regression. The probability of recurrence of the disease is around 50% in different series (Ersoz et al., 2018).

CASE REPORT

A 35-year-old female patient was admitted to our outpatient clinic with the complaint of sudden painless low vision in the right eye. It was learned from her history that she was treated with oral prednisolone due to the activation of ulcerative colitis. On examination, the best corrected visual acuity was 0.3 in the right eye and 1.0 in the left eye. Intraocular pressures of 12 and 15 mmHg in the right and left eyes.The anterior segment was normal in the slit-lamp examination. On fundus examination, punctate retinal pigmented epithelial detachment (PED) appearance with serous elevation in the macula and lower quadrant of the right eye; punctate PED appearance was detected in the left eye. Optical coherence tomography (OCT) of the patient revealed subretinal fluid and localized pigment epithelial detachment (PED) in the right macula and lower quadrant (Fig. 1), and localized PED in the left eye (Fig. 2) (Central macular thickness right: 309 microns; left: 267 microns).

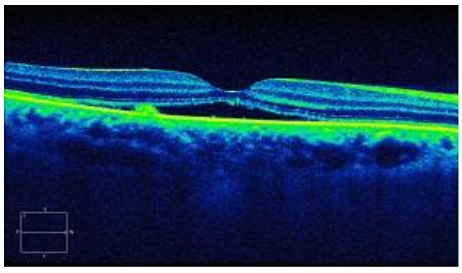

Fig. 1. The right macula and lower quadrant

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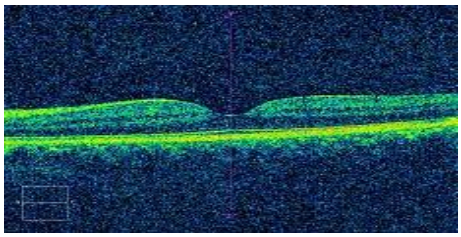

Fig. 2. Localized PED in the left eye

Fundus fluorescein angiography of the patient revealed hyperfluorescent leakage (inkblot) consistent with central serous chorioretinopathy (CSC) in the right eye and hyper fluorescence in areas matching the PED (Fig. 3).

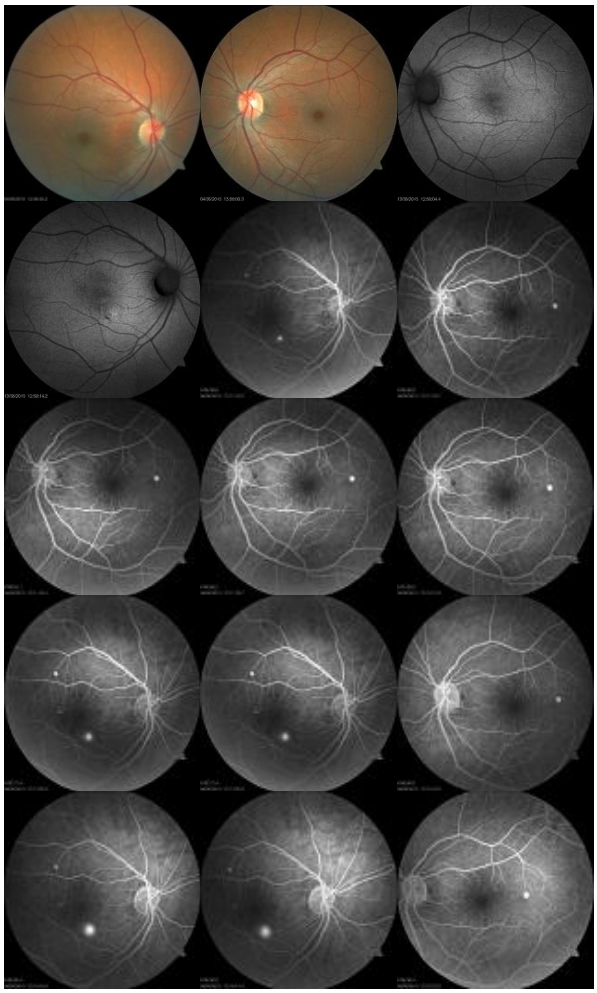

Fig 3. Fundus fluorescein angiography of the patient revealed hyperfluorescent leakage (inkblot) consistent with central serous chorioretinopathy (CSC) in the right eye

In consultation with gastroenterology, it was planned to reduce the steroid dose with the regression of activation. With the decrease in the steroid dose, it was observed that the subretinal fluid under the macula partially decreased and the subretinal fluid in the lower quadrant disappeared at the control after 3 weeks. On examination, the best corrected visual acuity was 0.3 in the right eye and 1.0 in the left eye. On OCT, the central macular thickness of the right eye was 242 microns (Fig. 4) and the central macular thickness of the left eye was 277 microns (Fig. 5).

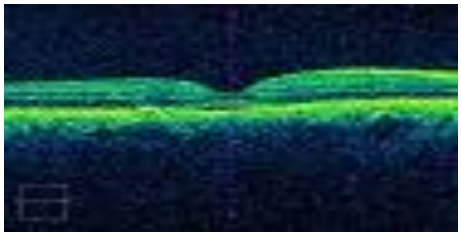

Fig. 4. The central macular thickness of the right eye was 242 microns

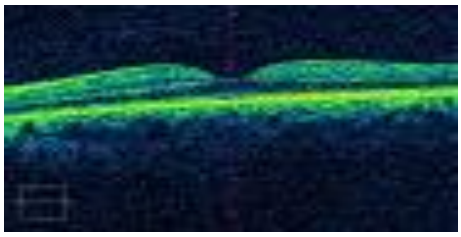

Fig. 5. The central macular thickness of the left eye was 277 microns

In the follow-up examination of the patient a month later, the best corrected visual acuity in the right eye was 0.5 and 1.0 in the left eye. Central macular thickness was measured as right: 227 (Fig. 6) left: 274 on OCT (Fig. 7). There was a punctuated PED on the right.

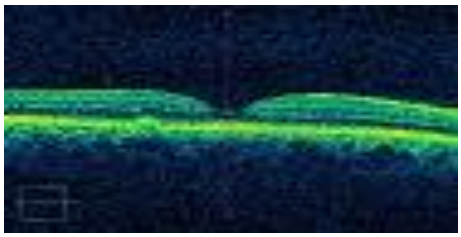

Fig. 6. Central macular thickness was measured as right: 227 on OCT

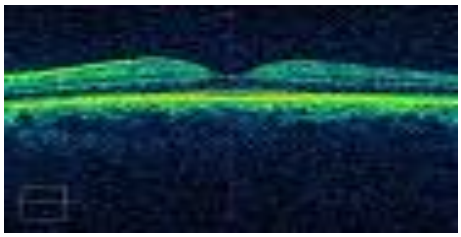

Fig. 7. Central macular thickness was measured as right: 274 on OCT

DISCUSSION

The increase in the permeability of the choriocapillaris and the deterioration of the outer retinal layers play an important role in the pathophysiology of CSC. Although exogenous steroid use is among the risk factors.Carvalho-Recchia et al. (1999) and Bouzas et al. (1999) reported in their studies that the risk of CRC development increased with the use of systemic corticosteroids. It is not understood how corticosteroids impair choroidal circulation (Carvalho-Recchia et al., 1999; Bouzas et al., 1999). While spontaneous recovery occurs in 3-6 months in most patients, follow-up is appropriate in most cases. If possible, discontinuation of the exogenous steroids used by consulting the necessary departments and changing the lifestyle to reduce the possible stress may affect the prognosis positively. The use of drugs such as infliximab, anti-TNF and intravenous cyclosporine which are alternatives to steroid therapy is controversial because of their risks such as kidney toxicity and infectious diseases (Geyshis et al., 2013).

Although the disease is defined as inflammatory choroiditis in the literature and inflammation plays a major role in its pathogenesis, it has been reported that it is the only condition worsened with corticosteroids (Darulch et al., 2015). If the feeding of the figure receptors continues it is often possible to achieve an excellent visual prognosis either spontaneously or with treatment. In cases with prolonged disease and bullous serous retinal detachment, figure receptors stay away from the choriocapillaris which is the oxygen source and as a result, ischemia and loss of figure receptors develop. This process may result in the development of choroidal neovascularization and retinal atrophy, loss of vision (Gemenetzi et al., 2010).

In diseases where steroid treatment is inevitable such as ulcerative colitis, the patient's current or past eye diseases should be questioned before the treatment is started. If visual symptoms are present, steroid treatment should be discontinued or alternative treatments should be applied, taking into account the risk-benefit ratio, by consulting an ophthalmologist (Samidh et al., 2011).

CONCLUSION

Activation of ulcerative colitis and use of oral steroids may lead to CSC.

Due to the relationship between steroids and CSC, these patients experience a treatment dilemma, whether they are treated for the primary disease or CSC and they require a multidisciplinary approach.

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S.E.Bayramova ORCHID: https://orcid.org/0000-0002-4500-3809