

Corticosteroid-associated atypical central serous chorioretinopathy in a patient with multiple sclerosis

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Dear Editor,

Multiple sclerosis (MS) is a chronic inflammatory, autoimmune, demyelinating disease of the central nervous system (CNS)^[1]. Neuro-ophthalmologic manifestations are one of the most common features occurring in the course of the disease. In particular, optic neuritis is a frequent cause of neuro-ophthalmologic manifestation in MS^[2]. However, when an MS patient complains of new visual symptoms, other possible differential diagnoses need to be ruled out, including corticosteroid associated central serous chorioretinopathy (CSC).

CSC is an idiopathic, often bilateral, asymmetric, recurrent disorder characterized by serous retinal detachment and/or retinal pigment epithelial (RPE) detachment. It occurs most commonly in healthy men in midlife. It is associated with leakage of fluid through the RPE into the subretinal space^[3]. The most consistent etiology of CSC is the use of glucocorticoids^[4-5].

In the term CSC, “central” refers to the form of the disease causing visual symptoms due to the presence of serous detachments in the macular area. Fundus imaging revealed that the patient in our study is atypical with extra-macular leaking sides.

CASE PRESENTATION

A 45-year-old female patient with a 2-year history of relapsing-remitting multiple sclerosis (RR-MS) presented to

our consultation clinic due to blurred vision and mild color desaturation of 15d duration in the left eye.

In her past medical history, she had a left optic neuritis as the first exacerbation in September in 2013 that she recovered totally with corticosteroid pulse therapy. Her second exacerbation was paresthesia and mild weakness in the left side of her body in May in 2014 for which she was again treated with corticosteroid pulse therapy, and recovered fully. She had not accepted to use any immunomodulatory drug. Two weeks earlier from her presentation to us, she was admitted to an MS center clinic due to tingling in her fourth and fifth fingers of the right hand, and in the lateral aspect of her right leg, and right sided leg weakness. She was considered to have a relapse after clinical and radiological examinations and she had received intravenous methylprednisolone (1 g/d) for 5 consecutive days. She had complained of blurred vision and color desaturation at the third day of the corticosteroid pulse therapy which she was told that she was suspected to have an optic neuritis as an ongoing part of her relapse in the same MS center. She was discharged with 100 mg/d oral prednisolone that was planned to taper down every 3d and glatiramer acetate was started as an immun-modulatory treatment for MS. Ten days after initiating oral steroid treatment, she was admitted to our university MS center due to her ongoing visual disturbances. A consultation was requested from our ophthalmology consultation clinic. She denied pain on eye movements, photophobia or injuries to the eye. Her past ocular history was unremarkable.

At neuro-ophthalmological examination her visual acuities were 20/20 in both eyes. The anterior segments of both eyes were unremarkable. Extraocular movements and alignment were normal. She had no pupillary abnormalities, in particular no relative afferent pupillary defect. Color vision by Ishihara plates was normal in each eye. The fundus of the left eye revealed serous retinal elevation approximately 2 disc diameters in size outside the fovea on the superior temporal vascular arcade. Similarly, in the right eye, there was also serous retinal elevation approximately 2 disc diameters in size outside the fovea on the inferior temporal vascular arcade and retinal pigment epithelial atrophy located temporal to the macula. Fluorescein angiography demonstrated bilateral focal dye leakage located within the macular region with a neurosensory retinal detachment outside the fovea (Figure 1). These findings were confirmed by spectral domain optical coherence tomography (SD-OCT) (Figure 1).

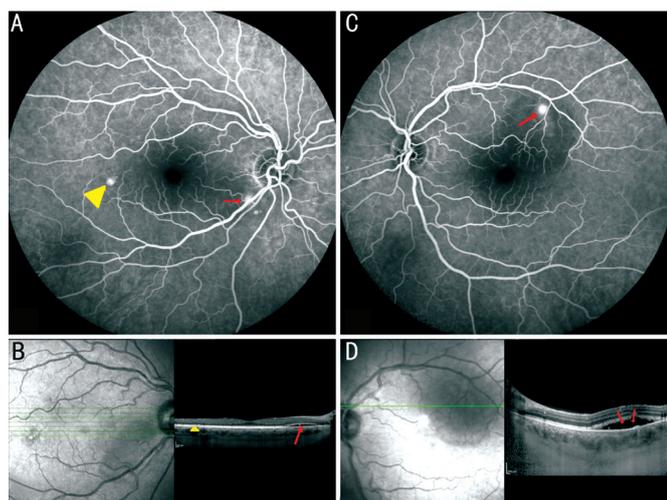


Figure 1 Fundus fluorescein angiography (FA) in both eyes A, C: Late-phase FA revealed pinpoint focus of hyperfluorescence (arrow), indicating fluorescein leakage through the RPE; B, D: OCT in the both eyes showing a serous retinal detachment (arrow) of leakage regions; A, B: RPE atrophy (arrowhead) in the right eye.

DISCUSSION

Given her history and findings, the patient was diagnosed with atypical CSC most probably due to usage of corticosteroid. Her oral steroid treatment was stopped immediately. Her SD-OCT showed complete resolution one and half months later (Figure 2). She did not have any MS relapse or CSC recurrence in her 10mo of follow-up duration. She continued to use her immunomodulatory drug for MS.

Our case describes the exacerbation of atypical CSC during steroid treatment in an MS patient whose complaint was erroneously diagnosed as optic neuritis as a part of her MS relapse.

She was a RR-MS patient whose disease onset relapse was optic neuritis in the left eye that recovered totally with corticosteroid pulse therapy. A relapse symptom can be any presentation of a CNS lesion, and it represents the unpredictability of the disease. However, the onset location can predict the location of subsequent relapses such that a previously affected optic nerve is about 6-fold more likely to be a target of a subsequent relapse affecting the same site^[6]. A relapse or worsening of a preexisting symptom might also occur in the course of steroid treatment in patients with MS. All these factors could cause diagnostic confusion as it does in our case.

Corticosteroids that speed up the recovery are used for the treatment of MS relapses^[7-8]. They have easily recognized common side effects such as gastrointestinal and psychiatric side effects^[9]. Besides, atypical CSC is a rarely seen one. The prolonging of the complaint and the unresponsiveness to corticosteroid led the neurologist to look for another reason for the visual problem and to request an Ophthalmology consultation. She was diagnosed as atypical CSC probably caused by iatrogenic exogenous hypercortisolism due to

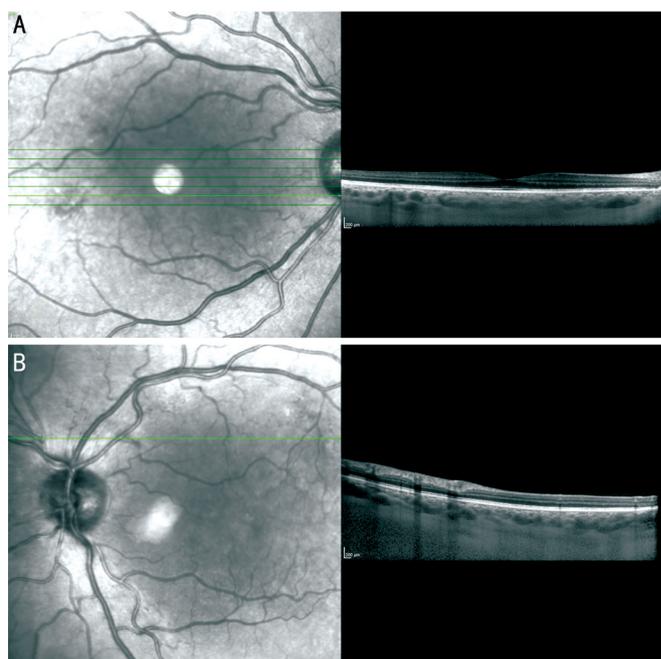


Figure 2 Optical coherence tomography of the right eye (A) and the left eye (B) showed complete resolution of neurosensory retinal detachment six weeks later after diagnosis.

steroid administration. The etiology of CSC appears to be multifactorial, including exogenous steroid use, endogenous hypercortisolism, hypertension, H. Pylori infection, use of alcohol, psychopharmacologic medications^[4-5,10]. However, increased cortisol was demonstrated to be the most consistent etiology^[4-5]. It was shown that elevated plasma cortisol aggravates the vascular permeability and capillary fragility in the choriocapillaris^[11]. In previous studies, development of CSC has been attributed to multiple routes of corticosteroids such as oral, intravenous, epidural, intra-articular, intranasal and inhaled. However, CSC due intravitreal and periocular steroid injections use were rarely reported^[3,12-13]. Corticosteroid-associated CSC may begin days to years after therapy with any form of drug delivery and it occurs bilaterally in 20% of patients. Our case's complaint was in the left eye yet her findings were in both eyes. We discontinued her corticosteroid treatment and observed complete resolution. If the fluid persisted, we could have planned to perform laser photocoagulation to extrafoveal sites of leakage to induce rapid remission^[3].

Symptoms of CSC may be indistinguishable from optic neuritis in MS and include sudden onset blurred vision in one or both eyes and decreased color vision^[14], as it was the case in our patient. On the other hand, sudden-onset of painless vision loss, normal color vision and normal pupillary reactions in the patient receiving corticosteroid raised suspicion for CSC. Nonetheless, patients may have a dual presentation. Thus, in daily practice, evaluation of MS patients presenting visual complaints should be performed in collaboration with an ophthalmologist and a neurologist.

After starting corticosteroid treatment in MS, atypical CSC is a rare condition and it can be mistaken clinically to be optic neuritis. Treatment regimens differed in these two conditions since lengthening corticosteroid treatment for MS relapse could result in permanent visual loss, once CSC occurs. The patients with MS who have complaints of new visual symptoms during corticosteroid treatment should be treated with an ophthalmologist so as to rule out other diagnostic possibilities.

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Conflicts of Interest: Kaya M, None; Kaya D, None; Idiman E, None.

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