Case Report

Multimodal Imaging for the Diagnosis of an Atypical Case of Central Serous Chorioretinopathy

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ABSTRACT

We report a case of a 52-year-old woman presented with atypical central serous chorioretinopathy (CSCR) that had been misdiagnosed as posterior uveitis and treated with systemic corticosteroids and immunosuppressive therapy, with subsequent severe chorioretinal damage. Diagnosis was straightened through multimodal imaging. Anatomical improvement was achieved after discontinuation of corticosteroids and intravitreal injection of bevacizumab. However, visual acuity remained severely impaired in one eye. Failure to differentiate atypical CSCR from inflammatory chorioretinal diseases may lead to severe and irreversible visual impairment. Multimodal imaging helps recognition of the atypical presentations of CSCR, avoiding misdiagnosis and inappropriate management.

Key words: Central Serous Chorioretinopathy, Corticosteroids, Multimodal Imaging, Prognosis, Uveitis

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INTRODUCTION

Central serous chorioretinopathy (CSCR) is a common disease characterized by serous detachment of the neurosensory retina and/or retinal pigment epithelium (RPE) that often involves the macula. Atypical and chronic or recurrent forms of CSCR may be overlooked or misdiagnosed as chorioretinal inflammatory conditions, leading to inappropriate use of corticosteroids.[1] This usually results in worsening of CSCR and irreversible chorioretinal damage and visual impairment. Various imaging modalities can be used to establish the definitive diagnosis. In this case we report the contribution of multimodal imaging approach for the diagnosis of an atypical case of CSCR.

CASE REPORT

A 52-year-old woman, with a history of well-controlled systemic hypertension, complained of decreased vision in both eyes of 3-year-duration. A diagnosis of idiopathic bilateral posterior uveitis had been made by her ophthalmologist based on a negative extensive comprehensive work-up for infectious and non-infectious diseases. The patient had been treated with several courses of oral prednisone, intravenous methylprednisolone, azathioprine, and intravenous cyclophosphamide with no improvement. The patient was referred to us to rule out a masquerade syndrome. On examination, her best-corrected visual acuity (BCVA) was 20/400 in the right eye (RE) and 20/50 in the left eye (LE). Results of anterior segment examination were unremarkable with no features of active or inactive anterior uveitis. Intraocular pressure was normal in both eyes. There was mild vitreous hemorrhage in the RE and normal vitreous in the LE. Dilated fundus examination of the RE showed normal optic disc, preretinal hemorrhage, subretinal fibrotic lesion in the macular area, epiretinal membrane (ERM), RPE changes, and a retinal detachment involving the periphery inferiorly without associated retinal breaks [Figure 1a]. Fundus examination of the LE showed area of macular RPE atrophy and area of RPE changes with no retinal detachment or other peripheral lesions [Figure 1b]. Fluorescein angiography (FA)

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revealed multifocal pinpoints, retinal neovascularization, and extensive RPE changes in the RE [Figure 1c], and multifocal pinpoints in the LE [Figure 1d]. Indocyanine green angiography (ICGA) showed dilation of choroidal veins and multiple areas of choroidal vascular hyperpermeability in both eyes [Figures 1e-h]. Optical coherence tomography (OCT) showed serous retinal detachment (SRD), ERM, and a subretinal hyperreflective lesion in the RE corresponding the fibrotic lesion seen clinically [Figure 1i]. B-scan ultrasonography of the RE confirmed the presence of retinal detachment without associated vitreous traction, choroidal tumor, or infiltration. Ultrasonography results of the LE were unremarkable.

A diagnosis of chronic CSCR exacerbated by corticosteroids and complicated by retinal neovascularization and subretinal fibrosis in the RE was retained. Corticosteroids were gradually tapered and immunosuppressive therapy was stopped. As photodynamic therapy was not available in our department, a single intravitreal injection of bevacizumab was performed in the RE. Three months later, BCVA remained unchanged (20/400) in the RE and improved to 20/32 in the LE. Intravitreal and preretinal hemorrhage in the RE had resolved, and the retina had reattached. Fluorescein and ICG angiographic features of active CSCR had resolved [Figure 2]. SRD had partially resolved in the RE on OCT.

DISCUSSION

Atypical CSCR may be misdiagnosed as Vogt Koyanagi Harada disease, sympathetic ophthalmia, posterior scleritis, multifocal choroiditis, serpiginous choroiditis, idiopathic posterior uveitis, or other uveitic entity that all usually require corticosteroid treatment.[1] The use of systemic corticosteroids in such cases is not only ineffective, but it usually exacerbates the condition, leading to bilateral, severe, and chronic CSCR with multifocal RPE detachments or diffuse retinal pigment epitheliopathy. Unusual findings can occur including acute bullous retinal detachment, subretinal fibrin, subretinal fibrosis, hard exudates, and even retinal neovascularization.[1-3]

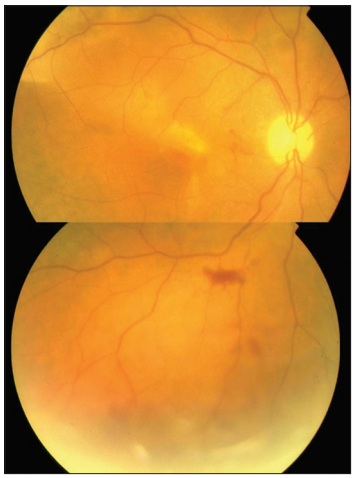

Figure 1a: Fundus photograph of the RE shows normal optic disc, preretinal hemorrhage, subretinal fibrosis, RPE changes, and inferior retinal detachment

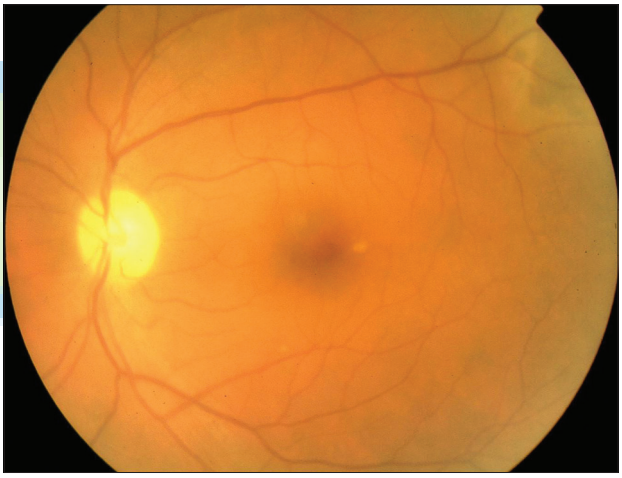

Figure 1b: Fundus photograph of the LE shows area of macular RPE atrophy and RPE changes

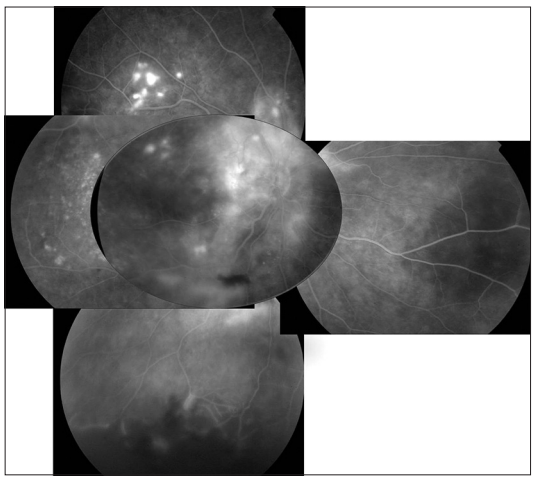

Figure 1c: Late-phase fluorescein angiogram of the RE shows juxtapapillary retinal neovascularization, extensive RPE changes and multifocal pinpoints.

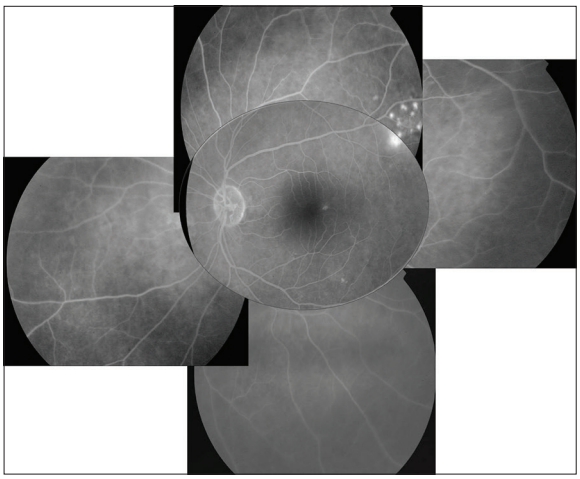

Figure 1d: Late-phase fluorescein angiogram of the LE shows multiple pinpoints

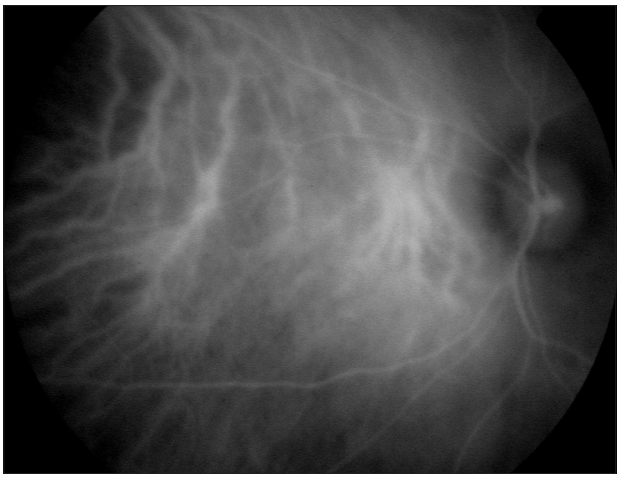

Figure 1e: Mid-phase indocyanine green angiogram of the RE shows dilatation of choroidal veins

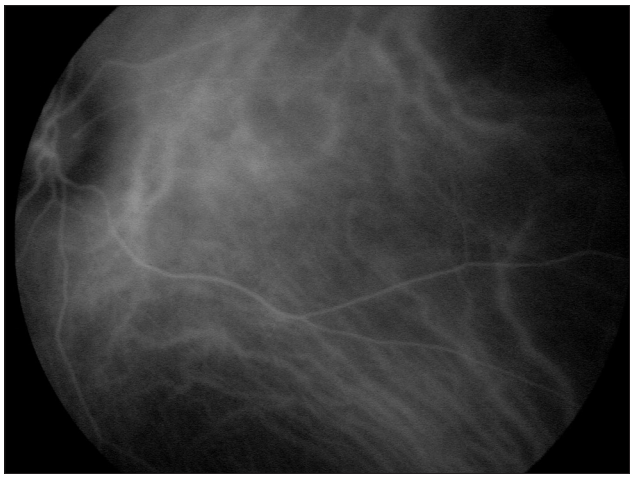

Figure 1f: Mid-phase indocyanine green angiogram of the LE shows dilatation of choroidal veins

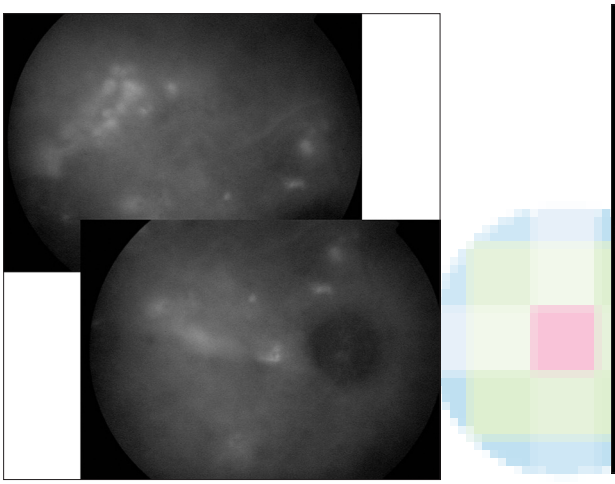

Figure 1g: Late-phase indocyanine green angiogram of the RE shows multiple areas of choroidal vascular hyperpermeability

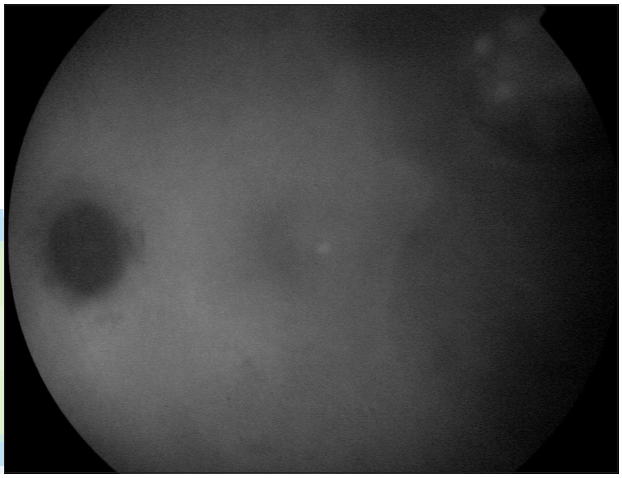

Figure 1h: Late-phase indocyanine green angiogram of the LE shows multiple areas of choroidal vascular hyperpermeability

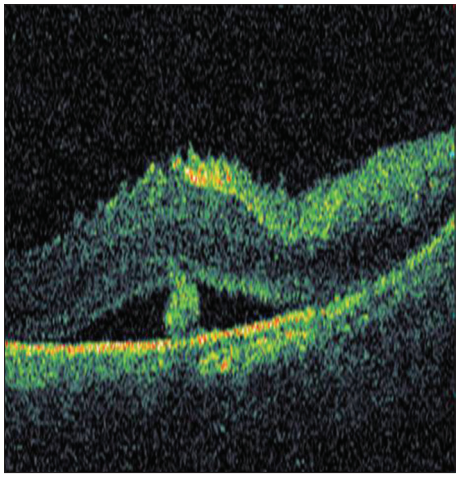

Figure 1i: OCT revealed SRD, ERM, and a subretinal hyperreflective lesion in the RE

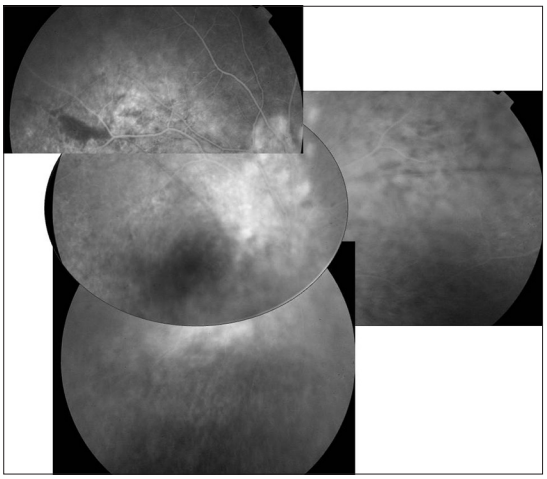

Figure 2a: Late-phase fluorescein angiogram 3 months later shows resolution of pinpoints in the RE

In our patient with CSCR exhibiting unusual and atypical findings, the use of multimodal imaging, including OCT, FA, ICGA, and ultrasonography, provides us clues for the definitive diagnosis. These mainly included multiple pinpoint leakage on FA, multifocal choroidal hyperpermeability on ICGA, dome-shaped pattern of SRD on OCT, and the absence of

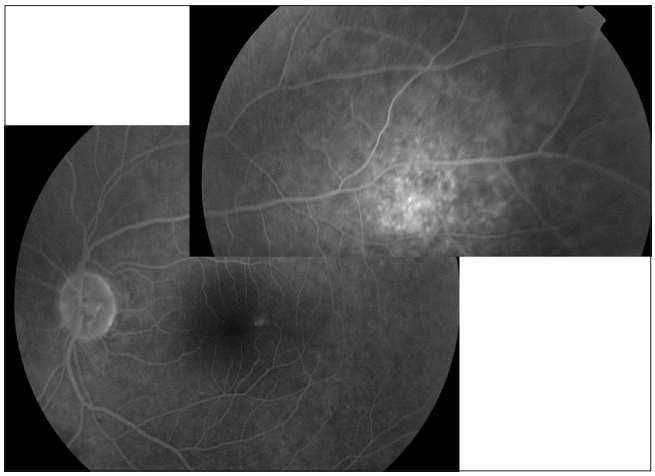

Figure 2b: Late-phase fluorescein angiogram 3 months later shows resolution of pinpoints in the LE

vitreoretinal traction or choroidal thickening or infiltration associated with retinal detachment on ultrasonography.

New imaging modalities including high-depth spectral domain OCT and auto fluorescence show specific features that may help in differentiating atypical CSCR from inflammatory conditions.[4,5]

In patients with corticosteroid-induced or worsened CSCR, discontinuation of corticosteroids should be the first management step. Many cases of CSCR actually improved following discontinuation of corticosteroid therapy with resolution of SRD, decreasing or disappearing of visual symptoms and improvement of VA.[1,6] Patients with persistent SRD despite discontinuation of corticosteroids may benefit from laser photocoagulation to active focal RPE leaks on FA, photodynamic therapy, transpupillary thermotherapy, acetalozamide, and intravitreal bevacizumab.[1,6,7] In our patient intravitreal bevacizumab helps in resolution of SRD and involution of preretinal neovascularization. However, anatomic improvement was not associated with concomitant VA improvement due to residual changes including extensive RPE changes, subretinal fibrosis, and ERM.

CONCLUSION

A careful clinical examination and appropriate use and interpretation of multimodal imaging are mandatory to differentiate CSCR from any chorioretinal inflammatory condition and to prevent severe and irreversible visual damage resulting from misdiagnosis and management mistakes.

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