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consistent with ischemic vasculitis. There have been a few previous reports about CSS patients without ANCA who developed CRAO. These cases may indicate that the ANCA status is not a useful criterion for the classification of ocular inflammation in CSS.

CSS is treated with 30-40 mg/day of prednisolone in a light case and a moderate case. In a severe case, it is treated with 60 mg/day prednisolone or by steroid pulse therapy combined with immunosuppressant. When it is steroid resistant, a high-dose intravenous gamma globulin may be used. Because peroneal nerve paralysis appeared during treatment with oral prednisolone, we thought that he was a severe CSS case. For that reason, steroid pulse therapy and low-dose cyclophosphamide were used together in this case.

According to previous reports, CRAO associated with CSS had a poor visual outcome. It might be due to low-dose systemic corticosteroids (prednisolone \( \leq 60 \) mg) or delay in performing steroid pulse therapy. In the present patient, steroid pulse therapy had already been started on the day before the occurrence of CRAO and the final visual acuity was 20/30. Therefore, early steroid pulse therapy may be effective for CRAO in CSS patients. But it remains possible that in this case, steroid pulse therapy increased platelets’ aggregation because coagulation parameters were not examined during a steroid pulse therapy. In addition, more cases are needed to confirm our hypothesis.

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Traumatic chorioretinal folds treated with intra-vitreal triamcinolone injection

Kook Young Kim, Hyung-Woo Kwak, Moosang Kim', Seung-Young Yu

A 34-year-old male visited the hospital due to decreased visual acuity in the left eye following an injury from a car accident. In the left eye, best-corrected visual acuity (BCVA) was hand motion and intraocular pressure (IOP) was 8 mmHg. Choroidal vasodilation and chorioretinal folds were observed by spectral domain-optical coherence tomography (SD-OCT). Topical and systemic steroid treatments did not improve the chorioretinal folds. Twelve months after the injury, intra-vitreal triamcinolone (4 mg/0.1 ml) was injected. Six months after intra-vitreal triamcinolone injection, BCVA in the left eye had improved to 20/100. Fundus examination showed improvement in retinal vascular tortuosity and SD-OCT revealed improvements in choroidal vasodilation and chorioretinal folds. Intra-vitreal triamcinolone injection (IVTI) was effective against traumatic chorioretinal folds with no recurrence based on objective observation by fundus photography and SD-OCT.

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Chorioretinal folds are a rare condition resulting from undulations in the choriocapillaris, Bruch's membrane, retinal pigment epithelium (RPE), and sensory retina. Chorioretinal folds can be idiopathic or due to orbital tumor, thyroid disorder, orbital cellulitis, papilledema, hypotony, scleritis, retinal detachment, uveitis, sclera buckling, or trauma. The exact pathogenesis of chorioretinal folds is unclear. Newell[1] suggested that the folds occur due to the attachment of the Bruch's membrane to the choriocapillaris and factors causing congestion in the choriocapillaris. Friberg[2] postulated that chorioretinal folds are caused by direct tension exerted at Bruch's membrane, which destroys the balance of choroidal tensile force in the choroid with a normal intraocular pressure (IOP). Management of chorioretinal folds varies depending on etiology. Thus, for proper management it is critical to determine the etiology through diagnostic tests.

To our knowledge, this is the first reported case of traumatic chorioretinal folds that did not respond to systemic steroid treatment but improved after intra-vitreal triamcinolone injection (IVTI) based on objective observation by fundus photography and spectral domain-optical coherence tomography (SD-OCT).

Case Report

A 34-year-old male was injured in a car accident. Facial computed tomography showed multiple facial fractures [Fig. 1a]. Best-corrected visual acuity (BCVA) was 20/20 in the right eye and 10-cm hand motion in the left eye. Spherical equivalents were -1.25 D in the right eye and -6.50 D in the left eye, which showed myopic changes. IOP was 17 mmHg in the right eye and 8 mmHg in the left eye. Fundus examination revealed vitreous hemorrhage and retinal hemorrhage. SD-OCT (Cirrus HD-OCT, Carl Zeiss Meditec, Dublin, CA, USA) showed a lamellar macular hole [Fig. 2a].

Three days after the injury, BCVA was 20/2000 and IOP was 9 mmHg in the left eye, and SD-OCT showed chorioretinal folds and choroidal vasodilation. Two weeks after the injury, facial reconstruction for multiple facial fractures was performed at the department of plastic surgery in our hospital [Fig. 1b]. One month after the injury, fundus examination revealed constant chorioretinal folds, vitreous hemorrhage, retinal vascular tortuosity, and SD-OCT showed undulations of the RPE–Bruch’s membrane complex and choroidal vasodilation in the posterior pole, and no progression of lamellar hole to full-thickness macular hole [Fig. 2b].

Treatment with topical prednisolone acetate and systemic prednisolone (30 mg/day) for 3 months led to absorption of the vitreous hemorrhage but no improvement in visual acuity, IOP, or chorioretinal folds, and spherical equivalents changed to -0.50 D in the left eye. After 12 months of treatment there was still no significant change in BCVA. Fundus examination showed retinal vascular tortuosity and continuous chorioretinal folds in the posterior pole region of the left eye [Fig. 3a]. The young patient wanted no more delay in treatment and hoped for more aggressive treatment. Through full explanation of the expected effects and possible complications of IVTI, informed consent was obtained from the patient before injection and intra-vitreal injections were performed using standard techniques. One week after IVTI (4 mg/0.1 ml), BCVA was 20/400 and IOP was 8 mmHg in the left eye. Six months after injection, BCVA was 20/100 and IOP was 14 mmHg in the left eye. Fundus photography revealed that retinal vascular tortuosity had disappeared and SD-OCT showed improvement of the chorioretinal folds and choroidal vasodilation [Fig. 3b].

Discussion

Chorioretinal folds develop as a result of various conditions such as orbital tumor, thyroid ophthalmopathy, pseudotumor, orbital cellulitis, papilledema, hypotony, choroidal melanoma, scleritis, retinal detachment, uveitis, sclera buckling, uveal effusion, or trauma. The mechanism underlying the development of the chorioretinal folds, however, is unknown.[1,2] Based on ultrasound, Cappaert et al.[3] suggested that scleral thickening and subsequent shrinkage have a role in the development of chorioretinal folds. Bullock and Egbert[4] concluded that forces compressing the Bruch's membrane as well as the adjacent RPE layer and choriocapillaris layer lead to the folds.

Figure 1: (a) Pre-operative facial computed tomography scan showing multiple fractures of the left inferior and maxillary sinus wall, and intact eyeball, (b) Postoperative facial computed tomography scan showing fixation of the fracture sites with titanium plates and screws, and Medpore®

Figure 2: (a) Pre-operative fundus photograph showing vitreous hemorrhage and retinal hemorrhage, (b) Postoperative fundus photograph showing improvement of chorioretinal folds and choroidal vasodilation.
Chorioretinal folds are usually narrow and located posterior to the equator with alternating dark and light bands. Most of the folds are orientated horizontally and point toward the optic disc, although they can also have vertical, oblique, irregular, and reticular patterns, and generally do not extend past the equator. The pattern of light and dark bands shown by fluorescein angiography may be explained by differences in RPE density. The banding patterns are inverted between fluorescein angiographic and autofluorescence images.\(^5\)

This case showed continuous folds of the RPE–Bruch’s membrane complex. Hypotony and cyclodialysis cleft cause chorioretinal folds. Although ultrasound biomicroscopy (UBM) did not show the definite cyclodialysis cleft, myopic changes in refractive index and phacodonesis were observed in comparison with the contralateral eye, which may be due to ciliary damage and a lasting low IOP associated with blunt trauma.\(^6\)

Kohno \textit{et al}.\(^7\) postulated that trauma to the eye, even when relatively mild, causes various types of injury to the choroidal vessels, such as delayed filling in choroidal veins, intra-choroidal leakage, delayed filling of the choroidal arteries, and changes in the choroidal vasculature together with impairment of the choriocapillaris observed on indocyanine green angiography (ICGA). In the present case, the choroidal vasodilation observed on SD-OCT might be related to choroidal vessel congestion, which is a predisposing factor for development of chorioretinal folds.

One treatment option for chorioretinal folds is administration of systemic steroids, which reduce the permeability of the outer blood–retinal barrier, induce resorption of exudation, and downregulate inflammatory stimuli. In this case, systemic steroids for 3 months did not affect the chorioretinal folds, whereas a single dose of IVTI (4 mg/0.1 ml) led to improvement of the chorioretinal folds as observed by SD-OCT. A higher therapeutic concentration in the vitreous can be achieved with IVTI compared with systemic steroid therapy, affecting not only the inner and outer retinal layers, but also the choroidal vessels, which do not respond to systemic steroid therapy. Triamcinolone acetonide has the capacity to reduce adhesion molecule expression and permeability of choroidal vessels, and exhibits vasoconstrictive effects that may contribute to its effectiveness in treating ocular neovascularization.\(^8,9\) In addition, that increased IOP caused by IVTI has contributed to the resolution of chorioretinal folds is considered.

In conclusion, IVTI induced the regression of traumatic chorioretinal folds due to reduction of permeability of the choroidal vessels, inflammatory reaction, and increased IOP. Therefore, IVTI is a considerable treatment option when traumatic chorioretinal folds do not respond to conservative treatment. The efficacy of IVTI in chorioretinal folds was observed objectively by fundus photography and SD-OCT in this case.

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