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CASE REPORT

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Intravitreal triamcinolone acetonide in sympathetic ophthalmia

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Abstract *Purpose:* To report the result of intravitreal triamcinolone acetonide in the treatment of sympathetic ophthalmia. *Methods:* A 29-year-old woman who suffered from sympathetic ophthalmia and who was being treated with systemic corticosteroid therapy received an intravitreal injection of 4 mg of triamcinolone acetonide. *Results:* By the 15th day after injection visual acuity had improved from 20/200 to 20/40 and serous retinal detachment had almost completely resorbed. Systemic corticosteroid therapy was reduced sequentially. By the third month after injection, the patient was in clinical remission. Her visual acuity was 20/20 and no serous detachment was observed. *Conclusions:* In this study, short-term improvement in the clinical picture of a patient with sympathetic ophthalmia after intravitreal triamcinolone acetonide injection was described. The results suggest that intravitreal triamcinolone acetonide injection may be an additional tool in the treatment of sympathetic ophthalmia.

Introduction

Sympathetic ophthalmia is a diffuse granulomatous panuveitis that occurs from a few days to several decades after penetrating ocular trauma. It can lead to bilateral blindness unless systemic immunosuppressive therapy is initiated in the early stage. Systemic immunosuppressive therapy, which often has to be maintained lifelong, is usually associated with severe side effects [7]. Intravitreal triamcinolone acetonide has recently been applied as treatment for various intraocular neovascular, proliferative or edematous diseases [2, 3, 5]. Jonas also showed that intravitreal triamcinolone acetonide was effective in a patient with advanced late-stage sympathetic ophthalmia [4]. The purpose of the present study was to report the effect of intravitreal triamcinolone acetonide in a patient in the acute stage of sympathetic ophthalmia.

Case report

A 29-year-old otherwise healthy woman presented with blurred vision in her right eye of 2 days' duration. She had experienced a penetrating injury to her left eye 10 years earlier. Emergency surgery was performed at a local hospital. Visual acuity was 20/63 in the right eye and no light perception in the left eye. Her left eye was phthisic. Biomicroscopic examination of the right eye showed moderate cells in the anterior chamber. Funduscopy of the right eye demonstrated multiple large areas of exudative retinal detachment. The intraocular pressure was 11 mmHg in the right eye. Fluorescein angiography showed multiple hyperfluorescent leaking foci of retinal pigment epithelium in the early phase with rapid dye staining, resulting in enlargement and coalescence of lesions (Fig. 1a). Indocyanine green angiography showed confluent areas of hypofluorescence repre-

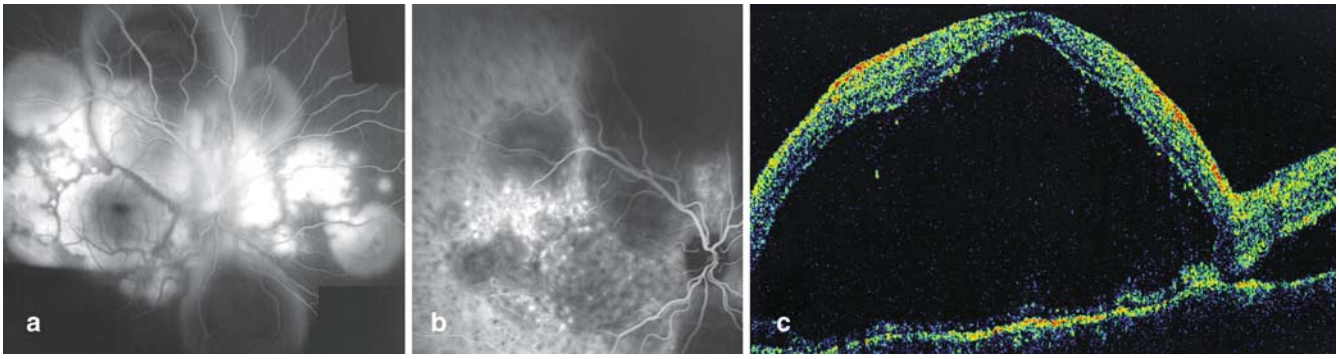

Fig. 1 (**a**) Fluorescein angiography showed multiple hyperfluorescent leaking foci of retinal pigment epithelium in the early phase with rapid dye staining, resulting in enlargement and coalescence of lesions. (**b**) Composite late-phase indocyanine green angiography study demonstrating confluent areas of hypofluorescence, representing blocked fluorescence from the overlying gravitating neurosensory detachment. Within these regions are focal areas of hypo- and hyperfluorescence, which may represent focal sites of active choroidal inflammation. (**c**) Optical coherence tomography showed large multiple serous retinal detachments involving the macular area

senting blocked fluorescence from the overlying gravitating neurosensory detachment. Within these regions were focal areas of hypo- and hyperfluorescence, which may have represented focal sites of active choroidal inflammation (Fig. 1b). Optical coherence tomography showed large multiple serous retinal detachments involving the macular area (Fig. 1c). The clinical diagnosis was sympathetic ophthalmia. The patient was immediately treated with methylprednisolone 1,000 mg/day intravenously for 3 days and then switched to oral prednisolone 2 mg/kg/day. Topical cycloplegics and corticosteroids were also given.

On the 10th day after the onset of sympathetic ophthalmia, the patient showed an initial clinical response to treatment, visual acuity in the right eye improved to 20/50 and inflammatory cells and serous detachment decreased. During this period, her body mass increased by 6 kg and she developed epigastric pain. Therefore, oral prednisolone was tapered to 1 mg/kg/day. The disease flared up while treatment was being tapered. On the 14th day after onset, the visual acuity in the right eye had decreased to 20/200. Detachment progressed to the whole retina. Despite resumed administration of oral corticosteroids in high doses (2 mg/kg/day), the condition progressively deteriorated. On the 17th day after onset, the patient was offered an intravitreal injection of triamcinolone acetonide (Kenacort-A; 40 mg/ml, Bristol-Myers Squibb, Princeton, NJ). Topical 0.5% proparacaine hydrochloride (Alcaine Alcon, Couvreur) was used for anaesthesia. An injection of 4 mg (0.1 ml) triamcinolone acetonide was performed through the inferior pars plana, 4 mm from the corneal limbus. The patient was fully informed about the experimental character of the treatment and had given her inform consent.

After the injection, oral prednisolone treatment was reduced to 1 mg/kg/day. By the 15th day after injection visual acuity had improved to 20/40, and intraocular pressure had increased to values ranging between 15 and 18 mmHg. The inflammatory cells in the anterior chamber were diminished and the serous detachment had almost completely resorbed (Fig. 2). By the third month after intravitreal injection of triamcinolone acetonide, the patient was in clinical remission. Her visual acuity was 20/20 and no serous detachment was observed. Prednisolone was gradually reduced and then

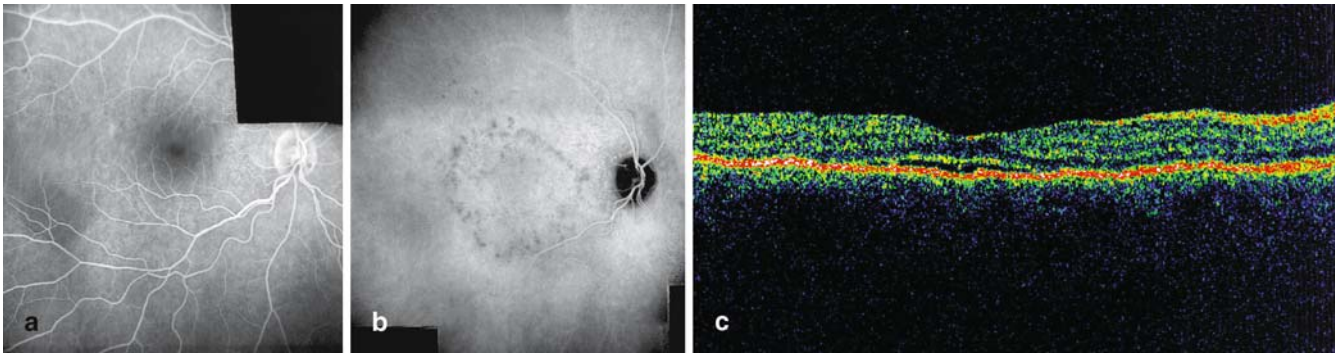

Fig. 2 On the 15th day after injection, fluorescein angiography (**a**), indocyanine green angiography (**b**), and optical coherence tomography (**c**) showed that the serous detachment had almost completely resorbed. Indocyanine green angiography also showed numerous hypofluorescent dark dots surrounding the macular area

discontinued during a further month. The ocular findings remained stable during the 8 months' follow-up. During follow-up no injection-related complications were encountered.

Discussion

The diagnosis of sympathetic ophthalmia is a clinical one depending essentially on the history of ocular injury by surgery or trauma followed by bilateral granulomatous uveitis. Occasionally, it may difficult to distinguish sympathetic ophthalmia from Vogt–Koyanagi–Harada syndrome. Typically, patients with Vogt–Koyanagi–Harada syndrome have bilateral localized serous detachments of the retina and have no history of trauma [6]. It was known that systemic immunosuppressive therapy is the main treatment for both diseases. It was also shown that intravitreal triamcinolone acetonide might be an additional tool in the treatment for sympathetic ophthalmia and Vogt–Koyanagi–Harada syndrome by decreasing intraocular inflammation and increasing visual acuity [1, 4]. The rationale for intravitreal corticosteroids parallels that established for other routes of corticosteroid administration, specifically the anti-inflammatory effect. However, the intravitreal route alleviates the pharmacologic issues of penetration and bioavailability. A potent dose of medication is delivered directly to its site of action with rapid onset.

We have described short-term improvement in the clinical picture of a patient with sympathetic ophthalmia after intravitreal injection of triamcinolone acetonide. The results suggest that intravitreal triamcinolone acetonide injection may be an additional tool in the treatment of sympathetic ophthalmia. Moreover, systemic corticosteroid use may be spared, shortened or even eliminated in selected cases of this disease when managed with intravitreal corticosteroids.

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