determinación de la patogenia del glaucoma es fundamental determinar el grado de actividad de la uveitis, el estado del ángulo mediante la gonioscopía, la facilidad de salida, el examen biomicroscópico y el fondo del ojo que permiten clasificar la uveitis subyacente y estado de las estructuras correspondientes.

El manejo del glaucoma en la uveitis, antiinflamatorio asociado a drogas antiglaucoma, cirugía filtrante, iridectomía o ciclocrioterapia, dependerá de diversos factores que es necesario analizar en cada caso entre los que destaca el nivel de visión, grado de actividad inflamatoria, mecanismo patogénico del glaucoma, tipo de uveitis, respuesta de la presión al tratamiento antiinflamatorio, a las drogas antiglaucoma, nivel de la presión ocular, estado de la papila del nervio óptico y estado del campo visual.

SUMMARY

Glaucoma is present in 20 to 50 percent of uveitis patients. Gonioscopy divides them according to the status of the chamber angle in open or closed angle secondary glaucoma. In open angle glaucoma aqueous outflow is obstructed by inflammatory debris, trabecular precipitates, steroids or trabecular fibrosis. It also may be secondary to hipersecretion or retinochoroiditis, the posibility of uveitis in a patient with chronic open angle glaucoma should not be discarded. A closed angle may be secondary to iris bombé or goniosinequias. The management of glaucoma in uveitis must consider factors such as response to antiinflamatory medication, antiglaucoma drugs, pathogenesis of the glaucoma, type of uveitis, level of intraocular pressure, visual acuity, state of the optic discs and visual fields. The analysis of these factors will determine medical treatment, filtering surgery, iridectomy or ciclo cryotherapy.

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Sympathetic Ophthalmia: A review of its Clinical Presentation and Management

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Sympathetic ophthalmia is a granulomatous inflammation affecting the uveal tract bilaterally and of unknown etiology characterized clinically by insidious onset and progressive course with exacerbations. It almost invariably follows a penetrating ocular injury, which may be accidental, surgically induced or due to perforation of the globe by infectious or neoplastic processes.

The onset of disease in the sympathizing eye is usually noted in the first three months following trauma to the "exciting" eye, but sympathetic ophthalmia has been reported to develop as early as five days or as late as 50 years following injury [1]. Prior to the advent of modern steroid and immunosuppressive therapy, the eventual visual outcome was quite poor with only 40-50% of patients retaining useful vision [2,3]; now, however, the clinical outlook is somewhat improved [4]. In this paper we will briefly review the major clinical features of this entity and will discuss management options currently available.

Sympathetic ophthalmia is an exceedingly rare entity. The incidence has been reported to be 0.9% following penetrating injury and 0.007% following intraocular surgery [5]; it has been reported as a complication of nearly every type of ocular surgery, including vitrectomy [1,6].

The sympathetic ophthalmia patient typically presents with a history of penetrating injury of the eye and a subsequent bilateral granulomatous uveitis. Subjectively, the patient may experience mild pain, photophobia, blurring of vision and accommodative fatique. On examination, there typically is ciliary injection, endothelial keratic precipitates, a thickened, sluggish iris, poorly responsive pupils and vitreous haze. Posterior segment changes may include papillitis,

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small yellow-white exudates beneath the retinal pigment epithelium (i.e., so-called Dalen-Fuch's nodules) and retinal perivasculitis. In some cases the choroiditis may be sufficiently exuberant to have produced exudative retinal detachments.

The diagnosis must usually be made on purely clinical grounds; no serological or immunological tests are available to assist in this diagnosis. Fluorescein angiography, however, may at times be quite helpful, typically showing multiple sites of choroidal leakage with later coalescence in areas of exudative detachment. This fluorescein angiographic picture is quite characteristic and similar only to that seen in Harada's disease [7,8]. Less frequently there may be early focal obscurations of the back-ground choroidal fluorescence with later staining, similar to the angiographic findings in acute posterior multifocal placoid pigment epitheliopathy [6].

Clinically, sympathetic ophthalmia must be differentiated from several other diseases, including "sympathetic irritation", reactivation of a pre-existing uveitis, lens-induced uvetis, infectious endophthalmitis and Vogt-Koyanagi-Harada syndrome.

As to the cause of this unusual bilateral granulomatous choroiditis, much has been speculated but little is actually confirmed. An infectious etiology has been postulated, including inflammatory induction by bacterial cell wall or virus derived antigens from subclinical infections following uveal injury or adjuvant-like materials generated within the inciting eye [9]. It is difficult to see, however, how this type of process could be so bilaterally symmetric. Other investigators have hypothesized an autosensitization mechanism in which uveal and/or retinal antigens are involved. Enhanced lymphocyte transformation to uveal-retinal extracts has been shown in patients with histologically diagnosed sympathetic ophthalmia [10,11]. More recent experimental studies by Rao and colleagues utilizing soluble proteins associated with retinal photoreceptors have produced an ocular inflammation in quinea pigs that is histologically quite similar to sympathetic ophthalmia [11]. These investigators postulate the release of retinal antigens to the lymphatic system via the penetrating injury, with subsequent induction of a delayed type of autoimmune reaction [13].

The only truly effective management of sympathetic ophthalmia is the prevention of its occurrence, which entails careful microsurgical wound toilet and prompt closure of all penetrating injures [1]. Of course, every attempt should be made to save any eye with a reasonable prognosis for useful vision, but in those eyes with no or barely discernible visual function and with demonstrable disorganization of the ocular contents, enucleation within two weeks after injury has long been advised to preclude the development of sympathetic ophthalmia. At one time it was believed that the use of steroids following penetrating injury would in some instances prevent the development of sympathetic ophthalmia; this has not proven to be the case [14].

There is still considerable controversy regarding the advisability of enucleating the inciting eye once sympathetic ophthalmia has commenced. Two recent papers [15,16] suggest that early enucleation of an inciting eye may improve the prognosis for the sympathizing eye, however, careful review of the data presented in these papers does not support this conclusion [17]. A review by Winter [3] of 257 cases of histologically proven sympathetic ophthalmia indicated no benefit to the sympathizing eye from enucleation of the inciting eye, whether performed briefly before, concomitant with, or subsequent to the development of sympathetic ophthalmia at various elapsed intervals following injury. Indeed, it is possible that the inciting eye may eventually provide the better visual acuity, and its enucleation would therefore deprive the patient of that visual potential [17].

Although steroids have not been shown to be effective in its prevention [14], they do constitute the mainstay of therapy of sympathetic ophthalmia. Marak [11] recommends that large doses be given early in the course of the disease, and continued for at least six months after the apparent resolution of inflammation. For the first week, 100 to 200 mg of oral prednisone is given daily, and then reduced to an every other-day dosage; steroids are eventually tapered following the clinical response of the uveitis. The systemic prednisone is supplemented with subtenon's injection of depo-steroids and topical drogs. Mydriatic and cycloplegic agents are, of course, used adjunctively.

In a number of patients, medical problems and/or systemic or ophthalmologic complications may prevent the long-term use of high doses of steroids. In these patients, supplemental treatment with immunosuppressive agents (methotrexate, azathioprine, or chlorambucil) has been shown to effectively suppress inflammation, allow reduction of corticosteroid therapy to nontoxic levels and, in some cases, induce an apparent remission of the disease. Andrasch and associates [18] presented a regimen for the use of these immunosuppressives consisting of prednisone, 10 to 15 mg orally per day, combined with azathioprine, 2 to 2.5 mg/kg/day,

or combined with chlorambucil, 6 to 8 mg orally once per day. They reported that a response to this regimen is usually noted within four weeks. Once remission is induced, the prednisone is reduced in 2.5 mg increments every one to two weeks until a maintenance dose of 2.5 mg orally per day is reached. Thereafter, slower tapering of the chemotherapy continues. They suggest that if intolerance or adverse effects to one chemotherapeutic agent are noted, the patient can usually be switched to another with no problem. Of course, during their course of chemotherapy all patients should be closely followed-up with the assistance of an oncologist/internist; marrow suppression, renal or hepatic toxicity, and the development of neoplasms can occur with the use of these agents. Nussenblatt and colleagues [19] recently reported suppression of uveal inflammation with systemic cyclosporine therapy. In light of serious side effects, it is highly recommended that all of these chemotherapeutic agents be reserved for use only in those cases of severe uveitis where conventional treatment with prednisone is not feasible or is ineffective.

As to prognosis of sympathetic ophthalmia, the only recent long-term follow-up study was performed by Mackly and Azar [4] who found that in patients treated with steroids a visual acuity of 20/60 or better was achieved in 64%. Exacerbations of sympathetic ophthalmia in 60% of the patients, in some instances with long intervals between observed relapses. Complications, including retinal detachments due to exudate, severe choroidal scarring, glaucoma, and cataracts, developed in 70% of their patients.

In conclusion, sympathetic ophthalmia is a serious entity, often with many exacerbations and a relentlessly progressive course that frequently results in very poor vision. Long-term follow-up of these patients is essential. It is hoped that with the use of large dose steroid therapy early in the course of the disease, and supplementation with immunosuppressive agents when indicated the prognosis in these patients need not be as grim as it has traditionally been. Increased understanding of the etiology of this disease will also someday assist us in more effectively preventing its occurrence and treating its manifestations.

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