Multifocal Choroiditis and Panuveitis

Immunomodulatory Therapy

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Purpose: To report our analysis of the efficacy of immunomodulatory therapy on the course of 19 patients with multifocal choroiditis and panuveitis (MCP).

Design: Retrospective, noncomparative, interventional case series.

Participants: Nineteen patients with multifocal choroiditis with panuveitis evaluated on the Ocular Immunology and Uveitis Service of the Massachusetts Eye and Ear Infirmary from 1978 to 2000.

Methods: Fifteen patients were treated with systemic immunomodulatory therapy; 4 patients (who refused therapy) were treated with systemic steroids. All patients were analyzed for control of inflammation, visual acuity outcome, and tolerance of immunomodulatory therapy.

Main Outcome Measures: Control of inflammation and visual acuity.

Results: Nineteen patients with bilateral MCP with a mean follow-up of 72.7 months were studied. Fifteen were treated with immunomodulatory agents, whereas 4 patients received only systemic steroids; these 4 developed serious systemic steroid-related complications, and 12 others had cataract and/or glaucoma related to chronic topical, regional, or systemic steroid use before immunomodulatory therapy. Two patients who refused immunomodulatory therapy lost considerable vision in three of their four eyes. Of the 15 patients treated with immunomodulatory drugs, 7 patients lost considerable vision in one eye on steroid therapy but maintained good vision in the other eye once immunomodulatory therapy was instituted. No patient lost vision in any eye once he or she was treated with immunomodulatory treatment.

Conclusions: Immunomodulatory therapy controls inflammation and preserves vision in patients with multifocal choroiditis and panuveitis. *Ophthalmology 2002;109:378–383 © 2002 by the American Academy of Ophthalmology.*

Nozik and Dorsch[1] described two patients with fundus lesions similar to those seen in the presumed ocular histoplasmosis syndrome (POHS) in 1973, except that unlike in POHS, vitreous and anterior chamber inflammation was present in the eye of these two patients. Investigations showed that these two patients had negative histoplasmin and tuberculin skin tests and normal chest x-ray films. Many articles have been published since this first report describing patients with multiple chorioretinal lesions, vitreitis, and sometimes with anterior chamber cells. Dreyer and Gass[2] first used the term "multifocal choroiditis and panuveitis" in 1984 to distinguish the condition from POHS and similar conditions (i.e., birdshot retinochoroidopathy, acute posterior multifocal placoid pigment epitheliopathy, and diffuse unilateral subacute neuroretinitis), to define the characteristic ophthalmoscopic, fluorescein angiographic, and electrophysiologic features of the disease, and to determine whether treatment with steroids improved either vision or symptoms.

Articles have previously been published with the purpose of differentiating multifocal choroiditis and panuveitis (MCP) from POHS,[3] defining the clinical features of the disease,[4] characterizing the indocyanine green angiographic features of the disease,[5] and describing its association with other conditions, especially myopia and sarcoidosis.[6] The disease has been discussed under various names: inflammatory pseudopresumed ocular histoplasmosis syndrome,[3] multifocal choroiditis associated with progressive subretinal fibrosis,[7,8] peripheral MCP,[9] multifocal choroiditis,[10] peripheral multifocal choroiditis,[11] and recurrent multifocal choroiditis.[12]

To define our inclusion and exclusion criteria for the study, we found it essential to start with specific inclusion criteria characteristics of MCP. The purpose of this report is to describe the results of treating MCP patients with immunomodulatory drugs.

Patients and Methods

Nineteen patients with MCP were seen on the Ocular Immunology and Uveitis Service of the Massachusetts Eye and Ear Infirmary during the period of 1978 to 2000. The inclusion criteria included the previously published diagnostic features of this relatively newly recognized disease entity:

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1. Bilateral, multiple (up to several hundred) choroidal lesions ranging in size from 50 to 350 μm, arranged singly, in clumps, or in linear clusters, situated in the posterior pole and/or the periphery. Acute lesions are yellow or gray and located at the level of the retinal pigment epithelium or deeper in the choroid. Older lesions are atrophic, "punched out," with variable amounts of pigment around or inside them[1–3] (Fig 1).
2. Presence of variable amounts of vitreous cells (vitreitis).[1–3]
3. Absence of diagnosable systemic disease.
4. Absence of features characterizing other diseases that could be similar (e.g., noncaseating granuloma or abnormal pulmonary function in sarcoidosis, human leukocyte antigen-A29 and abnormal electroretinogram in birdshot retinochoroidopathy, demonstration of any causative etiology).

Patients who had ocular manifestations that could be attributed to or classified under a different category or disease were excluded from the study.

The evaluation and treatment practice pattern for patients entering our service has been previously described.[13] Comprehensive history and general and ocular examinations are followed by directed laboratory evaluations, up to and including invasive (biopsy) studies as indicated to exclude infection from the differential diagnosis before proceeding to the stepwise algorithmic approach to antiinflammatory therapy.

Given the chronic and recurrent nature of MCP and the very limited long-term success of steroids in controlling this potentially blinding inflammation,[2,7,9,14,15] patients diagnosed with MCP on our service are offered the option of immunomodulatory therapy. The effects, potential side effects, and the follow-up routine (regular blood and urine tests and blood pressure check, depending on the medication used) are described in detail.

Case Reports

Case 1 (Patient 5)

A 22-year-old white female was referred to our service in December 1991 with a 10-month history of bilateral recurrent uveitis. The referring ophthalmologist thought her lesions looked like POHS, but the histoplasmin test was negative. She had complaints of blurry vision, photopsias, and floaters in both eyes. Her chest x-ray examination and blood work had been unrevealing. The patient's angiotensin converting enzyme, purified protein derivative intradermal skin test, chest x-ray, magnetic resonance imaging scan of the brain and orbit, and gallium scan were all negative or within normal limits. The chest x-ray and blood work were repeated during her follow-up. She had been seen by a dermatologist, a rheumatologist, an infectious disease specialist, and an allergist, and the only systemic diagnosis made was allergy to molds. The patient was being treated with topical prednisolone acetate, 1% four times daily to both eyes, and timolol maleate, 0.5%, twice daily in both eyes. On examination her visual acuity was 20/25 in the right eye and 20/50 in the left eye. Cells were observed in the anterior chambers, 2+ right eye and 3+ left eye, and 3+ cells were noted in the vitreous in both eyes. The fundus showed multiple small cream-colored chorioretinal lesions in the midperiphery in both eyes (active lesions), some inactive pigmented lesions, and bilateral disc edema. Fluorescein angiography showed hyperfluorescent spots in the early and late phases of the angiogram (corresponding to the clinically observed lesions). Indocyanine green angiography showed hypofluorescence at the site of the choroidal lesions. The patient was begun on diflunisal, 500 mg twice daily, and prednisone, 40 mg twice daily. The inflammation subsided, and prednisone was tapered and discontinued in 8 weeks. During the period from March 1994 to June 1996, the patient had multiple flare-ups, and her local ophthalmologist treated her with an increase in the frequency of topical steroids. The patient developed cystoid macular edema in both eyes and was then treated with regional periocular steroid injections and another course of oral steroids. In May 1997 the patient was offered the option of the immunomodulatory drug methotrexate as a steroid-sparing medication. Methotrexate was begun at 7.5 mg per week along with folic acid, 1 mg per day. The patient's visual acuity returned to 20/20 in both eyes. She has had no evidence of recurrent inflammation and has been off all systemic steroids for the past 3 years.

Case 2 (Patient 14)

In December 1985 a 31-year-old female was referred to our service for a second opinion regarding the management of her MCP of 12 years' duration. The patient had been treated with chronic topical and systemic steroids and with episodic regional steroid injections. The patient had required intracapsular cataract extraction in both eyes in 1975 without placement of intraocular lens implants. At the time of our first evaluation of the patient in 1985 her medications included prednisone, 60 mg every other day, topical prednisone acetate drops, 1% four times daily in both eyes, atropine sulfate, 1% three times daily in both eyes, and timolol maleate, 0.25% once daily in the left eye. The patient's best-corrected visual acuity was 3/200 in the right eye and 20/400 in the left eye. She had bilateral band keratopathy, 3+ flare and rare cells in the anterior chamber in the right eye, 2+ flare and rare cells in the anterior chamber in the left eye, bilateral aphakia, 3+ vitreous cells in the right eye, and 4+ vitreous cells in the left eye. Bilateral peripapillary atrophy, bilateral macular retinal pigment epithelial changes, bilateral disc edema, and active multifocal choroiditis in both eyes were noted. Laboratory studies and x-ray films were unrevealing. Cyclosporin A, 300 mg/day, was begun. It only partially controlled the inflammation, and so, azathioprine, 150 mg/day, was added. The patient's visual acuity improved to 20/200 in the right eye and 20/70 in the left eye, and the posterior segment inflammatory activity slowly resolved. But it took almost 3 years to reduce the systemic steroid dosage from 60 to 30 mg every other day, while maintaining the patient on cyclosporin A and azathioprine. By February 1996, the eyes had been free of all active inflammation for 12 months, and the patient's visual acuities were 20/200 in the right eye and 20/60 in the left eye. Bilateral optic atrophy, bilateral peripapillary atrophy, and fixed macular pathosis (retinal pigment epithelial abnormalities) were noted. The patient had now been receiving systemic steroid therapy for 22 years. In an effort to maintain complete control of inflammation and still overcome the steroid dependence for this patient, cyclosporin A and azathioprine were stopped, and chlorambucil, 6 mg/day, was begun. The dose of chlorambucil was adjusted according to the patient's response to steroid withdrawal and her hematologic parameters. The patient has been off all systemic medications since August 1998, without recurrence of ocular inflammation. Her current visual acuity is 20/200 in the right eye and 20/60 in the left eye. She has 2+ pallor of the right optic nerve and 1+ pallor of the left, macular scarring is present in the right eye, and the left eye has epiretinal membrane.

Table 1. Summarization of the Clinical

Patient	Sex	Age at onset	Prior Follow- up*	Follow-up (m)	Presenting Complaints	Presenting Signs
1	F	32	—	108	Decreased vision, floaters, photopsia	OD 20/20, OS LP. VC, CR lesions OU.
2	F	19	20 yrs	12	Decreased vision, floaters, photopsia	OD 20/25, OS 20/30. VC, CR lesions OU.
3	F	24	3 yrs	36	Decreased vision, floaters, photopsia	OD 20/200, OS CF. VC, SF.
4	M	19	—	96	Decreased vision, floaters, photopsia	OU 20/20. VC, CR lesions OU
5	F	22	10 ms	100	Decreased vision, floaters, photopsia	OU 20/25. VC, CR lesions OU
6	F	43	2.5 yrs	10	Decreased vision, floaters, photopsia	OD 20/50, OS 20/25. VC, CR lesions OU
7	F	27	2 yrs	43	Decreased vision, floaters, photopsia	OD 20/400, OS 20/20. VC, CR lesions OU, SF OD
8	F	35	6 ms	120	Decreased vision, floaters, photopsia	OD 20/40, OS 20/25. VC, CR lesions OU
9	F	80	2 yrs	5	Decreased vision, floaters, photopsia	OD 20/300, OS 20/200. VC, SF OU
10†	F	42	2 yrs	228	Decreased vision, floaters, photopsia	OD 20/20, OS 20/40. VC, CR lesions OU
11	F	74	—	60	Decreased vision, floaters, photopsia	OU 20/200. VC, CR lesions OU, ERM OD
12	M	35	—	78	Decreased vision, floaters, photopsia, photophobia	OD 20/25, OS 20/40. VC, CR lesions OU
13	F	44	—	19	Decreased vision, floaters, photopsia	OD 20/30, OS 20/40. VC, CR lesions OU
14	F	19	12 yrs	168	Decreased vision, floaters, photopsia	OD 20/200, OS 20/60. VC, CR lesions OU
15	M	13	—	5	Decreased vision, floaters, photopsia	
16	F	23	16 yrs	89	Loss of vision, floaters OD, glaucoma OS.	OD 20/40, OS no LP. VC, CR lesions OU
17	F	27	2 yrs	12	Floaters OU, decreased vision OS.	OD 20/30, OS 3/200. VC, CR lesions OU, Macular CNVM OS
18‡	F	37	3 yrs	12	Floaters, blurry vision OU, no response to steroid injection	OD 20/40, OS 20/30. VC, CR lesions OU
19	M	47	—	180	Decreased vision, floaters, photopsia OU.	OD 20/40, OS 20/50. VC, CR lesions OU

*Duration of disease before first consultation.

†This patient had normal upper body gallium scan, normal pulmonary function tests and no evidence of sarcoidosis.

‡This patient had no evidence of pulmonary or systemic tuberculosis; anti-tuberculosis treatment did not work for him.

AZA = azathioprine + = positive; C3/C4 = complement components 3/4.; CAB = chlorambucil; CF = counting fingers; CME = cystoid macular edema; CNVM = choroidal F = female; HM = hand movements; HSV = herpes simplex virus; IC = immune complexes; LP = light perception; M = male; MTX = methotrexate; TB = tuberculosis; VA = visual acuity; VC = vitreous cells.

Case 3 (Patient 7)

A 28-year-old white female was referred to our service in April 1996 for a second opinion regarding a 9-month history of peripheral multifocal chorioretinitis in her right eye. She was using topical prednisone, 1%, and timolol maleate, 0.5%, once daily in the right eye and oral prednisone, 20 mg/day. The patient's visual acuity was 20/50 in the right eye and 20/25 in the left eye. She had 0.5+ cells in the anterior chamber in the right eye and 2+ cells in the vitreous in that eye. There were multiple chorioretinal lesions in the right eye; some were active, and others were atrophic and/or pigmented. The systemic steroid was tapered, and because an infectious etiology for the patient's uveitis had not yet been excluded, serologic studies and subsequently a diagnostic pars plana vitrectomy were performed. Polymerase chain reaction analysis for herpes simplex virus, varicella-zoster virus, Epstein-Barr virus, tuberculosis, borreliosis, bartonellosis, psittacosis, and histoplasmosis were negative. An infectious disease consultant did not develop additional leads.

Inflammation in the right eye flared up on cessation of the systemic steroid therapy, which was started again at a dosage of 20 mg every other day. In July 1996, 1+ vitreous cells were observed in the left eye, and the patient's visual acuity dropped to 20/100 in the right eye and 20/40 in the left eye. The patient's visual acuities further deteriorated to 20/400 in the right eye and 20/60 in the left eye despite systemic steroid therapy; cyclosporin A, 200 mg/day, was begun. The systemic steroid was slowly tapered and eventually discontinued without recurrence of ocular inflammation. At the patient's most recent visit, February 25, 2000, she had no complaints, her eyes were quiet, her visual acuity was 20/320 in the right eye and 20/25 in the left eye. Her 3+ posterior chamber subcapsular cataract in the right eye was thought to account for much of the decreased vision in that eye.

Results

Nineteen patients satisfying the diagnostic inclusion criteria for MCP form the database for this report. Table 1 summarizes the clinical features of the study population. The disease was bilateral but not symmetric in all patients. Both eyes were affected at the same time or within a 12-month time interval. The mean (± standard deviation) age at onset was 34.8 years (range, 13–80). Fifteen patients were female and 4 were male (F/M = 15:4). The mean follow-up was 72.7 months (range, 5–228 months). Fifteen patients (30 eyes) were treated with immunomodulatory agents at some point during the course of their disease. Ten of these 15 patients had previously been treated with systemic steroids. Four

Features of the Study Population

Initial Treatment	Complications	Laboratory Findings	Last Treatment
AZA/CSA	Glaucoma, macular star OS	Negative	AZA, CSA
Systemic steroid	Unilateral glaucoma OD	Negative	CSA
Steroid, AZA/CSA	SF, duodenal perforation	Negative	Steroid, AZA, CSA
Diflunisal, systemic steroid	—	Negative	MTX, CSA
Topical steroid, antiglaucoma	Glaucoma, CME, disc edema OU	Negative	MTX
Systemic steroid	Bilateral cataract	Past EBV	Immuno. refused
Systemic and topical steroid	Unilateral glaucoma and SF OD, bilateral cataract	Past EBV	CSA
Acyclovir; systemic steroid	Cataract, glaucoma, and ERM OU	Chronic EBV	CSA
Systemic steroid	Glaucoma, SF OU	Elevated sIL-2R	Immuno. refused
Systemic steroid	Unilateral glaucoma	Noncaseating granuloma parotid, past EBV, elevated anticardiolipin antibody	MTX, leflunomide
Topical and systemic steroid	Unilateral cataract, glaucoma and ERM OU	Negative	MTX
NSAID, diamox, regional steroid	Posterior synechia, macular edema	Negative	Same as initial
MTX	Elevated liver enzyme; diabetes mellitus	Negative	Systemic steroid
CPA/AZA/CSA/CAB systemic steroid	Unilateral glaucoma, band keratopathy, macular scar, optic nerve atrophy OD	Negative	Off systemic treatment
Steroid dependent, MTX; ceftriaxone trial failure	Cushing, cataract	Negative	Systemic steroid, CSA, MTX
Regional and systemic steroid, NSAID	Glaucoma, optic atrophy, and retinal scars OS	HLA-B27 +	MTX
Systemic and topical steroid	Steroid-dependent, palpitations, water retention, bruises, mood changes, cataract OU, glaucoma OS	HLA-B27 +, EBV and HSV antibodies, cryoglobulin, ↑ IC, ↓ C3 and C4	MTX, and later AZA and steroid
Regional and systemic steroid. Anti-TB and Zovirax	Weight gain	Raji cell +, PPD +	Systemic steroid
AZA	—	Negative	CSA, AZA

neovascular membrane; CPA = cyclophosphamide; CR = chorioretinal; CSA = cyclosporin A; EBV = Epstein-Barr virus; ERM = epiretinal membrane; OD = right eye; OS = left eye; OU = both eyes; PPD =purified protein derivative test; SF = subretinal fibrosis; sIL-2R = soluble interleukin=2 receptor;

patients were not treated with immunomodulatory agents but were treated with systemic nonsteroidal antiinflammatory drugs or systemic steroids. Topical and regional steroids were used as adjunct treatment.

Documented steroid treatment–related systemic complications included duodenal perforation (case 3), cushingoid appearance, osteoporosis, and other complications (cases 15, 17, 18). Complications related to immunomodulation were seen in only one patient (case 13), whose elevated liver enzymes reverted to normal after stopping the immunomodulatory drug methotrexate. Twelve patients had cataract and/or glaucoma related to topical, regional, or systemic steroid use.

Of the 19 patients (38 eyes) in this study, 15 received immunomodulatory treatment during the course of the disease (cases 1–5, 7, 8, 10, 11, 13–17, and 19). Of the 38 eyes, 24 eyes of 16 patients (8 patients had good vision bilaterally and 8 patients had good vision unilaterally) were maintained at visual acuity of 20/60 or better (23 were maintained at 20/40 or better). Two patients (four eyes), who refused immunomodulatory treatment, lost considerable vision in three eyes (cases 6 and 9).

Of the 15 patients (30 eyes) who received immunomodulatory agents at some point during the course of their disease, 7 lost considerable vision in one eye (7 eyes) while receiving steroid therapy (cases 2, 7, 11, 14–17), but had good vision in the other eye preserved when chemotherapy was instituted. There was no vision loss for the patients who were treated with immunomodulatory drugs. Of the 30 eyes in this group, 20 have been maintained at 20/80 or better (1 eye at 20/80, 1 eye at 20/60, 18 eyes at 20/40 or better). One patient had unilateral traumatic macular scar, and another patient had bilateral subretinal fibrosis (cases 1 and 3) when they were first seen by us (Table 1).

Discussion

Nozik and Dorsch described two patients with fundus lesions similar to those seen in POHS in 1973.[1] However, in these two cases, vitreous and anterior chamber inflammation were present. Investigations showed that these two patients had negative histoplasmin and tuberculin skin tests and their chest x-ray films were within normal limits. Many articles have subsequently been published describing patients with multiple chorioretinal lesions together with vitreous cells (vitreitis) and (sometimes) anterior chamber

cells. In 1984, Dreyer and Gass[2] first used the term "multifocal choroiditis and panuveitis" to describe this entity.

MCP as a distinct entity is now defined as posterior or panuveitis and:

1. Bilateral, multiple (up to several hundred) choroidal lesions ranging in size from 50 to 350 μm, arranged singly, in clumps or in linear clusters, situated in the posterior pole and/or the periphery. Acute lesions are yellow or gray and located at the level of the retinal pigment epithelium or deeper in the choroid. Older lesions are atrophic, "punched out," with variable amounts of pigment around or inside them. The peripheral lesions may occur in curvilinear arrangements.[1–4]
2. Presence of variable amounts of vitreous cells (vitreitis).[1–4]
3. Absence of typical systemic disease associations.[1–4]
4. Absence of features characterizing other ocular diseases (e.g., human leukocyte antigen-A29 and abnormal electroretinogram suggesting birdshot retinochoroidopathy).[1–4,7–12]
5. Possible peripapillary changes, atrophy, or pigmentation[1–3] (Fig 2).
6. Possible presence of signs of anterior uveitis (e.g., anterior chamber cells, keratic precipitates or posterior synechia).[1–3]

Our experience and review of the literature suggest to us that MCP is generally resistant to long-term effective treatment with systemic and regional steroids. Palestine et al[15] found that only 25% of their cases had some benefit from steroid therapy, whereas Cantrill and Folk[7] reported that 40% responded and 60% progressed despite steroid treatment. Brown and associates[14] found that more than half their patients progressed to vision of 20/200 or less despite steroid treatment. And Nölle et al[9] reported their patients to be refractory to local and systemic steroid therapy, with deterioration in vision during steroid treatment. The resistance to long-term tolerable steroid treatment, together with the known side effects of long-term systemic steroid treatment, motivated us to examine the results of our experience in treating patients with MCP with immunomodulatory drugs.

It is clear that despite steroid treatment, MCP progresses to significant permanent visual loss in 60% to 75% of reported cases.[2,7,9,14,15] The vision loss occurs as a consequence of chronic or recurrent inflammation[2,7,9,14,15] (chronic macular edema, epiretinal membrane, other maculopathy, optic neuropathy, subretinal fibrosis, and subretinal neovascular membrane) and/or from steroid complications (glaucoma). A major impediment for steroid efficacy may be the inability of patients to tolerate the side effects of the dose of steroid that might otherwise be sufficient to be therapeutic.

Our philosophy in treating MCP has been one of complete intolerance to even low-grade inflammation and a limited tolerance to steroid use in patients for whom alternative anti-inflammatory medication is a reasonable option, in an effort to limit permanent structural damage to vital ocular structures.[16] Inflammatory activity is monitored both subjectively and objectively. The subjective signs of inflammation to which we pay attention include deterioration in visual acuity, floaters, photopsias, photophobia, and eye discomfort. The objective signs of inflammation to which we pay the most attention include yellow or gray active chorioretinal lesions, cells in the lacunae of the vitreous, and AC cells. We set our goal at complete resolution of inflammation, disappearance of recurrences and avoidance of medication-related side effects. Given the chronic and recurrent nature of MCP and the limited success of steroids in controlling this blinding inflammatory condition, we offer our patients the option of long-term immunomodulatory therapy as an alternative therapeutic approach. Our criteria for the selection of patients for immunomodulatory therapy and the guidelines for the collaborations for proper monitoring have been previously described.[17]

Just as in the case of immunomodulatory therapy for any other ocular inflammatory disease, so too for patients with MCP, the monitoring of the patient from the chemotherapeutic perspective must be performed by an individual who is, by virtue of training and experience, truly expert in such matters.[16–18]

Our long-term follow-up of 15 MCP patients, 20 eyes, treated with immunomodulatory drugs was not associated

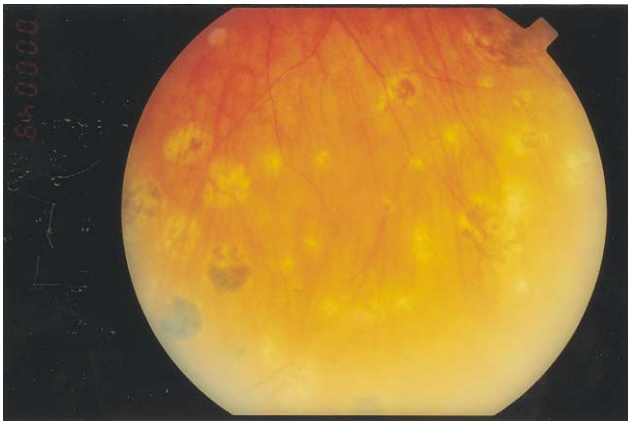

Figure 1. Multiple chorioretinal lesions; some are active (yellow) and some inactive (pigmented).

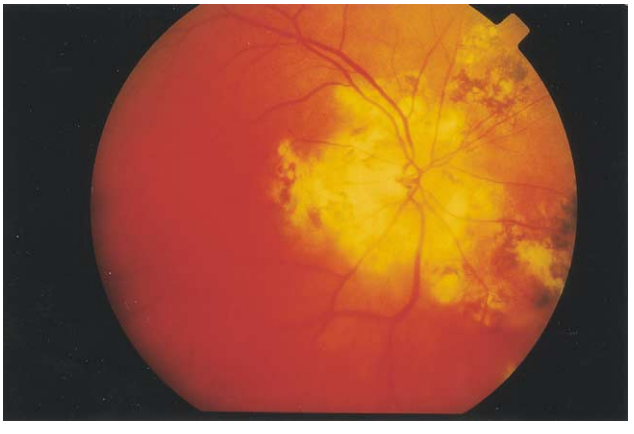

Figure 2. Peripapillary atrophy in a patient with multifocal choroiditis and panuveitis.

with any significant medication-related complications; methotrexate was stopped in one patient because of elevated liver enzymes, which reverted to normal after stopping the drug.

Conclusions

We conclude that immunomodulatory therapy can be effective and safe in controlling inflammation and preserving good vision in patients with the potentially blinding condition, MCP. A panel of 12 experts charged by the Executive Committee of the American Uveitis Society to analyze the world's literature on the matter of immunomodulatory therapy for ocular inflammatory disease agreed with this conclusion in its consensus position paper published recently.[19]

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