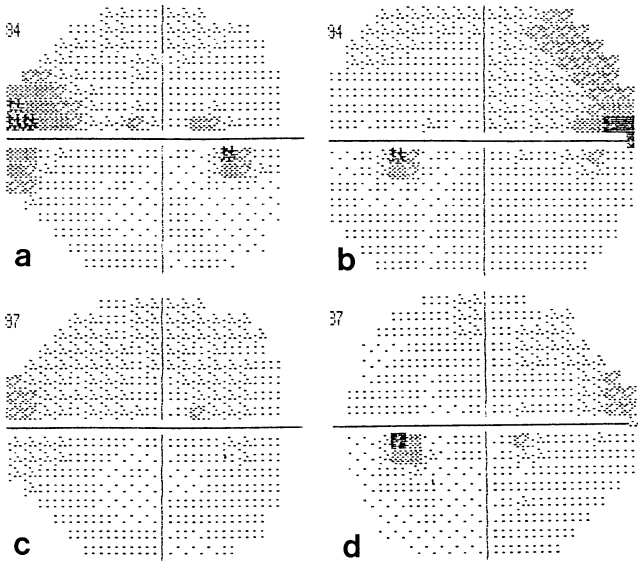

FIGURE 1. Visual fields of the patient with normal-pressure glaucoma. Right (a) and left (b) visual fields before treatment with methotrexate. Visual fields of the right (c) and left eyes (d) in the second year of the treatment.

cryoglobulin titers. She had a family history of glaucoma. Upon ocular examination, her corrected visual acuity was BE: 20/15. Slit-lamp examination was not remarkable, and the iridocorneal angles were open gonioscopically. Diurnal curves of intraocular pressures in both eyes were always in the low teens. She had bilateral glaucomatous cupping with inferior notching (RE: 0.7 and LE: 0.65) and visual field abnormalities. The patient received methotrexate for her connective tissue disease with a 7.5-mg to 12.5-mg dose each week. This treatment continued during last 3 years, except for a few periods of up to 3 months when she had infection and fever. During the treatment period, visual fields obtained with a perimeter showed improvement (Figure 1). Optic disk examination during the period without treatment disclosed new, bilateral splinter hemorrhages on the optic disks. As a part of ongoing studies on autoimmune mechanisms for glaucomatous optic neuropathy, sera from this patient were sequentially analyzed for immunoreactivity using Western blot analysis. We noticed a coincidental remission in serum immunoreactivities against retinal proteins with methotrexate treatment, assessed by a decrease in the density of the immunoreactive bands seen on Western blot analysis (Figure 2).

Methotrexate has been shown to effectively treat autoimmune diseases because it reduces both mononuclear cell proliferation and antibody synthesis.[4] Our observations show a coincident diminution of antibodies against retinal antigens during methotrexate treatment. Conversely, there is some evidence that after discontinuation of methotrexate treatment, systemic vasculitis may be observed in patients with rheumatic diseases.[5] Optic disk hemorrhages observed during the periods after cessation of the treatment may have a similar mechanism. To better understand

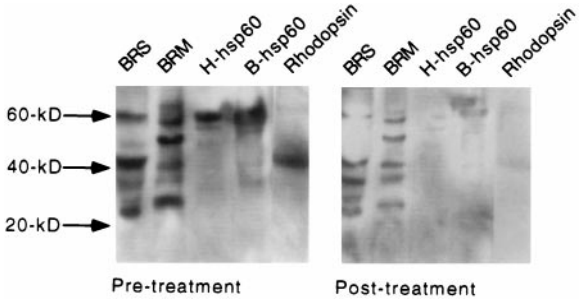

FIGURE 2. Western blot analysis of the patient with normal-pressure glaucoma. Before treatment with methotrexate, patient serum recognized retinal proteins, including heat shock protein 60-kD (hsp60) and rhodopsin. At the end of the 3-year period of treatment, immunoreactive bands faded. Each lane contains patient serum (1:1000) against bovine retinal supernatant (BRS), bovine retinal membrane (BRM) (15 μg/lane), purified human (H-hsp60) and bacterial hsp60 (B-hsp60), and rhodopsin (1 μg/lane) as labeled. Secondary antibody (goat anti-human IgG) dilution, 1:2000.

the clinical importance of these observations, the autoimmune mechanisms and effective treatment models of glaucomatous optic neuropathy must be studied further.

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Multiple Evanescent White Dot Syndrome in Older Patients

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Accepted for publication Dec 4, 1998.

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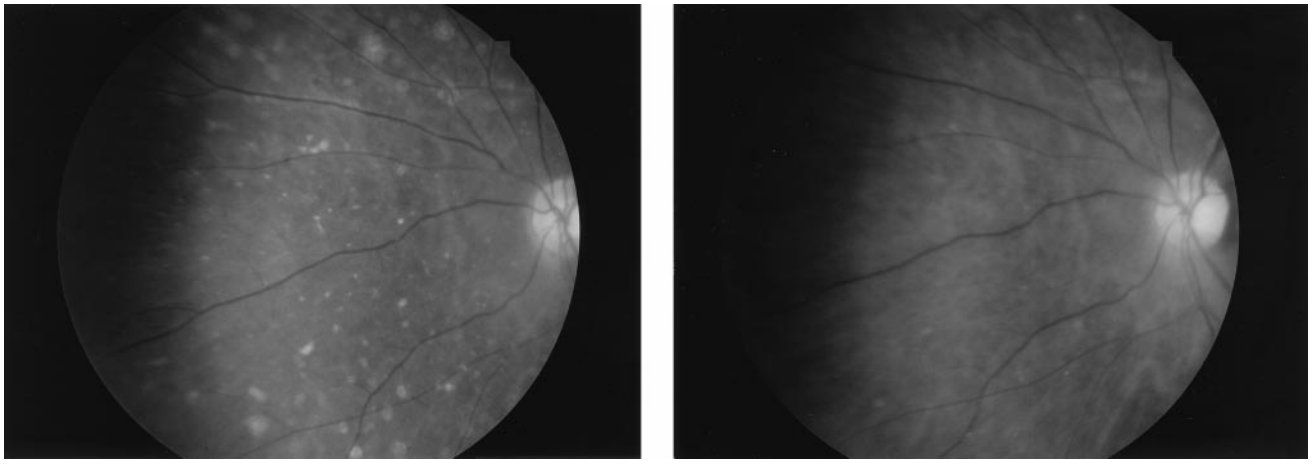

FIGURE 1. (Left) Fundus photograph of the left eye showing some of the numerous white dots in the nasal retina, posterior to the equator. (Right) Fundus photograph of the left eye taken 2 months later shows resolution of most of the white dots in the nasal retina, posterior to the equator.

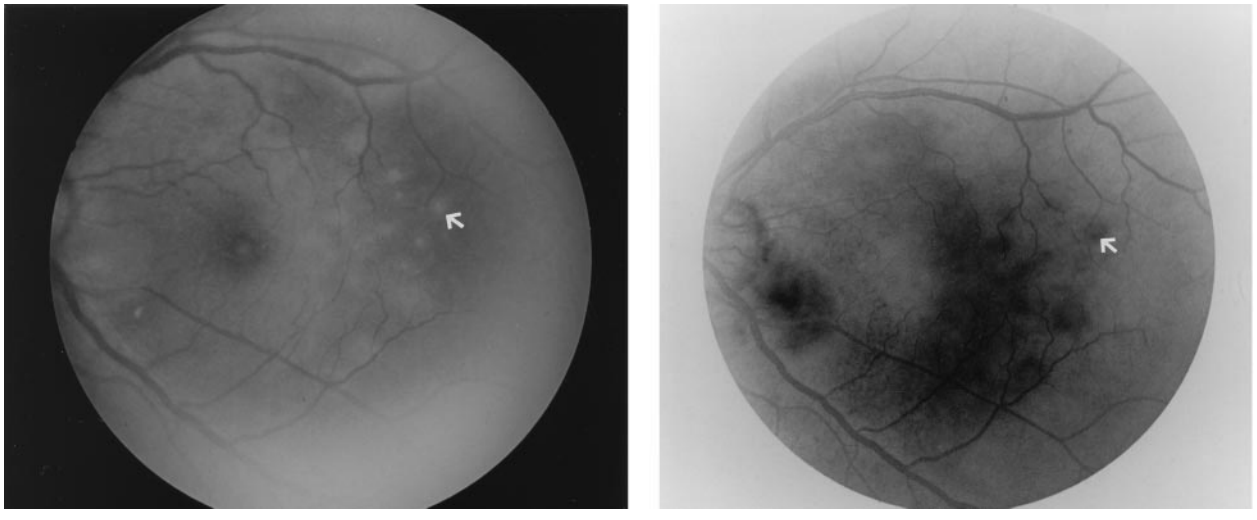

FIGURE 2. (Left) Fundus photograph of the left eye showing multifocal white, temporal, retinal lesions (arrow) with foveal granularity. (Right) Fluorescein angiogram showing hyperfluorescence of the retinal lesions in the temporal macula and deep diffuse leakage in inferonasal macula. Note the ring-shaped pattern of hyperfluorescence typical of multiple evanescent white dot syndrome. The fluorescent ring corresponding to the lesion in the left panel is identified by an arrow.

PURPOSE: To report two patients in their seventh decade who exhibited findings consistent with multiple evanescent white dot syndrome.

METHODS: Case reports of two patients referred for evaluation of decreased vision, visual field loss, and retinal white spots.

RESULTS: A 60-year-old man and a 67-year-old woman had photopsia, visual field loss, and decreased central visual acuity. Examination disclosed numerous white retinal spots, ranging from 50 to 400 μm, with eventual foveal granularity. Visual field testing showed an enlarged blind spot and peripheral field defects. Fluorescein angiography, electroretinography, and electrooculography results were consistent with multiple evanescent white dot syndrome. Eventually, the retinal lesions resolved in both patients and baseline visual acuity was recovered.

CONCLUSION: A diagnosis of multiple evanescent white dot syndrome should be considered in patients with retinal findings typical of multiple evanescent white dot syndrome, regardless of age. **(Am J Ophthalmol 1999; 127:725–728.**

MULTIPLE EVANESCENT WHITE DOT SYNDROME IS A multifocal inflammatory retinal disease that typically affects young women. The average age for previously

reported cases was 26 years, with a range of 14 to 47 years.[1–4] We report two patients, aged 60 and 67 years, who are older than the typical patient with multiple evanescent white dot syndrome.

• CASE 1: A 60-year-old Filipino man was initially examined with a 1-week history of temporal photopsia and peripheral visual field loss of his left eye. Symptoms began after he was struck by an automobile while crossing a street. Examination in the emergency room disclosed a left upper eyebrow laceration and superficial abrasions, but there was no internal injury or chest compression. He sustained no direct ocular trauma. Ocular history was negative.

Ophthalmologic examination showed initial best-corrected visual acuity of RE: 20/30 and LE: 20/40. Slit-lamp examination disclosed mild nuclear sclerosis. Ophthalmoscopic examination demonstrated numerous deep, flat, white retinal lesions, extending from the optic disk to the nasal, superior, and inferior equatorial regions. The lesions ranged in size from one-eighth to one-quarter disk diameter (Figure 1, left). There was no vitreous cell, subretinal fluid, retinal, or vitreous hemorrhage; hard exudate; Berlin edema; retinal tear; or retinal detachment. Visual field testing showed an enlarged blind spot with a temporal field defect.

Eleven days later, the patient's visual acuity measured LE: 20/60; the retinal lesions were unchanged. Five weeks after presentation, his visual acuity was LE: 20/40, photopsia were less frequent, and the temporal field loss was less dense. Examination disclosed fewer and less distinct retinal lesions. After 2 more weeks, his visual acuity improved to LE: 20/20, and almost all of the retinal lesions were resolved (Figure 1, right). Mild retinal pigment epithelial granularity was still present in the macular area. One month later, the retinal lesions, photopsia, and visual field defect had completely resolved. Follow-up examinations during the subsequent 18 months disclosed a visual acuity of LE: 20/20, with no recurrence of the lesions and no retinal tear or detachment.

• CASE 2: A 67-year-old white woman had sudden onset of blurred vision, photopsia, and a dark temporal shadow after an upper respiratory infection (rhinorrhea, low-grade fever, and chills). Her medical history was notable for hypertension and hypercholesterolemia. Initial ophthalmologic evaluation showed a best-corrected visual acuity of RE: 20/20 and LE: 20/200, with an unremarkable slit-lamp examination. Ophthalmoscopy disclosed multifocal deep, flat, white 50-μm to 300-μm retinal lesions in the macula (Figure 2, left) and midperiphery of the left eye. The fovea had a yellow granular appearance (Figure 2, left). Retinal evaluation of the right eye was normal. Goldmann visual field testing disclosed an enlarged blind spot with a cecocentral scotoma. Fluorescein angiography disclosed ring-shaped hyperfluorescent retinal lesions in the early phases with late staining (Figure 2, right). There was no disk staining. Laboratory evaluation included a positive antinuclear antibody titer of 1:64 (normal, less than 1:40), complete blood cell count, erythrocyte sedimentation rate, and chest x-ray, and negative tests for rheumatoid factor, fluorescent treponemal antibody, and purified protein derivative.

The patient's visual acuity in the left eye initially deteriorated to counting fingers during the first week. A 3-week course of oral corticosteroids was given. One month after initial presentation, her visual acuity improved to LE: 20/80. There was persistent photopsia, an afferent pupillary defect, a smaller blind spot, and resolution of most of the white retinal lesions. The left eye had a marked reduction in amplitudes of scotopic, photopic, and maximal response electroretinogram and the right eye had a normal electroretinogram response. The a-wave was severely attenuated in the left eye. The electrooculogram was abnormal in the left eye (Arden ratio: RE: 2.15 and LE: 1.40; normal: >1.85).

Four months after initial presentation, the patient's visual acuity had improved to LE: 20/30. The retinal lesions had resolved, and only mild residual foveal granularity remained. Her visual acuity stabilized at LE: 20/40 10 months after initial presentation.

Multiple evanescent white dot syndrome is a self-limited, usually unilateral disease typically found in young adults.[1] In a previous series, the patients ranged in age from the second to fourth decades.[1–4] The youngest patient reported with multiple evanescent white dot syndrome is a 14-year-old girl.[4] To our knowledge, our two patients represent the oldest patients reported with multiple evanescent white dot syndrome.

In patients with multiple evanescent white dot syndrome, visual acuity is usually mildly decreased to between 20/50 and 20/60. Our male patient's visual acuity was more typical of the degree of visual acuity loss in multiple evanescent white dot syndrome than our female patient's visual acuity. Both patients exhibited the typical yellow-white retinal lesions, ranging in size from one-third to one-fourth disk diameter and fading rapidly. Both patients showed the typical resolution of lesions within 3 months, with recovery of visual acuity to baseline levels. Both patients developed macular granularity but otherwise had no retinal scars or permanent visual field defects. Thus, the appearance of the white retinal lesions with typical rapid resolution, prominent photopsia, sudden vision loss with rapid recovery, and visual field abnormalities are all findings consistent with multiple evanescent white dot syndrome.

In addition, the electroretinogram findings and fluorescein angiographic findings of our female patient are consistent with multiple evanescent white dot syndrome.[1,3] The decreased electroretinogram amplitudes, marked attenuation of the a-wave, and abnormal electrooculogram are typical of multiple evanescent white dot syndrome, which is believed to be a diffuse outer retinal inflammatory disease. Our male patient did not have these studies performed because the clinical presentation was typical of

multiple evanescent white dot syndrome, and he was reluctant to have any ancillary tests performed.

Multiple evanescent white dot syndrome has been reported in men less commonly than in women.[2] There does not appear to be any racial predilection; multiple evanescent white dot syndrome has been reported in Asian patients.[3] Thus our Filipino male patient, although not a typical patient with multiple evanescent white dot syndrome, is not an exception.

Acute idiopathic blind spot enlargement has been reported to occur in patients as old as 57 years. Some authors have characterized acute idiopathic blind spot enlargement as a later phase of multiple evanescent white dot syndrome, whereas others have thought that multiple evanescent white dot syndrome represents one of several diseases that comprise acute idiopathic blind spot enlargement.[5] Our patients demonstrate the presence of multiple evanescent white dot syndrome and enlargement of the blind spot.

Our patients were atypical because of their advanced age compared with the typical age group affected by multiple evanescent white dot syndrome. They demonstrate that multiple evanescent white dot syndrome may occur in older patients and that patients do not always fit the rigid demographics of diseases. Thus, one should consider multiple evanescent white dot syndrome in the differential diagnosis of patients, regardless of age, who present with decreased visual acuity, photopsia, typical retinal white lesions, and visual field abnormalities.

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Laser Pointer Maculopathy

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Accepted for publication Nov 30, 1998.

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PURPOSE: To report a case of macular damage from a laser pointer.

METHOD: Case report. A 19-year-old woman had an acute reduction of visual acuity in the right eye after deliberately staring into a commercial class 2 laser pointer for approximately 10 seconds.

RESULTS: The patient's best-corrected visual acuity was RE: 20/40, and she had two small pericentral scotomata, as well as a hypopigmented ring-shaped lesion in the fovea. Within 8 weeks, her visual acuity improved to 20/20 and visual field returned to normal, but a subjective relative decrease in brightness of objects viewed by the right eye was apparent. Retinal pigment epithelial abnormality persisted.

CONCLUSIONS: Commercial laser pointers, commonly used for teaching and entertainment purposes, may cause notable macular damage if abused. Morphologically, this may manifest as foveal retinal pigment epithelial disturbance. (Am J Ophthalmol 1999;127:728–729.

A 19-YEAR-OLD WOMAN WITH NO PREVIOUS OCULAR history presented to our clinic with a 2-week history of decreased visual acuity in her right eye. She had directed a laser pointer into that eye from a distance of about 10 cm and stared at the light for approximately 10 seconds. Examination disclosed best-corrected visual acuities of RE: 20/40 and LE: 20/20. Anterior segments were normal in both eyes. Fundus examination disclosed a hypopigmented ring-shaped area around the right fovea, with attenuation of the foveal reflex; the left eye was normal. A red-free photograph demonstrated numerous hypopigmented foveal dots (Figure). Fluorescein angiogram showed a mild and irregular foveal hyperfluorescence in the right eye, compatible with a retinal pigment epithelial defect, and a normal appearance of the left eye. A computerized visual field, 10-2 threshold test (Humphrey Automated Perimeter; Humphrey Instruments, San Leanardo, California), showed two small pericentral scotomata in the right eye and a normal field in the left eye.

The laser pointer was labeled as class 2 laser diode, with a maximum output of less than 1 mW and a wavelength of 670 nm. We followed up with the patient and noted gradual improvement of her visual acuity to 20/20 within 8 weeks. The visual field returned to normal; however, after 3 months, the patient still noticed a relative reduction in the brightness of objects when they were viewed by the right eye. The foveal retinal pigment epithelial changes persisted at her last examination, 3 months after the acute event.

Laser pointers have been widely used in recent years in making presentations and are also popular as toys among adolescents. Most are class 2 or 3a red lasers, with outputs of up to 5 mW and wavelengths between 632.8 and 670.0 nm.[1] Their potential to cause retinal phototoxic damage is