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Outer retinal layer toxoplasmosis* **

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Abstract. It is widely held that ocular toxoplasmosis (1) involves inner retinal layers and (2) shows marked vitreous cellular reaction. This article reports on punctate outer retinal layer toxoplasmosis, a subset of ocular toxoplasmosis characterized by grey-white lesions of deep retina and retinal pigment epithelium, and associated with little or no overlying vitreous reaction. Acute lesions may resolve and become fine punctate white dots. Recognition of this uncommon presentation of toxoplasmosis is important, since this may allow for potentially efficacious therapy.

Introduction

Acute ocular toxoplasmosis is generally understood to be a process which involves inner retinal layers and manifests prominent overlying vitreous cellular reaction. However, in 1969 several cases of ocular toxoplasmosis were reported (Friedman and Knox 1969), which primarily affected outer retinal layers and showed little if any overlying vitreous reaction. Since that time further reports on this entity have not appeared. The purpose of this paper is to report additional cases and, thus, further to define punctate outer retinal toxoplasmosis, a subset of ocular toxoplasmosis characterized by multifocal punctate outer retinal lesions with little or no overlying vitreous involvement.

Case reports

Case 1

A 6.5-year-old white female was found to have decreased vision on a routine school screening examination. During an uncomplicated pregnancy, the patient's mother had begun eating raw hamburger meat. The family history was unremarkable.

Initial examination showed visual acuity of 20/20 in the right eye and 20/50 in the left. The anterior segments were unremarkable. The vitreous of the left eye showed fine posterior vitreous cells. Fundus examination in the right eye showed punctate white spots, approximately 20–75 μm in size, located at the level of the deep retina, and retinal pigment epithelium in the macular region (Fig. 1). Fluorescein angiography showed little in the way of hyperfluorescence (Fig. 1), which suggests that these white lesions may be at the level of the deep retina rather than of the retinal pigment epithelium.

The left eye showed a pigmented macular scar (Fig. 2). There were punctate deep white spots similar to those in the right eye, as well as active grey-white lesions involving the deep retina and underlying retinal pigment epithelium. There was no discernible overlying vitreous cellular reaction. At angiography the acute lesions blocked fluorescence early and stained late (Fig. 2).

Over the ensuing months the acute lesions in the left eye resolved to become punctate white spots (Fig. 3). Additional punctate white spots developed in several new areas and were similar to the ones seen in the right eye at the time of initial examination.

Six months after initial presentation, the right eye developed an acute lesion similar to that previously present in the left. This acute lesion involved the outer retinal layer and there was no overlying vitreous cellular reaction. After several months, the acute lesion in the right eye had healed, leaving a slightly pigmented scar.

Laboratory studies on this patient gave negative results except for positive toxoplasmosis hemoglutenin titers at 1:1024 on two separate occasions 6 months apart.

Case 2

A 17-year-old white female presented with metamorphosia of 1-month duration in the right eye. Visual acuity was 20/80 in the right eye and 20/20 in the left. The anterior segments were normal, and there were no vitreous cells. Funduscopy of the right eye showed a white retinal lesion in the macula, which appeared deep, at the level of the retinal pigment epithelium or outer retina. This resolved spontaneously, and acuity in the right eye improved to 20/20. There were several further episodes, which all resolved, and 6 years later acuity was 20/200 OD, with fine punctate spots at the level of the retinal pigment epithelium in the right macula. A further 1.5 years later the symptoms recurred. There was now active deep retinitis in the macula, with overlying vitreous cellular reaction. The toxoplasmosis titer was positive. Eight months later a new area of fundus activity was seen in satellite fashion in the left eye. Eventually these lesions resolved and became totally inactive. Ten years after initial presentation the right eye showed a chor-

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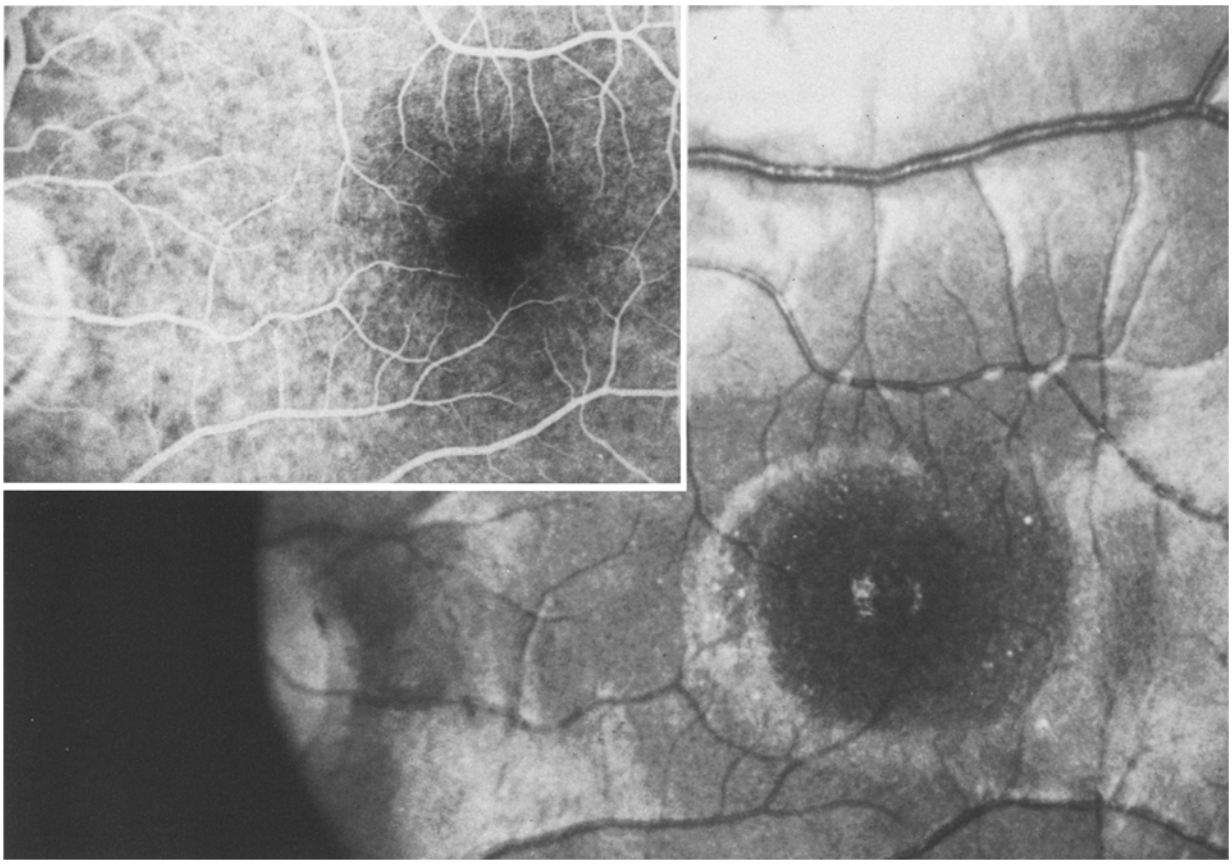

Fig. 1. Case 1. Black and white Kodachrome print of macula (right eye) shows punctate white spots at level of deep retina/retinal pigment epithelium. Fluorescein angiographic frame shown in *insert*

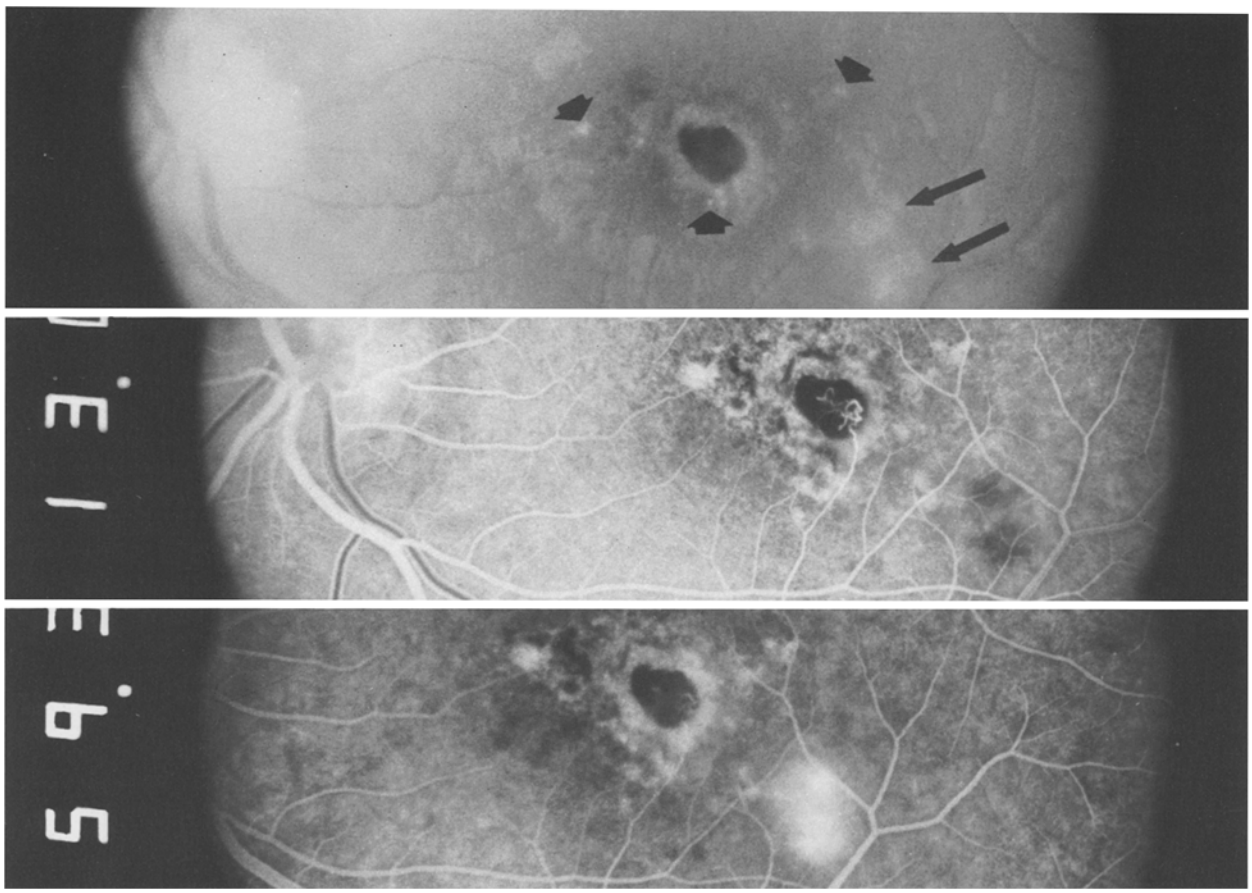

Fig. 2. Case 1. Top, black and white Kodachrome print, left eye. Macula shows punctate focal white spots (*short, wide arrows*) and active deep retina/retinal pigment epithelium lesions (*long, thin arrows*). Fluorescein angiogram at 13 s (*middle*) and 659 (*bottom*) shows active lesions (area corresponding to long, thin arrows in top part of figure)

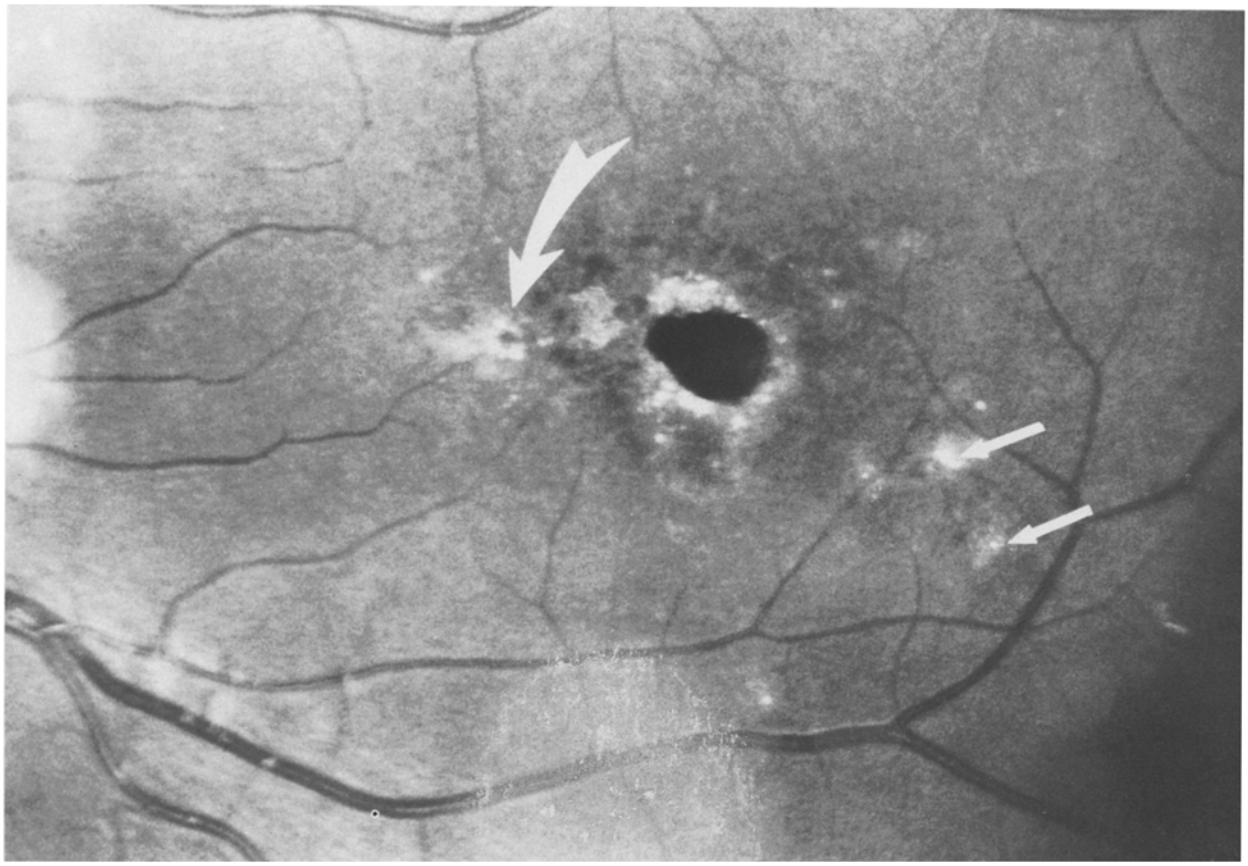

Fig. 3. Case 1. Active lesions in the left eye have resolved 6 months after initial presentation, leaving focal punctate deep white spots

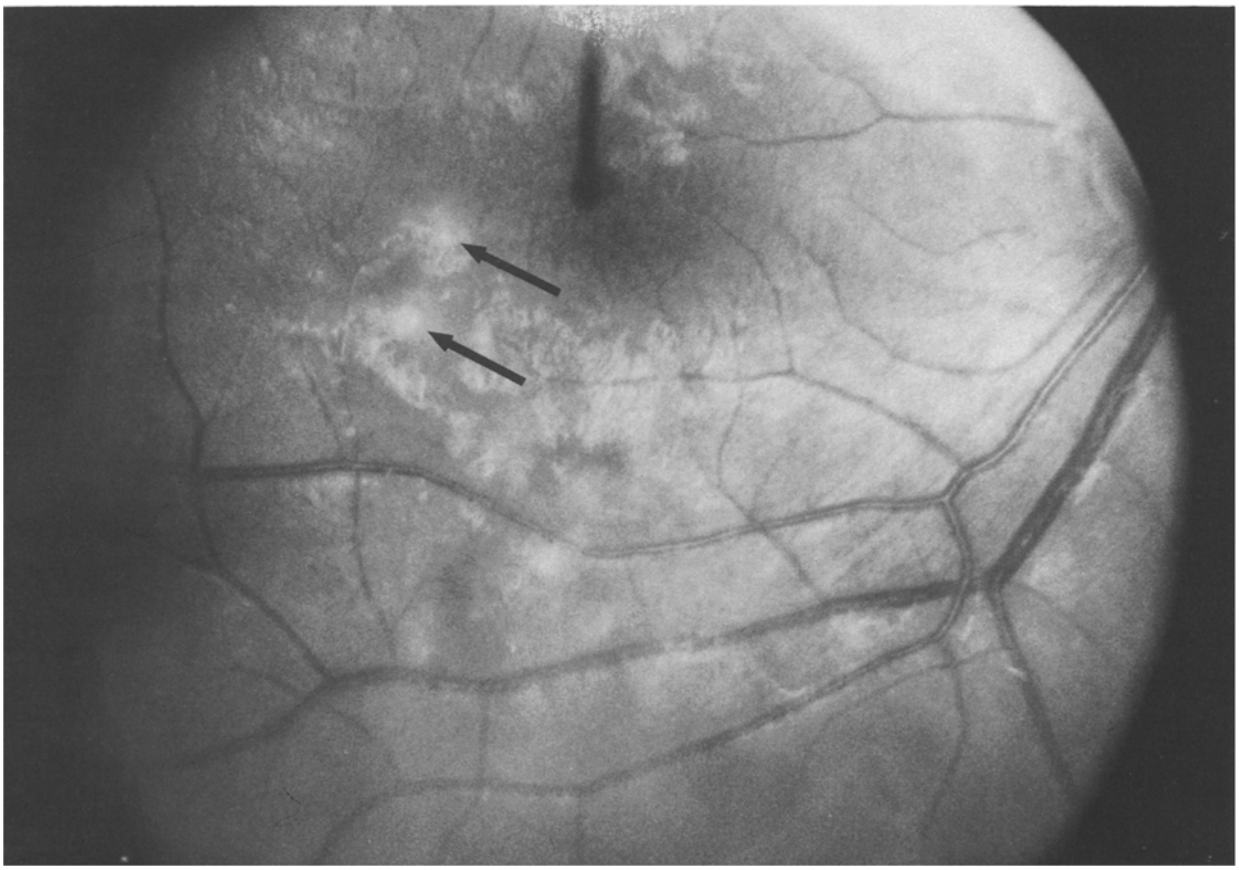

Fig. 4. Case 3. Active deep retinitis (*arrows*), right eye

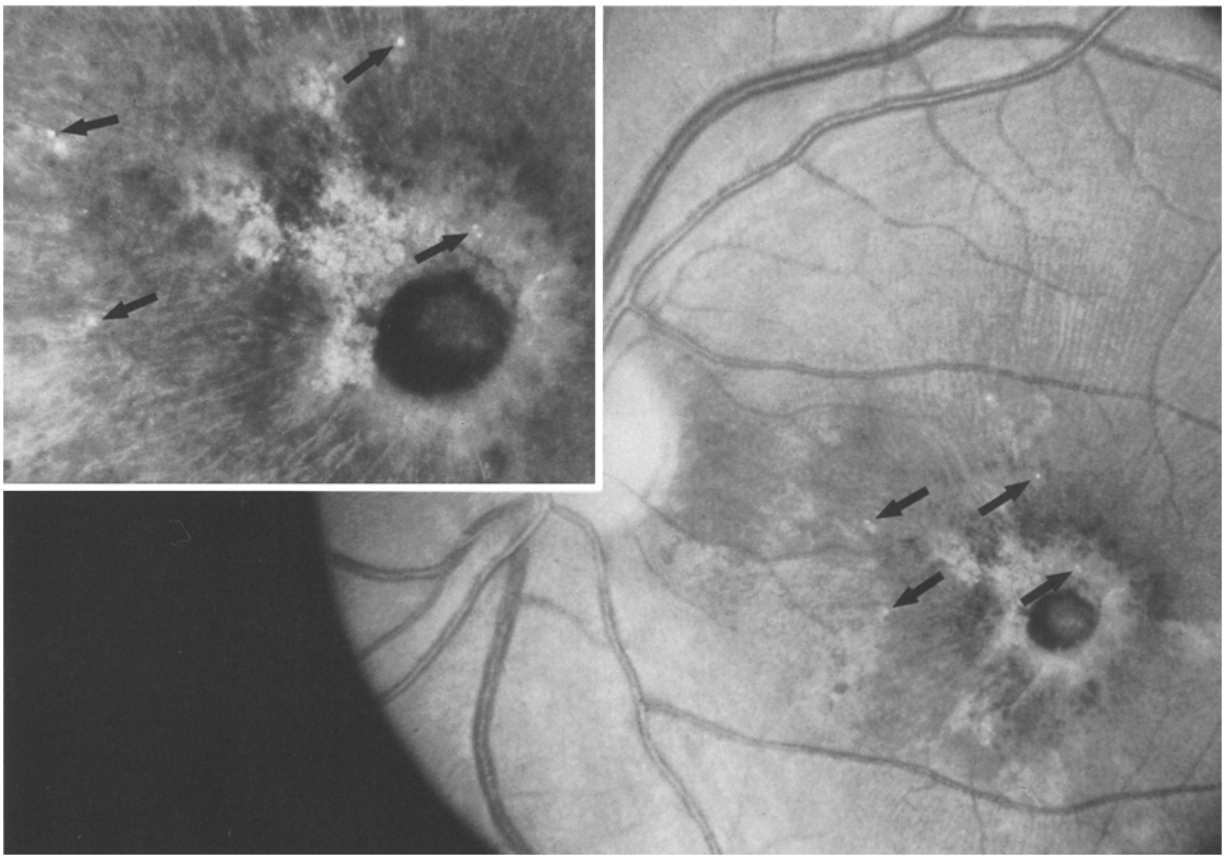

Fig. 5. Case 3. Punctate deep white spots (*arrows*), left eye. *Insert* at greater magnification

ioretinal scar, with satellite changes, similar to that which can occur with classic ocular toxoplasmosis.

Case 3

A 3-year-old asymptomatic girl failed a vision screening test. Her parents kept cats as pets, but otherwise the past history was unremarkable. Visual acuity was 20/25 in the right eye, and 20/200 in the left. Biomicroscopic examination of the anterior segment gave normal results. There were no vitreous cells. Funduscopic examination of the right eye revealed an active creamy deep retinitis inferior temporal to the foven (Fig. 4), with little if any overlying vitreous cellular reaction. The left eye showed an old pigmented chorioretinal scar in the macula, with discrete and deep punctate white spots (Fig. 5), just as in case 1. A quantitative immunofluorescent toxoplasmosis titer for IgG was positive at greater than 1:400. Systemic antitoxoplasmosis treatment was recommended, but declined by the patient's family.

Follow-up 1 month later showed resolution of a part of the active perifoveal lesion. However, 2 months afterward, there was prominent creamy white satellite recurrence of retinitis with only mild overlying vitreous cellular reaction. A month later, this was beginning to resolve. The patient never returned for follow-up.

Discussion

The diagnosis of ocular toxoplasmosis is, of necessity, presumptive since clinically one cannot isolate organisms from the eye. In practice, the diagnosis is based on clinical appearance substantiated by serological evidence of exposure to the parasite. This paper describes young patients with acute, recurrent, multifocal retinal lesions followed by scarring. Lesions occurred adjacent to each other in satellite fashion, and were associated with serological evidence of exposure to *Toxoplasma gondii*. These findings are sufficient to make a presumptive diagnosis of ocular toxoplasmosis (Owens et al. 1979; O'Connor 1975).

In the late 1960s a few unusual cases were described of what was felt to be toxoplasmosis characterized by initial involvement at the outer retinal layers, little if any vitreous cellular reaction, and (in these cases) serous elevation of the macula (Gass 1968; Friedman et al. 1969). Since that time, no new cases have been described in the literature, and outer retinal layer toxoplasmosis is not widely recognized.

This report describes additional clinical manifestations of the punctate outer retinal toxoplasmosis syndrome. This entity is a subset of the larger clinical syndrome of ocular toxoplasmosis. However, unlike classic ocular toxoplasmosis, the acute manifestations of outer layer toxoplasmosis are characterized by multifocal grey-white lesions, which appear at the level of the deep retina and retinal pigment epithelium. The lesions resolve slowly over time, recur in satellite fashion in adjacent areas, and are associated with only minimal evidence of vitreous reaction. The differential diagnosis of the acute lesion of outer retinal toxoplasmosis is that of similar deep grey-white changes wich may occur in acute posterior multifocal placoid pigment epitheliopathy, serpiginous choroiditis, and diffuse unilateral subacute neuroretinitis.

Resolution of the acute deep grey-white ocular lesions of outer layer toxoplasmosis is by healing to become punctate white spots. This is illustrated in case 1, where small white spots approximately 25–75 μm in size developed after the resolution of the acute lesions. Similar white lesions were present in the right eye of case 1 when the patient was first seen (Fig. 1). In case 3 these glistening punctate white spots were seen at presentation in the left eye (Fig. 5). It is not known what these punctate changes represent. The fact that these spots were not associated with angiographic changes in the right eye of case 1 suggests that they may represent focal outer-retinal gliotic scars. Though unlikely, it is also possible that they represent an encysted tissue form of *Toxoplasma*. Tissue cysts of *Toxoplasma* may be the same size as these lesions (Schlaegel 1968). The nature of these punctate changes will not be established until specimens can be examined histologically. Case 2 demonstrates that acute deep lesions may also eventually resolve by forming the more familiar and characteristic toxoplasmosis chorioretinal scar with satellite changes.

This paper confirms the findings of Freidmann and Knox (1969) that there exists a subset of ocular toxoplasmosis which initially and primarily affects the outer retinal layers, and has only minimal overlying vitreous reaction. Awareness of this presentation is important, since there is some evidence that treatment of toxoplasmosis may be effective.

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