Ocular Toxoplasmosis

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Infestation with the protozoan parasite, **Toxoplasma gondii** is commonly encountered in large segments of the population of both the temperate and tropical zones of the world. Judging from antibody tests on blood bank samples from ostensibly normal individuals, about one-third of the population of the San Francisco metropolitan area is currently infected. It is clear that most of these infections are asymptomatic; and in general, it can be said that unless Toxoplasma invades the eye or the brain, such infections remain asymptomatic.

Toxoplasmosis occasionally produces a febrile, lymphadenopathic disease in adults that closely resembles infectious mononucleosis. Small epidemics of this kind of disease have been described in Denmark and the United States , and isolations of Toxoplasma have been obtained from the blood and lymph nodes of the affected individuals; yet very few of these have developed ocular lesions. This has led Perkins and others to assume that virtually all ocular toxoplasmosis represents an early or late exacerbation of congenital toxoplasmosis. This suggestion remains largely unconfirmed, because most patients with toxoplasmic eye disease have no other stigmata of infection. Furthermore, serum specimens from the affected patients are generally not available from any time **prior** to the onset of eye symptoms. Thus, we lack the data that would show when a given patient converted from a negative test to a positive test. Once a patient has a positive dye-test, the serologic reaction usually remains positive for life, although the titer may decline considerably. Patients with congenital toxoplasmosis would therefore continue to be dye-test positive into the second and third decades of life when most toxoplasmic eye disease is first noted.

Only a few patients have been noted to convert from sero-negativity to sero-positivity during an acute illness that produced both retinochoroiditis and the classical signs of systemic toxoplasmosis. One case described by Hales and another described by Saari may fit into this category. Recently, a patient described by Michelson, et al. showed that isolated retinal lesions could arise **de novo** in a patient with acute acquired toxoplasmosis. This patient acquired the febrile, adenopathic form of the disease in adult life, and Toxoplasma organisms were isolated from one of her swollen lymph nodes.

Necrosis is the hallmark of the retinal lesion. Whether this is produced by the multiplicative activities of the parasite alone, or whether the lytic action of macrophages and lymphocytes is of equal importance remains to be seen. It is most likely that a combination of the two produces the zonal granulomas so characteristic of the disease. Recurrences are said by Frenkel and others to be the result of hypersensitivity to the organism. According to them, relatively few of the organisms released by the breakdown of Toxoplasma cysts in the retina invade new, previously uninvolved retinal cells.

DIAGNOSIS

The diagnosis of ocular toxoplasmosis is based on (1) the observation of a focal necrotizing retinochoroiditis; (2) the presence of serum antibodies to Toxoplasma (at any titer); and (3) the exclusion of other possible causes of necrotizing retinochoroiditis.

Congenital toxoplasmosis is usually bilateral in its ocular manifestations. A skull X-ray may show the presence of paraventricular calcifications.

In cases of adult retinochoroiditis where clinical picture is not clear or where there is serologic evidence of two or more possible etiologies, examination of the aqueous humor for antibodies may prove useful. If there is more antibody per unit of gamma globulin in the aqueous humor than in the serum, the patient can be assumed to be forming antibodies intraocularly. According to Desmonts, this is diagnostic for ocular toxoplasmosis.

TREATMENT

It is generally agreed that pyrimethamine and sulfonamides, or other antimicrobials such as clindamycin, should be administered for acute, newly acquired lesions, particularly if they threaten structures that are important for vision. Many practitioners treat recurrent retinal lesions with corticosteroids alone, feeling that the recurrences are merely manifestations of hypersensitivity. 'It is my feeling that proliferating organisms may be involved in all recurrent lesions, and that it is never safe to treat recurrent lesions

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with steroids alone. The patient should always be treated concomitantly with at least one antimicrobial agent known to be effective against the parasite. Small lesions that are nasal to the disc or lesions that occupy the retinal periphery are best treated by observation alone. Lesions that appear to be extending into the macula or papillo-macular bundle may require anti-microbial therapy plus corticosteroids. It is felt that injections of repository forms of corticosteroids into the subconjunctival space may be dangerous, because the multiplication of the parasite may be enhanced under these conditions.

TREATMENT SCHEDULE

Pyrimethamine, 75 mg per day for the first two days.
Pyrimethamine, 25 mg per day thereafter for 6 weeks.
Triple Sulfas, 2 g as a loading dose.
Triple Sulfas, 1.5 g four times a day thereafter for 6 weeks.
Folinic acid, 3 mg I.M. or by mouth twice a week.
Sodium bicarbonate, 1 tsp with meals three times a day.

or

Clindamycin 300 mg every 6 hours for 4 weeks (not FDA-approved).
Prednisone 80-100 mg per day for first week. Taper thereafter.
Prednisolone acetate drops (1%), p.r.n. for anterior uveitis.
Atropine sulfate 1% drops as needed to dilate the pupil.
Weekly blood count for W.B.C. and platelets while patient is on pyrimethamine-sulfonamide therapy.

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Infectious Causes of Uveitis

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TUBERCULOSIS

Documented cases of tuberculous uveitis are now rare. Within the past 10 years, only 3 histologically documented cases of tuberculous endophthalmitis have been reported in the English literature.

Woods reported an incidence of 1.5% ocular tuberculosis among patients hospitalized for pulmonary tuberculosis. Darrel (1) reported acute tuberculous panophthalmitis in a 73 yr. old patient with no other signs of active tuberculosis.

Clinical Features

I. Principal Symptoms

(1) Blurred vision, (2) Pain, (3) Redness, (4) Photophobia, (5) Floaters.

II. Cardinal Signs

(1) Conjunctival hyperemia and ciliary flush, (2) Mutton-fat keratic precipitates, (3) Cells and flare, (4) Dilated iris vessels, (5) Synechia formation, extensive, (6) Iris nodules (Koeppe or Busacca type), (7) Dense vitreous opacities: clumps of cells, strands, (8) Single or multiple choroidal nodules, may progress to large chorio-retinal granuloma invading the sclera or vitreous.

III. Diagnostic Studies

(1) Chest x-rays, (2) Tuberculin skin

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