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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test, (3) 24 hr. urine for acid-fast bacilli, (4) Iris biopsy, (5) Lymph node biopsy

IV. Treatment

(1) Streptomycin 1.0 Gm. 3 times a week for 4 weeks, (2) Isoniazid 100 mg. t.i.d. for 1 year, (3) Vitamin B6 10 mg. b.i.d. for 1 year, (4) P.A.S. 3 Grams t.i.d. for 6 mos. — 1 year, (5) Mydriatics (Cyclogyl, Neosynephrine, Atropine as needed for pain), (6) Steroid drops, p.r.n.

OTHER BACTERIAL CAUSES OF UVEITIS

As a complication of septicemia:

Staphylococcus aureus, Streptococcus sp., Leptospira icterohemorrhagic, Brucella, Neisseria gonorrheae, Salmonella typhi, Escherichia coli, Pneumococcus.

EXOGENOUS INFECTIONS

Listeria monocytogenes (2), Mycobacterium leprae, Neisseria gonorrheae, Mycoplasma hominis.

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THE OPPORTUNISTIC INFECTIONS: CANDIDA AND CYTOMEGALOVIRUS

Certain infectious agents that have been present in man's environment for centuries have recently come to the fore as causes of posterior uveitis. The appearance of these agents as prominent etiologic factors in uveitis can be attributed to man's manipulation of his own environment or to the greater average duration of malignant diseases, particularly of the hematopoietic system.

In case of *Candida* infections of the retina, for example, most cases can be traced to the use of prolonged intravenous hyperalimentation therapy administered through indwelling venous catheters. Man's skin is almost universally infested with *Candida*, yet the administration of solutions containing large amounts of amino acids through a catheter implaced in the skin is necessary to seed the blood stream with the fungus. Hyperalimentation as a form of post-surgical treatment has only become available during the last ten years. The use of improperly sterilized injection equipament on the part of narcotic addicts has also been shown to be responsible for a number of disastrous cases of *Candida* endophthalmitis.

The initial complaints of the patients are of floating spots and diminished visual acuity. Cotton-like, whitish lesions are seen on the retina.(1) Within a few days cellular aggregates may be seen in the vitreous; these often take the form of "puff balls" which contain the fungal agent as well as inflammatory cells. A careful history of the habits and health status of the patient is one of the most important clues as to the possible nature of the disease. One must suspect the disease in order to make a diagnosis, and early diagnosis is the key to successful treatment.

Isolation of the yeast from the tip of an in-dwelling catheter has helped to diagnose the lesion in a number of cases (2). Blood cultures are sometimes helpful if there is a persistent fungemia. Direct diagnosis has also been made by aspiration of the vitreous through a pars plana incision.

Treatment consists of the use of Amphotericin B by slow intravenous drip (in 5% dextrose in water) beginning with a dosage of 1 mg (to determine idiosyncrasy), and increasing the daily dosage by increments of 5 mg until signs of toxicity can be detected. These consist of BUN elevations, serum creatinine elevations, and decreases in the hemoglobin to pathologic levels. On the average, a total dosage of 1500 mg is aimed for. A new drug, 5-fluorocytosine holds promise for the treatment of *Candida* endophthalmitis. It is generally given in doses of 4-6 grams per day and may be administered concomitantly with Amphotericin B. About half of the known strains of *Candida* are resistant to it, however, and a large group of *Candida* strains have become resistant to it during therapy.

Once the *Candida* infection has left the surface of the retina and has invaded the vitreous, the prognosis for a complete cure diminished considerably. Vitrectomy has been advocated by some ophthalmologists under these circumstances.

Cytomegalovirus infections of the retina have been known for many years. The disease is seen principally in its congenital form where it may be confused with toxoplasmosis, particularly in neonates. In recent years it has been seen more commonly among adults, particularly in individuals receiving immunosuppressive therapy to retard the rejection of organ transplants (4, 5). Its lesions consist of exudative retinitis which often produces hemorrhage. The lesions tend to be less focal than those of toxoplasmosis. Like toxoplasmosis it often produces extensive retinal disease in patients who are immunosuppressed by overwhelming neoplastic processes. It has been seen in cases of Hodgkin's disease, multiple myeloma, and chronic lymphatic leukemia.

Active disease is usually accompanied by excretion of virus in the urine and by rising complement fixation titers in the serum. Treatment regimens have included the use of adenine arabinoside intravenously and the use of transfer factor by subcutaneous injection (6).

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SYPHILIS

Although documented cases of iritis are known to be associated with secondary lues, syphilis has not often been implicated in acute multifocal choroiditis. The "salt-and-pepper" fundus of congenital lues indicates that such a multifocal choroiditis must take place during fetal or early infantile life, and by the time such patients come to medical attention, the scars are usually well healed.

The multifocal chorioretinitis of lues produces necrotizing lesions in the retina as well as in the choroid. These lesions are often accompanied by exuberant pigmentation. Optic atrophy is also commonly seen. The disease must be assumed to be due to hematogenous dissemination of **Treponema pallidum** with multiple areas of focal infection in the retina and choroid. Its rarity in secondary ry lues and its relative frequency in congenital lues remain unexplained.

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TOXOCARIASIS AND OTHER PARASITIC UVEITIS

Toxocara is a common intestinal parasite of dogs (*T. canis*) and of cats (*T. cati*). Man may be an incidental host for this parasite if the infective ova are ingested. This generally occurs in small children who eat dirty contaminated with Toxocara eggs or who maintain such a close association with pets as to transfer the eggs from the external surface of the animal's body to their mounts. The swallowed ova, containing viable larvae, are digested in man's interestinal tract. The larvae penetrate the intestinal wall, gain access to the mesenteric vessels, and ultimately are distributed to the liver, spleen, lungs, and eyes, among other organs. A systemic disease, first recognized in 1952 and known as visceral larva migrans, often develops if large eosinophilia, hepatosplenomegly, and pulmonary infiltrates. Ocular involvement may take one of three forms: (1) diffuse endophthalmitis; (2) posterior pole granuloma, or (3) peripheral localized granuloma.

Ocular involvement usually does not occur in association with the systemic form of the disease, but the reason for this is not clear. Eosinophilia is present in the majority of the cases of visceral larva migrans but not in ocular toxocariasis. It is fruitless to search the stool of potentially infected individuals for ova or larvae, because these forms never reach the intestine of man, as they would normally do in the dog or cat.

Diagnosis of Toxocariasis

Diagnosis is based on the appearance of the ocular lesion and upon confirmation of the infection by serologic tests. The ELISA test (enzyme-linked immunosorbent assay) appears to be highly sensitive and highly specific. However, Biglan et al. have shown that titers of antibody may be higher in the vitreous than in simultaneously tested serum. Toxocariasis may be differentiated from retinoblastoma by the finding of eosinophils in the aqueous or vitreous humor. Lactic dehydrogenase (LDA) enzymes may be found in the aqueous humor of patients with retinoblastoma, but are not present in patients with toxocariasis.

Treatment of Toxocariasis

Toxocara infections in man have been treated with anthelmintics such as Thiobendazole; however, no medication has proven consistently effective. Furthermore, there is some evidence from the work of Byers and Kimura that the death of the worm may be accompanied by florid ocular inflammation, as is known to be the case in ocular onchocerciasis. There is some indication that corticosteroids, given by the oral route or by sub-Tenon's injections, may quiet the more exudative of the endocular inflammations.

Suggested Dosage

Prednisone 4-60 mg. per day.

Kenalog ® (Triamcinolone acetonide) 5-10 mg. (0.5-1.0 ml.) by injection.

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OTHER FORMS OF PARASITIC UVEITIS

Although **Ascaris** infestation is thought to be an uncommon source of ocular morbidity, a case of severe iridocyclitis with dislocation of the lens and secondary glaucoma was described by Calhoun. A subretinal Ascaris larva was followed in the fundus of a patient by Parsons for a period of 3 years, during which time the worm remained actively motile. Distribution of the larvae to the eye and brain must occur during the time that the larvae would normally exit from the alveolar capillaries of the lung. If the larvae are returned to the left ventricle of the heart, distribution of the errant larvae to many different organs is possible.

Cysticercosis is a serious cause of ocular morbidity in Mexico and other areas of the world where fecal contamination of food and water supplies is common. The problem arises from the ingestion of the eggs of **Taenia solium** or from reverse peristalsis in cases of intestinal obstruction caused by adult tapeworms. The ocular presentation of the larva is usually subretinal, although the vitreous and anterior chamber may occasionally be invaded. The treatment is generally surgical excision of the cysticercus, although some retinal cysticerci have been treated by photocoagulation, preceded by systemic corticosteroid therapy to prevent overly violent reactions to the larval antigens.

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Uveítes Reumáticas

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A região do trato uveal predominantemente envolvida nos casos de doença reumática é — fato já amplamente conhecido e estudado — a úvea anterior. Desta maneira, podemos encontrar, naqueles casos, uma irite pura ou, quando há comprometimento do corpo ciliar, uma iridociclite.

HISTÓRICO (Rocha, 1969)

1888 — Del Monte — "a mais frequente causa da irite é o reumatismo".

1909 — De Wecker — "irite reumatismal deveria ocupar o primeiro lugar, antes mesmo que a sífilis".

1925 — Charlin — "só é irite reumatismal quando temos antecedentes reumáticos evidentes".

1966 — Duke-Elder — "a irite reumática dos antigos autores é um têrmo que deve ser abandonado; as doenças articulares e musculares devem ser consideradas, como a irite, manifestações de doença sistêmica".

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