Toxocaral chorio-retinitis

Treatment of early cases with photocoagulation

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Since Wilder (1950) described nematode endophthalmitis in patients previously diagnosed as cases of pseudoglioma, and Nichols (1956) identified the nematode as *Toxocara canis*, it has been recognized that this organism, which is very common in puppies all over the world, is liable to infect the human eye, particularly in young children. Ashton (1960) demonstrated the pathology of the condition. Ocular toxocariasis manifests itself clinically as a granuloma at the posterior pole or as a chronic endophthalmitis. Duguid (1961) suggested that the granuloma occurred when a previous infection had conferred high resistance and low tissue sensitivity, while chronic endophthalmitis developed when sensitivity was high and resistance low. Perkins (1966) showed that chronic endophthalmitis was more common than granuloma.

However, as more cases of toxocariasis are diagnosed, other manifestations also appear to be quite common. A lesion resembling macular degeneration was recently described by Rubin, Kaufman, Tierney, and Lucas (1968) as being due to an intraretinal *Larva migrans*, most probably *Toxocara*, which produced a localized haemorrhage. Smith and Greer (1971) reported a case of acute anterior uveitis with subsequent development of a retinal mass at the ora serrata, which progressed to endophthalmitis and was found histologically to be toxocaral. This peripheral chorio-retinitis was also thought by Perkins (1966) to be due to *Toxocara* in a number of cases in children. Peripheral retinitis and chronic cyclitis in children were also reported by Hogan, Kimura, and O'Connor (1965) as being due to nematode infestation.

Granulomatous lesions in the macula in the three cases reported below were suggestive of toxocariasis on clinical and serological grounds; two were very early granulomatous lesions in adolescents.

We have tried a new form of treatment, namely photocoagulation, in these two early cases with satisfactory results.

Case reports

Case 1, a 16-year-old boy, presented on August 13, 1969, with defective vision in the right eye, which had been discovered by chance one month previously. He had been given topical and systemic steroids for the last month.

Examination

The visual acuity in the right eye was counting fingers at 1 m. There was no external abnormality, but ophthalmoscopy revealed a localized, slightly raised macular lesion, about two disc diameters across (Fig. 1). The left eye was normal and the visual acuity 6/5.

FIG. 1 Case 1 before treatment

Laboratory investigations

White blood count 10,500/mm.³, total neutrophils 75 per cent., eosinophils 5 per cent.; skin test + + +; complement-fixation test +; haemagglutination test +. Urine and stool normal. Blood Wassermann reaction and Mantoux test normal.

The boy had been in close contact with a puppy and the lesion was due to a *Toxocara* larva.

Treatment

Photocoagulation was used to destroy the larva *in situ* and stop the lesion progressing to a macular granuloma and possible endophthalmitis (Fig. 2).

Result

The photocoagulation scar became flat and has since remained unchanged (Fig. 3).

FIG. 2 Case 1 immediately after photocoagulation.

FIG. 3 Case 1 at follow-up after 6 months

Case 2, a girl aged 17 years, was first seen on August 24, 1971, complaining of defective vision in the left eye.

Examination

The visual acuity in the left eye was 1/60. No external abnormality was found, but fundus examination revealed a raised macular lesion surrounded by a fringe of blood.

The right eye was normal with visual acuity 6/6.

Laboratory investigations

The white blood count showed 3 per cent. eosinophilia and the skin test was strongly positive. Urine and stool normal. Blood Wassermann reaction and Mantoux test negative.

The family kept several dogs at their country house, and the lesion was thought to be due to infestation with *Toxocara*.

Treatment

Photocoagulation was applied and resulted in a small flat macular scar.

Case 3, a young woman aged 25, who had experienced rapid blurring and clouding of vision in the right eye for a month, consulted me on May 23, 1970.

Examination

The visual acuity was counting fingers at 50 cm., but anterior segment slit-lamp examination revealed no abnormality. Ophthalmoscopy showed a marked posterior vitreous haze and large raised macular mass.

Laboratory investigations

An eosinophilia of 7 per cent. with a strongly positive skin test suggested that the lesion was due to *Toxocara*.

Treatment

Local corticosteroids and atropine and subconjunctival decadron and systemic steroids caused marked clearing of the vitreous after 2 weeks.

Default

The patient did not return for follow-up and further treatment.

Comment

Early clinical diagnosis of ocular toxocariasis is often difficult. Children rarely complain, but occasionally a red eye with acute anterior uveitis and hypopyon may be noticed by the parents (Smith and Greer, 1971). Older children may present earlier, but even they may only accidentally become aware of defective vision in one eye. Young patients with a history of contact with dogs who show granulomatous lesions at the posterior pole, particularly with small surrounding haemorrhages, positive serological tests (especially eosinophilia), and a positive skin test, and absence of any other lesion in the affected or the contralateral eye, should be regarded as possible cases of toxocaral infestation and should be managed accordingly. The haemorrhage appears to be characteristic of recent lesions, the departure of the larva from the vessel, when its body is as large as the vessel diameter, being the cause of bleeding (Woodruff, 1970). Haemorrhage, which was noted in two cases by Ashton (1960), fringed the small granuloma in one of our cases. Rubin and others (1968) observed a retinal haemorrhage where a presumed *Toxocara* larva was seen to be

moving. The specificity of the skin test has been documented experimentally by Woodruff. (1970, 1971); the test appears to be so specific that experimental infestation with only twenty eggs in monkeys was sufficient to produce a positive reaction. A negative reaction had been obtained before the injection of *Toxocara canis*, even though there was pre-existing infestation with other nematodes including hook worm.

The intraocular infection may progress rapidly.

The treatment of toxocaral chorio-retinitis is not easy. Most of the cases so far reported had already progressed to severe endophthalmitis with retinal detachment, cyclitic membrane, or secondary glaucoma (Duguid, 1961; Nolan, 1968; Smith and Greer, 1971). Ashton (1960) stated that the best treatment was prevention, and that young dogs and cats in close contact with children should be regularly wormed. Nolan (1968) used subconjunctival depot corticosteroids for cases of toxocaral endophthalmitis. Woodruff (1970) reported good results with a standard course of diethylcarbamazine for 21 days. This drug is believed to kill the migrating larva which otherwise lives for a long time and continues to cause damage. In fifteen children treated by Woodruff (1971) with diethylcarbamazine, the eye remained quiet.

Our results with photocoagulation were satisfactory. The lesion was reduced to a limited flat scar, and the oedema around it subsided in a few weeks with slight improvement of vision. Central vision will be preserved if the lesion is not near the macula, and further progression of the inflammation will be prevented if the larva is killed.

Summary

Three early granulomatous macular lesions are reported. A history of contact with dogs, a positive skin test, and eosinophilia suggested that they were due to *Toxocara* infestation. Photocoagulation was tried in two cases with a satisfactory result, and this is recommended, particularly in early cases in which the lesion is not near the macula.

References

- ASHTON, N. (1960) *Brit. J. Ophthalm.*, **44**, 129
 DUGUID, J. M. (1961) *Ibid.*, **45**, 705
 ——— (1961) *Ibid.*, **45**, 789
 HOGAN, M. I., KIMURA, J. J., and O'CONNOR, G. R. (1965) *Trans. ophthalm. Soc. U.K.*, **85**, 39
 NICHOLS, R. L. (1956) *J. Parasit.*, **42**, 349, 363
 NOLAN, I. (1968) *Brit. J. Ophthalm.*, **52**, 276
 PERKINS, E. S. (1966) *Ibid.*, **50**, 169
 RUBIN, M. L., KAUFMAN, H. E., TIERNEY, I. P., and LUCAS, H. C. (1968) *Trans. Amer. Acad. Ophthalm. Otolaryng.*, **72**, 855
 SMITH, P. H., and GREEN, C. H. (1971) *Brit. J. Ophthalm.*, **55**, 317
 WILDER, H. C. (1950) *Trans. Amer. Acad. Ophthalm. Otolaryng.*, **55**, 99
 WOODRUFF, A. W. (1970) *Brit. med. J.*, **3**, 663
 ——— (1971) Personal communication