Toxocariasis in Sibs

J. A. KUZEMKO*

From the Department of Paediatrics, Churchill Hospital, Oxford

Toxocariasis is caused by larva of the nematode *Toxocara canis* or *catidis*. It has been found in a survey that in Southern England about 20% of dogs and cats are infected by the parasite (Woodruff, Thacker, and Shah, 1964). Man becomes infected by ingesting eggs which develop into larvae in his gastro-intestinal tract and then penetrate through the wall invading various organs in the body, most commonly the liver, lungs, and eyes. But any organ can be involved. The parasite usually persists in the larval stage in man, and can remain alive for a year or longer (Dent, Nichols, Beaver, Carrera, and Staggers, 1956). The lesions formed are of a granulomatous nature and consist of eosinophils, giant cells, and lymphocytes, which typically surround larvae and may lead to necrotic changes.

Two cases in sibs are described. It is suggested that the disease is much more common than is generally believed and, though it may run a benign course with full recovery, it seems, judging from the many reports of serious ocular manifestations (Ashton, 1960), that it should be treated as a potentially serious disease.

Case Reports

Case 1. A boy, aged 2 years, was admitted to hospital in April 1963, for investigation of hepatosplenomegaly. The mother complained that during the last two or three weeks his abdomen had become distended and seemed firm; he had been listless and restless and developed an unproductive cough. Up to that time he had always been well. Examination showed a well-nourished boy, weight 29 lb. (13.2 kg.), pyrexial (99.6° F. (37.6° C.) rectal) with generalized lymphadenopathy, enlarged liver (1.5 in. (3.8 cm.) below the costal margin) and spleen (2.5 in. (6.3 cm.) below the costal margin). There was a short systolic apical cardiac murmur and a right-sided inguinal hernia which was easily reducible. Investigation revealed white cell count 19,700/c.mm. with many atypical lymphocytes (70%), haemoglobin 10.2 g./100 ml., and a patch of bronchopneumonia at the right base.

Paul Bunnell test, toxoplasma dye test, quantitative skin sweat test for cystic fibrosis, liver function tests, Wassermann and Kahn tests, agglutination tests, blood sugar, and glucose tolerance tests were all negative. It was thought that he might have an atypical form of glandular fever. He settled well and within a fortnight was discharged home. Three weeks later he was again reinvestigated but all the findings were normal apart from an eosinophil count of 1,650/c.mm. Since his liver and spleen had diminished in size, no liver biopsy was done. He seemed in good health for the next three months when he was admitted again because 'he was well one day and off colour the next day and is awake most nights crying with what the mother thinks is abdominal pain'. There was now a history of exertional dyspnoea for one month and diarrhoea of two weeks' duration. Examination showed an enlarged liver (2 in. (5 cm.) below the costal margin), but only the tip of the spleen could be felt. Otherwise he seemed well. Investigations again showed an eosinophil count of 1,381/c.mm., a total white cell count of 12,200/c.mm., and haemoglobin 12.3 g./100 ml. Liver function tests were normal. His hernia was repaired and at the same time a bone-marrow biopsy was performed which showed an increase in eosinophil precursors. Tests for trichinosis and toxoplasmosis were again negative. Finally he was admitted again in July 1964, with his younger sister (Case 2) who was admitted for investigation of hepatomegaly. As on previous occasions, he was found to have an enlarged liver and a few shotty glands were palpable in the neck and both axillae. Hb 12.4 g./100 ml., white cell count 8,200/c.mm., with eosinophil count of 1,300; serum caeruloplasmin level of 60 mg./100 ml. (normal 30-40 mg./100 ml.). Total proteins 7.6 g./100 ml., albumin 4.6 g., globulin 3 g. A toxocara intradermal test was strongly positive. Throughout the time that he was under observation, no abnormal eye signs were ever recorded.

Case 2. Sister of Case 1, 18 months of age, was admitted in July 1964, because she seemed to be developing like her brother. There was a history of diarrhoea, intermittent attacks of abdominal enlargement, and breathlessness of a few weeks' duration. Examination showed an enlarged liver (2 in. (5 cm.) below the costal margin) and a few palpable lymph glands in the neck, axillae, and groins. The spleen was not felt. The fundi were normal. Investigations revealed haemoglobin 11.2 g./100 ml. White cell count 15,000/c.mm., with

Received November 18, 1964.

* Present address: Alder Hey Children's Hospital, Eaton Road, Liverpool 12.

total eosinophil count of 1,300. Serum caeruloplasmin level 55 mg./100 ml. Total proteins 7.4 g./100 ml., albumin 4.1 g., globulin 3.3 g. Toxocara intradermal test was strongly positive.

Environment. The family live on a farm, and keep puppies and cats. There are 8 other children but so far they have been clinically well. Toxocara intradermal tests were performed on the remaining members of the family. One child gave a doubtfully positive reaction, otherwise the tests were negative.

Discussion

The diagnosis of toxocariasis is essentially a clinical one. Liver enlargement, persistent eosinophilia, and hyperglobulinaemia are virtually diagnostic of the condition. A history of contact with puppies or kittens is also very helpful. Other features of the disease are usually non-specific and may include various skin rashes, asthmatic attacks, failure to thrive, or anaemia. If the larvae invade the eyes, choroiditis or even iritis may be present. Splenomegaly may also be present, and Snyder (1961) puts the incidence as high as 45%. Blindness or a mass in the eye simulating retinoblastoma are late but unfortunately common presentations (Ashton, 1960).

Recently a reliable intradermal test using toxocara antigen has been developed by Woodruff *et al.* (1964). In both our cases the tests were strongly positive. Although Jung and Pacheco (1960) described a haemagglutination test in toxocariasis, its value remains doubtful, and it is not generally used at present.

Diagnosis, however, is only the first and easier step. It is the question of treatment that is presenting great difficulty in management of these cases, for few treated cases have been described and hence the results of treatment are difficult to assess. Both our cases had a course of diethylcarbamazine citrate 30 mg./kg. for two weeks. It was noted that the liver and spleen decreased in size, and there was also an appreciable drop in the eosinophil count. However, it is too early to assess the full results of treatment. One feels, however, that some therapeutic agents should be tried in all cases. In an interesting report, Bourke and Yeates (1961) described the condition in a boy who presented at the age of 15 months in a similar way to our two cases. The child continued to thrive well up to the

age of 5 years, when he became blind in the right eye. Snyder (1961) quotes a similar patient who also became blind four years later. Pike (1960), following experiments on mice, is of the opinion that diethylcarbamazine citrate 10-30 mg./kg. of body weight given for three to four weeks may be very useful. The drug has virtually no serious side-effects apart from mild ones such as gastro-intestinal upsets, fever, or headache. ACTH has been tried with success in a fulminating case (Heiner and Kevy, 1956). Chloroquine diphosphate and 'thibenzole' are being tried in the U.S.A., but results so far are equivocal (Gellis and Kagan, 1964). Hence prevention remains the only safe weapon at disposal at present.

It is clear, therefore, that once the diagnosis is made, a child should be followed up for many years and particular attention paid to any eye changes. Meanwhile, a short course of diethylcarbamazine is worth a trial.

Summary

Toxocariasis in two sibs is described.

Diagnosis and treatment are briefly discussed.

I wish to thank Dr. B. W. Powell, Paediatrician to the Peterborough Group of Hospitals, for helpful criticism and encouragement and permission to publish the cases, Professor A. W. Woodruff of the London School of Tropical Medicine, for the generous supply of the antigen, and Dr. J. M. Walshe, of the Department of Experimental Medicine, Cambridge, for performing the serum caeruloplasmin estimations.

REFERENCES

- Ashton, N. (1960). Larval granulomatosis of the retina due to *Toxocara*. *Brit. J. Ophthalm.*, **44**, 129.
- Bourke, G. M., and Yeates, F. M. (1961). Blindness due to household pets (*Toxocara canis* infestation). *Med. J. Aust.*, **48**(2), 12.
- Dent, J. H., Nichols, R. L., Beaver, P. C., Carrera, G. M., and Stagers, R. J. (1956). Visceral larva migrans; with a case report. *Amer. J. Path.*, **32**, 777.
- Gellis, S. S., and Kagan, B. M. (1964). (eds.) *Current Pediatric Therapy*. Saunders, Philadelphia.
- Heiner, D. C., and Kevy, S. V. (1956). Visceral larva migrans: report of the syndrome in three siblings. *New Engl. J. Med.*, **254**, 629.
- Jung, R. C., and Pacheco, G. (1960). Use of a hemagglutination test in visceral larva migrans. *Amer. J. trop. Med.*, **9**, 185.
- Pike, E. H. (1960). Effect of diethylcarbamazine, oxphenarsine hydrochloride and piperazine citrate on *toxocara canis* larvae in mice. *Exp. Parasit.*, **9**, 223.
- Snyder, C. H. (1961). Visceral larva migrans. Ten years' experience. *Pediatrics*, **28**, 85.
- Woodruff, A. W., Thacker, C. K., and Shah, A. I. (1964). Infection with animal helminths. *Brit. med. J.*, **1**, 1001.