

# Symmetrical Optic Nerve Head Granulomas with Bilateral Vitreous Opacities

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Received: November 10, 2000  
Key Words: Optic nerve head granuloma, vitreous opacities, optic nerve

The larvae of the nematode Toxocara canis was first identified as a cause of intracocular inflammation by Nichols (1956) (1,2). Patients with ocular involvement are otherwise healthy and they have a normal white cell count with absence of eosinophilia; a history of pic is less common, and the average age at presentation is considerably older (7.2 years) compared with visceral larva migrans (5 years) (3). Ocular toxocariasis, was classified by Wilkinson and Welch in 1971 as (1) a chronic endophthalmitis-like picture, (2) posterior pole granuloma, and (3) peripheral inflammation (1-3), and mostly affects strictly one eye (3,4).

In this paper, we report an unusual case of bilateral ocular toxocariasis with chronic vitritis and bilateral vitreous opacities. The patient was a 7-year-old boy who presented to the Gullulu Eye Health Centre in March 1999. He gave a 3-month history of bilateral decreased visual acuity. On examination his corrected visual acuities were 6/10 right and 5/10 left. The intraocular pressure was 20 mm Hg on the right and 19 on the left. The eyes were normal on biomicroscopy. On posterior segment examination of the right eye, there were pigmented cells in the vitreous, which suggested chronic vitritis, and somewhat obscured fundus details. However, a round, white-solid, 1x3 disc diameter sized granuloma located at the optic disc with dense connective tissue strands in the vitreous cavity was noted (Figure 1-A). On the posterior segment of the left eye, there were also more densely pigmented cells in the vitreous; and a round, white-solid, 1x2 disc diameter sized granuloma located at the optic disc with an extension to the periphery at the inferior pupillary margin was noted (Figure 1-B).

In his history there was no contact with puppies or PICAs. Systemic examination was negative with no evidence of regional or generalized lymphadenopathy, skin lesion, pulmonary infiltration, arthropathy or hepatosplenomegaly. A number of laboratory tests were carried out. The X-ray examination of the chest showed no abnormalities. Complete blood count showed a slight leucocytosis of  $11 \times 10^9/L$ . Differential white cell count showed an eosinophilia of 41%. The serum Toxocara ELISA test (International Immunodiagnostic-U.S.A., ELISA test) and serology (Microbiology ELISA) was performed on 1:100 dilution serum samples and positive result obtained on 420 nm. Fluorescein angiography of both eyes confirmed that the optic disc fluorescence was masked at the site of lesion in the initial part of the dye transit. In the venous and late phases, there was progressive uptake of dye in the central areas of the mass (Figure 2-A, B, C).

## Case Report

In this paper, we report an unusual case of bilateral ocular toxocariasis with chronic vitritis and bilateral vitreous opacities. The patient was a 7-year-old boy who presented to the Gullulu Eye Health Centre in March 1999. He gave a 3-month history of bilateral decreased visual acuity. On examination his corrected visual acuities were 6/10 right and 5/10 left. The intraocular pressure was 20 mm Hg on the right and 19 on the left. The eyes were normal on biomicroscopy. On posterior segment examination of the right eye, there were pigmented cells in the vitreous, which suggested chronic vitritis, and somewhat obscured fundus details. However, a round, white-solid, 1x3 disc diameter sized granuloma located at the optic disc with dense connective tissue strands in the vitreous cavity was noted (Figure 1-A). On the posterior segment of the left eye, there were also more densely pigmented cells in the vitreous; and a round, white-solid, 1x2 disc diameter sized granuloma located at the optic disc with an extension to the periphery at the inferior pupillary margin was noted (Figure 1-B).

reported cases are of ocular lesions in children (3,7-8). Clinical reports of retinal lesions in adults due to presumed toxocaral infection are few in number (2,9). Posterior pole granulomas presentation is typically between the ages of 6 and 14 years (3). Our patient was in accordance with this definition. Although most common unilateral cases, bilateral cases have also very infrequently been reported (9,10). Yet none of the studies undertaken on bilateral diseases have so far reported bilateral vitreous symmetrical optic nerve head granulomas.

Zerodopsin studies in the past were inadequate because of their lack of sensitivity and specificity. In recent years, enzyme-linked immunosorbent assay using larval T. canis antigen has been shown to be both sensitive and specific for T. canis (10). However, laboratory reporting of a negative serum ELISA may lead to error in diagnosis (4). Clinicians should be aware that serum ELISA with ocular nematode infection by T. canis may be low as in our case (1) or negative (1,4). On the other hand the test may be strongly positive and therefore of great diagnostic significance, if carried out on intracocular fluid from an infected patient (1,8,9,10). This will be especially important in childhood where a clinically similar picture may be caused by retinodispersal (1). Hence, taking samples of aqueous humor involves an invasive procedure and so it is not a common method of examination. Definite diagnosis of ocular toxocarasis can be made only by identifying the larva histologically (7). Even if a diagnosis can be made there are no effective treatment procedures at present.

D, E, F). Ultrasound examinations identified a preretinal granuloma at the site of the optic disc.

Initially, the patient was given 500 mg of systemic prednisone, every other breakfast, and the dose was gradually tapered over a period of eight weeks. No beneficial result of this therapy was observed. Therefore the lesions were followed up for one year without therapy. The latest examination was performed in June 2000. The signs and symptoms remained unchanged in both eyes.

The clinical must differentiate toxocarasis from other causes of uveitis, particularly retinoblastoma, reduced ocular inflammation and prevent loss of vision and amblyopia. The diagnosis of ocular toxocarasis is based on the ophthalmoscopic appearance of the lesions, serology and a history of exposure to puppies. The clinical features of systemic infection or visceral larva migrans are usually absent when ocular involvement is detected. The ocular form appears as either a vitritis or as a focal inflammation that resolves slowly but sometimes produces a retinal detachment (6).

With the exception of bilateral vitreous reaction and optic nerve head granulomas, our patient was in good health, and the signs of systemic disease were absent. The physical examination and the hemogram did not support the presence of visceral larva migrans. However, the clinical impression of Toxocarasis endophthalmitis was supported by a weak positive enzyme-linked immunosorbent assay titre in the serum. There was no history of exposure to puppies. Nearly all

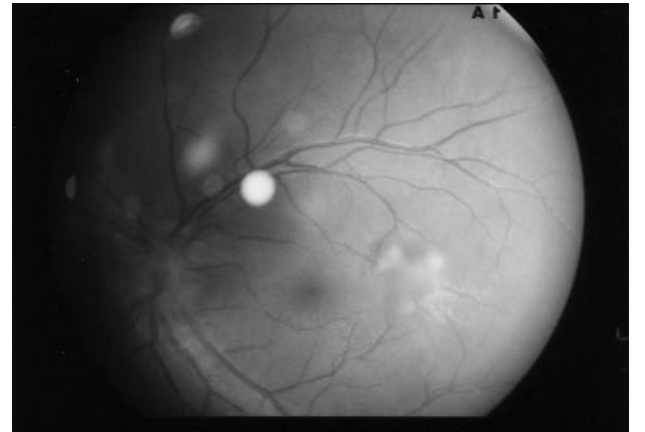
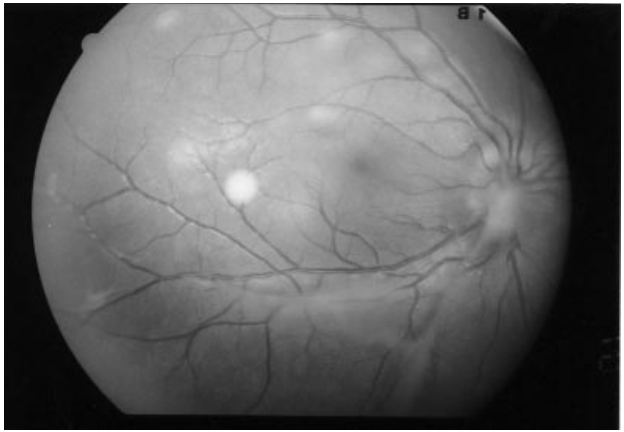


Figure 1. A: Optic nerve head granulomas of right eye with dense connective tissue strands in the vitreous cavity. B: Optic nerve head granulomas of left eye with an extension of peripheral vitreous at the inferior pupillary margin.

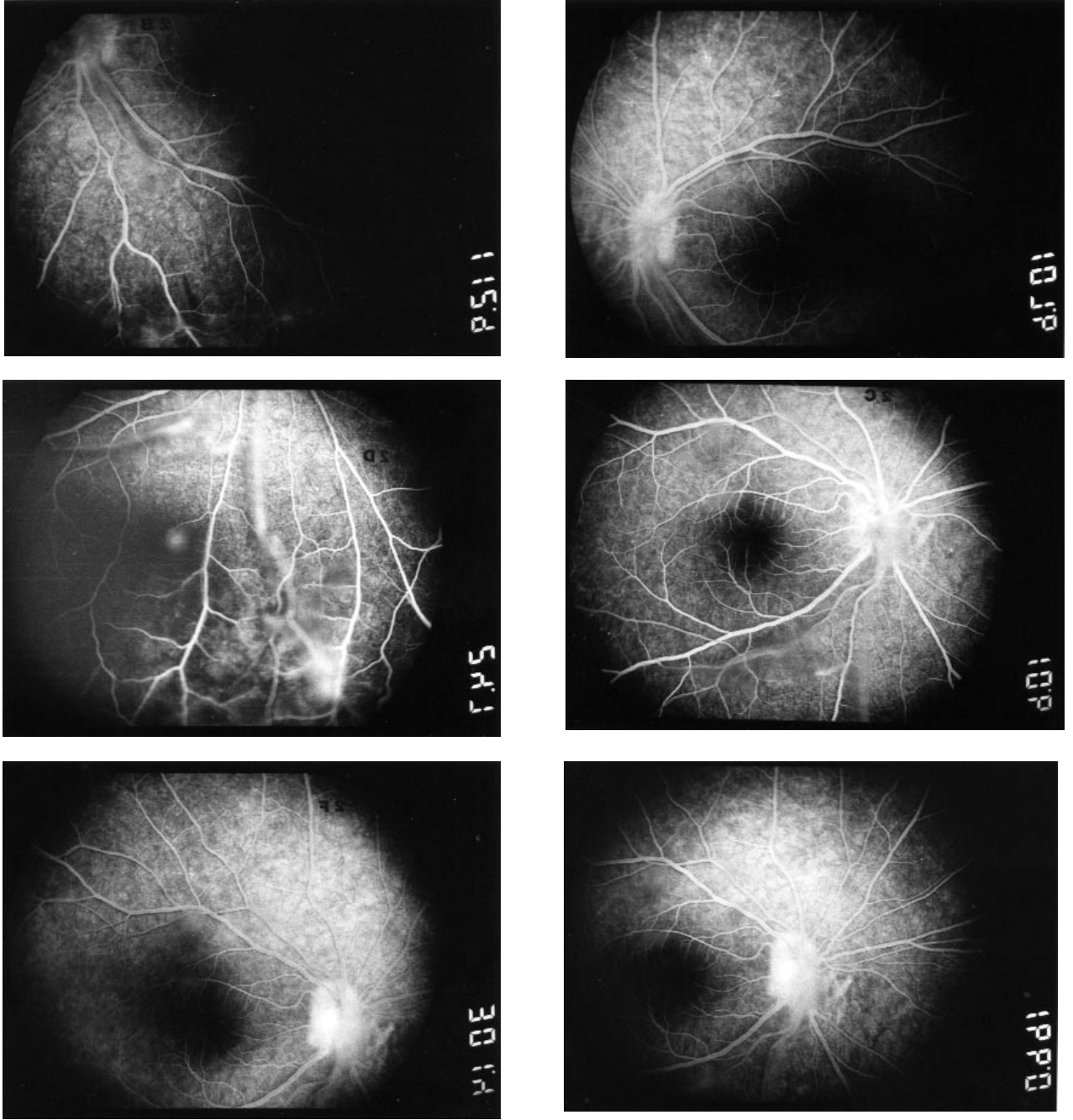


Figure 2. A, B: Fundus fluorescein angiography of right eye. S-C, D, E, F: Fundus fluorescein angiography of left eye. Notice in the initial part of the dye transit, masking of the optic disc fluorescence at the site of the lesion. In the venous and late phases, progressive uptake of dye in the central area of the mass.

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References

1. Takayanagi TH, Akao N, Suzuki R et al. New animal model for human ocular toxocariasis: ophthalmoscopic observation. *Br J Ophthalmol* 83: 967-72, 1999.
2. Paistick ER, Dean Hart JC: Ocular toxocariasis in adults. *Br J Ophthalmol* 60: 365-70, 1976.
3. Kanski JJ: Clinical Ophthalmology. Butterworth-Heinemann Ltd, London 1994, pp: 179-81.
4. Sharkey JA, McKay PC: Ocular toxocariasis in a patient with repeatedly negative ELISA titre to *Toxocara canis*. *Br J Ophthalmol* 77: 523-4, 1993.
5. Tan VT, Lumbroso L, LeHoang P, et al. Ultrasound biomicroscopy in peripheral retinovitreous toxocariasis. *Am J Ophthalmol* 127: 607-9, 1999.
6. Biglan AW, Glickman LT, Lopes LA. Serum and vitreous *Toxocara* antibody in nematode endophthalmitis. *Am J Ophthalmol* 88: 898-901, 1979.
7. Lyess RW, Eshley OE, Logan WC, et al. Ocular larva migrans: a case report. *Br J Ophthalmol* 71: 396-401, 1987.
8. Benitez del Castillo JM, Herreros G, Guillen JL, et al. Bilateral ocular toxocariasis demonstrated by aqueous humor Enzygme-Linked Immunosorbent Assay. *Am J Ophthalmol* 119: 214-6, 1992.
9. Beiran J, Cochavi O, Miller B. "Silent" ocular toxocariasis. *Eur J Ophthalmol* 8 (3): 192-6, 1998 Jul-Sep.
10. Maiti L, Ghoshal M, Aiyadi A, et al. *Toxocara canis* in apparently bilateral ocular site. *J Fr Ophthalmol* 13 (2): 303-8, 1990.