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Short Report

Ocular Toxocariasis with Bilateral Virtually Symmetrical Optic Nerve Head Granulomas

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The larva of the nematode Toxocara canis was first identified as a cause of intraocular inflammation by Nichols (1950) (1,2). Patients with ocular involvement are otherwise healthy and they have a normal white cell count with absence of eosinophilia; a history of pica is less common, and the average age at presentation is considerably older (7.5 years) compared with visceral larva migrans (2 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 inflammatory mass (1-5), and mostly affects strictly one eye (3,5).

In this paper, we report an unusual case of bilateral ocular toxocariasis with chronic vitritis and bilateral virtually symmetrical optic nerve head granulomas. Diagnosis was clinically and serologically confirmed.

Case Report

A 7-year-old boy attended 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, 1/3 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, 1/2 disc diameter sized granuloma located at the optic disc with an extension to the periphery at the inferior quadrant was noted (Figure 1-B).

In his history there was no contact with puppies or PICA. Systemic examination was negative with no evidence of regional or generalised 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 11×10^9/L. Differential white cell count showed an eosinophilia of 4.1%. The serum Toxocara ELISA test (International Immunodiagnostic-USA, Toxocara Serology Microwell ELISA) was performed on 1:100 dilution serum sample and positive result obtained on 450 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 area of the mass (Figure-2-A, B, C,

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

Initially, the patient was given 24 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 clinician must differentiate toxocariasis from other causes of uveitis, particularly retinoblastoma, to reduce ocular inflammation, and prevent loss of vision and amblyopia. The diagnosis of ocular toxocariasis 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 inflammatory mass that resolves slowly but sometimes produces a retinal detachment (6).

With the exception of bilateral inflammatory vitreous reaction and optic nerve head granuloma, 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 Toxocara 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 reported cases are of ocular lesions in children (3,5-8). Clinical reports of retinal lesions in adults due to presumed toxocaral infection are few in number (2,5,9). Posterior pole granuloma presentation is typically between the ages of 6 and 14 years (3). Our patient was in accordance with this definition. Alongside 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 virtually symmetrical optic nerve head granulomas.

Serodiagnostic 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 specific and sensitive for *T. canis* (6). 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 intraocular fluid from an infected patient (1,4,6,8). This will be especially important in childhood where a clinically similar picture may be caused by retinoblastoma (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 toxocariasis 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.

Figure 1. A: Optic nerve head granuloma of right eye with dense connective tissue strands in the vitreous cavity. 1-B: Optic nerve head granuloma of left eye with an extension to periphery at the inferior quadrant.

Figure 2. A, B: Fundus fluorescein angiography of right eye. 2-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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