CASE REPORT

A Rare Case of Subretinal Cysticercosis

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Summary

This is a case of a 25 year old lady whose eye had been infected by cysticercosis. This case highlighted that the inflammation was due to host immune response. She was treated with oral corticosteroid and the lesions regressed.

Key Words: Cysticercosis, *Taenia solium*, Subretinal cysticercosis

Introduction

A 25-year-old lady presented with blurring of vision of the left eye of ten days' duration. It was associated with left-sided headache and mild left eye discomfort. She had visited China one year ago during which she was exposed to cat's flea and developed rashes on the limbs, which resolved with treatment. She did not recall eating any raw food.

On examination of the left eye, visual acuity was 6/12, N5. The anterior segment examination was unremarkable. However, fundus examination showed a raised subretinal lesion about three-disc diameter in size located below the inferior arcade, with fluffy edges and subretinal blood centrally (Figure 1). There was mild vitritis overlying the lesion. There were two other focal areas of retinitis temporal to the fovea. The optic disc was hyperaemic with a blurred margin nasally. There were multiple chorioretinal scars at the posterior pole. A shallow exudative retinal detachment was noted inferiorly. The right eye was essentially normal.

Her full blood count was normal, erythrocyte sedimentation rate was raised and toxoplasma serology was negative. Since the subretinal lesion could be due to a parasite infection, serological test for parasites were done, including cysticercosis, which turned out to be positive. Other investigations, including MRI of the brain were normal. A diagnosis of subretinal cysticercosis was made. The patient was started on oral prednisolone, 60mg daily for one week, based on the knowledge that the inflammatory reaction in the eye occurs due to host immune response rather than the parasite itself. The lesion started regressing and the treatment was gradually tapered. While on 40mg daily of prednisolone, the patient was given anti-helminthic treatment by another ophthalmologist. Two weeks later (on 30mg daily of prednisolone), new lesions occurred at the superotemporal area and more vitritis was seen (Figure 2). The anti-helminthic was stopped and the prednisolone was stepped up to 40mg daily. The patient responded well to the treatment and the lesions regressed after about 3 months on treatment. However, the prednisolone was maintained at 10mg daily and stopped 2 months later.

Discussion

Cysticercus is the larvae of *Taenia solium*, a dangerous pork tapeworm in humans. Cysticercosis occurs when the eggs of Taenia solium are ingested. Cysticercosis usually occurs between the ages of 10 to 30 years old and has no sex predilection[1]. It is prevalent in areas where hygiene is poor particularly in Mexico, Central and South America, India, Southeast Asia, China and certain parts of Africa. This patient visited China one

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Fig. 1: Fundus picture of the left eye showing a raised subretinal lesion located inferiorly, associated with exudative retinal detachment. Multiple chorioretinal scars are also seen at the posterior pole.

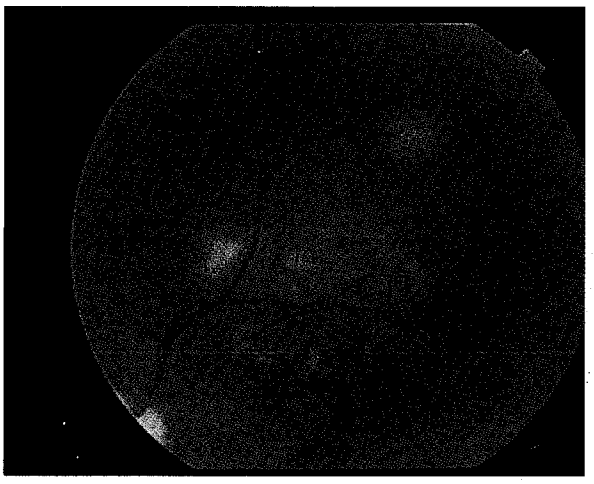

Fig. 2: Fundus picture showing the new lesions on the left eye

year ago, therefore there is a possibility that she got infected there.

Human cysticercosis occurs when a human becomes the intermediate host by ingesting contaminated food or water, or auto infestation by reflex peristalsis of eggs from a resident adult parasite. Cysticercosis may involve various parts of the body including the eye (13% to 46%), subcutaneous tissue (24.5%) and the brain (13.6%)[2]. It may also affect the skeletal muscle and heart muscle[1]. In the eye, the possible sites for encystment include subretinal space, vitreous, subconjunctival space, anterior chamber, recti muscles, lids, lacrimal gland and lens. Particularly in this patient, the cysticercosis occurred in the subretinal space. The larvae reaches the subretinal space via the posterior ciliary arteries[1,2]. The infection causes atrophic changes in the overlying retinal pigment epithelium as the cyst develops. Sometimes it may cause exudative retinal detachment and focal chorioretinitis. The inflammation in intraocular cysticercosis is more related to host immune response than the cysticercus itself[1]. However, the death of the parasite may lead to severe inflammation and endophthalmitis[1,2].

In this case, the diagnosis of cysticercosis was made by the clinical findings and supported by the serological test. The enzyme-linked immunosorbent assay (ELISA) that was used has a sensitivity of 50% and a specificity of 65% for cysticercosis. The diagnosis of cysticercosis can also be made by other methods. An anterior chamber tap showing a high eosinophil count may support the diagnosis[2]. In an opaque media, B-scan ultrasonography will show a curvilinear echo corresponding to the cyst wall. An A-scan analysis reveals two high amplitude echoes representing the anterior and posterior walls of the cyst[1,2]. A CT scan or MRI of the brain to look for solid, cystic or calcified nodules should be done to rule out brain involvement. Furthermore, the specific diagnosis of cysticercosis depends on biopsy material.

Treatment for cysticercosis is necessary because it has been reported that 80% of untreated cases result in severe ocular damage[2]. Anti-helminthic drugs such as praziquantel or albendazole have been found to be effective in central nervous system and skin cysticercosis[3]. Corticosteroids may be used in conjunction with these drugs to reduce the inflammatory response[1]. However, anti-helminthics are ineffective in intraocular cysticercosis[3]. This is probably due to insufficient concentration at that site. In this patient, corticosteroid treatment was given alone because it was believed that the inflammation was mainly due to host immune response rather than due to the parasite itself[1]. Anti-helminthics will cause energy

depletion, immobilization, and finally death of the parasites. When an anti-helminthic was added to the treatment, it was found that the inflammation increased. Therefore, it was postulated that probably the toxin released by the dead parasite gave rise to more inflammation. A high dose corticosteroid was used until regression of the lesions seen and then it was tapered down. A low dose of corticosteroid was maintained until all the lesions completely regressed and all surrounding oedema subsided. Diathermy, cryotherapy and photocoagulation had been advocated for the treatment of cysticercosis. However, these methods will give rise to severe intraocular inflammation due to the toxins released from the dead larval tissues[3]. Surgical removal of the cyst can also be done as a mode of treatment through either the transretinal or transcleral route[1,3]. Systemic corticosteroids are used before and after surgical removal of the cysticercus[2].

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