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The authors do not have any conflict or commercial interest related to the work.

Eye (2006) **20,** 611–612. doi:10.1038/sj.eye.6701928; published online 3 June 2005

Sir,

The Indian case of live worm in Diffuse Unilateral Subacute Neuroretinitis

There have been many reports of DUSN,[1] but none with a documented worm from India. We report a case of DUSN with live worm (size 1500–2000 μm) from South India, which was killed by a single shot of laser photocoagulation with subsequent visual improvement.

Case report

A 40-year-old, apparently healthy man from Kerala state, where filarial Burgia malayi was endemic, presented with right visual loss of 4 days duration. There was no history of contact with live animal. Right VA was 2/60 with RAPD and left was 6/6; otherwise there were unremarkable anterior segment findings. Right fundus revealed mild vitritis with a glistening white worm found in the temporal periphery among the diffuse subretinal tracts, Figure 1a. Full blood counts and stool examination were normal.

When the patient was scheduled for 532 nm Argon Green laser treatment on the same day, the worm had already migrated to the macula. Immediately, after a single shot of laser (spot size 200 μm, duration 150 ms, and power 160 mW) applied to the advancing end of the worm, it began to migrate to subfovea and further laser was abandoned, Figure 1b. A combination of antihelminthics Albendazole 400 mg stat[2,3] and Diethylcarbamazepine (100 mg tds for 21 days)[8] was started. On the next day, the worm was immobile with surrounding neuroretinitis, Figure 1c. The patient was put on oral corticosteroid 40 mg tapering over 21 days.

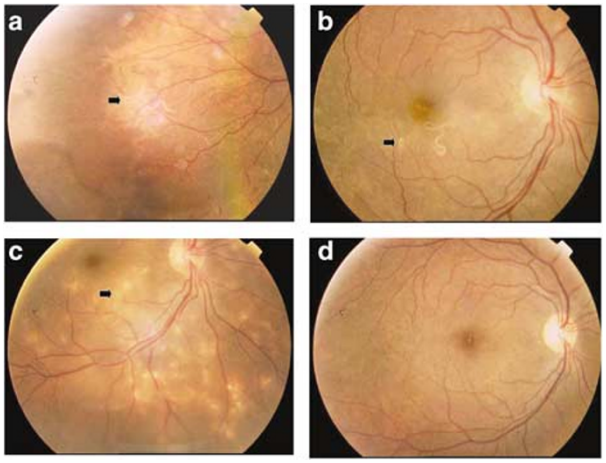

Figure 1 (a) Fundus picture of the right eye showing the worm (arrow) among crisscrossing subretinal tracts in the temporal midperiphery at presentation. (b) The same eye showing the migrated worm in the macular region with the arrow indicating the area where the laser was given. (c) The same eye showing the nonmotile dead worm (arrow) with surrounding serous detachment and new crops of evanescent white inflammatory lesions. (d) The same eye showing the resolved inflammatory lesions, 3 weeks postlaser.

After 3 weeks, his right vision improved to 6/60. There were diffuse pigmentary changes in the superior temporal fundus where the worm was first identified. However, there was no trace of the worm or of inflammation, Figure 1d.

Comment

DUSN is a clinical syndrome, caused by a motile, white, glistening nonsegmented nematode wandering in the subretinal space. It is characterized by vitritis, papillitis, and recurrent crops of white evanescent lesions, followed by severe visual loss, optic atrophy, and diffuse RPE degeneration.[4,5] At least two different sizes of the worm has been recorded: 400–1000 and 1500–2000 μm. The precise identity and portal of entry are still a mystery.[9] The visibility of motile worm is the gold standard in making the diagnosis of DUSN. However, identifying the small worm may be difficult and time consuming; therefore, fundus photography is useful to identify its location. The oral antihelmintics are ineffective, because of the impermeability of blood retinal barrier. It is recommended in those who have severe vitritis and in whom the worm cannot be easily identified.[8]

In our case, the death of the worm could not be attributed to oral antihelmintics, because the worm was found to be dead the next day after the laser. The worm was identified by its moving advanced end and we

managed to get a single laser shot. The use of oral steroid helped to control the inflammation caused by the dead worm and improved the final vision.

In most cases, it may not be easy to distinguish the head from the tail, especially in small worms. One approach is to lure the worm away from the macula with the aiming beam, followed by gradual burning over the worm with laser. A useful sign during the treatment is the slower uptake of the laser heat energy, seen as a fleeting translucency within the opaque laser spot.[6] When the live worm can be identified, laser photocoagulation is the treatment of choice[7,4,5]

Acknowledgements

The authors have no relevant financial interest in this article.

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Eye (2006) **20,** 612–613. doi:10.1038/sj.eye.6701936; published online 27 May 2005

Sir,

Ophthalmologic manifestations in headache, neurologic deficits, and cerebrospinal fluid lymphocytosis (HaNDL) syndrome with nonspecific frontal lesions and hyperthyroidism

Headache, neurologic deficits, and cerebrospinal fluid lymphocytosis (HaNDL) syndrome was first described as a migrainous syndrome with cerebrospinal fluid pleocytosis in 1981.[1] Recently, the ophthalmologic involvements in HaNDL have been emphasized.[2] We present ophthalmologic manifestations in an unusual case of HaNDL syndrome with nonspecific frontal lesions and hyperthyroidism.

Case report

A 39-year-old woman without personal or family history of migraine experienced 2 weeks of severe retrobulbar pain, headache and vomiting with no preceding viral illness. Her visual acuity and eye movement were normal. She had lid retraction in the right eye, bilateral papilloedema and bilateral tonic pupil. Goldmann perimetry showed bilateral enlargement of the blind spots. Additionally, the patient was diagnosed with hyperthyroidism 4 months previously. She was admitted to our hospital and a general neurological examination was unremarkable. She was not obese and she had no fever or any meningeal signs. All routine blood tests and haematochemistry were normal. No abnormal titres were found in the serum antibodies for any viruses. Several autoantibodies were negative (s-IL2 receptor, dsDNA, RNP, SS-A/B, c-ANCA, and p-ANCA).

Lumbar puncture revealed an opening pressure of 160 mmH_2O and 51.7 cells/mm^3 (lymphocytic predominance). Protein, glucose, and IgG in the cerebrospinal fluid were within normal limits and the oligoclonal band was negative.

Free T3 and T4 were within normal limits on admission, but the serum level of thyroid-stimulating hormone increased over time. While the antibodies for thyroid-stimulating hormone receptor and thyroglobulin were negative, the thyroid peroxidase antibody was positive. She was diagnosed with chronic thyroiditis (Hashimoto's disease) with rapid exacerbation.

T2-weighted magnetic resonance imaging (MRI) revealed small nonspecific areas of hyperintensity in the bilateral frontal lobe (Figure 1). She manifested horizontal gaze-evoked nystagmus, tinnitus and dysaesthesia of her bilateral fingers. These symptoms were temporary and self-limited. After 3 weeks , a repeat lumber puncture revealed 13.3 cells/mm^3. The papilloedema resolved following a 1-month course of acetazolamide.