

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.<sup>6</sup> When the live worm can be identified, laser photocoagulation is the treatment of choice<sup>7,4,5</sup>

### Acknowledgements

The authors have no relevant financial interest in this article.

### References

- Gupta A, Agarwal A, Dogra M. Retinal involvement in *Wucheria bancrofti* filariasis, Chandigarh. *Acta Ophthalmol* 1992; **70**: 832–835.
- Jung H, Hurtado M, Medina MT, Sanchez M, Sotelo J. Dexamethasone increases plasma levels of albendazole. *J Neurol* 1990; **237**: 279–280.
- Venkatesan P. Albendazole. *J Antimicrobial Chemother* 1998; **41**: 145–147.
- Hedges TR. Diffuse unilateral subacute neuroretinopathy. *Principles Pract Ophthalmol: Clin Pract* 2000; **3**: 2167–2171.
- Gass JD. *Stereoscopic Atlas of Macular Disease Diagnosis and Treatment*, Mosby Publisher: Saint Louis, USA. Vol 2, 1997, pp 622–628.
- Levada A, Davis J, Dennehy P. *Atlas Ophthalmol* 2000.
- Gass JD, Braunstein RA. Further observation concerning DUSN syndrome. *Arch Ophthalmol* 1983; **101**: 1689–1697.
- Gass JD, Callanan DG, Bowman CB. Oral therapy in diffuse unilateral subacute neuroretinitis. *Arch Ophthalmol* 1992; **110**: 675–680.
- Kazacos K, Raymond L, Kazacos E, Vestre WA. DUSN: probable cause. *Arch Ophthalmol* 1984; **102**: 967–968.

K Myint<sup>1</sup>, R Sahay<sup>2</sup>, S Mon<sup>1</sup>, VR Saravanan<sup>2</sup>, V Narendran<sup>2</sup> and B Dhillon<sup>1</sup>

<sup>1</sup>Eye Pavilion, Royal Infirmary of Edinburgh, Scotland

<sup>2</sup>Aravind Eye Hospital, Coimbatore, India

Correspondence: Dr K Myint,  
Princess Alexandra Eye Pavilion,  
Chalmers Street, Edinburgh EH3 9HA, UK  
Tel: +44 131 2211 846;  
Fax: +44 131 5363 897.  
E-mail: kmyintuk@yahoo.co.uk

*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.<sup>1</sup> Recently, the ophthalmologic involvements in HaNDL have been emphasized.<sup>2</sup> 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<sub>2</sub>O and 51.7 cells/mm<sup>3</sup> (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 lumbar puncture revealed 13.3 cells/mm<sup>3</sup>. The papilloedema resolved following a 1-month course of acetazolamide.

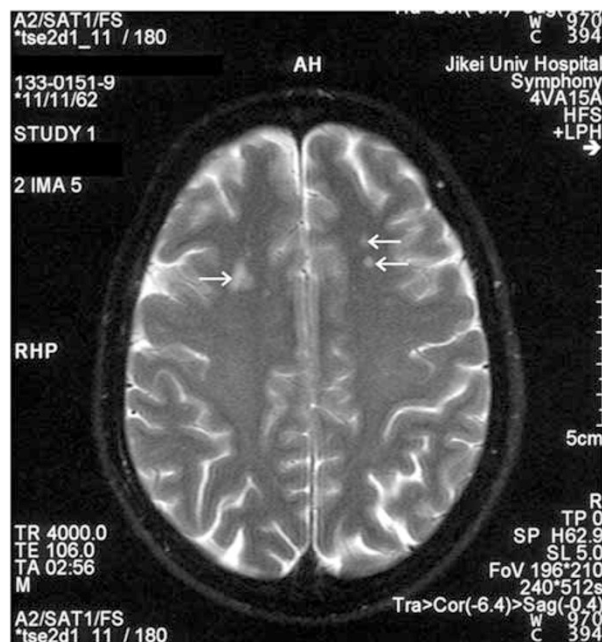


Figure 1 MRI showing multiple lesions (arrows).

**Comment**

The papilloedema, headache, cerebrospinal fluid lymphocytosis and temporary symptoms of horizontal gaze-evoked nystagmus, tinnitus, and dysaesthesia in the present case are highly suggestive of HaNDL. Berg *et al*<sup>3</sup> termed this syndrome HaNDL and the diagnostic criteria proposed included severe headache, temporary neurologic deficit, cerebrospinal fluid lymphocytosis and being self-limited. The present case fulfilled the diagnostic criteria and the disorder was differentiated from other migrainous syndromes. The differential diagnoses for migrainous diseases are listed in the Table 1. Barkana *et al*<sup>4</sup> has reported six cases with intracranial hypertension and unexplained cerebrospinal fluid pleocytosis. If these cases had temporary neurologic deficits, they also could be HaNDL. Although HaNDL is an uncommon disease, ophthalmologists must include HaNDL in any differential diagnosis used to assess patients with papilloedema and temporary neuro-ophthalmological signs. The neuroimaging abnormality we noted was similar to that mentioned previously in an advanced form of HaNDL.<sup>3,5</sup> The lid retraction in our case may be relevant to the hyperthyroidism. Tonic pupil may be due to a variety of causes including inflammation, vasculitis, or ischaemic processes that affect the ciliary ganglion in patients with autoimmune diseases.<sup>6-8</sup> The association of tonic pupil with hyperthyroidism and myasthenia gravis as part of an autoimmune disease has been previously reported.<sup>9</sup> In this case, the patient was associated with Hashimoto's

**Table 1** Differential diagnoses for migrainous diseases

	HaNDL	Pseudotumour cerebri	Meningitis	Migraine
Headache	+	+	+	+
Neuroimaging	-/+	-	-	-
Cerebrospinal fluid				
Pressure	↑	↑	↑	→
Cells	↑	→	↑↑	→
Temporary Neurologic deficits	+	-	-	-(+)
Fever	-(+)	-	+	-
Meningeal signs	-	-	+	-

disease, which is an autoimmune disease. Although the precise aetiology of the association remains obscure, the tonic pupils in this case could be related to an autoimmune disease. Thus far, no clear causal link has been established between Hashimoto's disease and cerebrospinal fluid lymphocytosis, although hyperthyroidism is known to cause increases in intracranial pressure.<sup>10</sup> The aetiology of HaNDL is unknown but an inflammation such as an allergic response after an infection or an autoimmune disease is more likely rather than an infection because of the noninfectious profile that is seen on cerebrospinal fluid analysis, the lack of fever, and a normal serum complete blood count. Autoimmune disorders might be one of causative factors of HaNDL. Further examples are required in order to clarify an aetiologic relationship between thyroid dysfunctions or autoimmune conditions and HaNDL.

**References**

- 1 Bartleson JD, Swanson JW, Whisnant JP. A migrainous syndrome with cerebrospinal fluid pleocytosis. *Neurology* 1981; **31**: 1257-1262.
- 2 Morrison DG, Phuah HK, Reddy AT, Dure LS, Kline LB. Ophthalmologic involvement in the syndrome of headache, neurologic deficits, and cerebrospinal fluid lymphocytosis. *Ophthalmology* 2003; **110**: 115-118.
- 3 Berg MJ, Williams LS. The transient syndrome of headache with neurologic deficits and CSF lymphocytosis. *Neurology* 1995; **45**: 1648-1654.
- 4 Barkana Y, Levin N, Goldhammer Y, Steiner I. Chronic intracranial hypertension with unexplained cerebrospinal fluid pleocytosis. *J Neuro-Ophthalmol* 2004; **24**: 106-108.
- 5 Masjuan J, Buisan J, Frutos T, Quereda C, Alvarez-Cermeno JC. The not so benign syndrome of transient headache with neurologic deficits and CSF lymphocytosis. *Neurology* 1998; **51**: 313-314.
- 6 Waterschoot MP, Guerit JM, Lambert M, de Barys T. Bilateral tonic pupils and polyneuropathy in Sjogren's

syndrome: a common pathophysiological mechanism? *Eur Neurol* 1991; **31**: 114–116.

- 7 Victor DI, Green WR, Stark WJ, Walsh FB. A non-permanent tonic pupil in rheumatoid arteritis. *Can J Neurol Sci* 1977; **4**: 209–212.
- 8 Levy NS, Kramer SG, de Barros T. Pupillary and accommodative abnormalities in the Vogt–Koyanagi–Harada syndrome. *Am J Ophthalmol* 1970; **69**: 582–588.
- 9 Jean D, Maes E. Hyperthyroidism, ophthalmoplegia and unilateral mydriasis. *Bull Soc Belge Ophthalmol* 1992; **245**: 29–32.
- 10 Stern BJ, Gruen R, Koeppel J, Aronson N, Krumholz A. Recurrent thyrotoxicosis and papilledema in a patient with communicating hydrocephalus. *Arch Neurol* 1984; **41**: 65–67.

K Shikishima<sup>1</sup>, K Kitahara<sup>1</sup> and K Inoue<sup>2</sup>

<sup>1</sup>Department of Ophthalmology, Jikei University School of Medicine, Tokyo, Japan

<sup>2</sup>Department of Neurology, Jikei University School of Medicine, Tokyo, Japan

Correspondence: K Shikishima, Department of Ophthalmology, Jikei University School of Medicine, 3-25-8, Nishi-shinbashi, Minato-ku, Tokyo 105-8461, Japan  
Tel: +81 3 3433 1111 ext. 3581;  
Fax: +81 3 3433 1936.  
E-mail: shiki@jikei.ac.jp

The authors have no commercial or proprietary interest in any material mentioned in this study. The authors also have received no financial support nor have any relationships with any entity with regard to the material presented in this study.

Part of this report was presented in July of 2004 at the 15th International Neuro-Ophthalmological Society Meeting in Geneva, Switzerland.

*Eye* (2006) **20**, 613–615. doi:10.1038/sj.eye.6701937; published online 27 May 2005

Sir,

### **Combined enucleation and orbitotomy for choroidal melanoma with orbital extension**

Extrascleral extension of uveal melanoma is observed in about 8% of cases and represents a poor prognostic factor.<sup>1,2</sup> Management of orbital melanoma is controversial. In general, small nodular extrascleral extensions noted incidentally on ultrasonography are managed by modifying enucleation so as to avoid transection of the area of involvement.<sup>3</sup> Beneficial role of

adjuvant radiotherapy in such cases remains questionable.<sup>3</sup> Diffuse orbital involvement is best managed by exenteration, although long-term survival advantage offered by this procedure is not known.<sup>4–8</sup> Larger but circumscribed orbital involvement may be excised via orbitotomy in conjunction with enucleation or proton beam radiotherapy.<sup>9,10</sup>

We report a case of slow growing choroidal melanoma that led to a circumscribed intraconal orbital extension that was managed successfully by a combined approach of enucleation and orbitotomy.

### **Case report**

A 76-year-old man presented with reduced vision of 3 years duration of the left eye in September, 1990. The past ocular history, personal, and family history was noncontributory. Visual acuity was 6/5 in the right eye and HM in the left eye. The right eye was normal. Left eye had normal anterior segment. Fundus evaluation showed a diffuse melanocytic choroidal thickening estimated to be 9 mm in base and 3 mm in thickness in the macular region (Figure 1a and b). Associated overlying RPE changes and subtotal inferior retinal detachment was also present. A diffuse choroidal melanoma was diagnosed. Patient refused all forms of treatment but agreed for periodic observation. The lesion continued to grow slowly with development of extrascleral extension and iris neovascularization and neovascular glaucoma in February, 1999 (Figure 1c). As the eye was not painful, patient refused enucleation. In February 2003, there was onset of proptosis with enlargement of orbital component of the melanoma (Figure 1d). Systemic evaluation was negative for hepatic metastasis. Once the patient was made aware of the possibility of orbital exenteration in the near future, he agreed to undergo enucleation combined with orbitotomy so as to avoid disfigurement associated with exenteration.

The surgery was performed under general anaesthetic. The initial surgical steps were of enucleation. Following disinsertion of all muscles, silk traction sutures were applied to the insertion of the recti muscles (Figure 1e). Superotemporal orbitomy was then performed through an upper eyelid crease incision (Figure 1f). Access to the intraconal orbit was gained by incising lateral orbital rim. The orbital melanoma could be palpated and it was isolated using blunt dissection. A long stump of optic nerve was cut and globe was removed with the orbital involvement. After haemostasis was achieved the orbitomy and enucleation wounds were closed with absorbable sutures (Figure 1g). There were no operative or postoperative complications. Ocular prosthesis was fitted 6 weeks postoperatively. Excellent cosmetic outcome could be achieved (Figure 1h). Patient continued to do well 1 year after the procedure.