# LETTERS TO THE JOURNAL

Sir,

**HTLV-I Associated Uveitis: A Report of Two Cases**

We report two patients with acute anterior uveitis associated with HTLV-I infection. One patient developed HTLV-I associated myelopathy (HAM) 3 months after the development of uveitis, characterised by a slowly progressive paraparesis, bladder dysfunction and constipation. The second patient had a history of anterior uveitis and was found to be HTLV-I positive at routine screening following the death of her daughter from HTLV-I associated adult T-cell leukaemia. We believe these to be the first reported cases of HTLV-I associated anterior uveitis in the United Kingdom.

*Case Reports*

*Case 1*

A 51-year-old Afro-Caribbean man presented to the casualty department at the Western Eye Hospital in February 1994 complaining of decreased vision and seeing floaters in the right eye. He had been resident in the United Kingdom since 1961 and 1 month prior to presentation had been on holiday in St Lucia. He had no past eye history and was systemically well. Examination revealed corrected Snellen visual acuities of 6/6 (R) and 6/5 (L). Fine granulomatous keratic precipitates were present in both eyes scattered throughout the corneal endothelia. There was 1+ of cells and 1+ of flare in both anterior chambers. Intraocular pressures were 10 mmHg (R) and 8 mmHg (L) and dilated fundoscopy revealed no abnormality. A diagnosis of mild granulomatous anterior uveitis was made and he was treated conservatively.

At routine follow-up a month later, Snellen visual acuities were 6/12 (R) and 6/6 (L). Large mutton fat keratic precipitates were now present in both eyes but there was no increase in the cellular activity in the anterior chambers. A few cells were present in the right anterior vitreous. Full blood count, erythrocyte sedimentation rate (ESR), serum angiotensin converting enzyme and treponemal serology were all normal and a plain chest radiograph revealed no abnormality. He was started on guttae betamethasone t.d.s. to both eyes. A month later at a routine clinic visit the visual acuities were 6/6 in both eyes. The keratic precipitates had resolved and no abnormalities were found in the anterior or posterior chambers. The course of topical steroid drops was reduced over 4 weeks and he was discharged from the clinic.

The first symptoms of HAM developed 3 months later with urinary retention, constipation and progressive paraparesis. Human T-cell leukaemia/lymphoma virus type I/II infection was diagnosed by the detection of serum and cerebrospinal fluid anti-HTLV antibodies in high titre by Fujirebio Serodia HTLV, and this was confirmed and serotyped as HTLV-I by Genelabs HTLV 2.3 WB. HTLV-I/II specific sequences from the HTLV-I *tax* gene were detected in peripheral blood lymphocytes following amplification by polymerase chain reaction primed with the oligonucleotides SK43/44. He had no further episodes of ocular inflammation until his death from HAM in July 1995.

*Case 2*

A 66-year-old Afro-Caribbean woman presented to the casualty department in January 1995 complaining of a painless red right eye with no visual disturbance. She had been resident in the United Kingdom since 1957 but had lived in the United States between 1975 and 1993. She had suffered a similar episode in the United States 4 years previously and had responded well to treatment with topical steroid drops. She was systemically well. She had tested positive for HTLV-I at routine screening following the death of her daughter from adult T-cell leukaemia in December 1993.

On examination, Snellen visual acuities were 6/9 (R and L). There was a mild right ciliary injection and 1+ of cells and flare in the right anterior chamber. Early nuclear sclerotic lens opacities were noted. No abnormalities were found in the vitreous or retina. A diagnosis of mild acute right anterior uveitis was made. She was treated with topical guttae betamethasone q.d.s. and guttae cyclopentolate t.d.s. to the right eye. Full blood count, ESR, serum

 

angiotensin converting enzyme and treponemal serology were all normal and a chest radiograph revealed no abnormality. Two weeks later the uveitis had cleared and the drops were reduced over 2 weeks. She had suffered no similar episodes 9 months later.

*Discussion*

HTLV-1 infection is strongly linked to the subsequent development of adult T-cell leukaemia/lymphoma and HTLV-I associated myelopathy (HAM), which is also referred to as tropical spastic paraparesis (TSP). It is also associated with other inflammatory conditions but causes asymptomatic lifelong infection in the majority of individuals.[1–3] Ocular manifestations associated with HTLV-I infection have been documented, particularly in Japan, and include a steroid-resistant vasculitis[4] and a granulomatous or non-granulomatous uveitis.[5] However, the exact role of HTLV-I in the pathogenesis of these disorders remains uncertain.[6]

In keeping with previous reports, our patients' anterior uveitides resolved completely with topical steroids. However, the development in case 1 of HAM 3 months after the initial presentation is in contrast with the results of a recent study of 32 patients with HTLV-I associated uveitis in which no patient developed neurological disease. Anterior uveitis has been described in patients with *pre-existent* HAM/TSP.[7]

HTLV-I is endemic in Japan, the Caribbean, Melanesia, Central Africa and parts of Central and South America, and in peoples who have migrated from these regions. Thus amongst the Afro-Caribbean community in the United Kingdom, the prevalence of HTLV-I is 2%,[8] although its prevalence in those with uveitis is unknown. The infection may be acquired by vertical transmission, as in case 2, and this emphasises the importance of a family history in the diagnosis and counselling of patients found to be HTLV-I positive.

Although the cause of uveitis in these two cases was ascribed to HTLV-I infection without intraocular biopsy, they highlight the need to consider HTLV-I infection in patients belonging to at-risk groups who present with uveitis of unknown aetiology, and the need for further studies into the prevalence of positive HTLV-I serology in such patients.

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Sir,

**Corneal Deposits and Penetrating Keratoplasty in a Patient with Hyperparathyroidism, Hyperlipidaemia and Multiple Myeloma**

Calcium deposition in the cornea is usually the result of long-standing ocular disease but may also be associated with underlying hypercalcaemia. Occasionally, corneal deposits may be the presenting sign of an underlying disorder of calcium and phosphate metabolism which may occur with hyperparathyroidism, sarcoidosis, chronic renal failure and hypervitaminosis D.[1,2]

Lipid deposition occurs in scarred vascular corneas; however, primary lipid keratopathy may be a manifestation of underlying hyperlipidaemia.[3,4] Crystalline corneal deposits have been described in association with multiple myeloma[5] and streptococcal infections of the cornea.[6]

A case in which corneal deposits were the presenting sign of both hyperparathyroidism and hyperlipidaemia is discussed. The recurrence and