Short report

Bell's palsy and HIV infection

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SUMMARY Unilateral infranuclear facial palsy developed in three young homosexual men. All three were positive for antibodies to human immunodeficiency virus (HIV). Two had persistent generalised lymphadenopathy, but the clinical criteria for the acquired immune deficiency syndrome (AIDS) were not fulfilled. There were no features of generalised neuropathy, and no other cause for facial palsy was evident. Recovery was excellent in each patient.

Bell's palsy is a common disorder, of unknown cause, sometimes associated with upper respiratory tract infection, with *Herpes zoster* or *Herpes simplex* infection of the facial ganglion, with other viral infections, or with diabetes mellitus.¹ Unilateral facial palsy may also occur secondary to trauma, otitis media, middle ear surgery, meningitis, sarcoidosis or with infiltration of the nerve by tumour. Recovery is complete in 50% of all cases and in 80% of young people with idiopathic Bell's palsy.² We report three young homosexual men in whom unilateral infranuclear facial palsy, in one consistent with the clinical syndrome of idiopathic Bell's palsy, and in two possibly related to *H zoster* infection, was found to be associated with HIV infection.

Case 1 A 26 year old single man presented with left facial weakness, of less than 6 hours duration, associated with subjective numbness of the left cheek. A month previously he had had chicken pox and for 18 months had noted lassitude and night sweats. Examination showed a complete left infranuclear facial palsy but no other abnormality. The sexual history revealed homosexual experience from the age of 19 years. He had practised insertive and receptive peno-anal intercourse, with 12 life-time partners, one of whom had also had intercourse in San Francisco. In the past he had had anal *H simplex* infection, and gonococcal proctitis. The CT head scan and chest radiographs were normal. The CSF contained 0.45 g protein/l and 5 lymphocytes/mm³. Link's IgG index was raised at 1.05 (normal <0.58) and the albumin index was 0.0078 (normal <0.0057). The CSF IgG was not

oligoclonal. The total blood leucocyte count was 4.3/nl (lymphocytes 54%). The lymphocytes were 53% T cells, with 25% helper T4 (normal 40-60%) and 47% suppressor T8 (normal 20-40%), giving a T4/T8 ratio of 0.53 (normal >1). HIV antibodies were detected by immunofluorescence and by ELISA in venous blood. The facial palsy recovered completely in 4 months.

Case 2 This 29 year old homosexual man presented with a complete left facial palsy that had developed overnight without other neurological symptoms. There were herpetic vesicles on the hard palate, with generalised lymphadenopathy. At this time the titre of antibodies to *Varicella zoster* rose from negative to 1:128, although there was a history of chicken pox as a child. The venous leucocyte count was 6.4/nl (40% lymphocytes). T cells comprised 36% of these lymphocytes; T4 20%, T8 15% (T4/T8 ratio 1.3). The HIV antibody test was found to be positive. He had practised insertive and receptive peno-anal intercourse during the previous 3 years, with 10 partners during the previous year. He had been treated for gonococcal and non-gonococcal proctitis and urethritis, and for hepatitis B in the previous year, and subsequently developed rubella, sinusitis, scabies, a penile ulcer and oral moniliasis. The facial palsy recovered completely in 5 months.

Case 3 A 46 year old male bisexual psychiatric nurse awoke with a left sided infranuclear facial palsy, with hyperacusis and with some discomfort in the region of the left ear. There were no other neurological abnormalities. He had been found to be HIV antibody positive 2 years previously, with persistent generalised lymphadenopathy, and had been treated for recurrent urinary tract infections during this time. Recovery was complete in 3 weeks.

Discussion

These three homosexual men presented with uni-

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lateral infranuclear facial palsy, two indirectly associated with *Varicella zoster* infection, and one without evident cause. The clinical features in each case were those of idiopathic Bell's palsy. All had positive HIV antibody tests, and two had persistent generalised lymphadenopathy. There were no signs of generalised neuropathy, and recovery from the facial palsy was complete.

Cranial neuropathies, including facial palsy, have been recognised in homosexual men with AIDS,^{3,4} often in association with symmetrical polyneuropathy. These neuropathies may develop in the few months after HIV infection or as part of the more chronic illness that develops with AIDS itself. In case 1 there was evidence of disturbance of the blood/brain barrier, as shown by the Link's and albumin indices, and in this patient the lymphocyte count in the CSF was at the upper limit of normal. The facial palsy in case 1 followed chicken pox, but these disorders are not known to be associated, and it is equally possible that the Bell's palsy in this patient occurred as a result of neurotropic infection of the facial nerve or facial ganglion by recently acquired HIV infection.⁵ In cases 2 and 3, Bell's palsy devel-

oped in the context of known HIV infection and with clinical and serological evidence of infection with *H Zoster*, although the latter was not in anatomical relation to the facial nerve itself. The possibility that Bell's palsy may be a feature of HIV infection, especially in young homosexual men, should be recognised.

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