Letters

a Wright-Codoc ataxiometer. A detailed description of the tasks is given in.⁸

All scores on the WAIS subtests were significantly lower compared with standard norms and to a control group (see table). His score on the Ravens Matrices placed AB at the lower end of the "intellectual average" group: assessment on the Matrices was done 12 months after the initial referral.

The control group consisted of 12 prisoners with a similar socioeconomic and criminal background matched on age and IQ who had no history of solvent abuse. Controls were given four assessments over a period of 1 year. Since they did not show any difference in performance between the third and fourth assessment, it was assumed that no further improvement due to practice would occur and testing was discontinued. AB was followed up at 3 monthly intervals over a period of 18 months. He gradually recovered to normal functioning on seven tasks, but after 18 months was still impaired on two tasks. Scores on word recognition had improved considerably but he still showed a sizeable difference from the control group.

After 18 months AB was given a standard neurological examination, performed by a visiting neurologist, which showed no signs of any residual focal or peripheral abnormalities. By this time we were also able to carry out magnetic resonance imaging which unfortunately was not obtainable at initial presentation. Imaging was performed on a Picker "Vista 1100" 0.15 tesla resistive MR System. MRI findings suggested minimal bilateral occipital and cerebellar atrophy but no discrete focal lesions were observed and the ventricular system appeared normal.

The study suggests that neuropsychological deficits can be detected in chronic solvent abusers after a period well beyond the acute effects of solvent use or withdrawal. However, even after extensive solvent abuse recovery is possible to a considerable degree, although after 18 months of abstinence normal functioning is still not fully reinstated in the present case. This is consistent with previous studies reporting permanent neurological and psychological damage in solvent abusers.⁹⁻¹¹ The nature of the recovery process and the extent of permanent damage as a consequence of solvent abuse are not yet understood and demand further investigation.

KLAUS D WIEDMANN*

KEVIN G POWER*

JT LINDSAY WILSON*

DM HADLEY†

*Department of Psychology,
University of Stirling,

Stirling,

FK9 4LA, UK

†Institute of Neurological Sciences,

MRI Unit,

Glasgow,

Scotland.

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Acute cerebellar syndrome complicating infectious mononucleosis

Sir: Neurological complications of primary Epstein-Barr virus (EBV) infection occur in up to 1% of cases^{1,2} and include meningo-encephalitis, aseptic meningitis, optic neuritis, facial palsy, transverse myelitis and polyneuritis.¹⁻⁸

We report the occurrence of a recognisable acute cerebellar syndrome due to infectious mononucleosis in two 15 year old

teenagers, one of whom suffered permanent, though mild, sequelae.

The pathogenesis of cerebellar involvement, which is infrequent and benign, with complete recovery in all reported cases,^{2,4-6} still requires elucidation. Direct viral replication seems most likely but post-infectious demyelination has been postulated in some reports.^{7,9}

A fifteen year old schoolgirl presented in May 1982 with a 2 week history of sore throat, malaise, abdominal pain, vomiting and generalised headaches. Four days prior to admission she became unsteady on her feet and noted horizontal diplopia, particularly on left lateral gaze. Examination revealed an alert and orientated girl who was afebrile. The pharynx appeared normal and apart from mild left supraclavicular fossa lymphadenopathy, general examination was normal. Neurological examination disclosed saccadic ocular pursuit, horizontal nystagmus and horizontal diplopia on lateral gaze to the right and left, and first degree downbeating nystagmus. A mild left lower motor neuron facial palsy and bilateral limb and truncal ataxia were present. The haemoglobin level was 144 g/l and the white cell count was $4.4 \times 10^9/l$. The neutrophil count was $1.32 \times 10^9/l$ (normal 2.0-7.5) and the lymphocyte count was $2.46 \times 10^9/l$ (normal range 1.5-4.0) with an occasional reactive lymphocyte. The differential slide agglutination test (Paul Bunnell-Davidson) for infectious mononucleosis was positive. Cerebrospinal fluid (CSF) examination revealed 18 leucocytes/ μl (95% lymphocytes, 5% neutrophils) and 0 red cells/ μl . No bacteria were seen. CSF glucose was 3.2 mmol/l (normal 2.8-4.4) and protein 0.22 g/l (normal 0.15-0.45). CSF IgG/albumin ratio was 0.8 (normal 0.04-0.24). Brain stem auditory evoked potentials were normal bilaterally.

She was managed conservatively and at 1 month follow-up had improved symptomatically. She still manifested gaze evoked horizontal nystagmus on lateral gaze bilaterally, upbeating nystagmus on upward gaze, downbeating nystagmus on downward gaze, mild limb ataxia and moderate truncal ataxia. At 6 months follow-up her physical signs were unchanged. At 5 years follow-up she was asymptomatic apart from slight horizontal diplopia on horizontal gaze bilaterally and an inability to run effectively. Examination revealed gaze evoked horizontal nystagmus on lateral gaze bilaterally, first degree upbeating nystagmus and first degree downbeating nystagmus. Very mild limb and truncal ataxia persisted.

The second case was a 15 year old schoolboy who presented in January 1987 with a 3

day history of slurred speech, difficulty with balance, dizziness and vomiting. Fourteen days previously he had felt unwell and developed a sore throat which had not responded to three injections of penicillin by his general practitioner, but had resolved as his neurological symptoms began. On examination he was mildly febrile (temperature 37.3°C) and alert and co-operative, but vertigo and vomiting, exacerbated by positional change, made him reluctant to move from the supine position. A fine macular rash was present over the trunk and the pharyngeal fauces were slightly injected. The cervical lymph nodes were palpable but lymphadenopathy and splenomegaly were not present. Kernig's sign was negative and there was no neck stiffness. The patient spoke with a slurring dysarthria. In the primary position of ocular gaze, fixational instability was present as demonstrated by irregular macrosaccadic oscillations. Horizontal and vertical ocular pursuit were interrupted by saccadic intrusions and gaze evoked horizontal nystagmus was present on lateral gaze to the right and, less so to the left. Moderate limb and truncal ataxia were present but the remainder of the neurological examination was normal. Plain cranial CT scan was normal and CSF examination revealed 2 mononuclear cells/ μ l, 0 red cells, protein 0.38 g/l, glucose 3.7 mmol/l and IgG/albumin ratio 0.16. Simultaneous plasma glucose was 6.3 mmol/l. CSF bacterial and viral cultures were negative. CSF cytomegalovirus and herpes simplex complement fixation test titres were negative and CSF EBV IgG fluorescent antibody and EBV IgM fluorescent antibody tests were negative. Plasma EBV IgG fluorescent antibody and IgM fluorescent antibody tests were positive indicating recent Epstein-Barr virus infection. Serum cytomegalovirus IgM (by ELISA) was not detected and CMV IgG (ELISA units) was 6 (not detected). The complete blood picture was within normal limits with a normal differential white cell count, no atypical cells and a normal erythrocyte sedimentation rate. The differential slide agglutination test (Monospot) was positive. The patient received intravenous acyclovir 10 mg/kg of body weight three times a day for 4 days. He remained afebrile after presentation and his nystagmus resolved on day three. Vomiting and vertigo persisted for the first week despite metoclopramide and prochlorperazine. Improvement was gradual and at 4 weeks after presentation the eye and limb movements were normal but minimal truncal ataxia persisted.

These two cases developed symptoms and signs of neurological dysfunction approximately 10 days following the onset of sore

throat and systemic upset due to EBV infection. Neurological involvement was not confined to the cerebellum in either case. The presence of bilateral lateral gaze palsies and a left lower motor neuron facial palsy in the first case suggested pontomedullary tegmental involvement and the first degree vertical nystagmus could be explained by involvement of the vestibulocerebellar connections. Vestibular involvement was manifested in the second case as severe vertigo.

The diagnosis of EBV infection in the first case was clinical and the Paul-Bunnell-Davidson test was positive, giving strong presumptive evidence of infectious mononucleosis.⁷ In addition, in the second case the serum IgM and IgG fluorescent antibody to EBV was positive.

Although central nervous system involvement may be the sole or major manifestation of infectious mononucleosis^{1,8} it most commonly occurs at the height of the systemic manifestations of the infection.⁴ The latent interval of 10 days between systemic and neurological dysfunction in our two cases suggested that a delayed antigen-antibody reaction may be involved in the pathogenesis. In some necropsy studies of fatal cases of infectious mononucleosis encephalitis, the presence of perivenous white matter demyelination has promoted the suggestion that a post-infectious encephalomyelitis is responsible for the neurological involvement.^{7,9} If this was so, we may have expected other evidence of demyelination, such as long tract signs in our cases but in neither case were the descending ventral brainstem pathways and ascending sensory pathways of the brainstem tegmentum affected.

Antibodies to EBV-viral capsid antigen (EBV-VCA) have been demonstrated in the CSF in two patients with mononucleosis-associated acute cerebellar syndrome² and a few cases of meningoencephalitis.^{10,11} Subsequently, virus¹² and lymphoid cells harbouring EBV¹¹ have been demonstrated in the CSF in mononucleosis-associated meningoencephalitis, suggesting that virus-altered B-lymphocytes can invade the CNS and may play a direct role in the pathogenesis of neurological sequelae of EBV infection.

Although acyclovir has been shown to inhibit oropharyngeal shedding of EBV (during the period of acyclovir administration),¹³ it does not have an established role in the treatment of EBV infection. Its use in the second case may not have affected the outcome.

We report these two cases of what has come to be recognised as an acute cerebellar syndrome complicating infectious mono-

nucleosis to increase awareness of this uncommon disorder of uncertain pathogenesis. Although the prognosis is generally favourable, the possibility of permanent neurological sequelae exists.

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DR. GRAEME J HANKEY

DR. MERCY SADKA

DR. ROSS S GOODHEART

DR. DAVID M HOFFMAN

Royal Perth Hospital

Box X2213, GPO, Perth

Western Australia 6001

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