

# PostScript

## LETTERS

### Medial medullary infarction with contralateral glossoplegia

There is a relative lack of information on the course of corticohypoglossal projections in the human brain stem,<sup>1,2</sup> especially in the medulla. Using transcranial magnetic stimulation and magnetic resonance imaging (MRI) in 11 patients with ischaemic lesions at different brain stem levels, Urban *et al* showed that pontine lesions at the ventral paramedian base close to the midline affected the contralateral corticohypoglossal projections, while lateral lesions at the pontine base affected the ipsilateral projections. Lesions of the dorsolateral and mediolateral medulla impaired only ipsilateral corticohypoglossal projections.<sup>3</sup>

We report a patient with contralateral glossoplegia in the ventromedial lesion of the upper medulla.

#### Case report

A 52 year old hypertensive man suddenly developed right sided paraesthesiae distal to the elbow and knee. The next day he had right hemiparesis. On neurological

examination, he was found to have left beating nystagmus on left lateral gaze. Unilateral tongue paresis was demonstrated by voluntary protrusion, during which the tip of the tongue deviated to the right (fig 1A). When the tongue was not protruded, the right half of the tongue base was seen to bulge. Neither atrophy nor fasciculation was observed in the tongue. There were no abnormalities on tongue electromyography. He had mild right hemiparesis, mildly diminished sensation in all modes, which gradually worsened, and truncal lateropulsion to the right. Brain MRI, done three days after symptom onset, showed an infarct in the ventromedial portion of the left rostral medulla extending deep into the dorsal portion (fig 1B and 1C). The paraesthesiae and sensory deficits disappeared spontaneously in two weeks, and the limb weakness and the glossoplegia gradually improved, with complete resolution two months after stroke onset.

#### Comment

This patient's glossoplegia was a supranuclear palsy because the tongue deviated to the contralateral side of the lesion and neither atrophy nor fasciculation was observed. Protrusion of the tongue is accomplished by the unopposed action of the normal contralateral genioglossus. The corticobulbar fibres

controlling the genioglossus muscles are crossed; the other tongue muscles appear to have bilateral supranuclear control.<sup>2,4</sup> With regard to the supranuclear projections to the hypoglossal nucleus in the brain stem, it is still unclear at what level they decussate over the midline to reach the contralateral nucleus. Urban *et al* showed that lesions of the ventral pontine base located close to the midline only impair the contralateral corticohypoglossal projections, while lesions extending to the lateral part of the basis pontis and dorsolateral lesions of the upper medulla near the pontine border affect the ipsilateral projections. They suggested that the main decussation of these fibres is located close to the pontomedullary junction.<sup>3</sup>

In this patient with contralateral supranuclear glossoplegia, the lesion was located on the ventromedial part of the rostral medulla. This finding shows that the corticohypoglossal projections in this patient are decussated at the upper medullary level (fig 1D), more caudally than the pontomedullary junction described by Urban *et al*.<sup>3</sup>

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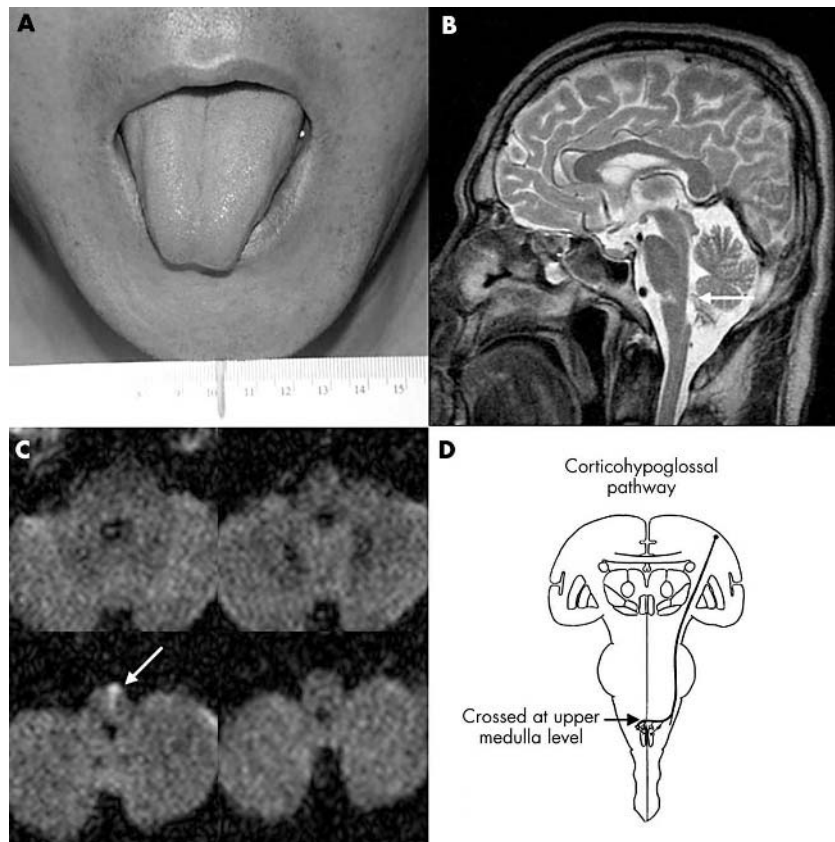
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### Recurrent hypertensive brainstem encephalopathy

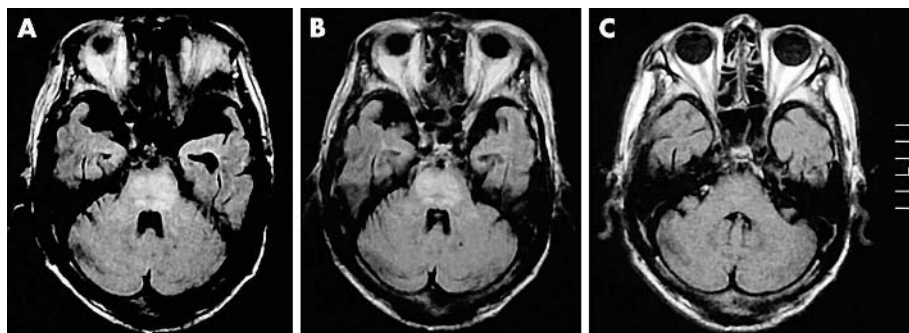
In hypertensive encephalopathy (HE), computerized tomography or magnetic resonance (MR) images usually exhibit predominantly posterior white matter involvement, similar to reversible posterior leukoencephalopathy syndrome (RPLS).<sup>1,2</sup> Recurrent HE is rare.<sup>3</sup> Here, we report a rare case of recurrent HE.

#### Case report

A 75 year old man with an unremarkable medical history presented with generalized convulsion and stupor following a headache in June 2003. On admission, his blood pressure (BP) was 200/120 mmHg. The T2 weighted MR images (T2WI) and fluid attenuated inversion recovery (FLAIR) images revealed hyperintense lesions and swelling in the brainstem (fig 1). There was no enhancement of these lesions after an



**Figure 1** (A) The protruded tongue deviating to the right about 1.5 cm from the midline. Magnetic resonance imaging (T2 sagittal (B) and diffusion weighted (C)) showed an acute unifocal ischaemic lesion in the left ventromedial part of the upper medulla. The schematic drawing (D) shows the proposed corticohypoglossal decussation at the upper medulla level.



**Figure 1** Fluid attenuated inversion recovery (FLAIR) images of representative levels of the pons. (A) On the first admission, the FLAIR images revealed hyperintense lesions and swelling. After blood pressure (BP) decreased, those findings disappeared spontaneously. (B) One year after the first admission, the FLAIR images revealed recurrent hyperintense lesions similar to those observed in the initial image. (C) One month later, after BP was found to be normotensive, the images revealed no abnormal findings.

injection of contrast media. Electroencephalography demonstrated no electric potentials of an epileptic discharge. Blood and cerebrospinal fluid analyses revealed normal findings without elevated levels of serum creatinine (18.0 mg/l) and urea nitrogen (200 mg/l). We treated the patient with antihypertensive drugs, after we noted that a decrease in his BP resulted in the rapid improvement of symptoms and the findings of T2WI and FLAIR images. One year later, he had a relapse of generalized convulsion. His BP was 230/100 mmHg. A fundoscopic examination of the eyes revealed retinal hypertensive atherosclerosis with Keith-Wagener II changes. T2WI and FLAIR images exhibited recurrent hyperintense lesions in the brainstem (fig 1), accompanied by slight hyperintense changes in the bilateral occipital lobes. The serum creatinine level was 21 mg/l, and the urea nitrogen level was 270 mg/l. The creatinine clearance rate was 20.6 ml/min. A hormonal survey revealed a plasma aldosterone concentration of less than 1.0 pg/ml because of secretory suppression caused by severe hypertension. The levels of other hormones, such as urinary and blood catecholamines, urinary vanillyl-mandelic acid (VMA), 17-hydroxycorticosteroids, 17-ketosteroids, and plasma renin activity were within normal ranges: urinary noradrenaline 158.7 µg/day (normal range (NR), 48.6 to 168.4), plasma noradrenaline 0.15 ng/ml (NR 0.07 to 0.31), urinary adrenaline 5.6 µg/day (NR 3.4 to 26.9), plasma adrenaline <0.05 ng/ml (NR <0.10), urinary dopamine 378.8 µg/day (NR 365.0 to 961.5), plasma dopamine <0.10 ng/ml (NR <0.10), and urinary VMA 3.3 mg/day (NR 1.5 to 4.3). Abdominal MR angiography revealed the occlusion of the left renal artery and the stenosis of the right renal artery. Furthermore, abdominal MR images revealed that the left kidney was atrophied, but did not reveal adrenal tumour. Thus, we diagnosed the patient as having recurrent hypertensive brainstem encephalopathy because of renal vascular hypertension and renal dysfunction. After adequate antihypertensive treatment, his symptoms immediately improved and the MR images exhibited normal findings (fig 1). By January 2005, he did not exhibit any recurrent symptoms.

## Discussion

Our patient presented with headache and generalized convulsion associated with marked hypertension. MR imaging revealed brainstem hyperintense lesions on T2WI and

FLAIR images. The prompt initiation and adequate administration of antihypertensive treatment led to a complete clinical recovery, with the disappearance of lesions on MR images. However, the patient's symptoms and abnormal findings on MR images were recurrent, a feature that has been rarely reported to date.

HE and RPLS are disorders that have been similarly categorized based on their symptoms and imaging findings. They are acute disorders that occur in patients with marked hypertension associated with central nervous system (CNS) symptoms, such as headache, convulsion, and altered mental status.<sup>1</sup> HE typically involves the posterior parieto-occipital lobes accompanied by some degree of brainstem and cerebellar involvements. However, HE has recently been reported to have a predominant involvement of the brainstem with minimal changes in the supratentorial structure.<sup>2</sup> The clinical diagnosis of hypertensive brainstem encephalopathy is difficult. The pontine T2W hyperintense lesion observed in our patient indicates glioma, infarction, postinfectious encephalomyelitis, radiation changes, or central pontine myelinolysis. A previous study suggested that retinal hypertensive signs are important for identifying the CNS manifestations of HE. The signs also allow the differentiation of HE from brainstem tumour.<sup>2</sup> Initially, we provisionally diagnosed the patient as having one of these conditions on the basis of his clinical symptoms. However, by simply decreasing his BP, the imaging findings became normal. Moreover, retinal examination demonstrated hypertensive changes, which are diagnostic features of hypertensive brainstem encephalopathy.

The proposed mechanism underlying HE involves the breakdown of autoregulation, resulting in the dilatation of cerebral arterioles, the disruption of the blood-brain barrier, and the breakthrough accumulation causing vasogenic oedema.<sup>1,2</sup> As the vertebrobasilar system and posterior cerebral arteries are sparsely innervated by sympathetic nerves,<sup>4</sup> the occipital lobes and other posterior brain regions may be particularly susceptible to the breakdown of autoregulation. In RPLS, most patients have hypertension as the precipitating factor, with comorbidities such as renal failure and numerous medications.<sup>1</sup> In a patient with pheochromocytoma, it was suggested that direct catecholamine toxicity might also have a role in the generation of brain lesions and vasospasm.<sup>2</sup> The levels of catecholamines in

our case were within normal ranges; therefore, we ruled out the diagnosis of pheochromocytoma. A previously reported case of a patient with recurrent posterior reversible encephalopathy syndrome had an unknown aetiology, and was accompanied by only mild HT.<sup>3</sup> Our patient may have developed susceptibility affected posterior circulation modified by renal failure. His recurrent HE might have been caused by a marked hypertension and renal dysfunction and his susceptibility to changes in BP, although we cannot rule out the possibility of the influence of other catecholamines that have not been evaluated.

In most patients with hypertension, renal stenosis is detected by angiography. In the chronic course of renal vascular hypertension, renal vascular changes lead to an acute increase in BP spontaneously. Renal vascular hypertension is sometimes diagnosed in hypertensive crisis accompanied by hypertensive encephalopathy.<sup>3</sup> In our patient, percutaneous intervention and surgical treatment were not performed because of severe renal failure. However, clinicians have to suspect this possibility in renal vascular hypertension, when patients present with hypertension, to prevent renal vascular changes and control hypertension similar to our present patient.

In conclusion, we suggest that there are patients susceptible to an increase in BP and recurrent HE such as our present patient, and that doctors must make a prompt diagnosis and provide immediate treatment, even if the actual aetiology of recurrent HE is not evident.

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## Diffuse cystic leucoencephalopathy after buprenorphine injection

Buprenorphine has been prescribed for opioid detoxification and subsequent maintenance therapy with favourable outcomes. Its high affinity for the mu receptor, higher analgesic potency than morphine, antagonistic effects at higher doses as a partial agonist, lower incidence of physical dependence, and mild withdrawal symptoms confer qualities that make it advantageous for the treatment of opioid dependence.<sup>1</sup> Buprenorphine, marketed under the trade name Subutex, may be administered sublingually, subcutaneously, or intravenously. We describe a severe neurological effect of intravenous buprenorphine exposure that resulted in diffuse cystic leucoencephalopathy.

### Case report

An 18 year old man was found unconscious in his bedroom by his girlfriend. He had no past history, and no family history of neurological or metabolic diseases. He was brought to the emergency room febrile, comatose and in severe respiratory failure (pH 7.399, pO<sub>2</sub> 84 mmHg, pCO<sub>2</sub> 42 mmHg, bicarbonate 24.9 mmol/L, on 100% non-rebreathing mask). He was intubated and treated presumptively for pneumonia with

intravenous imipenam and azithromycin. He did not require inotropic support. Physical examination revealed a drowsy and lethargic man, who had bilateral basal crepitations in the lungs. Apart from midrange pupils that were sluggishly reactive to light, the neurological examination was unremarkable. There was no evidence of external bruises, injuries or obvious needle track marks over his arms. Initial investigations were significant for polymorphic leucocytosis (total white cell count,  $8.08 \times 10^9/L$ ; polymorphonuclear leucocytes, 75%) and rhabdomyolysis (creatinine kinase, 8900 U/L; creatinine, 189  $\mu\text{mol/L}$ ). Serum and urine toxicology screen were positive for benzodiazepines.

Microbiological analysis of blood, urine, cerebrospinal fluid (CSF) and sputum samples did not yield any pathogens. Serology for *Mycoplasma*, *Leptospira*, and *Rickettsia* species, and influenza and Epstein-Barr viruses was negative. The patient's condition improved and he was extubated 2 days later. Bilateral pyramidal dysfunction, with spastic quadriplegia, hypertonia, hyper-reflexia, sustained clonus and extensor plantar responses were noted 5 days later. Pupils remained midrange and slowly reactive to light. He did not demonstrate any cranial neuropathies or cerebellar signs. Although bedside cognitive assessment was within normal limits, he was bradyphrenic. He did not manifest myoclonic jerking. Vibratory, proprioceptive, temperature and pinprick sensation were normal.

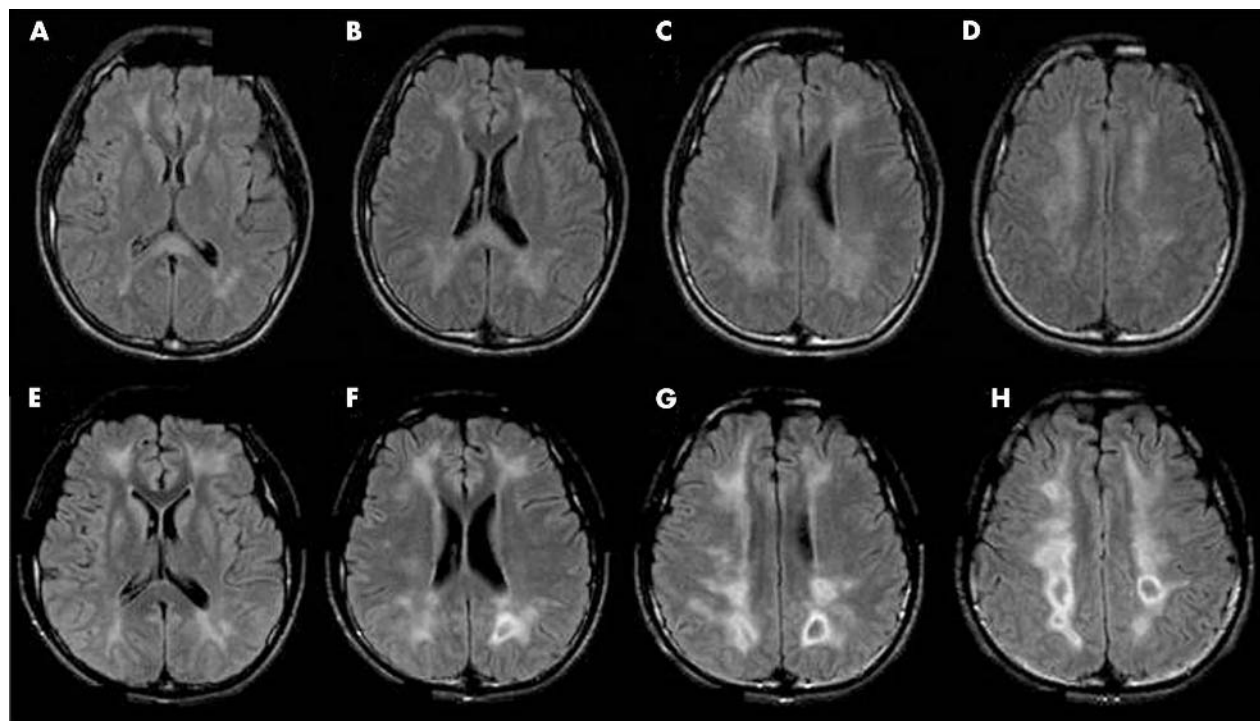
Brain CT scan and CSF examination were unremarkable (protein 0.39 g/L, glucose 3.6 mmol/L with normal cell counts and negative microbiological investigations including assays for neurotropic viruses). PCR for herpes simplex DNA in the CSF was negative and oligoclonal bands were absent. HIV and hepatitis B and C serologies were negative. Very long chain fatty acid and adrenocorticotrophic hormone levels were normal. T2

weighted and fast fluid attenuated inversion recovery (FLAIR) sequenced magnetic resonance images (MRI) of the brain (fig 1) revealed diffuse leucoencephalopathy within the periventricular, deep and subcortical white matter of the frontal and parietal lobes. The cerebellum and brainstem were normal. An electroencephalogram showed seizure activity that resolved with diazepam and sodium valproate. Periodic sharp wave complexes were not observed. Nerve conduction, somatosensory, brainstem auditory, and visual evoked response testing were all within normal limits.

Gradually, the patient recovered the ability to move his limbs and engage in conversation. He confessed to misusing benzodiazepines for chronic insomnia and to having injected sublingual buprenorphine tablets, crushed and dissolved in water, intravenously into his right external jugular vein on the day of admission. An empty bottle of buprenorphine tablets by his bed and a scar in the neck bore testament to this. He received intensive rehabilitation, and MRI brain was repeated 2 weeks later. The T2 and FLAIR hyperintensities previously seen were more extensive, with new areas of cystic degeneration (fig 1). He was put on a course of oral baclofen and discharged with amelioration but not ablation of limb spasticity. Power was rated at MRC grade 4/5 in all four limbs at the time of discharge.

### Discussion

The close temporal relationship of intravenous buprenorphine injection and symmetrical cystic leucoencephalopathy suggested to us a causal association. Intravenous buprenorphine was prepared in aqueous solution by crushing tablets of buprenorphine and dissolving them in water. We considered and excluded other differential diagnoses, such as acute disseminated encephalomyelitis (ADEM),



**Figure 1** (A–D) Axial T2 weighted fluid attenuated inversion recovery (FLAIR) MRI scan showing diffuse symmetrical hyperintense signal of the cerebral white matter. (E–H) More extensive areas of hyperintensities with new areas of cystic degeneration were observed 2 weeks later.

adrenoleucodystrophy, multiple sclerosis, metachromatic leucodystrophy, progressive multifocal leucoencephalopathy, hypertensive leucoaralosis, and hypoxic/ischaemic brain damage based on the clinical picture. The normal cerebrospinal fluid (CSF) pressure, protein, white cell counts, and absent oligoclonal bands exclude ADEM and infective causes. Adrenoleucodystrophy was excluded by normal levels of serum cortisol, ACTH, and very long chain fatty acids. The clinical, neurophysiological, and neuroradiological picture was not suggestive of multiple sclerosis. The patient had no history of hypertension, and hypoxic/ischaemic brain damage was unlikely, as the location of leucoencephalopathy was diffuse and not within the characteristic watershed territories. Genetic and metabolic causes of leucodystrophy could not be excluded, even though his normal neurocognitive developmental history, late onset, the close temporal relationship with injection of buprenorphine, and the absence of family history make them unlikely.

The direct role played by buprenorphine in causing the diffuse leucoencephalopathy is not known. As mu opiate receptors are expressed both in the brain and immune system (in lymphocytes and phagocytes), the injected buprenorphine could possibly have triggered an immunological response to neuronal tissues within the brain, severely affecting areas that are susceptible to the effects of demyelination and ischaemia, such as the subcortical white matter, thus resulting in diffuse leucoencephalopathy.<sup>2,3</sup> Although our patient denied the use of other recreational drugs, and repeated microbiological investigations did not reveal any pathogens, the possibility of impurities and additives present in the drug triggering an immunological and toxic reaction could not be excluded. The possibility of a synergistic effect on the neuroaxis of buprenorphine and benzodiazepine is raised as there have been reports of fatalities in patients receiving benzodiazepine and high dose buprenorphine.<sup>4,5</sup>

Intentional misuse of detoxification drugs should be suspected in a patient who presents with diffuse leucoencephalopathy. Further studies are needed to explain our observation—that is, whether the leucoencephalopathy occurred as a direct effect of buprenorphine or as an indirect effect of impurities and additives present in the drug.

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## Successful treatment of CMV ventriculitis immune reconstitution syndrome

In HIV infected patients, cytomegalovirus (CMV) disease of the central nervous system is usually seen when the CD4+ count is less than 100 cells/mm<sup>3</sup>. Recommended treatment includes intravenous ganciclovir, foscarnet, or both.<sup>1</sup> Ventriculitis is an uncommon but severe manifestation of CMV disease. Mortality is extremely high despite timely initiation of treatment. Little is known about CMV ventriculoencephalitis in the highly active antiretroviral therapy (HAART) era.

## Case report

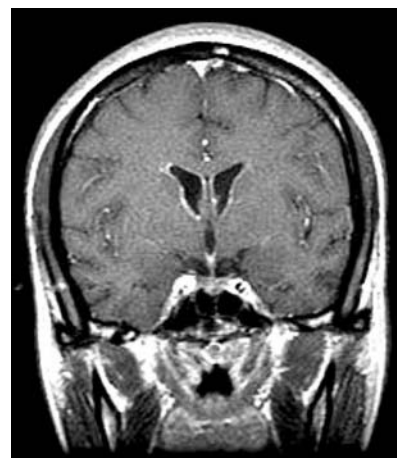
A 38 year old woman with no significant past medical history was diagnosed with *Pneumocystis jirovecii* (*Pneumocystis carinii*) pneumonia as her AIDS defining illness in April 2002. On 15 July 2002, HAART was initiated with zidovudine, lamivudine, and efavirenz. Her CD4+ count was 42 cells/mm<sup>3</sup> and her HIV-1 RNA level was 95 000 copies/ml. Serum CMV IgG was positive.

Ten days later, she was evaluated for headache of new onset. Computed tomography (CT) of her head was unremarkable. Cerebrospinal fluid analysis showed no white cells, a red cell count of 10/mm<sup>3</sup>, a protein level of 58 mg/dl, and a glucose of 40 mg/ml. Cryptococcal antigen serology was negative.

Two weeks later, after full compliance with her HIV therapy, she returned with headache, nausea, vomiting, and fever. Her temperature was 38.6°C (101.4°F). Neurological examination was normal and there was no meningism. Ophthalmological examination of the retinae with fully dilated pupils was normal.

The peripheral white blood count was 1800 cells/mm<sup>3</sup>. Serum chemistry was normal. Cryptococcal and histoplasma antigens were negative. Bacterial, mycobacterial, and fungal blood cultures were sterile. Chest radiography and repeat head CT were normal. CSF analysis showed 13 white blood cells/mm<sup>3</sup> (44% neutrophils, 44% lymphocytes, 12% monocytes), a red cell count of 4 cells/mm<sup>3</sup>, a protein level of 91 mg/dl, and a glucose of 30 mg/ml. Magnetic resonance imaging (MRI) of the brain showed abnormal contrast enhancement of the ependymal lining around the ventricles, consistent with ventriculitis (fig 1). CSF herpes simplex virus polymerase chain reaction (PCR) was negative. Both CSF PCR and bone marrow culture were positive for CMV.

Ganciclovir (5 mg/kg intravenously twice daily) was initiated. Seven days later, the patient was afebrile, but noted only mild improvement in her headache, nausea, and vomiting. Therefore, intravenous foscarnet (90 mg/kg twice daily) was added; her symptoms resolved within 21 days of combined therapy. At that time, her CD4+ count was 159 cells/mm<sup>3</sup> and the HIV-1 RNA level was <400 copies/ml. Follow up MRI showed improved appearance of the enhancement that was present at her initial diagnosis. CSF CMV PCR was subsequently negative. Valganciclovir (900 mg orally daily)<sup>2</sup> for maintenance treatment was added to her HAART regimen. Three months after completing CMV induction therapy, the patient's CD4+ count was 178 cells/



**Figure 1** Magnetic resonance imaging of the patient's brain at the time of her initial diagnosis. Note the abnormal contrast enhancement of the ependymal lining around the ventricles, with its characteristic "owl's eyes" appearance, a finding consistent with cytomegalovirus ventriculitis.

mm<sup>3</sup> and HIV-1 RNA was <50 copies/ml. Valganciclovir maintenance therapy was discontinued. There has been no recurrence of CMV disease over the 16 month period since discontinuing valganciclovir.

## Comment

Several points in this case are noteworthy. The patient was diagnosed as having CMV ventriculitis three weeks after initiating HAART, during a period of immune recovery. While immune reconstitution syndromes (IRS) involving CMV retinitis and pneumonitis have been reported, to our knowledge an IRS with CMV ventriculitis has not been described previously. The time to onset of IRS associated with CMV infections ranges from weeks to several months. Our patient developed a headache with a normal laboratory evaluation after 10 days of treatment. Three weeks after initiating HAART, her illness progressed to include fevers, persistent headache, nausea, and vomiting. While we cannot absolutely exclude the possibility that her CMV ventriculitis was a coincidental infection of a susceptible individual, we believe the timing of her illness with recent initiation of HAART is more consistent with a CMV associated IRS. Our patient was CMV seropositive and therefore latently infected when HAART was initiated. Her CD4+ count increased from 43 to 159 cells/mm<sup>3</sup> after 45 days of HAART. Additionally, between the 10th and 31st day of HAART, she developed CSF leucocytosis with progressive CSF protein elevation providing evidence of a significant CNS inflammatory reaction in a time interval consistent with IRS.

Studies addressing the best treatment for CMV ventriculoencephalitis in patients with AIDS are not definitive, although various comparative trials have addressed the treatment of retinitis. Current treatment options for CMV neurological disease include intravenous ganciclovir, intravenous foscarnet, or both.<sup>1</sup> In a study of HIV infected patients with acute CMV infection of the CNS, combination foscarnet-ganciclovir therapy resulted in clinical improvement or stabilisation in 23 of 31 patients, but 10 of these had



significant side effects necessitating discontinuation of at least one drug.<sup>3</sup> Our patient tolerated both foscarnet and ganciclovir for 21 days without significant side effects.

Given her initial presentation with advanced HIV-1 infection, we placed the patient on valganciclovir maintenance therapy (900 mg once daily) to prevent relapse of her CMV ventriculitis. In previous reports, maintenance therapy with intravenous ganciclovir prevented relapse of CMV encephalitis or myelitis in 13 of 23 AIDS patients.<sup>4</sup> Linear regression analysis of data from CMV and HIV seropositive patients shows that valganciclovir, 900 mg once daily, produces target AUC<sub>24</sub> values comparable to those achieved with intravenous ganciclovir at a dose of 5 mg/kg/day.<sup>5</sup> Our patient with CMV ventriculitis was prescribed oral valganciclovir maintenance therapy. Discontinuing maintenance therapy for CMV retinitis in patients who responded to HAART has been successful.<sup>6</sup> It is reasonable to follow the USPHS/IDSA guidelines for CMV retinitis and discontinue maintenance therapy when there has been a sustained (for example, more than six months) increase in CD4+ counts to >100–150 cells/μl in response to HAART. Based on our patient's robust CD4+ count improvement and complete suppression of HIV-1 viraemia, we judged her risk for CMV disease to be low and discontinued valganciclovir when her CD4+ count had been >100 cells/μl for three months. She has had no evidence of relapse of her CMV disease two years later.

In conclusion, CMV ventriculitis can present as an IRS in AIDS patients and should be considered in the differential diagnosis in a patient with CNS complaints, even in the absence of retinal disease. Aggressive treatment should be pursued, given the high mortality of the disease, and combined ganciclovir-foscarnet may be indicated based on the patient's presentation. The use of oral valganciclovir is a reasonable option for maintenance therapy.

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retinitis who have responded to highly active antiretroviral therapy. *Clin Inf Dis* 2002;34:394–7.

## BOOK REVIEWS

### Neuropsychological assessment, fourth edition

Edited by Muriel D Lezak, Diane B Howieson, David W Loring. Published by Oxford University Press, Oxford, 2004, pp 784, £59.50 (hardback). ISBN 0-19-511121-4.

So along comes a student—a medical colleague or speech and language therapist—and asks you for information about a certain type of neuropsychological assessment, form of cognitive impairment, or type of brain damage. If this falls under the auspices of one of your many areas of expertise then these queries are easy to field. However, they often stray into topics where our knowledge is much more limited. You also have a nagging doubt that much has been discovered about the subject since you studied it yourself as a student, not so many years ago ...

What we need in this situation is a handy textbook, or maybe series of books, in which a group of people have been kind enough to summarise the current state of knowledge with regard to types of brain damage, approaches to neuropsychological assessment and testing, types of perceptual, cognitive and affective impairments, and some information about useful neuropsychological assessments. On each of these specific topics, it is possible to find short and sometimes good guides (eg Hodges, 1994<sup>1</sup>). However, for a very authoritative and near-complete guide on all these different elements then the revised version of *Neuropsychological Assessment* (Lezak *et al.*, 2004) is very useful.

In this fourth edition, Lezak and colleagues have thoroughly updated the previous version despite the fact that the literature in each of these areas seems to grow exponentially. One consequence is that the book is much larger than previous versions but by producing a new edition, Lezak *et al.* have been able to cover and summarise many different types of brain diseases (including, for example, good summaries of non-Alzheimer neurodegenerative diseases), different domains of cognitive processes, and a large range of neuropsychological tests. I have been test-driving this book in my own lab for the last 4 weeks and have found that when I deal with queries from my colleagues and students, I have a new and helpful source of information.

**M A L Ralph**

## Reference

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### Book review palliative care in neurology

Edited by Raymond Voltz, James I Bernat, GianDomenico Borasio, Ian Maddocks, David Oliver, and Russell Portenoy. Published by Oxford University Press, Oxford, 2004, pp 435, £79.50 (hardback). ISBN 0-19-850843-3

This book is very opportune, with the increasing recognition of the need for specialist

palliative care in fields outside oncology. It is densely written and presented; however, it is very readable and would be of great use to those in specialist palliative care, who are developing an interest in neurology, as well as those who are specialist neurologists. The interface between neurology and palliative care is explored from several directions. The first section briefly surveys common neurological conditions such as stroke, motor neurone disease, dementia, Parkinson's disease, and human immunodeficiency virus/Creutzfeldt Jakob disease (HIV/CJD), giving a useful summary of the course and possible interventions in the disease process, and highlighting significant points and symptoms where specialist palliative care may be of assistance. The focus is always on integrated collaboration between the whole multi-disciplinary teams within the hospital and community, and between medical specialities. Areas of particular difficulty are handled well, such as the need for family support and ongoing bereavement work in families with muscular dystrophies, or involving relatives in decision processes for patients with limited capacity.

The second part of the book discusses the interface between neurological outcomes such as persistent vegetative state, locked in syndrome or quadriplegia, and palliative care. The chapters throughout the book commence with a case history, which neatly sets the scene and allows several points to be discussed, whilst keeping the focus patient centred. The third section explores the subject from the symptom rather than disease point of view. The pathophysiology and treatment of specific symptoms such as pain, nausea, fatigue, spasticity, and dysphagia are detailed and offer a good review of these areas. The sections avoid repetition with the first section, due to the tight focus and allows for a more detailed examination of both sides of the coin—disease process and symptom control. The next two sections revolve around the difficult areas of ethics, informed consent, advanced directives, and care of the dying patient. These chapters offer logic and clarity to often ignored and demanding areas, and are a good basis for further exploration. The contributors are all eminent in their fields, which is clear from the depth and clarity of the writing. Although the focus of the book is neurology/palliative care interface, many of the discussions and reviews are valid in any area of medicine. It should be required reading for all doctors in both disciplines.

**C E Urch**

## CORRECTION

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Michell A W, Raha S K, Barker R A, *et al.* A case of late onset sporadic Parkinson's disease with an A53T mutation in  $\alpha$ -synuclein (*J Neurol Neurosurg Psychiatry* 2005;76:596–7).

The authors wish to correct a typographical error in the primer sequence provided for exon 3. The NCBI database accession number was U46898 (HSACP03); primer pairs designed to amplify exon 3 were 3F: GAGGA CCTCTGTTAGCTGG, and 3R: GACTGATATG TTCTTAGAATGCTC.

In addition the order of authorship is incorrect. Dr Raha-Chowdhury should be in the senior (last) author position.