pain but little change in head control. After subsequent injections of 260 mg given over two days, he noted considerable improvement in the ability to drive and carry out other manual tasks in his work. He graded improvement in head control and pain as 50% and 80%, respectively, above baseline, compared with 50% and 50% after the previous BTX injections. He had transient mild tenderness in the injected muscles. After the first month he experienced a slight decline (10%) in function and pain control that remained constant for the next four months and then gradually declined again. With subsequent injections of phenol at about six monthly intervals, he has maintained his maximal level of improvement.

These two patients had moderately severe spasmodic torticollis that had improved only partially after previous treatment. In the first patient, BTX initially provided relief but became ineffective. In the second patient, BTX provided improvement but the side effect of dysphagia was nearly intolerable. Within 18 hours after phenol injections into cervical muscles, there was definite reduction of involuntary movements and pain, with functional improvement. Improvement was greater than after all previous treatments and persisted for six and five months respectively, after the initial series of phenol injections. The only side effect was transitory—namely, mild tenderness in the injected muscles.

In patients who become resistant to repeated injections of BTX, presumably due to formation of antibody to the toxin, it would be of great benefit to have an alternative treatment. Phenol may be of benefit in this situation and has the additional advantage of being inexpensive. If the promising response in these two patients is confirmed in a larger series of patients I am currently studying, EMG guided intramuscular phenol injections may prove to be an effective treatment for some patients with spasmodic torticollis.

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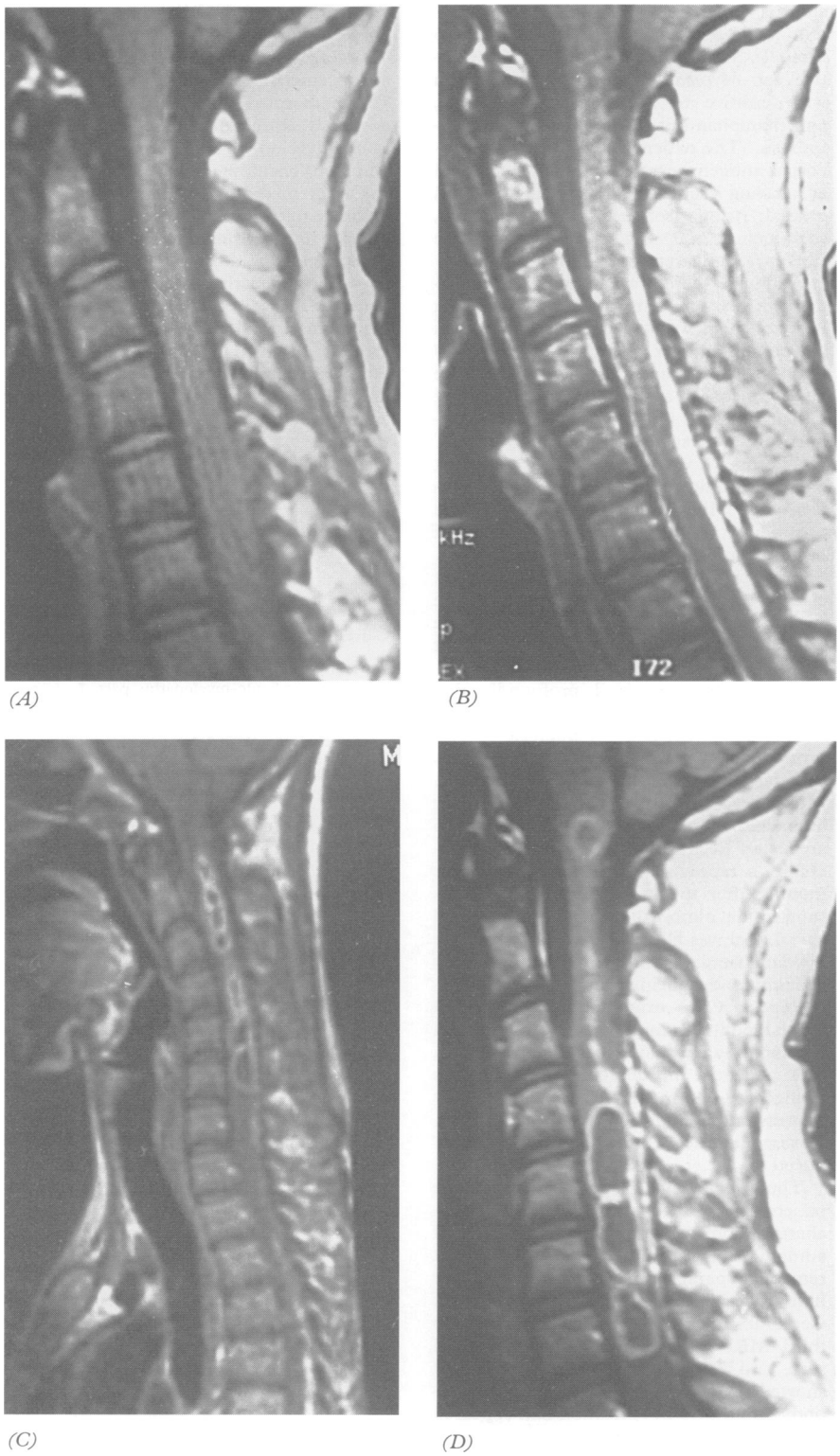

(A) Sagittal T1 weighted MRI of the cervical spine before and (B) after gadolinium—DTPA showing enhancement in the spinal meninges. (C) Sagittal T1 weighted MRI of cervical and dorsal spine after gadolinium—DTPA four months after the initial scan. Multiple gadolinium ring enhancing lesions are seen within the cervical cord. (D) Sagittal T1 weighted MRI of cervical cord showing further changes in the intramedullary cavities eight months after the initial scan. Intense gadolinium enhancement continues in some loculi, others have resolved, and a new locule has appeared at the cervicomedullary junction.

Tuberculous myelopathy: a serial MRI study

In two definitive publications in 1969 Wadia and Dastur delineated spinal meningitides with associated radiculomyelopathy with particular reference to tuberculosis.[1 2] The advent of MRI has meant that the nature of the intramedullary lesions can for the first time be defined during life. Furthermore, the natural history of spinal cord involvement in tuberculosis can be studied with serial MRI. The current study involves a single case followed up with a series of MRI examinations over an eight month period. It shows a previously unpublished pattern of cord involvement.

A 26 year old Samalian male refugee who had been in the United Kingdom for one year presented with a three month history of nausea and vomiting associated with increasingly severe headaches. He had no previous history of or exposure to tuberculosis. On admission to hospital he had neck stiffness only. Initial CSF examination

showed 33 lymphocytes/mm³, 7·33 g/l protein, and 1·4 mmol/l sugar (blood sugar 6·6 mmol/l). Subsequent CSF culture was positive for *Mycobacterium tuberculosis*, which was sensitive to isoniazid, pyrazinamide, and rifampicin but highly resistant to streptomycin. The patient was HIV and cryptococcal antigen negative. He was diagnosed as having tuberculous meningitis and treated with isoniazid (500 mg/day), rifampicin (600 mg/day), pyrazinamide (2 g/day), and streptomycin (750 mg/day).

Although there was an initial improvement in his symptoms after three weeks in hospital there was a rapid deterioration in his condition over a period of 24 to 48 hours, with dysphagia, dysarthria, and the development of a spastic tetraparesis that initially allowed him to walk with the support of one person. An enhanced brain CT was normal. It was assumed that the patient had had a brainstem infarct, which was confirmed on MRI of the posterior fossa. The possibility of spinal cord complications of his tuberculous meningitis was considered. Spinal cord MRI showed a normal cord but clearcut gadolinium enhancement in the spinal meninges (figure). Dexamethasone was added to his antituberculous treatment for four weeks.

The patient remained profoundly disabled with deteriorating lower limb function. He became paraplegic with pronounced flexor spasms. He was catheterised and fed by a gastrostomy tube. Muscle wasting developed in the upper limbs and there was associated upper limb areflexia. Because of his increasing disability, a repeat scan was carried out four months after the initial spinal MRI. This showed that almost the whole of the cervical spinal cord was filled with loculated discrete hypointense areas outlined by an intensely gadolinium enhancing rim.

Repeat CSF examination showed < 3 white blood cells/mm³, > 1 g/l protein, and 2 mmol/l sugar (blood sugar 4·4 mmol/l). The patient's antituberculous regime was changed to 900 mg/day rifampicin, 2 g/day pyrazinamide, and 500 mg/day isoniazid. Dexamethasone (16 mg/day) was reintroduced.

There was no major change in the patient's overall condition. He was discharged home six months after his initial admission. He resumed oral feeding but remained paraplegic with limited upper limb function: He remained catheterised. Cerebral function was intact. Follow up third MRI eight months after the initial scan and four months after the second showed no dramatic change from the second. A new area of loculation had appeared at the cervicomedullary function. Some of the earlier lesions were less obvious whereas others had remained unchanged.

The prominent tuberculous arachnoiditis described by Wadia and Dastur[1 2] can now be shown by the presence of gadolinium enhancement in the spinal dura-arachnoid complex on MRI. This was first shown in three out of the five cases reported by Chang *et al.*[3] This was the only obvious abnormality on first MRI of the present patient. Without the serial studies he would have been defined as a straightforward case of spinal arachnaditis.

The second and third scans showed multiple massive loculated lesions within the cervical cord. These were far too large for isolated tuberculomas. The radiological features fit most closely with intramedullary abscesses. In particular the high signal on T2 weighted scans and the thick rim of enhancement correspond well with the known morphological features of pyogenic abscesses. Intracranial tuberculous abscesses can develop while patients are on antituberculous chemotherapy.[4]

Earlier medical literature has tended to separate tuberculous radiculomyelitis completely from syringomyelia secondary to chronic arachnoiditis. Appleby in 1969 perhaps first clearly established the link between delayed myelopathy after chronic meningitis and syringomyelia. More recently MRI studies have confirmed this link.[5] The myelopathy in these cases is usually delayed by many years and gadolinium enhancement is not seen in the wall of these syrinxes.

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Bickerstaff's brainstem encephalitis associated with cytomegalovirus infection

Cytomegalovirus infection rarely induces encephalitis in a non-immunocompromised host. Bickerstaff's brainstem encephalitis was characterised by the acute onset of external ophthalmoplegia and cerebellar ataxia with CNS signs and symptoms.[1] We describe here the first case of Bickerstaff's brainstem encephalitis associated with cytomegalovirus infection.

A previously healthy 17 year old girl developed fever, headache, and general fatigue. Ten days later, drowsiness, diplopia, and walking difficulty developed. She had low grade fever, drowsiness, and neck stiffness. The right eye showed esophoria. External ophthalmoplegia was severe in both lateral directions and moderate in the right upward direction. Triceps reflexes were absent and biceps reflexes were reduced. Finger to nose and heel to knee tests showed dysmetria and decomposition. Romberg's sign was negative. She had a broad based gait and tandem gait was impossible. Muscle strength and sensory function were normal. Blood count, liver function, and cold agglutinins were normal. Serum cytomegalovirus-IgM antibodies were negative and HIV 1 antibody was absent. Serum IgG anti-GQ1b antibody tests[2] were positive. A lumbar puncture showed a high pressure (300 mm H_2O), and high values for cell count, protein, albumin, IgG, IgA, and anticytomegalovirus antibodies (table). Cytomegalovirus DNA was detected in the CSF by the polymerase chain reaction. Brain CT and MRI were normal. Electroencephalography showed irregular slow waves with an enhanced and prolonged response to hyperventilation. Electromyography and sensory conduction studies were normal. The patient was treated with acyclovir (1500 mg/day) for seven days. Clinical symptoms disappeared as follows; low grade fever disappeared on the 17th day of illness, drowsiness and neck stiffness on the 22nd day, ataxia on the 24th day, and the eye movement disorder on the 30th day; deep reflexes returned to normal on the 36th day. On the 30th day of illness, euphoria with pathological laughing appeared; she burst out laughing when our eyes met or we began to talk and this lasted for seven days. Reduction of anticytomegalovirus antibodies and cytomegalovirus DNA by the polymerase chain reaction in the CSF paralleled the clinical improvement. The serum anti-GQ1b antibody titre was also decreased. The EEG abnormalities disappeared. Titres of antibodies against herpes simplex virus, varicella-zoster virus, and Epstein-Barr virus in both serum and CSF showed no significant changes.

Although brain MRI and CT showed no abnormalities, the rapidly developed ataxia, drowsiness, ophthalmoplegia without downgaze disturbance, and abnormal EEG findings suggested the presence of a CNS lesion.[3] We therefore diagnosed the patient as having Bickerstaff's brainstem encephalitis.[2] Reduction of the increased CSF anticytomegalovirus antibodies and positive cytomegalovirus DNA paralleled the

Clinical data

	Day of illness				
	21	*28*	*42*	*56*	*76*
CSF:					
Cells (/mm³)	63	67	29	24	13
(monocytes (/mm³))	(59)	(65)	(27)	(22)	(11)
protein (mg/dl)	99	76	52	35	27
IgG (mg/dl)	9·6	8·0	4·9	2·8	2·8
CMV-IgG-FA (normal <1 ×)	1 ×	1 ×	<1 ×	<1 ×	<1 ×
CMV-IgG-ELISA (normal <200)	596	485	304	<200	<200
CMV-DNA-PCR	+	ND	ND	—	ND
Serum:					
IgG anti-GQlb antibody (normal <400)	6400	ND	3200	ND	1600
CMV-IgG-ELISA	3870	2700	3320	2270	3370

FA = indirect fluorescent antibody technique; ELISA = enzyme linked immunosorbent assay; ND = not done; PCR = polymerase chain reaction; CMV = cytomegalovirus.