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Short report

Magnetic resonance imaging in central pontine myelinolysis

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SUMMARY Magnetic resonance imaging (MRI) was performed in two patients in whom a clinical diagnosis of central pontine myelinolysis (CPM) had been made. MRI showed lesions in the pons in both cases about 2 years after the illness, at a time when the spastic quadriparesis and pseudobulbar palsy had recovered. The persisting abnormal signals in CPM are likely to be due to fibrillary gliosis. Persistence of lesions on MRI means that the diagnosis of CPM may be made electively, after the acute illness has resolved.

Central pontine myelinolysis (CPM) is a descriptive term for a distinct pathological entity in which primary demyelination of the basis pontis is the dominant feature.[1] Although formerly considered a rare condition, there have been numerous reports of cases fulfilling the pathological criteria for the diagnosis of CPM,[2] while recent clinical descriptions have suggested that it may be more common than previously recognised.[3,4] A major problem in the diagnosis of CPM in life has been the reliance on clinical criteria. Computed tomography of the brain[5–10] and brainstem evoked potentials[5] may indicate pontine abnormalities, but magnetic resonance imaging (MRI)[11–22] has shown quite distinctive changes. Moreover these may persist for some time after the illness. The purpose of this paper is to draw attention to the nature and significance of these persistent changes.

Case reports

The presenting clinical features of these two patients have been reported in detail previously[3] and only a brief summary will be given here.

Case 1. An 18 year old woman presented in the 20th week of her second pregnancy with hyperemesis gravidarum. Admission values for serum sodium and urea were 126 mmol/l and 86·5 mmol/l, respectively. Over the subsequent 2 days she was given 10 l of 0·9% sodium chloride and 6 l of 5% dextrose water. These measures corrected the serum sodium to 145 mmol/l and the urea to 14·7 mmol/l. Four days later she became anarthric. The next day she was irritable and

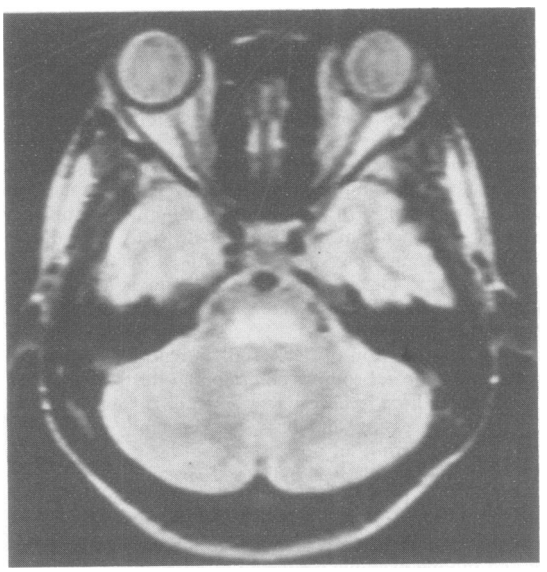

Fig 1 *(Case 1): 5 mm axial and sagittal slices of the brain and brain stem obtained with a Picker 0·5 Tesla MR scanner using a spin-echo sequence (SE 2000/60). There is a well defined area of high signal in the central pons.*

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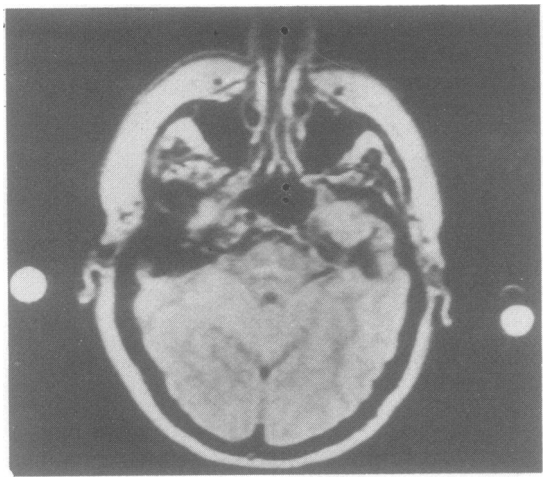

Fig 2 *(Case 2): Axial MR scans of brain stem as described in fig 1. The area of abnormal signal in the pons in this case shows a 'butterfly" pattern (see text).*

confused with bilateral 6th cranial nerve palsies and horizontal nystagmus. Wernicke's encephalopathy was diagnosed and parenteral thiamine, 250 mg, was administered. By the following day the abnormal ocular movements had improved, though there was now bilateral lower facial weakness and a left extensor plantar response. A spastic quadriparesis without sensory change evolved over the ensuing 2 weeks. These neurological deficits subsequently improved and 8 weeks after admission she was independent for daily activities.

At 37 weeks gestation she was delivered of a healthy male infant whose subsequent development has been normal.

MRI was performed 20 months after the illness, at which time clinical examination was normal. A well defined area of abnormal signal was present in the basis pontis sparing the ventral pons (fig 1).

Case 2. A 52 year old woman presented with drowsiness and a 1 week history of vomiting and diarrhoea. Her daily medication for hypertension included chlorthalidone, spironolactone, and cyclopenthiazide. She adhered strictly to a salt free diet. On examination she was confused, without other abnormal neurological signs. Shortly after admission she suffered a generalised seizure. The serum sodium was 99 mmol/l. Over the next 3 days she received 2 l of 2% sodium chloride, 2·5 l of 0·9% sodium chloride, parenteral frusemide, and oral sodium chloride and dextrose powder. The serum sodium level rose to 123 mmol/l. Thereafter, she developed increasing difficulty speaking and moving her limbs. One month after admission she was observed to be alert with a pseudobulbar palsy and spastic quadriparesis. Sensation was intact. During the following 2 months she improved and was mobile and independent at discharge.

When examined 2 years later, she exhibited slight dysarthria and mobile dystonic posturing of both upper limbs, left more than right. Deep tendon reflexes were symmetrically brisk. Her gait was wide-based and she was unable to walk heel-to-toe.

MRI revealed an area of abnormal signal in the pons (fig 2). Scattered abnormal signals also were evident in the cerebral white matter. These could have been associated with her preceding hypertension or extra-pontine myelinolysis. No lesions were identified in the basal ganglia or thalamus.

Discussion

The site, shape and size of the lesions in CPM demonstrated by MRI are identical to those described in pathological specimens. There may be large symmetrical lesions in the basis pontis, usually sparing the ventral pons,[2 11-23] or there may be smaller, "butterfly" or "trident" shaped lesions in the base of the pons as in case 2 (fig 2).[19 24]

These lesions of CPM, visualised by MRI, have now been reported by many authors,[11-23] and demonstrated at intervals ranging from weeks to months after resolution of the acute illness. In the two cases presented in this report, the lesions were still evident 20 and 24 months after recovery, at a time when one of the patients was normal on clinical examination. The nature of such persisting lesions is worthy of further comment.

Lesions are visualised on MRI largely because of an increase in tissue water proton content.[25] The cardinal pathological change of CPM is loss of myelin with preservation of axons, in the absence of a significant inflammatory reaction.[1 2] In the chronic lesion, intense fibrillary gliosis has been observed.[2] Loss of myelin per se, is unlikely to affect the MRI signal since the lipid protons in myelin make a negligible contribution to the normal brain signal.[26] On the other hand, the proliferation of astrocytes (with their abundant cytoplasm) in areas of gliosis would result in an increase in tissue water content per unit volume. As the signals in normal brain are produced by water protons,[26] it seems likely therefore that gliosis is making an important contribution to the MRI signal in the chronic lesion. The clinical recovery in the face of such gliosis is noteworthy.

The observation that residual gliosis after CPM may remain visible on MRI long after the critical phases of the illness, and when neurological deficits have improved or recovered, has several implications. The most important of these is that elective scanning may yield diagnostic information in patients suspected of having CPM. Thus, patients may be scanned when their clinical state permits, after the acute illness has resolved. Furthermore, it provides opportunity to obtain a clearer picture of the incidence of this condition, and may help resolve the controversy surrounding its aetiology and pathogenesis, particularly in relation to the treatment of hyponatraemia.[3 27]

Finally, the persistence of the MRI signals in CPM with clinical recovery must be taken into account when

interpreting MRI appearances in the context of a subsequent, unrelated illness.

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