

## Letters

### Cervical myelopathy due to ossification of the posterior longitudinal ligament

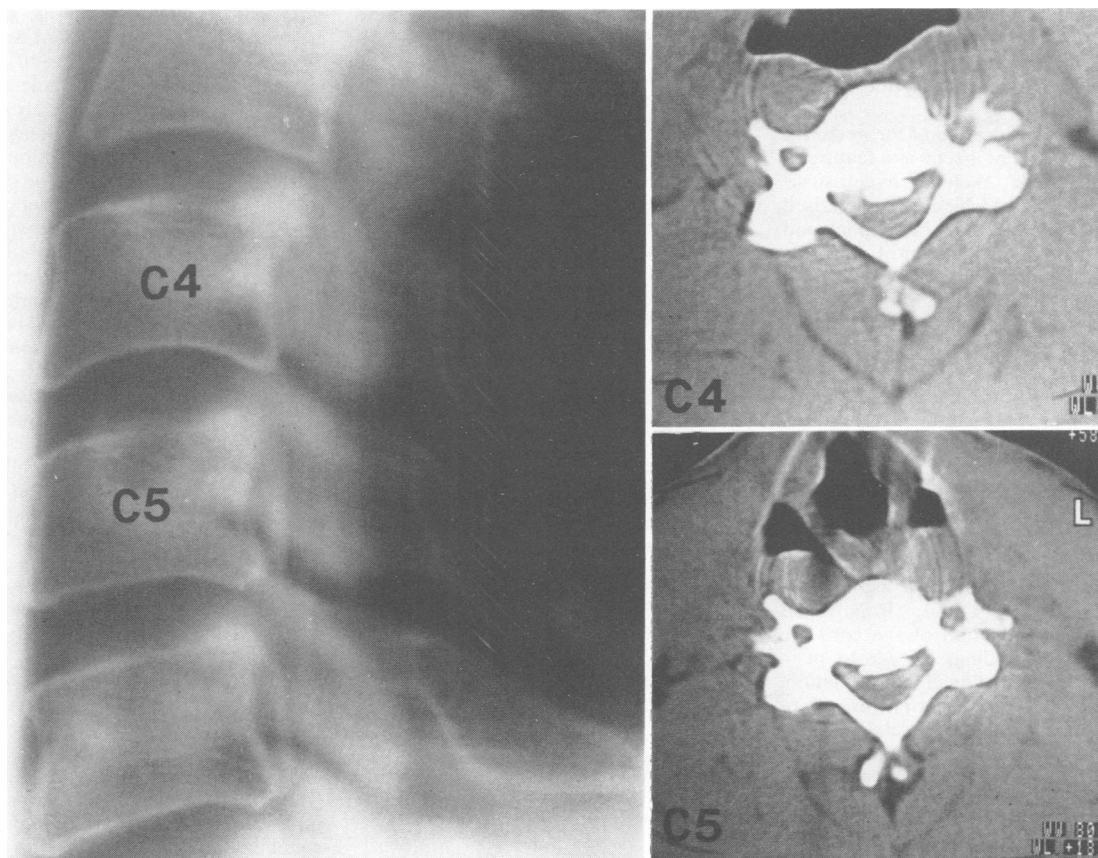
Sir: Ossification of the posterior longitudinal ligament as a cause of myelopathy was first reported in Japan<sup>1-3</sup> and was subsequently noted in the USA in non-oriental patients. The condition is not widely recognised in the UK and there has been only one previous British report.<sup>4</sup>

The patient was a 42-year-old West Indian factory foreman who had moved to the UK in 1966. He had been treated for

urethral discharge in 1975. There was no relevant family history. In December 1982 he had developed pain in the neck and right shoulder which lasted a few days. This was followed by numbness and tingling of all the digits of both hands and, one month later, by numbness of the left side of the trunk and left leg. These symptoms persisted unchanged. Except for difficulty in maintaining penile erection there were no other symptoms. He was admitted for investigation 3 months later. General physical examination, including neck movements, was normal. There was no limb weakness. Tendon reflexes were brisker in the right limbs, abdominal reflexes were

absent and both plantar responses were flexor. There was subjective impairment to pin prick over the left leg and left trunk with an indistinct high thoracic level.

Haemoglobin estimation, WBC, ESR, serum B12 and routine biochemistry were normal. Serum serology for syphilis (RPRT, TPHA, FTA) was positive but CSF examination was normal including negative serology. Chest radiographs were normal. Radiography of cervical spine and metrizamide myelography with CT scanning (fig) showed ossification of the posterior longitudinal ligament with anterior compression of the cord at C4 and 5. Although there was no evidence of



### Radiological findings

Fig A lateral view of the cervical spine and lateral tomography show ossific structures lying posterior to the vertebral bodies. CT scan with metrizamide demonstrated the lesion to be more extensive, from C3 to C6. It was discontinuous at the level of the discs and widest (3.7 mm) behind the C4 vertebral body. Compression of the cord is seen at C4 and C5, the sagittal diameter of the canal being reduced from 12.6 mm to 8.9 mm at C4 and from 12.2 mm to 9.4 mm at C5.

neurosyphilis, the patient was treated with procaine penicillin. Four months later there had been no significant change in either symptoms or signs.

Ossification of the posterior longitudinal ligament is of unknown aetiology and may have an incidence as high as 1–3% in the Japanese who have cervical radiology.<sup>3,5</sup> It may occur at any spinal level but is usually maximal in the cervical region. Although there is frequently no neurological involvement, the condition may cause a progressive myelopathy requiring decompressive laminectomy.<sup>3</sup> As would be expected, myelopathy tends to occur with greater degrees of encroachment of the cervical canal. Severe myelopathy was noted where ossification occupied more than 30% of the cross-sectional area of the spinal canal<sup>6</sup> but there are reported several cases of gross radiological change without symptoms. In this patient neurological disturbance was mild and not obviously progressive.

Occasionally ossification of the posterior longitudinal ligament is seen as a feature of diffuse idiopathic skeletal hyperostosis (Forestier's Disease) which is characterised by bone proliferation in both axial and extra-axial sites.<sup>8</sup> Ossification of the posterior longitudinal ligament is distinct from cervical spondylosis although the two conditions may co-exist.<sup>7</sup> In ossification of the posterior longitudinal ligament there is ectopic bone formation rather than calcification of the posterior longitudinal ligament. The ossification exceeds the anatomical limits of the posterior longitudinal ligament and is continuous with the vertebral bodies whereas the posterior longitudinal ligament itself attaches only to the annulus. Lastly, ossification of the posterior longitudinal ligament tends to be most marked at C3 to C5, a higher level than cervical spondylosis. Reports of pathological examination of the cord are limited but the findings appear to be a non-specific combination of demyelination, axonal loss and vascular changes, all of which are maximal in the dorsal and lateral region of the cord.<sup>9</sup>

Early changes are inconspicuous on lateral films of the cervical spine and are unlikely to be recognised unless the possibility of ossification of the posterior longitudinal ligament is considered. The extent of ossification is best shown by CT.<sup>5,6,10</sup> In this case CT was combined with metrizamide myelography which demonstrated clearly the level and extent of cord compression.

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#### Wernicke-Korsakoff syndrome with bilateral facial nerve palsies.

Sir: In 1881 Carl Wernicke described the first three cases of the syndrome which has

subsequently borne his name. The studies of Harper<sup>1,2</sup> indicate that the necropsy incidence of this condition is 2.8%, with only 20% of such cases being correctly diagnosed on clinical grounds. Nevertheless in the series of Victor *et al.*,<sup>3</sup> the incidence of ocular disorders and ataxia was high (96% and 87% respectively), and yet no sign of other cranial nerve palsies was present. A classical case of the Wernicke-Korsakoff syndrome was recently encountered, accompanied by severe bilateral facial weakness of lower motor neuron type. A housewife aged 29 years had had a considerable alcohol intake for at least 10 years, consuming about 6 bottles of beer (approximately 200 grams of alcohol) each day. She presented to her general practitioner some ten weeks before admission with right facial hyperaesthesiae, and bilateral parotid gland enlargement was noted. Two weeks prior to admission she was noted to be emotionally unstable and withdrawn, and became progressively confused and immobile. A right facial weakness was noted one week before admission. Examination revealed the patient to be obese and afebrile. She was dehydrated with bilateral parotid swelling and tender hepatomegaly. Neurological examination showed that she was drowsy and apathetic, and she obeyed only simple commands and mumbled occasional words. The fundi showed oedema of the nerve fibre layer with some linear haemorrhages, the discs appearing normal. Prominent horizontal and vertical nystagmus was present, with normal ocular movements. Severe bilateral facial weakness was evident involving all muscle groups; subsequent examination revealed no impairment of taste. Minimal lid movement was observed with corneal reflex testing, but there was no blink to menace. No apparent limb weakness could be detected, the reflexes in the arms being reduced and the leg reflexes were absent. The plantar responses were flexor. Sensory examination at a later date showed only a minimal distal impairment of pain in the toes.

Investigations showed a haemoglobin of 16.3 g/100 ml, a packed cell volume of 49%, and a mean corpuscular volume of 88 (range 80–96). The erythrocyte sedimentation rate was 52 mm/h. The white cell count was  $20.4 \times 10^9 \text{ mm}^{-3}$  with a neutrophilia of 72%. Serum biochemistry was consistent with pre-renal failure with a hypernatraemia of 154 mmol/l (range 137–145), a high urea of 58.5 mmol/l (range 3–8), and a high creatinine of 0.55 mmol/l (range 0.05–0.12). Bilirubin was normal, gamma glutamyl transpeptid-