EDITORIAL COMMENTARY

Subacute combined degeneration

Subacute combined degeneration of the spinal cord (SCD) is the most frequent neurological manifestation of vitamin B₁₂ deficiency. It is now seen much less often than in former years as the diagnosis is made earlier in most patients on haematological grounds. Nevertheless, patients in whom the initial manifestations are neurological still occur and it is a tragedy if early diagnosis is not made in this treatable condition. Presentation is either with a distally predominant sensory neuropathy, accompanied or preceded by myelopathy, and sometimes by optic atrophy or cognitive change. Suspicions that a sensory neuropathy may be related to vitamin B₁₂ deficiency can be raised by an upper limb onset and the occurrence of Lhermitte's symptom.

Hemmer *et al* in this issue (pp 822–827) document retrospectively the clinical, electrophysiological, and MRI findings in nine patients with SCD. In four the disorder was related to pernicious anaemia, in four others to gastrointestinal disease, and in one the explanation was not established. In all nine patients the salient clinical and electrophysiological changes were those of posterior column dysfunction, but with associated corticospinal tract degeneration in four, subcortical dementia in three, and depression in one.

Before treatment tibial somatosensory evoked potentials (SEPs) were abnormal in all patients whereas nerve conduction studies (NCs) were only abnormal in five. As originally shown by Fine and Hallett,¹ spinal SEPs become abnormal in SCD before changes develop in the peripheral nerves. Spinal MRI was undertaken in four of the present patients before treatment and in these and two others after treatment. In two of those imaged before treatment there were hyperintense lesions in the dorsal columns in T2 weighted images extending throughout most of the cervical cord. Such changes are non-specific but if seen, should

raise the suspicion of vitamin B₁₂ deficiency. All scans after treatment were normal.

The pathology of the changes in peripheral nerve is not as yet fully elucidated. Nerve biopsy studies have indicated an axonopathy.^{2–3} In the present study, NCs were normal in four. In two others they indicated an axonal sensory neuropathy and in one a sensory demyelinating neuropathy. The possible nature of the demyelination is of interest. Conceivably it could have represented superimposed immune mediated demyelination. Nerve biopsy might have been informative.

The abstract of the paper concludes with the advice that vitamin B₁₂ deficiency should be considered in the differential diagnosis of "all spinal cord, peripheral nerve and neuropsychiatric disorders". Although the range of manifestations of vitamin B₁₂ deficiency is certainly wide, I am not certain that I would request a vitamin B₁₂ assay in a pure motor neuropathy, for example. Vitamin B₁₂ deficiency is a classic neurological system degeneration in which particular sets of neurons are affected because of selective vulnerability. What this depends on is still not established as the precise biochemical defect in vitamin B₁₂ deficiency remains uncertain. The contending hypotheses have been discussed by Thomas and Griffin.⁴

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- 1 Fine EJ, Hallett M. Neurophysiological study of subacute combined degeneration. *J Neurol Sci* 1980;45:2–3.
- 2 Kosik KS, Mullins TF, Bradley WR, *et al*. Coma and axonal degeneration in vitamin B₁₂ deficiency. *Arch Neurol* 1980;37:590–2.
- 3 McCombe PA, McLeod JE. The peripheral neuropathy of vitamin B₁₂ deficiency. *J Neurol Sci* 1984;66:117–24.
- 4 Thomas PK, Griffin JE. Neuropathies predominantly affecting sensory or motor function. In: Asbury AK, Thomas PK, eds. *Peripheral nerve disorders* 2. Oxford: Butterworth, 1995:59–94.