VITAMIN B12 NEUROPATHY IN THE ABSENCE OF ANAEMIA — CASE REPORT

I S Chiew, K L Goh, T G Loh

ABSTRACT

A 70-year old man with neurological manifestations of vitamin B12 deficiency in the absence of anaemia is reported. The clue to the correct diagnosis is a raised mean corpuscular volume.

Key words: Vitamin B12 deficiency, neurological manifestations, raised MCV.

SING MED J. 1989; No 30: 221 — 222

INTRODUCTION

Subacute combined degeneration of the cord is the most serious neurological complication of deficiency of vitamin B12, and most commonly occurs with megaloblastic anaemia. These two conditions were considered as totally distinct when first observed by Leichterstein and Russel et al (1). Later observations proved that they were actually intimately associated, but the symptoms of anaemia usually precede the neurological manifestations. This neurological complication affected twenty to forty percent of earlier reported cases of pernicious anaemia (1). Only in exceptional cases subacute combined degeneration of the cord occurred in the absence of anaemia (1-5), but some of these cases subsequently developed megaloblastic anaemia if untreated or inadequately treated (1, 2). A case of subacute combined degeneration of the cord in the absence of anaemia is reported.

CASE REPORT

A 70 year old Chinese man presented with an unsteady gait, numbness and progressive weakness of his extremities for two months. He had no other symptoms of systemic illness. He had a well-balanced diet. He was a non-smoker and non-drinker, and had not been on any medication.

Physical examination revealed no anaemia. There was mild weakness of all four limbs, with brisk upper limb reflexes, suppressed knee reflexes, absent ankle jerks and extensor plantar response bilaterally. He had symmetrical hyperaesthesia of both hands and feet. Position and vibration sense were lost in both legs. Romberg's sign was positive.

Investigations showed a normal full blood count with haemoglobin of 14.5gm/dl, packed cell volume 44%, platelet count of 155 x 10^9/l, total white cell count of 5.9 x 10^9/l. The mean corpuscular volume was increased to 110.4 fl. A peripheral blood film examination revealed macrocytosis with some hypersegmented neutrophil nuclei. Serum vitamin B12 was decreased at 25 microgram/l (normal > 150) and serum folate was normal at 11.8 nanogram/ml (normal > 2.5). Malabsorption of B12 due to a lack of intrinsic factor was confirmed by Dicopac test (a modified Shilling test using Cobalt isotopes). The bone marrow aspiration examination was consistent with megaloblastosis. Mild generalised gastritis was seen at endoscopy. The gastric biopsy showed histological changes of chronic superficial gastritis with intestinal metaplasia. Serum antibody to intrinsic factor was not detected. Nerve conduction studies showed distal latency delays consistent with peripheral neuropathy.

The patient was commenced on intramuscular B12 injections as soon as the diagnosis of B12 deficiency was made. At follow-up two months later he had shown improvement in both symptoms and nerve conduction studies. Repeat haemoglobin was 14.2 g/dl and MCV 90.4 fl . . .

DISCUSSION

This 70 year old man had features consistent with subacute degeneration of the cord as indicated by the presence of pyramidal tract and dorsal column involvement. Though his haemoglobin was normal, his peripheral blood film and bone marrow were suggestive of megaloblastic changes. Defective absorption of vitamin B12 due to the lack of intrinsic factor was confirmed by the Dicopac test. Although serum intrinsic factor antibody was not detected, this does not rule out pernicious anaemia because it is only present in 50 to 60% of patients with proven pernicious anaemia (6). The classical presentation of vitamin B12 deficiency is a combination of a megaloblastic anaemia and subacute combined degeneration of the cord with peripheral neuropathy. Up to 95% of patients with pernicious anaemia have been reported to have neurological involvement, about a third of these experienced only mild symptoms such as paraesthesia or neuropsychiatric manifestations (5). A smaller proportion is associated with the more serious myelopathy. With better awareness, pernicious anaemia is diagnosed earlier and subacute combined degeneration of the cord is rarely seen nowadays. Occasionally, vitamin B12 deficiency presenting initially with features of myelopathy and peripheral neuropathy may be misdiagnosed. In such cases, and increased mean corpuscular volume, even if the haemoglobin is normal, should raise the suspicion of vitamin B12 deficiency as a cause.

The response to vitamin B12 replacement is generally very gratifying. The patient will experience an increase in strength and improved sense of well-being even before a haematological response is evident. The reversion towards a normal marrow begins a few hours after the initiation of treatment. Anaemia will be corrected over

Department of Medicine
Faculty of Medicine
University of Malaya
59100 Kuala Lumpur
Malaysia

I S Chew, MB chB (Sheffield),
K.L Goh, MRCP (UK),
T G Loh, MRCP (UK),

Correspondence to: Dr Chiew

several weeks. Prompt treatment is important in reversing the neurological damage. Delay in treatment may result in permanent neurological deficits despite an optimal replacement.

References

1. Jewesburg ECO. Subacute degeneration of the cord and achlohydric peripheral neuropathies without anaemia. Lancet 1954; ii: 307-12
2. Robertson EG, Wood IJ, Joske R et al. Gastric atrophy with subsequent pernicious anaemia. Lancet 1955; ii: 69-71
3. Melsom H, Kornstad S, Abilogaad U. Reversible neuropathy of vitamin B12 deficiency with normal haemoglobin and serum vitamin B12. Lancet 1977; i: 803
4. Perold JG. Vitamin B12 neuropathy in the absence of anaemia. S Afr M J 1981; 1: 570
5. Wintrobe MW. Clinical Haematology, 7th edition, Philadelphia:lea & Febiger 1974; 612-4
6. Fairbanks VF, Lennon VA, Kokman E, Howard FM. Tests for Pernicious anaemia : Serum Intrinsic Factor Blocking Antibody. Mayo Clin Proc 1983; 58: 203-4