Letter to Editor

**Vitamin B12 deficiency presenting with an acute reversible extrapyramidal syndrome**

Sir,

I read with interest the report of acute reversible extrapyramidal syndrome presumably caused by vitamin B12 deficiency by Sudhir Kumar.\(^1\) If this finding can be replicated, we may have another neurological manifestation caused by deficiency of vitamin B12. Probably from now on we should look for extrapyramidal manifestations in patients with deficiency of this vitamin.

Dr. Sudhir’s report also tells the importance of giving credence to simple investigations like complete blood count and peripheral smear examination. This could give unexpected and remarkable results as far as treatment is concerned at least in a few number of patients. Such observations could go a long way as far as clinical medicine is concerned.

I have a slight objection to the statement made by Kumar about the diagnosis. He states in the case report section “a diagnosis of acute onset parkinsonism with mild myeloneuropathy secondary to vitamin B12 deficiency was made” during the initial stage when the patient was seen. Because there had never been any report in the literature of such an observation in the past, it would have been difficult to attribute the patient’s symptoms and signs to vitamin B12 deficiency after the initial evaluation. But once we see that the patient responds to treatment, we can presume that probably, the manifestations were due to the vitamin deficiency.

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Reference


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**A case of sporadic Creutzfeldt Jakob disease with anterior visual pathway involvement**

Sir,

CreutzfeldtJakob disease (CJD) typically presents with rapidly progressing dementia, myoclonus and rigidity,\(^1\) although early cortical blindness is known in the Heidenhain variant.\(^2\) Early and predominant anterior visual pathway involvement is very rare in sporadic CJD.

A 61-year-old previously asymptomatic lady presented with a 3 months history of progressive, painless bilateral visual loss culminating in complete blindness. Behavioral alterations manifested two months later as apathy, decreased word output, and a fluctuating sensorium. Over the next couple of months she developed gait and limb ataxia, and limb dystonia. She had no history of blood transfusion or surgery and was a vegetarian who occasionally consumed fish but never meat or beef. We first saw her in the ninth month of her illness. She was doubly incontinent, had no visual regard, withdrew limbs to painful stimuli, and vocalized spontaneously and to painful stimuli with no meaningful verbalization. Menace and pupillary light reflex (direct and consensual) were absent, optic fundii showed bilateral primary optic atrophy, deep tendon reflexes were normal and plantar response flexor.

Over the next four weeks of hospital stay she developed spontaneous as well as action-induced myoclonus with axial and appendicular rigidity.

Serum biochemical tests, thyroid functions, anti-thyroid antibodies, fine needle aspiration cytology and ultrasonography of the thyroid, and serum B12 levels were all within normal limits. HIV and ANA serology were negative. Cerebrospinal fluid (CSF) showed 2 lymphocytes/um++, sugar of 97 mg/dl and protein of 21 mg/dl. CSF Grams and AFB stains and cultures were negative. Flash VEP recorded prolonged P100 latencies bilaterally (154 msec). The initial three weekly EEGs showed diffuse slowing with triphasic waves (Figure 1) while the last EEG showed generalized short interval sharp and slow wave complexes at 1 to 1.5 second (Figure 2). Noncontrast (1.5 Tesla) MRI brain showed age-related atrophy and bilateral posterior thalamic hyperintense signal changes on T2 and PD images (Figure 3).

Our patient started with progressive blindness followed over next ten months by dementia and myoclonus. Other causes were ruled out by appropriate investigations. CJD was considered initially though the optic neuropathy and the absence of the characteristic EEG findings by ten months into the illness raised questions. The subsequent evolution of dystonia, rigidity, myoclonus, and the characteristic periodic complexes on EEG and posterior thalamic hyperintensities on