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Vitamin B_{12} deficiency can be a cause of acute reversible parkinsonism and cognitive impairment in older adults

Dear Editor,

Vitamin B_{12} deficiency is common in older adults and tends to increase with age, due to the inadequate dietary intake and impaired absorption of vitamin B_{12} because of atrophic gastritis, bacterial overgrowth or long-term use of medications for comorbidities, including proton pump inhibitors and metformine.[1] Up to now, much evidence is available showing that vitamin B_{12} deficiency is associated with many neurological disorders, such as dementia, ataxia, paraesthesia, weakness, gait abnormalities, peripheral neuropathy, psychosis, mood disorders and so on.[1] In contrast, parkinsonian signs are common among older adults, with prevalence estimates that range from 15% to 40%.[2] Although both vitamin B_{12} deficiency and parkinsonism increase with age, the coexistence of them is exceedingly rare in elderly patients. In this report, it is presented that acute parkinsonism related to vitamin B_{12} deficiency in an older adult was improved completely with vitamin B_{12} supplements.

An 81-year-old man was brought to the geriatric center because of rapidly progressive gait and balance disorder, recurrent falls, and forgetfulness for 2 weeks. His medical history revealed chronic gastritis, and he had been taking rabeprazole for a long time. No other disease or new medication was recorded. He had no symptoms of myelopathy or neuropathy and daily fluctuations, visual hallucination or delusions according to the information obtained from his wife.

On physical examination, his vital signs were stable, but he had orthostatic hypotension. Assessment of his mental condition showed cognitive impairment, disturbances of attention and slight disorientation. He presented with right hand tremor, asymmetric rigidity (right upper limb), bradykinesia and severe postural instability. His Mini-Mental State Examination (MMSE) score was 21. He didn't have any pyramidal or cerebellar signs. Examination of other systems was normal. The cranial MR was performed and demonstrated cerebral atrophy and chronic ischemic alterations with no acute neurological signs. He had normal hemoglobin level, but high mean corpuscular volume (97 fl). Biochemistry showed normal liver, thyroid, and kidney functions, with no electrolyte imbalance. Based on these findings without acute and rapid progressive course, it was considered as typically Parkinson's Disease (PD). Serum folic acid and vitamin D were normal. However, it was shown that serum vitamin B_{12} level was 72 pg/mL (normal >200 pg/mL) and homocysteine level 18 μmol/L (normal <15 μmol/L). Vitamin B_{12} replacement was started with injections at a dose of 1000 mg/day for 5 days, than once a week for 4 weeks and monthly thereafter. It was remarkable that there was progressive clinical improvement at the end of fifth day of the supplement therapy, and then he discharged. At the time of discharge, he had mild bradykinesia, and no tremors. He was followed up periodically. After 3 months, vitamin B_{12} level was elevated (>1000 pg/mL), and homocysteine was decreased (9.6 μmol/L), and all his previous parkinsonian signs and orthostatic hypotension were dramatically improved, he was more independent in daily living activities, and had better cognitive performance. His Mini-Mental State Examination score increased from 21 to 25.

In this case, it was presented that parkinsonism developed as a result of a vitamin B_{12} deficiency, and improved completely with replacement therapy.

Parkinsonism, whose underlying causes are numerous, is a clinical syndrome characterized by tremor, bradykinesia, rigidity and postural instability.[2] Parkinson's disease (PD) is the most common, but a broad range of other causes including some toxins, drugs and metabolic diseases might lead to similar motor symptoms.[2] Parkinsonism secondary to vitamin B_{12} deficiency is extremely rare, and the pathophysiology of extrapyramidal signs in patients with a vitamin B_{12} deficiency is unclear. The relationship between the two could be explained by several hypotheses. First, a deficiency of vitamin B_{12} can lead to elevated homocysteine levels, as in the present case. There is evidence that increased homocysteine levels might accelerate dopaminergic cell damage through neurotoxic and ischemic effects.[3] Furthermore, it was reported that the vitamin B_{12} level

was lower in PD patients than healthy controls, and replacement therapy can delay homocysteine-induced atherosclerosis, and might possess anti-oxidant and anti-inflammatory activities, both dependent and independent of the hypothesized homocysteine-lowering activity.[4] Second, as enzymatic reaction, which is dependent on vitamin B_{12}, is the conversion of methylmalonic acid (MMA) to succinyl-CoA, vitamin B_{12} deficiency can cause increased serum MMA. Larnaout A *et al.* showed that MMA led to acute severe symmetrical basal ganglia necrosis in a post-mortem neuropathological study.[5] Therefore, MMA also might be responsible for the development of acute parkinsonism in the present case. However, showing the reduction of dopamine transporter ligand-binding by dopamine transporter single-photon emission computed tomography or metaiodobenzylguanidine scintigraphy could be better to diagnose PD in these patients. In addition, serum vitamin B_1, whose deficiency might have an affect on the development of PD, should be evaluated in such cases.[6]

In conclusion, parkinsonism and cognitive impairment might be due to vitamin B_{12} deficiency only. Therefore, before PD diagnosis, and levodopa treatment, clinicians should absolutely evaluate serum vitamin B_{12} levels, and adequate supplement therapy should be implemented in these patients.

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Ulcerated colonic mass mimicking malignancy in an elderly patient

Dear Editor,

A 68-year-old woman was hospitalized in our clinic due to complaints of fatigue, dyspnea and intermittent hematochezia for 2 months. The medical history of the patient included rheumatoid arthritis, hypertension, coronary heart disease, congestive heart failure and hypothyroidism. There was also a history of total abdominal hysterectomy and bilateral salpingo-oophorectomy for ovarian granulosa cell tumor 12 years previously, and the patient was in remission. Long-term medications in use were methylprednisolone, leflunomide, amlodipine, metoprolol, clopidogrel, atorvastatin, levotiroksin sodium, furosemide and lansoprazole. On physical examination, her blood pressure was 130/80 mmHg, body temperature was 36.5°C and the abdominal examination was unremarkable. The laboratory test results showed: hemoglobin 11.7 g/dL (11.7–15.5 g/dL), leukocytes 6.1×10^9/L ($4.1–11.2 \times 10^9$/L), platelets 120×10^9/L ($159–388 \times 10^9$/L), alanine transaminase 20 U/L (0–50 U/L), aspartate transaminase 13 U/L (0–50 U/L), creatinine 1.3 mg/dL (0.6–1.1 mg/dL), albumin 2.64 g/dL (3.5–5.2 g/dL), international normalization ratio (INR) 1.2 (0.8–1.2), procalcitonin 0.198 ng/mL (0–0.1 ng/mL) and C-reactive protein 0.682 mg/dL (0–0.8 mg/dL). Severe hematochezia occurred during follow up and the hemoglobin levels decreased to 8.9 g/dL. Colonoscopy was carried out on the patient and showed an ulcerated, irregularly shaped mass 3 × 4 cm in size in the proximal ascending colon (Fig. 1a). The histopathological examination of the mass biopsy revealed ulcerated granulation tissue consistent with cytomegalovirus